A REPORT ON MORTALITY AND MORBIDITY FOLLOWING 9,107
HYPOTENSIVE ANAESTHETICS*

BY

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This report covers the first ten years work with controlled hypotension (1950–1960) during which a series of 9,107 hypotensive anaesthetics has been administered. Of these 5,407 were administered by the author and the anaesthetic personnel of one hospital while the remaining 3,700 were administered personally in various hospitals and clinics. It would seem valuable to review the mortality and morbidity of this technique and to give for the first time an authentic analysis of a large series.

MORTALITY

There have been 9 deaths in association with 9,107 hypotensive anaesthetics (table I). This implies that death occurred at or after operation and that it was caused by the anaesthesia, the surgery or both. Of these 9 fatalities 4 followed cardiac arrest on the operation table. In 2 patients this was caused by inadequate ventilation and both responded to cardiac massage, but neither made a full recovery; 1 died after 13 days and the other after 28 days. Of the other 2 patients who suffered cardiac arrest 1 died from air embolism via the jugular veins, and the other from adrenal insufficiency.

The remaining 5 deaths occurred between 4 hours and 13 days after operation. In 2 instances death was due to respiratory obstruction, in one case caused by a spontaneous pneumothorax and in the other by airway difficulties in a coloured man half an hour after the operation had finished. The latter patient was resuscitated by cardiac massage but did not make a full recovery and died 4 days later. The third patient collapsed and died 20 hours after operation for block dissection of the glands of the neck for carcinoma. There is no clear indication from the records that inadequate ventilation, which can easily occur after this operation, played a decisive part in this fatal result but only that there was “cardiac collapse” in an old patient with malignant disease. The fourth fatality was caused by severe hypoglycaemia following an operation for osteomyelitis of the frontal bone in a diabetic tuberculous patient in whom pre-operative insulin was given in excessively large doses. Furthermore at that time the potentiation of insulin action by ganglion blocking drugs was not known.

The last fatality occurred 5 days after operation in a fat woman of 63 who was stone deaf. She underwent an extensive resection of the mandible for carcinoma and the blood pressure was not accurately controlled. The blood pressure was not accurately monitored, because this patient was operated on before the introduction of the oscillometer without which steady and accurate control is impossible. Her recovery from anaesthesia was apparently satisfactory and indeed she was discharged from the recovery ward the following day, but back in her own ward she was not co-operative and she did not appear to understand what was happening, although this aspect of her behaviour was difficult to assess because of her deafness. It is conceivable that her condition was caused by an acute mental disturbance precipitated either by operation or by hypotension or by both, and when she died 5 days later, from purulent bronchiolitis, no natural disease was found at autopsy to explain her mental condition. There is therefore some doubt as to the exact cause of this fatality.

Discussion.

From all these results it is apparent that death occurred four times because of inadequate ventilation (Case nos. 1, 2, 5, 6, in table I) in association with hypotensive drugs and surgery, and in
Table 1

Mortality.
Total 9 in 9,107 hypotensive anaesthetics.

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Cause of death</th>
<th>Time of occurrence</th>
<th>Time of death</th>
<th>Operation</th>
<th>Age</th>
<th>Sex</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Inadequate ventilation</td>
<td>During operation</td>
<td>28 days after operation</td>
<td>Decompression spine T.B.</td>
<td>F</td>
<td>16</td>
</tr>
<tr>
<td>2</td>
<td>Inadequate ventilation</td>
<td>During operation</td>
<td>13 days after operation</td>
<td>Facial plastic</td>
<td>F</td>
<td>42</td>
</tr>
<tr>
<td>3</td>
<td>Air embolism</td>
<td>During operation</td>
<td>During operation</td>
<td>Resection mandible, tongue and block dissection glands</td>
<td>M</td>
<td>24</td>
</tr>
<tr>
<td>4</td>
<td>Adrenal insufficiency</td>
<td>During operation</td>
<td>During operation</td>
<td>Plastic reconstruction of ear</td>
<td>F</td>
<td>40</td>
</tr>
<tr>
<td>5</td>
<td>Spontaneous pneumothorax</td>
<td>4 hours after operation</td>
<td>4 hours after operation</td>
<td>Excision mandible; block dissection glands neck</td>
<td>M</td>
<td>81</td>
</tr>
<tr>
<td>6</td>
<td>Acute respiratory obstruction</td>
<td>4 hours after operation</td>
<td>4 days after operation</td>
<td>Facial plastic</td>
<td>M</td>
<td>27</td>
</tr>
<tr>
<td>7</td>
<td>Cardiac collapse</td>
<td>20 hours after operation</td>
<td>20 hours after operation</td>
<td>Block dissection glands neck</td>
<td>F</td>
<td>63</td>
</tr>
<tr>
<td>8</td>
<td>Hypoglycaemic coma</td>
<td>During operation</td>
<td>4 days after operation</td>
<td>Osteoplastic forehead flap</td>
<td>M</td>
<td>45</td>
</tr>
<tr>
<td>9</td>
<td>Mental disturbance</td>
<td></td>
<td>5 days after operation</td>
<td>Excision mandible</td>
<td>F</td>
<td>63</td>
</tr>
</tbody>
</table>

Purulent bronchiolitis

A fifth patient (Case no. 7) this reason cannot be excluded with certainty. These results emphasize again what has been stated before—that the myocardium will not tolerate anoxia associated with hypotension. It is also vitally important to realize that residual ganglion paralysis after long-acting hypotensive drugs diminishes the capacity of the patient to withstand stresses such as respiratory obstruction or depression. When such drugs have not been given, a patient can often pull through these adverse conditions but a fatal outcome is very likely when the ganglia are paralyzed.

It is, therefore, chastening to realize that of these 9 deaths, 3 (Case nos. 1, 2, 6) were certainly avoidable by the basic principles of good anaesthesia, in particular by the maintenance of a clear airway and full oxygenation. Those associated with adrenal insufficiency and diabetes (Case nos. 4, 8) are, in the light of present knowledge of these conditions, avoided with greater certainty. It is doubtful whether the hypotensive technique was in any way responsible for the one death which occurred 20 hours after operation for block dissection of the glands of the neck (Case no. 7), whilst the patient with fatal air embolism (Case no. 3) died for reasons quite outside the control of the anaesthetist, and in association with an event which may occur at any time during an operation in the region of the great veins of the neck. The one unexplained death (Case no. 9) must be considered a likely complication of the hypotensive technique, particularly as the blood pressure was not carefully controlled but it must be remembered that this was an extensive and difficult operation in an unfit woman. This complication has not recurred in any other patient in this large series.

These figures give a mortality rate of 1 in 1,000 and it is tempting to compare this with the overall mortality rate at the Queen Victoria Hospital, East Grinstead, during the same 10-year period during which 30,950 operations were performed and 20 deaths occurred. But a detailed examination of these records shows that these deaths occurred from such a wide variety of causes that any attempt at comparison or statistics is quite worthless. Thus, for instance, this latter series includes death from coronary thrombosis 24 hours after operation and from the severe intoxication of burns 3 days after grafting, as well as deaths in the theatre from pulmonary
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embolism and from an obvious anaesthetic accident. It is, therefore, of no value whatever to lump all these together in order to give a neat figure for a mortality rate, for this has no meaning. It must be pointed out, however, that whereas inadequate ventilation was responsible for so many of the fatalities with hypotension, it did not figure prominently as a cause of death in the series without hypotension.

MORBIDITY

Cerebral thrombosis.

Cerebral thrombosis occurred once in this series. A female patient, aged 25 and physically fit, developed a left-sided hemiplegia 12–16 hours after full recovery from an extensive breast operation performed under hypotension. The operation was uncomplicated in every way and the systolic blood pressure, which had not been lower than 70 mm Hg at any time during operation, quickly rose to 90–100 mm Hg on return to bed. Consciousness was soon regained and thereafter the patient appeared normal in every way, but during the early hours of the following morning paralysis was discovered. Eventually she made an almost complete recovery. The most likely explanation of this complication is found in the fact that she was nursed in the sitting position after operation and was given an injection of chlorpromazine 50 mg, 4 hours after operation and morphine 10 mg, 6 hours after that. No check was made of the blood pressure after leaving the theatre. It was not at that time appreciated that chlorpromazine often induces a very considerable fall of blood pressure when given after hypotensive drugs and that its hypotensive action is further enhanced by morphine. There is little doubt that this patient suffered a prolonged period of hypotension, probably of severe degree, induced by these drugs together with her sitting position. This would not have occurred if a postoperative record of blood pressure had been kept as is now the practice for all patients. Chlorpromazine and other drugs with a hypotensive action should be avoided during the recovery period. The long-acting hypotensive agents (such as pentolinium and hexamethonium) paralyze ganglion transmission for many hours after operation and during this time all other drugs should be used with considerable caution.

Mental sequelae.

There is no evidence that mental changes of any kind have been induced by the hypotension. Disturbances in the elderly patient are more closely related to the mental stability of the patient and to the type, nature and success of the operative procedure. One male patient, aged 46 years, became aggressive and delirious 11 days after full recovery from an operation for forehead rhinoplasty under hypotension, and the condition was diagnosed by the psychiatrist as an acute paranoid episode. After treatment for 4 days he suddenly appeared to wake up and became normal. He apologized profusely for his previous accusations and obstructiveness and was thereafter normal with a satisfactory result after replacement of the flap. He committed suicide 6 months later after a recurrence of carcinoma of the stomach, for which he had received operative treatment previously and which diagnosis was known to him.

Occasionally the elderly patient is confused for several hours after operation, but this has been observed as frequently after non-hypotensive anaesthetics. There are occasional complaints of lethargy and depression after hypotension, but again not more frequently than when hypotension is not used. Indeed there have been many opportunities for comparing hypotensive and non-hypotensive anaesthetics in the same patient at consecutive operations, and they again point out the unreliability of such symptoms in assessing the possibility of mental effects arising from this technique. Complaints occur, sometimes with hypotension and sometimes without, in such a way as to indicate that the hypotension itself bears no relationship to the symptoms. On the other hand many anaesthetists support the observation that patients who have been subjected to extensive surgery under hypotensive ganglion blocking drugs are surprisingly fit after operation. This “anti-shock” effect appears clinically to be of real value in extensive operations.

The opinion of the Recovery Ward Sister at East Grinstead after many years experience of these patients is that recovery from anaesthesia with hypotension is indistinguishable from that without.
The vomiting rate as estimated on a recent series of 300 patients, half with hypotension and half without, shows no significant difference between the two series. Sex, age, operation, premedicant and anaesthetic drugs, are but some of the many factors affecting postoperative vomiting and it is therefore impossible to obtain two series for comparison in which all the variables are constant except blood pressure. It can, however, be stated with confidence that clinically there is no very obvious difference between those who have had hypotension and those who have not.

Cardiac massage.

One cardiac arrest with full recovery after cardiac massage occurred during the writer's early experience with the technique (Enderby, 1952). This was caused by inadequate ventilation (due to the position of the patient) at the commencement of hypotension, and only bears out again what has already been stated in this respect. It is included in this section because, although the patient made a full recovery, the necessity for such drastic action must be considered a part of the morbidity rate. It is perhaps unnecessary to point out that this complication is avoidable.

There was likewise one other collapse on the table shortly after the start of a thyroidectomy operation, again caused by respiratory obstruction, in this case due to a partially kinked endotracheal tube which had not been noticed earlier. There was a sudden fall of blood pressure from 70 mm Hg to an unrecordable level (again this occurred before the introduction of the oscillograph) and at once the table was changed to a head-down position and an incision made for cardiac massage. No sooner had the skin been incised than the patient made an equally sudden recovery. Cardiac massage was not performed and the operation was completed satisfactorily. The head-up position was partially responsible for the kinking of the intratracheal tube which was relieved by the change of position. It is quite clear too that the heart had not stopped beating when resuscitation was started and the increased venous return consequent on the change of position together with an improvement in the airway avoided an otherwise certain cardiac arrest and resulted in recovery.

Both these latter complications were in the writer's personal series of 3,700 operations, and it is worth recording that they both occurred during the first 500 operations of the series. They point dramatically to the absolute necessity for perfect anaesthesia as the only sound basis for safe hypotension. This complication has not reoccurred in the large number of hypotensive anaesthetics performed since then.

CONCLUSIONS

What conclusions can be drawn from this large series? How safe is hypotension and how justified are anaesthetists in adding ganglion paralysis to unconsciousness? The early mistakes which were responsible for one death (respiratory depression) should no longer occur, and indeed it seems fair to observe that the skill and knowledge of the trained anaesthetist is now capable of giving adequate hypotensive operating conditions with safety. Cardiac arrest during operation has not recurred since the early days, but this must be attributed to the increased care and skill devoted to an exact knowledge of blood pressure and anaesthetic requirements. Without this care and skill the risk of cardiac arrest is considerably greater with hypotensive drugs. These figures point out that such care and skill must be carried on into the immediate postoperative period and this makes the provision of skilled nursing a necessity during recovery.

The advantages of hypotension cannot be ignored in trying to assess justification for a technique with known dangers. From these results, however, it is fair to state that many of these dangers are now clearly understood, and indeed that the study of hypotensive anaesthesia has greatly enriched knowledge as to the mechanisms leading to sudden collapse and cardiac failure. The cause of death and the circumstances leading up to it are clearly understood in 8 of these 9 deaths which have been associated with hypotensive drugs. It is clear that these causes are in large part avoidable, but whereas the skill of the anaesthetist may be capable of avoiding them, it must be remembered that others are involved in the care of these patients and the level of skill throughout the surgical and nursing team may not be adequate to cope with every eventuality. But it must also be remembered, that where knowledge exists, skill will follow,
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particularly when that skill is obtained by careful and exact training.

SUMMARY

This series represents 10 years work by the staff of the Queen Victoria Hospital, East Grinstead (a total of 5,407 cases), together with a further 3,700 cases anaesthetized by the author in other hospitals and clinics.

Nine fatalities occurred in association with the 9,107 hypotensive anaesthetics. Four of these, and possibly a fifth, were caused by inadequate ventilation. Air embolism, adrenal insufficiency and diabetic hypoglycaemia caused the other fatalities. In one patient cerebral complications were possibly the result of the hypotension. During the same period there was a total of 30,950 operations at Queen Victoria Hospital, East Grinstead, and these showed an overall mortality of 20. No direct comparison is made between these figures and the hypotensive series because of the differing circumstances of death which they include, but it is significant to note that inadequate ventilation as the cause of death did not figure prominently, except in association with ganglion blocking drugs and hypotension.

Cerebral thrombosis occurred in one patient. There were no other cerebral or mental complications.

Cardiac massage successfully resuscitated one patient. Another patient collapsed at the start of operation but recovered before cardiac massage was commenced.

No significant difference was noted in the vomiting rate when compared with a similar series of non-hypotensive anaesthetics.

No other complications were reported which were attributable to the hypotension.

REFERENCE


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