SUMMARY

The interplay between stretch reflex and flexion reflex is discussed. A drug, which should have an inhibiting effect on the polysynaptic flexion reflex and on the gamma afferents was examined in walking tests in six children with cerebral palsy. The improvement obtained was probably due to a training effect. In 21 patients with different spinal cord lesions the flexion reflex of the lower limb was examined electromyographically by way of an electric nociceptive stimulus with needles inserted under the skin of the sole of the foot. This was done in a double-blind test, but no objective measurement could be obtained.

The flexion reflex was only obtained when the stimulus had a moving or nociceptive character. It was concluded that the flexion reflex probably has more qualities than withdrawal in defence. The importance of the widened receptive field in patients with paraplegia and a moving character of stimulus are stressed.

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SPASTICITY IN THE UPPER LIMB

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ONE of the results of the increase of wealth of society is that many more people are now owning and using motor-cycles and cars. This has resulted in an increased number of road accidents, some of these naturally involve the cervical spine and cause tetraplegia. The better understanding of the treatment of these injuries during the last 20 years, particularly with reference to the management of the chest and urinary tracts, means that many more tetraplegics are surviving. The long-term prognosis depends largely upon preventing chest and renal infections and pressure sores. The useful contribution that they can make to society and to leading a fuller and freer life depends almost entirely upon the useful function

that they can achieve in their arms and hands. Their ability to use their upper limbs is impaired by the presence of spasticity, contractures, lower motor neurone damage, pain and local trauma. The present paper is an analysis of the relative contribution that these factors make in those tetraplegics seen at the Liverpool Regional Paraplegic Centre during the last year.

Spasticity is a variable phenomenon which depends largely upon the level and the completeness of the injury to the cord. It may produce contractures as shown by limitation of abduction and internal rotation. The elbow may show limitation of extension, the wrist loss of full dorsiflexion. The hand shows a quite striking progression of changes:

1. Loss of flexion at the interphalangeal joints of the thumb and fingers.
2. Swelling of the hands and fingers with loss of the skin creases.
3. Swelling of the proximal interphalangeal joints.
4. Clawing of the hand, sometimes with hyperextension of the proximal phalanges, sometimes with tight flexion of the distal phalanges into the palm, the thumb may also be gripped and tightly clenched by the other fingers.
5. Clubbing of the finger nails.
6. Fixed deformity.

These changes in the joints are aggravated by lower neurone damage and are not the result of the spasticity alone, they may in turn produce pain and aggravate the spasticity. It is only by understanding the causes and relative contribution that the above features are making to the disability of the upper limb that any rational therapy can be devised.

Discussion

Sir Ludwig Guttmann (England). Professor Whitteridge warned us at the beginning of his masterly exposition of the physiological background of spasticity that his talk would be destructive and Dr. Rushworth warned that his study on the pathophysiology would add to the existing confusion. You all will agree with me that both papers were excellent contributions in elucidating this knotty problem by their emphasis on the importance of the afferent system with special reference to the functions of muscle spindle and Golgi tendon organs The awareness of the importance of afferent impulses arising from peripheral organs on the formation of the various pattern of spasticity has greatly influenced me in my own work on the spinal cord. This has led me to realise, for instance, the importance of posture of the paralysed limbs in the early stages of spinal cord transection on the reflex activity of the isolated cord. Details have been described in my monograph in Volume of Surgery of the *British Medical History of World War II*, in 1953.

Dr. Rushworth referred to Foerster's great pioneer work on posterior rhizotomy in the treatment of spasticity, who recognised the importance of afferent impulses on the motor reflex responses in the treatment of spasticity. Having been closely associated with Foerster for 10 years, and having carried out this operation myself, I can confirm that although the immediate effect of the elimination of all alternate posterior roots section is striking, the effect is not lasting. The spasticity returns in due course after resection of even a great number of posterior roots although in a rather more sluggish and rigid than brisk and dynamic form. Therefore, this method has been abandoned.

One has to consider whether other afferent pathways may be responsible for the return of spasticity after posterior root resection. In the past the question of afferents through the anterior roots was discussed, and I would like to ask Professor Whitteridge and Doctor Rushworth for their views in this respect.

Whitteridge, D. (Scotland). I do not believe in it.

Rushworth, G. (England). I second that. It can be shown in normal cases that if one simply deafferentates the spinal cord one gets rigidity. Professor Whitteridge has told us that one of the effects of deafferentation is degeneration of the input to motor neurons, upsetting the whole balance, and as a result muscular rigidity develops. Some years ago Dr. Oppenheimer, Dr. Hughes and myself described a patient who had an infiltrating glioma of the cervical cord which had cut off all the input to the motor neurons of the spinal cord and this patient had in fact intensely rigid muscles which we were unable to alleviate by any drugs. The only thing that reduced the spasm was complete block of the peripheral nerves or the brachial plexus.

Gurewitch, D. (U.S.A.). I would like to know the mechanics and the workings of that pneumatic splint which was shown by Dr. Pool as used in his study.

Pool, G. M. (Holland). The splint protects the skin against stimuli normally acting to retract the legs. It is a protective splint which is inflated just enough to have something between the skin. It has not a stretching effect but a relaxing effect.

Sir Ludwig. I just cannot understand that. How can this plastic bag prevent impulses from the skin? Is perhaps the leg in a position like a person in space?

Pool, G. M. Probably you could compare it with that. It is a protective air wall which prevents exteroceptive stimuli underneath the plastic splint.

Sir Ludwig. What kind of pressure do you apply to inflate the plastic splint?

Pool, G. M. Only a little.

Silver, J. (England). I, too, have tried the plastic splint. Sir Ludwig has taught us the value of extending the limbs in the early stages to prevent flexor spasms. I have found that if you place the limbs in extension the patient feels much more comfortable in that position. We got these plastic splints from the Air Sea Rescue Organisation. When the splints are inflated they keep the limbs straight, and tetraplegic patients have felt much more comfortable for a short time.

Cibeira, J. (Argentine). Is there any neurophysiological explanation for paraplegia in extension and flexion?

Sir Ludwig. The old concept was that paraplegia in flexion is an expression of complete spinal cord transection and paraplegia in extension of an incomplete lesion. This concept is no longer valid. The type of reflex-synergy depends greatly on the position of the limbs in the initial stages of paraplegia. Although the flexor group is preponderant in the response of the isolated cord, if from the beginning the paralysed legs are placed in extension and abduction one gets very little flexion response—in fact in many cases of complete transection the result is spasticity in extension. Vice versa, if in incomplete lesions the paralysed legs are neglected by keeping them flexed one can get the most frightful flexion spasticity.

McSweeney, T. (England). I would like to raise one or two small points on Dr. Silver's paper. One is the hand he showed which he quite rightly called the intrinsic miner's hand—the hand with the interosseus palsy. We see a similar condition in complete plexus lesion and in hemiplegics, and we believe that the fundamental lesion, as he suggested, is an intrinsic fibrosis of the small muscles of the hand. Despite an immense amount of surgery particularly in the U.S.A. we know of no really satisfactory solution for this—in fact the more one tries tendon transplants in the hand the more disappointed one becomes. The other small point is the lack of rotation at the shoulder, and I believe apart from the paresis and apart from muscle shortening many of these people, particularly the people over 40 with cervical lesions, develop an adhesive pericapsulitis of the shoulder. The elbow escapes, and the condition is not very dissimilar

from the hand-shoulder syndrome following trauma in people over the age of forty. The only reason I raise this point is the simple expediency of hydrocortisone injections into the shoulder capsule often unfreezes what in reality is an unfreezing process, pericapsulitis is in fact a self-resolving process but it may last something of the order of 18 months. The reason for treating is that the muscle contraction and the inability to use physiotherapy will finally freeze the shoulder to the side, unless you can persist with as much active movement as the patient has or as much passive movement as the physiotherapist can permit him. The other little point about that is that some years ago in the development of the hand-shoulder syndrome as a post-traumatic effect, many orthopædic surgeons were inclined towards a sympathectomy with terribly disappointing results and, no doubt, the same would apply in the treatment of the same condition arising as a result of cervical cord lesions.

THE USE OF DIAZAPEM (VALIUM) IN THE RELIEF OF SPASTICITY

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IN 1946, the short-acting muscle relaxant action of Mephenesin was described by Berger and Bradley (1946), and during the succeeding 20 years there has been a constant search for a drug with a longer action and minimal side-effects which could be of inestimable value as an adjunct to the well-proven uses of physiotherapy and posture in the relief of spasm. Such a drug should be able to help in the relief of discomfort in moderate severe spasticity, aid nursing and physiotherapy procedures, improve the activities of daily living and further reduce the number of patients for whom there would appear to be no alternative but surgical treatment.

In November 1961 the Riker Laboratories held a Symposium on Skeletal Muscle Spasm (1962) in London and, at that time, it had to be admitted that no really effective drug had yet been found. In October 1964, however, DeJak and Lowry (1965) read a paper at the Veterans Administration Thirteenth Clinical Spinal Cord Injury Conference claiming good or excellent results with the use of Diazapem in moderate doses, in 21 out of 27 patients who were suffering from severe spasticity. Their conclusions failed to convince a number of their colleagues who had been so often disappointed in the past and the need for properly controlled double-blind trials was emphasised.

Before and since that date a number of such trials have been published and there is increasing evidence that this drug is of definite value in a considerable number of cases and is worthy of further trial.

EXPERIENCE AT EDENHALL

In 1963 we were already using Diazapem empirically in spinal cord injury patients where the degree of spasticity appeared to be limiting treatment by physiotherapy. In many of these cases the daily programme of active rehabilita-