aided by eliciting a history of sudden strain or local trauma. Whereas an intra-abdominal mass disappears with contraction of the abdominal muscles, a haematoma of the rectus sheath usually remains palpable and indeed may become more discrete. Oddly enough there is often considerable delay before a tell-tale bruise appears in the skin over the mass which clinches the diagnosis.

Treatment varies according to circumstances. A small haematoma which is obviously subsiding should be left alone to resolve spontaneously. A large tense mass which is increasing in size is best managed by surgical evacuation. This not only allows the bleeding vessels to be ligated, but also gives dramatic relief of pain.

Orthopaedic Surgery in Spastic Paralysis

The relative merits of surgery and physical therapy in the management of children with spastic paralysis have been fiercely argued for the last 50 years. In the early part of this century ill-advised and indiscriminate surgery on nerves and tendons without proper analysis of the paralysis, deformity, and function in the affected limb often did as much harm as good. As a consequence there was a reaction towards reliance on physiotherapy alone, and this often resulted in a deformity and disability that careful and appropriate surgery could have prevented. In recent years the dividing lines between medicine and surgery have become much less sharp, and in the management of cerebral palsy the combined attentions of paediatrician, physiotherapist or physical medicine specialist, and orthopaedic surgeon can achieve more than any one specialist working alone.

The special place of orthopaedic surgery is in the management of deformity. Traditionally, splints, callipers, and braces have been used to try to prevent deformity and to improve function. Deformities that develop despite adequate physiotherapy and splintage are then corrected by reconstructive or salvage surgery. All too often, however, the deformities are so severe by the time that the orthopaedic surgeon is consulted that full correction cannot be achieved and the final functional result is unsatisfactory.

Little is known of the mechanisms that lead to the development of deformity in spastic paralysis. W. J. W. Sharrard[1] has pointed out that the intense overactivity of strong spastic muscles so often dominates the clinical picture that it may be forgotten that there is often functional weakness of the opponent muscles. Functional weakness of the dorsiflexors of the ankle may result in fixed equinus deformity, however efficient the splintage and however vigorously physiotherapy is pursued. When the dorsiflexors of the ankle are strong deformity often fails to develop, even when the calf muscles are severely spastic. This suggests that spastic paralysis has something in common with poliomyelitis, in which muscle imbalance produces deformity in the same way.

Once fixed deformity has developed the opposing muscles become progressively less efficient, and further increase of the deformity is inevitable. This concept suggests that there is a place for preventive surgery in correcting deformity before it becomes disabling and in trying to achieve a balance of muscle action. Surgery is indicated when the ankle, with the knee extended, cannot be dorsiflexed beyond a right-angled position.

Gastrocnemius recession or elongation of the tendo calcaneus achieves the triple effect of improving dorsiflexion, lessening the strength of the calf muscles, and reducing the input of sensory impulses from the tendo calcaneus to the spinal cord nuclei. Adductor tenotomy, with or without partial division of the obturator nerve, has been shown[2] to prevent dislocation of the hip in all but a few patients. Ilio-psoas transplantation may be used to prevent dislocation and to stabilize the hip in those in whom the hip adductors are severely paralysed. There are fewer indications for surgery in the upper limb, but it can often improve function if correctly applied.[3]

Carefully planned operations based on a proper evaluation of muscle function and potential deformity can allow a child to obtain the best possible function under the guidance of physiotherapist and paediatrician. Splints may often be avoided or discarded. The ugly, ape-like posture that has been regarded as the hallmark of the spastic child, but which is often the sign of uncorrected fixed deformity, need never develop. G. A. Pollock[4] has written that "orthopaedic surgery has a worth-while contribution to make in the treatment of cerebral palsy. When the cases are selected with care, the appropriate orthopaedic measures are skilfully performed, and when the patients are adequately supervised afterwards the benefits of surgery are greater than those provided by any other treatment, and they are achieved more quickly."

Extensive division of soft tissues to correct a deformity that has arisen in adults after traumatic paraplegia or in disseminated sclerosis may be well worth while, as has been shown by Evans[5] and by Hardy.[6] Surgery is less often needed in cases of hemiplegia in adults when physiotherapy and splintage after the acute episode have succeeded in preventing deformity, but sometimes a judiciously performed tenotomy or neurectomy (which may be able to be done under local anaesthesia) can confer a benefit which is out of all proportion to the extent of the operation.

[1] Sharrard, W. J. W., *Proc. roy. Soc. Med.*, 1964, **57**, 724.
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[3] Samilson, R. L., *Develop. Med. Child. Neurol.*, 1967, **9**, 109.
[4] Pollock, G. A., *J. Bone Jt Surg.*, 1962, **44B**, 68.
[5] Evans, D. K., ibid., 1963, **45B**, 616.
[6] Hardy, A. G., *Proc. roy. Soc. Med.*, 1959, **52**, 802.

Rheumatic Fever and Streptococcal Infection

The aetiological relationship between group A β-haemolytic streptococci and rheumatic fever is now firmly established. This is based on three main features. Firstly, there is the epidemiological relationship between upper respiratory infections with these organisms and the subsequent development of an acute attack of rheumatic fever.[1] Secondly, serological evidence of a recent streptococcal infection can be obtained in almost all patients with rheumatic fever if three or more antibodies to streptococcal products are looked for.[2] Thirdly, attacks of rheumatic fever may be prevented by the prophy-

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[4] Cayeux, P., Panijel, J., Cluzan, R., and Levillain, R., *Nature (Lond.)*, 1966, **212**, 688.
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lactic administration of antibiotics.[3] Nevertheless, the most convincing piece of evidence is still lacking—namely, the reproduction of the disease in animals by infection with an appropriate strain of streptococcus. For this reason the recent claim by workers at the Pasteur Institute in Paris[4] to have induced arthritis and carditis in mice infected with streptococci is of unusual interest.

Previous attempts to produce similar lesions in mice have failed because most strains of streptococci contain a proportion of highly virulent individuals (BM organisms) which kill the animals before lesions have time to develop. Strains deficient in these BM variants (so-called vm organisms) also fail to induce lesions because they are rapidly eliminated by the macrophages in the host. P. Cayeux and his colleagues[4] report having overcome this drawback by employing strains of streptococci relatively poor in BM organisms and by suppressing the activity of the host macrophages by treating the animals with a rabbit antimouse-macrophage antiserum one hour before injection of the organisms. Of 300 mice treated in this way 6% developed an arthritis of the wrists and ankles and about 50% enlargement of the heart. On histological examination the affected synovia were infiltrated with lymphocytes, plasma cells, and disintegrated polymorphs, and the affected hearts showed foci of necrosis and scattered areas of histiocytic infiltration. Both by microscopy as well as by bacteriological culture organisms were readily found in all the lesions.

Before these findings can be accepted as throwing light on the relationship between streptococcal infection and rheumatic fever the important differences between the human and mouse diseases must not be overlooked. In man there is a highly characteristic quiescent interval of two to three weeks between the primary streptococcal infection and the clinical appearance of joint or cardiac symptoms. In the mouse, on the other hand, arthritis was found to occur occasionally as early as the second day. In man, when special steps are taken to avoid contamination, the lesions are invariably sterile[5]; in the mouse they are obviously the result of local invasion by the living organisms. Rheumatic fever, even in severe epidemics of streptococcal infection, rarely affects more than 3% of the patients; in the infected mice the heart was affected in 50% of cases. Finally, the histological nature of the experimental lesions bore no resemblance either in the joints or in the heart to those of the rheumatic person. Thus readers may well conclude that the mechanism relating streptococci to rheumatic fever has yet to be produced in the experimental animal.

Carcinoma of Hypopharynx and Cervical Oesophagus

Carcinoma of the hypopharynx (post-cricoid) and the cervical oesophagus is fortunately uncommon. When it presents the primary lesion is usually extensive and often accompanied by metastatic nodes. Treatment, whether by radiotherapy or surgery,[1] has been generally unsatisfactory and 50% of patients have died after a distressing illness within the first year of treatment. Because fibrous strictures, oedema, and local recurrence commonly occurred after irradiation, the predominant symptom of dysphagia was not relieved, and, since tracheal or laryngeal obstruction supervened, the patient was left to die with a tracheostomy and permanent feeding through an oesophageal catheter or a gastrostomy. Not only was the rate of long-term cure poor but palliation was also unsatisfactory.

Surgeons have argued that pharyngolaryngectomy combined with unilateral or bilateral neck dissection in continuity, though carrying a mortality rate of 3% to 12%,[2 3] offered a better chance of survival. Pharyngeal reconstruction by skin flaps took a minimum of six weeks in non-irradiated cases and much longer in those previously irradiated—so long, in fact, that many reconstructions were never completed because of local recurrences and metastases. To overcome this delay in the restoration of swallowing immediate methods of reconstruction were devised using fascia or skin grafts round moulds, pieces of trachea, vascular homografts, and plastic prostheses, but all were bedevilled by salivary fistulas and strictures.[3-6] Further, of those patients suitable for surgery less than 20% survived five years, only a third more than two years,[7] and many died unrelieved of distress. Not surprisingly, the disease has been regarded as virtually incurable, and a safe, immediate method of restoring swallowing that will serve for what remains of the patient's life has been sought as a palliative.

Since pedicle grafts of stomach, jejunum, or colon had previously been used successfully for immediate oesophageal replacement, the method was extended to the pharynx.[1 7-11] Subcutaneous and retrosternal routes to by-pass the gullet were first used, but, as recurrences in the oesophageal stump were common, the initial excision was extended to include the whole oesophagus and the tunnel so created was used for routing the gastric[12-14] and colonic transplants. Free transplants of jejunum and colon, revascularized by anastomosis of the mesenteric vessels to convenient vessels in the neck, have also been used.[6 11]

However, all transplantations are formidable procedures and carry a mortality of 30%–50%. That is a high price to pay for palliation when it is known that two-thirds of the patients will die within two years, no matter what the extent of the excision. Recently a non-slip Polyvinyl tube has been made to replace the pharynx after a wide cervical excision.[15] The operative mortality from the procedure is low and the palliative results good, but after it patients must mince all the meat they eat. Occasionally the anterior parts of the larynx and trachea can be preserved to form a new anterior wall for the pharynx, the posterior wall being fashioned by a skin graft on to the prevertebral muscles.[11 16-18] A major sequel to pharyngolaryngectomy is a loss of voice, but after replacement with a colon graft several patients have developed a useful "oesophageal" voice.[19]

All these newer techniques have failed to offer a satisfactory outcome for most patients, and it is hoped that more modern radiotherapy techniques and cytotoxic drugs may offer better prospects.

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