Percutaneous coronary intervention (PCI) has reduced morbidity and mortality from cardiovascular disease. In 2009, an estimated 596,000 PCI procedures were performed. Major advances in PCI have included increasingly complex antiplatelet and antithrombotic regimens used in conjunction with PCI. Unfortunately, although these advances yield benefits, they also contribute to the occurrence of femoral vascular access site complications (VASCs), including hematoma, retroperitoneal hemorrhage, pseudoaneurysm, arterial occlusion, and arteriovenous fistula, which in turn are associated with increased morbidity, mortality, and costs. Risk factors predisposing patients to these complications are both modifiable (procedure technique, medications, hemostasis method) and nonmodifiable (sex, age, body mass index, blood pressure, renal function). Patients’ risks can be reduced by nurses who are knowledgeable about these risk factors and identify complications before they become problematic. (Critical Care Nurse. 2012;32[5]:16-30)

The reported incidence of VASCs during PCI is from 5.4% to 20%, depending on the definition and criteria used. VASCs remain an important source of increased morbidity, mortality, length of stay, and cost. The economic ramifications of VASCs are significant. Jacobson et al reported that the cost of PCI when bleeding complications arose was more than double the...
costs of uncomplicated PCI ($25,371 vs $12,279).\textsuperscript{4} Interventions aimed at reducing the risk of adverse events are likely to improve both financial and clinical outcomes.

Removing femoral sheaths and managing related complications after PCI are predominantly the responsibilities of nurses in many acute and critical care settings.\textsuperscript{3,12,13} Therefore, it is essential for nurses to understand the causes of and predisposing risk factors for VASCs. These risk factors can be categorized as modifiable and nonmodifiable.

**Modifiable Risk Factors**

The primary modifiable risk factors for VASCs are femoral access; medications administered before, during, and after the procedure; and hemostasis method. Although the interventional cardiologist controls the femoral access and medications ordered, the hemostasis method is controlled by the nurse unless a vascular closure device is deployed.

**Femoral Access**

Percutaneous entry through the femoral artery and vein approach for PCI is preferred because of the large diameter of those vessels,\textsuperscript{14} which improves the speed and simplicity of the procedure.\textsuperscript{15} VASCs at the femoral site are often associated with the location of the femoral puncture,\textsuperscript{7,15,16} the number of attempts,\textsuperscript{2,16} and catheter size\textsuperscript{17} (Table 1). To facilitate vessel entry and effective compression, the puncture should be above the femoral bifurcation but 1 or 2 cm below the inguinal ligament,\textsuperscript{7,8,15,19} which extends from the anterior superior iliac spine to the pubic tubercle (Figure 1). Many major and potentially lethal VASCs are related to punctures either too high or too low below the inguinal ligament.\textsuperscript{4} Table 2 describes the clinical findings of VASCs and the associated management.

\begin{table}
\centering
\caption{Femoral puncture location and associated complications\textsuperscript{a}}
\begin{tabular}{|l|l|}
\hline
Femoral puncture location: definition & Complications \\
\hline
Low stick: puncture below the femoral bifurcation & Pseudoaneurysm \\
& Hematoma \\
& Arteriovenous fistula \\
\hline
High stick: puncturing the inferior epigastric artery & Retroperitoneal hemorrhage \\
\hline
Posterior wall puncture: puncture through the back wall of the artery & Retroperitoneal hemorrhage \\
\hline
\end{tabular}
\textsuperscript{a} Based on data from Turi,\textsuperscript{7} Ragosta,\textsuperscript{8} Baim and Simon,\textsuperscript{15} Kamineni and Butman,\textsuperscript{18} and Rashid and Bailey.\textsuperscript{19}

\end{table}

\begin{figure}
\centering
\caption{Anatomical landmarks in relation to the femoral vessels.}
\end{figure}
<table>
<thead>
<tr>
<th>Complication</th>
<th>Description</th>
<th>Clinical findings</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hematoma</td>
<td>The most common vascular access site complication</td>
<td>Swelling surrounding the puncture site (visible)</td>
<td>Apply pressure to site 11</td>
</tr>
<tr>
<td></td>
<td>A collection of blood located in the soft tissue</td>
<td>Area of hardening under the skin surrounding the puncture site (palpable)</td>
<td>Mark the area to evaluate for any change in size 15</td>
</tr>
<tr>
<td></td>
<td>Occurs because of blood loss at the arterial and/or venous access site</td>
<td>Varies in size</td>
<td>Provide hydration 11</td>
</tr>
<tr>
<td></td>
<td>or perforation of an artery or vein</td>
<td>Often associated with pain in the groin area that can occur at rest or with leg</td>
<td>Monitor serial complete blood cell counts 21</td>
</tr>
<tr>
<td></td>
<td>May occur if the arterial puncture is below the femoral bifurcation</td>
<td>movement 22</td>
<td>Maintain/prolong bed rest 21</td>
</tr>
<tr>
<td></td>
<td>so the femoral head is not available to assist with compression 6,15,18</td>
<td>Can result in decrease in hemoglobin and blood pressure and increase in heart rate, depending on severity 21</td>
<td>Interrupt anticoagulant and antiplatelet medications if necessary 21</td>
</tr>
<tr>
<td></td>
<td>May occur if the arterial puncture is below the femoral bifurcation</td>
<td></td>
<td>Blood transfusion, if indicated 21</td>
</tr>
<tr>
<td></td>
<td>so the femoral head is not available to assist with compression 6,15,18</td>
<td></td>
<td>If severe, may require surgical evacuation 20</td>
</tr>
<tr>
<td></td>
<td>Can be fatal if not recognized early 21</td>
<td></td>
<td>Many hematomas resolve within a few weeks as the blood dissipates and is absorbed into the tissue 21</td>
</tr>
<tr>
<td>Retroperitoneal</td>
<td>Bleeding that occurs behind the serous membrane lining the walls of the abdomen/pelvis 21</td>
<td>Moderate to severe back pain 21</td>
<td>Provide hydration 21</td>
</tr>
<tr>
<td>hemorrhage</td>
<td>May occur if the arterial wall puncture is made above the inguinal ligament, resulting in perforation of a suprainguinal artery 21 or penetration of the posterior wall 7,8,15</td>
<td>Ipsilateral flank pain 21</td>
<td>Perform serial blood cell counts 21</td>
</tr>
<tr>
<td></td>
<td>Can be fatal if not recognized early 21</td>
<td>Vague abdominal or back pain 21</td>
<td>Maintain/prolong bed rest 21</td>
</tr>
<tr>
<td></td>
<td>May occur if the arterial wall puncture is made above the inguinal ligament, resulting in perforation of a suprainguinal artery 21 or penetration of the posterior wall 7,8,15</td>
<td>Ecchymosis and decrease in hemoglobin and hematocrit are late signs 7,21</td>
<td>Interrupt anticoagulant and antiplatelet medications if necessary 21</td>
</tr>
<tr>
<td></td>
<td>Can be fatal if not recognized early 21</td>
<td>Abdominal distention 7,21</td>
<td>Blood transfusion, if indicated 21</td>
</tr>
<tr>
<td></td>
<td>May occur if the arterial puncture is below the femoral bifurcation</td>
<td>Often not associated with obvious swelling 21</td>
<td>If severe, may require surgical evacuation 20</td>
</tr>
<tr>
<td></td>
<td>so the femoral head is not available to assist with compression 6,15,18</td>
<td>Hypotension and tachycardia 21</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Can be fatal if not recognized early 21</td>
<td>Diagnosed by computed tomography 21</td>
<td></td>
</tr>
<tr>
<td>Pseudoaneurysm</td>
<td>A communicating tract between the tissue and, usually, one of the weaker walls of the femoral artery, causing blood to escape from the artery into the surrounding tissue 21</td>
<td>Swelling at insertion site 22</td>
<td>Maintain/prolong bed rest 21</td>
</tr>
<tr>
<td></td>
<td>Possible causes include difficulty with arterial cannulation, inadequate compression after sheath removal, and impaired hemostasis 21</td>
<td>Large, painful hematoma 21</td>
<td>Small femoral pseudoaneurysms should be monitored; they commonly close spontaneously after cessation of anticoagulant therapy 21</td>
</tr>
<tr>
<td></td>
<td>May occur if the arterial puncture is below the femoral bifurcation</td>
<td>Ecchymosis 21</td>
<td>Large femoral pseudoaneurysms can be treated by ultrasound-guided compression, surgical intervention, or ultrasound-guided thrombin injection 22</td>
</tr>
<tr>
<td></td>
<td>so the femoral head is not available to assist with compression 6,15,18</td>
<td>Pulsatile mass 21</td>
<td></td>
</tr>
<tr>
<td></td>
<td>May occur if the arterial puncture is below the femoral bifurcation</td>
<td>Bruit and/or thrill in the groin 21</td>
<td></td>
</tr>
<tr>
<td></td>
<td>so the femoral head is not available to assist with compression 6,15,18</td>
<td>Pseudoaneurysms can rupture, causing abrupt swelling and severe pain 21</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Can be fatal if not recognized early 21</td>
<td>Suspect nerve compression when pain is out of proportion to size of hematoma 21</td>
<td></td>
</tr>
<tr>
<td></td>
<td>May occur if the arterial puncture is below the femoral bifurcation</td>
<td>Nerve compression can result in limb weakness that takes weeks or months to resolve 21</td>
<td></td>
</tr>
<tr>
<td></td>
<td>so the femoral head is not available to assist with compression 6,15,18</td>
<td>Diagnosed by ultrasound 21</td>
<td></td>
</tr>
<tr>
<td>Arteriovenous fistula</td>
<td>A direct communication between an artery and a vein that occurs when the artery and vein are punctured 22</td>
<td>Can be asymptomatic 24</td>
<td>Some arteriovenous fistulas resolve spontaneously without intervention 27</td>
</tr>
<tr>
<td></td>
<td>The communication occurs once the sheath is removed 22</td>
<td>Bruit and/or thrill at access site 29</td>
<td>Some arteriovenous fistulas require ultrasound-guided compression or surgical repair 20</td>
</tr>
<tr>
<td></td>
<td>Risk factors: Multiple access attempts’ Punctures above or below proper site level 21</td>
<td>Swollen, tender extremity 22</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Impaired clotting 21</td>
<td>Distal arterial insufficiency and/or deep venous thrombosis can result in limb ischemia 21</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Congestive heart failure 20</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Confirmed by ultrasound 21</td>
<td></td>
</tr>
</tbody>
</table>

Continued
High sticks are significantly linked with retroperitoneal hemorrhage resulting from the likelihood of puncturing the inferior epigastric artery. However, punctures below the proper access points do not eliminate the risk of retroperitoneal hemorrhage; penetration of the posterior wall of the artery during femoral puncture can also cause retroperitoneal bleeding (Table 1). 

Low sticks can predispose patients to pseudoaneurysm, hematoma, and arteriovenous fistula. When the groin is accessed at or below the level of the femoral bifurcation, the femoral sheath is put into vessels that are smaller than the common femoral artery. Depending on the size of the sheath used, these vessels may not be large enough to accommodate the sheath. As a result, access below the femoral bifurcation is more likely to lead to a VASC. When the groin site is accessed under optimal conditions, the femoral head can be used after sheath removal to achieve effective compression of the site and prevent bleeding complications. With low sticks, the femoral head is not available to assist with compression. Instead, pressure is placed against soft tissue, making effective hemostasis less probable. This can predispose patients to hematoma and pseudoaneurysm. Finally, low sticks are near the bifurcation vessels to other blood vessels. Various vein branches that run along or anterior to the bifurcation may be accessed during arterial puncture, resulting in an arteriovenous fistula.

Repeat or multiple punctures of the artery increase the likelihood that another artery or vein will be punctured, causing the development of VASCs. Increased sheath size increases the risk for vascular trauma and VASCs. Grossman and colleagues found that PCIs performed with 7F and 8F guides compared with 6F guides were associated with more use of contrast medium, renal complications, bleeding, VASCs,

<table>
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<th>Management</th>
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<tr>
<td>Arterial occlusion</td>
<td>Occlusion of an artery by a thromboembolism</td>
<td>Classic symptoms include the 5 Ps: Pain, Paralysis, Parasthesias, Pulselessness, Pallor, Doppler studies help localize the area</td>
<td>Treatment depends on size/type of embolus, location, and patient’s ability to tolerate ischemia in affected area</td>
</tr>
<tr>
<td>Incidence: &lt;0.8%</td>
<td>Most common sources: mural thrombus originating in cardiac chambers, vascular aneurysms, or vascular atherosclerotic plaques</td>
<td>Angiogram is required to identify exact location of occlusion site</td>
<td>Small thromboemboli in well-perfused arterial areas may undergo spontaneous lysis</td>
</tr>
<tr>
<td></td>
<td>Thromboemboli can develop at sheath site or catheter tip; embolization occurs during sheath removal</td>
<td></td>
<td>Larger thromboemboli may require thromboembolectomy, surgery, and/or thrombolytic agents</td>
</tr>
<tr>
<td></td>
<td>Prevention or at least reduction can be obtained by anticoagulation, vasodilators, and nursing vigilance</td>
<td></td>
<td>Distal embolic protection devices (ie, filters) may be placed if necessary</td>
</tr>
<tr>
<td>Femoral neuropathy</td>
<td>Nerve damage caused by injury of the femoral nerve(s) during access and/or compression of nerves by a hematoma</td>
<td>Pain and/or tingling at femoral access site, Numbness at access site or further down the leg, Leg weakness, Difficulty moving affected leg, Decreased patellar tendon reflex</td>
<td>Identification and treatment of the source, Treatment of symptoms, Physical therapy</td>
</tr>
<tr>
<td>Incidence: 0.21%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infection</td>
<td>Colonization by a pathogen Causes: Compromised technique, Poor hygiene, Prolonged indwelling sheath time, Femoral access closure device (closure devices have been linked with increased occurrence of infection)</td>
<td>Pain, erythema, swelling at access site, Purulent drainage at access site, Fever, Increased white blood cell count</td>
<td>Treatment of symptoms (eg, pain), Antibiotics</td>
</tr>
<tr>
<td>Incidence: &lt;0.1%</td>
<td></td>
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CriticalCareNurse Vol 32, No. 5, OCTOBER 2012 19
transfusions, major adverse cardiac events, and deaths.

Medications

As techniques for performing PCI have progressed through the years, so has the approach to anticoagulation before, during, and after the procedure. Combinations of oral and intravenous antiplatelet and antithrombin therapy are universally used for patients with acute coronary syndrome, including unstable angina and NSTEMI, who are undergoing PCI. These agents (shown in Table 3) are critical in reducing rates of mortality, adverse ischemic events (such as recurrent myocardial infarction), short- and long-term complications of PCI, and other major adverse cardiac events.

Antithrombins inhibit the coagulation factors that act in a complex cascade to form fibrin strands as part of the process of hemostasis. The antithrombins consist of unfractionated heparin (UFH), low molecular weight heparin (LMWH), and direct thrombin inhibitors (DTIs; eg, bivalirudin, argatroban). Antiplatelet agents can prevent the formation of blood clots by inhibiting the activation of platelets. In so doing, these agents prevent blood clotting, usually in high-flow areas of the circulation such as the arterial circulation. They have little effect in inhibiting thrombosis in the venous circulation. The antiplatelet agents include glycoprotein IIb/IIIa inhibitors, adenosine diphosphate inhibitors (eg, clopidogrel and prasugrel), and aspirin. It is the nurse’s responsibility to know the mechanism of action of each medication, verify and double check the type and dosage of medication(s) prescribed, and monitor the patients’ reactions to the medication(s) to reduce VASCs.

Unfractionated Heparin. The anticoagulant effect of UFH is mediated directly by inhibiting thrombin-activated conversion of fibrinogen to fibrin. UFH is used to prevent thrombosis before and during PCI. Heparin binds to thrombin, preventing the conversion of fibrinogen to fibrin. During the PCI, heparin activity is monitored by measuring activated clotting time, with a goal of maintaining times greater than 200 s. Activated clotting time indicates the effectiveness of high-dose heparin therapy by measuring the intrinsic clotting activity of the whole blood. Activated clotting time lacks correlation with results of other coagulation tests and is used to demonstrate the inability to coagulate rather than to quantify the ability to clot.

Low Molecular Weight Heparin. LMWH, like UFH, exerts its anticoagulant activity by activating antithrombin, which plays a role in restricting thrombus formation. Although it consists only of fragments of UFH, it is just as effective and has a half-life 2 to 4 times longer than that of standard heparin. Because LMWH has little effect on measurements of activated clotting time, it should not be used as a guide to anticoagulation therapy. Sheath removal when followed by manual or mechanical compression may be performed 4 hours after the last intravenous dose or 6 to 8 hours after the last subcutaneous dose. LMWH is a safe and effective alternative to unfractionated heparin.

Direct Thrombin Inhibitors. DTIs may be used as an alternative to UFH and LMWH. DTIs exert their effect by interacting directly with the thrombin molecule without the need for a cofactor. Unlike heparin, DTIs do not rely on antithrombin to provide anticoagulation but function by inhibiting thrombin that is bound to fibrin or fibrin degradation products. In addition, unlike heparin, DTIs have an antiplatelet effect.

Their pharmacokinetic profile precludes the need to measure activated clotting times during the procedure or before sheath removal. Additionally, it has been reported that the most commonly used DTI, bivalirudin, is associated with lower frequency of bleeding at the access site (4%) than are UFH or LMWH (plus glycoprotein IIb/IIIa inhibitors; 7%) in patients undergoing PCI (P < .001). DTIs offer many advantages over heparin, including the inhibition of both circulating and clot-bound thrombin; a more predictable anticoagulant response; inhibiting thrombin-induced platelet aggregation; and absence of induction of immune-mediated thrombocytopenia.

Glycoprotein IIb/IIIa Inhibitors. These inhibitors prevent the final pathway of platelet aggregation by attaching to fibrinogen and other proteins, blocking platelet aggregation and preventing thrombosis. Three parenteral agents—abciximab, eptifibatide, and tirofiban—are currently approved for clinical use by the Food and Drug Administration. They are often used in conjunction...
### Table 3: Dosing of anticoagulant and antiplatelet agents used in percutaneous coronary intervention (PCI)

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosing</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anticoagulant agents</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Bivalirudin (Angiomax)      | Loading dose: 0.75 mg/kg intravenous bolus (wait 30 minutes if patient received unfractionated heparin)  
                           Maintenance dose: 1.75 mg/kg per hour infusion during PCI | With addition of 600 mg clopidogrel, can be administered with or without unfractionated heparin for antithrombotic treatment in PCI and acute coronary syndrome24 |
| Enoxaparin                  | Loading dose: 30 mg intravenous bolus  
                           Maintenance dose: 1 mg/kg subcutaneous every 12 hours  
                           If patient received initial anticoagulant therapy <8 hours before PCI: no additional therapy  
                           If last subcutaneous dose >8 hours ago: 0.3 mg/kg intravenous bolus  
                           Creatinine clearance <30 mL/min: 1 mg/kg every 24 hours | No anticoagulation monitoring available27  
                           Risk for heparin-induced thrombocytopenia29  
                           Only partial reversal with administration of protamine sulfate26 |
| Unfractionated heparin      | Loading dose: 70-100 IU/kg intravenous bolus; if target values for activated clotting time are not achieved, administer additional heparin boluses 2000 to 5000 IU  
                           Loading dose on glycoprotein IIb/IIIa inhibitor: 60 U/kg intravenous bolus  
                           Administer for target activated clotting time of 200-250 seconds (target activated clotting time may vary by method of measurement)  
                           Dosing must take into account whether patient received initial medical therapy | Monitor by using activated partial thromboplastin time or activated clotting time at bedside26,29  
                           Reverse with protamine sulfate26  
                           Risk for heparin-induced thrombocytopenia29 |
| **Intravenous antiplatelet agents** |                                                                        |                                                                                                                                                                                                          |
| Abciximab (ReoPro)          | Loading dose: 0.25 mg/kg intravenous bolus  
                           Maintenance dose: 0.125 µg/kg per minute (maximum, 10 µg/min) infusion during percutaneous coronary intervention and for 12 hours after PCI | Administer with aspirin and heparin22  
                           Platelet aggregation returns to normal 24-48 hours after discontinuation25  
                           Monitor activated clotting time, activated partial thromboplastin time, hemoglobin, platelet count when given with heparin24 |
| Eptifibatide (Integrilin)   | Loading dose: 180 µg/kg intravenous bolus  
                           Maintenance dose: 2 µg/kg per minute during PCI; continue 18-24 hours after PCI  
                           Creatinine clearance <50 mL/min: Reduce both loading and maintenance dose infusion rate by 50%24 | Administer with aspirin and heparin11  
                           Platelet aggregation returns to normal 4-8 hours after discontinuation25  
                           Monitor activated clotting time, activated partial thromboplastin time, hemoglobin, platelet count when given with heparin11 |
| Tirofiban (Aggrastat)       | Loading dose: 0.4 µg/kg per minute intravenous for 30 minutes  
                           Maintenance dose: 0.1 µg/kg per minute infusion during percutaneous coronary intervention and continued for 18-24 hours after PCI  
                           Creatinine clearance <30 mL/min: Reduce both loading and maintenance dose infusion rate by 50%24 | Administer with aspirin and heparin12  
                           Platelet aggregation returns to normal 4-8 hours after discontinuation25  
                           Monitor activated clotting time, activated partial thromboplastin time, hemoglobin, platelet count when given with heparin12 |
| **Oral antiplatelet agents** |                                                                        |                                                                                                                                                                                                          |
| Aspirin                     | Daily dose: 162-325 mg daily orally (patients at high risk of bleeding may receive 75-162 mg/d)25,26 | Administer as soon as possible after hospital admission (if patient has not already taken aspirin)25 |
| Clopidogrel (Plavix)        | Loading dose: 300-600 mg orally  
                           Maintenance dose: 75 mg/d, ideally up to 1 year24 | Administer to patients not able to take aspirin because of hypersensitivity or significant gastrointestinal intolerance25  
                           Use in conjunction with aspirin25  
                           Antiplatelet effects are irreversible14  
                           Takes several days to achieve maximum effectiveness without a loading dose24  
                           Variability in patients’ responsiveness to the drug25 |
| Prasugrel                   | Loading dose: 60 mg orally  
                           Maintenance dose: 10 mg daily for ≥12 months after stenting24 | Antiplatelet effects are irreversible29  
                           Little variation noted in patients’ response24 |
with UFH or a DTI. The adjunct use of a glycoprotein IIb/IIIa inhibitor during PCI is effective and associated with improved in-hospital survival rates. However, the optimal timing of initiation of glycoprotein IIb/IIIa inhibitor therapy in patients with unstable angina or NSTEMI (ie, whether to administer therapy before or after PCI) and the optimal application of this therapy have not been determined. The 2011 guidelines on PCI from the American College of Cardiology Foundation/American Heart Association (ACC/AHA) support early administration of glycoprotein IIb/IIIa before catheterization for patients with unstable angina or NSTEMI undergoing PCI who are judged clinically to be at high risk of thrombotic events relative to bleeding risk. The guidelines further note that much of the research evaluating use of these agents for patients with STEMI was performed in the era before routine dual oral antiplatelet therapy and was evaluated largely by placebo-controlled comparisons.

More recently, 3 trials were done to evaluate glycoprotein IIb/IIIa antagonists as adjuncts to oral antiplatelet therapy in patients with primary PCI. The results bring into question whether glycoprotein IIb/IIIa antagonists provide additional benefit to STEMI patients who received dual antiplatelet therapy before catheterization. The ACC/AHA guidelines judge that routine use of glycoprotein IIb/IIIa antagonists for such patients cannot be recommended, although some patients (eg, those with a large thrombus burden or who received inadequate thienopyridine loading) may benefit. Clinical practices with these and other antithrombotic agents continue to evolve.

**Clopidogrel.** This oral antiplatelet agent specifically inhibits the P2Y12 adenosine diphosphate receptor on the platelet surface, preventing activation of the glycoprotein IIb/IIIa receptor complex, thereby reducing platelet aggregation. Platelets blocked by clopidogrel are affected for the remainder of their lifespan (~7-10 days). Clopidogrel reduces the frequency of ischemic complications after PCI and improves postintervention outcomes. 

Clopidogrel resistance (ie, decreased inhibition of platelet function after administration of clopidogrel) may occur in 30% of patients and may relate to factors such as bioavailability, noncompliance, underdosing, lower absorption, drug interference, or single nucleotide polymorphisms. Poor response to oral antiplatelet agents increases the risk of thrombotic events, including myocardial infarction, particularly after coronary angioplasty. The authors of a recent meta-analysis suspect that the cause of clopidogrel resistance is an interaction with glycoprotein IIb/IIIa inhibitors and the use of different cutoffs to identify nonresponders.

**Prasugrel.** This novel third-generation rapid-acting thienopyridine is a more potent blocker of the platelet P2Y12 receptor than is clopidogrel, and no resistance has been reported. The 60-mg loading dose achieves faster, more consistent, and greater inhibition of adenosine diphosphate–induced platelet aggregation than does 600 mg clopidogrel. Prasugrel produces a significantly greater effect than clopidogrel as early as 30 minutes after administration. Prasugrel is superior to clopidogrel in preventing ischemic events in patients with acute coronary syndrome undergoing PCI, even though there is an associated greater risk of bleeding. Prasugrel therapy should be individualized and targeted toward those patients with stents or patients with decreased platelet inhibition by clopidogrel. Patients with a history of cerebrovascular events have experienced significant harm from administration of prasugrel. Prasugrel has a black box warning, not to be used in patients with previous stroke or transient ischemic attack because of its greater tendency to cause intensive inhibition of platelet aggregation in general and the findings of increased levels of bleeding compared with clopidogrel.

**Aspirin.** Aspirin inhibits the cyclooxygenase enzyme, which stops prostaglandin synthesis and release, and inhibits prostaglandin synthetase action, which prevents formation of thromboxane A2, thus inhibiting platelet aggregation. Long-term aspirin therapy is recommended for patients who undergo any revascularization procedure, including PCI.

**Hemostasis Methods**

Currently, 3 main techniques are used to achieve hemostasis at the femoral access site after PCI: manual compression, mechanical compression, and vascular closure devices.

**Manual Compression.** Manual compression has been the “gold standard” for obtaining hemostasis at the vascular access site for years, but this standard has changed as new devices have come on the market.
The artery punctured is superior and medial to the skin puncture site, so pressure is applied as the sheath is removed by placing the index and middle fingers 1 to 2 cm above the site where the sheath enters the skin and applying pressure as the sheath is removed (Figure 2).\(^\text{67}\) Hemostasis is achieved by compressing the femoral artery against the femoral head.

Manual compression for some practitioners is not an option because it requires strength and the ability to hold a good compression for 15 to 20 minutes.\(^\text{6,21}\) If hand and arm fatigue develops during the procedure, the amount of pressure applied to the femoral artery may vary, causing VASCs.\(^\text{3}\)

**Mechanical Compression.** Mechanical compression involves the application of constant pressure on the artery to obtain hemostasis and allows hands-free catheter removal so that nurses can monitor the patient.\(^\text{3}\) There are 2 main types of compression: The C-clamp (CompressAR, Advanced Vascular Dynamics) and pneumatic (FemoStop, Radi Medical Systems AB, St Jude Medical, Inc).

The C-clamp consists of a flat metal plate, placed under the mattress at the patient’s hip to stabilize the device, and a C-clamp arm. A disposable translucent pad is attached to the tip of the C-clamp arm (Figure 3). The FemoStop device uses a small pneumatic clear pressure dome, a belt placed around the patient’s hips, and a pump with a manometer making it possible to adjust pressure to an optimal level (Figure 4). As with manual compression, the translucent pad or clear dome is placed 1 to 2 cm above the site where the sheath enters the skin and pressure is applied by pressing down on the C-clamp arm or adjusting the pressure with the pump. Mechanical compression does not cause hand and/or arm fatigue and is just as effective as manual compression in obtaining hemostasis.\(^\text{3,5,68}\) The translucent pad or clear dome provides easy visualization of the puncture site while the pressure is slowly released. It is important to remember that both manual and mechanical compression can be ineffective in obtaining hemostasis in patients who received low sticks.\(^\text{7}\)

**Vascular Closure Devices.** These devices first appeared in the 1990s as means of reducing time on bed rest and improving both hemostasis and patients’ comfort. A variety of devices seek to mechanically close the arterial puncture site during sheath removal in the catheterization laboratory in fully anticoagulated patients and shorten the time to hemostasis and ambulation.\(^\text{15}\) Three main types of vascular closure devices can be categorized by the mechanism of hemostasis, including
sutures, collagenlike plugs, and staples/clips (Figures 5-7). Suture-mediated closure devices tie off the femoral artery with sutures. Collagen plugs seal the puncture site by stimulating platelet aggregation and the release of coagulation factors, which results in the formation of a clot. Extravascular clips or staples are used to seal off the puncture site in the artery. Hemostasis is usually obtained shortly after deployment, allowing the patient to get out of bed and ambulate faster.

Vascular closure devices, when compared with the mechanical C-clamp and manual compression, all provide low and comparable complication risks following sheath removal in the era of antiplatelet
and antithrombotic therapies. Appropriate selection of patients by the physician is important, and the device should be placed only after confirmation of the vascular anatomy and the absence of significant local peripheral arterial disease. In cases in which vascular closure devices are not effective, manual compression must be applied to accomplish hemostasis.

### Nonmodifiable Risk Factors

Nonmodifiable risk factors for VASC are characteristics of patients that cannot be changed in the PCI setting. These include sex, advanced age, body mass index (BMI), hypertension, and renal dysfunction. Each of these factors alone, and especially in combination, can affect the likelihood that a patient will experience a VASC after a procedure.

#### Sex

An estimated 34% of the almost 600,000 PCIs in the United States annually are performed in women, and being female has been clearly identified as a risk factor for VASCs. Compared with men, women undergoing PCI are older and have a higher incidence of hypertension, diabetes mellitus, hypercholesterolemia, and comorbid disease. A nationwide study of 199,690 patients showed that women presented for PCI with unstable angina and/or NSTEMI more often than men did and had a significantly higher frequency of VASCs. These women were older than their male counterparts, although they had fewer high-risk angiographic features and higher ejection fractions. However, women have been observed to have atypical and sometimes ambiguous symptoms, which may have reached higher acuity by the time they arrive at the cardiac catheterization laboratory, thereby contributing to their level of complications.

#### Advanced Age

Advanced age, generally more than 70 years of age, is directly linked to increased incidence of VASCs. Results of a retrospective study of the incidence, predictors, and prognostic impact of periprocedural bleeding and transfusion in 10,974 patients undergoing PCI indicated that age was among the strongest predictors of major bleeding. It is generally agreed that with increasing age, patients are at increased risk of bleeding complications, possibly related to local vascular changes or more advanced vascular disease.

#### Body Mass Index

Researchers have identified a lower BMI (calculated as weight in kilograms divided by height in meters squared) as a risk factor for vascular complications in several studies. Mehta et al studied 2,325 patients with acute myocardial infarction who received primary PCI and reported that although obese patients (those with BMI ≥30) had more cardiovascular risk factors at baseline, they had fewer VASCs, shorter hospital stays, and fewer deaths in the hospital and at 12 months than did patients with a normal BMI. This difference may have been because the obese patients were a mean of 6 years younger than the patients with normal BMI or because obesity is related to impaired fibrinolysis and increased platelet aggregation. Delhaye et al further examined the role of BMI in records of 16,783 patients who underwent PCI. The patients were grouped according to 6 BMI groups: underweight (BMI <18.5), “normal” weight (BMI, 18.5-24.9), overweight (BMI, 25-29.9), class I obesity (BMI, 30-34.9), class II obesity (BMI, 35-39.9), and class III obesity (BMI ≥40). The incidence of major bleeding varied significantly throughout the BMI spectrum: from underweight (5.6%) to normal-weight (2.5%) to overweight (1.9%) to class I obese (1.6%) to class II obese (2.1%) to class III obese (1.9%) patients ($P < .001$). Compared with normal-weight patients, the risk of major bleeding was higher in overweight patients (odds ratio, 2.29 [95% CI, 1.56-3.38]) and lower in class I obese patients (odds ratio, 0.65 [95% CI, 0.47-0.90]).

#### Hypertension

Hypertension may increase patients’ risk for a VASC developing. In a study of 413 patients undergoing PCI, it was reported that patients with a higher systolic blood pressure (135 vs 129 mm Hg; $df=410$, $P=.02$) were significantly more likely to have complications than were patients with lower blood pressures. In a larger study of 13,819 patients, Manoukian et al found that the 644 patients (4.7%) who experienced major bleeding were more likely to have hypertension than were patients without major bleeding. Although elevated blood pressure during PCI and sheath removal may increase the risk of VASCs, no evidence-based blood pressure guidelines for PCI patients are currently available.

#### Renal Dysfunction

Renal dysfunction, defined as creatinine clearance less than...
60 mL/min,11 has been consistently identified as a major risk factor for bleeding in patients undergoing PCI.11,12,37,74,76,81 The underlying mechanism for such an association has been postulated to be advanced age, as well as the presence of more severe atherosclerosis and multiple comorbid conditions.76 Patients with renal dysfunction who are undergoing PCI are at increased risk of excessive dosing of anticoagulant and antiplatelet medications such as UFH and glycoprotein IIb/IIIa inhibitors, considering that most of these medications are eliminated via the kidneys (Table 3).

**Implications for Nursing**

The main goals of patient care after PCI include maintenance of hemostasis at the puncture site and assessment for VASCs. Achieving these goals requires diligent assessment of patients with frequent monitoring of vital signs, puncture site, and pulse check. Duration of bed rest and time to ambulation depend on the method of arterial closure and the patient’s overall clinical condition. Figure 8 is an assessment worksheet for units or individual nurses to use as a reference when getting report from the catheterization laboratory or assessment checks on the unit. The worksheet assists nurses in determining the patient’s baseline assessment so that any


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**Figure 8** Worksheet to guide nurses in asking the right questions when getting report from the catheterization laboratory.
procedural or medication-related complications can be promptly noted and addressed.

Never before has it been so clinically important to understand the predictors and effect of VASCs in PCI, acute coronary syndrome, and STEMI. Patients are increasingly treated with higher complexity regimens containing greater numbers of more potent oral and intravenous antiplatelet and antithrombin medications for longer periods. These factors can be expected to result in higher rates of VASCs. Critically ill patients admitted to the intensive care unit are at high risk for VASCs because of the presence of comorbid conditions such as renal failure, hypertension, and advanced age. These patients are also more likely to be heavily anticoagulated and to have had a high-risk, technically demanding procedure on an emergency basis.

Importantly, independent nursing judgments regarding the methods for sheath removal and frequency of monitoring should be based on current evidence and knowledge of the risks for complications, given the patient’s characteristics and the circumstances surrounding the PCI procedure. Nurses are in a good position to recognize VASCs when they occur and be knowledgeable about management techniques to resolve them should they become problematic. Understanding that VASCs have both modifiable and nonmodifiable risk factors helps nurses address issues that they can affect while ensuring that at-risk patients receive optimal monitoring and management. A thorough understanding of vascular access issues and prompt recognition of these complications are essential to minimize the substantial morbidity, mortality, and hospital costs associated with them. CCO

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