

Cardiac Emergencies

**Assessment
of the
Patient with
Cardiac Disease**

Chief Complaint

–Chest pain or discomfort

- Onset
- Provocation/palliation
- Quality
- Region/radiation
- Severity

Timing

–Dyspnea

Historical factors important in differentiating breathing difficulties include the following:

- Duration and circumstances of onset of dyspnea

- Anything that aggravates or relieves the dyspnea, including medications

- Previous episodes

- Associated symptoms

- Orthopnea

- Previous cardiac problems

–Syncope

Syncope is a brief loss of consciousness often caused by a sudden decrease in oxygenated blood to the brain.

The history of a syncopal event should include the following:

- Presyncope aura

- Circumstances of occurrence

- Duration of syncopal episode

- Symptoms before syncopal episode

- Other associated symptoms

Previous episodes of syncope

–Abnormal heartbeat and palpitation

Many patients are aware of their own heartbeat, particularly if it is irregular.

Important information to obtain includes:

- Pulse rate

- Regular versus irregular rhythm

- Circumstances of occurrence

- Duration

- Associated symptoms (chest pain, diaphoresis, syncope, confusion, dyspnea)

- Previous episodes and frequency

Medication (drug stimulant) or alcohol use

Significant Medical History

Is the patient taking prescription medications, particularly cardiac medications?

Is the patient being treated for any other illness?

Does the patient have any allergies?

Does the patient have risk factors for a heart attack?

Does the patient have an implanted pacemaker, ventricular assist device, or ICD?

Physical Examination

Primary survey

Should include a more in-depth evaluation of the patient's level of consciousness, respirations, pulse, and blood pressure.

Physical examination

Look

- Skin

- Jugular veins

- Peripheral and presacral edema

- Additional indicators of cardiac disease

Listen

Lung sounds

Heart sounds

Carotid artery bruit

Feel

Skin

Pulse

Thorax and abdomen

Pathophysiology and Management of Cardiovascular Disease

Acute Coronary Syndromes

AMI and unstable angina are part of a spectrum of clinical diseases, collectively known as acute coronary syndrome (ACS). ACS is the most common cause of sudden cardiac death.

–The primary goals of therapy for patients with ACS include the following:

Reducing the amount of myocardial necrosis that occurs in patients with MI.

Preserving left ventricular function

Preventing heart failure

Preventing major adverse cardiac events (death, nonfatal MI, the need for urgent revascularization)

Treating acute, life-threatening complications of ACS (e.g., VF, pulseless VT, symptomatic bradycardias, unstable tachycardias)

Atherosclerosis

Atherosclerosis is a disease process characterized by progressive narrowing of the lumina of medium and large arteries.

Major risk factors

Atherosclerosis occurs to some extent in all middle-aged and older people. The disease also occurs in some young people. Atherosclerosis is thought to have an inheritable component.

Effects

Atherosclerosis has two major effects on blood vessels.

First, it disrupts the innermost lining of the vessels. This causes loss of vessel elasticity and an increase in the formation of clots.

Second, the atheroma reduces the diameter of the vessel lumen. This reduces the blood supply to tissues.

Angina Pectoris

Angina pectoris is a symptom of myocardial ischemia; the term literally means “choking pain in the chest.”

Angina is caused by an imbalance between myocardial oxygen supply and demand.

The result is a buildup of lactic acid and carbon dioxide in ischemic tissues of the myocardium.

Stable angina

Stable angina usually is precipitated by the increased myocardial demand of physical exertion.

–Unstable angina

Unstable angina may occur during periods of light exercise or at rest. The pain usually lasts longer than it does in stable angina.

Management

Place the patient at rest physically and emotionally.

Administer oxygen if the patient is dyspneic, has signs of heart failure, or has an oxygen saturation level, as measured by an arterial blood gas sampling (Sao₂), of less than 90%.

Administer aspirin: 160 to 325 mg, non-enteric coated.

Initiate IV therapy.

If the patient reports pain, use pharmacologic therapy.

Monitor the ECG for dysrhythmias.

Obtain a 12-lead ECG at first medical contact, and record serial 12-lead ECGs during transport.

Also measure, record, and communicate any ST-segment changes.

Transport the patient to a PCI-capable hospital for evaluation by a physician.

Myocardial Infarction

Participating events

The process of MI is complex.

It generally begins with the formation of an atherosclerotic plaque involving the intimal layer of a coronary artery.

The plaque disrupts the smooth arterial lining and results in an uneven surface that creates turbulent blood flow.

The plaque may rupture.

Types and locations of infarcts

Emergency care is directed at the following:

If the patient is hypoxic, increasing the oxygen supply by administering supplemental oxygen
Decreasing the metabolic needs and providing collateral circulation.

Reestablishing perfusion to the ischemic myocardium as quickly as possible after the onset of symptoms.

ACS can be classified into one of three ischemic syndromes based on the rupture of an unstable plaque in an epicardial artery:

unstable angina

non-STEMI

STEMI.

Death of myocardium

When blood flow to the myocardium stops, a series of events begins.

Cells switch from aerobic to anaerobic metabolism, resulting in the release of lactic acid and an increase in tissue carbon dioxide levels.

These changes contribute to ischemic pain (angina).

As cells lose their ability to maintain their electrochemical gradients, they begin to swell and depolarize.

These initial changes are reversible.

Death following MI

Death within the first week or two after MI usually results from lethal dysrhythmias, pump failure, or myocardial tissue rupture.

Signs and Symptoms

- Agitation
- Anxiety
- Cyanosis
- Diaphoresis
- Dyspnea
- Nausea and vomiting
- Palpitations
- Syncope
- Sense of impending doom
- Weakness

–Management of an uncomplicated AMI

All patients are assumed to have an AMI.

–Fibrinolytic therapy

The AHA strongly recommends the following:

–Protocols using fibrinolytic checklists

–12-Lead ECG acquisition, transmission, and interpretation

–Experience in advanced life support

–Communication with the receiving institution

–Medical director with training and experience in STEMI management

-Continuous quality improvement

Heart Failure

Heart failure is a chronic condition that affects the chambers of the heart, making the heart unable to pump blood at a rate that meets the metabolic needs of the tissues.

The condition can occur in all age groups (usually a result of congenital heart disease in children) but more commonly is associated with high-output heart failure in older adults

Left-side heart failure

Left-side heart failure occurs when the left ventricle fails to work as an effective forward pump.

Right-side heart failure

Right-side heart failure usually occurs as a result of left-side failure.

Pulmonary edema

Caused by excess fluid in the lungs. It can occur from pneumonia, following exposure to certain toxins and drugs, and from being at high altitudes.

Management

Pulmonary edema is an acute and critical emergency that can lead to death unless it is treated rapidly.

Emergency care entails patient positioning, oxygenation, continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BPAP), ventilator support as needed, and pharmacologic therapy.

The EMT should place the patient in a sitting position with the legs dependent.

The EMT should administer oxygen using noninvasive positive pressure ventilation (CPAP or BPAP).

Cardiogenic Shock

Cardiogenic shock is the most extreme form of pump failure.

It occurs when left ventricular function is so compromised that the heart cannot meet the metabolic needs of the body.

The result is a significant decrease in stroke volume (resulting from ineffective myocardial contraction), cardiac output, and blood pressure; these decreases produce an inadequate supply of blood to the organs.

-Management

Prehospital care should include the following:

- Airway management and ventilatory support
 - Placement of the patient in a supine position
 - Insertion of an IV line with normal saline or lactated Ringer solution and infusion of fluids
 - Monitoring of etco2
 - ECG monitoring
 - Correction of dysrhythmias
 - Assessment of blood glucose level and correction if less than 60 mg/dL
- Frequent evaluation of vital signs

Cardiac Tamponade

The condition may have a gradual onset.

It may result from a cancerous growth or infection.

Signs and symptoms of cardiac tamponade include the following:

- Chest pain that increases with deep breathing or coughing

- Decreased systolic pressure (a late sign)

- Palpitations

Elevated venous pressure (an early sign) with associated JVD

Faint or muffled heart sounds

Shortness of breath

Low-voltage QRS complexes and T waves

Alternating amplitude and vector of P waves, QRS complexes, and T waves (electrical alternans)

Pulsus paradoxus

Tachycardia

–Management

The EMT must obtain a thorough history to attempt to identify the cause of the cardiac tamponade.

The EMT should perform a physical examination.

Thoracic Aneurysms and AAAs

“Aneurysm” is a nonspecific term that means “dilation of a vessel.”

An aneurysm may result from atherosclerotic disease (most common), infectious disease (primarily syphilis), traumatic injury, or certain genetic disorders (e.g., Marfan syndrome).

AAA and dissecting aneurysm of the aorta are presented here.

Abdominal aortic aneurysms

Rupture of an AAA may begin with a small tear in the intima.

This small tear allows blood to leak into the wall of the aorta.

Management

In most cases, prehospital care should be limited to gentle handling, oxygen administration if hypoxemic, cardiac monitoring (including a 12-lead ECG), initiation of two IV lines (18-gauge or larger) enroute to the receiving hospital and alerting the receiving facility to prepare for imminent surgery.

Acute dissecting aortic aneurysm

Acute dissecting aortic aneurysm is the most common aortic catastrophe.

Aortic dissection may cause the following:

- Syncope
- Stroke
- Absent or reduced pulses
- Unequal blood pressure readings
- Heart failure resulting from sudden aortic valve regurgitation
- Pericardial tamponade
- AMI

Signs and symptoms

Sudden, severe chest or upper back pain

Sudden severe abdominal pain

Loss of consciousness

Shortness of breath

Sudden difficulty speaking, loss of vision

Stroke-like weakness or paralysis of one side of the body

Weak pulse in one arm or thigh

Leg pain

Difficulty walking

Management

Relieving pain and transporting to a medical facility

Other care measures include the following:

Handling the patient gently

Reducing anxiety

Treating nausea and vomiting with antiemetics

Administering high-concentration oxygen

Beginning a large-bore IV line of crystalloid solution

Administering a beta blocker to maintain a heart rate of 60 to 80 beats/min

Giving analgesia per medical direction if the diagnosis is strongly suspected

Acute Arterial Occlusion

–The location of the ischemic pain often is related to the site of occlusion:

- Terminal portion of the abdominal aorta
- Iliac artery
- Femoral artery
- Mesenteric artery

Signs and symptoms

- Pain in the extremity that may be severe and sudden in onset or absent as a result of paresthesia
- Pallor (the skin also may be mottled or cyanotic)
- Lowered skin temperature distal to the occlusion
- Changes in sensory and motor function
- Diminished or absent pulse distal to the injury
- Bruit over the affected vessel
- Slow capillary filling
- Sometimes shock

Management

Acute arterial occlusion in an extremity is serious and painful.

The occlusion may be limb threatening if blood flow is not reestablished within 4 to 8 hours.

The affected limb should be immobilized and protected.

In addition, the patient should be transported for evaluation by a physician.

Noncritical Peripheral Vascular Conditions

Predisposing factors to venous thrombosis include the following:

- Primary
 - Paralysis after spinal cord injury
 - Fractures of hip, pelvis, or long bones
 - Coagulopathies
 - Multisystem trauma
 - Cancer (especially metastatic)
 - Major general or orthopedic surgery

Other causes

Pregnancy or immediate postpartum period

Birth control pills or menopausal estrogen treatment

Age older than 50 years

Obesity (doubles risk)

Immobility

Family history

Genetic blood clotting disorders

Physical inactivity (such as a long flight)

Acute deep vein thrombosis

- Signs and symptoms of acute DVT include the following:
 - Pain
 - Edema
 - Warmth
 - Erythema or blue discoloration
 - Tenderness

Management

Patients with acute DVT who are at a low risk for pulmonary embolism may be treated at home.

Prehospital care usually is limited to immobilization and elevation of the extremity and transport for evaluation by a physician.

Hypertension

Chronic hypertension

Chronic hypertension has an adverse effect on the function of the heart and blood vessels.

Chronic hypertension leads to an increase in cardiac afterload.

Any hypertension-related illness, such as pulmonary edema, dissecting aortic aneurysm, preeclampsia and eclampsia of pregnancy, or stroke, requires stabilization and prompt, appropriate management.

Hypertensive crisis

Conditions in which an increase in blood pressure, usually greater than 180/120 mm Hg, can lead to significant, irreversible damage to organs.

May cause/worsen the following clinical conditions:

- myocardial ischemia with hypertension

- aortic dissection with hypertension

- pulmonary edema with hypertension

- hypertensive intracranial hemorrhage

- toxemia

- hypertensive encephalopathy

- renal failure

–Hypertensive encephalopathy results solely from elevated blood pressure, causing elevated intracranial pressure.

–Prehospital management of patients with a hypertensive emergency includes the following:

- Supportive care

- Calming the patient

- Oxygen therapy if indicated

- IV line to keep the vein open

- ECG monitoring

- Rapid transport

Valvular Heart Disease

Valvular heart disease refers to any disease process that affects one or more valves of the heart: the mitral, aortic, tricuspid, or pulmonary valves.

When one or more of these valves become narrowed, hardened, or thickened (stenotic), the valves do not open or close completely.

Valvular heart disease can be congenital.

In other cases, it can develop slowly, or it may be acute.

Infectious Heart Disease

Infectious heart disease is caused by intravascular contamination by pathogens.

The infections can damage the muscles and valves of the heart.

Endocarditis

Endocarditis is an infection of the endocardium (inner layer of the heart).

Endocarditis usually results from a bacterium that enters the bloodstream (bacterial or infective endocarditis).

Endocarditis may develop slowly or may be sudden in onset.

Signs and symptoms include:

Abnormal urine color

Chills (common)

Excessive sweating (common)

Fatigue

Fever (common)

Joint pain

Muscle aches and pains

Night sweats

Nail abnormalities (splinter hemorrhages under the nails)

Paleness

Red, painless skin spots on the palms and soles (Janeway lesions)

Red, painful nodes in the pads of the fingers and toes (Osler nodes)

Shortness of breath with activity

Swelling of the feet, legs, abdomen

Weakness

Weight loss

- Nail abnormalities (splinter hemorrhages under the nails)
- Paleness
- Red, painless skin spots on the palms and soles (Janeway lesions)
- Red, painful nodes in the pads of the fingers and toes (Osler nodes)
- Shortness of breath with activity
- Swelling of the feet, legs, abdomen
- Weakness
- Weight loss

Pericarditis

Pericarditis is inflammation of the pericardium (the fibrous sac surrounding the heart). It usually is a complication of a viral infection.

Signs and symptoms include the following:

- Swelling of the ankles, feet, and legs (occasionally)

- Anxiety

- Difficulty breathing when lying down

- Crackles

- Decreased breath sounds

Chest pain caused by the inflamed pericardium rubbing against the heart

May radiate to the neck, shoulders, back, or abdomen

Often increases with deep breathing and lying flat; may increase with coughing and swallowing
Pleuritic chest pain (often relieved by sitting up and leaning forward)

- Pericardial friction rub
- Dry cough
- Fatigue
- Fever
- 12-Lead ECG changes
 - Diffuse ST elevation
 - PR-segment depression
 - Notched J point

Myocarditis

- Myocarditis is inflammation of the heart muscle.
- Signs and symptoms that may occur with the disease include the following:
 - Abnormal heartbeat, sometimes leading to syncope
 - Chest pain that may be severe
 - Fever and other signs of infection (headache, muscle aches, sore throat, diarrhea, rashes)
 - Joint pain or swelling
 - Leg swelling
 - Shortness of breath
 - Decreased urine output

Myocarditis may lead to heart muscle damage, and the patient may need to be treated for heart failure.

Dysrhythmias may need to be managed with antidysrhythmic and insertion of a pacemaker or an ICD.

Depending on the severity of the damage to the heart, the patient may recover completely or may have permanent heart failure.

Mechanical Circulatory Support Devices

Mechanical circulatory support devices are now commonly used in the treatment of severe heart failure as bridges to cardiac transplantation, as destination therapy for patients who are not transplant candidates, and as bridges to recovery.

These devices, which can be used to support the left or right ventricles, or both, restore circulation to the tissues, thereby improving organ function.

Two common devices are the left ventricular assist device and the total artificial heart.

Left Ventricular Assist Device

A left ventricular assist device (LVAD) is a battery-operated implantable pump that is increasingly being used as a destination therapy for patients not eligible for or awaiting transplant.

The LVAD enhances, rather than replaces, the left ventricular contractility. Common complications are power disconnection and driveline failure. Both will stop the pump.

Special care considerations

Because the LVAD assumes the pumping function of the left ventricle but provides flow in a continuous manner, patients who have newer devices have no palpable pulse or measurable blood pressure.

Some patients with an LVAD do not lose consciousness if VF or VT develops because their pump maintains enough flow to perfuse the brain.

If you are called to care for a patient with an LVAD, proceed as follows:

Check the power source.

Then assess the level of consciousness, airway, and breathing.

Auscultate heart sounds.

If it is a continuous-flow device, a whirring sound will be heard.

Monitor the ECG rhythm.

Assess the device for alarms.

The controller (usually around the patient's waist) will have a colored tag to indicate the type of device and a resource number to call in an emergency.

Some agencies carry a color-coded resource guide to assist with troubleshooting.

Start a large-bore IV line.

Attempt to assess noninvasive MAP.

Transport to the closest LVAD center.

Bring any LVAD equipment with the patient to the hospital.

Transport the patient's significant other to help troubleshoot problems with the device enroute.

Total Artificial Heart

A total artificial heart (TAH) replaces both ventricles.

The ventricles are removed when the device is implanted.

The right pump is joined to the right atrium and pulmonary artery, and the left pump is connected to the left atrium and aorta.

An external pneumatic driver powers the pumps.

Cardiac Arrest and Sudden Cardiac Death

- It is becoming increasingly evident that patients who cannot be resuscitated in the prehospital setting rarely survive.

This is the case even if the patient is resuscitated temporarily in the ED.

Care of the Patient after ROSC

Some patients survive cardiac arrest and have a ROSC.

Reestablishment of effective perfusion of organs and tissues.

Ideally, the patient is alert and awake.

Patients also may be comatose, yet still have full potential for recovery with a good neurologic outcome.

The AHA recommends measures to promote optimal outcomes.

Some of these methods include hemodynamic and ventilation optimization, immediate coronary reperfusion with PCI, and therapeutic hypothermia.

Hemodynamic and Ventilation Optimization

To avoid hypoxia in adults with ROSC after cardiac arrest, it is reasonable to use the highest available oxygen concentration until the arterial oxyhemoglobin saturation or the partial pressure of arterial oxygen can be measured.

Some patients survive cardiac arrest and have a ROSC.

In these situations, the principal objective of post-resuscitation care is reestablishment of effective perfusion of organs and tissues.

Ideally, the patient is alert and awake.

Immediate Coronary Reperfusion with PCI

A 12-lead ECG should be acquired as soon as possible.

These therapies include immediate coronary reperfusion (e.g., PCI), therapeutic hypothermia, and other treatment modalities to improve post-cardiac arrest survival.

Therapeutic Hypothermia

Maintaining the body temperature between 32°C (89.6°F) and 36°C (96.8°F) reduces the intracranial pressure, the cerebral metabolic rate, and the brain's demand for oxygen consumption.

In addition, it is thought to suppress many of the chemical reactions associated with reperfusion injury, including free radical production, excitatory amino acid release, and calcium shifts.

Please complete the online test for two (2) hours of continuing education.