IN THE HOSPITAL SETTING, emergencies typically occur in emergency departments (EDs) and intensive care units (ICUs). But many also take place in progressive care units or general nursing units. And when they do, they can cause marked anxiety for nurses—especially those unfamiliar or inexperienced with the drugs used in these emergencies.

Generally, the goal of using emergency drugs is to prevent the patient from deteriorating to an arrest situation. This article helps nurses who don’t work in ICUs or EDs to understand emergency drugs and their use.

Under normal circumstances, a registered nurse (RN) needs a physician’s order to administer medications. In emergencies, RNs with advanced cardiac life support (ACLS) certification can give selected drugs based on standing orders, relying on algorithms that outline care for certain emergencies. Wherever possible, nurses should strive to maintain proficiency in basic life support (BLS), as the latest research shows the importance of effective cardiopulmonary resuscitation. Some non-ICU nurses may want to pursue ACLS training as well.

Drugs for acute coronary syndrome
Acute coronary syndrome (ACS) refers to a spectrum of clinical manifestations associated with acute myocardial infarction and unstable angina. In ACS, a plaque in a coronary artery ruptures or becomes eroded, triggering the clotting cascade. A blood clot forms, occluding the artery and interrupting blood and oxygen flow to cardiac muscle.

Many healthcare providers use the acronym MONA to help them remember the initial medical treatment options for a patient with ACS.

M: morphine
O: oxygen
N: nitroglycerin
A: aspirin.

But keep in mind that while MONA might be easy to remember, the drugs aren’t given in the MONA sequence. They’re given in the order of OANM.

Oxygen
Oxygen (O₂) is given if the patient’s O₂ saturation level is below 94%. The heart uses 70% to 75% of the oxygen it receives, compared to skeletal muscle, which uses roughly 20% to 25%.

Aspirin
The standard recommended aspirin dosage to treat ACS is 160 to 325 mg, given as chewable “baby” aspirin to speed absorption. Aspirin slows platelet aggregation, reducing the risk of further occlusion or re-occlusion of the coronary artery or a recurrent ischemic event.

Nitroglycerin
To help resolve chest pain from ACS, nitroglycerin 0.4 mg is given sublingually via a spray or rapidly dissolving tablet. If the first dose doesn’t reduce chest pain, the dose can be repeated every 3 to 5 minutes for a total of three doses.

A potent vasodilator, nitroglycerin relaxes vascular smooth-muscle beds. It works well on coronary arteries, improving blood flow to ischemic areas. It also decreases myocardial oxygen consumption, allowing the heart to work with a lower oxygen demand. In peripheral vascular beds, nitroglycerin causes vasodilation and reduces preload and afterload, resulting in decreased cardiac workload.

If chest pain recurs once the initial pain resolves or decreases, the patient may be placed on a continuous I.V. infusion of nitroglycerin. Because of the drug’s vasodilatory effects, be sure to institute continuous blood-pressure monitoring.

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Morphine
If chest pain doesn’t resolve with sublingual or I.V. nitroglycerin, morphine 2 to 4 mg may be given every 5 to 15 minutes via I.V. push. An opioid acting primarily on receptors that perceive pain, morphine also acts as a venodilator, reducing ventricular preload and cardiac oxygen requirements.

As with nitroglycerin, the patient’s blood pressure needs to be monitored continuously. If hypotension occurs, elevate the patient’s legs, give I.V. fluids as ordered, and monitor for signs and symptoms of pulmonary congestion.

Other medications for ACS
Metoprolol may be used in the initial treatment of ACS. A cardioselective (beta_1 receptor) drug, it’s a beta-adrenergic blocker that dilates peripheral vascular beds, in turn reducing blood pressure, decreasing cardiac workload, and lowering cardiac oxygen demands. It also may have a mild analgesic effect in ACS-related chest pain. The patient’s blood pressure must be monitored. (See Be cautious with beta blockers.)

A primary goal of ACS treatment is to minimize muscle cell damage, which necessitates restoring blood flow to cardiac muscle. Drugs that may be used to reduce expansion of the arterial occlusion or restore blood flow to cardiac muscle include:

- heparin or enoxaparin (a low-molecular-weight heparin), which helps prevent the original clot from expanding and allows it to break down on its own; as a result, the vessel opens and new clot formation is inhibited.
- glycoprotein IIb-IIIa inhibitors, such as abciximab (Reopro). These drugs bind to glycoprotein IIb-IIIa receptor sites on platelets, preventing further aggregation and stopping expansion of the original clot or formation of new clots.
- fibrinolytics, such as reteplase (Retavase) and alteplase (Activase). These agents break down the original clot, opening the vessel for blood flow. (See Drugs used to treat acute coronary syndrome.)

Intervening for bradycardia
In bradycardia, the heart rate slows to a critical point and hemodynamic instability occurs. Usually, bradycardia is defined as a heart rate slower than 60 beats/minute (bpm). But in some patients, hemodynamic instability may occur at faster rates. This instability may manifest as dizziness, light-headedness, nausea, vomiting, hypotension, syncope, chest pain, and altered mental status. Atropine, epinephrine, and dopamine may be used to treat bradycardia, with dosages depending on the acuity and severity of hemodynamic instability.

For symptomatic patients, the healthcare team must determine the cause of bradycardia. In many cases, bradycardia results from use of other drugs, specifically other antiarrhythmics—for instance, beta blockers and calcium channel blockers. So those drugs may need to be withheld temporarily until their effects wear off. Beta blockers reduce circulating catecholamine levels, decreasing both the heart rate and blood pressure.

Typically, atropine is the drug of choice for symptomatic bradycardia. An anticholinergic and potent belladonna alkaloid, it increases the heart rate, which improves hemodynamic stability.

Epinephrine may be used as a secondary measure if atropine and temporary heart pacing don’t improve hemodynamic stability. Among other actions, epinephrine stimulates beta_1 receptors, causing cardiac stimulation, which in turn increases the heart rate.

Dopamine also may be used to support hemodynamic status by correcting hypotension. It enhances cardiac output, minimally increasing oxygen consumption and causing peripheral vasoconstriction.

If your patient is receiving these I.V. drugs, be sure to monitor for extravasation, which could lead to tissue damage. If possible, use a central line to deliver epinephrine and dopamine.

Intervening for tachycardia
Tachycardia, which usually refers to a heart rate faster than 100 bpm, may result from various cardiac mechanisms. The first step in choosing the right drug is to identify the origin of the arrhythmia. Most tachycardias are classified as one of two types:

- narrow-QRS-complex tachycardias (for instance, atrial fibrilla-
Each type calls for a slightly different treatment. Narrow-QRS-complex tachycardias with a regular rate generally are treated with adenosine, along with beta blockers, calcium channel blockers, and/or amiodarone or ibutilide.

With a wide-QRS-complex tachycardia, the first step is to determine if the arrhythmia is a ventricular tachycardia or is conducted with aberrancy. Wide-QRS-complex tachycardias with aberrancy call for the same treatment as narrow-QRS-complex tachycardias. On the other hand, ventricular tachycardia in a patient with a pulse is treated with amiodarone alone or with amiodarone in conjunction with synchronized cardioversion.

**Adenosine.** This general antiar-
This table lists some of the more common drugs used in the emergency treatment of arrhythmias, particularly bradycardia and tachycardia. Other types of arrhythmias may require other medications and interventions.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage and delivery</th>
<th>Indication</th>
<th>Action</th>
<th>Nursing implications</th>
</tr>
</thead>
</table>
| Adenosine           | 6 mg by rapid I.V. push, followed by 10 to 20 mL of normal saline solution (NSS) flush; may repeat a 12-mg dose twice, followed by NSS flush | Tachycardia | Depresses sinoatrial and atrioventricular node activity, slowing the heart rate | • Know that drug causes brief period of asystole. Monitor for underlying atrial activity during this time.  
• Be aware that drug triggers a flushing sensation. |
| Amiodarone          | 150-mg bolus given I.V. over 10 minutes, followed by continuous I.V. infusion at 1 mg/minute for 6 hours, then 0.5 mg/minute for 18 hours | Tachycardia | Reduces the heart rate                                                  | • Know that drug has long half-life (28 to 110 days), rarely affects blood pressure, and may cause thrombo-plebitis.  
• Monitor for drug allergy or reaction.  
• Mix infusion in glass bottle. |
| Atenolol, metoprolol| 5-mg I.V. bolus over 5 minutes; may repeat atenolol dose once and metoprolol dose twice | Tachycardia | Reduce catecholamines, leading to slower heart rate and lower blood pressure | • Monitor for bradycardia and/or pauses in heart rhythm.  
• Monitor blood pressure for hypotension. |
| Atropine            | 0.5 to 1 mg by I.V. push                          | Bradycardia | Increases the heart rate through anticholinergic effect               | • Be aware that dosages below 0.5 mg may further slow the heart rate.  
• Monitor for rebound tachycardia.  
• Monitor blood pressure for improvement. |
| Diltiazem           | 5 to 20 mg by I.V. push over 2 to 5 minutes, followed by I.V. infusion or additional 20 to 25 mg by I.V. push after 15 minutes | Tachycardia | Lengthens cardiac cycle, slowing the heart rate                        | • Monitor for bradycardia and/or pauses in heart rhythm.  
• Monitor blood pressure for hypotension.  
• Titrate dosage in small increments to achieve desired heart rate.  
• Begin oral drugs before stopping infusion, unless severe bradycardia, pauses in heart rhythm, and/or hypotension occur. |
| Dopamine            | 5 to 15 mcg/kg/minute by I.V. infusion           | Bradycardia | Stimulates dopamine receptors and increases cardiac output, with minimal increase in oxygen consumption; causes peripheral vasoconstriction | • Monitor for rebound tachycardia and/or hypertension.  
• Monitor blood pressure for improvement.  
• Titrate dosage in small increments to desired effect.  
• If possible, deliver via central line. |
| Epinephrine         | 2 to 10 mcg/minute by I.V. infusion              | Bradycardia | Stimulates beta<sub>1</sub> receptors, causing cardiac stimulation     | • Monitor for rebound tachycardia and/or hypertension.  
• Monitor blood pressure for improvement.  
• If possible, deliver via central line. |
Cardiac emergencies can occur at any time in any patient. Being familiar with the actions, dosages, rhythmic is used mainly as a diagnostic agent to identify the origin of an underlying narrow-QRS-complex tachycardia. It briefly depresses the atrioventricular (AV) node and sinus node activity. When given by rapid I.V. bolus, the drug's primary action is to slow electrical impulse conduction through the AV node. Be aware that adenosine commonly causes a few seconds of asystole, but because of its short half-life (6 to 10 seconds), the asystole usually is brief. The drug sometimes restores a normal sinus rhythm; if it doesn’t, calcium channel blockers and beta blockers may be given immediately to control the heart rate while amiodarone or ibutilide may be used to help restore a normal sinus rhythm.

**Diltiazem.** A first-line agent in controlling heart rate in narrow QRS-complex tachycardias, this drug can be used both in patients with preserved cardiac function and in those with impaired ventricular function (ejection fraction below 40%) or heart failure. (Verapamil, another calcium channel blocker, should be used only in patients with preserved cardiac function.)

A calcium channel blocker, diltiazem slows and/or blocks electrical impulse conduction through the AV node, reducing the number of impulses that arrive at the ventricular tissue and slowing the heart rate. It may cause hypotension secondarily to vascular smooth-muscle relaxation. Also, it may block impulses in some narrow-QRS-complex tachycardias that involve AV nodal reentry, thereby terminating the rhythm and restoring normal sinus rhythm.

**Other drugs.** Occasionally, selected beta blockers are used to help control the heart rate associated with narrow-QRS-complex tachycardias. They include metoprolol, atenolol, propranolol, and esmolol. Propranolol isn’t cardioselective and can affect pulmonary function, so it’s used less often. Typically, esmolol is given only in the ICU.

Atenolol is administered as a 5-mg I.V. bolus over 5 minutes. If the patient tolerates the dose and the arrhythmia persists after 10 minutes, an additional bolus of 5 mg may be given over 5 minutes. Metoprolol also is administered I.V. in 5-mg increments over 5 minutes; the dose may be repeated twice, to a total of 15 mg.

Don’t give beta blockers or calcium channel blockers to patients with narrow-QRS-complex tachycardias suspected of being pre-excitation arrhythmias, such as Wolf-Parkinson-White (WPW) syndrome. Such arrhythmias allow impulses to flow from the atria to the ventricles through an accessory or alternate pathway. Beta blockers and calcium channel blockers may increase the number of impulses arriving at ventricular tissue, further speeding the heart rate.

**Amiodarone.** This drug is used to treat certain narrow- and wide-QRS complex tachycardias identified as ventricular tachycardia or tachycardias of unknown origin. Although a class III antiarrhythmic, it has some properties of all antiarrhythmic classes. Its primary action is to block potassium channels in the cell, but it also prolongs the action potential duration, depresses conduction velocity, slows conduction through and prolongs refractoriness in the AV node, and has some alpha-, beta-, and calcium-channel blocking capabilities.

Dosing depends on circumstances. When used to treat ventricular tachycardia in patients with a pulse, runs of paroxysmal ventricular tachycardia, or narrow-QRS-complex tachycardias, amiodarone is given as a bolus of 150 mg over 10 minutes, followed by a continuous I.V. infusion starting at 1 mg/minute for 6 hours and then 0.5 mg/minute for 18 hours. If the patient is on nothing-by-mouth status for an extended time, the infusion can be kept running at 0.5 mg/minute. Otherwise, an oral dose usually is started before the infusion ends.

**Any time, any place**

Cardiac emergencies can occur at any time in any patient. Being familiar with the actions, dosages,
and rationales for commonly used emergency drugs will help you manage any crisis with confidence and efficiency.

Jolly M, Lincoff AM. Chapter 7, Medications used in the management of acute coronary syndrome. The Cardiac Care Unit Survival Guide. Philadelphia: Lippincott Williams & Wilkins; 2012.


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**POST-TEST • Emergency cardiac drugs: Essential facts for med-surg nurses**

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**Please circle the correct answer.**

1. The acronym MONA refers to the four drugs (morphine, oxygen, nitroglycerin, and aspirin) used initially to treat acute coronary syndrome (ACS). In which order should these drugs be given?
   a. MONA
   b. OANM
   c. OMNA
   d. MANO

2. The standard recommended dosage of aspirin for the treatment of ACS is:
   a. 75 to 100 mg.
   b. 125 to 150 mg.
   c. 160 to 325 mg.
   d. 325 to 350 mg.

3. If an initial dose (0.4 mg) of nitroglycerin doesn’t reduce chest pain, the dose can be repeated every 3 to 5 minutes, for a total of how many doses?
   a. Three
   b. Four
   c. Five
   d. Six

4. Which drug reduces ventricular preload and cardiac oxygen requirements?
   a. Reteplase
   b. Abciximab
   c. Enoxaparin
   d. Morphine

5. Which drug acts on beta1 receptors?
   a. Atelplase
   b. Aspirin
   c. Eptifibatide
   d. Metoprolol

6. What is the usual drug of choice for patients with symptomatic bradycardia?
   a. Atenolol
   b. Heparin
   c. Atropine
   d. Amiodarone

7. Patients with severe left ventricular failure should not receive:
   a. amiodarone.
   b. enoxaparin.
   c. metoprolol.
   d. nitroglycerin.

8. Which drug may cause a short period of asystole when given to treat tachycardia?
   a. Diltiazem
   b. Adenosine
   c. Amiodarone
   d. Metoprolol

9. Which drug lengthens the cardiac cycle, thus slowing the heart rate?
   a. Diltiazem
   b. Atropine
   c. Dopamine
   d. Epinephrine

10. For a patient who has hypotension stemming from bradycardia, what dosage of dopamine typically is given?
    a. 2 to 5 mcg/kg/minute by I.V. infusion
    b. 5 to 8 mcg/kg/minute by I.V. infusion
    c. 5 to 15 mcg/kg/minute by I.V. infusion
    d. 15 to 20 mcg/kg/minute by I.V. infusion

11. An example of a wide-QRS-complex tachycardia is:
    a. atrial flutter.
    b. heart block.
    c. atrial fibrillation.
    d. ventricular tachycardia.

12. Which drug is used to treat ventricular tachycardia in patients who have a pulse?
    a. Amiodarone
    b. Adenosine
    c. Atenolol
    d. Diltiazem

13. Patients with narrow-QRS-complex tachycardias suspected of being pre-excitation arrhythmias, such as Wolff-Parkinson-White (WPW) syndrome, should not receive:
    a. heparin.
    b. morphine.
    c. metoprolol.
    d. alteplase.

14. Which drug’s primary action is to block potassium channels in the cell?
    a. Dopamine
    b. Amiodarone
    c. Atropine
    d. Epinephrine

15. An infusion for which drug should be mixed in a glass bottle?
    a. Amiodarone
    b. Dopamine
    c. Epinephrine
    d. Heparin
Drugs for acute respiratory distress

Acute respiratory distress refers to a situation in which a patient becomes short of breath and may need emergency treatment to avoid an untoward outcome. For instance, acute pulmonary edema can result from changes in the Starling forces (hydrostatic and oncotic pressure) that allow ventilation in the lung fields; these changes permit fluid to enter the alveoli. Acute decompensated heart failure is a primary cardiogenic cause of acute respiratory distress and pulmonary edema.

Pulmonary edema manifests as rapid movement of fluid into the alveoli, causing acute shortness of breath, “wet” breath sounds (crackles in the lung fields), decreased ability to maintain adequate O₂ saturation, and in some cases pink, frothy sputum. Increased anxiety also may occur. Treatment focuses on restoring the Starling forces to normal. Usually, this entails reducing the amount of fluid entering the lung fields from the right side of the heart (called preload).

In many cases, pulmonary edema results from fluid overload. Typically, the patient’s fluid and sodium intake are limited and a diuretic (commonly furosemide) is given to eliminate excess fluid. Furosemide acts on the kidney’s proximal and distal tubules and the loop of Henle, causing excretion of water and some electrolytes (most notably potassium). The typical dosage is 20 to 40 mg by I.V. push delivered over 1 to 2 minutes. Watch the patient for hypotension, excessive diuresis, and hypokalemia; also monitor urine output closely. Be aware that supplemental potassium may be given orally to offset urinary potassium losses. As excess fluid is excreted, alveolar fluid returns to the intravascular compartment; shortness of breath, “wet” breath sounds, and anxiety levels decrease; and O₂ saturation returns to baseline.

Supplemental O₂ should be given during this emergency. Morphine may be used to relieve pulmonary congestion, lower myocardial oxygen demands, and reduce anxiety. Typically, morphine is given as 2 to 4 mg by I.V. push over 1 to 2 minutes. It usually causes a flushed feeling and can lead to hypotension and sedation, so be sure to monitor the patient carefully. (See Drugs used to treat pulmonary edema.)

Drugs used to treat pulmonary edema

This table presents selected drugs used to treat emergency episodes of acute pulmonary edema. To prevent further episodes, the healthcare team should identify and treat the underlying cause of pulmonary edema.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage and delivery</th>
<th>Action</th>
<th>Nursing implications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Furosemide</td>
<td>20 to 40 mg by I.V. push over 2 minutes; repeat if needed</td>
<td>Acts on proximal and distal tubules and loop of Henle to cause excretion of water and some electrolytes (most notably potassium)</td>
<td>• Monitor urine output to evaluate drug efficacy.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Monitor blood pressure.</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>• Monitor blood potassium level; give potassium supplements if needed and ordered.</td>
</tr>
<tr>
<td>Morphine</td>
<td>2 to 4 mg by I.V. push over 1 to 2 minutes</td>
<td>Relieves pulmonary congestion, lowers myocardial oxygen demands, and reduces anxiety</td>
<td>• Monitor for hypotension.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Watch for sedation.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Monitor respiratory effort and function.</td>
</tr>
<tr>
<td>Oxygen (O₂)</td>
<td>1 to 15 L via appropriate delivery device</td>
<td>Increases amount of oxygen available to red blood cells for delivery to body tissues</td>
<td>• Use appropriate delivery device for amount of O₂ delivered.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Use carefully in patients with chronic obstructive pulmonary disease.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Titrate dosage downward as appropriate.</td>
</tr>
</tbody>
</table>