activity after 25 days storage at 6°C. A few observations I have made on dried plasma show that, on reconstitution, pseudocholinesterase activity is within the normal range. If this finding is substantiated an intravenous infusion of plasma will be a valuable therapeutic measure as it is readily available, avoids the necessity for crossmatching and blood grouping, and the difficulty in obtaining fresh blood or plasma.

I suggest that in treating a patient who has a prolonged apnoea following suxamethonium, a nerve stimulator is of value, to confirm not the type but the degree of the neuromuscular block, the nature of which is complex and poorly understood. No anticholinesterase drugs should be administered but if, because of temporal or other factors, a hastened recovery is required, an infusion of plasma should be given intravenously.

GORDON H. BUSH
Liverpool

REFERENCES

Fromberg, H. F., Mannheimer, W. K., and Keats, A. S. (1960). Succinyldicholine esterase activity in blood. *Anesthesiology*, **21**, 64.

Harrison, B. L., Seward, E. H., and Skinner, L. C. (1954). Prolonged Scoline apnoea treated by blood transfusion. *Anaesthesia*, **9**, 21.

ADRENALINE AND HALOTHANE

Sir,—I have had my attention drawn to a frank report of two cases of cardiac arrest from adrenaline infiltration during halothane anaesthesia (*Brit. J. Anaesth.*, 1963, **35**, 51). There must be many similar cases of cardiac arrest which have remained unrecorded and your report of two case records is indeed timely and helpful. For some considerable time it has been customary in my department, when adrenaline infiltration into the para-vaginal tissues is about to be started, to ask the question "Is the patient receiving a halogenated anaesthetic?" I suggest that this simple drill might save near-disasters, especially with changes of surgeons and anaesthetists.

Recently recordings have been made on the effect on blood pressure readings of the infiltration of approximately 30 ml of 1:100,000 adrenaline in saline solution. Systolic pressures have risen as much as 50–60 mm of mercury in 2 minutes.

The blood pressure recordings with the times for rise and fall will be presented for publication together with other observations on the infiltration of adrenaline solutions into the para-vaginal tissues when non-halogenated anaesthetic agents are used.

J. R. E. JAMES
Carmarthen

HEAD'S "PARADOXICAL" REFLEX

Sir,—Dr. Murphy's discussion of Head's "paradoxical" reflex (*Brit. J. Anaesth.*, 1963, **35**, 277), and the failure to identify it in conscious man (Widdicombe, 1963), stimulates the suggestion that this reflex may be recognizable under certain conditions associated with anaesthesia. A typical situation is as follows: an elderly patient is given a relative overdose of morphine, sufficient to lower respiratory rate to 8–12 per minute, and after a "sleep" dose of thiopentone there is the expected apnoea. Respiration does not start spontaneously; but a single gentle pulmonary inflation will often produce a strong, sometimes prolonged, inspiratory effort. The effect can usually be repeated until spontaneous respiration restarts. I suggest that this may be Head's reflex *in action*, and the hypothesis could be made that morphine and thiopentone, while reducing the excitability of central inspiratory and/or expiratory neurons—or merely by decreasing the impulse traffic in their vicinity (Delisle Burns, 1963)—reveals this "inspiration-reinforcing" reflex partly by damping down Hering-Breuer and other vagal afferent activity; this differentiating action of morphine would thus resemble the effects of vagal cooling demonstrated by Head.

Since the Head reflex would appear earlier in inspiration and at a lower lung volume than the Hering-Breuer reflexes, its successful demonstration in man breathing normally seems a formidable proposition. Its apparent disappearance shortly after birth may merely indicate concealment, since this reflex could be partly concerned in the occasional deep breaths noted both during ether anaesthesia and normal respiration. "Paradoxical" is an unfortunate term, of course, since this refers only to its apparent opposition to the better known vagal inhibitory reflexes.

R. A. MILLAR
Cambridge

REFERENCES

Widdicombe, J. G. (1963). Respiratory reflexes from the lungs. *Brit. med. Bull.*, **19**, 15.

Delisle Burns, B. (1963). The central control of respiratory movements. *Brit. med. Bull.*, **19**, 7.

SUXAMETHONIUM APNOEA

Sir,—In the case of prolonged suxamethonium apnoea described by Vickers (*Brit. J. Anaesth.*, 1963, **35**, 260) the demonstration of post-tetanic facilitation after stimulation of the ulnar nerve was taken as being indicative of a non-depolarizing neuromuscular block, and the worsening of neuromuscular transmission after administration of an anticholinesterase was considered to be a perverse response.

It has recently been shown (Wislicki and Benzakein, 1963) that post-tetanic facilitation does occur with depolarizing blocking agents too, though only during lighter stages of their action, and not, as with non-depolarizing substances, when the block is complete.

In many instances of prolonged suxamethonium block, not all muscle fibres will be paralyzed and, apparently, those not blocked will show post-tetanic facilitation when stimulated through their motor nerve. This finding does not, therefore, exclude the presence of a depolarizing type of block and it cannot constitute the decisive criterion for the administration of neostigmine, edrophonium or other anticholinesterases.

LEO WISLICKI,
*Manchester**

REFERENCE

Wislicki, L., and Benzakein, F. (1963). Post-tetanic relief of neuromuscular block. *Arch. int. pharmacodyn.*, **142**, 23.

*Dr. Wislicki has now returned to Jerusalem.