[This page contains a mix of technical and medical discussions, including references to studies and practices related to anaesthesia and surgery. The text discusses the use of methylphenidate in the treatment of muscle spasticity and paralysis, as well as its effects on cardiac arrhythmia. There are also references to the influence of anaesthesia and surgery on plasma cortisol, insulin, and free fatty acids.]

**L'EFFET DE METHYLPHENIDATE (RITALIN) SUR LA SPASTICITE MUSCULAIRE APRES HALOTHANE**

*Méthylphénidate, un stimulant psychomoteur à faible action sympathomimétique, supprime complètement la spasticité musculaire et le tremblement, qui peuvent se produire comme compliqués d'une anesthésie à l'halothane. Les auteurs croient que la spasticité est due au rétablissement de l'activité spinale reflexe, qui précède celle des neurones moteurs supérieurs, de l'inhibition par l'anesthésie, et que le méthylphénidate agit en stimulant le reticulum du mésencéphale. La suractivité adrénergique du cœur, provoquée par le méthylphénidate, est empêchée par des petites doses d'un bloqueur beta et par la néostigmine.

**CORRESPONDENCE**

Paradox of Cardiac Arrhythmia in Anaesthesia

Sir,—With reference to the case described in Dr Borg's article (Brit. J. Anaesth., 1969, 41, 709), I wish to record a similar case. A patient with multiple extrasytostoses was to be anaesthetized for a temporal lobe brain tumour. Preparations were made to counter any instance of ventricular fibrillation. To my surprise the electrocardiograph tracing reverted to sinus rhythm soon after induction and remained so during the operation. Multiple extrasytostoses reappeared postoperatively. It seems as though general anaesthesia suppressed activity in some ectopic sites in the heart. As suggested by Dr Borg, the cause of the suppression may not be halothane since in this case anaesthesia was maintained using nitrous oxide, oxygen and tubocurarine.

V. M. DIVERKAR

 Bombay

Sticking Rotameters

Sir,—We have, over the last few years, renewed all our Boyle-type anaesthetic apparatus, and have had considerable trouble with the Rotameters in all the new pieces of equipment. The Rotameters have been sticking or working erratically because of electrostatic charges, and we have been able to show that the charge is on the surface of the glass both inside and outside the tube and not primarily on the bobbin.

Charges on the outside of the Rotameter tube are easily removed by spraying with an antistatic spray, but charges on the inside surface of the tube are more difficult to remove. We have found that coating the inside surface of the tube with an antistatic fluid at the time of servicing will prevent trouble for approximately one month, but the apparatus is usually serviced once every three months.

I am very anxious to find out how much of a problem this is to anaesthetists in Great Britain. I understand from the makers, that it is a considerable problem in Sweden and Canada, and they are investigating a method of making the tubes so that they will not pick up a static charge. However, if this is a problem to anaesthetists in this country, it would be worth looking for some way of treating existing tubes so that electrostatic charges do not build up.

I wonder whether I could ask your readers to write to me, letting me know if they have trouble with their Rotameters, whether this occurs often or only occasionally, and whether this is confined to the 9-inch tubes or whether they have trouble with the older machines in which 6-inch tubes are used.

J. CLUTTON-BROCK

Bristol

The Influence of Anaesthesia and Surgery on Plasma Cortisol, Insulin and Free Fatty Acids

Sir,—I was most interested in the recent paper by Dr R. S. J. Clarke and his colleagues (Brit. J. Anaesth., 1970, 42, 295) which came to more or less the same conclusions as ourselves (Allison, Tomlin and Chamberlain, 1969). However, I would like to offer some comments.

Dr Clarke states "The negative findings in relation to plasma insulin levels show that there has been no inhibition of insulin production during surgery". He bases this statement on the finding that the fall in plasma insulin after 30 minutes of intra-abdominal surgery (5 ± 1.6 μU/ml) was not statistically significant. If one examines this finding in the light of a rise in blood sugar at this time of 21 ± 2.2 mg/100 ml, then his results strongly suggest a suppression of insulin release.

Dr Clarke's conclusion based on statistical computation would only have been valid if there has been no change in blood sugar. Insulin levels must be interpreted in the light of such changes.

I look forward to reading Dr Clarke's findings on adrenaline secretion during major surgery. He states "the suggestion that adrenaline is liberated during major surgery has not been confirmed". In the absence of data on adrenal vein blood, it is difficult to comment on this statement, but I would find it difficult to explain the changes in blood sugar, free fatty acids and insulin on the basis of changes in plasma cortisol alone. I would also be surprised to learn that major surgery produced no increase in sympathetic activity.

S. P. ALLISON

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**REFERENCES**
