CARDIAC ARREST FOLLOWING ADMINISTRATION OF A HIGH CONCENTRATION OF HALOTHANE VAPOUR

BY

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A PATIENT suffered cardiac arrest after being subjected to intermittent positive-pressure respiration with an unsuspectedly high concentration of halothane. The cardiac arrest was successfully treated and the high halothane concentration was found to be due to a mechanical fault in the vaporizer used in the anaesthetic circuit.

CASE REPORT

The patient was a 59-year-old man, admitted for left inguinal herniorrhaphy. At examination, it was found that he showed symmetrical and generalized diminution of chest movement on respiration. On account of this it was decided to use endotracheal anaesthesia with controlled respiration. His physical condition was otherwise normal with blood pressure 140/80 mm Hg.

On the day of operation, the sequence of events was as follows.

Time

0815

Pethidine 50 mg, and atropine 0.65 mg intramuscularly.

0905

Thiopentone 250 mg and suxamethonium 60 mg intravenously. After inflation of lungs with oxygen and spraying of larynx and upper trachea with 4 per cent lignocaine solution, no. 10 cuffed endotracheal tube passed and cuff inflated. Manually controlled respiration with nitrous oxide and oxygen (7 and 2 l. per min).

0922

Pethidine 20 mg I-V. Patient taken into theatre (still on I.P.P.R.). During preparation of operation site, attempt at spontaneous respiration. Halothane vaporizer set at 0.5 per cent mark and the lung inflated 3 times; setting increased to 1.0 per cent and the lung inflated 5 or 6 times; setting was then advanced to 1.5 per cent and maintained there.

0924

Incision made deeper. No active bleeding; no arterial pulsation palpable.

(It is estimated that by this time the lungs had been inflated some 40 times with vaporizer set at 1.5 per cent halothane vapour, with an imposed tidal volume of 500–600 ml). Vaporizer and nitrous oxide turned off: high flow-rate of oxygen by I.P.P.R. Table tilted steeply head down. Left chest opened through incision in 4th interspace. Heart found to be in asystole. No contractions after 3 or 4 manual compressions. Incision extended up through 4th costal cartilage and pericardium opened; ventricular fibrillation seen. Two shocks with defibrillator set at 125 V resulted in regular cardiac contractions. Cardiac output poor: descending aorta compressed. Immediate re-establishment of ventricular fibrillation. Two more shocks at 125 V resulted in cardiac arrest: one manual compression resulted in normal, rhythmic and effective cardiac contractions.

0927½

(Full oxygenation by I.P.P.R. maintained throughout. No enlargement of pupils beyond mid-dilatation.)

0930

Herniorrhaphy commenced (chest and pericardium left unsutured). Nitrous oxide and oxygen by I.P.P.R. (7 and 2 l./min).

1000

50 ml of 50 per cent sucrose solution I-V.

1010

Herniorrhaphy completed. A shallow incision, 2 cm long, was found in anterior surface of upper lobe of left lung; lung sutured—no visible signs of gas leakage on I.P.P.R. Pericardium closed.

1025

Closure of chest completed (under-water seal drainage through low axillary line incision).

1040

Patient sent from theatre. Respiration normal in character; pharyngeal airway tolerated. Blood pressure 125/75 mm Hg. Pupils small, brisk reaction to light; slow lateral eye movements.

1200

Patient still unconscious. Breathing stertorous; considerable restlessness. 50 ml of 50 per cent sucrose intravenously.

1205

Breathing normal; no restlessness. Surgical emphysema found, extending from left shoulder to left costal margin; air heard to escape from chest wound at each expiration but under-water seal drainage apparently satisfactory.

1215

Chest radiograph showed no evidence of lung collapse, pneumothorax or fluid in chest.

1235

Surgical emphysema spreading. Decided to re-open chest on account of possibility of unsuspected injury to lung during intrathoracic manipulations. Atropine 0.65 mg intravenously.
Intubation with No. 10 cuffed tube after thiopentone 75 mg and suxamethonium 40 mg. I.P.P.R. with nitrous oxide and oxygen, 7 and 2 l./min. Chest re-opened; found that the sutures at lung incision (see note made at 1010) had cut out. Intercostal muscle graft applied over tears.

Chest closed, with under water seal drainage. Patient's condition good throughout. Blood pressure 135/90 mm Hg.

50 ml of 50 per cent sucrose intravenously. Blood pressure 145/100 mm Hg. Respiration quiet; airway tolerated. Allowed to leave operating theatre.

1011 Patient opened eyes, stared blankly on gentle stimulation and became restless. Incontinent of urine; no abnormal neurological signs. Chest radiograph showed partial collapse of left lower lobe (this re-expanded steadily over the next few days.)

Rousable to drink small quantities of fluid. Deeply asleep when undisturbed.

Removal of his own anterior chest drain and went for short walk. Resentful of restraint; paraldehyde given intramuscularly.

Able to carry on coherent, though aggressive conversation. Retrgrade amnesia, extending back for about two weeks.

During 3rd day, period of retrogate amnesia had shortened to 5 days, by 5th day, this period had become reduced to pre-operative 24 hours, and his mentality appeared to be normal. Discharged from hospital on 15th day, and when seen as out-patient 6 weeks after operation was in excellent health.

The concentration of halothane with the control set at 3 per cent was such that one short inspiration made it necessary for the observer to sit down quickly.

The vaporizer was then tested by measuring the residual volume of halothane after passing 7 and 2 litres per minute of nitrous oxide and oxygen over a known volume of the agent for 10 minutes, with the control dial set at 1.5 per cent. In turning the dial rapidly from the “Off” to the 1.5 per cent position, difficulty was encountered in moving it beyond the 1.3 per cent mark. It was found that 41 ml of halothane had been taken up by the 90 litres of gases passed through the vaporizer; without making corrections for the prevailing physical conditions, this gave a concentration of 8.8 per cent halothane in the issuing gases.

Similar, but more accurate, tests were then carried out at various points over the vaporizer's whole calibrated range. With the exception noted in the next paragraph, there was difficulty in moving the control dial past the 1.3 per cent position. Checking the total nitrous oxide and

### Table I

<table>
<thead>
<tr>
<th>Control dial setting</th>
<th>Halothane used (g.)</th>
<th>Per cent halothane (vapour)</th>
<th>Column 2 Per cent halothane (vapour)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.3%</td>
<td>0</td>
<td>0</td>
<td>27–19</td>
</tr>
<tr>
<td>0.5%</td>
<td>73</td>
<td>8.6</td>
<td>0.57</td>
</tr>
<tr>
<td>0.6%</td>
<td>49</td>
<td>8.9</td>
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<tr>
<td>0.7%</td>
<td>69</td>
<td>8.0</td>
<td>1.4</td>
</tr>
<tr>
<td>0.8%</td>
<td>82</td>
<td>6.7</td>
<td>2.1</td>
</tr>
<tr>
<td>0.9%</td>
<td>82</td>
<td>5.8</td>
<td>3.8</td>
</tr>
<tr>
<td>1.0%</td>
<td>41</td>
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</tr>
<tr>
<td>4.5%</td>
<td>9</td>
<td>9.4</td>
<td></td>
</tr>
</tbody>
</table>

Physical conditions:
Room temperature: 21°C.
Barometer: 1009 mb.
Flow rates:
- nitrous oxide, 7 l./min.
- oxygen, 2 l./min.
Place: Bristol.

Table of Vaporizer Performance.
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oxygen flow as indicated on the rotameters against a Parkinson-Cowan meter showed that the flow was 88.9 litres in 10 minutes.

The results of these tests are set out in the first column of table I, and it will be seen that the first tests at control settings of 0.5 per cent, 1.0 per cent and 1.5 per cent were repeated and that the results were not reproducible. It had been found that no difficulty was encountered in returning the control dial to the “Off” position, or in taking it to the 1.5 per cent mark during the repeat experiments.

The events related above took place on a Friday, and, after establishing the fact that the vaporizer was faulty (and taking it out of service), other members of the anaesthetic staff were asked whether they had observed any unexpected results when using the apparatus. A total of 4 cases was reported by two anaesthetists. Their patients, anaesthetized on the Wednesday and Thursday, had rapidly developed apnoea while spontaneously breathing halothane-nitrous oxide-oxygen mixtures with the vaporizer control set at concentrations of from 1 per cent to 1.5 per cent. It was established that the vaporizer was functioning correctly on the Monday.

On the following day (Saturday), the death occurred of the first patient who had suffered cardiac arrest. (This patient had been anaesthetized on the Thursday night and was the one mentioned by the Theatre Sister during the operation proceedings detailed above. Subsequent enquiries indicated that halothane had not, in fact, been used in this case.) At this time, the survival of the writer’s patient was by no means certain; it therefore seemed essential to have the vaporizer tested more accurately than had hitherto been possible, and by an independent expert. Dr. H. G. Epstein, of the Nuffield Department of Anaesthetics, Oxford, very kindly agreed to conduct the tests, at which the manufacturers of the vaporizer were represented. The vaporizer was removed from the Boyle’s apparatus, and, in the course of demonstrating to a colleague the intermittent stiffness of movement of the control dial, marked difficulty was again encountered in turning the dial around the 1.3 per cent position. The vaporizer was handed to Dr. Epstein with the dial in this position, and his first test was conducted at this setting. The results of his tests are set out in the second column of table I. The observed fall in halothane concentration from 27 per cent to 19 per cent during the two-minute period of the first test (at 1.3 per cent dial setting) was probably due to cooling effects in the vaporizer, resulting from rapid evaporation of liquid halothane.

When Dr. Epstein had completed his tests, the manufacturer’s representative dismantled the control mechanism of the vaporizer, and this is shown partly “exploded” in figure 1.

When completely assembled, the part indicated by (A) is screwed on the spindle (B) so that the hole (C) lies in the plane marked (D...D). The control dial (E) partly covers the hole (C), which is tapped to take an Allen-

FIG. 1
Partially “exploded” view of control mechanism of vaporizer.
headed grub-screw with a cone point. It is the hold of this grub-screw on the spindle (B) at a point in plane (D...D) which causes the spindle to rotate helically when the control dial is turned, for the helical groove (F) is engaged with a peg in the main body of the apparatus.

When the vaporizer is assembled in the process of manufacture, the part (A) is threaded on spindle (B) to a position which experience indicates as being approximately correct. The grub-screw is then tightened to hold the control mechanism together, the apparatus charged with halothane and tested by refractometry. The portion of the spindle (G) governs passage of gases through a port into the halothane-containing chamber, and a relatively small advancement of this part (resulting from helical rotation of the whole spindle) causes a comparatively large increased diversion of gases into the chamber and consequently high halothane enrichment of the finally emergent gas-halothane mixture. The actual (longitudinal) position of the grub-screw on the spindle is therefore critical, for on it depends the point at which the part (G) of the spindle, under the influence of the helical groove (F), begins to allow gases to pick up halothane vapour.

It frequently happens that the initial (trial) setting of the grub-screw is proved by the refractometer to be incorrect. The screw is then slackened and the part (A) moved axially along the spindle to a different position, the amount of alteration being governed by the refractometer reading considered in the light of manufacturing experience. The apparatus is then retested, and the whole process is repeated until acceptable refractometer readings are obtained.

Figures 2 and 3 show detail of the spindle around plane D...D (v. fig. 1). The indentations (H), (J), (K), and (L) make it seem likely that
four trials were necessary to adjust this vaporizer to satisfactory performance.

Now, each time the screw is tightened, its point becomes blunted by an amount depending on the relative hardness of screw and of spindle, and on the force used. Figure 4 is a silhouette photograph of the screw removed from the faulty vaporizer, and the flattened point is readily seen both there and in figure 5.

Reviewing the evidence, we found: (1) inconstant difficulty in turning the control dial; (2) a blunted grub-screw; (3) a scored helical mark on the spindle. From these three points, we can reasonably deduce the sequence of events to be as follows. With repeated, normal, to-and-fro rotation of the control dial, the blunted tip of the grub-screw gradually enlarged its (final) indentation on the spindle (blunting itself still further in the process). There came a time when a normal rotation of the dial caused the screw-point to slip out of its indentation. The control assembly (A) (fig. 1) was then able to rotate independently of the spindle, carrying the grub-screw with it, and it probably so rotated until the "point" of the screw caught in one of the trial indentations on the spindle. During this rotation of the control assembly on the spindle,
on to the spindle by a right-hand thread, so that clockwise rotation of the dial to the "Off" position, aided by frictional resistance imposed on the spindle by tension in the spring (N) (fig. 1), ensures that however much (within the structural limits of the vaporizer) the dial assembly has become unscrewed, this assembly will travel down its thread and reach the position at which positive locking to the "Off" position will occur.

DISCUSSION
There is no doubt but that blame for the clinical events detailed above rests solely with the anaesthetic management of the case.

The original intention was to maintain anaesthesia with nitrous oxide and oxygen by hyperventilation, with supplementary intravenous injections of pethidine and relaxant given as required. The halothane was added to tide over a lapse from good management, and was used in an amount and in a manner believed by the writer to be safe. Subsequent events and investigations show that assumption to have been incorrect, and it is therefore necessary to examine the pharmacology of the agent in relation to the circumstances of the particular case.

From the time of introduction of halothane, it has been recognized that it is, by itself, capable of inducing cardiac arrhythmias. Raventós (1956), using concentrations of the vapour of up to 4 per cent, showed that cardiac arrhythmias did not develop in dogs anaesthetized with halothane unless adrenaline or noradrenaline were injected intravenously. Hall and Norris (1958), recording the death of a dog after 12 minutes exposure to a 4 per cent concentration of halothane, and noting that prior to death the animal had complete apnoea and a normal e.c.g., they questioned the alleged cardiotoxicity of halothane. They state that in their work no severe arrhythmias developed until long after respiratory arrest. On the other hand, Stephen et al. (1958a) showed that complete heart-block developed in dogs artificially ventilated with 5 per cent halothane vapour. They noted that recovery sometimes followed mere discontinuance of the halothane and maintenance of normal ventilation with oxygen.

There is conflicting evidence on the arrhythmia-inducing effect of adrenaline during halothane anaesthesia. Raventós (1956) found that large doses of adrenaline or noradrenaline could be injected subcutaneously or intramuscularly in dogs without producing ventricular disturbances. In man, whose myocardium is probably more sensitive to the effects of adrenaline than is that of the dog, Junkin et al. (1957), MacKay (1957) and Marrett (1957) all reported the injection of adrenaline-containing solutions without clinically noticeable cardiac effect. However, Brindle et al. (1957) suggested that the subcutaneous injection of adrenaline during uncomplicated halothane anaesthesia in man may be hazardous. They based this statement on the results of monitoring the e.c.g. following administration of 500 micrograms of adrenaline in 1:1,500 cinchocaine solution during halothane anaesthesia (vapour concentration usually under 1 per cent): they demonstrated the occurrence of supraventricular extrasystoles, pulsus bigeminus and multifocal bigeminal rhythm.

In the case under discussion, it is reasonable to believe that an adequate depth of anaesthesia had been reached by the time the surgical incision was made—even if the vaporizer had been delivering only the indicated concentration of halothane. It therefore seems unlikely that adrenaline-induced sensitivity of this patient's myocardium to halothane was responsible for the cardiac arrest.

In his first series of halothane anaesthesias, Johnstone (1956) observed that vagal-type arrhythmias occurred when vapour concentrations of from 2.2 per cent to 3.2 per cent were used for maintenance of anaesthesia and he found that these arrhythmias were abolished by atropine; their incidence was eliminated by adequate preanaesthetic atropinization. The necessity for proper atropinization was emphasized by Brennan et al. (1957), who stated that the administration of halothane after an inadequate dose of atropine permitted activation of the parasympathetic innervation of the heart, and led to the consequent likelihood of development of cardiac inhibition and arrest.

Brennan (1957) pointed out that hyoscine was no substitute for atropine as a means of protecting the heart in these circumstances. The validity
of these observations is emphasized by some reports on cardiac arrhythmias associated with halothane anaesthesia. Burnap et al. (1958) state that “minimal medication with a belladonna drug” may have accounted for the fact that in a series of 102 patients, 22 developed arrhythmias such as nodal rhythm, bifocal and multifocal ventricular extrasystoles and bradycardia, while Hudon et al. (1957) reported arrhythmias in 14 per cent of e.c.g.'s taken during halothane anaesthesia. These latter authors do not state the atropine dosage used—in fact, 10 per cent of the patients in the series reported did not receive any atropine.

Delaney (1958), using atropine premedication of 0.6 mg, commented on the association of abnormal e.c.g.'s and laryngeal reaction to intubation under halothane anaesthesia, and compared the coupled rhythm and ventricular extrasystoles seen in two such cases with the similar disturbances noted by Hill (1932) during induction of chloroform anaesthesia and regarded by him as a “preventricular fibrillation stage” which could pass unnoticed clinically. As mentioned above, Brindle et al. (1957) noted similar e.c.g.’s after injection of adrenaline during halothane anaesthesia.

Although Stephen et al. (1957) reported using halothane in concentrations of up to 3.5-4 per cent for induction of anaesthesia, after premedication with atropine or oxyphenonium 0.6 mg, they regarded cardiac arrhythmias as occurring less often and as being of no more serious nature than those occurring during cyclopropane anaesthesia. Burns et al. (1957), using morphia and either atropine or scopolamine in unstated doses for premedication, and maintaining anaesthesia with concentrations of halothane of from 1 per cent to 1.5 per cent, reported that e.c.g. monitoring confirmed the suspicion that most of the observed cardiac irregularities were, in fact, ventricular extrasystoles.

Johnstone (1957) stated his belief that positive pressure inflation of the lungs with halothane in the presence of inadequate atropinization is very likely to lead to vagal inhibition and arrest of the heart. He and co-workers (Brennan et al., 1957) defined adequate atropinization as being achieved by giving 0.6 mg of the drug with the induction dose of thiopentone, or, if halothane is used for induction, by giving the same dose intravenously one minute before commencing induction.

Reports have appeared of 10 cases of cardiac arrest associated with halothane anaesthesia. Chang et al. (1957), Foster (1957), and Stephen et al. (1958b), 1 case each. Hudon et al. (1957), and Carson et al. (1959), two cases each. Abajian et al. (1958 and 1959) and Mazuzan (1959), 3 cases. In addition, Robson and Sheridan (1957) reported a case in which, clinically, cardiac arrest was believed to have occurred. Such relevant details of these cases as are available are summarized in table II, and the following points are noteworthy:

1. The general inadequacy (by Johnstone’s standards) of atropinization.

2. Where there are sufficient data, the uniform association of controlled or assisted respiration and the placing of the halothane vaporizer within the circle system.

3. With one exception, all the patients made complete recoveries after cardiac massage and “washing out” the halothane. (Compare this with the animal experiments of Stephen et al. (1958a).

4. The case reported by Chang et al. had received atropine, 0.65 mg, intravenously 5 minutes before cardiac arrest occurred. During those 5 minutes, the patient had been given an increased concentration of halothane by intermittent positive-pressure respiration. These circumstances give practical point to the observation that large doses (2.0 mg or more) of atropine are required to free the heart from vagal control (Goodman and Gilman, 1955).

5. The presence, in 4 of the cases, of circumstances which would result in hypoxia and/or hypercarbia, with the attendant possibility of increased adrenaline output.

In the case of cardiac arrest here described, it is unlikely that hypoxia or hypercarbia were present. Overdose with halothane undoubtedly was present. Waters (1951) says of chloroform “Display of a sudden extremely high concentration may act centrally on the heart and cause a drop in blood pressure which, if severe enough and sudden enough, may be accompanied by cardiac arrest”. It does not seem likely that this mechanism accounted for the present case, as the excessive concentration of halothane had been...
### Table II

*Analysis of details of other recorded cases of cardiac arrest associated with halothane anaesthesia.*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Authors</th>
<th>Premedication (in mg)</th>
<th>Anaesthetic circuit, and position of vaporizer (in or out of circuit)</th>
<th>Respiration (spontaneous, assisted, or controlled)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Chang et al.</td>
<td>Morphia, 10 Hyoscine, 0.65</td>
<td>Closed In</td>
<td>Controlled</td>
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<tr>
<td></td>
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<td></td>
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<tr>
<td>2</td>
<td>Hudon et al.</td>
<td>0.4 Levorphanol, 2</td>
<td>Semi-closed ? In</td>
<td>Controlled</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Hudon et al.</td>
<td>0.3 Pethidine, 50</td>
<td>Semi-closed ? In</td>
<td>Controlled plus hyperventilation</td>
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<td></td>
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<td></td>
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<tr>
<td>4</td>
<td>Foster</td>
<td>Papaveretum, 10 Scopolamine, 0.4</td>
<td>Closed In</td>
<td>Assisted</td>
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<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>5</td>
<td>Abajian et al.</td>
<td>No details given</td>
<td>Open-drop Closed In</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Maxuzan</td>
<td></td>
<td></td>
<td></td>
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<td>7</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Stephen et al.</td>
<td>0.6 Quinalbarbitone, 100</td>
<td>Semi-closed In</td>
<td>Assisted and, later controlled</td>
</tr>
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<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Carson et al.</td>
<td>Not stated</td>
<td>Circuit not stated</td>
<td>Probably controlled</td>
</tr>
<tr>
<td>10</td>
<td>Robson and Sheridan</td>
<td>Not stated</td>
<td>Vapourizer probably &quot;In&quot;</td>
<td>Controlled</td>
</tr>
</tbody>
</table>

Atropine 0.65 mg intravenously about 5 min before cardiac arrest, on account of blood pressure and pulse-rate fall. Then, halothane concentration increased, with I.P.P.R. Ventricular fibrillation developed after cardiac massage. Duration of arrest 1–2 min. Recovery.

Patient stated to have been in state of “severe vagotonia” before anaesthesia. Patient tilted to reverse Trendelenburg position immediately before cardiac arrest developed. Duration of arrest—4 min. Recovery. Successfully anaesthetized with halothane 7 days later (technique not stated). “Defective oxygen outlet” from apparatus subsequently discovered.

Patient aged 10 years, stated to be “obese”. Atropine 0.2 mg intravenously just before cardiac arrest (on account of resistance to I.P.P.R. at time of drawing out cæcum for appendectomy). Recovery. “Defective oxygen outlet” from apparatus subsequently discovered.

Hypotensive episode on increasing halothane concentration early in anaesthesia. Reversed on washing out halothane with oxygen. Second episode on increasing halothane concentration to deepen anaesthesia for closure of abdomen; not reversed by I.P.P.R. with oxygen. Duration of arrest not stated; cardiac massage not effective soon enough. Death after 10 days unconsciousness.

All three cases recovered.

The cases anaesthetized with an in-circuit vaporizer occurred at a time when the apparatus used gave unknown and virtually uncontrollable halothane concentrations. Authors emphasize their belief that halothane anaesthesia requires precision vaporization with concentrations which never exceed 2 per cent.

Cardiac arrest developed 2 min after commencement of suxamethonium drip and controlled respiration, using same concentration as had been given by assisted respiration for previous 1½ hours. Recovery.

Stated to have been due to overdosage with halothane. Stated to have followed hypoxia episode (due to mechanical obstruction of endotracheal tube).

During halothane induction, episode of upper respiratory obstruction. Thiopentone and suxamethonium intravenously. Within one minute of resumption of manual ventilation after intubation, e.g., showed complete A.V. block and atypical A.V. nodal rhythm. 30 sec later, no recordable blood pressure. Skin incision for thoracotomy oozed blood, so—oxygen by I.P.P.R., head-down tilt. methoxamine 10 mg intravenously. Rapid recovery of blood pressure, e.g., took 12 min to show "awake" pattern (after showing profound anaesthesia or cerebral anoxia). E.G. returned to normal 5 minutes after restoration of blood pressure.
administered for at least 4 minutes before cardiac arrest occurred.

It seems more likely that the cause of arrest was vagal inhibition of the heart following delivery by positive-pressure respiration of a high concentration of halothane to an inadequately-atropinized patient. It is possible that the dosage of atropine would have been adequate for the intended temporary administration of the agent in a low concentration. Apart from ensuring proper atropinization of planned halothane anaesthesias, it is now the writer’s practice to have atropine 0.65 mg in a syringe lying on the table of any anaesthetic apparatus which he is using and which is equipped for giving halothane.

It does appear from this case, and from the others listed in table II, that early recognition of impending cardiac arrest can lead to successful reversal of the phenomenon by simply washing out the halothane by efficient lung ventilation with oxygen. Prevention lies in proper pre-medication with atropine and the employment of means of vaporizing the halothane which at all times deliver safe concentrations of the agent to the patient—not merely into the vehicle gas stream. In this latter connection, accuracy in performance and simplicity of design of vaporizers are all important. Simplicity is accompanied by a minimum of possible mechanical failures—any of which may occur unsuspected by the anaesthetist until he finds himself confronted with a major disaster. Whatever design of vaporizer ultimately proves the best, there can be no substitute for watchfulness on the part of the anaesthetist—with this agent as with any other.

It is worth noting that, using the vaporizer described in the present case, 2 anaesthetists experienced unexpected effects in 4 patients. Until that time there had been no reason to suspect the vaporizer’s performance. It is very likely that, had those 4 cases been reported to a senior anaesthetist, the faulty state of the apparatus would have been discovered in time to prevent the occurrence of the clinical events described in this communication.

**SUMMARY**

A case of cardiac arrest associated with halothane anaesthesia with ultimate recovery is presented.

Cardiac arrest was probably due to vagal inhibition of the heart in an inadequately-atropinized patient, following intermittent positive-pressure respiration with an unexpectedly high concentration of halothane.

The existence of a mechanical fault within a calibrated vaporizer was responsible for the unduly high concentration of halothane. The investigation and the nature of the fault are described.

Other reported cases of cardiac arrest associated with halothane anaesthesia are reviewed and some common factors noted.

**ACKNOWLEDGMENTS**

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Following the discovery of the mechanical fault in this particular vaporizer, the manufacturers have designed modifications which will be applied to existing vaporizers and incorporated in all new ones.

**REFERENCES**


BOOK REVIEW


This book, designed to cover comprehensively the study of modern anaesthetic practice and as a text for reference to its many aspects, has wisely been produced in two volumes. That they total well-nigh one thousand pages is the clearest indication that the field of anaesthesia is expanding to increasingly spacious horizons. It might well be thought that to read this book was a formidable task but such is far from the case—a happy circumstance perhaps due to the varied styles of its many famous authors. It is no exaggeration to say that the editors have so arranged the material that the subjects follow one another like the chapters of a good story-book. This is especially true of the first volume which deals with Basic Principles—not usually conducive to easy reading. The second volume is devoted to Techniques, Special Fields and Hazards and here, of course, it has been less easy to maintain a sense of continuity. The idea of the book as a text for reference is more prominent in this second part.

In the first volume, specialized aspects of basic subjects are treated by acknowledged authorities. In addition to chapters on anatomy and special physiology, there are original contributions on such topics as premedication, intravenous anaesthesia and blood transfusion. The Signs of Anaesthesia are given a new slant and even if the author of this chapter insists on death as the endpoint of anaesthesia, it is refreshing to be conducted downwards through some new country from which a happy ascent to recovery can be anticipated. There is the novelty of a chapter on the neurophysiology of anaesthesia and the prediction that this could be "quite a chapter" proved to be indeed true. It could not be expected that every chapter would be of equal strength though the standard is exceedingly high. Nevertheless the excellent chapter on Basal Narcosis by means of Rectal Injection might well be fitted in with the Principles of Premedication.

The second volume contains an abundance of information on the many techniques of anaesthesia in their application to all branches of surgery. The authors, each of whom again is universally accepted as a master of his subject, deal thoroughly with the many modern practices of anaesthesia, and they indicate clearly where this specialty has spread its benefits and specialized knowledge into wider medical fields. Particularly [continued on page 185]