DEATHS ASSOCIATED WITH ANAESTHESIA AND SURGERY*

A CRITICAL ANALYSIS OF 200 CASES

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The first recorded death under general anaesthesia appears to have been reported in the *Lancet* in 1848—a 15-year-old girl was given chloroform for the removal of a nail from an infected toe. After a few breaths, she became rigid and apparently died very quickly. The jury's verdict was—'We are unanimously of opinion that the deceased, Hannah Greener, died from congestion of the lungs from the effect of chloroform, and that no blame can be attached to Mr. Meggison, surgeon, or to his assistant, Mr. Lloyd.'[1] The search for an ideal anaesthetic has proceeded ever since—so far without success.

In 1949 the Council of the Association of Anaesthetists of Great Britain and Ireland appointed a Committee of Investigation to examine all cases of death associated with anaesthesia.[2] This Committee has published numerous reports,[3-8] the most recent one in 1956 containing a record of 1,000 deaths associated with anaesthesia.[9]

Investigations by various Anaesthetic Study Commissions in the USA and Canada have furnished a number of reports.[10-13] The most sensational report from America was the one published in 1954, by Beecher and Todd, which

* A paper presented at the South African Medical Congress, Durban, September 1957.

was based on a study of deaths associated with anaesthesia and surgery in 10 institutions, covering the 5 years 1948-52[14] (see below).

In South Africa the 'Orenstein Committee' appointed by the Minister of Health to investigate deaths under anaesthesia, published a report in 1936.[15]

In 1954 the Transvaal Provincial Administration appointed a Commission to investigate all deaths associated with anaesthesia in the Transvaal public hospitals. As a result of the recommendations of this Commission, all public hospitals in the Transvaal were instructed to supply the Director of Hospital Services with full details of all deaths associated with anaesthesia, from 1 October 1955. A further directive also made it compulsory for all Transvaal public hospitals to supply this department every 3 months, from 1 January 1957, with details of all anaesthetics administered, and prescribed particulars of all deaths occurring in their surgical departments.[16] The scope of the investigation has therefore been widened, and is not limited to deaths associated with anaesthesia. The voluntary co-operation of several hospitals outside the Transvaal province, notably hospitals in Natal, has also been obtained in this project.

In 1955 the South African Society of Anaesthetists appointed a sub-committee to investigate the problem, and to collect data from anaesthetists in private practice. The present communication reports on the first 200 deaths *associated with anaesthesia only*, which have been received from the various hospitals during the last 2 years. In other words, it deals only with surgical deaths referred to a district surgeon for autopsy in terms of section 86 of the Medical, Dental and Pharmacy Act 1928, which reads as follows: 'The death of a person whilst under the influence of a general or local anaesthetic, or of which the administration of an anaesthetic has been a contributory cause, shall not be deemed to be a death from natural causes within the meaning of the Inquest Act (1919) or the Births, Marriages and Deaths Registration Act (1923) or any amendments of these Acts.'

One of the recommendations of the 'Orenstein Committee' published in 1936 was that this clause 'operates unfairly to anaesthetists, who are frequently called upon to anaesthetize moribund cases with the practical certainty of having to face a public inquest. The result is to create a state of mind in the anaesthetists which tends to endanger the safety of the patient by unduly hurrying the surgeon.'[15] This recommendation to amend the Act has not yet been accepted.

PRELIMINARY ANALYSIS OF DATA RECEIVED FROM HOSPITALS

The present report is of a preliminary nature and does not warrant conclusions on the relative safety of various drugs—upon which I hope to comment at a later date. At this stage deaths have been classified on the lines adopted in 1956 by the Committee of Investigation in Great Britain,[9] as follows:

CLASSIFICATION OF DEATHS ASSOCIATED WITH ANAESTHESIA AND SURGERY

1. Where it was reasonably certain that death was caused by the anaesthetic agent or technique or administration, or in other ways coming entirely within the anaesthetist's province.
2. Similar cases, but in which there was some element of doubt whether the agent or technique was entirely responsible for the fatal result.
3. Cases in which the patient's death was caused both by the surgical and anaesthetic techniques.
4. Deaths entirely referable to surgical technique, e.g. uncontrolled haemorrhage.
5. Inevitable deaths, e.g. cases of severe general peritonitis, but in which the anaesthetic and surgical techniques were apparently satisfactory.
6. Fortuitous deaths, e.g. those due to pulmonary embolism.
7. Cases the Committee could not assess despite considerable data.
8. Cases on which an opinion could not be formed on account of inadequate data.

In the present analysis, 89 deaths (44½%) fell into the first 3 categories (those where the anaesthetic played a major role) and comment will be mainly confined to these (Table I). The classification by race, sex and age is shown in Tables

TABLE I. 200 DEATHS ASSOCIATED WITH ANAESTHESIA AND SURGERY. CATEGORY AND SITE OF OPERATION

Site of Operation	*Anaesthetic (reasonably certain)* 1	*Anaesthetic (very probable)* 2	*Surgical and Anaesthetic* 3	*Surgical* 4	*Inevitable* 5	*Fortuitous* 6	*Unassessable* 7	*Inadequate Data* 8	*Total*	*Total Anaesthetic (1, 2, 3)*	*% Anaesthetic*
E.N.T.	3	1	3	—	3	—	1	1	12	7	58
Obstetric	3	—	1	2	3	3	2	1	15	4	27
G.U.	5	2	1	—	3	—	—	—	11	8	73
Stomach and Duodenum	2	—	2	1	3	—	1	—	9	4	44
Small and Large Bowel	9	5	8	1	25	—	2	2	52	22	42
Other Abdominal	2	—	2	6	5	—	2	—	17	4	23
Hernia (all types)	2	1	2	—	2	—	1	—	8	5	62
Thoracic	2	—	3	12	5	1	—	—	23	5	22
Endoscopy	2	1	1	—	—	—	—	—	4	4	100
Thyroid and other Neck operations	2	—	—	—	—	1	—	1	4	2	50
Dental and Oral	1	—	—	—	—	—	1	—	2	1	50
Orthopaedic	7	1	3	—	4	3	—	2	20	11	55
Plastic and Superficial	5	—	—	1	—	—	—	1	7	5	71
Neurosurgical	—	—	3	2	4	—	—	—	9	3	33
Gynaecological	1	—	—	1	—	—	1	1	4	1	25
Other	2	1	—	—	—	—	—	—	3	3	100
Total	48	12	29	26	57	8	11	9	200	89	44

II and III. The greater number of deaths in males can probably be ascribed to a high relative incidence of serious trauma among non-European males. The stage of anaesthesia at which death occurred appears in Table IV; the status of surgeon and anaesthetist in Table V.

TABLE II. AGE DISTRIBUTION

Decade	No. of Cases	% of Total
1	26	13
2	14	7
3	39	19·5
4	29	14·5
5	29	14·5
6	28	14
7	25	12·5
8	10	5
Total	200	100

TABLE III. RACE AND SEX DISTRIBUTION

	Eur.	Non-Eur.	Total
Males	43	77	120
Females	31	49	80
	74	126	200

TABLE IV. STAGE AT WHICH DEATH OCCURRED

Time of Death	No. of Cases	%
Induction	25	12½
Maintenance	88	44
½ hour post-op.	40	20
Later	47	23½
	200	100

TABLE V. STATUS OF SURGEON AND ANAESTHETIST

Status	Surg.	Anaesth.
Specialist	103	60
Registrar	48	77
G.P.	45	55
Intern	4	8
	200	200

Pre-operative Status

Pre-operative assessment of all cases roughly followed the 4 categories suggested by Adriani[17] (Table VI).

Category A. A patient having no systemic defects who is undergoing a major or a minor operation, and who is in a fit state generally before the operation.

Category B. A patient having a minor systemic defect who is undergoing a major operation, but whose general condition before operation is satisfactory.

Category C. A patient who is undergoing a major operation, but who has in addition to his surgical condition another disease which would not in itself prove immediately fatal, or whose general condition before the operation is very poor.

Category D. A patient who is undergoing a major operation, but who has in addition to his surgical condition a disease which itself might prove immediately fatal, or who is moribund from his surgical condition.

Such assessment of operative risk is largely a matter of opinion, as is also the grading of an operation as major or minor. Table VI has been compiled with full recognition of these difficulties.

TABLE VI. PRE-OPERATIVE STATUS

A	20	10% (Good).
B	40	20% (Fair).
C	84	42% (Poor).
D	56	28% (Moribund).
	200	100

COMMENT

1. *Resuscitation*

As 90 deaths (45%) occurred with abdominal operations—54 (27%) for acute intestinal obstruction (Table X) an inescapable deduction appears to be that lives may have been saved had more effective measures of resuscitation been adopted, not only at the time when the catastrophe occurred, but also in the immediate pre-operative period. One often reads of operations being commenced with a systolic blood pressure of 80 mm. Hg. I feel that except in cases of uncontrollable haemorrhage, or in those rarer cases where conscientous resuscitative measures soon show their ineffectiveness, no operation should be attempted until the systolic blood pressure has been stabilized to at least 100 mm. for about 30 minutes. As the correction of the hypovolaemia and electrolyte imbalance in any event is so important in the management of abdominal lesions, there is no excuse for its omission in cases coming to urgent operation. Vigorous attempts should be made to remove the appalling mortality not only under the influence of the anaesthetic but also before and after the operation. That the deaths of patients suffering from abdominal lesions do not occur only during the operation is shown from the figures obtained from one large teaching hospital. Here the total deaths for 1 year from peritonitis, intestinal obstruction, and viscus rupture numbered 45, out of which only 4 patients actually died whilst under the influence of the anaesthetic, i.e. 9%. The proper control of fluid and electrolytes in these cases is a problem which needs the urgent attention of both anaesthetists and surgeons.

2. *Cardiac Arrest*

There were 132 cases of cardiac arrest on the operating table. This figure includes arrest after severe haemorrhage or intracardiac manipulations during heart operations. Cardiac massage was attempted in only 82 of these cases (Table VII). The only effective treatment of cardiac arrest on the table is immediate thoracotomy and effective cardiac massage by the surgeon, followed by defibrillation if considered necessary.[15, 19] At the same time the anaesthetist must institute effective measures of artificial respiration. The common practice, reflected in Table VII, of blind intracardiac injection of vasopressor drugs before thoracotomy and massage is criticized for 4 reasons:

TABLE VII. CARDIAC ARREST ON OPERATING TABLE. 132 CASES

Pre-op. Status		Treatment	
A	11	Massage	82
B	32	Intracardiac Adrenaline	39
C	53	Intracardiac Coramine	31
D	36	Intracardiac, other drugs	18

(i) Valuable time is lost before the actual massage is begun.

(ii) These drugs have no effect in the presence of anoxia, and anoxia is the usual cause of cardiac arrest.

(iii) The intracardiac injection of adrenaline, especially in patients suffering from ventricular fibrillation as result of chloroform, cyclopropane, ethyl chloride, or trilene, may make matters worse.

(iv) Permanent damage may be done to the heart muscle and aberrant blood vessels may be injured.[20]

The main treatment of an arrest is to get oxygen to the brain and other vital organs as soon as possible, and this can only be done efficiently by effective massage *via* the thoracic route. Only when this fails should one resort to drugs like adrenaline, in small doses, eg. 0·3 ml.

3. *Premedication*

Apart from 4 cases of patients over 65 years old and 2 cases of patients under 12 years old who were given large doses of Omnopon—20 mg. (gr. ⅓)—pre-operatively and eventually died in respiratory failure, I do not think that premedication played a major role in causing mortality. One frail youth (99 lb.) with a temperature of 102° was given 0·8 mg. (gr. 1/75) of atropine and eventually developed ether convulsions. Atropine seems to be the most popular drug for premedication; it was used alone in 75 cases, and in combination with pethidine in 41 cases. Next in preference comes a combination of Omnopon and scopolamine—15 cases.

4. *Intravenous Barbiturates*

Of the 182 patients who were given general anaesthesia, in 116 induction was with thiopentone sodium (pentothal)—Table VIII. Out of this number, 22 succumbed from

TABLE VIII. DEATHS ASSOCIATED WITH THIOPENTONE SODIUM. 22 CASES

		Stage of Death		*Status of Anaesthetist*	
Total no. induced	116	Induction	13	Specialist	4
Primary cardiac failure	22			Registrar	11
Massage attempted	13	Commencement of operation	8	G.P.	6
Pre-op. Status					
C, 17. D, 5		Later	1	Intern	1

primary cardiac failure developed either during induction or at the beginning of the operation. The pre-operative assessment of all these patients was category C or D, and here I question the choice of pentothal as an induction agent, especially when administered by an inexperienced anaesthetist. I think that the main factor responsible for these deaths must be the pentothal, notwithstanding that some of the patients were given 50-100 mg. of scoline in addition. The following are a few typical anonymous case histories:

1. Adult patient, aged 50. Acute intestinal obstruction, mitral stenosis, and congestive cardiac failure. Pulse 90, respiration 20, B.P. 110/60. Premedication: 0·6 mg. of atropine (gr. 1/100) and 50 mg. of pethidine. Induction with 2½% pentothal (dose not stated) and 50 mg. of scoline. Oxygen under pressure and oral endotracheal tube. Patient became pulseless immediately. Cardiac massage done through abdomen with no effect. P.M. report: No additional information.

2. Adult patient. Peritonitis. General condition 'very toxic and ill'. Temperature 99°, pulse 98, respiration 20. Premedication: 1·2 mg. of atropine (gr. 1/50). 'Thiopentone, 250 mg., mixed with 25 mg. of methedrine given intravenously. Immediately pulse and respiration ceased. Cardiac massage without avail.' P.M. report: Generalized peritonitis following a ruptured appendix.

3. Adult woman. Placenta praevia, general condition 'fair'. Temperature 96°, pulse 116, respiration 20, B.P. 110/80. 'Tired, mild shock, moderate anaemia.' Premedication: 0·6 mg. of atropine (gr. 1/100). Pentothal, 375 mg., followed by 50 mg. of scoline. Immediate circulatory and respiratory arrest. Coramine, 1 ampoule intravenously and 1 ampoule intracardially *statim*. Cardiac massage 5 minutes later; no effect. P.M. findings: mitral stenosis.

It has often been stated that there are no *absolute* contra-indications to the use of thiopentone, and that patients usually die from overdosage; this is certainly not a view one would like to adopt in a teaching hospital. The potential dangers of this drug have been described in numerous text-books on modern anaesthesia, and it would seem unnecessary to repeat them here, except to mention that the two common complications are depression of respiration, and depression of circulation; and any person using thiopentone should be aware of the contra-indications, and know how to treat these two complications should they arise. Even the simple procedure of using a 2½% solution as routine might go a long way towards diminishing these complications. There is an excellent book on this subject by Dundee.[21]

5. *Inhalation Anaesthesia*

Of the 182 patients who were given general anaesthesia, in 130 the anaesthesia was either induced or maintained with an inhalation anaesthetic (other than nitrous oxide). A further 26 cases were given an intravenous agent plus nitrous oxide and oxygen for induction and/or maintenance.

Of all the inhalation agents mentioned in these records the death rate from trichlorethylene (trilene) appears to be the highest, and this agent deserves further discussion.

(*a*) *Trichlorethylene*. There were 10 cases of primary cardiac arrest in which I think trilene played the major role. I am reasonably certain that this agent was responsible for at least 6 deaths on the table, and was the probable cause of 4 more (Table IX). The deaths all follow more or less the

TABLE IX. DEATHS ASSOCIATED WITH TRILENE. 10 CASES

Reasonably certain	6	*Pre-op. Status*	*Stage of Death*
Probable cause	4	A 3	Induction 4
Attempted massage	6	B 3 C 3	Maintenance 5
Adrenaline before or after	7	D 1	After 1

same pattern as those described by Edwards *et al.*[9] and Norris and Stuart.[22] The following are 3 abbreviated anonymous case histories to illustrate the points mentioned in Table IX:

1. Young woman with abscess of the breast. Toxic. Temperature 103·4°, pulse 130, B.P. 130/80. Premedication: 1·2 mg. of atropine (gr. 1/50) ½ hour pre-op. Induction: Trilene, gas and oxygen—Boyle's machine. 'Two minutes after incision the patient gave a sigh, and pulse and respiration disappeared. Resuscitation: Artificial respiration with Boyle's machine, intracardiac adrenaline, and intravenous coramine; no massage.' Autopsy revealed no abnormality.

2. Young woman with abscess of the breast. Temperature 100·1°, pulse rapid. 'Very, very apprehensive.' Premedication:

100 mg. of pethidine, 0·4 mg. of scopolamine (gr. 1/150). Induction: gas, oxygen and trilene. 'After 2 minutes respiration suddenly ceased, and pupils dilated. No pulse to be felt. Intratracheal tube passed and pure oxygen insufflated. Intracardiac adrenaline given to no effect. No cardiac massage done. No operation was done.' Autopsy: Nothing definitely abnormal found.

3. Young man with a superficial haematoma on the leg. Preoperative condition, N.A.D. 'Trilene by means of a Cyprane inhaler. Patient stopped breathing within 1 minute, and all measures of resuscitation were unsuccessful, including oxygen and coramine'. Autopsy: no abnormality.

If primary cardiac failure during trilene anaesthesia is really a common occurrence, then the encouraging results reported recently from the use of 'fluothane' might make this drug a valuable substitute in cases where a non-explosive mixture is required.[23, 24]

(*b*) *Other Agents for Inhalation Anaesthesia.* Apart from the 10 deaths associated with trilene anaesthesia there were 9 other deaths for which, I think, the inhalation agents were mainly responsible. These include nitrous oxide, ether, ethyl chloride, vinesthene, chloroform and cyclopropane.

One patient developed cardiac arrest for an internal version during chloroform anaesthesia. Another adult male patient died after induction with nitrous-oxide anaesthesia. Autopsy revealed a tuberculous pericarditis.

There was one case of convulsions in a youth. Temperature 102° and multiple injuries. Atropine, 0·8 mg. (gr. 1/75), was given as premedication, and anaesthesia was induced with ethyl chloride and open ether (no nitrous oxide was obtainable at the time). After induction the patient was maintained with oxygen (2½ l./min.) and CO_2 (¼ l./min.), and ether through a Boyle's machine. The convulsions were controlled with flaxedil, but the patient eventually developed a cardiac arrest which did not respond to massage and intracardiac adrenaline.

6. *Muscle Relaxants*

In Beecher and Todd's report,[14] already referred to, a shadow was cast over the relaxant drugs. According to their statistics, covering more than half a million anaesthetics, these authors found that the death rate was 6-fold higher in patients who were given 'curare drugs' during surgical operations. The combination of ether and a 'curare drug' seemed to be the worst, and here the death rate rose from 1 : 1100 with ether alone, to 1 : 62 with ether plus curare or a similar compound. These authors mentioned the possibility that the muscle relaxants might have an inherent toxicity of which we are not yet fully aware. This, however, is not a view which is shared by pharmacologists.[25]

Recently also there were further disturbing reports about deaths associated with relaxant drugs, especially in elderly patients with a fluid and electrolyte imbalance—patients suffering from what is called 'neostigmine-resistant curarization'.[26]

Bearing these facts in mind, I made a special study of all those deaths in which I thought the relaxant drugs might have played a role—especially in elderly patients admitted with acute intestinal obstruction (Table X). Out of the 182 patients who died whilst under the influence of a general anaesthetic, 130 received relaxant drugs (Table XI). Going carefully through the records I found 19 cases only in which I think the relaxant drugs were involved. Out of these 19,

TABLE X. DEATHS FROM ACUTE INTESTINAL OBSTRUCTION (54 CASES)

Age	*No.*	1 *Type of Relaxant*	2 *Types of Relaxant*	*Ether*
Under 60	36	17	12	20
Over 60	18	8	6	9
Total	54	25	18	29

Pre-op. Status: B, 5; C, 30; D, 19.

TABLE XI. PATIENTS RECEIVING MUSCLE RELAXANTS (130 CASES)

Depolarizing only	41
Competitive only	37
Both	52
	130
Competitive+Ether	42

there were 7 cases where I think the relaxants were only a contributory cause of death. There were 9 other cases in which I think the relaxants were mainly responsible for the death, and 3 more cases of collapse after the injection of large doses of prostigmine—2·5-5 mg.

Out of the 9 deaths which I am reasonably certain were due to relaxants, 5 presented the same picture as that described by Hunter in his article on 'neostigmine-resistant curarization'.[26] All these 5 cases were admitted seriously ill, and were operated on for acute intestinal obstruction. Respiration was 'controlled' in all of them; 2 had only one type of relaxant—either tubarine or flaxedil; the other 3 had scoline in addition to curare or flaxedil. Only 2 of the 5 were given ether. I may add that the pre-operative status of 4 of the patients was C, and of the remaining one D.

The most acceptable theory about the cause of these neostigmine-resistant deaths is that advanced by P. A. Foster of the Karl Bremer Hospital, Bellville.[27] In his opinion, in patients with low cellular-potassium values the competition blockers may have a central depressant action, which is not reversed by neostigmine.

The obvious lesson to be learned from these unfortunate deaths is to remember that with elderly debilitated patients, especially for abdominal operations, one *must* keep the patient breathing all the time, and obtain further relaxation if it is necessary with an inhalation agent.

The remaining 4 patients whose death I attribute to relaxants (not associated with neostigmine-resistant curarization) in my opinion all died from under-ventilation. There were no deaths from prolonged apnoea due to intraperitoneal injections of neomycin, as was described recently.[28]

The relaxant drugs have come to stay, and to my mind they are the biggest boon to patient, anaesthetist and surgeon since the introduction of ether as an anaesthetic agent in 1846. I can see nothing wrong with their universal use, provided one is aware of their potential dangers, knows how to intubate and, above all, remembers that the respiration must be supported in every case.

In one teaching hospital, over a period of 12 months 6,045 of 10,005 patients receiving a general anaesthetic were also given muscle relaxants. I have carefully studied the 16 'anaesthetic deaths' reported during the same period, and I found 1 death only which, in my opinion, can be attributed to the use of a relaxant drug. This was a patient who was very ill and unconscious from a head injury, with much secretion in his bronchial tree, for which a bronchoscopy was performed every day. On the occasion of his death he was given 20 mg. of scoline in the ward with no

other anaesthetic. His pharynx and trachea were sucked dry, when his pulse suddenly disappeared. Cardiac massage was of no avail—the cardiac massage drum first had to be fetched from the operating theatre. This patient would probably have died in any case, after a day or two, but the technique adopted is open to criticism. On this basis the rate of mortality solely due to relaxant drugs for this hospital, was 1 : 6,000, which is much lower than for the safest inhalation agent.

From the figures quoted in the present series one gets the impression that the relaxant drugs are actually much safer than thiopentone, which should be regarded as 'public enemy no. 1', at least as far as South African anaesthetists are concerned.

7. *Regional Analgesia*

As far as I am aware, regional analgesia is not a technique that is very popular with South African anaesthetists. That it is a mistaken idea to regard local analgesia as the only anaesthetic for a bad-risk patient is proved by the fact that 18 deaths in the series fell in this category, mostly associated with bad-risk patients (the pre-operative status was A in 1 case, B in 5, C in 4, and D in 8). Of these 18 deaths, 3 took place in the induction stage (1 with convulsions), 9 during the operation (in 5 of these cases both local and general anaesthesia were administered), 4 immediately afterwards, and 2 later. In 4 of the cases of death during operation cardiac massage was practised.

Time is short, but I should like to mention a few of the more interesting cases. One young woman with a colloid goitre developed ventricular fibrillation after the injection of only 2 minims of 1 : 1,000 adrenaline in saline solution into the operating site. Another death under local analgesia was associated with the use of the 'lytic cocktail' as premedication. One patient apparently developed an unrecognized coronary thrombosis before caudal analgesia. One collapsed after the local injection of 800 mg. of procaine during tonsillectomy. One death followed after bronchoscopy under local analgesia with 2% anethaine and 1 c.c. of 5% cocaine injected through the crico-thyroid membrane; in this case 'cocaine and anethaine sensitivity tests beforehand (had) proved nothing abnormal'. A warning should be given against the use of 2% amethocaine for topical analgesia, for a number of deaths during examinations of the bronchial tree in which this drug has been used have recently been described by Adriani and Campbell.[29] The maximum dose of amethocaine recommended for spraying of the larynx and bronchial tree is 40 mg. of ½% watery solution, i.e. a total dose of 8 ml. in the spray.

8. *Asphyxia*

Under this heading are included the deaths of patients who collapsed from acute obstruction of the airway caused by aspiration of vomitus or blood (12 cases), or from asphyxia caused by excessive secretions or respiratory depression following on the use of relaxant or other drugs (11 cases). There were then 23 deaths in this category. Of these, 2 took place in the induction stage, 4 during the operation, 6 within ½ hour after the operation, and 11 later. The pre-operation status was A in 5 cases, B in 3, C in 13 and D in 2. In my opinion, death in most of the cases was due not only to hypoxia but also to hypercapnia.

I think a number of these deaths were preventable, either by a better selection of technique, or by more adequate post-operative treatment. One must strongly condemn the practice of giving morphine or other depressant drugs post-operatively to a restless patient when the patient is not fully awake and is still suffering from a considerable amount of respiratory depression.

The 12 deaths due to aspiration of blood or stomach contents into the bronchial tree deserve further comment; 6 of them resulted from operation for acute intestinal obstruction, 2 from emergency gastrectomies, 3 from tonsil operations, and 1 from a hip operation. The pre-operation status was A in 4 cases, B in 1, C in 5, and D in 2. In some cases a stomach tube was not passed beforehand; in others it was deliberately omitted because of the danger of increasing the haemorrhage. In yet other cases the vomiting was unexpected, and there was no suction apparatus or other measures readily available to cope with the acute emergency.

I feel strongly that the dangers associated with regurgitation and vomiting, not only during induction, but also during recovery from anaesthesia, are a subject which should be brought to the notice of anaesthetists, surgeons, and all nursing staff in charge of surgical patients. The simple method of adopting the Sims or 'tonsil' position as routine post-operative procedure for all unconscious patients under the influence of a general anaesthetic will go a long way to prevent these tragic deaths. Morton and Wylie[5] have written an excellent article, in which the deaths in Great Britain from this cause are analysed.

RECOMMENDATIONS

I have given an analysis of what I think are the more obvious causes of death in the 200 cases in this series. I should now like to submit a few suggestions which might serve as a basis for discussion with a view to improving the situation. They are not necessarily set out in order of importance:

1. *Recovery Rooms*

Anaesthetic recovery rooms should be provided in all future hospitals. In these rooms bad-risk patients can be nursed by experienced nursing sisters, under the expert eye of the anaesthetists and surgeons. These sisters' experience in anaesthetic problems should also be available to assist the anaesthetists in the theatre in special cases.

All hospitals should also keep proper resuscitative equipment (cardiac massage drums, suction apparatus, etc.) immediately available, not only in the operating theatres, but also in the X-ray rooms and other localities where anaesthetics are frequently administered.

2. *Departments of Anaesthesia*

Independent departments of anaesthesia should be established in all medical schools with status equal to that of other departments. In these departments students and post-graduates should receive lectures on the principles of modern anaesthesia, and research should be encouraged and subsidized.

3. *Compulsory Training of Interns*

The modern curriculum is far too full to teach a student how to administer an anaesthetic properly during his student years. I should like to recommend, before registration as a medical practitioner, at least 1 month full-time compulsory training in anaesthesia for the intern or, where this is not practical, the administration under supervision of at least 100 anaesthetics. In our own hospital this system has worked very successfully for the last 8 years.

Whatever may be the ideal, for a long time to come the bulk of the anaesthetics in this country will be given by general practitioners. In the Transvaal public hospitals, for instance, there are still 33 hospitals with no specialist anaesthetist on the staff,[30] and in the mine hospitals for Natives this is the rule rather than the exception.

4. *Anaesthetic Study Committees*

In all the large hospitals special committees should be appointed to investigate all anaesthetic deaths immediately after they have occurred, whilst the memory of the case is still fresh, and make recommendations for more adequate preventative measures in future.

5. *Compulsory Notification of all Deaths under Anaesthesia*

All such deaths should be notifiable to the Union Department of Health or some other responsible authority. This would ensure that *all* deaths were reported, and would be of invaluable help in research and for statistical purposes.

6. *Compulsory Instruction in Methods of Cardiac Resuscitation.*

With the ever-increasing number of investigations that are being carried out under anaesthesia on more or less moribund patients, not only by surgeons but also by physicians, radiologists, etc., there is an urgent need for instruction in the most effective methods of cardiac resuscitation. This might well form the subject for joint discussion by the S.A. Society of Anaesthetists and the S.A. Society of Surgeons.

Furthermore, I should like to recommend that effective 'notices' of how to act in case of sudden cardiac arrest should be displayed prominently in all operating localities.

7. *Qualified Assistants for Anaesthetists*

With some of the modern methods and modern techniques it is essential that the anaesthetist should have the help of a qualified assistant at major operations, especially neurosurgical, thoracic and abdominal operations. Moreover, the practice of making the anaesthetist without the aid of an assistant responsible for the actual administration of blood during operations should be condemned.

8. *Refresher Courses in Anaesthesia*

Refresher courses for general practitioners, nurses and other persons interested in modern methods of anaesthesia should be offered by all medical schools. With this recommendation should be coupled more effective training of the nursing staff in the care of the unconscious patient.

SUMMARY AND CONCLUSION

A preliminary report has been presented on the main facts of 200 deaths associated with anaesthesia and surgery. Interpretations have been personal, as have been the views expressed throughout—all may not agree with them.

Resuscitation still does not receive the attention it deserves. The treatment of cardiac arrest remains, in general, inadequate. Thiopentone sodium seems to be the most dangerous anaesthetic agent in this country—much more dangerous than muscle relaxants, provided that in elderly, debilitated persons respiration is assisted but not controlled. The death rate with Trilene seems to be unduly high. There are peculiar dangers in the use of some agents for regional and topical analgesia. Adequate pre- and post-operative care is essential in all cases to avoid the dangers of asphyxia.

A number of recommendations have been made apropos training and organization to improve the existing position.

Finally I should like to make an urgent appeal to all hospitals and private anaesthetists, not already cooperating in this investigation, to supply voluntary, anonymous information of all deaths associated with anaesthesia and surgery.

In conclusion, I quote the well-known anaesthetists' motto: '*There are no safe drugs, only safe anaesthetists.*'

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