14. DEEP VEIN THROMBOSIS AND PULMONARY EMBOLISM

Cheryl Hubner, RN, MS, CCRN

1. Define deep vein thrombosis (DVT).

DVT is a blood clot that has formed in a vein. It usually starts as a cluster of platelets and fibrin growing out of a vein valve pocket in an intramuscular venous sinus of the leg or along a vein wall that has been traumatized. The clot grows in the direction of blood flow, with the tail of the clot floating freely in the vein lumen. The clot continues to form, adheres more firmly to the vein wall, and blocks flow. The clot continues to grow both proximally and distally until the next vein branching is reached or until the clotting process is halted by anticoagulation therapy. A thrombus may form in any vein, but it is found most commonly in the lower extremities. Upper-extremity DVTs are more likely in patients who have a central venous catheter, are undergoing chemotherapy, or have a malignancy.

2. Define pulmonary embolism (PE).

PE is a free-floating particle that lodges in a pulmonary artery or arteriole. A massive PE usually lodges in a central pulmonary artery. A moderate-sized embolus lodges in an arteriole. Multiple tiny, undetectable emboli can shower the distal pulmonary arterioles, resulting in pulmonary
hypertension. Most PEs are fragments of a thrombus originating in the right heart or the deep veins of the upper extremities, thigh, or pelvis. Nonthrombotic causes of PE include amniotic fluid emboli, fragments of a trophoblast, cotton-wool fragments, talcum or starch particles, fat or fat cells, air, megakaryocytes, tumor fragments, and ova of parasites.

3. Why is DVT dangerous?
The two most serious consequences of DVT are PE and phlegmasia cerulea dolens (or venous gangrene). PE occurs in approximately 35% of patients with untreated DVT. Venous gangrene is rare, occurring in patients with extensive iliofemoral thrombosis. A late complication of DVT is chronic venous insufficiency, called postphlebitic syndrome. DVT leads to vein valve damage, chronic reflux across the damaged valve, increased venous pressure, chronic leg swelling, and skin ulcerations.

4. Why is PE dangerous?
The consequences of PE depend on the size of the embolus. A massive PE is often fatal. It causes severe pulmonary obstruction, which progresses rapidly to acute hemodynamic instability, right-sided heart failure, and hypoxemia. Pulmonary pressures rise only modestly, but right ventricular and atrial pressures rise sharply, resulting in right ventricular dilatation and failure. Most emboli do not obstruct the pulmonary vessel completely but allow some flow around them.

If the patient survives the immediate event, the clot begins to reorganize within a few hours. It becomes partially recanalized, allowing blood flow through it, and shrinks in size. Small fresh pulmonary emboli that lodge in the smaller arterioles dissolve spontaneously through a process called fibrinolysis. The pulmonary endothelium is a reservoir for plasminogen activator, which triggers fibrinolysis. A serious but rare consequence of PE is pulmonary infarction (death of embolized pulmonary tissue). Infarction is more likely if the embolus completely blocks a large artery or if the patient has preexisting lung and heart disease.

5. Why are critically ill patients at risk for developing DVT or PE?
Every patient admitted to a critical care unit should be considered at risk for a thromboembolic event and should receive appropriate prophylactic therapy. Critically ill patients frequently have some or all of the three factors that favor clot formation: stasis of blood, alterations in the blood coagulation system, and abnormalities of the blood vessel wall. Bedrest, casts, restraints, paralytic agents, local pressure, increased blood viscosity (from, for example, dehydration, polycythemia vera, sickle cell disease), hypotension, and states of low cardiac output contribute to stasis of blood, especially in the lower extremities. The stasis of blood allows activated clotting factors to accumulate in a localized area and form a clot. Stasis also decreases the liver’s ability to filter activated clotting factors from the blood stream. Malignancy, chemotherapy, trauma, pregnancy, oral estrogen therapy, acidosis, sepsis, surgery, induction of anesthesia, and burns activate clotting factors. Injury to the vein wall is caused by local trauma, acidosis, and bacterial endotoxins. Local trauma is caused by intravascular devices, burns, surgery, retractors, and manipulation of local veins during orthopedic surgery.

6. What congenital or acquired conditions increase the risk of thrombotic events?

<table>
<thead>
<tr>
<th>INHERITED HYPERCOAGULABLE STATES</th>
<th>ACQUIRED HYPERCOAGULABLE STATES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Factor V Leiden mutation</td>
<td>Diabetes</td>
</tr>
<tr>
<td>G20210A prothrombin gene mutation</td>
<td>Nephrotic syndrome</td>
</tr>
<tr>
<td>Dysplasminogenemias</td>
<td>Prolonged immobilization</td>
</tr>
<tr>
<td>Dysfibrinogenemia</td>
<td>Malignancy</td>
</tr>
<tr>
<td>Protein C, S, or antithrombin III</td>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>deficiency</td>
<td>Obesity</td>
</tr>
<tr>
<td>Heparin cofactor II deficiency</td>
<td>Paroxysmal nocturnal</td>
</tr>
<tr>
<td>Lupus anticoagulant disorder</td>
<td>Sickle cell disease</td>
</tr>
<tr>
<td>Homocysteinemia</td>
<td>Congestive heart failure</td>
</tr>
</tbody>
</table>
|                                  |                               | (From Schell: Critical Care Nursing Secrets, Philadelphia, 2001, Hanley & Belfus)
7. What are the two most common inherited hypercoagulable states?

The two most common inherited hypercoagulable states are activated protein C resistance, known as factor V Leiden mutation, and G20210A prothrombin gene mutation. Factor V Leiden is usually caused by a mutation that alters the binding site of factor V for activated protein C. It occurs in 5% of the general population and 20–40% of unselected patients with DVT.

8. Is a sequential compression device (SCD) more effective than a thromboembolic stocking (TED) in preventing a DVT?

Preventive measures are most successful when matched to the patient’s risk for developing a thromboembolic event. Thromboembolic stockings and foot exercises are appropriate prophylaxis in patients at low risk for DVT, who are well hydrated and mobile.

In patients at moderate risk, SCD prophylaxis is appropriate when initiated before a clot has started to form and when used consistently until the patient is ambulatory.

In patients at high risk, prophylaxis is best achieved with a combination of mechanical and pharmacologic therapy. Pharmacologic therapy may consist of an adjusted dose of oral anticoagulant therapy or fixed low-dose unfractionated heparin, 5000 U subcutaneously every 8–12 hours. Mechanical therapy can be provided by foot impulse technology or SCD. Neurosurgical patients are treated with an SCD alone because of the increased risk of bleeding when anticoagulant therapy is used. Low-molecular-weight heparin (LMWH) has been effective in preventing DVT in patients undergoing elective knee replacement. More research is needed to determine the applicability of LMWH to other populations.

9. What are the signs and symptoms of DVT?

The manifestations of a DVT vary widely from no symptoms to pain, fever, unilateral extremity swelling, and increased superficial venous patterning. Symptoms depend on the size and location of the thrombus and the adequacy of collateral flow through the superficial veins. Homans’ sign (calf pain on dorsiflexion of the foot) is a poor test for DVT because it is positive in about 50% of patients with DVT, and 40% of patients without DVT.

10. What are the signs and symptoms of PE?

The most common signs and symptoms of PE are dyspnea, pleuritic chest pain, and tachypnea, which occur in about 97% of patients. Eighty-one percent of patients with PE will have a partial pressure of arterial oxygen (PaO₂) < 80%, and 89% have an alveolar-arterial gradient > 20 mmHg. The signs and symptoms of a PE are related to the size of the occluded or partially occluded artery. Massive PEs lodging in a major pulmonary artery cause acute decompensation, hemodynamic collapse, jugular vein distention, shortness of breath, tachypnea, tachycardia, hypotension, and occasionally chest pain. A loud pulmonic second heart sound (P₂) may be auscultated. PEs of moderate size may present with pleuritic pain, dyspnea, slight fever, cough with blood-tinged sputum, tachycardia, and possibly a pleural rub. Small emboli are asymptomatic because they gradually obliterate the pulmonary capillary bed. In time, they cause pulmonary hypertension, a right ventricular heave, and a loud P₂.

11. What is compartment syndrome?

In patients with compartment syndrome, elevated pressure within an osteofascial compartment threatens the function of the leg or arm. Intracompartmental pressures are ≥ 30–40 mmHg. Lower intracompartmental pressures can cause ischemia in patients with low cardiac output. Increased pressure results in decreased perfusion to the extremity muscle, ischemia, and destruction of the muscle.

12. What are the symptoms of compartment syndrome?

- Pain in the affected extremity that increases with passive movement of the affected muscle
- Numbness, especially in the web space between the first and second toes
- Taut edema in the affected extremity
- Pulses are usually present in the extremity until the swelling is extreme

(From Schell: Critical Care Nursing Secrets, Philadelphia, 2001, Hanley & Belfus)
13. How do you differentiate DVT from compartment syndrome?

Both DVT and compartment syndrome present with similar symptoms: leg pain and swelling specific to the affected extremity. The quality of the pain is difficult to differentiate. The swelling with DVT may not be as tight as the swelling that accompanies compartment syndrome. The tense limb edema caused by compartment syndrome may feel hard like a football. In addition, patients with compartment syndrome may experience loss of sensation and movement in the affected extremity and complain of numbness between the first and second toes, decreased sensation in the foot, and decreased ability to dorsiflex the foot or wiggle the toes. In addition, the risk factors for DVT and compartment syndrome are different. Risk factors for DVT are listed in question 5. Risk factors for compartment syndrome are traumatic injury to the limb, prolonged period of hypoperfusion to the limb followed by reperfusion, and extreme swelling of a limb.

14. What tests are used to diagnose DVT?

Tests available to detect DVT are venography, impedance plethysmography (IPG), ultrasonography, and magnetic resonance imaging (MRI). Radionuclide studies, such as I-125 fibrinogen uptake, are rarely used except during research studies. Venography is the most sensitive test for DVT of the calf vein. Compression ultrasonography (duplex ultrasonography with color Doppler imaging) is highly reliable for DVT above the knee. MRI is excellent for detecting pelvic and lower extremity thrombi and for ruling out external compression of the pelvic veins due to other causes (e.g., tumors).

15. What tests can be used to confirm the diagnosis of PE?

The gold standard is the pulmonary angiogram. An intraluminal filling defect in the contrast-filled pulmonary arteries is considered diagnostic of PE. Ventilation-perfusion scintigraphy (V/Q scan) is a radioisotope lung scan. Technetium-labeled albumin macroaggregates or microspheres are injected intravenously. If perfusion is normal, no emboli are present. An abnormal perfusion scan may indicate PE or diffuse destructive airway disease, such as chronic bronchitis, infection, or areas of emphysema. Multiple perfusion defects are suspicious for PE. To perform a ventilation scan, the patient inhales krypton-81m. In PE the perfusion scan is abnormal, but the ventilation scan is normal.

Two new tests are magnetic resonance angiography (MRA) and contrast-enhanced spiral computed tomography (CT). MRA has a sensitivity of 75–100% and a specificity of 95–100%. Spiral CT has a sensitivity of 72% and a specificity of 95%; it is able to detect emboli as distal as the lobar or segmental arteries.

16. What nonspecific tests help to evaluate patients with possible PE?

An electrocardiogram (ECG) is used to identify right ventricular strain and to rule out myocardial infarction. Chest films are rarely helpful in diagnosing PE but help to rule out processes such as pneumonia and pneumothorax. PE sometimes causes distal hemorrhage, atelectasis, parenchymal infiltrates, and pleural effusions. If PE is present, the chest film may show a moderate increase in heart size, localized areas of underperfused lung, and increased density of the main pulmonary artery associated with peripheral cut-off vessels. These findings cannot be seen on a portable film.

Arterial blood gas analysis is helpful in documenting hypoxemia.

17. Why is it difficult to diagnose PE?

It is difficult to diagnose PE because most patients are too unstable to undergo pulmonary angiography, the most sensitive test, and because the V/Q scan is reported only in probabilities.

18. How accurate are V/Q scans?

The V/Q scan is less sensitive and specific, and requires some patient cooperation. Its probability score (high, intermediate, and low) is based on the presence of perfusion defects and the lack of ventilation defects. A high-probability scan correctly predicts the presence of PE in 86% of cases. However, PE is also present in 30–34% of cases with intermediate probability and 14–31%
Deep Vein Thrombosis and Pulmonary Embolism

of patients with low probability. The most reliable indication of a PE is a match between the probability of the V/Q scan and the probability of the clinical setting (history and physical findings). If the clinical setting and V/Q scan are highly probable for PE, the patient should be aggressively treated for PE. If the clinical probability of a PE does not match the V/Q scan results, the diagnosis of PE is correct in about 50% of cases.

19. What therapies are available to treat DVT in critically ill patients?
   The goals of therapy for acute proximal DVT are to prevent propagation of the thrombus, to limit leg edema, and to reduce symptomatic recurrence of DVT or PE. Therapies to limit thrombus propagation include anticoagulation, thrombolytic therapy, and surgical embolectomy. An infusion of weight-adjusted unfractionated heparin or LMWH is started immediately and continued until warfarin therapy has increased the international normalized ratio (INR) to 2–3 (usually 5 days). Leg elevation and bedrest are recommended to decrease edema and leg pain. Continued anticoagulation and inferior vena caval (IVC) interruption (e.g., IVC filter) are used to prevent recurrence of thromboembolism.

20. What therapies are available to treat PE in critically ill patients?
   The goals of therapy for patients with PE are hemodynamic stability, oxygenation, and prevention of recurrent PE. Therapies include anticoagulation, thrombolytic therapy, surgical or catheter embolectomy, oxygen, intubation and mechanical ventilation if needed, vasoactive medications, and consideration of vena caval interruption.

21. Is the INR or prothrombin time (PT) more accurate in determining therapeutic range for oral anticoagulation therapy?
   The INR is a more sensitive and consistent representation of coagulation status. PT measures the effects of oral anticoagulants on the blood’s ability to clot. When a blood sample is drawn for PT, the laboratory personnel add a specific amount of calcium to the blood, spin the blood down, and then add a thromboplastin reagent to the serum. Because thromboplastin reagents vary widely from manufacturer to manufacturer, an international sensitivity index (ISI) has been developed for each reagent. The laboratory compares the PT time to the ISI for the specific reagent and reports the results as an INR. The INR is relatively consistent between laboratories and allows the same patient’s blood to be tested at more than one laboratory. New point-of-care testing machines are programmed with the ISI score of the reagent and automatically calculate the INR for each test.

22. Why do some patients require a higher dose of heparin to reach therapeutic range?
   The anticoagulation effect of heparin is affected by many factors. Heparin is a naturally occurring glycosaminoglycan, consisting of alternating residues of uronic acid and glucosamine that are variably sulfated. The sulfation of the residues is a major determinant of the anticoagulation activity of a given heparin preparation. Not all preparations have the same anticoagulant activity. Heparin catalyzes antithrombin III to inactivate prothrombin and activated factor X (fXa). In the presence of a large thrombus, more heparin may be needed to achieve effective anticoagulation (partial thromboplastin time 1.5 × normal) because of the large amount of thrombin in the clot. Body weight, hereditary resistance to anticoagulants, and amount of heparin binding to plasma and endothelial cell proteins also affect heparin dosing.

23. What are some medications that interfere with the effectiveness of warfarin?
   
   **Commonly Used Medications that Influence the Anticoagulant Effect of Warfarin**

<table>
<thead>
<tr>
<th>INCREASE EFFECT</th>
<th>DECREASE EFFECT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allopurinol</td>
<td>Barbiturates</td>
</tr>
<tr>
<td>Antibiotics</td>
<td>Carbamazepine</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>Rifampin</td>
</tr>
<tr>
<td>Aspirin products</td>
<td>Antacids</td>
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   (From Schell: Critical Care Nursing Secrets, Philadelphia, 2001, Hanley & Belfus)
Commonly Used Medications that Influence the Anticoagulant Effect of Warfarin (Continued)

<table>
<thead>
<tr>
<th>INCREASE EFFECT</th>
<th>DECREASE EFFECT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Influenza virus vaccine</td>
<td>Sucralfate</td>
</tr>
<tr>
<td>Isoniazid</td>
<td>Colestipol</td>
</tr>
<tr>
<td>Phenytoin</td>
<td>Estrogens</td>
</tr>
<tr>
<td>Phenylbutazone</td>
<td>Adrenal corticosteroids</td>
</tr>
<tr>
<td>Thrombolytics</td>
<td>Spironolactone</td>
</tr>
<tr>
<td>Tolbutamide</td>
<td>Vitamin K</td>
</tr>
<tr>
<td>Vitamin E</td>
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</tbody>
</table>

24. What herbal remedies interfere with the effectiveness of warfarin?
Herbal remedies that have bioflavonoids may interfere with platelet aggregation and increase the patient’s risk of bleeding. Examples include feverfew, gingko biloba, grape seed extract, bilberry, ginger, garlic, and nettle leaves. In one case report, ginseng may have increased the patient’s hypercoagulability by decreasing the INR.

25. What medical conditions influence the effectiveness of warfarin
Patients with the following conditions are more sensitive to warfarin: cachexia, malabsorption syndromes, malnutrition, cancer, collagen diseases, congestive heart failure, diarrhea, fever, hepatic disorders, hyperthyroidism, pancreatic disorders, radiation therapy, renal insufficiency, thyrotoxicosis, and vitamin K deficiency.

Patients with the following conditions may require higher doses of warfarin to reach therapeutic levels: diabetes mellitus, edema, hereditary resistance to anticoagulants, hypercholesterolemia, hyperlipidemia, hypothyroidism, visceral carcinoma, excessive intake of vitamin K, and nephrotic syndrome.

26. What parameters should the nurse monitor to detect potential complications of anticoagulant therapy?
The most common complication of anticoagulation therapy is bleeding. The nurse should monitor and report bleeding from the gums, nose, urinary tract, invasive line sites, or surgical wounds as well as easy bruising and prolonged menses. Pain and swelling in a joint can also be a sign of bleeding. Other parameters to measure include signs of recurrent thrombotic events, thrombocytopenia, and warfarin-induced tissue necrosis. Heparin causes a slight fall in platelet count in about 25% of people, with gradual recovery despite continuation of therapy.

27. What is heparin-induced thrombocytopenia (HIT)?
HIT occurs in about 3% of patients receiving heparin therapy. It is a serious immunoglobulin-mediated response to heparin that may cause life- and limb-threatening thrombotic complications. HIT usually occurs 5–8 days after heparin has been started. Although LMWH is significantly less likely to cause HIT, platelet count should be measured every 2–3 days during therapy.

28. What patients are candidates for thrombolytic therapy?
Catheter-directed thrombolytic therapy is presently indicated for patients with acute leg pain and edema (duration < 14 days) caused by an iliofemoral DVT. Others who may benefit include young patients with femoropopliteal vein thrombi, patients with phlegmasia cerulea dolens (venous gangrene), and patients with extensive superior vena caval, subclavian, or axillary vein thrombosis. Thrombolytic therapy should not be used for patients with a short life expectancy, low risk of ambulatory venous hypertension (inability to walk), coagulopathies, or contraindications to lytic therapy. Thrombolytic therapy also is indicated for patients with a massive PE causing right ventricular dysfunction.

29. What patients are candidates for an IVC filter?
The most common indications for IVC interruption are recurrent thromboembolism despite adequate anticoagulation, complications of anticoagulation (e.g., significant bleeding), and

(From Schell: Critical Care Nursing Secrets, Philadelphia, 2001, Hanley & Belfus)
contraindications to anticoagulation. Less agreed upon indications are presence of a free-floating
tail of an IVC or iliac vein thrombosis, thromboembolism in a pregnant woman with or without
heparin therapy, and preparation for pulmonary embolectomy.

30. What special nursing considerations apply to patients with an intravenous filter?
Immediately after filter placement, patients should be monitored for signs and symptoms of
air embolism, bleeding from the insertion site, signs of retroperitoneal bleeding from accidental
perforation of the inferior vena cava, contrast dye-related renal failure, sepsis, and hemodynamic
compromise from migration of the filter. Patients also must be monitored closely for recurrent
DVT, because the presence of the filter increases the risk for DVT. Over time the filter may de-
crease venous return to the right side of the heart, causing edema below the level of the filter. As
the filter fills with clot and debris, superficial veins of the abdomen dilate. Enlargement of super-
ficial veins increases the risk that emboli may travel around the filter through the superficial
veins and reach the lungs. Most patients with IVC filters are continued on anticoagulation ther-
apy and must be monitored for all associated complications.

31. What is the role of LMWH in the prevention and treatment of DVT?
LMWH may replace unfractionated heparin as the anticoagulant of choice for both prevent-
tion and treatment of DVT. LMWH safely treats DVT in inpatient and outpatient settings. It has
several advantages over unfractionated heparin, including a greater affinity for anti-factor Xa and
a longer half-life. It is not affected by heparin-binding proteins in the blood. Because the effects
of LMWH are highly predictable, it is dosed on a per kilogram schedule and does not need to be
followed with blood tests for partial thromboplastin time.

32. What are the special nursing considerations in caring for patients with DVT or PE?
Patients with DVT or PE need detailed instructions about medical therapy, how to administer
their medications, signs and symptoms of bleeding or rethrombosis, and the importance of regu-
lar follow-up and laboratory testing. They also need emotional support to cope with an acute ill-
ness that may have chronic sequelae. Patients usually are encouraged to shave with a safety or
electric razor to prevent skin nicks. Dental care is important. Patients are encouraged to brush
and floss gently to prevent bleeding. If gum disease is a problem, the patient is encouraged to dis-
cuss possible treatments with a dentist. During the acute phase of care, health care providers try
to minimize the risk of bleeding by minimizing invasive procedures and monitoring the patient
closely for signs of bleeding. Patients with a DVT are measured for knee or thigh-high graduated
compression stockings before ambulation but after leg swelling has decreased. They also are en-
couraged to keep the affected extremity elevated as much as possible to reduce swelling. Warm
moist compresses occasionally are used to increase comfort and reduce leg swelling. Care must
be taken to prevent skin maceration due to moisture.

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(From Schell: Critical Care Nursing Secrets, Philadelphia, 2001, Hanley & Belfus)
1. Define acute coronary syndrome.

Acute coronary syndrome describes a dynamic spectrum of clinical conditions that begin with the rupture of an atherosclerotic plaque, platelet deposition, and thrombus formation. Arterial injury, extent and type of thrombus, and duration of ischemia determine the clinical presentation. These factors underlie the diagnosis of unstable angina, non–Q-wave, or Q-wave myocardial infarction.

2. What is the difference between non–Q-wave and Q-wave infarctions?

Development of a Q wave requires transmural death of the muscle, which permanently alters electrical conduction on the ECG. With subendocardial injury, electrical conduction is not permanently altered, and a Q wave does not develop; hence the term non–Q-wave infarct.

3. How are the acute coronary syndromes differentiated?

<table>
<thead>
<tr>
<th></th>
<th>UNSTABLE ANGINA</th>
<th>NON–Q-WAVE AMI</th>
<th>Q-WAVE AMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonspecific ST segment changes or ST depression</td>
<td>Yes</td>
<td>Yes</td>
<td>Maybe</td>
</tr>
<tr>
<td>ST segment elevation</td>
<td>No</td>
<td>Maybe</td>
<td>Yes</td>
</tr>
<tr>
<td>Development of Q waves after infarction</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Elevation of cardiac injury markers (CK-MB enzyme, troponin T or I)</td>
<td>No</td>
<td>Lower elevations</td>
<td>Higher elevations</td>
</tr>
<tr>
<td>LV function</td>
<td>Return to normal</td>
<td>Less damage</td>
<td>More damage</td>
</tr>
<tr>
<td>Complications</td>
<td>Few</td>
<td>Low</td>
<td>High</td>
</tr>
</tbody>
</table>

AMI = acute myocardial infarction, CK-MB = creatine kinase, myocardial-bound; LV = left ventricular.

4. What are the diagnostic criteria for acute myocardial infarction (AMI)?

At least two of the following criteria must be present:
1. History of ischemic-type chest discomfort
2. Change on serial ECG tracings
   • ST-segment elevation of 0.1 mV in two or more contiguous leads
   • Presence of new left bundle-branch block
3. Rise and fall in serum markers of cardiac injury

(From Schell: Critical Care Nursing Secrets, Philadelphia, 2001, Hanley & Belfus)
5. When do serum markers of cardiac injury begin to rise? When do they peak? For how long are they elevated?

<table>
<thead>
<tr>
<th>SERUM MARKER</th>
<th>POSITIVE LEVEL</th>
<th>BEGINS TO RISE (HR)</th>
<th>PEAK (HR)*</th>
<th>DURATION (DAYS)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>CK-MB</td>
<td>0–5 µg/L</td>
<td>4–6</td>
<td>10–24</td>
<td>2–3</td>
</tr>
<tr>
<td>Troponin T</td>
<td>0.1–0.2 µg/L</td>
<td>3–5</td>
<td>10–24</td>
<td>5–14</td>
</tr>
<tr>
<td>Troponin I</td>
<td>1.5–3.1 µg/L</td>
<td>3–5</td>
<td>14–18</td>
<td>5–9</td>
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<tr>
<td>Myoglobin</td>
<td>Serum levels double with q 2 hr serial samples</td>
<td>2–3</td>
<td>3–15</td>
<td>0.5–1</td>
</tr>
</tbody>
</table>

CK-MB = creatine kinase, myocardial-bound.
* Lack of standardized upper reference levels account for variations in values.

6. What are the current recommendations for emergency department (ED) treatment of patients with AMI?

- Relief of pain
- Supplemental oxygen (keep oxygen saturation > 90%)
- Assessment of hemodynamic stability
- Assessment of ECG electrical stability
- Identification of reperfusion candidates and rapid delivery of therapy
- Intravenous (IV) thrombolytic therapy started within 30 minutes from arrival at ED
- Percutaneous transluminal coronary angioplasty (PTCA) started within 60 minutes from arrival at ED
- Administration of aspirin, beta blockers, and glycoprotein IIb/IIIa inhibitors (unless contraindicated)
- Administration of IV nitroglycerin in patients with congestive heart failure (CHF), hypertension, persistent pain, or large anterior infarction
- Additional drugs to treat arrhythmias and hypotension (if present)

7. What early general measures are recommended for hospital care of patients with AMI?

- Selective ECG monitoring based on infarct location and rhythm
- Relief of pain
- Bedrest with bedside commode for initial 12 hours, and longer if the patient is hemodynamically unstable or pain persists
- Identification and management of high-risk patients
- Pulmonary artery catheter for severe or progressive CHF, shock, hypotension, or suspected mechanical complication (e.g., ventricular septal defect [VSD], mitral regurgitation [MR])
- Intraarterial pressure monitoring for severe hypotension (systolic blood pressure < 80 mmHg) or cardiogenic shock
- Intraaortic balloon counterpulsation for cardiogenic shock refractory to pharmacologic therapy, MR, VSD, incessant ventricular tachycardia, or bridge to PTCA or coronary artery bypass grafting (CABG)
- Arrhythmia management

8. What drugs may be used during hospital care of patients with AMI?

1. Continue beta blockers, glycoprotein IIb/IIIa inhibitors, and aspirin.
2. Angiotensin-converting enzyme (ACE) inhibitors within the first 24 hours in selected patients and before discharge for all patients with AMI and left ventricular ejection fractions < 40%.
3. IV heparin for patients receiving tissue plasminogen activator (Alteplase).
4. Unfractionated heparin or low-molecular-weight heparin for patients not treated with thrombolytic therapy and patients with non-Q-wave MI.

(From Schell: Critical Care Nursing Secrets, Philadelphia, 2001, Hanley & Belfus)
9. How is the patient with AMI prepared for discharge?
   • Exercise, vasodilator stress nuclear scintigraphy, or exercise echocardiography
   • Invasive testing for patients with persistent pain or hemodynamic instability
   • Management of lipids
   • In-hospital cardiac rehabilitation

10. How are thrombolytic drugs best administered?
    The primary intravenous thrombolytic agents are streptokinase (SK), tissue plasminogen activator (tPA), and anisoylated plasminogen streptokinase activator complex (APSAC). Thrombolytics are augmented by heparin and dosed as follows.

    | DRUG       | BOLUS | INFUSION          | TIME       | HEPARIN                        | INFUSION    |
    |------------|-------|-------------------|------------|--------------------------------|-------------|
    | SK         | None  | 1.5 million U     | 60 min     | 6 hr after SK/APSAC and when   | 1000 U/hr for 48 hr |
    | APSAC      | 30 mg | 5 min             | As above   |                                | As above    |
    | tPA        | 15 mg | 0.75 mg/kg up to 50 mg; then 0.5 mg/kg up to 35 mg | 30 min, 60 min | Maximum of 1000 U/hr           | Maximum of 1000 U/hr |

    SK = streptokinase, APSAC = anisoylated plasminogen streptokinase activator complex, tPA = tissue plasminogen activator, aPTT = activated partial thromboplastin time.

11. What are the most common complications of AMI? How are they identified?

    | COMPLICATION                        | METHOD OF IDENTIFICATION                                      |
    |-------------------------------------|----------------------------------------------------------------|
    | Postinfarction angina               | Ongoing monitoring of ST segment of culprit lesion             |
    | Arrhythmias                         | Monitor in lead with diagnostic specificity:                  |
    |                                     | • Leads V₁ and V₆ help to discriminate supraventricular from ventricular beats |
    |                                     | • Lead II is useful in amplifying atrial contribution          |
    | Left ventricular failure            | Monitor for early signs of heart failure:                     |
    | (size and intensity of infarct are predictive of development of heart failure) | • Increased heart rate                                         |
    |                                     | • Reduced blood pressure                                      |
    |                                     | • Extra heart sounds (S₃)                                      |
    |                                     | • Shortness of breath                                         |
    |                                     | • Decreased urine output                                      |
    | Rupture of intraventricular septum (e.g., VSD) | Development of new holosystolic murmur at left sternal border |
    |                                     | Pulmonary artery (PA) mixed venous saturation (SvO₂) is elevated by mixture of blood in left and right ventricles |
    | Rupture of papillary muscle         | Development of new holosystolic murmur starting at apex and radiating to axilla |
    |                                     | No increase in SvO₂                                            |
    | Pericarditis (inflammation of pericardial sac) | Monitor for localized chest pain (may be affected by position) and friction rub Usually manifests 2–4 days after AMI |

    BUN = blood urea nitrogen, ACE = angiotensin-converting enzyme, VSD = ventricular septal defect, IABP = intraaortic balloon pump.

12. What nursing interventions are appropriate for rupture of the intraventricular septum or papillary muscle?
    • Prepare for measures to reduce left ventricular afterload and facilitate forward flow (e.g., intraaortic balloon pump, vasodilators, inotropic support).
    • Prepare the patient for surgery.

(From Schell: Critical Care Nursing Secrets, Philadelphia, 2001, Hanley & Belfus)
BIBLIOGRAPHY


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