CARDIAC ARREST AND RESUSCITATION*

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PART I

Since the last World War many major medical advances have been made, none more dramatic or important than the resuscitation of the human heart in ventricular fibrillation. Every surgeon should know how to resuscitate the heart and how to use the necessary equipment, which should be available wherever surgical operations are performed. This is not an impracticable ideal but reality falls far short of it. Most hospitals possess an internal defibrillator, but how many can provide, at a moment's notice, a thoracotomy drum, ventilation equipment, and a full range of drugs? How many doctors know the difference between an internal and an external defibrillator and are conversant with their indications and limitations? Above all, how many of us are capable of instituting resuscitative measures with confidence and efficiency? The only external defibrillator in use in Johannesburg to my knowledge between 1960 and 1962 was one for animals in the experimental theatre of the medical school. This defibrillator saved several human lives.

While doctors and hospitals are still ill-equipped to fulfil their obligations to routine surgical patients, progress soars ahead. Kouwenhoven and his associates at Johns Hopkins Hospital,[27] without opening the chest, have resuscitated a significant number of patients with acute occlusion of the coronary artery, who would otherwise have died. It is clear that ventricular fibrillation, precipitated by acute coronary thrombosis, is not necessarily an expression of irreversible myocardial damage and, if the rhythm is successfully converted, the patient may have as good an outlook as that of the patient with a non-fatal attack. The techniques of cardiac resuscitation have now extended from the operating theatre to all departments of the hospital; indeed to wherever medicine is practised. A responsibility has been thrust upon every doctor, be he surgically or medically inclined, newly-qualified or long-experienced, to attempt to resuscitate an acutely arrested heart. Anyone may be chosen by chance to witness the collapse. Only the person present can do anything about it.

Historical

According to Norman Morris the history of cardiac arrest and resuscitation begins with Vesalius.[51] He attempted to resuscitate hearts in anoxic asystole by inflating the lungs through an endotracheal tube. No great advance was made until the work of Moritz Schiff (1823 - 1896) who, while professor of physiology in Florence, carried out many experiments on animals.[22] He understood more about cardiac resuscitation than most modern surgeons. The following quotation[36] is taken from his collected works published in 1896: 'But if one opens the thorax while air is slowly blown into the lungs and compresses the heart rhythmically with the hand to squeeze out blood, compressing the abdominal aorta at the same time so that more of the artificial circulation is directed to the head, taking care not to obstruct the coronary circulation, one can restore the heart as long as 11½ minutes after it has been arrested'. In Schiff's time the question of sudden cardiac arrest was exciting considerable discussion because of the recent introduction of chloroform anaesthesia in Edinburgh in 1847 by Sir James Simpson and of ether anaesthesia by William Morton, of Boston, in 1846. Two months after Simpson's announcement, the first death from chloroform occurred in Newcastle-upon-Tyne. In 1858 John Snow[49] described 50 deaths from chloroform. Ether, whose harmlessness had been extolled so enthusiastically in America, was not without its dangers, and by 1861 it had been implicated in 41 deaths.[23] However, though Schiff had clearly enunciated the principles of resuscitation, only sporadic attempts were made in man.[52, 58] To the surgeons of the late 19th century the human heart was an awesome thing and the possibility of resuscitating it must only have been entertained by visionaries and cranks. The great Billroth had this to say in 1875,[8] 'Let no man who hopes to retain the respect of his medical brethren dare to operate on the human heart'; while Paget[40] in 1896 was so far off the mark as to write, 'surgery of the heart has probably reached the limits set by Nature to all surgery.'

In early attempts at resuscitation the arrested heart was stimulated to beat by manual irritation through the diaphragm. Sir William Arbuthnot Lane (1902) was the first to succeed.[50] In 1906 Green[21] reviewed the world literature to find only 9 survivals. At that time Mauclaire[35] reported that 17 attempts had been made by the transthoracic route, with one survival. These were all cases of asystolic arrest, and it was not until 1942 that a case of ventricular fibrillation was resuscitated by Adams and Hand.[1] In this case the heart was defibrillated with procaine. Five years later a more important case was reported by Beck *et al.*,[4] in which after 70 minutes of ventricular fibrillation and massage the heart was defibrillated electrically. By 1954 Milstein and Brock[37] were able to find 17 reports of successful defibrillations, to which they added 9 further cases of their own. Cardiac surgeons are now so familiar with cardiac arrest that they no longer report isolated cases, but Stephenson[51] has established a 'cardiac arrest registry' and by 1958 had collected records of 1,710 cases, with 536 survivals (31%).

The magnificent physiological and experimental studies of the pioneers are today bearing abundant fruit. Early lack of success in human patients was due, not to insufficient knowledge, but to the timidity and conservatism of practising surgeons, who imagined the heart to be a fragile organ not to be desecrated after it had ceased to function effectively. We can now control the heartbeat and, provided oxygenation is maintained by extracorporeal circulation, it can be stopped or fibrillated with complete confidence that full function will return. Most of the techniques of today were introduced by men long since dead. William Harvey showed that manual stimulation was

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capable of restoring the beat of an arrested heart.[13] Friedburg[20] (1859) used an electric current to revive the arrested heart. Schiff (1896) clearly outlined the need for positive-pressure respiration and emphasized the effectiveness of cardiac massage. Arnaud[2] (1891) showed the importance of perfusing the coronary arteries with oxygenated blood by intra-arterial transfusion. Ringer[44] (1883) was the first to demonstrate the effects of calcium and potassium ions on myocardial contractility. Adrenaline was discovered in 1895 by Oliver and Schafer[39] and its intracardiac injection was utilized in 1919 by Von den Velden.[53] In 1920 Schott[47] described the method of external thumping of the praecordium now recognized to be effective in restarting certain hearts in asystole. The first clinical defibrillator was developed by Hooker, Kouwenhoven and Langworthy[24] in 1933 and the uses of this instrument were later elaborated by Wiggers.[56] The work of these great pioneers has been subjected to frequent re-appraisal and refinement, but they laid solid foundations upon which the present safety of cardiac surgery is based.

In Johannesburg the earliest resuscitation of a fibrillating heart by electric defibrillation was accomplished on 23 April 1954 by Mr. L. Fatti. It is possible that this was the first in South Africa because, as far as I know, the first electric defibrillator in this country was made for us in 1953 by Prof. A. Bozzoli, Head of the Department of Electrical Engineering, University of Witwatersrand. He constructed a machine to deliver the voltages and amperage recommended by Wiggers. This defibrillator still functions efficiently in our experimental theatre. Fatti's first case is worth recording.

Case 1. Mr. P.B., aged 38. This man had a tight mitral stenosis, for which a valvotomy was being done on 23 April 1954. During splitting of the commissures the heart fibrillated. It was immediately massaged and after 10 minutes 0·5 ml. of 1:1000 adrenaline was injected into the left ventricle. The heart became pink and fibrillated strongly and it was then stimulated with a single shock of 120 v. for 0·2 sec. Defibrillation was immediate and the heart recovered to beat synchronously. The patient awoke on the table without neurological disturbance. He was discharged from hospital 3 weeks later.

The defibrillator had been used once, unsuccessfully, 3 months earlier. In the 4 previous years, when cardiac surgery was in its infancy, Fatti dealt with 9 cases of cardiac arrest. Three were successfully resuscitated (33%), 2 being cases of ventricular fibrillation. They are recorded below (cases 2 and 3), and illustrate what was accomplished by determination and persistence before electrical defibrillation was available.

Case 2. Mrs. H.L., aged 37. A difficult, prolonged, but successful digital commissurotomy for mitral stenosis had been completed on 28 November 1951. The chest was closed, when suddenly the ECG showed ventricular fibrillation. The chest was immediately re-opened by Mr. G. R. Crawshaw and the arrhythmia confirmed. Cardiac massage was started. A coordinated beat returned within 5 minutes, but without sufficient force to maintain the circulation. Adrenaline was injected and the heart assisted manually. Ventricular fibrillation recurred. After a further 10 minutes of massage, the rhythm again converted spontaneously. Thereafter the heart was intermittently assisted by manual compression, but only after an hour of effort was it able to maintain the circulation unaided. After the operation the patient remained deeply unconscious for 48 hours. Her limbs were spastic and the pupils widely dilated. Her condition slowly improved but she remained confused, drowsy and aphasic. On 12 January 1951 she developed severe diarrhoea and became stuporose. Because of dehydration, intravenous therapy was necessary. Overnight she improved and next day, though weak, she was alert and able to speak for the first time since the operation. Thereafter she steadily improved. When seen in February 1963 she was mentally normal. She had led a useful and productive life.

In the second successful case procaine was the defibrillating agent.

Case 3. Mr. C.J., aged 34. This patient was in intractable congestive cardiac failure owing to a mitral stenosis, and on 15 December 1952 the commissures were split satisfactorily by digital pressure. After suturing the appendage, tachycardia developed and ventricular fibrillation rapidly supervened. Cardiac massage was commenced and the aorta compressed beyond the left subclavian artery. 100 mg. of procaine hydrochloride was injected intravenously and massage was continued. A weak coordinated beat developed within 5 minutes. 5 ml. of aminophylline was injected into the cavity of the left ventricle and thereafter tachycardia was followed by a forceful regular beat. The patient recovered consciousness slowly and was eventually discharged 6 weeks after surgery.

Definition of Cardiac Arrest

Twelve definitions of cardiac arrest were listed by Natof and Sadove[38] in 1958. The basic theme behind them all is that the heart can be resuscitated. 'Cardiac arrest' in the present context does not therefore apply to the cessation of the heartbeat of a person suffering from a chronic debilitating disease. Similarly it does not apply to death from a fulminating acute illness or uncontrollable haemorrhage. In 1963 Milstein[36] defined cardiac arrest as 'failure of the heart action to maintain an adequate cerebral circulation in the absence of a causative and irreversible disease.' This definition emphasizes the paramount importance of the cerebral circulation and the possibility of reversibility. It is probably as close as one can get to accuracy but 'sudden and usually unexpected failure of the heart to maintain circulation' (Reid *et al.*,[43] 1958), though less precise, is more descriptive of the condition as it presents in practice.

Definitions of cardiac arrest do not differentiate between ventricular asystole (standstill) and ventricular fibrillation. Even a coordinated but ineffectual ventricular beat may be responsible. According to Stephenson's records of 1,710 cases, ventricular fibrillation occurred in 11·3% of cardiac arrest. This figure is much lower than the experience of cardiac surgeons would indicate.

Under experimental conditions the heart can stop in either systole or diastole; systole can be caused by an excess of calcium ions in the circulation, diastole follows the injection of potassium. Clinically, ventricular standstill is always in diastole.

Presentation of Cases

Since acquiring an electrical defibrillator Mr. Fatti and I have treated 52 cases of cardiac arrest during thoracic operations or investigations (Table I). I have also been concerned with 11 other cases in operating theatres, wards, radiology departments, or doctors' consulting rooms (Table II). On 8 occasions I was not present when arrest developed, but was called to assist after resuscitative steps had been initiated. The series therefore totals 63 cases. 45 of these cases occurred with the chest open and 18 with the chest closed. Further, in 62 operations under cardiopulmonary bypass, asystole or ventricular fibrillation has either been purposely produced for a specific purpose or has developed spontaneously; conditions here are quite different

from those of cardiac arrest with an unsupported circulation and these cases are only used to illustrate the aetiology of ventricular standstill or fibrillation.

TABLE I. CASES OF CARDIAC ARREST OCCURRING DURING THORACIC SURGICAL PROCEDURES EXCLUDING OPEN-HEART SURGERY

	Number	*Successful restoration of heartbeat*	*Recovery to 'pre-arrest brain status'*
Mitral valvotomy	24	20 (83%)	14 (60%)
Congenital cyanotic heart disease	5	1 (20%)	0 (0%)
Congenital acyanotic heart disease	4	2 (50%)	1 (25%)
Pulmonary resection	7	4 (57%)	3 (43%)
Drainage of empyema	1	0 (0%)	0 (0%)
Bronchogram and bronchoscopy	6	5 (83%)	5 (83%)
Cardiac catheterization	1	1 (100%)	1 (100%)
Aortic valvotomy	4	2 (50%)	1 (25%)
Total	52	35 (67%)	25 (48%)

Incidence of Cardiac Arrest

According to Stephenson, between 7,500 and 9,000 cardiac arrests occur in operating theatres annually in the USA. Almost 12 million anaesthetics are administered there each year. Beecher and Todd,[7] in 1954, recorded the frequency of cardiac arrest in 10 large American university hospitals as 1 case in 1,500 anaesthetics administered. Of course, the frequency will vary with the nature of the surgery but, in general, a figure of 1 case in 1,500 - 2,000 anaesthetics appears to be the average. These figures do not include cases of acute circulatory collapse following cardiac infarction, anaphylactic reactions, drownings, electrocutions, heart-blocks and drug sensitivity. Beck *et al.*,[6] in 1956, estimated that 90% of sudden deaths from coronary artery disease are due to rhythm disturbance and only 10% to myocardial failure. Wood,[57] in 1956, expressed the belief that one-third of all deaths from myocardial infarction were due to sudden ventricular fibrillation or asystole.

TABLE II. CASES OF CARDIAC ARREST OCCURRING DURING NON-THORACIC PROCEDURES

	Number	*Successful restoration of heartbeat*	*Total recovery*
Viscero-cardiac reflexes	3	2	2
Drug sensitivity	2	1	1
Spinal operations — Wilson's rest	2	2	1
Drainage of subphrenic abscess	1	0	0
Haemorrhage	2	1	1
Coronary thrombosis	1	1	0
Total	11	7 (63%)	5 (45%)

In the 10 years 1954 - 63 covered by this survey, our 52 cases of cardiac arrest occurred during the course of about 10,000 anaesthetics, so that the incidence is 1 case in 200 thoracic procedures. The incidence was highest in direct heart operations, being about 1 in 10 closed operations for cyanotic heart disease, 1 in 40 mitral valvotomies, and 1 in 80 acyanotic congenital heart operations. There has been one cardiac arrest in every 300 pulmonary resections, 1 in 850 bronchoscopies or bronchograms, but none during oesophageal, mediastinal and chest-wall surgery or endoscopic investigations for these conditions. These are figures of a special branch of surgery where direct heart stimulation and acute circulatory obstruction result in an incidence 10 times greater than for other surgical procedures.

CAUSES OF CARDIAC ARREST

Clinical differentiation between ventricular fibrillation and standstill is impossible. A correct diagnosis can only be made by electrocardiography (Figs. 1 and 2) or by direct observation. Ventricular fibrillation, when fine and feeble, cannot be accurately felt, and conclusions drawn from palpation through the diaphragm or pericardium are often inaccurate. In our experience, ventricular fibrillation is twice as common as standstill (Table III). In addition, 43% of asystolic arrests have converted to ventricular fibrillation during massage (Table III).

Under cardiopulmonary bypass, ventricular asystole can be intentionally produced by total cardiac anoxia, by deep hypothermia, and by injecting potassium into the coronary arteries. If the aorta is clamped so as to stop the coronary circulation, the heart will continue to beat for 1 - 5 minutes. Initially it often accelerates for a few moments but, as cyanosis deepens, bradycardia develops and the ventricles become progressively more flaccid. On the other hand, obstruction of one main coronary artery will usually cause ventricular fibrillation. Provided the ventricles are protected from distension, the well-oxygenated heart will not fibrillate during cooling, but will pass into asystole at a temperature of 12 - 16°C. If stimulated or distended, it will fibrillate at 22 - 28°C.[33] A well-oxygenated healthy myocardium will withstand considerable manipulation at temperatures above 28°C, but an anoxic heart, or one grossly hypertrophied or diseased, will fibrillate when handled or incised. An electric current of 15 volts and less than 15 amps applied to the ventricles will cause fibrillation, and this technique is deliberately used to improve intracardiac operating conditions (Fig. 3). The oxygenated heart will often revert spontaneously to normal rhythm after the current is removed. If air bubbles enter all the coronary arteries evenly, the heart will arrest in asystole, but if they block one main coronary artery only, fibrillation usually results. If the right side of the heart is obstructed by air, asystole usually follows, but stimulation or massage will cause fibrillation.[34] These observations show that asystole is the usual response to uniform anoxia, whereas ventricular fibrillation follows stimulation, distension and unequal anoxia. A uniformly anoxic heart will fibrillate if irritated mechanically. The final state of cardiac death is asystole. The finding of asystole at resuscitation may therefore be a good or a bad sign. If sudden and acute, it is easily reversed, but asystole may be the end-result for a heart that has passed from ventricular fibrillation to exhaustion and death.

If the vagus nerve of a dog is stimulated with an alternating current of 3 - 6 v., the heart will immediately stop. If the current is maintained it will start to beat within 10 - 30 seconds but at a slow rate ('vagal escape'). This phenomenon also occurs in man and is prevented by atropine. Reflex cardiac arrest can follow the stimulation of any area where vagal nerve endings are found (Fig. 1). Reid and Brace[42] (1943) have shown that one of the commonest sites of origin of stimuli is the tracheobron-

Fig. 1. Vagus arrest and escape during intubation. ECG tracing taken during anaesthetic intubation showing a brief period of asystole. This tracing is comparable with that of experimentally produced vagus arrest and escape. The recovery rate is slower than the pre-arrest rate. *Acknowledgement to Dr. J. W. Mostert.*

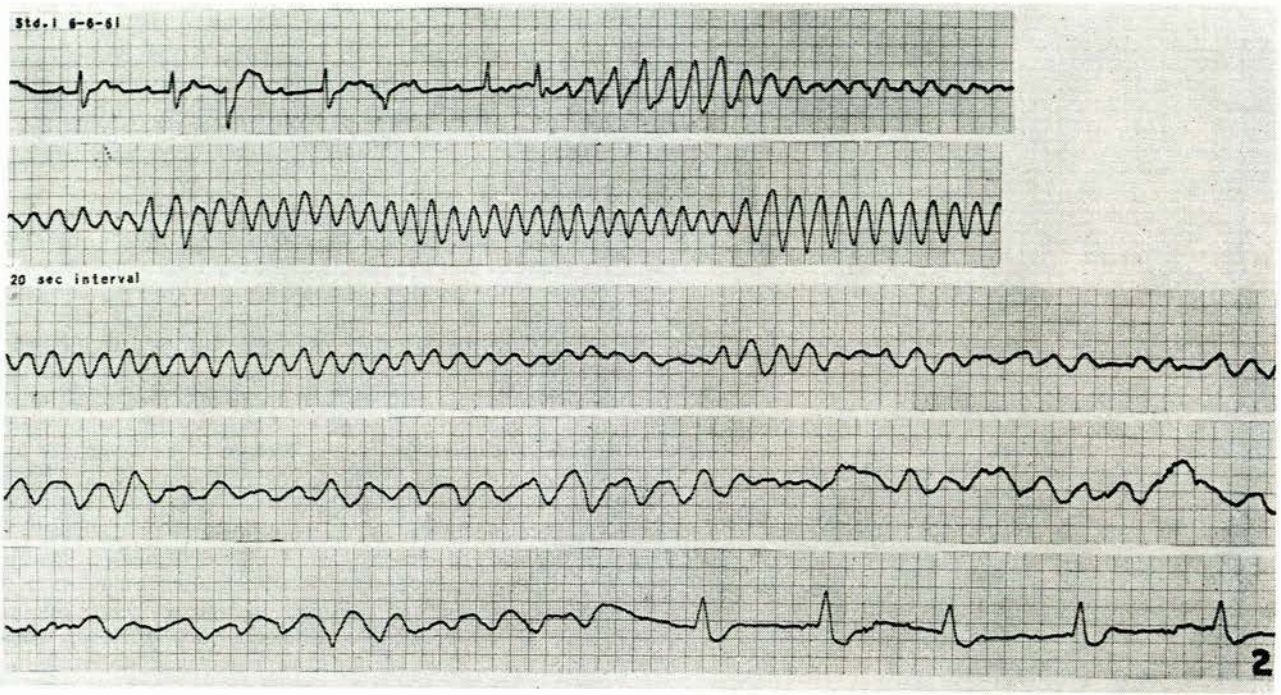

Fig. 2. ECG tracing taken during a Stokes-Adams attack. Ventricular fibrillation of the coarse vigorous variety developed. Spontaneous reversal. *Acknowledgement to Dr. J. B. Barlow.*

chial mucosa, and there are many recorded cases of arrest following tracheal intubation.

Adrenaline has long been implicated in the aetiology of ventricular fibrillation. In the presence of anoxia or of anaesthetic agents such as chloroform and cyclopropane, adrenaline injections are notoriously dangerous. Endogenous adrenaline secretion follows sympathetic stimulation from fear. It is produced in large quantities by the adrenal glands in response to hypoxia, hypercapnia, and haemorrhagic hypotension. Rapid intravenous injections of adrenaline lead to high serum-potassium levels as a result of release of potassium from the liver.[12] Adrenaline may cause cardiac arrest either by direct action upon the heart or by disturbing the calcium:potassium ratio of the blood.[10]

There are many possible causes of acute cardiac arrest and in any one case a combination of factors is usually involved. An attempt is made here to analyze the main causes in our cases (Table III).

Anoxia

Anoxia was implicated in 45 cases of cardiac arrest (71%). Anoxia has been due to respiratory causes such as ineffectual ventilation under anaesthesia or obstruction of the air passage by foreign bodies. Acute haemorrhage with systemic hypotension has been another cause of cardiac anoxia. Circulatory obstruction during intracardiac manipulations is a hazard that leads to anoxia but is peculiar to cardiac surgery. Two cases of cardiac arrest occurred during spinal operations with the patient in the prone position on a Wilson's rest. This position is dangerous, for the legs and head are dependent and the pressure exerted upon the chest and abdomen must seriously impede the venous return to the heart. In addition the anaesthetist must literally lift the thorax off the rest to ventilate the lungs.

TABLE III. ANALYSIS OF CAUSES AND NATURE OF CARDIAC ARREST AND RESULTS OF RESUSCITATION

		Initial arrest		*Asystole to*			
	No.	*Vent. fibr.*	*Asystole*	*Vent. fibr.*	*Successful resuscitation*	*Late deaths*	*Total recovery %*
Acute anoxia ..	29	16	13	5	21 (72%)	7	48%
Chronic hypoxia	11	6	5	3	2 (18%)	2	0%
Air emboli ..	5	4	1	1	4 (80%)	1	60%
Vagal reflex ..	5	3	2		4 (80%)	0	80%
Myocardial irritability ..	8	8	0		8 (100%)	1	87%
Toxaemia ..	2	2	0		0 (0%)		0%
Massive blood transfusion ..	3	3	0		3 (100%)	1	67%
Total ..	63	42	21		42 (67%)	12	48%

Chronic anoxia due to heart failure or respiratory disease is particularly dangerous (Table III). With these conditions it takes little extra to precipitate cardiac arrest. It is

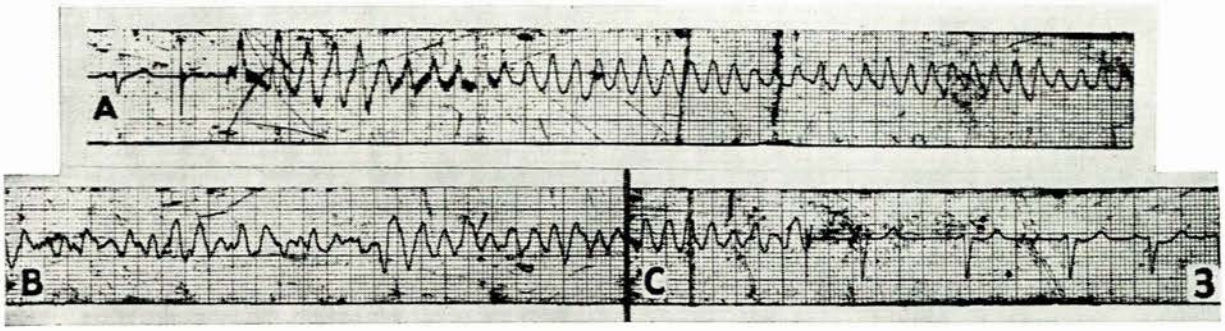

Fig. 3. ECG showing induced ventricular fibrillation and direct defibrillation during open-heart surgery under cardiopulmonary bypass. A=ventricular fibrillation induced by ventricular stimulation 15 volt 1 amp. B=ventricular fibrillation suitable for defibrillation. C=direct defibrillation.

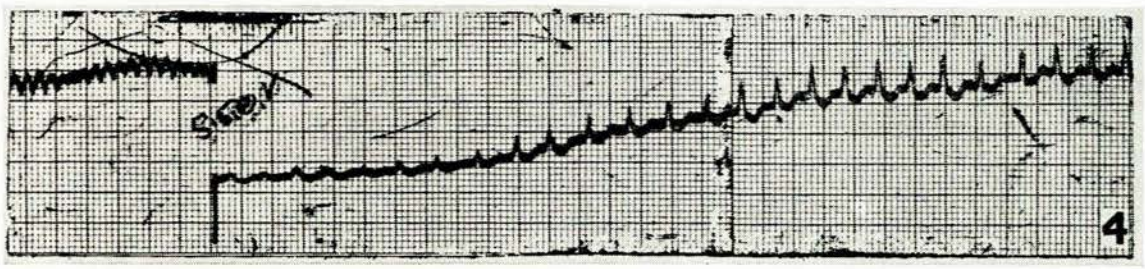

Fig. 4. ECG taken during external defibrillation. The ECG machine was disconnected while the shock was administered so as to protect the equipment. Idioventricular rhythm started immediately and the amplitude of the QRS complex rapidly increased. P waves begin to appear towards the end of the tracing.

sometimes necessary to operate on patients whose vital organs are already hypoxic, and then irreversible cerebral changes may occur without the heart having stopped.

Case 4. Mrs. M.A., aged 23. This patient had suffered from mitral stenosis for many years. When 8 months pregnant she was admitted to the Queen Victoria Maternity Hospital with pulmonary oedema. This proved refractory to the usual medical measures and urgent valvotomy was recommended. She resolutely refused consent and was only prevented from drowning in oedema fluid by positive-pressure ventilation with a Bird respirator administered through a face mask. She remained deeply cyanosed and dyspnoeic for 24 hours before permission for operation was granted. A rapid and complete valvotomy was performed on 11 August 1962 with a Tubb's dilator. Only during the commissurotomy, which lasted about 30 seconds, was the heartbeat unduly weak. It responded promptly to an injection of 5 ml. of 1-in-10,000 adrenaline and manual assistance for about 20 seconds. However, the patient remained deeply unconscious and died 48 hours later.

Left-sided *air embolism* causes cardiac arrest by obstructing the coronary circulation, while right-sided emboli block the pulmonary blood flow. In one case right-sided air embolism was due to neglect in supervising a rapid blood transfusion administered by pumping air into the blood bottle with a Higginson's syringe. In another it followed a per-cricoid bronchogram; the 'lipiodol' needle pierced the thyroid plexus of veins and when the patient coughed, air was forced around the needle into the veins and the patient collapsed. A left-sided air embolism was produced by inadvertently flushing air through a left ventricular needle inserted to record pressures at operation. A second left-sided embolism appeared apparently miraculously during mitral valvotomy. This could only have happened as a result of obstructing the blood flow to the left side, probably by compressing the pulmonary artery. When the left atrium empties of blood the contractions of the ventricle can suck air into the atrium around the finger.

The third case of left-sided air embolism is briefly recorded:

Case 5. P.M., aged 38. This Bantu male was subjected to thoracotomy on 16 April 1959 with the intention of removing the left lung, which was the site of a cavitating carcinoma. A Thomson's blocker was used to obstruct the left main bronchus. As the incision was made the anaesthetist reported that the heart had stopped. The chest was rapidly opened and air bubbles escaped from divided vessels. The heart was in standstill and its left chambers were distended with froth. Bubbles were present in the coronary vessels. Small punctures were made in the aorta, the left atrium, and a large coronary vessel from which frothy blood escaped. The ascending aorta was clamped and the heart massaged. Fibrillation supervened. 10 ml. of 1-in-10,000 adrenaline was injected and the fibrillation became coarse and forceful. The heart was defibrillated with a single shock. The heart beat well but it was decided to abandon the operation. The patient remained unconscious and died 8 hours later. Death resulted from cerebral air emboli. It seems likely that air was trapped under pressure beyond the bronchial blocker and was forced into a pulmonary vein in the wall of the cavity.

Vagal Reflex

Autonomic reflexes mediated by the vagus nerve may cause sudden cardiac arrest. Simpson[48] described 87 such cases in 1949. In his experience stimuli from the uterus and cervix, the throat and glottis, and the bladder and urethra, are most commonly involved. He emphasizes that the disaster is most liable to occur under light anaesthesia and that the effect is instantaneous and far too rapid for anoxia, pH changes, hypotension, or the production of endogenous adrenaline, to be responsible.

Vagal arrest usually occurs in patients who have been

inadequately premedicated with atropine or where the effects of atropine have worn off by the time the anaesthetic is given (Fig. 1). Milstein[36] makes a strong plea for the intravenous injection of atropine in adequate doses when the patient is about to be anaesthetized. He advocates a full dose of 1·0 mg. for adults and not the generally used dose of 0·6 mg.

In this series there are 5 cases of cardiac arrest where viscerocardiac reflexes were implicated (Table III). In 2 of them cardiac arrest occurred during intubation under light anaesthesia preparatory to tonsillectomy, and 2 other patients collapsed during bronchoscopy. The fifth died in a consulting room while an instrument was being inserted into the cervix for insufflation with air. Vagal reflexes cause standstill and in these cases thumping of the praecordium is particularly effective in restarting the heart.

Myocardial Irritability

Every cardiac surgeon knows that some hearts are more irritable than others and that mechanical stimulation alone sometimes causes ventricular fibrillation. A particularly irritable area of the heart is the region of the transverse sinus. The extremity of the medial commissure is another danger zone and 3 of our cases have fibrillated while the fused medial commissure of stenosed valves was being split digitally. This area is adjacent to the auriculo-ventricular bundle of His,[18] and the arrhythmia is probably caused by its stimulation. The anoxic heart is particularly irritable.

A minor coronary-artery occlusion may precipitate ventricular fibrillation, and cardiac 'irritability' may account for this happening in one case and not another. Beck and Leighninger[5] have ascribed fibrillation in these cases to 'oxygen differentials'. They postulate that electric currents develop at the junction of the oxygenated and anoxic areas of myocardium and that a current of injury asynchronously stimulates individual muscle fibres. Danese,[15] however, has shown that perfusion of a single coronary-artery branch with serum while the rest of the heart is perfused with blood does not result in ventricular fibrillation; this suggests that an oxygen differential is not responsible but rather that electrolytic changes across myocardial cells, or anoxic myocardial products that are swept away by the serum perfusion, are the cause.

During cardiac catheterization ventricular fibrillation is an ever-present hazard. Anoxia caused by heart disease, poor ventilation, or excessive sedation, is responsible in some cases. In my experience of 350 cardiac catheterizations, ventricular fibrillation has occurred once.

Case 6. Mr. H.K., aged 44. This patient had aortic stenosis, and a transthoracic left ventricular needle was inserted with the intention of measuring the pressure gradient across the aortic valve. Only at the third attempt was the ventricular cavity entered. Circulatory failure developed before pressures could be recorded. The needle was hastily withdrawn but ventricular fibrillation supervened. The examination was performed under general anaesthesia and ventilation was already under control. The chest was opened by Mr. L. du Plessis. Blood was present in the pericardium, showing that tamponade was a factor additional to myocardial irritability. Massage was started and when the heart was pink it was shocked electrically. Defibrillation was immediate and a coordinated beat returned, but after one minute fibrillation recurred. Again the heart was massaged and defibrillated but the sequence was repeated. Attempts were made, without success, to split the stenosed valve, and at the third attempt, after 10 ml. of 10% calcium lactate had been injected, the heart remained in sinus rhythm. The patient recovered and was eventually discharged, having declined valvotomy.

This is the only serious incident in 70 left ventricular punctures and more than 100 other left heart catheterizations. Runs of extrasystoles are not infrequent during both left and right heart catheterizations but these always cease when the catheter is withdrawn. We have had no arrests during angiocardiography. In 1950 Dotter and Jackson[17] collected 26 deaths associated with angiocardiography from 182 hospitals, and the mortality rate was 0·38%.

Toxaemia

Two patients with chronic abscesses developed ventricular fibrillation during operative drainage, and neither could be resuscitated. Both were seriously ill and care had been taken to avoid anoxia during induction of the anaesthetics. The deaths were attributed to toxic myocarditis. Recovery from cardiac arrest due to myocarditis following diphtheria, poliomyelitis or acute pyogenic infection is only remotely possible. However, when additional stimuli have been added, resuscitatory attempts are worthwhile. The following is an example of such a case (toxaemia, anaesthesia, and vagal inhibition).

Case 7. H.E., aged 7. This White boy had been admitted to the Fever Hospital with ascending paralysis of the Guillain-Barré type. For 6 weeks he had been treated with an Engström respirator. When his respiratory muscles were judged to have recovered sufficiently, mechanical ventilation was abandoned. A fortnight later he developed atelectasis of his left lung, his condition rapidly deteriorated, and he became hyperpyrexial. On 1 September 1962 bronchoscopy under pentothal and scoline anaesthesia was started, and as the bronchoscope was passed his pulse disappeared. External cardiac massage was immediately started and continued for 10 minutes, during which time the chest was cleansed and towelled and one was able to draw on sterile gloves. The left chest was opened and the heart found to be fibrillating. After 30 seconds' massage it defibrillated with a single shock. The tracheotomy was reopened and the patient was again ventilated mechanically. This was continued for 2 weeks. He is now well and ambulant.

Massive Blood Transfusion

Two patients developed ventricular fibrillation towards the end of protracted pulmonary resections accompanied by steady haemorrhage from division of dense adhesions. Blood loss had been continously measured and adequately replaced throughout the operation, and hypotension had never developed. One patient had received 7 pints of blood, the other 6. Both were successfully resuscitated. A third developed cardiac arrest 6 hours after an emergency gastrectomy for persistent ulcer haemorrhage. He had received 12 pints of blood. Many factors are probably concerned in this type of case. Le Veen *et al.*[29] have shown that serum potassium rises sharply when stored blood is transfused. Hypotension, hypercapnia and anoxia all induce the mobilization of potassium from the liver, and the calcium:potassium ratio may thereby be disturbed and cardiac contractility affected.[10] The massive blood transfusions used in open-heart surgery cause less biochemical disturbance, owing, no doubt, to the use of fresh heparinized blood and not stored citrated blood. Factors that must be avoided with all massive transfusions are pH changes and the use of cold blood that will cool the heart. The administration of calcium and digitalis[28] lessens the chance of cardiac arrest when large quantities of stored blood are transfused.

(To be continued)