Sir,—We read with interest the study by Takada and colleagues [1] on the effects of midazolam and flunitrazepam on the release of dopamine from rat striatum.

Recently, we postulated that benzodiazepines may reduce the synthesis, release and postsynaptic effect of dopamine centrally [2]. This action at the chemoreceptor trigger zone is a possible mechanism for the antemeric effect of benzodiazepines [2].

The study by Takada and colleagues [1] supports the hypothesis that benzodiazepines reduce dopamine release centrally. However, they suggest that the effect is mediated by benzodiazepine binding to the GABA_A receptor complex, causing inhibition of dopaminergic neuronal activity. The reason given for this conclusion is that the benzodiazepine effect on dopamine release was abolished by flumazenil.

It has been shown that flumazenil is also an adenosine antagonist [3]. Benzodiazepines block the re-uptake of adenosine and some of their central effects may be explained by an increased adenosine action [3]. Adenosine, a central neuromodulator, has been shown to inhibit nigrostriatal dopamine release by an adenosine-1-receptor mediated effect [4].

The alternative explanation for the reduced release of dopamine after administration of midazolam and flunitrazepam is that benzodiazepines block the re-uptake of adenosine, causing an adenosine-mediated reduction in dopamine release [2]. This effect can be blocked by flumazenil acting as an adenosine antagonist.

The regional differences in the effects of midazolam and flunitrazepam found by Takada and colleagues [1] may reflect the differing sensitivities of the drugs on adenosine neuromodulation, the GABA_A-benzodiazepine receptor complex or both. Obviously, further studies are required to clarify this problem.

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REFERENCES


Sir,—Drs Di Florio and Goucke suggest that our results could be explained by an inhibitory effect of midazolam and flunitrazepam on adenosine uptake [1], because adenosine also inhibits dopamine release [1].

Given the lack of selectivity of benzodiazepines, benzodiazepine receptor antagonists [2-4] and the ability of benzodiazepines to modify adenosine A_1 and A_2 receptors [5], the suggestion of Drs Di Florio and Goucke merits further consideration. However, it is possible that the effect of adenosine on dopamine release may still be mediated by the GABA_A-benzodiazepine chloride channel because of the close association of some adenosine receptors with this receptor complex [6].

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ONSET OF BLOCK WITH VECURONIUM AND BODY MASS INDEX

Sir,—I was interested in the demonstration by Drs Gill and Scott [1] that the onset of vecuronium 0.1 mg kg^{-1} was faster after induction with etomidate compared with thiopentone or propofol. I am concerned, however, that excessively heavy patients do not appear to have been excluded from the study.

It is stated that weights ranged from 50 to 110 kg. Body mass index is the weight divided by the height squared. According to a standard nomogram [2], in order to escape classification as "obese", a 110-kg male would have to be more than 190 cm (6'3") tall. Only if his height were to exceed 210 cm (6'11") would his weight be accepted as normal!

It has been shown that increasing the dose of vecuronium from 0.1 to 0.15 mg kg^{-1} markedly decreases onset time in patients within 20% of ideal body weight [3]. The administration of a neuromuscular blocker to an obese patient on an mg kg^{-1} basis constitutes an overdose. If the patient weighing 110 kg belonged to the etomidate group, the mean onset time would have been spuriously reduced.

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REFERENCES


However, none of the three groups differed statistically with regard to the weights of patients. Those in the etomidate group had a mean weight of 74.2 kg (range 50-90 kg). Therefore it is unlikely that this variable could have been a confounder in our results.

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A TRACHEAL TUBE PROTECTOR TO PREVENT KINKING

Sir,—A tracheal tube hidden under surgical drapes may be inadvertently occluded. For example, during surgery on the cervical region, the arms of surgical staff may involuntarily compress the tube. In such situations, the patient's upper teeth act as the fulcrum of a lever, leading to a kink in the tracheal tube at the lips. To solve such problems, special tracheal tubes such as the RAE preformed tube and a reinforced tube have been developed. However, these tubes are not used frequently and they are cumbersome for tracheal intubation and suction.

We were faced recently with difficulty in ventilation of the lungs of a patient because of kinking of the tracheal tube during anterior fusion of the cervical vertebra. To solve this problem, we devised a protector using a modified anaesthetic mask. We cut off part of the plastic dome of an infant mask to create a square hole approximately 15 x 15 mm to fit a tracheal tube (fig. 1). After tracheal intubation, the tube was mounted on the concave portion of the mask and then connected to an anaesthesia circuit. The mask was fixed to the face with adhesive tape (fig. 2). This simple equipment prevented the tracheal tube from kinking because of the weight of the drapes or the surgeon's arm. It was not an obstacle to surgery because of its small size.

We now routinely use this protector for surgery in the cervical region.

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A SIMPLE INDICATOR FOR IDENTIFYING THE EXTRADURAL SPACE

Sir,—Recognizing when the point of the needle pierces the ligamentum flavum and enters the extradural space is a critical step in performing extradural anaesthesia. Forty-three years ago, Macintosh proposed an ingenious device for identifying this space, based on the existence of negative pressure in the extradural space [1]. This indicator comprises an inflated rubber balloon attached to a Tuohy needle. When the tip of the needle enters the extradural space, the balloon deflates abruptly. However, the materials for this device are not readily available, and its assembly is awkward.

Based on the same principal, we have developed a simple device using common, readily available materials that are found in all operating theatres: a Foley catheter, a stopcock and a needle for i.m. injections. The extradural needle is attached to the female end of the stopcock using the male connector included in the set (Minipack, Portex). The Foley catheter Fr16 is cut transversely approximately 7 cm from its distal end and is attached to the side connection of the stopcock. The i.m. needle (21-gauge) is inserted into the narrow lumen of the Foley catheter leading to the balloon, and a syringe with a few millilitre of air is attached to this needle. The stopcock valve is adjusted to connect between the syringe and the Foley catheter, and the balloon on the catheter is inflated. After the needle is placed in the spinal ligaments, the stopcock valve is turned to seal the air in the balloon and open the connection between the Foley catheter and the extradural needle. This device is steadily advanced through the tissues with constant pressure. As soon as the needle enters the extradural space, the balloon abruptly deflates, indicating the location.

For correct and effective use, the device must be sealed so that the deflation of the balloon is abrupt. Furthermore, confirmatory tests for extradural puncture, including an aspiration test, must be carried out.

This technique is very useful for identifying the extradural space, especially at the thoracic and cervical levels, where a positive hanging drop sign is not always an infallible index of correct extradural localization [2].

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HAEMODYNAMIC EFFECTS OF PROPOFOL

Sir,—We read the article by Lindgren and colleagues [1] comparing propofol and thiopentone and congratulate the authors for a fine study on the use of propofol and its haemodynamic effects.