HYPOTHERMIC VENTRICULAR FIBRILLATION AND HALOTHANE

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Ventricular fibrillation and cardiac arrest have always been — and still are — the most dreaded complications of anaesthesia. Yet nowadays, in certain branches of surgery, fibrillation or arrest are deliberately induced with the sure knowledge that, if produced under controlled circumstances, they are *always* reversible. The opportunity provided by these deliberately induced fibrillations has been used to compare ventricular fibrillation onset temperatures in hypothermia where the anaesthetic agent was halothane, with those where the anaesthetic agent was not halothane.

Material and Method

Open-heart surgery at Groote Schuur Hospital and the Red Cross War Memorial Children's Hospital provided the indication for the use of hypothermia and deliberately induced ventricular fibrillation. The hypothermic cardiopulmonary bypass circuit has been described in full.[1] Briefly, the circuit used was one where arterialized blood was pumped through a heat exchanger, so cooling it; the cooled blood was then pumped into the femoral artery and so distributed *via* the aorta to the various tissues of the body, cooling them.

Temperatures were recorded, using the Electric Universal Thermometer, type TE-3,* from the lower mid-oesophagus. This temperature approximates best to the cardiac temperature.[2,3]

Results

The data presented are temperature readings obtained from 132 consecutive patients who had hypothermia deliberately induced for open-heart surgery. The total series of 132 patients has been divided into two groups; a group of 58 patients who had halothane administered during cooling, and a group of 74 patients who did not have halothane, but had curare and perhaps ether. In the end result 42 'halothane hearts' and 41 'curare hearts' fibrillated, i.e. 83 patients had ventricular fibrillation at some stage and 49 not.

The lower mid-oesophageal temperatures at which ventricular fibrillation occurred are shown in Table I. Fig. 1 shows graphically the percentage of all cases that reached a certain oesophageal temperature that had already fibrillated by the time that oesophageal temperature had been reached.

It may be seen from Fig. 1 that the curves plotted are in all 3 groups similarly 'S'-shaped, but that the 'halothane' group is quite markedly shifted to the right away from the 'curare' group; e.g. when 25°C. had been reached, 50% of the 'curare hearts' had fibrillated whereas at that temperature only 30% of the 'halothane' group had fibrillated, and the 50% level was only reached at 21°C.

Statistically the difference between the two groups is highly significant.

TABLE I. VENTRICLES THAT HAD FIBRILLATED AT OR ABOVE THE OESOPHAGEAL TEMPERATURE REACHED

Oesophageal temperature °C	*dTC/Ether*		*Halothane*	
	Ventricles that fibrillated	*No. of patients*	*Ventricles that fibrillated*	*No. of patients*
37	0	74	0	58
36	0	73	0	58
35	1	72	1	57
34	2	63	1	55
33	4	53	1	48
32	5	48	1	47
31	6	44	3	46
30	10	42	5	45
29	12	42	5	45
28	14	42	6	45
27	14	41	9	45
26	14	41	10	45
25	20	41	14	45
24	24	41	17	43
23	30	41	18	43
22	32	41	20	42
21	37	41	23	42
20	38	41	28	42
19	39	41	31	42
18	40	41	35	42
17	40	41	37	42
16	40	41	37	42
15	41	41	40	42
14			40	42
13			40	42
12			41	42
11			41	42
10			42	42

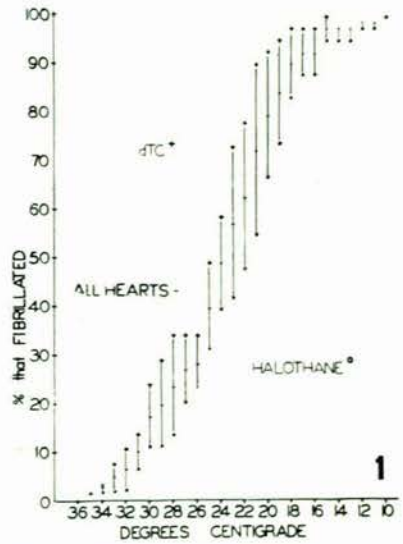

Fig. 1. The percentage of all patients who reached a certain oesophageal temperature who had already fibrillated by the time that oesophageal temperature had been reached.

DISCUSSION

From the results it is seen that when halothane was used the hearts tended to fibrillate at lower temperatures than those where curare and perhaps ether were used. Does halothane protect the heart against ventricular fibrillation?

Halothane, in spite of Orton and Morris'[4] speculation, does not itself protect the heart from anoxia[5] nor from hypercarbia,[6] and there is no reason to suppose that it should protect the heart from anything at all — in fact we know that halothane in its own right depresses the myocardium[7,8] and 'sensitizes' the heart to circulating catechol amines.[6]

In deep hypothermia there is an increase in the level of circulating catechol amines,[9] and the action of these is increased — which is only to be expected since the enzymatic action responsible for their removal has been slowed by the cold.[10] Administration of catechol amines to hypothermic hearts precipitates ventricular fibrillation.[11]

The sympatho-adrenal response in hypothermia may be blocked by bilateral sympathetic denervation or a sympathetic ganglion blocker like 'arfonad', thus reducing the concentration of circulating catechol amines, and this does in fact partially protect the heart against hypothermic fibrillation.[12]

Price *et al.*[13] showed that the level of circulating catechol amines in halothane anaesthesia is very low as opposed to diethyl ether anaesthesia.[14]

Hypothesis

In halothane anaesthesia the virtual absence of circulating catechol amines — in the absence of other stimulants to their presence such as anoxia or hypercarbia — means that as opposed to other anaesthetics, or lack of them, there is little circulating catechol amine to summate

* Manufactured by Elektrolaboratoriet, Copenhagen.

with that from any other cause of sympathetic nervous system discharge.

However, *if* catechol amines *are* released to any marked degree from any other cause of sympathetic nervous system stimulation, such as anoxia or hypercarbia, the halothane, far from protecting the heart, will in actual fact have sensitized the myocardium to the catechol amines.

SUMMARY

The incidence and temperature of onset of ventricular fibrillation in 132 patients having heart surgery performed using hypothermic cardiopulmonary bypass was determined. When halothane was the anaesthetic used hearts tended to fibrillate at lower temperatures than when curare and perhaps ether was used. The non-stimulation of the sympathetic nervous system by halothane is suggested as being the reason for this difference.

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REFERENCES

1. Barnard, C. N., Terblanche, J. and Ozinsky, J. (1961): S.Afr. Med. J., **35**, 107.
2. Cohen, D. and Hercus, V. (1959): Brit. Med. J., **1**, 1435.
3. Terblanche, J. and Barnard, C. N. (1962): S. Afr. Med. J., **36**, 337.
4. Orton, R. H. and Morris, K. N. (1959): Thorax, **14**, 39.
5. Stirling, G. R., Morris, K. N., Orton, R. H., Boake, W. C., Race, D. R., Kinross, F., Thomson, J. W. and Crosby, W. (1960): Brit. J. Anaesth., **32**, 262.
6. Black, G. W., Linde, H. W., Dripps, R. D. and Price, H. L. (1959): *Ibid.*, **31**, 238.
7. Morrow, D. H. and Morrow, A. G. (1961): Anesthesiology, **22**, 537.
8. Severinghaus, J. W. and Cullen, S. C. (1958): *Ibid.*, **19**, 165.
9. Brown, T. G. and Cotten, M. de V. (1956): Fed. Proc., **15**, 405.
10. Fuhrman, F. A., Crismon, J. M., Fuhrman, G. J. and Field, J. (1944): J. Pharmacol. Exp. Ther., **80**, 323.
11. Berne, R. M. (1954): Circulat. Res., **2**, 236.
12. Shumacker, H. B., Liberi, A., Boon, R. D. and Kajikuri, H. B. (1956): Ann. Surg., **143**, 223.
13. Price, H. L., Linde, H. W., Jones, R. E. and Black, G. W. (1959): Anesthesiology, **20**, 563.
14. Price, H. L. (1957): Clin. Sci., **16**, 377.