Severe open fractures of the tibia: the courage to amputate

The management of wounds is an ancient surgical skill. Their importance is neatly summarised in Hippocrates' famous aphorism, "If you wish to be a surgeon go to war." Today war has been largely replaced by the high velocity road traffic accident, and no wound poses more challenges than the severe open fracture of the tibia, a common injury in motor cycle and pedestrian accidents. Three anatomical features make these severe injuries difficult to treat: the tibia has a minimal soft tissue sleeve along its medial border; the vessels have a fixed point at the distal end of the popliteal artery; and the nerves are vulnerable to either direct contusion or traction injuries.

In contrast to his predecessor, who was equipped only to amputate, the modern surgeon is armed with an impressive array of techniques. Vascular damage can be accurately diagnosed and repaired; large soft tissue defects can be covered by transposing muscles; modern microsurgery can provide free composite grafts of muscle, bone, and skin; the tibial fragments can be rapidly and safely stabilised by an external fixator; and, finally, bone can be grafted reliably. Not surprisingly, amputation is considered a failure. There is, however, a tendency for exciting surgical advances to obscure fundamental aims, and there are only two—saving the patient's life and restoring function. Many patients face a long and debilitating period of rehabilitation punctuated by numerous operations. We must therefore know what quality of limb we can expect to achieve by trying to salvage a severely injured leg, and often we do not. The failure to subdivide these severe injuries into relevant subgroups makes nonsense of most published reports and explains the enormous variation in the incidence of non-union, infection, and late amputation.

The recent paper from Ohio by Caudle and Stern, although containing too few cases to draw a final conclusion, should stimulate us to redefine the place of primary amputation.¹ Using the classification of Gustilo *et al*,² they report the outcome of 63 type III open fractures of the tibia (a wound greater than 14 cm with severe muscle and soft tissue damage). Eleven were type IIIA, in which adequate soft tissue coverage of the fracture site was possible; in three cases non-union developed, but there were no deep infections and no secondary amputations. Of the 42 patients with type IIIB fractures, in which the soft tissue sleeve could not be directly restored, 15 developed non-union, 12 had associated deep infection, and seven required secondary amputation. There were only nine type IIIC cases, in which the open fracture was complicated by an arterial injury, but seven of the patients required secondary amputation and the two others had a poor functional result.

Poor results after vascular repair associated with severe open tibial fractures were also reported by Lange *et al*³; six of 12 cases required secondary amputation. These authors considered that an ischaemic time of over six hours and the disruption of the posterior tibial nerve were absolute indications for primary amputation. Other factors that produced a poor outcome were crush injuries in the presence of segmental fractures of the tibial shaft.

Henry De Mondeville in the fourteenth century said of his contemporaries, "More surgeons know how to cause suppuration than to heal a wound." Will our epitaph read,

"They knew how to operate but not how to restore function?" Unfortunately it requires more judgment and courage to do a primary amputation than it does to try to salvage the limb of a patient with a severe open fracture of the tibia.

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- 1 Caudle RJ, Stern PJ. Severe open fractures of the tibia. *J Bone Joint Surg [Am]* 1987;69:801-7.
- 2 Gustilo RB, Mendoza RM, Williams DN. Problems in the management of type III (severe) open fractures. A new classification of type III open fractures. *J Trauma* 1984;24:742-6.
- 3 Lange RH, Bach AW, Hansen SR Jr, Johansen KH. Open tibial fractures with associated vascular injuries. Prognosis for limb salvage. *J Trauma* 1985;25:203-8.

Adenovirus gastroenteritis

Adenoviruses are ubiquitous agents that infect people of all ages. Forty one antigenically distinct types (serotypes) of virus have been recognised, and most are readily recovered from the faeces. Adenoviruses have thus long been suspected to cause diarrhoea, but establishing a causal link has been difficult because of long term asymptomatic shedding of certain serotypes from the stools after adenovirus respiratory infection.¹ The confusion has been cleared by the discovery in stools of adenoviruses that could be visualised by electron microscopy but not grown in conventional cell cultures² and by the development of classification schemes that subgroup adenovirus serotypes into six groups (A-F).³ Thus we now know that the non-cultivable adenoviruses in the stools belong to a distinct group (F) and that these group F adenoviruses (serotypes 40 and 41)⁴ are strongly associated with diarrhoeal disease in children.^{5,6}

Disease develops after an incubation period of 8-10 days,⁷ and diarrhoea is the most prominent symptom.^{5,7} Stools are watery and non-bloody,⁵ and diarrhoea continues for seven to eight days.^{5,7,8} Diarrhoea induced by adenovirus type 41 may be more protracted than disease caused by adenovirus type 40.⁵ Vomiting occurs one to two days after the onset of diarrhoea⁵ in about four fifths of patients^{7,8} but is usually mild and lasts for only about two days.⁵ Fever also occurs in 40-90% of patients^{5,8} with a mean duration of two to three days.⁵ Severe dehydration is rare,⁵ but a death has been described.⁹ When necessary, patients can be rehydrated with standard oral rehydration schemes.¹⁰ Respiratory symptoms have been reported to be common by some⁸ but rare by others⁵—but group F adenovirus antigen has not been detected in respiratory secretions from patients with diarrhoea caused by group F adenoviruses.³ In one series temporary secondary lactose intolerance was reported in three of 32 patients and gluten intolerance in one of 32 after diarrhoea caused by group F adenoviruses.¹⁰

Most infections caused by group F adenoviruses occur in children aged under 2 years^{5,6,11} and in contrast to infections with rotaviruses show no important seasonal variation.^{5,6,11} Virus is excreted in the stool for seven to 14 days^{5,7} and presumably is spread by the faecal oral route. Transmission to family contacts (parents and older siblings) is rare,^{7,12}