At times attention has been given to the question of the best age at which to operate upon unstable feet, and lately discussions have again centred on this point. On the one hand it is thought that bony fusion cannot occur between tarsal bones which are largely cartilaginous, that deformity may consequently recur, and that the interference with growing bone may in itself cause deformity. While it is true that a full stabilising operation should not be performed on little children's feet, it is none the less possible by suitable less radical operations to ensure that no child carries the burden of an "iron" in addition to the burden of paralysis. Certainly I have had no cause to regret this policy since adopting it twenty years ago. Operations of a conservative nature have accordingly been carried out on the feet of children from five years of age upwards (the need does not arise earlier). They have been designed to render the foot stable without prejudice to growth or to a subsequent triple stabilising arthrodesis. In surprisingly few instances has a subsequent arthrodesis been necessary. Deformity has not recurred and growth has not been disturbed. It also appears that in the course of time fibrous ankylosis becomes bony.

Apart from sentimental and economic reasons against their use, it must be remembered that no appliance will prevent the progressive postural and structural calcaneus deformity which results from paralysis of the gastrocnemius and soleus. Nor can a child with an unstable foot and poor muscular control of the knee dispense with a caliper: give him a stable foot and he will need no appliance at all. After all, we do not want our splints to add to the child's handicap in life. The story told to the British Orthopaedic Association in 1934 (when this policy and procedure were first described) is not without its moral. It concerns a boy of ten with a six-year paralysis affecting both legs. He wore "irons"; he attended a special school for physically defective children; the school was about half a mile from his home. The city, in its benevolence, provided a small bus to take the children to and from the school. The bus could not hold all the children and so had to make two journeys each morning and each evening. Our little patient always travelled home on the first bus. Leaving the bus, he went into the house, quickly discarded his boots and irons and put on gym shoes. Then came the high-spot of his day; by hurrying back to school he was able to cheer the second bus-load on its way.

Although splints have their place, they should be kept in it. They should be used as a support and protection during the two or three years necessary to assess the future. The value of some surgical operations remains in doubt while that of others is beyond dispute. Stabilisation of the foot is in the latter group.

BRYAN MCFARLAND.

MECHANISM OF CORD INJURY WITHOUT VERTEBRAL DISLOCATION

One of the most puzzling features of injuries of the cervical spine is the lack of correlation between the severity of the cord lesion and the vertebral injury. In this issue of the Journal there is a contribution by Taylor on the mechanism of traumatic paraplegia without demonstrable injury of the cervical vertebrae. Until recently it was accepted that the cord lesion in this type of injury was caused by a transient dislocation and that reduction occurred by muscular action immediately the flexion violence ceased. This view is no longer tenable and it is now clear that even when there is no dislocation of the vertebrae the cord may be damaged by a massive extrusion of a disc or by forcible hyperextension of the neck.

In previous papers Taylor and Blackwood, and the present writer, described the mechanism of hyperextension injuries. We believed then that the cord injury was caused by a transient and often minor dislocation in extension, for post-mortem examination revealed a complete rupture of the anterior longitudinal ligament and the intervertebral disc. In his present paper Taylor reports a fatal case of paraplegia from a hyperextension injury where after a thorough post-mortem examination no abnormality could be demonstrated in the cervical spine. He then describes a series of experiments which go far to prove that

the cord injury in his case was caused by the interlaminar ligaments bulging into the spinal canal when the neck was forcibly hyperextended. He also suggests that this is the usual mechanism of the cord lesion in hyperextension injuries.

There must, however, be other factors which render the cord susceptible to injury. If it were not so, this might be a common catastrophe, for Taylor demonstrated considerable bulging of the interlaminar ligaments when the neck of a young adult was hyperextended. We know that paraplegia seldom complicates a hyperextension injury in a young adult and that most of those afflicted have advanced osteoarthritis of the cervical vertebrae. It has long been known that the cord may be compressed by osteophytes in the absence of any injury, and the margin of safety for the cord in a patient with advanced arthritic changes in the neck may be small indeed. Morton (*Journal of Bone and Joint Surgery*, 18, 893) observed that the symptoms of incipient paraplegia secondary to osteoarthritis of the cervical spine were aggravated by hyperextending the neck and that a partial spinal block was sometimes made complete by this manoeuvre. There are therefore sound reasons for accepting Taylor's suggestion that paraplegia in hyperextension injuries of the neck is caused by the cord being trapped between the bulging interlaminar ligament behind and the degenerate disc or adjacent osteophyte in front.

It is likely that this is the usual cause of paraplegia in patients in whom there is no radiographic evidence of vertebral injury or dislocation. But it is not the only cause. The cord may be contused by a massive disc protrusion as a result of a hyperflexion injury. For this reason the writer does not agree with Taylor that "cases of paraplegia without radiographic evidence of bone damage or displacement should be regarded as injuries caused by forcible hyperextension and treated as such by immobilisation in slight flexion." This position may cause further damage to the cord if it is kinked over an extruded disc. The problem is not as difficult as it appears, for careful examination of the patient will often reveal an injury of the face or forehead in a hyperextension injury, and of the vertex of the head in a hyperflexion injury. If in doubt it is safe to immobilise the neck in the neutral position, which cannot cause further damage to the cord by kinking over a disc protrusion or by bulging of the interlaminar ligament.

ROLAND BARNES.

**JOINT MEETING OF THE ORTHOPAEDIC
ASSOCIATIONS OF THE BRITISH COMMONWEALTH
AND THE UNITED STATES OF AMERICA**

A joint meeting of the Orthopaedic Associations of the English-speaking world will be held in London in the first week of July 1952—thus fulfilling the hopes and aspirations of British and American orthopaedic surgeons who met in London after the first world war, and met again with the surgeons of Canada after the second war. This time every nation of the English-speaking world will be represented. We will welcome them all; and we hope to honour them all.