CARDIAC ARREST

FEW TOPICS merit more frequent review than cardiac arrest. The occurrence of sudden cessation of circulation in a patient in whom it is not expected is a catastrophe which demands prompt and proper resuscitative efforts to prevent death or serious neurological injury. This catastrophe is increasing in frequency, and probably occurs between 8 and 9,000 times each year in the United States.

Investigation into the cause of circulatory arrest—and this includes ventricular fibrillation as well as cardiac standstill—reveals that, with the exception of ventricular fibrillation following electric shock, every instance of cardiac arrest appears to be due to a combination of circumstances. Some of these causes are understood and many are not. Among the agents recognized as contributing to cardiac arrest should be listed: hypoxia, hypercapnia, acidosis, vagal reflexes, and depressant drugs.

Anoxia or hypoxia has been considered by many surgeons as the most important cause of cardiac arrest. Hypoxia does not consistently result in sudden circulatory arrest in the laboratory animal, and, fortunately, cardiac arrest usually does not follow its clinical appearance. Although the association of hypoxia and circulatory arrest has been emphasized by Gerbode, Johnson, and Kirby 

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Hypercapnia, the accumulation of carbon dioxide in the body, may occur even in the presence of adequate oxygenation. Hypercapnia results in respiratory acidosis and hyperkalemia, and in its presence vagal inhibition of the heart is potentiated. There is little doubt that this may contribute to circulatory arrest in some cases. It follows that hypercapnia, and especially sudden changes in CO₂ level, should be avoided.
The importance of a vagal or vago-vagal reflex in causing cardiac arrest is often discussed but remains unsettled. Stimulation of the central end of the vagus nerve will cause ventricular standstill in the experimental animal, but even with continued stimulation vagal escape occurs. There are apparently no vagal fibers of the ventricles of the human heart. Reid, Stephenson, and others conclude the vagal stimulation will not cause cardiac arrest in the normal healthy heart. In the presence of myocardial disease or temporary myocardial depression by drugs, vagal inhibition of the heart may be of great importance.

Admitting that knowledge of the etiology of cardiac arrest is incomplete, it becomes important to recognize those situations in which it is likely to occur. The majority of instances of circulatory arrest are encountered in the operating room and at the present time occur once in approximately every 1,500 operative procedures. It does not seem coincidental that the first recognized case of cardiac arrest was reported within a few months of the introduction of anesthesia. Anesthetic procedures offer the hazard of vagal stimulation during intubation and extubation, the possibility of hypoxia and hypercapnia, and the depressant effect of anesthetic agents themselves. Beecher has pointed out the apparent increase in incidence of cardiac arrest when using multiple anesthetic agents, particularly including curare-like drugs.

Circulatory arrest during operation is more likely to occur when no pre-anesthetic medication is given, in poorly apprehensive patients, in children, and in patients with pre-existing heart disease. Furthermore, arrest has been reported most frequently during biliary and pulmonary operations. An increase in frequency during cardiac operations is not surprising.

Outside the operating room, cardiac arrest is well known in the recovery room, emergency room, endoscopy clinic, and radiology department, and its occurrence in any of these areas is associated with a poor prognosis.

Recognition of cardiac arrest is simple but deserves emphasis because of its urgency. Absence of pulsation in major arteries is all that is necessary to establish the diagnosis. Beck and others have repeatedly stated that more elaborate determinations only delay resuscitation. Recently, monitoring of the pulse during operation has been advocated, and it seems likely that some simple monitor with a warning system will come into common if not routine use in the future.

Successful management of cardiac arrest requires perfusion of the tissue with oxygenated blood within four minutes of cessation of circulation. The initial problem is restoration of an airway and adequate ventilation. This can be done by mouth-to-mouth breathing, as recently advocated by Elam and Benson, by mask breathing using an anesthesia machine, or preferably by endotracheal intubation. When good efforts to restore ventilation are begun, the managing physician should begin circulatory restoration by cardiac massage through the open chest. If operating in the abdomen, preliminary massage can be carried out through the intact diaphragm while the chest is being opened. It is important to emphasize to untrained persons how easily the heart itself may be cut in a too rapid thoracotomy. Massage is facilitated by opening the pericardium. The position of the surgeon during massage and the method of holding the heart are matters of individual preference. The possibility of myocardial injury or actual rupture from too vigorous massage should be mentioned. The initial rate of massage is 70 to 100 per minute, and the criteria of successful manual systole is the detection of a palpable peripheral pulse. Once massage is begun the requirements of the tissues for oxygen can be met and the problem becomes one of restoring a heart beat. After myocardial tone is regained, heart beat will usually begin spontaneously. Drugs are useful when myocardial tone does not respond to massage or when ineffective beat is initiated.

The appearance of ventricular fibrillation during cardiac resuscitation is common and not necessarily ominous. Defibrillation using an electric shock should be attempted after massage has restored good tone and color to the myocardium. If the usual single shock
at 120 volts (1.5-2 amp.) is ineffective, multiple shock and higher voltage should be used after intervening periods of massage. The value of high voltages, particularly in large hearts, has not been generally appreciated.

Several drugs may be helpful in cardiac resuscitation. Epinephrine is used to improve myocardial tone and conduction. It is injected directly into any accessible chamber of the heart. The use of epinephrine is not contraindicated in ventricular fibrillation. Calcium, as emphasized by Doctor Blick, increases myocardial contractility, but may be contraindicated in patients who have received digitalis.10

Circulatory arrest is always accompanied by metabolic acidosis. As Campbell has pointed out, acidosis diminishes response of the heart to presser amines. For this and other reasons, sodium bicarbonate should be injected intravenously in each case of cardiac arrest.

Isuprel may have a place in the treatment of intractable ventricular fibrillation.11 Potassium, barium, procaine, and heparin have been advocated but at the present time their value is uncertain.

Other stimulant drugs appear to have little place in the treatment of cardiac arrest.

During cardiac resuscitation, several adjuvant procedures are useful.

The patient should be placed in the Trendelenberg position to aid in perfusing the central nervous system, as well as to aid venous return to the heart.

Compression of the thoracic aorta may be advantageous in increasing flow to the brain. This occlusion should be relieved at 15 minute intervals to avoid spinal cord injury.

The influence of coronary perfusion on cardiac arrhythmia is very impressive during procedures involving cardio-pulmonary bypass. Some form of aortic perfusion should be considered in the unusual instances in which it is feasible.

Cardiac pacemakers are being widely advertised as essential hospital equipment. Electronic pacemakers are effective in treating ventricular asystole due to a conduction defect as in surgical trauma to the Bundle of His or in the Stokes-Adams syndrome due to other causes. They will not initiate effective contractions in the usual case of cardiac arrest.12

Defibrillation of the heart can be accomplished through the intact chest wall using the method and apparatus described by Kouwenhoven and Knickerbocker.13 This has been particularly useful in the cardiac catheterization laboratory where ventricular fibrillation is a definite hazard and where the diagnosis can be instantly made from the oscilloscopic monitor of the electrocardiogram. Closed chest defibrillation of the heart has also been successful after myocardial infraction and represents a real advance in the treatment of this form of cardiac arrest.

Recently, unpublished work by Kouwenhoven and Knickerbocker has shown that in the dog compression of the closed chest is effective in restoring circulation and arterial pressure up to 90 mm. hg. can be obtained.14 This procedure was investigated as a possibility in lengthening the period of effectiveness of closed chest defibrillation but has clinical possibilities of its own. Doctor James Jude of the Johns Hopkins Hospital has now successfully restored heart beat in three patients with asystole using external compression of the chest.9 This procedure cannot yet be placed in our permanent armamentarium, but it may find important uses in the future.

When the heart does not respond to resuscitative efforts, there is a difficult decision as to when to discontinue resuscitation. There can be no fixed rule in this regard, but it should be mentioned that recovery has followed periods of massage exceeding four hours. The length of time between arrest and massage and the presence of persistently dilated pupils are important factors in the decision to abandon resuscitation.

The over-all prognosis for permanent survival in cardiac arrest is exceedingly difficult to estimate, but the figure given by Stephenson is 29 per cent. There is no doubt that rapid recognition and prompt treatment will result in a high survival rate. In
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The major cause of death after successful restoration of heart beat is injury to the central nervous system. The clinical picture is that of unconsciousness and progressive decerebrate rigidity ending either in death within hours or survival with neurologic damage. Very occasionally recovery follows definite evidence of brain injury. It has been suggested that death is caused by brain swelling, which occurs in response to the original anoxic injury. If this is true, prevention of brain swelling might allow survival in cases when the original injury is not too severe. A series from the Johns Hopkins Hospital has been reported in which patients with evidence of central nervous system injury after cardiac arrest were treated by inducing hypothermia with impressive results,19,20 Spencer and Zimmerman have shown an increase in survival rate in animals subjected to hypothermia after circulatory arrest,21 and unpublished data from the University of Oklahoma indicates that the brain does swell following circulatory arrest.22 Work on the use of Urea after cardiac arrest has recently been presented by Javid at the Congress of the American College of Surgeons.23 At the present time, the induction of moderate hypothermia, or the use of Urea, seems justified in patients who show evidence of central nervous system injury after cardiac resuscitation.

A final word on the role of the surgeon in the problem of cardiac arrest. The surgeon logically has the greatest experience and interest in this catastrophe, and it is urged that he assume a hospital wide responsibility for efforts to prevent, recognize, and treat cardiac arrest. Someone must emphasize the continued necessity for proper evaluation of pre-operative patients, the value of pre-operative medication, and the need for special anesthetic care and perhaps monitoring during some procedures. Someone must see that the equipment for resuscitation is available to remote areas. Most

BIBLIOGRAPHY