DEATH ON THE OPERATING TABLE

By E. Faulkner Hill, M.D.
Senior Anaesthetist, Royal Infirmary, Manchester

and A. R. Hunter, M.D., F.R.F.P.S.G., D.A.
from Royal Infirmary, Manchester

DEATH on the table is fortunately not a common accident but its rarity makes it all the more difficult to obtain any definite information concerning its causation. The personal experience of a single anaesthetist, even over a long period, is unlikely to provide enough material for a study of the phenomenon. We have therefore collected from the records of the hospital which we serve the details of all the deaths which have occurred under anaesthesia in the last 14 years. In all 77 patients died on the table in this period. In a few cases no adequate explanation of the accident could be obtained from the information available, but in most the cause of death was clear enough. The cases are classified in Table I. It may seem in the light of modern knowledge that some of the patients should not have died, but it must be remembered that the period under consideration extends to 15 years ago. Also fully two-thirds of the anaesthetics were administered by junior members of the resident medical staff.

Each of the different groups in Table I calls for some comment.

In the 10 cases of death under spinal anaesthesia the anaesthetist was in the vast majority a recently qualified house-surgeon. All the patients were poor risks. The solutions were heavier than the cerebrospinal fluid and if the technique taught (viz. 3° to 5° Trendelenberg position with the head raised) was used there could be no possibility of the paralysis of the phrenic roots by the direct action of the solution. The cause of death must therefore be sought elsewhere than in respiratory paralysis, and in fact there were no records of this phenomenon. That the proximate cause of these catastrophes
TABLE I

Causes of Death on the Operating Table

1. Deaths due to Anaesthetic

<table>
<thead>
<tr>
<th>Anaesthetic</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spinal</td>
<td>10</td>
</tr>
<tr>
<td>Ether (convulsions)</td>
<td>6</td>
</tr>
<tr>
<td>Chloroform</td>
<td>4</td>
</tr>
</tbody>
</table>

2. Deaths due to Operation or Disease for which it was performed

<table>
<thead>
<tr>
<th>Disease</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moribund patient</td>
<td>10</td>
</tr>
<tr>
<td>Medullary compression</td>
<td>7</td>
</tr>
<tr>
<td>Uncontrollable haemorrhage</td>
<td>4</td>
</tr>
<tr>
<td>Shock</td>
<td>13</td>
</tr>
<tr>
<td>Thyroidectomy</td>
<td>14</td>
</tr>
</tbody>
</table>

3. Other Causes

<table>
<thead>
<tr>
<th>Cause</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspiration vomitus</td>
<td>2</td>
</tr>
<tr>
<td>Bronchial spill over</td>
<td>2</td>
</tr>
<tr>
<td>Unexplained: On the table</td>
<td>3</td>
</tr>
<tr>
<td>In bed immediately after</td>
<td>2</td>
</tr>
</tbody>
</table>

was heart failure seems to admit of no doubt, but as a spinal anaesthetic has no direct action on the myocardium the question arises as to how cardiovascular failure develops. In the experimental laboratory it has hitherto proved impossible to produce heart failure under spinal anaesthesia whatever the fall in blood pressure, provided the animal is supplied with oxygen. Anoxia therefore would seem to be the undeniable predecessor of the heart failure. There were many factors tending to produce anoxia in the patients of the present series. First and foremost their condition was unsatis-
factory. Some were obese; others were very ill; the medullary
centres were often depressed by drugs. The Trendelenberg
position added to their difficulties. Vasoconstriction was
probably inadequate even in the unaffected parts of the body
because the patients were too ill to have normal vasomotor
responses. Some must have had feeble fatty hearts. Their
respiratory centres lacked the stimuli they would normally
have received from the anaesthetised two-thirds of the body.
All these factors may have played a part in producing
anoxaemia in the blood entering the coronary arteries of the
heart. Under such circumstances a vicious cycle appears in
which the anoxia depresses cardiac function and thus
exaggerates the oxygen lack until such time as there is not
enough oxygen in the coronaries to allow the cardiac muscle
cells to carry on, and so the heart fails. Such a condition will
naturally develop more quickly in patients who are suffering
from hæmorrhage or dehydration. It is manifestly impossible
to evaluate the part played by each of these factors in the cases
studied, but many of them must have been operative. In
two patients death occurred shortly after the induction of
supplementary nitrous oxide and oxygen anaesthesia. In
these two instances it is almost certain that the added anoxia
produced by the administration of nitrous oxide with too little
oxygen to support life was the primary cause of the tragedy.
The 6 deaths from ether convulsions should occasion little
surprise. It was from Manchester in 1926 that the earliest
descriptions of the conditions came, and though they have
not been reported, there have been a few cases each year in
the area. It is significant, however, that in our own hospital
no deaths have occurred since 1941, i.e., since it became
generally known that an adequate dose of a barbiturate would
control the convulsions.
Chloroform deaths from primary cardiac failure are like-
wise becoming a thing of the past as this agent is falling into
general disuse. They still, however, occur occasionally.
Primary cardiac failure during thyroideectomy remains
depressingly common. In this series its incidence was
independent of the type of anaesthetic and premedication
given. Basal narcosis with avertin or paraldehyde did not
protect the patient. Death took place under nitrous oxide and oxygen alone. All the usual agents used as supplements were associated with such accidents. Ether, chloroform and trilene all were incriminated. The cause of the collapse was presumably ventricular fibrillation, the result of undue irritability of the cardiac muscle, produced by thyroid intoxication. The fact that 9 of the 14 patients in this group were over 40 supports this presumption, as it is the secondary form of the disease which is associated with other cardiac complications, notably auricular fibrillation.

Ten of the 77 patients in the series seemed to be almost in articulo mortis at the time at which operation was performed. There is nothing to be gained from further discussion of these cases. In every instance the patient was desperately ill, usually because of an infective condition, e.g., mediastinitis, spreading gangrene of the abdominal wall. Another had already had a cardio-omentopexy and was about to undergo repair of diaphragmatic hernia. He died shortly after a dose of avertin.

The neurosurgical unit deaths fall into two groups, those where compression of the vital centres was responsible and those where hemorrhage too severe to be controlled by ordinary methods took place. Accidents of the latter type are much less common nowadays with blood-bank facilities, but in this connection it should be noted that where extremely severe hemorrhage is anticipated fluid can be given much more quickly by a French's needle into an arm vein than through a gold cannula tied into the saphenous vein. Failure of the medullary centres during operations in their immediate neighbourhood is relatively rare. It took place only twice in the period under review. In two other cases the indirectly compressed hypothalamic centres in the floor of the third ventricle ceased to function during anaesthesia. Disturbance of both these areas of the brain progressing to a fatal issue is much more common in the immediate post-operative period. In patients with cerebellar abscesses, however, irreversible failure of breathing from spread of inflammatory oedema to the respiratory centre is not very common. It is therefore much more satisfactory to tap these abscesses under
local anaesthesia and to take the risk of wound infection, which in these days of penicillin and sulphonamides is almost negligible. A mastoid operation can if necessary be performed subsequently under general anaesthesia. Even so, special care must be taken to use only the lightest possible levels of anaesthesia and to prevent either oxygen lack or carbon dioxide excess, both of which are potent causes of cerebral oedema. If apnoea does appear the patient’s lungs should be rhythmically inflated with oxygen and 3–5 c.c. of nikethamide injected intravenously. This drug will often restart respiration in such cases.

The heading of shock is unsatisfactory and this group certainly includes some patients in whom the anaesthetic was at least in part responsible for the fatal issue. Some of the patients were found to be pulseless and apnoeic at the end of long abdominal operations. Two were air-raid casualties and one patient had bled severely during an incomplete abortion. In these last cases the administration of an anaesthetic and the slight loss of blood associated with the operation initiated a progressive decline in blood pressure which proved fatal before its seriousness was appreciated. In every instance in this group the anaesthetic was ether and it is probable that the fatal accident was due to a failure to appreciate the adverse effect of this agent on a patient with an already inefficient circulation. It is significant that no similar cases occurred under nitrous oxide and oxygen nor under spinal anaesthesia, though the latter is the method of choice in the hospital for emergency abdominal surgery.

Drowning of the patient in his own secretions is a well-recognized risk of anaesthesia. It happened four times in the present series. Two patients, both suffering from intestinal obstruction, died from aspiration of vomitus. In neither instance had the stomach been drained adequately before operation nor was there a stomach tube in situ at the time of the accident. In both cases the anaesthetic was given by a house surgeon. One operation was carried out under general anaesthesia; in the other case the patient collapsed under spinal anaesthesia, vomited and aspirated the vomitus while semi-conscious. There have been no accidents of this type
Death on the Operating Table 29

since 1934. It should be noted that the same mishap may complicate anaesthesia in cases of pyloric stenosis. It is therefore important not merely to teach nurses that the stomach should be washed out prior to such operations but to ensure that they know not to pass a Ryle’s tube so far into the stomach that it kinks and ceases to drain before the stomach is empty.

The other form of drowning during anaesthesia is a risk peculiar to major thoracic surgery in "wet" patients. When the pleura is opened the diseased lung collapses and ejects the fluid contained in the cavities within it into the bronchi and trachea, which are at once occluded. The patient becomes progressively more cyanosed and eventually dies of asphyxia. The same accident may occur in similar cases when a bronchoscope induces violent coughing. It is to be noted that even careful postural drainage before operation is not a complete safeguard against such tragedies. This accident has occurred only twice in the last 14 years in Manchester Royal Infirmary. A mishap which befell one of us at another hospital in the area indicates, however, by how narrow a margin such calamities may be averted.

A bronchiectatic young man aged 24 was undergoing a lobectomy under general anaesthesia. Shortly after the pleura was opened the movement of the bag on the machine ceased and the patient became cyanosed. Inflation of the lungs with oxygen caused only slight improvement in his colour. There were no moist sounds in the airway and suction applied to the catheter in situ produced no result. In desperation the anaesthetist removed the endotracheal tube and with difficulty inserted a fresh one. He was then able to improve the patient’s colour considerably by inflating the lungs with oxygen, but when anaesthesia was reinduced cyanosis again appeared. The chest was therefore rapidly closed and at the conclusion of the operation a quantity of extremely thick viscid pus was removed from the bronchi by means of a bronchoscope. The endotracheal tube and the suction catheter originally inserted were both completely blocked by the same thick pus. What was particularly terrifying about this case was the way in which sudden flooding of the tracheo-
bronchial tree occurred without a single moist sound in the airway to indicate what had happened.

In 5 cases no satisfactory explanation of the death could be elicited from the hospital records. In 2 instances the patient was found dead in bed shortly after the anaesthetic had been withdrawn. The presumptive cause of death was obstruction of the airway, and the lesson to be learned is the importance of educating nurses in the proper handling of the unconscious patient.

The frequency of death on the table with different anaesthetics is not easy to obtain. For reasons connected with the recent war the hospital statistics from 1939 to 1946 are incomplete. Details of all the anaesthetic deaths from 1933 to 1946 are, however, available. The crude death rate for this period during which approximately 124,500 operations were performed was 0.063 per cent. If, however, the 25,000 minor procedures which were carried out under local and gas anaesthesia without mortality are discounted the mortality becomes 0.78 per cent. The figure for death under spinal anaesthesia, 11 in 29,500 cases, or 0.049 per cent, compares very favourably with that for operations under general anaesthetics (0.077 per cent). This is particularly noteworthy, since spinal anaesthesia is the method of choice for emergency abdominal surgery. The only single group which calls for comment is that of thyroidectomy for thyrotoxicosis, which carries a mortality five times that for all other operations. The significance of this figure as regards the risk of surgical interference in the condition is clear enough but unfortunately no satisfactory means of prophylaxis has emerged from this study.

SUMMARY

The case records of 77 patients who died during anaesthesia have been analyzed. The various causes of death have been classified and where such is possible the means of prophylaxis of accidents of this kind has been indicated. Spinal anaesthesia had the lowest death rate on the operating table. The mortality in the operating theatre during thyroidectomy for toxic goitre was heavy.
### TABLE II
**Statistical Frequency of Death on the Table**

<table>
<thead>
<tr>
<th>Type of Anaesthetic</th>
<th>Number</th>
<th>Deaths</th>
<th>Death Rate per 100 Operations</th>
</tr>
</thead>
<tbody>
<tr>
<td>All cases (1933-46)</td>
<td>124,500</td>
<td>77</td>
<td>0.063</td>
</tr>
<tr>
<td>Major operations only</td>
<td>99,500</td>
<td>77</td>
<td>0.078</td>
</tr>
<tr>
<td>Major operations under general anaesthesia</td>
<td>77,000</td>
<td>66</td>
<td>0.087</td>
</tr>
<tr>
<td>Major operations under spinal anaesthesia</td>
<td>22,500</td>
<td>11</td>
<td>0.045</td>
</tr>
<tr>
<td>Thyroidectomy for thyrotoxicosis (1933-38)</td>
<td>1,528</td>
<td>5</td>
<td>0.32</td>
</tr>
</tbody>
</table>