CORRESPONDENCE

POTASSIUM DEPLETION AND THE CENTRAL ACTION OF CURARE

Sir,—Dr. Patrick A. Foster is to be congratulated for writing so freely about unsuccessful cases, and his paper ("Potassium Depletion and the Central Action of Curare," Brit. J. Anaesth. (1956), 28, 488) must have been read with great interest by many. I wonder however, whether his cases really illustrate the thesis which he maintains: after all, there are other causes of both central and peripheral neuromuscular depression.

For example, Dr. Foster's Case No. 7 was obviously in a poor state. She had been vomiting for 24 hours, no doubt her ability to breathe was limited by abdominal distension, she had apparently received inadequate intravenous therapy, and was given what may well have been an excessive dose of morphine for one so ill. Not surprisingly, she had "prolonged postoperative sleep" and "had no remembrance of induction or intubation." The comment states, "This is thought to provide an unequivocal instance of a central action of dTc", but, to me, it conveys no such impression. Does one need to postulate such an hypothesis to account for struggling or apparent unconsciousness on administering 10 mg of d-tubocurarine to such a patient?

By the way, is it common practice to administer neostigmine in such large doses? In the three cases in which the total dose is mentioned it is given as 15 mg, 15 mg, and 20 mg. With the doses of d-tubocurarine used, I should have expected 2.5 mg to produce complete reversal of the relaxant and I should have assumed that any subsequent failure to breathe was due to some other cause. Although I often use d-tubocurarine in comparable doses, my custom is to give an initial dose of neostigmine, 1 mg, followed, if necessary, by not more than two further doses of 0.5 mg. The total dose of 2 mg is only exceeded with the greatest reluctance. Surely doses of the order mentioned may have contributed substantially to the apnoea and must have played Old Harry with the heart and intestinal tract, to say nothing of every other organ in the body.

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Sir,—As with any new and unanticipated event, the records thereof are not as full of relevant data, nor as convincing, as could be wished. The article was an attempt to define a clinical picture from fragments collected in a number of cases of apparently similar nature. Whether the thesis is correct is at present a matter of opinion, awaiting further evidence. But there are not many known causes of persistent neuromuscular block by antidepolarizing agents where efficient circulation and ventilation have been maintained, where nitrous oxide without hypoxia has been the only narcotic, and where no postmortem lesion could be found.

I regret that case report No. 7, receiving Dr. Armstrong-Davison's attention, may have conveyed a wrong impression. Dr. Armstrong-Davison follows the pre-operative and post-operative course of his patients perhaps more thoroughly than many other anaesthetists, and his criticism is thus valued, but not here apposite. This lady was in good condition, and no extensive pre-operative preparation was considered necessary. She was not grossly distended nor dehydrated; in fact, she was the type of case that would not normally cause the anaesthetist anxiety, and who would have been unremarkable had the unorthodox technique not been used.

As an observer, not the administrator, of the anaesthetic, I was able to remark, I hope accurately, on the disappearance of eye signs usually thought to be absent only in relatively deep planes of unconsciousness. She came to the operating theatre obviously fully in touch with her surroundings, with subsequent recall of events com-