Achilles Tendon Reflex in Hypothyroidism and Myxoedema

SIR,—During the relaxation phase of a tendon reflex the muscle is electrically silent. "Brisk relaxation" of a muscle therefore cannot be caused by a lesion of the central nervous system as suggested by Dr. D. Maclean and others (14 April, p. 57). A more reasonable conclusion would be that hypothyroidism and hypothyroidism do not invariably delay muscle relaxation.—I am, etc.,

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Malignant Hyperpyrexia

SIR,—It is interesting to see in the letter from Dr. M. A. Denborough and others (24 March, p. 738) that their earliest recorded case of death from malignant hyperpyrexia occurred in 1921. Brit and Kalon mentioned another fatal case of malignant hyperpyrexia occurring in 1922. Both cases, of course, occurred well before the introduction of suc- cinyldicholine and halothane. These anaesthetic agents cannot therefore be specifically incriminated, as suggested by Dr. C. Langton Hewer (24 March, p. 738). Ether, cyclopropane, tri- chloroethylene, and methoxyflurane have probably also been responsible for inducing the syndrome, though it is rarely possible to be absolutely sure of this because of the multiplicity of drugs commonly used during anaesthetic procedures. Ether may well be less dangerous than the other agents, as halothane is potent in uncoupling oxidative phosphorylation in liver mito- chondria, the relative potencies being related to the partition coefficient of the agents between lipids and water. Dr. Hewer is, I am sure, correct in maintaining the separation of malignant hyperpyrexia from the syndrome of late ether convulsions. In fact he made an early con- clusion to that effect in his paper on the introduction of ether convulsions. It is, however, unlikely that ether convulsions had a single cause. The earlier literature on operative and post- operative hyperpyrexia and its fits contained many reports of pre-existing hyperpyrexia, hot operating theatres, excessive draping of the patient, and atropine premedication. Agents other than ether were at times responsible for identical convulsions and hyperpyrexia. The major contribution of Dr. Denborough and his colleagues was the recognition of a family of patients developing fatal hyperpyrexia with anaesthesia. Sporadic cases, however, do occur, and I agree with the suggestion that some of the cases recorded in the earlier literature as late ether convulsions may well have been suffering from malignant hyperpyrexia. If ether is a less potent agent, which induces malignant hyperpyrexia reactions less quickly than halothane and suxamethonium, then one may perhaps accept the six cases reported by Guedel as being attributable to malignant hyperpyrexia. Certainly it is not easy to see from what they suffered if not malignant hyperpyrexia, and Brit and Kalow, who have extensively reviewed the literature, accepted these as cases of malignant hyperpyrexia. Nevertheless, I agree that it is impossible to be certain of the diagnosis without the confirmation of a positive family history.

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More important than this discussion, however, is the elucidation of the mechanism underlying the malignant hyperpyrexia reaction. As mentioned previously, contrary to the findings of Ellis et al.1 and of Dr. Denborough and his colleagues (3 February, p. 272), we found no histological abnormality in muscle biopsy specimens from six individuals, all of whom had raised serum creatine kinase levels and relatives of patients who had suffered from malignant hyperpyrexia. In particular "moth-eaten" fibres, and pseudo-cores were not present. We did not find that halothane and suxamethonium inevitably induced the intracellular enzyme creatine kinase in muscle in vivo, which again differed from the findings of Ellis et al.2 Our physiological3 and biochemical4 studies failed to elucidate the underlying pathogenetic mechanism. Further studies of the muscle biochemistry and of the part played by thyroid hormones5 are therefore awaited with great interest.—I am, etc.,

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3 Hewer, C. L., British Medical Journal, 1930, 2, 300.

Treatment of Depression in General Practice

SIR,—The paper by Dr. D. A. W. Johnson (7 April, p. 18) requires comment because of the erroneous and misleading statements in it. The place the validity of many of his data is questionable owing to inadequacies of method. In the second place, even if accepted at face value, his results fail to justify his harsh criticism of his general practitioner colleagues.

Dr. Johnson fails to emphasize the accuracy of the G.P.'s diagnosis of depression. That 73 patients out of 91 interviewees whom satisfied his criteria is remarkable in view of the frequently repeated assertion that the G.P. does not recognize depression. Dr. Johnson then criticized G.P. treatment in terms of drug therapy, psychotherapy, and its involvement in agencies. In the second place, even if accepted at face value, his results fail to justify his harsh criticism of his general practitioner colleagues.

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