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Cardiac arrest and a review of emergency cardiac resuscitation

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CARDIAC ARREST AND A REVIEW OF EMERGENCY CARDIAC RESUSCITATION

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Attempts at reviving the patient whose heart has ceased beating have been made for many years, but only recently have a considerable number of these patients survived. Several methods of resuscitation have been described and utilized in the cardiac arrest patient. Of great interest to physicians today is the cardiac arrest patient and an evaluation of the methods which are being used for resuscitation.

Cardiac arrest is a relatively new technical expression; the condition it describes has been closely associated with anesthesia, but it can and has occurred under many kinds of circumstances. It can take place under local or regional anesthesia as well as under general anesthesia. It may strike during a simple procedure as well as during a long complicated one. Cardiac arrest occurs both in the young and in the old, in the patient with a good heart as well as the individual with a known heart impairment.

Cardiac arrest is the sudden cessation of demonstrable cardiac activity unexplained by gross hemorrhage, shock, or asphyxia.[3] There are only a few situations in medicine today where the physician feels almost completely helpless, and one of these is the sudden and unexpected cessation of cardiac activity.

Cardiac arrest is a much more frequent occurence than is generally realized. Many areas are able to report incidence as high as one case in one thousand anesthetics. Considerable data indicate that the actual incidence of cardiac arrest is increasing. Stephenson's[17] study showed that 12-20% of cardiac arrest patients

have the arrest outside the operating room. The major portion of cardiac arrests occur on the operating table, with most large hospitals having several cardiac arrests in the operating room every year. The disaster can occur at any point in the hospital and even outside the hospital in the home or in public.

Stone[18] reports that in his study of 148 arrests, eighty-seven occurred in the operating room and sixty-one in other areas of the hospital. The majority of arrests which occurred outside the operating room took place in the emergency room in seriously ill patients or with moderate-to-severe trauma.

Unfortunately, cardiac arrest does not occur only in the elderly or chronically ill. It may strike the relatively young adult who from all standards appears to be in good health. Electrocution, drowning, and suffocation from many causes occur daily. Sudden cessation of cardiac activity, when it is a sharp deviation from the course one would ordinarily expect from the patient's state of health, is certainly an indication that one should attempt some sort of resuscitative measure.[9]

The two conditions of cessation of the heart beat that confront us are cardiac standstill and ventricular fibrillation. Fibrillation exists when one or more areas of localized anoxia develop in the heart muscle, giving a checkerboard pattern of anoxia and distribution of oxygen. There is complete incoordination of the muscle fibers -- each fiber and fibril contracting at its own rhythm. The well-oxygenated pink muscle in juxta-

position to poorly oxygenated blue muscle produces an electrical condition which leads to muscular fibrillation. In cardiac standstill, the entire heart becomes anoxiated and is blue all over. A uniformly anoxiated heart such as this maintains equilibrium and does not fibrillate. The electrical response or discharge of the anoxic heart is sometimes normal even after the heart stops beating and may even persist for several minutes after the muscle itself fails to respond. If red blood or oxygenated blood flows to the anoxiated heart it frequently fibrillates. If the red blood is perfused into a branch of a coronary artery of a blue heart, fibrillation will most likely be produced. This blood produces no injury to muscle nor does it interfere with the electrical current through the heart. Therefore, fibrillation is dissociated from injury. We can assume it is produced by oxygen differential in the heart muscle itself and not by injury. This separation from injury explains many clinical paradoxes in which fibrillation and death occur without apparent injury and without any signs of recent disease in arteries or muscles.[2] Fibrillation can occur in any patient, even the young, energetic person. Standstill is more likely to occur in the severely damanged heart which becomes anoxiated. The baby with severe congenital heart disease usually dies with the heart stopping in standstill. In paralysis of the respiratory center, such as with brain tumor, the heart stops in standstill. In pneumonia, emphysema, cardiac failure, asphyxia choking, and drowning, the heart usually stops in standstill and there is usually no anginal pain associated with these

conditions. Fibrillation occurs in the presence of coronary artery disease with incomplete oxygenation of the heart muscle. A person who is electrocuted may develop fibrillation of the heart; a person who is struck by lightening also dies in cardiac fibrillation.

The most highly developed organ in the body is the brain and it is most easily damanged with lack of oxygen. Most authorities agree that the presence of anoxia for between three to five minutes will result in irreversible damage to the brain. The most highly developed centers as those for speaking, thinking, and memory are injured the quickest and are most likely to be permanently impaired by periods of anoxia greater than from three to five minutes. On many occasions cardiac resuscitation has restored the heart beat with the patient expiring some twelve to forty-eight hours later from cerebral edema because the circulation was not restarted soon enough following cardiac arrest.

The metabolism or oxygen needs of individuals vary. To illustrate, some persons are able to tolerate much higher altitudes than others before blacking out. The reaction of the cardiovascular system to diminishing oxygen shows considerable individual variation. In many cases of cardiac arrest there may be a certain degree of anesthetic cerebral hypoxia preceding the actual stopping of the heart. This would have a definite influence on the safe time limitation. In such a case it would be incorrect to assume that the brain had a deficiency of oxygen only from the time of

cessation of the heart beat. Here a certain degree of cerebral injury would have taken place before the anoxia of cardiac arrest.

There are several factors which must be considered in the cause and effect of cardiac stoppage, one of the more important ones being age. Briggs[3] feels that there is a steady increase in cardiac arrest beyond the age of thirty years, but here one must also consider the fact that there is an increase of the number of older people undergoing surgery along with an increase in the number of poor risk patients. In his study he determined there was one cardiac arrest for every 4,358 surgical and anesthetic procedures in age groups of from twenty to thirty years. In the age group of over eighty years, there was an arrest for every 219 procedures. Briggs table of statistics is as follows:

Decade	Percent of cases of surgical cardiac arrest in each age group over a ten year period
0 - 9 yrs.	5.0%
10 - 19	7.4%
20 - 29	12.6%
30 - 39	15.4%
40 - 49	17.2%
50 - 59	17.1%
60 - 69	15.0%
70 - 79	8.2%
80	1.9%

Stone[18] reports in his study of 148 cases of cardiac arrest that there is a relatively even distribution of approximately twenty cases of arrest for each decade of life up to the eighth decade in which group only seven cases were found.

Sex is another factor considered by Stephenson in cardiac

arrest. He reports that in every one hundred cases of cardiac arrest, sixty-one occur in the male and thirty-nine in the female. This ratio may indicate that the male is more susceptible to cardiac arrest or possibly that the male undergoes more operations of a type which predisposes to cardiac arrest.

Pre-operative condition of the patient is a very important factor in cardiac arrest. Poor pre-operative physical status predisposes to cardiac arrest. This is especially true if the patient has underlying cardiac disease. The patient with clinical evidence of cardiac disease such as coronary artery disease, generalized arteriosclerotic heart disease, hypertension, congenital heart disease, or acquired heart disease appear to be more prone to become victims of cardiac stoppage.

Vago-vagal reflex is important in the etiology of cardiac arrest and anoxia and hypoxia potentiate this reflex. Anoxia itself is felt to accentuate the inability of the specific tissue to form stimuli, especially when the latter has been depressed by the action of the anesthetic. Vagal reflexes may occur during tracheal intubation, during esophageal, tracheo bronchial or oral procedures, and during visceral manipulation (cardiac, great vessels, or biliary).

Hemorrhage is another important factor in the etiology of cardiac arrest. A considerable number of patients have excessive bleeding before surgery and others have no replacement of blood during surgery -- both contribute to heart stoppage.

Anoxia is a very important cause of arrest. The immediate effect on the brain and myocardium are the most serious. The heart and lungs may fail to function simultaneously or one may fail before the other. The functions of the heart and lungs are so interdependent that when one fails, failure of the other will follow shortly.

Improper utilization of drugs such as atropine may be listed as a cause or contributing factor of cardiac arrest. Atropine is a valuable drug in protecting the heart from reflexes that may be instituted through the vagus nerve or vago-vagal reflex. The vago-vagal reflex produces an overabundance of acetylcholine. The heart is sensitive to acetylcholine and may cease to beat if the vago-vagal reflex is stimulated. Some of the improper uses of acetylcholine are as follows:

1. Insufficient dose of belladonna as premedication.
2. Delay in beginning anesthesia or the operation, the medication having been given several hours before.
3. Prolonged operative procedures without readministering the atropine.

One important sign of cardiac arrest from the insufficiently suppressed vago-vagal reflex is a severe bradycardia. Anoxia can also cause bradycardia, therefore adequate pulmonary ventilation with oxygen must be maintained. Sudden shift in ph, hemorrhage, massive transfusion of citrated blood, cardiac arrhythmia induced by hypothermia or direct operation on the heart may cause cardiac

arrest in the operating room.

Drug reactions and the frequent practice of administering several drugs may be the cause of cardiac arrest. In the majority of instances the difficulty is over-dosage. Often the anesthetic agent itself appears to have a depressive affect on the myocardium. This is especially true in the elderly and the diseased. There is also an increase in cardiac arrest with the greater depth of anesthesia.

Outside the operating room and recovery room, coronary occlusion is the most frequent cause of cardiac arrest. Other causes of cardiac arrest are anoxia secondary to intracranial trauma, over-dose of sedatives, airway obstruction, altered pulmonary function due to trauma, excessive blood loss or chronic lung disease. Other causes include excessive blood loss, electrocution, and cardiac tamponade.[18]

Diagnosis of cardiac arrest must be prompt in order to begin resuscitative measures as soon as possible. Essentially the absence of peripheral pulse and disappearance of heart sounds indicate the heart has stopped functioning. The patient's color may be cyanotic and respiration may be gasping or absent. In some cases the heart may continue to beat after respirations have ceased. The pupils also dilate and do not respond to light. One should not waste time taking blood pressure and electrocardiogram unless the equipment is already attached to the patient. Also, one should not be misled by persistence of regular electrical

activity on the electrocardiogram when pulse and heart sounds have disappeared. In this instance the heart, even though it may be beating, has lost its effectiveness as a pump and resuscitation measures must be taken.[7]

This is the critical point where time is of extreme importance. Every study that has been made shows that the treatment of cardiac arrest must be instituted within three to four minutes after the heart has stopped. A delay of four minutes after the last beat will result in severe damage to both brain and viscera. In Stahlgren's[15] study, 94% of the patients who survived after cardiac arrest had resuscitation started within four minutes after the heart stopped. The other 6% who survived had resuscitation started over four minutes after cardiac stoppage and these individuals had many residual neurological defects. These studies were based on open chest cardiac massage which means the patient's heart had to be exposed and massaging action initiated within four minutes to save the patient from serious damage.

Several methods of cardiac resuscitation have been attempted and advocated. Thumping on the chest was one of the oldest methods used, along with mouth to mouth respiration. The first successful cardiac resuscitation reported was by Lane in 1902 in the operating room, but there was little development for nearly fifty years.[5] In 1947 Beck had the first successful treatment of ventricular fibrillation. Some progress has been made in the field, but revival of people from clinical death appears to be in its infancy

even today. Intensive research is being carried on in various countries throughout the world in this field.

Presently there are two major methods of cardiac resuscitation. These are open chest cardiac massage and closed chest massage. Electrical defibrillation can be utilized with either method.

Resuscitation requires that two basic functions be restored simultaneously. These are (1) blood must be pumped through the body and (2) there must be a gaseous exchange of oxygen and carbon dioxide. The brain will not survive unless the blood it receives has been oxygenated nor will the blue cyanotic heart defibrillate until it receives oxygenated blood. When arrest occurs, an attempt should be made to inflate the lungs with oxygen through an endotracheal tube as soon as possible. If equipment is not at hand at the time of arrest, mouth-to-mouth respiration or oxygen by mask should be used.

Open chest cardiac massage with an emergency thoracotomy is the oldest method of resuscitation, having been used for many years. Only one instrument is essential for the emergency thoracotomy and this is the scalpel. The operator should have a thorough and complete plan of action when attempting cardiac resuscitation. There are some procedures which should not be done, mainly because they waste valuable time. One should not delay excessively by listening for faint heart sounds. Do not change blood pressure cuff to other extremity to detect blood pressure. Do not wait for an electrocardiogram. Do not inject epinephrine through the chest wall into

the heart. Do not give a blood transfusion.

In the emergency thoracotomy the incision is made without preparation of the skin and without gloves. The incision is made parallel to the ribs, and the left pleural cavity is entered through the fourth or fifth intercostal space and extending from the sternum to the mid-axillary line. The ribs are forcibly spread apart and maintained in this position by inserting between them a short tube or some other available object. The hand is then thrust into the chest cavity and intermittent compression of the heart is begun immediately through the intact pericardium at the rate of sixty to eighty per minute. Small hearts can be compressed between the thumb anteriorly and four fingers posteriorly. Larger hearts may require bimanual compressions. The effectiveness of the compression can be checked by the character of the peripheral pulse and contraction of the pupils. If, after several minutes of cardiac compression the heart does not start beating, the pericardium should be opened to determine whether fine ventricular fibrillation is present. Resistant cases of standstill may respond to the use of certain drugs as outlined below. These drugs are injected into the chamber of the left ventricle, and compression of the heart forces them into the coronary circulation.

DRUGS USED IN CARDIAC ARREST

	DRUG	DOSAGE
I.	Epeniphine hydrochloride	1-2 cc 1:10,000 sol.
II.	Isoproterenal hydrochloride	1 mg. per kg.

	DRUG (Continued)	DOSAGE
III.	Calcium chloride	2-4 cc 10% sol.
IV.	Procaine hydrochloride	2-5 cc 1% sol.
V.	Procaineamide (Propestyl)	150-300 mg.

INDICATIONS FOR DRUG LISTED ABOVE

I. For flabby, dilated heart in standstill. May cause fibrillation.

II. For standstill. Stimulate S.A. node. Less apt to cause ventricular fibrillation than epeniphine.

III. To increase amplitude of contractions in feebly beating heart. May cause ventricular fibrillation.

IV. For persistent or recurrent ventricular fibrillation.

V. For irritable myocardium. Longer action than procaine.

Ventricular fibrillation, if present originally or if it develops after cardiac standstill, may sometimes be converted to normal sinus rhythm by electrical counter shock. Electrical defibrillation is most easily accomplished after the heart has been massaged and has regained its tone and pink color. Staryl[16] states that if the electrical pacemaker cannot be immediately applied, it is likely to fail even in the most favorable cases. He feels that in some cases the assumption that cardiac arrest is due purely to failure of the conduction system is not warranted and the arrest is actually the result of anoxia and hypercapnia in which the myocardium has been damaged. Often after cardiac arrest has been diagnosed, the attending person or persons may waste

valuable time attempting to procure and apply an electrical pacemaker or defibrillator. When electrical stimulation is finally started, the myocardium will be incapable of contracting. Thus, the primary therapy of cardiac arrest should still be cardiac massage, in order to keep some blood circulating to the heart and vital centers. A defibrillator or pacemaker may be obtained if available and applied later if there are indications for it. According to Jude[8] some 30% of cardiac arrests were with the heart in ventricular fibrillation. Nearly all these patients required defibrillation before spontaneous cardiac action could be returned. External electrical defibrillation has been developed by Hooker, Kouwenhoven, and Zoll. The fibrillation should be vigorous and rapid in order for defibrillation to succeed. This can occur only with a well oxygenated myocardium. Thus it is important to establish circulation by external heart massage while the defibrillator is being brought to the scene. The voltage recommended is 440 volts A.C. applied across the longitudinal axis of the heart for .25 seconds.

Jude states that the availability of an electrocardiogram may not always be present. Since it is desirable to reestablish cardiac action as soon as possible, it may be desirable to use blind application of the defibrillating shock when cardiac action has not resumed after five or six minutes of good ventilation, external massage, and drug therapy. He states that experimental studies on dogs have shown that 440 volts A.C. shocks for .25 seconds have

not had any detrimental effect on the normally beating heart.

The system of closed chest cardiac massage is a relatively new method of revival of cardiac arrest patients although there is a long history of similar experiments of attempts at maintaining circulation and revival of animals and patients. First attempts at cardiac resuscitation were made by Niehaus in 1880, but the first successful case was that of Igelerud in 1901.[18] Tournade and his co-workers were able to produce a blood pressure of 60-100 mm of mercury in dogs with cardiac arrest. Killish and Eve used a rocking technique of artificial respiration where the patient was tilted at a 60° angle from the horizontal in alternating head-down and feet-down positions. Their experiments showed a change of blood pressure in the atrium from 38-76 mm of Hg; they hypothesized that this change in pressure was sufficient enough to cause adequate flow to nourish the heart and brain. Rainer and Bullough treated cardiac arrest in the small child by lowering the head about 10° and placing an arm under the patient's knees and flexing the legs and thighs against the chest. They recorded eight successful resuscitations in children from two months to thirteen years of age.[10]

The most recent method of external heart massage was described by Kouwenhoven.[10] This procedure consists of pressure being applied to the lower sternum by the palm of the hand. The patient is placed in the supine position on a rigid support, such as the floor. The operator assumes a kneeling position, usually on the right side

of the patient and facing the patient. The operator should be positioned so he can use his body weight in applying pressure. The heel of one hand is placed over the lower sternum just cephalad to the xiphoid and the other hand is placed on top of it. Firm pressure is applied vertically downward from fifty to seventy times per minute. At the end of each pressure stroke the hands are lifted slightly to permit full expansion of the chest. The sternum should move 3-4 cm toward the vertebral column. The anatomy of the bony thorax is such that by compressing the sternum downward toward the vertebral column, the heart is squeezed between the two bony structures. The pericardium restricts the lateral movements of the heart, thus the heart is made to occupy a smaller area, forcing blood out the aorta into the systemic circulation. Relaxation of the pressure allows the heart to fill again. In the unconscious individual the thorax is unusually mobile, thus aiding in the resuscitation.

There is also some ventilation of the lungs accomplished by the cardiac massage and, if only one person is present, the cardiac massage should be the primary measure used for resuscitation. However, if more than one person is present, one should attempt to provide ventilation through mouth-to-mouth respiration.

In the adult patient the first few pressures are said to loosen the costal junction to allow adequate depression movement of several centimeters without fracturing the ribs. Erickson[4] reports in his study that the external cardiac massage was used on

twenty patients with an overall survival rate of 72%. Reports of being able to develop up to 150 mm of mercury by this method have been recorded in which systolic pressures of only 70-80 mm of mercury were recorded as maxiumun with open chest manual massage in the same patient. Records were made by indwelling catheters.

Several studies have been made comparing open chest and closed chest cardiac massage. Redding[12] made such a study using dogs in attempting to demonstrate whether closed chest cardiac massage was less effective in maintaining circulation than the open method. He utilized dogs in which ventricular fibrillation was produced and were placed under positive pressure breathing. In some of the dogs, closed chest cardiac massage was started thirty seconds after their hearts were started fibrillating. Massage was accomplished by compressing the sternum against the vertebral column at a rate of one hundred times per minute. This was carried on for twenty minutes and then an external defibrillator was applied over the manubrium sternum and over the apex of the heart with 480 volts shock for .25 seconds. On another group of dogs a similar procedure of starting ventricular fibrillation was employed, but instead of closed chest massage, a thoracotomy was performed and manual compression of the heart begun for twenty minutes. Then the electrode of an internal Berkmire defibrillator was applied to the ventricle (110 volts for .5 seconds). In both groups the carotid blood flow was determined just before initiation of ventricular fibrillation, then after one, five, ten, fifteen,

and twenty minutes of fibrillation and then again after one minute and thirty minutes following defibrillation. His results showed that the blood pressure in all the dogs dropped to zero immediately following initiation of fibrillation. In the dogs receiving the closed chest cardiac massage, the aortic pressure ranged from 35/0 mm to 80/35 mm of mercury in the first minute and carotid flow from 2.7 ml per minute to 15 ml per minute. Nine of the ten dogs reverted to a sinus rhythm immediately following the administration of 480 volt shock; the other dog required .8 ml of adrenalin and a second shock to restore circulation. The dogs that received the thoracotomy and open chest cardiac massage had a delay of 25-50 seconds while the chest was being opened before massage was started. The first minute of massage with the pericardium unopened resulted in an aortic pressure varying from 35/15 mm to 100/50 mm. Carotid flow was 5 ml per minute to 13 ml per minute. Each of the ten dogs reverted to sinus rhythm immediately following administration of 110 volts. Thirty minutes following restoration of circulation, the carotid flow was from 7-100% of that in the control group. The dogs receiving closed chest massage had carotid blood flow of 40-100% of that in the control group at thirty minutes following restoration of circulation.

The conclusion of this experiment was that within limits of the experiment, the artificial circulation produced by closed chest cardiac massage was comparable to that produced by thoraco-

tomy and manual systole. One must consider the difference in human and dog to relate it to similar responses and results one could expect in the case of human cardiac arrest.

Another experiment was discussed by Kouwenhoven[10] in which one hundred dogs were used. Adequate circulation was maintained by external cardiac massage for periods as long as thirty minutes with the dog in fibrillation. A closed chest defibrillatory shock resulted in immediate return of normal sinus rhythm. A simultaneous recording of blood flow in carotid artery, pressure from the femoral artery, and a cardiogram were taken during the procedure. The recordings demonstrated the normal pressure and flow before fibrillation, the fall of both during fibrillation and then the rise of blood pressure and carotid flow when cardiac massage was started. Vigorous fibrillation was maintained throughout the entire period and there was immediate return of normal sinus rhythm when the closed chest defibrillation shock was given. Both of these above experiments demonstrate that adequate cardiovascular circulation can be maintained in the dog by external heart massage.

Greenwich[6] was able to make recordings of aortic pressures in the human during closed chest cardiac massage. This cardiac arrest occured while retrograde left heart catherization was being performed . This afforded an opportunity to obtain direct aortic pressure while cardiac massage was being accomplished. The patient was a thirty-six year old white male who went into

ventricular fibrillation with the injection of hypaque. Attempts at transthoracic defibrillation with 150 volts internal defibrillator and also an external defibrillator resulted in no response. After five to eight minutes had elapsed, closed chest cardiac compression, as Kouwenhoven had described, was started. A blood pressure of 80/40 mm of mercury was obtained immediately from the catheter in the abdominal aorta and the patient's color improved considerably. He was ventilated continuously, first by mouth-to-mouth respiration for ten minutes and then intubated with intermittent positive pressure oxygen. After thirty minutes there was persistent fibrillation, so external massage was discontinued and thoracotomy was performed. As the pericardium was being incised, the rhythm changed spontaneously to ventricular tachycardia. Direct manual massage of the heart was started giving an aortic pressure of 100/65 mm of mercury. Adequate spontaneous cardiac contraction developed after a few minutes of cardiac massage and a fairly stable sinus rhythm developed with adequate blood pressure maintained for some time. Hypothermia was instituted to limit anoxia brain damage, but the patient never regained consciousness and died in a second bout of fibrillation. Post mortem exam revealed scattered focal hemorrhagic areas seen on cross-section of the myocardium, which were interpreted to be injuries sustained during direct manuel cardiac massage. No other abnormalities were found. This experiment illustrates that manual compression of the lower sternum can

produce substantial blood pressure in the distal aorta during fibrillation. The external massage pressure was recorded as being slightly less than direct massage. There were not injuries to the thoracic cage or lacerations of the heart, great vessels or other organs in this experiment.

Kouwenhoven[10] has reported in a study of twenty patients of various ages up to eighty years good success with external cardiac massage. Fourteen of the patients survived without central nervous system damage. The duration of massage varied from less than one minute to sixty-five minutes. He obtained records of blood pressure in seven of the cases with pressure of from 60 to 100 mm being recorded during the massage. Three of the patients were in ventricular fibrillation and it was necessary to utilize a closed chest defibrillatory shock to restore normal rhythm.

A typical case is one discussed at the Clinical Anesthesia Conference of the New York Society of Anesthesiology.[23] A twenty-nine year old white male was admitted for excision of tonsil tags. He was given no preoperative medications. Anesthesia began with oxygen and nitrous oxide. Halothane was given later and verbal contact was lost in four minutes. The nitrous oxide and halothane were then discontinued; succinylcholine was injected intravenously. An oraltracheal tube was passed with ease under direct supervision and then no pulse could be felt; the pupils dilated. The lower sternum was compressed at a rate of 40-50 times per minute and ventilation with oxygen maintained. A strong radial pulse was

obtained with each sternal compression. The pupils became miotic and spontaneous respiration began. Closed massage was continued for ten minutes. An electrocardiogram showed fibrillation and closed chest defibrillation was attempted without success. A thoracotomy was then performed with open chest direct defibrillation and cardiac rhythm returned to normal. The only after effect was two days of amnesia.

Another case reported by Kouwenhoven[10] was of a thirty-five year old woman who was to undergo cholecystectomy. She was given 8 mg of morphine sulfate, .4 mg of atropine, and 100 mg of phenobarbital for preoperative medication. She was taken to the operating room one and a half hours later where anesthesia was induced with thiopental sodium and succinylcholine. Intubation was attempted but difficulty was encountered. The patient became pulseless and cyanotic and her respiration disappeared. External cardiac massage was instituted, without artificial respiration. After two minutes a strong transthoracic pulse developed, together with spontaneous shallow respiration. Blood pressure returned to 130/80 mm of mercury and pulse beats to 100 beats per minute. Intubation was then accomplished and no further cardiac problems developed. The patient underwent cholecystectomy and had an uneventful recovery. She was discharged five days later without neurological signs or symptoms and subsequent follow-up examinations were normal.

Kouwenhoven reports on four similar cases, including a nine

year old boy undergoing mastoidectomy, an eighty year old woman having a thyroid operation, a twelve year old boy having an excision of a verruca linearis of the scalp with fluothane anesthesia and local infiltration with epinephrine solution, and another case of a forty-five year old man who was brought into the emergency room with myocardial infarction. All attempts at external cardiac massage were successful in these patients and none had residual effect other than the twelve year old boy who had transitory blindness and nystagmus present postoperatively. Within thirty-six hours his neurological findings returned to normal.

Many similar cases reports have appeared in literature recently and there undoubtedly are many attempts at cardiac massage, both successful and unsuccessful, which are not reported. It appears that the external heart massage, if done soon enough after arrest, will provide adequate circulation to maintain the heart and central nervous system and provide an opportunity to bring a defibrillator to the scene if necessary.

It was at first felt that the closed chest method of massage would be most useful in children, whose ribs are known to be flexible. This proved to be incorrect since the chest of the unconscious adult is also very flexible. However, there are some factors about the child's thorax which probably make him a better candidate for external cardiac massage. The thorax is elastic and compressible until puberty, when the sternum becomes rigid. The

musculature of the rib cage develops when the child approaches ten years and this also increases the rigidity of the thorax. The child's heart occupies a larger proportion of the thoracic cavity than does the heart of the adult. Thus, manipulation of the child's chest results in a higher degree of cardiac compression and a greater volume output of blood, which is a distinct advantage. The child's chest is also deeper than the adults, allowing more room for compression and expansion of the heart. The narrowness and small size of the young child's chest makes it easy to grip with the hands and compress.

McKelvie[11] reports of three cases in very small children who experienced cardiac arrest following application of a local anesthetic. He reports good results in these cases. He describes how, in the very small child, the operator may place one hand behind the left side of the chest and the other in front to effect good compression.

Sutherland[19] has discussed the effectiveness of cardiac massage in the stillborn infant. His studies were made over a four year period. The report included eleven cases of stillborn infants on which cardiac massage was carried out. There were six successes in the eleven cases. Six of the cases of stillborn infants followed shoulder arrest. Three of the five failures followed prolapse of the cord. His conclusions were that stillborn infants born after shoulder arrest present the clearest indication for cardiac massage and that when asphyxia is of long duration,

as with prolapse of the cord, the indication for cardiac resuscitation is less clear.

How does the closed chest cardiac massage compare to open chest cardiac massage as far as effectiveness is concerned? What are the advantages of each and their limitations? It appears from the preceding reviews of reported cases and experiments carried out on animals and human patients that the external cardiac massage compares favorably with the open chest heart massage, although it is doubtful if the closed method is as sure a method maintaining circulation as when the operator has his hand directly on the heart. Reviewing experiments on this subject, it appears that the blood pressure and carotid artery flow resulting from direct manual massage of the heart through a thoracotomy is slightly higher than that obtained through the external compression of the chest and heart. However, one must consider the other advantages and disadvantages of both methods.

The certain and most evident advantage of the external cardiac massage is the absence of the thoracotomy incision, which is a major operative procedure, even in the operating room. It is a procedure which is done with hesitancy and not without risk even in elective surgery cases. If nearly the same effects, as far as circulation to vital structures is concerned, can be obtained through a procedure which does not necessitate making a large incision through the thoracic wall, one finds it hard to find good basic reasoning for such a traumatic procedure.

Some complications of the thoracotomy and direct massage include: (1) There is usually a certain amount of injury and trauma to the myocardium where the operator has compressed the heart with his fingers. This has been shown by pathological review of the heart tissue of patients who have not survived the cardiac massage. (2) Infectious complications with empyema or wound abscess. (3) There is also trauma to the lung tissue and surrounding structures and more risk of injuring a major vessel when the heart and other thoracic organs are exposed. (4) It is also felt that pulmonary complications are more frequent following the open approach to heart massage. This is logical to assume for the thoracic pressure relationships are destroyed and the pain produced when the patient attempts to breathe after recovery from arrest hinders breathing, resulting in poor aeration of the lungs.

Another advantage of the external heart massage is the fact that more immediate resuscitation after cardiac arrest can be instituted without additional equipment. Stephenson[17] has stated that the most common cause of failure in cases of resuscitation in cardiac arrest is the delay in instituting cardiac massage. Personnel do not feel adequate enough to perform the thoracotomy. Their delay in initiating any procedure and also the lack of equipment would delay the treatment even further. With external massage the diagnosis can be made and treatment started immediately without undo apprehension or any delay in waiting for specific

equipment.

There are some advantages of the thoracotomy and direct massage. One of these is the fact that the heart and other surrounding organs are in direct view of the operator and he can plainly see what he is accomplishing. He has more definite control of the resuscitative procedure and is more sure of producing an adequate amount of pressure on the heart to establish sufficient circulating volume of blood.

Another advantage of the open chest massage is that it is much easier to accomplish electrical defibrillation by being able to apply the electrodes directly to the myocardium. It also requires a smaller voltage to bring about defibrillation when the electrodes are placed directly on the heart than when the heart is defibrillated through the chest wall. However, Kouwenhoven reports that the closed chest alternating defibrillator that has been developed in their laboratories has proved to an effective and reliable means of arresting ventricular fibrillation.

There is still another advantage of open chest massage. The heart being visible and readily accessible, drugs may be injected with more accuracy directly into certain areas of the myocardium. The aorta being in direct view and accessible makes it possible to clamp it off just beyond the origin of the great vessels and cause shunting of more blood into the cerebral and coronary circulation. This would be considered an advantage of direct manual massage of the heart.

There also is the questionable advantage as to the greater output produced by direct manual massage of the heart. From the studies presented previously there is some question as to the soundness of this presumed advantage over the external massage. Flagg[5] feels that more effective circulation can be produced by the hand directly on the heart than when the external compression of the heart is utilized. Kouwenhoven[10], on the other hand, feels that the external massage provides as satisfactory circulation of blood as does the open chest method without the added risk of thoracotomy. An annotation from the British Medical Journal[22] also states that maintenance of effective circulation is as efficient with external massage as open chest massage.

There is considerable difference between the two methods of resuscitation as far as fatigue of the operator, according to Redding[12] in his experiment with dogs. He found that a single operator could maintain the closed method of cardiac massage for a period of twenty minutes easily, but fatigue necessitated the use of more operators in the direct manual compression of the heart.

The external massage is not without complications. There have been occassional reports of rib fractures, hemothorax, hemopericardium, marrow emboli, and lacerations of the liver. These do not appear to occur frequently but still continue to be hazards, especially in the hands of the inexperienced operator.

The obvious advantage of the external massage -- simplicity --

may actually be a disadvantage if one considers the possible injuries that may result if the layman were to attempt the procedure without adequate training. A person who has fainted may be injured by having the thorax compressed with the external massage. The procedure of external cardiac massage should be carried out by those individuals who are familiar with the anatomy of the body and who have been trained to do this resuscitative measure. The important fact that must be emphasized is that the resuscitative procedure should be carried out only after definite diagnosis of cardiac arrest has been made. It is most desirable to have a physician make this diagnosis; but, can we deny the cardiac arrest patient a life saving procedure because there is no physician immediately available? This is a problem which will become more and more evident as more of the general public become aware of external cardiac massage. We must determine if this procedure would actually save more lives or if there would be more serious injuries due to improper and unnecessary attempts at cardiac massage. If this procedure is to be taught to personnel who are not physicians, they should be instructed also in determining if there is actually an absence of cardiac action. They should learn the sign of cardiac arrest and institute resuscitation measures only when they are certain an arrest has occurred.

Either open or closed massage may be ineffective if the operator is not sensitive to the fact that small changes in the position and manner of pressure may make large differences in the

peripheral pulse obtained. Most failures in resuscitation are due to combinations of various factors. The chief factor is the inexcusable delay between arrest and cardiac massage. Inexperience or unfamiliarity with a method of proper massage is another factor concerned with failure and a good reason the measures should be taught to all physicians particularly. Another important factor is the failure to establish adequate artificial respiration with sufficient air exchange. If this occurs the cardiac massage will be fruitless. Absence of equipment to defibrillate the heart will result in failure at resuscitative measures if the heart is fibrillating. Improper post resuscitative management also enters into the overall survival after the immediate resuscitative measures have succeeded.

When should resuscitative measures be abandoned? As long as massage appears adequate with a good peripheral pulse being maintained and pupils remain constricted, the effort should be continued. Stephenson[17] states there are a number of reported cases in which there appeared to have been severe neurological damage, but who continued to improve after several months. He feels the great majority of the patients who die after the heart has restarted will expire within the first twenty-four hours. In his record of 1,200 cases, he has permanent survival of 28%, but the heart beat was restarted in 56% of all the cases. He feels that by an increase in alertness and awareness of proper resuscitative measures, there is every reason to predict a survival rate

of over 50% in the next few years.

New mechanisims and procedures are now being designed and tested. Most of the mechanisms being tested for cardiac resuscitation consist of electronic and electrical devices to be used as defibrillators or as pacemakers in the patient with complete heart block. Attempts are also being made to design a mechanism which can be used for automatic external heart massage and at the same time act as a respirator and ventilate the patient.

These mechanisms will probably not be available for those patients who have a cardiac arrest outside the hospital. This group includes one-fourth to one-third of the total arrest patients.

It appears that closed chest cardiac massage should be the preferred method of approach to cardiac arrest outside the operating room. Even in the operating room where cardiac arrest occurs, it is the author's opinion that the preliminary use of external cardiac massage is justified and effectiveness should be evaluated by the status of the patient before the thoracotomy incision is made. Surgeons have been slow to accept the value of massage applied externally, being conditioned to the urgency of opening the thorax and massaging the heart within minutes of onset of circulatory arrest.

External massage is the method usually used at John Hopkins Hospital in the treatment of cardiac arrest and only rarely is the chest opened. Their studies have been documented many times by direct measurement of arterial pressures and by survival of

patients.[1]

External cardiac resuscitation may be life-saving in the patient who has arrest outside the hospital. Its value lies in its simplicity and the fact that there is no disfigurement of the patient. There would be less hesitation in starting massage and those most important first few moments need not be spent in deliberation.

No patient should be denied a chance to survive because a physician feels inadequate in thinking that the only method for treatment is thoracotomy and heart massage. The external heart massage has proven to be equally as effective and is simple in being administered. The human heart has been made to beat again after it has stopped. The heart may be too good to die and may need only a second chance to beat. The apparently fatal attack is not necessarily the end of life.

BIBLIOGRAPHY

1. Bahnson, H. T., Closed Chest Cardiac Resuscitation, Surgery GYN and OB. 112:622-623 (May) 1961.

2. Beck, C. S. and others, Death After a Clean Bill of Health, J.A.M.A. 174:133-134 (Sept. 10) 1960.

3. Briggs, B. D. and others, Cardiac Arrest, J.A.M.A. 160:1439-1447 (April 28) 1956.

4. Erickson, R. I. and others, An Effective and Simplified Method for Cardiac Resuscitation, Journal of Oral Surgery 19: 322-325 (July) 1961.

5. Flagg, P. J., Present Day Cardiac Resuscitation, Am J. of Cardiology 8: 297-299 (Aug.) 1961.

6. Greenwich, V. and others, Aortic Pressure During Closed Chest Cardiac Massage, Circulation Vol. XXIII: 593-595 (April) 1961.

7. Halswade, G. R., The Management of Cardiac Arrest, Surgery Clinics of North American 41: 315-321 (April) 1961.

8. Jude, J. R., Cardiac Resuscitation, Am. Heart J. 62: 286-287 (Aug.) 1961.

9. Jude, J.R. and others, Cardiac Arrest, J.A.M.A. 174: 1063-1070 (Dec. 16) 1961.

10. Kouwenhoven, W. B. and others, Closed Chest Cardiac Massage, J.A.M.A. 173: 1064-1067 (July 9) 1960.

11. McKelvie, W. B. and McKelvie, P., External Cardiac Massage in Young Children, British J. of Anesthesiology 33: 371-372 (July) 1961.

12. Redding, J. S. and others, A Comparison of Open Chest and Closed Chest Cardiac Massage in Dogs, Anesthesiology 22: 280-285 (March - April) 1961.

13. Safar, P. and others, Ventilation and Circulation with Closed Chest Cardiac Massage in Man, J.A.M.A. 176: 574-576 (May 20) 1961.

14. Safar, P., Closed Chest Cardiac Massage, Anesthesia and Analgesia 40: 609-613 (Nov.- Dec.) 1961.

15. Stahlgren, L. H. and Angelchik, J., Cardiac Arrest, J.A.M.A. 174: 226-233 (Sept. 17) 1960.

16. Staryl, T. E., The Role of Electrical Pacemaker in the Treatment of Unexpected Cardiac Arrest, Surgery of GYN and OB 112: 624 (May) 1961.

17. Stephenson, H. E.Jr. and others, Some Common Denominators in 1200 Cases of Cardiac Arrest, Annals of Surgery 137: 731-744 (May) 1953.

18. Stone, H. H., Cardiac Massage: A Report of 148 Cases, The American Surgeon 27: 495-501 (July) 1961.

19. Sutherland, J. M. and Epple, H. H., Cardiac Massage of Stillborn Infants, Obstetrics and Gynecology 18: 182-186 (Aug.) 1961.

20. Williams, G. R. Jr., and Lynch, M. F., Treatment of Cardiac Arrest by Closed Chest Massage, The Journal of Okla. State Med. Assoc. 54: 160-162 (April) 1961.

21. Article on Cardiac Arrest and Resuscitation, The Lancet Vol. II, No, 7164: 1333-1334 (Dec.17) 1960.

22. Article on External Cardiac Massage, Annotation from British Medical Journal 5249: 440 (Aug.12) 1961.

23. Clinical Anesthesia Conference -- Prepared by the Anesthesia Study Committee of the New York State Society of Anesthesiologists, Closed Chest Cardiac Massage, New York State J. of Med. 61: 2304-2306 (July 1) 1961.