ASPECTS OF THE MANAGEMENT OF CARDIAC ARREST

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The management of cardiac arrest has been well reviewed by Milstein[1] and Katz.[2] The purpose of this paper is to present 3 cases of cardiac arrest, and to discuss some points of interest in their treatment.

Case 1

Cardiac arrest occurred in a man aged 63 shortly after the induction of anaesthesia with 200 mg. of thiopentone and 25 mg. of succinylcholine.

The patient was inflated with oxygen *via* an endotracheal tube which was already in position, and the chest was opened within 2 minutes of the arrest. The heart appeared to be in asystole; so after cardiac massage for 3 minutes, 0·5 c.c. of 1 : 1,000 adrenaline diluted to 5 c.c. was injected into the left ventricle. This produced no evident change in the appearance in the heart, but when the pericardium was opened a fine fibrillation was observed which had not been seen through the pericardium. Massage was continued and after a further 5 minutes 2 c.c. of 1 : 10,000 nor-adrenaline was injected into the ventricle, and an intravenous nor-adrenaline infusion started. This was followed by some powerful but irregular contractions for a short time, after which ventricular fibrillation continued. After 40 minutes had elapsed from the time of cardiac arrest 4 c.c. of 2% lignocaine was injected into the left ventricle. The heart immediately became flaccid and unresponsive to any further attempts at resuscitation. A post-mortem examination showed a previous infarct and diffuse myocardial fibrosis.

Case 2

Cardiac arrest, due to an obstructed airway, occurred approximately 20 minutes after the end of an operation in a previously healthy young woman. The patient was wheeled back into the theatre and an endotracheal intubation attempted. This proved difficult and delayed the oxygenation of the lungs by a minute. The chest was opened approximately 6-8 minutes after the arrest. The heart was in asystole and flabby. Massage produced no improvement in tone and after 3 minutes, 0·5 c.c. of 1 : 1,000 adrenaline diluted to 5 c.c. was injected into the left ventricle. Fibrillation immediately followed. Massage was continued and 2 c.c. of 1 : 10,000 nor-adrenaline was then injected into the left ventricle. Some strong irregular beats then occurred, but ventricular fibrillation soon ensued and further efforts at resuscitation were unsuccessful.

Case 3

Cardiac arrest occurred shortly after the administration of 0·45 g. of thiopentone to a woman aged 32. There was a delay in the diagnosis of cardiac arrest, perhaps 2-3 minutes; the lungs were inflated with oxygen at the time. The chest was opened approximately 4 minutes after the arrest, and a large pericardial effusion was found. The heart was in asystole. After massage had been carried out for 3 minutes, 0·5 c.c. of 1 : 1,000 adrenaline was injected into the left ventricle. Fibrillation immediately started. After a further 3 minutes, 7 c.c. of 10% calcium gluconate was injected into the left ventricle. A strong and regular heart beat was the result. This patient was unconscious for 24 hours after the arrest and thereafter made a complete physical recovery, but she would have been permanently mentally defective had she not succumbed to a large pulmonary embolus 10 days after the operation.

THE MANAGEMENT OF CARDIAC ARREST

A routine procedure for the management of cardiac arrest which is simple and suitable for use in any operating theatre, will now be described under the following headings: (1) The diagnosis of cardiac arrest, (2) cardiac massage and pulmonary ventilation and (3) the use of a defibrillator if fibrillation is present, or the use of calcium gluconate if asystole is present.

A more complicated routine resembling a dramatic play is unnecessary, for the success of cardiac resuscitation depends

on the efficiency with which sections (1), (2) and (3) of the above-mentioned procedure are carried out.

Diagnosis of Cardiac Arrest

The diagnosis of cardiac arrest must be made very promptly for irreversible damage to the brain will occur unless an effective circulation is re-established within 3-4 minutes. Thus in case 3, although cardiac massage was successful in re-starting the heart, the patient survived the catastrophe as a mental cripple.

In recent years a number of useful electrical amplifiers have been produced which record the heart beat or pulse wave continuously so that the anaesthetist is warned when cardiac arrest occurs. Almost as effective is a long anaesthetist's stethoscope, the diaphragm of which can be fixed to the chest over the apex beat or, more conveniently in babies, placed under the supine patient. The heart beat can be auscultated continuously or as required, even when peripheral pulses are difficult to palpate and, if a 2-way cock is fitted to the stethoscope, another diaphragm can be fitted to the anticubital fossa so that blood pressures or heart beats may be noted without inconvenience. In case 1, cardiac arrest was diagnosed when it occurred because a stethoscope was fixed in position, while in case 3 no stethoscope was present in the theatre and the diagnosis was delayed. A continuous electrocardiographic record is the most effective way to diagnose any abnormality in the rate, rhythm or function of the heart which may occur during operation, but the use of the electrocardiogram for every surgical procedure is impracticable.

Cardiac Massage and Pulmonary Ventilation

Although pulmonary ventilation is ineffectual without a pumping heart, this subject will be mentioned first. Myocardial anoxia is the most frequent cause of cardiac arrest, so that when arrest occurs the anaesthetist promptly initiates adequate pulmonary ventilation with a high percentage of oxygen. It is often a quicker procedure to inflate the lungs initially with oxygen with a face mask until a suitable endotracheal tube and connections are selected. In case 2, initial oxygenation was delayed by an effort to insert an endotracheal tube.

Once cardiac arrest has been diagnosed the onus of initiating cardiac resuscitation rests with the medical practitioner present, and the procedures must be started immediately and should not be delayed to await the arrival of more skilled personnel.

If the abdomen is open at the time of arrest the heart can be massaged by squeezing the ventricle between the hand and the sternum from below the diaphragm. In children the method of resuscitation described by Rainer and Bullough,[3] can be tried. The knees are flexed and pressed onto the chest and alternately the legs are extended while pulmonary inflation with oxygen is carried out. I have used this method successfully on one occasion.

If either of these methods is not successful within 15-20 seconds, a transpleural approach is essential for effective massage and observation of the heart. An incision is made in the 4th intercostal space extending from the mid-axillary line to the sternum. A costal cartilage is divided and the ribs are spread by an assistant or by a rib-spreader. Unless this wide incision is made, a hand, or when necessary 2 hands, cannot be introduced into the thoracic cavity in a manner which will allow efficient cardiac massage. A valid generalization would be to say that on many occasions neither is the exposure adequate nor the massage effective.

If the legs are raised at the commencement of the massage, an increased venous return of blood will be made available at the crucial time, or alternatively a slight head-down posture can be instituted when the cardiac arrest is diagnosed. The heart should be compressed at the rate of 50-60 times, per minute between the palmar surfaces of the fingers and the thenar eminences in a manner which will produce a palpable peripheral pulse wave. In this way the cerebral circulation can be maintained and the necessary re-oxygenation of the myocardium can be achieved, which will often take several minutes. Until the myocardium presents a healthy pink appearance, the resumption of normal activity either spontaneously or following electrical or chemical stimuli will rarely occur.

The Treatment of Ventricular Fibrillation and Asystole

If after massage lasting several minutes the heart beat has not returned spontaneously, and if the examination of the heart through an opened pericardium shows that asystole is present, 5-10 c.c. of 10% calcium gluconate, or half this quantity of 10% calcium chloride, may be injected into the left ventricle. This will often initiate spontaneous contractions. If, however, ventricular fibrillation is present, defibrillation should be carried out. An electrical current of 110 volts at 1-3 amperes is applied to the heart muscle in the form of single or repetitive shocks lasting 0·1 second, by 2 large electrodes wrapped in cloth soaked in saline. The myocardial fibres are thrown into a refractory state simultaneously and remain quiescent for a short period. The sino-atrial node, because it usually recovers its excitability most rapidly, once again emits a regular impulse and initiates sinus rhythm. If defibrillation by single or repetitive shocks is unsuccessful, the injection of calcium gluconate should be tried. Electrical defibrillation is acknowledged as a good and effective treatment for ventricular fibrillation, but a defibrillator is often not available when required.

When the ventricle starts to fibrillate the muscle undergoes coarse uncoordinated contractions which are visible and palpable through the pericardium. Before this event the heart muscle has usually been exposed to a period of anoxia which has caused damage to the muscle cells and, therefore, to their membranous covering. This membrane has now become more permeable and begun to leak negative charges through its surface at various points. These areas become ectopic foci and initiate action potentials and consequently waves of muscular contractions, which pursue devious paths around the ventricles, following routes where the muscle has come out of its refractory period. There is no coordinated muscular contraction and no perfusion of blood through the coronary arteries; consequently a profound anoxia rapidly ensues until the whole muscle membrane is damaged and undergoes widespread repetitive firing. This causes localized weak muscular contractions seen as ripples along the surface of the muscle, and coarse fibrillation changes into fine fibrillation.

The basic aim in cardiac resuscitation, whether fibrillation or asystole exists, is to oxygenate the myocardium by pulmonary ventilation and cardiac massage, and to allow time for the muscle membrane to re-establish its selective permeability to ions. When this is achieved and a resting membrane

potential is built up, sinus rhythm is likely to initiate regular contractions.

The part played by calcium ions in muscular activity is not fully known, but the presence of calcium in the extracellular fluid assists in maintaining the integrity and selective permeability of the muscle membrane, and so facilitates the building up of the appropriate ionic concentrations on both sides of this membrane—which is essential for muscular contractions. A low concentration of calcium in extracellular fluid is known to cause repetitive firing in nerve and muscle tissue, as exhibited in tetany. The injection of calcium ions into the left ventricle stimulates the myocardium to contract and the fact that calcium ions can also induce a fibrillating heart to beat regularly is demonstrated in case 3. An increased concentration of calcium ions, if present in the coronary circulation for prolonged periods, will, however, increase the tendency towards fibrillation.

In the absence or failure of a defibrillator some authorities advocate the use of a local analgesic such as 10 c.c. of 1% procaine injected into the left ventricle. The effect of local analgesics is to stop the transfer of ions across the muscle membrane. Muscular fibrillation is stopped but so is muscular contraction. It is hoped that when normal conductivity is resumed after dispersal of the local analgesic sinus rhythm will ensue. This method can undoubtedly be successful and should be remembered as an alternative, but the heart may subsequently be difficult to start and massage may have to be prolonged. This difficulty is demonstrated in case 1 although, admittedly, 2% lignocaine is a very strong concentration of local analgesic.

Cardiac massage, while necessary to maintain the circulation, is an undesirable mechanical stimulus which can promote ectopic rhythms and which can also damage the myocardium.

The action of adrenaline on a healthy heart in addition to increasing the rate and force of contraction, is to increase the consumption of oxygen, reduce the refractory period of the muscle, and predispose to ectopic rhythms. It is therefore not surprising that when adrenaline is injected into a heart in asystole, ventricular fibrillation often occurs. If the myocardium is anoxic, fibrillation is more likely than ever to occur. The association of adrenaline with fibrillation is illustrated in the above 3 cases. The use of this drug in treating cardiac arrest, where the whole purpose is to restore regular contractions, is surely unwise, and particularly so when a defibrillator is unavailable. Yet adrenaline is probably the most commonly used drug in the treatment of cardiac arrest.

The use of intracardiac solutions of nor-adrenaline stimulates the myocardium to contract although the tendency to produce fibrillation is less than with adrenaline. Therefore, nor-adrenaline in doses of 1-2 c.c. of a 1:10,000 solution may be tried in cases of asystole when the other methods, detailed above, have had no success.

The mortality after cardiac arrest is generally very high indeed. The unsuspected anoxia which might have caused the arrest, might also have affected the heart and brain in an irreversible manner in some cases. In many cases, however, prompt and effective action will surely reduce the mortality.

SUMMARY

Three cases of cardiac arrest are reported and certain aspects of the management of cardiac arrest are discussed, viz the diagnosis of cardiac arrest, cardiac massage, and the treatment of ventricular fibrillation and asystole.

I should like to thank Dr. H. Grant-Whyte, Head of the Department of Anaesthesia, King Edward VIII Hospital, for his interest and advice, and Dr. S. Disler Medical Superintendent, King Edward VIII Hospital, for his permission to publish the cases.

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