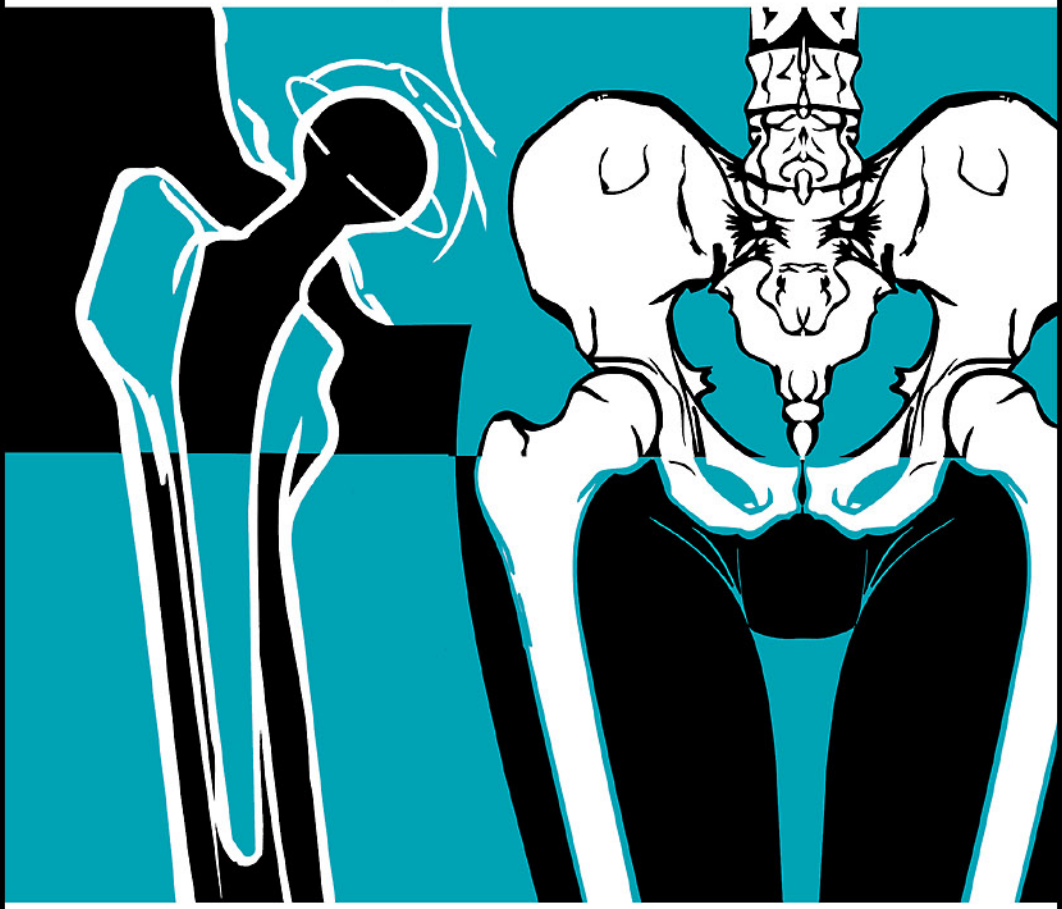
KEY TOPICS IN ORTHOPAEDIC SURGERY

I.M. NUGENT, J.P. IVORY and A.C. ROSS



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KEY TOPICS IN ORTHOPAEDIC SURGERY

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* Topic contributed by Tim Craft (MB, BS, FRCA), Consultant in Anaesthesia and Intensive Care, Royal United Hospital, Bath, UK.

ABBREVIATIONS

ABC	aneurysmal bone cyst
ACJ	acromioclavicular joint
ACL	anterior cruciate ligament
ADI	atlas-dens interval
AFO	ankle-foot orthosis
AIDS	acquired immunodeficiency syndrome
AP	antero-posterior
APB	abductor pollicis brevis
APL	abductor pollicis longus
APPT	activated partial prothrombin time
AVN	avascular necrosis
CD	Cotrel-Dubousset
CDH	congenital dislocation of the hip
CHD	congenital habitual dislocation
CIND	carpal instability, non-dissociative
CMA	congenital metatarsus adductus
CMC	carpometacarpal
CNS	central nervous system
CPPD	calcium pyrophosphate dihydrate
CRP	C-reactive protein
CSA	cross-sectional area
CSF	cerebrospinal fluid
CT	computerized tomography
CTEV	congenital talipes equinovarus
CVT	congenital vertical talus
CXR	chest X-ray
DDH	developmental dysplasia of the hip

DEXA	dual energy X-ray absorptiometry
DIP	distal interphalangeal
DISI	dorsal intercalated segment instability
DPA	double photon absorptiometry
DVT	deep venous thrombosis
ECG	electrocardiogram
ECRB/L	extensor carpi radialis brevis/longus
EDF	elongation-derotation-flexion
EDL	extensor digitorum longus
EHL	extensor hallucis longus
EIP	extensor indicis proprius
ELISA	enzyme-linked immunosorbent assay
EMG	electromyography
EPL	extensor pollicis longus
ESP	erythrocyte sedimentation disorder
ESR	erythrocyte sedimentation rate
EUA	examination under anaesthesia
FBC	full blood count
FCR	flexor carpi radialis
FDL	flexor digitorum longus
FDP	fibrin degradation product
FHL	flexor hallucis longus
FT	femoro-tibial
HIV	human immunodeficiency virus
HR	hallux rigidus
HVA	hallux valgus angle
IMTA	intermetatarsal angle
INR	international normalized ratio
IP	interphalangeal
JCA	juvenile chronic arthritis
JRA	juvenile rheumatoid arthritis
LLI	leg length inequality
LMWH	low molecular weight heparins
MCP	metacarpophalangeal
MFH	malignant fibrous histiocytoma
MRI	magnetic resonance imaging
MTP	metatarsophalangeal

MUA	manipulation under anaesthetic
NPS	nail-patella syndrome
NSAID	non-steroidal anti-inflammatory drug
OA	osteoarthritis
PA	postero-anterior, photon absorptiometry
PB	peroneus brevis
PCI	physiological cost index
PCL	posterior cruciate ligament
PE	pulmonary embolism
PFFD	proximal femoral focal deficiency
PIP	proximal interphalangeal
PL	peroneus longus
PMA	peroneal muscular atrophy
PMMA	polymethylmethacrylate
POP	plaster of Paris
PT	peroneus tertius
PTB	patellar tendon-bearing
PTH	parathyroid hormone
PTS	patellar tendon-supporting
QCT	quantitative computerized tomography
RA	rheumatoid arthritis
RSD	reflex sympathetic dystrophy
RVA	rib-vertebral angle
SAC	space available for the cord
SACH	solid ankle cushioned heel
SD	standard deviation
SL	scapholunate
SLAC	scapholunate advanced collapse
SLR	straight-leg raising
SPA	single photon absorptiometry
STS	soft tissue sarcoma
STT	scaphoid-trapezium-trapezoid
SUFE	slipped upper femoral epiphysis
TENS	transcutaneous nerve stimulation
TFCC	triangular fibro-cartilage complex
TFL	tensor fascia lata
THR	total hip replacement

TKA	total knee arthroplasty
TKR	total knee replacement
TL	triquetrolunate
TOS	thoracic outlet syndrome
TU	tuberculin unit
UHMWPE	ultra-high molecular weight polyethylene
VC	voluntary closing
VISI	volar intercalated segment instability
VMO	vastus medialis obliquus
VO	voluntary opening
VQ	ventilation-perfusion
WBC	white blood cells

PREFACE

Orthopaedic surgery is becoming increasingly sub-specialized. Few senior registrars approaching their consultant posts will possess real expertise in more than one or two sub-specialities in addition to a good basic grounding in general orthopaedic practice. Nevertheless, a high standard of general orthopaedic knowledge is expected and sought by the Royal Colleges in the final specialist examination.

This book has been written as a series of short articles which attempt to summarize current opinion on 100 'Key Topics' in orthopaedic surgery. Its principal aim is to help registrars and senior registrars in their revision for the FRCS (Part III) examination. We have, therefore, been dogmatic rather than discursive in areas of controversy and have deliberately tried to steer an uncontroversial middle course through those topics which most lend themselves to heated argument. We have kept references to a minimum in the knowledge that the weeks leading up to an examination are a time for consolidation rather than for the mastery of new facts and opinions. We hope that our preference for a prose style will find favour at a time when synopses and lists appear to be in the ascendant.

We thank Dr T.M.Craft for contributing the 'Anaesthesia' topic, all those at BIOS who have waited patiently for our manuscript, and our wives and families for their tolerance and support during the writing of this book.

I.M.Nugent
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ACUTE CERVICAL DISC DISEASE

Cervical disc protrusions are described as ‘hard’ or ‘soft’. A ‘soft’ disc protrusion is the result of prolapse of nuclear material through a tear in the annulus fibrosus. It is comparable to an acute lumbar disc protrusion and its aetiology is thought to be the same. A ‘hard’ disc is an osteophyte or osteophytic bar and may be part of generalized degenerative disc disease of the cervical spine. ‘Soft’ discs occur at a single level; ‘hard’ discs may be present at several levels. ‘Hard’ discs are dealt with more fully in the section on degenerative disease of the cervical spine.

Acute prolapse of a cervical intervertebral disc (‘soft’ disc protrusion) occurs most commonly in the fourth decade of life and affects more men than women (1. 4:1). It is associated with heavy lifting, cigarette smoking and board diving. The levels most commonly affected are C6/7 (70%) and C5/6 (24%) where the bending moments and weight carried are maximal. Mixter and Barr (1934) included four cases of acute cervical disc protrusion in their classical description of prolapsed spinal intervertebral discs. Patients with cervical disc disease are more likely to have lumbar disc disease.

Clinical problems

1. Neck and shoulder pain may be the only sign of an acute cervical disc prolapse. The pain is usually felt to one side of the midline. It is accompanied by stiffness of the neck and spasm of the paraspinal muscles, and may radiate to the shoulder or to the medial border of the scapula. It may be unilateral or bilateral. The pain probably arises from distortion of the annulus of the disc or from irritation of the surrounding structures.

2. Nerve root compression is usually unilateral. It is characterized by pain in the arm and may be accompanied by numbness, tingling and weakness in the distribution of the affected root. It is often exacerbated by movement of the neck, and by coughing and sneezing. It should be distinguished from other lesions of the brachial plexus and peripheral nerves, particularly Parsonage-Turner syndrome and thoracic outlet syndrome.

3. Myelopathy may be of sudden onset. The corticospinal tracts are commonly affected causing increased tone, hyperreflexia, clonus and upgoing plantar

(Babinski) responses. There may also be weakness and loss of proprioception in the lower limbs. If the disc protrusion is high in the cervical spine the arms may also be affected. The acute onset of a myelopathy is a surgical emergency. The level of the lesion should be confirmed by MRI, CT or myelography and the cervical cord decompressed immediately from the front. Myelopathy should be distinguished from multiple sclerosis, amyotrophic lateral sclerosis, posterolateral sclerosis, syringomyelia and spinal cord tumour.

Management

1. Investigation. An accurate history and examination should be followed by *plain radiographs.* The chief purpose of these is to exclude other causes of neck and arm pain. The normal cervical lordosis is frequently lost. Signs of disc space narrowing with anterior or posterior osteophytes may indicate degenerative disease but do not help in the localization of an acute disc protrusion. *MRI* is now the investigation of choice and has, in most cases, displaced cervical myelography and CT. It is indicated if the patient's symptoms fail to settle and the diagnosis is in doubt, or when surgery is being planned, in order to confirm the level (or levels) affected. *Cervical discography and root blocks* have no place in routine management.

2. Non-operative treatment. Analgesics, non-steroidal anti-inflammatory drugs (NSAIDs), rest and gentle physiotherapy are all helpful in the acute phase. A soft cervical collar may be of benefit if the patient is mobile. *Exercises* may be introduced once the acute pain has settled. Resisted neck exercises are beneficial as are general neck and shoulder strengthening manoeuvres. *Halter traction* in the position of comfort may relieve pain and spasm when the acute pain is severe. No more than 10 Ib of traction need be applied. Effective skin care around the lower jaw is essential and undue strain should not be placed on the temporo-mandibular joints.

3. Operative treatment is indicated if conservative treatment has failed to control severe pain or if there is an increasing neurological deficit or myelopathy. Posterolateral soft discs should be removed through a posterior fenestration. Hard discs or central soft discs should be removed through an anterior approach. The question of whether to fuse the affected level after anterior discectomy is controversial, as is the side from which the procedure is undertaken. For a right-handed surgeon, an approach through the right side of the neck is technically easier, but runs the risk of damaging the recurrent laryngeal nerve. Anterior fusion with grafting allows the disc height to be restored. After a stable fusion, most posterior marginal osteophyte will be resorbed.

Anterior discectomy and interbody fusion is indicated for midline hard and soft disc protrusions. It may also be used for lateral protrusions with less postoperative morbidity but a slightly increased risk of intra-operative complications. The recurrence rate is also slightly higher from this route. Techniques have been described by Bailey and Badgley, Smith-Robinson,

Cloward and Simmons and Bhalla. They vary principally in the method of preparing and siting the graft. Each gives good to excellent results in between 82% (Cloward) and 100% (Simmons and Bhalla).

Anterior discectomy without interbody fusion was first described by Hirsch in 1958. In 1971, Robertson compared this method with Cloward fusion and found it to be superior. However, current opinion tends to favour grafting using either the Smith-Robinson technique or the 'Keystone graft' method of Simmonds and Bhalla. There are a number of potential problems. Because of the absence of graft, disc space collapse almost always occurs with some angulation of the vertebral bodies. It is therefore necessary to remove all posterior osteophyte. The decision whether to resect the posterior longitudinal ligament to prevent it from buckling backwards into the canal is controversial, but if it is carried out there can be bleeding from the cut edges of the ligament, producing extradural haematoma. The long-term results of this operation are unknown, but it does appear to be inappropriate from an anatomical and biomechanical standpoint.

Posterior hemilaminectomy/foraminotomy and discectomy is indicated for patients with soft lateral disc protrusions. The results are excellent in over 90%. Persistent arm pain may be the result of missed fragments and should be re-investigated early and re-explored if necessary. Minor sensory changes may persist for up to 18 months. Causalgia occurs occasionally.

Complications

1. Of anterior decompression. Damage can occur to the carotid vessels, recurrent laryngeal nerve, oesophagus and larynx, thoracic duct, cervical sympathetic chain, vertebral arteries, spinal nerve roots and cervical cord. These may be minimized by a thorough knowledge of the anatomy of the region, gentle retraction and an adequate exposure with meticulous haemostasis.

2. Of anterior fusion. Anterior grafts may dislocate if insufficiently countersunk between the vertebral bodies. They may collapse with recurrence of symptoms if less than 6 mm in thickness. Non-union occurs in 10%, but symptoms may still have been relieved. The non-union rate is increased if more than one level is fused. Grafts which are too deep from front to back may compress the anterior cord and cause a myelopathy.

3. Of posterior discectomy. Damage may occur to the nerve roots, spinal cord and vertebral arteries. Recurrence of a soft disc protrusion occurs in 1% compared to 2–3% when excised from an anterior approach.

4. Operations on the wrong level may be avoided by meticulous identification of the level by pre- and intra-operative imaging, either by fluoroscopy or, better still, with plain films.

5. Other complications have been noted in the main text.

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Related topics of interest

Congenital neck anomalies (p. 98)
Degenerative disease of the cervical spine (p. 113)
Rheumatoid neck (p. 254)
Shoulder pain (p. 281)
Spinal fusion (p. 299)
Spinal infection (p. 302)
Spinal tumours (p. 310)
Thoracic outlet syndrome and cervical rib (p. 324)

ACUTE LUMBAR DISC DISEASE

Acute prolapse of a lumbar intervertebral disc commonly affects patients in the third or fourth decades of life. The gelatinous nucleus pulposus herniates through the relatively weak postero-lateral portion of the annulus fibrosus and posterior longitudinal ligament. The commonest levels affected are L4/5 and L5/S1. Infrequently, the L3/4 level is affected. Protrusion of the L5/S1 disc usually causes compression of the first sacral nerve root. Similarly, protrusion of the L4/5 disc tends to compress the fifth lumbar root. However, the size and site of the disc protrusion and the local configuration of the nerve root may all obscure this pattern. A large disc protrusion at L5/S1 can affect both the S1 and L5 roots, and a'far lateral' disc may compress the nerve root exiting the canal at the same level (e.g. an L4/5 disc may compress the L4 root).

Clinical problems

1. Acute postero-lateral disc prolapse. Acute disc prolapse is usually preceded by episodic low back pain with or without irritation of the nerve root. When the disc ruptures and the nucleus is extruded on to the nerve root, the patient's symptoms change to those of 'sciatica', often with loss of the back pain. There is pain, numbness and tingling in all or part of the distribution of the compressed nerve. The pain and tingling are usually exacerbated by coughing, sneezing, lifting or straining.

Examination of the back may reveal a loss of the normal lumbar lordosis and the presence of a 'sciatic scoliosis', a list or side-shift of the trunk on the pelvis. There may be accompanying paravertebral muscle spasm. The straight-leg raising (SLR) test is considered positive if it reproduces the patient's leg pain. A limited SLR provoking back pain does not necessarily indicate nerve root compression. Dorsiflexion of the foot and the 'bowstring' test may be used in conjunction with the SLR. Provocation of the leg pain by elevation of the opposite leg ('crossed leg pain') is virtually pathognomonic of a prolapsed intervertebral disc.

Compression of the L5 nerve root by an L4/5 disc causes pain in the buttock, lateral thigh and antero-lateral calf, radiating across the dorsum of the foot to the hallux and second toe with numbness and tingling in the same distribution. There

may be weakness of extensor hallucis longus (L5, S1) and sometimes tibialis anterior (L4, L5) but the ankle reflex (S1, S2) is usually preserved.

Compression of the Sl nerve root by an L5/S1 disc causes pain in the lateral aspect of the thigh and calf which radiates to the lateral aspect of the foot and the lateral three toes. Plantarflexion (S1, S2) may be weak and the ankle jerk (S1, S2) is often diminished or absent. The toe extensors and peronei (L5, S1) may be weak whether the L5 or Sl root is involved and are not reliable indicators of level.

Compression of the L4 nerve root by an L3/4 disc causes pain in the postero-lateral aspect of the thigh and the antero-medial part of the leg. The quadriceps femoris (L2, L3, L4) may be weak and the knee may feel unstable to the patient. The knee jerk (L2, L3, L4) may be absent.

2. Acute massive 'central' disc prolapse. This is rare but constitutes a surgical emergency. The onset is sudden following herniation of a massive amount of disc material into the spinal canal. Pain is felt in the back and radiates down the backs of both thighs and legs with numbness in the same distribution, often extending to the soles of the feet and perineum (saddle anaesthesia). The ankles are weak or paralysed, as are the sphincters of bladder and bowel. The ankle jerks are absent.

Management

1. Non-surgical. Since 80–90% of all postero-lateral disc protrusions resolve spontaneously in 3–6 months, most should be treated conservatively. Bed rest in the acute phase supplemented with NSAIDS and rescue analgesia will allow most patients to start to mobilize gently for short periods of time after a few days. Sitting (flexion) is actively discouraged at this stage. If patients cannot rest completely at home or if their pain cannot be controlled, hospital admission and opiate analgesia are appropriate. Bed rest for prolonged periods and muscle relaxants are of no value and may be harmful.

2. Investigations. Investigations are used to confirm a diagnosis which has been made clinically. Only if all the clinical signs, symptoms and investigations localize a disc protrusion to a specific side and level is the disc protrusion likely to be the cause of that patient's pain. Failure to appreciate this fact is responsible for much unnecessary and inappropriate surgery.

Investigation is indicated if the patient has failed to respond to conservative treatment, if there are progressive neurological signs, or if there is any suggestion of sphincter disturbance. *Plain radiography* is of no direct help in making the diagnosis, but can exclude malignancy, infection and tumour. It also confirms or refutes the normal pattern of segmentation of the lumbar spine. *Myelography* using metrizamide or iohexol is a very sensitive method of identifying neural compression. It was certainly more accurate than early *CT* (1984), but has the disadvantage of being invasive and associated with appreciable side effects. The relative accuracy of CT, myelography and *MRI* is not currently known as there

have been significant advances in each over the last few years. Given that MRI is non-invasive and does not use ionizing radiation, it has to be the investigation of choice to confirm acute lumbar disc prolapse. It should be borne in mind, however, that there is a false-positive rate (overdiagnosis) of up to 30%.

3. Surgical Chemonucleolysis involves the percutaneous injection of chymopapain into the nucleus pulposus. It should only be considered for protrusions which are shown to be deep to the posterior longitudinal ligament on MRI. Moderate improvement may be seen in up to 75% of patients. It carries a 0.04% risk of death due to anaphylaxis. There is concern about the number of patients who have severe residual low back pain. *Percutaneous nucleotomy* may be indicated in the same group of patients but avoids the use of chymopapain with its attendant risks. Satisfactory results have been claimed in about 70% of patients. *Microdiscectomy* and *minidiscectomy* yield similar results, with 90–95% good or excellent results. Both should be preceded by radiographic localization of the affected level. *Microdiscectomy* uses a shorter incision with less attendant soft tissue dissection. Minimal bone excision is required and the extradural fat is preserved. It is of greatest value when pre-operative investigations indicate a simple fresh postero-lateral disc prolapse. In these cases, the procedure should be uncomplicated and excellent results are obtained. *Minidiscectomy* uses a slightly longer incision but allows exploration of the whole length of the nerve root if there are doubts about sequestered fragments or non-discal compression of the root. No adequate long-term comparative study exists between the two procedures. *Laminectomy* can only be justified for acute central disc prolapse.

Complications

- *Neural damage* occurs in 0.8%.
- *Infection* occurs in less than 1% of simple discectomies.
- *Vascular damage* occurs to the vessels lying in front of the affected disc space in 0.05%.
- *Dural injury* occurs in 2.5–5% of cases.
- *Recurrent prolapse* at the same level occurs in 5–7% of cases.
- *Failed disc surgery* occurs in 5–10% of patients, who are not relieved of their leg symptoms. However, up to 30% are not relieved of their attendant backache. Patients should be warned pre-operatively that only 70% are relieved of their backache, and these for reasons which are not wholly understood.

Further reading

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Related topics of interest

Degenerative disease of the lumbar spine (p. 116)
Low back pain (p. 181)
Spinal stenosis (p. 306)
Spondylolysis and spondylolisthesis (p. 317)

AIDS

Acquired immunodeficiency syndrome (AIDS) is caused by the human immunodeficiency virus (HIV). By 1991, there were over 5000 cases of AIDS and 17000 cases of HIV infection. HIV is a retrovirus which infects host cells and uses its reverse transcriptase to synthesize DNA copies of its RNA. Once the DNA copy is incorporated into the host cell it causes further synthesis of the virus. HIV can be spread by unprotected intercourse with an infected person, by inoculation by blood, tissue or body fluids, and by parturition or breastfeeding. Seroconversion takes anything from 3 weeks to 6 months. Infection with HIV is characterized by an acute viral infection, an asymptomatic phase, a generalized lymphadenopathy and fmally by wasting, herpes zoster infection and neoplasms. AIDS is characterized by atypical pneumocystic chest infections, Kaposi sarcoma, candidiasis and coccidiomycotic infections.

Clinical problems

1. Diagnosis. The high risk groups are homosexuals, intravenous drug abusers, trauma cases, haemophiliacs and people from West Africa. Antibody testing (the ELISA or Western blot tests) will be positive if the HIV infection has raised serum antibodies. There may be an interval of up to 6 months between infection and seroconversion. An antigen test is available, but it is more expensive and not yet widely used.

2. Transmission from patient to orthopaedic surgeon. HIV may be spread in the operating theatre by sharps injuries, corneal contact or mucous membrane contact from aerosols. HIV probably cannot spread by direct contact of contaminated blood and intact skin. Hollow needles appear to increase the risk of transmission more than solid needles.

3. Transmission from surgeon to patient. There is only one defmite case of a dentist having transmitted the HIV virus to patients. The risk is small.

4. Transmission in blood and tissue products. Biood donors are screened for the HIV virus and all donations are tested using the antibody test. The risk of transmission is estimated to be one in a million transfusions. There are defmite recorded cases of transmission by bone and tendon allografts.

5. Orthopaedic manifestations. Atypical infections in bone may mimic tumours.

Management

1. Defmitely infected patient. If possible, they should be treated like uninfected patients, but the presence of open wounds or discharges of body fluids may require that the patient is nursed in a side room. In the operating theatre, all unnecessary equipment should be removed. Surgical staff should wear double gloves, glasses or a visor, a plastic apron and disposable gown, and plastic overshoes. The patient is towelled with disposable drapes. There should be the minimum use of sharps, meticulous haemostasis, no-touch technique and use of skin staples. Spillages should be cleaned with 1% hypochlorite, and after surgery, surfaces should be cleaned with 0.1% hypochlorite. Patients should be recovered in theatre. Shoes are left in the theatre for cleaning. Arthroscopes must be sterilized in glutaraldehyde for at least 20 minutes, but, because of its toxicity, many arthroscopes are autoclaved and the fibreoptic system is excluded by sterile plastic sheaths.

2. Suspected infected patient. All patients undergoing surgery should be screened. Before elective surgery, those at high risk should be offered counselling and testing. In emergency, and in cases where the patient refuses to be tested, the surgeon in charge of the case must assume responsibility for the precautions required. If in doubt, the patient is treated as infected.

3. Injury. If a member of staff is injured during an infected case, then he or she should have a blood sample taken for storage and later testing if required. The patient may be tested, ideally after counselling. The Royal College of Surgeons has stated that in the case of injury, the surgeon has the right to test a patient who has not previously given consent, but the surgeon must consider the need for this in the light of possible action should a complaint be lodged.

4. Infected surgeon. Infected surgeons must receive counselling and cease invasive surgery.

5. Blood products. All blood is screened by antibody testing but not by antigen testing. Blood products such as Factor VIII are heat-treated to inactivate the virus.

6. Human tissue. All donors must be screened. All live donors must be tested by antibody testing at the time of donation and after 3 and preferably 6 months. Cadaver donors must be screened, have an antibody test and if possible an antigen test. Secondary sterilization may be by heat, ethylene oxide or irradiation.

Further reading

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Related topic of interest

Allografts and bone banking (p. 12)

ALLOGRAFTS AND BONE BANKING

An allograft is tissue transplanted from one member of a species to another member of the same species. The most common orthopaedic allografts are of bone, articular cartilage, tendon and meniscus.

While autograft bone is the best material to replace bone lost after tumour resection, revision arthroplasty and trauma, it is in limited supply and its removal may be accompanied by pain or other complications at the donor site. Allograft bone is in unlimited supply but induces less new bone formation from surrounding tissues (osteoinduction), is mechanically inferior and may provoke an immunological reaction. Transplantation also carries the risk of transmitting an infectious disease and the transplant may itself be a focus for infection.

Bone banking

1. Donors. Bone may be retrieved from patients undergoing total hip arthroplasty (live donors) or from cadavers. Femoral heads are a suitable source of cancellous bone whereas cadaveric bone is best used to supply large structural allografts.

The age range from which cadaveric donors should be selected is 18–45 years. The lower age limit ensures skeletal maturity and avoids problems with open epiphyses. The upper age limit is imposed to exclude donors with osteoporosis or cartilage degradation.

2. Consent. Informed consent is obtained from live donors undergoing total hip replacement, and from the next of kin in cases of cadaver donation. This includes consent to obtaining blood for HIV testing.

3. Procurement. Femoral heads are retrieved in the operating theatre under sterile conditions. Cadaveric bone may be retrieved under sterile conditions and stored directly, or under non-sterile conditions if secondary sterilization is planned. Bone may be removed up to 24 hours after death provided the cadaver has been kept cold in the mortuary. Grafts are cleaned of soft tissue, measured, individually wrapped and labelled. A swab soaked in dimethylsulphoxide may help preserve the articular cartilage.

4. Screening. The history may exclude a number of potential donors with transmissible disease. Blood tests include HIV, hepatitis B and C, VDRL and cytomegalovirus. Live donors need a second HIV blood test at least 6 months

after procurement to detect seroconversion of previously negative donors. This will be replaced by the polymerase chain reaction blood test for HIV antigen when it becomes more readily available. Swabs are taken from all grafts and cultured for organisms.

5. Storage. Allografts may be stored at -20°C for up to 6 months or at -80°C for up to 5 years. Sterilized and freeze-dried allografts may be stored at room temperature indefmitely.

6. Sterilization. Grafts may be retrieved sterile and stored in a freezer (fresh-frozen): these require no further sterilization. Contaminated grafts or grafts not procured under sterile conditions require secondary sterilization. Irradiation is the commonest method employed. A dose of 25 kGy will kill *Bacillus subtilis* (the reference organism for sterilization) and HIV. Other sterilization methods use ethylene oxide or heat but these may further reduce the osteoinductive capacity of the allograft.

7. Re-implantation. The graft is allowed to thaw and is soaked in antibiotic solution. Prophylactic antibiotics should be given to the recipient. At re-implantation the allograft is swabbed again to ensure sterility and to act as a guide to antibiotic treatment should infection ensue. Rhesus-positive grafts should not be given to rhesus-negative women of child bearing age.

8. Documentation. Strict documentation is essential. All grafts must be clearly labelled. Blood and microbiology results must be reviewed before release of the graft for transplantation.

Complications

1. Infection. The prevalence of clinical infection in recipients of femoral head allografts should be negligible. Often, however, the complex nature of the surgery itself carries a high risk of infection. Infection complicates 10–15% of procedures in which large allografts are used. The allograft frequently has to be removed as a result and amputation may be required.

2. Fracture. Sixteen percent of large grafts fracture, on average, 28 months after implantation. This follows revascularization of the graft by host vessels which cause bone resorption. Intramedullary fixation may be protective. If a graft shows extensive resorption it should be removed and replaced. Articular fractures may be treated by joint replacement or internal fixation. Intercalary grafts can be grafted at the fracture site with autogenous bone and stabilized by internal fixation.

3. Non-union. Union of large allografts to host bone takes between 4 and 14 months. The incidence of non-union is 12% and is less for grafts with a large cancellous surface at the junction. Treatment is by autografting the junction.

4. Rejection. Immunological responses are detectable but rarely cause a clinical problem. Part of the reason for this is that freezing of bone diminishes its immunogenicity. Early rapid resorption or rejection of an allograft occurs in less

than 1% of cases. Patients who have fresh osteoarticular allografts appear not to reject the graft even without tissue typing or immunosuppression.

5. Failure of osteoarticular allografts. This occurs frequently in fresh-frozen allografts due to loss of viable chondrocytes. The success rates for fresh-frozen allografts used to fill post-traumatic defects are 75% at 5 years and 64% at 10 years. The graft must not be mechanically overloaded. The success rate is higher in unipolar grafts. Grafting for osteoarthritis and osteonecrosis has an unfavourable prognosis.

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Related topics of interest

Bone tumours—malignant (p. 68)
Hip arthroplasty (p. 150)
Spinal fusion (p. 299)

AMPUTATIONS AND PROSTHETICS

Between 5000 and 6000 new amputees are seen in limb-fitting centres in England each year. The commonest reason for amputation is vascular insufficiency (54%) followed by diabetes (20%), trauma (11%), embolism (4%) and malignancy (4%). The overall male to female ratio is 2.5:1, but this tends to equalize as the patients become older. Upper limb amputations usually occur in younger patients as a result of trauma.

Problems

1. The foot and ankle. Toe and ray amputations do not usually cause problems except in the case of the big toe and first ray. Trans-metatarsal, tarsometatarsal (Lisfranc) and midtarsal (Chopart) amputations may be indicated for trauma or infection but are poor choices in vascular disease. Talectomy with calcaneo-tibial arthrodesis (Boyd) and through-ankle (Symes) amputation avoid the equinus that occurs after amputations through the foot.

2. Below knee. A minimum of 9–10 cm should be preserved below the knee joint to allow satisfactory fitting. Either skew or long posterior (Burgess) flaps are used. Seventy-five percent of patients will walk with a prosthesis but with a 10–40% increase in energy expenditure. The overall mortality is 9%.

3. Through knee. The advantages of better socket suspension through retaining the condyles and end-load-bearing are probably outweighed by the disadvantages of poor healing and cosmesis except in patients who are not expected to walk again.

4. Above knee. A minimum of 12 cm above the knee joint is required to allow fitting of an artificial knee. A snug myoplasty should be fashioned. In the long term, only 20% are able to walk because of the increased (65%) energy expenditure. The overall mortality is 25%.

5. Pelvic disarticulation and hindquarter. Usually indicated for trauma or malignancy. Prostheses usually only worn by young patients.

6. Upper limb. Wrist disarticulation preserves pronation and supination. Below-elbow amputation should preserve at least 16–18 cm of the forearm below the tip of the olecranon. Above elbow amputations should aim to preserve at least 20–25 cm of the humerus. Equal flaps are normally used. Shoulder

disarticulation and forequarter amputations are only indicated for malignancy or high brachial plexus injuries with vascular damage.

Design

1. Sockets and suspension. A below-knee socket is termed 'hard' if the laminate is in contact with the skin, or 'soft' if it is foam-lined. Total contact casts help prevent oedema of the stump and improve the 'feel' of the limb. Patella tendon-bearing (PTB) sockets permit direct loading and are suspended by a strap above the condyles, while patellar tendon-supporting (PTS) sockets encapsulate the femoral condyles and the patella. The classical above-knee amputation quadrilateral socket is suction or waist-strap suspended and loads the ischial tuberosity. Newer CAD-CAM designs share the load between the gluteal structures and the greater tuberosity. Above-knee designs allow for 10° adduction of the hip to increase abductor strength.

Upper limb sockets are usually oval in shape and suspended by straps. Elbow flexion and control over the terminal device are effected by movements of the shoulder acting via cables.

2. Terminal device. The most popular foot is the solid ankle cushioned heel (SACH). Multi-axis designs allow for inversion and eversion while walking on uneven ground. Energy-storing feet are fitted with a cantilever spring to allow smooth roll-over, but are more expensive. The most effective upper limb terminal device is the split hook which may be either voluntary opening (VO), which is more popular, or voluntary closing (VC). Artificial hands tend to be passive devices used for cosmetic purposes. Mechanical hands are bulky and heavy.

3. Body. The body links the terminal device and the socket. It may be an endoskeleton with a soft exterior, or an exoskeleton with a hard load-bearing shell.

4. Joints. Knee joints for above-knee prostheses are chosen on an individual basis. Alignment and stability are important in design. If the centre of motion is placed behind the centre of gravity, control in stance is improved but flexion is made more difficult. If it is placed in front of the centre of gravity, the joint rotates easily but at the expense of control.

Constant friction joints are durable and useful in children but only allow single speed walking. Variable friction joints increase the resistance to flexion as the knee extends but are not as durable. Stance-phase control acts like a constant friction during swing but freezes in stance.

Polycentric four bar linkage knees allow for different stability in different phases of walking as the centre of rotation changes. Manual locking in extension is useful for weak patients. Fluid control dampens the swing into flexion and allows for a more even gait.

Single-axis elbow hinges are the most commonly used in below-elbow amputees, but in obese patients a polycentric hinge may prevent the soft tissues obstructing flexion. Above-elbow prostheses are fitted with an elbow joint,

which can be locked in a number of positions, and a friction turntable which allows the hand to be positioned in space before the terminal device is used.

Problems

1. Poorfit.

2. Poor alignment. This may produce varus or valgus strain, hyperextension of the knee or instability. A dorsiflexed foot on a below-knee prosthesis can cause increased pressure across the patello-femoral joint.

3. Neuromaformation.

4. Reflex sympathetic dystrophy.

5. Phantom limb. This is present in all amputees to some degree. It disappears more quickly in young patients. The foot tends to 'telescope' to the end of the stump. True phantom pain is not common, but when it occurs it is usually continuous with exacerbations, and may be protracted. Phantom pain should be managed in conjunction with the pain clinic.

Further reading

Symposium on amputations. *Annals of the Royal College of Surgeons,* 1991; **73:**133–78.

Related topics of interest

Bone tumours—malignant (p. 68)

Congenital upper limb abnormalities and deficiencies (p. 106)

ANAESTHESIA

Elective orthopaedic patients tend to be at the extremes of age. They may have multi-system disease and the procedures can have special hazards with anaesthetic implications. The necessary pre-anaesthetic investigation of patients scheduled for elective orthopaedic surgery will vary according to the nature of the proposed surgery and the health of the patient. Some general principles can, however, be stated. Difficulty with intubation, surgery for spinal deformities, prevention of deep venous thrombosis, and local anaesthesia merit further discussion.

Preoperative investigations

1. Blood tests. All patients undergoing intermediate or major surgical procedures should have their haemoglobin, platelets and electrolytes measured. All women who have not yet entered the menopause should also have their haemoglobin measured even for minor procedures under general anaesthesia. Determination of the blood group and, in most centres, cross-matching of blood is required before embarking on major surgery associated with appreciable blood loss. Other investigations should be tailored to the individual patient. For example, those taking regular medication which may alter renal function should have their urea electrolytes and creatinine measured.

2. ECG. A standard 12-lead ECG should be recorded in all patients with a history suggestive of cardiovascular disease and in those over the age of 60. Only 50% of old infarcts will be seen on a standard ECG, however, and arrhythmias are best detected with a 24-hour ECG. Stress testing may be performed using physical exercise in fitter patients and dipyridamole-induced coronary vasodilation in the immobile. Indications for further investigation include ST depression greater than 0.2 mV, early ST depression, little increase in blood pressure or heart rate, and the development of hypotension.

3. Chest X-ray (CXR). A CXR should always be performed in patients with a history suggestive of cardiovascular disease. Seventy percent of those with radiological evidence of cardiomegaly have an ejection fraction less than 50%. Evidence of left ventricular failure should also be sought. The yield of positive pulmonary signs from a CXR in an asymptomatic patient is low. Routine CXR in

such patients who have no clinical signs of pulmonary pathology is unwarranted.

Intraoperative problems

1. Difficulty with intubation. Patients with disease of the cervical spine resulting in stiffness, deformity or instability (e.g. ankylosing spondylitis or rheumatoid arthritis (RA)) may be difficult to intubate. Clinical tests to predict this are performed preoperatively, as well as plain radiographs of the head and neck. Controlled flexion and extension views are required to exclude atlanto-axial instability in patients with RA. These patients may also have temporo-mandibular arthritis, limiting jaw opening, and cricoarytenoid arthritis, which may prevent easy passage of a tracheal tube. Specialized fibreoptic-assisted intubation techniques may be required.

2. Spinal deformity surgery. Longstanding kyphoscoliosis results in a restrictive pulmonary defect with abnormal gas exchange. Pulmonary hypertension and right ventricular failure may ensue. Corrective surgery may prevent further cardiorespiratory deterioration but careful pre-operative assessment is essential. Pulmonary function tests and arterial blood gas analysis provide valuable information about the extent of respiratory impairment. Predictions may be made as to the likelihood of the patient being able to maintain adequate gas exchange during surgery. Anterior approaches to the spine requiring one-lung ventilation (OLV) are a particular challenge for the anaesthetist. A double-lumen tracheal tube is used to isolate the lungs and high inspired oxygen tensions are administered to maintain oxygen saturation. Spinal cord function is monitored using evoked potentials or a ‘wake-up test’ once the spine is straightened. Neuromuscular blockade is reversed and anaesthesia lightened until the patient moves his/her feet to command. An intravenous benzodiazepine or induction agent is then given immediately and anaesthesia deepened to permit the completion of the operation. Postoperative recall of the test is unusual.

3. Anaesthetic technique and DVT. The occurrence of DVT following elective total hip replacement performed under general anaesthesia may be as high as 75–80% if preventative measures are not taken. The use of spinal or epidural anaesthesia for total hip replacement or total knee replacement reduces the risk of DVT by about half. The mechanism for this apparent benefit is multifactorial. First, there are changes in lower extremity *blood flow* associated with regional anaesthetic techniques. Vasodilatation occurs following blockade of the sympathetic nervous system and results in an increase in lower extremity arterial inflow, venous emptying, and venous capacity. Second, epidural anaesthesia is known to inhibit the neuroendocrine *stress response* to major surgery. This response includes an increase in blood coagulability and a depletion of plasma fibrinolytic activity. Third, systemic levels of local anaesthetic agents may have

a direct effect on the *endothelium,* resulting in an increased formation of prostacyclin.

General anaesthesia with controlled ventilation, on the other hand, is associated with a reduced velocity of blood flow in the femoral vein. This is a consequence of reduced cardiac output following the induction of anaesthesia and reduced right heart return secondary to the increased intrathoracic pressure resulting from positive pressure ventilation.

Contraindications to spinal and epidural anaesthesia include the administration of thrombolytic therapy within the last 24 hours, known coagulopathies, significant thrombocytopenia and full anticoagulation. There does not appear to be an increased incidence of complications in patients receiving either epidural or subarachnoid anaesthesia who have also been given sub-cutaneous low-dose heparin or antiplatelet therapy.

Local anaesthesia

1. Mode of action. In low concentration, local anaesthetic agents delay, and in higher concentrations completely prevent, the migration of ions across the cell membrane. This stabilizing effect prevents propagation of the action potential not only in nerve cells but in any other cell which is capable of excitation (lignocaine may be used to treat certain cardiac arrhythmias for example).

2. Maximum dosage. The adoption of maximum safe doses in anaesthesia is an attempt to prevent the occurrence of toxic systemic levels. The likelihood of toxicity depends on the site of injection; intravascular injections are most likely to result in toxicity. Perivascular nerve blocks (e.g. the axillary brachial plexus) and infllltration of highly vascular areas (e.g. the subcostal space) also carry a greater risk of toxicity. The addition of adrenaline to lignocaine solutions will help reduce absorption into the blood stream. A 1% solution contains 10 mg/ml. Maximum recommended doses are: lignocaine 3 mg/kg; lignocaine with adrenaline 7 mg/kg; bupivacaine 2 mg/kg.

3. Signs of toxicity. Local anaesthesia should not be performed without immediate access to full resuscitation facilities and skills. The signs of systemic toxicity are not always related to dose and may include:

- *CNS:* stimulation followed by depression, restlessness, peri-oral tingling, tinnitus, tremor, convulsions and respiratory depression;
- *CVS:* hypotension, bradycardia, sweating and acute cardiovascular collapse;
- *Allergic reactions:* bronchospasm, urticaria or angioneurotic oedema.

These are rare. Treatment is supportive: barbiturates or benzodiazepines are used to control convulsions.

Further reading

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Related topics of interest

Kyphosis (p. 170)
Postural correction in ankylosing spondylitis (p. 233)
Rheumatoid neck (p. 254)
Scoliosis—early onset (p. 263)
Scoliosis—late onset (p. 267)
Thromboembolic disease (p. 328)

ANKLE INSTABILITY

Recurrent instability of the ankle usually occurs after repeated acute inversion injuries. The three parts of the lateral collateral ligament are partially or completely torn and heal in a lengthened position, resulting in lateral joint laxity. This commonly affects the anterior talo-fibular and calcaneo-fibular parts, allowing anterior subluxation of the talus in the ankle mortice (anterior draw sign of the ankle). It has been suggested that repeated 'going over on the ankle' may be due to proprioceptor damage in the ligament rather than laxity.

Isolated injuries of the medial collateral ligament are much less common and chronic instability is rare. Medial ligament injuries tend to occur in conjunction with ankle fractures.

There is no reliable information about the incidence of ankle ligament injuries, the incidence of instability or the risk of developing arthritis after ligamentous injury. The risk of developing instability is related to the severity of the initial injury and to the method and duration of treatment.

Clinical problems

Clinical examination in the acute phase is often difficult because of pain and swelling. If clinical or radiological evidence of subluxation is evident, examination under anaesthetic may be indicated.

Non-operative management

1. Diagnostic imaging. Stress radiographs are obtained with the ankle forcibly inverted. Significant instability is present if, on the AP projection, the talus tilts more than 6°. A lateral stress film is also important in order to detect anterior or posterior subluxation. Peroneal stress tenography may be useful in the detection of rupture of the lateral ligament but may eventually be replaced by MRI.

2. Physiotherapy. Minor degrees of laxity without subluxation may be treated with ice, ultrasound and 'wobble-board' exercises which are thought to improve proprioception.

3. Orthoses. Shoes with a lateral float on the heel may be of help. If disability is only related to certain sporting activities, a lateral supporting ankle orthosis

may be worn. Chronic conditions involving weakness, paralysis or arthritis may require a calliper.

Surgical management

Operative repair of acutely torn ligaments may be advisable in athletes. After early immobilization in plaster, mobilization in supportive orthoses is recommended. Adhesions are common after acute injury and may cause continuing disability unless appropriate rehabilitation is undertaken.

Surgical reconstruction may be appropriate for chronic instability with disabling symptoms in the presence of positive stress radiographs.

1. Watson-Jones tenodesis. The peroneus brevis tendon is divided proximally so as to give as long a distal segment as possible. It is then threaded through a drill hole in the fibula from back to front, through a vertical drill hole in the neck of the talus from above to below, and back to the fibula where it is secured. This technique reconstructs both the calcaneo-fibular and the anterior talo-fibular ligaments.

2. Evans procedure. The peroneus brevis tendon is divided proximally above the ankle. It is then re-routed and secured at either end of an oblique tunnel drilled in the fibula. This technique requires less length of tendon and may be used as a back-up procedure if required.

3. Fitton's operation. This procedure uses an isolated graft of the plantaris or peroneus brevis tendon. Its advantage is that it does not bridge the talus. The anterior talo-fibular ligament is reconstructed by passing the graft through four drill-holes: one horizontally through the lower fibula, one vertically through the neck of the talus, one horizontally through the body of the talus and another through the tip of the fibula.

4. Elmslies's operation (Chrisman-Snook modification). The peroneus brevis is split in half longitudinally and is pulled distally under the peroneal retinaculum. It is passed posteriorly through a horizontal hole in the fibula and secured at the entrance to the periosteum with sutures. Another hole is drilled posteroanteriorly through the lateral ridge of the os calcis near the attachment of the calcaneo-fibular ligament. The tendon may be secured here or, if long enough, sutured back on to itself anteriorly.

5. Prosthetic ligaments made of synthetic fibres (e.g. carbon fibre) have been advocated, but none have been universally successful.

The choice of procedure may be selected according to the direction of instability. The Fitton procedure is useful in cases of pure anterior subluxation but, if anterior laxity is combined with significant varus tilt of the talus, the Elmslie- type procedure may be more appropriate. If varus tilt exists without anterior subluxation, the Evans operation will control this at the expense of loss of foot inversion.

Complications

- Recurrent instability may cause degenerative ankle arthritis.
- Surgical reconstruction may achieve stability at the expense of reduced inversion range and eversion power.

Further reading

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Related topics of interest

Arthritis of the ankle (p. 28)
Rheumatoid arthritis (p. 245)

ANTERIOR KNEE PAIN

Anterior knee pain is commonest in adolescent girls and is frequently bilateral. It is usually caused by the increased pressure and stress on the patella associated with patello-femoral instability, patellar tilting or malalignment. It starts insidiously with anteromedial pain exacerbated by climbing stairs, squatting or sitting with the knees bent. The knee frequently gives way and may 'catch' or 'grate'. Examination of the lower limbs may reveal femoral neck anteversion, genu varum or valgum, external rotation of the tibia and squinting patellae. A quadriceps angle (Q) of more than 20° is abnormal. Patellar tracking should be observed from full extension to 30° of flexion as the patella enters the trochlear groove. Patellar tenderness may be elicited and patello-femoral compression is likely to be painful.

Other causes of anterior knee pain include osteoarthritis without malalignment, osteochondral fractures or chondral separations, osteochondritis dissicans, bipartite patellae, inflamed bursae and algodystrophy. Osgood-Schlatter's disease, Sindig-Larsen disease, vastus lateralis strain and patellar tendonitis (jumper's knee) should also be considered. Finally, arthroscopy may reveal a synovial shelf, synovitis or an inflamed fat pad (Hoffa's disease).

Evaluation

1. Subluxation. Subluxation occurs when the patella moves laterally or medially from its normal position in the trochlea. The patient complains of instability rather than pain. Osteoarthritis does not usually occur unless the articular cartilage has been damaged by dislocation. A tangential radiograph taken at an angle of 30° to a knee flexed at 45° allows measurement of the angle of congruence (Merchant) which is the relationship between the apex of the patella and the bisected trochlea (sulcus angle). It is pathological if more than 16° and gives a measure of subluxation.

2. Tilt. Lateral tilt results from the application of a traction force to the lateral portion of the patella. It can cause chronic pain especially in the lateral retinaculum which undergoes adaptive shortening. Increased pressure on the lateral facet may produce osteoarthritis. The lateral patello-femoral angle (the angle between the lateral patellar facet and the most prominent points of the

trochlea measured on a tangential radiograph with the knee flexed to 20°) should open laterally. Both CT and MRI can document the degree of patellar tilt accurately.

3. Tilt and subluxation. These patients have increased load on the lateral facet, decreased load on the medial facet, central patellar shear and instability. Osteoarthritis is frequent.

4. Articular damage. Arthroscopic examination of the knee is useful in documenting the site and severity of damage to the articular cartilage in those patients who require defmitive surgery. Damage to the articular surface (chondromalacia patellae) is defmed as grade 1 when there is only softening, grade 2 if there is fibrillation less than 1 cm in diameter, grade 3 if more than 1 cm and grade 4 if there is exposed bone. Arthroscopy may reveal other pathology which can be dealt with at the same time.

Treatment

1. Conservative. Stretching of the retinaculum and strengthening of the quadriceps (especially vastus medialis) may improve symptoms. An elastic knee support, non-steroidal anti-inflammatory tablets and avoidance of precipitating activities are also beneficial. Failure to respond to conservative measures suggest that arthroscopic assessment is indicated as a prelude to further surgery.

2. Operative.

(a) *Realignment.* This is indicated in cases where there is definite malalignment and a failure to respond to conservative treatment. A lateral retinacular release is likely to be of benefit if there is tilt and mild articular damage (grade 1/2). If there is severe damage (grade 3/4) a retinacular release may not be sufficient and an anteromedial tibial tubercle transfer is indicated. Mild degrees of patellar subluxation may respond to lateral release but the results are not predictable and the surgeon may have to resort to anteromedial transfer.

(b) *Chondrectomy.* Arthroscopic cartilage shaving may smooth roughened areas when these are extensive, improving symptoms in 84%. Excision and drilling to encourage fibrocartilage formation is appropriate if there are circumscribed areas of damage and yields satisfactory results in 60%. Spongialization (removal of the subchondral bone beneath damaged cartilage) is reported to produce good or excellent results in 79%.

(c) *Others.* Anterior (but not medial) tibial tubercle advancement improves symptoms in two-thirds of patients who have painful arthritis of the patello-femoral joint without evidence of malalignment. Resurfacing arthroplasty is only satisfactory in 55% of cases after 3–6 years. The results are better in osteoarthritis (65% satisfactory) when there is no tibio-femoral involvement. Patellectomy remains an operation of last resort: good results can be expected in 72%.

Complications

Distal or posterior advancement of the tibial tubercle may lead to osteoarthritis because of increased pressure. Loosening and fracture plague resurfacing procedures. Skin necrosis is the main problem following tibial tubercle advancement. Patellectomy weakens the power of the quadricepsby 15–30%.

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Related topics of interest

Arthritis of the knee (p. 39)
Arthroscopy (p. 49)
Osteotomies around the knee (p. 211)

ARTHRITIS OF THE ANKLE

Primary osteoarthritis of the ankle is rare. Secondary osteoarthritis occurs most commonly after ankle fracture, ligamentous instability or inflammatory arthritis. Despite being the most frequently injured weight-bearing joint, it has the lowest incidence of degenerative joint disease.

Clinical problems

1. Post-traumatic osteoarthritis. The incidence of osteoarthritis after ankle fractures is 20–40% and is related to the *severity of the injury* and the *accuracy of its reduction*. When an anatomical reduction is maintained until union, both internal flxation and closed treatment give comparable clinical results. The risk of developing post-traumatic osteoarthritis is also similar.

The severity of the injury can be quantified by the degree of displacement and comminution, and the presence or absence of a posterior malleolar fracture. Fractures of the tibial plafond are particularly likely to initiate arthritic change in the joint because of the degree of comminution of the articular cartilage.

The most reliable indicator of an *accurate reduction* is the talo-crural angle which reflects the amount of fibular rotation and shortening. On an antero-posterior X-ray of the ankle in internal rotation (the mortise view) a line is drawn parallel to the tibial plafond and a second between the tips of the malleoli. The talo-crural angle is the superomedial angle subtended when a perpendicular is dropped from the line parallel to the plafond. The norrnal range in adults is 83 ±4°. A talocrural angle which is more than 5° outside this range is associated with a poor prognosis.

Arthritis is more likely to occur after a fracture in which *lateral displacement of the talus* has not been fully corrected. This is due to a marked reduction in tibio-talar contact area which occurs with only small degrees of lateral displacement. The majority of arthritic change occurs in the first 12–18 months. The worst results are obtained in women over the age of 50.

Premature osteoarthritic change may also follow stage 4 *osteochondritis dissecans of the talus,* in which a free osteochondritic fragment is present in the joint.

2. Osteoarthritis of chronic instability. The presence of lateral ligament insufficiency with varus tilt of the talus in the mortise will cause premature degenerative change (see Ankle instability, p. 22).

3. Haemophilia. Haemophilia is a rare cause of ankle arthritis, but in haemophiliacs the ankle is the third most common joint to become arthritic.

4. Rheumatoid arthritis (RA). The ankle joint is symptomatic in approximately 50% of patients with lower limb involvement and is second only to the knee joint as a source of symptoms in the lower limb. It is rarely affected in isolation from the joints of the foot. The ankle joint appears to become involved as a result of abnormal stresses placed on it by deformity in other involved joints. The ankle may be painful without clinical deformity or radiographic evidence of degenerative change. Selective injections of local anaesthetic into surrounding joints may help to make the diagnosis. The tendon sheaths of the peronei and medial flexor tendons may also be involved. Chronic synovitis may cause tendon rupture. If tibialis posterior ruptures, the hindfoot may deform into valgus and precipitate secondary changes in the ankle joint.

There are three main patterns of ankle deformity in RA. The *valgus ankle* is seen with pes planus and subtalar involvement. It may progress to cause lateral impingement on the fibula and diastasis of the distal tibiofibular joint. The deformity is well tolerated and patients function well despite marked deformity. The *varus ankle* is rare but functionally poor. The *neutral equinus ankle* follows a chronic synovitis with progressive ankylosis and proximal migration.

Management

1. Investigation. AP and lateral weight-bearing radiographs with appropriate stress views or corrected views in an orthosis should be obtained. Tomograms and CT may give additional information. Arthrography is rarely useful in assessing the degree of arthritis but may help to identify loose bodies.

2. Physiotherapy. Physiotherapy is likely to give only temporary relief in established arthritis but may improve the symptoms of instability.

3. Shoe modifications. Shoe inserts and insoles can correct minor varus and valgus deformities. Boots with ankle supports may help with instability.

4. Splints and orthoses. Valgus, varus and equinus deformities may all be treated with an appropriate ankle-foot orthosis (AFO). This is most likely to be a calliper with or without a T-strap. Plaster of Paris casts may be required during episodes of acute synovitis.

5. Steroid injections. Intra-articular local anaesthetic and steroid injections can be used for acute exacerbations of all forms of non-infective arthritis.

6. Surgery. There are five basic types of procedure. Degenerative arthritis accompanied by lateral instability may be improved by lateral *ligament reconstmction* (e.g. Watson-Jones tenodesis). *Synovectomy* can produce good symptomatic relief in RA when medical treatment has failed and the articular cartilage is relatively undamaged. *Arthrodesis* is indicated in isolated ankle joint

osteoarthritis or in rheumatoid arthritis with unilateral ankle involvement, preferably in 'burnt-out' cases with good distal mobility of the foot. In bilateral disease, rising from a seated position is extremely difficult if both ankles are fused, particularly if the feet are stiff. The method of ankle arthrodesis depends on the experience of the surgeon and may include compression arthrodesis with an external clamp (e.g. Charnley) or using internal fixation with screws. There is a significant complication rate after ankle fusion including infection, poor wound healing (especially in rheumatoids), malunion and non-union. Non-union occurs in up to 25% of reported cases. The role of *arthroscopy* is well defined for the removal of loose bodies from the ankle and for drilling osteochondral defects in osteochondritis dissecans. Its role in the symptomatic relief of arthritis is less clear, but reports of arthroscopic ankle arthrodesis and synovectomy are beginning to appear. The results of ankle *arthroplasty* are not universally successful. The results of arthroplasty for post-traumatic osteoarthritis in young patients are particularly poor. Better results have been obtained in patients with rheumatoid arthritis and in elderly patients with osteoarthritis. Arthroplasty may be undertaken in individuals with stiff feet in whom bilateral arthrodesis would result in poor function.

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Related topics of interest

Hindfoot arthrodesis and osteotomy (p. 147)
Orthopaedic manifestations of haemophilia (p. 199)
Osteoarthritis (p. 202)
Osteochondritis dissecans (p. 205)
Rheumatoid foot (p. 248)

ARTHRITIS OF THE ELBOW

The elbow joint is involved in approximately half of those with rheumatoid arthritis, in whom it is often disabling. Osteoarthritis of the elbow is usually the result of trauma. Pain is frequently accompanied by swelling from soft tissue inflammation and may interfere with the ability to carry loads. Stiffness prevents accurate positioning of the hand in space.

Biomechanics

The elbow is composed of two independent uniaxial joints. The humero-ulnar joint is a hinge joint between the trochlea and the olecranon. The instant centre of flexion-extension is in the centre of the trochlea. The axis of rotation of the forearm is in a line drawn between the ulnar and radial heads. In extension valgus stress is limited equally by the medial collateral ligament, the capsule and the joint surface. Varus stress is limited equally by the lateral ligament, capsule and joint surface. In flexion, the medial collateral ligament provides most of the restraint to valgus stress and the joint surface prevents excessive varus deformation. The maximum elbow strength occurs at 90° of flexion: the joint reaction forces are greatest in extension. Over half of the axial load is borne by the radio-capitellar articulation.

Problems

1. Nodules and bursae. Large rheumatoid nodules and bursae overlying the olecranon may be excised if troublesome.

2. Synovitis. This is commonly seen in rheumatoid arthritis.

3. Loose bodies. These may be due to osteophytes, synovial osteochondromatosis, separation of fragments of the capitellum in osteochondritis dissecans and ununited fractures. Joint locking is the usual presenting symptom.

4. Instability. Injury or disease affecting the collateral ligaments, capsule or joint surface may make the elbow unstable.

5. Bone loss. This may occur as a result of trauma, e.g. after a comminuted fracture of the radial head, or in severe rheumatoid disease.

6. Proximal radio-ulnar joint disease. Localized pain on pronation or supination may suggest that the disease process is limited to the proximal radio-ulnar joint, in which case a more limited surgical approach may be considered.

7. Humero-ulnar disease.

Management

1. Diagnosis. After clinical evaluation and plain radiographs, arthrography, tomography, scintigraphy and MRI may assist diagnosis.

2. Treatment.

(a) Medical. NSAIDs, physiotherapy and steroid injections may all be beneficial.
(b) Surgical.

- *Arthroscopy.* Arthroscopy may aid diagnosis and allows the removal of loose bodies. There may be a role for drilling and debriding osteochondritic lesions.
- *Synovectomy.* In the early stages of rheumatoid arthritis, synovectomy relieves pain in 80–90% of patients but this figure drops to 50% at 5 years. There is rarely any lasting effect on movement.
- *Radial head excision.* This procedure is commonly performed at the time of synovectomy although there is some debate as to its role in early rheumatoid disease. In advanced disease of the proximal radio-ulnar joint it may restore significant pain-free function. Replacement by a silastic implant is not indicated for arthritis, since radial head excision gives a better range of movement with less pain.
- *Excision arthroplasty.* Excision of the joint surface to form a pseudarthrosis is of value in the treatment of young patients with post-traumatic arthritis, and for patients with a septic joint or a failed elbow arthroplasty. The results are worse in rheumatoid arthritis because of instability and the lack of muscular control.
- *Fascial interpositional arthroplasty.* This may be indicated for young patients with post-traumatic arthritis. A strip of fascia lata is placed between the contoured bone ends. The results are acceptable in 70%.
- *Replacement arthroplasty.* Joint replacement arthroplasty may use a constrained hinge, a semiconstrained sloppy hinge, which allows some varus-valgus toggle, or an unconstrained surface replacement relying on the natural ligaments and joint design for stability. Both semiconstrained and unconstrained prostheses produce 90% satisfactory results at 5 years in rheumatoid patients. The satisfactory results drop to 75% in young patients with osteoarthritis.
- *Arthrodesis.* This operation should be avoided if possible because function afterwards is poor. It may be indicated for sepsis or after failed joint arthroplasty. Sound fusion will only be achieved in half. The position of

fusion is 110° of flexion (for eating). If bilateral arthrodesis is undertaken, the other elbow should be fused at 65° for the purposes of toilet.

Complications

1. Loosening. Loosening requiring revision occurs in about 7% at 5 years.

2. Ulnar nerve. Up to 15% may develop some degree of neuritis and 3% nerve entrapment postoperatively. This may be reduced by routine superficial neurolysis at the time of surgery.

3. Skin, infection and soft tissues. Wound problems develop in up to 14%, but may be minimized by using a long postero-medial incision and suction drainage. Infection should occur in less than 2%. Triceps problems occur in 4%.

4. Instability. This occurs after 3.5% of primary arthroplasties. In cases of severe bone erosion or revision arthroplasty a semiconstrained prosthesis must be used. The lateral ligament is usually intact but if the anterior portion of the medial ligament is destroyed then a slip of flexor carpi ulnaris may be used to restore stability.

5. Fracture. Fracture of the humerus and ulna each occur in up to 5%.

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Related topics of interest

Rheumatoid hand (p. 251)

Rheumatoid wrist (p. 257)

ARTHRITIS OF THE HIP

Pain from an arthritic hip is normally felt in the groin or in the region of the greater trochanter and radiates down the front of the thigh to the knee and occasionally beyond. Pain is the usual reason for loss of mobility. Stiffness leads to inability to flex the hip sufficiently to tie shoe laces and put on socks or stockings. There may be apparent shortening of the leg from an adduction contracture. In patients with severe bilateral adduction deformities, there may be severe difficulty with personal toilet.

Clinical problems

1. Osteoarthritis. Osteoarthritis (OA) occurs in up to 40% of the population over 60 years.

In central type OA there is concentric reduction of the joint space and disease is often bilateral at presentation. It is more common in females and in older age groups.

In superior pole OA there is loss of joint space superolaterally. It is more common in men and is often related to a structural problem such as trauma, infection, developmental dysplasia, Perthes' disease, slipped upper femoral epiphysis and avascular necrosis. Medial buttressing may occur with thickening of the calcar. Progression is associated with superolateral migration.

The natural history of early OA is poorly understood but one study suggests that symptoms have deteriorated in only 17% at 10 years and that many improve. Progression is believed to be associated with obesity, superior pole OA, chondrocalcinosis elsewhere and, possibly, NSAID ingestion.

2. Rheumatoid arthritis. The hip is involved radiographically in 33% at 5 years from onset rising to 50% at 8 years. It is a late manifestation of the disease. A small group may deteriorate rapidly.

3. Juvenile chronic arthritis (JCA). In JCA the capital epiphysis fuses early giving rise to a short thick anteverted femoral neck. Because the lever arm of the abductor mechanism is shortened, the patient walks with a Trendelenburg gait. The femoral canal is often small and bowed. Flexion contractures occur early. In stage I disease both hip movements and radiographs are nearly normal. In stage

II, hip movements are reduced but the joint space remains virtually normal. In stage III there is little hip movement and the joint space is reduced radiologically.

4. Ankylosing spondylitis. Flexion contractures of both hips make personal toilet and walking difficult, especially if there is kyphosis. Sixteen percent of those aged 10–15 at onset, 10% of those aged 15–20 at onset and 1% of late presenters require total hip replacement.

5. Metabolic disease. The hips may also be affected by metabolic disease (including Paget's) in which degenerative disease supervenes.

6. Protrusio acetabuli. This may be primary (Otto's disease) or associated with rheumatoid arthritis, osteomalacia, Paget's disease, Marfan's syndrome and trauma. The femoral head migrates beyond the ilio-ischial (Kohler's) line.

Management

1. Assessment. This should include assessment of the functional problems caused by the arthritis. Patients with rheumatoid disease should also be assessed by a physiotherapist and an occupational therapist.

2. Conservative. Rest, a shoe raise, a walking stick in the opposite hand, NSAIDS and physiotherapy are appropriate for patients who do not need surgery. Those with rheumatoid disease may require second and third line medication.

3. Soft tissue surgery. Adductor and psoas tenotomy may be indicated for children with stage II JCA.

4. Osteotomy. Osteotomy may be indicated in early osteoarthritis, particularly when this is secondary to childhood disease. Enthusiasts report an 85% improvement in pain after intertrochanteric osteotomy. Abduction osteotomy is indicated if there is a limp, adduction deformity, motion in adduction beyond the deformity and painful abduction. Adduction osteotomy is indicated if there is an abduction deformity, motion in abduction beyond the deformity and painful adduction. Medial displacement of the distal fragment appears to be important in reducing the load borne by the hip. Patients with advanced OA who have less than 50° of flexion are not suitable for this type of surgery. In AVN, the osteotomy should bring an uninvolved area of the femoral head to the weight-bearing area. Pelvic osteotomy should be considered in those with incongruity resulting from acetabular dysplasia.

5. Anhrodesis. If hip arthrodesis is to be successful, the patient must have good pain-free function of the spine, opposite hip and both knees. The optimal position is 30° of flexion, 0–5° abduction and 10–15° external rotation. Ten percent go on to non-union. Seventy-eight percent are satisfied 20 years later although 57% have some back pain and 45% some knee pain. Thirteen percent will require revision to arthroplasty. There are many techniques described of which arthrodesis with cobra plate fixation is recommended.

6. Arthroplasty. Hip replacement relieves pain in 96% of patients with RA at 6–11 years. It should be appreciated that their functional requirements are less

than those of patients with OA. Mechanical loosening is likely with time and the infection risk is nearly twice normal because of poor wound healing.

Total hip replacement in JCA is also associated with a high failure rate (30%) at 6–18 years follow-up.

In secondary osteoarthritis due to acetabular dysplasia there may be considerable problems in seating the acetabular component. The choice lies between leaving the patient with a high hip centre or relocating the centre of rotation to the anatomical position with bone grafting. The latter is associated with high failure rate at 7 years if block grafts are used.

In cases of dysplasia and JCA the femoral neck may be highly anteverted and the shaft of small dimensions. Such cases may require customized implants. In cases of protrusio, the neck may need to be cut *in situ* and the sciatic nerve may lie closer to the field of surgery than normal. Relocation of the hip to the anatomical position is essential to avoid loosening.

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Related topics of interest

Developmental dysplasia of the hip (p. 119)
Hip arthroplasty (p. 150)
Rheumatoid arthritis (p. 245)
Young adult hip problems (p. 340)

ARTHRITIS OF THE KNEE

Arthritis of the knee commonly presents with pain on weight-bearing which radiates from the knee down the anterior aspect of the tibia. Night pain is less common than in hip disease. Swelling may be due to joint effusion, synovitis or osteophyte formation. Stiffness is often worse in the morning. Patients may complain of deformity.

Examination reveals an antalgic gait, the cause of the swelling and a restricted range of movement. Genu varum deformity is more common in osteoarthritis and genu valgum in rheumatoid arthritis. Weight-bearing radiographs of the osteoarthritic knee show loss of joint space, osteophytes, sclerosis and, rarely, cyst formation. In rheumatoid arthritis the radiographs show loss of joint space, erosions and deformity. The hip joints and spine need to be assessed clinically and, if necessary, radiologically. Synovial fluid should be analysed for micro-organisms, crystals, complement and immunoglobulins.

Clinical problems

1. Unicompartmental osteoarthritis. This may follow deformity, trauma or meniscectomy. A varus or valgus deformity develops with loss of joint space on the affected side. Treatment depends on the age of the patient and the severity of their symptoms. Arthroscopic assessment is recommended in younger patients.

2. Bicompartmental osteoarthritis. If both tibio-femoral joints are involved there is no place for osteotomy. The very frail and the very young may benefit from an arthroscopic washout. Total joint replacement offers the best relief of symptoms in the severely disabled.

3. Patello-femoral osteoarthritis. Anterior knee pain on climbing and descending stairs suggests patello-femoral involvement. In the presence of tibio-femoral arthritis total knee replacement may be appropriate. Isolated patello-femoral disease may be treated by tibial tubercle elevation, patellectomy or patello-femoral joint replacement.

4. Rheumatoid arthritis. The knee is affected at presentation in 20%, rising to 90% when the disease is of long standing. Synovial proliferation causes damage to ligaments, cartilage and bone. There is commonly a fixed flexion deformity, marked synovitis, joint effusion and progressive instability. Instability may be

caused either by involvement of the ligaments or due to bony destruction of the tibial or femoral condyles.

5. Seronegative arthropathy. The knee is frequently involved in Reiter's disease and the enteropathic arthropathies but less commonly in ankylosing spondylitis and psoriasis.

6. Crystal arthropathy. Pyrophosphate arthropathy with chondrocalcinosis occurs in the knee in over 50% of cases. Acute uric acid gout and chronic tophaceous gout may involve the knee.

7. Others. Synovial osteochondromatosis presents with locking due to loose bodies. The knee is the most common joint affected by haemophilia. Pigmented villo-nodular synovitis presents with swelling which becomes painful.

Management

1. Conservative. Symptoms from early disease may be controlled by NSAIDs, quadriceps exercises, ultrasound and ice. Knee bracing may control instability. Splintage in acute rheumatoid disease helps reduce disease activity but should be brief to avoid stiffness. Intra-articular steroid injection provides benefit in osteoarthritis of up to 4 weeks but lasts longer in rheumatoid arthritis and pyrophosphate arthropathy.

2. Arthroscopy. Washout, resection of unstable meniscal fragments and debridement of the knee may produce improvement in symptoms for up to 5 years in 66% of cases.

3. Osteotomy. Eighty-five percent of patients under the age of 60 years with unicompartmental disease have good or excellent results after osteotomy at 5 years. Perform proximal tibial osteotomy for varus deformity and distal femoral osteotomy for valgus deformity. In those with isolated patello-femoral disease consider a tibial tubercle elevation to unload the joint but note that it is associated with a high incidence of complications.

4. Unicompartmental knee replacement. The ideal candidate is over the age of 60 years, not obese, not extremely active, with unicompartmental disease, a functioning anterior cruciate ligament and minimal flexion deformity. Success is reported at 85–95% at 5 years falling to 70% at 10 years. Revision to total condylar knee replacement is possible but 14% require a further revision by 5 years.

5. Total knee replacement. Total condylar knee replacement has a success rate of over 90% at 10 years in osteoarthritic patients over 60 years of age. The revision rate by 10 years is 3%.

6. Patellectomy. Sixty percent are rendered pain-free but weakness, joint instability and extensor lag are common.

7. Patello-femoral joint replacement. Although the short term results are satisfactory, the longer term results are disappointing. Consider total knee replacement in appropriate patients.

8. Arthrodesis. This may be indicated after septic arthritis, in mono-articular arthritis and in neuropathic disease. Compression plate fixation and intra-medullary fixation are the methods most commonly employed.

9. Synovectomy. Seventy percent of patients over the age of 45 years with early rheumatoid arthritis may expect good results at 2 years after chemical synovectomy with yttrium, osmic acid or dysprosium ferric hydroxide. Open surgical synovectomy produces improvement in 75% at 2–5 years with deterioration to 55% at 9 years. Arthroscopic synovectomy produces comparable results but is less complete.

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Related topics of interest

Anterior knee pain (p. 25)

Osteotomies around the knee (p. 211)

ARTHRITIS OF THE SHOULDER

Arthritis of the shoulder is much rarer than that of the hip or knee. It is usually secondary to chronic destructive inflammatory disease or local trauma. Unilateral shoulder arthritis can often be managed symptomatically. Even when the glenohumeral joint is grossly restricted in its range, scapulo-thoracic movement may provide adequate function. Profound functional disability occurs when the condition becomes bilateral.

Clinical problems

1. Osteoarthritis of the glenohumeral joint. Primary osteoarthritis is rare and probably does not occur. Degenerative arthritis is usually secondary to trauma (e.g. following intra-articular fracture), or to a long-standing rotator cuff injury. It presents most commonly between the age of 50 and 60 with a history of painful arc syndrome and a decreased global range of movement. Rarely it may present as a complication of haemophilia, sickle cell disease, avascular necrosis or infection.

2. Osteoarthritis of the acromioclavicular joint (ACJ). This condition is common in the elderly and presents as painful swelling of the ACJ. It may be associated with previous shoulder trauma or pathology.

3. Rheumatoid arthritis of the glenohumeral joint. The glenohumeral joint is commonly involved in RA. The involvement is usually bilateral. There is marked synovial proliferation and, because the shoulder capsule is lax, there may be a large effusion. The patient usually presents with pain, stiffness and reduction in shoulder movements, particularly abduction. Synovitis of the rotator cuff may produce impingement and tenosynovitis around the long head of biceps may lead to rupture. Suprascapular nerve compression may also occur as a result of the synovitis and typically cause pain at the apex of the shoulder.

4. Rheumatoid acromioclavicular joint disease. The ACJ is invariably involved in RA. It may produce severe erosive arthritis with subsequent instability.

5. Idiopathic destructive arthropathy (Milwaukee shoulder). This is a rapidly progressive and destructive form of arthritis causing severe erosion of the glenohumeral joint, the acromion and ACJ. It is usually preceded by long-

standing rotator cuff pathology and is due to shedding of hydroxyapatite crystals from the torn rotator cuff. This initiates destructive lysosomal enzyme release from the synovium. It affects patients of 60 years or more with a history of chronic shoulder pain. There is an acute episode of swelling and instability with marked crepitus and loss of movement. Radiographs show severe erosion and subluxation. Response to treatment is poor.

Management

Investigations

If the primary pathology is evident, e.g. rheumatoid disease, haemophilia, infection or rotator cuff disease, the appropriate disease-specific investigations are carried out (see relevant section).

1. Radiographs. Plain radiographs showing the glenohumeral and acromioclavicular joints are essential. Characteristics of OA, RA, infection or traumatic arthritis may be evident.

2. Joint aspiration. Synovial fluid may be aspirated to confirm crystal arthropathy or to exclude infection.

3. Synovial biopsy. May be required to exclude malignant disease or pigmented villo-nodular synovitis.

Non-surgical treatment

1. Oral medication. Most shoulder arthritis can be controlled with simple analgesia and NSAIDs.

2. Splintage. Acute exacerbations, particularly of rheumatoid arthritis, may respond to short periods of rest in a sling or brace.

3. Physiotherapy. Local treatment, e.g. ultrasound, exercises and gentle manipulation, may maintain or regain range of movement.

4. Joint aspiration and intra-articular steroids. In RA, aspiration of a tense joint effusion and intra-articular corticosteroid injection may control symptoms. Subacromial injection may control degenerative arthritis associated with rotator cuff lesions.

Surgical treatment

1. Synovectomy. In early RA, before significant joint erosion has occurred, synovectomy can control symptoms and may delay progression of disease.

2. Arthroscopy. Joint debridement, shoulder decompression, removal of loose bodies and limited synovectomy may all give symptomatic relief. In young patients, excision of osteophytes and acromioplasty may defer more radical surgery.

3. Arthrodesis. Arthrodesis is rarely indicated in RA. The main indications are in the management of degenerative arthritis in younger patients with an otherwise normal limb or in patients considered unsuitable for arthroplasty. When performed, a position of 20° of abduction, 30° of forward flexion and 40° of internal rotation gives a good functional result.

Extra-articular arthrodesis fuses the humerus to the clavicle and the spine of the scapula by advancing bone blocks. The articular surfaces are left untouched and the shoulder is supported externally in a spica or with internal screw fixation. *Intra-articular arthrodesis* removes the remaining articular cartilage from the humeral head, glenoid and adjacent subacromial area. Position is maintained by screw fixation, plate and screw fixation. *Combined intra-articular and extra-articular arthrodesis,* employing a combination of the above techniques, may be used, particularly if the bone stock is poor.

4. Arthroplasty. Hemi-arthroplasty (prosthetic humeral head replacement) is primarily used for pain relief. It is commonly carried out for patients with RA or post-traumatic arthritis. It can be performed if total shoulder arthroplasty is not technically possible or in cases where the glenoid articular surface is well preserved. With *total shoulder arthroplasty (humeral head and glenoid surface replacement),* the best functional results are obtained in patients with OA because the rotator cuff is usually intact and bone quality is good. Such patients may regain a good range of movement as well as obtaining pain relief. *Glenoid excision* may be considered if replacement or arthrodesis are inappropriate (e.g. severe osteoporosis or infection) and can afford good pain relief. Proximal humeral excision arthroplasty provides poor functional and symptomatic results.

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Related topics of interest

Arthroscopy (p. 49)
Avascular necrosis of the hip (p. 53)
Orthopaedic manifestations of haemophilia (p. 199)
Rheumatoid arthritis (p. 245)
Rotator cuff injuries (p. 260)
Shoulder instability (p. 278)
Shoulder pain (p. 281)

ARTHRITIS OF THE WRIST AND FIRST CARPOMETACARPAL JOINT

Arthritis of the wrist and first carpometacarpal (CMC) joint may be primary (idiopathic) or secondary to fractures of the scaphoid or distal radius, Bennett's or Rolando fractures or scapholunate dissociation. Pain on stressing the wrist may indicate arthritis. Examination should be directed to the point of maximum tenderness. There is often a restricted range of movement with some crepitus. In the CMC joint, pain at the base of the thumb on pinch grip is characteristic. Axial rotation and grinding of the thumb are also painful. Radiographs usually reveal a loss of joint space, sclerosis and osteophyte formation. Cystic change may be present: intraosseous ganglia and rheumatoid arthritis should be considered as possible alternative causes.

Clinical problems

1. Scapholunate advanced collapse (SLAC). Degenerative change in the radiocarpal joint may be seen after fracture of the scaphoid or SL dissociation. Four stages are recognized. *Stage I* is characterized by beaking of the radial styloid and loss of joint space between the radius and the scaphoid. *Stage II* disease has more extensive involvement of the radioscaphoid articulation. In *stage III* the capitolunate and scaphocapitate joints are involved and in *stage IV* there is SL dissociation with proximal migration of the capitate.

2. Scaphoid-trapezium-trapezoid (STT). This is the second most common pattern in the wrist. It is frequently associated with CMC arthritis.

3. CMC arthritis. Most common in females over the age of 50 years. In power pinch, the joint surfaces shear and only the ulnar and the dorsal ligaments of the joint prevent subluxation. With time, the joint wears and the ligaments become weakened. Collapse of the thumb may occur producing a swan-neck deformity similar to the Nalebuff type II deformity seen in rheumatoid disease. Radiographs show progressive changes starting with widening of the joint space and mild subluxation in stage I, and progressing to more than one-third subluxation and small calcific deposits in stage II. Advanced changes with larger fragments and joint space narrowing occur in stage III and presage the major subluxation, sclerosis, cystic change and osteophyte formation characteristic of

stage IV. Involvement of the second metacarpal occurs in 86%, the scaphoid in 48% and the trapezoid in 35%. Carpal tunnel syndrome may occur.

Management

1. Diagnosis. Careful history-taking, examination and radiographic examination will reveal the diagnosis in most cases. Consider blood tests for rheumatoid disease in the presence of erosive changes. A bone scan may be helpful if the radiographs are equivocal. Arthrography and MRI may help in the diagnosis of early OA.

2. Arthroscopy. Arthroscopy may be indicated to assess the state of the joint surfaces, to remove loose bodies or to biopsy the synovium.

3. Conservative treatment. A trial of conservative treatment is indicated in most patients. Steroid injections, NSAIDs and splintage may be of benefit.

4. Triscaphe (STT) arthrodesis. This is advocated for STT arthritis when the CMC joint is unaffected. If the CMC joint is involved then it must be combined with excision of the trapezium. It is contraindicated in the presence of radioscaphoid OA.

5. Styloidectomy and excision of the proximal scaphoid fragment. These operations carry a reasonable chance of success if carried out for arthritis of the radioscaphoid joint secondary to non-union or avascular necrosis of a scaphoid fracture. Excision of the proximal fragment is indicated if less than one-quarter of the scaphoid is involved. If a larger fragment is removed the capitate will collapse back into the space left behind. Silastic spacers have been used but carry the risk of silicone synovitis. A folded tendon graft can be used.

6. Proximal row carpectomy. This procedure relieves pain in approximately 90% of patients. It must be undertaken before degenerative change occurs in the midcarpal joints. It is indicated in those for whom residual mobility is important. In manual labourers, an arthrodesis is a better option. Some residual pain is not uncommon: arthrodesis remains a salvage procedure. The triquetrum and lunate are excised completely. The scaphoid may be excised in its entirety or the distal half may be left to stabilize the thumb. If the latter option is chosen it should be accompanied by a styloidectomy.

7. Wrist arthrodesis. This may be indicated for severe radiocarpal arthritis. The wrist should be fused at 10–20° of extension. The compression plate technique is most commonly used. The carpus is pulled on to the radius to avoid impingement by the distal ulna. Complications include non-union, injury of the cutaneous branch of the radial nerve, sloughing of the wound edge and adherence of the extensor tendons.

8. CMC joint. If, in the early stages of the disease, the CMC joint is unstable, the ulnar ligament may be stabilized using half the flexor carpi radialis tendon (Eaton-Littler reconstruction). In younger patients with mild disease, an osteotomy of the metacarpal restores painless pinch grip in 84% at 12-year follow-up. In more severe cases an arthrodesis is indicated to provide a painless

grip. In the older patient excision of the trapezium provides reliable relief of pain but may be accompanied by instability of the thumb. If a silastic implant is used to fill the defect, subluxation or dislocation will occur in 5–20% and silicone synovitis in up to 50%. Resection of the trapezium with interposition of a rolled tendon or resurfacing by an artificial joint may provide more stability. Z-hyperextension deformity of the thumb MCP joint should be corrected at the same time by tenodesis, capsulodesis or arthrodesis.

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Related topics of interest

Carpal instability (p. 78)
Osteoarthritis (p. 202)
Rheumatoid hand (p. 251)
Wrist pain (p. 337)

ARTHROSCOPY

Arthroscopy has revolutionized the management of many joint conditions. Advances in technology have allowed clear visualization of joints and manipulation of intra-articular structures. Surgery can be performed with minimal morbidity. The techniques pioneered in the knee are now employed in many joints. Certain procedures which were previously impossible or technically demanding can now be undertaken (e.g. repair of a posterior meniscal tear).

Equipment

1. A rigid arthroscope with lenses allowing a selection of fixed-angled fields. Most commonly used is 30°. The diameter of the scope varies on the joint being examined, e.g. 4 mm for larger joints (knee and shoulder) and 1.7 mm or 2 mm for smaller joints (wrist, ankle and elbow).

2. Fibreoptic light source. High-output halogen bulb systems transmit light from the source to the scope along a multi-filament fibreoptic cable.

3. Television camera and monitor. Miniaturization of cameras has led to equipment which is easy to manipulate, thereby improving the comfort of the operator and reducing the risk of bacterial contamination.

The above equipment can be sterilized by immersion in a sterilizing fluid, e.g. Cidex (glutaraldehyde), which allows a rapid turnover of cases if necessary. Because of possible toxicity, glutaraldehyde sterilization should be carried out in an enclosed cabinet.

4. Additional instruments. A variety of hand-operated and powered instruments are available for an ever-increasing range of procedures. Electrocautery and lasers are available for both haemostatic cauterization and for cutting.

5. Irrigation with normal saline or Hartman's solution is used to distend the joint and to maintain a clear field of vision.

6. Tourniquet is usually used except in the most proximal joints (hip and shoulder).

Clinical applications

General indications for arthroscopy include diagnosis and assessment (although this may shortly be superseded by MRI), synovial biopsy, removal of loose bodies, excision of osteophytes, irrigation of septic arthritis, for drilling or shaving within the joint (e.g. osteochondritis dissecans, chondromalacia patellae), and synovectomy.

1. Lower limb

(a) *Hip arthroscopy* is performed infrequently.

- *Specific indications*—excision of torn labrum and removal of loose bodies.
- *Position*—supine on traction table with image intensifier.
- *Entry portals*—initial distension of joint with 50 ml of saline via a spinal needle under image intensifier control. The primary portal is located 4 cm lateral to the femoral artery and 4 cm inferior to the inguinal ligament. A further portal lateral to this can be used for instrumentation.

(b) *Knee*

- *Specific indications*—meniscectomy, meniscal repair, plica resection, lateral retinacular release, cruciate ligament repair.
- *Position*—supine.
- *Portals*—routinely a medial and lateral para-patellar tendon portal with additional portals sited as necessary for specific procedures.

(c) *Ankle*

- *Specific indications*—drilling of osteochondral defects of talus, ankle arthrodesis.
- *Position*—supine on table with knee flexed over the end. Joint distraction required for adequate access.
- *Portals:*

    a. Anterolateral: at tibio-talar joint line just lateral to peroneus tertius tendon.
    b. Anteromedial: at tibio-talar joint line just medial to tibialis anterior (this portal is made after transillumination from the lateral side to identify saphenous vein and nerve).
    c. Posterolateral: just lateral to the tendo-Achilles 1 cm distal to the anterior portals.

2. Upper limb

(a) *Shoulder*

- *Specific indications*—debridement of biceps tendon or rotator cuff tears, excision of labral tears, sub-acromial decompression for impingement, excision of calcific tendinitis, assessment of instability and repair.
- *Position*—lateral decubitus or deckchair positions. Distraction in 45° of abduction and 15° of forward flexion.
- *Portals*—distend joint with 50 ml of saline.

    a. Posterior: 2 cm inferior and 2 cm medial to the posterolateral corner of acromion (soft spot between infraspinatus and teres minor). Direct trocar towards coracoid.
    b. Anterior: midway between anterior lateral corner of acromion and coracoid.

(b) *Elbow*

- *Portals*—the two anterior portals require elbow to be flexed at 90° and the joint well distended to prevent neurovascular damage.

    a. Anterolateral: 3 cm distal and 1 cm anterior to lateral epicondyle just anterior to the palpated radial head.
    b. Anteromedial: 2 cm distal and 2 cm anterior to medial epicondyle.
    c. Posterolateral: 3 cm proximal to the tip of the olecranon at the lateral supracondylar ridge.

(c) *Wrist*

- *Specific indicatiom*—assessment of carpal instability (especially scapho-lunate dissociation), examination of joint surfaces, pre-operative assessment before fusion or after trauma. Excision of the triangular fibrocartilage complex.

  The joint is distracted using finger traps and a counter-weight then distended with 10 ml of saline.
- *Portals*—multiple portals may be needed. These are located between the extensor compartments at the wrist, and are labelled according to the compartment on the radial side (i.e. portal 1 between compartments 1 and 2, etc.)-

  Portal 3 (i.e. between extensor pollicis longus (3) and extensor digitorum communis (4)) is commonly used for radio-carpal joint examination.

  Mid-carpal examination is carried out using a portal 1 cm distal to portal 3. The position is confirmed by insertion of a hypodermic needle and distension with saline.

Complications

Complications are rare (around 1%) and are usually minor. These include haemarthrosis, infection, thrombo-embolic disease, anaesthetic complications and reflex sympathetic dystrophy.

1. Articular cartilage damage is probably the most common complication and is due to poorly placed entry portals. The long-term morbidity from this is unknown.

2. Damage to fat pad or menisci in the knee by misplaced portals.

3. Vascular damage. May occur because of direct injury from an inappropriately placed incision or from instruments. The popliteal vessels may be injured during meniscectomy or meniscal repair and the anterior tibial vessels during ankle arthroscopy.

4. Nerve damage. Peroneal and saphenous nerve injuries have been reported after meniscal repair. Shoulder arthroscopy may damage branches of the axillary nerve: the brachial plexus may be injured by traction.

5. Ligaments and tendons. Patellar tendon and collateral ligament injuries have been reported at the knee and rotator cuff injuries have occurred in the shoulder. The tendons of tibialis anterior and the peronei are vulnerable during arthroscopy of the ankle.

6. Extravasation of irrigation fluid occurs most commonly during shoulder arthroscopy and may cause compression damage to the neurovascular structures in the axilla.

7. Infection rate is 0.2% and has improved since the use of sterile cameras and waterproof drapes.

Further reading

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Related topics of interest

Anterior knee pain (p. 25)
Carpal instability (p. 78)
Musculoskeletal imaging (p. 190)
Rotator cuff injuries (p. 260)
Shoulder impingement syndromes (p. 275)
Shoulder instability (p. 278)

AVASCULAR NECROSIS OF THE HIP

Avascular necrosis (AVN) of bone occurs in the hip, knee, talus, shoulder and scaphoid. It frequently follows interruption of blood supply by trauma but is also associated with alcoholism, dysbarism (Caisson disease), steroids, radiation therapy, sickle cell disease and Gaucher's disease. The theories of aetiology include expansion of fat cells in the marrow and repeated intramedullary haemorrhage or thrombosis. Whatever the starting point, the outcome appears to be blockage of the osseous microcirculation. AVN most commonly occurs in the hips in the fifth decade and is bilateral in 50% of cases of idiopathic (Chandler's) disease but in up to 80% if steroid-induced.

Clinical staging

Clinical staging uses the system described by Ficat (1985).

Stage 0. This stage is pre-clinical and is only described in patients who have clinical involvement of the contralateral hip. One study suggests that 44% of hips with abnormally raised intramedullary pressure but without symptoms or signs will develop clinical disease.

Stage I. 50% present with sudden pain in the groin which is often worse at night. Abduction and internal rotation are limited. Radiographs show some blurring of the trabecular pattern with slight osteoporosis. The intramedullary pressure may be raised. Scintigraphy shows a cold spot which corresponds to the region of infarction of the weight-bearing surface of the femoral head demonstrated histologically.

Stage II. The clinical signs worsen and the radiographs show either localized or diffuse sclerosis in the femoral head. Small cysts may be seen. A bone scan will show increased uptake and histology reveals some spontaneous repair of the infarcted area by new bone.

Stage IIB. This is a transition phase between stage II and III disease characterized by the crescentic line of a subchondral fracture and flattening of the femoral head.

Stage III. There is a further flattening and a break in the articular margin with collapse of the sequestrated area into the femoral head.

Stage IV. This is the terminal phase with joint space narrowing due to loss of articular cartilage and the formation of osteophytes.

Management

The following discussion concentrates on techniques used in the management of AVN of the hip. Most of the available information is anecdotal since there have been few properly controlled studies. Similar results are seen following decompression, osteotomy and total knee replacement for ischaemic necrosis of the knee.

1. Imaging. The problem is how to identify the disease in its early stages before radiographic changes have taken place so that conservative therapy can be maximally effective. Scintigraphy may be helpful as isotope uptake is initially reduced and only increases as symptoms appear. Once AVN is established, the stage bears no relation to the bone scan findings. MRI appears to have a useful role in the early stages of the disease. Studies suggest a sensitivity of 87%, specificity of 83%, and positive and negative predictive values of 96% and 55%, respectively.

2. Intraosseous pressure studies. Ficat and Arlet recommend functional exploration of bone (FEB) by measurement of the bone marrow pressure and by intramedullary venography of the intertrochanteric area. They regard the upper limit of normal for intraosseous pressure as 30 mmHg, even 5 minutes after injection of 5 ml of saline (the stress test). They recommend repeating the test in the femoral head if the initial test is negative. In the second stage of FEB, they inject contrast and regard any reflux into the diaphysis or intramedullary stasis 15 minutes after injection as abnormal. The third stage of FEB is a core biopsy.

3. Core decompression. This aim of this procedure is to make the diagnosis, to relieve pain and to prevent further progression of the disease. A core measuring 6–8 mm in diameter and which extends to within 5 mm of the articular cartilage is removed. The procedure is only indicated for stages 0, I and II disease. At least 50–60% of those treated by core decompression progress radiologically by 3 years. Core decompression may be combined with fibular strut grafting in stage II disease. The fibula may be taken as a free vascularized graft or may be avascular. Core decompression has a complication rate of 5%: complications include femoral neck fracture and joint penetration.

4. Pulsed electromagnetic field. There is some evidence that using a pulsed electromagnetic coil for 6–8 hours a day may delay radiographic progression of stage I and II disease and even show improvement at 3-year follow-up.

5. Osteotomy. Sugioka described a transtrochanteric osteotomy which aims to rotate the uninvolved part of the femoral head into the weight-bearing area. His successful results were 91%, 88% and 73% in stage I, II and III disease respectively. Progression of the disease was associated with involvement of more than two-thirds of the femoral head on the pre-operative lateral radiograph, stage III and IV disease and steroid-induced AVN. Other authors have found 80%

satisfactory results in grades I and II but only 45% in stages III and IV. Significant complications (fracture and non-union) occur in up to 30%. These may be avoided by using blade-plate fixation rather than screws.

6. Total hip replacement. Total hip replacement is indicated for stage III and IV disease if symptoms are severe. Generally poor results are obtained with early loosening in up to 15% at a mean 6 years. This may rise to 45% in those treated for sickle cell disease.

Further reading

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Related topics of interest

Arthritis of the hip (p. 35)
Hip arthroplasty (p. 150)
Young adult hip problems (p. 340)

BIOMATERIALS

The factors which determine the success and longevity of an implant are its material properties, design and usage. The wide variations in implant design make use of a relatively small number of implant materials in different combinations.

Definitions

1. Stress. Stress is the force applied divided by the surface area. This load can be compressive, tensile, shear or torsional.

2. Strain. This is the relative deformation of a body as a result of loading, i.e. the change in length divided by the original length.

3. Elasticity. Young's modulus of elasticity (E), stress divided by strain, is the measure of stiffness in the elastic range of deformation where the body returns to its original size when the stress is removed.

4. Yield point (proportional limit). This is the point beyond which deformation is no longer elastic but plastic, i.e. there is permanent deformation of the material.

5. Ultimate strength. This is the maximum strength obtained by the material, the point at which the material fractures.

6. Brittle materials. These have a linear stress-strain curve up to the point of failure and so do not exhibit significant plastic deformation (e.g. PMMA cement).

7. Ductile materials. These undergo considerable plastic deformation before failure (e.g. metals).

8. Viscoelastic materials. The stress-strain behaviour changes with the rate of application of the stress, e.g. slow loading may cause an avulsion fracture of the lateral malleolus but rapid loading may cause ligament rupture.

Problems

1. Wear particles. Wear particles of cement, polyethylene, and metal have been implicated in the pathogenesis of aseptic implant loosening. The particles are found in the fibrous membrane surrounding loose implants. Smaller particles are

found in histiocytes and larger ones within giant cells. Activation of the macrophages causes release of kinins which stimulate osteoclastic resorption of bone.

2. Wear mechanisms. Adhesive wear is caused by the bonding of two articulating surfaces, usually polyethylene and metal. Continued motion causes the softer material to be transferred to the surface of the harder material. Abrasive wear is dependent on contact stress, surface hardness and surface roughness. A soft surface is abraded by a hard rough surface. Roughness depends on the distance between the peaks (asperities) and the troughs in the surface of a material. By making the articulating surface harder, the smooth fmish can be prolonged thereby reducing roughness by wear. High contact stresses between non-conforming surfaces increase wear. Fatigue may occur when peak stresses occur in a material, causing it to fracture or delaminate. Any wear debris may be caught between the articulating surfaces and cause third body wear.

3. Creep. This is the progressive deformation of a material over an extended period of time.

4. Corrosion. This is the chemical dissolving of materials. This may be due to the use of different materials (galvanic), fatigue cracks with areas of low oxygen tension (crevice), areas of high stress gradients (stress) and from small movements abrading the outside layer (fretting).

5. Fatigue. Repeated cycles of loading may cause failure of the material below its ultimate strength. It is related to the number and magnitude of the loading stress cycles.

6. Friction. Friction is the resistance to movement which occurs between two opposed surfaces. It may be deleterious in the case of moving joint surfaces or beneficial in the case of a screw holding in bone. Friction depends on load, not stress, i.e. is independent of surface area. Friction is proportional to load in a linear fashion over a wide range. Metal on metal, metal on UHMWPE (ultra-high molecular weight polyethylene), alumina (Al_2O_3) on UHMWPE and the natural hip joint have coefficients of friction of descending magnitude. Lubrication reduces friction. Hydrodynamic lubrication occurs because pressure in the fluid keeps bearing surfaces apart. In boundary lubrication a thin surface layer of lubricant uses surface properties to keep the surfaces apart. In elastohydrodynamic lubrication the lubrication film can transmit forces to the asperities on the opposite bearing surface and cause a relative smoothing of the surface. Weeping of fluid from a porous surface may help lubrication and a structured surface may hinder lateral flow of fluid, thereby keeping fluid between the surfaces (squeeze film). Lubrication in a natural joint is by a combination of boundary, elastohydrodynamic and weeping lubrication. In the replaced joint, lubrication is primarily elastohydrody namic.

Common materials

1. Ultra-high molecular weight polyethylene. This material is tough, ductile and has a low resistance to wear and friction. Wear increases in thin (<5 mm) components and in those which are metal-backed.

2. Metals. Cobalt-chrome, stainless steel and titanium have Young's moduli of decreasing magnitude, that is, they are increasingly flexible. All are stiffer than cortical bone, which is stiffer than PMMA and UHMWPE. A material with a high Young's modulus may cause stress-shielding when implanted in bone. Titanium has poor wear characteristics.

3. Polymethylmethacrylate bone cement has a low tensile strength and is weaker than cortical bone in compression. Reduction of voids by vacuum centrifugation and pressurization improves the cement strength and improves bone interlock.

4. Hydroxyapatite. Calcium phosphate ceramics are plasma sprayed on to metal to encourage bone ongrowth and ingrowth.

5. Alumina (Al_2O_3). Alumina ceramic has a small grain size, allowing a smooth fmish which reduces friction. It has the disadvantage of being brittle, and therefore needs to be of relatively large size (28–32 mm diameter) if it is to be strong enough for use as a prosthetic femoral head.

Further reading

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Related topics of interest

Biomechanics (p. 60)
Hip arthroplasty (p. 150)
Total knee arthroplasty (p. 331)

BIOMECHANICS

Biomechanics is the study of forces that act on the living body. *Kinematics* is the study of motion in terms of displacement, velocity and acceleration. *Kinetics* relates the action of forces on bodies to their resulting action. *Kinesiology* is the study of human motion.

Principles

1. Newton's first law. If the net external force on a body is zero then that body will either be at rest or will move uniformly (Ff=0).

2. Newton's second law. The rate of change of momentum of a mass *(m)* is proportional to the force applied (F); F=dmv/df=*ma,* where dmv is the change in momentum (rav) and df is the change in time (f).

3. Newton's third law. For every action there is an equal and opposite reaction.

4. Forces. Forces can be resolved into components, usually vertical (Fv) and horizontal (F/z), using sine and cosine functions. Summation of the components will allow calculation of the resultant force which has an equal and opposite equilibrant force. Useful functions to remember are:

sin 30=cos 60=0.5

sin 45=cos 45=0.7

sin 60=cos 30=0.9 approximately.

5. Moments. The rotational effect of a force (M) is the force (F) times the perpendicular distance from the point of rotation *(d).* Therefore *M=Fd* Nm (Newton-metres).

6. Angular velocity. Angular displacement is denoted by the Greek symbol Θ and is measured in radians. Angular velocity is the rate of change of angular displacement and is measured in radians per second. Angular acceleration is the rate of change of angular velocity, measured in radians per second per second.

7. Work. Work is the force applied to a body to cause motion times the distance moved. *W=Fd* Nm.

8. Energy. Energy is the capacity to perform work (Joules). Kinetic energy is the energy a body has because of its motion KE=l/2mv^2. Potential energy is stored energy that may be used to perform work.

Problems

When faced with a problem of forces acting on the human body, proceed to draw a free body diagram, i.e. the body separated from other bodies and with the forces acting on it. Select a coordinate system for resolving components. Apply Newton's laws, i.e. *EM*=0, *EF*=0 and solve for unknowns. Draw the diagrams for the following situations.

1. The elbow. Assume that the forearm exerts a force of 20 N at a distance of 15 cm from the elbow and that biceps acts at a distance of 5 cm from the elbow. Assume that the elbow is held at 90°. The moments around the elbow must equal zero because of Newton's first law. Therefore the moment exerted by biceps equals the moment exerted by the weight of the forearm. Therefore 20x15=*B*x5, where *B* is the force exerted by biceps. Therefore *B*=60 N. Since the sum of the forces acting at the elbow is also zero, the joint reaction force *(J)* plus the forces exerted by biceps and the weight of the forearm *(W)* are zero, i.e. *J*+*B*+*W*=0 (Newton's third law). Therefore J+60–20=0; J=−40 N.

Assume that the elbow is now held at 120° with the forearm horizontal. The line of action of the biceps is now 30° from the vertical. We must therefore use the vertical component *(B* cosine 30°) in our calculation. EM=0. Therefore 15 cos 30x*W–5*x*B* cos 30=0. *B*=300/5xcos 30=607 cos 30=67 N. EF=0. Assume the joint reaction force, y, acts at an angle of oc degrees to the vertical. We now resolve into vertical and horizontal components. Resolving into vertical components *W*+*B* cos 30+*Jv*=0, where *Jv* is the vertical component of *J*. Therefore *Jv*=20 - 67 x cos 30=-40 N. Resolving into horizontal components *Jh*+*Bh*=0, where *Jh* is the horizontal component of the joint reaction force. Therefore, *Jh*=*B* sin 30=67 sin 30=33 approximately. By Pythagoras' theorem, *J*=square root of sum of the squares of the opposite sides = square root of [40^2+33^2]= 52. The angle a can be derived from tan ot=*JhlJv*=33/40=0.825, etc.

2. The hip. The body weight *(W)* has a line of action through the midpoint of the pelvis, a distance B from the centre of the hip. The abductor muscles *(M)* act at a distance A from the centre of the hip and at an angle of 30° from the vertical. The joint reaction force *(J)* is at an angle of 30° to the vertical. Assume A is 5 and B is 12.5 cm. Forces *M, J,* and *W* can be resolved into horizontal and vertical axis components. *E*Moments=0. Therefore−5*Mv*+*12.5Wv*=0. *Wv*=*W* because the line of action of *W* is vertical. Therefore *Mv*=*2.5W*. *EF*=0. Therefore *Mv*−*W*+*Jv*=0. Since −Mv =2.5 *W,* then −2.5*W*−*W*+*Jv*=*O*.*Jv*=*3.5W*. *Jv*=*J* cos 30. Therefore *J*=*Jv*/cos 30=3.5*W*/0.9=*4W* approximately.

Further reading

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Related topic of interest

Biomaterials (p. 56)

BONE TUMOURS—BENIGN

Benign bone tumours may be classified into tumours of cartilage, fibrous tissue and bone. Like other musculoskeletal tumours they each have characteristic sites of predilection and presentation. Some features, however, pertain to the group as a whole.

Patients may present with pain over the lesion, or complain of a lump or deformity in a bone. The pain may be due to expansion of the lesion or may follow pathological fracture. Alternatively, a lesion may be noted incidentally on a radiograph.

Clinical problems

1. Cartilage tumours. Benign cartilage tumours may arise within bone (enchondromas) or from the surface of a bone (ecchondromas). They may be single or multiple. Solitary tumours arising within the bone are the solitary enchondroma, chondroblastoma and chondromyxoid fibroma. Solitary tumours arising from the surface of the bone are the osteochondroma (ecchondroma or solitary exostosis) and juxtacortical chondroma. Multiple tumours may be found in Ollier's disease (multiple enchondromas), Maffucci's syndrome (multiple enchondromas with soft tissue haemangiomas) and with hereditary multiple exostoses (multiple osteochondromas).

Lesions which are small, peripherally located, painless and single are usually benign. Lesions which are large, central, painful and multiple are more likely to be malignant. The likelihood of malignancy also increases with age.

Radiologically, stippled calcification within the lesion suggests the presence of cartilage. A cold technectium bone scan is a strong indicator of a benign lesion but a hot scan does not exclude it.

2. Fibrous tumours.

- *Unicameral bone cyst (solitary bone cyst).* These are most commonly found in the metaphysis of humerus and femur. Their cause is unknown. They are usually painless unless fracture supervenes. Treatment is by curettage and grafting, although drilling the cyst and injecting steroid is effective in many

smaller lesions. The development of a fracture often leads to spontaneous regression.

- *Aneurysmal bone cyst (ABC).* These can occur in any bone. They commonly involve the metaphyses of long bones and the vertebrae. They are eccentric, expansile, lytic lesions which expand and thin the cortex. The cyst is divided into spaces by a thin meshwork of trabeculae and is filled with blood. Treatment is by meticulous curettage and grafting. Up to 25% may recur.
- *Non-ossifying fibroma (fibrous cortical defect)* is a common fmding in young children but these have usually disappeared by the age of 11. They have a characteristic 'soap-bubble' appearance on X-ray with a well-defined zone of transition. Pathological fracture may occur.
- *Fibrous dysplasia* can affect one (monostotic) or several (polyostotic) bones. Rarely, it may be associated with brownish pigmented lesions on the skin over the site of the bony lesions in women with precocious puberty (Albright's syndrome). Bone is replaced with a benign fibrous stroma containing areas of metaplastic bone. Pathological fracture is common. The radiographic appearance has been described as containing 'bubbles and stripes'.
- *Giant cell tumour* constitutes approximately 5% of all bone tumours. It affects slightly more women than men (3:2) and has its peak incidence in the third decade. The distal femur, proximal tibia, distal radius and proximal humerus are the commonest sites. Patients present with a swelling of one end of a bone. This may be painful, but is invariably so if a pathological fracture is present. Radiographs show an expanding, lytic lesion in the epiphysis and metaphysis which extends to, and can fracture, the subchondral bone. There is a narrow zone of transition. Extension may occur into the soft tissues, or into the adjacent joint, in which case an effusion may be present.

3. Osseous tumours. Osteoid osteoma constitutes about 10% of all benign bone tumours and 4% of all bone tumours. It occurs most commonly in the second decade of life and is rare after the age of 30. Men are twice as likely to be affected as women. The commonest sites are the femur (27%), tibia (22%), posterior elements of the spine (10%), humerus, hand and fibula. The medial side of the femoral neck and the talus seem to be particular sites of predilection.

The presentation is of persistent pain which is worse at night and often completely relieved by salicylates. The diagnosis is often delayed. An osteoid osteoma (or osteoblastoma) in the spine typically presents as a painful scoliosis. There may be swelling and point tendemess if the lesion is on the surface of a subcutaneous bone. Radiographs show a small, round, relatively radiolucent lesion (the nidus), measuring less than 1 cm across, surrounded by dense sclerosis (normal reactive bone). The lesion is usually situated in the cortex of diaphyseal bone but may be subperiosteal or medullary.

Osteoblastoma is a rare tumour constituting only 3% of all benign bone tumours. It occurs at all ages but most commonly in the second or third decades of life.

Men are affected twice as often as women. It has a predilection for long bones, the posterior elements of the spine and the pelvis. It appears histologically similar to an osteoid osteoma but is usually larger (>1 cm) and can be locally aggressive. Pain is constant during the day but is less at night. It is less well relieved by salicylates and NSAIDs than osteoid osteoma. Radiographs usually show an eccentric lesion in the diaphysis or metaphysis of a long bone. These may reach a considerable size. There is surrounding sclerosis, but its density is variable.

Investigation

1. Plain radiographs. The plain radiograph remains the most useful single investigation. The following points should be appreciated in patients with a suspected benign bone tumour: the site of the lesion *within the skeleton* (appendicular/axial) and particularly if it is single or multiple; the site of the lesion *within the affected bone* and whether it is centrally or peripherally placed: a benign expanding lesion within the epiphysis is likely to be a chondroblastoma if the physis is open and a giant cell tumour if the physis is closed. The metaphysis of a bone can be involved in almost any tumour process: the diaphysis is a site of predilection for fibrous dysplasia, adamantinoma, histiocytosis (and Ewing's sarcoma). The *zone of transition* between tumour and bone should be narrow if the lesion is benign. This indicates that bone is forming in response to the tumour and is not being overwhelmed by it. Most benign tumours expand and thin the cortex of the bone without destroying it (ABC, enchondroma, fibrous dysplasia). There should be no soft tissue mass.

2. Scintigraphy. This is useful to delineate the extent of a benign lesion throughout the skeleton where these may be multiple (e.g. fibrous dysplasia, hereditary multiple exostoses, enchondromas (Ollier's disease), haemangioma, histiocytosis).

3. CT and MRI. CT is the best investigation for imaging cortical bone for reactive margination, fracture or permeation. MRI is preferred for defming the intramedullary spread of a tumour. It tends to overestimate the boundary of a tumour because of surrounding oedema.

4. Surgical staging. Enneking has grouped benign bone tumours into three surgical stages, latent, active and aggressive, based on their radiological appearance and clinical behaviour. Latent tumours cause no symptoms and exhibit no signs of aggression. At the other end of the scale, aggressive tumours may cause pain, enlarge more rapidly than latent or active tumours, penetrate the reactive bone around them and sometimes spread into surrounding soft tissues. The distinction between benign and malignant behaviour becomes blurred at this stage, and on occasion these tumours may metastasize.

5. Biopsy. The principles of biopsy for benign tumours are the same as those for malignant tumours (see above).

Management

Certain benign tumours require definitive surgical treatment.

1. Cartilage tumours. The indications for surgery are:

(a) to prevent pathological fracture,
(b) to remove a mass which is causing deformity of a bone or pressure effects, and
(c) where there is the possibility of malignancy.

Cartilage tumours should be completely excised if this will not cause significant morbidity. Intraosseous lesions may be curetted, but this must be done thoroughly with adjunctive cryosurgery or phenolization. Defects may be grafted, but may make the subsequent identification of a recurrence more difficult.

2. Giant cell tumour is most commonly treated by *curettage and grafting.* The recurrence rate after this procedure may be as high as 60% so various adjunctive procedures have been described. *Radical curettage,* drilling away the walls of the lesion with or without grafting, reduces the recurrence rate to around 15–20%.

Packing the cavity with polymethylmethacrylate (PMMA) cement after curettage is favoured in some centres. It is suggested that either the toxic effect of the monomer or the thermal effect of curing cement kills off residual cells at the margins of the cavity, resulting in a lower rate of recurrence. *Cryosurgery* has also been used, with similar claims. *Complete excision* with reconstruction of the skeleton has been advocated, but this is extensive surgery for an essentially benign condition. Occasionally, amputation is required for recurrence. Approximately 1 % of these tumours will metastasize, usually after surgery and to the lung where they can be successfully treated by local excision.

3. Osseous tumours. Treatment is by surgical excision, using intraoperative radiographs or radionuclide imaging and a hand-held detector. Only the nidus need be excised. It is usually bright red, in contrast to the ivory-like reactive bone surrounding it. Complete excision abolishes the pain. If pain persists, an incomplete excision should be suspected. True recurrence is rare.

Further reading

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Related topics of interest

Bone tumours—malignant (p. 68)
Bony metastases (p. 74)
Soft tissue sarcomas (p. 293)

BONE TUMOURS—MALIGNANT

Primary malignant bone tumours are rare and account for less than 1% of all deaths from malignant disease. Accurate estimates of their incidence are difficult to collect but it appears that there are no more than 350 new cases per year in the United Kingdom. The approximate incidence of the commonest bone tumours is osteosarcoma: 3/million/year, chondrosarcoma 1.5/million/year, Ewing's sarcoma 1/million/year and malignant fibrous histicytoma 0.5/million/year. Metastatic disease therefore remains by far the commonest malignant process to be found in bone.

The aetiology of bone sarcomas is unclear. Paget's disease predisposes to a highly malignant form of osteosarcoma in the elderly. Some benign cartilaginous tumours may undergo malignant transformation. Osteosarcomas may arise at the site of previous irradiation, particularly for giant cell tumour. Malignant fibrous histiocytoma and fibrosarcoma are seen in relation to bone infarcts and fibrous dysplasia. A specific gene translocation has recently been identified in cases of Ewing's sarcoma.

The most common presenting features are pain and swelling. The pain is non-mechanical and worsens with time. Commonly, the tumour arises adjacent to a joint and may restrict its movement. An effusion suggests that the tumour is very close to, or is involving, the joint. Pathological fracture is unusual. Tumours arising in the axial skeleton and enlarging into a body cavity typically present late. In most cases, plain radiographs will confirm the diagnosis.

The differential diagnosis is principally from metastatic disease, a benign tumour of bone, osteomyelitis, lymphoma or multiple myeloma, a simple cyst, fibrous dysplasia, bone infarcts, stress fractures and callus formation, eosinophilic granuloma, the 'brown tumours' of hyperparathyroidism and Paget's disease.

Clinical problems

1. Osteosarcoma is the commonest primary malignant bone tumour and accounts for about 20% of all bone sarcomas. It is a malignant tumour with a spindle-cell stroma which produces osteoid or immature bone. It can occur at all ages but is commonest in the second decade of life. It affects slightly more males than

females. The commonest sites are the distal femoral (25%) and proximal tibial (15%) metaphyses, followed by the proximal humerus (8%) and proximal femur (7%). Nearly 90% of all upper limb osteosarcomas are situated in the proximal humerus. Most present with pain and a bony mass. Occasionally, they may fracture. The serum alkaline phosphatase may be elevated.

The radiographic features of classical osteosarcoma are of a sclerotic intramedullary lesion of the metaphysis which expands and destroys bone. There is a wide zone of transition. More than 90% have penetrated the cortex of the bone at the time of diagnosis. Radial streaking of the extraosseous component has been described as a 'sunburst appearance'. Periosteal elevation at its edges provokes reactive bone formation (Codman's triangle).

There are several variants, of which the commonest are parosteal and telangiectatic osteosarcoma. Parosteal osteosarcoma arises from the outer surface of the bone and tend to be sessile and densely sclerotic. Telangiectatic osteosarcoma is rapidly growing and highly destructive, and has a poor prognosis.

The disease-free survival rate for all osteosarcomas improved rapidly in the 1980s from 25% at 5 years to just over 70%. This was directly related to the introduction of effective neoadjuvant chemotherapy and, to a lesser extent, the resection of lung metastases.

2. Chondrosarcoma is the second commonest tumour of bone (excluding myeloma) after osteosarcoma. It affects all age groups from the second decade onwards but is commonest between the ages of 35 and 55. It affects both sexes equally. The commonest sites are the pelvis (30%), proximal femur (15%), ribs (11%), shoulder girdle and around the knee.

Radiologically, the tumour may have a lobulated appearance and contain regions of different radiodensity. It may thin, expand and destroy bone or may cause thickening of the cortex. Typically, there is endochondral calcification.

Histologically, chondrosarcoma has to exhibit the organization and appearance of cartilage, to contain malignant cells that appear to be chondral in origin and to have a matrix that resembles that of normal cartilage. It may be difficult to distinguish from a benign lesion as mitoses are scanty in the lower-grade lesions. There are (at least) four distinct types: classical, mesenchymal, clear-cell and dedifferentiated. Each behaves somewhat differently. In broad terms, mesenchymal and dedifferentiated chondrosarcomas are highly malignant with a poor prognosis, while clear-cell chondrosarcoma is rather more benign. The classical chondrosarcoma can be of any grade but is usually towards the lower end of the spectrum.

Prognosis is directly related to the surgical staging. For a IA tumour, this approaches 100% at 5 years whereas the disease-free 5-year survival for a IIB tumour may be as low as 30% for tumours in unfavourable sites. Chondrosarcoma is not particularly chemosensitive or radiosensitive. High-dose methotrexate or adriamycin are sometimes used for high-grade tumours and radiotherapy for surgically inaccessible lesions.

3. Ewing's sarcoma (small round cell tumour of childhood). Ewing's sarcoma constitutes about 5% of all malignant bone tumours. It is commonest in children (age 5–20) although older patients may be affected. It has a slight male predominance. Its commonest sites are the pelvis and bones of the lower limbs, followed by the bones of the shoulder girdle and ribs. In long bones it tends to be diaphyseal, although the metaphysis of the proximal femur is a favoured site. Pain and swelling are accompanied by signs of a mild febrile illness. Ewing's frequently has to be distinguished both clinically and radiologically from a low-grade osteomyelitis. The usual radiographic appearance is of a diffuse moth-eaten lesion with a wide zone of transition. It may resemble an osteosarcoma with periosteal elevation and Codman's triangles.

Ewing's tumour is very radiosensitive. Radiotherapy used to be the mainstay of treatment but was accompanied, as might be expected in children, with severe local complications and long-term disability. More recently, surgical resection, adjunctive chemotherapy and interval resection of the primary lesion after chemotherapy and local radiotherapy have been advocated. It is not yet clear whether these offer significant long term benefits. The prognosis overall has improved from a 15% 5-year survival to around 50%.

4. Malignant fibrous histiocytoma (MFH) is, in histological terms, a relatively recent tumour. The diagnosis has only been made since the mid-1970s when it became possible to distinguish it from fibrosarcoma. Both are rare. Clinically and radiologically, MFH is very similar to any other stage II lesion of bone. It is seen most commonly in the distal femur, proximal femur, proximal tibia and proximal humerus. It tends to occur at an older age and often develops in or around old bony infarcts, fibrous dysplasia and Paget's disease. Unlike other sarcomas it may metastasize early to regional lymph nodes. It is characterized histologically by a storiform pattern of spindle-shaped fibroblast-like cells some of which contain lipid giving them a 'foamy' appearance. It has a poorer prognosis than other fibrosarcomas and most require radical amputation.

Management

1. Staging investigations. Staging should be carried out prior to biopsy. The aim is to determine the local extent and distant spread of the tumour. Local extent is best demonstrated by magnetic resonance imaging, although CT is also helpful. These should image the whole of the involved bone or bones to exclude 'skip' lesions and the adjacent soft tissues to demonstrate the extent of the extraosseous component of the tumour. Angiography is seldom needed. Distant spread is assessed using bone scintigraphy, plain radiography (PA and lateral) of the chest and CT of the lungs. A full blood count, plasma viscosity and biochemical screen should also be obtained.

2. Biopsy. Biopsy should be carried out by the surgeon who is going to undertake the definitive management of the patient. The aim is to acquire sufficient representative material to enable an experienced bone tumour

pathologist to make an accurate diagnosis. In many cases this may be obtained percutaneously using a Jamshidi or Tru-Cut biopsy needle but on occasion an open biopsy is indicated. Whichever method is used, the biopsy site must be placed along the line planned for the defmitive excision so that its track can be excised *en bloc* with the tumour. Open biopsy incisions should be placed longitudinally in the limbs. Dissection should be minimal and haemostasis meticulous to avoid contamination of adjacent tissue planes. The extraosseous portion of a bone tumour is most likely to yield active tumour and may not need decalcification. Incisional biopsy is favoured. If the cortex has to be breached, it should be sealed with a plug of methylmethacrylate cement provided that infection has been excluded. Any drain should be placed close to the incision so that its track may be excised with the tumour.

3. Surgical staging. After these investigations it is possible to stage the tumour accurately using the system devised by Enneking. This combines the tumour grade (I=low grade: II = high grade: III=low or high grade with metastases) and site (A=intracompartmental: B=extracompartmental) to give an alphanumeric descriptor which has been shown to correlate well with prognosis for the commonest types of bone tumour. For example, an osteosarcoma which has broken out of a long bone into the surrounding soft tissues but had not metastasized would be staged as a IIB tumour.

4. Adjunctive therapy has been discussed in relation to each of the tumours described. Neoadjuvant chemotherapy is an essential part of the modern management of osteosarcoma. Most regimens are based on high-dose methotrexate. All consenting patients should be entered into the current MRC osteosarcoma trial. Chemotherapy and radiotherapy have been used to treat most other bone sarcomas with varying results.

5. Resection. In all sarcoma resection, the margin of the excision determines the success of surgery, whether that surgery is ablative (amputation) or local (limb salvage). The margin is described as *intralesional* if an incomplete excision has been carried out and frank tumour has been left behind. A *marginal* margin indicates that dissection has been carried out through the pseudocapsule of the tumour (‘shelling out’ of a tumour). Tumour cells will almost certainly remain in the surrounding tissue and ‘skip’ lesions in the compartment. A *wide* margin excises the whole tumour with a surrounding layer of normal soft tissue but may leave ‘skip’ lesions within the affected compartment. A *radical* margin means the excision of the whole compartment or compartments in which the tumour arises.

The choice of margin is principally determined by the surgical staging: occasionally a palliative procedure may be indicated. A wide margin is needed for the control of stage I disease but will be insufficient for stage II disease unless satisfactory adjunctive therapy is available to deal with the ‘skip’ lesions. A radical margin is compatible with a local excision in some cases and in these will be as effective as radical amputation. Both wide and radical local excisions

demand the *en bloc* excision of the biopsy track and any other contaminated tissues.

6. Reconstruction. A local excision leaves a large defect in the skeleton. In a long bone the defect may be diaphyseal or osteoarticular. There are a variety of ways in which these can be reconstructed. A *diaphyseal defect* can be grafted using an avascular or vascularized autograft or a segmental allograft. Alternatively, a modular or custom-made endoprosthetic replacement may be used. Early reports of bone transfer using the Ilizarov apparatus suggest that this may be possible in highly selected cases. Rarely, a small diaphyseal defect can be treated by limb shortening. An *osteoarticular defect* can be treated by shortening and arthrodesis, arthrodesis using autogenous fibular strut grafts, modular or custom-made endoprosthetic replacement or osteoarticular allografting, with or without replacement of the joint surface with a standard prosthesis. Rarely, an excision arthroplasty is appropriate. *Defects in the pelvis and axial spine* may also be reconstructed using variants on the methods described above.

The choice of method depends on the age, activity and life expectancy of the patient, the type of tumour, the need for adjunctive therapy, the availability of prostheses and allografts and the individual experience of the operating surgeon.

Complications

Almost any complication can and has occurred in the treatment of patients with malignant bone tumours. Failure to control the disease by resection and adjunctive therapies can lead to local recurrence, metastasis and death while the complications of allograft and endoprosthetic replacement of large bone defects carry with them all the problems of joint replacement, but on a larger scale and with greater significance to life and limb. To give one example, the overall infection rate for allografts and EPRs is around 7% in centres of excellence.

For the general orthopaedic surgeon, inadvertent and inappropriate biopsy and the histological diagnosis of a primary bone tumour will cause most problems. Inappropriate placement of the biopsy site may make its subsequent *en bloc* excision difficult or impossible. If the biopsy specimen is inadequate or unrepresentative an accurate diagnosis will be impossible. The biopsy will then have to be repeated. Complications of bone biopsy include fracture, haematoma formation and infection. Each affects the prognosis adversely.

Further reading

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Related topics of interest

Allografts and bone banking (p. 12)
Bone tumours—benign (p. 63)
Bony metastases (p. 74)
Musculoskeletal imaging (p. 190)
Soft tissue sarcomas (p. 293)
Spinal tumours (p. 310)

BONY METASTASES

Metastatic deposits are the commonest malignancy of bone. They arise commonly from tumours of the breast, bronchus, thyroid, prostate and kidney. Less commonly, tumours of the skin (especially melanoma), oesophagus, colon and rectum, stomach and cervix will metastasize to bone: the spine, pelvis, femora, ribs and skull are most often involved. The overall incidence of metastasis in patients with primary tumours is not certain: it is related to the care with which post-mortem examination is carried out. It may be as high as 70–80%. A solitary deposit is present in only 9% of patients.

The mechanism by which tumours metastasize is not clear. It has been suggested that it is a three-phase process: the tumour cells have to break through the basement membrane, lyse the underlying stroma with proteases, and then move through the stroma until they encounter lymph and capillary channels. Once in the bloodstream, their presence on the venous side of the circulation of the liver and lungs, allied to their abundant capillary networks, make them prime targets for the growth of metastatic deposits. Spread to the spine has been explained by Batson, who identified a valveless system of veins linking the viscera of the abdominal and thoracic cavities with the vertebral veins (Batson's plexus). Metastases may cause local osteolysis or may excite an osteoblastic reaction in the surrounding bone: the metastases themselves are not histologically distinct.

Clinical problems

Any patient with a history of primary malignancy who presents with *bone pain,* particularly if accompanied by local tenderness, should be suspected of having a metastatic deposit at the site of the pain, especially if this is in the thoracic or lumbar spine. *Vertebral fracture* with collapse may occur. The extension of tumour into the spinal canal, a pathological burst fracture of the vertebral body, or direct invasion of the neural elements may cause a *myelopathy* or *radiculopathy.*

Pathological long bone fractures particularly affect the femur and humerus. Pain and local tenderness in relation to either of these should be investigated with plain radiographs so that incipient fractures may be identified and treated prophylactically.

Rarely, patients present with *hypercalcaemia* or the effects of *inappropriate hormone secretion* by the tumour mass.

Investigation

1. Plain radiography may show no abnormality. The vertebral body must lose 50% of its mass before a deposit can be discerned and the figure is similar for medullary bone. A typical osteolytic deposit destroys rather than expands bone (although thyroid cancer and adenocarcinoma of the kidney are notable exceptions), has an irregular margin with a poorly defined zone of transition and little soft tissue reaction. Pathological fracture may be seen. Osteoblastic deposits are most commonly seen with tumours of the prostate and breast, but are by no means confmed to these. Single lesions may be difficult to distinguish from primary bone tumours, particularly if there is no history of malignancy. Disease in patients over the age of 40 is more likely to be metastatic.

2. Laboratory investigations may be normal. The *ESR or plasma viscosity* may be elevated. *Hypercalcaemia* is present in 10%. It is indicative of extensive bone destruction or parathormone production by the tumour. The prognosis is usually poor. *Serum alkaline phosphatase* levels are proportional to the amount of bone regeneration taking place. *Serum acid phosphatase* levels are raised in metastatic prostatic carcinoma.

3. Scintigraphy will show the pattern of metastatic disease in a series of 'hot spots' unless the primary disorder is myeloproliferative. Further radiographs may be taken on the basis of the scintigram to demonstrate the most suitable site for biopsy.

4. MRI is the most sensitive method of demonstrating spinal metastases. It can also distinguish between osteoporotic and metastatic collapse in the same spine.

5. Biopsy should be carried out if there is any doubt that the skeletal lesion represents metastatic spread from a primary tumour of known histology. In practice, therefore, biopsy is almost always indicated. If the lesion is solitary and excision is considered, the guidelines given for biopsy of a primary bone tumour apply. Mostly, needle biopsy from the most accessible lesion is appropriate.

Management

1. Alleviation of symptoms. Pain control is essential. Analgesics and NSAIDs in combination with radiotherapy and fracture stabilization are usually effective. Occasionally more aggressive measures, such as cordotomy or amputation, are indicated for intractable pain. Care should also be paid to the patient's general condition: nutrition, hydration and metabolic balance should be carefully regulated.

2. Treatment of the metastatic process is usually undertaken in conjunction with medical oncologists and radiotherapists. A variety of techniques are

available depending on the nature of the tumour. Hormonal manipulation, chemotherapeutic agents and steroids are commonly used.

3. Spinal decompression. Vertebral compression fractures may be treated conservatively with radiotherapy and hormonal manipulation provided that the primary tumour is sensitive and that maximal irradiation has not been given to the spine on a previous occasion. If the cord is compressed by radioresistant tumour or if collapse of the vertebral body causes intractable pain, anterior vertebrectomy and stabilization should be carried out. Posterior decompression is to be condemned except in those rare cases of isolated posterior element involvement. Good primary stability can be achieved anteriorly using one of the anterior spinal fixations systems in conjunction with polymethylmethacrylate (PMMA), corticocancellous bone graft, an anterior jack or a titanium cage filled with cancellous bone graft.

4. Stabilization of pathological fractures. Metastatic lesions of long weight-bearing bones which destroy more than half the width of the cortex are at significant risk of fracture and should probably be stabilized prophylactically with an interlocked nail, using adjunctive PMMA cement augmentation if there is no chance that the lesion will heal spontaneously. Treatment is technically easier at this stage and the procedure may be carried out electively. It also prevents the pain of fracture and the ensuing distress to the patient. If the patient presents with an existing fracture, diaphyseal and metaphyseal fractures are stabilized with interlocked or reconstruction nails, fractures of the humeral or femoral heads can be treated by excision and replacement using standard prostheses while more extensive osteoarticular lesions may require the use of custom-made prostheses. Radiotherapy will usually be used adjunctively.

5. Excision of isolated lesions. In selected cases, excision of an isolated metastasis followed by reconstruction of the skeleton is appropriate. The best example is an isolated deposit from an adenocarcinoma of the kidney or thyroid affecting the proximal end of the femur. Wide local excision and reconstruction with a custom-made prosthesis eradicates what may be the only remaining tumour mass, while the use of an endoprosthetic replacement allows radiotherapy to continue and offers prolonged prosthesis survival if the patient remains well.

6. Radiotherapy is the mainstay of treatment for most of the common tumours that spread to bone. Between 80 and 85% will respond to a fractionated dose of between 30 and 50 Gy. Radiotherapy can relieve pain, lessen disability, reduce the growth of the metastatic tumour and decrease the risk of pathological fracture.

Further reading

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Related topics of interest

Bone tumours—malignant (p. 68)
Low back pain (p. 181)
Spinal tumours (p. 310)
Thoracic back pain (p. 320)

CARPAL INSTABILITY

The stability of the wrist depends on the contours of its constituent bones and on a system of extrinsic and intrinsic ligaments. The palmar radiocapitate, radioscapholunate and radiolunate ligaments are the most important stabilizers on the palmar aspect of the wrist, whereas the dorsal radiocarpal and intercarpal ligaments and the ligaments of the ulnocarpal space are the most important on the dorsal aspect. The carpus may be considered as an oval ring system in which the proximal and distal rows are joined on the radial side by the scaphoid and on the ulnar side by the triquetrohamate joint.

The wrist may also be thought of as having three longitudinal columns which transmit force from the hand to the forearm. The *radial* column consists of the radius, lunate, proximal two-thirds of the scaphoid, capitate, trapezoid and the second and third metacarpals. The *ulnar* column consists of the ulna, ulnocarpal complex, triquetrum, hamate and the bases of the fourth and fifth metacarpals. The *thumb* column consists of the base of the first metacarpal, the trapeziotrapezoid joint and the distal third of the scaphoid.

Carpal instability is defmed as a loss of normal alignment of the carpal bones caused by carpal injury or disease. It may be acute or chronic.

Clinical problems

1. Scapholunate dissociation. A fall on the supinated dorsiflexed wrist may result in scapholunate dissociation (rupture of the scapholunate and radioscapholunate ligaments) or a scaphoid fracture. In SL dissociation there is widening of the gap between the scaphoid and the lunate beyond the normal 2–3 mm, producing the 'Terry-Thomas' sign, best shown on a clenched-fist supinated AP radiograph. In the normal wrist, radial deviation causes the scaphoid to palmarflex with the lunate and the rest of the proximal row. SL dissociation results in palmarflexion of the scaphoid with dorsal angulation of the lunate. The 'cortical ring sign' seen on the PA radiograph is an end-on view of the scaphoid resulting from this palmar flexion: the lunate appears quadrilateral because it is dorsiflexed. On the lateral radiograph the lunate is dorsally angulated and the scaphoid is palmarflexed such that the scapholunate angle on the lateral radiograph exceeds the normal 30–60°. The capitate is therefore displaced dorsal

to the long axis of the radius with the lunate as an intercalated segment (dorsal intercalated segment instability or DISI).

2. Triquetrolunate (TL) dissociation. A fall which loads the ulnar side of the wrist with the carpus pronated may result in an injury of the triquetrolunate joint. In the normal wrist, ulnar deviation results in dorsiflexion and distal migration of the triquetral with dorsiflexion of the lunate. In TL dissociation the lunate assumes a palmarflexed position. On the PA radiograph the scaphoid appears foreshortened and palmarflexed and the cortical ring sign is present. The palmarflexed lunate appears triangular. On the lateral radiograph the lunate and scaphoid are palmarflexed so that the scapholunate angle is less than the normal 30–60°. The capitate lies to the palmar side of the long axis of the radius with the lunate as an intercalated segment (volar intercalated segment instability or VISI).

3. Ulnar translocation. This is rarely caused by injury and usually follows rheumatoid arthritis. The carpo-ulnar distance is reduced, i.e. the ratio of the distance between the head of the capitate and the long axis of the ulna divided by the length of the third metacarpal is less than 0.3.

4. Perilunate instability. Cadaver studies suggest that there are four phases of perilunate instability when the wrist is progressively loaded in extension, ulnar deviation and supination. Stage I is scapholunate diastasis, stage II is dorsal dislocation of the capitate (perilunate dislocation), stage III is triquetrolunate dissociation and stage IV is lunate dislocation.

5. Dynamic instability. This exists when routine radiographs are normal but the patient is able to produce the radiographic signs of instability by holding the wrist in an abnormal position.

6. Carpal instability non-dissociative (CIND). DISI and VISI may be seen in the absence of ligamentous injury to the proximal carpal row if the midcarpal joint has been injured or if the ligaments of the wrist are lax. A malunited fracture of the radius commonly results in dorsal subluxation of the carpus and instability.

Management

1. Clinical diagnosis. The patient normally complains of pain, weakness, instability and clicks or snaps. Localized tenderness must be elicited and clicks demonstrated. Manipulation of the scaphoid may reproduce the feeling of instability. Standard radiographs including the clenched-fist view are undertaken and additional radiographs are taken in positions of instability if necessary. A bone scan may be positive in doubtful cases. Arthrography with fluoroscopy may show ligamentous tears and abnormal carpal movements. Magnetic resonance imaging may be useful.

2. Treatment. This is controversial. For acute SL dissociation consider either manipulation under anaesthetic (MUA) and percutaneous pinning with plaster of Paris (POP) immobilization or operative repair of the ligaments. For chronic SL dissociation consider soft tissue reconstruction or scapho-trapezio-trapezoid

fusion. An acute TL dissociation may be treated by POP immobilization or operative repair. For chronic cases consider reconstruction, ulnar shortening if there is ulnar plus deformity, or TL arthrodesis. For ulnar translocation, relocation and radiolunate arthrodesis is probably the best option. For dynamic instability a trial of conservative measures is reasonable before undertaking operative reconstruction or arthrodesis.

Further reading

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Related topics of interest

Arthritis of the wrist and first carpometacarpal joint (p. 46)
Wrist pain (p. 337)

CHRONIC KNEE INSTABILITY

Chronic knee instability may occur as a result of undiagnosed acute injury, inadequate treatment or repeated trauma. Stability depends on the cruciate and collateral ligaments (primary stabilizers) and the extrinsic muscles (secondary stabilizers). Some individuals are less dependent on the primary stabilizers than others. Disruption of the complex supporting structures may produce either simple one-plane instability or more complex rotational instability. These can be classified according to the direction of instability noted on stress testing (e.g. anterior draw, pivot shift, etc.).

Clinical problems

Unidirectional instability

1. Medial instability in full extension. The knee opens on the medial side with valgus stress. This occurs with disruption of the medial collateral ligament, the medial capsular ligament, the anterior cruciate ligament (ACL), the posterior oblique ligament and the posterior capsule. Usually the posterior cruciate ligament is also disrupted.

2. Lateral instability in full extension. The knee opens in extension with varus stress due to disruption of the lateral capsular ligament, the lateral collateral ligament, the biceps tendon, the ilio-tibial band, the arcuate popliteus complex, the ACL and the posterior cruciate ligament (PCL).

3. Posterior instability. When the tibia moves posteriorly on the femur during the posterior drawer test or there is a posterior sag at rest there is disruption of the posterior cruciate ligament, the arcuate complex and the posterior oblique ligament.

4. Anterior instability. When the tibia moves forward on the femur during the anterior drawer test in neutral there is disruption of the anterior cruciate ligament, the lateral capsular ligament and the medial capsular ligament. This instability disappears with internal rotation of the tibia as the posterior cruciate ligament is tightened.

Rotatory instability

1. Antero-medial rotatory instability. On stress testing, the medial plateau of the tibia rotates anteriorly as the joint opens on the medial side. This occurs when the medial capsular ligament, the medial collateral ligament, posterior oblique ligament and the ACL are disrupted.

2. Antero-lateral rotatory instability. The lateral tibial plateau rotates forward in relation to the femur in 90° of flexion with lateral opening of the joint line. This occurs when the lateral capsular ligament, the arcuate ligament and the anterior cruciate ligament are disrupted. The jerk test and the pivot shift test are both positive.

3. Postero-lateral rotatory instability. The lateral tibial plateau rotates posteriorly on the femur with lateral opening of the joint space with the reverse pivot shift test. This occurs when the popliteus tendon, the arcuate ligament, the lateral capsular ligament and the PCL are deficient.

4. Postero-medial rotatory instability. This may occur with a hyperextension valgus force which tears the medial collateral ligament, the medial capsular ligament, the posterior oblique ligament, the ACL and the posterior capsule. The medial tibial plateau rotates posteriorly on the femur and opens medially with stress testing. The intact posterior cruciate is the axis of rotation.

Combined instability

Most severe rotatory instabilities are also accompanied by some degree of varus or valgus instability.

1. Antero-lateral-antero-medial rotatory instability. This is the most common combined instability: the anterior drawer test is markedly positive.

2. Antero-lateral-postero-lateral rotatory instability. Varus instability is marked, with disruption of all structures on the lateral side of the knee as well as the ACL.

3. Antero-medial and postero-medial rotatory instability occurs when all medial structures including the semimembranosus complex are disrupted in addition to the anterior and posterior cruciates.

Management

1. Clinical assessment. Clinical examination, often under general anaesthetic, is essential. *Arthroscopy* can assess the state of the articular surfaces and menisci and may confirm cruciate ligament injuries. *Plain radiographs* will exclude bony abnormality, e.g. an old tibial plateau fracture or ACL avulsion. *MRI* can be used as a non-invasive method for assessing the structures seen at arthroscopy.

2. Physiotherapy. Exercises to strengthen the quadriceps, hamstrings and gastrocnemius muscles may overcome a functional instability. If the knee recovers, dynamic stability surgery may be unnecessary.

3. Bracing. Instability associated with sport may be controlled by bracing.

4. Surgery. A complete examination and understanding of instability is required before reconstruction is undertaken, otherwise inappropriate or inadequate surgery may be performed.

- *Chronic medial instability.* Direct repair of medial structures may be possible. If direct repair is impossible, reconstruction with part of semimembranosus may be required. Provided that lateral and posterior structures are intact, a pes anserinus transfer will improve anteromedial instability.
- *Chronic lateral instability.* Less frequently requires repair than medial side. Lateral instabilities are rarely simple and usually include a rotatory instability.
- *Antero-lateral rotatory instability* may be stabilized using intra- or extra-articular procedures. *Extra-articular procedures* using an ilio-tibial band are divided into static check-rein techniques (e.g. Mackintosh, Muller) and dynamic procedures (e.g. Ellison). *Intra-articular procedures* using bone-tendon-bone free patellar tendon graft are currently popular. These may use autogenous tissues or allografts and may be undertaken as an open procedure or using the arthroscope. Tunnels are drilled in the tibia and femur using a jig which aligns the graft anatomically. Alternative methods use the semitendinosus double loop graft or a Mackintosh 'over-the-top' reconstruction in which a lateral strip of quadriceps and patellar tendon is passed through the intercondylar notch around the lateral femoral condyle and secured to the lateral epicondyle. Synthetic ligament reconstruction using carbon fibre, or Dacron is less successful than autogenous grafts but can be used for salvage procedures.
- *Postero-lateral rotatory instability.* There are two principal components: *repair of the postero-lateral complex,* using ilio-tibial band transfer to reconstruct the popliteus, biceps tendon transfer to restore the arcuate ligament and lateral gastrocnemius advancement, and *PCL reconstruction*. PCL reconstruction is only considered in cases of severe functional disability or early progressive degenerative change. Results are less predictable than ACL reconstruction. The techniques available are derived from those used in anterior cruciate surgery. Bony avulsions are reattached. Intrasubstance ruptures are either treated by transfer of one-third of the medial head of gastrocnemius through the medial femoral condyle via the intercondylar notch or using a bone-patellar tendon-bone free tendon graft. Reconstruction by tunnelling the medial hamstrings (semitendinosus or gracilis) has also been used.

Complications

Untreated chronic knee instability may progress and cause meniscal degeneration, meniscal tears and subsequent early degenerative change. It is not currently known how reconstruction affects this cycle.

Further reading

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Related topics of interest

Arthritis of the knee (p. 39)
Arthroscopy (p. 49)

CLUBFOOT

Congenital talipes equinovarus (CTEV) is a hereditary condition of polygenic inheritance to which environmental factors are added *in utero,* resulting in arrested development. It occurs in 1 in 1000 live births and is commoner in males (2:1). There is a flexion deformity at the ankle which exposes the antero-lateral dome of the talus, and medial and downward subluxation of the navicular which exposes the lateral neck of the talus. The medial and plantar fibro-elastic structures consequently shorten. There may be associated deformity of the talus (less often the calcaneum) with hypoplasia of the calf muscles. Clubfeet are classed as 'easy', when the deformity is less severe, is correctable in the same way as 'postural clubfoot' (see Congenital foot deformities, p. 88) and does not recur, or 'resistant' which needs protracted treatment and never returns to normal. Other associated congenital abnormalities are seen in 10–15% of patients with CTEV.

Management

1. Clinical examination. This reveals a characteristic picture with abnormal skin creases. Associated abnormalities (e.g. myelomeningocele, intraspinal tumour, diastematomyelia, polio, cerebral palsy) or any associated developmental syndrome (e.g. arthrogryposis, diastrophic dwarfism) must be identified.

2. Radiographs. Antero-posterior (AP) and lateral radiographs are required in a maximally corrected position. Radiographs of the other foot are obtained for comparison. The talo-calcaneal angles are measured. In the lateral projection the angle is normally 35–50°: in CTEV it is less than 25° and may have a negative value. In the AP projection of a normal foot the long axis of the talus points medially towards the first metatarsal and the long axis of the calcaneum points laterally towards the fifth metatarsal forming a 'V' of 20–40°. In CTEV this angle approaches zero as both axes tend to point towards the fourth and fifth metatarsals.

3. Non-operative treatment. The treatment of clubfoot begins at birth. The aim is to achieve a plantigrade, pliable, cosmetically acceptable, pain-free foot with as minimal a disruption to life as possible. Initially, it cannot be determined

which feet will respond to serial splintage and which will need surgery. Relapse can occur at any stage up to skeletal maturity.

Gentle *manipulation and splintage* with serial above-knee plasters or strapping (Robert-Jones technique) is carried out at weekly intervals for 6–8 weeks, and fortnightly thereafter. The principle of strapping is to produce an eversion force at the ankle from active knee movement. If a full correction has not been obtained by the age of 3 months, surgery is considered. If full correction has been obtained, splintage is maintained until the age of 6 months when a Dennis Browne bar is worn until walking and at night.

4. Operative treatment. Soft tissue release is carried out as soon as is technically feasible and safe (usually 6 months to 1 year). Bony procedures are in general reserved for the older child. The rationale for soft tissue release is that realignment of the talus, calcaneum and navicular allows remodelling of the articular surfaces. Posterior release is required if only forefoot adduction and heel varus have been corrected. Postero-medial release (Turco) or a circumferential release (McKay) is otherwise necessary. *Tendon transfer* is not routinely used as primary treatment but can be used to correct supination of the forefoot (e.g. tibialis anterior transfer to lateral cuneiform or fifth metatarsal). *Osteotomy* is required in residual or relapsed deformity in children over 6 years old. This can be, for example, an opening wedge osteotomy of the calcaneum to correct varus (Dwyer), a laterally based closing wedge osteotomy (Toohey/Campbell) or a closing wedge resection of the calcaneocuboid joint (Dillwyn Evans). *Triple arthrodesis* may be required as a salvage procedure for a stiff painful foot but is not performed before a skeletal age of 12 years as pseudarthrosis rates are high before this age. Also, unacceptable foot shortening is avoided. Correction of deformity using the *Ilizarov technique* may be useful in a resistant clubfoot.

Complications

Breaking of the foot can occur from excessive manipulation creating a ‘rocker-bottom’ deformity. Surgery may cause wound problems with scarring and stiffness. Severe complications can result in necrosis and even amputation. Acute hypertension has been recorded following surgical correction.

Further reading

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Related topics of interest

Congenital foot deformities (p. 88)
Flatfoot (p. 127)
Hindfoot arthrodesis and osteotomy (p. 147)
Neuromuscular disorders (p. 195)

CONGENITAL FOOT DEFORMITIES

Congenital foot deformities are either postural deformities or malformations. Postural deformities occur in 2% of live births and are intra-uterine distortions of a normally formed limb. These usually respond to non-operative treatment and often correct spontaneously. Malformations are defects of limb organogenesis and are more resistant to treatment.

Clinical problems

Postural deformities

1. Talipes calcaneo-valgus. The entire foot assumes a position of dorsiflexion and eversion with limited plantar flexion and inversion. It may be mild, moderate or severe: in severe cases the dorsum of the foot may touch the anterior surface of the tibia. There is no bone or joint abnormality and ossification is normal. It is the commonest deformity at birth (1 per 1000 live births) and is more common in females (1:0.6) and in firstborn. It is important to distinguish this from congenital convex pes planus. There is an association with flexible flat foot in the older child (see Flat foot, p. 127).

2. Talipes varus. The forefoot is adducted and inverted on an inverted hindfoot which dorsiflexes normally. Talipes varus must be differentiated from postural clubfoot. The deformity is fully correctable with no bone or joint abnormalities. Often the other foot has a valgus posture and is associated with other features of 'the moulded baby syndrome' (i.e. pelvic obliquity, scoliosis, etc.).

3. Talipes valgus. The abducted and everted forefoot with hindfoot eversion is the mirror image deformity of talipes varus and has similar features.

4. Postural metatarsus adductus (see Intoeing, p. 157).

5. Postural clubfoot. The forefoot is adducted and inverted, the hindfoot is inverted and the ankle is plantar flexed. There is normal clinical and radiological orientation of the talo-calcaneo-navicular joint and the talar head and neck are normally situated (compare with CTEV). Clinically there are normal skin

creases, the navicular does not abut the medial malleolus and the deformity is correctable.

Treatment. Postural deformities often resolve spontaneously. Occasionally they require gentle manipulation, strapping or splinting. Very rarely, soft tissue surgery is required.

Malformations

1. Congenital talipes equinovarus (CTEV) (see Clubfoot, p. 85).

2. Congenital convex pes valgus is a primary dorsal and lateral dislocation of the talo-calcaneo-navicular joint. The navicular articulates with the dorsal talus locking it in a vertical plantarflexed position *(congenital vertical talus* or *rocker-bottom foot)*. The forefoot is abducted and dorsiflexed at the midtarsal joint and the hindfoot is in equinovarus, causing a rigid deformity with a convex sole. The dorsal ligamentous and tendinous structures are shortened. Often it is seen in association with neuromuscular abnormalities.

- *Investigation.* Plain radiographs show that the calcaneum is in equinus whereas the forefoot is dorsiflexed. In the lateral view the longitudinal axis of the talus is parallel to the long axis of the tibia.
- *Treatment.* The objective is to reduce the navicular and calcaneum into a normal relationship with the talus and to maintain reduction. This can be achieved by closed methods if diagnosed early by holding the foot plantarflexed in plaster. Otherwise, open reduction and stabilization should be undertaken at 3 months after a period of stretching exercises. Occasionally, excision of the navicular is required. If management is delayed, a triple arthrodesis with a Grice-Green procedure may be indicated.

3. Tarsal coalitions (see Flat foot, p. 127).

4. Congenital metatarsus varus is a medial subluxation of the tarsometatarsal joints with inversion and an adduction deformity of all five metatarsals. It is the more resistant malformation equivalent of the postural metatarsus adductus (see Intoeing, p. 157).

5. Congenital ball-and-socket ankle joint is rare. The proximal articular surface of the talus is dome-shaped in both the lateral and antero-posterior plane. The distal tibia is reciprocally shaped. It is associated with tarsal coalition, hypoplasia of the fibula and congenitally short limb. The condition is usually asymptomatic but ankle sprains are more common.

6. Congenital cleft foot (lobster claw foot) is a form of ectrodactyly with absence of two or three central digital rays of the foot. A varus deformity of the hallux is common and the phalanges of the lateral ray deviate towards the midline. The condition is rare and in its typical form is bilateral, affecting hands and feet. This is an autosomal dominant condition. Treatment is tailored to each individual functional problem.

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Related topics of interest

Clubfoot (p. 85)
Flatfoot (p. 127)
Hindfoot arthrodesis and osteotomy (p. 147)
Neuromuscular disorders (p. 195)

CONGENITAL HAND DEFORMITIES

The upper limb forms between the 4th and the 7th week *in utero* although internal differentiation continues to take place until birth. Deformities may be inherited or acquired *in utero.* Assessment is difficult because of lack of communication with the young baby. Treatment is directed at the parents as well as the child. The following classification is of the American Society of the Hand.

Clinical problems

1. Failure offormation of parts

- *Transverse.* These present as 'congenital amputations' which may occur through the upper third of the forearm, the carpus, metacarpus or phalanges.
- *Longitudinal* These may occur along a border of the limb or may present as a segmental absence (e.g. thalidomide) where the hand may be joined to the elbow.

Radial club hand comprises hypoplasia or absence of the radius, which is replaced by a fibrous anlage, bowing of the ulna and radial deviation of the wrist. The thumb may be hypoplastic, rudimentary or absent. The humerus is short, the elbow is often stiff in extension and there is stiffness of the index finger. The condition is often associated with cardiac abnormalities and blood dyscrasias.

Cleft hand is typically familial and affects both hands. The middle ray including the metacarpal is absent and the child is left with a V-shaped defect. The atypical cleft is sporadic and affects only one hand. The metacarpal is present but more than one digit is involved and the child has a U-shaped defect.

2. Failure of separation or differentiation of parts. Syndactyly is the commonest of all hand deformities and occurs in up to 1:1000 live births. The third, fourth, second and first web spaces are affected in descending order of frequency. Complete syndactyly joins the fingers as far as the tips whereas incomplete syndactyly involves only part of their length. In complex syndactyly bony fusion is present: in simple syndactyly, only the soft tissues are joined. In acrosyndactyly there is bony fusion with a fenestration proximally.

Symphalangism is failure of differentiation of interphalangeal joints. Camptodactyly is a congenital flexion deformity of the proximal interphalangeal (PIP) joint, usually of the little finger, but occasionally affecting the thumb. Commonly bilateral, the deformity becomes most noticeable during the growth spurt. Clinodactyly is the result of deformity of the middle phalanx which, if severe, has a delta phalanx with a C-shaped epiphysis. Congenital trigger thumb, like any trigger fmger, must be distinguished from congenital clasped thumb due to hypoplasia of the extensor muscles.

3. Duplication (polydactyly). Duplications may be formed of soft tissue alone, or a fully formed extra digit may articulate with a normal metacarpal or with an extra metacarpal. In the thumb the metacarpal, proximal phalanx and distal phalanx may be bifid or duplicated and there may be triphalangism.

4. Overgrowth (hyperplasia). A rare sporadic unilateral condition which in 70% of cases involve more than one digit in the distribution of a peripheral nerve. The enlargement may be static or progressive.

5. Undergrowth (hypoplasia). Hypoplasia may be generalized, in which case the part must be fully formed but in miniature, or may shorten the metacarpals (brachymetacarpia), fingers (brachyphalangia) or digits.

In the thumb, the Blauth grading of hypoplasia is used:

(a) slightly small but otherwise normal;
(b) as (a) but with adducted first web and lax ulnar collateral ligament of the metacarpaphalangeal (MCP) joint;
(c) significant hypoplasia of skeleton, muscles and carpometacarpal joint;
(d) pouce flotant;
(e) aplasia.

6. Congenital constriction band syndrome. This often occurs with other abnormalities. The ring constriction may cause distal deformity, with or without lymphoedema, distal fusion (acrosyndactyly) or autoamputation.

7. Generalized skeletal abnormalities.

Treatment

The goals of treatment are to improve function and cosmesis by correcting deformity.

1. Splintage. For radial club hand, serial splintage initiated shortly after birth may help restore normal alignment of the hand on the forearm as a prelude to wrist centralization. Splinting may prevent triggering of the thumb and in mild cases of clasped thumb may prevent the occurrence of flexion deformities.

2. Timing of surgery. Congenital constriction rings require urgent surgery to decompress the limb. Complex syndactyly, syndactyly between digits of different length, acrosyndactyly, delta phalanges and club hand require early, but not urgent, treatment to prevent further deformity. Most other conditions should be

observed for a time. Many will not require surgery and the delay will allow the affected parts to grow, making surgery easier.

3. Techniques. Radial club hand is treated by fusion of the ulna to the carpus (centralization of the carpus). Thumb hypoplasia may be treated by pollicization of a finger or by free transfer of a toe to provide precision grip. Cleft defects can be closed with repair of deep transverse metacarpal ligaments. Syndactyly is treated by creating a commissure using local flaps and, if necessary, by skin grafting. Only one side of any digit should be operated on at any one time for fear of vascular injury. In duplication of the thumb at the MCP joint, the radial digit is sacrificed if the ulnar is of a good size so that the ulnar collateral ligament does not have to be reconstructed. Distal duplications may be treated by resection of the central wedge.

Further reading

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Related topic of interest

Congenital upper limb abnormalities and deficiencies (p. 106)

CONGENITAL KNEE ANOMALIES

Major congenital knee deformities are relatively rare and are frequently combined with other deformities of the hip and foot. They often accompany neuromuscular disorders.

Anomalies of the patella and extensor mechanism

1. Congenital absence of patella is rare and bilateral in 70% of cases. The profile of the knee is flattened in the flexed position. There may be little disability if the quadriceps are intact and of normal strength. The patella is congenitally absent or hypoplastic in the nail-patella syndrome (NPS) where there may be accompanying elbow deformities, iliac horns and dysplastic nails. NPS is a dominantly inherited condition linked with the ABO blood group gene.

Since the patella is unossified until the age of 3 years, the diagnosis of congenital absence is clinical. Treatment is directed at quadriceps function which may improve with strengthening exercises. In the presence of significant weakness, transfer of one or a combination of sartorius, gracilis, semitendinosus and biceps to the quadriceps tendon will increase power.

2. Congenital bipartite and tripartite patella. Two or more ossification centres are present in the patella in 15% of the population, usually bilaterally. The commonest site for an accessory centre is the superolateral pole (25%) then the lateral margin (20%) and the inferior pole (5%). The condition rarely causes disability.

3. Congenital dislocation of the patella. This is a rare familial condition caused by failure of medial rotation of the quadriceps myotome during development. It is characterized by irreducible lateral patellar dislocation. The patella is small and adheres to a hypoplastic lateral femoral condyle or ilio-tibial band.

Secondary deformity develops with growth. The tibia becomes abducted, laterally rotated and flexed on the femur. Knee extension is weak or absent and the child may present walking on his knees. It is frequently associated with Down's syndrome. Lateral radiographs show an absent quadriceps shadow and an AP projection of the tibia because of rotation.

Treatment is surgical as soon as the diagnosis is made. Quadriceps realignment is carried out with reduction of the patella, wide lateral release and medial stabilization including a Goldthwait procedure if necessary. Tibial osteotomy may be needed.

4. Congenital habitual dislocation (CHD). CHD is characterized by recurrent lateral dislocation of the patella when the knee is flexed. The primary anomaly is a contracture of vastus lateralis and the ilio-tibial band. It usually presents in mid-childhood with an inability to flex the knee when the patella is held in the midline. Surgical correction involves lateral mobilization and medial stabilization.

Knee hyperextension deformities

A knee that hyperextends is common in the presence of generalized joint laxity. A knee extending more than 20° is abnormal. Recurvatum may result from disturbance of epiphyseal growth at the upper tibia or lower femur but a true genu recurvatum occurs at the knee joint itself.

1. Simple genu recurvatum. Characterized by bilateral involvement with a normal range of movement. Usually identified by the parents as the child walks with a hyperextended knee. There is evidence of general joint laxity but it does not progress and by 3 years is self-limiting. No treatment is required in children under 7 years. A child who presents with pain and instability over the age of 7 years may need a closing-wedge flexion supracondylar osteotomy 2 cm above the distal femoral physis.

2. Fixed genu recurvatum. A severe deformity presenting at birth with unilateral restriction of knee flexion and associated hip or foot deformities. Very rare if not associated with congenital neurological conditions. The anterior knee is hollow with a transverse crease. Both femoral condyles are felt posteriorly.

Radiographs demonstrate contact between articular surfaces (cf. congenital subluxation). If untreated, secondary changes occur and the posterior tendons displace anteriorly to become extensors.

If passive flexion is greater than 50°, manipulation and stretching followed by splintage or serial plasters is adequate. More severe deformities or failure to respond to treatment in 2 months require surgical correction in the first year of life by quadriceps tendon V-Y elongation.

3. Congenital subluxation and dislocation. These are rare conditions. Their aetiology is multifactorial and includes both genetic and mechanical factors such as absence or hypoplasia of the cruciate ligaments, quadriceps fibrosis and contracture (localized arthrogryposis, congenital paralysis of knee flexors, anterior subluxation of the collateral ligaments and hamstring tendons). The tibia always moves anteriorly and the degree of hyperextension varies from 20° to 120°. Flexion may range from 0° to 90°. The deformity does not always prevent walking. Other associated deformities are DDH, fibular hypoplasia, congenital foot deformity, joint laxity and arthrogryposis and spina bifida.

Manipulation and the 2-weekly application of long leg plasters or spicas may be successful if commenced in the first week of life. Night splints may be used once flexion reaches 60°. With a combined knee and hip dislocation the desired positions may be maintained in a Pavlik harness. Treatment is usually complete at 6 months. Skeletal traction with progressive flexion may help in resistant cases.

Anterior release and quadriceps elongation is indicated if flexion is less than 30° at 3 months, if non-operative treatment fails or if the diagnosis is made in the older child. A resistant dislocation is often encountered in arthrogryposis or spina bifida.

4. Congenital discoid meniscus. This condition is not uncommon. It is usually lateral and bilateral. Discoid medial meniscus is rare (1:50). There are two subtypes: the *complete* discoid meniscus with normal peripheral attachments which is frequently asymptomatic and the symptomatic variety with *incomplete* peripheral attachments which is significantly thicker than normal and has an abnormal ligamentous attachment to the medial femoral condyle (ligament of Wrisberg). The posterior horn is not attached to the tibia and is, therefore, hypermobile. It may present at any age although commonly presents between 12 and 15 years. Due to increased meniscal mobility there is usually limited extension, with painful snapping and locking of the knee. In infancy it may present as an audible click (differential diagnosis DDH) which becomes more marked with walking. On examination a palpable or audible click may be evident with medial displacement into the inter-condylar notch when the knee is extended. It may present with lateral joint pain alone or with a history of locking without injury.

A wider lateral joint space can be seen on plain AP standing radiographs of the knee. Arthrography, MRI and arthroscopy will all confirm the diagnosis.

Treatment is aimed at conserving meniscal stock. Immobilization of the knee in a POP cylinder for 4 weeks may reduce symptoms to an acceptable level. Subsequently, arthroscopy with excision of a meniscal tear or a reshaping meniscoplasty may be helpful. Meniscectomy is carried out as a last resort for continuing instability, locking and pain.

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Related topics of interest

Clubfoot (p. 85)

Congenital foot deformities (p. 88)

Developmental dysplasia of the hip (p. 119)
Flatfoot (p. 127)
Neuromuscular disorders (p. 195)
Spina bifida (p. 296)

CONGENITAL NECK ANOMALIES

Towards the end of the third week of gestation, the mesoderm around the neural tube and notochord breaks up into a series of segmental structures called *somites.* At the beginning of the fourth week, the cells of the ventral and medial wall of the somite change their appearance and start moving towards either side of the neural tube where they form the mesenchymal *sclerotome.* Towards the end of the fourth week, these start to fuse in a cephalocaudal direction to form *primitive hemivertebrae* which fuse across the midline at 6 weeks to form the vertebral bcxiies of the spine. The dorsolateral mesenchyme differentiates into a hemilamina and pedicle so that at birth, there are three ossification centres, one in each hemilamina and one in the vertebral body. These fuse at the end of the first year of life. Failures of development, segmentation, migration or fusion during this process may give rise to congenital anomalies.

Clinical problems

1. Basilar impression is a deformity of the bones of the base of the skull which become indented by the upper cervical spine, with the result that the tip of the odontoid protrudes into the foramen magnum and encroaches on the brain stem. *Primary* basilar impression is congenital and may be associated with a variety of other cervical anomalies. A short neck and facial asymmetry are common. Symptoms tend to occur in adolescence. *Secondary* basilar impression is caused by softening of the base of the skull from osteomalacia, Morquio's disease, rheumatoid arthritis, Paget's disease, ankylosing spondylitis and renal osteodystrophy. The clinical features are those of crowding of structures in the foramen magnum, with brain stem compression, cranial nerve palsies, raised intracranial pressure from obstruction of the flow of CSF and vertebral artery insufficiency.

Traditionally, radiological assessment has used plain AP and lateral radiographs of the cervical spine on which can be drawn a series of lines. *Chamberlain's line* is drawn from the posterior lip of the foramen magnum (opisthion) to the dorsal margin of the hard palate, *McGregor's line* from the dorsal margin of the hard palate to the lowest point of the occiput and *McRae's line* from the opisthion to the lowest point of the anterior margin of the foramen

magnum. These lines are still used for screening purposes but *MRI* is now used to defme both bony margins and soft tissue incursion into the canal. The treatment of symptomatic patients is by appropriate decompression and fusion.

2. Atlanto-occipital fusion (occipito-cervical synostosis). There is partial or complete fusion of the arch of the atlas to the occiput resulting in an appearance similar to Klippel—Feil syndrome with a short neck, low hairline, torticollis, restricted neck movements and a high scapula. There are a variety of associated anomalies. Symptoms do not usually develop until middle age and may then be the result of basilar impression resulting in neck pain, myelopathy, cranial nerve or cerebellar compression, vertebral artery ischaemia and sudden death.

Non-operative treatment should be undertaken first as the operative morbidity and mortality are high. If this fails, traction should be applied in an attempt to achieve reduction of the deformity. If successful, posterior Cl/2 fusion may be undertaken with an improved outcome. If, however, reduction is impossible and there are signs of posterior compression of the cord, foramen magnum decompression may be needed. The outcome from this is very variable.

3. Anomalies of the ring of Cl. This is a very rare condition in which there is a hemiatlas with absence of the facet of C1 resulting in a severe progressive torticollis in young children. Vertebral artery anomalies commonly coexist: angiography is indicated if surgery is being considered. Satisfactory results may be obtained from O-C2 fusion if undertaken before the deformity becomes fixed.

4. Congenital laxity of the transverse ligament of the atlas. This occurs in 15–20% of those with Down's syndrome but is otherwise a diagnosis of exclusion in patients with atlanto-axial instability without a predisposing cause. It is demonstrated on lateral flexion/extension views. Radiological assessment of atlanto-axial instability is based on certain measurements. The atlas-dens interval (ADI) is normally no greater than 4 mm in the child and 3 mm in the adult. The transverse ligament must have ruptured if 5 mm of movement or more is present. On the posterior aspect of the dens there has to be sufficient room for the transmission of the spinal contents. This is referred to as the space available for the cord (SAC). This is measured from the posterior aspect of the dens to the nearest posterior structure, whether it is the posterior arch of the atlas or the foramen magnum. This should be more than 14 mm in the adult.

Contact sports and other hazardous activities should be avoided in asymptomatic patients. O-C2/C3 fusion is reserved for those with symptomatic atlanto-axial instability or neurological impairment

5. Congenital anomalies of the odontoid. The ossification centres in the odontoid are usually fused by the age of 5 years with the exception of the summit ossification centre at the tip which fuses by the age of 12.

Odontoid aplasia is very rare but results in complete absence of the basilar portion of the odontoid. It is best seen on an open-mouth view. *Odontoid hypoplasia* is diagnosed by tomography or MRI. The peg is short and squat. *Os odontoideum* is also identified by tomography which shows a rounded ossicle with a well-defined smooth edge. It may be of variable size. There is a wide gap

between the os and the hypoplastic dens and the normal contour of the dens on the through-mouth view is lost. The ossicle moves with the arch of the atlas.

It is thought that these conditions may be both congenital anomalies or acquired following trauma or infection. They are commoner in some of the skeletal dysplasias, Morquio's disease, Down's syndrome and in conjunction with Klippel-Feil syndrome.

They present either as an incidental radiographic finding or with neck pain, the symptoms and signs of atlantoaxial instability, myelopathy or brain stem ischaemia from compromise of the vertebral arteries. Neurological problems are thought to occur in 50% of patients.

Treatment is by Cl/2 or O-C2 (Gallie) fusion in patients who cannot be managed conservatively. This is preceded by a period in a halo-body vest to achieve reduction. Postoperatively, a Minerva cast is applied.

6. Klippel-Feil syndrome. First described in 1912, this is the syndrome of congenital cervical synostosis characterized by a short neck, low posterior hairline and restricted neck movement. Its aetiology is unknown. Occasionally (<20%), facial asymmetry, webbing of the neck and torticollis occur. It is associated with Sprengel's shoulder (25–35%), deafness, urinary tract anomalies (>30%), congenital heart disease (4.2–14%), synkinesia (involuntary paired movements of the hands), anomalies of the hand, kyphosis and scoliosis (60%). Symptoms do not occur until young adulthood. Cervical radiographs show fusion and disorganization of a number of cervical vertebrae. Flexion/extension views and CT/MRI help to define the anatomy of the anomaly. Orthopaedic management is of any neurological abnormality resulting from instability or degeneration of related uninvolved motion segments or of associated spinal deformity.

Further reading

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Related topics of interest

Degenerative disease of the cervical spine (p. 113)
Genetic abnormalities (p. 134)
Juvenile chronic arthritis (p. 162)
Neuromuscular disorders (p. 195)
Paralysed hand (p. 214)
Rheumatoid neck (p. 254)
Scoliosis—early onset (p. 263)
Shoulder pain (p. 281)
Spinal stenosis (p. 306)

Thoracic outlet syndrome and cervical rib (p. 324)

CONGENITAL PSEUDARTHROSIS AND DEFICIENCIES OF THE TIBIA AND FIBULA

Congenital deficiencies of long bones are subdivided by the Frantz-O'Rahilly classification (1961) into either *terminal* or *intercalary* deficiencies. Each type of deficiency is further subdivided into *transverse* or *longitudinal.* Tibial and fibular hemimelias may, therefore, be either terminal or intercalary paraxial deficiencies. This is reflected in the degree of the foot abnormality.

A *terminal deficiency* exists when there is a complete or partial absence of a whole body part distal to an abnormal proximal termination of a limb (transverse or complete paraxial hemimelia). In an *intercalary deficiency* the middle segment of the limb is missing but the proximal and distal segments are present (segmental paraxial hemimelia or phocomelia).

Congenital absence of the fibula (paraxial fibular hemimelia)

The fibula is the long bone most likely to be congenitally absent (then the radius, femur, tibia, ulna and humerus, in descending order). The most common variant is absence of the whole fibula followed by absence of the proximal fibula or occasionally absence of the middle of the fibula. In 15% there is an ipsilateral proximal femoral focal deficiency (PFFD) and almost all affected patients have some shortening of the ipsilateral femur. The foot may be normal but there is often an absence of up to three of the lateral rays.

Fibular hemimelia has been classified by Achterman and Kalamchi (1979), as follows:

Type I (hypoplasia ofthefibula)

Ia. In which the proximal fibular epiphysis is more distal and the distal fibular epiphysis more proximal than normal. There may be a ball-and-socket ankle joint.

Ib. A more severe deficiency with at least 30–50% of the fibula missing and no distal support to the ankle.

Type II (complete absence ofthefibula)

Angular deformities of the tibia are common and are associated with severe foot and ankle problems (tarsal coalitions and lateral ray deficiencies.)

Clinical problems

- Leg length discrepancy.
- Equino-valgus foot.
- Flexion contracture of the knee.
- Femoral shortening.
- Ankle and knee instability (if the child walks, he bears weight on the medial malleolus in the presence of severe deformity).
- Stiff hind foot with absent lateral rays.

Management

In cases of mild deformity when only one limb is affected, the tibia is straight and the foot normal; leg length discrepancy may be the only significant problem.

With moderate or severe deformities without associated PFFD children do better with an early Syme's amputation, usually around the age of 1 year.

A tibial osteotomy to correct angulation should be performed at the same time as Syme's amputation.

In cases of moderate or severe fibular hemimelia with PFFD, Syme's amputation should be postponed to retain limb length until the full extent of the deformity is evident and a combined treatment plan can be made. Ankle stabilization may be performed in the mean time.

Congenital absence of the tibia (tibial paraxial hemimelia)

Clinical problems

This is the converse deformity of fibula hemimelia with either complete absence of the tibia or a variable amount of the tibia remaining. It is often associated with PFFD or congenital short femur and may be bilateral in up to 30% of cases. The involved limb is short with a varus or calcaneo-varus foot. Initially the fibula articulates with the lateral femoral condyle, but with weight-bearing this dislocates and produces gross lateral instability. There is often a skin dimple over the front of the leg. Tibial hemimelia has been classified into four types on the early radiographic appearances by Jones *et al.* (1978)

Management

Diagnostic imaging, arthroscopy, ultrasound and MRI can now define the abnormality early.

Surgical reconstruction may employ the following techniques:

1. Fibular transfer. Stabilization of the knee may be achieved by fusing the fibula beneath the femur and re-attaching the extensor mechanism. Following a talectomy and medial soft tissue release, the ankle may be stabilized by fusing the fibula to the os calcis.

2. Distalfibulotalar arthrodesis.

3. Proximal tibiofibular synostosis is used when the distal fibula is deficient.

4. Distal tibiofibular synostosis is used when the proximal tibia is deficient.

5. Through-knee amputation is reserved for gross knee instability with severe contractures.

Congenital pseudarthrosis of the tibia

Clinical problems

Congenital pseudarthrosis of the tibia has an incidence of 1 in 250 000 live births. There is an association with neurofibromatosis. It has been classified into six types by Boyd (1982).

Type I. Anterior bowing and a defect in the tibia at birth.

Type II. Anterior bowing with an hour-glass constriction of the tibia at birth. The tibia fractures spontaneously before the age of 2. The tibia is tapered, rounded and sclerotic with an obliterated medullary canal. Most often associated with neurofibromatosis and has poor prognosis.

Type III. Pseudarthrosis in a congenital cyst at the junction of middle and distal thirds. Presence of bowing is variable. Good prognosis.

Type IV. Pseudarthrosis in a sclerotic segment of bone without narrowing of the tibia. A stress fracture develops which extends and fails to unite.

Type V. Pseudarthrosis in association with a dysplastic fibula. Prognosis similar to type II.

Type VI. Pseudarthrosis in an intraosseous neurofibroma or schwannoma.

Management

1. Non-surgical. Bracing may reduce tibial bowing. Treatment of fractures in plaster results in a high rate of non-union.

2. Surgical treatment requires adequate excision of the hamartomatous tissue which may extend across to the fibula. This leaves a significant bone defect which may be reconstructed by:

- *Osteosynthesis.* Internal fixation with an intramedullary rod and cancellous bone grafting. Additional support with external splinting is advised.
- *Vascularized fibular grafting* with internal or external fixation.
- *Bone transport with Ilizarov instrumentation.* Resection is performed and bone transport undertaken from a corticotomy in the proximaJ metaphysis.
- *Amputation.* Rarely considered as a primary procedure, but patients do well if it is performed early.

Electrical stimulation using internal implants has been tried. The results are variable.

Complications

1. Re-fracture or non-union following traditional bone grafting techniques occurs in more than 50% of cases.

2. Stiffness of the ankle and subtalar joint frequently occurs if prolonged treatment in a plaster cast has been required.

3. Limb shortening may occur from repeated operative procedures or as a primary feature of the disease.

4. Anterior angulation of the tibia. A valgus ankle from asymmetry of the distal tibial physis is a feature of the condition. Following grafting, progressive anterior angulation of the tibia is occasionally seen.

5. Infection. Repeated operations, soft-tissue scarring and internal fixation all contribute to a high rate of infection which is the commonest indication for amputation.

Further reading

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Related topics of interest

Amputations and prosthetics (p. 15)

Leg length inequality (p. 174)

Proximal femoral focal deficiency (p. 236)

CONGENITAL UPPER LIMB ABNORMALITIES AND DEFICIENCIES

The upper limb bud first appears on the 35th day of gestation and is fully formed by the end of the first trimester. In most cases the injurious agent or genetic abnormality producing the deformity or defect is not identified.

Clinical problems

1. Shoulder. Sprengel's shoulder consists of congenital elevation of the scapula with vertebral and rib abnormalities. It is usually unilateral but if bilateral suggests that there is cervical spine involvement. An omovertebral bar is present in one-third of cases but is not related to disability which is usually slight. Treatment is cosmetic and involves either excision of the superomedial corner of the scapula or mobilization to a more distal position.

2. Phocomelia. In phocomelia the intercalated segment of the humerus or forearm is absent and the limb terminates in a rudimentary arm or hand. It is usually bilateral. Function using the feet can be surprisingly good. Treatment is conservative.

3. Radial club hand (see Congenital hand deformities, P-91).

4. Ulnar club hand. This is much rarer than radial club hand. The most common form is deficiency of part of the ulna and the ulnar two digits. Alternatively, the whole ulna may be deficient with fusion of the radius to the humerus. Forearm bowing is caused by persistence of the cartilaginous anlage which may need to be excised to prevent further deformity. The radial head may dislocate from the humerus. Creation of a single-bone forearm with excision of the proximal radius may be required.

5. Dislocation of the radial head. The radial head may be dislocated in an anterior, posterior or lateral direction. Almost full movement of the elbow is present. The radius is usually bowed and the radial head is conical in shape. Usually no treatment is indicated.

6. Radioulnar synostosis. The radius and the ulna are joined just below the elbow on one, or more often both, sides. The radial head may be absent. The radius may arise directly from the ulna, or it may be intact but fused to the ulna just below the radial head. In some cases there is no bar connecting the two bones but the patient has the clinical signs of synostosis. The forearm is usually

in full pronation. Function is excellent because the shoulder compensates for the lack of rotation. Excision of the bar fails because the bone regrows. No treatment is usually required.

7. Congenital forearm amputation. The most common level for a transverse amputation is just below the elbow. It is usually unilateral. Function is normally excellent because the arm acts as a prop. A prosthesis will not be worn unless it is prescribed early. If the deformity is bilateral and the stump is of adequate length then a Krukenberg operation should be considered.

8. Congenital pseudarthrosis of the forearm. Congenital pseudarthrosis of the radius or ulna is very rare and is usually associated with neurofibromatosis. The radius can often be treated by dual onlay grafting. The distal fragment of the ulna is excised to remove the tether at the wrist.

9. Madelung's deformity. This is more common in girls than boys and does not usually present until adolescence. It is commonly bilateral and there is often a family history. It is associated with diaphyseal aclasis and Turner's syndrome. The aetiology involves damage to and arrest of the volar and ulnar part of the radial epiphyseal plate. The growing ulna impinges on the carpus, causing it to deviate radially. The ulna finally dislocates dorsally. In most cases it is worthwhile waiting until maturity before intervening because symptoms are mild. In the skeletally immature patient, a radial osteotomy and ulnar shortening may correct the deformity, but it is likely to recur with growth. In the mature child treatment can be symptomatic. If pain is arising from the ulnar-lunate complex then excision of the distal ulna with osteotomy of the radius may be carried out to correct the deformity. If the pain is arising from the wrist joint then arthrodesis may be considered.

Management

In most cases the deformity will be noticed at birth. A full family and obstetric history is obtained. In the older child the functional and cosmetic problems must be assessed and the patient's and parents' expectations evaluated. The potential for further growth may result in improvement of some deformities (e.g. post-traumatic) by remodelling and deterioration of others (e.g. Madelung's). In the majority of cases, function will not be improved by operation. A specialist opinion is advisable in the rarer conditions. Active treatment may be undertaken for the following reasons.

1. Cosmetic reasons. In Sprengel's shoulder there is rarely functional improvement following surgery which must therefore be considered only for cosmetic reasons.

2. To prevent further deformity. Splintage of a radial or ulnar club hand may correct or prevent further deformity. Excision of the ulnar anlage in ulnar deficiency is performed to prevent further ulnar deviation of the carpus.

3. To stabilize joints. Fusing the radius to the humerus in ulnar deficiency stabilizes the elbow. Arthrodesis of the wrist can be performed for Madelung's deformity if the wrist is very unstable.

4. To improve function. Creation of a pincer grip by the Krukenberg operation may improve function.

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Related topics of interest

Congenital hand deformities (p. 91)

Congenital pseudarthrosis and deficiencies of the tibia and fibula (p. 102)

CRYSTAL ARTHROPATHIES

This group of conditions is characterized by physical and chemical damage from crystal deposition which causes inflammatory degenerative change in articular cartilage.

Gout

Gout is not a single disease but is a group of disorders of purine metabolism in which crystals of urate from hyperuricaemic body fluids cause an inflammatory arthritis, tenosynovitis and bursitis. It is predominantly a disease of males (20:1) and is rarely seen in pre-menopausal females.

Gout and hyperuricaemia depend on genetic and environmental factors combining to produce either decreased excretion or increased production of uric acid.

Primary gout (95%) is, in 75% of cases, the result of diminished excretion of urate caused by one of a number of genetically determined defects in renal excretion resulting in an inability to respond to a raised purine load. In 25% of cases there is increased production of urate due to specific enzyme defects.

Secondary gout (5%) is the result of acquired conditions which cause over-production (e.g. malignant disease, polycythaemia, leukaemia) or failure of excretion (e.g. renal failure) of uric acid. It may also be caused by thiazide diuretics.

Pathology

Urate crystals are deposited in synovium, tendons and articular cartilage. Their presence excites a physical and chemical reaction causing acute inflammation. In chronic arthritis there is cartilage degeneration with periarticular cysts. Large deposits may cause cartilage and soft tissue collections (tophi). Juxta-articular bone may show extensive destruction and replacement by similar material.

Clinical problems

1. Acute gout. A sudden onset of severe joint pain, often in the metatarsophalangeal joint of the great toe (70%), traditionally known as podagra. In decreasing order of involvement, the ankle, knee, small joints of the feet and hands, wrist and elbow may all be involved. The affected joint is hot, red and swollen and excruciatingly painful. Acute attacks may be accompanied by fever, leukocytosis and a raised ESR.

2. Arthropathy. Single acute attacks rarely cause residual disability but recurrent attacks may cause progressive cartilage and bone erosion and secondary degenerative changes. Severe joint deformities may occur in chronic tophaceous disease. Tophi are deposits of urate surrounded by inflammatory and foreign body reactions and are principally seen in the cartilage of the ear and in bursae and tendon sheaths.

3. Extra-articular features. Urate stones, chronic urate nephropathy, diabetes, hypertension and ischaemic heart disease are associated with chronic gout.

Investigations

1. Blood tests. Serum urate levels may be raised but are not diagnostic.

2. Joint aspiration. Synovial fluid should be examined microscopically with both Gram stain and polarized light as the principal differential diagnosis is from septic arthritis. Gout crystals are negatively birefringent under polarized light.

3. Radiographs. These may show characteristic punched-out erosions or tophi flecked with calcium but are rarely helpful in the diagnosis of acute disease. The differential diagnosis is from cellulitis, septic arthritis, septic bursitis, Reiter's disease, rheumatoid arthritis and pseudo-gout.

Treatment

1. NSAIDs. The treatment of choice in acute disease. These should be given as early as possible after the onset of an attack. Indomethacin and naproxen are the most effective and are continued until the acute attack has subsided when the dose is lowered and continued for a further week. Salicylates and diuretics should be stopped if possible.

2. Colchicine is effective and may be used when NSAIDs are contraindicated. It can cause vomiting and diarrhoea and therefore needs close monitoring.

3. Prophylaxis. This should be considered after resolution of the acute attack in patients with recurrent arthritis, tophi or chronic gouty arthritis, and for those with a markedly raised serum urate.

Allopurinol reduces uric acid production but should not be started until the acute episode has resolved. Alcohol intake should be reduced and overall weight loss is encouraged. Severe calorie restriction should be avoided as it may precipitate an acute attack.

4. Surgery. Excision of an ulcerating tophus or a chronically inflamed bursa (e.g. olecranon or over bunion) may be required.

Joint replacement is rarely indicated for gout but may be required for secondary degenerative changes in the knees or hips.

Pseudo-gout and chondrocalcinosis

Chondrocalcinosis is a common familial condition which results in the deposition of calcium pyrophosphate dihydrate (CPPD) in the fibrous and articular cartilage of older patients. The crystals are formed by chondrocytes and may be produced by articular cartilage in the presence of other arthritic conditions. The menisci and articular cartilage of the knee are common sites where it is usually asymptomatic. Although it is most commonly idiopathic, it can also be associated with diabetes mellitus, hyperparathyroidism and idiopathic haemochromatosis.

Acute shedding of crystals into the joint may occur after a sudden change in the ionic calcium and pyrophosphate equilibrium triggered by an acute illness, surgery, or trauma. The presence of crystals in the joint space may provoke an acute synovitis (pseudo-gout).

Investigation

1. Blood tests are normal unless altered by intercurrent illness.

2. Radiographs may show CPPD in articular cartilage, knee menisci, labra of the acetabulum and glenoid, triangular cartilage of the wrist and symphysis pubis.

3. Aspiration of synovial fluid excludes septic arthritis. The crystals are positively birefringent under polarized light, as distinct from urate crystals.

Treatment

1. Rest or splintage of the joint.

2. NSAIDs for acute symptoms.

3. Joint aspiration and intra-articular corticosteroids.

4. Arthroscopy. joint irrigation and removal of loose bodies will improve symptoms.

5. Arthroplasty. The condition is commoner in the presence of rheumatoid and osteoarthritis, particularly in the knees, and arthroplasty is considered as necessary.

Further reading

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Related topics of interest

Arthritis of the knee (p. 39)
Septic arthritis (p. 272)
Spondyloarthropathies (p. 313)
Total knee arthroplasty (p. 331)

DEGENERATIVE DISEASE OF THE CERVICAL SPINE

The distinction between the normal aging of the cervical spine and pathological degeneration is blurred. In the normal aging cervical spine there are few changes until the age of 30. There is then progressive replacement of the glycosaminoglycan component of both nucleus pulposus and annulus fibrosus by collagen (fibrosis). The capacity of the nucleus to imbibe water drops by about 20% and the annulus becomes weakened by the decrease in structural protein. The vertebral end plates become sclerotic and vascular channels between the vertebral body and disc diminish in number. The result is a less elastic nucleus and an annulus which is more easily damaged. As the disc space narrows anteriorly due to circumferential bulging of the disc, the facet joints degenerate and become osteoarthritic. Each motion segment becomes more slack and traction osteophytes are seen at the margins of the vertebral bodies. The ligamentum flavum becomes hypertrophied and buckles inwards. Within the spinal canal, the neural structures may be compressed by osteophyte and bulging annulus ('hard' disc) or protruded nuclear material ('soft disc'), resulting in neck pain, radiculopathy or myelopathy. These changes are commonest at C5/6, followed by C6/7 and C4/5.

Clinical problems

1. Neck pain is accompanied by headaches in one-third of patients, and by unilateral or bilateral pain in the upper limbs of non-radicular pattern in two-thirds. The shoulders are three times as likely to be affected as the hands. Isolated neck pain is thought to be due to abnormalities in those structures innervated by the sinuvertebral nerve while non-radicular radiating pain probably arises in the facet joints which are segmentally innervated by branches of the posterior primary rami. Treatment of neck pain is hampered by this inability to be precise about the anatomical origin of the pain. Additional investigations, including MRI, discography, root blocks and provocative injection, have not been helpful.

The prognosis for patients with neck pain is that 80% will improve or become asymptomatic, while 20% will remain unchanged or will deteriorate. Surgical intervention for neck pain produces results that are no better than the natural

history of the disease itself. No clinical or radiological features correlate with the outcome.

2. Cervical radiculopathy is the result of nerve root compression at one or more levels. It may be caused by 'soft' or 'hard' disc protrusions. A 'soft' disc can protrude posterolaterally, causing compression of the ventral motor nerve root, or into the nerve root foramen where it compresses both dorsal and ventral roots causing the more typical mixed neuropathy. Central protrusion is more likely to cause myelopathy. 'Hard' disc protrusion is due to osteophytes which are posterolaterally placed on the disc margin or arise from a degenerate facet joint.

Pain and sensory changes in the approximate distribution of the nerve root are exacerbated by coughing, sneezing, straining and neck movement. There may be associated spasm of the cervical musculature. Objective motor and reflex changes occur later than sensory disturbance and are more commonly seen in cases of chronic nerve root compression.

3. Cervical myelopathy. Central protrusion of a 'hard' disc with infolding of hypertrophied ligamentum flavum will narrow the AP diameter of the canal and reduce the area available for the cord. Myelopathy may be due to direct compression, ischaemia or a combination of the two. It may be precipitated by minor flexion or injury, particularly in the presence of a congenitally narrowed canal. Patients complain of numbness, weakness and clumsiness in the hands and eventually in the feet and legs, with gait and sphincter disturbance. Corticospinal tract compression causes the increased tone, brisk reflexes, clonus and upgoing plantar reflexes of an upper motor neurone lesion. Anterior spinothalamic tract compression reduces the appreciation of crude touch and pressure. Lateral spinothalamic tract involvement impairs pain and temperature perception: compression of the posterior gracile and cuneate fascicles diminishes proprioception and vibration sense. Chronic repeated small insults, particularly if compounded by an ischaemic component, cause characteristic patchy change.

Investigation

1. Plain radiographs (AP and lateral) are obtained to confirm the clinical diagnosis and to exclude other disease particularly malignancy, infection and trauma. The distance from the back of the vertebral body to the corresponding lamina should be 17 mm. Reduction of this distance to 13 mm raises the suspicion of pathological narrowing. The correlation between a diameter of 10 mm and cord compression is high. Flexion/extension views are only obtained if instability is suspected.

2. Oblique views will show the foramina, pedicles and facet joints more clearly. Foraminal osteophytes are not thought to cause root compression until the cross-sectional area (CSA) of the foramen has been halved.

3. Special investigations aim to predict the site and presence of lesions which may be surgically treatable. In this respect, MRI (88%) is superior to *CT*

myelography (81%) which is, in turn, better than *myelography* (58%) or *CT* (50%) alone. CT is better than MRI at distinguishing bone from soft tissue and hence 'hard' from 'soft' disc protrusions. MRI can miss small foraminal osteophytes. At present, plain films and a good quality MRI are the investigations of choice for myelopathy, while either MRI or a contrasted CT are equally acceptable for radiculopathy.

Treatment

1. Non-operative. Neck pain, whether acute or chronic, can be alleviated by NSAIDs, simple analgesics, tricyclic antidepressants and, occasionally, steroids. Physiotherapy may include isometric exercises, stretching, postural techniques and advice on day-to-day ergonomics or, for acute pain, heat, ice packs, gentle manual manipulations and traction, massage or a soft collar.

2. Operative. Surgery may be indicated if severe pain is uncontrolled by conservative measures, for instability and for neurological deterioration from radiculopathy or myelopathy. The choices of approach and technique for surgery and its complications are the same as those described in Acute cervical disc disease (p. 1).

Further reading

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Related topics of interest

Acute cervical disc disease (p. 1)
Congenital neck anomalies (p. 98)
Osteoarthritis (p. 202)
Rheumatoid neck (p. 254)
Shoulder pain (p. 281)
Spinal stenosis (p. 306)

DEGENERATIVE DISEASE OF THE LUMBAR SPINE

The term ‘degenerative lumbar spondylosis’ is used to describe the radiological appearance of a spine in which syndesmophytes are present on the anterior margins of vertebral bodies, vertical disc height has been lost and osteoarthritic changes are present in the posterior apophyseal joints. This appearance may be accompanied by a clinical history of low back pain or may be asymptomatic.

Careful pathological studies have shown that degenerative changes are always present in the lumbar spine by middle age and may be seen in many spines by the age of 30. MRI studies have shown that abnormalities of disc morphology can appear in adolescence and may occur at multiple levels in patients with a single proven symptomatic disc protrusion.

Pathogenesis

1. The normal (young adult) disc. This consists of an annulus fibrosus made up of multiple concentric fibrous layers which surround a nucleus pulposus consisting of 80% water and a collagen and protein-polysaccharide ground substance. The disc acts as a shock absorber because the incompressible fluid core distributes pressure evenly to the vertebral end-plates and the annulus fibrosus.

2. Changes in hydration of the disc. By the age of 25 the water content of the annulus has dropped from 80% to 70%, where it levels out. The water content of the nucleus has dropped to 75% by this age but continues to fall until it approaches that of the annulus. The definition between nuclear and annular material becomes indistinct.

3. Cleft formation. As the disc approaches middle age, splits and clefts may appear in its substance. These are most commonly found in the posterolateral part of the disc. There may be a degree of vascular ingrowth reminiscent of a repair reaction and it has been suggested that repeated tears may extend through the whole annulus and predispose to prolapse of nuclear material.

4. Disc deformation and syndesmophyte formation. As the water content of the disc falls and clefts develop in the annulus, the disc becomes less elastic and is capable of greater deformation. The upper vertebral body tilts forwards around the axes of the apophyseal joints and the anterolateral rim of the disc bulges

outwards with accompanying loss of anterior disc height. Osteophytes develop on the anterolateral aspect of the vertebral body by a process of endochondral ossification pf the annulus. The spaces between them are filled with disc material. Further growth of the osteophyte occurs by subperiosteal new bone formation. Osteophytes which form posteriorly are not usually large enough to narrow the canal centrally, but laterally placed osteophytes may cause narrowing of one or both intervertebral foramina.

5. Apophyseal joint changes. The most important result of disc space narrowing is the effect which it has on the posterior apophyseal joints. These are placed under increasing load and become subluxated. The overriding facets protrude into the intervertebral foramen, reducing the area available for transmission of the nerve root. The overloaded apophyseal joints become osteoarthritic and the osteophyte formed around their margins further narrows the lateral recesses and nerve root canal. While osteoarthritis of the posterior column may sometimes occur in isolation, degenerative disc disease is almost always accompanied by change in the apophyseal joints.

6. Soft tissue changes. There is reactive proliferation of the capsule and adjacent ligamentum flavum which buckles into the posterolateral aspect of the spinal canal, further decreasing the cross-sectional area of the canal. Ultimately, spinal stenosis may develop either centrally or laterally.

7. Relationship of degenerative disc disease with low back pain. There is no doubt that advanced degenerative disc disease can be completely asymptomatic. Narrowing of the central canal, lateral recess or intervertebral foramen may result in the symptoms of spinal stenosis or nerve root compression. Low back pain may, in theory, also arise from distortion of any innervated structure, from microfracture, osteoarthritic change or from the ingrowth of innervated vascular tissue into clefts in the disc. In practice, these theories are extremely difficult to prove.

Further reading

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Related topics of interest

Acute lumbar disc disease (p. 5)
Low back pain (p. 181)
Spinal fusion (p. 299)
Spinal stenosis (p. 306)
Spondyloarthropathies (p. 313)
Spondylolysis and spondylolisthesis (p. 317)

DEVELOPMENTAL DYSPLASIA OF THE HIP

The incidence of developmental dysplasia of the hip (DDH) is 1 per 1000 live births and is four times commoner in females. The left hip is more frequently involved than the right (10:1). Bilateral DDH is commoner than involvement of the right side alone. Risk factors include a breech delivery, being first-born, a positive family history and caesarean section. There is an association with other musculoskeletal abnormalities (e.g. congenital torticollis and congenital foot deformities). Possible aetiological factors include mechanical influences, hormone-induced joint laxity, primary acetabular dysplasia and polygenetic inheritance.

Neonatal screening

All babies are examined for hip instability in the first week of life. The range of hip movement, particularly any reduction of abduction in flexion, abnormal skin contours, leg length inequality and the results of the Ortolani (to detect a dislocated but reducible hip) and Barlow tests (to detect a dislocatable hip) should be recorded.

Clinical problems

1. Neonate (0–6 months). Most dysplastic hips are diagnosed at this age. Correct diagnosis and treatment will result in a 95% cure rate. Dislocations *in utero* (teratologic dislocation) are resistant to treatment and are usually associated with other abnormalities, e.g. spina bifida or arthrogryposis.

2. Infant (6–18 months). These children are crawling or walking. The dislocated hip is no longer reducible simply by abduction. There is reduced abduction of the affected hip in flexion due to adductor contracture, asymmetrical skin folds and apparent shortening of the femur (Galeazzi sign). Bilateral dislocation may be difficult to detect if these signs are symmetrical. As a result of weight-bearing, the hip capsule is stretched, the psoas tendon obstructs reduction, the acetabular limbus inverts and the ligamentum teres hypertrophies and elongates. The femoral head becomes progressively flattened postero-medially, the neck drifts into anteversion and coxa valga develops. The

acetabulum is shallow. A subluxed or dislocated hip discovered after the neonatal period cannot be assumed to be due to DDH until other causes are excluded, e.g. cerebral palsy, spina bifida, infection, trauma.

3. Toddler (18 months to 3 years). These children are established walkers with a waddling (Trendelenburg) gait and hyperlordosis of the lumbar spine.

Radiological investigation

In a conscious young baby it is only possible to obtain an *AP view of the pelvis*. Certain lines and measurements aid diagnosis.

1. Hilgenreiner's horizontal line (drawn through the triradiate cartilages).

2. Perkins' vertical line (at right angles to Hilgenreiner' s horizontal line, through the anterior inferior iliac spine).

In combination, these lines produce quadrants which can be used to localize the femoral capital epiphysis. Before this ossifies, the metaphyseal beak of the proximal femur will lie in the inner lower quadrant in a normal hip.

3. Acetabular index. The angle between Perkins' vertical line and the line of slope of the acetabular roof. In the newborn it should be equal to or less than 30°.

4. Shenton's line. A line following the curve of the inferior margin of the superior pubic ramus down on to the inferomedial border of the femoral neck should be unbroken. Elevation of the lateral part reflects proximal migration of the femoral head and neck.

5. Teardrop sign (Kohler).

6. Femoral capital epiphysis shows delayed appearance and irregular maturation.

7. Hypoplasia of ipsilateral iliac wing.

8. Increased femoral neck-shaft angle.

Arthrography is performed under general anaesthetic. This outlines the articular surfaces of the acetabulum and femur thereby defining the contours of the joint. It can confirm a satisfactory concentric reduction and will outline abnormal soft tissues such as an inturned limbus, hypertrophic ligamentum teres or obstructive iliopsoas tendon.

Management

Clinical examination by an experienced assessor and a high index of suspicion when risk factors are present are the most important diagnostic assets in the newborn. At this age, ultrasound is the investigation of choice as X-rays are not reliable. Plain radiographs become useful at 3–4 months with development of the femoral capital epiphysis.

Treatment of DDH is largely related to the age at which it is first diagnosed.

1. 0–6 months. The *unstable dislocatable hip* needs no immediate treatment and may be reviewed at 3 weeks of age. If it has stabilized, no further action is

taken until *3–4* months when radiographs are taken to confirm a satisfactory position. The *dislocated hip* is reduced if possible and held in a Pavlik harness or Von Rosen splint. It is essential to confirm reduction in the splint by ultrasound or X-ray (on X-ray the femoral metaphysis should be directed towards the triradiate cartilage).

If the hip cannot be reduced primarily, skin traction should be applied for 2–4 weeks. At this stage, an arthrogram is carried out. If the hip remains unreduced, it should be reduced either by gentle manipulation, if possible, or opened, then immobilized in a full hip spica.

Splintage should be for a minimum of 3 months, or for 2 months after the hip is clinically stable. It is then removed and worn at night until the radiographic appearance of the hip has returned to normal. The child is reviewed every 3 months until walking, when a weight-bearing radiograph of the hip is taken. All patients with DDH should be followed up until skeletally mature.

2. 6–18 months. Surgery is usually necessary because of established secondary changes.

Pre-operative skin traction is applied until radiographs show that the femoral head is at the level of the true acetabulum (2–6 weeks). This avoids forced reduction and reduces the risk of avascular necrosis. A *percutaneous adductor tenotomy* is carried out to allow unrestricted abduction then an *arthrogram* is obtained to outline the block to reduction. If a closed treatment fails to maintain reduction in a child under the age of 18 months, an open procedure is indicated to remove the soft tissue obstruction and to obtain a concentric reduction in the acetabulum. It may be performed through an anterior, antero-medial or medial approach. Surgery is followed by the application of a hip spica (95° flexion, 45° abduction) for 10–12 weeks.

3. 18–36 months. These children require open reduction. A period of pre-operative traction is usually appropriate. A *femoral derotation osteotomy* is usually needed to correct the excessive femoral anteversion. Opinion varies as to whether it is necessary to carry out a *pelvic osteotomy* for acetabular dysplasia. Many believe that if the femoral head is satisfactorily seated in the acetabulum by the age of 3, acetabular remodelling will correct the pelvic abnormality over the subsequent 3 years. *Primary femoral shortening* may be needed if relocation of the femoral head cannot be achieved without forcible reduction because of soft tissue contracture. This also reduces the risk of avascular necrosis.

4. 3–8 years. Untreated DDH in this age group is difficult to manage. Secondary changes including shortening of periarticular structures and structural bony changes have occurred in both femur and pelvis. In *unilateral disease* surgery is advised. Pre-operative traction should not be used as 50% will develop AVN. These children need an open reduction with femoral shortening. They may also require a *pelvic osteotomy* and *soft tissue reconstruction* (e.g. capsulorrhaphy). In combined surgery, the best results are obtained with femoral derotation osteotomy and Pemberton osteotomy (Klisic procedure).

A variety of pelvic osteotomies may be considered. In *Salter innominate osteotomy,* the acetabulum, pubis and ischium are rotated on the symphysis pubis as a hinge. A wedge-shaped anterior iliac crest graft is inserted between the margins of the osteotomy and fixed in with pins. The acetabulum is transposed anterolaterally. In the *Pemberton osteotomy* (pericapsular osteotomy of ilium), a curved osteotomy using the triradiate cartilage as a hinge translates the acetabulum antero-laterally. *Redirectional acetabuloplasty* (i.e. Steel or 'dial' osteotomy) can be used in older children with residual dysplasia when remodelling cannot be predicted. It redirects the articular cartilage over the femoral head and requires joint congruency to be successful. *Shelf augmentation* is required when congruency cannot be obtained by osteotomy. It gives support laterally by interposing capsular fibrous tissue. *Chiari osteotomy.* medial displacement osteotomy gives lateral cover in the same way as the shelf procedure.

Bilateral disease up to the age of 5 years is treated in the same way as unilateral disease. Surgery is not recommended over the age of 5. Individuals are normally mobile without pain but may require reconstruction arthroplasty in adulthood.

Complications

Avascular necrosis of the femoral head is the most serious complication of treatment in infancy. This produces femoral head deformity, greater trochanteric overgrowth, leg length inequality and early degenerative change.

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Related topics of interest

Arthritis of the hip (p. 35)
Congenital foot deformities (p. 88)
Congenital hand deformities (p. 91)
Congenital knee anomalies (p. 94)
Flatfoot (p. 127)

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DUPUYTREN'S DISEASE

Dupuytren's disease is a common condition characterized by contractures of the palmar and digital fascia. It is twice as common in men and the incidence rises with age. It is thought to affect a quarter of the male population over the age of 70 in the United Kingdom. The incidence is subject to considerable geographical variation, and is greater in Westernized societies.

Dupuytren's disease is associated with knuckle (Garrod's) pads, plantar fibromatosis in the feet (Ledderhose's disease), Peyronie's disease, diabetes, alcoholism, and epilepsy treated with phenytoin, epanutin or phenobarbitone. It is no more common among smokers. There is a reduced incidence in rheumatoid patients. A history of trauma is not infrequently elicited but it is debatable whether blunt trauma and manual work are causative. It is likely that genetic predisposition is the most important factor and that the other factors determine the age of onset of the disease.

The palmar fascia and its associated ligamentous structures in the hand are referred to as 'bands'. When these become involved by the disease process, the normal longitudinal fibres of the palmar fascia thicken to become opaque white 'cords'. New fibroblasts lay down immature collagen around the cords. Contractures are thought to develop either by contraction of the myofibroblast or by progressive loss of extension as more new collagen is laid down. The digits most frequently involved are the ring and little fingers, followed by the middle fmger and thumb.

Clinical problems

1. The palm. The disease affects the three distal insertions of the longitudinal fibre system. The most superficial is inserted into the skin of the palm, the intermediate spirals around the neurovascular bundle (spiral band of Gosset) and the deepest passes around the tendon sheath at the level of the MCP joints. The earliest sign of palmar disease is nodule formation. The overlying skin blanches on full extension of the hand. Pit formation is caused by contracture of the vertical fibres.

2. The finger. Central cords form in the dermis and lie anteriorly in the midline. Lateral cords form lateral to the neurovascular bundle. The spiral cord

passes from the palm beneath the neurovascular bundle to emerge lateral to it. It then returns to the midline volar to the neurovascular bundle via Grayson's ligament.

3. Little finger. Cords arise from the abductor digiti minimi muscle and its overlying fascia with frequent displacement of the neurovascular structures.

4. Thumb. A cord may develop along the free border of the thumb web, causing adduction of the thumb. The longitudinal bundle of palmar fascia may be involved but flexion of the IP joint is rare.

Management

1. Conservative treatment. Splintage may delay progression of the disease but will not reverse it.

2. Indications for surgery. Surgery should be considered when deformities become fixed and the hand can no longer be placed flat on a table. Deformity of the proximal IP joint is more difficult to correct than that of the MCP joint and therefore needs earlier intervention.

3. Incisions. Either a longitudinal incision with a Z-plasty or a zig-zag incision should be used. The open-palm technique (McCash) allows drainage of blood and reduces pain. The involved skin in the finger is excised and covered with a full-thickness skin graft.

4. Fasciotomy may be indicated in elderly patients with longstanding cords or in cases of severe contracture with skin maceration, as a prelude to further fasciectomy.

5. Partial fasciectomy. Only the involved fascia is excised from the palm and fingers. Partial fasciectomy is just as successful as a radical excision of all the palmar fascia.

6. Contracted proximal IP joint. If the joint deformity is passively correctable then fasciectomy should correct the deformity, but if the deformity is fixed then fasciectomy is less predictable. Hyperextension of the MCP joint may compensate for mild contractures. More severe contractures may require release of the Cleland's ligaments or the check-rein ligaments holding the volar plate. Skin grafting is often necessary.

7. Amputation. This may be indicated in the elderly or infirm. It may also be the most expedient solution for patients with a severe fixed flexion deformity of the, proximal interphalangeal joint of the little fmger.

8 Postoperative care. High elevation for at least 24 hours is mandatory. No splintage is needed after a simple palmar fasciectomy and the hand may be mobilized early. After correction of digital contractures, splintage in plaster for at least a week is recommended. Thereafter, a thermoplasic splint may be worn between periods of exercise, and later, only at night. If skin grafts have been used, prolonged splintage is necessary to avoid contracture.

Complications

Haematoma formation in the fmger leads to scar formation and further contracture. Disease may recur locally or new disease may occur in a neighbouring site. Careful surgical technique should minimize digital nerve injury and skin necrosis.

Further reading

Burke FD, McGrouther DA, Smith PJ. *Principles of Hand Surgery.* Edinburgh: Churchill Livingstone, 1990; 293–312.

Related topic of interest

Congenital hand deformities (p. 91)

FLAT FOOT

A flat foot (pes planus) is a foot which has lost the normal longitudinal arch. This pulls the heel over into valgus and causes compensatory supination of the forefoot. Flat foot in adults and children is usually *flexible* but if the arch is not reformed either on weight-bearing or when standing on tip-toe it is termed *rigid.* Each form of flat foot may be congenital or acquired. Children with flat feet are often overweight and have generalized ligamentous laxity. They may be in a growth spurt and are often competitively athletic. They commonly present with abnormal shoe-wear or because of knock knees.

Clinical problems

Flexible flat foot

Congenital flexible flat foot is discussed under Congenital foot deformities (p. 88).

Acquired flexible flat foot may be due to:

- Ligamentous hyperlaxity. This is often familial but syndromal conditions (e.g. Marfan's syndrome and osteogenesis imperfecta) must be considered.
- Neuromuscular conditions (e.g. muscular dystrophy, peripheral nerve or spinal cord injuries and cerebral palsy).
- Joint disease (e.g. rheumatoid arthritis, osteoarthritis or traumatic degenerative arthritis) may cause a flexible flat foot which becomes rigid with time.

Flexible flat foot is classified as *mild* when, on weight bearing, the arch is depressed but still visible, *moderate* when the arch is not visible and *severe* when the medial border of the foot is convex and the head of talus is prominent. On non-weight bearing, the arch may be normal.

The indications for instituting treatment vary with the age of the child.

- *Age 0–3 years:* at this age the arch is normally filled with a fat pad; flat foot is normal. No treatment is needed. There is no evidence that the outcome can be

altered by intervention at this stage provided that the correct diagnosis is made.

- *Age 3–9 years:* asymptomatic children require no treatment. There are no lifetime studies of outcome in treated and untreated children with this condition but shorter term studies show no benefits from the use of corrective shoes or inserts. Children with symptoms related to the flat foot (e.g. pain in longitudinal arch after prolonged standing, calf aching) benefit from supportive shoes and inserts. It is rare to have symptoms in this age group and other pathology should be excluded.
- *Age 10–14 years:* asymptomatic children require no treatment. Symptomatic children of this age comprise the group which most commonly require treatment.

Rigid flat foot

Congenital convex pes valgus is discussed under Congenital foot deformities (p. 88).

Tarsal coalition is a condition in which varying degrees of union occur between two or more tarsal bones producing a rigid plano-valgus foot. It is due to failure of mesenchymal differentiation and is probably of autosomal dominant inheritance. Many patients have no pain and little deformity. The majority have a stiff valgus hindfoot and a reduction of the longitudinal arch. Perineal muscle spasm due to shortening of the muscle group is an acquired phenomenon producing the clinical entity of 'perineal spastic flat foot'. The two common types are calcaneonavicular and talocalcaneal.

1. Calcaneonavicular bar ossifies between the age of 8 and 12 years. Before this age symptoms are rare. Coalitions may be bony (synostosis), cartilaginous (synchondrosis) or fibrous (syndesmosis). The non-bony varieties are often more symptomatic. Seventy-five percent of patients with familial coalitions are asymptomatic.

2. Talocalcaneal bridge ossifies between the age of 12 and 16 years. The diagnosis is often not made until adulthood. Perineal spasm is more frequent and the cardinal sign is restricted subtalar movement. The middle facet joint is the most common site.

Coalitions present with vague dorso-lateral pain centred at the sinus tarsi. Patients complain of foot fatigue and difficulty walking on uneven surfaces. Rest relieves the pain.

There is usually decreased subtalar movement, with some degree of pes planus (compare sides). As a rule calcaneonavicular coalition is bilateral in 60% and talocalcaneal bilateral in 50%.

Management

Acquired flexible flat foot

1. Non-surgical. The mainstay of treatment is with a medial arch support in a leather shoe with a firm heel counter, an extended medial counter, a Thomas' heel and a medial heel wedge. Rarely, in the severe condition with marked heel valgus and talar head prominence, a custom made orthosis is required, moulded from the 'corrected foot' (i.e. neutral heel and forefoot and reconstituted medial arch). Leg pain is often due to relative contracture of the calf muscles and responds to passive stretching exercises.

Muscles play little role in maintaining the longitudinal arch, and therefore intrinsic or extrinsic muscle exercises have no primary role in managing flexible flat foot.

2. Surgical treatment is rarely required for the symptomatic flexible flat foot. It is considered for disabling pain only after extensive adequate conservative treatment, never for cosmesis. Surgery will reduce the range of movement in exchange for an improvement in the level of pain.

- *Os calcis displacement osteotomy.* This corrects excessive heel valgus (e.g. Dillwyn Evans or Anderson-Fowler)
- *Arthrodesis* (triple arthrodesis, talo-navicular or naviculo-cuneiform).

Acquired rigid flat foot

Investigation

- *Plain radiology.* The calcaneonavicular coalition is best demonstrated on the 45° lateral oblique X-ray. With a non-bony bar the adjacent margins of the bones are irregular and indistinct. Talocalcaneal coalition is best shown on the postero-superior oblique projection (coalition view). Beaking of the dorsal articular margin of the talus on a weight-bearing lateral radiograph may be the only radiological sign.
- *CT scanning* in the coronal plane at 3 mm increments is the most sensitive investigation.
- *MRI scanning* is particularly useful in the detection of non-bony bars.

Management

- *Non-surgical* Initial treatment is with rest or reduced activity, the use of inner heel wedges and medial arch support may diminish stress on the hindfoot. Cast immobilization for 6 weeks may be necessary.
- *Surgical treatment* is considered following failure of adequate conservative treatment.

Calcaneonavicular coalition

(a) *Excision ofcoalition.* In children under 16 years excision and inter-position of muscle or fat gives 75% complete relief of symptoms
(b) *Triple arthrodesis* is performed when there is degenerative change or following failure of selective excision. Often the primary treatment in patients older than 16years.

Talocalcaneal coalition

(a) Selective *excision ofa middle facet bar* gives good relief in younger patients (9–12 years).
(b) *Subtalarfusion* is performed when degenerative change is absent.
(c) *Triple fusion* is performed as a salvage procedure or when (a) or (b) is inappropriate.

Further reading

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Tachdjian MO. *The Childs Foot.* Philadelphia: WB Saunders, 1985.

Related topics of interest

Clubfoot (p. 85)
Congenital foot deformities (p. 88)
Intoeing (p. 157)
Knock knees and bow legs (p. 166)

GAIT ANALYSIS

Gait analysis may be used to study both normal and pathological gait. First, normal gait must be understood. The gait cycle is defined as the time interval between two successive heel strikes of one foot. Each leg in turn has a *swing phase* (from 'toe-off to 'heel contact') and a stance phase (from 'heel contact' to 'toe-off). The normal timing of the gait cycle comprises 60% stance phase and 40% swing phase for each limb. There is a 10% 'double support' period (i.e. both feet on the ground simultaneously) in normal walking which disappears when running.

Cadence (steps per minute), stride length (metres) and velocity (metres per second) are the basic indicators of walking ability. An abnormality of any of these produces an abnormal gait. All measurements obtained by gait analysis should be compared with normalized values for age, height and weight. There are different levels of complexity for measuring gait. The parameters may be measured with basic equipment such as a stop-watch, tape measure and talcum powder, or with high-powered technology.

Principles

Gait abnormalities have been classified in descriptive terms by the Postgraduate Medical School of New York into 15 categories (see Further reading), e.g. lateral trunk bending, circumduction, hip hiking, vaulting, etc. Some abnormalities are best observed from the side and some from in front or behind. Quantitative measurements in gait analysis are subdivided into kinematics (pattern of movement), kinetics (forces around joints), energetics (energy expenditure), dynamics (electromyographic).

1. Kinematics. The simplest assessment is done by visual gait analysis. Adequate analysis requires a walking distance of 10–12m. Introduction of video recording has overcome the two limitations of 'eye-balling' analysis. It provides a permanent record and allows slow motion replay to view high-speed events. This therefore reduces the number of times the patient has to repeat the walk. It can be used to show the patient and a record may be kept for comparison.

Assessment is required in three planes (coronal, sagittal and rotational). Further accuracy in timing the gait cycle may be obtained by using automated

foot switches, for example under the heel and forefoot, or by using an instrumented walkway. Accelerometers are also used to measure high-speed transient events (e.g. heel strike) and also for kinematic analysis of limb movement. Acceleration data can be integrated once to provide velocity and again to provide position.

2. Kinetics. Forces across joints can be measured but more commonly pressures beneath the foot from physical or electrical measuring systems are used. These give information on distribution of weight when standing or walking.

3. Energetics. Energy expenditure may be measured as:

(a) Oxygen consumption, by analysis of exhaled breath.
(b) Heart rate monitoring, e.g. physiological cost index (PCI).

$$\text{PCI} = \frac{\text{heart rate walking - heart rate resting}}{\text{velocity (beats per minute).}}$$

4. Dynamics. Electromyography (EMG) measures electrical activity in specific muscles. This is measured either by surface, fme wire or needle electrodes. EMG is only semi-quantitative and is mainly used to assess the relative timing of muscle contractions during the gait cycle, e.g. before muscle transfer procedures. In cerebral palsy, co-firing of antagonistic muscle groups will cause restricted movement at a joint and will increase energy expenditure.

Systems

Systems to measure individual aspects of gait are used for specific purposes. However, combined kinetic, kinematic systems produce capabilities greater than the sum of their parts. They can calculate, in addition, force and power and, if combined with EMG, provide a reliable and versatile means of gait analysis.

Applications

1. Clinical gait analysis. This analyses a single person and aims to improve that person's gait. It is carried out either in the planning stage of specific treatment or in order to monitor changes in a condition. The commonest clinical use of gait analysis is for patients with neuromuscular disorders. It is also a powerful adjunct to the treatment of cerebral palsy. After complete gait analysis, all soft tissue and bony corrective procedures may be undertaken under one anaesthetic. Such treatment aims to produce an energy-efficient gait.

2. Scientific gait analysis. This often requires different systems which can be be more invasive and less patient-friendly. Research applications may either be clinical (e.g. investigation of specific diseases) or scientific (e.g. biomechanics, performance in sport, physiological research).

3. Other applications

- To differentiate a pathological gait from a 'habit pattern' gait, e.g. to distinguish an idiopathic 'toe-walker' from a patient with a mild spastic diplegia.
- To plan the order of surgical operations in patients with multiple joint disease.
- To monitor results of knee ligament surgery.
- To assess hemiplegia (other than cerebral palsy).
- To design custom-made pressure-relieving insoles by computer-aided design and manufacture (CAD-CAM).
- To align and adjust prosthetic limbs.
- To quantify the outcome of surgical procedures.
- To help improve performance and technique in sport.

Gait analysis can therefore monitor the effects of modifying gait, whether this be by drug therapy, prostheses, orthosis, surgery or physical therapy.

Further reading

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Related topics of interest

Intoeing (p. 157)
Neuromuscular disorders (p. 195)
Paralysed lower limb (p. 218)
Spina bifida (p. 296)

GENETIC ABNORMALITIES

There are normally 46 chromosomes: 22 pairs of autosomes and two sex chromosomes. Recent advances in chromosome diagnosis include gene mapping and the technique of extended chromosome banding which allows more detailed examination of the chromosome structure for deletions and translocations.

Single gene inheritance (unifactorial)

Autosomal dominant

Autosomal dominant disorders are expressed in every generation. The affected individual has a 50% risk of transmitting the trait to any offspring. Males and females are equally affected. The severity of the disorder varies from generation to generation because of variable expression and penetrance. A number of dominantly inherited conditions are of interest to the orthopaedic surgeon including osteogenesis imperfecta tarda, the less severe form of osteogenesis imperfecta in which the patient survives into adult life. There are dominant types of the Ehlers-Danlos syndrome, e.g. Marfan's syndrome which may present with a scoliosis. Classic dwarfism (achondroplasia) and certain of its variants (hypochondroplasia, and dystrophic dwarfism) are dominantly inherited. The group of spondyloepiphyseal dysplasias, however, have varying forms of inheritance.

Neurofibromatosis is one of the more common autosomal dominant disorders and occurs in about 1 in 3000 persons in the general population. Pseudarthrosis of the tibia and scoliosis may present to the orthopaedic surgeon. Pseudarthrosis of the tibia occurs in about 1% of cases of neurofibromatosis, but up to 50% of pseudarthroses are associated with neurofibromatosis. The gene locus for neurofibromatosis has been identified and mapped on chromosome 17.

Less common conditions such as diaphyseal aclasis, cleidocranial dysplasia, nail-patella syndrome and osteopetrosis tarda are all autosomal dominant conditions and occasionally encountered by orthopaedic surgeons.

Autosomal recessive

Autosomal recessive disorders are expressed only in homozygotes in whom the recessive gene has been inherited from both parents. Males and females are equally affected. Consanguineous parents increase the risk of transmitting recessive disorders.

Osteogenesis imperfecta congenita and osteopetrosis congenita, the more severe forms of the conditions, are autosomal recessive. The mucopolysaccharide storage disorders (Hurler's and Morquio's disease) are similarly inherited and may present with orthopaedic problems including short stature. Individuals with these conditions are now being treated by bone marrow transplantation and are living longer.

Metaphyseal dysplasia (Pyle's disease), where there is a failure of remodelling in the long bone metaphysis, is a recessive condition and may present with skeletal deformities, particularly genu valgum. A variety of dwarfism, chondroectodermal dysplasia (Ellis-van Creveld syndrome), may present with problems related to limb hypoplasia.

Linked inheritance

X-linked dominant conditions are rare. The most important is hypophosphataemic or vitamin D-resistant rickets. Bowing and short stature may be prevented by treating the condition early with inorganic phosphate and vitamin D. In females, where the expression may be less penetrant, it may present as osteomalacia in later life.

X-linked recessive disorders are expressed in males and carried by non-expressing females. Medically important conditions such as haemophilia and muscular dystrophy are discussed elsewhere. Charcot-Marie-Tooth disease (peroneal muscular atrophy) is classically inherited as an X-linked recessive although certain forms with varying penetrance may be dominant.

A rare condition due to abnormal cellular copper transport (Menke's syndrome) may present to an orthopaedic surgeon because of fragile bones. The effects on the skeleton are widespread and include Wormian bones in the skull, periosteal new bone formation and thin porotic bones. Progressive cerebral degeneration results in early death. A type of Ehlers-Danlos syndrome (type IX—the occipital horn syndrome) is also related to abnormal copper metabolism and produces skeletal dysplasias.

Polygenic inheritance (multifactorial)

A multifactorial trait is determined by a combination of genetic and environmental factors. Such conditions include cleft lip and palate, some forms of congenital heart disease, and neural tube defects. Of particular interest to the orthopaedic surgeon are conditions such as congenital club-foot and congenital

hip dislocation. Such conditions tend to be familial. The threshold trait may be more common in one sex than the other. For such conditions, prediction of an affected offspring is less specific and counselling may recommend *in utero* investigations as used for the early detection of neural tube defects. The knowledge of increased incidence in certain families may advise prenatal measurement of cc-fetoprotein from blood or amniotic fluid and high resolution ultrasound to identify either spina bifida or anencephaly.

Disorders of chromosomal abnormalities

Down's syndrome is the commonest chromosomal abnormality. The orthopaedic surgeon is most commonly involved because of its association with atlanto-axial instability which has been reported in up to 20% of cases. A proportion of such children require atlanto-axial fusion. Down's syndrome children should be screened before engaging in sporting activity.

The Prader-Willi syndrome is a chromosomal abnormality characterized by obesity, hypotonia, short stature, hypogonadism and scoliosis. The condition is caused by a deletion in the long arm of chromosome 15. Such patients may need surgery for scoliosis and cause significant postoperative respiratory problems because of their weight.

Further reading

Wynn-Davies R. *Heritable Disorders in Orthopaedic Practice.* Oxford: Blackwell Scientific Publications, 1973.

Related topics of interest

Congenital foot deformities (p. 88)
Congenital hand deformities (p. 91)
Congenital neck anomalies (p. 98)
Congenital pseudarthrosis and deficiencies of the tibia and fibula (p. 102)
Congenital upper limb abnormalities and deficiencies (p. 106)
Developmental dysplasia of the hip (p. 119)
Knock knees and bow legs (p. 166)
Leg length inequality (p. 174)
Neuromuscular disorders (p. 195)
Orthopaedic manifestations of haemophilia (p. 199)
Scoliosis—early onset (p. 263)
Spina bifida (p. 296)

HALLUX RIGIDUS

Hallux rigidus (HR) is a term first used in 1888 to describe painful limitation of movement of the metatarsophalangeal joint (MTP) of the big toe. The condition probably starts with repeated trauma to the big toe in adolescence, which produces a chondral defect on the metatarsal head between the apex of the dome and the dorsal margin of the articular surface. As the hallux is extended, abutment of the proximal phalanx against the defect causes pain. Later, osteophytes appear on the dorsal articular margin and form a mechanical block to extension. Further progression results in the characteristic radiographic appearances of osteoarthritis. Other contributory factors including osteochondritis dissecans, hyperextension of the flrst metatarsal and pronation of the forefoot have been suggested.

Problems

1. Pain. Extension of the toe is painful and causes problems with walking.

2. Stiffness. Extension of the hallux is restricted and painful because of impingement on the chondral defect. The appearance of dorsal osteophytes further limits extension. Female patients complain of inability to wear high heels and young patients complain of pain in the push-off phase of running.

3. Activity. Hallux rigidus presents in early to middle adult life when patients are physically active. Exercise exacerbates the symptoms and the desire to exercise must be taken into consideration when planning treatment.

4. Footwear. The ridge of the dorsal osteophyte can be a source of irritation in footwear. High heels and flexible soied shoes also increase pain.

Management

1. Assessment. The restriction of activity and the degree of pain need to be established. Examination of the foot should include assessment of the rest of the foot to detect other abnormalities which may influence treatment (e.g. metatarsalgia). Standing AP and lateral radiographs must be obtained. Finally, the patient's expectations of treatment must be carefully assessed.

2. Conservative. NSAIDs and a change to rigid-soled footwear may alleviate symptoms.

3. Injection. There is anecdotal evidence that injection of local anaesthetic and corticosteroid followed by manipulation may provide significant relief.

4. Cheilectomy. Excision of the dorsal osteophyte (30% of the metatarsal articular surface) sufficient to allow 70° of passive dorsiflexion at surgery produces good results unless osteoarthritis of the joint is advanced.

5. Arthrodesis. This procedure can be performed by screw fixation, wire fixation, intra-osseous wire suture, inlay graft or by a variety of other methods. After 10 years, 90% of patients report excellent results. The toe should be arthrodesed in approximately 15–20° of valgus and in 10–15° extension.

6. Keller's arthroplasty. This procedure produces good or excellent results in 90% of patients at 5 years. It is particularly appropriate for the elderly patient as the rehabilitation is faster than for arthrodesis.

7. Osteotomy. Excision of a dorsally based wedge from the proximal phalanx (Kessel-Bonney operation) may be the treatment of choice in the adolescent and young adult with no radiographic evidence of osteoarthritis.

8. Silicone rubber arthroplasty. Replacement of the base of the proximal phalanx with silicone rubber produces satisfactory results in 86% of cases.

Complications

1. Metatarsalgia. The reported incidence of metatarsalgia is 20% after Keller's arthroplasty and 10% after arthrodesis. Patients with pre-operative metatarsalgia and those who expect to be very active after surgery will fare better with an arthrodesis.

2. Non-union. The rate of non-union depends on the technique of fusion. A cone type of fusion with screw fixation will be successful in 97% while intra-osseous wiring will fail to fuse in 30%: the majority of these are asymptomatic.

3. IP joint osteoarthritis. Radiographic changes of osteoarthritis of the IP joint will be present in 25% at 10 years: 10% will be symptomatic. Treatment may involve taking down the arthrodesis.

4. Silicone synovitis. Four years after a silicone rubber arthroplasty, 72% of cases will show erosive or cystic changes on radiographic examination.

5. Footwear. Female patients may complain of inability to wear high heels following arthrodesis.

Further reading

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Related topics of interest

Hallux valgus (p. 140)
Metatarsalgia (p. 187)

HALLUX VALGUS

The deformity of hallux valgus (HV) consists of lateral deviation of the big toe at the first metatarsophalangeal (MTP) joint with medial deviation of the first metatarsal. There is resultant prominence of the bone and soft tissues on the medial side of the first metatarsal head. The first MTP joint may be subluxated or dislocated: there may also be changes at the cuneiform-metatarsal joint. A hallux valgus angle (HVA) greater than 15° and an intermetatarsal angle (IMTA) of more than 9° are abnormal. Pronation of the big toe may occur when the HVA exceeds 30°.

The aetiology remains uncertain. There is undoubtedly a familial tendency. Race and the wearing of Western footwear are also strongly implicated. It is unclear whether metatarsus primus varus or the lateral deviation of the hallux is the primary causative factor. Predisposing mechanical factors should be sought. The distal metatarsal articular surface may be laterally orientated: the range of movement in the MTP joint may be restricted by degenerative change: the cuneiform-metatarsal joint may be hypermobile, or pronated and in valgus due to collapse of the medial arch or hindfoot valgus.

The treatment of HV is controversial.

Clinical problems

1. Prominent medial eminence. Younger people with relatively normal HVA and IMTA should have their existing footwear modified. If symptoms persist, a simple exostectomy may be indicated.

2. Mild to moderate HV in younger people (<55 years). Those with a HVA of 20–35° and an IMTA of less than 15° may be treated by bunionectomy and soft-tissue realignment (e.g. McBride procedure), or by distal metatarsal osteotomy (Mitchell's, Wilson's or chevron).

3. Severe HV in younger people (<55 years). Correction of the valgus pronated hallux is more difficult to achieve with a distal metatarsal osteotomy in patients under the age of 55 with a HVA of more than 35°. If the IMTA is more than 10°, a basal metatarsal osteotomy should be considered. Patients with high functional demands or those with degenerative disease in the MTP joint should have an arthrodesis.

4. Older patients. Patients over the age of 65 years usually do well after a Keller's operation. Patients with high functional expectations or evidence of pre-existing metatarsalgia should have an arthrodesis. The choice of procedure is most difficult in patients between the ages of 55 and 65.

5. Adolescent hallux valgus. A strong family history is common. Metatarsus primus varus is often present in the hypermobile flat foot. There may be abnormal medial obliquity of the cuneiform-metatarsal joint. Recurrence of the deformity is common and soft-tissue procedures alone rarely suffice. Those with an IMTA of more than 10° should have a metatarsal osteotomy. The location and type of this osteotomy does not affect the outcome. Any basal osteotomy should avoid an open physis. If deformity at the interphalangeal joint is pronounced, a closing wedge osteotomy of the base of the proximal phalanx is indicated.

6. Metatarsus primus varus. Younger patients with a HVA measuring more than 35° and an IMTA greater than 10° may benefit from a basal osteotomy and distal soft tissue procedure.

7. Overriding of the second toe. Treatment of the HV takes priority except in elderly patients when an amputation of the second toe may suffice.

Management

1. Assessment. Initial assessment must take account of pain, footwear problems, functional limitation and cosmetic factors. The valgus deformity and any rotational element should be accurately measured. The rest of the foot is examined for hindfoot causes of HV and any evidence of hallux rigidus or metatarsalgia.

2. Conservative. Initial treatment may simply involve a change to more capacious footwear. Splintage has little effect.

3. Bunionectomy. Usually successful in patients with symptoms related to the bony prominence but minimal deformity. It should be combined with a soft-tissue procedure.

4. Soft-tissue procedures. Most operations are variations on the McBride procedure with release of the adductor hallucis, transverse metatarsal ligament and lateral capsule combined with excision of the medial eminence and reefing of the capsule.

5. Osteotomy. The most common distal metatarsal osteotomies are the chevron, Mitchell and Wilson. Chevrons are usually stable enough to leave free of splintage. Basal osteotomies may be either medial opening-wedge, lateral closing-wedge or crescentic in construction. Basal proximal phalangeal osteotomies are usually of closing-wedge type.

6. Arthrodesis. The most common technique is a cone and socket type with screw fixation.

7. Keller's arthroplasty. Excision of the basal one-third to one-half of the proximal phalanx with trimming of the medial osteophyte. Soft tissue

interposition, wire stabilization and replacement by a silastic spacer are variations.

Complications

Overall, the results of 90% of any of these operations are satisfactory.

1. Recurrence of deformity. May follow inadequate bony correction or soft-tissue balancing, or may result from failure of fixation, instability of an osteotomy or stretching of a soft-tissue procedure. It occurs in up to 11% of McBride operations.

2. Metatarsalgia. Transfer metatarsalgia is caused by shortening the first metatarsal or by defunctioning the first ray. It can be a particular problem after Keller's operation.

3. Hallux varus. Follows overenthusiastic correction of the deformity, particularly by a McBride procedure or metatarsal osteotomy.

4. Hallux extensus. This may occur due to muscle imbalance after a McBride procedure. The MTP joint becomes extended and the interphalangeal (IP) joint flexed. It may also follow a Keller's arthroplasty or a dorsally angulated metatarsal osteotomy.

5. Non-union. Non-union may occur after any metatarsal osteotomy but particularly if the osteotomy has been sited in the metatarsal shaft. Non-union of arthrodeses is related to the stability of fixation and may occur in up to 30% of cases if interosseous wiring is used.

6. Avascular necrosis. May follow metatarsal osteotomy if there has been excessive dissection around the metatarsal head.

7. Other complications. Fracture of the metatarsal head at osteotomy, neuromas of the digital nerves and septic arthritis of the first MTP joint have all been reported.

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Related topics of interest

Hallux rigidus (p. 137)
Metatarsalgia (p. 187)

HAND INFECTIONS

Hand infections usually follow an open injury and rarely occur by haematogenous spread from a distant focus. They are more common among deprived social groups as a result of poor hygiene and nutrition. Other predisposing factors include diabetes mellitus, steroid and immunosuppressant therapy, ischaemia from vasculitis or Raynaud's disease and intravenous drug abuse.

The commonest infecting organisms are *Staphylococcus aureus* (50–80%), *Streptococcus* spp. and coliform bacilli, but following an open wound any organism may be present. A human bite sustained from a punch injury may be contaminated by *Eikenella corrodens* (and is associated with an osteochondral fracture in up to 60% of cases). *Bacteroides* and *Pasteurella multocida* are frequently isolated from animal bites.

Untreated infections may lead to rapid soft tissue destruction from vascular injury caused by increasing tension within the confined fascial spaces of the hand. Unless treatment is early and incisive, long-term stiffness with loss of function is inevitable. Prolonged sepsis may be due to inadequate treatment, the presence of a foreign body or immunosuppression.

Clinical problems

1. Paronychia. Infection of the nail fold often follows a minor penetrating injury. A chronic paronychia occurs in people whose occupation involves sustained immersion of the hand in water and is characterized by an inflamed eponychium with loss of the cuticle.

2. Apical. Infection in the small closed compartments of the apex of the pulp may produce small intensely painful abscesses.

3. Pulp space (felon). Loss of the normal pulp fluctuance is suggestive of infection. Delay in treatment may lead to destruction of the distal phalanx. In dentists, if the pulp has a vesicular appearance consider a herpetic whitlow.

4. Tendon sheath. The classical signs of Kanavel are a flexion deformity of the digit, uniform swelling and erythema, intense pain on extension and tenderness along the line of the tendon sheath. Involvement of the little finger may lead to inflammation of the ulnar bursa: similarly, involvement of the thumb may extend

to the radial bursa. The radial and ulnar bursae may communicate to produce a horseshoe abscess. Rarely, the other fingers may communicate with the ulnar bursa (3–5%).

5. Web space. Infection may occur in the space bounded by the natatory ligament distally and the attachment of the palmar fascia proximally and laterally to the tendon sheaths. The swelling may force the fingers apart.

6. Palmar spaces. Infection lies deep to the palmar fascia in the tight thenar, hypothenar and midpalmar spaces. The hand is often swollen on both the volar and dorsal aspects and the patient is systemically unwell. The fingers are held flexed at the metacarpophalangeal joints but can be moved. A collar-stud abscess may be present.

7. Septic arthritis. An overlying wound (especially human bite) with swelling, restricted motion, instability and discharging sinus are suggestive of septic arthritis. The differential diagnosis includes osteoarthritis, gout and rheumatoid arthritis.

8. Cellulitis. Acute presentation with a systemic illness and lymphangitis are suggestive of streptococcal cellulitis. Blistering distal dactylitis may be the forerunner of the cellulitis.

9. Other. Osteomyelitis in the hand usually follows soft tissue infection or a compound fracture. Haematogenous spread is rare. Gonorrhoea may cause tendon sheath infections or septic arthritis. Cat scratch disease is associated with vesicles around the scratch and a pronounced lymphadenopathy. Occupational infections include orf (sheep workers), erysipeloid (meat workers), pilonidal sinus (hairdressers) and infection with *Mycobacterium marinum* (swimming pool attendants). In cases of unusual tenosynovitis consider the possibility of tuberculosis or *Mycobacterium kansasii.*

Management

Early diagnosis is essential. If no pus is present, rest in a splint with high elevation and an appropriate antibiotic regime is usually effective. Frequent review is mandatory. Surgical drainage is required if the presence of pus is suspected or if there is failure to respond to conservative treatment. General anaesthesia should be used for all hand infections other than small collections in the terminal phalanx. The arm is elevated and a tourniquet applied. Drainage is carried out at the point of maximum tenderness unless anatomical structures prohibit this. Thorough irrigation, debridement and open drainage of wounds are essential. Postoperatively, elevation and splintage combined with systemic antibiotics should lead to rapid resolution of the local signs at which time the hand may be mobilized.

Patients with tendon sheath infections can be rehabilitated faster using a closed irrigation technique. At operation, rather than opening the whole tendon sheath, two short incisions are made and a fme catheter is introduced along its length. Irrigation usually continues for 48 hours, when the catheter is removed.

In punch injuries, the skin wound may be at some distance from the entry wound to the metacarpophalangeal joint when the hand is unclenched. Patients require excision of the wound and irrigation of the joint. Antibiotic therapy should include penicillin for *Eikenella,* a cephalosporin for *Staphylococcus* and anaerobic cover. Complications follow in up to 50% of cases. Surgical drainage should be avoided in cases of cellulitis and herpetic whitlow. Treatment of the hand in drug addicts is along conventional lines but the infection may be complicated by vascular insufficiency which causes a delay in healing.

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Related topics of interest

Osteomyelitis (p. 208)

Septic arthritis (p. 272)

HINDFOOT ARTHRODESIS AND OSTEOTOMY

Malalignment of the hindfoot impairs gait and prevents the wearing of normal footwear. Treatment is normally instituted early in childhood for most congenital causes in the hope of avoiding bony surgery. However, some cases relapse or are resistant to primary treatment and may require hindfoot arthrodesis or osteotomy. Congenital causes include talipes, congenital vertical talus, spinal dysraphism, cerebral palsy and peroneal muscular atrophy. Hindfoot arthrodesis may also be indicated in some cases of polio, post-traumatic arthritis and rheumatoid arthritis.

Clinical problems

1. Calcaneocavus. The neurologically determined calcaneocavus deformity can be treated by Grice-Green subtalar arthrodesis with transfer of tibialis anterior to the calcaneum in younger children to strengthen plantar flexion. Mild cases presenting after skeletal maturity are treated by crescentic osteotomy of the calcaneum to lengthen the foot combined with midtarsal osteotomy. In cases of severe deformity with poor function a triple arthrodesis is appropriate.

2. Cavovarus. Varus of the heel (e.g. from spinal dysraphism or cerebral palsy) causes supination of the forefoot and elevation of the medial arch. Once rigid, the foot must be treated by dorsal wedge tarsectomy and a Dwyer osteotomy. If severe, a triple arthrodesis may be a more reliable choice.

3. Varus. Isolated hindfoot varus from talipes may be treated by Dwyer osteotomy in children between the ages of 3 and 10. Alternatively, a simple oblique osteotomy of the os calcis with lateral and downward displacement of the heel will correct varus and preserve heel shape. If there is a short medial column as well as varus, then a Dillwyn-Evans osteotomy or cuboid decancellation (Tachdjian) is appropriate. In children over the age of 10 years with severe deformity, a triple arthrodesis should be performed.

4. Equinus. Isolated equinus deformity in talipes or polio is best treated by the Lambrinudi procedure.

5. Cavus. Persistent cavus may develop in the bean-shaped foot of talipes because of forefoot supination and adduction. A plantar release and Dillwyn-Evans procedure usually solves the problem.

6. Congenital vertical talus (CVT). Children over the age of 4 years with untreated CVT usually require open reduction of the talus with a Grice-Green subtalar arthrodesis. A child over the age of 12 years who has functional problems should be treated by triple arthrodesis.

7. Valgus. A valgus deformity of lower motor neurone lesions may be treated by Grice-Green arthrodesis or by medialization osteotomy of the calcaneus if valgus is severe.

8. Equinovalgus. For children between the ages of 4 and 9 with cerebral palsy and equinovalgus feet, the Grice-Green subtalar arthrodesis is the procedure of choice. Correction of the deformity and soft tissue balance are vital. Modifications of the original technique are aimed at stabilizing the calcaneum beneath the talus until union occurs. After maturity, a triple arthrodesis is indicated: this is more difficult to achieve in the valgus foot.

9. Planovalgus. In a small number of patients with flexible pes planus the foot loses its flexibility and its ability to reform the arches. In this situation, a triple arthrodesis may be indicated after skeletal maturity to correct valgus and reform the medial arch. Isolated subtalar fusion is not indicated because of the excessive strain placed on the talonavicular joint.

10. Flail foot. In poliomyelitis, pantalar arthrodesis (i.e. ankle and triple arthrodeses) may be indicated to eliminate the need for a long leg calliper if there is quadriceps weakness requiring a knee calliper.

Management

The following operations may be useful in the treatment of hindfoot deformity.

1. Dwyer osteotomy. Dwyer originally described a closing lateral wedge osteotomy for persistent cavovarus. He later modified this as an opening medial wedge osteotomy for varus deformity. However, because of skin problems most now use the closing wedge osteotomy from the lateral side of the os calcis. There are satisfactory results in 85%.

2. Crescentic osteotomy. This is a curved osteotomy of the calcaneus posterior to the subtalar joint for calcaneus deformity. The tuberosity of the calcaneum is shifted posterosuperiorly and held with a staple or wires.

3. Dillwyn-Evans procedure. This operation is a combination of an extensive medial soft tissue release with shortening of the lateral border of the foot by excision of a wedge of bone based on the calcaneocuboid joint. The wedge can be thicker dorsally to take care of equinus. Good results are reported in 80–98%.

4. Grice-Green arthrodesis. This is an extra-articular arthrodesis of the subtalar joint for valgus deformity which is effected by inserting a block of bone from the tibia or iliac crest into the sinus tarsi. Other methods of subtalar arthrodesis involve disruption of the subtalar joint surfaces and cancellous grafting with or without screw stabilization.

5. Lambrinudi arthrodesis. In this procedure, carried out for equinus deformity, a wedge of bone is removed from the plantar and distal parts of the

talus and the foot is repositioned on the rest of the talus which remains in equinus. It is, in effect, a triple arthrodesis with correction of the equinus by excision of a wedge from the talus.

6. Triple arthrodesis. This involves fusion of the subtalar, calcaneo-cuboid and talo-navicular joints. It can be combined with triplanar correction of the deformity by the use of wedge excisions. It is reserved until skeletal maturity. Pseudarthrosis of the talo-navicular joint is not infrequent and there is additional stress put upon the ankle joint.

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Related topics of interest

Clubfoot (p. 85)
Congenital foot deformities (p. 88)
Flatfoot (p. 127)
Neuromuscular disorders (p. 195)
Paralysed lower limb (p. 218)

HIP ARTHROPLASTY

Hip arthroplasty is indicated in patients with incapacitating pain which is refractory to conservative treatment. Arthrodesis and osteotomy should be considered. Total hip replacement is contraindicated in those patients who are medically unfit or who have active infection, neuropathic joints or progressive neurological disease. Resection (excision) arthroplasty may be considered in those with severe infection or poor mobility for other reasons. Resurfacing arthroplasty is no longer considered a worthwhile procedure because of failure rates of 20–50% at 5 years and extensive acetabular bone loss.

Components

1. Cemented femoral stem. There are two basic philosophies. The Charnley and Harris approach is to have a stem bonded to the cement to prevent migration. The Exeter (Ling-Lee) approach is to assume that all components migrate because of cement creep and to allow for this by using a tapered polished stem. Most components are made of stainless steel or a cobalt-chrome alloy and should not have sharp edges to avoid stress risers in the cement. The traditional approach to the cement mantle is to assume an even mantle of at least 3 mm all round. Some designs aim for metal-bone contact medially and laterally with cement filling the anterior and posterior aspects. Cement restrictors, careful bone preparation, retrograde injection with a gun and pressurization appear to improve penetration of the cement into trabecular bone.

2. Uncemented stem. Primary fixation is achieved by press fit. Secondary fixation occurs by ingrowth into porous coating (50–400 jim), mesh or hydroxyapatite. Proximal third coating is used for primary replacements. Complete coating is reserved for revision arthroplasty where the proximal bone stock is poor. Many components are made of titanium which appears to encourage bone apposition. The design endeavours to maximize fit and fill of the canal.

3. Cemented cup. Usually made of ultra-high molecular weight polyethylene (UHMWPE) which should be at least 7 mm thick. The addition of a flange and pressurization appear to improve the cement mantle and fixation. Metal backing

was introduced to reduce stresses in the cement but is associated with higher rates of wear and loosening.

4. Uncemented cup. Initial enthusiasm for screw-in metal rings with UHMWPE liners has been tempered by high loosening rates. Hemispherical press-fit sockets appear to provide the best results. Primary fixation is achieved by supplementary screws, under-reaming or anti-rotation pegs.

5. Head. 22 mm heads have low frictional torque and hence create less shear on the prosthesis-bone interface. However, they may have increased rates of linear wear and dislocation. Heads of 32 mm width permit an increased range of motion and improved stability but have an increased volume of wear and loosening. The best compromise appears to be 26–28 mm. Ceramic appears to have the lowest friction and wear on polyethylene but is prone to fracture and can only be manufactured in larger sizes. Cobalt-chrome and stainless steel are available in all sizes. Titanium has poor wear characteristics. There is renewed interest in metal-on-metal articulations because of improved manufacturing tolerances allowing improved friction and wear rates.

Complications

1. Wear. This is related to head size, the weight of the patient and the presence of a metal backing. Normal rates of wear are 0.1 mm per year.

2. Aseptic loosening. Failure because of aseptic loosening occurs at a rate of 1–2% per year. Wear debris appears to excite macrophage and osteoclastic responses causing loosening and bone loss. Radiographic loosening of the femoral component is defmed as change in position, cement fracture, circumferential lucency of 2 mm or more, or enlargement of radiolucent lines seen on serial radiographs in Gruen zones. Acetabular loosening is associated with lucencies of greater than 2 mm in all three DeLee/Charnley zones, progressive lucencies in one or two zones or change in position. Incapacitating pain and loss of bone stock are indications for revision. Loss of bone stock may be addressed by cement, bulk allografts or impaction of morcellized allograft.

3. Infection. Occurs in 1–2 % of arthroplasties. It is associated with diabetes, rheumatoid arthritis, immunosuppression, urinary instrumentation, alcoholism and psoriasis. Laminar air flow, antibiotic prophylaxis, UV lights, exhaust suits, reduced theatre traffic all help to reduce the incidence.

4. Dislocation. This occurs in 1–4% of arthroplasties. It is associated with small femoral head size, posterior approach, previous hip surgery, inadequate tension and malalignment of the components. Patient education is an important preventative measure. Splintage in an abduction brace or plaster cylinder for 3–6 weeks may be indicated after reduction. Revision of malaligned components in recurrent dislocation should be considered.

5. Heterotopic bone. Increased incidence is found in young men and in patients with ankylosing spondylitis, post-traumatic arthritis, hypertrophic arthritis and diffuse idiopathic systemic hyperostosis. Prophylaxis is indicated in

high risk groups using either indomethacin (1 month pre-operatively and 3 months post-operatively) or irradiation with 1000 rad post-operatively.

6. Nerve injury. The sciatic nerve may be injured during the posterior approach, resulting in a foot drop. The femoral nerve may be injured by traction.

7. Thromboembolism. After hip replacement the incidence of deep venous thrombosis is 40–80%, pulmonary embolism (PE) 1–10% and fatal PE 1% if preventative measures are not used.

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Related topics of interest

Arthritis of the hip (p. 35)
Biomaterials (p. 56)
Infected arthroplasty (p. 153)
Thromboembolic disease (p. 328)

INFECTED ARTHROPLASTY

Infection of a total joint arthroplasty may occur early, by contamination at the time of operation, or late, by haematogenous spread from a distant infected focus. Often, if the infection is primary, more than one organism may be cultured: secondary infection is usually caused by a single organism. The patient's principal complaint is of persistent pain. Examination may reveal inflammation or swelling around the scar and abscess or sinus formation.

Investigation

1. Plain radiographs. These may be normal or may show signs of loosening. In severe infection, the features of osteomyelitis may be present. The bone may be 'moth-eaten' with cloacae and sequestra. *Arthrography* is now infrequently used, but a *sinogram* may still be helpful in defming the location of a sinus and its communications.

2. Scintigraphy using combined technetium (^{99}Tc) and gallium (^{67}Ga) scanning is both accurate and reliable. ^{99}Tc uptake is increased in both aseptic loosening and infection whereas ^{67}Ga is taken up most avidly by polymorphs and is therefore more specific for infection. Indium-111 (^{111}In)-labelled leukocyte scanning is specific for infection but is usually reserved for cases in which there is doubt after using the other two methods.

3. Labomtory investigation includes a full blood count, C-reactive protein (CRP) and erythroyte sedimentation rate (ESR). Patients with chronic infection may be anaemic. The leucocyte count has been shown to be an unreliable indicator of infection. In patients with a stable, aseptic joint replacement the CRP seldom exceeds 20 mg/1. In patients who do not have RA, the combination of a CRP greater than 20 mg/1 and an ESR exceeding 30 mm/h strongly suggests infection. Serial blood samples should be obtained.

4. Identification of organism. The infecting organism may be identified by culture of draining fluid, joint aspirate or material obtained from the joint at open biopsy of the synovium and periprosthetic tissues. Recently, fme needle aspiration of the joint followed by radiometric culture has been described. This has a sensitivity of 87% and a specificity of 95% and may replace traditional methods of sampling and culture in the future. All specimens are sent for aerobic

and anaerobic culture and the sensitivity of each organism identified should be reported. Any organism may be pathogenic. The fmdings of low glucose, high lactate and high protein levels in synovial fluid are also suggestive of infection.

5. Bacteriology. Staphylococcus epidermidis is isolated from infected hip arthroplasty in 37–46% of cases. Multiple strains are present in 30%. If gentamycin-impregnated cement is used at the primary operation, the risk of infection is reduced by a factor of two, but 88% of those that become infected will grow gentamycin-resistant organisms. If gentamycin cement is not used primarily, the risk of gentamycin resistance is 16%. Other common organisms include *S. aureus, Streptococcus* spp., Gram-negative aerobic bacilli and Gram-negative anaerobes.

Management

1. Long-term antibiotic suppression. This is usually reserved for patients who are too unwell or who refuse revision. Long-term antibiotics almost never result in eradication of infection.

2. Debridement and antibiotics. Repeated aggressive debridement leaving the prosthesis in place has been advocated for early infection of total knee arthroplasty. However, the long term results in several series have proved to be unsatisfactory with reinfection rates of between 77% and 100% after 1 year. This approach cannot be supported currently.

3. Excision arthroplasty, Excision (Girdlestone) arthroplasty of the hip gives functional results which are markedly inferior to those of a revision arthroplasty. The leg is shortened, the patient walks with a profound limp and needs to use crutches. The worst cases are seen after resections for sepsis especially in cases where the upper femur has been resected and the patient is in poor health. Failure to remove all cement leads to recurrent disabling infections. The best results are obtained when the femoral neck has been preserved, contrary to traditional teaching. The result does not improve with time.

Excision arthroplasty after infected total knee arthroplasty has been carried out. There is no doubt that meticulous removal of all infected tissue and foreign material combined with intravenous antibiotics will eradicate most infections. However, the functional result is unsatisfactory in up to 80%, some pain remains in 73% and up to 40% are dissatisfied with surgery because of pain and instability.

Excision arthroplasty of the shoulder and elbow gives less satisfactory results than reimplantation, but may be appropriate for those patients in whom the infection has been eradicated, but who have poor residual bone stock. Excision arthroplasty at the ankle is less satisfactory than a successful fusion.

4. One-stage revision arthroplasty. One-stage revision arthroplasty is the best method of salvaging an infected total hip replacement provided that certain guidelines are observed. The infecting organism(s) must be identified and their sensitivities known. The patient then undergoes a thorough debridement with

removal of all sinuses, infected material, cement, cement-bone membrane and implant. If the organism cannot be identified or if it is of considerable virulence, a two-stage revision should be undertaken. The new prosthesis may then be implanted using cement loaded with antibiotics to which the organism is known to be sensitive. The place of impaction allografting in the revision of infected arthroplasty has not yet been established. Up to 90% of one-stage revisions will be successful.

One-stage revision for total knee is still less successful than two-stage revision. Comparable figures for total shoulder and total ankle replacement are not available.

5. Two-stage revision arthroplasty is currently the treatment of choice for infected total knee arthroplasty. The knee is thoroughly debrided with excision of all infected material and a spacer made of PMMA cement to which the appropriate antibiotics have been added is implanted. The patient is then mobilized in a brace or a POP cylinder and intravenous antibiotics are administered for at least 1 month. The interval between removal and reimplantation should be at least 6 weeks. Reimplantation should be delayed if clinical, radiological or laboratory tests, including repeated aspirations and culture, suggest that infection persists. This regimen has a success rate of about 90%. The results are functionally less satisfactory than those achieved for aseptic revision but are better than those of excision arthroplasty.

Two-stage elbow revision for infection is less successful than excision arthroplasty.

Prevention

1. Clean air enclosures. Ultra-clean air enclosures and whole-body exhaust-ventilated suits remain the 'gold standard'. Laminar-flow theatres are the minimal requirement.

2. Prophylactic antibiotics should be administered in high dose on induction of anaesthesia, then 6 and 12 hours after surgery. Second-and third-generation cephalosporins appear to be effective.

3. Surgical technique should be meticulous, with minimal tissue damage and strict asepsis.

4. Prophylaxis of bacteraemia. Dental manipulation, bacterial chest infection, genitourinary infection, gastrointestinal infection, surgery and any septic foci should be preceded or treated with prophylactic antibiotics.

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Related topics of interest

Hip arthroplasty (p. 150)
Musculoskeletal imaging (p. 190)
Rheumatoid arthritis (p. 245)
Septic arthritis (p. 272)

INTOEING

Intoeing may occur as a result of anomalies in the foot (metatarsus adductus), in the leg (internal tibial torsion) or in the hip (persistent femoral anteversion): rarely, these may occur together. The source of the intoeing can be identified by clinical examination without further investigations.

Clinical problems

1. Metatarsus adductus. Congenital metatarsus adductus (CMA) is adduction of the forefoot in relation to the midfoot and hindfoot. An associated congenital dislocation of the hip or acetabular dysplasia is found in 5%. The degree of deformity is determined clinically by the 'heel bisector angle'. In the normal foot this bisects the second and third toes. *Mild* CMA bisects the third toe, *moderate* CMA the third and fourth toes, and *severe* CMA the fourth and fifth toes and beyond. The deformity may be flexible or rigid and there may be an abnormal transverse crease on the medial border of the foot or a widened first web space. The primary deforming factor is relative shortening of the abductor hallucis muscle.

The natural history of untreated CMA is for spontaneous correction in about 85%. Most of these are mild cases with flexible deformities.

2. Internal tibial torsion. In the normal tibia, the foot is angled 15° lateral to the anteroposterior plane when the knee is flexed to 90°. The angle between the transmalleolar axis and the transverse axis of the knee changes from a mean of 5° at 1 year to 15° at adolescence. The normal range in adults is wide and varies between 10° and 40°. Approximately 10% of children have internal tibial torsion: most correct spontaneously. Whatever torsion exists at 7 years will persist into adult life. In the older child who attempts to avoid intoeing there may be a compensatory valgus deformity in the foot which is held everted and abducted. The child may therefore present with a flat foot.

3. Persistent femoral anteversion is abnormal femoral torsion which persists after 8 years of age. It is a distinct developmental abnormality, and does not correct spontaneously with growth. The main complaint is of abnormal gait and clumsiness. Clinically, there is an increased range of internal rotation and a decreased range of external rotation, which is best seen by examining the

child prone. When standing, the patellae face medially and there are often compensatory valgus and abducted feet. There is often a family history.

Management

1. Metatarsus adductus. In general, mild CMA will resolve without treatment. Moderate and severe cases will require active treatment, initially by serial stretching and casting, and only if this fails, by surgery. Surgery is required if the foot is painful, of unacceptable appearance, or causes difficulty in fitting shoes. The resistant deformity may be corrected surgically by release of the abductor hallucis insertion. In addition, in the child under 5 years, a capsular release of the tarso-metatarsal and intermetatarsal joints is performed. Basal metatarsal osteotomies are needed in the older child.

2. Internal tibial torsion. Treatment is controversial. Most correct spontaneously. *Mild* torsion (15° or less) will correct by 8 years old. *Moderate* deformity (15–30°) is likely to correct but should be reviewed at the age of 8 if intoeing persists. Children with severe torsion (>30°) require annual review and will require corrective tibial osteotomy at the age of 8 or earlier if secondary foot deformities or symptoms dictate.

3. Persistent femoral anteversion. No intervention is needed in cases of mild deformity with 15° or more of external rotation in the extended limb. When there is fixed internal rotation or less than 15° of external rotation, lateral rotation osteotomy should be undertaken before secondary foot deformities develop. The site of the femoral osteotomy and method of fixation is a matter of the surgeon's preference. The amount of correction aims to produce an equal range of internal and external rotation.

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Related topics of interest

Congenital foot deformities (p. 88)
Congenital knee anomalies (p. 94)
Developmental dysplasia of the hip (p. 119)
Flatfoot (p. 127)
Knock knees and bow legs (p. 166)
Osteotomies around the knee (p. 211)

IRRITABLE HIP

Irritable hip is a common condition of childhood characterized by an acutely painful hip. It was first described by Lovett and Morse in 1892 as "a short-lived and ephemeral form of hip disease". At that time the distinction from tuberculosis of the hip was important. The condition has many pseudonyms (transient synovitis, transitory hip arthritis, observation hip). The aetiology is unknown. Infection, allergic reaction and trauma are most commonly implicated. There are no specific diagnostic tests. The diagnosis is therefore one of exclusion and made in retrospect on the clinical course of the condition. The diagnosis is made once other conditions have been excluded by routine tests and clinical observation. The differential diagnosis is from Perthes' disease, septic arthritis, osteomyelitis, slipped upper femoral epiphysis, trauma, rheumatoid arthritis, tuberculosis and rheumatic fever.

There is no predilection for side. Bilateral disease is rare (approximately 1%). The age range is between 2 and 12 years (mean 6 years). The male to female ratio is 2:1.

Clinical features

The child presents with a limp (90%) and hip pain (80%) which can radiate to the knee (25%). Weight-bearing may not be possible (70%). In the majority of cases, symptoms have been present for less than 48 hours at presentation. There may be a history of similar episodes of pain. A history of recent infection, often of the respiratory tract, can be elicited in 30%. A significant history of trauma is present in less than 5% of cases.

On examination, the hip is held in slight flexion and external rotation. The range of movement is restricted in all planes, most markedly in abduction and internal rotation. Muscle spasm, particularly of the hip flexors and abductors, is seen and the hip is tender to palpation in the groin. Gauvain's test is positive (reflex contraction of abdominal muscles on internal rotation of an extended hip).

Investigations

1. Blood and bacteriology. A full blood count and erythrocyte sedimentation rate (or plasma viscosity) are traditionally performed to assist exclusion of septic arthritis. In true cases of irritable hip, the results are not significantly different from those of the normal population. Bacteriological investigation should include a throat swab, mid-stream urine and hip aspirate (see below) as a screen for infection.

2. Radiography. An AP X-ray of the pelvis and lateral projection of the hip aid in the exclusion of Perthes' disease or slipped upper femoral epiphysis. At presentation, less than 5% of radiographs are abnormal. A plain radiograph is an unreliable method of diagnosing a hip effusion.

3. Ultrasound may be used to diagnose an irritable hip. It is a sensitive test for diagnosing an effusion and has the advantages of being non-invasive and not requiring the use of ionizing radiation. In skilled hands, features of Perthes' disease or slipped upper femoral epiphysis may be seen. The presence of a significant effusion and severe symptoms is an indication for aspiration of the effusion under ultrasound control. This relieves the symptoms by reducing capsular distension and allows laboratory examination of the fluid.

4. Further investigations. Isotope bone scan and magnetic resonance imaging may be helpful in patients with persistent pain but otherwise normal investigations. Serological testing will exclude juvenile chronic arthritis.

Treatment

If septic arthritis has been excluded, the treatment is bed rest. A proportion may require hospital admission. The majority can rest at home. Hospital admission may be required for symptomatic control, social reasons or lack of compliance.

Skin traction is of no proven benefit. It may occasionally be required if compliance with bed rest is poor. There are potentially harmful effects of traction on the extended hip. Extension of the hip tightens the hip capsule and increases the intra-articular pressure. In theory, tamponade may cause avascularity of the femoral epiphysis, although there is no direct evidence to support this.

Complications

There is no evidence that an irritable hip which has been correctly diagnosed gives rise to complications. The incidence of Perthes' disease is no higher than in a matched normal population. It is, however, widespread practice to re-X-ray the hip 6 weeks after presentation, particularly if mild symptoms persist or if symptoms have recurred. There is anatomical and experimental evidence to suggest that intra-articular tamponade can cause avascular necrosis. To date, this has been demonstrated neither clinically nor radiographically.

It is important that the child's symptoms have resolved completely before mobilization. If mobilization is reintroduced prematurely symptoms tend to persist. Gauvain's test is a sensitive clinical indicator. The average time from presentation to mobilization is 5 days.

Further reading

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Related topics of interest

Developmental dysplasia of the hip (p. 119)
Juvenile chronic arthritis (p. 162)
Osteomyelitis (p. 208)
Perthes' disease (p. 230)
Septic arthritis (p. 272)
Slipped upper femoral epiphysis (p. 287)

JUVENILE CHRONIC ARTHRITIS

Juvenile chronic arthritis (JCA) is a heterogeneous group of diseases characterized by arthritis in one or more joints lasting more than 12 consecutive weeks in a child under the age of 16. Other diseases must have been excluded. Chronic inflammation of joints results in muscle weakness and osteoporosis. Joint hyperaemia may cause epiphyseal overgrowth (e.g. coxa magna). Partial involvement of the epiphysis may cause asymmetrical overgrowth and deformity (e.g. genu valgum). Either may cause limb length inequality.

Types of JCA

There are three distinct types of arthritis which are characterized by their initial presentation:

1. Pauciarticular onset. The commonest presentation (70%), which is defined as involvement of up to four joints within the first 6 months of the arthritis. It is usually of insidious onset. The arthritis may be painless. Children are usually under 5 years old: it is more common in girls (4:1). The most common presenting joint is the knee, followed by the ankle. There is a particular risk of developing chronic iridocyclitis which, if unrecognized and untreated, may lead to blindness. A high percentage have a positive anti-nuclear antibody. Prognosis is good and 70% will be in remission, with little functional disability after 15 years.

A subgroup, usually of boys, who present after the age of 9 years, have an associated plantar fasciitis, achilles tendonitis and features of ankylosing spondylitis. One quarter have a positive family history. It is associated with HLA-B 27 and tends to persist into adult life.

2. Polyarticular onset. This accounts for 20% of cases and is defined as involvement of five or more joints within the first 3 months of onset. The majority of patients are seronegative for IgM rheumatoid factor. It is more common in girls and can present at any age (peak 6–7 years). The presenting child is irritable, with a sudden reluctance to use a particular limb or limbs. Contractures or deformities may develop rapidly and hand involvement is common. These respond well to early treatment. Seropositive disease resembles adult rheumatoid arthritis and commonly presents in girls approaching the menarche. It affects the hands and feet initially and later involves the larger

joints. Patients may develop nodules and vasculitis as well as the extra-articular manifestations of rheumatoid disease. It tends to persist into adult life and is often referred to as juvenile rheumatoid arthritis (JRA).

3. Systemic onset. A systemic illness with myalgia or arthralgia occurs in 10%. Arthritis may not develop until later. Boys and girls are equally affected. It typically presents between the age of 1 and 4 years but can occur in adolescence or young adulthood. There is a spiking fever and a pink maculopapular rash which mainly affects the trunk. There may be a lymphadenopathy and hepatosplenomegaly. Rarely, it can progress to a life-threatening myocarditis, hepatic dysfunction or cerebral involvement. Its diagnosis is often difficult.

Clinical problems

There are specific clinical problems related to each joint. These are principally due to growth disturbance, contracture and joint destruction.

1. Hip. In younger children, joint inflammation accelerates fusion of both the acetabular and femoral head epiphyses causing acetabular dysplasia with subluxation. Flexion/adduction/internal rotation contractures occur commonly. The femoral neck becomes valgus and anteverted. Occasionally a patient with 'wind-swept' legs may be seen. A compensatory structural scoliosis may be generated by prolonged pelvic obliquity. Malposition of the hip may cause a secondary knee deformity. Joint destruction may be erosive or ankylotic dependent on the type of disease. Protrusio is common.

2. Knee. Asymmetrical overgrowth of the distal femoral epiphysis causes lengthening of the involved leg. The knee may become fixed in flexion and valgus in an attempt to compensate. External tibial torsion occurs in response to an internal rotation deformity at the hip. Joint destruction is not usually severe and rarely requires surgery.

3. Foot and ankle. Involvement of the ankle and hindfoot tends to produce a varus deformity whereas midtarsal involvement causes an equinus deformity.

4. Upper limb. At the *shoulder,* limitation of movement is more significant than pain. Radial overgrowth may cause dislocation of the radiohumeral joint at the *elbow.* There may also be overgrowth of the distal ulna at the *wrist,* producing pain and deformity. Severe fixed flexion deformity and subluxation can occur. The pattern of involvement in the *hand* is similar to that in adults. Surgery is rarely required in childhood.

Management

1. Investigation. Investigation should include the appropriate blood tests and radiographs. Joint aspiration is indicated in cases of suspected haemarthrosis or septic arthritis. *Blood tests* should include a full blood count (anaemia, leukocytosis, thrombocythaemia); erythrocyte sedimentation rate (raised in active disease); C-reactive protein (raised in acute disease); IgM rheumatoid

factor (usually negative except in a few with JRA) and anti-nuclear antibodies (more frequently present). *Radiographs* are not indicated initially for diagnosis but may help to exclude neoplasm, rickets, non-accidental injury, etc.

2. Non-surgical treatment. NSAIDs are the most important initial treatment. Ibuprofen and naproxen are commonly used. *Gold* and penicillamine are used immediately in seropositive JRA if erosions are seen. Intra-articular injections of *steroids* are used for exacerbations in large joints. Systemic steroids are reserved for serious systemic illness. *Cytotoxic drugs* are used in life-threatening amyloidosis. *Physiotherapy* is essential for preventing contracture and maintaining strength and movement. *Splintage* may overcome early contractures. *Traction* can overcome minor hip contractures or knee contractures with subluxation.

3. Surgery. Surgery for JCA may be divided into soft tissue procedures, bony procedures and arthroplasty. *Soft tissue procedures* include adductor and psoas release at the hip for mild contracture (conserving the obturator nerve); anterior release of the muscles arising from the ilium (including sartorius and rectus femoris) in addition to adductor and psoas release for severe contracture and lateral patellar release, synovectomy and posterior capsule, hamstring and gastrocnemius release at the knee. *Bony procedures* include epiphyseal stapling at the knee for leg length inequality or angular deformity; supracondylar osteotomy of the knee for varus, valgus, or flexion deformities; a dorsal closing wedge osteotomy for mid-tarsal equinus deformity and triple or midtarsal fusion which are occasionally required for fixed deformities in older children. Arthrodesis in large joints should be avoided in children with polyarticular disease.

Arthroplasty. particularly of the hip and knee, can be considered but is rarely performed in children. If undertaken, custom-made prostheses are usually required to conform to the small, osteoporotic bones and to correct profound femoral neck anteversion. Surgery often requires major soft tissue releases: poor muscle strength prolongs rehabilitation. Survivorship of these prostheses is shorter than in adult rheumatoids.

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Related topics of interest

Hindfoot arthrodesis and osteotomy (p. 147)

Knock knees and bow legs (p. 166)

Leg length inequality (p. 174)

Osteotomies around the knee (p. 211)
Rheumatoid arthritis (p. 245)
Young adult hip problems (p. 340)

KNOCK KNEES AND BOW LEGS

Children commonly present with either bow legs or knock knees. It is important to differentiate between the common physiological forms and rarer pathological forms. The term genu varum or genu valgum is used if the femoro-tibial angle is more than 2 standard deviations (SD) above or below the mean for a particular age. Lateral tibial bowing is common in early infancy (10–15° is normal up to the age of 1 year). Normally the limb is straight by the age of 2. Physiological knock knee becomes maximal between 3 and 4 years of age (greater in girls than boys) and the normal valgus angle (approximately 5°) is established by the age of 7 or 8. There are familial and racial variations.

Clinical problems

A pathological form should be considered in the presence of the following features: *positive family history* (bone dysplasia, syndromes or renal rickets), *short stature or disproportion* (bone dysplasia or endocrine disturbance), *asymmetry or unilateral growth plate damage or infection,* other *musculoskeletal abnormalities* or *a severe or progressive deformity* (Blount's disease). In all cases, development is inconsistent with the normal bow leg-knock knee sequence.

1. Physiological Genu varum is no longer physiological if it occurs over the age of 5 years or if the intercondylar distance is greater than 8 cm. Genu valgum in a child less than 1 year old or in an older child with an intermalleolar distance of more than 10 cm is abnormal. Both varus and valgus abnormalities are commoner in overweight children, in whom weight loss should be encouraged.

2. Tibia vara (Blount's disease). A progressive deformity of the upper tibia which usually presents in infancy as the child begins to walk. It is more common in the West Indies and Africa. Clinically, there is acute unilateral or bilateral angulation just distal to the knee. The cause is unknown, although infection, trauma, avascular necrosis, and a latent form of rickets have been suggested as causative factors. Early treatment with splinting may help. Established deformity often requires surgery at the age of 3 or 4 years.

3. Growth plate disturbance. Trauma may cause a partial physeal arrest and formation of a bony bridge. The most common site for this is the distal femoral

physis. Rarely physeal injury may cause accelerated growth. *Infection* is a rare cause either resulting from a primary infection or more commonly as an extension from osteomyelitis.

4. Fracture malunion of the femur or tibia. A specific fracture of the proximal tibial metaphysis with an intact fibula in children may produce a valgus deformity.

5. Bone dysplasia and 'syndromes' including forms of dwarfism (e.g. achondroplasia, Morquio's disease, Ellis-van Creveld syndrome) produce varus deformities. Epiphyseal and metaphyseal dysplasias may produce either valgus or varus deformity.

6. Bone softening. Rickets (dietary malabsorption, renal hypophosphatasia) is suspected with an increasing varus deformity and a positive family history of growth abnormality. The correction of the metabolic abnormality under 5 years old will allow deformities to resolve spontaneously. In older children corrective osteotomies may be necessary. Juvenile chronic arthritis may also produce angular deformity.

7. Osteoarthritis with thinning of articular cartilage and secondary bone erosion may cause deformity.

8. Miscellaneous conditions such as poliomyelitis, spina bifida and osteogenesis imperfecta may lead to deformity. The primary condition has usually been identified by the time of presentation.

Management

1. Screening examination. Records of height and weight and an careful history are obtained. The site and severity of the deformity should be defmed. The femoro-tibial angle is measured with a goniometer and the intermalleolar or intercondylar distance with a ruler.

2. Radiographs are indicated if a pathological form is suspected. An AP radiograph is taken on a long-leg film with the patella pointing directly forward. The entire length of the femora and tibiae should be visible. The hip-knee-ankle angles are measured. The site of the deformity is identified (i.e. localized or generalized, femur, tibia or both). A *skeletal survey* is required if a bone dysplasia is suspected. *Tomograms* are obtained if a physeal arrest is suspected.

3. Clinical photography for documentation.

4. Non-surgical treatment. The majority of deformities will correct spontaneously as growth occurs. Bracing for valgus deformities is disappointing. Varus bracing is technically easier. It is the treatment of choice in early Blount's disease and is of symptomatic help in osteoarthritis and rheumatoid arthritis.

5. Surgical treatment. Surgery may be needed in younger patients with progressing deformities (i.e. severe Blount's disease, osteogenesis imperfecta). Otherwise surgery is delayed until within 2 years of skeletal maturity. The aim is to make the knee joint horizontal. This may require an arthrogram to define the joint accurately. Deformities are corrected by *physeal stapling* or *osteotomy.*

Physeal stapling requires the use of three staples placed centrally, anteriorly and posteriorly. Depending on the site of deformity this may involve the femoral physis, tibial physis or both. The time and duration of treatment may be calculated from appropriate charts. Regular follow-up is required for timing of staple removal. Over-correction must be avoided.

Osteotomy should, in all cases, avoid the region of the tibial tubercle in the skeletally immature. Injury may cause premature physeal arrest and induce a tibia recurvatum. The choice of osteotomy depends on the nature of the deformity. An *opening wedge osteotomy* is carried out if a gain in length is required. A *closing wedge osteotomy* behaves more predictably and is more stable than an opening wedge. A *dome osteotomy* allows correction of alignment and rotation. *Corticotomy, callotasis and external fixation* (e.g. Ilizarov) may be appropriate if malalignment and malrotation are combined with shortening. *Multiple osteotomies and intra-medullary nailing* may be used in cases of severe generalized abnormality (e.g. osteogenesis imperfecta or severe rickets).

Complications

1. Degenerative arthritis may complicate severe untreated deformity.

2. Malunion and non-union may complicate treatment by osteotomy.

3. Recurrence of the deformity occurs particularly with growth plate abnormalities, osteogenesis imperfecta and rickets.

4. Vascular complications may follow osteotomy, e.g. anterior compartment syndrome or acute vascular occlusion from kinking of vessels following angular corrections.

5. Neurological complications. particularly involving the peroneal nerve, may result from direct or indirect injury at osteotomy, especially those attempting to correct severe valgus.

6. Soft tissue contractures can occur if excessive lengthening results.

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Related topics of interest

Congenital knee anomalies (p. 94)
Intoeing (p. 157)
Leg length inequality (p. 174)

Osteotomies around the knee (p. 211)

KYPHOSIS

Kyphosis is the term used to describe both the normal curvature and abnormal curvature of the dorsal spine. A dorsal curve of up to 30° is normal. Excessive angulation may either be due to a generalized increase in the dorsal curve or a localized *kyphos* (or *gibbus*). The age of presentation will suggest the likely pathology, e.g. a congenital cause in children and pathological fracture or Paget's disease in the elderly.

Clinical problems

Congenital kyphosis

This presents before the age of 10 and often in infancy. The deformity may be purely kyphotic or may present in combination with a rotational deformity. A developmental anomaly is the most likely cause and may be due to *absence of formation* of one or more vertebral bodies or *failure of segmentation*. These can occur at any level of the spine but are most common in the upper and lower thoracic spine or in the lumbar spine (e.g. myelomeningocele). In the upper thoracic spine there may be little visible deformity. It is more obvious in the lower thoracic spine, where there is usually a compensatory lumbar lordosis and ribcage deformity. Paraplegia may occur if the curvature exceeds 90°.

Radiology may define the primary vertebral anomaly, particularly in younger patients before secondary deformity develops, and may reveal fused ribs or spina bifida.

Plain lateral radiographs allow the definition of three types of congenital kyphosis (Winter *et al.,* 1973).

- Type I—absence of formation of vertebral body, usually dorsal hemivertebra, producing short severe curves which are unstable. Paraplegia develops in 20%, most commonly when sited in the upper thoracic or thoracolumbar regions. This usually develops around the adolescent growth spurt at approximately 12 years of age.

- Type II—failure of segmentation (congenital fusion) is a less severe deformity but over a longer segment. Not associated with paraplegia as progression of the curve is slow. Often associated with lumbar pain.
- Type III—this is a combination of types I and II. Progression and prognosis depend on individual characteristics.

MRI is used prior to surgery in cases of congenital kyphosis to exclude the presence of diastematomyelia and cord tethering. *Treatment*. Prognosis is poor in the type I deformity. Moderate and severe type I and III deformities without paraplegia require anterior and posterior spinal fusion. If type I deformity is diagnosed early, posterior fusion, preferably before the age of 3 years, is recommended. Type II deformities need posterior fusion only. If the patient presents with paraplegia, spinal cord decompression is required. An anterior decompression with strut grafting is followed by posterior fusion 2–4 weeks later.

Postural kyphosis

A cosmetic complaint of being 'round shouldered' often occurs in adolescent girls who are tall for their age. There is usually no associated pain. There is a general increase in the curvature of the thoracic spine with corresponding hyperlordosis in the lumbar spine. Shoulders and head are displaced forwards. On lying prone the deformity corrects completely. There is no limitation of movement and no spinal tenderness. The apex of curvature is at the normal level, i.e. mid-thoracic, and the curve is generalized.

Idiopathic adolescent kyphosis (Scheuermann's disease)

A growth disorder of vertebrae producing a kyphosis. It is commoner in girls (2:1). The apex of curvature is in the lower thoracic or upper lumbar spine. There is often a compensatory lumbar lordosis. It is essential to exclude congenital bony abnormalities and neuromuscular conditions (e.g. polio or muscular dystrophy). The aetiology is not understood. It has been proposed that it is:

- Primary cartilaginous abnormality (Scheuermann, 1921).
- Extrusion of disc material into vertebra following multiple minor injuries (Schmorl, 1951).
- Persistence of an anterior vascular groove resulting in vertebral collapse (Ferguson, 1956).
- Congenital growth disorder (Rathke, 1961).
- Secondary to vertebral body osteoporosis as the primary lesion (Bradford, 1976) as part as a generalized skeletal condition.

The differential diagnosis is from tuberculous and pyogenic infection.

Radiology. Characteristic diagnostic features are wedging of *at least* three vertebrae and an overall kyphosis of more than 40°. Wedging means an angle greater than 5° between adjacent vertebrae. The vertebral end-plates are irregular and the disc spaces are narrow and may have protrusion of disc material into the vertebral body (Schmorl's nodes). There is often osteopenia. In the later stages, the bone density recovers in the epiphysis and may produce an isolated ossicle at the antero-superior margin of the vertebral body or a dense rounded margin.

Treatment. If untreated, the kyphosis is progressive but usually self-limiting. If pain is a feature, bed rest is required with application of a plaster jacket. Usually treatment in a brace (e.g. Milwaukee) is effective. This also reduces the hyperlordosis. The brace is worn until there is radiological evidence of bone healing (at least 1 year) and is removed only for bathing and performing postural exercises. It is worn at night for 6 months after radiological healing.

Posterior fusion is only needed in cases of disabling pain or cord compression. The complication rates from such surgery are high.

Osteoporotic kyphosis

Women over 60 years old may develop postmenopausal osteoporotic vertebral collapse in the thoracic spine, leading to increased kyphosis. Fracture may be accompanied by an acute episode of localized back pain. Symptoms often arise from the compensatory lumbar lordosis in a degenerate stiff spine. 'Senile' osteoporosis affects men and women usually over 75 years who are relatively immobile. It may be necessary to exclude neoplasm or infection.

Ankylosing spondylitis

Severe kyphotic deformities may be seen in patients with ankylosing spondylitis. These are considered in a separate section.

Miscellaneous

Primary or metastatic neoplasms in thoracic vertebrae can cause deformity. Disc and vertebral infection, particularly spinal tuberculosis, must also be considered. Neurofibromatosis can cause a progressive kyphosis with a significant incidence of neurological complications.

Further reading

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Related topics of interest

Bony metastases (p. 74)
Postural correction in ankylosing spondylitis (p. 233)
Scoliosis—early onset (p. 263)
Scoliosis—late onset (p. 267)
Skeletal tuberculosis (p. 284)
Spinal infection (p. 302)
Spinal tumours (p. 310)
Thoracic back pain (p. 320)

LEG LENGTH INEQUALITY

Leg length inequality (LLI) may be due to an abnormally short or abnormally long limb. A short leg may be due to a congenitally short femur or tibia, congenital hemiatrophy, fracture malunion or growth plate injury. A long leg may be due to a congenital long limb in association with vascular anomalies or neurofibromatosis. Infection may destroy an epiphysis, causing shortening or, conversely, may cause overgrowth due to chronic osteomyelitis. Abnormalities of bone growth may produce a leg length discrepancy or bilateral short limbs (enchondromatosis, achondroplasia).

Clinical problems

1. LLI of less than 1.5 cm requires no corrective treatment.

2. LLI of between 1.5 and 3 cm in an adult should be managed with a shoe raise. Discrepancies of more than 3 cm are not usually treated with a shoe raise due to its bulk and unacceptable cosmetic appearance.

3. LLI of more than 4 cm leads to marked asymmetry of spinal rotation. It is generally accepted that discrepancies over 2.5 cm can give rise to problems in the spine. Leg length discrepancies of this magnitude may be treated by lengthening the short limb or shortening the longer limb. In general, an average or tall individual with less than 5 cm discrepancy should undergo shortening as this involves less morbidity. However, in some cultures it is unacceptable to shorten a limb. Limits on lengthening may be imposed by congenital abnormality particularly when there is a related soft tissue abnormality affecting muscles, nerves or joints. Often a combination of lengthening and shortening gives the best result.

Management

The aim of treatment is to obtain functional limb equality at maturity. Whether to shorten or lengthen depends on a large number of variables including age, aetiology and the type of deformity. Each programme of treatment is tailored to the individual. Lengthening, in particular, is a long-term programme requiring the full cooperation and commitment of the patient, their family and the medical

team. It must be stressed to both patient and parents that treatment cannot make a leg normal, particularly in cases of congenital deformity.

1. Clinical assessment. Measurement: accurate serial objective measurements of discrepancy, using either grid radiographs or computerized tomography, are required. *Bone age:* serial measurements of bone age are needed. Bone age is best assessed on a plain radiograph of the left hand in conjunction with the Tanner Atlas or Greulich and Pyle Atlas. *Height percentile* is recorded on standard percentile charts. *Corrective blocks:* prior to surgery, a standing film of the pelvis and lower limbs with the pelvis 'normalized' will provide information of the functional discrepancy particularly if there is coincidental foot, hip or spine deformity.

2. Limb shortening. During growth a discrepancy of 4 cm or less may be treated with epiphyseodesis. This involves excision of the growth plate through a short localized incision under X-ray control. The timing of growth arrest may be predicted using the constant annual growth technique (White and Stubbins), femoral and tibial growth charts (Green and Anderson) or the straight-line graph method (Mosley).

Discrepancies of up to 6 cm may be equalized with good cosmetic result by shortening a limb after maturity. This may be achieved by open subtrochanteric shortening with blade-plate fixation (Wagner technique) or by a closed intramedullary mid-shaft shortening technique.

3. Limb lengthening. The safe limit for lengthening is approximately 15% of the original bone length. Discrepancies greater than this are not correctable by one procedure and may require a combination of leg lengthening and contralateral leg shortening or more than one lengthening procedure. Should two procedures be required, they should be performed at approximately 10 years of age and then at maturity. Large discrepancies may only be corrected in acquired short limbs (i.e. with an otherwise normal limb) such as occur after injury to a physis or neonatal infection of the proximal femoral growth plate.

The following techniques may be used: *osteotomy and internal fixation* to correct angular deformity, periosteal stripping to stimulate bone growth in a young child or *corticotomy and callus distraction (callotasis)*.

Modern techniques of callotasis require a single planned operation. This involves application of a fixator, a corticotomy and a delay before distraction. Distraction proceeds slowly (e.g. 0.5 mm, four times a day) to the required length. The fixator is left in place until the callus has consolidated and is then removed as an out-patient procedure. The timing for removal of the fixator is often empirical but may be helped by radiographs, ultrasound or measurements of stiffness.

A unilateral external fixator is easier to apply and is normally used if pure linear lengthening is required. A circumferential fixator (e.g. Ilizarov) is used when correction of malalignment is also required, as angular correction can be achieved at the same time as lengthening. It is also used when greater

lengthening is required as the quality of the callus is better than that achieved by other methods. A hybrid of the two fixators can be used when appropriate.

Complications

1. Pin site infection and osteomyelitis. Pin site infection is more common with unilateral than with circumferential fixators.

2. Malalignment. Varus malalignment is frequently seen when lengthening the femur, and valgus deformity of the ankle and hindfoot when lengthening the tibia.

3. Fracture or bowing deformity may occur after fixator removal. This is most often seen in the presence of poor quality callus when lengthening a congenitally short femur.

4. Premature consolidation may occur and require re-operation.

5. Nerve injury. Paraesthesiae, transient weakness and nerve palsy may occur if lengthening is too rapid or if the limb is overlengthened.

6. Joint problems. Knee stiffness can occur during femoral lengthening. In the presence of an anterior cruciate-deficient knee, femoral lengthening may cause a postero-lateral rotational subluxation. Femoral lengthening may also destabilize a dysplastic hip.

7. Muscle contracture and weakness may follow lengthening in cases of congenital deformity with hypoplasia.

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Related topics of interest

Congenital pseudarthrosis and deficiencies of the tibia and fibula (p. 102)
Osteomyelitis (p. 208)
Proximal femoral focal deficiency (p. 236)

LESSER TOE DEFORMITIES

Poliomyelitis, Charcot-Marie-Tooth disease, myelodysplasia and cerebral palsy are all associated with deformities of the lesser toes. However, in the majority of cases the aetiology is unknown.

Clinical problems

1. Curly toe. Correctable flexion deformities of the lateral three toes at both the proximal and distal interphalangeal (PIP and DIP) joints with normal metatarsophalangeal alignment of the (MTP) joint. It is often familial and presents in childhood.

2. Overriding fifth toe. This is a congenital deformity in which the fifth toe is short, rotated and deviated medially. Pressure symptoms may be relieved by surgical correction using a V-Y plasty or the Butler procedure.

3. Overriding second toe. The second toe overlaps the hallux in childhood. In the adult, incompetence of the lateral capsule and collateral ligaments allow subluxation of the toe medially. Hyperextension of the MTP joint and a claw toe may follow.

4. Hammer toe. Hyperextension of the MTP joint accompanied by flexion at the PIP joint with the DIP joint in neutral alignment. Callosities develop on the dorsum of the PIP joint and on the weight-bearing tip of the toe just distal to the nail.

5. Claw toe. Hyperextension of the MTP joint accompanied by flexion deformity of both the PIP and DIP joints. Both claw and hammer toe deformities may follow a synovitis of the MTP joint whether the cause is idiopathic, traumatic or arthritic. Dorsal subluxation of the proximal phalanx occurs first and may be influenced by longitudinal crowding of the toes in the shoe. The result is change in line of pull of the extensor hood so that it only acts on the base of the proximal phalanx, weakening extension at the PIP joint. Weakness of the intrinsics because of dorsal subluxation and increased action of the flexors further contribute to the problem. Shortening of the capsule and collateral ligaments of the MTP and PIP joints, with bowstringing of the shortened extensor tendons, combine to make the deformities fixed.

6. Mallet toe. Flexion deformity of the DIP joint of the second toe causing pressure on the tip of the toe. It is common in diabetic patients with a peripheral neuropathy.

Management

1. Assessment. The severity of the patient's symptoms determine the need for treatment. Examination of the foot will indicate whether the deformity is flexible or fixed. Any underlying neurological cause should be identified. If foot ulceration or a mallet deformity is present, the patient should be assessed for diabetes.

2. Conservative. In cases of mild deformity, the patient or parents may only require reassurance. Advice about footwear and stretching shoes over pressure points may be all that is needed. Strapping deformities is no longer recommended.

3. Flexor tenotomy. In more severe cases of curly toe, flexor tenotomy has been shown to be as effective as flexor to extensor tendon transfer (Girdlestone's operation). Flexor tenotomy is also indicated for mallet toe deformities.

4. Extensor tenotomy and capsular release. A staged approach is recommended. The initial treatment for claw and hammer toes with hyperextension of the MTP joint is an extensor tenotomy of both the long and short extensors at the level of the MTP joint. This is combined with dorsal capsular release. If the proximal phalanx will not reduce, then section of the collateral ligament (including its plantar portion if necessary) will usually allow correction. Any further tendency to dislocation can then be minimized by section of the tight interossei.

5. Osseous decompression. In cases of severe claw toe or hammer toe deformity the MTP joint may have been dislocated for many years. Any attempt to restore the toe to its normal position will stretch the neurovascular bundle. The options include: proximal hemiphalangectomy if the MTP joint is still tight after capsular release, distal proximal hemiphalangectomy or generous bone resection for arthrodesis of the PIP joint if there is troublesome contracture, and proximal phalangectomy if both joints are severely contracted.

6. Arthrodesis. Arthrodesis of the PIP and DIP joints is indicated for severe hammer and claw toe deformities, but attention must be paid to the MTP joint.

7. Amputation. This may be indicated if there is severe deformity in an elderly patient.

Complications

1. Metatarsalgia. Defunctioning of the toe by dorsal dislocation increases the load borne by the metatarsal.

2. Neurovascular. Clumsy surgery, or overzealous attempts to straighten a toe without shortening it, may compromise the neurovascular bundles.

3. Mallet deformity. Arthrodesis of the PIP joint alone may result in a mallet toe deformity requiring arthrodesis of the DlPjoint.

4. Crossover deformity. Proximal hemiphalangectomy may cause weakening of the lateral structures causing overriding.

Further reading

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Related topics of interest

Hallux valgus (p. 140)

Metatarsalgia (p. 187)

LOW BACK PAIN

Low back pain is of epidemic proportions in the Western world. It is estimated that 80% of the population will have at least one significant episode of low back pain during their lifetime. In the working population, an employer can expect 50 in every 1000 workers to have an episode of low back pain each year. Consequently around 2000 working days are lost each year per 1000 employees: a total of 30 million working days are lost each year in the UK alone. Psychosocial factors, including job satisfaction, have been shown to play a considerable part particularly in those who have been off work for more than 3 months. Litigation and compensation tend to prolong the time to resolution during which illness behaviour may become established.

Clinical problems

- Degenerative disc disease.
- 'Facet joint syndrome'.
- Spinal stenosis.
- Prolapsed lumbar intervertebral disc.
- Spondylolysis and spondylolisthesis.
- Spinal infection.
- Spinal tumour.
- Extraspinal pathology.
- Illness behaviour.

Management

The history, clinical examination and subsequent investigations are aimed at establishing whether the patient has a condition with clear identifiable pathology or whether the symptoms are compatible with one of the low back pain syndromes (idiopathic or 'mechanical' low back pain). In the current state of knowledge (Nachemson, 1992), a consensus view is that surgery is virtually never indicated for the 'low back pain syndromes' because of the lack of validation available for these. Indeed, the whole subject of low back pain

continues to be one of speculation and unsupported assumptions. Unless clearly identifiable deformity, instability or neural compression can be demonstrated, surgery is best avoided.

The majority of patients with low back pain do not require surgery. The natural history in most cases of 'mechanical' low back pain is spontaneous resolution of symptoms within 1 week. For those that last longer, most will respond to a combination of rest, physiotherapy, exercises including swimming, NSAIDs, analgesics, antidepressants, manipulation and occasionally immobilization in a plaster jacket, brace or corset. Avoidance of certain activities that produce pain, elementary ergonomics, awareness of posture and patient education are factors that should be considered but are principally matters of 'common sense'.

Surgery may be indicated when conservative measures have failed to effect sufficient improvement in the patient's condition. Surgery is directed at the relief of neural compression, stabilization of painful unstable motion segments, debridement or drainage of infective lesions, the excision or palliation of spinal tumours and the correction of deformity. These conditions are dealt with in detail in other sections.

Further reading

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Related topics of interest

Acute lumbar disc disease (p. 5)
Degenerative disease of the lumbar spine (p. 116)
Spinal infection (p. 302)
Spinal stenosis (p. 306)
Spinal tumours (p. 310)
Spondyloarthropathies (p. 313)
Spondylolysis and spondylolisthesis (p. 317)

METABOLIC BONE DISEASE

Calcium metabolism

Over 99% of body calcium is in bones. Plasma calcium is distributed equally in bound and unbound forms. It is actively absorbed from the duodenum by an ATP-dependent process regulated by active vitamin D, and is passively absorbed in the jejunum. Ninety-eight percent is reabsorbed in the proximal tubule of the kidney.

Vitamin D3 is a steroid which is activated by UV light or produced from dietary vitamin D2. It is hydroxylated to 25-hydroxy-D3 in the liver and 1, 25 dihydroxy-D3 in the kidney. It promotes mobilization of calcium from bone, increases gut calcium and phosphate absorption by inducing calcium binding protein, and increases phosphate and calcium absorption in the kidney.

Parathyroid hormone (PTH) is an 84-amino acid peptide secreted by the chief cells in the parathyroid glands. It increases serum calcium by mobilizing calcium and phosphate from bone, increases the resorption of calcium from the kidney while increasing secretion of phosphate, stimulates production of vitamin D3 and increases absorption of calcium from the gut.

Calcitonin is a 32-amino acid peptide produced by the parafollicular clear cells in the thyroid. It is released in the presence of high plasma calcium and acts to lower calcium by decreasing osteoclastic resorption. It decreases calcium resorption in the kidney and absorption from the gut.

Clinical problems

1. Hypercalcaemia. Presents with polyuria, constipation, lethargy, disorientation, hyperreflexia, kidney stones, abdominal pain and bone resorption. The most common cause is malignancy.

- *Primary hyperparathyroidism* is caused by overproduction of PTH by an adenoma. Increased osteoclastic resorption produces osteopenia, osteitis fibrosa cystica (fibrous replacement of the marrow), brown tumours (with giant cells, red blood cells and haemosiderin staining) and chondrocalcinosis.

Radiographs show osteopenia, with ill-defined trabeculae and areas of radiolucency. Parathyroidectomy is curative.

Other causes of hypercalcaemia include multiple endocrine neoplasia, Addison's disease, kidney disease, sarcoid and excess alkali ingestion.

2. Hypocalcaemia. This presents with neuromuscular irritability (tetany, Chvostek's and Trousseau's signs), cataracts, a prolonged QT interval and fungal nail infections.

- *Primary hypothyroidism* may be iatrogenic following thyroid surgery. *Pseudohypoparathyroidism* (including Albright's hereditary osteodystrophy) is a rare genetic disorder of PTH receptors in target organs.
- *Renal osteodystrophy* is secondary to insufficient phosphate excretion in chronic renal failure. This is associated with a decrease in the plasma calcium which in turn stimulates PTH production resulting in secondary hyperparathyroidism but the phosphate cannot be excreted. Radiographs may show a 'rugger jersey' spine and soft tissue calcification. The underlying renal disease should be treated. 1-a-Hydroxycholecalciferol may be beneficial.
- *Hereditary vitamin D-dependent rickets* is a rare autosomal recessive disorder with a defect in the 1-hydroxylation of vitamin D3 giving rise to bone deformity. High-dose vitamin D is required.

3. Bone deformity. In rickets there is cupping, increased width and disorientation of the physis. Bone thinning, bowing of long bones, enlargement of the costochondral junctions (rachitic rosary), codfish vertebrae, trefoil pelvis, pathological fractures, Looser's zones and muscle hypotonia are all characteristic features.

- *Vitamin D-deficient rickets and adult osteomalacia* may be caused by dietary insufficiency (usually in Asian immigrants), malabsorption syndromes, renal impairment and chronic parenteral nutrition. The defect in mineralization results in large amounts of unmineralized osteoid. Treat with vitamin D and calcium.
- *Familial hypophosphataemic rickets (vitamin D-resistant rickets)* is an X-linked disorder of impaired renal reabsorption of phosphate with a very low plasma phosphate because of the impaired vitamin D response. Treat with phosphate and vitamin D.
- *Hypophosphatasia* is an autosomal recessive disorder caused by reduced levels of alkaline phosphatase required for bone matrix formation. Treat with phosphate.

4. Osteopenia.

- *Osteoporosis* is an age-related loss of bone mass. *Type I* is oestrogen-dependent post-menopausal loss of trabecular bone which produces vertebral and distal radial fractures. *Type II* is age-related and affects both trabecular and cortical bone to produce hip fractures. Risk factors include being Caucasian, smoking, heavy drinking and phenytoin ingestion (impaired vitamin D metabolism). Hyperparathyroidism, hyperthyroidism, Cushing's and malignancy must be excluded. Plain radiographs are only positive if there is over 30% loss of bone mass. Physical activity and calcium help prevent fractures in type II and oestrogen/progesterone therapy in type I if initiated within 6 years of the menopause. Other causes include osteomalacia, scurvy and marrow infiltrative disorders.

5. Increased bone density.

- *Paget's disease* affects 3% of the population. Increased osteoclastic resorption is accompanied by irregular bone formation with trabecular thickening, bowing and sclerosis. Increased hat size, deafness and high output cardiac failure may be seen. Increased alkaline phosphatase and increased urine hydroxyproline are characteristic. Pathological fractures commonly occur. Calcitonin inhibits osteoclastic bone resorption and diphosphonates (pyrophosphate analogues) act by inhibiting bone turnover. Paget's sarcoma occurs in up to 5% and carries a poor prognosis.
- *Osteopetrosis* is a group of disorders of increased sclerosis and obliteration of the medullary canal due to decreased osteoclast function. The most severe autosomal recessive form leads to hepatosplenomegaly, aplastic anaemia and the 'bone within a bone' appearance on radiographs. Bone marrow transplantation of osteoclast precursors, high dose calcitriol (1,25 dihydroxy-D_3)and steroids may be helpful. The autosomal dominant tarda form (Albers-Schonberg) presents with generalized osteosclerosis and a 'rugger jersey' spine but without haemopoietic problems.
- *Osteopoikilosis* is characterized by islands of cortical bone within the trabeculae and marrow of the long bones in the hands and feet. It is not premalignant.

Further reading

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Related topics of interest

Avascular necrosis of the hip (p. 53)
Osteoarthritis (p. 202)

METATARSALGIA

Metatarsalgia is pain arising from the metatarsal heads. Any condition which causes dorsal subluxation and clawing of the toes may cause the transfer of load from the toes to the metatarsal heads with consequent pain. Painful metatarsal heads may also occur from Freiberg's infraction and march fracture. Morton's metatarsalgia is caused by an interdigital neuroma.

Clinical problems

1. Pressure metatarsalgia. Rheumatoid arthritis and neuromuscular disease commonly cause dorsal subluxation and clawing of the toes. Excessive pressure is borne by the metatarsal heads which become painful. The increased load causes a painful hard callosity over the more prominent lateral side of the metatarsal head. In the first ray this is usually situated under the tibial sesamoid. The lesion has to be differentiated from a viral verruca and the rare neurovascular corn which is characterized by vessels running parallel to the plantar surface of the foot.

2. Freiberg's infraction. This condition most commonly occurs in the second and third decades. It is an example of an osteochondrosis in which avascular necrosis of the second, or rarely the third, metatarsal head is followed by a repair phase. The characteristic clinical features are of pain on weight-bearing, swelling due to synovitis and limitation of extension of the toe. Rarefaction is seen on the dorsal side of the head on early radiographs. Later, fragmentation and broadening of the head occur with thickening of the metatarsal shaft due to increased metatarsal load-bearing.

3. March fracture. This is a stress fracture of the second, third or fourth metatarsal shaft. Insufficiency of the first metatarsal causes the repeated transfer of excessive load on to the other metatarsals which may ultimately fracture. Pain on prolonged walking is associated with oedema of the forefoot. Radiographs show periosteal new bone formation.

4. Morton's metatarsalgia. Middle-aged women are more commonly affected than men and the condition is usually unilateral (85%). Patients complain of intermittent shooting pains or a constant ache in the region of the metatarsal heads, which is worsened by walking and relieved by rest or removal of their

footwear. There is frequently tenderness in the third web space and a palpable click on squeezing the metatarsal heads. If the neuroma is large the toes may be spread. Subjective numbness in the distribution of the common digital nerve is a frequent complaint but there is often no objective sensory loss. The characteristic histological features are perineural fibrosis, demyelination and degeneration of large fibres. The process is degenerative rather than a true 'neuroma'. The aetiology is obscure although ischaemia, entrapment or trauma have all been suggested. Abnormal responses from damaged nerve fibres probably account for the pain. Dorsal or plantar excision is usually followed by good relief of symptoms.

5. Sesamoiditis. Chronic inflammation or trauma may give rise to pain and tenderness localized to the tibial (or occasionally fibular) sesamoid. A bipartite tibial sesamoid occurs in 10% of the population. A bipartite fibular sesamoid is rare. Chondromalacia, osteochondritis dissecans and tendonitis of the flexor hallucis brevis may all occur. Sesamoid shaving or excision is indicated if conservative measures fail.

6. Verruca vulgaris. Viral plantar warts are associated with sharp margins and vessels running perpendicular to the surface. Paring by a chiropodist and curettage are the mainstays of treatment.

Management

1. Assessment. An accurate history and examination are essential. Radiographs will reveal a Freiberg's infraction and march fractures. Electromyographic studies may help in doubtful cases of Morton's neuroma but are not specific. Footprint analysis by simple Harris-Beath mats or by electronic force plates will reveal patterns of abnormal load-bearing in the foot.

2. Footwear. A broad thick-soled shoe with a metatarsal bar to relieve pressure may be sufficient. A metatarsal dome insert may be strapped on to the foot or may be part of an insole in the shoe. It relieves forefoot pressure if placed just behind the metatarsal heads. A short-term plaster cast may be necessary to ameliorate symptoms in those cases of Freiberg's and march fracture that do not settle with simpler remedies.

3. Metatarsal osteotomy. Patients with metatarsalgia which fails to respond to toe surgery and conservative measures will respond to an oblique metatarsal shaft osteotomy (Helal) in 85–90% of cases. This may be followed by increased loading on the remaining metatarsals.

4. Arthroplasty of the MTP joint. Those with a small intractable plantar keratosis beneath the lateral condyle of the metatarsal head will respond to shaving of the undersurface of the metatarsal head in 95% of cases.

5. Freiberg's infraction. Many treatments have been advocated including resection of the metatarsal head, resection of the base of the proximal phalanx, drilling and debridement of the defect and closing wedge osteotomy of the metatarsal head.

Further reading

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Related topics of interest

Hallux valgus (p. 140)
Lesser toe deformities (p. 178)

MUSCULOSKELETAL IMAGING

The wide variety of imaging techniques available permit greater accuracy in the diagnosis of musculoskeletal disease. Many of the newer techniques avoid irradiation but the most frequently used imaging techniques in orthopaedics continue to use X-rays.

Imaging using X-rays

1. Plain radiography remains the most useful method of diagnostic imaging, giving valuable information about the architecture of bones, joints and soft tissues.

2. Contrast studies. Most contrast media are iodine-based liquids; some are ionic water-soluble iodides while others are oil-based non-ionic compounds. The simplest form of contrast investigation is the *sinogram* or the *fistulogram*.

3. Arthrography has been widely used to image large joints. It can identify loose bodies, irregularities of articular cartilage and tears or deficiencies in ligaments. The use of a double-contrast technique allows a high degree of accuracy. In recent years, MRI has been preferred to arthrography in certain situations because it is non-invasive.

Arthrography of the knee was traditionally used to diagnose meniscal injuries, cruciate ligament deficiencies and cysts. It has gradually been supplanted first by arthroscopy and, more recently, by MRI.

Hip arthrography in children will outline the cartilaginous non-radio-opaque structures used to assess reduction in DDH and can be used to assess congruency in Perthes' disease, slipped upper femoral epiphysis and skeletal dysplasia. In adults, it is used to determine joint congruency in degenerative joint disease or avascular necrosis prior to surgical treatment. In the evaluation of loose total joint replacements, arthrography has given way to scintigraphy.

Shoulder arthrography remains a reliable method of demonstrating a tear of the rotator cuff and, in conjunction with CT, is a valuable way of demonstrating the anatomical abnormalities associated with shoulder instability. At the *elbow,* arthrography is principally used to demonstrate loose bodies or osteochondritis dissecans. At the *wrist* it is used to assess carpal instability or to identify tears of the triangular fibro-cartilage complex (TFCC).

4. Myelography has largely been replaced by non-invasive procedures (CT and MRI) in the assessment of lumbar disc disease. It is still used in some centres as an adjunct in the investigation of central spinal stenosis, and remains a valuable investigation when assessing acute central disc protrusion and spinal injuries. It is also used in the evaluation of cervical nerve root problems, often in conjunction with CT.

Oil-based contrast media are no longer used as they can produce chronic arachnoiditis. Metrizamide, a non-ionic iodine compound, is currently thought to be the least toxic and is isotonic with cerebrospinal fluid. It may be used throughout the length of the spinal cord if required.

5. Discography was used to assess the extent and severity of degenerative disc disease but this function has been replaced by MRI. It is, however, still used provocatively to identify a symptomatic motion segment.

6. Tomography generates a series of focused X-ray images at a specific distance to produce serial 'cuts'. These are useful in defming certain fractures (e.g. tibial plateau) and in the assessment of non-union and partial physeal arrest. Many of its previous roles have now been replaced by CT.

7. Xeroradiography. A technique using conventional X-rays with a positive image on special plastic-coated paper. It is a more sensitive method of examining soft tissues.

8. Computerized tomography (CT) has resolution characteristics which are superior to those of plain radiographs. It generates a series of axial images which can be reconstructed to provide three-dimensional information. CT can demonstrate small differences in tissue density: this allows good definition between tissues which can be enhanced by using a contrast medium. Cross-sectional slices of between 1 and 10 mm are possible. Its main applications are in the evaluation of spinal pathology, bone tumours, pelvic and hip abnormalities and fractures.

Radionuclide imaging

Photon emission by radio-isotopes can be picked up by a gamma camera and used to produce images.

Technetium (^{99}Tc) has a half-life of 6 h and is rapidly excreted, making it ideal for clinical use. When injected intravenously, technetium-labelled phosphates or diphosphonates become bone-seeking isotopes. Activity is recorded shortly after injection (the 'blood pool' phase) which reflects vascularity and at 3 hours (the 'bone phase'). Increased uptake in the blood pool phase reflects increased vascularity in the soft tissues, the commonest cause of which is inflammation. Increased uptake in the bone phase is the result of the increased osteoblastic activity of fracture healing, infection or tumour. Decreased uptake in the bone phase reflects avascularity (e.g. avascular necrosis or acute vascular insufficiency after a subcapital fracture). The main uses of technetium scanning are in the detection of metastatic deposits in bone; the diagnosis and

localization of early bone abscess or osteomyelitis; the diagnosis of stress fractures not seen on plain radiographs; the localization of osteoid osteomata and the diagnosis of femoral head ischaemia in Perthes' disease or avascular necrosis.

Gallium (^{67}Ga) is concentrated in inflammatory cells and can be used to detect infection. ^{111}Indium-labelled white cells can also be used to help localize infection.

Magnetic resonance imaging (MRI)

MRI measures the distribution density of hydrogen nuclei in tissues. It records the motion of the hydrogen nuclei measuring their relaxation times (Tl and T2). Compared to X-ray techniques, soft tissue contrast is superior and this can be further enhanced by Tl and T2 weighting. Specific sequences are produced by varying the transmitted radio-waves which helps to differentiate between specific types of tissue (inversion recovery, spin echo and saturation recovery). MRI allows scanning in transverse sagittal, coronal and oblique planes. High contrast between the muscle and fat results in clear definition of muscle planes. The dynamic flow characteristics of tissues mean that fast-flowing blood produces little signal compared to static or slow-flowing blood: it is therefore possible to distinguish between nerves, arteries and veins. Imaging of specific regions can be enhanced using surface coils: these improve signal quality and spatial resolution. Gadolinium injection is used if further defmition is required.

1. Tumours. The margins of both bone and soft-tissue sarcomas can be demonstrated very precisely. Malignant tumours often have prolonged Tl and T2 relaxation times. MRI is the most sensitive method of assessing intramedullary tumour extent as well as extraosseous spread. Image degradation due to metallic implants is less than with CT and MRI may be used to assess recurrence after surgery even in the presence of endoprosthetic replacements.

2. Spine. Normal nucleus pulposus gives a high signal on T2-weighted images due to the high water content. The spinal cord and surrounding epidural fat are both well visualized on Tl-weighted images whereas CSF and theca are seen more clearly on T2-weighted images. Disc disease, cord compression and nerve root compression are, therefore, well imaged. Spinal infection and inflammation are more specifically located than with radio-isotope scans. MRI is superior to X-rays for the diagnosis of osteomyelitis or discitis. Spinal tumours are well localized and the extent of the surrounding cord swelling is well defined. Conditions within the spinal canal such as demyelination, syringomyelia and dysraphism can be diagnosed.

3. Knee. MRI of the knee has superseded arthrography and is comparable in diagnostic accuracy to arthroscopy in the diagnosis of meniscal and cruciate ligament deficiencies. Meniscal tears are seen as a focus of high intensity (white areas within a normally low signal meniscus). Cruciate ligament tears, cysts, effusions and osteochondritis dissecans are well defined.

4. Hip. MRI is used for assessing AVN and, using a surface coil, labral tears and synovial pathology can be identified.

5. Shoulder. Rotator cuff tears and glenoid labral tears can be located.

Diagnostic uitrasound

High frequency sound waves with real-time imaging allow a dynamic examination. The technique is non-invasive and the equipment is highly portable. Diagnosis is, however, highly dependent on the operator. Because of a marked contrast in the echogenicity between solids and fluids, it is particularly useful in identifying haematomas (haemophilic cyst resolution), abscesses and intra-articular fluid (e.g. diagnosis and aspiration in irritable hip). It can be used in the diagnosis of prenatal conditions (developmental dysplasia of the hip, proximal femoral focal deficiency and limb agenesis). Its common role in orthopaedics is in the postnatal assessment of DDH and hip dysplasia.

Quantitative bone mineral analysis

Bone densitometry may be used to assess the progress of metabolic bone disease, osteoporosis and fractures. Two techniques are used, photon absorptiometry (PA) and X-ray absorptiometry. Single source techniques (SPA) primarily measure cortical bone and are most accurate in monitoring the density of the distal third of the radius. Double photon absorptiometry (DPA) uses two distinct photon energy sources (gadolinium) and allows measurements from the lumbar spine and the hips specifically as well as whole body measurements. Dual X-ray absorptiometry (DEXA) allows more precise and accurate measurements and can be used to determine both peripheral and central skeletal density. It is typically used in the lumbar spine and hip, and allows both anteroposterior and lateral measurements to be taken. Quantitative CT (QCT) can differentiate trabecular from cortical bone both in the periphery and centrally and is highly sensitive for measuring change in density in metabolic bone disease.

NEUROMUSCULAR DISORDERS

Neuromuscular disorders and their orthopaedic sequelae may be caused by disease of the upper motor neurone (e.g. cerebral palsy), the lower motor neurone (e.g. poliomyelitis) or of muscle. Severe or advanced cases are easy to classify but others can cause diagnostic difficulty. Problems arise because of instability from muscle weakness or joint abnormality and from deformity due to relative shortening of soft tissues, muscle imbalance and impairment of coordination.

Clinical problems

1. Cerebral palsy is used as an inclusive term to describe a group of non-progressive disorders occurring in young children in which disease of the brain causes impairment of motor function. The changes seen in the musculoskeletal system with time are due to the resulting abnormal growth and development of the child. The incidence is approximately 1 per 1000 live births. The diagnosis is often missed at birth particularly when this is premature. Classification is either neurological:

•	spastic	75%
•	athetoid	10%
•	ataxic	
•	atonic	8%
•	rigid	
•	mixed	7%

or anatomical:

- hemiplegia 35%
- diplegia 35%
- quadriplegia (total body involvement) 30%.

A useful functional classification defming walking ability and sitting ability helps management.

2. Poliomyelitis. A viral infection affecting the anterior horn cells in the spinal cord and brain stem producing a flaccid motor paralysis. The paralysis becomes maximal at 2-3 weeks. It may recover completely but any residual paralysis at 6 months will be permanent. Fibrous contractures develop over 4-6 months in the hamstrings, calf, pectoral muscles and elbow flexors as a primary feature of the disease. Secondary deformity due to muscle imbalance develops after approximately 1 year.

3. Spinal muscular atrophy. An autosomal recessive condition affecting the anterior horn cells and cranial nerve nuclei. It may present at birth or may not become evident until adolescence. The lower limbs are affected more than the upper limbs, and the proximal muscles more than distal.

4. Hereditary neuropathies and spino-cerebellar degeneration. The *hereditary motor and sensory neuropathies* include peroneal muscular atrophy (PMA) and Charcot-Marie-Tooth disease which are both characterized by slowly progressive distal weakness in the lower limbs. There are two main types, an autosomal dominant variety which manifests itself in adult life and an autosomal recessive variety which appears in childhood. The earliest signs are of pes cavus, with subsequent wasting and weakness of the intrinsic muscles of the foot. This spreads to involve the peroneal muscles, producing a superimposed varus deformity, and later the tibialis anterior, to produce a drop foot. Later the small muscles of the hand and forearm muscles are involved. EMGs show denervation.

Friedreich's ataxia is a dominant or recessive familial condition which presents in childhood as a cerebellar ataxia but which later develops features of peroneal muscular atrophy. It is progressive, with cardiomyopathy and dementia leading to death in the third decade. Surgery is usually unrewarding because of the ataxia.

5. Spina bifida (see p. 296).

6. Arthrogryposis is probably an intrauterine neuropathy and is characterized by joint stiffness and contractures. Knee deformities, clubfoot, and hip dislocation are common (see Further reading).

7. Muscular dystrophy is a primary disorder of muscle classified on genetic inheritance and clinical presentation (e.g. Duchenne: sex-linked recessive; limb girdle dystrophy: autosomal recessive; facioscapulo-humeral dystrophy: autosomal dominant). The commonest is Duchenne, which is often unsuspected until the child walks. Muscle bulk pseudohypertrophy is due to fat deposition and hides wasting. Patients die in their late teens and 20s due to respiratory failure. Spinal fusion can prolong their life expectancy.

Treatment

1. Cerebral palsy. Generally hemiplegics will walk unaided by 2–21/2 years and require an energy-efficient gait with a cosmetically acceptable upper limb.

Diplegics will need assistance but most will walk by 5 years. They need an energy-efficient gait particularly as they get older and their weight increases. Quadraplegics will not be able to walk but need a stable trunk and hips to enable them to sit upright.

Non-operative treatment consists of *physiotherapy,* which is aimed at teaching patterns of posture and movement, and *splinting or bracing* for support and to maintain correction of deformity.

Operative treatment has a limited role. The overall results of treatment depend on the mental function of the patient. The best results are obtained in patients with hemiplegia. Surgery is aimed at the prevention and correction of deformity and at achieving a reduction in the energy cost of walking. It is essential to operate on the primary abnormality and not on abnormalities from 'coping' responses. Gait analysis can help to differentiate between the two. The principal techniques employed are *soft tissue releases, muscle transfers* (e.g. Egger's hamstring transfer) and *osteotomies* to correct deformity (e.g. tibial osteotomy) or stabilize joints (e.g. femoral osteotomy to improve hip stability). *Dorsal selective rhizotomy* can help diplegics with good motor control to improve their gait efficiency.

2. Poliomyelitis. The *extent of involvement* must be charted accurately. This should include electromyography (EMG). *Prevention of secondary deformity* by early passive stretching and splinting is imperative. *Management of early deformity* is by physiotherapy using passive and active exercises with night splintage as necessary. Particular attention should be paid to flexion contractures at the hip and knee and equinus deformity at the ankle. Involvement of the ilio-tibial tract can produce spinal, hip and knee deformity. The management of *permanent paralysis* depends on the nature of the deformity. This is discussed in the sections on the paralysed hand and lower limb.

3. Hereditary neuropathies and spino-cerebellar degeneration. Early treatment of the cavus foot and clawing may prevent fixed deformity. In a *flexible foot* without secondary deformity, division of plantar fascia with release of abductor hallucis, abductor digiti minimi and the short flexors of toes may be undertaken. This is followed by toe surgery with flexor to extensor transfer in the lesser toes and a Robert Jones procedure on the hallux. When peroneal weakness becomes evident, transplantation of tibialis posterior to the dorsum of the fourth metatarsal base prevents deformity. In the *rigid foot* with fixed deformity, a wedge tarsectomy or triple arthrodesis with tibialis posterior transfer is necessary.

4. Muscular dystrophy is investigated by muscle biopsy, serum enzymes (especially creatine phosophokinase and transaminases) and electo-myography and nerve conduction studies which will distinguish it from a neuropathic process.

Non-operative treatment consists of *physiotherapy* to maintain a range of passive movement and to reduce progressive deformity, *active exercises* to strengthen normal muscles and *bracing* to hold the corrected deformity and

support weak muscles. *Surgery* may be needed to correct contracture or deformity.

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Related topics of interest

Clubfoot (p. 85)
Developmental dysplasia of the hip (p. 119)
Gaitanalysis (p. 131)
Genetic abnormalities (p. 134)
Paralysed hand (p. 214)
Paralysed lower limb (p. 218)
Spina bifida (p.296)

ORTHOPAEDIC MANIFESTATIONS OF HAEMOPHILIA

Haemophilia is a sex-linked recessive disorder affecting males, although one-third present as a result of spontaneous mutations. Many clotting deficiencies have been described of which the most important are haemophilia A (factor VIII deficiency) and haemophilia B (Christmas disease-factor IX deficiency).

The clinical effects of haemophilia depend on factor activity in the plasma. Patients may be mildly affected (50–100% of normal activity) with no clinical bleeding disorder, or may bleed severely after minor injury or occasionally spontaneously (1–5%), or may present with frequent spontaneous bleeds (0%). These last two groups can develop a haemo-arthropathy.

The mainstay of treatment for haemophilia is factor replacement. Self-administration immediately after injury or the onset of symptoms reduces morbidity. Infection with HIV from pooled blood products has had a major effect on the medical and surgical treatment of haemophilia. Not only are surgical staff at risk of cross-infection but the surgical insult can precipitate clinical AIDS in HlV-positive patients.

Problems

1. Acute soft tissue bleeds. Muscle bleeds may be spontaneous or may follow trauma. Treatment consists of compression bandaging, factor replacement and splintage. There is no place for surgery. Resolution is monitored by clinical examination and serial diagnostic ultrasound examinations. Bleeds into pelvic or para-spinal soft tissues may require prolonged bed rest and serial MRI or CT scans.

2. Acute haemarthrosis. Fifty percent of all bleeds occur into the knee, 25% into the elbow and 15% into the ankle. Acute bleeds into the hip joint are rare but chronic complications often develop (see below). Management is similar to acute soft tissue bleeds.

3. Haemophilic cysts and pseudotumours are produced by an expanding haematoma which is not controlled by adequate treatment. The pelvis and femur are the commonest sites in adults although the hands and feet are most commonly affected in children.

Simple cysts are contained within the fascial envelope of a muscle, do not involve bone and occur after approximately 3% of muscle bleeds. Cysts arising in muscle can involve the periosteum and can cause cortical thinning. *Pseudotumours* arise from subperiosteal or interosseous haemorrhage. Before factor replacement, the mortality from pseudotumours was more than 50%.

4. Chronic progressive synovial proliferation occurs as the result of haemosiderin deposition from repeated subacute haemarthrosis. It is therefore seen most commonly in the knee and elbow. The hyperaemic synovium is prone to further bleeding and a cycle of bleeding and haemosiderin deposition develops.

5. Chronic haemophilic degenerative arthropathy. The hip and elbow are the most common sites. Repeated bleeds cause progressive degenerative articular damage which can be severely destructive and debilitating. Analgesia, physiotherapy and orthoses are the primary treatment but surgery may be needed. Bleeding into the hip may raise the intra-articular pressure and cause avascular necrosis of the femoral head. The incidence of hip disease is therefore greater than might be expected from degenerative disease alone.

6. Severe progressive soft tissue contracture. Contractures that fail to respond to serial splinting can occur at the elbow and knee. Functionally, knee contractures are more significant and may require surgery (soft tissue release or osteotomy). This condition is less common with modern treatment.

Management

1. Radiology. Plain radiographs will identify pseudotumours which cause localized osteolysis with ill-defined margins and soft tissue shadowing. If subperiosteal, they elevate the periosteum and may mimic a primary bone tumour. Diagnostic ultrasound is useful in diagnosing soft tissue bleeds into the limbs and monitoring their resolution.

2. CT and MRI scanning are used for more inaccessible lesions, particularly in the pelvic and spinal musculature.

Treatment

1. Non-surgical. Compression bandaging, splintage and factor replacement is the standard treatment for acute bleeds and is followed by physiotherapy and rehabilitation. Chronic arthropathy is supported non-operatively as far as possible.

2. Surgery. Surgery in the management of haemophilia is limited. The presence of a ‘factor inhibitor’ is a contraindication to *elective* surgery. Standard surgical approaches and techniques should be applied. A pneumatic tourniquet is used wherever possible and only released when pressure dressings and splints are in place. Meticulous haemostasis, closure of dead space and the use of continuous haemostatic sutures is advisable. Drains and percutaneous pins are to

be avoided. Procedures should be planned well in advance, adequate factor cover is required and full HIV precautions are used by theatre staff for every procedure.

- *Total joint replacement.* Total hip and total knee replacements have been performed satisfactorily in haemophiliacs. Standard techniques using cemented prosthesis are recommended.
- *Synovectomy.* Open surgical synovectomy has been successful in the past but arthroscopic synovectomy has a reduced morbidity.
- *Arthrodesis.* Arthrodesis of the knee and ankle using internal fixation has certain advantages over arthroplasty in young individuals. It is a single procedure providing permanent relief of pain that should not require revision. Procedures which involve minimal bony excision and internal fixation are recommended.
- *Other* orthopaedic procedures for conditions not directly caused by haemophilia can be performed. Arthroscopic meniscectomy and removal of loose bodies have been described. Theoretically, any surgical procedure can be performed under appropriate factor cover but because of the attendant risk of cross-infection and the danger of sero-conversion following surgery, surgery is avoided wherever possible.

Complications

The complications of haemophilia are outlined above. Those of its treatment are an increased incidence of wound infections and delayed healing resulting from haematomata. Prolonged splintage causes joint stiffness. Currently, the main problems are related to the transmission and activation of HIV.

Further reading

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Forbes CD, Lowe GD. *Vnresolved Problems in Haemophilia.* Lancaster: MTP Press.

Related topic of interest

AIDS (p. 9)

OSTEOARTHRITIS

Autopsy studies have shown that the incidence of osteoarthritis is approximately 80% in the hip, knee and elbow at the age of 40 years, and rises to over 90% at the age of 80 years. However, its clinical features do not correlate well with radiological or pathological fmdings.

Problems

1. Aetiology. General 'wear and tear' is still considered an important aetiological factor. Changes due to aging of the articular cartilage are seen in areas of low contact stress, while progressive changes leading to osteoarthritis are seen in areas of high contact stress. The knee and hip joint are most commonly affected. The incidence in limbs paralysed by poliomyelitis is lower, suggesting that the presence of stability is not in itself preventative and that force transmission may be more important. There is no clear relationship between arthritis and obesity but the incidence is higher in diabetics. There appears to be a genetic predisposition to arthritis affecting the fingers (causing Heberden's nodes), posterior spinal facet joints and the knees. Primary OA has no obvious underlying aetiology while secondary OA follows trauma, slipped upper femoral epiphysis (SUFE), Perthes' disease, sepsis, etc.

2. Pathogenesis. Under compressive loads some of the superficial chondrocytes release lysosomal enzymes which cause breakdown of proteoglycan matrix. Loss of the ground substance results in relative dehydration, with loss of the ability of the cartilage to withstand compressive and shear forces. The net result is fibrillation and fissuring of the articular cartilage. More cell death follows with further release of degradative enzymes, thereby perpetuating the cycle of damage. In eroded areas the subchondral bone collapses with microfracture of the trabeculae and new bone formation.

3. Grading. Grade I change is characterized by superficial damage to the cartilage in load-bearing areas, with early fibrillation, pitting, grooves and blisters. There are no changes in the bone or synovium. *Grade II* change has more extensive destruction of cartilage in the load-bearing areas with deeper fibrillation and flaking. Early small osteophytes are seen. *Grade III* change is a total loss of cartilage in load-bearing areas with eburnation of the underlying

bone. Fibrillation and flaking of the remaining cartilage is usually present and there is sclerosis and deformation of articular surfaces with osteophyte formation. *Grade IV* changes include the complete loss of cartilage from large areas of the joint surface. Osteophytes are prominent and remodelling of the bone ends occurs. Capsular fibrosis gives rise to fixed deformity.

4. Natural history. The general tendency is for osteoarthritis to deteriorate but the rate at which this happens is variable. Up to two-thirds of patients with the lesser degrees of articular cartilage damage which are seen in chondromalacia patellae have symptoms which resolve in adult life.

5. Clinical Aching is related to the synovitis associated with degradation products. Since articular cartilage does not have a nerve supply, pain only originates from the bone if it is directly involved. Collapse of trabecular bone, new bone formation and hypervascularity are associated with the constant pain which is often felt at night. Stiffness and deformity follow insidiously. Swelling may be due to synovitis, effusion or osteophyte formation. Crepitus is often felt.

6. Radiological. Radiological features often lag a long time behind pathological features. There is often little correlation between the clinical features and the radiological appearances. The characteristic sequence of changes is narrowing of the joint space, formation of osteophytes at the articular margins, sclerosis and cyst formation. Osteophyte formation is due to metaplasia of fibroblasts of the synovial membrane. The new bone formation in response to trabecular microfracture accounts for the radiographic fmding of sclerosis.

Management

1. Tablets. Simple analgesics are appropriate for mild disease. NSAIDs inhibit prostaglandin synthesis and 70% of patients will show a satisfactory response. Femoral head collapse has been reported with the use of NSAIDs but appears to be an idiosyncratic reaction.

2. Intra-articular injection. Intra-articular injection of local anaesthetic and steroid tends to provide short-term relief of symptoms. Bone destruction has been observed following steroid injection.

3. Physiotherapy. Heat, cold, short wave diathermy and ultrasound may lead to symptomatic improvement. Exercises may restore mobility and alleviate pain. Use of a walking stick and adjustments to the home environment may also help.

4. Arthrodesis. This is particularly useful in the upper limb where pain-free stable joints are the goal of treatment.

5. Osteotomy. An osteotomy may alleviate pain by redistributing the forces around a joint so that more load is borne by relatively intact articular cartilage. The restoration of the joint space is due to fibrocartilage rather than articular cartilage. Pain relief may be partly due to relief of venous hypertension.

6. Arthroplasty. This is the most certain way to relieve pain and restore function.

7. Drilling. Arthroscopic lavage and drilling of the damaged joint surface in the early osteoarthritic knee does appear to produce relief of symptoms in about 70% of patients. The upward passage of cells from the marrow is accompanied by proliferation and metaplasia to form fibrocartilage. This layer does not, however, provide a lasting durable lining and hence the effect is likely to be temporary. Continuous passive motion may help this process of extrinsic repair by diffusion of nutrients in the synovial fluid.

8. Osteochondral allografting. Transplantation of osteochondral allografts has been accompanied by some success. Load-relieving osteotomy is recommended.

Further reading

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Related topics of interest

Arthritis of the hip (p. 35)
Arthritis of the knee (p. 39)
Hip arthroplasty (p. 150)
Osteotomies around the knee (p. 211)

OSTEOCHONDRITIS DISSECANS

The most common presentation is in a child aged between 10 and 13 years. Boys are affected twice as commonly as girls. Bilateral lesions are present in 25%.

In osteochondritis dissecans there appears to be an underlying abnormality of the epiphysis. A history of injury is present in half the patients. Endogenous trauma from discoid menisci or tibial spines, ischaemia, abnormal ossification and hereditary influences have all been suggested as possible causes. The condition needs to be distinguished from osteochondral fracture and chondral flaps and separations which are traumatic in origin.

Developing lesions are seen on radiographs as a single area of ossification which has developed separately from the main epiphysis. Neither the fragment nor the defect is corticated. In late lesions, concave defects with loose bodies arising from them are seen, in contrast to the flat defects of osteochondral fractures. Pain is the usual presenting feature: symptoms of a loose body may occur if the fragment separates.

The radiographic signs are diagnostic. Stage I shows subchondral compression; stage II lesions have a flap of osteochondral fragment which is still attached; stage III lesions are separated but still within the bone crater and stage IV lesions have progressed to produce a loose fragment.

Clinical problems

1. Knee. Eighty-five percent occur on the medial femoral condyle with 69% in the classical site on the lateral aspect of the medial femoral condyle. This is the area of contact of the patella during full flexion. Trauma to the patella in this position may explain the high incidence in this area. Lesions on the lateral condyle may be related to dislocation of the patella. Lesions uncommonly occur on the patella in association with patellar subluxation.

2. Talus. Forty-three percent occur in the middle third of the lateral portion of the talus and 57% on the medial surface, usually posteriorly. Trauma appears to be responsible for most of the lateral lesions and at least half of the medial lesions.

3. Capitellum. Usually seen in the throwing arm of athletes because of valgus stress causing compression of the radial head and the capitellum.

4. Hip. In the absence of Perthes' disease, sickle cell disease, Gaucher's, multiple epiphyseal dysplasia and avascular necrosis osteochondritis dissecans can be diagnosed. This rare condition affects the weight-bearing area of the femoral head.

Management

1. Children. The majority of children can be treated expectantly as 50% will heal with a good or excellent result. However, large defects on the weight-bearing surfaces and defects on the lateral femoral condyle are associated with osteoarthritis.

2. Early separation. In cases of early separation where the fragment is not displaceable, drilling through the fragment will produce radiographic healing in 82% by 1 year. Immediate weight-bearing is permitted and pain relief is usually very rapid. If there is a displaceable fragment of recent origin which is still attached, the base should be curetted or drilled before stabilization of the fragment by pins, wires, bone pegs or a Herbert bone screw. Weight-bearing should be protected until there is evidence of union. If the fragment is old, it should be removed and the defect curetted or drilled.

3. Loose body within the crater. If the lesion is in the non-weight bearing area remove the fragment, curette and drill the crater. If in the weight-bearing area consider reattachment after preparing the defect. An allograft, osteotomy or hemi-arthroplasty can be considered if the symptoms are very severe.

4. Talus. Displaced lateral lesions often fail to unite and should be treated by early excision of the fragment and curettage of the base. Undisplaced lesions and displaced medial lesions can be treated by plaster immobilization. Approximately 50% of displaced medial lesions will require surgery.

5. Elbow. In a young child, the arm should simply be rested. In the older child or adult, loose bodies may need to be removed and the capitellar defect drilled. Few athletes return to their previous standard of competitive throwing.

6. Hip. In children with minimal symptoms, observation is recommended. In the adult with severe symptoms, removal of the fragment, internal fixation of the fragment, drilling and grafting of the defect have all been reported with favourable results.

Complications

At an average follow-up of 33 years, 32% had radiographic evidence of moderate or severe osteoarthritis. Osteoarthritis is more likely if the defect is large or on the lateral femoral condyle.

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Related topics of interest

Anterior knee pain (p. 25)
Arthritis of the knee (p. 39)
Arthroscopy (p. 49)
Avascular necrosis of the hip (p. 53)

OSTEOMYELITIS

Acute infection of bone occurs directly by contamination of an open wound (exogenous) or indirectly by blood-borne (haematogenous) spread. The infection may be bacterial, tubercular, spirochaetal, fungal or parasitic. Haematogenous osteomyelitis is caused by a single organism; exogenous infections may be mixed. The commonest causative organism is *Staphylococcus aureus* (80%); next come *Streptococci,* which, with *Haemophilus influenzae,* are frequently responsible for osteomyelitis in children under 2 years. Gram-negative and anaerobic organisms are commonly isolated after trauma. Patients with sickle-cell disease are predisposed to *Salmonella* infections.

Acute blood-borne bacterial infection is commonest in children. The infection localizes in the metaphysis because of its rich blood supply. Pus spreads through the Haversian canals under pressure lifting the periosteum, which responds by laying down new bone. The combination of medullary spread and periosteal stripping leads to necrosis of the infected cortical bone. Simultaneous osteomyelitis and septic arthritis can occur in joints in which the synovial membrane extends down to the metaphysis or in the neonate where blood vessels cross the physis.

Osteomyelitis may become chronic as the result of inadequate treatment, a highly virulent organism or impaired host resistance. A chronic (Brodie's) abscess containing thick pus and fragments of necrotic bone (sequestrum) develops in a cavity surrounded by sclerosed bone. Separating the diseased bone from normal tissues is new bone (involucrum) laid down by the periosteum. Defects in the involucrum (cloacae) allow the continued leakage of pus which may result in sinus formation.

Clinical problems

1. Childhood osteomyelitis. Occurs most commonly in boys aged 7–8 years. The overall incidence is declining. Often there is a history of injury, a febrile illness or an infective focus. The child presents with pain in the limb and a reluctance to use it. He may be toxic or only vaguely unwell. The earliest physical finding is metaphyseal tenderness; erythema occurs much later. Local swelling indicates subperiosteal pus and fluctuance suggests a subcutaneous abscess.

2. Neonatal osteomyelitis. The diagnosis may go unsuspected because 75% are not acutely unwell at presentation. The proximal femur is involved in 45% of cases. If the infection spreads to the hip joint (Tom Smith's arthritis) the prognosis is poor. Local swelling and pseudoparalysis are common. Radiographs may show rarefaction of the affected bone and widening of the joint space.

3. Disseminated staphylococcal infection. This is of rapid onset with multisystem involvement. Most patients become toxic and shocked. The mortality is 13%: 38% have long term complications.

4. Subacute osteomyelitis. Insidious onset with a 1–2 week history of pain but minimal loss of function. The clinical picture is of tenderness without toxicity. Open biopsy and culture may be necessary to exclude malignancy. *S. aureus* is usually isolated. The prognosis is good after treatment with appropriate antibiotics.

5. Post-traumatic. Open fractures or penetrating injuries to bone may be complicated by osteomyelitis. Aim to prevent infection by thorough debridement, skeletal stabilization, soft tissue cover and antibiotics. Infected non-union of a fracture has a poor prognosis.

6. Chronic osteomyelitis (see below).

Management

1. Establish diagnosis. Plain radiographs are usually normal for the first 10 days. Later they may show soft tissue swelling, rarefaction of bone, periosteal elevation or a Brodie's abscess. Elevation of the white cell count and sedimentation rate are suggestive of infection but normal values do not exclude the diagnosis, particularly in neonates. Blood cultures will identify the infecting organism in 50% of cases. Scintigraphy is helpful in the spine and pelvis. It is positive in about 80%: false negatives occur in neonates. If doubt persists, an open biopsy is advised. The differential diagnosis includes trauma, tumour, cellulitis and erysipelas.

2. Resuscitation. Correction of dehydration is essential.

3. Antibiotics. Use 'best guess' antibiotic initially: flucloxacillin 200 mg/kg/day i.v. for 72 hours, then oral 100 mg/kg/day for at least 6 weeks. In neonates add fucidin 30 mg/kg/day for resistant staphylococci and ampicillin 150 mg/kg/day for *Haemophilus influenzae*. Use broad-spectrum antibiotics in post-traumatic osteomyelitis and include metronidazole for anaerobic organisms.

4. Surgical drainage. Open drainage is advised if there is swelling suggestive of an abscess, a severely toxic child or a failure to respond to antibiotics. The cortex is drilled to decompress the medulla and the wound loosely sutured over a drain.

5. Splintage. Relieves pain and aids healing.

Complications

1. Chronic sepsis. Repeated episodes of inflammation or sinus formation denote chronic infection. Tomograms or a CT scan may be necessary to identify a Brodie's abscess. Treat by excision of sinus tract, sequestrectomy and saucerization of cavities. The wound may be closed and the cavity bone grafted at a second operation after 10–14 days. Alternatively, the wound can be left open and irrigated daily until clean enough to allow bone grafting and delayed skin closure (Papineau). Consider excision of infected bone and bone transport for intractable cases. Long term antibiotics may be required. Amyloid and squamous cell carcinoma of the sinus occur rarely.

2. Pathological fracture. Avoid internal fixation.

3. Growth arrest or defonnity.

Further reading

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Related topics of interest

Leg length inequality (p. 174)
Musculoskeletal imaging (p. 190)
Septic arthritis (p. 272)
Skeletal tuberculosis (p. 284)

OSTEOTOMIES AROUND THE KNEE

Proximal tibial and distal femoral osteotomies are performed for arthritis or deformity around the knee. They may also be undertaken to unload a unicompartmental osteoarticular allograft. An osteotomy of the tibial tubercle (e.g. the Maquet osteotomy) may be performed for patello-femoral pain and a derotation osteotomy for torsional deformity. Genu recurvatum may be corrected by creating an anteriorly based opening-wedge or a posteriorly based closing-wedge tibial osteotomy.

Indications

1. Pain. The main indication is significant pain and disability from osteoarthritis.

2. Age. Best results are produced in patients under the age of 60 years at operation.

3. Unicompartmental disease. There should be radiographic or arthroscopic evidence that disease is confined to one compartment. Severe bone depression usually indicates involvement of the other compartment.

4. Stability. Instability is a contraindication.

5. Adequate range of movement. A flexion deformity of no more than 20° and a range of movement to at least 90° are required.

6. Varus deformity. A varus deformity of less than 10° is easily corrected by using a closing-wedge valgus osteotomy. Deformities of more than 10° are often accompanied by stretching of the lateral soft tissues and instability. Coventry recommends soft tissue reefing of the lateral side after osteotomy. Deformities of more than 10° may be difficult to correct with a closing wedge because there is insufficient room to remove a wedge of adequate size from the lateral side. Maquet recommends a barrel vault osteotomy to overcome these problems and believes that the soft tissues tighten after the deformity is corrected.

7. Valgus deformity. Advanced osteoarthritis of the lateral compartment is accompanied by loss of substance of the lateral femoral condyle and laxity of the medial collateral ligament. If a varus closing-wedge osteotomy is undertaken for this condition, it produces obliquity of the knee joint axis and a tendency for the femur to subluxate medially on the tibia. An obliquity of less than 10° does not appear to be clinically significant and medial tibial closing wedge osteotomy

with medial soft tissue reefing may be appropriate. However, if the obliquity exceeds 10°, the shift of loading from the lateral compartment will only be as far as the tibial spine and not to the intended medial compartment. Femoral osteotomy is therefore recommended for all degrees of valgus because it avoids obliquity of the joint and it is easier to achieve the required correction than by closing medial tibial wedge osteotomy.

Technique

1. Tibial lateral closing wedge. The femoro-tibial (FT) angle is determined on full-length weight-bearing radiographs. The aim of correction should be to produce 10° of valgus, i.e. to produce a FT angle of 170°. One millimetre of wedge resection gives approximately 1° of correction. A transverse incision is made and the common peroneal nerve is clearly identified. Division of the superior tibiofibular joint or resection of the fibular head allows closure of the osteotomy after removal of the wedge. Fixation may be external using plaster or an external fixator, or internal, using staples or a plate and screws.

2. Tibial barrel vault. An anterior approach to the knee is used. A separate short incision is made laterally to remove a segment of the fibula so that the osteotomy may be closed. Guide pins are inserted to measure the correction. Multiple drill holes are made between the tibial tubercle and the joint line and completed with an osteotome. Angular correction is measured using the guide pins to the degree determined by the pre-operative evaluation. Anterior translation of the tibial tubercle with the distal fragment can be achieved at the same time to reduce the load borne by the patello-femoral joint. Fixation is achieved using the same methods as for wedge osteotomy.

3. Distal femoral wedge osteotomy. The wedge size needed to produce a FT angle of 175–180° is estimated from pre-operative radiographs. A long anterior incision or medial and lateral incisions may be used. A medially based wedge is removed and the osteotomy is stabilized using a laterally applied blade-plate.

Complications

1. Clinical failure. The results of proximal tibial osteotomy deteriorate with time. Good or excellent results are seen in 85% at 5 years and 60% at 10 years. Poor results are more likely if the FT angle is more than 170° (i.e. less than 10° of valgus), in obese patients and in those over the age of 60 years at operation. Similar results are seen with femoral osteotomy.

2. Peroneal nerve palsy. This occurs in up to 7% and is caused by direct injury, proximal fibular osteotomy or transfixion by laterally placed external fixator pins.

3. Deep vein thrombosis. Deep vein thrombosis occurs in 40%. Prophylaxis is recommended.

4. Non-union of the osteotomy. This rare complication is best treated by bone grafting and internal fixation.

5. Avascular necrosis orfracture of the proximal fragment.

6. Vascular injury. This is rare and usually involves the anterior tibial vessels.

Further reading

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Related topics of interest

Arthritis of the knee (p. 39)

Arthroscopy (p. 49)

PARALYSED HAND

The hand is the means by which man manipulates his environment. It is also a potent organ of sensation. The remainder of the upper limb functions as a system of levers whereby the hand may be positioned accurately in space.

Paralysis is a state in which either the ability to move (motor paralysis) or feel (sensory paralysis) is lost. They may occur separately to varying extents, or together.

Motor paralysis may affect a single muscle, a group of muscles or all muscles depending on the cause of the paralysis. It may be spastic or flaccid. Spastic paralysis is caused by lesions of the central nervous system (upper motor neurone lesions) and results in increased tone, pathologically brisk reflexes with clonus, diminished or absent abdominal reflexes and upgoing plantar (Babinski) responses. Flaccid paralysis is caused by disease of the anterior horn cells or peripheral nerves (lower motor neurone lesions).

Psychological, hysterical or emotional paralyses are termed *unfeigned psychic paralyses* and vary between full-blown psychoses and minor hysterical reactions. These differ from *feigned paralyses* in which the patient deliberately sets out to deceive the examiner.

Clinical problems

1. Upper motor neurone lesions. These are divided into extrapyramidal and pyramidal lesions. *Extrapyramidal lesions* are characterized by spontaneous uncontrolled movements which are classified on their pattern of movement into athetosis, ataxia, chorea, hemiballismus, rigidity, tics and tremors. They range from Parkinson's disease to the functional disorders of musicians. *Pyramidal disorders* cause motor paralysis, the commonest causes of which are stroke and cerebral palsy. Other causes are cerebral infection, tumours and multiple sclerosis. Most are seen by neurologists or neurosurgeons, but may occasionally present to the orthopaedic surgeon.

2. Lower motor neurone lesions. These may be divided anatomically into those affecting the spinal cord and roots, the brachial plexus and peripheral nerves. These will be dealt with under separate headings.

3. Spinal cord and root lesions. Those seen most commonly by the orthopaedic surgeon include traumatic lesions of the cervical cord, avulsion injuries of the brachial plexus (preganglionic lesions), neuralgic amyotrophy (Spillane-Parsonage-Turner syndrome), and osteomyelitis, tumours and degenerative disease of the cervical spine affecting the spinal cord. Other important causes are craniovertebral anomalies, syringomyelia, subacute combined degeneration of the cord and motor neurone disease.

4. Brachial plexus lesions. These may be *traumatic* lesions, either from injury at birth or later by traction, penetration, irradiation or compression (including thoracic outlet syndrome), or *neoplastic* either arising in the brachial plexus (benign neurilemmomas and neurofibromas or malignant neurilemmomas or neurosarcomas) or involving it (malignant lumps in the neck).

5. Lesions of peripheral nerves. The commonest lesions of peripheral nerves are those caused by compression (i.e. peripheral nerve entrapment) or trauma. Also of importance are the ischaemic/compressive injury of Volkmann's ischaemic contracture and the varied causes of peripheral neuropathy. Tumours of peripheral nerves and leprosy are rarely seen but present specific problems.

Investigation

1. History and examination. The ability to be able to obtain a thorough medical history is essential. The diagnosis of the underlying condition causing paralysis depends on it. A competent examination of the nervous system and hand merely defmes the extent to which the hand and upper limb are involved.

2. Electromyography is the measurement and recording of action potentials from muscles at rest and when contracted voluntarily.

Normally, muscles exhibit no spontaneous activity at rest but produce characteristic triphasic action potentials when contracted voluntarily. Denervation or myopathy results in the inability of a muscle to contract and the production of involuntary discharges from single muscle fibres (fibrillation). After injury, these changes take 2–3 weeks to appear.

3. Nerve conduction studies. Direct electrical stimulation of a nerve evokes a characteristic response in a normal muscle. Measurements of the *motor nerve conduction velocity* can be calculated from the difference between reaction times to a stimulus applied at two different points. *Sensory nerve conduction velocity* can be recorded between paired sets of electrodes. The larger the nerve and the greater its myelin content, the faster the conduction velocity. The more peripheral the nerve and the older the patient, the slower the conduction velocity.

Management

1. Nerve repair. In almost all cases, acute nerve injury is best treated by primary repair, or, if this is impossible, by delayed repair or grafting. Repair in the first 3 months appears to give better results than that achieved at a later date. Repair

after 6 months gives significantly worse results. It is unclear whether there is a demonstrable difference between epineural and interfascicular suture techniques. With both techniques, magnification and meticulous operating technique are essential. Accurate bundle matching may also be beneficial. Grafts, either from the sural nerve or from the medial cutaneous nerve of forearm, should be used if there is a significant gap once the ends of the nerve have been trimmed back to reveal good healthy bundles. The definition of a significant gap is debateable, but it is clear that the repair should not be under tension, the nerve should not be mobilized excessively and adjacent joints should not be placed in extreme postures to close the gap artificially. Peripheral nerves regenerate at roughly 1 mm per day. An 'advancing' Tinel sign over the regenerating nerve demonstrates that regeneration is occurring but does not indicate the degree of functional recovery which will take place.

2. Nerve decompression. This is considered in the section on peripheral nerve entrapment.

3. Tendon transfer. There are literally hundreds of individual tendon transfers described for reanimating the paralysed hand. Of much greater importance is an understanding of the underlying principles which must be observed. These may be summarized as follows: the motor selected must be strong enough to perform the desired movement: a transferred motor will lose one MRC grade of power after transfer: muscles which have been paralysed are generally unsuitable for transfer: motors may be more effective when combined with selective arthrodesis of joints in the recipient structure: joints which are moved by dynamic tenodeses should be mobile and stable: the motor selected should have sufficient excursion to achieve the desired range of movement: the motor should be of a power equal to or greater than that of its antagonist: the line of pull of the transferred motor should be as straight as possible: the motor should be free to glide in its new situation.

4. Rehabilitation and pain control. The rehabilitation of the patient with a paralysed hand is as important as any surgical procedure undertaken to improve the patient's motor or sensory function. The aims are to prevent problems secondary to nerve degeneration (e.g. contracture), to restore function and to compensate for any residual disability. Daily (or inpatient) physiotherapy and occupational therapy may be needed if the patient is too young or insufficiently responsible to carry out their exercises reliably on their own. No patient will rehabilitate adequately if in pain. The commonest problems in the paralysed upper limb are painful neuroma formation and causalgia. If there are problems with pain control, all modalities of treatment offered by a modern pain clinic may be employed.

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Related topics of interest

Congenital neck anomalies (p. 98)
Degenerative disease of the cervical spine (p. 113)
Neuromuscular disorders (p. 195)
Paralysed lower limb (p. 218)
Peripheral nerve entrapment (p. 225)
Thoracic outlet syndrome and cervical rib (p. 324)

PARALYSED LOWER LIMB

The paralysed lower limb may be spastic or flaccid, depending on the cause of the paralysis. The causes themselves are discussed in the section on neuromuscular disorders. In this section, specific problems related to paralysis of particular muscle groups are addressed, as are the problems of instability, weakness, contracture and deformity.

Clinical problems

1. Hip dislocation may gradually develop in the patient with a flexion/adduction hip contracture secondary to gluteal paralysis. It may either be a true paralytic dislocation with weak muscles, stretched ligaments and a valgus anteverted hip, or a postural dislocation due to pelvic obliquity from scoliosis. It is important to differentiate between them as the former may be amenable to surgery at the hip and the latter is not.

2. Hip adduction/flexion contracture results from contracture of tensor fascia lata (TFL), iliopsoas, sartorius, and rectus femoris.

3. Hip abduction/flexion contracture results from an ilio-tibial band contracture. This may produce a pelvic obliquity or, if bilateral, a hyperlordotic lumbar spine. It is often associated with contractures of gluteus medius and minimus.

4. Knee flexion deformity occurs with paresis of the anterior thigh muscles in the presence of active flexors.

5. Genu valgum may develop if quadriceps weakness is associated with active hamstrings and TFL.

6. Genu recurvatum is produced in one of two ways. First, it may be the result of quadriceps weakness and contracture. The knee is forced into hyperextension when walking, eventually causing the anterior tibial plateau to tilt inferiorly. Second, it may be caused by weakness of the gastrocnemii and hamstrings, which become stretched. This often occurs in conjunction with a calcaneovalgus foot.

7. Achilles tendon contracture occurs due to relative over-activity of the calf in the presence of paralysed extensors. Patients with quadriceps insufficiency may

depend on a tight Achilles tendon to lock the knee in extension: this must be assessed before surgery is undertaken.

8. Plantar fascia contracture may contribute to a cavus deformity.

9. Foot muscle imbalance. See below.

Clinical examination with muscle charting is mandatory. Nerve conduction and EMG studies may help define the pattern of paralysis.

Non-operative treatment

1. Physiotherapy. Specific exercises for the affected limb and good general fitness to compensate for paralysis are equally important. Prone lying and stretching exercises may prevent a hip contracture.

2. Traction. Gradual stretching (Agnes Hunt method) may overcome hip contracture. Knee contractures can be treated with reverse dynamic traction.

3. Bracing. Callipers, straps, plastic orthoses and casts all have a major role in treating the paralysed lower limb either as static supports or as dynamic aids (quadriceps-enhancing splint). Splintage prior to fusion may be appropriate.

Operative treatment

1. Hip. Soft tissue release: open division of fascia lata, iliotibial band and lateral intermuscular septum, or release of hip flexors from pelvis (Soutter's operation). *Muscle transfer:* posterior transfer of iliopsoas through the ilium to the greater trochanter (Sharrard procedure). *Osteotomy:* varus derotation femoral osteotomy with adductor tenotomy for true paralytic hip dislocation. *Pelvic shelf procedure:* indicated with femoral osteotomy if there is difficulty maintaining reduction of the femoral head. *Arthrodesis:* last resort procedure for instability and pain. Contraindicated with associated abdominal muscle paralysis.

2. Knee. Soft tissue release: tendon releases medially (semimembranosus, semitendinosus, gracilis, and sartorius), and laterally (biceps femoris and tensor fascia lata) and posterior capsuloplasty to release flexion deformity. *Osteotomy:* genu valgum of more than 8°, fixed flexion despite soft tissue surgery or genu recurvatum may need the appropriate femoral or tibial osteotomy. *Tendon transfers:* for genu recurvatum due to quadriceps weakness in the presence of adequate hip flexors, gluteus maximus, and hamstrings the biceps femoris can be transferred medially and the semitendinosus laterally into the quadriceps tendon. *Arthrodesis:* can be used following failed surgery or inadequate musculature.

3. Foot and ankle. Between 2 and 12 years, most surgery is carried out to correct muscle imbalance. *Bony operations* are best left until near skeletal maturity (Grice-Green procedure excepted). Fusions will not prevent recurrence of a deformity unless muscle imbalance has been corrected. *Muscle transfers* are carried out for the loss of individual movements.

Paralysis of inversion: isolated *tibialis anterior palsy* causes weakness of dorsiflexion and inversion producing an equinovalgus deformity. Unopposed peroneus longus (PL) and extensor hallucis longus (EHL) produce a claw hallux deformity. This is prevented by anterior transfer of PL to the base of the second metatarsal and a Robert-Jones procedure (transplantation of the EHL to the first metatarsal neck and fusion of the interphalangeal joint of the hallux). *Isolated tibialis anterior and posterior paralysis* with unopposed peroneal activity leads to a fixed everted and flat foot. This is prevented by transfer of peroneus brevis (PB) to the navicular to replace the function of tibialis anterior.

Paralysis of dorsiflexion and inversion: weakness of both tibiales plus the toe extensors with normal activity of the peronei and the triceps surae produce an equinovalgus deformity. Initially this can be controlled by a below-knee iron with a toe-raising spring and inside T-strap. Transfer of PB to the dorsum of the foot (base of second metatarsal) may be necessary. Paralytic drop foot in an older patient may require a Lambrinudi fusion.

Paralysis of dorsiflexion, inversion, and eversion: weakness of the tibiales, peroneals, long toe extensors and flexor hallicus longus (FHL) with active triceps surae produces a severe equinovarus deformity resistant to physiotherapy or splintage. Hemi-transplantation of the lateral half of the triceps and Achilles tendon around the fibula to the cuboid is required.

Paralysis of eversion: weakness of peroneus tertius (PT) and extensor digitorum longus (EDL) is often seen in spina bifida or peroneal muscular atrophy. It responds well to lateral transplantation of tibialis anterior to the third metatarsal and of EHL to the first metatarsal neck.

Paralysis of plantarflexion: weakness of triceps surae in a growing child produces a calcaneus deformity. When secondary plantarflexes are preserved (peronei, tibialis posterior, long toe flexors) initial loss of function may be small. However progressive calcaneo-cavus is inevitable. Transfer of any combination of flexor digitorum longus (FDL), FHL, tibialis anterior, tibialis posterior, or the peroneals has been described.

Arthrodesis: the operations used most commonly are the Lambrinudi drop-foot arthrodesis, Grice-Green extra-articular subtalar arthrodesis, Elmslie's operation (fusion correction for calcaneocavus) and a triple fusion. For further details see Hindfoot arthrodesis and osteotomy, p. 147.

Further reading

Sharrard WJ. *Paediatric Orthopaedics and Fractures,* 3rd edn. Oxford: Blackwell Scientific Publications, 1993.

Related topics of interest

Amputations and prosthetics (p. 15)

Congenital foot deformities (p. 88)
Developmental dysplasia of the hip (p. 119)
Flatfoot (p. 127)
Hindfoot arthrodesis and osteotomy (p. 147)
Neuromuscular disorders (p. 195)
Spina bifida (p. 296)

PATELLAR INSTABILITY

Recurrent dislocation of the patella may follow an isolated injury but is most often the consequence of pre-existing anatomical abnormality.

Instability may be caused by deficiencies in either the dynamic or static stabilizing mechanisms of the patella. The *dynamic* component comprises mainly the vastus medialis obliquus muscle (VMO) which has relatively horizontal fibres and acts as a medial stabilizer. The *static* components include the shape of the patella and the shape of the femoral condyles. The *Q-angle* is the angle formed by the line of pull of the quadriceps muscle and the patellar tendon through the mid-point of the patella. This is a valgus angle and is greater in females than males (10° vs. 15°). The resultant lateral force vector when the knee is actively extended is therefore directed laterally. The angle is increased in genu valgum, persistent femoral anteversion, external tibial torsion or with a laterally positioned tibial tuberosity. A dysplastic patella, patella alta or hypoplastic femoral condyles may contribute to instability.

Clinical problems

1. Traumatic dislocation. This usually follows an isolated injury. If it occurs more than twice then it is thought of as 'recurrent'. Patients are often overweight, with knock knees, and patella alta. The patella usually dislocates during sport or while dancing and reduces spontaneously when the knee is extended. Dislocation is accompanied by a long tear of the medial capsule and patellar expansion which is manifested clinically by exquisite tenderness over the medial border of the patella. There is sometimes an associated osteochondral fracture.

2. Habitual dislocation. The patella dislocates every time the knee is flexed and reduces every time the knee is extended. The cardinal physical sign is that if the patella is held reduced, knee flexion is restricted. Flexion returns once the patella is allowed to dislocate again. Habitual dislocation may be caused by an abnormal attachment of the iliotibial band, a quadriceps contracture or a combination of the two. Continuing dislocation may lead to degenerative arthritis: treatment by surgical division of contractures and plaster immobilization in flexion is recommended.

3. Congenital dislocation (see Congenital knee anomalies, p. 94).

4. Recurrent dislocation or subluxation. The patient usually describes one or more episodes of acute traumatic dislocation with a characteristic description of the knee 'going out'. Subsequently there is a history of diffuse anterior knee pain with intermittent episodes of severe pain. There may be swelling and crepitus. There are two different patterns of malalignment which can be responsible for instability. *Subluxation in extension* occurs when the patella disengages from the trochlea in the last 30° of extension and moves laterally. The risk of dislocation occurs during flexion when the laterally placed patella passes down the outer side of the lateral condyle. This is caused by 'bowstringing' of a malaligned quadriceps mechanism. *Subluxation in flexion* is due to a tight lateral retinaculum. This can lead to excessive lateral pressure syndrome (ELPS) and premature lateral facet degeneration.

Management

1. Clinical Feel for effusion, crepitus and localized medial marginal tenderness. There may be evidence of generalized ligamentous laxity. The 'apprehension test' is positive. There is quadriceps wasting. The Q-angle may be increased.

2. Plain X-rays. The lateral view is helpful in determining patella alta (high-riding patella). The ratio of the length of the patella tendon to the length of the patella is usually equal to one. If less than 0.8, patella alta exists. The 'Merchant view' is a standard axial radiograph with the knee flexed at 45° and the X-ray beam at 30° to the horizontal. This demonstrates the position of the patella in the femoral groove and Merchant has described angles of congruence based on this projection. An osteochondral fracture may be seen on any of these views .

3. CT scan. Better visualization of the patella can be made with CT cuts in the mid-patella plane at 0°, 10°, 20° and 30° of flexion. The normally aligned patella should enter the trochlea at 20° of flexion, with the patella centred and untilted.

4. Non-surgical treatment. Strengthening exercises for the VMO. Avoidance of high resistance arc of motion exercise between 0° and 90° is important. Stretching the lateral retmaculum and the iliotibial band along with patella taping (McConnell) may render surgery unnecessary.

5. Surgical treatment. Before any procedure to correct malalignment is undertaken, *arthroscopy* should be carried out to assess the extent of damage to the articular cartilage and to exclude other intra-articular pathology. An assessment of patellar maltracking can be made visually.

Open or arthroscopic *lateral release* is most effective in those with subluxation in flexion. *Proximal realignment of the extensor mechanism* is achieved by realignment of the quadriceps tendon on the patella in conjunction with a lateral release. *Distal realignment of the extensor mechanism* using bony procedures involving the tibial tuberosity should not be performed until the apophysis has fused. The choice then is between distal and medial transplantation of the tubercle (Hauser technique) and medial transposition of

tibial tubercle (Elmslie-Trillat procedure). Medial transfer of the lateral half of patellar tendon (Roux-Goldthwait operation) can be used in younger patients. *Combined proximal and distal realignment* should be carried out if the Q-angle exceeds 10°. *Patellectomy* is only considered as a last resort.

Further reading

Arnbjornsson A *et al.* The natural history of recurrent dislocation of the patella. *Journal of Bone and Joint Surgery,* 1992; **74B:** 140–2.

Dandy D, Griffiths D. Lateral release for recurrent dislocation of the patella. *Journal of Bone and Joint Surgery,* 1989; **71B:** 121–50.

Goodfellow J, Hungerford JS, Woods C. Patello-femoral joint mechanics and pathology: 2. Chondromalacia patellae. *Journal of Bone and Joint Surgery,* 1976; **58B:** 291.

Related topics of interest

Anterior knee pain (p. 25)
Arthroscopy (p. 49)
Chronic knee instability (p. 81)
Congenital knee anomalies (p. 94)
Knock knees and bow legs (p. 166)

PERIPHERAL NERVE ENTRAPMENT

Peripheral nerve entrapment may result from compression, distraction, angulation or friction. Compression may occur when the space available for the passage of a nerve is insufficient. This usually follows an increase in tissue pressure due to trauma, pregnancy, metabolic or endocrine disturbance, tumour, malformation or vitamin deficiency. A nerve may also be compressed by a hypertrophied or anomalous muscle, scar tissue, tumour or bony deformity. Friction may occur when a nerve lying in its normal anatomical position is repeatedly moved over its surrounding structures by movement of an adjacent joint.

Mild compression of a nerve applied for a relatively short period of time produces a conduction block which is probably ischaemic in origin. If a nerve is chronically compressed it may be more sensitive to ischaemia. Ischaemia then causes episodes of conduction block with numbness and paraesthesia which, if prolonged, will result in infarction and demyelination.

Clinical problems -upper limb

1. Suprascapular nerve. Entrapment of the suprascapular nerve in the suprascapular notch is a rare cause of shoulder pain which is characteristically diffuse, deep and radiates to the posterior and lateral aspects of the shoulder and on to the chest wall. There is local tenderness over the notch. Weakness of supraspinatus and infraspinatus may limit abduction and lateral rotation of the shoulder joint but wasting is a late sign. The pain can be abolished by the injection of local anaesthetic into the notch under fluoroscopic control. The diagnosis should be confirmed by EMGs. *Non-operative treatment* is by rest, splintage, steroid injections and physiotherapy. *Operative treatment* is reserved for resistant cases when division of the suprascapular ligament or notchplasty may be carried out depending on the perceived source of nerve compression.

2. Thoracic outlet syndrome. See Thoracic outlet syndrome and cervical rib, p. 324.

3. Median nerve. The commonest cause of median nerve compression is *carpal tunnel syndrome*. The nerve is compressed as it passes through the fibro-osseous tunnel at the wrist. The condition is commonest in middle-aged women. It

classically causes numbness, tingling and pain in the radial three and a half digits. It is characteristically worse at night, often waking the patient from sleep. It is exacerbated by activities such as carrying a heavy shopping bag. The numbness may cause a decrease in the patient's ability to carry out fme manipulative tasks. As it worsens the pain becomes continuous. There may be wasting of the thenar muscles, sympathetic disturbance, and clumsiness with a positive Tinel sign and Phalen's (wrist flexion) test. The symptoms of carpal tunnel syndrome vary widely in practice, both in distribution and severity. *Sensory nerve conduction studies* are the most sensitive method of making the diagnosis. The degree of slowing of conduction does not correlate well with the clinical severity of the condition. *Non-operative treatment* consists of avoiding the activities which exacerbate symptoms, local steroid injections, NSAIDs, splintage and short-term diuretics. *Operative treatment* is by decompression of the tunnel through an interthenar incision. This tends to the ulnar side of the palm, thereby minimizing the risk of damage to the recurrent motor branch of the median nerve. In inflammatory conditions, a complete flexor tenosynovectomy should be considered. The commonest *complications* are tenderness over the incision, weakness of grip, stiffness of the fmgers and RSD. Haematomas, inadequate decompression and damage to the motor or digital branches of the nerve are well recognized. The superficial palmar arch is at risk if the incision is carried too distal.

There are three areas around the elbow in which the median nerve may be compressed. The first, 5 cm above the elbow, is at the aberrant *ligament of Struthers,* which runs from this point to the medial epicondyle. The second is just below the elbow where the nerve may be compressed by a band running in the pronator teres muscle *(pronator syndrome)* and the third occurs 5–8 cm below the lateral epicondyle where the *anterior interosseous nerve* may be trapped by the fibrous arch of flexor digitorum sublimis, or anomalous bands. The common symptom in each is pain, aching and heaviness after activity. The pain radiates from the site of compression distally. The neurological signs and symptoms may be vague.

4. Ulnar nerve. Ulnar nerve entrapment at the elbow is the second commonest site (after the carpal tunnel) for nerve entrapment in the upper limb. It may occur by compression beneath the aponeurotic arch of flexor carpi ulnaris *(cubital tunnel syndrome),* or may result from an isolated injury to the nerve, compression by bony deformity or scarring or as a result of recurrent subluxation of the nerve around the medial epicondyle.

The patient describes numbness and tingling in the ulnar nerve distribution followed by pain in the elbow and forearm which may disturb sleep. Motor signs include weakness of pinch with a positive Froment's sign; weakness of grip due to paresis of FDP to the ring and little fingers; loss of coordination of digital flexion and extension due to weakness of the interosseous and lumbrical muscles; small muscle wasting; and varying degrees of ulnar claw hand. If surgery is indicated, the choice lies between simple (Osborne) release, medial

epicondylectomy and anterior transposition of the nerve either to a subcutaneous or submuscular position. Transposition is not without morbidity. If not carried out widely, kinking or compression may occur particularly in the region of the medial intermuscular septum: if widely transposed, there is the risk of significant ischaemia.

Ulnar nerve entrapment can also occur more distally where the nerve enters the hand through *Guyon's canal*. This canal is bounded by the transverse carpal ligament, the volar carpal ligament, the hook of the hamate and the pisiform. It also contains the ulnar artery and vein. The most common causes are a ganglion from one of the carpal joints or an occupational injury. The nerve divides into its motor and sensory branches within the canal, and either or both may be affected. The dorsal sensory branch, which arises in the distal forearm, is spared.

5. Radial nerve entrapment is rare. It may occur high following trauma. *Supinator syndrome* may occur from idiopathic entrapment of the posterior interosseous nerve by the arcade of Frohse, but most cases are due to lipomas, cysts, bursae or ganglia, trauma or rheumatoid arthritis. In idiopathic entrapment there is a wrist drop with radial deviation (ECU: ECRL, ECRB and supinator are innervated proximal to the arcade) and absence of extension of the thumb and fmgers at the MCP joints (EDC, EIP, APL, APB and EPL). *Radial tunnel syndrome* (resistant tennis elbow) causes pain over the interosseous nerve which should be distinguished from the lateral epicondylar pain of tennis elbow. The superficial cutaneous branch of the radial nerve is frequently damaged but can occasionally become entrapped.

Lower limb

1. Lateral femoral cutaneous nerve entrapment (meralgia paresthetica) is quite common. It is a purely sensory neuropathy causing burning and numbness over the upper outer aspect of the thigh. There may be a positive Tinel sign over the inguinal ligament where it is pierced by the nerve. Pain is not usually severe. If it is, suspect an L3/4 disc protrusion or rarely, an intraspinal tumour.

2. Sciatic nerve entrapment is rare. In 6% of people, the peroneal portion of the sciatic nerve passes through the substance of piriformis where it may be entrapped *(piriformis syndrome)*. Symptoms are worse after activity, particularly running, and are exacerbated by internal rotation of the femur which compresses the nerve. There is local tenderness over piriformis. Treament is surgical, by division of one of the heads of the muscle. This syndrome is currently overdiagnosed and is not as clear-cut as the previous description would indicate. The sciatic nerve may also be trapped more distally.

3. Peroneal nerve entrapment is rare. Idiopathic entrapment may occur in the fibular tunnel. Most lesions are neuropraxic from acute trauma or compression. A foot drop with inversion results.

4. Tarsal tunnel syndromes. There are two syndromes, both of which are rare. *Posterior tarsal tunnel syndrome* is due to entrapment of the distal part of the

posterior tibial nerve at the level of the medial malleolus. The patient complains of pain in the foot in one or more of the three terminal branches of the posterior tibial nerve. It may wake them from sleep and is worse after activity. There is local tenderness and a positive Tinel sign behind the medial malleolus. *Anterior tarsal tunnel syndrome* is due to entrapment of the distal part of the deep peroneal nerve beneath the dense superficial fascia at the front of the ankle. There is local tenderness and pain at the site of the entrapment and altered sensation in the first web space.

Management

1. Investigation. Electrodiagnostic procedures help to establish the diagnosis of entrapment, to confirm the site or to quantify deterioration or recovery with time.

2. Non-operative treatment relies on avoidance of repetitive movements, splintage, local injection of anaesthetic or steroid, physiotherapy and short courses of diuretics.

3. Operative. The aim of surgical decompression is to relieve the pressure on the nerve by removing the anatomical structure causing compression. If this is due to a specific pathological structure, such as a ganglion in Guyon's canal or an aberrant vessel, no major problem should be encountered. An abnormal thickening of fascia may be divided. It is when the nerve has been released from any surrounding source of compression, but is still subject to repeated traction or distortion, that transposition to a different anatomical site may be needed.

Further reading

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Kopell HP, Thompson WAL. *Peripheral Entrapment Neuropathies.* Baltimore: Williams & Wilkins, 1963.

Related topics of interest

Neuromuscular disorders (p. 195)
Paralysed hand (p. 214)
Paralysed lower limb (p. 218)
Thoracic outlet syndrome and cervical rib (p. 324)

PERTHES' DISEASE

Perthes' disease is an osteochondritis of the hip which occurs as a result of ischaemic necrosis of the upper femoral epiphysis. It occurs most commonly between 4 and 8 years of age (range 2–16 years), is commoner in boys than girls (4:1), and is bilateral in 10% of cases. Occasionally, there is a family history but there is no evidence that it is an inherited disorder. There is a slightly increased risk with breech delivery and in lower socio-economic groups: it is commoner in urban populations. Children with Perthes' disease have a lower mean birth weight and a delayed bone age (average 21 months).

The aetiology is unknown. Up to 3% of children with irritable hips have Perthes' disease but there is no evidence that 'benign transient synovitis' is a causative factor. There is an association with excess femoral anteversion which is currently thought to be due to 'vascular embarrassment' to the proximal femur, possibly due to deficiency of the anterior anastomotic vessel from the circumflex arteries. Studies of other epiphyses in affected patients show that this may be a generalized process involving other sites synchronously.

Clinical presentation

The onset of Perthes' disease is usually insidious. The child may limp but pain is often only present on activity. The presentation is, therefore, usually delayed. Pain is located in the groin, thigh or knee. Some present with acute pain, often with a history of recent trauma. Examination shows a restricted range of motion, particularly of abduction and internal rotation. There may be a true leg length discrepancy.

A similar presentation may occur with septic arthritis, proximal femoral osteomyelitis and juvenile rheumatoid arthritis. In cases of bilateral disease hypothyroidism and multiple epiphyseal dysplasia must be considered.

Investigation

1. Plain radiographs. The diagnosis is made and the progress of the child monitored by plain AP and lateral radiographs. These identify the extent of

epiphyseal involvement (Catterall groups 1–4) and the radiological stage of disease.

The radiological staging is used to follow progress of the disease. At *stage I (initial phase)* there is a small radiodense ossific nucleus, the epiphyseal plate is irregular, the metaphysis appears blurred, there may be a crescent sign (subchondral radiolucent zone) and there may be widening of the medial joint space due to secondary cartilaginous hypertrophy. In *stage II (fragmentation phase)* the bony epiphysis fragments as cystic areas appear where dead bone is being resorbed. In *stage III (reparative phase)* normal bone density reappears and remodelling occurs. New bone is deposited at the medial and lateral edges of the epiphysis causing widening of the head. *Stage IV* is the healed phase in which bone quality is normal but the shape of the femoral head may or may not be normal. Often the femoral head and neck are widened but remain spherical (coxa magna) but, if recovery is poor, the head is ellipsoid and flattened and the acetabulum is reciprocally deformed.

2. Scintigraphy can help in the early stages if the diagnosis is in doubt and can also be used to assess the degree of epiphyseal involvement.

3. Magnetic resonance imaging is sensitive in locating areas of infarction ahead of radiographic changes and may help in predicting healing.

4. Arthrography can be used to assess congruency and 'hinge abduction' when planning treatment.

Prognostic factors

Certain clinical and radiographic factors determine prognosis. These include deformity of the femoral head, joint incongruity, age of onset of disease, growth disturbance due to premature epiphyseal closure, protracted course of disease, type and timing of treatment. Partial involvement or anterior femoral head involvement favours good prognosis.

1. Grading. Catterall has described four grades of Perthes' disease. In *group 1,* only the anterior part of the epiphysis is affected, so that on AP and lateral radiographs less than 50% of the epiphysis is involved. *Group 2* involves more than 50% of the epiphysis with sequestrated fragments limited to the anterior segment. Viewed in the AP plane, a central dense 'head within a head' appearance is characteristic. *Group 3* has additional features of flattening of the head and metaphyseal changes. *Group 4* shows extensive head involvement with femoral head collapse and extrusion of the epiphysis anteriorly, posteriorly and laterally. The Catterall grading predicts prognosis. Groups 1 and 2 left untreated have 90% good results, groups 3 and 4 left untreated have 90% bad results. It is not always easy to distinguish radiologically between groups 2 and 3.

2. 'At risk' signs. Certain additional radiographic 'at risk' signs seen during the active stage of disease are associated with a poor prognosis. These are: Gage's sign (radiolucency in the lateral epiphysis and metaphysis) and calcification lateral to the epiphysis (both potentially reversible with treatment);

metaphyseal radiolucencies; lateral subluxation (indicative of a widened head); horizontal growth plate.

'At risk' signs are reflected clinically by reduced motion and an adduction contracture. Catterall reported no poor results in patients without 'at risk' signs.

Treatment

Since the primary cause of Perthes' disease is not known, treatment is aimed at minimizing the secondary changes. Initially, the child with acute pain is treated on bed rest with traction until symptoms subside.

1. Non-operative. Catterall group 1 patients and children under 4 years need no treatment, but are reviewed clinically and radiologically every 3 months. Treatment of patients in other groups depends on the presence of 'at risk' signs. Treatment aims to contain the femoral head in the acetabulum to minimize deformity while spontaneous healing occurs. In the majority this means splintage in abduction/internal rotation braces (e.g. Newington, Toronto, Scottish Rite).

2. Surgery is reserved for those over 6 years old with group 3 or 4 disease or with at least two 'at risk' signs (or failure of treatment with a brace for whatever reason). Before considering surgery, arthrography is mandatory to confirm joint congruity. Most surgeons advocate a varus derotation femoral osteotomy, although some would chose a Salter innominate osteotomy for containment.

Hips that are already deformed, show signs of healing, or are more than 8 months after onset of symptoms are resistant to treatment. Indications for reconstructive surgery are a grossly deformed femoral head (cheilectomy), lateral subluxation (Chiari osteotomy), capital femoral growth plate arrest (trochanteric advancement).

Further reading

Catterall A. *Legg-Calvé-Perthes' Disease.* Edinburgh: Churchill Livingstone, 1982.

Related topics of interest

Anterior knee pain (p. 25)
Arthritis of the hip (p. 35)
Avascular necrosis of the hip (p. 53)
Developmental dysplasia of the hip (p. 119)
Hip arthroplasty (p. 150)
Slipped upper femoral epiphysis (p. 287)
Young adult hip problems (p. 340)

POSTURAL CORRECTION IN ANKYLOSING SPONDYLITIS

Ankylosing spondylitis (AS) tends to cause fixed flexion deformities of the spine, hips and knees. Severe combined flexion deformities of the hips and spine result in a profoundly flexed posture, with loss of the visual horizon, limitation in capacity to function outdoors, reduction in quality of life and clinical depression. The aim of non-operative treatment is to prevent the progression of deformity and to encourage ankylosis in an acceptable posture. Surgical treatment is reserved for patients with severe deformities who feel that their quality of life is wholly unacceptable.

Clinical problems

1. Kyphosis. A progressive flexion deformity can occur in the lumbar, thoracic or cervical spine. A deformity that is initially flexible may, if untreated or if conservative treatment fails, end up as a rigid kyphosis. There may be an associated rotational deformity or deformity in the coronal plane. In the cervical spine, a 'chin-on-chest' deformity makes eating and swallowing difficult and will significantly frustrate the passage of an endotracheal tube.

2. Ankylosis of the hips. Hip involvement is related to the age of onset of the disease. It is frequently bilateral and occurs most commonly in those who develop AS between the ages of 10 and 15 years: 16% of these will require a total hip replacement (THR) at some stage. Only 1% will require THR if the disease does not develop until the age of 30–40 years. Some hips cause little pain but gradually become ankylosed. THR may be indicated in the absence of pain if hip movement is very restricted or if a severe fixed flexion deformity is present.

3. Spinal fracture, particularly of the neck, may occur after trivial trauma. These tend to be shear fractures because of multi-segmental ankylosis. A previously static or slowly progressing painless fixed deformity increases in magnitude and becomes painful. There may be associated neurological impairment.

Management

The key is to determine the degree of functional disability that the deformity causes the patient. If this is not severe, there is no indication for surgery and the patient should be managed conservatively. If the deformity is severe, the magnitude and primary site of the deformity is identified. If the deformity is principally in the spine then Cobb's method may be used to measure the angular deformity. The most useful method for combined deformities is measurement of the *chin-brow* angle. This is the angle from the vertical of a line drawn from the forehead to the chin.

1. Non-surgical. Physiotherapy is the mainstay of treatment and concentrates on extension exercises of the hips and spine. These are carried out at least twice a day. If ankylosis is anticipated, strenuous efforts should be made to ensure that this occurs in the optimum position for hips, spine and knees. Immobilization for intercurrent disease should be avoided. *Medication,* preferably in the form of slow-release indomethacin, is recommended. *Radiotherapy* has been abandoned because of the risk of leukaemia and aplastic anaemia.

2. Surgical If the principal deformity is in the hips, *total hip replacement* should be undertaken before any spinal surgery is considered. Up to 90 degrees of correction may be achieved. If the primary deformity is in the lumbar or thoracic spine, correction may be achieved by *lumbar osteotomy* at one or more levels or by polysegmental osteotomy and transpedicular screw fixation. Lumbar osteotomy is traditionally carried out using a Smith-Peterson extension osteotomy at the L2/3 or L3/4 levels, with osteoclasis of the anterior longitudinal ligament. Concern has been expressed that this technique risks the major vascular complications listed opposite and there is therefore a trend towards using a posterior wedge osteotomy with its apex at the level of the anterior longitudinal ligament. This is achieved by emptying or compressing the trabecular bone within the vertebral body using a curette passed down each pedicle. The posterior wall of the vertebral body is then punched out or impacted into the vertebral body and the osteotomy closed under direct vision. No large series with long-term results are yet available. *Cervical osteotomy* is reserved for those with a severe primary deformity of the cervical spine. It is carried out in the lower cervical segments (usually C7/T1) where the intraspinal diameter is greatest. We favour fixation with a Hartshill cervical loop and sublaminar wiring. Nevertheless, a halo-body vest or Minerva cast should be applied. This may be discarded after 3 months if tomograms of the osteotomy site show good bone formation. A firm collar should be worn until 6 months have elapsed, to guard against accidental injury.

Complications

1. Loosening of THR. There is no evidence that the rate of loosening of THR in AS differs from that in OA but the information available is scant.

2. Heterotopic bone formation occurs after about 55% of total hip arthroplasties in AS. It is only of clinical importance in 9%. This is no greater than in hip replacement for OA. Motion is limited in only 2%. The risk is increased in patients who undergo a second or revision THR if they have had significant heterotopic bone formation around a previous THR, in patients with an infected THR and in those who have undergone THR for a completely ankylosed hip. Since prophylactic NSAIDs and irradiation both carry risks, these should be reserved for patients who are at risk of developing significant heterotopic bone formation.

3. Complications of spinal osteotomy. The complications of spinal osteotomy were initially high, with a 10% mortality from paralysis, aortic rupture and superior mesenteric artery syndrome. Other specific complications are nerve root injury and a high rate of ileus. These occur in addition to the other complications of spinal fusion. In recent years, the incidence of neurological problems has been reduced by spinal cord monitoring, wake-up testing and, in the US, by performing the procedure under local anaesthetic. The current mortality is around 1% and the risk of paralysis from cervical osteotomy perhaps 3%.

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Related topics of interest

Hip arthroplasty (p. 150)
Spinal fusion (p. 299)
Spondyloarthropathies (p. 313)

PROXIMAL FEMORAL FOCAL DEFICIENCY

The term proximal femoral focal deficiency (PFFD) covers a broad range of defects in which there is a deficient proximal femur and hip instability. The incidence is 1 per 50 000 live births. The child presents at birth with a short limb or limbs with a tendency to lie with the affected leg abducted and externally rotated. Most of those affected have other associated anomalies (e.g. fibular hemimelia, clubfoot, or cardiac, spinal and facial anomalies).

PFFD is classified using Aitken's classification (types I-IV). This classification is based on clinical and radiological appearances recognized by the second year of life.

Type I: the distal two-thirds of the femur is present. The acetabulum and femoral head are normal. The femoral neck is absent on early radiographs. By the end of the second year the head and neck have ossified but there may be a pseudoarthrosis resulting in coxa vara with a markedly shortened limb.

Type II: at birth, the distal half of the femur, the acetabulum and the femoral head are present. There is a pseudoarthrosis between the proximal shaft and the head. Ossification of the femoral capital epiphysis is delayed resulting in severe varus of the femoral neck.

Type III: at birth, appearances are similar to type II. The shaft is shorter and displaced more proximally. The acetabulum is dysplastic. The femoral head is absent. There is a single small ossific centre at the proximal femur. There is a pseudoarthrosis between the neck and the distal shaft.

Type IV: at birth, only the distal half of the femoral shaft is evident. It is abducted and the proximal end articulates with the side of the pelvis. The acetabulum and femoral head are absent. The hip is grossly unstable and the leg extremely short. This is often bilateral and fibular hemimelia is usually present.

Clinical problems

1. Psychological. The initial problem is with the parents of the child who have to come to terms with a disfiguring deformity, particularly when the condition is bilateral.

2. Leg length inequality (see p. 174).

3. Limb malposition. The affected limb is abducted and externally rotated making the fitting of a prosthesis more difficult.

4. Hip instability. When a suitable limb stump is available stability at the pelvis is required for efflcient walking.

Management

Initially the diagnosis must be confirmed. The differential diagnosis includes congenital short femur with coxa vara (developmental dysplasia of the hip), congenital absence of the femur and contralateral hemihypertrophy.

Investigation

1. Radiology. Plain radiographs of the pelvis and lower limbs are needed to confirm the diagnosis. The presence of a pseudoarthrosis is confirmed by arthrography.

2. Special investigations. MRI may be used to define the type of PFFD in the early stages.

Treatment

1. Type I. When a pseudarthrosis persists, treatment is the same for types I and II. If spontaneous union occurs the coxa vara and leg length inequality are treated as required.

2. Types II and III. Non-operative treatment consists of *traction* to correct the length of the soft tissues and reduce the abduction deformity and an *orthosis* with a pelvic strap to control rotation with an ischial bearing calliper to protect the hip. This is complicated by progressive varus deformity at the hip and knee flexion contracture which makes limb-fitting difficult.

Surgical treatment may involve stabilization of pseudoarthrosis and provision of an orthosis, stabilization of pseudoarthrosis and later reconstruction by rotationplasty or radical early surgery.

Stabilization and orthosis. Fusion of the distal shaft to the femoral neck is performed early (before the age of 6 months) to prevent varus deformity. If this provides a stable hip a prosthesis can be fitted. Knee fusion is then performed when growth is complete. The foot can either protrude through the prosthesis, be positioned in equinus within the prosthesis or be removed by Symes amputation.

Stabilization and reconstmction. The initial treatment is as above until early adolescence when reconstruction is performed by rotationplasty (Van Nes procedure). By external rotation osteotomy of the femur and tibia combined with arthrodesis of the knee, the foot is reversed. The ankle operates as a knee to control a ‘below knee prosthesis’. This procedure is only indicated in unilateral cases when the ankle can be situated at the level of the opposite knee. It is best done at approximately 12 years of age when growth is more easily predictable.

Normal ankle and foot movement and good muscle power at the ankle are prerequisites. A custom-build prosthesis and re-education for walking are then required.

Radical early surgery. At 3 years of age, the pseudarthrosis is explored and the knee arthrodesed. The growth plates are preserved. This is followed 2 months later by a Symes amputation. It risks premature fusion of growth plates and is not widely practised.

3. Type IV. Surgery usually has little to offer. The hip is completely unstable and the limb very short. Stability may be obtained by fusion of the distal femoral shaft to the pelvis. This may require a preliminary Chiari osteotomy to create a buttress. Fusion is carried out with the femur flexed at 90° to the pelvis, allowing the patient to sit with the knee fully extended. The stump may be too short for a prosthesis but it allows comfortable sitting.

Complications

- Premature growth plate fusion compounding leg length inequality.
- Neurovascular compromise following complex osteotomies.
- Rotationplasty is a complex procedure which may be followed by 'derotation'. The created 'knee' only flexes to 90° and therefore limits function, especially kneeling. The limb is cosmetically poor without the prosthesis and high suicide rates are reported following this procedure.

Further reading

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Related topics of interest

Clubfoot (p. 85)
Developmental dysplasia of the hip (p. 119)
Leg length inequality (p. 174)

REFLEX SYMPATHETIC DYSTROPHY

Reflex sympathetic dystrophy (RSD), algodystrophy and Sudeck's atrophy all refer to an exaggerated response to a painful stimulus, usually affecting an extremity. The painful stimulus is frequently traumatic but RSD has followed myocardial infarction, cerebrovascular accident and herpes zoster infection. The precise pathological pathway is unknown but appears to involve activation of both central and peripheral nervous systems. It is more common in females (2:1) in the age range of 20–40 years but can affect any age including children. It is more common in the lower limb than the upper and has been described in the spine, hip and shoulder. Some studies have found the incidence to be as high as 37% although the condition is usually recognized much less frequently than this.

Clinical problems

1. Pain. The pain is gradual in onset. It is often described as intense or excruciating and appears to be out of proportion to the injury. Adjectives such as 'lightning', 'burning', 'knife-like' and 'throbbing' may be used by the patient. The pain often has a non-dermatomal distribution and as time progresses becomes more diffuse. Patients describe hyperalgesia (increased sensitivity to noxious stimuli) and allodynia (pain provoked by stimuli not normally recognized as painful). Tenderness is often felt at joints distal to the injury.

2. Swelling. Swelling of the involved extremity is diffuse and may contribute to reduced range of movement. After the first few months the swelling becomes non-pitting.

3. Reduced movement. Movement is restricted by both pain and swelling. Further reduced movement increases the swelling thereby perpetuating the condition. Joint contractures follow prolonged immobility.

4. Skin changes. Vasomotor changes are common. Initially the skin has an increased blood flow with warmth and erythema. After a few months the skin becomes cold, tight and cyanotic because of reduced blood flow. The finger nails become grooved and pitted and the hair growth ceases. Hyperhydrosis is a frequent fmding.

5. Sensory changes. Hyperaesthesia, dysaesthesia and paraesthesia are common early problems.

6. Psychological factors. These patients often exhibit features of depression and anxiety. While some personality traits may make patients more susceptible to the condition, there is little scientific evidence for this and the psychological features exhibited may be part of a consistent response to chronic severe pain.

Management

1. Diagnosis. The clinical features usually herald the diagnosis but recognition is low. The diagnosis is made on the basis of two descriptors of neuropathic pain (burning, dysaesthesia, paraesthesia, allodynia and cold intolerance) and two signs of autonomic dysfunction (cyanosis, skin mottling, hyperhydrosis, oedema and a cool extremity). Radiographs may show localized areas of osteoporosis on a background of more diffuse reduction in bone density. Bone resorption is patchy and may involve any surface of the bone. These radiographic changes are less marked in children. Technetium scintigraphy usually shows an increased blood flow and peri-articular bone uptake. Thermography has been used but mainly for experimental screening studies. Haematological investigations are usually normal.

2. Physiotherapy. An early active exercise regime helps prevent the condition and is the mainstay of treatment. Adequate analgesia must be prescribed for this to be effective. The initial treatment may involve desensitization. Splintage may be necessary to prevent contractures. About 70% report improvement.

3. Transcutaneous nerve stimulation (TENS) and acupuncture. Fifty-seven percent of patients find improvement with TENS. Seventy percent of patients who have electro-acupuncture report improvement.

4. Sympathetic blockade. Regional sympathetic blockade can be carried out by intravenous injection under tourniquet of local anaesthetic and guanethidine which acts as a false transmitter, depleting nerve endings of noradrenaline. Alternatively, the sympathetic chain can be blocked by injecting local anaesthetic into either the stellate ganglion or the lumbar sympathetic chain. A catheter may be left in place to give continuous analgesia for a few days. The blocks allow intensive mobilization of the limb under powerful analgesia. Relief of symptoms may be expected in 76%. If the block is successful it can be repeated and made permanent by either chemical or surgical sympathectomy.

5. Psychological assessment. In most cases review by a clinical psychologist will detect any underlying psychiatric disorder and improve the patients' response to their chronic pain. Treatment with tricyclic antidepressants may improve symptoms in approximately 50% of cases.

6. Other modalities. Non-steroidal anti-inflammatory tablets, systemic corticosteroids, regional intravenous corticosteroids, propranolol and calcitonin may all give symptomatic improvement. There is a strong placebo response in this condition.

Prognosis

Early detection and aggressive treatment will improve the prognosis. However, the diagnosis is frequently overlooked for some time and chronic pain patterns develop. Symptoms are often resistant to treatment and approximately 50% of patients continue to have significant pain and disability. Communication with the patient, reassurance and treatment of anxiety are important aspects of management.

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Related topics of interest

Repetitive strain injury (p. 242)

Wrist pain (p. 337)

REPETITIVE STRAIN INJURY

Repetitive strain injury (RSI), cumulative trauma disorder and overuse syndromes are terms used to describe any condition of the upper limb which produces symptoms of pain associated with repetitive work. Many patients are suffering from recognisable conditions which will respond to appropriate treatment. However, the majority do not fit into any defmite diagnostic category and are labelled as having 'RSI' to suggest that they suffer from a condition which is attributable to repetitive actions in their employment. The paradox is that most patients with RSI have jobs which involve keyboard operation and not heavy repetitive manual work.

Clinical problems

This diagnosis has a number of characteristic features.

- It is a symptom complex which fails to respond to treatment and is followed by more widespread and continuing symptoms. No underlying pathology is found.
- The common aches of everyday usage are magnified so that there are continuing complaints in the absence of recognisable pathology.
- Repetitive physical activities at work are blamed as causative.
- Litigation against the employers for financial gain perpetuates and exacerbates the symptoms.

Medicolegal cases reached epidemic proportions in the late 1970s and early 1980s in Australia. There is no evidence that the number and rate of repetitive tasks on a keyboard correlates with this complaint. Heavy manual workers, who should be most affected, have a low incidence. Improved standards of working conditions and ergonometrics have not brought about a reduction but are associated with an increase in cases. The present increase in cases in the United Kingdom probably represents an increased preconceived disability in the workplace with an opportunity for compensation. There is little evidence of an industrial injury or disease. It is important to distinguish RSI from the following defmite clinical syndromes.

1. Tenosynovitis. This condition is relatively rare and usually follows rapid repetitive movements which are either unaccustomed or resumed after a vacation. Pain and swelling are localized to the tendon sheath, which usually involves the tendons of the first and second dorsal compartments. There is swelling and tenderness over the area of the sheath. Stretching the muscle-tendon unit and activation against resistance reproduce the pain. Crepitus is occasionally felt or heard. The symptoms settle after a few days rest or a change in the work routine.

2. Stenosing tenosynovitis. Fibrosis and stenosis may occur after inflammation of a tendon sheath. De Quervain's stenosing tenovaginitis affects the long abductor and short extensor of the thumb. There is no objective evidence that this condition is caused by rapid repetitive movements although exacerbation of the symptoms by such activities is common. Pain and tenderness is localized to the first dorsal compartment and Finkelstein's test confirms the diagnosis in the absence of osteoarthritis of the first carpometacarpal joint (see p. 46). This condition has also been described with the extensor pollicis longus tendon and that of extensor digiti minimi.

This term is also applied to stenosis of the synovial sheaths of the flexor tendons of the thumb and the other fingers in association with thickening of the tendon leading to a triggering.

3. Tendinitis is similar to tenosynovitis except that the condition affects the tissues around the tendon proximal to the tendon sheaths. Pain, swelling and tenderness are found at the point where the long abductor and short extensor of the thumb cross over the radial extensors of the wrist. There is frequently crepitus and therefore the condition is referred to as peritendinitis crepitans. The clinical course is the same as for tenosynovitis.

4. Flexor carpi radialis tendinitis presents with pain after strenuous exercise, and tenderness is localized to the line of flexor carpi radialis just proximal to the scaphoid tubercle. The condition is really a tenosynovitis rather than a tendinitis.

5. Carpal tunnel syndrome is common in the general population and although an industrial aetiology has been claimed there is little evidence that this syndrome is caused by a particular manual activity.

6. Upper limb enthesopathies. Golfer's and tennis elbow are common in the middle aged group and probably represent a mild degenerative condition of the fibrous connective tissue at the origin of the forearm flexor and extensor muscle. These conditions are likely to be exacerbated by, rather than caused by, repetitive physical work.

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Related topic of interest

Soft tissue disorders of the elbow and forearm (p. 290)

RHEUMATOID ARTHRITIS

Rheumatoid arthritis (RA) is the commonest form of chronic inflammatory joint disease. Typically it is an asymmetrical, erosive, deforming polyarthritis affecting both small and large peripheral joints. The course of the disease is often prolonged with exacerbations and remissions. The incidence is approximately 1%. It is more common in females (3:1) and typically begins in the third to fifth decade. The cause of RA is obscure but there appears to be a multi-factorial genetic predisposition.

Pathology

RA is characterized by both extravascular immune complex disease and disordered cell-mediated immunity producing chronic inflammation, granuloma formation and joint destruction. There is an initial synovitis, with infiltration by T-lymphocytes, plasma cells and macrophages followed by hypertrophy of the synovium and pannus formation. The pannus spreads across the joint surface, destroying the underlying cartilage and subchondral bone. Bone erosions occur earliest at the sites of synovial reflection. The joint capsule and its associated ligaments are progressively weakened by the inflammatory process. Muscles adjacent to affected joints atrophy. The result is progressive destruction of the joint with instability and subluxation. Subcutaneous 'rheumatoid' nodules, with characteristic central fibrinoid necrosis and granulomata, occur over bony prominences in 20% of cases.

Clinical problems

Onset is usually insidious with joint pain, stiffness, and symmetrical swelling of peripheral joints. Typically, small joints of the fmgers and toes are the first to be affected. In 15% of cases, the onset is an acute polyarthritis with severe systemic symptoms. In 10%, often middle-aged men, it may be more insidious with fatigue, malaise and weight loss without joint symptoms. Distinguishing it from malignant disease or chronic infection may be difficult.

The articular manifestations of RA are described in detail in other sections. The extra-articular manifestations of rheumatoid disease may be haematological, lymphatic, ocular, cardiac, pulmonary and neurological.

Diagnosis

The diagnosis of RA is made in patients with clinical features of inflammatory arthritis for at least 6 weeks, with four or more of the following criteria: morning stiffness greater than 1 hour; arthritis of three or more joints; arthritis of hand joints; symmetrical arthritis; rheumatoid nodules; rheumatoid factor; and radiological changes.

1. Serological tests. Rheumatoid factor (IgM) may be detected using the latex or the Rose-Waaler tests. Antinuclear factor is positive in 30%. Markers of active inflammatory disease are anaemia, thrombocytosis, raised C-reactive protein, increased plasma viscosity or increased erythrocyte sedimentation rate. Each of these is used to establish the diagnosis and follow disease activity.

2. Synovial fluid analysis excludes joint infection or crystal arthropathy.

3. Synovial biopsy is non-specific.

4. Plain radiography is used to follow progression of erosive joint disease.

Management

Because the aetiology of RA is unknown, its treatment is empirical and consists of symptom relief, suppression of active disease and maintenance of function to allow integration in society.

1. Splintage. In acute exacerbations, particularly at the knee, wrist and elbow, splintage may help prevent or correct deformity. POP immobilization is helpful for acute painful synovitis.

2. Physiotherapy. Exercise and correction of early fixed flexion can prevent permanent deformity (e.g. quadriceps exercises can prevent posterior subluxation of the knee).

3. Corticosteroid injections. Intra-articular injections are effective in the control of acute synovitis in large joints.

4. First line medication. NSAIDs and simple analgesics are used to relieve pain and stiffness but do not alter the course of the disease.

5. Second line medication (disease-modifying drugs) is used for more aggressive disease (anti-malarials, sulphasalazine, penicillamine and parenteral gold). Steroids are used systemically in severe exacerbations which fail to respond to the first line therapy or in life or sight threatening visceral disease, e.g. pericarditis, polyarteritis or scleritis. Prednisolone is the corticosteroid of choice.

6. Immunosuppressive therapy. These drugs may have dangerous side effects and their indications are limited. These include life-threatening extra-articular rheumatoid manifestations with active joint disease which has failed to respond

to high-dose steroids. Such drugs include azathioprine, cyclophosphamide and methotrexate.

7. Surgical treatment is described in the sections on rheumatoid foot, rheumatoid hand, rheumatoid wrist, shoulder arthritis, elbow arthritis, hip arthritis and knee arthritis.

Complications

Septic arthritis can complicate rheumatoid arthritis, especially in long-standing nodular seropositive disease. In debilitated or immunosuppressed individuals, the normal signs and indices of infection may be absent. S. *aureus* is the usual organism. Rheumatoid disease is the commonest cause of secondary amyloidosis.

Further reading

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Related topics of interest

Arthritis of the ankle (p. 28)
Arthritis of the hip (p. 35)
Arthritis of the knee (p. 39)
Arthritis of the shoulder (p. 42)
Arthritis of the wrist and first carpometacarpal joint (p. 46)
Carpal instability (p. 78)
Chronic knee instability (p. 81)
Hip arthroplasty (p. 150)
Juvenile chronic arthritis (p. 162)
Peripheral nerve entrapment (p. 225)
Rheumatoid hand (p. 251)
Rheumatoid neck (p. 254)
Rheumatoid wrist (p. 257)
Rotator cuff injuries (p. 260)
Shoulder impingement syndromes (p. 275)
Shoulder pain (p. 281)
Total knee arthroplasty (p. 331)

RHEUMATOID FOOT

The foot is involved in nearly 90% of patients with RA. Significant deformity at the hip or knee will result in abnormal loading of the foot and ankle. Surgery of the severely affected foot can be very beneficial. The patient's functional needs and expectations should first be assessed and a careful clinical examination carried out. Weight-bearing radiographs are required to assess the extent and degree of joint involvement. Patients with rheumatoid disease who undergo surgery are at higher risk because of poor peripheral circulation, steroid therapy, etc.

Clinical problems

1. Ankle. The ankle is involved in about 25% of those with RA. It usually presents late with pain and valgus instability and is often less disabling than disease in other joints.

2. Hindfoot valgus and subtalar arthritis. This may start with tenosynovitis of the tibialis posterior tendon and synovitis of the subtalar joint. Pain and tenderness are felt in the sinus tarsi. There may be impingement of the peroneal tendons. Radiographs of the ankle may show that the hindfoot valgus is caused by valgus instability of the ankle.

3. Collapse of the medial longitudinal arch. Rupture or weakening of the tibialis posterior tendon combined with talonavicular synovitis results in collapse of the medial longitudinal arch. The head of the talus subluxes medially and inferiorly from the navicular and may bear weight as part of the medial border of the foot.

4. Pronation of the forefoot. The collapse of the medial longitudinal arch combined with a valgus heel swings the forefoot into pronation. Pressure on the first ray may then contribute to hallux valgus if the first metatarsophalangeal joint is diseased. This is an example of the zig-zag deformity of RA. With sustained pressure the forefoot may adopt a supinated position relative to the mid and hindfoot which will only be detected when these are corrected.

5. Dorsal subluxation of the lesser toes and metatarsalgia. Synovitis of the MTP joints of the lesser toes occurs early in the disease. Weakening of the capsule and ligaments causes dorsal subluxation. The intrinsic muscles then lie

dorsally and can no longer flex the MTP joints and extend the interphalangeal joints. The result is dorsally subluxed MTP joints with hammer toe formation. The metatarsal heads are further exposed by distal migration of the forefoot pad. Painful callosities develop under the prominent metatarsal heads, over the prominent PIP joints and at the nail-pulp junctions.

Management

1. Footwear. If the midfoot has collapsed and the heel is in valgus, a medial arch support can be fitted. If the ankle joint is involved, a calliper with inside T-strap may stabilize the tibio-talar joint. A high toe box can be used to avoid pressure over the PIP joints. A metatarsal dome may relieve metatarsalgia.

2. Conservative. NSAIDs, local injections of steroid and brief periods of immobilization may relieve acute synovitis.

3. Synovectomy. In the early stages of the disease, synovitis of the ankle joint which fails to respond to medical treatment can be treated by synovectomy provided that there are no joint erosions or joint space narrowing. Persistent tenosynovitis of the tibialis posterior tendon can be treated by tenosynovectomy. Synovectomy of the subtalar joint is rarely rewarding.

4. Ankle. Early disease may respond to synovectomy and a calliper to restore stability. Arthrodesis often relieves pain but is associated with a high incidence of non-union (25%). The relief of pain allows patients to maintain their level of activity but may not improve it. Arthroplasty of the ankle does not give good long-term results. It is only indicated in those patients who have pan-talar disease and who would otherwise require a triple arthrodesis. It may also be beneficial in cases of bilateral disease, when one ankle can be fused and the other replaced.

5. Hindfoot. Arthrodesis of the hindfoot in the form of a triple arthrodesis is a reliable and useful operation. The talonavicular deformity must be corrected and the arthrodesis should be done with internal fixation of the talonavicular and calcaneocuboid joints to reduce the incidence of non-union.

6. Forefoot. Hallux valgus can be treated reliably either by arthrodesis or Keller's operation with temporary stabilization. Arthrodesis may stabilize the foot more reliably and prevent recurrence of the deformity. The lateral four toes and metatarsalgia can be treated by a variety of operations. It is probably not important whether the incision is placed on the dorsal or plantar surface of the foot unless the forefoot is not passively correctable, in which case a volar approach is preferred. Aim for adequate bone resection to relax the soft tissues and a smooth arcade of the metatarsal remnants. Fowler described a dorsal approach to excise the metatarsal heads with a plantar skin ellipse to replace the forefoot pad. Kates and Kessel performed the operation through a plantar incision. Clayton combined the operation with removal of the base of the proximal phalanges. Hammer toe deformity is best treated by PIP fusion. At the end of the operation the toes need to be held on the end of the metatarsals by

dressings or by temporary intramedullary wires. Overall results are satisfactory in 80–97% of cases. There is little to choose between the operative techniques.

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Related topics of interest

Arthritis of the ankle (p. 28)
Hindfoot arthrodesis and osteotomy (p. 147)
Lesser toe deformities (p. 178)
Metatarsalgia (p. 187)
Rheumatoid arthritis (p. 245)

RHEUMATOID HAND

Rheumatoid involvement of the hand causes functional problems and is frequently mutilating. Synovitis of the joints causes cartilage loss, subchondral bone erosion, swelling of the joint and reduced motion. Ligaments become stretched resulting in joint instability. Synovial swelling of the flexor tendon sheaths causes local pain, swelling and restriction of movement. The combination of joint, muscle and tendon involvement gives rise to zig-zag deformities.

Clinical problems

1. Metacarpo-phalangeal joint. Synovitis initially causes stretching of the extensor hood with ulnar dislocation of the extensor tendon. Ulnar drift of the fingers may be caused by thumb pressure in pinch grip, ulnar inclination of the metacarpal heads, the action of abductor digiti minimi, ulnar drift of the extensor tendons, ulnar force of the flexor tendons, intrinsic tightness or may be secondary to radial deviation of the wrist. Palmar subluxation may occur with stretching of the collateral ligaments and intrinsic tightness.

2. Intrinsic tightness. Spasm provoked by inflamed MCP joints is the most likely cause. If the MCP joint is held in extension it is not possible to flex the PIP joint fully. On radial deviation, the ulnar intrinsic is usually found to be tighter than the radial. In ulnar deviation, intrinsic tightness contributes to ulnar and volar subluxation of the fingers, weakness of power grip and swan-neck deformity.

3. Swan-neck deformity. This deformity is caused by weakening of the periarticular structures of the PIP joint by synovitis. Abnormal force is applied by tight intrinsic muscles. Some believe that weakness or adherence of the flexor digitorum superficialis causes hyperextension. With increasing hyperextension of the PIP joint the lateral bands of the extensor mechanism become adherent to the capsule dorsally. The zig-zag deformity causes flexion at the MCP andDIPjoints.

4. Boutonniere deformity. Synovial proliferation causes weakening and eventual rupture of the central slip of the extensor tendon. The lateral bands are pulled forward in the finger by the swelling of the flexor sheath to which they are

attached by the transverse retinacular fibres. Compensatory hyperextension occurs at the MCP and the DIP joints.

5. The thumb. The Nalebuff *type I* thumb is the most common. A boutonniere deformity develops because of weakness of the extensor pollicis brevis and ulnar subluxation of the extensor pollicis longus. Flexion of the MCP and extension of the IP joint result. The *type II* thumb is identical to type I but is caused by disease of the trapeziometacarpal (CMC) joint with secondary adduction of the first metacarpal. *Type III* is a swan-neck deformity due to disease of the CMC joint with flexion of the metacarpal. Secondary hyperextension occurs in the MCP joint with flexion of the IP joint. *Type IV* is due to disease of the CMC joint which produces adduction of the metacarpal with collateral ligament instability of the MCP or IPjoints.

6. Flexor synovitis. Swelling in the fingers restricts motion and may cause triggering.

7. Extensor tendon mpture and carpal tunnel syndrome. (see Rheumatoid wrist, p. 257).

Management

1. Assessment. The whole upper limb and the functional requirements of the patient must be assessed. Deformity, range of movement, tendon function, stability and sensation must be evaluated. Loss of precision, key and power grips all influence the usefulness of the hand. Routine radiographs of the wrist and hand, including Brewerton's views of the MCP joints, should be taken to evaluate the extent of joint destruction. Appliances and aids may help the patient so that surgery is not necessary. Surgery should be directed at improving function rather than anatomical correction.

2. Metacarpo-phalangeal joints. In the absence of joint destruction, synovectomy with a crossed intrinsic muscle transfer may restore normal alignment. Silastic joint replacement is indicated if there is joint destruction with pain and loss of function.

3. Proximal interphalangeal joint. In early swan-neck deformity, the PIP joint may be treated by dermodesis or flexor tenodesis and release of intrinsic tightness. Early boutonniere deformity may be treated by synovectomy with reconstruction of the central slip. Advanced cases of either deformity may require an arthrodesis or arthroplasty depending on the demands of the fmger, e.g. arthrodesis of the index finger for pinch grip and arthroplasty or arthrodesis in flexion of the little finger for power grip.

4. Thumb. In early boutonniere deformity, the extensor pollicis longus tendon can be inserted into the base of the proximal phalanx. Advanced cases require stabilization of the MCP joint in neutral flexion with 15° pronation for pinch grip. Adduction of the metacarpal in type III and IV deformities may require release of the adductor pollicis or the first dorsal interosseous. Underlying CMC joint disease may require synovectomy, silastic interposition or silastic

replacement of the trapezium. The MCP joint can then be treated by capsulodesis or tenodesis if the joint surfaces are acceptable, or by arthrodesis if they have been destroyed. In the type IV thumb, the IP joint may require stabilization.

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Related topics of interest

Arthritis of the wrist and first carpometacarpal joint (p. 46)

Rheumatoid wrist (p. 257)

RHEUMATOID NECK

Up to 86% of all patients with RA have radiological signs of involvement of the cervical spine. Twenty-five percent of all those admitted to hospital with RA show signs of atlanto-axial instability.

Deformity and instability occur as the result of rheumatoid involvement of the joints and ligaments. They are seen most often in patients who have had severe nodular disease, a high rheumatoid factor, corticosteroid treatment and longstanding (>10 years) disease. There are three basic patterns of disease: atlanto-occipital, atlanto-axial and subaxial. These may occur singly or in combination.

Clinical problems

1. Atlanto-occipital disease. Disease affecting *one* of the atlanto-occipital joints may cause the syndrome of non-rotational tilt of the head. This presents with occipital pain, tender points in the neck and tilting of the head to the affected side. If *both* joints are involved the head may 'settle' on the cervical spine and the odontoid migrates upwards into the foramen magnum causing atlanto-axial impaction and medullary compression.

2. Atlanto-axial disease. Anterior atlanto-axial instability is the commonest type. It is due to stretching or rupture of the transverse ligament and/or erosive synovitis of the odontoid and lateral facet joints. Rarely the odontoid may fracture. The atlas moves forward in relation to the odontoid compressing the spinal cord by a corresponding amount. More than 3 mm between the arch of the atlas and the front of the odontoid is pathological. MRI may show a mass of inflammatory granulation tissue around the odontoid arising from the adjacent joints and further compressing the spinal cord.

Posterior atlanto-axial instability is rare and is caused by erosive synovitis or fracture of the odontoid process.

3. Subaxial disease. This occurs late and involves several vertebrae in a 'stepladder' pattern of deformity.

4. Instability. May be asymptomatic or cause intractable pain in the neck and head, vertebrobasilar insufficiency or cervical myelopathy.

5. Myelopathy. Once established is usually rapidly progressive. Only slight subluxation is needed in the subaxial spine to produce a myelopathy because of the smaller diameter of the spinal canal. The more complex the combination of deformities the greater the likelihood of a neurological deficit. The chance of developing a myelopathy is greater in the atlanto-axial region if the subluxation measures more than 9 mm.

Management

Radiology. Plain lateral flexion/extension views of the neck should be taken with a doctor in attendance if instability is suspected. Plain films show atlanto-axial and subaxial instability but may be insufficiently clear to demonstrate atlanto-occipital instability because of osteoporosis and erosion of the odontoid. They should be available routinely before any surgical procedure under general anaesthetic is undertaken to avoid inappropriate cervical manipulation by the anaesthetist. Sixty percent of patients undergoing hip or knee replacement have radiological evidence of instability: half are asymptomatic. Atlanto-axial impaction can be measured using McGregor's line which is drawn from the posterior tip of the hard palate to the most inferior part of the curve of the base of the occiput. The tip of the odontoid should protrude no more than 4.5 mm above this line.

Magnetic resonance imaging (MRI) should be undertaken in all cases in which significant instability is suspected. Views obtained in flexion and extension will yield valuable additional information.

Treatment

1. Conservative. In most patients having minor degrees of instability. Soft collar and appropriate analgesia. Regular review to exclude neurological deterioration and/or instability. Indications for surgery are intractable pain, neurological deficit and myelopathy.

2. Surgical. Halo stabilization and arthrodesis. Problems arise due to the poor quality of the patient's bone and because rheumatoid patients are intolerant of halo-body vests. Posterior spinal fusion is preferred. This may be augmented with a combination of wire, rods or a contoured loop and PMMA cement. If there is significant anterior cord compression by pannus or migration of the odontoid peg, transoral decompression may be combined with posterior stabilization. Fusion usually takes at least 3 months to occur.

Complications

1. Pseudarthrosis. Rate increases with length of fusion. Occiput to C3 has 36% recurrence after a mean 2.6 years. An isolated atlanto-axial fusion has only a 5.

5% recurrence rate of deformity at a mean 9 years and protects against atlanto-axial impaction.

2. Death. Mortality rates between 27 and 42% have been reported after surgery. They may be higher in those with more advanced neurological deficit.

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Related topics of interest

Rheumatoid arthritis (p. 245)
Spinal fusion (p. 299)

RHEUMATOID WRIST

The wrist is the presenting joint in 2% of patients with rheumatoid disease but is ultimately involved in 90%. A pain-free stable wrist is important for optimal hand function. Rheumatoid disease of the wrist presents with pain, swelling and deformity. Secondary effects on the median nerve, extensor and flexor tendons may necessitate surgical intervention.

Clinical problems

1. Pan-carpal disease. Synovial proliferation involves the whole wrist joint, causing pain, stiffness and swelling. Later, there is destruction of the articular cartilage giving rise to a stable ball-and-socket type of joint which often progresses to a stable ankylosis.

2. Periscaphoid disease. Periscaphoid disease leads to scaphoid rotation and carpal instability. The carpus drifts into volar subluxation and carpal translocation (rotation of the scaphoid with a DISI pattern and ulnar synovitis) occurs. This significantly weakens grip strength.

3. Caput ulnae syndrome. Synovitis destroys the triangular fibrocartilage complex, thereby contributing to ulnar translation of the carpus with impingement of the ulna on the lunate. Prominence of the ulna, caused by supination of the carpus, gives rise to the piano-key sign. Depressing the ulnar head produces pain and radial rotation of the carpus.

4. Instability. The intercarpal ligaments and the wrist capsule become stretched and weakened. Power grip is diminished and lifting is difficult because the wrist is no longer stable.

5. Extensor tenosynovitis. Synovial proliferation of the extensor tendon sheaths occurs commonly and may cause attrition rupture of one or more extensor tendons to the fingers. This usually develops around the prominence of the elevated ulnar head (little and ring fingers) and at Lister's tubercle (extensor pollicis longus). Ulnar extensor tendon rupture may be confused with ulnar subluxation of the tendons in the hand, volar subluxation of the MCP joints or a posterior interosseous nerve palsy by synovial compression from the humero-radial joint.

6. Flexor tenosynovitis. This causes pain and reduced movement of the fingers. Flexor tendon rupture usually occurs over the scapho-trapezial joint and affects the tendon of flexor pollicis longus or around the hook of the hamate, affecting the flexors of the little fmger.

7. Carpal tunnel syndrome. This may occur from inflammatory synovial hypertrophy in the carpal canal or, rarely, by direct infiltration of the median nerve.

Management

1. Assessment. The function of the whole upper limb must be assessed. The goal of treatment is a pain-free stable wrist which acts as a platform for useful hand function. Adequate radiographs are essential.

2. Conservative treatment. The advice of a rheumatologist should be sought in the early stages of the disease regarding systemic therapy, intra-articular steroid injections and splintage.

3. The wrist joint. In the absence of articular damage, early synovectomy is recommended. Synovectomy is frequently performed too late. Conclusive proof that it slows progression of the disease is lacking. Wrist arthrodesis is simple and effective if the articular surfaces are damaged and the disease is unilateral. In bilateral disease, an attempt should be made to preserve movement in one wrist. Silastic interposition arthroplasty may be effective in eliminating pain if the joint is not subluxated. If the wrist is unstable but passively correctable, dorsal reefing of the capsule and temporary stabilization of the joint with a Kirschner wire may restore stability and allow movement. In the presence of fixed subluxation, silastic replacement arthroplasty may restore reasonable function. If the wrist extensors have been destroyed, wrist fusion, using the Mannerfelt technique, is the only option.

4. Distal radio-ulnar joint. Carpal translation (sliding of the carpus in an ulnar direction) should be treated by Darrach's procedure. This can be combined with a radio-lunate fusion to prevent further translation of the carpus. Translocation of the carpus is best treated by the Soave-Kapandji procedure.

5. Tenosynovectomy. Inflamed synovial tissue around the extensor tendons which is unresponsive to conservative measures should be excised to prevent tendon rupture. At operation, the extensor retinaculum is passed beneath the tendons to avoid secondary involvement by the synovium of the wrist joint. Flexor tenosynovectomy is usually combined with carpal tunnel decompression.

6. Tendon transfer. Extensor indicis may be transferred to restore extensor pollicis longus. Rupture of the extensor tendons to the ring and little fingers can be treated by suture to the tendon of the adjacent middle finger or extensor digiti minimi if still intact. In cases where three or four extensor tendons are ruptured, flexor digitorum superficialis can be transferred. Flexor pollicis longus rupture can be treated by transfer of flexor digitorum superficialis from the ring finger, tenodesis of the flexor tendon across the IP joint or arthrodesis of the IPjoint.

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Related topics of interest

Arthritis of the wrist and first carpometacarpal joint (p. 46)

Carpal instability (p. 78)

Rheumatoid hand (p. 251)

ROTATOR CUFF INJURIES

The rotator cuff consists of the tendons of supraspinatus, infraspinatus, teres minor and subscapularis blended with the fibrous capsule of the shoulder to form a hood over the glenohumeral joint. The actions of these muscles combine during elevation of the arm to draw the humeral head inwards and downwards thereby stabilizing it in the glenoid fossa. Impairment of this mechanism is indicated by loss of normal scapulothoracic rhythm.

The rotator cuff mechanism is susceptible to degenerative disease, trauma and inflammation which, singly or in combination, may cause it to rupture. The most vulnerable site is a point 1–2 cm medial to the insertion of supraspinatus on the anatomical neck of the humerus, where there is a area of precarious blood supply.

Rotator cuff rupture occurs more commonly in men (10:1) and in the dominant shoulder. Rupture can be complete or partial. A complete rupture extends through the full thickness of the tendon allowing direct communication with the subacromial bursa and the shoulder joint. A partial tear will cause local inflammation and a subsequent 'painful arc syndrome'. The size of the tear is also important: the uncommon complete massive rupture will cause permanent serious handicap unless surgically repaired. The order of frequency in which they present is: (1) supraspinatus rupture alone, (2) supraspinatus and infraspinatus, (3) a cleavage tear between supraspinatus and subscapularis, and (4) the rare complete avulsion of the supraspinatus/infraspinatus/teres minor complex.

Clinical problems

There are four separate clinical presentations. The chronic attritional type is by far the most common. Acute ruptures account for only 8% of all rotator cuff tears.

1. Acute massive rupture with fracture of the greater tuberosity. Acute anterior dislocation can cause a significant rotator cuff tear and cause permanent disability. Diagnosis is easier if there is an associated greater tuberosity avulsion. Often reduction of the fracture is achieved with reduction of the dislocation. This is considered significant if displaced more than 5 mm or angulated more than 45°. Regular radiographic follow-up is needed initially to ensure position is maintained.

2. Acute massive rupture without fracture is more difficult to diagnose. The patient is usually a man of over 50 who has fallen heavily on to his arm. There is loss of active abduction and acutely there is tenderness in the subacromial region. Local anaesthetic injected into the subacromial area will allow effective examination. The severity of injury is often not appreciated because of pain. If the diagnosis is delayed, the tendon retracts and effective treatment is made more difficult.

3. Acute-on-chronic massive rupture occurs in elderly patients after a trivial injury. Attrition of the avascular insertion over a long period of time results in a massive rupture and significant disability.

4. Chronic rupture presents as a subacromial painful arc syndrome (see below). A chronic tear of the rotator cuff should be considered when a patient with a subacromial arc syndrome is not responding to treatment.

Management

1. Clinical examination using a local anaesthetic injection will often give the diagnosis. When the patient elevates the arm there is a characteristic reversal of the scapulo-humeral rhythm seen as a hunching of the shoulder at the start of abduction. There is an inability to hold the arm in mid-abduction, but there is retention of a good range of passive movement. There is selective atrophy of infraspinatus and supraspinatus within 2 weeks of injury. There may be crepitus and a palpable defect over the humeral head.

2. Plain radiographs following acute injury and a suspected rotator cuff injury are mandatory. This will show a fracture or indirect signs of chronic cuff pathology (calcific deposits, arthritic changes, cystic change in humeral head, decreased acromio-humeral space, etc.).

3. Arthrography will confirm the diagnosis and give additional information if surgery is planned (e.g. loose bodies, labral tear, impingement, etc.).

4. Arthroscopy will demonstrate in addition the precise site and extent of a tear.

5. Ultrasound and MRl examination are useful imaging techniques but their roles are still being defmed.

Treatment

Ninety percent of patients with rotator cuff tears recover without surgery. The majority are older patients with tears in degenerate tendons in whom surgery is inappropriate.

Non-operative treatment is directed towards the relief of pain, stiffness, muscle spasm, and muscle atrophy. Physiotherapy, non-steroidal anti-inflammatory drugs, subacromial injections of local anaesthetic and steroid can all be used to good effect. The acute episode usually settles within 2–3 months after appropriate treatment.

Operative treatment is indicated early for acute injuries in active individuals. In those who are less active, surgery may be considered if there is insufficient improvement after 1–2 months of conservative treatment. In elderly or inactive patients, surgery is reserved for intractable pain.

The specific operative technique used for repair of the cuff depends on the site and extent of the defect. The available methods include direct repair, local transposition or advancement flaps. Fractures are reduced and fixed appropriately. Additional procedures such as anterior stabilization or acromioplasty may be indicated.

Complications

Rotator cuff tears are often a complication of other conditions (e.g. rheumatoid arthritis, osteoarthritis, calcific tendonitis) when cause and effect are difficult to identify. There is evidence of a 'cuff tear arthropathy' which develops with time in unrepaired lesions. This consists of atrophy of the glenohumeral articular cartilage, humeral head osteoporosis and subacromial impingement, which is difficult to treat.

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Related topics of interest

Arthritis of the shoulder (p. 42)
Shoulder impingement syndromes (p. 275)
Shoulder instability (p. 278)
Shoulder pain (p. 281)

SCOLIOSIS—EARLY ONSET

Early onset scoliosis may be congenital, with bony anomalies, or infantile idiopathic in type. The behaviour of these two basic types is entirely different.

Congenital

In the spine, congenital failure of segmentation may be apparent either as the complete absence of a disc, causing a block vertebra, or partial absence, producing a unilateral bar. The classification of congenital spinal deformity is therefore based on the underlying vertebral anomaly (e.g. semi-segmented hemi-vertebra, non-segmented hemi-vertebra, full-segmented hemi-vertebra, unilateral bar, etc.).

Clinical problems

Curve progression. It is important to be able to predict which of these anomalies will cause curve progression. Progression depends on the *type* and *extent* of the anomaly and its effect on vertebral growth. Unilateral failure of segmentation carries a poor prognosis: if this occurs at several levels the prognosis is even worse. The worst combination is a unilateral bar on one side and hemi-vertebra on the other. By contrast, balanced anomalies protect against progression.

The commonest anomaly is a unilateral failure of segmentation. Next comes hemi-vertebra, a bar and contralateral hemi-vertebra, a block vertebra and, least common of all, a wedge vertebra.

Girls are affected more than boys and appear to have a greater potential for progression. Progression is most obvious in the lower thoracic and thoraco-lumbar spine and least obvious in the upper thoracic region. Deformities which present soon after birth have the worst prognosis. All deformities deteriorate during the adolescent growth spurt. An observation phase is important in all cases. If the deformity is acceptable, the aim should be to prevent it from worsening. If the deformity is unacceptable, then correction may be attempted. Significant correction may be obtained without significant neurological risk in patients with idiopathic curves, whereas with most congenital deformities, where

curves tend to be rigid and conditions such as dysraphism coexist, the risk of neurological damage is high.

Management

1. Non-surgical. While a deformity is still acceptable, casting and bracing may be used. There is no evidence that this affects the natural history of the curve. In younger patients it may buy time to allow some spinal growth.

2. Surgical. Curves with significant progression potential may be fused *in situ* if the deformity is acceptable. Anterior surgery should precede posterior fusion, since posterior surgery alone may make the deformity worse.

If the deformity is unacceptable, spinal fusion is combined with corrective surgery. This surgery runs the highest risk of causing paraplegia due to the presence of associated cord anomalies and should therefore be preceded by an MRI scan or myelography.

Correction may be obtained with a one- or two-stage procedure. If a one-stage posterior fusion is undertaken, it may be followed by cast correction for 6 months or may be preceded and/or followed by halo traction. This will not achieve as much correction as a two-stage procedure with preliminary anterior excision of the intervertebral discs and end-plates with bone grafting of the defects. Instrumentation should be avoided if possible.

Unsegmented bars may be assessed by tomography, CT scanning or MRI. Conventionally, they are treated by localized grafting of the convex side of the curve to arrest growth, but the bar may be successfully excised, particularly if it only involves posterior structures. Hemi-vertebrectomy should be reserved for severe cases when there is no other surgical option.

Because the anomalies are congenital it is frequently necessary to operate at a younger age than in cases of idiopathic scoliosis. Whenever possible short segments are fused to minimize growth deformity. Only a 30% correction can be anticipated.

Complications

- Pseudarthrosis. 5–20% incidence.
- Curve progression.
- Loss of correction.
- Complications of halo traction: pin track infection, osteomyelitis, vault penetration, CSF leakage, failure of fixation, cranial nerve palsies and paraplegia.
- Paraplegia.
- Complications of any thoracic or abdominal procedure.

Early onset idiopathic scoliosis (infantile)

Clinical problems

This condition affects children from birth to 3 years of age. There are two clear subgroups: in 95%, the deformity resolves spontaneously; in the other 5% there is a high potential for progression. The condition may be a result of intra-uterine moulding. Other signs of the 'moulded baby syndrome', such as plagiocephaly and plagiopelvy on the same side as the convexity of the curve, may be present. It has been noted that patients with progressive curves exhibit retardation of growth or CNS development and have other congenital abnormalities. Males are more commonly affected than females (3:2) and three-quarters of the curves are convex to the left. Curve pattern is important: thoracic and thoraco-lumbar curves tend to resolve, whereas double structural curves tend to progress.

Management

1. Radiographic assessment. Congenital bony anomalies should be excluded. The rib-vertebral angle difference (RVAD) at the apex of the curve is a good predictor of progression. It is estimated by comparing the angle between the neck of the rib and the vertical axis of the vertebra to which it is attached on each side. The difference is related to the magnitude of vertebral rotation. If the difference is greater than 20°, the curve is likely to progress. The RVAD should be measured every 3 months. A curve which is progressing requires immediate treatment. A Cobb angle of more than 30° is also significant.

2. Non-operative treatment. The use of serial elongation-derotation-flexion (EDF) casts can influence the outcome of infantile curves. The cast is applied under general anaesthetic and is changed every 3 months. When the child is old enough, the cast can be discarded in favour of a brace. The treatment must be continued until the adolescent growth spurt has finished.

3. Operative treatment. Surgery is only considered for a rapidly progressive deformity which is resistant to conservative treatment. Fusion should be avoided wherever possible.

Anterior surgery to remove growth plates and discs may be combined with posterior instrumentation without fusion. This form of 'physiological' correction may achieve good correction without stunting growth. Pre-operative traction may be of greater benefit in this group than in the late-onset group.

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Related topics of interest

Kyphosis (p. 170)
Scoliosis—late onset (p. 267)

SCOLIOSIS—LATE ONSET

Late-onset idiopathic scoliosis presents after the age of 5 years and before skeletal maturity. Some degree of scoliosis is present in 15% of the general population. Curves greater than 10° occur in 2% of the population under 16 years. Clinically significant scoliosis is predominantly a female condition (10:1). Right-sided thoracic curves are the most common.

The clinical problem is principally one of disfigurement but severe curves (>60°) will progress, causing respiratory problems, arthritis with back pain and an overall reduction in lifespan.

Clinical problems

1. Spinal curvature. The location of the apex of the primary curve and the direction of rotation are evident at presentation: it is the magnitude of the curve which increases with growth. There are six main curve patterns.

- *Single major lumbar* (20%). The apex is at Ll or L2 with an average angle at maturity of 37°.
- *Single major thoracolumbar* (15%). The apex is at Tl 1 or T12 and produces a cosmetically significant deformity.
- *Combined thoracic and lumbar double major* (37%). These are balanced curves and are of cosmetically good appearance.
- *Single major thoracic* (22%). These tend to be of earlier onset. The apex is at T8 or T9 causing a marked rib deformity.
- *Cervicothoracic.* A very rare curve with the apex at T2 or T3.
- *Double major thoracic.*

2. Curve progression. Curves tend to progress steadily except during growth spurts, when they increase rapidly. Progression is more likely in girls than boys. The greater the curve and the higher it is in the spine at presentation, the more likely it is to progress. Double curves are more likely to progress than single curves.

3. Other complaints. Children may present sporadically with complaints other than spinal curvature, e.g. breast asymmetry, or pelvic obliquity which produces complaints of a 'dislocated hip' or leg length inequality.

Physical examination

1. General physical examination. This includes the assessment of leg lengths, tilt, list, asymmetrical skin creases and pelvic obliquity. Examination of the hips is mandatory. Height and weight are recorded on growth charts marked in centiles.

2. Neurological examination. Detailed examination must exclude neurological, neuromuscular and neoplastic disorders.

3. Back examination. Visual examination will define the magnitude and direction of the curve and the degree of rotation as demonstrated by scapular prominence over a rib hump, or unilateral fullness in the lumbar region. Any cutaneous signs of spinal dysraphism should be noted. Spinal movements and flexibility are assessed and documented.

4. Radiographic examination. Standing postero-anterior (PA) radiographs should include the whole spine and both iliac crests. Derotated (Stagnara) views may be obtained to allow more accurate angle measurements. Lateral flexion views will confirm clinical estimates of flexibility. Radiographs should not be taken at less than 6-month intervals.

5. Measurement of body-surface topography may be undertaken using non-invasive techniques such as Moire fringe surface topography or ISIS television scanning.

6. Measurement of the curve. The method described by Cobb is widely used. This is the angle formed by perpendiculars to the end plates of the superior and inferior end vertebrae of the major curve on the PA radiograph.

Management

The two most important factors to consider when planning treatment are the progression potential of the curve and the magnitude of the curve. The skeletal growth potential is related to the age at presentation, the time relation to the menarche and to skeletal maturity as determined by Risser's sign. The progression potential of adolescent idiopathic scoliosis is mild compared to many other types. If the magnitude of the curve at presentation is small (between 20 and 30°), the patient will do well with non-operative treatment. As the curve increases (up to 45°), the proportion of successful results diminishes.

Non-operative treatment

Non-operative treatment rarely improves the curve. If a deformity is acceptable at presentation, the aim is to prevent progression and maintain the acceptable

appearance of the curve. If a curve is already unacceptable at presentation, cosmetic improvement will only be achieved surgically.

1. Bracing. The Milwaukee brace (cervico-thoracic-lumbar-sacral orthosis) works by obliterating the lumbar lordosis and preventing forward flexion of the spine. Underarm orthoses (e.g. Boston brace) may be used for curves in the lower thoracic and lumbar spine. These work in the same way and are more acceptable than the Milwaukee brace. Braces are worn for 23 hours a day. The duration of treatment is debatable but it should certainly continue until general skeletal maturity and probably a year longer. Note, however, that spinal skeletal maturity is not achieved until the age of 25.

2. Spinal exercises. Stiffness of the spine should be avoided by exercise. Flexion exercises are probably inadvisable but pelvic tilt and lateral flexion exercises should be encouraged.

3. Cast bracing. This is used widely for infantile curves but less commonly for adolescent curves. An EDF cast is applied with the patient suspended in traction on a Cotrel frame. During growth spurts, regular cast changes are necessary.

4. Electrospinal stimulation. This relies on percutaneous and transcutaneous stimulation of paraspinal muscles. It developed from the belief that there is a neuromuscular basis to late-onset idiopathic scoliosis. No positive benefit has been identified.

Operative treatment

Surgery may be indicated if the magnitude of a curve is unacceptable or if its progression potential is high. Threshold values for considering surgery are around 45° in the thoracic region and 60° in the lumbar region. These are very dependent on individual acceptability. Thresholds of acceptability are becoming lower: they also change with age.

1. Posterior fusion with instrumentation. Segmental fusion with instrumentation (e.g. Harrington, Luque, Cotrel-Dubousset (CD)) has been the mainstay of surgical treatment for flexible curves of moderate size. Correction of rotational deformity has improved by the addition of sublaminar segmental wiring, transverse couplings and CD instrumentation. Fusion with autograft or allograft is used to stabilize the correction. A postoperative supporting cast is needed after Harrington instrumentation but is not essential after Luque or CD procedures.

2. Posterior segmental spinal instrumentation without fusion may be considered in young children whose curves are progressing despite brace treatment. Clearly, extensive fusion of the immature spine with stunting of longitudinal growth is undesirable. Subcutaneous instrumentation attempts to avoid periosteal stripping and premature fusion. The rods are replaced or lengthened every 6–12 months. Bracing is essential to avoid hook displacement.

3. Anterior transthoracic soft tissue release and apophysectomy. This allows greater correction of the curve by releasing anterior tension without compromising the spinal cord. It is followed by posterior fusion.

4. Anterior spinal instrumentation. Screws inserted into the centre of vertebral bodies and joined by rods or cables (e.g. Dwyer, Zielke and Webb-Morley) are effective at correcting short thoracolumbar and lumbar curves with lordoscoliosis.

5. Costoplasty. A persistent prominent rib hump may be reduced by excising segments of the rib neck. A moulded cast is applied afterwards.

Curves over 60° tend to be rigid. Skeletal traction (e.g. halo-femoral, halo-pelvic or halowheelchair) is sometimes used either as a preliminary measure or between anterior release and posterior stabilization in order to obtain a gentle correction.

Anterior soft tissue release alone may not achieve an acceptable correction for curves over 90°. In these cases, a closing wedge osteotomy of the apical vertebrae may be needed. Posterior stabilization is undertaken after an interval of 2–3 weeks, during which time the patient should be treated as for an unstable spinal fracture.

Complications

1. Neurological complications involving the spinal cord occur in approximately 1% of patients. These are minimized by performing an intra-operative wake-up test or using spinal cord monitoring.

2. Chest infection and pneumothorax are the commonest complications following anterior thoracotomy.

3. Urinary tract infection is particularly associated with indwelling catheters.

4. Pseudarthrosis should not occur in more than 1–2% of cases.

5. Rod breakage and hook displacement. Rod breakage usually follows pseudarthrosis whereas hook displacement is the result of technical error or inadequate bracing.

6. Others. The potential for complications in scoliosis surgery is almost limitless.

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Related topics of interest

Kyphosis (p. 170)
Scoliosis—early onset (p. 263)

SEPTIC ARTHRITIS

Joint infection is not common. It occurs by haematogenous spread, penetrating injury or by spread from neighbouring osteomyelitis, either because of the intracapsular location of the metaphysis in the hip or by transphyseal spread in the infant. A delay in diagnosis is frequent and may lead to severe joint destruction, especially in the infant. Approximately one-third occur under the age of 2 years and half by the age of 3. More than one joint is involved in 10% of cases. The sex incidence is equal. Immunocompromised and elderly patients are increasingly affected.

Intracapsular inflammation, synovial proliferation and exudate or transudate raise the intra-articular pressure. Proteolytic enzymes, fibrin adhesions causing loculation of pus and inadequate production of hyaluronic acid have been implicated in the destruction of articular cartilage. Subluxation and dislocation may ensue as periarticular ligaments are stretched and weakened.

Staphylococcus aureus is the most frequently implicated organism except in children aged between 6 months and 2 years when *Haemophilus influenzae* is more common. *Streptococci* and coliforms account for most of the rest. *Tuberculous arthritis* is occasionally seen. Gonococcal and syphilitic joint infection are now relatively rare in the Western world.

Clinical problems

1. The infant usually presents with septicaemia, failure to feed, irritability, fever or anaemia. A delay in diagnosis of a septic arthritis of the hip is common. Local signs include swelling of the leg, buttock or genitalia, lack of movement, asymmetrical skin creases, abnormal posture, and pain on palpation or movement of the hip.

2. The child presents with acute fulminating disease. Fever, tachycardia, effusion, muscle spasm and reluctance to move the joint are common. There may be evidence of another infective focus.

3. The adult frequently presents with an overlying wound or distal portal of entry but this may be absent in the immunocompromised patient. latrogenic causes are not infrequent. Infection may follow aspiration of haemarthroses, arthrograms and femoral venepuncture. Previous arthritis (especially

rheumatoid) is a predisposing factor. The classical signs of fever, erythema, effusion and severely restricted range of movement may be masked in the elderly or those on steroid therapy.

Management

1. Diagnosis. A raised white cell count is seen in 34% and a raised sedimentation rate in 53%: blood cultures are positive in only 41%. Distension of the joint capsule, increased joint space width or subluxation may be visible on plain radiographs. In the young child, erosion of the epiphysis may be seen if the diagnosis is delayed.

Aspiration of the joint yields a positive culture in 80%. Any fluid aspirated should undergo an immediate Gram stain and microscopy. Infection should be suspected if there are more than 50000 cells/ml joint fluid with more than 90% polymorphonuclear leukocytes. If pus is aspirated then the joint should be drained. If aspiration reveals no fluid then radiopaque dye should be injected to confirm correct placement of the needle. The differential diagnosis includes transient synovitis, rheumatic fever, acute osteomyelitis, Perthes' disease, haemophilia, rheumatoid arthritis, gout and pseudogout.

2. Drainage of the joint. The safest procedure is arthrotomy, especially in infants. After debridement and washout the capsule is left open to drain: the skin is closed without suction drainage. Poor results are obtained if surgery is delayed for more than 5 days. Repeated aspiration of subcutaneous joints has been recommended as a safe procedure in children over 1 year of age and in adults. If the response is not favourable in 24–48 hours then arthrotomy is indicated. This regime may avoid unnecessary arthrotomy. Arthroscopic washout of the knee joint in adults is popular but may have a higher recurrence rate. Continuous lavage systems are unnecessary.

3. Antibiotics. The 'best guess' policy is usually accurate in 95%, especially after the initial Gram stain. Most authors recommend cloxacillin and ampicillin until sensitivities are known. There are increasing reports of resistance of *H. influenzae* to ampicillin: chloramphenicol is a suitable alternative. Antibiotics must be given in high dose by the intravenous route. Instillation of antibiotic into the joint is unnecessary. The antibiotics may be given orally as soon as the general and local signs suggest that the infection is settling. Monitoring the sedimentation rate and C-reactive protein is helpful. The duration of treatment is empirical but it should last for at least 3 weeks.

4. Splintage. The involved joint should be splinted until the infection is resolving. The hip should be splinted in abduction to prevent dislocation. There is some experimental evidence that continuous passive motion may result in less stiffness than splintage.

Complications

- Destruction of the capital epiphysis.
- Dislocation.
- Destruction of the epiphyseal plate.
- Growth disturbance including coxa magna.

Further reading

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Related topics of interest

Hand infections (p. 144)
Osteomyelitis (p. 208)
Skeletal tuberculosis (p. 284)

SHOULDER IMPINGEMENT SYNDROMES

Shoulder impingement can be diagnosed when a patient has a positive impingement sign (pain on passive elevation) and a positive impingement test (disappearance of the pain following intrabursal injection of anaesthetic). Two main impingement syndromes are described.

Subacromial impingement

The supraspinatus tendon normally glides smoothly in the subacromial space which is bounded by the unyielding confmes of the osteoligamentous coracoacromial arch above and the humeral head below. The causes of subacromial impingement can be subdivided anatomically into *extrinsic factors* such as spurs, osteophytes or variations in the shape of the acromion or coracoid and *intrinsic factors* related to primary cuff pathology.

They may also be classified on a pathological basis as:

(a) *Vascular:* the relatively avascular area 1 cm medial to tendon insertion is progressive with age. This 'critical zone' corresponds to the common site of degeneration and rupture of the cuff.

(b) *Degenerative:* tendonopathy at the site of insertion is associated with inflammatory or degenerative changes in the joint. This may produce a bony spur, intratendinous tears (reflected as osteopenia or cyst formation at the greater tuberosity) or reparative granulation tissue.

(c) *Traumatic:* this includes displaced fractures of the greater tuberosity, acute or acute-on-chronic cuff rupture as well as overuse or repeated stressing in the overhead position (occupational or athletic), and shoulder instability with persisting inflammation.

(d) *Mechanical or anatomical:* the antero-inferior one-third of the acromion causes mechanical wear of the rotator cuff. Certain anatomical shapes of acromion are implicated (curved and hooked) particularly in external surface partial tears. In certain conditions the acromioclavicular joint or coracoacromial ligament can contribute.

In the absence of bony changes the problem may be due to a thickened coracoacromial ligament, or anterior laxity with tightness of the posterior capsule. No single factor causes impingement: it is a varying contribution from each that causes the different clinical presentations.

Subcoracoid impingement

This produces symptoms because of impingement between the coracoid process and the lesser tuberosity.

Clinical problems

1. Subacromial impingement (supraspinatus syndrome). There is a painful arc of movement in active and passive forward flexion and abduction. Classically, the rotator cuff impinges between 60° and 120° of abduction. *Acute primary* injury in young athletic individuals causes oedema and haemorrhage. There is diffuse pain located over deltoid, local tenderness over the greater tuberosity, muscle spasm and limitation of movement. Impingement sign and test are positive. *Acute recurrent* symptoms occur up to 40 years, with fibrosis and tendonitis. Symptoms are similar to above but recur with activity and become progressive. There is often muscle atrophy. *Chronic impingement syndrome* occurs over 40 years of age with progressive persistent disability. In addition, symptoms often include locking and night pain. Frequently there is either a rotator cuff tear or biceps tendon rupture and secondary bone changes.

2. Subcoracoid impingement occurs in younger patients with an intact rotator cuff. Symptoms are more anterior and impingement occurs with the arm in flexion and internal rotation. Soft tissue impingement occurs between the lesser tuberosity and the coracoid/coracohumeral ligament complex. This presents in gymnasts, javelin throwers, swimmers, tennis players and others. It may also occur after fractures of the coracoid or lesser tuberosity, tears of subscapularis or long head of biceps, or after scarring from an anterior dislocation with coracohumeral ligament tear.

Management

1. Establish diagnosis. Clinical examination localizes the site of maximal tenderness. There is a positive impingement sign and impingement test. A rotator cuff rupture must be excluded.

2. Plain radiographs show non-specific changes, including osteopenia, cysts, notching of the head, dystrophic calcification or ossification of the coracoacromial ligament. There is decrease in the radiographic space (rotator interval) between the humeral head and the underside of the acromion in cases of rotator cuff deficiency. A space of 5 mm or less indicates a significant lesion (normal=7–14 mm).

3. Arthrography will demonstrate an associated rotator cuff lesion, biceps tendon pathology, or instability.

4. Subacromial bursography may identify partial thickness tears with a normal arthrogram.

5. MRI and ultrasound are being used increasingly to replace invasive contrast studies.

6. Diagnostic arthroscopy.

7. Non-surgical treatment consists of steroid or NSAID and local anaesthetic injection into the subacromial bursa; physiotherapy and occasional splintage; aspiration of acute calcific tendonosis.

8. Surgical treatment may require management of rotator cuff tear (see Rotator cuff injuries, p. 260); excision of calcific deposits; decompression acromioplasty with coracoclavicular ligament excision; excision of distal clavicle and acromioclavicular joint when there are prominent inferior osteophytes; biceps tendon sheath decompression; or arthroscopic debridement and decompression.

Complications

Chronic impingement may lead on to rotator cuff rupture, adhesive fibrosis, or degenerative arthrosis.

Further reading

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Related topics of interest

Arthritis of the shoulder (p. 42)
Arthroscopy (p. 49)
Rotator cuff injuries (p. 260)
Shoulder instability (p. 278)
Shoulder pain (p. 281)

SHOULDER INSTABILITY

The glenohumeral joint is the most mobile joint in the body but this mobility is achieved at the expense of intrinsic stability.

Clinical problems

Shoulder instability may be classified as either *traumatic* or *atraumatic*. The shoulder may dislocate or subluxate in one or more directions.

1. Acute anterior dislocation is the commonest shoulder dislocation and accounts for 98% of all cases. The humeral head usually dislocates to a subcoracoid or infraglenoid position in response to forced external rotation in an abducted extended position.

2. Acute posterior dislocation occurs less commonly and the diagnosis is often missed. The dislocating force is internal rotation combined with adduction and flexion, and not uncommonly follows electric shock injury or convulsions.

3. Recurrent instability. The reasons for recurrent instability are numerous. Much depends on the site and nature of the damage caused by the initial trauma. Paradoxically, the greater the trauma, the lower the rate of recurrence. The age of the patient is also important. Recurrent dislocations occur in 90% of patients who have their first traumatic dislocation under the age of 20 years, in 60% who dislocate for the first time between the ages of 20 and 40, and in only 10% who dislocate over the age of 40 years. Men are four times more likely to redislocate than women. There is no single predictive lesion to indicate recurrent instability, although avulsion of the glenoid labrum (Bankhart lesion) is more likely to occur in younger patients and capsular stripping in older patients.

4. Subluxation. Occasionally, the injury may be insufficient to dislocate the joint, but may still cause sufficient capsular damage to allow painful subluxation.

5. Atraumatic shoulder instability is increasingly being diagnosed, and probably accounts for about 10% of chronic shoulder instabilities. About half have generalized ligamentous laxity. It is essential to distinguish between shoulder instability and shoulder laxity. Laxity is a sign and can be demonstrated on examination, instability is a symptom and may only be evident by apprehension on examination. Atraumatic instability may be voluntary or involuntary. *Voluntary atraumatic instability* is often multidirectional, bilateral,

painless and present since childhood. There is often a personality disorder and primary treatment is directed at counselling and physiotherapy, not surgery. *Involuntary atraumatic instability* presents in teenage years with recurrent painless dislocation. Instability is usually multidirectional with a background of generalized joint laxity. The first line of treatment is aimed at strengthening and educating the shoulder musculature to avoid dislocation. This may be a protracted period of physiotherapy and only if this is unsuccessful after at least 12 months should surgery be considered.

Management

1. Imaging. Most structural lesions associated with shoulder instability are in the soft tissues and are not seen on *plain radiographs*. Secondary evidence of recurrent dislocation (e.g. Hill-Sachs lesion) may confirm the clinical diagnosis. *Double-contrast arthrography* has, in the past, been the investigation of choice. This allows visualization of labral or capsular defects or an over-capacious axillary pouch with anterior laxity. Special methods of investigation now include plain or contrasted *computerized tomography (CT)* and *magnetic resonance imaging (MRI)*.

2. Non-operative treatment. All patients presenting with a recurrent dislocation should undergo a supervised rehabilitation programme. No further treatment may be needed if modification of work or recreational activities can be achieved.

3. Operative. There are numerous surgical procedures advocated for both anterior and posterior instability. Before selecting a particular procedure or combination of procedures, the precise pathological lesion must be appreciated.

Shoulder arthroscopy demonstrates the glenohumeral ligaments well and is probably more useful than CT in the diagnosis of recurrent subluxation. Arthroscopic anterior stabilization of the shoulder using a modified Bankhart technique is practised in some specialized centres, but currently runs a higher risk of failure than an open operation.

For *anterior instability,* repair of a labral tear and tightening of a stretched capsule may be sufficient (Bankhart or du Toit and Roux). In the absence of a labral tear, imbrication of the capsule and subscapularis (Putti-Platt), subscapularis muscle and tendon advancement (Magnuson-Stack), or the transfer of the coracoid process (with its attached short head of biceps and coracobrachialis) to the anterior glenoid (Bristow-Laterjet) may be necessary.

When a *multidirectional instability* has failed to respond to non-operative measures, a capsular shift procedure may be required (e.g. Neer). This involves detaching the joint capsule from the humeral neck and advancing and overlapping flaps depending on the direction of greatest instability.

Surgery for *posterior instability* is rarely required. When indicated this may require posterior capsulorrhaphy with transfer of the long head of biceps to the posterior glenoid rim (Boyd-Sisk). When a bony deficiency is considered

responsible a redirectional osteotomy and bone graft (posterior glenoplasty) or a posterior capsular shift with a posterior bone block (Warren) may be used.

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Related topics

Arthritis of the shoulder (p. 42)
Arthroscopy (p. 49)
Shoulder pain (p. 281)

SHOULDER PAIN

In the shoulder, disorders of the periarticular soft tissues are more common than arthritic conditions, and account for up to 95% of all shoulder pain. Abnormalities of the rotator cuff tendons are the commonest and most important *intrinsic* cause of painful shoulder syndromes and are often associated with mechanical derangement related to degenerative change, or with an inflammatory arthropathy. Less commonly, primary and secondary tumours or infection may occur. It is important that a clear plan of investigation is employed to identify these urgent cases at an early stage. The anatomical site of shoulder pain should be determined by the history and examination. *Extrinsic* causes of shoulder pain such as cervical nerve root or diaphragmatic irritation, myocardial disease and thoracic outlet syndrome should be identified. Pain in the shoulder may be localized (e.g. with acromioclavicular disease), but is more often diffuse and poorly localized. Pain produced by certain activities may suggest the underlying cause but since shoulder movement is a composite of gleno-humeral, scapulo-thoracic and acromio-clavicular movements, the cause may remain obscure. Tears and intrinsic rotator cuff tendonopathies must be recognized and treated (see Rotator cuff injuries, p. 260, and Shoulder impingement syndromes, p. 275).

Clinical problems

Shoulder pain is not restricted to any particular age group, and the peak incidence is different for each given cause. The causes of primary shoulder pain are discussed here: other causes are discussed elsewhere.

1. Calcific tendonitis may present in two ways. The *acute syndrome* occurs as a sudden attack, often at night without any clear precipitating factor, and is resistant to analgesics. The *sub-acute syndrome* comes on over several months with features of impingement but with an overlying element of rest pain. Calcification occurs 1–2 cm medial to the insertion of supraspinatus in contrast to secondary dystrophic calcification which is seen at the point of insertion. Separate formative (subacute) and resorptive (acute) phases can be identified. Radiographs show uniformly dense calcification with well defined margins in the former and ‘fluffy scalloped’ calcification in the latter.

2. Shoulder impingement syndromes (see p. 275).

3. Rotator cuff tendonopathies (primary and secondary) (see p. 260).

4. Frozen shoulder was described by Codman in 1934. Its cause is unknown and it can only be diagnosed when other intrinsic and extrinsic causes of pain have been excluded. Symptoms include diffuse pain (often at rest) which is worse at night. There is progressive restriction of active and passive movement, particularly external rotation. This is caused by prolific fibrosis involving the joint capsule, the rotator cuff tendons and the coracohumeral ligament producing an 'adhesive capsulitis'.

5. Biceps tenosynovitis is most commonly a secondary phenomenon but is primary in 5–10%. It is usually due to overuse or direct injury sustained at work or sport. Occasionally it is due to a congenitally shallow bicipital groove or to a tight transverse humeral ligament. Pain and tenderness directly over the tendon of the long head of biceps (directly anterior over proximal humerus in 30° of external rotation) confirms the diagnosis. Tendon rupture is rare in primary cases and usually follows injudicious corticosteroid injection into the tendon rather than the sheath.

6. Subacromial bursitis is commonly secondary to a degenerative or inflammatory process but can occur primarily after acute or repetitive trauma. It presents as a subacromial 'painful arc syndrome' between 60° and 120° of abduction.

7. Glenohumeral arthritis (see p. 42).

8. Acromioclavicular joint (ACJ) disorder causes pain localized over the joint. It is often evident on passive stressing of the joint (adducting the arm), with a 'painful arc' in the last 30° of arm elevation. Common causes of disease at this site are rheumatoid arthritis, trauma and post-traumatic arthritis, infection and tumour.

Management

Clinical examination should record any weakness or wasting, specific sites of tenderness, the active and passive ranges of movement, response to provocative tests of stability, any painful arc and the results of relevant haematological investigations. Local anaesthetic sensory testing is used to abolish pain and identify its specific site of origin. The subacromial bursa, ACJ, gleno-humeral joint, and long head of the biceps sheath are injected in turn.

1. Special investigations. Plain radiographs including true antero-posterior and axillary views are necessary: ACJ views are obtained if indicated. These will identify an arthritis or calcinosis and show indirect evidence of rotator cuff lesions, impingement or instability. *Double contrast arthrography,* with or without CT, will identify rotator cuff tears, capsular and labral lesions suggesting instability, and loose bodies.

Ultrasound is a non-invasive and cheap investigation and can identify rotator cuff lesions. It is operator-dependent and with the availability of MRI its role is unclear. *Scintigraphy* is used in assessment of tumour or infection but is

otherwise of limited value. *MRI* is becoming increasingly helpful in the diagnosis of soft-tissue lesions. Its value in the treatment of tumours around the shoulder is already established. *Arthroscopy and bursoscopy* are used increasingly for diagnostic purposes to identify rotator cuff lesions, capsular and labral defects and bicipital tenosynovitis. They are also used for operative treatment (see below).

2. Non-surgical treatment. The majority of conditions will respond to rest, physiotherapy, NSAIDs and local injection of steroids. Acute calcific tendonitis responds well to needling of the deposit under image intensifier control.

3. Surgery. The treatment of arthritis, impingement, instability and rotator cuff tears is discussed elsewhere. Open excision of chronic calcinosis as an isolated procedure or in combination with others is sometimes indicated. Arthroscopic decompression of the biceps tendon, synovial biopsy, or removal of loose bodies is carried out as necessary. EUA is carried out to exclude instability and MUA in patients with restricted movement due to frozen shoulder.

Further reading

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Related topics of interest

Arthritis of the shoulder (p. 42)
Arthroscopy (p. 49)
Rotator cuff injuries (p. 260)
Shoulder impingement syndromes (p. 275)
Shoulder instability (p. 278)
Thoracic outlet syndrome and cervical rib (p. 324)

SKELETAL TUBERCULOSIS

Tuberculosis (TB) is caused by infection with *Mycobacterium tuberculosis* in the majority of cases: *M. bovis* and the rarer mycobacteria account for the rest. The incidence is decreasing in the Western world but not in Africa and Asia where there are 400 new cases per 100 000 population per year. One-tenth of all those infected have musculoskeletal TB.

Clinical problems

1. Transmissal TB is spread by infected sputum. Patients inhale the tubercle which is phagocytosed in the alveoli forming a *Gohnfocus.* The phagocytes transport the tubercle to the regional hilar lymph nodes which, with the involved lung, form the *primary complex.* The tubercle multiplies in the phagocytes and a cell-mediated immune response occurs giving rise to a *granuloma.* The centre may undergo necrosis (caseation): attempts at healing are associated with calcification. The initial disease may be subclinical or may present with a pneumonitis. Re-activation may occur at any time. Spread to the bloodstream occurs either through the thoracic duct or by direct invasion of blood vessels by involved lymph nodes.

2. Spine. Involvement of the spine occurs in 50% of those with disease of the musculosketal system. The thoracolumbar segments are most commonly affected but any part of the spine may be involved. The infection normally starts in the vertebral body but can occasionally involve the posterior elements. Involvement of an end-plate may lead to spread across the disc into the neighbouring vertebrae. A paravertebral abscess frequently forms and may track along tissue planes to point at a site distant to the spine (e.g. psoas abscess pointing in the groin). Collapse of the vertebral body will cause an acute gibbus or kyphos. Involvement of C1/C2 may lead to erosion of the atlas and destruction of the odontoid peg. Instability frequently results. Neurological deficits may be gradual in onset or rapid and complete. Most are secondary to medullary and radicular inflammation rather than to extradural compression by abscess, tuberculoma or subluxation of a vertebra.

3. Septic arthritis. A monoarticular arthritis is the rule. The order of frequency of involvement is hip, knee, ankle, sacro-iliac joint, wrist and shoulder. The joint

is warm, but not hot like a bacterial septic arthritis. The synovium has a thickened doughy feel. Muscle atrophy is common and sinuses may occur. A decreased range of movement leads to a fibrous or bony ankylosis.

4. Osteomyelitis. Any long bone may be affected. The patient presents with localized pain and swelling. Dactylitis may affect the fmgers or toes. It is most common in children under 10 years and presents initially with swelling. Cystic TB also occurs in children and presents with circumscribed lesions in bones including the skull. Poncet's disease is thought to be related to a reactive type of polyarticular arthropathy.

5. Systemic features.

Management

1. Diagnosis. The history and examination often yield the diagnosis. An ESR may be helpful but is non-specific. The tuberculin test may be confused by the BCG. A strongly positive reaction to 1 tuberculin unit (TU) is strongly suggestive of infection while no reaction to 100 TU implies no infection. Five percent of infected individuals do not react to tuberculin.

2. Radiographs. Osteoporosis due to hyperaemia, with soft tissue swelling, loss of joint space and ragged subchondral bone are suggestive of TB. In the spine, adjacent vertebral bodies may be eroded, with loss of the involved disc space. Soft tissue swelling is best seen on CT or MRI scanning. More advanced destruction is associated with kyphosis. In children, a compensatory curve may be seen above or below the involved segment if the adjacent vertebrae have overgrown. The differential diagnosis is usually from tumour or staphylococcal infection.

3. Biopsy. Tissue may be collected for culture by biopsy and is grown on Lowenstein-Jensen medium for 4–8 weeks. Smears reveal slender beaded rods on ZN staining. Histology shows granulomas with giant cells, epithelioid cells and central caseation. In the synovium an epithelioid infiltrate is seen but granulomas may be absent.

4. Medical treatment. There are various chemotherapeutic regimes, of which one is rifampicin 450–600 mg, isoniazid 300 mg and pyrazinamide 1000–1500 mg daily for 2 months followed by rifampicin and isoniazid daily for 6 months. Treatment should continue for longer in children and in those with a spinal abscess. Patients must be monitored for 12 months after ceasing treatment. Hepatic and renal impairment are common with these drugs. Patients should also have rest and a good diet.

5. Surgery. Surgery is rarely required. A synovectomy can be considered in those with pronounced synovitis. An abscess may require drainage. In the spine, neurological impairment is usually secondary to inflammation and treatment should be medical not surgical. In very rare instances, decompression may be performed but only when there is defmite evidence of neural compression. Ankylosis of hip or knee may develop spontaneously: it may be necessary to

perform an arthrodesis. Joint arthroplasty should not be performed in active disease. In old affected joints, conversion of ankylosis or arthrodesis to arthroplasty should be covered by antituberculous therapy for 3 weeks pre-operatively and at least 6 months post-operatively. Arthroplasty in this situation is associated with a 12% sepsis rate.

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Related topics of interest

Osteomyelitis (p. 208)
Septic arthritis (p. 272)
Spinal infection (p. 302)

SLIPPED UPPER FEMORAL EPIPHYSIS

The incidence of slipped upper femoral epiphysis (SUFE) varies from 1 to 3 per 100000 and is more common in boys (3:2). The peak incidence occurs about 2 years earlier in girls than boys and is related to puberty. Individuals often have delayed bone age. The reported incidence of bilateral disease, in symptomatic cases, is up to 20% and up to 40% when a second asymptomatic hip is identified.

Experimental evidence shows that oestrogen can increase the strength of the growth plate and that growth hormone can decrease it. In both sexes, the strength of the growth plate is at its lowest at puberty. As in traumatic slipped epiphyses, the slip occurs through the hypertrophic layer of cells. There is a clinical association between SUFE and some endocrine conditions such as hypothyroidism, pituitary dysfunction and hypogonadism.

Clinical problems

The presenting symptoms are usually of pain in the groin, thigh or knee, and a limp. There are three recognized presentations categorized by the duration of symptoms.

1. Chronic slip. This is the most common type and presents with a gradual and progressive onset of symptoms lasting 3 weeks or more. The child walks with an antalgic Trendelenburg gait and the limb is externally rotated. Examination reveals reduced internal rotation (particularly in flexion), reduced flexion and abduction. Typically, the limb externally rotates on flexion of the hip. There is invariably a synovitis.

2. Acute slip. An acute slip usually presents with sudden severe symptoms, sometimes after a mild prodromal period lasting less than 3 weeks. There may be a history of insignificant trauma.

3. Acute-on-chronic slip. A sudden exacerbation of symptoms on top of a chronic pattern of symptoms.

Management

1. Radiology. Antero-posterior and true lateral views of both hips are required. Radiographic features of SUFE include a widened and irregular growth plate,

loss of height of the epiphysis, the 'blanch sign' (a localized increased density in the metaphysis), and an increase in the distance of the metaphysis from the medial wall of the acetabulum. In the lateral view the 'Klein line' drawn along the superior cortex of the femoral neck transects less of the epiphysis than the normal side. With a chronic slip, as the head slips posteriorly relative to the neck, the periosteum is stripped from the posterior and inferior surfaces. This produces an appearance of a varus retroverted neck. A CT scan can be useful in planning secondary treatment but does not aid the initial diagnosis.

2. Conservative treatment alone has no role in this condition. The aim of primary treatment is to prevent a progressive deformity without producing the recognized complications of treatment which are avascular necrosis of the femoral capital epiphysis, and chondrolysis. Failure to achieve this will lead to accelerated joint degeneration. Chondrolysis is identified by hip pain and stiffness, and by radiographic narrowing of the joint space, typically between 2 months and 1 year after diagnosis.

3. Primary treatment of SUFE is fixation of the femoral epiphysis *in situ.* The complications of treatment are chondrolysis and avascular necrosis and can be ascribed to operative technique. Chondrolysis is more common when fixation devices penetrate the joint. Avascular necrosis is associated with fixation devices placed in the superior part of the femoral head at the site of the lateral epiphyseal artery. This consistent artery supplies the weight-bearing part of the femoral head and injury to it may cause avascular necrosis. Fixation should be achieved by a single screw placed centrally in the epiphysis crossing the growth plate at 90°. The potential for remodelling exists following successful fixation *in situ,* with even moderate and severe slips recovering satisfactory function.

4. Secondary treatment may consist of *manipulation.* If this is performed on an acute slip, under anaesthetic, the incidence of avascular necrosis is high. When performed gradually, using traction, this complication is less common. *Osteotomy* can be required if deformity results and the congruity or arc of movement of the joint are unacceptable. The site of the corrective osteotomy can be at the level of the growth plate, the base of the femoral neck, or intertrochanteric. The more distal the chosen site, the lower the reported rate of avascular necrosis and chondrolysis.

5. Salvage procedures may be necessary in severe instances of avascular necrosis or chondrolysis not controlled by conservative measures. This may include valgus osteotomy, total joint replacement or arthrodesis.

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Related topics of interest

Arthritis of the hip (p. 35)
Avascular necrosis of the hip (p. 53)
Hip arthroplasty (p. 150)
Irritable hip (p. 159)
Osteoarthritis (p. 202)
Young adult hip problems (p. 340)

SOFT TISSUE DISORDERS OF THE ELBOW AND FOREARM

Elbow pain arising in the soft tissues occurs in 1–3% of the general population and is common in sportsmen, manual workers and in industrial workers who do repetitive tasks.

Clinical problems

1. Tennis elbow (lateral epicondylitis). This is commonest in the fourth and fifth decades and may be related to degenerative change in the connective tissues. It is also seen in amateur tennis players, where it is related to their backhand stroke, and in professionals in relation to their serve. The pain is of gradual origin and is usually well localized, but it may radiate to the extensor aspect of the forearm. Patients often complain of a weak grip and of dropping objects. Resisted wrist extension is painful. Tenderness is usually localized to the lateral epicondyle and supracondylar ridge. The pathology is uncertain but is probably that of a degenerative or traumatic tear of the tendo-periosteal attachment of extensor carpi radialis brevis and the common extensor muscle origin. Other theories include a humero-radial bursitis, inflammation of the annular ligament of the radius, the presence of a synovial fold between the humerus and radial head and posterior interosseous nerve entrapment.

2. Golfer's elbow is thought to be caused in the same way as tennis elbow but affects the medial epicondyle. It is less common.

3. Olecranon bursitis. Inflammation of the bursa between the insertion of triceps and the subcutaneous tissue is supposedly common in students as a resuult of resting on the elbow. It may become infected.

4. Pitcher's elbow. A repeated valgus strain on the elbow may cause pain over the ulnar collateral ligament. Lateral joint overload may be associated with osteochondritis dissecans of the capitellum and the radial head. Medial joint overload may lead to the development of posteromedial traction osteophytes. In children, epicondylitis or avulsion of the epicondyle may occur. Ulnar nerve entrapment or traction neuropathy rarely develop.

5. Ulnar nerve entrapment. The ulnar nerve may be compressed at the arcade of Struthers near the medial intermuscular septum, in the cubital tunnel and between the heads of flexor carpi ulnaris. An ulnar nerve palsy may occur due to

traction over a malunited fracture. Hypoplasia of the trochlea and an inadequate fibrous arch may result in subluxation or dislocation of the nerve to the front of the epicondyle. The diagnosis should be confirmed by EMG studies.

6. Posterior interosseous nerve entrapment. This may produce pain very similar to that caused by a lateral epicondylitis. Entrapment may occur at the extensor carpi radialis brevis, from adhesions about the radial head, an abnormal recurrent radial arterial fan, the arcade of Frohse, after fractures of the elbow or forearm, or from synovitis of the elbow in rheumatoid disease. Pain on resisted supination is characteristic. Some or all of the extensors may be affected. EMG may help the diagnosis.

Management

1. Clinical assessment. Careful history and examination will often locate the source of the problem. Tenderness over the epicondyle and supracondylar ridge is suggestive of tennis or golfer's elbow.

2. Investigations. Plain radiographs of the elbow should be performed to exclude any bony cause for the symptoms but in the majority of cases these will be normal. A bone scan will usually be normal. Magnetic resonance imaging may reveal areas of inflammation related to tennis elbow. It may also demonstrate other causes of ulnar nerve compression (e.g. ganglia). EMGs should be performed in cases of suspected posterior interosseous and ulnar nerve lesions.

3. Conservative treatment. Rest, steroid injections and physiotherapy will settle over 90% of cases of tennis elbow. There is some evidence that ultrasound reduces the rate of recurrence more than steroid injection. There is no convincing evidence that NSAIDs are beneficial. At least 50% of cases of ulnar nerve compression will respond to conservative treatment.

4. Arthroscopy. This is indicated in cases of pitcher's elbow where injury to the articular surface is likely and removal of loose bodies or debridement may be beneficial.

5. Surgery. The procedures described for tennis elbow include release of the common extensor origin, excision of part of the annular ligament, release of the fibrous arch of the superficial part of the supinator overlying the arcade of Frohse and osteotomy of the lateral epicondyle. There is no evidence that any procedure other than release is indicated, nor is there clear evidence that resistant tennis elbow is caused by posterior interosseous nerve palsy. In the case of ulnar nerve entrapment behind the medial epicondyle, simple release of the nerve should be carried out by dividing the fibrous roof of the cubital tunnel. Results are best in those with a short history and definite constriction. Transposition may devascularize the nerve and should be reserved for cases of subluxation of the nerve, obliteration of the tunnel by osteophyte, gross valgus of the elbow or when the constriction is proximal to the cubital tunnel. Transposition should be

submuscular and the medial intermuscular septum must be excised to avoid kinking of the nerve.

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Related topics of interest

Peripheral nerve entrapment (p. 225)

Repetitive strain injury (p. 242)

SOFT TISSUE SARCOMAS

Soft tissue sarcomas (STS) are uncommon, but not rare, and account for 0.5% of all malignancies in adults. They are at least five times as common as primary malignant bone tumours. Their incidence is estimated at between 13.5 and 20/ million/year. This appears to be increasing. Their pathogenesis is unknown. They affect all age groups. Fifteen percent affect those under the age of 15 years and 40% affect those over the age of 55. Males are affected more than females. They can arise anywhere, but the most common sites are the large muscle groups of the upper (20%) and lower (40%) extremities, the head and neck (10%) and trunk (30%), of which 10% are retroperitoneal. It is the extremity tumours which are most likely to present to the orthopaedic surgeon.

Classification

Sarcomas are classified according to the tissue type from which they originate. The commonest types are fibrosarcoma, malignant fibrous histiocytoma, liposarcoma, rhabdomyosarcoma, leiomyosarcoma, synovial sarcoma, malignant schwannoma and angiosarcoma. Rarely seen are alveolar soft parts sarcoma, extraosseous Ewing's sarcoma, clear-cell sarcoma of tendon sheaths and aponeuroses, epithelioid sarcoma and other rare varieties.

STS may be of high or low histological grade. High grade tumours are more common. Large tumours (>5cm) metastasize more commonly than small tumours. Spread is through the bloodstream to the lungs and liver. Regional lymph nodes are rarely affected (<4%) but their involvement indicates a poor prognosis.

Clinical presentation

STS typically present with a mass. Malignancy should be expected in any mass which is deeply seated in muscle, rapidly enlarging, painful or more than 5 cm in diameter. However, some STS (of better prognosis) may be small, superficial, painless and slowly enlarging.

Management

1. Staging is carried out either using the Enneking surgical staging (see Bone tumours—malignant, p. 68) or by the TNM system proposed by the American Joint Committee Task Force on Soft Tissue Sarcomas.

2. Biopsy is carried out using the principles described for malignant bone tumours. Tru-Cut biopsy may be used but sampling must be representative and the reporting pathologist must be experienced in the interpretation of small samples.

3. Resection aims to excise the whole tumour along with its surrounding myofascial coverings. *Superficial* tumours may be treated by wide local excision but deep intramuscular tumours usually require compartmental excision. The whole myofascial compartment containing the tumour is widely exposed and resected and its muscle attachments are sectioned close to their bony origin. Vascular resection and reconstruction are needed only rarely. The frequency of amputation in specialist units should be less than 15%.

Resection alone carries a high recurrence rate, which is determined by the margin of resection. Recurrence occurs in 25% after a radical compartmental excision, 50% after a wide resection, 90% after a marginal resection and 100% after an intralesional resection (where, of course, tumour is left behind). Surgery should almost always be followed by radiotherapy once the wounds have healed.

4. Radiotherapy alone can sterilize up to one-third of all STS, but in most cases should be used in conjunction with surgery. The patient should be seen preoperatively in joint consultation with a radiotherapist. For mobile tumours there is no proven difference between pre- and postoperative radiotherapy. Postoperative radiotherapy is preferred because it allows fuller histological assessment of the tumour and does not prejudice wound healing. Preoperative radiotherapy may be employed for fixed tumours and those in ‘difficult’ sites. In these cases surgery should be delayed for several weeks after irradiation.

5. Chemotherapy has proved disappointing in these tumours. No significant increase in long-term survival has been achieved by any of the agents which are currently available.

6. Prognosis. The major determinants of death from STS are the size and histological grade of the tumour at the time of treatment. Whether the tumour is within an anatomical compartment or outside it does not appear to be significant in terms of overall prognosis. The principal factors affecting local recurrence are probably tumour size, histological grade and margin of resection.

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Related topics of interest

Bone tumours—benign (p. 63)
Bone tumours—malignant (p. 68)
Bony metastases (p. 74)

SPINA BIFIDA

Spina bifida is a developmental abnormality of the neural tube and paraxial mesoderm which produces posterior element deficiencies. The incidence of spina bifida is estimated at 3 per 1000 live births, although there is wide geographical variation. The incidence is increased in first-born children and with lower social class. It is familial. As a result of antenatal screening and folic acid supplements in pregnancy, the incidence is falling. The neural tube has completed closure by the 25th day of foetal life. The extent of myelodysplasia depends upon the timing of the pathological process. Deformities range from total failure of closure (rachischisis) to mild forms (spina bifida occulta).

Clinical problems

1. Spina bifida occulta. Approximately 15% of clinically normal individuals have radiographic evidence of spina bifida occulta of the lower lumbar or upper sacral segments. In a small proportion there is cutaneous evidence in the form of a lipoma, haemangioma, naevus, hairy naevus or a skin dimple. Spina bifida occulta can cause cord tethering which becomes significant if surgery is contemplated for correction of other spinal deformities, e.g. idiopathic scoliosis.

2. Spina bifida aperta (cystica). Three main clinical types occur:

- *Closed myelomeningocele* and *spina bifida occulta.*
- *Simple meningocele:* consists of a herniation of dura and arachnoid filled with cerebrospinal fluid. This is most common in the lumbosacral and sacral regions. The lower limbs are not deformed at birth and neurological examination is normal. Operative repair within 48 hours is recommended.
- *Open myelomeningocele:* a failure of fusion in which the spinal cord is opened out as a neural plaque flush with the skin: 60% are in the lumbar or lumbo-sacral spine and 20% in the thoraco-lumbar spine. The degree of deformity and neurological abnormality may vary from none to severe involvement. Early operative closure prevents progressive paralysis and reduces infection. Hydrocephalus is common and requires shunting.

3. Spinal deformity. The spinal deformity in spina bifida is paralytic in origin in 60% of cases. The commonest deformity is a simple *lordosis* which progresses slowly with age and increasing weight. By contrast, a *kyphosis* associated with a high neurological lesion progresses rapidly at an early age. *Neonatal kyphosis* can be so severe that spinal osteotomy may be needed to obtain primary soft tissue cover of the defect. *Progressive adolescent kyphosis* (or a kyphos) may require correction to prevent skin ulceration. Progressive deformity often develops at the age of 5 or 6 years, particularly with high neurological lesions. It may initially be controlled by bracing but by 10 years may require operative spinal correction and fusion.

In 40% of cases, spinal deformity is secondary to a congenital bone anomaly situated at a level just proximal to the neurological lesion. The development of subsequent spinal deformity depends on the degree of asymmetric muscle activity, the nature of the anomaly and the presence of obesity with approaching adolescence. This causes difficulty with seating and walking. The lower the level of neurological lesion, the better the prognosis for the spine. A significant spinal deformity will be present in 80% of those reaching the age of 20.

4. Hip instability and contracture. Coronal plane asymmetry caused by muscle imbalance and the effect of gravity typically causes a long C-shaped lordo-scoliosis with pelvic obliquity and a 'wind-swept' hip deformity. The hip dislocates on the 'high' side and pressure sores commonly develop on the 'low' side. By addressing the lordo-scoliosis early, this sequence can be prevented. The aim is to produce a stable straight limb which allows the patient to stand with callipers, while retaining enough flexibility to allow sitting.

There are recognizable patterns of birth deformity dependent on the level of neurological defect. A lesion above Ll produces a balanced paralysis with flail limbs. Only splintage is required. With a lesion at or below Sl, there may be a pure flexion deformity. Usually the lesion is between Ll and Sl: hip dislocation is the commonest problem. In this group, 50% have dislocated hips by the age of 2 years as a result of muscle imbalance. Reduction may be obtained by closed or open methods with soft tissue release (e.g. adductor tenotomy). In contrast to cerebral palsy, the lack of skin sensation in patients with spina bifida restricts the use of postoperative splintage, and therefore muscle imbalances must be corrected. Psoas transfer around the hip may be needed.

5. Knee deformity. Knee deformities are less common than deformities of the hip or foot. Most are not severe and can be managed by gentle passive manipulation. Deformity of the knee should be treated after hip deformity has been corrected. Similarly, significant knee deformity should be corrected before foot deformity. *Fixed recurvatum* is secondary to paralytic shortening of the quadriceps and is treated by surgical quadriceps elongation. In a *stiff knee with a flail limb,* simple division of soft tissues to allow sitting is appropriate. This may require hamstring lengthening, hamstring tenotomy alone or in combination with transplantation of semitendinosus, biceps femoris and gracilis to the extensor mechanism. A resolving deformity may be corrected with a supracondylar

femoral osteotomy. *Fixed flexion* may require soft tissue or bony correction. *Fixed valgus deformity* occurs as a secondary effect on the lower femoral or upper tibial epiphysis. Appropriate correction may be obtained by femoral or tibial wedge osteotomies.

6. Foot deformity. The floppy balanced foot needs no surgery but requires well fitting footwear and appropriate orthoses. The aim is to prevent contracture by routine stretching and obsessive skin care. A fixed foot deformity may occur in the presence of muscle imbalance. The treatment is as for the specific foot deformity with tendon transfer if imbalance is present (see Paralysed lower limb, p. 218). Pes cavus and clawing of the toes may be the only deformity with poor intrinsic muscle strength. Claw toes are treated by flexor tenotomy or flexor to extensor transfer as indicated.

Further reading

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Related topics of interest

Congenital foot deformities (p. 88)
Congenital knee anomalies (p. 94)
Developmental dysplasia of the hip (p. 119)
Neuromuscular disorders (p. 195)
Paralysed lower limb (p. 218)
Scoliosis—early onset (p. 263)
Spinal fusion (p. 299)

SPINAL FUSION

Spinal fusion is undertaken for three basic reasons. First, to stabilize the unstable spine; second, to provide long-term correction of spinal deformity; and third, to relieve pain.

Indications

1. Stabilization. The spine may be rendered unstable by trauma, infection, tumour, iatrogenic injury (failed or inappropriately performed surgery), spondylolysis and spondylolisthesis and, to a lesser extent, degenerative disc disease.

2. Correction of deformity. Spinal fusion is used to secure a correction which has been obtained by mechanical means. Any purely mechanical means of correction will fail unless a fusion is undertaken concurrently and becomes soundly incorporated. Fusion may also be used on its own to prevent a deformity from deteriorating (fusion *in situ*).

3. Relief of pain. Spinal fusion for low back pain is based on the principle that by eradicating the movement between two adjacent vertebrae, pain arising from their articulations (two apophyseal joints and the intervertebral disc) will be eliminated. Studies on cadaveric spines have shown that rigid fixation of the anterior column does not entirely eliminate movement in the posterior column: nor does rigid fixation of the posterior column abolish all movement in the anterior column. For this, and other reasons, lumbar spinal fusion for low back pain in the absence of instability or deformity should be approached with extreme caution: the results obtained are less satisfactory than those achieved for instability.

Techniques

The spine may be approached from the front, from behind or circumferentially. Fusions may be augmented with instrumentation. The addition of instrumentation to correct deformity or to stabilize the spine is of undoubted benefit for the first few months after surgery. The use of instrumentation in the

treatment of low back pain without overt instability is more doubtful and is currently being reevaluated.

1. Anterior interbody fusion in the cervical spine can be carried out using the techniques described by Bailey and Badgley, Robinson and Smith, and Cloward. In each case an anterior discectomy is performed and a preshaped graft is fitted between the prepared vertebral bodies. Fusion rates are between 82% and 100%. In situations such as extensive tumour resection, where fusion is required over several levels, a fibular strut graft provides excellent anterior stability.

In the *lumbar spine,* anterior interbody fusion is probably best reserved for the treatment of failed posterior fusions and for the anterior correction of deformity. While several groups have reported good results (80–95% pain relief) after primary anterior fusion for degenerative disc disease, the rate of complications reported is also higher. When used as a salvage procedure, both the fusion rate and the success rate are about 56%.

2. Posterior lumbar interbody fusion is only carried out in the lumbar spine where the cauda equina can be displaced with relative safety. It is technically difficult to perform. Fusion rates of up to 90% have been reported with 80–85% good or excellent results.

3. Posterior interlaminar fusion. This technique is mostly confined to the cervical and thoracic spine. It has the disadvantages that it cannot be used if there has been a midline decompression and that it makes any subsequent surgery to the neural structures impossible without taking down the fusion. For these reasons, its uses are limited to fusion accompanying the correction of spinal deformity and for instability of the posterior column in the cervical and thoracic spines.

4. Postero-lateral fusion is probably the treatment of choice when a posterior fusion is required in the region from L3 to S1. A variety of methods have been described but all rely on the removal of bone from the facet joints, pars interarticularis and transverse processes while leaving the midline untouched. Fusion rates of up to 95% have been claimed, but a realistic estimate of success is nearer to 75%.

5. Intertransverse fusion is less destructive of the midline structures than postero-lateral fusion, but is more difficult to achieve because of the narrower base for grafting. It is most useful for lumbosacral fusion (alartransverse fusion) where fusion can be achieved between adjacent structures of relatively large surface area.

Complications

1. Pseudoarthrosis has been reported in all large series. Half of all patients with a radiological pseudoarthrosis will be asymptomatic. Pseudoarthrosis is suggested by pain, progression of deformity and the demonstration of movement at the fusion site. Re-exploration of the fusion is the most reliable means of diagnosis.

2. Neurological damage. The risk of neurological damage is increased by the use of fixation systems particularly when pedicle screws are used. Nerve root damage has been reported in up to 15%.

3. Damage to anterior structures is a particular problem in the neck (see Acute cervical disc disease, p. 1) but also exists in the lumbar spine. Any structure that is encountered may be damaged directly, by inappropriate retraction or compression.

4. Graft site pain is common. It may be avoided by the use of allografts, but at the expense of a lower fusion rate. The instillation of local anaesthetics at the end of the operation has been recommended.

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Related topics of interest

Acute cervical disc disease (p. 1)
Allografts and bone banking (p. 12)
Bony metastases (p. 74)
Congenital neck anomalies (p. 98)
Degenerative disease of the cervical spine (p. 113)
Degenerative disease of the lumbar spine (p. 116)
Kyphosis (p. 170)
Low back pain (p. 181)
Neuromuscular disorders (p. 195)
Postural correction in ankylosing spondylitis (p.233)
Rheumatoid neck (p. 254)
Scoliosis—early onset (p. 263)
Scoliosis—late onset (p. 267)
Spina bifida (p. 296)
Spinal infection (p. 302)
Spinal stenosis (p. 306)
Spinal tumours (p. 310)
Spondylolysis and spondylolisthesis (p. 317)

SPINAL INFECTION

Infections of the spine can involve the vertebral body, intervertebral disc, epidural space, paraspinal soft tissues and, rarely, the posterior elements of the spinal column.

Clinical problems

1. Acute pyogenic vertebral osteomyelitis. This accounts for between 8% and 18% of all cases of osteomyelitis. It is particularly prevalent in debilitated patients and those with diabetes. Males are more commonly affected than females (2:1). *S. aureus* is the commonest infecting organism. Spread of infection to the vertebra may be by local or haematogenous spread from a primary infected focus. The subchondral bone, being highly vascularized, is the site of predilection. From here, infection may spread directly into the disc and adjacent vertebral body or may break out anteriorly causing a retropharyngeal, paraspinal or psoas abscess. Posterior extension made lead to epidural abscess formation.

Infection may follow an acute or chronic course, depending on the virulence of the organism and the immunological status of the patient. The patient presents with severe back pain and spasm, low-grade fever and malaise and may be reluctant to move or bear weight. There may be mild neurological disturbance in the lower limbs. Paralysis may occur due to compression of the cord or cauda equina by an abscess, by collapse with kyphosis, or by vascular injury to the cord. Paralysis is most likely in the cervical spine and least likely in the lumbar spine.

The earliest changes are seen on scintigraphy and MRI. Radiographs show no changes for 2 weeks. There is then narrowing of the disc space with rarefaction of the end-plates and, if paraspinal abscess formation has occurred, a soft-tissue mass. Late changes are destruction of two or more adjacent vertebral end-plates with disc narrowing and angulation. Diagnosis is by positive identification of the causative organism either in blood cultures (25%) or on open (85%) or closed (70%) biopsy.

Treatment is by rest, immobilization and systemic antibiotics for at least 6 weeks. Surgery is indicated for abscess drainage, decompression of the cord or roots, correction of deformity, biopsy or the failure of non-operative treatment.

2. Pyogenic discitis is often of insidious onset with low back pain. Consequently, diagnosis may be delayed for many months. Scintigraphy and MRI are likely to show the earliest changes. Infection may start in the disc and spread to the adjacent vertebral body or may follow vertebral osteomyelitis. An isolated discitis may cause cord compression in up to 40% of cases. Treatment is then by anterior discectomy, debridement and stabilization followed by systemic antibiotics for 6 weeks. The disease in adults may be part of a spectrum which includes the pyogenic causes of infection of the disc and vertebral body in children.

3. Discitis of childhood. This is a rare condition of young children usually caused by 5. *aureus*. It tends to affect the lumbar spine. The child presents with malaise or feverishness, back or hip pain and a limp or unwillingness to walk. Radiographs are normal in the early stages. WBC and plasma viscosity may only be mildly elevated and blood cultures are often normal. The diagnosis is made early on scintigraphy or later, after 2 weeks, when the characteristic radiological changes may be appreciated. These include disc space narrowing and thinning and irregularity of the vertebral end-plate. Treatment is by bed rest and antibiotics in the first instance. Biopsy is reserved for those cases who fail to respond.

4. Epidural abscess. Pyogenic epidural abscess is rare. It may occur by haematogenous spread, local spread or after surgical intervention. The anterior epidural space is involved when there has been local spread from a pyogenic discitis, but the posterior epidural space is preferred because of its vascularity. *S. aureus* is the commonest infecting organism. Neural injury occurs from pressure or infective thrombosis of the vessels.

The clinical features progress through a recognized course. There is severe local back pain, root pain, diminished sensation, motor weakness and sphincter disturbance proceeding to complete paralysis if untreated. There may be systemic signs of infection.

Investigation should include FBC, viscosity and blood cultures. Radiographs may be normal. Scintigraphy will identify osteomyelitis of an adjacent vertebra. Myelography or MRI will demonstrate the abscess.

Treatment is by open surgical drainage. This should be carried out as soon as the diagnosis has been made. The appropriate intravenous antibiotics should be given for at least 2 weeks. If vertebral osteomyelitis is present, they should be continued for at least 6 weeks. The prognosis for neurological recovery is related to the duration and severity of paralysis.

5. Postoperative infection. The incidence of postoperative infection increases with the complexity of the procedure undertaken. The risk should be under 1% for simple discectomy but may rise to 5% for non-instrumented spinal fusions and be higher than this when instrumentation is used. The most commonly identified organisms are *S. aureus,* coagulase-negative *Staphylococcus* and

bowel organisms. Pre-operative risk factors are prolonged bed rest, malnutrition, extreme age, diabetes, infections at other sites and immunosuppression. Postoperatively, spinal orthoses and caudally placed drains are thought to increase the risk of infection. Patients present with the local and systemic signs of wound infection. Treatment should be prompt and aggressive. Mild infections may settle rapidly on the appropriate antibiotic but others should be meticulously debrided, layer by layer, until complete eradication of the infection is ensured. Major bone grafts and implants are left in place if possible but may have to be removed in cases of severe infection. Wounds may be closed primarily, secondarily after further debridement, or may be left to granulate. Parenteral antibiotics should be given for at least 6 weeks if the infection is severe.

6. TB. See Skeletal tuberculosis (p. 284).

7. Others. Rare causes of infection are *Brucella,* coccidiomycosis, echinococcus and hydatid disease.

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Related topics of interest

Low back pain (p. 181)
Musculoskeletal imaging (p. 190)
Osteomyelitis (p. 208)
Septic arthritis (p. 272)
Skeletal tuberculosis (p. 284)
Spinal fusion (p. 299)

SPINAL STENOSIS

Spinal stenosis is caused by narrowing of the spinal canal, either in its central portion, or peripherally in the lateral recess or nerve root canal. The peak age of presentation is in the fourth to sixth decades of life. Men are nearly twice as likely to be affected as women. Symptoms only occur when the contained neural elements are compressed. The mechanism by which this compression produces 'neurogenic claudication' is unclear. Ischaemia, mechanical irritation and a rise in intraosseous and/or extradural venous pressures have all been cited as possible causes.

Any or all of the structures abutting the canal may contribute to this compression. Commonly, in degenerative stenosis, the desiccated intervertebral disc bulges into the canal anteriorly, while the facet joints, hypertrophied by osteoarthritis, protrude inwards posterolaterally, carrying with them the buckled ligamentum flavum. This causes the characteristic trefoil shape of the spinal canal of central stenosis. If the facet joints enlarge anteriorly and the anterior disc bulge is broad-based, the lateral recesses and root canal will be compromised.

Clinical problems

The clinical presentation of spinal stenosis is remarkably constant, although it may be difflcult to distinguish between central and peripheral stenosis on purely clinical grounds. The presentation is of low back pain radiating to the buttock and leg which starts while walking. Both legs may be affected. Unlike vascular claudication, sensory symptoms, including paraesthesiae, dysaesthesiae and cramps, tend to overshadow complaints of pain although pain is not an uncommon symptom. The legs feel heavy or weak and 'drag', particularly when climbing hills or stairs. The symptoms are unlike those of sciatica, being unaffected by coughing or straining, and are seldom as sharp or lancinating. They are eased by forward flexion, sitting and resting and are exacerbated by forced extension. Rarely, there may be sphincter disturbance.

Physical signs may be entirely absent in early cases and may be rather non-specific when present. The patient may adopt a 'simian stance' with flexion at the hips and knees. Spinal movements may be restricted, particularly in those with degenerative disease. Straight-leg raising is frequently normal and

Lasegue's test is not predictable. Ankle reflexes may be depressed or absent but are not diagnostic. Some sensory loss may occur in the L4, L5 and Sl dermatomes. Motor loss should be sought after exercising to the claudication distance (or on stairs).

The international classification of spinal stenosis (Arnoldi *et al.,* 1976) is based on the clinical cause of the narrowing.

- *Congenital/developmental stenosis.* Either idiopathic or due to achondroplasia.
- *Degenerative stenosis.* Affecting the central or peripheral portions of the canal or when caused by a degenerative spondylolisthesis.
- *Combined stenosis.* Any combination of congenital or developmental stenosis combined with prolapse of the intervertebral disc.
- *Spondylolytic or spondylolisthetic stenosis.*
- *Postoperative stenosis.* Caused by previous laminectomy or fusion. Occasionally, chemonucleolysis may be implicated.
- *Post-traumatic.*
- *Miscellaneous.* Particularly Paget's disease and fluorosis.

Management

1. Plain radiography. The principal function of plain radiographs is to exclude other disease. They are seldom diagnostic. More specialized investigations are indicated for patients who may require surgery.

2. CT is currently the best method available for demonstrating spinal stenosis. It can distinguish clearly between each of the structures contributing to the stenosis whether central, lateral or in the nerve root canal. It is particularly good at identifying bony margins. In central stenosis, the sagittal diameter of the canal at the upper margin of the lamina should be less than 11.5 mm, the interpeduncular distance less than 16mm and the cross-sectional area less than 145 mm^2. These measurements are by no means infallible: most spinal surgeons and their radiologists place more emphasis on the relationship and general appearance of structures impinging on the spinal canal.

3. MRL MRI is fast approaching CT in delineating the pathological structures contributing to spinal stenosis and is superior in its ability to image the discal component. Its disadvantage is that bone gives a poor signal. MRI may consequently miss osteophytes which are contributing to the stenosis.

4. Myelography. This is the most sensitive method of identifying compression of the cauda equina and is equal to CT in making the diagnosis of spinal stenosis, although it is not particularly helpful in defming the cause. It is still less helpful in diagnosing compression of the lateral recess and of no value whatsoever in the nerve root canal. It should be reserved for 'difficult' cases with convincing clinical features in which the diagnosis or level of compression is in question after CT and MRI.

5. Other investigations include electromyography, and localization techniques using nerve root and facet joint injections. These are seldom needed in the first instance but may be helpful in the management of the multiply operated spine.

6. Non-operative treatment. A variety of methods have been used including bed rest, physiotherapy, NSAIDs, corsets, plaster jackets and epidural injections. Although benefits have been claimed for each of these, there is no objective evidence that they are of value. This does not, however, mean that every patient with spinal stenosis requires surgery.

7. Surgical decompression. Decompression is indicated if symptoms become intolerable, if a neurological deficit is progressing or if there is evidence of sphincter disturbance. It should be limited to those areas which, after appropriate clinical and radiological examination, are considered to be causing significant compression of the neural elements. The surgical approach should expose only the affected levels. The correct level/s should be identified from sacrum or radiologically (or both). There is seldom any need to excise the midline structures. Carefully placed fenestrations will give access to the cauda equina and nerve roots. Any bone, disc, facet joint or ligament which compresses the neural elements should be removed. The facet joint may be undercut to release the nerve in the lateral recess. If the nerve root canal is severely narrowed, an adequate decompression will only be obtained at the expense of resecting the facet joint. In this case, the affected spinal segment should be stabilized at the same time by fusion.

Complications

1. General complications. Wound infection, CSF leak, haematoma formation and damage to the retroperitoneal or abdominal viscera. These may occur after any posterior spinal surgery but their incidence should be low (<1%).

2. Inadequate decompression. The common causes are: inadequate planning; wrong level/s decompressed; inadequate decompression of the lateral recesses or root canals; insufficient number of levels decompressed.

3. Epidural scarring. This may be localized to scarring around a single nerve root or may be generalized.

4. Postoperative stenosis may be the result of inadequate decompression, the formation of a 'postlaminectomy membrane' or the development of further stenosis with the passage of time. Scar formation can be limited by the use of a minimally invasive approach, meticulous haemostasis and free fat grafts.

5. Postoperative instability and spondylolisthesis is unusual unless the decompression has removed complete facet joints or partes interarticulares. It is more likely to occur in a young patient after decompression at several levels for degenerative disease when the operation has included disc excision. The presence of anterior osteophytosis and extreme disc space narrowing may be protective. Destabilized levels can be treated by floating lateral mass fusions at the time of decompression.

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Related topics of interest

Low back pain (p. 181)

Spondylolysis and spondylolisthesis (p. 317)

SPINAL TUMOURS

Primary tumours of the spine are rare. They may be benign or malignant. The majority are metastases from breast, thyroid, bronchus, prostate, kidney, bowel or skin. Lymphomas and myeloma may also arise in the spine.

Clinical problems

1. Benign. The commonest benign conditions to affect the spine are haemangioma, aneurysmal bone cyst, giant cell tumour, eosinophilic granuloma and osteochondroma. Of particular interest are *osteoid osteoma* and *osteoblastoma*. These usually occur in adolescents who present with stiffness in the spine and a painful scoliosis. Most are in the pedicles and may be removed through a midline incision with resection of the transverse process to gain access to the pedicle. Fusion is unnecessary.

2. Malignant. Malignant myeloma and chordoma are the commonest primary malignancies of the cervical and lumbar spine. *Malignant myeloma* principally affects men over the age of 50. It shows marked predilection for the spine where it can cause severe pain and muscle spasm. Radiographs show one or more lytic lesions with little bony reaction. Scintigrams are frequently 'cold'. Laboratory investigations demonstrate an anaemia, increased plasma viscosity, hypercalcaemia and a paraprotein band on electrophoresis. The urine tests positive for Bence-Jones protein. The diagnosis may be confirmed by bone marrow biopsy if the disease is widespread. The treatment of choice is chemotherapy (often melphalan/prednisolone) and surgery is reserved either for direct biopsy of a plasmacytoma or for spinal stabilization. *Chordoma* is a rare malignant tumour of notochordal remnants which occurs either in the cervical or sacral spine. In the sacrum it produces a large soft tissue mass which usually enlarges anteriorly into the presacral space. Symptoms other than mild back pain may not occur until the tumour is of considerable size: the diagnosis may therefore be delayed. Like malignant myeloma, the scintigram may be 'cold'. Treatment is by wide resection in the hope of achieving a cure. Extensive procedures including radical sacrectomy with ligation and division of the cauda equina have been described with varying degrees of success. High resections are

always accompanied by sphincter disturbance for which provision has to be made.

3. Metastatic. See Bony metastases (p. 74).

Management

The management of any spinal tumour is directed at the diagnosis and treatment of the primary spinal lesion, prevention of neurological compromise, relief of pain, stabilization of the spine and correction of any deformity. The diagnosis may need to be confirmed by *biopsy*. This may be carried out percutaneously in the lumbar spine but should be open in the thoracic spine and neck. Many metastatic tumours can be palliated with radiotherapy and hormonal manipulation.

Surgery is indicated for defmitive treatment of a curable primary tumour, slowly progressive cord compression by tumour or vertebral collapse and intractable pain caused by instability or deformity. The way in which the tumour excision is carried out depends on the histological diagnosis and the extent of spread of the tumour. Most occur in the vertebral body and demand an anterior approach. *Stabilization* of the spine can be achieved using bone, PMMA cement and anterior and posterior implants. Anterior cages and jacks are particularly useful in this type of surgery and can be used to bridge at least two vertebral levels. Circumferential stabilization and fusion may be needed in cases of extensive tumour where all three columns are compromised. Long posterior stabilization and fusion using Harrington/Luque instrumentation can be a useful technique for patients with extensive metastatic disease.

1. Benign tumours. These are widely resected and the remaining defect in the spine is stabilized and grafted.

2. Primary malignant tumours. The principles of management of primary malignant tumours of the spinal column are the same as those described elsewhere for other musculoskeletal sarcomas. Wide local excision with adjunctive therapy should be followed by reconstruction of the excised spinal segments. This may present formidable problems because of the proximity of the neural elements and the difficulty in stabilizing long segments of the spine. *En bloc* excision of the tumour may be undertaken in one or two stages depending on the exact extent of the tumour. Radical two-stage resections excising the anterior vertebral body and pedicles, followed by posterior excision of the remaining parts of pedicle, lamina and spinous process are described but require considerable time and expertise to carry out.

3. Metastases. See Bony metastases (p. 74).

Further reading

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Related topics of interest

Bone tumours—benign (p. 63)
Bone tumours—malignant (p. 68)
Bony metastases (p. 74)
Low back pain (p. 181)

SPONDYLOARTHROPATHIES

This is a group of diseases characterized by an inflammatory arthritis with a negative test for IgM rheumatoid factor. They have common features which include sacroiliitis and/or spondylitis, asymmetrical oligo-arthritis, enthesitis and anterior uveitis. There is often a family association and a high prevalence of HLA-B27 antigen. The origin of these disorders appears to be an abnormal response to infection in a genetically predisposed person. The initiating organism has been identified in some.

Clinical problems

1. Ankylosing spondylitis (AS). This is a chronic inflammatory condition with a predilection for the sacroiliac joints and the spine which is characterized by progressive stiffening and fusion of the axial skeleton. It has a peak onset in the second and third decades and a male preponderance of 4:1. Ninety percent carry the histocompatibility antigen HLA-B27. First degree relatives show an increased incidence of psoriatic arthritis, inflammatory bowel disease and Reiter's syndrome. The arthropathy differs from rheumatoid arthritis in that it is asymmetrical and affects large joints more than small joints.

Its onset is insidious with episodes of low back pain and stiffness. Symptoms are worse in the early morning and after rest. Plantar fasciitis, achilles tendonitis, and chest pain aggravated by breathing due to involvement of costo-vertebral joints may be presenting features. During the course of the disease, 25% of patients have an attack of acute anterior uveitis. Early signs include a fixed lumbar lordosis and sacroiliac pain. With disease progression the spine becomes increasingly stiff and chest expansion decreases. Fixed dorsal and cervical kyphosis may occur and can be incapacitating when associated with hip disease (see Postural correction in ankylosing spondylitis, p. 233).

Extra-articular manifestations include iritis, aortic regurgitation, osteoporosis and pulmonary fibrosis.

The key investigations are confined to blood tests and radiology. The ESR is usually raised and the *rheumatoid factor* is negative. *Sacroiliac radiographs* show evidence of marginal sclerosis progressing to fusion. The *lumbar spine radiographs* show squaring of vertebrae due to ossification of the anterior

longitudinal ligament. There is syndesmophyte formation, erosion and sclerosis at the anterior corners of vertebrae and there are facet joint changes. Progressive ossification results in the typical 'bamboo' spine.

2. Reiter's disease. This is a triad of non-specific urethritis, conjunctivitis and arthritis following an episode of bacterial dysentery or chlamydia urethritis. When arthritis alone follows a urethritis or enteric infection the condition is called 'reactive arthritis'. A variety of organisms are associated with this condition and include *Salmonella, Shigella, Campylobacter, Yersinia* and *Chlamydia*.

The onset of symptoms is acute within 3 weeks of exposure to infection. There is an inflammatory oligo-arthritis affecting large or small joints of the lower limbs. There may be a systemic illness. Occasionally the onset is more insidious, presenting with a monoarthritis, commonly of the knee. The signs and symptoms of urethritis or conjunctivitis may have been trivial and ignored. Heel pain, either in the form of an achilles tendinitis or as plantar fascitis, may be a presenting feature. There may be dermatological evidence of a rash or nail dystrophy. Eye symptoms may be of mild bilateral conjunctivitis or, in 10%, acute iritis which may progress to glaucoma or blindness.

The arthritis is usually self-limiting, with spontaneous remission within 3 months. There is a recurrence rate of approximately 15% which may include low back pain and stiffness from sacroiliitis or the development of spondylitis. Twenty years after onset, 10% show evidence of disease including spondylitis, a chronic erosive arthritis or ocular manifestations.

The ESR is elevated in acute and chronic disease. A *full blood count* shows a polymorphonuclear leukocytosis or an anaemia in chronic disease. Serology with *rheumatoid factor* and *anti-nuclear factor* is negative. *Plain radiographs* show evidence of an erosive arthritis in affected joints. There may be a periostitis especially in the metatarsals. There may be large calcaneal spurs and the sacroiliac joints may resemble those in ankylosing spondylitis.

3. Psoriatic arthritis. This occurs in approximately 10% of patients with psoriasis. Onset is usually between 20 and 40 years of age. Arthritis affecting the distal inter-phalangeal joints is characteristic and is associated with the 'pepper pot' nail changes. However, more commonly, there is an asymmetrical or symmetrical seronegative arthritis which follows the rheumatoid pattern. Sacroiliitis and spondylitis resemble those seen in ankylosing spondylitis.

The *ESR* is mildly raised. *Rheumatoid factor* and *anti-nuclear factors* are negative. *Radiographs* confirm terminal IP joint involvement: axial skeleton changes resemble AS.

4. Inflammatory bowel disease and arthritis. Ulcerative colitis and Crohn's disease are associated with two distinct patterns of inflammatory arthritis. *Enteropathic synovitis* is an acute migratory non-erosive arthritis, most commonly affecting the knees, ankles, wrists and inter-phalangeal joints of the hands and feet. The arthritis usually accompanies exacerbations of bowel disease and resolves after bowel resection.

A disease pattern similar to *ankylosing spondylitis* may also accornpany bowel disease. It may precede the bowel symptoms and can continue independently irrespective of bowel remission or surgery.

5. Behget's syndrome is characterized by large joint arthritis, intestinal ulceration and encephalitis. Arthritis is often mono-articular and non-erosive and rarely requires surgery.

6. Whipple's disease is a rare disease with a migratory arthritis in association with gastrointestinal symptoms, skin pigmentation and general systemic upset. Diagnosis is made following a small intestinal biopsy to demonstrate the presence of p-aminosalicylic acid-positive macrophages.

Management

1. Ankylosing spondylitis. Physiotherapy aims to relieve pain and stiffness and to maintain maximum movement while avoiding deformity. It requires a regular exercise regime and training in posture. *NSAIDs* are used for symptomatic relief but do not alter the course of disease. *Corticosteroid injections* may help, particularly with plantar fascitis. *Systemic steroids* may be required for treatment of acute iritis. *Total hip replacement* may be required for pain relief or stiffness and *spinal extension osteotomy* to correct profound kyphosis (see Postural correction in ankylosing spondylitis, p. 233).

2. Reiter's disease. Treatment consists of rest or splintage in acute arthritis, NSAIDs for symptomatic control, joint aspiration and intra-articular steroids. Systemic corticosteroids may be required for acute iritis. The appropriate anti-microbial treatment should be given for the precipitating infection.

3. Psoriatic arthritis. NSAIDs are given for symptomatic relief. *Gold injections* may control the arthritis. *Immuno- suppressive drugs* may be required to control severe cutaneous disease and are also effective against the arthritis. Total joint arthroplasty is rarely indicated. The presence of local skin lesions does not affect healing. It is not clear whether the incidence of postoperative infection is increased.

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Related topics of interest

Kyphosis (p. 170)

Postural correction in ankylosing spondylitis (p. 233)

Young adult hip problems (p. 340)

SPONDYLOLYSIS AND SPONDYLOLISTHESIS

Spondylolysis is a condition in which there is a unilateral or bilateral defect in the pars interarticularis of one or more vertebrae. The condition may be asymptomatic and only recognized incidentally on radiographs of the lumbar spine.

Spondylolisthesis is the slipping forward of all or part of one vertebra on another. The classification of Wiltse *et al.* (1976) is used. They divided spondylolisthesis into five types to which a sixth has been added more recently.

Clinical problems

1. Type I (dysplastic). Congenital abnormality of the upper sacrum or the arch of L5 allowing slipping (olisthesis) at L5/S1. There are two subtypes: type IA, which presents early in life with a severe slip, axially orientated facet joints and wide spina bifida of L5/S1. Hamstring spasm is common. The neurological prognosis is better if the spine is widely bifid. In type IB, the facets are sagitally orientated and the posterior elements intact. A severe slip is less common but the cauda equina may be more tightly compressed resulting in leg pain, hamstring spasm and an altered gait pattern.

2. Type II (isthmic). The lesion is in the pars interarticularis. This may be either: (a) a stress fracture of the pars, or (b) elongation of the intact pars after healing of a stress fracture. Most cases start after the age of 5 years and may present in athletic individuals until early adulthood. High-grade slips occur most often early in the second decade and are four times more common in girls. Leg and back pain are the commonest symptoms. Further slipping in adulthood is uncommon.

3. Type III (degenerative). Due to longstanding instability of a motion segment. Occurs after the age of 40, with an increasing incidence into old age. Women are affected more than men. The symptoms are either those of unilateral nerve root compression causing sciatica, or of spinal claudication due to central compression of the cauda equina. Neurological sequelae are uncommon. The slip is usually self-limiting and seldom more than 30%.

4. Type IV (traumatic). Due to acute fractures in areas of the bony hook other than the pars.

5. Type V (pathological). Due to generalized or localized bone disease.

6. Type VI (postsurgical). Due to partial or complete loss of posterior bony and/or discogenic support secondary to surgery or to stress fractures of the inferior articular facets also secondary to surgery.

The congenital, isthmic and degenerative types occur most commonly. The degree of slip may be expressed as the percentage by which the rostral vertebra has slipped on the caudal (Taillard method) or may be graded from I to IV in 25% increments, grade IV being a 75–100% slip (Meyerding method).

Management

1. Children and adolescents. The younger the child the greater the risk of slip. The risk is four times higher for girls. Poor prognostic features include spina bifida, rounding of the superior surface of Sl and wedging of the body of L5. The indications for spinal fusion are severe symptoms lasting at least 6 months in the adolescent; lesser symptoms in a young child (under 10 years) particularly if the slip is progressive; intractable hamstring spasm and progressive slip. Posterolateral fusion *in situ* without decompression is recommended for most cases. Sciatica and hamstring spasm usually resolve with time. Very occasionally, late posterior decompression may be undertaken after fusion.

2. Adults. Conservative treatment is as for other forms of low back pain. The indication for surgery in isthmic spondylolisthesis is unremitting low back pain. The slip in the adult seldom increases significantly. Only the affected level should be fused unless this is L5/S1 and the L4/5 disc has been shown to be abnormal by MRI or provocative discography. In this case, most surgeons would extend the fusion to involve the upper segment. Concomitant posterior decompression is often carried out but is probably only indicated for severe leg pain.

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Related topics of interest

Low back pain (p. 181)
Spinal fusion (p. 299)
Spinal stenosis (p. 306)

THORACIC BACK PAIN

Pain localized to the back of the thorax may be caused by primary pathology arising outside the thoracic spine. Specific causes of thoracic back pain are often age-related. Examination of the thoracic spine is frequently unrewarding and the diagnosis is made on a detailed history and investigations. Advances in diagnostic imaging have made localization of thoracic spinal disorders more specific and their management more effective.

Clinical problems

Referred pain

1. Cervical spine. Pathology at the C5 or C6 levels may result in pain radiating to the mid- or inter-scapula area. C7 pain may radiate to the mid-thorax or to the angle of the scapula.

2. Cardiovascular. Pain may be referred from the myocardium to the thoracic spine, e.g. myocardial infarction or angina, or from an aortic aneurysm, or an inflammatory aortitis.

3. Pulmonary/diaphragmatic. Conditions such as infiltration by malignant disease or irritation from empyema or pulmonary infection may cause thoracic spinal pain.

4. Peripheral nerve pathology. A neuritis, herpes zoster or neuralgic amyotrophy related to the spinal accessory, the long thoracic, suprascapular or intercostal nerves may cause pain.

5. Ribs. Fractures occur commonly. Ribs may be infected either primarily, as a rare site of osteomyelitis, or secondarily from an adjacent empyema. Secondary deposits from malignant disease are more common than primary tumours and may present with thoracic back pain. Myeloma and eosinophilic granuloma are the commonest primary tumours affecting the ribs.

6. Muscular. Intercostal muscle strain or bruising. Inflammation at muscle insertions of pectoralis major, latissimus dorsi or the rhomboids may cause symptoms.

Pain arising in the vertebral column

1. Thoracic facet joint pain. Injury or degenerative change of the thoracic facet joints produces pain in the posterior thorax. There may be local tendemess at the affected levels.

2. Thoracic vertebra. Scheuermann's disease will on occasions present as back pain (see Kyphosis, p. 170). Pathological fractures often occur with no history of trauma, as a result of generalized osteoporosis. Primary or secondary tumours may cause symptoms with or without secondary fracture. Bone infection with pyogenic organisms, tuberculosis or aspergillosis may present as back pain. Paget's disease may involve the thoracic spine. Ankylosing spondylitis or Forestier's disease usually produce pain posteriorly and centrally in the thoracic spine, but with progressive involvement of the anterior longitudinal ligament the pain may become epigastric or sternal.

3. Thoracic disc disease. Disc degeneration evidenced by radiographic disc space narrowing is a common finding. Thoracic inter-vertebral disc rupture and prolapse is rare. It constitutes approximately 0.5% of inter-vertebral disc prolapse. There may be a history of trauma, often a road traffic accident. Many have an insidious onset with no obvious precipitating factor. The average age of presentation is 40–50 years old with average duration of symptoms at presentation of 2 years. The commonest site is from TIO to T12.

The symptoms are either of girdle pain encircling the thorax or piercing central pain radiating backwards from the sternum. Pain is aggravated by active elevation of the arms, by the heel strike when walking, coughing, sneezing or lying supine. A complaint may be no more than of non-specific lower limb weakness, paraesthesia or numbness.

On examination there is little muscular spasm, but usually tenderness over the spinous process above and below the affected disc. Sensory changes may be of hyperaesthesia only. In 50% of cases this is bilateral. The anterior primary rami supply the skin in front of the posterior axillary line and the posterior primary rami from this line to the middle of the back. Each ramus may be affected individually or both may be involved. Long tract signs due to cord compression may be present, e.g. spasticity, hyperreflexia, clonus and a positive Babinski reflex.

The differential diagnosis includes posterior primary ramus entrapment syndrome which produces pain of acute spontaneous onset, is usually unilateral and causes localized tenderness. Sensory change may occur in the distribution of the posterior primary ramus. There is no tenderness over the spinous process. Treatment is by local injection of anaesthetic and steroid. Costo-vertebral syndrome due to a severely deformed costo-vertebral joint may rarely be identified as a cause of symptoms.

Management

1. Investigation. Non-musculoskeletal causes should become evident from the history, general examination, or special investigations, e.g. ECG. The investigation of thoracic back pain of musculoskeletal origin requires standard *PA and lateral radiographs* of the thoracic spine. This may identify abnormal bone quality, disc space narrowing, abnormal calcification and soft tissue swelling. *Myelography* was once necessary to identify a thoracic disc lesion and intra- and extra-dural tumours: CT will localize bone infections, tumours and bony stenosis. However, *MRI* is now the investigation of choice for identifying and localizing soft tissue elements of thoracic spine pathology, including spinal cord pathology, thoracic disc disease or infection.

2. Treatment. The specific treatment of non-musculoskeletal causes of back pain is not discussed. Spinal tumours and spinal infection are discussed elsewhere.

More accurate location of the pathology using CT and MRI scans has improved the outcome of surgery for thoracic disc disease. The results from *posterior laminectomy and discectomy* are disappointing. It provides poor access to both central and lateral disc prolapses. *Lateral costo-transversectomy* is the best approach for lateral thoracic disc herniations and involves excision of a section of rib at the level of the involved disc through which the lateral extent of the disc may be approached and excised. *Anterior transthoracic discectomy* is the preferred approach for central disc herniations. It allows superior access for complete decompression and provides good access for vertebral fusion if required.

Complications

Thoracic disc disease occasionally presents with sudden onset of Brown-Sequard syndrome (unilateral paralysis with contralateral loss of pain sensation) or complete paraplegia.

Further reading

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Related topics of interest

Acute cervical disc disease (p. 1)
Acute lumbar disc disease (p. 5)
Bone tumours—benign (p. 63)
Bone tumours—malignant (p. 68)
Bony metastases (p. 74)
Kyphosis (p. 170)

Musculoskeletal imaging (p. 190)
Spinal infection (p. 302)
Spinal tumours (p. 310)
Spondyloarthropathies (p. 313)

THORACIC OUTLET SYNDROME AND CERVICAL RIB

Thoracic outlet syndrome (TOS) is a collection of several conditions in which the neurovascular structures in the root of the neck are compressed, thereby causing a variety of symptoms and signs in the shoulder and upper limb. It is four times more common in women than men and affects those in the second to fifth decades of life. Its aetiology is unclear but is probably related to the tortuous path taken by the first thoracic nerve root and the subclavian artery and vein as they curve up over the first rib before descending into the arm. Any condition which causes relative hypotonia of the muscles which elevate the pectoral girdle will cause the upper limb to droop in relation to the thorax and increase compression of the neurovascular bundle over the first rib. The greater angle of inclination of the first rib in men is probably protective. Compression of the neurovascular elements may also be caused by a cervical rib or a fibrous band arising from it, congenital anomalies of the shoulder girdle, post-fixing of the brachial plexus, hypertrophy or spasm of scalenus anterior and prolonged carrying of a heavy rucksack, although this more often causes compression of the upper trunk. Cervical ribs which are 5 cm or more long will impinge on the brachial plexus, usually lifting it upwards and forwards.

Clinical problems

The diagnosis of TOS is a clinical one: investigations are only useful to exclude conditions which may be confused with it.

The clinical features of TOS may be neurological, vascular or a combination of the two, depending on which structures are compressed. Patients describe continuous or intermittent throbbing or aching in the arm, particularly on the ulnar border of the forearm radiating into the ring and little fingers. This may be accompanied by paraesthesiae and numbness. It may be exacerbated by carrying heavy loads and by sudden unprotected movements of the head and neck. The pain may also radiate to the chest or scapula. Typically, overhead activities become impaired by ischaemic aching in the forearm and hand. They may describe pallor of the arm or flushing.

On examination there may be signs of vasomotor disturbance in the limb with small muscle wasting in the hand. Venous congestion may be present if the

subclavian vein is involved. There are a number of eponymous 'provocative tests' described. Adson's 'vascular test' suggests compression of the subclavian artery by scalenus anterior and only implies involvement of the brachial plexus. There are two modifications of this test which are thought to indicate compression of the subclavian artery by scalenus medius and pectoralis major, respectively. Wright's test (abduction and external rotation of the arm with the shoulder braced) is probably the most reliable test but should never be used in isolation from the others. Spurling's sign (pain produced by direct pressure over the brachial plexus) suggests irritability of the brachial plexus and is probably helpful. Roos' overhead exercise test and the 'military brace test' are positive in between 70 and 85% of those affected.

A number of these tests should be unequivocally positive in the hands of an experienced observer before the diagnosis is made with conviction.

Management

1. Investigation. Plain radiographs may show a cervical rib, or an enlarged transverse process which may indicate the presence of an anomalous fibrous band. The first rib or clavicle may be anomalous in size, shape or position. Nerve conduction studies may help exclude more distal problems particularly ulnar nerve compression at the elbow. Angiography is only of value in cases of subclavian aneurysm. MRI has not been of proven assistance.

2. Non-operative treatment. All patients should be treated by conservative means for at least 3 months. This should consist of exercises to strengthen the elevator muscles of the pectoral girdle and instruction in correct posture. Most patients will show some improvement. Only those with persistent severe symptoms should be considered for surgery.

3. Operative treatment. Surgical treatment is indicated in patients who have failed to respond sufficiently to conservative treatment, those with a significant neurological deficit and the rare cases of impending arterial occlusion.

Traditionally, patients were offered *anterior scalenotomy* or scalenectomy for TOS but the results were poor and there was a high rate of symptomatic recurrence.

The operation of choice for TOS *isfirst rib excision.* This may be carried out through a number of surgical approaches. The safest is the *transaxillary approach (of Roos).* During this procedure, the arm is held flexed at the shoulder thereby elevating the neurovascular structures from the upper surface of the first rib. This may then be approached through the axilla, taking care not to damage the intercostobrachial nerve, and the rib can be resected subperiosteally.

The first rib may also be resected though a *supraclavicular approach,* although access to the posterior part is more awkward. This approach is useful if there is doubt about the presence of a fibrous band passing through the brachial plexus, but must be carried out by a surgeon who is completely familiar with the

complex anatomy of the region. It is also the approach of choice for excision of a cervical rib.

First rib excision gives excellent or good results in approximately 75% of cases. This figure is not likely to improve until the diagnosis can be made more accurately.

Complications

The complications are those of surgery, once the correct diagnosis has been made.

1. Nerve injury. The intercostobrachial and long thoracic nerves are at risk of traction or laceration if the axillary approach is used. The lower trunk of the brachial plexus may be damaged particularly at the moment when the rib is divided posteriorly. The brachial plexus as a whole may be injured by excessive traction on the arm. The supraclavicular approach carries the risk of damage to the three supraclavicular nerves and to any part of the brachial plexus itself.

2. Vascular injury to the subclavian artery and vein have both been reported.

3. Causalgia occurs rarely, but with devastating results.

4. Pneumothorax is common, occurring in up to one-third of cases.

5. Recurrent or residual symptoms may be due to an inaccurate diagnosis, scarring at the site of surgery, compression of the neurovascular structures against the second rib, regeneration of the rib or an inadequate primary excision.

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Related topics of interest

Paralysed hand (p. 214)
Peripheral nerve entrapment (p. 225)
Shoulder pain (p. 281)

THROMBOEMBOLIC DISEASE

Deep vein thrombosis (DVT) occurs in 80% of patients undergoing major orthopaedic surgery (e.g. hip fracture or hip and knee replacement), and pulmonary embolism (PE) in 10%, if prophylaxis against thromboembolism is not undertaken. DVT is the commonest cause of emergency re-admission to hospital after total hip replacement. Most fatal emboli arise from deep vein thrombosis in the lower limb. The most effective way to prevent thromboembolic disease is to use routine prophylaxis for patients at risk. The risk of venous thromboembolism increases exponentially with age and becomes appreciable at 40 years with major illness, trauma or surgery. Other risk factors include obesity, immobility, previous DVT or PE, and malignancy. Patients with previous thromboembolic episodes have a postoperative risk of DVT greater than 50%.

During surgery, mechanical injury to vessel walls, venostasis, and hypercoagulability states (Virchow's triad) all contribute to DVT formation. There are changes both in coagulability and fibrinolysis after orthopaedic surgery.

Prophylaxis

Simple precautions such as discontinuing oral contraception, encouraging early postoperative mobilization, and using spinal or epidural anaesthesia reduce the risk of developing a DVT. Other preventative measures may be classified as *physical* or *pharmacological*.

Physical methods

1. Compression stockings. Graduated compression stockings should be put on immediately before surgery and worn until the patient is fully mobile once again. These decrease venous stasis in the limbs and may reduce the risk of developing a DVT.

2. Pneumatic compression boots and calf muscle stimulation have been shown to reduce the incidence of DVT in both orthopaedic and general surgery.

3. Foot pumps, which apply intermittent positive pressure to the sole of the foot, reduce venostasis and have been shown to reduce the risk of thromboembolic disease after total hip replacement and hip fracture.

Mechanical methods of prophylaxis do not increase the risk of bleeding, and may be used in circumstances where pharmacological methods are contraindicated.

Pharmacological methods

1. Heparin acts by potentiating the effect of endogenous coagulation inhibitor anti-thrombin III and inhibits platelet function. *Low dose subcutaneous heparin* (5000 international units (IU) 8–12 hourly) reduces the incidence of DVT and PE in high risk groups by approximately two- thirds. Treatment should be started 12 hours before surgery and continued for at least 12 days or until normal mobility is resumed. *Adjusted low dose subcutaneous heparin* administered 8 hourly and adjusted to maintain the activated partial thromboplastin time (APTT) within the desired range is more effective but more complicated to administer than low dose subcutaneous heparin. *Low molecular weight heparins (LMWH)* are derivatives of standard heparin and after subcutaneous injection have a greater bioavailability. The longer biological half-life allows a once daily injection which is as effective in preventing DVT as heparin. The timing of the preoperative dose is important. Low molecular weight heparins are potentiated by NSAIDs.

The potential complications of giving prophylactic heparin are bruising at injection sites and increased risk of wound haematomas. Contraindications include bleeding disorders, thrombocytopenia, haemophilia, liver or renal failure, peptic ulceration, aneurysm and cerebrovascular accident. Heparin should not be given within 6 hours of planned spinal anaesthesia.

2. Oral anticoagulants. Warfarin reduces the risk of developing a DVT after orthopaedic surgery. Starting with a low dose before surgery or starting after surgery will reduce the risk of bleeding. The recommended postoperative international normalized ratio (INR) is 2.0–3.0 for hip surgery. As daily monitoring of the INR is necessary, warfarin is usually reserved for high risk patients.

The contraindications to its usage are the same as those for heparin. In addition, it should be avoided in pregnancy as it may be teratogenic.

3. Dextrans. Dextran 70 reduces the risk of DVT after surgery for fracture of the femoral neck. It has to be given by intravenous infusion and increases the risk of intraoperative and postoperative bleeding and of anaphylaxis.

Diagnosis

DVTs may be confirmed using venography, duplex ultrasonography or plethysmography.

PE can be confirmed from arterial blood gases, chest X-ray, electrocardiography and ventilation-perfusion (V/Q) lung scan.

Treatment

Treatment for confirmed PE and DVT is full anticoagulation provided that there is no absolute contraindication to it: if there is, one or more of the mechanical forms of treatment may be used. Supplementary oxygen is required for the hypoxia associated with PE.

Heparin is usually given initially because it is effective immediately: warfarin is started at the same time but takes 2–4 days to reach therapeutic levels. Heparin is administered by continuous infusion (e.g. 1400 lU/h) after a loading dose of 5000 IU IV. Its activity is measured by the AFTT at a level 1.5–2.5 times the control value and may be monitored daily. It is stopped once oral anticoagulation with warfarin has been shown to be effective (INR between 2.0 and 3.0, or between 3.0 and 4.0 in patients with heart valves or recurrent thromboembolic disease).

Warfarin is a competitive antagonist of vitamin K, can be taken orally and is a more convenient long-term anticoagulant. The plasma half-life is 35 hours and it takes 1 week to reach a steady state. Warfarin should be continued for 3 months after the first venous thromboembolic episode.

The main complication is haemorrhage. In severe cases the effects of heparin may be reversed using protamine sulphate (1 mg for every 100 IU infused during the previous hour). For minor bleeding with warfarin, 1 mg of intravenous vitamin K will reduce the INR. For life-threatening bleeding, 5 mg of vitamin K should be given intravenously together with clotting factors.

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TOTAL KNEE ARTHROPLASTY

Total knee replacement (TKR) is indicated for patients who are severely incapacitated by pain from tricompartmental arthritis of the knee. Alternatives such as arthroscopic debridement, osteotomy and arthrodesis should be considered in the younger patient. Knee replacement is contraindicated in cases of extensor mechanism failure, active sepsis, previous arthrodesis and Charcot arthropathy.

Clinical problems

1. Unicompartmental disease. Most patients present with disease of the medial compartment and some varus deformity. Those with valgus knees may have isolated disease of the lateral compartment. In these cases, a unicompartmental replacement may be undertaken provided that the anterior cruciate and collateral ligaments are intact and that the knee flexes to at least 110°. At operation, it is important to ensure that the tibial cut is perpendicular to the long axis of the tibia. The affected compartment may be resurfaced using a standard metal-backed polyethylene tibial component and metal femoral component, or with a metal tibial tray and femoral component which articulate with an intercalated polyethylene meniscus.

2. Varus knee. Soft tissue balance in the varus knee is achieved by resection of medial osteophytes and elevation of a medial soft tissue sleeve from the upper tibia which includes the superficial medial collateral ligament. In severe cases, the semimembranosus tendon and posteromedial capsule may also be released.

3. Valgus knee. If the valgus deformity is severe, a lateral parapatellar approach is used. The popliteus tendon is released from the femur, especially in cases of lateral subluxation. The iliotibial band and posterolateral capsule are elevated from the tibia. The lateral collateral ligament and the lateral head of gastrocnemius may be released from the femur. In very severe cases, the biceps tendon may be lengthened: great care must be taken to protect the common peroneal nerve.

4. Fixed flexion deformity. Any anterior osteophytes are removed. The posterior capsule and posterior cruciate ligament are elevated from the posterior

surface of the tibia. The two heads of gastrocnemius are released and a posterior capsulotomy is carried out as necessary.

5. Limited flexion. A quadriceps V-Y plasty or patellar tendon Z-plasty may be used.

6. Patellar maltracking. A lateral release should be undertaken if the patella subluxates laterally when the knee is flexed. The superior lateral geniculate vessels must be preserved. The femoral component should be inserted in slight external rotation to facilitate tracking.

7. Tight flexion/ extension gaps. If the flexion gap is tight, more bone may be resected from the back of the femoral condyles. Alternatively, the tibial cut may be angled up to 5° backwards. If a cruciate-sparing prosthesis was planned, the PCL may be divided and a posterior-stabilized cruciate-sacrificing prosthesis implanted. If the extension gap is tight, more of the distal femur should be resected. If both flexion and extension gaps are tight, more tibia has to be removed.

8. Cruciate ligament. Unconstrained meniscal knees preserve both cruciate ligaments but are technically demanding. Semiconstrained knees sacrifice the ACL and depend on surface congruity for stability. The PCL may be sacrificed and replaced by a cam mechanism to allow an increased range of flexion and restoration of stability at the expense of some functional difficulty descending stairs.

Complications

1. Aseptic loosening rates. Survivorship rates for total condylar knees of 90% at 15 years and 97% at 10 years (for posterior-stabilized knees) have been reported. Lucent lines on radiographs are usually seen under the medial tibial plateau. Alignment and support for the tibial component with soft tissue balance are important.

2. Wear. Tibial tray thickness should be at least 8 mm.

3. Anterior knee pain. The incidence of patello-femoral joint pain after total knee replacement is reported to be between 16 and 50% if the patella has not been resurfaced.

4. Patellar fracture and patellar loosening. Fracture of the patella occurs in 1% of replaced knees. Sacrifice of lateral geniculate vessels, attempting replacement in a thin patella (<2 cm depth) or excessive resection are thought to be predisposing factors. Loosening of the patellar component occurs in 0.6%. Metal-backed patellar components have a higher loosening rate. The reoperation rate for patients who have undergone patellar replacement is 2.5% compared to 2. 4% in the non-resurfaced group.

5. Skin slough. This may occur if there are multiple scars around the knee, if the skin is of poor quality or if it has been much undermined at operation.

6. Infection. The average infection rate for osteoarthritic knees is 0.5% compared to 3% for rheumatoid arthritis. Constrained prostheses appear to become infected more easily.

7. Common peroneal nerve palsy. Occurs in 1–3% of cases and appears more commonly after correction of severe valgus or fixed flexion deformities.

8. Supracondylar fractures. Occur through osteoporotic bone and are best treated by ORIF using a dynamic condylar screw or blade plate.

9. Revision TKR and bone defects. Restoration of the joint line is imperative. The fibular head and adductor tubercles are points of reference. Small tibial bone defects may be treated by adequate tibial resection, but if they are more than 10 mm deep and excessive resection of good bone is required, alternative measures should be used. Small contained defects can be treated by using either morcellized graft or cement. Unconstrained defects or large contained defects may be replaced with block allografts or tibial wedges: in these cases the tibial component should be stemmed. Femoral defects may be treated in similar fashion. Severe bone loss may be treated using custom-made implants. Any patient with medial collateral ligament instability requires a constrained TKR.

Further reading

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Related topics of interest

Arthritis of the knee (p. 39) Osteotomies around the knee (p. 211)

TOURNIQUETS

A bloodless field in certain limb operations aids recognition of tissues, reduces operating time and reduces operative blood loss. The term 'tourniquet' was first introduced by Petit in 1718 for his device which had a screw mechanism to tighten a constricting band around the upper part of a limb destined for amputation.

The width of the cuff is critical. In the upper limb, the cuff should be 20% wider than the diameter of the upper arm: in the thigh it should be 40% wider (i.e. 20 cm). In the upper limb, the inflation pressure should be 50 mmHg higher than the systolic blood pressure: in the lower limb, it should be twice as high as the systolic blood pressure.

Types of tourniquet

There are two main types of tourniquet, *non-pneumatic* and *pneumatic. Pneumatic tourniquets* may be *non-automatic* or *automatic.*

1. Non-pneumatic. Before the common availability of pneumatic tourniquets, an Esmarch bandage was used as an exsanguinator and then retained as a tourniquet. The only time a non-pneumatic tourniquet is appropriate in contemporary surgery is for short operations on a finger or toe when a rubber catheter may be tightened around the base of the digit and held by an artery forceps.

2. Non-automatic pneumatic tourniquets consist of a cuff with a hand-operated pump and pressure gauge. This is a closed system with no automatic compensation for leaks in the system.

3. Automatic pneumatic tourniquets use a continuous supply of pressurized gas either from a bottle (air or nitrogen) or from a compressed air line in theatre. With an automatic system, cuff inflation is rapid and well maintained.

Tourniquet usage

1. Contraindications. Peripheral arterial disease particularly with large vessel involvement and atheroma. *Severe crushing injuries* where the distal circulation is critical. *Sickle cell disease:* this is an absolute contraindication in full-blown

sickle cell disease. They are best avoided in sickle trait, but if they must be used the limb must be fully exsanguinated before the cuff is inflated.

2. Exsanguination. Before the tourniquet is inflated, the limb should be exsanguinated. This may be achieved by *elevation* or *expression*. Optimum exsanguination by elevation occurs in the vertical limb elevated for 4 minutes. There is a combination of venous emptying due to gravity and reflex arteriolar constriction. Exsanguination by expression used to be achieved with an Esmarch bandage but this has now largely been replaced by the 'Rhys Davis' exsanguinator. Contraindications to its usage are venous thrombosis, malignancy or infection all of which may be spread by embolization. In frail patients, cardiac arrest can occur from circulatory overload if both lower limbs are exsanguinated at the same time.

3. Application. Pneumatic tourniquets should only be placed around the upper thigh or arm where there is sufficient muscle to distribute the pressure in the cuff evenly. This avoids creating local areas of high pressure with subsequent tissue necrosis. Pneumatic tourniquets are also used in regional anaesthesia, e.g. Bier's block. A layer of padding should be applied beneath the tourniquet cuff to protect the skin from pinching and abrasion as the cuff is inflated. During skin preparation, care must be taken to ensure that irritant or inflammable solutions do not pool beneath the cuff where they may cause burns. Patients with severe calcified atheroma may not allow vessel occlusion by normal cuff pressures. If the pressure is increased these vessels may fracture causing haemorrhage or distal embolization.

4. Duration. A colourless skin preparation is recommended so that the circulation in the distal limb can be determined more easily at the end of the procedure. The tourniquet must be used for the shortest time compatible with the satisfactory completion of the operation: for a healthy patient a maximum time of 1–2 hours. This may be excessive in the elderly or in patients suffering from diabetes or alcoholism. It is the responsibility of the surgeon to remove the tourniquet, to check the distal circulation and record the tourniquet time in the operating record.

Complications

1. Non-pneumatic tourniquet pressures. The pressure exerted on underlying tissues is unknown. The pressure under an Esmarch bandage may reach 900 mmHg in adults. Shearing forces can damage the skin particularly in the elderly or those with RA. Any incompressible structure lying deep to the tourniquet (e.g. protruding end of a fractured bone, orthopaedic implant, foreign body) may damage the skin or soft tissues with compression of the subcutaneous nerves.

2. Tourniquet paralysis syndrome is caused by cuff pressure rather than ischaemia. It consists of a flaccid motor paralysis with sensory dissociation. Pain sensation is often altered though temperature appreciation is usually preserved.

The colour and skin temperature are usually normal as are the peripheral pulses. EMG studies demonstrate nerve conduction block at the level of the tourniquet. Full recovery may take up to 3 months. Nerves in patients with diabetes, alcoholism and rheumatoid arthritis are more susceptible to pressure.

3. Post tourniquet syndrome is due to ischaemia. Following release of a tourniquet tissues swell immediately due to reactive hyperaemia and increased capillary permeability. This becomes more marked if the tourniquet time exceeds 2 hours. There is swelling in the digits and loss of normal skin creases. Joints (particularly in the hand) become stiff and the distal limb becomes pale when elevated and congested when dependent. There is subjective alteration in sensation and measurable weakness in the muscles. Irreversible mitochondrial changes are seen in muscle by 5 hours due to critical levels of acidosis. If the tourniquet time exceeds 2 hours, the tourniquet should be released for at least 20 minutes before reapplication. Problems can be minimized by elevating the limb after surgery and encouraging early active movement.

Further reading

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WRIST PAIN

Wrist pain may arise from the joint itself or from any of the structures around it. The pain is usually the result of trauma, inflammation or degeneration. Some conditions giving rise to wrist pain have already been described in other sections (Arthritis of the wrist and first carpometacarpal joint, p. 46; Carpal instability, p. 78; Repetitive strain injury, p. 242; Rheumatoid arthritis, p. 245).

Clinical problems

1. Swellings. Ganglia are the most common swellings at the wrist. On the palmar aspect, they usually lie lateral to the FCR tendon and close to the radial artery. On the dorsum, they arise either from the scapho-lunate joint or from the midcarpal joint where they should not be confused with a carpo-metacarpal boss. There is a 10% recurrence rate following excision which can be reduced by excising the neck of the ganglion. Aspiration and steroid injection cure 50%. Giant cell tumours, haemangiomas, squamous cell carcinomas and neurofibromas can also occur at the wrist.

2. Neurological Carpal tunnel syndrome is the commonest painful neurological condition arising at the wrist. *Pronator syndrome* arises from compression of the median nerve at the fibrous upper edge of flexor digitorum superficialis, at the edge of the lacertus fibrosus of biceps, or as it passes through pronator teres. The symptoms are similar to those of carpal tunnel syndrome but there is no night pain; Tinel's sign at the wrist is negative and conduction studies show delayed conduction in the upper forearm. *Ulnar tunnel syndrome* is due to compression of the terminal motor branch of the ulnar nerve in Guyon's canal where it is roofed in by the volar carpal ligament. The usual cause is a ganglion in the canal. If there is sensory loss on the dorsum of the little finger the compression is not in the canal but more proximal. Consider also anterior interosseous syndrome, cubital tunnel syndrome, thoracic outlet syndrome, and cervical root compression. Neuroma formation after surgery or iatrogenic nerve injury commonly occurs around the wrist, especially to branches of the superficial radial nerve.

3. Non-union. Non-union of the scaphoid occurs in 3.4% of conservatively treated fractures: some authorities quote higher rates. Displacement and

proximal third fractures carry a worse prognosis. Non-union of fractures of the base of the ulnar styloid may signify a serious disruption of the TFCC with radial deviation of the carpus.

4. Avascular necrosis. Avascular necrosis of the scaphoid occurs in up to 39% of proximal third fractures. Presier's disease is a vary rare idiopathic AVN of the scaphoid but even in this condition there is often a history of trauma. Kienbock's disease is avascular necrosis of the lunate. Ulnar minus variance occurs in 78% of those affected as against 23% of the normal population. Transient sclerosis is seen in 12.5% of cases of perilunate dislocation but does not progress to Kienbock's disease. The area of the bone closest to the radius has the worst blood supply. The stages are: *I,* slight sclerosis; *II,* stage I with fragmentation; *III,* stage II with collapse; *IV,* stage III with arthritis. A quarter improve spontaneously.

5. TFCC tears. Tears of the TFCC may occur in isolation or in conjunction with distal radial and ulnar fractures. Degeneration of the triangular cartilage is present in 30–50% ofcadavers.

6. DRUJ instability. Disruption of the distal radio-ulnar joint may occur in rheumatoid disease or after trauma. The ulnar head is usually displaced dorsally as the wrist supinates. Pain on demonstrating the 'piano-key' sign is characteristic.

7. Ulnar impingement. The lunate and triquetrum may abut the distal ulna in the ulna plus wrist which follows traumatic radial shortening. Cysts may be seen in the lunate. A bone scan is usually positive.

8. Pisiform pathology. The pisiform may fracture or be subject to degenerative disease.

Management

1. Diagnosis. Careful history and examination is the key to diagnosis. The exact location of tenderness often defmes the anatomical source of the problem. Restriction of movement should be noted and grip strength measured. The function of the tendons, nerves and vessels crossing the wrist must be assessed.

2. Investigations. Plain radiographs should be performed: stress views should be included if carpal instability is suspected. Tomography should reveal non-unions. Bone scans are helpful in cases where the lesion cannot be localized on radiographs and may indicate that radiographic findings are the site of active disease, e.g. cysts. MRI is useful in diagnosis of soft tissue lesions and in suspected avascular necrosis. EMG studies should be performed in cases of possible nerve entrapment.

3. Conservative. Plaster immobilization, steroid anaesthetic, physiotherapy and splintage may all be valuable.

4. Surgery. In stages I and II of Kienbock's disease, radial shortening has a success rate of 85–90%. Ulnar lengthening also is successful in 85–90% but the risk of non-union is higher (15%). STT fusion may provide relief by unloading

the ulna. Silicone replacement is no longer recommended because half fail in the long term. In stage IV disease consider wrist denervation, proximal row carpectomy and arthrodesis, in that order.

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Related topics of interest

Arthritis of the wrist and first carpometacarpal joint (p. 46)
Carpal instability (p. 78)
Repetitive strain injury (p. 242)
Rheumatoid wrist (p. 257)

YOUNG ADULT HIP PROBLEMS

Problems in the hip joint in adolescence and young adulthood follow congenital dysplasia or dislocation, Perthes' disease, slipped upper femoral epiphysis or infection (including tuberculosis). Avascular necrosis and post-traumatic arthritis may occur at any age. Patients complain of pain, stiffness and a limp. Occasionally they complain of locking and of the hip giving way.

Clinical problems

1. Acetabular dysplasia. Any insult to the growing acetabulum may result in a shallow acetabulum (centre-edge angle less than 30°). A break in Shenton's line indicates proximal femoral migration. In developmental dysplasia of the hip (DDH), the involved hemipelvis is often hypoplastic. Sclerosis may be seen on the lateral side of the acetabulum because of increased stress through this area. It is often associated with early symptoms and is a sign of further progression.

2. Femoral dysplasia. Normally, as growth proceeds, the femoral neck-shaft angle drops from 145° to 135° or less and the anteversion from 35° to 20° or less. In DDH, the femoral neck remains valgus and anteverted and the femoral canal tends to be hypoplastic.

3. Coxa magna. Overgrowth of the femoral head may occur in Perthes' disease or DDH. It is not associated with acetabular overgrowth and as a result the head becomes uncovered.

4. Abnormalities of the lateral acetabular epiphysis. The lateral acetabular epiphysis may fail to develop primarily or may have been excised at a previous operation. As a result the lateral portion of the femoral head is uncovered and secondary acetabular dysplasia may occur in which the epiphysis appears late and is unstable. There may be an associated degenerative tear of the acetabular labrum.

5. Soft tissue contractures. Soft tissue contractures occur as a result of dysplasia or deformity. They are of importance because of the risk of traction injury to the nerves and vessels if the deformities are corrected.

6. Leg length inequality. In coxa magna the femoral neck may be long and give rise to a long-leg dysplasia. Coxa vara produces a short-leg dysplasia with

restriction of abduction. Normal walking requires 10–15° of abduction to which must be added 10° for every extra centimetre of shortening.

Management

1. Clinical assessment. A history of aching on exercise suggests that the hip is beginning to decompensate. Continuous pain suggests degenerative change. Intermittent sharp pain suggests a labral tear. The onset of leg-length inequality because of fixed deformity usually presages rapid deterioration. A coxa magna with anterior subluxation may be felt as a lump below the ASIS (lump sign). Disappearance of the lump on flexion suggests a reducible subluxation. If the hip abducts on flexion, the femoral head is probably deformed. Gait, Trendelenburg test and the spine must all be assessed.

2. Imaging. Weight-bearing radiographs will often reveal joint space narrowing and evidence of dysplasia. A shoot-through lateral (Smith-Peterson view) and views of the femoral head in different degrees of rotation will show the extent of head involvement and congruency. CT, especially with 3-D reconstruction, will show the anatomy of the acetabulum and femoral canal. MRI will show the extent of head involvement in avascular necrosis.

3. Examination under anaesthetic and arthrography. This provides real-time imaging of the hip. After injection of dye, the hip should first be abducted. If the contrast pools medial to the femoral head, hinge abduction is present. The position of best congruity will usually be in flexion, adduction and external rotation.

4. Reducible subluxation. If the hip is shown to reduce in abduction, surgery should be performed to achieve this. Acetabular or femoral osteotomy should be performed depending on the site of the pathology. If more than 30° of correction is planned, surgery will be needed on both sides of the hip.

5. Irreducible subluxation. In those cases where no improvement in containment is possible the acetabulum must be augmented by a Chiari osteotomy or shelf procedure. A Chiari may be better if the femoral head shows significant lateral displacement.

6. Hinge abduction. Because containment is best in flexion and adduction, an extension abduction osteotomy will correct the deformity.

7. Unstable lateral segment. Buttressing by a shelf procedure will provide containment of the head.

8. Weak abductors. Distal transfer of the greater trochanter is indicated if the femoral neck is short and the trochanter high.

9. Arthrodesis. Severe degenerative changes in the hip may be an indication for an arthrodesis. Seventy-eight percent are satisfied at 20 years. Fifty-seven percent get back pain and knee pain occurs in 50%, with the ipsilateral knee being the most troublesome. Arthrodesis should be in 20–30° of flexion, and neutral in the other two planes erring on the side of slight external rotation. Internal fixation by cobra plate results in union in 92%.

10. Arthroplasty. In patients under the age of 50, THR fails in approximately 2% per year. The use of block autografts to reconstruct the acetabulum is associated with structural failure of the graft at 7 years. There is some evidence that a high hip centre may be better. In DDH small cemented components or customized uncemented implants may be required.

11. Arthroscopy. Arthroscopy of the hip is indicated for removal of loose bodies, excision of a labral tear and synovial biopsy.

Further reading

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Related topics of interest

Arthritis of the hip (p. 35)
Avascular necrosis of the hip (p. 53)
Hip arthroplasty (p. 150)

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