While Sigurdsson and his colleagues have devised a nice system for evaluating these arrhythmias, I believe that no discussion of arrhythmias during halothane anaesthesia is complete without documentation of arterial or end-tidal carbon dioxide. In the absence of this information, it is not possible to implicate operative site or a given anaesthetic as the cause of the arrhythmia. I believe the most likely cause is the combination of hypercarbia, halothane and surgical stimulation. I agree that this frequent occurrence of ventricular arrhythmias is disturbing and that tracheal intubation during adenoidectomy is preferable.

H. W. KARL

REFERENCES


Sir,—We agree with Dr Karl that the cause of the arrhythmias was probably a combination of halothane, hypercarbia and surgical stimulus as we have discussed in other papers (Sigurdsson, 1983; Sigurdsson et al., 1983; Sigurdsson and Lindahl, 1983). These indicated that the occurrence of ventricular arrhythmias during halothane anaesthesia for adenoidectomy was halved by intubation and reduced even more with controlled ventilation. The purpose of the present study (Sigurdsson, Werner and Fahraeus 1983) was more limited. Recently, several authors (Alexander, 1971; Alexander, Bekheit and Fletcher, 1972; Alexander and Murtagh, 1979; Lindgren, 1981) have suggested that widened QRS complexes occurring during oral surgery under halothane anaesthesia are usually aberrantly conducted supraventricular impulses, rather than ventricular extrasystoles. However, the evidence has not been convincing and our study was carried out in order to gather further information regarding this specific question.

G. H. SIGURDSSON

REFERENCES


CHANGES IN RESIDUAL VOLUME FOLLOWING OXYGEN BREATHING

Sir,—With regard to the article by A. B. Baker and R. Restall (1983) entitled "Changes in residual volume following oxygen breathing", there is a discrepancy between the mean control FRC of 3.09±0.7 in table I and the FRC before oxygen breathing in table V of 3.18±0.69. I have checked the mean and SD of FRC from the figures in table I and the mean and SD as quoted are correct. However, a paired Student's t test fails to demonstrate a significant reduction in FRC following oxygen breathing when FRC values in table I are compared with those in table IV.

In addition, analysis of tables I and IV indicates that FRC increased after oxygen breathing in six and not five subjects, as quoted in table VI. Furthermore, would it not have been a preferable statistical method to have compared a change in FRC with CC using correlation/regression analysis, rather than comparing two FRC with CC using correlation/regression analysis, rather than comparing two groups by t test derived by retrospective classification based on the results?

N. M. DEARDEN

REFERENCES


Sir,—Thank you for allowing me to reply to the correspondence from Dr Dearden. First, I am indebted to Dr Dearden for noticing a mistake in table I which had escaped my notice. The FRC value for the third subject R.C. should have read 2.28 instead of the 2.28 shown. The 2.28 was in fact the ERV (TLC) of 3.18±0.69. I have checked the mean and SD of FRC from the figures in table I and the mean and SD as quoted are correct. However, a paired Student's t test fails to demonstrate a significant reduction in FRC following oxygen breathing when FRC values in table I are compared with those in table IV.

For the second point, only 18 (instead of 19) of the subjects were compared because we could not obtain a closing capacity for R.L., although we tried. Thus, although the FRC did increase in six subjects following oxygen breathing, we could compare only five of these with the 13 who decreased their FRC when considering the effects of closing capacity. This is clear from a study of
Dr Dearden's last point is more complex. We did not consider that a correlation between CC and FRC(2) would be of any physiological significance to the study and that the correlation ($r = 0.6365$) is probably related to body size and not the experiment. We did consider a correlation between FRC(1) - CC and FRC(1) - FRC(2) to be physiologically significant, but thought that there was dependency between these two variables. The coefficient for this latter correlation is $r = 0.3201$ ($P = \text{n.s.}$), and even with some dependency between the variables there is no statistical correlation. The discussion in the article is thus unaltered and the conclusions remain the same.

May I repeat my apology for the mistake in table I and thank Dr Dearden for his constructive interest.

A. B. Baker
Dunedin

ATRACURIUM V. SUXAMETHONIUM IN A CASE OF ORGANOPHOSPHOROUS POISONING

Sir,—Atracurium is a selective non-depolarizing neuromuscular blocking drug which is rapidly metabolized by Hofmann elimination, and by ester hydrolysis independent of the plasma cholinesterase activity (Hughes and Chappie, 1981). In contrast, suxamethonium is a depolarizing agent which is rapidly hydrolysed by the plasma cholinesterase. The present case report compares the effect of the inhibition of the plasma cholinesterase by parathion poisoning on the neuromuscular block of atracurium with that of suxamethonium.

The patient, a 28-yr-old man (80 kg), attempted suicide by ingesting parathion 100 ml. The patient manifested the typical picture of parathion poisoning including coma, muscle twitches and respiratory failure which necessitated prolonged tracheal intubation and controlled ventilation. After 2 weeks, the patient developed acute erosive gastritis requiring gastrectomy. A few days later, he had tracheal erosion necessitating tracheotomy. During both operations, anaesthesia was maintained with nitrous oxide in oxygen. Relaxation was achieved during the first operation by atracurium, while suxamethonium was used in the second procedure. Neuromuscular transmission was monitored by the twitch response to supramaximal stimulation of the ulnar nerve at the wrist every 10 s. In the first procedure, an initial dose of atracurium 0.5 mg kg$^{-1}$ was required to produce complete neuromuscular block which lasted for 20 min. This was followed by maintenance doses of 5 mg every 10 min. The block was non-depolarizing in nature as manifested by tetanic fade and post-tetanic facilitation. In the second procedure, suxamethonium 0.5 mg kg$^{-1}$ produced complete neuromuscular block which lasted for 45 min; the block was depolarizing in nature.

The serum cholinesterase activity was measured using propionylthiocholine as substrate by the method of Dietz, Rubenstein and Lubrano (1973) (normal concentration in our population $4.12 \pm 1.44$ u. ml$^{-1}$). The plasma cholinesterase concentration in the patient was 0.04 u. ml on admission, and increased up to 0.56 u. ml$^{-1}$ after 3 weeks. Dibucaine No. was 89.

Parathion, an organophosphorous anticholinesterase, is the most widely used insecticide. Since, organophosphorous compounds can cross the blood–brain barrier, they inhibit central as well as peripheral acetylcholinesterase. These compounds can also inhibit the plasma cholinesterase (Barnes and Davies, 1951).

In this patient, the plasma cholinesterase was 0.04 u. ml$^{-1}$ on admission and increased to 0.57 u. ml$^{-1}$ after 3 weeks. In the presence of this marked decrease of the plasma cholinesterase activity, suxamethonium produced a markedly prolonged neuromuscular blockade. In contrast, the duration of action of the blockade produced by atracurium was not greater than normal.

The present report shows that atracurium is a relatively short-acting neuromuscular blocking drug even in the presence of low plasma cholinesterase activity, and hence can be used safely in such patients.

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REFERENCES

