CARDIAC ARREST

DONALD B. EFFLER, M.D. and EARL C. SIFERS, M.D.*
Department of Thoracic Surgery

UNEXPECTED cessation of the heart beat is the most serious of all operating room complications. If the patient does not have underlying cardiac disease this complication must be attributed directly to the anesthesia or to the surgical procedure. Popular acceptance of an entity is always followed by apparent increase in incidence; nevertheless we believe that cardiac arrest is occurring with greater frequency. We attribute this to more complicated anesthetic technic coupled with the surgeon's demand for longer and deeper level anesthesia. As more cases of cardiac arrest are reported, the emphasis appears to be on recognition and immediate therapy by surgical intervention. Equally important, in our opinion, is the greater need for emphasizing the cause of this unfortunate complication and its prevention.

Experience

During the last 4 years there have been 19 cases of proved cardiac arrest in the Cleveland Clinic. Prior to this time there were operating room deaths attributed to various causes such as apoplexy, coronary occlusion, embolus, drug reaction, status thymicolymphaticus, and shock; cardiac arrest, as a clinical entity, was not known. Regardless of the true cause of death in the early series, the present group of 19 proved cases represents a distinct increase in incidence. By "proved cases" we mean visual evidence of cardiac arrest during thoracotomy for resuscitation. Severe cyanosis, accompanied by imperceptible pulse, heart beat and blood pressure, is not conclusive evidence of arrest; as mentioned by Lahey and Ruzicka, spontaneous recovery may occur in this formidable state.[1] Spontaneous recovery does not occur in cardiac arrest.

These 19 cases have been distributed over most of the surgical and allied specialty services. In each instance a distinct effort has been made to critically evaluate the factors producing the arrest by presenting the case at open meeting for frank evaluation. Emphasis has been placed on the preoperative medication, anesthesia agents, position on the table, level of anesthesia, and nature of the procedure; every effort has been made to evaluate the patient's condition prior to surgery, and the anesthesia course immediately preceding the accident. Whereas one cannot expect the results of such an investigation to be accurate or even worth while in every instance, we believe that a better clinical understanding of the problem has resulted from these studies. In essence, the clinical observations gained from these 19 cases tend to confirm the experimental observations of Wiggers, Beck, Sloan, Young, and others who have

*Now at Bryn Mawr Hospital, Bryn Mawr, Pa.

investigated the cause and treatment of cardiac arrest.[2,3,4,5,6,7,8] Emergency thoracotomy for diagnosis and therapy was employed in every case. It is significant that ventricular fibrillation did not occur in this group of patients. After massage of the heart and control of respiration a sinus rhythm was established in each instance. This limited series tends to support Harris' contention that the reported rate of ventricular fibrillation is too high;[9] Harris states that the feeble undulations detected by electrocardiography in the failing heart do not contribute to death but are frequently interpreted as true fibrillation.

In the laboratory, arrest of a dog's heart may be produced by electric shock,[4] overdosage of drugs,[10] prolonged asphyxia or vagal stimulation in the presence of hypoxia.[7,8] It is the latter method that appears to be of greatest clinical significance. We believe that two factors are productive of cardiac arrest in the human heart; hypoxia and vagal inhibition. The search for a common denominator in 19 cases in which various anesthetic technics and surgical procedures were employed has led us to believe that oxygen deficiency coupled with vagal depression must be present to arrest a normal heart. These observations tend to corroborate the excellent work of Beck and Wiggers, who have repeatedly created the accident within the laboratory and have emphasized its clinical importance.[2,3,4,5,6] Most recently Sloan has shown the more exact relationship between hypoxia and vagal inhibition in producing arrest of the dog's heart.[7]

Another factor we believe to be of importance is hypercapnia associated with carbon dioxide retention. Two of our patients (table 1) both children approximately 2 years of age, suffered cardiac arrest after mechanical impairment of the endotracheal airway. Beecher[11] has emphasized the significance of high carbon dioxide tension during anesthesia and has repeatedly warned of its danger. According to Beecher the excessive retention of carbon dioxide within alveoli produces hypercapnia and acidosis; tachycardia and hypertension are clinical manifestations of increasing carbon dioxide retention. In the two cases mentioned there was mechanical impairment of the airway; the endotracheal tube had passed the carina creating occlusion of one bronchus and effectively reducing the ventilation of the contralateral lung. Both children developed progressive tachycardia and hypertension with abrupt onset of bradycardia and eventual arrest.

Hypoxia or stimulation of the vagi alone may produce serious alteration of the conductive mechanism in the heart with resultant hypotension and bradycardia. Regardless of the cause, the alteration in normal physiology is both recognizable and reversible. When both factors are present, however, depression of the heart may result in complete absence of conduction and arrest occurs.[7,8,9] In this event, release of the factor that is producing either hypoxia or vagal inhibition will not be effective unless active measures are taken to restore the heart beat mechanically. The clinical errors that produce both oxygen deficit and vagal stimulation are shared by the anesthetist and the surgeon. We believe that the commonest factors attributable to each may be categorized under two headings:

1. Anesthesia factors:
   a) Multiplicity of agents.
   b) Overdosage.
   c) Obstruction of airway.
   d) Injudicious use of tracheal aspiration.
   e) Complexity of technic.
2. Surgical factors:
   a) Insistence on "deep" anesthesia.
   b) Excessive tissue trauma.
   c) Inadequate blood or fluid replacement.
   d) Hyperpyrexia.
   e) Unphysiologic position of the patient—or abrupt changes of patient's position.

It must be emphasized that these clinical items are based on observation in the operating room and critical analyses of case histories of proved arrest. To what degree any one factor may produce oxygen deficit or create vagal stimulation is difficult to state. Nevertheless each of the listed factors has merited comment and elicited warnings by writers in the past. Potts has emphasized the importance of using minimum doses of medicine and as few agents as possible, since all are poisons;[12] he prescribes no barbiturates for children. Instances of hypoxia resulting from respiratory depression with excessive doses of morphine, pentothal or nitrous oxide, are familiar to all. Hyperpyrexia secondary to fever, dehydration, excessive draping of the patient or use of overheated operating rooms, will increase the oxygen requirement; in children[12] or severely ill adults, a serious oxygen deficiency may be created by uncontrolled body heat. Likewise the depletion of the patient's tidal volume by careless aspiration of the trachea and major bronchi under deep anesthesia is productive of temporary anoxia. The bronchial spasm produced by intubation under improper anesthetic level may be accompanied by impressive cyanosis, indicating profound hypoxia. Whereas changes in normal physiology occur frequently without serious consequences in operating rooms everywhere, the combination of unnecessary oxygen deficit with vagal inhibition under satisfactory conditions may result in fatal accidents.

Once cardiac arrest has occurred, the initiating factors may be obscured by the excitement and confusion that inevitably follows. The impression produced by a single case may have a profound effect upon both surgeon and anesthetist; more important, the impressions may be based on error and exert no value in prevention of future accidents. Too often the human tendency is to place the responsibility upon the patient and his disease. However, a critical analysis of every record obtainable assures a progressively clearer and more accurate understanding of the problem.

Whereas the sequence of events preceding cardiac arrest may be variable or obscure, a pattern of alteration in hemodynamics does occur. In most instances premonitory signs may be present and should be recognized if the accident is to be averted. Cyanosis always implies oxygen need and is a warning

sign! Hypoxia will usually produce a transient tachycardia, and perceptible elevation of blood pressures before the more serious duo of bradycardia and hypotension appear. Abrupt slowing of the pulse rate with accompanying hypotension is the predominant sign and invariably precedes arrest. An alert team of surgeon and anesthetist will accept this warning of impending disaster and make every effort to recognize and correct the underlying factor. To do so may abort the cardiac arrest as the circulatory alteration is still reversible.

Prevention

Of primary importance in prevention of cardiac arrest is recognition of the factors that may initiate oxygen deficit and vagal stimulation as well as the signs of impending cessation of heart beat. When the patient's condition shows definite change under anesthesia manifested by cyanosis, bradycardia and falling blood pressure, the alarm must be sounded. This alteration in hemodynamics may occur in the induction phase of anesthesia just as easily as in the course of a long complicated operation. At no time during the interval between the patient's induction and his return to consciousness are the patient and anesthetist entirely free of this potential hazard.

When the signs of impending arrest are recognized, the responsibility for correction of the cause of the factor falls equally on the surgeon and the anesthetist. Each must play his part in averting catastrophe.

1. Surgeon's Role:
   a) Abandon the operative procedure.
   b) Prepare to open the chest if arrest ensues.
   c) Assist in correcting blood deficit if this is a factor.
2. Anesthetist's Role:
   a) Establish airway; prompt intubation if needed.
   b) Control respiration with 100 per cent oxygen.
   c) Intravenous atropine sulfate.
   d) Continuous evaluation of pulse and blood pressure.

By sharp division of responsibility, unnecessary reduplication and confusion are avoided. When such emergency arises, those present must manage the problem; this is no time for frantic consultation with cardiologists or trusted colleagues who may be elsewhere in the hospital. Likewise, efforts by the surgeon to employ the time-honored methods of artificial respiration are only added hazards to the patient. Actually a calm, prompt effort to correct the anoxia by positive pressure via a patent airway, combined with release of the vagal stimulation by intravenous atropine sulfate, will usually suffice to abort the impending arrest.

Atropine sulfate may be an invaluable drug in such emergencies. This drug inhibits the parasympathetic effect of the vagus and its depressant action on the heart. The dosage will vary with the age and the size of the patient. The infant will tolerate a dose of 1/1000 gr. (Potts), the adult should receive 1/100 gr., or more. In all instances the drug must be given intravenously, as the

Department	Age	Physical Cond.	Scheduled Procedure	Anesthesia	Time of Arrest	Management	Outcome	Remarks
1. General Surg.	58	Fair	Whipple op. ca. pancreas	Continuous spinal and pentothal	Midway in operation	Delayed subdiaphragmatic massage and intracardiac adrenalin	Death on table	Anoxia due to hypotensive state; low blood volume secondary to inadequate replacement.
2. General Surg.	36	Good	D & C	Spinal and pentothal	Induction	Transthoracic massage	Recovery, complete	Preop. hemoglobin 8.0 Gm.; arrest followed lowering legs from lithotomy position.
3. General Surg.	46	Good	Vagotomy	Spinal and pentothal	Termination of operation	Transthoracic massage	Recovery, permanent cortical damage	Pentothal given to supplement waning spinal; respirations not assisted.
4. General Surg.	8	Fair	Excision of neuroblastoma left adrenal	Pento. induct. endotracheal GOE	Midway in operation	Transthoracic massage	Recovery—death in 6 hours due to cerebral decortication	Hypotensive state secondary to severe blood loss.
5. Neurosurgery	29	Good	Cervical rhizotomy	Pento. with endotracheal N_2O-O_2	During skin incision	Transthoracic massage	Recovery, complete	Induced hypotension (Page procedure) in upright position under anesthesia.
6. Neurosurgery	29	Good	Laminectomy	Spinal and pentothal	Induction	Transthoracic massage	Recovery—death in 1 week due to cerebral decortication	Abrupt change in position, supine to prone, under anesthesia—inadequate airway.
7. General Surg.	37	Good	Vagotomy	Spinal and pentothal	Prior to closure	Transthoracic massage	Recovery, complete	Mechanical obstruction of airway followed by cyanosis, bradycardia and arrest.
8. Thoracic Surg.	69	Fair	Esophageal resection	Pento. induction endo. GOE	Induction	Transthoracic massage	Recovery, complete	ECG evidence of acute coronary occlusion.
9. Thoracic Surg.	27 mos.	Fair	Patent duct	Endo. GOE	Completion of operation	Transthoracic massage	Recovery—died 48 hrs. later, cerebral damage	Hypercapnia due to CO_2 retention—endobronchial tube below carina.
0. Thoracic Surg.	62	Poor (gr. III heart d.)	Exploratory thoracotomy	Pento. induction endo GOE	Induction	Transthoracic massage	Recovery—died 7 days after, no cerebral damage	ECG evidence and postmortem evidence of myocardial infarction during induction.
11. Thoracic Surg.	16 mos.	Fair	Division patent duct	Endo GOE	Induction	Transthoracic massage	Recovery, complete	Hyperthermia, followed by hypercapnia with CO_2 retention. Operation completed after resuscitation.

Department	Age	Physical Cond.	Scheduled Procedure	Anesthesia	Time of Arrest	Management	Outcome	Remarks
12. Thoracic Surg.	66	Poor (grade IV heart d.)	Repair diaphragmatic hernia	Pento. induction	Completion of operation	Transthoracic massage	Recovery—died 10 days	Postmortem evidence of coronary infarction.
13. Thoracic Surg.	55	Poor (grade III heart d.)	Pericardectomy	Pento. and cocaine 4% induction	Following intubation	Transthoracic massage	Recovery, complete	Severe heart disease—arrest followed minimal anoxia associated with intubation.
14. Urologic Surg.	8 mos.	Fair	Nephrostomy (Urinary fistula following ureterosigmoidostomy	Ether	Induction	Transthoracic massage	Recovery, complete	Hypoxia secondary to massive l. hydrothorax. Marked impairment in pulmonary function at time of induction.
15. General Surg.	67	Fair	Ant. resection sigmoid	Continuous spinal; pentothal supplement	Midway in operation	Transthoracic massage	Recovery, complete	Obese patient in deep Trendelenburg's position—anoxia due to thoracic compression plus respiratory depression by pentothal without mechanical airway.
16. Ophthalmologic Surg.	65	Good	Enucleation cataract	Pentothal	Midway in operation	Transthoracic massage	Recovery—death in 3 hours from cerebral decortication	Anoxia due to inadequate airway—cyanosis not recognized under drapes.
17. Otolaryngologic Surg.	57	Poor	Bronchoscopy	Topical cocaine 4%	40 minutes after anesthesia (bronchoscopy cancelled)	Transthoracic massage	Recovery—death in 1 hour due to cerebral decortication	Chronic hypoxia with severe cardiopulmonary disease; anoxia precipitated by severe bronchospasm with topical anesthesia.
18. Thoracic Surg.	52	Poor	Bronchoscopy	Topical cocaine 4%	15 minutes after endoscopic procedure	Transthoracic massage	Recovery—minimal added cortical damage	Note: Diag. cardiac arrest made 3 months before during treatment elsewhere for intractable asthma; recovered with marked personality change.
19. Neurosurgery	37	Poor	Suboccipital craniotomy	Pentothal and local (procaine 1%)	During removal of skull plate	Transthoracic massage	Unable to restore normal heart rate	Pressure on medulla by intracranial cyst producing paralysis of respiratory center.

Table. An effort has been made to give a digest of the pertinent information concerning each of the 19 cases of proved cardiac arrest. Under the subject OUTCOME the term RECOVERED is used whenever the quiescent heart has been resuscitated and sinus rhythm restored. This does not mean that the patient has been saved; the ultimate fate of the patient is recorded separately.

failing circulation may not assimilate a subcutaneous or intramuscular medication.

Restoration of normal pulse and blood pressure is synonymous with correction of the underlying causative factor. There should be no residual cortical damage associated with this phase of hypoxia. The election as to continuation or abandonment of the surgical procedure is a matter of judgment. This decision will depend entirely upon the circumstances associated with the individual case.

Therapy

Much has already been written upon the surgical treatment of cardiac arrest. Nevertheless, for the sake of completeness and for the importance that the problem merits, a certain amount of repetition is justifiable.

Again we believe that the responsibility of therapy must be divided between the surgeon and the anesthetist. Each must carry out his assignment with dispatch and with a minimum of confusion and duplication of effort.

1. Surgeon's Responsibility:
    a) Prompt thoracotomy—4th, 5th, or 6th anterior interspace, left.
    b) Manual compression of the arrested heart for restoration of circulation.
    c) Visual inspection of the heart if recovery is not reasonably prompt.
    d) Intracardiac epinephrine, only (1) if restored beat is feeble, or (2) if there is no spontaneous recovery of beat following continuous manual compression.

Any incision that will give adequate access to the arrested heart is satisfactory. We do believe that the most desirable approach is through the left anterior thorax; the incision should be made with whatever blade is available and with complete disregard for the usual operating room rituals of skin sterilization and wound draping. Upon opening the chest it may be necessary to cut or fracture a costal cartilage to permit entry of the surgeon's hand. The wound may be retracted by the assistant employing his fingers or any form of available retractor. It is not necessary to open the pericardium, and manual compressions should be started immediately. The rate of compression will depend entirely on the rate of filling of the inert heart. Frequently the cardiac rhythm will respond promptly with only a few compressions by the surgeon's hand. When this occurs no further therapy need be carried out, but a period of observation is in order before closing the thoracic wound. When the heart beat is feeble or fails to return spontaneously, then intracardiac epinephrine in small doses is indicated. It has been our custom to use approximately ¼ cc. of epinephrine 1:1000 directly into the left ventricular chamber. Closure of the chest wall is a minor problem and can be accomplished with pericostal chromic catgut sutures and whatever material is available for closure of the muscle and skin layers.

2. Anesthetist's Responsibility:
   a) Prompt intratracheal intubation.
   b) Oxygen 100 per cent under intermittent positive pressure.
   c) Intravenous atropine sulfate, if bradycardia follows restoration of heart beat.

The importance of positive pressure oxygen therapy in resuscitation of the heart cannot be overemphasized. This is best carried out by means of a closed system employing the endotracheal tube. When properly executed, intermittent positive pressure anesthesia in itself can maintain a demonstrable circulation of blood through the lungs and to the brain. Correction of the anoxia by the anesthetist and intravenous administration of atropine sulfate, if indicated, will correct the initiating factors of hypoxia and vagal inhibition that produced the cardiac arrest.

Restoration of a normal sinus rhythm is not necessarily indicative of a return to normal physiology. Severe cortical damage will follow brief periods of anoxia and central nervous system damage may follow. It is for this reason that the emphasis on speed of recognition and initiation of therapy cannot be overemphasized. The exact period of time that may elapse between the onset of anoxia and severe cerebral damage is not known. It is dangerous for the surgeon to rely upon an arbitrary time interval before initiating the course of therapy that is clearly indicated.

Discussion

Cardiac arrest is a complication of anesthesia and surgery and is rarely a primary disease. For this reason we must look to ourselves in seeking the cause of a given accident; to blame the patient and his disease is fundamentally misleading. This unfortunate error has occurred more frequently than we care to admit even though it is often classified by another term. Every experienced surgeon and anesthetist can recall instances of sudden death that may well have been due to cardiac arrest.

In our experience cardiac arrest has occurred more often in the good risk patient than in the so-called bad risk person who is being subjected to a surgical procedure. This is an important observation on the principal causes of cardiac cessation which have been discussed. When a poor risk patient is brought to surgery meticulous care is used in the selection of anesthesia agents and he is closely scrutinized during the induction and subsequent course of anesthetic administration. Usually such a patient is subjected to the least amount of trauma and blood loss and is invariably given the benefit of a high supplementary oxygen intake. It is undoubtedly the combination of careful observation and basic precautions which eliminate the causative factors of cardiac arrest in these cases. The higher mortality rate associated with the poor risk patient is explained on complications other than true cardiac arrest. It is for this reason that the tragic sequela to cardiac arrest in the so-called

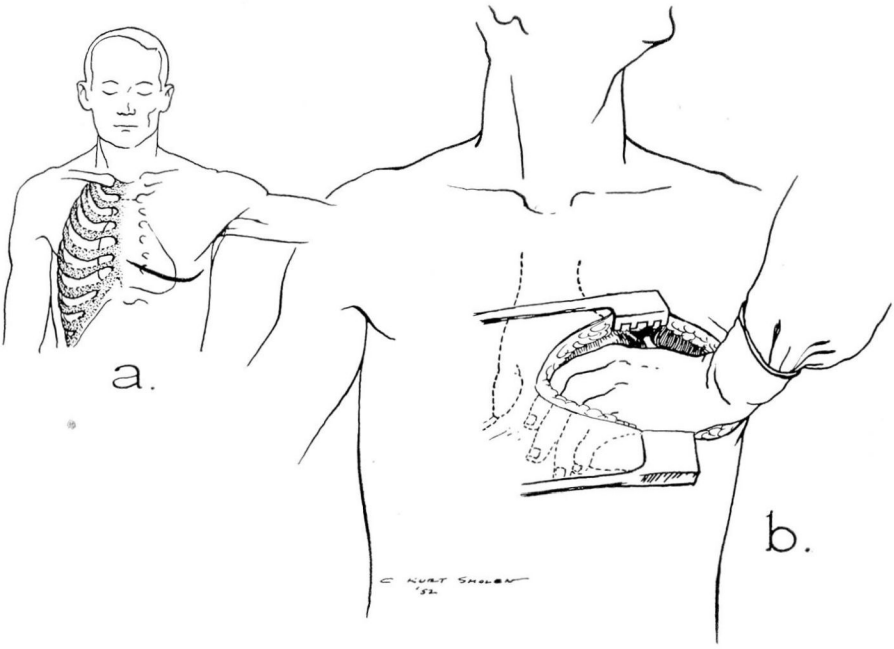

FIG. 1. Usual site of emergency thoracotomy for cardiac resuscitation is illustrated by diagram. Manual compression of the heart to maintain blood flow may be performed by alternate use of right and left hands to combat fatigue. The element of time is too precious to be sacrificed for technic! When necessary, resuscitation must be performed through an unprepared field by bare hand.

good risk patient must be kept in mind by all who practice surgery and anesthesiology. Until we are willing to accept the precursors of cardiac arrest as avoidable errors attributable directly to the anesthetist and surgeon there is little possibility that this dreaded accident will be eliminated from the list of operating room complications.

In the manner of the old fashioned fire drill it might be well for all surgeons and anesthetists to anticipate this tragedy and analyze the causative factors in past cases. Elimination of potential cause for hypoxia will, in itself, almost completely exclude the possibility of cardiac arrest in the good risk patient. A clear understanding of the roles of the anesthetist and surgeon in cardiac resuscitation may be of tremendous value in avoiding procrastination and confusion. Most important is a frank acceptance on the part of the operating team of the exact nature of the complication and its cause. The only stigma attributable to a case of cardiac arrest is that associated with failure to recognize the entity and delay or failure in carrying out the indicated resuscitation. The most important factor of all is time.

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