

tools to help cope with OA.

- *Braces, orthotics, walking aids.* A walking stick or wheezy walker may help. However, realignment braces for medial compartment or patellofemoral OA have not been shown to be helpful, nor have lateral wedge shoe insoles or knee taping. Advise sensible, supportive footwear.
- *Paracetamol.* Use paracetamol (acetaminophen) regularly, or before activity. Avoid combinations containing codeine or dextropropoxyphene. The newer 665 mg products marketed specifically for OA have no great advantage over the traditional 500 mg tablets; use either.
- *NSAIDs and COX-2 specific inhibitors (CSIs).* These are second-line drugs for more persistent pain not relieved by paracetamol or where there is evidence of inflammation, such as pain worse with resting and nocturnal pain. Systematic reviews found that oral NSAIDs probably reduce the pain of OA but there is no good evidence that NSAIDs are superior to paracetamol or that any one of the many NSAIDs is more effective than others.<sup>16</sup> The risk versus benefit equation always has to be weighed carefully. As a rule, use the lowest effective dose for a short period, then discontinue use if not effective. Evidence shows CSIs (celecoxib, etoricoxib) have a similar efficacy to other NSAIDs and a modest absolute reduction in GIT complications.<sup>18</sup> Significant risks of NSAIDs are:

gastric ulceration, erosion with bleeding

depression of kidney function (check kidney function beforehand)

hepatotoxicity

Topical NSAIDs and capsaicin have been shown to have a small benefit in pain relief over topical placebo preparations.<sup>12</sup>

*Note:* Change to a suppository form will not necessarily render the upper GIT safe from irritation.

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- *Intra-articular (IA) corticosteroids.* Corticosteroid injections have a modest place for offering short-term pain relief, as an adjunct to other measures. They can be particularly useful during an inflammatory episode of distressing pain and disability (e.g. a flare-up in an osteoarthritic knee), or where a joint replacement is either under consideration or contraindicated due to comorbidities or age.
- *Surgery.* Refer for surgical intervention if debilitating and intractable pain or disability, and when considering joint replacement. However, keep up to date with evidence from blinded ‘surgery vs sham-surgery trials’ and systematic reviews that suggest that the historic enthusiasm for knee and shoulder arthroscopies is no longer justifiable except in limited circumstances.<sup>19,20</sup>
- *Glucosamine, chondroitin, vitamin D, omega-3 fatty acids.* Evidence originally supporting oral glucosamine was from small trials prone to bias, including publication bias. Systematic

reviews suggest that it should no longer be recommended.<sup>14</sup> Nor should chondroitin, vitamins or omega-3 supplementation be recommended.

- *Viscosupplementation and stem cell therapy.* Intra-articular hyaluronic acid weekly for 3–5 weeks, especially for OA of knee. Conflicting evidence for efficacy.<sup>12</sup> Evidence originally supporting intra-articular injection of hyaluronic acid suffered the same fate as glucosamine, and no good evidence supports stem cell therapy.
- *Bisphosphonates.* These are used to prevent osteoporotic fractures, where appropriate, but do not advise they will have any impact on OA symptoms.
- *Contraindicated drugs.* For OA these include the immunosuppressive and disease-modifying drugs such as oral corticosteroids, gold, antimalarials and cytotoxic agents. Avoid long-term opioids, which won't help but will harm.

## Rheumatoid arthritis

RA, which is an autoimmune symmetrical polyarticular systemic disease of unknown aetiology, is the commonest chronic inflammatory polyarthritis and affects about 1–2% of the population. The disorder can vary from a mild to a most severe debilitating expression. About 10–20% of patients have a relentless progression and require aggressive drug therapy.<sup>21</sup> Urgent referral to a specialist is recommended.

Genetic factors may represent a risk of 15–70% of developing RA.

### The arthritis

RA generally presents with the insidious onset of pain and stiffness of the small joints of the hands and feet. The pain is persistent rather than fleeting and mainly affects the fingers where symmetrical involvement of the PIP joints produces spindling while the metacarpophalangeal joints develop diffuse thickening, as does the wrist (see FIG. 25.8). In 25% of cases RA presents as arthritis of a single joint such as the knee,<sup>13</sup> a situation leading to confusion with a spondyloarthropathy. Differential diagnosis is polyarticular gout.

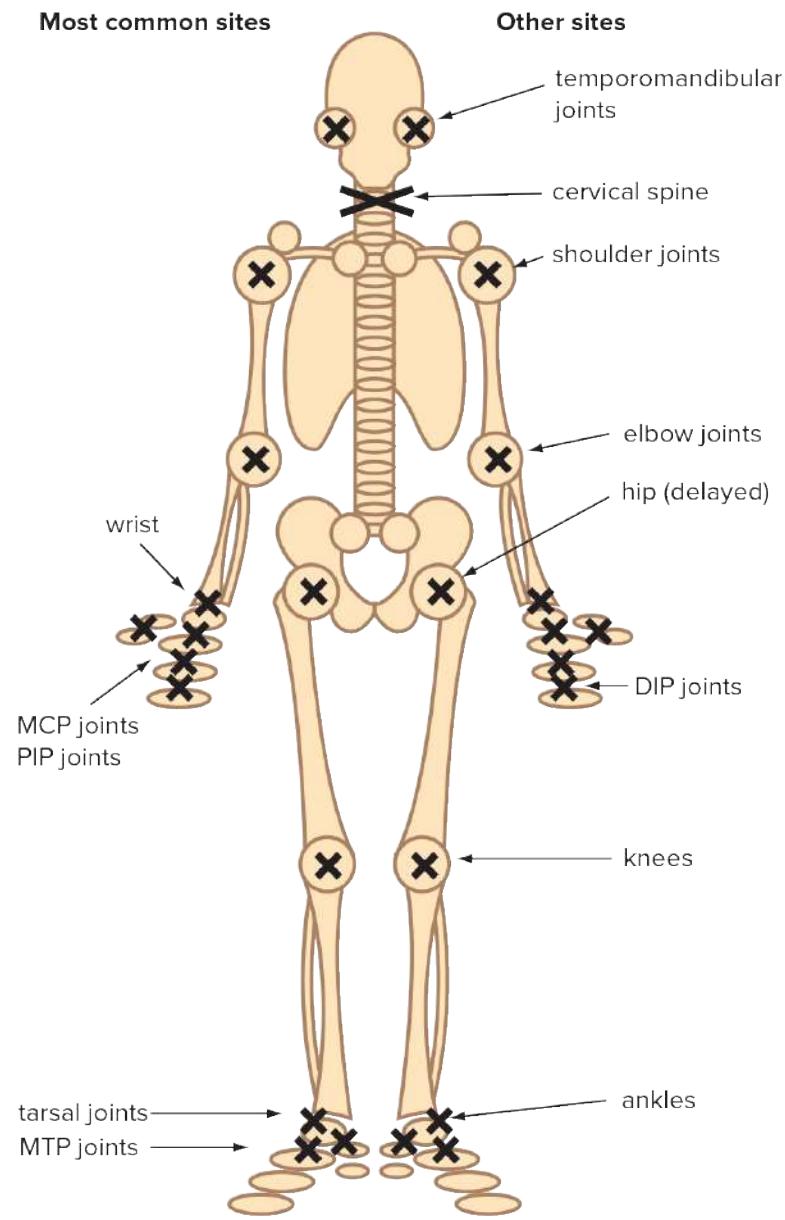


**FIGURE 25.8** Chronic rheumatoid arthritis showing classic features of deformities including subluxation of joints and rheumatoid nodules

## Joints involved

- Hands: MCP and PIP joints, DIP joints (30%)
- Wrist and elbows
- Feet: MTP joints, tarsal joints (not IP joints), ankle
- Knees (common) and hip (delayed—up to 50%)
- Shoulder (glenohumeral) joints
- Temporomandibular joints
- Cervical spine (not lumbar spine)

Refer to [FIGURE 25.9](#) .



**FIGURE 25.9** Rheumatoid arthritis: typical joint distribution

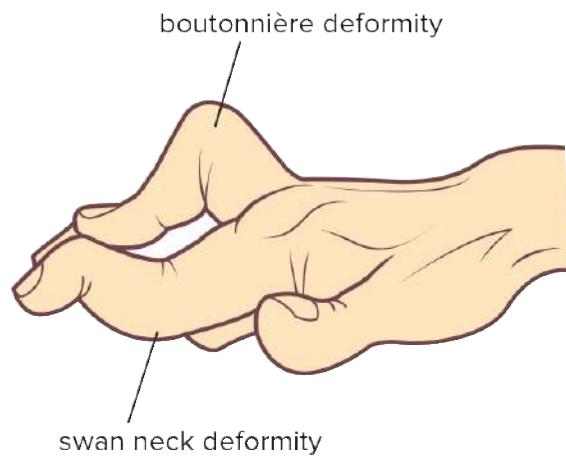
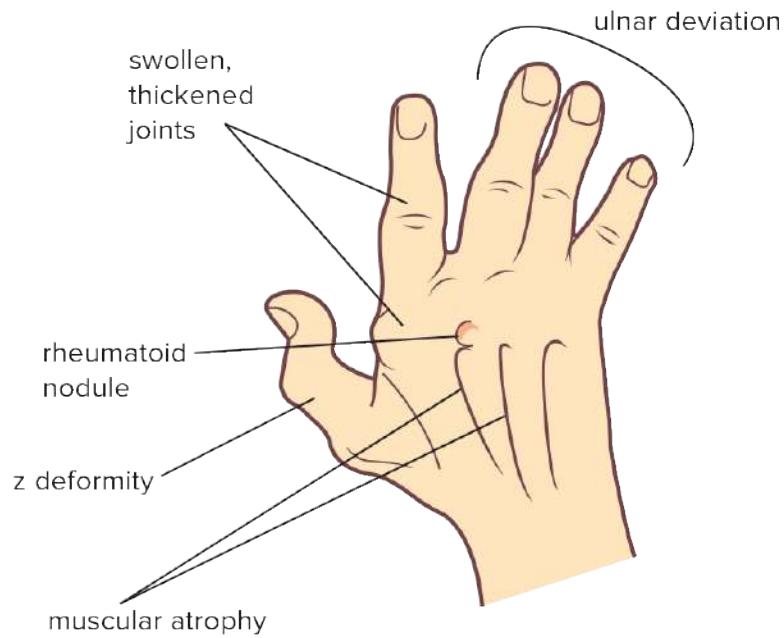
### Clinical features

- Insidious onset but can begin acutely (explosive RA)
- Any age 10–75 years: peak 30–50 years but bimodal 25–50 (peak age) and 65–75
- Female to male ratio = 3:1
- Joint pain: worse on waking, nocturnal pain, disturbed sleep; relieved with activity

- Morning stiffness—can last hours
- Rest stiffness (e.g. after sitting)
- General: malaise, weakness, weight loss, fatigue
- Disability according to involvement

## Signs

- Soft swelling (effusion and synovial swelling), especially of wrist, MCP and PIP joints, nodules
- Warmth
- Tenderness on pressure or movement
- Limitation of movement
- Muscle wasting
- Later stages: deformity, subluxation, instability or ankylosing
- Look for swan necking, boutonnière and z deformities, ulnar deviation (see FIG. 25.10 )



**FIGURE 25.10** Chronic rheumatoid arthritis: typical signs

- Check for a number of everyday functions, for example:

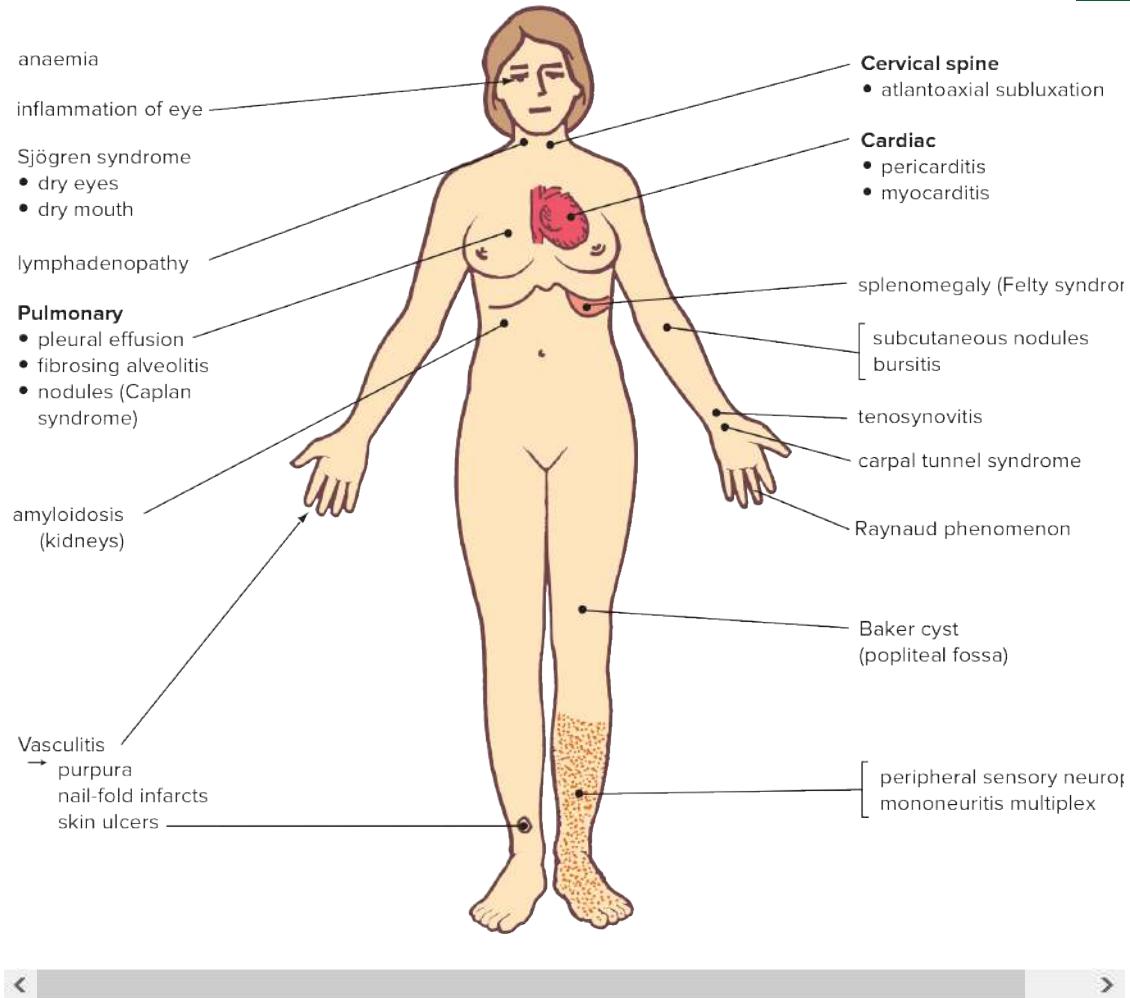
power grip (lifting a jug of water)

precision grip (using a key or pen), undoing buttons

hook grip (carrying a bag)

The various possible extra-articular manifestations are summarised in **FIGURE 25.11**.

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**FIGURE 25.11** Rheumatoid arthritis: significant non-articular clinical manifestations

## Investigations

- ESR/CRP usually raised according to activity of disease
- Anaemia (normochromic and normocytic) may be present
- Rheumatoid factor
  - positive in about 70–80% (less frequent in early disease)
  - 15–25% of RA patients will remain negative<sup>21</sup>

- Anti-cyclic citrullinated peptide (anti-CCP) antibodies: more specific for RA (94% specificity)<sup>12</sup>
- X-ray changes:
  - erosion of joint margin
  - loss of joint space (may be destruction)
  - juxta-articular osteoporosis
  - cysts
  - advanced: subluxation or ankylosing
- MRI—helpful for early diagnosis

Criteria for the diagnosis of RA are presented in TABLE 25.3 .

**Table 25.3** Revised criteria for features suggestive of rheumatoid arthritis<sup>12</sup>

- Family history of inflammatory arthritis
- Symptom duration of >6 weeks
- Early-morning stiffness of >1 hour
- Arthritis in three or more regions
- Swelling in five or more joints
- Bilateral compression tenderness of the metatarsophalangeal joints
- Symmetry of the areas affected
- Presence of rheumatoid nodules
- Rheumatoid factor positivity
- Raised inflammatory markers (ESR/CRP) in absence of infection
- Anticyclic citrullinated peptide antibody positivity
- Bony erosions evident on radiographs of the hands or feet, although these are uncommon in early disease

## Key points

- If the RA factor is positive, it is non-specific—order the anti-CCP antibody to confirm the diagnosis.
- RA has a strong cardiovascular risk factor.

## Principles of management<sup>12,22</sup>

- Give patient education support and appropriate reassurance. The diagnosis generally has distressful implications, and so the patient and family require careful explanation and support. Some have little or no long-term problems but even in mild cases, continuing care and medical supervision is important.
- There has been a radical shift from palliation to early induction of disease remission, to prevent joint damage and reduce morbidity from malignancy (especially lymphoma) and cardiovascular disease.
- Since many studies show disease progression in the first 2 years, relative aggressive treatment with disease-modifying antirheumatic drugs (DMARDs) from the outset is advisable, rather than to start stepwise with analgesics and NSAIDs only.<sup>23</sup>
- Use a team approach where appropriate, including an early specialist referral for obvious or suspected RA or positive anti-CCP for diagnosis and collaborative support.
- Fully assess the person's functional impairment and impact on home life, work and social activity. Involve the family in decision making.
- Make judicious use of pharmaceutical agents. For serious cases, consultant collaboration is essential.
- Review regularly, continually assessing progress and drug tolerance. The disease activity can be monitored with plain X-rays, ultrasound of hands (especially if hands are thick), CRP ± ESR.

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## Specific advice

- *Rest and splinting.* This is necessary where practical for any acute flare-up of arthritis.
- *Exercise.* It is important to have regular exercise, especially walking and swimming. Have hydrotherapy in heated pools.
- *Smoking cessation.* This is strongly recommended.
- *Referral.* Referring to physiotherapists and occupational therapists for expertise in exercise supervision, physical therapy and advice regarding coping in the home and work is important.
- *Joint movement.* Each affected joint should be put daily through a full range of motion to keep it mobile and reduce stiffness.
- *Diet.* Although there is no special diet that seems to cause or cure RA, a nourishing, well-balanced diet is common sense and obesity must be avoided. Some evidence supports both a

Mediterranean diet and vegetarianism.<sup>24</sup> There is also some evidence that avoiding animal fats (dairy products and some meats) and using fish oils is beneficial.<sup>25</sup>

Therapies used in the management of rheumatoid arthritis are presented in TABLE 25.4 .

**Table 25.4** Therapies used in the management of rheumatoid arthritis<sup>22,26</sup>

Education (rest, literature, weight loss, joint protection advice)

NSAIDs

Simple analgesics

DMARDs:

Conventional synthetic DMARDs

- Immunosuppressants:

azathioprine

cyclosporin

leflunomide

methotrexate

Biological DMARDs

- Cytokine inhibitors

anti-TNF  $\alpha$  agents: abatacept, adalimumab, certolizumab, etanercept, infliximab, golimumab, rituximab

anti-interleukin-1 agents; tocilizumab

Gold salts

Quinolones:

- hydroxychloroquine
- chloroquine

Others:

- D-penicillamine
- sulfasalazine

Glucocorticoids:

- oral prednisolone
- intra-articular
- intravenous (steroid ‘pulses’)

Fish body oil

Physical therapy (hydrotherapy, isometric exercises)

Occupational therapy (splints, aids and appliances)

Orthopaedic surgery (synovectomy, joint replacement, arthrodesis, plastic hand surgery)

## Chiropody, footwear, insoles

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Source: Reilly and Littlejohn<sup>22,26</sup>

## Management (pharmacological)

Best under consultant direction.

- NSAIDs are effective and still have a place, but the adverse effects are a problem.
- The use of DMARDs and biological DMARDs improves long-term outcomes.
- Methotrexate is the ‘backbone’ of treatment, and should be continued when starting other DMARDs.
- Supplementation with folic acid can improve gastrointestinal symptoms and reduce the risk of liver dysfunction.
- Beware of the increased risk of infection in patients on combination DMARD regimes.
- When indicated, vaccination for pneumococcus, influenza, hepatitis A and B and HPV is recommended for all DMARDs.
- Any pain should be managed with paracetamol or NSAIDs. Avoid opioid analgesics if possible.
- Glucocorticoids are appropriate for flares of RA.

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## Fish oil

Fish oil in doses to deliver 4 g of omega-3 long-chain polyunsaturated fatty acids daily (typically 0.2 g/kg) over several months has been shown to reduce symptoms and the need for NSAIDs through its anti-inflammatory activity.<sup>12,25</sup>

## Glucocorticoids

Oral use should be considered in those with severe disease as a temporary adjunct to DMARD therapy and where other treatments have failed or are contraindicated.

The dose is prednisolone 10 mg (o) daily. Avoid doses higher than 15 mg daily if possible.

Intra-articular injections of depot preparations are effective in larger joints.

## Disease-modifying antirheumatic drugs (DMARDs)

These agents target synovial inflammation and prevent joint damage. The choice depends on several factors, but is best left to the specialist coordinating care. In most patients with recently diagnosed RA, methotrexate is the cornerstone of management and should be commenced as early as possible.

Initial dose: methotrexate 5–10 mg (o) once weekly on a specified day, increasing to maximum of 25 mg weekly or SC depending on clinical response and toxicity. Add folic acid 5–10 mg twice weekly (not on the day methotrexate is given).<sup>12</sup>

Biological DMARDs (bDMARDs) are the newer agents, which should be considered if remission is not achieved with appropriate methotrexate monotherapy, ‘triple therapy’ or other combinations. All bDMARDs are more effective when combined with methotrexate. As a rule, don’t use two biologicals together.

*Warning:* All practitioners should be aware of the increased risk of infectious diseases such as the atypical pneumonias, tuberculosis and listeriosis while taking bDMARDs. All patients should report unusual or unexpected fever or symptoms. Injection site reactions are common.

### Standard initial drug therapy

Monotherapy with methotrexate (or occasionally another DMARD) is standard. Less than 20% will reach disease remission; if not achieved, increase the dose or consider combination therapy. Many people are managed on conventional DMARDs.

### Combination therapy

Consider standard triple therapy: methotrexate + sulfasalazine + hydroxychloroquine.

Triple therapy can be used if methotrexate monotherapy has failed or initially on diagnosis, depending on the severity of the disease. Monitoring for FBE, LFTs and annual eye checks is necessary.

Several other double combinations may be used (e.g. methotrexate with cyclosporin, leflunomide or a bDMARD).

## Connective tissue diseases

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The connective tissue disorders have the common feature of arthritis or arthralgia. Refer to CHAPTER 21 .

Arthritis is the commonest clinical feature of SLE (over 90%).<sup>13</sup> It is a symmetrical polyarthritis involving mainly small and medium joints, especially the proximal interphalangeal and carpal

joints of the hand. It is usually non-erosive and non-deforming, although deformities of fingers and thumbs can occur due to laxity of ligaments, tendons and capsules, causing joint instability.

The initial presentation is similar to RA.

Scleroderma can present as a polyarthritis affecting the fingers of the hand in 25% of patients, especially in the early stages. Soft tissue swelling produces a ‘sausage finger’ pattern.

Arthralgia and arthritis occur in about 50% of those with polymyositis/dermatomyositis and may be the presenting feature before the major feature of muscle weakness and wasting of the proximal muscles of the shoulder and pelvic girdles appear. The small joints of the hand are usually affected and it may resemble RA.

## Crystal arthritis

Arthritis, which can be acute, chronic or asymptomatic, is caused by a variety of crystal deposits in joints. The three main types of crystal arthritis are monosodium urate (gout), calcium pyrophosphate dihydrate (CPPD) and calcium phosphate (usually hydroxyapatite).<sup>27</sup> Refer to TABLE 25.5 .

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**Table 25.5** Crystal-induced disorders

| Crystals                               | Associated disease/syndrome       | Typical joints or region affected                                       |
|--|-----------------------------------|---|
| Monosodium urate                       | Acute gout                        | Metatarsophalangeal joint of big toe                                    |
|  | Tophaceous gout                   | Also: other foot joints, ankle, knee and patellar bursa, wrist, fingers |
|  | Asymptomatic                      |   |
|  | Chronic gouty arthritis           |   |
| Calcium pyrophosphate dihydrate (CPPD) | Acute pseudogout                  | Knee, wrist   |
|  | Destructive arthropathy (like RA) | In older people >60 years (average age 72)<br>F > M (2.7:1)             |
|  | Asymptomatic (most common)        |   |
|  |                                   |   |
| Basic calcium phosphate                | Acute calcific periarthritis      | Shoulder (supraspinatus)  |
|  | Destructive                       |   |

## ⌚ Gout (monosodium urate crystal disorder)

Gout is an abnormality of uric acid metabolism resulting in hyperuricaemia and urate crystal deposition. Urate crystals deposit in:

- joints—acute gouty arthritis
- soft tissue—tophi and tenosynovitis
- urinary tract—urate stones

Four typical stages of gout are recognised:

- *Stage 1*—asymptomatic hyperuricaemia
- *Stage 2*—acute gouty arthritis
- *Stage 3*—intercritical gout (intervals between attacks)
- *Stage 4*—chronic tophaceous gout and chronic gouty arthritis

Asymptomatic hyperuricaemia:

- 10 times more common than gout<sup>13</sup>
- Elevated serum uric acid ( $>0.42$  mmol/L in men,  $> 0.36$  mmol/L in women)
- Absence of clinical manifestations
- Usually does not warrant treatment

### Clinical features

Typical clinical features of gout include:<sup>12</sup>

- mainly a disorder of men (5–8% prevalence)
- onset earlier in men (40–50) than women (60+)
- acute attack: excruciating pain in great toe (see FIG. 25.12 ), early hours of morning
- skin over joint—red, shiny, swollen and hot
- exquisitely tender to touch

- relief with colchicine, NSAIDs, corticosteroids
- can subside spontaneously (3–10 days) without treatment



**FIGURE 25.12** Gout showing typical red, shiny, swollen arthritis of the first MTP joint with desquamation of the skin

### Causes/precipitating factors

- Foods: seafood, meat, liver, kidney
- Alcohol excess (e.g. binge drinking)
- Surgical operation
- Starvation, dehydration, acute illness
- Drugs (FACT: frusemide, aspirin, alcohol, cytotoxic drugs, thiazide diuretics)
- Chronic kidney disease
- Myeloproliferative disorders
- Lymphoproliferative disorders (e.g. leukaemia)
- Sugary soft drinks,<sup>28</sup> fruit juices containing fructose
- Cytotoxic agents (tumour lysis)
- Hypothyroidism

- Low-dose aspirin
- Others

## The arthritis

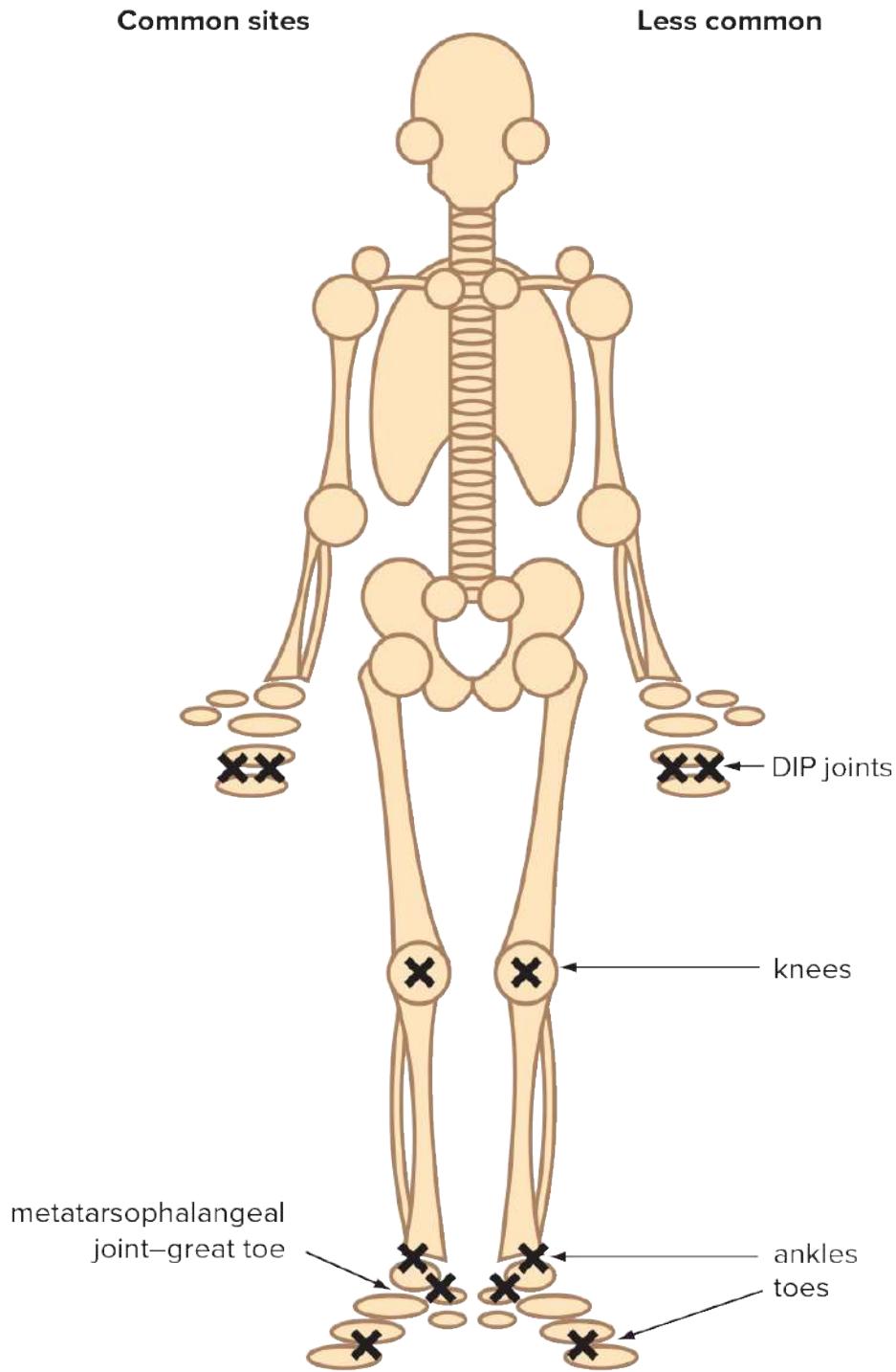
Monoarthritis in 90% of attacks:

- MTP joint great toe—75%
- other joints—usually lower limbs: other toes, mid foot, ankles, knees

Polyarticular onset is more common in old men and may occur in DIP and PIP joints of fingers.  
No synovial joint is immune.

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Refer to [FIGURE 25.13](#) .



**FIGURE 25.13** Gout: possible joint distribution

#### Other features

- Prone to recurrence

- Tophi in ears, elbows (olecranon bursa), big toes, fingers, Achilles tendon (can take many years)
- Can cause patellar bursitis
- Can get cellulitis (does not respond to antibiotics)

### Nodular gout

More common in postmenopausal women with kidney impairment taking diuretic therapy. Causes pain and tophaceous deposits around osteoarthritic interphalangeal (especially DIP) joints of fingers.

### Diagnosis

- Synovial fluid aspirate of affected joint, bursa or tophus → typical uric acid crystals using compensated polarised microscopy; this should be tried first (if possible) as it is the only real diagnostic feature
- Elevated serum uric acid (up to 30% can be within normal limits with a true acute attack)<sup>27</sup>
- X-ray: punched out erosions at joint margins

### Management

Management of gout includes these principles:

- good advice and patient education information
- provision of rapid pain relief
- preventing further attacks
- prevention of destructive arthritis and tophi
- dealing with precipitating factors and comorbid conditions (e.g. alcohol dependence, obesity, CKD, polycythaemia vera, diabetes, hypertension)

### The acute attack<sup>12,28,29</sup>

NSAIDs (except aspirin), in full dosage, are first-line and effective.

Give orally until symptoms abate (up to 4–5 days) then continue for one week

*or*

Corticosteroids:

prednisolone 15–30 mg (o) daily until symptoms abate,<sup>30,31</sup> then decrease gradually

*or*

local corticosteroid injection (but very painful) up to a maximum of two affected sites<sup>31</sup>

*or*

intramuscular (in difficult cases) e.g. tetracosactrin 1 mg

*or*

Colchicine:

- colchicine 1 mg (o) statim, then 0.5 mg 1 hour later as a single dose 1-day course (total dose is 1.5 mg)<sup>12,28</sup>

*Note:*

- Must be given early
- Avoid if kidney impairment
- Avoid use with macrolide antibiotics, e.g. clarithromycin, especially in CKD
- Avoid long-term use

*Note:*

- Avoid changes to urate-lowering therapy during an acute attack of gout
- Avoid aspirin and urate pool lowering drugs (probenecid, allopurinol, sulfipyrazone)<sup>30</sup>
- Monitor kidney function and electrolytes

## Long-term therapy

When acute attack subsides, preventive measures with the aim of treating through diet include:

- weight reduction
- a healthy, well-balanced diet
- avoidance of purine-rich food, such as organ meats (liver, brain, kidneys, sweetbread), tinned fish (sardines, anchovies, herrings), shellfish and game
- reducing red and processed meats, fried chips and sweet treats
- reduced intake of alcohol

- reduced intake of sugary soft drinks (fructose)<sup>32</sup>
- good fluid intake (e.g. water—2 litres a day)
- avoidance of drugs such as diuretics (thiazides, frusemide) and salicylates/low-dose aspirin
- wearing comfortable shoes
- avoidance of prolonged fasting

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## Prevention (drug prophylaxis)

*Allopurinol* (a xanthine oxidase inhibitor) is the first-line drug of choice: dose 100–300 mg daily.

Indications:

- frequent acute attacks (or even >1 attack in 12 months)
- tophi or chronic gouty arthritis
- kidney stones or uric acid nephropathy
- hyperuricaemia

Adverse effects:

- rash (2%)
- severe allergic reaction (rare)

Precautions:

- beware of kidney insufficiency and elderly patients—use lower doses
- beware of drug interactions:
  - azathioprine and 6 mercaptopurine—potentially lethal
  - amoxicillin—prone to rashes
- avoid initiating or changing allopurinol during an acute attack

## Method: treatment of intercritical and chronic gout

- Commence allopurinol 6–8 weeks after last acute attack.
- Start with 50 mg daily for 4 weeks and then increase by 50 mg every 2 to 4 weeks to maximum 900 mg daily.

- Check uric acid level after 4 weeks: aim for level <0.38 mmol/L.
- Temporarily add colchicine 0.5 mg bd or indomethacin 25 mg bd or other NSAIDs (to avoid precipitation of gout).

### **Second-line agents**

- Febuxostat (an alternative xanthine oxidase inhibitor): dose is 40 mg (o) daily initially for 2–4 weeks, increasing the daily dose by 40 mg every 2–4 weeks, to maximum dose 120 mg.
- Probenecid (uricosuric agent)—a second-line agent. Good for hyperexcretion of uric acid by blocking renal tubular reabsorption. Dose: 500 mg/day (up to 2 g).

*Note:* Aspirin antagonises effect.

### **Prophylaxis of a flare of gout<sup>12</sup>**

colchicine 0.5 mg (o) daily or bd

*or*

prednisolone 5mg (o) daily

*or*

an NSAID, e.g. diclofenac 25–50 mg (o) up to 200 mg/day

## **⌚ Calcium pyrophosphate crystal disorder (pseudogout)<sup>12</sup>**

The finding of calcification of articular cartilage on X-ray examination is usually termed chondrocalcinosis. This is mainly a disorder of the elderly superimposed on an osteoarthritic joint. The acute attack is similar to an acute attack of gout but it affects the following joints (in order):

- knee
- 2nd and 3rd MCP joints
- wrist
- shoulder
- ankle
- elbow

It can affect tendons, especially the Achilles tendon, and cause a fever resembling septic arthritis.

The crystals in synovial fluid are readily identified by phase-contrast microscopy. X-rays are helpful in showing calcification of the articular cartilage.

Management is based on aspiration and installation of a depot glucocorticosteroid by injection into the joint (if joint infection excluded) plus analgesia. Be cautious of using NSAIDs in the elderly—paracetamol is preferred. Colchicine can be used.

Treatment includes:<sup>12</sup>

indomethacin 50 mg (o) tds (if tolerated) until symptoms abate

*and/or*

colchicine 0.5 mg (o) tds until attack subsides

*and*

paracetamol 500–1000 mg (o) four times daily, if necessary

## The spondyloarthritides

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The spondyloarthritides are a group of related inflammatory arthropathies with common characteristics affecting the spondyles (vertebrae) of the spine. It is appropriate to regard them as synonymous with the seronegative spondyloarthropathies in contradistinction to RA, which is seropositive and affects the cervical spine only. Apart from back pain, this group tends to present with oligoarthritis in younger people. The arthritis is characteristically peripheral, asymmetrical, affects the lower limbs and can exhibit dactylitis (e.g. ‘sausage’ digits). Page 295

### Features<sup>33</sup>

- Sacroiliitis with or without spondylitis
- Enthesopathy (enthesitis), especially plantar fasciitis, Achilles tendonitis, costochondritis
- Peripheral arthritis, especially larger lower limb joints
- Extra-articular features (e.g. iritis/anterior uveitis, mucocutaneous lesions, psoriasisiform skin and nail lesions, chronic GIT and GU inflammation)
- Absent rheumatoid factor
- Association with HLA-B<sub>27</sub> antigen
- Familial predisposition

### The group of disorders

1. Axial spondyloarthritis, including ankylosing spondylitis
2. Reactive arthritis
3. Inflammatory bowel disease (enteropathic arthritis)
4. Psoriatic arthritis
5. Juvenile onset ankylosing spondylitis
6. Unclassified spondyloarthritis—partial features only

## Ankylosing spondylitis

This usually presents with an insidious onset of inflammatory back and buttock pain (sacroiliac joints and spine) and stiffness in young adults (age <40 years), and 20% present with peripheral joint involvement before the onset of back pain. It usually affects the girdle joints (hips and shoulders), knees or ankles. At some stage, over 35% have joints other than the spine affected. The symptoms are responsive to NSAIDs (see [CHAPTER 28](#) ).

### Key clinical criteria<sup>12</sup>

- Low back pain persisting for >3 months
- Associated morning stiffness >30 minutes
- Awoken with pain during second half of night
- Improvement with exercise and not relieved by rest
- Limitation of lumbar spine motion in sagittal and frontal planes
- Chest expansion ↓ relative to normal values
- Unilateral sacroiliitis (grade 3 to 4)
- Bilateral sacroiliitis (grade 2 to 4)

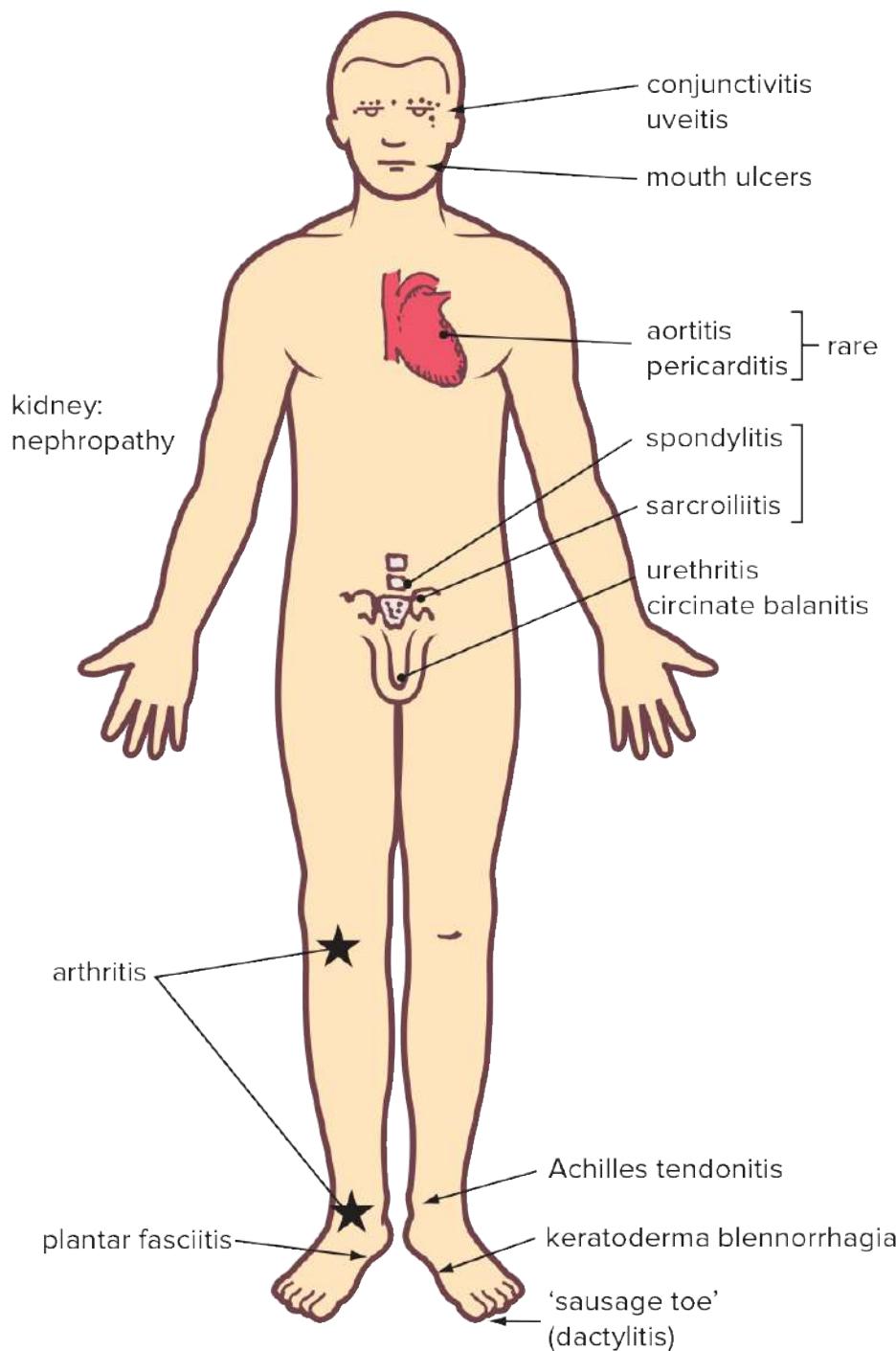
## Reactive arthritis

Reactive arthritis is a form of arthropathy in which non-septic arthritis and often sacroiliitis develop after an acute urogenital infection (usually *Chlamydia trachomatis*) or an enteric infection (e.g. *Salmonella*, *Shigella*).



**DxT** urethritis + conjunctivitis ± iritis + arthritis → reactive arthritis

The arthritis (Reiter syndrome), which commences 1–3 weeks post infection, tends to affect the larger peripheral joints, especially the ankle (talocrural) and knees, but the fingers and toes can be affected in a patchy polyarthritic fashion. Mucocutaneous lesions, including keratoderma blennorrhagica and circinate balanitis, may occur, although the majority develop peripheral arthritis only (see FIG. 25.14 ).



## ⌚ Enteropathic spondyloarthritidy

Inflammatory bowel disease (ulcerative colitis, Crohn disease and Whipple disease) may rarely be associated with peripheral arthritis and sacroiliitis.

## ⌚ Psoriatic arthritis

Like reactive arthritis, this can develop a condition indistinguishable from ankylosing spondylitis. It is therefore important to look beyond the skin condition of psoriasis, for about 5% will develop psoriatic arthropathy. It can have several manifestations:

1. mainly DIP joints
2. identical RA pattern but RA factor negative
3. identical ankylosing spondylitis pattern with sacroiliitis and spondylitis
4. monoarthritis, especially knees
5. severe deformity or ‘mutilans’ arthritis

## Unclassified spondyloarthritides

This category seems to be frequently encountered in family practice. The person clearly has a spondyloarthropathy but fails to meet the criteria for any one of the individual entities within the group. A typical patient is a young male in his third decade with a painful knee or other joint, unilateral (or bilateral) back pain with one of the enthesal problems (e.g. plantar fasciitis).

## Investigations for spondyloarthritides

- X-rays:
  - radiological sacroiliitis is central to the diagnosis
    - changes include narrowing of SIJs, margin irregularity, sclerosis of peri-articular bone and eventually bony fusion. Spondylitis usually follows
- ESR and CRP: most patients have an elevated ESR and CRP at some stage of their disease
- HLA-B<sub>27</sub>: this test has low specificity and has limited value except that it predicts risk to offspring if positive
- Microbiology: in patients with a history of reactive arthritis, cultures should be obtained from

the urethra, faeces, urine and blood<sup>32</sup>

## Principles of management

- Identify the most active elements of the disease and treat accordingly.
- Provide patient and family education with appropriate reassurance: this is vital. Stress that, although the disease is non-curable, treatment is effective and long-term prognosis generally good.
- Provide regular assessment and support.
- Give genetic counselling—in cases of ankylosing spondylitis with positive HLA-B<sub>27</sub>, the risk to offspring is significant.
- Give advice regarding work, especially with posture.
- Acute anterior uveitis requires prompt treatment and monitoring by an ophthalmologist.
- Refer for physiotherapy for exercises, stretching program, postural exercises and hydrotherapy. Appropriate physiotherapy slows deterioration in spinal function.<sup>33</sup>
- Consider referral for occupational therapy.
- Pharmacological agents:<sup>12</sup>

NSAIDs (e.g. indomethacin 75–200 mg (o) daily or 100 mg rectally nocte daily or ketoprofen 100 mg rectally nocte to control pain, stiffness and synovitis)

sulfasalazine (if NSAIDs ineffective)

intra-articular corticosteroids for severe monoarthritis and intralesional corticosteroids for enthesopathy

Refer for advice on above and especially for DMARD and bDMARD therapy.

## Cautions

- Careful monitoring is required with NSAIDs and sulfasalazine.
- Systemic corticosteroids are not indicated.
- Immunosuppressants (low-dose weekly methotrexate) and bDMARDs may be needed for severe intractable problems with psoriasis and reactive arthritis.
- These conditions should be managed in collaboration with a specialist.

## When to refer

---

- Consider referring most severe true inflammatory disorders for diagnosis and initiation of treatment (e.g. RA, spondyloarthropathy, connective tissue disorders and suspicion of a vasculitide)
- Osteoarthritis:
  - generalised joint pain
  - associated systemic symptoms
  - deteriorating joint function
  - intractable pain (especially at rest)
  - if surgical procedure is contemplated<sup>12</sup>
- Rheumatoid arthritis:
  - all patients initially
  - persistent inflammation of a joint or joints
  - patient ill and corticosteroids contemplated
  - if a surgical procedure is contemplated
- Spondyloarthropathies:
  - initial referral for confirmation of diagnosis and initiation of treatment
  - disease unresponsive to conventional treatment
  - sudden deterioration in symptoms, especially pain
  - onset of uveitis or other ocular complications
  - adverse drug reactions
- Undiagnosed arthritis in presence of constitutional symptoms
- Suspicion of a suppurative or serious infective condition (e.g. septic arthritis, endocarditis, brucellosis)
- Children with evidence of juvenile idiopathic arthritis (e.g. Still syndrome)

## Practice tips

- Morning stiffness and pain, improving with exercise = RA.
- Flitting polyarthritis and fever = rheumatic fever; ?endocarditis; ?SLE.
- Polyarthritis (usually PIPs) and rash = viral arthritis or drug reaction.
- If rheumatoid arthritis involves the neck, beware of atlantoaxial subluxation and spinal cord compression.
- If the patient is young—think of SLE.
- If a patient returns from overseas with arthralgia, think of drug reactions, hepatitis, Lyme disease, but if the pain is intense consider dengue fever.
- Consider the possibility of Lyme disease in people with a fever, rash and arthritis who have been exposed to tick bites overseas.
- If a patient presents with Raynaud phenomenon and arthritis, especially of the hands, consider foremost RA, SLE and systemic sclerosis.
- Avoid the temptation to apply on doubtful grounds a broad label such as arthritis or rheumatoid, or a precise diagnosis such as RA, and introduce drugs.<sup>34</sup>

## Patient education resources

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

- Gout
- Osteoarthritis
- Rheumatoid arthritis

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## 26 Anorectal disorders

*Duncan ill with very bad piles—operated on last night, or, since that sounds alarming, lanced. Can't really sympathise with that particular disease, though the pain is terrible. Must laugh.*

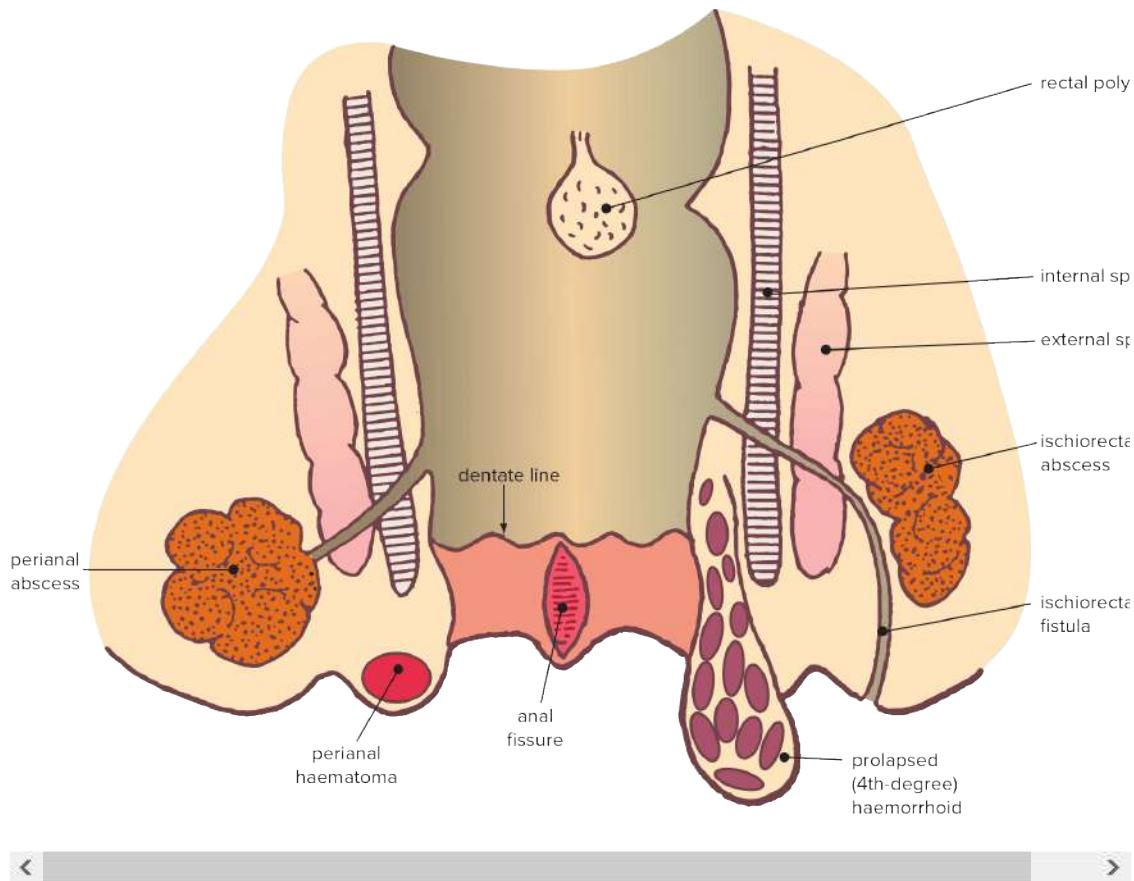
VIRGINIA WOOLF 1934, DIARY ENTRY

Anorectal problems are common in family practice and tend to cause anxiety that is often related to the fear of cancer. Although the majority of rectal bleeding and lumps have non-cancerous causes, the fear of cancer may be well founded, so it is important to consider it in any presentation of rectal bleeding.

Anorectal problems include:

- pain
- lumps
- discharge
- bleeding
- pruritus

Common anorectal conditions are illustrated in FIGURE 26.1 .



**FIGURE 26.1** Common anorectal conditions

## Anorectal pain (Proctalgia)

The complaint may be that defecation is painful or almost impossible because of anorectal pain.

### Causes

Pain without swelling:

- anal fissure
- anal herpes
- ulcerative proctitis
- proctalgia fugax
- solitary rectal ulcer

- tenesmus

Painful swelling:

- perianal haematoma
- strangulated internal haemorrhoids
- abscess: perianal, ischiorectal
- pilonidal sinus
- fistula-in-ano (intermittent)
- anal carcinoma

## Anal fissure

Anal fissures cause pain on defecation and usually develop after a period of constipation (may be a brief period) and tenesmus. Other associations are childbirth and opioid analgesics.<sup>1</sup>

Sometimes the pain can be excruciating, persisting for hours and radiating down the back of both legs. Anal fissures, especially if chronic, can cause minor anorectal bleeding (bright blood) noted as spotting on the toilet paper.

### Examination

On inspection the anal fissure is usually seen in the anal margin—90% are situated in the midline posteriorly (6 o'clock). The fissure appears as an elliptical ulcer involving the lower third of the anus from the dentate line to the anal verge (see FIG. 26.2 ).<sup>1</sup>



**FIGURE 26.2** Anal fissure with prominent skin tag situated in the mid posterior position of the anal verge: the 6 o'clock position

Digital examination and sigmoidoscopy are difficult because of painful anal sphincter spasm. If there are multiple fissures, Crohn disease should be suspected. Crohn fissures look different, being indurated, oedematous and bluish in colour.

In chronic anal fissures a sentinel pile is common and in longstanding cases, a subcutaneous fistula is seen at the anal margin, with fibrosis and anal stenosis.<sup>1</sup>

### Red flag pointers for anorectal pain

- Weight loss
- Change in bowel habits

- Fever >38°C
- Recurrent (consider Crohn disease)
- Exquisitely painful PR (consider abscess)

## Treatment

The aim is to disrupt the cycle of anal sphincter spasm, allowing improved blood flow to assist healing. Management is conservative: patients should avoid hard stools, and use warm salt (sitz) baths after bowel movements to relax the internal anal sphincter. A high-residue diet and avoidance of constipation (aim for soft bulky stools) may lead to resolution and long-term prevention. A combined local anaesthetic and corticosteroid ointment applied to the fissure, particularly before passing a stool, can provide relief but may not promote healing. A conservative treatment is the application of diluted glyceryl trinitrate ointment (e.g. Rectogesic 2% three times daily for 6 weeks to the lower anal canal) with a gloved finger gently inserted into the anal canal. It achieves healing rates of 50–70%, significantly more than placebo ointment.<sup>2,3</sup> Transient headache is the main adverse effect. An alternative is 2% diltiazem cream applied twice daily for 6–8 weeks. An acute anal fissure will usually heal spontaneously or within a few weeks of treatment involving a high-fibre diet, sitz baths or laxatives.<sup>4</sup>

Lateral internal sphincterotomy is indicated in patients with a recurrent fissure and a chronic fissure with a degree of fibrosis and anal stenosis.<sup>5</sup> This surgical procedure is the gold-standard surgical procedure. An alternative ‘chemical’ sphincterotomy, which is as effective as surgical treatment, is injection of botulinum toxin into the sphincter.

## § Proctalgia fugax (levator ani spasm)

### Clinical features

- Episodic fleeting rectal pain
- Varies from mild discomfort to severe spasm
- Last 3–30 minutes
- Often wakes the person from sound sleep
- Can occur any time of day
- A functional bowel disorder of unknown aetiology
- Affects adults, being more common in women

### Management<sup>6</sup>

- Explanation and reassurance re self-healing
- An immediate drink (preferably hot) and local warmth with firm flannel pressure to the perineum
- Salbutamol inhaler (2 puffs statim) worth a trial but anecdotal evidence only

Alternatives include glyceryl trinitrate spray for the symptoms or possibly antispasmodics, calcium-channel blockers and clonidine.

## Solitary rectal ulcer syndrome

These ulcers occur in young adults; they can present with pain but usually present as the sensation of a rectal lump causing obstructed defecation and bleeding with mucus. The ulcer, which is usually seen on sigmoidoscopy about 10 cm from the anal margin on the anterior rectal wall, can resemble cancer. Management is difficult and a chronic course is common. Treatment includes a high-residue diet and the avoidance of constipation.

## Tenesmus

Tenesmus is an unpleasant sensation of incomplete evacuation of the rectum. It causes attempts to defecate at frequent intervals. The most common cause is irritable bowel syndrome. Another common cause is an abnormal mass in the rectum or anal canal, such as cancer (e.g. prostate, anorectal), haemorrhoids or a hard faecal mass. In some cases, despite intensive investigation, no cause is found and it appears to be a functional problem.

## Perianal haematoma

A perianal haematoma (thrombosed external haemorrhoid) is a purple tender swelling at the anal margin caused by rupture of an external haemorrhoidal vein following straining at toilet or some other effort involving a Valsalva manoeuvre. The degree of pain varies from a minor discomfort to severe pain. It has been described as the ‘five-day, painful, self-curing pile’, which may lead to a skin tag. Spontaneous rupture with relief of symptoms can occur.

## Management

Surgical intervention is recommended, especially early in the presence of severe discomfort. The treatment depends on the time of presentation after the appearance of the haematoma.

1. *Within 24 hours of onset.* Perform simple aspiration without local anaesthetic using a 19 gauge needle while the haematoma is still fluid.
2. *From 1 to 3 days of onset.* The blood has clotted and a simple incision under local anaesthetic over the haematoma with deroofing with scissors (like taking the top off a boiled egg) to remove the thrombosis by squeezing is recommended. Removal of the haematoma reduces the chances of recurrence and the development of a skin tag, which can be a source of anal

irritation.

3. *Day 4 onwards.* The haematoma is best left alone unless it is very painful or (rarely) infected. Resolution is evidenced by the appearance of wrinkles in the previously stretched skin.

### Follow-up

Review in 4 weeks for rectal examination to examine for any underlying internal haemorrhoid that may predispose to further recurrence. Prevention includes an increased intake of dietary fibre and avoidance of straining at stool.

## Strangulated haemorrhoids

A marked oedematous circumferential swelling will appear if all the haemorrhoids are involved. If only one haemorrhoid is strangulated, proctoscopy will help to distinguish it from a perianal haematoma. Initial treatment is with rest and ice packs and then haemorrhoidectomy at the earliest possible time. It is best to refer for urgent surgery.

## Perianal cellulitis<sup>6</sup>

This occurs mainly in preschool and school-aged children. It is usually caused by *Streptococcus pyogenes*. Symptoms are perianal redness and pain on defecation. Check for a fissure. After swabbing, treat with oral cephalexin for 10 days.

## Perianal anorectal abscess

This is caused by infection by polymicrobial organisms in one of the anal glands that drain the anal canal.

### Clinical features

- Severe, constant, throbbing pain
- Fever and toxicity
- Hot, red, tender swelling adjacent to anal margin
- Non-fluctuant swelling

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Careful examination is essential to make the diagnosis. Look for evidence of a fistula, Crohn disease and anorectal cancer.

### Treatment

Drain via a cruciate incision, which may need to be deep (with trimming of the corners) over the point of maximal induration. A drain tube can be inserted for 7–10 days. Packing is not

necessary.

### Antibiotics

If a perianal or perirectal abscess is recalcitrant or spreading with cellulitis, use:

- metronidazole 400 mg (o) 12 hourly for 5–7 days plus
- cephalexin 500 mg (o) 6 hourly for 5–7 days<sup>7</sup>

## ¶ Ischiorectal abscess

An ischiorectal abscess presents as a larger, more diffuse, tender, dusky red swelling in the buttock. The presence of an abscess is usually very obvious but the precise focus is not always obvious on inspection. Antibiotics are of little help and surgical incision and drainage under deep anaesthesia is necessary as soon as possible.

## ¶ Pilonidal sinus and abscess

Recurrent abscesses and discharge in the sacral region (at the upper end of the natal cleft about 6 cm from the anus) can be caused by a midline pilonidal sinus, which often presents as a painful abscess. Once the infection has settled it is important to excise the pits, allow free drainage of the midline cavity and lateral tracks and remove all ingrown hair. Antibiotics, which should be guided by culture (e.g. cephalexin and metronidazole), are given to complement surgical drainage only if there is severe surrounding cellulitis.<sup>6</sup> Pilonidal means ‘a nest of hairs’ and the problem is particularly common in hirsute young men (see FIG. 26.3 ). Refer for excision of the sinus network if necessary, possibly marsupialisation.



**FIGURE 26.3** Shaving reveals a pilonidal sinus and a lateral sinus opening. It shows the characteristic tuft of hairs protruding from the midline sinus.

## ¶ Fistula-in-ano<sup>5</sup>

An anal fistula is a tract that communicates between the perianal skin (visible opening) and the anal canal, usually at the level of the dentate line. It usually arises from chronic perianal infection, especially following discharge of an abscess. It is common in Crohn disease. Symptoms include: recurrent abscesses; discharge of blood, pus or serous fluid; swelling and anal pain. A surgical opinion is necessary to determine the appropriate surgical procedures, which may be complex if it traverses sphincter musculature. One method is the Seton management, whereby thin silicone, silk or latex slings are inserted under general anaesthetic. This allows drainage and then guides surgical removal of the tracts.<sup>8</sup>

## Anorectal lumps

Anorectal lumps are relatively common and patients are often concerned because of the fear of cancer. A lump arising from the anal canal or rectum, such as an internal haemorrhoid, tends to appear intermittently upon defecation, and reduce afterwards.<sup>1</sup> Common prolapsing lesions include second- and third-degree haemorrhoids, hypertrophied anal papilla, polyps and rectal prolapse. Common presenting lumps include skin tags, fourth-degree piles and perianal warts (see TABLE 26.1 ).

**Table 26.1** Common anal lumps

### Prolapsing lumps

- Second- and third-degree haemorrhoids
- Rectal prolapse
- Rectal polyp
- Hypertrophied anal papilla

### Persistent lumps

- Skin tag
- Perianal warts (*condylomata accuminata*)
- Anal cancer
- Fourth-degree haemorrhoids
- Perianal haematoma
- Perianal abscess

## § Skin tags

The skin tag is usually the legacy of an untreated perianal haematoma. It may require excision for aesthetic reasons, for hygiene or because it is a source of pruritus ani or irritation. A tag may be associated with a chronic fissure.

## Treatment (method of excision)

A simple elliptical excision at the base of the skin tag is made under local anaesthetic. Suturing of the defect is usually not necessary. Perianal incisions/excisions rarely become infected.

## ¶ Perianal warts

It is important to distinguish the common viral warts from the condylomata lata of secondary syphilis. Local therapy includes the application of podophyllin every 2 or 3 days by the practitioner or imiquimod. Cryotherapy or diathermy are alternatives.

## ¶ Rectal prolapse

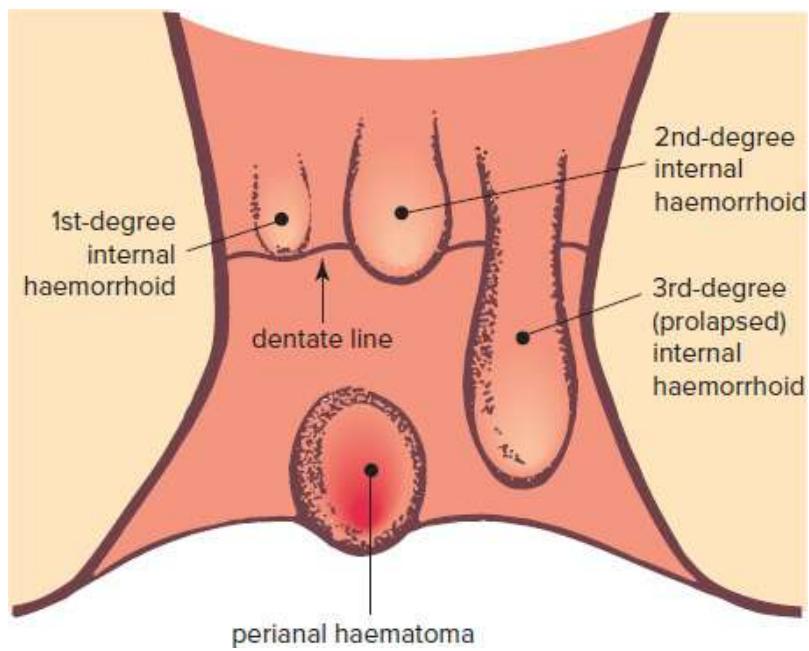
This is protrusion from the anus to a variable degree of the rectal mucosa (partial) or the full thickness of the rectal wall. It appears to be associated with constipation and chronic straining, leading to a lax sphincter. Features can include mucus discharge, bleeding, tenesmus, a solitary rectal ulcer and faecal incontinence (75%).

Visualisation of the prolapse is an important part of the diagnosis. Surgery such as rectopexy (fixing the rectum to the sacrum) is the only effective treatment for a complete prolapse.<sup>5</sup>

Temporary shrinking of a visible prolapse in an emergency situation can be achieved by a liberal sprinkling of fine crystalline sugar.

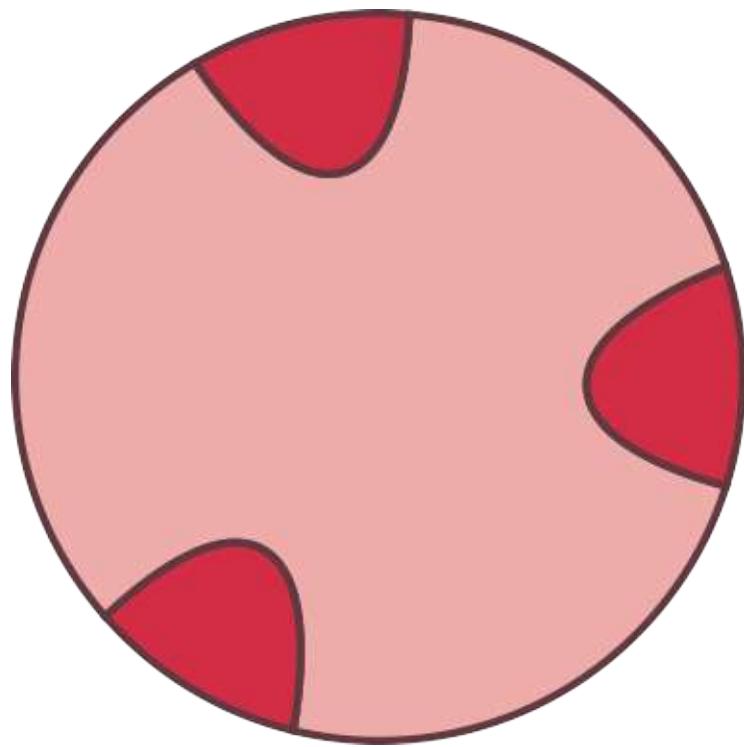
## ¶ Internal haemorrhoids

Haemorrhoids or piles are common and tend to develop between the ages of 20 and 50 years. In developed nations, roughly one in two adults has had a haemorrhoid by the age of 50.<sup>2</sup> Internal haemorrhoids are a complex of dilated arteries, branches of the superior haemorrhoidal artery and veins of the internal haemorrhoidal venous plexus (see FIG. 26.4 ). The commonest cause is chronic constipation related to a lack of dietary fibre and inappropriate bowel habit.



**FIGURE 26.4** Classification of haemorrhoids

Anatomically there are three classical sites, namely 3, 7 and 11 o'clock (see FIG. 26.5 ).



**FIGURE 26.5** Three sites of primary haemorrhoids, looking into the anus from

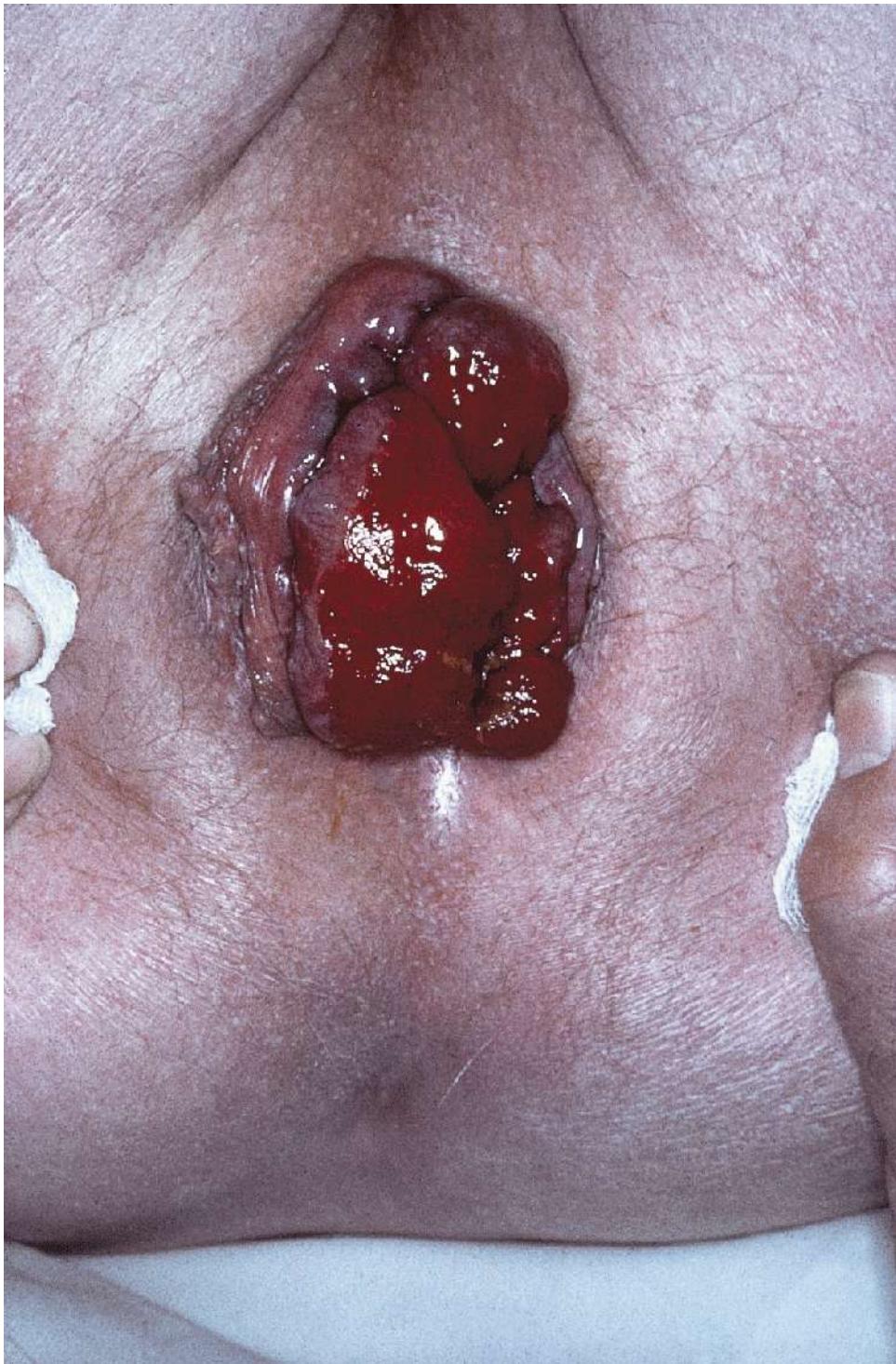
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## Clinical stages and pathology<sup>2</sup>

- *Stage 1:* First-degree internal haemorrhoids: three bulges form above the dentate line. Bright bleeding is common.
- *Stage 2:* Second-degree internal haemorrhoids: the bulges increase in size and slide downwards so that the person is aware of lumps when straining at stool, but they disappear upon relaxing. Bleeding is a feature.
- *Stage 3:* Third-degree internal haemorrhoids: the pile continues to enlarge and slide downwards, requiring manual replacement to alleviate discomfort. Bleeding is also a feature.
- *Stage 4:* Fourth-degree internal haemorrhoids: prolapse has occurred and replacement of the prolapsed pile into the anal canal is impossible.

## Symptoms

Bleeding is the main and, in many people, the only symptom. The word ‘haemorrhoid’ means flow of blood. Other symptoms include prolapse, mucoid discharge, irritation/itching, tenesmus, incomplete bowel evacuation and pain (see FIG. 26.6 ).



**FIGURE 26.6** Severely prolapsed haemorrhoids requiring surgery

## Treatment

Invasive treatment of haemorrhoids is based on three main procedures: rubber band ligation,

cryotherapy and sphincterotomy. Injection is now not so favoured, while a meta-analysis concluded that rubber band ligation was the most effective non-surgical therapy.<sup>9</sup> Surgery is generally reserved for large strangulated piles. The best treatment, however, is prevention; softish bulky faeces that pass easily prevent haemorrhoids. People should be advised to have an adequate intake of non-caffeinated fluids and a diet with enough fibre by eating plenty of fresh fruit, vegetables, wholegrain cereals or bran. They should respond to the urge to defecate and avoid straining at stool, complete their bowel action within a few minutes and avoid using laxatives.

## Anal discharge

---

Anal discharge refers to the involuntary escape of fluid from or near the anus. The causes may be considered as follows.<sup>5</sup>

1. *Continent*

- Anal fistula
- Pilonidal sinus
- STIs: anal warts, gonococcal ulcers, genital herpes
- Solitary rectal ulcer syndrome
- Cancer of anal margin

2. *Incontinent*

- Minor incontinence—weakness of internal sphincter
- Severe incontinence—weakness of levator ani and puborectalis

3. *Partially continent*

- Faecal impaction
- Rectal prolapse

## Anal (faecal) incontinence

---

An Australian survey suggested 1 in 9 adults suffer some degree of faecal incontinence, which is a common reason for institutionalisation of the elderly.<sup>10</sup> Patients may be reluctant to seek medical advice and doctors often do not ask specifically about the condition. The problem is as common in men as in women.

Apart from ageing, risk factors include perianal injury, such as childbirth injury, anal surgery,

irritable bowel syndrome and neurological disorders.

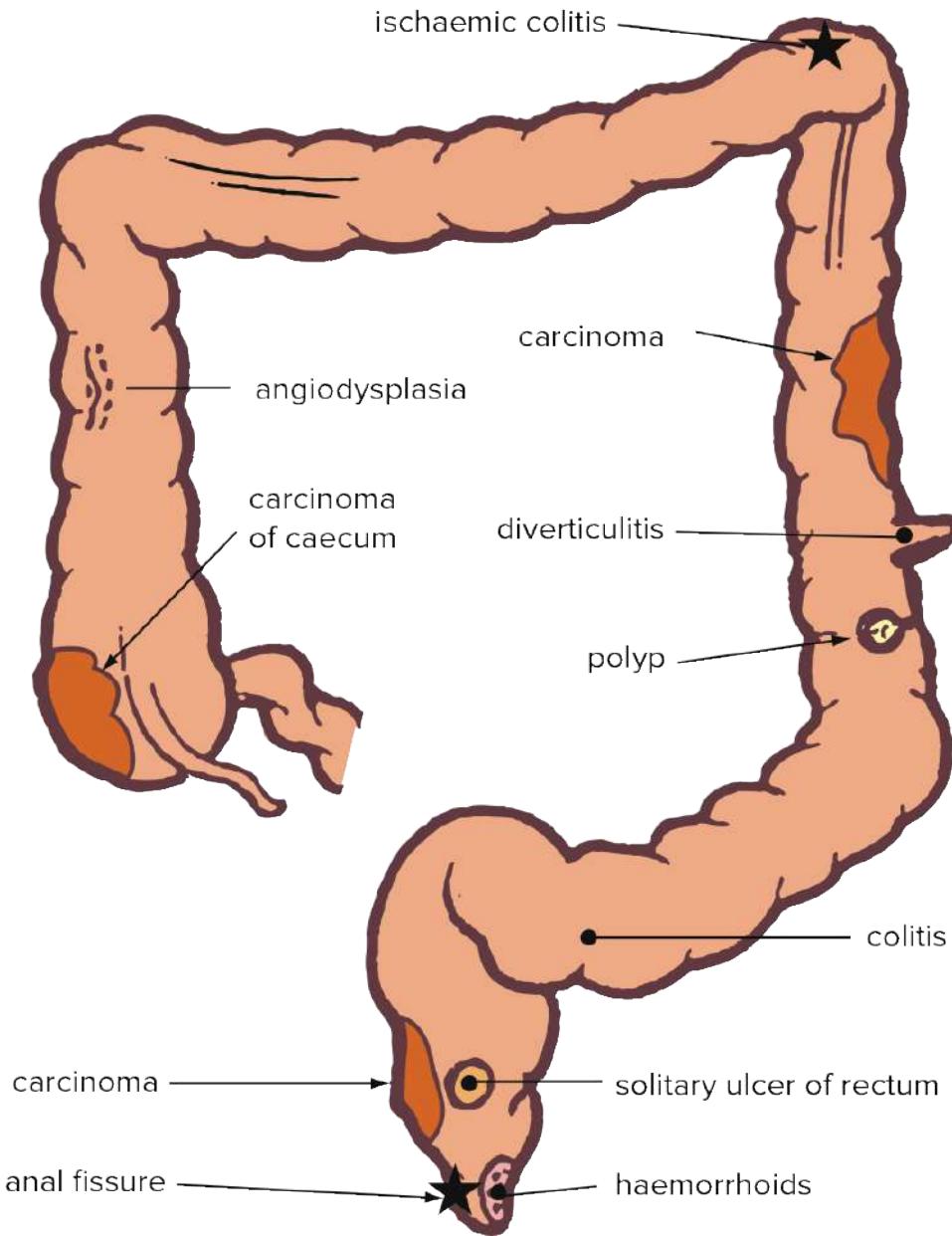
If there are symptoms of anal incontinence postnatally, early referral to a physiotherapist, continence nurse adviser or colorectal surgeon is advisable.<sup>11</sup>

Among the various treatments there are surgical possibilities, which vary from direct sphincter repair, directed injections such as collagen and silicone into the anal sphincter, and an artificial anal sphincter (e.g. Acticon Neosphincter). A colostomy may be the last resort. It is worth keeping in mind asking patients about the possibility of this problem and knowing ‘to whom to refer’.

## Rectal bleeding

---

Patients present with any degree of bleeding from a smear on the toilet tissue to severe haemorrhage. Various causes are presented in [FIGURE 26.7](#). Common causes are polyps, colon and rectal cancer, ischaemic colitis, diverticular disease and haemorrhoids.



**FIGURE 26.7** Various causes of rectal bleeding

Local causes of bleeding include excoriated skin, anal fissure, a burst perianal haematoma and anal cancer. A characteristic pattern of bright bleeding is found with haemorrhoids. It is usually small non-prolapsing (therefore not visible) haemorrhoids that bleed.

The nature of the blood (e.g. bright red, dark red or black) and the nature of the bleeding (e.g. smear, streaked on stool, mixed with stool, massive) gives an indication of the source of the bleeding (see TABLE 26.2). Black tarry (melaena) stool indicates bleeding from the upper gastrointestinal tract and is rare distal to the lower ileum. Those with melaena should be admitted to hospital.

**Table 26.2** Presentation and causes of rectal bleeding

|   |  |
|---|--|
| Bright red blood in toilet separate from faeces | Internal haemorrhoids  |
| Bright red blood on toilet paper                | Internal haemorrhoids<br>Fissure<br>Anal cancer<br>Pruritus<br>Anal warts and condylomata                                  |
| Blood and mucus on underwear                    | Third-degree haemorrhoids<br>Fourth-degree haemorrhoids<br>Prolapsed rectum<br>Mucosal prolapse<br>Prolapsed mucosal polyp |
| Blood on underwear (no mucus)                   | Ulcerated perianal haematoma<br>Anal cancer  |
| Blood and mucus mixed with faeces               | Colorectal cancer<br>Proctitis<br>Colitis, ulcerative colitis<br>Large mucosal polyp<br>Ischaemic colitis                  |
| Blood mixed with faeces (no mucus)              | Small colorectal polyps<br>Small colorectal cancer   |
| Melaena (black tarry stools)                    | Gastrointestinal bleeding (usually upper) with long transit time to the anus   |
| Torrential haemorrhage (rare)                   | Diverticular disorder<br>Angiodysplasia  |
| Large volumes of mucus in faeces (little blood) | Villous papilloma of rectum<br>Villous papilloma of colon  |
| Blood in faeces with menstruation (rare)        | Rectal endometriosis   |

Source: Reproduced with permission from Orlay G. *Office Proctology*. Sydney: Australasian Medical Publishing Company, 1987: 11–52.

Frequent passage of blood and mucus indicates a rectal tumour or proctitis, whereas more proximal tumours or extensive colitis present different patterns.

Substantial haemorrhage, which is rare, can be caused by diverticular disorder, angiodysplasia or more proximal lesions such as Meckel diverticulum and even duodenal ulcers. Angiodysplasias are 5 mm collections of dilated mucosal capillaries and thick-walled submucosal veins, found usually in the ascending colon of elderly people who have no other bowel symptoms. The bleeding is persistent and recurrent. The site is identified by technetium-labelled red cell scan or colonoscopy.

The history should also include an analysis of any associated symptoms such as pain, diarrhoea or constipation, presence of lumps and a sensation of urgency or unsatisfied defecation. The latter symptoms point to a rectal cause. Associated change of bowel habit suggests a diagnosis of cancer of the rectum or left colon. Bleeding from right colon cancer is often occult, presenting as anaemia.

The examination includes a general assessment, anal inspection, digital rectal examination and proctosigmoidoscopy. Eighty per cent of rectal tumours are within fingertip range. Even if there is an anal lesion, proximal bleeding must be excluded in all cases by sigmoidoscopy<sup>2</sup> and by colonoscopy if there are any bowel symptoms or no obvious anal cause or a doubt about a lesion causing the symptoms.

### Red flag pointers for rectal bleeding

- Age >50 years, especially new bleeding
- Change of bowel habit
- Weight loss
- Weakness, fatigue
- Brisk bleeding
- Constipation
- Haemorrhoids (may be sinister)
- Family history of cancer

## Pruritus ani

Pruritus ani, which is itching of the anus, can be a distressing symptom that is worse at night, during hot weather and during exercise. It is seen typically in adult males with considerable inner drive, often at times of stress and in hot weather when sweating is excessive. In children, threadworm infestation should be suspected. It may be part of general itching, such as with a skin disorder, or localised whereby various anorectal disorders have to be excluded. Seborrhoeic

dermatitis is a particularly common underlying factor. Consider also the more uncomfortable lichen sclerosus with its ivory white sclerotic plaques, which may also be present in the genital region.

## Signs

The skin changes can vary from minimal signs to marked pathology that can show linear ulceration, maceration or lichenification (see FIG. 26.8 ). Superficial skin changes can be moist and macerated or dry and scaly. Full anorectal examination is necessary.

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**FIGURE 26.8** Lichen chronicus simplex. Lichenification from scratching with longstanding pruritus (CHAPTER 114 )

## Causes and aggravating factors

- Psychological factors:
  - stress and anxiety
  - fear of cancer
- Generalised systemic or skin disorders:
  - seborrhoeic dermatitis
  - eczema

lichen sclerosus  
diabetes mellitus  
candidiasis  
psoriasis (look for fissures in natal cleft)  
antibiotic treatment  
worms: pinworm (threadworm)  
diarrhoea causing excoriation  
Crohn disease

- Local anorectal conditions:

haemorrhoids, skin tags  
fissures/fistula  
faecal incontinence  
warts

- Zealous hygiene or lack of hygiene

- Contact dermatitis:

- dyed or perfumed toilet tissue, soap, powder  
clothing
- Excessive sweating (e.g. tight pantyhose in summer)

## Diagnosis

- Urinalysis (?diabetes)
- Anorectal examination
- Scrapings and microscopy to detect organisms
- Stool examination for intestinal parasites

## Treatment

- Treat the cause (if known) and break the scratch cycle.

- Avoid local anaesthetics, antiseptics.
- Advise aqueous cream or a soap substitute to wash anus (instead of soap).
- Most effective preparations (for short<sup>13</sup> courses):  
methylprednisolone aceponate 0.1% in a fatty ointment; once daily until symptoms settle (up to 4 weeks)  
*or*  
hydrocortisone 1% cream/ointment  
*or*  
hydrocortisone 1% cream with clioquinol 3% or clotrimazole 1% (especially if dermatosis and *Candida* suspected)

If an isolated area and resistant, infiltrate 0.5 mL of triamcinolone intradermally. Fractionated X-ray therapy can be used if very severe. For a lichenified perianal area, use a potent corticosteroid ointment, e.g. betamethasone dipropionate 0.05% daily until clear.

Patient education about anal hygiene is essential.

### Practice tips for pruritus ani

- Most cases of uncomplicated pruritus ani resolve with simple measures, including explanation and reassurance.
- Avoid perfumed soaps and powders. Use bland aqueous cream or a mild soap substitute.
- Otherwise prescribe a corticosteroid, especially methylprednisolone aceponate 0.1%. Once symptoms are controlled, use hydrocortisone 1%.<sup>13</sup>
- Lifestyle stress and anxiety underlie most cases.
- In obese people with intertrigo and excessive sweating, strap the buttocks apart with adhesive tape.
- Consider perianal lichen simplex and lichen sclerosus in those presenting with ‘a sore bottom’.

## Patient education resources

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

- Anal fissure
- Haemorrhoids
- Pruritus ani

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## 27 Thoracic back pain

*The maladies that afflict the clerks aforesaid arise from three causes; first constant sitting, secondly the incessant movement of the hand and always in the same direction, and thirdly the strain on the midline from the effort not to disfigure the books by error or cause loss to their employers.*

THE PHYSICIAN RAMAZZINI 1713

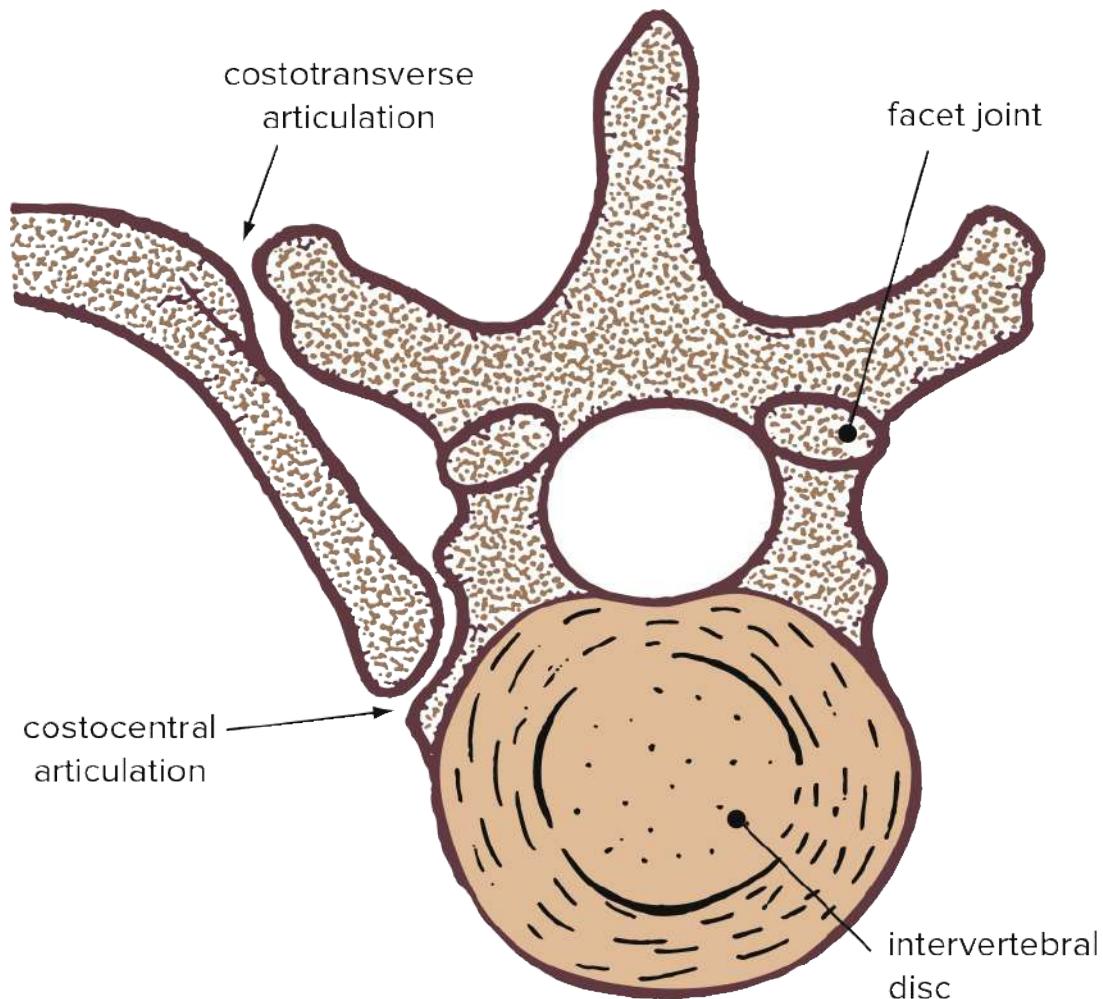
Thoracic (dorsal) or upper back pain, which is defined as pain localised between the neck and above the costal margin, is common in people of all ages. It accounts for 10–15% of all spinal pain and has a 1-year prevalence in 20% of adults. Dysfunction of the joints of the thoracic spine, with its unique costovertebral joints (which are an important source of back pain), is commonly encountered in medical practice, especially in people whose lifestyle creates stresses and strains through poor posture and heavy lifting. It is also referred to as non-specific thoracic spinal pain. Muscular and ligamentous strains may be common, but they rarely come to light in practice because they are self-limiting and not severe.

This dysfunction can cause referred pain to various parts of the chest wall and can mimic the symptoms of various visceral diseases, such as angina, biliary colic and oesophageal spasm. In similar fashion, heart and gall bladder pain can mimic spinal pain.

### Key facts and checkpoints

- The commonest site of pain in the spine is the costovertebral articulations, especially the costotransverse articulation (see FIG. 27.1 ).
- Pain of thoracic spinal origin may be referred anywhere to the chest wall, but the commonest sites are the scapular region, the paravertebral region 2–5 cm from midline and, anteriorly, over the costochondral region.
- Thoracic (also known as dorsal) pain is more common in patients with abnormalities such as kyphosis and Scheuermann disease.
- Trauma to the chest wall (including falls on the chest such as those experienced in body contact sport) commonly lead to disorders of the thoracic spine.

- Unlike the lumbar spine, the joints are quite superficial and it is relatively easy to find the affected (painful) segment.
- Intervertebral disc prolapse is very uncommon in the thoracic spine.
- The older person presenting with chest pain should be regarded as having a cardiac cause until proved otherwise.
- If the chest pain is non-cardiac, then the possibility of referral from the thoracic spine should be considered.
- The thoracic spine is the commonest site in the vertebral column for metastatic disease.
- Scheuermann disease, which affects the lower thoracic spine in adolescents, is often associated with kyphosis and recurrent thoracic back pain. Always inspect the thoracic spine of the younger patient for kyphosis and scoliosis, ideally at 9 years of age.
- Palpation is the most important component of the physical examination.



**FIGURE 27.1** The functional unit of the thoracic spine

## A diagnostic approach

A summary of the diagnostic strategy model is presented in [TABLE 27.1](#).

**Table 27.1** Thoracic back pain: diagnostic strategy model

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### Probability diagnosis

Musculoligamentous strains (mainly postural)  
Vertebral dysfunction (non-specific back pain)

### Serious disorders not to be missed

**Cardiovascular:**

- acute coronary syndromes, esp. myocardial infarction
- dissecting aneurysm
- pulmonary infarction
- epidural haematoma (blood-thinning agents)

**Neoplasia:**

- myeloma
- lung (with infiltration)
- metastatic disease

**Severe infections:**

- epidural abscess
- pleurisy
- infectious endocarditis
- osteomyelitis

Pneumothorax

Osteoporosis

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**Pitfalls (often missed)**

Angina

Gastrointestinal disorders

- oesophageal dysfunction
- peptic ulcer (penetrating)
- hepatobiliary
- pancreatic

Herpes zoster

Spondyloarthropathies

Costochondritis:

- Tietze syndrome

Fibromyalgia syndrome

Polymyalgia rheumatica

Notalgia paraesthesia

Chronic infection:

- tuberculosis
- brucellosis

---

**Seven masquerades checklist**

Depression

Diabetic radiculopathy

Spinal dysfunction

### Is the patient trying to tell me something?

Yes, quite possible with many cases of back pain.

## Probability diagnosis

The commonest cause of thoracic back pain is musculoskeletal, due usually to musculoligamentous strains caused by poor posture. However, these pains are usually transitory and present rarely to the practitioner. The problems that commonly present are those caused by dysfunction of the lower cervical and thoracic spinal joints, especially those of the mid-thoracic (interscapular) area.

Arthritic conditions of the thoracic spine are not overly common although degenerative osteoarthritis is encountered at times; the inflammatory spondyloarthropathies are uncommon.

The various systemic infectious diseases such as influenza and Epstein–Barr mononucleosis can certainly cause diffuse backache but should be assessed in context.

## Serious disorders not to be missed

A special problem with the thoracic spine is its relationship with the many thoracic and upper abdominal structures that can refer pain to the back. These structures are listed in TABLE 27.2 but, in particular, myocardial infarction and dissecting aneurysm must be considered. A complex problem described by neurosurgeons is the presentation of severe sudden thoracic back pain caused by an epidural haematoma related to aspirin or warfarin therapy. Visceral disease causing a rupture or leakage should be kept in mind.

**Table 27.2** Non-musculoskeletal causes of thoracic back pain

|                             |  |
|-----------------------------|--|
| <b>Heart</b>                | Myocardial infarction<br>Angina<br>Pericarditis  |
| <b>Great vessels, lungs</b> | Dissecting aneurysm<br>Pulmonary embolism (rare)<br>Pulmonary infarction<br>Pneumothorax<br>Pneumonia/pleurisy |
| <b>Oesophagus</b>           | Oesophageal rupture<br>Oesophageal spasm<br>Oesophagitis   |

## Oesophageal cancer

|                                       |  |
|---------------------------------------|--|
| <b>Subdiaphragmatic disorders of:</b> | Gall bladder<br>Stomach<br>Duodenum<br>Pancreas<br>Subphrenic collection |
| <b>Miscellaneous infections</b>       | Herpes zoster<br>Bornholm disease<br>Infective endocarditis              |
| <b>Psychogenic</b>                    |  |

## Cardiopulmonary problems

The acute onset of pain can have sinister implications in the thoracic spine where various life-threatening cardiopulmonary and vascular events have to be kept in mind. The pulmonary causes of acute pain include spontaneous pneumothorax, pleurisy and pulmonary infarction. Thoracic back pain may be associated with infective endocarditis due to embolic phenomena. The ubiquitous myocardial infarction or acute coronary occlusion may, uncommonly, cause interscapular back pain, while the very painful dissecting or ruptured aortic aneurysm may cause back pain with hypotension.

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## Osteoporosis

Osteoporotic pathological fracture, especially in people over 60 years, including both men and women, must always be considered in acute thoracic pain. The association with pain following inappropriate physical therapy such as spinal manipulation should also be considered.

## Acute infections

Infective conditions that can involve the spine include osteomyelitis, tuberculosis, brucellosis, syphilis and *Salmonella* infections. Such conditions should be suspected in young people (osteomyelitis), farm workers (brucellosis) and migrants from South-East Asia and developing nations (tuberculosis). The presence of poor general health and fever necessitates investigations for these infections.

## Neoplasia

Fortunately, tumours of the spine are uncommon. Nevertheless, they occur frequently enough for the full-time practitioner to very occasionally encounter metastatic disease.

The three common primary malignancies that metastasise to the spine are those originating in the lung, breast and the prostate (all paired structures). The less common primaries are the thyroid,

kidney, adrenals and malignant melanoma.

Reticuloses such as Hodgkin lymphoma can involve the spine. Primary malignancies that develop in the vertebrae include multiple myeloma and sarcoma.

Benign tumours to consider are often neurological in origin. The osteoid osteoma is aggravated by consuming alcohol and relieved by aspirin.

The tumours of the spine are summarised in TABLE 27.3 .

**Table 27.3** Significant tumours affecting the thoracic and lumbar spine

|                | Benign  | Malignant  |
|----------------|---|--|
| <b>Of bone</b> | Osteoid osteoma<br>Haemangioma<br>Osteoblastoma<br>Aneurysmal bone cyst<br>Eosinophilic granuloma   | Primary: <ul style="list-style-type: none"><li>• multiple myeloma</li><li>• lymphomas (e.g. Hodgkin)</li><li>• sarcoma</li></ul>   |
| <b>Spinal</b>  | Extradural: <ul style="list-style-type: none"><li>• lipoma</li><li>• neuroma</li><li>• fibroma</li></ul> Intradural: <ul style="list-style-type: none"><li>• neuroma</li><li>• ependymoma</li><li>• chordoma</li><li>• meningioma</li></ul> | Secondary: <ul style="list-style-type: none"><li>• breast</li><li>• lung</li><li>• prostate</li><li>• adrenals/kidney</li><li>• thyroid</li><li>• melanoma</li></ul> Direct spread: <ul style="list-style-type: none"><li>• stomach</li><li>• large bowel</li><li>• pancreas</li><li>• uterus/cervix/ovary</li></ul> |

Source: Reproduced with permission from Kenna C, Murtagh J. *Back Pain and Spinal Manipulation* (2nd edn). Oxford: Butterworth-Heinemann, 1997: 165–74.

The symptoms and signs that should alert the clinician to malignant disease are:

- back pain occurring in an older person
- unrelenting back pain, unrelieved by rest (this includes night pain)
- rapidly increasing back pain

- constitutional symptoms (e.g. unexplained weight loss, fever, malaise)
- a history of treatment for cancer (e.g. excision of skin melanoma)

## Red flag pointers for thoracic back pain<sup>2</sup>

The red flag pointers are similar to those for low back pain (see CHAPTER 28 ), especially with regards to trauma, malignancy and suppurative infection.

FBE, ESR, CRP and a plain X-ray of the thoracic spine should be the initial screening test in the presence of these pointers.

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A trap for the thoracic spine is lung cancer, such as mesothelioma, which can invade parietal pleura or structures adjacent to the vertebral column.

## Pitfalls

Pitfalls include ischaemic heart disease presenting with interscapular pain, herpes zoster at the pre-eruption stage and the various gastrointestinal disorders. Two commonly misdiagnosed problems are a penetrating duodenal ulcer presenting with lower thoracic pain and oesophageal spasm, which can cause thoracic back pain.

Inflammatory rheumatological problems are not common in the thoracic spine but occasionally a spondyloarthropathy such as ankylosing spondylitis manifests here, although it follows some time after the onset of sacroiliitis.

## Seven masquerades checklist

Spinal dysfunction is the outstanding cause in this checklist. Depression always warrants consideration in any pain syndrome, especially back pain. It can certainly cause exaggeration of pre-existing pain from vertebral dysfunction or some other chronic problem.

## Psychogenic considerations

Psychogenic or non-organic causes of back pain can present a complex dilemma in diagnosis and management. The causes may be apparent from the incongruous behaviour and personality of the patient, but often the diagnosis is reached by a process of exclusion. There is obviously some functional overlay in everyone with acute or chronic pain, hence the importance of appropriate reassurance to these patients that their problem invariably subsides with time and that they do not have cancer.

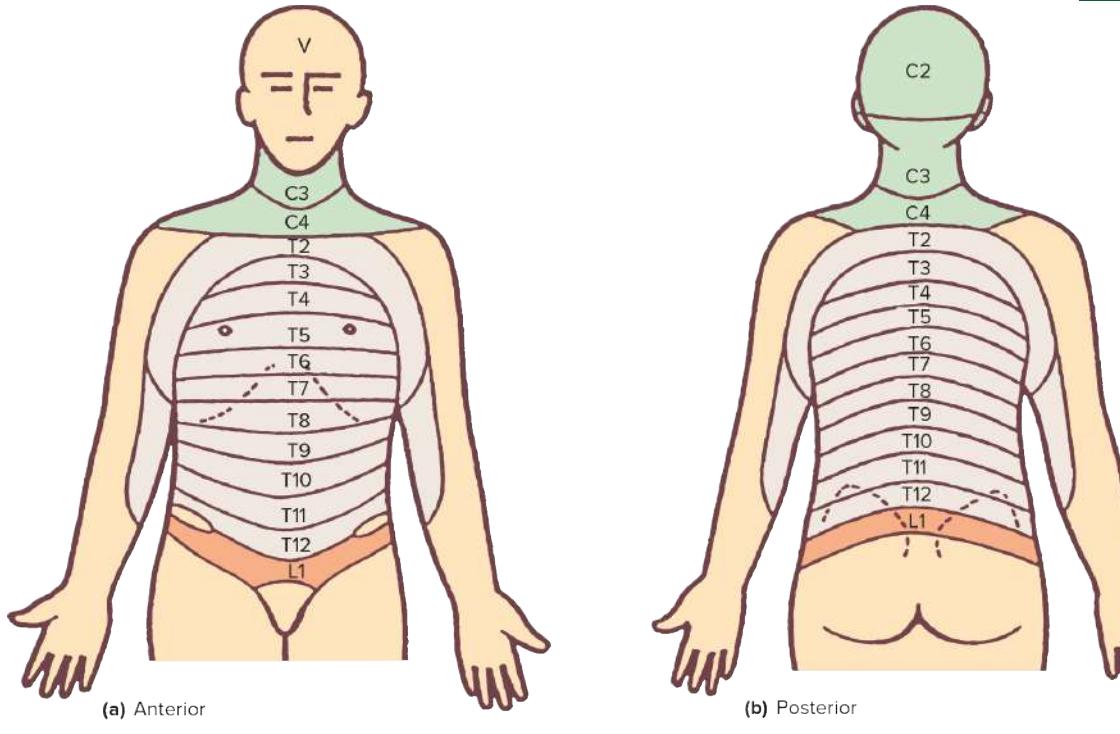
## Anatomical and clinical features

The functional unit of the thoracic spine is illustrated in FIGURE 27.1 . Although there is scant literature and evidence about the origins of pain in the thoracic spine,<sup>3</sup> the strongest evidence indicates that pain from the thoracic spine originates mainly from the apophyseal joints and rib articulations. Any one thoracic vertebra has 10 separate articulations, so the potential for dysfunction and the difficulty in clinically pinpointing the precise joint at a particular level are apparent.

The costovertebral joints are synovial joints unique to the thoracic spine and have two articulations—costotransverse and costocentral. Together with the apophyseal joints, they are capable of presenting with well-localised pain close to the midline or as referred pain, often quite distal to the spine, with the major symptoms not appearing to have any relationship to the thoracic spine.

Generalised referral patterns are presented in FIGURE 15.2 (see CHAPTER 15 ), while the dermatome pattern is outlined in FIGURE 27.2 .

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**FIGURE 27.2** Dermatomes for the thoracic nerve roots, indicating possible referral areas

Source: Reproduced with permission from C Kenna and J Murtagh. *Back Pain and Spinal Manipulation* (2nd edn), Oxford: Butterworth-Heinemann, 1997

The pain pattern acts as a guide only because there is considerable dermatomal overlap within the individual and variation from one person to another. It has been demonstrated that up to five

nerve roots may contribute to the innervation of any one point in the anterior segments of the trunk dermatomes, a fact emphasised by the clinical distribution of herpes zoster.

## Upper thoracic pain<sup>2</sup>

Dysfunction of the joints of the upper thoracic spine usually gives rise to localised pain and stiffness posteriorly but also can cause distal symptoms, probably via the autonomic nervous system.

A specific syndrome called the T4 syndrome<sup>4</sup> has been shown to cause vague pain and paraesthesia in the upper limbs and diffuse, vague head and posterior neck pain. Examination may reveal hypomobility of the upper thoracic segments. It can respond to spinal manipulation, which restores mobility.

However, most of the pain, stiffness and discomfort arise from dysfunction of the upper and middle thoracic segments with a presentation of ‘pain between my shoulder blades’.

## ⌚ Costovertebral joint dysfunction<sup>2</sup>

The unique feature of the thoracic spine is the costovertebral joint. Dysfunction of this joint commonly causes localised pain approximately 3–4 cm from the midline where the rib articulates with the transverse process and the vertebral body. In addition, it is frequently responsible for referred pain ranging from the midline, posterior to the lateral chest wall, and even anterior chest pain.

When the symptoms radiate laterally, the diagnosis is confirmed only when movement of the rib provokes pain at the costovertebral joint. This examination will simultaneously reproduce the referred pain.

Confusion arises for the clinician when the patient’s history focuses on the anterior chest pain and fails to mention the presence of posterior pain, should it be present.

## The clinical approach

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### History

The history of a person presenting with thoracic back pain should include a routine pain analysis, which usually provides important clues for the diagnosis. The age, sex and occupation are relevant. Pain in the thoracic area is very common in people who sit bent over for long periods, especially working at desks. Students and office workers are therefore at risk, as are breastfeeding mothers, who have to lift their babies.

Spines that are kyphotic or sciotic, or have a ‘hunchback’ secondary to disease such as tuberculosis and poliomyelitis, are prone to recurrent thoracic pain.

Older people are more likely to present with a neoplasm or osteoporosis. Osteoporosis is usually a trap because it is symptomless until the occurrence of a compression fracture. Symptoms following such a fracture can persist for 3 months.

Pain that is present day and night indicates a sinister cause.

Features of the history that give an indication that the pain is arising from dysfunction of the thoracic spine include:

- *Aggravation and relief of pain on trunk rotation.* The pain may be increased by rotating (twisting) towards the side of the pain but eased by rotating in the opposite direction.
- *Aggravation of pain by coughing, sneezing or deep inspiration.* This can produce a sharp catching pain which, if severe, tends to implicate the costovertebral joint. Care must be taken to rule out pneumonia and pleurisy.
- *Relief of pain by firm pressure.* Patients may state that their back pain is eased by firm pressure such as leaning against the corner of a wall.

It is very important to consider myocardial ischaemia during history-taking.

## Key questions

- Can you recall injuring your back? (Lifting something heavy or a fall onto your chest or back?)
- Is the pain present during the night?
- Do you have low back pain or neck pain?
- Does the pain come on after walking or any strenuous effort?
- Does the pain come on after eating or soon after going to bed at night?
- Have you noticed a fever or sweating at any time, especially at night?
- Have you noticed a rash near where you have the pain?
- What drugs are you taking? Do you take drugs for arthritis or pain? Cortisone?
- What happens when you take a deep breath, cough or sneeze?

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## Examination

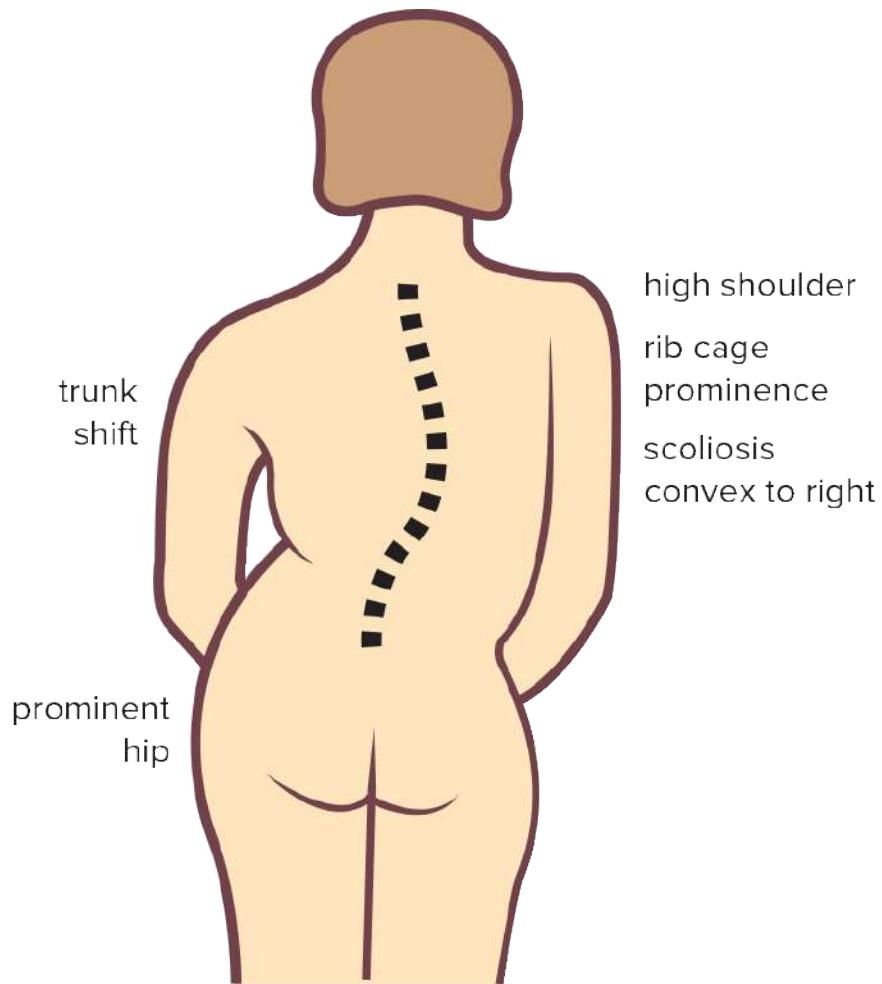
The examination of the thoracic spine is straightforward with the emphasis on palpation of the spine—central and laterally. This achieves the basic objective of reproducing the patient's symptoms and finding the level of pain. The 'LOOK, FEEL, MOVE, (consider) X-RAY' clinical approach is most appropriate for the thoracic spine.

## Inspection

Careful inspection, especially of posture, is important since it may be possible to observe at a glance why the person has thoracic pain. Note the symmetry, any scars, skin creases and deformities, ‘flat spots’ in the spine, the nature of the scapulae or evidence of muscle spasm. Look for kyphosis and scoliosis.

Kyphosis may be generalised, with the back having a smooth uniform contour, or localised where it is due to a collapsed vertebra, such as occurs in an older person with osteoporosis. Generalised kyphosis is common in the elderly, especially those with degenerative spinal disease. In the young it may reflect the important Scheuermann disease.

The younger person in particular should be screened for scoliosis (see FIG. 27.3 ), which becomes more prominent on forward flexion (see FIG. 27.5 ). Look for any asymmetry of the chest wall, inequality of the scapulae and differences in the levels of the shoulders. A useful sign of scoliosis is unequal shoulder levels and apparent ‘winging’ of scapula. When viewed anteriorly a difference in the levels of the nipples indicates the presence of scoliosis, or other problems causing one shoulder to drop. Inspection should therefore take place with posterior, lateral (side) and anterior views. For acute pain, check the skin for evidence of herpes zoster (rash or scars).



**FIGURE 27.3** Adolescent idiopathic scoliosis: typical configuration of the trunk and thoracic spine

### Palpation<sup>1</sup>

The best position is to have the patient prone on the examination table with the thoracic spine preferably in slight flexion, if the table head can be lowered.

Test passive extension of each joint with firm pressure from the pad of the thumbs or the bony hand (either the pisiform prominence or the lateral border of the fifth metacarpal). Spring up and down with a few firm oscillations, keeping the elbows straight, but being well above the patient. Ask if the pressure reproduces the pain.

Apart from asking 'Is that the pain?', note:

- the distribution of pain and its change with movement
- the range of movement

- the type of resistance in the joint
- any muscle spasm

Palpation must follow a set plan in order to reproduce the patient's pain. The sequence is as follows:

1. central—over spinous processes
2. unilateral—over apophyseal joints (2–3 cm from midline)
3. transverse—on side of spinous processes
4. unilateral—costotransverse junctions (4–5 cm from midline)
5. unilateral—over ribs (spring over posterior rib curve with ulnar border of hand, along axis of rib)

## Movements

There are four main movements of the thoracic spine to assess, the most important of which is rotation, as this is the movement that so frequently reproduces the pain where it is facet joint or costovertebral in origin.

The movements of the thoracic spine and their normal ranges are:

|                            |     |
|----------------------------|-----|
| 1. Extension               | 30° |
| 2. Lateral flexion L and R | 30° |
| 3. Flexion                 | 90° |
| 4. Rotation L and R        | 60° |

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Ask the patient to sit on the table with hands placed behind the neck and then perform the movements. Check these four active movements, noting any hypomobility, the range of movement, reproduction of symptoms and function and muscle spasm.

## Neurological examination

This includes sensory testing for altered sensation in a dermatomal distribution.

## Investigations

The plain X-ray and MRI scan is of no utility in patients with non-specific back pain but has a place in the presence of red flags, where the MRI is most appropriate to investigate suspected serious pathology. A plain X-ray may exclude basic bony abnormalities and diseases, such as

osteoporosis and malignancy. However, bear in mind that the majority of spinal X-rays and MRIs have abnormalities if you look hard enough, increasing steadily with age. Studies of lumbar and cervical MRIs in healthy volunteers find disc bulges in 70–90%.<sup>5</sup> CT scanning has a minimal role in the evaluation of thoracic spinal pain.

Other investigations to consider are:

- FBE and ESR/CRP
- serum alkaline phosphatase
- serum electrophoresis for multiple myeloma
- Bence–Jones protein analysis
- *Brucella* agglutination test
- blood culture for pyogenic infection and bacterial endocarditis
- tuberculosis studies
- HLA-B<sub>27</sub> antigen for spondyloarthropathies
- ECG or ECG stress tests (suspected angina)
- gastroscopy or barium studies (peptic ulcer)
- MRI scanning if myelopathy is suspected
- radionuclide bone scan if neoplastic or metabolic disease is suspected

## Thoracic back pain in children

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The most common cause of thoracic back pain in children is ‘postural backache’, also known as ‘TV backache’, which is usually found in adolescent schoolgirls and is a diagnosis of exclusion.

Important, although rare, problems in children include infections (tuberculosis, discitis and osteomyelitis) and tumours such as osteoid osteoma and malignant osteogenic sarcoma.

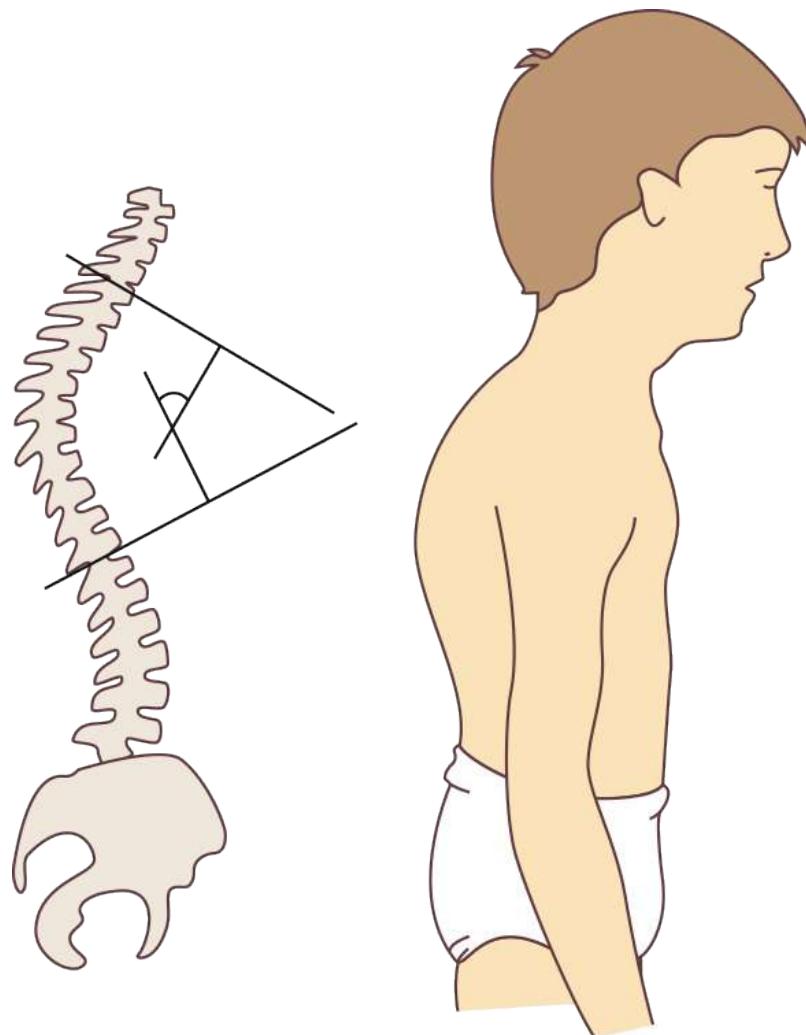
Dysfunction of the joints of the thoracic spine in children and particularly in adolescents is very common and often related to trauma such as a heavy fall in sporting activities or falling from a height (e.g. off a horse). Fractures, of course, have to be excluded.

Inflammatory disorders to consider are juvenile ankylosing spondylitis and spinal osteochondrosis (Scheuermann disease), which may affect adolescent males in the lower thoracic spine (around T9) and thoracolumbar spine. The latter condition may be asymptomatic, but can be associated with back pain, especially as the person grows older. It is the commonest cause of

kypnosis.

## § Kypnosis<sup>6</sup>

Kypnosis is the normal curve of the thoracic spine when viewed from the side. The normal range is 20–45° (see FIG. 27.4). An excessive angle (>45–50°) occurs with a kypnotic deformity. In children, a congenital cause is likely (present from infancy); in adolescents it is usually due to Scheuermann disease or is postural; in adults consider ankylosing spondylitis—and osteoporosis in the elderly. Tuberculosis of the spine can cause a gross deformity. Children with significant kypnosis should be referred for consideration of an intervention: exercises, bracing or surgery.



**FIGURE 27.4** Illustration of kyphosis, which is measured by the angle between the uppermost and lowermost inclined vertebrae on the lateral X-ray

## § Scheuermann disease

This is a structural saggital plane deformity with a dominant autosomal inheritance pattern affecting the T7, 8, 9 or T11, 12 areas.

## Clinical features

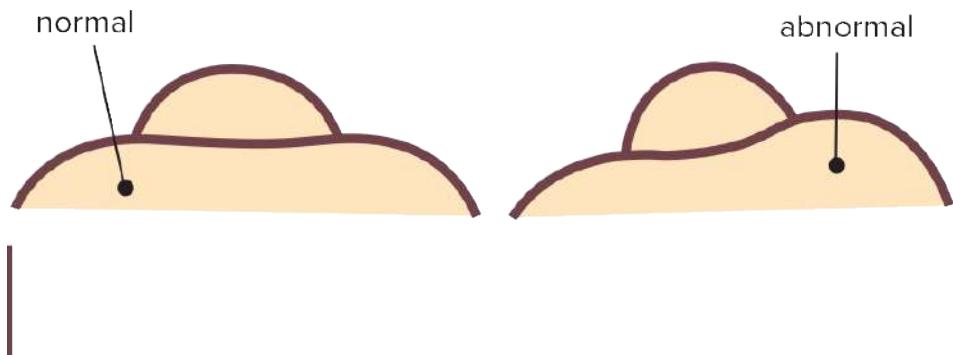
- Age 11–17 years
- Males > females
- Lower thoracic spine
- Thoracic pain or asymptomatic
- Increasing thoracic kyphosis over 1–2 months
- Wedging of the vertebrae
- Pain in the wedge, especially on bending (only 20% present with pain)
- Tight hamstrings, cannot touch toes
- Diagnosis confirmed by X-ray (lateral standing)—shows Schmorl node and anterior vertebral body wedging

## Treatment

- Explanation and support
- Extension exercises, postural correction and avoidance of sports involving lifting and bending have minimal evidence but are often suggested<sup>7</sup>
- Consider bracing or surgery if serious deformity

## § Adolescent idiopathic scoliosis

A degree of scoliosis is detectable in 5% of the adolescent population.<sup>8</sup> The vast majority of curves, occurring equally in boys and girls, are mild and of no consequence. Eighty-five per cent of significant curves in adolescent scoliosis occur in girls.<sup>8</sup> Inheritance is a factor. The highest incidence is in first-degree female relatives (12%). The scoliotic deformity develops at around 10 years of age. Such curves appear during the peripubertal period, usually coinciding with the growth spurt. A screening test is to note the contour of the back on forward flexion (see FIG. 27.5 ).



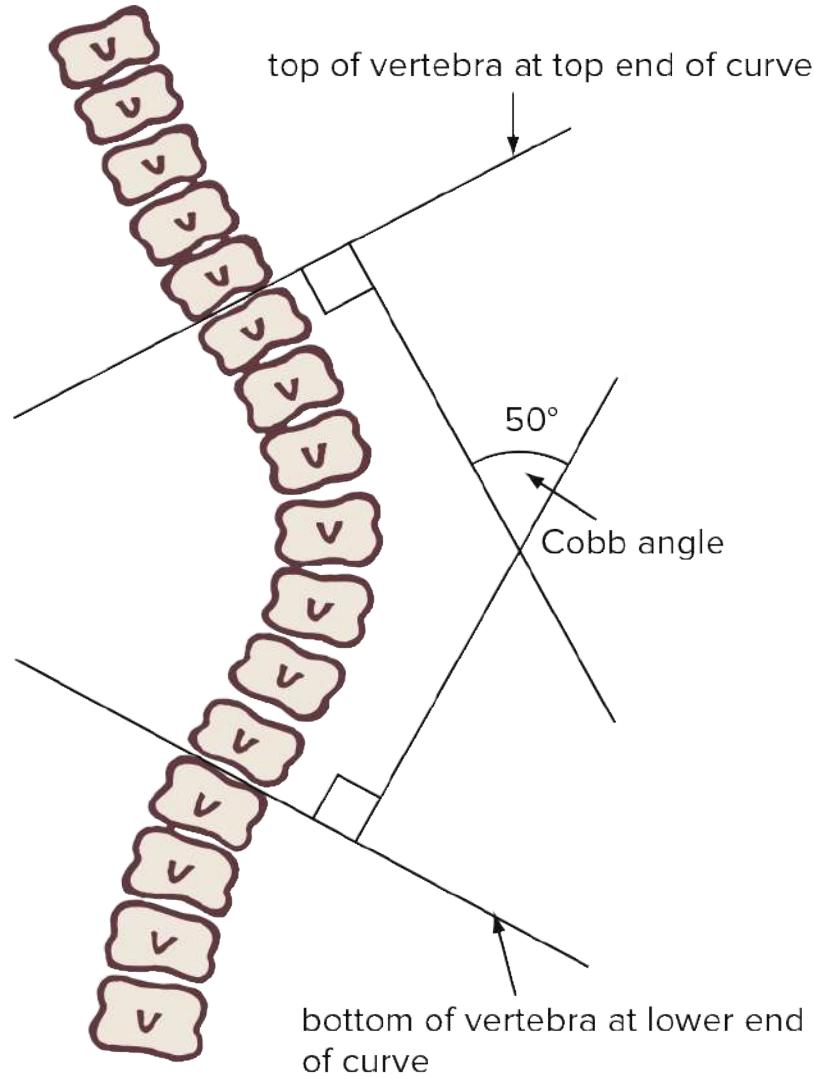
**FIGURE 27.5** Screening for adolescent idiopathic scoliosis: testing asymmetry by forward flexion. Viewed from behind the subject.

### The test

The subject stands with the feet parallel and together, and bends forward as far as possible with outstretched hands, palms facing each other, pointed between the great toes.

### Investigation

A single erect PA spinal X-ray is sufficient;<sup>9</sup> the Cobb angle (see FIG. 27.6 ) is the usual measurement yardstick.



**FIGURE 27.6** Scoliosis: the Cobb angle method of curve measurement

## Management

### Aims

- To preserve good appearance—level shoulders and no trunk shift
- Prevent increasing curve in adult life: less than 45°
- *Not* to produce a straight spine on X-ray

### Methods

- Braces:

Milwaukee brace (rarely used)  
high-density polyethylene underarm orthosis  
to be worn for 20–22 hours each day until skeletal maturity is reached.

- Surgical correction: depends on curve and skeletal maturity

### Guidelines for treatment

- Still growing:

|         |                                      |
|---------|--------------------------------------|
| <20°    | observe (repeat examination + X-ray) |
| 20–30°  | observe, brace if progressive        |
| 30–45°  | brace                                |
| ≥45–50° | operate                              |

- 
- Growth complete:

|      |             |
|------|-------------|
| <45° | leave alone |
| >45° | operate     |

Referral to consultant: >20°

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## Thoracic back pain in adults

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Elderly patients: thoracic back pain due to mechanical causes is not such a feature in the elderly, although vertebral dysfunction still occurs quite regularly. However, when the older person presents with thoracic pain, a very careful search for organic disease is necessary. Special problems to consider are:

- malignant disease (e.g. multiple myeloma, lung, prostate)
- osteoporosis
- vertebral pathological fractures
- polymyalgia rheumatica
- Paget disease (may be asymptomatic)
- herpes zoster

- visceral disorders: ischaemic heart disease, penetrating peptic ulcer, oesophageal disorders, biliary disorders

## Dysfunction of the thoracic spine

Also referred to as non-specific thoracic back pain, this is the outstanding cause of pain in adulthood presenting to the practitioner and is relatively easy to diagnose. It is often referred to as the thoracic hypomobility syndrome with the disorder arising in the facet joints, costovertebral joints and thoracic musculoligamentous structure, singularly or in combination. The most efficacious treatment for painful dysfunctional problems varies according to practitioners with a special interest in this area. There is a paucity of studies and evidence supporting the multiplicity of therapies, especially focal injections and physical therapy. Many claim and bear testimony that appropriate skilled mobilisation and manipulation therapy provides effective short-term, sometimes immediate, relief.<sup>10</sup>

### Typical profile:<sup>1</sup>

---

|                        |  |
|------------------------|--|
| Age                    | Any age, especially between 20 and 40 years  |
| History of injury      | Sometimes slow or sudden onset   |
| Site and radiation     | Spinal and paraspinal—interscapular, arms, lateral chest, anterior chest, substernal, iliac crest  |
| Type of pain           | Dull, aching, occasionally sharp; severity related to activity, site and posture   |
| Aggravation            | Deep inspiration, postural movement of thorax, slumping or bending, walking upstairs, activities (e.g. lifting children, making beds), beds too hard or soft, sleeping or sitting for long periods |
| Association            | Chronic poor posture   |
| Diagnosis confirmation | Examination of spine, therapeutic response to manipulation   |

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## Management

### First-line management

- Explanation with printed information
- Reassurance, including spontaneous recovery likely
- Continued activity according to pain level
- Back education program

- Analgesics, if required (e.g. paracetamol 1 g (o) qid or 1.33 g (o) 8 hourly), is first line
- Posture education and specific mobilising exercise program, esp. extension and rotation exercises to overcome stiffness
- Physical therapy: a short course of spinal mobilisation and manipulation (if appropriate) to help with pain and mobility

## Spinal mobilisation and manipulation

The evidence for manipulative therapies relieving back pain is generally disappointing; two Cochrane systematic reviews found that it is probably no better than placebo for acute pain, and very little better for chronic pain.<sup>11,12</sup> However, individual practitioners and patients seem to find immediate pain relief from spinal mobilisation or the more forceful manipulative therapy (used with extreme care, especially with osteoporosis). Many techniques can be employed, the choice depending on which part of the back is affected.<sup>9</sup>

## § Thoracic disc protrusion

Fortunately, a disc protrusion in the thoracic spine is uncommon. This reduced incidence is related to the firm splintage action of the ribcage. Most disc protrusions occur below T9, with the commonest site, as expected, being T11–12.

The common presentation is back pain and radicular pain that follows the appropriate dermatome so disc protrusion should be considered in those with neurological signs at thoracic levels. This may include a flaccid area of the lower abdominal musculature.

However, disc lesions in the thoracic spine are prone to produce spinal cord compression, manifesting as sensory loss, bladder incontinence and signs of upper motor neurone lesion. The disc is relatively inaccessible to surgical intervention, but may be reached via the transthoracic lateral approach.

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## § Syrinx

A syrinx usually comes to notice as a radiological finding in the presence of thoracic back pain when it may in fact be asymptomatic. It is a rare, fluid-filled neurological cavity within the spinal cord. It is usually a congenital anomaly, but a neoplasm needs to be excluded. An MRI of the spinal cord defines the problem. Any symptoms usually appear between adolescence and age 50, due to a central cord syndrome ([CHAPTER 51](#)). A syrinx usually begins at the cervical level and extends down. Treat conservatively, but refer to a specialist, who may consider surgical intervention if it is symptomatic.

## Muscle injury

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Muscular injuries such as tearing are uncommon in the chest wall. The strong paravertebral muscles do not appear to be a cause of chest pain, but strains of intercostal muscles, the serratus anterior and the musculotendinous origins of the abdominal muscles can cause pain. Injuries to these muscles can be provoked by attacks of violent sneezing or coughing, or overstrain, e.g. lifting a heavy suitcase down from an overhead luggage rack.

## Scapulothoracic joint disorders<sup>13</sup>

The gliding plane between the scapula and thoracic wall permits a considerable range of scapular movement, which contributes significantly to movement of the shoulder. Several muscles, including the rhomboids, serratus anterior and levator scapulae, help stabilise scapular movement and may be a source of pain in the scapular region.

### ⌚ Snapping scapula

The person complains of a loud cracking or snapping sound upon abduction of the scapula. There is often associated crepitus. Pain is felt along the medial scapular border. Some people develop a habit ('tic') of neurotically clicking the shoulder back and forth.

On examination, there is usually generalised hypermobility of the scapula, abnormal movement and tenderness to palpation along the medial edge on full abduction.

The cause (uncommon) may be an underlying bony abnormality such as a bony spur on the superior border of the scapula or an osteoma. X-rays should include a lateral view of the scapula to search for this possibility.

### Treatment

- Explanation and reassurance (if X-rays normal)—otherwise resect any bone abnormality.
- Avoid repeated scapular movement and 'trick' movements.
- Appropriate exercises under physiotherapy supervision.
- Infiltrate any very tender area in the muscle (with care) with local anaesthetic and steroid.
- Deep massage to the tender focus.

### ⌚ Scapulocostal syndrome

This condition causes localised pain and tenderness, often severe, along the upper part of the medial scapular border, with radiation around the chest wall and shoulder girdle to the neck. Pain is usually worse with prolonged shoulder use towards the end of the day. It is commonly seen in typists, gymnasts and other sportspeople. It is related to poor posture. The cause may include friction between the scapula and the thoracic wall, scoliosis, trauma and myofascial strain due to poor posture.

## Treatment

- Avoid the movements producing the pain.
- Posture and re-education exercises and scapula stretching.
- Deep friction massage.
- Local injections of local anaesthetic and corticosteroid into the tender area.

## ⌚ Winging of the scapula

The asymmetry may not be apparent until the patient tries to contract the serratus anterior against resistance by pushing the outstretched arm against a wall. There may be parascapular discomfort. The common cause is neurogenic paralysis of the serratus anterior muscle. Paralysis may result from injury to the long thoracic nerve (from C5, 6, 7 nerve roots) such as a neck injury or a direct blow to the suprascapular area and from injury to the brachial plexus such as excessive carrying of heavy packs, severe traction on the arm or forceful cervical manipulation. Most cases settle spontaneously, although it may take 1–2 years.

## Fibromyalgia, fibrositis and myofascial trigger points

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Fibromyalgia is relatively uncommon, but when encountered it presents an enormous management problem. It is not to be confused with so-called fibrositis or tender trigger points. Referral to a specialist with expertise in this condition or to a multidisciplinary pain clinic for the definitive diagnosis is recommended.

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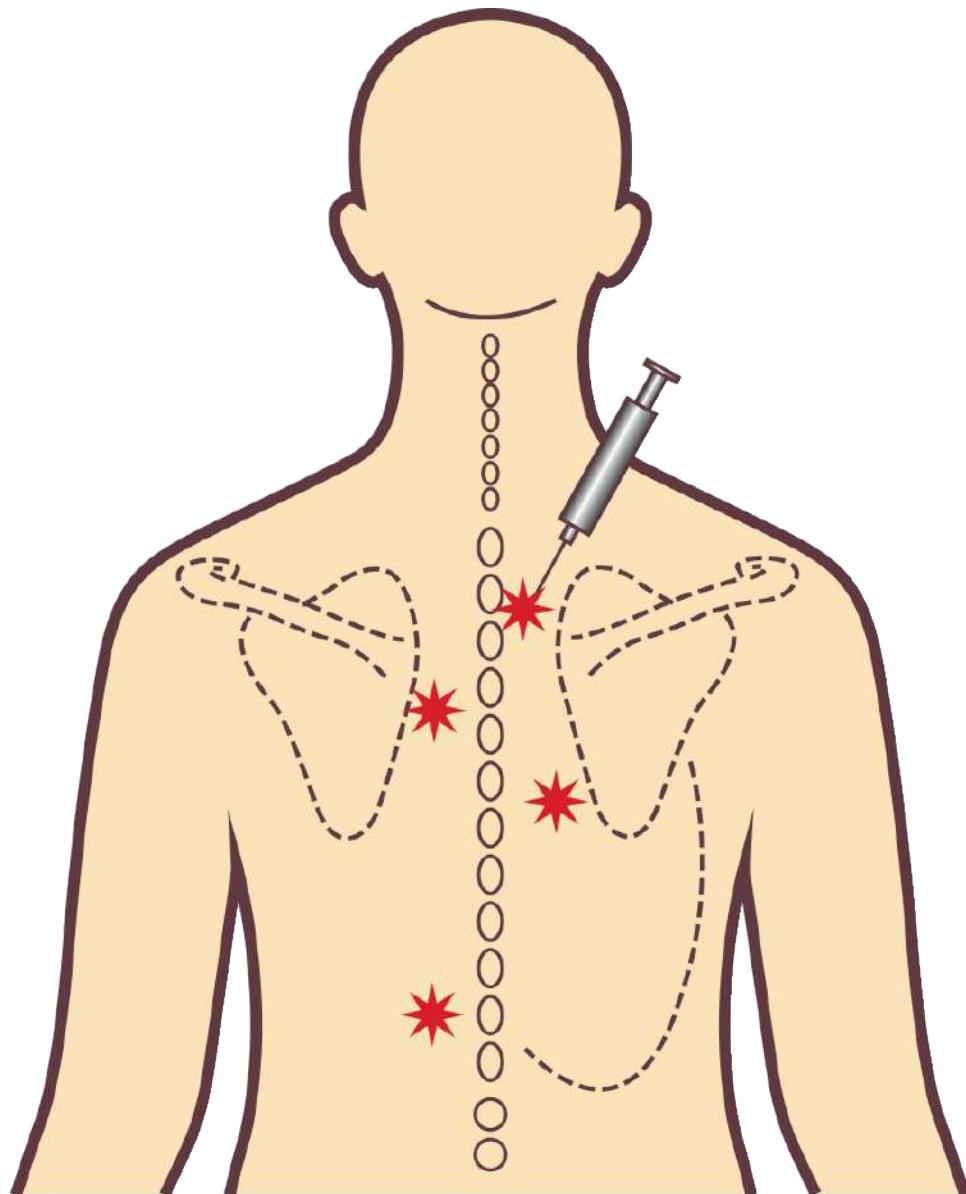
Fibrositis is not a diagnosis but a symptom, indicating a localised area of tenderness or pain in the soft tissues, especially of the upper thoracic spine. It is probably almost always secondary to upper thoracic or lower cervical spinal dysfunction.

## ⌚ Myofascial trigger points

As described by Travell and Rinzler in 1952,<sup>14</sup> a trigger point is characterised by local tenderness in a muscle that twitches upon stimulation and causes referred pain when subjected to pressure. However, under blinded conditions there is little consistency in the science behind reliably identifying those points.<sup>15</sup> Regardless, local injection is relatively easy and safe, and individuals may experience temporary pain relief.

## Treatment<sup>16</sup>

Identify the maximal point of pain and inject 5–8 mL of local anaesthetic (e.g. lignocaine/lidocaine 1%. Do not use corticosteroids) into the painful point (see FIG. 27.7 ). Post-injection massage or exercises should be performed.



**FIGURE 27.7** Injection for myofascial trigger points

## ⌚ Fibromyalgia syndrome<sup>17</sup>

AKA chronic diffuse non-inflammatory pain. Its pathophysiology is poorly understood.

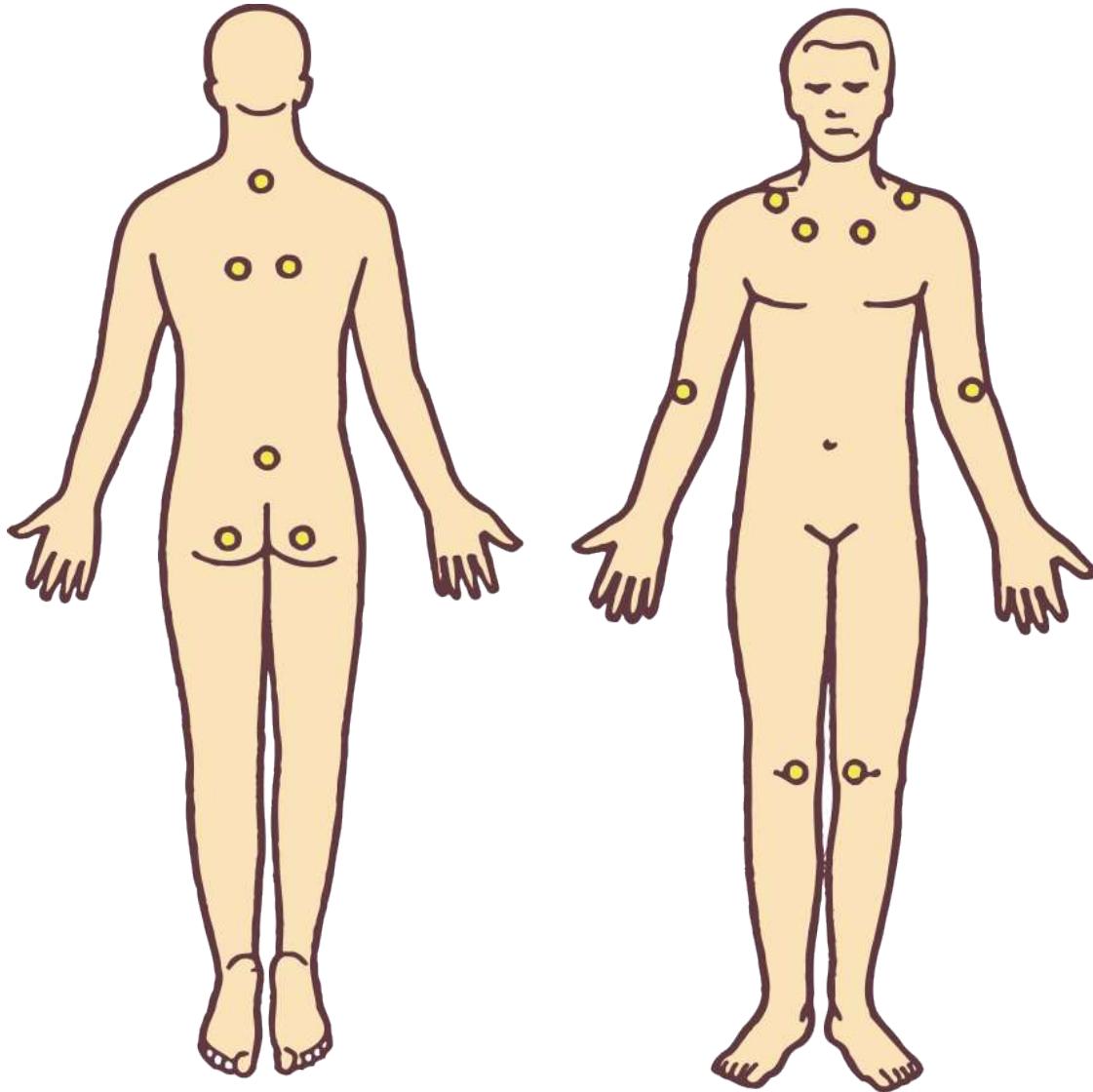
### Clinical features

The main diagnostic features are:<sup>18</sup>

- 1. a history of widespread pain (neck to low back) affecting all four body quadrants

- 1. fatigue, sleep problems, cognitive disturbance
- 2. pain in 11 of 18 tender points on digital palpation (the original definition in 1990)
- 3. pain for at least 3 months

These points must be painful, not tender. Smythe and Moldofsky have recommended 14 of these points on a map as a guide for management<sup>17</sup> (see FIG. 27.8). No consistent measurable investigations have been identified. If ESR/CRP are elevated, look for alternative diagnoses.



**FIGURE 27.8** Fibromyalgia syndrome: typical tender points (the tender point map represents the 14 points recommended for use as a standard for diagnostic or therapeutic studies)

## Other features

- Female to male ratio = 4:1
- Usual age onset 29–37 years: diagnosis 44–53 years
- Positive family history
- Psychological disorders (e.g. anxiety, depression, tension headache, irritable digestive system)

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This disorder is difficult to treat, and the GP needs to coordinate care and be responsive to individual needs as they evolve over time.<sup>18</sup> Management requires considerable explanation, support (counselling) and reassurance. Best evidence to date supports the value of educational programs and regular aerobic exercise.<sup>18,19,20</sup> Treat pain (simple analgesics—paracetamol) and depression (psychologist referral, antidepressants) on their merits. Consider referral to a specialist or fibromyalgia clinic. Referral to allied health practitioners can be very useful.

*Note:* NSAIDs and narcotics are of no proven benefit.

## Medication (often disappointing but worth a trial)

Antidepressants (of short-term value, where relevant);<sup>21</sup> start low then monthly increments as tolerated:

amitriptyline 10–50 mg (o) nocte

*or*

dothiepin 25–75 mg (o) nocte

*or*

duloxetine 30 mg (o) mane, increasing to 60 mg over 2 weeks<sup>22</sup>

*Note:* NSAIDs and narcotics are of no proven benefit.

## Diffuse idiopathic skeletal hyperostosis (DISH)

DISH is ligamentous ossification of the spine leading to progressive stiffness and possible pain in some parts. Possible associated metabolic disorder, e.g. diabetes. Ankylosing spondylitis is a differential diagnosis.

There is no specific treatment. Manage as for spinal dysfunction.

## Serious pitfalls

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The following points regarding serious vertebral organic disease are worth repeating in more

detail.

## Metastatic disease<sup>23</sup>

Secondary deposits in the thoracolumbar spine may be the first presenting symptoms of malignant disease. Any patient of any age presenting with progressive severe nocturnal back pain should be regarded as having a tumour and investigated with a technetium bone scan as part of the primary investigations.

Secondary deposits in the spine can lead to rapid onset paralysis due to spinal cord infarction. Many such metastases can be controlled in the early stages with radiotherapy.

## Multiple myeloma

Osteoporotic vertebral body collapse should be diagnosed only when multiple myeloma has been excluded. Investigations should include an ESR, Bence–Jones protein analysis and immunoglobulin electrophoresis.

Early treatment of multiple myeloma can hold this disease in remission for many years and prevent crippling vertebral fractures (see [CHAPTER 17](#) ).

## Infective discitis, vertebral osteomyelitis and epidural/subdural abscess

Severe back pain in an unwell patient with fluctuating temperature (fever) should be considered as infective until proved otherwise. Investigations should include blood cultures, serial X-rays and nuclear bone scanning. Biphasic bone scans using technetium with either indium or gallium scanning for white cell collections usually clinch this diagnosis.

Strict bed rest with high-dose antibiotic therapy is usually curative. If left untreated, vertebral end plate and disc space collapse is common and extremely disabling. Consider tuberculosis osteomyelitis in people at risk. Suspect an epidural abscess in the presence of persistent and increased back pain. Percuss the spine for localised tenderness (see [CHAPTER 20](#) ).

## When to refer

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- Persistent pain or dysfunction—refer to a physiotherapist or exercise specialist.
- Evidence or suspicion of a sinister cause (e.g. neoplasia, infective discitis/osteomyelitis in a child).
- Suspicion of cardiac or gastrointestinal referred (persistent) pain.
- Significant adolescent scoliosis or kyphosis (e.g. Scheuermann disease).

# Patient education resources

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

- Exercises for your thoracic spine
- Fibromyalgia
- Scoliosis

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## Practice tips

- Feelings of anaesthesia or paraesthesia associated with thoracic spinal dysfunction are rare.
- Thoracic back pain is frequently associated with cervical lesions.
- Upper thoracic pain and stiffness is common after 'whiplash'.
- The T4 syndrome of upper to mid-thoracic pain with radiation (and associated paraesthesia) to the arms is well documented.
- Symptoms due to a fractured vertebra usually last 3 months and to a fractured rib 6 weeks.
- The pain of myocardial ischaemia, from either angina or myocardial infarction, can cause referred pain to the interscapular region of the thoracic spine.
- Beware of the old trap of herpes zoster in the thoracic spine, especially in the older person.
- Consider multiple myeloma as a cause of an osteoporotic collapsed vertebra.
- Examine movements with the patient sitting on the couch and hands clasped behind the neck.
- Spinal disease of special significance in the thoracic spine includes osteoporosis and neoplasia, while disc lesions, inflammatory diseases and degenerative diseases (spondylosis) are encountered less frequently than with the cervical and lumbar spines.
- It is imperative to differentiate between spinal and cardiac causes of chest pain: either cause is likely to mimic the other. A working rule is to consider the cause as cardiac until the examination and investigations establish the true cause.
- Always X-ray the thoracic spine following substantial trauma, especially after

motor vehicle accidents, as wedge compression fractures (typically between T4 and T8) are often overlooked.

- Plain X-ray, CT scan or MRI are of no utility in patients with non-specific thoracic back pain.
- MRI is the most appropriate imaging to investigate suspected serious pathology.

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## 28 Low back pain

*Last Wednesday night while carrying a bucket of water from the well, Hannah Williams slipped upon the icy path and fell heavily upon her back. We fear her spine was injured for though she suffers acute pain in her legs she cannot move them. The poor wild beautiful girl is stopped in her wildness at last.*

FRANCIS KILVERT 1874

Low back pain accounts for at least 5% of general practice presentations. It is a massive problem worldwide. The most common cause is minor soft tissue injury, but patients with this do not usually seek medical help because the problem settles within a few days.

Most back pain in patients presenting to GPs is postulated to be due to dysfunction of elements of the mobile segment, namely the facet joint, the intervertebral joint (with its disc) and the ligamentous and muscular attachments. This problem, often referred to as mechanical back pain, will be described as vertebral dysfunction—a general term that, while covering radicular and non-radicular pain, includes dysfunction of the joints of the spine, although the specific origin in most instances cannot be determined. It is therefore appropriate to refer to this as ‘non-specific back pain’.<sup>1</sup>

### Key facts and checkpoints

- Back pain accounts for 2.6% of all presenting problems in Australian general practice.<sup>2</sup>
- In the US it is the commonest cause of limitation of activity in those under the age of 45.<sup>3</sup>
- Approximately 85–90% of the population will experience back pain at some stage of their lives, while 70% of the world’s population will have at least one disabling episode of low back pain in their lives.<sup>3</sup>
- At least 50% of these people will recover within 2 weeks and 90% within 6 weeks, but recurrences are frequent and have been reported in 40–70% of

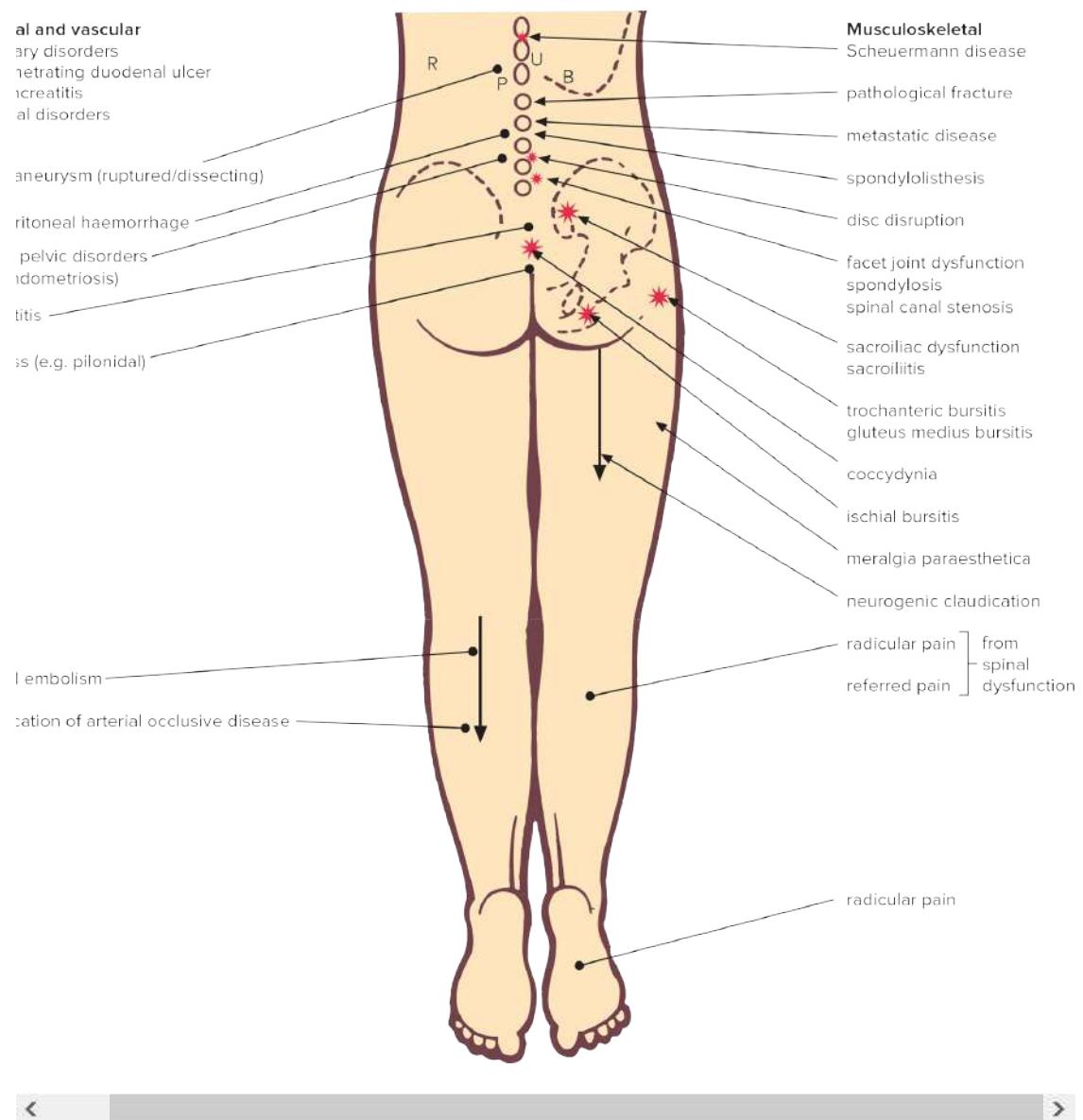
patients; 2–7% develop chronic pain.<sup>4</sup>

- It most commonly occurs in those aged 30–60 years, the average age being 45 years.<sup>5</sup>
- It is difficult to assign a specific pathoanatomical cause in acute back pain (perhaps 8–15%),<sup>6</sup> but the most common cause is probably a minor muscle/ligament strain (often don't present to a doctor), followed by dysfunction of the intervertebral joints of the spine ('mechanical back pain') and spondylosis (synonymous with osteoarthritis and degenerative back disease).
- L5 and S1 nerve root lesions represent most of the cases of sciatica presenting in general practice. They tend to present separately but can occur together with a massive disc protrusion.
- An intervertebral disc prolapse is causative in only 6–8% of cases of back pain,<sup>3</sup> and only a small fraction of those require urgent diagnosis and surgical treatment.

## Causes of low back pain

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To develop a comprehensive diagnostic approach, the practitioner should have a clear understanding of the possible causes of low back and leg pain (see FIG. 28.1 ) and of the relative frequency of their clinical presentations.



**FIGURE 28.1** Relevant causes of back pain with associated buttock and leg pain

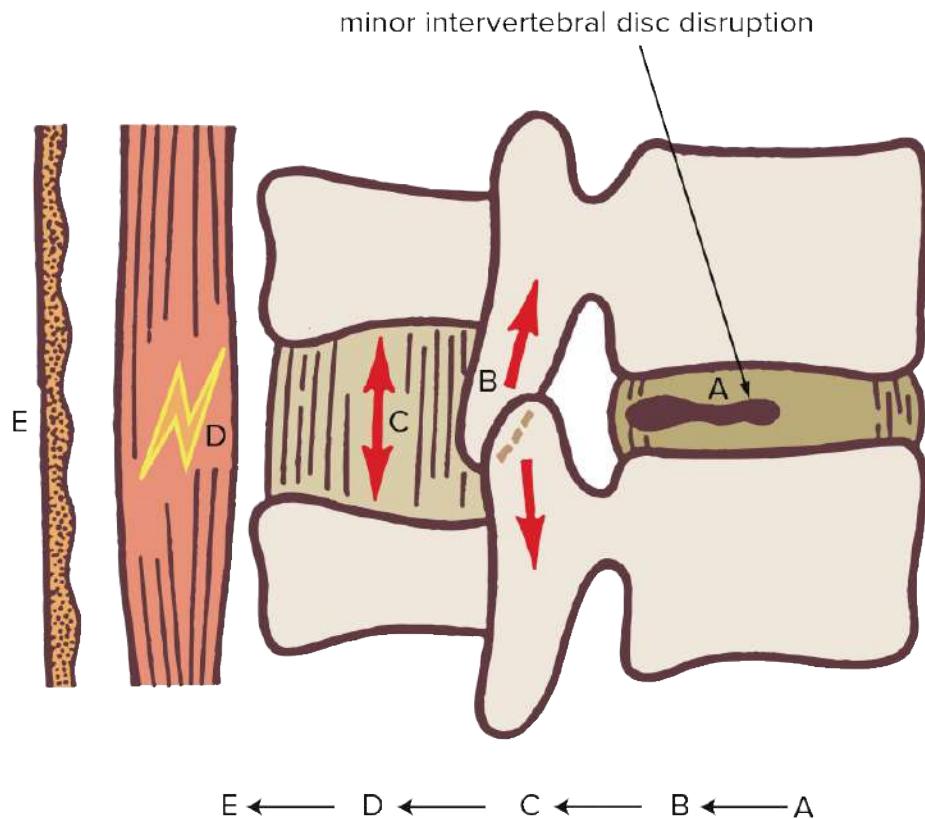
## Anatomical and pathophysiological concepts

Studies have focused on the importance of disruption of the intervertebral disc in the cause of back pain. A very plausible theory has been advanced by Maigne<sup>5</sup> who proposes the existence, in the involved mobile segment, of a minor intervertebral derangement (MID). He defines it as ‘isolated pain in one intervertebral segment, of a mild character, and due to minor mechanical cause’.

The MID always involves one of the two apophyseal joints in the mobile segment, thus initiating

nociceptive activity in the posterior primary dermatome and myotome.

Maigne points out that the functional ability of the mobile segment depends intimately upon the condition of the intervertebral disc. Thus, if the disc is injured, other elements of the segment will be affected.



**FIGURE 28.2** Reflex activity from a MID in the intervertebral motion segment.

Apart from the local effect caused by the disruption of the disc (A), interference can occur in the facet joint (B) and interspinous ligament (C) leading possibly to muscle spasm (D) and skin changes (E) via the posterior rami.

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

In theory, any structure with a nociceptive nerve supply may be a source of pain. Such structures include the ligaments, fascia and muscles of the lumbosacral spine, intervertebral joints, facet joints, dura mater and sacroiliac joints.<sup>7</sup>

Actually, pain can theoretically arise from any innervated structure in the region of the spine. It can be neurogenic, spondylogenetic, viscerogenic, vasculogenic or rarely psychogenic.

## A diagnostic approach

A summary of the diagnostic strategy model is presented in TABLE 28.1 .

**Table 28.1** Low back pain: diagnostic strategy model

**Probability diagnosis**

Vertebral dysfunction (non-specific low back pain)

Musculoligamentous strain/sprain

Spondylosis (degenerative OA)

**Serious disorders not to be missed**

Cardiovascular:

- ruptured aortic aneurysm
- retroperitoneal haemorrhage (anticoagulants)

Neoplasia:

- myeloma
- carcinoma of pancreas
- metastases

Severe infections:

- vertebral osteomyelitis
- epidural abscess
- septic discitis
- tuberculosis
- pelvic abscess/PID

Osteoporotic compression fracture/other fracture

Cauda equina compression

**Pitfalls (often missed)**

Spondyloarthropathies:

- ankylosing spondylitis
- reactive arthritis
- psoriasis
- bowel inflammation

Sacroiliac dysfunction

Spondylolisthesis

Claudication:

- vascular
- neurogenic/spinal canal stenosis

Paget disease

Prostatitis  
Endometriosis

### Seven masquerades checklist

Depression  
Spinal dysfunction  
UTI

### Is the patient trying to tell me something?

Quite likely. Consider lifestyle, stress, work problems, malingering, conversion reaction.

Note: Associated buttock and leg pain included.

## Probability diagnosis<sup>7</sup>

The commonest cause of low back pain is vertebral dysfunction or mechanical pain, which then has to be further analysed. The term can embrace musculoskeletal strain, discogenic and posterior ligament pain, and facetogenic dysfunction/pain.

Degenerative changes in the lumbar spine (lumbar spondylosis) are commonly found in the older age group. This problem, and one of its complications, spinal canal stenosis, is steadily increasing along with the ageing population.

## Serious disorders not to be missed

It is important to consider malignant disease, especially in an older person. It is also essential to consider infection such as acute osteomyelitis and tuberculosis, which is often encountered in recent immigrants, especially those from Asia and central Africa. The uncommon epidural or subdural abscess should also be kept in mind, especially if any fever (see CHAPTER 20 ). These conditions are considered in more detail under infections of the central nervous system. For pain or anaesthesia of sudden onset, especially when accompanied by neurological changes in the legs, consider cauda equina compression due to a massive disc prolapse and also retroperitoneal haemorrhage. It is important to ask patients if they are taking anticoagulants. See TABLE 28.2 .

**Table 28.2** ‘Red flag’ pointers to serious low back pain conditions<sup>8</sup>

Age >50 years or <20 years; consider osteoporosis  
History of cancer  
Temperature >37.8°C  
Constant pain—day and night esp. severe night pain  
Unexplained weight loss

Symptoms in other systems, e.g. cough, breast mass  
Significant trauma; sometimes mild trauma  
Features of spondyloarthropathy, e.g. peripheral arthritis (e.g. age <40 years, night-time waking)  
Neurological deficit, e.g. numbness, paraesthesia in limb  
Drug or alcohol abuse, especially IV drug use  
Use of anticoagulants  
Use of corticosteroids  
No improvement over 1 month  
Possible cauda equina syndrome:

- saddle anaesthesia
- recent onset bladder dysfunction/overflow incontinence
- bilateral or progressive neurological deficit

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## Pitfalls

The inflammatory disorders must be kept in mind, especially the spondyloarthropathies, which include psoriatic arthropathy, ankylosing spondylitis, reactive arthritis and inflammatory bowel disorders such as ulcerative colitis and Crohn disease. The spondyloarthropathies are more common than appreciated and must be considered in the younger person presenting with features of inflammatory back pain (i.e. pain at rest, relieved by activity). The old trap of confusing claudication in the buttocks and legs, due to a high arterial obstruction, with sciatica must be avoided.

### General pitfalls

- Being unaware of the characteristic symptoms of inflammation and thus misdiagnosing one of the spondyloarthropathies.
- Overlooking the early development of malignant disease or osteomyelitis; if suspected, and an X-ray is normal, a radionuclide scan should detect the problem.
- Failing to realise that mechanical dysfunction and osteoarthritis can develop simultaneously, producing a combined pattern.
- Overlooking anticoagulants as a cause of a severe bleed around the nerve roots, or corticosteroids leading to osteoporosis.
- Not recognising back pain as an infective complication of IV drug use.

### Red flag pointers for low back pain

The 'red flag' symptoms or signs (see TABLE 28.2 ) should alert the practitioner to a serious health problem and thus guide selection of investigations, particularly appropriate imaging of the lumbar spine.

## Seven masquerades checklist

Of these conditions, depression and urinary tract infection have to be seriously considered. For the young woman with upper lumbar pain, especially if she is pregnant, the possibility of a urinary tract infection must be considered even in the absence of urinary symptoms such as dysuria and frequency.

Depressive illness has to be considered in any chronic pain complaint. This common psychiatric disorder can continue to aggravate or maintain the pain even though the provoking problem has disappeared.

## Psychogenic considerations

Chronic back pain is more likely to occur in people who have become anxious about their problem or who are under excessive stress. It may be necessary to probe beneath the surface of the presenting problem. Consider counselling, or a therapeutic trial of an antidepressant medication where appropriate. The possibility of malingering should also be considered in some circumstances, although with great caution.

Low back pain following lifting at work poses a problem that causes considerable anguish to doctors, especially when the pain becomes chronic and complex. Chronic pain may be the last straw for those who have been struggling to cope with personal problems; their fragile equilibrium is upset by the back pain. The importance of a caring, competent practitioner with an insight into all facets of his or her patient's suffering, organic and functional, becomes obvious. The tests for non-organic back pain are very useful in this context.

## Yellow flag pointers

This term has been introduced to identify psychosocial and occupational factors that may increase the risk of chronicity in people presenting with acute back pain.  
Consider psychological issues if:

- abnormal illness behaviour
- 'fear avoidance': concern re pain on activity
- compensation issues
- unsatisfactory restoration of activities

- failure to return to work
- unsatisfactory response to treatment
- treatment refused
- atypical presenting physical signs

## Nature of the pain

The nature of the pain may reveal its likely origin. Establish where the pain is worst—whether it is central (proximal) or peripheral. The following are general characteristics and guides to diagnosis:

- aching throbbing pain = inflammation (e.g. sacroiliitis)
- deep aching diffuse pain = referred pain (e.g. dysmenorrhoea)
- superficial steady diffuse pain = local pain (e.g. muscular strain)
- boring deep pain = bone disease (e.g. neoplasia, Paget disease)
- intense sharp or stabbing (superimposed on a dull ache) = radicular pain (e.g. sciatica)

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A comparison of the significant features of the two most common types of pain—mechanical and inflammatory—is presented in [TABLE 28.3](#).

**Table 28.3** Comparison of the patterns of pain for inflammatory and mechanical causes of low back pain<sup>9</sup>

| Feature        | Inflammation                           | Mechanical                                |
|----------------|--|---|
| History        | Insidious onset                        | Precipitating injury/previous episodes    |
| Nature         | Aching, throbbing                      | Deep dull ache, sharp if root compression |
| Stiffness      | Severe, prolonged<br>Morning stiffness | Moderate, transient                       |
| Effect of rest | Exacerbates                            | Relieves                                  |
| Effect of      | Relieves                               | Exacerbates                               |

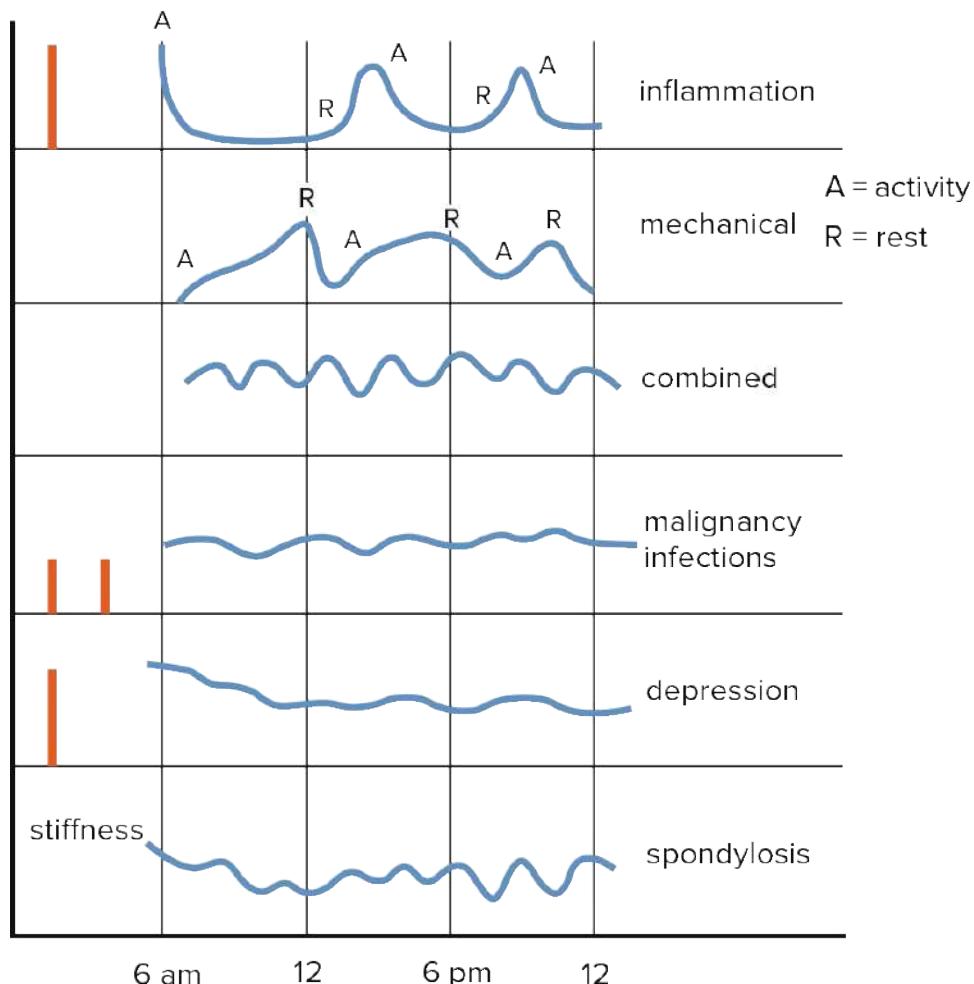
|           |  |                                 |
|-----------|--|---------------------------------|
| activity  |  |                                 |
| Radiation | More localised, bilateral or alternating | Tends to be diffuse, unilateral |
| Intensity | Night, early morning                     | End of day, following activity  |

## The clinical approach

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### History

Analysing the history invariably guides the clinician to the diagnosis. The pain patterns have to be carefully evaluated and it is helpful to map the diurnal variations of pain to facilitate the diagnosis (see FIG. 28.3 ).



**FIGURE 28.3** Typical daily patterns of pain for conditions causing back pain.

Note conditions that can wake people from sleep (red spikes) and also the pattern of combined mechanical and inflammatory pain.

It is especially important to note the intensity of the pain and its relation to rest and activity. In particular, ask whether the pain is present during the night, whether it wakes the patient, is present on rising or whether it is associated with stiffness.

Continuous pain, present day and night, is suggestive of neoplasia or infection. Pain on waking suggests inflammation or depressive illness. Pain provoked by activity and relieved by rest suggests mechanical dysfunction, while pain worse at rest and relieved by moderate activity is typical of inflammation. In some patients the coexistence of mechanical and inflammatory causes complicates the pattern.

Pain aggravated by standing or walking that is relieved by sitting is suggestive of spondylolisthesis. Pain aggravated by sitting (usually) and improved with standing indicates a discogenic problem.

Pain of the calf that travels proximally with walking indicates vascular claudication; pain in the buttock that descends with walking indicates neurogenic claudication. This latter problem is encountered more frequently in older people who have a tendency to spinal canal stenosis associated with spondylosis.

## Key questions

- What is your general health like?
- Can you describe the nature of your back pain?
- Was your pain brought on by an injury?
- Is it worse when you wake in the morning or later in the day?
- How do you sleep during the night?
- What effect does rest have on the pain?
- What effect does activity have on the pain?
- Is the pain worse when sitting or standing?
- What effect does coughing or sneezing, or straining at the toilet have?
- What happens to the pain in your back or leg if you go for a long walk?
- Do you have a history of psoriasis, diarrhoea, penile discharge, eye trouble or severe pain in your joints?
- Do you have any urinary symptoms?

- What medication are you taking? Are you on anticoagulants?
- Are you under any extra stress at work or home?
- Do you feel tense, depressed or irritable?

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## Examination

### Physical examination

The basic objectives of the physical examination are to reproduce the symptoms, detect the level of the lesion and determine the cause (if possible) by provocation of the affected joints or tissues. This is done using the time-honoured method of joint examination—look, feel, move and test function. The patient should be stripped to a minimum of clothing so that careful examination of the back can be made. A neurological examination of the lower limb should be performed if symptoms extend below the buttocks. If cauda equina syndrome is suspected, perform a rectal examination to check for flaccidity.

A useful screening test for a disc lesion and dural tethering is the slump test.<sup>10</sup>

The main components of the physical examination are:

1. inspection
2. active movements: note which ones reproduce the symptoms
  - forward flexion
  - extension
  - lateral flexion (R and L)
3. provocative tests (to reproduce the symptoms)
4. palpation (to detect level of pain)
5. neurological testing of lower limbs (if appropriate)
6. testing of related joints (hip, sacroiliac)
7. assessment of pelvis and lower limbs for any deformity (e.g. leg shortening)
8. general medical examination, including rectal examination if appropriate

### Important landmarks

The surface anatomy of the lumbar region is the basis for determining the vertebral level. Key

anatomical landmarks include the iliac crest, spinous processes, the sacrum and the posterior superior iliac spines (PSISs).

- The tops of the iliac crest lie at the level of the L3–4 interspace (or the L4 spinous process).
- The PSISs lie opposite S2.

## Inspection

Inspection begins from the moment the patient is sighted in the waiting room. A person who is noted to be standing is likely to have a significant disc lesion. Considerable information can be obtained from the manner in which they arise from a chair, move to the consulting room, remove shoes and clothes, get onto the examination couch and move when unaware of being watched.

The spine must be adequately exposed and inspected in good light. Patients should undress to their underwear; women may retain their bra and ideally provide them with a gown that opens down the back. Note the general contour and symmetry of the back and legs, including the buttock folds, and look for muscle wasting. Note the lumbar lordosis and any abnormalities, such as lateral deviation. If lateral deviation (functional scoliosis) is present it is usually away from the painful side.

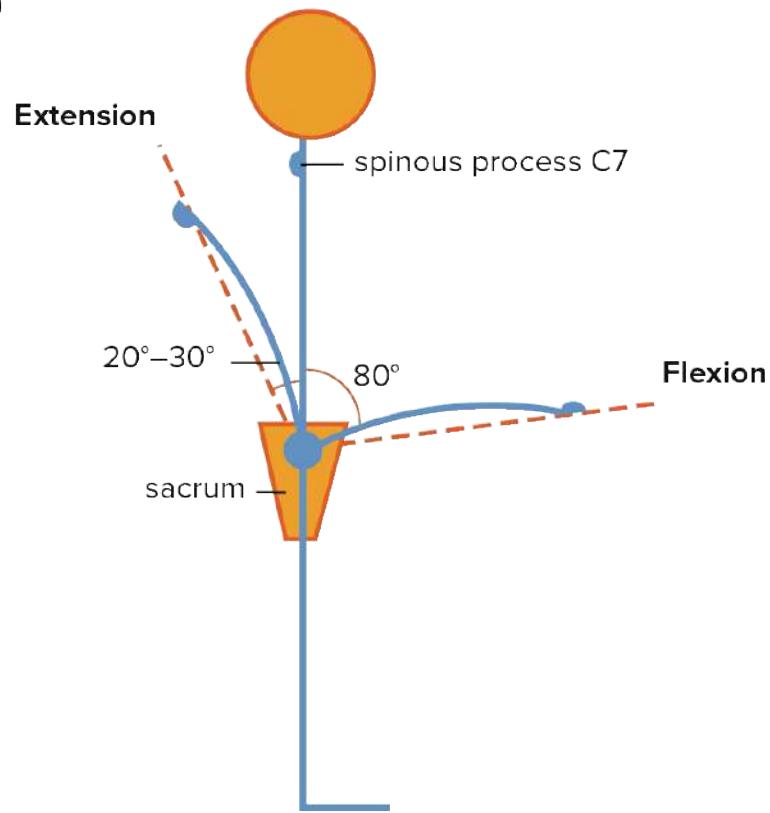
Note the presence of midline moles, tufts of hair or haemangioma that might indicate an underlying congenital anomaly, such as spina bifida occulta.

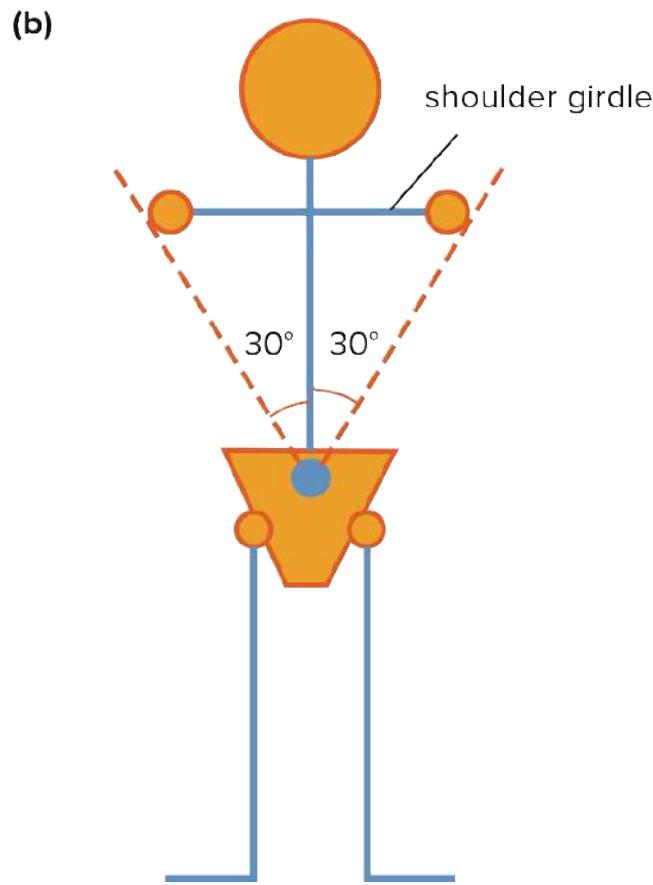
## Movements of the lumbar spine

There are three main movements of the lumbar spine. However, rotation, which mainly occurs at the thoracic spine, is not usually measured. The movements that should be tested, and their normal ranges, are as follows:

- extension (20–30°) (see FIG. 28.4A )
- lateral flexion, left and right (30°) (see FIG. 28.4B )
- flexion (75–90°: average 80°, or record where fingertips reach) (see FIG. 28.4A )

(a)





**FIGURE 28.4 (a)** Degrees of movement of the lumbar spine: flexion and extension, **(b)** degree of lateral flexion of the lumbar spine

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

Measurement of the angle of movement can be made by using a line drawn between the sacrum and large prominence of the C7 spinous process.

### Palpation

Have the patient relaxed, lying prone, with the head to one side and the arms by the sides. The levels of the spinous processes are identified by standing behind the patient and using your hands to identify the L4 spinous process in relation to the top of the iliac crests. Mark the important reference points.

Palpation, which is performed with the tips of the thumbs opposed, can commence at the spinous process of L1 and then systematically proceed distally to L5 and then over the sacrum and coccyx. Include the interspinous spaces as well as the spinous processes. When the thumbs (or other part of the hand such as the pisiforms) are applied to the spinous processes, a firm pressure is transmitted to the vertebrae by a rocking movement for three or four ‘springs’. Significant reproduction of pain is noted.

Palpation occurs at three main sites:

- centrally (spinous processes to coccyx)
- unilateral—right and left sides (1.5 cm from midline)
- transverse pressure to the sides of the spinous processes (R and L)

### **Straight leg raising (SLR) test (Lasègue test)**

This test is a passive test by the practitioner. The patient lies supine with both knees extended and the ankle dorsiflexed. The affected leg is raised slowly, keeping the knee extended. If sciatica with dural irritation is present, 20° to 60° of elevation causes reproduction of pain.

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### **Slump test**

The slump test is an excellent provocation test for lumbosacral pain and is more sensitive than the SLR test. It is a screening test for a disc lesion and dural tethering. It should be performed on those who have low back pain with pain extending into the leg, and especially for posterior thigh pain.

A positive result is reproduction of the patient's pain, and may appear at an early stage of the test (when it is ceased).

Method:

1. The patient sits on the couch in a relaxed manner with knees at the edge of the table.
2. The patient then slumps forward (without excessive trunk flexion), and then places the chin on the chest.
3. The unaffected leg is straightened.
4. The affected leg only is then straightened (see FIG. 28.5 ).