# CARDIAC RESUSCITATION[1]

W. A. DODDS, M.D., and P. G. ASHMORE, M.D., F.R.C.S.(C)[2]

CARDIAC RESUSCITATION may be necessary for patients who have developed cardiac arrest or ventricular fibrillation, or in whom the cardiac impulse is so weakened as to result in an inadequate circulation.

In a proportion of these patients the cardiac condition is expected as a terminal event owing to hopeless pathological conditions and resuscitation would be undesirable. However, a large number of cases occurring in the operating theatre during surgery or anaesthesia, or even on the ward, are worthy of our best efforts at resuscitation.

In 1946, Barber and Madden (1) summarized the case reports to that date and mentioned that the first successful cardiac resuscitation had been reported in 1902 by Starling and Lane. Previous to this there had been reports of experimental resuscitation on dogs. They also reported that up till then there had been 48 survials (33 per cent) in 143 attempts.

In 1941 Hamilton Bailey (2) reported four successful attempts out of forty as his personal experience. In 1954 Johnson and Kirby (3) reported 52 per cent successful out of nineteen cases occurring in their operating rooms and mentioned other cases occurring on the wards as unsuccessful owing to the time which elapsed before treatment was instigated.

In 1953, Stephenson, Reid, and Hinton (4) gathered information on 1,200 cases by personal communication and correspondence. There was not necessarily any uniformity of treatment in these cases. In this large series there was a 28 per cent survival figure—94 per cent of the survivals had treatment instigated within the first four minutes and in 6 per cent the treatment began after four minutes. They also reported that if cases from thirty large American centres are considered, there was approximately one case of cardiac arrest per 2,000 anaesthetics or surgical procedures. Miller *et al.* (5) reported an incidence of 1:850 and Cooley (6), reporting on 878 cases of pulmonary stenosis, gave a figure of 1:19. The variation in these figures is due to the type of surgery involved. Stephenson *et al.* (4) found that one-fifth of all cases occur in children under ten years of age and that approximately one in ten patients needing resuscitation requires treatment for ventricular fibrillation.

As anaesthetists, we are primarily interested in cases which occur in the operating theatre. However, there is a growing awareness of the need for cardiac resuscitative measures on the wards. Therefore, it would seem logical for hospitals to have mobile equipment, capable of being moved quickly to all parts of the hospital and suitable for dealing with all types of cardiac resuscitation. Of the 1,200 cases previously mentioned, 132 occurred outside the operating theatre and

[1]Presented at the Western Divisions' Meeting, Canadian Anaesthetists' Society, Calgary, Alberta, March 13, 1958.

[2]Departments of Anaesthesia and Surgery, Vancouver General Hospital, and the University of British Columbia, Vancouver, B.C.

in 17 of these the patients survived. It is, of course, not possible to resuscitate a heart through a thoracotomy if oxygen under pressure is not available.

The aetiology of cardiac arrest may be discussed under four headings.

*Anoxia or hypercarbia.* In the final analysis of these cases, it is often found that $O_2$ lack and $CO_2$ excess are the underlying causes. In fact, it is becoming increasingly evident that without anoxia the other three causes are probably not of great significance. Anoxia of the myocardium may occur for many reasons, such as oxygen lack in inspired gases, obstruction of the airway, deficient oxygen exchange in the alveoli or, due to coronary artery disease, inadequate circulation from blood loss, interference with venous return, or anaemia. The position of the patient on the table is often a factor.

*Neurogenic stimuli* occurring owing to instrumentation of the larynx as in intubation, bronchoscopy or oesophagoscopy may produce serious cardiac arrhythmias. These reflexes may be abolished by deeper anaesthesia or the use of atropine. Other surgical reflexes occurring during traction on the viscera may be abolished or lessened by deeper anaesthesia or atropine. However, it would appear that most reflexes capable of producing cardiac disturbances of a serious nature are avoided by adequate oxygenation and reasonbly deep anaesthesia. The reported frequent occurrence of cardiac arrest during intubation and extubation can to a large extent be avoided by the routine use of atropine preoperatively, also the use of 100 per cent oxygen under positive pressure before either intubation or extubation, as well as the use of Nupercaine or Xylocaine ointment on endotracheal tubes.

*Anaesthetic agents* may produce serious cardiac disturbances. The parasympathomimetic agents, such as cyclopropane and pentothal or other barbiturates, may produce undesirable vagal reflex actions. Also, deep anaesthesia from total anaesthetic agents, such as cyclopropane, Fluothane, and ether, may depress the myocardium, decrease cardiac output, and thus produce coronary insufficiency with resultant myocardial anoxia. Since the routine use of curare agents for relaxation was introduced, the danger of over-dosage from anaesthetic agents has decreased. Other agents, such as trilene, chloroform, and also cyclopropane, which increase cardiac irritability may in the presence of anoxia produce serious cardiac arrhythmias.

Finally, cardiac manipulation as in cardiac surgery increases the danger of arrest—especially if the heart is dislocated from the pericardium causing kinking of the great vessels and therefore a reduction in coronary flow. Increased cardiac irritability—produced by hypothermia—is an added hazard during cardiac manipulation and has led to ventricular fibrillation. Recent use of moderate hypothermia in the range of 30–32° C. has lessened the likelihood of serious arrhythmias if the myocardium does not become anoxic.

The recognition of the need for cardiac resuscitation is primarily the duty of the anaesthetist. During cardiac surgery with the heart exposed, the diagnosis is not difficult to make. However, during other types of surgery, the anaesthetist in the normal course of events is continually checking the pulse, the pressure, and the colour of the patient and he may observe for dilated pupils and a change in

respiration. He might check with the surgeon as to the colour of the wound and also ask him to palpate a large artery in the wound if he is uncertain. An anaesthetist who suspects cardiac arrest should never hesitate to disturb the surgeon in an effort to clarify the situation.

Auscultation of the praecordium as a diagnostic aid is usually of very little value and time should not be wasted unless information can be quickly obtained. Routine use of a praecordial stethoscope to monitor cardiac activity is very useful, particularly for paediatric cases.

The electrocardiogram, if already recording, is invaluable and gives an accurate diagnosis of ventricular fibrillation—or the onset of serious arrhythmias which precede ventricular fibrillation—and also shows the onset of cardiac arrest. The electrocardiogram, if being monitored continually, may give enough warning so that serious cardiac difficulties may be avoided. Unfortunately, a great many patients undergo surgery and anaesthesia without the benefit of continuous electrocardiography. Many simple types of cardiac monitors are becoming available and will perhaps prove of value.

If all other methods fail, direct vision of the heart through a left thoracotomy is the best method for accurate diagnosis.

As already stated, the anaesthetist is in the foreground as far as diagnosis is concerened and may actually find himself in the same position as far as treatment is concerned. There have recently been an increasing number of occasions when cardiac resuscitation has been carried out by non-surgical personnel. Cardiac difficulties do not always wait until the surgeon is present—they may occur in the anaesthetic room or in the recovery room as well as in the operating room. Anaesthetists perhaps have more chance to gain experience in treating these cases as they are continually present in the theatre and available in an emergency.

Recent types of surgical procedures have caused increased interest in ventricular fibrillation. Besides the patient undergoing ordinary surgery, who for one cause or another may develop this difficulty, we now must deal with the hypothermic patient whose myocardium is irritable and who in the presence of anoxia may develop serious arrhythmia; and also with the open cardiac patient receiving total perfusion by means of the pump oxygenator, who may have potassium or acetylcholine arrest, and who is apt to develop either inadequate circulation due to loss of myocardial tone or even ventricular fibrillation when an attempt is made to restart the heart. In our hospital, both hypothermic techniques and the use of the pump oxygenator are managed by the anaesthetists, and these duties have added to our interest in the treatment of cardiac arrest and arrhythmias.

Time is of the utmost importance. The interval between cardiac arrest and the death of nerve tissue in the central nervous system was shown experimentally by Wienberger *et al.* (7) in 1940 to be 3½ min. Clinically this time may be set at 3–5 min. However, if anoxia or a gradual hypoxia has been present for some time before actual cessation of circulation, this time may be considerably reduced, as stated by Cooley (6) in 1950. In 1927, Hamilton Bailey (2) suggested that at the onset of cardiac arrest the time should be noted and each minute called out loudly by an observer. This may seem to be elementary, but in our experience we

are often in doubt as to the exact time of cessation of circulation. Once the diagnosis is made the heart should be exposed through a thoracotomy in the fourth left interspace—care being taken to avoid the internal mammary artery. The incision should extend well out into the mid-axillary line to allow for adequate exposure. Sterile preparation of the skin is not warranted for this incision. At the same time, artificial respiration with 100 per cent oxygen should be instituted through an endotracheal tube; 10° Trendelenburg may or may not be advantageous. Mechanical positive and negative respiration has some slight effect on the maintenance of circulation as stated by Thompson and Roche (8) in 1947.

A short period of time may be spent in attempting to massage the heart through the diaphragm if the abdomen is open. However, thoracotomy is by far the best route. In our hospital, if the diagnosis is in doubt, it is suggested that we cut the skin over the fourth left interspace and if there is no bleeding open the chest. However, we have not got to the point where we prepare the left chest for all surgical procedures.

When the heart is exposed, a short preliminary examination and even massage may be attempted before opening the pericardium. If there is no rapid return to a normal beat, the pericardium shoulc be incised anterior to the phrenic nerve. At this time, by observing the heart, a diagnosis of arrest or ventricular fibrillation can be made. In any event, massage should be started immediately. Our experimental work on dogs undergoing hypothermia[3] and cardiopulmonary bypass, as well as on clinical patients, has led us to believe that the best method of massage is to use two hands, one in front and one behind the heart, and pump the blood from the apex to the base of the heart by compressing the hands. The rate is to some extent governed by the filling but usually approximates sixty per minute. By this means, an arterial pressure of 40–60 mm. Hg should be maintained. The use of two hands makes it easier to maintain this pressure and also lessens the danger of rupturing the ventricle with the thumb as reported by Hurwitt and Seidenberg (9) in 1953 and Haight and Sloan (10) in 1955. Also, two-handed massage is not as tiring for the operator. In the presence of reduced blood volume, periodic aortic compression may aid in keeping adequate cerebral blood pressure. Intra-arterial transfusion may also be considered if the anaesthetist keeps in mind the dangers of excess citrate (11) and potassium (12). The adequacy of the massage may be judged by whether a femoral or carotid pulse can be felt—it is not usual to be able to produce a radial pulse.

In order to massage satisfactorily, a rib-spreader should be available to ensure enough room. In small infants, it may be more practical to massage the ventricles against the sternum by using two fingers.

At this stage the operator should observe the tone of the myocardium—if there is loss of tone it is difficult to maintain arterial pressure and 3–5 cc. of 10 per cent $CaCl_2$ may be injected into the left ventricle or left atrium. It is necessary now to definitely diagnose if the ventricle is arrested or fibrillating. If arrested, massage should be continued and tone maintained until an adequate beat returns. It is best to avoid fibrillation, if at all possible, because of the increased

[3]Supported by a grant from the British Columbia Tuberculosis Society.

oxygen consumption of the myocardium (13). If fibrillating, the oxygenation of the myocardium is observed. It is our experience that a fibrillating ventricle will improve its colour by massage and also the fibrillatory movement will change from a fine to a coarser movement. If the fibrillating ventricle is flaccid, it is often beneficial to inject adrenalin 2–4 cc. 1:10,000 into the left heart and to continue massage until the peripheral vasoconstriction improves venous return to the heart. When the myocardium regains tone and colour, and the fibrillating is coarse, the defibrillator is used. The ventricles are shocked by placing the electrodes across the septum and a single shock of 170–220 v. at 1.5 amp. for 0.2 sec. is used. If this treatment does not stop the fibrillating and produce arrest or a co-ordinated beat, massage is restarted to maintain the circulation. The shock is repeated and, if not successful, a series of three shocks repeated rapidly, as suggested by Wiggers (14) in 1940, is used. In our experience it has not been difficult to convert ventricular fibrillation to an adequate conducted beat in normothermic or hypothermic animals or patients.

The voltage required varies with the size of the heart and its electrical resistance as stated by McMillan *et al.* (15) in 1952. Beattie and Blades (16) in 1953 reported the resistance of the human heart to be approximately 50 ohms; therefore, 170–220 v. should be adequate for all hearts. It is necessary to moisten the electrodes with normal saline and to place them carefully to ensure contact so that the myocardium is subjected to the total current. It is also important to reduce the time of shock to a minimum (less than 0.5 sec.) to avoid burning the myocardium. Following successful defibrillation, massage should probably be continued as cardiac arrest may persist or, if the ventricles are beating, the impulse at first may not be adequate. Resuscitative procedures should be continued until return of cardiac action seems unlikely or until there is known irreversible central nervous system damage. There are reports of return to normal function after massage lasting up to 7 hr. There is one case reported by Haight and Sloan (10) in 1955 of successful resuscitation after 3½ hr. of massage, including rupture of the ventricle through an old infarction.

All available information should be weighed carefully during the treatment to help arrive at a prognosis or to decide when treatment should be discontinued—information such as time elapsed without adequate circulation, cardiac condition preoperatively, dilatation of pupils, and maintenance of circulation by massage. As a rule, 1–2 hr. is a long time to continue treatment. Some increased prognostic ability may be gained by the use of the electro-encephalogram.

Cardiac emergencies of this nature arising during cardiac surgery may be better dealt with if the cardiac abnormality such as valvular stenosis or other defects are corrected before or during massage. (17)

Chemical defibrillation, using procaine and potassium, has not been successful in our laboratory. Defibrillation may be accomplished, but these drugs seem to poison the myocardium with resultant dilatation which is not responsive to treatment.

Our experience in the laboratory with the prevention of ventricular fibrillation by means of prostigmin or the injection of the S-A node with procaine has been unsatisfactory, as it has with the use of respiratory alkalosis by hyperventilation.

When a conducted beat returns and the blood pressure is maintained adequately by the heart itself, all bleeding points should be stopped, the pericardium may or may not be closed. The chest is closed using underwater drainage and the patient treated, as necessary, for respirations, blood volume, vasopressors, and fluids. An indwelling catheter may be of value in assessing kidney function, and antibiotics should be left in the chest.

The most important aspect of the treatment of cardiac arrest is prevention. With adequate oxygenation of the myocardium, serious cardiac arrhythmias will probably not occur. In an experimental series using dogs, it was found in our laboratory that in the hypothermic animal subjected to an 8-min. cardiac occlusion, cardiac irregularities due to reduced conductivity developed in the first 30 sec. and progressed throughout the occlusion time. However, when the coronary arteries were perfused through the left subclavian artery with small quantities (2 cc./kg./min.) of arterialized and heparinized blood these arrhythmias either failed to develop or reverted to normal.

The next most important factor in treating cardiac emergencies is observance of time. It should not be difficult to restore cardiac action, but the central nervous system is the chief site of damage—and this damage occurs early. Central nervous system damage has, however, been shown by Stephenson *et al.* (4) to improve gradually over many months in patients who survive. Kountz (18) in 1936 described perfusing post-mortem hearts and restoring cardiac action after 30 min., 60 min., and even 5–6 hr. It is not likely that the use of an artificial pacemaker will be of value in these patients as the conducting mechanism of the heart is intact. The use of external defibrillator should also be limited to a very few patients in whom ventricular fibrillation can be diagnosed without observing the heart and where profound anoxia of the myocardium has not had time to develop.

## Summary

Need for cardiac resuscitation occurs in approximately 1:1,000 to 1:2,000 surgical procedures. Early recognition by the anaesthetist is important. The surgeon may be required to assist in the diagnosis.

The final diagnosis is best made by observing the heart through a left thoracotomy. The thoracotomy incision should be large, in the fourth left interspace and avoiding the internal mammary artery.

The most common cause is myocardial anoxia with or without reflex activity, anaesthetic agents, or cardiac manipulation.

Early instigation of treatment is important. Respiratory resuscitation should be carried on at the same time as cardiac resuscitation. Cardiac massage is best maintained using two hands and should be started within 4 min. Myocardial tone may be improved using calcium chloride or adrenalin. Ventricular fibrillation is treated by electrical shock, 170–220 v., 1.5 amp., and 0.2 sec.

Defibrillation should not be attempted until the myocardium is oxygenated and the fibrillating movements become coarse. Cardiac massage may be necessary after defibrillation. Cardiac defects such as valvular stenosis should be corrected during massage if possible. All treatment should be aimed at reducing the total

time of cerebral anoxia. When resuscitation has been completed, careful post-operative care is necessary.

The best method of treatment is prevention.


## Résumé

On pratique la resuscitation cardiaque pour traiter l'arrêt cardiaque, soit la fibrillation ventriculaire, soit une activité cardiaque inadéquate. Il s'impose de recourir à cette sorte de traitement dans 1:1000 à 1:2000 des interventions chirurgicales. La cause la plus fréquente de cette situation est l'anoxie du myocarde avec ou sans l'aide d'une activité réflexe d'agents anesthésiques ou d'une manipulation cardiaque. Le diagnostic, habituellement, est fait par l'anesthésiste qui observe les signes vitaux et, finalement, en pratiquant une thoracotomie gauche. Le traitement peut être fait par un anesthésiste, un chirurgien ou toute autre personne qualifiée et ce traitement consiste à faire un massage cardiaque précoce, à employer du chlorure de calcium ou de l'adrénaline et, s'il y a lieu, un défibrillateur électrique. On pratique un massage plus adéquat en se servant de ses deux mains. La défibrillation par choc électrique s'opère facilement lorsque le myocarde a été oxygéné par le massage et que les mouvements fibrillatoires du ventricule sont passés de petits à plus gros.

Les facteurs les plus importants sont la mise en œuvre précoce du traitement et la capacité de maintenir une circulation adéquate par le massage.


## REFERENCES


1. Barber, R F., & Madden, J L. Historical Aspects of Cardiac Resuscitation. Am. J. Surg. *70*: 135 (1945).
2. Bailey, H. Cardiac Massage for Impending Death under Anaesthesia Brit. Med. J. 2: 84 (1941).
3. Johnson, J, & Kirby, C K. Cardiac Standstill and Ventricular Fibrillation. Am. Pract. 5: 264 (1954).
4. Stephenson, H. E, Reid, L C, & Hinton, J W. Some Common Denominators in 1200 Cases of Cardiac Arrest. Ann. Surg. *137*: 731 (1953).
5. Miller, F. A., Brown, E B., Buckley, J. J, Van Bergen, F. H.; & Varco, R. L. Respiratory Acidosis. Its Relationship to Cardiac Function and other Physiologic Mechanisms. Surgery *32*: 171 (1952).
6. Cooley, D. A. Cardiac Resuscitation during Operations for Pulmonic Stenosis. Ann. Surg. *132*. 930 (1950).
7. Wienberger, L W, Gibbon, M H, & Gibbon, J. H., Jr. Arch. Neurol & Psychat. *43*. 615 (1940).
8. Thompson, S. A., & Roche, E. F. The Effect of Mechanical Artificial Respiration upon the Maintenance of the Circulation. Surg., Gynec. & Obst. *84*: 1059 (1947).
9. Hurwitt, E. S., & Seidenberg, B. Rupture of the Heart during Cardiac Massage. Ann. Surg. *137*: 115 (1953).
10. Haight, C, & Sloan, H. Successful Cardiac Resuscitation despite Perforation of the Heart during Massage. Ann. Surg. *141*. 240 (1955).
11. Cookson, B. A., Costos-Durieux, J., & Bailey, C. P. The Toxic Effects of Citrated Blood and the Search for a Suitable Substitute for Use in Cardiac Surgery. Ann. Surg. *139*: 430 (1954).
12. Melrose, D. G., & Wilson, A. O. Intra Arterial Transfusion: The Potassium Hazard. Lancet *1*: 1266 (1953).

13. JARDITZKY, O., GREENE, E. A., & FORBES, V. Oxygen Consumption of the Completely Isolated Dog Heart in Fibrillation. Circ. Res. *4*: 144 (1956).
14. WIGGERS, C. J. The Physiologic Basis for Cardiac Resuscitation from Ventricular Fibrillation: Method for Serial Defibrillation. Am. Heart J. *20*: 413 (1940).
15. MCMILLAN, I. K. R., COCKETT, F. B., & STYLES, P. Cardiac Arrest and Ventricular Fibrillation. Thorax 7: 205 (1952).
16. BEATTIE, E. J, KESHISHIAN, J. M, AMES, N. B., & BLADES, B. Ann. Surg. *137*: 504 (1953).
17. MILSTEIN, B. B., & BROCK, SIR RUSSELL. Ventricular Fibrillation during Cardiac Surgery. Hosp. Rep. *103*. 213 (1954).
18. KOUNTZ, W. B. Revival of Human Hearts. Ann. Int. Med. *10*: 330 (1936).

## ERRATUM

BRADSHAW, A. K., FRASER, ROBERT S., & MCINTYRE, J. W. R. The Effect of Chlorpromazine on Pulmonary and Systemic Arterial Pressure in Dogs. *Canadian Anaesthetists' Society Journal*, vol. 5, no. 3 (July, 1958), p. 338.

TABLE I: line 1 should read *Dosage 0.5 mg. kg.*; line 8 should read *1 mm. Hg.*