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
Hershey, Pennsylvania

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 anesthesia 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
Lund

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
Leeds

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 3.90 litre instead of the 2.28 shown. The 2.28 was in fact the ERV (TLC) volume for R.C. and was inadvertently transcribed twice and not noticed. This corrects the mean ± SD for FRC in table IV as stated in the article, so the conclusions remain the same.

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