# CARDIAC ARREST AND RESUSCITATION*

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Part II

## DIAGNOSIS OF CARDIAC ARREST

Precious moments are lost when surgeon and anaesthetist hesitate to diagnose cardiac arrest. The circulation must be restored within 3 minutes; there is no time to double-check. When anoxia has slowly built up before actual arrest, time is even shorter. The only decision to be made is whether the circulation is insufficient to maintain brain viability. Should a hasty thoracotomy reveal a weakly beating heart, this is cause for congratulation, not reproach. We should recognize that our hesitancy stems from an ardent wish to be clear of disaster; this is a natural reaction but it helps neither the doctor in his predicament nor the patient. When a patient drops dead in a ward there is seldom panic or diagnostic doubt; the patient looks dead, he is pulseless, and his pupils are dilated. No responsibility attaches to anyone. Nowadays, however, even in the ward, there is no escape. The thoracic surgeon need only look at the heart. The abdominal surgeon must feel the near-by aorta or palpate the heart through the diaphragm. If the body cavities are not open, the carotid pulse must be sought, and if that is absent one must prepare for action. If the pupils are seen to dilate at the same time, then the diagnosis is established. An observant anaesthetist will know the previous size of the pupils and will know whether they were already affected by atropine. Even so, the enormously wide pupillary dilatation of anoxia is unmistakable. Fortunately the natural revulsion of most doctors against the opening of the chest need no longer be a psychological cause of delay. External cardiac massage can be commenced immediately.

## CLOSED-CHEST RESUSCITATION OF VENTRICULAR FIBRILLATION

If one looks into the nooks and crannies of most operating theatres, a printed form will be found outlining the steps that must be taken to resuscitate the heart. Occasionally a large notice with bold lettering will be prominently displayed. The place for such a schedule is the brain, not the wall. Each step must be automatic; reaction must be instinctive and correct. The immediate object is to recirculate oxygenated blood to the brain. Unless this is done within 3 minutes of collapse, the patient will be surely dead. The lungs must therefore be ventilated and the heart intermittently compressed. Once these two steps are effective, the dire emergency is under control.

### *Ventilation*

In the operating theatre the task of ventilation should present no difficulty. A well-fitting face mask is adequate and rhythmic compression of the oxygen bag will inflate the lungs. The chest must move freely with each squeeze of the hand. There is no place for doubt. The muscles are all flaccid and offer no resistance to movement. If the chest does not move the airway is blocked or the mask ill-fitting. Support of the angle of the jaw will usually correct the former by preventing the base of the tongue from blocking the larynx. The pharynx must be cleared of blood, vomitus or secretion. A skilled person should introduce an endotracheal tube as soon as possible. This must be large, or else a balloon must be inflated to block the trachea around the tube. A small tube in a capacious trachea is as useless as a loosely fitting face mask. Time should not be wasted in trying to learn the technique of intubation; a pharyngeal airway and a face mask are enough.

In the absence of oxygen or anaesthetic equipment, take comfort in the fact that expired air contains 16% oxygen. Elam *et al.*[19] have shown that it is possible to maintain normal blood oxygen and carbon dioxide values in curarized patients by insufflating mouth to mask or mouth to tracheal tube, particularly if the operator overventilates slightly. The subject is placed supine with the head tilted well back to open the airway. The nostrils are pinched with the fingers or obstructed by pressure of the operator's cheek. The operator takes a deep breath and exhales lips to lips into the mouth, forcefully for adults, more gently for children, and with a mere puff for babies.[36] With children the nose and mouth can be enclosed in the operator's lips. Ventilation should be maintained at a rate of 15 - 20 per minute and between each breath the operator withdraws his face so as not to impede passive exhalation from elastic recoil of the subject's lungs and chest wall. Gastric distention and the risk of infection to the operator, as well as the aesthetically repugnant nature of the technique, are disadvantages of an otherwise efficient and universally applicable method of ventilation. The oropharyngeal airway designed by Safar and McManon[45] overcomes the hygienic objections to mouth-to-mouth breathing. This consists of 2 Gueddel-type tubes mounted base to base in reverse (Fig. 5). There are tubes of different sizes, for adults and for children. Mouth-to-mouth breathing or its modification is effective even with the chest open.

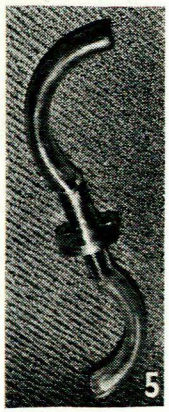


*Fig. 5.* Oropharyngeal airway for emergency ventilation.

### *Cardiac Massage*

Restoration of the circulation takes equal precedence with ventilation. The one is useless without the other. If collapse has occurred in any circumstance other than an intrathoracic operation the following procedure should be followed.

(*a*) The patient's head is steeply lowered if he is lying on an operating table or else his legs are elevated. This improves the venous return to the heart and increases the cerebral blood pressure.[14]

(*b*) Three sharp thumps with the fist are administered to the praecordium.[47] This is most effective in Stokes-Adams attacks, the condition for which it was originally described. If a pulse does not appear within a few seconds,

fibrillation is assumed and the next step must be taken.

(*c*) External cardiac massage was introduced by Kouwenhoven *et al.*[27] in 1960. They intended it to be used in conjunction with external defibrillation. Provost and Batelli[41] long ago established that electrical defibrillation is only successful if applied within a minute of the onset of fibrillation; otherwise the heart must first be massaged to restore the degree of oxygenation essential for defibrillation. The patient is laid on an unyielding surface, and the floor is often the only one available. The base of the left hand is placed over the lower sternum with the right hand cupped over it. (Figs. 6A and 6B). The elbows are bent and then sharply straightened to press the left hand vertically downwards so as to compress the heart between

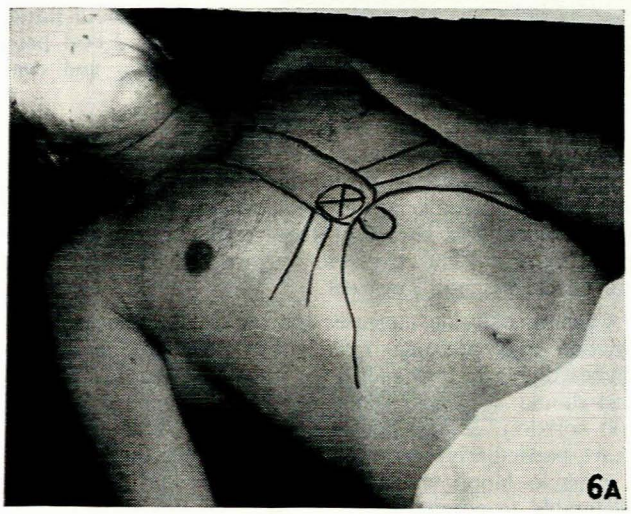


*Fig. 6A.* Outline of sternum and costal margin. Manual compression is applied at the point marked X.

the sternum and the vertebral bodies. The sternum must be appreciably moved and the operator must adjust his body position so as to use his weight in applying pressure. After each compression the arms are relaxed. With small patients less force is exerted, and with infants light pressure with two or three finger tips is sufficient. It is useless to go through the motions of external massage without being certain of its efficacy. Someone must always have a finger on a pulse and the femoral artery is the most convenient. The pulse produced by effective external massage can be felt even with an obese subject. The impulse is more prolonged than a normal pulse and has an unmistakably eddying quality. The pupils will dilate widely within seconds of circulatory arrest, but under effective massage they contract and must remain so throughout resuscitation. It may be necessary for relays of operators to work at the massage, for it is a tiring business. The technique is soon learnt by observation, and onlookers can be coopted to do their stint. Milstein[36] (1963) was able to record ankle-vein and radial-artery pressures during external massage. A systemic blood pressure of 75/35 mm.Hg was produced while the venous pressure rose only negligibly. These direct observations contradict the assertion of Weale and Rothwell-Jackson[54] (1962) that closed-chest massage is less effective than direct cardiac massage. In my experience, external cardiac massage is capable of maintaining an effective circulation for upwards of an hour. It is simple, it requires no courage and little skill, and it can be instituted without delay. Closed-chest massage has been used at the Johns Hopkins Hospital since 1961, and during that time it has not been necessary to open the chest of any patient with acute cardiac arrest.

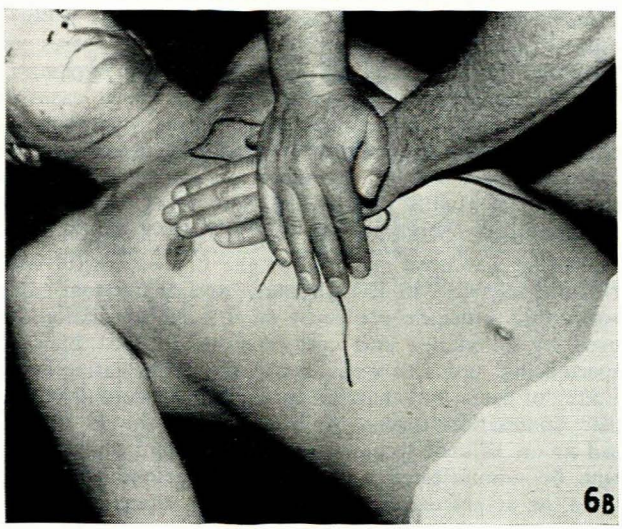


*Fig. 6B.* Position of the hands for external cardiac massage.

Closed-chest cardiac massage may cause rib fractures. This has happened in 2 of my cases. It is particularly liable to occur in elderly or obese subjects because the chest is brittle or because it is difficult to compress the heart until the sternum has been fractured.[46] Baringer *et al.*[3] produced chest fractures in one-third of their cases, but with increasing experience their incidence has dropped. They also reported laceration of the liver, but this should be avoided by pressing accurately over the sternum and avoiding excessive force. McLeod[31] has reported rupture of the heart at the entry of the inferior vena cava into the right atrium. Jude *et al.*[25] have described pericardial effusion and tamponade in patients with myocardial infarcts under anticoagulation therapy.

Throughout the period of external massage pulmonary ventilation must be maintained, for chest-wall compression does not move an adequate amount of air into the air passages. Safar *et al.*[46] showed that the tidal volume of patients with cardiac arrest undergoing external massage without ventilation was negligible. An unfortunate single-handed operator must take time off to inflate the lungs by mouth-to-mouth breathing for three or four breaths every half minute until assistance arrives. There must be close cooperation between the operator and the ventilator. If the chest is compressed while the lungs are inflated, the intrapulmonary pressure may rise so sharply that alveoli will rupture. To avoid this, the lungs should be inflated while the operator is relaxing between compressions.

*Defibrillation*

The circulation produced by cardiac massage never equals that of the normally beating heart. Under cardiac

massage respiratory movements are never sufficient to maintain oxygenation, and consciousness is never regained. Deep gasping respiratory efforts are an agonal sign of ominous portent. A cyanosed, flaccid, weakly quivering heart will not defibrillate, but massage will cause it to become pink, to regain tone, and to fibrillate coarsely and vigorously (Figs. 2, 3 and 4). Wiggers[56] did not have a single case of spontaneous defibrillation in 208 dogs treated by cardiac massage alone. However, spontaneous defibrillation does occur in man. Among our 52 cases of ventricular fibrillation under cardiopulmonary bypass, 12 reverted spontaneously to normal rhythm. Of 21 cases of ventricular fibrillation complicating mitral valvotomy, 4 defibrillated spontaneously. This usually happens when the heart has fibrillated for no reason except irritability, and conversion is usually rapid. One must never wait for this to happen and thereby delay electrical defibrillation.

Procaine was used in 1942 for the first successful defibrillation in man.[1] In 1940 Burstein and Marangoni[11] had shown that procaine protected dogs from fibrillation induced by adrenaline and cyclopropane. In 1951 Blalock[9] reported that procaine was a successful defibrillating agent in 50% of cases, but that the force of the resulting ventricular contraction tended to be weak. It should not be used as an adjunct to electrical defibrillation. Should procaine be administered and the heart become weak and atonic, its action can be antagonized by adrenaline.

Potassium salts should be excluded from the list of resuscitation drugs. D'Halluin[16] showed that potassium chloride diminishes myocardial tone and in excess causes diastolic asystole. Potassium can only be condemned, for in defibrillating doses it is likely to cause irrevocable atonic arrest. Its action is antagonized by calcium salts, which correct potassium hypotonia, but the pendulum is difficult to control and will all too readily swing back to ventricular fibrillation.

The action of electrical defibrillation is to render all the asynchronously beating muscle fibres refractory at the same time. After a short period of asystole a coordinated beat returns. The shock must be strong enough to stop the contraction of every muscle fibre. MacKay *et al.*[30] have shown, in dogs, that shocks of 0·09 to 0·4 amps. always cause ventricular fibrillation and never defibrillate, whereas shocks of 1·5 - 3·8 amps. always defibrillate and never fibrillate. The defibrillating shock is now standardized, and with directly applied electrodes the machine should be capable of delivering a current of 1 amp. with a voltage range from 80 to 250 and a stimulus duration of 0·1 to 0·5 seconds. Large electrodes are necessary if heart burns are to be avoided; they should be 7·5 cm. in diameter for adults and 2·5 cm. for children. The external defibrillator designed by Kouwenhoven *et al.*[26] delivers 5 amps. at a potential of 480 volts with a 60-cycle alternating current and a duration of 0·25 seconds. The external defibrillator developed by the Cardiovascular Research Unit of the University of the Witwatersrand[32] has voltage selection between 350 and 750 volts with a fixed current duration of 0·1 second and a maximum current flow of 15 amp.; it requires external electrodes of at least 7·5 cm. in diameter.

Once adequate ventilation and artificial circulation have been established, preparations must be made for defibrillation. The sooner this is done the better. Even if an external defibrillator is not available, external massage should be started while preparations for thoracotomy are being made. The left chest is opened through the 5th intercostal space, the incision extending from the sternum into the axilla. The internal mammary artery may be injured, and when the chest is eventually closed it should be ligated. The ribs are forcibly retracted with a rib spreader to allow comfortable access of the hands. The lung is retracted and the pericardium slit widely open in front of the phrenic nerve. The circulation will once again be arrested and it is necessary to recommence massage. Both hands should be used, the heart palmed between them and compressed. After each compression the hands must be completely relaxed so as to allow the heart to fill. Compression should be rapid and firm, and both hands should have wide contact with the ventricles. Local pressure by thumb or finger may cause bruising and even perforation.

Hearts that defibrillate most easily are pink, possess good tone, and are fibrillating vigorously. In order to obtain these conditions it may be necessary, in addition to massage, to inject drugs. Only two are useful—adrenaline and calcium. The choice of these should be injected into either ventricle and the heart vigorously massaged until the effects become apparent.

Calcium chloride increases the force of ventricular contraction and prolongs systole by direct action on the myocardium. It is the drug of choice in cardiac asystole and should be injected in doses of 5 - 10 ml. of the 1-in-100 solution. It also improves the tone of a fibrillating heart, particularly where the catastrophe has occurred after a massive blood transfusion. It is safe and is probably preferable to adrenaline.

Adrenaline hydrochloride increases the rate and force of myocardial contraction and by causing vascoconstriction raises the peripheral resistance. It may be effective in restoring the beat in asystole, but often precipitates ventricular fibrillation. The dose is 5 - 10 ml. of a 1-in-10,000 solution.

Once the heart is suitably fibrillating, the electrodes are applied, one on each side of the ventricular mass. First a single shock of 80 - 120 volts for 0·1 second is given. If this is not successful the voltage may be increased or the duration of shock lengthened. Each shock will cause general tonic convulsions of the body. Should fibrillation persist after 2 or 3 intermittent shocks, the heart must again be massaged and, if necessary, a further injection of adrenaline or calcium given, preferably different from the one previously administered. When conditions are again suitable the method of Wiggers[55] should then be used, namely a series of 3 - 5 shocks of 0·1 second at 150 volts at 1-second intervals. 'Serial defibrillation' is often successful after single shocks have failed. Patience and persistence will be rewarded on occasions; one patient successfully resuscitated after 60 minutes of massage interspersed with 12 shocks.

After defibrillation the ventricles will remain motionless for a few moments, and the heart must not be touched for fear of precipitating ventricular fibrillation. There may be a delay of 5 or even 10 seconds before coordinated beats

start. Should asystole persist longer the outlook is grim indeed; the heart is probably beyond resurrection. A feeble beat must be manually assisted, when the heart may either respond or revert to fibrillation.

There should be no hurry to close the chest. Ventricular fibrillation may recur, and half an hour should be given to observation. During this time bleeding points can be controlled, the pleura drained, and the wound sterilized and draped.

If an external defibrillator is available, surgical venturesomeness is unnecessary. External defibrillation suffers from the disadvantage that the heart cannot be seen and assessed as suitable for defibrillation. If ECG is available, feeble fibrillation can be recognized by the low amplitude of its undulations. Suitable fibrillating conditions must otherwise be assumed after a period of vigorous external massage. In all my cases adrenaline has been given. In one the injection was made intravenously and the defibrillating shock administered 30 seconds later. This is the most suitable route of administration. In the others, the drug

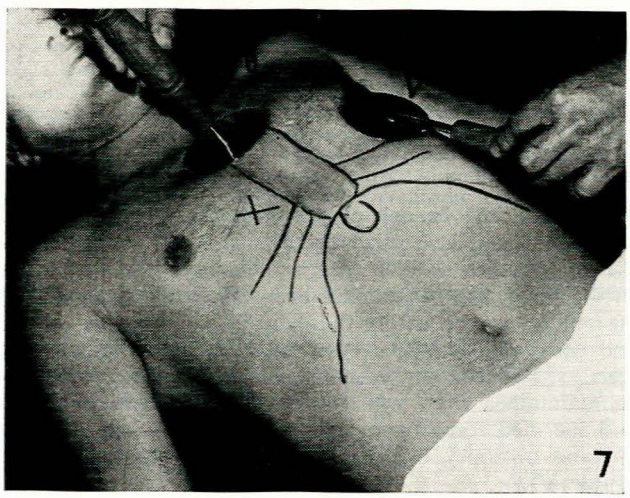


*Fig. 7.* Position of electrodes for 'vertical' electrical defibrillation.

was injected through the chest wall into the heart with a size-18 needle; no pericardial haemorrhages were caused as judged clinically in the 3 survivors and in the fourth at autopsy. This must be followed by vigorous massage for about 15 seconds before defibrillation is attempted. One electrode is placed over the manubrium sterni, the other over the region of the heart's apex (Fig. 7). They are liberally smeared with electrode paste to effect good contact. Though the handles are heavily insulated I prefer different assistants each to hold an electrode. For adults a voltage of between 400 and 750 is selected, according to the subject's size and obesity. With children a shock of between 250 and 400 volts is adequate. For small children a conventional internal defibrillator can be used at a voltage of 150 - 200. The method of shocking is the same as for internal defibrillation. At first single shocks are given and if these fail 'serial' defibrillation should be attempted. If the 'vertical' position of the electrodes is ineffective, 'transverse' shocks should be given with one electrode placed over the lower sternum at the same level as the praecordial electrode (Fig. 8). In 3 of my cases rhythm conversion followed single vertical shocks and the fourth was successful after transverse serial defibrillation. Success is determined by keeping a finger on the femoral artery and feeling for the pulse, which returns rapidly. On one occasion an ECG was connected and restoration of isometric ventricular contractions was obvious.

## RESULTS

An adequate heartbeat was restored in 42 of our 63 cases (66·6%). During the post-resuscitation period a further 12 patients died from cardiac or cerebral causes directly

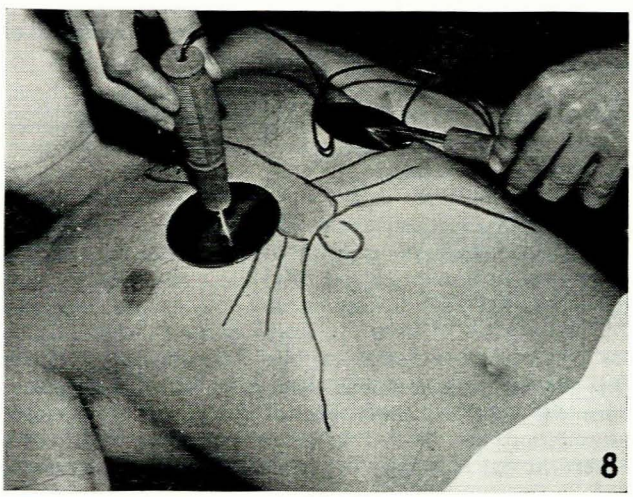


*Fig. 8.* Position of electrodes for 'transverse' electrical defibrillation.

attributable to the cardiac arrest (Table IV). Jude *et al.*[25] have taken restoration to the 'pre-arrest central nervous system status' to be the criterion for success and on this basis our success rate has been 47·5%. Patients who develop cardiac arrest are often poor surgical risks liable to other complications, and indeed 4 patients died later from causes unrelated to cardiac arrest.

There is a close similarity between the results of the two groups treated by direct cardiac massage (Table IV) even though in one group the chest was already open. However, Tables I, II and III show that it is not valid to consider cardiac arrests with open chest in a single group. 60% of patients whose hearts arrested during mitral valvotomy recovered fully, whereas no patient suffering from chronic hypoxia survived. It is obvious that the result will depend to an appreciable extent upon the nature and severity of the disease that predisposed the heart to fail. There were, in fact, no survivors among 13 patients with chronic toxaemia, cyanotic heart disease or congestive cardiac failure. The more sudden the arresting stimulus, the better the chances of recovery. Arrests caused by vagal reflexes and myocardial irritability happen almost instantaneously and sometimes in the absence of anoxia. In our hands more than 80% of these have survived and almost invariably the heart has been resuscitated within a matter of minutes. Where the causative factors have operated

over the course of minutes or hours the heart's powers of recovery are impaired and the results are correspondingly poorer. When anoxia has slowly built up during the operation before finally precipitating arrest, between one-third and one-half of the patients have died. These are the results which have been obtained with cases of air emboli, acute anoxia and massive blood transfusion. (Table III.)

The cases that occurred when the chest was closed but were resuscitated by either open or closed methods are essentially comparable in nature and causation (Tables V and VI). The results following closed-chest resuscitation are better than those following open resuscitatory measures, the final survival figures being 71% and 45% (Table IV).

TABLE IV. COMPARISON OF RESULTS OF OPEN AND CLOSED METHODS OF RESUSCITATION

| | *No.* | *Successful cardiac resuscitation* | *Post-resuscitation deaths* | *Total recoveries %* |
|---|---|---|---|---|
| Arrest and resuscitation during thoracotomy | 45 | 29 (64%) | 9 | 44% |
| Arrest with chest closed (open-chest massage) | 11 | 7 (64%) | 2 | 45% |
| Arrest with chest closed (closed-chest massage) | 7 | 6 (86%) | 1 | 71% |

The numbers are few but they gain added significance from the fact that only one of the 7 cases of closed resuscitation was treated in a surgical operating theatre; 3 were in the radiology department, 2 in the wards, and

TABLE V. LIST OF CASES OF CARDIAC ARREST OCCURRING WITH CLOSED THORAX BUT TREATED BY OPEN-CHEST RESUSCITATION

| *Age and sex* | *Cause* | *Site* | *Duration of internal massage* | *Cardiac resuscitation* | *Survival* |
|---|---|---|---|---|---|
| 49M | Anoxia. Toxaemia. Amoebic abscess | Operating theatre corridor | 60 min. | Failed | No |
| 34M | Dilatation of cervix | Doctor's consulting room | 90 min. | Failed | No |
| 3F | Foreign body trachea. Bronchoscopy | Operating theatre | 15 min. | Succeeded | Cerebral death 24 hours |
| 44M* | Aortic stenosis. Left ventricular puncture | X-ray department | 10 min. | Succeeded | Yes |
| 10M | Intubation for tonsillectomy | Operating theatre | 10 min. | Succeeded | Yes |
| 13M | Intubation for tonsillectomy | Operating theatre | 5 min. | Succeeded | Yes |
| 38F | Foreign body trachea. Bronchoscopy | Operating theatre | 7 min. | Succeeded | Yes |
| 42M | Anoxia Wilson's rest for lumbar disc | Operating theatre | 10 min. | Succeeded | Yes |
| 47M | Anoxia Wilson's rest for lumbar disc | Operating theatre | 90 min. | Succeeded | Cerebral death 48 hours |
| 13M | Per-cricoid bronchogram. Air embolism | X-ray department | 120 min. | Failed | No |
| 47M | Procaine injection for removal of naevus from hand | Operating theatre | 60 min. | Failed | No |

* Case 6.

1 in a maternity department. On the other hand, 8 of the 11 open resuscitation cases were in surgical operating theatres, where skilled personnel and facilities for emergency treatment are more readily available. It would seem, therefore, that the better results are directly ascribable to the method of resuscitation used. Once one accepts that external cardiac massage is as effective as direct cardiac massage, this is not surprising, for the external massage can be started immediately and by anyone.

*Personal Experience of Closed Resuscitation*

It is testimony to the efficacy of external massage that 3 of our patients began spontaneous respirations within 1 - 3 minutes of defibrillation. In none was the external defibrillator immediately available, and this caused un-

TABLE VI. LIST OF CASES OF CARDIAC ARREST TREATED BY EXTERNAL CARDIAC MASSAGE

| *Age and sex* | *Cause* | *Site* | *Duration of external massage* | *Defibrillation* | | *Survival* |
|---|---|---|---|---|---|---|
| | | | | *Ext.* | *Int.* | |
| 7M† | Toxaemia. Bronchoscopy | Operating theatre | 10 min. | | + | Yes |
| 33F‡ | Sensitivity to quinidine | Ward | 18 min. | + | | Yes |
| 57M | Gastric haemorrhage, hypotension. Massive transfusion | Ward | 80 min. | + | | Yes. Recurrent haem. 2nd day, died |
| 83F | Coronary thrombosis | Radiology department. Routine chest X-ray | 90 min. | + | | No |
| 32F | Haemorrhagic shock. Caesarean section | Labour ward | 120 min. | Failed | | No |
| 4F | Bronchiectasis B'copy and B'gram | Radiology department | One thump | Not necessary | | Yes |
| 5F | B'copy prior to B'gram | Radiology department | One thump | Not necessary | | Yes |

† Case 7. ‡ Case 8.

necessary delay. In one of the 2 deaths, the patient had been inadequately ventilated and was so far gone that her skeletal muscles scarcely reacted to shocks of 750 volts. In the other, the patient had been efficiently managed but 90 minutes passed before the defibrillator could be located and delivered. The heart was successfully defibrillated by serial shock, but an adequate blood pressure could not be maintained even with intravenous hypertensive agents and she died some hours later. This woman was 83 years old and necropsy revealed a massive myocardial infarct. (Table VI.)

The following is an example of successful closed resuscitation.

*Case 8.* Mrs. A.C., aged 33. This patient had a mitral valvotomy by Mr. L. Fatti 3 weeks previously. An attempt was being made to convert her atrial fibrillation with quinidine. She had been on gradually increasing doses of the drug for 3 days, felt nauseated and unwell, and had been told to remain in bed. On the day of her collapse (11 September 1962) she was given 6 grains of quinidine 3-hourly and had already had 24 grains. She became confused at 5 p.m. and left her bed to walk along the passage. Dr. H. van Hasselt and I witnessed her collapse and immediately hurried to her. Her carotid pulse was absent and her pupils widely dilated. Dr. van Hasselt started mouth-to-mouth breathing while external cardiac massage was commenced. Her colour improved, her pupils constricted, and a forceful femoral pulse was produced. Oxygen, an endotracheal tube and a rebreathing bag were obtained and satisfactory ventilation was soon established. Mr. J.-J. Brossy and I took turns at massage. The external defibrillator was in the experimental operating theatre at the medical school and 18 minutes elapsed before it arrived. The electrodes were placed in the vertical axis of the heart and a shock of 650 volts was given. Defibrillation was immediate. A good forceful femoral pulse returned. The patient breathed spontaneously within a minute and opened her eyes within 5 minutes. She was then lifted off the floor and returned to her bed. Within half an hour she

recognized and spoke to her husband. Next day she was confused and remembered nothing of what had happened during the previous 3 days. Mental recovery was complete. She complained only of pain over the xiphisternum, which had been injured by the compression. She left hospital 3 weeks later. Her auricular fibrillation had not been corrected by the defibrillating shock.

We have had 2 cases of successful resuscitation of cardiac standstill by external praecordial thumping (Table VI). Both responded and massage was unnecessary. Five out of seven cases of cardiac arrest have therefore been successfully resuscitated without opening the chest. I very much doubt whether these results could have been duplicated by open-chest resuscitation. It is upon this experience that I feel impelled to advocate closed-chest resuscitation.

*Post-resuscitation mortality.* The complications that develop after resuscitation depend largely upon the effectiveness of the artificial circulation during the period of arrest. Rapid revival will usually have the happy result of prompt complete recovery. Delay, prolonged or ineffectual massage, and cardiac trauma from rough handling, will lessen the chances of uncomplicated recovery. Fifteen of our patients died after the heartbeat had been restored, 3 from causes unrelated to the cardiac arrest as follows: (1) One died from massive cerebral embolism owing to displacement of atrial clot during hasty commissurotomy because of the onset of ventricular fibrillation. (2) Another died after 18 days from massive myocardial infarction. The atrium had split during valvotomy, causing massive haemorrhage. The left circumflex coronary artery was occluded by a suture in controlling haemorrhage from the depths of the atrioventricular groove. This precipitated ventricular fibrillation but resuscitation was none the less possible. (3) After one successful external resuscitation the patient died during the second day from massive recurrent gastric haemorrhage, the condition for which he was originally treated.

*Cerebral complications.* Twelve patients recovered after resuscitation, with cerebral symptoms (Table VII), of whom 7 died from 3 hours to 6 days later. The signs ranged from

TABLE VII. POST-RESUSCITATION COMPLICATIONS

| | *Patients* | *Survivors* | *Deaths* |
|---|---|---|---|
| Cerebral .. .. .. | 12 | 5 | 7 |
| Hypotension .. .. .. | 5 | 2 | 3 |
| Arrhythmia .. .. .. | 5 | 3 | 2 |
| Empyema .. .. .. | 1 | 1 | 0 |

cerebral irritation to profound coma. None with the latter survived. Cerebral irritation has lasted 2 - 3 weeks with survival. Patients with post-resuscitation cerebral symptoms should be submitted to tracheostomy and should probably be treated by mechanical ventilation. Full oxygenation must be maintained, and mechanical ventilation is the only certain way of preventing respiratory failure.

*Cardiac complications.* Hypotension and cardiac arrhythmia are common. These may be effects of myocardial anoxia during the period of arrest, and bruising of the heart may also be a factor. Two patients died from recurrent ventricular fibrillation within a few hours of resuscitation. One patient survived after a prolonged worrying period of atrial-flutter fibrillation. Extrasystoles, transient partial heart-block, and atrial fibrillation, have occurred. In these cases digitalis or procaine are helpful. Three patients with prolonged postoperative hypotension succumbed; they were treated with intravenous 'levophed' and showed minimal cerebral signs; they died from renal failure 4 and 8 days later (today they would have better chances of survival, in the hands of the hospital's Renal and Respiratory Resuscitation Units).

## RECOMMENDATIONS

No-one can say that a heart that has stopped suddenly and unexpectedly cannot be revived. The report of Jude *et al.*,[25] which is supported by our own results, shows that 70% of sudden cardiac arrests can be reversed. No-one can predict that a particular person whose heart has unexpectedly stopped will, if resuscitated, not have a long or useful or happy life ahead. Kouwenhoven has reported a 62% survival rate out of 114 cases of cardiac arrest, of which 89 (with a survival rate of 53%) occurred outside the operating theatre or even outside a hospital.[36] In the face of unexpected cardiac collapse there is no place for philosophical debate on the merits of reviving an apparently dead person. The patient's life depends on the resourcefulness of the nearest man. Training and organization can prepare him to act correctly. This can best be fostered by forming a hospital cardiac resuscitation committee to educate the staff in resuscitation techniques and to establish an emergency routine. One envisages this committee's functions as follows:

1. *Education.* The medical and nursing staff should attend demonstrations on resuscitation techniques. Emphasis should be on practice, not theory. The effort should be to establish a set drill. Repetition is necessary so that reaction is automatic. Interns, registrars and nurses should practise the routine in their wards or departments.

2. *Organization and equipment.* The hospital should be divided into zones each fully equipped so that there is no need to send elsewhere for drugs or instruments. Each zone must possess a mobile resuscitation trolley containing all the necessary drugs (there are only 4—adrenaline, calcium chloride, atropine, and procaine), a thoracotomy set, an internal and external defibrillator, a suction apparatus, and if possible an ECG oscilloscope. Several mouth-to-mouth airways must be kept in every ward, operating theatre, and special department, for with this and external massage the brain can be kept viable until the resuscitation trolley arrives. Laryngoscopes, intratracheal tubes, airways, facemasks, rebreathing bags, and a simple ventilator, should be kept in the trolley. Drugs must be fresh, electric points in good repair, oxygen cylinders full, and the defibrillators in working order.

3. *An alarm system.* Some system must exist whereby assistance can be summoned to the emergency. A loudspeaker would be the most effective so that precise directions could be given. It should be used only for emergencies, not for routine communication.

4. *Enquiries.* After every unexpected cardiac arrest, whether successfully resuscitated or not, the committee should meet the medical and nursing staff involved. A frank discussion is required and a pathologist's report should be available if the patient died. Each step must be analyzed to learn what mistakes were made, so as to avoid them in the future. Only good can come from calm scrutiny, but the enquiry must be a domestic affair without judicial air and without intent of apportioning blame. Acute cardiac arrest is a desperate affair and failure can never be blamed. Neglect to make the attempt can be.

## SUMMARY

1. The history of the development of internal and external methods of resuscitating the acutely arrested heart is traced. Several cases, believed to be among the first successful resuscitations of the fibrillating heart in South Africa, are presented.

2. A personal experience of the treatment of 63 cases of sudden cardiac arrest is described. Since this article was prepared 3 further cases have occurred (see Addendum). The factors responsible for the catastrophe are analyzed. In thoracic surgical practice anoxia is the factor most often responsible. Viscerocardiac reflexes and cardiac irritability are sometimes the prime cause and in these cases successful resuscitation, provided it is instituted promptly and effectively, should always be possible.

3. The techniques of open and closed cardiac resuscitation are described. The methods advocated by Kouwenhoven and his colleagues, of the Johns Hopkins Hospital, have been used in 10 cases with 7 recoveries (70%). Prompt artificial ventilation, effective external massage, and external electrical defibrillation as soon as is possible, will restore the heartbeat in as many as 70% of cases of sudden cardiac arrest following conditions such as coronary thrombosis, anaphylactic reactions, and acute anoxic arrests. Resuscitatory efforts must be immediate and effective. The chest wall must be seen to move freely, a pulse must be felt with each cardiac compression, and the pupils must remain small. These conditions can be attained in all cases where immediate skilled treatment has been instituted. At every hospital an external electrical defibrillator is an essential item of equipment in every ward block, operating theatre, radiology department, and casualty department. External methods of resuscitation should replace internal massage and direct heart defibrillation.

4. Recommendations are made concerning the establishment of hospital committees to encourage the teaching of methods of resuscitation to hospital staffs. Only those present can be of assistance to a person who has collapsed with cardiac arrest.

I must thank Mr. L. Fatti, F.R.C.S., for allowing me to include in this survey cases he has treated. He pioneered cardiac surgery in this country and had to face the frustrations and disappointments of cardiac arrest at an early stage. It is because of this experience and his efforts to overcome the problem that I am able to present a series of cases with results equal to the best reported in the literature.

## ADDENDUM

Since the completion of this paper my Unit of the Department of Thoracic Surgery has been concerned with the resuscitation of 3 further cases of acute cardiac arrest (Table VIII). Dr. P. Obel and Dr. W. Scott each resuscitated a case and Dr. van Hasselt was involved with myself in a third. All the attempts were successful in restoring cardiac rhythm, but one patient died as a result of irreversible cerebral damage. The total is therefore 7 successful resuscitations out of 10 attempts (70%).

TABLE VIII. ADDITIONAL CASES OF ACUTE CARDIAC ARREST TREATED ENTIRELY BY CLOSED METHODS SINCE APRIL 1963

| *Age and sex* | *Cause* | *Site* | *Duration of resuscitation* | *Comment* | *Survival* |
|---|---|---|---|---|---|
| 55M | Stokes-Adams attacks with ventricular fibrillation | Ward & X-ray dept. | 3 Episodes each lasting 2-10 minutes | Repeated attacks occurred over course of 3 hours before a catheter electrode was introduced to control attacks | Yes |
| 65M | Anoxia owing to tracheal obstruction by pus. Bilateral pneumonia | Foyer of operating theatre | 14 min. | Complete cerebral and cardiac recovery | Died suddenly (heart failure) 3 weeks later |
| 40F | Anoxia during attack of status asthmaticus | Ward | 30 min. | Profound cerebral damage | Died 4 days later |

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