# 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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# 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 cultures[2] 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 diarrhoea[5] 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 some[8] but rare by others[5]—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]

unlike with rotavirus. Outbreaks of diarrhoea caused by group F adenoviruses have, however, occurred among young children in closed communities.[2 7 13]

In the developed world, after rotaviruses, group F adenoviruses are the most common viruses associated with infantile gastroenteritis and are detected in between 4%[11] and 8%[5] of stools from children with diarrhoea. Seroepidemiology shows that they are common throughout the world,[14] but their importance in developing countries is not clear. One study from Brazil showed that they are present in only 2% of children with diarrhoea.[15]

Infection with group F adenovirus can be presumptively diagnosed when adenovirus is seen in stool specimens by electron microscopy but the virus fails to grow in conventional cultures. Adenoviruses from other groups may also fail to grow,[3] however, which makes this criterion unreliable as well as retrospective. Immunoassays have been developed[16-18] to identify specifically group F adenoviruses in stools, and with the development of monoclonal antibodies to such viruses[19 20] such tests should become more widely available.

In conclusion, two adenovirus serotypes (types 40 and 41) are an important cause of diarrhoea in young children. Many other adenovirus serotypes are also shed in faeces and are sometimes detected in diarrhoeic stools but are not proved causal agents. There is therefore a clear need for definitive tests for group F adenoviruses to allow accurate diagnosis of adenovirus gastroenteritis.

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# The downs and ups of infant mortality

The death rates of young children are, in my opinion, among the most important studies in sanitary science. In the first place, their tender young lives, as compared with the more hardened and acclimatised lives of the adult population, furnish a very sensitive test of sanitary circumstances; so that differences of the infantile death rate are, under certain qualifications, the best proof of differences of household condition in any number of compared districts. And secondly, those places where infants are most apt to die are necessarily the places where survivors are most apt to be sickly....[1]

JOHN SIMON, 1858

Infant mortality has long been regarded as a sensitive indicator of the state of the population's health. News of the rise in the infant mortality rate (mortality under one year after live birth) for England and Wales from 9·4 for every 1000 live births in 1985 to 9·6 in 1986 was announced by the Office of Population Censuses and Surveys on December 15[2] but emerged only slowly into the public and political consciousness. The rise provoked varied responses, ranging from the suggestion that "it may well be that it is a statistical error"[3] to talk of a link with the plight of children awaiting cardiac operations and the financial problems of the Hospital for Sick Children at Great Ormond Street.[4]

It is ironic that the accuracy of the infant mortality rate should be questioned. Although they are not without problems, birth and death registration data are probably more reliable than the statistics about National Health Service activity[5] that are so often quoted by politicians "backwards and forwards like tennis balls,"[6] and, unlike these statistics, they are related to defined populations.[7]

The figure shows the infant and perinatal (stillbirths plus mortality in the first week after live birth) mortality rates together with the components into which they are commonly divided. The rates for 1986 were not a bolt from the blue but a continuation of trends already apparent from preceding years. By 1985 early neonatal mortality (mortality in the first week after live birth) was no longer falling as rapidly as in the late 1970s and early 1980s, and the same pattern can be seen in the stillbirth rate. As a result perinatal mortality, which fell by an average of 8·0% annually between 1975 and 1983, fell by only 3·0% in 1984, 3·2% in 1985, and 2·1% in 1986. In contrast, the postneonatal mortality rate (mortality over 1 month of age but under 1 year) remained static apart from minor fluctuations between 1976 and 1982 and then fell in 1983 and 1984. Although the small rise that followed in 1985 was unremarkable in itself, it meant that the larger rise in 1986 was not entirely unexpected.

Last time the infant mortality rate went up, in 1970, the picture was different: the rise occurred in the early neonatal period. Subsequent analysis showed that the high mortality was largely concentrated in the second quarter of 1970.[8] It was suggested that the severe influenza epidemic in late 1969 and early 1970 may have adversely affected women in the first trimester of pregnancy and thus led to an increased incidence of low birthweight[9] and mortality[8] in their babies.

There is also little parallel with the rise in perinatal mortality in Wales in 1981. This increase, which led to a major inquiry,[10] followed an exceptionally low rate in 1980. The rate for 1982 was, however, in line with the downward trend seen in the late 1970s. As Wales is about the same size as an average English NHS region its infant and perinatal