SOME MECHANISMS OF CARDIAC ARREST DURING ANAESTHESIA

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The advent of thoracic surgery has been followed by a renewal of interest in the mechanisms and treatments of the acute cardiac catastrophes which may occur during clinical anaesthesia. It has now become apparent that the cessation of cardio-dynamics during anaesthesia may be due either to ventricular fibrillation or to asystole unprecedented by fibrillary phenomena. Numerous authors have acclaimed the success of direct cardiac massage, and other manoeuvres, in the treatment of cardiac arrest during thoracic surgery; some have even gone so far as to advise immediate diagnostic thoracotomy when the peripheral pulse ceases to be palpable in the anaesthetized patient (Rao et al., 1954; Milstein and Brock, 1954; Mullens, 1955).

It is obvious that direct cardiac massage is the only treatment for an established cardiac arrest. This treatment can be instituted promptly and with comparative ease in patients undergoing or about to undergo thoracic surgery. It should be remembered, however, that most cardiac arrests under anaesthesia occur in patients prepared for other types of surgical operations; under these circumstances the accurate diagnosis of asystole or ventricular fibrillation may be virtually impossible in the absence of cardiographic tracings. In my experience the absence of a peripheral pulse is certainly not always an indication for immediate thoracotomy to determine the state of the myocardium. Therefore, in an attempt to counter the possible overenthusiastic acceptance of cardiac massage in the treatment of cardiovascular collapse during anaesthesia, it is proposed to demonstrate that it may be possible, with the more liberal use of the electrocardiograph, to anticipate major cardiac upsets in the anaesthetized patient and to take steps to avoid their occurrence.

Many reports of animal experiments have thrown some light on the problem, but the results are controversial. It will be clear to those of us engaged in the practice of clinical anaesthesia that the final solution must inevitably be found through the medium of comprehensive clinical observation. It is doubtful whether the complex pathological and metabolic abnormalities which we regularly encounter in our everyday work will ever be reproduced in the experimental animal.

The first investigation into the aetiology of cardiovascular collapse during anaesthesia was reported by John Snow in 1858. He observed that most catastrophes occurred in the inductive phases of anaesthesia and appeared to be directly related to the inhalation of excessively high concentrations of the vapour. Cardiac inhibition could be prevented by restricting the vapour concentration to 2 per cent or less; the inhalation of higher concentrations often caused stoppage of the heart before the cessation of respiration. Snow’s work was repeatedly confirmed (Kirk, 1893; Brodie and Russell, 1900; Embley, 1902). It was demonstrated that the inhalation of chloroform vapour caused reflex inhibition of the dog’s heart as the result of chemical stimulation of vagal receptors in the air passages. Brodie and Russell (1900) made the important observation that, whereas stimulation of the peripheral end of the cut vagus seldom if ever caused permanent arrest of the heart before the cessation of respiration, Snow’s work was repeatedly confirmed (Kirk, 1893; Brodie and Russell, 1900; Embley, 1902). It was demonstrated that the inhalation of chloroform vapour caused reflex inhibition of the dog’s heart as the result of chemical stimulation of vagal receptors in the air passages. Brodie and Russell (1900) made the important observation that, whereas stimulation of the peripheral end of the cut vagus seldom if ever caused permanent arrest of the heart, stimulation of the pulmonary branches of the vagus produced intense cardiac inhibition; they confirmed and extended the work of Snow and showed that chemical stimulation of vagal receptors in the lower air passages caused reflex arrest of the heart.

Levy (1912), using the electrocardiograph for the first time in experimental anaesthesia, emphasized his belief that chloroform syncope in cats was due to an hyperexcitability of the ventricles leading to ventricular tachycardia and ventricular fibrillation. This hyperexcitability of the ventricles
was associated with overactivity of the sympathetic nervous system. He was very critical of the views of Snow and his supporters and refused to accept that the vagus played any part in the suppression of the heartbeat during anaesthesia. In the following years Levy's thesis was generally accepted and the vagal concept tended to be overlooked. It is not improbable that the principal reason for the general acceptance of Levy's work was the fact that his descriptions were supported by indisputably accurate cardiograms.

More recently, Sealy et al. (1954) and Young et al. (1954) disputed the suggestions that autonomic activity played any part in cardiovascular collapse during anaesthesia. They demonstrated that retention of carbon dioxide during anaesthesia in animals caused a rise in the plasma potassium to cardiotoxic levels. A sudden lowering of the alveolar CO₂ level, after a prolonged period of hypercarbia, was followed by a still further increase in the plasma potassium content which precipitated fatal ventricular fibrillation. They made the important observation that electrocardiographic changes were the only important signs of impending cardiovascular collapse; these changes occurred sufficiently early to permit the institution of successful corrective measures.

These three theories—the vagal concept of the earlier workers, the sympathetic hyperirritability of Levy, and the potassium intoxication of Sealy—represent our knowledge of the factors which predispose to sudden cardiac arrest during anaesthesia in animals. I have no doubt that the findings of these three groups of workers are perfectly correct but I hope to show that the criticisms of the latter groups were unjustifiable. I feel quite certain that sudden cardiovascular collapse during clinical anaesthesia may be due to any one of the three mechanisms described, in addition to others not yet fully understood.

During the past eight years I have made extensive use of the electrocardiograph during clinical anaesthesia in all types of patients undergoing major and minor surgical procedures. It has now become apparent that there are at least five different and clearly defined sequences of cardiac derangement which may occur in man and which may lead to complete arrest of the heart. The object of the present paper is to present a series of case reports, complete with cardiograms, illustrating some of the changes of cardiac action which may end in arrest. I will endeavour to show that autonomic overactivity of either the sympathetic or parasympathetic nervous systems, potassium intoxication, and interference with the coronary circulation, produce specific electrocardiographic changes which, if identified in the early stages, may provide sufficient warning of impending arrest to permit the early application of successful resuscitative measures.

**Arrêt by Vagal Overactivity.**

This is the mechanism first identified by Snow but later denied by many workers because they were unable to cause permanent arrest of the animal heart by stimulation of the peripheral end of the cut vagus. I think that these workers were unwise in assuming that the cardiac effects induced by stimulation of the peripheral end of the cut vagus were necessarily similar to those induced reflexly—with irritant anaesthetic vapours—of the innumerable vagal receptors situated in the air passages. Furthermore, we cannot deny the possibility of vagal arrest of the human heart simply because it is impossible to stop an animal's heart by direct vagal stimulation. We should remember that the human autonomic nervous system is subject to variations of activity which do not obtain in animals—variations which seem to be related to conditions such as peptic ulceration, essential hypertension, certain forms of cardiac arrhythmia, bronchial asthma, and other states now referred to as syndromes of adaptation or stress.

By the simple experiment of filling the lungs with a mixture of gases containing an irritant vapour, and at the same time preventing coughing and straining (Johnstone, 1951a; 1953b), it is possible in some individuals to induce a condition which simulates death so closely as to be indistinguishable from it clinically and electrocardiographically, especially in curarized subjects. The following case illustrates this mechanism.  

**Case 1**

Healthy male, 41 years. Herniorrhaphy. No preoperative drugs given. Anaesthesia induced with thiopentone and maintained smoothly for fifteen minutes with nitrous oxide, oxygen, and trichlorethylene. Ether was then added and respiration assisted. A few minutes later the patient became profoundly collapsed and pulseless; the cardiograph revealed cardiac arrest (fig. 1, d). During the period of arrest the respiratory movements were shallow and gasping. The ether and
trichlorethylene were immediately turned off and the patient ventilated with oxygen; three minutes later the cardiovascular system had returned to normal and the patient made an uneventful recovery. The electrocardiographic details of the case are illustrated on figure 1.

![Fig. 1](image-url)

Male, 41 years. All Lead 2.
A. Before induction. Sinus rhythm 60/minute.
B. After thiopentone induction. Sinus rhythm 120/minute.
C. Trichlorethylene anaesthesia for fifteen minutes. Sinus rhythm 50/minute.
D. Ether added six minutes previously and respiration assisted. Patient apparently dead. Complete atrioventricular block with ventricular standstill.
F. Two minutes later. Sinus rhythm 60/minute.
G. Three minutes later.

By courtesy of the British Heart Journal (1951), 13, 47.

I cannot say whether a cardiac arrest induced in this manner can be permanent, but, when one reads the published reports of fatal arrests during anaesthesia, I feel that it is reasonable to assume that this mechanism was involved in some of them. It has been shown that small doses of pethidine (Johnstone, 1951b) and of morphine (Embley, 1902; Cohn, 1913) may increase the sensitivity of vagal reflexes and, thereby, predispose to cardiac arrest when irritant vapours are inhaled. The muscarinic action of neostigmine and of suxamethonium (Johnstone, 1955) may precipitate vagal cardiovascular collapse in sensitive patients unprotected by atropine.

This type of arrest should be regarded as an ever-present risk when anaesthesia is induced or maintained with ether, ethyl chloride, chloroform, cyclopropane, and trichlorethylene. It may also occur when the pulmonary branches of the vagus are stimulated by surgical manipulations (O'Shaugnessy, 1936; Edwards, 1938). The transition from normal pulse rate to complete arrest requires one or two minutes, depending on the intensity of stimulation. I suspect that cyanotic pallor and cessation of respiration are relatively late signs of cardiac arrest of this type. Normal cardiac function can be restored either by withdrawing the irritant vapour and ventilating with nitrous oxide and oxygen, or by administering atropine intravenously. Atropine—0.5 mg to 1.0 mg—will prevent or abolish this type of cardiac inhibition and is at present the only known antidote. Judging from the few cases of complete vagal arrest of the heart which I have observed during clinical anaesthesia I think that spontaneous recovery of the heart is possible after sixty seconds complete stoppage, providing the precipitating agent is promptly removed. Atropine, administered by intravenous injection, reaches its peak vagolytic effect in twenty or thirty seconds. During electrocardiographic observations on many hundreds of patients I have never seen any suggestion of active potentiation of vagal activity following the intravenous administration of the drug. I am not aware of any effort to determine whether other vagolytics, with the exception of gallamine triethiodide (Johnstone, 1955), are suitable substitutes for atropine in this respect. Pethidine, which is known to have a weak atropine-like effect in animals (Eisleb and Schaumann, 1939), provides no protection against reflex cardiac inhibition; it appears to increase the sensitivity of the pulmocardiac reflex by preventing bronchiolar constriction when the irritant vapours are inhaled (Johnstone, 1951b). The use of intravenous barbiturates and nitrous oxide mixtures for the induction and maintenance of anaesthesia completely eliminates the risk of this type of arrest.
during anaesthesia in all cases except those involving surgical stimulation of the vagus nerve and its pulmonary branches.

The Ventricular Tachycardia—Fibrillation Sequence.

This is the disturbance first observed by Oliver and Schafer (1895) and later confirmed electrocardiographically by Levy (1912). It has repeatedly been shown to be caused by overactivity of the sympathetic nervous system (Elliott, 1912; Bathia and Burn, 1933; Cattell, 1923; Allen et al., 1940), and occurs classically when an overdose of adrenaline or noradrenaline is administered to an anaesthetized subject. It is interesting to note that Snow (1858) drew attention to the fact that sudden and unexpected death was not an uncommon occurrence in the early stages of surgical operations in the pre-anaesthetic days; Levine (1952) observed that acute fear has precipitated ventricular tachycardia in a susceptible patient.

The induction of anaesthesia with agents such as chloroform, ether, cyclopropane, and trichlorethylene, which have repeatedly been shown to increase the secretion of adrenaline, is often associated with the appearance of severe cardiac arrhythmias, especially in nervous or excited patients. The cardiac disturbance consists of a rapidly progressive increase in the sinus rate, increase in the amplitudes of the P and T waves, the appearance of ventricular extrasystoles which gradually increase in frequency till they completely dominate the cardiac rhythm; in fatal cases the extrasystolic beats are soon followed by ventricular fibrillation. A similar sequence of events may be observed when carbon dioxide retention is allowed to occur during the maintenance of anaesthesia with cyclopropane, chloroform, trichlorethylene, thiopentone, and most other agents. More detailed descriptions of the aetiology and control of these arrhythmias have already been published (Johnstone, 1950 et seq.).

I have now come to regard the occurrence of extrasystolic beats during anaesthesia as an indication that ventilation, whether spontaneous or controlled, is inefficient and has permitted the retention of carbon dioxide; this applies to anaesthesia with all agents, including cyclopropane and trichlorethylene. The use of the thiopentone—nitrous oxide—oxygen—curare sequence does not in any way predispose to the more severe degrees of ventricular arrhythmias, though occasional extrasystoles and bigeminy may be observed when the elimination of carbon dioxide is inadequate. The only other circumstances in which I have observed these arrhythmias are during surgical manipulation of the heart and after the administration of sympathetic stimulants to anaesthetized patients.

I have been unable to observe that oxygen lack plays any part in their production, and reliable evidence seems to indicate that it may prevent their appearance (Harris, 1946; Zeigler, 1948). Levy (1913) observed that asphyxia and deep anaesthesia protected the animal heart from the effects of adrenaline administered during chloroform anaesthesia. He also noted that spontaneous cardiac irregularities were less likely to occur during chloroform anaesthesia when the systolic pressure was less than 100 mm Hg, or when artificial respiration was performed. After prolonged deep anaesthesia in man, or in the presence of surgical shock, carbon dioxide retention has not the same tendency to cause cardiac arrhythmias during cyclopropane or trichlorethylene anaesthesia. It is now well established that procaine will abolish ectopic cardiac rhythms through its depressant effect on the conducting mechanisms (M'Clendon et al., 1951). It would appear, therefore, that the appearance of ectopic disturbances during anaesthesia indicates the effects of excessive adrenaline on the intact Purkinje cells. When the activity of these cells is depressed by deep anaesthesia, procaine, vascular hypotension, or hypoxia, the irritability of the ectopic centres of impulse formation is correspondingly depressed. If the depression of these cells is excessive or prolonged, the sino-atrial and the atrioventricular nodes may cease to function. It is also possible, however, that the depression of cardiac irritability by the above mentioned factors may be related to suppression of the sympathetic and adrenal activity.

An important feature of the extrasystolic arrhythmias is that they can be abolished by stimulation of the vagal pulmocardiac reflex in the manner described in the previous section. The insufflation of ether vapour provides a convenient method for doing this and is most effective in dealing with the severe arrhythmias which
may be precipitated by the administration of adrenaline or noradrenaline during anaesthesia (Johnstone, 1953a). Conversely, in the presence of increased sympathetic activity, the sudden abolition of vagal function by vagolytic drugs such as atropine or gallamine may release the ectopic centres of impulse formation normally held in restraint by the inhibitory effect of the vagus (Levy, 1912; Johnstone, 1951a, 1955).

Recognition of the disturbance of cardiac rhythm due to sympathetic overactivity is difficult without the aid of an electrocardiograph. It is doubtful whether multifocal ventricular tachycardia can be accurately recognized by inspection of the exposed heart. Levy (1912) emphasized that multifocal ventricular tachycardia was indistinguishable from the coarse type of ventricular fibrillation except by electrocardiographic methods. This point should be carefully considered when assessing the merits of the treatment of cardiac derangements diagnosed by direct inspection of the heart. Ventricular tachycardia is certainly not an indication for cardiac massage as it can be remedied by more conservative means. Ventricular fibrillation, on the other hand, indicates a seriously damaged myocardium which may require assistance by massage.

Cardiac Disturbances due to Electrolytic Imbalance.

Since the estimation of the blood electrolytes was facilitated by the introduction of the flame photometer (Barnes et al., 1945) it has become more obvious that the electrolytes play an important part in the maintenance of cardiovascular efficiency during anaesthesia and surgery. The sodium, potassium, and chloride ions have received most of the attention, and of these potassium is the most interesting from the anaesthetist’s point of view. It is now generally accepted that potassium excess (Marchand and Finch, 1944) or potassium deficiency (Lewis et al., 1954; McAllan, 1955) may lead to severe cardiovascular collapse. The effects of anaesthesia on the patient with potassium deficiency have not yet been clearly defined. There are, undoubtedly, aspects of potassium deficiency which are of much importance in relation to relaxant drugs and somatic myoneural function; this matter is at present under consideration and the evidence is incomplete. Potassium intoxication is encountered less frequently than deficiency but is liable to have more serious consequences.

The potassium ion is normally present in relatively high proportions in the intracellular spaces. Under certain conditions, such as metabolic acidosis and glycojenolysis (Mudge, 1953; Keating et al., 1953), respiratory acidosis (Cattell and Civin, 1938; Abrams et al., 1951), or after the administration of adrenaline (D’Silva, 1936; Laonson and Greig, 1953), it moves from the intracellular to the extracellular spaces with the result that the level in the plasma rises. A similar rise may follow excessive intake of potassium. In the presence of any renal function the rise in plasma potassium is dealt with immediately by an increased excretion of potassium in the urine. When kidney function has been stopped, however, by conditions such as calculus anuria, obstruction of both ureters, pyelonephritis, glomerulonephritis, or severe systemic infections, a progressive rise occurs in the level of the plasma potassium and results in ventricular fibrillation. The sequence of electrocardiographic changes associated with potassium intoxication is diagnostic of the condition (Hoff et al., 1941), consisting of:

1. Increase in the amplitude of the T wave which loses its normal rounded contour and becomes pointed.
2. The S wave becomes deeper and wider.
3. The P wave disappears.
4. The widening of the ventricular deflection progresses gradually to complete disorganization of the complex with the appearance of ventricular fibrillation.

Several of these changes are illustrated in the following case.

**Case 2**

Male, 39 years. Oliguria for 14 days, with anuria for the previous 48 hours. Six months history of backache. Heart, lungs, and abdomen appear normal. Blood pressure 150/90 mm Hg, blood urea 247 mg per cent, haemoglobin 9 g per cent. For cystoscopy and retrograde catheterization of the ureters. Premedication atropine 0.5 mg. General anaesthesia considered suitable because of some restlessness and mental confusion. An electrocardiogram taken immediately before induction revealed signs suggestive of potassium intoxication of the heart (fig. 2, A).

Anaesthesia was induced with thiopentone 200 mg injected intravenously, followed by suxamethonium 50 mg as soon as the patient was asleep. Apnoea followed the injection of these drugs. After about fifteen
seconds of complete apnoea it was noticed that the P waves of the cardiogram had disappeared, the QRS became wider and deeper, and the amplitude of the T wave increased (fig. 2, c). An endotracheal tube was promptly inserted and the patient efficiently ventilated with nitrous oxide and oxygen; the cardiogram returned to its pre-operative appearance within thirty seconds (fig. 2, d). Brisk spontaneous respiration returned in two or three minutes and both ureters were successfully catheterized in twenty minutes. During this time a further 100 mg of thiopentone and 50 mg of suxamethonium were administered intravenously without causing any electrocardiographic changes.

At the end of the operation it was decided to administer some carbon dioxide to augment the respiratory movements which were depressed by overventilation. Carbon dioxide 20 per cent in oxygen was given for thirty seconds and caused a brisk increase in the respiratory movements. Coincident with the onset of respiratory stimulation, changes occurred in the cardiogram similar to those seen after induction. These changes progressed rapidly to what appeared to be ventricular flutter or fibrillation (fig. 2, h), in spite of efficient ventilation. At times it was thought that a very feeble pulse could be felt in the carotid arteries, though none could be felt in the radial artery. Respiration remained brisk, rather like that associated with hypoxic stimulation of the respiratory centre. Ten minutes later the patient became restless and removed his endotracheal tube. A blood sample, taken immediately after the collapse at the end of the operation, later revealed the serum potassium to be 9.35 m equiv./litre (36 mg per cent).

After twenty minutes, during which time his respiration was brisk and the cardiovascular system unchanged, 4 ml of 1 per cent procaine hydrochloride were injected intravenously; this caused further deterioration of the cardiogram (fig. 2, i), the peripheral pulse disappeared completely, and restlessness ceased. Fifteen minutes later there was a slight improvement in the cardiogram (fig. 2, j) and some restlessness returned. One hour later (fig. 2, k) there was no appreciable change in the patient's condition and it was decided to give procaine amide. 500 mg were injected intravenously over a period of five minutes and caused deterioration similar to that caused by procaine hydrochloride (fig. 2, l). Thirty minutes later 100 ml of 50 per cent glucose solution with 50 units of insulin were injected intravenously and caused little change in the patient's condition (fig. 2, m). One and a half hours later (fig. 2, n), when it was felt that the patient was dying, it was decided to repeat the glucose-insulin therapy and a similar dose was again administered intravenously. Within five minutes of the completion of the injection, which was four hours after the onset of the collapse, a sudden and dramatic improve-

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M. Ten minutes later, after the first glucose-insulin therapy.
N. Four hours after the induction of anaesthesia, and immediately before the second glucose-insulin therapy.
O. Five minutes later.
P. Ten minutes later.
Q. Three days later, serum potassium within normal limits.

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Male, 39 years. All tracings, except A, are Lead 2.

A. Before induction. Leads 1, 2, and 3. Large tented T2 and T3.
B. After thiopentone induction.
C. After suxamethonium and fifteen seconds apnoea.
D. Thirty seconds later, efficiently ventilated.
E. After twenty minutes anaesthesia.
F. Thirty seconds later, carbon dioxide started.
G. One minute later.
H. Five minutes later, immediately before the injection of procaine hydrochloride.
I. Two minutes after the procaine injection.
J. Fifteen minutes later.
K. Two hours after the induction of anaesthesia.
L. Two minutes later, after the injection of procaine amide.
M. Ten minutes later, after the first glucose-insulin therapy.
N. Four hours after the induction of anaesthesia, and immediately before the second glucose-insulin therapy.
O. Five minutes later.
P. Ten minutes later.
Q. Three days later, serum potassium within normal limits.

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Fig. 2

[Diagram showing various heart rate and wave patterns]
ment occurred in both the clinical and electrocardiographic states of the patient: he became fully conscious and the cardiogram reverted to its pre-operative appearance (fig. 2, p); the serum potassium at this time had dropped to 8.05 m equiv./litre (31 mg per cent). The improvement was maintained and ion-exchange resins restored the serum potassium to normal levels in three days (fig. 2, q).

The patient passed urine normally for a few days after the catheterization of the ureters, his blood chemistry and cardiovascular system becoming quite normal. Anuria then returned, apparently due to obstruction of the ureters by a retroperitoneal tumor. Bilateral nephrostomies were made, one under spinal anaesthesia and one under general anaesthesia, without complication. One month later the presence of an inoperable retroperitoneal tumor was confirmed by laparotomy; this operation was performed under general anaesthesia and was not associated with any cardiovascular disturbance as his plasma potassium was within normal limits. At the present time, which is over twelve months after the original cystoscopy, the patient is active and ambulant.

This case illustrates several interesting points. Firstly, it shows how the cardiac effects of potassium intoxication can be dangerously intensified by respiratory acidosis induced by endogenous or by exogenous carbon dioxide. Secondly, the effectiveness of the glucose-insulin therapy of potassium intoxication (Seldin and Tarail, 1949; Merrill et al., 1950) has been demonstrated. Thirdly, it illustrates the need for accurate observation of the sequence of events which precede the onset of serious electocardiographic disturbances during anaesthesia. Inspection of tracings figure 2, c and k, might give the impression that a ventricular tachycardia was present; however, having seen on the oscilloscope that the abnormality was preceded by a progressive widening of the QRS complex associated with a deepening of the S wave, elevation of the T wave, and disappearance of the P wave, one can better describe the cardiogram as illustrating a diffuse intraventricular block: this is a condition fundamentally different from ventricular tachycardia.

It is generally accepted that ventricular tachycardia, as it occurs during anaesthesia, is due to a hyperexcitability of the Purkinje network and is not in any way related to blockade or depression of the specialized tissue such as occurs in potassium intoxication (Winkler et al., 1938; Winkler and Hoff, 1943). The onset of ventricular tachycardia is preceded by a characteristic sequence of changes: the amplitude of the P wave increases, the QRS and PR intervals generally become smaller, isolated ventricular extrasystoles then appear, followed rapidly by bigeminy (coupling), and eventually all the sinus beats are replaced by extrasystoles; the arrhythmia can be abolished at any of these stages either by eliminating carbon dioxide more efficiently, or by vagal stimulation in the manner described above.

Realizing that the cardiac changes associated with potassium intoxication are due to impairment of conduction in the Purkinje network, it is obviously illogical and dangerous to treat with procaine hydrochloride or amide which can only increase the degree of block still further, as shown in this case.

**Spontaneous Coronary Thrombosis during Anaesthesia.**

Obliterative disease of the coronary arteries is becoming an increasingly more frequent finding in pre-operative examinations. Its precise relation to anaesthetic drugs and techniques has not been defined. It is obvious, however, that patients with this disease usually survive major surgical and anaesthetic procedures without myocardial deterioration. The following case history, which describes the only example of acute coronary thrombosis I have encountered during anaesthesia, illustrates an important point in the anaesthetic management of such patients.

**Case 3**

Male, 63 years. Severe angina of effort. No oedema or cyanosis. Lungs normal. Blood pressure 140/100 mm Hg. Left stellate ganglionectomy twelve months previously with transient relief from pain. For thoracic cardiac neurectomy. Premedication atropine 0.5 mg with pethidine 50 mg administered subcutaneously one hour before operation. Quite calm and without pain or discomfort on arrival at theatre. Blood pressure and cardiogram unchanged (fig. 3, A). Anaesthesia was induced with 250 mg of thiopentone injected intravenously, followed by suxamethonium 50 mg and endotracheal intubation. There were no significant changes in the cardiogram following induction and intubation (fig. 3, B). Three minutes later the effects of suxamethonium disappeared and the patient began to cough and strain on the endotracheal tube. An attempt to control the coughing and straining with a further dose of relaxant was delayed for about three minutes because of considerable difficulty in finding a suitable vein. Thirty seconds after the onset of the coughing the QRS complex of Lead 2 became smaller and the ST segment elevated (fig. 3, C); two minutes later the rate dropped to 50 beats a minute and the ST shift became more pronounced, the pulse being imperceptible at the wrist. During the next forty minutes the ventricular complex became wider, the rate slowed, and the heart eventually stopped (fig. 3, D, E, F). Cardiac
massage was performed in the early stages of the collapse but was soon abandoned as the coronary arteries felt hard and brittle.

The changes which occurred in Lead 1 in this case are interesting and might possibly prove misleading if too much emphasis were to be placed on the examination of isolated single-lead cardiograms. Prior to induction it shows a sinus rhythm with an inverted T wave. No significant changes occurred during induction and intubation. During the period of coughing, however, the T wave became erect to the normal position (fig. 3, C) and the tracing appeared to be improved although the patient was in extremis. This type of change illustrates the importance of having serial tracings in at least two leads when a collapse is anticipated or is being investigated. Errors of diagnosis may occur when isolated single-lead tracings are taken after the collapse has supervened.

Myocardial Ischaemia during Mitral Valvotomy.

During the operation of mitral valvotomy the cardiogram often shows signs of myocardial ischaemia for a short time immediately after the removal of the surgeon's finger from the valve orifice. Whilst the commissures of the valve are being split, a multifocal ventricular rhythm almost invariably replaces the normal supraventricular complexes (fig. 4, F, G, H); this arrhythmia appears to be due to tactile stimulation or stretching of Purkinje fibres, as its onset coincides exactly with the insertion of the finger into the valve ring and it subsides immediately after the finger is withdrawn into the cavity of the auricle—it is not necessary to withdraw the finger from the auricle to abolish the arrhythmia. When the supraventricular complexes reappear they generally show a decrease in rate and displacement of the ST segment (fig. 4, F, G, H). With each successive attempt at splitting the commissures the rate becomes slower and the ST segment more displaced until the degree of displacement may be extreme (fig. 4, i); at this stage it is impossible to feel a peripheral pulse. I think that this change in the position of the ST segment indicates myocardial ischaemia secondary to vascular obstruction at the mitral valve. The ST segment resumes its normal iso-electric position within one or two minutes after the withdrawal of the finger from the valve orifice (fig. 4, J, K, L, M). It is therefore advisable to observe the behaviour of the ST segment when several attempts at valve splitting are required. A severe degree of shift indicates the need for a pause to permit the return of the coronary circulation; neglect to observe these changes may lead to myocardial anoxia and cardiac arrest.
Accidental Coronary Occlusion during Cardiac Surgery.

The intimate relation of the left coronary artery to the base of the left auricular appendage is well known. This artery, and particularly its circumflex branch, is liable to compression between the anterior wall of the left ventricle and the handle of the clamp which is applied to the auricular appendage immediately before the insertion and immediately after the withdrawal of the surgeon's finger. When the clamp is reapplied after the valve has been split, the blood pressure is usually very low and the lumen of the coronary artery can be easily obliterated. A more complete description of the mechanics of this type of coronary occlusion has recently been published by Mudd et al. (1954).

Occlusion of a large branch of a coronary artery naturally leads to a sudden and profound collapse of the patient with a characteristic sequence of electrocardiograph changes. These changes are illustrated in the following case.

**CASE 4**

Female, 26 years, with mitral stenosis. For valvotomy. Premedication atropine 0.5 mg, with pethidine 50 mg. Anaesthesia induced with 200 mg of thiopeptone injected intravenously, intubated during suxamethonium paralysis, and maintained with nitrous oxide, oxygen, and full curarization. The patient's condition remained very satisfactory during the preliminary stages of the operation and also whilst the valve was being split (fig. 5, A–F). Immediately after the reaplication of the auricular clamp to facilitate closure of the auricular appendage, the patient became pulseless, pallid, and faintly cyanotic; direct inspection of the heart revealed a slow and very weak contraction. Coincident to the onset of the collapse there occurred a sudden elevation of the ST segment and the sinus rate became slower (fig. 5, G,H,I). The auricular suture was rapidly completed and the clamp removed; this was followed by a dramatic improvement in the patient's condition both clinically and electrocardiographically (fig. 5, J, K). Recovery was uneventful.

H. Third attempt at valve splitting. Again a multifocal ventricular tachycardia gives way to a sinus rhythm when the finger is withdrawn. The sinus rate is now slower, the T wave deeply inverted, and the displacement of the ST segment is increasing rapidly.

I. End of fourth and successful attempt at valve splitting. There is now an extreme degree of ST shift.

J. Three minutes later, auricular sutures being inserted. The ST shift is less.

K. Five minutes later.

L. Ten minutes later.

M. Fifteen minutes later. The tracing has now reverted to its pre-operative appearance.

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A. Before induction.
B. Twenty minutes later, chest open under nitrous oxide, oxygen, and full curarization.
C. Auricular clamp applied. Sinus rhythm interrupted by auricular extrasystoles.
D. First attempt at valve splitting. Shows multifocal ventricular tachycardia giving way to sinus rhythm when the finger is withdrawn from the valve.
E. Second and successful attempt at valve splitting. Tracing again shows a ventricular rhythm giving way to a sinus rhythm when the finger is withdrawn from the valve. The T wave is now diaphasic and the ST segment displaced.
F. One minute later. ST segment in normal position.
G. Thirty seconds later, auricular clamp in situ. The S wave has suddenly become wider and deeper.
H. Lead I. Ten seconds later. Note the extreme elevation of the ST segment.
I. Lead I. Fifteen seconds later. Patient apparently moribund.
J. Lead 2. Thirty seconds later, after the removal of the auricular clamp. ST segment and T wave normal.
K. Lead 2. Ten minutes later. Patient's condition now satisfactory.

Fig. 5
Female, 26 years. Mitral valvotomy.
All tracings, except H and I, are Lead 2.

DISCUSSION
These notes describe six sets of circumstances which may lead to complete cardiac arrest. They depict cardiac collapse due to vagal overactivity, to sympathetic overactivity, to potassium intoxication, and to acute coronary insufficiency induced in three different ways. Each type of collapse was preceded by characteristic electrocardiographic changes which provided good warning for the institution of specific resuscitative measures. In addition to these mechanisms there are others which have not been fully described: those due to asphyxia, acute haemorrhage, acute adrenocortical insufficiency (Lewis et al., 1953; Galante et al., 1954; Root, 1955), hypothermic fibrillation (McWilliam, 1887), obstruction of the pulmonary artery (Masson and Branwood, 1955), and others not yet identified. It is not improbable that each of these conditions in the early stages may cause a characteristic sequence of electrocardiographic changes calling for specific remedies.

In extreme cases of cardiovascular collapse the electrocardiograph may provide the only evidence of life and may save the patient from unnecessary cardiac massage, as the following case illustrates.

Case 5
Female, 32 years. Excellent condition, with normal cardiovascular and respiratory systems. For dilatation of the cervix and curettage of the uterus. Morphia 15 mg with atropine 0.5 mg administered subcutaneously ten minutes before the induction of anaesthesia. The operation was completed uneventfully in ten minutes under thiopentone 300 mg with gallamine 40 mg injected intravenously, respiration being spontaneous throughout. On leaving the theatre the patient was asleep and in good condition. Ten minutes later, whilst awaiting transport back to the ward, she was found to be apparently dead: the face showed a waxy-cyanotic pallor, the pupils were dilated and the corneal reflex absent, there was no evidence of spontaneous respiration, no pulsation could be felt in either the radial or carotid arteries, and the heart sounds could not be heard. An electrocardiograph was attached immediately and, at the same time, a tightly fitting endotracheal tube was inserted and the lungs briskly inflated with oxygen; the pulmonary inflation was designed not only to oxygenate the lungs but rhythmically to compress the heart. The first tracing was obtained about thirty seconds after the start of pulmonary inflation and shows only one aberrant ventricular complex (fig. 6 A). Ten seconds later the aberrant beats had increased in frequency (fig. 6, B), and one and a half minutes later a bigeminal rhythm had appeared and a slow pulsation became palpable in the carotid artery (fig. 6, C). Ten minutes later the cardiovascular system was quite normal apart from a slight degree of partial atrioventricular block.
Artificial respiration was continued for a further fifty minutes before spontaneous respiration returned. Two hours later, the patient was conscious and rational. No evidence of neurological sequelae was detected and recovery was uneventful.

I feel reasonably certain that virtually complete cardiac arrest had occurred in this patient as the result of asphyxia. The response to brisk pulmonary inflation was dramatic and provided clinical support for the experimental observation of Thompson et al. (1946) who, with the aid of radio-active sodium ions in the heparinized blood of dead dogs, demonstrated that pulmonary inflation produced a distinct circulation of the blood of the dead animals. I have also observed dramatic recoveries in a few children who collapsed and became pulseless after periods of intense laryngeal spasm during the induction or recovery from inhalational anaesthesia.

Cardiac massage has been recommended as the primary treatment of circulatory collapse when the peripheral pulse ceases to be palpable (Milstein and Brock, 1954; Rao et al., 1954; Mullens, 1955). It is obvious that such treatment may be quite commendable and is easily performed on patients prepared for thoracic surgery. In other branches of surgery, however, the decision to perform this treatment is a most harrowing experience for all concerned and must inevitably be associated with delay in a proportion of cases, resulting, at best, in patients with crippling neurological sequelae. The instantaneously recording electrocardiograph provides the answer to many of these problems.

I cannot accept that inability to detect pulsation in a large artery is invariably an indication for prompt cardiac massage. On several occasions, whilst using the “total spinal” technique (Griffiths and Gillies, 1948) for major pelvic surgery, I have been unable, for various reasons, to feel any pulsation in the peripheral arteries; in all these cases the cardiogram has remained unaltered in the three limb leads and the patients have made uneventful recoveries without the need of resuscitation. On one occasion, whilst performing an abdominoperineal resection of the rectum under a “total spinal”, the surgeon was unable to feel pulsation in the iliac arteries; in spite of this the cardiogram remained perfectly normal, the operation was completed without the need of supportive therapy, and the patient made an uneventful recovery.

Traction or pressure in the region of the coeliac plexus in some patients causes intense peripheral vasoconstriction with complete disappearance of the arterial pulse but persistence of the heart beat. Similarly, the use of excessively high or sustained intra-alveolar gas pressures during the ventilation of curarized patients may render them pulseless, yet the heart continues to beat and the cardiogram remains unaltered for a time. A further cause of pulselessness during operation was recently encountered and provides evidence of the value of continuous electrocardiography during anaesthesia.

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**Fig. 6**

Female, 32 years. Curettage of the uterus. All Lead 2.

A. Thirty seconds after the start of pulmonary inflation. Shows only one aberrant ventricular beat.
B. Ten seconds later. Aberrant beats increase in frequency.
C. After one and a half minutes of brisk pulmonary ventilation. Atrioventricular dissociation with ventricular extrasystoles coupled to the AV nodal beats. Feeble carotid pulse felt, 30 per minute.
D. Five minutes later, pulse palpable at wrist, 44 beats a minute. Atrioventricular dissociation.
E. Fifteen minutes later. Sinus rhythm, 47 beats a minute. Slight degree of atrioventricular block.
SOME MECHANISMS OF CARDIAC ARREST DURING ANAESTHESIA

Case 6

Middle-aged, thin female, for exploratory laparotomy. Known to have a malignant carcinoid of the intestine with secondaries in the liver. Severe vaso-motor complications, of the type described in detail by Thorson et al. (1954), occurred periodically as the result of a toxin (enteramine or serotonin) secreted by the tumours. During these attacks she developed a patchy cyanosis, orthopnoea of the bronchial-asthmatic type, and the peripheral arteries became quite pulseless; consciousness and the cardiogram remained relatively unaltered. Anaesthesia was induced with thiopentone 200 mg, suxamethonium 50 mg, and continued with nitrous oxide and oxygen through an orotracheal tube with controlled respiration using 15 mg of d-tubocurarine. A severe vasomotor attack commenced five minutes after induction and persisted throughout the operation which lasted forty-five minutes. During this time the patient was quite pulseless, the limbs cold and patchily cyanosed, and the lungs difficult to inflate; apart from occasional diaphragmatic twitches, the only sign of life available to the anaesthetist was the cardiogram which remained significantly unaltered. Anaesthesia was induced with thiopentone 200 mg, suxamethonium 50 mg, and continued with nitrous oxide and oxygen through an orotracheal tube with controlled respiration using 15 mg of d-tubocurarine. A severe vasomotor attack commenced five minutes after induction and persisted throughout the operation which lasted forty-five minutes. During this time the patient was quite pulseless, the limbs cold and patchily cyanosed, and the lungs difficult to inflate; apart from occasional diaphragmatic twitches, the only sign of life available to the anaesthetist was the cardiogram which remained significantly unaltered throughout the operation. The peripheral circulation returned as the skin sutures were being inserted and the recovery of consciousness and muscle power were not delayed.

Several authorities have suggested that the electrocardiographic monitoring of the heart during clinical anaesthesia should be the responsibility of the cardiologist. Several important papers have been published by cardiologists, particularly in relation to cardiac surgery (Hill, 1932; Zeigler, 1948; Campbell and Reynolds, 1952; Jaruszewski et al., 1953; Campbell and Reynolds, 1954; Milstein and Brock, 1954). These reports indicate that severe cardiac disturbances often occur during anaesthesia but in most instances the observers were unable to define the precise factors which were causing the disorders of cardiac function. Writing as an anaesthetist, I should like to emphasize that the majority of cardiac disturbances, which I have described or referred to (Johnstone, 1950 et seq.) are due not so much to the anaesthetic agents, as to the manner in which they are used; and they are usually amenable to a physical modification of the anaesthetic technique. The accurate diagnosis of a cardiac abnormality occurring in an anaesthetized patient is based on inspection of the cardiogram combined with a detailed assessment of the clinical details of the anaesthesia—the method of ventilation, the reaction to inflation, the resistance of the air passages, the autonomic reactions to the various drugs and surgical manipulations, the fluctuations of blood pressure and pulse rate, and numerous other details with which the experienced anaesthetist is familiar.

It would appear, therefore, that the final solution to the clinical complexities of cardiovascular collapse during anaesthesia and surgery will be provided by cardiologists who are skilled anaesthetists, or by anaesthetists who are familiar with the behaviour of the human heart as depicted on the cardiogram. It is obvious that there are several different mechanisms of cardiac arrest and it is hoped that eventually we may be able clearly to define the circumstances under which they may occur and to take steps to counteract them successfully. As the number of complex drugs and techniques increases, the greater becomes the need for detailed observation of cardiovascular function during anaesthesia. It is not sufficient that this type of research should be confined to research centres for the simple reason that each anaesthetist, fortunately, encounters a relatively small proportion of the cardiovascular collapses which actually occur.

After several years experience with the electrocardiograph during clinical anaesthesia I am now quite convinced that the accurate determination of the aetiology of a cardiovascular collapse or arrest is virtually impossible in the absence of electrocardiographic records. There is a certain amount of truth in the statement that the cardiograph may record a complex when the patient is already dead. It has been my experience, however, that a tracing obtained in these circumstances shows abnormal features in at least one of the limb leads. When serial tracings are obtained before the onset of the collapse, a characteristic sequence of changes will be recognizable, from which it may be possible to determine the resuscitative measures most likely to benefit the patient. I do not wish to suggest that it is impossible to administer a safe anaesthetic without the aid of an electrocardiograph: experience always teaches us instinctively to avoid numerous pitfalls. Careful pulse palpation combined with a knowledge of pharmacology should prevent such complications as vagal arrest or ventricular tachycardia, and perhaps even the potassium intoxication syndrome. In the management of dangerously ill patients, and in the assessment of new techniques, the electrocardiograph provides information which is un-
obtainable by any other means. It is an instrument which is easily available to every anaesthetist and, quite apart from its research potential, it often provides much-needed reassurance when dealing with the critically ill patient.

SUMMARY

(1) Six different mechanisms of cardiovascular collapse during clinical anaesthesia have been described. It has been shown that each of them is associated with a characteristic sequence of electrocardiographic changes. They include collapse due to vagal overactivity, to sympathetic overactivity, to potassium intoxication, and to acute coronary insufficiency induced in three different ways.

(2) The prevention and treatment of each of these mechanisms have been described.

(3) The importance of the cardiogram in the diagnosis and treatment of cardiovascular collapse during anaesthesia has been emphasized. It is suggested that the cardiological management of the patient during anaesthesia should be the responsibility of the anaesthetist.

(4) It is claimed that pulselessness is not always an indication for immediate cardiac massage. Some of the circumstances in which the anaesthetized patient may become pulseless have been discussed.

(5) The anaesthetic management of a case of malignant carcinoid secreting "serotonin" has been described.

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SOME MECHANISMS OF CARDIAC ARREST DURING ANAESTHESIA


BOOK REVIEW

*The Medical Annual*. Edited by Sir Henry Tidy and R. Milnes Walker. Published by John Wright and Sons Ltd., Bristol. Price 32s. 6d.

The 404 pages of ten years ago now extend to 548.

The short section on anaesthesia and analgesia is again written by Dr. Langton Hewer. He once again emphasizes the importance of keeping an open airway, advice one would have supposed unnecessary but for the illustration on the previous page which shows in how many different ways it can be obstructed by the very means taken to ensure it. In these days of long operations and mechanical devices, the monotony of his task may render the anaesthetist temporarily oblivious of his surroundings. If this be so the “Drip alarm” figured on page 30 will no doubt provide a rude awakening and a quick recall to a lively sense of his responsibilities.

Chlorpromazine has a long paragraph to itself.

Dr. Hewer refers to the well-known case of Woolley and Roe v. The Ministry of Health and Others. He says: “It has now been established in law that ampoules of analgesic solutions stored in antiseptic fluid can become contaminated even if no crack is visible.” It is important for anaesthetists to remember this, but for the sake of truth they must also remember that it has not been established in fact. The trial decided that there was no evidence of negligence. It did not discover the cause of the damage. One witness suggested that it was the anaesthetic itself—the light novocain—that had caused the mischief; another that it was the 1 in 40 carbolic in which the ampoules were stored. It is extremely doubtful if either of these statements will stand the test of time. What we do know is that alcohol will cause such damages as were sustained but there was no evidence at the trial to this effect.

A short paragraph on analgesic drugs by Dr. Andrew Wilson concludes this section on the subject.

Dr. Wilson later on in the *Annual* gives an account of the varying views on Nalorphine and Professor Anderson contributes an article on the use of chlorpromazine in psychiatry which also may be of interest to anaesthetists.

E. Falkner Hill