CENTRAL AND SENSORY TRANSMISSION

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I have greatly enjoyed hearing Dr. Feldberg's most interesting paper, and find it particularly entertaining that so much of the newest evidence presented in favour of neurohumoral transmission mechanisms in the central nervous system comes, not only from an electrophysiologist, but from such a high-voltage spark as J. C. Eccles. With reference, however, to the suggestion relayed from Canberra that the direct, Group I inhibitory pathway between antagonistic muscles in a myotatic unit is not, in fact, monosynaptic, but includes an internuncial link, I must state most strongly that this is incorrect. With careful timing of afferent volleys, direct reflex inhibition is well established when the inhibitory precedes the test volley by 0.2 msec., and reaches maximum with a volley interval of 0.5 msec. (3). There are, it is true, other internuncially-relayed inhibitory reflex pathways traversed by impulses of muscle-receptor origin, but the inhibitory pathway between antagonists within the myotatic unit is strictly monosynaptic.

I advise a policy of caution with regard to theories of humoral transmission mechanisms at central synaptic junctions. Probably most of us here feel biased towards the "chemical" viewpoint, if only in the interests of economy of hypothesis. But simply because we would like it that way is not a sufficient reason for accepting it. There are, it is true, a number of suggestive observations, and Dr. Feldberg's first "flash" from Canberra may mean that the existence of one central cholinergic synaptic transmission mechanism has been demonstrated. However, my feeling is that "neurohumorists", if I may call you this, are necessarily so conditioned in favour of chemical transmission, that the sudden rejection of his former creed by an old and formidable electrical antagonist seems to some a sufficient substitute for positive and substantial proof of the existence of such mechanisms in the central nervous system. Take Dr. Shaw's statement read yesterday by Dr. Feldberg: "now that Eccles has abandoned electrical transmission in the central nervous system, there is no reason for thinking it plays a part in ganglionic transmission." I cannot see how Eccles' views on central synapses should in any way affect one's thinking about ganglia. If there is a case for chemical transmission in ganglia, surely it rests solidly on its own feet.

A critical evaluation of the very interesting experiments of Brock, Coombs and Eccles with intracellular recordings from motoneurones (1) reveals that rather sweeping conclusions have been drawn from somewhat slender evidence, as far as transmission mechanisms in monosynaptic pathways are concerned. True, excitatory afferent volleys usually cause depolarization, and inhibitory volleys hyperpolarization, at any rate in some motoneurones. These apparently "active" changes in membrane potential can be explained by a humoral hypothesis, but until the possibility had been tested, they could equally well represent active membrane responses to, say, current flow in different directions. It is not valid to assume identical properties for peripheral axonal and nerve-cell soma

membranes—in fact, there is much direct evidence to the contrary (4, 5). However, an attempt to examine directly this aspect of soma membrane behavior was made last year by Dr. Brock and myself, using double internal micropipettes, stimulating across the membrane with one and recording by way of the other channel (2). Active responses to rectangular depolarizing current pulses were always seen, but none to hyperpolarizing pulses, even though the latter "inhibited" reflex discharge and antidromic invasion. In the light of these observations it therefore seems valid to say that hyperpolarization associated with monosynaptic central inhibition is not likely to be a post-synaptic response triggered simply by a specifically-orientated flow of electric current about presynaptic terminals. A "humoral" mechanism for this aspect of reflex inhibition seems more probable, though unproven. Still less evidence is available in the case of monosynaptic excitatory action. Evidence of a more positive kind will be necessary before the nature of these transmission processes can be properly understood.

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