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PART I

(A) SPASTICITY

THE SPINAL CORD AND SPASTICITY

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THOUGH the arguments for Sherrington's view that spinal shock is due to withdrawal of a predominantly excitatory influence from higher centres have lost nothing of their force, it is perfectly possible that part of the increased spinal activity seen within hours of recovery from spinal shock in animals is due to release from descending inhibition paths. Eccles and Lundberg (1959) have shown that though the monosynaptic flexor reflexes at this time are depressed the polysynaptic reflexes are enhanced. It has also been shown that fusimotor fibres to the muscle spindles of flexor muscles are inactive presumably due to lack of descending impulses.

These changes are overshadowed by the changes in reflex activity which develop over the next three weeks with the onset of spasticity. A number of attempts have been made to test the excitability of reflex centres directly, using electrical stimuli applied to afferent nerves.

An increase in monosynaptic reflexes was found by McCouch, Austin, Liu and Liu (1958), after hemisection of the cord in cats, and Teasdall, Magladery and Ramey (1958), and Teasdall, Villablanca and Magladery (1965) found an increase in polysynaptic reflexes after hemisection. Unfortunately we know little of the mechanisms of this increase in excitability of polysynaptic pathways. The older literature speaks of death of spinal interneurons after cord section. We know complete deafferentation of neurones can give rise to transsynaptic degeneration presumably as a result of disuse, but we know nothing of the cellular mechanisms. Presumably cord section could result in such a degree of deafferentation of some interneurons that they die.

The situation has become more complex since the extraordinary degree of elaboration of the muscle spindle has been realised. The sensitivity of the muscle spindle is controlled by the central nervous system to a greater extent than is any other nerve ending, and it is possible that this control is disturbed in spasticity, so that more excitatory impulses than usual reach the spinal cord. Such a situation would not be detected by applying electrical stimuli direct to afferent nerve trunks.

Recently Van der Meulen, Gilman and Denny Brown (1966) have shown that in spastic monkeys there is an increase in the sensitivity to stretch of extensor but not flexor muscles following lesions of the corticospinal tract. Denny Brown (1966) thinks that heightened proprioceptive effects in spastic limb are due to an imbalance of a drive to extrafusal fibres, and fusimotor drive to the muscle spindles.

Recent work on the muscle spindle has distinguished between its sensitivity to a movement of stretch, a dynamic or phasic sensitivity, and sensitivity to

maintained stretch, or static sensitivity. It is clear that the largest afferent fibres from muscle in Group Ia come from the primary sensory receptors on the muscle spindles. These end in annulospinal structures on nuclear bags, but also send branches to form similar annulospinal endings on nuclear chain fibres. In addition there are smaller afferent fibres which make up 90 per cent. or more of Group II of the afferent fibres from muscle, which arise from secondary or flower spray endings exclusively found on nuclear chain intrafusal fibres some way away from the central annulospinal endings.

FIG. 1

FIG. 2

Figure 1.—Projection plan of the equatorial region of a large spindle unit of a rectus femoris tendon spindle consisting of three nuclear-bag and six nuclear-chain fibres. Proximal end of equatorial region on the left. Reconstructed from 50 serial 12 μ transverse sections; de Castro silver method.

Figure 2.—Graphs comparing the discharge rates of a primary and a secondary ending in response to applied stretches of 4 mm. to soleus muscles and recorded from dorsal root filaments in different decerebrate cats. Both animals showed good rigidity and had intact motor roots. The muscles were at similar medium resting tensions.

Primary ending conduction rate of impulses 90 m/sec. responses plotted in three upper curves.

○—○ stretch applied at 50 mm./sec.;
x—x 23 mm./sec.;
○—○ 9 mm./sec.

Secondary ending conduction rate of impulses 31 m/sec. responses plotted in three lower curves.

○ - - - ○ stretch applied at 52 mm./sec.;
x - - - x 27 mm./sec.;
○ - - - ○ 11 mm./sec.

The resting discharge rates before stretch are on the extreme left; the stretches were applied between the vertical dotted lines and on the extreme right are the discharge rates at the beginning of the periods of maintained stretch.

Recently Sybil Cooper (1961) began the process of clarifying our understanding of the function of primary and secondary afferent fibres by studying them in the decerebrate cat with intact motor innervation. In this preparation primary afferent fibres identified by their conduction rates were found to be sensitive to phasic stretch, but to be much less sensitive to static stretch. On the other hand, secondary fibres were almost insensitive to the rate of stretching but were very sensitive to static stretch.

Subsequently P. B. C. Matthews (1964) showed that there were in fact two different kinds of efferent fusimotor fibres. One increased the phasic sensitivity of the primary afferents, and had no effect on static sensitivity, whereas the other increased the static sensitivity of both primary and secondary endings. The simplest explanation is that whereas the phasic sensitivity of primary fibres is due to annulospinal endings on nuclear-bag fibres, the static sensitivity of primary endings is due to the annulospinal endings on the nuclear-chain fibre, and that

FIG. 3

Diagrammatic comparison of the responses of 'typical' primary and secondary endings at various stimuli. The drawings are based on the reviewer's personal observations as well as those in the literature. The responses are drawn as if the muscle were under moderate initial stretch and as if there were no fusimotor activity.

phasic or static properties are related to physical difference between nuclear-bag and nuclear-chain intrafusal muscle fibres. It seems likely that there is a larger viscous component in the response to stretch of the nuclear-bag fibre than in the nuclear-chain fibre. Quite recently I. A. Boyd (in the press) has observed a slower rate of shortening and lengthening during contraction in the nuclear-bag than in the nuclear-chain fibres.

Though this is now clear, the difference in the central effects of primary and secondary afferent fibres is still obscure. It is well established that primary endings have monosynaptic connections with motoneurons and that they are responsible for tendon jerks and fast reactions to change of length. They can also show maintained responses when stimulated by static fusimotor fibres and may therefore play an important part in maintained stretch reflexes. The secondary endings have been said to be purely flexor in effect whether they come from flexor or extensor muscles. This view is derived from stimulating the whole of Group II fibres electrically. Group II, however, also includes some afferent fibres from muscular septa and if these fibres are nociceptive, as seems likely, then their central flexion producing effects would be prepotent. The results of Perl (1958) are also consistent with the presence in Group II of an admixture of prepotent nociceptive fibres. In view of the slow rate of change of discharge in these muscles, their activity must be summed centrally over a considerable time, and it is likely that analysis of their activity by single electrical impulses is singularly ineffective. Undoubtedly they have only polysynaptic connections to motoneurons and at present the question of their function must be left open.

Just as we know little of the balance of descending tracts on motoneurons,

we know little of the effects of descending tracts, probably mainly reticulospinal on phasic and static fusimotor neurones.

Finally we must be cautious in applying results from cats directly to man. It is well known that the corticospinal tracts end in interneurons in cats, and few if any fibres end directly on motoneurons. This is not so in monkeys, in whom a large number of corticospinal fibres end on motoneurons, and presumably the same is true in man. The distribution of 'disuse atrophy', to use the old term, may well be different in cats and man. While therefore our present knowledge of mechanisms of spasticity in spinal man is incomplete, there is every reason to hope for a flood of exact information in the near future.

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SOME STUDIES ON THE PATHOPHYSIOLOGY OF SPASTICITY

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MUCH of our understanding of the nature of spasticity has been derived, in the first instance, from experimental work in animals, and it will be necessary to describe briefly some of this before I describe the approaches which we have made to this subject in the human patient.

The experiments of Sherrington (1898) on the origin of decerebrate rigidity in the cat and monkey were an important starting-point. These experiments drew attention again to a curious phenomenon of muscular rigidity which had been observed by Magendie and others and which appeared within a few minutes of severing the brain-stem in many animals. Sherrington demonstrated its dependence upon sensory input to the central nervous system, not from cutaneous receptors but derived from the stiff muscles themselves, so that when appropriate dorsal roots were divided the muscular rigidity was locally abolished. Muscle spindles were already known to be present in skeletal muscles, and, a few years earlier, Sherrington (1893) himself had shown that they were sensory organs. We will