Practical Orthopedic Examination MADE EASY®

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Second Edition

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To
My doting wife Neeta Verma
My loving son Siddhant
and
All my respectable teachers
and
Caring students...

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Preface to the Second Edition

After moving out of my parent institute, I realised that there are a few cases that postgraduates encounter during exams which are not covered in the current text. Brief teaching experience at Lady Hardinge Medical College and associated hospitals reaffirmed the need for second edition incorporating some more cases. I have not changed the original text to a large extent for simple reason that the basic approach to questions and their answer remain the same. Minor changes have been done in examination section of hip, knee and spine and ankle and foot cases. Additions are in the form of new cases for malunited distal radius fracture and intertrochanteric fractures. Hand section has been upgraded to large extent and now additionally carries cases on cut tendon injuries, carpal tunnel syndrome and malunited distal radius fractures. Some more questions have been added to Chapters 11 and 12. My current practice as joint replacement and arthroscopy surgeon has segregated me from most of the usual instruments and implants, so I could not gather enough courage to add chapter on instruments and implants, though I feel there an erstwhile need for the same. I promise to include section on instruments and implants and surgical exposures that are commonly asked in exams in future reprints/edition. I am thankfully obliged to my previous mentor Prof PP Kotwal for encouraging me to remain in academics and timely guiding me for current needs, he himself is a great academician. My current head Dr VP Singh had immensely cooperated by omitting my inconsistencies at work and extracurricular activities while providing me time and support to finish the writing work. New author, Dr Ram Kinkar Jha has given good support to revise spine section and writing carpal tunnel syndrome. Dr Aditya Soral has re-contributed in the form of new inputs and editing few deficiencies in the previous text. I am thankful to all the DNB students for making

the text a success, while myself being able to continue with my initiation of helping in academics for postgraduate students.

Most importantly in the end, I urge all DNB students who have read or are reading the current text (or even previous text) for providing me with regular inputs either at Facebook (Manish Kumar Varshney) or writing to me at manish.varshne@tatamotors.com (*y* unknowingly removed from this new e-mail ID). This will help me in generating the quality work useful to all.

Manish Kumar Varshney

Preface to the First Edition

The first edition of *Practical Orthopedic Examination Made Easy* has emerged after giving immense thought over the imminent need for a text that could serve to guide the postgraduates in the field for preparing through the ultimate acknowledgement of degree. The book specifically serves the stratified group of people preparing for orthopedic practical exams but also it does help a beginner in the field; gain theoretical knowledge and grab some basics often not found in modern orthopedic textbooks.

It has been an endeavor to keep the text as simple and directed to maintain interest of the reader. Also preservation of continuity in the text and standardization of information has been specifically dealt with. This gives an advantage for reader rehearse the examination scenario virtually and identify weaknesses to improve upon and strengths to maintain. While providing the most frequently asked questions and the 'expected' answers, consideration has also been given to some uncommon or unique questions with diligent answers. I have tried to provide as non-controversial an answer as possible with direct impact on the next possible questions while simultaneously being adequate. Still some controversial answers are detailed and are expected to be accepted by prevailing popular choice.

The examination points given at the beginning of every region serve as a quick synopsis along with comments on some uncommon tests that are still uncommonly asked. Figures have deliberately been omitted to make the material less bulky which considering the same are easily found in standard textbooks for orthopedic clinical examination. This work should be perceived in the deserved perspective and is not expected to substitute the standard orthopedic textbooks that have been the masterpieces in the field. When read as contemporary to the

standard textbooks, this work helps one understand the meaning of 'reading between lines' that is often the basis of some formidable questions stumbling even some avid book lovers. Organization of the book into regional affections and pertinent cases is more deliberate than accidental considering the relevance of 'regions' to examination cases. Some short cases however, have to be clubbed into miscellaneous section as they are either common to various sections or are full sections by themselves. Named signs, tests, procedures have been kept only to bare minimum necessary level as they are stressed only by an exceptionally uncommon unrealistic examiner unless one heads for a gold medal. No work is impeccable (that's why future editions follow) and readers' comments are delightfully welcome to improve upon the present text.

I personally wish appreciation to the various chapter contributors, colleagues and my seniors and junior residents in bringing this book to fruition in a timely manner. Particularly the constructive criticism and helpful suggestions given by Dr Mohit Singh in formatting the book needs mention. The book is a concise upshot of training and teaching from the expert and experienced teachers at Department of Orthopaedics, All India Institute of Medical Sciences to whom I am grateful.

All my sincere dedications and gratitude towards my wife Mrs Neeta Verma, will always fall short of the immense help I received from her in preparing the major part of this work.

Finally, I am highly indebted to the dedicated team of M/s Jaypee Brothers Medical Publishers(P) Ltd, New Delhi for giving constant encouragement and sage advice in the preparation of this book.

I wish the readers astounding success in examinations and accomplishments!

Manish Kumar Varshney

Contents

How to Approach Exams?

WHAT IS EXPECTED IN EXAMS?

After the immense hard work at orthopaedic residency program and practical experience spree it's difficult to put forward the argument in favour of a one or two-day assessment of the candidate deciding his fate to procure degree. Everyone has to still undergo the same stereotyped manner of judgment deciding one's 'fitness' for the degree. Nevertheless it is a matter of fact that the examiners have also undergone the same course over the years making it easier for a candidate to pass exam if it is taken in its "true perspective" which is often unknown! Candidates often find it an 'uphill' task to 'please' the examiners in exams for want of understanding of the main purpose of exams and the "desired perspective". The following points are projected to give an outline about what exactly is expected from candidate:

- *The way a candidate approaches the patient*: This should ideally be reflected upon as a firm but sober and professional approach to the patient (*always remember to be gentle and kind, be keen, be confident, be smart, be professional – it's not too much an ask!*). Give patient enough time (from your stipulated time, don't be thrift – believe me it helps; sometimes you will find that the patient tells you the diagnosis!). Explain every procedure to the patient and confirm the doubtful points to streamline your 'history taking' part. Follow the basic norms like to examine the normal side first for comparative assessment etc. Always pay particular attention to the surroundings, e.g. footwear, assistive appliances and make a note of them before starting 'your' "probation". This will tell the examiner that you know how to start solving the problem.

- *The way a candidate approaches the given problem*: Perhaps the most difficult part in the absence of practical experience (*good judgment comes from experience and experience from bad judgment*). A hands-on experience in out-patient department which is often time constrained and theory and academic classes give excellent opportunity to present patients to 'well-experienced' consultants – who are surely examiners for

others and possess immense experience – never flee away from them (Mistakes teach you the most – *The biggest fault of life is to think that you have none*). Ward rounds and patient work-up immensely help develop one's skills. Examination anxiety imposes a sense of hurry, and uncertainty about cases throws up disarray in approach which is often striven in the end by a quick fix solution that indeed jumbles up the problem turning on the vicious cycle. Always attempt to develop a unique but well-manageable and adequate approach for most common cases. Regular practice should make one comfortable in exams.

- *The way a candidate presents the problem and his viewpoint*: After examining the case appears the 'true' encounter whereby you will be assessed as regards your capability to communicate with the examiner about the case. For this *you should be a part orator, part clinician and part politician!* You should be smooth and focused in presentation with tactful representation of the facts leading to diagnosis but not projecting at the same time to the examiner that you are biased by a diagnosis. Keep your mind open to anything and be unprejudiced for criticism.

- *The understanding about the given problem and possible solutions*: Always answer to yourself in the following sequence of the problem and solution (What is the problem? → What can be done? → Why should it be done? → When to do it? → How to do it?; *What, Why, When, How*). Just making a diagnosis in a long case and particularly in a short case does not surmise the assessment. One is required to know the basic etiopathogenesis and management perspectives of the same. Particularly for some cases being a specialist in the field you may even be asked of the popular historical perspectives (*so you also need to be a historian!*). The treatment or management options you give require that you know fully about them and that it should fit the case. Don't beat around the bush, take your time. No one will fail you for being sloppy if your answers are correct, as even

the best of clinicians are perplexed sometimes by simple problems in unusual circumstances!

- *Explanation of most common solutions*: You should be the 'boss' of the most common procedures performed. Any question asked right-left or centre should be answered with the ease of cutting butter with a 'spoon' in summers.

HOW TO PREPARE FOR EXAMS?

This is the shortest written portion of this book specifically reiterating the fact that it's individual's way of learning, however, there may be some advice of help.

- Refer and follow the standard practice.
- Continuously practice and revise the important cases.
- Be focused.
- Develop communication skills! (Difficult; but of tremendous help, most candidates who fail are just unable to express what they actually mean although they are correct in essence).

DO's AND DON'Ts IN EXAMS

There are a few things that should be done and more importantly few that should not be done:

Do's
- Be calm and patient!
- Be tactful but considerate
- Be clear in perspective
- Be confident with apt knowledge
- Be frank
- Accept your mistakes if pointed out, be flexible, open-minded
- Finish with a smile to patient and examiner.

Don'ts
- Be in a hurry or anxious or overzealous
- Try to be clever

- Try to jumble up problems or become a researcher with innovative ideas!
- Mess up with facts or try guessing work!
- Be overconfident and arrogant
- Be dominative
- Argue
- Show disappointment or frustration over disagreement
- Make hasty conclusions!

HOW TO READ THIS BOOK?

A concise yet comprehensive attempt to recreate the questions may make some aspects of the book difficult to grab! It is well accepted that basic concepts regarding the management and diagnostic perspectives differ depending upon the personal and practiced algorithms by various 'experts'. You too may differ in various respects being amateur in the field (*Information consists of differences that make a difference*). The book definitely does not give a holistic approach to all the cases, but is a willful attempt to introduce the basics for revision with address to some intricacies of the cases that may need further refinement according to the local practice. It is imperative that at the important juncture of exams one needs support from the internal examiners who are basically 'internal experts' and would be able to better explain the 'locally practiced' protocols. I would appeal the readers to go through the book as-it-is once and then practice the cases. Mutual/group discussions over particular problems make fundamentals clear and easy to grab. Don't be in an illusion by 'illegitimate' concepts that are unacceptable. The book has been prepared after considerate and careful analysis of the various standard texts (more than 21 textbooks) and review papers (more than 650) that are acceptable to most by and large (the acceptability threshold may however differ!). The concepts given are standard for orthopaedic practice, but personally I will prohibit quoting of the text or concepts or guidelines given herein, one should adhere to standard textbooks or reviewers to this rescue. I feel here to reiterate the fact that what is written in this book is not

a hypothesis or personal research work, to give a sense of security to the readers. You will gradually come to know the source for manuscript while reading your textbooks. The read times are calculated approximations depending upon the general practice and exposure of graduates, complexity of topic, importance of topic and compactness of information provided. This, in general holds true but 'exceptions are a rule' so one may diverge considering his own knowledge and exposure and the local prevalence of particular topics (e.g. shoulder may be omitted fully while hip may be read twenty times!). General flow of questions in exams not necessarily follows the pattern given herein and in a "virtually-realistic" exam it is bound to differ, but one is always questioned of the reasons for giving a particular diagnosis, possible differential diagnoses (with reasons), management protocols, etc. That is primarily stressed upon in this compilation. Some stereotyped questions are oft repeated like 'What will you do next', the answer to which is often wrong in an anxious environment! And will hence be found in most of the chapters so that it becomes a habit of the candidate to correctly reply the same as it is the watershed between the diagnosis (considered less important part by the candidate) and management (more stressed part).

Try reading the chapters from beginning unless revising particular concepts as continuity has been specifically dealt with, otherwise some concepts may be overlooked. Teaching notes or text in italics is often ancillary and sometimes includes controversial and unsettled issues needing individual judgment for acceptance. Lastly, where a large amount of knowledge is presented with alternative procedures, the underlined text serves as the standard or most commonly followed method, but is again subjected to individual assessment for want of standardization.

In spite of the best efforts one may not accomplish the desired. The disheartening fate dictates some lapse in fundamental approach which all cannot be laid down in a narration and entails experience – remember hair don't just turn

grey in sun (*Experience enables you realize a mistake when you make it again!*). I surely do not mean that one needs to 'fail' to understand the cause of failure but it should be emphasized that prevention by a masterly carved approach pays rewards. There is no short-cut to success and exhausting effort with indelible persistence is required to become a specialist. Destiny is not a matter of chance but of choice, and the choice is still yours till you have to face exams. You can achieve what you want with a good strategy. Strategy building is not just a subject of fighting wars or winning elections – remember those times you devised time to see a movie or attend friend's marriage by quickly shuffling day's work – that's also strategy.

The Hip

{The hip joint is by far the most popular and common case given to candidates. The candidates are supposed to know all details of the case, especially the examination. Final diagnosis may not matter as much as the approach to it; however it should not be waywardly different from the expected ones. Being so frequent a case and multitude of approaches to the same a detailed presentation covering some controversial aspects also have been remarked.

Read: 6-8 times (MS and DNB candidates)}

EXAMINATION POINTS
FOR A HIP CASE

Before starting examination, always have a look around room to look for assisting devices like crutches/walker/stick and have a look at the footwear for shoe raise, etc.!

HISTORY TAKING

1. *Pain:* Onset (acute – traumatic/infective/reactive/muscular; insidious – degenerative, arthritic, osteonecrosis, TB (chronic infection)), duration, character (sharp shooting pain of trauma vs. dull aching pain of early osteonecrosis and arthritis to throbbing of infection), diurnal variation ('night cries' of tuberculosis, morning stiffness and pain of ankylosing spondylitis and mono-articular rheumatoid arthritis and RA), pain present at first step – arthritis but pain appearing after exertion – uncommonly of hip joint origin except early osteonecrosis, progression over time (undulating course – chronic disease, undulating course with sudden increase – collapse in osteonecrosis or fracture e.g. bone cyst or SCFE). Pain localized to groin suggests hip origin, but patient pointing pain over anterolateral region/ back of thigh suggest pain of upper lumbar and lower lumbar origin respectively. Pain that increasingly involve leg during exertion 'thermometer pain' suggests LCS. If pain is reported to be presented laterally above trochanteric region in c-fashion it suggests femoroacetabular

impingement. Note important negative history; pain in multiple joints (arthropathy), pain of spine origin as above and associated history e.g. pregnancy. Importantly, unexplained knee pain especially anterolateral region mandates hip examination. In itself pain is rarely of help in pointing out hip disorder.

2. *Limp:* A very important symptom that quite reliably localizes site of pathology. First symptom to appear in TB hip (even before pain). Note onset in relation to pain, progression, assistance in walking. Painless limp is often congenital (DDH, coxa vara, dysplastic disorders) or due to healed disease with deformity (healed infection with ankylosis or ankylosing spondylitis) or neuromuscular disorder (poliomyelitis/cerebral palsy).

3. *Stiffness (in patients language it is better to refer this as to 'Limitation of movement'):* Indicates spasm secondary to inflammatory disorder or enthesopathy or cartilage eburnation. Often seen following treatment of primary disorder or prolonged immobilization. Morning stiffness is often characteristic of non-infective inflammatory disorder.

4. *Deformity:* Often patient will be able to tell shortening but very infrequently you will find a patient telling you flexion/rotational deformities! Ask about when first noticed, association with pain, progression.

5. *Swelling:* Small swellings are often masked by bulk of muscle, large progressing swellings like that of TB hip and acute marked pyogenic infections should be indicated. Other swellings are often superficial and arise due to unrelated disorders (lymphadenopathy, saphena varix etc). Old unreduced dislocation is hardly mentioned by patient as swelling in gluteal region unless specifically asked for.

6. Describe the event (usually trauma) into mode (RTA, fall from height, fall of heavy object, slip on floor, missing stairs etc), site of injury, post injury mobility and ability to bear weight, injury to other regions, treatment received.

7. End your history by current disability experienced by patient, mobility status of the patient before event in question and finally mentioning all negative history:

– Trauma, fever, history of contacts for TB, affections of other joints, treatment history for TB, birth disorder. (*never forget to ask about symptoms in other hip and ipsilateral and contralateral knee, ankle and foot and mention it*)

Past history: TB and treatment, respiratory, renal, dermatological, neurological disorder, hematological disorder, connective tissue disorder, organ transplants, liver disorder, trauma and treatment, congenital/ developmental disorder and treatment, surgery around hip, diabetes, hypertension, and work tolerance (for treatment planning and anesthesia).

Personal history: Occupation, diet, smoking, alcohol intake, addiction.

Family history: Dysplasia, inflammatory disorder, storage disorders.

General Examination

Apart from other routine examination specifically look for clubbing, skin, lymphadenopathy (external/internal iliac, paraaortic), abdomen for psoas abscess and rarely tubercular ileitis, hemophilia, dysplasia, hypermobility syndrome.

Local Examination

Expose form nipples below and cover private parts to avoid embarrassment.

Standing

Gait (*See section 10a*)

Inspection

From front: A̲ttitude and A̲lignment (always start with 'A'– hip flexion, knee (patella) pointing out (external rotation) and vice versa and knee flexion, foot pointing out/in, equinus at ankle. Balance (front – flexion at hip and lateral – tilt to side). Level of ASIS, pelvic tilt, swelling (s), skin for scar/sinus/loss of creases, dilated veins, discoloration. Wasting of quadriceps,

prominent muscle (e.g. adductor spasm). Hernia sites and perineal widening.

From side: Lordosis of spine, pelvic tilt, trochanteric prominence, flexion at hip, knee and equinus at ankle, skin as above.

From behind: PSIS level (dimple of Venus), midline shift (natal cleft), and curvature of spine, lumbar triangle for fullness, lordosis, gluteal wasting, gluteal folds and symmetry, skin as above.

Note the skin for (SEADS): Swelling, erythema, atrophy (of appendages), discoloration, suppuration (scars and sinuses)

Palpation: (*mark all bony points – both ASIS, greater trochanter, pubic tubercle, PSIS, ischial tuberosities and iliac crest*)

Anteriorly: Note temperature from back of hand, ASIS levels, groin tenderness at base of Scarpa's triangle (hip joint is 2 cms below and lateral to mid inguinal point), femoral pulsations (best palpable just inferolateral to midinguinal point), swelling and abscesses, palpate femur along length.

From side: Trochanteric upriding, tenderness, broadening, thickening, levels of iliac crest.

From back: Tenderness over SI joint (lies just distal to PSIS), gluteal tenderness (short external rotators underlying gluteus can be a cause of pain), coccygeal tenderness (coccygodynia), tenderness over ischial tuberosity (weaver's bottom; bursitis), gluteal fold tenderness (gluteus maximus tendinitis), feel for swelling (spherical, smooth, bony hard of head of femur in dislocations) also feel for soft tissue swellings and abscesses.

Medially: Adductor spasm, ludloff's sign (tenderness over anteromedial aspect of thigh at base of Scarpa's triangle – lesser trochanteric affections)

Supine

Inspection and palpation: Same as above but rotational deformities can be better commented. Confirm above findings.

Percussion: Firm percussion at heel (Anvil sign – elicits pain at hip in inflammatory conditions)

Deformities:
- Fixed flexion deformity: First reveal the deformity by doing Thomas test
- Fixed adduction and abduction deformity: By squaring the pelvis.
- Fixed rotational deformities: There is no indirect way of measuring/estimating it. The deformity has to be measured using goniometer. One can take center of heel to 2nd toe as reference or medial border of foot as reference.

Movements: (*for all movements hold the pelvis firmly with left hand with thumb at the ASIS and fingers embracing trochanter – to detect even the slightest movement of pelvis, hold the affected limb with left hand*)
- *Flexion:* First reveal deformity by Thomas test then passively move the limb further and measure the range taking couch as reference.
- Adduction and abduction: First square the pelvis then measure the further range of respective movement taking body midline as reference (*if someone speaks 20º of abduction then it usually means 20º abduction beyond deformity; which is more appropriately expressed as range say 20-40°*).
- *Rotational movements:* Measure both in extension and 90º flexion at hip (if possible), in extension; measure further movement possible from deformity in that direction but for opposite movement measure from zero position (patella horizontal to ceiling facing directly up). In flexion measure from zero position only (hip flexed at 90º and leg parallel to midline).
- *Extension:* Measure in prone position
  (*Remember always to speak the movements of affected limb in comparison to other (deemed normal) limb, for e.g., flexion of 25º - 120º as compared to 0º-135º in left hip*)

Measurements: (*Limb length discrepancy*)
1. *Apparent length:* Measure in an unsquared pelvis with limbs lying parallel (the comfortable supine position of patient) from *Xiphisternum* to medial malleolus.

2. *True length:* Square the pelvis and put the limbs in mirror image (viz. flexion at hip, knee) and then measure from *ASIS* to medial malleolus. More reliably measured by wooden block method.
3. *Wasting:* Measure thigh circumference 15 cms from medial knee joint line.
4. Femoral (thigh) and tibial (leg) lengths
5. Galeazzi's sign and Ally's test.
6. *Bryant's triangle:* First square the pelvis. Draw a line from ASIS laterally horizontal (perpendicular to midline), draw a line joining tip of trochanter to ASIS (hypotenuse); draw a line passing straight up from tip of trochanter to intersect first line (base). Measure all in cms.
7. Femoral anteversion (Craig's test, Ryder method)
8. *Kothari's parallelogram (ML Kothari): unsquared pelvis* – Join both ASIS and drop perpendicular from each ASIS to midline, measure the angle formed between lines – this gives idea of Adduction/abduction in affected hip (*can be used as adjunct to classical method and is easy)*

Sitting

Usually done in patients who cannot stand. Look for posture and spine especially ankylosing spondylitis.

Special tests:
1. Qualitative assessment of supratrochanteric shortening:
   a. *Nelaton's line:* Turn the patient lateral to *bring affected side up* and flex the hip to 90°. Join ischial tuberosity (often the most distal part is the one most easily palpable) to ASIS. Supratrochanteric shortening is present if tip of greater trochanter crosses this line. As such it is drawn *only on the affected side*.
   b. *Shoemaker line:* In supine position join tip of trochanter to ASIS and extrapolate it to abdomen crossing umbilicus. Draw a similar line from *other side.* Normally the lines cross at or above umbilicus in midline. In unilateral supratrochanteric shortening the crossing always misses the midline and lies on the opposite side

below umbilicus. In bilateral symmetrical deformities the crossing point is below umbilicus but may be in center (if perfectly symmetrical).
   c. *Chiene's test:* Lines joining both ASIS and both tips of trochanter should be parallel – converge on the side of upriding.
   d. *Morris bitrochanteric test:* Interpretation as for hypotenuse of Bryant's triangle.
2. *Tests for stability:*
   a. Active SLRT (Stinchfield's test): If patient is unable to do then it indicates dislocation/fracture of neck or hip joint instability.
   b. Trendelenburg test
   c. Telescopy
   d. Ortolani's sign and Barlow's maneuver
3. Ober's test to test for ITB contracture (*See section 3a 'examination'*)
4. *Gauvain's sign:* spasm of abdominal muscles on initiating rotatory movements of hip in active tuberculosis, seen in stage of synovitis.
5. *Ely's test (Rectus phenomenon):* Tests rectus tightness – In a prone patient passive flexion of knee leads to flexion at hip joint.
6. Noble compression test for iliotibial band friction
7. *Yeoman's test:* Active hip extension against resistance to test Gluteus maximus tendinitis.
8. *Phelp's test:* To test gracilis tightness – In prone position, abduct the limbs then flex knee to 90° (relaxing gracilis) if further abduction present it indicates contracture.
9. *Tripod sign:* In sitting position passively extend knee fully if patient leans back and supports himself with both hands and extension at hip – indicates hamstring tightness (remember Lasegue's sign should be negative otherwise this could very well be positive in sciatica).
10. *Piriformis test (FADIR):* Flexion, adduction and internal rotation in lateral position stretches piriformis – produces pain in piriformis tendinitis/syndrome. *Same test if produces pain at groin is useful for femoroacetabular impingement (called impingement sign).*

11. *Patrick's test; (FABER) test; Jansen's sign:* Flexion, abduction and external rotation at hip putting lateral malleolus at patella – pain produced at SI joint. *Same test if produces pain at groin is useful for femoroacetabular impingement.*

12. *Erichson's pelvis compression test:* Press iliac crests together – pain over SI joint.

13. *Gaenslen's test:* Patient at edge of bed with ipsilateral hip hanging out. Flex both hips together then ask to release ipsilateral limb to extend hip – pain indicates SI joint affection.

14. *Yeoman's test for Sacroilitis:* Passive hyperextension of thigh in a prone patient.

15. *Fulcrum test:* To test stress fracture of femur – keep forearm below mid-thigh and press knee.

16. *Desault's sign:* On passively rotating femur, the tip of greater trochanter subtends an arc as the pivot is at the head of femur, but if the neck is fractured (non-union) then tip of trochanter remains stationary and rotates along longitudinal line passing through tip of trochanter itself (long axis of femur).

17. *Alli's sign:* Relaxation of the fascia between trochanter and iliac crest (seen in fractures of hip).

18. *Gill's sign:* Swollen hip due to effusion feels thicker than other hip felt with thumb at base of scarpa's triangle and four fingers over buttocks.

19. *Ludloff's test:* Inability to raise thigh in sitting position specially against resistance (avulsion of lesser trochanter)

20. *Sectoral sign:* Seen in Osteonecrosis – reduced internal rotation in extension that improves when checked in flexion.

21. *Gear-stick sign:* Limitation of abduction in extension but abduction improves with flexion of hip. These are due to replacement of diseased segment by healthy portion of bone so that impingement effect is gone (osteonecrosis and Perthes).

22. *Figure of '4' sign:* Click felt on making figure of 4 in osteonecrosis due to collapse of subchondral bone and left over shell of cartilage.

23. *Schober's test:* (*See section 8a 'measurements'*).

24. *McFarland's test:* During flexion, hip points to opposite shoulder but in SCFE and Osteonecrosis with superolateral collapse the hip points to ipsilateral shoulder.

Per rectal examination: For central fracture dislocation, otto pelvis (protrusio acetabuli).

Distal neurological and vascular examination: Check for distal motor deficit especially in sciatic nerve distribution, reflexes and sensations. Palpate all peripheral pulses (popliteal, anterior and posterior tibial, dorsalis pedis)

Always conclude with the status of other hip and ipsilateral and contralateral knee and ankle.

Some common classic findings as an example:
- Posterior dislocation: FADIR deformity with true (supratrochanteric) and apparent shortening, upridden trochanter (crosses Nelaton's line), head palpated in gluteal region, limitation of abduction and external rotation, telescopy and Trendelenburg positive, Gill's sign positive.
- DDH: Upridden trochanter, adduction contracture, absent/reduced femoral pulsations, asymmetric thigh folds, widened perineum (bilateral dislocations), Galeazzi's sign shows femoral shortening, higher gluteal fold, restricted abduction, increased internal rotation, limited hip abduction with 90° knee flexion, positive Barlow's maneuver and Ortolani's sign. In a young child 'duck-like' or 'sailor's gait' is seen (B/L) or a lurching gait (U/L), increased lordosis, true shortening as above, absent/reduced femoral pulses, positive Trendelenburg sign.

1. What are the prerequisites of examining hip joint?
Ans: The following points should be observed:
1. Patient should be lying supine on a couch or other firm mattress positioned away from wall.
2. Expose from below nipples and cover private parts.
3. Female attendant should accompany while examining a female patient.

4. Make patient comfortable by discussing with him and explaining the procedure before proceeding.
5. There should be ample light in the examining room preferably daylight.
6. Warm-up your hands before palpation.
7. Avoid hurting the patient.
8. Examine in standing, supine and sitting positions.

2. What is Narath's sign?

Ans: Absent (or feeble) femoral pulsations due to lack of bony support below (head of femur); seen in old unreduced dislocations, DDH, squeal of septic arthritis, pestle and mortar type TB hip, central fracture dislocation of hip, arteriosclerosis of femoral artery (The Narath's sign should not be spoken as positive or negative – always mention that femoral pulses are bilaterally comparable or femoral pulses on affected site are feeble/not palpable).

3. What are the causes of asymmetry of gluteal folds?

Ans: DDH, gluteal abscess, gluteal atrophy, fixed pelvic obliquity, gluteus maximus contracture, hematoma/collection/psoas abscess in gluteal region, tumors.

4. What are the sites of psoas abscess that can be clinically examined?

Ans: Abdomen (iliac fossae), base of scarpa's triangle, gluteal region, supratrochanteric region, anteromedial aspect of thigh (sub-sartorial canal and through adductor hiatus), popliteal fossa, lumbar triangle at back, flanks.

5. What is the lymphatic drainage of hip "or" what groups of lymph nodes do you examine for hip case?

Ans: External iliac, internal iliac, paraaortic group of lymph nodes, also some drainage to deep inguinal group of lymph nodes is present.

6. What is the nerve supply of hip joint and what is its importance?

Ans: Variegated nerve supply:

1. Femoral nerve via rectus femoris
2. Obturator nerve anterior division
3. Accessory obturator nerve
4. Nerve to quadratus femoris
5. Superior gluteal nerve.

Pain of hip joint may be referred to knee joint (via femoral and obturator nerve) and may be the primary presentation in early pathology. Knee joint is also supplied by femoral nerve via vastus medialis, posterior division of obturator nerve apart from sciatic nerve (genicular tibial and common peroneal)

7. How do you identify various bony points?

Ans: *ASIS:* Slide metal end of tape (or thumb) gently along the inguinal ligament superolaterally in the groin fold and the first bony prominence encountered is ASIS.

Pubic tubercle: Lies about 2-2.5 cm lateral to pubic symphysis (serves as attachment for inguinal ligament)

Tip of greater trochanter: From ASIS go along iliac crests and mark some 5 cms arc a hand breath behind ASIS. From here gently descend down and the first bony point felt is tip of greater trochanter, confirm this by gently rotating thigh. (Alternately some people may mark it by going along femoral shaft and the place where your thumb dips is tip)

PSIS: Marked at dimple of Venus.

Ischial tuberosity: Patient lateral and hip flexed at right angle feel right in the center of gluteal region and mark the base (most prominent part).

8. Why do you choose Xiphisternum as the bony landmark for measuring apparent length?

Ans: A fixed easily identifiable reference in *midline* of body is required to compare apparent length, the following landmarks are available:
1. Suprasternal notch
2. Xiphisternum

3. Umbilicus
4. Symphysis pubis.

Landmark 1 lies too high (unexposed) and is quite wide – may give erroneous measurement. Landmark 3 is not fixed as it can move with slightest of hand movement. Landmark 4 lies below the level of hip joint and as a rule no landmark below the hip is chosen. So it is better to choose 2.

9. Does the true length measurement change with hip position?

Ans: The line ASIS → medial malleolus passes lateral to hip joint so moving from full abduction to adduction increases the limb length by 1-2 cms. Also flexion deformity gives erroneous limb shortening, hence mirrored limb position with squaring of pelvis is recommended.

10. Why and how do you square pelvis?

Ans: Squaring is done to remove the effect of compensation by body (e.g. apparently lengthening a shortened limb), by reversing the deformity produced due to compensation. Squaring is done by abducting the limb in fixed abduction deformity and vice versa. Moving the limb this way always first corrects the fixed deformity then only movement occurs at joint.

11. What are the fallacies of squaring pelvis?

Ans: Squaring is not possible for:
- Absent ASIS due to previous surgery
- Fixed pelvic obliquity/ scoliosis
- Malformed pelvis
- Deformed pelvis e.g. following trauma
- Ankylosing spondylitis with sixed spinal and hip deformities

12. What does apparent length measure?

Ans: As such nothing specific! It is only a rough guide and a confirmation to visual appearance of shortening/

lengthening. Interpretation is possible only in conjunction with true length.

13. **What do you understand by abduction and adduction deformities?**

Ans: These are coronal compensatory mechanisms of body for limb length inequality. Abduction deformity denotes apparent lengthening and adduction deformity denotes apparent shortening.

Simple deformities: These are present in early stages only; these can only be detected while examining the patient standing! And they often go away on lying down – that's why not fixed. (*They are referred to as 'adduction/abduction deformity'*). Movement in direction opposite to deformity may still be possible as the deformities are not fixed.

Fixed deformities: Over time without treatment the deformities become fixed due to soft tissue contracture whereby 'movement is still possible in the direction of deformity but not in the other direction'. (*They are referred to as 'Fixed abduction/adduction deformity'*)

Lastly limb may get ankylosed in one of the deformities and there it is called '*Fixed in abduction/adduction*'.

They are measured by squaring pelvis or by Kothari's parallelogram.

14. **What are various compensatory mechanisms and what is their importance?**

Ans: Compensation for hip deformity occurs at:
- Spine (often lumbar)
- Pelvis (coronal tilt or flexion/extension)
- Hip joint itself (coronal deformity)
- Knee
- Ankle
- Foot
  They are physiological response of body to a pathological condition and must not be thought of as a disorder! They:
- Conceal deformity

- Maintain equilibrium while standing/ walking by shifting center of gravity
- Stabilize the joint (remember they '*fix*' the joint)
- Make up for the loss

15. Can all deformities around hip be compensated?

Ans: No, there is a limit to compensation for rotational deformities which often remain revealed unless very mild. Only coronal and saggital plane deformities are corrected, that to only a certain extent.

16. How much shortening can be effectively compensated without producing ankle equinus?

Ans: Approximately one and a half inch (around 3-3.5 cms).

17. What is Thomas test?

Ans: Test for indirectly revealing and measuring flexion deformity.

18. What are the prerequisites of performing Thomas test?

Ans: The following should be observed:
   1. General prerequisites of examination
   2. Unilateral deformity (bilateral measured by modification)
   3. No bony ankylosis in other hip/knee.
   4. Other hip should be painless, ipsilateral hip should not be very painful
   5. No fixed pelvic/spinal deformity

19. How do you perform this test?

Ans: Stand on right side of couch, explain the procedure. Pass your hand behind back volar side up. Manipulate the asymptomatic limb to flex the hip and knee to fullest obliterating lumbar lordosis. Check for ischial tuberosity movement simultaneously and it should not rise off couch (this denotes over-flexion). Ask patient to hold the knee in same position. Passively gently extend the affected limb at thigh to correct any overcorrection, correct any accidental abduction at hip and measure the angle (couch to back of thigh) after reconfirming obliteration of

lordosis. Alternate method – ask patient to flex both limbs so that knees touch chest. Then ask to release the affected limb while holding the normal knee to chest.

(*Passing hand beneath the back only checks for the presence of lumbar lordosis, inability to pass the hand means obliteration of the same. Do not insinuate your hand again while measuring the flexion deformity as, one it is uncomfortable and two it may reproduce the deformity decreasing the exact measurement!*)

20. What will you do for bilateral deformity?

Ans: I will do the test in prone position (modification) – both lower limbs dangling off the couch. Support both thighs and obliterate lumbar lordosis in direct vision then measure the flexion deformity from imaginary horizontal parallel to floor. Other more practical way is to test in lateral position and obliterate lumbar lordosis.

21. What precautions you should take care of and what are the drawbacks of this test?

Ans: One should not allow over-flexion and lifting off pelvis. There should be no abduction in the affected limb while measuring deformity – it spuriously reduces the deformity. *Drawbacks:*

1. Exact flexion contracture cannot be commented if adduction/abduction deformity coexists
2. Not comfortable for a patient with already painful hip
3. Inaccurate in obese and uncooperative patients
4. Difficult to do prone test for bilateral deformity.

22. What is the principle behind Thomas test?

Ans: Fixed saggital plane deformity is compensated by pelvic extension (flexion deformity) and vice versa. This is produced by lumbar lordosis which shares the compensation. The combined effect allows a patient to walk with feet touching the ground in conjunction with knee flexion. While we flex the normal hip the deformity in pelvis first gets corrected (something akin to squaring pelvis) then lumbar lordosis is corrected simultaneously revealing the deformity.

23. Can we test by manipulating affected hip?

Ans: Of course we can, it will be similar to squaring pelvis but the patient with painful hip will be hurt and also such a maneuver will make him apprehensive. This is what is done in alternative maneuver described above.

24. Why do you keep the other limb flexed?

Ans: To keep the pelvis fixed in corrected position.

25. What are the causes of false-positive Thomas test?

Ans: Wrong technique is the most common cause.
Others are:
- Fixed pelvic obliquity in scoliosis and polio
- Exaggerated lordosis in obese individuals
- Malformed pelvis.

26. What are the movements of pelvis?

Ans: Internal movements (within bones – minimal); external movements (extension – superior pelvis moves forward; flexion – superior pelvis moves backwards). Just to remember one F—K's in Flexion.

27. How much flexion deformity can be hidden by lumbar lordosis?

Ans: Up to 30°.

28. What are the causes of increased lumbar lordosis?

Ans: The various pathological and physiological causes of increased lumbar lordosis are:
- *Suprapelvic:* Pregnancy, obesity, spondylolisthesis, constitutional
- *Pelvic:* Congenital defects, rickets and developmental defects
- *Infrapelvic:* B/L or U/L flexion deformity of hip, DDH

29. Which movement is first lost in hip pathology?

Ans: Extension followed by rotations, adduction, abduction, and lastly flexion (this sequence is followed in synovial disorders – increased volume whereas those in which

there is direct injury to cartilage abduction is lost earlier due to adductor spasm – adductors are stronger!)

30. What is the position of rest for hip joint?

Ans: This is the position in which the volume of hip joint is maximal: 10° each of flexion, abduction and external rotation.

31. How do you do Trendelenburg test?

Ans: There are at least four different methods vaguely described in various textbooks. The following is the most standard method studied and described.

1. Stand behind patient. Observe the angle between pelvis (line joining iliac crests) and ground. Ask patient to stand on unaffected side first, lifting affected side foot and flexing hip between neutral and 30° and knee to clear foot off the ground (this is done to nullify the effect of rectus femoris). Note the position of pelvis. {In some patients to maintain balance either a supporting stick can be used on the hand of weight-bearing hip or examiner can support both shoulders}.

2. Then ask patient to raise the affected side of pelvis as high as possible. (One may provide support to the patient by holding arm of the weight-bearing side). Correct any tendency to lean over the weight bearing side by bringing shoulders at same level.

3. Repeat the same on affected side (the side to be tested). *Interpretation:* Normally ("negative test") one is able to lift the other side (watch iliac crest) without losing balance for at least 30 sec and the lift is equal to the abduction possible at that hip.
   Gluteal folds have been long propagated as 'standard' reference for judging pelvic lift but a lot of limitations arise primarily due to muscle wasting so common in hip disorders. PSIS is a good reference if there is significant gluteal wasting or folds are asymmetrical, however it is too near to midline to judge pelvic lift and be taken as a primary reference.

Alternately one can less reliably stand in front of patient and support patient's palm. Perform the test the same way but notice the pressure transmitted by patient's palm when they attempt to balance. Increased pressure in opposite side in an attempt to gain support from you suggests positive test.

32. What constitutes a positive test?

Ans: Any of the following constitute a "positive test": (Abnormal response)
1. Maximal elevation not achieved.
2. Sustained elevation not achieved (for 30 sec) – Delayed abnormal response.
3. Iliac crest not elevated (pelvis parallel to ground).
4. Pelvis drops down (opposite iliac crest).

33. What does this test assess?

Ans: It is a screening test to check integrity of abductor mechanism comprising of head and acetabular socket as fulcrum, neck and trochanteric region as lever (site of effort), abductor (primarily Gluteus medius aided by TFL) as power, load is the lower limb distal to trochanteric region. Hip is a class – 3 lever! Insufficiency at any level is reflected as a positive test.

34. What can produce a false positive test?

Ans: Painful hip, poor balance, uncooperative patient, costo-pelvic impingement (as in scoliosis).

35. What can produce a false negative test?

Ans: Use of suprapelvic muscles by patient, use of psoas and rectus femoris, wide lateral translocation of trunk to allow balance over the hip as a fulcrum.

36. What are the prerequisites for doing this test?

Ans: There are a lot of limitations that itself are frequently seen in hip pathologies, so it is not a very good test:
• It should not be a very painful hip (spuriously positive).

- There should be no abduction or adduction deformity in any hip.
- Quadratus lumborum must be normal (affected in polio) – this effects a normal Trendelenburg test.
- In obese and patients with medial shift of lower limb mechanical axis the test may be pseudo-positive.
- Sacroilitis may produce a positive test.

37. Who described Trendelenburg test?

Ans: Originally described by "Duchenne de Boulogne" in 1867 but was rediscovered by Friedrich Trendelenburg in 1895 for assessment of DDH.

38. What are the causes of a positive Trendelenburg test?

Ans: *Gluteus medius paralysis:*
- Polio, L5 radiculopathy, girdle muscular dystrophy, cerebral palsy (and other disjunctive disorders) etc. *Power of 5/5 MRC grade is required for normal response ("negative test") any power less than or equal to 4 will produce positive test. Failure of lever:*
- Trochanteric avulsion, fracture neck of femur, coxa vara *Disruption of fulcrum:*
- Dislocated hip, DDH or subluxating hips, Perthes disease, SUFE, osteonecrosis. *Gluteal inhibition:*
- Painful hip due to arthritis/infection, Sacroilitis.

39. Is it possible to mask trendelenburg's sign or resulting gait?

Ans: (*An impractical but a very conceptual question – concept can be utilized in conservatively managing unreconstructible abductor paralysis*). Yes, if one carries a weight of 6-7 Kg on the affected side while walking, the lurch can be largely obliterated. This is due to shift of center of gravity towards the affected side and hence masking the weakness.

40. How do you test for telescopy?

Ans: Patient supine on bed. Fix pelvis by left hand (trochanter should be embraced by fingers). Flex hip to more than

60° (preferably to 90°) and adduct the limb slightly (to put hip in vulnerable position) then apply a Push-Pull force along the axis of shaft and note first the translation of trochanter and secondly crepitus from joint. (*Remember this is a provocative test and offensive one to the patient – be gentle!*) (*Now, whether to push or pull first – don't be concerned, result will not change!*)

41. What are the prerequisites for telescopy?

Ans: Hip and knee should have flexion range of movement; adduction should be possible at hip, firm mattress, ideally a painless hip.

42. What are the causes of a telescoping hip?

Ans: Causes in hip joint *per se:*
- Girdlestone arthroplasty,
- Old unreduced dislocation with lax structures
- DDH
- Pathological dislocation e.g. TB
- Charcot's joint
- Perthes disease
- SCFE
- Squeal of septic arthritis
- Osteonecrosis with collapse
- TB hip: Mortar and pestle type, destroyed head, wandering acetabulum
  Causes out of hip joint:
- Non-Union fracture neck femur
- Non-Union fracture intertrochanteric

43. What are Ortolani's and Barlow's tests?

Ans: Better called Ortolani's test and Barlow's maneuver. These are basically for evaluation of DDH.

Ortolani test (1947); Froelich (1911); LeDamanu (1912): Hips flexed 90° and knees flexed. Start from adducted position and slowly abduct hip whilst exerting pressure over greater trochanter. Reducible dislocated hip gives 'clunk of entry' feeling. Usually observed till 3 months of age.

Barlow's maneuver (1962): Modified Ortolani's test. His test has two parts first one similar to Ortolani's test but the hips are sequentially tested in 45-60° flexion (hip is more likely to be unstable in this position while the other one fixes pelvis). 2nd part is Barlow's test for dislocatable hips – exert outward pressure on hip; if it dislocates but reduces then it indicates dislocatable but not dislocated hip.

44. What is thickening and broadening of trochanter?

Ans: Thickening of trochanter is identified by holding it between index finger and thumb whereas broadening is observed by palpation over trochanter. Causes are often same!

- Tumors, cysts, malunited (or uniting or non-union) intertrochanteric fractures, postoperative (fixation of fractures around hip, osteotomy), infection, Perthes.

45. Interpretation of Bryant's triangle?

Ans: *Shortening of base:* suggests upriding of greater trochanter seen in – coxa vara, coxa breva, destruction of head, non-union fracture neck femur, old dislocations, girdlestone arthroplasty, Perthes disease.

Shortening of perpendicular only: Suggests internal rotation of femur or central migration of head – old unreduced posterior dislocation, central fracture dislocation etc.

Shortening of hypotenuse: It is always associated with shortening of either or both of other lines (*Remember Pythagoras theorem*).

Isolated shortenings are rare and a combination is often seen:

Base + perpendicular shortening (definite hypotenuse shortening): Central fracture dislocation, neck resorption, head resorption, protrusio acetabuli.

Base shortened but perpendicular lengthening: Non-union fracture neck femur, anterior dislocation of hip, SCFE, girdlestone arthroplasty.

Base shortened + lengthened perpendicular: Malunited fracture neck femur

All lines lengthened: Coxa magna, coxa valga.

46. What are the fallacies of Bryant's triangle measurement?

Ans: Useless in bilateral affections, if ASIS cannot be palpated, lines are quite arbitrary and errors are easy to occur.

47. What are the various sites of referred pain of hip joint?

Ans: Various patterns observed are:
   a. *Obturator pattern:* Pain deep in groin radiating to medial thigh.
   b. *Posterior pattern:* Deep in the buttock (most common).
   c. *Femoral pattern:* Pain from front of joint radiating along anterior thigh.
   d. *Lateral pattern:* Over greater trochanter radiates along lateral thigh to knee.
   e. Uncommonly pain can also radiate to leg and foot.

48. What are the criteria for making diagnosis of ankylosing spondylitis?

Ans: *Rome criteria* (1963): *Ankylosing spondylitis (AS) present if Bilateral Sacroilitis + any of the below:*
   • Low back pain with stiffness for > 3 months
   • Pain and stiffness in thoracic region
   • Limited motion in lumbar region
   • Limited chest expansion
   • History of iritis or its sequel

   New York criteria (1968): The following clinical criteria:
      1. Limitation of motion of the lumbar spine in anterior flexion, lateral flexion and extension
      2. History of pain in thoracolumbar junction or in the lumbar spine
      3. Limitation of chest expansion to $\leq$ 2.5 cm (1 inch)
   • Definite AS = Grade 3-4 bilateral Sacroilitis + at least one clinical criteria "or" grade 3-4 unilateral or grade 2 bilateral Sacroilitis + clinical criteria '1' or both '2' & '3'
   • Probable AS = Bilateral Sacroilitis without any clinical criteria.

BASDAI (Bath Ankylosing Spondylitis Disease Activity Index) detects the inflammatory burden of disease. It can help establish the diagnosis of AS in the presence of other factors like HLA-B27 positivity, persistent buttock or back pain that resolves with exercise, and X-ray and MRI involvement of sacroiliac joints. *(Apart from HLA-B27, two new genes have been recently associated with AS – ARTS1 and IL23R)*

49. Who described Kothari's parallelogram?

Ans: Dr. Manu Kothari described this alternative maneuver to look for coronal plane deformities at hip joint without squaring the pelvis *(He was a second year MBBS student at that time and retired as professor of anatomy from KEM Mumbai).*

50. What are the common conditions affecting hip bilaterally?

Ans:
- Osteonecrosis of hip (bilateral in 50%)
- Perthes disease (10%)
- Coxa Vara (50%)
- SUFE/SCFE (25-30%)
- DDH (30-40%).

CASE I:
TUBERCULOSIS (TB) OF HIP JOINT

Diagnosis

The patient is a 17 year old male with 6 months old conservatively managed TB of right hip on Anti-tubercular therapy (ATT) in healing stage. Patient has 2.5 cm of true shortening (supratrochanteric) with flexion deformity of 20°, adduction deformity 15° and internal rotation deformity of 10° with limitation of all movements. Patient is able to do activities of daily living but not his other routine activities.

(From here viva can take any direction as this is an open and adequate diagnosis; like – how do you measure flexion/adduction deformity/true

shortening or what is the range of flexion etc. – this often entails from the fact that there are multiple candidates and a comparison is coherent to mark candidates, hence common questions are legible).

1. Why do you think it is tuberculosis of hip?

Ans:
- Protracted history with insidious onset pain; night cries
- Age group (As a rule <10 year old patient population affected; TB spine:TB hip=10:7)
- Association with progressive limp that appeared early
- Typical deformity
- Restriction of movements in all direction ('Global' restriction of movements)
- Relief with treatment
- Wasting and constitutional symptoms.

2. What is your differential diagnosis?

Ans: I will give a differential according to more common possibilities first:

1. *Subacute septic arthritis:* Heals quite early with treatment otherwise atypical forms closely resemble TB (TB in Indian subcontinent gets benefit of doubt and is often the first clinical diagnosis).
2. *Proximal femoral osteomyelitis with reactive hip involvement:* Thickening/broadening of trochanter, irregular proximal femur, sinuses, reactive effusion give (FABER) deformity.
3. *Osteonecrosis of femoral head with early cartilage eburnation:* Limp and deformities appear late.
4. *Late onset Perthes disease:* Less common, less flexion deformity, movements not limited in all directions, should not respond to treatment.
5. *Mono-articular rheumatoid:* Very uncommon, occurs in middle aged/elderly, should not respond to ATT.
6. *Non-union fracture neck of femur:* Trauma, characteristic initial history, treatment history, telescopy, deformity (flexion, external rotation).

7. *Central fracture dislocation of hip:* Movement in one plane is preserved (*however even in TB in Indian patients flexion is often present due to practice of squatting which is a daily practice*): Trauma is definitely present.

8. Ankylosing spondylitis: oops you are given the whole patient not just a joint – examine in totality (*don't go so close to a tree that you lose the site of forest*).

3. What can be your other differentials?

Ans: Depending on specific symptomatology I will classify my differentials into:

1. Limitation of movements:
   a. Irritable hip
   b. UMN lesion: Spasms overcome by pressure, not painful, no wasting.
   c. Reflex irritation from lymph nodes etc.

2. Limp:
   a. DDH: Certain movements exaggerated
   b. Coxa vara: $\uparrow$ external rotation, adduction – external rotation deformity
   c. Perthes disease
   d. Irritable hip.

3. Pain:
   a. Osteomyelitis and septic arthritis
   b. SCFE
   c. Early poliomyelitis
   d. Irritable hip.

This question has been deliberately added to complete the remotest possibility for TB hip differential and enabling the candidate face examiner's lethal weapon –"And… "

4. Why do you call it healing "or" in other words what are the signs of active disease?

Ans: There is no rest pain, night cries ('starting pains'), no joint swelling, Gauvain's negative, characteristic deformity of stage III.

5. How do you stage TB hip?

Ans: Clinical staging (this is the sequence of untreated TB, treatment may arrest it anywhere!):

Stage	Pathology	Attitude
1. Stage of apparent lengthening (due to pelvic tilt to compensate for abduction deformity. >75% pain free movements, No true shortening)	Synovitis/ Effusion	FABER (flexion, abduction, external rotation)
2. Stage of apparent shortening (pelvic tilt to compensate adduction deformity (spasm), movements restricted beyond 50%, true shortening either none or <1 cm)	Early arthritis	FADER (Flexion, adduction, external rotation)
3. Stage of true shortening. (Fixed deformities, movements restricted to <25%, Real shortening >1 cm)	Advanced arthritis	FADER with shortening
4. Stage of aftermath and destruction (wandering acetabulum, pathological dislocation, destruction of head, fibrous ankylosis etc.)	Ongoing gross destruction	Shortening $\uparrow^s$ further, deformities can vary depending on the final outcome.

6. Why do the deformities differ in different stages?

Ans: In stage I due to effusion and $\uparrow^d$ requirement for space hip goes into FABER deformity, later in stage II the deformities are due to spasm of muscles. Flexors and adductors are stronger than other groups so characteristic

FADER deformity is exhibited. Moreover irritation of inferomedial joint capsule by debris irritates obturator nerve causing adductor spasm and direct irritation of iliopsoas occurs by underlying swollen, hyperemic capsule. In stage III there is eburnation of cartilage and generalized spasm increasing bony contact and enhancing destruction but above two groups dominate to maintain FADER deformity.

7. Can you still see flexion, abduction, internal/external rotation deformity in a patient in stage II/III?

Ans: YES:
- Patient treated by prolonged traction
- Patient maintained in hip spica
- Elderly or debilitated patients who prefer to lie in lateral position and continue with initial posture for relief of pain, moreover they have weak muscles
- Destruction of iliofemoral ligament (inverted Y-ligament of Bigelow)
- Patient who continues to bear weight in the initial deformed position.

8. What is the cause of night cries?

Ans: Destruction of cartilage exposes the subchondral nerve endings. In the night with muscle relaxation, the splinting effect is taken away and the bony surfaces rub across each other causing severe pain.

9. How will you confirm diagnosis?

Ans: I will get an X-ray of pelvis done (AP) view with radiographs of right hip in AP and lateral projections. *Blood tests:*
- Serial ESR
- ELISA: IgG and IgM response to A60 Antigen complex (interspecific antigen and is common to typical and atypical mycobacteria) – sensitivity of 60-80%
- PCR is highly sensitive and specific *(lots of them are available, still more reliable and specific is the RNA testing but is much expensive)*

Skin tests: False positives and negatives high; strongly positive reaction in previously negative patient is highly suggestive (but hardly found in an adult patient).

Synovial fluid examination/ synovial biopsy/bone biopsy:

- ↑ Protein, poor formation of mucin clot etc. in synovial fluid examination
- Staining (Ziehl-Neelson)
- Culture:
  - Provides ultimate diagnosis
  - But is very slow (Rapid alternatives are BACTEC TB-460, BACTEC MGIT960 etc.)
- Rapid methods:
  - Centrifuged samples
  - Thin-layer and gas-liquid interphase chromatography for detection of lipids and long-chain fatty acids
  - Radioactive labeled DNA probes: specific to diagnose family, genus, species, sub-species – result in 2-8 hours
  - MDR rapid diagnosis: genotypic analysis of six codon regions rpoB531, rpoB526, rrs513, rpsL43, embB306, and katG315
  - Molecular hybridization techniques.

Staining requires around 10,000 bacilli/mm^3, culture requires 1000 bacilli/mm^3.

10. What do you expect to see on an X-ray?

Ans: As per the examination I expect to see involvement of both femur and acetabulum with reduction of joint space, cystic sclerotic lesions in head and acetabulum, acetabular widening and/or destruction of femoral head, upriding of greater trochanter, flexion adduction and external rotation deformity, with or without subluxation of joint and displacement of fat planes.

11. Does this confirm your diagnosis?

Ans: Yes ☺ or No ☹.

12. How do you clinico-radiologically classify TB?

Ans: Shanmugasundaram classification:

1. Normal hip
2. Travelling acetabulum
3. Dislocating type
4. Perthes type
5. Mortar and pestle type
6. Atrophic type
7. Protrusio acetabuli

(More commonly in *Adult* the Atrophic form; *in children*: normal, perthes and dislocation types, while mortar & pestle, wandering and protrusion types are seen in *both*)

13. What are the common radiological signs of TB hip?

Ans: Osteoporosis is the earliest sign, 'Cat-Bite' lesion in articular margin, ↓ joint space, destructive changes. Bony lamellae undergo osteoporosis in early stages (osteoclasts and howships lacunae) later with healing there is osteosclerosis (osteoblastic activity).

14. What are the radiological signs of healing?

Ans: The following are seen in healing TB:
1. ↑ in thickness of trabeculae
2. ↑ density of bone
3. Recommencing of epiphyseal growth.

15. What is the pathology of an untreated hip?

Ans: *Synovial type:* Hypertrophy, congestion, unhealthy hypertrophic granulation tissue formation (pannus) → cartilage destruction → fibrosed and thickened synovium → granulation tissue bridges bony surfaces → fibrous ankylosis.

Bony extension type: Occurs via subperiosteal space or direct metaphyseal spread → cartilage is the only barrier → once breached leads to growth plate destruction → deformities and shortening.

Clinical types of hip joint TB:
- Granular form: Common in adults, protracted course, less destruction, ↓ tendency for cold abscess formation.
- Caseous type: Severe constitutional symptoms, more common in childhood, destruction and abscess common.

• Tubercular rheumatism (Poncet's disease): Asymmetric polyarthritis + focus of infection.

16. What are rice bodies?

Ans: Accumulation of fibrin and articular cartilage.

17. What is the focus of infection in TB?

Ans:
  1. Synovial: often protracted course
  2. Osseous:
      i. Acetabular side
     ii. Head of femur "Babcock's triangle": It is an area which is the watershed between obturator and femoral circulation and bone is weaker in this region. It lies towards the cervical side of lower part of head and proximal part of neck in lower half near epiphyseal line.
    iii. Neck of femur: see above
     iv. Trochanteric region.

18. How will you treat this case?

Ans: The aim is to obtain ideal outcome of a painless, mobile, stable hip. But the patient's disease has already been present for 6 months and is in stage III so treatment has to be provided according to present situation. I will continue with chemotherapy and advice for heliotherapy, liberal diet, fresh air, restrain from exertion until healing, traction to correct and maintain deformity with intermittent mobilization.

19. What is the role of chemotherapy and what is the regime?

Ans: Chemotherapy is absolutely essential and should be a combination chemotherapy (at least one of which is bactericidal) and prolonged for long to kill persisters. WHO regime for skeletal TB is apparently inadequate and we follow intensive phase for 3 months of four drugs (INH, RIF, PZE, ETH) by which time the results of culture sensitivity are available to evaluate primary resistance, followed by extension phase for 4 months of three drugs

(INH, RIF, PZA), and followed by continuation phase for 11 months of two drugs (INH, RIF). (*Please follow your institutional regime if it differs from above or when in doubt you can always bank upon WHO regime, Category III and ignore all above*). However, in general the intensive phase (aka initial phase) kills the rapidly multiplying extracellular bacteria while continuation phase is aimed at killing the dormant bacteria and preventing recurrence.

{*In Asian sub-continent one is expected to know the whole regime, drugs (first and second line), dosage, side-effects, and interactions for ATT. You should refer to the standard pharmacologic texts for this – it's essential!*}

20. What is the role of traction?

Ans: Traction is used for:
1. Overcoming spasm and deformity due to same
2. Providing/enforcing rest
3. Maintaining length and functional position of limb
4. Maintaining joint space (separating capital and acetabular cartilage)
5. Correction of deformities (stretching contractures)
6. Pain relief
7. Prevent complication like subluxation and dislocations, wandering acetabulum.

21. How much traction do you give?

Ans: Traction is usually decided on age of patient and 0.45 kg/ year of traction is given:
- Give bilateral traction as:
  – Unilateral may increase abduction deformity
  – Pelvic tilt may also increase
- Slight abduction is advisable as:
  – Compensates real shortening
  – Usually a tendency towards adduction is seen in convalescence.

22. What is the role of surgery in TB?

Ans: Surgery is indicated either to obtain tissue for diagnosis and/or do a formal debridement in clinically non-

responsive TB. Other type of surgery is done to manage the deformities and provide an optimized outcome:

1. Painless, stable, immobile (fixed) joint: Hip arthrodesis
2. Corrective osteotomy
3. Painless, unstable, mobile joint: Modified girdlestone arthroplasty
4. Painless, stable, mobile: Total hip replacement

23. What are the indications for surgery?

Ans: The following conditions needs to be managed by surgery in TB hip:

1. Clinically non-responsive TB hip: Excision of focus.
2. Failure to obtain acceptable outcome (unacceptable deformity) after completion of conservative treatment.
3. Painful healed disease due to secondary osteoarthritis.

24. What do you mean by excision of focus?

Ans: Removal of all diseased tissue from the joint which is often the synovium and any obvious bony focus/lesion. The material is thoroughly curetted out and sent for histopathology and bacteriological diagnosis (reconfirm diagnosis). Limb is immobilized in acceptable position following procedure. Surgery should be done in proper ATT cover (6 weak – 2 months pre-surgical cover).

25. What are the complications of this surgery?

Ans: One or more of the following:

1. Fulminant progression of disease
2. Osteonecrosis of femoral head
3. Pathological fracture of femoral neck
4. Slippage of capital femoral epiphysis
5. Chondrolysis
6. Pathological dislocation of hip.

26. What will you do in this patient?

Ans: As the patient is responding to conservative treatment, I will continue the same till healing then depending on the outcome I will choose a procedure.

27. What do you expect in this patient?

Ans: Patient in stage III of disease is unlikely to have a well formed painless joint. I expect a painful healed joint with features of secondary osteoarthritis at the end of treatment.

28. What will you do then?

Ans: I will give patient the options (see above) and explain in detail the functional limitations and merits of each. Also I will assess the functional demands of patient.

29. What does the patient want?

Ans: Patient belongs to poor laborer family and principally requires joint for doing hard work. I would prefer doing a hip arthrodesis for him.

30. What are the indications of arthrodesis in TB hip?

Ans: Four main indications:

1. Young active adult patient doing hard work and putting lot of stress on joint
2. Failure to arrest disease after 1 year of supervised treatment
3. Relapse and recurrence of pain and deformity after conservative treatment (now with the advent of good total hips and chemotherapy people prefer doing THR)
4. Destructive disease viz. formation of sequestra in head of femur or acetabulum (again people may prefer THR)

In general a young adult patient with no life limiting (focus on remaining survival) or activity limiting (e.g. rheumatoid arthritis) unilateral hip disease and is not a candidate for osteotomy/mold arthroplasty. Also one who desires a standing work rather than sitting.

31. What are the types of hip arthrodesis?

Ans: *Intraarticular:*

- Central dislocation and internal compression arthrodesis of charnley.
- Watson-Jones transarticular nail arthrodesis

- Intramedullary arthrodesis of Onji
- Cobra-plate arthrodesis
  Extraarticular:
- Ilio-femoral arthrodesis of Albee
- Ischio-Femoral arthrodesis of Brittain
  Pararticular (usually done to augment intraarticular procedure):
- Davis muscle-pedicle arthrodesis.

32. Which one will you do and why?

Ans: I will do intraarticular arthrodesis using cobra plate for the following reasons:
   1. Joint debridement can be done simultaneously.
   2. Large raw surfaces can be carved out to enhance chances of union.
   3. More secure fixation and one directly addresses the diseased site without changing the anatomy elsewhere.
   4. In destroyed joints space can be filled with bone graft with good approximation in intraarticular methods.

33. What is the role of extraarticular arthrodesis?

Ans: These were practiced on the premises that opening up diseased joint will flare up the infection and there was lack of availability of good chemotherapy. However, these procedures destroy the anatomy of hip joint making any future procedure difficult.

34. In what position do you fix joint?

Ans: Flexion of 30°: As a general rule 1° per year of age above 10 years till a maximum of 30° above 25 years of age (rationale is that ongoing compensation develops in children with flexible spine and in adults up to 30° of flexion can be hidden by lumbar lordosis). Flexion provides necessary ground clearance and lets one hide fixed joint while sitting.

   Adduction/abduction: Previously favoured abduction no longer holds true as it leads to later development of frontal plane knee deformity and also gait is better with hip fused in adduction. Prefer either neutral or 5° of adduction.

Rotation: No objective data but 0-15° external rotation preferred.

35. What is Brittain's method of extraarticular arthrodesis?

Ans: Consists of first doing a sub-trochanteric osteotomy followed by incising ischium just below the acetabulum *through* osteotomy. Then a massive tibial graft is pushed into the defect through osteotomy. Alternatively, in a healed case (ankylosed) with adduction and flexion deformity a subtrochanteric McMurray's osteotomy can be done.

36. What are the contraindications of arthrodesis?

Ans: Should not be done in:
1. Ongoing uncontrolled active infection: Wait at least one year after infection has healed.
2. Opposite hip, ipsilateral knee already arthrodesed.
3. Severe degenerative changes in lumbar spine, opposite hip, ipsilateral knee.

37. When can you do THR in TB hip?

Ans: Classsically the concept was to do THR after 10 years (every examiner must be acquainted to this – safest option). Ambitiously done THR even after one year of healed infection under ATT cover gave comparable results (this appears to be a safer choice). Now with the advent of chemotherapy, people have attempted THR after 6 weeks of ATT cover under the premises that fixation for spinal TB with ongoing infection has got fair results and change to chronic osteomyelitis and implant failure *per se* due to infection uncommonly occurs (most ambitious choice if you speak may be unacceptable to many!).

(*Howsoever difficult this question and answer seems due to uncertainity – basic principle is that the disease must show good response to ATT and thorough excision of focus must be done else joint will come out with pus!*)

38. What is girdlestone arthroplasty and who described it?

Ans: Girdlestone arthroplasty in essence comprised of extensive debridement of septic joint and surrounding soft tissue

with free drainage creating a 'type' of Excisional arthroplasty. Excisional arthroplasty *per se* was first described by A. White (1849). In extensive review of literature various interesting facts are revealed and there is quite a great misconception for the procedure. Gathorne Girdlestone described his operation in 1923 (not the other oft quoted dates) as a modification of Robert Jone's operation (1921) done for ankylosis of hip joint whereby greater trochanter with its attached muscles used to be attached to resected end of neck to produce pseudoarthrosis. Since then there are as many modifications as there are descriptions.

39. What do you do in girdlestone arthroplasty?

Ans: The original description as sent by girdlestone in a telegram to Sir Robert Jones on 15th July 1926 is described below:

- Transverse incision ≈ 5 inches long centered 1 inch above trochanter is used to expose deep structures.
- Make two transverse cuts to remove all involved gluteal muscles, trochanter and 3-4 inch wide and one inch deep bone is removed from depths (this includes superolateral acetabulum).
- Curette out all carious bone and decide whether to leave bone for ankylosis or remove further to create pseudoarthrosis! (*Here it appears that this procedure was not to create pseudoarthrosis only rather a method to do joint debridement (saucerisation for drainage) in long standing infections of hip with outcome as either ankylosis or pseudoarthrosis*)
- Suture the flaps to periosteum in depths to prevent proud granulation tissue to appear
- Loosely pack the wound with Vaseline gauge.

40. What is the difference between originally described girdlestone arthroplasty and its modification?

Ans: The most popular modifications are firstly that of Taylor (1950) using Smith-Petersen approach. Additionally,

trimming of acetabular rim was done to provide opposing surfaces, pain relief, deformity correction and movements. Nelson (1971) described soft tissue interposition to achieve pseudoarthrosis. The other is that of Grauer et al (1989) who described four different levels of proximal femoral resections and one by Nagi (1997) who did a subcapital osteotomy and sutured anterior capsule. He reposed the neck back into acetabulum. Basically, now the concept is to retain adequate amount of bone and no acetabular surgery or extensive muscle debridement, using posterior approach.

41. **How will you manage a patient of girdlestone arthroplasty in post-operative period?**

Ans: Again very confusing. The principle is to create stout pseudoarthrosis by fibrous tissue and avoid any lateral impingement. Nagi recommends 6 weeks of post-operative traction. Tuli and Mukherjee (1981) recommend 6-8 weeks skeletal traction and further 6-8 weeks skin traction (total 3 months) followed by walking in caliper for 1 year. It is advisable to put patient on skeletal traction in Thomas splint with Pearson's attachment for first 6 weeks (important to prevent external rotation) in 30-50° abduction with radiographic demonstration of distraction at operative site. After first week of surgery patient should be encouraged to mobilize hip and knee. This will be followed by skin traction for further 6 weeks and then mobilization with bucket top caliper for at least 6 months.

42. **What can you do to address the hip instability arising after this osteotomy?**

Ans: Instability and shortening arising out of resection arthroplasty can be partially addressed by pelvic support osteotomy of the Milch and Batchelor type.

43. **What is Milch-Batchelor osteotomy?**

Ans: It is a resection angulation osteotomy (RAO/PSO) described as a two stage procedure by Batchelor and Carry (1943, London)

1. Release of pelvis and restoration of femoral mobility by resecting femoral head and neck
2. Reestablishment of stability by means of PSO (Schanz). The two stages were combined into one by Milch and Gruca (New York) as traction after stage 1 and immobilization after stage 2 wasted a lot of precious time for mobilization of joint. The essence of osteotomy is 'post-osteotomy' angle that should place proximal femur congruent to lateral pelvic wall (mean lateral pelvic wall tilt = 205-210°) else the aim would be defeated. Classically iliofemoral approach was used sacrificing the nerve to tensor fascia lata. The angulation osteotomy is done at the level of ischial tuberosity. Distal fragment was abducted (virtual lengthening to compensate true shortening) and internally rotated else spontaneous external rotation would again destabilize the pelvic support.

44. What other types of PSO you know of?

Ans: Schanz, Ganz, Lorenz (bifurcation osteotomy), McMurray's etc.

45. What is the role of PSO?

Ans:
1. Surgically shifts the shaft of femur near the center of gravity of the body so that the wt. bearing axis is more along the axis of femur
2. Supports pelvis by creating medial fulcrum
3. Improves adductor function by causing valgus
4. Abduction of distal fragment causes of gain of length.

46. What is Phemister triad?

Ans: Classically described for tuberculosis of hip consists of:
1. Juxtaarticular osteoporosis
2. Peripherally located osseous lesion
3. Gradual narrowing of joint space.

47. What triangles you know in relation to hip joint?

Ans: Learn the following:
1. Babcock's triangle

2. Ward's triangle: Between primary tensile, primary compressive trabeculae and calcar portion of neck – relevant in osteoporosis and fixation of hip fractures
3. Fairbank's triangle: Coxa vara
4. Bryant's triangle
5. Scarpa's triangle (femoral triangle)
6. Abductor triangle: formed between gluteus medius, ilium, neck of femur (displays abductor mechanism).

48. What is the role of manipulation under anesthesia?

Ans: Manipulation under anesthesia is indicated in healing disease with less severe deformities to:

1. Attempt gaining mobility of hip while on treatment when articular cartilage is supposedly 'preserved'
2. Provide a functional position (correcting the deformity) to hip lest it goes in fibrous ankylosis when cartilage is irreparably damaged and functions cannot be regained.

CASE II:
NON-UNION FRACTURE NECK OF FEMUR
('THE UNSOLVED FRACTURE'- SPEED)

Diagnosis

The patient is a 52 year old male with 7 months old non-union fracture of femoral neck following trauma treated conservatively. There is 30° fixed flexion deformity with 20° external rotation deformity and 20° adduction deformity and true supratrochanteric shortening of 3 cm. The patient is unable to do his routine activities.

1. What makes you think this is a case of old fracture neck of femur?

Ans:
• Middle aged (or elderly) patient with history of definite trauma and inability to bear following injury
   – Tenderness over midinguinal point

- Desault's sign positive
- Telescopy test positive, trendelenburg's positive.
- Shortening and external rotation.

2. Why is it not non-union fracture intertrochanteric?

Ans: There should be irregularity over trochanter with broadening and thickening. Tenderness should be at trochanteric region rather than mid-inguinal point.

3. Why is it not old anterior dislocation of hip considering the deformity?

Ans: There is often extension deformity with lengthening in low types of dislocation. Moreover, head is not palpable in the classical sites.

4. Why do you think that head is located in this patient?

Ans: Femoral pulses are bilaterally comparable (Narath's sign). (*Also see examination for palpable head in dislocated hip*)

5. What will be your differential diagnosis?

Ans: I will put forward the following differentials (*It is always better to speak your answer and diagnosis itself this way – as I presented a very hypothetical and classic case for understanding which is hardly the case in exams, although I cannot predict or present here all pathological presentations!*):

1. Old ununited Fracture intertrochanteric right femur
2. Old Fracture head of right femur: Telescopy often absent or minimal
3. Malunited fracture acetabulum posterior wall/superior wall/both: very difficult to differentiate but often internal rotation deformity is observed as the hip is unstable and dislocates/subluxates posteriorly – not always true, so it is the closest but rarest differential to be given in exams (DNB candidates beware)
4. Old treated TB hip with bony changes (wandering acetabulum/mortar-pestle type): All said and done traumatic event with typical follow-up should be absent.

6. What are the causes of non-union in fracture neck femur?

Ans: Non-union here is predominantly due to combination of mechanical (points 1-4 below) and biological (points 5-10) disruptions:

1. Morphologic features: High fracture angle 60°-90° ($\uparrow$ shear angle)
2. Displaced fracture: Garden's III/IV
3. Fracture comminution – Posterior comminution (affects adequacy of reduction, angulation and stability of fixation)
4. Inadequate reduction and stability of fixation
5. Poor bone quality (osteoporosis)
6. Injury to vascularity: Direct and tamponade effect (Deyerle): Remember head of femur is already a 'hypovolemic bone' (PET studies) even small disturbances put vascularity at risk.
7. Absence of cambium layer in periosteum
8. Chondrogenic factors in synovial fluid that inhibit callus formation and consolidation
9. Lack of hematoma formation: Synovial fluid prevent hematoma formation
10. Washing away and dilution of osteogenic factors. Patient's age (It only decides treatment!), gender, Osteonecrosis has no influence with non-union # neck femur.

7. Which type of non-union do you see here?

Ans: Atrophic type.

8. How does duration of non-union affect planning?

Ans: Increased duration of fracture is counterproductive in the following ways:

1. Resorption at fracture ends (Resorption begins as early as 3 weeks)
2. Contractures prevent adequate lengthening and reduction
3. Acetabular cartilage damage

9. How do you radiographically assess the fracture?

Ans: Poor prognostic factors:

1. ↑ # angulation
2. Osteopenia
3. Bone loss
4. Osteonecrosis
5. Calcar comminution
6. Varus angulation
   Lateral projection:
1. Flexion/extension
2. Posterior comminution
   MRI/Bone scan to look for viability of head.

10. How do you radiographically assess osteoporosis?

Ans: Singh and Maini index on AP radiographic evaluation of trabeculae:

Grade 6 (normal): All trabeculae present

Grade 5: Loss of trochanteric and secondary tensile, attenuated secondary compressive

Grade 4: Loss of secondary compressive, attenuation of primary tensile

Grade 3 (definite osteopenia): Break in primary tensile

Grade 2: Marked loss of primary tensile

Grade 1: Only primary compressive seen but they are also reduced.

11. When do you call a fracture neck of femur to be a Non-Union?

Ans: 3 months following fracture.

12. What are the various treatment options available?

Ans: The followings have been successfully practiced (*And should be individualistically given to the patient!)*:

1. ORIF with cancellous bone grafting
2. ORIF with fibular grafting
3. ORIF with vascularized bone grafting
   a. Free vascularized fibula
   b. Muscle pedicle bone grafting
4. Neck reconstruction

5. Osteotomy
6. Arthrodesis
7. Arthroplasty
8. Girdlestone type of resection arthroplasty.

13. How do you plan surgery?
Ans: Clinical assessment:
1. Age of patient
2. Presence of Osteonecrosis
3. Prior hip symptoms: Osteoarthritis etc.
4. Co morbidities: Smoking etc.
5. Duration from injury
6. Fracture variables:
   a. Site of fracture
   b. Fracture configuration.

14. How would you classify non-union fracture neck femur?
Ans: Sandhu et al (Predictive classification):
- Fracture surfaces
  – Irregular
  – Smooth
- Size of proximal fragment
  – 2.5 cm or more
- Gap b/n fragments
  – Upto 1 cm
  – More than 1 cm
  – More than 2.5 cm
  Three groups (decided on a combination of above)
- Group three – worst results.

15. What are the guidelines?
Ans: First look for Osteonecrosis:
1. *Osteonecrosis + Nonunion:*
   a. <50 years: Pedicle grafting vs. arthrodesis vs. osteotomy (McMurray's type)
   b. >50 years: Arthroplasty
2. *Nonunion + anatomy preserved (NO Osteonecrosis):*
   a. <65 years: Osteosynthesis

      i. ORIF with vascularized grafting

      ii. ORIF with fibular grafting

   b. >65 years: arthroplasty

3. Non-union with destroyed anatomy (e.g. neck resorption) (NO Osteonecrosis)

   a. <65 years: Osteotomy (Pauwel's type) (in a subset of younger people <40 years – Neck reconstruction also is a suitable option)

   b. >65 years: Arthroplasty (*This option has become so favourable that for this situation nearly all patients >40 years are offered arthroplasty!)*

16. What are the various muscle pedicle grafts described?

Ans: There are various muscle based grafting techniques described:

1. *Muscle pedicle bone grafting*

   a. Quadratus femoris based (Judet, Meyers et al)

   b. Gluteus medius based (Hibbs)

   c. Anterior trochanteric bone grafting (Das and Balasubramaniam – modified Hibbs)

   d. Sartorius based (Li et al)

   e. Tensor fascia lata based (Bakshi)

   f. Gluteus maximus based (Onosun et al)

2. *Muscle pedicle "grafting"*

   a. Gluteus medius based (Frankel and Derian)

   b. Vastus lateralis based (Stuck and Hinchey)

3. *Muscle pedicle periosteal (Myoperiosteal) graft*

   a. Quadratus femoris based periosteal grafting

4. *Combined!!*

   a. Often used in eastern Asian countries

      i. Combined Sartorius + deep circumflex femoral artery based iliac crest

      ii. Quadratus based + osteoperiosteal anterior grafting

5. *Neck reconstruction*

   a. Devising a trough like rectangular box in the region of resorbed neck and filling with bone graft

   b. Using cage and autologous cancellous bone grafting

17. What is the advantage of muscle pedicle bone grafting?

Ans: Following are the advantages of using these grafts:
- There is no substitute for original biological joint
- Always give a fair chance to save a salvageable joint
- Vascularized bone graft may additionally take care of Osteonecrosis
- Pedicle grafts are less cumbersome than free grafts with comparable results.

18. What are the principles of this grafting technique?

Ans: *Principles:*
- Vascularized grafts shown to increase the vascularity of devascularized head (Stuck and Hinchey, Frankel and Derian)
- The hypovolemic head is converted into normovolemic head
- Often spontaneous revascularization after ORIF/CRIF stops at antero-superior region leading to segmental collapse which is taken care of.

19. What is the role of osteotomy in treating Non-union fracture neck of femur?

Ans: Osteotomy alters both mechanical and biological environment around non-union site which may enhance healing or at least provide relief to the patient (McMurray's concept):
1. Altering mechanics – Medial shift of line of weight bearing
2. Correction of deformity: Rotational deformity can be corrected, shortening compensated by apparent lengthening (Pauwel's)
3. Realignment of limb during movement
4. Relaxation of joint capsule
5. Increased vascularity
6. Psoas relaxation providing pain relief by a mechanism similar to hanging hip of Voss
7. Improved congruity of joint surfaces (only if deformed due to Osteonecrosis)
8. Improved leverage and stability

9. Relief of pressure by muscles
10. Re-distribution of tensile forces at # line to compressive forces → *Arm chair effect*.

20. What is arm chair effect?

Ans: As above (*I understand that this does not explain what one is curious about*). In McMurray's osteotomy the distal fragment is placed directly under the head so weight is directly transmitted from head to shaft bypassing neck so tensile shearing forces are converted to compressive forces. Now just imagine and compare yourself getting up from a chair without arms and a chair with arms. In the first instance forces are concentrated around knee in a tensile manner (unless you support them with your hand) whereas in the second instance you will get off the chair pushing at the arms which are more or less situated at knee level or sometimes even in front, dissipating in effect the shearing stresses across knee. This is the effect of an arm-chair (dissipating the tensile forces at the lateral border of fracture) which was recommended originally for osteoarthritis of knee and hip.

21. What are the various osteotomies described around hip for treating Non-union?

Ans: Classically two landmark osteotomies with various modifications are cited:

1. Lineal osteotomy: Medial displacement osteotomy first described by Haas revised by McMurray and Leadbetter
2. Angulation osteotomy described by Schanz with modification by Pauwel's.

22. What is McMurray's osteotomy?

Ans: It is aptly described as medial displacement oblique intertrochanteric pelvic support osteotomy.

23. What are the principles of McMurray's osteotomy?

Ans: Conditions for success:

1. Upper end of shaft must be just below the edge of acetabulum
2. There must be union between portions of divided femur.

24. For what condition did he describe this osteotomy and what are the prerequisites?

Ans: Osteoarthritis of hip joint with pain, stiffness and deformity. There should be a minimum of 70° flexion at hip joint (90° ↓ general anesthesia). It should not be done in coxa magna, loss of sphericity of head both in AP and lateral projections, dysplastic acetabulum, subluxation of head, inflammatory disease, and ankylosing spondylitis.

25. How do you do this osteotomy and how to fix it?

Ans: The line of osteotomy goes from the base of greater trochanter obliquely up (10-15°) to exit just above lesser trochanter. After doing medial displacement of distal fragment fixation was done by Wainwright-Hammond spline (plate). By doing adduction it used to tilt the proximal fragment into valgus making the fracture line horizontal.

26. What are the disadvantages of this osteotomy?

Ans: Causes shortening, lurching gait, frequent nonunion at osteotomy site, difficult future THR (so it is recommended not to displace by >50%), predisposes to genu valgum of ipsilateral knee.

27. What is Pauwel's osteotomy?

Ans: Pauwel's repositioning valgus intertrochanteric osteotomy re-places the pseudoarthrosis site to remove the shear forces. The osteotomy is aimed to enhance fracture healing and other benefits like:
   1. Equalizing limb lengths (virtually)
   2. Lateralization – reducing tendency to genu valgum
   3. Early mobilization by fixing osteotomy.

   Planning: Body forces subtend an angle of 16° at hip joint. The anatomical axis is at an angle of 8-10° to body forces, so the pseudoarthrosis site is subjected to forces at around 25°. Subtract this from the pseudoarthrosis angle (vide Pauwel's classification). This gives the wedge angle to be resected at osteotomy site. The same principle applies for

Murray's modified osteotomy classically described for pseudoarthrosis of femur neck.

28. Which test would you use to decide instability at hip joint?

Ans: Telescopy test: Significant telescopy (> 1cm trochanteric excursion in one direction) is a good indicator of unstable hip, whilst absence of the same does not substantiate stability. Telescopy is seen due to absorbed neck, comminution at fracture site, tearing of capsule in high impact injuries.

29. Which other test can you use?

Ans: Due to limitations of telescopic test (*See Q 38&39 on Page 27*) active SLRT (Stinchfield test) can be performed. This test may however be fallacious (false negative) in impacted fragments, capsular contracture, and leverage of distal fragment on acetabular margin. It may be absent (false positive) in a frail patient and cannot be done in hemiplegia or paraplegic patient.

30. What are various closed Reduction maneuvers for fracture neck of femur?

Ans: *Maneuvers in extension:*
   1. Whitman
   2. Deyerle
   3. Swiontkowsi.
      Maneuvers in flexion:
   1. Leadbetter: flexion → internal rotation → circumduction to abduction and extension; check by resting heel on palm, if it rests without externally rotation then it is a secure reduction.
   2. Flynn
   3. Smith-Peterson method ("gentle Leadbetter" method)

31. How do you make an assessment of alignment?

Ans: Assessment of alignment can be done by any one or a combination of following:

1. Garden's index: AP – 160° and lateral – 180°, radiographs required 155-180° acceptable
2. Lowell's S-curves: image intensification
3. McElvenny: 'Hat on hook' position
4. Lindequist and Tornkvist criteria of good reduction: ≤ 2mm displacement, AP Garden angulation of 160-175° and lateral angulation of ≤10°.

32. What is the shape of fracture line in fracture neck of femur?
Ans: Spiral.

33. How do you classify fracture neck of femur?
Ans:
1. Garden's classification (complete/incomplete; degree of displacement – Garden's index – trabecular disposition in AP (160°) and lateral (180°) projections):
   i. Incomplete, Valgus impacted fracture with trabecular displacement (↑ Garden's index in AP, may be normal in lateral)
   ii. Complete, undisplaced ± impaction
   iii. Complete, displaced (partial displacement <50%)
   iv. Complete, displaced >50% and dissociation between proximal and distal fragments so that proximal one realigns with acetabular trabeculae.
   Eliasson et al, according to displacement in femoral neck fractures divided fractures into undisplaced (= Garden 1 & 2) and displaced (= Garden 3 & 4) types.
2. Linton's classification:
   i. Fracture in adduction (varus displacement/angulation: Garden's index ↓)
   ii. Fracture in abduction (valgus displacement/valgus angulation)
   iii. Intermediate type.
3. Pauwel's (higher the shear angle more will be the stresses and hence unstable fracture):
   i. Angle of # line <30°
   ii. Angle of # line 30° to 50°
   iii. Angle of fracture line >50° and ≤ 70°

4. Anatomical: subcapital, transcervical, basicervical
5. AO
6. Current classification (Caviglia; Osorio and Commando): 5 types depending upon # completeness, contact, angulation and comminution.
7. Stress fracture neck of femur (Fulkerson and Snowdy)
    i. Tension stress fracture → superolateral aspect of neck, ↑ risk of displacement
    ii. Compression stress fracture → inferomedial aspect, ↓ risk of displacement
    iii. Completely displaced fracture neck of femur displaced.
8. Classification in children (Delbet and Collona):
    i. Transepiphyseal: Involves physis with/without dislocation of femoral head from acetabulum. Flexion, abduction and external rotation deformity.
    ii. Transcervical #: (most common) Most are displaced and unstable. Osteonecrosis proportional to degree of displacement
    iii. Cervicotrochanteric: 2nd commonest, similar to basicervical
    iv. Intertrochanteric: Good fracture.

34. What is the blood supply of femoral head?
Ans: The blood supply to *femoral head* is derived from three primary sources (as described by Crock), the metaphyseal system, retinacular system and the foveolar system as follows:
1. **Extracapsular arterial ring [ECA]**: This is the chief system giving rise to both intramedullary and extramedullary arterial systems. The ECA gives less prominent metaphyseal branches to intertrochanteric region which also supply the head through neck (intramedullary metaphyseal system). It is located at the base of femoral neck and is formed –
   • Posteriorly by branch of medial circumflex femoral artery
   • Anteriorly by branch of lateral circumflex femoral artery more often a branch of profunda femoris artery (main branch of femoral artery)

- *Ascending cervical branches* of ECA (aka Epiphyseal arteries of Trueta or retinacular arteries) arise from ECA (more prominent system) and ascend up the neck partly also supplying the neck in due course –
  i. Divided into anterior, posterior, medial, and lateral groups
  ii. Anteriorly these vessels penetrate the capsule at intertrochanteric line while posteriorly they pass underneath the orbicularis fibers of the capsule
  iii. Lateral group (*lateral ascending cervical vessels)* is the most important group carrying major portion of blood supply to head and neck of femur

2. **Sub-synovial intraarticular arterial ring of Chung** (*Circulus articuli vasculosus of Hunter)* is formed from lateral ascending cervical vessels:
   - Located at the margins of articular cartilage on surface of neck of femur
   - It is either a complete or incomplete ring
   - Provides epiphyseal vessels (that penetrate the head just outside the articular cartilage to supply major portion of head)

3. **Artery of ligamentum teres**
   - Branch of obturator artery (more often) or medial circumflex femoral artery
   - Variable supply in adults
   - Supply head around the region of fovea

   The metaphyseal *femoral neck* is supplied by a cruciate shaped anastomosis between:
   - Branches from ascending cervical arteries
   - Branches from sub-synovial intraarticular arterial ring
   - Intramedullary branches of superior nutrient artery system
   - Metaphyseal vessels from intertrochanteric region

   This rich anastomosis makes the neck a very unlikely site for avascular necrosis.

35. How do you look for protrusio acetabuli and what are the various causes of the same?

Ans: Distance between medial wall of acetabulum and the pelvic brim (iliopectineal line) Sotelo-Garza and Charnley (1978):

- Grade I: 1-5 mm (mild)
- Grade II: 6-15 mm (moderate)
- Grade III: >15 mm (severe).

Causes

1. Familial/idiopathic (Otto pelvis)
2. Rheumatoid arthritis and JCA
3. Osteoporosis
4. Osteomalacia and Rickets
5. Marfans syndrome (45% have protrusio, 50% of these are unilateral and 90% associated with a scoliosis)
6. Pagets disease
7. Ankylosing Spondylitis
8. Osteoarthritis (occasionally)
9. Acetabular fractures
10. Osteogenesis Imperfecta.

CASE III:
OSTEONECROSIS OF FEMORAL HEAD

Diagnosis

The patient is a 32 year old male with Rt. hip flexion deformity of 30°, adduction deformity of 25°, internal/external rotation deformity of 20° with true supratrochanteric shortening of 1.5 cms. I would like to give a differential diagnosis of: 1. Osteonecrosis, 2,3,4,5...

(*It is important to understand that Osteonecrosis is by itself a differential diagnosis for various disorders nearly all of which have the end result of producing secondary osteoarthritis, however the development of head deformity is an early finding with Osteonecrosis*

with characteristic progression. The deformities are not characteristic and with development of secondary osteoarthritis the movements are also lost early! So as a rule always try to give the diagnosis as a differential diagnosis and be safe)

1. What is your differential diagnosis?

Ans: Typical differential diagnoses include:

1. Tuberculosis of hip: (Old cases of Osteonecrosis only with restriction of most movements)
2. Transient osteoporosis of hip in females.
3. Primary osteoarthritis of hip (once the osteoarthritic changes develop in Osteonecrosis): deformity of head and sectoral signs absent.
4. Old Perthes disease, femoral head deformity due to epiphyseal/other dysplasia with development of secondary osteoarthritis.
5. Old Femoral head fracture with secondary osteoarthritis.
6. Monoarticular rheumatoid is as such rare and if mentioned then should be last as it is a diagnosis of exclusion!

2. Why do you keep Osteonecrosis as your first differential?

Ans: History:

1. Single joint involvement (ankylosing spondylitis), Insidious onset, slow progression
2. Characteristic course
3. No constitutional symptoms
4. Deformities do not match with staging of TB hip (viz. even in stage III the movements are fairly preserved)
5. No history of trauma.

3. What are the diagnostic criteria for osteonecrosis of hip?

Ans: For non-traumatic osteonecrosis of hip (Japanese investigating committee, 1990):

Major criteria
(*There should not be any joint-space narrowing or acetabular changes for 1-3 to be positive*):

1. Radiological (depression of femoral head, demarcating sclerosis in the femoral head, crescent sign)
2. Bone scan (cold-in-hot)
3. MRI (low intensity band on T1-weighted image)
4. Histology (trabecular and marrow necrosis).

Minor:
1. Radiological (depression of femoral head with joint-space narrowing, cystic radiolucency/mottled necrosis, flattening of the superior portion of femoral head)
2. Bone scan (cold 'or' hot)
3. MRI (homogeneous/inhomogeneous low intensity without a band pattern)
4. Symptom (hip pain with weight bearing)
5. History (corticosteroid or alcohol usage)
   Definitive osteonecrosis: ≥ 2 positive major criteria
   Probable osteonecrosis: One positive major criteria or ≥ 4 positive minor criteria at least one radiological.

4. What are the causes of Osteonecrosis of hip?

Ans: *Idiopathic* form (Chandler's disease) is the most common (? COL2A1 gene mutation/P-glycoprotein or alcohol metabolizing enzyme polymorphism etc). The other causes are:

1. *Trauma:*
   a. Fracture neck of femur
   b. Dislocation (posterior >> anterior but one to one case wise incidence is more common in anterior dislocation)
2. *Corticosteroids:* 'Threshold' cumulative dose of 2000mg for prednisolone within 2-3 months, 4.6 fold $\uparrow$ in incidence with every additional 10mg intake.
3. *Alcohol:* consumption of >400ml/week (this is equivalent absolute alcohol content!) – 9.8 fold $\uparrow$ in incidence (*This value was notably not rounded off – remember the value of "g"*) {DRINK YEARS = weekly alcohol consumption X years)
4. Coagulation disorders (hyper-coagulability)
5. Hyperlipidemia
6. *Dysbarism:* Does not occur <17 psi, not believed to be a risk factor <30 months

7. SLE and connective tissue disorders
8. Organ transplantation (metabolic changes and chemotherapy including steroids)
9. Liver dysfunction
10. Radiation
11. *Pregnancy:* Small body frame & relatively large weight gain.
12. Smoking (>20 pack years, pack years = packs per day X years)
13. Hyperuricemia
14. Myeloproliferative disorders
15. HIV infection.

5. What are various pathogenic mechanisms?

Ans: Direct cellular toxicity, extraosseous arterial, extraosseous venous, intraosseous extravascular, intraosseous intravascular, multifactorial.

6. What is the difference between two most common causes of osteonecrosis?

Ans: In the idiopathic form the lesion is characteristically located in the antero-superior region. The involvement could be bilateral in more than half of the cases (some texts may say ? 75%). Post-traumatic cases have often total involvement with isolated joint involvement.

7. How will you confirm your diagnosis?

Ans: I will get the radiographs of pelvis with both hips, involved hip in AP and lateral projections.

8. How much time does it take to be evident on radiographs?

Ans: Perhaps the earliest changes appear by 2 months which are easily discernable by 6 months.

9. How do you stage osteonecrosis?

Ans: Ficat and Arlet classification (4 stage), modified Ficat-Arlet (stage '0', subdivision of stage II), university of Pennsylvania classification (see table).

Ficat-Arlet (modified) classification

Stage	Symptoms and signs	X ray	Bone scan
0	None	—	? $\downarrow$ uptake
I	None/Mild	—	$\downarrow$ uptake
II	Mild	Altered density A. Sclerosis/cysts/ normal joint line, normal head contour B. **Crescent sign** (subchondral #), flattening	$\uparrow$ uptake
III	Mild to moderate	Loss of sphericity, collapse	$\uparrow$ uptake
IV	Moderate to severe	$\downarrow$ joint space, acetabular changes	$\uparrow$ uptake

Original Ficat's classification did not include sub-classification of stage II.

University of Pennsylvania Classification (Steinberg et al)

Stage	0	I	II	III	IV	V	VI
Findings	All present technique normal/ non-diagnostic Histology +ve	X-ray, CT normal	X-ray– mottled and sclerotic cysts, porosis	X- ray- **crescent sign**	X- ray– flatten- ing of anterior surface	IV + narrow- ing of joint space/ aceta- bular changes	Complete joint destruc- tion
Techniques	—	X-ray, CT, Scinti- graph, MRI	X-ray, CT, Scinti- graph, MRI	X-ray, CT only Quanti- tate – X-ray and MRI	X-ray, CT only Quanti- tate – X-ray only	X-ray only	X-ray only

Contd...

		Quanti-tate-MRI	Quanti-tate –MRI and X-ray		
Sub-classi-fication	NO	QUANTITATION (Stage II-V)			NO
		% area involved	Length of crescent	% surface collapse	Location
		A - <15% (minimal)	A <15%	and dome depression	Medial
		B – 15-30% (moderate)	B 15-30%	A <15%, <2 mm	Central
		C - >30% (severe)	C >30%	B = 15-30%, 2-4 mm	Lateral
				C >30%, >4mm	

There are numerous other classification that are regionally followed, association research circulation osseous, Japanese orthopaedic association, Marcus et al, Sugioka, Kerboul et al, Smith et al etc.

10. What are the tests for hemodynamic function?

Ans: These tests are for research purpose and not available for population at large, however they are positive in stage '0' Ficat-Arlet/Pennsylvania classification.

1. Intramedullary pressure (at rest = 10-20mm Hg, rapid saline injection $\rightarrow$ rises by about 15 mm Hg): In osteonecrosis both increase by 3-4 times of normal

2. Venography.

11. Do you know of any classification based on MRI only?

Ans: Schimuzu (1994) MRI grades for treatment:

In general:

a. The extent (area of coronal femoral head involvement) determined at the outset does not change

b. Lesion <1/4 in extent and medial 1/3 in location (portion of weight bearing surface involved) – grade I, rarely collapse.

c. Lesion up to ½ in extent and $1/3$-$2/3^{rd}$ of weight bear surface collapse in ≈ 30% cases (Grade II)

d. Lesion > ¼ in extent and > $2/3^{rd}$ of weight bearing surface (Grade III) collapse within 3 years in 70% cases.

12. How will you plan treatment based on classification (Steinberg)?

Ans: Grade 0 – Reassure and treat symptomatically keep in close observation (non-weight bearing/ restricted weight bearing has been shown to be of NO benefit, lipid-lowering agents, anticoagulants, iloprost prostacyclin inhibitor), intravenous bisphosphonates, shock wave therapy, hyperbaric oxygen, PEMF are all indeterminate. *In general all lesions in precollapse stage (Ficat IIA and below, Steinberg II and below) have good chances with joint preserving procedures, post collapse stages – the disease is bound to progress, so in any case a surgical procedure is indicated. Only stage '0' at the present time can be observed – but is hardly ever diagnosed as it is preclinical and preradiological!!. So why talk of conservative treatment? Most of idiopathic cases are sequentially bilateral – this treatment is to preserve the opposite hip)*

Grade I – Core decompression, percutaneous drilling (multiple drill holes with 3.2 mm drill bit), muscle pedicle bone grafting

Grade II (precollapse) – Age < 45years; core decompression with/without bone grafting, osteotomy, muscle pedicle bone grafting

Age >45 years; limited hemi-surface replacement/total hip replacement

Grade III (crescent) – Younger patients → bone grafting, limited hemiresurfacing arthroplasty (post-traumatic cases), (?) osteotomy. Older patients → total hip arthroplasty.

(Bipolar and hemiarthroplasty have been shown to be a POOR performer as of now for various potential but unproven reasons, so an option of bipolar arthroplasty has been intentionally dropped here).

Late cases

Steinberg IV-VI / Ficat III/IV – total hip replacement/ osteotomy(?IV)/ arthrodesis/ girdlestone (nowadays will be considered criminal!). In younger patients bone grafting (trap door etc) still remains treatment of choice (stage IV), some cases might also fit into osteotomy.

If one does not want to follow this complicated version, here is the *simpler one*!

70-86% will progress (for non-traumatic one and less for a traumatic etiology but surely spontaneous resolution is remote!) hence there is no role of conservative treatment!!

In general (Ficat classification followed here):
- Core decompression with/without bone grafting → for Ficat IIa or earlier
- Osteotomy → stage II and (?) III
- Trap-door/light-bulb procedures → stage IIb and III (hardly ever done, better keep quite unless asked specifically, you can score better in stronger fields!)
- Arthroplasty/arthrodesis → stage III and IV (avoid arthrodesis in majority – you see, most idiopathic cases are bilateral!)

So here you will find you have maximum choice for stage III Steinberg, but practically speaking this is the most difficult stage to treat.

13. What is core decompression and how you do it?
Ans: Also called 'Forage' this procedure is typically used to decompress the hypertensive head by creating a hole extending till necrotic area. This often gives immediate pain relief. It can be done as an:
- Isolated procedure or
- With adjuvant (like electric current stimulation, BMP, demineralised bone matrix (DBM)), or
- With bone grafting (non-vascularized/ vascularized) *Hungerford Technique:* Using Jewett nail starter (12mm) Ficat (modified) – 10mm window with 8 mm central core made by Michele trephine and two additional channels made by 5-6 mm trephine.

Entry point is just distal to vastus lateralis origin in metaphyseal to prevent stress fracture.

Post-op: single hip → PWB walk on two crutches → 3 point gait

Bilateral cases → PWB on two crutches → 4 point gait.

14. What is the rationale of core decompression?

Ans: Femoral head is likened to Starling's resistor (thin vessels in unyielding bony channels). Femoral hypertension is relieved by drilling the head and neck. The benefits are thought to arise from:

Biological changes:
a. ↓ intra-osseous pressure
b. Revascularization through channel or (fibular intramedullary canal if used)
c. Prevents additional ischemic events.

Mechanical changes:
a. Removal of necrotic bone removing the obstruction to revascularization
b. Subchondral graft supports the cartilage.

15. What are the various methods of core decompression and bone grafting?

Ans: Suggested first by Phemister, now often used:
a. Cancellous (Modified Ficat)
b. Cortical (vascularized fibula, non-vascularized strut graft)
c. Muscle pedicle bone graft: Usually for traumatic osteonecrosis.
d. Osteochondral grafting
   i. Trapdoor procedure: raise a chondral flap, curette out bone, fill with cancellous graft and struts, replace flap
   ii. Light-bulb procedure (Rosenwasser et al): above through a metaphyseal cortical window.

16. How do you insert fibula?

Ans: Harvest fibula (90-100mm), split it into two, mallet through the core with canal portion along the wall (split and flip over the graft). This provides a facilitative pathway for revascularization channels.

17. What are the principles of free vascularised fibular grafting?

Ans: (Urbanik). Harvest cancellous graft from ilium and 13 cm of fibula with vascular pedicle (peroneal artery and branches). Make a core 2 mm wider than fibular girth, insert fibula and make an anastomosis between peroneal stump and lateral circumflex femoral vessels, stabilize fibula with K-wire. Vastus lateralis should be released to prevent kinking and pressure on lateral circumflex femoral vessels. Iliac graft (Autogenous cancellous) is inserted into the necrotic region and around fibula before inserting fibular graft (it can also be obtained from greater trochanter). Odds favour the use of free vascularised fibular grafting strongly over non-vascularised bone grafting.

18. What is the role of osteotomy?

Ans: Goals:

1. ↓ in intramedullary pressure/ venous hypertension (biological)
2. Removal of lesion from weight bearing area (bio-mechanical)
   a. Giving time to heal
   b. Decreasing progression.
3. Restoration of blood supply (biological)
4. Other effects as (*See Q 19 on Page 53*).

19. What are the various osteotomy options?

Ans: Following osteotomy principles are described, exact procedure and choice depends on site of lesion, extent of lesion (ideally should be <200° combined necrotic angle), early stages, surgeon's preference:

1. Merle D' Aubigne type curved varus osteotomy to load the most lateral portion (at least >20° of normal femoral head laterally and <160° combined necrotic angle required). (*note – McMurray type varus displacement osteotomy or Pauwel's type varus angulation osteotomy has also been successfully used by some*).

2. Valgus extension osteotomy (Pauwel's): Moves necrotic portion laterally considerably enlarging the weight bearing surface and loads the capital drop osteophyte (? The osteophyte is well developed only in stage IV)

3. Sugioka's anterior rotational osteotomy: Moves necrotic area anteriorly (up to 90° rotation can be done), done through trochanteric (hence trans-trochanteric, based on vascular pedicle of medial circumflex femoral artery). At least 36° of preserved lateral femoral head should be there with combined necrotic angle <200°. First rotational osteotomy was described by Wagner and Zeiler using a double intertrochanteric osteotomy that could rotate the necrotic fragment by 180° degrees. Posterior rotational osteotomy is also described (Atsumi T.) that has theoretically the advantage of medially shifting the medial circumflex femoral artery and relieving tension with posterior rotation. These are very technically demanding with inconsistent results.

4. Flexion intertrochanteric osteotomy (Schneider): Places posterior healthy portion into weight bearing area.

5. Valgus flexion osteotomy (Scher and Jakim) with Autogenous bone grafting.

   (*If you are confused with Pauwel's osteotomy for was it varus or a valgus angulation osteotomy – then it means you are reading the text well!. Pauwel described angulation osteotomy for osteoarthritis hip! Varus osteotomy (Pauwel's I) and valgus osteotomy (rather Valgus extension osteotomy – Pauwel's II were both described by him. This should take away the confusion – Hurray!!).*

   (*Now a note on which osteotomy to be mentioned in exam – Frankly speaking please read the algorithm for planning treatment first. While answering, only mention 'osteotomy', if further asked mention – 'intertrochanteric osteotomy to unload the necrotic segment' – if still asked then tell that 'I will plan osteotomy according to site of femoral head involvement' and bank upon valgus extension/flexion osteotomies or varus osteotomy; others are difficult and one may not have ever seen – see below*).

(Just a learning note – osteotomy is better suited for post-traumatic osteonecrosis, in these cases although osteonecrosis extends more distally into femoral head but it responds favourably to joint preserving and revascularization procedures and is often non-progressive).

20. How do you plan osteotomy?

Ans: On X-ray and MRI I will evaluate the following:

1. Look at the site of involvement (anterior – then do a flexion osteotomy, antero-superior – valgus flexion osteotomy, posterior – extension osteotomy, posterosuperior – valgus extension osteotomy, superior with >30° (now >20°) of preserved head – curved varus or McMurray's type, >36° preservation – rotational transtrochanteric).

2. Whether it satisfies the criteria and prognostic factors:
   a. Kerboul et al (X-Ray, combined necrotic angle – measured sum of necrotic arc measured in degrees from the center on AP and Lateral views): Small combined necrotic angles are ≤150°, medium angles are between 151° and 200°, and large angles are >200°.
   a. Modified Kerboul (X-Ray and MRI – central coronal and sagittal cuts used to calculate the combined necrotic angle): grade 1 (<200°), grade 2 (200° to 249°), grade 3 (250° to 299°), and grade 4 (≥300°).

   Grade I/small necrotic angles (Kerboul) are ideally suitable for osteotomy.

21. What is Bakshi's procedure?

Ans: Muscle pedicle bone grafting using either quadratus femoris, tensor fascia lata, sartorius, gluteus medius muscle with multiple drilling of the necrotic fragment. It can be done till stage III (Ficat) in younger patients (DR. D.P. Bakshi did it in patients up to 60 years old). Internationally, however, the results have not been very acceptable for stages above IIA.

CASE IV:
PERTHES DISEASE

1. What is Perthes disease?

Ans: It is a self limited disease of hip in children produced by varying degrees of idiopathic osteonecrosis of capital femoral epiphysis.

2. What are the various synonyms?

Ans: LEGG-CALVE-PERTHES disease, pseudocoxalgia (Calve {France}), arthritis deformans juvenilis (Perthes {Germany}), precoxalgia (Soudart), coxa vara capitalis (Levy), coxa plana (Waldenstrom). Legg was from USA. (*What's in a name? – the one you call rose would smell as sweet by any other name*)

3. Why is it more common in boys and what are gender differences for this disease?

Ans: This is more common in males (M:F=4:1), peaks at around 6 years. Anterior anastomosis is often incomplete in boys. Females have earlier onset and prognosis is worse. More common in whites than blacks and Indians possibly due to early establishment of foveolar blood supply in the latter.

4. What is the blood supply of head in children?

Ans: Till 3 years of age metaphyseal and retinacular are the two major sources of blood supply. During growth of head (4-8 years) the metaphyseal vessels are obliterated and retinacular vessels are the only significant source of blood supply which enter as lateral epiphyseal vessels. Lateral epiphyseal vessels are predominantly divided into posterosuperior and posteroinferior, occlusion of former leads to development of osteonecrosis in anterolateral aspect of femoral head (proposed vascular hypothesis for Perthes disease, *Trueta hypothesis*). After 8 years foveolar supply is developed enough and dual channels are

established (i.e. retinacular and foveolar). By 16-18 years growth plate is disappeared and all three groups supply head (i.e. metaphyseal in addition to above).

5. What is Caffey's hypothesis?

Ans: Intraepiphyseal compression of blood supply leads to osteonecrosis.

6. Why do you call it Perthes disease?

Ans: There is:
1. Protracted course and insidious onset of limp followed by pain
2. Limitation of abduction and internal rotation (painless limp is the usual presentation, limited abduction in flexion is the first usual sign)
3. Minimal shortening (true)
   TB hip is the closest differential and cannot be adequately differentiated so always give a differential diagnosis. However flexion deformity is not very prominent in Perthes disease and the patient's clinical findings will not usually fit the stages of TB.

7. What is the other differential diagnosis?

Ans: In the absence of trauma irritable hip is the other close differential. Other differential diagnosis include TB hip, irritable hip, SUFE.

8. How will you differentiate irritable hip from Perthes?

Ans: Irritable hip has less strong male dominance (2:1), and occurs in early age group (mean=3 years). Average duration of symptoms is often less than a week (>6 weeks in Perthes).

9. How will you confirm diagnosis?

Ans: I will get an X-ray done; the following findings support the diagnosis of Perthes:
1. Lateral subluxation: CE angle >20°
2. Small femoral capital epiphysis: Temporary cessation of enchondral ossification, continued articular cartilage growth

3. Flattening and loss of sphericity with subchondral fracture (Caffey's sign, typically occurs in anterolateral region as there are maximum weight bearing stresses and bone resorption)
4. Increased width of epiphysis
5. Gage sign
6. Broadening and shortening of femoral neck
7. Decreased neck shaft angle
8. Convex growth plate
9. Overgrowth of greater trochanter
10. Step like irregularities in growth plate
11. Metaphyseal rarefaction
12. Sagging rope sign
13. Head within head appearance
    Acetabulum is comparatively well preserved! (Important in differentiating TB hip).

10. What are head at risk signs?

Ans: Clinical head at risk signs:
1. Age >8 yrs:
   a. Younger patient has more time for remodeling the defect
   b. Acetabulum in older patient loses its potential to develop
   c. Older patient due to more weight are likely to damage epiphysis further.
2. Female sex:
   a. Girls tend to skeletally more mature than boys of same age so less remodeling time
   b. Age for age, girls tend to be more severely affected.
3. Obesity
4. Limitation of range of motion:
   a. Flexion less than 80° and abduction <30°
5. Increasing adduction contracture
6. Subluxating hips.

   Radiographic head at risk signs (Catterall):
1. Lateral subluxation of femoral head
2. Calcification lateral to epiphysis:
   a. Indicates extruded cartilage
   b. Increased pressure on cartilage by acetabular lip

3. Metaphyseal cysts (*Not in original Catterall*):
   a. Metaphyseal changes represent replacement by adipose tissue then fibrocartilage
   b. Step like deformity in growth plate due to cartilage necrosis.
4. Gage sign:
   a. 'V' shaped defect in lateral part of bony epiphysis, indicates cartilaginous overgrowth.
5. Horizontal growth plate: due to premature physeal closure.
   Scintigraphic head at risk signs:
1. Failure of revascularization of lateral column
2. Decreased activity of physis
3. Anterolateral extrusion of epiphysis
4. Disappearance of previously present lateral column
5. Intense metaphyseal activity.

11. How do you classify Perthes disease?

Ans: The most useful classification is of Herring (Lateral pillar classification for disease severity): femoral head is divided into 3 columns and classified on the basis of lateral pillar height (middle column is the extent of sequestrum on AP view, epiphysis lateral to it is the lateral column and one medial to it is the medial column).

Herring A: Lateral pillar height normal

Herring B: Lateral pillar height reduced by <50%

Herring C: More than 50% reduction in height of lateral pillar.

Catterall groups

Group 1: Anterior head of femur, no sequestrum, no crescent sign, and epiphyseal height maintained.

Group 2: Anterolateral 1/3 to ½ femoral head, sequestrum +, lateral pillar maintained. (Subgroup 2 ½: lateral pillar maintained but there is radiolucency).

Group 3: Anterolateral ¾ femoral head involved, subchondral fracture extends to posterior half, lateral pillar not preserved (reverse group 3 – affects ¾ femoral head but in anteromedial region).

Group 4: Entire femoral head, dense sequestrum, subchondral # extends throughout the head.

Salter and Thompson

A. Extent of subchondral # less than half of femoral head
B. Extent is more than half of femoral head.

12. How do you manage the cases?

Ans: Simple algorithm based on age, range of motion and radiologic staging.

Group I: <6 years old → 50-70% do well. Containment done in rare cases with persistent loss of motion with Herring C/ Catterall III or IV with head at risk signs, Salter and Thompson group 'B'.

Group II: 6-8 years (Bone age more important than chronological age):

A. Symptomatic treatment for Herring group A, Catterall I/II with no head at risk signs, Salter and Thompson group A with symptomatic treatment and range of motion exercises.
B. Herring group B/C with persistent loss of motion, Catterall III/IV with head at risk signs, Salter and Thompson group B → containment indicated.

Group III: ≥ 9 years:

A. As above: Observation
B. As above: Surgical containment.

13. What are the principles of treatment?

Ans: Principles:

A. Restoration and maintenance of movements: by counterpoised split Russell traction, analgesics, serial abduction splintage, adductor tenotomy (<7yrs) or adductor + iliopsoas tenotomy (>8yrs) may be required if spasm increases discomfort.
B. Reduce hip irritability
C. Prevent ball from extruding/collapsing (containment)
D. Regain a spherical femoral head and resumption of weight bearing and full activity.

14. How would you do containment?

Ans: Bracing or surgical:

Bracing:
  A. Non-ambulatory recumbent weight relieving:
     a. Abduction broomstick plaster casts
     b. Bivalved hip spica cast
     c. Milgram hip abduction orthosis
  B. Ambulatory static affected limb only:
     a. Harrison hip containment splint
  C. Ambulatory dynamic both limbs
     a. Petrie cast
     b. Scottish rite orthosis
  D. Ambulatory unilateral orthosis
     a. Trilateral socket orthosis.

   Surgical:
  A. Salter's innominate osteotomy/ Elizabethtown osteotomy: best done between 6-8 years, <20% epiphyseal extrusion
  B. Femoral varus derotation osteotomy (VDO): best done between 5-8 years and if arthrogram shows head containment in abduction and medial rotation
  C. Combination
  D. Lateral shelf.

15. What are the pre-requisites and contraindication of bracing?

Ans: Prerequisites:
  1. Full range of motion
  2. Entire femoral head containable
  3. Motor strength and balance should be adequate
  4. Arthrography: Essential to judge congruency throughout range of motion, rule out hinge abduction.

   Contraindicationsa ≡ indications for surgery
  1. Persistent/recurrent loss of movements
  2. Progressive collapse
  3. Psychosocial problems
  4. Non-compliant.

16. What is the end-point for bracing?

Ans: Orthosis can be discontinued when:

1. Disease enters healing stage
2. Increased density of femoral head appears
3. Medial segment of femoral head increases in size
4. Metaphyseal rarefaction ossifies
5. Intact lateral column
6. Complete subchondral ossification.

17. What are the prerequisites for Salter's osteotomy?

Ans: Prerequisites:

1. Non-irritable hip
2. Normal or near normal range of movements
3. Absence or minimal deformity of head
4. Concentric containment can be achieved.

18. What are the advantages of Salter's and VDO osteotomy?

Ans: The exact procedure to be chosen is controversial. Combination of innominate osteotomy and VDO may achieve containment avoiding complications of each (Staheli). The advantages of Salter's osteotomy are:

1. Lengthening of extremity
2. Anterolateral coverage
3. Avoidance of second surgery for plate removal (see VDO)

   Advantages of VDO:

1. Achieves anteversion and coverage simultaneously
2. Decompress femoral head
3. Decreases stresses across hip joint
4. Tends to enhance remodeling process.

19. When should one restrain from surgical containment?

Ans: Contraindications:

1. Persistent limitation of movements
2. Deformed femoral head
3. Age <5years
4. Presence of significant physeal involvement.

20. What are the indications of reconstruction surgeries?

Ans: These surgeries are done to correct residual deformities as follows:

1. Hinged abduction: If hips are congruent in adduction and have movements beyond point of congruency then – valgus extension osteotomy
2. Malformed femoral head: Garceau's chielectomy
3. Large malformed femoral head with 'mushrooming': Chiari's osteotomy
4. Coxa magna: Shelf augmentation (Staheli)
5. Femoral epiphyseal arrest (irrelevant overgrowth of greater trochanter): Trochanteric advancement. (Note – trochanter tip should be ideally at the level of center of femoral head and $1^{1/2}$ times the distance of radius of femoral head from center of femoral head.

21. What are the causes of shortening in Perthes disease?

Ans: The following account for shortening:

1. Fixed flexion deformity
2. Fixed abduction deformity
3. Destruction of femoral head
4. Coxa vara.

22. Theoretically can you think of a procedure that can be done in opposite limb (normal) to 'contain' the affected hip?

Ans: (*This s a very conceptual question but hypothetical one*!) one can do shortening of normal limb that will produce relative abduction deformity in affected limb and hence effect containment!

23. Why is soft tissue release contraindicated in hinged abduction?

Ans: (Salter's procedure as a rule involves ilio-psoas release in Perthes disease). Soft tissue release will enhance the tendency of hinged abduction.

CASE V:
CONGENITAL AND DEVELOPMENTAL COXA VARA

1. Why do you call it congenital coxa vara?

Ans: Findings:
1. Deformity (shortening) since birth, painless limp since walking age, progressive increase in deformity
2. Associated congenital deformities
3. Prominent greater trochanter
4. Upridden trochanter
5. Shortening (progressive)
6. Limited abduction
7. On flexion the knee points to same shoulder
8. Adduction, external rotation deformity
9. No telescopy (differentiate from DDH).

2. What is the cause of this deformity?

Ans: Can be divided into following causes:
1. Primary (often bilateral)
   a. Congenital: (present at birth and often associated with femoral shortening, cleidocranial dysostosis etc.)
   b. Developmental (presents later in childhood):
      i. As a part of PFFD (proximal focal femoral deficiency)
      ii. Multiple epiphyseal dysplasia
      iii. Achondroplasia
      iv. Hypothyroidism.
2. Secondary to:
   a. Perthes disease
   b. Infection
   c. Trauma
   d. Iatrogenic (treatment of DDH).

3. Why does this deformity worries an orthopaedician?

Ans: This is a progressive deformity with decreasing neck shaft angle with increasing age and weight bearing and may ultimately lead to pseudoarthrosis of neck. The gait and mechanics of hip are at a disadvantage producing early degenerative arthritis. The disease has a poor prognosis after 8 years of age.

4. How do you classify congenital coxa vara?

Ans: Severity classification based on angular measurements:
1. Hilgenreiner's epiphyseal (H-E) angle:
   a. Angle > 60° – Coxa vara almost always progresses
   b. Angle < 45° – Deformity almost always corrects over time and observation
   c. Angle between 46° to 90° – Natural history not clear, correct at the first sign of progression
2. Neck-Shaft angle (somewhat less reliable):
   a. Angle <100°: Surgically correct
   b. Angle >110°: Observe
   c. Angle between 100 and 110°: Close follow-up and correct if progresses.

5. What are the goals of treatment?

Ans: Goals are to:
- Restore neck shaft angle (to around 140° (135° - 150°) or H-E angle to < 45°)
- Reduce shortening
- Reduce abductor lurch.

6. How will you treat this patient?

Ans: The treatment of choice is subtrochanteric valgus osteotomy (*And I suppose the only one practiced!*). This restores neck shaft angle and provides virtual lengthening to limb. The disadvantage is that there is premature physeal closure in majority even without a direct physeal injury.

7. What do you see on X-ray?

Ans: Following findings are seen:
1. Decreased neck-shaft angle
2. Shallow acetabulum
3. Widened teardrop
4. Fairbank's triangle: 'Y' shaped defect present in the inferior part of capital physis and adjacent metaphysis, it is a defect in enchondral ossification with cartilage deposition at abnormal place.
5. Upridden trochanter
6. Short femur.

CASE VI:
SLIPPED CAPITAL FEMORAL EPIPHYSIS

(*This is a very unlikely case in Asian subcontinent as the disease is quite uncommon here. If given at all the cases will be of chronic slips*)

1. Why do you call it Slipped capital femoral epiphysis (SCFE)?

Ans: Findings:
1. Obese child, intermittent limp with or without pain
2. History of minor trauma followed by typical course
3. Shortening
4. Attitude of flexion, abduction, external rotation with waddling gait
5. Lower gluteal fold on same side
6. Restriction of abduction, internal rotation and flexion
7. Increased extension, adduction and external rotation
8. Increased femoral retroversion
9. Increased external rotation with straight leg raising/ flexion of hip due to tense posterior capsule
10. When child sits → thigh is held in external rotation and adduction resulting in tendency to cross the leg on uninvolved side.

2. What are your differential diagnoses?

Ans: Differentiate from:
1. TB hip: (here one finds characteristic deformities with movement restricted in all directions)
2. Perthes disease: (mild to moderate coxa vara may be difficult to differentiate just on the basis of history but here you will find restricted extension and also the age of onset is earlier in Perthes)
3. DDH: (head palpably outside joint, increased movements).

3. What is SCFE?

Ans: SCFE (or SUFE {slipped upper femoral epiphysis}) is postero-inferior slip of proximal femoral epiphysis in

relation to metaphysis. In correct terminology it is better defined as an antero-superior slip of femoral neck relative to epiphysis.

4. How do you classify SCFE?

Ans: The accepted classifications in the order of preference:
1. Stable and unstable slip (it also has a good prognostic value and fairly decides treatment):
   a. Stable slip is defined as ability of child to ambulate with or without crutches
   b. Unstable slip is where the child cannot walk with or without support.
2. Severity of slip (linear displacement of epiphysis relative to metaphysis, angular displacement of epiphysis relative to long axis of femoral neck, % surface contact remaining):

	Linear displacement (%)	*Angular displacement (deg)*	*Contact (%)*
Preslip	0	0	100
Minimal	<33	<30	58
Moderate	33-50	30-60	39
Severe	>50	>60	22-0

3. Duration of slip:

	Features	*Signs and symptoms*
Preslip	Widened physis, No displacement	Unknown
Acute	Displacement	< 3 weeks
Acute on chronic	Displacement + remodeling	
Chronic – early	With/without remodeling, physis open	>3 weeks
Chronic – late	Remodeling and closed physis	> 3 weeks
Chronic – late with osteoarthritis	Sclerosis; osteophytes, decreased joint space	

4. Direction of slip:
   a. Varus slip: common
   b. Valgus slip: these patients usually have a relative coxa valga with a more laterally tilted physis.

5. How will you manage this case?

Ans: I will assess and plan treatment after assessing the slip radiologically (AP and cross table lateral radiograph).

6. What are the treatment goals?

Ans: Goals:
   1. Prevent further slip
   2. Physeal closure
   3. Maintain adequate function
   4. Avoid complications like osteonecrosis and Chondrolysis

   Principles:
   1. All children with SCFE and open physis need treatment
   2. Simplest procedure that will achieve the goals should be done
   3. Patients with closed physis primarily require proximal femoral osteotomy for unacceptable gait, functional limitations and cosmetic deformity.

7. What are the treatment options?

Ans: Stable SCFE:
   1. *In situ* fixation: With single 6.5 mm (or 7.0 mm) cannulated partially threaded titanium screw
   2. Epiphysiodesis
   3. Proximal femoral osteotomy
      a. Cuneiform (Fish/Dunn): Sort of open reduction, highest complication rate
      b. Basilar neck osteotomy: Compromise surgery not very popular
      c. Intertrochanteric/Subtrochanteric: least complication and manipulates joint from 'outside'
   4. Spica cast immobilization

   Unstable SCFE:
   1. *In situ* fixation: As above without any 'intentional' reduction unless the slip is so severe that fixation cannot

be achieved in that case gentle single attempt at reduction to bring the slip in the 'chronic' position or a suitable position for fixation can be attempted. Whatever the consensus is towards immediate (within 24 hrs).

2. Epiphysiodesis (Schmidt technique)
3. Cuneiform osteotomy (acute-on-chronic slip)
4. Surgical dislocation of hip, open reduction, femoral osteotomy and fixation of SCFE (Ganz, described recently) – not very much in vogue.
   Always explain the risk of osteonecrosis and Chondrolysis with procedures, the closure one operates to the physis higher the chances.

8. What will you do for the other hip?

Ans: *There is a lot of controversy in addressing this issue*. In general it is logical to fix a disease that is known to have grave complications, in spite of the current knowledge that 20-35% are bilateral and majority of surgeries may be unnecessary.

CASE VII:
LATE SEQUELAE OF SEPTIC
ARTHRITIS OF HIP JOINT

1. Why do you call it sequel of septic arthritis?

Ans: Aka Tom-Smith arthritis
   Findings:

1. History of infectious arthritis in neo-natal/peri-natal period or infancy (typically < 2 years of age)
2. Prematurity
3. Shortening (true)
4. Increased gluteal creases
5. Wasting
6. Abductor lurch
7. Absent femoral pulses
8. Upridden trochanter
9. Adduction, external rotation deformity

10. Hypermobile joint (movements increased in all direction, typically identified by increased extension, abduction and internal rotation)
11. Desaults's positive.

2. What is your differential diagnosis?

Ans: DDH (may be bilateral, other congenital abnormalities else difficult to differentiate).

3. What are the various outcomes of septic arthritis?

Ans: Outcomes of septic arthritis in general! *You were not asked for a specific case – keep your mind open.*

1. Dislocated joint
2. Ankylosis of hip
3. Osteomyelitis of femur and persistent infection
4. Myositis ossificans
5. Contractures and various deformities primarily flexion and adduction
6. Coxa breva
7. Coxa magna.

4. How do you classify late sequelae of septic arthritis?

Ans: Radiological classification of severity of residual femoral head deformities (Choi et al):

Type I: (complete resolution of infection)

IA: Normal head (no residual deformity)

IB: Coxa magna (mild)

Type II: (epiphysis, physis and metaphysis are involved)

IIA: Coxa vara with deformed head

IIB: Progressive coxa vara/valga due to asymmetrical premature physeal closure

Type III: (primary involvement of neck with angular deformities)

IIIA: Coxa vara/valga with severe anteversion/retroversion

IIIB: Pseudoarthrosis of neck

Type IV: (unstable hips)

IVA: Destruction of femoral head and neck with small remnant of neck

IVB: Complete resorption of femoral head and neck and no articulation of hip.

Hunka gave a similar classification (there was no equivalent of subtype IIIA and IV and V were equivalent to IVA and IVB).

5. What will you do for this patient?

Ans: I will get an X-ray done to confirm the diagnosis, look for the geometry of hip joint and determining the type.

6. What are the treatment options?

Ans: The treatment options are decided based up on the following factors:

1. Age of patient
2. Type of sequelae
3. Abductor lurch
4. Movements and stability
5. Limb shortening
6. Previous reconstructive procedures.

Algorithm (simplified) for surgical management in accordance with various types:

Type I: Observation

Type II:

Type IIA: Observation

Type IIB: Observation, if deformity increases (as it will) – realignment procedures depending upon magnitude and age (sufficiently mature as a compromise to have a good fixation and minimal future deformity progression). Valgus osteotomy (for coxa vara), varus osteotomy for coxa valga.

Type III:

Type IIIA: Correction of angular deformity as above with derotation osteotomy to correct version.

Type IIIB: Valgus osteotomy with achieving union at pseudoarthrosis by bone grafting.

Type IV (*In general for type IV one can always safely recommend pelvic support osteotomy of Schanz type, Lorenz or Milch batchelor type*) other reconstructive options are as below:

Type IVA: Age $\geq$6 years – ilizarov hip reconstruction osteotomy with distal lengthening.

Age < 6years: Trochanteric epiphysis well formed and hyaline cartilage cover present – open reduction, Modified Harmon's procedure with distal transfer of greater trochanter.

Type IVB: age $\geq$ 6years – ilizarov hip reconstruction osteotomy with distal lengthening.

Age < 6 years – greater trochanteric arthroplasty, varus osteotomy, acetabular osteotomy to provide coverage. Failed IVA/IVB procedures are treated with ilizarov hip reconstruction osteotomy with lengthening.

7. What are the principles of Harmon's procedure?

Ans: Harmon's procedure (as described by L'Episcopo and Harmon separately) involved repositioning of cartilage remnant attached to neck into acetabulum facilitated by longitudinal femoral osteotomy (modified Harmon's as described by Choi et al provides a neck lengthening effect whereby a cartilage graft taken from iliac apophysis is placed into incomplete longitudinal femoral osteotomy). Often additional coverage procedures are required in the form of chiari osteotomy and additional limb lengthening later is often needed.

8. What are the principles of trochanteric arthroplasty?

Ans: This procedure banks upon the growth potential remaining in the uninvolved greater trochanter and remodeling potential which theoretically at least moulds towards the shape of femoral head if subjected to pressure and stresses in acetabulum. The osteotomy is done just below lesser trochanter and medial based wedge is removed. Vastus lateralis and medius attachments are retained to prevent devascularisation. Abductors are transferred distally. Pelvic osteotomy is required to provide additional coverage.

9. What are the limitations of trochanteric arthroplasty?

Ans: Limitations:

1. Avascularity of proximal segment
2. Gradual loss of correction due to femoral remodeling and straightening
3. Abductor weakness
4. Stiffness
5. Degenerative arthritis of hip
6. Difficult future total hip arthroplasty.

10. What is the role of pelvic support osteotomy?

Ans: Better choose Milch Batchelor osteotomy and describe as dealt in other section (Q43, case1). Lorenz bifurcation osteotomy puts proximal and of distal fragment into acetabulum with capsular interposition. These osteotomies provide stability and displace the line of weight bearing medially without approaching jip joint. Femoro-pelvic impingement may produce later degeneration and pain. In a nutshell, PSO:

1. Surgically shifts the shaft of femur near the center of gravity of the body so that the wt. bearing axis is more along the axis of femur
2. Supports pelvis by creating medial fulcrum
3. Improves adductor function by causing valgus
4. Abduction of distal fragment causes of gain of length.

11. What is ilizarov reconstruction osteotomy and lengthening?

Ans: This is a one step reconstruction for stability of hip joint by valgus subtrochanteric femoral osteotomy and lengthening with medialization (to compensate for abduction attitude) distally. The valgus osteotomy is overcorrected to eliminate any further adduction that may impinge on pelvis or cause valgus stress at knee.

CASE VIII:
DEVELOPMENTAL DYSPLASIA OF HIP

Findings

1. Trochanter upridden and palpated over Nelaton's line
2. Adduction contracture
3. Absent femoral pulses
4. Asymmetrical thigh folds ($\uparrow$ on the side of dislocation)
5. Widened perineum (bilateral dislocation)
6. Galeazzi's sign
7. Telescopy
8. Higher buttock fold on the affected side
9. Head palpably out of acetabulum
10. Movements – restricted abduction, increased internal rotation
11. Positive trendelenburg's sign
12. Positive Barlow's and Ortolani's sign
13. Duck-like or Sailor's gait
14. Increased lordosis
15. Positive impingement test (older child; flexion, adduction, internal rotation produces pain)
16. Positive instability test (tests anterior instability; extension, abduction and external rotation. '*?usefulness*)
17. Shortening.

1. Why is it not sequelae of septic arthritis?

Ans: Lack of typical history, restriction of abduction goes against it.

2. What are other differentials?

Ans: Various other differentials for this condition are:
1. Coxa vara
2. Pathological dislocation
3. Paralytic dislocation of poliomyelitis
4. Cerebral palsy.

3. What are the Hart's classic signs of hip dysplasia?

Ans: Three signs:
1. Limited hip abduction in 90° hip flexion
2. Ortolani's sign (first three months only)
3. Apparent shortening of thigh with hip and knee in flexion.

4. How do you define DDH?

Ans: Partial/complete displacement of femoral head from acetabulum because of inadequacy of acetabulum. Klisic introduced the term DDH (second 'D' represents both displacement and dysplasia but I prefer the latter).

5. Where is the defect in DDH?

Ans: The primary defect lies in acetabulum (deficient development 'dysplasia') whereby a constant defect is seen in the development of acetabular labrum (also a characteristic of lower forms where hind limbs are rudimentary). The femoral defect is secondary, however soon they become complementary! A ridge of thickened articular cartilage forms over posterosuperior wall of acetabulum 'Neolimbus' (Ortolani) over which head of femur rides in and out → clunk or 'Scatto' (Ortolani).

6. What are the obstacles for reduction of hip?

Ans: Obstacles:
1. Pericephalic insertion of capsule
2. Ligamentum teres
3. Inverted limbus
4. Iliopsoas muscle
5. Capsular adhesions
6. Pulvinar thickness in acetabulum
7. Transverse acetabular ligament pulled up along with ligamentum teres.

7. What will you do for this patient?

Ans: I will get an X-ray done to evaluate various parameters, ultrasonography and measurement of angles and for follow-up. If required an MRI (to detail the soft tissue problems).

8. What are the various radiographic findings?

Ans: Typical radiographic findings include:

- Perkin's quadrant's:
    - Normal hip → lower medial quadrant
    - Subluxated hip → upper medial quadrant
    - Dislocated hip → lower lateral quadrant
    - High dislocation → upper lateral quadrant
- "D" distance normally < 7.5 mm; "H" distance normally > 4-5mm
- Medial gap (distance separating proximal femur and a line perpendicular to lateral margin of ischium): Normally <4mm; 5mm → suspicious; ≥6mm → dislocation
- Tear drop: Normally appears by 4-6 months; failure to appear >6 months is pathological (open/closed/crossed/reversed/U-shaped/V-shaped are all associated with DDH.
- Increased acetabular index: Tells acetabular dysplasia and its severity
- C-E angle of Wiberg: Tells degree of subluxation; decrease or reversal of angle is seen depending upon degree of dislocation; normal is >19° (6-13 years)

9. What are the boundaries of tear drop?

Ans: Formed by lateral wall of lesser pelvis medially, wall of acetabulum laterally, curved line inferiorly and upper limit marked by acetabular notch.

10. How do you classify DDH?

Ans: Various types and classification based on etio-pathology, radiology, MRI, reducibility etc. are described. For prognostic and descriptive purpose the following may be noted:

Klisic subgroups:
- DDH "at risk": family history, breech, female child, 1st born, oligohydramnios, associated deformities (torticollis, clubfoot etc.)
- DDH: hypoplastic with limited abduction
- DDH: reducible dislocation with jerk of entry

- DDH: reducible dislocation with jerk of exit
- DDH: subluxation and limited abduction
- DDH: dislocation with limited abduction, femoral shortening and telescopy

Graf's classification {simplified} (based on ultrasonogram)

Type	α-angle	β-angle	Description	Treatment
I	>60°	<55°	Normal	None
II	43-60°	55-77°	Delayed ossification	*? (I mean 'controversial' - don't think it as a question)*
III	<43°	>77°	Lateral subluxation	Pavlik harness
IV	Immeasurable	Immeasurable	Dislocation	Pavlik harness/ closed/open reduction

Etio-pathological types:
- *Teratologic type:* Intra-uterine dislocation in early fetal life, complete acetabular malformation, marked displacement of femoral head, tight soft tissues, and fixed deformities.
- *Paralytic type:* Associated with AMC, spina bifida etc.
- *Diastrophic dysplasia:* Mucopolysaccharidosis.

11. What is the direction of hip dislocation in DDH?

Ans: Depending upon various pathological positions:
- *In extreme flexion:* Posterior dislocation over acetabular rim
- *Lateral rotation:* Stretching of anterior capsule (seen in neglected DDH with increased anteversion) – anterior dislocation {*I got this case in DNB exam!*}
- *Adduction and flexion:* Lateralization of head and dislocation over posterolateral acetabular rim.

12. What is Ilfeld phenomenon?

Ans: Clinical examination for infantile DDH is difficult and results are poorly reproducible due to various reasons

(crying baby, tense baby, hungry baby, hurried/ inexperienced doctor, too firm a grip 'white knuckle sign of Salter')

13. How will you plan treatment?

Ans: Various factors decide the treatment. Age, type of dislocation (etio-pathological, radiologic), reducibility all have a direct bearing.

Up to 3 months of age:

- Ultrasound (USG) positive acetabular dysplasia, negative Ortolani: treat prophylactically under supervision with 'triple diaper'.
- USG positive, Ortolani's positive, dislocatable/ subluxatable hip (especially >3-6 weeks) → abduction splint like Pavlik harness, Tubingen splint (less chances of osteonecrosis). Assess every 6 weeks.

Over the age of 3 months:

- Reducible hip → abduction/flexion splintage (3-6 months)
- Irreducible hip → reduce by traction ('human position' of 90° flexion and mild abduction, higher abduction → osteonecrosis) after arthrogram → successful (head should come at least to the upper edge of acetabulum), hold in spica for 8 weeks → mobilize in abduction splint. Follow for 2 years.

  – Fails → Open reduction

14. What is the role of Pavlik harness?

Ans: Usually the first choice during 1-6 months. Hips must be flexed >90° with proximal metaphysis pointing to triradiate cartilage and child must lie supine (*note- hip need not be reducible on clinical examination before application of harness, however chances of reduction of high dislocations are much less!*). Reduction obtained → continue for 6 weeks otherwise discontinue. Appearance of notch above acetabulum and improved acetabular development (↓ acetabular angle) indicate success. Four patterns of dislocation are seen with the use of Pavlik harness:

1. *Superior:* Additional flexion required

2. *Inferior:* Reduce flexion
3. *Lateral:* Gradual reduction can be anticipated
4. *Posterior:* Difficult to treat, usually requires adductor release.

15. What are the contraindications of Pavlik harness?

Ans: Do not use if:
- Child is in walking age
- Hip cannot be centered towards triradiate cartilage with 90-110° flexion.
- Dislocation develops several weeks after birth
- Dislocation associated with muscle imbalance:
  – Meningomyelocele,
  – Down's syndrome, marfan's syndrome etc.

16. What is the role of arthrogram?

Ans: Good investigation to assess the depth and stability of reduction. Width of medial dye pool indicates the likely stability of reduction. Fair reduction has 5-6 mm of dye pool. Poor reduction has >6 mm with difficult to hold reduction.

17. How will you do open reduction?

Ans: *Indications:*
1. Femoral head lying persistently above triradiate cartilage.
2. Arc of reduction and redislocation <25°
3. Femoral head remains laterally displaced >6 weeks (because of hourglass constriction)
4. Previous failed reduction.
   Either use median adduction approach as described by Ferguson or by Ludloff's incision or by anterior approach ('Bikini' incision). Anterior approach preferred for high dislocations, expected difficult reduction, significant growth difference. This additionally allows pelvic osteotomy and anterior capsular reefing. Clear all the obstacles for reduction and hold the reduction with K-wire. Assess stability of reduction; if stable but inadequate coverage for head (age between 2-8 years → Do a pelvic

osteotomy (Salter/Pemberton), age >8 years → needs acetabular procedure like Ganz's, Steel's or Dega's osteotomy). If unable to reduce with adequate stability then plan for additional procedure like femoral shortening/derotation and assess coverage as above.

18. What are the advantages of femoral osteotomy?

Ans: It reduces chances of osteonecrosis, Chondrolysis (by reducing pressure on femoral head) and redislocation.

19. What are the complications of combining acetabular procedure with femoral shortening?

Ans: Posterior dislocation particularly if derotation also done simultaneously.

20. What is the upper age of reduction of hip?

Ans: Difficult guidelines! In general unilateral cases have significant gait asymmetry and functional limitation than bilateral also complication rate is higher when both hips must be reduced
  • So for unilateral dislocation reduction can be attempted up to 9-10 years of age
  • For bilateral cases results are very unsatisfactory >8 years of age and probably the natural outcome of untreated bilateral dislocation is better than the results of treatment.

21. What are the indications of Salter's and Pemberton osteotomy?

Ans: The following are the indications:
  1. Failure to achieve stable reduction by open method due to acetabular incongruity or deficiency
  2. Progressive subluxation of hip after either conservative/operative treatment
  3. Failure of acetabulum to remodel
     Salter's type of osteotomy is done for rotational stabilization (if acetabulum has abnormal direction) and capsular reefing; Pemberton osteotomy is indicated in acetabular dysplasia (abnormal shape).

22. What are the indications for Salter's osteotomy?

Ans: This osteotomy can be done from 18 months to 8 years of age for unilateral cases and up to 5 years for bilateral cases. The acetabular roof is directed forward, downward and laterally to stabilize hip in functional position. Indications:
- Failure of acetabular angle to improve within 2 years following reduction
- Persistent mild to moderate dysplasia at 5 years.

23. What are the prerequisites and where do you do this osteotomy?

Ans: *Prerequisites:*
1. Concentric reduction of hip joint
2. Good range of movements
3. Mild to moderate dysplasia only

Site: just above acetabulum running transversely from anterior inferior iliac spine to greater sciatic notch, lower fragment displaced forward, downward and outward.

24. Where is the hinge of rotation for this osteotomy?

Ans: Pubic symphysis.

25. What are the disadvantages of this osteotomy?

Ans: *All procedures have some!*
- *Unstable osteotomy:* Requires fixation
- Correction limited by the size of graft
- Defect created in posterior acetabulum with narrowing of joint space

26. What is Pemberton osteotomy?

Ans: Pemberton's incomplete pericapsular osteotomy achieves stabilization by rotating the anterosuperior portion of acetabulum forwards, laterally and downwards. Posterior portion remains undisturbed. The osteotomy is often done in the presence of moderate to severe acetabular dysplasia but there should be adequate remodeling duration left so it is done for ages from 18 months to 6-7 years.

27. Where is the hinge for this osteotomy?

Ans: Triradiate cartilage.

28. What is Chiari osteotomy?

Ans: Chiari osteotomy is a medial acetabular displacement osteotomy over intact hip capsule for inadequacy to obtain concentric reduction. The osteotomy is aimed at:
- Deepening acetabulum
- Displacing the femoral head medially to reduce stresses
- Covering a subluxed head but not a dislocated head
Over time hip capsule transforms into fibrocartilage. The indications for the osteotomy are as follows:
1. Symptomatic patient in adolescent age up to middle age
2. Femoral head has little to no support
3. False acetabulum is not too high.

29. How does chiari osteotomy differ from Salter's or Pemberton's osteotomy?

Ans: Salter's and Pemberton's osteotomy alter the orientation of acetabulum in order to cover the anterolateral defect present in these cases whereas Chiari provides a new lateral extension to existing acetabular roof.

30. What is Shelf procedure?

Ans: Staheli shelf procedure is also described for hip that are not concentrically reducible. Shelf is made from outer table of ilium over supero-antero-lateral aspect of femoral head and augmented with ample graft.

31. What are the various palliative procedures?

Ans: These procedures are indicated in symptomatic patients where reduction is no longer possible by either closed/ open reduction methods. Arthrodesis, arthroplasty, osteotomy all have their own indications as discussed briefly. Arthritic pain in a young heavy labourer can be managed by arthrodesis. Osteotomy of Schanz type to align the proximal fragment with pelvis and distal fragment parallel to weight bearing axis.

CASE IX:
POLIOMYELITIS AFFECTION OF HIP

1. What will you do for paralytic hip instability?

Ans: First I will assess whether it is a complete dislocation, incomplete dislocation or hip subluxation. Assessment of muscle power is also important as the abductors and extensors are often involved leading to flexion and adduction contracture in unstable hip.

2. What is incomplete dislocation?

Ans: The hip dislocates in adduction and flexion but is otherwise stable in abduction. This often presents as 'snapping hip'.

3. How will you manage complete dislocation?

Ans: For a true dislocation I would like to determine whether the cause of dislocation lies within the joint (viz coxa valga) or periarticular soft tissues (muscle paralysis/ ligament stretching) {type one Somerville}. In the other type the dislocation is secondary to pelvic obliquity/ scoliotic deformity.

4. How will you manage first type?

Ans: I will correct deformity by traction to gradually gain abduction and reduce the hip joint till acetabular level. Deformity in hip joint proper needs osseous correction, so I will do varus osteotomy and correct anteversion for coxa valga. Simultaneous adductor contracture needs adductor tenotomy and other weak muscles also need management. If the hip is not reducible then after release of contractures I will make Staheli shelf to provide a superior support for the head. If everything else fails hip arthrodesis can be done. The second type needs specific correction (spinal deformity) by addressing the causative factors.

5. How will you manage hip contracture?

Ans: The deformity at hip is typical of iliotibial band contracture producing FABER deformity. This is supplemented by iliopsoas, sartorius and rectus femoris. The deformity and

compensatory mechanisms due to iliotibial band and tensor fascia lata (TFL) contracture are progressive and should be promptly corrected. There are two methods of correction:

1. Stretching: Suitable for mild deformities; causes pain, damages tissues and is unreliable for moderate to severe deformities. Hence for these deformities prefer option '2':-

2. Release of muscles and soft tissue: Soutter's or Ober's release at hip and Yount's release (iliotibial band) at knee. Soutter's release primarily is aimed at correcting flexion deformity by releasing flexors of hip; additional correction can be achieved by releasing iliopsoas, anterior joint capsule and abdominal muscles from crest. Ober's addresses the FABER deformity by releasing iliopsoas, rectus, Sartorius, gluteus medius and minimus and TFL and joint capsule. Campbell release and transfer of iliac crest is another option.

6. How will you manage abductor and extensor weakness?

Ans: The gluteus medius and maximus weakness needs to be addressed primarily for abductor and extensor weakness respectively. Gluteus maximus weakness produces extensor lurch and increased lumbar lordosis, whereas the medius weakness produces trendelenburg gait.

Gluteus maximus paralysis: Difficult to compensate by transfer due to lack of powerful muscles. Erector spinae transfer (Ober's) or posterior shift of TFL origin can be done to produce some functional compensation.

Gluteus medius paralysis: There are two muscles commonly used for compensating the paralysis:

1. External oblique transfer (Thomas, Thompson) to greater trochanter and adductor release (or transfer): This has following advantages over iliopsoas transfer:
   a. Synergistic transfer
   b. Maintaining power around hip joint
   c. Preserving ilium for other bony procedure if needed
   d. Additional of external muscle power

2. Iliopsoas transfer: Works for combined medius and maximus paralysis. Sharrard's modification of mustard

operation is often used. Here psoas along with whole of the iliacus muscle is transferred to outer surface of ilium and tendon is attached to greater trochanter which now acts as abductor and extensor.

CASE X:
OLD UNREDUCED DISLOCATIONS
AND FRACTURE DISLOCATION

FINDINGS

History

- Trauma (often significant)
- Treatment with failed reduction/ neglected/ traction

Examination

- FADIR in posterior dislocation (commoner ones)
- Shortening
- Wasting
- ASIS often high due to adduction deformity
- Absent femoral pulse
- Round, bony hard, globular (spherical) swelling palpable in gluteal region
- Crepitus
- Movements often only present in sagittal plane (flexion)
- Inverted Bryant's triangle
- Sciatic nerve injury (foot drop)

1. What are your differential diagnoses?

Ans: *The above characteristic findings (the pathognomonic palpable head in gluteal region) are often absent in other disorders*:

1. Pathological dislocation (TB hip, Septic arthritis)
2. TB hip with destruction of head (will have painful global restriction of movements in active stage or fibrosis ankylosis or hypermobile unstable hip in healed disease)
3. AVN hip with destruction of head will have secondary osteoarthritis, and often external rotation deformity

4. Old unreduced fracture neck of femur (external rotation deformity, limited shortening).

2. What will you do next?

Ans: I will confirm my diagnosis radiologically (X-ray pelvis AP projection and AP and cross leg lateral of involved hip).

3. What will you find on X-rays?

Ans: The following are the characteristic and ancillary findings:
- Loss of congruity of femoral head and acetabulum
- Broken Shenton-minard line
- Proximal migration of trochanter
- Smaller obturator foramen
- Smaller ilium
- Less prominent lesser trochanter
- Adducted femur.

The last few signs are ancillary signs of flexion and internal rotation of hip.

4. What are the causes of chronic old unreduced posterior dislocation of hip?

Ans: Various causes:
1. Inability to identify/Quack treatment
2. Incarcerated acetabular or femoral head fragment (types III, IV, V)
3. Buttonholing of femoral head through capsule
4. Inversion of labrum and obstruction to reduction
5. Incarceration of piriformis
6. Posterior unstable acetabular fractures (type II, III, IV).

5. What will you do next?

Ans: I will get CT-scan of the pelvis to assess associated fractures of acetabulum, MRI to look for vascularity of head and condition of acetabular soft tissues.

6. How will you manage the patient?

Ans: The patient will be assessed on the basis of associated injuries (Classify as per Thompson and Epstein classification of posterior dislocation of hip) and vascularity

of head and medical condition of patient (fitness for major interventions and pre-operative ambulatory status).

7. What is the Thompson and Epstein classification?

Ans: Classified posterior dislocation of hip on the basis of fractures of acetabulum and femoral head fractures:

1. Type I: With no or minor acetabular fracture
2. Type II: With a large chunk of posterior wall of acetabulum
3. Type III: With comminuted rim fracture of acetabulum
4. Type IV: With rim and floor fracture of acetabulum
5. Type V: With fracture of head.

8. How will you treat the patient?

Ans: *Type I:* < 3 months old with viable head → closed reduction ↓ GA → {if fails then} Gupta's heavy traction method

Type I: > 3 months old with viable head → Gupta's method vs. open reduction (preferred) using anterior or anterolateral approach

Type II: <3 months (viable head) → reconstruction of acetabulum and open reduction (preliminary heavy traction often required)

Type II: >3 months (viable head) → arthroplasty vs. arthrodesis (as while trying open reduction the acetabular cartilage will often be damaged irreparably in an attempt to curette the soft tissues)

Type III: As for type II

Type IV and Type V: Viable head → reconstruction and open reduction must be tried for young patients for older patients' → arthroplasty is better

Nonviable head (any type any duration): Arthroplasty is the procedure of choice vs. arthrodesis.

9. What is Gupta's method for closed reduction with traction?

Ans: Gupta and Shravat devised the method for closed reduction of hip < 3 months old using traction and abduction. Apply skeletal upper tibial traction with 18 kg weight and sedate the patient. When head comes at or

below the acetabular margin then gradually abduct the limb and reduce weight @ 3.6 kg every fourth day. Once the reduction is achieved then traction with 7 kg weight is maintained for 2 weeks. Patient is allowed NWB for 4 weeks followed by gradual weight bearing to full weight bearing after 3 months. The aim is to obtain concentric reduction.

10. Why the duration of three months is critical?

Ans: After 3 months soft tissue often develops inside the acetabular cavity that prevents reduction of head by closed methods.

11. What is the role of arthrodesis?

Ans: Arthrodesis is a good proposition for young patients. However, achieving solid bony union is often difficult in the setting of osteonecrosis.

CASE XI:
MALUNITED/OLD NEGLECTED FRACTURE INTERTROCHANTERIC

{Unusual case but given commonly in endemic areas like eastern India. Importantly concepts of intertrochanteric fixation and methods are assessed rather than the case itself.

Read times: 4-6 (DNB and MS candidates)}

Diagnosis

This is a 68-year-old female with 6 months old malunited united fracture of right proximal femoral region** most likely intertrochanteric (or a pathological fracture if known!). There is 30° adduction deformity, 40° external rotation deformity and 2.5 cm of true supratrochanteric shortening of right lower limb.

Findings

1. Externally rotated limb at hip, adduction attitude may be seen in long standing (>3 months) malunions.
2. Shortening (supratrochanteric).
3. Prominence at the region of greater trochanter.

4. Broadening and irregularity over greater trochanter
5. Prominent trochanter (that may be posteriorly displaced)
6. External rotation deformity and adduction deformity at hip.
7. Decreased abduction, external rotation and, extension at hip.
8. Movements often painful in all directions due to impingement of soft tissue.
9. Trendelenburg positive (usually in old age muscle power is weak and long standing muscle inhibition because of pain producing hypotrophy and varus at fracture manifests as positive Trendlenburg sign).
10. Able to do active SLRT (malunited fractures should not hamper SLR).
11. Telescopy negative (do only if painless!)
12. Shortening of base of bryant's triangle, increased hypotenuse and perpendicular.

Important note: Old neglected intertrochanteric fractures are commonly pathological. History taking and examination should incorporate finding the primary cause of fracture. Traumatic neglected cases as may be found in remote areas are less likely to be kept in exams as they are rare.

1. Why do you call this an intertrochanteric fracture malunion, would you like to present any differentials that have been excluded?

Ans: See above for findings in support of intertrochanteric fracture malunion. Differentials include:
   a. Malunited basicervical fracture neck femur** (really cannot be differentiated from malunited IT fracture so never commit specific diagnosis as NOF or IT rather use term like "region")
   b. Malunited subtrochanteric fracture.
   c. Congenital coxa vara.
   d. Neoplastic pathology (cyst/GCT) at greater trochanter.
   e. Sequelae of septic arthritis of hip.
   f. Neglected dysplastic dislocation of hip.

2. What will you do next and why?

Ans: I will get X-ray of the involved hip in anteroposterior and lateral projections to look for configuration and status of

fracture (union). I will also look for any primary pathology (neoplasia/osteoporosis) that might be responsible for the fracture.

3. What pathology do you suspect?

Ans: Osteoporosis, Osteomalacia, Neoplastic pathology (metastasis mainly, primary tumor at the region are uncommon for this age).

4. What are the problems to patient?

Ans: Shortening, Trendelenberg lurch, reduced movements, pain (if recent), hip arthritis in long run.

5. How will you manage this patient?

Ans: Malunited fracture – Assess the movements at hip. If the movements are reasonably preserved and muscle power is regained then I will discuss with the patient corrective surgical options vs conservative method. Patient will gain limb length with corrective valgus osteotomy, abductor lurch will improve and pain due to abnormal transfer of forces across the hip joint will be relieved. Patient will have to, however, be bedridden for a week and slowly regain movements over a month to full weight bearing walk and usual complications of surgery viz. infection, non-union, etc. With conservative management there is a high chance for development of hip degenerative arthritis.

Old-neglected fracture (>3 weeks old) – If the fracture is <6 weeks old (mobile on fluoroscopy) I will do open reduction of the fracture with bone grafting and fix using sliding hip screw and start patient on graduated hip mobilisation and muscle strengthening program. Fracture > 6 weeks old (not mobile on fluoroscopy) I will wait for fracture union and graduated muscle strengthening program, osteotomy will be planned after around 1 year for fracture to consolidate.

6. How will you manage a fresh intertrochanteric fracture?

Ans: After assessing the medical condition of the patient and discussing the willingness for surgery, I will treat the patient as follows.

For stable intertrochanteric fractures I will use sliding hip screw.

For an unstable fracture pattern I will preferably use an intramedullary fixation.

7. How do you define an unstable intertrochanteric fracture?

Ans: Presence of any or combination of:
   a. Four part fracture
   b. Medial cortical comminution (loss of calcar support)
   c. Reverse obliquity of main fracture line
   d. Large and separate posterior greater trochanteric fragment
   e. Subtrochanteric extension.

8. How will you fix a stable intertrochanteric fracture?

Ans: I will use sliding hip screw (or dynamic hip screw, AO).
   There are a lot of devices mentioned in the literature like variable angle sliding hip screw, talon compression hip screw, Medoff plate, percutaneous compression plate, etc. but speak only those in exams that you have seen or used and can defend for further questions!

9. What is the entry point for lag screw used in sliding hip screw system?

Ans: Approximately two cm below the vastus lateralis ridge.

10. What is the ideal position of lag screw?

Ans: The lag screw should be placed in center-center position of femoral head. Previous recommendations of inferior and posterior placement of the screw to prevent superior and anterior cutout no longer hold true as they actually increase the TAD. Eccentric placement of screw also places more rotational stress at the fracture site and may cause early failure.

11. What should be ideal angle of barrel plate used?

Ans: First understand that, for unstable fractures there is usually a posteromedial defect. Second the joint reaction forces are transmitted at around 160°. Now Barrel plates are available in angle ranges from 130° to 155°.

Posteromedial fragments are most difficult to reduce even partially. When a larger angle plate is used one is actually putting the screw eccentrically (mean neck shaft angle is 129°) so that the head will be in valgus reduction to produce a center-center screw position. This valgus reduction increases the medial gap and makes the construct unstable. Also it is difficult to negotiate the guide wire off the medial cortex to center it in the head. For such unstable construct some form of osteotomy is required to improve medial loading. Otherwise the load to failure of these angle construct is higher for stable fractures as the bending forces are minimal (screw nearly parallel to resulting forces) and fracture is perpendicular to the forces due to valgus reduction.

When one uses the 135° barrel plate (as is commonly practiced) the posteromedial opening is less and one comfortably places the screw at the center of head reducing the potential of iatrogenically displacing the anatomical fracture reduction. Although the bending moment is higher than larger angle barrel but it is preferred for anatomical reduction.

12. What is TAD?

Ans: TAD is tip apex distance first referred by Baumgaertner. The distance is sum total of the distance in mm of screw tip to apex of the medial femoral head curve in anteroposterior and lateral radiographs after magnification correction. Ideal is 11 mm to 25 mm, but surgeons tend to achieve TAD <20 mm.

13. Why do you call this a lag screw?

Ans: The Screw slides in the barrel plate and has no other fixed end. By itself it cannot produce compression. The screw only acts to facilitate collapse. Compression is aided by separate compression screw put at the rear of lag screw.

14. When should you avoid giving compression at fracture site?

Ans: In osteoporotic fractures and weak bones there is a high chance of the screw pull out from cancellous bone so compression should be avoided.

15. When should you put short barrel plate?

Ans: Barrel length is chosen to facilitate optimal sliding and collapse of the fracture without letting the screw jam inside barrel under bending moment (vertical force). The larger the length of screw outside the barrel higher is the bending moment at screw barrel junction jamming the screw. Again, if a smaller length screw is put then screw may touch the barrel before complete fracture consolidation has occurred. Short barrel length (25 mm) is used for screw lengths ≤80 mm while otherwise standard barrel length (38 mm) is preferable.

16. What is the thread length for DHS lag screw?

Ans: The thread length for standard DHS lag screw is 22 mm, thread diameter is 12.5 mm and shaft diameter is 8 mm.

17. What are the options for intramedullary fixation of fracture IT?

Ans: Gamma nail (second and third generation), Proximal femoral nail (short and long and PFN-a), intramedullary hip screw, trochanteric hip screw, trochanteric antegrade nail.

18. Can all unstable intertrochanteric fractures be managed with intramedullary fixation?

Ans: NO. Intramedullary fixation is typically suitable for subtrochanteric extension, reverse obliquity fracture and medial cortical comminution hampering medial support. IT fractures where there is no lateral wall support are also good candidates for intramedullary fixation. Fractures with large posterior fragment or four part fractures where greater trochanteric fragment is not localized or reduced well are not well suited for intramedullary fixation due to entry point problems. These are better fixed with tension band wiring of greater trochanter fragment and additional trochanteric stabilising plate with a sliding screw fixation. Alternatively dynamic condylar screw or condylar blade plate can be used but patient has to delay weight bearing. *(DCS or CBP provide a prosthetic lateral wall of metal, it is unclear how well a support can these provide!)*

19. Why you do not use DHS for fixation of reverse oblique fracture?

Ans: DHS is unable to resist the medial displacement tendency of distal shaft fragment. With continued fracture consolidation the proximal fragment has no good screw purchase (lag screw only is meant for controlled collapse and not fracture fixation) and has constant abductor pull. The distal fragment has constant adductor pull from muscles. Now the fracture line (reverse oblique slanting upwards medially) is such that it will not resist medial displacement of the distal shaft fragment so the fixation will surely fail. Intramedullary device is best suited to fix the fracture. DCS or CBP can be used provided there is no medial comminution (medial continuity and contact is imperative) else they will fail ultimately in varus collapse.

20. What are the pros and cons of using intramedullary implant?
Ans:

Advantages:

   a. Ability to fix majority of fractures.
   b. Short surgical time and less blood loss for unstable fractures (no difference comparing to DHS fixation for stable IT fractures).
   c. Smaller moment arm so better able to bear tensile forces and lesser calcar strain is produced. A sliding hip screw produces 1.5 times the calcar strain of a normal femur while intramedullary implant produces <10% of strain.
   d. Better controlled collapse as the bending moment at lag screw for nail is lesser than DHS.
   e. Biomechanically the nail construct is more stiff (so more stable) than DHS for torsional and bending forces.

Disadvantages:

   a. Abductor injury while insertion.
   b. Difficult revision by arthroplasty if fails
   c. Anterior thigh pain due to impingement of unlocked nail tip.
   d. Curvature mismatch of nail and femora may produce iatrogenic fracture.

e. Stress concentration at tip of nail predisposing to fracture.

f. Costlier implant.

21. What is "Z-effect" and reverse Z-effect?

Ans: 'Z-effect' and 'reverse Z-effect' are complications that arise from fixation of unstable proximal femoral fractures with PFN having two screws (newer version has single blade). Z-effect is lateral migration of caudal screw, varus collapse and perforation of femoral head by superior screw while reverse Z-effect is lateral migration of cephalic screw, varus collapse and femoral head cutout by inferior screw. Cause is not fully elucidated, however varus fixation of fracture, severe medial comminution, inappropriate entry point and poor bone quality have been provisionally implicated.

22. Do you know of any intraoperative procedure to stabilize an unstable fracture?

Ans: There are various osteotomies that can be done to improve the contact between fragments (in unstable fractures) and load transmission and were devised to be used with fixed angle nail plate design like the *Smith-Petersen nail plate*:

a. Dimon-Hughston medial displacement osteotomy

b. Sarmiento valgus osteotomy

c. Wayne County lateral displacement reduction.

There is no evidence to support the use of these osteotomies with sliding hip screw. Instead one should aim at obtaining an anatomical reduction and fixing with a standard device.

23. What are the mechanisms of failure of DHS?

Ans: DHS fails primarily if the fracture is not consolidated and stabilized whence still instability remains at the fracture site by virtue of fracture configuration and screw has either jammed or slide to limit has already occurred. In this instance the construct behaves like a fixed angle nail plate device failure involved any of the four modes:

   a. Penetration of the nail through head into hip joint.
   b. Pulling out of plate and shaft screws (varus collapse)
   c. Cutout of lag screw superiorly through head
   d. Bending and break at barrel plate junction.

24. What is the role of arthroplasty for treatment of unstable intertrochanteric fractures?

Ans: Due to higher failure rates for fixation of unstable intertrochanteric fractures and difficult revision following failed fixation many surgeons have resorted to arthroplasty as primary treatment modality. The modality has become increasingly popular and promising. Both cemented and cementless; hemi- and total hip arthroplasties have been used with comparable or better outcomes in terms of *postoperative complications*. Cone prostheses (cementless) are more popular as there is a higher chance of cementless stems subsiding into the wider femoral canal following osteolysis. The treatment is, however costly and requires specialist.

25. Would you like to do anything for poor bone quality?

Ans: I will start the patient on pharmacotherapy for osteoporosis with calcium, vitmin D and oral bisphosphonates. If the patient is willing I will prefer Intravenous bisphosphonate to avoid adverse effects with the use of oral compounds and ensure compliance. I will avoid putting compression screw during surgery.

26. What are the management options for poor bone purchase in osteoporosis?

Ans: Using bone cement in femoral head to improve lag screw purchase can be done else implants specific to minimize screw cut-out should be used like:
   a. Delta bolt.
   b. Spiral blade instead of screw.
   c. Injecting cement into the screw tract before putting the screw.
   d. Talon compression hip screw.

27. Can you start the bisphosphonates at fracture healing?

Ans: I will start the bisphosphonate (either alendronate or zoledronic acid) at the earliest. I will take care to make the patient calcium replete for 3-4 days before starting bisphosphonates. There is no evidence till date to delay bisphosphonate therapy. They do not delay callus. Real concern is sequestration of bisphosphonates at the fracture healing site so that with reduced systemic drug efficacy may be lost. There is no answer to this but one can use weekly bisphosphonates to allow frequent body loading with the drug rather than giving yearly administered drugs.

28. Can you start Parathormone at acute stage and with surgical procedure?

Ans: rPTH has been found to improve bone healing in animals. It can be systemically or locally administered. Locally administered drug has been found to improve implant anchorage.

29. How do you classify intertrochanteric fractures?

Ans: The question has been put in last as there is no consistently followed classification. Instead usually the surgeons see a fracture and recall a matching 'type' from various classifications. The commonly followed is Evans (1949) as it differentiates stable from unstable types and is easy. AO/OTA classification has been found to be more consistent if regularly followed.

Evan's classification for intertrochanteric fracture:

Type I: Undisplaced 2-fragment fracture

Type II: Displaced 2-fragment fracture

Type III: Three-fragment fracture without posterolateral support (displaced greater trochanter fragment)

Type IV: Three-fragment fracture without medial support (displaced lesser trochanter or posteromedial calcar fragment)

Type V: Four-fragment fracture without posterolateral and medial support (combination of Type III and Type IV)

R: Reversed obliquity fracture

The Knee

EXAMINATION POINTS
FOR A KNEE CASE

HISTORY

1. *Pain:* Onset, duration, severity, character (achingdegenerative, tumor; throbbing - infection), progression (insidious- degenerative and mechanical; acute- traumatic and infection), diurnal variation (morning- inflammatory; evening – mechanical and degenerative; night- inflammatory and tuberculosis), activity related (degenerative viz. osteoarthritis), radiation (usually to calf- degenerative and mechanical), pain after prolonged flexion (patellar disorders; "theater sign"), bar or vice like pain suggests patella baja (low-riding patella), pain at other sites (inflammatory, referred pain from lower back), previous history/episodes.

2. *Deformity:* Varus (rickets, osteoarthritis, post-traumatic, etc), valgus (rickets, rheumatoid arthritis), recurvatum (poliomyelitis, generalized joint laxity), flexion (effusion, infection, sequelae, scurvy, hemophilia), triple deformity (tuberculosis), bizarre (Charcot's), broadening (hemophilia, arthritis-osteophytes), patellar (alta, baja pronounced as *baha*), lateral subluxed/dislocated, small, double-bipartite), deformity at other joints especially hips (congenital) and small joints (inflammatory).

3. *Swelling:* When? (Onset and duration), How? (traumatic or atraumatic - infection, inflammatory, tumor), How long? Associated symptoms? (Fever, pain, etc). Previous aspiration "Recurrent" - Straw colored (hydrarthosis-inflammatory, osteoarthritis, loose body; all due to synovial irritation), pus (pyarthrosis- septic arthritis), blood-colored (haemarthrosis-haemophilia, traumatic), Amber colored (Pigmented villonodular synovitis (PVNS)). Long-standing painless swelling may be meniscal cysts and benign tumors, viz. Osteochondroma (painless), painful swellings – tumors (malignant) and hematoma in soft tissues.

4. *Laxity:* {English = Laxity; French = Instability or Instabilitè}. Specifically enquire about "Going out" of knee – Anterior

cruciate ligament (ACL) tear, Patellar dislocation or subluxation (subluxation usually presents with anterior knee pain). Symptom of "Giving Way" (knee failing to provide support fully – sort of apprehension ± pain) especially on walking on uneven surfaces usually indicates "Interposition" (meniscus, cartilage-free bodies, synovial membrane, etc); "Cartilage damage"; "muscle weakness" (Polio, neuropathy), "generalized joint laxity" (*Marfan's etc. See chapter 5; Case II; Q 11).*

5. *Locking:* [Inability to extend fully : free flexion] "True Locking" also called meniscal locking is usually due to meniscal tear but may also be caused by loose bodies– "funny" feeling in joint (joint mice) or ACL stump. "False locking" also called patellar locking rather "catching" may also be caused by hypertrophied fat pad.

6. *Limitation of movement or stiffness:* Painful (inflammatory, degenerative, traumatic, tumors), painless "mechanical" (soft tissue- contractures of muscles, tendon, fascia, post-operative, inadvertent- total knee replacement, arthrodesis; bony – malunited fractures, tumors, osteophytes). *Intra-articular (usually painful, bony, sudden stop- firm end point, post-surgical); extraarticular (developmental, painless, soft-end point).*

7. *Other clinically relevant features:*
   a. Noise from joint: Crepitus, snapping syndromes, "Thud" of discoid meniscus. "clunks" and "clicks" of menisci.
   b. Evaluation for IKDC score.

EXAMINATION

General and Systemic Examination: 'Rickets, hypermobility syndrome (Down's syndrome, Larsen, Marfan's, Ehler-Danlos), haemophilia, rheumatoid, dysplasia, myopathy'

LOCAL EXAMINATION

Prerequisites

1. Position, lighting, draping: Examine both knees (limbs) together. Expose from flanks to ankle for examination of

knee joints. Examine in ambient day light. Adequately drape the patient to avoid embarrassment.

2. Firm couch for supine examination.
3. Examine the patient first in standing position then the gait and lastly in "lying down" position (may require prone position also for some tests). However, if the patient has significant pain in standing or walking then postpone it till the end.
4. Keep other ("normal") joint in same position for comparison, e.g. valgus in a knee with flexion contracture should be commented only by observing the normal joint in same degree of flexion (*Ideally speaking varus is definitely pathological and can be commented upon even in a flexion deformity, however, it is less well accepted for valgus deformity*).
5. Be gentle and always explain to the patient what you are going to do and what you expect. Warn if it is going to be painful and be sorry if it hurts!

Inspection

Patient standing (Kneel down in front of patient to look head on):
1. Attitude: "Front and Side"- extension at knee joint and hip joint with foot plantigrade planted fully and patellae looking forwards.
2. Alignment: Patient looking to horizon and you looking head on (i.e. "front only") - knee and medial malleoli barely are touching each other with great toe pointing forwards.
   (Always speak of "normal alignment" and not as normal joint as there is inbuilt varus in recurvatum deformity).
   a. Genu valgum
   b. Genu Varum
   c. Windswept deformity

Now inspect joint from "all around"
1. Swelling: Generalized, localized (tumor, bursitis, meniscal cyst, Baker's cyst, Osgood-Schlatter disease, Sinding-Larsen-Johanson syndrome, Jumper's knee- patellar tendinitis, Hoffa's disease), thickened synovial band seen as swelling at side (meniscal tear).

2. Effusion (fullness of peri-patellar fossae)
3. Skin: Neurofibromatosis
4. Scars
5. Sinus
6. Patella and extensor apparatus
    a. Bipartite patella (swelling at superolateral aspect)
    b. Squinting patella (increased femoral ante-version)
    c. Frog-eye patella (patella facing out – femoral condyle hypoplasia, subluxed patella, increased Q angle, etc)
    d. Patella magna (osteophytes) and breva (hypoplasia)
7. Wasting especially of vastus medialis.

Gait: (Table 3.1)

Table 3.1: Gait

Examine from anterior and posterior perspectives:
1. Valgus thrust gait (single joint- osteoarthritis, malunited tibial plateau #)
2. Valgus thrust with circumduction (both joints affected)
3. Varus thrust gait (osteoarthritis, malunited tibial plateau #, lateral lig. complex injury.
4. Varus recurvatum thrust (postero-lateral laxity)
5. Duck-footed (Slew-footed) gait (increased femoral anteversion)

Lateral perspective:
1. Antalgic gait
2. Stiff knee gait
3. Flexed knee gait

Examination in sitting position:
1. Patella Alta and Baja
2. J-sign (bony or soft tissue pathology at trochlea – trochlear dysplasia, internal femoral torsion, tense lateral retinaculum)
3. Dynamic patellar tracking (N- straight line) and patellar tilt.

Examination in supine position: Confirm above findings report any change in findings as correction of version and deformity (correction denotes intra-articular deformity). Special attention to effusion and flexion deformity should be given as small amount may be hidden in standing.

Palpation

Anterior aspect:

1. Temperature and Effusion
   a. Fluid shift (visible fluid wave ≈ 15 ml) – mild effusion.
   b. Palpable fluid wave (cross fluctuation ≈ 30 ml) moderate effusion.
   c. Ballotable patella sign (patellar tap test) - gross effusion.
   d. Trans-illumination
   e. Palpate synovium (from above below feel for thickened bands/doughy thickenings which "rolls" under the finger)
2. Patella and extensor apparatus:
   a. Patellar facet tenderness (push patella medially–feel undersurface)
   b. Patellar/Quadriceps tendinitis (Jumper's knee)
   c. Apical patellar tenderness and bipartite patella (Base)
   d. Patellar glide/ Slide (<1 cm each side, Sage sign) and tracking
   e. Patellar lift ("push and lift", N= none on lateral side, minimal over medial aspect <10°)
   f. Patellar grind test (push patella over femur with palm and flex the joint)
   g. Fairbank's apprehension test (Glide patella laterally – flex the joint – +ve at around 30°)
   h. Tibial tubercle
3. Wilson's test (osteochondritis dissecans) – lift ankle → flex knee 90º → extend knee → pain due to ACL impingement

Medial aspect:

1. Joint line tenderness (90º flexion: typically elicited medially; laterally obscured by iliotibial band (ITB))
2. Pes Anserinus and bursa, meniscal cysts
3. Medial collateral ligament (MCL) - both femoral and tibial ends

Lateral aspect:

1. Lateral collateral ligament (LCL), meniscal cysts

2. Iliotibial band and Gerdy's tubercle
3. Ober's test (lateral decubitus position; knee flexion to
   $90°$ → hip abduction $40°$ → hip extension as permitted →
   gentle hip adduction; tests ITB contracture – inability to
   adduct hip)
4. Allis test (Galleazi sign) – differentiate femoral and tibial
   shortening

Posterior aspect (prone):
1. Confirm and measure flexion deformity
2. Baker's cyst (most prominent in extension) and other
   swellings (aneurysm, lymphadenopathy, etc.)
3. Craig's test (Ryder method)

Manipulation and Special Tests

Tests for menisci:
1. McMurray's test (MM – fully flexed knee; external rotation
   at hind foot + varus stress → extend knee; LM – internal
   rotation + valgus; patient feels pain and examiner feels click
   (uncommon): pain in initial, middle and late extension
   "suggests" involvement of posterior, middle portion and
   anterior horn)
2. Apley's distraction-compression test – prone patient;
   stabilize thigh with your knee → pull leg up in $90°$ flexion
   → rotate foot internally and externally: painful → abandon
   test: no pain → perform in compression; medial joint line
   pain- MM tear and vice versa)
3. Cabot's maneuver – figure of "4" produces lateral joint line
   pain
4. Childress' test – in deep squat walk patient feels respective
   joint line pain in fully flexed knee in meniscal tear;
   retropatellar pain suggests patellofemoral arthritis
5. Passive extension test (Bounce home test) – respective joint
   line tenderness during forced extension of knee after
   maximal active knee extension.

Tests for stability:

- *Valgus and Varus laxity*
    1. Valgus and Varus stress test
        a. In extension – damage to MCL and postero-medial capsule (valgus stress); LCL and posterolateral ligament complex (varus stress)
        b. In 20º flexion – more specific for MCL/LCL
    2. Cabot's position
    3. Henri Dejour "frog position" – simultaneous evaluation of both knees' lateral structures.

- *Anterior laxity*
    1. Lachman's test and Trillat modification
    2. Anterior drawer test in neutral, internal and external rotation
    3. Pivot shift test of McIntosh and Galway
        a. Trace pivot shift "pivot glide" of Henri Dejour
        b. Dejour's test (pivot shift in extension)
        c. Hughston's jerk test
        d. Losse test
        e. Flexion-Rotation Drawer test

- *Posterior laxity*
    1. Godfrey's sign – posterior tibial sag in 90/90 position
    2. Muller's test
    3. Posterior drawer test and drop-back phenomenon
    4. Quadriceps active drawer test of Daniel

- *Posterolateral instability*:
    1. External rotation recurvatum test (varus recurvatum test)
    2. External rotation test
    3. Reverse pivot shift test

- *Anterolateral rotatory instability (ALRI)*
    1. Slocum's test

Measurements

- *Linear measurements*
    1. Apparent length

2. True length
3. Femoral and tibial length
4. Inter-malleolar (Genu valgum) and distance between two knees (Genu varum)

- *Circumferential measurements*
    1. Quadriceps wasting (15 cms from knee joint line; > 2 cms is significant)
- *Angular measurements*
    1. Varus and valgus at knee
    2. Q angle
    3. Tuber sulcus angle
- *Torsional measurements*
    1. Tibial torsion
    2. Femoral anteversion

Neurological and vascular status (At least offer to examine)
1. Palpate lower limb pulses (compare)
2. Sensory and motor distribution of tibial, common peroneal and femoral nerves
3. Reflexes (knee, ankle, tibialis posterior)

Test	Method	Interpretation	Grading	Comments
Lachman's test	20-30° flexion Hold femur with L hand; R hand holds tibia (thumb on tibial tuberosity- "Trillat") (compare) Watch end-point and anterior translation (Gerdy's tubercle/	Soft endpoint-ACL tear Firm with ↑ translation ↑ incomplete tear <2 mm translation + firm end point −ve test	Difference* from N <2 mm. >10mm consider MCL tear also. Grade I-"feel" of +ve test. Grade II-visible anterior translation. Grade III-passive subluxn of the tibia with the patient supine. Grade	First described by Torg. Sensitivity = 0.86 Specificity = 0.91 Best negative predictive value Overall better test to rule out and rule in ACL tear

Contd...

Contd...

Test	Method	Interpretation	Grading	Comments
	medial condyle)		IV-ability of the patient to actively sublux the proximal tibia	
Anterior drawer test	90° flexion Hold leg with both hands – thumbs on anterior tibial plateau. Index fingers check hamstrings. Watch anterior translation and end-point (compare)	-do- *External rotation* – ↑ – anteromedial rotatory instability, ? MCL tear *Internal rotation* – Normally decreased translation, ↑– ?Significance.	<2 mm- insignificant 2-5 mm = nearly N >5 mm* = abnormal *= Ensure PCL is not torn else false ↑ translation	Sensitivity = 0.2 Specificity = 0.88 Limitations: 1. Acute injury- painful 2. Door- stopper effect of meniscii 3.Hamstring spasm and checkrein effect 4. Normal ↑ laxity in 90° flexion
Pivot shift test	Internal rotation of leg + valgus at knee → flex knee. "Pivot" = Gerdy's tubercle "Axis"= PCL	Visible or palpable shift of Gerdy's tubercle (reduction of tibia) posteriorly at around 20-30˚ flexion	Dynamic test – "NO" grades. {you start from a posterior subluxed femur to reduce tibia over femur}	Sensitivity = 0.18-0.48 Specificity = 0.97-0.99 Best positive predictive value Limitations: 1. Flexion deformity 2. Acute injury

CASE I: RECURRENT
DISLOCATION OF PATELLA

Diagnosis

The patient is a 20-year-old female/male with recurrent/ habitual dislocation/subluxation of R/L patella with 5 cms circumferential wasting of thigh muscles. There is increased femoral-anteversion/tibial external torsion/patella alta/ tight lateral retinaculum.

Common positive findings:
- Standing:
  - Varus/valgus
  - Squinting/Frog-Eye patella
  - Patella alta
  - ↑ pronation of foot
- Sitting
  - Tracking
  - Lateral tilt (patella tilts down laterally)
- Supine
  - ↑ Q-Angle
  - Patellofemoral crepitus and effusion
  - Lateral tibial tubercle (+ ve Bayonet's sign)
  - Hypoplastic femoral condyle
  - Quadriceps atrophy
  - Patellar glide (> 2 quadrants laterally /or < 1 Quadrant medially- Sage sign)
  - ↓ patellar lift
  - Fairbank apprehension test
  - Ober's test (HDP)
  - Ely's test (HDP, Quadriceps contracture) *(See Chapter 2; Examination points; special tests; test 5)*

1. What is the difference between recurrent and habitual dislocation/subluxation of patella?

Ans.
- In recurrent dislocation of patella (RDP) there is tendency of patella to dislocate/subluxate (abnormal tracking) making patient "insecure". Patella does not always

dislocate/subluxate with flexion. In habitual dislocation of patella (HDP), patella comes off the joint with every flexion and patient is less bothered (habitual).

- HDP is a sub-group of chronic patellar dislocations (other one being permanent). Mostly recurrent dislocation follows traumatic event or is developmental (increased femoral anteversion or external tibial torsion) whereas habitual cases are most often than not due to congenital deficiencies or quadriceps myofibrosis.

- In RDP flexion occurs to full range without dislocation patella whereas in HDP patellar dislocation is "required" to complete flexion at knee joint so it is an example of "compensatory" pathology to pursue a physiological function.

- Secondary deformation of tibia (external torsion, valgus at knee) may develop in habitual dislocation of patella due to tight ITB/Quadriceps contracture while these are predisposing factors for RDP. Fixed extension contracture of knee may be a finding at birth in HDP.

- Recurrent dislocation/subluxation cases report to hospital (that's why kept in exam) usually during adolescence while many habitual cases present either during first decade or not at all till late (neglected – remember they take it as a habit!).

2. What factors may lead to recurrent patellar dislocation?

Ans. *Bony:* Patella Alta, patella breva, trochlear dysplasia (shallow trochlea), Genu valgum, Genu recurvatum, hypoplastic femoral condyle, external tibial torsion, increased femoral anteversion.

Soft Tissue: Vastus medialis obliqus hypoplasia, vastus lateralis hypertrophy, lax medial retinaculum, tight lateral retinaculum, deficient medial patellofemoral ligament, hypermobility syndrome.

HDP (Please see CASE III): Quadriceps contracture (multiple intra-muscular injections), hypoplasia of patella, femoral dysplasia/hypoplasia, ITB contracture.

3. What are the restraints for patella?

Ans. Static: Shape of patella, trochlea, retinaculum, patellofemoral distance, medial capsule, patella-femoral and patella-tibial ligaments.

Dynamic: vastus medialis obliqus (pulling patella medially at 50-55º).

4. What is Q-angle?

Ans. Q-angle (short for Quadriceps angle) represents the mean vector angle of quadriceps pull on patella. It represents the dynamic "instability" of patella – the greater it is the more unstable patella will be! First described by Brattstrom, it is clinically measured by intersection of lines formed between center of patella and ASIS and that between patellar center and tibial tuberosity (*in standing position*). Prerequisites are that patella should be centered in trochlea otherwise in an already dislocated patella angle will be falsely reduced (so measure it in 15-20° flexion of knee with relocated patella). Various factors outlined in Q2 can cause ↑ Q-Angle. N values (supine) 8-10° males and 15°±5° in females while in standing 14° for males and 17° for females is normal. Q-angle >8° in sitting position is abnormal. To remove the effect of femoral rotation tubercle-sulcus angle can be measured. In sitting position with knees flexed 90° intersection of line joining tibial tuberosity and center of patella and per-pendicular to patellar center gives tubercle-sulcus angle. N is <5° in males and <8° in females. An increase in this angle also signifies lateral shift of tibial tuberosity.

Increased Q-angle leads to patellar subluxation, Chondromalacia patellae, and excessive foot pronation.

5. What is J-sign?

Ans. Denotes one of the forms of abnormal dynamic patellar tracking (maltracking). Normally patella glides in a straight line with minimal sideways shift at the end of extension in the trochlear groove, however, due to various reasons (see above) patella may have excessive

lateral shift at the end describing an inverted "J" trajectory. Other forms of maltracking include subluxation (+ ve congruence angle even after 10° of flexion, Tilt (tilt angle <80°) and combination of above. Dynamic patellar tracking is judged in sitting position while patient extends knee from 90° flexion to full extension as above. Active patellar tracking is checked in full extension asking the patient to contract quadriceps and observing patellar shift which should be more superior than lateral.

6. Can you classify patellar instability?

Ans. Yes, it is based on two factors—(1) Patella Alta: Insall-Salvati index <1.3, and (2) Generalised joint laxity.
   • Grade I: (1) and (2) absent
   • Grade II: (1) absent (2) present
   • Grade III: (1) present (2) absent
   • Grade IV: (1) and (2) present

7. Now after clinical assessment what will you do and why?

Ans. I would like to get roentgenograms of involved and normal knees in antero-posterior, lateral, axial (infrapatellar/axial/skyline – like Merchant's view) views. AP view may show osteochondral fragment, loose bodies.
   Axial view demonstrates:
   – Tilt angle (angle between lateral patellar facet and femoral condyle – always open laterally minimum 8°)
   – Congruence angle (N = 6°).
   The lateral view demonstrates:
   – Trochlear flatness ("Y" sign or "crossing" sign where trochlear and lateral condylar shadow merge, N= parallel)
   – Trochlear convexity ("X" sign - lines cross)
   – Patellar height.

8. How do you access patellar height?

Ans. On lateral X-ray plate
   1. Insall-Salvati ratio = diagonal patellar length / patellar tendon length

2. Modified Insall-Salvati ratio = articular patellar length/ dist; from articular surface to tibial tuberosity
3. Blackburne peel ratio = articular patellar length/ distance from articular surface to tangent from tibial plateau
4. Blumensaat line: In 30° knee flexion line extending from intercondylar notch should touch lower pole of patella. Patella above this line – Alta, Below – Baja. Considered non-reproducible by many.

9. How will you manage acute injury?

Ans. *In acute stage:*
- PRICE (pain control, rest, ice, compression, elevation)
- In addition TENS
  In repair phase:
- Restore ROM, neuromuscular control, muscular strength, muscular endurance
  In maturation phase:
- Functional progression back to activity
- US, massage, joint mobilization, fascial stretching.

10. How will you manage this case?

Ans. The answer should be based upon assessment of:
1. Q-angle, Insall-Salvati ratio
2. Skeletal maturity, activity level (athlete)
3. Presence of associated pathology like osteochondral fracture, chondromalacia.

In general the simplest procedure that achieves realignment and patellar stability should be done.
- Recurrent subluxation, normal Q-angle, tight lateral structures – lateral retinacular release (arthroscopic or open)
- Above + medial laxity – Insall, Madigan
- Above but Q angle >20° - Roux-Goldthwait/ Galleazi (skeletally immature); Elmslie-Trillat (skeletally mature)
- Above + distal mal-alignment – Hughston, Modified Elmslie-Trillat

- Athlete requiring rapid resumption of activity – repair medial patellofemoral ligament and vastus medialis in addition to lateral release
- Patient with chondromalacia – Fulkerson type osteotomy
- Insall-Salvati ratio >1.2 – Simmons procedure.

11. Why do you see wasting in RDP?
Ans. Quadriceps inhibition.

CASE II: ANGULAR DEFORMITY OF KNEE (GENU VARUM AND GENU VALGUM)

Diagnosis

The patient is a 16-year-old male/ female with 17º Genu varum/ valgum deformity of R/L/Both knee(s) most probably secondary to "*etiology*" (idiopathic/rickets/epiphyseal dysplasia/ trauma/tumour/infection, etc). There is increased femoral anteversion (Genu varum) with 2 cms lengthening/shortening and 4 cms wasting of quadriceps muscle.

Common Findings

Standing:
- Genu varum/valgum
- External (valgum)/internal (in toeing) rotation of foot
- Patella subluxation/dislocation (lateral in valgum)
- Flat feet (valgum)

Gait:
- Abnormal foot progression angle
- Varus/Valgus thrust gait
- Stigmata of associated disease
  – Rickets
  – Dysplasia (acral shortening)
  – Cerebral palsy
  – Ligamentous laxity

Supine:
• Increased hip external rotation in extension and tibial in-torsion (varum) – measure thigh-foot angle. (For demonstration of deformities in supine position *remember* to rotate the limb so that patella faces ceiling – this unmasks various torsional deformities of bones and reveals true joint deformities and malalignment!)
• Ober's test

1. How do you define genu varum/valgum?
Ans. These are angular deformities (coronal malalignment) of lower limb; some centers include it under orthopaedic cosmetology. Valgum (knock-knees) or varum (bow-legs) refer specifically to abnormal coronal "alignment" in which the leg is shifted away from midline (Valgum-medial angulation) or towards it (Varum-lateral angulation). In a normal person standing with heels/knees touching each other, ASIS-center of patella-center of malleoli-second toe/web are in a straight line, deviation of this line to inside of knee is varus at knee and vice versa. Upto 1 cm separation can be allowed for soft tissue in obese patients.

2. How do you measure these?
Ans. Measured in standing position. Two methods:
1. Femoro-tibial alignment: Angle formed between lines joining ASIS to center of patella and line joining center of inter-malleolar line to center of patella. Subtract normal valgus from the measured alignment (7° for males, 8° for females >7 yrs) for a valgus malalignment and add the same for varus. Varum or Valgum is said to exist if the angle is outside two standard deviations from the normal for males and females for that age.
2. Measure the intermalleolar distance in cms (normal < 10 cms) using measuring tape for valgus, and distance between two knees for varus (normal < 8 cms). It can also be expressed in "finger-breadth" albeit crudely. For unilateral deformities measure from midline (plumb line from nape of neck).

The first method is more specific and reproducible, however, it is limited by age factor and charts are not readily available. Second method although crude but can be readily pursued to judge the progression of deformity. Also distance in cms is not absolute for all – same distance is much more significant for a short statured person.

3. How do you differentiate if the deformity is in femur or tibia?

Ans. Supine position in a relaxed patient, flex the knee fully (heel touching ischial tuberosity is normal alignment) –
- If the deformity disappears altogether, then it should have been in femur,
- If it persists it should be tibial,
- If it is partially corrected then it could be in both.

   This is because the non-weight bearing posterior femoral condyles are usually relatively spared (Heuter-Volkman law) of deformity (unless it is epi/meta-physeal dysplasia/trauma, etc). In full flexion the tibia is in articulation with posterior femoral condyles; if the deformity is tibial it persists however if femoral – it corrects. There is usually only a partial correction in dysplasia. Patient can also be made to squat to demonstrate this. This method is usually applicable for metabolic and some developmental causes only. Ideally it should be assessed radiologically.

4. What is physiological malalignment?

Ans. Child is born with genu varum (physiologic) which due to persistence of tight posterior capsule of hip (external rotation) and internal tibial torsion. This over-corrects to physiologic valgus by 24-36 months of ambulation. Valgus should correct to adult levels by 7 years of age. In females the valgus sometimes keeps correcting upto 16 years also. So varus should be pathological after 2-3 years of age and valgus after 11 years of age (> 2SD of normal physiologic alignment). {Heath CH. Staheli LT. Journal of Pediatric Orthopedics. 13(2):259-62, 1993 Mar-Apr}

5. What are the signs of rickets?

Ans.

1. Infant rickets symptoms:
    a. Deformed skulls
    b. Late-closing fontanelles
    c. Rib-breastbone joint enlargement
    d. Delayed milestones
2. Knobby enlargements on the ends of bones
3. Distorting pelvis under weight
4. Spinal curvature
5. Restlessness
6. Lack of sleep
7. Retarded growth
8. Mental retardation
9. Thin top of skull (craniotabes)
10. Thin back of skull
11. Bossing (frontal bossing)
12. Harrison groove
13. Beading where rib joins cartilage (rachitic rosary)
14. Bowed legs
15. Knock-knees
16. Weak muscles (floppy-baby syndrome, rickety myopathy)
17. Pot belly and widening of perineum
18. Deformed chest (pigeon chest)
19. Weak ribs
20. Abnormal teeth development
21. Tooth decay
22. Fragile bones if untreated
23. Fractures if untreated (especially greenstick fractures)
24. Double malleoli sign
25. Windswept deformity (Tackle deformity)
26. Tetany spasms.

6. What would you do next?

Ans. I would like to get an AP and lateral view of both lower limbs fully including femur and tibia in standing position. If not possible then I will get orthoscannogram/

computed scannogram of both lower limbs. (*Remember for genu varum radiographs are indicated only if child is short [dwarfism], asymmetric deformity, age >3 yrs, progressive deformity*).

7. What do you see on X-rays?

Ans. Look for

1. Confirmation of diagnosis
2. Site of disease
3. Degree of malalignment (magnitude in degrees–tibio-femoral angle, mechanical axis, and metaphyseal-diaphyseal angle for differentiating *tibia vara* from genu varum >16° = tibia vara)
4. Associated disorders and assessment of physes
   a. Signs of rickets:
      i. Rarefaction of the provisional zones of calcification
      ii. Irregularly frayed and cupping metaphysis
      iii. Deepened physes, rarefaction of the margins of the epiphyses
      iv. Diffuse rarefaction of the shafts with thinning of the cortex and coarsened texture
      v. In healing rickets – zones of provisional calcification become denser than the diaphysis.

8. What would you do to confirm the location of disease "or" what if the malalignment is not very clear on X-ray?

Ans. Measure CORA (center-of-rotation of angulation)
   This reveals:
   • Site "apex" of deformity (femoral/tibial/metaphyseal/disphyseal sometimes at joint line!)
   • Number of deformity in complex deformities/ both femoral and tibial involvement
   • Magnitude
   • Planning guide
   • Reveals additional deformities like bowing of bones and torsional deformities.

9. How will you manage this case?

Ans. Reply only after considering age, deformity magnitude and deformity site, activity of underlying disease process.

- *Age:* In general *varus* malalignment should be corrected ASA underlying disease is settled because varus would not correct itself with age and is deemed as a progressive deformity. Bracing for genu varum is not favoured but for tibia vara as special case bracing can be undertaken for children ≤ 3 yrs, Langenskiold's grade upto II (? III), medial physeal slope <50° and metaphyseal-diaphyseal angle between 10° and 15° (surgical treatment is indicated for grade ≥ III, failure of orthotic management {upto 1yr}, medial physeal angle > 60°, obese child, female gender, late onset tibia vara). Genu *valgum >15° in female >11 yrs and male >12 yrs should be corrected.* Osteotomy for genu valgum is indicated by some at age >12yrs female and >14yrs for male (Note - *The females mature early for skeletal age. This is partly in controversy to the finding that genu valgum may correct till 16 years of age (see above), but I am afraid, that's the way it is!*). (*Another query needs to be addressed – What is 2SD from normal. It is 5° but varies regionally. This means 7 or 8 + 5 = 12 or 13, but the guidelines of 15° takes into account the error during measurement and the grey zone of regio nal differences so that unnecessary surgeries are avoided*).

- Take into account the *growth spurts left* for the patient as deformity progresses rapidly during growth spurts. If patient can wait, then do it after both growth spurts are crossed. However, if deformity is severe and disabling or progressing rapidly then correction may be undertaken after explaining the parents and patient appropriately.

- *Deformity site:* Better assessed by CORA **–** do intervention ideally at the site "apex" revealed by CORA.

- *Deformity magnitude:* Severe deformities put limb at risk of shortening (closed osteotomy) and neurovascular damage (open osteotomy). Usually upto 25° of (mild) deformity correction is successfully accepted (≈ 2.5 cms shortening (closed) or 2.5 cms excursion for neurovascular

bundle (open)). More severe deformities (mode-rate – 25-40°, severe - >40°) can be treated by staged surgery or simultaneous femoral and tibial compensatory osteotomies (viz. open lateral femoral and closed medial tibial for genu valgum)/Ilizarov method.

- *Underlying disease:* If active, (viz. rickets) then first treat the disease then only operate as if done in active disease the intervention may fail (non-union) or the deformity may recur!

  The stages of healing rickets and their impact:

  1. Acute stage: The normal rounded physis is replaced by cloudy area with indistinct center(s) of ossification. Metaphysis is splayed and periosteum may be thickened.
  2. Second stage: Mottled, irregular epiphysis. Broader and ragged metaphysis. Periosteal thickening disappears.
  3. Third stage: Epiphyseal shadow is denser and regular in outline and a dense line appears at the metaphysis due to deposition of calcium.
  4. Fourth stage: Clearly defined bone and normal calcium content. Metaphysis may still be broadened.

  Any osteotomy or surgical procedure must **not** be done before **stage three** has appeared.

10. What are the advantages and disadvantages of your method?

Ans. Often the answer rests finally on open/closed wedge osteotomies. "Usually" but not always (you can always defend your answer – till you remain logical of course) it is deemed better to do a lateral procedure in femur and medial on tibial side (*remember to mention partial fibulectomy with your chosen open/closed tibial osteotomy!*).

Open wedge osteotomy:
Advantages:

1. Avoids shortening and hence a second procedure to correct limb lengths.
2. Open lateral femoral osteotomy avoids vascular complications associated with closed medial femoral osteotomy for genu valgum.

3. Open medial tibial osteotomy for genu varum averts the possible neurological complications of lateral closed osteotomy of tibia.

Disadvantages:
1. Need for graft with associated complications like non-union, graft displacement and graft site morbidity.
2. Need for fixation to hold the osteotomy in desired position and maintain correction.
3. Collapse of graft and losing correction.
4. Stretching neurovascular bundle and morbidity.
5. Delayed weight bearing.

Closed wedge method:
Advantages (apart from above):
1. Periosteal splint at apex of osteotomy is a good stabilizer
2. Compression of osteotomy site with weight bearing

Disadvantages:
1. Shortening
2. Medial femoral closing wedge osteotomy needs careful protection of vessels
3. Visualisation and fixation of lateral tibial closing wedge osteotomy is hindered by fibula and also requires protection of common peroneal nerve.

11. What precaution will you take before performing osteotomy and how will you fix your osteotomy?

Ans. Apart from taking into consideration as above, I will first perform derotation (as decided by intorsion or extorsion) and then decide the amount of wedge required for removal as derotation frequently reduces the amount of wedge required grossly. Plate and screw (DCP, LCDCP, LCP, Condylar blade plate, DCS, L-Plate), Puddu plate (has metallic wedges of varying sizes to hold osteotomy and prevent collapse – open osteotomy), Thick K-wires; Crossed Steinman pins and POP cast, External fixator, POP cast only. *(I am not sarcastic but please choose your option).*

12. What other osteotomies you know of and what are the complications?

Ans. Dome osteotomy (Barrel-Vault osteotomy - additional rotational component can be corrected, avoid bump at the site of osteotomy, minimal alteration of true limb length). Oblique osteotomy, proximal tibial osteotomy with physeal resection (tibia vara), intraepiphyseal osteotomy and elevation of medial tibial articular surface (tibia vara), progressive lateral opening osteotomy (genu valgum). Complications apart from above include infection, implant failure, non-union, loss of reduction, recurrence, chronic pain, compartment syndrome (prevent by doing simultaneous fasciotomy of anterior compartment).

13. Are there methods other than osteotomy?

Ans. Yes, hemi-epiphysiodesis, hemiphyseal stapling, Ilizarov ring fixation system "corticotomy" and gradual correction, callotasis (that may be done with an external fixator).

14. What are the disadvantages of hemiepiphysiodesis/ hemiepiphyseal stapling?

Ans. Hemiepiphysiodesis is a permanent method whereas hemi-epiphyseal stapling is a temporary measure. First of all it is not a corrective method rather it is a compensatory measure controlling the physiologically normal side! Disadvantages are complete arrest and hence production of opposite deformity, asymmetric physeal arrest producing complicated deformities, breakage/ extrusion and joint penetration of staples, overlying bursitis, second surgery for removal. They are suitable in only a small group of patients (in a narrow age-group range and mild to moderate deformity upto 15°). It can be done only in patients in whom growth potential exists and may have to be repeated or followed with other procedures (for under correction/ overcorrection due to

unexpected physeal fusion!). Results and correction are unpredictable; also there is risk of compensatory overgrowth and recurrence of deformity after removal of staples. Stapling is more often indicated in genu valgum as there are infrequent; if at all any associated rotational deformities of bones.

15. How much correction do you aim to achieve?

Ans. A tricky question! Ideal is to correct exactly the amount of deformity as has been measured which should be true for "all static" deformities. However, it is nice to understand that no amount of varus is acceptable at knee whereas to err on side of added valgus can be accepted (Remember- deformities anywhere in body in the direction of physiologic bow/angulation can be accepted and remodeling is possible). Further for progressive deformity like *tibia vara* where physeal defect can somewhat be countered by valgus, "over-correction" ($\approx 5°$) should be done, this also holds true for stapling. In general otherwise, under-correction of deformities is commonly practiced as any over-correction (so-called "mal-correction") will get magnified as the child grows, whereas magnification in the direction of original deformity is less disheartening to patient and parents and can be re-corrected!

16. What is pseudo valgum and varum?

Ans. Excessive femoral anteversion and compensatory tibial extorsion produces appearance of genu valgum. Femoral anteversion only or with tight posterior hip capsule without tibial compensation appears like genu varum in supine position.

17. What is hemichondrodiastasis?

Ans. Asymmetrical <u>physeal</u> lengthening ("distraction") by fixator to correct angular deformities.

18. What deformity (ies) does iliotibial band contracture produce?

Ans.
- *At lumbar spine:* Ipsilateral lumbar scoliosis, increased lumbar lordosis.
- *At pelvis:* Pelvic obliquity due to abduction contracture
- *At hip:* Flexion, abduction, external rotation (FABER)
- *At knee:* Genu valgum and knee flexion contracture
- At leg: External tibial torsion with or without knee subluxation
- *At Ankle and foot:* (secondary deformities) – talipes equino varus, heel varus
- *Whole leg:* Shortening (true), initially there may be apparent lengthening due to abduction contracture at hip and pelvic obliquity.

19. What is triple deformity of knee?

Ans. Classically described for tuberculosis of knee. However, it is also found in various long standing chronic diseases like rheumatoid arthritis and in ITB contracture.

Deformity components:
1. *Flexion deformity at knee* (due to chronic nature of diseases and synovial effusion knee is commonly kept in position of maximum joint space – 30° which persists to produce flexion deformity of knee).
2. *Posterolateral subluxation of tibia* (most of chronic destructive diseases joint subluxates laterally – reasons are not clear; one explanation could be that in rheumatoid disease which commonly affects females there is already more physiological valgus which leads to increased stresses over lateral joint compartment, other more plausible one is that in destructive diseases there is exaggeration of the physiological alignment – remember there is a physiological valgus and posterior tibial slope in a normal knee joint).
3. *External rotation of tibia over femoral condyles*– various reasons; quadriceps pull, popliteus action, ITB, etc.

Sometimes *quadruple deformity complex* is described where *genu valgum* is added as a component of deformity (however this is not a primary patho-mechanism and develops secondary to tibial subluxation).

CASE III: QUADRICEPS CONTRACTURE

Diagnosis

The patient is a 7-year-old male/female with quadriceps contracture of R/L side with restricted flexion, patella alta and patellar hypoplasia. There is associated subluxation of the knee (anterior!) and habitual subluxation of patella. Patient is unable/able to perform activities of daily living.

(**Note:** Arguments may be raised to present it as a case of habitual dislocation of patella but remember that habitual dislocation of patella is "secondary" to primary quadriceps contracture – ask from history if patellar subluxation developed later).

Common Findings

Presents in one of these forms (at birth – stiff extended knee, congenital recurvatum, congenital dislocation; as toddlers progressive painless loss of flexion at knee; later childhood – habitual dislocation of patella; in adults as painful knee due to habitual patellar dislocation and arthritis)

Standing

- Genu recurvatum
- ↑ lumbar lordosis, flexion at hip (to relax rectus)
- Posterior knee (femoral condylar) prominence (knee subluxation)
- Lateral patellar subluxation
- Patellar hypoplasia, patella alta
- Reduced knee creases
- Scars of previous surgery/ injections
- Wasting

Gait:
- Stiff knee gait
- Walk with internal rotation of leg
- Valgus thrust gait

Sitting:
- Dimple over thigh (tethering of quadriceps)
- Lateral position of tibial tuberosity and bayonet sign

Supine:
- ↓ knee flexion
- Habitual dislocation of patella
- Ober's test
- +ve Ely's test (prone)

1. What is the commonest cause of quadriceps contracture?

Ans. In children and adolescents acquired form due to multiple injections (antibiotics, tetanus anti-serum, etc) in quadriceps is the commonest cause. Other causes are infusions into thigh, idiopathic (congenital), surgical (e.g. plating for fracture femur), fracture femur and malunion/tethering of muscle, infection (osteomyelitis in which muscle has adhered to bone) and myonecrosis (post myositis). In adults post surgical intervention is the most common cause. It is a "progressive" disease in children with on-going fibrosis especially idiopathic forms. The muscle most commonly affected is vastus lateralis (in post-injection cases; idiopathic/congenital form - imtermedius). Vastus medialis is least involved. The congenital form arises out of still elusive pathology and is deemed to resemble sternocleidomastoid tumor of torticollis, contracture akin to those in clubfoot and sprengel shoulder, localized arthrogryposis congenita, Seddon's ellipsoidal infarct of VIC (however this has distinct etiopathology and pathogenesis). Natarajan (Kini Memorial Oration 1968) showed that Vastus imtermedius inherently has a precarious blood supply and additional infusions/injection into it may further jeopardize the vascularity and hence induce fibrosis due to injury.

2. How will you differentiate contracture of vastus lateralis/ intermedius from rectus femoris?

Ans. In both there is limitation of knee flexion range of motion however in rectus femoris contracture there is additional positive Ely's test and concomitant flexion at hip. It should be noted however that combined contractures are difficult to assess.

1. Vastus imtermedius only: ↓ knee flexion + genu recurvatum and hyperextension
2. Vastus lateralis only: 1 + genu valgum and lateral patellar subluxation
3. Rectus femoris only: 1 + +ve Ely's test + hip flexion!
4. Combined: 1+2+3+ quadriceps wasting (vasti) and cord like tightened rectus on flexion.
5. Gracilis contracture (uncommon): +ve Phelps test.

3. What are your differentials?

Ans. *Chronic dislocation of patella* (permanent (congenital) type – stiffness never precedes dislocation, presents at birth, permanent and irreducible, "Flexion" contracture, genetic and syndromic – Larsen, Down, AMC, Nail-patella syndrome, Rubinstein-Tyabi, Ellis-van Creveld syndrome and Diastrophic dysplasia, patellar hypo/aplasia), *Arthrogryposis multiplex congenita, congenital dislocation of knee* (present at birth, hereditary, >females (3 times), 1/ 3rd bilateral, round condyles, absent suprapatellar pouch, absent or hypoplastic cruciates, quadriceps contracture – acquired, hamstrings and ITB – subluxed anterior, associated abnormalities common: DDH, CTEV, AMC, Larsen, cleft lip and palette, imperforate anus, etc), patellar aplasia, congenital genu recurvatum, post-polio residual paralysis, congenital quadriceps hypoplasia (localized muscle dysplasia of unknown origin).

4. Where else can you find similar affections?

Ans. Post-injection contracture of Deltoid (Shanmu-gasundaram) and Gluteus maximus extension contracture of hip.

5. What will you do next?

Ans. I will get AP and lateral views of knee along with AP view of pelvis and femur are required. I will access and look for primary pathology (? infection, fracture) and secondary developments in joint (viz. patellar displacement, flattening of femoral condyles, anterior tibial subluxation of tibia, genu recurvatum, fragmentation of superior/inferior pole of patella).

6. How do you plan surgery?

Ans. Factors to consider (Thompson)

1. Whether rectus femoris is also involved
2. How well can this muscle be isolated during surgery
3. How well can the muscle be developed after surgery
   Aim for at least 0° to 90° of flexion ROM (critical arc) at knee joint.

Options:

- Only rectus femoris involved: Sasaki type rectus release.
- Early stage with no joint changes: Only proximal release (Sengupta)
- More extensive involvement: Thompson/Payr type quadricepsplasty[*]
- Genu recurvatum: Femoral osteotomy
- Arthritis and extensive involvement: Arthrodesis.
  [*]Simultaneous Patellectomy is indicated if the deep surface of patella is grossly involved.

7. What precautions will you take perioperatively?

Ans. Explain to the parents for sure that there would be some degrees of extensor lag following quadricepsplasty that may however resolve later (hence proximal release was devised). During surgery obtain as much hemostasis as possible to avoid re-contracture and hemarthrosis. Immobilisation should be done in flexion (90° (Sengupta), 50° less than that obtained on table (Thompson)) for 2-3 days followed quickly by continuous passive motion.

8. **How will you manage habitual dislocation of patella (HDP)?**

Ans. HDP conceptually differs from RDP. Managing HDP on lines of RDP will aggravate the disease. In HDP restricted flexion is hardly seen and hence is quietly accepted by patient. In early cases management of quadriceps contracture with/without medial retinacular plication and sartorius reinforcement may give success other wise in late cases with patellofemoral arthritis patellectomy and reconstruction of extensor mechanism may be undertaken. Restricting patella from dislocating prevents flexion also from occurring *(See Chapter 3; Case I; Q 1).*

CASE IV: EXOSTOSIS (OSTEOCHONDROMA *ICD 10: C40-C41; ICD-O: 9210/O*)

Diagnosis

The patient is a 14-year-old male with 4×3 cm pedunculated swelling over distal femur on medial aspect possibly solitary exostosis (if multiple then rephrase sentence with… multiple exostoses and do not use the term "possibly"). There is true shortening of 2 cms and genu valgum/varum (multiple form).

Common Findings

Inspection:
- Lump in metaphyseal region of bones
- Joint deformity
- Patellar subluxation
- Associated swellings
- Shortening

Palpation:
- Non-tender bony hard spherical/ovoid lump 4 x 3 cms arising from femoral metaphyseal region (metaphysis is a radiological term so better call it "distal femur"!) over medial aspect. The swelling is directed away from the knee joint. Surface is irregularly bosselated with distinct edge. Swelling

is arising from underlying bone and is pedunculated/sessile. Overlying skin is free from swelling. The swelling is non-compressible/reducible/pulsatile and there is no fluctuation. There is no abnormal mobility (fracture) and no distal neuro-vascular deficit.

- Examine patella.
- Examine other swellings

Movements of Adjacent Joint

Measure:
- Genu valgum
- Shortening
- Q-angle

1. Is this solitary or multiple?

Ans. Look for other swellings typically around shoulder, elbow, wrist, scapula (vertebral border), ankle, cristal border of ilium, neurocentral synchondroses of vertebrae and medial and lateral ends of clavicle.

2. Why do you think it is exostosis?

Ans.

- Hard painless lump present for long duration in a skeletally immature patient.
- The swelling is arising and located at the growing end of bone.
- Swelling is growing (directed) away from the growing end of bone (growing ends of bones are 'towards the knee and away from elbow')
- Characteristic findings and underlying bone is not showing clinical signs of involvement (tenderness/irregularity/scalloping).
- There are no signs of aggressive tumour.
- Swelling is pedunculated (not true for sessile ones).

3. What are the other names for this swelling?

Ans. (Solitary) Osteochondroma, biotrophic osteoma, osteocartilaginous exostosis; (Multiple) hereditary multiple

exostoses (HME), osteochondromatosis, diaphyseal aclasis (Sir Arthur Keith), Metaphyseal aclasis (Greig), dyschondroplasia, hereditary deforming chondrodysplasia.

4. Is this a true tumour of bone "or" what is the process of development "or" pathogenesis?

Ans. No – this is a developmental disturbance of bone growth (developmental enchondromatous hyperplasia) typically affecting bone remodelling (Keith) hence representing failure of normal "tubulation" of bone (Jansen). Tendency of disease is usually inherited (doubtful for solitary form) and present at birth but manifests itself only after 7-8 years of age during growth spurt. The growth of swelling stops with physeal closure (cartilage may grow to form sarcoma later but of-course!). It affects bones that grow by combined enchondral and membranous ossification. Bones growing 'only' by enchondral or only membranous ossification are very uncommonly involved. Exostoses arise at those parts where cartilage ossification comes to be surrounded by subperiosteal bone (i.e. at the growing ends). From irregularity of ossification, cartilage cells fail to ossify. Henceforth if central cartilaginous cells are involved they form enchondroma but if peripheral ones are involved they form exostoses.

Various theories have been advanced – isolation of islet of cartilage cells, defective anchorage of germinal cartilage cells, physical-stress theory (focal embryonal cells at tendon insertions converted into hyaline cartilage), clonal/ neoplastic theory (three loci isolated: *EXT* Genes for HME – *EXT1* is in **8**q23-q24, *EXT2* is on **11**p11-p12, and *EXT3* is on chromosome arm **19**p) {It is not mandatory to remember all these theories as hardly ever they have any impact on management – examiners also will never nail you for not remembering these; they are only mentioned here for comprehensiveness. You will not be forgotten however if you do not know the first paragraph!}.

5. If it is a defect of remodelling then why it does grow?

Ans. The inner surface of cartilage cap (hyaline cartilage resembling growth plate, normally <3 mm) is involved in enchondral ossification and is responsible for growth in size. Swelling arises from physeal plate but with time and longitudinal growth of bone is shifted to metaphysis.

6. What is the mode of inheritance?

Ans. HME is inherited as autosomal dominant trait with variable expressivity and high penetrance although sporadic forms are also known to occur; may also occur with metachondromatosis, Langer-Giedion syndrome, and trichorhinophalangeal syndrome type II (TRP II), and DEFECT 11 syndrome. Male : Female for HME = 1:1 and for solitary = 2:1. Boys are somehow more severely affected by the disease.

7. What is your differential diagnosis?

Ans. *For HME* – Trevor's disease☺ (dysplasia epiphysealis hemimelica), multiple epiphyseal dysplasia☺, dominant carpotarsal osteochondromatosis of Maroteaux, multiple enchondromatosis☺, bizarre parosteal osteochondromatous proliferation (BPOP) – the Nora lesion. *For solitary osteochondroma* – Organized subperiosteal hematoma☺, traumatic osteoma ('rider's bone☺' of adductor magnus, traumatic osteoma of brachialis), pelligrini-steida disease☺, parosteal osteosarcoma☺, myositis ossificans☺, non-ossifying fibroma, soft tissue ossifying lipoma, Pedunculated lesions hardly have any differentials, sessile lesions are often confused with above. [Again - we do not have perverted memories and cannot remember all of these; the common ones are marked with☺].

8. What will you do next "or" how will you confirm your diagnosis?

Ans. I will get the AP and lateral roentgenographic views of the extremity.

9. What would you see on X-ray?

Ans. *I would like to confirm my diagnosis:* The lesion may be sessile or pedunculated. Outgrowth usually arises from metaphysis of a long bone with stalk continuous with the cortex and oriented away from epiphysis. Outline is well demarcated. The medullary canal is typically continuous with the parent bone (important to differentiate from parosteal osteosarcoma). Sessile swellings arise as plateau like irregular swellings with variable (smooth to irregular) outline. Cartilage cap may form a bump in soft tissue shadow. *{Computed tomographic scans may be required for evaluation of axial lesions and detailed study. MRI may be helpful in demonstrating continuity of medulla with parent bone, cartilage cap, and malignant degeneration}*

10. What are the complications of exostoses "or" what are operative indications for exostosis?

Ans. These are also the usual presenting features apart from cosmetic deformity!

1. Mechanical
   a. Locking knee
   b. Muscular restriction
   c. Subluxation/dislocation of joints (proximal and distal radio-ulnar joint and distal tibio-fibular joint)
   d. Reduced or loss of movements
   e. Large pelvic osteochondromas may obstruct parturition!
2. Compression
   a. Musculotendinous
   b. Neurologic
      i. Paresthesias
      ii. Paralysis
   c. Vascular
      i. Vessel displacement
      ii. Stenosis/occlusion
      iii. Pseudoaneurysm

3. Growth
    a. Angular deformity (genu valgum, varum, manus varus, cubitus valgus/varus)
    b. Cosmetic deformity
4. Trauma
    a. Fracture
        i. Nonunion (fibrous)
        ii. Infarction of cartilage cap
        iii. Ischaemic necrosis
    b. Bursitis
5. Malignant degeneration
    a. Benign
        i. Chondroma
    b. Malignant
        i. Chondrosarcoma
        ii. Osteosarcoma
        iii. Malignant fibrous histiocytoma

11. When do you suspect malignant transformation in exostosis?

Ans. *Clinically:*
- Continued growth of lesion after skeletal maturity
- Sudden appearance of pain in adulthood
- Sudden enlargement of pre-existing lesion in an adult.

Radiologically:
- Stippled calcification or variable mineralization of cartilage cap
- Soft tissue mass in vicinity
- Loss of distinctive bony margin
- Cap size >1.5 cms measured by USG or MRI (only raises suspicion; there is no one-to-one relationship)

12. How does this tumor differ from *de novo* tumours?

Ans. Malignant degeneration (uncommon complication 0.5-50% in HME – lower value probably true, <1% in solitary form) is more common in central lesions (pelvis, scapula, ribs and vertebra). They have a better prognosis than *de novo* lesions and rarely ever metastasize.

13. How will you treat this case?

Ans. Frame your answer considering following facts:

- Majority of lesions are asymptomatic and incidental findings. Small lesions can be left alone and followed under surveillance. Avoid surgery unless necessary in skeletally immature patients where lesion is close to physis as there is risk of physeal injury.
- Prophylactic excision is indicated for lesions in vicinity of prominent vessels to prevent aneuryms formation and pelvic ones in females!
- Symptomatic or unsightly lesions and those with complication (see above) are excised extraperiosteally.
- Secondary deformities in limb (particularly in HME) require corrective osteotomies and/or reconstruction (distal and/or proximal radio-ulnar joints, distal tibio-fibular joint).

14. How do you excise an exostosis?

Ans. Resection of exostosis is done extraperiosteally (complete extraperiosteal resection), flush with the parent bone or by marking a window of normal bone at the base (wide marginal excision if malignant degeneration is suspected) along with overlying bursa. Some people prefer wide marginal excision in all cases.

15. What do you mean by extraperiosteal resection?

Ans. No attempt is made to elevate the periosteum or perichondrium and the lesion is resected *en masse*. Care should be taken not to cut through cartilage and in sessile lesions the cartilage at the exostosis–host bone junction should be removed. After excision examine the mass for completeness of excision and send for histopathology. Assessment of bone defect is made as in excising sessile lesions enough cortex may be removed to weaken the underlying bone that may require support in POP cast/ slab.

16. Why do these lesions recur?

Ans. Incomplete excision especially of cartilage cap is the commonest cause of recurrence. Abnormal cartilage remnants at the excised margins and also if an attempt is made to cut through cartilage during surgery or a biopsy is attempted from the lesion pre-operatively (core and trephine biopsies are not advised – do an excision biopsy as above straight away) are known causes of recurrence.

17. What are the techniques for deformity correction?

Ans. Underlying principles of deformity are same. For knee deformity please see CASE II.

Forearm deformities are predominantly due to growth abnormality of ulna; radius is less commonly deformed leading to ulnar subluxation of carpus (manus varus). Restriction of rotation is due to exostosis growing into interosseous membrane, sigmoid notch of radius and excessive bow of bones. Cubitus valgus may develop secondary to radial head dislocation.

Masada et al classified forearm with HME (Table 3.2)

Operative techniques for correction of deformity:

- Distal radius hemiphyseal stapling
- Radial head excision
- Differential forearm lengthening
- Derotational osteotomy of both bones.

Table 3.2: Classification of forearms with HME

Type	Ulna	Radius
I	Short, with distal osteochondromas	Bowed
IIa	Short, with distal osteochondromas	Radial head dislocated, with proximal osteochondromas
IIb	Short, with distal osteochondromas	Radial head dislocation
III	Relatively unaffected	Short, with distal osteochondromas

CASE V: POLIO KNEE

Diagnosis

The patient is an 11-year-old F/M with postpolio residual paralysis with genu recurvatum deformity of 30° at R/L/ (bilateral) knee for past 5 years and quadriceps weakness.

1. Why do you call it postpolio residual paralysis?

Ans.

- Flaccid paralysis
- Only motor involvement
- Not present since birth
- Involved muscle groups in a limb without preference to extensors/flexors or proximal/distal
- Non-progressive paralysis.

2. What are your differential diagnoses?

Ans.

- *Meningitis:*
  - Meningoencephalitis (mumps, coxsackie)
  - Pyogenic meningitis (modified by antibiotic chemotherapy)
  - Tubercular meningitis
- *Infective polyneuritis (Guillian-Barré-syndrome):* paresthesia common, bilateral symmetrical Paralysis, increased CSF protein
- *Myopathy:* Develops often during adolescence, specific muscle groups involved, progressive disorder.

3. How many types (serotypes) of polio virus you know of?

Ans. Three types (genre: enteroviridae, family: picor-naviridae, RNA (+) virus, isolated first by Karl Landsteiner)

- Type I: Mahoney (PV1) – most common
- Type II: Lansing (PV2) – least common, most commonly causes paralytic polio.

- Type III: Leon (PV3) – vaccine associated paralytic polio

Transmits by feco-oral route. Can infect only humans, higher primates, old world monkeys with CD155 Ag. Humans serve as reservoir. Infects upper and lower oropharynx, CNS (bulbar type, central brain stem nuclei), spinal type (ventral horn cells), gut, meninges. Immunity is heterogenic to all types (i.e. no cross-immunity – 2nd infection possible). Incubation period range 3-35 days.

4. How many types of polio infection you know "or" is paralytic polio a common form of disease?

Ans.

Form	Proportion of cases
Asymptomatic (abortive polio)	90-95%
Minor illness (abortive polio)	4-8%
Non-paralytic *aseptic meningitis*	1-2%
Paralytic poliomyelitis	0.1-0.5%
a. Spinal Polio	79% of paralytic cases
b. Bulbospinal Polio	19% of paralytic cases
c. Bulbar Polio	2% of paralytic cases
d. Encephalitis	Spastic paralysis, seizures

5. What are phases of symptomatic disease?

Ans. Three phases:

1. Acute phase (5 - 10 days): Stage of onset of paralysis usually 2 - 3 days after fever begins
   a. Preparalytic-fever, headache, neck rigidity, painful spasm, muscle tenderness
   b. Paralytic- if brainstem affected (bulbar polio) respiratory muscle paralysis

Progression ceases when fever settles, acute phase terminates 48 hrs after fever normally (may last up to 2 months)

2. Convalescent phase (may last 18 months): Spontaneous recovery –
   a. Aims to eliminate deforming tendency, restore ROM and train co-ordination, rebuild muscle power by hot packs, passive movements, positioning (spine – firm mattress or Dunlop pillo with intermitent prone position; lower limbs – knees in slight flexion and foot in splint; upper limb – felt sling), muscle re-education, stimulation for standing reflexes, pool therapy (Hubbard tank bath)
3. Chronic phase (residual paralysis) – aims to prevent or correct deformity.

6. What orthopaedic apparatus are used during convalescent phase and residual phase?

Ans. Apparatus is required to protect weak muscle, prevent deformity and support limb.

Upper limb
- Abduction shoulder splint: Now rarely given for deltoid paralysis and protection from effects of gravity and shoulder subluxation.
- Cock-up wrist splint
- Spinal brace for axial involvement (often severe cases)

Lower limb
- Below knee appliances: For deformity correction and prevention – bar on the side of deviation and strap at the angulation (For genu valgum: outside bar and inside T-strap)
- Weight bearing caliper.

7. What is the aim of orthopaedic procedures in residual stage of disease?

Ans.
1. Correction of soft tissue contracture
2. Improvement of function and prevention of deformity by tendon transfer and stabilization procedure

3. Correct limb length discrepancy
4. Eliminate external supports.

8. How do you grade muscle power?

Ans. MRC grading (Medical research council, Great Britain)
   0. Total paralysis
   1. Barely detectable contracture/ flicker
   2. Not enough power to act against gravity
   3. Strong enough to act against gravity
   4. Still stronger with activity against resistance (not normal)
      i. 4 (+) Good resistance but not normal
      ii. 4 (0) Resistance moderate
      iii. 4 (-) Resistance weak and easily overcome
   5. Full power.

9. What are the causes of genu recurvatum in polio knee?

Ans. It is a progressive deformity!

Two types:
   1 Caused by structural bony and articular changes following quadriceps paralysis
   2 Relaxation of soft tissues on posterior aspect of knee joint.

10. What are the differences in the two types?

Ans. In the *first type* primary pathology is in quadriceps but hamstrings and triceps sure are normal so there is loss of anterior soft tissue support with maintained posterior support. Gradually tibial condyles get elongated posteriorly with reversal of posterior tibial slope. Posterior bow of upper meta-diaphysis and subluxation then develops. This is due to patient walking with hyper-extension of knee using anatomical knee locking or hand to knee gait. The prognosis for correction of deformity is excellent and should aim at (Irwin):
   - Restoration of limb alignment (Irwin proximal tibial posterior closing wedge osteotomy, Storen modification of Campbell osteotomy, flexion femoral osteotomy of Mehta and Mukerjee (1991))

- Rectification of cause of deformity (anterior transfer of hamstring (biceps femoris and semitendinosus) tendons to strengthen quadriceps)

The *second type* of knee is due to relaxation of posterior capsular structures and weakness in calf and hamstring muscles. This type is often also associated with calcaneo-valgus foot deformity. The hamstrings often subluxate anteriorly and become extensors of knee. There may be flattening of femoral condyles. Prognosis is less certain after correction of this type as no muscles are available for transfer, underlying cause cannot be corrected and deformity can recur. Bracing for up to moderate deformities prevents progression but has to be discarded later. Perry outlined the principles for successful surgical correction:

1. Fibrous tissue mass must be of sufficient strength to withstand stretching force generated.
2. Healing tissues must be protected.
3. Alignment at ankle be at least neutral or should be achieved so before surgery.

'Triple tenodesis of knee' for paralytic genu recurvatum is often the only option for these patients.

11. How will you manage quadriceps paralysis?

Ans. Hamstring tendon transfer (semitendinosus and biceps femoris). As such the muscles available for transfer are hamstrings, tensor fasciae latae and sartorius but the latter are insufficient to replace quadriceps function.

12. Why do you transfer semitendinosus with biceps femoris?

Ans. To prevent lateral dislocation of patella

13. What are the prerequisites for this transfer "or" what will you look for before doing this transfer?

Ans. The power of hip flexors (to clear foot off ground) and abductors along with triceps surae (climbing stairs) must be adequate. There must not be any ankle equinus (to

prevent post-op hyperextension at knee). Flexion contracture at knee must be released before transfer.

14. How would you prevent post-op hyperextension from developing after tendon trans-fer?

Ans. Assuring adequate power in triceps surae pre-op, preventing immobilisation of knee in hyperextension, correcting talipes equinus before resuming weight bearing. After tendon transfer hyperextension should be prevented otherwise it becomes a progressive deformity and genu recurvatum ensues.

15. How do you treat flexion contracture of knee joint?

Ans. Flexion deformity (contracture) develops either due to tight ITB or due to powerful hamstrings in the setting of weak quadriceps. Various treatment options with respect to the severity of deformity and etiology are as follows:

– Only tight ITB: ITB release and lateral intermuscular septum division (Yount)

– Tight hamstrings (contractures 15-20°): posterior hamstring lengthening and capsulotomy or wedge plaster cast treatment

– Deformities up to 40°: Initial treatment in reverse dynamic traction then as above or femoral osteotomy {for combined ITB and Hamstring tightness})

– More severe deformities: Supracondylar extension osteotomy along with posterior release either in single stage (up to 70°) or in two stages (>70°); often 5-10° of recurvatum is aimed at.

16. What is flail knee?

Ans. When there is weakness of both extensors and flexors leading to instability in all directions the knee is called flail knee. For such knee no muscles are left that can stabilize the joint for day-to-day activities. Management involves using a long brace with lock or if preferable to a heavy labor–Arthrodesis of knee joint.

17. What will you do for bilateral flail knee?

Ans. Options are providing locking knee brace on both sides or better an arthrodesis on one side and locking knee brace on other. It is prudent to give the patient an "arthrodesis trial" by putting the limb in cylinder POP cast.

18. What is the role of total knee replacement for paralytic polio knee?

Ans. Typically indicated for pain relief for arthritic changes. Quadriceps strength of 3/5 is a prerequisite for this procedure as TKR does not allow hyperextension and patient may not be able to walk! Results are poor even for pain relief for quadriceps power <3/5. Total constrained prosthesis is often advised.

CASE VI: INJURY TO ANTERIOR CRUCIATE LIGAMENT (ACL)

1. What is your diagnosis?

Ans. Anterior instability of the knee joint due to ACL tear.

2. What are the points in favour of your diagnosis?

Ans. *Symptoms:*
- Pain
- Swelling
- Popping or snapping sensation
- Feeling of give way
- Instability.

Signs:
- Anterior drawer test positive
- Pivot shift test positive
- Lachman's test positive.

3. What is your differential diagnosis?

Ans. PCL tear: This can mimic ACL tear due to presence of false positive anterior drawer and reverse pivot shift test.

4. What is the cause of false negative drawer test?

Ans. The drawer test can falsely negative due to:
- Haemarthrosis and hamstring spasm in acute injury, knee cannot be flexed till 90°
- Door stopper effect of the posterior horn of medial meniscus.

5. How do you grade instability?

Ans. Grading of instability is done as follows:
- 1+ Joint surface separate 5 mm or less (forward subluxation of tibia under femur)
- 2+ Joint surface separate 6 to 10 mm.
- 3+ Joint surface separate > 10 mm

6. Which is the most sensitive test for diagnosis of ACL tear?

Ans. Lachman's test.

7. What are the advantages of Lachman's test?

Ans.
- Highly specific for ACL rupture
- Not hampered by posterior horn of meniscus
- Not hampered by haemarthrosis
- Less painful because the muscles are relaxed
- Not hampered by sprained or partially ruptured medial collateral ligament
- Performed in functional position of flexion of knee. Can be performed when there is a fracture close to knee.

8. What are the various modifications of anterior drawer test?

Ans. Weatherwax described a modified anterior drawer test in which the lower leg is supported in the examiner's axilla. It is relatively difficult to establish a specific position of tibial rotation with this technique, but anterior displacement is easily recognized.

The Noyes test can be performed from the same initial position without significantly changing the hand position. Varus and valgus laxity also can be tested by slightly adjusting the placement of the fingers.

Feagin recommends performing 90° drawer tests with the patient in the sitting position. Gravity pulls the tibia downward and helps to relax the muscles. The advantages claimed are that anterior displacement of the tibia can be more easily perceived and confirmed and that the rotational response of the proximal tibia (medial and lateral compartmental translation) also can be evaluated using this technique.

9. What is the significance of "end point" in stress testing?

Ans. There are two discernable end-points in stress testing for disruptions of ligaments about the knee:

1. "Hard" implying a firm, definite stop
2. "Soft or mushy" a less distinct and less sudden stop. Following are the types of end points and their interpretations:
   - Firm end point with haemarthrosis: Implies an acute partial rupture.
   - Firm end point without haemarthrosis: implies an old partial rupture or elongation.
   - Soft end point with haemarthrosis: Complete rupture.
   - Soft end point without haemarthrosis: Old complete rupture, acute complex ligamentous injury.

10. What is "door stopper" effect of meniscus and its role in diagnosis of ACL tear?

Ans. With knee flexed to 90° for classic anterior drawer sign, medial meniscus, being attached to tibia, abuts against acutely convex surface of medial femoral condyle and has "door-stopper" effect, hindering anterior translation of tibia. With knee extended however, the relatively flat weight-bearing surface of femur does not obstruct

forward motion of meniscus and tibia when anterior stress is applied.

11. What is the significance of performing anterior drawer test in doing in different degrees of rotations of tibia?

Ans.

1. 90° flexion with internal tibial rotation
   I. (Nil) Iliotibial tract and PCL intact. The test is not made positive by rupture of the ACL and posteromedial structures, because the internal rotation "locks" the joint by tightening the posterolateral ligaments, the iliotibial tract, and especially the PCL.
   II. (Slight): Rupture of ACL. Injury of arcuate complex and iliotibial tract, possible lesion of medial and posteromedial structures.
   III. (Marked): Rupture of ACL and PCL. Lateral and posterolateral structures, lesion of iliotibial tract.

2. 90° Flexion with neutral tibial rotation
   I. (Nil) Medial and lateral capsuloligamentous structures intact. ACL may be torn.
   II. (Slight): Lesion of medial and/or lateral structures. Possible rupture of ACL. With a firm end point, a PCL ligament rupture must be excluded.
   III. (Marked): Rupture of ACL and lesion of medial and posteromedial and/or lateral and posterolateral structures. Possible rupture of PCL

3. 90° Flexion with external tibial rotation
   I. (Nil) : Medial and posteromedial structures intact.
   II. (Slight): Rupture of medial and postero-medial structures.
   III. (Marked): Rupture of ACL, medial and posteromedial structures.

12. How do you classify knee instabilities?

Ans.

I. Single plane instability simple or straight.
   a. One plane medial

 b. One plane lateral
 c. One plane posterior
 d. One plane anterior
 II. Rotatory instability
   a. Anteromedial
   b. Anterolateral
     i. In flexion
     ii. Approaching extension
   c. Posterolateral
   d. Posteromedial
 III. Combined instability
   a. Anterolateral-anteromedial rotatory
   b. Anterolateral-posterolateral rotatory
   c. Anteromedial-posteromedial rotatory.

13. What is single plane instability and what is its significance?

Ans. Single plane instability is the one that is present only in single sagittal or coronal plane that can be tested by appropriate stress testing.

One-plane *medial instability with the knee in full extension* is apparent when, as the abduction or valgus stress test is performed, the knee joint opens on the medial side. This indicates disruption of the medial collateral ligament, the medial capsular ligament, the anterior cruciate ligament, the posterior oblique ligament, and the medial portion of the posterior capsule. One-plane medial instability detected in 30° of knee flexion indicates a tear predominantly of the medial compartment ligaments.

One-plane *lateral instability with the knee in extension* is apparent on adduction or varus stress testing when the knee opens on the lateral side; that is, the tibia moves away from the femur. This indicates disruption of the lateral capsular ligament, the lateral collateral ligament, the biceps tendon, the iliotibial band, the arcuate-popliteus complex, the popliteofibular ligament, the anterior cruciate ligament, and, often, the posterior cruciate ligament. One-

plane lateral instability detected only with the knee in 30° of flexion may be present in minor lateral complex tears or may be normal when compared with the opposite knee.

One-plane *posterior instability* is apparent when the tibia moves posteriorly on the femur during the posterior drawer test. This indicates disruption of the posterior cruciate ligament, the arcuate ligament complex (partial or complete), and the posterior oblique ligament complex (partial or complete).

One-plane *anterior instability* is present when the tibia moves forward on the femur during the anterior drawer test in neutral rotation. It indicates that disrupted structures include the anterior cruciate ligament, the lateral capsular ligament (partial or complete), and the medial capsular ligament (partial or complete).

The anterior drawer sign is positive in neutral rotation when the anterior cruciate ligament is disrupted with immediate or subsequent stretching of the medial and lateral capsular ligaments. In this type of instability the test becomes negative as the tibia is internally rotated because in this position the posterior cruciate ligament becomes taut.

14. What are the rotatory tests to diagnose ACL and other ligamentous disruptions?

Ans. Slocum Anterior Rotary Drawer test, Jerk test of Hughston and Losee, Lateral Pivot Shift Test of MacIntosh, Flexion Rotation Drawer Test of Noyes, External Rotation Recurvatum Test, Reverse Pivot Shift Sign of Jakob, Hassler, and Staeubli, Tibial External Rotation Test, Posterolateral Drawer Test are the rotatory tests to diagnosis ligamentous disruptions.

15. What investigations will you do?

Ans. X-rays may show bony avulsions of tibial spine.
MRI – Edema in ACL substance or avulsions from tibial or femoral ends.

16. What will you do to treat the patient?

Ans. I will do arthroscopic ACL reconstruction using quadrupled hamstring tendon graft. The treatment options available include non-operative management, repair of the anterior cruciate ligament, either isolated or with augmentation, and reconstruction with either autograft or allograft tissues or synthetics.

{Young athletic patients with complete ACL tear are treated with arthroscopic ACL reconstruction whereas old patients with partial ACL tear can be managed conservatively.in these two extremes the surgeon has to decide the treatment}.

17. What are the various auto grafts for ACL reconstruction?

Ans. The most common current graft choices are bone-patellar tendon–bone graft and the quadrupled hamstring tendon graft.

18. What are various graft options for ACL reconstruction?

Ans.

Autograft:

- Patellar tendon (bone-patellar tendon-bone)
- Hamstring tendon
- Semitendinosus
- Gracilis
- Central quadriceps
- Achilles tendon
- Multiple looped
- Fascia lata/Iliotibial band
- Meniscus!
- Reharvested patellar tendon

Allograft:

- Patellar tendon
- Hamstring
- Fascia lata/Iliotibial band
- Achilles tendon
- ACL

- Tibialis anterior
- Peroneal tendon

Synthetic:
- Goretex
- Dacron
- Carbon filaments
- Polyester

Engineered graft:
- Fabricated collagen.

19. What type of exercises are preferred in patients with ACL reconstructed knees rehabilitation and why?

Ans. Closed chain exercises

20. What are closed chain exercises and what is their rationale?

Ans. Resisted quadriceps exercises put strain on the anterior cruciate ligament, particularly in terminal extension if the limb is not bearing weight, these are called open-chain exercises (the foot is in air and the chain is hence open). In an effort to protect the graft (reconstructed ACL) during quadriceps exercises, it has been suggested that the foot be in contact with the couch (making the chain closed). The knee joint is thus so loaded that during movements the graft is protected from shearing stresses and perhaps the contours of the joint help stabilize the knee and protect the graft, these are also called as closed-chain exercises.

CASE VII: INJURY TO POSTERIOR CRUCIATE LIGAMENT

1. What is your diagnosis?

Ans. Posterior instability of the knee joint due to PCL tear.

2. What are the points in favour of your diagnosis?

Ans. *Symptoms:*
- Pain

- Swelling
- Popping or snapping sensation
- Feeling of give way
- Instability

Signs:
- Posterior drawer test positive
- Reverse pivot shift test positive
- Lachman's test positive
- Absence of normal proximal tibial step (1 cm) when traced along femoral condyles anteriorly
- Telltale signs as scar mark of injury over proximal leg
- Godfrey's sign (posterior sagging of tibia)

3. What is the differential diagnosis of PCL tear?

Ans. ACL tear can mimic PCL tear due to presence of false positive posterior drawer and pivot shift test.

4. What is the cause of false negative drawer test?

Ans. In acute injury with hemarthrosis and hamstring spasm, knee cannot be flexed till 90°.

5. What investigations will you do?

Ans. X-rays may show bony avulsions of tibial spine.
MRI- Edema in PCL substance or avulsions from tibial or femoral ends.

6. What will you do to treat the patient?

Ans. (*The reconstructive outcome may not be as productive for PCL as for ACL reconstruction hence the guidelines are quite different*)

Guidelines:

Acute PCL avulsions:
- Large bony fragment – treat with either arthroscopic or open screw fixation.
- Small fragment (non-fixable):
  - Posterior tibial translation <10 mm – quadriceps exercises and rehabilitation

- Posterior tibial translation >10 mm – PCL reconstruction
- Young athletic patients with complete PCL tear are treated with arthroscopic PCL recon-struction whereas old patients with partial PCL tear can be managed conservatively.

Chronic PCL avulsions/tear:

- Chronic posterolateral instability (+):
  - Get AP hip to ankle radiograph in extension
    - Normal alignment→ rehabilitate
    - Varus → valgus osteotomy → relief → rehabilitate otherwise do PCL reconstruction (if still symptomatic and degenerative changes absent) else consider arthroplasty (degenerated joint)
- Chronic pain an/or instability (>10-15 mm posterior displacement)
  - Quadriceps exercises and rehabilitate
    - Improvement → continue and watch for degenerative changes → PCL reconstruction if progresses.
    - No improvement→ follow as for 'varus' above.

7. How will you reconstruct PCL and what are various options?

Ans. The treatment options available include non-operative management, repair of the posterior cruciate ligament either isolated or with augmentation, and reconstruction with either autograft or allograft tissues or synthetics. *(See Chapter 3; Case VI; Q 18)*

Foot and Ankle

{The foot and ankle is a difficult topic for examination and in itself is a speciality topic. The cases are often short cases but long case of a polio foot can be presented.

Read times 3-5 times, 5-7 times for polio foot; MS and DNB candidates}

EXAMINATION POINTS FOR FOOT AND ANKLE CASES

HISTORY

1. *Age:* CTEV present since birth, TEV secondary to polio, neural tube defects, etc. appear later. CVT noticed at walking age-around 1 year.
2. *Sex:* Boys common in CTEV.
3. *Pain:* Duration, Site, radiation, type, character, aggravating factors, relieving factors, diurnal variation and postural variation.
4. *Swelling:* Duration, onset (preceding trauma, fever, other joints involvement, morning stiffness), progress (always increasing as in tumors, regressive as in trauma or increase with on and off reduction as in infection), aggravating factors (walking in subtalar arthritis), relieving factors (antibiotics in infection or chemotherapy in tumours), effect of any treatment received, diurnal and postural variation. Associated with deformity in other foot.
5. *Limp:* Onset, duration, painful or painless, progressive or not.
6. *Instability:* Duration, onset (post-traumatic), unilateral or bilateral (ligament laxity), on even or uneven surfaces (in stiff subtalar joint)
7. *Deformity:* Onset (at birth [CTEV] or appeared later [acquired clubfoot]) (appears at around 1 year in CVT) (after an episode of fever and myalgia with weakness of limb muscles in polio), progress (congenital is less progressive than acquired), any treatment received (casts, surgeries), response to any such treatment.
8. *Associated diseases:* Fever with myalgia and weakness of limbs in polio.

EXAMINATION

GENERAL EXAMINATION

Examine hip and spine for congenital hip dislocation, myelo-meningocele, spinal dysraphism, Arthrogryposis multiplex congenita.

LOCAL

Prerequisites

1. Patient must be sitting at edge of table with legs hanging freely.
2. Entire lower limb from lumbo-sacral spine to tips of toes must be examined.
3. Examine foot during gait, standing and in non-weight bearing position.
4. Neurological examination of lower limbs should be done as deficits produce different deformities of foot and toes.
5. Footwear examination.

Inspection

Gait

Antalgic, short limbed, foot drop, equinus, stiff 1st MTP joint.
Anterior aspect:
1. *Alignment:* Great toe (Hallux valgus/ varus), other toes (claw, hammer, mallet), relations of forefoot, midfoot, hindfoot w.r.t each other and lower leg (include tibia vara, rotation).
2. *Condition of skin:* Any discolouration, ulcers, dilated veins, edema (pitting or non-pitting and up to what level).
3. *Toes:* Notice transverse skin creases at I-P joints (sometimes lost in polio). Also note thickened cornified skin over dorsum (heloma durum) seen in toe deformities. Toe nail deformities in fungal infections. Paronychia (seen as swelling around base and sides of nail). Ingrown toenail.
4. Osteophytes medially over 1st MTP joint is called bunion and over lateral aspect of 5th MTP joint is called bunionette.

5. Tendons of EHL and EDL are visible over foot and anterior aspect of ankle by active contraction of muscles. (Remember pneumonic for structures medial to lateral is The Himalayas Are Not Dry Places-stands for tibialis anterior, extensor Hallucis longus, anterior tibial artery, anterior tibial nerve, extensor digitorum longus and Peroneus tertius).
6. Relation of medial and lateral malleoli: Normally lateral is below and posterior to medial malleolus.
7. Any swelling over malleoli: Seen in trauma, tendinitis.
8. Anterior crest of tibia and subcutaneous border may show swelling, deformities.

Lateral aspect
1. Visualize lateral malleolus, 5th metatarsal base, tendo-achilles and peroneus brevis tendon. Note for any swelling.

Posterior aspect
1. *Alignment:* Varus/valgus of hindfoot. "Too many toes sign" – more than 2 toes visible from behind means abduction of forefoot, usually associated with pes planus.
2. *Heel:* Size (any broadening), pattern and position.
3. Tell patient to stand on tips of toes (windlass effect-inversion and increased height of medial longitudinal arch)
4. Plantar fat pad, calcaneal tuberosity (abnormally increased prominence of superior aspect is Hagelund's deformity or pump-bump)
5. Retro-calcaneal bursa: Bursitis.
6. *Achilles tendon:* tendinitis, rupture (2-6 cms above insertion), swelling at level of malleoli is seen in tendonitis and over whole length is seen in rupture.
7. Calf atrophy (compared to normal): residuum of CTEV, TA rupture or prolonged immobilization.

Medial aspect
1. *Medial longitudinal arch:* Cavus or planus or rocker bottom deformity (in diabetics or improperly treated CTEV)
2. *Bony prominences:* Medial malleolus, head of 1st MT, calcaneal tuberosity and navicular tuberosity (prominent in accessory navicular).

3. Tibialis posterior tendon made visible by active contraction. Remember pneumonic for structures underneath flexor retinaculum of ankle: The Doctors Are Never Happy-stands for (from anterior to posterior aspect) tibialis posterior, flexor digitorum longus, posterior tibial artery, posterior tibial nerve and flexor Hallucis longus.

Plantar aspect
1. Callosity suggests point of weight bearing. Normally seen over metatarsal heads and lateral margin of foot. Painful calluses over MT heads are seen in toe deformities like claw toes and hammer toes with hyperextension of MTP joints.
2. Corns are localized thickening of skin over pressure areas. Two types: Hard or soft.
3. *Ulcerations:* Diabetes, abnormal bony prominences
4. Warts or fungal infections (tineasis)

Palpation

Anterior
1. Local rise of temperature
2. *Tenderness:* Over anterior tibial crest (in stress fractures). Over talar dome: Palpated anterolaterally with maximal passive plantar flexion at ankle (in OCD). Over navicular in Kohler's. Over talo-navicular joint in osteoarthritis. Also palpate cuneiforms, metatarsals (stress fracture esp. in 2nd and 3rd MT. Over 1st MTP joint in bunions, gout and septic arthritis). 2nd MTP joint (Freiberg's infarction).
3. Tenderness in interdigital spaces suggests Morton's neuromas (commonest b/w 3rd and 4th metatarsal heads).
4. *Swelling:* Over stress fractures. Osteophytes over joints. Effusion of joint: Cross fluctuation can be demonstrated between anterolateral and antero-medial swellings in full plantar flexion. Also seen between posterolateral and posteromedial swellings in full dorsiflexion. In between anterior and posterior swellings, it's seen in neutral position.
5. Tendons (whether they are taut, tenderness, lump or any gap seen in ruptures, diffuse swelling, crepitus): Tibialis anterior, EHL, EDL and peroneus tertius.

6. Toes palpated for corns, ingrown toe nails.
7. Tinel's sign over deep peroneal nerve (at site of dorsalis pedis artery) present in anterior tarsal tunnel syndrome.

Lateral
1. Lateral malleolus, anterior talo-fibular ligament and calcaneo-fibular ligament for swelling, tenderness.
2. Peroneal tendons (can't distinguish 2 separately)
3. Calcaneum, its tuberosity (in Sever's disease), Calcaneocuboid joint.
4. Over sinus tarsi in subtalar arthritis.
5. Over fibular shaft: Stress fractures.

Posterior
1. Over gastro-soleus. In Tendo-Achilles rupture, tenderness, gap and swelling are felt 2-6 cms above TA insertion.
2. Over posterior tuberosity of calcaneum: Tender swelling in retro-calcaneal bursitis

Medial
1. Medial malleolus and subcutaneous border of tibia
2. Head of talus(by eversion of foot)
3. *Navicular tuberosity:* Tender swelling seen in accessory navicular.
4. Tendons of FHL, FDL and T.P.
5. Tinel's signs over posterior tibial nerve and medial and lateral plantar nerves.

Plantar
1. Callosities-tender
2. Sesamoids for tenderness.
3. Plantar fascia-tenderness at calcaneal attachment in fasciitis, tenderness on hyperextending toes, painful nodules.
4. Plantar fat pad-tenderness,

Range of Motion

Ankle: Dorsiflexion (normal 20°) and plantar flexion (normal is 50°) – tested with forefoot in inversion and hindfoot neutral. Leg is held with one hand and foot is grasped such that head

of talus is gripped in hand to exclude any movement at the subtalar and midtarsal joints. If there is equinus deformity, assess passive dorsiflexion with knee extended and flexed *(In isolated gastrocnemius contracture, more dorsiflexion is possible with knee flexed as it relaxes muscle arising above knee. In isolated soleus contracture, knee position does not affect range of dorsiflexion. If both muscles are involved, slight increase in passive dorsiflexion is noted with knee flexed but still it is not within normal range).*

Subtalar joint: Inversion (normal is 40º) and eversion (normal is 20º). Examined with patient prone (hold dorsum of foot with one hand such that head of talus is stabilized between thumb and index, hold calcaneum with thumb and index of other hand and perform movements).

Forefoot: Abduction and adduction (normal is jog). With calcaneum stabilized in neutral position.

Great Toe: Extension (Normal is 70º) and flexion (normal is 45º) at MCP joint. Flexion (90º) and extension (0º - neutral) at IP joint.

Lesser toes: Flexion and extension (normal is 40º) at IP joint and at MCP joint (40º and 0º respectively). Also test for adduction (movement towards 2nd toe) and abduction of toes.

Test for muscles individually:
• Grossly ankle plantar flexors are tested by toe walking.
• Ankle dorsiflexors by heel walking.
• Evertors by walking on medial border.
• Invertors by walking on lateral border.

Measurements

1. Longitudinal: True and apparent length of whole limb, heel length (from tip of medial malleolus vertically down to point of heel), foot length both medial (back of heel to tip of great toe) and lateral (back of heel to tip of 5th toe).
2. Circumferential: At thigh, calf and foot (at height of medial longitudinal arch)
3. Broadening of ankle seen with calipers is seen in inferior tibio-fibular diastasis.

Distal neurovascular deficit:
- Palpate for anterior tibial (in between tendons of EHL and EDL), dorsalis pedis and posterior tibial (behind FDL 1 finger breadth behind medial malleolus) arteries.
- Complete neurological examination of lower limb.
- Sensory examination – Sural (lateral border of foot and ankle), deep peroneal (1st web space), superficial peroneal (dorsum of foot), saphenous (medial leg), posterior tibial (plantar aspect of heel) and digital nerves (adjacent sides of interspace).

Lymphadenopathy: Inguinal and popliteal

Special tests: (All done with leg hanging freely at edge of table)
1. *Anterior drawer test:* Grasp just above ankle with one hand and hold heel with the other. Gently pull heel forward with an internal rotatory movement to foot. Observe for amount of anterior translation and prominence of talar head anterolaterally. Difference of 3-5 mm in laxity between two sides with a soft end point or skin tenting anterolaterally by talar dome is significant. It tests anterior talo-fibular ligament.
2. *Inversion stress test (varus stress):* It tests calcaneo-fibular ligament. Maximally dorsiflex ankle and apply inversion stress to calcaneous. Abnormal inversion of talus at ankle (not movement at subtalar joint) compared to opposite side is significant (No definite numeric criteria).
3. *Peroneal tendon instability test:* Rotate ankle from maximal dorsiflexion to eversion to plantar flexion to inversion. Palpate posterior to lateral malleolus. If peroneal tendons subluxate or dislocate anterior to malleolus, suggests instability.
4. *Thomson's test*
5. *O Brien's needle test*
6. *First metatarsal rise test:* Done for tibialis posterior tendon. Patient is made to stand. From behind patient, rotate leg into external rotation. If 1st metatarsal rises off ground, it suggests tibialis posterior insufficiency. Normally, it remains in contact with ground. (The test can also be performed in

other way – Rose test: On dorsi-flexing great toe, tibia
rotates externally)

7. *Morton's test:* Compress 1st and 5th metatarsal heads
together. If a neuroma is present, he will complain of pain
in affected interspace.

8. *Homan's test:* Pain in calf on passive dorsiflexion of ankle
suggests DVT.

9. Pain behind heel on toe walking suggests pre-achilles
bursitis and on heel walking suggests post Achilles bursitis.
Pain on both suggests Achilles tendonitis.

CASE I:
CONGENITAL TALIPES
(L. TALUS=ANKLE; PES= FOOT) EQUINOVARUS

{The diagnosis is quite evident however one will be judged
based on the ability of ability to distinguish it from paralytic
conditions at least theoretically and clarity of approach towards
management.

Read 4-6 times (M.S. and DNB candidates)}

Diagnosis

The patient is a 4 year old male child with idiopathic neglected
clubfoot deformity of both feet without any associated spinal
dyraphism or syndromic association.

Findings

Foot

• Small foot; stretched thin skin on dorsolateral aspect and
thrown into creases along the medial aspect
• Scars and callosities (if patient is ambulatory)
• Head of talus palpable over foot
• Lateral convex border and medial concavity with furrows
• Heel ('small') rotated medially and drawn up (empty heel)
with deep crease over posterior aspect

- Ancillary findings:
  – Extrinsic/intrinsic type
  – Genu valgum

Gait: "stumbling" gait

ROM: ankle, knee, inversion and eversion at subtalar joint

Other examination (A very important question – What else would you like to examine?):

- Hip for DDH
- Spine for dysraphism (Meningomyelocele)
- Cerebral palsy
- AMC
- Polio – tight ITB (always check!)
- Cleft lip, palate, exomphalos and congenital hernia
- Sensation of foot
- Examine the power of gluteus maximus and quadriceps femoris in particular as weakness of above can lead to compensatory equinus at ankle.

D/D: congenital dislocation of ankle, tibial hemimelia

1. Why do you call it congenital?

Ans: The following tabulated points should help you answer this question!

Congenital clubfoot	*Acquired clubfoot*
Deformity since birth	*Naipe*
Bilateral (≈50%), 70% male	*Naipe*
Deep medial crease commonly present	*Naipe*
Small and wider foot	Often not
"Absent" heel	Maintained heel
Atrophied calf with cylindrical leg	General lower limb involvement with calf maintaining shape
Internal tibial torsion + t	Not present unless grossly neglected for long
Other congenital abnormalities may be present	Typical cause for deformity is evident
Neurological examination normal	Motor/sensory/combined deficit
Achilles attached medially	Not so (*Naipe*)

2. What are the deformities in clubfoot?

Ans: CTEV is a deformity in which foot is turned inwards to varying degrees with *[more precise but clinically impractical definition is – rotatory subluxation of Talocalcaneonavicular joint complex (otherwise called subtalar complex) with talus in plantar flexion and subtalar complex in medial rotation and inversion]:*

1. Equinus at ankle
2. Varus and internal rotation of heel (*varus of heel is equivalent to inversion at subtalar joint*)
3. Forefoot adduction with supination
4. Cavus of midfoot
5. Internal torsion of tibia (*this is controversial and probably secondary deformity – better avoid it unless pressed to answer!)*
6. Other:
   – Atrophy of calves and smaller circumference than other side
   – Smaller foot.

   {Please note that first three typically describe CTEV (Q: thence should be – What comprises CTEV) however if you are asked a specific question like deformities in clubfoot then answer all of above}

   Le Noir's proposed joint malalignment as the cause of deformities (ankle-equinus, subtalar-inversion, Chopart and Lisfranc-adduction)

3. How do you look for equinus, heel varus and adduction deformities?

Ans: The evaluation of deformities appears arbitrary in most of the texts. I am presenting the concepts from standard protocol podiatric examination that should be acceptable to many.

   Heel varus (frontal plane alignment of calcaneum): normal alignment of calcaneus is neutral – the line of attachment of tendo-achilles to center of heel is perpendicular to horizontal reference line. Look at the foot of child (preferably making him stand on ground) from behind. Assess the alignment as above. If the line

is directed inwards then it indicates heel varus. (*Note – heel varus/valgus is same as heel inversion/ eversion*)

Supination of foot: While observing the foot from behind two curved depressions concave outwards are obvious above and below the lateral malleolus. These curves are symmetrical in a normal foot. If the curve below the lateral malleolus is shallow/flat/convex outwards then it indicates supinated foot. Similarly exaggeration of curve indicates pronated foot.

Equinus: if patient is able to stand then make him stand on ground with knee in extension otherwise passively dorsiflex the foot to maximum possible. A normal patient will stand with plantigrade foot (ball of great toe and heel simultaneously touching the ground). In equinus deformity the patient will not be able to touch the heel or the heel and great toe ball are not in the same horizontal plane. For quantification measure the distance between center of heel and great toe, else measure the angle foot makes with perpendicular to long axis of foot. (Grading (*subjective – I : cannot walk on heel, II: smaller heel with appearance of cavus, III: splaying of forefoot with exaggeration of II, IV: clawing of toes with III*))

Forefoot adduction: While examining from behind, heel masks quite a substantial amount of foot (in front) and barely great (medial) and little (lateral) toe prominence are observable – which are equal on medial and lateral aspects. In forefoot adduction great toe is very prominently seen in totality and lateral aspect is empty.

Cavus: Observe both the medial longitudinal arch height and curve congruence. In pes cavus the arch becomes high and is acutely curved up posteriorly. One can measure the distance of floor to apex of arch to quantify the same. To make cavus more prominent one can ask the patient to stand on toes (cavus is exaggerated due to windlass effect – whereby due to forced dorsiflexion of MTP joint the plantar fascia is stretched).

To get an understanding of foot scoring one should read the FPI (foot posture index) – which is graded from -12 to +12. The relevance to CTEV is not there but the basics of observation can be understood.

4. What is supination and pronation of foot?

Ans: Supination is a combination of:
- Adduction at fore foot
- Internal rotation and plantar flexion at ankle
- Inversion at subtalar joint
- Medial arch elevation
  Pronation is a combination of:
- Forefoot abduction
- Hindfoot eversion
- Dorsi flexion at ankle
- Depression at medial arch

5. What is meant if equinus at ankle corrects with knee flexion but appears on extension?

Ans: It implies that the contracture is in the gastrocnemius component of gastrosoleus complex. If there is partial correction then either there is additional ankle stiffness or soleus contracture of lesser magnitude than that of gastrocnemius.

6. Can you tell the etiology of idiopathic CTEV?

Ans: *Still elusive*! Some theories are as follows:
1. Intrauterine packaging defect: (excessive packing – primi; large baby; oligohydramnios)
2. Neuromuscular defect (Isaacs): spina bifida; AMC
3. Fetal developmental arrest in fibular stage (Bohm)
4. Germ-plasm defect (Manufacturing defect!)
5. *Defective cartilage enlage of talus* (Irani Sterman)
6. Retracting fibrosis "Crimp" (Ippolito and Ponseti)
7. Anomalous tendon insertion (Inclan)
8. Myoblast in medial fascia (Zimny et al)
9. Heredity: polygenic multifactorial trait: 1:35 chances if sibling affected; 1:3 if other twin affected. Deletion of chromosome 2 (2q 31-33) related to CASP 10 gene (Heck et al), Edward's syndrome

10. Infective pathogens (Carney et al): enteroviruses – conflicting evidence
11. Electromagnetic radiation and toxins (maternal and/or paternal smoking, drugs, "ectasy" use during pregnancy)
12. Vascular theory: absent anterior tibial artery in patients or posterior tibial artery in parents!
    (I agree it's too much; and is always bad – to pass in exam concentrate and learn other things rather than wasting time to learn all of these – same for next Q!)

7. What syndromes are associated with CTEV?

Ans:

1. AMC	7. Diastrophic dwarfism
2. Streeter's dysplasia	8. Larsen syndrome
3. Prune belly syndrome	9. Down syndrome
4. Tibial hemimelia	10. Opitz symdrome
5. Mobius syndrome	11. Pierre-Robin syndrome
6. Freeman-Sheldon syndrome (whistling face)	12. Fetal alcohol syndrome

8. What are types of clubfoot?

Ans: Unexpected question! If asked at all:

Remember – primary (idiopathic) and secondary types (muscular type – AMC; osseous type – tibial hemimelia; neuromuscular; CP, polio, trauma etc) are actually "varieties" of clubfoot.

Clinical types (Kawashima and Uhtoff 1990) are as follows:

	Type I (Extrinsic, Non-rigid)	*Type II (Intrinsic/ Rigid)*
Foot	Normal size; mild varus	Smaller; marked varus
Heel	Normal size can be brought down with ease; minimal varus	Small; elevated; cannot be brought down with ease; marked varus
Creases	Normal	Deep medial, posterior and plantar creases(s)
Telescopy!	Absent	Present

Other types for severity classification viz. non-rigid/rigid/ teratologic and Goldner's subtypes are better unmentioned as they are not standardized and hardly used.

9. What are the aims of treatment?

Ans: Aim is to obtain a supple, plantigrade foot with good function

Objectives of correction are to correct the deformity early, correct deformity fully and develop the muscle power of the limb sufficiently to maintain correction.

10. What is the manipulative correction technique for clubfoot?

Ans: There "are" two popular manipulative techniques:
1. Kite and Lovell's
2. Ponseti's

It's just a matter of preference and comfort as to who uses which one! (I would prefer 2 in practice and especially exams!)

11. What is Kite's manipulative correction of clubfoot?

Ans: Generally described as sequential correction of deformities in the order <u>a</u>dduction of forefoot→<u>i</u>nversion at subtalar joint→<u>v</u>arus at heel→<u>e</u>quinus at ankle (remember A→I→V→E). However, if carefully read and followed kite's method also did simultaneous correction of deformities. Whatever – most importantly get the forefoot deformity corrected so that it points outward 20° followed by hindfoot deformity which is key stone to function of deformed foot and must be brought into a vertical plane BEFORE correcting ankle equinus. (Why?) – dorsiflexion to correct equinus before correcting inversion locks the subtalar joint decreasing the chances of further correction and the foot may break in midfoot region! – Rocker bottom deformity.

12. When to begin?

Ans: Day "1": manipulation by mother. Thumb rests on talus and press forefoot into abduction repeat at least six times

in a day. Correction achieved around talus (*Talus is least displaced but most deformed bone*). DO NOT UNTWIST – it increases cavus. After 6 weeks or according to institutional policy serial weekly/fortnightly casting in manipulated position should begin.

13. What defines the end of treatment?

Ans:
- No adduction/inversion deformity
- Hollow on dorsum of foot previously occupied by talar head.
- Passive movement to full calcaneovalgus position
- Child is able to evert and dorsiflex foot voluntarily to about right angle
- "Squat test"

14. What is Ponseti method of correction of clubfoot?

Ans: Manipulative casting technique to simultaneously correct the deformities beginning from pronation and correcting equinus in end. The concept of this technique is relaxation of collagen and atraumatic remodeling of joint surfaces. Simultaneous correction is achieved at talonavicular, calcaneocuboid and talocalcaneal joints. To begin, the "pronation twist" is corrected by supinating the forefoot to bring it in alignment with hindfoot. The tarsal bones distal to talus are then abducted in *supinated foot* so that navicular comes in front of talus and cuboid in front of calcaneous and calcaneous glides below talus into corrected position. Remember foot is not pronated (sort of untwisting) rather correction is achieved simultaneously by moving lisfranc joint (tarso-metatarsal), naviculo-cuneiform joint, chopart joint (mid-tarsal) and subtalar joint by above technique of abduction in foot in supination and equinus. Above knee casts are applied every 5-7 days. A total of about 70° of abduction is achieved before last cast is applied. Equinus is corrected at the end. Percutaneous Achilles tenotomy is done just before the application of last cast that heals in about 3 weeks. This

is followed by physiotherapy and immobilisation of foot in POP cast in corrected position (Pirani et al). To prevent relapse correction is maintained in foot abduction bar (three months following manipulation 14-16 hrs a day in "night and naps" till three years or as accessed by physician) with feet in 70° abduction and 15° dorsiflexion (without Dennis-Browne plates). For unilateral deformity keep normal foot in 30° outward rotation and neutral dorsiflexion.

Some say that the order of correction of deformities in Ponseti is C(cavus)→A(adduction)→V(varus)→E(equinus), but the method originally described corrected all of them simultaneously; even equinus which if residual was corrected with tenotomy.

15. What is accelerated Ponseti protocol?

Ans: Here casts are applied at 5 day interval and there is low threshold for Achilles tenotomy.

16. What is Kite's error?

Ans: Kite considered that the forefoot is in absolute adduction and emphasized that lateral deviation (abduction) of forefoot by putting pressure on calcaneo-cuboid joint laterally holding the heel will correct adduction deformity (Kite's error- this prevented abduction of calcaneum which is in adduction). However, actually the whole foot is in adduction with forefoot in relatively more adduction than hindfoot. This error was rectified in Ponseti method of manipulative correction where simultaneous abduction is done at Lisfranc, calcaneo-navicular, Chopart line and subtalar joint laterally putting pressure at talus head without touching heel. Total of 70° abduction is thus achieved.

Also heel varus does not correct by everting calcaneum – he did not realize that calcaneum will not evert if not laterally rotated.

17. How do you apply casts?

Ans: 2" roll for age <2 months and 3" rolls for age >2months. Use eversion tug principle and apply snugly fitting cast

with only sufficient cotton (not overstuffing cotton which presses out with time losing correction). Cast should not show your fingers and should be smooth enough. Casts should be applied up to groin with knee in 90° flexion to relax gastrosoleus complex and aid in equinus correction also it prevents cast from slipping out!

18. **Can you use any other method?**

Ans: "Strapping/Taping" in circumstances where cast correction is difficult/impossible viz. premature infant with multiple anomalies, monitored infant in intensive care – feet required for blood sampling or I.V drug infusions!

19. **What is spurious correction?**

Ans: Apparent correction without actual correction or development of unrelated deformities due to faulty manipulation:
- Rocker bottom foot
- Beanshaped foot
- Skewed foot
- Fractures
- Flat top talus!

20. **How will you manage this patient?**

Ans: I will get a true A-P and Lateral X-ray done to confirm diagnosis and for scoring foot (viz Dimeglio scoring and classification). A-P: {talocalcaneal angle = 20°-50° (normal), CTEV <20°}; {tarso-1st metatarsal angle – up to 30° valgus, varus angulation is seen in CTEV}. Lat:{talocalcaneal angle = 25°-50°, <25° in CTEV}.

I will correct the foot by Ponseti method of manipulative cast correction. Score foot by Pirani scoring:
Scores six clinical signs [A,B,C,D,E,F]

0- normal

0.5- moderately abnormal

1- Severely abnormal

Midfoot score (MS): Three signs comprise the MS, grading the amount of midfoot deformity between 0 and 3.

1. Curved lateral border [A]
2. Medial crease [B]
3. Talar head coverage [C]

Hindfoot score (HS): Three signs comprise the HS, grading the amount of hindfoot deformity between between 0 and 3.

1. Posterior crease [D]
2. Rigid equinus [E]
3. Empty heel [F]

Tenotomy will be done if HS >1 and MS<1 and head of talus is covered.

Treat as above and plot score on graph – roadmap of Ponseti treatment.

If deformity not corrected then soft tissue release will be required.

21. What is soft tissue release for clubfoot?

Ans: Indications include: neglected clubfoot (<4yrs), resistant clubfoot or deformity (ies), relapse/residual deformities.

Very infrequently required for Ponseti method of correction of clubfoot. Residual or resistant deformities can be corrected by specific releases (most commonly posterior for equinus).

Various releases are described for clubfoot and include posterior release, posteromedial release, extensile posteromedial release, combined posteromedial and posterolateral release, complete subtalar release etc. timing of release is an issue. French do release within few weeks to months of age capitalizing on the remodelling potential of growing foot. Turco however considers adequate time to be 1-2 years as the anatomical details are clear, under/over correction are not magnified as foot grows, better somatotypic assessment can be done and correction is maintained by ambulatory child. Simon's considers 8cm as good criteria and not age.

Soft tissue release must address all pathoanatomic structures:

Turco described one stage posteromedial release. Carol emphasized plantar fascial release and calcaneocuboid joint osteotomy as forefoot adduction and supination were not addressed by Turco. Goldner emphasized on correction of talar rotation by complete release of tibiotalar joint leaving subtalar joint to prevent valgus overcorrection. McKay and Simons described "complete subtalar release" which was actually a peritalar release with release of interosseous ligament and talonavicular and calcaneocuboid joints.

Posterior release only for persistent equinus; full posteromedial plantar and lateral release if all deformities persist.

22. What structures do you release in posteromedial soft tissue release (PMSTR) of McKay (modified)?

Ans: *Incisions:*
- Turco's: hockey stick posteromedial incision
- Cincinnati: circumferential incision
- Caroll's two incision technique: posteromedial and a small lateral incision over subtalar joint.

Medial release:
- Posterior and medial subtalar joint capsule (preserve interosseous ligament)
- Talo-navicular joint capsule
- Spring ligament
- Y-ligament
- Medial calcaneocuboid joint capsule
- Knot of henry
- Abductor hallucis
- Lengthening of posterior tibial tendon, FHL, FDL
- Plantar fascia, quadrates plantae origin

Posterior release:
- Ankle joint capsule
- Subtalar joint capsule
- Achilles tendon Z-lengthening
- Posterior talo-fibular ligament

Lateral release:
- Lateral subtalar joint capsule
- Peroneal tendon sheath
- Calcaneofibular ligament
- Lateral talo calcaneal ligament
- Extemsor digitorum brevis origin, calcaneocuboid ligament, inferior extensor retinaculum, cubonavicular ligament may be released in resistant cases.

Structures preserved: dorsal structures, medial neurovascular bundle, deep deltoid ligament, interosseous ligament.

Talo-navicular joint with subtalar joint is often fixed with smooth K-wires.

23. What are the complications of operative treatment?
Ans:
1. Neurovascular damage, bony damage, physeal damage, wound dehiscence
2. Undercorrection: most commonly due to inadequate post-op maintainance
   a. Equinus
   b. Heel varus
   c. Forefoot adductin
   d. Cavus
3. Overcorrection:
   a. Valgus overcorrection: caused by cutting interosseous ligament/aggressive casting. Treatment requires medial column shortening and lateral column lengthening and medial translation of talus.
   b. Forefoot abduction
   c. Calcaneous deformity
   d. Pes planus
4. Skew foot: forefoot adduction and hind foot valgus
5. AVN talus/navicular
6. Sinus tarsi syndrome
7. Dorsal navicular subluxation: produces a short cavovarus foot. Caused due to incomplete release or loss of talonavicular reduction. Treat by midfoot release with repeated plantar release and TA lengthening.

8. Dorsal bunion (Hallux flexus): Occurs due to loss of depressing strength of peroneus longs on 1st metatarsal as compared to dorsal long and short toe flexors with weak plantar flexion. Treat by 1st ray realignment and dorsal FHB transfer as toe extensor with MTP joint release.

24. What do you understand by terms neglected, resistant, recurrent, relapsed clubfoot?

Ans: *Neglected clubfoot:* when the patient does not receive treatment (conservative/operative) by walking age – typically 9 months

Recurrent clubfoot: When one or many or all deformities recur "during" the course of treatment- typically during manipulative correction, which was/were successfully corrected previously.

Relapsed clubfoot: When one or many or all deformities recur after successfully achieving correction of all deformities i.e. after end of treatment.

Resistant clubfoot: better called persistent clubfoot – when correction is not obtained in any or all of the deformities by manipulation/surgical methods (commonly due to inappropriate technique)

Most common **persistent deformities** are forefoot adduction and supination whereas true **recurrent deformity** is most commonly equinus.

25. What is the role of tendon transfer in clubfoot?

Ans: Indicated for poor foot positioning during walk or excessive inter foot progression angle or muscle imbalance. Commonly done either to correct evertor insufficiency or triceps surae insufficiency. Minimum age for tendon transfer is 5 years.

Anterior tibial tendon transfer: done for dynamic inversion/supination of midfoot when there is "relative" evertor insufficiency and patient bears weight on lateral

aspect of foot. *Split type {SPLATT – split anterior tibialis transfer}*: lateral arm rerouted from retinaculum subcutaneously to cuboid or lateral cuneiform. *Entire tendon*: inserted just lateral to midline in a comfortable tarsal bone – provides dorsiflexion and eversion without excessive abduction (power lost by 1 grade!)

Transfer for calcaneous gait (Triceps Surae insufficiency): overlengthening of TA is best prevented than treated – diagnose as early as possible. Peronei, tibialis posterior (TP) or long toe flexors can be used. Peroneus brevis split and rerouted into calcaneal tuberosity with tenodesis of distal stump to longus to prevent evertor insufficiency. Tib. Anterior transfer is NOT recommended as it leads to dorsiflexion paralysis and high stepping gait.

26. **What is the role of bony procedures and when will you do them?**

Ans: Please note that algorithms may differ from person to person. Simplified version can be put as follows:

Deformity	Age	Treatment
Metatarsus adductus	>5 yrs	Metatarsal osteotomy
Hindfoot varus	<2-3 yrs	Mod McKay procedure
	3-10 yrs	Dwyer osteotomy (heel varus) Dillwyn-Evans (if medial column is short) Litchblau (if lateral column is long)
	10-12 yrs	Triple arthrodesis
Equinus		Posterior release (mild-moderate deformity). Lambrinudi procedure (severe deformity). Excision of a portion of talar head/navicular. Distal tibial dorsiflexion osteotomy (salvage).

Contd...

Cavus	> 6yrs	Japas "V" osteotomy Akron mid-tarsal osteotomy (dome osteotomy) Trans-Midtarsal osteotomy (Köse et al)
All three deformities	>10 yrs	Triple arthrodesis
Persistent in-toeing gait	If persists for >2 yrs following correction	For a and b – supramalleolar osteotomy just proximal to distal tibial physis correcting 35° external rotation
a. True internal tibial intorsion		
b. Medial spin of hind foot in ankle mortice		For c – Evans/Litchblau.
c. Medial deviation of forefoot due to talar neck deviation		
Neglected clubfoot or secondary clubfoot	Cuneiform tarsectomy	Adult patients
	Talectomy	Patients with myelomeningocele – can be modified for patients with severe, resistant idiopathic clubfoot
	Wedge tarsectomy	Hardly ever done! Neglected clubfoot 8-11 yrs. Remove dorsolateral based wedge

Principles and brief description:

Older the patient more likely is the need for combined procedure

2-3 yr good candidate for modified McKay procedure

>5yr (some say >4yr) almost always require corrective osteotomies.

Dwyer osteotomy: Originally a medial opening wedge (taken from tibia) osteotomy – may increase equinus.

Modified to lateral closing wedge osteotomy that reduces wound healing problems, equinus, improves chances of union. Overall advantages – preserves subtalar motion, does not hinder with future procedure and can be combined with with other procedure.

Dillwyn-Evans (lateral column shortening): Originally a 4 stage procedure! (first 3 – soft tissue releases and 4th one – calcaneocuboid fusion after partial excision). Now done in single stage, may produce hindfoot stiffness in long run.

Litchblau procedure (lateral column shortening): essentially a calcaneocuboid arthroplasty (preferred for a too long lateral column). Excise distal part of anterior calcaneal process or take wedge from calcaneum – creating calcaneocuboid pseudoarthrosis. Less stiffness.

Triple arthrodesis: may be done as two stages (↓bone resection but ↑chances of AVN) or a single stage procedure (↓ AVN). Essentially a modified Lambrinudi type triple arthrodesis, here most of correction is done around calcaneus and not around talus (as in Lambrinudi). Aimed to correct deformity and maintain (>10 yr), it is the ultimate salvage procedure. Can be used for neglected clubfoot and varus/valgus overcorrected foot. Crucial NOT to undercorrect – slight valgus and pronation is favoured. Most difficult joint to fuse is talonavicular joint as stresses are concentrated at this joint and mobility is marked.

27. What is JESS fixator?

Ans: JESS – "Joshi External Stabilisation System" developed by Dr. B.B.Joshi (Mumbai). *Fractional, differential distraction* used to *sequentially* correct deformities. Distraction continued until approximately 20 degrees of dorsiflexion and overcorrection of the forefoot deformities was achieved. Maintained in this overcorrected position for twice as long as the distraction phase by casts/braces. May require soft tissue release simultaneously or before fixator application.

28. What is the role of Ilizarov fixator?

Ans: Uses the principle of *Distraction Histogenesis* to differentially correct the deformities. Correction slow enough to protect soft tissues; correction at the focus of deformity, *simultaneous* three-dimensional, multilevel correction; deformity correction without shortening the foot. Can be applied even in patients with failed previous procedures.

29. Do you know of any other non-operative treatment for clubfoot apart from Ponseti's?

Ans: Montpellier and Dimeglio method of adding CPM (Continuous passive motion) during earliest portion of treatment (usually 1st month of life).

Botox (botulinum toxin) injection into tendo Achilles

30. What are characteristics of CTEV shoes?

Ans: Robert Jones shoes are pronation shoes that have:
- Straight inner border to prevent forefoot adduction with medial containment bar.
- Outer shoe raise (out flare) to prevent foot inversion
- NO heel (high counter) to prevent equinus.
  They not only maintain correction and prevent relapse of deformities but may also correct mild residual deformities in a flexible foot.

31. Can you name some Indian authors associated with work in CTEV?

Ans: Dr. R. L. Mittal – local rotational skin flap for neglected clubfoot and extensive soft tissue release for posteromedial contracture.

Prof B Mukhopadhyaya – neglected clubfoot "Patna procedure"

Prof Duriaswamy – (SJH – insulin injection produced) induction of CTEV in chick embryo

Dr. B.B. Joshi – JESS

32. What is Dennis Browne splint?

Ans: Better called Dennis Browne bar aka abduction bar. It consists of a metal (original) or a polypropylene bar with

shoes attached to ends over foot plate (aluminum) rotated outwards of midline. The shoes are open-toe high top with straight medial border and the foot was strapped using adhesive tapes. Modified splints are made of polypropylene and are light, shoe are made of soft canvas with rigid medial border and are secured with Velcro straps. Problems with original splint were weight, pressure sores, injury to infants and parents and crib!

For further reading one may read Dimeglio classification

CASE II:
CONGENITAL VERTICAL TALUS

Diagnosis

The patient is a 1-year-old male child with bilateral rigid flatfoot most probably due to congenital vertical talus with foot size of 11 cms (medially and laterally) without any associated spinal or lower limb abnormality.

1. Why do you call it congenital vertical talus?
Ans.

History:
- Deformity in foot present since birth (often noticed when patient starts to walk)
- Toeing out when walking
- Difficulty to fit shoes

Examination:
- Severe uncorrectable equino-valgus deformity of hindfoot.
- Rocker bottom foot (loss of medial longitudinal arch with prominent rounded talar head as lowermost part of arch).
- Forefoot is abducted, pronated and dorsiflexed with fixed dorsal subluxation of navicular over talar head.
- Lateral toes are outward looking and everted.
- Soft tissues (tendons of tibialis anterior, long toe extensors and 3 peronei) on dorsolateral side of foot are contracted.

- Deep creases inferior and lateral to lateral malleolus.
- Tendo-Achilles contracture
- Callosities beneath anterior end of calcaneus and along medial border of foot superficial to talar head.
- Tendons of peroneus longus, brevis and tibialis posterior are tight and may come to lie anterior to malleoli (acting as dorsiflexors rather than plantar flexors).

Essential components of deformity:
– *Fixed equinus at ankle*
– *Fixed dorsal dislocation of navicular over talar head*)

  Always additionally examine spine (for meningo-myelocele, neurofibromatosis), hips and knees (equino-valgus may be compensatory to knee deformity!).

3. What are the various differential diagnoses that you will consider?

Ans.
1. Flexible flat foot
2. Inflammatory and infective foot disorders
3. Neurological like AMC and meningomyelocele
4. Compensatory: Tight Tendo-Achilles with/without equinus deformity, external rotational deformity of lower limb.

4. What will you do next?

Ans. I will do anterior and plantar flexion stress lateral views along with the routine PA and oblique projections.

5. What do you see on X-rays?

Ans. Calcaneum is in equinus. Talus points vertically downward with its long axis almost parallel to that of tibia. Navicular is dislocated dorsally over talar head.

In maximally plantarflexed (stress) view, navicular dislocation can be reduced in flexible flatfoot but not in CVT. Talus remains vertical. Both talar-1st metatarsal angle and tibio-talar angle do not correct.

In maximally dorsiflexed (stress) view, equinus at calcaneum is fixed.

I will also measure the various angles as below: Angles:

1. Lateral view:
   a. Increased talo-calcaneal angle
   b. Disrupted talar-first metatarsal angle
   c. Line along long axis of talus and calcaneum passes inferior to cuboid(normally crosses cuboid)
2. A-P view:
   a. Increased talar-1st metatarsal angle (shows marked forefoot abduction)
   b. Increased talo-calcaneal angle.

6. Why do you use 1st metatarsal for angles and not navicular, though navicular is the one dislocated?

Ans. Because navicular ossification centre appears at 3 years. Till then 1st metatarsal is taken for calculation.

7. What syndromes are associated with CVT?

Ans.

1. CDH, CDK,CTEV of opposite foot
2. Myelomeningocele, sacral agenesis, caudal regression syndrome
3. Spinal muscular atrophy
4. Neurofibromatosis
5. Trisomy 13-15,18
6. AMC
7. Prune belly syndrome
8. Marfan's
9. Multiple pterygium syndrome.

8. How do you classify CVT?

Ans. *Lichtblau types:*

I. Teratogenic: Associated with CDH, often bilateral with + family history, very rigid deformities.
II. *Neurogenic:* Myelomenigocele, neurofibromatosis: Less rigid.
III. *Acquired type:* Intra-uterine malposition.

9. What is oblique talus?

Ans. Condition where all deformities are present but navicular can be reduced over talar head in plantar flexion.

10. How do you treat CVT?

Ans. In initial stages, serial corrective casts are applied. Aim is not to correct the deformities (which is impossible), but to make skin and soft tissues supple. Forefoot is pulled into plantar flexion and inversion while calcaneus is dorsiflexed and heel cord stretched. Surgery is the definitive treatment.

11. When do you operate?

Ans. Surgery is to be done at the earliest possible time, preferably before 6 months of age.

12. What are the surgical modalities?

Ans. Modality used depends on age of presentation; however, the following four aspects are to be essentially followed:

1. Talo-navicular joint reduction-the main aim
2. Tendo-Achilles lengthening with/out capsulotomy of ankle and sub-talar joints. (Remember distal transverse cut in TA is made laterally in CVT because foot is in valgus while it is made medially in CTEV as foot is in varus).
3. Long toe extensors (EHL, EDL, peroneus tertius and tibialis anterior) lengthening with Z-plasty with calcaneo-cuboid reduction.
4. Dynamic sling of tibialis anterior through a drill hole in talar neck–to prevent plantar-flexion.

Depending upon age the various modalities are:

1. *1-4 years:* Open reduction and realignment of talonavicular, calcaneo-cuboid and subtalar joints. Navicular excision, partial talectomy and decancellation of tarsal bones may be required for reduction in children older than 3 years. Lateral column lengthening may need to be added to navicular excision.

2. *4-8 years:* Open reduction (as above), soft tissue release and extra-articular (Grice-Green) subtalar arthrodesis. May use intra-articular arthrodesis with screw fixation (Dennyson Fulford) as well. A gap of 6-8 weeks is usually given between release and arthrodesis. Some surgeons perform talonavicular reduction with subtalar bone-block in one stage followed 6-8 weeks later by posterior release and tibialis anterior sling.

3. More than 12 yrs: Triple arthrodesis.

Open reduction can be done in single stage or two-stages.

In one-stage surgery, use either Cincinnati incision or combined lateral (for tendo-Achilles and calcaneocuboid reduction) and medial incision (for talonavicular reduction).

{Kumar, Cowell and Ramsey technique uses 3 incisions, i.e., medial, lateral (over sinus tarsi) and posterior (for T.A.)}

In two-stage surgery, 1st stage consists of dorsolateral release, i.e., reduce forefoot over midfoot. Second stage is postero-lateral release i.e., reduce midfoot over hindfoot.

Reduction is held with threaded K wires across talonavicular and subtalar joints.

Attempt to reconstruct talonavicular ligament should be done. Long leg cast with knee flexed and joints reduced is applied for 2 months. Then wires are removed and another long leg cast is applied for 1 month. A short leg cast is continued for 1 more month after which an ankle-foot orthosis is applied for 3-6 months.

13. Which ligaments are released in the soft-tissue surgery?

Ans.

1. Talonavicular capsule
2. Tibio-navicular ligament
3. Interosseous talo-calcaneal ligament and superficial part of deltoid ligament
4. Calcaneo-cuboid
5. Calcaneo-fibular
6. Plantar calcaneo-navicular (spring).

14. What are common complications of CVT?
Ans.

1. AVN of talus- when operated.
2. Callosities in untreated/inadequately treated cases.
3. Residual feet pain.

CASE III:
POLIO AFFECTION OF
FOOT AND ANKLE

{Polio foot in particular is a difficult topic. Basic understanding of deformities and their cause is essential to initialize the viva session. The treatment of polio depends on deformities present. Always look for muscles that are active and suitable for transfer for the muscles weak or paralyzed}

Diagnosis

The patient is a 10-year-old male/female with post polio residual paralysis (PPRP) of left/right lower limb with equinovalgus deformity which is partly correctable with shortening without trophic ulcers.

1. Why do you say it's polio?
Ans.

1. Asymmetric, patchy, lower motor neuron type paralysis (AFP)
2. Non progressive
3. No sensory loss
4. Prior paralytic poliomyelitis with evidence of motor neuron loss, as confirmed by history of the acute paralytic illness, signs of residual weakness and atrophy of muscles on neuromuscular examination, and signs of nerve damage on electromyography (EMG).
5. A period of partial or complete functional recovery after acute paralytic poliomyelitis, followed by an interval (usually 15 years or more) of stable neuro-muscular function.

6. Symptoms that persist for at least a year.
(*Remember paralytic limbs are colder than their counterparts*)

2. What are the differentials?

Ans. Myopathy, neurological disorder or injury.

3. What is critical arc of motion?

Ans. 15° dorsiflexion (minimum required for heel strike and squatting) to 15° plantar flexion (minimum required for push off).

4. What is ideal age for tendon transfers and bony procedures in polio?

Ans. Wait for at least one and a half years after the paralytic attack, i.e., after convalescent stage has passed off and residual stage has started. The following principles apply:

– Tendon transfers in skeletally immature patients
– Extra-articular arthrodesis between 3-8 years
– Triple arthrodesis after 10-11 years
– Ankle arthrodesis after 18 years

Ideally tendon transfers should be done after 5 years when the child can be trained adequately for rehabilitation. Results are, however, better after age of 10-11 years. This should be supplemented with bony procedures (esp. arthrodesis) which should be done before or along with tendon transfers. Bony blocks to prevent opposite motion may be necessary with tendon transfers, esp. in children. This is because in pediatric patients, growth along with slight muscle imbalance even after tendon transfers causes recurrence of deformities. For example, if tendon is transferred to cause dorsiflexion of ankle, then posterior bony block to prevent plantar flexion may be done simultaneously.

First the plantar and dorsiflexion movements are to be balanced before inversion and eversion. Among the two, plantar flexion strength is to be regained first. If sufficient muscles are not available for transfer, then joints

are to be arthrodesed. For example, if invertors and evertors are not available, do triple arthrodesis so that only plantar and dorsiflexors are needed to be balanced.

It is better to be too tight than to be too loose when attaching tendons to new locations.

It is better to wait for 3 weeks in upper limbs and 4-6 weeks in lower limbs for physiotherapy to start after tendon transplants.

Surgical stabilization of joints (arthrodesis) is ideally done after skeletal maturity.

You should know the actions of groups of muscles namely tibialis anterior, tibialis posterior, long toe dorsiflexors, long toe plantar flexors and peronei. Transfer to midline if only plantar and dorsiflexion are to be balanced. If you need eversion, transfer it laterally and transfer medially if inversion is needed. For example, if you need to transfer a muscle to gain dorsiflexion and eversion, transfer it to dorsal tarsus (to gain dorsiflexion) and among the tarsal bones, laterally (to gain eversion).

Deformity progresses till bone and soft tissue growth ceases. Any splint/passive stretching cannot prevent development of a deformity. It can only slow down the process.

5. Do you prefer tendon transfers or excision of stronger muscles? Why?

Ans. Transfers are preferred because excision causes further atrophy of paralytic part. If excised, 7-10 cms of tendon is to be excised to prevent re-union by fibrous tissue.

6. Which is the commonest deformity in polio foot?

Ans. Most common is equinovalgus (tibialis anterior paralyzed) followed by calcaneocavovarus and then equinovarus. (Valgus is more common than varus).

7. What is the aim of treatment in foot surgery in polio?

Ans. Aim is to get a foot which is
   • Stable–no abnormal varus/valgus in hindfoot and ankle on weight bearing

- Plantigrade – even distribution of weight on hindfoot, midfoot and forefoot on weight bearing.
- No significant fixed deformity–without any inversion-eversion deformity, with foot to be able to be brought to right angle passively.
- Adequate muscle power– >3 power in active dorsi and plantar flexors of ankle, i.e., sufficient power to clear the toes off the ground.

8. What is Peabody's classification of polio foot?

Ans.

1. Limited extensor-invertor insufficiency
2. Gross extensor-invertor insufficiency
3. Evertor insufficiency
4. Triceps surae insufficiency.

9. How do you manage limited extensor-invertor insufficiency?

Ans. Weakness of tibialis anterior is the main feature producing equinus and cavus or planovalgus.

Treatment includes transfer of EHL (extensor hallucis longus) to base of 1st metatarsal along with plantar fasciotomy. It may be reinforced by peroneus tertius to same site. Plano valgus of long standing duration often becomes fixed may require triple arthrodesis. Fixed equinus may additionally require Tendo-Achilles lengthening.

10. What is gross extensor-invertor insufficiency and how to manage it?

Ans. There are two types of extensor-invertor insufficiencies:
   – Type A: Weakness of tibialis anterior and toe extensors (EHL and EDL) with a normal tibialis posterior
   - Type B: Weakness of tibialis anterior, toe extensors (EHL and EDL) and tibialis posterior.

Treatment for Type A:
   Deformity is equinus or equinovalgus.

Prefer transfer of peroneus longus to 1st cuneiform. Another option is peroneus brevis transfer that may be attached to dorsum of foot in midline (when TP and peroneus longus balance each other) or over 4th ray (when TP is strong) or 1st or 2nd ray (when peroneus longus is stronger than TP).

Treatment for Type B:

Transfer of both peroneus longus and brevis to dorsum of foot.

In both the above triple arthrodesis may be combined if bony deformities are fixed.

11. What is evertor insufficiency and how do you manage it?

Ans. Evertor insufficiency is characterized by weakness of peronei.

Treatment depends on extent of weakness:
  – Mild: EHL transfer to 5th metatarsal base.
  – Moderate: Tibialis anterior transfer to cuboid with EHL transfer to 1st metatarsal. Tibialis anterior may be transferred more medially if tibialis posterior is also weak so that it balances peroneal weakness.
  – Complete: Tibialis posterior to cuboid (anterolateral aspect) or lateral cuneiform

Triple arthrodesis may be combined with any of the above.

Split tibialis anterior transfer may be used as well.

12. How do you treat triceps surae insufficiency?

Ans. Earlier you treat this muscle imbalance, the better is the result.
  – Calcaneus deformity: Posterior transfer of tibialis anterior through interosseous membrane along with transfer of EHL to dorsum of foot, usually 1st metatarsal base. Most successful operation that restores heel to toe gait and tip toe walking.

– Calcaneovarus (peronei weak and strong TP – produces varus): Transfer both TP and flexor hallucis longus to calcaneus.
– Calcaneovalgus (TP weak with strong peronei): Transfer peronei to calcaneus.
– Calcaneocavus (both TP and peronei are strong and intact): Transfer both peronei and TP to calcaneus.

Triple arthrodesis is done if deformities are uncorrectable and associated with bony deformities.

13. What joints are fused in triple arthrodesis? What are commonly used types?

Ans.

– Aim is to reduce the number of joints that the paralyzed muscles have to stabilize.
– Angulatory or rotatory deformities of knee and leg should be corrected immediately after this procedure; otherwise there will be recurrence of deformities.
– The most important joint in the tarsus, determining the mobility of ankle and tarsal complex, is the talonavicular joint and hence any procedure should tackle this joint.
– A stable ankle with no varus – valgus instability is an absolute requirement. Or else, there may be recurrence of deformities or residual instability of ankle and foot complex. If anterior structures around ankle are found lax as in a forced plantar flexion X-ray, then either tendon transfers anteriorly to reinforce dorsiflexors or ankle arthrodesis should accompany triple arthrodesis. Other contraindications include unstable knee joint, painful OA of ankle, severe trophic ulcers and age less than 11 years.
– Ideal position of foot after triple arthrodesis:
  • Hindfoot in 50 valgus
  • Transverse tarsal joints in 0-50 abduction and forefoot in <100 varus
  • The medial border of foot should be straight

- The heel and 1st and 5th metatarsal heads should be in the same plantigrade plane and heel should be in exact mid position (Mild valgus will not harm but varus will definitely)
– Incisions include Kocher, Ollier and antero-lateral exposure to ankle with/ without accessory medial incision over talo-navicular joint.

Types:

1. *Hoke* (1921): Subtalar arthrodesis with resection, reshaping and re-implantation of the *head and neck of the talus* with *posterior displacement*.(Remember there is re-implantation of talar neck and posterior displacement of entire foot which is less than Dunn's type)

2. *Dunn* (1922): Subtalar arthrodesis with excision of *navicular and part of head and neck of talus* with *posterior displacement* which depends on the amount of bony resection. (remember navicular is completely excised and not re-implanted and posterior displacement is more than Hoke's and depends on amount of bony resection). Both Hoke and Dunn's types cause posterior displacement and hence increase lever arm posteriorly helping weakened plantar flexors of ankle. Both did not emphasize calcaneo-cuboid joint fusion initially. Both are especially useful with weakened plantar flexors of ankle.

3. *Ryerson* (1923): Arthrodesis of subtalar, talonavicular and *calcaneo-cuboid* joints. Correct hindfoot varus by bony cuts on either side of subtalar joint. Forefoot varus-valgus and abduction-adduction by cuts on talo-navicular and calcaneo-cuboid joints. This method is particularly useful when plantar and dorsiflexors of ankle balance each other.

4. *Lambrinudi*: Similar to Ryerson except that a break is created in tarsal bones into which reshaped talar head and neck is fitted to correct the forefoot equinus. Calcaneus remains in equinus at ankle, but equinus

deformity is corrected at subtalar joint. By locking the talus in equinus at subtalar joint and realigning rest of tarsus over talus, correction is achieved.

5. *Siffert, Forster and Nachamie*: Dorsal cortex of navicular is excised. Inferior part of talar head and neck is removed such that the superior part along with attached soft tissues anterior to ankle joint remains as a beak into which remaining navicular is locked. Stapling may be required to maintain this alignment.

Complications include: Pseudoarthosis of joints (most common complication and talo-navicular joint is most common joint), residual deformities like supination of forefoot and varus-valgus at hindfoot and midfoot, avascular necrosis of talus, varus-valgus imbalance at ankle, osteoarthritis of ankle, painful foot (pseudo-arthrosis and callosities).

14. What is the appearance of foot after triple arthrodesis?

Ans. Ideal foot after triple arthrodesis (Hoke and Thomson):
   – Looks natural in shoes
   – No external rotation on long axis of foot when standing or walking
   – No need for brace
   – Appears natural when bare
   – Weight is evenly distributed over plantar surface
   – Axis of ankle is at right angle to that of foot and well forward
   – No pain
   – Patient can control ankle joint motion.

15. Define hammer toe, claw toe and mallet toe.

Ans.
   1. Hammer toe is hyperextension of metatarsophalangeal (MTP) and distal interphalangeal joints (DIP) with hyperflexion of proximal interphalangeal (PIP) joint (usually involves single digit and associated with callus on dorsum of PIP joint).

2. Claw toe means hyperflexion at both interphalangeal joints with hyperextension at MTP joint (usually involves many toes and associated with callus on dorsum of PIP joint and tip of toe). The term claw toe is most likely derived from the affected toe's similarity in appearance to the claw of an animal or talon of a bird.

Deformity	MTP Joint	PIP Joint	DIP Joint
Hammer toe	Dorsiflexed or neutral	Plantar flexed	Neutral, hyper-extended, or plantar flexed
Claw toe	Dorsiflexed	Plantar flexed	Plantar flexed
Mallet toe	Neutral	Neutral	Plantar flexed
Curly toe	Neutral or plantar flexed	Plantar flexed	Plantar flexed

16. What are types of clawing?

Ans. *Two types:*

1. Swing phase: Due to weakness of ankle dorsiflexors, long toe extensors substitute for them. So during swing phase, when ankle dorsiflexors are to be active, long toe extensors contract producing clawing. It is more marked when Tendo-Achilles is contracted.
2. Stance phase: Due to weakness of triceps surae, long toe flexors are used to substitute them. So during push off phase of stance, when plantar flexors of ankle (triceps surae) are to be active, long toe extensors contract producing clawing.

17. How does clawing present?

Ans. *Common presentation of clawing:*

– Pain at the dorsal PIP joint from an impingement of the toe on the shoe.
– Callus or erythema over the dorsal PIP joint (friction with shoe).

– Patients may report pain at the tip of the toe (pressure against the point of the distal phalanx). There may be callus at the tip of the toe and a malformed nail.

– Pain from MTP joint due to synovitis (following persistent hyper-extended position and instability).

– Callus or soft corn on the medial border of the claw toe (seen in clawing of the fourth or fifth toe but is less common, impingement of the lateral claw toe on the adjacent toe).

– Metatarsalgia: Increased pressure beneath the metatarsal head and distal migration of the plantar fat pad with hyperextension of the MTP joint.

18. How do you treat clawing?

Ans. Foot deformities should be corrected before surgery for clawing, as clawing correction may be unnecessary after foot deformity correction. Add surgery for clawing if necessary after above. It depends on type of clawing:

– In swing phase clawing, first restore active dorsiflexion of ankle and correct equinus deformity. Add surgery for clawing if necessary after above.

– In stance phase clawing, restore active plantar flexion and correct cavus.

Indications: Pain due to clawing. (Contraindications: Poor vascularity to the toe and poor skin quality)

Splint used for post-op immobilization is Lambrinudi splint: It has felt pads on which plantar surfaces of toes rest and wires to hold onto the toes.

Surgical procedure:

Girdlestone-Taylor procedure: Transfer long toe flexors onto dorsal expansion of extensor tendons. This enables long toe extensors to act as intrinsic muscles of foot in producing active plantar flexion of MTP joint while extending IP joints. This surgery is more useful when clawing is primarily due to weakness of intrinsics of foot. (Swing phase).

19. What is the mechanism of great toe clawing?

Ans. Clawing of great toe is caused by insufficiency of dorsiflexors of ankle with normal long toe extensors (EHL) or long toe flexor (FHL) that try to overcome respective weak ankle dorsiflexor (tibialis anterior) or plantarflexor (triceps surae) and in the attempt produce secondary clawing of great toe.

20. How do you treat great toe clawing?

Ans. *Modified Jones procedure:* Done for clawing associated with Tendo-Achilles contracture. It consists of attachment of EHL into the 1st metatarsal neck and arthrodesis of inter-phalangeal joint. Distal part of EHL is attached to soft tissues over dorsum of the proximal phalanx.

Dickson-Diveley procedure: For clawing caused by insufficiency of plantar flexors of ankle and persists after appropriate foot stabilization and tendon transfers to restore active plantar flexion of ankle. It consists of three steps: Transfer of EHL to FHL around the medial side of 1st metatarsal head. Arthrodese interphalangeal joint and then suture distal part of EHL to soft tissues over proximal phalanx.

21. What is the difference between Jones and modified Jones procedure?

Ans. In modified Jones procedure:
- Two incisions are used
- IP joint arthrodesis is done
- Excision of tendon sheath.

22. What are advantages of modified Jones over Jones?

Ans.
1. Less chances of hypertrophic scar
2. Less chances of pseudo-arthrosis at interphalangeal joint.
3. Regeneration of EHL is less likely.

23. What is clawfoot and how to treat it?

Ans. Cavus of foot with clawing of toes due to intrinsic tightness is clawfoot (*Equinus of forefoot is called cavus*). It may be associated with severe ulcerations beneath metatarsal heads.

1. Mild and correctible: Metatarsal bar on shoes with metatarsal pad during day and a splint with metatarsal bar for use at night can be used.
2. Mild, not correctible:
   a. Peroneus longus to peroneus brevis transfer (Bentzon).
   b. Arthrodesis of IP joints of all toes.
3. Moderate:
   a. Steindler's fasciotomy
   b. Dwyer's calcaneal osteotomy
   c. Japas 'V' osteotomy of the tarsus
4. Severe:
   a. Anterior tarsal wedge resection
   b. Hoke/Dunn triple arthrodesis along with TP transfer to dorso-lateral tarsus.

24. What is Steindler's fasciotomy?

Ans. It is done for cavus deformity along with other procedures or as a single operation. The structures released are:
1. Plantar fascia
2. *Extra-periosteal* stripping of following muscles: (Avoid periosteal stripping to prevent bone formation that may cause pain on weight bearing)
   a. Abductor hallucis
   b. Flexor digitorum brevis
   c. Abductor digitorum brevis
3. Long plantar ligament
   If deformity is not corrected, insert Steinmann pin longitudinally into calcaneum from tip of heel. Apply corrective below knee cast.

25. What is Japas osteotomy? What are the advantages?

Ans. 'V' shaped osteotomy of tarsus often done to correct moderate cavus in children 6 yrs or older.

Advantages: No shortening, widening of foot. No bone is excised.

(Teaching note: Apex of V is proximal and at the highest point of cavus, usually within the navicular. One limb of V extends laterally through cuboid to lateral border of foot and the other through medial cuneiform to medial border. The proximal border of distal fragment of osteotomy is depressed plantarward while the metatarsal heads are elevated, thus lengthening the plantar surface of foot. Plantar fasciotomy should be done).

26. What is dorsal bunion?

Ans. It is a deformity where MTP joint of great toe is flexed with dorsiflexion of 1st metatarsal and plantar flexion of great toe. A small exostosis is formed on dorsum of 1st metatarsal head. MTP joint may even subluxate. The plantar part of joint capsule and flexor hallucis brevis may both contract.

Three types:

1. Weak peroneus longus with a strong tibialis anterior: Causes unopposed dorsiflexion of 1st metatarsal and cuneiform. Plantar flexion of great toe is secondary to create point of weight bearing. Most commonly due to improperly planned peroneus longus transfer away from its normal insertion.

2. Due to paralysis of all muscles controlling foot except triceps surae and long toe flexors which are strong. They become active in push-off phase causing active plantar flexion of toes including great toe. Flexor hallucis brevis adds to effect.

3. Along with hallux rigidus and flatfoot with rocker-bottom deformity.

27. How do you manage dorsal Bunion?

Ans. General guidelines:

– If bunion is flexible – correct muscle imbalance between ankle motors.

– If rigid – do 'Lapidus' procedure: Imbrication (double breasting) of dorsal capsule of the 1st metatarsophalangeal joint along with capsular release on plantar aspect. Transfer of tibialis anterior from 1st metatarsal to 2nd and 3rd cuneiform. Transfer of FHL to base of 1st metatarsal through a tunnel passing from plantar to dorsal aspect so that FHL now acts as a plantar flexor of 1st metatarsal. Plantar based wedge osteotomy of cuneiform-metatarsal with/without naviculo-cuneiform joints to correct dorsiflexion of 1st metatarsal.

28. How do you treat equinus deformity of ankle in polio?

Ans. When plantar flexors are stronger than dorsiflexors or contracture secondary to posture and gravity in a flail foot.

It is treated by Tendo-Achilles lengthening without posterior capsular release of ankle and subtalar joint. This should be accompanied by tendon transfers to restore active dorsiflexion of ankle and a surgery which is either of:

1. Campbell's posterior bone block: A bone block is constructed on posterior aspect of talus and calcaneum such that it will impinge on tibia to restrict plantar flexion of ankle.
2. Lambrinudi arthrodesis
3. Pantalar arthrodesis
4. Ankle joint arthrodesis.

29. What is pantalar arthrodesis? What are the indications?

Ans. Surgical fusion of ankle, subtalar, talo-navicular and calcaneo-cuboid joints (i.e., triple + ankle arthrodesis; *pan = whole)*. The foot is preferably fused in neutral or slight valgus at hindfoot, equinus at ankle (5° for males and 15° for females). Equinus may be adjusted according to shortening (increase for a shorter leg).

Indications:

1. Equinus or calcaneus with lateral instability of foot and whose leg and foot muscles are not strong enough to control foot and ankle when only foot is stabilized.
2. Recurrence after Lambrinudi or Campbell's procedure.

Contraindications:

1. Fixed flexion deformity.
2. Uncontrolled recurvatum deformity of knee- functioning hamstrings or triceps surae.

   It can be done in one stage (Steindler) or two-stage (Liebolt and King). In the latter, foot stabilization is done first by Hoke's triple arthrodesis followed by ankle arthrodesis.

30. How do you treat Talipes equino-varus?

Ans. Equino-varus occurs with weak peronei and (normal or weak) ankle dorsiflexors with comparatively stronger tibialis posterior, triceps surae. Removal of this strong TP is essential in management.

   Treatment depends on age of patient:

1. *Skeletally immature:* Braces (double bar, with inner iron and outer T strap with 90° ankle stop with footpiece attached to leg in external rotation), serial corrective casts, Tendo-Achilles lengthening with/without posterior capsular release of ankle/subtalar joint, Steindler's release.
2. *Skeletally mature:* Triple arthrodesis with/without Steindler's release. If deformity is not fully corrected, then 4-6 weeks later, Tendo-Achilles lengthening with Jone's transfer can be done.

   Both the above age groups should be treated with muscle balancing operations.

   • Commonest is anterior transfer of tibialis posterior (through interosseous membrane as by Ludloff or around medial surface of tibia as by Ober) onto anterolateral tarsus. If there are no functional ankle dorsiflexors for transfer, then combine triple

arthrodesis or posterior bone block simultaneously with TP transfer.
- If triceps is weak, transfer T.P. posteriorly to calcaneus.
- If T.P itself is weak, transfer tibialis anterior to midline dorsally or slightly lateral.
- Tibial external rotational deformities more than 30º should be corrected by de-rotational osteotomies of tibia and fibula.

31. How do you treat paralytic talipes cavovarus?

Ans. This deformity is due to strong tibialis posterior and long toe flexors or due to strong foot intrinsic muscles in an otherwise flail foot.

Treatment is by:
1. Excision of short toe flexors with a segment of plantar fascia along with motor branches of lateral plantar nerve.
2. Excision of motor branches of medial and lateral plantar nerves (Garceau and Brahms).

32. How do you treat equinovalgus?

Ans. This is due to weak tibialis anterior and/ or tibialis posterior associated with strong peronei and strong, contracted triceps surae. Forefoot goes into abduction and pronation.

Treatment depends on age:
1. *Skeletally immature:* Correction of deformity with
   a. Double bar brace with 90° ankle stop and inside T-strap, shoe with medial arch support and medial heel wedge.
   b. Wedging casts
   c. Tendo-Achilles lengthening may be required.
      Once correction is achieved, then extra-articular subtalar arthrodesis (Grice-Green) with anterior transfer of peroneus longus and brevis (4-6weeks later) is done. Tendo-Achilles lengthening may be required along with this to correct equinus. Ideal age is 4-12 years

In isolated tibialis anterior paralysis with valgus deformity, transfer peroneus longus or EDL to first cuneiform.

In isolated tibialis posterior paralysis: Peroneus longus/FDL/FHL/EHL is trans-ferred through sheath of tibialis posterior onto plantar aspect of navicular.

2. *Skeletally mature:* Triple arthrodesis with/without TA lengthening followed 4-6 weeks later by anterior transfer of peroneus longus and brevis tendons and Jones operation.

33. How to treat a calcaneus (calcaneo-cavus) deformity?

Ans. Weakened triceps surae along with normal ankle dorsiflexors causes calcaneo-cavus.

In skeletally immature patient: Ankle brace with posterior elastic strap or shoes with elevated and posteriorly extended heel can be used. Early tendon transfers are the rule.

In skeletally mature patient, plantar fasciotomy and triple arthrodesis (preferably Hoke's or Siffert's) should be done.

Tendon transfers are a rule. It should be done at earliest possible time in both the above age groups. Options include peronei and tibialis posterior. In addition, tibialis anterior can be transferred posteriorly into calcaneus if EHL and EDL are normal and are transferred to the proximal aspect of dorsum of foot. FHL can be transferred if above muscles are not strong enough.

When sufficient muscles are not available for transfer, pantalar arthrodesis is the option.

CASE IV:
TENDO-ACHILLES RUPTURE

{Getting a case with acute rupture is quite unlikely and one must prepare for neglected injuries. A theoretical knowledge of various causes and diagnoses will be legible to pass. One should have a clear idea of management.

Read 2-3 times (DNB candidates), M.S candidates are very unlikely to get this case in preference for other examination cases}

Diagnosis

The patient is a 47-year-old male with 3 months old neglected rupture of tendo-Achilles of left side.

Findings

History:
- Sudden onset pain over back of ankle associated with snap and inability to walk
- Acute onset swelling
- Previous history of minor trauma
- No treatment taken
- Labourer

Examination:
- Scar mark, ?tenderness, ?bruise (acute cases)
- Palpable gap/irregularity (old cases) on posterior aspect of lower leg in the region of tendo-Achilles
- Inability to toe-walk
- Positive:
    - Thompson-Simmonds calf squeeze test
    - O'Brien's needle test
    - Copeland Sphygmomanometer test
    - Matles knee flexion test
- Weakness of gastrocsoleus and painful plantar flexion (especially when resisted).

   1. How do you test for Tendo-Achilles discontinuity?
- **Ans:** Apart from history and local palpable gap and plantar flexion weakness, the special tests that help diagnose the discontinuity of tendo-achilles are:
    - Thompson-Simmonds-Doherty test: patient prone: Squeeze the calf muscle of patient – passive plantar flexion of foot demonstrates continuous tendon.

[*after 7 days (neglected cases) due to intervening scar formation the test may be falsely negative*]

- Needle test of O'Brien: Insert a hypodermic needle 10 cm above the insertion of tendo-Achilles so that its tip is just inside the tendon. Alternately plantar and dorsiflex foot. If the outer portion of needle points cranially on dorsiflexion the tendon is supposed to be intact

- Sphygmomanometer test: Wrap the cuff around calf region and inflate it to 100 mm Hg, if then on dorsiflexion of foot pressure rises to 140 mm Hg then it indicates intact tendon.

- Knee flexion test: With patient prone ask the patient to flex knee to 90°, neutral position or dorsiflexion of ankle suggests torn tendon.

- Reverse Silfverskiold test (not very popular): With knee in full extension (ankle dorsiflexion here is solely restricted by tendo-Achilles) measure the range of dorsiflexion at ankle (more on injured side compared to the normal side).

- Single leg heel raise test: Ask the patient to stand on injured leg with heel raised (not possible with torn Achilles tendon).

2. What are the fallacies of Thompson test?

Ans: A positive Thompson-Simmonds test is supposed to clinically demonstrate complete rupture; in particular dependent on integrity of soleal part of the tendon. O'Brien however demonstrated that rupture of gastrocnemius part also produces a positive test and hence described the needle test. Conversely also in cases where the gastrocnemius aponeurosis is separate from soleus (see Q3 Section 4e); the Thompson test may be falsely positive as the "squeeze" has predominant effect on gastrocnemius muscle belly rather that soleus! The treatment decisions are often based on whether tear is complete or partial and whether soleus is involved rendering Thompson test insufficient.

3. Why is 10 cm chosen as a point for O'Brien test?

Ans: The following reasons explain the rationale:

1. In type-I junction (gastrocnemius aponeurosis separate from soleus) the gastroc. Tendon joins the soleal tendon 12 cm proximal to insertion. In type-II junctions (gastroc. Inserting into soleus itself) the tendon is composite from beginning. In any case the tendon is a single unit some 10 cm proximal to insertion.

2. At the same level the plantaris tendon is comfortably medial to Achilles tendon while the sural nerve migrates laterally preventing false negative test or nerve injury respectively.

4. What is tennis leg?

Ans: Rupture of gastrocnemius musculotendinous junction is called tennis leg, other types of rupture are complete Achilles tendon rupture and partial tears.

5. Why is the tendo-Achilles called so?

Ans: According to the famous legend Achilles was the warrior of Homer's Iliad who was made invincible by his mother by immersing him in the river Styx holding his heel which remained untouched by water and hence vulnerable. This tendon being the strongest tendon and 'location' resemblance in paradox has been so labeled.

6. What is the blood supply of this tendon?

Ans: Three sources (deriving vascularity from both posterior tibial and lateral peroneal artery):

1. Musculotendinous junction
2. Surrounding connective tissue
3. Bone-tendon junction

The vascular supply is disputably precarious in mid-portion or 2-6 cm from insertion of tendon, and skin directly posterior to the tendon is relatively sparsely supplied.

7. Where does the rupture commonly occur and what are the causes of rupture?

Ans: The weak area of tendon is supposed to be 2-6 cm above insertion, where the vascularity is supposed to be precarious; however there is no site preference with trauma! Various causes listed are as follows:

1. Collagen disorders (genetic)
2. Inflammatory and autoimmune mechanisms
3. Degenerative and repetitive trauma
4. Drugs (corticosteroids, fluroquinolones)
5. Exercise induced hyperthermia within tendon
6. Mechanical theory (eccentric loading/sudden loading with incomplete synergism of agonist muscles/inefficient plantaris which is not able to maintain tension in tendo-Achilles)
7. Ischemic injury to tendon (age related, vasculitis and collagen disorders, torsional ischemia due to vasoconstriction of intratendinous vessels).

8. What are the various pathogenic mechanisms?

Ans: Various factors lead to development of weakness of this tendon that ultimately fails under load. The various pathological changes that have been proposed finally leading to weakness and hence rupture of tendon are tendinosis, paratendinitis, and paratendinitis with tendinosis. The term tendinosis describes various degenerative changes within tendon (hyaline, mucoid, myxoid, fatty, fibrofatty, etc) that may arise out of various above listed causes. Often tendinosis is not symptomatic and is realized only on rupture of tendon. Repetitive trauma or inflammatory conditions produce paratendinitis which may be later accompanied with tendinosis and is often painful before rupture.

9. What will you do next for this patient?

Ans: I will confirm the diagnosis (only if there are atypical findings on clinical examination) by radiological investigations.

10. What are the findings on plain X-ray?

Ans: Lateral projection shows:
- Loss of posterior border of Kager's triangle or complete disappearance (fat filled triangular space in front of tendo-Achilles) is suggestive of torn tendon.
- Toygar's sign involves measurement of angle of posterior skin-surface seen on lateral projection. With disappearance of triangle the angle increases to 130-150°.

11. What is the most specific and sensitive investigation to diagnose a tendon rupture?

Ans: MRI (contrast enhanced is better if infection is suspected).

12. How will you manage this patient?

Ans: After confirming complete rupture of Achilles tendon a symptomatic patient with disability needs surgical reconstruction. I will mobilize the gastrocnemius and soleal components of the complex and after flexion of knee and plantar flexing the ankle, try to approximate the ends without tension and do an augmentation with plantaris tendon (or turn down of two flaps of gastrocnemius aponeurosis). If the ends cannot be approximated then I will use the peroneus brevis dynamic tendon transfer (rather 'splint') of White and Kraynick for reconstruction of tendon.

13. Are there any guidelines for the treatment of chronic Achilles tendon rupture?

Ans: Classifications exist but are not popular and couture treatment is often practiced:
1. Myerson's classification:
   a. Type-1 defect: 1-2 cm long → End to end repair and posterior compartment fasciotomy.
   b. Type-2 defect: 2-5 cm → V-Y lengthening with or without tendon transfer.
   c. Type-3 defect: > 5 cm → Tendon transfer alone or combined with V-Y advancement and augmentation.

2. Kuwada's classification:
   a. Type-I: Partial tear → Conservative management
   b. Type-II: Complete tear → 3 cm defect → end to end repair
   c. Type-III: 3-6 cm defect → debride + tendon transfer ± augmentation
   d. Type-IV: > 6 cm defect → debride + tendon graft ± augmentation

   (Confused! Better not be – leave apart others; the authors themselves did not evaluate utility of the classifications. Be assured you will not be asked these unless you are getting Gold medal!)

14. How do you manage acute tears?

Ans: Simply if the tendon ends can be approximated then I will use an end-to-end suturing with modified Kessler method and plantaris augmentation. If the ends cannot be approximated then I will use complex reinforcement method such as Lindholm's technique with turn down gastrocnemius aponeurotic flap with plantaris augmentation. Postoperatively I will immobilize the repair in above knee cast in flexion at knee and plantar flexion at ankle. After two weeks sutures are removed and further immobilization in below-knee cast is done which is followed by gradual dorsiflexion of ankle over further two weeks and protected mobilization.

15. What other methods are available for repair of tendon?

Ans: Fascial reinforcement, artificial tendon implants, marlex and collagen prostheses.

16. Do you know of any other method of repair?

Ans: Percutaneous method of Ma and Griffith.

17. What is the role of non-operative treatment?

Ans: Constant fight between supporters of non-operative vs. operative treatment remains unabated. With more recent publications the disparity has more clouded. It is for a surgeon's preference and to his experience to decide

between the two. A combined conservative and orthotic regime has recently been reported with excellent results. These methods are based on the premises of not injuring the already precarious blood supply to tendon through anterior mesentery and paratenon and are definitely (and should be) used for partial tears.

18. Do you know any popular surgeon who suffered from Achilles tendon rupture?

Ans: John Hunter in 1767 suffered complete tear while dancing! He treated himself with calf bandaging and raising shoe heel with excellent results.

19. Why is re-rupture common after repair or reconstruction?

Ans: Native Achilles tendon is rich in Type-I collagen while the healing tendon has high percentage of weaker and poorly organized type-III collagen less resistant to stretch.

20. What factors determine good healing of tendon?

Ans: Preservation of paratenon, good approximation, prolonged immobilization in equinus position are important for good healing of tendon.

The Shoulder

{For most of the candidates the shoulder joint will be an unexpected case and for this reason most are not required to know a lot about the same. An unfortunate DNB candidate is but 'at risk' of getting one and will be expected to know at least some important points. What follows gives a very comprehensive examination schema of the joint (more than enough for all) and a brief review of concepts for cases.

Read: 6-10 times! (DNB candidates, MS candidates may either skip or read 2-3 times). This is a difficult case (difficult both to understand and present especially the unstable shoulder!!}

EXAMINATION POINTS FOR A SHOULDER CASE

History Taking

1. Pain:
   a. *Onset:* Acute (infective, traumatic); insidious (inflammatory, sub-acute and chronic infections like TB).
   b. *Duration:* Protracted course in inflammatory process and adhesive capsulitis, TB.
   c. *Radiation:* To back of shoulder, axilla, outer aspect of upper arm.
   d. *Aggravating factors:* Movements aggravate most of the painful conditions.
   e. *Character:* Throbbing severe in pyogenic infections and traumatic conditions.
   f. *Relieving factors:* Rest, massage, analgesics (duration of relief, complete and incomplete relief should be specifically asked).
   g. *Relation to trauma:* Dislocations, fractures and fracture dislocations may be a cause of recurrent instability (also enquire the treatment given and duration of immobilization and post-interventional physiotherapy to judge the stiffness and instability).
   h. *Movements:* The movements that aggravate pain (early abduction – supraspinatous tear, painful arc – supraspinatous; flexion – biceps, internal rotation (reaching back) – subscapularis.
   i. Fever: Association is helpful for infective conditions.

2. *Swelling:* Spontaneous onset (infective, PVNS, reactive effusion, inflammatory, haemophilia, degenerative, etc.) or related to trauma (haemarthrosis).
3. *Limitation of movements:* Onset (spontaneous over a period – adhesive capsulitis, sub-acute infections), treatment related (post-traumatic, post-surgical).
4. *Lack of power:* Recurrent subluxation/ dislocations, dead arm syndrome.
5. *Instability:* Voluntary/involuntary, associated with which movement, direction, how frequent, onset and duration, associated neurological injury/ weakness.

Also ask for causes of radiating pain (gastric/ duodenal affections, diaphragmatic affections, cardiopulmonary and mediastinal disorders), polyarthralgia.

Past history: Diabetes, hypertension, neurological disorders (epilepsy), haematological disorder, and tuberculosis.

Examination

General for ligamentous laxity *(See chapter 5 : Case II Q11)*

Inspection

- Attitude (Carriage/Posture):
  - *Anterior dislocation of shoulder:* Elbow kept away (abduction) and slightly in front and external rotation with support of the opposite hand.
  - *Posterior dislocation:* Adduction and internal rotation
  - *Deltoid contracture:* Abducted and drooping of shoulder
  - *Klippel-Feil syndrome:* High webbed neck
  - *Sprengel shoulder:* Scapula higher than uninvolved side
  - Lateral scapular slide (in throwing athletes) scapula of dominant side drawn away from midline.
  - *Prescapular abscess:* Shoulder kept in flexion, abduction (away from irritating pus).
  - *Pyogenic arthritis:* Flexion mild abduction and slight external rotation.

- From front (compare from other side): Sternal notch, sterno-clavicular joint, clavicle and its contour, supra/infra clavicular fossae, acromio-clavicular (AC) joint, preglenoid fossa, anterior axillary fold, coracoids prominence, deltoid mass and shoulder contour, pectoral muscle, sternocleidomastoid muscle and alignment of chin to suprasternal notch
- *From behind:* Midline and alignment of nape of neck to both shoulders, trapezius, medial border of scapula (winging due to serratous anterior weakness or sometimes due to rhomboids and trapezius also), spinous process of scapula, angle of scapula, supra/infraspinatous fossae, posterior axillary fold, 'soft spot' (1 cm medial and 2 cm inferior to angle of acromion) to look for swelling.
- *From top:* AC joint, contour of shoulder
- *From medial aspect:* Swelling of lymph nodes, sebaceous gland infection.

  At all the sites examine the skin for (SEADS) – swelling, erythema, atrophy (of appendages), discoloration, suppuration (scars and sinuses).

Palpation

Note for temperature rise and superficial tenderness then proceed to regional and deep palpation as below.
- *Anteriorly:* Sterno-clavicular joint, clavicle, AC joint, acromion (for os acromiale), subacromial bursa (tenderness just anterior to acromion), long head of biceps (for tendinitis – palpate along 1-4 cms in front of acromion anteriorly with 10° internal rotation of shoulder), myositis mass, pectoralis major tendon (by pressing both palms together), supraclavicular fossa (for brachial plexus injury and 'burners/stingers' due to mild involvement of plexus).
- *Lateral aspect:* For deltoid mass, step-deformity due to inferior subluxation of shoulder
- *Posterior aspect:* Soft spot for swelling
- Medial aspect for pulsations of axillary artery

Movements

- *Anterior flexion (Forward flexion):* Normally up to 160-180º.
- *Abduction:* Look for scapulo-humeral rhythm while patient does abduction, after 90º abduction patient externally rotates the arm. Shrugging of shoulder with abduction often indicates chronic rotator cuff insufficiency. Note for painful arc where abduction is relatively painless in the initial few degrees of abduction and the pain is reported during further arc and may again abate in terminal degrees (suggests supraspinatous impingement/partial tear). Inability to initiate abduction is due to supraspinatous insufficiency while inability to maintain abduction denotes deltoid insufficiency.
- *Adduction:* Ask patient to take the limb forward and also compare the cross-chest adduction.
- Internal rotation in 0º abduction and 90º abduction (normal = 45º): 'Apley scratch test' is more functional to evaluate internal rotation although it also requires some extension (normal IR is up to 80º). In Apley scratch test ask the patient to try and reach back and note the internal rotation in terms of the spinal level reached with thumb (normal=T7 for women and T9 for men).
- External rotation in 0º abduction and 90º abduction: In abduction the 'neutral position' is while forearm is pointing directly in front with elbow flexed (normal ER is up to 90º).
- Total active elevation: The patient is instructed to raise the arm 'in plane of scapula' which is some 20-30º from sagittal plane.
- *Scapular protraction:* Ask patient to bring forward scapulae in 'hunched position' by shrugging the shoulders forward.
- *Scapular retraction:* Ask patient to pull back the shoulders in 'attention attitude': Alternate retraction and protraction may elicit 'snapping scapula' syndrome.

Test for muscle power: Latissimus dorsi (climbing rope maneuver or hanging on beam maneuver), serratus anterior (push against wall – winging of scapula), deltoid (palpate the shoulder contour while asking patient to actively abduct shoulder), trapezius ("shrugging shoulder" – *one does it many times in exams!*),

rhomboids ('attention attitude'), pectoralis major (asking patient to press both the palms together in front); other muscles as below.

Measurements

- Apparent length (from seventh cervical spine to radial styloid {patient standing})
- Arm length (from angle of acromion to lateral epicondyle).
- Mid-arm circumference
- Anterior and posterior axillary folds
  Palpate pulses (radial, ulnar, brachial, axillary)

Special Tests

- Rotator cuff pathology:
  - *Neer's impingement sign:* Passively do maximal forward flexion of shoulder which reproduces the pain due to approximation of anterolateral acromion with rotator cuff and greater tuberosity.
  - *Neer's impingement test:* Inject local anesthetic in subacromial bursa and repeat the test, pain disappears.
  - *Hawkins impingement reinforcement test (Hawkin's and Kennedy):* Passively forward flex the shoulder to 90° and then internally rotate while maintaining the shoulder position. Pain produced indicates rotator cuff pathology or subacromial bursa involvement and is due to rotation of greater tuberosity and subacromial bursa into the acromion and coraco-acromial ligament arch. Similar pain can be produced due to rare coracoids impingement.
  - *Drop arm test:* Passively abduct the arm to fullest and then ask the patient to slowly bring the arm down. After few degrees of retrieval the arm suddenly drops down like a dead limb. Positive in extensive rotator cuff tear and deltoid paralysis.
  - *Subscapularis liftoff test of Gerber (lumbar lift-off test):* Ask patient to reach his back and try to lift the hand away

from back. Pain produced in this maneuver at lesser tuberosity suggests subscapularis tendinitis and inability to perform this test or weakness suggests subscapularis inefficiency.

– Belly press maneuver for subscapularis if patient cannot internally rotate shoulder.

– *Jobe's test:* For supraspinatous – ask patient to abduct arm to 90° and then bring it in 30° forward flexion (from coronal plane) followed by internal rotation (like 'emptying a can of water'). This maneuver produces pain in supraspinatous tendinitis. Further in the test, power of muscle can be tested by asking patient to push the elbow to ceiling and examiner actively resisting it.

– *Passive rotation test:* Passively rotate the shoulder through full range of external and internal rotation in 90° abduction while palpating the front area of shoulder. Popping sensation can be felt in hypertrophied sub-acromial bursa and torn irregular rotator cuff.

– *Test for infraspinatous and teres minor:* Ask patient to forcefully externally rotate while arm is by the side of the body, resistance to this maneuver can be applied and may produce pain at the greater tuberosity in tendinitis of these muscles.

• Tests for shoulder instability:
  – *Anterior instability (Provocative tests): It is good to remember the sequence in which these tests are done (apprehension test → Augmentation test → Relocation test → Release test) as this is a continuous array of test that should be done in a single sitting. Note, however, should be made that the position for performing this array of test is supine and not sitting/standing as had been advised discretely for individual tests.*
  – 'Apprehension test' (originally given by Rowe and Zarins and renamed by Silliman and Hawkins): Patient *supine* – abduct (90°), externally rotate the shoulder, feeling of giving way (apprehension) is taken as positive.
    • 'Augmentation test' of Silliman and Hawkins: To above test, apply anteriorly direct force i.e., extend

shoulder if previous test does not elicit apprehension – positive if patient resists or 'apprehends'.

- *Relocation test (of Jobe):* If above maneuver produces pain instead of apprehension then apply a posteriorly directed force – relieves pain (indicates instability) this can be further tested.
- *Release test of Silliman and Hawkins:* Release the posterior force that relieved pain – patient again complains of pain/apprehension in a positive test.
- *LOAD-SHIFT test:* Load the glenoid with humeral head and do anterior-posterior shift to assess laxity.

– Anterior and posterior instability:
  - *Drawer test "shoulder lachman test" of Gerber and Ganz:* With patient supine, hold proximal humerus in mild abduction and pull and push it forward and backward after stabilizing scapula with other hand and grade the instability. Grade '0' = no translation, '1' = translation up to glenoid rim but not over it, '2' = translation beyond rim with spontaneous reduction and '3' = dislocation and locking of head.
  - *Modified drawer for posterior instability:* Forward flex the arm and apply axial load along humerus to sublux the head out of glenoid – pain and palpable shift suggest posterior labral tear.
  - *Jerk test:* With patient seated abduct the arm to 90° and apply a downward force, then adduct the arm till front of scapular plane when head may subluxates then abduct the arm behind scapular plane when head reduces with a jerk.
  - *Circumduction test:* Perform circumduction in abduction while palpating the posterior aspect for subluxation of head.
  - *Posterior apprehension test of O'Driscoll:* Arm positioned as in Hawkin's test – produces pain and is relieved by injection of local anesthetic.
  - *Posterior subluxation test of Clarnette and Miniaci:* Axially directed force along arm in adduction, 70-90° flexion and internal rotation with the other hand feeling for posterior subluxation of head.

- – Inferior instability:
  - • *Feagin maneuver:* Place the abducted arm of patient on your shoulder and apply a inferiorly directed force to sublux the head inferiorly.
  - • *Sulcus sign 'inferior drawer test' of Neer:* Pull the patients arm downward while it is by the side of patient's body, formation of a sulcus beneath acromion suggests inferior laxity or multidirectional instability. Grade '1'=1cm, '2' = 2cm, '3' = 3cm; >2 indicates a capacious capsule and specific laxity of rotator interval
- – Multi-directional instability:
  - • Compression rotation test for glenoid labrum
  - • O'Brien's test for superior labral injuries
  - • Snyder's biceps tension test
  - • Sulcus sign >2.
- • Tests for dislocated shoulder:
  - – *Hamilton ruler test:* Keep a straight ruler along lateral aspect of arm – in a normal person it does not touch the acromion angle but in dislocated shoulder it does. This will also be positive in any affection of head of humerus.
  - – *Callaway's test:* Girth of shoulder is normally symmetrical – increases in effusion, suppuration, dislocation of shoulder.
  - – *Duga's test:* The elbow cannot be brought to the midline of body
  - – *Bryant's test:* In anterior dislocation of shoulder the anterior axillary fold is elongated.
- • Tests for thoracic outlet syndrome:
  - – *Adson's test:* Abduct the arm by 30° and ask patient to take a deep breath while palpating the pulse (feel the character and compare with other side), then ask the patient to tilt his head towards the same side – reduction or diminution of pulse suggests thoracic outlet syndrome.
  - – *Wright's maneuver:* In the above test abduct the shoulder to 90° and externally rotate the arm.
  - – *Roos test:* Abduct the arm and flex elbow to 90° then externally rotate the shoulder so that the hand faces up.

Ask patient to clench and release fist 15 times. Paresthesia/pain/cramps/ weakness suggest thoracic outlet syndrome.

– *Halsted's test:* While patient standing with arm by the side ask patient to turn the head to other side and extend the neck. Give a downward traction and feel for diminution/obliteration of pulse.

– *Hyperabduction test:* Abduct and hyperextend both the arms (behind the body) simultaneously. Feel for diminution of pulse on affected side.

• Tests for biceps tendinitis:
  – Yergason test: Arm by side and elbow flexed to 90°. Patient is asked to flex elbow and pronate while examiner resists. Pain is felt on anterior aspect of shoulder.
  – *Speed test:* Forward flex the arm and extend the elbow fully. Supinate the upper limb and apply a downward force with patient resisting against it. Pain is felt in the region of bicipital groove

• Tests for SLAP lesion (superior labrum anterior to posterior)
  – *O'Brien's test:* Tested by asking patient to resist when their limb is in 90° flexion and 10° adduction with thumb pointing down. Production of pain is positive which relieves with thumb pointing up.
  – *"Crank test" of Liu et al:* With arm in 160° of abduction in scapular plane apply an axial force with forced internal and external rotation (*like Apley's grinding test for menisci*).

CASE I:
TUBERCULOSIS OF
SHOULDER JOINT

1. What findings support the diagnosis of tuberculosis in this case?

Ans.

History:

• Young adult to middle aged male (TB shoulder is not at all common in children, also this type of TB is one where often a pulmonary focus can be found).

- Insidious onset pain
- Rapidly accompanied by loss of movements and stiffness
- Gradual progression over time of both complaints
- Exacerbation in night and partial relief with analgesics
- Constitutional symptoms – evening rise of fever, night sweats, weight loss, loss of appetite, asthenia, etc.
- Negative history – trauma, polyarthralgia, cervical spondylosis, exertion associated pain

Examination:
- Wasting
- Tenderness (deep joint)
- Spasm of surrounding muscles
- Painful limitation of movements in all directions
- Shortening of arm

Swelling and abscess formation or sinuses are usually absent in TB shoulder as the dry form is much more common than the wet form.

2. What is your differential diagnosis?
Ans. Periarthritis (adhesive capsulitis), primary osteoarthritis of shoulder, osteonecrosis of humeral head and secondary osteoarthritis, rheumatoid arthritis.

3. What are the foci of infection in TB shoulder?
Ans. Glenoid process, humeral head, synovium.

4. How do you classify TB shoulder?
Ans. Clinical types:
1. Caries sicca (dry form – no abscess formation, pus accumulation or sinus formation)
2. Wet suppurative form (swelling, sinus formation common)

Dry form is much more common of the two and has an onset commonly in middle-aged patients. Wet form is less common and is seen in younger patients who are often nutritionally deficient and compromised.

Staging TB shoulder is often not productive as the stages progress so fast that often the patient would be first seen

or diagnosed in advanced arthritis or Ankylosis stage. Hardly if ever one would see a patient in synovitis stage.

5. How would you establish the diagnosis?

Ans. *Please see Chapter 2 : Case I; Q 9.*

6. What are the radiological features?

Ans.

- Generalized osteopenia around shoulder
- Reduction of joint space
- Involvement of both the humeral head and glenoid fossa
- Lytic lesions in humerus and/ or glenoid, later healing stages show lytic-sclerotic regions
- Reduction of subacromial space (in sicca form; it may increase in wet form)
- Wet form often presents with honey combing of proximal humerus.

7. What is your differential on X-ray?

Ans. Radiologic features have *various* differential*s*:

- Rheumatoid arthritis
- Urate arthropathy
- Primary osteoarthritis
- Giant cell tumour of humerus
- Periarthritis (this is the most uncommon radiological differential, however, it clinically closely resembles TB – so getting an early X-ray is imperative).

8. How will you treat the patient?

Ans. This is not as complicated as in TB spine/ hip! Management depends upon the stage in which the patient first presents. Patient presenting relatively early when articular surfaces are well maintained (there may be joint space narrowing) should be started on ATT with gradual mobilisation of shoulder, after adequate pain relief and good response shoulder strengthening exercise should begin to prevent associated morbidity (soft tissue contractures and instability). When patient presents with destroyed (partial or complete) articular surfaces then pain

relief (apart from healing obviously) is the primary goal and immobilization in spica cast in functional position should be done. If the patient fails to achieve painless joint or progresses poorly then excision of focus (debridement) and intraarticular arthrodesis in functional position will be done.

9. What is the functional position of shoulder?

Ans. *As such there are wide variations in the guidelines that are referred.* It is simplified into 30-30-30, which means 30° each of abduction, flexion and internal rotation; or 20-30-40. It is important to understand that one should be able to provide that position where patient can feed himself well and do routine activities. 'Salute position' (with wide abduction) is typically not recommended.

10. How do you judge the position of arthrodesis intraoperatively?

Ans. Abduction is judged by the position of arm in relation to body. Forward flexion is also judged so or in lateral position from the midline. For internal rotation which is supposedly the most important component (and hence should be accurate), after abduction and forward flexion, flex the elbow to 90° so that the hand is in a line between sternum and axilla, then move the hand in a range that thumb is able to reach chin.

11. What are the various methods for shoulder arthrodesis?

Ans. Extra-articular (not preferred): Watson-Jones, Putti, Steindler, etc.

Intra-articular:
- External fixation (Charnley and Houston)
- Internal fixation (Cofield, AO group, Richards)
  Combined intra and extra-articular arthrodesis (Uematsu).

12. What are the problems associated with AO compression arthrodesis?

Ans. Difficulty in bending the plates and prominence of screws (so Richard used reconstruction plates).

CASE II:
THE UNSTABLE SHOULDER

Diagnosis

The patient is a 28-year-old male with post-traumatic anterior shoulder instability for past 4 years. There is no associated neurological deficit and patient is able to pursue his routine activities.

1. What do you mean by shoulder instability and how do you classify it?

Ans. Shoulder instability is both a symptom and sign! Pathomechanically it can be described as inability of the head of humerus to center itself within glenoid fossa during any/some/all of the movements. This, however, is not a practical measure to judge instability, which can only be labeled by careful evaluation of patient symptomatology and examination. Various classifications have been poised to 'divide' instabilities, however, they are all seemingly plagued by impracticality and imprecision. None can substitute the experienced clinicians evaluation and management.

Classification by:

- Type: 'dislocation' vs. 'subluxation'
- Etiology: Macrotrauma, microtrauma (repetitive) or atraumatic
- Direction: Anterior, posterior, inferior, superior, multi-directional.
- Volition: Involuntary vs. voluntary
- Comprehensive (Matsen et al): TUBS (Traumatic, unidirectional, Bankart, Surgical treatment) vs. AMBRI (Atraumatic, Multidirectional, Bilateral, Rehabilitation, Inferior capsular shift in some cases)

    The last one is probably useful in order to group the patients but many patients may fall in between the two groups due to mixed features making it difficult for an average surgeon.

2. What do you mean by 'chronic dislocation'?

Ans. Dislocation for >6weeks

3. How do you recognize posterior dislocation on X-ray?

Ans. The following points help diagnosing posterior dislocations that are often missed:

- Absence of normal elliptical overlapping shadow of humerus with glenoid on routine AP view
- Vacant glenoid sign: Most of glenoid is 'vacant' of humeral head on routine AP view aka 'positive rim sign'
- Trough sign: Caused by reverse Hill-Sach's lesion
- Loss of profile of humeral neck: "Light-Bulb" sign
- Void in inferior or superior glenoid fossa.

4. What defines multi-directional instability?

Ans. Simplified version would be instability in ≥ 2 planes in any combination (this makes it easier to fit the patient into TUBS/ AMBRI).

5. What is Bankart's lesion?

Ans. Avulsion of fibrocartilagenous labrum with anterior capsule and periosteum (combined) is referred to as Bankart's lesion, when this also involves some part of glenoid bone then it is called "bony Bankart".

6. What is Hill-Sachs lesion and reverse Hill-Sachs lesion?

Ans. Impingement Osseous defect (compression fracture) in the posterosuperior humeral head is called Hill-Sachs lesion (seen in anterior dislocation), when similar defect is present in anterior humerus head then it is called reverse Hill-Sachs lesion (seen in posterior dislocation).

7. What are the various risk factors for the development of recurrent shoulder instability?

Ans.

1. Younger age group (75-80% in <20 years, 50% in 20-30 years)
2. Posterior dislocation > anterior
3. Repetitive microtrauma (athletes and overhead activities)
4. Direct anterior dislocation rather than anteroinferior dislocation
5. Associated bony lesions (bony Bankart, Hill-Sach, etc.)
6. Poor compliance with rehabilitation program.

8. What are the various restraints for shoulder?

Ans. Importantly it is the most mobile joint of body and by virtue of compromise into stability for mobility it is also the most frequently dislocated one! The restraints have classically been divided into static and dynamic but (*it is the human nature to create and resolve confusion*) schools are divided over the concept. The following table may help to give an insight that is bound to improve over time:

Factor	Function	Abnormality
STATIC STABILISERS		
Congruity of humeral head to glenoid fossa (glenoids are pear shaped with surface contour and are not matching the humeral head) {head is 1.5-2 times larger in diameter with smaller radius of curvature, around 3 times in surface area)	- Centering of head in concavity - Stability ratio and balance stability angle - Directional load transmission by virtue of version, and conjoint movement of surfaces - "Bony-Cam effect" of scapular inclination	Osseous maldevelopment (dysplasia) Osseous destruction (trauma, infection – glenoid destruction, Hill-Sach's lesion, reverse Hill-Sach lesion) Articular desturction (inflammation, infection, primary, posterior chondrolabral avulsion of glenoid rim)
Humeral head and coracoacromial arch	- Concavity-compression effect	Iatrogenic (acromioplasty in rotator cuff tear), floating shoulder, coracoid avulsion
Glenoid labrum	- Increased depth and surface area - anchors ligaments and capsule	Bankart lesion (osseous, soft tissue, reverse osseous), SLAP, ALPSA (anterior labor-ligamentous

Contd...

Factor	Function	Abnormality
		periosteal sleeve avulsion), POLPSA (posterior labrocapsular periosteal sleeve avulsion), Kim lesion (concealed incomplete avulsion of postero-inferior aspect of labrum)
Capsule (surface area of capsule is twice that of humeral head)	- Posterior portion restrains anterior translation and vice versa	Capsular tear, plastic deformation of capsule
Negative intra-articular pressure, synovial fluid	- Vacuum effect (effective atmos-pheric pressure), cohesive effect	Capsular rupture, defect of the rota-tor interval, capsular laxity, and capsular injury, arthritis and degenerative conditions with loss of synovial fluid
Coracohumeral ligament/superior glenohumeral ligament	- Limits external rotation and inferior translation in adduction and posterior translation in flexion	Lesion of the rotator interval (between supra-spinatous and sub-capsularis), rotator cleft lesions (bet-ween superior and middle glenohume-ral ligament, situ-ated deep to interval)

Contd...

Factor	Function	Abnormality
Middle glenohumeral ligament	- Limits external rotation and inferior translation in adduction and anterior translation in midabduction (45°)	Bankart lesion and capsular injury, HAGL (humeral avulsion of gleno-humeral liga-ments), RHAGL (reverse HAGL)
Inferior glenohumeral ligament complex	- Limits anterior, posterior, and inferior translation in abduction (45-90°)	Bankart lesion and capsular injury, HAGL, RHAGL
Posterior aspect of the capsule	- Limits posterior translation in the flexed, adducted, and internally rotated shoulder	Posterior capsular laxity and injury, POLPSA

DYNAMIC STABILISERS

Factor	Function	Abnormality
Rotator cuff	- Dynamic com-pression of the joint; steering effect	Overuse injury (fatigue) and rupture, interval/cleft injury
Biceps (long head)	- Dynamic restraint to anterior and superior translation	Lesion of the superior portion of the labrum, anterior and posterior (SLAP lesion) and rupture

Typically speaking the static and dynamic stabilizers are complementary and in any given position of shoulder both supplement each other's function. Mere congruence of glenohumeral articulation will be ineffective without ligamentous support and 'dynamic' muscular 'com-pressors'. Similarly during night time when the muscles

are relaxed still the rotator cuff (deemed to be dynamic stabilizer) is in enough tension to restrain the joint from dislocation thus having by itself a 'static' effect.

9. How do you evaluate the patient for unstable shoulder?

Ans. I will methodically examine the patient as above to be able to decide:

1. The articulation(s) involved (glenohumeral, acromio-clavicular, scapulothoracic, sterno-clavicular) that are responsible for the complaints of patient
2. Whether there is decentering of head
3. Direction(s) of instability
4. Mechanical factors responsible for decentering of head
5. Dynamic factors responsible for decentering
6. Age and psychological status of patient amenable for treatment
7. Possibility of repairing the pathologic factor

   I will proceed with no touch examination while listening to patient's complaints and asking him to position the limb/maneuver that produces the symptoms. Then manipulative examination will be done (*Always perform the manipulative tests first on uninvolved shoulder).*

10. What else will you like to do?

Ans. I will also do an examination under anesthesia (gold standard) and radiologic investigations to look for bony articulation (CT scan), capsuloligamentous abnormalities (MRI scan).

11. How will you look for ligamentous laxity?

Ans. Look for the following, Beighton criteria (9 sites in total, *4 are bilateral*):

1. More than 10° hyperextension of the elbows
2. Passively touch the forearm with the thumb, while flexing the wrist.
3. Passive extension of the fingers or a 90° or more extension of the fifth finger (Gorling's sign). This is used as a "Screening Test".

4. Knees hyperextension greater than or equal to 10º (genu-recurvatum).
5. Touching the floor with the palms of the hands when reaching down without bending the knees. This is possible as a result of the hypermobility of the hips, and not of the spine as it is commonly believed.

   A score of 4/9 (on Beighton score) is a major criterion in evaluating the patient on Brighton criteria.

12. What is the role of muscles as stabilisers?

Ans. The surrounding muscles are "compressors" of shoulder joint that can be produced practically in any position. They should not be classified as depressors or elevators in the perspective of shoulder stability.

13. What is the rotator cuff?

Ans. The rotator cuff is a musculo-tendinous structure that surrounds the shoulder joint from all sides except inferior. It is formed by four muscles that attach to the tuberosities and have the following functions:

- Subscapularis (lesser tuberosity) – principle anterior compressor, chiefly in an internal rotator supplied by upper and lower subscapular nerve
- Supraspinatous (greater tuberosity) – principle superior compressor, chiefly is an abductor supplied by suprascapular nerve
- Infraspinatous (greater tuberosity) – principle posterior compressor, chiefly is an external rotator supplied by suprascapular nerve
- Teres minor (greater tuberosity) – complements infraspinatous, supplied by axillary nerve

14. How will you manage the patient?

Ans. I will put the patient on a supervised plan of physiotherapy for muscle balancing and strengthening exercises. The progress is judged by symptomatology. If the patient fails to improve over a prolonged program (more than six months) I will consider surgery for unilateral instability. Multidirectional instability (such as

due to ligamentous laxity) needs to be carefully evaluated with respect to various psychological factors, the treatment options are very limited here.

Operative reconstruction is contraindicated for voluntary, habitual dislocations in a psychologically unstable patient.

15. How will you operatively manage the instability?

Ans. For a given instability there are numerous variations that have to be individually judged. A practical guide would be as follows:

1. Labral injuries producing unidirectional instability: Bankart's repair ± capsular shift of Neer
2. '1' + capsular injury (often anterior) {capsulolabral injury}: Bankart repair with capsular reconstruction of Jobe's or capsular shift.
3. '1' + bony eburnation: Bankart's repair with glenoid osteoplasty (Eden-Hybbinette or similar procedure)
4. For similar posterior lesions postero-inferior glenoplasty of Metcalf can be done
5. Loss of coraco-acromial arch with anterosuperior escape of humeral head: "dismal injury" – reconstruction is not possible with present techniques: 'Reverse shoulder arthroplasty'
6. Posterior instability due to capsuloligamentous inefficiency: Capsular shift of Tibone
7. Multi-directional instability: Inferior capsular shift of Neer and Foster (or its modification) with repair of the mechanical factor if possible.
8. Hill-Sach's lesion: Derotation osteotomy + capsular reefing "or" bone grafting of the lesion (Neer) "or" tight anterior repair (± Putti-Platt subcapsularis double breasting).
9. Reverse Hill-Sach's lesion: Modified McLaughlin procedure.
10. If >40% of head involved: Shoulder arthroplasty.
11. Loss of glenoid rim:
    a. Up to 20%: Repair the detached labrum and capsule back to the intact glenoid cartilage to render the defect extracapsular.

   b. Up to 25%: Reattachment of the fragment "or" Bristow-Helfet procedure if the fragment cannot be reattached.

12. Reconstruction of capsuloligamentous deficiencies due to previous surgeries by tendon graft from humeral attachment

13. Deficient tendon of subscapularis: Hamstring tendon graft

14. Instability associated with denervation/paralysis/ irreparable detachment of muscles: glenohumeral arthrodesis.

16. What is the role of arthroscopic procedures?

Ans. The reconstruction of a lot of injuries to 'mechanical' capsuloligamentous and labral injuries (Bankart's, SLAP, POLPSA, rotator cuff tears, capsular tears/ laxity, etc.) is now possible with meticulous and advanced arthroscopic procedures. The posterior lesions are less amenable to arthroscopy and need expertise.

The Elbow Joint

{Short cases are very popular, however, long case may also be given typically to a DNB candidate – it's better to be prepared. Attempts to cover all the necessary aspects have been made in the following text.

Read: 5-7 times for a DNB candidate, 3-4 times for a MS candidate}

EXAMINATION POINTS
FOR AN ELBOW CASE

History

1. *Pain:* Onset, site (bony – epicondylar, supracondylar, olecranon, radial head, joint line; soft tissue – extensor origin, flexor origin, bursae), duration, radiation, association with fever, trauma.
2. *Swelling:* Onset, duration, localized (laterally over soft spot 'Anconeus triangle' or any soft tissue site– mitotic pathology) or generalized, associated symptoms.
3. *Limitation of movements:* Associated with swelling/trauma/ manipulation/immobilization/massage/fever/iatrogenic.
4. *Deformity:* Congenital vs acquired, association with trauma, fever and swelling, treatment related, Charcot's arthropathy.
   Additional history: Massage, haemophilia, manipulation attempts.

Examination

Inspection

- *Attitude*: Alignment of forearm in relation to arm (carrying angle) compare from normal, flexion deformity.
- *From front*: Flexion crease (1-2 cms above joint line it is at the level of interepicondylar axis), swelling (epitrochlear lymph nodes, etc.), medial and lateral epicondyle, biceps tendon and lacertus fibrosus, common extensor and flexor origin mass.

- *From lateral aspect*: Anconeus triangle and fullness over soft spot, common extensor origin, prominence of triceps tendon (elbow dislocation), olecranon process, radial head (dislocation), olecranon bursa, biceps mass.
- *From medial aspect*: Medial epicondylar prominence, supracondylar depressions, common flexor origins
- *Posterior aspect* (in 90º flexion): Olecranon process, triceps bulge, paraolecranon fossae, three point relationship.

Palpation

- *Superficial palpation*: Temperature, superficial tenderness
- *Deep palpation*:
  - Epicondyles, olecranon process, olecranon fossae, radial head, capitellum (Panner's disease)
  - Myositis mass, any swelling with all characteristics (site, size, shape, surface, margins, consistency, tenderness, compressibility/ reducibility, pulsatility)
  - Supracondylar ridges: Palpate both medial and lateral ridges simultaneously for irregularity, thickening, discontinuity, spur, loss of contour
  - Three-point relationship
  - Ulnar nerve (thickening, subluxation, tenderness)
  - Joint line (for tenderness)
  - Brachial artery pulsations

Movements

- Flexion (0º-140º), extension (reversal of flexion)
- Hyperextension up to 10º is normal
- Supination (85º), pronation (70-80º)

Measurements

- Linear measurements:
  - *Arm length (medial and lateral):* Lateral length measured from angle of acromion to lateral epicondyle, medial length is measured from medial epicondyle to an

imaginary soft tissue point on the inner aspect of arm just against the deltoid insertion.

– *Forearm length:* Lateral length measured from lateral epicondyle to radial styloid process and medial length from medial epicondyle to ulnar styloid process.

– *Three point relationship measurements (in 90° flexion):* Measurement of olecranon tip to lateral and medial epicondyles and interepicondyler distance.

• *Angular measurements:* Angle formed between arm and forearm axis. (Axes are marked by joining the mid-point of lines joining the above bony points, viz. epicondylar line at elbow, interstyloid line at wrist, line joining deltoid insertion and a point against it).

• *Circumferential measurements:* For wasting – measure mid-arm circumference and forearm circumference at a convenient point (usually 7 cms below medial epicondyle).

Neurological examination: Motor power of elbow flexors, extensors, supinators, pronators, biceps jerk, triceps jerk and supinator jerk and sensory testing.

Special Tests

• *Varus stress test:* Test in 30° flexion to disengage olecranon.
• *Valgus stress test:* Test in 30° flexion
• *Pivot shift test (for posterolateral rotator instability):* For anterior ulnar collateral ligament by applying valgus and axial compressing forces to elbow and supination torque to forearm. This maneuver produces rotator subluxation of ulnohumeral joint which is maximum at 40° of elbow flexion.
• Tests for lateral epicondylitis:
    – Resisted wrist extension test
    – Long finger extension test: Pain produced just distal to lateral epicondyle suggests lateral epicondylitis (cf-radial tunnel syndrome)
    – Wringing test
    – Chair test
    – Jug test

- – *Cozen's test:* To an extended elbow and wrist ask the patient to make fist and passively flex the wrist – produces pain at the common extensor origin
  - – Mill's maneuver: With wrist and elbow extended pronate forearm.
  - – Broom test
  - – Rolling-pin test
  - – Stir-fry test
- *Resisted pronation:* For pronator syndrome
- *Resisted flexion and pronation:* For medial epicondylitis
- *Elbow flexion test* for cubital tunnel syndrome.
- *Long finger extension test:* Pain produced 4 finger breadths below lateral epicondyle suggests radial tunnel syndrome
- *Resisted elbow flexion and resisted forearm supination:* For median nerve compression at lacertus fibrosus

Also examine the shoulder and forearm as the compensations of elbow movements are often taken up at shoulder and the illeffects are often sustained by forearm.

1. What are the prerequisites of elbow examination?

Ans.
1. Examine both elbows together.
2. Examine both elbows in identical position.
3. Expose whole upper limb from shoulder girdle to fingers.
4. Arms lying by the side of chest is the most comfortable position for the patient.

2. What are the interpretations of three point relationship measurements?

Ans. The olecranon, lateral and medial epicondyle form a near-equilateral triangle in 90° flexion at elbow. In disruptions of elbow joint, the measurements are altered and can be interpreted as below:

1. *Decreased length of medial limb:* Posteromedial dislocation, medial rotation of fractured fragment
2. *Decreased length of lateral limb:* Posterolateral dislocation, lateral rotation

3. *Increased length of medial limb:* Fractured medial epicondyle/condyle
4. *Increased length of lateral limb:* Fracture lateral condyle
5. *Increased base (interepicondylar distance):* Malunited fracture intercondylar humerus.

3. What are the fallacies of three-point relationship?
Ans.
- Displaced fracture of medial/lateral epicondyle
- Surgical intervention done leading to altered morphology and inability to palpate epicondyles
- Excision arthroplasty of elbow
- Lateral spur formation
- Ankylosed elbow in extension
- Charcot's arthropathy

4. What is the importance of anconeus triangle?
Ans. The triangle is bounded by radial head, lateral epicondyle and olecranon tip. The triangle directly overlies joint capsule and anconeus muscle so this 'soft spot' becomes distended early in effusions of elbow joint. Synovial biopsy from elbow joint, injections into elbow joint can be directly given by approaching this triangle. Anconeus is an important muscle in EDS studies and can be easily approached here.

5. How do you look for rotational deformities at elbow joint?
Ans. Internal/external rotational mal-alignment at elbow can be measured by measuring the external and internal rotation at shoulder in 90° abduction and while the arm is by the side (*the movements are to be always compared from other side*). Internal rotation deformity at elbow will restrict external rotation at shoulder and complementary increased internal rotation. This deficit or excess can be measured and is a quite accurate measure of deformity. For this measure to be effective there should be no rotational limitation at shoulder or torsional deformity of

humerus and that the deformity should be unilateral. (*Remember that this measure is often only an approximation as the dominant shoulder has an increased physiological rotation giving fallacious comparison for deformities of left elbow and masking the ipsilateral deformities*)

Alternative method (Yamamoto et al): Make the patient bend forward and fully internally rotate and hyper-extend the shoulder (in an attempt to touch the back). Lift the hand off the back (in a normal the hand can either be not lifted up or it is minimal). The angle made by forearm to the horizontal when compared to the opposite side gives an idea of internal rotation deformity.

6. How do you palpate for radial head?

Ans. Locate lateral epicondyle with elbow flexed to 90° then go just in front of it where a depression is first felt immediately stopped by bony silhouette of radial head. Confirm by gently rotating forearm for transmitted supination and pronation.

7. How do you look for flexion deformity of elbow?

Ans. Make sure that the interepicondylar line is horizontal to the ground before assessing the flexion deformity. (*Note – in the presence of flexion deformity, never comment on valgus deformity as even the physiological valgus varies with flexion of elbow. Similarly, however, one can say that there is varus deformity but do not attempt to quantify (measure) it when flexion deformity coexists as classically these measurements have been described in elbow extension*).

CASE I: TUBERCULOSIS OF ELBOW JOINT

Diagnosis

The patient is a 22-year-old female with active tuberculosis of left elbow on treatment for past 2 months with flexion deformity of 30° and further flexion of 90° (30-120°). There is restriction of pronation and supination to 0-30°.

1. Why do you call it tuberculosis of elbow?

Ans. History:
- Insidious onset of pain and swelling
- Prolonged symptoms with gradual progression
- Swelling (boggy) with effusion at elbow joint
- Limitation of movements over a duration
- Healing sinuses/scar
- Wasting of forearm and arm muscles
- Axillary lymphadenopathy

2. What is your differential diagnosis?

Ans.
- Myositis ossificans
- Pigmented villo-nodular synovitis
- Subacute septic arthritis
- Partially/incompletely treated septic arthritis
- Mitotic pathology of muscles/synovium
- Charcot's arthropathy
- Clutton's joints

3. What are the foci of infection for elbow joint tuberculosis?

Ans. Olecranon, distal humerus, radial head and uncommonly synovial membrane.

4. What will you do to confirm diagnosis?

Ans. AP and lateral projections of elbow joint which reveals diffuse osteopenia, lytic foci in relevant bone, periosteal reaction, decreased joint space. Late stages may additionally show deformation of bone ends, pathological posterior dislocation of joint, spina ventosa of proximal ulna.

   For other diagnostic modalities please see (*See Chapter 2; Case I; Q 9*)

5. What do you mean by the term spina ventosa?

Ans. *Spina = long/ short slender bone, ventosa = dilated with air.* The loculated expansile appearance resembles a dilated

bone due to air. This deformity is often seen in thin long slender bone of hands and feet.

6 How will you manage the patient?

Ans. Early diseases with maintained outline of bones and somehow preserved joint space are expected to return to good function with healing of disease. I will put the patient on chemotherapy (*For regime please see chapter 2; Case 1; Q 19*) and supervise for the response. Advanced disease with destruction of bone ends and pus collection and discharging sinuses are better managed with early excision of focus and chemotherapy with immobilization in functional position.

7. What are the other surgical options?

Ans.

Excisional arthroplasty: For disease healed in unacceptable position or in a case of advanced arthrodesis/Ankylosis if the patient desires a mobile joint.

Arthrotomy and excision of focus: For uncertain diagnosis, non-responders or advanced disease.

Arthrodesis: For a heavy manual labourer.

Arthroplasty: Principles are same as discussed elsewhere (*See Chapter 10; Elbow Arthroplasty*).

8. What is the optimal position of elbow arthrodesis?

Ans. Optimal position of arthrodesis varies from patient to patient and should be determined by experimentally immobilizing the limb using POP cast or brace. For unilateral arthrodesis 90° flexion and mid-prone position is often acceptable. Bilateral cases may be arthrodesed in 65° for one limb in supination for personal hygiene and the other in 110° flexion in mid-prone position for ability to reach mouth, however, this is highly undesirable to do so!

CASE II:
THE STIFF ELBOW

Diagnosis

The patient is a case of post-traumatic stiff elbow with flexion contracture for past two years with 60º flexion deformity and further 30º range of motion.

> **1. How much functional range of movements is required at elbow joint?**

Ans. For activities of daily living 100º flexion with the functional range of arc from 30º-130º and 100º rotation (50º each of pronation and supination) is required.

> **2. How do you classify stiff elbow?**

Ans.

Extrinsic Causes

- Soft tissue
    - Capsulo-ligamentous
    - Muscular
- Ectopic ossification

Intrinsic Causes

- Intra-articular adhesions
- Loss of articular cartilage
- Gross distortion resulting from inadequate or failed reduction.

> **3. What are the various causes of stiff elbow?**

Ans.
- Post-traumatic
    - Joint incongruity
    - Dislocation/Subluxation
- Heterotopic ossification
- Burns

- Coronoid/olecranon/Radial osteophytes
- Loose bodies
- Triceps/Biceps adhesions
- Chronic infection
- Inflammatory arthritis
- Patient non-compliance
- Post surgery.

4. How will you differentiate extrinsic from intrinsic stiff elbow?

Ans. Intrinsic stiff elbow gives a bony stop to movements while extrinsic stiffness presents with comparatively soft stop. There is associated deformity in coronal plane in intrinsic stiffness. Movements are grossly painful in intrinsic causes due to early development of arthritis. In extrinsic causes the contracture stands out on pursuing movements. Wasting of all groups of muscles is more evident in intrinsic stiff elbow.

5. How will you manage this patient?

Ans. Evaluate with radiological investigations (X-ray and/or CT) and then categorization and management as below:
- *Acute:* ORIF, continuous passive motion, splints (dynamic and static)
- Sub-acute (<6 months): Splints, anti-inflammatory drugs, close follow-up
- Chronic (> 6 months): X-ray ± CT scan
  - Ectopic ossification present
  - No ectopic ossification:
    - Extrinsic causes: Release
    - Intrinsic causes:
      - Articular: Soft tissue release or distraction arthroplasty for acceptable surfaces otherwise elbow arthroplasty (>50 per cent cartilage destroyed)
      - Impingement: Coronoid/Olecranon: Excision of bony stop.

6. What will you do for this patient?

Ans. I will put the patient on conservative regime of stretching, strengthening and mobilisation with analgesics. Then based upon the progress (if functional range of movement not regained) I will decide surgery.

7. What surgery will you do?

Ans. I will do anterior contracture release (Column procedure). Then I will reassess the movements on table if there is restriction of flexion then I will do a posterior release also.

8. What is Column procedure?

Ans. Posterolateral incision → Dissection between ECU and Anconeus. Separate extensor tendon from joint capsule and LCL. Expose anterior capsule and release it as wide as possible. For posterior release dissection proceeds between ECRL and triceps and posterior capsule is released. Look for and excise the olecranon and coronoid osteophytes. Immediate CPM is begun followed by dynamic splintage after 3 weeks and gradual weaning over 3 months.

9. What is Bhattacharya procedure?

Ans. Elbow arthrolysis procedure of Bhattacharya included the following:
- Removal of capsular contracture
- Mobilizing brachialis and triceps from lower humerus
- Restoration of trochlear pulley
- Minimal removal of bone block without excising articular surface
- Post-operative course:
  - Instill 25 mg hydrocortisone acetate in joint with 2-5 cc of hylase
  - Compression bandage with splint in full extension
  - 2nd dose of hydrocortisone with 2-4 cc of lignocaine on 7th to 10th day.

10. What are the contraindications of arthrolysis/ soft tissue release?

Ans. Soft tissue release procedures should not be done in the following:

1. Significant alteration of the articular contour
2. Loss of cartilage >50 per cent
3. When release of one or both collateral ligaments
4. Motor deficiency or spasticity.

11. What are various arthroplasty options for elbow?

Ans. The intra-articular (intrinsic) causes need the following: Distraction arthroplasty, fascial interposition arthroplasty, replacement arthroplasty.

12. What is distraction arthroplasty?

Ans. Keeps the injured articular surfaces distracted while simultaneously providing joint motion and protection to collateral ligaments.

Indications:

- Adjuvant to capsule release if ligaments are damaged, or
- Significant dissection making intraoperative motion difficult, or
- > 50 per cent joint surface void of cartilage, or
- Modified joint contour.

13. What is fascial interposition arthroplasty?

Ans. It is an alternative for a poorly articulated or ankylosed joint where painless movements are desired and elbow replacement cannot be done. *Indications:*

1. Young patients with post-traumatic ankylosis of elbow with intact broad contour of distal humerus.
2. Young adult stage I and II rheumatoid arthritis with intact bone.

14. What materials can be used for interposition arthroplasty?

Ans. *Natural:* Fascia, fat patch
Synthetic: Synthetic membranes

15. What is ectopic ossification?

Ans. Formation of bone at abnormal places.

Types:
- Heterotropic ossification: Formation of mature lamellar bone in non-osseous tissue (dystrophic process involving ligaments and capsule; can be metastatic).
- Myositis ossificans (a misnomer): Benign localized <u>reactive</u> proliferative lesion occurring within soft tissues (muscles) that normally do not ossify.
- Periarticular ossification: Collection of calcium pyrophosphate crystals in soft tissue (lacks trabecular pattern).

16. What are the various causes of myositis ossificans?

Ans. Classified into three types (etiological classification):
1. Traumatic (myositis ossificans circumscripta, myoosteosis, ossifying hematoma): Contusions, tearing, post-operative.
2. Neurogenic: Injury to neural axis
3. Myositis ossificans progressiva – inherited disorder
   More than 1/3rd cases are idiopathic.

17. Which muscle is commonly involved in myositis ossificans at elbow?

Ans. Brachialis muscle, others can be pronator teres and brachioradialis.

18. What are the clinical features of myositis ossificans?

Ans. Clinical features depend upon the phase in which patient is seen:
- Acute/pseudoinflammatory phase (3rd day – 3rd week): pain, swelling and ↓ ROM
- Subacute/Pseudotumor phase (3-6 weeks): painless hard mass with raised temperature locally.
- Maturation (3-6 months)
- Resolution (few cases only).

19. How do you classify ectopic ossification around any joint?

Ans. Functional classification (Hasting and Graham):

Class I: Radiologically evident ectopic ossification without clinical limitation

Class II: Subtotal, functional, limitation of motion

A: In flexion and extension plane

B: In pronation and supination plane

C: In both planes

Class III: Ankylosis that eliminates motion (A,B,C as above).

20. What is the differential diagnosis of myositis ossificans?

Ans. The following are often confused with myositis ossificans:
1. DVT in acute phase
2. Osteogenic sarcoma
3. Mesenchymal chondrosarcomas
4. Synovial sarcoma
5. Calcified lipoma
6. Hemangiomas (phleboliths).

21. What is 'zonal' phenomenon?

Ans. This applies to the functional orientation of fibroblasts to osteoblasts in all ossifying masses. Myositis ossificans matures from inside to outside. Center is less mature (higher mitotic figures and cellular atypia), mature cells in periphery. 'Reverse zoning' is seen in tumours. This helps differentiate myositis from otherwise very difficult to differentiate osteoid forming tumours typically osteosarcoma histologically.

22. What are the available treatment modalities for ectopic ossification?

Ans. Prophylaxis:
- Chemotherapeutic agents:
  - *Acute phase:* Ice, compression, maintainance of ROM (CPM), support the limb, avoid massage/forceful passive mobilisation.
  - *Bisphosphonates:* Merely delay the appearance of mass – now recommended now
  - NSAIDS: Started on the first post-operative day and continued for 2 weeks

- Calcitonin ? efficacy – also not recommended
- Thalidomide (for myositis ossificans progressiva)
• Radiation therapy: Low dose external beam radiation within 96 hrs (20Gy/10 fractions. Coventry (1981) is recommended, other regimes are 10Gy/5 fractions, 6-8 Gy/5 fractions)
Excision: Surgery

23. What are the indications of surgery?

Ans. Criteria for doing surgical treatment:
- Functionally limiting joint stiffness (mechanical obstruction) or neurological or vascular complications due to mass are the only finite indications for excision. Relative indications include non-cosmetic bump and if the patient demands surgery.
  Prerequisites:
- Radiographic union of fracture
- Radiographic evidence of intact articulating surfaces
- Mature mass
- Soft tissue equilibrium (don't touch a hot metabolically active mass)
- Stabilized traumatic brain injury and motivated patient.

24. How do you assess maturity of mass?

Ans. Note the following points:
- No local rise of temperature, no oedema
- Normal ESR and alkaline phosphatase
- Bone scintigraphy – investigation of choice (≥2 bone scans showing normal/ decreased uptake)
  In general 18 months is accepted as the sufficient time needed for maturation.

25. What is the surgical protocol?

Ans. Delayed intervention (>18 months) is recommended and has the advantage of finding a metabolically quiescent bone in a tissue in equilibrium. Also it gives additional time for associated injuries (traumatic brain damage) to heal and stabilize.

26. How will you surgically manage ectopic ossification around elbow?

Ans. After taking care as above I will resect the ossified mass taking care for the following:

- Incision should be selected to be able to resect all ectopic bone
- Decompress the compressed nerve
- Resection of anterior and posterior capsule
- Debride coronoid process
- Clear olecranon fossa
- Excision of terminal 1-1.5 cms of olecranon
- Correction of elbow instability
- Transposition of ulnar nerve
- Preserve collateral ligaments and annular cartilage of radial head
- Start prophylaxis.

27. What are the problems of delayed treatment?

Ans. Delay leads to:

1. Progressive and advanced contracture
2. Potential articular cartilage destruction
3. Prolonged infirmity.

CASE III: THE UNSTABLE ELBOW

Examination and Findings

- History:
  - Antecedent trauma
  - Overuse sports injury
  - Congenital anomalies
  - Surgery to elbow
  - Erosive arthropathy
  - Generalized ligament laxity
  - Locking/clicking with clunk when elbow supinated in extension half way through ROM
  - Assess patient's needs and demands
- Generalized joint laxity
- Arthropathy (Charcot's/ Syphilitic)

- Elbow:
    - External signs of injury/surgery
    - Tenderness over collateral ligaments
    - Stress tests:
        • Valgus
        • Varus
    - Milking maneuver
    - Radiocapitellar compression test
    - Lateral pivot shift test
    - Valgus laxity in pronation
    - Valgus extension overload test
    - Ulnar nerve: Tinel's test

1. What are the various restraints to elbow joint?
Ans. Dynamic: Anconeus, triceps, brachialis
Static:
- Primary
    - Ulnohumeral articulation (only stabilizes in <20° and >120° flexion)
    - MCL
    - LCL
- Secondary:
    - Radial head
    - Capsule
    - Common flexor pronator and extensor origin
    Stabilizers of elbow for various stresses:

Valgus stress:
- Primary:
    - MCL-
        • Anterior bundle: Principle-stabilizer in 30°-120° flexion
        • Posterior bundle: Co-restraint
- Secondary:
    - Radial head
- Tertiary:
    - Flexor-Pronator muscle groups (FCR,FDS)

Varus stress
- Primary:
  - LCL and annular ligament complex
- Secondary:
  - Extensor muscles with fascial bands
  - Intermuscular septa

2. What is "fortress concept" for elbow stability?

Ans. According to O'Driscoll:

The ulnohumeral articulation, lateral collateral ligament (LCL), medial (anterior portion) collateral ligament (MCL) serve as outer "wall" and radio-humeral, common extensor and common flexor origin as inner "wall" of fortress as protection against stresses.

3. How do you classify the elbow instability?

Ans. Five criteria have been used to classify elbow instabilities (see table).

Articulation involved	Proximal radio-ulnar joint (radial head)	Ulnohumeral and radiohumeral (elbow)	Both (divergent)
Direction of displacement	Anterior Posterior	Varus/valgus Anteroposterior Mediolateral Posterolateral rotatory instability (PLRI)	Posterior
Degree of displacement	Subluxation	Perched	Complete
Timing	Acute Chronic	Acute/chronic Recurrent	Acute
Associated fractures	None	Radial head Olecranon Coronoid	None

4. What are the various patterns of elbow instability seen?

Ans.

Valgus instability
- Athletic chronic overuse injuries
- Repetitive overload
  - Intrinsic (muscular)
  - Extrinsic (external tensile overload)

Varus instability
- PLRI
- Recurrent isolated radial head instability

  Anterior instability

  Posterior instability.

5. What are the various causes of elbow instability?

Ans.
- Acquired causes
  - Simple dislocation
  - Fracture dislocation
  - Fractures in association with ligament injuries
  - Pure ligament injuries
  - Arthropathies
  - Hyperextension injury
  - Repetitive valgus stress
- Congenital causes
- Iatrogenic causes.

6. What is the 'unified concept' of elbow instability?

Ans. The unified concept (pathomechanics) of clinical spectrum of acute and chronic elbow instability – "circle concept" (Hori's circle)
- Three sequential stages of elbow instability.
- The Hori's circle progresses from lateral to medial side.
- It may pass through soft tissue or bone or both.

7. What is the most common mechanism of injury producing unstable elbow?

Ans. Valgus strain in a supinated elbow under axial compression is the most common mechanism of injury, which

is seen in fall on outstretched hand. The stresses begin laterally and pass medially 'around elbow' joint anteriorly or posteriorly depending on the position and either through bone or soft tissue or both.

(*Most chronic affections of elbow affect the MCL due to peculiar disposition of elbow in valgus, however, most acute injuries affect LCL due to positional valgus stress at the time of injury!*).

8. What is the sequence of tissue injury?

Ans. The circle in progression meets three distinct stages that can arrest anywhere depending upon the severity of injuring force:

- *Stage I:* LCL disruption → Posterolateral rotatory subluxation → may undergo spontaneous reduction.
- *Stage II:* Additional anterior and posterior disruption → Coronoid perched over trochlea → may reduce with minimum force.
- *Stage III:* Subdivided into three parts-
  – IIIA: Anterior medial collateral ligament intact → stable in pronation
  – IIIB: Entire MCL disrupted → varus, valgus and rotatory instability following reduction.
  – IIIC: Entire distal humerus devoid of soft tissue attachment → unstable even in 90° flexion in cast.

9. What are the associated fractures with this injury?

Ans. Coronoid, radial head, posterior capitellum, medial and/ or lateral epicondyle.

10. What are your differential diagnoses?

Ans. As the patient presents with pain on activity and feeling of 'not enough strength'; the following serve as important differentials:

Anterior: Anterior capsular tear, pronator teres syndrome, annular ligament disruption

Posterior: Traction apophysitis, triceps tendonitis/ rupture, valgus-extension overload syndrome

Medial: Medial epicondylar physeal fractures, medial epicondylitis, snapping elbow syndrome, medial elbow instability, ulnar neuritis

Lateral: Panner's disease, radial head fracture, radio-capitellar overload syndrome, PLRI.

11. How will you investigate the patient?

Ans. If the clinical examination is equivocal then manipulation under anesthesia is worth considering in a symptomatic patient after ruling out above conditions.
Radiology:

- *Plain X-rays:* To look for associated bony injury, degenerative changes, ectopic bone formation, posteromedial osteophytes
- *Stress radiographs:* Varus and valgus stress, posterolateral stress
- *MRI and MR Arthrography:* Rupture of collateral ligaments and muscle groups
- *CT Arthrography (86 percent sensitive and 97 percent specific):* Undersurface tear in MCL → 'T-Sign'
- Arthroscopy: ?value.

12. How will you decide treatment in this case?

Ans. I will undertake the following course (please refer to classification especially type III for a good understanding of the algorithm and the various constraints and 'circle concept'):

1. Reduce and test stability clinically if stable → splint/sling and early mobilisation and muscle equilibrium and strengthening exercises
2. If unstable in '1' then pronate and retest, if stable → hinged brace, forearm pronated and gradual mobilisation
3. If unstable in '2' then fully flex and check for upto 30° extension block, if stable → hyperflexion and pronation immobilization with 30° extension block and gradual mobilisation
4. If unstable in '3' then → operate!

13. How will you decide surgery?

Ans. Surgical treatment algorithm (depends much on the identified ligamentous injuries and radiological investigations):

1. If all constraints can be repaired → ORIF and repair ligaments/tendons

2. No to '1' but ulno-humeral articulation intact → excise unfixable radial head/ replace radial head and repair ligaments/ tendons

3. No to '2' but ulno-humeral joint can be fixed → ORIF coronoid and or olecranon and may excise/ replace radial head, repair ligaments and tendons

4. No to '3' then look for 'type' of coronoid fracture: For type I/II coronoid fracture → suture it , ORIF or replace/ partially excise radial head and repair ligaments and tendons. For type III coronoid fracture → ORIF coronoid and apply hinged distractor.

14. What is the terrible triad of elbow injury?

Ans. Combination of elbow dislocation and radial head and coronoid fractures.

15. How will you manage this injury?

Ans. Fundamental to management is to convert complex dislocation into simple dislocation as above.

16. What is the cause of recurrent dislocation of elbow?

Ans. Exceedingly rare, causes may be mishappen trochlear notch of ulna, lax collateral ligaments. Both congenital and post-traumatic forms are documented. Manage as above for post traumatic ones else for congenital forms → arthroplasty

17. What is recurrent dislocation of radial head?

Ans. Disruption of annular ligament produces anterior subluxation or dislocation of radial head especially during pronation. Diagnosis done by finding of locking/ popping of elbow, decreased ROM, crepitus over radial head ± tenderness, apprehension to hyper pronation.

18. What is your differential?

Ans. PLRI, monteggia fracture-dislocation, congenital dislocation of radial head, dislocation in association with cerebral palsy.

19. How will you treat recurrent radial head instability?

Ans. Annular ligament reconstruction, radial head excision.

CASE IV:
CUBITUS VARUS

Diagnosis

The patient is a 10-year-old male child with right side cubitus varus deformity for 7 months due to malunited supracondylar fracture of humerus without any distal neurological deficit.

1. How do you define cubitus varus deformity?

Ans. When the forearm is deviated inwards with respect to arm at elbow with resulting lateral angulation *in full extension*, we call it cubitus varus. (*Note- Literally speaking any reduction of physiological valgus **is also cubitus varus** – although there is no true varus angulation! This further implies that even cubitus rectus is 'cubitus varus' deformity of elbow so when you do the correction of deformity then you add the normal valgus (by measuring at other elbow) to give full correction – opinions should not differ*)

2. What is the anatomical alignment at elbow?

Ans. Forearm is aligned in a valgus of 8-15º (higher value for females and lower for males) with respect to arm in full extension with a medial angulation.

3. What if the angulation is lost?

Ans. If the alignment is neutral we call it cubitus rectus 'deformity'. It is still a 'deformity' as it deviates from the normal for population.

4. Why do you call it post-traumatic malunited supracondylar fracture?

Ans. There is history of trauma with relevant treatment.
On examination:
- Irregularity over medial and lateral supracondylar ridges
- Maintained three-point relationship
- Medial epicondyle tip is higher
- Hyperextension at elbow
- No widening at intercondylar region
- Internal rotation deformity with restricted external rotation and increased internal rotation
- No deficiency in the region of trochlea.

5. Why the rotational deformities do not manifest?

Ans. The much more mobile shoulder joint compensates for the same so often they go unnoticed by patient or relatives.

6. Is it a progressive deformity?

Ans. No, it is a static deformity *(also see Q 7)*.

7. What are the other causes of cubitus varus/ what is your D/D?

Ans. Apart from above:
1. Congenital (progressive)
2. Malunited fracture lateral condyle (progressive, if due to hyperemia and overgrowth)
3. Trochlear osteonecrosis (often non-progressive)
4. Malunited intercodylar # (static)
5. Malunited medial condyle # (static).

8. Why is supracondylar fracture so common and why does it occur at this site?

Ans. The following reasons account for the same:
1. Thin wafer of bone
2. Olecranon impinges at the fossa with leverage and delivers point stress

3. Soft tissues often are lax in children which account for possible hyperextension
4. The bone is actively undergoing remodeling at the site during 6-10 years of age
5. Large number of actively growing physes in vicinity account for increased vascularity at the region and resultant relative hyperemia.
6. Periosteal attachment to olecranon fossa (results in constant transcondylar failure), the capsule is tight anteriorly and hinges the olecranon tip against fossa.

9. What is the cause for a higher frequency of postero-medial type fracture?

Ans. The following bring the distal fragment medially:
   1. Pronation of forearm at fall
   2. Eccentric pull of biceps
   3. Medial column collapse
   4. Oblique fracture line.

10. What are the displacements of a supracondylar fracture?

Ans.
   1. Medial displacement
   2. Medial tilt
   3. Internal rotation
   4. Posterior displacement
   5. Posterior tilt
   6. Proximal migration.

11. What neurovascular structures are particularly at risk in a supracondylar fracture?

Ans. Radial nerve in posteromedial type fracture displacement, median nerve in posterolateral type, anterior interosseous, ulnar (flexion type – 2-3 per cent of all supracondylar fractures are flexion type) all may be damaged often neurapraxia and that too of mixed type is seen. Brachial artery is particularly at risk in posterolateral displacement with median nerve which is further placed at risk by ulnar-sided tether of supratrochlear branch.

12. What are the complications of supracondylar fracture?

Ans. *Early:*
- Compartment syndrome
- Nerve injury
- Vascular injury

Late:
- Malunion
- VIC
- Myositic ossificans
- Cubitus varus
- Cubitus valgus
- Tardy ulnar nerve palsy
- Chronic nerve entrapment in healed callus (Metev's sign).

13. What are the clinical signs of an irreducible fracture?

Ans. Button holing of the fragment through anterior muscles particularly brachialis which is evident as puckering.

14. How do you manage the acute supracondylar fracture?

Ans. Type I fracture (Gartland's) and most of type II are managed using closed reduction and plaster slab immobilization in hyperflexion and pronation (in posteromedial type) or supination (in posterolaterally displaced fracture) [some people recommend skin traction or overhead skeletal olecranon screw traction]. Type III fractures being unstable are managed by closed/ open reduction and percutaneous pinning. {posteromedial >> common than posterolateral = 3:1}.

15. Why do you immobilize in hyperflexion?

Ans. In types I and often in type II the posterior periosteum is spared. This acts as posterior splint, additional splinting is obtained by tightening of triceps posteriorly.

16. What precaution do you take while reducing fracture and applying slab?

Ans. One needs to keep a constant check of vascularity by keeping finger over radial pulse.

17. What precaution do you take while pinning the fracture?

Ans. After closed reduction lateral pin is inserted first followed by medial pin after bringing elbow to 90° flexion (it reduces risk of ulnar nerve injury).

18. How do you look for adequacy of reduction?

Ans. For coronal plane alignment a true AP view of elbow is evaluated and compared with the normal side for Baumann's angle and intact olecranon fossa. A crude approximation is that a change of 5° in Baumann's angle will produce a 2° cubitus varus at the malunion. Other measures are humero-uoln-wrist alignment (apparently more accurate than Baumann's angle) and metaphyseal-diaphyseal angle. For rotational alignment (axial mal-alignment) one should look for any deformation in the tear drop in a true lateral view and that anterior humeral line must transect capitellum (other measures like shaft-condylar angle and coronoid line can also be evaluated). Finally saggital plane malalignment (Flexion-Extension) is also looked for in a true lateral view.

19. What is Baumann's angle?

Ans. Angle subtended between the tangent to lateral physeal line and long axis of diaphysis of humerus (normal = 64-81°; mean=72°).

20. What are the indications of open reduction?

Ans.
- Open fracture
- Irreducible fracture
- Associated neurovascular injury
- Flexion type fracture
- Delayed presentation (>1 week).

21. How do you look for cubitus varus deformity on a lateral view?

Ans. "Crescent sign" due to overlapping of capitellum on olecranon.

22. What are the associated deformities with cubi-tus varus?

Ans. Cubitus varus (medial tilt and lateral angulation) is accompanied with:
1. Internal rotation
2. Extension of distal fragment
3. Shortening
4. Medial shift

'3' and '4' are seen in severe forms only.

23. What deformities may get corrected over time?

Ans. As this deformity is not in the physiological direction of elbow so correction, if, at all, is minimal. Only extension deformity (partially) and to a minimal extent medial shift is corrected over time. This is the reason why this cosmetic deformity often persists.

24. Is the ulnar nerve at risk in cubitus varus deformity?

Ans. Not as commonly as in cubitus valgus but for sure there is reported tardy ulnar nerve palsy in cubitus varus deformity. This is due to medial shift of triceps which narrows the cubital tunnel or fibrous band across the heads of flexor carpi ulnaris.

25. How will you differentiate between a lateral condyle fracture and supracondylar fracture clinically?

Ans. The following points must be noted (see table):

Findings	Lateral condyle	Supracondylar #
Three-point relationship	Disturbed	Maintained
Arm length	Normal	May be reduced
Thickening of supracondylar ridges	Lateral only	Both sides
Instability	To varus stress	None
Fixed flexion deformity	Present	Absent

Contd...

Findings	Lateral condyle	Supracondylar #
Movements	Restrictions of rotation or flexion/ extension often seen	None often (decreased external rotation at shoulder is due to bony deformity at elbow)
Hyperextension	Restricted	Often hyperextension seen
Complications	Delayed by months to years	Usually immediate and neurovascular
Long term	Arthrosis common	Very rare

26. What is the other name of this deformity?

Ans. Gunstock deformity.

27. Why is it called so?

Ans. The deformity resembles the loading stock of old long barrel guns.

28. What will you do for this patient?

Ans. I will confirm my diagnosis radiologically and then do a modified French osteotomy (lateral closing wedge osteotomy).

29. What is the cause of fishtail deformity?

Ans. This is seen in old supracondylar fractures and is due to osteonecrosis of trochlea producing a smooth gentle curve. Trochlea is supplied by two sources. A distal fracture line disrupts the supply to lateral part of medial trochlear crista that comes through medial condyle. The other type of sharp angular wedge is seen after fracture of lateral condyle and is due to persistence of gap between lateral condular physis ossification center and medial ossification of trochlea.

30. Who modified French osteotomy?

Ans. Bellemore modified the original osteotomy described by French.

31. What is the difference between modified and original French osteotomy?

Ans. The following are the differences:

French osteotomy	Modified French osteotomy
Posterior longitudinal approach	Posterolateral incision
Detach lateral half of triceps	Whole triceps detached
Ulnar nerve explored	No exploration of ulnar nerve done
Medial periosteum hinge	Medial periosteal and bony hinge

32. How do you correct the rotation while doing osteotomy?

Ans. The proximal lateral screw is posterior to the coronal plane and distal slightly anterior, this disposition leads to external rotation of distal fragment when the wires are tightened.

33. What are the methods of fixing the osteotomy?

Ans. Screws and metallic suture wire, crossed thick K-wires, Steinman pin, external fixator, staples, plate and no fixation (POP cast only).

34. What other osteotomies do you know of?

Ans. Dome osteotomy (advantage of not producing shortening, less lateral bump), obliqe osteotomy with derotation, three-dimensional osteotomy (Uchida et al), step-cut osteotomy (Derosa and Graziano), medial opening wedge osteotomy with bone graft (King and Secor).

35. When will you plan for osteotomy?

Ans. Prerequisites:

1. At least one year following fracture (adequate duration for remodeling of bone and tissue equilibrium to regain)
2. Patient demanding surgery

36. What will you explain to the patient?

Ans. Always explain that this osteotomy is being done only for cosmetic correction and that no functional benefit

must be expected out of it. Also the lateral bump often persists after osteotomy. The complications of osteotomy should be thoroughly explained.

37. What are the complications of osteotomy?

Ans. Stiffness!, nerve injury, persistent deformity (under-correction), non-union, lazy S-deformity of elbow, etc.

38. What is pseudo cubitus varus deformity?

Ans. Lateral spur formation is one of the most common deformities after lateral condyle fracture due to lateral elevation of periosteum by displaced fragment. In patients with no real change in angle the lateral pro-minence produces appearance of mild cubitus varus.

CASE V:
CUBITUS VALGUS

1. What are the causes of cubitus valgus deformity?

Ans.
1. Non-union fracture lateral condyle
2. Malunited supracondylar fracture humerus
3. Osteonecrosis of lateral trochlea
4. Malunited intercondylar fracture
5. Radial head fracture dislocation
6. Medial epiphyseal injury and growth stimulation.

2. Why do you call it fracture of lateral condyle?

Ans. Following points favour # lateral condyle:
1. Disturbed three-point relationship
2. Lateral supracondylar ridge thickening
3. Widening of intercondylar region
4. Flexion deformity
5. Tenderness
6. Abnormal mobility of lateral condyle
7. Varus stress test positive
8. Ulnar nerve (may be involved as ulnar neuritis).

3. When do you call lateral condylar fracture to be not united?

Ans. After 12 weeks (3 months)

4. What are the components of lateral condyle?

Ans. Lateral condyle comprises of:
1. Lateral trochlear crista
2. Lateral condylar physis and epicondyle
3. Capitellum and lateral metaphyseal region.

5. Why is non-union common in lateral condyle fractures?

Ans. The following factors have been put forward:
1. Poor circulation to metaphyseal fragment: Only a very small portion of lateral condyle is extra-articular and nearly all of blood supply enters from here. Damage to the same is quite common (trauma/iatrogenic) causing non-union and osteonecrosis.
2. Fragment bathed by synovial fluid
3. Forces exerted by muscles arising from condylar fragment keeping it displaced
4. Displaced fragment with opposition of articular cartilage to the proximal fracture surface – in this scenario union is impossible.

6. Which type of fracture is it?

Ans. Epiphyseal intra-articular fracture.

7. How do you classify lateral condyle fracture?

Ans. *Milch classification:*

Type I: Line passing from metaphysis to capitello-trochlear groove traversing through ossification center, push-off injury (a″ Salter-Harris type IV) – stable, heals often with bony bar.

Type II: Fracture line passing from metaphysis coursing through physis and exiting through intercristal groove (medial to lateral crista) out of ossification center, pull off injury (a″ Salter-Harris type II) – unstable type more common type.

Jackob's classification: (Stages of displacement)

Stage I: Undisplaced with intact articular surface (Badelon modification – displacement < 2 mm)

Stage II: Complete fracture through articular surface

Stage III: Fragment rotated, displaced laterally and proximally allowing translocation of olecranon and radial head.

Finnbogason and associates (classifies only minimally displaced fractures ≤ 2 mm)

Type A: Minimal/no gap on radial/dorsal side. # not continuous to epiphyseal cartilage.

Type B: As above but # line continues to articular surface. Lateral # gap > medial.

Type C: As of B but # gap as wide medially as laterally.

8. What muscles remain attached to lateral condyle?

Ans. ECRB (major blood supply) and ECRL, the fracture line runs between ECRL and brachioradialis.

9. What are the displacements of lateral condyle?

Ans. Lateral condyle is rotated both in coronal and vertical axis. There is 180º rotation in coronal plane and 90º vertical directions totalling the displacement of 270º. Milch type II is more likely to displace and sublux the elbow whereas type I injury is more likely to rotate.

10. What is the cause of development of cubitus valgus deformity in lateral condyle fractures?

Ans. In type I fracture with healing there may be development of bony bar or arrest of lateral growth due to injury to ossification center (see classification). This leads to cubitus valgus deformity.

Type II fractures cannot be held securely in place with conservative methods being inherently unstable, so there is always a tendency towards lateral and proximal migration, hence development of cubitus valgus with non-

union. Even in united fractures there could be formation of bony bar which tethers lateral growth.

11. Why is the cubitus valgus of concern?

Ans. Not all cubitus valgus deformities will concern us, but particularly the one from lateral condyle non-union or sometimes with united # is "progressive". This may lead to changed elbow mechanics or neurological complications or both and thus concerns us.

12. What are the causes of progression?

Ans.

1. Non-union with progressive lateral and proximal migration (type II)
2. Damage to ossification center (type I)
3. Bony bar formation (type I and II)
4. Overgrowth of medial condyle (theoretical).

13. How does tardy ulnar nerve palsy develop?

Ans. Tardy ulnar nerve palsy develops due to:

1. Stretching of nerve due to medial angulation and hence resulting lengthier course
2. Friction induced perineuritis, and
3. Adhesions causing entrapment of the nerve in cubital tunnel.

14. How will you manage this patient?

Ans. I will confirm the diagnosis by getting X-ray done in AP and lateral projections and also assess the displacements, condition of physis and span of metaphyseal fragment.

15. What will you do?

Ans. I will assess the patient for surgical management in a symptomatic patient based on Flynn criteria:

- Large metaphyseal fragment
- Displacement of < 1 cm from joint surface
- Open viable lateral condylar physis

Surgical management is absolutely required for a progressive deformity, associated tardy ulnar palsy or elbow instability. The patient should be symptomatic!

16. What surgery will you do?
Ans.

- For a patient with established non-union (deformity <20º) having a large metaphyseal fragment, minimal migration and an open physis → open reduction (debride fibrous tissue to expose bone), *in situ* **fixation** in compression with a screw (cancellous/cortical) and bone grafting along with anterior transposition of ulnar nerve.
- For patients with concern of cosmetic deformity (≥ 20º) *supracondylar corrective osteotomy (medial closing/ step cut/ dome) and rigid in situ compression fixation along with transposition of ulnar nerve must suffice.*
- Patients with displaced fragments require → translocation of fragment debriding the fracture surface and rigid fixation with bone grafting and transposition of ulnar nerve. This is particularly needed when in situ fixation is useless like in conditions where the articular cartilage opposes fracture surface.
- Asymptomatic non-progressive non-union in an adult patient with tardy ulnar nerve palsy requires transposition of ulnar nerve.

    {*Some more clarifications over need and choice for open reduction:*

    Open reduction is a sort of "extensive surgery" for this non-union – often associated with complications chiefly stiffness that 'excels' the benefits from surgery. So it's a better idea on table to preliminarily fix lateral condyle with K-wires and look for limitation of movements and drop open reducion if significant limitation is produced. This is true for fragments that are moderately displaced and can be easily repositioned. For a grossly rotated fragment it's probably better to leave it 'as it is' unless grossly symptomatic explaining the risk of osteonecrosis}.

17. Why is ulnar nerve transposition always required?

Ans. Even after correction of deformity and fixation, tardy ulnar nerve palsy may develop so transposition must be done.

CASE VI:
OLD UNREDUCED MONTEGGIA FRACTURE DISLOCATION

Diagnosis

The patient is a 9-years-old male child with 2 year old unreduced Monteggia fracture dislocation. There is flexion deformity of 20º with restricted extension, supination and pronation.

1. Why do you call it old unreduced Monteggia fracture dislocation?

Ans. Findings:

- History of trauma
- Cubitus valgus
- Radial head palpable anteriorly in elbow rather than its original location
- Ulnar bow
- Foreshortening of forearm more over radial aspect
- Restricted supination and pronation.

2. What are your differentials?

Ans.

- Traumatic isolated radial head dislocation
- Congenital radial head dislocation
- Congenital radio-ulnar synostosis
- Rheumatoid arthritis (older patient)
- Recurrent radial head dislocation.

3. How do you define old-unreduced Monteggia fracture dislocation?

Ans. No strict definition, but when closed reduction is not possible then it is often called old unreduced dislocation which is arbitrarily chosen to be 3 weeks.

4. What will you do next?

Ans. I will confirm my diagnosis on AP and lateral X-rays of elbow joint with arm and forearm including wrist joint.

5. How do you differentiate this with congenital radial head dislocation?

Ans.

Findings	Congenital	Traumatic
Bilateral	Common	Uncommon
Trauma	Absent (may be present)	Often remembered (and necessary for clinical diagnosis)
Radial head shape in anterior dislocation	Dome shaped (rounded) without central depression	Normal
Radial head shape in posterior dislocations	Elongated and narrow head	Normal
Ulnar bow	Present (often palmar in anterior dislocation and dorsal in posterior)	Absent unless remodeling failed to occur
Capitellum	Hypoplastic or absent	Normal
Other congenital anomalies	Present	Absent

6. What are the possible complications of unreduced Monteggia fracture dislocation?

Ans.
- Tardy radial nerve palsy
- Tardy ulnar nerve palsy
- Progressive cubitus valgus
- Restriction of movements
- Functional impairment (weakness, stiffness)
- Arthrosis and pain over lateral aspect of elbow.

7. Which movement is more severely restricted in Monteggia fracture dislocations?

Ans. Supination

8. What nerve injuries are frequently associated with this injury?

Ans. Posterior interosseous nerve, anterior interosseous nerve, ulnar nerve in that order of occurrence.

9. How do you classify Monteggia fracture dislocations?

Ans. Bado classification of Monteggia lesion:

Type I (60%): Anterior radial head dislocation with pronated bicipital tuberosity; #ulnar diaphysis at any level with anterior angulation (children > adults)

Type II (15%): Posterior/posterolateral radial head dislocation; # ulnar diaphysis with posterior angulation (exclusive to adults)

Type III (20%): Lateral/anterolateral radial head dislocation with # ulnar metaphysis (exclusive to children)

Type IV: Anterior dislocation of radial head with # proximal 1/3rd radius and # ulna at same level.

10. What do you understand by the term Monteggia lesion?

Ans. Monteggia in 1814 described a traumatic lesion of ulna (# between base of olecranon and proximal third of ulna) with dislocation of radial head. Over time various patterns of injuries with similar mechanism of production have been included into the group "Monteggia lesions" that is defined as 'A group of lesion having common a radio-humero-ulnar joint dislocation associated with ulnar # at various levels or with lesions of wrist'.

11. What are Monteggia equivalents?

Ans. Type I and II have been classically described to have equivalents.

Type I:
1. Anterior dislocation of radial head (pulled elbow, nursemaids elbow, minimal Monteggia)
2. # ulnar diaphysis with # radial head
3. # neck of radius
4. # ulnar diaphysis with # proximal radius (always # radius proximal to ulnar #)

5. # ulnar diaphysis with anterior dislocation of radial head and # olecranon
6. Posterior dislocation of radial head with # ulnar diaphysis with or without # proximal radius
7. Wrist lesions may also be found with type I Monteggia lesions or 'equivalents'
   a. Radio-ulnar dislocation
   b. Slipped distal radial epiphysis
   c. # distal radius
   d. # distal radial diaphysis with sprained DRUJ (Galeazzi's lesion)

   Type II:
1. Epiphyseal fracture of dislocated radial head of fracture radial neck.

12. How do you look for ulnar bow?

Ans. On a lateral projection radiograph make a longitudinal line from olecranon to distal ulnar metaphysis along dorsal border. Perpendicular distance of ulna from this line of > 1 mm is significant and denotes bow.

13. What will you do for this patient?

Ans. I will do open reduction of radial head with annular ligament reconstruction and ulnar lengthening/corrective osteotomy.

14. What else would you do?

Ans. I will fix the radial head using transcapitellar wire.

15. How will you do open reduction of radial head?

Ans. Using Boyd's approach, radial head and joint are exposed. Capsule and/or annular ligament are removed if they hinder reduction and radial head is replaced into position after inspection.

16. How will you reconstruct annular ligament?

Ans. Modified Bell-Tawse (Steel and Peterson modification) using triceps aponeurosis. A drill hole is made in ulna

obliquely from medial aspect to exit from near coronoid (where original annular ligament is attached); then the tendon is passed which does not completely encircle the radial neck. This has the advantage of avoiding notching of radial head and constriction to prevent future growth disturbance and also stabilizes it more securely with posteromedial force. Immobilize in 60° supination for 6 weeks in POP cast.

17. What other materials can be used for reconstruction of annular ligament?

Ans. Natural: Central/lateral slip of triceps aponeurosis, lacertus fibrosus, forearm fascia, Palmaris tendon, chromic catgut ligature, Fascia lata graft.
Synthetic: (Not very popular) mersilene tape.

18. What are the indications of ulnar osteotomy?

Ans. Malunited ulna with shortening or angulation needs correction for reduction and maintenance of radial head in position.

19. At what level will you do the osteotomy?

Ans. At the apex of deformity.

20. Till when can you do open reduction?

Ans. As such till 12 years of age the results are good. However, there is no absolute contraindication for the procedure but complications like stiffness, dystrophic ossification are more common. In asymptomatic children without neurological compromise or progression of deformity masterly inactivity is the best option. However, for any of the above one can give a fair trial of reduction. In skeletally mature patients radial head excision can be safely done. Done in an skeletally immature patient the progression of deformity and consequent nerve injury are common.

The question can have other meaning of asking time limit after injury till which reduction can be done. For this there

is again absolutely no duration limit. The only recommendation is ASAP. Reductions done within 3-6 months have a good prognosis, results however, are not disappointing for longer dislocations.

21. What are the contraindications of doing reduction and repair?

Ans. Deformed radial head, flattened capitellum, valgus deformity of radial neck.

Wrist and Hand

EXAMINATION POINTS FOR A HAND CASE

{The topic is difficult with respect to the examination as various specifications are to be remembered and detailed. Precision and tests are tough to learn.
Read: 4-6 times for MS and DNB candidates}

History taking

(In a broad division try to first ascertain what you are dealing with – deformity/neurological condition/painful or inflammatory condition, then it will be prudent to proceed):

- *Pain:* Onset (injury/spontaneous; acute/insidious), type, location, duration, remote injury (RSD), aggravating and relieving factors, activity restrictions, guarding of wrist and hand.
- *Swelling:* Onset (as above), location, duration (also whether temporary or permanent), relieving and aggravating factors, flare-ups (severity, frequency, duration), previous treatment if any, association with fever/stiffness, discolouration.
- *Deformity:* As above + abnormal contours, palm and finger alignment.
- *Loss of function:* (A key factor as hand in itself is an organ, 45 per cent of hand function is utilized for grasp, 45 per cent for pinch (key, tip, chuck pinch), 5 per cent for hook function and in rest 5 per cent hand functions as a paper weight (most primitive function))
- *Loss of power:* Any decreased strength or dexterity.
    Apart from above it is very important to ask for history of hospitalizations and treatment (type, dose, frequency, response and side effects), details of trauma if present, dominant hand, effect on occupational functioning and ADL.

Examination

Inspection (keep the hand on a pillow with whole ipsilateral and contralateral upper limb exposed):

Alignment

- Whether viewed from dorsal or volar aspect with fingers and thumb in adduction – the forearm, wrist and hand (middle finger) should be in a straight line (axial alignment). Ulnar deviation of MCP joint and radial deviation of wrist are observed in rheumatoid arthritis (RA).
- Dislocation of PIP joint is visible as a step off (sagittal malalignment). Rupture of extensor slip in RA produces flexion deformity of fingers at MCP joint (Vaughan-Jackson lesion). Dinner fork deformity is seen in Colle's fracture.
- Rotational malalignment is judged in two ways:
  – Ask patient to partially flex the fingers together at MCP joint – nails of index finger and those of ring and little finger face away from that of long finger in supinated hand.
  – Ask patient to flex fully each finger in turn – the fingers should unfailingly point towards the scaphoid tuberosity.

Dorsal aspect:
- *Nails:* vasculitic changes (local infarction – RA), splinter haemorrhage, periungal telengiactases (SLE, scleroderma), pin-size pitting (psoriasis), hyperkeratosis, onycolysis, discolouration, ridges, anaemia, dilated capillary loops over nail fold, paronychia, subungal haematoma.
- Fingers (examine from DIP to MCP): Mallet finger (avulsion of EDC, EPL for thumb), redness, sausage shaped digits, nicotine stains, arthritis mutilans, tophi, swan neck/ boutonniere deformity, Z-deformity of thumb, Bouchard's nodes, Heberden nodes, Garrod pads, ulnar deviation, benediction attitude, clawing of fingers, contractures, telengiectasia, mucous cyst, inclusion cyst, sebaceous cyst, skin and appendages (normally there are no hair from MP and distal).
- Hand: Dropped knuckle (# metacarpal), fore-shortening of metacarpal, wasting in first web space, wasting of interossei, skin and appendages, carpal bossing.
- Wrist: Head of ulna (prominent in pronation disappears in supination), silver fork deformity, ulnar deviation, volar

subluxation, volar-ulnar subluxation, cystic swelling (ganglion).

Radial aspect:
- Thumb and 1st MCP joint (basilar joint): (Skier's thumb or gamekeeper's thumb), swelling (1st MCP arthritis).
- Wrist: Anatomical snuffbox (bound dorsally by EPL and volarly by APL and EPB) for swelling.

Volar aspect:
- Fingers and palms: As above + pulp spaces for pits (Raynaud's phenomenon) and swelling (Felon), swelling over volar aspect of finger (GCT tendon sheath or cystic swelling), jersey finger (avulsion of FDP), flexion arcade of fingers, palmar skin for pits and cords/ nodules (Dupuytren's contracture), signs of flexor tendon sheath infection (Kanavel's signs – fusiform swelling extending along MP and PP into distal palm + tenderness along volar aspect + finger held in flexed position + passive extension causes pain), thenar and mid palmar spaces for infection, thenar and hypothenar wasting.
- Wrist: As above + tendon of Palmaris longus (ask patient to touch the tips of thumb and little finger and flex the wrist – tightens the palmar fascia and makes tendon prominent), FCR, FCU, compound palmar ganglion.

Ulnar aspect:
- Hypothenar wasting, head of ulna (capita ulna syndrome – in RA the volar subluxation of carpals and dorsal subluxation of ulnar head accentuates the deformity with prominent head of ulna).

Palpation

Make a note of temperature.
Dorsal aspect
- *Fingers:* for mallet finger, collateral ligaments especially for thumb, joint swelling and effusion, MCP joint for collateral ligament tenderness.

- Metacarpals for deformity, tenderness, first metacarpal base for Bennett's fracture.
- Wrist for radial styloid, De Quervain's disease (tenovaginitis of 1st dorsal compartment – APL, EPB), lister tubercle (2 cm ulnar to radial styloid), anatomic snuff box (for dorsal branch of radial artery, # scaphoid, 3-4 mm distal to it is basilar joint), 2nd dorsal compartment (ECRL, ECRB, intersection syndrome, ganglion is most likely to occur here), 4th dorsal compartment (EDC tendons in RA), TFCC just distal to ulnar styloid.

Palmar aspect
- As in inspection confirm the findings of felon, flexor sheath infection, mid-palmar space, thenar space
- Swelling of ganglion, GCT tendon sheath, trigger finger (catching of tendon in pulley)
- Tubercle of scaphoid, hook of hamate, pisiform, pisiohamate ligament and Guyon's canal, FCR, median nerve (between Pl and FCR), volar carpal ligament (proximal limit corresponds with the distal radial crease).

Movements

Wrist motion (with elbow flexed in 90º) – dorsiflexion (60-70º), palmar flexion (60-80º), radial (20º) and ulnar deviation (30-40º)

Finger movements: Flexion at DIP (70-90º), PIP (110º), MCP joint (80-90º). Hyperextension at MCP joint is quite normal and is maximum at Index finger (up to 70º). Other method is to measure the finger tip to transverse palmar crease distance for serial assessment and follow-up. Restricted flexion at IP joints can be due to capsular contracture/ intrinsic contracture or extrinsic tightness. Perform Bunnell-Littler test to differentiate the two. Passively flex the IP joint with MCP in extension followed by MCP in flexion. Restriction due to intrinsic muscle tightness will increase the passive flexion at IP joint with MCP flexion due to relaxation of intrinsic muscles. In extrinsic tightness the flexion decreases due to tightening of structures with MCP flexion hence restricting flexion further.

Abduction and adduction: Abduction and adduction is possible at MCP joint due to "cam effect" – the metacarpal heads are shaped like cam (larger volar articular component as compared to linear distal) so that in extension the collateral ligaments are lax allowing movement in coronal plane (this is also the reason for 'James position' of immobilization). This is not possible at IP joints – bicondylar joints without a 'cam'. The presence of abduction and power of adduction is more important than the range of abduction! (just compare with other side).

Thumb movements: Flexion, extension, adduction, abduction, opposition

Motor power and muscle/tendon function:
- Wrist flexors (FCR/ FCU), extensors (ECRL, ECRB, ECU), radial deviators (FCR, ECRL, ECRB, APL), ulnar deviators (ECU, FCU)
- Finger flexors (FDP – hold PIP individually and ask patient to flex DP; FDS – ask patient to flex the PIP while holding other three fingers in extension, this defunctionalises the FDP as it is a single unit muscle). This test is less reliable for index finger as the FDP to IF may be separate.
- Abduction (dorsal interossei) and adduction (palmar interossei) of fingers, card test.
- Thumb flexion (FPL, FPB), radial abduction or extension (APL), palmar abduction (APB), adduction (Adductor pollicis), opposition (APB, OP), froment's test (Adductor pollicis)

Sensation Testing

Percussion: *Tinel's test*
Vascularity
Neurological examination for various nerves (*See Chapter 7 : Case I*)

Special tests:
- Carpal tunnel compression tests
  1. Tinel's sign
  2. Phalen's test: Wrist flexed by gravity for 60 sec

3. Durkan's median nerve compression test: Manual pressure over median nerve at carpal tunnel for 30 sec
4. Reverse Phalen's test: Wrist and fingers extended for 2 minutes
5. Tourniquet test: Arm tourniquet inflated above systolic pressure for 60 secs
6. Hand elevation test: Hand elevated above for 60 sec
7. Wrist flexion and carpal compression test: In a supinated forearm with flexed wrist compress the median nerve with direct pressure
8. Closed fist test: Tight fist for 60 sec

- Stability testing:
  1. MCP joint
  2. IP joint
- Carpal instability:
  1. Scaphoid shift test of Watson: Tests scapholunate instability
  2. Luno-triquetral ballottement test for lunotriquetral joint
  3. Mid-carpal instability
- DRUJ instability:
  1. Piano key test
  2. DRUJ compression test
- TFCC test: TFCC compression test
- Grind test for basilar (trapezio-metacarpal joint) joint arthritis
- Finkelstein test for de Quervain's disease.
- Allen's test for radial and ulnar arterial circulation.

CASE I:
PERIPHERAL NERVE INJURIES

{This is a very important and favourite topic that is bound to come if case is present. The diagnosis is simple and straightforward to those who have practiced the case otherwise not only examination or diagnosis but the management will also be formidable!

Read: 6-8 times (MS Orth and DNB candidates}

Diagnosis

The patient is a 28-year-old male with post-traumatic, non-recovering, complete right high radial nerve palsy for past 9 months (with or without non-union/ united fracture of humerus).

1. What do you mean by high/low nerve (radial/median/ulnar) palsy?

Ans. *Radial nerve:*

Complete palsy (very high): Triceps paralysed (injury in axillary region)

High: Triceps and often anconeus preserved but rest all paralysed (injury around radial groove till it pierces septum).

Low: BR and ECRL preserved (≡ posterior interosseous nerve (PIN) palsy; ECRB in 58 per cent cases is supplied by PIN so it may also be spared in some cases).

For ulnar and median (*See Chapter 7 : Case II; Q 21-23*)

2. Why do you call it non-recovering?

Ans. The nearest muscle to the site of injury (Brachioradialis) has not been reinnervated and the tinel's is still localized at the site of injury.

3. What function is lost in nerve (radial/median/ulnar) palsy?

Ans.

Radial:
1. Inability to extend fingers (1,2,3,4,5) {low} and wrist {low+high}
2. Inability to stabilize the wrist (wrist drop) and thumb (radial abduction of thumb) {low+high}
3. Loss of grip strength (accessory forearm flexion) {High}
4. Accessory forearm supination {Very High}
5. Sensory loss (radial 2/3 dorsal sensation)
   Ulnar (also *See Chapter 7: Case II; Q21, 23, 29, 30*):
1. Loss of grip strength (impairment of power grip > precise grasp) {High}

2. Flexion of distal phalanx 4,5 {High}
3. Digital balance 4,5 {High}
4. Loss of finger function (flexion (partial), adduction, abduction) {High+Low}
5. Loss of thumb adduction and weakness of thumb flexion {High+Low}
6. Sensory loss (medial 1½ digits – Low; Ulnar 1/3 volar – High)

   Median nerve:
1. Loss of thumb opposition, finger stabilization 1,2 {Low+High}
2. Weakness of wrist and partial loss of finger flexion 2,3; complete for 1 {High}
3. Forearm pronation {High}
4. Sensory loss (Radial volar 2/3 hand)

   Combined low median and ulnar nerve:
1. Thumb opposition+ adduction + flexion (partial)
2. Finger abduction and adduction
3. Finger stabilization
4. Sensory loss (volar hand + dorsal ulnar 1½)

4. What is the course of radial, ulnar and median nerves?

Ans. *Radial nerve (C5-T1):* Formed in front of subscapularis from posterior cord → passes anterior to lattissimus dorsi muscle → to pass through triangular space accompanied with profunda brachii artery to posterior aspect of humerus into radial groove (not spiral groove; radial groove is few mm above spiral groove) where it is separated from bone by medial head of triceps coursing obliquely laterally → pierces the lateral intermuscular septum around 122 mm above lateral epicondyle → passes into anterior compartment emerging beneath brachioradialis → deep branch (PIN) pierces supinator muscle to emerge into extensor compartment of forearm → forms cauda equina of spinner 8 cms distal to elbow joint.

Ulnar nerve (C8-T1, rarely C7 also): Formed from medial cord of brachial plexus → runs inferomedial to axillary artery to continue behind brachial artery over triceps muscle → passes straight to posterior aspect of medial epicondyle in "ulnar groove" between medial epicondyle and olecranon process → passes between two heads of FCU (the so-called "cubital tunnel" better called humero-ulnar aponeurosis → remains deep to FCU overlying FDP muscle accompanied with ulnar artery → passes superficially; emerging beneath FCU to reach Guyon's canal at wrist.

Median nerve (C6-T1): Formed from medial and lateral roots of median enters arm in close relation to brachial artery. In the cubital fossa nerve lies lateral to brachialis tendon and passes b/n two heads of pronator teres → gives AIN → continues in forearm sandwiched b/n FDS and FDP → emerges just proximal to wrist b/n FDS and FCU → passes through carpal tunnel → lies anterior and radial to FDS tendons → divides in hand into recurrent motor and sensory branches.

5 What will you do for this patient?

Ans. I will do a thorough examination of motor, sensory loss. Look for reinnervation (e.g., brachioradialis for motor recovery – radial, FCU for ulnar, etc. and advancing tinel's for nerve regeneration). I will also document the findings with EDS. I will get X-rays of the arm in AP and lateral projection to look for status of fracture.

6. What are the requirements of a patient with radial nerve palsy?

Ans.
1. Wrist extension
2. Finger (MCP) extension
3. Combination of thumb extension and abduction
   (*What is available? - motors innervated by ulnar and median nerve)*

7. What about sensory deficit?

Ans. The sensory deficit is notable but really not a disability and is partially covered up by lateral cutaneous nerve of forearm. So it can be neglected unless there is painful neuroma.

8. How does status of fracture affect the treatment?

Ans. Osseous union should be promptly addressed to stabilize the limb and prevent additional injury to soft tissues especially nerve. In this case I will have to prepare for bony surgery also, according to the findings. Had the duration been less (say 3 months) then I would have fixed the bone along with exploration of a non-recovering nerve. Fracture in acceptable position with recovering nerve would have been managed with POP immobilization otherwise surgery to fix fracture was indicated even in recovering nerve.

9. What is the role of non-operative treatment?

Ans. Goals:
1. Maintaining full ROM.
2. Preventing contractures particularly of 1st web space.
   It is undertaken while waiting for spontaneous nerve recovery and obviously in the interim period to surgery. Usually a dynamic cock-up splint (external splint) is used for this purpose. However, internal splintage can also be done.

10. How will you treat this patient?

Ans. I will do tendon transfer.

11. What are the prerequisites for tendon transfer?

Ans. In order of importance:
1. Correction of contractures (all joints must be supple)
2. Adequate strength of the transferred tendon
   a. 85 per cent of power is a must (Steindler) – graded as good power

    b. "Omer" stated that a muscle loses at least one grade of strength after transfer so for useful post-operative movements 4/5 power is a must.

3. Straight line of pull: No pulley is ideal or minimum number of pulleys should be made.
4. One tendon – One function, i.e., flex or extend
5. Synergism: Synergistic transfer should be preferred as much as possible, however, there are a lot of violations of this rule.
6. Expendable donor: There should be no functional morbidity following use of a tendon
7. Tissue should be in equilibrium (tissue equilibrium – termed by Steindler)
   a. Soft tissue induration should subside
   b. No reaction in wounds
   c. Joints are supple
   d. Scars should be as soft as possible
8. Pass tendon below fascial planes/sheaths and not below incision line/scar (best b/n subcutaneous fat and fascial sheath).
9. Amplitude of transferred tendon should be as near to the original tendon for which the transfer is being done.
10. Try preserving the nerve and vascular supply to muscle and vascular supply to tendon.
11. Insertion of the tendon should be as close to the insertion of paralyzed tendon; at same angle; if split transfer then keep both slips in same tension.
12. Try to restore sensibility of distal organ before treatment.
13. Arthrodesis/joint procedure should be done before tendon transfer.
14. The disorder should be a non-progressive one.
15. Keep dissection to a minimum around the muscle to be transferred and achieve meticulous hemostasis to prevent adhesion formation.

12. What if the amplitude of transferred tendon is less than the tendon for which transfer has been contemplated?

Ans. Amplitude can be increased by two ways:

1. Converting a monoarticular muscle into bi/multiarticular, e.g., (FCU/FCR → EDC). The effective amplitude of motion is enhanced due to tenodesis effect by active volar flexion of wrist.

2. Muscle release from surrounding, e.g., brachioradialis transfer (*violates pt. 15 Q 11*).

13. When will you do a tendon transfer?

Ans. In general, till the time a functional recovery is expected to occur; one can wait else do a tendon transfer.

Early transfer can be done if there is gap > 4 cms or there is excessive scarring/skin loss over the nerve, some may also include injection palsy and gunshot injuries in this category. Otherwise, transfer is usually done after waiting for around 1 year following injury (whether an interim repair has been attempted or not). (Remember the exact time for transfers as such vary according to the nerves involved and surgeon's preference).

14. Why do you wait for a year?

Ans. I will give adequate time for the nerve to regrow and possibly re-innervate the muscle. Neuro-muscular junctions in the muscle degenerate if not re-innervated in about a year's time, so it is prudent to do tendon transfer after year as the likelihood of regaining function is meager. Moderate to severe atrophy of muscles is seen by 3 months, mod-severe fibrosis seen by 11 months, beyond 3 years there is fragmentation and disintegration of muscle fibers; hence ideal reinnervation can be expected after 1-3 months, functional reinnervation up to 1 years, no reinner-vation >3 yrs.

15. What are the various tendon transfers for radial nerve injury?

Ans. I. JONES transfer *(violates point no. 6 Q 11)*:

Classical (1916):
1. PT → ECRL and ECRB;
2. FCU → EDC III-IV;
3. FCR → EPL; EDC II, EIP

Classical (1921):
1. PT → ECRL and ECRB
2. FCU → EDC III-IV
3. FCR → EDC II, EIP, EPL, EPB, APL

Modified JONES: PT → ECRB and rest as above

II. FCR transfer (of Starr; Brand; Tsuge)
1. PT → ECRB
2. FCR → EDC II-V
3. PL → rerouted APL

III. Boyes Transfer (Superficialis transfer)
1. PT → ECRB
2. FCR → APL and EPB
3. FDS III → EDC II-V via interosseous membrane
4. FDS IV → EIP; EPL via interosseous membrane

IV. FCU Transfer (Standard Transfer, this is not modified Jones transfer)
1. PT → ECRB
2. FCU → EDC
3. PL → rerouted EPL

16. Which one will you do?

Ans. {Look for PL and ECRB power in examination}. I will do FCU transfer. (*If PL is absent then say Boyes transfer. If the innervation to ECRB is not lost then PT transfer is unnecessary* ☺)

17. What options are available if PL is absent?

Ans. Options in the order of preference:
1. Do Boyes transfer.
2. Substitute FDS III and IV for PL (Tsuge and Goldner)

3. Use brachioradialis (*possible only in PIN palsy*)
4. Use FCU transfer to both thumb and finger extension *(violates pt no. 4)*.

18. What is meant by rerouted EPL?

Ans. It means that EPL is taken out of dorsal retinaculum (in the region of snuff box and junction is made between PL and EPL). This gives a combination of abduction and extension force on thumb.

19. What are the problems with standard transfer (FCU transfer)?

Ans. The main problem is of excessive radial deviation which is due to:
1. Removal of the only preserved ulnar deviator (FCU) in radial nerve palsy.
2. PT → ECRB transfer. ECRB is a radial deviator (albeit less than ECRL)
3. In PIN palsy ECRL may be spared that leads to excessive radial deviation.

20. How will you avoid them?

Ans. Mainly by two methods:
1. Planning itself: If you are too concerned by radial deviation then do FCR/Boyes transfer
2. Alter the insertion of tendon by centralizing the ECRL (attach to IV metacarpal) or alternately one may attach the proximal resected end of ECU to (PT → ECRB) transfer which is not preferred as it limits total excursion.

21. What is internal splint and what is its rationale?

Ans. *Principles*:
1. Should not ↓ function in remaining hand
2. Should not create deformity
3. Should be phasic or capable of phasic conversion
   E.g., PT → ECRB transfer described by Burkhalter for radial nerve palsy.

Indications:

1. Substitute function during nerve regrowth eliminating the need of splintage.
2. Helper following reinnervation → aiding power of normally innervated muscle
3. Substitute in cases where results of repair are poor or nerve irreparable.

22. Which type of nerve is radial nerve?

Ans. Radial nerve is a primary motor nerve with a small sensory component. Ulnar and median nerves are mixed nerves.

23. Does the type of nerve have any impact on outcome of repair?

Ans. Yes, primary functioning nerves whether sensory or motor have a better outcome as the chances of cross union and resultant fiber atrophy are minimum and also single modality (sensory/motor) dominant nerves have similar fibers which reform better.

24. What is Holstein Lewis fracture and what is its importance?

Ans. Spiral fracture of M/3rd and L/3rd junction of humerus with proximal spike over lateral aspect just at the site where radial nerve touches humerus. Commonly radial nerve palsy was thought to be associated with fracture.

25. What will you do for a patient who comes with fracture of humerus with radial nerve palsy?

Ans. I will wait for return of nerve function as according to Steindler's formula for nerve regeneration [(1mm = 1Day) + 30 days for motor recovery to manifest (N-M junction) and nerve ends to sprout]. Usually for, e.g., nerve injury is 90-120 mm above lateral condyle. Now for brachio-radialis to get reinnervated that arises 20 mm above lateral condyle it will take 70-100 days + 30 days = 4-5 months. This is the time duration one can wait (calculate as for other fractures). This is based on the

premises that often the nerve injury is apraxia or axonotomesis with nerve in continuity and fair recovery is seen in over 90 per cent cases.

26. Will you always follow this regime 'or' will you always prefer to wait?

Ans. No, I will do early exploration of radial nerve for palsy associated with:
1. Radial nerve injury secondary to manipulation of fracture (absolute indication)
2. Open fractures
3. Fractures in which satisfactory alignment is not possible by closed methods
4. Fractures with associated vascular injury
5. Patients with multiple trauma.

27. If you find a gap on exploration what will you do?

Ans. I will see whether nerve ends can be coapted without tension. If not then I will mobilize the nerve and still if not possible then I will do nerve grafting.

28. How will you assess the tension at suture site?

Ans. Wilgis → take a single suture bite with 8-0 suture and if a knot cannot be tied without tension (or if it breaks) then there will be unacceptable tension at suture site (or a gap >4 cms).

Millesi → gap of ≥ 2.5 cms after keeping the limb in functional position indicates possibility of tension.

Elbow flexion >90° or wrist flexion >40° required for nerve approximation indicates tension.

Brooks: If gap cannot be closed after mobilizing the nerve then there is bound to be tension.

29. How will you overcome nerve gap?

Ans. Following in isolation or combination are often required:
1. Mobilization
2. Transposition
3. Limb positioning

4. Resection osteotomy
5. Nerve stretching and bulb suture (neuroma to glioma suture)
6. Neuromatous neurotisation (e.g., intercostal nerve for brachial plexus)
7. Nerve grafting
8. Nerve crossing (ulnar → median)
9. Addition of non-neural tubes (e.g., vein segment).

30. How much mobilization can be done for a nerve?

Ans. Depends on the type of nerve but in general mobilization >6-8 cms ↓ perfusion. (≈ 8 per cent tension ↓ venular flow, 10-15 per cent - tension: blood flow arrest).

31. What are various types of nerve grafting?

Ans. The various options are:
1. Trunk grafting using full-thickness segment of major nerve trunk (disadvantage – central necrosis/total graft dissolution)
2. Cable graft using multiple strands of cut nerve sewn at both ends (drawback – wastes axons and ignores anatomic localization of function)
3. Pedicle grafting often preferred for high combined ulnar and median nerve palsy where ulnar nerve is used as a pedicle graft to repair median nerve.
4. Interfascicular nerve graft (group fascicular nerve grafting)
5. Individual fascicular nerve grafting – often done for paucifascicular nerve, e.g., ulnar nerve at elbow or for thin/terminal nerves, e.g., motor thenar branch of distal digital N.
6. Free vascularised nerve graft.

32. From where one can harvest nerve for graft?

Ans.
1. Autogenous:
   a. Lateral cutaneous nerve of thigh
   b. Medial brachial and antebrachial cutaneous nerve

  c. Radial sensory nerve
  d. Sural nerve (up to 40 cms of graft)
  e. Lateral cutaneous nerve of forearm (up to 20 cms)
  f. Terminal branch of PIN (for digital nerves)
2. Autologous vessels and muscle
3. Allograft nerve
4. Artificial conduits (veins/collagen conduits).

33. What if you find a sharp cut on exploration?
Ans. I will do nerve repair.

34. What are various types of nerve repair you know of?
Ans. *Again a tricky question, begin by speaking one of the following – if unacceptable switch to other!*
   Depending on duration from injury:
1. Primary repair – within hours
2. Delayed primary – within 5-7 days
3. Secondary – any repair > 7 days
   Depending on the technique used:
1. Epineural
2. Group fascicular
3. Individual fascicular (funicular).

35. What do you mean by conditioning effect?
Ans. In clean sharp injuries a delay of 2-3 weeks was often advocated on the premises that a 'primed' neuron will regenerate faster at its peak metabolic activity due to "conditioning effect". Conditioning effect presupposes that axons regenerate quickly if they have been damaged previously.

36. What is the structure of a peripheral nerve?
Ans. Epineurium has two parts-internal and external, former permeates the nerve ensheathing individual fascicles and the latter is a condensation of collagen encasing the fascicles as a group. Epineural fibrosis and scar formation after nerve injury is a function of epineurium.

Perineurium is an extension of blood-brain-barrier made up of up to 10 concentric lamellae of flattened cells that are 'dove-tailed'. Removal of perineurium causes nerve function to fail. Up to 15 per cent stretch, there is no injury to perineurium but it fails for stretch >20 per cent. Endoneurium acts as a packing material of collagen tissue. There is no elastin. Participates in the formation of Schwann cell tube.

37. What is fascicle?

Ans. Termed funiculus by Sunderland it is the smallest unit of nerve that can be manipulated surgically.

38. What do you understand by Wallarian degeneration?

Ans. It is the reactive change of a nerve to injury whereby distal stump is cleared of axoplasm and myelin along with regenerative changes in proximal stump. It is initiated by macrophage ingrowth stimulating Schwann cells. Schwann cell proliferation peaks around 3rd day and continues up to 2 weeks. The distal stump once cleared by Schwann cells and macrophages is left in the form of a tube that shrinks in size. Proliferating Schwann cells form 'bungers bands'. The proximal stump degenerates till nearest node of ranvier from where new sprouts grow (2-5 sprouts within 6 hrs). Those sprouts that establish end organ contact persist rest all (those entangled at scar site 'scar delay' and those missing their 'receptive tubes') are pruned away.

39. What is topographic sensitivity?

Ans. Reinnervation of correct muscle within motor system or correct patch of skin in sensory system.

40. How do you classify nerve injuries?

Ans. Seddon's classification:
Neurapraxia (≡ 'Sunderland's type I); only physiological disruption of nerve function – recovers of its own.

Axonotomesis (≡ 'Sunderland's type II-IV); physiological disruption with partial anatomical (increasing) disruption of nerve – recovery possible (70%) but later may need surgical intervention in higher grades.

Neurotemesis (≡ 'Sunderland's type V); complete anatomical and physiological disruption of nerve – always needs surgical intervention.

41. What is type VI nerve injury?

Ans. Combination of types I-V (added by Mackinnon)

42. What is intrinsic minus hand?

Ans. *Fingers:* Hyperextension at MCP joint + flexion at IP joints ± adduction of fingers
Thumb: adduction and hyperextension at MCP joint + Flexion at IP joint.

43. What are synergistic muscles?

Ans. Synergism is the endless repeated coordination of anatomically different groups of muscles to perform a given action e.g., wrist extensors + finger flexors + finger adductors "or" wrist flexors + finger extensors + finger abductors.

44. What is the earliest sign of nerve recovery?

Ans. Advancing tinel's sign

45. What is Tinel's sign and what is its utility?

Ans. Paresthesia (*fornication*) experienced along the nerve distribution *(not at the percussion site)* on gentle percussion from distal to proximal over the nerve (1917, Jules Tinel)

Cause: Bare young hyper-excitable unmyelinated sprouts from injured proximal end.

Seen in: Sunderland's grades II-V.

Importance:

1. Advancing Tinel's can be used to calculate and gauge progression of recovery (spontaneous or following repair)

2. Non-progressing Tinel's indicates interruption of nerve regeneration

3. Static Tinel's at injury site and one present distally also indicates poor prognosis.

4. *Advancing* Tinel's seen only in grades II and III. (IV and V grades show it only after repair)

   Fallacies:

1. Few sensory sprouts and partial regeneration may give a false positive Tinel's sign.

2. No estimate of motor recovery should be made from Tinel's sign as it indicates sensory recovery from sensory sprouts.

3. Single time estimation is also useless and needs to be regularly followed.

46. What do you mean by autonomous zone, intermediate zone and maximal zone?

Ans. Autonomous zone is the area exclusively (no other nerve has interference in this zone) supplied by nerve in question. While clinically testing for sensory loss a somewhat larger area is outlined that contains overlap from other nerve and this is intermediate zone. In actual there is a still larger area that can only be tested by blocking the other nerves, however, it is clinically irrelevant as it has predominant brain representation by other nerve, this is maximal area.

47. What are the autonomous zones of radial, ulnar and median nerves?

Ans. Radial nerve: Coin shaped region over the dorsum of stretched 1st web space.

Median nerve: Volar distal aspect of index finger.
Ulnar nerve: Volar ulnar aspect of distal little finger.

48. What is EDS?

Ans. EDS (electro diagnostic studies) comprises of EMG (electromyography), NCS (nerve conduction studies) and

Strength-duration curve. Normal muscle is electrically silent on EMG (embryonic muscle show fibrillation till ≈ 6 weeks of foetal life). Denervated muscle starts showing fibrillation potential by 18-21 days (three weeks). If renervation occurs fibrillation potential decreases and motor unit action potential (MUAP) of low magnitude appear. Giant MUAP are seen in a partially denervated muscle which is additionally reinnervated by nearby nerve.

Normal conduction velocity ≈ 50 m/sec (slightly more in sensory nerves), demyelization reduces speed; unmyelinated fibers have ≈ 10 m/sec of conduction velocity. Sunderland type I injury may show delay at the site of injury but otherwise NCS and EMG is normal.

49. What is strength-duration curve?

Ans. A graph plotting the intensity of electrical stimulus to the length of time it must flow to produce response.

50. What is chronaxie and rheobase?

Ans. Rheobase (Rheos = current or flow; base = foundation) is the minimal amount of stimulus strength that will produce a response when applied indefinitely (practically a few milli-seconds).

Chronaxie (chronos = Time; axie=axis) is the stimulation duration that yields a response when stimulus strength is set to exactly 2 × rheobase.

51. What is F-wave and H-reflex?

Ans. They are part of NCS (others are motor and sensory CS): F-wave – stimulation of motor nerve and recording action potential from muscle supplied by it. The stimulus travels up the nerve to spinal cord then back to limb. It measures the conduction b/n nerve and spinal cord (others measure conduction within limb).
H-reflex – in this case afferent impulse travels up the sensory nerve and travels down motor nerve to produce discharge.

52. What is the importance of upward kink in SD curve?

Ans. It indicates partial denervation.

53. What do you mean by opposition of thumb?

Ans. (1) Thumb abduction → (2) MCP flexion → (3) Internal rotation and pronation → (4) Radial deviation of proximal phalanx → (5) Motion of thumb towards fingers.

54. What tendon transfer will you do for low ulnar nerve palsy?

Ans. Reconstruction system:

Requirements:
   i. Thumb adduction
   ii. MCP flexion

Available:
   i. Wrist extensors
   ii. FDS
   iii. Index proprii (EIP)

Transfers:
   1. Thumb adduction: ECRB → abductor tubercle (Smith/Boyes).
   2. MCP flexion with integrated IP flexion: ECRL (Brown's 4 tailed; EF4) or FCR (if flexion contracture at wrist); tendon grafts passed volar to transverse carpal ligament attached to A2 pulley or radial band of dorsal apparatus *(see Chapter 7: Case II)*.
   3. Palmar arch and adduction of little finger: EDM tendon split and ulnar half transferred to radial collateral ligament of PP or radial band of dorsal apparatus.
   4. Thumb-Index finger 'tip pinch': Accessory slip of APL to 1st dorsal interosseous and MP joint arthrodesis. If MP joint already arthrodesed EPB may be transferred.
   5. Volar sensations: Proximal medial digital nerve translocated to distal ulnar digital nerve.

55. What are methods for high ulnar nerve palsy?

Ans. 1, 2, 3, 4, 5 as above +:

1. Wrist flexion (ulnar side) {*not frequently done*}: FCR → FCU or PL → FCU

2. DIP flexion for RF and LF: FDP (IF and MF) → tenodesed to FDP (RF and LF).

56. Which transfer is done to obtain opposition with adduction of thumb?

Ans. FDS (RF) transfer through pulley of volar carpal ligament (Royle Thompson); *remember that FDS transfer can be done for low ulnar nerve palsy in place of one described above but for high ulnar nerve palsy FDP is paralyzed so this is contraindicated. It's always more comfortable not only to practice and master one technique but for exams also to remember only one method and not get confused!* Other very commonly asked transfer is littler's ("muscle") transfer of Abductor digiti minimi.

57. What reconstructive method will you use for combined low median and ulnar nerve palsy?

Ans. This is a severe injury with complete volar anesthesia and intrinsic palsy. Make the hand supple first and do transfers as below:

1. Finger intrinsic function: Brand's EF4 using ECRB or Brown's EF4 using ECRL.

2. Opposition for thumb: Do Riordan's transfer (FDS through FCU pulley) or Burkhalter transfer (EIP). But it is better to remember thumb adduction as suggested in low ulnar palsy as it also works for high median and ulnar and high median and low ulnar combined palsies: Of course this does not give opposition! *(remember – we do not have perverted memories – make it simple).*

3. Sensory as above.

58. What is combined high median and ulnar nerve?

Ans. What patient loses?

1. Sensation of hand

2. Thumb abduction/opposition
3. MP joint flexion
4. Finger flexion
5. Wrist flexion
   What must be replaced?
1. Sensation of hand
2. Thumb abduction/opposition
3. MP joint flexion
4. Finger flexion
5. Wrist flexion

   Transfers:
   Thumb adduction: ECRB → adductor tubercle
   Thumb opposition → Burkhalter (EIP)
   Finger flexion: ECRL → FDP and tenodesis of all fingers
   Wrist flexion: ECU → FCU
   But this may make extension of wrist quite weak, so one may do wrist arthrodesis that spares all wrist extensors for transfer quite safely.

59. What is Camitz transfer?

Ans. PL → APB, but done only in partial low median palsy to provide opposition (in reality a pseudo-opposition) e.g., carpal tunnel syndrome.

60. What is the cause of nerve 'palsy' in postinjection nerve palsy?

Ans. As such injection into or through nerves should not cause palsy, however, some drugs act as fibrosing agent (e.g., tetracyclines, preservatives with injectable diclofenac) that lead to 'progressive' fibrosis. This constricts the nerve and cause palsy.

61. What is the treatment?

Ans. Do not wait, and if the patient presents early > 3 weeks do neurolysis (endoneurolysis). Late cases (> 3 months) showing no recovery should be treated with tendon transfer. For immediate presentation one can wait for up

to no longer than 3 weeks to give benefit for recovering from possible neurapraxia due to local nerve injury or oedema.

62. How do you classify fibrosis?

Ans. Millesi Types (added to Sunderland classification):
A. Epifascicular epineurium involved.
B. Interfascicular epineurium involved.
C. Endoneurium involved.
   Type I/II and A/B need neurolysis.

63. What are the types of neurolysis?

Ans. Epineural, perineural (interfascicular), endoneural, hemicircumferential neurolysis.

64. What are the various tests for ulnar nerve palsy?

Ans. Following are the tests in the order of importance:
  1. Froment's sign/Bunnel 'O' sign/newspaper sign: Paralysis of 1st dorsal and 2nd palmar inter-osseous muscle with Adductor pollicis paralysis. Patient flexes thumb as a trick/compensatory maneuver.
  2. Card test: To test palmar interossei (PAD= palmar-adduct; DAB=Dorsal-abduct)
  3. Wartenberg sign: EDM unopposed by 3rd palmar interossei.
  4. Jeanne's sign: Loss of key pinch (Adductor pollicis)
  5. Bouvier's maneuver: Passive block of MCP hyper-extension facilitates IP extension.
  6. Duchenne's sign: loss of MCP flexion
  7. Pitres-stut sign: Inability to cross fingers e.g. IF on MF tests P1D2.
  8. Pitres-stut sign 2: Inability to make cone with extended fingers
  9. Pitres-stut test: Inability to radial and ulnar deviate MF.
 10. Masse's sign: Wasting and loss of metacarpal arch.
 11. Pollock sign: Inability to flex DIP of RF and LF (paralysis of ulnar half of FDP)
 12. Look for wasting in 1st web space (Ist dorsal interosseous)

13. Impairment of precision grip.
14. Andre-Thomas sign.

65. **What are anomalous innervations for ulnar nerve?**
Ans.
1. Martin-Gruber anastomosis: In proximal forearm b/n Median/AIN and ulnar nerve – additional intrinsic innervations.
2. Riche-Cannieu: Motor ulnar branch with recurrent branch of median – complete/partial intrinsic innervations
3. Ulnar nerve always contains fibers from C8 and T1; additional C7 in 5-10 per cent → FCU
4. FDP innervation – all ulnar to all median
5. Dorso-ulnar surface of hand may be innervated by radial nerve.

66. **What are the boundaries of Guyon's canal?**
Ans. Wall formed proximally by pisiform and distally by hook of hamate
Floor by transverse carpal ligament, hamate and triquetrum bones
Roof by pisio-hamate ligament.

67. **Can you outline the formation of brachial plexus and its branches?**
Ans. *(Learn this by heart as it may be asked anywhere and at any instance)*

Formation:
From C5-C8 nerve roots just lateral to scalene muscles, if a component of C4 received then it is termed pre-fixed; if from T1 – post-fixed. Roots → Trunks (Upper C5,6; middle C7; lower C8,T1) → Divisions (anterior and posterior; behind clavicle) → Cords – named after their relation to axillary artery (lateral – anterior division of upper and middle trunk; medial – anterior lower; posterior – posterior divisions of all three trunks)

Branches from the roots

1. Dorsal scapular nerve (C5) supplies rhomboids and levator scapulae and runs down deep to levator scapulae.
2. Nerve to subclavius (C5,6) supplies subclavius.
3. Long thoracic nerve (C5,6,7) supplies serratus anterior

Branches of the upper trunk (lower and middle have no branches)

Suprascapular nerve (C5, 6) supplies supra-spinatus and infraspinatus

Branches of the Lateral cord *(L2M)* (anterior divisions of upper and middle trunk)

1. Lateral pectoral nerve (C6) supplies upper half of pectoralis major
2. Lateral head of median nerve (C6,7)
3. Musculocutaneous nerve (C5, 6, 7) supplies coraco-brachialis, biceps and brachialis and then becomes lateral cutaneous nerve of the forearm.

Branches of the medial cord *(M4U)* (anterior division of lower trunk)

1. Medial pectoral nerve (C7,8) supplies the sternocostal fibers of pectoralis major
2. Medial cutaneous nerve of the arm (T1)
3. Medial cutaneous nerve of the forearm (C8, T1)
4. Medial head of median nerve (C8 and T1) joins the lateral head and supplies most of the flexor muscles of the forearm, the three thenar muscles and two lumbricals.
5. Ulnar nerve (C7,8 and T1) supplies the ulnar forearm flexors and most of the intrinsic muscles of the hand.

Branches of the posterior cord *(ULNAR)*

1. Upper subscapular nerve (C6,7) supplies subscapularis
2. Lower subscapular nerve (C6,7) supplies subscapularis (lower part) and teres major
3. Nerve to latissimus dorsi (Thoraco dorsal nerve (C6,7,8))
4. Axillary nerve (C5) passes backwards through the quadrilateral space in contact with the neck of the humerus and supplies deltoid and teres minor.

5. Radial nerve (C5,6,7,8 and T1) supplies muscles of the extensor compartment of the arm and forearm leaves the axilla through the triangular space below teres major.

68. List the muscles innervated by radial, median and ulnar nerves?

Ans. **Radial**: Arm – triceps, anconeus, radial half of brachialis, brachioradialis, ECRL, ECRB

Forearm: Supinator, EDC, ECU, EDM, APL, EPB, EPL, EIP

Median: Forearm: PT, FDP (IF, MF), FDS (I-IV), FPL, PL, PQ

Hand: APB, FPB (partial), OP, Lumbricals (I, II)

Ulnar: Forearm: FDP (RF, LF), FCU

Hand: Palmaris brevis, flexor digiti minimi, ADM, Opponens digiti minimi, adductor pollicis, flexor pollicis brevis (partial), all interossei, lumbricals (III, IV).

69. What are the various tests for median nerve?

Ans. *Signs:* Pointing index, ape thumb deformity (adducted thumb) – Simian 'hand', thenar wasting, sensory deficit.

Tests:

1. Clasp test: Ask patient to clasp both hands – IF remains extended
2. Pen test
3. Loss of opposition
4. Kiloh-Nevin sign: Ask patient to form 'O' with IF and thumb using tips – patient will extend DIP of IF and IP of thumb making peacock's eye instead.

70. What is Hilton's law?

Ans. It states that a nerve passing near a joint supplied that joint.

{*It is difficult at all times to understand learn and remember tendon transfers, as a rule difficult aspects will not be asked and if you pass in concepts then you may be forgiven for not knowing all the details. What you are expected to know:*

Type of palsy (high/low, complete/incomplete, recovering/non-recovering)

Basic principles of decision making
Examination of motor and sensory innervation and deficits
You should know how to examine:
FDS and FDP separately, wrist flexors and extensors, demonstrate PL, movements of thumb and muscles especially intrinsic muscles and their nerve supply.
Additionally you should know thoroughly the origin, course and muscle innervation of three major nerves of UL
Extensor compartments, carpal tunnel and structures, guyon's canal may also be asked}

CASE II:
LEPROSY HAND

{Important but difficult case as regards the treatment part which is quite similar to peripheral nerve injuries and needs constant revision otherwise *'all is volatile'*. Identification may be easy and barring few specific examination and characteristic points there is nothing "special" in leprosy. Approach the case as for peripheral nerve injury that helps learning then grab the specific points for leprosy. *Personally I will advise you to follow the practice in your institute for a given set of nerve involvement and not get confused in exams – remember not only does the eyes see what mind knows but also the mind knows what eyes see!*

Read: 6-8 times (MS Orth and DNB candidates)}

Diagnosis

The patient is a 28-year-old male with claw hand deformity of left hand due to high ulnar nerve palsy as a residual of leprosy.

Findings: See Q1.

1. **Why do you call it Hansen's disease?**
Ans. History: Long duration → Paresthesia (ants crawling), patch (area) of numbness, glove and stocking anesthesia, paralysis, trophic ulcer, epistaxis, pedal edema. Take h/o residence in endemic area, family h/o, occupational h/o, any other disease etiology leading to thickened nerves.

Skin lesions: Macular, papular, nodules, infiltration ulcers, burns, scar.

Palpate: Decreased sweating, roughness, scaling,

Nerves: (Cutaneous, superficial, peripheral)
Test for anesthesia
Motor weakness
Cardinal signs of Hansen's: (three)

1. Anesthetic lesion
2. Nerve enlargement
3. Demonstration of *Mycobacterium leprae* in lesions

   Other examination:

a. Ear lobe infiltration: Thickening and nodularity of ear lobes
b. Superciliary madarosis (loss of lateral third eyebrows) late feature of LL
c. Gynaecomastia (testicular atrophy) late cases of LL
d. Pedal edema
e. Trophic changes and ulcer formation (metatarsal heads and lateral border of foot)
f. V and VII cranial nerves (only cranial nerves to be involved in leprosy)
g. Lymph nodes (lepromatous spectrum)
h. Hepatosplenomegaly (occasional in lepromatous)

2. What nerves are commonly involved and how do you examine them?

Ans. In the order of involvement: Ulnar (mixed, high, low), median at wrist (then high), common peroneal at fibular neck, facial, radial (superficial at wrist and distal forearm, deep at arm), posterior tibial (clawing of toes and loss of arch), greater auricular, supraorbital.

   i. Supraorbital and supratrochlear: Examine from front → run thumb/index finger from midline to lateral margin of forehead); 2 cm from midline
   ii. Greater auricular: Turn head to side → make sternocleido-mastoid prominent → look for thickened nerve crossing the muscle → run thumb/IF above below to feel as it slips under.

iii. Supraclavicular: 2 cm lateral to medial end of clavicle.
iv. Radial nerve in radial groove (arm) and over radius (lateral aspect of forearm)
v. Ulnar nerve in cubital tunnel: Right side with left hand and vice versa!
vi. Ulnar and radial cutaneous nerves over medial and lateral aspects of wrist
vii. Median nerve: Flex elbow and deep palpation of nerve at wrist between flexor tendons
viii. Common peroneal nerve: Patient sitting → flex knee and feel for nerve 2 cms below fibular head
ix. Posterior tibial nerve: 2 cms below and behind medial malleolus.
x. Sural cutaneous nerve: Between heel and lateral malleolus.

3. What is the old name of leprosy?

Ans. "Elephantiasis graecorum". In India it has been known as "Maharog" since ages.

4. What is meant by "lepra" and "kushtha"?

Ans. Lepra = bark/scaling; Kushtha = eating away.

5. Who discovered *M. leprae* and what was his hypothesis?

Ans. Gerhard Hendric Armauer Hansen (1873): first identified it as an infectious disease rather than 'older' hereditary concept.

6. Why are nerves involved and that too in a peculiar distribution?

Ans. *M. leprae* enters through naked axons in epidermis → perineural cells → endoneurium → localizes to Schwann cells → it enters nerves through endoneural blood vessels. Schwann cells are present only in peripheral nerves and the nerves are at relatively cooler regions of body hence particularly affected. (V and VII are the only cranial nerves to be involved in leprosy).

7. How does nerve damage occur?

Ans. Immune reaction to bacterial antigens is the primary cause of nerve damage. Also peripheral nerves (located in cooler areas) are common sites for trauma → localize infection; and also they pass through unyielding fibro-osseous tunnel hence getting further traumatized following thickening. In tuberculoid spectrum there is primary damage by caseating necrosis and in lepromatous spectrum there is slow ongoing fibrosis due to unchecked bacterial proliferation in Schwann cells with secondary ischemic damage to nerve (Vasculitis).

8. What is "Jogerson Lewandoz" law?

Ans. An infection when controlled by immunological mechanisms result in granuloma formation.

9. How will you test the integrity of dermal nerves?

Ans. Pilocarpine test

10. What is methylene blue test?

Ans. One of the tests to prove that hypopigmented patches are due to leprosy. Inject (36.5 ml) methylene blue → after around 6 days the patches become blue and retain stain due to lipid content of lepra cells.

11. What is the D/D of thickened nerves?

Ans. Hereditary sensory neuropathy, primary neuritic amyloidosis, Dejerine-Sotta's disease, Refsum's disease.

12. What is morphological index?

Ans. Percentage of viable bacilli after counting 200 bacilli.

13. What is bacteriological index?

Ans. Number of bacteria in an average microscopic field. Cochrane and Ridley scales commonly used to grade it from 1+ to 6+.

14. How do you classify leprosy?

Ans. WHO- Single lesion paucibacillary (SLPB), paucibacillary (PB), multibacillary (MB).

Ridley-Jopling (1962): True tuberculoid (TT), borderline tuberculoid (BT), borderline borderline (BB), borderline lepromatous (BL), lepromatous leprosy (LL); modified in 1971 to include subpolar tuberculoid (TTs) and subpolar lepromatous (LLs) types.

IAL (Indian Association of Leprologists) {1951 → 1953 → 1981}: Tuberculoid, borderline, lepromatous, indeterminate, pure neuritic. Previous maculoanesthetic type now clubbed with tuberculoid.

NLEP (clinical) classification: Non-lepromatous (N) → tuberculoid, maculoanesthetic, pure neuritic; lepromatous (L); (N/L) → borderline and indeterminate.

Madrid (more useful):

Types – lepromatous (macular, diffuse, infiltrative, major tuberculoid and pure neuritic), tuberculoid (macular, minor tuberculoid, major tuberculoid, pure neuritic)

Groups – dimorphous, indeterminate (divided into macular and neuritic)

15. What are lepra reactions?

Ans. Allergic inflammatory process which is not a part of infective process either in its spread or resolution although it may be associated one or more of these processes. The disease itself is chronic and active for a long time with bouts of exacerbating and sudden attacks of disease. They describe only an episode in major disease. These reactions are more common in lepromatous than borderline or tuberculoid spectrum.

16. What are the types of lepra reaction?

Ans. *Type I:* Observed around neural elements and skin lesions of borderline and tuberuloid types. They are likely due to bacterial antigens which are killed by therapy and get exposed to Cell-Mediated immunity. The reaction is

typically akin to lepromin reaction with presence of epithelioid cells as the hallmark. It may be upgrading (reversal) or downgrading types. Upgrading (reversal) reaction is a BT to TT shift and downgrading one is a shift to lepromatous spectrum with new lesions having their characteristics.

Type II: (*Aka* – erythema nodosum leprosum, Roseolar leprosy, Lepra fever) seen in lepromatous spectrum with/without treatment. This reaction resembles "arthus phenomenon" – Ag-Ab complex formed at specific sites. Reaction is particularly located around medium sized vessels.

	Type 1	*Type 2*
Spectrum	BT, BB, BL	BL,LL
Lesion	Existing lesion develop erythema and edema	New modules arise in crops
Nerve damage	Frequent and severe	Not so
Systemic signs	Not common	Fever, malaise, arthralgia, lymphadenitis
Other organs	Iritis, orchitis, glomerulonephritis do not develop.	Common
Course	Relapse infrequent	Common
AFB	Not seen	Broken bacteria
Investigations	Normal routine	Urine – albuminuria
Pathogenesis	Type-4 Ag-Ab reaction	Type 3 Ag-Ab reaction
Histopathology	Disorganized granuloma	Vasculitis

Type 3 (Lucio phenomenon, Erythema necroticans): seen in Mexican origin only, vascular lesion with diffuse infiltration, does not respond to Thalidomide. Large number of organisms seen.

17. What are the types of ENL?

Ans. Two types: Intermittent (divided into mild and severe forms) and continuous (no reaction free period).

18. What are the bony changes in leprosy?

Ans. *Specific:* Osteitis, periosteitis, bone cysts, bone resorption (longitudinal/concentric/combined).

Non-specific: Erosions, osteomyelitis, osteopenia, septic arthritis.

Minor changes: Honey combing, pseudocyst, enlarged nutrient foramina, stippling.

19. What is reaction hand?

Ans. Subcutaneous nodules develop over dorsum of hand in type 2 reaction. Infiltrating edema with pain and functional incapacity is often present. If these features are associated with arthritis of IP joint → reaction hand.

20. What is frozen hand?

Ans. Subcutaneous nodules heal by leaving scar (involves palmer skin and aponeurosis) → Contracture produced by fibrosis pulls up finger at MCP joint → pull in various directions produces multi-forme deformities in fixed position known as frozen hand.

21. How do you define low ulnar nerve palsy?

Ans. Anatomically – distal to olecranon fossa. Clinically – involvement of intrinsic hand muscles.

22. What is high ulnar nerve palsy?

Ans. Anatomically – proximal to ulnar fossa. Clinically; involvement of ulnar half of FDP and Flexor Carpi Ulnaris.

23. What do you understand by low and high median nerve palsy?

Ans. Low median nerve palsy typically is defined by involvement around wrist (carpal tunnel). Motor loss to OP, APB, FPB, 1st and 2nd lumbrical with variable loss of palmar cutaneous sensation. High median nerve palsy in addition has motor loss to FDS, radial half of FDP, and thumblong flexor (FPL), weakness of pronation (pronator quadratus).

24. What is deformity, disability, impairment?

Ans. *Deformity:* Alteration in form/shape/appearance of body part which is visible.

Disability: Deterioration in one's ability (capacity) which is felt by the patient. Also defined as inability (or difficulty) to carry out certain tasks.

Impairment: Anatomic, physiological and/or psychological abnormality or loss resulting from disease/disorder that may be temporary or permanent.

25. What are the types of deformity in leprosy?

Ans. *Specific (LL/BL):* Intrinsic plus finger, twisted finger, banana finger, reaction hand, frozen hand.

Paralytic (BB/Neuritic): Claw hand, wrist drop, claw toes, foot drop.

Feet: Trophic ulcer, scars and contractures, short digits.

26. What is the chemotherapy for leprosy?

Ans. MDT (Multi Drug Therapy)

Multi-bacillary (MB): Regimen for adults: Rifampicin 600 mg (per month), Dapsone 100 mg daily, Clofazimine 300 mg per month and 50 mg daily. {Children (10-14yr) – half dose}.

Paucibacillary (PB): Rifampicin 600 mg monthly, Dapsone 100 mg daily. Single lesion (SLPB): Rifampicin 600 mg, Ofloxacin 400 mg, Minocycline 100 mg (ROM regime).

27. How long will you continue treatment?

Ans. MB: Life long monotherapy (dapsone); MDT till skin smears negative; FDT (fixed dose treatment) 24 pulses (in 36 months); **FDT 12 pulses in 18 months (1995)**.

PB: 6 pulses of PB – MDT.

SLPB: ROM therapy.

Treatment of reactions: **Steroids (types I and II)**; thalidomide (I); colchicines (II), **chloroquine (I and II)**, cyclosporine till subsidence of lesions and new lesion do not appear.

28. How do you diagnose and treat neuritis?

Ans. Suspect neuritis in: tenderness over nerve trunk with thickening

Swelling/redness/increased temperature

Some functional loss in distribution.

Conservative (medical decompression): Anti-inflammatory drugs; prednisolone (60 mg/kg reduced by 10 mg every week till three weeks → good response treat with 30 mg/kg till 6 months); heat; splint → if no improvement in 3 weeks then operate.

Operative: Indications:

1. Recent onset incomplete paralysis not responding to conservative treatment
2. Sudden onset complete paralysis attributed to neuritis
3. Continued pain even in presence of paralysis
4. Deterioration/progression of disease on conservative treatment
5. Symptomatic nerve abscess

Procedures:

1. External decompression by removing constricting ligaments and /or arching tendon fibers, medial epicondylectomy for ulnar nerve
2. Reduction of angulation stress: E.g. anterior transposition of nerve
3. Decompression:
    a. Epineurectomy
    b. Hemicircumferential neurolysis (epineurium dissected only from hemicircumference of nerve to preserve blood supply)
    c. Interfascicular neurolysis

Splinting and exercises (muscle strengthening – physiotherapy; functional re-education – occupational therapy).

29. What is claw hand?

Ans. Claw hand is deformity of hand due to functional malposition with clawing of fingers (in position of extension or hyperextension at MCP joint and flexion at

IP joints) and loss of cascade (normal disposition of fingers in relaxed hand). It can be due to low/high ulnar nerve paralysis (partial claw hand) or due to combined ulnar and median nerve paralysis (total or complete claw hand). Other causes are soft tissue contractures, hand compartment syndrome and sequel, forearm compartment syndrome and VIC.

30. What is reversal of grasp or how does clawing functionally affect patient?

Ans. Major hand functions include: Grasp, pinch, hook, and grip. To achieve all or any of these functions hand must first achieve lumbrical position (MCP flexion and IP extension) followed by gradual MCP → PIP → DIP → flexion occurring in coordination. In intrinsic paralysis (intrinsic minus hand) extension at MCP occurs due dominance and DIP and PIP sequences occur before MCP flexion pushing the object away before grasping.

31. How will you assess this claw hand for treatment?

Ans. *Claw deformity* → forearm vertical on table with wrist neutral → ask patient to actively extend fingers → assess severity of deformity.

Assisted angle: Assesses extensor lag in IP joint. Patient actively extends finger with examiner stabilizing MCP joint (examiner basically replenishes lumbrical function) → if assisted angle is present then flex MCP joint until maximum active extension of IP joint is possible. If assisted angle >30º then passive (static) procedures are contraindicated.

Contracture angle: Measures residual fixed flexion at IP joint. (Examiner passively extends the finger to fullest and measures the residual angle). Joint stiffness/skin/capsule contracture causing contracture angle <30° is correctable by physiotherapy.

FDS power: If FDS is involved then routine procedures are contraindicated.

FDP power: if FDP to RF or LF is < grade 4 MRC then use of FDS of these fingers is contraindicated.

Volkmann's sign: If positive then stretching by physiotherapy is required before surgery.

Hypermobility of joints if present then use weaker muscles for transfer to prevent later development of swan neck deformity.

32. What are the prerequisites for surgery in leprosy hand?

Ans. Determined by various factors:
- Good clinical response to anti-leprosy treatment
- No attacks of reaction or neuritis during previous 6 months.
- No tenderness over nerve
- Deformity for at least 1 year (no possibility of spontaneous recovery)
- No joint contracture/joint damage
- < 10° hyperextension at PIP joint and <15° ulnar deviation of fingers {ideally should be absent}
- Isolated contraction of muscle to be transferred must be practiced
- Compensatory abnormal movement pattern must be unlearnt and eliminated
- Supple hand
  Most importantly explain to the patient that only motor function will be restored and not sensory function.

33. How will you treat claw hand?

Ans. Primary requirement in claw hand is to provide flexion at MCP joint.

Secondary is to achieve flexion at IP joint and coordination and power.

I will get an X-ray done to look for condition of bones and joints.

Passive (Static) treatment methods to provide flexion at MCP joint (Achieve only first goal and also do not address adduction of fingers)
- Zancolli's anterior capsulorrhaphy of MCP joint.

- Extensor diversion graft (Srinivasan)
- Palande's technique (capsulorrhaphy and flexor pulley advancement)
- ECRL tenodesis
  Tendon transfer (dynamic):
  Even if RF and LF are involved, transfer of tendon to all four tendons must be done to provide coordinated and powerful movements:

1. *Sublimis transfer (Modified Stiles-Bunnel FF_4T phasic transfer)* – FDS of RF → 4 tail/slips → IF (ulnar side) and rest of fingers (radial side). Attach to dorsal lateral extensor expansion. Specific attachment provides in addition adduction of fingers. Tendon should pass anterior to MCP joint (Bunnel originally used all 4 FDS slips that led to intrinsic plus deformity).

2. *Zancolli Lasso:* Transfer of FDS of RF/MF to all fingers. Here a lasso is created around A2 pulley system (modified Lasso) to provide dynamic MCP flexion (remember unassisted angle should be <30º).

3. Extensor to flexor 4 tailed (EF_4T non-phasic, Brand): ECRL tendon with fascia lata or palmaris or plantaris extension attached as mentioned. Brand originally described it with ECRB tendon and later modified with ECRB/ECRL.

4. Palmaris longus many tailed graft – if fingers are hypermobile. (This weaker muscle will not cause swan neck deformity).

5. Extensor indices transfer.

34. How do you ascertain adequate tension?

Ans. Karat's method: Maintaining range of excursion between 4-6 cms.

Other method is to clinically pull one tendon slip that should just start excursion in nearby slip.

35. How will you address thumb function?

Ans. Adduction of thumb lost in ulnar nerve (Adductor pollicis, FPB - partial) and major thenar muscle functions (APB, FPB, OP) lost in median nerve palsy (Claw thumb) – so

treatment required mostly in total claw hand. Aim is to provide pronation (Total claw hand) and adduction (ulnar). Most commonly FDS to ring finger rerouted around pisiform bone (Riordan's opponensplasty) → divided into two slips → one attached to dorsum of MC (adduction) and other to dorsum of terminal IP joint (pronation). Other transfers include lateralization of EPL tendon at wrist, APL rerouting, ECU rerouting, FPL transfer, intermetacarpal bone block.

36. What ancillary surgeries may be required?

Ans. Web space contractures → Z-Plasty (with or without skin graft or Groin)

Arthrodesis of MCP and IP joint (bone destruction and non-salvageable joints)

Surgery for Boutonniere's, Swan neck and mallet finger deformities may be required.

37. What are the prerequisites for tendon transfers in foot?

Ans. The following must be looked for:
- No planter ulcer or septic focus.
- No fixed equinovarus deformity or tarsal disorganization
- Condition of muscles: Powerful tibialis posterior (Grade V or at least IV+)
- No contracture of tendo Achilles
- At least 20° ankle dorsiflexion
- Conditioning of tibialis posterior muscle for voluntary contraction (training).

38. How will you manage Hansen's foot drop?

Ans. Main aim is stability to have reasonable gait and stance. I will get X-ray done to see status of bones.
- Early foot drop: I will do passive mobilization, splinting, physiotherapy to strengthen muscles till one year as spontaneous recovery is possible till that time.
- Established foot drop: Tibialis posterior transfer (non-phasic) → split into two → attach each into EDL/EHL. Two routes:
   1. Circumtibial route is easier but pull is oblique.

2. Trans-interosseous membrane: Direct pull but more chances of adhesion.

Other method is to insert it into ligaments of foot or middle cuneiform (not preferred)

Incomplete lesions with active peronei → do a transfer of both peronei and tibialis posterior otherwise tendency to eversion is increased.

39. How will you manage Claw-Toes?

Ans. Claw-toes arise due to posterior tibial nerve palsy (Three stages – 1. Hyperextension of MTP joint: Toe tip touches ground: Toe-tip ulcer; 2. Increased hyperextension: Toe tip off ground: Increased IP joint flexion: Toe dorsum ulcer; 3. Increased MTP hyperextension: Dorsal dislocation of toe; Toe Ball ulcer.)

1. If toes are supple: Flexor to extensor transfer: FDL transferred to dorsum of toe and extensor tendon.
2. Stiff toes with contracture: Shortening of toes (excision of middle phalanx) and inactivation of long flexor tendons with arthrodesis.

40. How will you manage foot ulcers?

Ans. Three types:

1. Lepromatous: MDT for leprosy and nitrofurazone ointment.
2. Stasis ulcer (dorsum of foot and medial and lower third of leg): Local hygiene, antisceptic cream, dressing, elastic compression bandage and elevation, POP cast and skin grafting.
3. Trophic ulcer (plantar ulcer caused by prominent metatarsal heads, sensory deficit, weight bearing, increased plantar fascia tension, loss of protective function of sesamoids in FHB):
   a. *Acute (swollen hot foot with abscess):* Drain-age, wash and irrigate, regular dressing, elevate limb, antibiotics.
   b. *Chronic:* Regular dressing with antisceptics (or MSGP (Magnesium sulfate, glycerine, pro-flavine) solution), POP cast, rest and immo-bilization (contraindicated for

secondary complications – infection), Remove slough, clean and dress to enhance granulation, split thickness skin grafting, amputation.

41. What is "Hot foot syndrome" and how will you manage it?

Ans. Acute neuropathic disintegration of foot → results from neurological deficit. Foot is hot and swollen with collapsed suspension system.

42. What other deformities can be found in foot?

Ans. Cocked up toe due to unopposed extensor action (treat by extensor tenotomy); Rosette toes (bunching of toes) treated by corrective bandaging, neuropathic foot can be managed by triple arthrodesis but chances of failure are high.

43. What vaccines are available for Hansen's dis-ease?

Ans. Live *M. leprae*, killed *M. Leprae*.
Killed *M. Leprae* with adjuvant
Chemically modified *M. leprae*
BCG + *M. Vaccae*
Delipified *M. leprae*
M. Omega (ω vaccine) {ICMR Delhi}
ICRC bacillus.

44. How do you make smears?

Ans. *Slit smears:* Incise 5×3 mm and scrape material from posterior auricular patch.
Snip smears: Small piece of skin removed and crushed under slide.

45. What is histoid leprosy and lazarine leprosy?

Ans. Histoid leprosy is seen in LL patients with or without relapse. It has variable nodules, does not undergo ENL, absence of globi and clears on treatment.
Lazarine leprosy (Lepra Manchada) are ulcerating lesions arising over trunk and extremity seen in BT leprosy (also seen in Lucio leprosy).

46. What is SFG index?

Ans. Solid, fragmented and granular index:
2-0-0 (all solid) {SFG index = 10}
0-0-2 (all fragmented) {SFG index = 0}

47. What are the characteristics of *M. leprae*?

Ans. It is an atypical (slow growing, oxidizes DOPA) partial (weakly) acid-fast bacillus that looks like a bundle of cigars "globi" under microscope.

48. What are the stains for *M. leprae*?

Ans. Ziehl-Neelsen and Fite-Faracco stains with the later one being better as the bacilli are acid-fast for only a particular duration of their lifetime and Fite-Faracco sort creates acid fastness in bacilli. (Hansen originally used osmic acid).

CASE III: DUPUYTREN'S DISEASE

Diagnosis

The patient is a 52-year-old male with dupuytren's contracture of both hands involving the ring and little fingers.

Findings

- Painless nodules/cords over palmar aspect
- Garrod nodes/knuckle pads over PIP joint dorsally
- Painless flexion contracture of fingers
- Thinning of subcutaneous fat
- Adhesion of skin to contracture
- Pitting/dimpling of skin
- Lesion's of plantar fascia (Ledderhose disease)
- Plastic induration of penis (Peyronie disease)

1. What is dupuytren's contracture?

Ans. It is a proliferative fibroplasia of palmar tissue predominantly involving the palmar fascia and palmo-digital extension. The proliferation is seen in the form of

nodules and cords that may soon after develop in secondary flexion deformity of fingers.

2. What are various risk factors for the development of this disease?

Ans. Male sex (M:F=10:1, occurs earlier in males), Scandinavian and celtic origin, diabetes mellitus, epilepsy, alcoholism, pulmonary TB, vascular insufficiency (?Free radicals, ?Platelet derived growth factor) and cigarette smoking and heredity (autosomal dominant with variable penetrance).

3. How do you classify dupuytren's disease?

Ans. There are three phases:
1. *Early:* Skin changes + loss of normal architecture + skin pits
2. *Intermediate:* Nodules and cords
3. *Late phase:* Above + contractures. This has four stages:
   i. Ring finger MCP joint contracture
   ii. Ring finger MCP + PIP joint contracture + Little finger MCP
   iii. Above + Little finger PIP joint and MCP joint of middle finger
   iv. Above + DIP joint hyperextension of ring or little fingers or both.

4. Which finger is most commonly involved?

Ans. Ring finger (followed in order of involvement by little, middle, index and lastly thumb).

5. What are dupuytren's nodules?

Ans. It is a firm soft-tissue mass originating in the superficial components of palmar and digital fascia which is fixed to both skin and deep fascia. They are usually well-defined and localized. In the palm they are located adjacent to distal palmar crease while in fingers the nodules are commonly found at PIP joint or at the base of fingers. They are often painless but may become symptomatic

when associated with stenosing tenosynovitis due to direct pressure on A1 pulley. Over time they get regressed to be replaced by cord.

The dorsal side equivalents are Garrod nodes (rare) or knuckle pads which are prevalent in bilateral disease and particularly the presence of former should suggest one to search other sites for similar affection (Ledderhose and Peyronie).

6. How does the nodule form?

Ans. The earliest change is the thickening of Grapow fibers (these connect the fascia to dermis). There is hence the development of thickening of skin associated with rippling and dimpling. The local proliferation continues to form a nodule or if skin retraction occurs then a pit. (*Skin pit caused by full thickness skin retraction is a reliable sign of early dupuytren's disease and is latter replaced by nodule or cord*).

7. What is the pathogenesis of cord?

Ans. Cords are often the future of nodules, however, they may arise de novo. Remember the cords or nodules are by themselves not de novo structures but often the fibroblastic proliferation in normal anatomical structures that become thickened by myofibroblastic activity and deposition of type III collagen. The cords involve the palmar, palmodigital and digital regions and the exact distribution is complicated necessitating sound anatomical knowledge of hand, however the important features are presented here.

For development of flexion deformity at MCP joint the pretendinous cord is most important.

The spiral cord is the one that extends through palm to digits and has four origins; the pre-tendinous band, the spiral band, the lateral digital sheet, and the Grayson ligament.

PIP joint contracture results from involvement of central cord, lateral cord, spiral cord, retrovascular cord.

DIP joint contracture emerges from contracture of retrovascular and lateral cord.

8. What is the differential diagnosis?

Ans.

1. Non-dupuytren's disease: Occurs in diverse ethnic groups (Dupuytren's disease occurs in whites), unilateral, usually single digit, often associated with trauma, can spontaneously improve (rarely needing surgery)

2. Epithelioid sarcoma

3. Occupational thickening of skin and hyperkeratosis

4. Localized PVNS, palmar ganglion, inclusion cyst are differentials for large dupuytren's nodule

5. Post-traumatic contracture.

9. How do you treat the patient?

Ans. General guidelines are as follows:

Observation is limited to a patient with static disease and minimal contracture or functional compromise.

Surgery is the keystone to treatment for majority. Typical indications are flexion deformity of >30° at MCP joint and flexion contracture of 15° at PIP joint in the presence of a well-developed cord. The following procedures are done with their individual merits and demerits:

1. *Percutaneous fasciotomy:* Preferred for palmar cords in older patients. Higher recurrence rate; limited dissection required but greater danger to nearby tendons.

2. *Fasciectomy:* Partial, regional, limited remains the most widely done procedure. Lower recurrence rates.

3. *Segmental aponeurectomy:* Through multiple small C-shaped incisions in palm or digits segments of diseased fascia are removed.

4. Total fasciectomy and digital Z-plasties: Higher complications

5. *Dermofasciectomy:* Simultaneous excision of skin and diseased tissue. Low recurrence rates but limited to recurrent disease or treatment failures or very severe disease due to extensive dissection needed.

6. *Salvage procedures:* Amputation (PIP flexion deformity >70°, recurrent disease with exuberant scar tissue), dorsal

wedge osteotomy of proximal phalanx, arthrodesis of PIP joint with partial resection of proximal phalanx, arthroplasty of PIP joint.

Surgical release of PIP joint contracture is needed for residual deformity >40°.

10. What are the complications of surgery?

Ans. Various complications are known, viz., neuro-vascular injury, hematoma, infection, stiffness, reflex sympathetic dystrophy, recurrence (true recurrence – disease at the operative site or disease extension – recurrence outside the primary surgical site), and inclusion cyst formation.

11. What can you offer to a patient unwilling for surgery?

Ans. Local agents have been tried with variable efficiency:
- Calcium channel blockers for early disease
- Collegenase for advanced disease
- Trypsin and hyaluronidase (enzymatic fascio-tomy)
- Steroid injection and local γ-interferon injections.

12. What are the poor prognostic factors?

Ans. Male sex, family history, ulnar side lesions, alcoholism and epileptic patients, bilateral disease, etc are associated with poor prognostic factors.

CASE IV: FLEXOR TENDON INJURY

Diagnosis

Forty two-year-old male with 15 days old complete cut injury to FDS and FDP of ring and middle fingers of right hand in zone II.

Findings

- Scar mark over palmar aspect of hand healed with primary/secondary intention
- Loss of finger flexion cascade

- Palpable gap at the injury site
- Palpable nodule (of retracted tendon) proximal to injury site (commonly at A2 pulley or at FDS chiasm or in palm)
- Inability to actively flex the finger at PIP and DIP *(examine both for all injured fingers)* of RF and MF
- Loss of sensation distal to the injury site (cut injury to digital nerve(s).   Loss of sweat and two point discrimination

1. Why do you say it is a cut of both FDS and FDP?

Ans: *(Also see section 7a. examination of hand, motor power and muscle/tendon function).* There is complete absence of finger flexion at both PIP and DIP joints on testing active finger flexion.

2. How do you interpret the finger flexion cascade to localize tendon injury?

Ans: (*Observing flexion cascade is better than probing the wounds in many circumstances*). Allowing the wrist to drop free into extension there is a passive tenodesis effect of long flexors that bring all four fingers into a smooth flexion cascade ('arcade') with incremental flexion from IF to LF. Also there is flexion at IP joint of thumb. Any break in this smooth transition indicates pathology (the interpretation is true for Zone 1-3 injuries and some zone 4 injuries).

A finger held with slight flexion at both IP joints but with a break in cascade → FDS injury

A finger is straighter than others but there is slight flexion at PIP joint → FDP injury *(if there is no history of cut injury then suspect FDP avulsion; "Jersey finger" – especially for RF).*

A completely straight finger → injury to both FDP and FDS.

3. What else would you like to know from patient's history of injury that has a bearing on management?

Ans: I would specifically enquire if the finger was in flexion or extension at the time of injury.

4. What is the implication?

Ans: Injury inflicted at finger flexion suggests that the level of wound; cut FDS and cut FDP will be different and is a favorable situation as 'bunching' at repair is less likely with minimal chances of cross union and adhesions or mechanical block later during rehabilitation training.

An injury in extension would although make it easier to find the cut structure through smaller incision, however there are higher risk of healing process and tendons becoming one scar unit.

5. What in examination would prompt you to look for possible other injuries?

Ans: Cut injury of both tendons in zone 2 will raise alarm for associated digital neurovascular injury. Cut injury at zone 3 often implicates injury to digital nerves and superficial arch injury. Cut flexor tendons except Palmaris longus at Zone 4 should be explored for median nerve injury. Similarly cut injury to FCU often indicates ulnar neurovascular injury.

6. What are the zones of hand?

Ans: Verdan described 5 zones for flexor region of hand as shown in table below:

Zone	Limits (with suggested modification)
1	Insertion of FDP tendon at DP to insertion of FDS at MP
2	(No Man's land) Insertion of FDS at MP to A1 pulley – corresponds to distal palmar crease
3	A1 pulley to distal limit of transverse carpal ligament (only the central limited part of tendons supplied by mesotenon and attached to lumbricals are proposed to be included in this zone)
4	(Enemy territory) Underneath the transverse carpal ligament – between proximal and distal limits (synovial coverage of flexor tendons extends up to 4 cm proximal to proximal limit and some authors include this also in zone 4)

Contd...

Zone	Limits (with suggested modification)
5	Muscles tendons and other structures proximal to proximal limit of carpal ligament. Extensive laceration of muscles in this region is termed "Spaghetti wrist/full house injuries"

(*Remember only the classical ones unless you aspire to become a hand surgeon or encounter a hand surgeon in exams!*)

7. Why is zone 2 called 'no man's land'?

Ans: Bunnell (1918) introduced the term "no man's land" for zone 2 following dismal results of primary repair and admonished surgeons to primarily remove the tendon and graft later. Problems primarily arise due to limited space fibro-osseous tunnel containing two tendons and multiple pulleys and minimal areolar tissue to allow gliding despite best of repairs. Since then with advances in suture material and techniques along with better understanding the term should be upgraded to "some-man's land".

8. What will you do for this patient?

Ans: (Considering the wound is favorable – healed without infection/necrosis). I will ensure suppleness of joints and absence of reflex sympathetic dystrophy. I will explain the need of exploration and tendon repair with arrangements for tendon grafting and repair of digital nerve to the patient. Prognosis and need of all these procedures will be explained to the patient.

(*Any injury more than three weeks old is a strong candidate for tendon grafting as the suture retaining strength of tendon decreases and myostatic contractures develop precluding apposition of tendons without excessive IP flexion*)

9. What care will you take during repair?

Ans: Proper exposure using mid-lateral incisions or Bruner incisions or both. Minimal tissue handling, avoid

devascularization of tendons, avoid bunching (stuffing), strong enough repair for early mobilization, maintaining or reconstructing pulley system.

I will expose the injury site through the transverse wound and a mid-lateral incision (as nerve is also involved). I will milk the tendons through the sheath using gentle pressure on palm and flexing the MP joint; else I will expose the retracted tendons in palm and pass a catheter through the digital sheath with tendons tied to the same and deliver through wound.

10. What are Bruner incisions?

Ans: Extensile zigzag incisions suitable for pure tendon injuries that keep away the scar site from repair as much as possible and do not produce function limiting wound contracture.

11. What should be the characteristics of suture material for repairing tendon?

Ans: The suture should have high tensile strength; have easy knotability; having minimal tissue response; not be extensile, for preventing gaping; be absorbable late after healing of tendon and have ease of use.

12. What suture technique will you use for repair?

Ans: I will use locked cruciate core suture technique (or otherwise modified Kessler) using 4-0 ethibond along with Lin-locking epitendinous suture (or simple locked epitendinous suture) using 6-0 prolene to 'tidy up' the repair.

13. What tendons will you repair?

Ans: I will repair the FDP and one slip of FDS using four-strand technique. However if FDS is cut proximal to Camper's Chiasm then I will repair both using the core suture and epitenon repair technique. If the cut in both tendons are at the same level then only repair the FDP.

14. Why will you use the four strand technique?

Ans: It is agreed upon that more the number of suture filaments cross the repair site the more is strength of initial repair (time-zero). However, the strength of repair is weakest at fifth-day and may drop anywhere from half to one fifth of time-zero strength. Now it is believed that some 14.7N strength is required to flex finger against moderate resistance so that 3-5 times this measure (around 45N) is required for the time-zero suture techniques. Most of the 4-strand suture techniques easily surpass this 45N limit (especially the Becker and locked cruciate techniques – 60N) also some strength is provided by epitendinous technique (10-50%). Two strand techniques are hence weak and 6 and 8 strand techniques are too bulky possibly.

15. What other suture techniques you know of?

Ans: The following are various suture techniques:
- Two strand techniques: Bunnell (condemned), Mason-Allen, modified Kessler
- Four strand techniques: Strickland, Lee, Robertson, Becker, modified Becker and Locked cruciate (McLarney)
- Six-strand techniques: Savage, Sandow, Lim
- Eight-strand technique: Silva

"Strands" mean the number of suture filaments actually crossing the repair site.

16. How will you protect tendon surfaces and repair?

Ans: The following should be practiced: Using a strong nonabsorbable suture, employ at-least two independent sutures, using atraumatic technique, minimize exposed knots and sutures, minimize exposed raw surfaces of tendon, no strangulation of blood supply to tendon ends.

17. What if repair is not possible?

Ans: There is a high chance that the repair is not possible (difficult after two weeks) and then I will arrange for

tendon graft using Palmaris longus tendon in single stage if the peritendinous tissue is healthy (not scarred and provide gliding) else I will use a two-stage grafting technique (less likely).

18. What is two-stage tendon grafting and rationale?

Ans: *(Ensure supple skin, sensate digit, adequate vascularity and full passive ROM of IP joints)*. If the tendon bed and peritendinous tissue are badly disrupted so that the gliding sheath for tendon is not available and /or pulley system is damaged, I will place a silicone sheath in place (as spacer) so that pseudo-sheath develops around it which will act as tendon sheath for the future graft preserving or reconstructing A2 and A4 pulley. Stage II performed at least 3 months after; requires extrasynovial grafts from palmaris longus or plantaris or toe extensor passed through the formed tunnel using rail road technique.

19. How will you manage injury to pulley?

Ans: Out of eight pulleys (5-annular and 3-cruciate) A2 and A4 are the most important *(oblique pulley for thumb)* to prevent bowstringing of flexor tendon (A2 pulley) and reducing the angle of attack (A4 pulley) for flexor tendon to provide smooth flexion. Primary repair is possible only if care has been taken to deliver the tendons through L-shaped incision across the pulley. Else a damaged pulley will be reconstructed using extensor retinaculum sheath (distal half to third) or Palmaris longus tendon graft.

(*It is easier to remember the important pulley locations A2 is located in proximal half of proximal phalanx and A4 is located in middle third of middle phalanx*)

20. How will you manage tendon injury in zone 1?

Ans: Avulsion injury of FDP – 'Jersey finger' is classified into three types (Leddy and Packer):

- Type I – Avulsion injury with proximal tendon stump retracted into palm. Urgent repair required as tendon degeneration (both nutrient vinculae ruptured) and

myostatic contractures are highly likely. Delayed presentations (>21 days) require DIP fusion, FDP reconstruction around FDS (not through chiasm), no treatment or excision of FDP.

- Type II – Tendon retracts to FDS decussation at PIP joint (commonest). Short vinculum only is ruptured. Repair to bone.
- Type III – Larger piece of bone gets caught at the level of A4 pulley. ORIF using pull-out suture or suture anchors.

Cut injury of FDP is treated depending on the length of distal stump left. If more than 0.75 cm of distal stump remains – repair the tendon under A4 pulley (modified Kessler). For small stump either advance the tendon and attach to volar plate (*the volar plate moves with distal phalanx*) or bone. If Quadriga effect is expected then one may consider conversion to 'superficialis finger' or suture the proximal tendon to distal stump and bring out the suture through fingertip as pull out sutures.

21. What is 'superficialis finger'?

Ans: For small distal stump in cut FDP tendon in zone 1 the proximal stump is sutured to middle phalanx and the proximal end of distal stump is sutured to the neck of middle phalanx to prevent hyperextension deformity at DIP. The method is also used for non-reconstructible pulley in zone 2 with bowstringing of FDP.

22. What will you do for zone 4 injury?

Ans: As the space is limited so repairs are limited to terminal digital flexors. I will repair FPL and Index profundus independently while profundi to middle, ring and little fingers are combined anatomically into single unit. Superficialis tendons to repair should be so chosen that they will not be in contact with any other tendon repair. I will keep the carpal ligament open.

23. How will you manage the patient in postoperative period?

Ans: I will manage the patient with Strickland active motion protocol (See table below) designed for four-strand repair with epitendinous suture (Indiana protocol). This incorporates the early active motion exercises with tenodesis motion in a Kleinert-type hinged splint.

0-3 days	0-4 weeks	4 weeks	5 weeks	6 weeks	8 weeks
Dorsal blocking splint (DBS) with wrist in 200 and MCP in 500 flexion Tenodesis splint (TS) allowing 300 wrist extension and full wrist flexion, maintaining MCP in 500 flexion	Passive DIP extension with PIP and MCP flexion (DBS) (15 repetitions/2hrs) Passive PIP extension with DIP and MCP flexion (DBS) (15 repetitions/2hrs) Tenodesis exercises within hinged splint (TS) (15 repetitions/ 2hrs)	Dorsal blocking splint removed during exercise Tenodesis exercise continue (TS)	Active IP flexion with MCP extension followed by full digital extension	Blocking exercise if active tip to distal palmar crease > 3 cm	Progressive resistive exercises begin Unrestricted use of hand at 14 weeks

24. What is the rationale of this program?

Ans: From the original description of Kleinert using the elastic flexion pull against active extension various observations have modified the postoperative regime.

- Passive DIP extension with PIP and MCP flexion glides FDP away from FDS suture site
- Passive PIP extension with DIP and MCP flexion glides both tendons away from wound
- Combined MCP flexion and wrist extension produces the least tension on repaired site
- Tendon loses its tensile strength in first 2 weeks and gliding function by 10 days.
- Work of flexion (resistance to tendon gliding) at 1 week is least for repairs protected for first 3 days compared to those mobilized immediately (during edema) or at 7 days

- IP joint flexion is critical to preventing adhesion (it is difficult to determine in Kleinert regime if flexion is really occurring at IP joints or MP joint only)
- Wrist joint with greatest moment arm for flexor tendons produces greatest tendon excursion so tenodesis exercises have been added to active exercises.

25. What is Kleinert regime for postoperative rehabilitation of tendon repair?

Ans: The protocols for tendon repair have moved a full circle from early active mobilization in 1920s to immobilization to passive mobilization (Kleinert and Duran) to active mobilization (last two decades). Kleinert passive mobilization protocol involved the use of a protective splint with wrist and MP joints in flexion and IP joints in extension; whereby active extension was encouraged while flexion was passive using elastic rubber-band. This slowly progressed to progressive resistive exercises.

26. What is Quadriga effect?

Ans: Quadriga (Roman 4-horsed chariot – single driver holding reins to 4 horses) syndrome was first described by Verdan in 1960. The common muscle belly of FDP to four fingers (and analogous EDS – extensor quadriga) limits independent function of flexor tendons to individual finger. Also the muscle belly can contract effectively only if excursion of all the tendons is normal. Thus, if one of the tendon is fixed or its amplitude altered then proportional effect will be evident in all the other slips (reduced flexion) and often produces forearm pain. Pseudoquadriga is produced by fixed contractures of IP joints.

27. How much advancement is considered critical to produce Quadriga effect?

Ans: More than 1 cm advancement has been found to clinically produce quadriga effect.

28. What is lumbrical plus finger?

Ans: The term refers to extension (paradoxical) at IP joints (the action of lumbricals) on attempted flexion. This condition is seen in cases where the tendon graft is either too long or sutured in lax tension or has ruptured in attempted reconstruction for zone 2 or sometimes 3 injuries using tendon graft. The force in these cases is transmitted through the lumbrical muscle tendon unit instead of flexor tendon and hence paradoxical movement is produced. It is important to demonstrate full 'passive' flexion before making diagnosis of lumbrical plus finger.

(*Remember and do not confuse: The superficialis finger is a treatment method while lumbrical plus finger is a complication of reconstruction*)

29. Why is maintaining IP joint extension and MP flexion for immobilization so stressed upon?

Ans: During immobilization; MP joints should be maintained in more than 50° flexion as the collateral and accessory collateral ligaments are lengthiest in 50-70° flexion at MP joint. In case stiffness develops the ligaments will hence not contract and limit the flexion during rehabilitation, however immobilization in extension will produce contracted ligaments and capsule that are difficult to stretch later by physiotherapy. This differential length (19 mm in flexion and 14-17 mm in extension) is due to differential radii of metacarpal head and eccentric volar placement (Cam effect) which places PP farthest from metacarpal epicondyle (the origin of collateral ligaments) during flexion. The effect is not seen in IP joints and prevention of flexion contracture here is priority (hence extension). Such a cast with MP joint in flexion and IP in extension is called "Clam Digger cast".

30. How do you manage partial lacerations?

Ans: Tendon lacerations of up to 90% of their thickness are stronger than completely cut repaired tendons so a partial laceration must never be made complete. In general

lacerations up to 25% may be simply trimmed. Epitenon repair is done for lacerations involving 25 to 50% of tendon width. Larger lacerations are repaired as for a complete tear without disturbing the remaining intact tendon.

CASE V:
CARPAL TUNNEL SYNDROME
(TARDY MEDIAN PALSY)

(Simple case to diagnose and present and is good to score marks. Simple and straight questions and answers.

Read times 4-5 MS and DNB candidates)

Diagnosis

The patient is a 40-year-old female with non-traumatic incomplete right low median nerve palsy most likely due to carpal tunnel syndrome.

1. Why do you call this carpal tunnel syndrome?
Ans:

A: *Short history-*

1. Tingling and numbness sensation in the typical median nerve distribution in the radial three and a half digits (thumb, index, long, and radial side of ring) prominent in night ("nocturnal acroparesthesia"). Variable amount of deep throbbing pain present diffusely over hand climbing to forearm or arm may be described.

2. In late cases symptoms may progress to gritty or numb feeling in fingers, weakness of grip or pinch and diminished finger dexterity.

3. Tinel's and Phalen's test (Wrist hyperflexd for 60 secs; acceptable if Tinel's positive; sensitivity 0.59, specificity 0.93) are positive.

4. On inspection of the right hand there is thenar muscle wasting. The thumb cannot be opposed to the fingertips. Testing for APB revealed MRC power grade 3 compared

to the opposite normal side with reduced muscle bulk and tone present. {In advanced cases one may find thumb to be lying in the plane of the palm – a simian thumb (ape-thumb deformity). There may be atrophy of the pulp of the index and main fingers; dystrophic nail changes. There may be signs of ulceration or burns seen in the hand or fingers. However, testing for FPL revealed normal power. (Distinguishes with high median nerve palsy)}

2. What other differentials have you ruled out?

Ans: Also called CTS mimics:
- Median nerve contusion
- Cervical radiculopathy (double-crush syndrome)
- Thoracic outlet syndrome
- Pronator syndrome
- Idiopathic brachioplexitis (Parsonage-Turner syndrome/ neuralgic amyotrophy)
- Intracranial neoplasm
- Multiple sclerosis
- Cervical syringomyelia
- Pancoast tumor
- Peripheral nerve tumor (schwannoma, hamartoma, etc.)
- Lower trunk brachial plexopathy
- Ulnar neuropathy
- Radial neuropathy
- Generalized neuropathy (diabetes/mononeuritis multiplex)
- Churg-Strauss syndrome.

3. What else would you like to examine?

Ans: Examination of neck to exclude any proximal abnormality (like double crush syndrome). Examine for causes of proximal median nerve compression.
  1. Supracondylar process of humerus (also known as ligament of Struthers).
  2. Bicipital aponeurosis: Resist elbow flexion with forearm supinated.
  3. Between heads of pronator teres : Resist forearm pronation with elbow extended/negative Phalens test at wrist.

4. Proximal arch of FDS (Isolate long finger PIP joint flexion).

Simple test is to look for FPL weakness that identifies high median nerve palsy.

4. How do you perform Tinel's test and what is the significance?

Ans: The Tinel's sign is elicited by percussing in the midline from 2.5 cm proximal to 4 cm distal to the wrist crease. Presence of paresthesia along with a positive Phalen's test has sensitivity of 0.41 and specificity of 0.9.

5. Are there any other tests you can do to identify critical median nerve compression?

Ans: *Durkan's test:* Manual pressure over carpal tunnel for 30 secs – positive if paresthesia (electric test or tingling) elicited in the median nerve distribution. More sensitive than Phalen's test (acceptable if Tinel's positive).

Wrist extension test (Reverse Phalen's test; pray position): Active extension of wrist for 2 minutes (acceptable if Tinel's positive).

Tourniquet test (Gilliat and Wilson): Arm tourniquet inflated above systolic pressure for 60 secs – not significant.

Closed fist sign: Making tight fist for 60 secs – significant only if one more sign positive.

Hand elevation test: Hand elevated above head for 60 secs – not significant.

Ames test: To detect malingering. Make a fist and ask to press the fists together – identifies malingering if positive.

6. What are the other names for carpal tunnel syndrome?
Ans: Acroparesthesia, thenar neuritis, median neuropathy at wrist.

7. What are other clinical tests that can be performed in this case?
Ans: Hand diagram (Patient marks site of pain or altered sensation on outlined hand diagram), Hand volume stress

test (Hand volume measured by displacement, repeat after seven minutes stress test and ten minutes rest), static two point discrimination, moving two point discrimination, vibrometry, Semmes-Weinstein monofilaments.

8. Do you know of any invasive test?

Ans: Direct measurement of the carpal tunnel pressure by Wick method. Normal pressure inside carpal tunnel is 2.5 mm Hg. Complete intraneural flow stasis is seen at pressure above 80 mm Hg. 'Critical pressure' for microvessels to cause Obliteration and consequent ischemia is 40-50 mm. In patients with CTS the pressure has been found to be elevated to a range of 12-43 mm Hg.

9. How will you investigate this case?

Ans: I will get Electrodiagnostic studies (nerve conduction velocities and electromyography):

- A distal motor latency of more than 4.2 ms and a sensory latency of more than 3.5 ms are considered abnormal.
- There is increased median-to-ulnar latency difference of the fourth finger SAP (>0.5 ms).
- Electromyography may show signs of nerve damage, including increased insertional activity, positive sharp waves, and fibrillations at rest, decreased motor recruitment, and complex repetitive discharges in Adductor pollicis muscle.

10. What do you achieve with EDS?

Ans: Confirm the clinical suspicion of CTS. EDS-
   1. Localizes the lesion.
   2. Depicts involvement of motor, sensory fibers; defines physiologic basis (axon loss demyelination)
   3. Severity of lesion.
   4. Time course of lesion (evidence of reinnervation or ongoing axonal loss).

   In preoperative work-up EDS allows quantification of severity and type of lesion. Should the outcome of surgery is less than satisfactory it can be of some value in litigation.

It should not be used as a substitute for clinical examination as EDS is more sensitive and may come positive even in 'clinically silent' patients.

11. Is there any role of CT/Ultrasonography/MRI in carpal tunnel syndrome?

Ans: *Ultrasound* dynamic stress testing in neuromuscular ultrasound may demonstrate compression of median nerve between contracting thenar muscles ventrally and taut tendons dorsally. Mean diameter may decrease by 40% while in normal population there is a tendency of no change or median nerve enlargement by 17%. Cross-sectional area of <9.9 sqmm is taken as a cutoff for median nerve compression at pisiform level.

CT shows the bony structures clearly, but does not define the soft tissues accurately.

MRI shows high soft-tissue contrast may demonstrate space occupying lesion as a cause. Most importantly MRI is useful in postoperative failed cases (investigation of choice) to look for real canal widening, incomplete ligament resection, scarring or algodystrophy.

12. What is carpal tunnel and what are its boundaries?

Ans: A cylindrical, inelastic osseo-ligamentous space (open on both sides) bound dorsally by concave carpal arch (floor) and volarly by unyielding carpal ligament. The depth varies from 10-13 mm.

Carpal ligament (roof) attaches to the hook of the hamate, triquetrum, and pisiform medially; and the scaphoid, trapezium ridge, and fibroosseous flexor carpi radialis sheath laterally.

13. What are its contents?

Ans: The most ventral (palmar) structure in the carpal tunnel is the median nerve. Lying dorsal (deep) to the median nerve in the carpal tunnel are the nine flexor tendons (4FDS, 4FDP, 1FPL) to the fingers and thumb. *Total of 10 structures in tunnel.*

14. What is the commonest site for median nerve compression in the tunnel?

Ans: The compression has been localized to the thickest part of TCL which is somewhere 1 cm distal to the proximal border of ligament. This level has been identified to be in line with hook of hamate.

15. What are the risk factors for carpal tunnel syndrome?

Ans: Certain risks factors have been identified but are not proven *causative* factors:
- Increased age (30-60 yrs)
- Female gender (F:M=3)
- Obesity
- Cigarette smoking
- Vibrations associated with job tasks (rock drilling) – exertional CTS
- Repetitive movements of wrist and finger flexion
- Keyboarding (computer associated disease).

16. What are the causes of CTS?

Ans: Causes can be:
1. Idiopathic
2. Anatomic:
    a. Acute form (fracture, crushing hand injury, hemorrhage, burn, median artery thrombosis, infection, pregnancy)
    b. Distal radius malunion
    c. Carpal canal stenosis (deformity congenital or acquired)
    d. Anomalous structures (palmaris profundus, proximal origin of a Lumbrical, reversed palmaris longus, anomalous branch of radial artery)
    e. Space occupying lesions (ganglion, lipoma, fibroma, synovial sarcoma, neuroma, neurofibroma, hemangioma)
3. Occupational (exertional CTS)
4. Systemic
    a. Pregnancy
    b. Endocrinopathy (diabetes mellitus, thyroid disease, growth hormone)

c. *Congestive heart failure*

d. *Collagen and autoimmune diseases* (tenovaginitis, rheumatoid arthritis, scleroderma, gout, chondrocalcinosis, etc.)

e. *Amyloidosis*

f. *Polyneuropathy*

g. *Alcoholism*

h. *Myeloma*

i. *Consequential forms* (oral contraceptives, anticoagulants, lack of vitamin B6)

j. *Children's forms*

k. *Congenital diseases* (mucopolysaccharidosis, mucolipidosis).

17. Carpal tunnel has tendons and nerves as contents but can you think of any muscular cause of carpal tunnel syndrome?

Ans: Yes, aberrant muscles (lumbrical, reversed palmaris longus, palmaris profundus) can decrease the volume of carpal tunnel.

18. Are there any vascular causes of CTS?

Ans: Yes, anomalous branch of radial artery, hypertrophied or aberrant median artery and vein, hemangioma, A-V malformation in the tunnel.

19. How will you manage this case?

Ans: *The following is based on general consensus.* Mild to moderate cases that are not rapidly advancing and are not due to acute cause (see above) should be given a fair conservative trial. I will operate advanced and late cases only on an urgent basis and counsel for elective surgery to patients showing evident denervation in median nerve distribution and pronounced sensory loss particularly supported by electrodiagnostic studies.

20. What is non-surgical treatment for carpal tunnel syndrome?

Ans: Splinting, activity modification supplemented with oral medications are mainstay of conservative management of CTS.

Splinting: Especially at night and intermittently during day provide good relief in majority of cases, particularly those having positive Phalen's test.

Oral medications: Anti-inflammatory medication ± neurotropic vitamins (B6, methylcobalamin) ± steroids ± diuretics to reduce carpal tunnel pressure.

Local adjunctive measures like ultrasonics, laser therapy and iontophoresis have variable non-documented effects.

21. What is the role of corticosteroid injections?

Ans: Steroid injections provide rapid relief but there is no documented benefit after 3 months over oral steroids and over long term when compared to splinting and NSAIDs. There is no concrete information as to type, dose or location of injection. They may be of help in patients with symptoms less than 1-year, normal two-point discrimination, no thenar atrophy, less than 1-2 msec prolongation of sensory motor latencies and no denervation potentials (mild or moderate cases). Failure to improve with steroid injections is a poor prognostic factor and even surgical release may not be helpful in these cases.

22. What are the poor prognostic factors where conservative treatment often fails?

Ans: Age older than 50 years, duration of symptoms longer than 10 months, constant paresthesia, stenosing flexor tenosynovitis, and a positive Phalen test result in less than 30 seconds.

23. What are surgical approach to carpal tunnel syndrome?

Ans:
  1. Open carpal tunnel release
     a. Wrist-palm incision (Milford, Taleisnik, Phalen, etc.)
     b. Double incision technique by Wilson
     c. Palm only incision (Lee et al)
     d. Minimal open release (Bromley et al)
  2. Endoscopic technique (single portal technique of Agee and double incision technique of Chow)

24. **What are the complications of open release?**

Ans: Iatrogenic nerve injury (palmar or motor cutaneous branch), injury to thenar branch (commonly aberrant), scar tenderness, pillar pain (most common complication following release due to laceration of palmar cutaneous branch), weakened grip strength, injury to superficial palmar arch and recurrence.

25. **What are the contraindications for endoscopic release?**

Ans: Wrist stiffness, proliferative synovitis and space occupying lesions.

26. **What is the role of internal neurolysis during open release?**

Ans: Once popular this has been absolutely abandoned and is counterproductive.

27. **What are common causes of failed release and persistent symptoms or recurrent CTS?**

Ans: Incomplete ligament release, fibrosis/painful scar, tendon adhesions to nerve, missed double-crush syndrome, recurrent tenosynovitis. Others are reformation of the flexor retinaculum, scarring in the carpal tunnel, median or palmar cutaneous neuroma, palmar cutaneous nerve entrapment.

28. **How will you manage failed CTS?**

Ans: A very difficult problem even for experts. Reevaluate with EDS studies, MRI is a must. At revision surgery neurolysis of median nerve with fat or muscle transfer and vein wrapping are some of the methods described to improve results.

CASE VI:
MALUNITED DISTAL RADIUS FRACTURE

{This is a very common case to see in our subcontinent. Not a frequent exam case but in regions or places where large number of DNB students appear for exams, the case may be

kept. It is a fact that not many surgeons expertise in treatment of malunited distal radius fractures so the questions will be more directed to treatment of distal radius fractures rather.

Read times 6-8 times to grab the different concepts}

Diagnosis: This is a 56-year-old female with a malunited distal radius fracture. There is 1 cm of radial shortening and reduced grip strength.

Findings of a typical malunited Colles fracture.
1. Inspection
    a. Dinner fork deformity
    b. Shortening of forearm
    c. Mannus rectus or valgus
    d. Swelling at wrist
    e. Prominent ulnar head.
2. Palpation (above +)
    a. Distal radial swelling
    b. Irregular distal radius
    c. Tenderness at carpal region or dorsal wrist joint line (late cases due to degenerative changes) or wrist instability.
    d. Wrist widening.
3. Movements
    a. Reduced palmar flexion, ulnar deviation and combination of other movement loss depending on stiffness and wrist instability
    b. Reduced forearm movements (supination and pronation).
4. Reduced grip strength.
5. Shortening of radius.
6. Wrist widening (measurements).

   1. What type of malunion is this, would you like to be more specific?
Ans: This appears to be extraarticular malunion but I would need radiographs to confirm absence (or presence) of intraarticular extension of original fracture. Dorsal malunions are further classified as type I (correctable DISI) or type II (fixed DISI).

2. What other malunions you know of?

Ans: Apart from intraarticular and extraarticular malunions, can also be subgrouped as volar (Smith's) or dorsally (Colles) displaced malunions with shortening.

3. What would you like to do for this patient?

Ans: Firstly I would like to get radiographs to primarily evaluate the fracture. I will order standard AP, lateral and one clenched fist X-ray of wrist (to look for carpal instability).

4. What else can you expect to see on X-rays?

Ans: Reflex sympathetic dystrophy (algodystrophy) changes, associated ulnar styloid fracture.

5. What are the consequences of malunion that concern patient?

Ans: Intraarticular malunions produce irregular cartilage surface (late degenerative changes). Extraarticular malunions alter intracarpal, radiocarpal and DRUJ (distal radioulnar joint) mechanics. Grip strength is reduced. TFCC (**T**riangular **f**ibro-**c**artilage **c**omplex) strain may produce constant ulnar pain on activity. Altered loading patterns and premature osteoarthritis is quite possible.

6. How do you define malunion of distal radius on radiographs?

Ans: There is no constant definition. Agreed values are as follows: radiocarpal (RC) malunion – RC joint stepoff of > 1 mm. Dorsal malunion– 10 degrees of extension and more (greater than 20 degrees from normal). There is still more disagreement on malunion in coronal plane (< 10 degrees of radial inclination is considered significant), acceptable distal radius shortening (loss of 5 mm or more of radial height is considered significant), acceptable displacement of DRUJ surface and malunion of ulna. (*It's all dependent on onlooker, in view of an experienced surgeon a displacement is a displacement and malunion if united! – really*

a bookish definition, but I am sorry). Any malunion associated with radiocarpal subluxation is a significant malunion and needs corrective action.

7. What is the incidence of malunion?

Ans: Most common complication following distal radius fracture. Around 23-25% radiological malunion is seen. Clinically symptomatic malunion is very low somewhere around 5-7% following various treatment methods.

8. What are the values for various parameters of distal radius?

Ans: Radial inclination = 17 – 25 degrees (22 degrees av.)

Palmar/volar tilt/inclination = 2–20 degrees (11 degrees av.)

Radial length/height (from distal radius lunate fossa to radial styloid) = 9.9 – 17.3 mm (mean of 11 mm), <9 mm is significant.

Ulnar variance (from distal radius lunate fossa to ulnar articular surface) = –2.3 to ± 4.6 mm (average 0.9 mm), on a zero rotation view.

9. What is the most concerning aspect of distal radius malunion?

Ans: Dorsal tilt disables most of the mechanics around distal radius that concerns all patients, additionally for a manual labourer radial length is of additional concern as it weakens the grip strength. For intraarticular malunion development of wrist arthritis is a big concern (some 91% patients develop arthritis having step-off >2 mm while only 11% develop arthritis if the step-off is <2 mm), and should be managed with priority.

10. How will you manage dorsal tilt?

Ans: I will do an osteotomy (at the or as close to) from dorsal approach and do tricortical iliac bone grafting under fluoroscopy control to reproduce the distal radius anatomy (as compared to the normal side). Similarly for

other malunions wedge-shaped grafts in different lengths and stabilised using volar or dorsal locked plates (as the case may be) are used – nothing very special.

11. How will you manage intraarticular malunion?

Ans: I will get a CT scan of the distal radius malunion and plan surgery accordingly. In general the simple intraarticular malunions and malunions with dorsal subluxations are better dealt with dorsal approach while volar radiocarpal subluxation with malunion are better treated with volar approach. There is a trend to avoid incising the volar stouter capsule for good functional outcome and also familiarity of intraarticular reduction through dorsal approach (die-punch reduction) makes this approach preferable. DRUJ and TFCC needs specific management on a case to case basis.

12. What various named fractures do you know?

Ans: *Colles (pronounced as 'collis' – English literature) fracture (Pouteau fracture – French):* Dorsal displacement and tilt, volar displacement and tilt, impaction and supination. Fracture of ulnar styloid may or may not be a component.

Smith's fracture (Smith-Goyrand fracture): Reverse of Colles.

Barton's fracture: Intraarticular *shear* fracture of *dorsal* rim of radius with radiocarpal subluxation.

Reverse Barton's fracture (volar Barton fracture; Smith-Goyrand II): Fracture of *volar* rim of radius with radiocarpal subluxation (many people consider volar Barton as Barton's fracture – this is incorrect it is reverse Barton's fracture. Also Smith fracture 1 is extraarticular fracture with volar displacement and Smith fracture 2 is intraarticular fracture with volar subluxation as described).

Chauffeur's fracture: Radial styloid fracture.

Hutchinson's fracture: Radial styloid fracture ± scapholunate disruption.

Moore's fracture: Distal radius fracture with ulnar dislocation and entrapment of ulnar styloid fracture under annular ligament.

Die-punch fracture: Fracture through lunate fossa (depressed fracture).

13. What is Colles fracture?

Ans: As typically described by Colles – extraarticular fracture occurring at the corticocancellous junction of distal radius some one and a half inch above (around 4 cm) the carpal surface, more so a transverse fracture throwing the carpus dorsally with dorsal displacement and angulation at the fracture site.

14. How do you classify distal radius fractures?

Ans: There are a lot of classifications but the preferred are Melone's, universal and AO (remember any one), Fernandez and Frykman's are of historical significance only.

Melone's classification: Based on distal radius '4 parts' divided into (a) radial styloid, (b and c) dorsal medial and dorsal lateral parts (of lunate fossa) and (d) radial shaft.

- **Type I:** Stable, undisplaced and minimally comminuted (Colles equivalent)
- **Type II:** Comminuted, stable displacement of medial complex.
  - Posterior displacement – die – punch #: moderate to severe displacement;
  - Displaced # involving radioscaphoid joint – involves more than simple radial styloid
- **Type IIb (irreducible)** – 'double die-punch' # (irreducible injury)
  - Dorsal medial component fragmentation – persistent radiocarpal incongruity > 2 mm
  - Requires open reduction ± bone grafting for restoration of articular congruity
- **Type III:** Die-punch or lunate load fracture (depressed fracture)

– Involves additional # from shaft of radius that projects into flexor compartment (volar spike)
- **Type IV:** transverse split of articular surfaces, wide separation with rotational displacement
  - ORIF with plating ± bone grafting.

15. **What is Kapandji method of fixation of distal radius?**

Ans: Intrafocal 'double basket like' pinning typically described for extraarticular fractures. The k-wires pass through the fracture site (interfocal) and act to buttress the distal fragment in position resisting the displacing forces.

16. **What is the role of external fixator for fixation of distal radius?**

Ans: Typically indicated for open fractures for wound management. It has been used for distraction (ligamentotaxis) reduction of severely comminuted fractures to maintain alignment. The role of spanning external fixators has reduced now with the introduction of locked plates (internal fixator) and anatomic reduction. Non-spanning dynamic fixators have come into vogue for ease of application, minimally invasive surgery and no implant remaining in the body after removal at fracture union.

The Spine

{Spine cases are considered formidable and are sometimes simple and sometimes tricky. Its best to prepare thoroughly these cases as there is high probability of faltering in exams for limited exposure and differing concepts that are undergoing rapid and extensive change – I am not sure if concepts would change while this chapter is in writing!

Read 7-9 times for M.S and DNB candidates, I am afraid that topic could not be more simplified, as much of classical and recent developments have to be mingled}

EXAMINATION OF SPINE

HISTORY TAKING

- Pain (*Try to search for 'pain generators' and whether it is referred from other site, give particular attention to 'red flag signs'*):
  - *Onset (acute/insidious):* Acute in post-traumatic, jerk, pyogenic infection (discitis), tubercular infection; while insidious onset in mechanical low back pain, spondylitis, facet joints and vascular structures, metastasis (unless precipitated by trauma), primary bone tumors, hemangioma.
  - *Site:* Axial (localized pain) at all spinal levels often result from 'mechanical' (non-neurogenic) structures like musculotendinous structures, zygapophyseal joints, vertebrae and annulus fibrosus whilst radiating pain is due to the involvement of neurological structures that may be related to disc herniation, degenerative process, neuroforaminal stenosis or other space-occupying lesions, intrinsic diseases of cord/nerve root (Herpes zoster).
  - *Nature:* throbbing acute unbearable pain result from acute pyogenic infections, traumatic/pathological fracture (barring osteoporotic #), and severe constant pain may result from cauda equina syndrome, advanced tubercular infection, acute cervical/dorsal/lumbar strain.

Dull constant aching pain is often the result of mechanical problems originating as above.

– *Radiation:* The radiated (from other site) and radiating (to other site) pain are both noticed. Pain from lumbar or cervical regions often radiates as 'lightening sensation' in the limbs while dorsal segments radiate in 'Band-like' fashion. The general rule for radiating pain (from neurological structures) is simple and constant in that 'it is the spinal level of pathology and not the exact structure involved that determines the 'radiation pattern'. Axially radiated pain from various sites that require mention include aorta, carotid arteries, costovertebral and costotransverse articulations, pancreas, lung, pleura, gallbladder, stomach and proximal duodenum, diaphragm, kidneys, ureter, pelvic organs in female. Affections other than spine may also present with radiating pain and include lower trunk brachial plexopathies, neuropathies, heart and pericardium to upper limb and sacroiliac joint, sacrum (stress #), ilia and hip joints.

– *Diffuse/localized:* Localized pain often result from focal injury to mechanical structures whereas diffuse pain is often radiated.

– *Aggravating and relieving factors:* Rotation and extension was classically attributed to zygapophyseal joints. Forward flexion is painful in discogenic pain. Excursion as such is painful in inflammatory conditions of spine. Neuroforaminal stenosis causes neurogenic claudication that comes up after exertion and is relieved by forward flexion/sitting. Whereas sitting and getting up from seated position is often painful in discogenic pain, sleeping with knees and hips flexed often relieves pain.

• *Trauma:* Note the following – energy dissipated from impact, direct/indirect (like lifting heavy weight, seat belt injuries)

• *Deformity:* Onset, duration, sudden increase (collapse in pathological fractures), association with pain and fever (tuberculosis), stiffness (ankylosing spondylitis).

• Loss of power/weakness, balance.

Other history: Genitourinary complaints, gynecological complaints and all pertinent as above, Marfan's syndrome, Ankylosing spondylitis, vascular claudication.

Past medical history: Diabetes mellitus, tuberculosis, hypertension, hematological disorder, pulmonary disorder, treatment for osteoporosis, neurological disorder (like epilepsy, Parkinsonism), HIV status (pre-op workup).

Personal history: Smoking, alcohol, drug addiction.

EXAMINATION

Inspection

1. Attitude and deformity: 'military attitude', levels of shoulder, forward bending (scoliosis, kyphosis, ankylosing spondylitis), side bending (list).
2. From side:
    a. Kyphotic deformities (normal thoracic kyphosis is 21-33° measured by Cobb's method)
        i. Knuckle: due to single vertebra
        ii. Gibbus: 2-3 vertebra
        iii. Short angular/rounded kyphosis: small segment of spine involved from 4-8 vertebrae
        iv. Rounded kyphosis: ankylosing spondylitis, senile osteoporosis, Scheuermann's disease, osteomalacia.
        v. Loss or partial reduction of kyphosis: Early tuberculosis with 'boarding' due to spasm, flat back syndrome, iatrogenic (post-scoliosis correction).
    b. Lordosis of spine (average lumbar lordosis is 40°-60°, cervical lordosis = 20°-40°, thoracic kyphosis = 20°-45°):
        i. Increased lordosis:
            1. Females
            2. Obese
            3. Spondylolisthesis
            4. Fixed flexion deformities at hip
            5. Compensatory to increased thoracic kyphosis
            6. Constitutional.

    ii. Reduced lordosis (flattening/reversal)
1. 'Sniffing position' of cervical spine where face of patient is thrust out anteriorly due to flexion at cervico-thoracic junction but extension at upper segments (ankylosing spondylitis)
2. PIVD (Prolapsed intervertebral disc)
3. Infection
4. Lumbar flatback: osteoporotic/traumatic anterior wedging, advanced degeneration of disc, long thoracolumbar spinal fusion.
5. Ankylosing spondylitis.

3. From back:
   a. Position of head (centrally aligned over pelvis): Plumb line – drop a plumb line from occiput 'inion' and for a straight spine it should pass between the clefts of buttocks
   b. Hair line
   c. Webbing of neck/short neck
   d. Position of shoulders
   e. Comparative position of scapular spine
   f. Scapular angles
   g. Step-off deformity (spondylolisthesis)
   h. Kyphotic and lordotic deformities (as above – its better only to mention them as 'confirm of above findings' rather than stressing them much as most examiners do not like these to be examined from behind!)
   i. List: "A list is an abrupt planar shift (no definite curvature) of the spine above a certain point to one side in coronal plane" – usually seen in lumbar region.
   j. Iliac crest
   k. Posterior superior iliac spine (dimple of venus)
   l. Lateral body margin
   m. Skin for
      i. Lumbar lipoma, hair patch
      ii. Port wine stain (of spina bifida/meningomyelocele)
      iii. *Café au lait* spots
      iv. Nodular skin swelling (neurofibromatosis)
      v. Dermal hemangioma

      vi. Scars
     vii. Sinuses
    viii. Paravertebral spasm
      ix. Swelling
        1. Meningolyelocele
        2. Paravertebral abscess
        3. Hernia
   n. Scoliosis:
      i. Sidedness: (right/left sided convexity)
     ii. Forward bending for making it more prominent (Adam's test) and looking for flexibility, site of primary curve by noting the site of rotation and 'Razor-back' deformity
    iii. Lateral bending to look for possible correction and hence flexibility
    iv. Compensatory curves (note rotation is localized only to the primary curves and rest all other curves are better termed compensatory unless there are two primary curves in an unusual case!)

4. From front:
   a. Head seated squarely over shoulders and chin positioned over sternal notch
   b. Hyoid bone
   c. Thyroid cartilage
   d. Sternocleidomastoid muscle
   e. Sternum: Pectus excavatum, pectus carinatum
   f. Umbilicus

5. Gait: Shuffling gait (posterior cord syndrome), slapping foot gait (high stepping gait), broad-based gait (halting gait), Alderman's gait, festinating gait, antalgic gait, etc. (*See Section 10a*)
   a. Heel walking: to test ankle dorsiflexors (L5 root).
   b. Toe walking: to test ankle plantarflexors (S1 root).

Palpation: Confirm the findings of inspection
   1. Local temperature
   2. Tenderness:
   a. Direct pressure (superficial tenderness often indicate affections of skin), for deep tenderness give direct pressure over spinous process

b. Twist (rotator) tenderness: push the spinous process to either side in an attempt to rotate the vertebra
c. Thrust tenderness: by gently 'thrusting' the spine with a closed fist.
3. Palpation of all spinous processes from inion (note C2, C7, T1, T12) above to coccyx below to identify defect (spina bifida), thickening/deformity (congenital, affections of posterior elements in mitotic pathology), iatrogenic defect (laminectomy) and alignment (normally straight) and abnormal prominence of spinous processes (upper spinous process becomes prominent in all anterior wedging conditions that distract open the spine from behind like traumatic/osteoporotic/tubercular anterior wedging and retrolisthesis while the lower spinous process is made prominent by forward subluxation of spine as in fracture dislocations and spondylolisthesis)
4. Kyphotic and lordotic deformities as above
5. Soft tissue and paraspinal gutters: interspinous region for supraspinous ligament and sites for Pott's abscess
6. From front palpate the carotid tubercle (tubercle of Chassaignac), sternal notch and deformities.

Percussion:
• Spinous process
• Costotransverse joints.

Movements:
• *Flexion:* cervical spine – chin to chest, lumbar spine – forward bending (measured by the final position of trunk in relation to vertical plane or distance of fingers from floor {normal = 90° or < 10 cm finger floor distance)}
• *Extension:* cervical spine – looking roof, lumbar spine – backward bending (normally 20-30°), often limited in patients with facet arthropathy and in foraminal narrowing as extension further narrows the foramina.
• Lateral rotation to right and left side for cervical spine (normal = 80°)

- Lateral bending (thoracic spine) – floor-fingertip distance
- For flexion and extension of thoracic spine (which is minimal) make the patient sit on a wooden straight back chair and ask to bend forward and backwards. More accurate measurement can be made by direct measurement (see below).
- Rotation in sitting position (for lumbar spine) – measure angle between plane of shoulder to pelvis.

Measurements:
- Mark two points from T1 to S1 and a point at L1. Measure and ask patient to bend forward, increase of T1-L1 by >8 cm and L1 to S1 by more than 8-10 cm is normal.
- Modified Schober's test: mark two points – one point 10 cm above and the other 5 cm below lumbosacral junction. Measure the distance before and after forward flexion. At least there must be an increase of > 5 cm otherwise it is pathological.
- Chest expansion – abnormal is <2.5 cm than the average normal value for age and sex (measure at the level of the 4th intercostal space). For males upto 65 years normal is 5.5-7 cm and females 4-5.5 cm. >65 years of age for males 3-4 cm and females 2.5 to 4 cm. *You can safely refer to 5.0 cms as the normal for most healthy adults.*
- Iliocostal distance (from front)
- Iliooccipital distance (from back)

Neurological examination:
- Cervical spine: lateral rotators (right by left sternocleidomastoid and vice versa), forward flexors (both SCM together), extensors (posterior intrinsic muscles and trapezius) and lateral benders (scalene).
- Beevor's sign (for lower thoracic nerve roots): patient performs a quarter squat with hands behind head. Watch the movement of naval up/down or to any side which indicates involvement of lower abdominal musculature (below T9)
- Examination of limb muscles (in distribution, myotomes)
  – Bulk

- Tone
- Power
- Coordination
- Balance
- Involuntary movements (chorea, athetosis, dystonia, tics, myokymia, hemiballismus, asterixis, myoclonus)
- Sensory examination:
  - Fine touch and sharp-dull discrimination.
  - Crude touch and sterognosis
  - Two point discrimination
  - Pressure
  - Temperature (cold and hot)
  - Vibration sense
  - Positional sense (proprioception)
- Vasomotor examination: starch-iodine test, Guttman's test
- Reflexes:
  - Superficial (UMN dependent reflexes)
    - Trapezius reflex (C3-C4)
    - Deltoid reflex (C5-C6)
    - Scapular reflex (C5-T1)
    - Abdominal muscle reflexes (T7-L1)
    - Cremastric (T12, L1)
    - Anal wink reflex(S2-S4): used to determine end of spinal shock
    - Bulbo-cavernosus (S2-S4)
    - Plantar reflex (Babinski sign) – this is a pathological reflex and appears only in UMN lesions
    - Throckmorton's reflex (percuss the dorsum of foot in MTP joint region – great toe extension with flexion of other's is normal)
  - Deep (LMN local reflexes with inhibition from UMN) – lost in LMN lesions and becomes hyperactive in UMN lesions (*a cervical level is UMN for lumbar levels!*)
    - Biceps (C5-C6)
    - Triceps (C6-C7)
    - Brachioradialis (C6-C7)

- ◆ Inversion of radial jerk (C5-C6)
- ◆ Knee (L2-L3-L4)
- ◆ Ankle (S1-S2)
- ◆ Medial hamstring (L5)
- ◆ Tibialis posterior (L5)
- – Ankle clonus and patellar clonus (Due to loss from UMN inhibition).

Special Tests

Nerve root tension signs:
1. Lhermitte's sign (technically a symptom): electric shock like sensation radiating into limbs. If the sensation comes with cervical spine flexion then cervical pathologies are more evident and if it comes with trunk flexion then it indicates thoracic cord lesion. The sign was first described for multiple sclerosis.
2. Spurling's maneuver: extension and rotation of cervical spine produce radicular pain, it is pseudoradicular if the pain radiates to occiput or scapula or limbs but not below elbow.
3. Upper limb tension test 1 (ULTT1) for C5, C6 and C7 and is considered median nerve dominant.
4. ULTT2: stretches C6 and C7, two variants – median and radial nerve dominant.
5. ULTT3: stretches C8 and T1 – ulnar nerve dominant.
6. Axial cervical spine compression test.
7. Straight leg raising test (Passive).
8. Crossed SLRT (well leg SLRT, Frajersztajn test): extremely sensitive for L4-5 and L5-S1 disc.
9. Lasegue's test (Bragard's test): (does not increase the discomfort to hamstrings as in SLRT) – stop at the hip flexion where pain is induced and dorsiflex ankle and foot → radicular pain exacerbated, strengthens the diagnosis of sciatica.
10. Modified Lasegue's test (Kernig's maneuver): raising the leg with knee flexed and slowly extending knee.
11. Bowstring sign of McNab: perform SLRT→ stop at the hip flexion where pain is produced→ flex knee to 90° → press

the sciatic nerve in popliteal fossa → strengthens the impression of sciatica.

12. Cram test: similar to Lasegue's test → flex hip of a supine patient then extend the knee. Reproduction of pain indicates positive test.

13. Slump test: patient seated → flex dorsal spine with extended cervical spine → flex cervical spine also→ ask patient to straighten one leg → ask patient to dorsiflex the foot. Pain at any point means positive.

14. Femoral nerve stretch test (reverse SLRT): for L2-L4 – prone patient → lift the thigh keeping buttocks stable.

15. Single leg hyperextension test for spondylolysis: ask patient to stand with one leg extended than other (straddle position) → ask patient to lean backwards→pain produced on the affected side.

16. Tests to increase the intrathecal pressure:
    a. Valsalva maneuver
    b. Milgram's test: maintain active SLRT for 30 secs once in >30° and <30° hip flexion. If positive in <30° hip flexion then it indicates PIVD
    c. Naffezier's test: jugular compression in seated position→ pain in lumbar region suggests intrathecal space occupying lesion in lumbar region.

1. How do you localize a spinal level from examination?

Ans: The following findings are classical for a particular level along with other findings:

Level	Sensory findings	Motor deficit	Reflex involved
C4	Lateral neck region	Trapezius	Trapezius reflex
C5	Middle deltoid region in arm ("Regiment badge area") - *Axillary nerve*	Deltoid, Biceps (partial)	Biceps reflex, deltoid reflex
C6	Dorsum of first web space (Thumb); lateral forearm - *musculocutaneous nerve*	Biceps brachii, wrist extensors	Brachioradialis (supinator) reflex, inversion of radial reflex, biceps (weakness)

Contd...

Level	Sensory findings	Motor deficit	Reflex involved
C7	Long finger	Wrist flexors, long finger extensors, triceps	Triceps jerk, scapular reflex
C8	Little finger, ulnar aspect of palm - *Ulnar nerve*	Long finger flexors, Hand intrinsic	Scapular reflex
T1	Medial aspect of elbow and lower arm - *Medial brachial cutaneous nerve*	Intrinsic muscles of hand typically interossei	-do-
T2	Medial upper arm and adjacent chest		
T4	Nipple		
T10	Umbilicus	Flexion of trunk (Beevor's sign)	Abdominal reflex
L1	Anterior proximal thigh at groin fold (inguinal ligament) - *ilioinguinal nerve*	Iliopsoas	Cremastric reflex
L2	Mid-thigh (pocket region) - *Lateral, anterior and medial femoral cutaneous nerves of thigh*	Iliopsoas	
L3	Lower thigh and medial aspect of patella - *obturator nerve*	Quadriceps	Patellar tendon reflex (partial)
L4	Medial aspect of leg and ankle - *Saphenous nerve*	Tibialis anterior	Patellar tendon reflex
L5	Lateral and anterolateral aspect of leg, dorsum first web space (medial plantar nerve and lateral cutaneous nerve of calf)	EHL, EDL, Gluteus medius	Tibialis posterior reflex, medial hamstring reflex
S1	Lateral aspect of foot over plantar aspect, posterior calf - *lateral plantar nerve*	Gastro-soleus, peronei, gluteus maximus	Tendo Achilles reflex, plantar reflex
S2	Posterior thigh region, proximal calf	Lax external sphincter in rectal examination	
S3-5	Perianal area	-do-	Anal reflex, bulbocavernosus reflex

2. **How do you test for plantar reflex and what is its interpretation?**

Ans: (*Explain the procedure to the patient*). Stroke the lateral aspect of foot from heel upwards making a 'J' at the region of metatarsal heads. Normally there is a progressive sequence of:

1. Toe flexion
2. Ankle dorsiflexion
3. Inversion of foot

This denotes intact innervation up to L5-S1 level. Mute plantar response can occur in a normal individual and in spinal shock. The pathological appearance of a different sequence is a Babinski reflex (extensor plantar response) which is characterized by:

1. Extension of great toe
2. Fanning out of other toes
3. Ankle dorsiflexion
4. Knee flexion followed by ipsilateral hip flexion
5. Contralateral hip and knee extension (withdrawal reflex)

This dramatic sequence can be overridden in a conscious patient and only first few stages are seen in a typical UMN lesion.

3. **What are the other ways of eliciting this reflex?**

Ans:

   – Squeezing the heel cord (Gordon's sign)
   – Sqeezing the calf
   – Pressing firmly the medial border of leg/tibial crest (Oppenheim's sign)
   – Stroking lateral malleolus.

4. **What other pathological reflexes appear in a UMN lesion?**

Ans:

• Hoffman's reflex {palmar flexion of thumb (and observing a pincer movement between thumb and index finger) on rapidly tapping the distal phalanx of middle finger from palmar aspect}

- Inverted radial reflex
- Tromner sign – similar to Hoffman's reflex but here the middle finger is elevated and distal phalanx is flicked towards palm
- Crossed adductor's sign – ipsilateral patellar reflex causes contralateral thigh adductors to contract
- Chaddock's sign – abduct little toe and release it to slap against other toes or flick third and fourth toe down rapidly to look for great toe dorsiflexion.
- Clonus!

  (*Note that they may be present in a patient with brisk reflexes and may not necessarily indicate pathology, however asymmetry is of significance*)

  5. What is the bladder dysfunction associated with spinal cord injury?

Ans: The following should help explain the mechanism of two different cord bladder functioning. First there are two differently innervated muscles in bladder control. One is the bladder motor (detrusor) which is under local spinal control S2-4, whereby any stimulus that arises due to bladder filling leads to contraction of bladder and emptying. The other one is the innervations of sphincters which is inhibited (relaxed) by local reflex (S2-4) and stimulated by sympathetic system (L1-2). Now secondly there is a higher control which has an inhibitory influence on local reflex (S2-4).

Now if local reflex is released from all upper inhibitions (as happens in UMN lesion) the bladder becomes an '**Automatic bladder**' which will contract as soon as it fills up and there will be detrusor hypertrophy due to hyperactive local reflex (S2-4). If this local reflex is lost (as will happen in S2-4 lesions like cauda equina syndrome – LMN lesion) the bladder will not be able to contract due to its lost motor supply and also the sympathetic (L1-L2) reflex is still activating the sphincters that additionally prevent emptying. This type of bladder keeps filling till the capacity is exceeded when the urine dribbles due to

exceeded competency of sphincters – 'atonic bladder'. With time, however intramural reflexes develop whence with overfilling the bladder may contract due to direct detrusor stimulation – the so called '**Autonomous bladder**'. (*Remember the aphorism – the one who is released from higher control becomes 'automatic' whilst the one who gains self control due to loss of virtually everything becomes 'autonomous'*)

6. How do you predict the level of cord involvement with the vertebral (spinal) level?

Ans: The following is the rule of thumb for an adult:

Vertebral level	*Cord level*
C1-C7	Add one to know corresponding cord level
T1-6	Add two
T7-9	Add three
T10	L1 and L2
T11	L3 and L4
T12	L5 and S1
L1 (cord ends at lower border of L1)	Rest sacrals and coccygeal segment
Below L1	Cauda equina

7. What is clonus?

Ans: It is a pathological hyperreflexia of normal deep tendon reflexes produced due to release of the normal reflex from higher control. Sustained clonus (> 5 beats of clonus) is significant and represent UMN lesion. Ill-sustained clonus can occur in an otherwise normal individual.

8. What do you do to make Reflexes more prominent if they are not elicited by normal maneuver?

Ans: I will do Jendrassik's maneuver by asking patient to either clench his teeth tight or pulling the interdigitated fingers of both hands apart to enhance the reflexes.

9. How do you differentiate Rigidity vs. Spasticity?

Ans: Both represent types of hypertonia. *Rigidity* is pathologically due to *extrapyramidal* affection. It can be of

cogwheel type (jerky or intermittent regular resistance to motion) or lead pipe type (present throughout the range of motion). *Spasticity* is more of practical concern in the context of spine examination and is due to affection of *pyramidal system* (corticospinal pathway). It is classically a UMN lesion and is of clasp-knife type whereby resistance is offered only to the initial part of movement and disappears on persisting with the same. A hysterical hypertonia classically increases with the effort applied.

10. What is central cord syndrome?

Ans: Central cord syndrome occurs in the cervical level often due to hyperextension injuries or in an elderly due to stenotic cervical canal.

The upper limb is more severely affected with initial affection of pain and temperature and later involvement of upper limb motor function and lower limb with progression. The syndrome may also be produced by central canal syrinx or sometimes intraspinal tumors.

11. What is anterior cord syndrome?

Ans: Due to involvement of ventral portion of cord (interruption of ascending spinothalamic tracts and descending motor tracts. It is characterized by:
- Loss of pain and temperature sensation
- Loss of motor control below the affected level
- Preservation of proprioception and crude touch

12. What is Brown-Sequard syndrome?

Ans: Hemisection injury of spinal cord:
- Ipsilateral motor loss below the level
- Loss of reflexes at the level
- Contralateral loss of pain and temperature one to three levels below the level of lesion
- Ipsilateral loss of proprioception and position sensation and crude touch
- Band of hyperesthesia at the level

CASE I:
SPINAL TUBERCULOSIS (POTT'S DISEASE)

Clinical Findings

- Pain and stiffness of spine: pain may be referred to arms, intercostal neuralgia in cervical and thoracic regions. In thoracolumbar region 'girdle pain' or epigastric pain may be indicated.
- Paravertebral spasm and 'boarding'
- Kyphotic deformity
- Tenderness (deep thrust and rotational)
- Night cries
- Abscess formation and paraspinal swelling.
- Constitutional symptoms – Evening rise of temperature, night sweats, anorexia, weight loss, asthenia, tachycardia, anemia
- Gait: upper thoracic disease – 'military attitude'; lower thoracic and upper lumbar disease, 'Alderman's gait'
- Paralysis

In Heroin addicts a distinct syndrome has been reported having acute toxic reaction with fever, back pain, weight loss, night sweats, and rapidly developing neurological deficits.

1. What are your differential diagnoses?

Ans: Apart from spinal tuberculosis:

1. Pyogenic osteomyelitis and discitis
2. Metastasis from primary tumor elsewhere (older age group)
3. Primary tumor of spine (younger people – think of lymphoma, as such anything is possible like multiple myeloma, GCT, ABC)
4. Actinomycosis of spine
5. Brucellosis, *Salmonella typhi, V. cholera* pyogenic discitis (as D/D for paradiscal type)
6. Eosinophilic granuloma (Calve's disease)
7. Hemangioma

8. Early thoracic disease has the following differentials
   a. Rickets
   b. Scoliosis
   c. Osteochondroses (Scheuermann's disease)
   d. Schmorl's disease
9. Isolated ivory vertebra:
   a. Lymphoma, Paget's disease, osteoblastic metastasis.

2. How does the organism reach vertebral column?

Ans: Associated active infection is seen in < 10% cases (*most commonly – pulmonary and urogenital, uncommon – cutaneous or lymphadenopathy*). The infection reaches spine mainly via:
   a. Arterial vascular channels
   b. Batson's perivertebral venous plexus
   Other uncommon routes are direct spread from mesentry or cysterna chlyi, direct implantation.

3. Why is Pott's disease most common in dorsolumbar region?

Ans: >50% cases seen in D-L region due to:
   a. Greater extent of movements
   b. Degree of weight bearing and hence microfractures
   c. Larger area of spongy cancellous bone
   d. Proximity to kidney and cistern chili
   In decreasing order of involvement; D-L region → lumbar → upper dorsal → cervical → sacral
   Children have higher incidence of cervical spine TB.

4. What are various types of tubercular involvement of spine?
Ans:
   a. Paradiscal involvement: infection spreads through epiphyseal arteries→ disk and adjacent vertebral bodies. This type is more common in lumbar lesions and is overall the most common type.
   b. Complete lesions: destruction of one/two vertebral bodies, > in children under 10 years→ prone to late onset paraplegia.

c. Central lesions: infection spreads through Batson's plexus/posterior vertebral artery. Concentric collapse of body. Minimal discal involvement or paravertebral shadows. This type is more common in dorsal spine.

d. Anterior lesions: infection under anterior longitudinal ligament. Collapse and discal involvement late.

e. Posterior lesions (appendicial/apophyseal spinal TB): single pedicle involvement (Winking owl appearance), spinous process involvement (Beakless owl). Increased chances of neurological deficit due to proximity to spinal canal and often delayed diagnosis (in lumbar appendicial TB neurological deficit is late to appear due to ample space in canal).

f. Skip lesions: lesions separated by 2-3 normal vertebral levels

g. Spinal tumor syndrome: lesion starts at posterior margin with cord compression by ensuing granulation tissue.

h. True tuberculosis arthritis; at occipito-atlanto-axial joint.

5. Why do you see bony Ankylosis in spine with healing whereas fibrous Ankylosis in hip joint?

Ans: Hip joint is a synovial joint with hypertrophied synovial membrane and granulation tissue (pannus) that intervenes the apposition of cancellous bony surfaces, also the cartilage destruction is often not complete thus with healing disease there is fibrous Ankylosis. In spinal TB there is apposition of large cancellous surfaces preceded by destruction of intervening disc; this is aided by paraspinal spasm and weight transmission acting as compression devices. (*In fact absence of bony fusion and fibrous pseudoarthrosis are 'at risk' factors for late recurrence of disease*)

6. What are the differences in childhood and adult spinal TB?

Ans:

1. Average vertebral loss in children is nearly twice that of adults

2. Collapse is rapid and severe in children due to flexibility
3. There is constant increase in deformity in children due to growth even after healing of disease
4. Children are more prone to late onset paraplegia.

8. Is there a method to predict the future deformity?

Ans: The deformity at 5 years (follow-up) can be predicted with a fair level of accuracy by calculation of pretreatment vertebral body loss according to the following formula:

$Y = a + b*X$

Y= final deformity (in degrees) at 5 years follow-up, X= pretreatment vertebral body loss, a=5.5 and b=30.5 are constants.

9. What are the various types of collapse in spinal TB?

Ans: Collapse in TB spine:
1. Telescopic – along the long axis (more common in lumbar spine)
2. Flexion – in the sagittal plane of one spinal segment on other (dorsal spine).

10. Why does the collapse so happens?

Ans: Weight is transmitted through articular processes (anterior ones) located posteriorly in cervical and lumbar spine (from where the line of weight bearing pass) whereas in dorsal spine the line of weight bearing passes anteriorly through body (anterior column) so flexion collapse occurs.

11. What is cold abscess?

Ans: Cold abscess consists of TB debris, disintegrated bone lamellae, serum, caseous material, granulation tissue, bone marrow, TB bacilli.

12. Where all does cold abscess track into?

Ans: Cervical lesions:
– Retropharyngeal abscess: collection behind prevertebral fascia

– Spread lateral to posterior triangle of neck and present at posterior border of sternocleidomastoid muscle
– Mediastinum → mediastinitis (may be life threatening)
– Axilla/cubital fossa (along brachial plexus)

Dorsal lesions:
– Paravertebral abscess (*upper dorsal spine – squaring of mediastinum, mid-dorsal spine – fusiform swelling*)
– Extrapleural space
– Intrapleural space → empyema thoracis
– Along intercostal nerves and vessels

Lumbar lesions:
– Psoas abscess
– Petit's triangle/lumbar triangle
– Scarpa's triangle
– Posterior aspect of thigh/popliteal region.

13. What are the radiological findings of spinal tuberculosis?

Ans: Osteopenia, decreased disc space, loss of paradiscal margins are the earliest and most common findings:

Severe thoracic disease may show up as 'Bird-Nest appearance' due to crowding of ribs and 'eggs' as destroyed vertebral bodies.

In anterior lesions erosion of anterior part of vertebral body without much change in disc space shows up as 'aneurysmal phenomenon' akin to erosion by aortic aneurysm.

In heroin addicts the features may be atypical including ivory vertebra.

14. What other imaging modalities you know of?

Ans: MRI is the imaging modality of choice as both osseous and soft tissue details are evident. Intervertebral disc height is often preserved in TB whereas they are destroyed quite early in pyogenic infection. Also in gadolinium enhanced images the abscess shows peripheral enhancement whereas granulation tissue solid mass enhances *in Toto*. The role of computed tomography scan (cannot differentiate granulation tissue from abscess) and

scintigraphic (neither sensitive nor specific) scanning have lost ground to MRI.

15. How do you classify Pott's spine?

Ans: Clinico-radiological classification of tubercular spondylitis (Kumar 1988)

   I. Predestructive: straightening of curves, paraspinal spasm, marrow edema (MRI); < 3 months

  II. Early-destructive: $\downarrow$ disc space, paradiscal erosion (K: <10°), marrow edema + osseous break, marginal erosions or cavitations; 2-4 months

 III. Mild angular kyphos: 2-3 vertebrae involved (K: 10-30°); 3-9 months

 IV. Moderate kyphos: >3 vertebrae involved (K: 30-60°); 6-24 months

  V. Severe kyphos: > 3 vertebrae involved (K: >60°); >2 year

Stage III and further all have vertebral body destruction and collapse + appreciable kyphos.

16. How do you classify Pott's paraplegia?

Ans: *Remember Pott's paraplegia is more common in dorsal and cervicodorsal lesions as the spinal canal is narrower and there is propensity towards kyphosis and retropulsion. In the lumbar lesions it is less common as the canal is wider and cord ends at lower border of L1.*

Girdlestone and Griffith's classification:

1. Early onset paraplegia – develops within 2 years of disease onset.

2. Late onset paraplegia – develops after 2 years of onset.

Hodgson's classification (etiological):

1. Paraplegia due to extrinsic causes: viz. abscess, sequestrate, discal fibrosis, pathological subluxation/dislocation of vertebrae, transverse ridge of bone anteriorly

2. Paraplegia due to intrinsic causes: viz. meningitis/ meningomyelitis, inflammatory thrombosis, etc.

Seddon's classification:

• Paraplegia of active disease

• Paraplegia of healed disease

17. What are the causes of Pott's paraplegia?

Ans: Active disease (good prognosis):
- Compressive pathology:
  - Inflammatory edema
  - Granulation tissue and Caseous tissue with sequestrated material
- Infective vasculitis
- Spinal tumor syndrome
- Pathological dislocation of spine
- Direct infiltration of bacilli into cord with ensuing inflammatory process.

Healed disease (mechanical pathology with guarded prognosis – treatment surgical):
- Stretching of cord over bony ridge at apex of deformity (Internal Gibbus)
- Progressive constriction of cord due to epidural fibrosis.

18. How do you stage/grade paraplegia?

Ans: Kumar's clinical staging

Stage 1: patient unaware, examination reveals extensor plantar response or ankle clonus (sustained)

Stage 2: patient has in-coordination and reports weakness but manages to walk with support

Stage 3: patient confined to bed 'paraplegia in extension' and variable sensory blunting (<50%)

Stage 4: "paraplegia in flexion" with sphincters involved, some also include flaccid paraplegia in this stage, sensory involvement >50%

(*In-coordination is the earliest symptom and clonus is the earliest sign*)

19. What is the cause of paraplegia in extension?

Ans: During progression of disease it is a stage when there is differential involvement of fibers in cord (predominantly related to the types of fibers) whereby the inhibitory pathways to local reflexes are lost but motor power and intrinsic tone of muscles is still there so this produces a 'spastic' state and the antigravity muscles predominate producing paraplegia in extension and brisk reflexes with clonus.

20. What is the cause of flaccid paraplegia?

Ans: It is the end-stage in evolution of disease whereby all motor control and muscle tone is lost due to severe compression and possibly arachnoiditis.

21. How will you manage the patient?

Ans: After confirming diagnosis, I will manage the patient on middle path regimen and start ATT with brace stabilization and continue close supervision to assess and look for surgical indications. If the progression is good the course is followed.

22. What is middle path regimen?

Ans: In short it can be stated as 'drugs for all and surgery for failure'; *the above protocol described comes under middle path regimen.* This includes symptomatic supervised closely observed conservative treatment with expectation of improvement and includes rest, ATT, gradual mobilization, aspiration of large abscesses and collections to reduce discomfort, management of sinuses and close observation with clinic-radiological progress.

23. What ATT course do you give?

Ans: *Please see Section 2b case I,Q 19*

24. Why DOTS is not preferred?

Ans: DOTS (directly observed treatment in supervision) is often not possible for orthopedic patients as they are often unable to visit the center regularly due to disability! Also uncomplicated musculoskeletal tuberculosis is classified as class 1 in WHO regime that can be given intermittently. *Do learn WHO regime from standard pharmacological texts.*

25. Is there any condition when you will not give ATT during perioperative period?

Ans: There is only one condition where ATT is not required in perioperative period – late onset paraplegia from progressive deformity in a patient with healed inactive disease.

26. What are the indications for surgery in Pott's paraplegia/paresis?

Ans: Indications for patients on middle path regimen:

1. No neurological recovery even after 4 wks of chemotherapy
2. Development of neurologic complications during chemotherapy
3. Recurrence of neurological complications during chemotherapy
4. Worsening of neurological complications during chemotherapy
5. Advanced cases of neurologic involvement (stage 4)
6. Rapidly advancing paresis which is advancing daily
7. Common indications (whether or not neurological complications present): patients with prevertebral cervical abscess and difficulty in deglutition, dorsal spine involvement with spasmodic respiration, older patients in whom one would like to avoid complications of prolonged recumbency.

27. What are the indications of surgery in a patient who does not have neurological complications?

Ans:

1. Failure of clinical improvement after 6-10 weeks of ATT (modified from original middle path regimen that observes for 3-6 months – most examiner observe for upto 3 months)
2. Recurrence of disease
3. Primary drug resistance or history of irregular chemotherapy
4. To prevent deformity
    a. Adult: Vertebral body loss >1 in dorsal and D-L regions and >1.5 in lumbar region
    b. Children who present with a kyphus >30° before start of treatment
5. Rare indications:
    a. To establish diagnosis (only when CT-guided biopsy inconclusive)

b. In patients with persistent sinuses and abscess
c. Tuberculosis of cervical spine with paravertebral abscess causing difficulty in deglutition and respiration.

28. Why do you perform surgery?

Ans: Surgery is performed to:
1. Drain abscess
2. Debride sequestrated bone
3. Decompress spinal cord
4. Stabilize the spine for prevention or correction of deformity

(*Basically this is a play question! Option '1' and '2' achieve '3' and '4' has often to be added to '2' in multisegment involvement,* this question estimates whether you are aware of surgical procedures and their role)

A combination of all is often required.

29. If there is no improvement for a patient on middle path regimen then what will you do?

Ans: This is an indication for surgical decompression.

30. How will you decompress?

Ans: *Choose one of the options as below!*
1. Abscess drainage around spine/pelvis
2. Abscess drainage + limited debridement
3. Radical debridement of disease focus and anterior arthrodesis + bone grafting
4. Above + posterior fusion to prevent progression of deformity

It is fair to choose option '1' for limited disease viz. single level perispinal collection without any neurological complications or any evidence of instability but better to choose option '3' for adult patients and option '4' for children for ≥ 2 level disease where operative procedure is planned.

Option '2' is not very much favored and most spine surgeons agree to the conclusions of working party on tuberculosis of spine in Medical Research Council trial that Hong Kong procedure of radical debridement and anterior strut grafting is superior.

Nowadays option '1' is practiced as a minimally invasive guided procedure like ultrasound guided pigtail drainage of abscess that is quite satisfactory for limited disease.

31. How will you manage disease and drain abscess at various locations?

Ans: C*ommon routes are mentioned with special situations in sub-tabs*:

- Cervical spine: Avoid rupturing into septic pharynx, so evacuate by neck (extraoral); except in emergency when direct posterior pharyngeal wall incision may be given
  - 1st and 2nd cervical vertebrae → approached transorally with or without supplementary occiput to C2 fusion
  - 3rd – 7th cervical vertebrae → standard anterior approach between carotid sheath and esophagus (anterior triangle), or through posterior triangle.
- Thoracic region: Do costo-transversectomy (for abscess drainage only) additional decompression of anterior spinal canal would need extrapleural anterolateral decompression (ALD)
  - Cervicothoracic region → sternum splitting approach 'or' anterior approach
  - If anterior arthrodesis also required then use transpleural approach instead of costotransversectomy.
  - Lateral rachiotomy (modified costotransversectomy) is reserved for late-onset paraplegia with large kyphotic deformity where lateral exposure of dura is required.
- Lumbar region: Direct drainage along lateral border of sacrospinalis between last rib and rest of ilium
  - Dorsolumbar junction → left sided abdominothoracic approach through bed of 11th rib
  - Lumbosacral junction → extraperitoneal route/ paramedian transperitoneal route.

32. What is Hong Kong procedure?

Ans: This is a radical debridement of spine with anterior strut grafting whereby spine is approached anteriorly. The

sequestrated and caseous material is removed up to bleeding bone up and below and back to posterior longitudinal ligament (*the decompression should go back to duramater in cases of neurologic deficit when spinal cord decompression is necessary*). Angular deformity if present is corrected with the strut graft.

33. What are the choices for strut graft?

Ans: In terms of preference:
1. Full thickness iliac crest
2. Vascularized rib graft
3. Rib strut graft
4. Vascularized fibula
5. Fibular cortical strut graft.

34. What is anterolateral decompression?

Ans: Position patient in right lateral position (avoids venous congestion and excessive bleeding, lung and mediastinal contents fall anteriorly). Semicircular (Convex laterally) incision centered at the pathological site spanning a distance 5-6 cm proximally and distally and apex of incision 9-10 cm laterally is placed (Capner incision). Skin with fascia lifted to minimize bleeding. 2-4 ribs identified and traced up to transverse process. Ribs are cut some 8-9 cm from transverse and lifted up and removed. Transverse process removed from their base completing costotransversectomy (Menard procedure). For ALD identify the intercostal nerves and vessels laterally, tracing them medially guides the pedicles which are then removed along with diseased vertebral bodies over posterolateral aspect hence decompressing anterolateral part of spinal canal (*the question may be asked in the form like – what structures are removed in ALD?*). Some 3-6 cm of anterolateral canal should be exposed for proper decompression *completeness of which is judged by the reappearance of evident cord pulsations and passing a small red rubber catheter up and below the spinal canal for adequate space.* In younger children posterior fusion on the other side can be done.

35. What is the role of laminectomy?

Ans: Now condemned as it is ineffective in decompressing the anterior part of cord (*which is the commonest site of* compression) and in addition renders the spinal segment unstable (unless circumduction fusion done simultaneously). It can be considered however only for limited cases in patients in whom the lesion is posterior (appendicial TB) or those with spinal tumor syndrome (posterior epidural tuberculoma).

36. Is it recommended to use instrumentation in spinal tubercular "infection"?

Ans: Anterior surgery alone is often ineffective and *posterior instrumentation is often additionally* required as a staged procedure or in single sitting which is now an established modality. Also posterior fusion alone without instrumentation or anterior surgery is considered as a poor planning as it does not control progressive kyphosis. Implant associated infections are related to altered local environment and bacterial thriving around implants due to relative inadequacy and ineffectiveness of host defenses and antibiotics respectively. Biofilm formation plays a significant role in evading defense mechanisms and safeguarding bacteria from chemotherapy. *Mycobacterium tuberculosis* has limited tendency to adhere or produce biofilms so risk of persistent infection is smaller. This has led to now an increasingly popular use of anterior instrumentation.

37. What are the indications for anterior instrumentation?

Ans: Often the graft failure occurs if they are used for disease that spans *more than two disc levels*. In these cases the option is either to use simultaneous anterior instrumentation or a two staged surgery. In the two staged surgery where first a posterior instrumented fusion is done followed by anterior debridement and fusion, the first stage prevents loss of correction.

38. How will you manage dorsal spine TB?

Ans: Mehta and Bhojraj have proposed a system for *surgical* management dorsal spine TB (based on type of involvement):

Group A: paradiscal/central involvement without deformity – transpleural debridement with fusion (no instrumentation)

Group B: 'A' + deformity – above + posterior instrumentation

Group C: 'A' but too ill to undergo transpleural surgery – transpedicular decompression and posterior instrumentation

Group D: posterior involvement only – posterior decompression only

39. What are the factors that affect the surgical outcome?

Ans: Good outcome can be expected in:
- Minimal destruction and small graft required for maintenance
- Good intraoperative correction
- Involvement of lower lumbar segments

Poor outcome more common in:
- No perioperative chemotherapy
- Poor nutrition
- Vertebral body loss > 2
- Junctional lesions
- Marked preoperative kyphosis
- Frank instability
- Post-debridement defects requiring grafts spanning > 2 disc spaces.

40. How does graft failure occur?

Ans: Four types:
1. Displacement
2. Fracture
3. Absorption
4. Subsidence

41. Why is deformity especially kyphosis of concern?

Ans: There is:
- Foreshortening of trunk
- Short stature
- Cosmetic problems
- Reduced pulmonary function
- Secondary cardiac and respiratory problems
- Progression leads to internal cord stretching and late onset paraplegia

42. What is K-angle?

Ans: K-angle (short for kyphosis angle) is a measure of present kyphotic deformity. There are two methods to measure the same, one along the posterior border of vertebrae (Dickson 1967) and the other by dropping tangents to end plates of upper and lower end vertebrae. Angle >60° indicates severe deformity and requires surgical correction or anterior transposition of cord.

43. What are the risk factors for severe increase in deformity?

Ans: The following are associated with increase in deformity
1. Patients < 10 years of age at onset
2. Initial kyphosis > 30°
3. Vertebral body loss >1.5
4. Involvement >3 vertebral bodies
5. Evidence of instability in X-ray
6. Computed tomography showing involvement of both anterior and posterior structures
7. Children who have no/partial fusion.

44. What are 'spine at risk' signs?

Ans: Four:
1. Facet dislocation
2. Retropulsion sign
3. Lateral translation sign
4. Topple sign.

(*Score = maximum of 4, minimum of 0; score >2 associated with higher increase in final deformity*)

45. Can you classify deformity progression in children?

Ans: Three main types of deformity progression are seen in children during growth spurt:

I. Continued deformity throughout entire growth period
   a. Deformity increases continually after active period
   b. There is a lag period of 3-6 yrs

II. Shows beneficial effects during growth with a decrease in deformity
   a. Immediately after growth period
   b. After a lag period of 3-6 yrs

III. No major change in deformity.

46. Does level of spine involved have a bearing on deformity progression?

Ans: Yes,
- Dorsal level has higher deformity at presentation that progresses fast and the articular facets are horizontal so there is early subluxation and dislocation
- Dorsolumbar junctional disease – has worst prognosis because of inherent instability at the transitional zone from kyphosis to lordosis and loss of protection of rib cage
- Lumbar has best prognosis: there is often telescopic collapse due to narrowness of pedicles.

47. What factors are associated with poor recovery of cord?

Ans:
1. Inactive disease with poor mechanical compression
2. Complete paralysis
3. Paralysis for >1 year
4. Presence of severe kyphosis
5. Older age and poor nutrition
6. Presence of myelomalacia and syringomyelia on MRI.

48. What surgery will you do for healed disease with paraplegia?

Ans: Anterior decompression with removal of healed disease and stabilization with bone graft with/without anterior instrumentation.

49. What are the indications of surgery to prevent deformity in spinal Tuberculosis?

Ans:
1. Loss of 0.75 vertebral levels in dorsal/dorsolumbar region and loss of 1.0 in lumbar region
2. Children with > '2' spine at risk signs.

50. How will you manage severe kyphotic deformity?

Ans: For patients without neurological complications manage as per *Q 27*. For paraplegics with severe kyphotic deformity do anterior transposition of cord using exprapleural anterolateral approach. A more aggressive and probably better method is to stabilize spine posteriorly followed by anterior debridement and bone grafting in healed disease using transthoracic approach. For healed disease anterior debridement followed by posterior instrumentation and anterior fusion using titanium cages filled with bone grafts is used.

51. How will you manage craniovertebral lesions?

Ans: Behari et al classified patients into four grades depending up on presentation and respective treatment:
1. Grade I: Neck pain only and no pyramidal signs – treat with brace immobilization and medical therapy only
2. Grade II: Independent patient but minor disability – treat as above
3. Grade III: Partial disability requiring assistance with activities of daily living – anterior decompression followed by posterior fusion
4. Grade IV: Severe disability with respiratory compromise – treat as for grade III.

52. What are the advantages of costotransversectomy?

Ans:
– Attacks the main cause of paraplegia
– Drainage is away from cord
– Does not weaken spine
– No great operative risk

This does not decompress the anterior compressive pathology that requires ALD.

CASE II:
LUMBAR DISC DISEASE (PROLAPSED INTERVERTEBRAL DISC DISEASE)

1. **What is the cause of pain in PIVD?**

Ans: The following have been pointed variously as cause of pain:
- Irritation of nerve root due to:
  – Compression (? – pure compression produces only motor and sensory changes without pain so inflammation should coexist, *dorsal root ganglion* is very sensitive to compression and vibratory forces and may be an important pain generator)
  – Stretching
  – Friction
  – Occlusion of vasa nervorum (by inflammatory emboli/thrombi/vasculitis)
  – Degeneration of nerve fibers (due to relative ischemia – 'cry of dying nerves')
  – Combination of above (*this has been deliberately added here if you are exhausted of etiology then you can 'extend' your viva with this universal option – beware this can offend an ostentatious examiner*)
- Persistent pain due to facet joint arthropathy ('vertebrogenic pain' from zygapophyseal joints, the capsule and synovial folds possess pain fibers)
- Engorgement of extradural veins
- Tearing of annulus fibrosus containing sensory nerve endings ('sinuvertebral' nerve) present in outer thirds of annulus
- Biological factors (now considered much important):
  – Neurochemical changes due to localized or systemic inflammatory changes against extruded nucleus.

2. **What fibers are responsible for mediation of pain?**

Ans: Small, myelinated (A-delta) fibers and unmyelinated C fibers.

3. What are various types of disc herniations?

Ans: Pathological staging (Eismont and Currier) clarifies the variously used terms:

1. Dehydration, dessication with early degeneration of disc material (the 'degenerative disc disease')
2. Prolapsed of nucleus pulposus (circumferential symmetric disc extension around the vertebral border) within annulus fibrosus ('Disc Bulge') {Contained herniation}
3. Disc 'protrusion' consists of focal or asymmetric extension of disk beyond vertebral border. {Contained herniation}
4. 'Extruded' disc material through annulus but is in continuity with the remaining nucleus but not through posterior longitudinal ligament ('Disc extrusion') {Contained herniation}
5. 'Sequestrated' disc material through both annulus and posterior longitudinal ligament which is not continuous with the disc material ('Disc sequestration') {Uncontained herniation}
6. 'Migrated' disc identifies disc material displaced from the site of extrusion (may or may not be sequestrated).

To this is added "Intermittent disc herniation of 'Falconer' or 'concealed disc' of Dandy – this is not obvious in position of flexion on table but can be reproduced by hyperextension of spine.

Contained herniations mean that disc is still subligamentous. '3' and '4' may present as a firm ridge with herniation across the canal.

4. What are the various sites of disc herniation?

Ans: Topographically disc herniation can be:

1. Central zone
2. Lateral recess: includes the colloquial variably used terms like 'paracentral', 'posterolateral', 'juxtacentral'
3. Foraminal
4. Extraforaminal zone

'3' and '4' are commonly indicated in the term "far lateral disc herniations".

5. What do you mean by the term Sciatica?

Ans: Sciatica is the common term for 'referred pain' to lower limb and can occur in other lumbar spine disorders.

6. What are your differentials?

Ans: The following must be considered:
1. Inflammatory disorders:
   a. Infections
   b. Ankylosing spondylitis
2. Vertebral tumors both primary and metastatic
3. Nerve tumors: Ependymoma, meningioma, neurinoma (most likely to resemble PIVD – pain increases on coughing)
4. Spondylolisthesis
5. Radiculitis
6. Spinal stenosis, foraminal stenosis/lateral recess syndrome
7. Piriformis syndrome
8. Gynecological/genitourinary condition.

7. Why is the symptomatology more often episodic spanning months to years of symptom free intervals sometimes?

Ans: There can be regression in symptoms due to:
1. Degeneration of compressed nerve fibers
2. Adjustment of nerve root in displaced position
3. Diminution of swelling of herniation
4. Disappearance of edema of nerve root
5. Disappearance of central excitatory state
6. Spontaneous resolution of inflammation.

8. Are there any features that help you judge the site of prolapsed disc?

Ans: The following features can serve as 'localising signs' and help distinguish the various herniations:

Central disc:
- Asymmetric muscle weakness and wasting in legs
- Radicular sensory loss over sacrum, perineum, and back of leg

- Impotence, urinary frequency and or retention

Peripheral disc herniation:

- Segmental root pain
- Corresponding segmental muscle weakness and wasting
- Absent segmental reflexes
- Late development of sphincter disturbance and bilateral signs

9. What do you understand by 'axillary' and 'shoulder' presentation of disc?

Ans: These describe the presentation of herniated material in relation to nerve root. Axillary presentation means that disc material is inferomedial to the nerve root whereas 'shoulder' presentation is understandably superolateral to the nerve root.

10. What is its significance?

Ans: (*It's in the very human nature to flee away from irritating factors*). The body tries to 'take away' and reduce stretching of the nerve 'over' the 'irritating disc' by adjusting the body posture and giving some respite. This is done by 'tilting' towards the affected side in axillary presentation and away from the affected side in shoulder presentation. This presents as the typical list observed in PIVD.

11. What are the red flag signs and its importance?

Ans: Red flag sign's in patient history should alert one for an early MRI, these include:

1. Constitutional symptoms like fever, chills, sweats, weight loss, anorexia
2. History of significant trauma
3. History of malignancy
4. Osteoporosis
5. Age > 50 years, <18 years
6. Severe or progressive neurological deficit and multi root deficits
7. Ongoing infection
8. History of immunosuppression.

12. What are the various presentations of back pain?

Ans: Fairbank and Hall classification:

Type I: "Simple' or 'non-specific' back pain – acute attack, no obvious reason, radiates upto buttocks; never below knee, worse with sitting better with activity. Allright with activity. Treatment is activity

Type II: "Chronic back pain" – chronic persisting signs and symptoms, insidious onset, pain may progress from back → buttocks → thigh → legs depending on severity, so-called 'thermometer pain'. Tends not to respond to physiotherapy.

Type III: "Neurogenic claudication" – walking related back pain, intolerant of walking or standing straight

Type IV: "Unclassifiable" – tumor, infection and psychogenic back pain.

13. How will you differentiate between neurogenic and vascular claudication?

Ans: The following features favor neurogenic claudication:

- Patient feels pain with weakness in legs so that they may fall but in vascular claudication it is often a cramp or tightness that progresses to pure pain.
- Patient has a variable walking distance and has no relation to speed of walking whereas in vascular claudication the walking distance constantly decreases and appears early with brisk walking.
- Symptoms appear proximally (back → buttock → leg) and progress distally in neurogenic claudication whereas they are reverse (leg) in vascular claudication.
- Patient or spouse often notes a flexed posture after some walking in neurogenic claudication whereas limp often occurs early in vascular one.
- Easier to walk uphill (due to inherent flexion of spine while going up) in neurogenic.
- Riding a bicycle is not painful in neurogenic claudication.
- Often not possible to 'walk through' the pain (as can be done in vascular claudication).

- Longer duration of recovery from pain in neurogenic claudication – 5-30 minutes (1-3 minutes in vascular).
- Taking simply a flexed posture reduces the pain and gives respite in neurogenic claudication but patient has to sit and take rest in vascular claudication.

14. How do you localize a segmental lesion?

Ans: Based on the clinical finding of positive stretch testing and corresponding involvement in respective sclerotome, myotome, and deep and/or superficial reflexes.

15. What are the findings for lumbar fourth segment involvement?

Ans: The following findings support lumbar fourth nerve root involvement:
- Positive sciatic nerve stretch test
- Weakness of tibialis anterior and/or knee extension (quadriceps muscle)
- Sensory loss over medial leg and ankle
- There may be weakness of patellar tendon reflex
- Patient is unable to walk over heel (or in weakness with power >3/5 there would be fanning of toes with loss of arch).

16. What are the findings in fifth lumbar root involvement?

Ans: Following would be seen apart from positive stretch testing:
- Weakness of EHL, EDL, knee flexion (hamstrings), TFL, Gluteus medius (hip abduction)
- Sensory loss over lateral and anterolateral leg and dorsum of foot (typically in the first web space – deep peroneal nerve territory)
- Weakness or mute posterior tibialis reflex and medial hamstring reflex
- Positive trendelenburg sign (variable)
- Trendelenburg gait (variable)

17. When do you call sciatic stretch test to be positive?

Ans: Firstly the stretch test should be performed passively (Passive SLRT). The nerve is stretched only during 35-70°

range of movement in SLRT (*A typical L5 or S1 nerve is deformed by 2-6 mm in SLRT*). In the first 35° the slack in the nerve is taken up and beyond 70° the deformation of sciatic nerve occurs beyond spine and is not sensitive for radicular pathology. So a stretch test for being positive should elicit pain during this range. Sometimes with intense acute inflammatory reaction it may not at all be possible to lift the leg. This can be taken as positive with other findings but should prompt for an urgent search for pathology. A mere feeling of 'posterior stretch' is NOT a positive SLRT and is common between 70-90° range (*Not all of us are athletes*).

18. What is the interpretation of Cross-leg SLRT?

Ans: Cross-leg SLRT (well leg SLRT) is quite specific and sensitive for prolapsed symptomatic disc. This test is positive due to micromotion of ipsilateral (affected side) nerve root on performing the test in asymptomatic limb. This test signifies that the disc is central or that there is a large lateral recess herniation and it is more specific for a 'free' disc fragment.

19. What does the segment testing indicate with respect to level of herniation?

Ans: A herniated disc produces radiculopathy in nerve root that crosses the disc at that level. For example in L4-L5 disc herniation L5 nerve root lies in the lateral recess that presents with the radicular signs. This is due to the peculiar disposition of nerve roots in the spinal canal. Understand the following thoroughly. Nerve root leaves the *cauda equina* ('cord') one level 'above' its exiting foramen (so L5 nerve root that will exit the L5-S1 foramen beneath L5 pedicle will leave the cauda equina 'cord' at L4 vertebral body). From here it descends obliquely in the lateral recess crossing the lower body of L4 and L4-L5 disc and turns outwards beneath the pedicle of L5 before ever coming in vicinity of L5-S1 disc. This topography of nerve root distribution explains that for a given level of disc

herniation the affected nerve root will be the lower nerve root. Hence L4-L5 disc herniation will affect L5 nerve root as the L4 nerve root has already exited before ever coming in vicinity of L4-L5 disc and S1 nerve is still in cauda equina that will separate from it at middle of L5 vertebral body which lies below disc.

Caution – this holds true only for a typical moderate sized posterolateral (lateral recess) disc herniation.

Various factors modify the radicular presentation as below and now should be understood after grasping the above said:

- Size of disc: A large sequestrated disc can affect the nerve against it and any nerve root in vicinity (so in above example L5, S1 and sometimes L4 may be affected)
- Presentation of disc: Axillary disc tend to displace down and medially so a sequestrated fragment can involve the classical nerve root at the level and one level below (in above example L5 and *S1*). Similarly a shoulder presentation can involve in addition nerve root of one level above (but is quite uncommon)
- Far lateral disc herniations (foraminal and extraforaminal) may spare the classical nerve root but involve the exiting nerve (so in above example L4 will be involved).

20. What is cauda equina syndrome?

Ans: As a disc herniation can involve a nerve root so also it can involve cauda equina if the herniation is central! The weakest part of annulus fibrosus is posterolateral and then posterocentral. L4-L5 disc is the usual culprit. Clinical diagnosis rests on several components of central disc herniation viz.:

- Pain radiating along the back of thighs and legs (often symmetrical)
- Numbness in buttocks, back of legs and sole
- Perineal sensory deficit, decreased rectal tone
- Paralysis in L5 and S1 supplied muscles of foot and TFL and gluteus medius

- Loss of tibialis posterior, medial hamstring and ankle (gastrosoleus) reflex.
- Trendelenburg sign and gait
- Bowel or bladder or both incontinence (they travel centrally in cauda equina) with post-void residue of >50-100 ml.
- Babinski's reflex would be mute.
  Such a case is most unlikely as this is a surgical emergency!

21. What will you do for patient?

Ans: After clinically examining the patient I would confirm my diagnosis using radiological and neurological investigations.

22. What radiological investigations would you order?

Ans: I will get an AP and lateral projection X-rays of lumbosacral spine after preparing patient.

23. What will you see on X-ray?

Ans: I will observe the following:
1. Physiological spine curves
2. Reduction of suspected or other disc space ('*vaccum phenomenon*' is quite characteristic for PIVD in conjunction with clinical findings)
3. Auxillary findings of canal stenosis, osteophytes, facet joint arthropathy
4. Osteopenia ('red flag' sign)
5. Spondylolisthesis.

24. What else would you like to do?

Ans: I will get MRI study of L-S spine done. If I find osteopenia or suspected inflammatory process I will order for gadolinium enhanced study.

25. If MRI scan shows a large disc with other discs what will you do?

Ans: The anatomical level should be ideally correlated with neurological level, so I will order electrodiagnostic studies (EMG-NCV) for the same.

26. What do you want to look for in EDS?

Ans: Confirmation of:
- Radiculopathy (whether single level or multiple)
- Evidence of denervation/renervation (partial/complete)
- Associated myopathy if present.

And at last whether this corresponds to the anatomical level determined above. Often in piriformis syndrome where there is entrapment of sciatic nerve in piriformis muscle rather than spine; the clinical tests and radiological investigations are inconclusive and EDS may reveal the etiology.

27. After establishing diagnosis how will you proceed?

Ans: I will discuss the results of investigations with the patient and explain various treatment options then decide accordingly.

28. What is the role of conservative treatment?

Ans: If a symptomatic patient with Fairbank type '2', '3', '4' opts for conservative treatment or the patient is in type '1' category then the following modalities are used in conjunction:
- Rest: Often not indicated for more than 2-3 days. It is just to tide over the acute painful stage and make patient feel comfortable. Advise patient to rest with a pillow beneath knee. Prolonged recumbency potentiate prolonged disability and continued or augmented pain
- Lifestyle changes: tobacco cessation, limited alcohol intake, weight management
- Physical training: toning back muscles and stretching tight muscles and strengthening exercises for weak muscles, Pilates (incorporates Zen meditation and yoga)
- Ultrasound massage, microwave or shortwave diathermy, electrical stimulation (TENS)
- Acupuncture, osteopathic manipulation, magnets, intradiscal electrothermal anuloplasty (IDET)
- Pharmacological treatment: analgesics and muscle relaxants with or without night sedation.

- Adjunctive pharmacological treatment for osteoporosis, facet joint arthropathy (disease modifying agents for osteoarthritis), etc. (TNF-α inhibitors and systemic steroids are *not* approved for this condition!), membrane stabilizers, antidepressants, etc.

29. If a patient does not progress after a fair duration of above treatment but is still not willing for surgical treatment what else can you do?

Ans: I will opt for semiinvasive procedures like epidural injection or chemonucleosis.

30. What is the philosophy and indications of epidural injection?

Ans: Considering altered 'biological' environment and inflammation as an important cause of pain extradural deposition of steroids is a good proposal. The indications are as follows:
 1. Patient with acute signs and symptoms:
    a. Painful SLR/femoral nerve stretch test
    b. Sciatic scoliosis
    c. Appropriate neurological deficit
 2. Patient with acute on chronic attacks with long symptom free intervals
 3. Patient with acute on chronic symptoms with a different level of disc pathology (e.g. after previous surgery).
   Complications could be headache, sciatic pain during treatment, transitory muscle weakness.

31. What is chemonucleosis?

Ans: It is an alternative form of treatment for PIVD as well as formal discectomy/percutaneous nucleotomy. Enzymatic digestion of proteoglycans reduce the water holding capacity of nulceus pulposus which shrinks. Effect takes 4-8 days. Chymopapain (heat labile cysteine protease from carica papaya) or chymodiactin (less heat labile, easier production) is used.

Indications:
1. Single level disc/prolapsed disc
2. Failed response to adequate conservative treatment
3. Classical history and MRI documented disc

Contraindications:
1. Known sensitivity
2. Cauda equina syndrome
3. Pregnancy
4. Previous surgery at the level
5. Sequestrated or free disc fragment
6. Demonstrated spinal/lateral recess stenosis
7. Ongoing litigation.

32. What are the indications of operative treatment?
Ans: *Indications:*
1. Progressing neurological deficit
2. Cauda equina syndrome
3. Severe peripheral neurological deficit viz. foot drop
4. Failure of conservative treatment to relieve pain and neurological signs and symptoms
5. Severe persistent pain and disability for more than 1 year.

Prerequisites:
- Compressive pathology concordant with patients signs and symptoms
- Motivated patient with a strong will to return to work
- No pending/ongoing litigations
- No psychological issues.

33. What are the various options available for surgical removal of disc?
Ans:
- Spinal fusion
- Options (for radiculopathy and nerve root compression – decompression and discectomy):
  1. Standard open discectomy
     a. Laminectomy
     b. Laminotomy
     c. Fenestration

    2. Microdiscectomy (using microscope)
    3. Endoscopic discectomy
    4. Percutaneous automated discectomy.

I will prefer option 1b (for larger disc with displaced nerve root) otherwise 2 (microdiscectomy) due to shorter hospital stay and faster return to work.

– Intervertebral disc replacement.

34. Are there any factors that determine a favorable result? (reverse for unfavorable result)

Ans: Presence of only radicular symptoms positive tension signs, no back pain, non-involvement of workmen compensation act, higher socioeconomic status, minimal psychosocial stressors.

35. What are the signs of complete discectomy?

Ans: There is no absolute answer as to how much disc to remove. Fragments that are loose and easily reached should be removed to minimize recurrence. Aggressive end-plate curettage should be avoided. The goal of the surgery is not complete discectomy but removal of enough material to relieve symptoms and prevent recurrence. After removal of disc, the inflamed nerve root may be erythematous but should move freely and easily with a very gentle retraction. Congestion of epidural vessel is reversed. This is the time to methodically inspect the epidural space medially around the root, anterior to the sac, distally to the level of the inferior pedicle, as well as proximally for other possible fragments.

36. What will you do if there are no further fragments but the root is not free?

Ans: Consider a foraminotomy.

37. What is the usual amount of disc material removed in discectomy?

Ans: Around 5 gm in open discectomy but varies markedly with the level of operation and surgical technique (average 1-7 gm).

38. What are the indications for fusion?

Ans: Posterior fusion can be done after laminectomy or facetectomy during disc removal. The indications are as follows:
1. Associated listhesis
2. Congenital malformation
3. Advanced intervertebral arthrosis
4. Instability due to bone removal
5. Need to return for heavy manual work.

McNab's indications for fusion
1. < 50 year old with normal disc height and root entrapment associated with changes in posterior facet joint necessating joint excision
2. Progressive history of backache associated severe sciatic pain and nerve root irritation where mechanical instability is deemed to be the more important cause
3. Recurrent episodes of low back pain in otherwise emotionally stable patients.

39. What are the various methods for fusion?

Ans: The basic principle for performing fusion is to prevent further segmental motion which is thought to be an important pain generator (posterior spinal segments or disc itself – anterior pain generator). The various methods to achieve this are briefed below
1. Posterolateral intertransverse process fusion ± instrumentation: most common procedure but leaves the disc behind
2. Posterior lumbar interbody fusion (PLIF) ± instrumentation: requires instrumentation as potentially destabilizes the segment for immediate postoperative period
3. Transforaminal lumbar interbody fusion (TLIF) ± instrumentation: approach to disc more lateral than PLIF, near total disc excision so more solid fusion than PLIF
4. Anterior lumbar interbody fusion: anterior approach which is somewhat better tolerated but does not reliably

decompress posterior neural elements and is relatively unstable as it depends only on compressive graft fit

5. Circumferential (anterior and posterior) fusion: combines ALIF and PLIF, but more extensive.

40. What are the causes of failure of open discectomy?

Ans: The following may account for failure:
1. Removal of lamina >25% or predisposes to facet fracture
2. Recurrent/persistent disc herniation
3. Epidural fibrosis
4. Arachnoiditis
5. Lumbar canal stenosis
6. Wrong level and wrong side
7. Emotionally unstable patient/patient requiring workman's compensation.

41. What is percutaneous nucleotomy or percutaneous automated discectomy?

Ans: aka Percutaneous aspiration discectomy.

Indications:
1. Herniation within disc: incapacitating or radicular leg pains made worse by standing or sitting and eased by lying down
2. Failure of conservative treatment for >6 weeks
3. Myelographic/MRI evidence of subannular disc herniation consistent with patient's symptoms and signs
4. At least two of the following:
   a. Weakness of plantar flexors/extensors
   b. Wasting of above muscles
   c. Lost/diminished reflexes
   d. Sensory deficit in specific distribution.

42. What are the complication of discectomy?

Ans: Complications of discectomy:
- Recurrent symptoms/Failed back syndrome: e.g. due to Wrong Level, Recurrent Herniations (same or different level)
- Dural tear, CSF leak: requires resurgery
- Nerve root injury

- Infection (discitis)
- Arachnoiditis
- Epidural fibrosis
- Epidural hematoma
- Cauda equina syndrome
- Iatrogenic Instability

(Additionally one may read the steps of discectomy for a more extensive viva session!)

CASE III:
SCOLIOSIS

Findings

History:
- Insidious onset deformity of spine first noted accidentally
- Gradually progressive deformity
- Painless deformity (there may be associated general weakness)

Examination:
- Features of any of the following:
  – Polio
  – Neurofibromatosis
  – Von Recklinghausen's disease
  – Down's syndrome
  – Marfan's syndrome
  – Hurler's syndrome
  – Meningomyelocele/spina bifida occulta
  – Osteogenesis imperfecta
  – Charcot-Marie-Tooth disease
  – Friedrich's ataxia
- Or none: Idiopathic variety
- Raised shoulder on the convex side in thoracic curves (affected side), on concave side in lumbar curves.
- Scapula rotated outwards and forwards with elevation on convex side
- Thoracic/thoracolumbar/lumbar curve with convexity to right/left side

- Forward protrusion of chest wall on affected
- Increased flank creases on opposite (concave side)
- Higher ASIS and PSIS on concave side
- Adam's anterior bending test: more prominence of structural curve on convex side with hump (rib hump in thoracic and thoracolumbar curves), partial/complete correction of compensatory curves or non-structural scoliosis
- Spinous processes turned into concave side
- Plumb line to judge balance of curve
- Flexibility testing (flexible/Rigid curve):
  – Forward bending
  – Pushing the curve from convex side and noting the correction
  – Lifting the patient up from head
- Measuring the:
  – Distance of plumb line from center (midline)
  – Iliocostal distance reduced on concave side
  – Ilio-occipital distance reduced on the side of decompensation (under correction) – often to the side of convexity
  – Wasting in limbs
  – Limb length inequality
- Neurological examination of limbs
- Limb length measurements
- Hip examination for fixed deformities and a quick look at knee and foot/ankle deformities
- Ober's test
- Gait pattern

(*It is imperative to note the following:*
- *Family history of Charcot-Marie-Tooth disease, Friedrich ataxia, Marfan's syndrome, Neurofibromatosis (they will not tell if you do not ask!)*
- *Age of parents (Down's syndrome)*
- *Growth and developmental history (to judge further curve progression – 'time left'):*
  – *Secondary sexual characteristics*

- – *Menarche*
- – *Milestone achievement*
- *Symptoms for spinal pathology (possible neuromuscular cause):*
  - – *Gait*
  - – *Coordination*
  - – *Bowel and bladder function and specifically late onset bed-wetting.*
- *Midline defects (possible neuromuscular scoliosis):*
  - – *Hairy patch*
  - – *Swelling/mass*
  - – *Port wine patch (Naevi)/Café au lait spots*
  - – *Skin dimples*
- *Congenital malformations (possible congenital scoliosis)*
  - – *Skull deformities*
  - – *Cleft lip/palate*
  - – *Mandibular hypoplasia/small chin*
  - – *Tapering/small ears*
  - – *Malformations of limbs*
  - – *Transverse/longitudinal defects*
- *Always note the non-structural causes of scoliosis – tumor, infection, herniated disc (these often are painful whilst the structural scoliosis only produces mild discomfort and not characteristic pain pattern)*

  This schema should make you answer the following questions quite reasonably.

Diagnosis

The patient is a 12-year-old female with right sided thoracic structural scoliosis with lumbar compensatory scoliosis. There is no clinical evidence of midline spinal defects or neuromuscular affection and the limb lengths are equal. There is no evidence of associated congenital defects.

1. **How do you define scoliosis?**
Ans: Lateral curvature of spine of >10° with associated rotation of vertebrae.

2. What are good prognostic signs in scoliosis?

Ans:
- Male sex (although overall males predominate but females are likely to have large curves)
- Late onset (>10 years) idiopathic scoliosis
- Gradual progression (or static curves)
- Single lumbar/thoracolumbar curve.

3. Why do you call thoracic curve as structural and lumbar curve as compensatory and why not other way around?

Ans: The Adam's forward bending test reveals rotation that is accentuated on forward flexion. The rib hump becomes more prominent. While the lumbar curve partially corrects and does not show any rotational component on forward flexion.

4. What is the basis of Adam's test?

Ans: Technically spine is an asymmetrical cylinder with long anterior and short posterior distance. With forward flexion the anterior distance is 'cramped' and posterior distance 'open' up if the spine is straight. It is imperative to the fact that unless there is advanced anterior wedging of vertebrae which is uncommon, the scoliosis *is actually a lordotic deformity* most prominent at the apex. Forward flexion compresses the lordosis and buckles out the lordosis to side making rotation more prominent.

In a rotational deformity where the anterior part of spine (vertebral body) has rotated away from their anatomical location the 'fixed' deformities would get accentuated by virtue of the technically 'posterior line' now getting compressed and throwing rotated vertebral bodies further into disarray. While the compensatory curves by virtue of being flexible realign to reproduce the normal biomechanics and hence 'reduce'.

5. What are the interpretations of Adam's test?

Ans: Adam's test tells the following:
1. Site of primary curve
2. Flexibility of spine

3. By virtue of accentuation of curve the test also tells us the 'future' of ongoing process in terms of expected outcome.

6. What do you understand by the term 'postural' scoliosis 'compensatory' scoliosis and 'structural' scoliosis 'or' is postural scoliosis same as compensatory scoliosis?

Ans: *"Postural scoliosis"*: A curve that corrects totally on bending forward/lying down/traction or other maneuvers. Clinically there is NO rotation of vertebrae (*this defies the definition of "scoliosis"- however there is supposedly no other better term! please also read 'list in examination'*), there is no structural change.

"Compensatory" scoliosis/curve (secondary curve/minor curve): Curve developed as a compensatory measure to 'structural' or pathological defect elsewhere. There may be rotation but it is NOT FIXED (except otherwise in 'intermediate idiopathic scoliosis' where the compensatory curve also show clinical rotation, this type is fortunately uncommon). The curve and rotation disappears if the primary 'defect' elsewhere is corrected.

"Structural scoliosis": the structural defect lies in the curve which shows FIXED ROTATION of vertebra and spinous process (and ribs). This rotation is persistent on clinical examination and needs intervention to correct.

'Postural and compensatory' are etiological terms and are not technically subset of each other so always choose the correct word!

7. What are the characteristics of a compensatory curve due to limb-length inequality?

Ans: This type of curve begins at lumbosacral junction and is convex to the short side. There is no compensatory curve between the curve and pelvis, this differentiates it from all other curves except congenital forms that may extend to sacrum.

8. **Is it possible to have a step-off "offset" head on thorax which is compensated well over pelvis?**

Ans: Yes, in high thoracic curves with cervical compensation, the head is dragged by cervical spine to one side and appears as an offset but may be well compensated over pelvis.

9. **How do you determine the flexibility of curve?**

Ans: By various distraction and manipulative tests:
1. Forward bending "Adam's test"
2. Lifting the patient up from head
3. Lateral bending
4. Pushing the curve from convex side.

10. **What are the findings that can point the etiology to paralytic curve?**

Ans: The following are the common findings in a paralytic curve (due to PPRP)
1. High thoracic curves (may also be seen in congenital scoliosis, rarely in neurofibromatosis and virtually never in idiopathic scoliosis)
2. Collapse of ribs on convex side in contrast to the often present forward thrusting chest in idiopathic variety
3. Almost certain progression
4. Appearance within one-two years of disease process
5. Muscle charting then reveals other findings (PPRP patient have normal sensory examination).

11. **What are the characteristics of neuromuscular curves?**

Ans: Common features of neuromuscular curves include:
- Large curves early in life
- Stiff curves due to secondary contractures
- Progressive curves that progress even out of rapid growth period
- Long 'C' curves
- Sagittal plane deformity
- Presence of pelvic obliquity

12. Is it necessary for the shoulder to always remain up on the convex side?

Ans: No, in cervicothoracic curves shoulders may remain at the same level. More importantly in idiopathic scoliosis with double thoracic curve the shoulder on the convex side is lower than the other side! This has been called *Signe d'épulae* (the sign of shoulder).

13. What are the interpretations of superficial abdominal reflex?

Ans: An absent reflex may not be abnormal but an asymmetric reflex demands further comprehensive evaluation by advanced radiological investigations to rule out spinal pathologies most prominently syringomyelia.

14. How will you further evaluate the patient?

Ans: I will get radiographs of the whole spine in AP and lateral projections with Lateral AP right and left bending films and 'Hump view' to characterize the curve. Additionally I will obtain the AP view of pelvis and optional radiographs of left hand and wrist to estimate bone age.

15. What information will you get from X-rays?

Ans: The following information can be retrieved:
  1. Etiology:
     a. Congenital – failure of formation (type I deformity) like
        i. Segmented hemivertebra
        ii. Semi-segmented hemivertebra
        iii. Incarcerated hemivertebra
        iv. Non-segmented hemivertebra
     b. Congenital – failure of segmentation (type II deformity) like
        i. Formation of bar (partial failure) – anterior, posterior, lateral, mixed
        ii. Block vertebra (complete failure)
     c. Congenital failure of formation and segmentation (Type III deformity)

d. Paralytic curve: high curves with sloping and collapsed ribs on convex side
e. Neurofibromatosis: *s*hort, *s*harp, *s*tiff (rigid) curves
f. Idiopathic
2. Site of primary curve (regional classification) and classification into types (King's or SRS)
3. Severity (magnitude) of deformity
4. Compensatory curves
5. Rotation (Moe and Nash method etc)
6. Prognostication (RVAD)
7. Compensation
8. Flexibility and amount of correction possible (bending films).

16. What information is obtained from bending films?
Ans:
- Determine the curve type – i.e. structural curves
- Determination of flexibility of curves – by calculation the flexibility index of curves
- Determine the fusion level in lumbar spine
  - The distal extent can be estimated by the flexibility of disc below the chosen distal fusion vertebra
  - Centering the distal planned fusion vertebra over sacrum.

17. What is flexibility index?
Ans: Flexibility index = {Cobb's angle on standard PA film – Cobb's angle on a bend film}/Cobb's angle on standard PA film × 100

18. How do you classify curve?
Ans: Regional classification of primary curves is based on the location of 'Apical vertebra':
- Thoracic curve: 2nd thoracic vertebra to T11-T12 disc at apex
- Thoracolumbar curve: 12th thoracic or 1st lumbar vertebra at apex
- Thoracolumbar – Lumbar main curve: L1-L2 disc to 4th lumbar vertebra at apex

- Cervicothoracic curve: C7 or T1 at the apex.
- Lumbosacral curve: L4-5 disc to S1

Sometimes it is difficult to identify a single vertebra at apex when instead disc is found – then term 'apical disc' is used.

19. What is apical vertebra and what are its characteristics?

Ans: Apical vertebra is the vertebra situated at the apex of curve and its location determines the regional type of curve (see above):
- It is situated farthest from midline
- It is the most rotated vertebra
- It is the least tilted vertebra into the curve
- Lordosis is maximal at the apical vertebra
- It is at the center of primary curve (often but not necessarily).

20. What are end vertebrae?

Ans: These determine the extent of primary curve on AP projection:
- These are the vertebrae 'most tilted' into the curve
- These are the last vertebrae in the curve to show rotation (least rotated vertebrae)
- They are surrounded by disc spaces that are either parallel (neutral disc) or the subsequent disc shows opening into concavity
- The pedicle width is equal on both the sides.

These vertebrae are helpful for measuring the magnitude of deformity and determine the level till which fusion is to be achieved during surgery. Any one of the above findings are sufficient to define an end vertebra.

21. What is then a neutral vertebra?

Ans: This is specifically in relation to a double primary curve where a single vertebra marks the junction of two curves. This vertebra is surrounded by discs that open into the convexity of their respective curves and there is no neutral disc.

22. How do you measure the magnitude of deformity?

Ans: After identifying end vertebrae and apical vertebra, magnitude of scoliosis can be measured by three popular methods:

1. Cobb's method: (simpler and more reproducible of the two) – draw parallel lines from the upper border of upper end vertebra and from lower border of the lower end vertebra and drop perpendiculars from a convenient point of these lines to intersect at a comfortable location. Measure the angle formed at intersection.

2. Ferguson's method: mark the center of end vertebrae and the apical vertebra. Join the centers and measure the angle at intersection.

3. Whittle and Evans protractor method (Oxford Cobbometer): This is principally based on Cobb's method but directly yields the measure and obviates the need to draw lines.

23. What is the criticism of Cobb's method?

Ans: The following are the criticisms against Cobb's method:

1. Firstly (and the one difficult to understand) it is a non-linear expression of the curve size and progressively underestimates the true magnitude of deformity: Understandably, the rotational deformity (3-D) in transverse plane is the true deformity in scoliosis that secondarily produces a lateral curve (*remember the aphorism – 'rotation + lordosis' = lateral curvature*). This means that measurement of rotation and its change will vary linearly with the true change in deformity (*extremely sorry to introduce unavoidable mathematical terms*). Arguably hence measurement of deformity in coronal plane (AP radiograph) will not give 'true' picture. Thus Cobb's angle measurement will not truly determine the corresponding deformity change (hence non-linear) and should not be used as a population measure. This also precludes its expression in "percentage change" of deformity; it will be a logarithmic equation.

2. Interobserver variation of up to 5° has been observed. This is of concern as curve progression by 5° over a given

period above a certain deformity is often taken as a measure of curve progression!

(All above is theoretical, most of the prognostication, classification and management guidelines runs on Cobb's angle measurement. Nothing in prejudice but changing this practice would mean like redefining chemistry with subatomic particles other than proton, neutron and electron!)

24. What are the methods to measure rotation of vertebrae?

Ans: The rotation on A-P projection is noted when the pedicle shows progressive 'ecclipse'.

1. Nash and Moe: measuring pedicle distance from sides of vertebral body, grade '0' when they are equidistant and grade '4' when pedicle is past the center.
2. Perdriolle templates.

25. What is RVAD (Rib-vertebral angle distance)?

Ans: A method to prognosticate idiopathic thoracic curves in infancy described by Mehta. The measurement is done at the apical vertebra – draw lines along the neck of ribs adjoining apical vertebra and measure the angle they subtend with vertical drawn at the center of vertebra. The angles measured on two sides are subtracted and if the difference (RVAD) is >20°; the curve is likely to progress.

26. How do you assess the maturity in terms of bone age?

Ans: Bone age indirectly and roughly determines the time remaining for the curve to progress so that what will be the ultimate outcome. Various methods in terms of popularity are as follows:

1. Risser's sign: divide the iliac apophysis into four equal regions and grading is done according to the extent of ossification from anterior to posterior with riser IV indicating complete capping of apophysis and corresponds to completion of vertebral end plate growth and static curve (Risser '0' = no ossification of apophysis, '5' = fusion of apophysis to ilium). (*Some growth of curve still can occur, riser IV is not a full-stop!*)
2. Triradiate cartilage (open – immature).

3. TW3 and particularly digital skeletal age (DSA) is considered more reliable if at all one would like to assess skeletal age by radiological methods.
4. TW2 (Tanner and Whitehouse second) – comparing the standard film of AP projection of left hand and wrist against the standards referred.
5. TW1: various anthropometric measures
6. Vertebral ring apophysis ossification (Bick et al).
Other measures like chronological age and menarche are unreliable. Height velocity in turn is a somewhat reliable method.

27. How do you classify curves in idiopathic scoliosis?

Ans: King and Moe types for thoracic curves:
1. Type I: lumbar>thoracic (or equal) with lumbar curve less flexible
2. Type II: thoracic>lumbar (or equal) with thoracic curve less flexible and lumbar curve crossing midline
3. Type III: thoracic>lumbar with very flexible lumbar curve that does not cross midline.
4. Type IV: single thoracic curve and L4 tilted into the curve with L5 balanced over pelvis.
5. Type V: double structural thoracic curve.
Coonard expanded King's classification to include lumbar, thoracolumbar and triple curve patterns giving nine types. Lenke et al classification (2001) is more comprehensive adding sagittal alignment to the classification – utilizes three components of spine to produce classification viz. the six curve types, lumbar modifier and thoracic *kyphosis* modifier.

28. How do you look for sagittal alignment and what is its importance?

Ans: On a lateral radiograph drop a plumb line from center of C7 vertebral body. The line normally is anterior to the thoracic curve, posterior curve and passes through the posterior corner of S1 (*sagittal vertebral axis*). SVA is said to be present if the line passes plumb line is anterior to the anterior aspect of S1 and negative SVA exists when it is posterior to anterior body of S1. Generally the lumbar

lordosis is 20-30° more than thoracic kyphosis for maintaining sagittal balance which should be taken care for while correcting the curves.

29. How do you prognosticate and differentiate infantile, juvenile and adolescent idiopathic scoliosis?

Ans:

	Infantile	*Juvenile*	*Adolescent*
Age at presentation (definition)	Up to 3 years	4-9 years	10-20 years
Frequency	Moderate	Infrequent	Commonest form (>50%)
Side dominance	Left thoracic, Left:Right = 2:1	Right: Left = 6:1	Right: Left = 8:1
Associations	Developmental disorders like DDH and congenial deficiencies like heart disease often found	Often none (look for Hypermobile joints)	None Moderate (around ¼th of curves progress)
Risk of curve progression	< 6 month low, > 1 year - high, most of curves <6 month of age resolve (90%)	High (progression during growth spurts)	Quite effective for curves <30°
Bracing	May delay progression of curve but they ultimately progress	Reduce rate of progression but often fail (the early the onset the poorer is outcome)	Posterior fusion ± instrumentation (preferred) < 11 year: add anterior fusion
Surgical management	< 8 year: instrumentation only 8-11 years: anterior + posterior fusion with/without instrumentation >11 year and closed triradiate cartilage: posterior fusion (± instrumentation)	< 8 year: instrumentation only 8-11 years: anterior + posterior fusion with/without instrumentation >11 year and closed triradiate cartilage: posterior fusion (± instrumentation)	

30. How do you manage the patient conservatively?

Ans: Conservative treatment is a supervised treatment planning that is decided on the following points for idiopathic scoliosis:
- Type of bracing
- Duration of bracing
- Curve magnitude and progression
- Skeletal maturity

The following serve as guidelines for bracing:
1. Patient with Risser sign < II with curve progression >5° and magnitude up to 20°.
2. Patient with Risser sign < II with curve magnitude >20°
3. Patient with Risser sign II or more with curve magnitude >30°

The following may be kept on observation:
1. Patient with Risser sign ≥ II and curve magnitude <30°
2. Patient with Risser < II with a non-progressive curve up to 20°

Bracing is done for 20-22 hours in a day. CTLSO (cervicothoracolumbosacral, Milwaukee brace) or TLSO (thoracolumbosacral, Boston or Miami brace) are often used with former preferred for upper thoracic curves. The threshold of surgery for upper thoracic (apex above T7) curves is kept lower.

31. What are the general operative guidelines for idiopathic scoliosis?

Ans: The surgery for scoliosis has evolved over time:
- Classical posterior fusion and POP cast gave way to – Harrington's rod instrumentation which utilized the principle of distraction and compression for correction
- Luque (1970s) then introduced 'segmental' instrumentation utilizing sublaminar wires. This system utilized translation and derotation for achieving correction in contrast to Harrington's instrumentation
- The system of segmental instrumentation was expanded with the introduction of 'hook and rod' constructs (mid 80s)

- This further expanded with introduction of pedicular screws in 1990s. Now pedicular screws for all levels are there that give excellent stabilization early rehabilitation.

What has remained constant over the years is the necessity to obtain a solid fusion of spine irrespective of the instrumentation used.

Indications:

1. Inability to control a curve with bracing
2. Curve >50° (some say >40° for immature spine and >50° for mature spine)

Principles:

- Selective fusion of primary curve is the rule while allowing compensatory curves to spontaneously correct
- Always include the end vertebrae in fusion
- Use anterior procedure in immature patients (infantile and juvenile) to prevent crank shaft phenomenon.

Type I curve: this requires fusion of both thoracic and lumbar curves (T4-L3/4) with multisegment construct using translation and derotation technique for correction. Screw and rod construct can also be effectively used.

Type II curve: fusion of thoracic curve alone with careful derotation at apical vertebra. This is probably done better with the use of all-screw rod construct and translating the spine in addition to rotating the rod. Failing to achieve apical derotation locks the lumbar spine and keeps it from decompensating.

Type III and IV: fuse the primary curve up to end vertebrae.

Type V: both the curves should be fused. There is a tendency of upper curve to correct when major curve is fused. Guidelines by Lenke (upper curve >30° that corrects <20°, grade ≥ I rotation at apical vertebra, T1 tilt into concavity, elevation of shoulder on convex side of upper curve) may help the surgeon to decide necessity of upper curve fusion.

As regards the need of anterior instrumentation (fusion often done from proximal to distal end vertebrae), types II-IV can be effectively managed with anterior procedure

taking special care for preventing future kyphotic collapse by combining posterior procedure in immature spine (see table above). For type I curve combined approach is generally reserved for curves >80°. For lumbar and thoracolumbar curves maintenance of lordosis is an important aspect that can be done by using titanium cage with bone grafts anteriorly and/or rigid rod systems.

Last is the consideration of thoracoplasty to reduce the 'rib hump' that remains often uncorrected after scoliosis surgery. Posterior rib resection by either sectioning medial portion of ribs (the razor ridge) only, or by sectioning and suturing the remaining portion of rib to medial border; both seem to be effective.

32. What is crankshaft phenomenon?

Ans: In cases of immature spine with significant growth potential if only posterior fusion is done then due to continued growth of anterior portion the spine rotates over posterior fused portion as axis (like a crankshaft) and the deformity increases. Risk factors are young age at surgery and significant curve remaining at the time of arthrodesis.

33. How will you manage congenital scoliosis?

Ans: The issue of conservative vs. operative is not as confusing for congenital scoliosis as conservative management hardly has any role. The real issue is regarding what type of surgical procedure to use and obviously when? (*Question should not have been put in answer but this question is very much required to proceed!*)

As opposed to idiopathic scoliosis an early surgical procedure is desirable in congenital scoliosis.

Principles:

• Operate early to prevent structural changes in spine (problem of height gain is immaterial as without intervention there is only abnormal 'width' gain rather than height)

- Surgical means of correction vary with nature of anomaly, location, curve size and flexibility and age of child.

  The following serve as general guidelines:

- Posterior fusion: reserved for small curves with limited remaining growth potential for anterior elements (to avoid crankshaft)
- Combined anterior and posterior fusion: deformity with a large growth potential
- Convex hemiepiphysiodesis: prerequisites –
  - ≤ 6 vertebrae involved
  - Curve <70°
  - Age < 6 years
  - Absence of pathological congenital kyphosis or lordosis
- Hemivertebra excision: reserved for children with unacceptable deformity, fixed lateral translation of curve and hemivertebra located at the apex of curve (safest being lumbar and lumbosacral spine)
- Use of instrumentation: for large curves in children > 5 years. Intraspinal anomalies and rigid curves are contraindication for its use.

34. What are different types of Growing rod used for surgical treatment of Scoliosis?

Ans: There are 3 systems of growing rod: single growing rod, dual growing rods, and the vertical expandable titanium prosthetic rib implant. Each system has its advantages and disadvantages.

   The current expandable spinal implant systems appear more effective in controlling progressive scoliosis, allowing for spinal growth and improving lung development. All have a moderate complication rate, especially rod breakage and hook displacement.

35. What is principle behind Growing rod?

Ans: Most operations addressing spinal deformity in the young child actually arrest growth. This may have unfavorable effects on the growth of the thorax, lungs, and the size

of the trunk. The growing rods theoretically allow for continued but controlled growth of the spine. One or two rods span the curve percutaneously (minimally invasive). The rods attach to the spine at the top and bottom of the curve with hooks or screws. The curve can usually be corrected 50% at the time of the first operation. The child is then followed every six months to have the spine "lengthened"(differential distraction) about one centimeter to allow the child's growth. Often, the rods are kept longer than usual so lengthenings are less invasive than the initial procedure and only involve opening through one incision. Children have to wear a brace to protect the instrumentation. When the spine has grown and correction is acceptable, remove instrumentation and perform a formal spinal fusion operation.

36. What are the complications of growing rod?

Ans: Hook displacement, pedical screw loosening, and rod breakage.

37. What is Shilla procedure?

Ans: The Shilla procedure uses the same ideas as growing rods, but does not require multiple lengthening procedures. The apex of the curve is fused and special screws are used at the top and bottom of the spine. These screws can slide along the rods as the child grows.

38. What is Shilla?

Ans: Shilla is the name of hotel where McCarthy the originator for system was staying and got this idea.

39. What is the role of bracing in congenital scoliosis?

Ans: Practically none. During observation of long flexible curves bracing can be applied but these are uncommon.

40. What are the problems with Bracing?

Ans: Compliance, have to wear until skeletal maturity, stressful for family/patient, lung function affected, compensatory curves can increase, some progress even after wearing brace.

Miscellaneous Short Cases

CHRONIC OSTEOMYELITIS

{Very important and typical case, aims to score candidate's knowledge of infection and approach towards management with respect to bone (*Just making diagnosis will not suffice as it is quite straight forward*).

Read: 3-5 times (MS orth and DNB candidates)}

Diagnosis

The patient is a 31-year-old M/F with 3-years-old chronic osteomyelitis of R/L tibia with deformity (angulation and/or shortening) (with or without pathological fracture). There is associated stiffness of knee joint.

Common Findings

History

• Bone pain, past h/o trauma/surgery/swelling or discharge. History of discharging bone pieces is virtually pathognomonic

Inspection

• Sinus fixed to bone or healed puckered scar
• Exposed necrotic bone
• Soft tissue (contractures)

Palpation

• Bony tenderness
• Fixed sinus/scar
• Deformity
• Irregular thickened bone
• Irregular surface
• Surgical hardware

Nearby joints

Soft tissue

1. Why do you call it as chronic osteomyelitis and how do you define chronic osteomyelitis?

Ans. Characteristic history and examination as given. Also note in history classic 'Walenkamp' phenomenon – classic h/o cyclical pain increasing to severe and deep tense pain subsiding with pus breakage and temporary healing. Chronic osteomyelitis is by definition "bone infection predicated on pre-existing osteonecrosis", and there is no relation of preceding time duration; it is chronic in literal terms that infection persists for long! *Osteonecrosis usually takes around 3 months to establish (and separation of sequestrum from parent bone) hence most texts refer this time duration, however, strictly speaking this may not be acceptable to majority.*

2. Who first coined the term osteomyelitis?

Ans. Nelaton (1834) popularized the use of term "osteomyelitis" probably first instigated by Reynaud in 17th century.

3. How do you differentiate a tubercular sinus from pyogenic sinus?

Ans. The following points help one differentiate the two:
   1. Tubercular sinus has bluish margins and is undermined.
   2. There is serous discharge unless there is superadded infection or extravagant infection.
   3. Anaesthesia or paresthesia in the skin surrounding the sinus.

4. How do you clinically differentiate cellulitis from osteomyelitis?

Ans. Cellulitis has peau d'orange appearance of skin with features of lymphangitis and bleb formation. Osteomyelitis has given (see examination) features.

5. What will you do next?

Ans. Radiograph of involved extremity and adjacent joints in both AP and lateral projections to confirm diagnosis and plan treatment.

6. What do you see on X-ray and does this confirm your diagnosis?

Ans.

- Sequestrum
- Involucrum formation
- Ill-defined bone destruction with areas of remodelling and local irregular cortical thickening
- Loss of corticomedullary differentiation
- Focal cortical defects → cloacae
- Soft tissue scars

   The features confirm my diagnosis of chronic osteomyelitis

7. What is the differential diagnosis for chronic osteomyelitis?

Ans. *Tumour*: Ewing's sarcoma, eosinophilic granulomas, lymphoma, osteoid osteoma, intraosseous ganglion and osteosarcoma

Trauma: Stress fracture

Osteonecrosis: Especially of hip, knee (femoral condyles – with corticosteroid therapy or HIV infection)

Miscellaneous: Fibrous dysplasia, Paget's disease, cellulitis, haemophilia, Gaucher's disease (pseudo-osteitis), Caffey's disease (infants), hypervitaminosis A, chronic cutaneous and subcutaneous infections (deep mycotic infections).

8. What is sequestrum and what is its importance?

Ans. Separated microscopic/macroscopic necrotic fragment of usually cortical bone (which appears radiodense) surrounded by infected granulation tissue and pus (radiolucent) from viable parent bone. (*Parent bone is important as otherwise a bone graft used to treat infected nonunion or to fill Brodie's abscess cavity may also be called Sequestrum!*)

Diagnostic importance: Necessary to make diagnosis of chronic osteomyelitis

Biological importance: It serves as a focus for continuing infection leading to recurrent bouts of acute and chronic osteomyelitis.

(*It takes ≈ 2-3 months for sequestrum to isolate and separate. Sequestrum may be absent in children <12 months of age – whole bone becomes a sequestrum as periosteum is loosely attached, also small sequestra may be absorbed over time*)

9. Why is the sequestrum dense on X-ray?
Ans.

1. Avascular - $\downarrow$ resorption of bone (unable to take part in remodeling)
2. Surrounded by radiolucent granulation tissue and pus enhancing the contrast
3. Hyperemia of surrounding bone due to inflammation making it relatively osteopenic.

10. What are different types of sequestra?
Ans. *According to shape:*

- Pencil like – infants
- Cylindrical/tubular – infants
- Ring – external fixator
- Conical – amputation stump
- Annular – amputation stump
- Coralliform – Perthes

Consistency:

- Coke like – Tuberculosis
- Feathery – Syphilis
- Sand like – tubercular osteomyelitis in metaphysis

Coloured:

- Black – amputation stump and long exposure of necrotic bone to air while also attached to parent bone (formation of ferrous sulphide), fungal infection
- Green – pseudomonal osteomyelitis

Miscellaneous:

- Muscle – Volkmann's ischemic contracture.

11. What are gross characteristics of sequestra and how will you recognize it during surgery?

Ans. Ivory white brittle piece of bone with smooth (pus facing) and rough (granulation tissue) surfaces lying free from parent bone in the cavity. Does not show any punctuate capillary bleeding (paprika sign). Sinks in water and has a dull note to percussion. It has a closed haversian canal on histopathology.

12. What is involucrum and its importance?

Ans. Immature, subperiosteal, reactive, living new bone formation around a dead bone.

Biological importance: It walls off abscess; it surrounds and can merge with parent bone or become perforated with holes 'cloacae' through which pus and granulation tissue may pout or pus discharges.

Treatment related importance: Only if three walls of sequestrum are seen fully on two perpendicular views should sequestrectomy be undertaken otherwise a pathological # will result and it will get converted into infected nonunion.

(*It takes 10 days for new bone to be radiologically visible after subperiosteal deposition. Physiological and pathological variants (D/D of involucrum) include*

a. 50% infants <6 months age $\rightarrow$ B/L symmetrical thin layer along diaphysis of femur, radius, humerus
b. Hypervitaminosis A
c. Metastatic leukaemia
d. Neuroblastoma.

13. What is the common site of osteomyelitis and why?

Ans. Metaphysis:

  i. Hair pin bend arrangement of arterioles (Hobo).
  ii. Relatively less phagocytosis ($\downarrow$ reticuloendothelial system activity, {Hobo 1921})
  iii. Sluggish flow (Treuta)
  iv. Tortuous blood vessels and skimming of bacteria

v. Dead (apoptotic) and degenerating cartilage cells from physeal plate serving as medium for bacterial growth (Duthie and Barker 1955)

vi. Microfractures and local haematoma common in young active children (Morrissy).

14. What are the causes for diaphyseal osteomyelitis?
Ans.

- Long standing osteomyelitis in children (grows out to diaphysis)
- Post-traumatic osteomyelitis
- Implant related osteomyelitis
- Tubercular osteomyelitis
- Drug abusers (Heroin addicts – pseudomonas)
- Immunocompromised (fungal)
- Salmonella osteomyelitis (often bilateral and may be symmetrical).

15. In what forms of osteomyelitis will you not see a periosteal reaction?

Ans. HIV associated osteomyelitis, tubercular osteomyelitis (some cases), long standing resolving osteomyelitis that began in childhood.

16. What is the cause of chronicity of infection?
Ans.

1. Presence of unabsorbed and retained sequestra serving as a constant source of infection
2. Unobliterated cavities (dead spaces alive with bacteria!)
3. 'Microbiological shift' (change of aerobic cocci to gram-negative or anaerobes)
4. Multiple types of bacteria "mixed infection" and anti-microbial resistance.

17. How will you decide management?

Ans. Work-up to confirm osteomyelitis and individualize management and classify host (Cierny Mader classification; See next page)

Blood investigations include hemogram with ESR, LFT, RFT, BS (F and PP), albumin, prealbumin, transferrin (for judging nutritional status of patient)

Advanced imaging for localizing lesion and treatment planning.

Anatomic type

Stage 1: Medullary osteomyelitis (endosteal)

Stage 2: Superficial osteomyelitis (surface only)

Stage 3: Localized osteomyelitis (full thickness cortical involvement and cavitation)

Stage 4: Diffuse osteomyelitis (that are mechanically unstable)

Physiologic class

A host: Healthy

B host:

Bs: Systemic compromise

Bl: Local compromise

Bls: Local and systemic compromise

C host: Treatment worse than the disease

Factors affecting immune surveillance, metabolism and local vascularity

- *Systemic factors (Bs):* Malnutrition, Renal or hepatic failure, diabetes mellitus, chronic hypoxia, immune disease, extremes of age, immunosuppression or immune deficiency
- *Local factors (Bl):* Chronic lymphedema, venous stasis, major vessel compromise, arteritis, extensive scarring, radiation fibrosis, small-vessel disease, neuropathy, tobacco abuse

Young children are included as super A1+++ hosts

As such it is a redundant classification for practical purposes and treatment guidelines based on this classification are hard to find.

18. How will you manage this patient?

Ans. After investigation and work-up with confirmation of diagnosis I will do sequestrectomy and saucerisation of the containing/parent bone *(also see Q 30)*.

19. How will you perform?

Ans. Explain the procedure to patient. Under anaesthesia, position and clean and prep the patient. Methylene blue dye is injected through the sinus tract to clinically mark (live tissue stains grey and dead one blue) the various tracts and culprit sequestrum. No attempt is made to cut through sinus tracts and they are removed *in toto* with careful dissection. There is often a bone defect at the site of pus discharge which if adequate is used to remove the Sequestrum otherwise defect is elongated in oblong fashion with rongeur or multiple drill holes and completing window. This is followed by removal of sequestrum and granulation tissue. The removed material is sent for histopathology, staining, culture and antibiotic sensitivity. Eburnated necrotic bone is removed till healthy bone is encountered taking care not to risk parent bone's strength. In the end an oblong saucer shaped cavity with smoothened edges is aimed for. Thorough inspection is done to search any additional sequestra and lavage is done. Cavity management and wound closure over drain is performed.

20. What are the prerequisites of surgery (sequestrectomy and saucerisation) for chronic osteomyelitis?

Ans. *Absolute:*

Radiological: Well-formed involucrum surrounding the discretely visible sequestrum adequately at least 2/3rd diameter of bone (3 intact walls on two views ensure 3/4th intact walls!).

Clinically: Symptomatic patient with pus discharge or chronic unrelieved disabling pain due to osteomyelitis per se and type A/B host.

Ethical: Salvageable limb and patient willing for prolonged and multiple treatments.

Relative:

Stopping antibiotic treatment at least one week prior to surgery (otherwise cultures will be misleading).

21. **What is *leukergy*?**

Ans. Agglomeration of WBC's in peripheral venous blood seen in association with burns, polycythemis rubra vera, ischemic heart disease etc. Otremisky et al (1993) used it to diagnose and monitor early sepsis.

22. **What is the role of ^{99}Tc bone scintigraphy?**

Ans. It serves as a screening tool only ($\approx$ 10 per cent specificity).

Phase I- arterial (flow) phase

Phase II- venous phase

Phase III- focal bone uptake

Phase I + Phase II $\rightarrow$ +ve with -ve phase III $\rightarrow$ soft tissue infection

All phases +ve $\rightarrow$ true skeletal infection (False +ves – post surgical, implants)

(*Teaching note: Ga^{67} scintigraphy provides the best way to detect vertebral osteomyelitis radiographically, In^{111} labeled leukocyte imaging is the test of choice for osteomyelitis elsewhere in body*).

23. **What is leukocyte imaging (Indium^{111} labeled)?**

Ans. Increased specificity (over Ga^{67} scanning) which can be further increased if used in conjunction with sulphur colloid scans that delineate areas of normal bone activity whereas leukocyte scan highlight the involved regions. 'Incongruence' of In^{111} labeled leukocyte scans and sulphur colloid scans is highly suggestive of infection.

24. **What is the Role of MRI and CT scans?**

Ans. MRI (contrast enhanced) has very high specificity and sensitivity and a negative MRI effectively rules out infection. CT scans may be done to plan surgery.

25. **What is the role of sinogram?**

Ans. Most important investigation to do before surgery. It demonstrates sinus tracts and their source and hence most important guide to surgery.

26. When can we see septic arthritis secondary to osteo-myelitis?

Ans. In infants < 6 months (Trueta) when there are physio-logical connection between epiphyseal and metaphyseal vasculature through physeal plate.

In intra-articular location of metaphysis – proximal humerus, neck of femur, proximal radius, distal fibula in adults.

27. What were the various modalities of treatment in pre-antibiotic era?

Ans. 'Rest and Poultice' method – till discharge of sequestrum occurred spontaneously

Carrel and Dakin – acute clinical sterilization of wounds using Dakin's solution in a closed system.

W. Howes (1874) advocated use of sequestrectomy and granulation of wound.

Winnett Orr – laid the fundamental principles of wide drainage and rest between World War I and II (immobilize fracture in POP in best position → thorough debridement → pack wound with vaseline gauge → keep immobilized and don't disturb wound till granulation and healing → repeat if complications occur)

Use of maggots – Connors used it in American civil war and Baer and Eastman during World War I.

28. What are the causes of knee stiffness in osteomyelitis of femur?

Ans.
1. Quadriceps tethering/sinus
2. Sympathetic effusion
3. Patellofemoral and tibiofemoral adhesions
4. Quadriceps contracture (multiple surgery)
5. Cast immobilisation.
6. Reflex sympathetic dystrophy.

29. What is Klemm's triad?

Ans. Determines the outcome in osteomyelitis:
1. Vitality and stability of bone

2. Virulence and antibiotic sensitivity of bone
3. Condition of soft tissue envelope.

30. Outline the principles for treatment of chronic osteomyelitis?

Ans. Treatment for osteomyelitis is initiated along the following path:

1. Thorough debridement of necrotic tissue and bone: Sinus tract, necrotic bone, infected granulation tissue, sequestra, eburnated bone is removed and saucerisation done for providing wide window.
2. Stabilization of bone
3. Obtaining intra-op cultures
4. Dead space management
5. Soft tissue coverage
6. Limb reconstruction
7. Systemic antibiotic treatment

31. How do you assess adequacy of necrotic bone removal?

Ans. i. Dead bone removed till "Paprika sign": punctuate bleeding from Haversian system → healthy bone (not reliable – tourniquet)
ii. "Laser Doppler" intra-op – cumbersome.

32. What is pulsed lavage?

Ans. Irrigation with 10-14 litres of normal saline using fluid pressure 50-70 pounds/sq. in. and 800 pulses/min. There is no indication for adding antibiotics or emulsifying agents.

33. How do you manage dead spaces following wound excision?

Ans. Healing by secondary intention is discouraged as scar formed is itself relatively avascular and frequently breaks down.

1. Antibiotic beads:
   a. *Non-bio degradable:* PMMA beads impregnated with (gentamycin, vancomycin, tobramycin, clindamycin,

cefazolin). Local concentration of up to 200 times that of systemic are achieved. Short term (10 days), long term up to 80 days and permanent placements are described. Beads need removal as granulation tissue may make removal difficult later. Beads contain 2.4-3.6 gm tobramycin per 40 gm cement and 1-4 gm vancomycin /40 gm cement. Beads are placed from deepest to most superficial. Open wound treatment and suction–irrigation methods are incompatible with this treatment.

b. *Biodegradable beads:* Calcium hydroxyappetite (up to 12 weeks), poly (D,L-lactide) – initial burst followed by sustained release, polyglycolic and poly lactic acid beads

2. Local muscle, myocutaneous flap, free flap, composite flap, etc.
3. Cancellous bone graft (Papineau)

34. What are the disadvantages of antibiotic impregnated beads?

Ans.
- Require second surgery for removal
- Local immune compromise
- Local MIC active only for 2-4 weeks
- Act as substrate for bacteria in long term – glycocalyx formation.

35. What is bead pouch technique?

Ans. Often used now a days as an interim measure between wound excision and definitive skin coverage. The cavity is filled with antibiotic impregnated beads completely and covered by sterile transparent/lucent adhesive covering to prevent secondary wound infection. It has the advantage that no dressings are required during this period.

36. What is glycocalyx?

Ans. It is now applied as a general term to extracellular polysaccharide material and probably glycerol techoic acid

of bacteria aka "the sweet husk of cell". It specifically protects bacteria from phagocytosis, recognition, helps cling to inert implant material and form biofilms that may contain numerous colonies of bacteria safely hidden from host immunity, e.g. *streptococcus epidermidis*. Biofilms stimulate release of PGE_2 from monocyte that inhibits T lymphocyte proliferation, B-lymphocyte blastogenesis and immunoglobulin production; also it interferes with white cell chemotaxis and degranulation. For this reason implants have to be removed for complete eradication of infection.

37. What are Morrey and Peterson's criteria for acute osteomyelitis?

Ans. They determine the likelihood of having osteomyelitis (Acute).

Definite: Pathogen isolated from bone or adjacent soft tissue or there is histologic evidence of osteomyelitis.

Probable: A blood culture is positive in the setting of clinical and radiographic features of osteomyelitis.

Likely: Typical clinical settings and definite radiographic evidence of osteomyelitis are present and there is a response to antibiotic therapy.

38. How do you diagnose acute osteomyelitis?

Ans. Peltola and Vahvanen's criteria (diagnosis requires two of the following four criteria)
1. Purulent material on aspiration of the affected bone
2. Positive findings of bone tissue or blood culture
3. Localized classic physical findings
   a. Bony tenderness
   b. Overlying soft tissue oedema, erythema
4. Positive radiological imaging.

39. What are the differences of osteomyelitis affecting different age groups?

Ans.

Feature	Infancy	Childhood	Adult
Source	Umbilical cord/ hematogenous	Hematogenous	Direct inoculation
Location	Epi-meta-physeal, intra-articular	Metaphyseal	Meta-diaphyseal
Constitutional symptoms	Failure to thrive	Marked symptoms of inflammation	Moderate
Local temperature	Raised little, pseudoparalysis	Raised	Moderate
Adjacent joint	Septic arthritis common	Less frequent	Uncommon, affected in intra-articular metaphysis or long standing infections with pus tracking under periosteum and perichondrium
Periosteum	Perforated by pus	Extensively lifted by pus	Locally lifted by pus
Sequestrum formation	Less	Very frequent and usually one or two	Small and multiple
Chronicity	Less chances	More	May persist for life
Deformity	Shortening	Shortening, angulation, lengthening!	No effect or shortening in bone loss

40. What are the complications of chronic osteomyelitis?

Ans.
1. Recurrences and relapses
2. Limb length discrepancy
3. Pathological fractures
4. Septic arthritis
5. Septicemia
6. Joint stiffness
7. Soft tissue abscess formation and cellulitis
8. Soft tissue contractures
9. Amyloidosis
10. Squamous cell carcinoma of the sinus tract.

41. What is Garre's osteomyelitis?

Ans. This is a chronic form of disease (sclerosing non-suppurative osteomyelitis of Garre) characterized by symmetrical thickening of bone and irregularity but with conspicuous absence of abscess and sequestra (remember Brodie's has abscess but no sequestra). Children and young adults are often affected. Low grade anaerobic infection could be the cause. Guttering the bone or drilling multiple holes may alleviate pain. Should be differentiated from Paget's and Osteoid Osteoma.

42. Is there any classification method that provides estimated rehabilitation time for patient?

Ans. Yes, May's classification:

Type	Description	Rehabilitation time
I	Intact tibia and fibula	6-12 weeks
II	Intact tibia and fibula but	3-6 months
III	Tibial defect ≤ 6 cms long and intact fibula	6-12 months
IV	Tibial defect > 6 cms with intact fibula	12-18 months
V	Tibial defect > 6 cms with unstable fibula	>18 months

NON-UNION, PSEUDOARTHROSIS AND MALUNION

{With the exception of non-union of fracture neck femur and pseudoarthrosis of spine in ankylosing spondylitis (Anderson's lesion) these are given as short cases. Malunions are uncommonly kept for MS exam but may very well be given in DNB exams. Again malunions around hip are given more often as long case, if at all.

Read: 3-5 times (MS and DNB candidates)}

CASE I:
NON-UNION OF LONG BONES

Diagnosis

The patient is a 26-year-old male with nonunion of fracture R/L lower third both bones (if fibula is also involved) leg for one year. There is postero-medial angulation at middle and distal third junction with 2 cms shortening of leg and 20° external rotation of distal fragment. There is restricted dorsiflexion at ankle.

Common Findings

Inspection:
• Scar, discolouration (due to plaster treatment), sinus(es) – infection
• Deformity (angular/rotational)
• Shortening
• Bayoneting of fragments
• Swelling (hypertrophic)
• Wasting

Palpation:
• Temperature
• Tenderness (deep palpation and palpate with nail of your thumb)
• Palpable defect (palpate with nail of thumb running from above down)

- Abnormal mobility (check in two planes)
- Crepitus
- Loss of transmitted movements (rotate the distal part)
- Telescopy and distraction of fragments (lax non-unions and pseudoarthrosis)
- Prominence or absence of other bone (fibula) and its status

Movements: Check for ROM of nearby joints as stiffness due to previous treatment is common.

Neurovascular status distally

Both iliac crests and legs for bone graft.

1. **How do you define non-union, delayed union, slow union, and pseudoarthrosis?**
Ans. *Non-union:* Simply, when a fracture fails to unite permanently of its own. Comprehensively "non-union of fracture is said to exist when the fracture shows clinically, radiologically and biologically no signs for progression of healing after giving adequate time (US FDA considers this as 9 months with failure of progression for 3 consecutive months) for the type and site of fracture which will not unite unless some fundamental alteration in the line of management is undertaken" (quite vague and demands speculation for future events). According to Brinker non-union is a fracture that according to treating physician will not heal unless some intervention is required. This is a permanent end situation.

Delayed union: Fracture is said to have gone into delayed union when healing has not advanced at the average rate for the location and type of fracture. This is a "temporary" phase (fracture still shows progress towards healing) and will progress to permanency of union or sometimes non-union.

Slow union: Here the fracture takes longer than usual to unite but passes through the stages of healing without departure from normal both clinically and radiologically. The ultimate outcome is union. Some people differentiate between slow union as being slow to start with from

delayed union where fracture struggles in later stages of healing.

Pseudoarthrosis: This is a distinct nonunion characterized by hypermobility at the fracture site with cleft formation between ends and lined by pseudo-capsule. The cavity is usually fluid-filled.

2. What are the causes of non-union?

Ans.

Local factors:

Fracture related:

1. Open
2. Infected
3. Comminuted
4. Segmental
5. Fractures of irradiated bone
6. Intra-articular fractures

Treatment related:

1. Inadequate immobilization (most common)
2. Inadequate reduction
3. Inadequate fixation
4. Inadequate blood supply
5. Inadequate soft tissue cover
6. Interposition of soft tissue
7. Inadequate apposition "Distraction"

Systemic:

1. Age (elderly)
2. Mal-nutrition (Albumin< 3.4 g/dl; lymphocyte count <1500/mm^3)
3. Corticosteroid therapy
4. Immunosuppressive treatment
5. Systemic (hepatic, renal) disease
6. Metabolic bone disease
7. Anticoagulants
8. Burns
9. Smoking
10. Alcohol
11. Radiation

3. How do you classify non-union?

Ans.

Infected non-union:

Umiarov's classification

> *Type I:* Normotrophic without shortening
>
> *Type II:* Hypertrophic with shortening
>
> *Type III:* Atrophic with shortening
>
> *Type IV:* Atrophic with bone and soft tissue defect usually with shortening
>
> *May's classification for Tibia* (*see Chapter 9: Case I, Q 42*) (actually it is a classification for osteomyelitis of tibia, however type III, IV, V include non-union)
>
> *Type III:* Tibial defect 6 cm or less with intact fibula
>
> *Type IV:* Tibial defect >6 cm, intact fibula
>
> *Type V:* Defect > 6 cm with no usable fibula.

Non-infected non-union:

Weber and Cech (modified Muller and 'Judet and Judet' classification systems)

Hypervascular non-union:

1. "Elephant foot": Inadequate fixation/immobilisation, premature weight bearing
2. "Horse hoof": Moderately unstable fixation
3. "Oligotrophic": Inadequate apposition, displaced fracture

Avascular non-union:

1. Torsion wedge: Intermediate fragment healed to one main fragment of bone but not to other
2. Comminuted (necrotic): Has one or more necrotic fragments
3. Defect (gap): Loss of intermediate fragment
4. Atrophic: Ends porotic and atrophic usually loss of blood supply

Paley's classification for tibial non-union (clinical and radiological):
- Type A (<1cm bone loss)
  – A1: Mobile deformity
  – A2: Fixed deformity
    • A2-1: Stiff non-union without deformity
    • A2-2: Stiff non-union with fixed deformity
- Type B (>1cm bone loss)
  – B1: Bony defect no shortening
  – B2: Shortening but no defect
  – B3: Both (shortening with defect)

4. What is the basis of Weber and Cech classification?

Ans. Viability of bone ends determined by strontium[85] uptake.

5. How do you clinically determine that this is a non-union and not delayed union or pseudoarthrosis?

Ans. Presence of *painless abnormal mobility* at the site *in two perpendicular planes* is deemed pathognomonic of non-union. Other findings are:
- Crepitus
- Telescopy of fragments (lax non-union)
- Loss of transmitted movements
- Palpable gap or defect in avascular nonunions.

   Patient is unable to bear weight if it is in lower limb. In delayed union there is residual pain at fracture site and tenderness on manipulation of the fracture with abnormal mobility usually in one plane. Abnormal mobility if at all present is not gross. Pseudoarthrosis is characterized by gross abnormal mobility typically in all directions and crepitus is often absent as the ends are covered by fibrocartilage.

6. Is it necessary that a non-union has to be painless?

Ans. No, following are the causes of painful non-union:
1. Infected non-union
2. Interposition of soft tissue especially viable muscle and nerves

3. Non-unions of intra-articular fractures
4. Bayonet non-unions impinging surrounding tissues with adventitial bursa formation and bursitis.

7. How do you clinically differentiate a stiff from a lax non-union?

Ans. Stiff non-unions have arc of motion ≤ 7° (classically < 1 cm defect) that is not adequately bridged by fibrocartilage to resist movements grossly. Often only micromotion can be elicited; even the patient may bear weight partially in such cases. They are also called non-mobile non-unions (or short fibrous non-unions). Lax non-unions (Arc of motion >7°) on the contrary are inadequately supported by connective tissue and demonstrate the classical signs of non-union. These are also called long non-unions or mobile non-unions.

(Intra-articular short fibrous nonunion (fibrous Ankylosis) are often painful and the concept of painless nonunion does not apply to them.)

8. Do all non-unions demonstrate abnormal mobility? (Or can you tell me something about non-mobile non-unions)

Ans. Not necessarily. As above stiff nonunions typically hypertrophic ones may not at all demonstrate abnormal mobility, also some site specific nonunions like in diaphyseal tibial non-union where fibula is intact mobility may be masked at least in mediolateral plane. Some metaphyseal tibial and femoral non-unions also ingeniously mask abnormal mobility only on the sheer basis of proximity to mobile joint – judgment testifies the surgeon's clinical competence. *(In these cases give the clinical diagnosis as malunion if deformity is present but there is no tenderness or if tenderness is present then – uniting fracture when time frame from injury is short or delayed union if time period is long. If there is no deformity/tenderness/abnormal mobility then case will not be kept in exam and better search for something else or you are grossly missing something!)*

9. What are the common sites of occurrence of nonunions?

Ans. Typical sites include – fracture femoral neck, fracture of carpal scaphoid, fracture neck of talus, fracture lateral condyle humerus, fracture capitellum, fractures of distal third shaft of tibia.

10. What are the causes of non-union for fracture shaft of tibia?

Ans. Distal third of tibia is poorly surrounded by muscles so that segmental periosteal blood supply (derived from anterior tibial artery) is limited.

Secondly these fractures are often open and caused by high velocity injury further compromising the blood supply.

Surgical intervention is an added risk factor for jeopardizing the blood supply.

Lastly in middle third fractures of tibial shaft the endosteal blood supply derived from posterior tibial artery that supplies 90 per cent of cortex is most severely affected.

11. How do you clinically identify infected non-union?

Ans. Following are the features of infected non-union: (Infected non-unions fall into Cierney Madar type IV osteomyelitis)

1. Painful non-union (painless if there is significant gap (>1cm) filled with mature fibrous tissue)
2. Raised local temperature
3. Discharging sinus
4. Scar healed by secondary intention with "puckering".
5. Tethered skin
6. Irregularity of bone on either side (representing osteomyelitis)

(*It is not always that you get an active infection with non-union, more often than not it is a quiescent infection. Healing of infection in bone is virtually equivalent to reversing virginity of a girl! So if active infection is not seen then make diagnosis of "infected non-union with quiescent infection" – there is no term like quiescent infected non-union! Similarly healed*)

infected non-union {which means that non-union has healed which was infected} should also not be used ideally speaking. Also healing of infection cannot be clinically "detected". Single plane mobility with tenderness at fracture site and lesser duration of fracture would be clinically diagnosed as "delayed union")

12. What will you do next?

Ans. Radiograph of involved extremity (to confirm diagnosis) with adjacent joints.

13. Does this confirm your diagnosis?

Ans. YES ☺ or No! ☹

Describe the location, type of non-union along with condition of bones and ancillary findings of infection and soft tissue. Comment on joints, status of hardware and deformities.

Delayed union:
• Slight resorption of bone ends with "Wooly" appearance [No e/o sclerosis or very slight].
• Medullary canal is open at both ends.
• Fracture line is clearly visible.
• External and internal callus are minimal.

Nonunion:
• Marked sclerosis of ends with rounding off appearance.
• Medullary canal closed.
• Diffuse osteoporosis of both fragments.
• Fracture gap persists and widened due to unsuccessful bridging.
• Proximal end convex and distal end concave (pseudo-arthrosis).

14. How does infection affect fracture healing?

Ans. Infection is not an etiological factor for nonunion i.e., it does not cause non-union; fractures are known to unite in the presence of infection. However, active ongoing uncontrollable infection is a predisposing factor for non-

union of fracture. Various mechanisms are responsible to this effect:

- Dissection of pus through planes and periosteum – devascularising the ends.
- Fragmentation and dissolution of fracture hematoma.
- Inflammatory mediators that promote fibrous tissue formation.
- If fixation was done then implant failure occurs destabilizing the fragments.
- Increased catabolic response at fracture ends than anabolic activity (this also causes porotic ends).

15. How does non-union develop?

Ans. The inherent tendency of fracture is to heal and to this end there is a fine interplay of innumerable factors that also have a reserve capacity, i.e., some deviations are tolerated but not constant and gross distractions from normal process. Quality of bone, vascularity, protection and stress are some of important mechanical and biological factors that can be modulated to produce positive effect. Response to stress is as follows:

- Intermittent compressive hydrostatic stress (pressure) → Chondrogenesis
- Intermittent strain → Fibrogenesis
- Low levels of mechanical stimuli (micro motion)
  - Good vascularity → Ossification (Osteogenesis)
  - Poor vascularity → Chondrogenesis.

Type of healing also depends on type of stabilization: (Q: What are the various types of callus response with *your* chosen mode of treatment?)

1. Cast (Closed treatment) → Periosteal bridging callus and interfragmentary enchondral ossification
2. Compression plate → Primary cortical healing (cutting cone type)
3. Intramedullary healing → Early – Periosteal bridging callus and late – Medullary callus

4. External fixator → depends on rigidity
   a. Less rigid – periosteal bridging callus
   b. More rigid – primary cortical healing
5. Inadequate immobilisation with
   a. Adequate blood supply → Hypertrophic non-union (failed endochondral ossification)
   b. Poor blood supply → Atrophic (Avascular) non-union
6. Inadequate reduction with displacement at fracture site → Oligotrophic nonunion.

16. What are various types of bone grafts?

Ans. Bone grafts or bone transplants are natural bone tissues obtained to provide either structural stability (cortical) or stimulate osteogenesis (cancellous) or both (usually). (Bone graft substitute are synthetic or semi-synthetic derivatives that have various effects on bonehealing.)

Vascularised: Fibula (free vascularise/double barrel fibula transfer, centralization of fibula, reversed flow vascularised pedicle), iliac crest, greater trochanter, coracoid process (with muscle), rib.

Non-vascular graft:

Cortical: Fibula, tibia, autoclaved diaphyseal grafts.

Cancellous: Iliac crest mulberry grafts, tibial condyles, femoral condyles, olecranon, excised head of femur, posterior superior iliac spine, malleoli.

Cortico-cancellous: Tibial upper metaphysis, tricortical iliac graft.

Mesh cage bone graft technique: Metallic cage (slightly smaller than adjacent bone) filled with cancellous bone chips and demineralised bone matrix.

Grafts may also be classified as autograft, syngraft (isograft – from identical twin), allograft (homograft), xeno(hetero)graft or according to shape, composition and method of application (onlay (single or dual of Boyd)),

inlay, intercalary, peg (neck femur, epiphysiodesis), H-graft, chip, shell, osteoperiosteal, pedicle, sliding and intra-medullary (augments stability, improves screw purchase, added potential for intramedullary healing between host bone and graft, e.g. humeral non-union).

17. What is the mechanism by which grafts enhance healing?

Ans. Biological properties of bone grafts are osteoinduction (process that supports the mitogenesis of undifferentiated mesenchymal cells, leading to the formation of osteo-progenitor cells that form new bone), osteoconduction (property of a matrix that supports the attachment of bone-forming cells for subsequent bone formation) and osteogenesis (combination of above to form bone). Osteogenic property is defined as propensity to generate bone from bone forming cells.

18. How do grafts heal?

Ans. Cancellous (osteoinduction more prominent than osteo-conduction): By a process called "creeping substitution" (Phemister) whereby capillaries invade the tissue followed by granulation tissue and macrophages that resorb the bone while simultaneously also the process of osteogenesis is going on over the scaffold of graft.

Cortical (mainly serves as structural graft, osteo-conduction prominent): Process is somewhat similar but slow resorption occurs along haversian canals and then regeneration occurs. This may take years to complete depending on size and vascularity.

19. What grafting options are available for infected non-union?

Ans.

1. Papineau open bone grafting (Three stage technique): (Rhinelander, Higs, Roy-Camille, etc also described similar techniques)
   a. Debridement (usually multiple at 5-7 days interval) with intramedullary stabilization

b. Grafting: Once granulation tissue appears use "match-stick" cancellous grafts 3-6 cms × 3 mm × 4mm in overlapping circular fashion with or without saline irrigation and wound packing.

c. Skin cover: Either spontaneous or various flaps/graft for coverage

2. Mini-Papineau
3. Postero-lateral bone grafting of tibia (Harmon's method, especially if anterior skin is unhealthy)
4. Phemister bone grafting
5. Friedlander technique – thorough debridement + stabilization and closure followed by open bone grafting
6. Ilizarov method with or without bone grafting
7. Huntington's two-stage tibialization of fibula for infected gap nonunion (quiescent infection).

20. What is phemister bone grafting and modified phemister grafting?

Ans. DB Phemister (1947!) devised a technique for treating delayed unions and established non-union of tibia (even infected ones) by placing "sub-periosteal" "cortical" bone grafts (onlay) across fracture site. The fracture site was not opened. Modification (Charnley and Forbes) included placing "cortico-cancellous" grafts "subcortically" by raising "osteoperiosteal" flaps. Charnley additionally used "Shingling" (raising osteoperiosteal flaps of bone longitudinally across fracture site) originally devised by Naughton Dunn (1939). Later, process of "Petalling" ('fish-scaling' originally described by Uhtoff) was also combined to above; Forbes actually analyzed the results of Charnley method and also used petalling in combination. Petalling has the effect of creating artificial ensheathing callus at ends which is connected by process of shingling.
Philosophy of Phemister grafting:

• Fibrous union should not be broken down by refreshing or resection as it disturbs the ongoing healing process and increases instability.

• Nonunion heals if induced to do so

- Rigid immobilisation is unnecessary
- Can be used in presence of recent sepsis provided graft is inserted through normal tissues away from fracture site.

21. What are the various modalities of enhancing fracture healing?

Ans.

1. Autogenous (vascular/non-vascularised) Bone grafting
2. Bone marrow injection (app. 20 ml of marrow harvested from iliac crest or greater trochanter for nonunions < 5mm)
3. Stem cell injection (100-150 ml bone marrow centrifuged to produce 2-4 ml aspirate containing multipotent marrow cells and osteogenic stromal cells)
4. Platelet rich plasma and related peripheral blood concentrates
5. Electrical: What can be modulated?
   a. Piezoelectric effect of Yasuda
   b. Bioelectric/steady state potentials (Friedenberg and Brighton)
   How?
   1. Constant direct current stimulation (invasive percu-taneous electrodes) – stimulates inflammatory like response – stage I of fracture healing
   2. "Capacity coupled generators" (alternating current) – affects cAMP; collagen synthesis and calcification during repair
   3. PEMF (pulsed electro magnetic field, time varying inductive coupling) – calcification of fibrocartilage (not fibrous tissue)
6. Ultrasound
7. External shockwave treatment
8. Bone morphogenic proteins
   a. BMP-2 (rhBMP-2; 1.5 mg/kg in collagen sponge)
   b. BMP-7 (rhBMP-7 aka OP-1{osteogenic protein – 1})
   c. BMP-14 (GDF-5/MP-52)

22. What are bone graft substitutes?

Ans.

Osteoinductive
- Allograft bone
- Demineralized bone matrix (contains Type-I collagen, non-collagen protein and osteoinductive growth factors like TGF- superfamily, BMP, GDF)
- Purified human BMP
- OP-1 device
- INFUSE (rhBMP-2/ACS{absorbable collagen sponge})

Osteoconductive
- $CaPO_4$ – available as powder, ceramics (coralline, synthetic, etc. prepared by *sintering* (heating to temp. >1000ºC) cements and composite
- $CaSO_4$
- Allograft (stored at (–60ºC); tested for HIV, HBV, HCV, Syphilis- India; additionally for HTLV-I and II and CMV-US, sterilized by γ-irradiation/Ethylene oxide) Hydroxyapatite
- Bioactive glass (beads of silica, calcium oxide, disodium oxide and pyrophosphate)

Osteogenic and osteopromotive
- Selective cellular retention (Cellect)
- Bone marrow aspirate injection/implantation
- Platelet-rich plasma and blood concentrates
- Stem cells.

23. What is distraction osteogenesis?

Ans. Actually it is distraction histogenesis. This is based on the philosophy of tissue generation under tension– "A living tissue (capable of regenerating) when put under steady traction can be lengthened to any extent in the line of tension vector by virtue of increased metabolism and vascularity". For bones the principles of compression and distraction osteogenesis both apply. Additionally, Ilizarov method can be used to its best by "accordion maneuver" and "trampoline effect" to enhance union.

- Small segment non-unions (short stiff non-unions): can be crushed by pressure effect in compression and then growth of fibrocartilage ensues which slowly ossifies due to trampoline effect if patient is bearing weight.
- Diseased or malunited segments can be removed for relatively longer nonunions and if the resulting gap <4 cms then acute docking in compression followed by lengthening can be done.
- For long and atrophic nonunions where the gap will be >4 cms; its better to hold out the extremity to its length and do bone transport (internal/external/combined, as there is risk of kinking neurovascular bundle) in various modes:
    – Monofocal
    – Bifocal
    – Trifocal

24. What is corticotomy?

Ans. Best defined as "an open, subperiosteal, low-energy partial osteotomy of bone cortex, followed by manual osteoclasis of the remainder of cortical circumference maximally preserving the periosteum, endosteum and bone marrow with its blood supply as well as the muscle and soft tissue surrounding the bone". The term "compactotomy" is specifically used for diaphyseal corticotomy. Torsional corticotomy usually requires "external rotation" to prevent stretching radial and peroneal nerves!

25. What are various types of corticotomy?
Ans.
1. *Transverse:* Lengthening, correction of deformity, bone transport
2. *Longitudinal:* To widen bone (overcoming defect in one of two bones improve shape of a thin atrophic limb
3. *Splinter:* Splints off a piece of bone with attached periosteum, soft tissue and skin – bridging non-union, eliminate partial bony defects
4. *S-shaped corticotomy:* Chronic osteomyelitis

5. Complete
6. *Partial:* To correct bow in bones, e.g. osteogenesis imperfecta.

26. Where in bone will you do corticotomy?

Ans. Should be done at the CORA (apex of deformity) if done to correct deformities. For lengthening and other purposes metaphyseal location is best suited:

- Regenerate is of best quality
- The nutrient artery has already branched and it is easier to preserve the same
- Lastly the origins and insertions of muscles are adapted for extremity growth (that takes place at ends of bone!) – so lengthening at ends of bone is more physiological.

    CORA basically is a locus (Center Of Rotation of Angulation) from where all deformities in that plane can be addressed. Co-existence of various types of deformities (e.g. angular, translational, rotational, etc) at various sites makes assessment of CORA more difficult and then multiple CORAs exist. For uniplanar angular deformity correction of deformity along the bisector of deformity (CORA) corrects the deformity completely. Calculation of CORA is a bit complicated but it should be sufficient to remember that there are two methods for doing so. First one is trigonometric which is more accurate; the other one is graphical method which is more of approximation.

27. What is the physiological effect of distraction osteogenesis "or" tell us some non-orthopaedic uses of distraction osteogenesis?

Ans. There is increase in blood supply of limb by 330 per cent so it can be used for treatment of peripheral vascular disease and trophic ulcer.

28. What are complications of corticotomy?

Ans. Damage to vascularity, displacement after corticotomy, incomplete corticotomy, premature consolidation.

29. How do you assess regenerate?

Ans. Radiologically:

1. No defects/shark bite lesions on 3 sides (if present – accordion maneuver)
2. Complete ossification of radiolucent central growth zone
3. Uniform radiographic density that is half way between density of adjacent bones.

30. How do you reduce pin site infection?

Ans. Proper care

Proper tension

Using special pins:

- Titanium pins (900 per cent less chances of infection, steel pins inhibit respiratory burst of granulocytes)
- HA (hydroxyapatite) coated pins
- Silver coated pins (antimicrobial action of silver ions).

31. What is the rate of distraction and when do you start it?

Ans. Distraction at corticotomy site is done at the rate of 1mm/day in equally divided intervals usually four (each 0.25 mm). Ideal will be to use motorized continuous motion distracters. Rate of distraction can be increased (up to 2 mm/day) in children or oblique corticotomy whereas it should be reduced in older age groups/ Diaphyseal corticotomy/ poor bone quality. Distraction is started (latency) usually on 4th or 5th day former one being favoured for children, oblique corticotomy. Waiting for an additional day or two (≈7days) may be required for high energy, diaphyseal, comminuted corticotomy or accidental osteotomy, osteopenic/sclerotic bone and fragments rotated >30° for rotational osteoclasis.

32. What are the various types of bone transport?

Ans.

1. *External bone transport:* Combined bone loss replacement (up to 5-7 cms) with correction of deformities and limb lengthening (can be monofocal {single corticotomy} or

bifocal {dual site corticotomy and movement in opposite directions towards each other})

2. *Internal bone transport using olive/hooked wires:* For defects 7-10 cms and larger
3. *Combined bone transport:* For larger defects due to major bone loss combined with limb deformities, deep soft tissue scars and local blood supply insufficiency.

33. What are advantages and disadvantages of your technique?

Ans. External bone transport is easier and can simultaneously correct deformity and shortening but produces more skin scarring and is inadequate for major bone loss. Internal bone transport is better for larger bone defects and easier for patient as fewer wires are involved but it is difficult to apply and does not have enough "compression effect" at docking site.

34. For fracture of legs, which has better prognosis as regards union – a fracture of both bones or fracture of tibia only?

Ans. Fracture of both bones as non-fractured fibula quite often produces distraction at fracture tibia and prevents apposition of displaced fracture tibia. Even in simple fractures of both bones, fibula may unite early and become load sharing reducing axial loading of tibia. Partial fibulectomy may allow closer apposition of tibia and with weight bearing union could be enhanced.

35. How do you decide the treatment for non-unions?

Ans. Treatment options are decided on the basis of type of non-union, location (epi/meta/diaphysis, age, bone quality, functional demands, movements at nearby joints, secondary changes in soft tissue, infection, prior treatments and presence of deformity and shortening. Most hypervascular non-unions will heal if adequately stabilized in optimum position combined with bone

grafting. Sometimes biological enhancements (bone marrow, stem cells, electrical, etc) may alone be used if fracture is acceptably aligned. Deformity correction may be done acutely with open methods if deformity is not grotesque (<4 cm shortening; <10-15° angulation). For larger deformities gradual correction would be a better option. Infected non-unions require specialized modalities (see above). Avascular non-unions often require diligent measures and planning for achieving union along with adjuncts.

(*You should be a part surgeon, part detective, and part historian. History has a way of repeating itself, without a clear understanding and appreciation of why previous treatments have failed; the learning 'curve' becomes a 'circle')*

36. How much shortening is acceptable for treating nonunions of various bones?

Ans. Shaft humerus: 4-5 cms

Both bones forearm: up to 4 cms (proximal third of radius and distal 5-8 cms of ulna are dispensable and can be sacrificed for defects in these regions). Ideology is that elbow joint is primarily formed by proximal ulna and wrist joint by distal radius – this holds true for creating single bone forearm in complicated lesions.

Lower limb bones: Less than 3 cms (up to 1.5 inch of deformity can be hidden without producing ankle equinus)

37. How do you treat epiphyseal (intraarticular) non-unions?

Ans. These are usually oligotrophic type. Inter-fragmentary screw (e.g. cannulated lag screw) with arthroscopic intra-articular setting of bone and arthrolysis if required is most recommended. This may be followed by neutralization plating.

38. What is SCONE?

Ans. Slow Compression Over Nail Using External Fixator.

39. What is dynamisation?

Ans. Supporting efforts at healing by providing or producing micromotion at fracture site – generally recommended for delayed unions.

Nails: Remove the screw that is farthest from non-union (advantage – minimally invasive with immediate return to weight bearing, disadvantage – axial and rotational instability)

External fixator: This is both therapeutic (axial loading → further bony union) and diagnostic (↑pain at fracture site suggests fracture has still not united). Achieved by removal/loosening/exchange/farther shifting of external struts spanning nonunion.

40. What principles underlie enhancing healing by exchange nailing?

Ans. Improves local mechanical environment (two ways) and local biological environment (two ways).

Mechanical advantage:
1. Larger diameter nail (usually 2-4 mm larger) – stronger and more stiff construct
2. Widening and lengthening of isthmic portion of medullary canal - ↑stability due to ↑cortical contact area

Biological advantage:
1. Reamings – local bone graft
2. Reaming - ↓ medullary blood flow → dramatic ↑in periosteal blood flow and periosteal bone formation

Exchange nailing is applicable for both viable and non-viable nonunion. Preferred if circumferential bone loss <30 per cent (failure rate high if defect >2 cms and/or >50 per cent circumferential bone loss)

(*Additional information – "ultrasound adjuncts" have not been found useful for nonunions with intramedullary nail in situ*)

41. What is the role of synostosis and amputation for managing nonunions?

Ans. Synostosis entails creation of bony continuity between paired bones above and below nonunion site functionally creating one bone extremity, e.g. single bone forearm.

Amputation; least preferred and is indicated for:

1. Frail, elderly unfit patient with infected nonunion
2. Unreconstructable neurologic function which precludes restoration of purposeful limb function
3. Patient wishes to discontinue medical and surgical treatment
4. When ultimately all viable reconstructive methods have failed and left over plans will produce less satisfactory function than with amputation
5. When reconstruction is impossible!

42. What are various types of callus response?

Ans. Callus is like sex – it's natural, it unites two ends, and it requires a bit of movement.

Primary callus response:

• Begins to form after two weeks of injury
• Derived from *cambium layer* of periosteum and exuberant *external callus* beneath the intact periosteum
• Spread from fracture end
• Undergoes involution and **does not** cause bone union
• Independent of environmental and hormonal influences

External bridging callus:

• Under the control of hormonal and mechanical factors
• Paracrine hormonal influences principally determine the blood supply
• Inhibited by rigid fixation
• Seen in cast immobilization and interlocking nails

Late medullary callus:

• Seen in combination with external bridging callus
• Dependent on medullary vascularity and independent of environment
• Not inhibited by rigid fixation

- Seen with plate fixation

  (*Teaching note: Source of osteoprogenitor cells*:
  1. *DOPC: Previously determined osteopro-genitor cells – Present in the inner layer of periosteum (cambium and marrow)*
  2. *IOPC: Inducible osteoprogenitor cells derived from undifferentiated soft tissue cells*)

CASE II:
PSEUDOARTHROSIS OF TIBIA

Diagnosis

The patient is a 6-year-old M/F with anterolateral angulation of left tibia at lower third middle third junction for 4 years. There is 1 cm true shortening of leg and wasting of 2 cms over calf muscles and patient is walking with short limb gait. There is associated right sided scoliosis of dorsal spine and café-au-lait spots over trunk.

Common Findings

Inspection:
- Anterolateral angulation of leg
- Foot deformity (type VI)
- Ankle valgus curly toes
- Signs of neurofibromatosis
- Deformities in other limbs (fibrous dysplasia)

Palpation:
- Thinning of tibia at angulation
- Tenderness
- Abnormal mobility (pathological fracture)
- Fibula and its status
- Wasting
- Constriction bands
- Contracted and prominent tendo Achilles

Associated Findings

- Other limb deformities
- Previous surgical procedures, etc.

1. What is your etiological diagnosis?

Ans. I would like to give differential diagnosis of congenital pseudoarthrosis of tibia with neurofibromatosis (see below), congenital longitudinal deficiency of tibia (paraxial tibial hemimelia), congenital longitudinal fibular deficiency (paraxial fibular hemimelia), fibrous dysplasia, nonunion of tibia (if previous surgical procedures have been done and trauma +), Rickets, post osteomyelitic pathological fracture, fibrous dysplasia.

2. What would you do to confirm your diagnosis?

Ans. I would get X-rays of leg with knee and ankle joints in A-P and lateral projections.

3. What do you see on X-rays?

Ans. There is a pathology involving L/3rd and M/3rd junction of tibia with area of sclerosis/lucency (cyst formation). There is thinning of tibia or hour glass constriction and fibula is also dysplastic. (You may find frank nonunion at the site). This suggests the diagnosis of congenital pseudo-arthrosis of tibia (first described by Hatzoecher 1708, Barbar associated with Neurofibromatosis; incidence = 0.005 per cent, 1:190000 live births).

4. When you do not see pseudoarthrosis of tibia then why do you call it congenital pseudoarthrosis of tibia?

Ans. Congenital pseudoarthrosis of tibia is a misnomer it is often not a congenital one neither is there a primary or true pseudoarthrosis. The nonunion (pseudoarthrosis) develops usually after birth due to congenital 'defect' in tibia. The defect lies in bone, periosteum, surrounding tissue and possibly nerve or vascular supply (likened to hamartoma) weakening the bone.

5. What are the diseases most commonly associated with congenital pseudoarthrosis of tibia?

Ans. Neurofibromatosis and fibrous dysplasia.

6. How do you look for pseudoarthrosis and what is its impact on congenital pseudoarthrosis of tibia?

Ans. Crawford criteria for neurofibromatosis (At least two of the following):
1. Multiple café-au-lait spots (>5 mm and at least 5)
2. Positive family history
3. Definitive biopsy
4. Characteristic bony lesions such as congenital pseudo-arthrosis of tibia; short sharply angulated, stiff (3S) scoliosis of spine.

   Fibroma molluscum (subcutaneous nodules) are not present until adolescence but they are typical of chronic disease

   The neurofibromatosis does not have any effect on the type of treatment or prognosis of disease.

7. How do you differentiate between skin lesions of neurofibromatosis and those of fibrous dysplasia?

Ans. The café-au-lait spots of congenital pseudoarthrosis of tibia have smooth edge (coast of California) however, in fibrous dysplasia the lesions have appearance of coast of Maine (irregular edge).

8. How do you classify congenital pseudoarthrosis of tibia?

Ans. Various classifications; Crawford, Boyd's, Anderson system

Boyd	Description	Anderson equivalent
I	Patients born with anterior bowing and tibial defect	
II	Anterior bowing + hourglass contracture (# by 2 years) associated with neurofibromatosis (worse prognosis)	Dysplastic type

Contd...

Contd...

Boyd	Description	Anderson equivalent
III	Bone cyst	Cystic type
IV	Sclerotic segment of tibia (no narrowing), usually develops stress type fracture – nonunion	Late/Sclerotic type
V	Also have dysplastic fibula	Fibular type
VI	Intraosseous neurofibroma/ Schwannoma (Rarest type) Foot deformity (CTEV/Streeter's band associated)	Clubfoot/ congenital band type

9. What is conservative treatment for congenital pseudo-arthrosis of tibia?

Ans. Done for patients without fracture or pseudoarthrosis (pre-pseudoarthrosis). Total contact plastic clamshell orthosis, AFO (prior to walking), KAFO (infant starting to walk). Worn indefinitely full-time; if fracture does not occur tibial bowing usually gradually improves and reformation of medullary canal often requires up to 5-10 years. If there is sufficient straightening of tibia, medullary canal is reformed and adequate cortical thickness then orthosis may be discontinued. Explain to parents that this is not a permanent remedy and may be of little use; ultimately surgery including amputation may be needed.

10. What are goals of surgical treatment and what are various modalities available?

Ans. Goals:
- Union at fracture site
- Maintaining union
- Obtaining acceptable limb length
  Treatment options:
1. McFarland type posterior bypass graft from opposite tibia (this is prophylactic grafting done in imminent fracture cases, inadequate bone structure, child whose activity cannot be controlled!)

2. EyreBrook's delayed bone grafting (older the patient with presentation greater are the chances of success)
3. Boyd's dual onlay cortical bone grafting with bicortical screw fixation (bone of parents/allograft): Early success but much ultimate failure.
4. Farmar's cross-legged vascularised pedicle bone grafting from opposite tibia : Problematic as it is cumbersome and potential for infecting uninvolved tibia
5. Sofield miller's double proximal and distal osteotomy followed by reversal of tibial shaft with intramedullary nailing: Reversing healthy bone to involved site may stimulate osteogenesis
6. Intramedullary nailing with iliac crest bone graft (Charnley)
7. Intramedullary bone graft with vascularised fibula and iliac crest: Transfixes ankle and subtalar joint (retrograde insertion of rod)
8. Microvascular fibular graft (contralateral), rib, iliac crest with excision of tibial pseudoarthrosis
9. Electric current stimulation of osteogenesis
10. Ilizarov method: Compression, compression + tibial legthening, compression followed by distraction, distraction alone for hypertrophic nonunion
11. Amputation (McCarthy); Boyd/Symes type to produce end bearing stump:
    a. Failed three surgical attempts
    b. Shortening >5cms
    c. Deformed foot
    d. Prolonged hospitalization
    e. Pseudoarthrosis <2.5 cms from ankle joint.

11. What are the complications of treatment?
Ans.
- Refracture
- Limb length discrepancy
- Ankle and subtalar stiffness
- Progressive anterior angulation
- Ankle valgus
- Donor site morbidity.

AMPUTATION STUMP

{Simple case, having good chances to be put up in DNB exam. Very commonly performed surgery so practical aspects must not be of concern. Some theoretical aspects of course need polishing.

Read: 2-3 times (DNB candidates), MS Orth may get a short question instead!}

Diagnosis

The patient is a 37-year-old male with healed, mature below knee amputation stump of right leg done 15 cms from knee joint line with skewed flaps. The patient is using below knee prosthesis for mobilization for last 3 years.

If you know the reason for doing amputation –include in diagnosis.

Findings

- Conical amputation stump covered with soft tissue (muscle and skin)
- No protruding or impinging bony ends
- Healed incision line without sinus/discharge/ excoriation/ erythema/ulcers/keloid formation
- There are no callosities
- Vascularity of the stump is fair.

1. How do you define amputation?
Ans. Amputation is a procedure where a body part is removed through one or more bones. (cf – disarticulation where it is removed from joint)

2. What are the indications for doing amputation?
Ans. Amputation is philosophically required for:
   1. Dead: Complete irreversible loss of vascular supply to limb

2. Dying: E.g. PVD, Tumour, burns.
3. Deadly (Dangerous): necrotizing fasciitis, gas gangrene, etc
4. Damn nuisance (Damn useless limb or Damn painful limb): Trauma/chronic osteomyelitis, nerve injury, trophic ulceration/ neuropathic disorder, congenital, complete irreparable brachial plexus injury, etc.

3. What are the goals of amputation?
Ans.
- Ablation of diseased tissue
- Reconstruction
- Optimize patient function and reduce morbidity (Palliation)
- Provide a physiological end organ.

4. Why do you call flaps skewed?
Ans. The scar line is anterior from midline in coronal plane having long posterior flap.

5. What are various methods of configuring of flaps?
Ans. *Principles:*
- Use defined flaps in elective amputation with apex of flaps just distal to planned level of bony resection
- Use any available flaps in trauma aiming to preserve length
- Tailor flaps at least as long as diameter of the stump
- Thick flaps without undermining/undue dissection
- Flaps should not adhere to bone
- No dog ears/unnecessary folds.

Options:
- Equal anterior and posterior flaps: Suits most amputations.
- Equal medial and lateral flaps (Scandinavian flaps – especially for Peripheral Vascular Disease (PVD))
- Long posterior flap (Skewed flap) – commonly done in B/K amputation and PVD.

6. **What is the ideal length for amputation stumps at various places?**

Ans. Amputation level is not primarily decided by ideal length at least now with the advent of modern prosthesis. The most distal level that will heal without complications and provide a functional stump should be chosen.
- A/K amputation:
  - 12 cms from medial joint line or 18 cms below greater trochanter tip.
- B/K amputation:
  - Ideal level is at musculotendinous junction of gastrocnemius. Ideal length would be 15 cms from medial knee joint line (stumps < 12 cms are less efficient and <6 cms do not function as B/K stump); minimum working length is 9 cms
  - Rule of thumb – allow 2.5 cms (1 inch) for every 30 cms (12 inch) height otherwise preoperatively flex the knee at right angle and if the residual tibia extends at least three finger breadth beyond insertion of medial hamstring tendons then patient can be fitted for below-knee prosthesis.
- A/E amputation:
  - 20 cms from acromion
- B/E amputation:
  - 18 cms from tip of olecranon

7. **How do you determine adequacy of vascularity of flaps?**

Ans. *Clinically:* Skin temperature, level of dependent rubor, feel pulses (the level till pulses can be felt will support the skin adequately even distally some 5-8 cms can survive by collateral circulation)

Investigations:
- Doppler: ?inaccurate in elderly patients (calcified vessels) – Ankle brachial pressure index >0.45 provided 90% chances of healing
- Toe systolic B.P. – 55 mmHg is minimum required for distal healing

- Transcutaneous PO_2 minimum 35 mm Hg for healing
- Arteriogram or digital subtraction angiography
- Skin blood flow (Xe^{133} clearance)/thermography/ Thallium scanning, etc largely research tools
- Intraoperatively adequate bleeding form flaps and oozing from muscles (remember to deflate tourniquet).

8. How will you assess the patient for amputation (Or what precautions will you take before proceeding with amputation)?

Ans.

- Tissue perfusion as above
- *Immune competence and nutritional status of patient for adequate healing:* Serum albumin at least 3 gm/dl, Hemoglobin level at least 10 gm/dl, lymphocyte count > 1500/ml
- *Systemic:* Control diabetes, optimize Renal, cardiac and liver functions.
- *Psychological:* Early plan for return to func-tion, pre-operative counseling, rehabilitation assessment
- *Pre-operative pain control:* Coordination with experts in pain management.

9. What intra-operative precautions will you take for tailoring an ideal stump?

Ans.

- Planning flaps as mentioned.
- Muscles Divide some 5 cm distal to level of bone resection, stabilise muscle mass to prevent sensation of instability during prosthetic fitting and ambulation
- *Nerves:* Divide cleanly with sharp knife after gentle traction and allow to retract at least 1 inch. Large nerves should be ligated for they contain vessels, e.g. sciatic nerve
- *Vessels:* Large arteries and veins should be doubly ligated to secure hemostasis. In general haematoma

formation should be prevented by meticulous hemostasis.

- *Bone:* Plan a level as this is often the reference point (apart from flaps). No sharp ends or margins, bevel the end to give desired shape and should not impinge or come in direct contact (and adhere) with skin flaps.
- *Closure:* There should be no tension at margins, interrupted monofilament sutures to give good scar.
- *Drains:* If infected or haematoma expected.
- *Dressing:* Compression soft dressing or moulding dressing reinforced by POP cast/slab.
- Prevent contractures!

10. What do you mean by muscle stabilization?

Ans. Muscles must be so secured that they do not wiggle under the effect of prosthetic fitting during mobilization as it gives a sensation of constant discomfort.

Methods :

- *Myodesis (preferred):* Here the muscle or tendon is attached to bone by drill holes. Should not be done for ischemic limb (↑chances of wound breakdown). It effectively counterbalances the antagonists, provides good strength and minimizes atrophy, maximizing residual limb function, prevents contractures.
- *Myoplasty:* Here the muscle is sutured to periosteum/ fascia of opposing group (antagonists) for counterbalancing action.

11. How will you prevent contractures?

Ans. The following are the various methods to prevent contractures:

- Make the patient lie prone.
- Muscle setting exercises
- Myodesis
- Early mobilization and pain control (prevents spasm)
- Immediate post-operative prosthetic fitting.

12. What are the complications of amputation surgery?

Ans.

Early

- Wound haematoma
- Flap breakdown, infection (especially in diabetics), clostridial infection secondary to perineal contamination
- Joint contracture
- Wound pain, phantom sensation, phantom pain

Late

- Joint contracture, instability
- Pain due to pressure of ill fitting socket, phantom pain, neuroma
- Stump oedema due to venoconstriction, unstable - too much soft tissue left failure to perform myodesis
- Skin verrucous hyperplasia, skin maceration, fungal infection / intertrigo, blisters, abrasion, atrophy, callosities, follicular hyperkeratosis, sycosis barbae, allergic reactions to material of cup or liner
- Bone spur formation - due to periosteal bone formation - avoid periosteal stripping osteoporosis, fracture
- Cosmesis sitting asymmetry, bulbous stump e.g. symes in females, severely scarred stump

13. Why do you prefer distal amputation?

Ans. More acceptable, ethically preserves as much normal tissue as possible, less energy expenditure and higher compliance.

Energy expenditure depends up on:

- Length of residual limb (amputation level), unilateral/ bilateral
- Reason for amputation
- Aerobic capacity and cardiopulmonary efficiency, patient age and fitness
- Speed of gait - walking speed decreases with more prox amputation, gait symmetry
- Weight of prosthesis and weight concentration

Long BKA	10 per cent +	beyond baseline
Medium BKA	25 per cent +	(energy
Short BKA	40 per cent +	consumption)
Average AKA	65 per cent +	
Hip disarticulation	100 per cent +	

14. What is the timing of prosthetic fitting?
Ans.
- Immediate (immediate post-operative prosthesis {IPOP}) *(See Chapter 10: Prothetics and Orthotics Q12)*
- Prompt (7-10 days): With evidence of stump healing ("Preparatory prosthesis")
- Early (≈ 3 weeks) after the stump has healed
- Late – after the stump has fully matured – less chances of wound breakdown.

   Stump modulation occurs for up to 6 months post operatively; therefore, the patient should be assessed regularly. Usually a new socket has to be given after 2-3 months. The patient should be explained thoroughly that repeated visits in the early period are essential and normal!

15. What do you understand by pre-prosthetic care?
Ans. Postoperative/preprosthetic care – this phase includes the time period during which the amputation stump undergoes maturation.

   Goals of preprosthetic care:
- Promotes wound healing
- Reduces post surgical edema
- Molding of residual limb
- Preventing flexion contractures
- Maintain muscle strength in the affected limb
- Maintain muscle strength in the unaffected limb
- Maintain body symmetry.

16. What is pylon prosthesis?
Ans. Pylon or endoskeletal (means – structure of "prosthesis", do not confuse with osteointegrated prosthesis) prosthesis

with adjustments began to appear in 1960 for use as temporary limbs. Ultimate concept being an adjustable endoskeletal structure that could be carried into definitive prosthesis, the pylon being covered with resilient foam shaped to match the contralateral leg. Basic technique involves fitting plaster socket on operating table that incorporates an aluminum fitting to which pylon can be attached bearing an artificial foot. Patient is made to touch down the prosthesis within 24 hrs and can bear weight in ensuing days. The benefits are as follows (not all are accepted by community without controversy at large):

1. Control of post-operative edema
2. Reduction of post-operative pain
3. Improved wound healing
4. Early gait training and walking
5. Reduced hospital stay
6. More rapid stump maturation
7. Earlier fitting of definitive prosthesis
8. Psychological benefit to patient "waking up with arm/ leg"
9. Prevents contractures
10. More frequent sparing of knee joint when amputation is done for PVD

Difficulties:

1. Cast loosening with edema subsidence
2. Pistoning effect can increase edema and wound breakdown
3. Heaviness of cast, impedes walking
4. Muscle atrophy.

17. What are the advantages of through knee amputation?
Ans. Advantages:

1. Large end bearing surfaces of distal femur are preserved
2. Long lever arm controlled by strong muscles is created
3. The prosthesis used on the stump is stable.

18. **What is *inactive residual extremity syndrome* and how do you manage it?**

Ans. Some patients in spite of being treated with well performed amputation experience persistent residual extremity pain, swelling, a sense of instability and inability to tolerate extended prosthetic ambulation. Extremity hence under-goes atrophy and becomes a passive participant in mobilization. This symptom complex is known as inactive residual extremity syndrome. The *Ertl osteomyoplastic lower extremity amputation reconstruction* is described to treat such patients for trans tibial and trans femoral amputations.

Technique: Osteoperiosteal flaps are raised from end stump of bone and bone further resected → the osteoperiosteal flaps are closed over medullary canal sealing it off (osteoplasty) → superimposed myoplasty attaching antagonizing muscle groups is performed → skin is contoured to underlying myoplasty.

19. **What is phantom limb sensation and phantom limb pain?**

Ans. Phantom limb sensation is the feeling that amputated limb is still present sometimes in contorted positions. In early days the sensation is often accompanied with pain that can be managed with analgesics. It is later replaced by intermittent paresthesia and still further by mere awareness of absent limb. Some patients experience *"jacitation"* – distressing phantom limb pain with involuntary jerking of stump. This is resistant to treatment and is more likely to be present in patients with chronic pain and sepsis before amputation. Cause is not known but "trigger points" may be identified in some; other proposed mechanisms include absence of normal afferent barrage (C-fiber atrophy) especially at night, hyper-sensitive axonal sprouts from cut nerve ends, changes in spinal cord physiology (altered peptide concentrations, etc.), extrapolation by brain of limb location.

EXAMINATION OF SWELLING

{Examination of a bony swelling is not only interesting but a vast topic, viva of which can proceed in any direction sometimes to the weirdest of extents. Only the most basic aspects of it are presented here that are a must for candidate to learn and without which only an exceptional examiner would forgive. The details of various tumour types are better learnt from standard textbook(s)

Read times: 2-4 for MS and DNB candidates; better judge it from your capability.

Diagnosis

The patient is a 17-year-old male with solitary, non-tender benign looking swelling over upper leg. The swelling is fixed to tibia, around 6 × 4 cms, spherical, bony hard, smooth, non-lobulated with defined edges. The swelling is non-fluctuant, non-compressible or reducible, non-pulsatile and does not transilluminate. The overlying skin (and muscle) is not fixed (*rather* tethered) to the swelling.

Look for solitary/multiple lesions, type of bone (long/flat), site affected (metaphysis/diaphysis/epiphysis)

1. **Why do you call it a benign swelling?**
Ans. {*Brief history suggests that swelling appeared insidiously <u>followed</u> by pain and that the swelling has progressed from the size of a lemon to pear in last 6 years*}.

   On inspection:
   • The skin is not stretched or shiny smooth.
   • There are no dilated veins.
   • There is no discolouration.
   • There are no secondary skin changes (inflammation, ulcer formation or fungation, loss of skin appendages)

   On palpation:
   • Local temperature not raised.
   • Non-tender swelling

- Well-defined edge (margins are for ulcer!)
- Smooth surface
- Not-tethered to skin or surrounding soft tissue
- No pain on bearing weight or movements
- No evidence of pathological fracture (*not a very consistent finding for differentiating a benign from a malignant swelling – better drop it!*)

2. How do you determine that the swelling is fixed to or emanating from bone?

Ans. The swelling is continuous with bone when I palpate the edges of the swelling. There is no intervening tissue between bone and swelling. The swelling congruently moves with the movements of bone. The swelling arises from beneath the muscles which overlie it.

3. What else would you like to examine?

Ans.
- Sessile or pedunculated
- Deformity in the bone secondary to the swelling
- ROM of nearby joints
- Lymphadenopathy.

4. How will you differentiate that the swelling is below the muscle or above it?

Ans. Ask the patient to tense the muscles overlying the swelling by appropriate resistive maneuver, e.g. pressing the knee down for an anteriorly located lower thigh swelling. Observe the following:
- Movement of the swelling in the direction of muscle fibers and perpendicular to it
- Prominence of swelling

If the swelling becomes more prominent on tensing the muscle it indicates that the swelling overlies the muscle otherwise disappearance of swelling suggests that it is deep to the muscles. A swelling arising from the muscle itself shows variable change depending upon where it lies within muscle fibers and the support for swelling increases

if it lies in the superficial fibers making it more prominent and vice versa. Now here the assessment of mobility takes an important role.

The mobility of a swelling that overlies the muscle is not changed while that of a swelling arising from muscle is lost when movement is attempted in the direction of fibers or perpendicular to a taught muscle. (*Swelling that underlies the muscle often disappears unless it is large and bony or stony hard!*)

5. How do you determine if a swelling is soft/ firm/hard on clinical examination?

Ans. A *stony hard* swelling is not indentable offering some friction or a 'gritty feeling' to the examining hand. *Bony or woody hard* swelling is also not indentable but is smooth or lobulated. *Rubbery swelling* is hard to firm or firm like touching the nose tip. A swelling that is soft and squashable offering some resilience is *'spongy'.* Swelling feeling like your ear lobe (spongy and squashable) is a *'soft'* swelling.

Bony swelling may have some typical surface characteristics like 'crackling egg-shell consistency' of a giant cell tumour (GCT), variable consistency (firm on surface to bony hard at pedicle) for an osteochondroma. Swelling may be indentable like a 'ping pong' ball in aneurysmal bone cyst (ABC).

6. What is your differential diagnosis?

Ans. Benign bone tumours like osteochondroma, osteoma, giant cell tumor, osteoid osteoma, Chondromyxoid fibroma, osteoblastoma, ABC.

{Learn and remember the tumor distribution as per Diaphyseal location, metaphyseal location, epiphyseal location, which may be asked for enumeration – it is always better to develop your own list! A brief list is presented here}:

- *Epiphyseal lesions:* Chondroblastoma, chondrosarcoma, giant cell tumour
- *Metaphyseal lesions:* Any lesion!

- *Diaphyseal lesions:* Ewing's sarcoma, osteoblastoma, lymphoma, adamantinoma, fibrous dysplasia, eosinophilic granuloma
- *Flat bones:* Ewing's sarcoma, metastasis, myeloma, chondrosarcoma
- *Vertebral body:* Giant cell tumour, metastasis, Ewing's sarcoma, eosinophilic granuloma
- *Posterior elements of vertebra:* Aneurysmal bone cyst, osteoid osteoma, osteoblastoma.

7. How do you stage benign bony swellings?

Ans. In Enneking classification the benign bone tumors are classified according to activity of the tumour:
- *Latent:* Remains static or heals spontaneously like non-ossifying fibroma
- *Active:* Progressive growth limited by natural barriers like ABC
- *Aggressive:* Progressive growth, invasive, not limited by natural barriers like GCT.

8. What is Enneking classification of bone tumours?

Ans. Enneking classified the malignant and benign bone tumours.

Enneking classification of malignant bone tumors is based on a combination of parameters (grade of tumor (G1 – low grade, G2 – high grade), compartment involved (T) and metastasis (M)).

Compartment is defined as a natural barrier to tumour extension like bone, fascia, synovial tissue, periosteum or cartilage. The tumour may be intracompartmental (T1) or extracompartmental (T2).

Stage	Grade	Site	Metastasis
IA	G1	T1	M0
IB	G1	T2	M0
IIA	G2	T1	M0
IIB	G2	T2	M0
III	G1/G2	T1/T2	M1

Stage I is a low grade tumour while II is high grade one. Sub-stage 'B' means an extracompartmental tumor and stage III is a metastatic tumour.

AJCC (American Joint committee Classification) for staging of bone sarcomas is more comprehensive as follows:

Stage	Grade	Primary tumour	Metastasis in regional lymph nodes	Distant metastasis
IA	G1/G2	T1	N0	M0
IB	G1/G2	T2	N0	M0
IIA	G3/G4	T1	N0	M0
IIB	G3/G4	T2	N0	M0
III	NOT DEFINED!			
IVA	Any G	Any T	N1	M0
IVB	Any G	Any T	Any N	M1

G1 – well differentiated, G2 – moderately differentiated, G3 – poorly differentiated, G4 – undifferentiated. T1 – tumour within cortex; T2 – tumour outside the cortex. N0 – no regional lymph node metastasis, N1 – regional lymph node metastasis

9. What is the stage of tumour when it presents with an extraosseous component?
Ans. Often stage IIB

10. What will you do next?
Ans. I will get an X-ray of the involved region in at least two perpendicular planes.

11. What will you see on an X-Ray?
Ans. Specifically I will look for parent bone and changes (site, extent, cortical breach, etc.), tumour mass, destruction pattern, periosteal reaction and tumour mineralization patterns (osteoid, chondroid, reactive bone mineralization).

12. What are the various types of periosteal reactions?

Ans. Takes 10 days to three weeks to appear:

- *Type I:* Solid periosteal reaction
  - *Shell formation:* Simultaneous bone removal of bone from endosteal surface with surface deposition of bone. Thickness depends on the balance between the two
  - *Smooth shells:* Associated with benign tumours
  - *Lobulated shells:* Due to focal variation in growth rate
  - *Ridged shell:* Trabeculated, septated or soap-bevel reaction. This is due to uneven destruction. Seen in non-ossifying fibroma, GCT, ABC, enchondroma and some malignant tumours like chondro/fibrosarcoma, plasmacytoma and metastasis from thyroid, melanoma, renal.

- *Type II:* Continuous periosteal reaction with cortical persistence
  - *Solid periosteal reaction:* Multiple layers of bone deposition due to chronic stimulation of bone by a relatively slow growing bony lesion, e.g. osteoid osteoma, sub-acute osteomyelitis. It is referred to as cortical thickening or hyperostosis.
  - *Single lamellar reaction:* Single layer of new bone in the form of a uniform radiodense line considered hallmark of benign process, e.g. histiocytosis X, osteoid osteoma, fractures!
    - Dense undulating periosteal reactions are a variant seen with low-grade osteomyelitis, osteoarthropathy associated with paraneoplastic lesions or varicose veins, periosteitis
  - *Lamellated reactions:* Seen with active bone-destroying lesions like acute osteomyelitis, Ewing's sarcoma and osteosarcoma
  - *Parallel speculated reaction:* (Hair-on-end appearance) seen with more rapidly destroying malignant process due to bone formation along the radial neovascularisation

- *Type III:* Interrupted periosteal reactions seen with aggressive tumours reaching sub-periosteal location after cortical breach:
  - *Buttress:* Solid bone formation at cortical margins of a slowly but constantly growing lesion
  - *Codman angle:* Represents the buttress lesion in an aggressive neoplasm. First described by Ribbert was elaborated upon by Codman in 1926. It can be seen in any lesion that aggressively lifts the periosteum like acute osteomyelitis, ABC, osteoma, osteosarcoma, chondrosarcoma. They result from subperiosteal bleeding and bone formation and are themselves free from tumour!
- *Type IV:* Complex periosteal reactions
  - *Divergent speculated pattern aka sunburst pattern:* Combination of reactive bone formation and malignant bone formation, it contains areas of reactive and sarcoma bone with intervening areas of cellular tumour and tumour products. It is highly suggestive of osteosarcoma but can also be seen in osteoblastic active metastasis and haemangioma.

13. What are the various types of bone destructions?

Ans. Bone destruction has to be analyzed on the following grounds before grouping them:

1. Pattern of bone destruction and configuration of marginal interface
2. Cortical breach
3. Presence of sclerotic rim
4. Expansion of parent bone

Type I: Geographic bone destruction:
A. Geographic lesion with sclerotic margin: Seen in non-ossifying fibroma, unicameral bone cyst, chondroblastoma, fibrous dysplasia
B. Geographic lesions without sclerotic margins: GCT
C. Geographic lesions with ill-defined margins: Seen in GCT, fibrosarcoma, and chondrosarcoma. Aggressive

benign neoplasms such as enchondroma, chondroblastoma, desmoplastic fibroma.

Type II: Moth-eaten destruction (regional invasion): Multiple scattered holes that vary in size interspersed with apparently preserved bone. This pattern is seen in Ewing's sarcoma, primary lymphoma, chondrosarcoma, fibrosarcoma and osteosarcoma, sometimes acute osteomyelitis.

Type III: Permeative destruction: Poorly demarcated with indistinct margins. Can be seen in numerous destructive processes not limited only to neoplastic (round cell tumours, angiosarcoma, high grade chondrosarcoma, etc.), mechanical, inflammatory (acute osteomyelitis) and metabolic lesions of bone.

14. What else would you like to do and why?

Ans. I would get detailed radiological investigations to determine and plan management:

- Bone scan:
  - Intramedullary extent of tumour – classically the bone was cut 6 cms proximal to the lesion demarcated by scintigraphy
  - Polyostotic disease
  - Metastasis
  - Biological activity of tumour
- CT scan: Bony extent and margins of tumour and cortical breach.
- MRI:
  - Medullary extent
  - Soft-tissue component and extent
  - Involvement of vital structures
- Angiography:
  - Tumour blood supply
  - Embolisation to reduce intra-operative bleeding
  - Relationship of major vessels to tumour
  - Feasibility of intra-arterial chemotherapy.

15. Which bone tumour metastasizes to bones?

Ans. Osteosarcoma

16. What will you do for this patient?

Ans. Management planning is done only after characterization of the tumour that includes doing a bone biopsy before proceeding any further.

17. What are the principles of bone biopsy?

Ans.

1. Should be done at the conclusion of staging (*to avoid radiological artifacts*)
2. Avoid sampling error, take multiple samples from the lesion (this is necessary for sarcomas but single specimens may suffice for carcinomas)
3. The biopsy tract should be incorporated into the planned surgical incision
4. Biopsy should be done from the representative tissue
5. Biopsy tract should be the shortest way to tumour
6. Should not violate more than one compartment
7. Should not be done from intermuscular planes
8. Should be remote from the main neurovascular bundle
9. Try not to violate the cortex and take sample from the extracortical bone tissue (to prevent weakening the bone by creating a stress riser defect)
10. Bone window should not have any sharp corners (avoiding stress risers) – prefer an oblong window
11. Avoid transverse incisions (prefer longitudinal incisions)
12. Do a sharp dissection, close with a sub-cuticular stitch
13. Obtain meticulous hemostasis (to avoid haematoma formation); a drain if absolutely needed must be placed in the line of incision, or through the wound
14. For an incisional biopsy take tumour tissue, reactive tissue, pseudo-capsule, capsule
15. Biopsy should be ideally done by the surgeon going to finally operate the patient.

18. What are the various types of biopsy?

Ans.

1. FNAC (not very useful for sarcomas)
2. Core/Trephine biopsy using a 14-gauge needle (usually recommended for most bone tumours)
3. Incisional biopsy (for failed core biopsy, vascular bone tumours, ABC)
4. Excisional biopsy (usually done for small lesions <2-3 ms in longest diameter).

19. What surgery will you do for this patient and what are the various advantages and disadvantages of the resection procedures?

Ans. Four types of excision based on the relationship of dissection plane to the tumour and pseudocapsule.

1. *Intralesional excision:* Performed within the tumour mass and removes only a portion of tissue, the pseudocapsule and macroscopic tumour are often left behind
2. *Marginal excision:* Dissection plane passes through the pseudocapsule, may leave microscopic tissue behind
3. *Wide (en-bloc) excision:* Removal of tumour tissue, pseudo-capsule, and a cuff of normal tissue (some > 1 cms) peripheral to the tumour tissue in all directions. This is often the desired procedure for a sarcoma but the normal tissue is a matter of controversy
4. *Radical excision:* Removal of tumour and entire containing compartment, taking care of skip metastasis

   1, 2 done for benign lesions or for palliative procedures in metastases. 3, 4 for curative malignant lesion. Amputation can be 2, 3, and 4 depending upon the plane through which it passes.

20. What is the difference between a satellite lesion and a skip lesion?

Ans.

"Skip lesions" are present in the same anatomical compartment; they are 'not' in continuity with the main tumour mass that forms outside the pseudocapsule.

"Satellite lesions", in turn, are formed within the pseudocapsule and are in continuity with the main tumour mass.

21. What is three-strike rule?

Ans. The four key components are:
1. Bone
2. Nerves
3. Vessels
4. Soft tissue envelop
   If any of the three key components are involved then limb salvage is not worth considering 'three-strike rule'.

22. What are the types of chemotherapy?

Ans.
- *Adjuvant chemotherapy:* Given after surgical procedure for control of residual tumour and metastasis or micro-metastasis.
- *Neo-adjuvant chemotherapy:* Given before the surgical procedure and after it ('Sandwich technique') often for a limb salvage procedure. It is aimed to:
  – Chemotherapeutic drugs start acting immediately helping control micrometastasis
  – Reduce the stage of tumour (at least 90 per cent kill is desired, if < 90 per cent then change agents – *there is no evidence of increased survival in those that do not respond to initial/ deemed most effective agents*)
  – Determine the response of tumour to chemotherapy (sort of 'sensitivity testing' for drugs)
  – Reduce mass and vascularity of tumour
  – Buy time for surgical intervention and prosthetic design
- Definitive chemotherapy: For tumours that are highly chemosensitive (Ewing's sarcoma)
- Palliative: For tumours that cannot be resected.

23. What is the role of radiotherapy?

Ans. Adjuvant radiotherapy had been largely replaced with chemotherapy. Use of mega-voltage and orthovoltage radiation to control micrometastasis and tumour volume

reduction has now but immensely helped the limb salvage proce-dures. It can be given in neoadjuvant or adjuvant modes. Radiotherapy acts by rupture of chemical bonds of complex molecules, generation of highly reactive free radicals and hydrolysis, direct DNA damage and strand breakage and lastly to damage to vascularity which is quite prominent in sarcomas. The damage is more pronounced in rapidly dividing cells (high turnover) but success is dependent on the rate of recovery of surrounding normal tissue.

Dosage is measured in terms of Rad (radiation absorbed dose) which is a measure of the energy imparted to the matter by ionizing radiation per unit mass (1 Rad – 0.01j/kg). Grays (Gr) = 1 joule of energy absorbed by a mass of 1 kg tissue (= 100 Rad). Radiation is given in daily fractionated doses resulting in repair of normal tissues but exponential kill of tumour tissue. Multisite radiation (multiaxial radiation) protects the normal tissue from excessive radiation but the total energy delivered to the tumour is high often in the form of 'limit movement' (large amount of energy delivered in packets over few seconds). Tumours that are amenable to radio-therapy are few and include Ewing's sarcoma, osteo-sarcoma, myeloma.

24. What is the most common soft tissue tumor in children?
Ans: Hemangioma.

25. What is the most common soft tissue tumor in adults?
Ans: Lipoma.

26. What is the most common malignant soft tissue tumor in children and adults?
Ans: It is rhabdomyosarcoma for children and malignant fibrous histiocytoma for adults.

27. What is the most common primary benign and malignant bone tumor?
Ans: Osteochondroma and osteosarcoma.

28. What is the most common bone tumor of hand?
Ans: Enchondroma.

29. What is the most common secondary benign bone tumor?
Ans: Aneurysmal bone cyst.

30. What is the most common secondary malignancy of bone?
Ans: In the order – malignant fibrous histiocytoma, osteosarcoma and fibrosarcoma.

31. What is the most common sarcoma of foot and ankle?
Ans: Synovial sarcoma.

32. What is the most common sarcoma of hand?
Ans: Epithelioid sarcoma.

33. What are the common anterior spinal tumors?
Ans: Giant cell tumor, hemangioma, eosinophilic granuloma, metastasis, chordoma, multiple myeloma.

34. What tumors do you find in posterior spinal elements?
Ans: Aneurysmal bone cyst, osteoid osteoma, osteoblastoma.

35. Can you tell characteristic immunostains for some tumors?
Ans: Lymphoma (+CD20), Ewings sarcoma (+CD99), Chordoma (Keratin, S100), Adamantinoma (Keratin).

36. Is genetics associated with bone tumors or they are developmental?
Ans: Yes, it's only a fact of matter that we have not identified all! Some common ones are:
   • Ewing's sarcoma t(11:22)(q24:q12)
   • Synovial sarcoma t(X:18).

37. What are the various osteosarcoma variants?
Ans: 11 variants (first three are *forms of osteosarcoma*):
   • *Osteosarcoma of jaw*

- *Multicentric osteosarcoma*
- *Secondary osteosarcoma*

Variants of primary osteosarcoma:

1. Conventional
2. Low grade intramedullary
3. Low grade superficial (parosteal and periosteal)
4. High grade intramedullary
5. High grade superficial
6. Small-cell variant
7. Chondroblastic variant
8. Giant cell rich osteosarcoma
9. Osteoblastic
10. Fibroblastic
11. Telangiectatic.

VOLKMANN'S ISCHAEMIC CONTRACTURE AND COMPARTMENT SYNDROME

{Often considered as a difficult case needing specialized review and guidance. The case is moderately important and is a good choice for examiners. Diagnosis is often clear and emphasis is on examination and identifying other lesions (nerve injuries) and approach to treatment.

Read: 4-6 times (MS Orth and DNB candidates)}

Diagnosis

The patient is a 14-year-old male with post-traumatic *(if etiology known then mention it viz. supracondylar fracture humerus)* moderate VIC of left forearm of 7 months duration with median nerve involvement.

Findings

- Wrist flexion
- Volkmann's sign
- Pronated forearm, Wasting
- Flexed elbow
- Cord- like induration on the flexor side, extensors affected/ spared

- MP joints flexed/extended and IP joints flexed/extended
- Paraesthesia or anaesthesia in the hand and fingers
- Flexed and adducted thumb
- Claw hand
- Deformity and trophic changes due to ulnar and median nerve involvement.

1. Why do you call it Volkmann's ischemic contracture (VIC)?

Ans. History of trauma followed by swelling and functional loss. Slowly developing contracture. Wasting and fibrosis of muscles, involvement of skin with scar (contracted). Typical posture of hand and attitude of limb. Volkmann's sign positive and other findings as above.

2. What is Volkmann sign and what is its significance?

Ans. Inability to *actively* extend fingers (at IP and/or MCP joints) without flexing wrist "and" *passive* extension of fingers possible only with wrist flexion (or conversely wrist flexion with passive finger extension). It differentiates the deformity due to nerve palsy and those due to intrinsic muscle contracture of long flexors. Also it can differentiate between intrinsic minus hand from long flexor contracture.

{*One may wonder why the Volkmann sign is not described in a supposedly simpler way – "finger flexion with passive wrist extension", the explanation is – try on yourself and you will appreciate that it is a normal finding! – the tenodesis effect*}

3. What is your differential diagnosis?

Ans. Cases are usually very evident but some milder versions may be confused with:

- Post traumatic haematoma and resulting contracture
- Osteomyelitis and muscle involvement either by intervention or disease process
- Haemangioma of forearm muscles
- Pseudo-VIC (tethering of muscle often FDP to healing fracture).
- Burns.

4. Who described VIC?

Ans. Richard Von Volkmann of Halle, Germany (1881) resulting from tight bandage for injured extremity.

5. How will you test FDS contracture in isolation?

Ans. Flex FDP to fullest to relax it completely then stretch FDS.

6. What is Seddon's ellipsoid?

Ans. Seddon described ischemic zone of injury usually following brachial artery injury that acquires ellipsoid shape which is in general different from conical ischemic zones observed in lung and liver ischemia. The long axis of the ellipse runs usually around anterior interosseous artery with center just above mid-forearm. Nerve at the center is the one most affected in VIC; which is often median nerve whereas ulnar nerve often is at periphery and is variably involved. FDP and FPL lying on either side of vessel are the most severely affected muscles. Necrotic muscle is colloquially termed "muscle sequestrum".

7. How do you classify VIC?

Ans.

> *Seddon:*
> *Grade I:* Ischaemia
> *Grade II:* Ischaemic contracture
> *Grade III:* Ischaemic contracture with nerve involvement
>
> *Zancolli:*
> *Type I:* Contracture involving forearm muscles with normal intrinsic muscles
> *Type II:* Contracture involving forearm muscles with paralysis of intrinsic muscles
> *Type III:* Contracture involving forearm muscles with contracture of the intrinsic muscles
> *Type IV:* Combined type

Tsuge (Types):

Mild (aka localized VIC): Further subdivided into proximal (pronator teres chiefly involved) or distal (pronator quadratus) or middle third (flexor digitorum profundus) involvement. No or mild nerve involvement, e.g. ulnar nerve in proximal type. Often presents with involvement of RF/MF but in advanced cases there may also be involvement of IF/LF.

Moderate: Deep muscles (FDP, FPL) + superficial muscles (FDS, wrist flexors, pronator teres) + involvement of thumb and nerve involvement.

Severe: Moderate + extensors (often partial) and skin involvement + severe contractures and joint stiffness.

8. Why is median nerve more commonly involved?

Ans. Median nerve is often entrapped at the center of ischemic ellipsoid and contracting cicatrix in later stages hence it suffers maximum damage. Ulnar nerve situated at periphery of the zone is variably involved depending on the severity of disease and involvement. Radial nerve is involved only in extensive disease process spilling on to extensor region.

9. What muscles are most commonly involved?

Ans. FDP (partial or complete) and FPL are the first ones to be involved; then depending on the severity of involvement FDS, PT, flexors of the wrist, pronator quadratus, extensors in that order may be affected.

10. What assessment will you do before surgery?

Ans. Muscle groups involved and spared (for tendon transfer), nerve involvement, skin involvement (there may not be enough initial space available for free vascularised transfer due to skin contracture). Thence we stage the disease and decide treatment.

11. When can you do surgery?

Ans. Ideal is to wait for at least 3 months {Seddon; for necrotic region to segregate and spontaneous recovery of muscle

and nerves stops (some say 6 months)} and should be done within a year (Tsuge) to produce good results. Deformity is said to be established if the interval from injury is more than 6 months.

12. How will you treat this case?

Ans. I will get X-ray done to look for the status of bones (malunion/cross-union/nonunion) as they also require treatment. Then I will do initial physiotherapy and stretching to reduce stiffness and improve contractures and do muscle slide operation with neurolysis.

13. What are the various modalities of management?

Ans. Treatment is devised according to the stage of *presentation (this staging is not the stage of disease progression/severity – the Tsuge classification)*

I. Acute Stage (up to 24 hrs): Treat like compartment syndrome.

II. Sub-Acute or delayed stage (from 24 hrs to 3-6 months): Presenting as edema and induration of forearm with paresthesia or anaesthesia and motor weakness or loss. The deformities are progressing during this stage. It is an opportunity to improve sensations and motor function and prevent stiffness and deformity. Mobilization and supervised physiotherapy (Dynamic splinting, stretching) is undertaken. Surgery is indicated if there is neurological impairment, failure of conservative treatment or radiological (MRI) evidence of fibrosis. Neurolysis and displacement of nerve from contracting cicatrix to subcutaneous plane, excision of scar is done. If nerve damage is severe then excision followed by grafting may be attempted.

III. Established VIC (Tsuge classification of disease severity): Mild type:

a. Stretching and physiotherapy: If muscle mass is available.

b. Tendon transfer/lengthening when there is loss of muscle mass by Z-Plasty (of involved FDS, FDP, FPL tendons) or FDS to FDP transfer {Parkes} (by attaching

cut distal end of FDS to cut proximal end of FDP "motorization of FDP").

c. The other way of transfer is to transfer extensor tendon to flexor. For involvement of multiple tendon units prefer muscle sliding operation (Page's operation) or less favourably proximal row corpectomy.

Moderate type (Classic type): Initial stretching followed by:

a. Muscle sliding operation (of Maxpage) with neurolysis (preferred for preserved muscle mass).

b. When there is no useful finger flexion left or there is proximal skin problem then BR/ECRL transfer to flexors (FPL and FDP respectively) and complete release of contracture and neurolysis is the usual option. Other options are proximal row carpectomy or forearm shortening by 2-3 cms (Garre's operation).

Severe type: Preferred treatment includes early excision of all necrotic tissue with complete neurolysis of ulnar and median nerves to give them fair chance to recover (at least 3 months). This can be *followed by tendon transfer* as above or if no tendons are available then Gracilis (muscle after expanding skin with tissue expanders) or lattissimus dorsi/medial gastrocnemius (Myocutaneous) free innervated muscle graft. Carpectomy (Griffiths) and wrist arthrodesis are other uncommonly performed operations in isolation.

In old cases with no available motor, severe contracture and non-salvageable nerve injury; a combined procedure of flexor tenodesis with intermetacarpal fusion with thumb in opposition can be done. Non-salvageable (damn useless) limb can even be amputated.

Thumb function needs to be addressed in moderate and severe types: Release of 1st web space contracture followed by reconstruction of motor function by use of extensors or by intermetacarpal fusion with thumb in opposition.

(Always begin answer by "stretching and physiotherapy to correct contractures and stiffness" then better choose appropriate surgery as above, those underlined are now standard)

14. Why is tendon lengthening not preferred?

Ans. Lengthened tendons often fail to function due to re-contracture and adherence to skin and each other. Further it is ineffective for paralyzed muscles.

15. What is muscle slide operation?

Ans. It is the distal slide (by 3 cms) of flexor pronator mass subperiosteally from common flexor origin and interosseous membrane protecting carefully the ulnar nerve and anterior interosseous nerve, artery and vein. FCU may need to be elevated till the level of wrist. This is followed by anterior transposition of ulnar nerve. The muscles are dissected in the order (ulnar nerve → FCU → PT → FDP → PT (distal origin) → Palmaris longus and FCR → FDS → FCU at interosseous membrane). Advantages are – simple surgery, can be completed in one operation and secondary tendon transfers are possible after this procedure.

16. What are the disadvantages of muscle slide operation?

Ans.

1. Ineffective for paralyzed muscle.
2. Some scar tissue is left behind.
3. There is a risk of recurrence of contracture with growth of the bone.
4. There is a decrease in strength of grip especially in flexion of the DIP joint.
5. All flexor muscles are treated alike irrespective of the severity of damage.

17. What is the most common cause of VIC?

Ans. Long bone fractures either as a primary cause or secondarily due to ill-supervised or ignored treatment. Fracture both bones of leg is the most common cause of compartment syndrome (not of proximal tibial metaphyseal fracture!). In the upper limb fracture supracondylar humerus in children is the most common cause.

18. What are the various compartments in forearm?

Ans. Four:

1. Superficial volar (FDS, FCR, FCU, PL)
2. Deep volar (FDP, FPL, PQ)
3. Dorsal
4. Mobile wad of Henry. (Brachioradialis, ECRL, ECRB)

19. Can you name some eponyms with the name Volkmann?

Ans. Richard Von Volkmann:

- Volkmann's ischaemic contracture (VIC)
- Volkmann's deformity (Congenital talar subluxation/ dislocation)
- Volkmann's splint (for fracture of lower extremity)
- Volkmann's triangle (postero-lateral corner of tibia)
- Volkmann's spoon (sharp spoon to curette away carious bone or other diseased bone)
- Volkmann's cheilitis (lower lip swelling, ulceration, crusting and mucus gland hyperplasia)
  Alfred Wilhelm Volkmann (Father of Volkmann RV)
- Volkmann's canals in bone.

TORTICOLLIS *[TORTUS (L.) – TWISTED; COLLUM (L.) – NECK]*

1. What is your diagnosis?

Ans. My case is a patient of congenital muscular torticollis (CMT) of right side in a male child of one and a half years. There is associated CTEV of right foot.

2. Why do you say that this is torticollis and how do you determine the side?

Ans. On inspection –

- There is contracture of right side sternocleidomastoid muscle.
- The head and neck are tilted towards involved side.
- Ear of the ipsilateral side is touching the shoulder.
- The chin is lifted towards the left side (child seems to be looking in the direction opposite to the side which is involved).

On palpation:
- There is tightness and thickening of the sternocleidomastoid muscle of right side.
  Also examine for identifying the etiology of torticollis (*See Q 4 and 5*).

3. What is the cause of congenital muscular torticollis?

Ans. CMT is caused by fibromatosis within the sternocleidomastoid muscle. It may involve the muscle diffusely, but more often it's localized near the clavicular attachment of the muscle. *Various hypothesis proferred are:*
- Malposition of the foetus *in utero* (resulting in intrauterine or perinatal compartment syndrome of sternocleidomastoid muscle more common towards right side).
- Birth trauma (with resultant haematoma formation followed by muscular contracture – *pseudotumour formation*)
- Infection
- Vascular injury (and fibrosis of muscle).

4. What are the other musculoskeletal disorders that you will look for?

Ans. Developmental dysplasia of hip (associated in 7 to 20% of CMT), metatarsus adductus, talipes equinovarus.

5. What are the other possible causes of torticollis in a child?

Ans. *Pediatric local etiology*
- Congenital causes, such as pseudotumour of infancy, hypertrophy or absence of cervical musculature, spina bifida, hemivertebrae, and Arnold-Chiari syndrome
- Otolaryngologic causes, such as vestibular dysfunction, otitis media, cervical adenitis, pharyngitis, retropharyngeal abscess, and mastoiditis
- Esophageal reflux
- Syrinx with spinal cord tumour
- Traumatic causes, such as birth trauma, cervical fracture or dislocation (atlanto axial rotator subluxation), and clavicular fractures

- Juvenile rheumatoid arthritis
  Pediatric compensatory etiology
- Strabismus with fourth cranial nerve paresis
- Congenital nystagmus
- Posterior fossa tumour
  Pediatric central etiology
- Dystonias include torsion dystonia, drug-induced dystonia, and cerebral palsy.

6. How will you treat a case of CMT?

Ans. Evolution of CMT takes around 12 to 24 months so any surgical intervention should be deferred until evolution of fibromatosis is complete.

In infancy stretching of the sternocleidomastoid by manipulation should be done.

- Manual – by parents
- Plaster casts.
  Surgical intervention can be done after the age of 2 years and the options are:
- Unipolar or bipolar release of sternocleidomastoid muscle
- Selective denervation
- Dorsal cord stimulation
  Post-operatively immobilisation in corrected position by plaster casts, hard cervical collar or head halter traction for 3-6 weeks is done depending on the tolerance of child. Physical therapy including manual stretching of the neck to maintain the overcorrected position is then immediately begun. Manual stretching should be continued three to five times daily for 3-6 months.

7. What are the complications of surgery?

Ans. Complications include injury to spinal accessory nerve or nearby vasculature including the jugular veins and carotid artery. Other complications include neck muscle atrophy, loss of muscle control, instability, variable numbness or sensory loss, pain, and neck deformity.

8. What are the features in a child presenting late?

Ans. In a delayed presentation there are fixed anatomical changes that are hard to reverse. These include:
- Facial asymmetry
- Elevation of ipsilateral shoulder
- Front-occipital diameter of the skull may become less than normal.

9. What precaution will you take before treating a child presenting with delayed presentation?

Ans. One should clearly explain to the parents that the fixed anatomical structures will/may not reverse in spite of the corrective surgery and the surgery only cosmetically corrects the deformity. One should also not be aggressive as the chances of complications are much higher due to secondary contracture and shortening of neurovascular structures.

10. Why do you then do the surgical correction?

Ans. For a neglected case the primary indication for doing surgery is cosmetic correction of unsightly 'wryneck' that has immense psychological implications for a school going child. Also without surgery the deformities become fixed and may even progress. However, the above should be clearly followed and borne in mind.

Miscellaneous Topics

GAIT

{Gait analysis is an important part of examination for lower limb and spine evaluation and no candidate would be spared if he does not possess the requisite knowledge of the same. The topic is difficult and needs lots of practice and guidance for proper presentable knowledge.

Read: 5-7 times (MS Orth and DNB candidates)}

1. **What do you understand by gait?**
Ans. Gait is a dynamic posture allowing bipedal unassisted mobility by virtue of sound interplay between coordination and balance primarily of the lower limb and pelvis. Gait involves complex neuromuscular coordination of the lumbar spine, pelvis, hips and those structures distal to them. Any dysfunction in the lower limb will become observable during gait.

2. **What do you understand by walking, running and jumping?**
Ans. *Walking:* Bipedal unsupported gait pattern where at any time at least one foot is in contact with the ground.
Running: Gait pattern where least at some point of time feet are in air and one foot touches the ground in alternation.
Jumping (both feet): Where at any point of time either both feet are touching the ground or are in air. In single foot jump the other foot never comes in contact with ground!

3. **What are the prerequisites for gait evaluation?**
Ans. The following needs to be closely conformed to:
   1. Gait should be observed in all three planes with and without shoes (bare foot).
   2. Patient should be covering the private parts only during the gait examination.
   3. Observe gait while the patient walks along walkway of 1.1 m wide and 6m long.

4. What is gait cycle?

Ans. It consists of two distinct phases – the swing phase and the stance phase with their sub-phases.

   A. Swing phase (40% of the gait cycle – remember body tries to preserve energy)
      1. Acceleration phase
      2. Mid swing phase
      3. Deceleration phase
   B. Stance phase (60% of the gait cycle – *even while walking we rest more*; actually balance if more important than speed. So as a corollary one is more 'unstable' while running).
      1. Heel strike
      2. Foot flat
      3. Mid stance
      4. Heel off (Push off)
      5. Toe off
      Two periods of double support:
       I. Immediately after the initiation of stance phase
      II. Just before the end of stance phase

5. What are the various dysfunctions that can influence the gait?

Ans.
   1. Neurological (may involve dysfunction of one or a combination of the following parts of the CNS and/or PNS):
      a. Motor: CVA, Cerebral palsy
      b. Sensory: Tabes dorsalis, blindness
      c. Cerebellum: Friedreich's ataxia
      d. Basal ganglia: Parkinson's disease
   2. Systemic disease:
      a. Joint disease: Rheumatoid arthritis, Osteoarthritis, JRA
      b. Crystal arthropathies: Gout
      c. Muscle disease: Duchenne's muscular dystrophy, Dermatomyositis
      d. Bone disease: Rickets, Osteomalacia, Paget's disease

3. Structural:
   a. Limb length inequality: DDH, Polio, femoral/tibial fracture
   b. Alignment disorder: Coax valga/vara, Genu varum/ valgum, tibial/femoral torsion.

6. How to describe gait?

Ans. *Observe the following anatomical & functional features during gait*: (Observe from front, back and side)

1. Head position
2. Shoulder position
3. Arm swing
4. Trunk position/rotation
5. 'Base'
6. Pelvic tilt
7. Limb motion
8. Thigh segment
9. Patellar position/rotation
10. Knee position
11. Tibial position/rotation
12. Ankle
13. Calcaneal position
14. Navicular position
15. Mid-tarsal joint
16. Metatarsals
17. Toe position
18. Foot position/shape
19. Propulsion
20. Swing phase

   The patient is walking with a bipedal unsupported gait having equal smooth alternating and rhythmic stance and swing phases with a normal base. The patient walks with a steady head in coronal plane centered over the shoulders. The shoulders are at same height. There is no trunk sway or rotation. Arm swing is equal and alternate on both sides. There is no pelvis drop on either side and the limbs move in coordination alternately during swing phase. During stance phase the patellar and knee heights

are equal and there is no pelvic drop. The foot on both sides maintains its arch and stance phase progresses smoothly through heel strike, foot flat, mid-stance, heel off and toe off. This patient has a normal gait!

(First mention bipedal or single limb or three/ four point gait commenting type of support used. Then comment on the two phases of gait, their length, smoothness, coordination, alternation and any abnormality. Then one may differ and would describe the pathological features first (like short stance phase with ipsilateral shoulder dip and trunk sway, etc. for antalgic gait) and then describe the other normal findings. Otherwise one may describe the features observed during swing and stance phases sequentially – both are correct and choice is yours – *Always remember to conclude with your diagnosis of type of gait)*

7. What do you understand by base of gait?

Ans. The distance between medial borders of both foot (normally 2-4 inches).

8. What is 'double support' during gait cycle?

Ans. Also known as 'double leg stance'. During gait cycle the body is supported on both foot for a brief period that extends from somewhere between push-off and toe-off on one foot and between heel strike and foot-flat on contralateral side. This comprises some 10% of gait cycle.

9. What is cadence and its importance?

Ans. Number of steps taken in one minute (normal 90-120/ min). The period of double support is inversely proportional to cadence.

10. Can you comment on some common types of gait and their mechanism?

Ans. The various types of gait observed are (see also(Chapter 3):

1. *Trendelenberg's gait (abduction lurch gait):* It may be unilateral or bilateral. It depends upon the abductor lever

arm (*See Chapter 2; Examination points for hip case, Q 33*) any abnormality of which leads to Trendelenberg's gait. When present unilaterally, the patient lurches on the affected side and the pelvis drops on the opposite hip. Bilateral Trendelenberg's gait is also known as *waddling gait*.

2. *Short limb gait:* The patient lurches to the affected side and the pelvis drops to the same side (different from Trendelenberg gait in which the pelvis drops to the opposite side). If the shortening is less than 4 cm, it is compensated by the hyperextension at the knee and equinus at the ankle. Typical short limb gait is seen only when the shortening is more than 4 cms.

3. *Antalgic gait:* Patient walks with shortened stance phase (avoids taking weight on the same limb). Any condition leading to pain in the lower limb (from hip to foot) leads to antalgic gait.

4. *Stumbling gait:* Gait of bilateral CTEV.

5. *Waddling gait or duck walk gait:* It is wide base gait with increased lumbar lordosis, the patient sways to the same side after putting weight on the limb. Seen commonly in pregnancy, bilateral DDH, osteomalacia and myopathies.

6. *Knock knee gait:* While walking the knees point to each other and cross each other and the feet are kept apart. Typical in-toeing gait except the position of the knees on flexion are crossed or near to each other.

7. *Quadriceps gait or 'hand to knee gait' or 'five fingers quadriceps gait':* In cases of weakness of the quadriceps (PPRP – quadriceps are the most common muscles affected in the polio) the patient walks by supporting his knee with his hand during the extension to bear weight. The typical gait is produced to stabilize the knee. The patient does this typical action externally or by putting his hand through the pocket.

8. *Gluteus maximus gait or 'extension lurch gait':* The gait is rarely seen these days as the most common cause was the weakness of the gluteus maximus due to PPRP. The patient lurches backward during walking.

9. *Gluteus medius gait:* The gait is similar to the Trendelenberg's gait as the abductor forces of the hip are affected.

10. *Stiff hip gait:* The patient walks by moving his whole pelvis along with the affected side (swaying to opposite side to clear ground); there is no or minimal movement at the affected hip.

11. *Stiff knee gait:* The patient lifts his pelvis during the swing phase of the gait cycle to get the ground clearance.

12. *High stepping gait or Russian march gait:* The gait is seen in cases of foot drop, patient lifts the affected limb higher and puts the forefoot first over the ground while entering into the stance phase. (Remember- normal gait cycle starts with heel strike)

13. *Scissors gait:* This pattern of gait is seen in cases of weakness with spasticity (weakness is more than the spasticity) of the both lower limbs (CVA, cerebral palsy, early stages of lathyrism etc.). One leg crosses directly over the other with each step as the blades of the scissors. This is also called as the circumduction gate.

14. *Hemicircumduction gate:* The patient moves his limb while dragging his body along with the limb in hemicircle. The pattern is seen in cases of hemiparesis (CVA, cerebral palsy).

15. *Stamping gait:* It is seen in cases of loss of proprioception (sensory ataxia, tabes dorsalis, Vitamin B_{12} deficiency, alcoholism). The patient walks as he has no idea where his foot is leading to thumping noise over the ground due to sudden striking of the foot. The pattern can be imagined as descending the stairs in complete darkness.

16. *Short shuffling gait or festinating gait:* Difficulty in starting and stopping the gait cycle with forward stooping posture and short steps. Typically seen in cases of Parkinsonism.

17. *Charlie Chaplin gait:* This is seen in cases of alkaptonuria, external rotation deformity of tibia and flat feet with valgus deformity of ankle.

18. *Drunkards or reeling gait:* The patient has wide base gait and he swings to each side with tendency to fall with every step. This is seen in cases of cerebellar lesions or alcohol poisoning.

19. *Hysterical gait (Helicopod gait):*The patient walks in bizarre fashion with a tendency to fall on every step; often seen in females with a typical tendency to fold in themselves. Typically the patient does not fall, or falls in safer position and places hence are never hurt. The pattern is present in front of others only.

20. *Calcaneus gait:* The patient has broad heel due to malunion of the calcaneum fracture. The patient has no calcaneal pick up and push off due to weakness of triceps surae. The patient walks with tendency to rotating the foot outwards and genu recurvatum.

21. *In-toeing gait:* Seen in – clubfeet, metatarsus varus, medial tibial torsion, genu valgum, femoral intorsion, acetabular dysplasia.

22. *Peg-Leg gait:* Congenital vertical talus.

11. What are the various crutch gaits and the gait patterns?

Ans. Depending upon the support assistance provided by the crutch and weight bearing limb the gait patterns are divided on the total points of contact bearing the weight at a time.

Four-point gait
• Partial weight bearing both feet
• Maximal support by crutches
• Constant shift of weight over points

Gait-pattern:
1. Advance right crutch
2. Advance left foot
3. Advance right crutch
4. Advance right foot

Three-point gait
• Requires good balance and arm strength
• Non-weight-bearing one foot
• Faster gait
• Can be used with walker assistance

Gait pattern (assuming right foot is affected):

1. Advance left foot with both crutches
2. Advance right foot
3. Repeat sequence to move forward

Two-point gait

- Partial weight bearing both feet
- Minimal crutch support
- Faster than a four-point gait

Gait pattern:

1. Advance left foot and right crutch
2. Advance right foot and left crutch
3. Repeat sequence to move forward

12. What do you understand by "swing-through" and "swing-to" gaits?

Ans. These are the manner in which the feet are brought forward in two-point gait.

Swing to gait:

- Weight bearing both feet
- Requires stability and arm strength
- Faster gait
- Can be used with walker

Gait pattern:

1. Advance both crutches
2. Lift feet $\rightarrow$ swing forward $\rightarrow$ land feet next to crutches.
3. Repeat the sequences again to move forward

Swing through gait:

- Weight bearing both feet
- Requires stability and arm strength
- Most advanced gait

Gait pattern:

1. Advance both crutches
2. Lift both feet $\rightarrow$ swing forward $\rightarrow$ land feet in front of the crutches
3. Repeat the sequences again to move forward

13. What is the advantage of walker and what are the various types of walker?

Ans. Walker provides more support and stability than a cane or a pair of crutches (*the obvious difference between a four-wheeler and a two-wheeler).*

Types of walker:
1. *Pick up walkers:* One that has to be picked up and moved forward with each step, it does not permit a natural walking pattern and is used for patients who have poor balance or limited cardiovascular reserve or who can't use crutches.
2. *Rolling walkers:* It allows automatic walking and is used by the patients who cannot lift or who inappropriately carry a pick-up walker.

14. What is the role of cane during walking?

Ans. Cane provides an additional point of contact that assists patient during gait by redistributing the weight and hence shifting the centre of gravity and line of weight bearing:

Types of canes:
- Quad canes (four footed canes) provide more stability
- Single foot cane one

Placement of the cane
- 15 cm lateral to the base of fifth toe
- Hold in the hands over less affected (or unaffected) side (line of weight bearing shifts to the side where cane is held; unloading the affected side)
- Hold the handle of cane at the level of greater trochanter.

PROSTHETICS AND ORTHOTICS

1. What is prosthesis?

Ans. Prosthesis is a device designed to replace as much as possible the function or appearance of a missing limb or body part.

2. How does it differ from an Orthosis?

Ans. An orthosis (orthos (*G.*) – straight) is a device designed to supplement or augment the function of an existing limb or body part. It controls the abnormal movement or allows restricted normal movements. The following can be devised as functions of splints:

1. Static splints (Immobilize or stabilize joints)
   – Protection
   – To put joints to rest
   – To decrease inflammation
   – To decrease pain
   – To prevent undesired motion
   – To resolve fixed joint contractures (e.g. serial casting)
   – To substitute for lost muscle function
   – To substitute for loss of a digit
2. Dynamic splints (Mobilisation or traction to joints)
   – To resolve tendon tightness
   – To resolve joint contracture
   – To increase activity range on motion to given joints
   – To increase muscle strength.

3. How do you classify prosthesis?

Ans. Prosthesis may be classified in three broad categories:

1. **Endoskeletal**: Most widely used for lower limbs throughout the world. This type of prosthesis consists of a central structural tube to which a joint and socket can be attached. The central tube is mostly made of carbon fiber or aluminum. This basic structure can then be covered by an outer cosmesis in form of shaped foam.
2. **Exoskeletal**: In these prostheses, the main structural component is the "outer visible skin". Now a days, mostly DURAL (aluminum alloy) or plastic laminates are used. Majority of the upper limb prosthesis are of plastic exoskeletal structure.
3. **Temporary "pylon" prosthesis**: This consists of two self locking side struts resembling above knee calipers. They are rarely used in the current circumstances.

   The term *pylon* is also used to describe the central structural tube in the endoprostheses.

4. What components should you specify while prescribing prosthesis?

Ans. While prescribing prosthesis, following details have to be furnished:
1. Type of prosthesis
2. Level of amputation
3. Type of socket
4. Material of socket
5. Hip, knee, elbow mechanism
6. Foot/ankle or hand/terminal appliance
7. Suspension
8. Cosmesis

5. What are various types of sockets?

Ans. From the viewpoint of patient, socket is the single most important factor in prosthesis.
- Sockets may be made of various materials for E.g. Leather, polypropylene, fiberglass, etc.
- Sockets may be standard sockets, which are worn over a stump sock. Now a days, suction sockets are available which remain in close contact to the skin and are worn without a sock.
- Recently, silicone impregnated sock or sock lined by a layer of polyurethane gel have been developed to increase comfort and reduce sweating.

6. What are various types of suspension mechanisms?

Ans. The prosthesis may be:
A. Suspended with the help of belt, cuff or sleeve.
B. Self suspending: These may again be divided into mechanical or suction.

7. What are the various knee mechanism options available?

Ans. The knee systems have met the most advance-ment, the simplest being single axis constant friction type. Other mechanisms are:

Type	Typical patient	Advantages	Disadvantages
Single axis, constant friction	Sedentary, poor access to maintenance	Inexpensive Durable Easy to maintain Light weight	No swing phase control Gait optimal only at one speed Little inherent stability
Weight activated stance phase control (safety knee)	Elderly Neurologic compromise	Inexpensive Durable Provides stability during heel strike and stance phase	No swing phase control Gait optimal at one speed only Reduced flexibility
Polycentric four bar knee joint	Knee disarticulation Amputation	Can be less expensive Stable Hydraulic swing phase control may be added Inherent stability	Bulky Poor cosmesis Increased weight Frequent maintenance
Hydraulic or pneumatic swing phase control	Active young patients with access to maintenance facility	Best swing phase control Adapts to speed Best gait pattern	Expensive More frequent maintenance Heavier Bulky
Manual locking knee	Elderly/weak Unstable	Maximum stability Very light	Unnatural gait Must be unlocked to sit

8. What are the various foot and ankle mechanisms available?

Ans. Main groups include:
1. Articulated ankle joints
2. Dynamic response and energy storing foot

3. Non-dynamic response and/or energy storing foot
The simplest mechanism is SACH which stands for "solid ankle, cushion heel". It consists of a non-articulated ankle and non-dynamic response foot. It is least efficient.
Articulated ankle has the following disadvantages:
   • Heavier
   • More frequent maintenance
   • When combined with dynamic response foot, it prevents loading of toes to provide push off.

9. What is SCAH foot?

Ans. SACH stands for (solid ankle cushion heel). No true ankle joint, contains a 'simulated ankle joint' by compressed wedge shaped rubber heel. Consists of a solid wooden keel, high density rubber for dorsum, low density rubber for toes and plantar aspect (ensures smooth transition from toe-off to heel strike, variable density rubber for heel. The advantages are as follows:
   • Absorbs the impact of heel strike.
   • $\downarrow$ vertical excursion of centre of gravity.
   • Allows some simulated movement of metatarsal head.

10. What is Jaipur foot?

Ans. Developed by Prof P.K. Sethi. Contains two wooden keels (broken keel). There is provision of toe break. The fundamentals if these types of prosthetic foot would get cleared by the following table:

	Jaipur foot	*SACH foot*
1	Appearance similar to normal foot	Dissimilar to normal foot
2	Can be worn without shoe but if someone wishes to wear one he can use a flat heel shoe.	Requires a closed shoe to protect and hide it.
3	No restriction of movements at ankle as the metallic keel (carriage bolt) is confined to ankle only and all movements take place at natural site.	Solid ankle consisting of long wooden keel restricts movements in nearly all directions.

Contd...

	Jaipur foot	*SACH foot*
4	Squatting possible (dorsiflexion adequate)	Not possible
5	Cross-legged sitting is possible (adequate forefoot adduction and internal rotation possible)	Not possible
6	Walking on uneven ground possible (good inversion and eversion)	Suitable for walking only on level ground
7	Barefoot walking possible (cosmetic skin colour, no heel and toe height difference)	Not possible

11. What are the various upper limb prostheses available?

Ans.

1. **Passive/cosmetic**: They are useful in those individuals who want a near life like cosmesis or those who have a high level amputation and they want light weight prosthesis for cosmetic reasons. These are non-functional.

2. **Body powered**: This involves the use of gross body movements to move the components through a system of harnesses and straps.

   a. *Advantages:*
   - Does not depend on battery power
   - Quicker reaction time
   - Less expensive
   - Durable
   - Maximum proprioception

   b. *Disadvantages:*
   - Repetitive injury to activating muscles and joints
   - Limited pinch force control of terminal device
   - Harnessing effect

3. **Externally powered (myoelectric, switch control):** Myoelectric components are controlled by voluntary muscle action via an electronic signal. The signal is picked up and amplified by electrodes placed over muscle fibers and the downloaded over to a computer to provide a

specific function. Before prescribing this type of prosthesis, myoelectric testing has to be done.

a. *Advantages:*
   – Better cosmesis
   – No need for harness
   – Prevents repetitive injury
   – Increased anatomic function
   – Voluntary wrist rotation

b. *Disadvantages:*
   – Heaviest
   – Slower response time
   – Expensive
   – Frequent maintenance
   – Battery dependent
   – Less durable, non water proof
   – Longer training period

4. Hybrid systems: Hybrid system has a combination of body powered and myoelectric components. This combination reduces the weight as well as the cost of the prosthesis.

Various upper limb units:

1. Terminal devices- passive or powered (myoelectric/body powered) hand or hook.
2. Wrist units- both body powered and myoelectric units are available.
3. Elbow units- passive, body powered and myoelectric units
4. Shoulder- only manual units are used.

12. What is immediate post-operative prosthesis?

Ans. Immediate post operative prosthesis- this consists of a specialized dressing covered by a plaster cast molded to provide the patient with weight bearing areas to enable them to ambulate as soon as possible.

The IPOP incorporates an adapter in the distal end that has a removable pylon with a prosthetic foot attached.

Disadvantages of IPOP
   – Pistoning action due to plaster loosening, leading to tissue breakdown

– Repeated changing of casts
– Heaviness of cast leading to reduced movement of contained joints and subsequent muscle atrophy
– Difficulty in regular monitoring of the wound
– Auxiliary suspension may be required.

13. What is post-prosthetic care?

Ans.
1. Balancing, stretching and muscle strengthening exercises may be started as soon as possible. This helps in maintaining flexibility, prevent flexion contractures and preserve muscle mass and strength.
2. An aerobic conditioning program must be designed and incorporated in the rehabilitation process.
3. Gait training should begin with first step. Initially walking stick or walker may be allowed but there use should be terminated as soon as possible.
4. In case of upper limb amputations, early fitting of prosthesis and promotion of two handed function leads to reduced rejection rate.

14. What is Milwaukee brace?

Ans. A brace designed by Blount, Schmidt and Bedwell (1958) known by the city of origin of these men. This brace is used for controlling curve deterioration and to maintain the post-operative correction in patients with scoliosis. The brace has three parts:
– The pelvic mould
– Vertical bars
– Mandibulo-occipital assembly

The pelvic girdle uniformly fits over the iliac crests and is the most important part of brace. It helps reduce lordosis, derotates the spine, and corrects frontal deformity. Uprights have localized pads to apply transverse force which is effective for smaller curves. The main corrective force is the thoracic pad which attaches to two posterior and one anterior upright. Discomfort from the same creates a righting response. Neck ring is another

corrective force and is designed to give longitudinal traction. The throat mould allows the use of distraction force without producing jaw deformity.

Complications

- Pain
- Skin breakdown
- Jaw deformity
- Unsightly appearance
- Increased energy expenditure with ambulation, etc.

WOUND INFECTION, WOUND COVERAGE AND DRESSINGS

{This is typically a topic for DNB exam ward rounds and hardly if ever would be of any utility to MS Orth candidate. The practical utility is however immense and one may read it just out of interest!

Read: 3-5 times for DNB candidate}

1. Describe the wound.

Ans. Wound or ulcer is basically described under following headings:

1. *Inspection*
   a. Size & shape
   b. Number
   c. Position
   d. Edge
   e. Floor
   f. Discharge
   g. Surrounding area
2. *Palpation*
   a. Tenderness
   b. Edge and margin
   c. Base
   d. Depth
   e. Bleeding

    f. Relations to deeper structure
    g. Surrounding skin

- *Inspection findings:* This is a single, oval shaped, roughly 3 cm in length, ulcer (wound) situated over middle one third of right leg on the medial aspect in healing stage. The edge of the ulcer is sloping and pale and smooth granulation tissue is visible at the floor. Discharge is scanty. Surrounding skin is normal. *(look for appendages and colour).*

- *Palpation findings:* The ulcer is slightly tender to palpation and its 5 mm in depth. The base is indurated. It is mobile and not fixed to the deeper structures.

2. What else you would like to examine in this case?

Ans. I would like to examine the regional lymph nodes, peripheral pulses, vascular insufficiency and sensory status *(for neurological status–neuropathic ulcer).*

3. How do you classify an ulcer?

Ans. Ulcers can be classified either on the basis of clinical type or on the basis of pathological type.

    Clinical types are – spreading ulcer, healing ulcer, chronic or callous ulcer.

    Pathological ulcer:
- Non-specific (traumatic, arterial, venous, neurogenic)
- Specific (tuberculous, syphilitic, actinomycotic)
- Malignant (epithelioma, Marjolin's ulcer, rodent ulcer)

4. What are the characteristics of a healing ulcer?

Ans. The floor of a healing ulcer is covered with pinkish or red healthy granulation tissue. The edge is reddish with granulation, while the margin is bluish with growing epithelium. The discharge is slight and serous if at all present.

5. How can you say that an ulcer is a chronic ulcer?

Ans. These ulcers show no tendency towards healing. The floor is covered with pale granulation tissue. Discharge may

be scanty or absent. The base is considerably indurated and so is the edge and surrounding skin.

6. When an ulcer or wound is called as infected?

Ans. Clinical indicators of infection seen on wound examination are -
- Poor quality granulation tissue
- Thinning of granulation tissue
- Increased volume of exudates
- Pain
- Formation of adherent fibrinous slough.

7. Classify a wound?

According to the type	According to the extent	According to local environment of wound
Incisional wound	Deep wet wound	Necrotic wound
Partial thickness wound	Deep dry wound	Sloughing wound
Full thickness wound	Shallow wet wound	Granulating wound
	Shallow dry wound	Epithelializing wound

8. What is the principle of wound management and prerequisites for an ideal dressing material?

Ans. Wound management is based on stepwise approach consisting of:
a. Assessment of both patient health and wound characteristics
b. Planning the course of treatment
c. Cleansing and debridement (establishment of open edge), application of dressing and adjunct therapy.
   The ideal dressing should:
   - Protect from external forces
   - Allow appropriate gaseous exchange
   - Provide moist environment
   - Maintain high humidity
   - Provide optimum pH (slightly acidic)

- Discourage infection
- Provide thermal insulation
- Reduce odour
- Absorb exudates
- Be easy to apply
- Be cost and resource effective.

9. What is the most accepted dressing method-wet dressing or dry dressing?

Ans. Wet dressing is most accepted method and conversion from wet to moist dressing is ideally suited in most cases:

Moist	*Dry*
Reduced risk of infection	Encourages scabformation
Reduced healing time	Delays healing
Faster re-epithelisation	Increases pain
Better cosmetic result	May produce scar tissue

10. Classify different dressing materials with examples.

Ans. Classified on the basis of construction and function.
1. Absorptive
   a. Gauze
      - Plain/Impregnated
      - Foams and polymer dressing
      - Alginates
2. Occlusive (Moisture maintain) dressing
   a. Film dressings
   b. Hydrocolloids
   c. Hydrogels
3. Others
   a. Oxidized regenerated cellulose and collagen
   b. Silicone skin-substitute dressing
   c. Amnion
   d. Homograft/Xenograft

11. Where will you use paraffin gauge?

Ans. It is most commonly used in non-infective granulating wounds and on pink epithelializing wounds. Its major

advantage is that it's non–sticky and can be left for several days.

12. Where will you use iodine or zinc impregnated gauge?

Ans. Skin graft donor site & graft.

13. What is foam dressing?

Ans. It consists of hydrophilic polyurethane open cell sheets and is highly absorptive. It provides moist environment and thermal insulation. It's useful in exudative wounds and needs to be changed in 3-4 days. No role in dry wound.

14. What are alginates?

Ans. They are calcium, sodium salt of alginic acid. These are highly absorbent and haemostatic in nature and also provide moist environment. Commonly used in highly exudative wounds and infective wounds.

15. What are hydrocolloids?

Ans. It consists of two layers; an inner layer which consists of caboxy methylcellulose polymer absorbs exudates and forms gel. The outer layer which is of polyurethane seals the wound. It allows limited moisture and gas transmission and is impermeable to bacteria. It also has fibrinolytic property and is mainly useful in light to moderate level exudative wounds. E.g. Duoderm, nuderm, comfeel. Mainly used in venous ulcers, pressure ulcers, diabetic ulcers, 1st and 2nd degree burns.

16. What is composition of betadine?

Ans. It is iodine combined with polyvinylpyrrolidone. It is broad spectrum - effective against gram positive negative bacteria, fungi, viruses and protozoa. Its preoperative application before surgical incision reduces colonization. It decreases fibroblast proliferation and neovascularisation in chronic wound.

17. Tell something about hydrogen peroxide?

Ans. It is inexpensive and widely available. It uses free radical oxygen radical which scavenges the infective pathogens. Mainly used for cleaning and removing loose debris. It maintains aseptic environment but decreases neodermal regeneration and fibroblast proliferation.

18. What is EUSOL?

Ans. EUSOL stands for Edinburg University Solution. It is basically a Chlorinated lime and boric acid solution 0.25%weight/volume of available chlorine. Better activity with freshly prepared solution.

19. What is oxoferrin?

Ans. It is an aqueous solution which contains bio-catalytically activated oxygen carrier. O_2 has good tissue penetration capacity thus promotes phagocytosis. It provides good cleaning and good local defense against infection.

20. What are the properties of silver ointment used for dressing?

Ans. Silver ions destroy bacterial cell wall, enzymes, DNA synthesis. Additional anti- inflammatory properties – reduces TNF-α which is responsible for rapid wound healing. It is useful in infective contaminated wound.

21. What are the constituents of neosporin ointment?

Ans. It is a combination of neomycin, bacitracin and polymyxin-b. It is effective against wide range of gram positive and gram negative bacteria.

22. What is traditionally known as three layer dressing?

Ans. The three layer dressing consists of:
   1. Contact layer (non-adherent material)
   2. Absorptive layer
   3. Binding layer (tape)

23. Enumerate the stages or grades of pressure sore?

Ans. Given by Shea, recommended by national pressure ulcer advisory panel.

- *Stage I:* Non-blanching erythema (after 30 sec of pressure) of intact skin (partial thickness wound)
- *Stage II:* Partial thickness skin loss involving epidermis, dermis or both (partial thickness wound)
- *Stage III:* Full-thickness skin involved and extending till fascia (full thickness wound)
- *Stage IV:* Full-thickness skin involved and extending to supporting structures (bones, muscles) (full thickness wound)

PRINCIPLES OF FRACTURE FIXATION

{The topic is added only in part to supplement the answers as the viva may be detailed to include the basics of plating and nailing principles. This makes it imperative to have the basic theoretical knowledge also of these. I fully understand that one has a good practical exposure of the same.

Read: 2-4 times for MS and DNB candidates}

1. What are the various modes in which a plate can be applied?

Ans. The following are the various modes of plate fixation:

1. *Neutralisation:* Used to protect primary fixation from torsion, bending, shear. The longer the plate the greater the neutralization capacity.

2. *Buttressing:* Plate directly counters the bending, compressive and shear forces at # site to the applied axial compressive forces. Often used to stabilize peri-articular and intraarticular fractures. Epi-physeal and metaphyseal fractures can displace to produce angular deformity.

3. *Compression mode:* Reduces, stabilize and compreses the fracture site (transverse and short oblique) when lag screw fixation is not possible.

4. *Bridging mode:* To maintain length and alignment of a severely comminuted and segmental fracture. The plate goes over the main injury site as 'bridge' without further disturbing the fracture region.

5. *Tension band:* (Adopted by Pauwels): Plates applied over the tensile surface prevent distraction of fracture fragment and by virtue of eccentric holding, also produces counteractive (compression) forces at the opposite side. Thus the distraction forces are converted into compressive forces due to eccentric hold/ fixation.

6. *Antiglide:* The plate is applied in such a way as to hold the distal fragment indirectly and convert the displacing compression forces into reduction compressive forces, for ex., in Weber type B fractures the proximal spike of distal fragment is caught in the plate, with weight bearing the axial forces are transformed to compression forces at fracture site as the fragment is caught between fracture and plate.

7. *Locked plates:* Where the internal fixation is actually a type of external fixation with the screws locked to both the plate and bone providing an extremely rigid construct. This is beneficial for treatment of complex metaphyseal fractures and osteoporotic fractures but not simple diaphyseal fractures as the callus response may be suppressed and often compression at fracture site is not deliberately tried.

2. What are the principles of buttress plating?

Ans. This plating acts as a retaining wall if applied properly:
- Exactly contour the plate
- Screws should hold plate from moving with respect to bone
- Screw applied closest to the fracture through oval hole is said to be in buttress mode, minimizing axial movement at fracture.

- Buttress plate applies force perpendicular to bone (compression plate applies force parallel to bone).

3. What is spring plate?

Ans. Plate is affixed to only one of the two fragments such that metallic properties of plate help reduce the fracture and hold it (Buttress mode).

4. How do you achieve compression at fracture site?

Ans. By:
1. Overbending the plate
2. Compression device
3. Using plates with dynamic holes that have two inclined and one horizontal cylinders merged together and leads to production of compression with screw tightening (DCP/ LCDCP).

5. What is a 'Wave plate' and what are its uses?

Ans. Similar to bridge plate but the plate is contoured away from fracture site. This gives space to insert bone graft in comminuted fractures and at pseudoarthrosis site. In treating nonunions this plate allows for better in-growth of blood vessels, increases the area for bearing stress (decreasing stress at fracture site), can also act as a tension band by compressing the opposite side.

6. What other modalities (aside from plate) can be used to utilize tension band principle and where all can it be used.

Ans. K-wires with metallic suture wires (encirclage wires), screws with encirclage wiring, simple encirclage wiring can all be used at:
- Greater trochanter (femur)
- Patella
- Olecranon
- Greater tuberosity (humerus)
- Acromio-clavicular joint

- Ulnar styloid
- Medial and lateral malleoli
- Tibial tuberosity fracture.

7. Where do you apply plate in a bone?

Ans. On the tensile surface:
- Humerus – anterior in extension and posterior in flexion! (In elbow stiffness associated with non-union the posterior surface is the tensile surface)
- Radius – lateral
- Ulna – posterior/posterolateral (actually in proximal third posteromedial is tensile but it is difficult to apply plate there)
- Femur – anterolateral
- Tibia – none specifically!

8. What is the principle of nailing?

Ans. Three-point fixation for snugly fitting and elastic nails. The solid unreamed interlocking nail acts as splints only. Nail is a load sharing device so that one does not see stress shielding and resulting osteoporosis. There is transmission of force also through the bone and hence fracture site which helps in bone formation and remodeling at fracture.

9. What is the principle of K-nail?

Ans. Kuntsher called the nailing as elastic nailing and described the mode of action of nailing to be elastic impingement or radial compliance to explain the mechanism of fixation. According to him the nail was released from the elastic constraint as soon as it is in the medullary canal and expands to grip the canal from inside. However, the mode of action is now considered to be three-pint fixation only. K-nail provides angular (bending), translational (horizontal displacement) and to some extent torsional (rotatory) stability, provides axial compression facilitating callus response.

10. What are various generations of nailing?

Ans. Nailing has evolved over years and consecutive advancements can be grouped under three generations:

- 1st generation nailing included K-nail, V-nail, clover-leaf nail, etc. that primarily acted as splints. The rotational stability was minimal and primarily relied on snug fit.

- *2nd generation nailing:* The major advantage was improved rotational stability due to locking screws at either ends. They also relied on snug fit. The proximal femoral entry was piriformis fossa in all. Here the nails were classified into centromedullary (Schneider self broaching nail, Hansen-Street diamond nail, Huskstep nail), cephalomedullary (G-K nail, SUN nail, MDN nail, etc.), caudocephalic nails (distal femoral nails), etc. depending on primary mode of action or technique of insertion. This 'was' the most dynamic phase of nail evolution.

- *3rd generation nailing:* With the aid of CAD various design changes to make the nails as anatomical as possible and to aid the insertion and stability have led to development of multiple curve nails and multiple fixation systems, viz. greater trochanteric entry point for femoral nail with additional lateral curve, femoral nail with femoral neck fixation, tibial nail with malleolar fixation, etc.

11. What do you understand by nail length and working length?

Ans. The nail length is considered from the following viewpoints:

1. Total nail length
2. Length of nail bone contact
3. Working length

Total nail length is primarily an anatomical consideration. Nail-bone contact is difficult to calculate but practically determines the resistance to motion which is directly proportional to length of nail-bone contact.

Working length is the length of nail spanning the fracture site from its distal point of fixation in proximal

fragment to the proximal point of fixation in distal fragment. This represents the part of nail not supported by bone and carries load across the fracture site.

12. What are the implications of working length?

Ans. Working length determines:

- Bending stiffness – inversely proportional to the square of working length
- Torsional stiffness – inversely proportional to the working length
- Strength of construct – the smaller is the working length the stronger is the construct

{Interlocking screws reduce the working length in torsion by fixing the nail to specific part of bone. This also explains why you should impact the fracture before locking the nail. Similarly reaming the canal improves the working length by enhancing the nail-bone contact towards fracture}.

13. What are the methods of reducing 'Hoop Stresses' in nailing?

Ans. Hoop stresses (circumferential expanding forces on the bone walls) are generated when nail is inserted into medullary canal. These may rupture the bone and depend on the insertion force (axial forces converted into radial forces) which, in turn depends on the resistance offered by medullary canal. The following can be done to reduce the hoop stresses:

1. Using a flexible nail
2. Over-reaming the canal
3. Selecting proper entry point.

14. What is dynamisation?

Ans. In general static locking is preferred in comminuted, segmental, long-oblique and spiral fractures. Dynamic locking is otherwise preferable if at least 50% cortical contact is established. Dynamic locking provides *in situ* dynamisation with weight bearing (some 5 – 10 mm

reserve is offered by most modern nails). Sometimes, however, in delayed union 'proximal or distal' screw is removed to allow telescoping and compression at fracture site and aid healing. This allows axial compression but also takes away the rotational stability. Dynamisation is preferably done between 6-10 weeks.

ARTHROPLASTY OF HIP, ELBOW AND SHOULDER

{The basis of writing this chapter is to acquaint the students with the most popular arthroplasty procedures and not at all to detail them! Only those procedures have been dealt that have some significant mention in the book elsewhere – so deliberately quitting knee arthroplasty. It should be remembered however that the hip arthroplasty is so commonly performed now a days that it is a favourite question once you cross the hurdles of examination points and diagnosis.

Read: 3-5 times (MS Orth and DNB candidates)}

ARTHROPLASTY OF HIP

1. What is arthroplasty?

Ans. Arthroplasty is a joint reconstructive procedure using natural or synthetic substitutes or reduction methods (excisional) that alter the structure and function of the joint.

2. What is the principle of low friction arthroplasty as propounded by Sir John Charnley?

Ans. Low friction arthroplasty principles of Sir John Charnley had the following components:

1. Thick plastic socket of high molecular weight polyethylene – this is more suited than a metal bearing as it wears less and works best at high loads and slow speed. It is able to self lubricate in dry state.
2. Small diameter femoral head of stainless steel: The smaller the head the less the wear.
3. Greater trochanter transferred to more lateral position: Increasing offset

4. Fulcrum displaced medially: Centre of artificial joint is more medial than natural joint.

3. What are various types of hip arthroplasty?

Ans.

Resection arthroplasty: Girdle stone type

Interposition arthroplasty: Various natural and synthetic materials interposed between two articulating surfaces. The materials used are muscle, fascia, fat, synthetic membranes (historically – wood! By Carnochan, gold foil by Sir Robert Jones), etc.

Cup arthroplasty: Special interposition arthroplasty in which a cup was used to separate femoral and acetabular surfaces. Smith-Peterson used glass that was then changed to vitallium cup.

Endoprostheses are of following two types:
1. Hemi-arthroplasty:
   - Monoblock non-modular prosthesis like Austin-Moore and Thompson prosthesis
   - Bipolar prosthesis (Bateman's prototype prosthesis, etc.)
   - Tripolar prosthesis (Jumbo head prosthesis)
2. Total joint replacement arthroplasty:
   - Cemented THR
   - Non-cemented THR
   - Hybrid THR
   - Surface replacement arthroplasty (classically double cup arthroplasty)
   - Custom made THR with variable calcar/ femoral replacing systems.

4. What is cemented THR?

Ans. A cemented THR is one in which both the femoral and acetabular components are fixed to bone with a cement interface.

5. What do you understand by hybrid THR?

Ans. In hybrid system only the femoral component is cemented whereas the acetabular component is used in a non-cemented fashion.

6. Why was the need of this system felt?

Ans. Over the evolution of THR in the past 50 years it was seen that in a cemented system the acetabular component fails more frequently to the femoral component. There were more complications of cement related bone degeneration like osteolysis on the acetabular side. So with the development of non-cemented systems acetabulum was fixed to bone without cement interface. However, as regards the femoral component, sturdy fixation of the stem and early mobilisation was possible with the use of cement interface. Also if at all revision would be required it often is limited to changing of acetabular component or liner so cementing of femoral component was retained as a 'permanent' stem. The other major rationale emanates from different mechanisms of failure of cemented acetabular and femoral componenets (see below).

7. What are the various mechanisms of failure of cemented acetabular and femoral components, can you correlate these to development of hybrid system?

Ans. Acetabular (cemented) component predominantly fails due to immunological induced bone lysis destroying the fixation ultimately. This induction is done by wear particles.

Femoral (cemented) component whereas loosens predominantly for mechanical reasons. Early failure is related to thin cement mantle whereas long term failure is due to loosening at cement metal interface at the tip and higher up seen more frequently with first-generation cementing techniques.

Cementless fixation of femoral stem developed with the hope of reducing above however fared poorly and on the contrary more frequent and progressive lysis was

observed seen in anatomic modular locking (AML), Allopro prosthesis (APR), Harris-Galante prosthesis (HGP) and porous coated anatomic (PCA) systems.

Considering the above results one would naturally be intended to use a cemented femoral and cementless acetabular component (hybrid system).

(*Philosophies and schools differ and may continue to do so endlessly – may be till total robotic age comes up! When there will be no 'soft tissue' and only metal prevails*)

8. What decides the use of a cemented or noncemented or a hybrid THR?

Ans. Primarily the decision is based upon the age of patient and bone stock (Dorr classification type A – complete funnelisation (ideal for cementless stem) type C – No funnel, B – intermediate). Requirement for mobility and disease condition for which replacement has been planned are also important while deciding type of prosthesis to use. Elderly patients with poor bone stalk are better dealt with cement augmentation of bone and hence cemented THR. Also for patients requiring early mobilisation and those with limited longevity (viz. tumour patients) cemented components are preferred. Non-cemented prostheses are preferred for younger patients.

9. Can you name some cemented femoral and acetabular components that have evolved over time?

Ans. Cemented acetabular cups:
- Charnley's plastic cup
- Modified Charnley's cup
- Buchholtz cup
- Peg cups, etc
  Cemented femoral stems (these may be curved/<u>straight</u>, collared/<u>collarless</u>, textured/<u>smooth</u>, bowed/<u>straight</u>):
- McKee-Farrar (metal on metal)
- Charnley femoral stem (metal on plastic) with 22.25 mm head

- Muller femoral stem with 32 mm head (medial ridge - 'saber') and with a curve to ease insertion
- Harris femoral component (HD-2): 32 mm head with moderate undercut and oval neck around 3 cm of proximal stem was precoated with PMMA for better fixation.
- Amstutz femoral component (TR-28): Modified Charnley component to a head size of 28 mm and a thicker stem (but not wider).
- Aufranc-Turner component: Similar to Muller cup but the head is more undercut.
  (*A collarless, polished, straight stem subsides well into the cement and converts load into hoop stresses*)

10. What are the various cementing techniques?
Ans. Over a time refinements in the way cement was applied to the components have led to developments into generations in cementing:
- *First generation:* Finger packing, no distal plug.
- *Second generation:* Distal plug of canal, 'preparation' of canal with pulsed lavage, distal centraliser and use of cement gun to insert the cement in a retrograde fashion.
- *Third generation:* precooling of cement, vaccum mixing and centrifugation, pressurization of cement by use of proximal seal.
- *Fourth generation:* In addition, this uses a proximal centra-lizer to ensure symmetric cement mantle.

  During the same periods there were some 'implant characteristics' that improved however they are not grouped into generations. In the first generation the implants had 'sharp' borders that used to split the mantle and were prone to 'mid-stem pivot effect' whereby there was excessive medial pressure in the proximal portion and lateral pressure distally. In the second generation the implants were made of superalloys and sharp corners were removed. During third generation the surface characteristics were improved to increase bonding.

11. How much cement mantle is considered adequate?

Ans. 2-5 mm. The place where cement is missing or very thin is called 'mantle defect' and is often the site of failure.

12. What is cement disease?

Ans. "Cement disease" applies to the previously prevailing concept of ill effects on the body and local bone due to leakage of hot monomer, thermal injury during curing phase, degradation and release of particles including contrast. Seemingly there is no strong biological backing for this mundane term.

13. What are the various types of cements available?

Ans. There is no standard classification but the available types of cements evolved on the needs of different types of procedures:

- *High viscosity cements:* Standard arthroplasty procedures now not preferred.
- *Low viscosity cements:* The most commonly used cement, surface replacement and vertebroplasty (very low viscosity cement).
- *Antibiotic impregnated cements:* Revision and infection in arthroplasty, spacer, cement beads for defect management in chronic osteomyelitis.
- Cold curing cements (Mjoberg) using butyl methacrylate.
- Biodegradable aqueous gel phase cement.

14. What are the components of standard cement?

Ans. Cement is provided as biphasic module (2-3 parts powder and one part monomer liquid). The solid phase comprises powder form microbeads (1-100µm) of polymerized component (PMMA) with opacifier (barium sulfate or zirconium oxide) and initiator (benzoyl peroxide). The liquid phase consists of monomer methyl methacrylate and co-initiator (aka activator – Dimethyl-p-Toulidene, DMPT) along with stabilizers to prevent autopoly-merisation (hydroquinone and /or ascorbic acid). The

colouring agents like chlorophyllin can be added to any phase while antibiotics are added only to solid (powder) phase.

15. What are the various antibiotics that can be used?

Ans. Most commonly used are aminoglycosides, vancomycin. However, the β-lactams, cephalosporins, macrolides, quinolones, doxycycline can all be used. Basically the antibiotic should be heat stable, water soluble, hypo-allergic, bactericidal and available as a powder.

16. What are the various phases of polymerization?

Ans.

1. Mixing phase: Wetting
2. Waiting phase: Swelling + polymerization, ↓ viscosity, sticky dough
3. Working phase: Chain propagation, ↓ movability, ↑ viscosity.
4. Setting phase: chain growth finished, no movability, high temperature

   There is shrinkage of the mix finally and the whole process is exothermic (heat of polymerization = 43-46°C.

17. What is the effect of precooling/ warming of prosthesis?

Ans. Both are deleterious. Precooling leads to shrinkage of the material at cement-prosthesis interphase – early loosening. Prewarming leads to ↓ conduc-tive capacity of prosthesis → heat necrosis → early loosening. (*So warm prosthesis till body temperature*)

18. What are the effects of vacuum mixing and centrifu-gation?

Ans. Vacuum mixing results in ↑ in bending strength by 15-30%.

   Centrifugation improves the fatigue strength by ≈ 9%.
   Precooling of monomer, polymer and mixing vessels
↓ number and volume of pores.

19. What are the various bearing surfaces?

Ans. The most popular one is metal (cobalt-chrome alloy) on UHMWPE (ultra high molecular weight polyethylene). The others are:

- Metal on metal
- Ceramic on UHMWPE
- Ceramic on ceramic (alumina on alumina third generation bearings)
- Failed bearing surfaces:
  – Ceramic on metal: High frictional torque and wear
  – Titanium on polyethylene: High wear typically third body wear.

20. What are the various types of cementless stems?

Ans. There are three methods of cementless stem fixation:

1. *Press-fit:* Moore and Thompson prototype stems. Lord and Sivash femoral stems. These rely on the development of bone 'around' stem to give a tight fit.

2. *Macro-interlock:* Here the press-fit is supplemented by mechanically carving out ribs, threads, steps, etc. in the stem.

3. *Porous coated stems (modern stems):* The porous coating could be of hydroxyappatite (ceramic pore size ≈ 50 µm) or spongy metal porous coating in the form of small spherical beads (cobalt-chrome/ titanium, pore size of 50-400µm) or mesh applied by sintering or diffusion bonding. Bone 'ingrowth' is considered optimal if micromotion is <20µm at bone-implant interface. Some examples are AML, HGP, PCA stems. For porous coated ceramic stems; the cells migrate into the pores (osteo-conduction) and bone may form from the coating itself! The following characteristics are found:

   a. *Diaphyseal (distal) fit or metaphyseal (proximal) fit:* Latter is preferred. In the former one there is complete porosity over the surface that may stress shield the metaphysis due to early distal fit and lead to metaphyseal bone loss and are also prone to cause thigh pain. The metaphyseal fit stems to have only proximal porous coating.

b. *Anatomical or straight stems:* The former have proximal posterior bow and a variable distal anterior bow (only for revision long stems). This mandates side determination for prosthesis as anteversion is also additionally built into the neck.

c. *Circumferential or patchy porous coating:* Newer designs have circumferential coating earlier designs had patchy coating that served to circulate particulate debris around the stem – "effective joint space".

d. *Collared or a collarless stem:* Collar in a cementless stem is useless! As if collar fits before stem fit – stability is compromised, if stem fits before collar is seated it is ineffectual.

21. What is the rationale for using recently launched large head bearings?

Ans. Large head with limited endoprosthetic components that waste a very limited amount of patients bone has been popularized as surface replacement arthroplasty. Also there are various modular components which can fit large metal bearings on standard endoprosthetic components. The highly polished surfaces provide very limited wear and improved stability and range of motion as compared to the conventional arthroplasty.

22. What are various types of wear seen in THR?

Ans. Wear is the loss of rubbing surfaces due to repetitive motion and friction.

1. *Abrasive:* Due to rubbing of two hard surfaces
2. *Adhesive:* Rubbing of a soft surface onto a hard one in which the former is transferred as a thin film over the latter.
3. *Fatigue:* Due to repetitive loading
4. *Corrosive wear:* Due to different types of metals (Galvanic), etc.

Linear wear: With linear wear the head penetrates into the acetabulum due to high contact pressure (linear distance travelled by head). This wear is more common with smaller heads (say 22 mm). (*The smaller heads may also*

penetrate the acetabulum due to 'cold flow' of plastic – plastic deformation).

Volumetric wear: This is due to frictional torque and is more with larger (say 32 mm) heads! *(You see why we use 28 mm heads. The larger heads have again come in vogue due to improvement in plastic characteristics – the highly cross-linked polyethylene)*

23. What are the indications of total hip arthroplasty?

Ans. Primary and secondary osteoarthritis, osteonecrosis, inflammatory arthritis, dysplastic hip, PFFD, pathological fracture of proximal femur, conversion of arthrodesis into arthroplasty, etc.

24. What are the contraindications of hip arthroplasty?

Ans. *Absolute:* Active or latent infection at local/ distant site, medically unfit patient with a high risk to benefit ratio.

Relative: Neuropathic arthropathy, rapid bone destruction, insufficiency of abductor mechanism, rapidly progressive neurological disease.

25. What are the complications of THR?

Ans. Various complications are seen; all related to major surgical procedure and to prosthesis fixation itself:

Immediate: Bleeding and vascular injury (external iliac, obturator, superior gluteal), sciatic/ femoral/ obturator/ peroneal nerve injury, fracture of femur/acetabulum, overreaming and unstable implant, bladder injury.

Early: Fat embolism, deep vein thrombosis, thromo-embolism, dislocation, infection. Renal failure.

Late: Loosening and osteolysis, wear, thigh pain, lurch, protrusion, heterotopic ossification, peri-prosthetic fractures.

ELBOW ARTHROPLASTY

1. What are the indications of elbow arthroplasty?

Ans. Age >60 years, advanced arthritis or post-traumatic destruction of joint in a low demand patient.

2. What are the various types of elbow prosthesis?

Ans. Semi-constrained (linked prosthesis) like GBS III elbow and Coonard-Morrey prosthesis and unconstrained or unlinked prosthesis like Kudo and IBP elbow.

3. What are the prerequisites for unconstrained prosthesis?

Ans. There should be good bone stock, little deformity, stable capsuloligamentous support.

4. What are the indications for unconstrained prosthesis?

Ans. Elderly patients with rheumatoid arthritis, painless ankylosed elbow, e.g. Juvenile rheumatoid arthritis.

5. What is the prosthetic choice for post traumatic elbow?

Ans. Constrained (linked) prosthesis as the capsuloligamentous structures are damaged.

6. What are the indications for constrained elbow prosthesis?

Ans. Deficient bone stock, unstable capsuloligamentous support, deformed joint.

7. What is Bakshi's sloppy hinge prosthesis?

Ans. It is a type of semi-constrained linked prosthesis with a loose hinge (sloppy) to partially compensate for the rotational stress on the prosthesis hinge.

8. What are the limitations after total elbow arthroplasty?

Ans. The person cannot lift weight >5 Kg and should avoid contact sports for life time.

SHOULDER ARTHROPLASTY

1. What are the indications of shoulder arthro-plasty?

Ans. In general shoulder arthroplasty is recommended for patients with symptomatic gleno humeral arthritis (osteoarthritis, rheumatoid arthritis), traumatic arthritis, osteonecrosis, rotator cuff arthropathy and four part non-reconstructible proximal humerus fractures.

2. What are the various options available?

Ans.
- Hemiarthroplasty
- Total shoulder arthroplasty
- Reverse shoulder arthroplasty

3. When do you decide between hemi and total shoulder replacements?

Ans. The demarcation is vague and controversial but the following guidelines are generally followed: Always first look at two crucial components to be restore – arthritis and instability.

Hemiarthroplasty (HSA):
- Rough and destroyed humeral articular surface with intact glenoid cartilage with enough glenoid to stabilize the humeral prosthetic head
- There is insufficient bone to support glenoid component with irreparable cuff tears
- Fixed upward displacement of humeral head relative to glenoid
- History of remote joint infection
- Heavy demands anticipated for the joint
- Four-part non-reconstructable fracture of humeral head.

Total shoulder arthroplasty (TSA)
- Incongruent joint surfaces
- Normal or reparable cuff tears
- Loss of articular cartilage on both surfaces

Reverse total shoulder arthroplasty (rTSA)
- Arthritis and/or instability from non-reconstructable soft tissue or osseous defects
- Posterior aspect of capsule and rotator cuff have been lost.
- 'Anterio-superior escape' due to coracoacromial arch deficiency (wear, fracture, acromioplasty!)
- Slackened deltoid unable to lift the humerus for abduction 'pseudoparalysis'.
- Failed previous conventional arthroplasty.

4. What is reverse total shoulder prosthesis?

Ans. This prosthesis involves making glenoid 'ball' that articulates with concave humeral trumpet shaped component, in effect it is reversal of the normal anatomical joint.

5. What are the prerequisites for hemiarthroplasty?

Ans. *(This is the most popular procedure world over. Total shoulder needs expertise and over all the indications are not very clear. Reverse total shoulder is mentioned only for candidate to have an idea that this relatively new system exists. The experience is very limited and results not extensively quantified or qualified. So at the summit it becomes clear that if asked about shoulder arthroplasty then hemiarthroplasty will be the choice-unless furiously refrained to by an occasional examiner)*

- Concentric glenoid consisting of eburnated bone
- Non-concentric glenoid that can be converted to a smooth concentric surface by reaming
- The humeral head can be centered in the glenoid by soft tissue balancing and glenoid preparation
- The surgeon is proficient with soft tissue and osseous procedures!

6. What is the rationale for development of reverse total shoulder arthroplasty?

Ans. There are various limitations for conventional shoulder arthroplasty that could be addressed by reverse total shoulder arthroplasty as follows:

- Limitation of TSA/HSA to manage glenohumeral translation: For the exquisite ROM of a normal shoulder there is translation of humeral head especially at the ends. This is limited in perfectly conforming joint surfaces of TSA/ HSA.
- Limited fixation of glenoid component to bone in TSA
- Limited intrinsic stability of TSA/ HSA: (*See above for indications of rTSA*). The TSA/ HSA can be done for the following conditions:
    - Arthritis/instability due to deficiency of humeral head (HSA)

- Arthritis/instability due to deficient glenoid that can be reconstructed (TSA)
- Arthritis + acute reparable rotator cuff tears (HSA/ TSA)
- Arthritis + excessive capsular laxity: Tightening + large head HSA/ TSA with increased lateral offset and tissue balancing
- Arthritis + upward displacement of humeral head (intact coraco-acromial arch) (HSA/ TSA)

• Limited ability for compensation of deltoid dysfunction (*These are the most preferred explanations for development and use of rTSA, results are beginning to come and have been satisfactory*)

7. What are the limitations of shoulder arthro-plasty?
Ans.
- Skin, vascular, osseous deficiency
- Infection
- Deltoid deficiency, limited scapular mobility
- Unfit patients (medical, emotional, motivational issues)

8. What are the differences of shoulder arthro-plasty as compared to hip arthroplasty?

Ans.
- Shoulder arthroplasty depends upon soft tissue balancing primarily
- The humeral head is in retroversion (femoral head is anteverted)
- Glenoid is in minimal retroversion (acetabulum is anteverted)
- The approach to shoulder is anterior (hip is posterior/ lateral)
- The glenoid surface (concave surface) is small and humeral surface (convex surface) is larger (in THR femoral head is smaller than acetabulum) (*This 'issue' is also resolved by reverse shoulder!!*).

Long Questions for Theory Examinations

SURGICAL TECHNIQUES AND APPROACHES

1. Bone grafting, types and technique. Describe bone graft substitutes.

IMAGING IN ORTHOPEDICS

1. Role of ultrasonics in orthopedics.

2. Role of MRI and CT scanning in orthopedics.

THE HIP

1. Describe the pathoanatomy of DDH with relevance to the treatment and the role of pelvic osteotomy.

2. What is the etiopathogenesis of SCFE (SUFE)? Describe in detail the management of acute slip with a note of the advantages and disadvantages of various treatment methods for chronic slip.

3. Classify Perthes' disease with a note over the significance of changes in head and describe the management in various stages with a note on bracing and its role.

4. Give a note of the clinical and pathological aspects of septic arthritis. Describe the management of sequelae of septic arthritis.

5. Describe the etiopathogenesis of tuberculosis of hip and management of TB hip in stage III.

6. How will you manage ankylosed hip in a patient with ankylosis of both hip joints? Describe the principles of low-friction arthroplasty.

7. Describe the pathogenesis and management of Coxa vara.

8. Describe the vascular supply of head. What is the role of steroids in production of osteonecrosis of femoral head and how do you manage stage three Ficat and Arlet hip in a 40-year-old male.

9. Describe the causes of loosening in THR and management of failed hip (also learn for TKR).

10. Describe the various dislocations around hip and management of a case of old neglected posterior dislocation of hip and its complications.

11. Classify fracture neck of femur in adults and what are the various complications. Outline the management of subcapital fracture neck of femur in a 35-year-old male.

12. How do you classify fracture of intertrochanteric region of femur? Describe the advances in management of intertrochanteric fractures.

13. Define femoral anteversion and clinical evaluation. What is the role of femoral anteversion in hip biomechanics, development and clinical diagnosis?

14. Classify pelvic fractures and management of unstable patient with vertically unstable pelvis injury (basically also rule out abdominal injury).

15. Classify fracture acetabulum and management.

16. Briefly describe the valgus osteotomy of proximal femur and its role in orthopedics with merits and demerits.

THE KNEE JOINT

1. Classify knee instability. How will you manage ACL deficient knee?

2. Classify patellar instability. How will you manage a case of habitual dislocation of patella in a 16-year-old female?

3. How will you manage a 60-year-old male with Kellgren grade 3 bilateral knee osteoarthritis? What is high tibial osteotomy and list the various methods to perform the same?

4. Describe the pathology of osteoarthritis and the biomechanics of malaligned knee. What is the role of surgery in correction of malalignment?

5. What are the various causes of anterior knee pain? Describe the pathoanatomy and management of patellofemoral overload syndrome.

6. Enumerate various methods of ACL reconstruction including types of grafts used and their relative merits and demerits.

7. How will you approach a patient with unstable knee? Briefly describe the treatment algorithm.

THE LEG, FOOT AND ANKLE

1. Describe the principles of distraction histogenesis. How will you manage a 21-year-old male with post-traumatic 8 cm limb shortening with anterior angulation of 30° of lower third tibia?

2. Describe the pathoanatomy of clubfoot and the role of talo-calcaneo-navicular complex. How will you manage a 6-year-old child with neglected clubfoot?

3. Orthopedic management of leprosy foot.

4. Clinical features and management of old neglected Achilles tendon rupture?

THE SHOULDER JOINT

1. Morbid anatomy of unstable shoulder and management.

2. Etiopathogenesis of rotator cuff tear and its management.

THE ELBOW AND ARM

1. Management of neglected elbow dislocation.

2. Management of stiff elbow.

3. What are the various restraints for elbow and how will you manage an unstable elbow?

4. Describe in detail the pathoanatomy of un-united fracture of lateral condyle and detail its management.

5. What is nonunion? How will you manage a case of non-union of humerus in a 30-year-old male?

THE HAND AND FOREARM

1. Describe in detail the etiopathogenesis, clinical and management perspectives of congenital radio-ulnar synostosis.

2. What is Madelung's disease? Describe in detail the management.

3. What do you understand by intercarpal instabilities? Describe in detail the morbid anatomy and management perspectives.

4. A 45-year-old female presents with paresthesia and pain over radial 31/2 fingers, described in detail the morbid surgical anatomy, clinical features and management of the patient.

5. Classify and describe Kienbock's disease with respect to etiology and surgical management of various stages.

6. Classify fracture scaphoid. What are the various complications, describe in detail the management of scaphoid non-union?

7. What is tardy ulnar nerve palsy (ulnar neuritis), describe the surgical management?

8. How will you manage 6-month old case of post-traumatic radial nerve palsy associated with non-union of fracture humerus?

9. Describe the components of extensor mechanism of fingers. Classify and describe the surgical management of Boutonnière and Swan neck deformities.

10. What are the various spaces in hand, describe with respect to deep hand infections and pathogenesis?

11. What are the various zones of flexor tendons? Describe the management of zone II injury?

12. Describe the etiopathogenesis, classification, anatomy and manipulative and surgical management of Volkmann ischemic contracture.

13. Describe the anatomy of peripheral nerve. How will you manage a case of high ulnar nerve palsy.

14. Outline the formation of brachial plexus and describe the course of ulnar nerve. What are the various sites of compression of ulnar nerve and clinical and management perspectives?

15. Anatomy and pathology of brachial plexus injury. How will you differentiate preganglionic from post-ganglionic brachial plexus injury?

THE SPINE

1. Describe the structure of intervertebral disc. Describe in detail the clinical presentation of PIVD L4-L5 disc and current surgical management perspectives.

2. What is scoliosis and how do you classify the same. Describe the management of idiopathic scoliosis in a 17-year-old girl?

3. Describe the structure of spinal cord with respect to the location of spinal tracts. Describe in detail the rehabilitation of a traumatic paraplegic patient.

4. How do you define and classify spondylolisthesis. How will you manage grade III L5-S1 listhesis.

5. Classify traumatic injury to spine and describe the various spinal cord syndromes.

6. Describe the diagnostic and management perspectives of failed back syndrome?

TUMORS

1. Describe in detail the etiopathogenesis, histopathology and management of giant cell tumor of bone.

2. Classify and describe the osteosarcoma of long bones and its syndromic associations. Outline the management of a case of osteosarcoma of proximal tibia and the role of immunotherapy.

3. How will you approach a patient with metastasis and unknown primary?

4. Principles, evolution and practice of limb salvage surgery.

5. Describe in detail the radiologic and pathologic features of Ewing's sarcoma. Describe the surgical management of chemosensitive tumors.

6. Detail the work-up for a metastatic tumor to bone. Classify spinal metastasis and how will you manage a 55-year-old female with localized spinal metastasis with paraparesis?

GENERAL ORTHOPEDICS, INFECTIONS AND FRACTURES

1. Describe the clinical anatomy of iliopsoas muscle and iliotibial tract.

2. What is osteoporosis; describe in detail the diagnostic and management perspectives?

3. What is the blood supply of talus? Classify and describe the management of fractures of talus.

4. Describe the etiopathological advances of cerebral palsy and classify the disease. When and how will you surgically manage a case of knee deformity due to cerebral palsy?

5. Biological therapy for osteoporosis.

6. Immunotherapy for rheumatoid arthritis.

7. Describe the management of postpolio residual paralysis and foot deformities in a 12-year-old child.

8. What is pott's paraplegia? Classify and describe the causes and management of a case of late onset paraplegia?

9. Antitubercular chemotherapy.

10. What is gas gangrene and responsible pathogens? Describe the management of gas gangrene of lower limb (also learn for necrotizing fasciitis).

11. What are the various mechanisms for joint lubrication?

12. Describe in detail the clinical biomechanics of hip joint and correlate the use of rehabilitative devices.

13. Describe in detail the structure of physis. Classify physeal injuries. How will you manage a case of type three physeal injury (S-H) of distal femoral epiphysis?

14. Describe the management of disaster at state level (this was the long question in my DNB exam!!).

Short Questions for Theory Examinations

1. Anterolateral decompression.
2. Spinal osteotomy.
3. Posterior fusion.
4. Pedicular screw.
5. Steps of discectomy (Fenestration).
6. Bankart's repair.
7. Putti-Platt repair.
8. Anterior approach to shoulder for open reduction.
9. French and modified French osteotomy and difference.
10. Ulnar nerve transposition.
11. Maxpage release.
12. Carpal tunnel release.
13. Darrach's reconstruction at wrist.
14. Wrist fusion, types and indications.
15. Drainage of various hand spaces.
16. Opponensplasty.
17. Southern approach, Harding's approach to hip.
18. Subtrochanteric valgus osteotomy.
19. McMurray's osteotomy.
20. Pauwels osteotomy.
21. Core decompression.
22. Girdlestone arthroplasty.
23. Shelf procedure.
24. High tibial osteotomy.
25. Approaches for total knee arthroplasty.
26. ACL repair.
27. Phemister grafting.
28. Sofield's osteotomy.
29. Posteromedial soft tissue release.
30. Dwyer's osteotomy.

31. Lichtblau osteotomy.
32. Jone's transfer.
33. Lambrinudi, Grice-Green arthrodesis.
34. Hallux valgus.
35. Hallux rigidus.
36. Rigid flatfoot.
37. Arches of foot.
38. Fungal infections of foot.
39. Calcaneal spur.
40. Trophic ulcer.
41. Diabetic foot.
42. Talocalcaneal bar.
43. Congenital pseudoarthrosis of tibia.
44. Varicose ulcer.
45. Congenital constriction band.
46. Fibular hemimelia.
47. Endobutton.
48. Patella alta, radiology and pathoanatomy.
49. Complications of TKA.
50. Bursae around knee, Clergyman's knee.
51. Arthroscopy of knee, optical principles and port designing.
52. Applied anatomy of quadriceps, quadriceps contracture.
53. Triple deformity of knee.
54. Osgood-Schlatter disease.
55. Osteochondritis dissecans.
56. Pivot-shift test.
57. Anterior drawer test.
58. Ober's test and ITB contracture.
59. Proximal focal femoral deficiency.

60. Bone cement.
61. Aseptic loosening.
62. Trendelenburg's gait.
63. Snapping hip.
64. Impingement syndrome.
65. Congenital pseudoarthrosis of clavicle.
66. Sprengel shoulder.
67. Cervical rib, thoracic outlet syndrome.
68. Milwaukee shoulder.
69. Subacromial bursitis.
70. Tennis elbow.
71. Congenital radial head dislocation.
72. Allen's test.
73. Mucous cyst and differentials with a note on giant cell tumor of tendon sheath.
74. Principles of tendon transfer.
75. Strength-Duration curve.
76. Trigger finger.
77. Lobster hand.
78. Dupuytren's contracture.
79. Tenodesis.
80. Neurolysis.
81. Mallet finger.
82. Management of moderate VIC.
83. Radial club hand.
84. Thumb aplasia.
85. Flexor pulley.
86. Glomus tumor.
87. Groin flap, skin grafting.
88. Myocutaneous flap.

89. Spina bifida.
90. Spinal shock.
91. Definition of global instability.
92. Klippel-Feil syndrome.
93. Fractures of spine and mechanism.
94. Lumbar canal stenosis.
95. Vertebra plana.
96. Microwave diathermy.
97. TENS.
98. Floor reaction orthosis.
99. Splints for radial and ulnar nerve palsy.
100. Milwaukee brace.
101. Jaipur foot and SACH foot.
102. Closed chain exercises.
103. Pavlik harness.
104. Hydrotherapy.
105. Lumbar and brachial plexus.
106. Structure of giant cell and giant cell lesions.
107. Structure of peripheral nerve and Wallerian degeneration.
108. Blood supply of scaphoid.
109. Vertebra plana.
110. Myositis ossificans progressiva.
111. Floating knee.
112. Stress fracture, Pathological fracture, Avulsion fracture.
113. LCDCP, DCP, LCP, interfragmentary compression (principles of lag screw).
114. Fracture disease.
115. AO principles of fracture management and evolution of ideology.
116. Biological fixation.

117. Reflex sympathetic dystrophy and complex regional pain syndrome.

118. Functional cast bracing.

119. Piezoelectric effect and bone healing.

120. Fracture healing and modulating factors, fracture healing enhancers.

121. Management of polytrauma patient and principles of triage.

122. Advanced trauma life support.

123. Management of pelvic injuries.

124. Crush syndrome.

125. Ankle fracture – classification and management.

126. Factors leading to non-union lateral fracture of condyle and management.

127. Fat embolism.

128. Pulmonary embolism.

129. Low molecular weight heparins and prevention of deep vein thrombosis.

130. Septic shock.

131. Fluid and electrolyte imbalance.

132. Tension band principle.

133. Classification of compound fractures and management of compound tibial fractures.

134. Biodegradable implant.

135. Use of silicone in orthopedics.

136. Compartment syndrome – diagnosis and management.

137. Classification and management of calcaneal fractures.

138. Classification and management of talus fractures.

139. Plaster of Paris, resin based fiber cast.

140. Soft tissue coverage of lower limb.

141. Acromioclavicular dislocations.

142. Sterno-clavicular dislocations.
143. Trans-scapho-perilunate dislocation.
144. Carpometacarpal dislocation.
145. Lisfranc dislocation.
146. Divergent elbow dislocation.
147. Marble bone disease.
148. Osteogenesis imperfecta classification and management.
149. Pseudofractures, role and actions of Vit D3.
150. Neoosteogenesis.
151. Renal osteodystrophy.
152. Renal rickets.
153. Uses of LASER in orthopedics.
154. Uses of ultrasonics in orthopedics.
155. Charcot's arthropathy.
156. Gas gangrene.
157. Utility of MRI in differentiating infective from mitotic pathology.
158. Role of bone scans in diagnosing orthopedic infection and inflammation.
159. Role of FDG PET in metastatic work-up.
160. Synovial disorders of joints.
161. Adamantinoma.
162. Diagnosis and management of solitary plasmacytoma.
163. Diagnosing and differentiating osteoid osteoma and osteoblastoma.
164. Methods and critical analysis of bone grafting and bone graft substitutes.
165. Café-au-lait spots.
166. Principle of limb salvage surgery.
167. Role of immunotherapy in bone tumors.
168. Compound palmar ganglion.

169. Regimen of ATT and drug interactions.
170. Histology of tubercular granuloma.
171. Fungal disease of bone.
172. Brodie's abscess.
173. Sequestrum.
174. Granulomatous osteomyelitis.
175. Role of reaming in femoral osteomyelitis.
176. Obstetrical palsy, its etiopathogenesis and management.
177. Stem cell therapy in orthopedics.
178. Posterior approach to hip joint.
179. Steps of fenestration for PIVD.
180. Steps of anterolateral decompression.
181. Use of tourniquet in orthopedics.
182. Radionuclide bone scan.
183. Bearing surfaces for THR.
184. Implant infections.
185. Trochanteric flip osteotomy for hip exposure.
186. Tribology.
187. Femoroacetabular impingement syndrome.
188. Autoimmune disorders in orthopedics.
189. Anti-CCP test and significance in rheumatoid arthritis.
190. Pseudotumors and management (hemophilia and metal on metal hip arthroplasty).
191. Pigmented villonodular synovitis.
192. SEMLR (Single event multiple level resections).
193. Quadriga effect.
194. Giant cell tumor.
195. Paraffin wax and role in orthopedics.

Etc.etc..etc…etc…. virtually everything has been asked in DNB theory exams!!! And what you consider has not been asked will be asked in coming times?!?!?!