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CARDIAC ARREST ASSOCIATED WITH SURGICAL PROCEDURES: A REVIEW OF 300 CASES*

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This paper is based on 300 cases of cardiac arrest associated with surgical operations occurring in various South African public hospitals. This report deals with fatal cases only, and they were all included in the returns of deaths associated with anaesthesia (as defined in terms of section 86 of the Medical, Dental and Pharmacy Act of 1928) submitted to the CSIR Anaesthetic Deaths Research Unit during the last 4 years (1955–1959).

Definition and Pathology

For the purpose of this article, we can define cardiac arrest as a sudden and complete cessation of cardiac function during the administration of an anaesthetic or the performance of a surgical operation, not due to irreversible shock or uncontrollable haemorrhage. By cessation of cardiac function we mean the inability of the left ventricle to pump the blood to vital organs like the heart muscle, brain, liver and kidneys.

Under this definition of cardiac arrest we include 3 specific pathological conditions, viz. (1) cardiac standstill or arrest in asystole, (2) cardiac arrest in ventricular fibrillation, and (3) feeble contractions of the myocardium not strong enough to pump the blood to the vital organs. In Table I 300 cases are classified accordingly.

TABLE I. TYPE OF ARREST (300 CASES)

	No. of Cases	*% of Total*
Cardiac standstill	150	50
Ventricular fibrillation	31	10
Unknown	119	40

There are 3 points which help to differentiate cardiac arrest from other causes of death associated with surgical operations. After all, every death is eventually due to a cessation of cardiac function. These points are the following: (1) The condition generally occurs suddenly and unexpectedly; (2) the actual cause of arrest is often difficult to determine; and (3) the condition is often avoidable and with prompt and adequate treatment usually reversible.

Incidence

In the absence of detailed statistics of the number of anaesthetics administered, we are not yet in a position to determine the exact incidence of this condition in South African public hospitals. We hope to be able to do this in a subsequent report. There is no doubt in our own minds, however, that this condition is on the increase, and this is in accordance with opinions expressed by overseas authors.[1-4] From our records we think 3 factors are mainly responsible for the increase during the last decade:

1. Owing to improved anaesthetic and surgical techniques more operations are being performed on bad-risk and even moribund patients, and this has led to more cases of cardiac arrest on the operating table.

2. The fact that cardiac surgery is now practised in a number of centres has also swelled the numbers; in the present series 35 cases fell in this category.

3. Modern anaesthetic techniques have been blamed by some surgeons, but we feel that lack of training and ignorance on the part of the anaesthetist responsible have more influence than the actual methods adopted.

More than one-third of our cases occurred during abdominal operations (105), with thoracic (35) and orthopaedic cases (29) taking second and third places (Table II).

TABLE II. SITE OF OPERATION[5] (300 CASES)

	No. of Cases	*% of Total*
ENT	7	2·3
Obstetrics	15	5·0
Genito-urinary	19	6·3
Stomach and duodenum	16	5·3
Small and large bowel	52	17·3
Other abdominal	32	10·6
Hernia (all types)	5	1·6
Thoracic	35	11·6
Endoscopy	14	4·6
Thyroid and other neck operations	5	1·6
Dental and oral	5	1·6
Orthopaedic	29	9·6
Plastic and superficial	5	1·6
Neurosurgical	15	5·0
Gynaecological	18	6·0
Other	28	9·3

Cardiac arrest is supposed to be commoner in children under 10 years and in adults over 60 years, but from the figures reflected in Table III it is obvious that no age is really immune to this catastrophe.[6]

AETIOLOGY

Hypoxia of the myocardium is usually considered as the chief cause of cardiac arrest.[7]

From this investigation it would appear, however, that excepting when the heart was pathological before operation, or in cases of asphyxia, or massive haemorrhage, *hypoxia alone* very seldom led to *a sudden cessation of cardiac function.* There were usually other factors responsible for precipitating the arrest and this will be considered in more detail presently.

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TABLE III. AGE DISTRIBUTION (300 CASES)

Decade	No. of Cases	% of Total
1	50	16·6
2	19	6·3
3	54	18·0
4	54	18·0
5	46	15·3
6	41	13·6
7	24	8·0
8	10	3·3
9	2	0·6

TABLE IV. SOME PRECIPITATING FACTORS

	No. of Cases
Pre-operative status of patients	88
Hypercapnia	70
Drug intoxication ..	61
Stimulation of peripheral autonomic nerves:	
(*a*) Vagus ..	15
(*b*) Sympathetic	3
Human factors ..	18

We also believe that when an arrest eventually occurs it is a summation of subliminal stimuli on the myocardium. These stimuli can be cumulative and additive, and for this reason short operations are probably less dangerous than prolonged procedures, especially on bad-risk patients.

In our analysis of these records we are of opinion that the factors shown in Table IV played a role in precipitating the arrest. In some cases more than one factor was considered responsible, and in others it was often difficult, and even impossible, to determine the exact cause.

1. *Pre-operative Status of Patient*

An assessment was made of the physical status of each patient before operation and all patients were classified into 4 categories A, B, C, and D as described in a previous article.[8] From Table V it will be seen that the 300 deaths include 191 poor-risk and moribund patients. Although in the moribund patient an arrest can take place at any time even without an operation or the administration of an anaesthetic, one must in some cases criticize the wisdom of operating when the prognosis is obviously hopeless from the start. We consider that in 88 cases in the list the pre-operative status of the patient was mainly responsible for the cardiac arrest. A number of these patients were suffering from thoracic diseases, and others were mostly bad-risk abdominal cases suffering from hypovolaemia and electrolyte disturbances which should have been corrected before operation.

2. *Hypercapnia*

Hypercapnia, irrespective of the cause of the accumulation of the carbon dioxide, is considered an important factor in the aetiology of cardiac arrest by most authors.[3,4,9-16] One should, however, remember that when hypercapnia occurs during the course of an anaesthetic it is usually accompanied by a certain degree of hypoxia. In this series we found 70 cases where we were of opinion that the hypercapnia (with an associated hypoxia) played a role in precipitating the arrest. The majority of these patients were either senile or suffering from chronic pulmonary or cardiac disease. We also have a clinical impression that malnourished patients who were not adequately prepared pre-operatively were more susceptible to the harmful effects of hypercapnia. Other factors responsible for this condition were inadequate ventilation of the patient during anaesthesia, especially when relaxant drugs were administered, too deep anaesthesia, and anaesthetic techniques which did not adequately remove carbon dioxide from the anaesthetic mixtures.

Hypercapnia appears to be particularly dangerous when it is accompanied by direct or reflex vagal stimulation, especially during operations in the neck and thorax. Young *et al.*[17] showed, by experiments on dogs, that the inhalation of 20% carbon dioxide and 80% oxygen until the pH of the blood is reduced may greatly augment the effect of vagal stimulation on the heart. As a contrast, it was found that a hypoxia produced by the inhalation of 10% oxygen, even when it was accompanied by hypercapnia and a low blood pH, reduced the effect of vagal stimulation on the myocardium. Stewart also found that cardiac arrest occurred very rapidly in normal dogs after hypercapnia and vagal stimulation in the thorax. In dogs suffering from chronic hypoxia before operation, the production of a hypercapnia did not augment this reflex.[15]

Another important cause of cardiac arrest associated with the accumulation of carbon dioxide is the so-called *post-hypercapniac ventricular fibrillation*. Brown and Miller have demonstrated on dogs that the danger period in association with hypercapnia follows immediately after the cessation of inhalation of high concentrations of carbon dioxide. When these dogs again inhale atmospheric air, ventricular fibrillation rapidly sets in.[13]

Sealy *et al.*[14] also investigated this condition, and came to the conclusion that hypercapnia produced in experimental animals was always accompanied by hyperkalaemia, and that the cessation of inhalation of carbon dioxide was immediately followed by a further increase of the plasma potassium. During this period ventricular fibrillation occurred very rapidly, apparently owing to the increased serum-potassium values. This may be one explanation for a sudden cardiac arrest which is sometimes reported at the end of a long and successful operation.

3. *Drug Intoxication*

An absolute or relative overdose of a drug used by the anaesthetist or surgeon during the course of an operation, or sensitivity to it, can easily precipitate a cardiac arrest on the operating table. In this category we include local analgesics, muscle relaxants, and drugs like prostigmine, adrenaline, diodine and pitocin. Polypharmacy, or the use of buckshot techniques when one drug would have served the purpose just as well, should also be mentioned in this connection.

In this series we found 61 cases where we think that the drug *per se* played a role in the aetiology of the arrest. Under this category we include 51 cases occuring during induction of anaesthesia.

As mentioned in a previous report,[8] the indiscriminate use of thiopentone (pentothal) in bad-risk patients is still mainly responsible for a sudden cardiac arrest during the stage of induction. We must again warn against the use of concentrated solutions or the rapid injection of this drug in elderly or bad-risk patients and would strongly advise the occasional anaesthetist to make use of alternate techniques when confronted with bad-risk cases. Thiopentone was the drug responsible for most of the 51 cases of arrest reported during induction.

TABLE V. PRE-OPERATIVE STATUS OF PATIENT (300 CASES)

	No. of Cases	% of Total
A (Good) ..	22	7·3
B (Fair) ..	87	29·0
C (Poor) ..	125	41·6
D (Moribund)	66	22·0

TABLE VI. PREMEDICATION

	No. of Cases	% of Total
Atropine ..	257	85·6
Scopolamine	33	11·0
Morphine ..	11	3·6
Pethidine ..	108	36·0
Other drugs	20	6·6

4. *Stimulation of the Peripheral Autonomic Nerves*

Cardiac arrest caused by direct or reflex stimulation of certain nerves associated with the autonomic nervous system has been reported frequently:[18]

(*a*) *Cardiac arrest from excessive stimulation of vagus nerves.* Vagal stimulation, associated with light anaesthesia and inadequate premedication, is often mentioned as a cause of cardiac arrest. We are of the opinion, however, that a fit patient, who has received an optimum dosage of atropine pre-operatively, very seldom develops a vagal inhibition during the anaesthetic, especially when muscle relaxants are used in conjunction with light anaesthesia. In this series we found 15 cases where we believe stimulation of the vagus played a part in the arrest. The majority of these cases occurred in poor-risk patients during bronchoscopies or in bad-risk cases during abdominal operations. Most of them received atropine beforehand (Table VI), but it might well be that by the time the arrest occurred the effect of the atropine had already worn off.

Sloan[19] believes that hypoxia plays an important role in the stimulation of the vagal reflex, but, from the work of Young *et al.*[7] on dogs, it would appear that carbon dioxide retention and not hypoxia is mainly responsible for augmenting this reflex during surgical operations.

(*b*) *Cardiac arrest from excessive stimulation of the sympathetic nervous system.* The usual causes of stimulation of the sympathetic nervous system during operations under general anaesthesia are oxygen lack, asphyxia, direct stimulation of splanchnic nerves, or chemical action of adrenergic substances, especially adrenaline.[7] In this respect the use of adrenaline in local analgesic mixtures in conjunction with trichlorethylene, halothane, cyclopropane or chloroform can easily cause ventricular fibrillation. We came across 3 cases where the indiscriminate use of adrenaline precipitated such an attack.

5. *Human Factors*

The training and judgment of the anaesthetist and surgeon and the choice of anaesthetic technique may play an important part in the final result. One cannot help feeling that some of these cases would have fared better if they had been operated on by the 'chiefs' instead of being left to junior personnel. We found 18 cases where we think that better judgment on the part of the anaesthetist might have avoided an arrest. The status of the medical personnel dealing with these 300 cases is summarized in Table VII.

In spite of all the factors mentioned above, cases still occur where it is impossible to determine the exact cause of the arrest. This may be because the data are inadequate, but, even with adequate details, we still came across 26 cases which did not fit into any of the above categories.

DIAGNOSIS AND TREATMENT

With the exception of operations in the thorax or the continuous use of an electrocardiogram, the diagnosis is usually established by the anaesthetist, and he must immediately ask the surgeon to begin the necessary treatment at once.

No time must be wasted in looking for a stethoscope in order to ascertain whether the heart is still beating. When a patient suddenly stops breathing and develops an ashen-grey colour, and the carotid artery cannot be palpated, one must assume that the patient is suffering from cardiac arrest, and cardiorespiratory resuscitative measures must be instituted immediately. Other signs are the absence of audible heart sounds, pupils which are dilating, and a blood pressure that cannot be measured. One should not, however, waste time in looking for these signs.

The only accurate method of diagnosis is an immediate thoracotomy and palpation or inspection of the heart by the surgeon. Should a diagnostic thoracotomy be done unnecessarily, free bleeding of the thoracic arteries will be found, and the incision need not be extended any further.

In most cases of cardiac arrest there is a critical time-limit of about 3 minutes in which to restore the circulation, and if one does not institute resuscitative measures *immediately*, permanent brain damage is likely to follow.

The stage at which the arrest occurred is reflected in Table VIII, from which it will be seen that approximately 1/6th of the cases occurred during induction, 2/3rds during the maintenance of anaesthesia, and 1/6th immediately after the end of the operation.

Stage at which death occurred. The majority of patients (217) were considered dead within 1 hour of the arrest (Table IX). Seventy patients succumbed within 24 hours after successful massage and another 10 patients lived for at least 24 hours.

Treatment. The detailed treatment of this acute emergency has been described elsewhere. Suffice it here is it to say that the ideal treatment consists of immediate thoracotomy by the surgeon, followed by manual massage and defibrillation where necessary, whilst the anaesthetist institutes effective measures of pulmonary ventilation. In the absence of adequate resuscitative equipment, a laparotomy and massage through the diaphragm by the surgeon, together with mouth-to-mouth breathing by the anaesthetist, is better than no treatment at all.[20] That the treatment of the majority of cases under review left much to be desired is reflected in Tables X – XIV.

TABLE VII. STATUS OF SURGEON AND ANAESTHETIST (300 CASES)

Status	*Surgeon*	*Anaesthetist*
Specialist ..	145	86
Registrar ..	55	105
G.P. ..	92	102
Intern ..	8	7

TABLE VIII. STAGE AT WHICH ARREST OCCURRED (300 CASES)

Period of Arrest	*No. of Cases*	*% of Total*
Induction ..	51	17·0
Maintenance	199	66·3
Within ½ hr. post-op. ..	50	16·6

TABLE IX. STAGE AT WHICH DEATH OCCURRED (300 CASES)

Time of Death	*No. of Cases*	*% of Total*
Under 1 hr.	217	71·6
Under 24 hrs.	70	23·6
Over 24 hrs.	10	3·3
Not stated	3	1·0

TABLE X. ROUTE OF MASSAGE (300 CASES)

	No. of Cases	*% of Total*
Abdominal..	82	27·3
Thoracic ..	122	40·6
No massage	96	32·0

TABLE XI. COMMENCEMENT OF MASSAGE (204 CASES)

	No. of Cases	*% of Total*
Under 3 min.	87	42·2
Over 3 min.	68	33·3
Not stated	49	24·0

TABLE XII. NO MASSAGE: PRE-OPERATIVE STATUS OF PATIENT (96 CASES)

	No. of Cases	*% of Total*
A (Good) ..	5	5·2
B (Fair) ..	27	28·1
C (Poor) ..	46	47·7
D (Moribund)	18	18·7

TABLE XIII. NO MASSAGE: SUMMARY OF TREATMENT (96 CASES)

Drug	Intra-cardiac	Intra-venous	Intra-muscular	Sub-cutaneous
Adrenaline	34	5	0	1
Coramine	9	24	14	0
Noradrenaline ..	5	9	0	0
Methedrine ..	5	11	3	0
Steroids (cortisone, etc.)	0	7	2	0
Neosynephrine ..	0	1	0	0
Megamide	0	2	0	0
Picrotoxin	0	1	0	0

Massage plus injections .. 152 cases
Massage only .. .. .. 42 cases
Oxygen only .. .. .. 13 cases

TABLE XIV. MASSAGE PLUS INJECTIONS: SUMMARY OF TREATMENT (152 CASES)

Drug	Intra-cardiac	Intra-venous	Intra-muscular	Sub-cutaneous
Adrenaline	116	26	—	1
Coramine	32	47	14	—
Noradrenaline ..	25	38	—	—
Methedrine ..	6	39	3	—
Atropine	6	18	—	—
Calcium	31	4	—	—
Other drugs ..	14	59	5	—

DISCUSSION

Although the exact aetiology of this emergency is still a matter of conjecture and argument amongst surgeons and anaesthetists, there is no doubt about the treatment to be followed once an arrest has occurred. From the records we consider that some lives might have been saved had more effective measures of cardiorespiratory resuscitation been instituted immediately after the condition was noticed. Other cases, we feel, might have benefited by more efficient measures of resuscitation before operation, or by a better choice of anaesthetic technique.

As already stated, the majority of arrests (105 of the 300 fatal cases) occurred during abdominal operations, in which a number of the patients suffered from a hypovolaemia and, in our opinion, would have benefited considerably from a detailed pre-operative examination of their fluids and electrolytes followed by replacement therapy where necessary.

Except in cases of uncontrollable haemorrhage, we still believe that no operation should be attempted in the shocked patient until the systolic blood pressure has been stabilized at 100 mm. Hg and the haemoglobin raised to at least 70%.[8] In the treatment of haemorrhagic shock, one can take it for granted that when the systolic blood pressure of a normal adult has dropped below 100 mm. Hg he has lost at least 2½ pints of blood and replacement of this volume by whole blood is absolutely imperative before operation.

In considering the 51 cases of cardiac arrest during induction of anaesthesia, an overdosage of anaesthetic agent or faulty anaesthetic technique cannot be completely ruled out and the anaesthetist, therefore, may not be entirely blameless.

We are obliged again to warn against the indiscriminate use of thiopentone (pentothal) in bad-risk cases, especially by the occasional anaesthetist. When this drug is injected too rapidly, or in too large a dosage, the immediate effect may be a fatal fall in blood pressure, probably due to direct action of the drug on the myocardium.[21] If, therefore, it is used in patients suffering from hypotension or myocarditis, it is advisable to give inhalations of 100% oxygen beforehand, and inject the drug very slowly, preferably in a 1¼% solution.

Most authors are agreed that the ideal treatment of a cardiac arrest, as far as the surgeon is concerned, is immediate cardiac massage through the thoracic route.[9,20] In the 300 fatal cases under review, this was only done in 122 cases. Our main criticism, however, of the methods employed for cardiac resuscitation is that in 96 cases no attempt was made to massage the heart at all; in these cases the prognosis was hopeless right from the start. In most of them an abortive attempt was made to revive the patient by means of subcutaneous, intramuscular, intravenous or intracardiac injections. *We cannot help feeling that the injection of drugs before massage is attempted is a waste of time and should be strongly condemned in the absence of an effective circulation, as the drug cannot possibly reach the coronary arteries.*[8] Furthermore, the injection of drugs like adrenaline intracardially, especially when associated with the inhalation of agents like halothane, trichlorethylene, chloroform or cyclopropane might even be harmful and precipitate attacks of ventricular fibrillation.

We were also impressed with the variety of drugs that were being used, some of which are known not to have any effect on the myocardium. Nikethamide (coramine) may be of use as an analeptic, but it is of little value as a cardiovascular stimulant, and in cases of cardiac arrest it would be much wiser to ask the nurse for a scalpel instead of a syringe filled with the traditional ampoule of coramine.[22]

Another point worth mentioning is that of the 31 fatal cases with ventricular fibrillation, only 16 were treated with an electric defibrillator. In the remaining 15 cases there was no electric defibrillator available. As every heart may theoretically go into fibrillation after an arrest, we seriously recommend, for the attention of the authorities, the installation of defibrillation equipment in all hospitals where major abdominal surgery is being undertaken. We take it for granted that in all hospitals where thoracic surgery is being done electric defibrillators would be readily available.

In those cases in which the treatment appears to have been adequate, why did the patient not recover? We believe that the following factors were mainly responsible:

(*a*) Inexperience on the part of the staff dealing with the emergency and the lack of a proper programme of action once they were faced with an actual case. We think that all surgical and anaesthetic staff should meet at least once a year in order to become acquainted with the latest methods of cardiac resuscitation. At such a meeting a programme of action could be drawn up by the surgeons and anaesthetists and this should be displayed prominently in all localities where surgical operations are being performed.[20] If possible, cases of cardiac standstill and ventricular fibrillation together with effective methods of treatment should be demonstrated on dogs.

(*b*) In a number of cases the massage was begun too late. In order to obtain the best results the surgeon should start the actual massage within *90 seconds* of the arrest.

(*c*) Even though in some cases the massage was attempted in time, the heart might not have been pumped strongly enough to force the blood into the vital organs and maintain a satisfactory blood pressure.

(*d*) In a number of cases the patients were moribund or suffering from chronic heart disease and here the prognosis was more or less hopeless from the beginning.

(*e*) In some cases, especially in the smaller hospitals, the equipment was inadequate to deal with the acute emergency. Every hospital or nursing home, no matter how small, should have immediately available for emergency use at least a cardiac massage drum, suction apparatus, and an apparatus for applying oxygen under pressure.[20]

Finally, we should like to commend to our readers the use of hypothermia after successful massage in cases of suspected cerebral oedema or in patients with hyperpyrexia. Recently some successes were reported from this technique even in cases of arrest in excess of 5 minutes.[23,26]

REDUCTION OF MORTALITY FROM CARDIAC ARREST

As cardiac arrest appears to be on the increase, what can we do in future to reduce the alarming mortality rate which accompanies this emergency? Although the condition is not entirely preventable we feel that strict observance of the following points might assist materially in lowering the death rate:

1. *Pre-operative Period*

(*a*) A detailed history and a thorough examination of the patient before operation, especially in cases of emergency. In routine cases the patient should be examined on admission, and the blood pressure and pulse rate should again be taken immediately before induction of the anaesthetic in order to gauge the effect of fear and premedication.[24] If there is a history of the patient's having taken chlorpromazine (largactil), cortisone, promethazine (phenergan) or reserpine over a long period, appropriate pre-operative treatment should be instituted or the operation postponed.[25]

(*b*) Optimum premedication at the right time, especially with atropine, to reduce the chances of vagal inhibition.

(*c*) Correction of hypovolaemia and electrolyte imbalance before operation, especially in major abdominal and thoracic surgery.

2. *During Operation*

(*a*) The right choice of anaesthetic and the use of techniques with which the anaesthetist is thoroughly familiar.

(*b*) Careful observation of the patient with special reference to respiration, pulse rate, blood pressure and replacement of blood loss; and avoidance of hypoxia and respiratory acidosis.

3. *Immediate Post-operative Period*

(*a*) Wait until the patient is awake before injecting depressant analgesics like morphine, omnopon or pethidine.

(*b*) Unless contra-indications exist, nurse the unconscious patient on his side until he has recovered consciousness—Sims or tonsil position.

(*c*) Whenever possible, keep the patient in a recovery room until his condition is satisfactory.

SUMMARY AND CONCLUSIONS

We have given a review of the aetiology and treatment of 300 *fatal cases* of cardiac arrest associated with surgical procedures and collected from various South African public hospitals over a period of 4 years.

In a number of cases we consider the treatment was inadequate either through lack of knowledge or lack of proper resuscitative equipment.

We feel that more lives may be saved in future if every anaesthetist and every surgeon is made more acutely aware of the danger of cardiac arrest and knows exactly how to act when confronted with such an emergency. For this purpose we advocate regular meetings between surgical and anaesthetic staffs so that all members of the surgical team can receive proper instruction in the latest methods of cardiac resuscitation.

Eternal vigilance is the price of safety

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