CORRESPONDENCE

ALTHESIN IN THE WOLFF–PARKINSON–WHITE SYNDROME

Sir,—Following two reports on electrocardiographic changes at induction in patients with the Wolff–Parkinson–White (W–P–W) syndrome (Hannington-Kiff, 1968; Campkin and Moore, 1969), Kadis and Gianelly (1973) question the use of thiopentone which appears to produce further aberrant conduction. Diazepam was suggested as the best induction agent, but its effect can be unpredictable in the younger adult patients, some failing altogether to become unconscious (Brown and Dundee, 1968). Since Althesin possesses useful anti-arrhythmic properties (Cundy, 1973) it may be a worthwhile alternative.

A 45-year-old female patient with asymptomatic W–P–W syndrome was undergoing laparoscopic ligation of the Fallopian tubes. Physical examination was normal apart from obesity and numerous varicose veins in both legs. A routine e.c.g. before operation showed the characteristic features of the W–P–W syndrome although there was no history of attacks of tachycardia.

The patient was not premedicated, but atropine 0.2 mg was given slowly at induction. Anaesthesia was induced with Althesin 5 ml given over 2 min, and diazepam 5 mg. The trachea was intubated with the aid of suxamethonium 100 mg after pre-treatment with tubocurarine 3 mg, and before surgery was allowed to commence pentazocine 30 mg was administered. The e.c.g. was recorded at frequent intervals during the induction procedure (standard leads 1 and 2). No electrocardiographic modifications were observed at any time. After induction, anaesthesia was maintained with Althesin 1 ml every 5 min and tubocurarine (total 40 mg for a 1-h procedure). The lungs were ventilated artificially with 70% nitrous oxide in oxygen. Both anaesthesia and surgery proceeded uneventfully and recovery was normal. The heart rate was monitored continuously with a chest lead, but no changes in the e.c.g. occurred.

It would appear from this case that Althesin is a possible alternative to thiopentone for induction (and also for maintenance) of anaesthesia in these patients. The e.c.g. changes reported with thiopentone were suggestive of acute myocardial infarction, but were shown to be the result of further aberrant conduction as enzyme concentrations (s.g.o.t., s.g.p.t., LDH) remained normal. Aberrant conduction, although much less serious, may have profound circulatory effects (McIntosh and Morris, 1966; Boba, 1978).

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REFERENCES


COMPARISON OF TRACHEAL CUFFS

Sir,—The article entitled “A Method for Comparing Endotracheal Tubes” (Homi et al., 1978) provides additional evidence to support the view that it is unnecessary to have a tracheal wall pressure greater than the peak airway pressure. However, in deducing the tracheal wall pressure for a low residual volume cuff from the compliance curves, the authors make the assumption that the cuff inflates to the same shape in air as it does in the confines of the trachea. This is clearly not so and the tracheal wall pressure will vary along the length of the cuff. Therefore, the resulting “calculated” pressure can only be an approximation. One major problem in the use of compliance curves is that the physical properties of a cuff will vary between individual tubes as a result of manufacturing tolerances and in a given tube they can vary with both temperature and time. For the high residual volume cuff, it has been shown that the intra-cuff pressure is equal to the tracheal wall pressure (Carroll, 1973; Crawley and Cross, 1975) and so compliance curves are unnecessary.

It is not always acceptable to use minimal leak ventilation as this may not provide protection against aspiration. In these situations additional air will need to be added to the cuff. For a low residual cuff the effects of this are not immediately apparent, whereas for the high residual cuff the effect can be seen readily by using a simple pressure gauge to measure the intra-cuff pressure and hence the tracheal wall pressure. Indeed, with the high residual volume cuff, additional air added to the cuff will, in the first instance, increase the resting cuff pressure without necessarily increasing the peak intra-cuff pressure. This is not so for a low residual volume cuff, as has been demonstrated again by Homi and his colleagues in their figure 5. From this it can be seen that the addition of 1 ml of air to the cuff almost doubles the calculated tracheal wall pressure.

Homi and his colleagues refer to a high transmitted pressure necessary for a seal in high residual volume cuffs “particularly along any folds or ridges in the cuff”. They quote the reference as an earlier paper by myself (Cross, 1973). In fact, the higher pressures referred to were not “particularly along any folds” but were generally increased intra-cuff pressures necessary to seal any folds. This was only the case in the in vitro studies. Later work (Crawley and Cross, 1975) showed that these greater pressures were