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REPLY

The suggestion by Naguib and Gyasi that an interaction may exist between tamoxifen or danazol and atracurium is interesting and warrants further investigation. Tamoxifen is a nonsteroidal drug with strong antiestrogen properties, which are considered secondary to its ability to compete with estrogen for binding sites in target tissues. It is used in the treatment of advanced breast cancer in postmenopausal women. Tamoxifen is prepared as a tablet with carboxymethylcellulose calcium, magnesium stearate, mannitol and starch as inactive ingredients.[1] Danazol is a synthetic androgen which suppresses the pituitary–ovarian axis, has weak androgenic activity, and possibly binds directly to gonadal steroid receptors. It is used in the treatment of endometriosis, fibrocystic breast disease and hereditary angioedema. Danazol (Danocrine) also is supplied in capsule form prepared with benzyl alcohol, gelatin, lactose, magnesium stearate, parabens, sodium propionate, starch, talc and food coloring.[2]

Tamoxifen and danazol both contain ring structures, bind to receptors and are prepared with magnesium stearate. The effect of the molecular structure and receptor binding ability on the interaction of these drugs and muscle relaxants merits attention. Magnesium sulfate is known to potentiate the effects of muscle relaxants. Magnesium stearate may have similar properties. Also, danazol is structurally similar to the adrenocorticosteroids which have been reported to potentiate or antagonize the effects of nondepolarizing blockers.[4,5]

In summary, we feel that Drs. Naguib and Gyasi introduce an important point and we agree that atracurium and perhaps other nondepolarising relaxants should be used cautiously in patients receiving these drugs until definitive information is available about their interactions.

The points raised by Dr. Donati are well taken. The actual total dose of atracurium administered was 85 mg (1.44 mg·kg^{-1}) over the 2.5-hour procedure. This included 25 mg initially followed by three additional doses of 20 mg. Unfortunately, due to our error, the incorrect dosage was described in the case report. During the operation, the response of the adductor policis to ulnar nerve stimulation was assessed visually. In addition, contractions of the facial muscles in response to stimulation of the facial nerve were also assessed visually. Although, as pointed out by Dr. Donati, there may be differences between the sensitivities of various muscles to non-depolarizing blockers, our patient did not exhibit this phenomenon.

We recognize that even trained observers may overestimate the degree of contraction to nerve stimulation; however, we do not routinely use recording equiment in every patient who demonstrates muscle weakness at the conclusion of an anaesthetic. We agree that succinylcholine, gentamycin, enflurane and d-tubocurarine can alter the response to atracurium and this was discussed in our report.

The last comment made by Dr. Donati certainly is valid. We agree that all the necessary information could have been obtained by the responses to train-of-four stimulation and that we may have confused the picture by using tetanic (100 Hz) stimulation. Even if train-of-four responses are assessed prior to tetanic stimulation, post-tetanic potentiation may persist for a long time. Train-of-four response provides information about the degree of neuromuscular block, but up to 70 per cent of receptors have to be blocked before fade becomes evident. To assess the degree of recovery from block this information may not be sufficient and a normal response to tetanic stimulation (100 Hz) is more reassuring. We recognize that post-tetanic distortion of twitch may be prolonged and may lead one to overestimate recovery from blockade.

In spite of all the complicating factors, we believe that this patient was weak due to atracurium, particularly when we consider that only 85 mg (1.44 mg·kg^{-1}) were used during the 2.5 hr operation and no supplemental dose was administered in the last 30 min.

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Estimation of blood loss in the operating room

To the Editor:

A common way to estimate blood loss in the operating room is to subtract the amount of irrigation fluid used from the total volume of blood and fluid in the suction bottle. This technique requires a record of irrigation fluids and this may lead to omissions or inaccuracy.

We wish to remind readers of the usefulness of the micro-haematocrit method to determine the packed cell volume (PCV) of the blood and fluid in the suction bottle, and therefore to estimate blood loss. The following simple calculation estimates the amount of blood in the fluid:

$$\text{Blood loss} = \frac{\text{volume in the suction bottle} \times \text{suction PCV}}{\text{patient PCV}}$$

Many methods are used by surgeons and anaesthetists to estimate blood loss. Accurate methods are more complex and therefore less likely to be easily applied.[1–4] The method we describe has its own inbuilt errors. The suction bottle sample must be well mixed or the formation of clot and effects of gravity will produce an inaccurate sample. The assumption that the PCV of the patient stays constant throughout the procedure is erroneous as a decreasing haematocrit would tend to overestimate the blood loss. At the same time haemolysis from trauma and irrigating fluids tends to give an erroneously low PCV value for the suction fluid and this would underestimate the amount of blood loss. Blood that is not suctioned must be measured by other means.

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Ketamine induction for a patient with hyperinsulinism treated with oral diazoxide

To the Editor:

The usefulness of oral diazoxide in the management of hyperinsulinism, because of its direct inhibitory action on release of pancreatic insulin, is well documented.[1] Diazoxide is a nondiuretic benzothiadiazine derivative and has potent vascular dilating activity. Burch and McLeskey[2] reported marked hypotensive episodes related to thiopentone administration in two patients with hyperinsulinism treated with oral diaxoxide. They postulated three mechanisms of the untoward drug interactions between diazoxide and thiopentone: (1) displacement of diazoxide from protein binding sites by thiopentone; (2) increased unbound thiopentone due to preoccupation of binding sites by diazoxide; (3) a combination of the first two mechanisms, resulting in an additive effect on depression of blood pressure.

Ketamine, instead of thiopentone, might be the intravenous agent of choice for induction of anaesthesia in patients with hyperinsulinism treated with oral diazoxide, because of its cardiovascular-stimulating properties and limited plasma protein binding.[3] We wish to report our experience with the use of ketamine for induction.

A 1-year 8-month old 12 kg boy with hyperinsulinism, treated with oral diazoxide 10 $mg \cdot kg^{-1} \cdot day^{-1}$ was scheduled for pancreatectomy. Diazoxide was continued until the morning of the surgery, because of the previous profound hypoglycaemic episodes upon discontinuation of diazoxide.