ADVANCED CARDIAC LIFE SUPPORT PROVIDER
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Overview

If you have not certified in 2015 BCLS guidelines, return to the site to purchase and test now.

In a respiratory or cardiac arrest, the goal of the healthcare team is to restore ventilation, oxygenation and circulation. By following a systematic approach, the healthcare team preserves the integrity of the neurologic system to its highest degree. As in responding to any emergency, always begin by being sure the scene is safe.

After determining the scene is safe, as an ACLS provider you will begin by determining the patient’s level of consciousness, and proceeding through the steps outlined in the Systematic ACLS Algorithm.
**BCLS ASSESSMENT**

*If you have not certified in 2015 BCLS guidelines, return to the site to purchase and test now.*

After determining the scene is safe, the healthcare provider will begin the BCLS assessment. BCLS affords the healthcare provider an opportunity to restore or support ventilation, oxygenation and circulation without the use of advanced airways or drug administration.

<table>
<thead>
<tr>
<th>ASSESSMENT</th>
<th>ACTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Check Victim for Responsiveness</td>
<td>“Are you okay?”</td>
</tr>
</tbody>
</table>
| Activate Emergency Response/Get AED | Shout for help  
Call 911  
Get an AED or send someone for one |
| Check for Breathing and Pulse | Check for breathing for no more than 10 seconds  
Check for pulse for no more than 10 seconds  
No pulse: begin CPR, compressions first  
Pulse: begin rescue breaths |
| Defibrillation              | No pulse: check for shockable rhythm  
Shock as indicated  
Follow prompts to provide CPR |
**Primary Assessment- ABCDE**

If the patient is unconscious, the healthcare provider will begin the Primary Assessment. This assessment will continue until the patient reaches the next level of care, or at the same time as ACLS is conducted.

<table>
<thead>
<tr>
<th><strong>ASSESSMENT</strong></th>
<th><strong>ACTION</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Airway – Patent?</td>
<td>• Maintain patency with head tilt-chin lift, oropharyngeal or nasopharyngeal airway</td>
</tr>
<tr>
<td>Advanced airway indicated?</td>
<td>• Utilize advanced airway management if necessary, ensuring correct CPR/ventilation integration</td>
</tr>
<tr>
<td>Airway device properly positioned?</td>
<td>• Continue to monitor waveform capnography</td>
</tr>
<tr>
<td>Tube position rechecked regularly?</td>
<td></td>
</tr>
<tr>
<td>Breathing-</td>
<td>• Supplementary oxygen when indicated</td>
</tr>
<tr>
<td></td>
<td>• Monitor ventilation/oxygenation adequacy</td>
</tr>
<tr>
<td></td>
<td>• Avoid excessive ventilation</td>
</tr>
<tr>
<td>Circulation- Effective chest compressions?</td>
<td>• Monitor CPR effectiveness, quantitative waveform capnography, intra-arterial pressure</td>
</tr>
<tr>
<td>Cardiac rhythm?</td>
<td>• Defibrillation/cardioversion</td>
</tr>
<tr>
<td>IV/IO access established?</td>
<td>• IV/IO fluids</td>
</tr>
<tr>
<td>Defibrillation/cardioversion indicated?</td>
<td>• Monitor temperature and glucose</td>
</tr>
<tr>
<td>Pulse stable?</td>
<td>• Appropriate drugs</td>
</tr>
<tr>
<td>Volume needed?</td>
<td>• Monitor perfusion</td>
</tr>
<tr>
<td>Disability-</td>
<td>• Monitor neurologic function</td>
</tr>
<tr>
<td></td>
<td>• Assess responsiveness</td>
</tr>
<tr>
<td></td>
<td>• Assess level of consciousness/pupil dilation</td>
</tr>
<tr>
<td>Exposure-</td>
<td>• Remove clothing for exam</td>
</tr>
<tr>
<td></td>
<td>• Look for signs of trauma</td>
</tr>
<tr>
<td></td>
<td>• Look for medical alert bracelets</td>
</tr>
</tbody>
</table>
Secondary Assessment

In the secondary assessment, the healthcare provider gathers patient medical history information, as well as “H & T’s”. During this assessment, the healthcare provider will develop a differential diagnosis and begin treatment of underlying causes. The H & T’s are potentially reversible conditions leading to cardiac arrest.

<table>
<thead>
<tr>
<th>H</th>
<th>T</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolemia</td>
<td>Tension pneumothorax</td>
</tr>
<tr>
<td>Hypoxia</td>
<td>Tamponade</td>
</tr>
<tr>
<td>Hydrogen ion</td>
<td>Toxins</td>
</tr>
<tr>
<td>Hypo-/hyperkalemia</td>
<td>Thrombosis- pulmonary</td>
</tr>
<tr>
<td>Hypothermia</td>
<td>Thrombosis – coronary</td>
</tr>
</tbody>
</table>

Diagnosis and Treatment

Act quickly to identify and manage potentially reversible underlying problems that may be causing cardiac arrest.

- Most common PEA causes are hypovolemia and hypoxia, possibly reversible
- ECG may reveal underlying causes of cardiac arrest
- Recall the H and T’s for possible causes of PEA

Hypovolemia

Hypovolemia is a state of decreased blood volume, specifically a decrease in blood plasma volume, characterized by sodium depletion. It is a common cause of PEA and presents as a rapid, narrow-complex tachycardia with an increase in diastolic and decrease in systolic blood pressures. Hypovolemia may be an underlying cause of hypotension, resulting in PEA. Hypovolemia may be caused by trauma, internal hemorrhage and dehydration. PEA with narrow-complex tachycardia can be treated with volume infusion.
**Cardio-Pulmonary Conditions**

In PEA, acute coronary conditions impacting large amounts of heart muscle may be present. Patients with occlusion of the left main or proximal left anterior descending coronary artery may have cardiogenic shocking, progressing to both cardiac arrest and PEA.

Patients without pulmonary embolism have no demonstrated benefit to receiving fibrinolytic treatment during CPR however, when pulmonary embolism is suspected, fibrinolytic treatment should be administered. Pulmonary embolism obstructs the flow to the vasculature, causing acute right heart failure.

Volume infusion may be beneficial during the periarrest period of pericardial tamponade, a potentially reversible condition. Healthcare providers can effectively treat tension pneumothorax once identified.

**Toxins and Overdoses**

Hypotension from peripheral vascular dilatation and myocardial dysfunction may result from overdose or exposure to drugs and toxins. Rapidly progressing toxin effects should be treated aggressively, as they are often reversible.

Treatment options may include prolonged CPR, adjunct agents, renal dialysis, intra-aortic balloon pumping, IV lipid emulsion, drug antidotes and electrolyte disturbance treatment.
Overview

ACLS scenarios include the skills learned in Chapter One. Healthcare providers should be sure they have certified in 2015 BCLS and have thoroughly reviewed all information in Chapter One prior to proceeding to the following six scenarios including respiratory arrest, acute coronary syndrome, stroke, cardiac arrest, bradycardia and tachycardia.

SCENARIO: RESPIRATORY ARREST

Healthcare providers must be able to assess, intervene and manage scenarios for unconscious, unresponsive adults, including distinguishing between respiratory distress, respiratory failure and respiratory arrest.

- Consider using other drugs in rapid sequence intubation
- Adult respiratory rate averages 12 - 16/min
- Maintain tidal volume of 8 - 10 mL/kg

Respiratory Distress

- Abnormal respiratory rate or effort
- Nasal flaring, retractions
- Hypoventilation
- Changes in respiratory sounds
- Changes in skin color
- Changes in mental status
- Tachycardia
**Respiratory Failure**

- Bradypnea, apnea
- Tachypnea
- Tachycardia
- Bradycardia
- Cyanosis
- Coma
- Increased, decreased, absence of respiratory effort

**Respiratory Arrest**

- Absence of breathing
- Typical of head injury, drowning, airway obstruction

Treatment with tidal volume of 500 – 600 mL is typically sufficient. Healthcare providers should note visible chest rise. In airway obstruction scenarios, healthcare providers should ventilate with a bag-mask, with any pressure-relief valve bypassed in order to use high pressures in order to cause the chest to visibly rise.

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*Healthcare providers should take caution against excessive ventilation which may cause gastric inflation, regurgitation and aspiration. Excessive ventilation may also cause decreased venous return to the heart and increased intrathoracic pressure.*

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- Monitor oxygen and titrate to maintain 94% or greater saturation
- Maintain open airway with head tilt chin lift or jaw thrust
- Provide basic ventilation through most appropriate technique
Basic Ventilation Techniques

- Head tilt-chin lift
- Jaw thrust
- Mouth to mouth
- Mouth to nose
- Pocket mask
- Bag-mask

Airway Adjuncts

Nasopharyngeal:

For use in patients who are conscious, semiconscious or unconscious with intact gag and cough reflex

- Select the correct size – should not be so large as to cause nostril blanching; measure length by distance of tip of nose to earlobe
- Lubricate airway – use anesthetic jelly or other water soluble lubricant
- Insert- through nostril perpendicular to plane of face
- Resistance – rotate or attempt other nostril

Oropharyngeal:

For use in patients who are unconscious, without intact gag or cough reflex

- Clear – mouth and pharynx of blood, vomit or other secretions using rigid pharyngeal suction tip
- Select the correct size – Measure flange of OPA at corner of mouth, tip at the angle of mandible
- Insert- curve upward toward hard palate
- Rotate – as OPA passes through oral cavity, rotate 180 degrees or insert at 90 degrees into mouth and turn down toward posterior pharynx as it advances

OPAs may obstruct the larynx or cause trauma if too large. OPAs may obstruct airway with tongue if too small. Check spontaneous respirations immediately after insertion of NPA or OPA. If respirations are not adequate or absent, begin positive-pressure ventilations immediately.
Suctioning

Suction immediately if there are large amounts of blood, vomit or secretions.

**Portable suction devices with a force of -80 to -120 mmHg**

**Wall-mounted device with a airflow of greater than 40 L/min and vacuum of greater than -300 mm Hg**

**Adjust suction for pediatric or intubated patients**

**Oropharyngeal Suctioning:**
- Measure catheter prior to suctioning, avoiding insertion beyond the distance from tip of nose to earlobe
- Suction by occluding catheter’s side opening, simultaneously withdrawing and rotating
- For use of rigid suction, tip into the oral cavity, advancing beyond the tongue to the oropharynx

**Endotracheal Tube Suctioning:**
- Use a sterile technique
- Insert catheter into ET tube, being sure to not occlude side opening
- Suction by occluding side opening only, simultaneously withdrawing and rotating
- Do not attempt for more than ten seconds
**Advanced Airway Ventilation**

- Laryngeal mask airway
- Laryngeal tube
- Esophageal-tracheal tube
- ET tube

*Advanced airway ventilation rates for cardiac arrest are once every six seconds.*

*Advanced airway ventilation rates for respiratory arrest are once every 5 - 6 seconds.*

**Endotracheal Tube**

<table>
<thead>
<tr>
<th>Prepare by assembling equipment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perform ET intubation</td>
</tr>
<tr>
<td>Inflated cuffs on tube</td>
</tr>
<tr>
<td>Attach ventilation bag</td>
</tr>
<tr>
<td>Confirm correct placement by physical exam and continuous waveform capnography</td>
</tr>
<tr>
<td>Secure tube placement</td>
</tr>
<tr>
<td>Monitor placement</td>
</tr>
</tbody>
</table>
SCENARIO: ACUTE CORONARY SYNDROMES

Healthcare providers must be able to assess, stabilize and manage patients with ACS. In all ACS cases, the healthcare provider initializes the 12-lead ECG and categorizes patients into one of three possible ECG categories. The three categories present different care and management strategies.

![ECG Categories Diagram]

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**ACS Drugs**

Initial treatment is focused on relieving ischemic discomfort, dissolving clots and inhibiting platelets and thrombin.

- Aspirin
- Nitroglycerin
- Fibrinolytic therapy
- Heparin
- Oxygen
- Opiates

Adjunct to initial therapy include:

- Beta-blockers
- Glycoprotein IIb/IIa inhibitors
- ACE inhibitors
- ADP antagonists
- Statin therapy

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*A number of clinical syndromes may be associated with coronary atherosclerosis including non-ST elevation, (NSTE-ACS) and STEMI.*
ACUTE CORONARY SYNDROMES

Victim displaying symptoms of Ischemia or Infarction.

Monitor and support ABCs and be ready to give CPR or use AED. Give aspirin and nitroglycerin, oxygen, or morphine if necessary. Retrieve 12-lead ECG and if ST elevation occurs, alert the hospital with a transmission of interpretation, while being sure to record time of onset and original medical contact. Hospital should mobilize medical resource to respond. If prehospital fibrinolysis is considered, use the fibrinolytic checklist.

Concurrent ED assessment (less than 10 minutes):
Check the vital signs and examine oxygen saturation. Obtain IV access. Give victim a short targeted history physical exam. Finish fibrinolytic checklist and observe contraindications. Retrieve original cardiac marker levels and the initial studies of electrolytes and coagulation. Obtain the portable chest x-ray.
Immediate ED general treatment:
If oxygen saturation is below 90%, begin oxygen at 4L/min, titrate. Aspirin 160-325mg. Nitroglycerin sublingual or spray. If discomfort not taken away by nitroglycerin, give Morphine IV.

ECG Interpretation.

ST elevation or new LBBB; suspicious for injury. ST-elevation MI also knows as STEMI.

Begin adjunctive therapies and be sure to not delay reperfusion.

If time from symptom onset is less than 12 hours: Therapy defined by criteria. Door to balloon inflation: goal of 90 minutes. Door to needle inflation: goal of 30 minutes

ST depression or T-wave inversion; suspicious for ischemia. NSTE ACS.

Time from symptom onset is greater than 12 hours: Early Invasive strategy if:

Normal changes in ST segment or T-wave: Low-Intermediate risk ACS.

Consider sending the victim to appropriate bed or unit for further monitoring and care.
MANAGING ACS

Initial assessment and action may begin in an out of hospital environment, conducted by EMS responders, including providing oxygen, aspirin, morphine and nitroglycerin. EMS should also obtain an initial 12-lead ECG. Depending on the results, EMS responders should complete the fibrinolytic therapy checklist and notify the hospital of an AMI-STEMI patient arriving.

Upon hospital arrival, healthcare providers should review the results of the out of hospital 12-lead ECG, or immediately perform one if it has not yet been conducted. 12-lead ECG results should be analyzed within the first ten minutes of the patient’s arrival, followed immediately by categorizing based on the results of the ST segment or left bundle branch block presence.

Chest Discomfort Indicating Possible Ischemia

- Chest fullness, squeezing, pressure or pain in the center lasting several minutes
- Discomfort spreading to neck, jaw, shoulders and one/both arms
- Spreading to back and shoulder blades
- Light-headedness, fainting, dizziness, nausea, sweating, vomiting
- Shortness of breath
**Oxygen and Drug Administration**

**Aspirin**

- 160-325 mg non-enteric-coated aspirin, immediate inhibition of thromboxane A(2) production.
- Inhibits platelet cyclooxygenase (COX-1)
- Reduces coronary reocclusion, recurrent events after fibrinolytic therapy
- Better absorbed when chewed vs. swallowed
- 300mg rectal aspirin suppositories for patients with nausea, vomiting, peptic ulcer or upper GI disorders

**Oxygen**

- High inspired-oxygen tension to maximize arterial oxygen saturation
- Helps support oxygen delivery when cardiac output limited
- Administer to patients who are dyspneic, hypoxemic, display signs of heart failure or have arterial saturation less than 90%
- Titrate oxygen therapy to noninvasively monitored oxyhemoglobin saturation 90% or greater

**Nitroglycerin**

- Reduces ischemic chest discomfort
- Administer 1 sublingual tablet every 3 – 5 minutes; up to 3 doses
- Administer only for stable patients (SBP greater than 90 mm Hg)
- Not for use in patients with inferior wall MI, RV infarction, hypotension, bradycardia, tachycardia, recent phosphodiesterase inhibitor use

**Opiates**

- Administer if no results from nitroglycerin
- Provides central nervous system analgesia
- Reduces adverse effects of neurohumoral activation, catecholamine release, myocardial oxygen demand
- Decreases systemic vascular resistance

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*Nonsteroidal anti-inflammatory drugs are contraindicated with the exception of aspirin. Nonselective and COX-2 selective drugs should not be administered during STEMI due to increased risk of reinfarction, mortality, hypertension, heart failure and myocardial rupture.*
In-Hospital Treatment and Assessment

The First 10 Minutes

- Vital signs, evaluate oxygen saturation
- IV access
- Brief history, physical exam
- Fibrinolytic checklist, contraindications
- Blood sample for cardiac marker levels, electrolytes and coagulation
- Portable chest x-ray within 30 minutes

General Care

- Oxygen if hypoxemic or signs of heart failure
- Aspirin
- Opiate if no response to nitrates
- Nitroglycerin

ST-Segment Deviation Based Classification

<table>
<thead>
<tr>
<th>STEMI</th>
<th>ST elevation</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSTE-ACS</td>
<td>ST depression or dynamic T-wave inversion</td>
</tr>
<tr>
<td>Low-/Intermediate –risk ACS</td>
<td>Normal or nondiagnostic ECG</td>
</tr>
</tbody>
</table>

STEMI

- Complete occlusion of epicardial coronary artery typical
- Early reperfusion therapy achieved with primary PCA or fibrinolytics
- Reperfusion reduces mortality, saves heart muscle
- Fibrinolytic therapy within first hour show 47% reduction in mortality
Early Reperfusion

- STEMI patient’s first physician should confirm 12-lead ECG
- Determine risk/benefit of reperfusion therapy
- Direct administration of fibrinolytic therapy or PCI team activation
- Follow recommended time guidelines

<table>
<thead>
<tr>
<th>PCI</th>
<th>Arrival to balloon inflation within 90 minutes; non-PCI facilities, first medical contact to device less than 120 if PCI considered</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fibrinolysis</td>
<td>Arrival to needle (beginning fibrinolytic agent infusion) no more than 30 minutes</td>
</tr>
<tr>
<td>Fibrinolytic Ineligible</td>
<td>Consider transfer to PCI facility; door to departure less than 30 minutes later</td>
</tr>
</tbody>
</table>

PCI

Primary PCI is the preferred reperfusion strategy. Rescue PCI is early after fibrinolytics for patients with persistent occlusion of the infarct artery. PCI is has been proven to be superior to fibrinolysis in patients with combined end points of death, reinfarction and stroke presenting 3 to 12 hours after onset.

Best option for STEMI management when performed arrival to balloon time less than 90 minutes

Primary PCI also acceptable to patients arriving at non-PCI hospitals if initiated within 120 minutes

PCI preferred for patients with contraindications to fibrinolytics
**Fibrinolytic Therapy**

“Clot busters” are administered to patients presenting with J-point ST-segment elevation greater than 2 mm in leads V(2) and V(3) and 1 mm or more in every other lead. It is also administered where there is new LBBB without contraindications. Normal flow is achieved in approximately 50% of patients administered fibrin-specific agents.

- No contraindications, fibrinolytic therapy is an option for reperfusion in STEMI patients with onset of symptoms within 12 hours
- ST-segment depression in early precordial leads equivalent to ST-segment elevation in others
- Not recommended for patients presenting more than 24 hours after onset of symptoms

**Adjunct Treatments**

In addition to sublingual or spray nitroglycerin, aspirin, oxygen, morphine and fibrinolytic therapy:

- Glycoprotein IIb/IIa inhibitors
- IV nitroglycerin
- Beta blockers
- Unfractionated/low molecular weight heparin
- Bivalirudin
- P2Y(12) inhibitors

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*Intracerebral bleeding and major hemorrhage in STEMI patients may be caused by inappropriate dosing and monitoring of heparin therapy. Careful dosing, use and duration is paramount in effective heparin therapy.*

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**IV Nitroglycerin**

IV nitroglycerin is widely used in ischemic syndromes, although has not been demonstrated to reduce mortality in STEMI. It can be titrated in patients with unstable hemodynamics. Patients presenting with recurrent chest discomfort unresponsive to sublingual or spray nitroglycerin, pulmonary edema and hypertension may benefit from IV nitroglycerin.

<table>
<thead>
<tr>
<th>Ischemic chest discomfort relief</th>
<th>Pulmonary edema/hypertension relief</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Titrate</td>
<td>• Titrate</td>
</tr>
<tr>
<td>• Maintain SBP greater than 90 mm Hg</td>
<td>• Limit SBP drop to 10% of baseline in normotensive patients</td>
</tr>
<tr>
<td>• Limit SBP drop to 30 mm Hg</td>
<td>• Limit SBP drop to 30 mm Hg below baseline in hypertensive patients</td>
</tr>
</tbody>
</table>
SCENARIO: ACUTE STROKE CASE

While out of hospital stroke care focuses on rapid assessment and identification of stroke patients, in-hospital care for stroke patient care focuses on rapid determination if the patient is appropriate for fibrinolytic therapy, therapy administration and consideration for additional treatment options.

Drugs for stroke treatment include:
- Nitroprusside
- Glucose D(50)
- Fibrinolytic agent (rtPA)
- Aspirin
- Enalaprilat
- Nicardipine

Early recognition of ischemic stroke patients is critical as IV fibrinolytic treatment is recommended to begin within 3 hours of symptom onset, and endovascular therapy within 6 hours. Following the Chain of Survival minimizes brain injury and gives the stroke patient the best opportunity for recovery.

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The Cardiac Chain of Survival

*Early Recognition – Early Dispatch — Early Transport — Early Treatment*
ACUTE STROKE

Observe symptoms of possible stroke. Activate emergency response system.

Support ABCs and give oxygen if necessary. Begin stroke assessment. Record time of symptom onset. Triage to stroke center. Notify the hospital. Check glucose if able to.

Assess patient’s ABCs and vital signs. If hypoexemic, give oxygen. Perform lab assessments and gain IV access. Observe glucose and respond if necessary. Begin neurologic screening. Activate stroke team. Order CT scan or brain MRI. Retrieve 12-lead ECG.

Study patient’s history. Establish symptom timeline. Begin neurologic exam.

If CT scan shows no hemorrhage:
- Look for fibrinolytic exclusions
- Redo neurologic exam

If patient remains a candidate for fibrinolytic therapy:
- Give rtPA
- Provide no anticoagulants or antiplatelet treatment for 1 day
- Review risks and benefits with family and patient prior to treatment

Start the post-rtPA stroke pathway
- Monitor BP protocol and watch for neurological deterioration
- Emergent admission to ICU or stroke unit

If they are not a candidate for fibrinolytic therapy:
- Administer aspirin

If CT scan shows hemorrhage:
- Consult with a neurologist or neurosurgeon

Start stroke or hemorrhage pathway
- Admit patient to stroke or intensive care unit
Stroke Diagnosis and Treatment Steps:

1. **Detection**: early recognition of symptoms
2. **Disptach**: early activation of 911
3. **Delivery**: EMS identification and transport
4. **Door**: triage to stroke care
5. **Data**: evaluation and management
6. **Decision**: therapy selection
7. **Drug/Device**: fibrinolytic or endovascular therapy
8. **Disposition**: rapid stroke unit admission

**Treatment Timeline**

For acute ischemic stroke patients, the time dependent benefit of fibrinolytic therapy is far shorter than for those with ST-segment elevation MI. The critical time period for IV fibrinolytic therapy begins at the point of onset of symptoms.

<table>
<thead>
<tr>
<th>Event</th>
<th>Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immediate general onset</td>
<td>10 minutes</td>
</tr>
<tr>
<td>Immediate neurologic assessment</td>
<td>25 minutes</td>
</tr>
<tr>
<td>CT head scan</td>
<td>25 minutes</td>
</tr>
<tr>
<td>Interpret CT scan</td>
<td>45 minutes</td>
</tr>
<tr>
<td>Fibrinolytic therapy, timed from arrival</td>
<td>60 minutes</td>
</tr>
<tr>
<td>Fibrinolytic therapy, timed from onset</td>
<td>3 hours</td>
</tr>
<tr>
<td>Endovascular therapy, timed from onset</td>
<td>6 hours</td>
</tr>
<tr>
<td>Admission to unit</td>
<td>3 hours</td>
</tr>
</tbody>
</table>
Signs & Symptoms

- Sudden weakness face, arm, leg, particularly one-sided
- Difficulty speaking
- Difficulty comprehending
- Confusion
- Difficulty walking
- Vision disturbance
- Dizziness, loss of balance
- Severe headache

Cincinnati Prehospital Stroke Scale (CPSS)

| Facial Droop – Have the patient smile | Normal – both sides move equally  
| Abnormal- one side does not move as well as the other |
| Arm Drift – Have patient close eyes, raise both arms straight out, palms up | Normal – both arms move the same  
| Abnormal – one arm does not move or drifts downward |
| Abnormal Speech- Have patient repeat a phrase | Normal – patient repeats using correct words without slurring  
| Abnormal – Patient uses incorrect words, slurs or can’t speak |

EMS ACTIONS:

- Support ABCs – provide supplementary oxygen
- Perform CPSS
- Establish Timeline – determine onset of symptoms
- Triage during transport
- Alert receiving hospital
- Check glucose
## IN-HOSPITAL ASSESSMENT/STABILIZATION

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Assess ABCS</td>
<td>Airway, breathing, circulation, baseline vitals</td>
</tr>
<tr>
<td>Provide Oxygen</td>
<td>Supplementary oxygen to hypoxemic patients</td>
</tr>
<tr>
<td>IV Access/Blood Samples</td>
<td>Baseline blood count, coagulation studies, glucose</td>
</tr>
<tr>
<td>Glucose</td>
<td>Treat hypoglycemia</td>
</tr>
<tr>
<td>Neurologic Assessment</td>
<td>Conduct NIH Stroke Scale Assessment</td>
</tr>
<tr>
<td>Activate Stroke Team</td>
<td>Notify team, arrange consultations</td>
</tr>
<tr>
<td>CT Brain Scan</td>
<td>Prompt reading by physician</td>
</tr>
<tr>
<td>12-Lead ECG</td>
<td>Identify recent/ongoing AMI, arrhythmias as cause of embolic stroke; identify coexisting myocardial ischemia</td>
</tr>
</tbody>
</table>

### STROKE TEAM NEUROLOGIC ASSESSMENT

Review of the patient’s history, physical exam, establishment of onset of symptoms and neurologic examination is performed by the stroke team, neurovascular consultant or emergency department physician. This should be completed within 25 minutes of the patient’s arrival.

### CT SCAN FOR HEMORRHAGE

The CT scan is a critical step in the assessment of acute stroke patients. Non-contrast CT scans differentiate ischemic from hemorrhagic strokes. This should be completed within 25 minutes of the patient’s arrival and read within 45 minutes of arrival. If CT scan is not available, the patient should immediately be transferred to an appropriate facility.

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*Do not give aspirin, heparin or rtPA until intracranial hemorrhage has been ruled out by CT scan.*
HEMORRHAGE VS NO HEMORRHAGE – THE NEXT STEP

FIBRINOLYTIC THERAPY

In cases where CT shows no hemorrhage, fibrinolytic therapy should be considered. Additional assessments should be performed to determine eligibility. The neurologic exam should be repeated. Inclusion and exclusion criteria should be reviewed. If the patient’s neurologic function is returning to normal, fibrinolytics may be unnecessary. Patients may still receive fibrinolytic therapy despite the presence of one or more contraindications if the benefit outweighs the risk.
INCLUSION CRITERIA

- Diagnosis of ischemic stroke causing neurologic deficit
- Symptom onset less than 3 hours before treatment
- Patient is 18+ years old

EXCLUSION CRITERIA

- Significant head trauma within prior 3 months
- Possible subarachnoid hemorrhage
- Arterial puncture at noncompressible site within previous 7 days
- Intracranial hemorrhage history
- Elevated blood pressure
- Active internal bleeding
- Acute bleeding diathesis
**Anticoagulants and antiplatelet treatment should not be administered for 24 hours after rtPA administration. A follow-up CT scan showing no intracranial hemorrhage is recommended after 24 hours.**

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**ENDOVASCULAR THERAPY**

Endovascular therapy is recommended for acute ischemic stroke patients who qualify based on inclusion criteria. This treatment option may increase the treatment window up to six hours following the onset of symptoms.

**MECHANICAL CLOT DISRUPTION/STENT RETRIEVERS**

Select acute ischemic stroke patients may benefit from mechanical clot disruption or retrieval with a stent. Patients must meet the following criteria:

- NIHSS score of 6 or greater
- Age 18 years +
- Receiving IV rtPA within 4.5 hours of onset of symptoms
- Prestroke mRS score of 0 to 1
- Causative occlusion of internal carotid artery or proximal MCA

**HYPERTENSION MANAGEMENT IN rtPA PATIENTS**

| Acute reperfusion therapy eligible except blood pressure greater than 185/110 mm Hg | • Labetalol 10-20 mg IV over 1-2 minutes, repeat 1 time
| | • Nicardipine IV 5 mg/h, titrate up to 2.5 mg/h every 5-15 min, max 15 mg/h
| | • Consider hydralazine, enalaprilat
| Management during and after rtPA | • Monitor blood pressure every 15 min for 2hrs from rtPA therapy start
| | • Systolic bp 180-230 MM Hg or diastolic bp 105-120 mm Hg labetol 10 mg IV with continuous IV infusion 2-8/mg/min OR nicardipine IV 5 mg/h, titrate up to 2.5 mg/h every 5-15 min, maximum 15 mg/h
**SCENARIO: CARDIAC ARREST – VT/PULSELESS VT**

For a pulseless patient not responding to BLS, the Adult Cardiac Arrest Algorithm is the most important guideline for assessment and management. The algorithm identifies two different courses of action for a cardiac arrest patient: a shockable rhythm (VF/pulseless VT) and nonshockable rhythm (asystole/PEA). Many patients demonstrate VF during cardiac arrest. VF and pulseless VT both require CPR and are treated with AED. These steps are outlined on the left side of the Adult Cardiac Arrest Algorithm. On the right side, actions for nonshockable rhythms are outlined for PEA and Asystole scenarios.
Adult Cardiac Arrest Algorithm

1. Start CPR and be sure to give oxygen and attach monitor and defibrillator.

2. If shockable rhythm detected: V fib/VT

3. Shock the patient

4. Give CPR for 2 minutes and obtain IV/IO access.

5. Is a shockable rhythm present?

6. If yes, provide a shock.

7. Give 2 minutes of CPR and give epinephrine every 3.5 minutes. Consider capnography.

8. Is a shockable rhythm present?

9. If shockable rhythm not detected: Asystole/PEA

10. Give CPR for 2 minutes, obtain IV/IO access, give epinephrine every 3.5 minutes, and consider capnography.

11. Is a shockable rhythm present?

12. If no give 2 minutes of CPR and treat reversible causes.

13. If yes, go to steps 5 or 7.
**VF/pVT IN CARDIAC ARREST**

Beginning with high quality CPR immediately is critical while preparing for use of a manual defibrillator or AED. Minimizing interruptions of chest compressions during CPR is most effective. Once a defibrillator has analyzed and advises the healthcare provider to deliver a shock, the correct energy dose should be utilized. For monophasic defibrillators, administer a single 360-J shock. For biphasic defibrillators, healthcare providers should follow the manufacturer’s recommendations, often displayed on the device. Early defibrillation significantly increases the patient’s survival rate, decreasing approximately 10% every minute defibrillation is delayed.

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*Two minutes of CPR should resume immediately after delivering a shock, beginning with chest compressions. Refer to BLS certification for CPR ratios and guidelines.*

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**Vasopressors**

If IV/IO access is available during CPR, epinephrine should be administered after the second shock, 1 mg IV/IO, repeating every 3 – 5 minutes.

Epinephrine hydrochloride is administered for beta-adrenergic effects, primarily vasoconstriction which increases cerebral and coronary blood flow.

**Shock and Antiarrhythmics**

Antiarrhythmic drugs should be considered for administration before and after shock.

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*Amiodarone should be the first drug considered as it improves the rate of ROSC and hospital admission in patients with refractory VF/pulseless VT.*

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# ANTIARRHYTHMIC DRUGS

<table>
<thead>
<tr>
<th>Drug</th>
<th>Actions and Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Amiodarone</strong></td>
<td>• 300 mg IV/IO bolus&lt;br&gt;• Consider following additional 150 mg IV/IO&lt;br&gt;• Blocks sodium channels&lt;br&gt;• Exerts noncompetitive antisypathetic action&lt;br&gt;• Lengthens cardiac action potential</td>
</tr>
<tr>
<td><strong>Lidocaine</strong></td>
<td>• Consider if amiodarone not available&lt;br&gt;• 1 – 1.5 mg/kg IV/IO first dose&lt;br&gt;• Follow by .5 - .75 mg/kg IV/IO at 5-10 minute intervals&lt;br&gt;• Maximum dose 3 mg/kg&lt;br&gt;• Increases electrical stimulation threshold of ventricle&lt;br&gt;• Blocks permeability of neuronal membrane to sodium ions</td>
</tr>
<tr>
<td><strong>Magnesium Sulfate</strong></td>
<td>• Consider for long QT interval associated torsades de pointes&lt;br&gt;• Loading dose 1 – 2 g IV/IO diluted in 10 mL&lt;br&gt;• Administered as IV/IO bolus over 5-20 minutes&lt;br&gt;• Suppresses atrial L- and T-type calcium channels&lt;br&gt;• Classified as sodium/potassium pump agonist</td>
</tr>
</tbody>
</table>
## Physiologic Monitoring

| End-tidal CO(2) | • Blood delivery during CPR main determinant of PETCO(2)  
|                | • Less than 10 mm Hg for intubated patients suggests unlikely ROSC  
|                | • Abrupt PETCO(2) increase to normal value of 35-40 mm Hg reasonable ROSC indicator  
|                | • PETCO(2) less than 10 mm Hg, improve chest compressions and vasopressor therapy  

| Coronary Perfusion Pressure/Arterial Relaxation Pressure | • CPP increase correlates with myocardial blood flow and ROSC  
|                                                         | • Surrogate for CPP is arterial relaxation, measured by intra-arterial catheter  
|                                                         | • Arterial relaxation pressure less than 20 mm Hg, improve chest compressions and vasopressor therapy  

| Central Venous Oxygen Saturation | • Changes in SCVO(2) may reflect changes in oxygen deliver due to cardiac output changes  
|                                 | • SCVO(2) measured by oximetric tipped central venous catheters  
|                                 | • Placement in superior vena cava or pulmonary artery  
|                                 | • Normal range 60-80%  
|                                 | • Less than 30%, improve chest compressions and vasopressor therapy  

*Monitoring physiologic parameters with invasive monitors may optimize CPR and detect ROSC*
DRUG ACCESS ROUTES

Intravenous

- Peripheral IV preferred route if central line access not available
- Central line access may cause CPR interruptions and complications during insertion
- Be aware of vascular laceration, hematoma and bleeding
- Central line insertion in noncompressible vessel contraindication to fibrinolytic therapy
- Peripheral line establishment does not interrupt CPR
- Delay of 1-2 minutes of drugs to central circulation via peripheral line
- Drugs via peripheral line should be administered via bolus injection
- Elevate extremity 10-20 seconds to facilitate drug delivery

Intraosseous

- Provides safe drug delivery route if IV access not available
- IO access can be established for all aged patients
- Can be achieved in 30-60 seconds
- Preferred over ET route
- Can be used for any drug that could otherwise be administered via IV

Endotracheal

- Should be considered only if IV and IO administration not possible
- Optimal dose of most drugs via ET unknown
- Drug dose via ET typically 2 – 2.5 times that administered via IV
- Periodic CPR stops required to ensure drug has not regurgitated up ET tube

Fluid Administration

- Titrate fluid administration and vasoactive or inotropic agents to optimize blood pressure
- Mean arterial pressure 65 mm Hg or greater is reasonable goal
- Hypovolemic patients, ECF volume typically restored with normal saline or lactated Ringer’s
- Avoid D(5)W as it reduces serum sodium rapidly

OPIOID OVERDOSE

Naloxone is an opioid receptor antagonist in the spinal cord, brain and GI system and can reverse CNS and respiratory depression associated with opioid resuscitative emergencies. Administration may be via IV, intramuscular, intranasally, subcutaneously, nebulizer or ET tube.

*Administer naloxone immediately at a dose of 2 mg IN or 0.4 mg IM/IV,*

*repeating every four minutes if necessary*
Check if patient is unresponsive and attempt to reach nearby help. Send somebody to call 911 and obtain AED and naloxone.

If victim is not responsive and they are not breathing or only gasping, start CPR. If alone, complete 2 minutes of CPR before leaving to call 911 and obtain naloxone and AED.

Give naloxone as soon as possible. 2mg dose for intranasal or 0.4mg dose for intramuscular. Administer again after 4 minutes.

Is the person responsive? This includes purposeful movement, regular breath, moaning, etc.

If no: Continue CPR and use AED as soon as possible. Maintain this routine until person becomes responsive or advanced help arrives.

If yes: Continue to check for the person’s responsiveness and monitor breathing until advanced help arrives. If responsiveness stops, begin CPR and administer naloxone.

OPIOID ALGORITHM
SCENARIO: ADULT CARDIAC ARREST/PEA

PEA includes a group of rhythms organized or semi-organized but lacking palpable pulse, including:

- Sinus rhythm
- Ventricular escape rhythms
- Post-defibrillation idioventricular rhythms
- Idioventricular rhythms

Healthcare team members continue high-quality CPR throughout the BLS, primary and secondary assessments. CPR is then interrupted for no more than 10 seconds for rhythm and pulse checks. The pulseless patient with an organized rhythm is on the monitor is PEA and steps from the PEA pathway on the algorithm should be conducted next.

IV/IO access and airway management should be established. Healthcare team members should simultaneously assess the patient for underlying causes of PEA.

NONSCHOCKABLE RHYTHM

- No electrical activity, go back to step 10
- Organized electrical activity, try to palpate a pulse for 5-10 seconds
- No pulse, resume CPR for 2 minutes beginning with chest compressions
- Palpable pulse with organized rhythm, begin post-cardiac arrest care

SHOCKABLE RHYTHM

- Rhythm check indicates shockable rhythm, resume CPR beginning with compressions while defibrillator charges
- Switch to left side of algorithm, perform steps according to VF/pVT sequence beginning with steps 5 or 7

Hypovolemia and hypoxia are the most common underlying causes of PEA, both of which are reversible
CPR Quality
Push at least 2 inches and at a rate of 100-120/min for chest compressions. Be sure to minimize interruptions. Avoid the excess of ventilation. Switch compress or every 2 minutes. If no advanced airway is available, maintain a 30 compression to 2 breath ratio.
Quantitative waveform capnography: If PEEP is less than 10 mmHg, improve CPR quality.
Intra-arterial pressure: If diastolic pressure is less than 20 mmHg, improve CPR quality.

Shock Energy for Defibrillation
Biphasic: Use manufacturer recommendation. If that is unknown, use the maximum dose available. Subsequent doses should be equivalent in energy and a higher dosage should always be considered.
Monophasic: 360 J

Drug Therapy
Epinephrine dosage: 1 mg/3-5 minutes
Amiodarone dosage:
Dosage 1 of 300mg
Dosage 2 of 150mg

Advanced Airway
Endotracheal intubation or supraglottic advanced airway.
Do waveform capnography to ensure and monitor correct ET tube placement. Give 1 breath every 6 seconds with compressions once advanced airway in place.

Return of Spontaneous Circulation
Pulse and blood pressure. Greater than 40 mm Hg PETCO2. Spontaneous arterial pressure waves.

Reversible Causes
Hypovolemia
Hypoxia
Hydrogen Ion
Hypo/Hyperkalemia
Hypothermia
Tension Pneumothorax
Cardiac Tamponade
Toxins
Pulmonary Thrombosis
Coronary Thrombosis

1. Start CPR and be sure to give oxygen and attach monitor and defibrillator.
2. If shockable rhythm detected: VF/VT
3. Shock the patient
4. Give CPR for 2 minutes and obtain IV/IO access.
5. Is a shockable rhythm present?
   No
   11. If no: Give 2 minutes of CPR and treat reversible causes.
   Yes
5. If yes, provide a shock.
6. Give 2 minutes of CPR and give epinephrine every 3-5 minutes. Consider capnography.
7. Is a shockable rhythm present?
   No
   12. If signs of ROSC are not present go to steps 6 or 7.
   Yes
   Go to steps 5 or 7
8. If yes, provide a shock.
9. If shockable rhythm not detected: Asystole/PEA
10. Give CPR for 2 minutes, obtain IV/IO access, give epinephrine every 3-5 minutes, and consider capnography.
**SCENARIO: ASYSTOLE**

Asystole is a cardiac arrest rhythm with no discernible electrical activity on ECG. Healthcare providers should confirm the flat line on the monitor is true asystole and is not:

- Another rhythm that appears as a flat line
- Operator error
- Loose leads or leads not connected
- Loss of power
- Signal gain too low

**MANAGING ASYSTOLE**

High-quality CPR should be performed throughout the BLS, primary and secondary assessments. Interrupt CPR for no more than 10 seconds in order to perform rhythm check. Once determined as asystole, resume CPR beginning with chest compressions for 2 minutes. IV/IO access should be established while CPR is being performed. As soon as IV/IO access is established, 1 mg IV/IO epinephrine should be administered every 3-5 minutes without interrupting CPR. CPR should only be interrupted every 2 minutes to check rhythm.

**NONSHOCKABLE RHYTHM**

- No electrical activity, go back to step 10 or 11
- Electrical activity and organized, try to palpate pulse
- No pulse, continue 2 minutes of CPR beginning with chest compressions, Return to step 10
- Has pulse and organized rhythm, begin post-cardiac care

**SHOCKABLE RHYTHM**

- Prepare to deliver shock
- Resume chest compressions during charging if indicated
- Perform steps according to VG/pVT sequence, beginning with steps 5 or 7
TERMINATING RESUSCITATION

IN HOSPITAL

- Consider time from collapse to CPR
- Consider time from collapse to defibrillation
- Comorbid disease
- Prearrest state
- Initial arrest rhythm
- Response to resuscitation measures

OUT OF HOSPITAL

Continue efforts until:

- Restoration of spontaneous circulation, ventilation
- Presence of criteria indicating irreversible death
- DNR is presented
- Scene becomes unsafe for responder
SCENARIO: BRADYCARDIA

Bradycardia management steps include:

- Identifying slow-rate signs and symptoms
- Distinguishing those symptoms from unrelated causes
- Diagnosing the type of AV block
- Administering atropine
- Initiating transcutaneous pacing
- Administering epinephrine or dopamine
- Calling for expert consultation

Bradycardia is a rhythm disorder with a heart rate less than 60/min such as third degree AV block or sinus bradycardia

SIGNS AND SYMPTOMS

Identify the signs and symptoms of bradycardia through patient history and focused physical.

- Chest pain or discomfort
- Shortness of breath
- Altered level of consciousness
- Fatigue
- Weakness
- Dizziness
- Light-headedness
- Syncope
- Hypotension
- Diaphoresis
- Pulmonary congestion
- Congestive heart failure or PE

ASSESSMENT

Healthcare providers should assess the patient’s heart rate, determining whether or not it is less than 50/min. A primary assessment should then be conducted including maintaining an open airway, assisting breathing if necessary, assessing blood pressure, obtaining a 12-lead ECG and establishing IV access. Next, a physical exam and focused-history should be conducted to identify and treat potential contributing factors.
Determine if bradycardia is causing the symptoms or another illness is causing bradycardia.

BRADYCARDIA WITH PULSE ALGORITHM

1. Assess if clinical condition is appropriate. Heart rate typically less than 50/min if bradyarrhythmia.

2. Maintain airway and assist patient with breathing if necessary. If hypoxemic, give oxygen. Identify rhythm by cardiac monitor and monitor blood pressure and oximetry. Obtain IV access. Don’t delay treatment, but obtain 12-Lead ECG if available.

3. Is persistent bradyarrhythmia causing hypotension, acute altered mental status, signs of shock, chest discomfort, heart failure?

4. If no: Monitor and observe the person.

5. If yes: Give atropine. If the atropine is not effective: Transcutaneous pacing, dopamine infusion, or epinephrine infusion.

6. Consider consulting an expert and transvenous pacing.
TREATMENT SEQUENCE

Severity of the patient’s presentation determines the treatment sequence for bradycardia. Symptomatic bradycardia patients may be pre-cardiac arrest and require multiple simultaneous interventions. Delays in treatment should be avoided.

For type II second-degree or third-degree AV block or third-degree AV block with new wide QRS complex with a likely infranodal tissue block, reliance on atropine should be avoided. TCP or b-adrenergic treatment is preferred while patient is being prepared for transvenous pacing. When a bradyarrhythmia is unresponsive to atropine, b-adrenergic infusion is appropriate.

ATROPINE

Atropine is the most effective drug for acute symptomatic bradycardia if there are no immediately reversible causes by reversing cholinergic-mediated decreases in heart rate and AV node conduction.

For bradycardia, administer atropine 0.5 mg IV every 3-5 minutes to a total dose of 0.4 mg/kg

Maximum total atropine dose 3mg

Atropine doses of less than 0.5 mg may result in slowing the heart rate
PACING

TCP is a noninvasive treatment for symptomatic bradycardia and should immediately be considered by healthcare providers in unstable patients with high-degree heart block when IV access is not available, particularly in patients not responsive to atropine.

Electrical and mechanical capture should be confirmed after initiating pacing. The pacing rate should be set to the lowest effective rate. The patient should be reassessed to evaluate hemodynamic stability and symptom improvement.

TCP may be painful. Administer analgesics and sedatives with caution. Some may decrease blood pressure and alter mental status.

<table>
<thead>
<tr>
<th>Parenteral Benzodiazepine</th>
<th>For anxiety and muscle contraction</th>
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<tbody>
<tr>
<td>Parenteral Narcotic</td>
<td>For analgesia</td>
</tr>
<tr>
<td>Chronotropic Infusion</td>
<td>Use as soon as available</td>
</tr>
<tr>
<td>Consultation</td>
<td>Obtain expert consult for transvenous pacing</td>
</tr>
</tbody>
</table>

INDICATIONS FOR TCP USE

- Hemodynamically unstable bradycardia
- Hypotension
- Ischemic chest discomfort
- Acute heart failure
- Unstable condition likely attributed to bradycardia
- Bradycardia with symptomatic ventricular escape rhythms

TECHNIQUE

1. Place pacing electrodes on chest
2. Turn pacer on
3. Set demand rate to 60/min. Adjust once pacing established.
4. Set current milliamperes output 2 mA above dose at which consistent capture observed
**ASSESSMENT**

The goal of TCP is to improve the patient’s clinical status, as opposed to targeting a specific heart rate. After beginning pacing, adjust the rate based on the patient’s response. If the symptoms are attributable to bradycardia, typical patient’s improve to a rate of 60 to 70/min.

If pacing appears to be ineffective, chronotropic drug infusion is recommended as an alternative to stimulate heart rate.

| Epinephrine                  | • Initiate at 2 to 10 mcg/min  
|                              | • Titrated to patient response |
| Dopamine                     | • Initiate at 2 to 20 mcg/kg per minute  
|                              | • Titrated to patient response |

**STANDBY PACING**

Acute ischemia of conduction tissue and pacing centers may cause several bradycardic rhythms in ACS patients. Clinically stable patients may decompensate or become unstable over minutes to hours resulting from worsening conduction abnormalities.

TCP electrodes should be placed on patients with acute myocardial ischemia or infarction with the following rhythms in anticipation of clinical deterioration:

- Newly acquired left, right or alternating bundle branch block or bifascicular block in AMI setting
- Symptomatic sinus node dysfunction, severe symptomatic sinus bradycardia
- Asymptomatic Mobitz type II second-degree AV block
- Asymptomatic third-degree AV block

**EPINEPHRINE AND DOPAMINE**

Healthcare providers should assess the patient’s intravascular volume status and avoid hypovolemia when using epinephrine and dopamine because they are vasoconstrictors. If atropine is inappropriate or fails for the patient, epinephrine and dopamine infusion may be an alternative. Epinephrine infusion should be administered in a dose of 2 to 10 mcg/min and titrated to patient’s response. Dopamine infusion at 2 to 20 mcg/kg per minute and titrate to patient’s response.
SCENARIO: STABLE AND UNSTABLE TACHYCARDIA

Tachycardia, symptomatic or asymptomatic, may be attributed to a number of causes. Determining whether pulses are present is the primary key to managing tachycardia patients. Healthcare providers should determine whether or not the patient is stable when the patient has a pulse by assessing rhythm and condition. If tachyarrhythmia is sinus tachycardia, the healthcare provider should attempt to identify its cause.

UNSTABLE TACHYCARDIA

Unstable tachycardia occurs when heart rate is too fast for the patient’s condition. Excessive heart rate causes symptoms due to the following:

- Cardiac output is reduced because heart is beating so fast, possibly causing pulmonary edema, coronary ischemia and hypotension
- Condition between atrium and ventricles, or ventricles themselves reduce cardiac output because the heart is beating ineffectively

Signs and symptoms include:

- Hypotension
- AHF
- Altered mental status
- Shock
- Ischemic chest discomfort

 Quickly determine whether tachycardia is producing hemodynamic instability and symptoms or if symptoms are producing tachycardia.
ADULT TACHYCARDIA WITH PULSE ALGORITHM

Assess if clinical condition is appropriate. 150/min or more is typical tachycardia heart rate.

Maintain airway and assist patient with breathing if necessary. If hypoxicemic, give oxygen. Identify rhythm by cardiac monitor and monitor blood pressure and oximetry.

Is persistent tachycardia causing hypotension, acute altered mental status, signs of shock, chest discomfort, heart failure?

If no:
Gain IV access and 12-lead ECG if possible. Vagal maneuvers. If regular, adenosine. B-blocker or calcium channel block. Consider consultation of an expert.

If no:
Wide QRS? Greater than or equal to 0.12 seconds?

If no:
Gain IV access and obtain 12-lead ECG if possible. Consider adenosine only if monomorphic and regular. Also consider antiarrhythmic infusion and expert consultation.

If yes:
Synchronized cardioversion. Consider sedation. If narrow complex is regular, consider adenosine.

Doses/Details

Synchronized Cardioversion:
- Recommended initial dosage:
  - Narrow regular: 50-100J
  - Narrow irregular: 120-200J
  - Wide irregular: defibrillation dose (not synchronized)
  - Wide regular: 100J
- Adenosine:
  - Initial dose of 6 mg rapid IV push, followed by NS flush
  - Second dose: 12 mg, if necessary

Antiarrhythmic Infusions for Stable Wide-QRS Tachycardia:
- Procainamide Dose:
  - 20-50mg per minute until suppression of arrhythmia. Hypotension ensues. QRS duration increases by greater than 50%, or the maximum dosage (17mg/kg) is provided.
  - Maintenance infusion: 1-4mg/min
  - Avoid if QT or CHF are prolonged.
- Amiodarone Dose:
  - Initial dosage of 150mg over 10 minutes. If VT occurs, repeat.
  - Follow this by maintenance infusion of 1mg/min for initial 6 hours.
- Sotalol Dose:
  - 100mg over 5 minutes, but avoid if a prolonged QT is present.
CARDIOVERSION

Healthcare providers must be able to distinguish when cardioversion is indicated and what type of shock to administer.

Unsynchronized shock:

- Does not advise when to deliver shock
- Healthcare provider must determine when to deliver shock
- Shock may be delivered randomly at any point in cardiac cycle
- Higher energy level than synchronized cardioversion
- Use for pulseless patient, deteriorating patient or if uncertainty regarding monomorphic or polymorphic VT present

Synchronized shock:

- Advises when to deliver shock
- Sensor determines peak of QRS complex to deliver synchronized shock
- Avoids delivery of shock during cardiac repolarization
- Lower energy than unsynchronized cardioversion
- Use for unstable atrial fibrillation, atrial flutter, SVT or monomorphic tachycardia with pulses

Deliver initial 200-J synchronized shock – monophasic cardioversion

Deliver initial 120 – 200-J synchronized shock – biphasic cardioversion

For both, increase energy dose in steps for subsequent attempts
<table>
<thead>
<tr>
<th>Step</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Sedate all conscious patients unless the patient is unstable or deteriorating quickly.</td>
</tr>
<tr>
<td>2</td>
<td>Activate the defibrillator (monophasic or biphasic).</td>
</tr>
<tr>
<td>3</td>
<td>Attach leads from monitor to patient and make sure that rhythm is displayed properly. Place adhesive electrode pads on the patient.</td>
</tr>
<tr>
<td>4</td>
<td>Push the sync control button to enter synchronization mode.</td>
</tr>
<tr>
<td>5</td>
<td>Look for an indicator of sync mode, specifically the markers on the R wave.</td>
</tr>
<tr>
<td>6</td>
<td>Until sync markers occur with each R wave, adjust the gain of the monitor.</td>
</tr>
</tbody>
</table>
| 7    | Choose appropriate energy level. Deliver monophasic shocks in the sequence:  
   - Unstable atrial fibrillation – Initial Dose of 200J  
   - Unstable monomorphic VT – Initial Dose of 100J  
   - Other unstable SVT/atrial flutter – Initial Dose of 50 to 100J  
   - Polymorphic VT and unstable – Treat as VF with high-energy shock  
Biphasic waveforms of lower energy are usable if documented to be equivalent or superior to reported monophasic shock success. Extrapolation from elective cardioversion of atrial fibrillation supports an initial biphasic dosage of 120 to 200J with increase in dosage if necessary. For specific recommendations, consult the manufacturer. |
| 8    | Shout: “Charging defibrillator – Stand Clear!” to members of team. |
| 9    | Push the charge button. |
| 10   | Clear the patient once the defibrillator is charged. |
| 11   | Push the shock button. |
| 12   | Study the monitor. If tachycardia is still present, increase the energy level. |
| 13   | Activate sync mode after each shock delivery. Most defibrillators will default back to unsynchronized mode after a synchronized shock delivery. This default allows immediate shock delivery if cardioversion creates VF. |