CHAPTER 120
Common Paediatric Orthopaedic Diseases

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Introduction

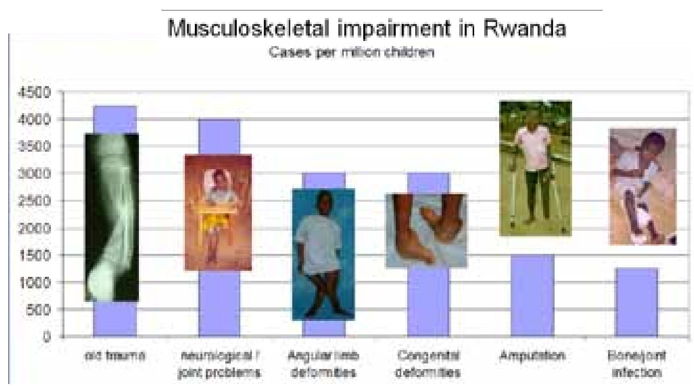
Globally, traumatic injuries are amongst the 10 leading causes of morbidity and mortality in children and adolescents, with most of the burden borne by low- and middle-income countries (LMICs), in particular in Africa, South-East Asia, and the Western Pacific Region.[1] Furthermore, for every injury-related death, an estimated four or five children with severe injury-related disabilities are a significant drain to their families and health systems troubled by already low resources.[2] Traumatic injury is estimated to get worse over time, in particular in the developing world, largely due to the increasing prevalence of road traffic injuries, especially in Africa.[3]

Most posttraumatic disabilities are due to injuries to the upper or lower extremities or to the spine.[4,5] For the sheer volume of musculoskeletal injuries seen everywhere in Africa, rural or urban, any hospital accepting all types of emergencies would require significant orthopaedic surgical services.[6] Furthermore, with more than 50% of the population under the age of 15 years in most developing countries, a specific familiarity with paediatric orthopaedic traumatic injuries would be required.

In most LMICs, very few orthopaedic surgeons are available to deal with these many surgical problems, and even fewer are formally trained *paediatric* orthopaedic surgeons. As such, most orthopaedic surgical services throughout Africa have been provided by general surgeons or orthopaedic clinical officers with some surgical training.[7] With the literature on the efficacy of African traditional bonesetters being limited, and in general negative,[8–10] it is likely that generalist surgeons and nonphysician clinical officers will continue to provide the vast majority of orthopaedic surgery in Africa for the foreseeable future.

Apart from the traumatic orthopaedic problems African children must endure are the additional surgical problems of congenital deformities, infections, and other conditions affecting the musculoskeletal system. Figure 120.1 presents data from Rwanda from what is probably the first national, population-based survey done in sub-Saharan Africa examining the extent of musculoskeletal impairment and treatment needs in children.[11] Beyond the sizeable burden of the acute traumatic issues already mentioned, data extrapolation from the results of this study estimates that 50,000 children in Rwanda are in need of orthopaedic surgery for *old* traumatic problems, neurological impairments, nontraumatic angular deformities (e.g., rickets), congenital anomalies (e.g., clubfoot), and bony infections.

The purpose of this chapter is to introduce newer, evidence-based approaches to some old and persistent orthopaedic surgical problems, not only in sub-Saharan Africa but also throughout the developing world. Bach[7] argues the traditional orthopaedic mantra of "Never close an open fracture nor open a closed one" in African centres, given improvements in sterilisation equipment, orthopaedic implants, availability of antibiotics, and so forth. Furthermore, failures of conservative orthopaedic care can result in significant disability. Utilisation of a public health, evidence-based approach to musculoskeletal surgical problems may reduce the sizeable morbidity of orthopaedic problems in African children. Specific problems discussed will include clubfoot, an approach to open fractures to reduce posttraumatic chronic osteomyelitis, and evidence to support an open surgical approach to supracondylar fractures of the paediatric elbow. Also addressed are angular deformities related to polio and nutritional rickets.



Source: Atijosan O, Simms V, Kuper H, Rischewski D, Lavy C. The orthopedic needs of children in Rwanda: results from a national survey and orthopaedic service implications. J Pediatri Orthop 2009; 29:948–951.

Figure 120.1: The musculoskeletal problems that exist beyond the acute orthopaedic trauma needs for children in Rwanda. A major part of a public health–oriented, evidence-based approach to surgical problems is the need for accurate data to plan health services.

Congenital Idiopathic Clubfoot in Africa

Introduction

Clubfoot (CF) is perhaps the most prevalent, congenitally disabling musculoskeletal problem in the world today, leading to a lifetime of unnecessary morbidity if untreated. This condition always requires treatment, and in the past required a major surgical procedure from an experienced paediatric orthopaedic surgeon, with long-term follow-up. With few experienced surgeons and impoverished conditions, most CF in the developing world has been neglected, with resultant major disability. Over the last two decades, however, the developed world's evidence based movement in orthopaedic surgery has identified an essentially nonoperative clinical approach to CF called the Ponseti method.[12–14] An independent critical review of the available literature supports the Ponseti approach, and in Africa, Tindall and colleagues[15] have shown that even trained clinical officers (nondoctors, not just non-orthopaedists) can successfully manipulate idiopathic clubfeet using the Ponseti technique in Malawi. Similar findings elsewhere in Africa have demonstrated that a physiotherapist-delivered Ponseti programme can effectively manage CF in low-resource settings.[16]

Although not without some problems, the Ponseti method may significantly reduce the burden of this major orthopaedic disabler in many LMICs by reducing the need for major operative interventions.

Demographics

Clubfoot deformity is the most common congenital cause of ambulatory disability in the developing world, with a worldwide incidence of approximately 1 in 1,000 live births (1 in 500 in Malawi), the male-to-female ratio is 2:1, and it is bilateral in 50% of cases.[17] Omololu et al.[18] has shown, by a prospective study at the University of Ibadan, that the most common congenital orthopaedic malformations—more than 50% of all congenital musculoskeletal (MSK) malformations—were CF, with hip dysplasia accounting for only 2.2 % of all cases, for comparison.

Aetiology/Pathophysiology

Many theories exist regarding the aetiology of clubfoot, but none have been proven. These theories cite foetal development arrest, contracting fibrosis, and myogenic and neurogenic causes.

Clinical presentation

History

Clubfoot is not infrequently a component of an underlying syndrome, and therefore a complete history and physical are required. CF, for example, can be associated with hand anomalies, certain forms of dwarfism, arthrogryposis, myelomeningocele, and many other afflictions. Furthermore, the spine should be examined for evidence of spinal dysraphism at the base of the spine, such as a hairy tuft, haemangioma, lipoma, among others. A unilateral cavus foot, especially one that was not as evident at birth but has *developed* over time, could be secondary to an intraspinal condition such as a diastomatomyelia. A neurological examination is normal in idiopathic clubfoot.

Note that a significant genetic component exists for the transmission of CF.

Physical examination

The CF deformity is present and obvious at birth, and it is usually not a difficult clinical diagnosis. CF presents as a complex, three-dimensional congenital deformity that includes the components of hind-foot equinus, hind-foot varus, and mid-foot adductus and medial rotation (Figures 120.2 and 120.3). Another name for CF frequently seen in the literature is congenital talipes equinovarus (CTEV or TEV). It is the equinus component (the tight Achilles tendon) that differentiates it from severe metatarsus varus of the midfoot, another congenital foot anomaly evident at birth.

The CF deformity varies in severity, with classic or "idiopathic" clubfoot being most common. There is generalised hypoplasia of the entire leg on a limb affected with CF. The hind-foot, in particular the talus, is most dysplastic. The talar neck is shortened and angled medially and plantar-ward. The navicular articulates with the deformed medial head of the talus. All the hindfoot and midtarsal bones are hypoplastic, and the cartilage, ligaments, and muscles of the foot are hypoplastic and contracted.

The example of idiopathic clubfoot deformity in the newborn shown in Figure 120.2 is suitable for management with the Ponseti method. Treatment should begin immediately for best clinical outcomes. In contrast, neglected clubfoot is unsuitable for the Ponseti method of treatment and requires complex reconstructive surgery by an experienced surgeon. Functionality of the individual patient must be assessed carefully as it may be prudent to not submit the patient to difficult and somewhat risky surgical procedures. The child shown in Figure 120.3 would require several staged operations on each foot, and before this, he would likely need soft tissue releases for chronic knee contractures. This amount of surgery is beyond the capacity of most health systems in Africa. Clearly, treatment with the Ponseti method from birth would have prevented this chronic disability, and at a relatively low cost.

Currently, there is no widely used rating system for the severity of CF, but in general the more fixed a deformity, the greater the severity. The severity of clubfoot deformity is suggested by the extent of stiffness and size differentiation of the feet if unilateral.

Source: Courtesy of Dr. Norgrove Penny, Senior Advisor for Physical Impairments, CBM International; Uganda Sustainable Clubfoot Care Project.

Figure 120.2: Bilateral clubfoot deformity in a newborn. Note the foot plantar flexion, hindfoot varus, and midfoot adductus.

A

B

Source: Courtesy of Dr. Norgrove Penny, Senior Advisor for Physical Impairments, CBM International; Uganda Sustainable Clubfoot Care Project.

Figure 120.3: (A) An example of neglected clubfeet in an older child. (B) The foot deformity in this particular case is of such severity that sustained ambulation was too painful and it became easiest for the child to walk on his knees. Many children do ambulate upright on their deformed feet.

Investigations

Little consensus exists on the role of radiography in the diagnosis and management of idiopathic CF because the diagnosis in the newborn is readily made clinically. Ossification of the foot bones is slight even in healthy newborns, so the expenditure for radiographs would seem quite redundant. In neglected CF, radiographs may help the experienced surgeon to plan operative management; however, this is beyond the scope of this discussion.

Management

The general goal of management for CF of any kind and from any aetiology is to restore to the patient a pain-free, plantigrade foot. Here we discuss idiopathic CF diagnosed early, essentially after birth.

The Ponseti approach is a technique of serial manipulations and casting of the affected leg, beginning almost immediately after birth; most cases require a very minor surgical procedure—a percutaneous heel-cord tenotomy.[12] Ponseti demonstrated by using long-term studies spanning four decades, that his technique is so effective that the rate of primary operative treatment for clubfoot has decreased from more than 80% to less than 5% in many populations. Moreover, treatment results have been shown to be as effective as surgery in terms of overall function and reduction of risk of recurrence.

The clubfoot is manipulated through a series of sequential steps:

Step 1: The forefoot is supinated and the first metatarsal is dorsiflexed to correct the cavus deformity of the foot.

Step 2: While the forefoot remains in supination, the forefoot is abducted while counter pressure is applied to the head of the talus. This corrects the varus and medial deviation of the foot.

Step 3: Finally, dorsiflexion of the fully abducted foot corrects the equinus.

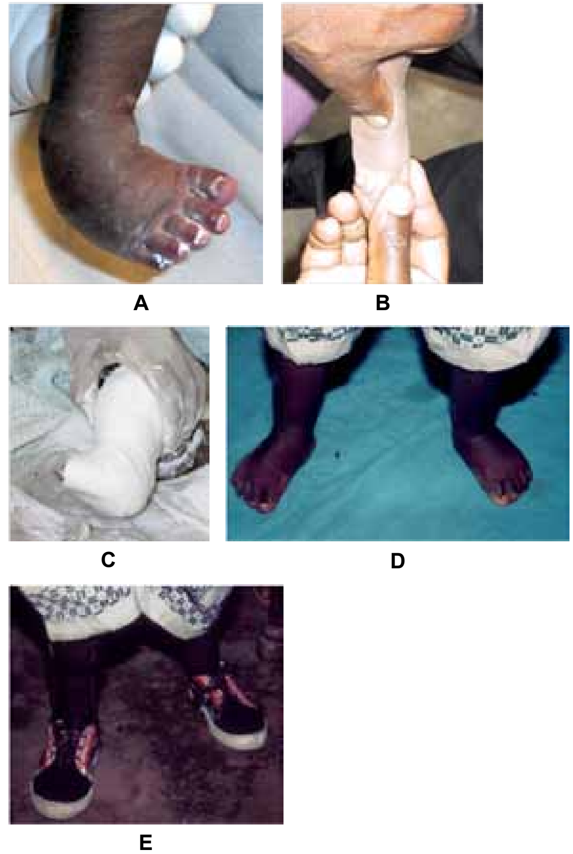
Step 4: The treatment consists of the application of serial casts to the manipulated foot, followed by orthotic management with a foot-ankle orthosis (FAO).

Treatment should begin immediately after birth, if possible. The clubfoot typically corrects after the application of four to five long-leg plaster casts that are applied to the manipulated foot with the knee at 90° (Figure 120.4). These casts are changed every 4 to 5 days. The final cast should be in a position of 70° of abduction and 20° of dorsiflexion. A percutaneous tenotomy of the Achilles tendon is necessary in 80% of the cases. If a tenotomy is performed, the last cast is worn for 3 weeks. After the removal of the final cast, an FAO is worn to prevent relapses. The FAO should be worn for 23 hours per day for the first 3 months and at naptime and nighttime only until 3–4 years of age. Relapses are correctable with the Ponseti method if caught early enough.[12,13]

The Ponseti treatment is *early* management of clubfoot and in general is not used in the clinical management of advanced neglected clubfoot. Neglected clubfoot treatment usually requires complex surgery, which cannot be taught to nonspecialists and should be referred to larger centres with experienced surgeons. However, some recent work in Brazil has shown some success in using low-cost Ponseti methods for treatment of neglected CF presenting after walking age, and more work may show less invasive techniques suitable for well-established cases of CF.[19]

Postoperative Complications

The stiffer the clubfoot, the less likely manipulation and casting by using the Ponseti technique will work. It is important to start treatment as soon as possible. If, after 3 months of Ponseti treatment, the foot is still not corrected, then a surgical procedure will likely be required at some point. Proper training on the Ponseti technique, with emphasis on the sequence of manipulation and casting being very important, is required. Dr. Norgrove Penny, Senior Advisor for Physical Impairments, CBM International, Uganda Sustainable Clubfoot Care Project, has found that percutaneous tenotomy has a definite learning curve among trainees.



Source: Courtesy of Dr. Norgrove Penny, Senior Advisor for Physical Impairments, CBM International; Uganda Sustainable Clubfoot Care Project.

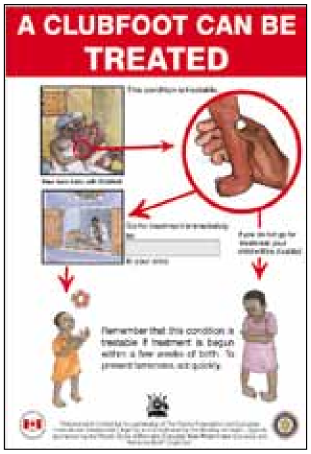
Figure 120.4 (A) A newborn idiopathic clubfoot deformity. (B) One step in the sequential Ponseti technique of serial manipulation of the clubfoot prior to (C) serial casting. All the sequences of the Ponseti method are not shown here. (D) A successful example of the general goal of all clubfoot care: a pain-free, plantigrade foot that can fit in normal shoes (E).

Prognosis and Outcomes

The Ponseti method achieved complete deformity correction in 95% of patients in a series by Morcuende et al.[20] Malawi has set up a Ponseti programme in 25 health districts and corrected 67% of cases to a functional plantigrade foot; however, difficulties with the supply of plaster and splints and patient compliance have affected the results, requiring more research.[17] Pirani et al.[21] have shown very impressive results in the management of idiopathic CF identified at birth. Their work in Uganda underscored the importance of ideal conditions. Due to the significant demand made on both the parents of affected children and the local health care systems, clinical success on a large scale may be more challenging than anticipated.

Prevention

In 2002, Uganda adopted a national strategy of clubfoot care as a public health prevention of disability programme. After six years, the Uganda Sustainable Clubfoot Care Project (USCCP) has collected data to suggest that the Ponseti clubfoot care approach is effective and sustainable.[21] Rural health care workers, including midwives and traditional birth attendants who come into regular contact with mothers and infants, were trained to diagnose CF and to refer infants with this deformity to local orthopaedic officers, who in turn were trained to treat these patients by the Ponseti method. A key to the programme was that orthopaedic officers and sensitized paramedical personnel were assigned to all district and regional hospitals. Early management is key to the success of the Ponseti method and part of the major challenge is community education to find the patients to start treatment (Figure 120.5).



Source: Courtesy of Dr. Norgrove Penny, Senior Advisor for Physical Impairments, CBM International; Uganda Sustainable Clubfoot Care Project.

Figure 120.5. The Uganda Sustainable Clubfoot Care Project public education poster, which should be near every infirmary, birth site, hospital, and paediatric facility.

Ethical Issues

Every effort should be made to promote programmes such USCCP.[21] Surgeons, clinicians, and community leaders must advocate for the early diagnosis and referral to a centre providing Ponseti techniques and ongoing management (see Figure 120.5). It behooves surgeons and researchers to inform their respective health ministries and politicians of the success of programmes such as USCCP.

Secondary Prevention of Osteomyelitis Using Optimal Surgical Principles for Open Fractures

Introduction

Bickler et al.[22] showed that of all surgical conditions in the Gambia, osteomyelitis alone accounted for 15% of total hospital inpatient days, second only to burns. Although haematogenous osteomyelitis may be a more frequent cause of chronic bony infection in African children, posttraumatic osteomyelitis is also an important cause.[23] Indeed, from a surgeon's public-health-approach point of view, the open fracture is a more important cause because the surgeon can *secondarily* prevent posttraumatic osteomyelitis with an appropriate and timely surgical debridement. Haematogenous osteomyelitis can be prevented primarily through nonsurgical means, such as vaccination programmes,[24] improved nutrition, and poverty alleviation, which in general are not the purview of the African surgeon. From a cost-effectiveness point of view, the resources needed to treat an established posttraumatic osteomyelitis are likely much higher than those utilised to treat an acutely open fracture.[26]

It is the authors' contention that because open fractures are so common, many textbooks assume that their management is a readily available skill even in the primary health care worker's set of competencies. The sections on open fracture care in both King's *Primary Surgery*[26] or in the World Health Organization (WHO) *Surgical Care at the District Hospital*[27] each occupy less than half a page and suggest "careful wound toilet" without a detailed outline of the steps needed in appropriate debridement and irrigation and treatment of the open wound. It is the purpose of this section to give the salient details of open fracture management, in particular the precise factors important for optimum open fracture debridement.

Demographics

Ikem et al.[28] studied open fracture in Ile-Ife, Nigeria, and found the majority of open fractures were Gustilo type II or III open fractures (Table 120.1), indicating a higher risk of subsequent infection of lower-extremity fractures. Nearly half (45.8%) of these open fractures went on to fulminant infection, with delay in initial wound management being the major predisposing factor to subsequent infection.

Table 120.1: Gustilo classification of open fractures

Gustilo classification	Description
Type I	An open fracture with a wound <1 cm long and clean.
Type II	An open fracture with a laceration >1 cm long without extensive soft tissue damage, flaps, or avulsions.
Type III	Massive soft tissue damage, compromised vascularity, severe wound contamination, marked fracture instability.
Type IIIA	Adequate soft tissue coverage of fracture despite extensive soft tissue laceration or flaps, or high-energy trauma irrespective of the size of the wound.
Type IIIB	Extensive soft tissue injury loss with periosteal strippingand bone exposure; usually associated with massive contamination.
Type IIIC	Open fracture associated with arterial injury requiring repair.

Source: From Gustilo RB, Anderson JT. Prevention of infection in the treatment of 1,025 open fractures of long bones, retrospective and prospective analyses. J Bone Joint Surg 1976; 58-A:453–458.

Aetiology/Pathophysiology

Ikem and colleagues[28] found that in Nigeria *Staphylococcus aureus* and coagulase-negative staphylococci were the most common organisms associated with open fracture, and delay in initial wound management was a major predisposing factor to wound infection. Fortunately, these organisms are readily treated with antibiotics, but only if supplemented with surgical debridement.[25]

Clinical Presentation

History

The history of an injury resulting in an open fracture is important on a number of levels. The time from injury to treatment has been established as an important factor in the outcome of the injury. As important may be the mechanism of injury, with low-energy injuries (e.g., falls) usually resulting in a Gustilo type I or II fracture, and a high-energy trauma (e.g., motor vehicle crashes) resulting in a type III open fracture, with major tissue loss and possible vascular injury. One must always ask the extent of wound contamination at the injury site—for example, whether the contamination was from a farm, an environment notoriously bad for multiorganism and clostridium infections.

Comorbidities with the injury are also important in that a hypotensive patient cannot adequately perfuse injured tissue, making it more susceptible to inoculation by infecting organisms. Preinjury comorbidities, such as HIV, sickle cell anaemia, malaria, and malnutrition, can all affect susceptibility to infection in damaged tissue.[29]

The history should also include tetanus immunisation, as tetanus continues to be a problem in LMICs.[30] Allergy to antiobiotics must also always be determined.

Physical examination

The accepted management of open fractures, where there is disruption of skin and soft tissue such that there is exposed bone to the environment, is surgical debridement and irrigation within 6 hours of the injury.[31]

In general, it is appropriate to consider type I and II fractures as low-energy wounds and type III fractures as high-energy wounds.[33]

Investigations

Plain radiographs can help the clinician decide the extent of debridement necessary and bony comminution (fragmentation of bone) within the fracture. Avascular pieces of bone will require removal from the wound during debridement because the avascularity of bone fragments will render them foci of infection in the wound if not removed.

Arguably, resources for taking and processing cultures of wounds in acute injuries should not be routinely utilized. Specimens for gram stain, culture, and sensitivity really play a role only in identifying organisms responsible for chronic infections in bone, such as established osteomyelitis.

Management

The goals of treatment for open fractures in children are to avoid infection, achieve soft tissue coverage and bony union and to restore function.

Presurgical management

Acute trauma resuscitation, tetanus prophylaxis, starting of antibiotics, removal of gross debris, sterile irrigation and sterile dressing, and immobilisation should be carried out initially. The theatre is prepared for surgery. Although general anaesthesia should be used ideally, in particular with type III fractures for more aggressive debridement, effective regional blocks of upper and lower extremities with local anaesthetic can be used for open fracture wound care. Lack of general anaesthetic capability should not be used as a reason not to wash out an open fracture. There is some evidence that early administration of antiobiotics may protect against infection even if surgical debridement is delayed.[33]

Surgical management

Surgical management involves irrigation and debridement of open fractures. The necessary equipment includes a basic bone set (osteotome, curette, rongeur), 6–12 liters of normal saline, and wide-bore tubing for copious gravity irrigation as well as bulb and syringe for irrigation. The surgical procedure is as follows:

1.Open laceration is extended longitudinally to provide exposure.

2. Fracture ends are exposed.

3. Devitalised material is removed.

4. Bone ends are cleared of all debris, including haematoma (contaminated), with curette.

5. One litre of normal saline (or available solution) per centimeter of wound is used for irrigation.

6. Suture with 3-0 nylon.

7. Incisions are closed and the open wound is left open; if in doubt, the wound is left open but tendons, nerves, and vessels are covered.

A tourniquet can be applied for proximal vascular control, should it be required; however, the tourniquet should not be inflated in order to determine viability of tissue by bleeding. Any and all avascular tissue, including any bone fragments that no longer have soft tissue attachments (and hence blood supply) must be removed due to risks of becoming infectious foci.

Treatment of wounds by specific Gustilo type

For type I and II fractures, open wounds are opened further by longitudinal incisions proximal and distal to the open wound in order to expose the entire fracture site, (fragments and soft tissue) so that all areas can be inspected and debrided. A 1-cm wound, or even a puncture wound, more often than not hides a larger cavity below the surface. The sharp edge of the fractured bone usually has damaged muscle, leaving large cavities occupied by haematoma, which should be assumed contaminated until surgically debrided.

For type III fractures, principles similar to those for type I and II fractures apply. Sharp debridement is essential and does less damage than scraping healthy tissue with curettes. Wound debridement is done in a centripetal fashion,[34] and bony ends are delivered outside of the wound while the ends are meticulously cleaned of debris. The use of prophylactic fasciotomies should be considered. Repeat debridement is performed in 48–72 hours in order to excise devitalised tissue in type III fractures.

Types of solutions for irrigation

Normal saline is superior to other proposed irrigation agents because it is isotonic and does not disturb the body's natural healing processes.[32] The cost of sterile normal saline makes its availability a concern; a Cochrane Database Systematic Review[35] of tap water for cleansing has shown that in washing out open fractures, in the absence of potable water, boiled and cooled water or distilled water can be used as irrigation agents. Cyr and colleagues[36] studied the use of 5% sodium hypochlorite (Dakin's solution) in ground-derived field water, and showed that the treatment could virtually eliminate the bacterial burden and make the water useable.

Volume of solution for irrigation

Evidence for the optimum volume of solution to use in irrigation is minimal, but in general, the recommendation is 3 liters for a type I fracture, 6 liters for a type II fracture, and 9 liters for a type III fracture.[37] Another guideline is to use 3 liters of solution followed by 1 liter for each centimeter of open wound.

Solution delivery system for wound

High-pressure pulsatile lavage may injure already traumatised tissue and may drive bacteria deeper into the wound.[37] Low-pressure irrigation methods, such as continuous gravity irrigation or bulb syringe and suction irrigation, is efficacious in the removal of foreign material.[32]

Wound closure and fracture stabilisation

A soft tissue closure plan should be developed, but bone and wound coverage should not compromise excision of nonviable or contaminated tissue. A discussion on fracture stabilisation is beyond this review; however, protection of soft tissue should be the initial concern. Pain control is also important, and provisional stabilisation can always be revised. When in doubt, do not close the wound completely. In low-energy type I fractures that are treated less than 24 hours following the open injury, consideration can sometimes be made for primary closure. This is an approach used in the best of health care settings; it may not be realistic in the African setting. If in doubt, it is best to leave the wound open.

Antibiotics

The best "antibiotic" is an excellent wound debridement. In other words, no amount or duration of antibiotics can substitute for a timely and well-performed surgical debridement. Antibiotic prophylaxis for open fractures can depend on local factors and availabilities. In general, prophylaxis includes a first-generation cephalosporin, plus or minus an aminoglycoside, plus or minus penicillin, all depending on the type and extent of wound contamination.

Melvin et al.[32] have developed a detailed algorithm for management of open tibial fractures in the high-resource setting, but it still is useful to the low-income setting, in particular, in helping decision making for primary amputation following severe type III open fractures with neurovascular loss.

Postoperative Complications

The main adverse outcome of surgical debridement is ongoing infection and delayed union of the fracture. A low threshold for re-debridement of open wounds should be maintained at all times; for type III fractures, a routine re-debridement and irrigation at 48–72 hours after the first surgery should be considered almost routine.

Vascularity of tissue, including arterial supply and venous drainage, is the single most important determinant of complications following an open fracture.[34] Knowledge of watershed areas of blood supply to bone in the body is important, but is beyond the scope of this review.

Prognosis and Outcomes

Bach et al.[38] have shown in Malawi that modern management of open fractures in experienced hands can achieve clinical results similar to that seen in higher-income clinical settings. Chronic disability in their cohort of patients at a free-at source hospital was seen in only 12% of patients following open fractures. This work should stand as an indicator that much can be done in Africa to reduce the burden of posttraumatic osteomyelitis, as the prevalence of open fractures in children will likely increase with the increasing incidence of motor-vehicle crashes, in particular vehicles hitting children.[2]

Prevention

Prompt surgical intervention to remove the potential for infection in open fractures can be considered a form of *secondary* prevention. *Primary* prevention would be an intervention to avoid the traumatic event leading to the open fracture, such as streetlights at night so car-child impacts might be avoided. Secondary prevention by timely, aggressive, and extensive debridement and irrigation of an open fracture prevents the establishment of osteomyelitis.

Bach et al.[38] found that in Malawi only 72% of patients reached the hospital within 24 hours of their open fracture. Although the Gustilo and Anderson[31] recommendation of irrigation and debridement within 6 hours may be unrealistic at the present time for much of Africa, the most recent evidence from a North American setting suggests that chronic infection can be avoided following open fracture if the patient presents within a maximum of 24 hours.[39] However, an equally important factor is the quality of the debridement and irrigation.

Ethical Issues

The causes of delayed management of open fractures requires study, but little work has been done in this area in developing countries. Clearly, the effects of poverty, malnutrition, and clinical co-morbidities (e.g., human immunodeficiency virus (HIV), anaemia, sickle cell anaemia, malaria, tuberculosis (TB)) all will have profound effects on wound healing, bony union, and avoidance of infection.

Supracondylar Elbow Fracture in Children

Introduction

Supracondylar fractures (SCFs) in children are common injuries throughout the world and are also the most common injuries requiring surgical intervention. They also cause the most complications and poor outcomes if not adequately treated, in particular when suboptimally managed with poor local resources and expertise.[40] Every attempt should be made to manage these problematic fractures with closed reduction and percutaneous pinning, but there is an arguably growing consensus that the threshold for performing an open reduction should be lowered.[41] It is likely that a careful open reduction may be safer than multiple attempts at closed reduction and acceptance of a poorly reduced fracture. Furthermore, Howard[42] has described little difference in outcomes in closed versus open reductions, and the historical worries of excessive stiffness and heterotopic ossification following open reductions are likely unfounded.

In the developing world, other issues may favour open reduction, such as delayed presentation, where closed reductions of a type III displaced SCF after one week can be very difficult and perhaps even unsafe. Furthermore, it is the experience of one of the authors (PJM), working in Asia, that open reduction of SCF in the absence of x-ray assistance in the operating room (OR) can ensure anatomic reduction and accurate placement of percutaneous pins. Therefore, for practical reasons, open reduction of the paediatric supracondylar fracture in LMICs may be indicated, and it is the purpose of this section to briefly introduce the concept of open reduction via an anterior approach to the elbow for the typical extension-type supracondylar fracture.

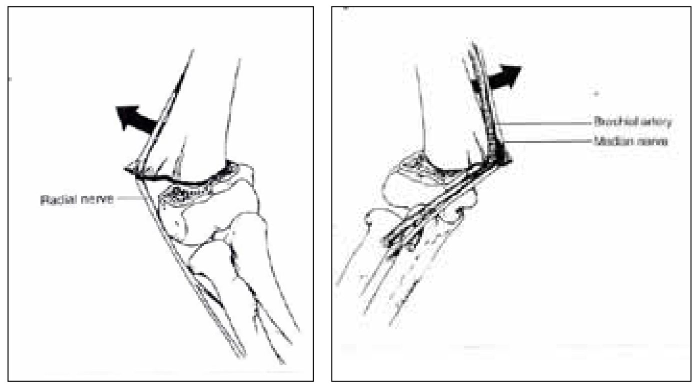
It must be emphasized, however, that if an adequate closed reduction and percutaneous pinning can be achieved, it is the preferred management. However, in light of clear indications for open reduction and internal fixation (e.g., vascular or nerve deficit, irreducible fracture), an anterior approach as described herein aids in the vast majority of severe SCF of the paediatric elbow.

Demographics

Supracondylar fractures of the paediatric elbow are the most common operative fractures in children in both the developed[43] and developing[40] worlds. In the developing world, SCF often presents late and treatment is delayed, increasing the need for an open reduction.[44,45] Typically, more males have SCF, and the left elbow appears to be more at risk. The peak age of 5 to 8 years seems to correlate with the peak of hyperextensibility of the child's elbow.

Aetiology/Pathophysiology

Most SCFs displace in extension (95%), either posteromedially or posterolaterally, threatening important neurovascular structures as a result (Figures 120.6 and 120.7). Flexion injuries are far less common. The degree of fracture displacement likely indicates the risk to neurovascular structures.



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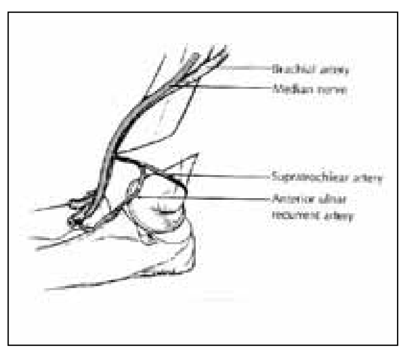
Source: Beaty JH, Kasser, JR, eds. Rockwood and Wilkins' Fractures in Children, 6th ed., Lippincott, Williams and Wilkins, 2006, with permission.

Figure 120.6: (A) In a posteromedial type III fracture, if the spike of bone from the leading edge of the proximal humeral fragment penetrates the brachialis muscle laterally, the radial nerve may be tethered. (B) In a posterolaterally displaced type III fracture, the spike can tether the median nerve and the brachial artery together.



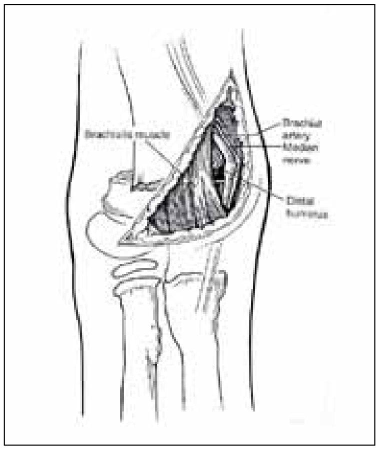
Source: Beaty JH, Kasser, JR, eds. Rockwood and Wilkins' Fractures in Children, 6th ed., Lippincott, Williams and Wilkins, 2006, with permission.

Figure 120.7: Mechanism of vascular injury to the brachial artery in typical extension-type supracondylar fracture of the elbow. Increasing displacement likely increases the extent of injury to the artery. Timely closed reduction, even in the emergency room, often will restore a feeble or absent radial pulse.

Clinical Presentation

History

Mechanism of injury details are important (e.g., how high was the fall? what was the position of the elbow during the fall?). Other injuries, especially ipsilateral shoulder/wrist/forearm injuries are important for management. The last meal, which hand is dominant, and previous injuries to the elbow may be important. Past immunisations and past general anaesthetic complications should be queried.



Source: Beaty JH, Kasser, JR, eds. Rockwood and Wilkins' Fractures in Children, 6th ed., Lippincott, Williams and Wilkins, 2006, with permission.

Figure 120.8: The anteromedial approach: A curvilinear anterior approach with the proximal arm of the incision being superomedial can address the neurovascular bundle (NVB) that is at risk in most type III SCFs. The proximal humeral fragment is often buttonholed through the brachialis muscle, and the NVB is tented over this. Because of the muscle damage anteriorly, the subcutaneous haematoma there can be used to guide the incision. Since most of the damage is done, direct visualisation allows for anatomic reduction and pinning of the fracture after the NVB is relieved or repaired.

Physical examination

The preoperative evaluation involves an obvious deformity with the elbow usually extended. Documentation of neurological status of the median, ulnar, and radial nerves as well as assessment of the pulse are important. A pulseless hand that is warm and with normal capillary filling can often be followed, but serial examination is important to ensure the hand remains viable despite an absent or weak pulse. Serial examination is important to watch for the evolution of a compartment syndrome. A cold, pulseless hand requires exploration of the brachial artery, as described in the Management section of this chapter. In 10–20% of cases there is a neurological deficit, the most common being the anterior interosseous nerve (AIN), which motors the deep flexor of the index finger and flexor polllicis longus.

A haematoma on the anterior elbow surface indicates muscle damage to the distal brachialis and biceps muscle and suggests the possibility of bone fragment invagination into muscle. An anterior haematoma on the skin is a good landmark to guide the anterior incision for an open approach to the fracture because following the skin incision, the deeper dissection is usually already done by the sharp fracture fragment, and just below the incision the fracture is often immediately accessible (Figure 120.8). Furthermore, the neurovascular structures (Figures 120.6–120.8) at risk for injury are easily found anteriorly.

Investigations

Plain anteroposterior (AP) and lateral radiographs suffice to show the type of fracture for classification purposes and for treatment. There are two types of fractures: the extension type (95%) and flexion type.

Gartland's classification for extension fractures recognizes that anterior cortex fails first with a resultant posterior displacement of the distal fragment:[46]

Management

Past concerns and reluctance to open the paediatric elbow due to concerns of stiffness are unfounded, and many authors have shown that open reduction is safe and effective.[47,48] Fleuri-Chateau et al.,[41] Gennari et al.,[49] and Beaty and Kasser[43] have demonstrated that open reduction of SCF is safe and effective and concluded that the threshold for open reduction should be lowered. It is the authors' contention that open reduction will likely be safer and more effective for severe, irreducible fractures, in particular for the surgeon who deals relatively infrequently with these fractures. A brief review of the pertinent anatomy before surgery helps to appreciate the location of the neurovascular bundles demonstrated in Figures 120.6 and 120.7.

Open reduction improves the ability for satisfactory reduction in the absence of imaging intensification. With an anterior transverse incision, the fracture fragments can be pieced together and held with the fingers in position while K-wires are drilled retrograde from the distal into the proximal fragment. Without x-ray imaging, the position of the K-wires is appreciated because the surgeon can see where the K-wire is going into bone through the open wound.

Koudstaal et al.[50] demonstrated that the anterior approach to displaced extension type supracondylar fractures was preferable to other open techniques in terms of safety and simplicity. It is a compelling approach in that it exposes directly the threatened neurovascular bundle (see Figure 120.8) and the subcutaneous haematoma anterior to the elbow joint usually means the dissection deep to the skin incision down to bone has already been done by the proximal fracture sharp edge. Once skin is incised, exploration with the finger is often direct to bone.

Anterior approach to the paediatric elbow

Type I:	nondisplaced fracture
Type II:	displaced fracture with intact posterior cortex;
Type III:	displaced fracture with no cortical contact (posteromedial > posterolateral)

1. A transverse incision is made just above the flexion crease of the elbow. Significant bruising often is seen where the incision is going. The incision can be extended distally on the ulnar aspect of the transverse incision and proximally on the radial side if needed (see Figure 120.8).

2. The NVB may be very superficial, and may disappear into the fracture site.

3. The brachial artery and median nerve are identified.

4. Dissection is proximal to distal.

5. Soft tissues (including periosteum) are removed from the anterior fracture site.

6.The fracture is reduced under direct vision.

7. The fracture is pinned in the usual fashion with 0.062 K-wires.

8. With a fracture, the reduced viability of the arm usually recovers whether or not the brachial artery is functioning. The collateral blood supply is usually adequate.

Following open reduction of the fracture the fracture can be easily reduced with no soft tissue interposition and can often be palpated to be anatomic. Thus, an image intensifier may not be required, although when present it should be used to assess the adequacy of reduction. Percutaneous pinning of the supracondylar fracture is then carried out, with two lateral pins usually; if still unstable, a supplementary medial pin is used.

Vascular injury

Note that the pulse often returns with a timely closed reduction, and this is the first approach to the displaced supracondylar fracture without a pulse.

Surgical exploration of an injured artery for the purposes of repair is a tertiary hospital activity by an experienced surgeon and is not practical in many African settings where supracondylar fractures occur. Fortunately, many pulseless hands with supracondylar fractures are warm and viable with very good capillary refill and do not require explorarion. Howard's review[42] of the best (level IV) evidence for this issue from the literature concludes that the pulseless, viable hand following supracondylar fracture should be treated by observation, with vascular interventions only when the pulseless hand appears cold and nonviable.

Postoperative Complications

Postoperative complications include the following:

- *Infection:* Pintract cellulitis, abscess, septic arthritis (especially if there are multiple lateral pins).
- *Nerve injury:* AIN injury is most common with a posterlateral type fracture; radial nerve injury is common with a posteromedial fracture displacement; and the ulnar nerve can be injured iatrogenically if a medial pin is used.
- *Compartment syndrome:* Watch for increased swelling, hard compartments, pain with passive flexion and extension of fingers, and often pain control. Close serial examination of the patient postoperatively is required, and a return to the theatre for compartmental fasciotomies must always be considered.
- *Growth disturbance:* This is rarely the cause of malalignment, which is usually due to malreduction.
- *Stiffness:* Patient may lose 10–20 degrees of terminal extension.

Prognosis and Outcomes

Farley et al.[51] have demonstrated that nonpaediatric orthopaedic physicians who treat SCF infrequently can manage these difficult fractures with outcomes and complication profiles similar to paediatric orthopaedists. Cubitus varus has an incidence of approximately 5% and is usually of cosmetic significance, usually with a good functional range of motion. The major poor outcome comes with compartment syndrome.

Prevention

Every district hospital and any moderately busy surgical centre in Africa—or anywhere in the world, for that matter—will see this common fracture, so local expertise should develop to deal with the most challenging aspects of these fractures. The anxiety with open SCF management should be reduced with the debunking of the concept that opening a paediatric elbow is somehow ill-advised or incompetent. Knowledge of the basic anatomical structures, as discussed in this section, makes this approach safe.

Ethical Issues

Two surgical Africa's exist: the bush hospital with no or minimal surgical resources and the slowly developing African district hospital surgical infrastructure, where an open approach to fractures is now safer, almost to the level of developed countries. In the former environment, open management is perhaps ill advised, whereas in the latter, basic surgical techniques should allow for safe and efficacious open reduction of SCF and the development of local expertise for management of this very common fracture, which is prone to a number of complications.

Poliomyelitis-Related Long Bone Angular Deformity

Despite decreasing rates of new cases of polio due to worldwide vaccination programmes, residual deformities secondary to polio continue to be common in Sub-Saharan Africa, India, and much of the developing world.[52] The literature on the orthopaedic management of polio is extensive and cannot be reviewed here other than to discuss some general principles. A detailed account of polio and the large number of procedures available to manage the musculoskeletal problems associated with this disease is available in the sizeable literature, in particular the classic surgical chapter by Tachdjian.[53] The emergence of "postpolio syndrome" (PPS), a condition that develops 20 to 30 years following the initial onset of polio, appears to be of increasing importance to clinicians and is reviewed briefly in this section. Although PPS has been recognized for more than 100 years, it appears important now because of the large epidemics of poliomyelitis that occurred in the 1940s and 1950s.

Demographics

WHO has indicated that polio has reemerged in a number of African countries, including Angola, Chad, and the Democratic Republic of the Congo, with increases due mostly from a lack of immunisations. The agency estimates that 75% of children on the African continent receive vaccines, and efforts are being taken to raise the number to 85%.[54]

As of 2010, in only four countries (Afghanistan, India, Nigeria, and Pakistan)[54] was polio endemic, compared with 125 countries in 1988.[55] It is important to note that unvaccinated adults traveling to endemic countries can contract polio.

Aetiology/Pathophysiology

Poliomyelitis is an acute infectious viral disease that initially invades the gastrointestinal and respiratory tracts and then spreads haematogenously to the central nervous system. The polio virus has a particular affinity for the anterior horn cells of the spinal cord, in particular the cervical and lumbar enlargements of the cord, and for specific motor nuclei of the brain stem.[53]

Clinical Presentation

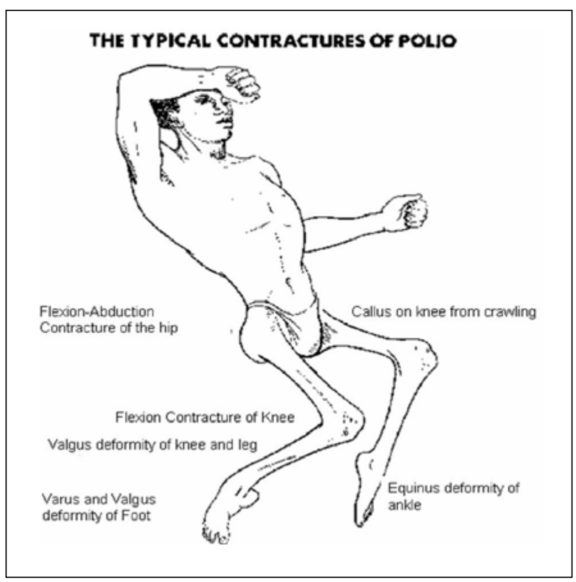
History

The phases of polio

The course of polio is divided into acute, convalescent, and chronic phases, with the acute phase, lasting 5 to 10 days, being the period when paralysis may occur. The acute phase is considered terminated 48 hours following the return of normal temperature in the patient.

Following the acute phase of polio is the convalescent phase, or the recovery stage. Here, the acute symptoms and muscle tenderness disappear and the paralysed muscles start to recover. This stage can last for up to 2 years after the acute onset of polio. During this entire period, there is gradual recovery of the muscles, rapidly usually in the first 6 months, then more slowly. Motor power plateaus by 12 months in most patients.

The period after 18 to 24 months is called the residual-paralysis stage. No recovery of muscle power occurs in this stage, and deformities that occur are due to imbalance of muscle power and poor posture. There is also disuse atrophy of muscles and shortening of the leg due to interference with growth. In severely neglected cases, gross fixed deformities of the hip, knee, and foot occur with severe wasting of muscles (Figure 120.9).[56] Impoverished children with extensive paralysis and gross deformities literally have to crawl on all fours to move from place to place.



Source: Adapted from Vidyadhara S, Rao SK, Shetty MS, Gnanadoss JJ. Poliomyelitis. e-Medicine, updated 20 November 2008. Accessed 24 February 2010.

Figure 120.9: Contractures possible in polio after the chronic, residual phase with treatment addressing muscles and limbs generally neglected.

Postpolio Syndrome

Postpolio syndrome has received considerable attention recently with late manifestations of poliomyelitis developing in patients 20 to 30 years after the occurrence of the acute illness. An estimated 25–60% of the patients who had acute polio may experience these late effects of the disease.[56]

The specific cause or causes of PPS are unknown, with both pathophysiologic causes and functional causes. Pathophysiologic causes may include chronic poliovirus infection, death of the remaining motor neurons with aging, or damage to the remaining motor neurons caused by increased demands or secondary insults, and immune-mediated syndromes. Functional aetiologies for PPS include greater energy expenditure as a result of weight gain and muscle weakness caused by overuse or disuse.

Postpolio syndrome is characterised by neurologic, musculoskeletal, and general manifestations. Musculoskeletal manifestations include muscle pain, joint pain, spinal changes such as spondylosis and scoliosis, and secondary root and peripheral nerve compression. General manifestations include generalised fatigue and cold intolerance. The slowly progressive muscle weakness occurs in those muscle groups already involved, such as the quadriceps and calf muscles.

Physical examination

Physical examination of the limbs and joints is key in assessing the nature of extremity contractures, in particular the relationship of contractures to neighbouring joints and the spine. Experienced functional assessment skills by orthopaedic surgeons, orthopaedic clinical officers, physiotherapists, and occupational therapists are important to plan a comprehensive treatment plan for the polio patient.

Investigations

A history with a detailed orthopaedic and neurological clinical examination is essential to define the clinical problems to be addressed in the management of poliomyelitis. Depending on the stage of polio, serial examinations of the patient guide the therapeutic regimen. The rate and extent of muscle recovery is also appreciated only with serial examinations—monthly, if possible. When muscle power in a polio patient shows no improvement over a 3-month period, it is unlikely to recover any more functionally and serves as an important prognostic tool. At this point, appropriate orthotic treatment should be considered to improve function. If functional motor improvement is continuing, then orthotic/splint management may, in fact, thwart recovery.

Diagnostic criteria for postpolio syndrome include:

- A prior episode of paralytic poliomyelitis with residual motor neuron loss (which can be confirmed through a typical patient history, a neurologic examination, and, if needed, an electrodiagnostic examination, which in the African setting is unnecessary or unavailable.
- A period of neurologic recovery followed by an interval (usually 15 years or more) of neurologic and functional stability.
- A gradual or abrupt onset of new weakness or abnormal muscle fatigue (decreased endurance), muscle atrophy, or generalised fatigue.
- Exclusion of medical, orthopaedic, and neurologic conditions that may be causing the symptoms mentioned above.

Management

Treatment

Treatment in the acute stage is mainly medical, including general supportive treatment for fever and irritation, for the prevention of secondary respiratory infection, and for any respiratory paralysis. The paralyzed legs are supported by plaster splints or pillows and sandbags to keep the hip joints in slight flexion and in neutral rotation. The knee joint is held at 5° of flexion, and the foot is supported in a neutral, 90° position. Splinting relieves pain and spasm and prevents the development of deformities. Physiotherapy is needed to maintain range of motion and should be implemented from the very start of the acute phase of the disease. Prevention of joint contracture is much easier to treat, and less costly in dollars and manpower, than established joint contractures.

The general goals of management of poliomyelitis were established by the International Society of Prosthetics and Orthotics consensus conference on poliomyelitis at Hammamet, Tunisia, in 1997,[58] and include:

- overcoming the effects of paralysis;
- correcting deformities;
- restoring joint mobility;
- relieving pain; and
- restoring limb length discrepancy.

In low-resource settings, management is challenged by the need for a comprehensive treatment plan for each patient, with priorities focused on:

- addressing ambulation;
- prevention of deformity, especially during growth;
- decreasing the needs for bracing;
- addressing the upper extremity; and
- managing spinal deformity.

In the ideal setting, the orthopaedic physican/surgeon and the physiotherapist should be involved early with the polio patient in order to *prevent* many of the residual deformities that result from muscle imbalances, which result in the muscle and tendon contractures that impair function and produce joint contractures as well as the pain and disability associated with these.

Physiotherapy and rehabilitation

Physical therapy prevents or corrects deformity through passive stretching exercises and by increasing motor strength through active exercising of muscles that remain active following the chronic phase (after 18 months postexposure). Perhaps most important, physical therapy includes functional training, which enables the patient to learn methods to overcome the handicaps of physical disability from polio.

Orthotics and appliances

The primary objectives of orthotic management according to Tachdjian[53] include:

- supporting walking to increase functional activity;
- protecting a weak muscle from overstretching;
- augmenting weak muscles or substituting for absent muscles;
- preventing deformity and malposition; and
- correcting deformity by stretching contracted muscles.

In general, dynamic splinting is superior to static splinting; however, cost constraints preclude the manufacture of dynamic splints in many developing countries.

Surgery

Surgery is indicated when deformities interfere with the activities of daily living (ADLs), to stabilise joints that have become unstable due to chronic muscle imbalance, and to improve motor function via tendon transfer (Figure 120.10). A plethora of operative procedures, including fasciotomy, joint capsulotomy, tendon transfers, osteotomy, and bony arthrodesis, can be used to address the paralytic deformities of the upper or lower extremities seen in polio; however, a detailed account of these procedures is beyond the scope of this brief review. Leg length discrepancy is common in polio from shortening of the paralyzed leg, and many procedures are available to correct this. Patient selection is extremely important, requiring some surgical experience, and has been well described by Krul.[59]

Figure 120.10: Large improvements in quality of life and productivity are seen following the management of postpolio paralysis. Resource-intensive care requires physiotherapy, appliances such as braces and walking aids, and also surgery; in this case, to relieve hip and knee contractures and lengthen tight heel-cords.

Postoperative Complications

The outstanding chapters in Tachdjian's *Pediatric Orthopedics*[53] describing surgery for all of the deformities related to polio and similar flaccid paralytic diseases include discussion of postoperative complications. Speigel[58] has also referenced many of the classic surgical articles for the management of polio.

Prognosis and Outcomes

Two primary factors are important when considering the prognosis of polio with ideal clinical management in place:

1. *The severity of the initial paralysis:* If total muscle paralysis persists beyond the second month, then severe motor cell destruction is likely; this is in contrast to partial paralysis, for which the prognosis is better.

2. *The diffuseness of the regional distribution of paralysis:* A weak muscle surrounded by paralyzed muscles is less likely to recover than if it were next to strong muscles.

Perhaps a larger factor in prognosis is scarcity of timely physical therapy resources to address contractures and provide range of motion for the patient. For this reason, Speigel[58] has stated that when resources are scarce, younger patients and those with lesser degrees of deformity should be prioritized.

Prevention

In recent years, some attention has focused on vaccine-associated paralytic poliomyelitis (VAPP), in which cases of poliomyelitis are caused by the oral vaccine. The risk of polio contraction from vaccine is extremely low, however, perhaps 1 case in 2.5 million doses, with those at risk including immunocompromised hosts and infants receiving their first dose. WHO has recommended that, to prevent this rare complication, children be inoculated first with inactivated vaccine, followed by oral attenuated vaccine.[60,61] The surgeon can act as community advocate in support of the widespread use of approved national vaccination programmes (Figure 120.11).

Source: Courtesy of UNICEF, with permission.

Figure 120.11: Poster promoting polio vaccine in Sierra Leone.

Ethical Issues

"Rehabilitation" surgery in the orthopaedic sense is meant to promote improvements in function.[20,58,59] Often, this surgery requires a very experienced surgeon; the clinical decision to operate is often a more difficult task than doing the actual operation itself. Furthermore, rehabilitation surgery should be performed only when there is patient access to physical therapy and orthotics. Resources are limited, thus limiting access to the benefits of this surgery to only a small number of Africans.

Nutritional Rickets and Angular Deformities in Orthopaedics

Introduction

Nutritional rickets (NR) constitutes a high burden of morbidity and mortality among children in Africa as the precursors of NR (i.e., poor socioeconomic status, low birth weight, protein-energy malnutrition, and common childhood infections) all continue to be common on the African subcontinent.[62] Surgeons and orthopaedic officers will likely not be providing the medical management of NR but will be referring children with significant angular deformities. This brief discussion of the orthopaedic management of NR will concentrate on the long bone angular deformities seen in the lower extremities.

Demographics

Nutritional rickets remains a public health problem in many countries, despite dramatic declines in the prevalence of the condition in many developed countries since the discoveries of vitamin D and the role of ultraviolet light in prevention. The disease continues to be problematic among infants in many communities, especially among infants who are exclusively breast-fed, infants and children of dark-skinned residents living in temperate climates, infants and their mothers in the Middle East, and infants and children in many developing countries in the tropics and subtropics, such as Nigeria, Ethiopia, Yemen, and Bangladesh.

Aetiology/Pathophysiology

Rickets has a long aetiological list; this discussion concentrates on NR, although the orthopaedic manifestations and surgical management may be similar for all aetiologies of ricketseal MSK disease. Rickets is caused by decreased levels of calcium or phosphate, leading to abnormal mineralisation of the skeleton. It occurs in growing children, and both the cartilage of the physes and the bones are involved.

Vitamin D deficiency is a major cause of rickets among young infants in many countries, because breast milk is low in vitamin D and its metabolites and social and religious customs and/or climatic conditions often prevent adequate ultraviolet light exposure. In sunny countries, such as Nigeria, South Africa, and Bangladesh, such factors may not apply.

Akpede et al.[63] showed that deficiency or reduced availability of dietary calcium may be of at least equal importance with vitamin D deficiency in the aetiology of NR in the Sahel savanna in Nigeria. In some parts of Africa, deficiency of calcium, phosphorus, or both in the diet may also lead to rickets, especially in societies where corn is predominant in the diet. Calcium and vitamin D intakes are also low in infants who are fed vegan diets, particularly lactovegans, and monitoring of their vitamin D status is essential. Dietary calcium deficiency and vitamin D deficiency represent two ends of the spectrum for the pathogenesis of nutritional rickets, with a combination of the two in the middle.

Clinical Presentation

History

By definition, rickets is observed only in growing children, although the effects may be observed later in life if untreated. No sexual predilection is noted. Agarwal et al.[64] showed that delayed walking in New Delhi children may be the initial manifestation of NR in children, and that the majority of ricketic nonwalkers will start to walk within 2 to 5 months of appropriate treatment.

Physical examination

Children affected with rickets present with short stature, often under the third percentile, as well as muscle weakness and frontal bossing of the skull with enlargement of suture lines. In more severe instances in children older than 2 years, vertebral softening can lead to kyphoscoliosis, the kyphotic segment known as the rachitic "catback" deformity. However, the physical ailment most disabling and that is most amenable to surgical intervention is the angular deformity of the lower extremity, either valgus or varus.

Weight bearing produces the angular deformities usually bowlegs (varus) and knock-knees (valgus). Oginni et al.[65] showed that children in Nigeria between the ages of 2 and 5 years with large angular deformities in their knees likely had rickets. They further showed that young children with NR generally had varus deformities at the knees. Children a bit older showed a more bimodal deformity presentation, either varus or valgus. Older children with NR were more likely to display valgus deformities (Figure 120.12).

Source: Courtesy of Dr. Norgrove Penny, Senior Advisor for Physical Impairments, CBM International; Uganda Sustainable Clubfoot Care Project.

Figure 120.12: Two children with severe valgus (knock-kneed) deformities secondary to NR. Had they been younger walking children when their NR developed they would have more than likely had bowed, or varus, deformities. Correction of valgus deformities puts the peroneal nerves at risk and they must be mobilised surgically at the time of surgical correction to prevent postoperative drop-foot deformities.

In the chest, knobby deformities result in the rachitic rosary along the costochondral junctions. The weakened ribs pulled by muscles also produce flaring over the diaphragm, which is known as the Harrison groove. The sternum may be pulled into a pigeon-breast deformity.

Investigations

The biochemical diagnosis of NR is often the role of the paediatrician or medical consultant, and will not be discussed here. The treating surgeon will use x-rays largely to manage children with the MSK manifestations of NR. Radiographic findings of NR include widening of the metaphyses and the appearance of flaring or cupping at the physis (growth plate at the end of the long bone), in particular, those near the knee and the distal radius.

Management

Knowledge of the use of a variety of external fixation devices, from the circular frames developed by Ilizarov[66] in Russia to the uni- and multi-planar frames, is key for the surgeon managing the deformities of NR. Not all deformities require an external fixator, and the benefits of an acute correction via angular osteotomy include a one-time surgical procedure with the reduced risks of infection and other complications. Slow correction by external fixation, usually for more severe and complex deformities, requires considerable training and resources. A "one-time" osteotomy to correct a modest angular deformity may be

Source: Courtesy of Dr. Norgrove Penny, Senior Advisor for Physical Impairments, CBM International; Uganda Sustainable Clubfoot Care Project.

Figure 120.13:. A case of angular deformity from nutritional rickets in a young boy. A uniplanar external fixator, of a kind not readily available in Africa, was used to completely correct this child's gross angular deformity to a normal slight genu valgum.

appropriate in the district, "up-country" hospital, where ordinary orthopaedic hardware, such as plates or K-wires, can be used. However, the more severe and complex angular deformity, more suited for an external fixator, is more appropriate in the specialty hospital setting (Figure 120.13) due to the need for the complex frames.[20]

Considerable surgical experience is needed for the large, complex, sometimes three-dimensional (3D) deformities seen. Fortunately, most deformities from NR appear to be uniplanar, either valgus or varus, unlike the 3D deformities seen with posttraumatic malunions. Selection of internal versus external fixation is only one problem that must be solved in limb angular-deformity surgery. Another is the decision as to where exactly the "centre of rotational deformity" (CORA) lies in the long bone because this determines where the boney osteotomy will be performed.[67] Considerable preoperative planning is required here, and a detailed discussion of the appropriate use and intricacies of angular deformity surgery is beyond the scope of this chapter.

Postoperative Complications

The list of complications from surgery for angular deformity is lengthy and is beyond the scope of this chapter. With respect to complications affecting the efficacy of surgical management, the benefits of experience and preoperative planning cannot be underestimated.

Prognosis and Outcomes

There is a paucity of survey data on the types of paediatric orthopaedic conditions that require surgery and the factors influencing their outcome in most parts of sub-Saharan Africa. Akinyoola and colleagues[68] showed that the most common indications for surgery in Nigeria were angular knee deformities (from Blount's disease and rickets) and clubfoot. Wound infection was the most common postoperative complication (8.2%). The patient's age, length of preoperative hospital stay, length of operation, and intraoperative blood loss above 200 ml were statistically significant factors adversely affecting the surgical wound outcome. Most of these factors for poor outcome were patient- and environment-related and were considered preventable.

Prevention

The only systematic review of NR in Africa was carried out in Ethiopia in 2005,[62] and much work is needed to address nutritional, genetic, and clinical management issues with NR. Prevention of surgical issues of NR is best approached through primary care nutritional and poverty alleviation.

Thacher et al.[69] showed Nigerian children with rickets have a low intake of calcium and have a better response to treatment with calcium alone or in combination with vitamin D than to treatment with vitamin D alone. Since Nigerian children with rickets had low calcium intake, treatment should focus on dietary supplementation with calcium or a combination of calcium and vitamin D.

Ethical Issues

African mothers' awareness about rickets is still very low and is a major reason for late presentation or complete failure to seek treatment for rickettsial deformity. Lack of funds, poor compliance with treatment, prolonged treatment periods, and lack of information on where to seek treatment were among the important factors affecting completeness of treatment of knee deformity due to rickets in Nigeria.[70] Increased and sustained public health programmes are necessary for prevention, and it has been argued that health policy should incorporate free surgical fees for children with established rickettsial knee deformity to encourage community participation in the management of this preventable condition.

Evidence-Based Research

Table 120.2 presents results of a Rwandan national survey on the orthopaedic needs of children. Table 120.3 presents a USCCP training program for congenital clubfoot in Uganda. Table 120.4 presents a systematic review of treatment of open fractures of the tibia in children.

Table 120.2: Evidence-based research

Title	The orthopaedic needs of children in Rwanda: results from a national survey and orthopaedic service Implications
Authors	Atijosan O, Simms V, Kuper H, Rischewski D, Lavy C
Institution	Ministry of Health, Rwanda; Nuffield Orthopaedic Center, Oxford, UK; London School of Hygiene and Tropical Medicine, London, UK
Reference	J Pediatr Ortho 2009; 29:948–951
Problem	Lack of objective, population-based data on the magnitude of orthopaedic problems in Rwandan children and the types of services required, such as surgical services.
Intervention	A national survey of musculoskeletal impairment prevalence using cluster sampling techniques and an urban/rural demographic split matching national population distribution data.
Comparison/ control (quality of evidence)	The data are of high quality because this is a population-based survey. The weakness of the study is that some estimates are extrapolations. The survey also did not measure acute trauma, which any planning scheme would have to include.
Outcome/ effect	Establishment of benchmark data.
Historical significance/ comments	This study is possibly the first national-level, population-based study in sub-Saharan Africa to attempt to quantify the "global" burden of disease in children's orthopaedics for the purposes of treatment planning and provision. From a public health approach, disease surveillance is essential to determining burden levels for resource allocation planning. Very few benchmark studies exist for surgical problems in orthopaedics in developing countries.

Table 120.3: Evidence-based research

Title	Towards effective Ponseti clubfoot care: the Uganda Sustainable Clubfoot Care Project (USCCP)
Authors	Pirani S, Naddumba E, Mathias R, Konde-Lule J, Penny JN, Beyeza T, Mbonye B, Amone J, Franceschi F
Institution	Uganda Ministry of Health; University of British Columbia, Vancouver, Canada.
Reference	Clin Orthop Relat Res 2009; 467:1154–1163
Problem	Congenital clubfoot, one of the most common congenital musculoskeletal deformities worldwide.
Intervention	Ponseti technique of clubfoot care; not suitable for neglected cases of clubfoot.
Comparison/ control (quality of evidence)	Level IV, therapeutic study.
Outcome/ effect	USCCP has trained 798 healthcare professionals to treat foot deformities at birth. Ponseti clubfoot care is now available in 21 hospitals in Uganda.
Historical significance/ comments	Landmark programme to address the congenital deformity causing the most physical disability in the world. Ongoing assessment of USCCP-treated clubfoot cohort with a high level of evidence demonstrated that this programme can be applied almost anywhere in the developing world.

Table 120.4: Evidence-based research

Title	Open fractures of the tibia in the pediatric population: a systematic review
Authors	Baldwin KD, Babatunde OM, Huffman GR, Hosalkar HS
Institution	University of Pennsylvania, Children's Hospital of Philadelphia, Philadelphia, Pennsylvania, USA
Reference	J Child Orthop 2009; 3:199–208
Problem	How the treatment for open fractures in children has evolved over the past three decades, based on Gustilo type.
Intervention	Comparison of infection rates between different types of open fractures.
Comparison/ control (quality of evidence)	Systematic review of paediatric studies only with 14 studies with linear regression analysis insures level IV evidence.
Outcome/ effect	No significant difference in odds of infection between type I and type II fractures; type III fractures have 3.5-fold and 2.3-fold greater odds of infection than type I and type II fractures, respectively.
Historical significance/ comments	A strong relationship exists between Gustillo subtypes and odds of infection; union rates are delayed with increasing severity of injury.

Key Summary Points

Congenital Idiopathic Clubfoot

1. The Ponseti method of idiopathic clubfoot treatment can effectively treat clubfoot (CF) identified at birth with perhaps >90% effectiveness with only a small, percutaneous tenotomy required under local anaesthetic. This has changed the approach to CF worldwide and benefits low- and middle-income countries by relatively low-cost intervention.
2. African studies have shown that effective Ponseti treatment can be done with nonspecialist physicians and nonphysician health care workers.
3. Neglected clubfoot in older children will likely require surgery in experienced hands; however, some work suggests Posenti techniques may be used in neglected cases.
4. Percutaneous tenotomy is required in 80% of cases, and this outpatient procedure done under local anaesthetic can take some experience to master.
5. An important factor in effecting Ponseti treatment is bringing patients from rural settings to the district centres offering treatment. Community-based, public health approaches are needed.

Secondary Prevention of Osteomyelitis

1. Haematogenous osteomyelitis is probably the biggest cause of osteomyelitis in children in Africa; however, open fractures are another major cause, and open fractures are amenable to prevention by timely surgical techniques.
2. Surgeons can practise secondary prevention by immediate surgical debridement of open fractures.
3. Type I and II open fractures need urgent care and can be done with simple tools, such as local anaesthetic and simple, available, but copious solutions. Low-energy type I fractures can wait for surgery until the next morning, but should not be left until the following evening (i.e., should be treated within 24 hours).
4. Open fractures should not be treated without opening the wound. This is true even for what appears to be merely a puncture wound over the fracture site, since the cavity deep to the wound is often much larger than anticipated and can contain a haematoma that can become contaminated and the focus of a fulminant infection.
5. Type III open fractures require immediate surgical attention and antibiotics and, ideally, transfer to an experienced surgical facility where a secondary debridement should be planned almost routinely prior to or simultaneous to the definitive management of the fracture.
6. Work done in Africa shows that the disability from open fractures can be reduced almost to the level seen in developed countries when proper treatment is done.

Supracondylar Elbow Fracture

1. Open procedures on the paediatric elbow do not cause excessive stiffness or heterotopic ossification.
2. Open reduction of a paediatric supracondylar fracture (SCF) is safer than multiple attempts at reduction of a grossly displaced fracture and acceptance of a poor reduction.
3. An excellent approach to the typical type III extension SCF is a transverse anterior approach just above the flexor crease because once through the skin, much of the dissection has been done by the fracture and bone is easily seen, as are at-risk neurovascular structures.
4. Open reduction allows for anatomic reduction of fracture fragments by palpation, and appreciation of placement of K-wires, even without x-ray control or fluoroscopy.
5. A warm, pulseless, but viable hand associated with a type III SCF can be observed; a cold, pulseless hand needs immediate attempt at closed reduction to resume circulation and, if unsuccessful, then immediate open reduction.

Poliomyelitis-related Long Bone Angular Deformity

1. The emergence of postpolio syndrome, a condition that develops 20 to 30 years following the initial onset of polio, appears to be of increasing importance to orthopaedic surgeons.
2. Unvaccinated adults traveling to polio-endemic countries can contract polio.
3. Delay of referral of polio patients to the orthopaedic officer, physiotherapist, or surgeon is a major cause of severe joint contractures and the resulting severe functional impairment.
4. To be effective in polio patients, surgery requires dedicated pre- and postoperative physio- and occupational therapy.
5. Surgeons can also play important roles in their communities in advocating for preventative interventions, such as surveillance and immunisation programmes.

Nutritional Rickets and Angular Deformity

1. Nutritional rickets (NR) continues to be a significant problem in the developing world, although prevention interventions are effective.
2. Long bone angular deformity of the lower extremity is the primary problem dealt with by the orthopaedic officer or surgeon.
3. Surgeons and orthopaedic officers can play leading roles in their communities in advocating for prevention interventions for NR.

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