

ADVANCED CARDIAC LIFE SUPPORT OVERVIEW

This overview is intended to be used as a study guide in conjunction with the Advanced Cardiac Life Support Textbook. It is based upon “Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care” Supplement to Circulation, Volume 102, Number 8, August 22, 2000 International Consensus on Science

Emergency Cardiac Care includes all responses necessary to deal with sudden and often life-threatening events affecting the cardiovascular, cerebrovascular, and pulmonary systems. It specifically includes:

1. Recognition of early warning signs of heart attack, and stroke, efforts to prevent complications, reassurance of the victim, and prompt availability of monitoring equipment.
2. Provision of immediately BLS at the scene when needed
3. Provision of ACLS at the scene as quickly as possible to defibrillate, if necessary and stabilize the victim before transportation
4. Transfer of the stabilized victim to a hospital where definitive cardiac care can be provided.

The most important link in the ECC system in the community continues to be the LAYPERSON.

CHAIN OF SURVIVAL – the highest potential for survival from cardiac arrest is achieved only when the following sequence of events occurs as rapidly as possible:

1. Recognition of early warning signs
2. Activation of the EMS system
3. Basic CPR
4. Defibrillation
5. Management of the airway and ventilation
6. Intravenous administration of medications

*The need to preserve cerebral viability must be stressed both in research endeavors and in practical interventions. The term *Cardiopulmonary-cerebral resuscitation* has been used to further emphasize this need.

PREVENTION – CPR PARADOX – Fully 50% of men and women in western society with serious coronary artery disease experience their first signs of the disease in a dramatic way – sudden cardiac arrest.

ETHICAL ASPECTS OF CPR AND ECC

The Principle of Futility – Medical treatment is futile if its purpose cannot be achieved. The 2 major determinants of medical futility are LENGTH OF LIFE AND QUALITY OF LIFE. Key factors are the underlying disease before cardiac arrest and expected state of health after resuscitation.

CRITERIA FOR NOT STARTING CPR – scientific evaluation has shown that there are no clear criteria to predict the futility of CPR accurately. Therefore, it is recommended that all patients in cardiac arrest receive resuscitation unless;

- The patient has a valid DNAR order
- The patient has signs of irreversible death: rigor mortis, decapitation, or dependent lividity
- No physiological benefit can be expected because the vital functions have deteriorated despite maximal therapy for such conditions as progressive septic or cardiogenic shock
- Withholding attempts to resuscitate in the delivery room is appropriate for newly born infants with confirmed gestation <23 weeks or birth weight <400g, anencephaly, confirmed trisomy 13 or 18

CRITERIA FOR TERMINATING RESUSCITATIVE EFFORTS – In the hospital the decision to terminate resuscitative efforts rests with the treating physician. The most important factor associated with poor outcome is time of resuscitative efforts.

The responsible clinician should stop the resuscitative effort when he or she determines with a high degree of certainty that the arrest victim will not respond to further ACLS efforts.

Available scientific studies have shown that, in the absence of mitigating factors, prolonged resuscitative efforts for adults and children are unlikely to be successful and can be discontinued if there is NO RETURN OF SPONTANEOUS CIRCULATION AT ANY TIME DURING 30 MINUTES OF CUMULATIVE ACLS. If return of spontaneous circulation of any duration occurs at any time, however, it may be appropriate to consider extending the resuscitative effort. Other issues such as drug overdose and severe prearrest hypothermia should be considered when determining whether to extend resuscitative efforts.

DO NOT RESUSCITATE ORDERS

- In the United States a physician's order is necessary to withhold CPR
- The scope of a DNAR order may be ambiguous. DNAR order does not preclude interventions such as administration of parenteral fluids nutrition, oxygen, analgesia, sedation, antiarrhythmics or vasopressors.
- The order can be written for an individual in a specific clinical situation.
- Carry no inherent implications for limiting other forms of treatment. Admitting a patient with a DNAR order to an intensive care unit is

consistent with the attitude that all patients deserve the best available care, regardless of the existence of a DNAR order.

ETHICAL ISSUES AROUND OUT-OF-HOSPITAL RESUSCITATION

Healthcare professionals are expected to provide BLS and ACLS as part of their professional duty to respond. There are, however several exceptions:

1. When a person lies dead, with obvious clinical signs of irreversible death
2. When attempts to perform CPR would place the rescuer at risk of physical injury
3. When the patient or surrogate has indicated that resuscitation is not desired.

Quality of life should never be used as a criterion to withhold CPR, conditions such as irreversible brain damage or brain death cannot be reliably assessed or predicted.

In the United States, EMS protocols must recognize and plan for adults and children who have advance directives limiting resuscitation. Such out-of-hospital DNAR protocols must be clear to physicians, patients, family members and loved ones, and prehospital healthcare professionals.

HONORING OUT-OF-HOSPITAL DNAR ORDERS

CPR and ACLS should be initiated if there is good reason to believe that;

- A DNAR order is invalid
- That the patient may have changes his/her mind
- The interest of the patient is in questions

Out of hospital DNAR orders should not be confused with advance directives or living wills. Advance directives and living wills require interpretation by a physician and need to be formulated into the treatment plan with specific orders for resuscitation that are consistent with the patient's wishes. An inappropriate out-of-hospital resuscitation (i.e. intubation) can be corrected by responsible physicians in the ED or the hospital. *"Withholding resuscitative efforts at the initial collapse is ethically and morally equivalent to withdrawing resuscitative efforts at the terminal event."*

TERMINATING EFFORTS:

A number of studies over the past decade observe that less than 1% of patients transported with continuing CPR survive to hospital discharge. The new ACLS asystole algorithm has been modified to bring the issues of termination of resuscitation to the forefront.

The asystole algorithm focuses on the following questions:

Asystole Persists

- Time to terminate resuscitative efforts?
- Are all BLS/ACLS interventions completed?
- Has asystole persisted for several minutes?
- Consider opposing family attitudes toward stopping efforts.

Clinical Features that Change Predictive Accuracy

- Young age
- Toxins or electrolyte abnormalities
- Profound hypothermia
- Drug overdose

FAMILY PRESENCE DURING RESUSCITATION ATTEMPTS

American Heart Association now recommends that family members be asked whether they wish to be in the same room with the patient during the resuscitation attempts.

Accompanied by a calm, experienced social worker or nurse, families can view the professional efforts made by medical personnel attempting to save their loved one. Afterward they rarely ask the recurring question was everything done??

Retrospective reports on these efforts note positive reactions from family members – many of whom said that they felt a sense of having helped their loved one and of easing their own grieving process. When surveyed before they had observed resuscitative efforts and a loved one's death, the majority of family members stated a preference for being present during resuscitation. Family members who were present during resuscitative efforts reported that it helped them to adjust to the death of their loved one and most indicated they would do so again. Standard psychological exams suggest that family members who were present during efforts demonstrated less anxiety and depression and more constructive grief behavior than family members who were not present. HEALTHCARE PROVIDERS SHOULD OFFER THE OPPORTUNITY TO FAMILY MEMBERS WHENEVER POSSIBLE.

ORGAN AND TISSUE DONATION – PREHOSPITAL

EMS agencies should consider contacting the organ procurement organization in their region to discuss the need for tissue from donors pronounced dead in the field.

ADULT BASIC LIFE SUPPORT

BLS ROLE IN STROKE AND ACUTE CORONARY SYNDROME MANAGEMENT

1. Rescuers should “phone first” for unresponsive adults. Exceptions: “phone fast” (provide CPR first) for adult victims of submersion, trauma and drug intoxication.
2. Prehospital BLS providers should identify possible stroke victims and provide rapid transport and pre-arrival notification to the receiving hospital to increase the likelihood of their eligibility for IV fibrinolytic therapy.
3. Patients with suspected stroke merit the same priorities for dispatch as patients with acute myocardial infarction or major trauma.
4. Victims of suspected ischemic stroke (with pre-arrival notification) should be transported to a facility capable of initiating fibrinolytic therapy within 1 hour of arrival unless that facility is >30 minutes away by ground ambulance.

BLS RESPONSE TO CARDIOPULMONARY EMERGENCIES:

Early CPR prevents VF from deteriorating to asystole, may increase the chance of defibrillation, contributes to preservation of heart and brain function and significantly improves survival. Early defibrillation is the single greatest determinant of survival for adult victims of cardiac arrest.

When the initial ECG is obtained, most adults with sudden nontraumatic cardiac arrest are found to be in VF. *Time from collapse to defibrillation is the single greatest determinant of survival.* Survival declines by 7-10% for each minute without defibrillation. More than 12 minutes after arrest and arrest survival rate is only 2-5%.

Most arrests in infants or children are related to airway or ventilation problems rather than sudden cardiac arrest. In these victims rescue support (especially rescue breathing) is essential and should be attempted first, before activation of the EMS system.

In Adults “phone first” except:

- Submersion/near drowning
- Arrest associated with trauma
- Drug overdoses

In a single study of dispatcher assisted CPR, chest compression only bystander CPR was associated with survival equivalent to chest compressions plus ventilation for victims of witnessed arrest. For reasons of simplicity and elimination of barriers to action, we recommend chest compression only CPR for use in DISPATCHER ASSISTED CPR instructions to untrained bystanders.

RECOGNITION AND ACTIONS FOR ACUTE CORONARY SYNDROMES

Of all patients with ACS, approximately half will die before reaching the hospital. Of patients who reach the hospital an additional 25% will die within the first year. In 17% of patients, ischemic pain is the first, last and only symptom.

Early diagnosis, treatment of AMI significantly reduces mortality, decreases infarct size, improves regional and global left ventricular function and decreases the incidence of resultant heart failure.

ACTION STEPS FOR LAY RESCUERS INCLUDE:

1. Recognize the signs and symptoms of ACS
2. Have the victim sit or lie down
3. If discomfort persists for >5 minutes, activate the EMS
4. After activating the EMS system, give the victim supportive care including rest, reassurance and use of the recovery position.

PREHOSPITAL MANAGEMENT:

Nitroglycerin is effective for relief of symptoms and it dilates coronary arteries and reduces ventricular preload and oxygen requirements. If the patient with chest pain has nitroglycerin and his systolic blood pressure is >90mmHg, the BLS ambulance provider can assist the patient taking NTG. The patient can take up to 3 NTG tablets at intervals of 3-5 minutes. After administration of each NTG monitor blood pressure closely for signs of hypotension.

If local protocol permits, the BLS ambulance provider should administer Aspirin 160-325mg enroute. ASA inhibits coronary re-occlusion and recurrent events after fibrinolytic therapy and reduces mortality in ACS. Routine out of hospital administration of NTG and ASA by BLS ambulance providers is expected to reduce morbidity and mortality from AMI.

ALS provider should administer MONA (morphine, oxygen, nitroglycerin and aspirin.) 12 Lead ECG's are recommended and significantly reduces time to treatment.

RECOGNITION OF STROKE AND ACTIONS FOR PATIENTS WITH SUSPECTED STROKE

Stroke is a leading cause of death and brain injury in adults. Now fibrinolytic therapy offers the opportunity to limit neurological insult and improve outcome in ischemic stroke.

PRESENTATION OF STROKE:

It is impossible to distinguish between a TIA and a stroke at the time of onset. If the symptoms resolve within 24 hours the event is classified as a TIA. TIA's are a significant indicator of stroke risk. Approximately one fourth of patients presenting with stroke have had a previous TIA.

Although both ischemic and hemorrhagic stroke can be life threatening, ischemic stroke rarely leads to death within the first hour.

STROKE MANAGEMENT:

Can be remembered by use of the mnemonic of the 7 D's

1. DETECTION
2. DISPATCH
3. DELIVERY
4. DOOR
5. DATA
6. DECISION
7. DRUG

LAYPERSON BLS CARE FOR STROKE

1. Rapid access of the EMS system is essential as soon as signs and symptoms of a stroke appear.
2. Delays in transport and initial hospital evaluation occur if the victim or family contacts the family physician or transports the stroke victim by private care to the hospital
3. Currently in the US only one half of stroke victims use the EMS system for transport to the hospital
4. 85% of strokes occur at home

PREHOSPITAL CARE:

1. Selection of a receiving hospital capable of administering fibrinolytic therapy.
2. Should require same high dispatch treatment and transport priorities for as for patients with trauma and AMI.

Airway compromise is relatively common after stroke. Cardiac arrest is relatively uncommon, although many stroke victims demonstrate arrhythmias including ventricular tachycardia and atrial fibrillation in the first hours and days after stroke.

TREATMENT:

1. Priority dispatch and response
2. Initial assessment and management, including support of airway, oxygenation, ventilation and circulation.
3. Rapid identification of stroke using standardized stroke scale
4. Rapid transport to a stroke center capable of delivering fibrinolytics within 1 hour of arrival.
5. Pre-arrival notification of the hospital

BLS ambulance providers should establish the time of onset of signs and symptoms of stroke; this timing has important implications for potential therapy. The onset of symptoms is viewed as the beginning of the stroke, and eligibility for fibrinolytic therapy ends 3 hours from that time.

The abbreviated out of hospital neurological examination should include a validated tool such as the Cincinnati Prehospital Stroke Scale.

CINCINNATI PREHOSPITAL STROKE SCALE

1. FACIAL DROOP (have patient show teeth or smile)

- Normal – both sides of the face move equally
- Abnormal – one side of the face does not move as well as the other side

2. ARM DRIFT (patient closes eyes and hold both arms straight out for 10 seconds)

- Normal – both arms move the same or both arms do not move at all
- Abnormal – one arm does not move or one arm drifts down compared with the other

3. ABNORMAL SPEECH (have the patient say “you can’t teach an old dog new tricks”)

- Normal – patient uses correct words with no slurring
- Abnormal – patient slurs words, uses the wrong words, or is unable to speak

Interpretation: If any of these 3 signs is abnormal, the probability of a stroke is 72%.

Once the diagnosis of stroke is suspected, time in the field should be minimized and the patient prepared for immediate transport to a stroke center.

RESUSCITATION SEQUENCE

No victim should undergo the more intrusive procedures of CPR (positioning, opening the airway, rescue breathing, or chest compressions) until need has been established by the appropriate assessment.

1. **ASSESS RESPONSIVENESS** – After determining that the scene is safe, the rescuer arriving at the side of the collapsed victim must quickly assess any injury and determine whether the person is responsive.
2. **ACTIVATE THE EMS SYSTEM** by calling the appropriate local emergency response system telephone number. The person who calls the EMS system should be prepared to give the following information:
 - Location of the emergency
 - Telephone number from which the call is being made
 - What happened: heart attack, auto crash, etc
 - Number of persons who need help
 - Condition of the victim
 - What aid is being given to the victim
 - Any other information requested. To ensure that EMS personnel have no more questions, the caller should hang up only when instructed to do so by the EMD
3. **AIRWAY** – if the victim is unresponsive, the rescuer will need to determine whether the victim is breathing adequately. To assess breathing, the victim should be supine with an open airway.
4. **POSITION THE VICTIM** – supine on a firm, flat surface. Rescuer should be at the victim's side.
5. **OPEN THE AIRWAY** – if no evidence of head or neck trauma use the head tilt-chin lift; remove any visible foreign material or vomitus from the airway.
6. **BREATHING** – place your ear near the victim's mouth and nose while maintaining an open airway. Look for chest rise and fall; Listen for air escaping during exhalation and feel for the flow of air. This evaluation should take no more than 10 seconds.
 - If you are not confident that respirations are adequate, proceed immediately with rescue breathing. Lay rescuers are taught to provide rescue breathing if “normal” breathing is absent.
7. **RECOVERY POSITION** – is used for unresponsive, but breathing patients with a pulse.
 - The victim should be as near a true lateral position as possible, with the head dependent to allow free drainage of fluid.
 - The position should be stable
 - Avoid any pressure on the chest that impairs breathing

- It should be possible to turn the victim on his or her side and to return to the back easily and safely, with concern for a possible cervical spinal injury.
 - Good observation of and access to the airway should be possible.
 - The position itself should not cause an injury to the victim.
8. PROVIDE RESCUE BREATHING – BE PREPARED TO DELIVER APPROXIMATELY 10-12 BREATHS PER MINUTE – approximately 1 breath every 4-5 seconds. The number of breaths delivered differs throughout the world and there is no data to suggest that one is better than another.
- To reduce the risk of gastric inflation, deliver slow breaths at the lowest tidal volume that causes the chest to rise.
 - Mouth to nose breathing may be the best way of providing ventilation while rescuing a victim submerged in water.
9. BAG VALVE MASKS – most common method of delivering ventilation. BVM's are most effective when 2 trained rescuers work together, one sealing the mask and the other squeezing the bag.
- Cricoid pressure is effective in preventing gastric inflation, reducing the risk of regurgitation and aspiration.
10. CIRCULATION – When laypersons use the pulse check, they require a long time to decide and are only 65% accurate. Therefore the *lay rescuer should not rely on the pulse check to determine the need for chest compressions. The pulse check will no longer be taught in layperson CPR courses. Instead they will be taught to look for normal signs of circulation including normal breathing, coughing or movement in response to rescue breathing. Healthcare professionals should continue to utilize the pulse check prior to compressions.*
11. CHEST COMPRESSIONS – a rate of approximately 100 per minute will result in delivery of fewer than 100 compressions by the single rescuer who must interrupt chest compressions to delivery rescue breaths. Compressions should be at a ratio of 15 to 2 for 1 or 2 rescuers until the airway is secured. Once the airway is secured with a cuffed tracheal tube, compressions may be continuous and ventilations may be asynchronous with a ratio of 5:1.
- The heel of the hand is placed on the lower ½ of the sternum
12. COMPRESSION ONLY CPR – mouth-to-mouth rescue breathing is a safe and effective technique that has saved many lives. However if a person is unwilling or unable to perform mouth-to-mouth for an adult victim, chest compression only CPR should be provided rather than no attempt at CPR being made. Current evidence indicates that the outcome of chest compression without mouth-to-mouth ventilation is significantly better than no COPR at all in the setting of adult cardiac arrest.

FOREIGN BODY AIRWAY OBSTRUCTION

Heimlich Maneuver with Responsive Victim Standing or sitting

Stand behind the victim, wrap your arms around the victim's waist, and proceed. Make a fist with one hand. Place the thumb side of your fist against the victim's abdomen, in the midline slightly above the navel and well below the tip of the xiphoid process. Grasp the fist with your other hand and press the fist into the victim's abdomen with a quick inward and thrust. Repeat the thrusts until the object is expelled from the airway or the victim becomes unresponsive. Each new thrust should be a separate and distinct movement administered with the intent of relieving the obstruction. The Heimlich maneuver is repeated until the object is expelled or the victim becomes unresponsive. When the victim becomes unresponsive, the EMS system should be activated, and the lay rescuer will attempt CPR. The healthcare provider will proceed with the sequence of actions to relieve FBAO in the unconscious victim.

Chest thrusts may be used as an alternative to the Heimlich maneuver when the victim is in the late stages of pregnancy or is markedly obese.

Healthcare providers only in the unresponsive/unconscious victim with complete FBAO should use the finger sweep. This sweep should be performed if the victim is responsive or is having seizures.

AUTOMATED EXTERNAL DEFIBRILLATOR

The following are major guideline changes related to use of the automated external defibrillators in Basic Life Support:

1. Early defibrillation within 5 minutes of EMS call receipt is a high priority goal.
2. Healthcare providers with a duty to perform CPR should be trained, equipped and authorized to perform defibrillation.
3. For in hospital defibrillation, early defib should take place <3 minutes in all areas of the hospital and ambulatory care facilities.
4. Evidence supports establishment of public access defibrillation programs in the following cases:
 - Reasonable probability of one AED use in 5 years.
 - An EMS Call to Shock time interval of < 5 minutes cannot be reliably achieved with conventional EMS services
5. Use of AED in children is not recommended
6. Biphasic defibrillation has equivalent or higher efficacy for termination of VF or VT compared with higher energy escalating monophasic waveform defibrillators

AED is the greatest advance in the treatment of VF cardiac arrest since the development of CPR. Extraordinary survival rates as high as 49% have been reported with AED programs. With the inclusion of AED use as a BLS skill, BLS now encompasses the first 3 links in the Chain of Survival.

EARLY DEFIBRILLATION is important for a number of reasons:

1. The most frequent initial rhythm in witnessed sudden cardiac arrest is VF
2. The most effective treatment for VF is electrical defibrillation
3. The probability of successful defibrillation diminishes rapidly over time
4. VF tends to convert to asystole within a few minutes

The performance of CPR while awaiting the arrival of the AED appears to prolong VF contributing to preservation of heart and brain function. Basic CPR however, is unlikely to convert VF to a normal rhythm.

SPECIAL AED SITUATIONS:

WATER – There is a small possibility that rescuers or bystanders may receive shocks or minor burns if they are exposed to water. Water on the skin of the chest can also provide a direct path of energy from one electrode pad to the other and can decrease the effectiveness of the shock delivery to the heart. Quickly remove the victim from freestanding water and dry the victim's chest before using the AED.

CHILDREN – Cardiac arrest is less common in children than adults. AED is not recommended for use in infants and children.

TRANSDERMAL MEDICATIONS – AED electrodes should not be placed directly on top of the transdermal medication patch. The patch may block delivery of energy from the electrode pad to the heart and may cause small burns to the skin. Medication patches should be removed and the area wiped clean before the AED electrode pad is attached.

IMPLANTED PACEMAKERS/ICD'S – Placement of an AED electrode pad directly over an implanted medical device may reduce the effectiveness of defibrillation attempts. Instead, place the pad at least 1 inch away from the implanted device.

OPERATION OF THE AED

1. Place the victim supine.
2. Power ON the AED
3. Attach electrode pads
4. Analyze the rhythm
5. Clear the victim and press the SHOCK button

AED's can be left in place during transport, but never use the analyze button while the patient is in transport, because the movement of the ambulance can interfere with rhythm assessment and artifact can simulate VF.

See Algorithm in Appendix

AED TRAINING Public Access Defibrillation (PAD) implies expanded use of AED's in the community to the broadest possible number of rescuers while maintaining safety and effectiveness.

LEVEL ONE – Nontraditional Responders – persons other than healthcare personnel, such as police, firefighters, security personnel, sports marshals, ski patrol members, ferryboat crews, and flight attendants, whose job duties require them to respond in an emergency.

LEVEL TWO – Targeted Responders – worksite responders who may be called “citizen responders”. Include employees of companies, corporations or public facilities with established PAD programs.

LEVEL THREE – Responders to Persons at High Risk – family members and friends living with or visiting persons at high risk for cardiac emergencies.

The PAD program planners should attempt to coordinate PAD programs with the Local EMS system.

SKILL MAINTENANCE:

Survey results and experience in rural communities have demonstrated that emergency responders may go several years without treating a patient in cardiac arrest. Every program director must determine how to ensure correct performance. At present many programs provide practice drills every 3 to 6 months and have found this interval satisfactory.

NEW GUIDELINES FOR FIRST AID

Unintentional injury continues to be a major cause of morbidity and mortality. It is the 5th leading cause of death in the United States. 26. Million people hospitalized and 34.9 million seen in emergency departments.

SPECIFIC EVIDENCE BASED GUIDELINES:

THERMAL BURNS To treat a thermal burn, remove the victim from the source of injury as soon as possible. Immediately cool the burn with cold – but not ice cold- water. Immediate cooling of burns with cold water is supported by a large number of observational clinical studies. Cooling of burns has many beneficial effects.

- Pain relief
- Reduced formation of Edema
- Reduced infection rates

- Reduced depth of injury
- More rapid healing, reduced need for grafting
- Reduced mortality.

Optimal healing and the lowest mortality rates were noted with water temperatures of 68 to 77 degrees. Excessive cooling with ice water (32 degrees) resulted in hypothermia and increased mortality rates. Cooling should continue at least until pain is relieved and probably for a total duration of 15-30 minutes. Cooling should not delay transportation to a medical facility.

Remove all non-adhering clothing and jewelry that can be removed without force from burn area.

Leave blisters intact.

Cover the burn with a clean dressing if available.

Do not apply lotions, creams, ointments or home remedies to the burn area.

ELECTRICUTION AND ELECTRICAL BURNS:

52,000 trauma admissions per year are due to electrical injuries.

Cardiopulmonary arrest is the primary cause of immediate death in persons who have sustained an electrical injury. Cardiac arrhythmias, including ventricular fibrillation, ventricular asystole and ventricular tachycardia that progress to ventricular fibrillation may occur as a result of exposure to low or high voltage current. Respiratory arrest may result from electrical injury to the respiratory center in the brain or from titanic contractions or paralysis of the respiratory muscles.

Factors that determine the nature and severity of injury include the magnitude of energy delivered, voltage, resistance to current flow, type of current, duration of contact, and current pathway. High-tension current causes the most serious injuries, but fatal electrocutions may also occur with low-voltage household current. Skin resistance, the most important factor impeding current flow, can be reduced substantially by moisture, converting a low voltage injury into a life threatening one.

Once the power is off, assess the victim, who may need CPR, defibrillation, and treatment for shock and thermal burns. Appropriate precautions must be taken because musculoskeletal and spinal cord injuries may be present. All victims of electric shock require medical assessment.

POISONING

Can be caused by solids, liquids, gases, and vapors. Ingestion may be unintentional or self-inflicted. The telephone number of the local poison control center should be prominently displayed at home or worksites where poisonous substances are present.

Do not enter any area where victims are unconscious without knowledge of the agents to which the victims have been exposed and without the required protective equipment.

If the poison is a gas or vapor, remove the victim from the area. If the victim's skin has been exposed, thoroughly flush it with running water until EMS personnel arrive.

Evaluate the victim for adequacy of airway, breathing and circulation and provide basic life support. Place symptomatic victims who are breathing spontaneously in the recovery position. Regardless of symptoms, transport all victims who ingest a poison in a suicide attempt to the nearest Emergency Department. **DO NOT ADMINISTER ANYTHING BY MOUTH UNLESS ADVISED BY A POISON CONTROL CENTER.**

At this time there is insufficient data to support or exclude administration of ipecac to induce vomiting. The potential danger of aspiration and the lack of clear-cut evidence of a benefit support our recommendation: do not administer ipecac unless specifically directed by a poison control center or other authority.

Administration of activated charcoal by first aid rescuers is not recommended.

HEMORRHAGE

First aid responders have a responsibility to protect themselves and must understand and practice protection against blood-borne diseases. Consider all body fluids from victims to be infectious.

After the hemorrhage is controlled, wash your hands thoroughly and change blood soaked clothing.

Nosebleeds can be treated by having the victim bend forward at the waist and pinch the nasal alae with the thumb and index finger.

Active bleeding – apply direct pressure with the flat portion of your fingers or the palm of your hand over a sterile dressing or clean pad. If the bleeding does not stop, apply more pressure. If the dressing becomes saturated, apply a second dressing over the first. If a barrier is unavailable and the victim is conscious have the victim apply pressure directly to the bleeding source.

Extremity bleeding – elevate the extremity. If severe bleeding continues despite application of firm pressure, add arterial pressure by applying pressure to the brachial artery if upper extremity and femoral artery if bleeding is from the lower extremity.

Arterial tourniquets however cause injury as a result of ischemia after 90 minutes. Complications include bleeding, injury to soft tissues, nerve and vascular injury, and paralysis. Tourniquets applied by first aid providers usually cause venous rather than arterial occlusion and often increase rather

than hemorrhage. They should be used only as a last resort for massive hemorrhage that is not controlled by other methods and only by persons skilled in their use.

ALTERED MENTAL STATUS

Signs and symptoms of an altered mental state include loss of consciousness, confusion, combativeness, disorientation, headache, inability to move a body part, dizziness, problems with balance and double vision. Any sudden change in level of consciousness requires medical evaluation.

First aid measures include removing the victim from a potentially dangerous environment; evaluating airway, breathing, and circulation; maintaining body temperature; and placing the victim in a recovery position. If the victim is known to have diabetes and is able to swallow, give him or her a drink contained glucose.

Fainting is a momentary loss of consciousness. Minor pain, sudden fright, or standing in one position for prolonged periods, especially in a hot environment, are precipitating factors in susceptible persons. First aid measures include protecting the victim from injury, placing victim in supine position, checking airway, breathing and circulation. The victim usually regains consciousness within a few seconds and has no alteration in mental status once consciousness is regained.

HEAD TRAUMA

Head injury should be suspected when any of the following has occurred:

- The victim fell from a height greater than his or her own
- When found the victim was unconscious
- The victim sustained a blunt force injury (from impact with or ejection from a car)
- The victim's injury was caused by diving, lightning strike or electrocution, or the victim's head protection or helmet was broken or insufficient
- The victim sustained a high impact sports injury.

First aid responders should gather information on the mechanism of injury whether an alteration in mental status has occurred, and the presence and duration of unconsciousness.

A concussion is an alteration in mental status, especially confusion and amnesia, and may or may not include a loss of consciousness. Because the signs and symptoms may be transient, the first aid responder's observations at the scene provide EMS personnel with important information.

TREATMENT:

1. Determine whether the victim's location poses a danger to the victim or you, and remove the victim from the site if it does.
2. Assess and provide CPR to an unconscious, non-breathing, pulseless patient.
3. Assess the victim's risk of vomiting and ability to protect the airway
4. Assess and control bleeding
5. Maintain the victim's body temperature
6. Stabilize the cervical spine in high risk situations

SPINAL CORD INJURIES AND CERVICAL SPINE IMMOBILIZATION

An overwhelming majority of spinal cord injuries occur during the primary traumatic event. Current practice incorporates the evaluation of specific pain, distribution of tenderness, neurological deficits, and mechanisms of injury to assess the risk of spinal and spinal cord injuries.

First aid responders should suspect an unstable spinal cord with any of the following:

- Injury was caused by a force sufficient to result in loss of consciousness
- Injury occurred on the upper part of the body, especially the head and neck
- Injury resulted in altered mental status
- There is evidence of drug or alcohol abuse.

If spinal cord injury is suspected, do not allow the patient to move in any direction. Immobilize the victim's head, neck and trunk.

SEIZURES

10% of all people will have a seizure during their lifetime and 1-2% will have recurrent seizures. Although seizures are rarely fatal, injuries related to seizures are relatively common.

The general principles of first aid management of seizures are:

1. Prevention of injury
2. Assurance of an open airway
3. Reassurance of an open airway after the seizure ended.

Do not restrain the victim during a seizure or place an object in the victim's mouth. To prevent aspiration of secretions, place the seizure victim in a recovery position as soon as possible after the seizure has stopped.

After a seizure it is not unusual for the victim to be unresponsive or confused for a short time. Activate the EMS system if:

1. A seizure lasts more than 5 minutes or is recurrent
2. The victim exhibits any respiratory problems
3. The victim has sustained an injury
4. Unresponsiveness or confusion lasts more than 5 minutes after the seizure has stopped.

Musculoskeletal Trauma

Closed soft-tissue injuries include joint sprains and muscle contusions. The basic principle in first aid for soft tissue injuries is to decrease hemorrhage, edema and pain. Application of ice is effective for reducing pain and duration of disability. The best way to apply ice is to use a plastic bag. Refreezable packs of gelled solutions are inefficient. To prevent cold injury to the skin it is best to limit application of ice to 20 minutes at a time.

Assume that any injury to an extremity includes a bone fracture. Cover open wounds with a sterile dressing if available. Stabilize the extremity, but do not straighten it if it is deformed. If a deformed extremity appears blue and there is no distal pulse, this is a critical emergency.

OVERVIEW OF RECOMMENDED CHANGES IN ACLS FROM GUIDELINES 2000

The high level of participation of international experts changes profoundly the way all future resuscitation guidelines will be developed.

PHARMACOLOGY OF RESUSCITATION

- **AMIODARONE** (Class IIb) and **procainamide** (Class IIb recommended ahead of Lidocaine and adenosine for the initial treatment of hemodynamically stable wide-complex tachycardia, especially in patients with compromised cardiac function.
- **AMIODARONE** and **sotalol** (not approved in US) are new agents recommended at Class IIa agents for the treatment of stable monomorphic and polymorphism ventricular tachycardia.
- **BRETYLIUM** References to Bretylium have been dropped from the ventricular fibrillation algorithm. The world's natural sources of Bretylium appear to be nearly exhausted. Bretylium remains acceptable to use but it is no longer recommended. High incidence of side effects, particularly hypotension in the post resuscitation setting.
- **LIDOCAINE** agent that suffered during new emphasis on evidence. Efficacy is poor and methodologically weak. The evidence supporting amiodarone is much stronger and justifies use of amiodarone before Lidocaine in the opinion of many. The conference experts concluded that Lidocaine could continue to be used for VF/VT but that given the

antiquated evidence, it merits only an indeterminate class of recommendation.

- **MAGNESIUM** has shown effectiveness only in the treatment of known hypomagnesaemia states and torsades de points.
- **VASOPRESSIN (arginine vasopressin)** may be a more effective pressor agent than epinephrine for promoting the return of spontaneous circulation in cardiac arrest. Vasopressin (40U IV, not repeated) may be substituted for epinephrine as an alternative. The lower adverse effects profile may be the major indication for Vasopressin.
- **HIGH DOSE EPINEPHRINE** - has not shown that routine use of initial and repeated or escalating doses of Epinephrine can improve survival in cardiac arrest. Some troublesome evidence indicates that cardiac arrest survivors who received high dose epinephrine have more post resuscitation complications than survivors who received the standard dose. Because of the potential for harm, high dose epinephrine is not recommended.

VENTILATION

- The experts recommend a reduction in the ventilation tidal volume for patients not in cardiovascular collapse to approximately one half of that recommended previously. Higher volumes increase risk of gastric inflation without improving blood oxygenation.
- **TRACHEAL INTUBATION** should be attempted only by healthcare providers experienced in performing this skill. Only personnel with advanced life support training and documented skills should attempt tracheal intubation. ALS providers unable to obtain regular field experience (non-evidence based guidelines: 6-12 times per year) should use alternative, noninvasive techniques for airway management.
- In the absence of a bag mask device or authorization to perform tracheal intubation, healthcare providers may use alternative airways (laryngeal mask airway, esophageal tracheal Combitube, pharyngotracheal lumen airway).
- **Confirm Tracheal Tube Position** – by using nonphysical examination techniques. **THIS IS THE MOST IMPORTANT NEW RECOMMENDATION FROM GUIDELINES 2000.** These include **esophageal detector devices, qualitative end tidal CO2 indicators, and capnographic and capnometric devices.**
- **TUBE DISLODGE MENT** – after a successful tracheal tube insertion may be occurring at much higher rates than previously suspected. During long transport efforts in the out of hospital setting, restless intubated patients can be fitted with a cervical collar and immobilized with sandbags or some other validated technique to prevent accidental tube dislodgement.

DEFIBRILLATION

- Healthcare providers with a duty to perform CPR need to be trained, equipped and authorized to use an AED
- Hospitals need to establish a comprehensive program for in hospital early CPR and early defibrillation.
- Hospitals need to establish programs to achieve early defibrillation throughout the facilities and in related patient care areas.
- Public access defibrillation programs have the potential to reduce one of the major health problems VF induced cardiac arrest.
- AED's are recommended for public sites with a high probability of at least one use every 5 years.

ACUTE CORONARY SYNDROMES

- **The prehospital 12-Lead ECG** improves prehospital diagnosis, reduces hospital-based time to treatment, identifies patients requiring reperfusion, contributes to mortality reduction and facilitates triage to cardiac centers with interventional facilities.
- **Prehospital fibrinolytic therapy** is beneficial when the transport of patients with acute infarction from home to the hospital is prolonged and should be considered by busy EMS systems. If the total time exceeds 60 minutes, consider prehospital fibrinolytics.
- **Angioplasty is an alternative to fibrinolytic therapy** in centers with high volume and experienced operators. Patients in cardiogenic shock who are <75 years of age need transportation to cardiac interventional centers for initiation of primary angioplasty and intra aortic balloon placement. Benefit occurs, however, only when door to balloon times average <90 minutes. Patients who are not eligible for fibrinolytic therapy should be transferred to these centers. Patients with large anterior infarctions, low blood pressure (<100mmHg), increased heart rate or rales more than one third of the was up
- **Ib/IIIa Inhibitors** for patients with non-Q wave infarction and high-risk unstable angina provides clinically significant benefit.
- **ASA, Beta Blockers** all patients with Acute MI including non-Q wave infarction need ASA and Beta Blockers in the absence of contraindications. Patients with a large anterior infarction, left ventricular dysfunction and ejection fraction <40% need early angiotensin-converting enzyme inhibition in the absence of hypotension.

STROKE

- **IV Recombinant Tissue Plasminogen Activator (rtPA)** improves neurological outcome when administered within 3 hours of stroke onset. The urgency should equal that of an acute MI, with ST segment elevation.

- The use of rtPA in patients with symptoms onset between 3 and 6 hours of presentation at an emergency department is under investigation. While subgroups of such patients may benefit from fibrinolytic treatment, routine use is not currently recommended.
- **Prourokinase** – has been found to improve neurological outcome in patients treated within 3 to 6 hours in one completed but unpublished study.
- **EMS systems should implement a prehospital stroke protocol** to rapidly identify patients who may benefit from fibrinolytic therapy.

POST RESUSCITATION CARE

- Following cardiac arrest, do not actively rewarm patients who are mildly hypothermic.
- Ventilatory values in patients who require mechanical ventilation should be maintained within the normal range. Hyperventilation may be harmful and should be avoided.

TOXICOLOGY

- **COCAINE** use is associated with serious ventricular arrhythmias and acute coronary syndromes. The use of Beta-blockers in patients with cocaine associated acute coronary syndromes has caused coronary vasoconstriction and should be avoided. Nitrates should be first line therapy together with benzodiazepines.
- **Tricyclic Overdose** – Hypotension or ventricular arrhythmias occur. The induction of systemic alkalosis is the treatment of choice. Antiarrhythmic agents such as Lidocaine or procainamide have not been studied in this setting.
- **Opiate Overdose** – Acute respiratory failure and hypoxemia occur. Reverse these by mechanical ventilation before administration of naloxone. This will reduce the incidence of pulmonary edema and serious arrhythmias associated with abrupt catecholamine elevation.

Overview of ACLS

ACLS includes:

1. Basic life support
2. Use of Advanced equipment special techniques for establishing and maintaining effective ventilation and circulation
3. ECG monitoring 12-Lead interpretation and arrhythmia recognition
4. Establishment and maintenance of Intravenous access
5. Therapies for the treatment of patients with cardiac or respiratory arrest including stabilization in the post resuscitation phase.
6. Treatment of patients with suspected acute coronary syndromes, including acute MI
7. Strategies for rapid assessment and treatment with rtPA of eligible stroke patients.

ACLS includes the knowledge, training, and judgment required to use these skills and the ability to perform them.

DEFIBRILLATION

Survival to hospital discharge is directly and negatively related to the time interval between onset of VF and the first shock. All healthcare providers with a duty to perform CPR should be trained, equipped and authorized to perform defibrillation. The use of defibrillation now transcends both ACLS and BLS care.

ENERGY REQUIREMENTS: Defibrillation depends on the successful selection of energy to generate sufficient current flow through the heart to achieve defibrillation while at the same time causing minimal electrical injury to the heart. A shock will not terminate the arrhythmia if the energy current is too low. Functional and morphological damage may result if energy and current are too high. There is no definite relationship between body size and energy requirements for defibrillation in adults. Transthoracic impedance does play an important role.

SHOCK ENERGIES The traditional recommended energy for the first monophasic shock is 200J. The energy level for second and third shocks can be either the same (200J) or as high as 360J. Even a failed shock at one energy may be successful if simply repeated. Any given energy has a constant probability to achieve defibrillation. Repeated shocks even at the same energy level add to the probability of successful defibrillation.

Higher current flow will occur with subsequent shocks even at the same energy, because transthoracic impedance declines with repeated shocks. A more predictable increase in current occurs when shock energy is increased. This supports second shocks of high energy. A compromise between these positions is the use of a range of energies (200-300J). Increase the current and deliver a third shock of 360J immediately if 2 monophasic shocks fail to defibrillate the heart. If VF is initially terminated by a shock but then recurs later in the arrest, deliver subsequent shocks at the previously successful energy level.

CARDIOVERSION

- **Atrial Fibrillation** recommended initial energy for cardioversion is 100-200 J. Atrial flutter and paroxysmal Supraventricular tachycardia generally require less energy. An initial energy of 50 – 100 Joules
- **Ventricular Tachycardia** The amount of energy required for VT depends on the morphological characteristics and rate of the arrhythmia. Monomorphic VT (regular form and rate) with or without a pulse responds well to cardioversion shocks at initial energies of 100J. Polymorphic VT (irregular morphology and rate) responds similarly to VF. The initial shock should be 200J

Transthoracic Impedance:

Factors that determine transthoracic impedance include energy selected, electrode size, paddle skin coupling material, number and time interval of previous shocks, phase of ventilation, distance between electrodes and paddle electrode pressure.

Electrode Position The standard placement is one electrode just to the right of the upper sternal border below the clavicle. The second electrode to the left of the nipple with the center of the electrode in the midaxillary line.

An acceptable alternative is to place the apex paddle anterior, over the left precordium and the other paddle posterior to the heart in the right infracapular location.

Synchronized Cardioversion is recommended for hemodynamically stable wide complex tachycardia requiring cardioversion, Supraventricular tachycardia, atrial fibrillation and atrial flutter. The VT patient who is pulseless, unconscious, hypotensive, or in severe pulmonary edema should receive unsynchronized shocks to avoid the delay associated with attempts to synchronize.

Asystole There is no evidence that attempting to defibrillate asystole is beneficial. Examine the rhythm in 2 leads to help differentiate technical artifact.

ADJUNCTS FOR OXYGENATION, VENTILATION AND AIRWAY CONTROL

Oxygenation Devices: Rescue breathing using exhaled air will deliver approximately 16 to 17%. During cardiac arrest and CPR tissue hypoxia occurs because of low cardiac output with reduced peripheral oxygen delivery and a resulting wide arteriovenous oxygen difference.

In patients with acute MI, supplemental oxygen reduces both the magnitude and the extent of ST segment changes on the ECG. Oxygen should be administered at 4L via nasal cannula for the first 2-3 hours for all patients with suspected acute coronary syndromes. The use of oxygen beyond 3-6 hours is indicated for patients with continuing or recurrent ischemia, complicated infarcts with congestive heart failure, or arrhythmia until hypoxemia has resolved.

Masks: For mouth to mask ventilation we recommend masks equipped with a 1 way valve that diverts the victim's exhaled gas. Mouth to mask ventilation has been shown to be superior to that with bag valve mask devices in delivering adequate tidal volumes on manikins. The efficacy of face shields has not been compared with that of other devices and at this time face shields are recommended for BLS lay rescuers only.

Bag Valve Devices Most commercially available adult bag mask units have a volume of approximately 1600ml. The volume is much greater than currently recommended tidal volumes for COPR. When the airway is unsecured, as with a mask versus a tracheal tube, the possibility of over ventilation with gastric inflation, subsequent regurgitation, and aspiration becomes a significant concern. An oral airway should be inserted and if possible the head elevated if no concern for neck injury exists. Slow gentle ventilation minimizes the risk of gastric inflation. A satisfactory bag valve unit should have:

1. A self refilling bag
2. A non jam valve system allowing for a maximum oxygen inlet flow of 30L/min
3. A no pop off valve
4. Standard 15/22mm fittings
5. A system for delivering high concentrations of oxygen through an ancillary oxygen reservoir
6. A true non-rebreathing valve
7. The capability to perform satisfactorily under all common environmental conditions

Automatic Transport Ventilators

Studies have revealed that ATV are as effective as other devices used in prehospital care in intubated patients. ATV's are considered to provide advantages over alternative methods of ventilation:

- In intubated patients they free the rescuer for other tasks
- In unintubated patients the rescuer has both hands free for mask application and airway maintenance
- Cricoid pressure can be applied with one hand while the other seals the mask
- Once set, they provide a specific tidal volume, respiration rate and minute ventilation.

Studies have observed improved lung inflation or absent gastric inflation when ATVs were compared with other devices including mouth to mask, bag valve mask and manually triggered devices. This is due to the lower Inspiratory flow and longer inspiratory times.

Oxygen Powered, Manually Triggered Devices

Oxygen powered manually triggered devices have been used in prehospital care for >25 years despite a paucity of high level scientific evidence supporting their use. Oxygen powered manually triggered devices are not recommended at this time.

AIRWAY ADJUNCTS

- **Oropharyngeal Airways** – should be reserved for obtunded unconscious patients who are not intubated. Incorrect insertion can displace the

tongue into the hypopharynx and insertion can displace the tongue into the hypopharynx and result in airway obstruction.

- **Nasopharyngeal Airways** – Especially useful in patients with trismus, biting, clenched jaws or maxillofacial injuries. They should be used with caution in patients with suspected fracture at the base of the skull.
- **Alternative Airways** – Alternative airways include the laryngeal mask airway (LMA), the esophageal-tracheal Combitube (ETC) and the pharyngotracheal lumen airway (PTL) to achieve good outcomes with these devices, healthcare providers must maintain a high level of knowledge and skills through frequent practice and field use.
- **Esophageal-Tracheal Combitube** is an invasive double-lumen airway with 2 inflatable balloon cuffs that is inserted without visualization of the vocal cords. Assessment of the location of the distal orifice is then made and the patient is ventilated through the appropriate opening. The advantages of the ETC over the face mask are similar to those of the tracheal tube over the face mask.
 - Isolation of the airway
 - Reduction of risk of aspiration
 - More reliable ventilation
- **Tracheal Intubation** – in the absence of a protected airway, providing adequate lung inflations may require pharyngeal pressures sufficient to cause gastric inflation, subsequent regurgitation and the potential for aspiration.
 - Proceeded by preoxygenation of at least 3 minutes.
 - Ventilation adjunct of choice because it keeps the airway patent, permits suctioning, ensures delivery of high concentration of oxygen, provides a route for meds, facilitates delivery of a selected tidal volume, protects airway from aspiration.

Unless initial training is sufficient and ongoing practice and experience are adequate, fatal complications may occur. Rates for failure to intubate are as high as 50% in EMS systems with low patient volume and providers who do not perform intubation frequently. Those who perform tracheal intubation require either frequent experience or frequent retraining. EMS systems should keep record for each provider documenting the number of intubations performed and success rates.

- **Confirmation of Accurate Placement of Tracheal Tube – Primary Confirmation**
 - As the bag is squeezed, listen over the epigastrium and observe the chest wall for movement
 - If the chest wall rises appropriately and stomach gurgling is not heard, listen to the lung fields; left and right anterior, left and right midaxillary and once again of the stomach.

- If there is continue doubt about correct tube placement, use the laryngoscope and look directly to see whether the tube is passing through the vocal cords.
- If tube seems to be in place, reconfirm the tube mark at the front teeth (this was noted after the tube was inserted 1 to 2 Apply cardiac monitor and pulse oximetry and record baseline strip. Past the vocal cords)

DEVICES TO ASSIST CIRCULATION

Compared with standard CPR, CPR adjuncts generally require additional personnel, training or equipment. The added effort may increase forward blood flow during CPR from 20-100% - levels that are still significantly less than normal cardiac output. Maximum benefits are reported when adjuncts are begun early in the treatment of cardiac arrest.

- **IAC CPR** includes manual compression of the abdomen by an extra rescuer during the relaxation phase of chest compression. Two randomized clinical trials for in hospital cardiac arrest have shown statistically significant improvement in outcome measures. No survival benefit for out of hospital arrest has been shown.
- **High Frequency Rapid Compression Rate CPR** – Compressions >100minute have been advocated as a technique for improving resuscitation from cardiac arrest. Some studies but not all show that rapid compression rates improve cardiac output, aortic and myocardial perfusion pressures, coronary blood flow and 24 hour survival. Shows some promise for improving CPR. Outcome studies in humans are needed to determine the efficacy of this technique in the management of patients in cardiac arrest.
- **ACD CPR** Decreasing intrathoracic pressure during the decompression phase of CPR is thought to enhance venous return and thereby prime the pump for the next compression. It is performed with a hand held device equipped with a suction cup to actively lift the anterior chest during compression. Early laboratory and clinical data show that acute hemodynamic parameters such as arterial blood pressure and vital organ perfusion are superior with use of ACD compared with standard CPR. There is some concern that the extra force and energy applied to the chest wall during ACD CPR tends to induce higher incidence of rib fractures than that with standard CPR.
- **Mechanical Piston CPR** Mechanical devices that depress the sternum are not a substitute for manual external chest compression but rather an adjunct to be used by trained personnel to optimize compression and reduce rescuer fatigue in prolonged resuscitative efforts. A disadvantage of any mechanical chest compression device is the potential for interrupting chest compression for extended periods while setting up and initiating compressions.

PHARMACOLOGY – AGENTS FOR ARRHYTHMIAS

MONITORING AND ARRHYTHMIA RECOGNITION

For patients with acute myocardial infarction or severe ischemia, the greatest risk for serious arrhythmias occurs during the first hour after the start of symptoms.

Interpret all ECG and rhythm information within the context of total patient assessment. Full diagnosis requires assessment of the patient's metabolic and acid base status. In specific clinical settings consider the possibility of proarrhythmic drug effects, adverse drug effects from intentional or unintentional overdose or drug toxicity occurring with normal dosing patterns. After initial training ACLS providers require regular updates in their rhythm expertise combined with evaluation sessions.

Professionals at the ACLS level should be able to recognize the following arrhythmias:

- Sinus Bradycardia
- Atrioventricular blocks of all degrees
- Premature atrial complexes
- Supraventricular tachycardias
- Preexcited arrhythmias (associated with an accessory pathway)
- Premature ventricular complexes
- Ventricular tachycardia
- Ventricular Fibrillation
- Ventricular Asystole

Most wide-complex tachycardias are ventricular in origin. If a patient is pulseless, in shock or congestive heart failure, such rhythm should always be assumed to be ventricular.

Administration of Medications During Cardiac Arrest – Correct Priorities

On close evidence review it is recognized that few drugs are supported by strong evidence. Therefore during cardiac arrest, the administration of drugs is secondary to other interventions.

Central Venous vs. Peripheral Infusions

A peripheral vein (antecubital or external jugular) should be the first choice. Peak drug concentrations are lower and circulation times are longer when drugs are administered via peripheral sites compared with central sites. When given via a peripheral vein drugs require 1 to 2 minutes to reach the central circulation but the delay is appreciably shorter with a central venous route.

If peripheral access is used, administer IV drugs rapidly by bolus injection; follow with a 20cc bolus of IV fluid and elevate the extremity for 10-20 seconds.

Avoid attempts at central line placement in patients who are candidates for pharmacological reperfusion.

Tracheal Drug Administration – If the tracheal tube has been placed before venous access, epinephrine, Lidocaine and atropine can be administered via the tracheal tube. Doses should be 2-2.5 times the recommended IV dose.

Pass a catheter beyond the tip of the tracheal tube, stop chest compressions, spray the drug solution quickly down the tube, and follow immediately with several quick insufflations to create a rapidly absorbed aerosol, then resume chest compressions.

Hemodynamically Stable Wide Complex Tachycardias

The criteria for hemodynamically stable wide complex tachycardias are:

1. Regular tachycardia at a rate greater than the upper limit of sinus tachycardia at rest (>120/min)
2. Uniform (Monomorphic) QRS configuration of >120ms in duration
3. No signs or symptoms of impaired consciousness or tissue hypoperfusion.

The patient must be stable enough to allow time for rhythm diagnosis or transport to a facility more capable of diagnosing the rhythm. The drugs used for most tachycardias lower BP. Therefore patients should have blood pressures high enough to permit use of these drugs. Otherwise the drug induced hypotension will require immediate cardioversion to end the abnormal rhythm.

TREATMENT

Focused on the need to establish a rhythm diagnosis before initiating treatment in stable patients. When circumstances and expertise allow, ACLS providers should make a reasonable attempt to distinguish hemodynamically stable VT from SVT with aberrancy. A history of coronary artery disease or other structural heart disease suggests ventricular in origin. A history of previous aberrant rhythms, accessory pathways, preexisting bundle-branch block, or rate-dependent bundle-branch blocks suggests SVT with aberrancy if the QRS matches that observed with the tachycardia.

12-Lead ECG Always obtain a 12 Lead ECG before and during pharmacological interventions and after conversion to a regular rhythm. If the 12 lead is not diagnostic, an esophageal lead may be helpful if the equipment and experts who can interpret tracings are available.

A carefully evaluated 12 lead permits identification of AV dissociation. The loss of a 1 to 1 relationship between atrial activity and ventricular response is a highly specific criteria for VT.

When initial care provides state whether a wide complex tachycardia is ventricular or Supraventricular in origin, they are wrong in more than 50% of cases. Their most frequent error is to evaluate VT and then misdiagnose it as SVT

LIDOCAINE

Evidence does not support the use of Lidocaine to discriminate between perfusing VT and SVT of uncertain origin. Lidocaine will effectively suppress ventricular arrhythmias associated with AMI and ischemia once they occur. The prophylactic use of Lidocaine causes higher mortality and has been abandoned.

Studies suggest that Lidocaine is ineffective for termination of hemodynamically stable VT and have found Lidocaine to be less effective than IV procainamide or Sotalol. Lidocaine is acceptable but other drugs are preferred over Lidocaine in each VT scenario.

ADENOSINE

The principle therapeutic effect of adenosine is to slow AV nodal conduction. Not effective agent for common forms of ventricular arrhythmias for pre-excited atrial arrhythmias such as atrial fibrillation or atrial flutter. Adenosine is used for narrow complex tachycardia only.

PROCAINAMIDE have shown efficacy in treating a broad variety of arrhythmias including SVT with and without aberrancy and VT. Procainamide is effective at terminating SVT because of its ability to alter conduction across an accessory pathway.

AMIODARONE – is also effective for SVTs because it alters conduction through the accessory pathway. Amiodarone becomes the Antiarrhythmic of choice after failure of adenosine if the patient's cardiac function is impaired and the ejection fraction is <40% or there are signs of congestive heart failure. Effective in treating hemodynamically unstable VT and VF. Both procainamide and amiodarone have vasodilatory effects and negative inotropic properties, which can destabilize hemodynamic status.

PROARRHYTHMIC ANTIARRHYTHMICS

Proarrhythmias are serious tachyarrhythmias or bradyarrhythmias seemingly generated by Antiarrhythmic agents. All Antiarrhythmic agents have some degree of proarrhythmic effects. The rhythm called torsades de pointes accounts for the majority of tachycardic proarrhythmic episodes. Sequential use of 2 or more antiarrhythmic drugs compounds the adverse effects, particularly for Bradycardia, hypotension and torsades de pointes. Never use more than 1 agent unless absolutely necessary. In most patients when an appropriate dose of a single Antiarrhythmic medication fails to terminate an arrhythmia, turn to electrical cardioversion rather than a second medication.

Amiodarone and Lidocaine cause the least additional impairment of LV function. Because of its broad Antiarrhythmic spectrum and lesser negative inotropic effect, amiodarone now dominates the management of tachycardias.

Hemodynamically Stable Monomorphic VT

Consider VT stable if there are no symptoms or clinical evidence of tissue hypoperfusion or shock. Guidelines now recommend treatment of hemodynamically stable VT with IV procainamide, IV Sotalol, IV Amiodarone or IV Beta Blockade. Each of these is preferable to IV Lidocaine.

Although Lidocaine can be administered rapidly with minimal effect on blood pressure studies suggest that it is relatively ineffective for termination of VT and less effective against VT than other medications.

Amiodarone is the agent of first choice.

POLYMORPHIC VT

VT with varying QRS morphology. It is usually irregular in rate, hemodynamically unstable and likely to quickly degenerate to VF. It is often associated with ischemic heart events or electrolyte or toxic conditions. A unique form of Polymorphic VT is called torsades de pointes, which usually occurs in a setting of Bradycardia and a prolongation of the QT interval. Hemodynamically unstable Polymorphic VT should be treated using the VF/Pulseless VT algorithm. Effective Antiarrhythmic agents include Amiodarone, Lidocaine, and Procainamide.

VENTRICULAR FIBRILLATION/PULSELESS VT

It is reasonable to add pharmacological therapy after at least three shocks, delivered in rapid sequence fail to restore a stable perfusing rhythm. Patients with shock refractory arrhythmias should be considered for pharmacological therapies sooner rather than later, the likelihood of benefit declines rapidly with the duration of arrest.

Once the IV line is placed, vasopressors (epinephrine or vasopressin) are administered following by another attempt to defibrillate. Shocks must not be delayed until IV line is established and medications have been delivered.

A randomized comparison between amiodarone and Lidocaine found a greater likelihood of successful resuscitation with amiodarone.

Procainamide administration in cardiac arrest is limited by the need for slow infusion and uncertain efficacy in emergent circumstances.

Evidence supports the use of IV amiodarone following epinephrine to treat shock refractory cardiac arrest due to VF or pulseless VT. Amiodarone restores spontaneous circulation and improved early survival to the hospital in adults. However, no pharmacological interventions for cardiac arrest have yet been

found to improve survival to hospital discharge. AS CURRENTLY AVAILABLE AMIODARONE MUST BE DRAWN UP FROM A 6ML GLASS AMPULE INTO A SYRINGE AND THEN DILUTED TO 20CC BEFORE INJECTION. PRELOADED SYRINGES ARE NOT AVAILABLE BECAUSE AMIODARONE ADHERES TO THE PLASTIC SURFACE OF PLASTIC SYRINGES.

PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA

Is a regular tachycardia exceeding the expected limits of sinus tachycardia (>120) at rest with or without discernible PZ waves that is usually of abrupt onset and abrupt termination.

Initial use of vagal maneuvers and IV adenosine in all patients without contraindications with PSVT continues to be recommended. Adenosine can provoke bronchospasm and should be used cautiously in patients with reactive airway disease. Cardiac denervation after transplantation may render patients hypersensitive to the bradycardic effects of adenosine.

In patients with preserved LV function calcium channel blockers (Verapamil, diltiazem) and beta blockers (esmolol, metoprolol) remain supported by previous evidence. Amiodarone, propafenone, flecainide and sotalol in the presence or absence of an accessory pathway are effective.

In the setting of significantly impaired LV function, caution should be exercised in administering drugs with negative inotropic effects.

ATRIAL TACHYCARDIA (ECTOPIC ATRIAL TACHYCARDIA, MAT)

Ectopic atrial tachycardia is distinguished from sinus tachycardia on the 12 Lead by presence of an abnormal P wave configuration and P waves axis. It is important to distinguish MAT from AF, because both can result in an irregularly irregular rhythm. MAT is distinguished from AF by the presence of P waves having 3 or more different morphologies preceding QRS complex.

Digitalis may be effective in slowing heart rate but not in terminating ectopic arrhythmias. Digitalis also has been associated with provoking ectopic atrial tachycardia. Other useful drugs for ectopic atrial tachycardia or MAT include amiodarone, flecainide, and propafenone.

MANAGEMENT PRINCIPLES FOR ATRIAL FIBRILLATION/FLUTTER

Hemodynamically unstable rapid response atrial fibrillation or flutter should be electrically cardioverted immediately, regardless of the duration of the arrhythmia.

Pharmacological rate control is the recommended initial treatment for stable rapid atrial fibrillation/flutter (>120/min) regardless of its duration. Specific drug treatment depends on the presence or absence of impaired LV function.

- Adenosine is an inappropriate drug to use for atrial fibrillation/flutter because of its ultra short duration of action. (Class III)
- In patients with preserved LV function, beta-blockers, calcium blockers and digitalis are reasonable agents for rate control.
- In patients with congestive heart failure, digitalis, diltiazem, and amiodarone are recommended.
- In patients with pre excited atrial fibrillation/flutter, beta-blockers, calcium channel blockers, and digitalis are contraindicated. If electrical cardioversion is not feasible, desirable, or successful, such patients with preserved LV function may be treated with procainamide or amiodarone IV flecainide, propafenone or sotalol (not available in all countries)
- Efforts should be made to minimize the risk of thromboembolic complications that are strongly related to the duration of the arrhythmia before cardioversion (>48hours)
- Electrical cardioversion is the preferred treatment for restoration of sinus rhythm
- Pharmacological cardioversion is recommended if electrical cardioversion is not feasible or desirable or is unsuccessful in maintaining sinus rhythm.

ANTIARRHYTHMIC DRUGS AND THE ARRHYTHMIAS THEY TREAT

ADENOSINE – is an endogenous purine nucleoside that depresses AV node and sinus node activity. Most common forms of PSVT involve a reentry pathway including the AV node. Adenosine is effective in terminating these arrhythmias.

- Produces a short-lived pharmacological response because it is rapidly metabolized.
- Half life is less than 5 seconds
- Dose is 6mg IV fast bolus followed by 20cc saline bolus
- Follow with 12mg IV fast bolus followed by 20cc saline bolus

More likely to precipitate persistent hypotension if the arrhythmia is not terminated. **Use of Adenosine to discriminate VT from SVT with aberrancy in hemodynamically stable wide complex tachycardia of uncertain origin is controversial, and such a practice should be discouraged. Adenosine should be used only when a Supraventricular origin is strongly suspected.**

AMIODARONE (IV) – is a complex drug with effects on sodium, potassium and calcium channels as well as alpha and beta-adrenergic blocking properties. The drug is useful for treatment atrial and ventricular arrhythmias.

- Amiodarone is also helpful for ventricular rate control of rapid atrial arrhythmias in patients with severely impaired LV function when digitalis has proved ineffective

- Recommended after defibrillation and epinephrine in cardiac arrest with persistent VT or VF
- Effective for control of hemodynamically stable VT
- Adjunct to electrical cardioversion refractory PSVT's, atrial tachycardias
- Control rapid ventricular rate due to accessory pathway conduction

In patients with severely impaired heart function IV amiodarone is preferable to other Antiarrhythmic agents for atrial and ventricular arrhythmias. Has greater efficacy and lower incidence of proarrhythmic effects than other Antiarrhythmic drugs under similar circumstances. Administered at 150mg over 10 minutes, followed by 1mg/min infusion for 6 hours and then repeated as necessary for recurrent or resistant arrhythmias to a maximum total daily dose of 2 grams.

In cardiac arrest due to pulseless VT or VF. Iv amiodarone is initially administered as a 300mg rapid infusion diluted in a volume of 20-30 cc of saline or dextrose in water.

The major adverse effects are hypotension and Bradycardia, which can be prevented by slowing the rate of drug infusion or can be treated with fluids, pressors, chronotropic agents or temporary pacing.

ATROPINE – reverses cholinergic mediated decreases in heart rate, systemic vascular resistance, and blood pressure.

- Useful in treating symptomatic sinus Bradycardia.
- Beneficial in the presence of AV block at the nodal level or ventricular asystole
- Should not be used when infranodal (Mobitz Type II) block is present.

DOSE: for PEA or Asystole is 1.0mg and repeated in 3 to 5 minutes to a total dose of 0.04 mg/kg. A total dose of 3mg results in full vagal blockage in humans. Vagolytic dose of atropine should be reserved for asystolic cardiac arrest.

Atropine is well absorbed through the tracheal route of administration.

Atropine should be used cautiously in presence of AMI or infarction because excessive increases in rate may worsen ischemia or increase the zone of infarction.

BETA ADRENERGIC BLOCKERS – have potential benefits in patients with acute coronary syndromes, including patients with non-Q wave MI and unstable angina. In the absence of contraindications they should be given to all patients with suspected AMI and high-risk angina.

- Reduces incidence of VF
- Adjunctive agent with fibrinolytic therapy beta blockage may reduce the rate of nonfatal re-
- Reduces the rate of re-infarction and recurrent ischemia. They also reduce mortality if administered early to fibrinolytic ineligible patients.

- Atenolol, metoprolol and Propranolol have been shown to reduce the incidence of VF significantly post infarct

Side effects include bradycardias, AV conduction delays and hypotension. Decompensation and cardiogenic shock after administration are infrequent provided that administration to patients with severe congestive heart failure is avoided and patients with mild and moderate congestive heart failure are monitored closely with appropriate diuresis.

Contraindications include second or third degree heart block, hypotension, severe congestive heart failure, and lung disease associated with bronchospasm.

BRETYLIUM – has been removed from ACLS treatment algorithms and guidelines because of a high occurrence of side effects, the availability of safer agents at least as efficacious and the limited supply and availability of the drug.

CALCIUM CHANNEL BLOCKERS: VERAPAMIL AND DILTIAZEM

Verapamil and diltiazem are calcium channel blocking agents that slow conduction and increase refractoriness in the AV node.

- They may terminate arrhythmias that require AV nodal conduction for their continuation.
- Control ventricular response rate in patients with AF, Aflutter or MAT
- My decrease myocardial contractility and may exacerbate congestive heart failure in patients with severe LV dysfunction.
- Adenosine is the drug of choice for terminating narrow complex PSVT

Dose 2.5-5mg IV given over 2 minutes. Repeated doses of 5-10mg may be administered every 15-30 minutes to a maximum of 20mg. Should only be given to patients with narrow complex PSVT or arrhythmias known with certainty to be of Supraventricular origin. Verapamil should not be given to patients with impaired ventricular function or heart failure.

Diltiazem at a dose of 0.25mg/kg followed by a second dose of 0.35mg/kg is equivalent to efficacy to Verapamil.

DOPAMINE – endogenous catecholamine agent with dose related dopaminergic and beta and alpha-adrenergic agonist activity. At doses between 3 and 7.5 ug/kg per minute, acts as Beta agonist, increasing heart rate and cardiac output. The inotropic effects of dopamine are modest compared with those of dobutamine. Dopamine is regarded as the safer agent and has displaced Isoproterenol as the preferred catecholamine for bradycardias in which atropine is either ineffective or contraindicated.

FLECAINIDE – approved in oral form only in the United States.

- Ventricular arrhythmias and for Supraventricular arrhythmias in patients without structural heart disease.
- Should be avoided in patients with impaired LV function.
- Increased mortality in patients with AMI.

IBUTILIDE – is a short acting Antiarrhythmic. Ibutilide acts by prolonging the action potential duration and increasing the refractory period of cardiac tissue.

- Recommended for acute pharmacological conversion of atrial flutter or AF or as an adjunct to electrical cardioversion in patients in whom electrical cardioversion alone has been ineffective.
- Administered IV diluted or undiluted as 1mg (10cc) over 10 minutes

ISOPROTERENOL – Pure beta-adrenergic agonist with potent inotropic and chronotropic effects.

- Increases myocardial oxygen consumption, cardiac output, myocardial work and can exacerbate ischemia and arrhythmias in patients with ischemic heart disease, congestive heart failure, or impaired ventricular function.
- Immediate, temporary control of hemodynamically significant Bradycardia when atropine and dobutamine have failed and transcutaneous and transvenous pacing are not available.
- For symptomatic Bradycardia it should be used (if at all) with extreme caution
- Not indicated in patients with cardiac arrest or hypotension

LIDOCAINE – Antiarrhythmic drug available for treatment of ventricular ectopy

- Prophylactic administration of Lidocaine is not recommended.
- Toxic to therapeutic balance is delicate.
- Second choice behind other alternative agents (amiodarone, procainamide, or sotalol)
- Half-life increases after 24-48 hours and dosage should be decreased after 24 hours.
- In arrest 1-15.mg/kg IV

MAGNESIUM – Severe magnesium deficiency is associated with cardiac arrhythmias, symptoms of cardiac insufficiency and sudden cardiac death.

- Magnesium deficiency should be corrected if present
- In emergent circumstances, magnesium 1-2 grams is diluted in 100cc and administered over 1 to 2 minutes
- Rapid administration of magnesium may cause clinically significant hypotension or asystole and should be avoided.

- Magnesium may be an effective treatment for Antiarrhythmic drug induced torsades de pointes even in the absence of magnesium deficiency.
- The routine prophylactic administration of magnesium in patients with AMI is no longer recommended in cardiac arrest except when arrhythmias are suspected to be caused by magnesium deficiency or when the monitor displays torsades de pointes.

PROCAINAMIDE – suppresses both atrial and ventricular arrhythmias.

- Acceptable for the pharmacological conversion of Supraventricular arrhythmias (particularly AF and atrial flutter) to sinus rhythm
- To control rapid rate due to accessory pathway conduction in pre-excited atrial arrhythmias and for wide complex tachycardias that cannot be distinguished as being Supraventricular or ventricular.

Given as an infusion of 20mg/min until

1. The arrhythmia is suppressed
2. hypotension ensues
3. the QRS complex is prolonged by 50% from original duration
4. A total of 17mg/kg

Bolus administration results in toxic concentrations and significant hypotension. The maintenance infusion rate of procainamide is 1 to 4mg/min. Should be reduced in renal failure.

Procainamide should be avoided in patients with pre-existing QT prolongation and torsades de pointes.

PHARMACOLOGY: AGENTS TO OPTIMIZE CARDIAC OUTPUT AND BLOOD PRESSURE

EPINEPHRINE – produces beneficial effects in patients during cardiac arrest, primarily because of its alpha-adrenergic receptor stimulating properties. Increase myocardial and cerebral blood flow during CPR. The value and safety of the beta-adrenergic effects are controversial because they may increase myocardial work and reduce subendocardial perfusion.

Results from 4 clinical trials compared high dose epinephrine with standard dose. No statistically significant improvement in rate of survival to hospital discharge occurred. 8 Randomized clinical studies involving more than 9000 cardiac arrest patients have found no improvement in survival to hospital discharge or neurological outcome, even in subgroups with initial high dose epinephrine compared with standard doses.

High dose Epinephrine may lead to increased myocardial dysfunction and occasionally a severe toxic hyperadrenergic state in the post resuscitation period.

HIGH DOSE EPI IS NO LONGER RECOMMENDED TO ROUTINE USE.

Epinephrine has good bioavailability following tracheal delivery in administered appropriately. Dose should be at least 2 to 2.5 times the peripheral IV dose.

The recommended dose of epinephrine is 1.0 mg (10ml of a 1:10,000) solution administered IV every 3 to 5 minutes during resuscitation.

VASOPRESSIN - naturally occurring antidiuretic hormone. In unnaturally high doses vasopressin acts as a non-adrenergic peripheral vasoconstrictor. Vasopressin acts by direct stimulation of smooth muscle V1 receptors.

- Not recommended in conscious patients with CAD because the increased peripheral vascular resistance may provoke angina pectoris
- After a short duration of ventricular fibrillation during CPR, vasopressin increased coronary perfusion pressures, vital organ blood flow, ventricular fibrillation median frequency and cerebral oxygen delivery. Similar results were found with prolonged cardiac arrest and PEA
- Produces no skeletal muscle vasodilatation or increased myocardial oxygen consumption during CPR because it has no beta-adrenergic activity.

Vasopressin is an effective vasopressor and can be used as an alternative to epinephrine for the treatment of adult shock refractor VF. Vasopressin may be effective in patients with asystole or PEA as well, however as of 2000 we lack sufficient data to support an active recommendation to use vasopressin in PEA or asystole.

NOREPINEPHRINE - naturally occurring potent vasoconstrictor and inotropic agent. Cardiac output may increase or decrease in response to norepinephrine, depending on vascular resistance, the functional state of the left ventricle and reflex responses.

Myocardial oxygen requirements may be increased, mandating cautious use in patients with ischemic heart disease.

DOPAMINE is a catecholamine like agent and a chemical precursor of norepinephrine that has both alpha and beta receptor stimulating actions.

- Stimulates the heart through both alpha and beta
- In periphery it releases norepinephrine from stores but the vasoconstricting effects of norepinephrine are countered by activity at the DA receptors producing vasodilation in physiological concentration.
- Reserved for hypotension that occurs with symptomatic Bradycardia after resuscitation.

- Dopamine in combination with other agents, including dobutamine, remains an option in the management of post resuscitation shock.
- Should not be mixed with sodium bicarbonate or other alkaline solutions in the IV line because some data indicates that it may be inactivated in alkaline solutions.
- Therapy should be tapered gradually not discontinued abruptly.
- Dosage ranges from 5 to 20 ug/kg/min. In excess of 1-ug/min system and splanchnic vasoconstriction
- Dose range of 2-4 ug/kg/min dopamine is primarily a dopaminergic agonist with little inotropic effect.

DOBUTAMINE – potent inotropic agent useful in the treatment of severe systolic heart failure.

- Stimulating effects that increase myocardial contractility in dose dependent manner, accompanied by a decrease in left ventricular filling pressures.
- Increase in stroke volume frequently induces reflex peripheral vasodilation so that arterial pressure may remain unchanged.
- Usually dose is 5-20 ug/kg/min
- Elderly patients have a significantly decreased response to dobutamine.
- Doses of >20 ug/kg/min increases in heart rate of >10% may induce or exacerbate myocardial ischemia.

AMRINONE AND MILRINONE phosphodiesterase III inhibitors that have inotropic and vasodilatory properties.

- More significant effect on preload than catecholamines and the hemodynamic effects are similar to dobutamine.
- Approved for use in severe heart failure or cardiogenic shock that is not adequately responsive to standard therapy.

CALCIUM – Although calcium ions play a critical role in myocardial contractile performance and impulse formation, retrospective and prospective studies in the cardiac arrest setting have shown no benefit from the use of calcium

- Hyperkalemia, hypocalcemia or calcium channel blocker toxicity is present calcium is probably helpful

DIGITALIS – have limited use as inotropic agents in ECC. Digitalis decreases the ventricular rate in some patients with atrial flutter or fibrillation by slowing atrioventricular nodal conduction.

NITROGLYCERIN – relax vascular smooth muscle. NTG is the initial treatment of choice for suspected ischemic type pain or discomfort.

- The therapeutic effect may last up to 30 minutes.
- IV NTG permits more controlled titration in patients with acute coronary syndromes, hypertensive urgencies or congestive heart failure.
- Use cautiously in patients with inferior MI. Nitrates are contraindicated in patients who are preload dependent with right ventricular infarction.
- Hypovolemia blunts the beneficial hemodynamic effects of NTG and increases the risk of hypotension.
- Nitrate induced hypotension responds to fluid replacement therapy.
- Low doses (30-40 ug/min) predominantly produce venodilation; high doses (150-500 ug/min) lead to arteriolar dilatation as well.
- Prolonged administration of NTG >24 hrs may produce tolerance.

SODIUM NITROPRUSSIDE – potent, rapid acting direct peripheral vasodilator useful in the treatment of severe heart failure and hypertensive emergencies.

- Direct venodilatation causes decreases in right and left ventricular preload resulting in relief of pulmonary congestion and reduced left ventricular volume and pressure
- If Intravascular volume is normal or high reduction in Peripheral vascular resistance is usually accompanied by increased stroke volume minimizing the fall in blood pressure
- If hypovolemia, likely to cause excessive drop in BP with reflex tachycardia
- Particularly useful in severe heart failure caused by regurgitant valvular lesions or aortic insufficiency and mitral regurgitation
- NTG is less likely to lower coronary perfusion pressure and is more likely to increase blood supply to ischemic areas of the myocardium.
- NTG is the preferred vasodilator in acute MI especially when complicated by CHF.
- Dose: 0.1 to 5 ug/kg/min but higher doses may be needed

SODIUM BICARBONATE

- Adequate alveolar ventilation is central for control of acid base balance during cardiac arrest and the post arrest period
- Laboratory and clinical data indicate that;
 - Does not improve the ability to defibrillate or survival rates
 - Can compromise coronary perfusion pressure
 - May cause adverse effects due to extracellular alkalosis, including shifting the oxyhemoglobin saturation curve or inhibiting release of oxygen
 - May induce hyperosmolarity and hypernatremia

- Produces carbon dioxide, which is freely diffusible into myocardial and cerebral cells and may paradoxically contribute to acidosis
- Exacerbates central venous acidosis
 - Indicated with pre-existing metabolic acidosis, Hyperkalemia or tricyclic or phenobarbitone overdose.
 - May be helpful in long arrest situations guided by bicarbonate concentration or calculated base deficit obtained by blood gas analysis

POST RESUSCITATION CARE

Return of Spontaneous Circulation After a No Flow Cardiac Arrest

Cardiovascular and hemodynamic derangements are common when spontaneous circulation returns (ROSC) after arrest. They include hypovolemic shock, cardiogenic shock and vasodilatory shock associated with the systemic inflammatory response syndrome (SIRS).

Multiple pathogenic factors contribute to the post resuscitation syndrome:

- Reperfusion failure
- Reperfusion injury
- Cerebral intoxication from ischemic metabolites
- Coagulopathy

FOUR PHASES OF POST RESUSCITATION SYNDROME (Dependent on the degree and duration of organ ischemia:

1. Almost one half of post resuscitation syndrome deaths that occur take place within 24 hours of the vent. Cardiovascular dysfunction tends to normalize over 12-24 hours. Microcirculatory dysfunction from multifocal hypoxia leads to rapid release of toxic enzymes and free radicals into the cerebrospinal fluid and blood.
2. Over 1-3 days cardiac function and systemic function improve but intestinal permeability increases, predisposing to sepsis. Several organs have progressive dysfunction, particularly the liver, pancreas and kidneys.
3. Finally, days after the cardiac arrest serious infection occurs and the patient declines rapidly.
4. Death

Principal Objective of post resuscitation phase is complete reestablishment of regional organ and tissue perfusion. The immediate goals of care are to:

- Provide cardiopulmonary support to optimize tissue perfusion, especially to the brain

- Transport the prehospital cardiac arrest patient to the ED and then a properly equipped critical care unit.
- Attempt to identify the precipitating cause of the arrest
- Institute measures such as Antiarrhythmic therapy to prevent recurrence.

OPTIMAL RESPONSE TO RESUSCITATION:

- Patient is awake, responsive and breathing spontaneously.
- Apply ECG monitor leads and supplement oxygen
- IV infusion with normal saline
- Change peripheral or central IV lines placed without proper sterile technique.
- Consider the use of an Antiarrhythmic agent that was used successful during resuscitation and continue infusion for several hours
- Consider the precipitating cause
- Perform laboratory investigations, including chest x-ray, determination of arterial blood gases, electrolyte, glucose, serum creatinine, blood urea, nitrogen, magnesium and calcium levels.

TEMPERATURE REGULATION

Regional cerebral metabolic rate determines the regional blood flow requirements of brain. Temperature elevation above normal can create a significant imbalance between oxygen supply and demand and impair brain recovery. **TREAT FEVER AGGRESSIVELY.**

Hypothermia:

After cardiac arrest, hemodynamically stable patients who develop a mild degree of hypothermia spontaneously after cardiac arrest should not be actively rewarmed. Mild hypothermia may be beneficial to neurological outcome and is likely to be well tolerated. However, hypothermia should not be induced.

RESPIRATORY SYSTEM - various degrees of respiratory dysfunction

- Pay special attention to potential complications of resuscitation, pneumothorax, misplacement of tracheal tube
- If high oxygen concentrations are needed it is important to establish whether the cause is pulmonary or cardiac dysfunction.

Ventilatory Parameters

Recent evidence supports the theory that sustained hypocapnea (low PCO₂) may worsen cerebral ischemia. If the patient is hyperventilated at this stage the additional cerebral vasoconstriction resulting from a low PCO₂ may further decrease cerebral blood flow and worsen cerebral ischemia.

With restoration of cardiac output, metabolic acidosis usually corrects over time and hyperventilation should not be used as a primary treatment modality. The use of buffer therapy is also not indicated and should be used for specific indications only.

CARDIOVASCULAR SYSTEM

- Serial vital signs and urine output
- 12-lead ECG tracings compared to previous tracings if available
- Chest x-ray
- Serum electrolyte levels

Serum marks levels may be elevated because of resuscitation efforts alone as global ischemia occurs during arrested or low flow states.

- Assess circulating volume and ventricular function
- AVOID EVEN MILD HYPOTENSION because it can impair recovery of cerebral function
- Intra-arterial assessment of blood pressure is usually more accurate in these patients and allows better titration of potentially dangerous catecholamine infusions

RENAL SYSTEM

The bladder must be catheterized so that urine output can be measured hourly and an accurate volume can be established.

Furosemide may maintain urine output despite developing renal failure. Dopamine at low doses does not improve splanchnic blood flow or provide specific renal protection and is no longer indicated in acute oliguric renal failure.

Progressive renal failure is indicated by a steadily rising serum urea nitrogen and creatinine, usually with Hyperkalemia and mortality and comorbidity are high in these patients who often require dialysis.

CENTRAL NERVOUS SYSTEM

- Cessation of circulation of 10 seconds results in a deficiency of oxygen supply to that brain that causes unconsciousness.
- 2-4 minutes glucose and glycogen stores of the brain are depleted
- 4-5 minutes ATP is exhausted.

Auto regulation of cerebral blood flow is lost after extended hypoxemia or hypercarbia, or both and cerebral blood flow becomes dependent on cerebral perfusion pressure.

Any elevation of intracranial pressure or reduction in systemic mean arterial pressure may reduce cerebral perfusion pressure and further compromise

cerebral blood flow. Optimize cerebral perfusion by maintaining a normal or slightly elevated mean arterial pressure.

Normothermia should be maintained and seizure activity controlled with Phenobarbital, phenytoin, or diazepam or barbiturate

The head should be elevated and maintained in a midline position to increase cerebral venous drainage.

GASTROINTESTINAL SYSTEM – a nasogastric tube should be inserted if bowel sounds are absent and in those patients with a reduced level of consciousness who are mechanically ventilated.

SYSTEMIC INFLAMMATORY RESPONSE SYNDROME (SIRS) AND SEPTIC SHOCK

Complex process that may be triggered by a variety of initial insults, such as trauma, burn or infection. The inflammatory response results in tissue damage and initiates a self-perpetuating process that results in local tissue damage.

The goal of hemodynamic management is normal tissue oxygen uptake. Initial management consists of volume replacement. Following volume replacement an inotrope or vasopressin is usually required.

When sepsis is suspected, empirical antibiotic therapy is indicated and should be directed at common and usual organisms.

ACUTE CORONARY SYNDROMES (ACUTE MYOCARDIAL INFARCTION)

Major Guidelines and Recommendations:

I. Prehospital Care

- A. Implementation of out of hospital 12-Lead ECG diagnostic programs is recommended in urban and suburban paramedic systems.
- B. Out of hospital fibrinolytic therapy is recommended when a MD is present or out of hospital transport time is >60 minutes
- C. When possible patients at high risk for mortality or severe left ventricular dysfunction with signs of shock, pulmonary congestion, heart rate \geq 100 beats per minute, systolic blood pressure <100 should be triaged to facilities capable of performing cardiac catheterization and rapid revascularization.

II. Reperfusion Therapies

- A. Early fibrinolytic therapy as a standard of care for acute ST segment elevation myocardial infarction

- B. PCI including angioplasty/stent is Class I recommendation for patients <75 years of age with acute syndromes and signs of shock.
- C. Patients in whom fibrinolytic therapy is contraindicated should be considered for transfer to interventional facilities
- D. Heparin is currently recommended for patients receiving selective fibrinolytic agents.
- E. Heparin dosing with fibrinolytics is changes to reduce the incidence of intracerebral hemorrhage and minimize reocclusion. 60U/kg followed by infusion of 12Ukg/hour with a maximum of 4000U bolus and 1000U/h infusion

III. New Therapy for Unstable Angina/Non-Q wave MI

- A. Glycoprotein IIb/IIIa inhibitors are commended for patients with non-ST segment elevation MI or high-risk unstable angina.
- B. IIb/IIIa inhibitors have incremental benefit in addition to conventional therapy with unfractionated Heparin
- C. Low molecular weight heparin is an alternative to UFH for the treatment of non Q wave MI and unstable angina
- D. Troponin positive patients are at risk for major adverse cardiac events and should be considered for aggressive management.

PATHOGENESIS

Patients with coronary atherosclerosis in whom these clinical syndromes develop have various degrees of coronary artery occlusion. Typically ACS is caused by rupture of a lipid-laden plaque with a thin cap. Most these plaques are not hemodynamically significant before rupture.

A partially occluding thrombus produces symptoms of ischemia that may be prolonged and may occur at rest. At this stage the thrombus is platelet-rich. Therapy with antiplatelet agents such as aspirin an GP IIb/IIIa receptor inhibitors, is most effective at this time. Fibrinolytic therapy is not effective and paradoxically may accelerate occlusion by causing the release of clot-bound thrombin, which further activates platelets. Patients with such a thrombus are at highest risk of progression to MI. The clot causing Q-wave MI is rich in thrombin and fibrin, in these patients fibrinolysis or PCI (angioplasty/stent) may limit the size of infarct.

OUT OF HOSPITAL MANAGEMENT

Early Defibrillation

Half of the patients who die of AMI do so early, before reaching a hospital. In most of these deaths the presenting rhythm is VT or VF. VF occurs in 4% to 18% of patients with infarctions. Once the patient reaches the hospital the VF rate is approximately 5%.

- Fibrinolytic therapy reduced the occurrence of VF primarily within the first 3 hrs. The presence of primary VF increases in hospital mortality and complications but does not appear to increase long-term mortality.
- All EMS and dispatch systems should have a trained and dedicated staff to respond to cardiac emergencies
- All vehicles that respond to cardiac emergencies should carry a defibrillator and staff skilled in its use.
- Ideally an EMS system should have enough trained personnel that a first responder can be at a victim's side anywhere in the system within 5 minutes.
- Patients with AMI have high risk of sudden cardiac death. *An out-of-hospital EMS system that can provide immediate defibrillation is mandatory.*
- If VF occurs under observation and immediate defibrillation occurs many patients will survive.

When patients with Acute Coronary Syndromes including MI and other ischemic syndromes reach the ED and hospital critical care unit, their risk of sudden cardiac death due to lethal arrhythmias falls dramatically. This decline in risk stems from a combination of early reperfusion, administration of Beta-blockers and other adjunctive agents used in the reperfusion era.

PATIENT EDUCATION AND DELAYS IN THERAPY.

Potential delay during in hospital evaluation include *door to data, from data to decision and from decision to drug.*

Patient delay to symptom recognition constitutes the longest period of delay to treatment. The elderly, women, persons with diabetes, and hypertensive patients are likely to delay partly because they tend to have atypical symptoms or unusual presentations.

Education of patients is the primary intervention to reduce denial or misinterpretation of symptoms. Public education campaigns have been effective in increasing knowledge of symptoms and signs of heart attack.

OUT OF HOSPITAL ECG'S

Out of hospital performance of electrocardiography and transmission of the ECG to the ED speeds the care of patients with AMI.

Diagnostic quality ECG's can be successfully transmitted for approximately 85% of patients with chest pain. Recording an ECG increases the time spent at the scene by only 0 to 4 minutes. There is no difference between the quality of information collected out of hospital and that received by cellular transmission at the base station.

- The use of out of hospital ECG and a chest pain evaluation form leads to more rapid initiation of reperfusion therapy without substantially delaying out of hospital time.
- Many studies have shown significant reductions in hospital-based time to treatment with fibrinolytic therapy in patients with AMU identified before arrival.
- The US National Heart Attack Alert Program recommends that EMS systems provide out of hospital 12 lead ECG's to facilitate early identification of AMU and that all advanced lifesaving vehicles be able to transmit a 12 lead ECG to the hospital.
- In recent studies the out of hospital ECG group was also significantly more likely to receive fibrinolytic therapy, primary angioplasty, or CABG. The in hospital mortality rate was 8% among patients with prehospital ECG and 12% among those without prehospital ECG.
- The safety, feasibility and practicality of obtaining prehospital 12 leads are well documented.
- American Heart Association recommends the implementation of out of hospital 12-Lead ECG diagnostic programs in urban and suburban paramedic systems.

CARDIOGENIC SHOCK AND OUT OF HOSPITAL FACILITY TRIAGE

The incidence of cardiogenic shock in the GUSTO I study was 11% and an aggressive strategy (cath lab) was associated with a lower mortality than that associated with fibrinolytic therapy. The use of early invasive interventions is more common in the United States than in other countries, but patients who underwent revascularization had better survival in all countries.

- Class I recommendation for PCI in patients with shock who are <75 years of age.
- When possible transfer patients at high risk for mortality or severe LV dysfunction with signs of shock
 - Pulmonary Congestion
 - Heart Rate >100 and SBP <100 to facilities capable of cardiac catheterization and rapid revascularization
 - Pts with contraindications for thrombolysis should also be transferred to interventional facilities when benefit from reperfusion exists

OUT OF HOSPITAL FIBRINOLYSIS:

Early small trials yielded conflicting results about the efficiency and efficacy of this strategy.

- Administration of fibrinolytics out of hospital period appears to reduce mortality when transport times are long
- Recommended that prehospital fibrinolytics be administered in the field when a physician is present or transport time is >60 minutes.

INITIAL GENERAL MEASURES

1. Oxygen – all patients complaining of ischemic type chest discomfort.
 - a. Nasal Cannula
 - b. Evidence that supplemental oxygen may limit ischemic myocardial injury
 - c. May reduce the amount of ST segment elevation.
2. Nitroglycerin – effective analgesic for ischemic type chest discomfort.
 - a. Hemodynamic effects including dilation of coronary arteries and peripheral arterial bed and venous capacitance vessels.
 - b. Administer nitrates with extreme caution if at all, to patients with suspected right ventricular (RV) infarction (50% of inferior wall MI's.)
 - c. Indicated for initial management of pain and ischemia with patients of AMI without hypotension except in patients with RV infarction.
 - d. Indicated in the first 24-48 hours
 - e. Particularly useful with patients who have hypertension, CHF or large anterior wall MI.
3. Morphine – although NTG effectively relieves ischemic pain due to ACS it should not be used as a substitute for narcotic analgesia which is often necessary to relieve pain associated with MI
 - a. Indicated for continuing pain unresponsive to nitrates.
 - b. Effective in patients with vascular congestion complication AMI because of its favorable hemodynamic effects.
4. Aspirin – should be given to all patients suspected of ACS as soon as possible unless there is a documented allergy.
 - a. Dose 160-325mg causes rapid and near total inhibition of thromboxane
 - b. Reduced death from MI in ISIS 2 and its effect as additive to that of streptokinase.
 - c. Chewable aspirin is absorbed more quickly than PO in early hours of infarction, particularly if morphine has been given. Aspirin suppositories are safe and recommended for patients with severe nausea, vomiting, or disorders of the upper gastrointestinal tract.

RISK STRATIFICATION

Patients with Chest Pain Suggestive of Ischemia: Probability of Significant CAD Based on Clinical Features and Presenting ECG

High Risk	Intermediate Risk	Low Risk
Prior MI or life threatening arrhythmia episode Known CAD	Definite clinical angina in young age.	Possible Angina
Definite clinical angina	Probable angina in older age	1 risk factor, not diabetes
Dynamic ST changes with chest pain symptoms	Possible Angina Diabetes 3 other Risk Factors	T wave inversion <1mm
Marked T wave changes in anterior precordial leads	ST depression <1mm T wave inversion >1mm (leads with dominant R waves)	Normal ECG

Patients with Chest Pain Suggestive of Ischemia: short Term Risk of Death and Non Fatal MI

High Risk of Death or Non-Fatal MI	Intermediate Risk of Death or Non-Fatal MI	Low Risk of Death of Non-Fatal MI
Prolonged continued pain not relieved by rest (>20 mins)	Prolonged angina (>20 min) but resolved at time of evaluation, moderately high likelihood of CAD	Angina increased in frequency, severity or duration
Pulmonary edema related to ischemia S ₂ or rales	Rest angina >20 minutes or relieved with NTG	Lower activity threshold before angina
Hypotension with Angina	Age >65 years	1 risk factor, not diabetic
Rest angina with Dynamic ST segment changes >1mm	Dynamic T wave changes and angina	New onset angina >2 wk to 2 mos before presentation
Elevated serum troponin I or I	Pathological Q waves or ST segment depression <1mm in multiple lead groups	

ST SEGMENT ELEVATION IN AMI

Patients with ST segment elevation should be rapidly evaluated for reperfusion therapy. Strategies for reperfusion include the administration of fibrinolytics and primary PCI

Fibrinolytics

- The most significant advance in treatment of cardiovascular disease in the last decade.
- Major determinants of myocardial salvage and long term prognosis:
 - Short time to reperfusion
 - Early and sustained patency of the infarct related artery with normal flow
 - Normal micro vascular perfusion

Risk Benefit of Fibrinolytic Therapy

Most of the reluctance to administer fibrinolytic agents is related to the risks of hemorrhage and intracranial bleeding.

- Therapeutic benefits were seen up to 12 hours but were greatest when fibrinolytics were administered in the first 3 hours. The benefits were less impressive in inferior wall infarction except when associated with RV infarction.
- The incidence of stroke increases with age and the relative benefit of fibrinolytic therapy is reduced. Older age is the most important baseline variable predicting non-hemorrhagic stroke.
- Associated with a small but definite increase in the risk of hemorrhagic stroke with contributes to the early hazard of therapy in the first day.
- Not recommended if > 12 hours past onset of symptoms

REPERFUSION THERAPY: PCI (Angioplasty/Stenting)

Clinical trials support the finding that direct coronary angioplasty is potentially superior to fibrinolytic therapy in the restoration of infarct patency.

- Provides higher rates of normal flow
- Is successful in >90% of patients
- Associated with lower rates of reocclusion and post infarction ischemia than fibrinolytic therapy

A major criticism of primary angioplasty is the need for on-site catheterization facilities and experienced operators. Triage by EMS personnel of patients with large anterior infarction and those with severe LV dysfunction may attenuate this problem. Door to balloon times are Suboptimal but experienced centers can reduce this delay so that reperfusion times may be comparable to those of fibrinolytic therapy.

GP IIb/IIIa INHIBITORS

Platelet adhesion, activation, and aggregation may result in formation of an arterial thrombus and are pivotal in the pathogenesis of ACS.

GP IIb/IIIa Inhibitors improve clinical outcome in patients with unstable angina or Non ST segment elevation MI

NON-DIAGNOSTIC ECG

Screening of patients with non-diagnostic ECG's and ischemic or atypical chest pain in the ED an area of clinical, legal and economic significance. Patients with a non-diagnostic ECG who have indeterminate or low risk of MI should receive aspirin and other therapy as clinically indicated while undergoing serial cardiac studies to assess ongoing cardiac necrosis or unstable coronary syndromes.

IT IS IMPORTANT TO OBTAIN SERIAL ECG TRACINGS DURING EVALUATION IN THE EMERGENCY DEPARTMENT.

COMPLICATION AMI

- I. Cardiogenic Shock, LV Power Failure and CHF**
 - A. Infarction of 50% of the LV myocardium usually results in cardiogenic shock and death.
 - B. Incidence of cardiogenic shock remains relatively constant (7.5%) and mortality is still high, averaging 50-70%

The ejection fraction of the heart falls when the amount of blood pumped with each beat (stroke volume) decreases. The ventricle dilates with an increase in end diastolic volume. These changes may increase myocardial oxygen consumption, increase ischemia in viable or distant myocardium and extend infarction.

The combination of hypotension and pulmonary edema constitutes clinical cardiogenic shock.

Initial therapy includes IV diuresis and preload and afterload reduction with nitrates. Intra-aortic balloon counterpulsation and transfer the patient to the cardiac catheterization has been shown to reduce mortality.

Fibrinolytic therapy has not been shown to consistently improve outcome in patients with cardiogenic shock and may have several limitations.

When possible triage high risk patients with cardiogenic shock or refer them to cardiovascular facilities with interventional specialists.

RIGHT VENTRICULAR INFARCTION

RV ischemia or infarction may occur in up to 50% of patients with inferior wall infarction.

Clinically manifested by jugular venous distention, Kussmaul's signs and various degrees of hypotension.

- Suspect in patients with inferior wall infarction, hypotension and clear lung fields.
- Right sided ECG leads using V4R is sensitive (90%) and is a strong predictor of in hospital complications and mortality.
- Routinely consider reperfusion in these patients.
- Dependent on maintenance of RV filling pressures to maintain cardiac output. AVOID USE OF AGENTS THAT REDUCE PRELOAD, SUCH AS NITRATES AND DIURETICS BECAUSE SEVERE HYPOTENSION MAY DEVELOP.
- Initial therapy consists of volume lading with 500cc of NSS up to 1-2Liters.

ADJUNCTIVE THERAPY FOR ACUTE CORONARY SYNDROMES

HEPARIN

- Recommended to all patients with diagnosed AMI
- 17% reduction in mortality and 22% reduction in the risk of re-infarction
- Heparin has been shown to increase patency of infracted artery when rt-PA is used.

Beta Adrenergic Receptor Blockers

- Reduce the size of infarct in patients who do not receive fibrinolytic therapy
- Reduce incidence of Ventricular Ectopy and fibrillation
- In patients who receive fibrinolytic agents, they decrease post infarction ischemia and non-fatal MI
- Start within 12 hours of onset of infarction
- Useful as an adjunct to morphine and to help control ventricular response in atrial fibrillation.

CALCIUM CHANNEL BLOCKERS

- Have not demonstrated a reduction in mortality or combined cardiovascular end points
- Beta Blockers are first line
- May be added as an alternative or additional therapy if Beta-blockers are contraindicated.

ACE I THERAPY

- Has improved survival in patients with AMI.
- Reduction in mortality is seen early after onset
- Should be given within the first day after MI when the patient is stable and after reperfusion and initial measures and other therapies have been started.

METABOLIC MANIPULATION OF THE INFARCT: GLUCOSE-INSULIN-POTASSIUM

- GIK therapy for patients with AMI may be helpful
- Easily administered and associated with few adverse effects.

VENTRICULAR RHYTHM DISTURBANCES

- The incidence of primary VF is highest (3-5%) in the first 4 hours after coronary occlusion then declines markedly. Primary VF should be distinguished from secondary VF occurring in the setting CHF or cardiogenic shock.
- *The practice of routine prophylactic administration of Lidocaine has been abandoned.*
- Routine IV administration of Beta Blockers to patients without hemodynamic or electrical contraindications, is associated with reduced incidence of primary VF
- Prophylactic treatment of arrhythmias or treatment of asymptomatic WARNING arrhythmias is not recommended.

BRADYCARDIA AND HEART BLOCK – INDICATIONS FOR PACING DURING AMI

- One third of patients with AMI develop sinus Bradycardia. Because of increased vagal tone it is often seen in patients with inferior wall MI's secondary to occlusion of the right coronary artery when that artery supplies the AV nodes.
- Initial treatment with atropine is indicated only when serious signs and symptoms are related to the decreased rate
- 2nd or 3rd degree AV block complicates approximately 20% of MI's
- Only rarely will a patient die of heart block. It is not an independent predictor of mortality and it is a poor predictor of mortality in patients who survive to discharge.
- In general treatment is not usually required.
- Avoid Atropine in Type II and 3rd degree with a wide QRS complex.

Indications for Transcutaneous Patches/Pacing

1. Hemodynamically unstable Bradycardia
2. Mobitz II
3. 3rd Degree Block
4. Bilateral BBB (alternative BBB or RBB and alternating LBBB)
5. Left anterior fascicular block
6. Newly acquired or age indeterminate LBBB
7. RBBB or LBBB and first degree AV block

ATRIAL FIBRILLATION COMPLICATING AMI

New onset atrial fibrillation complicating MI occurs in 10-15% of patients. It is associated with increasing age, large infarcts, LV hypertrophy and CHF.

- If rapid rate resulting in ischemic symptoms or hemodynamic compromise, immediate cardioversion is indicated.
- In stable patients, beta blockade is indicated.

Mortality is increased when atrial fibrillation develops in the setting of AMI.

ACUTE STROKE

Major Guideline Changes

- Intravenous administration of tissue type Plasminogen activator for patients with acute ischemic stroke and no contraindications is recommended
 - Within 3 hours of onset of stroke symptoms (Class I)
 - Between 3-6 hours of onset of stroke symptoms (Class Indeterminate)
- Intra-arterial fibrinolysis within 3-6 hours after the onset of symptoms may be beneficial in patients with occlusion of the middle cerebral artery (Class IIB)

Stroke is the disruption in blood supply to a region of the brain that causes neurological impairment. Stroke is ranked among the top 3 leading causes of death in most countries and is the leading cause of brain injury in adults.

Classified into 2 major categories: ischemic and hemorrhagic.

- 85% of all strokes are ischemic.

Fibrinolytic therapy now offers an opportunity to possibly limit the extent of neurological damage to improve outcome in stroke patients.

Early treatment of stroke depends strongly on recognition of the event by the patient, family members or bystanders.

ROLE OF EMS IN STROKE CARE

- Rapid activation of the EMS system is essential to optimize care. Patients who use the EMS system arrive at the hospital after than those who do not.
- Quickly transport patients to a stroke center and notify the facility before arrival.
- Only 50% of patients use the EMS system (85% of strokes occur at home)

7 D's of Stroke Management

- Detection
- Dispatch
- Delivery
- Door
- Data
- Decision
- Drug

EMS dispatchers must prioritize the call for a suspected stroke patient as they would for a victim of AMI or serious trauma and dispatch the appropriate EMS team.

AIRWAY AND VENTILATION

Airway obstruction may be a major problem in acute stroke. Hypoxia and hypercarbia can occur as a result of inadequate ventilation contributing to cardiac and respiratory instability. Aspiration of secretions or gastric contents is a serious complication associated with considerable morbidity and mortality.

VITAL SIGNS

Check vital signs frequently to detect abnormalities and changes. Abnormal respirations are particularly prevalent in comatose patients and usually reflect serious brain dysfunction.

Cardiac arrhythmias may contribute to the cerebral thromboembolism or they may be the consequence of brain injury.

NEUROLOGICAL EVALUATION

- Stroke screen or scale
- Time of onset of stroke signs
- Level of consciousness
- Type of Stroke (hemorrhagic versus nonhemorrhagic)
- Location of Stroke
- Severity of stroke

EMERGENCY DIAGNOSTIC STUDIES

CT is the most important diagnostic test for differentiating between infarction and hemorrhage or other intracranial masses. CT with contrast.

MRI is not part of the routine evaluation of acute stroke. It is very sensitive and will detect some lesions missed by CT, but is not superior to CT and is time consuming and may hamper continuous observation of acutely ill patients.

Emergency cerebral angiography is performed in many patients with subarachnoid hemorrhage in anticipation of aneurysm clipping.

EMERGENCY MANAGEMENT

- Rapidly identify, evaluate and treat all patients with signs and symptoms of acute stroke
- IV access en route to the hospital
- Normal saline at a rate of 50cc/hr unless hypotensive
- Avoid rapid infusions which increase the risk of cerebral edema
- Do not administer Dextrose in water
- Correct hyperglycemia and hyperthermia
- Do not routinely administer oxygen to non hypoxic (O₂ Sats >90%) stroke victims with minor or moderate strokes.

MANAGEMENT OF ELEVATED BLOOD PRESSURE

- Many patients have HTN after stroke, but few require emergent treatment
- Antihypertensive treatment is reserved for patients with markedly elevated BP or specific medical conditions.
- Antihypertensive therapy can be harmful and lower the cerebral perfusion pressure and lead to worsening of the stroke.
- Use of short acting nifedepine is CONTRAINDICATED

In candidates for fibrinolytic therapy, however, strict control of BP is required to reduce the potential for bleeding. Fibrinolytic therapy is not recommended for patients who have a systolic BP >185 or a diastolic >110 at the time of treatment.

MANAGEMENT OF INCREASED ICP

Death during the first week commonly is caused by brain edema and increased ICP. Fortunately only 10-20% of stroke patients develop brain edema sufficient to cause clinical deterioration.

- Elevation of the head (20-30 degrees)
- Avoidance of hypoxemia and hypoventilation
- Control of agitation and pain

ANTICOAGULANT THERAPY

Efficacy of anticoagulants in acute stroke has not been established. Heparin is frequently administered to patients with acute ischemic stroke but its value is unproved.

Heparin may help prevent recurrent embolism or propagation of a thrombus, but it may lead to bleeding complications including brain hemorrhage.

Aspirin, Heparin, Ticlid reduce the risk of subsequent stroke in patients with TIA and should be started within the first few days after TIA.

ADVANCED CARDIAC LIFE SUPPORT FOR THE EXPERIENCE PROVIDER

OVERVIEW

FIVE QUADRADS APPROACH

- Applied to all patients in cardiac arrest
- Applied to patients in an unstable condition steadily deteriorating toward arrest

1. ABCD Primary Survey (Quadrad 1)

- A. AIRWAY - Open the Airway
- B. BREATHING - Provide positive-pressure ventilation
- C. CIRCULATION - Perform chest compressions
- D. DEFIBRILLATION - Identify and shock VF/pulseless VT

2. ABCD Secondary Survey (Quadrad 2)

A. AIRWAY -

- Determine the effectiveness of the initial ventilation and airway techniques
- Perform endotracheal intubation if indicated

B. BREATHING -

- Determine that the endotracheal tube is patent and properly placed
- Provide positive-pressure ventilation through the endotracheal tube
- Ensure that the chest wall moves with each ventilation
- Listen for bilateral breath sounds

C. CIRCULATION -

- Continue chest compressions
- Obtain IV access
- Attach monitor leads
- Identify rhythm and rate
- Measure blood pressure
- Administer medications appropriate for rhythm and vital signs

D. DIFFERENTIAL DIAGNOSIS - Consider possible causes of the cardiac emergency

3. ALWAYS RIGHT (Quadrad 3)

- Oxygen-IV-Monitor-Fluids

4. VITAL SIGNS (Quadrad 4)

- Temperature, blood pressure, heart rate, respirations

5. TANK - TANK - PUMP - RATE

- A. Tank (too large vs. too small; due to systemic vascular resistance)
- B. Tank (too low vs. too full; due to volume)
- C. Pump (pumping poorly vs. pumping well; due to cardiac performance)
- D. Rate (too slow vs. too fast) due to arrhythmias

DEVELOPING THE DIFFERENTIAL DIAGNOSIS
THE H'S AND T'S

The H Causes	Assessments	Treatments
Hypovolemia <ul style="list-style-type: none"> • Occult bleeding • Anaphylaxis • Pregnancy with gravid uterus 	<ul style="list-style-type: none"> • History/exam • Hematocrit • B-HCG test 	<ul style="list-style-type: none"> • Volume • Blood
Hypoxia <ul style="list-style-type: none"> • Inadequate oxygenation 	<ul style="list-style-type: none"> • Breath sounds • Tube placement • Arterial blood gases 	<ul style="list-style-type: none"> • Oxygen • Ventilation • Forceful CPR • ET tube stable
Hypothermia/Hyper <ul style="list-style-type: none"> • Profound hypothermia • Heat Stroke 	<ul style="list-style-type: none"> • Touch • Core Body temperature 	<ul style="list-style-type: none"> • Active/passive external rewarming • Active/passive internal rewarming
High and Low Electrolyte Levels <ul style="list-style-type: none"> • Potassium, sodium, magnesium, calcium 	<ul style="list-style-type: none"> • History/exam • Risk Factors 	<ul style="list-style-type: none"> • Calcium • Bicarbonates, insulin, glucose
Hypoglycemia Hyperglycemia <ul style="list-style-type: none"> • Low glucose = insulin reactions • Diabetes ketoacidosis • Nonketonic, hyperosmolar coma 	<ul style="list-style-type: none"> • History/exam • Lab tests 	<ul style="list-style-type: none"> • Fluids • Potassium • Insulin • 50% glucose
Hydrogen Ion <ul style="list-style-type: none"> • Acidosis • Diabetic ketoacidosis • Drug overdoses • Renal failure 	<ul style="list-style-type: none"> • Clinical setting • Arterial blood gas • Lab tests 	<ul style="list-style-type: none"> • Forceful CPR • Optimal perfusion • Hyperventilation • Bicarbonate

The T Causes	Assessments	Treatments
Trauma <ul style="list-style-type: none"> • Massive trauma • Electrocution • Lightning • Near-drowning 	<ul style="list-style-type: none"> • History • Clinical Setting • Physical Exam 	<ul style="list-style-type: none"> • Rescuer Safety • Reverse triage • Early ETT placement • Treat asystole longer
Tension Pneumothorax <ul style="list-style-type: none"> • Asthma as possible cause • Trauma • COPD, blebs • Ventilators +positive pressure 	<ul style="list-style-type: none"> • Risk factors • Diminished lung sounds • Tracheal deviation • Distended neck veins 	<ul style="list-style-type: none"> • Needle compression • Chest Tube
Thrombosis, lungs <ul style="list-style-type: none"> • Pulmonary embolus 	<ul style="list-style-type: none"> • Risk factors • History • Echo or VQ can 	<ul style="list-style-type: none"> • Volume • Dopamine • Heparin • ?Thrombolytics
Thrombosis, heart <ul style="list-style-type: none"> • AMI • Other acute coronary syndromes 	<ul style="list-style-type: none"> • Prearrest symptoms • ECG • Serum markers 	<ul style="list-style-type: none"> • MONA, pressors • Emergent PTCA • Empiric rt-PA • Balloon pump • CABG
Tamponade, cardiac <ul style="list-style-type: none"> • Trauma • Renal failure • Chest compressions • Carcinoma • Central line perforations 	<ul style="list-style-type: none"> • Risk factors • History • Prearrest picture • Distended neck veins • Echo 	<ul style="list-style-type: none"> • Volume • Pericardiocentesis • Thoracotomy
Tablets (drug and toxin overdoses) <ul style="list-style-type: none"> • TCAs, phenothiazines • B-Blockers, calcium channel blockers • Cocaine, digoxin, aspirin, acetaminophen 	<ul style="list-style-type: none"> • Risk Factors • History and toxidrome 	<ul style="list-style-type: none"> • Specific antidotes • Bicarbonate • Glucagon, calcium • Long CPR • Cardiopulmonary bypass

CARDIOVASCULAR QUADRAD

FOUR DETERMINANTS OF CARDIAC OUTPUT

1. Heart Rate (Electrical System)
2. Pump Performance (Contractility)
3. Tank Size (vascular tone, resistance)
4. Tank Contents (volume)

*When multiple overlapping problems:

1. First correct rate problems
2. Second, correct tank problems with fluid (volume), and sometimes pressors (resistance)
3. Third, treat pump problems with pressors, inotropes or both

MAJOR ERRORS

1. Using fluids or pressors when hypotension is due to tachycardia or bradycardia.
2. Using pressors instead of volume when hypotension is caused by tank problems
3. Using fluids when tank is already full and the problem is due to the pump.

QUADRAD	ASSESS	MANAGE
Primary ABCD Survey		
<ul style="list-style-type: none"> Airway (C-Spine) 	<ul style="list-style-type: none"> Open? Suspicious mechanism 	<ul style="list-style-type: none"> Chin life, jaw thrust; head tilt Chin life, ;jaw thrust; no head tilt, stability; backboard; immobilize
<ul style="list-style-type: none"> Breathing 	<ul style="list-style-type: none"> Moving Air? 	<ul style="list-style-type: none"> Give 2 breaths, obstructed airway protocols
<ul style="list-style-type: none"> Circulation 	<ul style="list-style-type: none"> Pulse? 	<ul style="list-style-type: none"> Chest compressions - Basic CPR
<ul style="list-style-type: none"> Defibrillation 	<ul style="list-style-type: none"> Attach AED; paddles; monitor 	<ul style="list-style-type: none"> Shock if VF or pulseless VT
Secondary ACD Survey		
<ul style="list-style-type: none"> Airway (C-spine) 	<ul style="list-style-type: none"> Adequate? Gag? Pooled secretions? Noisy breathing 	<ul style="list-style-type: none"> Remove obstructions; suction; Oropharyngeal airway; nasal trumpet endotracheal intubation; nasotracheal intubation; surgical airway
<ul style="list-style-type: none"> Breathing (OXYGEN) 	<ul style="list-style-type: none"> Visualized cords? Tube in? Chest x-ray, pneumothorax? Flair chest? Open chest? Cyanosis? Moving air? Order ABG, attach O2 saturation monitor 	<ul style="list-style-type: none"> Adjust ETT, needle decompression, chest tube, cover release sucking chest wound, order ventilator Provide oxygen via nasal cannula, face mask, nonrebreather mask, use hyperventilation and PEEP
<ul style="list-style-type: none"> Circulation (IV-Monitor-Fluids) 	<ul style="list-style-type: none"> HR? BP? Monitor; assess rhythm Send blood for type and crossmatch; labs (A,A,A,A,B,B, Tox) (Alc, ASA, APAP, amy Bhcg, Bili 	<ul style="list-style-type: none"> Start IV; give rhythm appropriate medications; order fluids plus rate; order blood; stop visible hemorrhage serious hematocrits, If pregnant, place on left side.
QUADRAD	ASSESS	MANAGE
<ul style="list-style-type: none"> Differential 	<ul style="list-style-type: none"> Assess with the D- 	<ul style="list-style-type: none"> Begin managing

Diagnosis (THINK!)	CP-D-E-E-F-F-F-G-G system <ul style="list-style-type: none"> Consider differential diagnosis by H's and T's 	identified diagnosis
Disability - D-CP-D	<ul style="list-style-type: none"> Mental Status? Pupil response? GCS; best eye, vocal, motor response For altered mental status use "Coma Protocol" and reassess The same regimen can be remembered as DONT (Dextrose, D50W; Oxygen, Narcan, Thiamine) 	<ul style="list-style-type: none"> Coma Protocol, Give D50, thiamine 100mg IV, give narcan 2mg IV check oxygenation Assess response
Expose - examine - extremities	<ul style="list-style-type: none"> Completely expose patient; perform quick visual check for gross injuries, pregnancy, signs of skin lesions, skin temp, medic alert. Check extremity pulses 	<ul style="list-style-type: none"> Stabilize obvious injuries; restore pulse to compromised extremities
Finger - Foley - flip	<ul style="list-style-type: none"> Perform rectal, vaginal exam; check for injuries to pelvis, perineum or genitalia, then insert Foley Flip: log-roll patient to check back areas 	<ul style="list-style-type: none"> Foley to straight drainage; send for analysis, including Tox; observe urine output rate
Gastric tube - gunk	<ul style="list-style-type: none"> Check contraindications to nasal insertion Observe aspiration for blood, pills, odors 	<ul style="list-style-type: none"> Instill gunk (activated charcoal 50g plus cathartic) down gastric tube for suspected drug Ods <p>Note that several toxicology councils consider gastric lavage of little value</p>
QUADRAD	ASSESS	MANAGE
History	<ul style="list-style-type: none"> Take expanded history in increase differential diagnosis 	<ul style="list-style-type: none"> History delayed until patients stabilized. Use family friends, EMS personnel

<ul style="list-style-type: none"> • O2-IV-Monitor-Fluids 	<ul style="list-style-type: none"> • Assess response to both surveys, check labs, x-rays 	<ul style="list-style-type: none"> • Continue management of identified diagnoses
<ul style="list-style-type: none"> • Temp-BP-HR-Respirations 	<ul style="list-style-type: none"> • Assess excessive low or high temperatures 	<ul style="list-style-type: none"> • Continue management as indicated
<ul style="list-style-type: none"> • Tank-tank-pump-rate 	<ul style="list-style-type: none"> • Consider problems in these categories 	<ul style="list-style-type: none"> • Continue management as indicated

COMPLICATED ACUTE MYOCARDIAL INFARCTION

I. CARDIOGENIC SHOCK

- A. CHF is the most common cause of mortality following MI (10-12% of MI patients present in shock, with a 70-80% mortality rate.
- B. Thrombolytic therapy is less effective probably secondary to hypotension, poor perfusion and decreased coronary flow
- C. Best Outcome - IABP, Aggressive Medical Therapy and Primary PTCA
- D. Patients with shock (SBP <100) and tachycardia (>100) should be transported to interventional facilities
- E. *Thrombolytic therapy should not be withheld pending transfer unless contraindications exist

II. INFERIOR WALL INFARCTION - RIGHT VENTRICULAR INFARCTION

- A. Right Coronary Artery is dominant in most patients
- B. Impairs flow to the Right Ventricular Marginal Branch, causing ischemia and RV infarction
- C. RV infarction is present in one third of inferior wall infarcts
- D. ½ of these patients have hemodynamic instability
- E. RV infarction doubles the MI mortality rate

Right Ventricular Triad

- 1) Clear Lungs
- 2) Jugular Vein Distention
- 3) Hypotension

*All patients with inferior wall infarct should have right-sided ECG done

- 1mm ST elevation in V4R is the single most predictive finding for RV ischemia/infarction
- Causes RV dilation (limited by pericardium)
- Both systolic and diastolic dysfunction

TREATMENT

- Support of preload is important to maintain cardiac output

- Factors that decrease preload (Nitrates, Morphine, Volume Depletion, diuretics) may worsen hemodynamics
- Increase preload and decrease afterload
- Inotropic support (Dobutamine) if 1-2 liters of NSS fails to correct hypotension
- Prompt cardioversion of atrial fibrillation if hemodynamics are compromised.

III. HEART BLOCK WITH INFERIOR WALL INFARCTION

- A. 2nd and 3rd degree blocks occur in 20% of inferior wall infarcts
- B. 2/3 within the first 24 hours
- C. Most respond to Atropine and are not hemodynamically significant
- D. Heart Block is associated with increased mortality (not directly related to the heart block but because it is usually related to larger infarction size)

IV. HYPOTENSION/CONGESTIVE HEART FAILURE

- A. Left ventricular dysfunction
- B. Combination of MI and ischemia results in decreased systolic dysfunction
- C. Nitrates, Diuretics, Morphine, oxygen
- D. Angiotensin converting enzymes (ACE) inhibitors are effective afterload reducer which has been shown to decrease mortality and attenuate myocardial remodeling
- E. Beta Blockers benefit mild to moderate heart failure with infarction by blunting excess catecholamine effects during infarction
- F. Diuretics must be monitored closely. Hypotension and severe heart failure are contraindications.

IV. MECHANICAL COMPLICATIONS

- A. Cardiac rupture is the second most common cause of in hospital mortality.
- B. Most commonly occur on day 5-7 but quicker (24-48 hours) in patients who have received thrombolytics
- C. Presence of new murmur should raise suspicion
- D. Parasternal thrill (if the rupture involves the septal wall)
- E. Portable Echo
- F. LV free wall rupture is usually fatal and presents with chest pain and PEA
- G. CT surgery is difficult but can be life saving

V. POST INFARCTION ANGINA

- A. Recurrent chest pain with ST elevation means infarction extension
- B. The patient's mortality is more than doubled
- C. Heart failure in these patients is common

- D. Patients should receive Heparin (PTT 50-70)
- E. Expeditious coronary angiography
- F. Raise suspicion of pericarditis and pulmonary emboli

VI. VENTRICULAR ARRHYTHMIAS

- A. Ventricular fibrillation increases short term but not long term mortality
- B. Following primary VF, Lidocaine is the drug of choice
- C. Amiodarone for non arrest rhythms or frequently recurring VF or hemodynamically significant VT

VII. POTASSIUM/MAGNESIUM

- A. Low Potassium is associated with increased ventricular arrhythmias
- B. Early trials show mortality benefit from Magnesium (reduced CHF) due to its myocardial protectant properties

CARDIOVASCULAR EMERGENCIES (STATION ONE)

I. RISK FACTORS FOR HEART DISEASE

- Male gender
- Smoker
- Hypertension
- Middle Age

II. SUDDEN DEATH ASSOCIATED WITH CAD >80% of patients, Ventricular Fibrillation being the most common rhythm

- A. Most Prehospital AMI deaths are related to VF
- B. Most VF deaths occur during the first hour of symptoms
- C. In hospital deaths often occur secondary to decreased cardiac output in the first 24-48 hours of treatment
- D. Mortality is directly related to the size of infarction
- E. Effective 911 system is the first step to improved survival.

III. EMS PERSONNEL

- A. Trained and equipped to recognize and treat VF
- B. Can start MONA (Morphine, Nitro, Oxygen and ASA)
- C. Early Emergency Department Notification
- D. 12-Lead ECG for diagnosis
- E. Thrombolytic Screening

IV. EMERGENCY DEPARTMENT AMI PROTOCOL

- A. ECG screening within 10 minutes
- B. Door to Drug (Thrombolytics) 30 minutes
- C. Door to Balloon Time = 90 minutes
- D. Oxygen, IV, Monitor and Morphine
- E. Reperfusion for ST segment elevation
- F. Rule out contraindications
- G. Consider PTCA if ineligible for thrombolytics
- H. Angiography for cardiogenic shock
- I. ASA
- J. Beta Blockade (all patients without contraindications)
- K. IV Nitroglycerin for initial 24-48 hours in patients with AMI and CHF, large anterior wall infarction, persistent ischemia or HTN
- L. Serum Markers
- M. Electrolytes; coagulation studies
- N. Chest x-ray

V. ELECTROCARDIOGRAM

- A. ST Elevation - high specificity for evolving MI; assess for reperfusion
- B. ST Depression - Consistent with/strongly suggestive of ischemia; defines a high risk subset of patients with non-Q wave MI or unstable angina
- C. Non Diagnostic or Normal ECG - further assessment is usually needed; evaluation protocols may include repeat ECG or continuous ST monitoring; serial cardiac markers, myocardial imaging or 2D echo.

VI. CORONARY ARTERY DISTRIBUTION

A. Left Side

- 1. Septal Wall of the Left Ventricle
- 2. Anterior and Lateral Walls of the LV
- 3. Inferior Wall (10% of patients)
- 4. Both Bundle Branches

B. Right Side

- 1. Inferior wall of the Left Ventricle
- 2. Posterior wall of the LV (90% of patients)
- 3. AV Node (90% of patients)
- 4. Right Ventricle

VII. ASSOCIATED CONDUCTION PROBLEMS

A. Left Side Infarction

- 1. Advanced (2nd or 3rd) Degree heart blocks with wide QRS which tends to degrade faster or to convert to asystole without warning
- 2. Bundle Branch Blocks
- 3. TCP is treatment of choice for wide QRS heart blocks

B. Right Side Infarction

- 1. Heart Block, commonly AV nodal, transient, responds well to Atropine.
- 2. Always consider Right Ventricular Infarction

*Inferior Wall Myocardial Infarction can be caused by either RCA (90%) or dominant circumflex artery occlusion.

VIII. RIGHT VENTRICULAR INFARCTION

- A. Recognize clinical clues - need for right ECG leads
- B. Avoid nitrates and other vasodilators (may cause hypotension)

- C. Thrombolytics and PTCA improve right ventricular ejection fraction and decrease the incidence of heart block
- D. PTCA is indicated for shock, in patient's age <75 years.
- E. If hypotension - 1-2 liters NSS in 250-500cc boluses
- F. Dobutamine is particularly effective if hypotension persists
- G. IABP if concomitant LV dysfunction

IX. ARRHYTHMIA MANAGEMENT

- A. AV synchrony is important - cardiovert hemodynamically unstable atrial fibrillation.
- B. AV synchronous pacing may be efficacious

X. TREATMENTS TO CONSIDER

- A. Oxygen
- B. Nitroglycerin
- C. Analgesia
- D. ASA
- E. Beta Blockers
- F. ACE Inhibitors
- G. Magnesium
- H. Lidocaine
- I. Reperfusion (PTCA or Thrombolytics)

PHARMACOLOGY

NITROGLYCERIN

MECHANISMS OF ACTION

- Dilates coronary arteries
- Suppresses coronary artery spasm
- Increases collateral blood flow
- Analgesic
- Reduces myocardial oxygen use
- Reduces LV workload - decreases afterload

ADMINISTRATION

- SL, spray (avoid topical or long acting nitrates)
- IV 10-20 ug/min; limit SBP drop to 10% if normotensive, 30% if hypertensive

INDICATIONS

- Suspected ischemic chest pain
- Unstable angina
- Acute pulmonary edema
- Hypertension in AMI

CAUTIONS

- Caution if SBP >90mm Hg
- Beware in Right Ventricular Infarction
- Hypovolemic patients are prone to severe hypotension

MORPHINE SULFATE

EFFECTS

- Analgesia plus venodilation reduces ventricular preload and oxygen requirements

INDICATIONS

- Treatment of ischemic pain not relieved by nitroglycerin; also useful to redistribute blood volume in patients with pulmonary edema.

CAUTIONS AND COMPLICATIONS

- Do not use in patients with suspected hypovolemia
- If hypotension develops, elevate the patient's legs and administer normal saline

ASPIRIN

EFFECTS

- I. 160-325mg causes immediate and near-total inhibition of thromboxane A₂ production. Reduces coronary reocclusion and recurrent events after Thrombolytic therapy. Also important for treatment of unstable angina

INDICATIONS:

- II. All patients with suspected acute coronary syndromes
- III. Particularly reperfusion candidates, unless hypersensitivity to ASA

DOSE/ROUTE

- I. ASA is absorbed faster when chewed than when swallowed in the early hours after infarction, particularly if morphine has been given.
- II. ASA suppositories (325mg) can be used safely and are recommended for patients with severe nausea, vomiting, or upper gastrointestinal disorders

BETA BLOCKERS

MECHANISM OF ACTION

- I. Block sympathetic nervous system stimulation of heart rate and contractility
- II. Block B₂ mediated sympathetic vasodilation, resulting in vasoconstriction and increased vascular resistance.

EFFECTS

- I. Can reduce infarction size in patients not receiving Thrombolytic therapy.
- II. Reduce short term mortality
- III. In patients receiving Thrombolytic therapy, they decrease post infarction ischemia and nonfatal MI
- IV. The incidence of VF is reduced

INDICATIONS

- I. All pts with Q-wave infarction without contraindications
- II. All patients with non-Q-wave infarction without contraindications
- III. Treatment of recurrent or continuing pain associated with AMI

- IV. Tachyarrhythmias associated with catecholamine excess (i.e. atrial fibrillation)

ABSOLUTE CONTRAINDICATIONS

- I. Severe congestive heart failure
- II. Hypotension (SBP <100mm Hg)
- III. Bronchospasm
- IV. 2nd or 3rd degree AV Block

CAUTIONS

- I. Mild/moderate CHF
- II. Bradycardia (HR <60 bpm)
- III. History of Asthma
- IV. IDDM
- V. Severe peripheral vascular disease

HEPARIN

MECHANISM OF ACTION

- I. Indirect thrombin inhibitor (at AT III)

INDICATIONS

ST ELEVATION INFARCTION

- II. Adjunct to Thrombolytic therapy with fibrin specific agents
- III. Adjunct for PTCA
- IV. High Risk for systemic emboli (large anterior wall, atrial fibrillation, previous embolus, known LV thrombus)

ST DEPRESSION INFARCTION/UNSTABLE ANGINA

- I. Conjunction with ASA
- II. Recommended for all High Risk Patients

ACE INHIBITORS

EFFECTS

- I. Early oral ACE inhibition reduces mortality
- II. Reduces CHF associated with MI
- III. Prevent adverse LV remodeling, delay progression of heart failure, and decrease sudden death and recurrent MI

INDICATIONS (Greatest benefit in patients with)

- I. Anterior infarction
- II. Prior infarction
- III. Heart failure
- IV. Clinical signs of left ventricular dysfunction (ejection fraction 40%)

DOSE, ADMINISTRATION

- I. Not administered during the first 6 hours after MI
- II. Once stable a low oral dose is initiated and titrated to the full dose within 24 to 48 hours

POTASSIUM AND MAGNESIUM SULFATE

EFFECTS

- I. Potassium deficiency but not magnesium deficiency associated with arrhythmias, sudden death
- II. Recent studies suggest no reduction in AMI mortality with Magnesium administration

RECOMMENDATIONS

- I. Correction of documented deficits, especially in patients receiving prior diuretics
- II. Episodes of torsades-de-pointes-type VT associated with a prolonged QT interval.
- III. Use in High Risk patients with MI, such as those not receiving thrombolysis or the elderly
- IV. Routine use in AMI is not recommended

THROMBOLYTIC THERAPY FOR ACUTE CORONARY SYNDROMES

INDICATIONS

- I. Chest pain suggesting MI
- II. ST segment elevation $<0.1\text{mV}$ in 2 or more contiguous ECG leads or new or presumably new bundle branch block
- III. Time to therapy <12 hours
- IV. Age <75 years

ABSOLUTE CONTRAINDICATIONS

- I. Previous hemorrhagic stroke

- II. Other stroke or cerebrovascular accident within 1 year
- III. Active internal bleeding (menses excluded)
- IV. Suspected aortic dissection

RELATIVE CONTRAINDICATIONS AND CAUTIONS

- I. Severe uncontrolled HTN
- II. Current use of anticoagulants (INR >2.5); known bleeding disorder
- III. Recent trauma (within 2-4 weeks) including head and traumatic CPR or major surgery <3weeks.
- IV. Recent (2-4 weeks) internal bleeding; active peptic ulcer disease
- V. Pregnancy
- VI. For streptokinase allergy or prior exposure

PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY

** In experienced centers PTCA can restore vessel patency and normal flow the a 90% success rate.

Primary PTCA is most effective for the following:

- I. Cardiogenic shock, age <75 years if performed <18 hours from onset of shock and <36 hours from onset of ST elevation.
- II. As an alternative to thrombolytic therapy in acute ST elevation or Q wave or new LBBB MI patients, if performed <12 hours from onset of pain
- III. In patients with indications for reperfusion but with a contraindication to thrombolytic therapy

Best results are achieved at PTCA Centers with the following Characteristics:

- I. Center volume >200 procedures per year
- II. Individual operator volume of >75 procedures per year
- III. Balloon dilation within 90 minutes of AMI diagnosis

RECOMMENDATIONS FOR INTRA-AORTIC BALLOON COUNTERPULSATION

- I. Stabilize cardiogenic shock for angiography and revascularization
- II. Acute mitral regurgitation or VSD pending angiography and repair
- III. Recurrent intractable ventricular arrhythmia with hemodynamic instability
- IV. Refractory post-MI angina pending angiography

May be helpful with:

- I. Hemodynamic instability, poor LV function, persistent ischemia with large area of myocardium at risk; reperfusion not possible or anticipated.

SERUM MARKERS

Blocked blood flow to the myocardium leads to cellular (myocytes) ischemia, which causes injury and may result in cell death or necrosis with loss of cell membrane integrity and release of cellular macromolecules

New serum markers achieve earlier diagnosis with greater sensitivity (able to detect very small amounts of markers) and specificity (markers are never elevated unless cell death has occurred).

Serum markers can now differentiate between non-Q-wave MI (serum markers released) and unstable angina (no serum markers released)

Serum markers can provide useful risk stratification and prognostic information.

ELECTROLYTE ABDNORMALITIES LEARNING STATION TWO

I. POTASSIUM

- I. Affects cardiovascular activity and is essential for muscle and neurological function
- II. Small changes have large consequences
- III. Of all electrolytes rapid changes in K⁺ cause most life threatening consequences
- IV. K⁺ is a major intracellular cation
- V. Electrical charge across cell membrane accounts for function. Magnitude of charge is determined by transmembrane K⁺ gradient
- VI. Primary source is food. Average intake 1mEq/kg/day
- VII. May reduce by promoting intracellular shift or renal excretion or by reducing GI absorption
- VIII. Cardiovascular effects of K⁺ are reflected in ECG and by direct measurement of serum

HYPERKALEMIA

- I. Normal 3.5-5 mEq/L
- II. Serum Hyperkalemia does not imply a total body excess of K⁺
- III. Neurons and muscles are most affected by Hyperkalemia
- IV. Suggested by ECG Changes
 - A. Peaked T-waves
 - B. Flattened P waves
 - C. Prolonged PR interval
 - D. Widened QRS Complex
 - E. Deepened S wave
 - F. Atrioventricular Rhythm
 - G. Sine-wave formation
 - H. Ventricular Fibrillation – cardiac arrest

SYMPTOMS

- I. Weakness
- II. Ascending paralysis
- III. Respiratory Failure

CAUSES

- I. Diet
- II. Medication
- III. Renal Failure
- IV. Redistribution fall of pH
- V. Aldosterone Deficiency
- VI. Pseudohyperkalemia (Hemodialysis)

TREATMENT

- I. Remove the source or cause of Hyperkalemia
- II. Stabilize myocardial cells (calcium antagonizes potassium effect)
- III. Chemically shift potassium intracellularly
 - A. Glucose and insulin
 - B. Sodium Bicarbonate
 - C. Beta Blockers

- I. Physically remove excess K+
 - A. Diuretics or dialysis
 - B. Ion-exchangeresins

HYPOKALEMIA

Defined as a low measured serum potassium.

- II. Normal = 3.5- 5.0 mEq/L
- III. Serum hypokalemia does not imply a total body deficit
- IV. Neurons and muscle are the cells most affected by hypokalemia
- V. Serum hypokalemia may be suggested by ECG changes
 - A. U waves
 - B. T-wave flattening
 - C. ST segment changes
 - D. Cardiac arrhythmias, especially if the patient is receiving digitalis

SIGNS AND SYMPTOMS

- I. Neuromuscular symptoms
 - A. Weakness
 - B. Fatigue
 - C. Paralysis
 - D. Respiratory Difficulty
 - E. Rhabdomyolysis
- II. Gastrointestinal Symptoms:
 - A. Constipation
 - B. Ileus

- III. Nephrogenic Diabetes Insipidus

CRITICAL ACTIONS

- I.** Assess the Five Quadrads
- II.** Obtain and evaluate the ECG
- III.** Obtain a stat serum potassium and other electrolytes
- IV.** Obtain a stat blood gas if there is a question of pH disturbance
- V.** Obtain a stat CBC and hematocrit
- VI.** Begin directed resuscitation or intervention

- VII.** Reassess patient status and electrolytes frequently
- VIII.** Obtain additional history and ancillary studies as time and conditions allow
- IX.** Prepare for transport to the critical care unit, admission, or discharge as determine by response to intervention

TREATMENT

- I. Give potassium
 - A. Orally
 - B. Intravenously (Decreased renal output)
 - C. Estimate total body K⁺ deficit
 - 1. Dependent on age and muscle mass
 - 2. Rough Estimate: Decrease in serum K⁺ is equivalent to a deficit of 150-400 mEq/L of total body K⁺ in a steady state

SODIUM

Is the major ECF cation. Total body sodium is normally half of the total K⁺ content. Because it is extracellular it is more accessible than other electrolytes to rapid and dramatic changes in concentration and quantity by the renal and GI system. Governed by renin-angiotensin, aldosterone and antidiuretic hormones affecting primarily the kidneys.

CRITICAL KNOWLEDGE

- I.** Normal range for Sodium 135-145 mEq/L
- II.** Extracellular fluid volume is affected by total body sodium
- III.** Serum sodium concentration is affected by extracellular water volume
- IV.** Total body sodium cannot be determined from the serum sodium concentration (this is estimated in conjunction with the physical examination)
- V.** Changes in serum sodium concentration may suggest a change of total body sodium in a steady state
- VI.** Serum osmolality is a measure of the solute concentration in the ECG. Serum tonicity is a measure of the driving force for water of the active solutes across the cell membrane.
- VII.** Rapid changes in serum sodium are accompanied by similar changes in K⁺
- VIII.** The physical condition of the patient reflects changes in serum sodium concentration. The more rapid the changes, the more striking the physical changes.
- IX.** There are no significant ECG changes with hypernatremia or hyponatremia.

CRITICAL ACTIONS

- I.** Assess the Five Quadrads
- II.** Stabilize the patient's vital signs

- III. Measure serum and urine sodium concentration
- IV. Measure other serum and urine electrolytes
- V. Estimate and measure the serum and urine osmolality
- VI. Obtain and Evaluate ECG
- VII. Consider possible means of altering factors causing the sodium abnormality.

HYPERNATREMIA

Excess of sodium. Caused by an excess of sodium relative to water.

Excessive water losses most often caused by renal and gastrointestinal routes. Most physical manifestations expressed as changes in the patient's neurological status.

CRITICAL KNOWLEDGE

- Normal range = 135-145
- Major reasons are impaired thirst mechanisms, chronic debilitating illnesses, disordered or altered mental status, primary neurological disorders or lack of access to water.
- Treatment consists of replacing water and sodium to avoid rapid fall in serum sodium and osmolality and resultant extravascular fluid shifts and cerebral edema fluids.

HYPONATREMIA

Low measured serum sodium. Caused by an excess of water relative to sodium. Most cases are caused by reduced renal water excretion in the presence of continued water intake. Severe, rapid onset usually results in neurological symptoms, slower onset causes changes in the respiratory and cardiovascular systems.

TREATMENT

Aimed at finding the causes while treating the symptoms. The history and physical exam are best resources for determining the etiology, time of onset and volume status.

CALCIUM

Most abundant mineral in the body and is essential for skeletal support and neuromuscular function. Also extracellular cation and is actively pumped out of the cells with sodium and therefore able to antagonize both potassium and magnesium.

CRITICAL KNOWLEDGE

- Normal range - 8.5 to 10.5 mg/dL
- Total serum calcium is a balance between GI absorption, renal excretion and skeletal mass exchange
- Calcium and sodium both exchange with potassium in the renal tubules during reabsorption

CRITICAL ACTIONS

- Assess the Five Quadrads
- Determine serum pH, calcium, magnesium, albumin, BUN, creatinine, and electrolyte concentrations
- Obtain and evaluate the ECG
- Begin directed intervention and treatment of the abnormality
- Obtain additional history, laboratory studies and monitoring data as indicated

HYPERCALCEMIA

Primary hyperparathyroidism and malignancy account for more than 90% of the reported cases and are most often evident from history and physical. Primary disorder is due to increased entry of calcium from the skeletal reservoir and GI tract with decreased renal clearance.

CRITICAL KNOWLEDGE

- Normal range = 8.5 to 10.5 mg/dL
- Symptoms will appear at 12-15mg/dL
- CNS effects are due to decreased permeability of membranes
- Depression, weakness, fatigue, and confusion occur with mild elevations of calcium
- Hallucinations, disorientation, hypotonicity and coma develop at critical levels
- Cardiac effects are due to alteration in action potential.
- Contractility increases until 15-20 mg/dL after which myocardial depression occurs. Automaticity is decreased
- Ventricular systole is shortened and arrhythmias occur
- ECG Changes
 - Shortened QT intervals
 - Prolonged PR and QRS intervals
 - Increased QRS voltage
 - T-wave flattening and widening
 - Notching of QRS
 - AV Block progressing to arrest
- GI effects include dysphagia, constipation, peptic ulcers and pancreatitis
- Vascular effects cause hypertension

TREATMENT

- Begin treatment at 12mg/dL
- Start large bore IV
- Infuse NSS at 300-500cc to correct fluid deficits, restore filtration and promote calcium excretion
- Continue NSS at 100-200cc/hr
- Consider Plicamycin, calcitonin, glucocorticoids, or oral phosphates as alternatives

HYPOCALCEMIA

Most causes are dependent on absorptive, excretory, and hormonal functions.

CRITICAL ACTIONS

- Assess five quadrads
- Large bore IV
- Obtain and evaluate ECG
- Treat acute symptomatic hypocalcemia with 10% calcium gluconate 90-180 mg IV over 10 minutes
- Measure response every 4-6 hours
- Correct magnesium, potassium and pH abnormalities

MAGNESIUM

Fourth most abundant mineral in the body. It is primarily an intracellular cation and participates in a host of enzymatic actions and hormonal actions, most involving energy production through glycolysis and adenosine phosphorylation.

CRITICAL KNOWLEDGE

- Normal 1.3 - 2.2 mEq/L
- Fifty percent is found in bone and in insoluble form
- Majority in total body water is intracellular
- Renal excretion is normal.
- Magnesium is necessary for adequate energy production

CRITICAL ACTIONS

- Assess the five quadrads
- Obtain definitive history
- Obtain and evaluate ECG
- Begin directed intervention and treatment

HYPERMAGNESEMIA

Clearance is through the kidneys. Usually does not occur without the presence of some renal insufficiency. Symptoms include muscular weakness. ECG changes are reflected in electrolyte shifts of potassium.

CRITICAL KNOWLEDGE

- Concentration > 2.2

CRITICAL ACTIONS

- Assess the five quadrads
- Obtain magnesium and electrolyte levels
- Obtain a current history
- Perform a physical exam
- Obtain and evaluate ECG
- Begin directed intervention and treatment
- Reevaluate frequently to assess treatment response

HYPOMAGNESEMIA

Most often due to decreased absorption or increased renal, GI loss or secondary to alterations of parathyroid hormone. Symptoms include muscular irritability, altered mentation, and atrial and ventricular rhythm disturbances. Treatment consists of supplementation given IV or IM

CRITICAL KNOWLEDGE

- Concentration <1.4 mEq/L
- ECG changes are
 - Prolongation of PR and QT intervals
 - Nonspecific depression of ST interval
 - Flattening or inversion of precordial P waves
 - Widening of QRS
 - Torsades de pointes
 - Atrial Arrhythmias
 - Worsening of digitalis toxicity

CRITICAL ACTIONS

- Assess five quadrads
- Obtain history
- Obtain and evaluate ECG
- Immediately correct severe hypomagnesemia IV or by IM injection
- Definitive treatment is determined by history and diagnosis
- Reevaluate frequently

EMERGENCY TREATMENTS FOR HYPERKALEMIA

Therapy	Dose	Effect Mechanism	Onset of Effects	Duration of Effects
Sodium Bicarbonate	1mEq/kg IV bolus	Shifts	5-10 minutes	1-2 hours
Calcium Chloride	5-10 mL IV 10% Solution (500-1000mg)	Antagonize	1-3 minutes	30-60 minutes
Insulin plus Glucose	Regular insulin 10 U IV plus 1 ampule glucose	Shifts	30 minutes	4-6 hours
Diuresis with Lasix	40-80 mg IV bolus	Removes	When diuresis	When diuresis ends
Cation-exchange resin	Kayexalate 15-50 g PO or PR plus sorbitol	Removes	1-2 hours	4-6 hours
Peritoneal dialysis Hemodialysis	Per institution	Removes	As soon as started	Until dialysis completed

NORMAL VALUES

SERUM VALUES	
Sodium	135-145 mEq/L
Potassium	3.5-5.0 mEq/L
Chloride	95-105 mEq/L
Bicarbonate	24-26 mEq/L
Osmolality	280-295 mEq/L
Osmolal gap	<10 mOsm/L
Anion Gap	9-16 mEq/L
Urea	10-20 mg/dL

Clinical Problem	Typical ECG Findings	Treatment Approaches
Hyperkalemia	• Tall peaked T waves	• Sodium Bicarb

ECG changes are listed in order of increasing K ⁺	<ul style="list-style-type: none"> • Prolonged PR • Prolonged QT • P waves diminished • ST segment depression • Sine-wave PEA • Wide complex tachycardias 	<ul style="list-style-type: none"> • 1mEq/kg IV bolus • Calcium chloride 5-10 mlIV • Reg insulin 10U IV plus 1 amp D50 • Furosemide 40-80mg IV • Kayexalate 15-50 g PO/PR plus sorbitol • Peritoneal dialysis or hemodialysis
Hypokalemia	<ul style="list-style-type: none"> • U waves become prominent • T waves flatten • ST segment becomes depressed • QT interval prolonged • QRS widens • Wide complex tachycardias 	<ul style="list-style-type: none"> • If 3-3.5 need 100mEq replacement • If 2.5-3.0 need 200 mEq replacement • Infuse 10-40 mEq/h
Hypercalcemia Normal total calcium 8.5-10.5 mg/dL Normal ionized calcium = 4.2-4.8	<ul style="list-style-type: none"> • QT intervals markedly shortened • Automaticity decreased • ST segments shortened and depressed • T waves widen • Bundle branch blocks may occur • Second degree heart block 	<p>Urgent Treatment</p> <ul style="list-style-type: none"> • NS bolus: induce diuresis • Lasix 40-100 mg IV • Replace K⁺ and mag <p>CARDIAC ARREST</p> <ul style="list-style-type: none"> • ACLS plus • Magnesium sulfate 1-2 g IV • K⁺ 1-2 mEq/min • NS bolus • Diuretics
Hypocalcemia Normal total calcium = 8.5 - 10.5 mg/dL Normal ionized calcium= 4.2-4.8 mg/dL	<ul style="list-style-type: none"> • Prolonged QT • May have VT or torsades 	<p>Urgent Treatment</p> <ul style="list-style-type: none"> • Calcium Chloride 10% (1 g in 10ml = 272 mg) Give 100mg in 100cc D5W in 10-20 minutes • IN the next 6-12 hrs give a total of 1 gram by infusion <p>Cardiac Arrest</p> <ul style="list-style-type: none"> • Full ampule of calcium chloride
Hypomagnesemia Normal = 1.5-2.0 mgEq/L Hypomag <1.4	<ul style="list-style-type: none"> • Prolonged PR and QT • Wide QRS • ST depression • Broad flat T wave 	

Severe <0.5		
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ELECTROLYTE PROBLEMS AND ECG ABNORMALITIES

Electrolyte Problem	Typical ECG Abnormalities
Hyperkalemia	<ul style="list-style-type: none"> • Prolonged PR • P waves almost disappear • Tall, peaked T waves • ST segment depression, S and T waves merge • Sine-wave PEA, wide complex tachycardia
Hypokalemia	<ul style="list-style-type: none"> • U waves become more prominent • T waves flatten • ST segment becomes depressed • QT interval becomes more prolonged • Wide complex tachycardias
Hypercalcemia	<ul style="list-style-type: none"> • Key: QT interval markedly shortened • Automaticity decreased • ST segments shortened and depressed • T waves widen • BBB • Second degree heart block
Hypocalcemia	<ul style="list-style-type: none"> • Prolonged QT interval due to prolonged ST segment • May experience VT or torsades de points
Hypomagnesemia	<ul style="list-style-type: none"> • Prolonged PR and QT intervals • Wide QRS complex • ST depression • Broad, flat T waves with precordial T wave inversion

ECG ABNORMALITIES AND CLINICAL PROBLEMS

TYPICAL ECG ABNORMALITIES	CLINICAL PROBLEMS
P waves diminished	<ul style="list-style-type: none"> • Hyperkalemia
PR intervals prolonged	<ul style="list-style-type: none"> • Hyperkalemia • Hypomagnesemia • Hypercalcemia • B blockers • Calcium channel blockers • Tricyclic antidepressants • Neuroleptics
QRS Widened	<ul style="list-style-type: none"> • Hypomagnesemia • Hypercalcemia • Beta Blockers • Tricyclic antidepressants
QT Interval Prolonged	<ul style="list-style-type: none"> • Hyperkalemia

	<ul style="list-style-type: none"> • Hypocalcemia • Tricyclic Antidepressants • Neuroleptics • Calcium Channel Blockers
QT Interval Shortened	<ul style="list-style-type: none"> • Hypercalcemia
ST Segment Depression	<ul style="list-style-type: none"> • Hyperkalemia
ST Segment Shortened	<ul style="list-style-type: none"> • Hypercalcemia • Beta Blockers
T waves tall and peaked	<ul style="list-style-type: none"> • Hyperkalemia • Beta Blockers
T waves Wider	<ul style="list-style-type: none"> • Hypercalcemia
T waves flatter	<ul style="list-style-type: none"> • Hypomagnesemia
U waves appear	<ul style="list-style-type: none"> • Hypokalemia

RED FLAGS FOR ELECTROLYTE ABNORMALITIES

1. Abnormal vital signs
2. Altered mental status
3. History of
 - Prolonged chronic disease
 - Gastrointestinal or renal disease, malnutrition
 - Diabetes, other endocrinopathies
 - Cancer
 - Alcohol or drug abuse

Don't wait for results of laboratory analysis to identify an electrolyte abnormality - instead BE PROACTIVE LOOKING FOR problems

LEARNING STATION THREE ENVIRONMENTAL EMERGENCIES

1. Near drowning
2. Life threatening asthma
3. Hypothermia
4. Anaphylaxis
5. Lightning strike
6. Cardiac arrest associated with trauma
7. Cardiac arrest associated with pregnancy
8. Electric shock

I. NEAR DROWNING

- A. No modification of standard BLS is necessary
- B. In water resuscitation requires flotation devices and training.
External chest compression cannot be performed in the water
- C. Suspect spinal injuries and treat as a trauma patient
- D. Do not attempt to drain water from lungs
- E. Remove foreign bodies in the airway
- F. Vomiting and regurgitation are very common.
- G. All immersion victims who require resuscitation should be transported.

ADVANCED LIFE SUPPORT

- A. No modification of standard ALS is necessary
- B. Early trachea intubation is indicated to
 - Improve oxygenation, intermittent positive-pressure ventilation and reduce Paco₂
 - Suction the tracheobronchial tree
 - Apply continuous positive airway pressure (CPAP or PEEP)

PROGNOSTICS

- A. Predictors of outcome can be unreliable in the prehospital setting
- B. Patients reaching the hospital with a spontaneous circulation and breathing have good outcomes

DEFINITIONS:

DROWNING - refers to submersion that causes either immediate death or death within 24 hours

NEAR DROWNING - submersion that does not result in immediate death or death within 24 hours

The most impressive effects of submersion are those related to hypoxia, not the chemical composition of the submersion fluid (unless it is contaminated, i.e., septic tank)

PATHOPHYSIOLOGY

- A. Hypoxemia is the major insult; duration determines outcome
- B. Rule out associated conditions; trauma, alcohol intoxication, hypothermia
- C. Potential neurologic insults; hypoxia, trauma
- D. Potential pulmonary insults: pulmonary edema, intrapulmonary shunting, surfactant inactivation, ARDS, aspiration

II. LIFE THREATENING ASTHMA

Severe asthma can lead to several forms of sudden cardiac death.

- Severe bronchospasm, leading to asphyxia
- Tension pneumothorax, often bilateral
- Cardiac arrhythmias
- Use of B adrenergic agonists
- Hypotension and bradycardia mediated by vasovagal reflexes
- Cardiac conduction disease

Most deaths occur outside the hospital. The major clinical action should be aggressive treatment of all acute severe asthmatic crisis before deterioration to full arrest.

INTERVENTION KEYS

- A. Oxygen - use sufficient inspired oxygen to achieve a PaO₂ of 90mmHg. Use high flow oxygen by mask, be ready to intubate
- B. Nebulized B₂ Agonists - Metaproterenol has become the cornerstone of most therapy. Escalating doses of 5-10 mg every 15-20 mins up to three times.
- C. IV Corticosteroids - Begin corticosteroid therapy immediately with status asthmaticus. Methylprednisolone 2mg/kg as first dose repeated every 6 hours or Hydrocortisone 10mg/kg every 6 hrs.
- D. Nebulized Anticholinergic Therapy - Ipratropium bromide dose inhalers or as a moist nebulization at a dose of 0.5mg with B₂ agonists
- E. IV Aminophylline - used if B₂ or corticosteroids fail. Aminophylline may produce significant side effects. Loading Dose 5mg/kg given over 30-45 mins, followed by infusion of 0.5mg to 0.7mg/kg/hr
- F. Intravenous Magnesium Sulfate - not consistently effective
- G. IV B Agonists - Isoproterenol IV over several hours can be effective for severely ill patients. 0.1 ug/kg/min to max of 0.6 ug/kg/min
- H. Intravenous Epinephrine - Aggressive approach for life threatening situations. 2-10 ml of 1:10000 over five minutes and repeated in five minutes
- I. IV Sodium Bicarbonate - acidosis is known to counteract beneficial effects of sympathomimetic amines.

J. Tracheal Intubation

- Provide adequate sedation with ketamine
- Paralyze the patient
- Once intubated some patients can be effectively managed with permissive hypercarbia in which there is elective hypoventilation
- Inhaled, volatile anesthetics are powerful bronchial smooth muscle relaxants
- Ketamine
- Assisted exhalation or lung massage

III. HYPOTHERMIA

May increase interval that cardiac arrest and reduced blood flow during resuscitation can be tolerated. Severe hypothermia causes bradycardia and a slow ventilatory rate. It also can cause ventricular irritability

KEY INTERVENTIONS

- A. Prevent further heat loss due to evaporation from wet garments, cold environments and wind
- B. Cautiously transport, avoiding rough movement and excessive activity, which can cause VF. Do not delay urgently indicated procedures such as intubation or the introduction of intravascular catheters or a pacemaker.
- C. Patients with core temps <34 C can be rewarmed by internal active rearming.
- D. Because of the “after drop phenomenon” active external rearming should be performed to only truncal areas.

BLS MODIFICATIONS DURING ARREST

- A. Prevent further heat loss
- B. Avoid rough movement and excess activity
- C. Take up to 30-45 seconds to confirm pulslessness or apnea
- D. Do not attempt active external rearming
- E. Continue efforts until patient is rewarmed
- F. Successful resuscitation without neurologic problems have been reported after 70 minutes of arrest followed by two hours of BLS before active rearming was started by cardiopulmonary bypass.
- G. Some patients may be hypothermic because they have cooled down after having a normothermic arrest. Do not attempt resuscitation under these circumstances.

ALS MODIFICATIONS DURING ARREST

- A. May have reduced response to pacemaker stimulation, defibrillation and cardioactive drugs, drugs may accumulate to toxic levels
- B. If core temp is <30C give a max of 3 shocks for VF or VT until patient is warmed.
- C. Needle electrodes are preferred for ECG monitoring

- D. Active internal rewarming can be started in the field but must not delay transport
- E. Patients who have been hypothermic for hours may need large amounts of fluids during rewarming
- F. Cachectic, malnourished or alcoholic patients should receive Thiamine 100mg IV early during rewarming

Although hypothermia is a differential diagnosis, you must look beyond it for a reason for the exposure:

- Cardiac arrest may stop circulation before the victim cools
- Psychiatric disease, disorientation, drugs, or alcohol can impair thought
- Head trauma or other injury may immobilize the victim in the cold
- Alcohol produces vasodilation, which reduces the body's attempt to conserve heat.
- Victims of avalanches may have associated asphyxia and injury

MILD: 34 TO 36 C

- Lethargy
- Vasoconstriction
- Shivering
- Cold diuresis
- Increased Oxygen Demand

MODERATE: 30 to 34C

- Lethargy - stupor
- Dilated pupils
- Bradycardia, arrhythmias, hypotension
- Muscle rigidity, cessation of shivering
- Decreased O₂ demand

SEVERE: < 30 C

- Stupor - coma
- Dilated, non-reactive pupils
- Potential malignant arrhythmias
- Difficult to detect breathing
- Difficult to detect pulse, blood pressure

Standard gas thermometers do not read below 34C

ECG CHANGES

- Osborne or J waves
- T-wave inversion

- Prolonged PR, QRS and QT intervals
- Arrhythmias may include bradycardia, slow AF, VF or asystole

AFTER DROP PHENOMENON

1. Initial active external rewarming leads to;
2. Peripheral vasodilation (BP drops)
3. Cold blood from dilated peripheral vessels carries high lactic acid levels to core vessels
4. Cold acidotic blood causes drop in core temperature
5. Temperature drop and acidosis provoke serious arrhythmias

REWARMING METHOD BASED ON CORE TEMPERATURE

34C - 36C

- Passive rewarming (remove wet clothing, warm blankets)
- Active external rewarming (most convenient: IV solution bags heated in microwave, convective heating methods; (Bair blankets), radiant heat shield, convective heat (heating pads)

30C - 34C

- Passive rewarming (completely dried off; warm blankets)
- Active external rewarming to truncal areas only

<30C

- Active internal rewarming

IV. ANAPHYLAXIS

DEFINITIONS

- *Anaphylaxis* is usually used for hypersensitivity reactions mediated by IgE
- *Anaphylactoid reactions* are similar to, but do not depend on hypersensitivity.
- The manifestations and management of anaphylaxis and anaphylactoid reactions are similar so that the distinction is so unimportant in relation to the treatment of an acute attack.

SIGNS AND SYMPTOMS

Both may present clinically with angioedema, bronchospasm and hypotension.

- Some patients may die from acute irreversible asthma or laryngeal edema without having more generalized manifestations
- Other symptoms include urticaria, rhinitis, conjunctivitis, abdominal pain, vomiting, diarrhea and a sense of impending doom.

- The patient may appear either flushed or pale
- Cardiovascular collapse is the most common manifestation. It is caused by vasodilation and loss of plasma. Any cardiac dysfunction is due principally to hypotension and underlying disease, or epinephrine that has been administered.

Most common causes

- Insect stings
- Drugs
- Contrast Media
- Some Foods
- Peanut and tree nut allergies

KEY INTERVENTIONS

1. Recline victims in a position of comfort and transport to a hospital
2. Administer oxygen at high flow rates
3. Give epinephrine IM to all patients with clinical signs of shock, airway swelling, or definite breathing difficulties Dose: 300-500 ugs repeated after 5-10 minutes
4. Administer antihistamine IV
5. Provide inhaled salbutamol (not approved in the US) or epinephrine if airway obstruction is a major feature.
6. Give crystalloid if hypotension is severe and does not respond rapidly to Epinephrine
7. Raise the patient's legs as a useful interim measure
8. Inject high dose IV corticosteroids after severe attacks to help avert late sequelae, especially for asthmatics already on steroids
9. Keep under observation for at least 24 hours.

KEY INTERVENTIONS DURING ARREST

- Death is due to profound intravascular collapse
- Rapid volume expansion is an absolute requirements (as large as 4-6L over 15-30 minutes)
- High Dose Epinephrine IV
- Antihistamine IV
- Steroid Therapy
- Asystole/PEA
- Prolonged CPR - common error, failure to recognize cardiac arrest associated with anaphylaxis as imminently recoverable.

V. LIGHTNING STRIKE AND ELECTRICAL SHOCK

Lightning injuries have a 30% mortality rate. Up to 70% of survivors sustain significant morbidity.

- The primary cause of death in lightning strike is cardiac arrest which may be due to primary F or more frequently, due to ventricular asystole
- Lightning acts as an instantaneous, massive direct current countershock, depolarizing the entire myocardium at once and producing asystole.
- In many cases cardiac automaticity may restore organized cardiac activity and sinus rhythm may return spontaneously.
- Respiratory arrest due to thoracic muscle spasm and suppression of the respiratory center may continue after return of circulation
- Unless ventilatory assistance is provided, hypoxic cardiac arrest may occur.

REVERSE TRIAGE

Patients who are most likely to die of lightning injury if no treatment is forthcoming are those who suffer immediate cardiac arrest, usually with asystole and thoracic muscle spasm.

- When multiple victims are struck simultaneously, usual triage priorities should be reversed. Rescuers should give the highest priority to patients in respiratory or cardiac arrest.
- Patients who do not suffer cardiac arrest have an excellent chance of recovery because subsequent arrest is uncommon.

VI. CARDIAC ARREST ASSOCIATED WITH TRAUMA

Most cardiac arrests with trauma are either PEA or asystole. Remember that VF may initiate or follow a traumatic incident

KEY INTERVENTIONS

Making the correct diagnosis and fixing the underlying problem may be the only chance of resuscitation.

BLS MODIFICATIONS DURING CPR

- Head tilt should not be undertaken in any patient with cervical injury suspected or clinically or due to mechanism of injury
- Presence of blood, vomitus, and other secretions in the mouth must be ascertained before basic ventilatory procedures are undertaken
- Compressions and ventilations may require greater care and attention to detail in the presence of fractures to the chest wall or sternum
- External bleeding must be stopped as a priority to conserve blood volume.

ALS MODIFICATIONS DURING ARREST

AIRWAY

1. Tracheal intubation must be done with inline cervical immobilization.

2. Nasotracheal intubation must be undertaken with caution or may be contraindicated if a fracture of the anterior base of the skull is present or likely
3. Cricothyrotomy may be required in the event of massive facial damage to open, maintain, and secure the airway

BREATHING

1. Possible presence of preexisting pneumothorax, constant attention must be paid to auscultation of the chest throughout resuscitation
2. Sucking chest wounds must be appropriately sealed
3. Once intubated, simultaneous ventilations and compressions may result in development of pneumothorax. Synchronized ventilations and compressions in a ration of 1:5 may be required with damaged thoracic cage.
4. Consider NG tube to decompress the stomach of the trauma patient.

CIRCULATION

1. Bleeding must be controlled
2. Exclude pericardial tamponade as a priority especially in PEA
3. Adequate and aggressive volume replacement

EMERGENCY MEDICATIONS

1. Before medications in the exposed trauma patient, consider hypothermia
2. Because of the probability of pulmonary bleeding, administration of ET drugs is unwise with chest injury
3. Maintenance meds being taken by the patient must be ascertained if possible because these can affect the outcome of the traumatic response.

Prognosis for traumatic arrest is poor, especially those from blunt injuries. If return of spontaneous circulation is not obtained on the scene, continued resuscitation and transport to a trauma center is probably futile.

DEADLY DOZEN TRAUMATIC INJURIES

LETHAL 6 - you have <10 to 15 minutes to identify and treat

1. Airway obstruction
2. Tension pneumothorax
3. Penetrating cardiac injury
4. Open pneumothorax
5. Massive hemothorax
6. Flail chest

HIDDEN 6 - not immediately life-threatening: maximum time allowed < 60 minutes

1. Thoracic aortic disruption
2. Tracheobronchial injury
3. Myocardial contusion
4. Diaphragmatic tear
5. Esophageal injury
6. Pulmonary contusion

VII. CARDIAC ARREST ASSOCIATED WITH PREGNANCY

CAUSES OF ARREST

- A. Most commonly a maternal arrest is related to the changes and events faced at the time of delivery.
- Amniotic fluid embolism
 - Eclampsia
 - Drug toxicity (magnesium sulfate, epidural anesthetics)
- B. Other causes are related to the physiologic changes associated with pregnancy itself.
- Congestive cardiomyopathy
 - Aortic dissection
 - Pulmonary Embolism
 - Hemorrhage from pregnancy related pathology
- C. Causes unrelated to the pregnancy.

KEY INTERVENTIONS TO PREVENT ARREST

1. Place any distressed or compromised pregnant patient in the left lateral position.
2. Manually displace the uterus
3. Give 100% oxygen
4. Give fluid bolus
5. Immediately reevaluate any drugs being administered

BLS MODIFICATIONS DURING ARREST

1. Relieve aortocaval compression by manually displacing the gravid uterus
2. Use a wedge to displace the uterus to the left side of the abdomen

ALS MODIFICATIONS DURING ARREST

1. Consider rich variety of possible causes, i.e., magnesium sulfate toxicity, drug overdose, medication toxicity and iatrogenic events

EMPTY THE UTERUS

With Mom in arrest, the blood supply to the fetus rapidly becomes hypoxic and acidotic. The blood flow returning the mother's heart becomes blocked by the uterus. You are going to lose both the other and the infant unless you can get blood returning to the mother.

WHEN STANDARD BLS AND ALS FAIL

If chance of fetal viability, consider immediate perimortem cesarean section (ideally within 5 minutes from the arrest to the delivery of the infant)

LEARNING STATION FOUR TOXICOLOGIC EMERGENCIES

BLS MODIFICATIONS DURING ARREST

1. Rescuer Safety
2. Move the patient to a toxin-free area before starting CPR
 - Be aware that you may be in a hazmat situations
 - Remove hazardous material from the victim's skin or clothing before starting CPR
 - Remove the patient from the source of inhalation or skin contamination
 - Secure the scene if others could be exposed and affected

ALS MODIFICATIONS DURING ARREST

Same as BLS Modifications

ECG FINDINGS AND TREATMENT FOR COMMON TOXINS AND DRUG OVERDOSES

Clinical Problem	ECG Findings	Treatment Approaches
Cyclic Antidepressants and Major Tranquilizers	<ul style="list-style-type: none"> • ST and T wave changes • QRS wide • QT Long • R Axis deviation • BBB • AV Conduction blocks • Broad slurred QRS • Ventricular Arrhythmias • PEA 	<ul style="list-style-type: none"> • Hypervent to pH=7.5 • Benzodiazepine or Phenobarbital for seizures • Sodium Bicarbonate 1-5 mEq/kg over 1 to 2 minutes • Bicarb infusions 50-100 mEq at 150 to 200cc/hr • Magnesium 1-2 g IV bolus if unstable, over 1 to 5 minutes if stable • Pressors if needed (norepinephrine, epinephrine, high dose dopamine) • Charcoal hemoperfusion • Cardipulmonary bypass
Cocain	<ul style="list-style-type: none"> • Sinus Tachycardia • SVT • VF/VT • Cocaine induced AMI 	<ul style="list-style-type: none"> • Delay thrombolytics until enzyme levels confirm MI • NTG or nitroprusside (0.1-5 ug/kg per minute IV) to lower BP and HR • Benzodiazepines for increased BP, HR • Phentolamine 1-10 ug titrated as a dilute solution over 2-3 minutes to lower BP
Clinical Problem	ECG Findings	Treatment Approaches

Cocaine (cont)		<ul style="list-style-type: none"> • Labetalol (5-20 mg IV) consider for severe HTN CARDIAC ARREST <ul style="list-style-type: none"> • Use Lido cautiously • Space EPI to q 5-10 mins • Consider propranolol or labetalol
Beta Blockers	<ul style="list-style-type: none"> • Bradycardia • AV Block • Wide QRS • Peaked T waves • ST Changes • Cardiac deterioration leading to PEA 	<ul style="list-style-type: none"> • Saline bolus • Atropine • Epinephrine • Glucagon 1-5mg IV over 1 minute • Dopamine • Calcium chloride (5-20 ml IV) • Pacing • Pressors
Calcium Channel Blockers	<ul style="list-style-type: none"> • Sinus bradycardia • QRS widening • QT Long • Heart Blocks • PEA 	<ul style="list-style-type: none"> • Saline bolus • Calcium chloride • Glucagon • Epinephrine • Dopamine • Repeat Calcium Chloride • Dobutamine, norepinephrine and/or isoproterenol • Pacing • Cardiopulmonary bypass
Digitalis Ingestion	<ul style="list-style-type: none"> • Many arrhythmias are possible 	<ul style="list-style-type: none"> • Volume replacement • Potassium replacement • Magnesium replacement • Digoxin specific antibodies (Digibind)
Narcotic Overdose	<ul style="list-style-type: none"> • No specific ECG effects • 	<ul style="list-style-type: none"> • Standard ACLS plus • Naloxone 2mg IV repeat q 2-5 minutes • Naloxone infusion 0.8-1.0 mg/hr and titrated to effect (Mix 8-10 mg in 1000 cc D5W)
Benzodiazepine Ingestion	<ul style="list-style-type: none"> • No specific ECG effects • Bradycardia then asystole following respiratory depression 	<ul style="list-style-type: none"> • Romazicon 0.2mg IV over 15 seconds • Repeat at 1 minute intervals