Anaesthetic-Associated Mortality

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SUMMARY

The most important measure of the safety of anaesthesia for the patient is the incidence of anaesthetic-contributory death. Evidence is presented from an ongoing survey of anaesthetic-associated mortality at Groote Schuur Hospital, that the incidence of anaesthetic-contributory death has decreased from 0,33 to 0,22 per 1 000 anaesthetics. Reasons for this improvement in the safety of anaesthesia and some of the clinical lessons that emerge from the survey are discussed. The incidence of cardiac arrest in the operating theatre (0,63‰ anaesthetics) and the results of resuscitation (19% survival) are presented.

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Anaesthesia is a highly important adjunct to the care of the patient. Anaesthesia is not therapy which corrects a deformity, which restores health, or stays death; it simply makes possible the acts which accomplish these things. So above all it must be safe. In no field of medical practice does the precept 'to do no harm' have more cogency than in anaesthesia. Whatever advances are claimed for anaesthesia, they can only be regarded as real advances if they result in increased safety for the patient. However, to measure this safety is difficult. Though one may postulate the most fundamental index of the safety of anaesthesia to be the incidence with which factors related to the administration of an anaesthetic cause or are contributory to a patient's death, such a postulate immediately poses the problem of apportioning blame. Patients who die under anaesthesia do so more often from the disease or the surgery undertaken to cure it, than from the anaesthesia. To measure this incidence we must separate as best we can, anaesthetic-contributory deaths from their parent population of anaesthetic-associated mortality.[2]

At Groote Schuur Hospital I have conducted a contemporary survey of mortality associated with anaesthesia since 1956. Data on the anaesthetic-associated mortality of 15 years in over 300 000 anaesthetics are now available. Findings during the first 9 years of this survey were reported in 1968.[1] The data of the last 6 years (1967 - 1972 inclusive) are reported here and compared, where relevant, with the former study.

DEFINITIONS AND METHOD

For the purposes of this article anaesthetic-associated death is defined as a death occurring during or within 24 hours of anaesthesia, or a failure to regain consciousness after anaesthesia in a patient conscious before anaesthesia. The choice of a period of 24 hours after anaesthesia is arbitrary. It embraces a period adequate to permit identification of anaesthetic-contributory death, without becoming unmanageable. Extension of this study to a surveillance of the whole postoperative period, although desirable in some respects, would add considerably to its difficulties. It is acknowledged that, in these circumstances, a small number of deaths to which anaesthesia was a major contributory factor, such as some late deaths from aspiration pneumonia, might have been missed.

For each case of anaesthetic-associated mortality, the clinical records and an account of the conduct of the anaesthesia were obtained from the anaesthetist concerned. This information and the autopsy report were examined from 3 points of view:

1. Was the administration of the anaesthetic the cause of, or a major contributory factor to, the patient's death? Or was the death primarily due to the patient's disease or the surgical procedure being undertaken?

2. If it was considered to be an anaesthetic-contributory death, what was the precise clinical cause—what departure was there from accepted clinical practice?

3. Was the death preventable? Was there a clinical lesson to be learnt?

With regard to the first question, besides the two groups of cases implied: (*i*) anaesthetic-contributory deaths, and (*ii*) death due to other causes, there emerged a need for a third group: deaths associated with the surgery of desperation, operations on the moribund, for which, though an anaesthetic aetiology may not have seemed directly responsible, it could often not be entirely excluded; circumstances in which anaesthesia might be regarded as being necessarily, but unavoidably, contributory, were classified as 'inevitable death'.

GENERAL INCIDENCE

This 6-year survey covers about 141 000 anaesthetics. This is a crude total and includes all anaesthetics, from simple casualty procedures to open-heart surgery. An obvious refinement of a study such as this would be an analysis of mortality associated with specific operations or anaesthetic procedures. This has not been done. For comparison, relevant figures from the previous survey are included in parentheses in Table I.

This survey yielded an anaesthetic-associated mortality of 253 cases, a rate of 1,8/1 000 anaesthetics. Anaesthesia was considered to be the sole or major contributory factor in 12% (31 deaths) of this anaesthetic-associated mortality—an anaesthetic-contributory death-rate of 0,22/1 000 anaesthetics.

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TABLE I. GROOTE SCHUUR HOSPITAL: ANAESTHETIC-ASSOCIATED MORTALITY IN 140 653 ANAESTHETICS
(1967 - 1972 (incl.), 1956 - 1966 in parenthesis)

	No. of deaths	Deaths/ 10^3 anaesth.
Total operative mortality	1 315 (2 026)	9,35 (11,38)
Anaesthetic-associated mortality	253 (414)	1,80 (2,33)
Anaesthetic-contributory deaths	31 (58)	0,22 (0,33)
Other causes	147 (261)	1,04 (1,47)
Inevitable	75 (95)	0,53 (0,53)

$\frac{ACD}{AAD} \times 100 = 12\%$ (14%)

$\frac{ACD}{TOM} \times 100 = 2{,}3\%$ (2,8%)

AAD = Anaesthetic-associated death
ACD = Anaesthetic-contributory death
TOM = Total operative mortality

Looked at against the background of the total care of the surgical patient, anaesthesia and its mismanagement can be said to have been responsible, by means possibly preventable, for 2,3% of the total surgical mortality.

In the sense that the rate of anaesthetic-contributory death has decreased over the 6 years from the previous 9-years' report (0,22/1 000 from 0,33/1 000) anaesthesia may be said to have become a little safer for our patients. This increased safety may be a reflection of a fundamental, though gradual and undramatic, change that has occurred in anaesthetic practice during the last decade. This change follows the gradual acceptance of the routine use in all clinical anaesthesia of the elementary monitoring of vital signs, over and above the old-fashioned recording of systolic blood pressure, such as ECG, central venous pressure and blood gas monitoring.

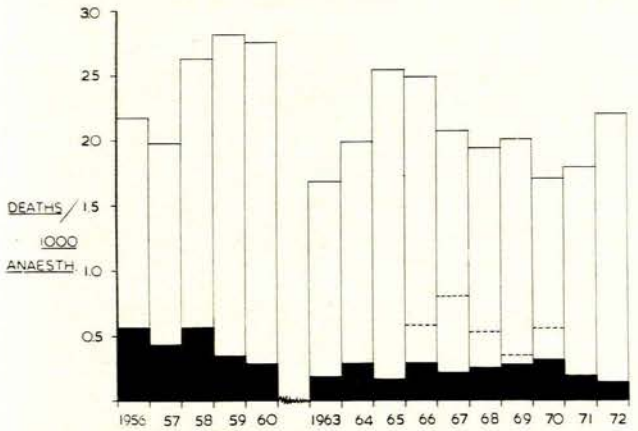

Fig. 1. Anaesthetic-associated mortality and anaesthetic-contributory death at Groote Schuur Hospital 1956 - 1972. (Open columns = anaesthetic-associated deaths; black columns = anaesthetic-contributory deaths.)

When anaesthetic-associated and contributory death rates are graphically represented on an annual basis (Fig. 1), although the initial downward trend in the latter is obvious, it appears that in this institution an anaesthetic-contributory death rate of 0,2/1 000 anaesthetics, regrettably constitutes an irreducible minimum. This is depressing when one considers that anaesthetic-contributory death is essentially preventable. Perhaps, in mitigation, it may be said that this is a teaching institution with a large, constantly changing staff, which results in a dilution of experience. It seems as though every trainee is educated through the same mistakes. Every trainee must be taught to acquire the right clinical reflexes. Constant and adequate supervision of trainees by experienced, specialist staff is only part of the answer, for the most enduring lessons for the trainee come only from the final acceptance of total clinical responsibility.

MECHANISMS OF ANAESTHETIC-CONTRIBUTORY DEATH

The causes and mechanisms of anaesthetic-contributory death were examined in terms of the following simple concepts (Fig. 2). The basic requirement for life is perfusion of tissue with oxygenated blood. Failure to perfuse tissue (and more particularly the brain) with an adequate

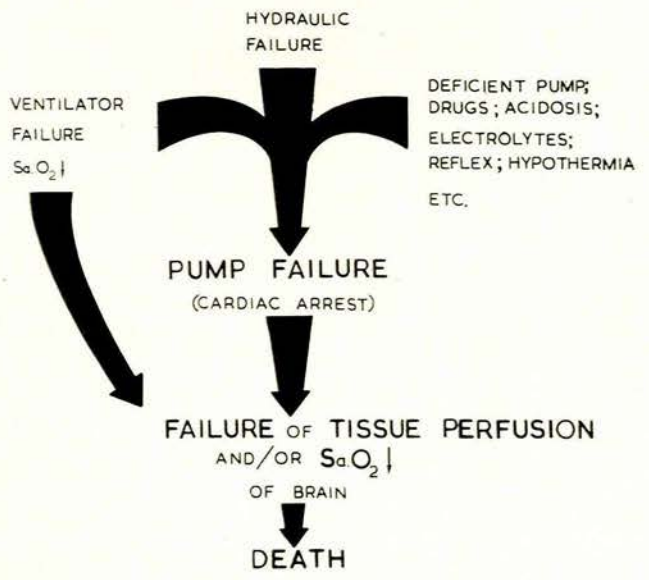

Fig. 2. Mechanisms of anaesthetic-contributory death.

supply of oxygenated blood, is the basic cause of death. In broad terms, this occurs when there is a failure of either the ventilator or the pump or both. While the former may directly cause cerebral death, the final common pathway is more usually pump failure and cardiac arrest. Cardiac arrest, although often caused by anoxia, has a wider aetiology, the most prominent factor of which, in a surgical sense, is a failure of the hydraulic system—hypovolaemia, compounded by the vasodilation of anaesthesia and its sequelae of concomitant poor circulatory homeostasis, acidosis, and electrolyte changes. It must be realised that the greater proportion of anaesthetic-contributory deaths present as cardiac arrest during operation. In some cases the reasons may be apparent before the event occurs, but more often they may escape notice until the dramatic arrest of the heart. At this stage it may be difficult to elucidate the cause in retrospect.

Analysed in these terms, the 31 deaths in this study are presented in Table II. This study reveals, like its predecessor, the same two clinical situations most frequently causally associated with anaesthetic-contributory death. These 2 situations between them are responsible for just under one-half of all anaesthetic-contributory deaths, or for the same number of anaesthetic-contributory deaths as all the other listed causes put together. These two situations are complications of intubation and hypovolaemia.

TABLE II. GROOTE SCHUUR HOSPITAL: CAUSES OF ANAESTHETIC-CONTRIBUTORY DEATHS IN 140 653 ANAESTHETICS (1967 - 1972 (incl.), 1956 - 1966 in parenthesis)

	No. of deaths	Deaths/ 10^5 anaesth.
Ventilator failure		
$SaO_2\downarrow$		
F_IO_2 technical mishap	**0 (1)**	**0 (0,5)**
Vom. regurg. inhal.	**2 (3)**	**1,4 (1,7)**
Sec. bronchial obstruct.	**2 (2)**	**1,4 (1,1)**
Bronchospasm	**1 (0)**	**0,7 (0)**
Comp. intubation	**7 (8)**	**4,9 (4,4)**
Relax. related	**3 (9)**	**2,1 (5,1)**
Inadequate postop. care	**3 (3)**	**2,1 (1,6)**
Pump failure		
(circulatory haemostasis)		
Hypovolaemia	**8 (12)**	**5,7 (6,7)**
Hypervolaemia—overhyd.	**2 (1)**	**1,4 (0,5)**
urovert	**0 (2)**	**0 (1)**
Cardiac arrest—direct causes	**2 (10)**	**1,4 (5,6)**
uncertain	**1 (6)**	**0,7 (3,3)**
Miscellaneous		
Incompatible blood	**0 (1)**	**0 (0,5)**
	31 (58)	**22 (33)**

F_IO_2 = Inspired oxygen concentration.
Cardiac arrest—direct causes = Cardiac arrest due to factors listed at upper right of Fig. 2.
Overhyd. = Overhydration.

Complications of Intubation

Endotracheal intubation is indeed a necessary technique for the anaesthetist. Complications are simple and mechanical, but lethal, unless diagnosed and corrected immediately. The reason for failure is often a false sense of security engendered by the very presence of the endotracheal tube, together with, as often as not, a lack of experience. A brief look at these 7 cases is instructive, as one lesson in particular emerges strongly. Over half of these deaths followed the technical failure of intubation in the presence of anatomical difficulties. All could have been easily anticipated before anaesthesia by adequate clinical examination, yet in all instances succinylcholine was given before the anaesthetist had assured himself that he could intubate or ventilate the patient. The anatomical difficulties were caused by carcinoma of the larynx and oesophagus, cervical tuberculous spondylitis, and torticollis.

One case was an example of that ever-lurking trap—the overinflated, herniated cuff—a lesson that needs constant repetition, as there often is, as in this case, a period of many minutes between the inflation and the herniation of the cuff, with subsequent respiratory obstruction.

One death followed kinking of the endotracheal tube when the patient was turned prone. The last, less culpable than the others, followed the obstruction of the right side of a Carlen's tube by distortion caused by an aneurysm of the ascending aorta.

Inadequate Postoperative Supervision

Disappointing is the appearance in this study of 3 cases due entirely to inadequate postoperative supervision. One death was caused by the direct attachment of a line from an oxygen cylinder to an endotracheal tube left *in situ* by the anaesthetist for postoperative IPPV. A second was due to the booby-trap of the endotracheal tube catheter-mount of a nasal endotracheal tube becoming detached unobserved inside the nose in a patient requiring postoperative IPPV.

I should like to draw particular attention to the third case, for it is an example of a condition to which the now widespread use of respiratory-depressant, narcotic, analgesic drugs, such as Fentanyl, exposes patients—a condition for which the name 'Ondine's curse' is appropriate. As with the nymph, the patient, while recovering from anaesthesia, though apparently awake, seems to have forgotten how to breathe, unless he is stimulated. This particular patient, having initially regained consciousness after uncomplicated anaesthesia for a herniorrhaphy, relapsed into unconsciousness and apnoea while in the care of a nurse in a busy theatre corridor. This event was not reacted to until cardiac arrest with some anoxic jactitation occurred. Although cardiac massage and resuscitation were successful, the brain damage sustained was irreparable.

Vomiting and Regurgitation

More encouraging is the very low incidence of death due to vomiting, regurgitation, and inhalation. Such deaths often feature in studies of anaesthetic-associated mortality.

Relaxant-Related Deaths

Noteworthy too, is the great reduction in relaxant-related deaths compared with the previous study—deaths due to mismanagement of prolonged curarisation. There is really little excuse for such deaths today.

Hypovolaemia

Under this heading are included those cases in whom a frank failure of circulatory homeostasis has followed induction of anaesthesia, a failure that appears to have resulted from inadequate venous return. These circumstances proved to be the most common, single cause of anaesthetic-contributory death. Once circulatory failure had ensued, metabolic acidosis and concomitant electrolyte disturbance may have been important in the causation of the cardiac arrest. But I have chosen to call attention in my classification to the initial hypovolaemia, as this situation is eminently correctable. Some workers stress this by classifying these deaths as due to 'inadequate preparation of the patient'.[2]

These cases, who also feature prominently in other surveys,[1,3,4] present most commonly, as did half of the 8 listed in Table II, as postinduction hypotension proceeding to cardiac arrest. The precipitating factor is often the exhibition of thiopentone to the sick, old, and arteriosclerotic who have evidence of ischaemic heart disease, or the administration of thiopentone to the shocked. A typical example is the old man with diabetic gangrene, presenting for amputation of a leg. Whether it be the vasodilatation, or the direct depressant effect of thiopentone on the myocardium, the situation is readily preventable by judicious hydration to normal CVP levels before induction of anaesthesia and avoidance of thiopentone.

Two of these deaths followed sudden blood loss in the presence of vasodilatation, the result of sympathetic paralysis: 1 from an epidural anaesthetic, and 1 from the use of a uterine antispasmodic, isoxuprine.

I wish to stress that in none of these 8 patients was the central venous pressure monitored, and in 4, the failure of circulatory homeostasis was precipitated by thiopentone.

Hypervolaemia

The reverse situation is hypervolaemia from overhydration. This usually results in pulmonary oedema when spontaneous respiration commences again at the conclusion of the anaesthesia. Two such cases are recorded here of a situation essentially avoidable by the substitution of fluid replacement guesswork, with CVP monitoring.

Cardiac Arrest in the Operating Theatre

This subject is exhaustively covered in the literature. I intend to summarise only the causes and the outcome of treatment in the cases occurring in Groote Schuur Hospital during the period of this survey. I have excluded from consideration all cardiac surgery in which so often cardiac arrest and its reversal are almost part of the operative technique. I have, however, included 5 cases of cardiac trauma from stab and gunshot wounds. The causes and results of treatment are set out in Table III.

TABLE III. GROOTE SCHUUR HOSPITAL 1967 - 1972: OPERATING ROOM CARDIAC ARRESTS IN 140 653 ANAESTHETICS

Causes				Resuscitation		
				Failed	Initial success	Ultimate success
Anaesthetic $SaO_2\downarrow$	Bronch. obstruc. spasm	2				
	Comp. intubation	8				
	Inhal. vom. regurg.	2				
	Underventilation	3	44%			
$Q\downarrow$	Hypovolaemia	13				
	Hypervolaemia	2				
	Cardiac arrest—direct causes	7				
	uncertain	2	39 (0,27°/₀₀)	21 (54%)	18 (46%)	12 (31%)
Other						
Haemorrhage + massive transf.		33				
Toxaemia refract. shock		8				
Cardiac trauma + tamponade		5	56%			
Ischaemic heart disease		2				
Diffuse neuronal injury		1				
Embolus (pulmonary)		1	50			
	Total		89 (0,63°/₀₀)	60 (68%)	29 (32%)	17 (19%)

$SaO_2\downarrow$ = Reduced arterial oxygen saturation.
$Q\downarrow$ = Decreased circulation/perfusion.

During the period under study, 89 patients suffered cardiac arrest during anaesthesia and operation, an incidence of 0,63/1 000 operations. Various anaesthetic problems were the sole or major contributory factor in the causation of 44% of all cardiac arrests.

As in the causation of anaesthetic-contributory death, the two principal faults were failure to maintain normovolaemia and complications of endotracheal intubation.

Worthy of comment in that group of anaesthetic-contributory cardiac arrests thought to be drug-induced, was 1 case in whom the cardiac arrest appeared to follow the administration of halothane (with adequate ventilation) to a parkinsonian patient being treated with L-dopa. Although this combination has been suggested as a potential cause of arrest because of th emobilisation of tissue catecholamine stores by L-dopa,[5] we are not aware of its having been so recorded.

Other drug-induced arrests followed the administration of succinylcholine, halothane in the presence of underventilation, and the hypotensive drug, trimetaphan, in the presence of a recent myocardial infarct—all well-known anaesthetic hazards.

The commonest single cause of cardiac arrest in the operating room, and responsible for 30% of cardiac arrests in this study, was gross haemorrhage with the deleterious effects of the concomitant massive transfusion. While many of the problems which follow the latter have been solved by monitoring of acid-base and electrolyte status, and the warming of transfused blood,[6] the basic problem is surgical and circumstantial.

With the present-day accepted use of vital-function monitoring in clinical anaesthesia, and the training of anaesthetists in prompt and informed resuscitative procedures, one could hope for a reasonable salvage from cardiac arrest occurring during surgery right under the eye of the anaesthetist, but the commonest cause—uncontrollable haemorrhage—precludes this.

In Table III the results of resuscitation from cardiac arrest are expressed as: 'failed'—death in operating theatre; 'initially successful'—patient survives to return to ward; 'ultimately successful'—patient survives to be discharged from hospital.

It can be seen that about one-third of all patients suffering from cardiac arrest survived to leave the operating theatre and one-fifth eventually left hospital. The prognosis for those which were the result of anaesthetic mismanagement (0,27/1 000) was slightly better. While about one-half survived to return to the ward, resuscitation was wholly successful for about one-third in that the patient was ultimately discharged alive from the hospital.

CONCLUSION

Whatever advances we claim for our speciality, anaesthesia, it must be safe for the patient. Surveys and studies such as this are somewhat pedestrian and tedious in the extreme to conduct, but if consistently and conscientiously carried out, they provide not only the clinical lessons by which we can improve our practice, but also the only means of measuring and evaluating the safety of anaesthesia.

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