FIGURE 28.5 The slump test: one of the stages

5. Both legs are straightened together.

6. The foot of the affected straightened leg is dorsiflexed.

Note: Take care to distinguish from hamstring pain. Deflexing the neck relieves the pain of spinal origin, but not hamstring pain.

Significance of the slump test

- It is positive if the back or leg pain is reproduced.
- If positive, it suggests disc disruption.
- If negative, it may indicate lack of serious disc pathology.
- If positive, one should approach manual therapy with caution.

Neurological examination[10]

A neurological examination is performed only when the symptoms, such as pain, paraesthesia, anaesthesia and weakness, extend into the leg.

The importance of the neurological examination is to ensure that there is no compression of the spinal nerves from a prolapsed disc or from a tumour. This is normally tested by examining those functions that the respective spinal nerves serve, namely skin sensation, muscle power and reflex activity.

The examination is not daunting but can be performed quickly and efficiently in 2 to 3 minutes by a methodical technique that improves with continued use. The neurological examination consists of:

1. quick tests: walking on heels (L5), walking on toes (S1)
2. dural stretch tests: slump test, straight leg raising
3. specific nerve root tests (L4, L5, S1): sensation, power, reflexes

Main nerve roots

Refer to FIGURE 28.6 and to TABLE 55.3 .

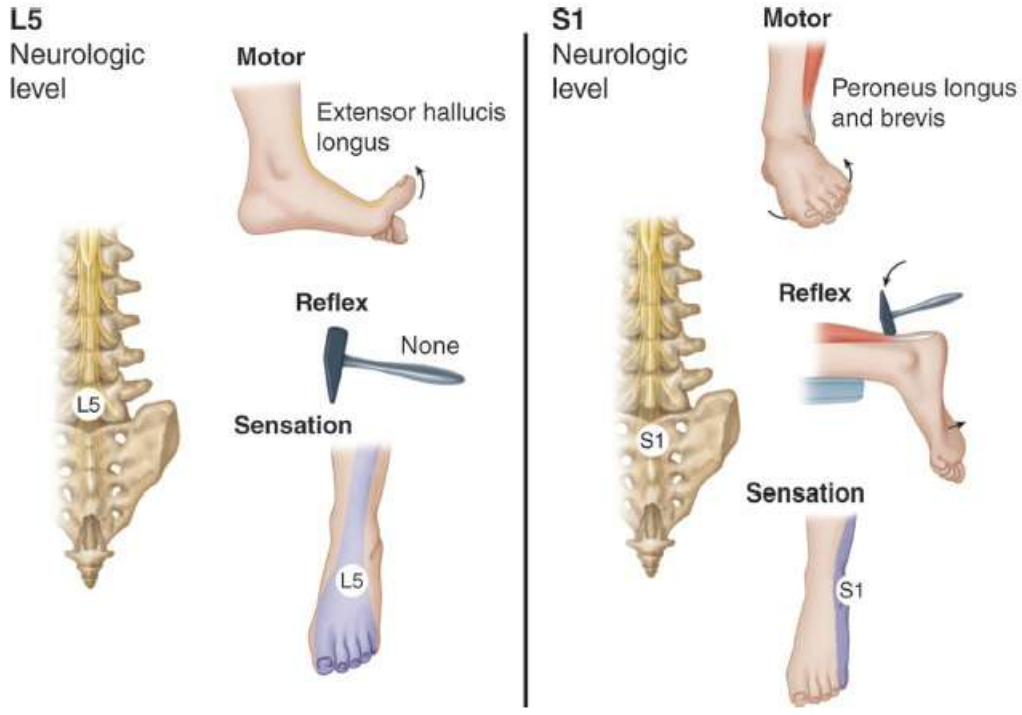


FIGURE 28.6 The main motor, sensory and reflex features of the nerve roots L5 and S1

L3:

- femoral stretch test (prone, flex knee, extend hip)
- motor: extension of knee
- sensation: anterior thigh
- reflex: knee jerk (L3, L4)

L4:

- motor: resisted inversion foot
- sensation: inner border of foot to great toe
- reflex: knee jerk

L5:

- motor: walking on heels, resisted extension great toe
- sensation: middle three toes (dorsum)
- reflex: nil

S1:

- sensation: little toe, most of sole
- reflex: ankle jerk (S1, S2)

Other examination

The method of examining the sacroiliac and hip joints is outlined in CHAPTER 54 .

Investigations

Investigations for back pain can be classified into three broad groups: front-line screening tests; specific disease investigations; and procedural and preprocedural tests.

Plain X-rays of the lumbar spine are not recommended in acute non-specific low back pain (pain <6 weeks) in the absence of ‘red flags’ as they are of limited diagnostic value and no benefits in physical function are observed.[1] Even when a red flag is present, strictly adhering to X-raying causes problematic false-positive results; for example, night pain is present in 44% of cases, yet

less than 1% have serious pathology.[6]

Screening tests

These are most important for the patient presenting with chronic back pain, especially in the presence of ‘red flags’, when serious disease such as malignancy, osteoporosis, infection or spondyloarthropathy must be excluded. The screening tests for chronic pain are:

- plain X-ray
- urine examination (office dipstick)
- FBE; ESR/CRP
- serum alkaline phosphatase
- PSA in males >50 years

Specific disease investigation

Such tests include:

- peripheral arterial studies
- HLA-B_{27} antigen test for ankylosing spondylitis and reactive arthritis
- serum electrophoresis for multiple myeloma (paraprotein)
- PSA for possible prostate cancer
- *Brucella* agglutination test
- blood culture for pyogenic infection and bacterial endocarditis
- bone scanning to demonstrate inflammatory or neoplastic disease and infections (e.g. osteomyelitis) before changes are apparent on plain X-ray
- tuberculosis studies
- X-rays of shoulder and hip joint
- electromyographic (EMG) studies to screen leg pain and differentiate neurological diseases from nerve compression syndromes
- radioisotope scanning
- technetium pyrophosphate scan of SIJ for ankylosing spondylitis

- selective anaesthetic block of facet joint under image intensification
- selective anaesthetic block of medial branches of posterior primary rami and other nerve roots

Procedural and preprocedural diagnostic tests

These tests should be kept in reserve for red flag pointers to chronic disorders, especially mechanical disorders, that remain undiagnosed and unabated, and where surgical intervention is planned for a disc prolapse requiring removal.

Depending on availability and merit, such tests include:

- CT scan
- myelography or radiculography
- discography
- MRI

Summary of diagnostic guidelines for spinal pain

- Continuous pain (day and night): think neoplasia or infection.
- The big primary malignancy is multiple myeloma.
- The big three metastases are from lung, breast and prostate.
- The other three metastases are from thyroid, kidney/adrenal and melanoma.
- Pain with standing/walking (relief with sitting) = spondylolisthesis.
- Pain (and stiffness) at rest, relief with activity = inflammation.
- In a young person with inflammation, think of ankylosing spondylitis.
- Stiffness at rest, pain with or after activity, relief with rest = osteoarthritis.
- Pain provoked by activity, relief with rest = mechanical dysfunction.
- Pain in bed at early morning = inflammation, depression or malignancy/infection.
- Pain in periphery of limb = discogenic → radicular or vascular → claudication or spinal canal stenosis → claudication.
- Pain in calf (ascending) with walking = vascular claudication.
- Pain in buttock (descending) with walking = neurogenic claudication.

- One disc lesion = one nerve root (exception is L5–S1 disc).
- One nerve root = one disc (usually).
- Two or more nerve roots—consider neoplasm.
- The rule of thumb for the lumbar nerve root lesions is L3 from L2–3 disc, L4 from L3–4, L5 from L4–5 and S1 from L5–S1.
- A large disc protrusion can cause bladder symptoms, either incontinence or retention.
- A retroperitoneal bleed from anticoagulation therapy can give intense nerve root symptoms and signs.

Back pain in children

The common mechanical disorders of the intervertebral joints can cause non-specific back pain in children, which must always be taken seriously. Like abdominal pain and leg pain, it can be related to psychogenic factors, so this possibility should be considered by diplomatically evaluating problems at home, at school or with sport. It is helpful to remember that tight hamstrings are associated with non-specific back discomfort with poor forward flexion.

Especially in children under the age of 10, it is very important to exclude organic disease. Infections such as osteomyelitis and tuberculosis are rare possibilities, and ‘discitis’ has to be considered. This painful condition can be idiopathic, but can also be caused by the spread of infection from a vertebral body. It has characteristic radiological changes.

Tumours causing back pain include the benign osteoid osteoma and the malignant osteogenic sarcoma. Osteoid osteoma is a very small tumour with a radiolucent nucleus that is sharply demarcated from the surrounding area of sclerotic bone. Although more common in the long bones of the leg, it can occur in the spine.

In older children and adolescents (in whom back pain is common) the organic causes of back pain are more likely to be inflammatory, congenital or from developmental anomalies and trauma.

A prolapsed intervertebral disc, which can occur in adolescents, can be very unusual in its presentation. There is often marked spasm, with a stiff spine and lateral deviation, which may be out of proportion to the relatively lower degree of pain.

Other important conditions to consider are Scheuermann disease (which largely affects thoracic spine) and early-onset ankylosing spondylitis.

Spondylolisthesis can occur in older children, usually due to a slip of L5 or S1, because the articular facets are congenitally absent or because of a stress fracture in the pars interarticularis. It is necessary to request standing lateral and oblique X-rays.

Consider a lumbar stress fracture in the adolescent, which can be caused by activity involving rotation and extension of the spine such as cricket fast bowling. It has an insidious onset and requires a high index of suspicion.

Back pain in the older person

Mechanical spinal dysfunction is still the most common cause of back pain in the elderly and may represent a recurrence of earlier dysfunction. It is amazing how commonly disc prolapse and facet joint injury can present in the aged. However, degenerative joint disease is very common and, if advanced, can present as spinal stenosis with claudication and nerve root irritation due to narrowed intervertebral foraminae.

Special problems to consider are malignant disease, degenerative spondylolisthesis, vertebral pathological fractures and occlusive vascular disease.

Acute back and leg pain due to vertebral dysfunction

Mechanical disruption of the vertebral segment or segments is the foremost cause to consider, while the main serious clinical syndromes are secondary to disruption with or without prolapse of the intervertebral disc, usually L4–5 or L5–S1.

TABLE 28.4 presents the general clinical features and diagnosis in acute back pain (fractures excluded) following vertebral dysfunction: the symptoms and signs can occur singly or in combination.

Table 28.4 Clinical features and diagnosis of vertebral dysfunction leading to low back and leg pain[10]

Clinical features	Frequency	Diagnosis
Syndrome A (surgical emergency) Saddle anaesthesia (around anus, scrotum or vagina) Distal anaesthesia Evidence of UMN or LMN lesion Loss of sphincter control or urinary retention Progressive weakness of legs peripherally and areflexia (often bilateral)	Very rare	Spinal cord (UMN) or cauda equina (LMN) compression (FIG. 28.7)

Syndrome B (probable surgical emergency) Anaesthesia or paraesthesia of the leg Foot drop Motor weakness Absence of reflexes	Uncommon	Large disc protrusion, paralysing nerve root
Syndrome C Distal pain with or without paraesthesia Radicular pain (sciatica) Positive dural stretch tests	Common	Posterolateral disc protrusion on nerve root or disc disruption
Syndrome D Non-specific lumbar pain (unilateral, central or bilateral) ± Buttock and posterior thigh pain	Very common	Disc disruption or facet dysfunction or unknown (non-specific) causation

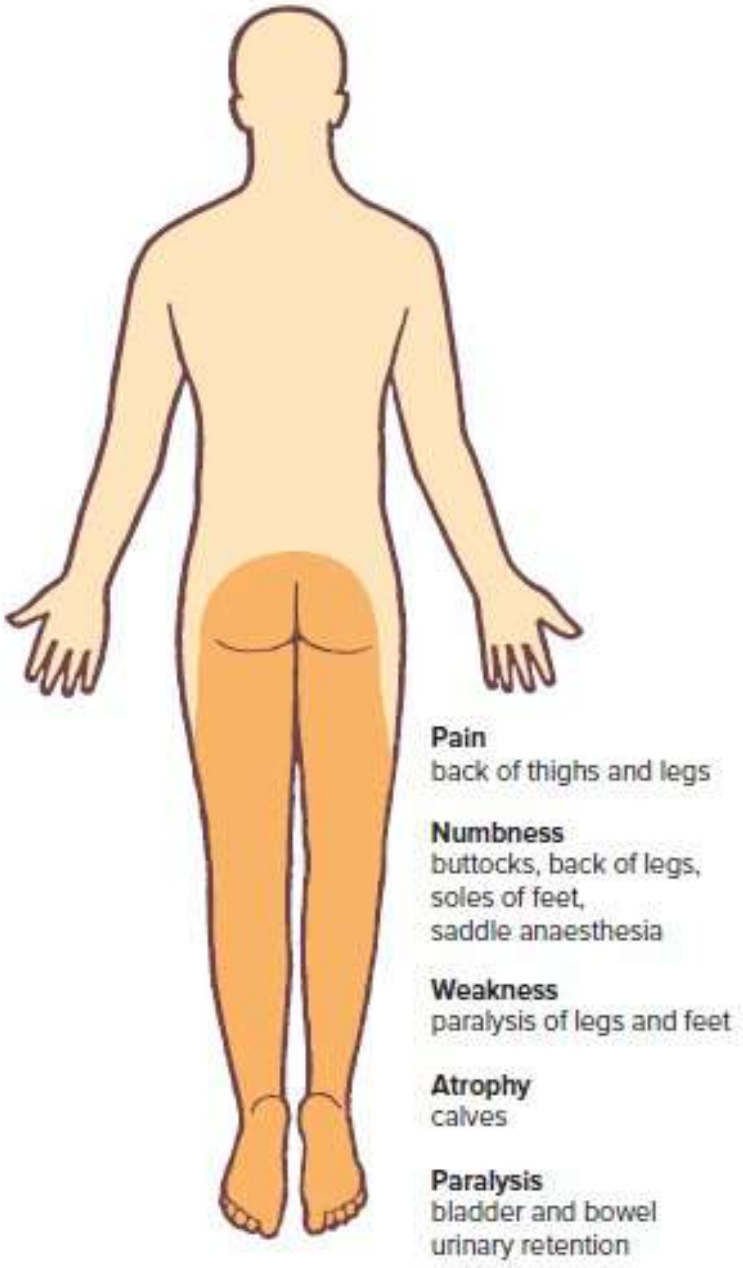


FIGURE 28.7 Cauda equina syndrome due to massive prolapsed intervertebral disc

Fortunately, syndromes A and B are extremely rare, but, if encountered, urgent referral to a surgeon is mandatory. Clinical features of the cauda equina syndrome are presented in FIGURE 28.7 . Syndrome B can follow a bleed in those taking anticoagulant therapy or be caused by a disc sequestration after inappropriate spinal manipulation.

Vertebral dysfunction with non-radicular pain (non-

specific back pain)

This outstanding common cause of low back pain is considered to be due mainly to dysfunction of the pain-sensitive facet joint. The precise pathophysiology is difficult to pinpoint.

Typical profile[9]

Age	Any age—late teens to old age, usually 22–55 years
History of injury	Yes, lifting or twisting
Site and radiation	Unilateral lumbar (may be central), refers over sacrum, SIJ areas, buttocks
Type of pain	Deep aching pain, episodic
Aggravation	Activity, lifting, gardening, housework (vacuuming, making beds, etc.)
Relief	Rest, warmth
Associations	May be stiffness, usually good health
Physical examination (significant)	Localised tenderness—unilateral or central L4, L5 or S1 levels, may be restricted flexion, extension, lateral flexion
Diagnosis confirmation	Investigation, which is usually inappropriate, invariably normal (or false positive)

Note: Diagnosis made clinically.

Management[1,8,9]

- Activity directed by degree of pain but normal activity encouraged from outset
- Reassure that it is rarely serious and usually settles in time
- Back education program
- Analgesics—paracetamol
- Consider short-term oral NSAIDs (particularly if inflammatory pattern)
- Exercise program and swimming (as tolerated)—conflicting evidence re efficacy
- Physical therapy—mobilisation, manipulation (for persistent problems, but conflicting evidence) (see later in this chapter)

Current evidence for acute low back pain can be summarised as follows:[4,8,10]

- beneficial—advice to stay active and reassurance, NSAIDs
- likely to be beneficial—analgesics and stretching (reduces period of morbidity)
- lacking firm evidence—spinal mobilisation/manipulation,[11] back exercises, trigger point injections, acupuncture

For chronic low back pain (pain >12 weeks):

- beneficial—back exercises, multidisciplinary treatment program
- possible benefit—weight loss, analgesics, NSAIDs, trigger point injections, spinal mobilisation/manipulation

Radiculopathy

Radicular pain, caused by nerve root compression from a disc protrusion (most common cause), tumour or narrowed intervertebral foramina typically produces pain in the leg related to the dermatome and myotome innervated by that nerve root. Leg pain may occur alone without back pain and vary considerably in intensity.

The two nerve roots that account for most of these problems are L5 and S1, and the commonest disc lesion is L4–5, closely followed by L5–S1. A disc can be confined, extruded or sequestrated. Most settle with time (6–12 weeks). The management is outlined at the end of this chapter and under ‘Sciatica’ (see CHAPTER 55).

Spondylolisthesis

About 5% of the population have spondylolisthesis but not all are symptomatic. The pain is caused by extreme stretching of the interspinous ligaments or of the nerve roots. The onset of back pain in many of these people is due to concurrent disc degeneration rather than a mechanical problem. The pain is typically aggravated by prolonged standing, walking and exercise. The physical examination is quite diagnostic.

- Physical examination (significant): stiff waddling gait, increased lumbar lordosis, flexed knee stance, tender prominent spinous process of ‘slipped’ vertebrae, limited flexion, hamstring tightness or spasm
- Diagnosis confirmation: lateral X-ray (standing) (see FIG. 28.8)

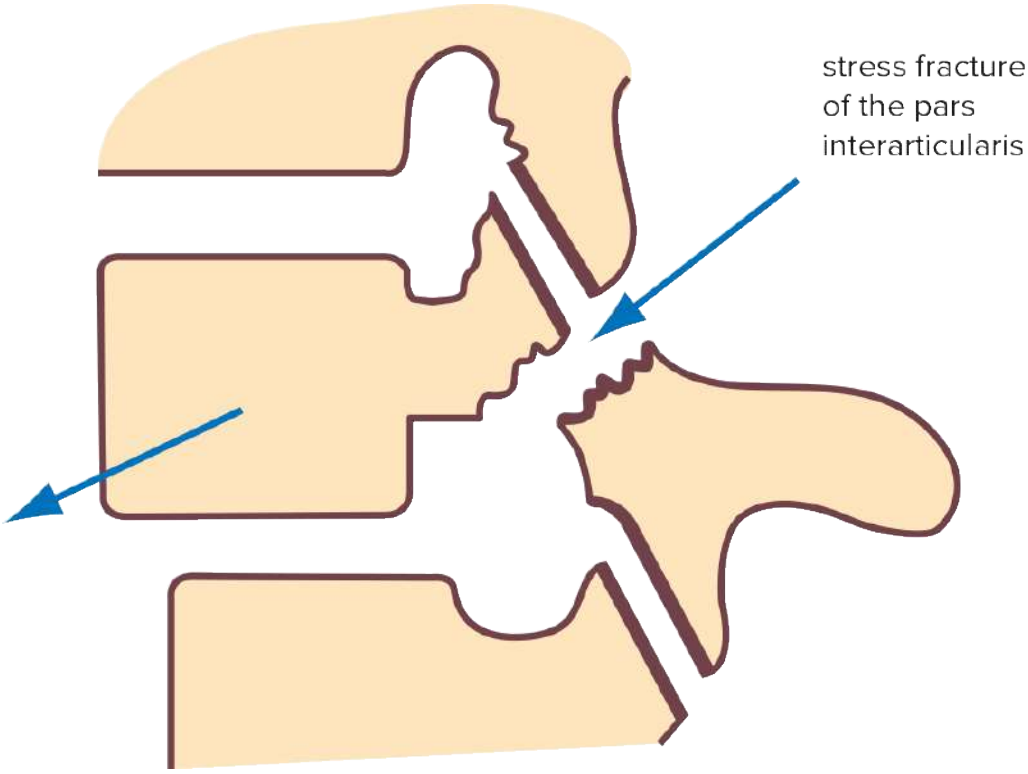


FIGURE 28.8 Spondylolisthesis: illustrating a forward shift of one vertebra on another

Management

This instability problem can be alleviated with relief of symptoms by getting patients to follow a strict flexion exercise program for at least 3 months. The objective is for patients to ‘splint’ their own spine by strengthening the abdominal and spinal muscles.

Extension of the spine should be avoided, especially hyperextension. Gravity traction might help. Recourse to lumbar corsets or surgery (for spinal fusion) should be resisted, although it is appropriate in a few severe intractable cases.

Lumbar spondylosis

Lumbar spondylosis, also known as degenerative osteoarthritis or osteoarthrosis, is a common problem of wear and tear that may follow vertebral dysfunction, especially after severe disc disruption and degeneration.

Stiffness of the low back is the main feature of lumbar spondylosis. Although most people live with and cope with the problem, progressive deterioration can occur, leading to subluxation of the facet joints. Subsequent narrowing of the spinal and intervertebral foramen leads to spinal canal stenosis (see FIG. 28.9).

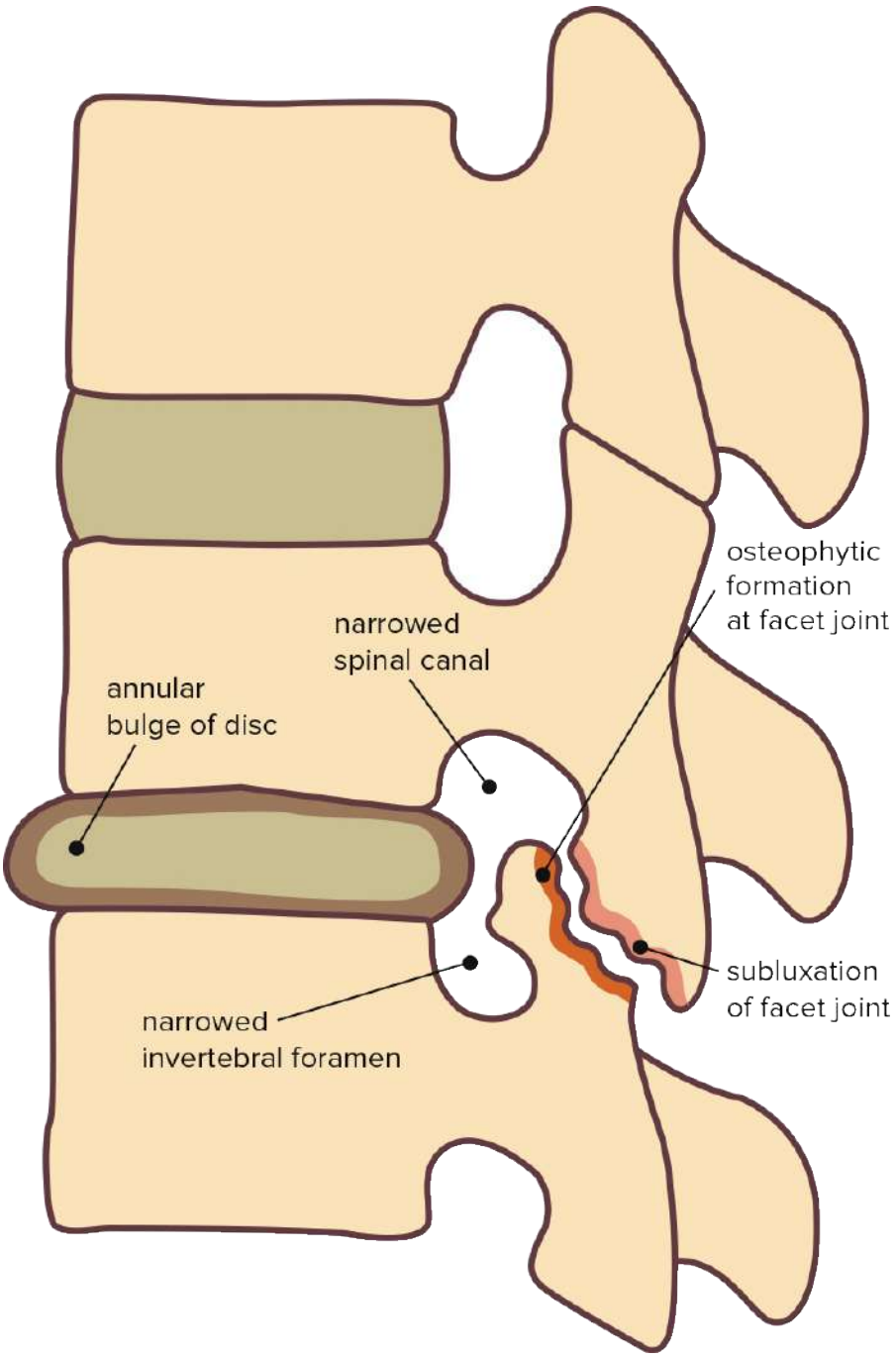


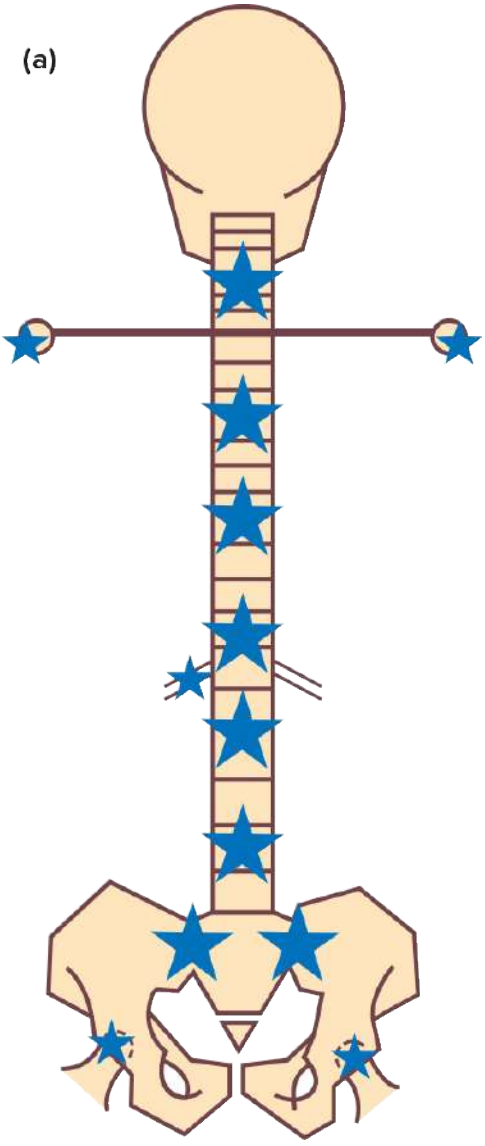
FIGURE 28.9 Lumbar spondylosis with degeneration of the disc and facet joint, leading to narrowing of the spinal canal and intervertebral foramen (spinal canal stenosis)

Management

- Basic analgesics (depending on patient response and tolerance)
- NSAIDs (judicious use)
- Appropriate balance between light activity and rest
- Exercise program and hydrotherapy (if available)—physiotherapy supervision
- Regular mobilisation therapy may help
- Consider trials of electrotherapy such as TENS
- Consider decompressive surgery for spinal canal stenosis (see CHAPTER 55)

The spondyloarthritides

The seronegative spondyloarthropathies are a group of disorders characterised by involvement of the sacroiliac joints with an ascending spondylitis and extraspinal manifestations, such as oligoarthritis and enthesopathies (see FIG. 28.10 ; refer to CHAPTER 25). The pain and stiffness that are the characteristic findings of spinal involvement are typical of inflammatory disease: namely, worse in the morning, may occur at night and improves rather than worsens with exercise.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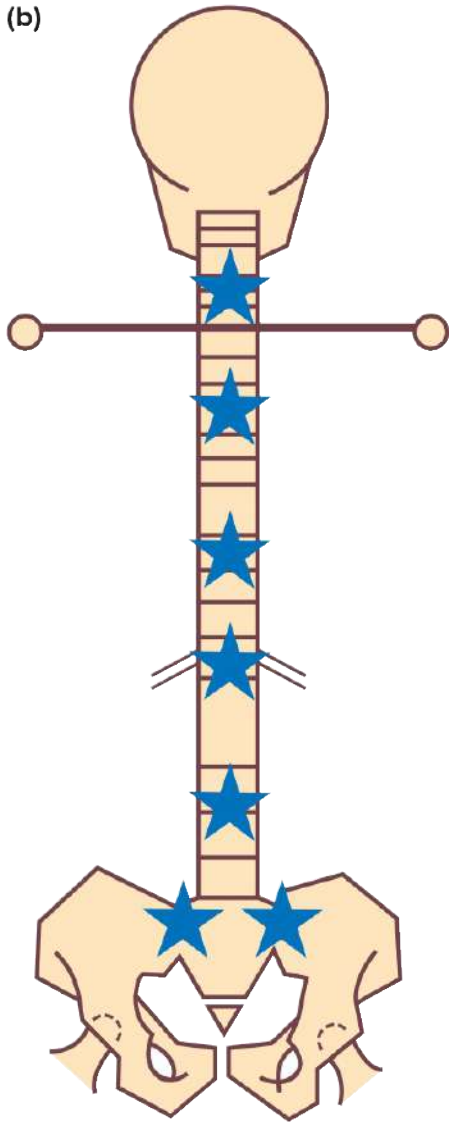

FIGURE 28.10 **(a)** Ankylosing spondylitis and psoriasis: main target areas on vertebral column and girdle joints and ribs, **(b)** Crohn disease and ulcerative colitis: main target areas of enteropathies. Reactive arthritis targets the lumbar spine and sacroiliac joints only.

The main disorders in this group are ankylosing spondylitis, psoriatic arthritis, reactive arthritis and the inflammatory bowel diseases. Hence the importance of searching for a history of psoriasis, diarrhoea, urethral discharge, eye disorders and episodes of arthritis in other joints.

Treatment

The earlier the treatment, the better the outlook; the prognosis is usually good (see earlier in chapter). Refer to consultant for shared care. The basic objectives of treatment are:

- prevention of spinal fusion in a poor position
- relief of pain and stiffness
- maintenance of optimum spinal mobility

Malignant disease

It is important to identify malignant disease and other space-occupying lesions as early as possible because of the prognosis and the effect of a delayed diagnosis on treatment.

With respect to the neurological features, more than one nerve root may be involved and major neurological signs may be present without severe root pain. The neurological signs will be progressive.

If malignant disease is proved and myeloma is excluded, a search should be made for the six main primary malignancies that metastasise to the spine (see FIG. 28.11). If the bone is sclerotic, consider prostatic secondaries, some breast secondaries or Paget disease.

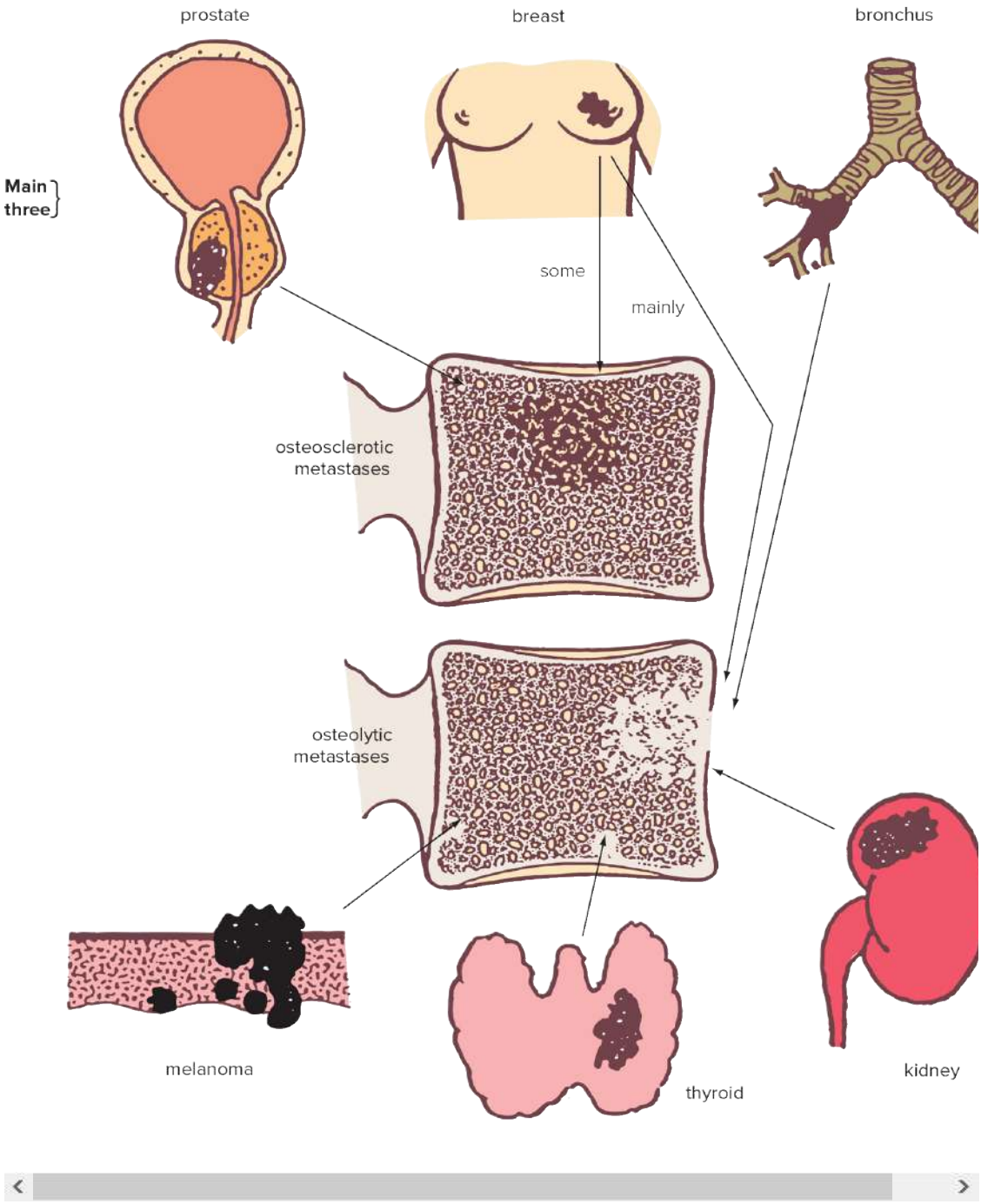

FIGURE 28.11 Important primary malignancies metastasising to the spine. Note the difference between sclerotic and osteoporotic metastases; multiple myeloma also causes osteoporotic lesions.

Non-organic back pain

Like headache, back pain is a symptom of an underlying functional, organic or psychological

disorder. Preoccupation with organic causation of symptoms may lead to serious errors in the assessment of back pain. Any vulnerable aching area of the body is subject to aggravation by emotional factors.

Depressed people are generally less demonstrative than those with extreme anxiety and conversion disorders and malingerers, so it is easier to mistakenly overlook the non-organic basis for their problem.

Where relevant, a trial of antidepressants for a minimum of 3 weeks can be reasonable, and quite often a positive response with relief of backache eventuates.

Failure to consider psychological factors in the assessment of low back pain may lead to serious errors in diagnosis and management. Each instance of back pain poses a stimulating exercise in differential diagnosis. A comparison of organic and non-organic features is presented in TABLE 28.5 .

Table 28.5 Comparison of general clinical features of organic and non-organic based low back pain[9]

Symptoms	Organic disorders	Non-organic disorders
Presentation	Appropriate	Often dramatic
Pain	Localised	Bilateral/diffuse Sacrococcygeal
Pain radiation	Appropriate Buttock, specific sites	Inappropriate Front of leg/whole leg
Time pattern	Pain-free times	Constant, acute or chronic
Paraesthesia/anaesthesia	Dermatomal Points with finger	May be whole leg Shows with hands
Response to treatment	Variable Delayed benefit	Patient often refuses treatment Initial improvement (often dramatic) then deterioration (usually within 24 hours)
Signs		

Observation	Appropriate Guarded	Overreactive under scrutiny Inconsistent
Tenderness	Localised to appropriate level	Often inappropriate level Withdraws from probing finger
Spatial tenderness (Magnuson)	Consistent	Inconsistent
Active movements	Specific movements affected	Often all movements affected
Axial loading test	No back pain (usually)	Back pain
SLR ‘distraction’ test	Consistent	Inconsistent
Sensation	Dermatomal	Non-anatomical ‘sock’ or ‘stocking’
Motor	Appropriate myotome	Muscle groups (e.g. leg ‘collapses’)
Reflexes	Appropriate May be depressed	Brisk hyperactive

Assessment of the pain demands a full understanding of the patient. One must be aware of his or her type of work, recreation, successes and failures; and one must relate this information to the degree of incapacity attributed to the back pain.

Patients with psychogenic back pain, especially the very anxious, tend to overemphasise their problem. They are usually demonstrative, the hands being used to point out various painful areas almost without prompting. There is diffuse tenderness even to the slightest touch and the physical disability is out of proportion to the alleged symptoms. The pain distribution is often atypical of any dermatome and the reflexes are almost always hyperactive. It must be remembered that patients with psychogenic back pain—for example, depression and conversion disorders—do certainly experience back pain and they do not fall for the traps set for the malingerer. A validated risk stratification tool such as the STarT Back screening tool (SBST) can be used to quantify psychosocial risk for levels of pain or disability as low, medium or high in order to guide prognosis and treatment.[10]

Treatment options for back pain

General aspects of management[1,8]

The aim of treatment is to reduce pain, maintain function, and minimise disability and work absenteeism and importantly the risk of chronicity.

Advice to stay active. Evidence from randomised controlled trials confirms that, in people with acute low back pain, advice to stay active speeds symptomatic recovery, reduces chronic disability and results in less time off work compared with bed rest or usual care.[8] Encourage the person to stay at work or return early if possible. Keep moving despite discomfort.[9]

The caring knowledgeable therapist. Evidence supports the positive value of education and reassurance from a confident, supportive and knowledgeable therapist.

Patient education. Appropriate educational material leads to a clear insight into the causes and aggravation of the back disorder plus coping strategies. This can be a component of cognitive behaviour therapy (if considered appropriate).

Heat. Thermography is beneficial. Heat in the form of heat bags, hot flannels and similar methods can be of benefit, especially in the first 2–4 weeks of acute low back pain. Small trials using cold applications were equivocal.[12] Evidence shows that heat is more effective than placebo for pain relief. [8]

Exercises. An early graduated exercise program as soon as the acute phase settles has reasonable evidence supporting it in primary care.[13] All forms of exercise (extension, flexion and isometric) appear to be equally effective (see FIG. 28.12). Supervised swimming is an excellent activity for back disorders. Physiotherapist supervision is optimal.

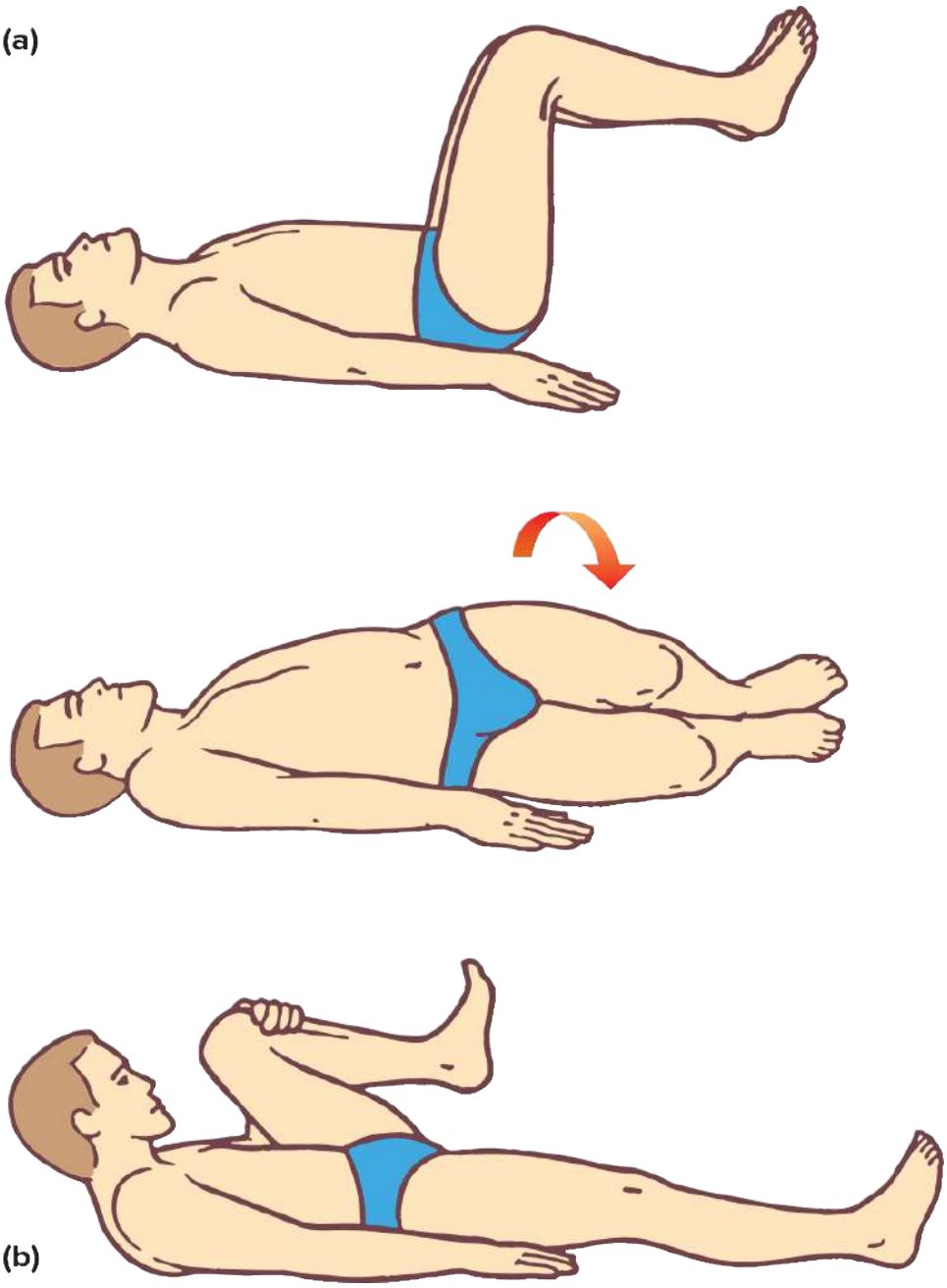

FIGURE 28.12 Examples of exercises for low back pain: **(a)** rotation exercise, **(b)** flexion exercise

Studies support the use of exercises for chronic back pain rather than acute pain.[4]

Pharmacological agents

Basic analgesics[14]

Evidence indicates that paracetamol is ineffective for non-specific low back pain.[15] However, individual patients may and do experience a benefit and, because of that and its safety profile, a trial of paracetamol may be considered if NSAIDs are contraindicated or not effective.[8]

NSAIDs[14]

NSAIDs fare somewhat better than paracetamol in systematic reviews, showing some improvements in pain and disability compared to placebo.[16] They may be particularly useful where there is clinical evidence of inflammation, especially with the spondyloarthropathies, severe spondylosis and in acute radicular pain, to counter irritation on the nerve root. The various different NSAIDs (including COX-2 inhibitors) appear to have roughly equal efficacy.

Muscle relaxants

Muscle relaxants (benzodiazepines, e.g. diazepam, or baclofen) are effective in the management of non-specific low back pain.[17] However, the common adverse effects (sedation, neurological, addiction) require that they be used with caution and for short periods only.

Opioids[8,14]

The role of opioids is limited because any pain-control benefits are outweighed by the potential risks. They are not recommended but may be considered when paracetamol and NSAIDs are not recommended or are unable to provide pain control.

Injection techniques

Trigger point injection

There is limited evidence (e.g. a trial injecting the most painful part of the medial iliac crest) that local anaesthetic injections may be effective for relatively isolated points using 5–8 mL of local anaesthetic.[18]

Chymopapain

This enzyme has been advocated for the treatment of acute nuclear herniation that is still intact. The indications are similar for surgical discectomy. However, studies show that although it is more effective than placebo, it is less effective than surgical discectomy.[19]

Facet joint injection

Corticosteroid injection under radio-image intensification is widely used in some clinics. The procedure is delicate and expertise is required. Best evidence to date does not support the use of these injections.[18]

Epidural injections

Injections of local anaesthetic with or without corticosteroids are sometimes used for chronic pain, especially for nerve root pain. The most recent Cochrane review indicates that corticosteroids don't differ from placebo injections, and local anaesthetic agents have not been studied alongside placebo.[18] If chosen, a reasonable option is the caudal (trans-sacral) epidural injection for persistent sciatica using 15 mL of half-strength local anaesthetic only (e.g. 0.25% bupivacaine) (see FIG. 28.13).

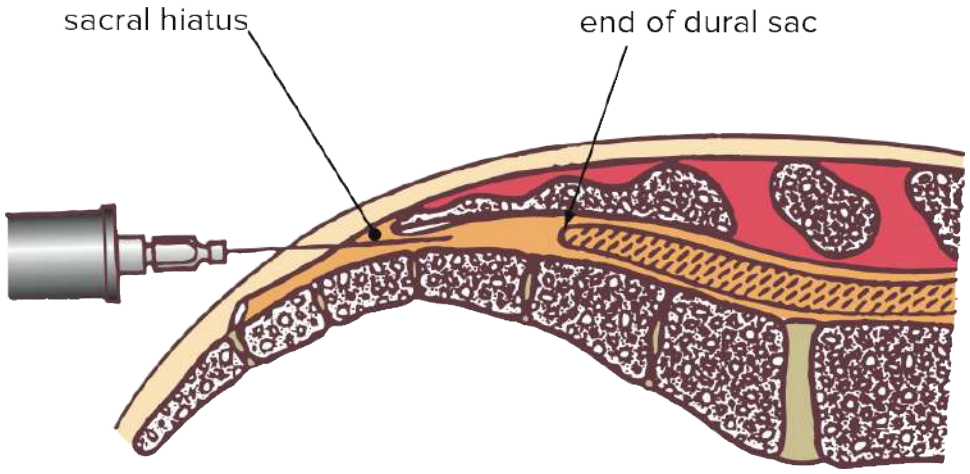

FIGURE 28.13 Caudal epidural injection: the needle should lie free in the space and be well clear of the dural sac

Physical therapy

Active exercises are the best form of physical therapy (see FIG.28.12a, b).

Passive spinal stretching at the end range is a safe, effective method (see FIG. 28.14). Spinal mobilisation is a gentle, repetitive, rhythmic movement within the range of movement of the joint. It is safe and modestly effective, and a variation of stretching.

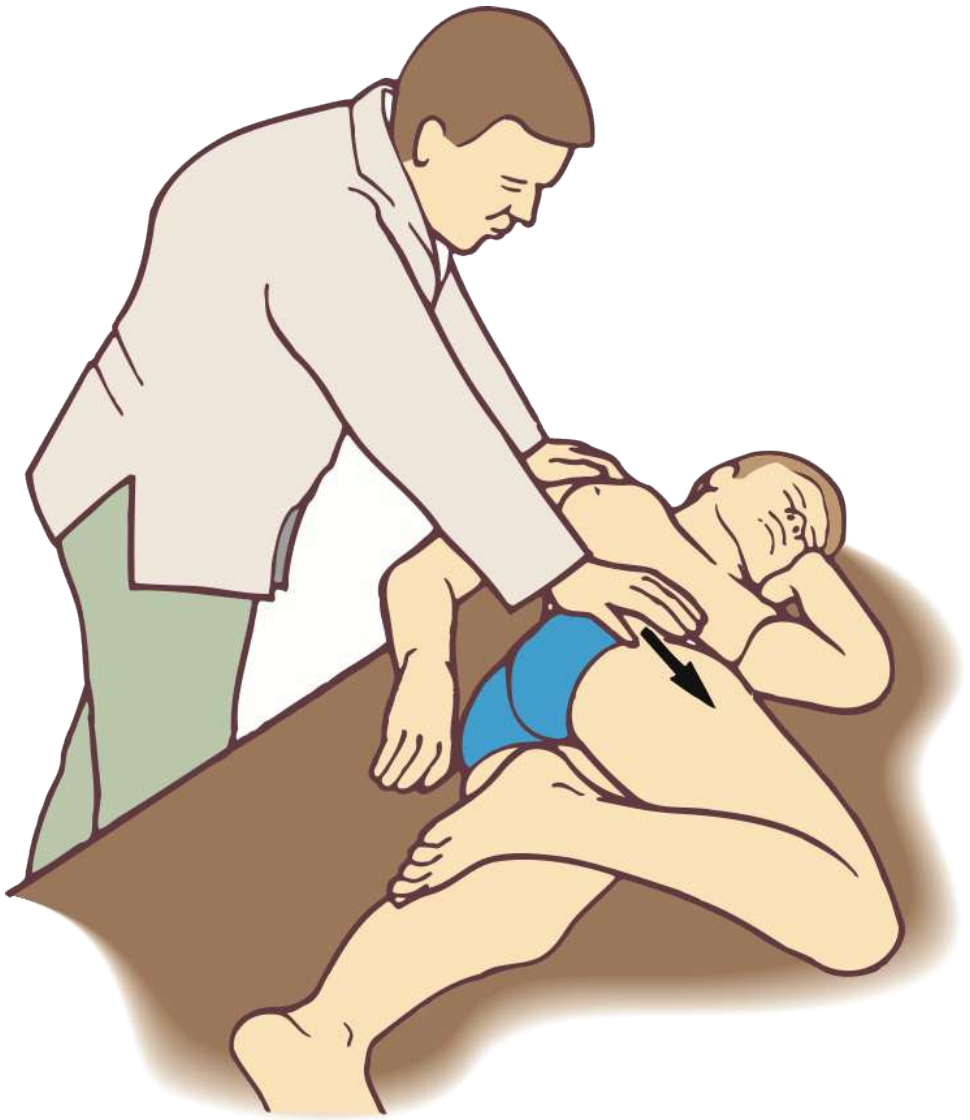

FIGURE 28.14 Lumbosacral spinal stretching technique (for right-sided pain): a traditional technique. Illustration shows direction of line of force.

Source: Reproduced with permission from C Kenna and J Murtagh. *Back Pain and Spinal Manipulation*. Sydney: Butterworths, 1989

Spinal manipulation is a high-velocity thrust at the end range of the joint. It seems to produce a faster response, but requires greater skill. Evidence is conflicting, but it may possibly be effective for uncomplicated dysfunctional low back pain (without radicular pain), especially acute pain (see FIG. 28.15).[4,8] In chronic low back pain, regular spinal manipulation results in a slight improvement at one month that disappears by six months.[20] Adverse effects are uncommon, but can be serious.

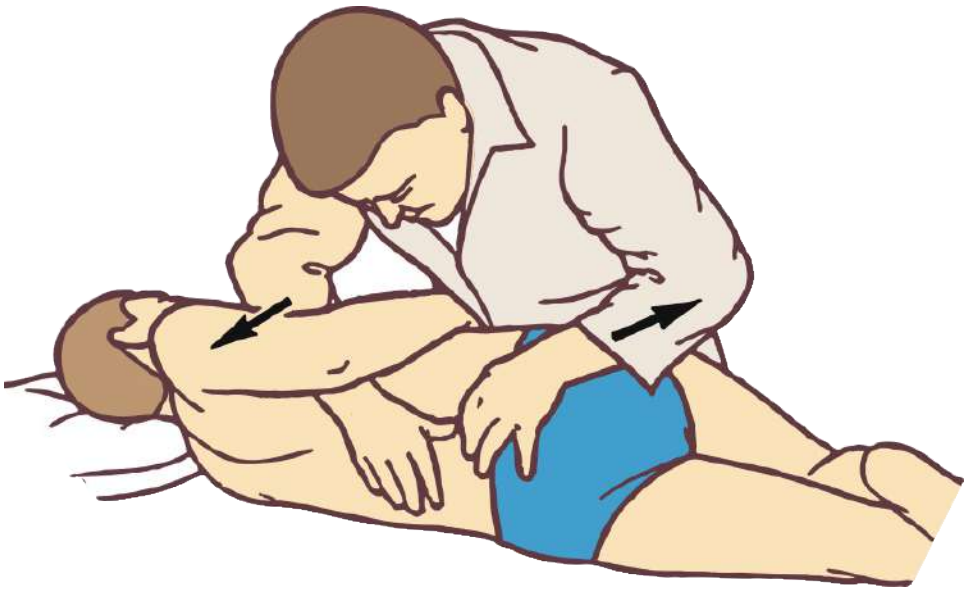

FIGURE 28.15 Lumbar spinal stretching manipulation: illustration of the specific technique for the L4–5 level with arrows indicating the direction of applied force

Source: Reproduced with permission from C Kenna and J Murtagh. *Back Pain and Spinal Manipulation*. Sydney: Butterworths, 1989

Other treatments

Because back pain is so common, so frustrating and seemingly so resistant to most treatments except the passage of time, there is a vast array of suggested therapies that are not supported by independent evidence. Few have been 'proven' not to work (that proof takes enormous effort) but the GP who advocates them should be aware they are relying on anecdote more than science. Where benefits are marginal or non-existent, it is important to preference safer, cheaper interventions over those that require significant time, money or risk.

- Hydrotherapy
- Traction (little or no impact)[21]
- TENS
- Therapeutic ultrasound
- Facet joint injection
- Posterior nerve root (medial branch) blocks with or without denervation (by cryotherapy or radiofrequency)
- Percutaneous vertebroplasty (injection of bone cement into fractured vertebra of osteoporosis)

- Deep friction massage (in conjunction with mobilisation and manipulation)
- Acupuncture (acute: no evidence, chronic: evidence of short-term relief)[22]
- Biofeedback
- Gravitational methods (home therapy)

- Lumbar supports (don't prevent pain; conflicting studies on relieving pain)[23]

Management guidelines for lumbosacral disorders (summary)

The management of 'mechanical' back pain depends on the cause. Since most of the problems are mechanical and there is a tendency to natural resolution, conservative management is quite appropriate. The rule is: 'if patients with uncomplicated back pain receive no treatment, one-third will get better within 1 week and by 3 weeks almost all the rest of the other two-thirds are better'.[24,25] Practitioners should have a clear-cut management plan with a firm, precise, reassuring and conservative clinical approach.

The problems can be categorised into general conditions for which the summarised treatment protocols are outlined.

- Acute pain = pain less than 6 weeks
- Subacute pain = pain 6–12 weeks
- Chronic pain = pain greater than 12 weeks

Acute low back pain[8]

The common problem of low back pain caused by facet joint dysfunction and/or limited disc disruption usually responds well to the following supportive therapy (see box). The typical patient is aged 20–55 years, is well and has no radiation of pain below the knee.[25,26]

Most of these patients can expect to be relatively pain-free in 14 days and can return to work earlier than that (some may not miss work and this should be encouraged).

Management of non-specific acute low back pain (summary)[27]

- Explanation and reassurance about no evidence of serious damage or disease; cognitive behaviour therapy

- Back education program
- Encouragement of normal daily activities, including work, and taking responsibility for own management
- Optional non-opioid analgesics (NSAIDs)
- Prescribe exercises (provided non-aggravating)
- Physical therapy: stretching of affected segment, consider spinal mobilisation or manipulation (if no contraindication)[8,14,25]
- Review in about 5 days (probably best time for consideration of physical therapy)
- No investigation needed initially

Sciatica with or without low back pain

Sciatica is a more complex and protracted problem to treat, but most cases will gradually settle within 12 weeks (refer to CHAPTER 55). Conservative management is usually recommended in the first 6–8 weeks.

Acute[8]

- Explanation and reassurance
- Back education program
- Resume normal activities as soon as possible
- Regular non-opioid analgesics with review as the patient mobilises
- NSAIDs for 10–14 days, then cease and review (low-quality evidence for mild improvement, but with side effects)[28]
- If severe pain unrelieved, add an opioid that works well for the patient, such as tapentadol SR 50 mg (o) bd as necessary, for short-term use[27]
- Walking and swimming
- Weekly or 2-weekly follow-up
- Consider: a course of corticosteroids for very severe pain,[8] e.g. prednisolone 50 mg for 5 days, then 25 mg for 5 days, gradually tapering to 3 weeks in total.

  or

30 mg daily mane for 3 weeks, tapering to 0 over next 2 weeks (efficacy not clearly established)

Chronic

- Reassurance that problem will subside (assuming no severe neurological defects)
- Consider epidural anaesthesia (if slow response)
- Explore depressive symptoms: consider amitriptyline 10–25 mg (o) nocte increasing to maximum 75–100 mg or duloxetine

Note: An important controlled prospective study comparing surgical and conservative treatment in patients with sciatica over 10 years showed that there was significant relief of sciatica in the surgical group for 1–2 years but not beyond that time. At 10 years, both groups had the same outcome, including neurological deficits.[29] Surgery has a limited role.

Sacroiliac dysfunction

See CHAPTER 55 .

Chronic back pain

The basic management of the patient with uncomplicated chronic back pain should consider the following options:

- back education program and ongoing support
- encouragement of normal activity
- exercise program
- mindfulness-based stress reduction (evidence based)
- paracetamol (e.g. 500 or 665 mg (o) 8 hourly) although not tested in any RCT
- NSAIDs for 14 days (especially if inflammation, i.e. pain at rest—relieved by activity and poor response to non-pharmacological treatment) and review
- antidepressants (but only if depressed)[30]
- trial of mobilisation or manipulation (at least three treatments)—if no contraindications[19,31] (low-quality evidence)
- consider a multidisciplinary rehabilitation team approach or a ‘back school’ (but evidence again suggests just trivial improvement)[32]

General guidelines for surgical intervention for radiculopathy

Absolute

- Bladder/bowel control disturbance; perineal sensory change
- Progressive motor disturbance (e.g. significant foot drop, weakness in quadriceps)

Relative

- Severe prolonged pain or disabling pain
- Failure of conservative treatment with persistent pain (problem of permanent nerve damage)
- If all four of the following criteria are met:[8]

  leg pain equal to or worse than back pain

  positive straight leg raise test

  no response to conservative therapy after 4–6 weeks

  imaging shows a lesion corresponding to symptoms

Prevention of further back pain

Patients should be informed that an ongoing back care program should give them an excellent outlook. Prevention includes:

- education about back care, including a good layperson's reference
- golden rules to live by: how to lift, sit, bend, play sport and so on
- an exercise program: a tailor-made program for the patient

When to refer

Urgent referral

- Myelopathy, especially acute cauda equina compression syndrome
- Severe radiculopathy with progressive neurologic deficit

- Spinal fractures

Other referrals

- Recalcitrant spinal canal stenosis
- Neoplasia or infection
- Undiagnosed back pain
- Paget disease
- Continuing pain of 3 months' duration without a clearly definable cause

Practice tips

- Back pain that is related to posture, aggravated by movement and sitting, and relieved by lying down is due to vertebral dysfunction, especially a disc disruption.
- The pain from most disc lesions is generally relieved by rest.
- Plain X-rays are of limited use, especially in younger patients, and may appear normal in disc prolapse.
- Remember the possibility of depression as a cause of back pain; if suspected, consider a trial of antidepressants.
- If back pain persists, possibly worse during bed rest at night, consider malignant disease, depressive illness or other systemic diseases.
- Pain that is worse on standing and walking, but relieved by sitting, is probably caused by spondylolisthesis.
- If pain and stiffness is present on waking and lasts longer than 30 minutes upon activity, consider inflammation.
- Avoid using strong analgesics (especially opioids) in any chronic non-malignant pain state.
- Bilateral back pain is more typical of systemic diseases, while unilateral pain typifies mechanical causes.
- Back pain at rest and morning stiffness in a young person demand careful investigation: consider inflammation such as ankylosing spondylitis and reactive arthritis.
- A disc lesion of L5–S1 can involve both L5 and S1 roots. However, combined L5

and S1 root lesions should still be regarded with suspicion (e.g. consider malignancy).

- A large central disc protrusion can cause bladder symptoms, either incontinence or retention.
- Low back pain of very sudden onset with localised spasm and protective lateral deviation may indicate a facet joint syndrome.
- The T12–L1 and L1–2 discs are the groin pain discs.
- The L4–5 disc is the back pain disc.
- The L5–S1 disc is the leg pain disc.
- Severe limitation of SLR (especially to less than 30°) indicates lumbar disc prolapse.
- A preventive program for dysfunctional back pain based on back care awareness and exercises is helpful.
- Remember that most back problems resolve within a few weeks, so avoid overtreatment.

Patient education resources

Hand-out sheets from *Murtagh's Patient Education* 8th edition:

- Backache
- Exercises for your lower back
- Sciatica
- Spondylosis

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29 Bruising and bleeding

My pa is one mask of brooses both blue and green.

CHARLES DICKENS (1812–1870), *NICHOLAS NICKLEBY*

Many people present with the complaint that they bruise easily but only a minority turn out to have an underlying blood disorder. Purpura is bleeding into the skin or mucous membranes, appearing as multiple small haemorrhages that do not blanch on pressure. Smaller purpuric lesions that are 2 mm or less in diameter (pinhead size) are termed petechiae, while larger purpuric lesions are called ecchymoses (see FIG. 29.1).

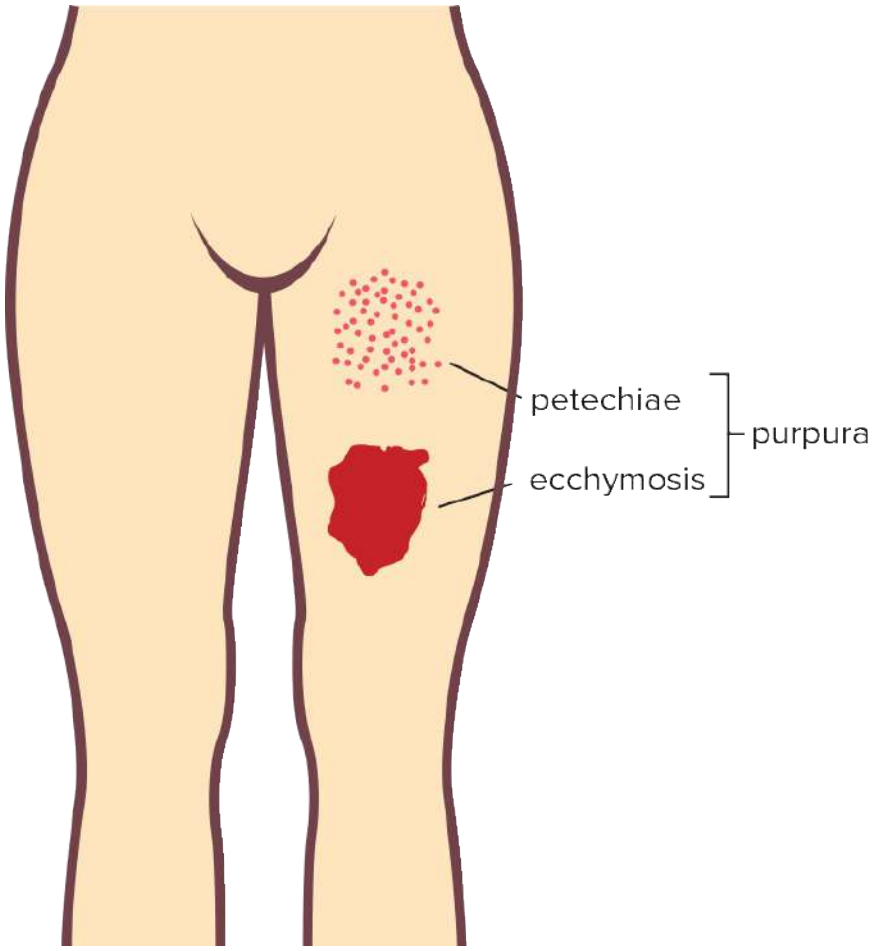

FIGURE 29.1 Purpuric rash (petechiae and ecchymoses)

Bruises are large areas of bleeding that result from subcutaneous bleeding. If bruising is abnormal and out of proportion to the offending trauma, then a disturbance of haemostasis is suggested (see FIG. 29.2). The three spontaneous, intrinsically linked pathways that arrest bleeding following injury are vasoconstriction, formation of a platelet plug and activation of coagulation factors.

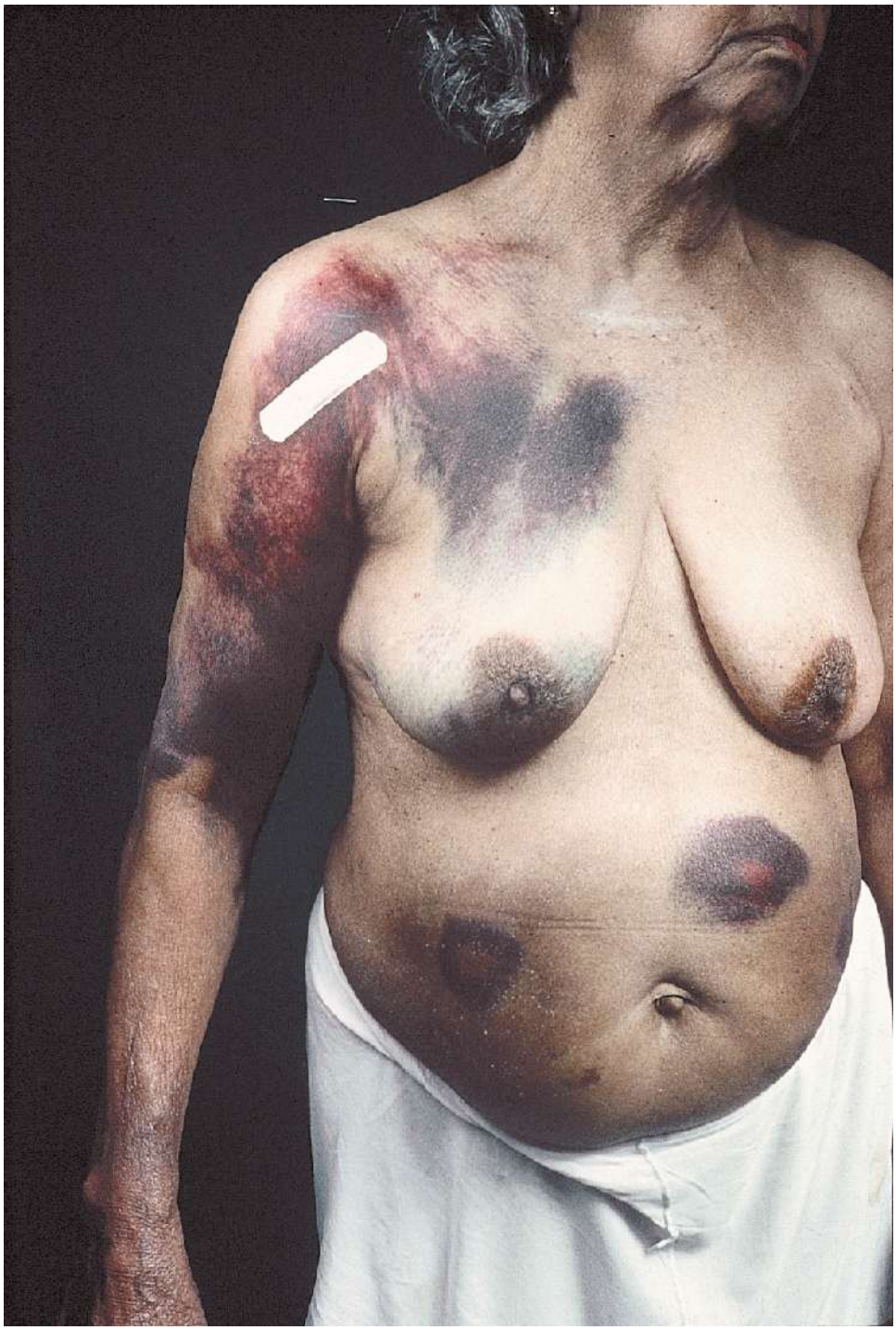

FIGURE 29.2 Severe bleeding in a woman with diabetes and systemic fibrinolysis. Note the bleeding following insulin injections into the abdominal wall and an injection into the shoulder joint.

Photo courtesy Hatem Salem

Differential diagnosis

‘Palpable purpura’ due to an underlying systemic vasculitis is an important differential problem. The petechiae are raised so finger palpation is important. The cause is an underlying vasculitis affecting small vessels (e.g. polyarteritis nodosa).

The decision as to which individuals require investigation is difficult and depends on whether the haemostatic defect is due to local or systemic pathology.[1] The ability to identify a bleeding disorder is important because of implications for surgery, pregnancy, medication and genetic counselling.

Key facts and checkpoints

- Purpura = petechiae + ecchymoses.
- Abnormal bleeding is basically the result of disorders of (1) the platelet, (2) the coagulation mechanism or (3) the blood vessel.
- There is no substitute for a good history in the assessment of bleeding disorders.
- The first step is an assessment of personal and family histories.
- When someone describes ‘bruising easily’ it is important to exclude thrombocytopenia due to bone marrow disease and clotting factor deficiencies such as haemophilia.
- The commonest cause of an acquired bleeding disorder is drug therapy (e.g. aspirin, NSAIDs, cytotoxics and oral anticoagulants).
- Bleeding secondary to platelet defects is usually spontaneous, associated with a petechial rash and occurs immediately after trauma or a cut wound.[1] The bleeding is usually mucosal (e.g. bleeding from gingiva, menorrhagia, epistaxis and petechiae).
- Bleeding caused by coagulation factor deficiency is usually traumatic and delayed (e.g. haemorrhage occurring 24 hours after a dental extraction in haemophilia).
- Laboratory assessment should be guided by the clinical impression.
- The routine screening tests for the investigation of a true bleeding disorder can occasionally be normal, even despite a severe haemorrhagic state. Second-line investigations will need to be undertaken.

Causes of clinical disorders

Bleeding disorders can result from:

- coagulation deficiencies (reduction or inhibition of circulatory coagulation factors)
- platelet abnormalities: of platelet number or function
- vascular defects: of vascular structure or endothelium

Bleeding disorders can also be divided into impaired primary or secondary haemostasis. Primary haemostatic disorders which are the most common include von Willebrand disease (vWD), thrombocytopenia and platelet function disorders. Secondary causes include disorders of fibrin formation and the haemophilias.[2]

A list of differential diagnoses of systemic bleeding disorders is presented in TABLE 29.1 .[1]

Table 29.1 Classification of bleeding disorders[1]

Vascular disorders

Inherited

- Hereditary haemorrhagic telangiectasia
- Connective tissue disease, e.g. Marfan syndrome
- Easy bruising syndrome

Acquired

- Senile purpura
- Infection, e.g. dengue, meningococcal
- Henoch-Schonlein purpura
- Corticosteroid purpura
- Vitamin C deficiency (scurvy)
- Painful bruising syndrome

Platelet disorders

Inherited

- Fanconi syndrome
- Glanzmann disease

Acquired (immune)

- Idiopathic thrombocytopenic purpura
- Aplastic anaemia
- Drug induced thrombocytopenia, e.g. heparin
- Thrombotic thrombocytopenia purpura

- Post-transfusion purpura

Non-immune

- Disseminated intravascular coagulation
- Myeloproliferative disorders
- Kidney failure/uraemia
- Bone marrow replacement (e.g. leukaemia) or failure

Coagulation disorders

Inherited

- Haemophilia A
- Haemophilia B
- von Willebrand disease (types 1, 2 and 3)

Acquired

- Disseminated intravascular coagulation (DIC)
- Vitamin K deficiency
- Oral anticoagulation therapy or overdose
- Acquired haemophilia
- Liver disease

The clinical approach

Differentiation of coagulation factor deficiencies and platelet disorders as the cause of a bleeding problem can usually be determined by a careful evaluation of the history and physical examination.

History

Factors that suggest the presence of a systemic bleeding defect include:

- spontaneous haemorrhage
- severe or recurrent haemorrhagic episodes, e.g. epistaxis
- bleeding from multiple sites, e.g. mouth, bladder, bowel
- bleeding out of proportion to the degree of trauma
- cutaneous bleeding
- gastrointestinal bleeding

- postpartum haemorrhage
- bleeding from tooth extraction/oral cavity
- menstrual history, e.g. menorrhagia
- muscle haematomas or haemarthrosis

If a bleeding diathesis is suspected it is essential to determine whether local pathology is contributing to the blood loss (e.g. postoperative bleeding, postpartum bleeding, gastrointestinal haemorrhage).

Diagnostic tips

- Platelet abnormalities present as early bleeding following trauma.
- Coagulation factor deficiencies present with delayed bleeding after initial haemostasis is achieved by normal platelets.
- A normal response to previous coagulation stresses (e.g. dental extraction, circumcision or pregnancy) indicates an acquired problem.
- If acquired, look for evidence of MILD: Malignancy, Infection, Liver disease, Drugs.
- A diagnostic strategy is outlined in TABLE 29.2 .

Table 29.2 Purpura: diagnostic strategy model

Probability diagnosis
Simple purpura (easy bruising syndrome)
Senile purpura (common on limbs of older people after minimal trauma)
Corticosteroid-induced purpura
Immune thrombocytopenic purpura
Henoch–Schönlein purpura
Liver disease, especially alcoholic cirrhosis
Increased intravascular pressure, e.g. coughing, vomiting
Serious disorders not to be missed
Malignant disease: • leukaemia • myeloma
Myelodysplasia
Aplastic anaemia

Myelofibrosis

Severe infections:

- septicaemia
- meningococcal infection
- measles
- typhoid
- dengue/chikungunya
- HIV and other blood-borne viruses (e.g. Hepatitis C)

Disseminated intravascular coagulation

Thrombotic thrombocytopenic purpura

Fat embolism

Pitfalls (often missed)

Haemophilia A, B, vWD

Post-transfusion purpura

Trauma (e.g. domestic violence, child abuse)

Rarities:

- hereditary telangiectasia (Osler–Weber–Rendu syndrome)
- Ehlers–Danlos syndrome
- scurvy
- Fanconi syndrome

Seven masquerades checklist

Drugs: many examples (see Medication record)

Anaemia:

- aplastic anaemia

Psychogenic factors

Factitial purpura

Family history

A positive family history can be a positive pointer to the diagnosis:

- sex-linked recessive pattern: haemophilia A or B
- autosomal dominant pattern: vWD, dysfibrinogenaemias
- autosomal recessive pattern: deficiency of coagulation factors V, VII and X

Enquire whether the person has noticed blood in the urine or stools and whether menorrhagia is

present in women. A checklist for a bleeding history is presented in TABLE 29.3 . The actual size and frequency of the bruises should be recorded where possible and if none are present at the time of the consultation the patient should return if any bruises reappear.

Table 29.3 Checklist for a bleeding history

Skin bruising	Tonsillectomy
Epistaxis	Other operations
Injury	Childbirth
Domestic violence	Haematuria
Menorrhagia	Rectal bleeding
Haemarthrosis	Drugs
Tooth extraction	Family history
Unusual haematomas	Comorbidities (e.g. liver disease, kidney disease)

Key questions

- How long has the problem been apparent to you?
- Do you remember any bumps or falls that might have caused the bruising?
- What sort of injuries cause you to bruise easily?
- Have you noticed bleeding from other areas such as your nose or gums?
- Have you noticed any rashes or blood blisters in your mouth?
- Has anyone in your family had a history of bruising or bleeding?
- What is your general health like?
- Do you have any tiredness, weight loss, fever or night sweats?
- Did you notice a viral illness or sore throat beforehand?
- How much alcohol do you drink?
- What happened in the past when you had a tooth extracted?
- Have you ever had painful swelling in your joints?

Note: A validated bleeding assessment tool is a useful guide to the diagnosis of vWD.

Medication record

It is mandatory to obtain a complete drug history. Examples of drugs and their responses are:

- vascular purpura:

  prednisolone/other steroids

- thrombocytopenia:

  cytotoxic drugs

  carbamazepine

  gold

  sodium valproate

  heparin

  ranitidine

  sulfonamides

  quinine, quinidine

  thiazide diuretics

  penicillins, vancomycin

  chloramphenicol

- functional platelet abnormalities:

  aspirin and other antiplatelet drugs

  NSAIDs

- coagulation factor deficiency:

  warfarin

  direct oral anticoagulants (e.g. dabigatran, rivaroxaban)

Examination

Careful examination of the skin is important. Note the nature of the bleeding and the distribution of any rash, which is characteristic in Henoch–Schönlein purpura. Senile purpura in the elderly is usually seen over the dorsum of the hands, extensor surface of the forearms and the shins.

Purpura on the legs indicates platelet disorders, meningococcal septicaemia and paraproteinaemias; on the fingers and toes indicates vasculitis.

Note the lips and oral mucosa for evidence of hereditary telangiectasia. Blood-filled vesicles of the oral mucosa (wet purpura) is a strong risk factor for intracranial haemorrhage in immune thrombocytopenic purpura and requires urgent intervention. Gum hypertrophy occurs in monocytic leukaemia. Search for evidence of malignancy, such as sternal tenderness, lymphadenopathy and hepatosplenomegaly. Examine the ocular fundi for evidence of retinal haemorrhages. Urinalysis, searching for blood (microscopic or macroscopic), is important.

Investigations

The initial choice of investigations includes a full blood count, blood film and basic coagulation studies. Basic coagulation studies include prothrombin time (PT), international normalised ratio (INR) and activated partial thromboplastin time (aPTT).

Further tests if coagulation defect suspected:

- fibrinogen level
- thrombin time (TT)

If platelet pathology suspected:

- platelet count and blood film
- platelet function analyser (PFA-100)

If inherited disorders suspected:

- factor VIII
- vW factor activity
- vW factor antigen

The full blood examination and blood film is useful in pinpointing the aetiology. Platelet morphology gives a diagnostic guide to inherited platelet disorders. Other sophisticated tests, such as von Willebrand screening and platelet aggregation (e.g. PFA-100), can be advised by the consulting haematologist. One of considerable value is the bone marrow examination, which is useful to exclude the secondary causes of thrombocytopenia, such as leukaemia, other marrow infiltrations and aplastic anaemia.

Other tests to consider: ESR/CRP, blood group, autoimmune screening, kidney function tests, LFTs, serology for blood-borne infections, ferritin, plasma electrophoresis, skin biopsy. Be cautious of pseudo-thrombocytopenia due to laboratory error or platelet clumping—exclude on a blood film and consider a repeat collection.

A summary of appropriate tests is presented in TABLE 29.4 and of blood changes for some coagulation factor deficiencies in TABLE 29.5 .

Table 29.4 Laboratory investigation checklist for the easy bruiser

Full blood count
Platelet count
Prothrombin time (PT) and international normalised ratio (INR)
Thrombin time (TT)
Activated partial thromboplastin time (aPTT)

Table 29.5 Blood changes for specific coagulation factor disorders

	Haemophilia A	vWD	Vitamin K deficiency
PT	Normal	Normal	↑
aPTT	↑	↑	↑
TT	Normal	Normal	Normal

Abnormal bleeding in children

Abnormal bleeding in children is not uncommon and once again the clinical history, particularly the past and family history, provides the most valuable information. It is important to keep non-accidental injury in mind in the child presenting with ‘easy bruising’. However, it is appropriate to exclude a bleeding disorder, especially a platelet disorder. Vigorous coughing or vomiting in a child can cause petechiae on or around the eyelids.

Coagulation disorders, including haemophilia and vWD, are usually suspected on clinical grounds because of widespread bruising or because of prolonged bleeding following procedures such as circumcision and tonsillectomy.

A common condition is haemorrhagic disease of the newborn, which is a self-limiting disease usually presenting on the second or third day of life because of a deficiency of coagulation factors dependent on vitamin K. The routine use of prophylactic vitamin K in the newborn infant has virtually eliminated this problem.

Idiopathic (immune) thrombocytopenic purpura (ITP) is the commonest of the primary platelet

disorders in children. Both acute and chronic forms have an immunological basis. The diagnosis is based on the peripheral blood film and platelet count. The platelet count is commonly below 50 000/mm^3 (50×10^9/L). Spontaneous remission within 4 to 6 weeks occurs with acute ITP in childhood.[3]

The commonest vascular defects in childhood are:

- anaphylactoid (Henoch–Schönlein) purpura
- infective states
- nutritional deficiency (usually inadequate dietary vitamin C)

Henoch–Schönlein purpura[4]

HSP, which is a type of IgA vasculitis, is the commonest vasculitis of children. It affects the small vessels, producing a leucocytoclastic vasculitis with a classic triad of non-thrombocytopenic purpura, large joint arthritis and abdominal pain. It is diagnosed clinically by the characteristic distribution of the rash (palpable purpura) over the lower limbs, extending onto the buttocks (see FIG. 29.3), but it can also involve the upper limbs, trunk and even the face.

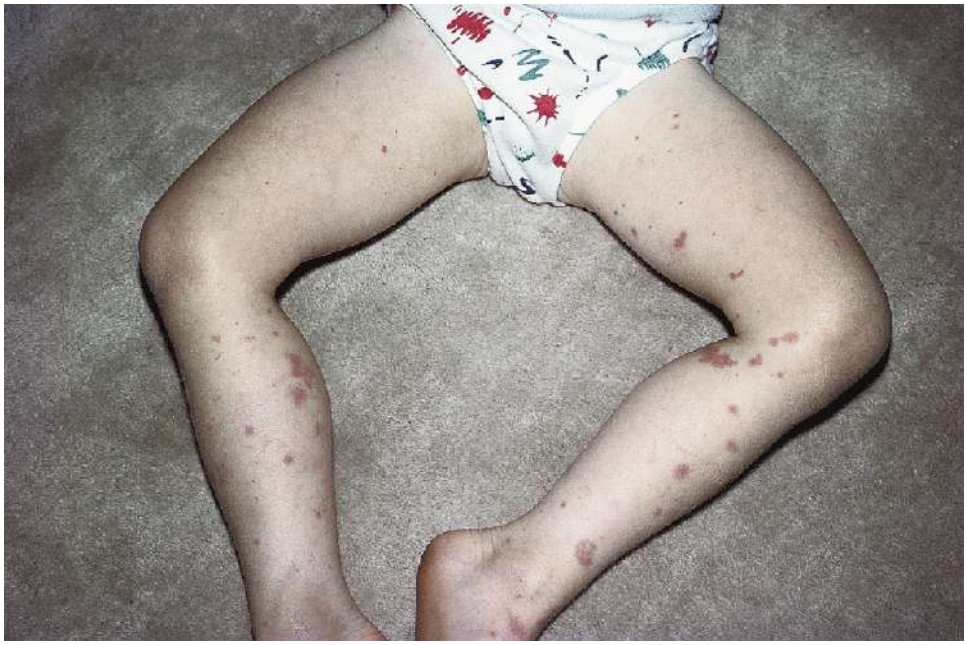

FIGURE 29.3 Henoch−Schönlein purpura in a 5-year-old boy showing the typical distribution of the rash on the lower limbs

The onset of HSP typically follows an upper respiratory tract infection including a group A streptococcal tonsillopharyngitis.

The bleeding time, coagulation time and platelet counts are normal. The prognosis is good; most recover fully in a few months.

Clinical features

- All ages, mainly in children 2–8 years
- Rash, mainly on buttocks and legs (see FIG. 29.4)[5]
- Rash can occur on hands, arms and trunk
- Arthritis (in two-thirds): mainly ankles and knees
- Abdominal pain—colicky (vasculitis of GIT)
- Haematuria (in 90%): reflects nephritis

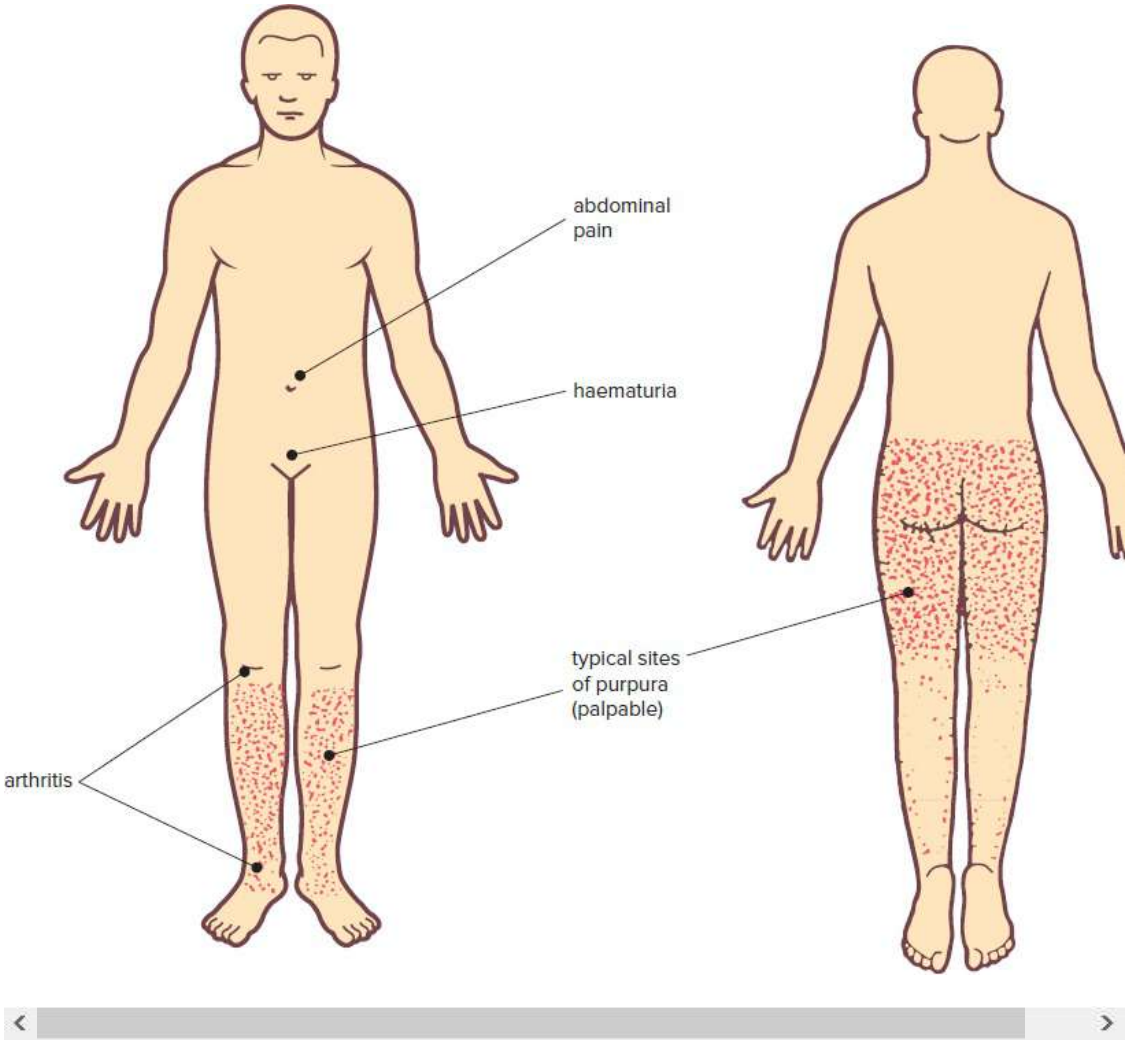

FIGURE 29.4 Henoch–Schönlein purpura: typical distribution

Associations

- Kidney involvement—deposition of IgA immune complex (a serious complication)
- Melaena
- Intussusception
- Scrotal involvement

Investigations

- FBE (if abnormal platelets or white cells, consider alternative diagnosis)
- Urine: protein and blood; spun specimen, micro for casts

Management

- Largely symptomatic—analgesics
- No specific therapy
- Short course of steroids for abdominal pain (if intussusception excluded)
- If haematuria: follow-up urine microscopy and kidney function especially if no resolution (in approximately 5%)

DxT arthralgia + purpuric rash ± abdominal pain → Henoch–Schönlein purpura

Practice tip

Beware of CKD in HSP.

Infective states

The purpura associated with severe infections, such as meningococcaemia and other septicaemias, is due primarily to a severe angiitis. Disseminated intravascular coagulation usually follows.[3]

Vascular, platelet and coagulative disorders

The features of vascular disorders are:

- easy bruising and bleeding into skin
- ± mucous membrane bleeding
- investigations normal

Abnormal bleeding in older people

In older adults, the outstanding causes are senile purpura and purpura due to steroids.[6] The cause in both instances is atrophy of the vascular supporting tissue.

Simple purpura (easy bruising syndrome)

This is a benign disorder occurring in otherwise healthy women usually in their 20s or 30s. The feature is bruising on the arms, leg and trunk with minor trauma. The woman may complain of heavy periods. However, major challenges to the haemostatic mechanism, such as dental extraction, childbirth and surgery, have not been complicated by excessive blood loss.

Factitial purpura

Unexplained bruising or bleeding may represent self-inflicted abuse or abuse by others. In self-inflicted abuse, the bruising is commonly on the legs or areas within easy reach of the patient.

Platelet disorders

The features are:

- petechiae ± ecchymoses
- bleeding from mucous membranes
- platelet counts <50 000/mm^3 (50 × 10^9/L): normal reference range 150–400 × 10^9/L

Immune (idiopathic) thrombocytopenic purpura

Clinical features[6]

- Acute onset in children
- Easy bruising and petechiae
- Epistaxis, bleeding gums and menorrhagia common

- No systemic illness
- Splenomegaly rare
- Isolated thrombocytopenia: platelets may be <20 000/mm^3
- Other blood cells normal
- Otherwise normal physical examination
- Normal bone marrow with normal or increased megakaryocytes (acute leukaemia and aplastic anaemia should be excluded)

DxT bruising + oral bleeding + epistaxis → ITP

The two distinct types caused by immune destruction of the platelets are:

- acute thrombocytopenia of childhood—usually in children, usually postviral
- chronic ITP—autoimmune disorder, usually in adult women; all cases should be referred to a specialist unit

Acute thrombocytopenia of childhood

This is caused by a reaction to a viral infection resulting in the production of cross-reacting antibodies against platelets.

There is an early risk of spontaneous haemorrhage, so refer/admit to hospital, especially if the platelet count is <30 × 10^9/L or there is active bleeding.

The prognosis is good, invariably self-limiting—90% resolve in 6 months. It may recur with further viral infections. The rest pass into chronic ITP.

Bleeding is treated with immunoglobulin IV or steroids (prednisolone or dexamethasone).

Chronic idiopathic (immune) thrombocytopenic purpura

Chronic ITP is a relapsing illness that rarely undergoes spontaneous remission and may require treatment with steroids, intravenous immunoglobulin or biological agents, e.g. rituximab. Ask about drug history. Some require splenectomy, but this operation is avoided where possible, especially in young children, because of the subsequent risk of severe infection, particularly with *Streptococcus pneumoniae*.[6] (Refer later in this chapter.)

Thrombotic thrombocytopenic purpura

This is an uncommon life-threatening syndrome of haemolytic anaemia, thrombocytopenia and extremely high LDH. Clinical features include fever (non-infectious) and neurologic and kidney abnormalities. The defect is in the absence of a specific protease in the plasma.

Coagulation disorders

The features are:

- ecchymoses
- haemarthrosis and muscle haematomas
- usually traumatic and delayed

The inherited disorders such as haemophilia A and B are uncommon and involve deficiency of one factor only. The acquired disorders, such as disseminated intravascular coagulation (DIC), occur more commonly and affect several anticoagulation factors (see TABLE 29.6). Antibodies may also develop against specific clotting factors.

Table 29.6 International nomenclature of clotting factors

Factor	Common synonyms
I	Fibrinogen*
II	Prothrombin*
III	No longer used
IV	Calcium
V	Proaccelerin
VI	No longer used
VII	Proconvertin (tissue factor)
VIII	Antihaemophilic factor A Antihaemophilic globulin
IX	Antihaemophilic factor B (Christmas factor)
X	Stuart–Prower factor
XI	Plasma thromboplastin antecedent
XII	Hageman factor, contact factor

Inherited coagulation disorders

A list of the inherited bleeding disorders is included in TABLE 29.1 . The better-known disorders are vWD, haemophilia A and haemophilia B (Christmas disease).

von Willebrand disease[7]

This is the most common disorder of haemostasis (incidence 1% of population) and is usually a mild problem with an excellent prognosis.[7] There are multiple subtypes—type I, the mildest, accounts for about 75%.

Clinical features

- Autosomal dominant inheritance (common types)
- Equal sex incidence
- Classically presents with mucocutaneous bleeding
- Prolonged bleeding time
- Bleeding tendency exacerbated by aspirin
- Platelets normal (common types)
- Defective platelet adhesion at site of trauma combined with factor VIII deficiency[7]
- aPTT prolonged
- Positive vW factor antigen (low)
- vW factor ristocetin (low)
- vW factor collagen binding assay
- Menorrhagia and epistaxis common
- Haemarthroses rare

DxT menorrhagia + bruising + increased bleeding—1. incisions 2. dental 3. mucosal → vWD

Treatment

- No specific treatment
- Avoid aspirin, NSAIDs, IM injections
- Be cautious of surgical and dental procedures
- Preparations that help include desmopressin acetate (DDAVP), factor VIII concentrates and tranexamic acid (especially for minor procedures)

Haemophilia A

Clinical features

- Spontaneous haemarthroses, especially knees, ankles and elbows, are almost pathognomonic
- X-linked recessive pattern of inheritance
- Invariably only males affected (1 in 5000)
- Females theoretically affected if haemophiliac father and carrier mother
- The human factor gene has long been identified
- Severity levels:

    severe—bleed spontaneously

    moderate—bleed with mild trauma or surgery

    mild—bleed after major trauma or surgery
- Deficiency of factor VIII
- aPTT prolonged
- Normal prothrombin time and fibrinogen
- Many seropositive for HIV, hepatitis B or C (factor VIII concentrate transmission)
- Low platelet count should suspect HIV-associated ITP[7]

DxT spontaneous haemarthrosis + muscle bleeds + delayed bleeding → haemophilia A

Treatment

- Infusion of recombinant factor VIII concentrates[8]

- Avoid aspirin

Haemophilia B (Christmas disease)

- Identical clinical features to haemophilia A
- Also an X-linked recessive hereditary disorder
- Incidence of 1 in 30 000
- Deficiency of coagulation factor IX
- Same laboratory findings as haemophilia A apart from specific factor assays
- Treatment is with recombinant factor IX concentrates

Splenectomy

Main indications:

- immune thrombocytopenic purpura
- haemolytic anaemias, esp. hereditary spherocytosis
- hypersplenism
- trauma
- Hodgkin/non-Hodgkin lymphoma

Post-splenectomy management[9]

Immediate problem is thrombocytosis (↑ platelets to 600–1000 × 10^9/L) for 2–3 weeks with risk of thromboembolism.

Long-term risk is overwhelming infection (S. pneumoniae [especially], *Haemophilus influenzae* and meningococcus), especially in young children in the first 2–3 years post-splenectomy. For elective surgery give immunisation at least 2 weeks before surgery. Best under specialist guidance. Lifelong prophylaxis should be considered in select patients such as those severely immunocompromised.

Prophylaxis

- Education about risks and early recognition of infection (special care with malaria)

- Pneumococcal and meningococcal vaccines—depends on age and should be guided by immunisation guidelines
- *Haemophilus influenzae* type B vaccine—once only if not immunised
- Influenza vaccine—annual
- Long-term penicillin may be indicated: amoxicillin daily or phenoxymethylpenicillin bd
- Urgent hospital admission if infection develops

Management principles for abnormal bleeding[1]

- Make the correct diagnosis.
- Stop or avoid drugs affecting the haemostatic system.
- Control bleeding episodes with appropriate drugs, blood products and local measures, such as simple compression or topical haemostatic agents.
- Infuse appropriate blood components for the treatment of coagulation factor deficiencies and some platelet disorders (e.g. factor VIII for haemophilia A, fresh frozen plasma for multiple factor deficiency).
- Refer patients with identified defects to a consultant haematologist or haemophilia centre.
- Supervise advanced planning in patients intending pregnancy, surgery or dental extraction.

When to refer[1]

- Management of haemorrhage is not amenable to simple measures such as local therapy with simple compression and other measures.
- Elective surgery or pregnancy is being planned.
- Platelet count $<30 \times 10^9$/L.

Practice tips

- A careful history and physical examination will usually pinpoint the cause of the bleeding disorder.
- Drug therapy can lead to unmasking of pre-existing haemostatic disorders (e.g. platelet dysfunction induced by aspirin may cause spontaneous bleeding in patients with underlying vWD).

- Think of disseminated intravascular coagulation (DIC) in any acutely ill patient with abnormal bleeding from sites such as the mouth or nose, venepuncture or with widespread ecchymoses. The clinical situations are numerous, such as septicaemia, obstetric emergencies, disseminated malignant disease, falciparum malaria and snake bites.
- Be cautious of non-prescription therapies affecting oral anticoagulants or causing platelet dysfunction (e.g. *Ginkgo biloba*).

Resources

Post surgical management—splenectomy. Spleen Australia.

Bleeding Assessment Tool: https://www.southernpath.com.au/media/6606/thrombophilia.pdf

Coagulation cascade: https://en.wikipedia.org/wiki/coagulation

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30 Chest pain

There is a disorder of the breast marked with strong and peculiar symptoms, considerable for the kind of danger belonging to it. The seat of it, and sense of strangling, and anxiety with which it is attended, may make it not improperly be called angina pectoris.

WILLIAM HEBERDEN (1710–1801)

The presenting problem of chest pain is common yet very threatening to both patient and doctor because the underlying cause in many instances is potentially lethal, especially with chest pain of sudden onset. These patients require rapid evaluation with a 12-lead ECG. The causes of acute chest pain are summarised and presented in FIGURE 30.1 .

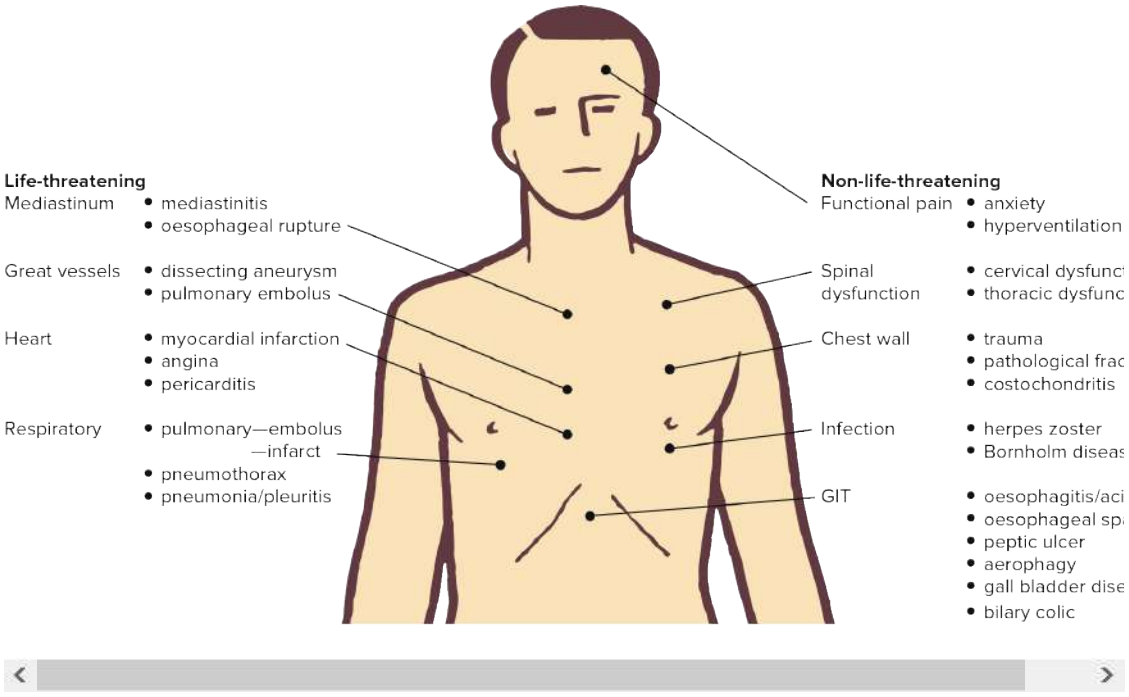

FIGURE 30.1 Causes of acute chest pain

Key facts and checkpoints

- Chest pain represents an acute coronary event until proved otherwise.
- Immediate life-threatening causes of spontaneous chest pain are:

  myocardial infarction (MI) and unstable angina (acute coronary syndromes: ACS)

  pulmonary embolism

  aortic dissection

  tension pneumothorax
- The main differential diagnoses of ACS include aortic dissection, pericarditis, oesophageal reflux and spasm, biliary colic and hyperventilation with anxiety and Takotsubo stress-related cardiomyopathy.
- The history remains the most important clinical factor in the diagnosis of ischaemic heart disease. With angina, a vital clue is the reproducibility of the symptom.
- Consider unstable angina = pre-myocardial infarction.
- Unrecognised MI is roughly equally divided into atypical or silent. More common with diabetes, hypertension, elderly and females.

A diagnostic approach

The diagnostic strategy model (see TABLE 30.1) can be used to analyse chest pain according to the five self-posed questions.

Table 30.1 Chest pain: diagnostic strategy model

Probability diagnosis
Musculoskeletal (chest wall)
Psychogenic
Gastro-oesophageal reflux disease
Angina

Serious disorders not to be missed

Cardiovascular:

- acute coronary syndromes
- aortic dissection
- pulmonary embolism/infarction
- myocarditis

Neoplasia:

- lung cancer
- tumours of spinal cord and meninges

Severe infections:

- pneumonia/pleuritis (pleurisy)/empyema
- mediastinitis
- pericarditis

Pneumothorax, esp. tension

Oesophageal rupture

Pitfalls (often missed)

Mitral valve prolapse

Oesophageal spasm

Biliary colic/acute cholecystitis

Herpes zoster

Fractured rib (e.g. cough fracture)

Costochondritis

Spinal dysfunction

Muscular tear

Rarities:

- Takotsubo cardiomyopathy
- pancreatitis; gall bladder disease
- Bornholm disease (pleurodynia)
- cocaine inhalation (can ↑ ischaemia)
- hypertrophic cardiomyopathy

Seven masquerades checklist

Depression (possible)

Anaemia (indirect)

Drugs (e.g. cocaine)

Spinal dysfunction

Is the patient trying to tell me something?

Consider functional causes, especially anxiety or panic with hyperventilation (e.g. Takotsubo syndrome), opioid dependency.

Probability diagnosis

The commonest causes encountered in general practice are musculoskeletal or chest wall pain and psychogenic disorders. The former is a very important yet often overlooked cause and sometimes inappropriately referred to as fibrositis or neuralgia. Causes include costochondritis, muscular strains, dysfunction of the sternocostal joints and dysfunction of the lower cervical spine or upper thoracic spine, which can cause referred pain to various areas of the chest wall. Gastro-oesophageal reflux may be difficult to distinguish from angina. Angina is common and must always be considered. If angina-like pain lasts longer than 15 minutes, myocardial infarction must be excluded.

Red flag pointers for acute chest pain

- Dizziness/syncope
- Pain or heaviness/pressure in arms L > R, jaw
- Thoracic back pain
- Sweating/diaphoresis
- Palpitations
- Syncope
- Haemoptysis
- Dyspnoea
- Pain on inspiration
- Pallor
- Past history: ischaemia, diabetes, hypertension

Serious disorders not to be missed

The usual triad of malignancy, myocardial ischaemia and severe infections (see TABLE 30.1) must be considered. In addition, other uncommon cardiovascular catastrophes, such as a dissecting aortic aneurysm and pulmonary embolus, must be excluded, especially in those at risk.

Spontaneous pneumothorax should also be considered, especially in a young male of slight build. Malignancies of the lung are relatively common and may present as pain when the previously asymptomatic tumour invades nerves or the spine.

The severe infections that cause chest pain include pneumonia/pleurisy, pericarditis and mediastinitis.

Pitfalls

Unfortunately, myocardial infarction and angina are often missed. Referred pain from spinal dysfunction, especially if referred anteriorly, is commonly overlooked. Other pitfalls include a cough fracture of a rib, herpes zoster (prior to the eruption) and gastrointestinal disorders, including oesophageal spasm, reflux and cholecystitis. Mitral valve prolapse can cause chest pain, although the mechanism is unclear: think of it in an unwell female prone to palpitations and chest pain. The pain tends to be sharp, fleeting, non-exertional and located near the cardiac apex.

General pitfalls

General pitfalls include:

- not being ‘coronary aware’ in patients presenting with chest pain
- referred pain from spinal disorders, especially of the lower cervical spine
- labelling chest pain as psychological in an anxious person presenting with acute chest pain
- assuming that pain radiating down the inside of the left arm is always cardiac in origin
- being unaware that up to 20% of myocardial infarctions are silent, especially in elderly patients, and that pulmonary embolism is often painless if the main pulmonary veins are involved

Seven masquerades checklist

Of this group, spinal dysfunction is possible. Disc lesions from the lower cervical spine are unlikely to cause chest wall pain, but dysfunction of the facet joints of this area of the spine and the upper thoracic spine are common causes of referred pain to the chest wall. Nerve root pain from spinal problems is rarely found in the chest wall. Pathological fractures secondary to osteoporosis or malignancy in the vertebrae cause posterior wall pain. Cocaine users can present with chest pain.

Psychogenic considerations

With psychogenic causes the pain can occur anywhere in the chest, and tends to be continuous and sharp or stabbing rather than constricting. Associated symptoms, particularly during a panic

episode, include palpitations, deep breathing, fatigue, tremor, agitation and anxiety. Abnormal stress, tension, anxiety or depression may precipitate the pain, which often lasts hours or days.

The clinical approach

History

A meticulous history of the behaviour of the pain is the key to diagnosis. The pain should be analysed into its usual characteristics with the SOCRATES system (see CHAPTER 82). Association with serious medical problems such as diabetes, Marfan syndrome, anaemia and connective tissue disorders (e.g. SLE, RA) should be kept in mind. Consider coronary risk factors. The ability to take a detailed history will obviously be limited with severe acute pain.

Associated symptoms

- *Syncope.* Consider myocardial infarction, pulmonary embolus and dissecting aneurysm.
- *Pain on inspiration.* Consider pleuritis, pericarditis, mediastinitis, pneumothorax and musculoskeletal (chest wall pain).
- *Thoracic back pain.* Consider spinal dysfunction, acute coronary syndromes, angina, aortic dissection, pericarditis and gastrointestinal disorders such as a peptic ulcer, biliary colic/cholecystitis and oesophageal spasm.

Key questions

- Where exactly do you get the pain? Can you pinpoint it?
- Does the pain travel anywhere?
- Can you give me a careful description of the pain?
- How long did the pain last and could you do anything to relieve it?
- Is the pain brought on by exertion and relieved by rest?
- Do cold conditions bring it on?
- Do you have any other symptoms, such as breathlessness, faintness, fever, nausea or vomiting, dizziness, weight loss, sweating or back pain?
- Is the pain made worse by breathing or coughing, or by movement or pressing on that area?
- Is there any blood in any sputum you bring up?
- Is your pain associated with what you eat and drink? Or with a bitter taste?

- Do you get the pain on stooping over and after lying in bed at night?
- Do antacids relieve your pain?
- Have you noticed a rash where you get the pain?
- Have you had a blow to your chest or an injury to your back?

Examination

The examination should focus on the following areas:

- general appearance—evidence of atherosclerosis (senile arcus, thickened vessels), pale and sweating (myocardial infarction, dissecting aneurysm or pulmonary embolus), hemiparesis (? aortic dissection)
- pulses—both radial and femoral—check for nature of pulse and absence of femoral pulses
- blood pressure, temperature, respiratory rate, oxygen saturation
- palpation of chest wall, lower cervical spine and thoracic spine—look for evidence of localised tenderness, pathological fracture, spinal dysfunction, herpes zoster
- palpation of legs—check for evidence of deep venous thrombosis
- examination of chest—check for evidence of pneumothorax
- auscultation of chest:

    reduced breath sounds, hyper-resonant percussion note and vocal fremitus → pneumothorax

    friction rub → pericarditis or pleurisy

    basal crackles → cardiac failure

    apical systole murmur → mitral valve prolapse

    aortic diastolic murmur → proximal dissection (aortic regurgitation)

Note: In the presence of a myocardial infarction, the examination may be normal but the patient, apart from being cold, clammy or shocked, may have muffled heart sounds, a gallop rhythm, a systolic murmur. With an aortic dissection the patient may also appear cold, clammy and shocked, but may show absent femoral pulses, hemiparesis and a diastolic murmur of aortic regurgitation.

- upper abdominal palpation—check for tenderness suggestive of gall bladder disease or peptic ulceration

Possible findings on examination of a person with chest pain are presented in FIGURE 30.2 .[1]

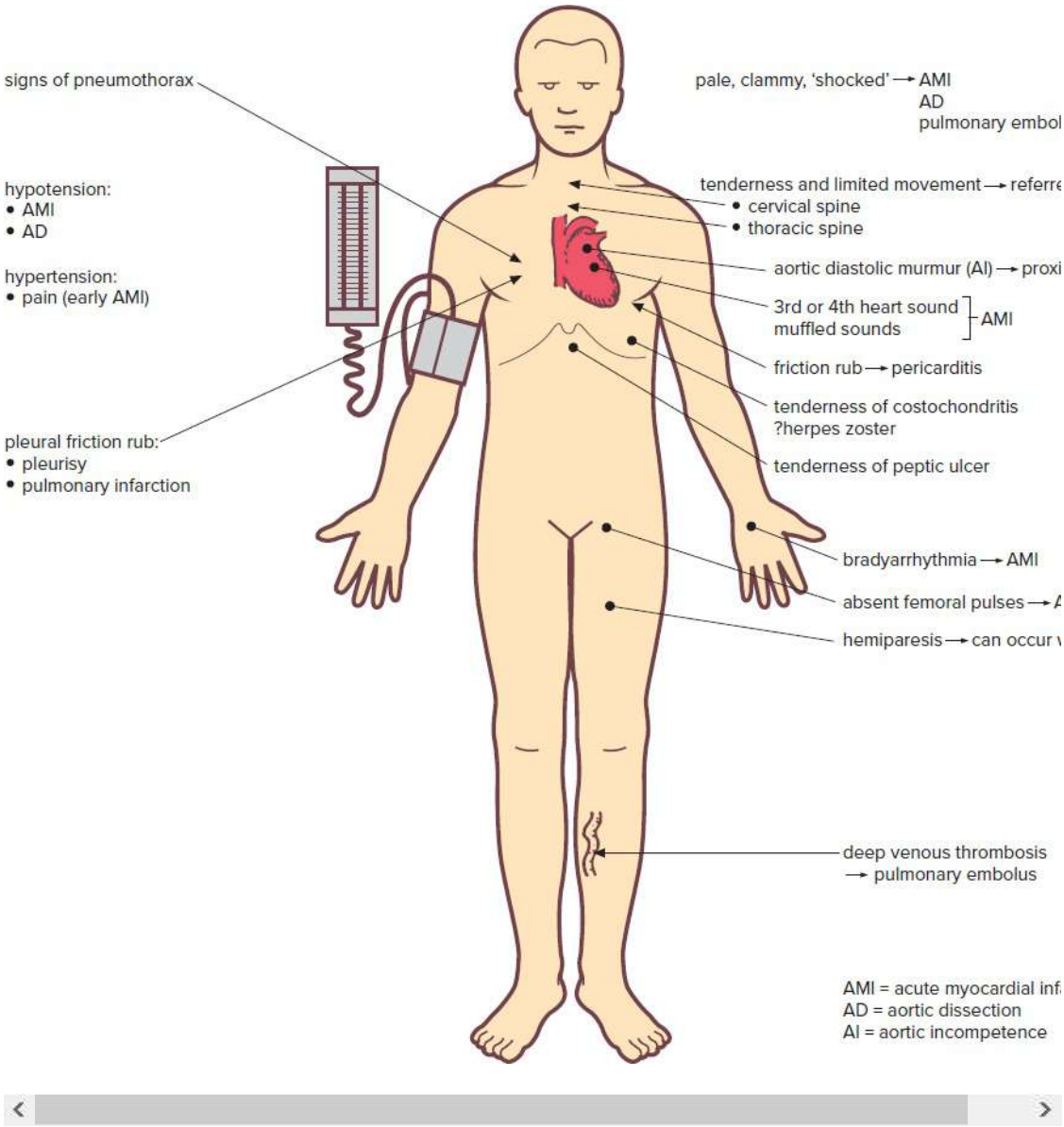

FIGURE 30.2 Possible examination findings in a person with chest pain

Investigations[1]

The following investigations to aid diagnosis are available, although the majority are sophisticated and confined to hospitals with high-technology imaging departments. The fundamental tests that are readily available to the GP—ECG, chest X-ray and cardiac enzymes—should help confirm the diagnosis in most instances. Anyone with undifferentiated chest pain should have a 12-lead ECG.

Electrocardiogram (ECG)

This may be diagnostic for ischaemia and myocardial infarction, although it is important to bear in mind that it may be normal with both, including the early minutes to hours of an acute infarction.

It can be helpful to differentiate between myocardial infarction, pulmonary embolism and pericarditis. The ECG in pulmonary embolism may be normal but if massive may show right axis deviation, right BBB and right ventricular strain. Pericarditis is characterised by low voltages and saddle-shaped ST segment elevation.

Exercise stress test

This is the key test for defining chest pain as cardiac in origin. Physical stress, such as the motor-driven treadmill or a bicycle ergometer, is used to elicit changes in the ECG to diagnose myocardial ischaemia.

Exercise thallium scan

This radionuclide myocardial perfusion scan using thallium can complement the exercise ECG.

Ambulatory ECG Holter monitor

This monitor is especially useful for silent ischaemia, variant angina and arrhythmias.

Chest X-ray

The routine CXR is taken in full inspiration. Ask for an expiration film if pneumothorax is suspected.

Blood glucose

Tests association with diabetes.

Haemoglobin and blood film

Anaemia is a possible associated factor.

Serum enzymes

Damaged (necrosed) myocardial tissue releases cellular enzymes, which are markers of this damage:

- troponin T and troponin I (the key marker)–on arrival and 2 hours later (ADAPT trial protocol)
- creatinine kinase (CK) and creatinine kinase–myocardial bound fraction (CK–MB)[2]

- myoglobin

Transthoracic echocardiography

This can be used in the early stages of myocardial infarction to detect abnormalities in heart wall motion, when ECGs and enzymes are not diagnostic. Stress echocardiography can be useful where standard exercise testing has been unhelpful.

Transoesophageal echocardiography (TOE)

More sensitive and is the investigation for dissecting aneurysm (immediate diagnosis), prosthetic valves and embolisation.

Isotope scanning

1. Technetium-99m pyrophosphate studies:
    - myocardium—to diagnose posterolateral myocardial infarction in the presence of bundle branch block
    - pulmonary—to diagnose pulmonary embolism
2. Gated blood pool nuclear scan (radionuclide ventriculography)—this scan tests left ventricular function at rest and exercise in patients with myocardial ischaemia.

Angiography (arteriography)

Angiography, including CT angiogram, should be selective:

1. coronary—to evaluate coronary arteries
2. pulmonary—to diagnose pulmonary thromboembolism
3. coronary CT
4. MRI

Coronary calcium scan

The calcium score, which is elevated by high-speed non-invasive CT, shows the quantity of calcium in the coronary vessels. It indicates risk for a coronary attack. A score of 0 reflects an excellent medium-term prognosis.

Oesophageal studies

- Endoscopy

- Barium swallow
- Oesophageal manometry
- Radionucleide transit studies

Spine—X-ray

- Cervical spine
- Thoracic spine

Site, radiation and features of chest pain syndromes

Myocardial ischaemia[1]

Coronary artery disease includes the acute coronary syndromes (unstable angina and myocardial infarction), stable angina and other variants of angina.

The typical retrosternal distribution of myocardial ischaemia is shown in FIGURE 30.3 . Retrosternal pain or pain situated across the chest anteriorly should be regarded as cardiac until proved otherwise.

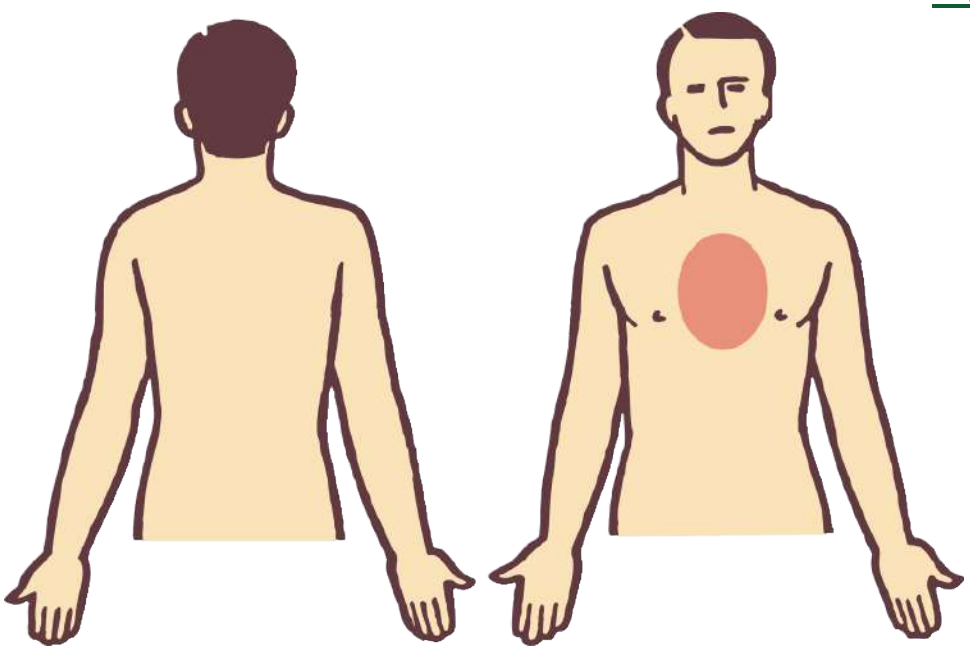

FIGURE 30.3 Pain of myocardial ischaemia: typical site

The wide variation of sites of pain—umbilicus to jaw, including neck, inside of arms, epigastrium and interscapular—should always be kept in mind (see FIG. 30.4). Pain is referred into the left arm more commonly than into the right arm. The best predictor of AMI is pain in both arms. The best predictors for ruling out AMI are pleuritic chest pain, sharp pain and pain reproduced by palpation.[3]

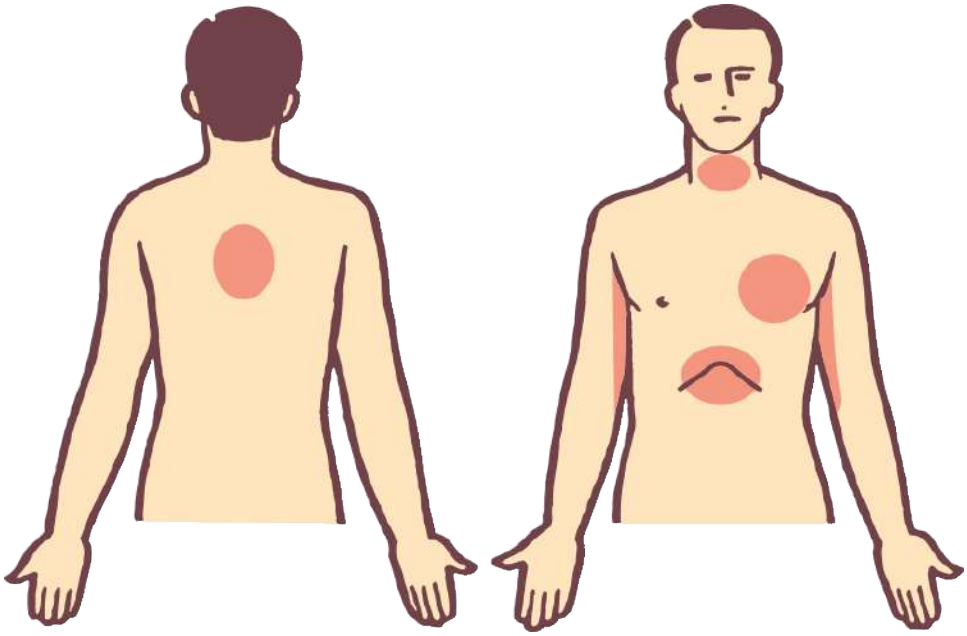

FIGURE 30.4 Pain of myocardial ischaemia: other sites

The quality of the pain is usually described as pressure, heaviness or tightness. The patient often uses the clenched fist sign to illustrate a sense of constriction.

The radiation of pain will assist in differentiating ischaemic pain from that caused by pericarditis. Enquiry about precipitating and relieving factors will enable a differentiation to be made between ischaemic pain and the almost identical pain caused by reference from the spine. Associated symptoms include dyspnoea, dizziness, nausea and vomiting, and sweating (diaphoresis).

If a retrosternal pain almost identical with that of myocardial ischaemia is precipitated not by exertion, but by bending, lifting, straining or lying down, consider oesophageal reflux or spasm. This is frequently confused with ischaemic heart disease and can cause radiation into the left arm.

Stable angina. The pain of angina tends to last a few minutes only (average 3–5 minutes) and is relieved by rest and glyceryl trinitrate (nitroglycerin). The pain may be precipitated by an arrhythmia.

The types of acute coronary syndromes are summarised in TABLE 30.2 .[5]

Types of acute coronary syndromes

Table 30.2 Types of acute coronary syndromes

	Serum markers		
	Creatinine kinase	Troponin	ECG at evaluation
Unstable angina			
Low risk	Normal	Non-detectable	Normal
High risk	Normal	Detectable	ST depression
Myocardial infarction			
Non-ST elevation (NSTEMI)	Elevated	Detectable	ST depression, no Q wave
ST elevation (STEMI)	Elevated	Detectable	± Q wave or new LBBB

STEMI = ST elevation myocardial infarction

1. *Myocardial infarction.* Ischaemic pain lasting longer than 15–20 minutes is usually infarction. However, it can resolve in a few minutes or within 24 hours. The pain is typically heavy and crushing, and can vary from mild to intense. Occasionally the attack is painless, typically with diabetes. Pallor, sweating and vomiting may accompany the attack.

2. *Unstable angina.* This term includes rest angina, new onset effort angina, post-infarct angina and post-coronary procedure angina. Severe ischaemic chest pain can last 15–20 minutes or more. It is classified as low-risk or high-risk ‘minor myocardial damage’.

For management purposes it is best to classify the clinical presentation of acute ischaemic chest pain as an ST elevation myocardial infarction (STEMI) or a non-ST elevation acute coronary syndrome (NSTEACS), which includes NSTEMI and unstable angina.

Aortic dissection

Chest pain is present in 75% of dissections. The pain—which is usually sudden, unrelenting, severe and midline—has a tearing or ripping sensation and is usually situated retrosternally and between the scapulae (see FIG. 30.5). It radiates to the abdomen, flank and legs. An important diagnostic feature is the inequality in the pulses (e.g. carotid, radial and femoral). There may also be occlusion of the coronary or renal arteries with appropriate symptoms and signs. Hemiplegia, aortic incompetence or cardiac tamponade can occur. Investigations include transoesophageal electrocardiogram, CT angiogram and MRI.

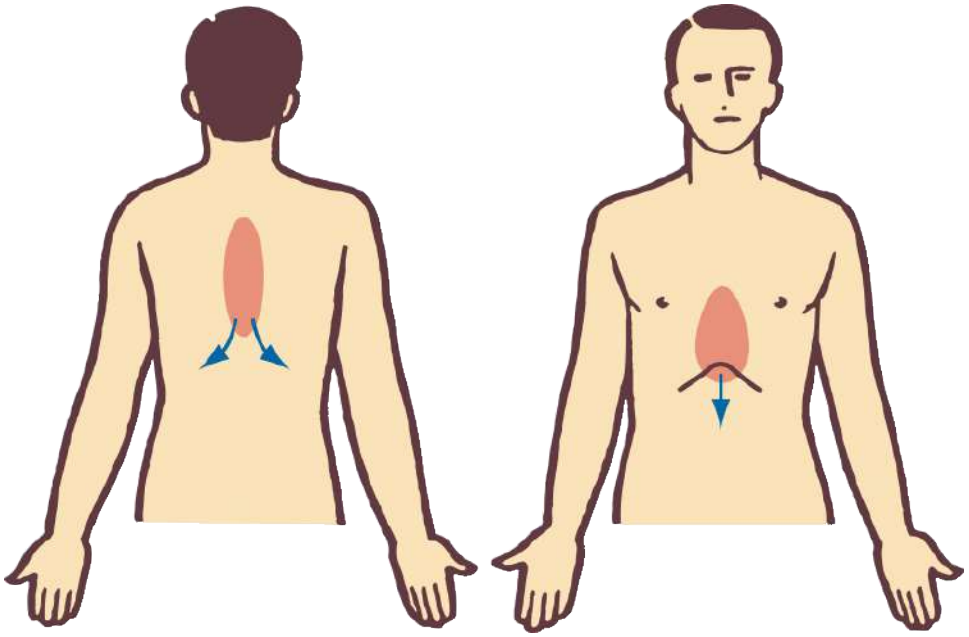

FIGURE 30.5 Pain of aortic dissection

Pulmonary embolism[4]

This may have a dramatic onset following occlusion of the pulmonary artery or a major branch, especially if more than 50% of the cross-sectional area of the pulmonary trunk is occluded. There are three general types: massive (obstructive shock or BP < 90 mmHg), submassive (acute without hypotension but right ventricular dysfunction) and non-massive (low risk).

The diagnosis can present clinical difficulties, especially when dyspnoea is present without pain. It can be asymptomatic. Embolism usually presents with retrosternal chest pain (see FIG. 30.6) and may be associated with syncope and breathlessness. In addition, hypotension, acute right heart failure or cardiac arrest occurs with a massive embolus. The physical examination can be deceptively normal. Pulmonary infarction is generally less dramatic than embolism and it is usually accompanied by pleuritic chest pain and haemoptysis. It complicates embolism in about 10% of patients. The diagnosis is usually confirmed by a CT pulmonary angiogram (best) and/or V/Q scan (see later in chapter) and ECG (look for T-wave inversion V1–V4). The Wells score for risk stratification is a useful probability guide. The D-dimer test is useful for helping ‘rule out’ a PE where it is already unlikely.

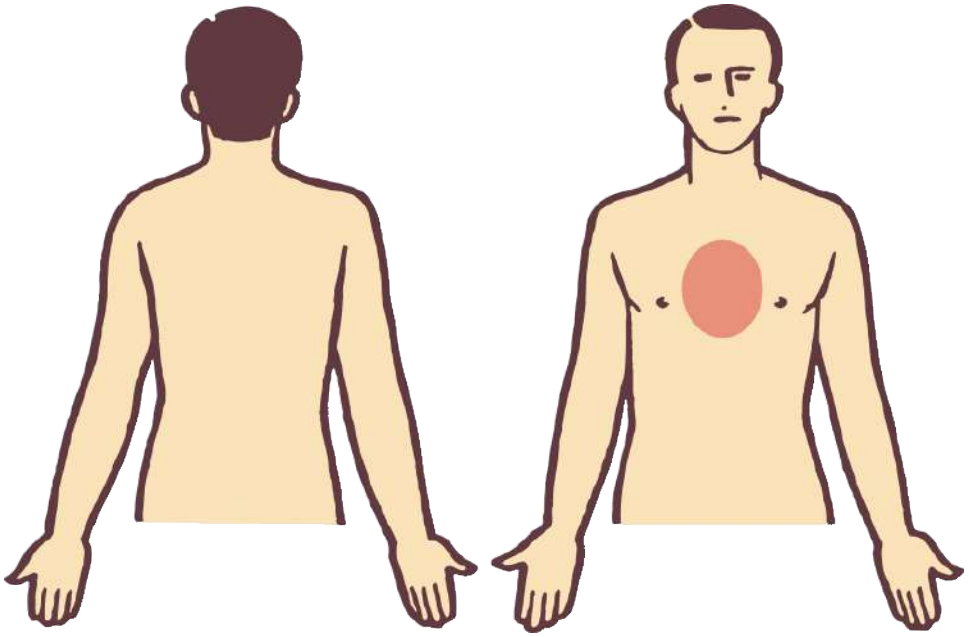

FIGURE 30.6 Pain of pulmonary embolism

Pleuritis[6]

Inflammation of the pleura is due to underlying pneumonia (viral or bacterial), pulmonary infarction, tumour infiltration or connective tissue disease (e.g. SLE).

Clinical features

- Often sudden onset
- Pain usually localised without radiation
- Sharp knife-like pain
- Continuous pain with sharp exacerbations
- Aggravated by inspiration, sneezing and coughing
- May be associated dyspnoea, cough, haemoptysis

Epidemic pleurodynia (Bornholm disease)

Unilateral severe knife-like intermittent chest pain and adjacent upper abdominal pain (‘the devil’s grip’) following an URTI—affecting any age (i.e. younger average than MI). Other symptoms include fever, malaise, headache, and tender truncal muscles. It is caused by a Coxsackie B virus. CXR is normal; diagnosis is by exclusion. It usually settles within a week

with simple analgesics. Recurrences may follow.

Acute pericarditis[6]

The clinical presentation depends on the cause, whether viral (commonest), a connective tissue disorder, bacterial, uraemia or post-AMI. It may be idiopathic. There may be fever, malaise, fatigue and anxiety.[6]

- Signs: friction rub, tachycardia, paradoxical pulse
- Investigations: ECG, CXR, echocardiography

Pericarditis causes three distinct types of pain:

1. pleuritic (the commonest), aggravated by cough and deep inspiration, sometimes brought on by swallowing; worse with lying flat, relieved by sitting up and leaning forward
2. steady, crushing, retrosternal pain radiating to neck and arms that mimics myocardial infarction
3. pain synchronous with the heartbeat and felt over the praecordium and left shoulder

Occasionally, two and rarely all three types of pain may be present simultaneously (see FIG. 30.7).

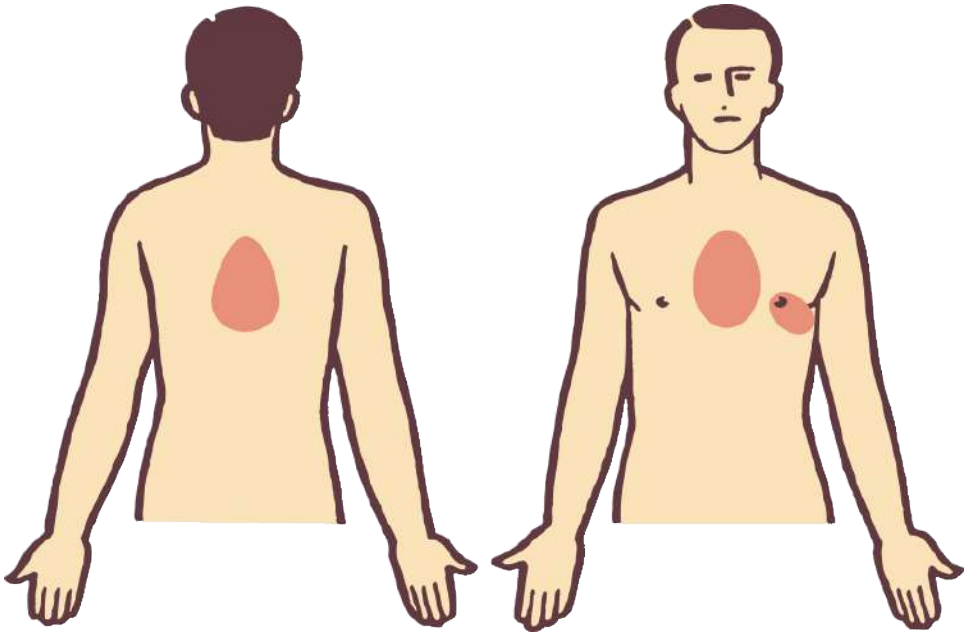

FIGURE 30.7 Pain of pericarditis

The cardinal sign is a pericardial friction rub (often transient). Treatment depends on the cause—

may be colchicine (3 months) plus aspirin or ibuprofen (1–2 weeks).[7]

Spontaneous pneumothorax

The acute onset of pleuritic pain and dyspnoea in a person with a history of asthma or emphysema is the hallmark of a pneumothorax. It is due to a rupture of a subpleural ‘bleb’ or a small air-containing cyst. It often occurs in young, slender males without a history of lung disorders. The pain varies from mild to severe and can be felt anywhere in the chest, sometimes being retrosternal. Typical pain distribution is shown in FIGURE 30.8 . The diagnosis is made on expiration film.

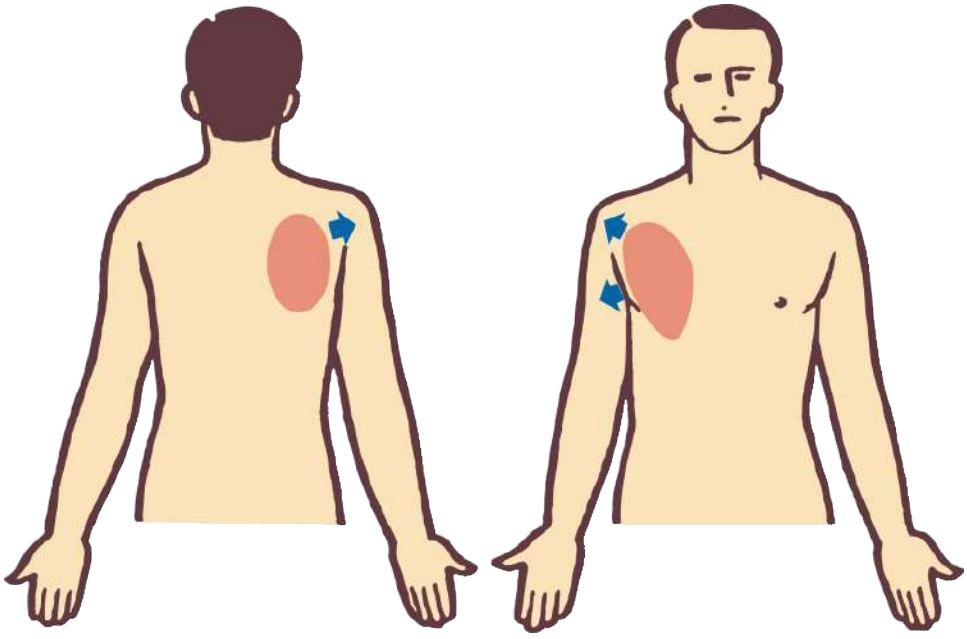

FIGURE 30.8 Pain of pneumothorax (right side)

If a tension pneumothorax becomes painful and dyspnoea becomes rapidly more intense, urgent decompression of air is essential (see later in chapter). A comparison of the serious causes of acute chest pain is summarised in TABLE 30.3 .

Table 30.3 A comparison of the serious causes of acute chest pain

	Myocardial infarction	Angina	Pulmonary embolus	Aortic dissectic
Pain intensity	+ → + + + +	+	+ → + + +	+ + + + +
Pain quality	Heavy	Heavy	Dull	Tearing

	Crushing Vice-like Burning	Aching Tightness Burning	Heavy	Searing
Pain site	Deep retrosternal	Deep retrosternal	Retrosternal	Anterior chest
Pain radiation	Throat/lower jaw Left arm (often) Right arm (uncommon) Back (uncommon)	As for infarction	Lateral chest (pleuritic)	Front to back chest Down back to abdomen Arms
History	Family, risk factors	Family, risk factors	Phlebitis Calf pain Immobility Surgery Malignancy	Atherosclerosi Hypertension ?Marfan
Associated symptoms	Pallor, nausea, sweating, vomiting, dyspnoea, syncope	Strangling in throat	Dyspnoea, syncope, sweating, vomiting, cyanosis, agitation, haemoptysis	Syncope, pallo cyanosis, Neurological: • hemipare • paraplegi
Pulse	Variable arrhythmias	Variable arrhythmias	Tachycardia	Unequal, some absent
Cardiac auscultation	± Gallop rhythm murmur of MI	S_3 during attack	↓ Pulmonary S_2, S_3 or S_4	± Murmur of Al
Chest auscultation	Basal crackles		± Adventitious sounds	
Chest X-ray			± Localised oligaemia or infarction	Widening of mediastinum
ECG	Q waves ST elevation T inversion	Normal or ST depression	R axis deviation S_1, Q_3, T_3 sign	May show myocardial infarction

	(variable)			
Special definitive diagnostic tests	Serum enzymes: troponin I or T Cardiac scanning	Stress ECG Coronary angiography Technetium scanning Enzymes	Lung scanning CT pulmonary angiography V/Q scan	TOE Ultrasound Aortic angiography CT scan

Oesophageal pain

Gastro-oesophageal reflux can cause oesophagitis characterised by a burning epigastric or retrosternal pain that may radiate to the jaw. Consider oesophageal rupture if sudden onset after endoscopy. The pain is aggravated or precipitated by lying flat or bending over, especially after meals, and is more frequent at night. The pain is worse if oesophageal spasm is present. Oesophageal motor disorders, including spasm, may occur in isolation. The pain may radiate uncommonly to the back (see FIG. 30.9). It may be precipitated by eating, especially hot or cold food and drink, and may be relieved by eating or by glyceryl trinitrate (nitroglycerin) and other nitrates. Features differentiating angina-like oesophageal pain and cardiac pain are presented in TABLE 30.4 . Gastrointestinal causes of chest pain are summarised in TABLE 30.5 .[1]

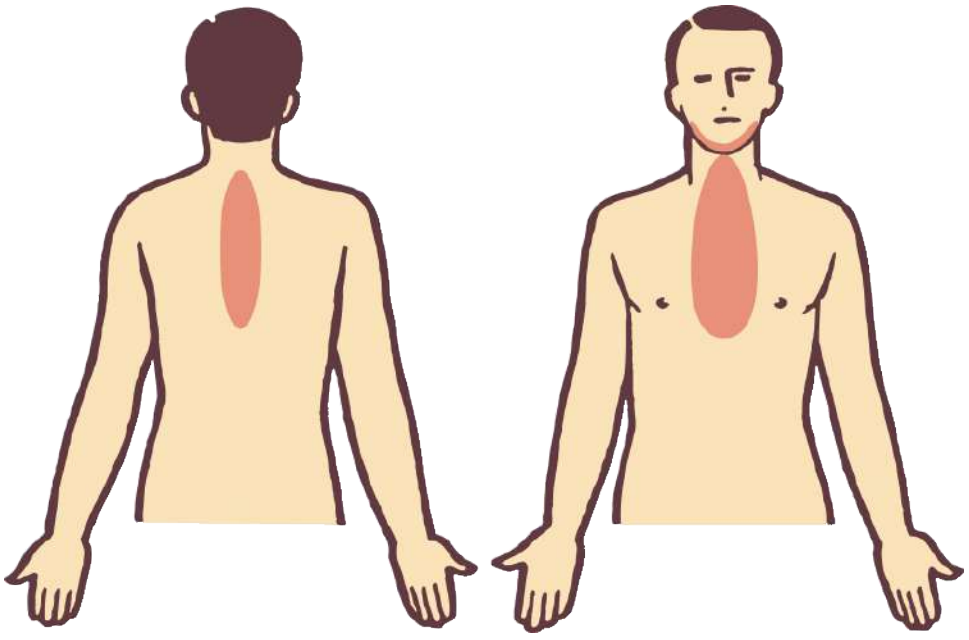

FIGURE 30.9 Oesophageal pain

Table 30.4 Features differentiating angina-like oesophageal pain and cardiac pain

	Favour oesophageal	Favour cardiac	Non-discriminating
Precipitating factors	Meals, posture	Consistently with exercise	Emotion
Relieving factors	Antacids		Rest, nitrates
Radiation	Epigastrium	Arm	Back
Associated symptoms	Heartburn, regurgitation, dysphagia	Dyspnoea	Sweating

Table 30.5 A comparison of gastrointestinal causes of chest pain

	Acid reflux	Oesophageal spasm	Peptic ulcer	Gall bladder disease
Site	Epigastric	Deep retrosternal	Deep retrosternal	Right hypochondriu
Radiation	Retrosternal Throat	Back	To back (DU)	Below right scapula Tip right shoulder
Quality	Burning	Constricting	Gnawing	Deep ache
Precipitation	Heavy meals Wine/coffee Lying Bending	Eating hot/cold food and drinks	Eating: • GU: 30 min • DU: 2–3 hours	Fatty food
Relief	Standing Antacids	Antispasmodics Nitroglycerin	Antacids	Getting onto hands and knees

Associated symptoms	Water brash	Dysphagia	Dyspepsia	Flatulence Dyspepsia

GU = gastric ulcer; DU = duodenal ulcer

Spinal pain

The commonest cause of pain of spinal origin is vertebral dysfunction of the lower cervical or upper dorsal region (see CHAPTER 27). The spinal problem may be a disc prolapse (relatively common in the lower cervical spine, but rare in the upper thoracic spine) or dysfunction of the facet joints or costovertebral joints causing referred pain. This referred pain can be present anywhere in the chest wall, including anterior chest, which causes confusion with cardiac pain (see FIG. 30.10). The pain is dull and aching. It may be aggravated by exertion, certain body movements or deep inspiration. The old trap for unilateral nerve root pain is herpes zoster.

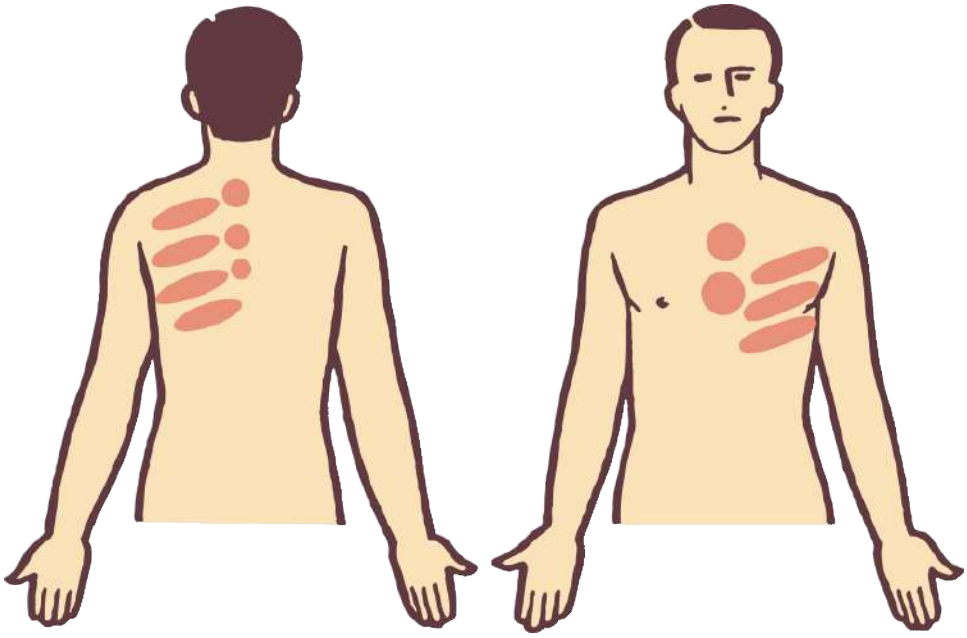

FIGURE 30.10 Possible pain sites for thoracic spinal dysfunction (left side)

Costochondritis[6]

This causes mild to moderate anterior chest wall pain that may radiate to the chest, back or abdomen. It is usually unilateral, sharp in nature and exaggerated by breathing, physical activity or a specific position. It may be preceded by exercise or a URTI and can persist for several months. It is diagnosed by eliciting tenderness at the costochondral junction of the affected ribs and needs to be differentiated from Tietze syndrome, where there is a tender, fusiform swelling at the costochondral junction (refer to CHAPTER 93).

Psychogenic pain

Psychogenic chest pain can occur anywhere in the chest, but often it is located in the left submammary region, usually without radiation (see FIG. 30.11). It tends to be continuous and sharp or stabbing. It may mimic angina but tends to last for hours or days. It is usually aggravated by tiredness or emotional tension and may be associated with shortness of breath, fatigue and palpitations.

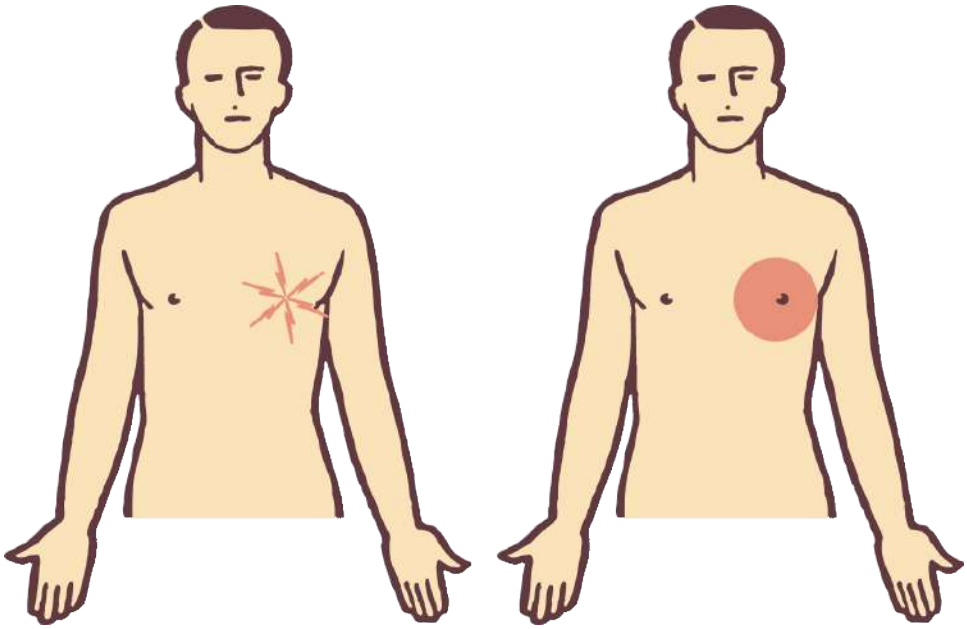

FIGURE 30.11 Typical sites of psychogenic pain

Da Costa syndrome (effort syndrome) is recurrent attacks of stabbing left-sided submammary pain, usually associated with anxiety ± depression.

Takotsubo cardiomyopathy or ‘broken heart syndrome’ presents with acute chest pain and shortness of breath. It mimics an acute anterior myocardial infarction. It is caused by a major catecholamine discharge following an emotionally stressful event, resulting in apical left ventricular ballooning. Treatment is usually with aspirin, beta blockers and ACE inhibitors. Most recover completely.

Chest pain in children

Chest pain in children is rarely the result of serious pathology but is an important complaint, especially in adolescents. A US study has shown that the mean age for childhood chest pain is 11.9 years.[8] Most cases are of unknown aetiology (probably many are psychogenic), while common causes include musculoskeletal disorders, cough-induced pain, costochondritis,

psychogenic disturbance (includes hyperventilation) and asthma.[8] See TABLE 30.6 .

Table 30.6 Causes of chest pain in children: diagnostic model[8]

Probability diagnosis

Musculoskeletal (chest wall pain)

- cough strain (10%)
- injury
- muscle strain
- costochondritis
- precordial catch syndrome (stitch in side)
- asthma

Note: The most common category is 'unknown' (21%)

Serious disorders not to be missed

Vascular

- ischaemic pain: structural cardiac conditions
- arrhythmias (e.g. PSVT)

Infection

- pericarditis
- myocarditis
- pneumonia
- herpes zoster

Other:

- pneumothorax
- POTS syndrome

Pitfalls (often missed)

Kawasaki syndrome

Breast disorders

Cocaine inhalation

Rarities:

- Bornholm disease
- oesophagitis or gastric pain

Is the patient trying to tell me something?

Psychogenic: stress, anxiety, depression (10%)

Chest pain in children younger than 12 years old is more likely to have a cardiorespiratory cause, such as cough, asthma, pneumonia or heart disease, while chest pain in adolescents is more likely to be psychogenic.

Causes of musculoskeletal pain include strains to pectoral, shoulder or back muscles after excessive exercise, and minor trauma from sports such as football or wrestling.

Breast problems can present as chest pain.

Cardiac causes

Myocardial ischaemia is very rare in children but should be considered in any child with exercise-induced chest pain, adolescents with longstanding diabetes and children with sickle-cell anaemia.

Precordial catch (Texidor twinge or stitch in the side)[9]

This complaint, which is common in children and adolescents, presents as a unilateral low chest pain that lasts usually 30 seconds to 3 minutes, typically with exercise, such as long-distance running. The pain is relieved by straightening up and taking very slow deep breaths followed by shallow breaths.

Chest pain in the elderly

Chest pain is a very important symptom in the elderly as the life-threatening cardiovascular conditions—myocardial infarction and angina, dissecting aneurysm and ruptured aorta—are an increasing manifestation with age. The elderly patient presenting with chest pain is most likely to have angina or myocardial infarction. Other important disorders to consider are herpes zoster, cough fracture of the rib, malignancy, pleurisy, pulmonary embolus and gastro-oesophageal reflux.

Angina pectoris

Main features

- There is a 2–3% incidence between 25 and 64 years.[10]
- The history is the basis of diagnosis.
- Angina is an oppressive discomfort rather than a pain, typically transient and lasting <10 mins.
- It is mainly retrosternal: radiates to arms, jaw, throat, back.
- It may be associated with shortness of breath, nausea, faintness and sweating.

- It occurs during exercise, emotion, after meals or in cold.
- It is relieved within a few minutes with rest.
- Physical examination is usually not helpful, except during an attack.
- Mitral valve prolapse, oesophageal spasm and dissecting aneurysm are important differential diagnoses.
- The causes of angina are summarised in TABLE 30.7 .

Table 30.7 Causes of angina

Coronary artery atheroma
Valvular lesions (e.g. aortic stenosis)
Rapid arrhythmias
Anaemia
Rarities:
• vasculitis
• trauma
• collagen disease

Note: Look for fever and tachycardia. Rule out anaemia and thyrotoxicity.

Variants[1,10]

- *Stable angina.* Pain occurs with exertion and is usually predictable with no symptom change during the past month.
- *Unstable angina* (also referred to as crescendo angina, pre-infarct angina and acute coronary insufficiency). It is increasing angina (severity and duration) over a short period of time, precipitated by less effort and may come on at rest, especially at night. It may eventually lead to complete infarction, often with relief of symptoms. It is due to unstable plaque.

  Nocturnal angina. Pain occurs during the night. It is related to unstable angina.

  Decubitus angina. The pain occurs when lying flat and is relieved by sitting up.

  Variant angina or Prinzmetal angina or spasm angina.[5] The pain occurs at rest and without apparent cause. It is associated with typical transient ECG changes of ST elevation (as compared with the classic changes of ST depression during effort angina). It can lead to infarction and cause arrhythmias. It is caused by coronary artery spasm.

Aids to diagnosis

ECG

This may be normal or show ischaemia or evidence of earlier infarction. During an attack it may be normal or show well-marked depression of the ST segment, symmetrical T-wave inversion (see FIG. 30.12) or tall upright T waves.

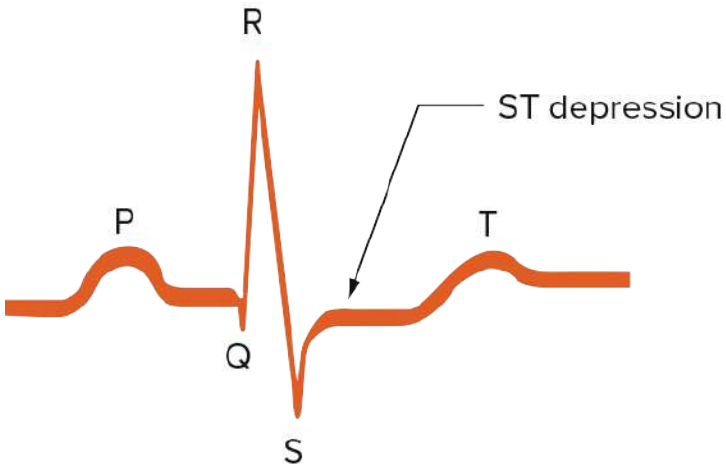

FIGURE 30.12 Typical ECG pattern for angina pectoris: this tracing is usually observed during an attack

Note: There is no specific ECG of angina; the most that can be said is that an ECG is consistent with angina

Exercise ECG

This is positive in about 75% of those with severe coronary artery disease and should be performed if the diagnosis is in doubt, for prognostic reasons or to aid in the timing of additional investigations (e.g. coronary angiography). A normal stress test does not rule out coronary artery disease.

Exercise thallium-201 scan

This test is helpful in some difficult circumstances such as in the presence of left branch bundle block (LBBB), old infarction and Wolff–Parkinson–White (WPW) syndrome (when exercise test is of little use) and with mitral valve prolapse, which gives high false-positive tests. It helps determine the presence and extent of reversible myocardial ischaemia since thallium is only taken up by perfused tissue.

Ambulatory ECG Holter monitoring

Occasionally useful for detecting intermittent rhythm disturbances.

Gated blood pool nuclear scan

This test assesses the ejection fraction, which is an index of ventricular function and thus aids assessment of patients for coronary artery bypass surgery.

Echocardiography

This assesses global and regional wall motion abnormalities, valvular dysfunction and pericardium status.

Coronary angiography

This test accurately outlines the extent and severity of coronary artery disease (see FIG. 30.13). It is usually used to determine the precise coronary artery anatomy prior to surgery. CT angiography provides a safer alternative in many circumstances.

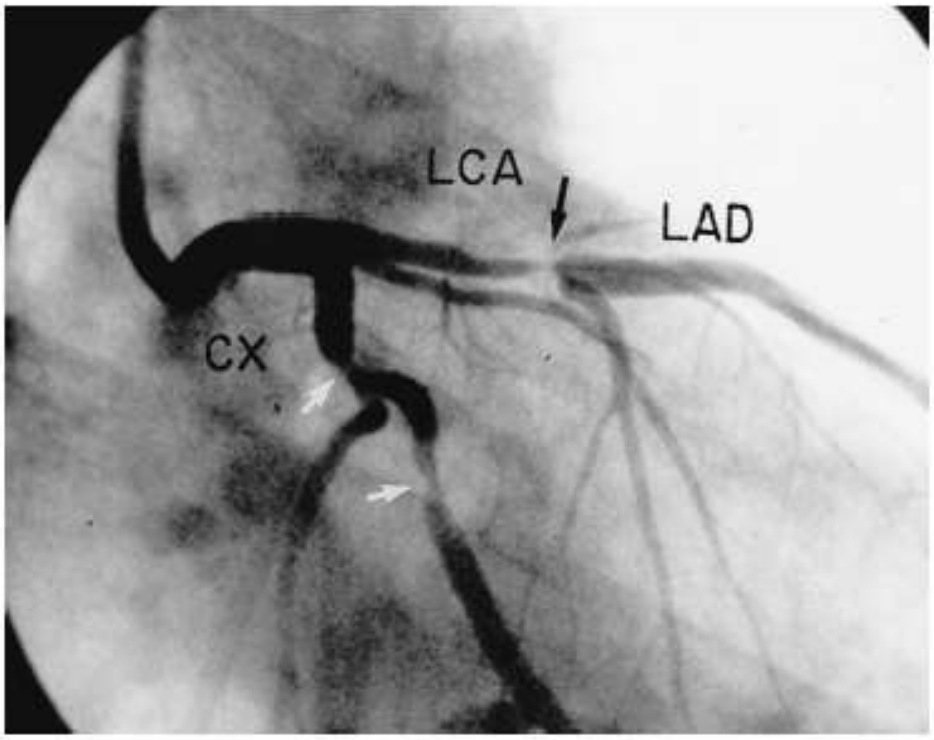

FIGURE 30.13 Coronary angiogram of a left coronary artery (*LCA*) with a tight stenosis in the proximal left anterior descending (*LAD*) artery (*black arrow*). The circumflex artery (*CX*) has two moderately severe stenoses (*white arrows*).

The relationship between the degree of angina and coronary artery disease is not clear-cut. Some people with severe angina have normal coronary arteries.

Indications for coronary angiography are presented in TABLE 30.8 .

Table 30.8 Indications for coronary angiography

Strong positive exercise stress test
Suspected left main coronary artery disease
Angina resistant to medical treatment
Suspected but not otherwise proven angina
Acute coronary syndromes
Angina after myocardial infarction
Patients over 30 years with aortic and mitral valve disease being considered for valve surgery

Management of stable angina

Preventions

This is especially important for those with a positive family history and an unsatisfactory lifestyle. Modification of risk factors:

- no smoking
- weight reduction
- healthy eating/optimal low-fat diet
- exercise
- control of hypertension
- control of diabetes
- control of blood lipids

General advice for stable angina

- Reassure patient that angina has a reasonably good prognosis: 30% survive more than 10 years;[10] spontaneous remission can occur.
- Attend to any risk factors.
- If inactive, take on an activity such as walking for 20 minutes a day.
- Take regular exercise to the threshold of angina.

- If tense and stressed, cultivate a more relaxed attitude to life—consider a stress management/relaxation course.
- Avoid precipitating factors.
- Don’t excessively restrict lifestyle.

Medical treatment[5,10]

The acute attack and episodic angina

- Nitrates:

  glyceryl trinitrate 300–600 mcg tab sublingually, max 1800 mcg

  or

  glyceryl trinitrate SL 400 mcg metered dose spray: 1 spray; repeat after 5 minutes if pain persists (maximum three doses)

  or

  isosorbide dinitrate 5 mg sublingually; repeat every 5 minutes if pain persists (maximum 3 tablets)

  or

  aspirin 150 mg (o)

  or if intolerant to nitrates

  nifedipine 5 mg capsule (suck or chew)

Tips about glyceryl trinitrate:

- if pain persists for longer than 10 minutes despite two doses of nitrates, take a third dose and call for an ambulance
- warn about headache and other side effects
- sit down while administering
- take ½ (initially) or 1 tablet or 1 spray every 5 minutes
- take a maximum of 3 doses in 15 minutes
- keep tablets out of light and heat—discard the bottle after being opened for 3 months or after 2 days if carried on the person

- advise patient to get medical advice if no relief after 3 doses

Note: Avoid nitrates if sildenafil or vardenafil used in the previous 24 hours or tadalafil in the previous 5 days.

Prevention of angina[5]

Moderate stable angina

- Regular predictable attacks precipitated by moderate exertion. For prevention:

  add to aspirin (if not contraindicated)

  beta blocker, e.g. atenolol 25 mg (o) once daily, increasing to 100 mg if required

  or

  metoprolol 25 mg (o) twice daily, increasing to 100 mg if required

  plus nitrates

  glyceryl trinitrate 5–15 mg (transdermal patch) daily (use for 14 hours only)

  or

  isosorbide mononitrate 30 mg (o) SR tablet mane, increasing to 120 mg if required

Note: Aim for a daily nitrate-free interval.

Persistent angina

- Not prevented by beta blocker:

  add

  a dihydropyridine calcium-channel blocker (CCB)

  nifedipine CR 30–60 mg (o) once daily

  or

  amlodipine 2.5–10 mg (o) once daily

  plus nitrates

If beta blocker contraindicated (use a non-dihydropyridine calcium-channel blocker):

  diltiazem MR 180–360 mg (o) daily

or

verapamil MR120–480 mg (o) daily

Refractory stable angina

Consider adding nicorandil 5 mg (o) bd, increasing after a week to 10–20 mg bd or replacing the CCB with perhexiline

or

ivabradine and seeking specialist advice

Unstable angina

Includes onset of angina at rest, abrupt worsening of angina and angina following acute myocardial infarction.

- Should be hospitalised for stabilisation and further evaluation. May need IV nitrate therapy.
- The objectives are to optimise therapy and consider coronary angiography with a view to a corrective procedure.

Rules of practice

- For variant angina (spasm) use nitrates and calcium antagonist (avoid beta blockers).
- As a rule, avoid the combination of verapamil and a beta blocker (risk of tachycardia and heart block).
- Do not combine a dihydropyridine CCB with a non-dihydropyridine CCB.
- Tolerance to nitrate use is a problem, so 24-hour coverage with long-acting preparations is not recommended.
- Consider using the potassium-channel-opening vasodilator nicorandil 5 mg (o) bd to 10–20 mg (o) bd (after 1 week). Can use as alternative to long-acting nitrates. The new agent ivabradine can be considered.
- Nitrates can be used prophylactically prior to any exertion that is likely to provoke angina (e.g. glyceryl trinitrate spray or tablet *or* isosorbide dinitrate 5 mg tablet)
- Avoid nitrates if the patient has used a 5 phosphodiesterase inhibitor in the past 1–5 days.

Non-medical treatment[5]

Percutaneous intervention (PCI) and coronary angioplasty (the gold standard)

A common technique is dilating coronary atheromatous obstructions by inflating a balloon against the obstruction—percutaneous transluminal coronary angioplasty (PTCA) (see FIG. 30.14)—and maintaining patency with intracoronary stent devices.

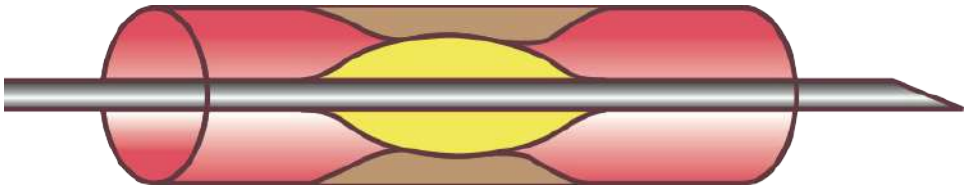

FIGURE 30.14 Percutaneous transluminal angioplasty (PTCA) with an inflatable balloon

Two complications of the balloon inflation angioplasty are acute coronary occlusion (2–4%) and restenosis, which occurs in 30% in the first 6 months after angioplasty.[10]

Intracoronary stents

The evidence supporting coronary stents for stable angina is steadily narrowing due to better designed RCTs.[11] The first ever truly double-blind trial (ORBITA) in 2017 showed no improvement in exercise time vs sham surgery, even for those with severe coronary artery disease (but stable angina). This is supported by other studies showing that adding a stent to optimal medical treatment, in terms of death and MI rates, results in either modest or no improvement. However, stents do result in a reduction in angina symptoms in stable angina, and they also reduce deaths and MIs in acute coronary syndromes.

PTCA followed by stenting is the most favoured procedure to maintain patency of the obstructed coronary vessel (see FIG. 30.15). Drug eluting stents, which include drugs such as pimecrolimus, sirolimus or paclitaxel, can be used as well as the bare metal stent. Stent patients require long-term antiplatelet agents (e.g. aspirin plus clopidogrel) (specialist advice is required).

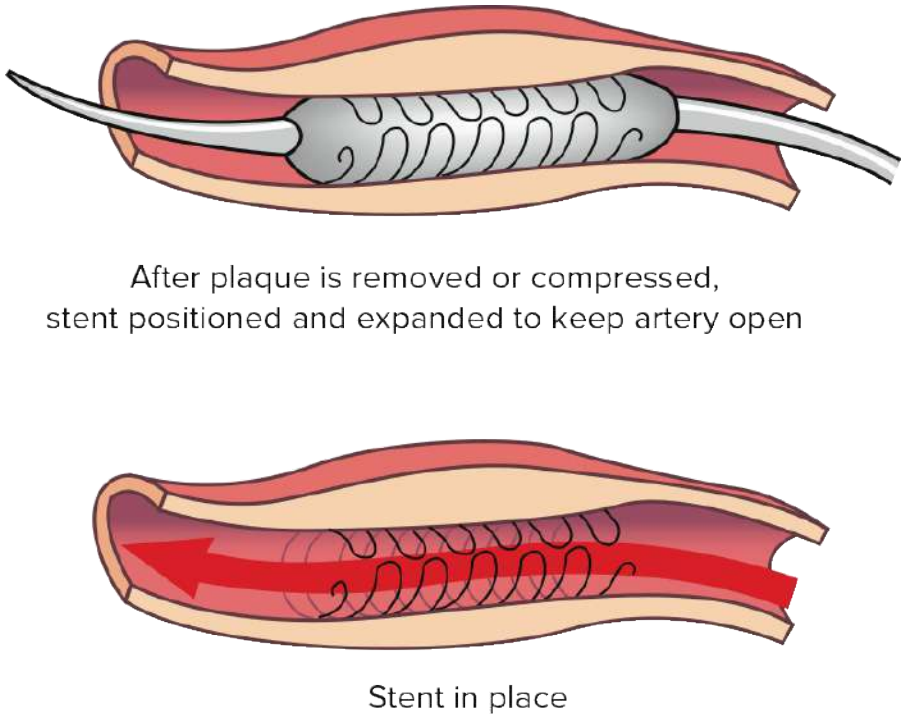

FIGURE 30.15 Illustration of stenting a coronary artery

Coronary artery surgery

The main surgical techniques in current use are coronary artery bypass grafting (CABG) using either a vein (usually the saphenous) (see FIG. 30.16) or internal mammary arterial implantation (see FIG. 30.17) or both, and endarterectomy.

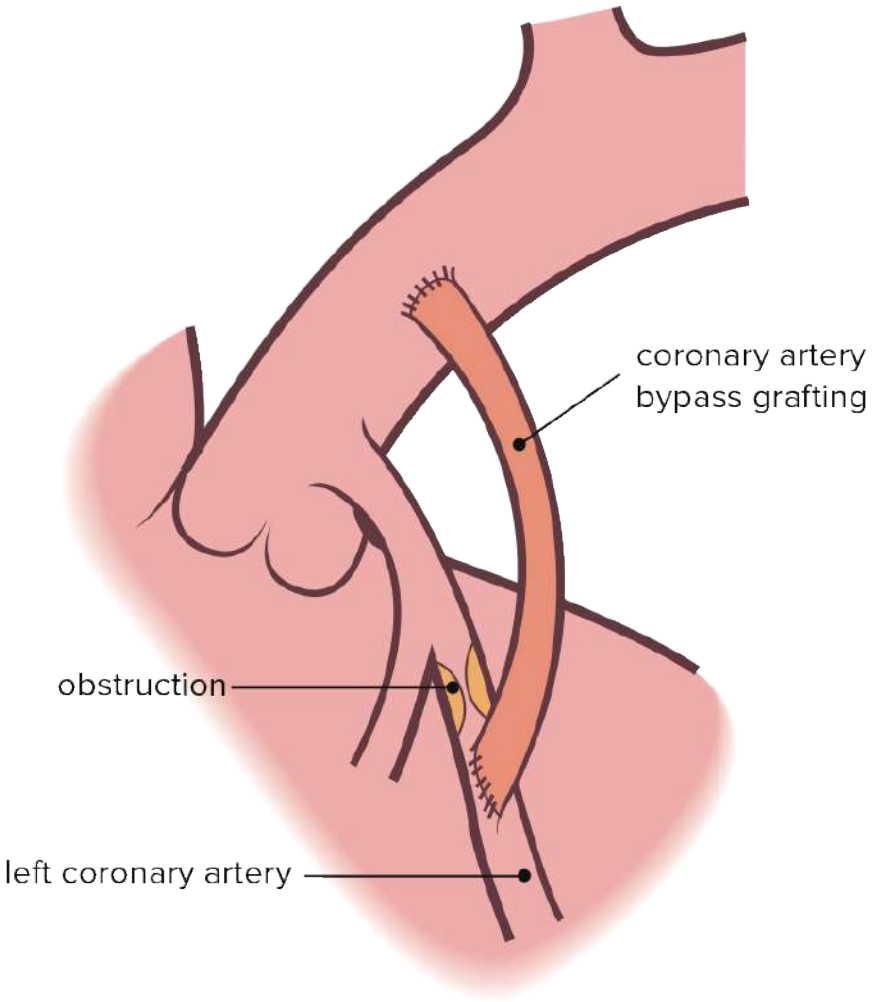

FIGURE 30.16 Coronary artery bypass grafting to relieve coronary obstruction

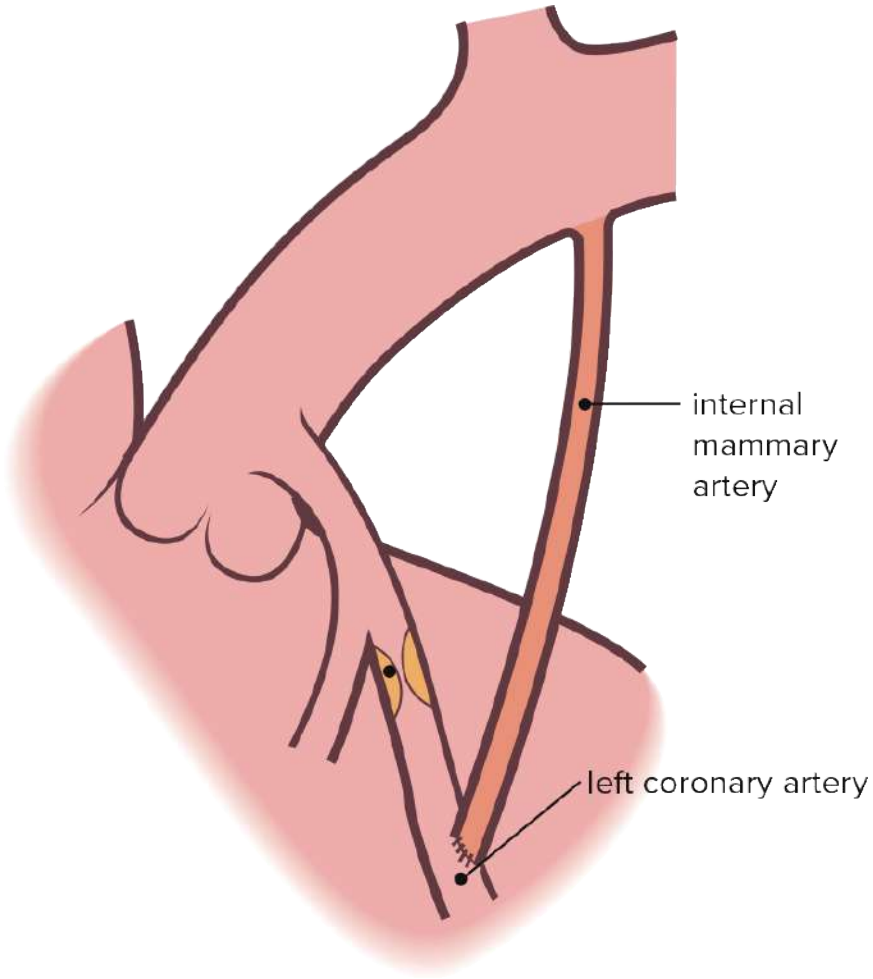

FIGURE 30.17 Internal mammary arterial transplantation to relieve obstruction

Symptomatic patients with significant left main coronary obstruction should undergo bypass surgery, and those with two or three vessel obstruction and good ventricular function are often considered for angioplasty or surgery. A significant improvement in the quantity and quality of life can be expected.

Myocardial infarction

Clinical guidelines[12]

- Variable pain; may be mistaken for indigestion
- Similar to angina but more oppressive
- So severe, patient may fear imminent death—*angor animi*

- About 20% have no pain. These have a high mortality rate
- ‘Silent infarcts’ in females, elderly and those with diabetes or hypertension.
- 60% of those who die do so before reaching hospital, within 2 hours of the onset of symptoms
- In those who arrive alive, hospital mortality is 8–10%[12]
- Like CVA, seems to peak at 6–10 am

Diagnosis is based on 2 out of 3 criteria: history of prolonged ischaemic pain, typical ECG appearance, and rise and fall of cardiac enzymes.

Aetiology

- Thrombosis with occlusion
- Haemorrhage under a plaque
- Rupture of a plaque
- Coronary artery spasm

Signs

These may be:

- no abnormal signs
- pale/grey, clammy, dyspnoeic
- restless and apprehensive
- variable BP with pain ↓ heart pump failure
- variable pulse: watch for bradyarrhythmias
- mild cardiac failure: third or fourth heart sound, basal crackles

Investigations

1. *ECG.* The ECG is valuable with characteristic changes in a full thickness infarction. The typical features (see FIG. 30.18) are:

   - the Q wave: broad (>1 mm) and deep >25% length R wave

     occurs normally in leads AVR and V1; III (sometimes)

     abnormal if in other leads

occurs also with WPW and ventricular tachycardia (VT)

usually permanent feature after full thickness AMI

- T wave and ST segment:

  transient changes (inversion and elevation respectively)

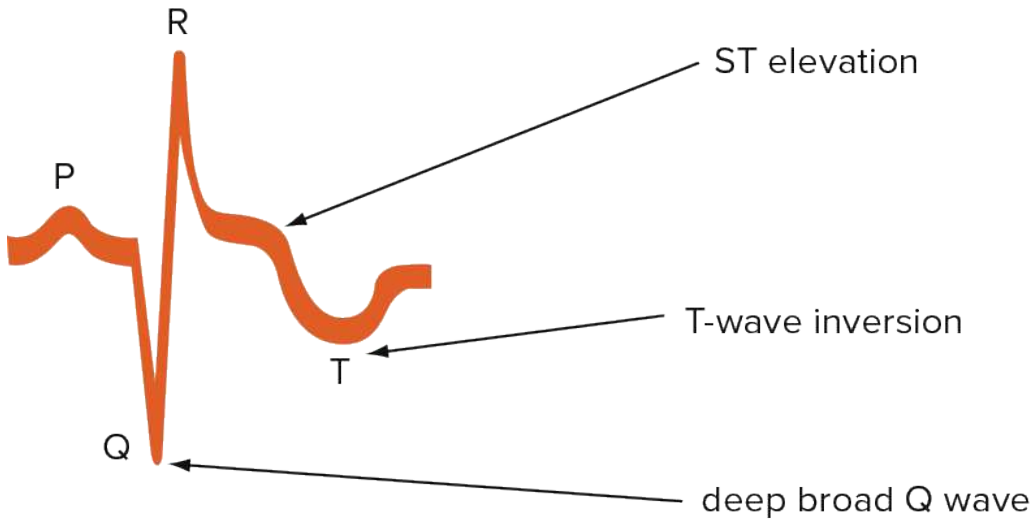

FIGURE 30.18 Typical ECG features of myocardial infarction, illustrating a Q wave, ST elevation and T-wave inversion

The typical progression is shown in FIGURE 30.19 .

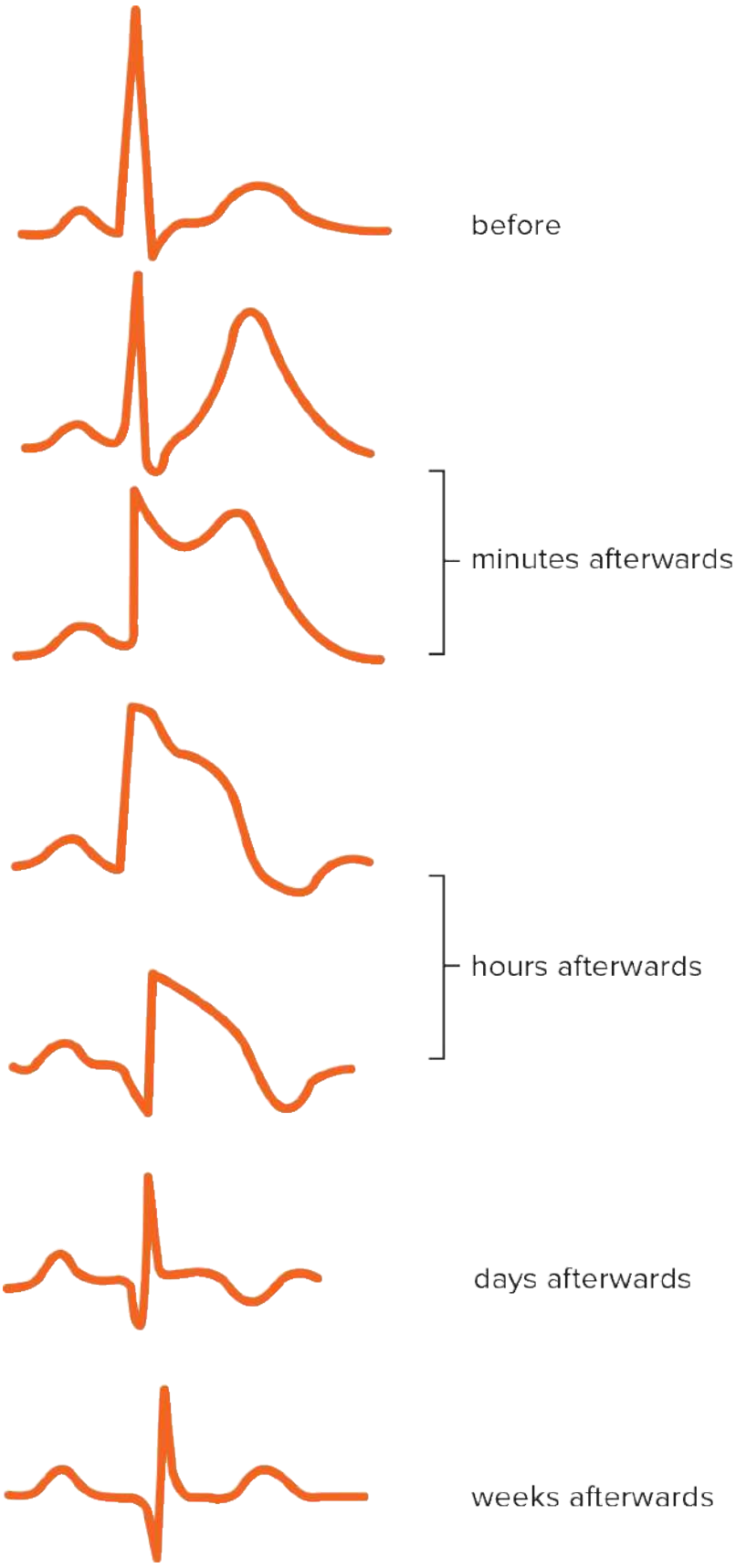
before
minutes afterwards
hours afterwards
days afterwards
weeks afterwards

FIGURE 30.19 Typical evolution of ECG changes with myocardial infarction

Note:

- Q waves do not develop in subendocardial infarction.
- The strategies for management of AMI are based on the distinction between Q wave (transmural) or non-Q wave (subendocardial) infarction.
- Q wave infarction has been proved to benefit from thrombolytic therapy, but non-Q wave infarction has not.
- If new LBBB, think AMI (in LBBB no Q wave).
- A normal ECG, especially early, does not exclude AMI. Q waves may take days to develop.

2. *Cardiac enzymes.* The typical enzyme patterns are presented in FIGURE 30.20 . As a rule, large infarcts tend to produce high serum enzyme levels. The elevated enzymes can help time the infarct:

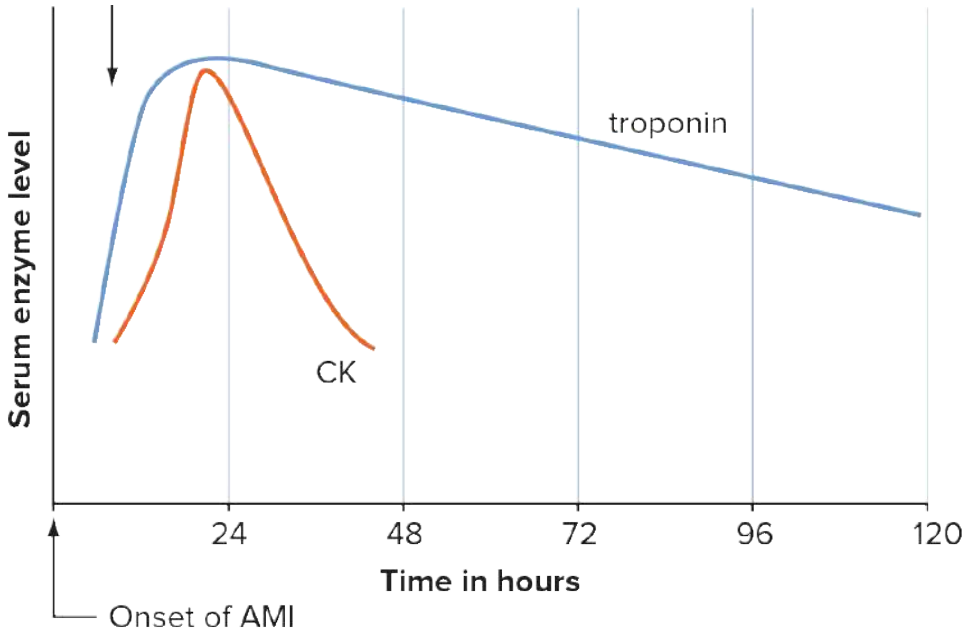

FIGURE 30.20 Typical cardiac enzyme patterns following myocardial infarction

- troponin I or T:

  the preferred test

starts rising at 3–12 hours, peaks at 24 hours and persists for about 5–14 days

positive in unstable angina

raised in aortic dissection and kidney impairment

may have to wait until 10 hours before recording a negative result

not useful for repeat MI

both proteins, I and T, provide same information

reference interval <0.1 µg/L

- creatinine kinase (CK):

  after delay of 6–8 hours from the onset of pain it peaks at 20–24 hours and usually returns to normal by 48 hours

  CK–MB: myocardial necrosis is present if >15% of total CK; unlike CK, it is not affected by intramuscular injectionsFurther management of STEMI

3. *Technetium pyrophosphate scanning*

   - Performed from 24 hours to 14 days after onset.
   - Scans for 'hot spots', especially when a posterolateral AMI is suspected and ECG is unhelpful because of pre-existing LBBB.

4. *Echocardiography.* This is used to assist diagnosis when other tests are not diagnostic.

Note: The clinical diagnosis may be the most reliable, as the ECG and enzymes may be negative.

Management of acute coronary syndromes

General principles[12,13]

- Aim for immediate attendance if suspected.
- Pre-hospital: make diagnosis, assess risk, ensure stability. A 12-lead ECG should be arranged ASAP.
- Call a mobile coronary care unit.
- Achieve coronary perfusion and minimise infarct size.
- Prevent and treat cardiac arrest; have a defibrillator available to treat ventricular fibrillation.

- Optimal treatment is in a modern coronary care unit (if possible) with continuous ECG monitoring (first 48 hours) and a peripheral IV line (consider intranasal oxygen only if hypoxaemic <94% saturation).
- Pay careful attention to relief of pain and apprehension.
- Establish a caring empathy with the patient.
- Give aspirin as early as possible (if no contraindications): 300 mg chewed or dissolved sublingually.
- Prescribe a beta blocker and an ACE inhibitor early (if no contraindications).

Note: For a STEMI it is important to re-establish flow as soon as possible, usually by either thrombolytic therapy or primary angioplasty (preferably with stenting). Rescue angioplasty is usually used when large infarcts have not perfused at 60–90 minutes.[5]

Hospital management[5,12]

- As for first-line management.
- Confirm ECG diagnosis: STEMI or NSTEACS.
- Take blood for cardiac enzymes, particularly troponin, urea and electrolytes.
- Organise an urgent cardiology consultation for risk stratification and a decision whether to proceed to coronary angiography and coronary reperfusion with PCI (or CABG) or with thrombolysis.

Management of STEMI[13]

The optimal first-line treatment for the patient with a STEMI is urgent referral to a coronary catheter laboratory ideally within 60 minutes (the golden hour) of the onset of pain for assessment after coronary angiography for percutaneous transluminal coronary angioplasty (PTCA). If available and performed by an interventional cardiologist it has the best outcomes (level I evidence).

The principle is to achieve rapid reperfusion via primary angioplasty with a stent (optimal stent status currently under evaluation).

Adjunct therapy will include dual antiplatelet therapy—aspirin and a $P2Y_{12}$ inhibitor (clopidogrel, ticagrelor or prasugrel) and anticoagulation with low molecular weight heparin or unfractionated heparin, a statin and an ACE inhibitor.

Urgent reperfusion guidelines

- Within 60 minutes of symptom onset STEMI: PCI (optimal)

- Within 90 minutes of onset STEMI: PCI (acceptable)
- If these targets are not reached: fibrinolysis within 30 minutes of arrival
- For patients presenting >12 hours after onset of symptoms, consider reperfusion if:

  continuing ischaemia

  viable myocardium

  major complications, e.g. cardiogenic shock[5]

First-line management acute chest pain of possible cardiac origin (e.g. outside hospital)

- Perform an ECG and classify ACS into STEMI or NSTEACS, and notify the medical facility that will receive the patient (discuss over the telephone). The ECG is the sole test required to select patients for emergency perfusion
- Oxygen 4–6 L/min only if hypoxaemic (aim to keep P_aO_2 >90%)
- Secure an IV line (withdraw blood for tests, especially troponin levels)
- Glyceryl trinitrate (nitroglycerin) 300 mcg (½ tab) SL or spray 400 mcg (every 5 minutes as necessary to maximum of three doses). Beware of sildenafil (Viagra) and related drugs use and bradycardia—correct with atropine
- Aspirin 300 mg
- Morphine 2.5–5 mg IV statim bolus: then 1 mg/min every 5–10 mins until pain relief (up to 15 mg) or fentanyl 25–50 mcg IV (If feasible, it is preferable to give IV morphine 1 mg/min until relief of pain; this titration is easier in hospital.)

Management of NSTEACS

NSTEACS can progress to a STEMI; therefore all patients with NSTEACS should have ongoing monitoring in hospital and their risk stratified to direct management decisions.

Reperfusion therapy[14,15]

All patients with acute myocardial infarction should be considered for admission to a coronary care unit for monitoring and expert care. The decision of reperfusion therapy by PCI or fibrinolytic therapy will be determined by unit policy based on availability of PCI.