CORRESPONDENCE

POSTOPERATIVE EFFECTS OF METHYLPHENIDATE

Sir,—Drs Dodson and Fryer (1980) suggest that methylphenidate drug may be of value in increasing wakefulness, stimulating respiration and preventing shivering during the early postoperative period.

I believe that it is not only worthwhile but imperative to report briefly two unpleasant experiences after methylphenidate administration.

Patient 1. A 60-yr-old hypertensive female received general anaesthesia for breast biopsy followed by modified radical mastectomy. Before operation the arterial pressure was controlled with methyldopa. Anaesthesia was induced with thiopentone, pancuronium was administered to provide neuromuscular blockade and the trachea was intubated. Anaesthesia was maintained with nitrous oxide in oxygen, 0.2% halothane delivered from a Fluotec Mark II vaporizer and fentanyl 2ug/kg body weight. During the surgical procedure systolic arterial pressure fluctuated between 17.3 kPa and 19.5 kPa. At the end of surgery the neuromuscular blockade was antagonized with atropine and neostigmine. The patient responded to verbal commands and systolic pressure was found to be 21.28 kPa. A few minutes later, when she started shivering, methylphenidate 20 mg was administered i.v. slowly. Five minutes later the patient became unconscious and apnoeic. It was thought that she had a cardiac arrest, but peripheral pulses were palpable and the systolic pressure was increased to 37.2 kPa. This abrupt increase in pressure obviously caused cerebral oedema, or possibly a cerebral episode. The lungs were ventilated manually with oxygen and air and frusemide 80 mg and prednisolone 50 mg i.v. were given. The patient responded favourably and recovered completely. Urine output was about 1500 ml and systolic pressure decreased to 23.9 kPa.

Patient 2. A 42-yr-old female received general anaesthesia for excision of a breast lump. Before operation arterial pressure and e.c.g. were normal. During the early period after operation and while the patient was recovering smoothly with systolic pressure 17.29 kPa, methylphenidate 20 mg was administered i.v. slowly because the patient started shivering. Within the next few minutes she became unconscious and apnoeic. Systolic arterial pressure was found to be 29.26 kPa. Frusemide and prednisolone were given i.v. with artificial ventilation of the lungs. The response was satisfactory and recovery uneventful.

Postoperative shivering is unpleasant for the patient and undesirable for the anaesthetist. It is known that oxygen consumption may be doubled. In both these cases methylphenidate was administered to stop and not to prevent shivering.

After these experiences with methylphenidate, we keep the patients as warm as possible and give oxygen via a Ventimask. We found that methylphenidate 10 mg given i.v. slowly is quite sufficient to stop shivering and does not affect the cardiovascular system.

Drs Dodson and Fryer did not exclude or at least did not mention hypertension, tachycardia (of whatever cause) and cardiovascular disease. Their study included patients of 70 years. Did the authors correlate the age with the restlessness observed in the four patients after the methylphenidate administration? They claim increased respiratory stimulation in the early period after operation. At that time I would expect an increase in c.n.s. oxygen consumption and an increased oxygen consumption by the heart (increase in arterial pressure and in heart rate, increased work).

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REFERENCES


Sir,—Only moderate increases in arterial pressure and heart rate had been reported after clinical use of methylphenidate, and we observed such increases. Table III of our article reports a mean systolic arterial pressure increase of 2.14 kPa after halothane—methylphenidate, and a mean heart rate increase of 22 beat min⁻¹. The increases were smaller after fentanyl—methylphenidate, and the largest increase in systolic arterial pressure was 6.8 kPa.

Potentiation of the inotropic effect of noradrenaline on the dog's heart by methylphenidate has been reported by Maxwell and others (1961). Increased catecholamine secretion occurs during shivering, and arterial pressure also increases; Borotoluzzi and others (1963) report a pressure of 30.1 kPa during shivering and before administration of methylphenidate, with a decrease in arterial pressure after the administration of the drug. Both patients of Dr Fassoulaki were shivering when the methylphenidate was given when increased sympathetic activity must have been present. The importance of such activity in causing adverse reactions to methylphenidate is supported by Brichard and Johnstone (1970). Ventricular extrasystoles or tachycardia occurred in five patients with a mild respiratory acidosis, and this could be treated or prevented by oxprenolol.

Methylphenidate may increase myocardial and c.n.s. oxygen consumption, but we suggest that this is probably less than the known large increase in muscle oxygen consumption which accompanies shivering. Although the administration of oxygen and warming are useful measures in the management of shivering, there may be occasions when the use of methylphenidate is considered. We agree with Dr Fassoulaki that the smallest effective dose should be used, and her two reports suggest that particular care should be taken in administering methylphenidate to hypertensive patients, especially when increased sympathetic activity is present, for example, when shivering is established.

We did not find any correlation between age of patient and restlessness. We did not claim increased respiratory stimulation, but that “there was significantly less deterioration (in respiratory function) when methylphenidate was given with halothane.”

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