SUDDEN CARDIAC COLLAPSE

Cardiac Arrest and Its Treatment

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SUDDEN cardiac collapse is a complication that may occur during any phase of anesthesia. Its appearance is sudden, dramatic, frustrating and frequently fatal. It is of vital concern to all anesthesiologists.\textsuperscript{1,4} While numerous reports describe its occurrence during induction and maintenance of anesthesia,\textsuperscript{5,6,15} few refer to the period of emergence from anesthesia, during transport of the patient to his room or during his stay in the postanesthesia recovery room or intensive care unit. An attempt is made in this presentation to review the literature concerning sudden cardiac collapse in the postanesthetic period.

There is considerable misunderstanding\textsuperscript{1,5,16} in the use of the term "cardiac arrest." The term has been employed to refer to a limitless variety of catastrophes occurring in the operating pavilion. The terms "cardiac arrest" and "cardiac standstill" have been used interchangeably. Many regard the term "cardiac arrest" as vague, inadequate and inaccurate. There is a need for clarification and a definition of terms. Recently, "sudden cardiac collapse" was proposed to refer to the precipitous failure of effective myocardial contraction.\textsuperscript{5} This may result from 1 cardiac asystole or 2 from ventricular fibrillation. The pulse disappears suddenly and the blood pressure becomes unobtainable in both. The myocardium is noncontractile in asystole and irritable with incoordinate and ineffective contraction in ventricular fibrillation.\textsuperscript{5} Cardiac collapse may also result from a rapid sustained tachycardia with subsequent ineffective systole as seen in irreversible shock.

INCIDENCE AND ETIOLOGY

It is not possible at this time to establish the incidence of sudden cardiac collapse during the postanesthetic period. There is not only an insufficiency but also a lack of uniformity, accuracy and classification of data. It is generally accepted, however, that the incidence of postanesthetic sudden cardiac collapse is increasing due to a multiplicity of factors.\textsuperscript{5,6,16,17} Recently a Cardiac Arrest Registry has been established with the cooperation of physicians from all parts of the world.\textsuperscript{1} It is hoped it will determine the incidence of sudden cardiac collapse in the postanesthetic period.

The etiologic mechanisms of sudden cardiac collapse in the postanesthetic period are multiple and complex.\textsuperscript{1,5} They concern the anesthetic management, the operation performed and particularly the physiological and pathological derangements of the patient. Certain factors are predisposing and others precipitating in nature.\textsuperscript{1,2,6,13} Of the former group, the most significant appear to be hypotension, hypovolemia, anemia and asphyxia. The precipitating factors of the latter group consist of (1) nervous stimuli, \textit{i.e.}, somatic and visceral stimuli, (2) chemical stimuli resulting from hypoxia, hypercarbia, asphyxia, acidosis, or hyperpotassemia, and (3) physical stimuli, \textit{i.e.}, cardiac tamponade, anomalies of thoracic organs, position of the patient, etc. Occasionally a predisposing influence may produce sudden cardiac collapse without any precipitating stimulus.\textsuperscript{6} Some believe the nervous factor plays the dominant and often the sole role in producing sudden cardiac collapse.\textsuperscript{5,11,12} Porter and French\textsuperscript{11} emphasize that different somatic and visceral stimuli will produce cardiac arrest during light anesthesia and that the basic mechanisms involved in many cases are (1) stimuli applied during, and (2) a stage of distorted reticular formation. Such mechanisms are central in action.

Others strongly feel that chemical stimuli are the significant factors involved in precipitating sudden cardiac collapse following anesthesia as well as before and during its administration.\textsuperscript{44-53} Johnson and Kirby\textsuperscript{47} state hypoxia is the most important factor. Redd \textit{et al.}\textsuperscript{48} state that in guinea pigs, "lack of oxy-
gen is more important than lack of substrate in producing irreversible cardiac arrest," and that "cardiac arrest achieved by lack of oxygen, substrate or both, is irreversible, and results from cellular exhaustion." Hypercarbia and asphyxia, as well as hypoxia, may occur in the immediate postanesthetic state, particularly after injudicious or excessive use of relaxants or improper position of the patient during his transport to the postanesthesia recovery room.

LeVeen and collaborators emphasized potassium intoxication following massive blood transfusion as a significant chemical factor in precipitating sudden cardiac collapse. Fifty of 157 cases of sudden cardiac collapse were due to hyperkalemia following banked blood transfusions. Many of this series occurred in the postanesthetic period.

Physical factors may also precipitate sudden cardiac collapse. Although admittedly their influence in the postanesthesia period is secondary to nervous and chemical stimuli, a physical factor such as cardiac tamponade can occur in the postanesthetic period and may precipitate sudden cardiac collapse.

**Postanesthetic Complications**

Postanesthetic complications involve various systems or organs of the body and may occur while the patient is being transported to his room or while he is in the postanesthesia recovery unit. Adriani and others state that some of these are conducive to sudden cardiac collapse. Eckenhoff emphasized some preoperative warnings of potential operating room deaths. In an analysis of 200 operative and postoperative fatalities, Weiss stressed the need for adequate supervision in postanesthesia recovery rooms and intensive care units. A case of cardiac arrest occurring 20 hours postoperatively when tracheal suction was attempted, has been reported.48 In 83,442 anesthetic administrations, 7 of 20 cases of sudden cardiac collapse occurred after completion of the surgical procedure; 3 during emergence from anesthesia in the operating room, 1 upon arrival in the recovery room, and 3 patients 1–8 hours after operation. The etiological factors in the 7 cases were chemical in nature: respiratory depression, chronic and acute hypotension, hypovolemia and peripheral vascular collapse. The nervous factor considered so important in producing sudden cardiac collapse during induction and endotracheal extubation was fatal in 8 cases during nasotracheal aspiration. Seven of these were in postoperative patients. Vagal stimulation, in the presence of asphyxia was thought to be the precipitating factor. It is thus becoming increasingly apparent that sudden cardiac collapse can and does occur in the postanesthetic period.

Some of the more significant postanesthetic complications causing sudden cardiac collapse are briefly discussed.

**Respiratory System.** (1) Hypoxia, (2) hypercarbia, and (3) asphyxia.

Hypoxia is the most important single factor in sudden cardiac collapse. However, hypercarbia and asphyxia also are effective in seriously disturbing myocardial function. They may occur in acute or chronic form, in the improperly supervised postanesthesia patient. Some of the causes of asphyxia in such patients are: (1) apnea and/or hypventilation due to deep anesthesia, effects of curare derivatives, injudicious use of narcotics, prone position, undetected pneumothorax or massive atelectasis, or respiratory obstruction; (2) uncontrolled coughing spasm; (3) reflex apnea from naso-endotracheal aspiration; (4) convulsions, particularly in hyperthermic children.

**Cardiovascular System.** (1) Hypotension may also cause sudden cardiac collapse, particularly in the aged and poor-risk patient. Its occurrence, in transit of the patient, or in the inadequately supervised postanesthesia room, may lead to fatal ventricular fibrillation. It may be due to hypovolemia from inadequate fluid replacement during operation, unexpected hemorrhage after surgery, afibrinogenemia, position of the patient on the recovery room stretcher, deep general anesthesia, ruptured viscus, cardiac arrhythmias, cardiac decompensation, embolism, hyperventilation and peripheral vascular collapse.

(2) Cardiac arrhythmias such as paroxysmal tachycardia, nodal rhythms, or heart block when appearing and detected after surgery, should be treated promptly. Such arrhythmias may be due to a variety of causes discussed elsewhere in this Symposium.

**Gastrointestinal Tract.** Postoperative vomiting and aspiration may cause severe asphyxia.
and thus a cardiac asystole or fibrillation. Acute gastric distention, produced by positive pressure respiration, particularly in the infant, poor-risk or aged may similarly produce sudden cardiac collapse.

Miscellaneous. (1) Deep hypothermia is conducive to sudden cardiac collapse. Rewarming hypothermic patients should start early enough so that temperatures of at least 36°C should be reached before transport to recovery units.

Diagnosis and Prognosis

The diagnosis of sudden cardiac collapse must be prompt and unequivocal if resuscitation is to be successful. Delays in instituting effective treatment gravely affects prognosis.1-2 Cerebral anoxia of four minutes duration in a well-oxygenated patient at the time of sudden cardiac collapse results in irreversible damage to the brain.61 In an hypoxic patient, cerebral anoxia of one minute or less may produce similar results. Vital organs, such as the heart and kidneys sustain significant though reversible damage in direct proportion to the period of indecisive treatment.66,67

Various procedures facilitating the diagnosis of sudden cardiac collapse have been recommended. Briefly, these are: (1) development of a "sense of awareness" of the possible occurrence of sudden cardiac collapse and a thorough appreciation of its etiology and symptomatology;1-8,14,15,16,26,30,47,64,65 (2) frequent recording of pulse rate, blood pressure, respiratory1-6 rate; and (3) the prophylactic use of monitors.11,62-76 The alert physician should sense impending complications and not be caught "off guard" or unprepared. He and all other attendants should know and fully appreciate certain warning signs which include:

(1) Sudden disappearance of radial, femoral, or carotid pulsations.
(2) Unobtainable blood pressure.
(3) Absence of wound bleeding.
(4) Pallor of skin or cyanosis.
(5) Cessation of cardiac impulse.
(6) Electrocardiogram showing asystole or ventricular fibrillation.
(7) Apnea or sudden gasping respiration.
(8) Dilated, fixed pupils in later stages.

Although the recording of the electrocardiogram and the electroencephalogram has been recommended during some operations,11,77,78 and also during all surgical procedures,29 the use of monitors on postoperative cases has received but slight attention. In fact, monitoring of vital signs of patients being transported immediately after surgery has been virtually ignored. The pulse rate, blood pressure and minute volume of respiration, checked with a ventilometer, should be evaluated and proper corrections made before attempts to transport the patient. In transit, the anesthesiologist's fingers should record the carotid pulse continuously and the blood pressure cuff should be ready for instant use. If there is evidence of hypoventilation before transit, then either the patient should be kept in the operating room and be given assisted respiration until there is a normal minute volume or be transferred with assisted respiration being administered by an endotracheal tube or face mask in place and connected to a portable source of oxygen. Some have recommended the use of portable monitors76,79,80,81 with visual or auditory recording. However, it has been reported that cardiac electrical activity may appear normal with an ineffective myocardial contraction and unobtainable blood pressure. Further, if attention is not focused on the sound in auditory monitor recording, one may fail to hear it stop. Monitors are thus not foolproof and sole reliance upon them for diagnosis is foolhardy, time-consuming and unwarranted.

Beck has emphasized certain "Don't's" in the diagnosis of sudden cardiac collapse. If pulse or blood pressure is unobtainable,75 he recommends:

(1) Do not waste precious moments.
(2) Do not keep on checking blood pressure and listening for heart sounds.
(3) Do not take an electrocardiogram.
(4) Do not strike the precordium with your fist.
(5) Do not inject adrenaline into the heart.
(6) Do not produce painful stimuli.
(7) Do not give an intra-arterial blood transfusion.

Prognosis in patients with sudden cardiac collapse is always guarded and to be considered poor unless proven otherwise. However, it is dependent chiefly upon: (1) rapid
restoration of an effective oxygen system, and (2) rapid resoration of the heart beat and effective circulation.

Other favorable prognostic signs are the return of consciousness, normal size of pupils, a normal electrocardiogram and electroencephalogram and the absence of twitchings, convulsions and hyperthermia.

**Prophylaxis**

In no other phase of medicine is prevention more important than anesthesiology. The more significant prophylactic procedures are:

(1) **Thorough preoperative evaluation and preparation of the patient.** Some preoperative warnings of potential operating room deaths and the necessity for their detection and prompt correction have been stressed. The patient's physiological status should be determined and recorded. After checking the history, physical examination, roentgenogram and laboratory data of the patient, all derangements such as anemia, hypovolemia, electrolyte imbalance, significant cardiac arrhythmia, hypotension, cyanosis, dyspnea, hypoventilation, respiratory obstruction, undue apprehension, pain, retching or vomiting should be corrected before anesthesia. The preoperative medication consisting of an analgesic, hypnotic, anesthetizing mixture and/or a belladonna derivative should be ordered depending upon the needs as well as the age, and weight of the patient and type of anesthesia to be administered. Then, on the basis of the physiological status and the surgery contemplated, the optimum selection of anesthetic agent and technique should be made.

(2) A smooth induction of anesthesia, that is free of excitement, sudden hypotension, struggling, respiratory obstruction, cyanosis, nausea or vomiting, is imperative for safe anesthesia. Complications during induction may lead insidiously to sudden cardiac collapse in the recovery period.

(3) **Maintenance of anesthesia** should be at optimum depth to avoid overdose, hypoxia, hypercarbia, or the appearance of adverse effects. Further, correction of cardiac arrhythmia or of hypotension due to blood loss, anesthetic agent, reflex activity or position of the patient should be promptly instituted.

(4) **Emergence from anesthesia** should be smooth and uncomplicated. Excitement, respiratory obstruction, hypotension, and vomiting must be avoided or taken care of immediately.

(5) **Lifting and positioning** the patient from the operating room table and the recovery room stretcher must be done slowly, carefully and with adequate help. Precipitous hypotension or severe respiratory obstruction among other complications may occur from sudden jerky, lifting of patients. The pulse rate, blood pressure and minute volume of respiration and the depth of anesthesia should be checked before moving patients and adequate corrections made of any significant deviations. Such vital signs should be checked again after the patient has been placed on the stretcher (preferably) in the supine position with the head in slight extension. The blood pressure cuff and sphygmomanometer should remain on the patient during transport. No unconscious patient should be transported in the prone position even with an endotracheal tube in place. The dangers of disturbing circulation and/or impeding respiration are too great. Patients should be transported on a stretcher to the recovery room or intensive care unit by the anesthesiologist (and not an orderly), who should monitor the pulse continuously and keep a close watch on the rate and amplitude of respiration.

(6) **Upon arrival in the postanesthesia recovery room,** the pulse rate, blood pressure, respiratory rate and amplitude should be immediately recorded. The color of the skin and the presence of gag, laryngeal and swallowing reflexes should also be noted. Before departing, the staff anesthesiologist or nurse in charge of the recovery unit should be briefed concerning the course of and complications during the administration of anesthesia.

(7) **Certain monitors** employed during the course of anesthesia may be used in the recovery room. However, it must be emphasized that they are only adjuncts to clinical symptomatology and are not fool-proof. The electrocardiogram, ventilometer, cardiac meter and pacemaker are most commonly recommended. Bellville, Gain and Elton in
particular stress the prophylactic usefulness of the electroencephalograph. Both the electrocardiogram and the electroencephalograph can be of important prognostic use in the recovery room during treatment of sudden cardiac collapse. Parsons 36 recommends electrocardiographic monitoring of every patient in every operating room and claims his hospital to be the first to adopt this prophylactic procedure. Recently, bedside measurement of alveolar carbon dioxide tension by $2N$ NaOH absorption in a lucite chamber has been made possible. 36

(8) The postanesthesia recovery room or intensive care unit should have adequate staff—consisting of anesthesiologists, graduate nurses and orderlies—equipment, and medications. Various recommendations concerning equipment and drugs have been made. The following has been found satisfactory for a postanesthesia recovery room in a 600 bed general hospital. 4

**Basic Supplies**

**Equipment:**
- Intravenous sets
- Cut-down tray
- Thoracotomy tray
- Endotracheal tubes with connectors
- Nasal and pharyngeal airways
- Tracheotomy set
- Tracheotomy tubes (silver and rubber-ruled)
- Hypothermia machine
- Ambu resuscitator
- Puritan humidifier
- Suction with catheters
- IPPB machine

**Monitors:**
- Stethoscope and blood pressure apparatus
- ECG machine
- EEG machine
- External defibrillators
- Internal defibrillators
- Pace-maker

**Drugs:**
- Epinephrine (1:1,000)
- Nephentermine (2 ml. amp. = 30 mg.)
- Neosynephrine (1 ml. amp. = 10 mg.)
- 1-Norepinephrine (4 ml. = 8 mg.)
- Isoproprylevarteronal
- Cedilanid (0.8 mg. amp.)
- Ouabain (2 ml. = 0.5 mg.)
- Ca Gluconate 10 per cent
- Ca Cl2 10 per cent
- NaHCO3
- Molar sodium lactate
- Atropine sulfate (1 ml. = 0.4 mg.)
- KCl 10 per cent
- Quindine
- Procaine amide (500 mg. amp.)
- Procaine (1 per cent)
- Pentothal (2.5 per cent)
- Sucinylcholine (1 ml. = 20 mg.)
- Aminophylline (20 ml. = 500 mg.)
- Benadryl (1 ml. = 10 mg.)

(9) Cooper and others recommend digitalization as a prophylactic procedure when sudden cardiac collapse occurs to aid the myocardium and to prevent hyperpotassemia.

(10) The inauguration of training programs for the prophylaxis as well as the therapy of sudden cardiac arrest has been highly recommended. Others have formed in their hospitals a “cardiac arrest team,” a “sudden cardiac collapse committee” or presented lectures periodically to the professional staff. All of these efforts have served to develop a keen sense of awareness for sudden cardiac collapse and therefore constitute commendable prophylaxis.

**Therapeutics**

Successful treatment of sudden cardiac collapse is based upon (1) a prompt diagnosis, (2) immediate institution of a well-planned method of treatment, and (3) teamwork. Loss of time means loss of life. As Davis has stated, failure of adequate and prompt treatment will result in a sudden death, death after 1–2 days, or survival of the patient indefinitely “without any intelligence.”

Many therapeutic regimes for sudden cardiac collapse have been recommended. Beck believes the resuscitation procedure should consist of two parts: part 1—the emergency restoration of respiration, and part 2—the res-
sudden cardiac collapse is to pound the chest forcibly. He states that no harm can take place. The basis of the procedure is to apply a mechanical stimulus to the myocardium by the blow on the sternum. The same rationale is the basis for "needling" the heart. Bailey 93 found this procedure ineffective. Pounding and needling are ineffective in ventricular fibrillation.

Zoll and his collaborators 23, 24 have reported success with the use of an external defibrillating current and of a stimulating current in treating ventricular fibrillation or cardiac asystole respectively. However, Hosler et al. 200 showed that in dogs while external defibrillation resulted in asystole of the heart, the return of blood pressure did not follow unless the heart was massaged. He concluded that when a trained team and equipment are available, a few seconds might be spent in external countershocking. If blood pressure fails to return, then thoracotomy and manual cardiac massage should be performed.

The use of thoracotomy with manual cardiac compression has been practiced enthusiastically and occasionally with avowed blindness. It is contended by Beck et al. 79 that thoracotomy, with manual cardiac compression in the operating pavilion or postanesthesia recovery room, not only facilitates diagnosis but also improves blood flow to the myocardium before the application of defibrillating or stimulating currents or drugs for resuscitation. While this is true, the question has been asked all too often, wasn’t the thoracotomy performed unwisely in some cases. What is even more disconcerting are some of the reports of thoracotomies done on patients in medical wards, emergency rooms and on the street. Little need be said about the sepsis, blood loss, trauma and legal action invited by such heriocics. In a timely paper, Carter 71 has questioned whether surgical intervention was always warranted. He reports (1) positive indications for thoracotomy with good prognosis, (2) positive indications for thoracotomy with poor prognosis, and (3) positive contra indications for thoracotomy. Southworth 82 lists four questions for internists to guide them in their decision to attempt thoracotomy with manual cardiac massage. The questions are: (1) Has the patient the fundamental health to justify restoration of life? (2) Can you guarantee that there is still time to institute massage (less than four minutes have elapsed)? (3) Have you the training, the equipment and the assistance necessary to undertake the procedure (both cardiac massage and assisted respiration) and to carry it through to a successful conclusion? (4) Is the arrest iatrogenic? Many clinicians are becoming more conservative and state that after sudden cardiac collapse is suspected or diagnosed, the left anterior chest wall should be pounded intermittently for 10-15 seconds before a thoracotomy is performed. McMullan 101 believes that thoracotomy is the best procedure for the treatment of ventricular fibrillation. On the other hand, Stoffregen 22 states that it should be the last procedure to be carried out, after the use of positive-negative respiration with oxygen, Trendelenburg position, and norepinephrine infusion for support of the circulation for the first two minutes. The complications of a thoracotomy with manual cardiac compression are many, some of which are sepsis, cardiac tamponade, rupture of the myocardium, and pneumothorax. Because of these major disadvantages, some have hesitated to institute this form of therapy.

Recently, Kouwenhoven and his colleagues 96 have developed a promising and effective method for the resuscitation of sudden cardiac collapse. This technique requires minimal skill and no equipment. They state "anyone, anywhere can now institute cardiac resuscitative procedures. All that is needed are two hands." After an initial experimental study on
dogs, they report a 70 per cent permanent survival in 20 patients resuscitated with this method. The method consists of applying pressure on the sternum, rhythmically at 60 times per minute, with arms of operator extended, and with the heel of the left hand on the lower third of sternum and the right hand over the left. Such pressure compresses the heart between the sternum and the vertebral bodies. Movement of the sternum toward the vertebrae should be about 3–4 cm. Closed chest massage was applied from 1–65 minutes in these cases with systolic pressures produced during massage ranging from 60–100 mm. of mercury. Three patients with ventricular fibrillation were defibrillated by a closed chest route after initial use of the sternal pressure. These as well as 11 of 14 with cardiac asystole survived. Although Kouwenhoven and his associates claimed that their method provided some ventilation of the lungs, Safar and Holley have challenged this. In 12 patients with cardiac asystole or ventricular fibrillation proven electrocardiographically and 18 others where blood pressure was unobtainable, Safar confirmed the effectiveness of closed chest cardiac massage in restoring effective circulation, but in 30 healthy curarized adults, sternal compression alone, failed to provide adequate ventilation of the lungs. Inasmuch as some patients recover after artificial respiration alone, and rib and costal fractures have occurred with closed-chest massage, Safar recommends positive-pressure inflation of the lungs with air or oxygen first before sternal compression. If a pulse is not palpable in a large artery after 3 to 5 lung inflations, then sternal compression should be started by a second resuscitator, at 1 second intervals, 4 times after each lung inflation. Lung inflation should continue at a rate of 12 per minute. Someone should monitor the pulse. The authors state that one operator cannot effectively ventilate the patient and provide sternal compression. After starting closed-chest cardiac massage, asystole or ventricular fibrillation should be diagnosed electrocardiographically and attempts made to defibrillate the fibrillating heart or to initiate a beat in the asystolic myocardium by the proper current applied externally.

It would appear that the combined technique of adequate ventilation and closed chest cardiac massage is the most adequate, simplest and readily adaptable method yet devised to treat sudden cardiac collapse. However, several pertinent questions have been raised recently. Does closed-chest cardiac massage result in less effective circulation when cardiac arrest occurs under conditions that permit thoracotomy? Would vigorous compression of the sternum against the vertebral column result in trauma to the heart more severe than thoracotomy? Accordingly, open chest and closed-chest cardiac massage has been compared in a series of 20 dogs with artificially induced ventricular fibrillation. The artificial circulation produced by closed-chest cardiac massage was comparable to that produced by open-chest cardiac massage. When the hearts were defibrillated, circulation was restored effectively after application of either method. Trauma with closed-chest cardiac massage included mediastinal hemorrhage, fractured ribs and cartilages and laceration of the liver, when maximal force was applied to the closed chest. Minimal trauma was noted when moderate force was applied to the chest. Closed chest cardiac massage was considerably less fatiguing to the operator. Redding and Cozine further point out, in unpublished observations, that the circulation, resulting from closed-chest massage, combined with ventilation by intermittent positive pressure breathing appeared adequate to reoxygenate the myocardium in asphyxiated dogs.

These studies confirming those of Safar and Kouwenhoven should deter surgeons and other physicians from instant thoracotomy at the drop of a heart beat. A less dramatic but effective procedure can now be carried out in the operating pavilion and post-anesthesia recovery room, and as the authors state "anywhere, anytime." The final evaluation of this method, however, depends upon further trial and confirmation.

Other recommendations have been made during the past few years to aid in the treatment of sudden cardiac collapse. These include intra-arterial transfusion with oxygenated blood, drug therapy, and the use of hypothermia. The advantages of intra-arterial transfusion are minimized by the loss of time required to carry out the technique.
and also by the trauma to the artery. Unless the chest is open and the aorta already accessible for transfusion, the maximum benefit of this procedure is not achieved.

Drug therapy in the treatment of the sudden cardiac collapse patient, has been stressed by Rowe and others. They believe that such therapy, not only aids in the restoration and maintenance of effective cardiac action but also in combating cerebral edema, pulmonary congestion and renal insufficiency. Digitalization of patients with sudden cardiac collapse is advised because of potential or actual cardiac decompensation and also to counteract hypotension. LeVeen states that "digitalis, by preventing the passage of potassium into the cell, will enable the myocardium to function properly in the presence of toxic levels of this ion," particularly in patients receiving massive transfusion of old blood.

Cooper et al. reports that mephentermine is a valuable cardiotonic drug and should be given to patients recovering from sudden cardiac arrest and from hypotension. It increases cardiac output, blood pressure, and stroke work and decreases peripheral resistance. Recently, Young and Kaupp have employed urea to minimize cerebral edema resulting from sudden cardiac arrest. Young administered urea after a 7-8½ minute cardiac-arrest period in dogs, and observed that the cerebral edema and the increased intracranial pressure were definitely decreased. However, Kaupp and his collaborators believe that cerebral edema is an over-emphasized factor in the post-cardiac arrest syndrome. They state that the principal determinant to recovery is hypoxic injury to the neurons not beneficially influenced by the reduction in brain volume. Further, despite the decrease in brain swelling in dogs and monkeys, hypotonic urea did not materially decrease the mortality or morbidity in the treatment of sudden cardiac collapse. Nevertheless, Benson and Murphy recently reported that urea induced serum hyperosmolality lowers cerebrospinal fluid pressure without adverse responses and therefore should be employed early in the treatment of cerebral hypoxia. It appears that the therapeutic value of urea in the treatment of post-cardiac arrest syndrome is not yet established.

Hypothermia is another adjunct in the treatment of patients with sudden cardiac collapse. Cessation of cerebral blood flow for more than 4 minutes in man results in cortical cellular destruction. Such hypoxic injury is manifested clinically as unconsciousness, hypotonicity, convulsion, coma or death. The duration of hypoxia generally determines the prognosis. Such prognosis is favored by the early institution of hypothermia. Rosomoff and Holaday estimated that at 28-30°C the oxygen needs of the brain are reduced 50 per cent. Wolfe reported a 30 per cent survival without cerebral damage and an additional 40 per cent prolongation of life in dogs after a 5-minute period of ventricular fibrillation followed by hypothermia to 31°C for 24 hours. All uncooled control dogs expired.

Feldman, Ruben and Surks report complete neuropsychological recovery in a 2 year old girl with hypotonicity, convulsions, and coma in the immediate post-arrest period upon the application of hypothermia with a water-cooled mattress. Lore et al. claim to have prevented residual brain damage with the use of hypothermia following cardiac arrest in a 12 day old infant, one of the youngest survivors of cardiac arrest on record. Benson et al. report that in 19 patients resuscitated after sudden cardiac collapse with resulting neurological damage, 50 per cent survived who were treated with hypothermia and only 14 per cent survived who were not cooled. Hypothermia was continued for 3 hours to 8 days at 30-32°C. They conclude that the addition of hypothermia is clinically significant and its use is justified in all cases of cardiac arrest with apparent neurological injury. There appears little doubt that hypothermia is advisable as an adjunct in the treatment of sudden cardiac collapse to prevent permanent neurological sequelae.

The treatment of sudden cardiac collapse is dependent upon continuing processes of prophylaxis, research, and education. Only recently, Jude reported 96 per cent success in resuscitating patients with sudden cardiac collapse in the operating room or in the postanesthesia recovery room with the combined method of ventilation and restoration of circulation by closed-chest cardiac massage. On
the basis of the successes reported in the recent literature, the following regime is recommended at this time, when sudden cardiac collapse is suspected or diagnosed in the operating room, postanesthesia recovery room, or intensive care unit. It is assumed that at least two of the operating team or recovery room personnel are immediately available. Both should act simultaneously.

**Maintenance or Restoration of Ventilation (First Operator)**

(a) The first operator should maintain or restore adequate ventilation preferably with oxygen by intermittent positive pressure through an endotracheal tube at a rate of 14–16 times per minute. 

(b) If a tube is not available, then oxygenation with intermittent positive pressure with a mask, should be administered at the same rate. 

(c) If no equipment is available, then mouth-to-mouth or mouth-to-nose breathing at 14–16 times per minute should be carried out. 

(d) The carotid, femoral, or radial pulse should be checked continuously.

**Restoration of Circulation (Second Operator)**

(a) The second operator should simultaneously perform closed-chest cardiac massage at 60–80 times per minute for at least 2–4 minutes to correct myocardial and cerebral hypoxia or until a carotid or radial pulse is detected. The combined method of ventilation and closed-chest cardiac massage has been used for an hour with successful results. 

(b) Obtain an electrocardiogram within 4 minutes if possible to determine if ventricular fibrillation or cardiac asystole is present. 

(1) If ventricular fibrillation is present, the operator should apply an external defibrillating current (440 volts for 0.25 seconds) for 1 or 2 countershocks. Continue with external cardiac massage. Since there is a possibility of a spark developing with the use of the external defibrillator, wet towels over the electrodes or other suitable precautionary methods should be used when flammable anesthetic agents have been employed. If external defibrilla-

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muscels and pleura. Care must be taken to avoid puncturing the lungs. The intercostal space should then be spread manually and the heart promptly palpated to determine asystole or ventricular fibrillation. It should be compressed by the fingers and the palm of the hand rhythmically at 72 times per minute. This technique should be known by all anesthesiologists, for as Stephenson states "the physician most readily available is the person who should make the incision." The anesthesiologist may be the only physician in the post-anesthesia recovery room or intensive care unit.

Supportive and Symptomatic Treatment

(a) Calcium gluconate (10 per cent solution) should be injected into the left ventricle in a dose of 1–2 Gm. (4–8 ml.) after myocardial is oxygenated and externally defibrillated or when cardiac asystole is detected by the electrocardiogram to facilitate and increase the strength of myocardial contraction.

(b) Epinephrine—Injection of epinephrine (4 ml. of 1:10,000) might be injected into the left ventricle if CaCl₂ or other cardiotonic drugs have failed to increase the irritability and strength of myocardial contraction.

(c) Mephenytoine intravenous injections given intermittently (1 ml. = 15 mg.) are highly recommended to increase the strength of myocardial contraction and to raise blood pressure.⁴⁴

(d) Digitalization, preferably with cedilanid in two doses of 0.8 mg. each, given 2 hours apart is desirable for impending myocardial decompensation. Digitalization has also been recommended to prevent hyperpotassiumia, particularly after massive transfusions with old blood.⁴⁵ ⁴⁶

(e) Atropine sulphate in doses of 0.5 mg. should be given in the left ventricle in cases of cardiac asystole.

(f) Phenylephrine, isopropylterenol and lactopinephrine as an infusion, given intermittently, have also been recommended for elevating and maintaining blood pressure.⁴⁷

(g) Sodium bicarbonate solution in a dose of 44.5 mEq. should be given intravenously in an adult if cardiac collapse persists beyond 5 minutes. It may be repeated every 10 minutes for 6 doses. Its main effect is to reverse metabolic acidosis readily detected by proper laboratory procedures.

(h) The early use of hypertonic urea (40 Gm. of urea in 250 ml. of 5 per cent dextrose and water) given intravenously will lower cerebrospinal fluid pressure and decrease cerebral edema.⁴⁹

(i) The use of hypothermia should be considered when the pulse rate and blood pressure have been restored close to normal range and there is continued unconsciousness, respiratory paralysis, muscle spasticity, twitchings, convulsions, or complete areflexia. It should then be promptly instituted and maintained at 30–32 C. for at least 48 hours. Rewarming should then be instituted. If muscle spasticity, twitchings, or unconsciousness return, hypothermia should be resumed for another 24–48 hours.

(j) Other procedures such as correction of hypovolemia, use of Trendelenburg position 15 degrees, tracheal suction, urinary bladder catheterization should be carried out.

(k) Intermittent electrocardiograms should be recorded to determine possible cardiac irregularities or damage.

(1) Frequent electroencephalographic tracings will be of aid in prognosis. Normal tracings are favorable signs; continued flattened tracings indicate a grave prognosis.

Initiation of resuscitative efforts should depend upon the patient’s capability of being resuscitated with at least a possibility of a normal existence. Termination of efforts at cardiac resuscitation depend essentially upon the patient’s condition. The best criteria are (1) failure to maintain blood pressure without continued use of pressor agents, (2) progressive electrocardiographic evidence of myocardial deterioration, (3) electroencephalographic evidence of progressive brain damage, and (4) continued unconsciousness and areflexia.

REFERENCES


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