




DISEASEDEX™ Emergency Medicine
Cardiac Arrest Sample Document

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Cardiac Arrest

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Summary

0.1 CRITICAL FOCUS

- A. GENERAL: Any patient with absent spontaneous respirations and pulses has sustained a cardiopulmonary arrest.
1. Basic life support must be initiated promptly, followed by advanced cardiac life support (ACLS), which includes airway management and correction of circulatory compromise. Evaluation for causative factors critical.
 2. Arrhythmia management is a key to successful resuscitation, utilizing rapid defibrillation (most critical step in cardiac arrest due to ventricular fibrillation) and pharmacologic therapy.
 3. Continuous monitoring of pH, PCO₂, PO₂, and rhythm is essential.
- B. IV LINES: Large-bore IV access must be available for fluid resuscitation and medication delivery; alternative routes include endotracheal administration and intraosseous infusion (children).
- C. AIRWAY MANAGEMENT:
1. ESTABLISH OPEN AIRWAY: Tongue is most common cause of airway obstruction in the unconscious victim.
 - a. HEAD TILT-CHIN LIFT MANEUVER: Method of choice for opening the airway.
 - b. JAW-THRUST MANEUVER: Safest approach to opening the airway of a victim with suspected neck injury; technically difficult and fatiguing.
 - c. ENDOTRACHEAL INTUBATION:
 - (1) Should be done as soon as possible in the course of resuscitation by an experienced healthcare provider. Once inserted, ET tube should be secured and placement verified with primary (physical exam) and secondary (nonphysical exam, eg, end-tidal CO₂ detector) techniques.
 - (2) If ET has been placed and there is a delay in obtaining venous access, several medications may be administered via ET route, including epinephrine, atropine, and lidocaine.
 2. BREATHING:
 - a. RESCUE BREATHING:
 - (1) WITHOUT OXYGEN: Give 2 breaths slowly over 2 seconds initially, followed by 10 to 12 breaths per minute (1 breath every 4 to 5 seconds). A volume of 700 to 1000 mL (approx 10 mL/kg) is sufficient for most adults. Because of the wide variation in the size of children, precise volumes cannot be recommended. Experts state the correct volume for each breath is that which causes the chest to rise.
 - (2) WITH OXYGEN (>=40%): Smaller tidal volume of 400 to 600 mL (6 to 7 mL/kg) delivered over 1 to 2 seconds, followed by 10 to 12 breaths per minute (1 breath every 4 to 5 seconds).
 - b. CHEST COMPRESSION TO VENTILATION RATIO:
 - (1) ADULTS AND CHILDREN 8 YR AND OLDER: For patients with unprotected airway (not intubated), ratio is 15:2 for both one- and two-rescuer CPR. Once airway is secured, continuous compressions and asynchronous ventilations with ratio of 5:1 is recommended.
 - (2) CHILDREN UNDER 8 YR: 5 compressions to 1 ventilation for one- and two-rescuer CPR.
 - c. CHEST COMPRESSION-ONLY CPR: Recommended for use when the rescuer is unwilling or unable to perform mouth-to-mouth rescue breathing or in dispatch-assisted CPR.

3. OXYGEN: Administer 100% O₂ via bag-valve-mask or mouth-to-mask, or via endotracheal tube.

D. CIRCULATION:

1. DETERMINE PULSELESSNESS:

- a. LAY RESCUERS: Assess for absence of signs of circulation (normal breathing, coughing, or movement). Pulse check no longer recommended for lay rescuers.
- b. HEALTHCARE PROVIDERS: For children ≥ 1 yr old and adults, use carotid artery. For children < 1 year of age, brachial artery is recommended.

2. EXTERNAL CHEST COMPRESSIONS:

- a. ADULTS: 100 times/minute, 1.5 to 2 inches, 50% compression duration.
- b. CHILDREN: 100 times/minute, 1 to 1.5 inches, 50% compression duration.
- c. INFANTS: At least 100 times/minute, 0.5 to 1 inch, 50% compression duration.

E. ELECTRICAL THERAPY:

1. DEFIBRILLATION: Place anterior paddle beneath right clavicle at upper sternal edge; place apical paddle to left of nipple with center of paddle at midaxillary line.

- a. ADULTS: 200 J on initial attempt, 200 to 300 J on second attempt, and 360 J on third and subsequent attempts for ventricular fibrillation and pulseless ventricular tachycardia.
- b. CHILDREN: 2 J/kg, 2 to 4 J/kg, 4 J/kg.

2. TRANSCUTANEOUS PACING: Initial pacing method of choice; consider use at an early point in the process.

- a. Indicated for all adults with symptomatic bradycardia; used in conjunction with pharmacologic therapy.
- b. Not recommended routinely for bradyasystolic arrest; if used at all, it should be used as soon as possible after onset of arrest.

F. CEREBRAL RESUSCITATION: Current recommendations include the standard methods of CPR to quickly establish return of carotid blood flow and cerebral perfusion and to correct hypoxemia and acid-base imbalance.

G. VASOPRESSORS:

1. VASOPRESSIN:

- a. INDICATIONS: An alternative to epinephrine for treatment of ventricular fibrillation or pulseless ventricular tachycardia refractory to initial shocks.
- b. DOSAGE (ADULTS): 40 U IV as single, one-time dose.

2. EPINEPHRINE:

- a. INDICATIONS: Treatment of ventricular fibrillation or pulseless ventricular tachycardia refractory to initial shocks. May be considered in asystole and pulseless electrical activity.
- b. DOSAGE:
 - (1) ADULTS: 1 mg (10 mL of 1:10,000 soln) IV; may repeat Q3-5MIN.
 - (2) CHILDREN: STANDARD-DOSE: 0.01 mg/kg (0.1 mL/kg of 1:10,000 soln) IV or IO (ET dose: 0.1 mg/kg (0.1 mL/kg of 1:1000 soln)). May repeat in 3 to 5 min at standard-dose or high-dose. HIGH-DOSE: 0.1 mg/kg (0.1 mL/kg of 1:1000 soln) IV, IO, or ET; may repeat Q3-5MIN, as needed.

H. ANTIARRHYTHMICS:

1. AMIODARONE:

- a. INDICATIONS: Shock-refractory ventricular fibrillation or pulseless ventricular tachycardia.
- b. DOSAGE:
 - (1) ADULTS: 300 mg IV push. If VT/VF recurs, consider second dose of 150 mg IV; max dose 2.2 g/day.
 - (2) CHILDREN: See PHARMACOLOGIC TREATMENT for pediatric recommendation.

2. PROCAINAMIDE:

- a. INDICATIONS: Intermittent/recurrent ventricular fibrillation or pulseless ventricular tachycardia.
- b. DOSAGE (ADULTS): 30 mg/min IV infusion until arrhythmia is suppressed, QRS prolongs by 50%, or a maximum of 17 mg/kg is given.

3. MAGNESIUM:

- a. INDICATIONS: Torsades de pointes; suspected hypomagnesemic state
- b. DOSAGE (ADULTS): 1 to 2 g IV over 1 to 2 min.

I. ATROPINE:

1. INDICATIONS: Symptomatic sinus bradycardia, asystole, or pulseless electrical activity.
2. DOSAGE (ASYSTOLE):

- a. ADULTS: 1 mg IV; may repeat Q3-5MIN to total dose of 0.04 mg/kg.
 - b. CHILDREN: 0.02 mg/kg (min dose: 0.1 mg) IV or IO (max single dose: child, 0.5 mg; adolescent, 1 mg); may repeat once in 5 min. Max total dose: child, 1 mg; adolescent, 2 mg.
3. DOSAGE (BRADYARRHYTHMIAS):
- a. ADULTS: 0.5 to 1 mg IV Q3-5MIN until adequate response or until 0.04 milligram/kilogram has been given.
 - b. CHILDREN: child, 0.5 mg; adolescent, 1 mg. May be repeated once.
4. DOSAGE (PULSELESS ELECTRICAL ACTIVITY): ADULTS: If PEA rate is slow, give 1 mg IV; may repeat Q3-5MIN up to a total dose of 0.04 mg/kg.

J. CALCIUM CHLORIDE:

Of limited use in setting of cardiac arrest. Indications include hyperkalemia, hypocalcemia, calcium antagonist toxicity.

K. SODIUM BICARBONATE:

Indications for use during cardiac arrest are extremely limited. May be considered under following conditions: known preexisting hyperkalemia; tricyclic antidepressant overdose; patient is intubated and the arrest is prolonged; upon return of spontaneous circulation after prolonged arrest.

0.2 CLINICAL PRESENTATION

A. The patient may be pale, cyanotic, or have a mottled appearance. There will be no evidence of respirations, or agonal respirations may be present. Pulses will be absent.

B. Other clinical signs and symptoms will depend on the underlying cause of the cardiopulmonary arrest, eg, abdominal distention with intraabdominal bleeding, abnormal temperature with hypothermia or hyperthermia, or deviated trachea and absent breath sounds with tension pneumothorax.

0.3 DIAGNOSTICS

A. LABORATORY:

1. The rapidity with which available laboratory procedures can be obtained is an important determinant of their use.
2. END-TIDAL CO₂ (ETCO₂): Detection of significant ETCO₂ (indicated by color change on colorimetric ETCO₂ detector) correlates well with successful endotracheal intubation; failure to detect ETCO₂ may indicate esophageal intubation. Use of ETCO₂ detectors has been endorsed by ACEP.
3. Monitoring of ABGs is essential.
4. Other lab data that may be beneficial, eg, potassium levels, carboxyhemoglobin level or toxicology screens, depend on the clinical situation.
5. Fingerstick glucose determinations may be inaccurate in the setting of shock.

B. RADIOLOGY:

1. CHEST FILM: Should be obtained after endotracheal tube placement. Correct placement should be determined clinically with or without ETCO₂ measurement.

0.4 DIFFERENTIAL DIAGNOSIS

A. Although the entity of cardiopulmonary arrest is not difficult to recognize, the underlying cause poses significant problems in many situations.

1. Pediatric arrest is usually a respiratory arrest followed by cardiac arrest.
2. Other clues that may indicate the cause of the cardiopulmonary arrest include:
 - a. Absence of pulses with chest compression (may indicate hypovolemia secondary to dehydration or hemorrhage, tension pneumothorax, or cardiac tamponade).
 - b. Recurrent or recalcitrant ventricular fibrillation (may be due to an underlying metabolic problem, especially potassium disorders and hypoxia).
 - c. Inability to ventilate (may indicate an airway foreign body).

1.0 CLINICAL PRESENTATION

1.1 INTRODUCTION

1.1.1 ETIOLOGY

A. PROGNOSIS:

1. GENERAL:

- a. Sudden cardiac arrest is major cause of death in developing countries; despite years of experience with CPR, postarrest survival continues to be low (Ballew, 1998).
- b. A cumulative meta-analysis indicates survival to hospital discharge is 6%, regardless of initial rhythm (Nichol, 1999).

2. IMPROVED OUTCOME:

a. GENERAL:

- (1) In a study of 1029 cardiac arrests, survival was higher when the collapse occurred outside the home, probably because of availability of witnesses, age, comorbidity, and underlying etiology (Litwin, 1987).
- (2) Factors associated with greater likelihood of survival following out-of-hospital arrest include early CPR and VF or VT as arrest rhythm (Rainer, 1997; Weston, 1997; Giraud, 1996; Gaul, 1996). The time to defibrillation is the major determinant of survival in cardiac arrest secondary to VF (AHA/ILCOR Guidelines, 2000; Stiell, 1999; Nichol, 1999; Heavens, 1998; White, 1998).
- (3) Successful outcome of inhospital CPR was related to arrest within 24 hours of hospitalization, short duration of CPR, and the absence of cardiogenic shock, sepsis, acute renal failure, cancer, and pneumonia (Rozenbaum, 1988).
- (4) Advanced life support (ALS) training was associated with improved survival in a study of 12,417 trauma patients (Messick, 1992).

- b. CHILDREN: Factors that positively influenced outcome included CPR duration (<5 minutes), number of epinephrine doses, and location of arrest. Age, sex, cardiac rhythm, and acid-base variables did not significantly affect outcome (Barzilay, 1988).

3. POOR OUTCOME:

a. ADULTS:

- (1) Of 249 victims of out-of-hospital cardiac arrest, only about 10% of patients surviving to hospital admission survived to hospital discharge. Bystander CPR had little impact on survival, and age/gender had no predictive powers. There were few or no survivors with arrest rhythms of primary asystole, severe bradycardia, or PEA (Gaul, 1996). Other studies have noted improved survival with bystander CPR (Giraud, 1996; Weston, 1997; Ballew, 1997).
- (2) Status epilepticus was predictive of poor outcome (survival and recovery of consciousness) in comatose adult survivors of CPR (Krumholz, 1988).
- (3) In persons with implantable cardioverter-defibrillators, sudden death is characterized by poor LV function and hemodynamically unstable ventricular tachyarrhythmias (major cause) (Li, 1996).
- (4) Serum neuron-specific enolase levels >33 mg/mL are an early predictor of poor neurologic outcome in survivors of cardiac arrest (Fogel, 1997).

- b. ELDERLY: Inpatient resuscitation of geriatric patients predictive of poor outcome was related to witnessed arrest, short duration, presence of sepsis, cancer, increased age, increased number of medication doses, asystole, or pulseless electrical activity (Taffet, 1988; Murphy, 1989).

c. CHILDREN:

- (1) The outcome for cardiac arrest is much poorer than for respiratory arrest (Zaritsky, 1987; Schindler, 1996). Following out-of-hospital arrest, overall mortality is 90% to 95%; for inhospital arrest, mortality is 85% to 90% (Zaritsky, 1998).
- (2) In a study of 101 pediatric arrests, survival to hospital discharge was associated with presence of a palpable pulse on presentation, short transport time, short duration of resuscitation, and few doses of epinephrine; no patients who required >20 minutes of resuscitation or >2 doses of epinephrine survived (Schindler, 1996).

4. SHORT- & LONG-TERM SURVIVAL: In one study, 46% patients initially resuscitated from out-of-hospital cardiac arrest survived to hospital discharge. Of this 46%, neurologic status was normal or mildly impaired in 89% and 91% could be discharged directly to home; estimated four-year survival was 68%. Factors predicting mortality on follow-up were increased age, heart failure, and cardiac arrest not due to definite myocardial infarction (Cobbe, 1996).

1.5 COMPLICATIONS

A. RUPTURE, GASTRIC

1. Has been reported as a complication of successful CPR (Reiger, 1997).

B. RUPTURE, SPLEEN

1. Has been reported as a complication of CPR (Stallard, 1997).

C. INFARCTION, MYOCARDIAL

1. Troponin T helpful in evaluating for AMI in patients following DC countershock. One study found troponin T levels were not significantly elevated in patients following cardioversion; however, both total CK and CK-MB were significantly elevated (Grubb, 1998).
2. CK-MB not useful in evaluating for AMI in patients following DC countershock. One study found both total CK and CK-MB were significantly elevated in patients following elective cardioversion. Strong correlation seen between CK-MB and total shock energy used (Grubb, 1998).

D. TRAUMA, IATROGENIC

1. Cardiac injury may be caused by myocardial damage related to CPR efforts (Mullner, 1996).
2. Iatrogenic injuries are rare in children receiving CPR (3% in one series), despite prolonged resuscitation performed with variable degrees of skill. Therefore, abuse should be considered whenever traumatic injuries are encountered (Bush, 1996).
3. US is readily available, noninvasive means of assessing traumatic complications of CPR in nontraumatic cardiac arrest patients. May identify injuries, help guide procedures, and serve as means to assess pharmacologic effects on cardiac performance during CPR (Corbett, 1997).

E. NEUROLOGIC SEQUELA

1. PROGNOSTIC INDICATORS:

a. CLINICAL FINDINGS:

- (1) Following survival of cardiac arrest, the presence of reactive pupils, oculocephalic reflexes, spontaneous respirations, and purposeful response to painful stimuli is associated with a much higher likelihood of neurologic recovery. However, initial neurologic examination does not exclude high quality neurologic survival among exceptional patients with many unfavorable signs (Earnest, 1979; Steen-Hansen, 1988).
- (2) Return of respiratory movements, pupillary response to light, coughing and/or swallowing, and ciliospinal reflexes within 1 h following the return of cerebral circulation have been correlated with improved cerebral recovery (Jorgensen, 1979).
- (3) Long-term survival and neurologic recovery following cardiac arrest followed by CPR associated with patient being awake on admission, or awakening to follow simple commands within 2 d following cardiac arrest, and with good neurologic status at the time of discharge from the hospital (Earnest, 1980).

b. LABORATORY FINDINGS:

- (1) Serum neuron-specific enolase (NSE) levels may be useful in evaluating neurologic outcome following return of spontaneous circulation (Schoerhuber, 1999; Fogel, 1997). Serum NSE levels >33 mg/mL are an early predictor of poor neurologic outcome in survivors of cardiac arrest (Fogel, 1997).
- (2) CSF enzyme markers of damaged cerebral cells (eg, LDH, AST, CPK, CPK isoenzymes, lactate, pyruvate) measured by 24 to 48 h after recovery from cardiac arrest may be useful as further prognostic signs of future cerebral function and brain viability (Mullie, 1981; Vaagenes, 1980).
- (3) Serum levels of protein S-100, a neurochemical marker, may be valuable in determining extent of hypoxic neuronal damage following cardiopulmonary arrest (Rosen, 1998; Astudillo, 1996).

2.0 LABORATORY DATA

2.1 GENERAL DISCUSSION

A. The rapidity with which available laboratory procedures can be obtained is an important determinant of their use. Monitoring of ABGs is essential. Other lab data that may be beneficial, eg, potassium levels, carboxyhemoglobin level or toxicology screens, depend on the clinical situation.

2.3 ELECTROLYTES

A. POTASSIUM

1. May be beneficial, depending on the clinical situation.

B. POTASSIUM, INCREASED

1. A study of 22 patients during closed-chest cardiopulmonary resuscitation (CC-CPR) revealed that significant hyperkalemia occurs in some patients during CC-CPR and may play a role in wide-complex pulseless electrical activity (PEA). The success of calcium chloride in treating wide-complex PEA may result from this phenomenon (Martin, 1989).

2.4 CHEMICAL SURVEY

A. CARDIAC ENZYMES

1. ISOENZYME, CK-MB

- a. CK-MB not useful in evaluating for AMI in patients following DC countershock. One study found both total CK and CK-MB were significantly elevated in patients following elective cardioversion. Strong correlation seen between CK-MB and total shock energy used. (Grubb, 1998).
- b. Another study found similar results. CK-MB elevations were associated with duration of CPR, cardiogenic shock, and AMI; however, troponin T elevations were only associated with AMI (Mullner, 1998).

2. TROPONIN T

- a. Troponin T helpful in evaluating for AMI in patients following DC countershock. One study found troponin T levels were not significantly elevated in patients following cardioversion; however, both total CK and CK-MB were significantly elevated (Grubb, 1998).
- b. Another study found similar results. CK-MB elevations were associated with duration of CPR, cardiogenic shock, and AMI; however, troponin T elevations were only associated with AMI (Mullner, 1998).

B. PROTEIN, S-100

1. A neurochemical marker that may be valuable in determining extent of hypoxic neuronal damage following cardiopulmonary arrest (Rosen, 1998; Astudillo, 1996). One preliminary study found patients with serum S-100 levels greater than or equal to 0.2 mcg/L had 100% mortality within 14 days postarrest. Nearly 90% of patients with levels less than 0.2 mcg/L survived 14 days postarrest (Rosen, 1998).

2.6 ARTERIAL BLOOD GASES

A. OVERVIEW

1. Essential in management of cardiac arrest victims.

B. ACIDOSIS, METABOLIC

1. Profound metabolic acidosis in association with cardiac arrest has been reported following restraint of severely agitated patients. Extremely combative behavior during prone-position restraint and concomitant cocaine intoxication led to dramatic lactic acidosis and cardiopulmonary arrest (Hick, 1999).

2.9 MISCELLANEOUS

A. CARBOXYHEMOGLOBIN

1. May be beneficial, depending on the clinical situation.

B. TOXICOLOGY SCREEN

1. May be beneficial, depending on the clinical situation.

C. NEURON-SPECIFIC ENOLASE

1. May be useful in evaluating neurologic outcome following return of spontaneous circulation. One small, preliminary study found measurement of neuron-specific enolase (NSE) at 72 hours following successful resuscitation is predictive of neurologic outcome. Patients with bad neurologic outcomes (cerebral performance category (CPC) 3 or 4) had significantly higher NSE compared with patients with good neurologic outcome (CPC 1 or 2) (Schoerhuber, 1999).
2. Serum NSE levels >33 mg/mL are an early predictor of poor neurologic outcome in survivors of cardiac arrest (Fogel, 1997).

3.0 RADIOLOGIC DATA

3.2 PLAIN FILMS

A. RADIOGRAPHY, CHEST

1. Should be obtained to verify endotracheal tube placement. Placement should also be verified clinically, with or without end-tidal CO₂ measurement.

3.3 CONTRAST STUDIES

A. ARTERIOGRAPHY, CORONARY

1. In survivors of out-of-hospital cardiac arrest who have no obvious noncardiac cause of the arrest, immediate coronary angiography with angioplasty is safe and feasible when performed by an experienced team and may improve long-term outcome (Spaulding, 1997).

3.6 ULTRASOUND

A. OVERVIEW

1. Readily available, noninvasive means of assessing traumatic complications of CPR in nontraumatic cardiac arrest patients. May identify injuries, help guide procedures, and serve as means to assess pharmacologic effects on cardiac performance during CPR (Corbett, 1997).
2. Compared with standard landmark-oriented approach for obtaining femoral vein catheterization in patients requiring IV access during CPR, real-time US-guidance is faster and provides lower rate of inadvertent arterial catheterization and higher rate of success (Hilty, 1997).

B. ECHOCARDIOGRAPHY, TRANSESOPHAGEAL

1. TEE was studied in 48 patients undergoing CPR and was found useful in identifying the etiology of cardiac arrest. Diagnoses included cardiac tamponade, myocardial infarction, pulmonary embolism, aortic dissection, aortic rupture, and others (van der Wouw, 1997).

C. ECHOCARDIOGRAPHY

1. Helpful in diagnosing pulmonary embolism as cause of cardiac arrest in conjunction with history and clinical findings (Kurkciyan, 2000; Bottiger, 1994).

4.0 DIAGNOSTIC AIDS

4.2 MISCELLANEOUS

A. CARBON DIOXIDE, END-TIDAL

1. GENERAL:

- a. Capnometry (measurement of exhaled end-tidal carbon dioxide (ETCO₂)) reflects ability of circulation to deliver CO₂ to the lungs (Berg, 1996).
- b. Use of ETCO₂ detectors has been endorsed by ACEP (ACEP, 1995).

2. INDICATIONS:

- a. Class IIa recommendation (acceptable, probably effective). Presence of exhaled CO₂ indicates proper ET tube placement. Continuous ETCO₂ devices can confirm ET tube placement within seconds of intubation attempt and can detect dislodgment of ET tube. If no exhaled CO₂ is detected (during cardiac arrest), a second technique should be used; Class IIb recommendation (acceptable, possibly effective) (AHA/ILCOR Guidelines, 2000).
- b. ETCO₂ is useful in monitoring effectiveness of CPR after 1 min of resuscitation (Ward, 1998; Berg, 1996). Correlates with cardiac output generated during CPR if minute ventilations are held fairly constant (Ward, 1998; Dickinson, 1999).
- c. May be helpful in predicting return of spontaneous circulation (Ward, 1998; Mauer, 1998).

3. FINDINGS:

- a. In patients in full arrest, ETCO₂ levels often are 1 to 5 mmHg, compared with 40 mmHg in patients with normal circulation. Rising CO₂ levels indicate more successful compressions; when rhythm is restored, levels rise to 15- to 25-mmHg range (Ward, 1998; Falk, 1988; Callahan, 1989).
- b. Animal studies indicate that initial ETCO₂ is markedly elevated during CPR after asphyxial cardiac arrest, with much higher levels than after VF cardiac arrest; in each case, initial ETCO₂ appears to reflect alveolar CO₂ prior to CPR (Berg, 1996).
- c. A 150-patient, prospective study of out-of-hospital cardiac arrest demonstrated that an ETCO₂ less than or equal to 10 mmHg measured 20 minutes after the initiation of ACLS accurately predicts death. The authors suggest CPR may reasonably be terminated in such patients (Levine, 1997). Other investigators have observed similar results (Wane, 1995; Asplin, 1995).
- d. Administration of sodium bicarbonate during resuscitation causes a significant, but transient, increase in ETCO₂. Increases noted within 1 minute of administration and return to baseline within 2 minutes of dose (Ward, 1998).

6.0 TREATMENT

6.1 TREATMENT SUMMARY

- A. GENERAL: Any patient with absent spontaneous respirations and pulses has sustained a cardiopulmonary arrest.
 1. Basic life support must be initiated promptly, followed by advanced cardiac life support (ACLS), which includes airway management and correction of circulatory compromise. Evaluation for causative factors critical.
 2. Arrhythmia management is a key to successful resuscitation, utilizing rapid defibrillation (most critical step in cardiac arrest due to ventricular fibrillation) and pharmacologic therapy.
 3. Continuous monitoring of pH, PCO₂, PO₂, and rhythm is essential.

B. IV LINES: Large-bore IV access must be available for fluid resuscitation and medication delivery; alternative routes include endotracheal administration and intraosseous infusion (children).

C. AIRWAY MANAGEMENT:

1. ESTABLISH OPEN AIRWAY: Tongue is most common cause of airway obstruction in the unconscious victim.
 - a. HEAD TILT-CHIN LIFT MANEUVER: Method of choice for opening the airway.
 - b. JAW-THRUST MANEUVER: Safest approach to opening the airway of a victim with suspected neck injury; technically difficult and fatiguing.
 - c. ENDOTRACHEAL INTUBATION:
 - (1) Should be done as soon as possible in the course of resuscitation by an experienced healthcare provider. Once inserted, ET tube should be secured and placement verified with primary (physical exam) and secondary (nonphysical exam, eg, end-tidal CO₂ detector) techniques.
 - (2) If ET has been placed and there is a delay in obtaining venous access, several medications may be administered via the endotracheal route, including epinephrine, atropine, and lidocaine.
2. BREATHING:
 - a. RESCUE BREATHING:
 - (1) WITHOUT OXYGEN: Give 2 breaths slowly over 2 seconds initially, followed by 10 to 12 breaths per minute (1 breath every 4 to 5 seconds). A volume of 700 to 1000 mL (approx 10 mL/kg) is sufficient for most adults. Because of the wide variation in the size of children, precise volumes cannot be recommended. Experts state the correct volume for each breath is that which causes the chest to rise.
 - (2) WITH OXYGEN ($\geq 40\%$): Smaller tidal volume of 400 to 600 mL (6 to 7 mL/kg) delivered over 1 to 2 seconds, followed by 10 to 12 breaths per minute (1 breath every 4 to 5 seconds).
 - b. CHEST COMPRESSION TO VENTILATION RATIO:
 - (1) ADULTS AND CHILDREN 8 YR AND OLDER: For patients with unprotected airway (not intubated), ratio is 15:2 for both one- and two-rescuer CPR. Once airway is secured, continuous compressions and asynchronous ventilations with ratio of 5:1 is recommended.
 - (2) CHILDREN UNDER 8 YR: 5 compressions to 1 ventilation for one- and two-rescuer CPR.
 - c. CHEST COMPRESSION-ONLY CPR: Recommended for use when the rescuer is unwilling or unable to perform mouth-to-mouth rescue breathing or in dispatch-assisted CPR.
3. OXYGEN: Administer 100% O₂ via bag-valve-mask or mouth-to-mask, or via endotracheal tube.

D. CIRCULATION:

1. DETERMINE PULSELESSNESS:
 - a. LAY RESCUERS: Assess for absence of signs of circulation (normal breathing, coughing, or movement). Pulse check no longer recommended for lay rescuers.
 - b. HEALTHCARE PROVIDERS: For children ≥ 1 yr old and adults, use carotid artery. For children < 1 year of age, brachial artery is recommended.
2. EXTERNAL CHEST COMPRESSIONS:
 - a. ADULTS: 100 times/minute, 1.5 to 2 inches, 50% compression duration.
 - b. CHILDREN: 100 times/minute, 1 to 1.5 inches, 50% compression duration.
 - c. INFANTS: At least 100 times/minute, 0.5 to 1 inch, 50% compression duration.

E. ELECTRICAL THERAPY:

1. DEFIBRILLATION: Place anterior paddle beneath right clavicle at upper sternal edge; place apical paddle to left of nipple with center of paddle at midaxillary line.
 - a. ADULTS: 200 J on initial attempt, 200 to 300 J on second attempt, and 360 J on third and subsequent attempts for ventricular fibrillation and pulseless ventricular tachycardia.
 - b. CHILDREN: 2 J/kg, 2 to 4 J/kg, 4 J/kg.
2. TRANSCUTANEOUS PACING: Initial pacing method of choice; consider use at an early point in the process.
 - a. Indicated for all adults with symptomatic bradycardia; used in conjunction with pharmacologic therapy.
 - b. Not recommended routinely for bradyasystolic arrest; if used at all, it should be used as soon as possible after onset of arrest.

F. CEREBRAL RESUSCITATION: Current recommendations include the standard methods of CPR to quickly establish return of carotid blood flow and cerebral perfusion and to correct hypoxemia and acid-base imbalance.

G. VASOPRESSORS:

1. VASOPRESSIN:

- a. INDICATIONS: An alternative to epinephrine for treatment of ventricular fibrillation or pulseless ventricular tachycardia refractory to initial shocks.
- b. DOSAGE (ADULTS): 40 U IV as single, one-time dose.

2. EPINEPHRINE:

- a. INDICATIONS: Treatment of ventricular fibrillation or pulseless ventricular tachycardia refractory to initial shocks. May be considered in asystole and pulseless electrical activity.
- b. DOSAGE:
 - (1) ADULTS: 1 mg (10 mL of 1:10,000 soln) IV; may repeat Q3-5MIN.
 - (2) CHILDREN: STANDARD-DOSE: 0.01 mg/kg (0.1 mL/kg of 1:10,000 soln) IV or IO (ET dose: 0.1 mg/kg (0.1 mL/kg of 1:1000 soln)). May repeat in 3 to 5 min at standard dose or high-dose. HIGH-DOSE: 0.1 mg/kg (0.1 mL/kg of 1:1000 soln) IV, IO, or ET; may repeat Q3-5MIN, as needed.

H. ANTIARRHYTHMICS:

1. AMIODARONE:

- a. INDICATIONS: Shock-refractory ventricular fibrillation or pulseless ventricular tachycardia.
- b. DOSAGE:
 - (1) ADULTS: 300 mg IV push. If VT/VF recurs, consider second dose of 150 mg IV; max dose 2.2 g/day.
 - (2) CHILDREN: See PHARMACOLOGIC TREATMENT for pediatric recommendation.

2. PROCAINAMIDE:

- a. INDICATIONS: Intermittent/recurrent ventricular fibrillation or pulseless ventricular tachycardia.
- b. DOSAGE (ADULTS): 30 mg/min IV infusion until arrhythmia is suppressed, QRS prolongs by 50%, or a maximum of 17 mg/kg is given.

3. MAGNESIUM:

- a. INDICATIONS: Torsades de pointes; suspected hypomagnesemic state
- b. DOSAGE (ADULTS): 1 to 2 g IV over 1 to 2 min.

I. ATROPINE:

1. INDICATIONS: Symptomatic sinus bradycardia, asystole, or pulseless electrical activity.
2. DOSAGE (ASYSTOLE):
 - a. ADULTS: 1 mg IV; may repeat Q3-5MIN to total dose of 0.04 mg/kg.
 - b. CHILDREN: 0.02 mg/kg (min dose: 0.1 mg) IV or IO (max single dose: child, 0.5 mg; adolescent, 1 mg); may repeat once in 5 min. Max total dose: child, 1 mg; adolescent, 2 mg.
3. DOSAGE (BRADYARRHYTHMIAS):
 - a. ADULTS: 0.5 to 1 mg IV Q3-5MIN until adequate response or until 0.04 milligram/kilogram has been given.
 - b. CHILDREN: 0.02 mg/kg (min dose: 0.1 mg) IV or IO; max single dose: child, 0.5 mg; adolescent, 1 mg. May be repeated once.
4. DOSAGE (PULSELESS ELECTRICAL ACTIVITY) (ADULTS): If PEA rate is slow, give 1 mg IV; may repeat Q3-5MIN up to a total dose of 0.04 mg/kg.

J. CALCIUM CHLORIDE: Of limited use in setting of cardiac arrest. Indications include hyperkalemia, hypocalcemia, calcium antagonist toxicity.

K. SODIUM BICARBONATE: Indications for use during cardiac arrest are extremely limited. May be considered under following conditions: known preexisting hyperkalemia; tricyclic antidepressant overdose; patient is intubated and the arrest is prolonged; upon return of spontaneous circulation after prolonged arrest.

6.2 NON-PHARMACOLOGIC TREATMENT

A. AIRWAY MANAGEMENT

1. OVERVIEW

a. NEED FOR VENTILATION:

- (1) Debate continues about need for ventilation in first few minutes of CPR (Hallstrom, 2000; Ewy, 2000; Becker, 1997, per recommendations of the Ventilation Working Group). If the arrest is prolonged, ventilation does improve the rate of return of spontaneous circulation (Safar, 1998; Braslow, 1997; Gazmuri, 1999; Idris, 1996).
- (2) Chest compression-only CPR is recommended for use when the rescuer is unwilling or unable to perform mouth-to-mouth rescue breathing or in dispatch-assisted CPR; Class IIa recommendation (acceptable, probably effective) (AHA/ILCOR Guidelines, 2000).

b. RATE:

- (1) ADULTS: Recommendation for both one- and two-rescuer CPR is 15 compressions to 2 ventilations for patients with unprotected airway (not intubated); Class IIb recommendation (acceptable, possibly effective). Once airway is secured, continuous compressions and asynchronous ventilations with ratio of 5:1 is recommended (AHA/ILCOR Guidelines, 2000).
- (2) INFANTS AND CHILDREN: Ratio of 1 ventilation for every 5 chest compressions is recommended for both one- and two-rescuer CPR (AHA/ILCOR Guidelines, 2000).

2. VENTILATION, BAG-VALVE-MASK

a. RECOMMENDATION:

- (1) Administration of 100% oxygen via mouth-to-mask or bag-valve-mask at a ratio of 2 ventilations to 15 compressions is recommended for patients with unprotected airway (not intubated); Class IIb recommendation (acceptable, possibly effective). Once airway is secured, continuous compressions and asynchronous ventilations with ratio of 1 ventilation to 5 compressions is recommended (AHA/ILCOR Guidelines, 2000).
- (2) When bag-valve-mask ventilations are ineffective due to facial trauma, and tracheal intubation is unavailable, difficult, or contraindicated, a rapid percutaneous approach to the trachea must be employed.

b. CONTROVERSY:

- (1) Debate continues about need for ventilation in first few minutes of CPR (Hallstrom, 2000; Ewy, 2000; Becker, 1997, per recommendations of the Ventilation Working Group). If the arrest is prolonged, ventilation does improve the rate of return of spontaneous circulation (Safar, 1998; Braslow, 1997; Gazmuri, 1999; Idris, 1996).
- (2) A randomized trial, involving 241 patients with witnessed out-of-hospital cardiac arrest, found patients treated with bystander-initiated chest compressions alone had similar outcomes to those treated with bystander-initiated chest compressions and mouth-to-mouth ventilation. Survival to hospital discharge was higher in patients treated with chest compressions alone, although difference was not significant (Hallstrom, 2000).

c. VENTILATORY VOLUMES (AHA/ILCOR Guidelines, 2000):

- (1) WITHOUT OXYGEN: Give 2 breaths slowly over 2 seconds initially, followed by 10 to 12 breaths per minute (1 breath every 4 to 5 seconds). A volume of 700 to 1000 mL (approx 10 mL/kg) is sufficient for most adults; Class IIa recommendation (acceptable, probably effective). Because of the wide variation in the size of children, precise volumes cannot be recommended. Experts state the correct volume for each breath is that which causes the chest to rise.
- (2) WITH OXYGEN ($\geq 40\%$): Smaller tidal volume of 400 to 600 mL (6 to 7 mL/kg) delivered over 1 to 2 seconds, followed by 10 to 12 breaths per minute (1 breath every 4 to 5 seconds); Class IIb recommendation (acceptable, possibly effective).

d. COMPLICATIONS:

- (1) One study involving over 700 patients with in-hospital cardiopulmonary arrest found initial use of bag-valve-mask resulted in regurgitation in over 12%; initial use of laryngeal mask airway resulted in regurgitation in only 4% (Stone, 1998).
- (2) Ventilation with bag-valve-mask resulted in significantly more gastric inflation compared with laryngeal mask airway or Combitube(R) (Doerges, 1999).

3. VENTILATION, MOUTH-TO-MASK

a. GENERAL (AHA/ILCOR Guidelines, 2000):

- (1) A well-fitting mask is an effective adjunct for use in artificial ventilation. This technique is easier to use and produces larger tidal volumes than the bag-valve-mask technique because both hands are used to maintain airway patency and a good mask fit.
- (2) Mouth-to-mask ventilation with supplemental oxygen should be used until an endotracheal tube or esophageal airway is in place unless personnel are extensively trained in the use of bag-valve-mask devices.

b. RECOMMENDATION: Administration of 100% oxygen via mouth-to-mask or bag-valve-mask at a ratio of 2 ventilations to 15 compressions is recommended for patients with unprotected airway (not intubated); Class IIb recommendation (acceptable, possibly effective). Once airway is secured, continuous compressions and asynchronous ventilations with ratio of 1 ventilation to 5 compressions is recommended (AHA/ILCOR Guidelines, 2000).

c. CONTROVERSY:

- (1) Debate continues about need for ventilation in first few minutes of CPR (Hallstrom, 2000; Ewy, 2000; Becker, 1997, per recommendations of the Ventilation Working Group). If the arrest is prolonged, ventilation does improve the rate of return of spontaneous circulation (Safar, 1998; Braslow, 1997; Gazmuri, 1999; Idris, 1996).

(2) A randomized trial, involving 241 patients with witnessed out-of-hospital cardiac arrest, found patients treated with bystander-initiated chest compressions alone had similar outcomes to those treated with bystander-initiated chest compressions and mouth-to-mouth ventilation. Survival to hospital discharge was higher in patients treated with chest compressions alone, although difference was not significant (Hallstrom, 2000).

4. INTUBATION, ENDOTRACHEAL

a. GENERAL: Current recommendations for artificial ventilation are administration of 100% oxygen via mouth-to-mask, bag-valve-mask, or endotracheal tube. Ventilations provided via endotracheal tube do not need to be synchronized with chest compressions; asynchronous ventilations at rate of 10 to 12 per minute with 100% oxygen should be performed (AHA/ILCOR Guidelines, 2000).

b. INDICATIONS: Cardiac arrest not responsive to early defibrillation.

c. ADULT TUBE SIZE: 7.0- to 8.0-mm diameter ET tubes are most commonly used for adult females and males. The ET tube should have a high volume/low pressure cuff (AHA/ILCOR Guidelines, 2000).

d. PEDIATRIC TUBE SIZE (AHA/ILCOR Guidelines, 2000):

(1) Tube size can be estimated using the following formula: age (in years) divided by 4, plus 4; or, a tube of the same diameter as the child's small finger can be used. A 3-mm tube is the smallest size tube allowing tracheal suction.

(2) An uncuffed tube should be used in children below the age of seven or eight years. Cuffed tubes are used in older children; a cuffed tube must be treated as if it were 0.5 mm larger (eg, 6.0 mm cuffed = 6.5 mm uncuffed).

(3) The following guidelines may be helpful in estimating the correct tube size:

AGE	ENDOTRACHEAL TUBE SIZE (mm)	SUCTION CATHETER SIZE (F)
Preterm infant	2.5 to 3.5 uncuffed	5 to 8
0 mo to 1 yr	3.5 to 4.0 uncuffed	8
1 yr/small child	4.0 uncuffed	8
3 yr/child	4.5 uncuffed	8 or 10
5 yr/child	5.0 uncuffed	10
6 yr/child	5.5 uncuffed	10
8 yr/child to small adult	6.0 cuffed	10 or 12
12 yr/adolescent	6.5 cuffed	12
16 yr/adult	7.0 cuffed	12

e. NASOTRACHEAL TUBE: Easier to secure, more comfortable, and less traumatizing to the vocal cords. The cuff should be inflated to a pressure less than 25 mmHg and should allow a small air leak during peak inspiration. The cuff should not be deflated periodically as this leaves the airway unprotected and permits aspiration.

f. TUBE PLACEMENT: After intubation, it is mandatory to verify tube placement using both primary and secondary techniques (AHA/ILCOR Guidelines, 2000):

(1) PRIMARY TECHNIQUES: Involve physical examination:

(a) Assess first breath delivered via bag-valve device. Auscultate over epigastrium and observe the chest wall for movement during ventilation. If stomach gurgling is heard and chest wall does not move, the esophagus has been intubated. Remove ET tube and reattempt intubation.

(b) Auscultate chest to verify equal breath sounds over lateral chest; helps identify ET tube in right main stem bronchus.

(2) SECONDARY TECHNIQUES: Include nonphysical examination techniques using electronic or mechanical devices:

(a) Esophageal detector device creates suction at the tracheal end of the ET tube by use of syringe or bulb. If ET tube is in the esophagus, the syringe or bulb will fail to reexpand.

(b) End-tidal CO₂ detector is valuable in confirming ET tube placement (Hayden, 1995; Bhende, 1995); Class IIa recommendation (acceptable, probably effective). Presence of exhaled CO₂ indicates proper ET tube placement. Continuous ETCO₂ devices can confirm ET tube placement within seconds of intubation attempt and can detect dislodgment of ET tube. If no exhaled CO₂ is detected (during cardiac arrest), a second technique should be used; Class IIb recommendation (acceptable, possibly effective).

(3) FOLLOW-UP: Once ET placement has been verified and secured with purpose-built commercial device, obtain a chest x-ray; ET tube tip should be 1 to 2 cm above carina. Insert bite block or oropharyngeal airway to prevent patient from biting down and occluding airway (AHA/ILCOR Guidelines, 2000).

g. LIGHTED STYLET ENDOTRACHEAL INTUBATION:

(1) DESCRIPTION: The lighted stylet is a light bulb affixed to a flexible shaft that is inserted through an endotracheal tube, in the same manner as a traditional stylet. When the trachea has been intubated, the anterior neck is illuminated; absence of illumination indicates esophageal intubation (Reed, 1995).

(2) ADVANTAGES: There is no need for a laryngoscope. The presence of secretions or blood in the airway does not impede intubation.

(3) DISADVANTAGES: There is no direct visualization of the vocal cords, which is the most reliable indicator of tracheal intubation.

5. ESOPHAGEAL-TRACHEAL COMBITUBE

a. DESCRIPTION (AHA/ILCOR Guidelines, 2000):

(1) An invasive double-lumen airway that can ventilate the trachea, either by direct endotracheal placement or via an esophageal obturator mechanism, irrespective of whether the esophagus or trachea is intubated (Reed, 1995; Atherton, 1993).

(2) Inserted without visualization of vocal cords. Oxygenation and ventilation provided via one of two proximal ports, depending on whether tube was placed in trachea or in esophagus.

b. INDICATIONS: Consider for short-term use in prehospital setting when endotracheal intubation is not possible; Class IIb recommendation (acceptable, possibly effective) (AHA/ILCOR Guidelines, 2000).

c. ADVANTAGES:

(1) Isolates airway, reduces risk of aspiration, provides more reliable ventilation than face mask. Also requires less training and maintenance of skills than endotracheal intubation (AHA/ILCOR Guidelines, 2000).

(2) Ventilation with Combitube(R) tube results in significantly less gastric inflation compared with bag-mask ventilation (Doerges, 1999).

d. DISADVANTAGES: May cause esophageal trauma. Incorrect identification of distal tube position, leading to use of incorrect port for ventilation, may lead to fatal complications (AHA/ILCOR Guidelines, 2000).

e. OUTCOME: Successful insertion reported in 70% to 100% of cases (AHA/ILCOR Guidelines, 2000).

6. CRICOTHYROTOMY

a. GENERAL: A rapid percutaneous approach to the trachea; allows temporary ventilation and oxygenation of patient.

b. INDICATIONS:

(1) ADULTS: Should be used when bag-valve-mask ventilations are ineffective due to facial trauma and tracheal intubation is unavailable, difficult, or contraindicated (eg, cervical spine trauma) (Hawkins, 1995).

(2) CHILDREN: Should be considered for children with complete upper airway obstruction when other methods have failed to provide a patent airway. In children <8 years of age, the subglottic cricoid ring (inferior to the cricoid membrane) forms the narrowest segment of the airway, and cricothyrotomy may not be effective.

c. TECHNIQUE: May be performed via percutaneous or surgical technique.

d. COMPLICATIONS: Include bleeding, false passage, esophageal perforation, and subcutaneous or mediastinal emphysema.

e. ADVANTAGES: Generally preferred over tracheotomy and does not have long-term complications when properly employed (Brantigan, 1976).

7. VENTILATION, PERCUTANEOUS TRANSTRACHEAL

a. DEFINITION: Temporary airway management technique that is utilized until a more definitive airway can be secured. High-pressure (50 or 60 psi) oxygen is delivered in intermittent jets, usually at 12 to 20 breaths/minute.

b. GENERAL:

(1) Provides immediate oxygenation and ventilation via a large bore (12- to 16-gauge) IV catheter inserted transtracheally through the cricoid membrane. Use in various clinical settings has demonstrated effective pulmonary gas exchange with minimal complications (Gazmuri, 1999; Depierraz, 1994; Nakatsuka, 1992).

(2) In general, this is a temporary procedure; oxygenation can be maintained for approximately 30 minutes; beyond this point, progressive hypercapnia due to inadequate ventilation occurs (Manning, 1995).

- c. INDICATIONS: Alternative airway management when airway obstruction cannot be relieved by other techniques and when oral or nasal intubation is time-consuming, dangerous, unsuccessful, or contraindicated (Gazmuri, 1999; AHA/ILCOR Guidelines, 2000; Nakatsuda, 1992; Benumof, 1994).
 - d. CONTRAINDICATIONS: Relative coagulopathy; complete airway obstruction, although this is being debated, especially if large (8.5 Fr) catheter is used (Manning, 1995).
 - e. TECHNIQUE (Manning, 1995):
 - (1) Palpate space between inferior surface of the thyroid cartilage and the upper surface of the cricoid; insert tip of 12- to 16-gauge over-the-needle plastic or Teflon IV catheter at a 30- to 45-degree angle caudally through cricoid membrane.
 - (2) Aspirate with syringe during insertion; air return indicates entrance into laryngotracheal lumen.
 - (3) Thread catheter in (to hub of catheter); connect catheter to pressurized (approximately 50 psi) oxygen. If high pressure oxygen not available, may use an oxygen-regulator system at maximum (15 L/min) flow rate (suboptimal but acceptable).
 - (4) Provide 1-second inflations at a rate of approximately 12 per minute.
 - f. COMPLICATIONS: Subcutaneous emphysema; hemorrhage, most often involving the thyroid; aspiration; esophageal perforation with gastric dilation; catheter kinking, and laryngeal pneumatocele.
8. AIRWAY, PHARYNGOTRACHEAL LUMEN
- a. DESCRIPTION: Double-lumen tube inserted blindly into oropharynx; either an esophageal or tracheal placement is possible. Following assessment of placement, patient is ventilated through lumen (port) that provides lung inflation. Airway seal is accomplished by inflation of large proximal oropharynx balloon. A smaller, distal balloon is then inflated to provide a tracheal seal (tracheal position) or a esophageal obturator (esophageal position) (AHA/ILCOR Guidelines, 2000).
 - b. ADVANTAGES: Aspiration risk is limited but not completely eliminated; provides oxygenation and ventilation (AHA/ILCOR Guidelines, 2000).
 - c. DISADVANTAGES: Presence of pharyngotracheal lumen airway may complicate endotracheal intubation. Further studies are needed before widespread use can be recommended (AHA/ILCOR Guidelines, 2000)
9. AIRWAY, LARYNGEAL MASK
- a. DESCRIPTION: A tube with cuffed mask-like projection at the distal end. Introduced through mouth into pharynx and fits over larynx, similar to the way a face mask fits over the face. Once inserted, cuff is inflated, establishing a secure open airway (AHA/ILCOR Guidelines, 2000; Reed, 1995; Samarkandi, 1994; Brain, 1983).
 - b. ADVANTAGES:
 - (1) Easy to insert; minimal training required; no need for laryngoscope. Useful when access to patient is limited, in presence of potential unstable neck injury, or appropriate positioning for endotracheal intubation is impossible; Class IIb recommendation (acceptable, possibly effective) (AHA/ILCOR Guidelines, 2000).
 - (2) One study involving over 700 patients with in-hospital cardiopulmonary arrest found initial use of laryngeal mask airway resulted in regurgitation in 4%; initial use of bag-valve-mask resulted in regurgitation in over 12% (Stone, 1998).
 - (3) Ventilation with laryngeal mask airway resulted in significantly less gastric inflation compared with bag-valve-mask ventilation (Doerges, 1999).
 - (4) Provides a more secure and reliable means of ventilation than face mask (AHA/ILCOR Guidelines, 2000).
 - c. DISADVANTAGES: May fail to occlude the esophagus, leading to esophageal insufflation and increased risk of aspiration; may be too large to insert if airway swelling is present.
10. OXYGEN
- a. Therapy with 100% inspired oxygen should be administered to all arrest victims as soon as available (AHA/ILCOR Guidelines, 2000; Zaritsky, 1998).
11. VENTILATION, MECHANICAL
- a. GENERAL: Used as a temporizing measure to provide ventilatory support. May be administered invasively (eg, endotracheal tube, tracheostomy) or noninvasively (eg, face or nasal mask). Modalities used may include assist/control, intermittent mandatory, synchronized intermittent mandatory, and pressure support ventilation. Adjuncts include continuous positive airway pressure, positive end expiratory pressure, permissive hypercapnia, and tracheal gas insufflation.
 - b. CAUTION: Physicians managing mechanically ventilated patients must be experienced and knowledgeable in the monitoring and therapeutic modalities used. (FOR FURTHER INFORMATION, SEE CLINICAL REVIEW: MECHANICAL VENTILATION)
12. OXYGEN SENSOR, TRANSCUTANEOUS
- a. Transcutaneous PO₂ and PCO₂ sensors may be used to continuously measure oxygen and carbon dioxide partial pressures in the blood during CPR (Tremper, 1980, 1981).

B. RESUSCITATION, CARDIOPULMONARY

1. OVERVIEW

a. RECOMMENDATION:

- (1) Compression rates of 100 per minute appear to generate greater forward blood flow and improved coronary perfusion (AHA/ILCOR Guidelines, 2000; Maier, 1984). Arterial pressure during chest compression is maximized when compression duration is 50% of the compression release cycle (AHA/ILCOR Guidelines, 2000).
 - (a) ADULTS: Approximately 80 and preferably 100 times per minute, with sternal depression of 1.5 to 2 inches.
 - (b) CHILDREN: Approximately 100 times per minute, with a sternal depression of 1 to 1.5 inches (Nadkarni, 1997).
 - (c) INFANTS: Approximately 120 times per minute, with sternal depression of 0.5 to 1 inch (Nadkarni, 1997).
- (2) If patient is in bed, a board should be placed beneath patient's back (AHA/ILCOR Guidelines, 2000). One study, utilizing a systematic mechanical analysis, found chest compressions performed on bed without board reduced depth and effectiveness of compressions (Boe, 1999).

b. MECHANISM:

(1) GENERAL:

- (a) Experimental evidence shows an increase in systolic and mean BP and in carotid blood flow during CPR (Rudikoff, 1980). Both increased intrathoracic pressure and direct compression of the heart may be responsible for varying portions of blood flow and tissue perfusion during closed chest CPR (Halperin, 1996).
- (b) The key determinant of successful resuscitation is the aortic to right atrial pressure gradient during the diastolic (or relaxation phase) of CPR. The gradient correlates with coronary blood flow (Paradis, 1991; Niemann, 1992).

(2) INCREASED INTRATHORACIC PRESSURE:

- (a) Several studies have found that increased intra-thoracic pressure is actually responsible for the flow of blood during CPR. The heart is thought to act as the passive conduit, with systolic and mean arterial pressures and blood flow to the carotid artery augmented by increased thoracic pressure, without direct compression of the heart (Niemann, 1981; Criley, 1981; Ornato, 1989). Higher compression pressures than those currently used may improve arterial systolic pressure and flow during closed-chest CPR (Ornato, 1989).
- (b) Presumably, blood flows during CPR because the intrathoracic pressure is transmitted into the extrathoracic arteries to a greater extent than into the extrathoracic veins. This is reflected in an extrathoracic arterial-venous pressure gradient. The unequal transmission of intrathoracic pressure into the extrathoracic arteries and veins results from the presence of venous valves and unequal arterial and venous capacitance and collapsibility. Arteries resist collapse, and therefore transmit the intrathoracic pressure into the extrathoracic arterial bed.
- (c) One study of 22 humans had aortic arch, jugular venous bulb, and right atrial catheters placed and pressures measured during CPR. The results indicated that most patients have functional venous valves at the thoracic inlet and the potential for a thoracic pump mechanism for cerebral perfusion (Paradis, 1989).
- (d) The mechanism of increasing intrathoracic pressure, however, does not explain why adequate arterial pressure, carotid flow, and cardiac output can be maintained during CPR in laboratory animals with vented chest tubes (Babbs, 1981). It has been suggested that chest compression causes the pleura to seal the chest tubes and thus create a closed system capable of transmitting the intrathoracic pressure equally (Chandra, 1981).

(3) DIRECT COMPRESSION:

- (a) Increasing intrathoracic pressure alone does not provide a gradient for blood flow or adequate perfusion pressure in the coronary arteries (Niemann, 1982). Inadequate coronary artery blood flow may exist in closed-chest CPR unless enough external compression force is applied to directly compress the heart (Ditchey, 1982).
- (b) Direct compression of the heart during closed-chest CPR may augment coronary blood flow (Ditchey, 1982), stroke volume, cardiac output, and regional blood flow to the cerebral hemispheres and kidneys (Luce, 1981).
- (c) Increasing the duration of external cardiac compression for at least 50% of cycle time also has been shown to be a more effective form of CPR (Halperin, 1996; Taylor, 1977).

c. EFFICACY:

- (1) A survival rate of 70% for closed-chest CPR initially reported (Kouwenhoven, 1960). Since that time, numerous series in both the hospital and prehospital setting have demonstrated much lower survival rates (Eynon, 1996).
- (2) In one study of 294 patients with in-hospital cardiac arrest, 44% survived initial resuscitation but only 14% were eventually discharged from the hospital (Bedell, 1983).
- (3) Among 1,562 out-of-hospital cardiac arrests in King County, Washington, 36% of patients were admitted to the hospital but only 19% were discharged (Eisenberg, 1982).
- (4) Factors identified as important in increasing survival from out-of-hospital cardiac arrest have included the presence of ventricular fibrillation as the initial rhythm and reduced times to initiation of CPR and defibrillation (Weaver, 1986; Aprahamian, 1986; Cummins, 1985).
- (5) An observational, prospective, population-based study, involving over 1100 patients with out-of-hospital VF, found 90 seconds of CPR prior to defibrillation improved survival when response interval was greater than 4 minutes compared with immediate AED use and defibrillation prior to CPR (Cobb, 1999).
- (6) A randomized trial, involving 241 patients with witnessed out-of-hospital cardiac arrest, found patients treated with bystander-initiated chest compressions alone had similar outcomes to those treated with bystander-initiated chest compressions and mouth-to-mouth ventilation. Survival to hospital discharge was higher in patients treated with chest compressions alone, although difference was not significant (Hallstrom, 2000).

d. COMPLICATIONS:

- (1) Fractures of the ribs and sternum, flail chest, rupture of the liver, spleen, and right and left ventricles, hemopericardium, hemopneumothorax, bone marrow emboli, and pulmonary barotrauma have all been reported (Rosenthal, 1986; Hillman, 1986). Retinal hemorrhage may occur (Kramer, 1993).
- (2) In a canine model of cardiac arrest, there was no significant correlation between method of CPR (standard CPR, high-impulse compression CPR, interposed abdominal compression CPR) and type of CPR-induced trauma found at autopsy; the most serious injuries were pulmonary hemorrhage and hepatic lacerations (Kern, 1986a).
- (3) Fatigue by CPR provider can result in a decay in the quality of chest compressions (Hightower, 1995).

e. PREGNANCY: In their 2nd and 3rd trimesters, pregnant women should be positioned on their left lateral side as much as possible (may be accomplished with a small wedge under right hip). The gravid uterus blocks the return of blood from the inferior vena cava; effective CPR cannot be done when woman in 3rd trimester lies on her back (AHA/ILCOR Guidelines, 2000).

f. ADJUNCTS: CPR-PLUS, a hand-held noninvasive device, provides feedback to rescuer regarding correct applied chest compressions. One preliminary study, involving 40 trained nurses performing CPR on manikins, found CPR performed with CPR-PLUS feedback resulted in significantly improved number of satisfactory compressions; rescuers reduced excessive compression pressure and incorrect hand positioning with CPR-PLUS feedback (Elding, 1998).

g. DISCONTINUING RESUSCITATION:

- (1) The American College Emergency Physicians (ACEP) policy statement indicates prehospital resuscitation efforts may be discontinued in specific, well-defined situations. Normothermic patients with asystole or wide-complex, pulseless bradyarrhythmias (less than 60 beats/minute) unresponsive to adequate trial of resuscitation, possibly including airway management, CPR, defibrillation, cardiac pacing, medications. In addition, field termination of resuscitation efforts should include appropriate involvement of medical control and grief support system to assist family members and friends (ACEP, 1998).
- (2) The Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care state treating physician may consider termination of resuscitation efforts (AHA/ILCOR Guidelines, 2000):
 - (a) When the treating physician is highly certain that the arrest victim will not respond to additional ACLS efforts.
 - (b) If there is no return of spontaneous circulation at any point during 30 minutes of total advanced cardiac life support, unless mitigating factors are present (eg, severe hypothermia, drug overdose).
 - (c) If there is return of spontaneous circulation of any duration during course of resuscitation, it may be appropriate to extend resuscitation efforts.

2. RESUSCITATION, CARDIOPULMONARY, IAC

a. GENERAL:

- (1) Several authors have suggested that the concept of increased intrathoracic pressure can be applied to increase the effectiveness of CPR. One proposed technique involves interposed abdominal compression CPR (IAC-CPR) (Sack, 1992, 1992a).
- (2) External pressure is applied to the abdomen during relaxation phase of chest compression. IAC-CPR denotes CPR with interposed abdominal compressions. The compressions are applied manually over the mid-abdomen, and pressure is maintained for about 50% of cycle time, exactly opposite the application of chest compression (AHA/ILCOR Guidelines, 2000; Babbs, 1985).

b. INDICATIONS:

- (1) ADULTS: An alternative to standard CPR for in-hospital resuscitation efforts when adequately trained personnel are available; Class IIb recommendation (acceptable, possibly effective) (AHA/ILCOR Guidelines, 2000).
- (2) CHILDREN: Clinical studies are needed to determine role in treatment of pediatric cardiac arrest (Zaritsky, 1998).

c. MECHANISM: There are two probable mechanisms for the hemodynamic effects of abdominal counterpulsation (Babbs, 1985, 1999).

- (1) Compression of the abdominal aorta during chest recoil forces blood retrograde toward the brain and heart. Aortic diastolic pressure is increased, with increased cardiac output and peripheral perfusion (Babb, 1985). Data from a mathematical model of CPR hemodynamics suggest cardiac output increases from 1.3 to 2.4 with the addition of abdominal counterpulsation (Babb, 1999).
- (2) Abdominal counterpulsation may "prime" the intrathoracic pump mechanism similar to the action of the atria in a normal heart (Babb, 1985). Data from a mathematical model of CPR hemodynamics suggest IAC-CPR increases abdominal aortic pressure leading to increased thoracic aortic pressure; action is similar to intra-aortic balloon counterpulsation (Babb, 1999).

d. HEMODYNAMIC EFFECTS:

- (1) IAC-CPR improves systolic and diastolic pressure, cardiac output, cerebral blood flow, and oxygenation (Berryman, 1984; Walker, 1984; Voorhees, 1983, 1984; Ralston, 1982).
- (2) In one study of 14 patients, although IAC-CPR significantly increased diastolic arterial pressure, the mean and diastolic AV pressure differences were not significantly increased (Howard, 1987).
- (3) In another study of six patients, addition of IAC to standard CPR significantly increased systolic and diastolic arterial pressures; however, the diastolic AV pressure difference was not increased (McDonald, 1985).
- (4) The central diastolic AV pressure difference is the primary determinant of artificial circulation during CPR; when venous pulses are larger than arterial pulses myocardial flow may be compromised (Babbs, 1985).
- (5) Another prospective, randomized study on 33 patients suggested that cardiac output (as evidenced by end-tidal PCO₂) is significantly increased during interposed abdominal compression CPR, and may increase the return of spontaneous circulation (Ward, 1989).

e. EFFICACY: In a randomized, prospective study of 143 cardiac arrest patients, IAC-CPR produced a significantly higher rate of return of spontaneous circulation and survival compared with standard CPR (Sack, 1992a). This result was not observed in a similar study (Mateer, 1985).

3. RESUSCITATION, CARDIOPULMONARY, ACD

a. DESCRIPTION: Active compression-decompression-CPR (ACD-CPR) is a mechanical CPR alternative aimed at improving hemodynamics during CPR (Babb, 1999).

- (1) Involves use of positive and negative pressure alternatively applied to chest wall with use of plunger-type device. Negative pressures of up to -30 lb are utilized (Babb, 1999; Shultz, 1994).
- (2) Data from a mathematical model of CPR hemodynamics suggest ACD-CPR increases cardiac output from 1.3 to 1.6 L/min compared with standard CPR (Babb, 1999).

b. INDICATIONS:

- (1) ADULTS: An alternative to standard CPR for resuscitation efforts when adequately trained personnel are available; Class IIb recommendation (acceptable, possibly effective) (AHA/ILCOR Guidelines, 2000).
- (2) CHILDREN: Clinical studies are needed to determine role in treatment of pediatric cardiac arrest (Zaritsky, 1998).

c. EFFICACY: Several studies have shown favorable clinical long-term outcomes (Palisance, 1997, 1999; Tucker, 1994; Lurie, 1994; Cohen, 1993); several studies have shown neutral results (Stiell, 1996; Schwab, 1995; Mauer, 1996; Luiz, 1996).

- (1) ACD-CPR may result in greater return of spontaneous circulation (Tucker, 1994; Cohen, 1993) and has been associated with higher end-tidal CO₂ levels (Orliaguet, 1995).

- (2) A 4-year, randomized, single-center, population-based study, involving over 300 patients with out-of-hospital cardiac arrest, found no clinical benefit in patients treated with ACD-CPR versus standard CPR (Skogvoll, 1999).
- (3) A prospective, controlled trial, involving 576 patients with out-of-hospital cardiac arrest found use of ACD-CPR did not provide any clinical benefits (eg, survival to hospital admission, neurologic outcomes, survival to hospital discharge) over standard CPR (Nolan, 1998).
- (4) A prospective, randomized trial, involving 120 patients with out-of-hospital cardiac arrest, failed to demonstrate difference in end-tidal CO₂ during ACD-CPR versus standard CPR (Mauer, 1998).
- (5) The French Active Compression-Decompression Cardiopulmonary Resuscitation Study Group found use of ACD-CPR significantly improved 1-year survival compared with standard CPR, based upon data from 750 patients with out-of-hospital cardiac arrest. Of note, all patients alive at one-year follow-up had witnessed cardiac arrest (Plaisance, 1999).
- (6) Clinical studies are needed to determine role of ACD-CPR in treatment of pediatric cardiac arrest (Zaritsky, 1998).

4. MESSAGE, CARDIAC, MECHANICAL

a. DESCRIPTION:

- (1) A mechanical compressor (Thumper(R)) with a universal programmable timer to allow unlimited variation in both the ventilatory cycle and the chest compression cycle. Allows standardization and reproducibility of techniques for external chest compression and the sequencing of ventilation.
- (2) The device has proven to be a valuable research tool for evaluation of CPR techniques; however, it has not found general usage in prehospital settings and has been used clinically in only specific cases, including prolonged CPR in hypothermia, near-drowning, or prolonged transportation times (Kirscher, 1980).

b. INDICATIONS (AHA/ILCOR Guidelines, 2000):

- (1) ADULTS: An alternative to standard CPR for resuscitation efforts when circumstances make manual chest compressions difficult (ie, certain transport situations, lack of adequate personnel); Class IIb recommendation (acceptable, possibly effective).
- (2) CHILDREN: Clinical studies are needed to determine role in treatment of pediatric cardiac arrest.

c. EFFICACY:

- (1) Mechanical chest compression may be superior to manual chest compression in generating higher mean arterial blood pressures (McDonald, 1982). In a randomized study in humans, mechanical compression of the chest was comparable to manual CPR compression (Cohen, 1993).
- (2) Data from a randomized trial involving 20 patients with out-of-hospital cardiac arrest suggest use of mechanical chest compression results in higher cardiac output, as measured by ET-CO₂, compared with human chest compression; however, outcomes did not differ between groups. Additional studies are needed to determine effect on patient outcome (Dickinson, 1999).

5. RESUSCITATION, CARDIOPULMONARY, SCV

a. GENERAL:

- (1) Several authors have suggested that the concept of increased intrathoracic pressure can be applied to increase the effectiveness of CPR. Techniques studied have included simultaneous compression ventilation (SCV) (AHA/ILCOR Guidelines, 2000).
- (2) These techniques have had varying experimental results and their clinical uses have not been established. Their routine use is not recommended (AHA/ILCOR Guidelines, 2000).

b. TECHNIQUE: SCV CPR describes chest compressions given simultaneously with ventilation. Reported rates of chest compression range from 40 to 60 per minute, with 50% to 60% compression duration and airway pressures from 60 to 110 cmH₂O; this technique had been combined with abdominal binding in some studies (Chandra, 1980; Bircher, 1981, 1982; Babbs, 1982).

c. HEMODYNAMIC FINDINGS:

- (1) Experimentally and in a limited number of clinical applications, SCV-CPR has been shown to increase systolic arterial pressure, carotid blood flow, and cardiac output (Chandra, 1980; Babbs, 1982; Redding, 1981; Kern, 1987).
- (2) Increasing the ventilatory pressure of interposed ventilations during CPR only slightly increases cardiac output (Babbs, 1980a).
- (3) No differences in arterial PO₂ and PCO₂ were demonstrated in one study of SCV vs standard CPR. Central venous pressures were significantly higher using the new method (Redding, 1981).
- (4) SCV-CPR does not appear to improve myocardial perfusion (Kern, 1987) and may actually worsen it (Martin, 1986).

d. CEREBRAL BLOOD FLOW:

- (1) Whether SCV-CPR improves or worsens cerebral blood flow remains unknown. Increases in intrathoracic pressure during chest compression are transmitted to the cranial vault via nonvalved

veins and CSF; the change in ICP is similar with conventional and SCV-CPR but is greatly increased by abdominal binding (Guerci, 1985).

(2) Experimentally, SCV-CPR with near normal intracranial pressures results in a lower cerebral perfusion pressure and decreased sagittal sinus PO₂ compared with standard CPR (Bircher, 1981).

(3) When higher intracranial pressures (45 mmHg) are delivered with SCV-CPR, however, the actual cerebral perfusion pressure (intracranial pressure minus arterial pressure) is increased secondary to a greater increase in carotid artery pressure (Koehler, 1981).

(4) Other studies suggest that regional blood flow to the brain is higher with SCV-CPR than blood flow to other organs (eg, heart, kidneys).

e. OUTCOME: There was no difference in 24-hour survival, and neurologic deficit scores in survivors were similar, in one experimental study of conventional, SCV, and vest CPR (Kern, 1987).

6. RESUSCITATION, CARDIOPULMONARY, HIGH-FREQUENCY

a. Has been suggested as an alternative for improving resuscitation following cardiac arrest. Preliminary human studies suggest improved hemodynamics with manual techniques (Kern, 1992; Swenson, 1988). Further studies are needed to define role in management of patients with cardiac arrest (AHA/ILCOR Guidelines, 2000).

7. RESUSCITATION, CARDIOPULMONARY, VEST

a. A pneumatically cycled circumferential thoracic vest system (vest CPR) has shown promising hemodynamic improvement in a preliminary study of cardiac arrest patients (Halperin, 1993). Large, multicenter trials of this technique are underway.

b. May be considered an alternative to standard in-hospital CPR or during ambulance transport only when adequate well-trained personnel are available; Class IIb recommendation (acceptable, possibly effective) (AHA/ILCOR Guidelines, 2000).

8. RESUSCITATION, CARDIOPULMONARY, COUGH

a. DESCRIPTION (Criley, 1981, 1983):

(1) A method of closed chest self-augmentation of blood flow in patients who are sufficiently awake to follow commands shortly after the onset of cardiovascular collapse preceding cardiac arrest.

(2) Patients experiencing asystole or ventricular fibrillation in an acute care setting, or during cardiac catheterization studies, can be induced to cough vigorously and thereby maintain arterial pressure and consciousness until definitive therapy can be administered.

b. MECHANISM:

(1) Coughing during cardiopulmonary collapse produces an arterial pulse, opens the aortic valve, generates anterograde blood flow, and can maintain consciousness during circulatory arrest.

(2) Experimentally, "cough CPR" has been reproduced in laboratory animals, and this suggests that pressure gradients in the thorax produce flow of blood out of the thoracic reservoirs by establishing a pressure gradient between the intrathoracic vessels and the extrathoracic outlets (Rosborough, 1981).

(3) In one report of patients with either ventricular fibrillation or tachycardia, 23 of 230 patients (10%) were treated with either a precordial thump or told to cough, and all were successfully converted (Caldwell, 1985).

9. RESUSCITATION, CARDIOPULMONARY, LIFESTICK

a. Involves a combination of interposed abdominal compressions and active-compression-decompression CPR. Self-adhesive compression pads applied to chest and abdomen facilitate active compression and decompression of chest and abdomen (Babb, 1999).

b. Data from a mathematical model of CPR hemodynamics suggest Lifestick CPR results in cardiac output of 3.1 L/min, greater than 2-fold increase over standard CPR (Babb, 1999). A preliminary animal study found similar results; 2-fold increase in coronary perfusion pressure noted. Significant improvements noted in successful resuscitation and 48-hour survival with Lifestick CPR compared with conventional CPR (Tang, 1997).

C. DEFIBRILLATION

1. OVERVIEW

a. INDICATIONS: Immediate defibrillation indicated for all patients with ventricular fibrillation or pulseless ventricular tachycardia (AHA/ILCOR Guidelines, 2000).

b. EARLY DEFIBRILLATION:

(1) The time to defibrillation is the major determinant of survival in cardiac arrest secondary to VF ((AHA/ILCOR Guidelines, 2000; Nichol, 1999; Heavens, 1998). Patients with VF have been found to have markedly improved survival with shorter resuscitation times ((AHA/ILCOR Guidelines, 2000; Pionkowski, 1983; Kerber, 1983).

(2) A prospective study of adult cardiac arrests treated by both first-responders and paramedics

found that the need for additional ALS (other than intubation) by the paramedics was associated with poor overall prognosis. Although aggressive ALS measures increased the number of survivors, 80% had poor neurologic outcome and 50% died within a year of discharge (Callaham, 1996).
(3) The OPALS (Ontario Prehospital Advanced Life Support) study Phase II found rapid defibrillation, accomplished by multisystem changes in EMS delivery, significantly improved survival in patients with out-of-hospital cardiac arrest; a 33% relative increase in survival reported (Stiell, 1999).
(4) Early defibrillation by first responders has been shown to dramatically decrease mortality and long-term neurologic sequela in patients with VF. Overall, 40% were discharged home without significant neurologic deficit following out-of-hospital treatment with automatic external defibrillators by first responders (ie, police or paramedics) (White, 1998).

c. ENERGY REQUIREMENTS:

- (1) The energy requirement for successful closed-chest defibrillation has been a source of continuing controversy. The AHA/ILCOR recommendation is a defibrillation attempt at 200 joules; if not successful, repeat shock at 200 to 300 joules, and if still unsuccessful, a third attempt utilizing 360 joules should be made ((AHA/ILCOR Guidelines, 2000).
- (2) Studies of defibrillation attempts in obese individuals, as well as from prospective investigations of defibrillation of heavy-weight animals, suggest that levels of energy greater than 400 joules were required for successful defibrillation (Geddes, 1974; Tacker, 1979).
- (3) Prospective studies, however, have reported that patients weighing 40 to 225 kilograms (selecting out those patients with agonal ventricular fibrillation) had a success rate of 98% to 99% defibrillation after shocks of just under 200 joules (2.7 joules per kilogram) (Gascho, 1979).
- (4) Studies of current flow delivered during ventricular fibrillation demonstrated that defibrillation occurred at 0.35 amps per kilogram, or approximately one-third the amperage suggested by the advocates of high-output defibrillation (Patton, 1979).
- (5) Clinical experience has indicated that much lower energy requirements are needed for transthoracic, as well as for open-chest, defibrillation of human beings. Although the controversy continues, the energy ranges employed in prospective clinical studies indicate that the elapsed time, the patient's diagnosis, the clinical setting, and the technique determine the outcome of defibrillation (Crampton, 1980; Adgey, 1979).
- (6) The use of biphasic defibrillation shocks rather than standard monophasic damped-sine wave shocks have been shown to have similar defibrillation efficacy while utilizing less energy. This results in less post-shock myocardial dysfunction (Reddy, 1997).

d. PADDLE LOCATION (AHA/ILCOR Guidelines, 2000):

- (1) Defibrillator paddles (standard paddle size is 8 to 8.5 cm in diameter) are currently placed in the anterior and apical locations for use in transthoracic defibrillation. The anterior paddle is placed to the right of the upper sternum below the clavicle; the apical paddle is placed to the left of the nipple with the center in the midaxillary line.
- (2) An alternative paddle placement involves placing the "apex" paddle anteriorly over the left precordium and the "sternum" paddle posterior to the heart over the right infrascapular location.

e. TRANSTHORACIC RESISTANCE:

- (1) Has been measured in the standard paddle positions and found to range widely from 15 to 170 ohms, with a mean of approximately 80 ohms. This wide variation in transthoracic resistance seems to be related to chest size (Kerber, 1981). The impedance to delivered energy can be reduced significantly by conducting gel and firm pressure on the electrodes during defibrillation.
- (2) Transthoracic resistance also decreases between the first and second shock at the same energy level. Orientation of electrodes in a posteroanterior pathway for flow of current is more efficient at lower levels of energy, especially in atrial defibrillation, and this also may apply to ventricular defibrillation (Crampton, 1980).
- (3) One study of pediatric transthoracic impedance indicated that the larger adult electrode paddles will minimize transthoracic impedance and should be used when the child's thorax is large enough; otherwise, the pediatric paddles should be used. This transition occurred at about 10 kilograms of body weight (Atkins, 1988).

f. DRUG INTERACTIONS: The presence or absence of certain drugs may affect the eventual outcome of defibrillation during cardiopulmonary resuscitation.

(1) EPINEPHRINE:

- (a) The alpha-adrenergic effects of epinephrine are probably most beneficial, producing increased myocardial and CNS blood flow during CPR. Others believe that the beta-adrenergic effects of epinephrine may increase myocardial work and decrease subendocardial perfusion (Callaham, 1990).
- (b) Coarse VF may be more susceptible to countershock because it occurs early in arrest.

(2) DIGOXIN:

(a) The presence of digoxin may predispose the myocardium to ventricular arrhythmias following successful defibrillation. One study has suggested that the deleterious interaction between digoxin and electric countershock occurs directly within the myocardial cell (Jones, 1980).

(b) Digoxin intoxication, in particular, greatly sensitizes the heart to electrical discharge; the use of delivered energies of 300 to 400 joules for defibrillation in the presence of digoxin intoxication may result in prolonged periods of heart block or asystole, which favor the re-emergence of ventricular fibrillation (DeSilva, 1980).

g. VENTILATORY PHASE:

(1) The phase of ventilation may play a significant role in the successful outcome of transthoracic defibrillation (Ventriglia, 1984). A study in laboratory animals showed a significantly higher transthoracic impedance with inspiration compared with expiration and a significant decrease in defibrillation success rate when shocks were delivered in full inspiration (10%), compared with expiration (50%) (Ewy, 1980).

(2) When the cardiopulmonary resuscitation efforts include the use of manual or bagged ventilations, most defibrillation occurs during the phase of relaxed expiration, when the operator stands away from the patient during the defibrillation attempt. However, these data may have more significance for patients being resuscitated on a mechanical ventilator or using "new" CPR.

h. COMPLICATIONS:

(1) The issue of myocardial injury and complications from transthoracic defibrillation remains confusing. Most information about myocardial injury derives from experiments in laboratory animals, and there are few investigations in humans.

(2) Experimental studies have shown that more than 100 shocks (between 180 and 220 joules) will produce no overt cardiac damage (Crampton, 1980). Myocardial damage is greatest when small electrodes are used and the shocks are closely spaced chronologically (DeSilva, 1980).

(a) Gross pathologic and microscopic examination of hearts of defibrillated laboratory animals has demonstrated a median toxic peak delivered current of 5.8 amps/kg and a median toxic delivered energy of 30 joules/kg (ie, morphologically detectable myocardial damage in 50% of animals studied) (Babbs, 1980a).

(b) Although this report suggests a wide safety margin for myocardial damage, a report of in vitro animal myocardial cells shows a high incidence of secondary arrhythmias following increasing energy doses for successful defibrillation (Jones, 1980a).

(3) Clinical data suggest that although some myocardial damage may occur with repeated defibrillation shocks, elevation of the CK-MB isoenzyme occurs only rarely (Reiffel, 1979).

(a) Fewer than 50% of patients receiving over 400 joules delivered energy for defibrillation have a rise in the concentration of cardiac isoenzymes (Crampton, 1980).

(b) Another report showed that positive myocardial scintigrams in multiply defibrillated patients do not occur unless there is an associated transmural myocardial infarction (Werner, 1980).

(c) Complications of countershock (eg, arrhythmias) may occur without significant changes as measured by enzyme elevation or abnormal scan. In clinical studies, excessive energy countershocks produced a higher incidence of post-shock arrhythmias, including PEA, asystole, and heart block (Weaver, 1982; DeSilva, 1980; Kerber, 1983; Crampton, 1980).

(4) Successive high energy shocks release tissue potassium, increase intracardiac temperatures, depress left ventricular function and myocardial contractility, and increase myocardial diastolic stiffness.

(a) Delivery of higher energies for defibrillation seems likely to induce myocardial injury, which has led some authors to recommend that the minimum voltage required for successful defibrillation always be used (Jones, 1980a).

(b) As the energy of the shock increases, ventricular arrhythmias occur more often and may become life-threatening, particularly if the subject has already received a cardiac glycoside (Lown, 1978).

2. DEFIBRILLATION, MANUAL

a. RECOMMENDATION:

(1) ADULTS: Initial shock of 200 joules, followed by a second attempt at 200 to 300 joules. With failure of the second shock, defibrillate a third time at 360 joules. The three shocks should be delivered one right after the other in a stacked sequence without pausing for a pulse check or CPR

(AHA/ILCOR Guidelines, 2000).

(2) CHILDREN: Initial shock of 2 joules/kilogram, followed by a second attempt at 2 to 4 joules/kilogram. With failure of the second shock, defibrillate a third time at 4 joules/kilogram (AHA/ILCOR Guidelines, 2000).

(3) PREMEDICATION: According to some animal studies, when VF has been prolonged (eg, ≥ 8 minutes), administration of high-dose epinephrine should precede electrical countershock (Niemann, 1992). The advantage to this is that it establishes a brief period of coronary artery perfusion.

(4) RECURRENT VF: If ventricular fibrillation recurs during the arrest sequence, defibrillation should be reinitiated at the energy level that was previously required for successful defibrillation (AHA/ILCOR Guidelines, 2000).

b. CAUTION: Electrical arcing, fire, and burns can result. Minimize the presence of oxygen-enriched spaces in the vicinity of the paddles; apply paddles firmly (ECRI, 1994).

c. EARLY DEFIBRILLATION:

(1) The time to defibrillation is the major determinant of survival in cardiac arrest secondary to VF (AHA/ILCOR Guidelines, 2000; Nichol, 1999; Heavens, 1998). Patients with VF have been found to have markedly improved survival with shorter resuscitation times (Pionkowski, 1983; Kerber, 1983; AHA/ILCOR Guidelines, 2000).

(2) A prospective study of adult cardiac arrests treated by both first-responders and paramedics found that the need for additional ALS (other than intubation) by the paramedics was associated with poor overall prognosis. Although aggressive ALS measures increased the number of survivors, 80% had poor neurologic outcome and 50% died within a year of discharge (Callahan, 1996).

(3) The OPALS (Ontario Prehospital Advanced Life Support) study Phase II found rapid defibrillation, accomplished by multisystem changes in EMS delivery, significantly improved survival in patients with out-of-hospital cardiac arrest; a 33% relative increase in survival reported (Stiell, 1999).

(4) Early defibrillation by first responders has been shown to dramatically decrease mortality and long-term neurologic sequela in patients with VF. Overall, 40% were discharged home without significant neurologic deficit following out-of-hospital treatment with automatic external defibrillators by first responders (ie, police or paramedics) (White, 1998).

3. DEFIBRILLATOR, AUTOMATED EXTERNAL

a. FUNCTION:

(1) The AED is a simple device that can be used by nonprofessional rescuers to treat out-of-hospital cardiac arrest; allows those with no medical skills to operate it with minimal or no training.

(2) Adhesive electrodes are applied to sternum and apex and attached to defibrillator with flexible cables, allowing hands-free defibrillation. AED is able to monitor and analyze the cardiac rhythm and direct the operator to deliver a shock, eliminating the need for operator interpretation of rhythm (AHA/ILCOR Guidelines, 2000).

b. TYPES: Defibrillator devices that range from being "fully" automated, semiautomated, to manually operated (AHA/ILCOR Guidelines, 2000).

(1) FULLY AUTOMATED EXTERNAL DEFIBRILLATOR: Limited training is required. Operator must attach defibrillator pads and turn on the device; a shock will be delivered if VF or VT with rate greater than a preset rate is recognized. Are available only for special situations.

(2) SEMIAUTOMATED EXTERNAL DEFIBRILLATOR: Also called a shock-advisory AED. Device automatically analyzes cardiac rhythm and then "advises" the operator to press a "shock" control to deliver the shock. Final decision to defibrillate is left to the operator.

c. INDICATIONS (AHA/ILCOR Guidelines, 2000):

(1) An important, life-saving addition to basic life support interventions. AED use has been added to the American Heart Association and the International Liaison Committee on Resuscitation for trained lay rescuers and healthcare personnel.

(2) Early defibrillation should be provided in the community and accomplished within 5 minutes from EMS call.

(3) Early defibrillation also should be provided in hospital and medical facilities (eg, ambulatory surgery centers); defibrillation should be accomplished within 3 minutes; Class I recommendation (acceptable, definitely effective).

d. EFFICACY:

(1) OUT-OF-HOSPITAL:

(a) A study in a two-tiered urban-suburban EMS system found that use of AEDs by first-responder EMTs did not improve survival from sudden cardiac death. These data do not support routinely equipping initial responders with AEDs as isolated enhancement and question its cost-effectiveness (Sweeney, 1998).

(b) Population density is an independent predictor of survival from out-of-hospital cardiac arrest; there is little benefit with AED used in BLS services in rural areas (population densities <100/sq mi) (Stapczynski, 1997).

(c) Earlier studies of patients in cardiac arrest treated with an AED by first-responding firefighters before arrival of paramedics found that the device can be used as an adjunct to basic life support, and its use may improve survival by shortening time to defibrillation; overall impact of AED on community survival rates remained uncertain (Weaver, 1986a, 1988).

(d) A randomized, controlled study of 321 cardiac arrest patients comparing effectiveness of EMT use of AEDs vs standard defibrillators found no significant differences in hospital admission or discharge, sensitivity for VF or specificity for non-VF, or rates of defibrillation to a non-VF rhythm between treatment groups (Cummins, 1987).

(e) In another study, none of 113 patients with cardiac arrest who were treated by AEDs survived; the reasons for these poor results may have included failure of bystanders to provide CPR, delays in calling an ambulance, and inadequate availability of defibrillators (Gray, 1987).

(f) A retrospective, cohort study, involving 310 patients with out-of-hospital cardiac arrest, found time to first defibrillation was the most important determinant of both successful shock and survival (Heavens, 1998).

(g) An observational, prospective, population-based study, involving over 1100 patients with out-of-hospital VF, found 90 seconds of CPR prior to defibrillation improved survival when response interval was greater than 4 minutes compared with immediate AED use and defibrillation prior to CPR (Cobb, 1999).

(h) AED use by first responder police officers significantly decreased time to first defibrillation. Although overall survival of patients during intervention period was not different from historical control group, improved survival was noted when police arrived at scene first and shocked patients with AED compared with patients shocked by EMTs (Mosesso, 1998).

(i) A small, retrospective, cohort study of children with out-of-hospital cardiac arrest found AED accurately recognized and advised in all cases of nonshockable rhythm; VF was correctly identified in 88% of cases. Accurate rhythm identification had a sensitivity of 88% and specificity of 100% (Atkins, 1998).

(2) INHOSPITAL: A multicenter trial, involving 79 patients monitored in electrophysiology laboratory or cardiac care unit, found inhospital use of fully AEDs appropriately delivered or advised in all arrhythmic episodes. Performance of AED had a sensitivity of 100% and specificity of 98.8%; average response time was 22 seconds (Mattioni, 1999).

e. SPECIAL SITUATIONS: The following situations require modification to use of AED (AHA/ILCOR Guidelines, 2000):

(1) WATER: Victim should be moved from standing water and chest should be dried prior to AED use. If victim has sustained a diving injury or other potential spinal cord injury, cervical spine should be immobilized prior to transfer or resuscitation.

(2) CHILDREN: Use of AED is not recommended in children under 8 yr. AED use in children over 8 yr, weighing over 25 kg, is recommended. Additional studies are needed to determine appropriate energy doses.

(3) TRANSDERMAL MEDICATIONS: AED should not be placed over transdermal medication patches; medication should be removed and area wiped clean prior to AED pad application.

(4) IMPLANTED PACEMAKERS/ICD: AED pad should be placed at least 1 inch from implanted device.

4. DEFIBRILLATION, PREGNANCY

a. Transthoracic defibrillation during pregnancy should proceed as any other defibrillation during CPR, as death of both the mother and the fetus will almost certainly follow unsuccessful defibrillation (AHA/ILCOR Guidelines, 2000).

b. Defibrillation with successful termination of ventricular fibrillation using 300 joules has been reported, with no electrocardiographic changes in the fetus (DeSilva, 1980).

5. DEFIBRILLATION, HYPOTHERMIA

a. Although hypothermia itself increases the propensity of the ventricles to fibrillate, experimental data shows that hypothermia alone does not increase the defibrillation threshold; therefore, the dose for defibrillation is probably unchanged from the dose in the normothermic patient (Tacker, 1981; Ventriglia, 1984).

b. If VF is present, the hypothermic patient should be shocked three times; if unsuccessful, further attempts

at defibrillation and intravenous medications should be avoided until core body temperature is greater than 30 degrees C (86 degrees F). Until rewarming is accomplished, successful defibrillation may not be possible (AHA/ILCOR Guidelines, 2000). (FOR FURTHER INFORMATION, SEE CLINICAL REVIEW: HYPOTHERMIA)

6. DEFIBRILLATION, OPEN CHEST

a. Direct open-chest defibrillation with epicardial paddles requires 10 to 20 joules of delivered energy and will give a 90% successful defibrillation rate. The same dose may be repeated if necessary (Kerber, 1981).

7. CARDIOVERTER-DEFIBRILLATOR, IMPLANTABLE

a. GENERAL: Patients with implantable cardioverter-defibrillators (ICD) are at high risk for ventricular arrhythmias. If VF or VT is present in patient with ICD, external defibrillation should be performed immediately. The ICD will become nonoperative following initial series of shocks and will reset when nonfibrillatory rhythm occurs (AHA/ILCOR Guidelines, 2000).

b. SPECIAL CONSIDERATIONS:

(1) Although the rescuer may feel the shock if the ICD discharges while touching the patient, it will not be dangerous to rescuer.

(2) ICD units are protected against damage caused by external defibrillation but will need a readiness check following any external defibrillation. External defibrillation paddles should be placed at least 1 inch from ICD device. ICD units are protected against damage caused by external defibrillation but will need a readiness check following any external defibrillation (AHA/ILCOR Guidelines, 2000).

(3) If external defibrillation attempts of 360 J are unsuccessful, chest electrode position should be changed (eg, anterior-apex to anteroposterior) and the shock repeated. Change in position may increase transcardiac current flow and facilitate defibrillation.

8. DEFIBRILLATION, BIPHASIC, WAVEFORM

a. Biphasic shocks, an alternative defibrillation waveform, accomplish defibrillation at lower energy levels than traditional monophasic shocks. Biphasic waveforms deliver current that initially flows in positive direction for specific duration; direction of current flow is then reversed and moves in negative direction (AHA, 1998).

b. A preliminary, multicenter, prospective, randomized, blinded study, involving nearly 300 patients during testing of implantable defibrillator, suggest 130-joule biphasic truncated transthoracic shocks defibrillated as effectively as 200-joule standard transthoracic monophasic shocks. In addition, fewer ST-segment abnormalities noted postshock in patients who received biphasic shock compared with monophasic shock (Bardy, 1996).

c. Use of biphasic shocks may facilitate several advances in AED technology, including smaller size, lower price, more lightweight design, and improved battery life (AHA, 1998; Bardy, 1996).

D. PRECORDIAL THUMP

1. GENERAL: The chest thump, or precordial blow of the fist, has been reported to convert ventricular fibrillation.

2. INDICATIONS: At present, the chest thump is not recommended for termination of ventricular fibrillation when proper defibrillation equipment is available. A solitary precordial thump is recommended in patients with monitored ventricular fibrillation and in witnessed cardiac arrests when a defibrillator is unavailable (AHA/ILCOR Guidelines, 2000).

3. EFFICACY:

a. In patients with VF or VT, 23 of 230 patients (10%) were treated with either a precordial thump or told to cough, and all were converted successfully (Caldwell, 1985).

b. Among 50 patients who received prehospital thumps for VT or VF, thumping for VF was always useless. Twelve of 27 VT patients remained in VT and 12 were converted to VF, PEA, or asystole. Overall, only 3 patients benefited from the maneuver and 12 were clearly harmed (Miller, 1984).

4. ADVERSE EFFECTS: Unlike electrical cardioversion, the thump cannot be synchronized to a safe part of the cardiac cycle. The detrimental effects of an ill-timed thump are more likely to occur if the dysrhythmia is long-standing and if acidosis and hypoxia have supervened (Ewy, 1986).

E. THORACOTOMY

1. CHEST COMPRESSION, OPEN

a. GENERAL:

(1) Not an adjunct for maintaining artificial circulation but a special technique for providing near-normal cerebral and cardiac circulation (Kern, 1991, 1987; Geehr, 1986; Sanders, 1985, 1984; Del Guercia, 1965).

(2) Used early in cardiac arrest after a short period of unsuccessful closed-chest CPR (Kern, 1987; Sheikh, 1994). Not effective when applied late (Geehr, 1986). Not justified for routine use in CPR (AHA/ILCOR Guidelines, 2000).

b. INDICATIONS:

- (1) Penetrating chest or abdominal trauma leading to cardiac arrest; Class IIb recommendation (acceptable, possibly effective) (AHA/ILCOR Guidelines, 2000).
- (2) Cardiac arrest secondary to hypothermia, massive pulmonary embolism, cardiac tamponade (refractory to pericardiocentesis), or abdominal hemorrhage; Class IIb recommendation (acceptable, possibly effective) (AHA/ILCOR Guidelines, 2000).
- (3) Significant chest deformities (eg, flail chest, scoliosis, lordosis) precluding adequate closed-chest compressions; Class IIb recommendation (acceptable, possibly effective) (AHA/ILCOR Guidelines, 2000).
- (4) Blunt trauma leading to cardiac arrest (should not be used if vital signs are absent in the field) (Girardi, 1999).
- (5) May be considered in pregnant patients after 15 minutes of continuous CPR (Lee, 1986).
- (6) When the closed-chest procedure has proved to be unsuccessful and the duration of arrest is <20 min (Robertson, 1991).

c. HEMODYNAMIC FINDINGS: In experimental studies, open-chest cardiac massage (OCCM) markedly increased carotid artery blood flow while lowering venous and intracranial pressure; this resulted in higher cerebral and systemic perfusion pressures and may lead to improved cardiac and cerebral recovery (Alifimoff, 1987).

- (1) OCCM provided significantly better coronary artery blood flow and the success of resuscitation was inversely related to the duration of inadequate coronary blood flow (Sanders, 1985).
- (2) Dog studies also have shown better brain preservation or cerebral blood flow with OCCM than with closed-chest methods, even after 30 minutes of compressions (Bircher, 1984; White, 1985). OCCM was not associated with the increased intracranial pressure seen with SCV-CPR (Bircher, 1983).
- (3) OCCM with a cross-clamped descending aorta is superior to closed-chest and other forms of open-chest CPR as measured by cerebral blood flow, cardiac index, mean arterial pressure, and stroke index (Bircher, 1981).
- (4) In three patients requiring both open- and closed-chest CPR, cardiac index and stroke volume index were twice as high with the open method (Del Guercio, 1965).
- (5) OCCM provides greater mean coronary perfusion pressures compared with closed-chest CPR (Boczar, 1995).

d. EFFICACY:

- (1) Numerous series prior to 1960 involving 10 to 1200 patients reported success rates ranging from 10% to 40%. Most of these reports involved surgical patients already in the operating room who had received multiple anesthetic agents, and documentation of the cardiac rhythm at the time of arrest was usually not done (Rosenthal, 1986).
- (2) Since that time there have been case reports of patients resuscitated with OCCM, even after prolonged unsuccessful closed-chest massage (Hachimi-Idrissi, 1997; Paradis, 1992; Sanders, 1985).
- (3) Does not improve survival in children who have undergone ≥ 20 minutes of closed-chest CPR.
- (4) Emergency cannulation of descending thoracic aorta during thoracotomy to establish rapid vascular access and fluid resuscitation has been described (Girardi, 1999).
- (5) A study of 16 patients undergoing 5 minutes of closed-chest CPR followed by 5 minutes of open-chest CPR did not show significant differences in acidosis between the two techniques (Henneman, 1988).

e. TIMING OF OCCM:

- (1) Among 49 patients with witnessed, nontraumatic out-of-hospital cardiac arrest, OCCM did not improve survival compared with standard CPR. However, total arrest and field times prior to initiation of OCCM averaged almost 20 minutes in these patients, possibly precluding resuscitation (Geehr, 1986).
- (2) OCCM may offer no advantage over standard CPR after a prolonged period of inadequate myocardial perfusion (Sanders, 1985).

f. REQUIREMENTS:

- (1) Should be performed within 15 minutes from onset of cardiac arrest; therefore, use is limited.
- (2) Well-coordinated, multidisciplinary team with operating room facilities and thoracic surgery back-up to close the thoracotomy if the resuscitation is successful (Sanders, 1988).

g. COMPLICATIONS: Incidence unknown. Reported complications include trauma to the heart, lung, aorta, and esophagus and infections of the thorax (Rosenthal, 1986).

2. VENTRICULAR ASSISTANCE, MECHANICAL

a. Direct mechanical ventricular assistance utilizing a glass cup lined by a silastic diaphragm that can be inflated and deflated has been available since the mid-1960s. This technique requires thoracotomy.

- b. Further evaluation is required before this device can be recommended for general use (McCabe, 1983).
- 3. MASSAGE, CARDIAC, MINIMALLY INVASIVE DIRECT
 - a. This technique uses a small, intercostal incision through which a simple plunger-like device is placed directly onto the heart with the pericardium left intact. Manual depression of the device compresses the ventricles, producing an artificial systole (Buckman, 1995, 1997).
 - b. In animal studies, this technique has been shown to produce hemodynamic values and metabolic indices of organ perfusion similar to formal OCCM (Buckman, 1995, 1997).

F. INTRAVENOUS LINE

1. GENERAL:

- a. Intravenous access is critical for administering emergency medications to adults and children. However, during cardiac arrest, nonpharmacologic therapies (eg, CPR and defibrillation) are primary interventions and drug administration is secondary (AHA/ILCOR Guidelines, 2000).
 - (1) Current American Heart Association/International Liaison Committee on Resuscitation standards recommend cannulation of a peripheral vein (ie, antecubital or external jugular) if IV access was not obtained prearrest (AHA/ILCOR Guidelines, 2000).
 - (2) Central (eg, internal jugular or subclavian) lines should be used if they are in place when the arrest occurs; otherwise, the antecubital vein should be the site of choice so as not to interrupt CPR (AHA/ILCOR Guidelines, 2000).
- b. Studies utilizing the inferior vena cava in low-flow states have shown this to be a less effective route than superior vena cava administration (Dalsey, 1984; Hedges, 1984). Studies have demonstrated a significant delay in arrival of drugs at the heart when peripheral IV sites are used for injection during CPR; distal wrist and hand veins and distal saphenous veins in the leg are the least favorable sites for IV access.
- c. Compared with standard landmark-oriented approach for obtaining femoral vein catheterization in patients requiring IV access during CPR, real-time US-guidance is faster and provides lower rate of inadvertent arterial catheterization and higher success rate (Hilty, 1997).

2. SEQUENCE (AHA/ILCOR Guidelines, 2000):

- a. If a central venous line is in place at the time of arrest, it should be used for drug administration.
- b. If no vein has been cannulated, the antecubital vein is the site of first choice because CPR may have to be interrupted for placement of a central line.
- c. If the circulation is not quickly restored after initial drug administration through a peripheral line, a central line should be placed by an experienced operator with minimum interruption of CPR.
- d. If an endotracheal tube has been placed and there is a delay in obtaining venous access, epinephrine, atropine, and lidocaine may be administered via the endotracheal route.
- e. Sublingual vein injection has been described as a potentially life-saving route of vascular access in the absence of all other traditional techniques (Rothrock, 1993).

3. CHILDREN: The American Heart Association/International Liaison Committee on Resuscitation recommend a protocol for the establishment of vascular access during resuscitation (AHA/ILCOR Guidelines, 2000).

- a. If a central line in place at the time of arrest, it should be used; Class IIa recommendation (acceptable, probably effective). If not, an experienced provider may attempt insertion of central venous catheter. Femoral vein commonly used; probably safest and easiest to cannulate.
- b. Peripheral venous access permits administration of fluids and drugs if access can be established rapidly.
- c. In infants and children, intraosseous access should be considered if venous access cannot be achieved rapidly. Provides an excellent alternative to IV access.

G. INFUSION, INTRAOSSEOUS

1. GENERAL: Excellent alternative when venous access not available in pediatric patients; Class IIa recommendation (acceptable, probably effective) (AHA/ILCOR Guidelines, 2000).

2. INDICATIONS:

- a. Critically ill or injured children ≤ 6 years when IV fluids and medications are urgently needed in life-threatening conditions and traditional methods of intravascular access are unavailable, unacceptably delayed, or unsuccessful (Zaritsky, 1998; Rosetti, 1985; Berg, 1984; Parrish, 1986; Glaeser, 1986, 1988; Brunette, 1988).
- b. Specific situations in which intraosseous infusion may be beneficial include:
 - (1) Fluid resuscitation in hypovolemic shock (Ponaman, 1990).
 - (2) Initiation of vasopressor therapy in cardiac arrest (Ponaman, 1990).
 - (3) Poorly accessible veins due to burns, circulatory collapse, hemorrhage, severe dehydration, trauma, allergy (Rosetti, 1985; Goldstein, 1990; Walsh-Kelly, 1986; Glaeser, 1986; McNamara, 1987) or few veins available because of thrombosis due to prior use, obesity, edema (MacGregor, 1990).
 - (4) Initial correction of metabolic acidosis (Ponaman, 1990).
 - (5) Administration of anticonvulsants in patients with status epilepticus (Ponaman, 1990).

- (6) Administration of succinylcholine to facilitate emergency airway control (Tobias, 1990).
 - (7) Limited IV skills (MacGregor, 1990).
 - (8) Prehospital care of young children with cardiopulmonary arrest when lack of vascular access is life threatening (Seigler, 1989; Miner, 1989).
 - (9) Preliminary data suggest possible use in nontraumatic resuscitations in adults and older children, particularly in critical care situations when there is difficulty obtaining vascular access by other routes (Iserson, 1986, 1989; Valdes, 1977).
3. CONTRAINDICATIONS: Femur or tibia fracture; significant overlying cellulitis; known osteogenesis imperfecta (Seigler, 1989).
4. TECHNIQUE:
- a. SITE:
 - (1) In children ≤ 6 years of age, proximal tibia 2 to 3 cm below tubercle on anterior medial surface or distal femur 2 to 3 cm above external condyles in midline may be used; tibia is preferred site, especially in infants (AHA/ILCOR Guidelines, 2000; Rosetti, 1985; Parrish, 1986; McNamara, 1986; Ponaman, 1990).
 - (2) The iliac crest and medial or lateral supramalleolar area are alternative sites (Iserson, 1986). Medial malleolus has been used successfully in adults (Iserson, 1986, 1989).
 - (3) The sternum should NOT be used in young children and infants (Tocantins, 1945; Quilligan, 1946).
 - b. NEEDLES:
 - (1) TYPE:
 - (a) Commercially prepared intraosseous devices with guards to prevent too deep penetration and mechanisms that provide ease in stabilizing and securing the needle are available (Melker, 1990).
 - (b) Alternatively, a large-bore short bone marrow needle or an 18- to 22-gauge spinal needle may be used (Parrish, 1986; Ponaman, 1990). Bone marrow needles are preferred because blockage with bony material and bending are less likely, but the more readily available spinal needle with stylet is acceptable (Berg, 1984; Rosetti, 1985; McNamara, 1986).
 - (c) If necessary, a standard (14-, 16-, or 18-gauge) or 21-gauge "butterfly" hypodermic needle (MacGregor, 1990) may be used for short-term infusion (may be blocked by bone spicules during insertion).
 - (2) SIZE FOR AGE (Ponaman, 1990):
 - (a) CHILDREN < 18 MONTHS: 18- to 20-gauge spinal or intraosseous needle.
 - (b) CHILDREN > 18 MONTHS: 13- to 16-gauge bone marrow or intraosseous needle.
 - c. INSERTION (Rosetti, 1985; McNamara, 1987; Parrish, 1986; Ponaman, 1990):
 - (1) Strict aseptic technique must be followed. Clean skin with povidone-iodine solution.
 - (2) Restrain extremity; place sandbag beneath leg.
 - (3) Infiltrate area with local anesthetic (1% lidocaine) if patient is conscious.
 - (4) Insert needle 1 to 3 cm below tibial tuberosity on flat anteromedial surface or 2 to 3 cm above external condyles in anterior midline of femur. Insert needle almost perpendicular to skin or directed inferiorly at about 60 degrees (tibia) or superiorly (femur) away from epiphyseal plate.
 - (5) Advance needle to periosteum. Apply firm pressure with a boring or screwing motion until penetration of marrow occurs (detected by sudden lack of resistance or "give").
 - (6) Remove stylet. Attach a 10-mL syringe, filled halfway with 0.9% NaCl and connected to a T-connector, to the needle. Aspirate marrow to confirm placement.
 - (7) Attach IV tubing to needle for infusion by gravity or pressure (up to 41 mL/min with pressure 300 mmHg).
 - (8) After insertion, it is imperative that a member of the resuscitation team be responsible for protecting the needle from displacement.
 - (a) An intraosseous needle with protective guards may be adequately secured with tape.
 - (b) To secure a bone marrow or spinal needle, a hemostat or clamp may be attached across the needle at the level of the skin and secured to the leg with tape.
 - (c) Secure the limb to a padded board, immobilizing the extremity.
 - d. CONFIRMATION OF NEEDLE PLACEMENT (Berg, 1984; Ponaman, 1990):
 - (1) Aspiration of bloody marrow contents (if no aspirate observed, introduce fluid under pressure; if infiltration not observed, may proceed with administration of medications (Seigler, 1989)).
 - (2) Attempts at flushing needle with 5 to 10 mL 0.9% NaCl meet with only slight resistance and little oozing.
 - (3) Free flow of infusion (flow under pressure without progressive edema of surrounding tissues).

(4) Rigid fixation of needle (needle feels firmly inserted and stands upright without support).

5. FLUIDS/DRUGS:

a. Many fluids and drugs have been infused with clinical efficacy. Most have not been studied from the perspective of short- or long-term effects on the bone marrow. The following have been given via the intraosseous route:

- Antibiotics (eg, penicillin, sulfonamides)
- Antitoxins
- Atropine
- Blood and blood products
- Calcium chloride
- Calcium gluconate
- Crystalloids (D5W, NS, Ringer's lactate, Normosol, Hartman' solution)
- Contrast material
- Dexamethasone
- Dextran 40
- Diazepam
- Diazoxide
- Digitalis
- Dobutamine
- Dopamine
- Ephedrine
- Epinephrine
- Glucose
- Heparin
- Hydroxyethyl starch
- Insulin
- Lidocaine
- Phenytoin
- Sodium bicarbonate
- Succinylcholine and other anesthetic agents

(Parrish, 1986; Valdes, 1977; Walsh-Kelly, 1986; Glaeser, 1986; Berg, 1984; Rosetti, 1985; Heinild, 1947; Meola, 1944; Tarrow, 1952; McNamara, 1987).

b. Not adequate to serve as method for rapid fluid replacement. Even with use of external pressure device on fluid reservoir, flow rate through needle not high enough to be effective in volume resuscitation, eg, in patients with trauma or GI bleeding (Iserson, 1986, 1989; Valdes, 1977; Hodge, 1987).

c. HYPERTONIC/IRRITATIVE SOLUTIONS:

(1) Intraosseous administration of hypertonic solutions may be associated with an increased risk of osteomyelitis (Heinild, 1947).

(2) Although administration of sodium bicarbonate and glucose in hypertonic solutions and of highly alkaline phenytoin without problems has been reported (Glaeser, 1986; Walsh-Kelly, 1986).

(3) When tested in animal models, these solutions were found to be safe (Spivey, 1987; Brickman, 1988).

(4) Their use may NOT be contraindicated in emergency situations, although dilution is advised (Glaeser, 1986; Parrish, 1986; Rosetti, 1985; McNamara, 1986).

6. DURATION:

a. Attempts to obtain vascular access by other means should be continued and intraosseous infusion maintained only until an alternative route is available (Glaeser, 1986; Miner, 1989; Hodge, 1987).

b. Infusion must be discontinued within 12 hours; second intraosseous infusion may be started at alternate location (Ponaman, 1990).

7. MECHANISM:

a. Because long bones contain abundant marrow sinusoids that are drained by large medullary venous channels that promptly empty into systemic venous circulation, the medullary cavity is noncollapsible space allowing rapid entry into central circulation (Rosetti, 1985; Parrish, 1986).

b. Medullary space is replaced with fat cells at 2 to 3 years of age (McNamara, 1986).

8. ADVANTAGES:

a. Provides simple, reliable, and rapid (<60 sec) vascular access (AHA/ILCOR Guidelines, 2000) .

b. Achieves peripheral to central infusion transit times comparable with those achieved by IV route (Cameron, 1989).

- c. Serum drug levels, peak effect, and duration of action comparable to those following central and peripheral IV access (Brickman, 1988; Walsh-Kelly, 1986; Spivey, 1985; Glaeser, 1988; Orłowski, 1990).
 - d. Tibial and femoral insertion sites have fixed anatomic relationships to guide insertion, with few critical structures to injure (Glaeser, 1986; Mayer, 1986; Rosetti, 1985).
 - e. Insertion site relatively distant from performance of cardiopulmonary resuscitation and airway control measures (Glaeser, 1986; Mayer, 1986; Rosetti, 1985).
 - f. Low risk of known complications, especially compared with percutaneous central venous access (further studies are needed to define effects on erythropoiesis and bone growth) (Glaeser, 1986; Mayer, 1986; Rosetti, 1985).
 - g. Can infuse large volumes of fluid and a wide variety of drugs, compared with endotracheal route (Glaeser, 1986; Mayer, 1986; Rosetti, 1985; Orłowski, 1990).
 - h. Can be used successfully by prehospital care providers; IO needle can be placed in back of moving emergency vehicle (Smith, 1988; Seigler, 1989; Miner, 1989).
9. COMPLICATIONS: Significant complications rare (<1%); primarily related to improper technique, infusions of hyperosmolar fluids, or long-term use (Berg, 1984; Parrish, 1986; Glaeser, 1986). The following complications or sequelae have been reported:
- a. OSTEOMYELITIS: No cases reported since 1977 (Brickman, 1988). Reported in early series, primarily in association with prolonged infusion, placement in bacteremic patients, and use of hypertonic solutions (Massey, 1950; Rosetti, 1985).
 - b. SUBPERIOSTEAL/SUBCUTANEOUS EXTRAVASATION:
 - (1) Rare; may occur if needle does not penetrate marrow cavity, resulting in leakage around needle and possible pressure necrosis (Berg, 1984; Walsh-Kelly, 1986; Quilligan, 1946; Rosetti, 1985).
 - (2) Placing 2 punctures in close proximity may result in leakage of infused fluid through one site and out the second (Rosetti, 1985; Parrish, 1986).
 - c. PERIOSTITIS:
 - (1) Slight periostitis that clears in 2 to 3 weeks may be noted at injection site (Massey, 1950; Rosetti, 1985).
 - (2) Roentgenographic follow-up revealed round defects of bone up to 6 weeks after infusion; resolution of all changes occurred 1 year after infusion (Heinild, 1947).
 - (3) Growth plate damage is potential complication but has not been reported (Rosetti, 1985).
 - d. INFUSION FAILURE: Occurred in about 10% of >4200 infusions in adults and children; may result from inability to aspirate marrow secondary to abnormally dense bone with small marrow cavity or marrow replaced by fat (Rosetti, 1985).
 - e. LOCAL ABSCESS/CELLULITIS: Possible complications related to long-term infusion and poor technique (Berg, 1984).
 - f. FAT/BONE MARROW EMBOLISM: Found in 100% of lungs of animals and patients studied; occurrence not related to drug or solution infused; pressurized maintenance into IO needle does not increase incidence of amount of embolization. Not of immediate clinical importance and does not result in fat embolism syndrome or ARDS that might complicate resuscitation, except possibly in patients with intracardiac right-to-left shunt (Orłowski, 1989).
 - g. MEDIASTINITIS/STERNAL PUNCTURE: Mediastinal infection and fatal sternal perforation have occurred with use of the sternum for intraosseous infusion (Turkel, 1954; Quilligan, 1946; Tocantins, 1945); risk eliminated by limiting procedure to tibia or femur.
 - h. BONE MARROW NECROSIS: Reported in association with IO injection of sodium bicarbonate, 50% dextrose in water, lidocaine, and epinephrine (Orłowski, 1989).
 - i. TIBIA FRACTURE: Reported after multiple unsuccessful IO needle insertion attempts in same bone (La Fleche, 1989).
 - j. COMPARTMENT SYNDROME: May result from deep placement of needle through both anterior and posterior cortex, resulting in deposition of fluid in popliteal space, particularly if fluid is infused under pressure (Moscati, 1990).

H. DRUG ADMINISTRATION, ENDOTRACHEAL

1. INDICATIONS: If an endotracheal tube has been placed and there is delay in obtaining venous access, epinephrine, atropine, naloxone, and lidocaine may be administered via the endotracheal route (AHA/ILCOR Guidelines, 2000). Should be used only if intravenous or intraosseous routes are not available due to less reliable drug delivery (AHA/ILCOR Guidelines, 2000; Zaritsky, 1998).
2. RECOMMENDATION (AHA/ILCOR Guidelines, 2000):
 - a. ADULTS: Medications administered via tracheal tube should be given at 2 to 2.5 times the intravenous dose. Medications should be diluted in 10 milliliters of distilled water or normal saline followed by 5 manual ventilations.

b. CHILDREN: During pediatric resuscitation, the recommended dose of epinephrine is 10 times the dose given intravenously; Class IIb recommendation (acceptable, possibly effective). Doses of other drugs should be increased when given via tracheal tube compared with intravenous route. Medications should be diluted in 5 milliliters of normal saline followed by 5 manual ventilations.

I. CARDIAC PACING

1. OVERVIEW

a. INDICATIONS (AHA/ILCOR Guidelines, 2000):

(1) Emergent pacing indicated for patients with serious signs and symptoms related to slow rate. Signs include low BP, shock, pulmonary congestion, congestive heart failure; symptoms include chest pain, shortness of breath, decreased level of consciousness.

(2) Routine use is not indicated in asystolic arrest; however, if performed early, may be used in combination with drug therapy.

b. TYPES:

(1) TRANSCUTANEOUS PACING: Recommended as initial pacing method of choice. Easily applied, easily operated, widely available and noninvasive (AHA/ILCOR Guidelines, 2000).

(2) TRANSVENOUS PACING: Utilizes endocardial stimulation of the right ventricle via an electrode passed through a central vein. Key to successful pacing is placement of the catheter tip into the apex of the right ventricle (AHA/ILCOR Guidelines, 2000).

c. EFFICACY: Studies evaluating emergency pacing have been conflicting but generally not encouraging.

(1) A number of studies report electrical capture in 33% to 77% of patients but return of pulses in only 7% to 8%. In one study, there was a significant improvement in both electrical and mechanical capture rates with intraventricular administration of epinephrine and sodium bicarbonate (White, 1985a).

(2) There are generally no long-term survivors (Tintinalli, 1981; Ornato, 1984; Paris, 1985; Hazard, 1981; Olson, 1985). In three studies there were one, three, and four survivors, respectively (Syverud, 1986; Roberts, 1984; Hazard, 1981).

(3) Although enhanced survival using automatic external cardiac pacing has not been demonstrated, the technique is reliable, easy to use, and free of complications; this suggests that further investigation in the prehospital setting may be warranted (White, 1985b).

(4) TCP should be considered early in cardiac arrest situations (AHA/ILCOR Guidelines, 2000).

Prehospital transcutaneous pacing was associated with generally long pacing times and did not improve outcome in one prospective, controlled trial of 89 patients (Hedges, 1987).

d. COMPLICATIONS:

(1) Ventricular perforation.

(2) Arterial/venous injury at insertion site (transvenous pacemaker).

(3) Air embolism.

(4) Pneumothorax.

(5) Pacemaker wire fracture.

(6) Atrial and ventricular arrhythmias.

(7) Sepsis.

(8) Thromboembolism.

2. CARDIAC PACING, TRANSCUTANEOUS

a. GENERAL: There is no supporting evidence for routine use of temporary transcutaneous pacing (TCP) in asystole. TCP is unlikely to improve prognosis of asystolic patients regardless of early application (Cummins, 1993). To be effective, if at all, TCP should begin early and be done simultaneously with CPR and drugs (AHA/ILCOR Guidelines, 2000).

b. MECHANISM: During transcutaneous pacing, the heart is stimulated by electrical impulse delivered by external electrodes placed on the skin of the chest. The impulse is conducted across the chest wall to activate the myocardium. Used for short intervals as a bridge until transvenous pacing can be initiated or until the underlying cause of the arrhythmia (eg, hyperkalemia, drug overdose) can be corrected (AHA/ILCOR Guidelines, 2000).

c. INDICATIONS (AHA/ILCOR Guidelines, 2000):

(1) Emergent transcutaneous pacing indicated for patients with serious signs and symptoms related to slow rate. Signs include low BP, shock, pulmonary congestion, congestive heart failure; symptoms include chest pain, shortness of breath, decreased level of consciousness; Class I recommendation (acceptable, definitely effective).

(2) Routine use is not indicated in asystolic arrest. However, if performed early, may be used in combination with drug therapy for asystole.

d. COMPLICATIONS (AHA/ILCOR Guidelines, 2000):

- (1) Pain from electrical stimulation of skin and from muscular contractions caused by pacing (requires sedation for the awake patient).
- (2) Failure to capture.
- (3) Failure to recognize ventricular fibrillation because of pacer artifact.
- (4) Induction of ventricular fibrillation or other dysrhythmia.
- (5) Tissue damage from prolonged transcutaneous pacing (including burns in pediatric patients).

3. CARDIAC PACING, TRANSVENOUS

- a. INDICATIONS: Early use is encouraged, less effective once cardiac arrest occurs. Transcutaneous pacing should be used as bridge until transvenous pacing is established (AHA/ILCOR Guidelines, 2000).
- b. MECHANISM: Utilizes endocardial stimulation of the right ventricle via an electrode passed through a central vein. Key to successful pacing is placement of the catheter tip into the apex of the right ventricle (AHA/ILCOR Guidelines, 2000).

J. BYPASS, CARDIOPULMONARY

1. INDICATIONS:

- a. May be instituted using femoral veno-arterial bypass; permits rapid control of circulation and respiratory gas exchange.
- b. May play role in pediatric resuscitation efforts in controlled situations for patients at high-risk for lethal arrhythmias during diagnostic and therapeutic procedures (eg, cardiac catheterization or catheter ablation) (Cochran, 1999).
- c. May be useful in cardiac arrest secondary to potentially reversible condition, eg, hypothermia, drug overdose (AHA/ILCOR Guidelines, 2000).
- d. Further studies are needed to define role in therapy; Class indeterminate (no recommendation until further evidence is available) (AHA/ILCOR Guidelines, 2000).

2. EFFICACY:

- a. Effective with inhospital and hypothermic out-of-hospital cardiac arrest, but CPB for prolonged (CPR >20 minutes) normothermic arrest patients has more disappointing results (Brader, 1989).
- b. Has been used successfully in pediatric patients in cardiac arrest refractory to pediatric advanced life support measures. Successful resuscitation with CPB reported 40 minutes after onset of cardiac arrest in 2 patients (Cochran, 1999).
- c. A prospective, uncontrolled ED study found use of CPB for treatment of refractory cardiac arrest is feasible and effective. Ten patients treated with CPB had return of spontaneous circulation; however, there were no long-term survivors. Additional studies need to address prevention/treatment of reperfusion injury and improving long-term outcomes (Martin, 1998).
- d. A retrospective, 7-year study, involving 21 patients with cardiac arrest, found a overall survival of 36%. Extracorporeal resuscitation was only effective in patients with treatable underlying conditions such as pulmonary embolism (Younger, 1999).

K. PERFUSION, AORTIC ARCH

1. New techniques of resuscitation involving direct access of the aorta followed by occlusion, infusion, or counterpulsation are undergoing laboratory trial (Paradis, 1996).

L. HYPOTHERMIA, INDUCED

1. Preliminary data indicate that, in comatose survivors of out-of-hospital cardiac arrest, moderate hypothermia (33 C) induced by surface cooling in the ED and maintained for 12 h in the ICU improves outcome, with no increase in complications. Further studies are warranted (Bernard, 1997).

M. MONITORING, HEMODYNAMIC

1. OVERVIEW

a. GENERAL (Ornato, 1993):

- (1) Direct measurement of the hemodynamic parameters during CPR requires mechanical equipment that may not be available in the emergency department setting.
- (2) However, assessments of the hemodynamic status that should be available to emergency physicians include blood pressure and central venous pressure monitoring; in special situations, arterial lines and pulmonary artery catheters may be utilized.
- (3) Arterial lines are especially useful for careful monitoring, particularly in the patient with peripheral shut-down.

b. COMPUTERS: Computer-assisted cardiovascular monitoring provides pre-arrest information of heart rate; arterial, venous, and pulmonary pressures; core and peripheral temperatures; urine output; cardiac output; cardiac index; and pulmonary capillary wedge pressures. Also measures myocardial contractility, eg, dP/dT max. However, this type of monitoring can only be available in an intensive care setting where the patients are known to the medical team; even in these patients only limited data may be available in the low output states of cardiopulmonary resuscitation.

c. RIGHT-ATRIAL MONITORING: Placement of right-atrial (RA) catheters in patients during arrest allows venous access and measurement of RA pressures (Paradis, 1989).

2. MONITORING, ARTERIAL PRESSURE

a. Direct measurement of mean arterial blood pressure during CPR has been advocated as a better indicator of the adequacy of closed-chest compression (McDonald, 1981).

b. AORTIC ARCH CATHETERIZATION: An aortic arch (Ao) catheter can be placed via the femoral artery and can provide determination of Ao-radial artery (RA) gradient and serial arterial blood gases. Ao-RA gradient information helps determine optimal therapy based on real-time diagnostic data (Paradis, 1989).

3. MONITORING, CARDIAC OUTPUT

a. Cardiac output determination during CPR with the conventional thermal dilution or dye dilution techniques may be inaccurate in low output states.

N. RESUSCITATION, CEREBRAL

1. OVERVIEW

a. GENERAL:

(1) The increasing effectiveness of CPR has made evident the need for improving cerebral resuscitation as well. The morbidity and mortality associated with brain injury resulting from global ischemia is a growing problem (Knopp, 1980).

(2) Among patients with sudden cardiac death outside of hospitals in whom CPR is started within 4 minutes of collapse, 60% recover with normal brain function, whereas those with longer arrest periods have a recovery rate of only 10%, primarily because of post-ischemic encephalopathy (Safar, 1980).

(3) CPR must provide 20% to 30% of baseline cerebral cortical blood flow to maintain cerebral viability during cardiac arrest (Safar, 1984; Niemann, 1984a).

(4) At present, general recommendations for improving cerebral viability following CPR include the standard methods for CPR to quickly establish a return of carotid blood flow, cerebral perfusion, and the correction of hypoxemia and acid-base imbalance.

b. PATHOPHYSIOLOGY:

(1) MECHANISMS OF CEREBRAL INJURY:

(a) DERANGEMENT OF CELLULAR FUNCTION: Sudden total cessation of brain circulation results in (1) depletion of oxygen stores resulting in unconsciousness within ten seconds; (2) depletion of glucose and glycogen stores and cessation of even low-energy producing anaerobic metabolism within about four minutes; and (3) exhaustion of high-energy phosphate reserves (phosphocreatine), stopping all energy-requiring reactions within about five minutes (Safar, 1978). Large ionic gradients across cell membranes (eg, calcium, sodium, potassium) decay rapidly, and equilibrium may occur within about five minutes of complete ischemic anoxia (Krause, 1986).

(b) MICROVASCULAR DAMAGE: Results in the no-reflow phenomenon and cerebral hypoperfusion following ischemia (Bircher, 1985a).

(c) REPERFUSION: Vulnerable neurons develop disaggregation of polyribosomes, peroxidative damage to unsaturated fatty acids in the plasma membrane, and alterations in the structure of the Golgi apparatus; protein synthesis also is inhibited (White, 1996).

(2) LACTATE:

(a) Although the oxygen supply is depleted almost immediately following cessation of blood flow, glucose is provided initially by glycogen breakdown. As glycolysis occurs there is a build-up of lactic acid, which may be a mediator of cerebral damage.

(b) The effects of acid-base disturbances on the brain during resuscitation are unclear. Ischemic brain injury is exacerbated by persistent cerebral blood flow at rates less than 15% of normal or by preischemic hyperglycemia; this may be a result of lactic acid accumulation secondary to continuing anaerobic metabolism of glucose. Attempts to generate a similar injury experimentally have failed, however, and correction of brain acidosis with agents that cross the blood-brain barrier has also been unsuccessful (Krause, 1986).

(c) In the setting of hyperglycemia, incomplete ischemia may be more detrimental than complete ischemia; in general, however, incomplete ischemia is less detrimental than complete ischemia.

(3) CALCIUM:

(a) With failure of ATP-dependent ionic pumps, there is a loss of cellular potassium and influx of calcium, sodium, and chloride. Accumulation of calcium within the cell appears to be mediated by failure of intracellular and membrane calcium homeostasis and overall increased membrane permeability to calcium (Bircher, 1985a).

(b) Ischemic calcium overloading leads to myofibrillar contracture of arterial walls, activation of phospholipases with liberation of free fatty acids from membrane lipids, and the conversion of xanthine dehydrogenase to xanthine oxidase (the superoxide generating form of the enzyme) (Krause, 1986).

(c) Sequestration of calcium within the mitochondria may occur, leading to uncoupling of oxidative phosphorylation.

(4) REPERFUSION INJURY: Significant tissue damage from cardiac arrest may occur during the period of reperfusion. Several mechanisms appear to be involved, including the no-reflow phenomenon, build-up of lactic acid, continued influx of calcium through membranes damaged during the ischemic period, and production of oxidative free radicals (White, 1996; Babbs, 1985a).

(a) NO-REFLOW PHENOMENON: There is a marked reduction in cerebral blood flow 1 to 3 hours after resuscitation (White, 1983, 1983a). Initially, there is a transient period of hyperemia during which blood flow is greater than normal but unevenly distributed; after 15 to 30 minutes, cellular edema begins to develop. Cerebral blood flow may be about 40% to 50% lower than pre-ischemic values as a result of microcirculatory changes, including capillary sludging, endothelial damage, and vasospasm. The mechanism for this is thought to be calcium dependent (Krause, 1986).

(b) FREE RADICALS: Molecules with unpaired electrons are produced in excess during the reperfusion phase due to the abundance of reducing equivalents such as NADPH, the action of xanthine oxidase and NADPH-cytochrome P-450 reductase, the sudden reappearance of molecular oxygen, and other factors. Common oxygen-derived free radicals include diatomic oxygen, the superoxide radical, the hydroxyl radical, the hydroperoxyl radical, the alkoxy radical, and the peroxy radical (Rogers, 1989). Immediately following reperfusion, superoxide dismutase and other systems that normally destroy superoxide ions are overwhelmed. There is a sharp rise in the concentration of superoxide radicals, with conversion of some to highly deleterious hydroxyl radicals by iron-catalyzed reactions. Hydroxyl radicals attack protein and lipid molecules causing widespread chain reactions that alter cellular architecture. The process may be accelerated by liberation of more free iron ions from ferritin molecules and damage to lysosomal membranes (Babbs, 1985a; Safar, 1985). Calcium loading of the mitochondria liberates mitochondrial iron (Safar, 1985).

(c) LIPID PEROXIDATION: Calcium-triggered phospholipase breaks down membrane phospholipids to free fatty acids; iron may then trigger formation of hydroxyl-induced free lipid radicals which cause irreversible lipid peroxidation. Among the free fatty acids produced by lipid peroxidation of membranes, arachidonic acid accumulation leads to synthesis of prostaglandins (especially thromboxane) and leukotrienes (Safar, 1985). Thromboxane may be responsible for intravascular coagulation, vascular occlusion, and cerebral vasospasm, while endoperoxides increase production of free oxygen radicals, which may lead to tissue damage and vascular paralysis.

(d) IRON: Free iron is required to catalyze lipid peroxidation of membrane Phospholipids and may be released from its normal sites within the cell during ischemia. This may occur secondary to reduction of ferric iron stored in ferritin molecules by superoxide or by interaction of NADH with ferritin to release ferrous iron (Krause, 1986).

(e) Animal models have shown some success in using free radical scavengers, eg, allopurinol, to improve neurologic outcome following ischemic injury (Rogers, 1989). Insulin-class growth factors have been shown to have neuron-sparing effects against damage produced by free radicals, ischemia and reperfusion (White, 1996).

(5) HISTOLOGY:

(a) Two principal cellular changes noted in the brain are ischemic cell change (neurons with shrunken, dark-staining cytoplasm and pyknotic nuclei) and status spongiosus (swelling of perineuronal and perivascular glial elements). There is little correlation between the histopathologic features and the clinical manifestations of ischemia.

(b) Neurons in the hippocampus CA1 and CA4 zones and cortical layers III and V are selectively vulnerable to death after ischemia and reperfusion (White, 1996).

2. ANTICEREBRAL EDEMA AGENTS

- a. MODALITIES: Cerebral edema treatment modalities may include head elevation, osmotic therapy, diuretics, rapid sequence intubation, barbiturates, hyperventilation, mechanical decompression.
- b. CAUTION: Physicians managing cerebral edema must be experienced and knowledgeable in the monitoring techniques and therapeutic modalities used to treat cerebral edema. (FOR FURTHER INFORMATION, SEE CLINICAL REVIEW: INCREASED INTRACRANIAL PRESSURE)

3. CALCIUM ANTAGONISTS

- a. Calcium ion has been implicated as a possible cause in the production of increased cerebrovascular resistance leading to post-resuscitation cerebral hypoperfusion. A number of animal models have shown improved neurologic recovery after cardiac arrest in those animals treated with calcium channel blockers, including flunarizine, lidoflazine, verapamil, magnesium sulfate, nimodipine, and nifedipine (White, 1983, 1982; Winegar, 1983; Wauquier, 1985; Edmonds, 1985; Krause, 1986).
- b. One uncontrolled clinical trial also suggested an improved outcome in those patients treated with the calcium antagonists verapamil and magnesium sulfate (Schwartz, 1985). Nimodipine was shown to increase survival in 19 patients with ventricular fibrillation following out-of-hospital cardiac arrest compared with controls; patients in the nimodipine group also recovered normal consciousness within 24 hours and were discharged home significantly more often than controls (Roine, 1987).
- c. RECOMMENDATION: Although calcium channel blockers are promising drugs (Safar, 1983; Newberg, 1984), their use in cerebral resuscitation is still considered investigational (Rogers, 1989).

4. NEUROTRANSMITTERS

- a. Ischemia may result in massive release of excitatory neurotransmitters that cause neurologic injury independent of the direct damage produced by the initial ischemic phase. N-methyl-D-aspartate (NMDA) receptor blockers are being evaluated for cerebral resuscitation following ischemia (Rogers, 1989).

O. RESUSCITATION, WITNESSED

1. GENERAL:

- a. Defined as presence of relatives during medical resuscitation. Although process is becoming more established in medical practice, it is not in widespread use (Boyd, 2000).
- b. Witnessed resuscitation should be offered to family members whenever possible (AHA/ILCOR Guidelines, 2000; Resuscitation Council, 1996; Wise, 1996). Routine exclusion of relatives may no longer be appropriate (Robinson, 1998).
- c. Additional studies are needed to identify any psychological stressors for healthcare providers involved in witnessed resuscitation (Boyd, 2000).

2. TECHNIQUE (AHA/ILCOR Guidelines, 2000; Goldstein, 1997):

- a. Family members should be given the opportunity to be present during resuscitation efforts. In addition, they should be free to leave resuscitation room, if desired, without feeling uncomfortable.
- b. An experienced team member needs to provide a full explanation to family prior to witnessed resuscitation. In addition, the resuscitation team leader needs to be notified before witnessed resuscitation occurs. If family decides to witness the resuscitation efforts, a designated team member without other resuscitation responsibilities should stay with relatives to address their needs.

3. BENEFITS:

- a. Family members reported satisfaction with their decisions to stay with patient during resuscitation and did not experience any adverse psychological effects as a result of witnessed resuscitation, based upon a pilot study involving 25 relatives of patients undergoing resuscitation (Robinson, 1998).
- b. A poll of 23 pediatric physicians found presence of parents during resuscitation was viewed as a negative experience by 3 physicians. In 2 cases, a nurse did not accompany parents during resuscitation and in 1 case, resuscitation was unnecessarily prolonged due to parents' presence (Goldstein, 1997).

4. CONTROVERSY:

- a. Concerns have been raised regarding the affect of witnessed resuscitation on healthcare providers and relatives (Boyd, 2000; Wise, 1996), although preliminary studies have not demonstrated this (Boyd, 2000).
- b. Medico-legal concerns have also been raised. Witnessed resuscitation by relatives of adult patients may be considered a breach of patient confidentiality (Stewart, 1997). Others disagree, stating humane considerations outweigh the legal and patient confidentiality issues (McLauchlan, 1997).

P. ANGIOPLASTY, CORONARY

1. In survivors of out-of-hospital cardiac arrest who have no obvious noncardiac cause of the arrest, immediate coronary angiography with angioplasty is safe and feasible when performed by an experienced team and may improve long-term outcome (Spaulding, 1997).

Q. ORGAN AND TISSUE DONATION

1. If resuscitation attempts are unsuccessful in preventing death or brain death, the patient may be eligible for organ or tissue donation (AHA/ILCOR Guidelines, 2000).
2. Healthcare providers are required by law to make families aware of the option of organ and tissue donation.
3. Organ procurement organizations can provide assistance with donor identification and management, organ recovery, and allocation (AHA/ILCOR Guidelines, 2000). (FOR FURTHER INFORMATION, SEE CLINICAL REVIEW: ORGAN AND TISSUE DONATION)

6.3 PHARMACOLOGIC TREATMENT

A. VASOPRESSORS

1. VASOPRESSIN

- a. INDICATIONS (AHA/ILCOR Guidelines, 2000):
 - (1) An alternative to epinephrine for treatment of ventricular fibrillation or pulseless ventricular tachycardia refractory to initial shocks; Class IIb recommendation (acceptable, possibly effective).
 - (2) Is NOT currently recommended for treatment of asystole or pulseless electrical activity; Class indeterminate (no recommendation until further evidence is available).
- b. RECOMMENDATION (ADULTS): 40 units intravenously as single, one-time dose (AHA/ILCOR Guidelines, 2000).
- c. AVAILABLE FORMS: Pitressin(R) (injection).
- d. DOSING IN SPECIAL SITUATIONS: Dose adjustment not required in hepatic insufficiency.
- e. MAJOR ADVERSE REACTIONS: Allergic reactions; tremors; bronchospasm; profuse sweating and pounding sensation in head; cardiac arrest.
- f. PRECAUTIONS: Caution in epilepsy, migraine, asthma, CHF, coronary artery disease, chronic nephritis.
- g. MONITORING PARAMETERS: Blood pressure; signs of water intoxication (drowsiness, listlessness, headaches); plasma and urine osmolality; serum sodium.
- h. EFFICACY:
 - (1) May be as effective as (AHA/ILCOR Guidelines, 2000) or possibly superior to (Chugh, 1997; Morris, 1997) epinephrine.
 - (2) In an uncontrolled study of refractory VF, spontaneous circulation was restored in 8 patients following administration of vasopressin, with 3 patients surviving to hospital discharge (Lindner, 1995).
 - (3) Animal studies have shown that vasopressin is more effective than epinephrine in increasing perfusion pressure (Lindner, 1996a), coronary perfusion pressure (Babar, 1999), vital organ flow (Wenzel, 1999), cerebral oxygen delivery (Prengel, 1996), and VF median frequency (Strohmeier, 1996).
- i. MECHANISM OF ACTION: A naturally occurring antidiuretic hormone that at high doses is a powerful, nonadrenergic, peripheral vasoconstrictor. Has positive effects similar to epinephrine but without adverse effects; does not increase myocardial oxygen consumption or cause skeletal muscle vasodilatation (AHA/ILCOR Guidelines, 2000).

2. EPINEPHRINE

- a. INDICATIONS (AHA/ILCOR Guidelines, 2000):
 - (1) VENTRICULAR FIBRILLATION/PULSELESS VENTRICULAR TACHYCARDIA:
 - (a) Employed after failure to respond to initial CPR and shocks; Class IIb recommendation (acceptable, possibly effective).
 - (b) Also may be used for patients unresponsive to vasopressin administration after 10 to 20 minutes; Class indeterminate (no recommendation until further evidence is available).
 - (2) ASYSTOLE: Indicated as soon as an intravenous line has been secured; if the patient has been successfully intubated prior to IV access, may give via the endotracheal route. Class indeterminate (no recommendation until further evidence is available).
 - (3) PULSELESS ELECTRICAL ACTIVITY: Employed after venous access established or endotracheal tube inserted. Increases heart rate and the strength of contraction, and may increase cardiac output. Class indeterminate (no recommendation until further evidence is available).
 - (4) BRADYARRHYTHMIAS: Unresponsive to atropine and transcutaneous pacing; given as intravenous infusion. Class indeterminate (no recommendation until further evidence is available).
- b. RECOMMENDATION (AHA/ILCOR Guidelines, 2000):
 - (1) STANDARD-DOSE:
 - (a) ADULTS: 1 milligram (10 milliliters of 1:10,000 solution) intravenously initially; may repeat every three to five minutes (endotracheal route: 2 to 2.5 milligrams).
 - (b) CHILDREN (INITIAL DOSE): 0.01 milligram/kilogram (0.1 milliliter/kilogram of 1:10,000 solution) intravenously or intraosseously (endotracheal route: 0.1 milligram/kilogram (0.1

- milliliter/kilogram of 1:1000 solution)). If unsuccessful, repeat initial dose every three to five minutes or proceed to high-dose epinephrine (Zaritsky, 1998).
- (2) HIGH-DOSE: Not recommended for routine use, but can be considered if standard dose fails (AHA/ILCOR Guidelines, 2000).
- (a) ADULTS: Up to 0.2 milligram/kilogram intravenous push every three to five minutes. Class indeterminate (no recommendation until further evidence is available).
- (b) CHILDREN (SECOND AND SUBSEQUENT DOSES): May repeat initial dose (0.01 milligram/kilogram), or give high-dose epinephrine of 0.1 milligram/kilogram (0.1 milliliter/kilogram of 1:1000 solution) intravenously, intraosseously, or endotracheally; may be repeated every three to five minutes (Zaritsky, 1998).
- (3) INTRAVENOUS INFUSION (SYMPTOMATIC BRADYCARDIA): 2 to 10 micrograms/minute via continuous intravenous infusion.
- c. AVAILABLE FORMS: Adrenaline(R) (injection).
- d. MAJOR ADVERSE REACTIONS: May contribute to refractory VF; tachycardia; palpitations; hypertension; arrhythmias; pulmonary edema; lactic acidosis, precordial pain; gangrene (with intramuscular injection); syncope (children).
- e. PRECAUTIONS: Contraindicated in narrow-angle glaucoma. Caution in elderly and patients with cardiovascular disease, hypertension, diabetes or hyperthyroidism; increased risk of arrhythmias with digitalis. Caution in patients receiving beta-blockers; the unopposed alpha action may cause severe hypertension.
- f. MONITORING PARAMETERS: Electrocardiogram, blood pressure, heart rate.
- g. MECHANISM OF ACTION:
- (1) Increases heart rate, contractile force, arterial blood pressure, systemic vascular resistance, and automaticity in a perfusing heart but its actions in cardiac arrest are very different. Epinephrine improves coronary artery and cerebral perfusion during CPR (Otto, 1984; Koehler, 1985).
- (2) The alpha-adrenergic actions of epinephrine are thought to produce improved coronary blood flow secondary to peripheral vasoconstriction (Ralston, 1984).
- (a) Peripheral vasoconstriction raises peripheral vascular resistance and increases aortic diastolic pressure. The latter is a major determinant of coronary blood flow. There is a consistent relationship between aortic diastolic pressure, coronary perfusion pressure, and success of resuscitation.
- (b) In canine studies, administration of epinephrine resulted in increased perfusion of the brain in addition to the heart (Ralston, 1984; Schleien, 1986).
- (c) Epinephrine also appears to enhance cerebral oxygen uptake secondary to the increase in cerebral blood flow (Schleien, 1986).
- (3) The value and safety of epinephrine's beta adrenergic effects are controversial. Some authors believe that they increase myocardial work and reduce subendocardial perfusion. Others believe that these effects are beneficial through an increase in inotropy and coarsening ventricular fibrillation.
- h. EFFICACY:
- (1) Data suggest that at current recommended dosages, epinephrine does not primarily convert VF or promote electrical defibrillation.
- (2) Several large randomized trials of high-dose epinephrine (HDE) have failed to confirm a survival benefit for HDE versus standard-dose epinephrine (Gueugniaud, 1998; Brown, 1992; Stiell, 1992; Callahan, 1992; Choux, 1995).
- (3) Secondary analysis of data from the Ontario Trial of Active Compression-Decompression Cardiopulmonary Resuscitation (OTAC), involving 773 patients with in-hospital cardiac arrest, suggest epinephrine does not improve resuscitation outcomes, even after controlling for confounding factors (van Walraven, 1998).
- (4) The European Epinephrine Study, a randomized, prospective study involving over 3300 patients with out-of-hospital cardiac arrest, found no difference in long-term survival in patients treated with repeated high-dose epinephrine versus those treated with repeated standard-dose epinephrine, although return of spontaneous circulation and survival to hospital admission were higher in patients receiving high-dose epinephrine (Gueugniaud, 1998).
- (5) Unfavorable neurologic outcomes reported in patients receiving increasing cumulative doses of epinephrine, based upon data from a retrospective cohort study involving 178 patients. Cumulative epinephrine dose was an independent predictor of poor neurologic outcome even after controlling for duration of arrest (Behringer, 1998). Additional studies are needed to further delineate neurotoxic effects of epinephrine (Behringer, 1998; Cummins, 1998).

3. NOREPINEPHRINE

- a. In a study of 816 adult cardiac arrest patients, norepinephrine produced no additional survival benefit when compared with standard- and high-dose epinephrine (Callahan, 1992).

B. ANTIARRHYTHMICS

1. AMIODARONE

- a. INDICATIONS: Shock-refractory ventricular fibrillation or pulseless ventricular tachycardia; Class IIb recommendation (acceptable, possibly effective) (AHA/ILCOR Guidelines, 2000).
- b. RECOMMENDATION (AHA/ILCOR Guidelines, 2000):
 - (1) ADULTS: 300 milligrams intravenous push. If ventricular fibrillation or pulseless ventricular tachycardia recurs, consider a second dose of 150 milligrams intravenously; maximum dose, 2.2 grams/day.
 - (2) CHILDREN:
 - (a) For pulseless ventricular tachycardia or fibrillation, give 5 milligrams/kilogram rapid intravenous bolus or intraosseous injection.
 - (b) For perfusing tachycardias, give 5 milligrams/kilogram intravenously or intraosseously over 20 to 60 minutes. Maximum dose, 15 milligrams/kilogram/day (AHA/ILCOR Guidelines, 2000). Alternatively, administer 5 milligrams/kilogram loading dose in incremental doses of 1 milligram/kilogram intravenously over 5 to 10 minutes (Perry, 1996).
- c. AVAILABLE FORMS: Cordarone(R) (injection).
- d. DOSING IN SPECIAL SITUATIONS: Safety and efficacy in pediatric patients have not been established (de Vane, 2001). Reduce dose in hepatic insufficiency.
- e. MAJOR ADVERSE REACTIONS:
 - (1) Acute pulmonary toxicity; hypotension; bradycardia; heart block; new ventricular fibrillation; sustained ventricular tachycardia; reduced cardiac contractility, particularly in patients with depressed left ventricular function; QT prolongation; agranulocytosis and hepatitis. Associated with <1% incidence of torsades de pointes.
 - (2) Amiodarone has been found to cause leaching of plasticizers (eg, DEHP) from IV tubing (including PVC tubing) that may adversely affect male reproductive tract development during fetal, infant, and toddler stages of development. In addition, fatal "gasping syndrome" has been reported in neonates (<1 month of age) following amiodarone administration secondary to the benzyl alcohol preservative used in amiodarone (de Vane, 2001).
- f. PRECAUTIONS: Contraindicated in severe sinus-node dysfunction causing significant sinus bradycardia; second- and third-degree AV block. Can elevate digoxin levels greater than 100%; concomitant use with amiodarone can increase blood levels of quinidine, procainamide, and flecainide approximately 30%. Caution with use in pediatrics.
- g. MONITORING PARAMETERS: Amiodarone plasma concentrations (therapeutic range, 1 to 2 micrograms/milliliter); blood pressure; cardiac output; EKG.
- h. MECHANISM OF ACTION: Substantial antiarrhythmic activity secondary to several different electrophysiologic mechanisms. The most important of these appears to consist of a sympatholytic action and for slowing of phase 4 depolarization together with a prolongation of repolarization.
- i. EFFICACY: A randomized, prospective, double-blind, placebo-controlled study involving over 500 patients suffering out-of-hospital VF cardiac arrest initially treated with 3 or more defibrillations found amiodarone significantly improved survival to hospital admission compared with placebo. Trial did not have sufficient power to determine benefit of amiodarone on survival to discharge (Kudenchuk, 1999).

2. PROCAINAMIDE

- a. INDICATIONS: Use in shock-refractory ventricular fibrillation or pulseless ventricular tachycardia is acceptable but not recommended; prolonged administration time unsuitable for cardiac arrest. Class IIb recommendation (acceptable, possibly effective) for intermittent/recurrent ventricular fibrillation or pulseless ventricular tachycardia (AHA/ILCOR Guidelines, 2000).
- b. RECOMMENDATION (ADULTS): 30 milligrams/minute intravenously until arrhythmia is suppressed, QRS widened by 50%, or up to maximum of 17 milligrams/kilogram is given (AHA/ILCOR Guidelines, 2000).
- c. AVAILABLE FORMS: Pronestyl(R) (injection).
- d. DOSING IN SPECIAL SITUATIONS: Increase dosage interval in renal insufficiency.
- e. MAJOR ADVERSE EFFECTS: Blood dyscrasias; hypotension (IV use); SLE; confusion or hallucinations; diarrhea.
- f. PRECAUTIONS: Contraindicated with history of allergy to procaine, complete AV block, myasthenia gravis; caution in digitalis intoxications, organic heart disease, renal or hepatic insufficiency.
- g. MONITORING PARAMETERS: Blood pressure (IV use); ANA and CBC (prolonged use).

3. LIDOCAINE

- a. INDICATIONS: Use in shock-refractory ventricular fibrillation or pulseless ventricular tachycardia; Class indeterminate (no recommendation until further evidence is available) (AHA/ILCOR Guidelines, 2000).
- b. RECOMMENDATION (AHA/ILCOR Guidelines, 2000):
 - (1) ADULTS: 1 to 1.5 milligrams/kilogram intravenous bolus. If no response, may repeat 1.5 milligrams/kilogram intravenous bolus in 3 to 5 minutes to a total of 3 milligrams/kilogram.
 - (2) CHILDREN: 1 milligram/kilogram intravenous or intraosseous infusion for loading dose; maintenance infusion of 20 to 50 micrograms/kilogram/minute.
- c. AVAILABLE FORMS: Xylocaine(R) (injection).
- d. DOSING IN SPECIAL SITUATIONS: Reduce dose in patients with hepatic insufficiency, congestive heart failure, cardiogenic shock, and in patients over 70 years of age.
- e. MAJOR ADVERSE REACTIONS: Neurotoxicity (paresthesias, muscle twitching); convulsions; respiratory depression; coma.
- f. PRECAUTIONS: Use with caution in patients with hepatic insufficiency or congestive heart failure; avoid concurrent use with the skeletal muscle relaxants.
- g. MONITORING PARAMETERS: Electrocardiogram; plasma concentrations.
- h. MECHANISM OF ACTION: A Class IB fast sodium channel blocker. Decreases the duration of the action potential and decreases automaticity. May predispose some patients to reentrant cardiac arrhythmias.
- i. EFFICACY: Secondary analysis of data from the Ontario Trial of Active Compression-Decompression Cardiopulmonary Resuscitation (OTAC), involving 773 patients with inhospital cardiac arrest, suggest lidocaine does not improve resuscitation outcomes, even after controlling for confounding factors (van Walraven, 1998).

4. MAGNESIUM SULFATE

- a. INDICATIONS: Torsades de pointes; suspected hypomagnesemic state (AHA/ILCOR Guidelines, 2000).
- b. RECOMMENDATION (AHA/ILCOR Guidelines, 2000):
 - (1) ADULTS: 1 to 2 grams intravenously over one to two minutes.
 - (2) CHILDREN: 25 milligrams/kilogram intravenously over 10 to 20 minutes.
- c. AVAILABLE FORMS: Magnesium Sulfate Injection(R).
- d. DOSING IN SPECIAL SITUATIONS: Dose reduction required in renal insufficiency.
- e. MAJOR ADVERSE REACTIONS: Magnesium toxicity, especially in renal failure (hypotension, hypothermia, circulatory collapse and respiratory paralysis); calcium gluconate is antidote (10 to 20 mL of 10% solution).
- f. PRECAUTIONS: Contraindicated in heart block and oliguric renal failure; use with extreme caution in renal insufficiency; cardiac conduction abnormalities can occur in digitalized patients if calcium is required to treat magnesium toxicity; CNS depression is accentuated by other depressant-type drugs; excessive neuromuscular blockade with concomitant neuromuscular-blocking agents (succinylcholine).
- g. MONITORING PARAMETERS: Serum magnesium level (toxic, above 4 mEq/L); disappearance of patellar reflex is indicative of toxicity.
- h. MECHANISM OF ACTION: Unknown. Restoration of intracellular potassium via an effect on membrane NA-K-ATPase, blocking effects on slow calcium channels and normalization of membrane repolarization abnormalities secondary to anti-arrhythmic drugs have all been postulated.
- i. EFFICACY:
 - (1) In the 156-patient, randomized, placebo-controlled MAGIC trial, empiric magnesium sulfate failed to improve return of spontaneous circulation or survival in patients with inhospital cardiac arrest (Thel, 1997).
 - (2) Another prospective, randomized, double-blind, placebo-controlled trial found similar results. Magnesium was administered prior to any other drugs in patients with out-of-hospital cardiac arrest; no significant difference in survival noted between groups (Fatovich, 1997).

5. BRETILIUM TOSYLATE

- a. Has been removed from advanced cardiac life support treatment algorithms for several reasons, including availability of safer agents, high incidence of adverse reactions, and limited availability of drug (AHA/ILCOR Guidelines, 2000).

C. ATROPINE

1. INDICATIONS (AHA/ILCOR Guidelines, 2000):

- a. Symptomatic sinus bradycardia; Class I recommendation (acceptable, definitely effective).
- b. AV block at nodal level; Class IIa recommendation (acceptable, probably effective).
- c. Ventricular asystole.
- d. Pulseless electrical activity if rate is slow.

2. RECOMMENDATION (AHA/ILCOR Guidelines, 2000):

a. ASYSTOLE:

(1) ADULTS: 1 milligram intravenously; may repeat every three to five minutes up to a total dose of 0.04 milligram/kilogram (endotracheal route: 1 to 2 milligrams diluted in up to 10 milliliters of normal saline).

(2) CHILDREN: 0.02 milligram/kilogram (minimum dose: 0.1 milligram) intravenously or intraosseously (maximum single dose: child, 0.5 milligram; adolescent, 1 milligram); may repeat once in five minutes (maximum total dose: child, 1 milligram; adolescent, 2 milligrams). May be administered endotracheally in doses two to three times the intravenous dose diluted in 3 to 5 milliliters.

b. BRADYARRHYTHMIAS:

(1) ADULTS: 0.5 to 1 milligram intravenously every three to five minutes until adequate response or until 0.04 milligram/kilogram has been given.

(2) CHILDREN: 0.02 milligram/kilogram (minimum dose: 0.1 milligram) intravenously or intraosseously; maximum single dose: child, 0.5 milligrams; adolescent, 1 milligram. May be repeated once.

c. PULSELESS ELECTRICAL ACTIVITY (ADULTS): If PEA rate is slow, give 1 milligram intravenously; may repeat every three to five minutes up to a total dose of 0.04 milligram/kilogram.

3. AVAILABLE FORMS: Generic preparations (injection).

4. DOSING IN SPECIAL SITUATIONS: Reduce dose in renal failure; dose reductions not required in hepatic insufficiency; reduce dose in elderly patients.

5. MAJOR ADVERSE REACTIONS: CNS toxicity (tremor, delirium, hallucinations); urinary retention; tachycardia; hypotension or hypertension; large doses - CNS depression (antidote: physostigmine).

6. PRECAUTIONS: Contraindicated in glaucoma, adhesions (synechiae) between iris and lens of eye; caution in prostatic hypertrophy, pyloric obstruction, CHF, and bladder neck obstruction.

7. MONITORING PARAMETERS: EKG, vital signs (IV use).

8. EFFICACY: Secondary analysis of data from the Ontario Trial of Active Compression-Decompression Cardiopulmonary Resuscitation (OTAC), involving 773 patients with inhospital cardiac arrest, suggest atropine does not improve resuscitation outcomes, even after controlling for confounding factors (van Walraven, 1998).

D. CALCIUM CHLORIDE

1. INDICATIONS: Hyperkalemia; hypocalcemia; calcium antagonist toxicity (AHA/ILCOR Guidelines, 2000; Zaritsky, 1998).

2. RECOMMENDATION: NOTE: Calcium and bicarbonate may form precipitate and should not be administered together (AHA/ILCOR Guidelines, 2000):

a. ADULTS: 2 to 4 milligrams/kilogram of a 10 percent solution intravenously; may repeat at 10-minute intervals as needed. Should be administered slowly if heart is beating.

b. CHILDREN: 20 milligrams/kilogram (0.2 milliliter/kilogram) of a 10 percent solution intravenously over 10 to 20 seconds during cardiac arrest; may be repeated once after 10 minutes, if needed. For perfusing patients, administer dose over 5 to 10 minutes. Further doses should be based on measured calcium deficiency.

3. AVAILABLE FORMS: Generic preparation available (10% solution).

4. MAJOR ADVERSE REACTIONS: May precipitate refractory asystole digitalis-intoxicated patient. When used in asystole, may decrease the chance of a pacemaker cell initiating conduction because of a decrease in automaticity of Purkinje fibers. Tissue necrosis from extravasation; hypercalcemia (prolonged use); hypotension, bradycardia (rapid IV infusion); avoid IM use.

5. PRECAUTIONS: Caution in digitalized patients (possible arrhythmias); avoid administration with sodium bicarbonate in same IV solution (precipitate).

6. MONITORING PARAMETERS: Continuous cardiac monitoring (bradycardia); serum calcium levels.

7. EFFICACY: Overall, retrospective and prospective studies have failed to demonstrate benefit from the use of calcium in cardiac arrest (AHA/ILCOR Guidelines, 2000). Research on the use of calcium antagonists indicates that the use of calcium is questionable and may be detrimental in the setting of cardiac arrest (Donovan, 1985; Stempien, 1986).

a. PULSELESS ELECTRICAL ACTIVITY (PEA):

(1) May be useful in specific subsets of patients with cardiac arrest, specifically those with PEA (Hughes, 1986; Vincent, 1987). Helpful when PEA secondary to hyperkalemia; should be treated with calcium chloride followed by insulin, glucose, sodium bicarbonate, nebulized albuterol, diuresis (AHA/ILCOR Guidelines, 2000).

(2) A prospective, blinded study comparing calcium with saline in PEA noted that calcium had a significant positive effect on resuscitation rate, although outcome in the form of discharge from the

hospital was not affected. Patients with QRS duration >0.12 seconds showed a response to calcium, while those with normal QRS did not (Stueven, 1985).

b. ASYSTOLE: The use of calcium in asystole is controversial and probably is not effective (Redding, 1983). In a study of 73 patients given either calcium (n=39) or saline (n=34) for asystole, no difference in outcome was noted. In one animal model of countershock-induced asystole, calcium had no effect (Niemann, 1985).

E. THEOPHYLLINE

1. Theophylline antagonizes the bradycardic effect of endogenous adenosine that is released from the myocardium during ischemia.
2. Theophylline promptly restored a stable heart rhythm in 11 of 15 cardiac arrest patients who were refractory to epinephrine and atropine (Viskin, 1993). Confirmatory studies are needed.
3. In a double-blind, placebo-controlled, randomized animal study of VF, the addition of theophylline to standard ACLS interventions did not improve the incidence of return of spontaneous circulation or 1-hour survival (Burton, 1997).

F. THROMBOLYTICS

1. Successful resuscitation following tPA administration has been reported in 3 acute MI patients with witnessed cardiac arrest in ED unresponsive to advanced life support measures; each had refractory ventricular tachycardia or ventricular fibrillation. Findings suggest selective use of thrombolytic therapy in AMI patients following witnessed arrest may be beneficial (Tiffany, 1998).
2. Use of bolus thrombolytics for patients with massive pulmonary embolism requiring CPR has been reported; successful in 10% to 100% of patients. Prompt diagnosis, using patient history, clinical findings, echocardiogram or angiogram, and prompt treatment essential (Kurkciyan, 2000; Bottiger, 1994).
3. Additional studies are needed to define role of thrombolytics in resuscitation efforts (Newman, 2000; Tiffany, 1998; Abu-Laban, 1998). The TPA in PEA study, an ongoing, double-blind, randomized, placebo-controlled study scheduled to include 230 patients, is evaluating the use of TPA in selected cases of cardiac arrest (Abu-Laban, 1998).

G. SODIUM BICARBONATE

1. INDICATIONS: Indications for use during cardiac arrest are extremely limited. May be considered under the following conditions (AHA/ILCOR Guidelines, 2000):
 - a. Known preexisting hyperkalemia.
 - b. Tricyclic antidepressant overdose.
 - c. Patient is intubated and the arrest is prolonged.
 - d. Upon return of spontaneous circulation after prolonged arrest.
2. RECOMMENDATION (AHA/ILCOR Guidelines, 2000):
 - a. ADULTS: If sodium bicarbonate is used, give 1 milliequivalent/kilogram intravenously as the initial dose, titrate subsequent doses according to ABG results.
 - b. CHILDREN: May be considered following establishment of airway, hyperventilation, CPR, and epinephrine administration. If sodium bicarbonate is used, give 1 milliequivalent/kilogram intravenously or intraosseously; titrate subsequent doses based upon ABG results.
3. AVAILABLE FORMS: Sodium bicarbonate (injection).
4. DOSING IN SPECIAL SITUATIONS: Reduce dose in renal insufficiency.
5. MAJOR ADVERSE REACTIONS:
 - a. Inhibition of oxygen release secondary to shift in oxyhemoglobin saturation.
 - b. Induces hyperosmolarity and hypernatremia.
 - c. Produces paradoxical acidosis, which may depress cerebral and cardiac function.
 - d. May cause extracellular alkaloses, which may reduce the concentration of ionized calcium, decrease plasma potassium, induce a left shift on the oxyhemoglobin dissociation curve, and induce malignant arrhythmias.
 - e. May inactivate catecholamines administered simultaneously.
6. PRECAUTIONS: Contraindicated when pH is in the desired range of greater than 7.3, when alkalosis is present, for the treatment of respiratory acidosis.
7. MONITORING PARAMETERS: Arterial blood gases, mixed venous blood gases, or both.
8. EFFICACY: Studies have failed to observe beneficial effects from sodium bicarbonate administration (van Walraven, 1998; Guerci, 1986; Niemann, 1984).

H. CALCIUM ANTAGONISTS

1. INDICATIONS: Although calcium channel blockers are promising drugs (Safar, 1983; Newberg, 1984), their use in cerebral and cardiac resuscitation is considered investigational (Rogers, 1989; Martin, 1984).

2. MECHANISM:

- a. Calcium may play a deleterious role in cerebral ischemic states by its effect on mitochondrial oxygen consumption and cytosol energy production (White, 1980).
- b. Calcium channel blockers counteract this action and may, in turn, improve cerebral blood flow, preserve oxygen consumption, and decrease vascular resistance (Martin, 1984; White, 1982; Edvinson, 1979).
- c. Its effect on cerebral vasospasm has been shown to be highly beneficial.

3. EFFICACY:

- a. A number of animal models have shown improved neurologic recovery after cardiac arrest in those animals treated with calcium channel blockers, including flunarizine, lidoflazine, verapamil, magnesium sulfate, nimodipine, and nifedipine (White, 1983, 1982; Winegar, 1983; Wauquier, 1985; Edmonds, 1985; Krause, 1986).
- b. One uncontrolled clinical trial also suggested an improved outcome in those patients treated with the calcium antagonists verapamil and magnesium sulfate (Schwartz, 1985).
- c. Nimodipine was shown to increase survival in 19 patients with ventricular fibrillation following out-of-hospital cardiac arrest compared with controls; patients in the nimodipine group also recovered normal consciousness within 24 hours and were discharged home significantly more often than controls (Roine, 1987).

I. NARCOTIC ANTAGONISTS

1. NALOXONE

- a. A number of studies have documented that, as a result of stress, endorphins are systemically released and may contribute to shock because of their myocardial depressant action. In one animal model of post-countershock PEA, naloxone seemed to be of benefit in restoring a perfusing rhythm (Rothstein, 1985).

J. STEROIDS

1. Two hundred sixty-two comatose cardiac arrest survivors were treated with no, low, medium, or high doses of glucocorticoids without any improvement of survival or neurologic recovery rates. The routine clinical practice of administering glucocorticoids after global brain ischemia may be associated with complications and cannot be recommended (Jastremski, 1989).

7.0 DISPOSITION

7.2 HOME CRITERIA

- A. All U.S. emergency department patients must be screened, stabilized, and discharged in accordance with the EMTALA (COBRA) law.

7.3 CONSULT CRITERIA

A. ORGAN AND TISSUE DONATION:

1. If resuscitation attempts are unsuccessful in preventing death or brain death, the patient may be eligible for organ or tissue donation.
2. Healthcare providers are required by law to make families aware of the option of organ and tissue donation.
3. Organ procurement organizations can provide assistance with donor identification and management, organ recovery, and allocation. (FOR FURTHER INFORMATION, SEE CLINICAL REVIEW: ORGAN AND TISSUE DONATION)

7.4 TRANSFER CRITERIA

- A. All U.S. emergency department patients must be screened, stabilized, and discharged in accordance with the EMTALA (COBRA) law.

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