CARDIAC IRREGULARITIES IN CATS UNDER HALOTHANE
ANAESTHESIA

BY

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Experiments carried out during the past eighteen months on the effects of halothane on the cardiovascular system of cats may throw further light on some aspects of the work of Raventós and Dee (1959) on cats anaesthetized with azeotropic mixtures of halothane and ether. This brief summary of the relevant results has, therefore, been prepared. It is hoped that a more detailed account of the investigation will be published in due course.

METHODS

All the experiments were performed on healthy young adult cats. The anaesthetic agents were delivered from a standard Boyle's anaesthetic apparatus, a Fluotec or an Oxford Mark I vaporizer being incorporated as necessary. Halothane or ether was vaporized using a mixture of equal parts of nitrous oxide and oxygen at a total flow rate of 6 litres per minute. Cyclopropane was added to the nitrous oxide/oxygen mixture to give the required concentration while the total gas flow rate was maintained at 6 litres per minute.

Anaesthesia was induced by placing the cat in a large glass tank into which the anaesthetic agents were introduced through narrow bore tubing. When third plane anaesthesia had been reached, the cat was removed from the tank and placed on its back on the operating table. Anaesthesia was maintained using either (i) a cat face mask and Magill attachment, or (ii) a No. 2 or No. 3 plain Magill endotracheal tube and T-piece, endotracheal intubation being performed under direct vision after the intravenous administration of 3 mg of suxamethonium chloride.

Standard limb lead electrocardiograms from subcutaneous needle electrodes and the blood pressure from a cannulated femoral artery were recorded simultaneously on a direct writing multichannel recorder (Cambridge Instrument Co. Ltd.).

RESULTS

Sixteen cats were anaesthetized with halothane and anaesthesia was maintained with a concentration of 1 per cent of this agent. Fourteen of these animals developed a cardiac irregularity within the first hour of anaesthesia. The onset of this irregularity was delayed and it was never seen until the animal had been anaesthetized for at least 15 minutes. The character of the irregularity was constant (fig. 1). A sudden increase in the concentration of the halothane to 2 per cent was followed by an immediate return to a normal electrocardiographic pattern, but the irregularities reappeared within a few minutes. Returning the concentration of halothane to 1 per cent then had no effect on the electrocardiogram but increasing the strength to 3 per cent again resulted in an immediate, but temporary, return to normal rhythm.

The irregularities were also abolished, but with a delay of a few seconds, by positive pressure hyperventilation. This effect was not obtained when 5 per cent carbon dioxide was added to the gases used for hyperventilation. Estimation of the carbon dioxide content of serial arterial blood samples taken during these manoeuvres showed that the disappearance of the irregularities coincided with a fall in the carbon dioxide content of the blood.

For control purposes three cats were anaesthetized with ether and anaesthesia maintained with
10 per cent ether vapour for the first hour and 20 per cent ether vapour for the second hour. No cardiac irregularities were observed in these three cats. A further two cats were anaesthetized with cyclopropane and anaesthesia maintained with 12.5 per cent cyclopropane. One of these cats exhibited a cardiac irregularity similar to that seen under halothane anaesthesia. In this animal positive pressure hyperventilation abolished the irregularity.

DISCUSSION
It is clear from these results that cardiac irregularities occur in cats anaesthetized with 1 to 3 per cent of halothane vapour and are not as uncommon as is suggested by Raventós and Dee (1959). The tracings of the irregularity have been examined in some detail and study of longer lengths than that shown in figure 1 suggests that they show either interference dissociation or multifocal extrasystoles. The abolition of the irregularities by measures designed to reduce the carbon dioxide content of the blood indicates that, as suggested by Johnstone (1956), hypercapnia predisposes to their onset. This fact, coupled with the failure of section of the vagus nerves to abolish the established arrhythmia, favours the latter alternative. It is hoped that further investigations will clarify this point.

A most important feature of the results is the delay before the onset of the irregularity and this might explain why the irregularities noted by Raventós and Dee in one cat appeared to start only after the inhalation of the azeotropic mixture.

ACKNOWLEDGMENTS
We wish to thank Mr. D. E. Stevenson, B.Sc., B.V.Sc., M.R.C.V.S., for performing the estimations of carbon dioxide in the arterial blood samples and Imperial Chemical Industries (Pharmaceuticals) Ltd. for generous supplies of Fluothane.

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HALOTHANE AND THE BLOOD SUGAR

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It has been known since the beginning of the century that diethyl ether anaesthesia produces an increase in the blood sugar content (Underhill, 1906). Most other volatile anaesthetics give rise to a similar response. As each new agent has become available it has been investigated with this in view and it seemed desirable to carry out a study of this kind for halothane. For apart from a short note on the matter in a more general paper (Stephen et al., 1957) no detailed investigation had until recently been made.

METHODS

Twenty patients in all were studied. Ten of them underwent general surgical operations. In them an initial specimen of venous blood was taken either immediately before induction or immediately before turning on the halothane. Subsequent specimens were withdrawn at intervals during anaesthesia. These patients were premedicated with morphine 10 mg and atropine 0.6 mg, or papaveretum 20 mg and hyoscine 0.4 mg. Anaesthesia was induced with a sleep dose of thiopentone. Suxamethonium in a dose of 40–50 mg was given and an endotracheal tube passed while the patient was paralyzed. Ten neurosurgical patients were anaesthetized by a similar technique, described in detail elsewhere (Hunter, 1954, 1958). Specimens were withdrawn immediately before turning on halothane and at intervals of approximately half an hour during the subsequent administration of the drug. The halothane was administered either from a Boyle’s bottle calibrated at a temperature of 70°F or with the aid of a Fluotec vaporizer. By either method the percentage of halothane varied within the limits of 0.7 and 2 per cent. No attempt was made to keep the concentration at one single level. It was modified to meet the needs of the individual patient and the stage of the operation in progress. Blood sugar determinations were carried out in duplicate by the method of Maclean (1919), which has in this laboratory a standard error of ± 5 per cent.

RESULTS

The blood sugar changes in the patients are shown in figures 1 and 2. It will be apparent that in the general surgical cases the results were somewhat irregular. In the neurosurgical cases, however, the blood sugar level remained constant over long periods of halothane administration. In neither group was there any great deviation from normal levels, nor was there any suggestion of the constantly climbing blood sugar content which regularly appears during other forms of inhalation anaesthesia with volatile agents.

DISCUSSION

During the administration of diethyl ether it has been noted that the blood sugar content doubles itself in half an hour, and provided that there is enough glycogen in the liver it will increase by the same amount in the second hour (Mackay, 1928; Cantarrow and Gehret, 1931). A similar state of affairs was shown by the author to hold for methyl n-propyl ether (Hunter, 1950) and hyperglycaemia has also been shown to occur in the patient anaesthetized with cyclopropane (Neff and Stiles, 1936). The results of this investigation indicate that the blood sugar changes produced by halothane in concentrations of up to 2 per cent are negligible by comparison.

It is almost universally accepted that the rise in blood sugar during ether anaesthesia is due to adrenaline release produced by the action of the drug on the centres of the brain which govern autonomic activity (Banerji and Reid, 1933; Brewster et al., 1952; Bunker et al., 1952). The fact that halothane does not cause a rise in
HALOTHANE AND THE BLOOD SUGAR

Fig. 1
The blood sugar changes in general surgical patients anaesthetized with nitrous oxide, oxygen and halothane. (The lines are broken to indicate a period of anaesthesia in which no halothane was given.)

Fig. 2
The blood sugar changes in neurosurgical patients anaesthetized with nitrous oxide, oxygen and halothane. (Zero time indicates the moment of adding a halothane supplement to the anaesthetic mixture. It was usually some 20 to 50 minutes after induction. The broken line indicates a rise in blood sugar produced by injecting local anaesthetic solution containing adrenaline.)
the blood sugar is almost certainly confirmatory
evidence of its power of depressing the activity
of the sympatho-adrenal system. This fact is, of
course, manifested more obviously in clinical
anaesthesia by hypotension and peripheral vasodilatation, but it would seem that the basic
mechanism in both hypotension and relative
absence of blood sugar change is the same.

Since this work was completed Keating (1959)
has published evidence that the blood sugar con-
tent rises in Jamaicans under the influence of
halothane. It is, however, to be noted that the
rise in his cases, though undoubtedly significant,
was not commensurate with that occurring with
diethyl ether anaesthesia, nor are the final blood
sugar levels greatly different from those reported
in the present series.

ACKNOWLEDGMENTS
The blood sugar estimations were carried out by the
staff of the Biochemical Department of the Clinical
Laboratory of Manchester Royal Infirmary. The
author is indebted to Mr. H. Varley, the senior
biochemist, for his kind co-operation in this work.

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