100 of these same patients that the exhibition of no more than 2 per cent halothane during induction has a detectable effect \((P=0.06)\) on BSP retention 5 days after surgery, when compared with a series of patients in whom halothane-concentration never exceeded 1 per cent. SGPT changes in the same patients, though by themselves not significant, support the conclusion that halothane (rather than the patients' pre-operative condition or the nature of surgery) interferes with the conjugating and excretory functions of the liver as shown by the BSP changes and increases the normal breakdown of liver cells as indicated by the elevated GPT levels in the blood. Admittedly, it is a far cry from abnormal liver function tests to clinically significant liver damage, but the tendency is there and should be weighed during the selection of anaesthetic agents and techniques. The facts can be verified by any interested anaesthetist in about 2–3 months in his own hospital and he can then draw his own conclusions from them. Our conclusion is that halothane is a pleasant and useful anaesthetic for prime-risk patients (who, after all, constitute the majority of our clientele) but it is not the agent of first choice for special-risk individuals, particularly if the operation involves opening the abdomen. It seems to us further that the safe administration of halothane requires greater caution and precision than many would have thought: this should also be considered in its choice.

We see nothing in these statements that would warrant the wholesale abandonment of a popular anaesthetic on the one hand or the visceral reaction of serious investigators on the other. The controversy has taught us something about the patients' liver and a great deal more about their physicians' spleen. Why not let it go at that?

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REFERENCE


Carbon dioxide elimination during insufflation anaesthesia

Sir,—I was interested in the article by Drs. Zeitlin, Short and Fielding (Brit. J. Anaesth., 37, 117) but I would like to suggest that the title is somewhat misleading. Apnoea was obtained by paralysis with suxamethonium, and nitrous oxide and oxygen were insufflated at a rate of 10 l./min. As would be expected, carbon dioxide retention occurred at a slightly lower rate than in a control series without insufflation. True insufflation anaesthesia as practised in the early 1920s, mainly for upper abdominal surgery, was quite different and air-ether or nitrous oxide-oxygen-ether was given at approximately three times this rate. Professor M. S. Pembrey (1914) worked out the physiology of the method at a flow rate of 30 l./min of air and found that the carbon dioxide content of the blood was lowered and concluded that this was the cause of the apnoea. I have myself given several hundred administrations using a flow rate of this order and found that operating conditions were almost ideal with excellent relaxation, little capillary oozing, and no perceptible respiratory movements. Two of my colleagues have recently anaesthetized a series of patients with the true insufflation technique using modern monitoring and it was found that while several subjects appeared to be apnoeic, slight respiratory movement could be detected. The cause of the virtual apnoea is still obscure as the carbon dioxide levels were within normal limits. The plane of narcosis was consistently light as evidenced by rapid recovery while there was virtually no positive intrapulmonary pressure. It is hoped to publish this work quite shortly and it could be argued that under certain circumstances, the technique compares favourably with more modern ones using relaxants. While not subscribing entirely to Sir Heneage Ogilvie's famous dictum, "Most new things in the end turn out to be wrong" (Lancet, 1956, 1, 115), it is possible that some anaesthetic techniques in use 45 years ago were not quite so bad as is commonly supposed.

With regard to the heartbeat causing some respiratory exchange, this is surely evident from the fact that the pulse rate can often be counted by watching the bag in a closed circuit, especially in young subjects.

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REFERENCES


FATAL CEREBRAL ANOXIA FOLLOWING THE USE OF LIGNOCaine SPRAY FOR BRONCHOSCOPY

Sir,—In our article under this title (Brit. J. Anaesth., 37, 61) we quoted Astra Hewlett Ltd. as recommending a maximum dose of 6.5 ml of 4 per cent lignocaine (260 mg). It has been pointed out that this refers to the amount suggested in the United States, and the manufacturers in Great Britain, while suggesting that 2 ml of 4 per cent solution (80 mg) is sufficient for bronchoscopy, recommend that no patient should be given more than 5 ml (200 mg).

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CORRECTION

Sir,—It is regretted that there is an error in the paper, "A study of the arterial clearance of Xenon 133 in man" (Brit. J. Anaesth., 37, March 1965). On page 153 in the seventh paragraph of the "Methods" section, the sentence beginning "In two conscious patients . . ." should read "In four conscious patients . . .".

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