induced hypotension.

Unfortunately, they did not monitor the end-expiratory carbon dioxide during the operation. Previously, we have observed abnormal decreases in the capnograms after the insertion of hip prostheses using acrylic cement. These represent wash-out curves typical for pulmonary embolism as described by Smalhout and Kalenda (1975). As an example figure 1 shows such a curve obtained from a 78-yr-old female during general anaesthesia after the insertion of a hip prosthesis using acrylic cement.

We want to draw attention to the fact that, contrary to the authors' opinion, a decrease in cardiac output combined with a decrease in arterial pressure is not necessarily the consequence of peripheral vasodilatation, but can also be the result of the afore mentioned embolism in the lung (Halmagyi, Starzeccki and Horner, 1964). This can also explain their results in the control group.

If, in dogs, a miliary pulmonary lesion is induced, sodium nitroprusside will cause a decrease in pulmonary artery pressure and pulmonary vascular resistance, but no change in cardiac output (Colley, Cheney and Hlastala, 1979). This is probably the consequence of an increased blood flow to the unaffected parts of the lung (Hill, Sykes and Reyes, 1979).

Thus, in our view the haemodynamic changes following the application of acrylic cement are most probably the consequence of embolism in the lung, which during induced hypotension is masked by some drugs depressing the protective response of hypoxic pulmonary vasoconstriction (Editorial, 1979).

JAAP J. DE LANGE
LEO H. D. J. BOOIJ
Amsterdam

REFERENCES


Sir,—In reply to the letter from De Lange and Booij, may I make the following comments:

We did not monitor the end-expiratory carbon dioxide. However, in a subsequent investigation we measured blood-gas tensions in 50 patients just before and at 1, 2, 4, 6 and 8 min after the insertion of acrylic cement, in patients operated under normotension, extradural anaesthesia, and trimethaphan, nitroprusside and nitroglycerin hypotension. There were no changes noticed in carbon dioxide, but a definite pulmonary hypoxic response was encountered under normotension and extradural blockade. However, changes in arterial \( P_{O_2} \) were insignificant after the implantation of acrylic monomer during trimethaphan, nitroprusside and nitroglycerin hypotension.

A. K. VAZEERY
Stavanger

THE LARYNGEAL MASK
Sir,—Dr Brain (1983), in his article on the Laryngeal Mask states that: "During IPPV (with a Manley Blease ventilator) . . . the readings obtained from the Wright respirometer (expired minute volume) were all greater than the inspiratory minute volume as measured by rotameter readings, presumably reflecting the known inertial characteristics of this respirometer . . . ."

We believe that this explanation is inadequate and offer an alternative.

Measurements of inspired and expired minute volumes were made at different points in the circuit, the rotameters being proximal to, and the respirometer distal to, the ventilator. Thus gas volume measurements were made at different pressures.

Assessment of the accuracy of Wright's mechanical respirometer (Nunn and Ezi-Ashi, 1962) suggests that its inertial characteristics are unlikely to account for the reported difference (14.68%).

Back pressure as a result of the resistance of a ventilator in series causes rotameters to underestimate delivered gas volume, and we believe this to be the major factor operating.

J. H. L. ANTROBUS
A. HUTCHINSON
Leeds

VENTRICULAR ARRHYTHMIA OR SUPRAVENTRICULAR ARRHYTHMIA WITH ABERRANT CONDUCTION

Sir—Sigurdsson, Werner and Fahrennaa (1983) reported the occurrence of anomalous QRS complexes in more than half of a group of children undergoing adenoidectomy. The anaesthetic technique included 2.5 - 3% halothane in 50% nitrous oxide and oxygen, spontaneous ventilation and no tracheal tube. Halothane is known to displace the \( P_{CO_2} \)-ventilation response curve to the right and to reduce its slope (Severinghaus and Larson, 1965).

The resulting hypercapnia is likely to be even more severe in the absence of an endotracheal tube. Hypercapnia increases plasma catecholamine concentrations and has been implicated in contributing to arrhythmias occurring during halothane anaesthesia (Benumof, 1981). We studied 31 children undergoing cleft palate repair under halothane anaesthesia (end-tidal halothane was \( 0.8 \pm 0.4\% \)), nitrous oxide and oxygen via an endotracheal tube (Karl et al., 1983). We controlled ventilation to produce a normal or low end-tidal carbon dioxide concentration. Despite the addition of adrenaline 0.5 - 14.3 \( \mu g \) kg\(^{-1}\) (mean value 5.3 \( \mu g \) kg\(^{-1}\), SD 3.2) in 1% lignocaine for local haemostasis, we saw no ventricular or supraventricular arrhythmias.
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 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 Murch, 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

REFERENCES

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

REFERENCE

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 noticing a mistake in table I which had escaped my notice. The FRC values in table I are compared with those in table IV.

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REFERENCES