# 44 Paediatric orthopaedics

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- Legg–Calvé–Perthes disease
- METABOLIC BONE DISEASE Rickets
- NEUROMUSCULAR CONDITIONS
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- OF THE SKELETON
- Osteochondritis dissecans
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- Septic arthritis
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- Spina bifida
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- disease)

# ABNORMALITIES OF THE KNEE AND LOWER LEG

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# Anterior knee pain

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In adolescents the extensor mechanism of the knee is a common site of knee pain. Osgood–Schlatter disease is a traction apophysitis of the patellar tendon insertion. Pain, tenderness and swelling at the tibial tubercle, exacerbated by exercise, are diagnostic and radiographs are unnecessary (although, in unilateral cases, it may be important to exclude other diagnoses such as a malig - nancy). Treatment is relative rest and analgesia, and the condition resolves once the apophysis has fused. Patellofemoral pain is common and often attributed to an adolescent growth spurt: symptoms are exacerbated either by activity or by resting with the knee flex ed. Alterations to activ - ity levels and sitting position and physiotherapy to stretch and strengthen both the hamstrings and the quadriceps muscles usually result in a return to normal within a few months. Patellofemoral pain may be associated with patellar maltracking and/or instability . Physiotherapy dev elops the quadriceps muscles, particularly the vastus medialis oblique (VMO), and counteracts wasting secondary to the pain. Many operations impr ove patellar tracking and these include options for realignment of the extensor mechanism both proximally and distally . Anterior knee pain

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Children report back pain less frequently than adults, although >50% will have had one episode by late adolescence. Back pain in a child is a ‘red flag’ for serious spinal pathology; however, if it is mild, intermittent or occurring only on strenuous activity , it is usually self-limiting. Many adolescents do su ff er posture- related discomfort. Physiotherapy to improve core strength and stability reduces symptoms if exercises are performed regularly . Summary box 44.17 ‘Red flag’ symptoms and signs for spinal pathology /uni25CF /uni25CF /uni25CF /uni25CF All ‘red flag’ signs require urgent investigation with a full blood count (FBC), erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), plain radiograph and MRI or other - imaging. Other causes of back pain include intra-abdominal, renal and systemic pathology . Summary box 44.18 - Other spinal conditions /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

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# Blount’s disease

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The aetiology of the disordered growth in the posteromedial proximal tibial physis is unknown. The infantile form is more Walter Putnam Blount , 1900–1992, Professor of Orthopaedic Surgery , Marquette University , Milwaukee, WI, USA, described this condition in 1937. disease a ff ects all ethnic groups. The child presents with progressive and often severe tibia vara with significant intoeing. The radiographic features are diagnostic ( Figure 44.27 ). Treatment is surgical: following correction of limb align - ment via an osteotomy , an epiphysiodesis of the remaining physis prevents recurrence. In unilateral cases, the limb – once straightened – is short and concomitant tibial lengthening with an external fixator is often an attractive option.

Figure 44.27 Standing leg length and alignment radiograph of a child with bilateral asymmetrical bow legs. Both proximal medial tibial physes/epiphyses are abnormal: this is bilateral Blount’s disease.

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# Brachial plexopathy

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Neonatal brachial plexus injury is still common, with a devas - tating e ff ect on upper limb function, particularly if antigravity motor activity has not recovered by 6 months. Physiotherapy is the mainstay of early treatment to maintain muscle length and joint range of movement and thus reduce the risk of glenohumeral dislocation. Neural repair may be necessary in the infant. Later surgical interventions aim to release joint/ muscle contractures and improve function, perhaps with tendon transfers. Brachial plexopathy

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# CLINICAL DILEMMAS The limping child

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TABLE 44.18 A guide to the clinical assessment of the limping child. Symptom onset: sudden or gradual? Symptom duration Concurrent events: recent viral infection, trauma, new sport? General health: is the child well? TABLE 44.19 Age at presentation of certain hip conditions. Age (years) Diagnosis 1–3 Sepsis Late presenting developmental dysplasia of the hip 3–10 Transient synovitis Perthes’ disease 11–15 Slipped upper femoral epiphysis

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# CONGENITAL AND DEVELOPMENTAL ABNORMALITIES

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# Cerebral palsy

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Cerebral palsy (CP) is caused by a non-progressive insult to the developing brain in the perinatal period; in most cases only risk factors, such as prematurity , rather than specific causes, such as hypoxia (hypoxic ischaemic encephalopathy [HIE]), can be identified. The e ff ects of CP may only become apparent as the child grows and fails to reach developmental milestones. Investigations may identify the aetiology and predict the pattern of the CP: premature babies may show evidence of periventricular leukomalacia (PVL) on MRI, associated with a spastic diplegia and relative preservation of intellectual function. , 1806–1875, neurologist, worked successively in Boulogne and Paris, France, but never

Aim of treatment Restores joint range (but results in relative muscle weakness) correction of /f_i xed deformity Restores mechanical alignment and allows muscles to work in a more ef /f_i cient manner Improves posture/function; reduces pain Reduce spasticity – not useful in dystonia Improve lower limb mechanics

/uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF In general, the pattern of involvement can be classified according to the anatomical site involved and the e ff ect on muscle tone ( Table 44.16 ). The prognosis for walking can be predicted by identifying evidence of neurological develop ment, i.e. gaining motor skills and losing primitive reflexes. The age-related Gross Motor Function Classification System (GMFCS) has five categories that relate to mobility and prog nosis (GMFCS /uni00A0 I – near-normal versus GMFCS /uni00A0 V – wheel chair based). Many children with GMFCS /uni00A0 V CP have multiple associated problems and a short life expectancy . An important aspect of management is the control of high tone. Tone can be reduced with drugs (e.g. diazepam and baclofen). Alternatively , neuromuscular blockers such as botulinum to xin allow a focal reduction in tone by preventing acetylcholine release at the neuromuscular junction. The e ff ect is temporary , giving a ‘window’ during which the physio therapists can stretch agonists and strengthen antagonists. It is important to di ff erentiate between dynamic and fixed contractures; the latter will not respond to tone management or splinting. The classic CP gait patterns demonstrate flexor spasticity The child with spastic diplegia has problems at all levels of the lower limb. Single-event multilevel sur gery (SEMLS) is pop ular, and gait analysis (both observational and computerised) contributes to the selection of an appropriate management plan for an individual patient. Computerised analysis provides objective evidence of joint movement and mechanics in mul tiple planes ( Figure 44.37 ). Appropriate bone and soft-tissue procedures can then be planned. Botulinum toxin helps with postoperative pain and spasm. In the child with total body involvement (TBI) and high muscle tone, hip subluxation leading to dislocation is common ( Figur e 44.38 ). Current thinking is that symmetry and pelvic position are important so the hips should be kept in joint with early surgical intervention if necessary . Aggressive management of a spinal deformity initially concentrates on seating position and subsequently emphasises spinal bracing or surgery .

Overall, it is important to remember that, in adulthood, independent mobility , even if in a wheelchair, and e ff ective communication are the most important requirements. - Summary box 44.20 - - Cerebral palsy /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF -

Characteristics Tone Commonest type of abnormality; due to pyramidal system damage Spastic (‘high’) Velocity-dependent increased muscle tone and brisk re /f_l exes Dyskinetic Dystonic Increased tone but reduced activity – stiff movements Choreoathetoid Low tone but increased activity – uncoordinated jerky movements Due to damage in the extrapyramidal system Generalised low tone, loss of muscle coordination Ataxia Due to cerebellar damage No one tone/movement disorder predominatesw Mixed Combination of spasticity and dystonia is common Hypotonia Usually a phase (which may last years) before the features of spasticity develop Site Unilateral Hemiplegia Arm more affected than leg Bilateral Diplegia Legs more affected than arms Total body involvement Often signi /f_i cant intellectual impairment and associated dif /f_i culties Brain injury is non-progressive Classi /f_i ed as unilateral or bilateral involvement: hemiplegia, diplegia or TBI Tone may be high, low or variable but there is always a generalised, relative muscle weakness In ambulant children, gait analysis may be used to plan management In TBI, primary concerns are hip subluxation and spinal deformity

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/uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF In general, the pattern of involvement can be classified according to the anatomical site involved and the e ff ect on muscle tone ( Table 44.16 ). The prognosis for walking can be predicted by identifying evidence of neurological develop ment, i.e. gaining motor skills and losing primitive reflexes. The age-related Gross Motor Function Classification System (GMFCS) has five categories that relate to mobility and prog nosis (GMFCS /uni00A0 I – near-normal versus GMFCS /uni00A0 V – wheel chair based). Many children with GMFCS /uni00A0 V CP have multiple associated problems and a short life expectancy . An important aspect of management is the control of high tone. Tone can be reduced with drugs (e.g. diazepam and baclofen). Alternatively , neuromuscular blockers such as botulinum to xin allow a focal reduction in tone by preventing acetylcholine release at the neuromuscular junction. The e ff ect is temporary , giving a ‘window’ during which the physio therapists can stretch agonists and strengthen antagonists. It is important to di ff erentiate between dynamic and fixed contractures; the latter will not respond to tone management or splinting. The classic CP gait patterns demonstrate flexor spasticity The child with spastic diplegia has problems at all levels of the lower limb. Single-event multilevel sur gery (SEMLS) is pop ular, and gait analysis (both observational and computerised) contributes to the selection of an appropriate management plan for an individual patient. Computerised analysis provides objective evidence of joint movement and mechanics in mul tiple planes ( Figure 44.37 ). Appropriate bone and soft-tissue procedures can then be planned. Botulinum toxin helps with postoperative pain and spasm. In the child with total body involvement (TBI) and high muscle tone, hip subluxation leading to dislocation is common ( Figur e 44.38 ). Current thinking is that symmetry and pelvic position are important so the hips should be kept in joint with early surgical intervention if necessary . Aggressive management of a spinal deformity initially concentrates on seating position and subsequently emphasises spinal bracing or surgery . Overall, it is important to remember that, in adulthood, independent mobility , even if in a wheelchair, and e ff ective communication are the most important requirements. - Summary box 44.20 - - Cerebral palsy /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF -

Characteristics Tone Commonest type of abnormality; due to pyramidal system damage Spastic (‘high’) Velocity-dependent increased muscle tone and brisk re /f_l exes Dyskinetic Dystonic Increased tone but reduced activity – stiff movements Choreoathetoid Low tone but increased activity – uncoordinated jerky movements Due to damage in the extrapyramidal system Generalised low tone, loss of muscle coordination Ataxia Due to cerebellar damage No one tone/movement disorder predominatesw Mixed Combination of spasticity and dystonia is common Hypotonia Usually a phase (which may last years) before the features of spasticity develop Site Unilateral Hemiplegia Arm more affected than leg Bilateral Diplegia Legs more affected than arms Total body involvement Often signi /f_i cant intellectual impairment and associated dif /f_i culties Brain injury is non-progressive Classi /f_i ed as unilateral or bilateral involvement: hemiplegia, diplegia or TBI Tone may be high, low or variable but there is always a generalised, relative muscle weakness In ambulant children, gait analysis may be used to plan management In TBI, primary concerns are hip subluxation and spinal deformity

# Complications of bone and joint sepsis

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Treated appropriately , most cases of sepsis resolve with no sequelae. However, significant complications can occur, particularly in terms of chronic infection and where there has been damage to the joint and/or the physis and the epiphyseal growth centres. In the neonate, vascular channels pass through the physis, connecting the metaphysis with the epiphysis, and a poorer outcome may ensue ( Figure 44.40b ). Orthopaedic follow-up should be continued until normal growth patterns are documented. Meningococcal sepsis The often debilitating, late orthopaedic sequelae of meningo coccal septicaemia are secondary to endotoxin-induced micro vascular injury and ischaemic physeal damage ( Figure 44.42 Tuberculosis Globally , tuberculosis is common. The clinical presentation is often insidious, with malaise and weight loss combined with a boggy joint swelling, muscle wasting and joint contractures. Spinal deformity and neurological symptoms are particular problems. Sir Philip Noel Panton , 1877–1950, Consultant Adviser in Pathology , Ministry of Health, UK. Francis Valentine , 1897–1957, described Panton–Valentine leukocidin as a dermo-necrotic and leukocidal toxin while working with Philip Panton at the Hale Clinical Laboratory , London, UK, in 1932. - Chronic relapsing/recurrent multifocal osteomyelitis The radiographic features suggest subacute or chronic osteo - - myelitis (or tumour) but laboratory and histopathological - findings are non-specific and cultures negative. This is an ). inflammatory ( not infective) condition. Discitis Children who refuse to weight bear and complain of back pain may have discitis. The aetiology of this condition may be infective or inflammatory but if vertebral bodies are involved, infection is assumed.

Figure 44.42 Anteroposterior leg length and alignment radiograph of an adolescent who had meningococcal septicaemia as a child. He has a right below-knee amputation. Many of his lower limb physes are not growing well so he has deformity of his remaining right proximal tibia, a short left tibia and an overgrown /f_i bula. His right femur is also short.

Brodie’s abscess Chronic infections may present with radiographic features of a sclerotic walled cyst.

Figure 44.43 Anteroposterior radiograph of a knee showing meta physeal corner fractures that are considered to be pathognomonic of non-accidental injury.

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# Congenital pseudarthrosis of the tibia

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This rare condition presents with an anterolateral bow of the tibia with or without a fracture. Classic radiographic changes are noted and 50% are associated with neurofibromatosis. Once fractured the tibia is reluctant to heal. Long-term orthotic treatment may be necessary , with subsequent surgical procedures designed to obtain bony union and restore leg length ( Figure 44.28 ). Summary box 44.11 Abnormalities of the knee and lower leg /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

# OCD – better prognosis in children than in adults Discoid meniscus – usually lateral, may require surgery Anterior knee pain – treatment usually conservative Fibular hemimelia – associated with abnormalities from the foot proximally (foot worse than hip); the tibial bow has an anteromedial

apex Blount’s disease – clinically, a sharp proximal tibial angulation Congenital pseudarthrosis of the tibia – the tibial bow has an anterolateral apex Apex posteromedial tibial bow – the bow improves with time but the limb may be short Figure 44.28 Anteroposterior radiograph of a child showing a congenital tibial pseudarthrosis and abnormal /f_i bula. She was born with a bowed lower leg that subse

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# Congenital radial head dislocation

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The dislocation is usually posterolateral, compared with the classic traumatic anterior dislocation ( Figure 44.33 ). Some restriction of elbow joint movement and forearm rotation is noted along with discomfort on activity . Surgical treatment should be avoided in children. - Summary box 44.15 Upper limb abnormalities /uni25CF /uni25CF - /uni25CF

Radial club hand is frequently associated with other congenital anomalies, for example the VACTERL or Holt–Oram syndromes Radioulnar synostosis presents with a /f_i xed forearm position Congenital radial head dislocation is usually posterolateral

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# Congenital talipes equinovarus (the ‘club foot’)

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In true congenital talipes equinovarus (CTEV) the three- dimensional deformity is fixed ( Figure 44.29 ). Intrauterine moulding can cause an identical pattern that is postural and therefore correctable. Incidence and aetiology The incidence varies from 1 to 6 per 1000 live births, depending on ethnic di ff erences. It is more common in boys and is bilateral in 50% of cases. A family history is common but inheritance is multifactorial. The diagnosis may be made during an antenatal ultrasound: the sensitivity is higher in bilateral cases, which are more likely to have a syndromic association. In some countries, the detection of CTEV may be grounds for a termination of the pregnancy but the parents should be reassured that, with treatment, their child will function as well as his/her peers. Most cases are idiopathic but because the outcome varies with the aetiology it is important to consider the cause when planning treatment ( Table 44.10 ). Many idiopathic cases will have some weakness of the evertor muscles. Pathology The talonavicular joint is subluxed, with the navicular displaced medially with respect to the talar head. Ligaments, particularly the calcaneofibular ligament, and tendon sheaths, such as the posterior tibial tendon sheath, are shortened and thickened and contain contractile myofibroblasts. The gastrocsoleus and posterior tibial muscles are smaller than normal, with reduced myofibrils and increased connective tissue, possibly because of a local neuromuscular abnormality . The vascular supply via the dorsalis pedis may be diminished. It remains unclear which abnormalities are primary and which occur as the deformity develops. Clinical assessment The postural club foot may benefit from physiotherapy stretches but, by definition, it must be normal by 3 months of age. Shafique Pirani , contemporary , Clinical Professor, Department of Orthopaedic Surgery , University of British Columbia, V ancouver, Canada. Alain Diméglio , contemporary , orthopaedic surgeon, Montpellier, France. In contrast, the structural idiopathic club foot has fixed deformity with elements of hindfoot equinus and varus, midfoot adductus and pronation of the first ray , giving the appearance of forefoot cavus. The heel feels ‘empty’ as the calcaneus is pulled up by the shortened tendo-Achilles. There is a deep medial and a single posterior crease. All children with structural club-foot deformity will have a small calf and foot. Tibial shortening may become apparent with growth. Children should be examined carefully for signs of intraspinal pathology . Both the Pirani and Diméglio classification systems ar e based on the appearance of the foot in its position of max - imal correction. They predict tr eatment response and hence outcome.

TABLE 44.10 Several different types of club foot are recognised. Type Example Postural Idiopathic Neuromuscular Spina bi /f_i da; arthrogryposis Syndromic Trisomy 15 or Disastrophic Dysplasia or Amniotic Band Syndrome (b) Figure 44.29 (a) Anteroposterior photograph of a foot showing the classic deformities associated with a club foot. The hindfoot (not seen) is in equinus and varus, there is midfoot cavus and the forefoot lies adducted and apparently supinated, although it is actually pronated relative to the hindfoot.

# (b) Untreated congenital talipes equinovarus in a child aged 7 years. Shoe wear is dif /f_i cult although some indepen

dent mobility is possible.

Club foot /uni25CF /uni25CF /uni25CF /uni25CF Treatment Ponseti method The Ponseti method corrects foot deformity in 95% of idio pathic cases without the need for a formal surgical release and is the treatment of choice for all feet. Treatment commences within a few weeks of birth. A specific set of manoeuvres, followed by a series of well-moulded above-knee plaster casts, results in gradual correction of the deformity ( Figure 44.30 The head of the talus is the fulcrum around which the rest of the foot rotates. After the forefoot has been corrected, most feet lack 15° of dorsiflexion and require a percutaneous Achilles tenotomy (performed under local anaesthetic in the clinic setting). Once corrected the foot position is maintained by a foot abduction orthosis (FAO) that holds the foot in external rotation and slight dorsiflexion. The FAO is worn full time for 3 months and at ‘night and nap time’ f or up to 4 years. Poor compliance with the FAO is associated with a higher relapse rate. Recurrent deformity can be treated with further plasters, but a tibialis anterior tendon transfer (TATT) may be required around the age of 2.5–4 years for persisting dynamic supination. Feet treated with the Ponseti method are less sti ff , less likely to be painful and less subject to overcorrection than those treated surgically . The Ponseti method also works reasonably well in non-idiopathic feet but both the failure and relapse rates are higher. Surgical treatment If conservative treatment fails, surgical intervention is required, ideally before walking age; this is more likely in non-idiopathic deformities. Surgical release is generally performed ‘à la carte’, with sequential release of the pathologically tight structures via either Turco or Cincinnati incisions to reduce the subluxated joints. Stabilisation may include temporar y Kirschner wire fixation. Following correction, wound closure can be di ffi cult but the Cincinnati incision heals well by secondary intention during the postoperative casting period. Ignacio Ponseti , 1914–2009, faculty member of the University of Iowa, USA. Born in Menorca, fled Spain during the Civil War because of the political situation, worked as a general practitioner in Mexico and then went to Iowa to train in orthopaedics. The technique that bears his name only became popular years after he retired – but it brought him back to work for another 20 years. Vincent J Turco , 1916–1999, Chief of Orthopedic Surgery , St. Francis Hospital, Hartford, CT , and Assistant Clinical Professor, Yale University Medical School, New Haven, and the University of Connecticut Medical School, Farmington, CT , USA. Martin Kirschner , 1879–1942, Professor of Surgery , Heidelberg, Germany , introduced the use of skeletal traction wires in 1909. - Good or excellent results can be achieved but sti ff ness and over- or undercorrection are common complications. ). Surgery for recurrent deformity r equires a careful assess - ment of the forefoot, hindfoot and tibial torsion. The foot becomes progressively sti ff er with each surgical intervention. Summary box 44.13 Treatment of

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Multiplanar deformity: hindfoot equinus and varus, midfoot adductus and forefoot cavus Incidence is 1–6 per 1000 live births, more common in boys and with a familial tendency Most cases are idiopathic but neuromuscular causes include spina bi /f_i da and arthrogryposis Scoring systems (Pirani/Diméglio), are used to assess severity Corrected Uncorrected Figure 44.30 A series of casts documenting the stepwise correction of the foot deformity with the Ponseti method of serial manipulation and casting. The Ponseti method of serial casting is successful in 95% of feet when de /f_i ned as avoiding formal surgical release The standard sequence of manipulations is: C – correction of the apparent forefoot cavus by elevation of the /f_i rst ray A – gradual forefoot abduction to 60°, and simultaneous V – correction of the hindfoot varus E – correction of hindfoot equinus usually follows a percutaneous Achilles tenotomy, which is an integral part of treatment TATT is used to correct dynamic supination in older children If the Ponseti method fails: Surgical release addresses posterior, medial, plantar and lateral structures, and results in a stiffer foot than one treated conservatively

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In true congenital talipes equinovarus (CTEV) the three- dimensional deformity is fixed ( Figure 44.29 ). Intrauterine moulding can cause an identical pattern that is postural and therefore correctable. Incidence and aetiology The incidence varies from 1 to 6 per 1000 live births, depending on ethnic di ff erences. It is more common in boys and is bilateral in 50% of cases. A family history is common but inheritance is multifactorial. The diagnosis may be made during an antenatal ultrasound: the sensitivity is higher in bilateral cases, which are more likely to have a syndromic association. In some countries, the detection of CTEV may be grounds for a termination of the pregnancy but the parents should be reassured that, with treatment, their child will function as well as his/her peers. Most cases are idiopathic but because the outcome varies with the aetiology it is important to consider the cause when planning treatment ( Table 44.10 ). Many idiopathic cases will have some weakness of the evertor muscles. Pathology The talonavicular joint is subluxed, with the navicular displaced medially with respect to the talar head. Ligaments, particularly the calcaneofibular ligament, and tendon sheaths, such as the posterior tibial tendon sheath, are shortened and thickened and contain contractile myofibroblasts. The gastrocsoleus and posterior tibial muscles are smaller than normal, with reduced myofibrils and increased connective tissue, possibly because of a local neuromuscular abnormality . The vascular supply via the dorsalis pedis may be diminished. It remains unclear which abnormalities are primary and which occur as the deformity develops. Clinical assessment The postural club foot may benefit from physiotherapy stretches but, by definition, it must be normal by 3 months of age. Shafique Pirani , contemporary , Clinical Professor, Department of Orthopaedic Surgery , University of British Columbia, V ancouver, Canada. Alain Diméglio , contemporary , orthopaedic surgeon, Montpellier, France. In contrast, the structural idiopathic club foot has fixed deformity with elements of hindfoot equinus and varus, midfoot adductus and pronation of the first ray , giving the appearance of forefoot cavus. The heel feels ‘empty’ as the calcaneus is pulled up by the shortened tendo-Achilles. There is a deep medial

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TABLE 44.10 Several different types of club foot are recognised. Type Example Postural Idiopathic Neuromuscular Spina bi /f_i da; arthrogryposis Syndromic Trisomy 15 or Disastrophic Dysplasia or Amniotic Band Syndrome (b) Figure 44.29 (a) Anteroposterior photograph of a foot showing the classic deformities associated with a club foot. The hindfoot (not seen) is in equinus and varus, there is midfoot cavus and the forefoot lies adducted and apparently

# supinated, although it is actually pronated relative to the hindfoot. (b) Untreated congenital talipes equinovarus in a child aged 7 years. Shoe wear is dif /f_i cult although some indepen

dent mobility is possible.

Club foot /uni25CF /uni25CF /uni25CF /uni25CF Treatment Ponseti method The Ponseti method corrects foot deformity in 95% of idio pathic cases without the need for a formal surgical release and is the treatment of choice for all feet. Treatment commences within a few weeks of birth. A specific set of manoeuvres, followed by a series of well-moulded above-knee plaster casts, results in gradual correction of the deformity ( Figure 44.30 The head of the talus is the fulcrum around which the rest of the foot rotates. After the forefoot has been corrected, most feet lack 15° of dorsiflexion and require a percutaneous Achilles tenotomy (performed under local anaesthetic in the clinic setting). Once corrected the foot position is maintained by a foot abduction orthosis (FAO) that holds the foot in external rotation and slight dorsiflexion. The FAO is worn full time for 3 months and at ‘night and nap time’ f or up to 4 years. Poor compliance with the FAO is associated with a higher relapse rate. Recurrent deformity can be treated with further plasters, but a tibialis anterior tendon transfer (TATT) may be required around the age of 2.5–4 years for persisting dynamic supination. Feet treated with the Ponseti method are less sti ff , less likely to be painful and less subject to overcorrection than those treated surgically . The Ponseti method also works reasonably well in non-idiopathic feet but both the failure and relapse rates are higher. Surgical treatment If conservative treatment fails, surgical intervention is required, ideally before walking age; this is more likely in non-idiopathic deformities. Surgical release is generally performed ‘à la carte’, with sequential release of the pathologically tight structures via either Turco or Cincinnati incisions to reduce the subluxated joints. Stabilisation may include temporar y Kirschner wire fixation. Following correction, wound closure can be di ffi cult but the Cincinnati incision heals well by secondary intention during the postoperative casting period. Ignacio Ponseti , 1914–2009, faculty member of the University of Iowa, USA. Born in Menorca, fled Spain during the Civil War because of the political situation, worked as a general practitioner in Mexico and then went to Iowa to train in orthopaedics. The technique that bears his name only became popular years after he retired – but it brought him back to work for another 20 years. Vincent J Turco , 1916–1999, Chief of Orthopedic Surgery , St. Francis Hospital, Hartford, CT , and Assistant Clinical Professor, Yale

University Medical School, New Haven, and the University of Connecticut Medical School, Farmington, CT , USA. Martin Kirschner , 1879–1942, Professor of Surgery , Heidelberg, Germany , introduced the use of skeletal traction wires in 1909. - Good or excellent results can be achieved but sti ff ness and over- or undercorrection are common complications. ). Surgery for recurrent deformity r equires a careful assess - ment of the forefoot, hindfoot and tibial torsion. The foot becomes progressively sti ff er with each surgical intervention. Summary box 44.13 Treatment of club foot /uni25CF /uni25CF /uni25CF

Multiplanar deformity: hindfoot equinus and varus, midfoot adductus and forefoot cavus Incidence is 1–6 per 1000 live births, more common in boys and with a familial tendency Most cases are idiopathic but neuromuscular causes include spina bi /f_i da and arthrogryposis Scoring systems (Pirani/Diméglio), are used to assess severity Corrected Uncorrected Figure 44.30 A series of casts documenting the stepwise correction of the foot deformity with the Ponseti method of serial manipulation and casting. The Ponseti method of serial casting is successful in 95% of feet when de /f_i ned as avoiding formal surgical release The standard sequence of manipulations is: C – correction of the apparent forefoot cavus by elevation of the /f_i rst ray A – gradual forefoot abduction to 60°, and simultaneous V – correction of the hindfoot varus E – correction of hindfoot equinus usually follows a percutaneous Achilles tenotomy, which is an integral part of treatment TATT is used to correct dynamic supination in older children If the Ponseti method fails: Surgical release addresses posterior, medial, plantar and lateral structures, and results in a stiffer foot than one treated conservatively

# DEVELOPMENT OF THE MUSCULOSKELETAL SYSTEM

DEVELOPMENT OF THE MUSCULOSKELETAL SYSTEM

The upper limb bud forms on the lateral wall of the 4-week embryo, followed promptly by the lower limb bud. By 2 /uni00A0 months, di ff erentiation of the limb elements is complete. Most congenital limb anomalies arise during this period. Three coordinated signalling centres control limb devel opment. The apical ectodermal ridge (AER) guides Carl Hueter , 1838–1882, Professor of Surgery , University of Greifswald, Germany . Richard von Volkmann , 1830–1889, Professor of Surgery , Halle, Germany . Julius Wol ff , 1836–1902, Professor of Orthopaedic Surgery , Berlin, Germany . mesodermal di ff erentiation in a proximal-to-distal direction, controlling digit formation via the production of fibroblast growth factors (FGFs). The mesodermal zone of polaris - ing activity (ZPA) directs anteroposterior development via the sonic hedgehog protein, which is itself sustained by FGFs. The ectodermal driven wingless-type (Wnt) signalling centre develops dorsoventral axis configuration and limb alignment. Certain limb anomalies are directly related to alterations in these centres. Experimentally , removal of the AER leads to a truncated limb, similar to a congenital amputation, and pre - vents interdigital necrosis resulting in syndactyly . An additional ZP A results in a mirror duplication of the distal limb. Factors causing fetal limb anomalies may also influence other organ formation, resulting in potentially life-threa tening disorders. Summary box 44.1 Development of the musculoskeletal system /uni25CF /uni25CF /uni25CF /uni25CF

Presentation and management of other childhood hip • conditions Management of club foot • Problems associated with musculoskeletal infection in • childhood Occurs 4–8 weeks after fertilisation AER controls proximal-to-distal differentiation and interdigital necrosis ZPA directs posterior-to-anterior differentiation Wnt in /f_l uences dorsal-to-ventral differentiation

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# Developmental dysplasia of the hip

Developmental dysplasia of the hip

- DDH defines the spectrum of hip instability , ranging from the hip that is in joint but has a shallow (dysplastic) acetabulum and may be ‘pushed out’ (Barlow positive) to the dislocated hip that is irreducible (Ortolani negative). The clinical picture varies with the pathology and the age at presentation: neonatal hips may be unstable, a toddler may limp, adolescents may experience exercise-induced pain and an adult may have pain - secondary to degenerative arthritis. Incidence The incidence of neonatal instability is approximately 20 per 1000 live births, whereas that of true dislocation is approxi - mately 2 per 1000 live births; many hips stabilise spontaneously . Aetiology of developmental dysplasia of the hip /uni25CF Gender . Four to five times more common in girls, possibly related to hormonal factors causing temporary joint laxity in the peripartum period. /uni25CF Breech presentation . More common in breech babies, particularly with the extended breech position. /uni25CF Birth order . More common in firstborns and in the left hip because of the common fetal position (left occipito - anterior) in a tight primigravid uterus where movement is restricted. /uni25CF Oligohydramnios . Restricts fetal movement. The presence of other postural deformities (torticollis and metatarsus adductus) raises the possibility of DDH. /uni25CF Family history . A positive family history significantly increases the risk of DDH. /uni25CF Regional and racial variation . More common in certain regions and in certain races because of a combina - tion of genetic, environmental and cultural factors. /uni25CF Swaddling the legs together exacerbates hip insta - bility , whereas carrying the baby astride the carer’s hip or back encourages hip flexion and abduction that improve stability . Hip dislocation is often found in association with gener - alised syndromes or neuromuscular conditions. These terato - logical hips ar e often resistant to the simpler treatments and a holistic approach to the child’s overall condition and prognosis must be taken. Diagnosis Neonate /uni25CF Clinical assessment and screening . In many coun - tries, neonates are screened for limitation of hip abduction and hip joint instability . In the UK, as part of the newborn and infant physical examination (NIPE) guidelines, the hips are examined again at 6 weeks. The knees and hips are flexed and the thigh held b y the examiner with the thumb trochanter. The hips are abducted gently: if abduction is limited, the hip may be dislocated. The examiner’s finger then lifts the greater trochanter upwards; a soft clunk – the Ortolani test – with improved hip abduction signifies hip reduction ( Figure 44.13a ). If the hip does abduct fully , then the flexed hip is brought back to neutral and then adducted while downward pressure is applied to the knee with the examiner’s thumb and palm: an unstable hip may dislocate or sublux – the Barlow test ( Figure 44.13b With an irreducible hip there is no clunk of reduction but there will be limitation of abduction. Bilateral dislocation may be missed

because abduction is symmetrical and ab duction may be normal when there is low muscle tone and joint laxity . In a dislocated hip, the femoral head may be palpable in the buttock. /uni25CF Ultrasonographic assessment . Ultrasonography defines the anatomy and the stability of the hip joint. It is used as a screening tool (universally or selectively for ‘at risk’ patients) ( Figure 44.14 ). Screening scans should be performed between 4 and 6 weeks of age and treatment, when necessary , started by 6 weeks. The sonographic appearance of most hips impro ves (in terms of both hip stability and acetabular dysplasia) spontaneously as the baby grows. /uni25CF Radiography . Plain radiographs are used from 4–5 months of age, when the relationship of the femoral ossific nucleus to the acetabulum can be assessed; late ossi fication of the nucleus is common in DDH ( Figure 44.15 Infant Hip checks, looking for limitation of abduction in more than 90° of flexion and limb shortening, are part of developmental monitoring. Child Children present with a Trendelenburg gait and/or unilateral tiptoeing, as the a ff ected leg is short. Abduction in flexion is Friedrich Trendelenburg , 1844–1924, successively Professor of Surgery at Rostock (1875–1882), Bonn (1822–1895) and Leipzig (1895–1911), Germany . The neonatal clinical examination must ask and answer the following questions: /uni25CF /uni25CF /uni25CF /uni25CF ). /uni25CF /uni25CF - limited and there may be an extra thigh crease. The signs may be subtle and easily missed in an unsteady toddler. If both hips are a ff ected there will be a waddling gait and a lumbar lordosis. - Adolescent Discomfort after exercise is common but the pain may be in the knee. In all age groups, radiographs may show dysplasia, sublux - ation or dislocation. Summary box 44.6 - ). Diagnosis of DDH /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

(a) Figure 44.13 Line diagram illustrating the (a) Ortolani and (b) Barlow tests for developmental dysplasia of the hip. For the Barlow test the femur must be at 90° to the bed. Is the hip dislocated? If so, is it reducible (Ortolani positive) or not (Ortolani negative)? If the hip is not dislocated, is it dislocatable (or subluxable)? If so, it is Barlow positive If the hip is not dislocated or dislocatable, is it clinically normal? If so, do the risk factors in the history still demand further assessment with an ultrasound scan or plain radiograph? Based on the history and clinical examination and con /f_i rmed by appropriate investigations All neonates are screened clinically (Barlow and Ortolani tests) at birth and at 6 weeks Ultrasound is used as a selective screening test in ‘at-risk’ babies Radiography is useful from 4 months onwards Older children present with a limp and/or tiptoeing and a lumbar lordosis in bilateral cases (b)

a (c) Management The objective is to obtain a stable, congruous reduction of the femoral head within the acetabulum while avoiding damage to the capital epiphysis (avascular necrosis [A VN]), which causes sti ff ness and proximal femoral deformity . Neonate Owing to the peripartum hormonal e ff ects many neonatal hips are unstable. Most stabilise spontaneously by 6 weeks. Hips that remain unstable or that are dislocated at rest are treated with harnesses or splints that obtain and maintain reduction with the hip abducted and flexed. Joint stability is monitored with ultrasound scanning. Most harnesses ( Figure 44.16 ) allow controlled mo vement while splints hold the hips more rigidly and may carry a greater risk of A VN and femoral nerve palsy . If the hips fail to relocate or stabilise, treatment should be discontinued. Christian Morin , contemporary , French paediatric orthopaedic surgeon. Heinrich Hilgenreiner , 1870–1954, German surgeon and orthopaedist. George Perkins , 1892–1980, Professor of Surgery , St Thomas’ Hospital, London, UK,

described signs by which to diagnose congenital dislocation of the hip in 1928. a α α a b Infant Successful harness treatment is unusual after the age of 4–6 months. For the late-presenting hip or one that fails conservative treatment, an examination under anaesthetic may achieve a closed reduction. A psoas/adductor release can

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be performed if the arthrogram suggests they are blocking reduction or limiting stability . Postoperatively , a spica cast maintains hip reduction. If the hip is irreducible or can only be held reduced in an extreme position then treatment should be abandoned and an open reduction considered via a medial or anterior approach. Summary box 44.7 Management of early DDH /uni25CF /uni25CF /uni25CF /uni25CF Child A medial approach open reduction can be performed between 6 and 24 months of age. An anterior approach to the hip (from 9–12 months of age) allows for a simultaneous capsulorrhaphy . In the older child, a pelvic osteotomy may be required to reorientate the acetabulum, and femoral shortening or derotation osteotomies will improve stability ( Figure 44.17 Arnold Pavlik , 1902–1962, Czech orthopaedic surgeon, became famous mainly for the development of a functional, active method of treating developmental dysplasia of the hip. Surgery is contraindicated in children over the age of 6–8 years in bilateral cases and the age of 8–10 years in unilateral cases ( Figure 44.18 ). Adolescent Hips are often dysplastic and subluxated. If the hip is reducible, the joint can be reconstructed with a combination of pelvic and femoral osteotomies. For the irreducible hip, acetabular augmentation may reduce symptoms and delay the onset of degenerative change. ).

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Management of late-presenting DDH /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Secondary procedures and complications At follow-up, acetabular remodelling is assessed with the acetabular index or the centre–edge angle ( Figure 44.19 Surgery may be required for residual dysplasia or subluxation. A VN with trochanteric overgrowth causes a Trendelenburg limp: the outcome is poor (

Figure 44.20 ). Occasionally , a leg length di ff erence needs treatment. There is an increased risk of osteoarthritis and hip arthroplasty later in life.

The older the child, the more likely it is that they will require surgery Femoral osteotomy improves hip stability Pelvic osteotomy redirects or reshapes the acetabulum The potential for acetabular remodelling decreases after the age of 3–4 years Avascular necrosis is a risk with all DDH treatment

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Figure 44.20 ). Occasionally , a leg length di ff erence needs treatment. There is an increased risk of osteoarthritis and hip arthroplasty later in life.

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Developmental dysplasia of the hip

- DDH defines the spectrum of hip instability , ranging from the hip that is in joint but has a shallow (dysplastic) acetabulum and may be ‘pushed out’ (Barlow positive) to the dislocated hip that is irreducible (Ortolani negative). The clinical picture varies with the pathology and the age at presentation: neonatal hips may be unstable, a toddler may limp, adolescents may experience exercise-induced pain and an adult may have pain - secondary to degenerative arthritis. Incidence The incidence of neonatal instability is approximately 20 per 1000 live births, whereas that of true dislocation is approxi - mately 2 per 1000 live births; many hips stabilise spontaneously . Aetiology of developmental dysplasia of the hip /uni25CF Gender . Four to five times more common in girls, possibly related to hormonal factors causing temporary joint laxity in the peripartum period. /uni25CF Breech presentation . More common in breech babies, particularly with the extended breech position. /uni25CF Birth order . More common in firstborns and in the left hip because of the common fetal position (left occipito - anterior) in a tight primigravid uterus where movement is restricted. /uni25CF Oligohydramnios . Restricts fetal movement. The presence of other postural deformities (torticollis and metatarsus adductus) raises the possibility of DDH. /uni25CF Family history . A positive family history significantly increases the risk of DDH. /uni25CF Regional and racial variation . More common in certain regions and in certain races because of a combina - tion of genetic, environmental and cultural factors. /uni25CF Swaddling the legs together exacerbates hip insta - bility , whereas carrying the baby astride the carer’s hip or back encourages hip flexion and abduction that improve stability . Hip dislocation is often found in association with gener - alised syndromes or neuromuscular conditions. These terato - logical hips ar e often resistant to the simpler treatments and a holistic approach to the child’s overall condition and prognosis must be taken. Diagnosis Neonate /uni25CF Clinical assessment and screening . In many coun - tries, neonates are screened for limitation of hip abduction and hip joint instability . In the UK, as part of the newborn and infant physical examination (NIPE) guidelines, the hips are examined again at 6 weeks. The knees and hips are flexed and the thigh held b y the examiner with the thumb trochanter. The hips are abducted gently: if abduction is limited, the hip may be dislocated. The examiner’s finger then lifts the greater trochanter upwards; a soft clunk – the Ortolani test – with improved hip abduction signifies hip reduction ( Figure 44.13a ). If the hip does abduct fully , then the flexed hip is brought back to neutral and then adducted while downward pressure is applied to the knee with the examiner’s thumb and palm: an unstable hip may dislocate or sublux – the Barlow test ( Figure 44.13b With an irreducible hip there is no clunk of reduction but there will be limitation of abduction. Bilateral dislocation may be missed because abduction is symmetrical and ab duction may be normal when there is low

muscle tone and joint laxity . In a dislocated hip, the femoral head may be palpable in the buttock. /uni25CF Ultrasonographic assessment . Ultrasonography defines the anatomy and the stability of the hip joint. It is used as a screening tool (universally or selectively for ‘at risk’ patients) ( Figure 44.14 ). Screening scans should be performed between 4 and 6 weeks of age and treatment, when necessary , started by 6 weeks. The sonographic appearance of most hips impro ves (in terms of both hip stability and acetabular dysplasia) spontaneously as the baby grows. /uni25CF Radiography . Plain radiographs are used from 4–5 months of age, when the relationship of the femoral ossific nucleus to the acetabulum can be assessed; late ossi fication of the nucleus is common in DDH ( Figure 44.15 Infant Hip checks, looking for limitation of abduction in more than 90° of flexion and limb shortening, are part of developmental monitoring. Child Children present with a Trendelenburg gait and/or unilateral tiptoeing, as the a ff ected leg is short. Abduction in flexion is Friedrich Trendelenburg , 1844–1924, successively Professor of Surgery at Rostock (1875–1882), Bonn (1822–1895) and Leipzig (1895–1911), Germany . The neonatal clinical examination must ask and answer the following questions: /uni25CF /uni25CF /uni25CF /uni25CF ). /uni25CF /uni25CF - limited and there may be an extra thigh crease. The signs may be subtle and easily missed in an unsteady toddler. If both hips are a ff ected there will be a waddling gait and a lumbar lordosis. - Adolescent Discomfort after exercise is common but the pain may be in the knee. In all age groups, radiographs may show dysplasia, sublux - ation or dislocation. Summary box 44.6 - ). Diagnosis of DDH /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

(a) Figure 44.13 Line diagram illustrating the (a) Ortolani and (b) Barlow tests for developmental dysplasia of the hip. For the Barlow test the femur must be at 90° to the bed. Is the hip dislocated? If so, is it reducible (Ortolani positive) or not (Ortolani negative)? If the hip is not dislocated, is it dislocatable (or subluxable)? If so, it is Barlow positive If the hip is not dislocated or dislocatable, is it clinically normal? If so, do the risk factors in the history still demand further assessment with an ultrasound scan or plain radiograph? Based on the history and clinical examination and con /f_i rmed by appropriate investigations All neonates are screened clinically (Barlow and Ortolani tests) at birth and at 6 weeks Ultrasound is used as a selective screening test in ‘at-risk’ babies Radiography is useful from 4 months onwards Older children present with a limp and/or tiptoeing and a lumbar lordosis in bilateral cases (b)

a (c) Management The objective is to obtain a stable, congruous reduction of the femoral head within the acetabulum while avoiding damage to the capital epiphysis (avascular necrosis [A VN]), which causes sti ff ness and proximal femoral deformity . Neonate Owing to the peripartum hormonal e ff ects many neonatal hips are unstable. Most stabilise spontaneously by 6 weeks. Hips that remain unstable or that are dislocated at rest are treated with harnesses or splints that obtain and maintain reduction with the hip abducted and flexed. Joint stability is monitored with ultrasound scanning. Most harnesses ( Figure 44.16 ) allow controlled mo vement while splints hold the hips more rigidly and may carry a greater risk of A VN and femoral nerve palsy . If the hips fail to relocate or stabilise, treatment should be discontinued. Christian Morin , contemporary , French paediatric orthopaedic surgeon. Heinrich Hilgenreiner , 1870–1954, German surgeon and orthopaedist. George Perkins , 1892–1980, Professor of Surgery , St Thomas’ Hospital, London, UK, described signs by which to diagnose congenital dislocation of the hip in 1928. a α α a b Infant

Successful harness treatment is unusual after the age of 4–6 months. For the late-presenting hip or one that fails conservative treatment, an examination under anaesthetic may achieve a closed reduction. A psoas/adductor release can

(c) Figure 44.14 Ultrasound images of an infant hip. (a) Normal hip with a high angle and a Morin index of 50% (de /f_i ned as the percentage of the femoral head covered by the acetabulum, i.e. the portion lying below the horizontal red line). (b) Grossly dysplastic hip with a low angle and a Morin index of <50%. This hip is likely to be unstable on dynamic ultrasound scanning, i.e. Barlow positive. (c) A dislocated hip joint (dislocated femoral head, red

# arrow; ‘empty’ acetabulum, white arrow). Figure 44.15 Anteroposterior pelvic radiograph showing Hilgenrein

er’s line (a) and Perkins’ line (b). The femoral head (ossi /f_i c nucleus) of a normal hip lies in the inner lower quadrant. The right hip is normal; the left hip has developmental dysplasia of the hip.

be performed if the arthrogram suggests they are blocking reduction or limiting stability . Postoperatively , a spica cast maintains hip reduction. If the hip is irreducible or can only be held reduced in an extreme position then treatment should be abandoned and an open reduction considered via a medial or anterior approach. Summary box 44.7 Management of early DDH /uni25CF /uni25CF /uni25CF /uni25CF Child A medial approach open reduction can be performed between 6 and 24 months of age. An anterior approach to the hip (from 9–12 months of age) allows for a simultaneous capsulorrhaphy . In the older child, a pelvic osteotomy may be required to reorientate the acetabulum, and femoral shortening or derotation osteotomies will improve stability ( Figure 44.17 Arnold Pavlik , 1902–1962, Czech orthopaedic surgeon, became famous mainly for the development of a functional, active method of treating developmental dysplasia of the hip. Surgery is contraindicated in children over the age of 6–8 years in bilateral cases and the age of 8–10 years in unilateral cases ( Figure 44.18 ). Adolescent Hips are often dysplastic and subluxated. If the hip is reducible, the joint can be reconstructed with a combination of pelvic and femoral osteotomies. For the irreducible hip, acetabular augmentation may reduce symptoms and delay the onset of degenerative change. ).

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# Discoid meniscus

Discoid meniscus

This invariably a ff ects the lateral meniscus, which is abnor - mally thick and covers most of the tibial plateau. The child presents with a painful clunk on knee extension. MRI is usually diagnostic. Surgery is indicated for relief of pain or mechanical symptoms. Discoid meniscus

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# Fibular hemimelia

Fibular hemimelia

In fibular hemimelia there is a congenital failure of formation of the lateral ‘column’ of the lower leg ( Figure 44.26 and Table 44.9 ).

# TABLE 44.9 Classical radiographic features of /f_i bular hemimelia.

| Foot and ankle | Absent lateral rays; tarsal coalition; ball-and-socket ankle joint |
|---|---|
| Lower leg | Absent or de /f_i cient /f_i bula; short, bowed tibia |
| Knee | Absent tibial spine (no cruciate ligament); de /f_i cient lateral femoral condyle |
| Femur | Relative hypoplasia |
| Limb length and | Short; external rotation with/without alignment valgus |

Management is tailored to the severity of the deficiency . Treatment options range from a shoe raise, through multiple episodes of limb equalisation surgery to amputation for the worst cases. An early prediction of the leg length discrepancy at maturity allows a realistic treatment plan to be devised for the patient, which should include consideration of a contra- lateral epiphysiodesis.

Figure 44.26 Anteroposterior (a) and lateral (b) radiographs of two lower limbs showing some of the features of a /f_i bular hemimelia: absent /f_i fth ray in the foot, absent /f_i bula and deformed tibia.

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# Flat foot

Flat foot

All children (<3 years) have flat feet with a fat pad obscuring the arch. Over time, the longitudinal arch develops but 15% of adults have flat feet influenced by familial and racial factors. All flat feet have a flattened medial arch with a valgus heel but two types are distinguishable ( Table 44.3 ). The painless, flexible flat foot needs no treatment. Orthoses do not alter the natural history but can alleviate symptoms if they are present. The symptomatic, rigid flat foot is due to inflammation or a tarsal coalition and requires investigation and medical or sur gical management ( Figure 44.4 ). (tibiofemoral angle) /H11002 /H11002 /H11002 /H11002 /uni25CF /uni25CF /uni25CF /uni25CF - Summary box 44.2 Normal variants /uni25CF /uni25CF /uni25CF - /uni25CF

30 Knee angle 25 20 15 10 5 0 5 10 15 Valgus (°) Varus (°) 20 1 2 3 4 5 6 7 8 91 01 Age (years) Figure 44.3 Graph to show the normal tendency of limb alignment to change from varus to valgus with growth; normal is slight valgus after the age of 7–8 years. Type Characteristics Flexible On tiptoe the arch returns and the heel corrects into varus Subtalar joint movements are full and pain free Rigid On tiptoe the arch fails to return and the heel remains in valgus Subtalar joint movements are restricted and often painful Figure 44.4 Oblique radiograph of the foot that shows the most common form of tarsal coalition: an incomplete calcaneonavicular bar (arrow). Legs are often bowed until age 2 years and then knock-kneed until age 6–7 years Neuromuscular pathology must be excluded in toe walkers, particularly when the onset is late Intoeing or extoeing is associated with excessive femoral or tibial torsion or foot deformity Flexible, pain-free /f_l at feet require no treatment

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# Generalised skeletal dysplasias

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Achondroplasia Achondroplasia is caused by a gain-in-function mutation in the FGFR3 (fibroblast growth factor receptor 3) gene, located on the short arm of chromosome (Chr) 4, which a ff ects enchondral bone formation. It is autosomal dominant. Patients present with disproportionate short stature where the limbs are shorter than the trunk, together with classical clinical and radiographic features ( Figure 44.7 ). Underdevelopment of the foramen magnum and spinal stenosis can cause neurological di ffi culties. Correction of limb alignment may be necessary and limb-lengthening techniques are used in some countries. Hereditary multiple exostoses An autosomal dominant condition related to a loss-of function mutation in either the EXT1 (Chr 8q) or gene (Chr 11p), leads to dysregulated growth and exostosis formation. Exostoses consisting of a cartilaginous cap on a bony stalk may be sessile or pedunculated. They grow as the child grows and may cause cosmetic or functional di ffi culties, - EXT2

# Figure 44.5 A child with arthrogryposis multiplex congenita and featureless upper limbs (no skin creases or muscle de /f_i nition). He mobilises with the help of knee–ankle–foot orthoses. Figure 44.6 Radiograph of a child born with proximal femoral focal

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(a) (b) Figure 44.7 Achondroplasia. (a) A child with achondroplasia: his upper limbs are short and his hands do not reach midthigh. (b) Standing leg length/alignment radiograph of a different child demonstrating short limbs, widened metaphysis, an overlong /f_i bula and slight bowing. The acetabulum is horizontal and the pelvic wings seem square: classical features of achondroplasia. Achondroplasia affects enchondral ossi /f_i cation and presents with disproportionate short stature Exostoses may cause functional and/or cosmetic problems Patients with Ollier's disease (multiple enchondromatosis) often have lesions in the hands and feet Figure 44.8 Radiograph of the knee showing multiple broad-based osteochondromas.

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Enchondromatosis (Ollier’s disease) Enchondromas arise from chondrocyte rests within the medul - lary canal of tubular bones: they consist of mature hyaline cartilage ( Figure 44.9 ). Larger lesions

may show calcification on radiographs and vertical lucent streaks (representing cartilage columns) in the metaphysis. Pathological fractur es are common. In Ma ff ucci’s syndrome there are also soft-tissue haemangiomas and lymphangiomas (see Chapter 42 ). Fibrous dysplasia This common disorder is often a chance radiographic finding, particularly in its monostotic form. It is a localised defect in osteoblastic di ff erentiation and maturation in which normal bone is replaced by fibrous stroma. With polyostotic fibrous dysplasia, limb deformity and pathological fractures are common. In patients with precocious puberty and Coast of Maine café-au-lait spots, the diagnosis is McCune–Albright syndrome ( Figure 44.10 ). Summary box 44.3 Congenital and developmental abnormalities of the skeleton /uni25CF /uni25CF /uni25CF

(a) (b) Figure 44.7 Achondroplasia. (a) A child with achondroplasia: his upper limbs are short and his hands do not reach midthigh. (b) Standing leg length/alignment radiograph of a different child demonstrating short limbs, widened metaphysis, an overlong /f_i bula and slight bowing. The acetabulum is horizontal and the pelvic wings seem square: classical features of achondroplasia. Achondroplasia affects enchondral ossi /f_i cation and presents with disproportionate short stature Exostoses may cause functional and/or cosmetic problems Patients with Ollier’s disease (multiple enchondromatosis) often have lesions in the hands and feet Figure 44.8 Radiograph of the knee showing multiple broad-based osteochondromas.

Figure 44.9 Anteroposterior radiograph of the index /f_i nger of a child showing a solitary enchondroma (arrow): note the opacity in the soft tissues, which represents the extent of the cartilaginous lesion.

# INFECTION

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# Intoeing gait

Intoeing gait

Intoeing is defined as a negative foot progression angle and results from one or more lower limb torsional anomalies ( Figure 44.1 and Table 44.2 ). Persistent femoral neck anteversion presents clinically with excessive internal rotation at the hip joint, which is best assessed with the patient pr one ( Figure 44.2a ). All femurs are anteverted at birth but as the femur lengthens it rotates with spontaneous improvement in the anteversion. If, by 10–12 years, a persistent deformity is associated with functional di ffi culties, corrective osteotomy may be justified. In such cases, the child has no ability to externally rotate the extended hip. In others, compensatory external tibial torsion may develop, in which case the foot progression angle will be normal but the child may have symptoms of the miserable malalignment syndrome, including knee pain and feelings of instability . Internal tibial torsion is assessed by the thigh–foot angle and is commonly associated with physiological tibia vara in infants ( Figure 44.2b ). Spontaneous correction occurs by age 4, as the tibia rotates with growth. Metatarsus adductus ( Figure 44.2c ) is usually flexible and corrects by age 2–4 years. For the more rigid foot, stretching, Surgical release is rarely indicated.

TABLE 44.2 Common sites and causes of intoeing gait in childhood. Site Cause Femur/hip Persistent femoral neck anteversion Tibia Internal tibial torsion Foot Metatarsus adductus o o

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- 20 Figure 44.1 Foot progression angle: a positive angle represents an extoeing gait; a negative angle, an intoeing gait. (a) (a) (b) External Internal thigh–foot thigh–foot angle angle (c) Normal Metatarsus adductus Figure 44.2 Assessment of the torsional pro /f_i le: all assessments are done with the child prone. (a) Femoral neck anteversion measured as the range of internal hip rotation with the hip extended and the knee /f_l exed. Craig’s test measures the degree of internal rotation present when the greater trochanter is at its most prominent (also called the trochanteric prominence test). (b) The thigh–foot angle measures the angle between the relaxed hindfoot and the thigh. (c) The bean- shaped foot of metatarsus adductus viewed from above: a curved lateral border with/without a medial crease.

Extoeing is less common but results from relative femoral retroversion, external tibial torsion or flexible flat feet. The child may walk late because of poor balance associated with the foot posture and overall alignment. Gait improves with growth/time. T oe walking is a phase in normal gait development. If the gait does not mature to a heel–toe pattern by 3 years, physiotherapy may help, and older children benefit from surgical lengthening of a contracted gastrocsoleus complex, if it is present. If toe walking starts after walking age, a spinal or neuromuscular aetiology such as a tethered cord or a muscular dystrophy must be considered; in the unilateral case, an orthopaedic cause for a short leg, such as a dislocated hip, must be excluded. Intoeing gait

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# Introduction

INTRODUCTION

Immature skeletons heal rapidly and can remodel with growth but physeal injury or muscle imbalance may lead to progressive deformity . The conservative treatment of common conditions, such as developmental dysplasia of the hip (DDH), considers remodelling ability with an understanding of the Hueter– V olkmann principle and Wol ff ’s law ( Table 44.1 ): improving the biomechanical environment may reverse abnormal growth. In contrast, in conditions such as Blount’s disease, a poorly functioning growth plate leads to asymmetrical growth and deformity . Advances in genetics and molecular science have improved our understanding of certain conditions and may lead to new treatments. /uni25CF /uni25CF /uni25CF

TABLE 44.1 Laws governing the remodelling of bone. Hueter–Volkmann principle Compressive forces inhibit growth Tensile forces stimulate growth Wolff’s law Bone deposition and resorption depend on the stresses applied

# Knock knees and bowlegs

Knock knees and bowlegs

All children start life with bowlegs, often accompanied by internal tibial torsion. By the age of 2–3 years they have devel oped knock knees, which regress towards the normal adult tibiofemoral angle of 7° valgus by age 7 ( Figure 44.3 ). The intercondylar or intermalleolar distance is often used to quantify the deformity but radiographs are needed when the defor mity is sev ere, asymmetrical or symptomatic. The most common pathological causes are previous trauma, rickets or a skeletal dysplasia. Knock knees and bowlegs

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# Kyphosis

Kyphosis

When a kyphosis exceeds the normal 20–50° the cause may be postural or structural. Scheuermann’s disease presents as a progressive structural adolescent kyphosis characterised radiologically by >5° vertebral wedging at three adjacent levels with end-plate changes. The aetiology is unknown. Treatment ranges from physiotherapy and bracing to surgery . William Adams , 1820–1900, described the forward bending test for scoliosis in 1865. John R Cobb , American surgeon, wrote a paper in 1948 on how to measure the angle on a radiograph in scoliosis. Holger Werfel Scheuermann , 1877–1960, radiologist, The Municipal Hospital, Sundby , Copenhagen, Denmark, described juvenile kyphosis in 1920. - Summary box 44.16 Scoliosis /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Figure 44.34 Anteroposterior radiograph of the spine demonstrating multiple congenital vertebral anomalies including hemivertebrae. The arrow points to one of the congenital vertebral anomalies. Figure 44.35 Clinical photograph of the Adams forward bend test that demonstrates the

# presence of a rib hump on the right and a promi

nence of the lumbar paravertebral muscles on the left. Multiplanar deformity includes a rotational component Aetiology may be congenital (underlying bony malformation), neuromuscular, syndromic or idiopathic A leg length discrepancy causes a postural scoliosis Adolescent idiopathic scoliosis is the most common structural scoliosis Back pain associated with scoliosis may be due to infection or tumour Treatment depends on the severity and likelihood of curve progression – it varies from observation, through bracing to surgery

Figure 44.36 Posteroanterior radiograph of a spine with a scoliosis (right thoracic), with a Cobb angle of 40°.

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# Learning objectives

Learning objectives

To be familiar with: Physiological versus pathological development of the • musculoskeletal system Diagnosis and treatment of developmental dysplasia of • the hip Learning objectives

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# Legg–Calvé–Perthes disease

Legg–Calvé–Perthes disease

Incidence and aetiology This rare condition, characterised by the development of A VN of the proximal femoral epiphysis, predominantly a ff ects boys aged 4–7 years; 10% develop bilateral disease. Although the aetiology is unclear, factors such as socioeconomic deprivation and passive smoking have been implicated. Other causes of femoral head A VN must be considered, particularly in bilateral cases ( Table 44.6 ). /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Pathology Once established the process follows a well-described course. The avascular change may a ff ect all or part of the femoral epiphysis. If the avascular bone collapses, this is followed by revascularisation, resorption and fragmentation of the dead ossific nucleus within the cartilaginous femoral head, and finally by reossification and regeneration (‘healing’) of the bony epiphysis. In this respect, Perthes’ disease is a self-limiting condition but, during the collapse and fragmentation phases, femoral head deformity occurs as the cartilage ‘follows’ the Legg, Calvé and Perthes all described osteochondritis of the head of the femur independently in 1910. Arthur Thornton Legg , 1874–1939, orthopaedic surgeon, The Children’s Hospital, Boston, MA, USA. Jacques Calvé , 1875–1927, orthopaedic surgeon, La Fondation Franco-Americaine, Berck Plage, Pas-de-Calais, France. Georg Clemens Perthes , 1869–1927, Professor of Surgery , Tübingen, Germany . ). shape of the reossifying epiphysis. This change in shape is irreversible and has a permanent e ff ect on hip function. Diagnosis The history , clinical examination and anteroposterior and ‘frog’ lateral pelvic radiographs make the diagnosis. An intermittently painful hip (or knee) with a limp and irritability or restriction of hip movements requires investigation. The radiographic features vary with the disease stage and may not correlate with the clinical condition ( Figure 44.21 ).

TABLE 44.6 Causes of avascular necrosis of the femoral head. Steroids Infection/surgery/previous injury Perthes’ disease Sickle cell disease Hypothyroidism Multiple epiphyseal dysplasia will show AVN-like appearances in both femoral heads AI CE (b) Figure 44.19 Anteroposterior pelvic radiographs demonstrating the acetabular index (AI) and the centre–edge (CE) angle: (a) normal hips; (b) the left hip shows residual dysplasia. The AI is increased when compared with the normal right hip. The left CE angle would be smaller too, but it has not been measured on this radiograph.

Management The prognosis, and hence the management, is influenced by the extent of A VN and the degree of collapse. The Herring classification is popular but can only be applied when the head is in the fragmentation phase. If the anterolateral portion of the head is preserved, the prognosis is good. Treatment aims to minimise femoral head deformity and the risk of secondary acetabular dysplasia by maintaining a good range of joint movement with analgesia and physio y . The use of crutches and/or wheelchairs is discour therap aged because they promote a flexion/adduction posture. Brace management does not alter the natural history . The role of operative treatment is controversial. Surgery ormed early to prevent deformity secondary to can be perf te to ‘salvage’ a poor mechanical femoral head collapse or la situation when deformity is limiting movement ( Table 44.7 Summary box 44.9 Legg–Calvé–Perthes disease /uni25CF /uni25CF /uni25CF /uni25CF

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Figure 44.20 Anteroposterior pelvic radiograph of a 7-year-old girl who developed avascular necrosis secondary to a postoperative wound infection following a closed reduction of her dislocated left hip. Note the destruction of the femoral head and the proximal femoral physis, so that the femoral neck is short and the greater trochanter relatively high (arrow). Most common in boys aged 4–7 years AVN leads to femoral head collapse; the return of the blood supply heralds the resorption and reossi /f_i cation phases that allow the femoral head to ‘heal’ The prognosis is better in younger children (and in boys), who have more remodelling potential before skeletal maturity Management aims to maintain femoral head sphericity Treatment may be non-surgical (to maximise range of movement) or surgical (early for containment or late for ‘salvage’) (b) Figure 44.21 Anteroposterior pelvic radiographs of Perthes’ disease demonstrating whole head involvement: (a) right-sided disease; the process is in an early phase and the area of dense necrotic bone is visible; (b) there has been collapse and fragmentation. The Herring classi /f_i cation relates to the height of the lateral pillar (lateral portion of the epiphysis) in the fragmentation phase of the disease.

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Figure 44.20 Anteroposterior pelvic radiograph of a 7-year-old girl who developed avascular necrosis secondary to a postoperative wound infection following a closed reduction of her dislocated left hip. Note the destruction of the femoral head and the proximal femoral physis, so that the femoral neck is short and the greater trochanter relatively high (arrow). Most common in boys aged 4–7 years AVN leads to femoral head collapse; the return of the blood supply heralds the resorption and reossi /f_i cation phases that allow the femoral head to ‘heal’ The prognosis is better

in younger children (and in boys), who have more remodelling potential before skeletal maturity Management aims to maintain femoral head sphericity Treatment may be non-surgical (to maximise range of movement) or surgical (early for containment or late for ‘salvage’) (b) Figure 44.21 Anteroposterior pelvic radiographs of Perthes’ disease demonstrating whole head involvement: (a) right-sided disease; the process is in an early phase and the area of dense necrotic bone is visible; (b) there has been collapse and fragmentation. The Herring classi /f_i cation relates to the height of the lateral pillar (lateral portion of the epiphysis) in the fragmentation phase of the disease.

# METABOLIC BONE DISEASE Rickets

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In rickets, the primary problem is inadequate mineralisation of growing bone ( Table 44.5 ). In severe cases the classic radiographic features are seen at all physes with significant deformity ( Figure 44.11 ). Medical treatment improves mineralisation and deformity corrects with growth. Once the medical condition has stabilised, surgery may be indicated to treat residual limb deformity . Guided growth techniques are often used in preference to osteotomies. Burrill Bernard Crohn , 1884–1983, gastroenterologist, Mount Sinai Hospital, New Y ork, NY , USA, described regional ileitis in 1932.

# TABLE 44.5 Common causes of rickets. Nutritional Reduced intake of vitamin D and calcium Environmental Inadequate exposure to sunlight Gastrointestinal Crohn’s disease, gluten-sensitive enteropathy disease Genetic X-linked hypophosphataemia (excess

# FGF23 production) Renal disease End-stage renal failure, renal tubular anomalies Secondary hyperparathyroidism may be present Figure 44.10 Standing leg length/alignment radiograph of a child with polyostotic /f_i brous dysplasia. The diaphyseal lesions have a ‘ground

glass’ appearance. The bones ar e often deformed and the limb may be short, as seen on the right. The femur has fractured pr eviously and one intramedullary nail remains in place. Figure 44.11 Radiographs in cases of rickets demonstrate widened physes with cupped, /f_l ared metaphyses.

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# NEUROMUSCULAR CONDITIONS

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Joint stability and limb function rely on the complex integra - tion of the musculoskeletal and neurological systems. Damage to either leads to one of several conditions linked only by the during the period of skeletal growth. Management is directed at helping the child cope with their disability , minimising further deterioration and maximising function. It is important to have an understanding of what the damage is and what the future holds ( Table 44.14 ). Spina bifida and polio are classic lower motor neurone lesions, whereas cerebral palsy and head injuries a ff ect upper motor neurones and the higher centres. There are often other disabilities such as blindness, epilepsy and intellectual di ffi cul ties to consider. /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF In children, even if the initial insult to the neuromuscular system is non-progressive, the e ff ects of the insult change with growth. Damage at any level of the neuromuscular system leads to an alteration in tone and muscle imbalance associ ated with decreased control of movement. Abnormal muscle pull, particularly in combination with the e ff ects of gravity , alters bone growth, leading to deformity and joint contrac ture. Muscles are relatively weak and, with body growth and a weight increase, they are no longer strong enough to control a heavier limb, particularly when deformity means they are working a t a mechanical disadvantage. A multidisciplinary approach to management is essential. Physiotherapy and orthotic management may reduce the need for surgical intervention and postoperatively they ensure that the surgical benefits are maximised. In conditions such as Duchenne muscular d ystrophy there is substantial evidence for the benefits of certain surgical procedures; however, in other conditions (cerebral palsy) there are fewer such long-term validated studies. /uni25CF /uni25CF Guillaume Benjamin Amand Duchenne (Duchenne de Boulogne) held a hospital appointment. ment, muscle length and tendon excursion. This is easier to achieve in patients with a flaccid paralysis or low tone. The maintenance of muscle strength is also important. The use of splints, positioning techniques and seating and sleeping sys - tems is common with the aim of preventing fixed contractures. Surgery has a valuable role in the management of selected patients ( Table 44.15 ). The surgeon must understand that altering ankle posture may a ff ect knee and hip posture/function and vice versa. - The patient m ust have the intellectual ability and motivation to recover fr om the surgical procedure. Some of the factors mentioned previously ( Table 44.14 ) must be considered in any holistic approach to the patient. Summary box 44.19 Principles of treatment of neuromuscular conditions /uni25CF /uni25CF /uni25CF /uni25CF - -

TABLE 44.14 Factors to be considered in the assessment of a neuromuscular disability. Is the insult to the neurological system progressive or non- progressive? Is it located centrally or peripherally? Is it general or focal? Is it associated with other abnormalities or not? If the insult is not neurological, is it myopathic? TABLE 44.15 General types of surgical procedure that may be

considered in the management of a patient with a neuromuscular condition. Surgical procedure Lengthening of the muscle–tendon unit Tendon transfer Improves functional movement; rebalances muscle forces, after Release of joint contracture; correction of bony deformity Fuse/stabilise/relocate joints Neurological procedures: selective dorsal rhizotomy (SDR) intrathecal baclofen pumps (ITB) Leg equalisation procedures A neurological defect, whether progressive or not, may cause progressive deformity with skeletal growth A multidisciplinary approach is essential Primary therapy aims to maintain range of movement and prevent /f_i xed contractures with an emphasis on managing tone and position Surgery has a limited role in the management of neuromuscular conditions

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# NORMAL VARIANTS

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Many normal variants of growth and development cause parental concern. The common problems relate to tripping and falling, an intoeing gait, bowlegs, knock knees and flat feet. In general, if they are symmetrical, symptom-free and in an - otherwise normal child, they require no intervention. If the are functional problems, further investigation may be required. NORMAL VARIANTS

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# Non-accidental injury

Non-accidental injury

No child is exempt but some children are at particular risk, including those under 3 years of age, those with disabilities and those in families su ff ering socioeconomic deprivation. A careful clinical assessment is required ( Figure 44.43 and Table 44.20 ). Characteristic patterns should alert the clinician to the possibility of non-accidental injury (NAI) ( Table 44.21 ). NAI occurs in di ff erent forms: emotional, physical, sexual and neglect. When suspected it should be discussed with child safeguarding teams. All injuries should be documented carefully . It may be prudent to admit the child until further checks have been made. /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

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TABLE 44.20 Factors that raise concern in the clinical assessment of suspected non-accidental injury. History Delay in seeking medical advice Variable story Mechanism inconsistent with injury pattern Examination Unexpected bruising to the buttocks/back of legs ‘Finger-mark’ bruises Bruises of various ages Burns, deep scratches, etc. TABLE 44.21 Fracture patterns with a high speci /f_i city for non-accidental injury. Multiple fractures at different stages of healing/old fractures Posterior rib fractures Corner or bucket-handle metaphyseal fractures Scapular fractures Any fracture in a child below walking age

Bulstrode CJK, Wilson-MacDonald J, Eastwood DM et al Oxford textbook of trauma and orthopaedics , 2nd edn. Oxford: Oxford University Press, 2017. Flynn JM, Weinstein SL (eds). Lovell and Winters pediatric orthopaedics vols 1 and 2, 8th edn. Philadelphia, PA: Lippincott, Williams & Wilkins, 2020. org/pediatric-orthopaedics (accessed 25 March 2021). . (eds). Kocher MS, Mandiga R, Zurakowski D et al . Validation of a clinical prediction rule for the di ff erentiation between septic arthritis and transient synovitis of the hip in children. J Bone Joint Surg Am , 2004; 86 (8): 1629-35. Non-accidental injury

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# OF THE SKELETON

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Although many skeletal abnormalities are identified ante - natally or at birth, others become apparent with growth. Skeletal disorders are often linked to focal or generalised soft-tissue abnormalities; the presence of a skin dimple or a ‘featureless’ limbs of a child with arthrogryposis multiplex congenita (AMC) ( Table 44.4 and Figure 44.5 ). /uni25CF /uni25CF /uni25CF /uni25CF Many anomalies require little treatment and cause minimal functional disability whereas others, such as proximal femoral focal deficiency (PFFD), pose considerable challenges to both the patient and their doctors. In these cases the functional and cosmetic needs of the child and family must be balanced against available resources and expertise ( Figure 44.6 ). Despite advances in limb reconstruction techniques there are few high-quality data from skeletally mature patients to support their widespread use. Concurrently , significant advances are occurring with amputation prosthetics, which may result in better patient-reported outcome scores (PROMSs), particularly in certain regions.

TABLE 44.4 Classi /f_i cation of congenital limb malformations. Category Example Failure of formation of parts Transverse Congenital amputation of the forearm/lower limb Longitudinal Fibula hemimelia Failure of differentiation Radioulnar synostosis; vertebral body fusion Duplication Extra digits Overgrowth Gigantism; macrodactyly Undergrowth Congenital constriction Often affects hands/feet with poor band syndrome formation of the digits distally Generalised skeletal Skeletal dysplasia, e.g. abnormalities achondroplasia

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# Osteochondritis dissecans

Osteochondritis dissecans

Osteochondritis dissecans (OCD) a ff ects the lateral aspect of the medial femoral condyle of the distal femur (but also the talus and the humerus). An osteochondral fragment becomes partially or completely separated from the joint surface. Magnetic resonance imaging (MRI) is the best method for demonstrating the site, extent and stability of the lesion. In mild cases, the osteochondral fragment remains attached and heals, particularly if treated early with activity modification. If it detaches, partially or completely , mechanical symptoms Robert Bailey Osgood , 1873–1956, Professor of Orthopaedic Surgery , Harvard University Medical School, Boston, MA, USA. Carl Schlatter , 1864–1934, Professor of Surgery , Zurich, Switzerland. Osgood and Schlatter described osteochondritis of the tibial tubercle independently in 1903. fixation of the fragment or to remove a loose body . Y ounger c hildren have a better prognosis. Osteochondritis dissecans

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# Osteomyelitis

Osteomyelitis

As with septic arthritis, bone infection is usually caused by haematogenous spread. Infection starts in the metaphyses of long bones, where the slow flow through the looped vessels combined with microtrauma encourages seeding of infection during a bacteraemia ( Figure 44.41a ). Inflammation follows

# Figure 44.39 Ultrasound scan of a hip joint. A large effusion is dis

tending the joint capsule. The dotted line represents the distance between the femoral neck and the joint capsule.

and, if purulent material forms, the pressure e ff ects secondary to the abscess formation lead to bony destruction. Pus passes through cortical bone and when it does so it elevates the strong periosteum, which may render the cortical bone avascular. As in cases of trauma or tumour, the periosteal elevation is a potent stimulus for new bone formation. In cases of untreated or chronic infection this new bone or involucrum may surround the dead bone, the sequestrum, leading to a ‘bone-within-a bone’ appearance ( Figure 44.41b ). The presentation and investigation of osteomyelitis can be similar to those for joint sepsis. The di ff erentiation between the two may be di ffi cult and a sympathetic joint e ff usion may occur with metaphyseal osteomyelitis. Thus, if there are no organ isms seen on microscopy of a joint aspirate, the possibility of a coexisting osteomyelitis must be considered. The metaphysis of a long bone may be intracapsular and infection may spread - easily into the joint once the periosteum is breached. In the neonate, proximal femoral osteomyelitis and septic arthritis are essentially the same condition ( Figure 44.41c ). - General principles for the management of infection should be followed. Pus needs to be drained but otherwise the treatment is medical. Debate continues over the duration of

# (b) Figure 44.40 Septic arthritis of the right hip: (a) anteroposterior

(AP) pelvic radiograph with subtle signs of right hip subluxation; pelvic radiograph 6 months later showing destruction of the femoral head secondary to late treatment of a septic joint. sinusoidal colonies vessel Trauma Ve in Artery (with bacteraemia) (b) Dead and dying bone s Bone absces Periosteum Pus Cortex Medullary cavity (c) Joint cavity (b) AP Bone absces s Periosteum Figure 44.41 (a–c) Diagrams illustrating the pathology underlying the development of osteomyelitis. The longer the infection goes

# untreated the greater the destruction, with the possibility of sequestrum forma

tion and secondary joint infection.

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Occurs by haematogenous spread, enhanced by microtrauma In untreated and/or chronic osteomyelitis, new involucrum envelops dead sequestrum In addition to antibiotics, treatment consists of: Rest/splintage of affected limb Analgesia A joint effusion may be sympathetic, a primary septic arthritis or caused by direct spread from the adjacent metaphyseal infection Treatment involves: Drainage of pus when present Appropriate and often prolonged antibiotic therapy: parenteral and then oral Treatment of the underlying condition, e.g. nutritional de /f_i ciency, sickle cell disease

Osteomyelitis

As with septic arthritis, bone infection is usually caused by haematogenous spread. Infection starts in the metaphyses of long bones, where the slow flow through the looped vessels combined with microtrauma encourages seeding of infection during a bacteraemia ( Figure 44.41a ). Inflammation follows

# Figure 44.39 Ultrasound scan of a hip joint. A large effusion is dis

tending the joint capsule. The dotted line represents the distance between the femoral neck and the joint capsule.

and, if purulent material forms, the pressure e ff ects secondary to the abscess formation lead to bony destruction. Pus passes through cortical bone and when it does so it elevates the strong periosteum, which may render the cortical bone avascular. As in cases of trauma or tumour, the periosteal elevation is a potent stimulus for new bone formation. In cases of untreated or chronic infection this new bone or involucrum may surround the dead bone, the sequestrum, leading to a ‘bone-within-a bone’ appearance ( Figure 44.41b ). The presentation and investigation of osteomyelitis can be similar to those for joint sepsis. The di ff erentiation between the two may be di ffi cult and a sympathetic joint e ff usion may occur with metaphyseal osteomyelitis. Thus, if there are no organ isms seen on microscopy of a joint aspirate, the possibility of a coexisting osteomyelitis must be considered. The metaphysis of a long bone may be intracapsular and infection may spread - easily into the joint once the periosteum is breached. In the neonate, proximal femoral osteomyelitis and septic arthritis are essentially the same condition ( Figure 44.41c ). - General principles for the management of infection should be followed. Pus needs to be drained but otherwise the treatment is medical. Debate continues over the duration of

# (b) Figure 44.40 Septic arthritis of the right hip: (a) anteroposterior (AP) pelvic radiograph with subtle signs of right hip subluxation; pelvic radiograph 6 months later showing destruction of the femoral head secondary to late treatment of a septic joint. sinusoidal colonies vessel Trauma Ve in Artery (with bacteraemia) (b) Dead

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# Other foot and ankle conditions

Other foot and ankle conditions

Most postural deformities such as metatarsus adductus and calcaneovalgus feet improve spontaneously . Congenital vertical talus (CVT) is rare and fre - quently associated with neuromuscular conditions such as ‘rocker-bottom’ foot with dorsal dislocation of the navicular on the talus ( Figure 44.31 ). A ‘reverse’ Ponseti method with a limited surgical approach to reduce and Kirschner (K)-wire the talonavicular joint and a percutaneous Achilles tenotomy may be preferable to more extensive surgical releases. In tarsal coalition there is failure of segmentation of adjacent tarsal bones. School-aged children present with hindfoot pain and recurrent ankle sprains. The most common coalitions are talocalcaneal and calcaneonavicular ( Figure 44.4 ). Radiographs, computed tomography (CT) or MRI are used to confirm the diagnosis. Treatment is initially conservative, but if the coalition requires surgical excision this should be performed before degenerative changes develop. Other causes of foot pain in children include the osteo chondroses, in which the radiographic changes are similar to A VN. These ‘heal’ but the change in shape of the a ff ected bone may lead to secondary joint sti ff ness and loss of function. (Sim ilar ‘diseases’ a ff ect the lunate [Kienböck’s] and the capitellum of the humerus [Panner’s].) /uni25CF Freiberg’s osteochondrosis presents with forefoot pain and avascular change in the second metatarsal head. Symptomatic bony spurs and osteochondral fragments may need excision but often it is asymptomatic and seen as an incidental finding on a radiograph. /uni25CF Köhler’s disease presents with dorsal forefoot pain and swelling in young children. The navicular becomes avascu lar with alteration in the ossification process. /uni25CF Sever’s disease (enthesopathy of the calcaneal apoph ysis) presents with heel pain related to activity . Tightness in the calf muscle complex may be a contributing factor. The ‘features’ on a radiograph are, in fact, part of normal growth and de velopment. Flexed, medially curved ‘curly toes’ are common and rarely need treatment. Strapping is ine ff ective. Flexor tenotomy is used when there ar e symptoms or cosmetic concerns. Summary box 44.14 Other foot and ankle conditions /uni25CF /uni25CF /uni25CF /uni25CF

CVT – presents as ‘rocker-bottom’ foot Tarsal coalition – presents as a stiff, painful /f_l at foot Osteochondroses – almost always self-limiting Curly toes are common – most do not need treatment

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# PAIN Congenital deformities

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Congenital vertebral deformities are failures either of formation (a hemivertebra) or of segmentation (unilateral or bilateral fusions or bars). The clinical result is usually a scoliosis ( Figure 44.34 ). Treatment should be based on the potential for curve progression. When a kyphosis develops, progressive neurological deficit is common. Bracing is ine ff ective for congenital vertebral deformities. PAIN Congenital deformities

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# Polio

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# Postural abnormalities

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Many babies are subjected to moulding pressures in utero . At birth they exhibit ‘postural abnormalities’, such as torticollis, calcaneovalgus feet and plagiocephaly , which improve with time and/or stretching exercises.

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# Radial club hand

Radial club hand

This longitudinal failure of formation is commonly associ - ated with other malformations, for example as part of the V ACTERL (abnormal vertebrae, anus, cardiovascular system, trachea, oesophagus, renal system and limb buds) syndr ome. The clinical problem depends on whether the thumb is present and functional ( Figure 44.32 ). Treatment is a balance of conservative measures, including physiotherapy and splinting, and judicious surgery to centralise and stabilise the hand and wrist on the single bone forearm. Thumb reconstruction may ) but be technically challenging. In later childhood, forearm length - ening may be considered.

affecting the hand. Anomaly De /f_i nition Treatment Extra/accessory Excise/amputate when digits necessary Failure of separation Syndactyly Separation with/ of digits without skin grafting for functional or cosmetic reasons Trigger thumb Release of the A1 (digit) pulley of the /f_l exor tendon sheath Surgical treatment of Abnormal angulation Clinodactyly the delta phalanx if of the digit in the (usually the /f_i fth deformity is progressive radioulnar plane digit) or interfering with hand function Splinting/ Fixed /f_l exion Camptodactyly physiotherapy; surgery deformity of (usually the /f_i fth rarely indicated the proximal digit) interphalangeal joint Figure 44.32 Anteroposterior radiograph of a radial club hand demon strating a short radius, a deformed ulna and an absent thumb.

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# Radioulnar synostosis

Radioulnar synostosis

Failure of proximal separation of the embryonic radius and ulna means that the forearm has no ability to pronate/supi nate. The hand, on the end of the forearm, is therefore in a fixed position along the arc from full pronation–neutral–full supination. The child presents if this fixed position results in functional di ffi culties. Osteotomy of the forear m bones changes the fixed position (for example, from pronation to neutral) but does not restore movement. The choice of the postoperative position depends on hand dominance, cultural considerations and functional demands. Undoing the synosto sis is not successful. Mary Clayton Holt , 1924–1993, cardiologist, The London Hospital for Women and Children, London, UK. Samuel Oram , 1913–1991, cardiologist, King’s College Hospital, London, UK. Holt and Oram described this syndrome in a joint paper in 1960.

# (b) Figure 44.33 Radial head dislocation: (a) lateral radiograph of a forearm showing a proximal radioulnar synostosis with a congenital posterolateral dislocation of the radial head. Note the underdeveloped radial head and neck and compare with (b) , a lateral radiograph of a traumatic

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# Scoliosis

Scoliosis

The term ‘scoliosis’ describes spinal deformity in three planes: lateral curvature is the most obvious deformity while the rota tional component is most apparent in forward flexion when the rib asymmetry creates a ‘rib hump’ ( Figure 44.35 ). The cause may be idiopathic, neuromuscular, related to a syndrome or congenital. Both the aetiology and the age of onset a ff ect the natural history ( Table 44.12 ). In general, the earlier the onset, the more likely the deformity is to be progressive. As most lung development occurs in early childhood, the management of early-onset scoliosis must preserve growth: casting techniques or the use of ‘growing rods’ may be appropriate. The adolescent idiopathic curve is the most common, a ff ecting girls more than boys. Idiopathic scoliosis is generally not painful and, therefore, in the presence of significant pain tumour and infection must be excluded. The Cobb angle is a radiological measurement that defines se verity and guides treatment ( Figure 44.36 ). Curves <20° do not need treatment, progressive curves of 25–40° may be braced and those >40° are considered for surgery , which involves instrumenting and fusing the spine (see also Chapter 37 ).

TABLE 44.12 Classi /f_i cation of idiopathic scoliosis. Type Age at onset Early onset <10 years Adolescent 11–18 years Adult Onset at maturity

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# Septic arthritis

Septic arthritis

Joint infection is usually secondary to haematogenous spread but direct inoculation can occur, for example during a neonatal venepuncture. Diagnosis can be di ffi cult in the very young and in those presenting with overwhelming sepsis. Neonates, children with immunocompromise and those with sickle cell

Figure 44.38 Anteroposterior pelvic radiograph of a child with spastic cerebral palsy. The right hip is dislocated: none of the head lies medial to the vertical Perkins line. The acetabulum is dysplastic. The left hip , knee is in abduction. There is often a ‘windswept’ appearance with one leg stiff in abduction and the other stiff in adduction. The red line demon

strates the pelvic obliquity; many children also have a scoliosis. Note the signi /f_i cant constipation; this can cause signi /f_i cant pain, which will increase spasm and increase the pain still further.

the di ff erentiation between joint sepsis and transient synovitis of the hip can also be di ffi cult. Classically , the child presents with pain, fever and a reluctance to move the joint; in the lower limb, this implies a reluctance to weight bear. On examination, local tender ness and painful restriction of movement ar e apparent and in superficial joints inflammation may be obvious, with a hot, swollen joint. Investigations include FBC, ESR, CRP and blood cul tures. Plain radiographs help e xclude other diagnoses and may identify osteomyelitis. Ultrasound scans of deep joints, such as the hip, will identify joint e ff usions ( Figure 44.39 ). MRI is considered the investigation of choice b ut this resource is not available to all (in a timely manner) and, in young children, it requires a general anaesthetic. Good clinical skills, regu lar patient review and a high index of suspicion are still the most valuable tools. Four clinical predictors can di ff erentiate between septic arthritis and transient synovitis ( Table 44.17 Pus in a joint is destructive: the proteases produced by leu kocytes destroy both the bacteria and the collagen matrix of the articular cartilage. A VN may occur secondary to pressure e ff ects or ischaemic infarction. The treatment of a presumed septic arthritis therefore requires the prompt removal of pus from the joint and appropriate adequate antibiotic therapy . Pain relief and rest are also important, as are the general health and nutrition of the patient. The joint is aspirated and, if pus is confirmed, a formal washout is mandatory; standard teaching states that the joint must be opened, irrigated and free drainage encouraged via the capsulotomy . Recent literature supports repeated aspiration/irrigation via a large-bore cannula or a small arthroscope for all joints except the hip. Antibiotic usage is guided by local hospital policy , the source of the infection, the Gram stain and, in due course, the culture and sensitivity of the organism identified. Joint instability , particularly in the hip joint ( Figure 44.40 ), may require the reduced joint to be splinted while the inflammatory process settles. /uni25CF /uni25CF /uni25CF /uni25CF Hans Christian Joachim Gram , 1853–1938, Professor of Pharmacology (1891–1900) and of Medicine (1900–1923), Copenhagen, Denmark, described this method of staining bacteria in 1884. cus aureus . Streptococcal infection is also common and other organisms are more prevalent in certain age groups, e.g. the neonate, in certain conditions, e.g. sickle cell disease, or in cer - tain countries. The Haemophilus influenzae type B (Hib) vaccine - has essentially eliminated H. influenzae as a cause of infection, but in some countries Kingella kingae has taken its place. Improvement is judged clinically and by monitoring the inflammatory markers. Reaccumulation of pus does occur and - must be suspected and treated promptly if the child fails to improve. Summary box 44.21 Septic arthritis - /uni25CF /uni25CF ). /uni25CF - /uni25CF /uni25CF

TABLE 44.17 Septic arthritis. (a) The clinical predictors of Kocher et al . (2004) for the diagnosis of septic arthritis: History of fever >38.5°C Non-weight-bearing Erythrocyte sedimentation rate >40 /uni00A0 mm/h 9 White cell count >12 /uni00A0×/uni00A0 10 /L (b) The value of the clinical predictors of Kocher et al . in determining the likelihood of a joint being septic: Number of positive Predicted probability of joint sepsis predictors 0 2.0% 1 9.5% 2 35.0% 3 72.8% 4 93.0% Diagnosis is dif /f_i cult in neonates and the immunocompromised Typical presentation is pain, fever and a reluctance to move the joint or weight bear Investigations should include FBC, ESR, CRP , blood cultures and appropriate imaging studies, combined with astute clinical skills Pus in a joint destroys articular cartilage and causes avascular necrosis of intra-articular epiphyses Treatment is prompt removal of pus, appropriate antibiotic therapy, pain relief and splintage

Septic arthritis

Joint infection is usually secondary to haematogenous spread but direct inoculation can occur, for example during a neonatal venepuncture. Diagnosis can be di ffi cult in the very young and in those presenting with overwhelming sepsis. Neonates, children with immunocompromise and those with sickle cell

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# Slip of the capital (upper) femoral epiphysis (SCF

Slip of the capital (upper) femoral epiphysis (SCFE/SUFE)

The physis connects the proximal femoral epiphysis (the femoral head) to the metaphysis (femoral neck). In certain physiological or pathological conditions a ‘stress fracture’ through the physis allows the epiphysis to displace as it would with an intracap - sular femoral neck fracture, so the leg lies short and externally /uni25CF /uni25CF /uni25CF /uni25CF rotated. There is painful limitation of hip movement. Hilton’s law , which states that a joint is supplied by the same nerves as the muscles that move the joint, explains why many children present with knee pain although the pathology is in the hip. Incidence and aetiology SCFE is rare, with an incidence of approximately 5 per 100 /uni00A0 000 population. Boys are a ff ected most commonly . The peak incidence is related to the start of puberty and hence is earlier in girls. As a result of hormonally stimulated growth, the strength of the physis, its resistance to shear and its orien tation are reduced. The hip is therefore ‘at risk’ and normal forces, exacerbated by obesity and repetitive minor trauma, precipitate a slip. Other conditions such as hypothyroidism, renal failur e and previous radiotherapy treatment (local or to the pituitary) also increase the risk. Diagnosis The diagnosis is suggested by the history and examination and confirmed on plain radiographs ( Figure 44.22 ). Displacement is often more obvious on a lateral view and the diagnosis can be missed if only the anteroposterior radiograph is checked. Classification A SCFE can be classified according to three parameters: timing, severity and stability . The onset of symptoms divides slips into those that are acute, chronic or acute on chronic. Slip severity is assessed on the lateral radiograph in terms of percentage uncovering of the metaphysis ( Table 44.8 ) or by measuring John Hilton , 1805–1878, surgeon, Guy’s Hospital, London, UK. Wayne O Southwick , 1923–2016, American surgeon and academic, first chairman of the Department of Orthopaedics and Rehabilitation, Yale University , New Hav en, CT , USA, from 1958 to 1979. Randall Loder , contemporary , Professor of Orthopaedic Surgery , Philadelphia, PA, USA. the slip angle of Southwick ( Figure 44.23 ). An unstable slip is defined by Loder as one in which the patient cannot bear weight on the limb. -

Timing Type of procedure Comments Early Femoral osteotomy Varus and derotation Consider an opening wedge osteotomy to maintain length Innominate osteotomy Shelf acetabuloplasty Intermediate Arthrodiastasis Hinged distraction to allow movement, primarily /f_l exion/extension Late Femoral osteotomy Valgus With extension to undo a /f_i xed /f_l exion deformity or /f_l exion to remove the anterior bump from impinging on the acetabulum Arthrotomy To remove osteochondral fragments Head–neck osteoplasty After physeal closure Trochanteric epiphysiodesis or Epiphysiodesis not effective after age 7–8 To improve lever arm function distal transfer years Contralateral limb Distal femoral epiphysiodesis Aim To cover (‘contain’) the vulnerable femoral head To reduce deforming pressures on the femoral head To improve joint congruity and hence function; to improve joint mechanics To improve head shape by reducing femoroacetabular

impingement and increasing head/neck offset To reduce leg length discrepancy and effects on hip joint mechanics Figure 44.22 Anteroposterior pelvic radiograph demonstrating a mild slip of the upper (capital) femoral epiphysis on the left side. A line drawn along the upper margin of the femoral neck should transect the femoral head (right side); if it does not do so (left side) a slip is present. There are many other radiographic features that help to con /f_i rm the diagnosis but the changes are often subtle and may be seen /f_i rst on the frog lateral view.

Management Following an acute episode the patient is often unable to weight bear and the slip is considered to be unstable . Displacement is often moderate or severe. This situation is equivalent to a displaced intracapsular femoral neck fracture. This means that an acute unstable SCFE is an emergency . The A VN risk is considerable ( Figure 44.24 ). With the reduction in muscle spasm that accompanies a general anaesthetic, a gentle repositioning of the femoral epiphysis occurs as the externally rotated limb is lifted into the neutral position using no force. A capsulotomy reduces the tamponade e ff ect on the epiphyseal vessels. T o be e ff ective such treatment should take place within 24 hours of injury . If delayed, the A VN rate may increase. With chronic slips the patient is able to weight bear, albeit with pain, and the slip is stable. Screw or pin fixation relieves pain and movement improves but there will be per manent reduction in abduction, flexion and internal rotation ( Figure 44.25 ). The leg will be slightly short. In the chronic severe slip it may be impossible to place a screw in a satisfactory position centrally within the epiph ysis. Once healed, there may be significant, persistent defor mity leading to restriction of joint movement. In these cases a realignment osteotomy may be considered. As with all osteo tomies, the closer the correction is to the site of deformity , the better the outcome. However, in this situation the centr of rotation of angulation (CORA) for the deformity is at the of the physis; the risk of A VN or chondrolysis may be level unacceptably high with an osteotomy at this level, and so an intertrochanteric osteotomy could be considered. The slipped epiphysis is associated with a ‘cam’ type of femoroacetabular in situ - - - - e impingement and this may require treatment with a head–neck osteoplasty to restore the o ff set between the head and neck. Bilateral slips do occur and prophylactic pinning of the normal but ‘at-risk’ hip may be indicated.

Normal a b c 12° TABLE 44.8 Grading of the severity of slip of the capital femoral epiphysis. Slip severity Metaphysis uncovered (%) Mild <33 Moderate 33–66 Severe

> “ 66 SCFE b a 40° Figure 44.23 The Southwick slip angle c is measured on a lateral radiograph and denotes how far the epiphysis has slipped off the metaphysis. The value on the normal side must be subtracted from the value on the abnormal side to get the true value. SCFE, slip of the capital femoral epiphysis. (a) (b) Figure 44.24 Anteroposterior pelvic radiograph showing a left-sided acute severe unstable slip of the capital femoral epiphysis: (a) at presentation; (b) following partial repositioning and /f_i xation with a cannulated screw. When this heals, because of the incomplete reduc

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Summary box 44.10 Slip of the upper (capital) femoral epiphysis /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Figure 44.25 Anteroposterior radiograph showing screw /f_i xation in situ of a case of bilateral chronic slip of the capital femoral epiphysis. Note the position of the screw: the more severe the slip, the more proximal and more anterior the screw entry point must be on the femoral neck. Occurs in prepubertal children, boys more commonly than girls Often presents with knee pain, and a short and externally rotated leg Classi /f_i cation systems relate to timing, severity and stability – all affect the prognosis Most slips are pinned in situ with a single screw into the centre of the epiphysis AVN is a feared complication of both the condition and its treatment

Slip of the capital (upper) femoral epiphysis (SCFE/SUFE)

The physis connects the proximal femoral epiphysis (the femoral head) to the metaphysis (femoral neck). In certain physiological or pathological conditions a ‘stress fracture’ through the physis allows the epiphysis to displace as it would with an intracap - sular femoral neck fracture, so the leg lies short and externally /uni25CF /uni25CF /uni25CF /uni25CF rotated. There is painful limitation of hip movement. Hilton’s law , which states that a joint is supplied by the same nerves as the muscles that move the joint, explains why many children present with knee pain although the pathology is in the hip. Incidence and aetiology SCFE is rare, with an incidence of approximately 5 per 100 /uni00A0 000 population. Boys are a ff ected most commonly . The peak incidence is related to the start of puberty and hence is earlier in girls. As a result of hormonally stimulated growth, the strength of the physis, its resistance to shear and its orien tation are reduced. The hip is therefore ‘at risk’ and normal forces, exacerbated by obesity and repetitive minor trauma, precipitate a slip. Other conditions such as hypothyroidism, renal failur e and previous radiotherapy treatment (local or to the pituitary) also increase the risk. Diagnosis The diagnosis is suggested by the history and examination and confirmed on plain radiographs ( Figure 44.22 ). Displacement is often more obvious on a lateral view and the diagnosis can be missed if only the anteroposterior radiograph is checked. Classification A SCFE can be classified according to three parameters: timing, severity and stability . The onset of symptoms divides slips into those that are acute, chronic or acute on chronic. Slip severity is assessed on the lateral radiograph in terms of percentage uncovering of the metaphysis ( Table 44.8 ) or by measuring John Hilton , 1805–1878, surgeon, Guy’s Hospital, London, UK. Wayne O Southwick , 1923–2016, American surgeon and academic, first chairman of the Department of Orthopaedics and Rehabilitation, Yale University , New Hav en, CT , USA, from 1958 to 1979. Randall Loder , contemporary , Professor of Orthopaedic Surgery , Philadelphia, PA, USA. the slip angle of Southwick ( Figure 44.23 ). An unstable slip is defined by Loder as one in which the patient cannot bear weight on the limb. -

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or Epiphysiodesis not effective after age 7–8 To improve lever arm function distal transfer years Contralateral limb Distal femoral epiphysiodesis Aim To cover (‘contain’) the vulnerable femoral head To reduce deforming pressures on the femoral head To improve joint congruity and hence function; to improve joint mechanics To improve head shape by reducing femoroacetabular impingement and increasing head/neck offset To reduce leg length discrepancy and effects on hip joint mechanics Figure 44.22 Anteroposterior pelvic radiograph demonstrating a mild slip of the upper (capital) femoral epiphysis on the left side. A line drawn along the upper margin of the femoral neck should transect the femoral head (right side); if it does not do so (left side) a slip is present. There are many other radiographic features that help to con /f_i rm the diagnosis but the changes are often subtle and may be seen /f_i rst on the frog lateral view.

Management Following an acute episode the patient is often unable to weight bear and the slip is considered to be unstable . Displacement is often moderate or severe. This situation is equivalent to a displaced intracapsular femoral neck fracture. This means that an acute unstable SCFE is an emergency . The A VN risk is considerable ( Figure 44.24 ). With the reduction in muscle spasm that accompanies a general anaesthetic, a gentle repositioning of the femoral epiphysis occurs as the externally rotated limb is lifted into the neutral position using no force. A capsulotomy reduces the tamponade e ff ect on the epiphyseal vessels. T o be e ff ective such treatment should take place within 24 hours of injury . If delayed, the A VN rate may increase. With chronic slips the patient is able to weight bear, albeit with pain, and the slip is stable. Screw or pin fixation relieves pain and movement improves but there will be per manent reduction in abduction, flexion and internal rotation ( Figure 44.25 ). The leg will be slightly short. In the chronic severe slip it may be impossible to place a screw in a satisfactory position centrally within the epiph ysis. Once healed, there may be significant, persistent defor mity leading to restriction of joint movement. In these cases a realignment osteotomy may be considered. As with all osteo tomies, the closer the correction is to the site of deformity , the better the outcome. However, in this situation the centr of rotation of angulation (CORA) for the deformity is at the of the physis; the risk of A VN or chondrolysis may be level unacceptably high with an osteotomy at this level, and so an intertrochanteric osteotomy could be considered. The slipped epiphysis is associated with a ‘cam’ type of femoroacetabular in situ - - - - e impingement and this may require treatment with a head–neck osteoplasty to restore the o ff set between the head and neck. Bilateral slips do occur and prophylactic pinning of the normal but ‘at-risk’ hip may be indicated.

Normal a b c 12° TABLE 44.8 Grading of the severity of slip of the capital femoral epiphysis. Slip severity Metaphysis uncovered (%) Mild <33 Moderate 33–66 Severe

> 66 SCFE b a 40° Figure 44.23 The Southwick slip angle c is measured on a lateral radiograph and denotes how far the epiphysis has slipped off the metaphysis. The value on the normal side must be subtracted from the value on the abnormal side to get the true value. SCFE, slip of the capital femoral epiphysis. (a) (b) Figure 44.24 Anteroposterior pelvic radiograph showing a left-sided acute severe unstable slip of the capital femoral epiphysis: (a) at presentation; (b) following partial repositioning and /f_i xation with a cannulated screw. When this heals, because of the incomplete reduc

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tion it is likely that the metaphysis will impinge on the acetabulum (femoroacetabular impingement) during movement, causing pain and leading to degenerative change.

Summary box 44.10 Slip of the upper (capital) femoral epiphysis /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Figure 44.25 Anteroposterior radiograph showing screw /f_i xation in situ of a case of bilateral chronic slip of the capital femoral epiphysis. Note the position of the screw: the more severe the slip, the more proximal and more anterior the screw entry point must be on the femoral neck. Occurs in prepubertal children, boys more commonly than girls Often presents with knee pain, and a short and externally rotated leg Classi /f_i cation systems relate to timing, severity and stability – all affect the prognosis Most slips are pinned in situ with a single screw into the centre of the epiphysis AVN is a feared complication of both the condition and its treatment

# Spina bifida

Spina bifida

The extent of the disability varies with the level of the lesion: upper motor neurone involvement will produce spasticity while the more classic lower motor neurone lesion produces a flaccid paralysis. Muscle imbalance leads to secondary joint a ff ect the choice of surgical and non-surgical options. Many children require a ventriculoperitoneal (VP) shunt to drain the hydrocephalus that develops following closure of the myelo cele. With growth, a tethered cord or a blocked VP shunt may develop with a deterioration in the neurological picture. (a) (b) (c) Many types of muscular dystrophy exist that vary in severity and distribution of involvement. Surgical intervention aims to - improve quality of life. This is best achieved by operating early to release joint contractures and maintain the ability to walk with a good spinal posture.

70 60 50 40 30 Hip /f_l exion 20 10 0 –10 0 20 40 60 80 100 80 70 60 50 40 30 Knee /f_l exion 20 10 0 0 20 40 60 80 100 25 20 15 10 5 0 –5 –10 Ankle dorsi /f_l exion +ve Ankle plantar /f_l exion –ve –15 –20 –25 0 20 40 60 80 100 Figure 44.37 Gait analysis graphs such as these demonstrate the normal range of joint movements (green band) at the hip (a) (b) and ankle (c) during the stance and swing phases of the gait. The abnormal joint ranges are shown in red (right leg) and blue (left leg) and demonstrate the excessive hip /f_l exion, lack of knee extension and abnormal ankle mechanics associated with the ‘crouch’ gait of a child with cerebral palsy.

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# Spondylolisthesis

Spondylolisthesis

Spondylolysis defines a defect in the pars interarticularis of the vertebra. There are six types including congenital and trau matic. Spondylolisthesis occurs when the upper vertebra slips forward on the lower; it is graded according to the percentage slip , measured by relating the slipped vertebra to the one below ( Table 44.13 ). Mild slips are often asymptomatic and require no treat ment. Treatment (physiotherapy , bracing and surgery) depends on the degree of slip and symptoms; mechanical back pain may respond to conservative methods, but neurological involvement usually requires sur gical intervention. In torticollis the head is tilted towards and rotated away from the tight sternocleidomastoid muscle. Congenital torticollis is usually secondary to intrauterine moulding but may present with a fixed sternocleidomastoid contracture or a palpable ‘tumour’ within the muscle. Most cases resolve with stretching but persistent cases develop facial asymmetr y requiring release of the origin and/or insertion of the sternocleidomastoid muscle. Acquired torticollis is rare and may be caused by inflam - mation/infection, ocular problems, atlantoaxial rotatory sub - luxation or a posterior fossa tumour.

TABLE 44.13 Classi /f_i cation of spondylolisthesis according to severity of the slip. Grade Percentage slip 0 No slip 1 <25 2 26–50 3 51–75 4

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# disease)

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Osteogenesis imperfecta (OI) represents a spectrum of condi tions linked by a qualitative and/or quantitative abnormality of collagen production. Most identified mutations a ff ect the collagen genes. The bone may break easily but it heals promptly and well. All structures containing collagen may be a ff ected, accounting for the ligamentous laxity , blue sclerae and poor teeth in some phenotypes. Cyclical bisphosphonate treatment decreases bone resorp tion and turnover. This reduces bone pain and the fracture rate, promoting weight-bearing mobility and bone strength ( Figure 44.12 ). Following fracture, treatment options range from simple casting techniques to mor e specialised surgical procedures to correct/maintain bony alignment while allowing growth. Intramedullary techniques f or stabilisation of fractures or osteotomies are preferred to plate fixation. Rehabilitation should start promptly . Summary box 44.4 Metabolic bone disease /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Thomas Geo ff rey Barlow , 1915–1975, orthopaedic surgeon, Salford Royal Hospital, Salford, UK. Marius Ortolani , 1904–1987, orthopaedic surgeon, Instituto Provinciale Per L’Infanzia di Ferrara, Italy , described this test in 1937.

Rickets, from nutritional or other causes, is characterised by a failure of bone mineralisation X-linked hypophosphataemic rickets is a dominant condition, affecting boys and girls; in some countries, treatment is with monoclonal FGF23 antibodies In OI: There is defective type I collagen production In severe forms frequent fractures lead to progressive deformity, which in turn increases fracture risk Systemic treatment with bisphosphonates reduces the fracture rate Figure 44.12 Radiograph of a child with osteogenesis imperfecta who has been treated with cyclical bisphosphonates. Multiple growth lines are visible in addition to intramedullary devices in both the femur and tibia.

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