Contemporary interest in resuscitation was historically related to anesthetic death. Primitive techniques of anesthetic administration, loss of airway control, and psychologically influenced sudden death contributed to unanticipated respiratory and cardiac arrest. Airway obstruction has remained the principal factor in asphyxial death, necessitating crucial preservation of respiratory function during induction of anesthesia.

Early, disorganized overdose and arrest interventions included: application of cold water, manual artificial respiration, heat, friction and galvanic battery application.

Cardiopulmonary resuscitation, after years of research and experimentation became an integrated plan of attack: mouth-to-mouth ventilation and maneuvers eliminating pharyngeal obstruction were proven effective; internal and external cardiac massage was incorporated and definitive drug therapy began with epinephrine, strychnine, caffeine, carbon dioxide, amyl nitrate, coramine, metrazol and procaine. Defibrillation proved electricity converted ventricular fibrillation to normal sinus rhythm.

Significant lethality still occurs from anesthetic-induced cardiac arrest, despite technological advances. Causes of operating room cardiac arrests are numerous and include sudden death syndrome. Constant vigilance distinguishes variable patient response. Immediate recognition and coordinated intervention assures success.

Causes of early anesthetic arrests
Fifteen-year-old Hannah Greener was the first patient to die under general anesthesia, losing her life on January 28, 1848, as she received a chloroform anesthetic for removal of an ingrown toenail. Her death, 18 months after the public demonstration of ether in Boston, immediately generated substantial concern and discussion. A painstaking investigation was implemented, but a second chloroform-related death occurred before any conclusions could be accepted. These incidents understandably troubled James Y. Simpson, chairman of obstetrics at Edinburgh, who had enthusiastically incorporated routine use of chloroform in his midwifery practice. Simpson's motivation to investigate chloroform's effects had been encouraged by Liverpool chemist David Waldie and research on the drug's properties, furnished by the French physiologist Flourens. He observed with growing anxiety as inquiries and analytical discussions of the French Academy of Medicine, Hyderabad Commissions, Royal Medical and Chirurgical Society, British Medical Association and the American Medical Association's newly organized Commission on Anesthesia established the occurrence of anesthetic-induced cardiac arrest.
Growing recognition of adversity associated with employment of general anesthesia stimulated interest in resuscitation, and in 1858, John Snow described 50 cases of sudden death under chloroform anesthesia due to what he delineated as paralysis of the heart.

Though frequent deliberations have persisted since Hannah Greener's death, all anesthetic agents and techniques have been associated with cardiac arrest, a disaster chronically resulting from an assortment of errors that include inexperience, inattentiveness and ignorance regarding pharmacological interactions.

Four anesthetics were in everyday use at the turn of the century: ether, chloroform, nitrous oxide and ethyl chloride. The accepted order of safety placed ether first, nitrous oxide second, ethyl chloride third and chloroform last. Not until the early 1920s were ethylene and cyclopropane clinically tested, approved and accessible anesthetic agents.

Prevailing methods of ether administration condoned dripping it onto a towel or an Esmarch mask as the patient breathed spontaneously. Somewhat later, delivery was managed by means of a Connell hook tube or Connell nasopharyngeal tube. Endotracheal tubes were rarely used, especially in the period before the technique of intubation was widely accepted.

While fatalities with ether were rarer than with chloroform, they were known to occur at any time during the course of anesthetic administration. Problems were often caused by anesthetic concentrations that were too high. Respiratory arrest under ether was regarded as less dangerous. The heart was not as depressed and usually remained beating until the concentration could be diluted and respirations resumed. Death under deep ether anesthesia was caused by respiratory paralysis. Danger signs indicating the onset of this condition included sudden pupillary dilatation, pallor and the loss of facial tone. Contributing factors to the onset of this problem were fright and struggling on induction, irregular respirations and a variety of idiosyncratic reactions. It was recognized that behavior during the induction of an anesthetic was adversely affected by a history of alcohol or drug use. Crying children were considered especially dangerous to the anesthetist, and pediatric inductions were cautiously initiated with dilute ether vapor, which was meticulously regulated when the anesthetic concentration was increased. Preoperative morphine was given for fear prevention and atropine eliminated vagal response.

Nitrous oxide was routinely administered according to McKesson's technique of primary and secondary saturation. Primary saturation was obtained by delivering 100% nitrous oxide until the first anoxic sign was observed. Then 5-10% oxygen was added to the nitrous oxide. The patient was usually unconscious but could still move. Secondary saturation was achieved by giving 100% nitrous oxide until the patient developed signs of the profound plane: a dilated pupil that did not react to light, prolonged exhalation, rigid muscles and cyanosis. One or two breaths of 50-100% oxygen was then given to relieve the anoxic spasm, leaving the patient relaxed and anesthetized.

Use of ethyl chloride embraced hazards analogous to those of chloroform in that relatively small doses could precipitate cardiac standstill. Large doses were known to directly paralyze the heart muscle. This anesthetic was customarily administered in doses of 5-10 cc by means of a closed mask that exposed the patient to partial asphyxia. Although there was minimal excitement and struggling during the induction, it was difficult for an anesthetist to determine the proper time for the surgeon to commence the operation.

Dennis Jackson had designed a closed-circuit inhaler for ethyl chloride in 1916, but dispensing the drug by this method was regarded as a dangerous practice. The recommended procedure was to drip it on two layers of gauze placed over the patient's mouth and nose, limiting the amount of anesthetic applied to 5 or 10 cc. Anesthesia was produced in 1½ to 2 minutes. The patient recovered rapidly, usually without vomiting. Ethyl chloride was an insidious respiratory depressant and an anesthetist could easily be deceived by observing a patient's apparently normal color and heart action for some minutes after respiration had ceased.

Chloroform continued to be tolerated in anesthetic practice, despite an increasing regard for its hazards, because it was convenient for the patient and the anesthetist. Its action was significantly more rapid than the other available anesthetics, the taste was not disagreeable, and it precipitated less suffocation, excitement, and postoperative nausea and vomiting. Anesthetizing a patient with chloroform was discouraged if there was documented history of Graves disease, hemorrhage, expectation of prolonged surgery or an existing acidotic condition.

Accumulating evidence corroborated the significant possibility of death during chloroform induction. Of 20 patients, expiring in 1896 while receiving chloroform, half succumbed within 6 to 15 minutes after induction had commenced.

Most chloroform fatalities occurred as a result of reflex vagal stimulation. Sudden cardiac arrest was frequently the consequence of nasal, laryngeal, tracheal or pulmonary irritation, which was aggravated by chloroform vapor in concentrations ap-
proaching 2%. Sufficient anesthetic depth, reducing the probability of arrest, was assured when vagal hyperexcitability was thoroughly abated.

Asphyxia, associated with the onset of laryngospasm, propagated cardiac dilation, vagal stimulation and finally heart failure, rapid in its onset from chloroform’s direct cardiac action. The heart, particularly vulnerable under light chloroform narcosis, was susceptible to ventricular fibrillation. Respirations sometimes resumed briefly after the heart had stopped, but arrested circulation could not be restored. Failure of the circulation, therefore, preceded the final respiratory arrest and was the true cause of death.8

Maintaining an even anesthetic plane and remaining continually vigilant for signs of trouble were the keys to avoiding an incident. An anesthetic plane manifesting complete muscular relaxation and absence of sensitive reflexes afforded the best protection for the patient and the anesthetist. An incident, developing late during an anesthetic course, frequently resulted from an anesthetist’s inexperience, either in preventing the anesthetic concentration from becoming too high or failing to observe and react to the danger signals of overdose: an increase in a shallow or irregular respiratory pattern, a slow or rapid pulse, or cyanosis and dilation of the pupil.8

Attention to the condition of the patient’s respiratory state was essential throughout the administration of an anesthetic but crucial during the period of induction due to dilemmas that often arose at this time. If the patient held his breath, reacting to an excess of chloroform vapor, the anesthetist had to prudently withdraw the inhaler, as the next breath would often be extraordinarily deep and the patient would receive an overdose. This problem was also confronted when inducing a crying child or if a surgeon cut prematurely, before the patient had entered the third stage of anesthesia. When administering chloroform, it was imperative to use only the amount of anesthetic necessary to keep the patient anesthetized. An uneven pattern of administration resulted in the onset of excitement, struggling, respiratory cessation, overdosage and shock. Autopsy in these individuals revealed nothing beyond the phenomena of asphyxial death: distended heart and congested veins.8

Every death that occurred on the operating table was not always attributable to anesthetic mismanagement. Patients had died for apparently unexplained reasons in preanesthetic days. One story implicated the French surgeon Desault. He was reported to have merely drawn his fingernail over a patient’s perineum to mark an incision line, when this individual cried out in pain and died.8

Anecdotes of sudden death, concerning apprehensive patients who died after simply entering the operating room or had suffered respiratory arrest and death following placement of the face mask before anesthetic was actually applied, occurred periodically in early anesthetic literature.10 One incident, of particular importance in the history of anesthesia, occurred when Simpson was about to try chloroform on a patient for the first time. The orderly, bringing him the anesthetic, fell and spilled the chloroform. No other chloroform was readily obtainable and Simpson decided to perform the hernia surgery without anesthesia. His patient succumbed when the skin incision was made. Had Simpson used the chloroform for this case with the occurrence of this accident, the drug’s introduction into anesthetic practice would undoubtedly have been suspended or terminated.8

**Early attempts at intervention**

Reports of early anesthetic administration and resuscitation procedures were described and recorded by two female medical students, Jennie I. Chapin and Harriet Bottsford, in dissertations written during their respective senior years of medical education at the Woman’s Medical College of Pennsylvania in Philadelphia. These students had personally observed administration of ether and chloroform at Pennsylvania Hospital during the years 1866-1875.

*An Abstract on the Subject of Chloroform* was written by Jennie I. Chapin in 1866. Commenting on techniques of resuscitation, she explained that the patient was immediately placed supine if the anesthetic had been administered with the patient sitting, which it often was. Cold water was then applied to the head, face and chest, while heat and friction was applied to the bulk of the body. Artificial respiration was started by one of the manual methods while ammonia was applied to the nostrils.11

Harriet Bottsford’s description of resuscitative methods in her 1875 dissertation, *Anaesthesics*, included the application of a galvanic battery (faradization). One of the battery’s electrical poles was placed on the spinous processes of the cervical vertebrae, the other on the precordial region. She explained that the objective of this procedure was to arouse the respiratory and cardiac ganglia, but did not indicate the positive and negative placement of the poles. She additionally reviewed the use of a surgical opening into the cricothyroid membrane for artificial respiration and the use of inversion resuscitation, a method dating from 1770, incorporating head-down suspension of the victim and gravitational force to circulate blood to the brain.12 13
Application of bilateral traction to the ascending mandibular rami was first described by Esmarch in 1878 and has remained the most commonly used method during anesthesia when a face mask is used. Extension of the head has also been recognized and practiced for many decades. Although the mouth-to-mouth maneuver is the oldest form of artificial respiration, it was abandoned for nearly a century in favor of the manual methods, the most popular and widely taught being the Sylvester method, the Schafer prone pressure technique and the Holger-Nielsen maneuver.14

The Sylvester method was performed by positioning the victim on his back. Inspiration was produced by raising the arms above the head. The victim’s arms were then carried forward, folded across the chest and pressed to produce expiration. The Schafer prone pressure technique was instituted by applying pressure to the victim’s back, forcing the abdomen against the diaphragm.Expiration was produced by compression of the lungs. Release of this pressure produced inspiration.15

Holger-Nielsen resuscitation was widely taught and practiced during and after World War II. A resuscitator knelt at the head, facing the prone victim. The head was turned laterally and the arms were flexed with the hands beneath the head. Inspiration was provided by compressing the lower posterior chest. Inspiration was created by the resuscitator rocking backward, lifting the victim’s arms upward at the elbows and pulling them forward. This hyperextended the spine, decreased pressure on the anterior chest and increased thoracic volume.15

Open-chest cardiac massage was first demonstrated on dogs in 1882 by Moritz Schiff, professor of physiology, in Florence, Italy. He described this procedure of opening the thorax to provide direct, rhythmic cardiac compressions while simultaneously insufflating air into the lungs. Schiff documented that using this method, heartbeat could be reestablished 11 1/2 minutes after cessation.14 Although the first report of its use on a human being by Neilhaus was not a successful attempt, this technique gained acceptance early in this century within the hospital setting, as it manifested increased survival with sensory and motor recovery.14,16 It was not until 1901 that the first successful resuscitation, using open-chest massage was reported to have been carried out by Ingelsrud in Norway.14 Lane and Gray, in 1902, resuscitated two anesthetized patients by squeezing the heart through a diaphragmatic incision and reported some of the first successful human resuscitations. Until 1960, internal cardiac massage remained the only acceptable procedure.17

Numerous reports on the effectiveness of open-chest cardiopulmonary resuscitation have been extremely variable. Stephenson, in 1958, reported a 29% survival in 1,710 cases. Lahey’s smaller study in 1950 claimed a survival rate of 80-100%. Stone’s report of 148 cases published in 1961 indicated no survivors.14 Talbot, reviewing reports and statistics in 1965, estimated prevailing survival with sensory and motor recovery between 22% and 33%.16

Attempts at closed-chest compression of the thorax are thought to have been first performed by John Hovard in the mid-eighteenth century, a method he abandoned after having the misfortune to break a patient’s ribs in front of a police inspector.18 Esmarch, during early open-chest experimentation, proposed pressure application to the left thorax for circulatory reestablishment as part of the widely used Sylvester maneuver (chest-pressure, arm-lift) with the patient placed in the Trendelenburg position.14 Koenig published a description of external compression in 1885 and claimed successful resuscitation of six patients by this technique.14 Maass reported the resuscitation of two patients in 1891, one a pediatric patient who was resuscitated from anesthetically related cardiac arrest by closed-chest massage, combined with utilization of artificial ventilation in the manner described by Koenig. He reported successful results, explaining that the pupils, previously dilated as a result of circulatory failure, constricted and resumed response to light, and that carotid pulses became palpable during and after chest compression. He elaborated that this type of chest compression had a minimal effect on pulmonary ventilation, and few untoward effects on the sternum.14

Drug therapy in cardiac resuscitation began with Crile’s introduction of epinephrine in 1904, now regarded as one of the great advances in the treatment of sudden cardiopulmonary arrest. Crile found that early use of epinephrine markedly improved resuscitation efforts.14

Strychnine and caffeine were used in early resuscitation protocols. Strychnine, the primary alkaloid of nux vomica, increased the reflex excitability of the spinal cord and of the medullary centers. Through action, chiefly on the dorsal gray matter of the cord, therapeutic doses produced a tonic effect on the alimentary canal, improved muscle tone and provided limited respiratory and vasomotor stimulation.4 Caffeine, one of the methyl derivatives of xanthine, increased reflex irritability of the central nervous system in a descending direction. Its stimulation of the respiratory medullary centers was the conspicuous effect. Therapeutic doses of caffeine were found to be more potent and prolonged in action than strychnine.8

In 1918, Henderson and Haggard recom-
mended postoperative inhalation of air containing 1-8% carbon dioxide, asserting that ether could be more rapidly eliminated by respiratory stimulation. While controversy existed regarding the harmlessness of carbon dioxide, this theory was also practiced during induction to hasten attainment of equilibrium between anesthetic vapor and the tissues, permitting use of lower anesthetic concentrations. Protocol for anesthetic-induced cardiac arrest, found in Sollmann's 1917 pharmacology text, specified that anesthesia be terminated and the patient's head lowered to permit the heart and medullary centers the benefit of remaining circulation. Alternating rhythmic compressions of the epigastrium and cardiac region were then initiated at a rate of 40 per minute. Recommendations for drug therapy included an intravenous infusion of 1 liter of saline containing 1 cc of 1:1000 epinephrine and unspecified doses of strychnine or caffeine. Artificial ventilation with 100% oxygen or room air was viewed as acceptable, though 100% oxygen probably had no advantage over room air if not immediately accessible. Electrical stimulation (faradization) to the phrenic nerve or heart was additionally reviewed. Commentary suggested that initiation was prompted by desperation, rather than by a rational view of what was actually accomplished by its performance. While phrenic nerve stimulation caused diaphragmatic contraction and inspiration, this technique was not thought to be as effective as artificial respiration. Arguing that an additional consequence might well be vagal stimulation, Sollmann suggested that time required to procure and adjust the necessary apparatus might be better spent in effectively performing artificial respiration.

Treatment of overdose and resuscitation, taken from Glaister's 1921 British text on toxicology, included artificial respiration by patient inversion and rhythmic tongue contractions, tracheotomy, application of electric current to the phrenic nerves, inhalation of amyl nitrate and strychnine administration by subcutaneous injection. A prepared anaesthetis was to have available a catch forceps for grasping the tongue and a hypodermic syringe containing strychnia solution.

Development of modern resuscitation

Critical investigations conducted after 1958 with unconscious patients, illustrated upper airway effects of head and mandible position. X-ray studies of anesthetized patients confirmed that the base of the tongue collapses against the posterior pharyngeal wall, obstructing air flow in the supine, lateral or prone position. Obstruction also occurred if the head was in the neutral position or anteriorly flexed. It was demonstrated that continuous support of the head and jaw, combined with extension of the head and mandible, would usually provide a patent pharyngeal air space.

Dripps and his associates conducted studies on intubated, curarized subjects, measuring the amount of air moved by the manual methods in use during the early 1950s. Recognizing the period of permissible apnea might only be one or two minutes in a hypoxic patient, these studies were conducted with the intention of identifying the manual method that provided the best ventilation, considering encumbrances faced by the operator. The Schafer prone pressure method moved about 500 ml with each compression, while the Holger-Nielsen method exchanged an average of 1,050 ml per respiratory cycle. Dripps verified that the Silver method moved about the same quantity of air as the Holger-Nielsen technique. Modifying these techniques by alternating chest compression and movement of the hips or shoulders moved an average of 1,150 ml in these unconscious, curarized, intubated individuals. Dripps determined that all of these methods could move an adequate volume of air, provided the trachea was intubated. He also demonstrated that considerable energy on the part of the resuscitator was required. Limited air movement transpired if the airway did not remain patent and the patient was not promptly intubated. Dripps endorsed the Silver method as the most serviceable, as it provided the maximum volume of air movable to the least expenditure of energy by the resuscitator.

Elam and Greene concurrently studied mouth-to-mouth and mouth-to-mask maneuvers, encouraging universal application of these techniques. These techniques became uncontested standards for intervention in respiratory failure, as with minimal effort, oxygen saturation and carbon dioxide exchange could be maintained for protracted periods.

External cardiac compression ultimately earned recognition in 1960, when Kouwenhoven, Jude and Knickerbocker reported in JAMA a 70% resuscitation accomplishment in 20 patients, asserting that systolic pressures between 60-100 mmHg had been generated and sustained. Three of these resuscitations involved patients in ventricular fibrillation who were externally defibrillated with alternating current.

Ventricular fibrillation had for centuries been recognized as a terminal event. Prevost and Batelli were able to show in 1899 that powerful electric shocks applied directly to the heart could convert ventricular fibrillation to sinus rhythm. The first clinical application of this theory was reported in
1947, when Beck successfully resuscitated a 16-year-old boy from ventricular fibrillation by applying alternating current directly to the heart. Following further development of the external defibrillator by Zoll and Kouwenhoven, use of electric shock became common in the operating room.11

Cardiac arrest protocols reviewed by Adriani, in 1947, implied that Schafer's method of artificial respiration was acceptable for general purposes, but insufflation employing anesthesia apparatus was more convenient for operating room episodes.19 Adriani encouraged the use of 100% oxygen for resuscitation, discredited carbon dioxide inhalation and strongly supported the performance of a tracheotomy, asserting that consequences from failure to adequately access the airway were far worse than those of the surgery.

Respiratory failure drug intervention included intravenous administration of 100 mg of 10% metrazol and 250 mg of 25% coramine. These served to stimulate the medullary centers and the carotid body which in turn reflexly activated the respiratory center.

Adriani repeatedly stressed superlative artificial respiration, warning that the oxygen desperately required by the patient could not be administered from an ampule.20

Management of circulation sanctioned internal cardiac massage through the chest or abdomen, administration of intracardiac injections of epinephrine, fluid resuscitation and use of cortical extract. Compressions were judged to be effective if peripheral pulses were palpable, pupils were found to be constricted, lung inflations were satisfactory and skin color improved. If sinus rhythm was returned, 50 mg of a 1% solution of procaine was administered intravenously for stabilization.

Support for these protocols was further substantiated from a study of 45 patients, between 1925-1954, who received open-chest massage and proper artificial respiration within four minutes of arrest. Twenty-six of them (58%) recovered without neurological deficit.21

Comments on contemporary practices

Factors related to the specific environment and the skill of personnel contribute profoundly to a patient's anesthetic risk. Accepting that one cannot deny an anesthetic to a patient urgently requiring an operation, estimates of an individual's anesthetic risk remain significantly intuitive; however, an improper anesthetic choice, technique or management increases overall risk.17 Determination for safe anesthesia considers the individual patient, required surgery and operator skill.1

Anesthetic drugs and administrative techniques combined with surgical stress are inherently toxic, produce arrhythmia and can result in fatality, propagating well-documented progressive mortality after the first six to eight hours in adults and beyond the first hour in geriatric and infant patients.23,24

An anesthetic-related death can always occur during induction as a result of pulmonary aspiration or failure to secure an airway, resulting in hypoxic insult.4 Preventable problems leading to cardiac arrest under anesthesia have been attributable to an assortment of mishaps including onset of high spinal or epidural obstruction of endotracheal tubes, esophageal intubation, failure of the oxygen supply, ventilator disconnections, accidental infusion of air embolus and administration of the wrong drug.24

Jude and Elam postulated in 1965 that asphyxia and anesthetic overdose could be implicated once in approximately 1,500 to 2,000 general anesthetic administrations and argued that five to ten catastrophes could easily occur every year in a large institution.14 In 1976, Collins placed overall operating room mortality at one death in every 1,000 to 2,000 operations, implying that anesthetic-related deaths represented one incident in every 3,000 to 5,000 anesthetics administered. He characterized existence of an obligatory death rate, attributable to pharmacologic and surgical stressors and error development related to inattention and fatigue.1

Tinker reiterates that although a lack of vigilant care and poor judgement are unquestionably contributing factors in anesthetic related death, patients can and do die despite optimal anesthetic care. In 1986, he ascertained that between one and three patients died for every 10,000 anesthetics administered, primarily as a result of anesthetic administration, and he commented that more than 50% of those deaths would be currently judged preventable.22

As Pierce has substantially summarized, many authorities in anesthesia advocate universal monitoring that incorporates electrocardiography, continual oxygen analysis, pulse oximetry and capnography, pressure sensitive disconnect alarms, automatic recording of blood pressure and attention to body core temperature.25 This concept was recently further supported by Tinker's work concerning the American Society of Anesthesiologists' closed claims analysis of malpractice cases and Eichhorn's recent investigations in the nine hospitals comprising the Harvard Department of Anesthesia. Tinker's review of malpractice cases established that prevalent use of pulse oximetry and capnometry was decisive in prevention of anesthetic mishaps. Approximately one-third of the cases carried to
judgement or settlement and more than half of the deaths might have been prevented if additional, currently available monitoring had been utilized.\textsuperscript{26}

Working with these guidelines for safety monitoring, particularly oxygenation, ventilation and circulation, Eichhorn reported an impressive accident-rate reduction from 1979 through mid-1988. During this period, 1,001,000 ASA physical status I and II patients were anesthetized in components of his department with an occurrence of one accident in 91,000 and one death in 200,200 anesthetics. He related that comparing anesthetic morbidity and mortality rates was complicated due to the dearth of large scale studies, resistance from physicians and insurance companies to record review of adverse events and conflicting definitions of anesthetic-related occurrences.\textsuperscript{27}

While monitoring is not intended to replace human supervision, mistakes may often be detected with an alarm system before patient injury occurs. Immediate recognition and initiation of intervention is the best assurance of overall success in treatment.\textsuperscript{24}

Cardiac arrest develops from impaired electrical cardiac activity, myocardial contractility, or impaired venous return and cardiac output. Therefore, indications for resuscitation involve cardiac arrest resulting from cardiovascular collapse, ventricular fibrillation or ventricular standstill.\textsuperscript{4} Dysrhythmia is a frequent occurrence, appearing in 60-90% of patients that undergo anesthesia and surgery. Intubation, hypertension and vagal reflex can all produce cardiac ischemia.\textsuperscript{21}

Appropriate initial actions for the anesthetist, following recognition of lethal dysrhythmia sudden arrest include turning off all anesthetic agents, calling immediately for required help, flushing the circuit with 100% oxygen and ventilating the patient. The circuit may contain excess anesthetic concentration and should be adequately flushed. A patent airway for the victim should be quickly secured; intubation should be performed rapidly. External chest compressions should be initiated if required, the defibrillator should be obtained and charged and emergency drugs should be made available.\textsuperscript{3}

Basic life support is the emergency first aid procedure that encompasses recognition of airway obstruction, respiratory arrest, and cardiac arrest and application of cardiopulmonary resuscitation (CPR). Performance of CPR involves providing and maintaining a patent airway and the institution of artificial ventilation and circulation with external cardiac compressions. Advanced cardiac life support (ACLS) is continued basic life support and the use of adjunctive equipment, intravenous infusion, drug administration, cardiac monitoring, control of dysrhythmia and postresuscitation care.\textsuperscript{14}

Definitive therapy is directed toward the specific cause of the arrest. Personnel who provide ACLS must possess thorough ECG interpretation skills and understand the immediate and potential dangers of life-threatening and lethal cardiac dysrhythmia.\textsuperscript{29}

Ventricular fibrillation and asystole (cardiac standstill) are always associated with absence of pulse and perfusion. The life-threatening events include ventricular tachycardia and third-degree heart block. A pulseless victim of ventricular tachycardia may have a ventricular rate rapid enough to prevent proper refilling of the heart. Some individuals experiencing this dysrhythmia do have a pulse, but ventricular tachycardia can deteriorate rapidly to ventricular fibrillation.

Complete heart block causes cardiac arrest unless an idioventricular or low nodal pacemaker maintains ventricular contractions. A pulseless victim possessing an organized ECG pattern is exhibiting electromechanical dissociation (EMD). True EMD is a symptom of extensive myocardial damage, although cardiac tamponade and massive pulmonary embolus may mimic the phenomenon.\textsuperscript{26}

Intraoperative deaths have multiple causes, embracing surgical, anesthetic and individual patient factors, including the occurrence of natural death during surgery.\textsuperscript{4} Approximately 350,000 Americans die annually of sudden death syndrome, a natural death occurring within one hour after the collapse of an apparently healthy individual. Almost all of these deaths are assumed to be the result of a fatal arrhythmia or cardiac arterial spasm, but a disconcerting number of occurrences do not correlate with the autopsy findings and psychological factors have been suggested as a precipitating cause. While this problem in no way accounts for the majority of deaths associated with anesthesia, its existence as a problem remains significant.\textsuperscript{21} It is a lingering reminder from the past that those who perform surgery and administer anesthesia must be constantly vigilant to variable patient response in the operating room.\textsuperscript{3}

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