INTERNATIONAL EDITION

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Davidson's Principles and Practice of Medicine

24th Edition

70th ANNIVERSARY EDITION

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ELSEVIER

Clinical evaluation and management in a patient with chronic pain or in the palliative care setting

character and radiation of pain

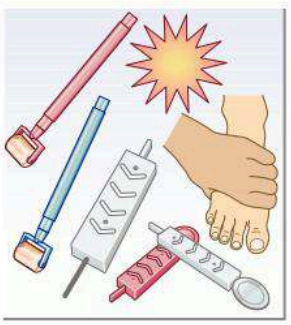
Take careful history, recording

Consider psychological aspects of pain assessment

Assess mood and screen for depression

Conduct general examination

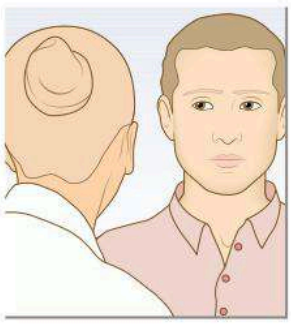
Check gait and whether using a walking aid



Assess pinprick, fine touch and heat/cold sensation

Conduct neurological examination

Educate patient on nature of pain and promote self-management



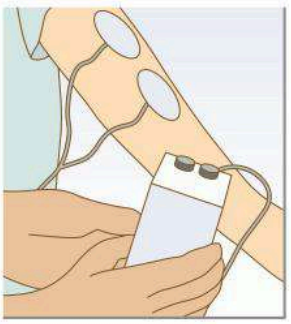
Formulate management plan with patient and set goals

Optimise medication

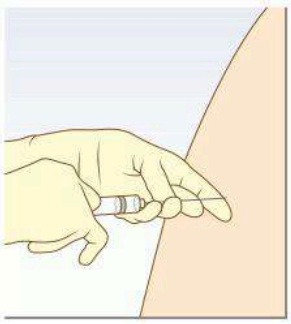
Consider psychological therapies and mindfulness

Increase physical activity

Consider yoga, pilates or tai chi



Consider TENS and acupuncture



Consider nerve block or ablation

(TENS = transcutaneous electrical nerve stimulation)

8.8 Endogenous opioids and opioid receptors

Endogenous ligand	Receptor (IUPHAR)	Alternative classification	Potential sites	Pharmacological effects
Endomorphin 1 and 2 Met-enkephalin Dynorphin A Dynorphin B	MOP	Mu	Brain, spinal cord, peripheral nerves, immune cells	Analgesia, reduced gastrointestinal motility, respiratory depression, pruritus
Leu-enkephalin Met-enkephalin β-endorphin	DOP	Delta	Brain, spinal cord, peripheral nerves	Analgesia, cardioprotection, thermoregulation
Dynorphin A Dynorphin B β-endorphin	KOP	Kappa	Brain (nucleus accumbens, neocortex, brainstem, cerebellum)	Analgesia, neuroendocrine (hypothalamic–pituitary axis), diuresis, dysphoria
Orphanin FQ (nociceptin)	NOP	Orphan	Nucleus raphe magnus, spinal cord, afferent neurons	Opioid tolerance, anxiety, depression, increased appetite

It is thought that opioids used clinically act through the MOP.
(IUPHAR = International Union of Basic and Clinical Pharmacology)

8.9 Commonly used opioids

Opioid	Typical starting dose	Route	Oral morphine equivalent	Comments
Morphine	10 mg	Oral	10 mg	Most widely used
Codeine	30–60 mg; max 240 mg/24 hr	Oral	3–6 mg	Metabolised to morphine by *CYP2D6* enzyme
Dihydrocodeine	60 mg; max 240 mg/24 hr	Oral	6 mg	Metabolised to morphine by *CYP2D6* enzyme
Tramadol	100 mg; max 400 mg/24 hr	Oral	10 mg	Metabolised to o-desmethyl tramadol by *CYP2D6* enzyme
Oxycodone	6.6 mg	Oral	10 mg	More predictable bioavailability than morphine
Buprenorphine	5 µg/hr	Transdermal	12 mg/day	Patch change usually every 7 days (frequency of change dependent on manufacturer and dose); advantages in impaired renal function
Fentanyl	12 µg/hr	Transdermal	30 mg/day	Use with care in opioid-naïve patients; patch change usually every 72 hrs
Tapentadol	50 mg; max 600 mg/24 hr	Oral	20 mg	Use with care in opioid-naïve patients
Hydromorphone	2 mg	Oral	10 mg	Semi-synthetic; hepatic metabolism
Morphine	3 mg	Subcutaneous, intramuscular, intravenous	10 mg	Mainly used for acute pain or palliative care

long-term use are needed. Additionally, there is increasing concern about potential harm from long-term use. This includes addiction, dependence, opioid-induced hyperalgesia, endocrine dysfunction, fracture risk (especially in older people), overdose and cardiovascular events, with many of these adverse effects being dose-related. Doses of more than 50 mg morphine equivalents per day may be detrimental, with an increased in harm at doses of >90 mg morphine equivalents per day. National and international guidelines have changed to reflect this, with most only recommending short- to medium-term use of opioids in carefully selected patients, as part of a holistic management plan. Regular review is essential to assess ongoing benefit, and any opioid trial should have clear goals, and a plan for cessation if these are not reached. A suggested strategy for using strong opioids in chronic pain is shown in Box 8.10.

Psychological therapies

The aims of psychological therapy are to increase coping skills and improve quality of life when facing the challenges of living with chronic pain. There are a range of ways in which psychological therapies can be delivered, including individual one-to-one sessions, group sessions, multidisciplinary pain management programmes, or web-based or telephone-based programmes.

There is good evidence for the use of a cognitive behavioural therapy (CBT)-based approach for chronic pain, delivered either individually or in a group. The overall aim is to reduce negative thoughts and beliefs, and develop positive coping strategies. The interaction between thoughts, behaviours and emotions is explored, and a problem-focused approach is used in therapy delivery.

Relaxation techniques, such as biofeedback and mindfulness meditation, require a degree of stillness and withdrawal, with regular practice required for sustained benefit (see 'Further information'). Acceptance and commitment therapy (ACT) is based on CBT principles but also uses components of mindfulness to improve psychological flexibility in the context of living with chronic pain.

Stimulation therapies

These range from minimally invasive procedures like acupuncture and transcutaneous electrical nerve stimulation (TENS) to more invasive techniques such as spinal cord stimulation.

8.10 Use of opioids in chronic pain: Always use as part of a holistic management plan

Step	Factors to take into account	Comment
1. Assess suitability for opioids	Type of pain	Neuropathic pain and chronic widespread pain less likely to respond
	Likelihood of dependence	Increased risk in those with history of alcohol and substance misuse
	Co-morbidity	Avoid use in conditions where adverse effects more likely: Chronic obstructive pulmonary disease Chronic liver disease Chronic kidney disease
2. Discuss with patient	Discuss potential benefits	Improvement in pain Improvement in function
	Discuss adverse effects	Nausea Constipation Drowsiness
	Establish treatment goal	Improvement in function
3. Plan treatment trial	Set timescale	Define duration of treatment Agree frequency of review
	Agree on dose	Aim for lowest effective dose Set upper dose limit
	Agree on stopping rules	Consider stopping if: Treatment goal is not met There is no dose response Tolerance develops rapidly

Acupuncture (Fig. 8.6) has been used successfully in Eastern medicine for centuries. The mechanisms are incompletely understood, although endorphin release may explain, in part, the analgesic effect. Acupuncture is particularly effective in pain related to muscle spasm, with some evidence of short-term benefit for patients with low back pain. Similar mechanisms probably apply to TENS, which is worth considering in many types of chronic pain. Neuromodulation, using implanted electrodes in the epidural space (or, more recently, adjacent to peripheral nerves), has been shown to be an effective option for neuropathic pain, including failed back surgery syndrome and chronic regional pain syndrome (see below). Specialist assessment and ongoing support is necessary, as there are many potential complications, including infection, malfunction and battery failure. The likelihood of success is increased when this technique is used within the context of multidisciplinary assessment and management.

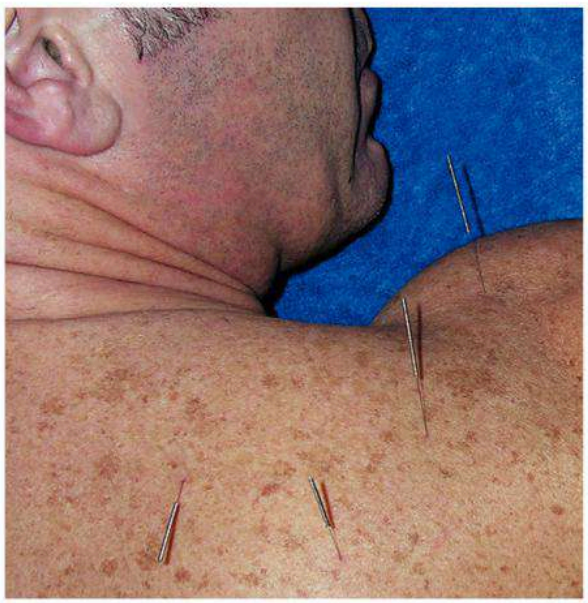


Fig. 8.6 Acupuncture.

Complementary and alternative therapies

Complementary techniques, such as herbal medicines, vitamins, homeopathy and reflexology, have been used for the treatment of chronic pain but with little evidence of efficacy. It should be noted that herbal medications may interact with conventional drugs, causing adverse effects as the result of drug–drug interactions. St John's wort (*Hypericum perforatum*) interacts with many drugs, including many antidepressants used in chronic pain, with increased serotonergic effects. Grapefruit may also increase the risk of serotonergic effects with some antidepressants. *Ginkgo biloba* may interact with paracetamol to increase bleeding time.

Nerve blocks and nerve ablation

The use of specialist nerve blocks and nerve ablation therapy can be considered for pain that is unresponsive to less invasive approaches. If these are being considered, they should form part of a multidisciplinary management plan, with the aim of restoring function and reducing pain. Local anaesthetic with or without depot glucocorticoid (non-particulate for neuraxial administration) can be effective in some circumstances. Examples include occipital nerve blocks for migraine or cervicogenic headache and trigger point injections for myofascial pain. If there is limited compression of a spinal nerve root, the nerve root injections into the epidural space may help settle symptoms and avoid the need for surgical intervention. Neurodestructive procedures can also be employed for intractable pain but are rarely used outside the palliative care setting.

Chronic pain syndromes

Chronic pain is a feature of several recognised syndromes, which are discussed in more detail below.

Neuropathic pain

Neuropathic pain is defined as 'pain associated with a lesion or disease of the somatosensory nervous system'. Neuropathic pain may be acute, such as in sciatica, which occurs as the result of a prolapsed disc, but is most problematic when it becomes chronic. Neuropathic pain causes major morbidity; in a recent study, 17% of those affected rated their

8.15 Adjuvant analgesics in cancer pain

Drug	Example	Indications	Side-effects*
NSAIDs	Diclofenac	Bone metastases, soft tissue infiltration, liver pain, inflammatory pain	Gastric irritation and bleeding, fluid retention, headache Caution in renal impairment
Glucocorticoids	Dexamethasone 8–16 mg per day, titrated to lowest dose that controls pain	Raised intracranial pressure, nerve compression, soft tissue infiltration, liver pain	Gastric irritation if used together with NSAID, fluid retention, proximal muscle myopathy, delirium, Cushingoid appearance, candidiasis, hyperglycaemia
Anticonvulsants	Evidence strongest for: Duloxetine Gabapentin Pregabalin	Neuropathic pain of any aetiology	Mild sedation, tremor, delirium Exacerbation of opioid-related side-effects
Tricyclic antidepressants	Amitriptyline Nortriptyline (less sedative)	Neuropathic pain of any aetiology	Sedation, dizziness, delirium, dry mouth, constipation, urinary retention Avoid in cardiac disease Exacerbation of opioid-related side-effects
NMDA receptor blockers	Ketamine	Severe neuropathic pain (only under specialist supervision)	Delirium, anxiety, agitation, hypertension

*In old age, all drugs can cause delirium.
(NMDA = *N*-methyl-D-aspartate; NSAIDs = non-steroidal anti-inflammatory drugs)

particularly where life expectancy is more than 3 months. Coeliac plexus blocks can be helpful for visceral pain, such as in pancreatic cancer. Lateral cordotomy to disrupt the spinothalamic tracts (either open or percutaneous) may be considered for unilateral chest wall pain, such as may occur in mesothelioma, where life expectancy is limited.

Radiotherapy

Radiotherapy is the treatment of choice for pain from bone metastases (see Box 8.13) and can also be considered for metastatic involvement at other sites. All patients with pain secondary to bone metastases should be considered for palliative radiotherapy, which can usually be given in a single dose. Some patients experience a transient flare of pain after radiotherapy and this can be managed by 24–48 hours of dexamethasone (4–8 mg once in the morning).

Physiotherapy

Physiotherapy has a key role in the multidisciplinary approach to a wide spectrum of cancer-related symptoms, including the prevention and management of pain, muscle spasm, reduced mobility, muscle wasting and lymphoedema. Rehabilitation in palliative care has expanded and now includes pre-habilitation, which involves the use of proactive focused exercise to maintain muscle mass during cancer chemotherapy and in other chronic conditions such as COPD.

Psychological techniques

As with chronic pain, there is increasing use of psychological techniques in cancer pain management, which train the patient to use coping strategies and behavioural techniques. Other issues related to the specific experience of a cancer diagnosis and cancer treatment may be complex, and individual therapy in addition to group-based approaches can be helpful.

Stimulation therapies

Acupuncture and TENS are low-risk stimulation therapies that may be useful in palliative care for management of pain and nausea. Both are particularly useful for secondary muscle spasm and TENS is increasingly used for bone pain.

Complementary and alternative therapies

Palliative care patients often seek symptom relief from both complementary and alternative therapies. While the evidence base is poorly developed, individual patients can gain significant benefits from the complementary therapies as outlined earlier in this chapter. It is critically important that patients are encouraged to discuss any alternative medicines they are considering, given the potential interactions with other therapies.

Breathlessness

Breathlessness is one of the most common symptoms in palliative care and is distressing for both patients and carers. Patients with breathlessness should be fully assessed to determine whether there is a reversible cause, such as a pleural effusion, heart failure or bronchospasm; if so, this should be managed in the normal way. If symptoms persist, additional measures may be necessary. There are many potential causes of dyspnoea in cancer patients and in other chronic diseases; apart from direct involvement of the lungs, muscle loss secondary to cachexia, anxiety and fear can all contribute. A cycle of panic and breathlessness, often associated with fear of dying, can be dominant. Exploration of precipitating factors is important and patient education about breathlessness and effective breathing has been shown to be effective. Non-pharmacological approaches that include using a hand-held fan, pacing and following a tailored exercise programme can help. There is no evidence to suggest that oxygen therapy reduces the sensation of breathlessness in advanced cancer any better than cool airflow, and oxygen is indicated only if there is significant hypoxia. Opioids, through both their central and their peripheral action, can palliate breathlessness. Both oral and parenteral opioids are effective and are now licensed for this indication in Australia. A low dose should be used initially and titrated against symptoms, unless opioids are already being prescribed for pain, in which case the existing dose can be increased further. If anxiety is considered to be playing a significant role, a quick-acting benzodiazepine, such as lorazepam (used sublingually for rapid absorption), may also be useful.

Cough

Persistent unproductive cough can be helped by opioids, which have an antitussive effect. Troublesome respiratory secretions can be treated with hyoscine hydrobromide (400–600 µg every 4–8 hours), although dry mouth is a common adverse effect. As an alternative, glycopyrronium can be useful and is given by subcutaneous infusion (0.6–1.2 mg in 24 hours).

vaccination, such as those aged 60 years or over, immunocompromised individuals or those with chronic liver disease. It may also be effective in an outbreak of hepatitis, in a school or nursery, as injection of those at risk prevents secondary spread to families.

There is no role for antiviral drugs in the therapy of HAV infection.

Hepatitis B

The hepatitis B virus (HBV) consists of a core containing DNA and a DNA polymerase enzyme needed for virus replication, surrounded by surface protein (Fig. 24.25). The virus and an excess of its surface protein (known as hepatitis B surface antigen, HBsAg) circulate in the blood. The virus replicates and assembles within hepatocytes but is not directly cytotoxic; hepatocyte damage occurs during immune-mediated clearance of infected hepatocytes.

Hepatitis B is one of the most common causes of chronic liver disease and hepatocellular carcinoma worldwide. Approximately 250 million people have chronic HBV infection. There is large regional variation, with the highest rates seen in the Western Pacific and African regions.

Hepatitis B can cause acute or chronic infection. The risk of developing chronic HBV infection depends on the source and timing of exposure. Infections may occur via a number of routes (Box 24.38). In endemic areas, vertical transmission from mother to child in the perinatal period is the most common cause of infection and carries the highest risk of ongoing chronic infection (90%–95%). In contrast, exposure to HBV at an older age is much more likely to result in an acute hepatitis, with chronic infection developing in less than 5% of adult-acquired HBV.

In perinatal infection, adaptive immune responses to HBV may be absent initially, with apparent immunological tolerance. Although HBV-specific T-cells are detectable in this setting, they are weak and functionally impaired. The mechanisms underlying this are incompletely understood.

Chronic hepatitis can lead to cirrhosis or hepatocellular carcinoma, usually after decades of infection (Fig. 24.26). Chronic HBV infection is a dynamic process that can be divided into five phases (Box 24.39); these are not necessarily strictly sequential, however, and not all patients will go through all phases. Phases are considered as either 'infection' where circulating HBsAg is detectable but is not resulting in any current liver injury, or 'hepatitis' where this is accompanied by liver inflammation and fibrosis.

Investigations

Assessment of hepatitis B infection relies on looking at a combination of viral markers (surface antigen, e-antigen and viral load) and liver markers (ALT and fibrosis markers) to determine the stage of disease.

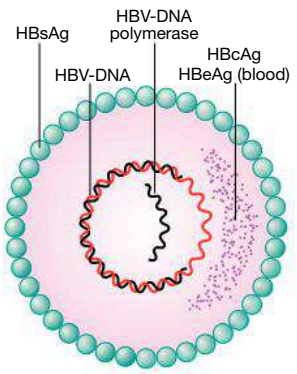


Fig. 24.25 Schematic diagram of the hepatitis B virus. Hepatitis B surface antigen (HBsAg) is a protein that makes up part of the viral envelope. Hepatitis B core antigen (HBcAg) is a protein that makes up the capsid or core part of the virus (found in the liver but not in blood). Hepatitis B e antigen (HBeAg) is part of the HBcAg that can be found in the blood and indicates infectivity. (HBV = hepatitis B virus)

24.38 Risk factors for the acquisition of hepatitis B infection

Vertical transmission

- Hepatitis B surface antigen (HBsAg)-positive mother (especially if e-antigen-positive)

Horizontal transmission

- Sexual transmission
- Intravenous drug use
- Infected unscreened blood products
- Tattoos/acupuncture needles
- Needlestick injury
- Sharing toothbrush/razor
- Close living quarters/playground play as a toddler (may contribute to high rate of horizontal transmission in Africa)

Serology

HBV contains several antigens to which infected persons can make immune responses (Fig. 24.27); these antigens and their antibodies are important in identifying the stage of HBV infection (see Box 24.39 and Box 24.40), in addition to direct assessment of viral load by polymerase chain reaction (PCR) for HBV DNA.

Hepatitis B surface antigen Hepatitis B surface antigen (HBsAg) is the main indicator of active infection, and a negative test for HBsAg makes HBV infection very unlikely. The exception is in acute liver failure from hepatitis B, where the liver damage is mediated by viral clearance and so HBsAg may be negative at presentation, with evidence of recent infection provided by the presence of hepatitis B core (anti-HBc) IgM (see below). In resolving acute HBV infection, antibody to HBsAg (anti-HBs) usually appears after about 3–6 months and persists for many years or perhaps permanently. Anti-HBs implies either a previous infection, in which case anti-HBc (see below) is usually also present, or previous vaccination, in which case anti-HBc is not present.

Hepatitis B core antigen Hepatitis B core antigen (HBcAg) is not found in the blood, but antibody to it (anti-HBc) appears early in the illness and rapidly reaches a high titre, which subsides gradually but then persists. Anti-HBc is initially of IgM type, with IgG antibody appearing later (see Fig. 24.27 and Box 24.40).

Hepatitis B e antigen Hepatitis B e antigen (HBeAg) is part of the core antigen that is detectable in the blood and can be used as an indicator of viral replication. Seroconversion to e antigen (i.e. loss of HBeAg and development of anti-HBe antibody) indicates a partial immune control of the virus and is associated with a significant drop in viral load. This typically occurs after 10–30 years in perinatally acquired infection, but within 3–6 months in adult-acquired acute infections.

Viral load and genotype

HBV-DNA can be measured by PCR in the blood. Viral loads are usually highest (in excess of 10^7 copies/mL) in HBeAg-positive infection, making this the most infectious phase. Once patients undergo HBeAg seroconversion, viral loads drop to less than 10^5 copies/mL in HBeAg-negative infection. Around 15%–20% of patients will subsequently develop further rises in viral load, due to mutations in the pre-core or core promoter region (Fig. 24.28). Such patients are classified as having HBeAg-negative chronic hepatitis.

Measurement of viral load is important in detecting flares of HBeAg-negative hepatitis and in monitoring response to antiviral therapy. Ten HBV genotypes (A–J) can also be identified using PCR. These affect the likelihood of response to pegylated interferon.

Management of acute hepatitis B

Full spontaneous recovery occurs in more than 95% of adults following acute HBV infection. Fulminant liver failure due to acute hepatitis B

Lifestyle advice

Weight loss has a substantial beneficial effect on symptoms if the patient is obese and is probably one of the most effective treatments available for OA of the lower limbs. Strengthening and aerobic exercises also have beneficial effects in OA and should be advised, preferably with reinforcement by a physiotherapist. Quadriceps strengthening exercises are particularly beneficial in knee OA. Shock-absorbing footwear, pacing of activities, use of a walking stick for painful knee or hip OA, and provision of built-up shoes to equalise leg lengths can all improve symptoms.

Non-pharmacological therapy

Acupuncture and transcutaneous electrical nerve stimulation (TENS) can be effective in knee OA. Local physical therapies, such as heat or cold, can sometimes give temporary relief.

Pharmacological therapy

If symptoms do not respond to non-pharmacological measures, paracetamol should be tried. Addition of a topical NSAID and then capsaicin for knee and hand OA can also be helpful. Oral NSAIDs should be considered in patients who remain symptomatic. These drugs are significantly more effective than paracetamol and can be successfully combined with paracetamol or compound analgesics if the pain is severe. Strong opiates may occasionally be required. Antineuropathic drugs, such as amitriptyline, gabapentin and pregabalin, are sometimes used in patients with symptoms that are difficult to control, but the evidence base for their use is poor. Neutralising antibodies to nerve growth factor have been developed and are an effective treatment for pain in OA, but they are not yet licensed for routine clinical use.

Intra-articular injections

Intra-articular glucocorticoid injections are effective in the treatment of knee OA and are also used for symptomatic relief in the treatment of OA at the first CMC joint. The duration of effect is usually short, but trials of serial glucocorticoid injections every 3 months in knee OA have shown efficacy for up to 1 year. Intra-articular injections of hyaluronic acid can help to an extent in knee OA, but the treatment is expensive and the effect short-lived. In the UK they have not been considered to be cost-effective by NICE.

Neutraceuticals

Chondroitin sulphate and glucosamine sulphate have been used alone and in combination for the treatment of knee OA. There is evidence from randomised controlled trials that these agents can improve knee pain to a small extent (3%–5%) compared with placebo.

Surgery

Surgery should be considered for patients with OA whose symptoms and functional impairment impact significantly on their quality of life despite optimal medical therapy and lifestyle advice. Total joint replacement surgery is by far the most common surgical procedure for patients with OA. It can transform the quality of life for people with severe knee or hip OA and is indicated when there is significant structural damage on X-ray. Although surgery should not be undertaken at an early stage during the development of OA, it is important to consider it before functional limitation has become advanced since this may compromise outcome. Patient-specific factors, such as age, gender, smoking and presence of obesity, should not be barriers to referral for joint replacement.

Only a small proportion of patients with OA progress to the extent that total joint replacement is required, but OA is by far the most frequent indication for this. Over 95% of joint replacements continue to function well into the second decade after surgery and most provide life-long, pain-free function. Up to 20% of patients are not satisfied with the outcome, however, and a few experience little or no improvement in pain. Other surgical procedures are performed much less frequently. Resurfacing of the femoral head can be considered for younger patients with hip OA (age <60) as an alternative to total joint replacement. Tibial osteotomy represents an alternative to total joint replacement in younger patients with knee OA and can be effective at improving pain by altering alignment of the lower leg. Cartilage repair is sometimes performed to treat focal cartilage defects resulting from joint injury.

Crystal-induced arthritis

A variety of crystals can deposit in and around joints and structures in the spine and cause an acute inflammatory or even chronic inflammatory arthritis or disease (Box 26.41). Several factors influence crystal formation (Fig. 26.23). There must be sufficient concentration of the chemical components (ionic product), but whether a crystal then forms depends on the balance of tissue factors that promote and inhibit crystal nucleation and growth. The inflammatory potential of crystals resides in their physical irregularity and high negative surface charge, which can induce inflammation and damage cell membranes. Crystals may also cause mechanical damage to tissues and act as wear particles at the joint surface. They can reside in cartilage or tendon for years without causing inflammation or symptoms and it is only when they are released that they trigger inflammation. This may occur spontaneously but can also result from local trauma, rapid changes in the concentration of the components that form crystals, or in association with an acute phase response triggered by intercurrent illness or surgery. In the longer term, a reduction in concentrations of the solutes that form crystals causes dissolution of crystals and remission of the arthritis.

Gout

Gout is the most common inflammatory arthritis in men and in older women though robust epidemiological data on the prevalence of CPPD are lacking. Gout is caused by deposition of monosodium urate monohydrate crystals in and around synovial joints.

Epidemiology

The prevalence of gout is approximately 1%–2%, with a greater than 5:1 male preponderance. Gout has become progressively more common over recent years in affluent societies due to the increased prevalence of obesity and metabolic syndrome, of which hyperuricaemia is

i 26.41 Crystal-associated arthritis and deposition in connective tissue

Crystal	Disease
Common	
Monosodium urate monohydrate	Acute gout
	Chronic tophaceous gout
Calcium pyrophosphate (CPP)	Acute CPP crystal arthritis
	Chronic CPP crystal arthritis
	Chondrocalcinosis
	Axial disease (e.g. crowned dens syndrome)
Basic calcium phosphate (BCP)	Calcific periarthritis
	Calcinosis
Uncommon	
Cholesterol	Chronic effusions in rheumatoid arthritis
Calcium oxalate	Acute arthritis in dialysis patients
Extrinsic crystals/semi-crystalline particles:	
Synthetic crystals	Acute synovitis
Plant thorns/sea urchin spines	Chronic monoarthritis, tenosynovitis

24th Edition

Davidson's Principles and Practice of Medicine

Well over two million medical students, doctors and other health professionals around the globe have owned a copy of ***Davidson's Principles and Practice of Medicine*** since it was first published over 70 years ago. Now in its **24th Edition**, this thoroughly updated textbook describes the pathophysiology and clinical features of the most frequently encountered conditions in the major specialties of adult medicine, and explains how to recognise, investigate, diagnose and manage them. Taking its origins from Sir Stanley Davidson's much-admired lecture notes, ***Davidson's*** has endured because it keeps pace with how modern medicine is taught and provides a wealth of trusted information in an easy-to-read, concise and beautifully illustrated format.

Key features

- **Part 1 'Fundamentals of Medicine'** - provides an account of the principles of genetics, immunology, infectious diseases, population health, oncology and pain management, along with a discussion of the core principles behind clinical decision-making and good prescribing.
- **Part 2 'Emergency and Critical Care Medicine'** - covers medical emergencies in poisoning, envenomation and medicine in austere environments, as well as common presentations in acute medicine and the recognition and management of the critically ill.
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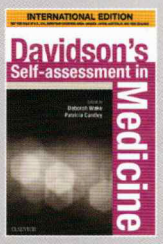

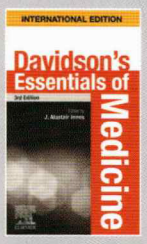

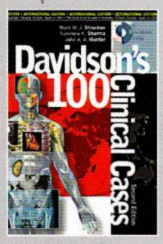

REVIEWS

Beautifully constructed with superb clarity of style – Davidson's *continues to provide for students, doctors and other health professionals a sound basis for the practice of medicine.*

Royal Society of Medicine and Society of Authors Medical Book Awards

This book comes through where others fail: an excellent textbook, easy to read and superb value.

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