STANDARDS OF PRACTICE


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ABBREVIATIONS

ADP = adenosine diphosphate, CI = confidence interval, CKD = chronic kidney disease, CLD = chronic liver disease, COX = cyclooxygenase, DAPT = dual antiplatelet therapy, DIC = disseminated intravascular coagulation, DOAC = direct oral anticoagulant, FDA = Food and Drug Administration, 4F-PCC = 4-factor prothrombin complex concentrate, INR = International Normalized Ratio, ITP = immune thrombocytopenia, LMWH = low molecular weight heparin, PT = prothrombin time, PTT = partial thromboplastin time, ROTEM = rotational thromboelastometry, SSRI = selective serotonin reuptake inhibitor, TEG = thromboelastography, UFH = unfractionated heparin, VKA = vitamin K antagonist, VTE = venous thromboembolic disease, VWF = von Willebrand factor

PREAMBLE

In 2012, the Society of Interventional Radiology (SIR) published its first consensus practice guidelines regarding the periprocedural management of coagulation status for percutaneous image-guided interventions (1), which was subsequently revised in 2013 with a discussion of newer anticoagulant agents (2). The present update to both documents incorporates a multidisciplinary approach to the periprocedural management of coagulation status with emphasis on the patient’s clinically relevant comorbidities, and will be divided into 2 parts: part I will review classes of anticoagulation medications and clinical considerations common to patients requiring percutaneous image-guided interventions; part II will discuss recommendations.

METHODOLOGY

The Standards Division of SIR provides evidence-based clinical practice documents to ensure patient safety and enhance the delivery of patient care. Standards Division members are leaders in the field of interventional radiology from the private and academic sectors of medicine who dedicate the vast majority of their professional time to performing interventional...
INTRODUCTION

The management of patients with coagulopathies and patients receiving anticoagulation and antiplatelet therapy undergoing minimally invasive image-guided interventions is complex and evolving. As the United States population ages, the American Heart Association anticipates an increased use of long-term anticoagulation medications to prevent stroke associated with nonvalvular atrial fibrillation and to prevent and treat venous thromboembolic disease (VTE) (8). Thus, it is imperative that interventional radiologists understand the causes and pathophysiology of their patients’ coagulopathies to determine the best course of action in mitigating bleeding and thromboembolic risks in the peri-procedural period. Herein, coagulation physiology, anticoagulant and antiplatelet medications, laboratory testing, and challenges of peri-procedural coagulation management in patients with specific clinical conditions such as cirrhosis, renal failure, and cardiac disease are reviewed.

LABORATORY TESTS USED IN THE EVALUATION OF HEMOSTASIS

The prothrombin time (PT) test assesses the tissue factor (ie, extrinsic) pathway, and the activated partial thromboplastin time (PTT) test assesses the intrinsic pathway. These tests are often used to assess bleeding risk before procedures. Both are also affected by the common pathway factors. These tests were developed to identify the cause of bleeding in symptomatic patients, and mild to moderate prolongation of these laboratory values has not been shown to predict bleeding risk in a nonbleeding patient (11,12).

The International Normalized Ratio (INR) was developed to standardize warfarin monitoring because of variability in tissue thromboplastins used in the PT reagent. INR is calculated as (PTR) INR where PTR = prothrombin time ratio (PT of patient/PT of control) and ISI = international sensitivity index, a value assigned to each PT reagent after calibrating against a WHO standard that has an ISI of 1.0. PTT reagents are even more variable than PT reagents in clinical laboratories, and local standards should be verified.

Other tests include the thrombin time test, fibrinogen assay, and D-dimer assay. D-dimers are produced by the action of plasmin (fibrinolytic system) on a cross-linked fibrin clot and are often used to diagnose disseminated intravascular coagulation (DIC) or to aid in ruling out acute thrombosis. Various coagulopathies, the use of anticoagulant medications, or liver disease can be associated with abnormalities of the results of these routine coagulation tests (Table 1). Therefore, the entire clinical picture must be taken into consideration to understand the patient’s true bleeding risk.

There are several classes of anticoagulant medications, such as low molecular weight heparin (LMWH) and most direct oral anticoagulants (DOACs), that may not cause derangements in activated PT and/or PT/ INR but still increase bleeding risk. Routine laboratory monitoring, with the exception of periodic assessment of renal function, is not required for patients receiving DOACs because these medications have predictable pharmacokinetics and anticoagulant effects (13), and there is no US Food and Drug Administration (FDA)–approved DOAC laboratory assay (14). All DOACs may affect routine coagulation test results, but not in ways that allow for reliable quantitative measurement of the anticoagulation effect (8).

However, the following tests may be used to evaluate for the presence of DOACs: thrombin time, ecarin clotting time (dabigatran), and anti-factor Xa activity (rivaroxaban, apixaban, edoxaban). Most clinical decisions concerning DOACs can be made by knowing the creatinine clearance and time of last drug ingestion (8).

Platelet count is also frequently assessed as part of preprocedural laboratory testing. For normal hemostasis, a platelet count of 5 × 10^11/L is sufficient; however, moderate to severe thrombocytopenia as well as moderate to severe platelet dysfunction have been shown to increase bleeding risk during procedures (10,15). With normally functioning platelets, a platelet count > 50 × 10^11/L is generally sufficient to reduce bleeding risk for most high-risk image-guided interventional procedures (15,16), whereas a platelet count < 20 × 10^11/L is associated with an increased bleeding risk (15).

Although they are not incorporated into routine laboratory testing, thromboelastography (TEG) and rotational thromboelastometry (ROTEM) deserve mention. TEG/ROTEM has been used for “point-of-display” testing during liver transplantation since the 1980s (17) and more recently to differentiate between coagulopathic versus perioperative surgical-associated bleeding to guide the transfusion of blood products (18,19). Thromboelastography assesses the viscoelastic properties of clot formation in whole blood from the initiation of clot through clot lysis, thereby allowing the evaluation of the kinetics of a patient’s coagulation system. Authors have advocated that it has distinct benefits for patients with cirrhosis in whom traditional coagulation tests are known to be inaccurate (17,20). A recent randomized controlled trial suggested that a TEG-guided transfusion strategy may lead to significantly lower use of blood products.
Figure 1. The coagulation cascade. The coagulation cascade is initiated by the tissue factor pathway that includes tissue factor (TF) exposed on damaged or altered cell surfaces. When tissue factor binds to small amounts of factor VIIa, the complex is called extrinsic tenase, as it will convert factor X to factor Xa. Factors Xa and Va on the cell surface, along with Ca$^{2+}$, form prothrombinase complex, which converts prothrombin (factor II) to thrombin (factor IIa). Thrombin has 2 actions. First, it contributes to self-regulation of the tissue factor pathway by activating tissue factor pathway inhibitor (TFPI), which will shut down the tissue factor pathway; and second, thrombin will initiate the intrinsic pathway by activating factors XI and IX. Factor Xla will also convert factor IX to factor IXa, which, along with factor VIIIa, forms an intrinsic tenase to convert factor X to factor Xa. This is the main amplification pathway to generate thrombin. The thrombin converts fibrinogen to fibrin monomers. Factor Xlla cross-links monomers to polymerize and stabilize the clot. Antithrombin (AT) inhibits thrombin and factor Xa to regulate thrombin generation. Free thrombin also binds to thrombomodulin (TM) on endothelium and converts protein C to activated protein C (APC); protein S acts as a cofactor. The APC inactivates factors Va and VIIa to regulate thrombin generation.
medications in this group are extrapolated from surgical literature in which 5.6% of coronary bypass patients experienced severe life-threatening hemorrhage with this medication compared with 4.2% receiving placebo, with no statistically significant difference between the groups (34). Ticlopidine is known to cause thrombotic thrombocytopenic purpura and neutropenia and is therefore rarely used today (35). Clopidogrel is commonly used, and less perioperative bleeding was noted when clopidogrel was stopped 5 days before surgery in the Clopidogrel in Unstable angina to prevent Recurrent ischemic Events trial (34). Prasugrel has a stronger antiplatelet effect than clopidogrel, and platelet activity normalizes at 7 days after discontinuation (36). Ticagrelor directly inhibits the P2Y12 receptor, with a greater antiplatelet effect than clopidogrel and a faster platelet recovery time (37). Cangrelor is an intravenous, direct P2Y12 inhibitor that has a rapid onset of action with a short half-life of 3–6 minutes. It is used in acute coronary care for the prevention of periprocedural myocardial infarction or stent thrombosis, with patients being transitioned to an oral thienopyridine agent postprocedurally. Cangrelor can also be used as a “bridge” therapy option for patients receiving oral thienopyridines before surgery (38).

**Phosphodiesterase Inhibitors**

Phosphodiesterase inhibitors reduce ADP-induced platelet aggregation. They are weak antplatelet agents with associated bleeding risk that is considered to be very low. Cilostazol is used in treating symptomatic peripheral arterial disease and improves walking distance and overall quality-of-life metrics (39). It has commonly been associated with minor side effects such as headache and diarrhea, and more recently with reports of

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**Figure 2.** Mechanisms of action of common anticoagulant medications. VKA (warfarin) decreases the functional levels of factors II, VII, IX, and X by 15%–30% versus baseline (administered orally). UFH potentiates the action of antithrombin to predominantly inhibit thrombin (factor IIa) and, to a lesser extent, factor Xa (administered intravenously). LMWH potentiates the action of antithrombin to predominantly inhibit factor Xa and, minimally, thrombin (factor IIa), and fondaparinux potentiates antithrombin to inhibit factor Xa only (both administered via subcutaneous injection). Oral direct factor Xa inhibitors rivaroxaban, apixaban, edoxaban, and betrixaban inhibit factor Xa without antithrombin. Direct thrombin inhibitors can be administered orally (eg, dabigatran) or intravenously (eg, argatroban and bivalirudin).

**Table 1. Interpretation of Routine Coagulation Tests**

<table>
<thead>
<tr>
<th>PT/INR</th>
<th>PTT</th>
<th>Fibrinogen</th>
<th>D-Dimers</th>
<th>Thrombin Time</th>
<th>Platelet Count</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Liver disease, vitamin K antagonist, factor VII deficiency, oral factor Xa inhibitors</td>
</tr>
<tr>
<td>Normal</td>
<td>↑</td>
<td>Normal</td>
<td>Normal</td>
<td>↑</td>
<td>Normal</td>
<td>Unfractionated heparin, dabigatran</td>
</tr>
<tr>
<td>Normal</td>
<td>↑</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>With history of bleeding: factor VIII, IX, or IX deficiency</td>
</tr>
<tr>
<td>Normal</td>
<td>↑</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Without history of bleeding: lupus anticoagulant, factor XII deficiency</td>
</tr>
<tr>
<td>↑ ↑</td>
<td>↑ ↑</td>
<td>↓ ↓</td>
<td>↑ ↑</td>
<td>↑ ↓</td>
<td>↓ ↓</td>
<td>Acute disseminated intravascular coagulation</td>
</tr>
<tr>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>↑ ↑</td>
<td>Normal</td>
<td>Normal</td>
<td>Acute thrombosis (nonspecific)</td>
</tr>
</tbody>
</table>

INR = International Normalized Ratio; PT = prothrombin time; PTT = partial thromboplastin time.
Table 2. Properties of Antiplatelet Agents

<table>
<thead>
<tr>
<th>Drug (Brand Name)</th>
<th>Mechanism of Action</th>
<th>Half-Life</th>
<th>Drug Elimination (h)*</th>
<th>Test to Detect Drug Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Thienopyridines</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cangrelor (Kengreal)†</td>
<td>Thienopyridine (reversible)</td>
<td>3.6 min</td>
<td>0.33</td>
<td>Platelet aggregometry, VerifyNow P2Y12†</td>
</tr>
<tr>
<td>Clopidogrel (Plavix)†</td>
<td>Thienopyridine (irreversible)</td>
<td>6 h</td>
<td>30</td>
<td>Platelet aggregometry, VerifyNow P2Y12†</td>
</tr>
<tr>
<td>Prasugrel (Effient)†,§</td>
<td>Thienopyridine (irreversible)</td>
<td>3.7 h</td>
<td>20</td>
<td>Platelet aggregometry, VerifyNow P2Y12†</td>
</tr>
<tr>
<td>Ticagrelor (Brilinta)†</td>
<td>Thienopyridine (reversible)</td>
<td>7 h</td>
<td>35</td>
<td>Platelet aggregometry, VerifyNow P2Y12†</td>
</tr>
<tr>
<td>Ticlopidine (Ticlid)†</td>
<td>Thienopyridine (irreversible)</td>
<td>13 h</td>
<td>65</td>
<td>Platelet aggregometry, VerifyNow P2Y12†</td>
</tr>
<tr>
<td><strong>NSAIDs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin†</td>
<td>COX-1 inhibitor</td>
<td>2–3 h</td>
<td>10–15†</td>
<td>PFA-100, platelet aggregometry, VerifyNow ASA†</td>
</tr>
<tr>
<td>Aspirin/dipyridamole (Aggrenox)†</td>
<td>COX-1 and phosphodiesterase inhibitor</td>
<td>13 h</td>
<td>65†</td>
<td>PFA-100</td>
</tr>
<tr>
<td>Celecoxib (Celebrex)</td>
<td>COX-2 inhibitor</td>
<td>8–12 h</td>
<td>40–60</td>
<td>NA</td>
</tr>
<tr>
<td>Diclofenac (Voltaren)</td>
<td>COX-2 inhibitor</td>
<td>1–2 h</td>
<td>5–10</td>
<td>NA</td>
</tr>
<tr>
<td>Diflunisal (Dolobid)</td>
<td>COX-1 and -2 inhibitor</td>
<td>8–12 h</td>
<td>40–60</td>
<td>NA</td>
</tr>
<tr>
<td>Ibuprofen (Motrin)</td>
<td>COX-1 inhibitor</td>
<td>2–4 h</td>
<td>10–20†</td>
<td>NA</td>
</tr>
<tr>
<td>Indomethacin</td>
<td>COX-1 inhibitor</td>
<td>5–10 h</td>
<td>25–50</td>
<td>NA</td>
</tr>
<tr>
<td>Ketorolac (Toradol)</td>
<td>COX-1 and -2 inhibitor</td>
<td>5–6 h</td>
<td>25–30†</td>
<td>NA</td>
</tr>
<tr>
<td>Ketoprofen (Orudis)</td>
<td>COX-1 and -2 inhibitor</td>
<td>2–5 h</td>
<td>10–25†</td>
<td>NA</td>
</tr>
<tr>
<td>Meloxicam (Mobic)</td>
<td>COX-2 inhibitor</td>
<td>15–20 h</td>
<td>75–100</td>
<td>NA</td>
</tr>
<tr>
<td>Nabumetone (Relafen)</td>
<td>COX-2 inhibitor</td>
<td>22–30 h</td>
<td>110–150</td>
<td>NA</td>
</tr>
<tr>
<td>Naproxen (Aleeve)</td>
<td>COX-1 and -2 inhibitor</td>
<td>12–17 h</td>
<td>60–85†</td>
<td>NA</td>
</tr>
<tr>
<td>Piroxicam (Feldene)</td>
<td>COX-1 and -2 inhibitor</td>
<td>45–50 h</td>
<td>225–250</td>
<td>NA</td>
</tr>
<tr>
<td>Sulindac (Clinoril)</td>
<td>COX-1 and -2 inhibitor</td>
<td>16 h (active metabolite)</td>
<td>80</td>
<td>NA</td>
</tr>
<tr>
<td><strong>Glycoprotein IIb/IIIa inhibitors</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abciximab (ReoPro)†</td>
<td>Glycoprotein IIb/IIIa inhibitor</td>
<td>10–30 min</td>
<td>2.5</td>
<td>PFA-100</td>
</tr>
<tr>
<td>Eptifibatide (Integrilin)†</td>
<td>Glycoprotein IIb/IIIa inhibitor</td>
<td>2.5 h</td>
<td>12.5</td>
<td>PFA-100</td>
</tr>
<tr>
<td>Tirofiban (Aggrastat)†</td>
<td>Glycoprotein IIb/IIIa inhibitor</td>
<td>2 h</td>
<td>10</td>
<td>PFA-100</td>
</tr>
<tr>
<td><strong>Phosphodiesterase inhibitors</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cilostazol (Pletal)</td>
<td>Phosphodiesterase inhibitor</td>
<td>10 h</td>
<td>50†</td>
<td>NA</td>
</tr>
<tr>
<td>Dipyridamole (Persantine)</td>
<td>Phosphodiesterase inhibitor</td>
<td>10 h</td>
<td>50</td>
<td>NA</td>
</tr>
</tbody>
</table>

COX = cyclooxygenase; NA = not applicable; NSAID = nonsteroidal antiinflammatory drug; PFA-100 = platelet function analyzer-100 (this test has replaced bleeding time to assess primary hemostasis, ie, platelet function and von Willebrand disease).

†The plasma concentration of a drug is halved after 1 elimination half-life. After 5 half-lives, the amount of drug remaining is approximately 3%, which is considered to be negligible with regard to therapeutic effect for most classes of drug. However, complete drug elimination may not always reflect the time to return to normal hemostasis for all drug classes (eg, abciximab and aspirin), and specific drug-withholding recommendations are provided in table 6 of part II of this document.

‡Time to drug elimination may vary with these drugs in patients with renal failure as a result of renal excretion of the medications.

§In cases of antiplatelet-associated life-threatening bleeding requiring reversal, there are no specific antidotes to the medications themselves; however, platelet transfusions may help control bleeding/symptoms.

The US Food and Drug Administration issued a Black Box Warning for prasugrel, which should not be used in patients with active pathologic bleeding, history of ministrokes or stroke, or those requiring an urgent need for surgery, including coronary artery bypass graft surgery.

†VerifyNow P2Y12 and VerifyNow ASA are point-of-care devices that can detect a patient’s resistance to thienopyridines or acetylsalicylic acid (ASA). If a patient is resistant to these medications, the normal recommended withholding times may not apply.
of cardiovascular adverse events and also bleeding (40,41). Cilostazol has not been shown to increase bleeding time when used alone or with ace-
tyalicylic acid (42,43). If the medication is discontinued, after 5 half-lives,
less than 5% of the drug remains in the plasma, and improvements in
platelet aggregation have been demonstrated (44,45). Dipyridamole can be
used alone or in a combination extended-release form with aspirin for the
secondary prevention of stroke or transient ischemic attacks. When used in
combination, an increased risk of bleeding has been reported (46,47).

Glycoprotein IIb/IIIa Inhibitors
Glycoprotein IIb/IIIa inhibitors prevent platelet aggregation by binding to the
receptor site on the glycoprotein IIb/IIIa complex where fibrinogen
normally attaches. Abciximab, eptifibatide, and tirofiban are very potent
antiplatelet agents with very short half-lives (30–45 min) that are given
intravenously during percutaneous coronary interventions and used pri-
marily in the acute coronary care setting (48–50). Abciximab causes irre-
versible inhibition, and, although the half-life is short (10–30 minutes),
dissociation from the receptor requires hours and recovery of platelet
function is slow, with normal hemostasis achieved between 24 and 48 hours
after drug discontinuation (51,52). Eptifibatide and tirofiban have faster
dissociation times, with normalization of platelet aggregation occurring
between 4 and 8 hours after drug discontinuation (53,54). Increased peri-
operative bleeding has been noted following cardiac and vascular surgery
(55), but there are no studies on interventional procedures for patients
receiving glycoprotein IIb/IIIa inhibitors (31). The effects of these medi-
cations can be partly reversed with platelet transfusion.

Anticoagulant Agents
Table 3 summarizes the properties of anticoagulant medications.

Vitamin K Antagonists
Warfarin, the most common vitamin K antagonist (VKA) used clinically,
inhibits vitamin K epoxide reductase and vitamin K reductase in the liver,
thereby decreasing carboxylation of γ-glutamyl acid residues, which are
required for factors II, VII, IX, and X and proteins C and S to function
normally. The full anticoagulation effect of warfarin is achieved at
approximately 3–5 days, when the levels of factors II and X are sufficiently
decreased (31). When the activity of factors II and X are reduced to 30%–
15% of normal, the corresponding INRs are 2.0–3.0, respectively (56). The
presence of clotting factors at concentrations of > 40% is considered
adequate for surgical hemostasis (57). Although the use of VKAs has
decreased during the past 5 years as a result of the introduction of DOACs,
warfarin remains the anticoagulant agent of choice in many clinical condi-
tions, including mechanical heart valves, left ventricular assist devices,
and antiphospholipid antibody syndrome. The effect lasts for 5–7 days
(10,58).

There are several options for reversing the effects of VKA. A 4-factor
prothrombin complex concentrate (4F-PCC; Kcentra; CSL Behring, King
of Prussia, Pennsylvania) is the only US FDA-approved drug for VKA
reversal and should be administered according to local hospital-based
anticoagulation-reversal protocols (59,60). It contains all vitamin K–
dependent factors (II, VI, IX, and X) and natural anticoagulants (proteins
C and S). A randomized clinical trial comparing 4F-PCC with plasma for
VKA reversal (60) showed similar hemostatic efficacy in bleeding patients,
whereas another randomized clinical (59) trial showed superior hemostatic
efficacy of 4F-PCC versus plasma for VKA reversal in patients needing
urgent surgical or invasive procedures. The thromboembolic events were
similar in both studies for 4F-PCC and plasma, whereas volume overload
was higher in the plasma arm than in the 4F-PCC arm (61). Vitamin K–
dependent factors achieved hemostatic levels within 30 minutes following
4F-PCC administration, compared with several hours for plasma. If 4F-PCC
is unavailable, plasma may be used for VKA reversal; however, plasma
requires an infusion time that is 8 times longer than that for 4F-PCC (62).
Side effects of plasma infusion include volume overload, acute lung injury,
alлерgic reactions, or infections (63). Oral vitamin K can be administered to
reverse the effect of VKA for elective procedures. The intravenous
administration of 3 mg of vitamin K, diluted in 25–50 mL of normal saline
solution and infused slowly over a period of 15–30 minutes, can also
reverse VKA effect within 18 hours before major surgery, with adequate
hemostatic levels of factors (64), and has been found to be safe in some
studies (58,65). However, the FDA has issued a Black Box Warning for the
risk of anaphylactoid reactions associated with the intravenous “push” or
subcutaneous administration of vitamin K.

Heparins
Heparins (unfractionated or LMWH) are the most commonly used paren-
teral anticoagulant agents, particularly for the acute treatment of thrombo-
embolic disease or coronary syndromes. LMWH and unfractionated heparin
(UFH) potentiate the anticoagulant effects of antithrombin by many thou-
sand fold to neutralize thrombin and factor Xa. UFH predominantly inhibits
thrombin more than factor Xa (4:1 ratio) and hence prolongs PTT. Because
of the short half-life of UFH (60–90 min), waiting for 4 hours after dis-
continuing heparin and checking PTT or anti-Xa level is sufficient to
normalize the bleeding risk before any procedure (66). By contrast, LMWH
inhibits factor Xa more than thrombin (4:1 ratio) and hence generally does
not affect PTT at therapeutic doses. An anti-Xa assay can be used to
monitor LMWH if needed, especially in patients at extremes of body weight
or with impaired renal function. LMWH has a half-life of 4–6 hours, which
requires waiting for at least 24 hours before a procedure to normalize the
bleeding risk (67). Fondaparinux is a synthetic pentasaccharide that binds to
antithrombin and potentiates its effect only on factor Xa. It is often used in
place of LMWH, especially in patients with heparin-induced thrombocy-
topenia, and should be treated like LMWH.

Protamine is a heparin reversal agent. A 1-mg dose of protamine
neutralizes 100 IU of UFH, but the goal should be to neutralize only 80% of
UFH estimated at the time of protamine infusion, as excess protamine will
itself function as an anticoagulant (68). Similarly, protamine can be used to
partially neutralize LMWH at doses of 1 mg per milligram of LMWH
within 8 hours of the last dose or 0.5 mg per milligram of LMWH if beyond
8 hours (68).

Parenteral Direct Thrombin Inhibitors
Parenteral direct thrombin inhibitors, including bivalirudin and argatroban,
are increasingly encountered in clinical practice because they block
thrombin directly, resulting in a more predictable anticoagulant effect
compared with UFH. They have very short half-lives (15–40 min) and a
rapid onset of action. Argatroban is metabolized by the liver, whereas
bivalirudin is metabolized by plasma enzymes, making bivalirudin a safer
option for patients with renal or hepatic dysfunction (69). Although there
are no reversal agents, bleeding risk should normalize 2–4 hours after
discontinuation of these drugs (69).

DOACs
All DOACs are indicated for nonvalvular atrial fibrillation to prevent
thromboembolic events and are also FDA-approved for the treatment and
prevention of deep vein thrombosis and pulmonary embolism. In clinical
trials (70–72), DOACs have shown significantly lower rates of intracerebral
hemorrhage compared with VKA, but dabigatran and rivaroxaban were
associated with a higher incidence of gastrointestinal bleeding. In general,
DOACs have a rapid onset of action (within approximately 2 h) and have
short half-lives (approximately 9–17 h). Other advantages include no need
for laboratory monitoring, no effect of diet, and fewer drug interactions
compared with VKAs. However, certain clinical conditions may require
knowledge of DOAC plasma levels and effects, such as patients who
require emergent major surgeries and procedures, present with severe
bleeding, or develop thrombosis while receiving DOACs.

Direct Thrombin Inhibitor
Dabigatran etexilate is the only currently available oral direct thrombin
inhibitor (DTI). It is a prodrug that is metabolized by plasma and intestinal
proteases to the active drug dabigatran. It is given twice daily and is
excreted by the kidneys. Thus, the risk of bleeding associated with
# Table 3. Properties of Anticoagulant Medications

<table>
<thead>
<tr>
<th>Drug (Brand Name)</th>
<th>Mechanism of Action</th>
<th>Half-Life</th>
<th>Drug Elimination (h)*</th>
<th>Test to Detect Drug Effect or Presence</th>
<th>Reversal Agent (Brand Name)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin K antagonist</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Warfarin (Coumadin)</td>
<td>Inhibits function of factors II, VII, IX, and X</td>
<td>40 h</td>
<td>200</td>
<td>PT/INR or chromogenic factor X</td>
<td>4F-PCC (Kcentra), plasma†</td>
</tr>
<tr>
<td>Heparins</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low molecular weight: enoxaparin (Lovenox) and dalteparin (Fragmin)</td>
<td>Indirect factor Xa inhibition</td>
<td>2–6 h‡</td>
<td>10–30</td>
<td>Anti-Xa assay</td>
<td>Protamine</td>
</tr>
<tr>
<td>Unfractionated</td>
<td>Inhibits thrombin more than factor Xa</td>
<td>1.5–2 h†</td>
<td>7.5–10</td>
<td>PTT, anti-Xa assay</td>
<td>Protamine</td>
</tr>
<tr>
<td>Direct thrombin inhibitors</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Argatroban (Acova)</td>
<td>Direct thrombin inhibitor</td>
<td>50 min</td>
<td>4</td>
<td>PTT or TT</td>
<td>None</td>
</tr>
<tr>
<td>Bivalirudin (Angiomax)</td>
<td>Direct thrombin inhibitor</td>
<td>25 min</td>
<td>2†</td>
<td>PTT or TT</td>
<td>None</td>
</tr>
<tr>
<td>Dabigatran (Pradaxa)</td>
<td>Direct thrombin inhibitor</td>
<td>12–17 h</td>
<td>60–85§</td>
<td>TT, ecarin clotting time</td>
<td>Idarucizumab (Praxbind)</td>
</tr>
<tr>
<td>Factor Xa inhibitors</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apixaban (Eliquis)</td>
<td>Direct factor Xa inhibitor</td>
<td>15 h</td>
<td>75§</td>
<td>Anti-Xa assay, apixaban assay where available</td>
<td>Andexanet alfa (Andexxa) PCC</td>
</tr>
<tr>
<td>Betrixaban (Bevyxxa)</td>
<td>Direct factor Xa inhibitor</td>
<td>37 h</td>
<td>185§</td>
<td>Anti-Xa assay</td>
<td>Andexanet alfa (Andexxa)</td>
</tr>
<tr>
<td>Edoxaban (Savaysa)</td>
<td>Direct factor Xa inhibitor</td>
<td>9–14 h</td>
<td>45–70§</td>
<td>Anti-Xa assay</td>
<td>Andexanet alfa (Andexxa) PCC</td>
</tr>
<tr>
<td>Fondaparinux (Arixtra)</td>
<td>Indirect factor Xa inhibitor</td>
<td>17–21 h</td>
<td>85–105§</td>
<td>Fondaparinux assay</td>
<td>Andexanet alfa (Andexxa)</td>
</tr>
<tr>
<td>Rivaroxaban (Xarelto)</td>
<td>Direct factor Xa inhibitor</td>
<td>9–13 h</td>
<td>45–65§</td>
<td>Anti-Xa assay, rivaroxaban assay where available</td>
<td>Andexanet alfa (Andexxa) PCC</td>
</tr>
</tbody>
</table>

4F-PCC = 4 factor–prothrombin complex concentrate; INR = International Normalized Ratio; PT = prothrombin time; PTT = partial thromboplastin time; TT = thrombin time.

*The plasma concentration of a drug is halved after 1 elimination half-life. After 5 half-lives, the amount of drug remaining is approximately 3%, which is considered to be negligible with regard to therapeutic effect for most classes of drug. However, complete drug elimination may not always reflect the time to return to normal hemostasis for all drug classes, and specific drug-withholding recommendations are provided in table 6 of part II of this document.

† Plasma only if 4F-PCC is unavailable.

‡ The range of half-life times presented for the heparin classes of drugs reflect times for intravenous and subcutaneous administration.

§ Time to normal hemostasis may vary with these drugs in patients with renal failure as a result of renal excretion of the medications.
dabigatran is increased in patients with renal impairment. In a bleeding patient or someone requiring an emergent intervention, the presence of dabigatran can be assessed by thrombin time, which is exquisitely sensitive to even very low levels of dabigatran; a normal thrombin time measurement excludes the presence of dabigatran. Idarucizumab, a humanized antigen-binding fragment monoclonal antibody, is a specific reversal agent for dabigatran with a rapid onset of action; 2 doses of 5 g are given 15 minutes apart (73). Dialysis can also remove dabigatran from the circulation.

**Direct Factor Xa Inhibitors**

By binding to factor Xa, direct factor Xa inhibitors, including rivaroxaban, apixaban, edoxaban, and betrixaban, decrease the conversion of prothrombin to thrombin, ultimately limiting the conversion of fibrinogen to fibrin clot. These agents are excreted by the kidneys, and thus renal impairment may prolong their clearance. Prolongation of PT/INR is variable with these medications, so measurement of anti-Xa activity, as is done in patients receiving LMWH, may help to detect their presence in the plasma of patients needing urgent or emergent intervention. An anti-Xa activity of < 0.2 U/mL should be safe for most interventional procedures. Andexanet alfa, a recombinant factor Xa molecule that acts as a decoy, was recently approved by the FDA as a specific reversal agent for all factor Xa inhibitors, LMWH, and fondaparinux (74). In animal experiments and human ex vivo and in vitro studies (75), prothrombin complex concentrates (50 U/kg) were shown to be somewhat effective in neutralizing factor Xa inhibitors.

**Other Medications**

Over-the-counter herbal medications and supplements are commonly used as self-remedies by patients (76,77). The majority of common herbal medications, such as gingko biloba, ginseng, licorice, and garlic, affect hemostasis through a variety of pathways, usually culminating in the inhibition of platelet function. Many of these herbal agents can affect the efficacy of other medications, including DOACs and warfarin. St. John’s wort increases the metabolism of warfarin and decreases its circulating blood time, thereby increasing the risk of thrombosis (78). Ginkgo is known to increase bleeding risk in patients who are also taking cilostazol and warfarin (79). Studies do not conclusively demonstrate an increased bleeding risk. Nevertheless, the interventionalist is encouraged to ask the patient about the use of nontraditional treatments and consult with a pharmacist on their potential implications in patient care given their propensity for drug–drug interactions.

Selective serotonin reuptake inhibitors (SSRIs) are commonly prescribed antidepressant medications. These drugs decrease platelet serotonin uptake from the blood. As serotonin plays a role in platelet aggregation, SSRIs have an inhibitory effect on platelet aggregation, and risk of bleeding approximates that associated with low-dose ibuprofen (31). However, studies have shown that SSRI use can be associated with surgical bleeding in breast and orthopedic surgery (80,81) and that the risk of gastrointestinal bleeding increases with SSRIs and concurrent antithrombotic therapy (82,83). Combined use of SSRIs and NSAIDs or low-dose aspirin increased the observed/expected ratios of upper gastrointestinal bleeding to 12.2 (95% confidence interval [CI], 7.1–19.5) and 5.2 (95% CI, 3.2–8.0), respectively, in a large epidemiologic study (83). The risk of bleeding was also increased with concomitant anticoagulant agent use and in patients with chronic liver disease (CLD) (84). Although routine discontinuation is not advocated, for patients at high risk, multidisciplinary discussion with the treating physician may be of benefit to aid in clinical decision-making.

**NUANCES OF COAGULATION SPECIFIC TO SELECT CLINICAL COMORBIDITIES**

**CLD**

The interpretation of coagulation test results in patients with cirrhosis or CLD can be difficult, and it is incorrect to assume that patients with CLD with prolonged PT/INR are autoanticoagulated; very large epidemiological studies (85) have demonstrated that patients with CLD have almost twice the thrombotic risk as the general population. In fact, patients with CLD have rebalanced primary and secondary hemostasis (Table 4) (86). The coagulopathy of CLD is unique because the liver synthesizes procoagulant factors, with the exception of factor VIII and VWF, and all-natural anticoagulants (eg, proteins C and S and antithrombin) that regulate thrombin generation. The routine coagulation tests used to assess hemostasis, PT/INR and PTT, reflect only decreased procoagulant factors, not the concomitantly decreased natural anticoagulants, thereby resulting in an inaccurate assessment of hemostasis. For example, it would be incorrect to interpret an INR of 3 in a patient with CLD as representing a significant increase in bleeding risk; as a result of the decrease in production of procoagulant and natural anticoagulant factors, the plasma of the patient with CLD actually has the same amount of thrombin generation as the plasma of a normal patient (87). Results from TEGER support these hypotheses and argue against the use of plasma administration to correct abnormal laboratory values, as patients with CLD with very prolonged PT/INR have normal clotting times or normal reaction times when assessed by ROTEM or TEG, respectively (17). Many patients with advanced CLD also have hypofibrinogenemia (< 100 mg/dL) and a degree of dysfibrinogenemia (86).

Most patients with advanced CLD have moderate thrombocytopenia (platelet counts of 40–80 × 10^9/L). Thrombocytopenia in CLD is multifactorial: thrombopoietin is reduced; patients are deficient in folate and vitamin B12; there may be an element of bone-marrow suppression especially in the setting of hepatitis C, and as many as 80% of patients have splenomegaly with platelet sequestration (86). However, it is important to note that the adhesive function of these platelets is actually enhanced, as VWF function is increased by 4–5 fold versus normal. The VWF function is further enhanced by the reduced amount of ADAMTS13 enzyme that regulates VWF multimer sizes. Thus, overall primary hemostasis is rebalanced (86), and, similar to the interpretation of PT/INR values, quantitative evidence of thrombocytopenia periprocedurally does not always imply increased bleeding risk. Therefore, the involvement of a transfusion medicine or hemostasis specialist may be in the best interest of a patient with CLD when determining when platelet transfusion or other agents should be used.

A recent prospective study of 363 patients with cirrhosis with thrombocytopenia who underwent 852 invasive procedures (88) showed that postprocedural bleeding is rare in patients with CLD and unrelated to platelet counts. Ten postprocedural bleeding episodes (1 per 84 procedures) were reported, but none of the patients who had platelet counts < 50 × 10^9/L (n = 49) experienced any bleeding. The authors concluded that the recommendation to transfuse platelets when the platelet count is < 50 × 10^9/L is not substantiated by this case series and that postprocedural bleeding is not predicted by INR or platelet count (88). Reported results of treatment with agonists of the thrombopoietin receptor, which are designed to increase platelet counts, are mixed. A randomized controlled clinical trial (89) comparing the use of eltrombopag (Promacta; Novartis, Basel, Switzerland) versus placebo in patients with cirrhosis was discontinued because the eltrombopag arm exhibited increased incidences of portal vein thrombosis, even though bleeding events were similar in both arms (89).

Avatrombopag is the newest FDA-approved thrombopoietin receptor agonist for patients with cirrhosis with thrombocytopenia who are scheduled to undergo a procedure. Two randomized controlled studies (90) were conducted with the primary endpoint of whether the use of avatrombopag would result in a platelet count of 50 × 10^9/L before the procedure, thereby avoiding the need for platelet transfusion (90). The drug must be taken daily for 5 consecutive days with the procedure to be scheduled 5–8 days after the last dose (91). Avatrombopag increased platelet count to > 50 × 10^9/L in 66% of patients receiving a high dose (60 mg for platelet count < 40 × 10^9/L) and 88% of patients receiving a low dose (40 mg for platelet count of 40–50 × 10^9/L) compared with 23%–38% of patients receiving placebo (90). Patients receiving avatrombopag required fewer platelet transfusions than patients receiving placebo (P < .0001). Avatrombopag did not cause increased thrombotic complications, nor was there any difference in bleeding events between groups of patients receiving placebo and avatrombopag. Although both studies met their primary endpoints, the utility of this drug remains questionable in procedures associated with low to medium bleeding risk in view of the results presented by other authors (20,89), which seem to suggest that the accepted thresholds of INR < 1.5 and...
platelet count of $50 \times 10^9/L$ before a procedure do not predict bleeding risk and may not be the correct safety thresholds for patients with cirrhosis undergoing invasive procedures.

**Chronic Renal Failure**

The pathogenesis of chronic kidney disease (CKD)-related bleeding is tied to (i) primary hemostatic defect secondary to an abnormal platelet–endothelial interaction caused by the presence of a middle molecule that interferes with VWF function and (ii) anemia, as red cell mass has a rheologic effect on platelets and provides ADP for platelet activation (92). As a result, patients with CKD have an increased tendency to experience bleed at baseline (93), during endovascular procedures (94), and when receiving antiplatelet agents or anticoagulation (95). Risk exists at all CKD stages, but is most pronounced in patients with uremia (92,96). Therefore, CKD should be accounted for when considering an endovascular procedure and reinitiation of postprocedural anticoagulation and antiplatelet therapy. Unfortunately, there are no well-validated tests to aid in assessing a patient’s periprocedural bleeding risk (92). Other platelet function-related tests (eg, platelet aggregation) are not routinely available in most centers. Finally, CKD should be considered when offering preprocedural medication-related recommendations, as the bioavailability of many medications is influenced by renal function. This is specifically true for many anticoagulant agents, including LMWH, fondaparinux, and DOAC (97). Typically, “hold” times, particularly before elective procedures, will be relatively prolonged in the presence of CKD.

**Thrombocytopenia**

A low platelet count is associated with increased bleeding (15,98), but bleeding risk and management options differ depending on the etiology of thrombocytopenia.

**Immune Thrombocytopenia**

Immune thrombocytopenia (ITP) is defined as a platelet count < $100 \times 10^9/L$ that is caused by autoantibodies, which results in immune destruction of platelets (99,100). Primary ITP is an acquired immune disorder, whereas secondary ITP is associated with other underlying autoimmune disorders, such as systemic lupus erythematosus, HIV, or underlying immune dysregulation syndromes, such as common variable immunodeficiency (101). Most patients with ITP have large platelets in peripheral blood as a result of the rapid and premature release from megakaryocytes, and these are typically hyperfunctional compared with normal-sized platelets (102,103).

First-line therapy for ITP can include corticosteroid agents, with many patients achieving a response within 2–4 weeks (104). Corticosteroids are relatively contraindicated (105,106). Splenectomy is often used as a last resort: surgical splenectomy and splenic embolization have been used in this setting to provide similar levels of platelet response. Surgical splenectomy has initial responses as high as 80%, with sustained response rates decreasing to 66% (105,106). Platelet transfusions alone are often ineffective in increasing platelet counts because autoantibodies will destroy transfused platelets within minutes unless they are given with intravenous immunoglobulin.

Although the safety of minimally invasive image-guided procedures in patients with hematologic disorders such as ITP has not yet been satisfactorily established, it is widely accepted that there is an increased risk of hemorrhage in these patients. A recent study (107) compared endoscopy procedure-related bleeding in patients with ITP or aplastic anemia versus the procedural outcomes in matched control subjects without hematologic disorders. The endoscopic interventions included low-risk procedures such as endoscopic biopsy and high-risk procedures including polypectomy, endoscopic resection, and endoscopic retrograde cholangiopancreatography with sphincterotomy. The study (107) showed that bleeding occurred in 9.7% of procedures among the patients with thrombocytopenia, compared with 3.1% in the control patients ($P = .003$). Bleeding occurred after 20% of all high-risk procedures, and the incidence of bleeding was significantly increased in patients with a platelet count less than $50 \times 10^9/L$.

**Nonimmune Thrombocytopenia**

Patients with non–immune-mediated etiologies of thrombocytopenia will often respond well to platelet transfusions, with the exception of those with splenomegaly, in which cases transfused platelets are sequestered in minutes (108). Cancer-related thrombocytopenia will be discussed in more detail in the following section. Platelet consumption in “platelet-rich thrombi” is the underlying mechanism in heparin-induced thrombocytopenia and thrombotic thrombocytopenic purpura, and platelet transfusions are relatively contraindicated (109).

**Cancer**

Cancer is associated with increased risk of venous and arterial thrombosis (110). Patients with a history of cancer and VTE > 12 months and no other risk factors, as well as patients with active cancer, defined as having been treated within the previous 6 months or receiving palliative therapy, are considered to be at moderate risk for periprocedural thromboembolism, ie, an annual risk of arterial thromboembolism of 5%–10% and a 1-month VTE risk of 2%–10% (110–112). The risks are even higher for patients with advanced-stage cancer, high-risk cancer histologies (eg, stomach, pancreas, lung), high-risk biomarkers, thrombotic event within 3 months, or thrombophilia, ie, an annual risk of arterial thromboembolism > 10% or 1-month VTE risk > 10% (110–112). Thrombocytopenia is also common as a result of the disease (usually hematologic malignancies) or a consequence of treatment (113). Adequate knowledge of this patient population and the impact of pre- and postprocedural anticoagulation management is essential,

### Table 4. Rebalanced Hemostasis in Chronic Liver Disease

<table>
<thead>
<tr>
<th>Primary Hemostasis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Platelets</td>
</tr>
<tr>
<td>Thrombopoietin</td>
</tr>
<tr>
<td>Bone marrow function</td>
</tr>
<tr>
<td>Splenomegaly</td>
</tr>
<tr>
<td>Nutritional deficiency</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Secondary Hemostasis</td>
</tr>
<tr>
<td>Procoagulants</td>
</tr>
<tr>
<td>Factors I, II, V, VII, IX, X, XI, XIII</td>
</tr>
<tr>
<td>Anticoagulants</td>
</tr>
<tr>
<td>Procoagulant</td>
</tr>
<tr>
<td>Antithrombin, protein C, protein S</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Fibrinolytic System</td>
</tr>
<tr>
<td>Plasminogen and α2 antiplasin</td>
</tr>
<tr>
<td>TPA = tissue plasminogen activator; VWF = von Willebrand factor.</td>
</tr>
</tbody>
</table>

\[ VWF(VWF) \] / ABSTRACTIONS:  

- Plasminogen and ADAMTS13

- Antithrombin, protein C, protein S

- Factor VIII

- Procoagulant

- Platelet aggregation

- Autoantibodies

- Platelet consumption

- Thromboplastin

- Platelet-rich thrombi

- Heparin-induced thrombocytopenia
as these patients frequently undergo procedures for diagnosis, curative therapy, or palliation.

It is not uncommon to encounter patients with cancer who are receiving anticoagulation (110). Tafur et al (114) prospectively followed 2,182 chronically anticoagulated patients who were referred for peri-procedural anticoagulation management to estimate the 3-month incidences of thromboembolism, major bleeding, and survival. In this cohort, 20% of all patients (n = 435) had active malignancy, and the indication for anticoagulation was VTE in 50% (n = 218). This study (114) showed distinct differences between patients with and without cancer: the VTE rate was higher (1.2% vs 0.2%; P = .001), the major bleeding rate was higher (3.4% vs 1.7%; P = .015), and the survival rate was reduced in patients with cancer (95% vs 99%; P < .001). Patients with cancer receiving anti-coagulation and undergoing bridging therapy had higher rates of peri-procedural VTE and major bleeding compared with patients without cancer who were receiving chronic anticoagulant therapy (114); however, the procedure-specific bleeding risk (ie, procedures with low vs high bleeding risk) did not significantly impact the incidence of major bleeding in patients with cancer.

Thrombocytopenia and its severity may depend on the type of malignancy, stage of cancer, or treatment (113). However, the bleeding risk has not been well established in patients with cancer with thrombocytopenia undergoing procedures (115,116), a finding acknowledged in the 2018 American Society of Clinical Oncology Clinical Practice guideline update “Platelet Transfusion for Patients with Cancer” (117), in which the recommendation for a minimal threshold platelet count for the performance of a major invasive procedure was noted to be supported by low-quality evidence and associated with a weak strength of recommendation.

DIC

DIC is characterized by systemic activation of coagulation, with the potential to cause thrombotic and hemorrhagic events. It is a heterogeneous syndrome that may present as an acute and life-threatening emergency or as a chronic asymptomatic process (118). DIC is typically triggered by an underlying event such as sepsis, trauma, or obstetric complications. The pathophysiology typically includes 4 main steps. First, there is a procoagulant exposure. The source of the procoagulant (eg, tissue factor, bacteria-derived lipopolysaccharide, cancer procoagulant) will vary depending on the underlying cause of DIC. Second, the coagulation cascade is activated, leading to the formation of micro- and macrothrombi consisting of fibrin and platelets in the microvasculature and/or larger vessels. Third, fibrinolysis is activated at sites of thrombi formation, resulting in the production of D-dimers and fibrin degradation products. When present in significant amounts, fibrin degradation products can interfere with systemic fibrin clot formation and platelet aggregation. Finally, end-organ and tissue damage may occur from arterial thrombosis, reduced perfusion, and bleeding (119,120). A patient with DIC can have significant intraprocedural and postprocedural bleeding, which may be difficult to control. Given the complex disease process of DIC and heterogeneous presentation of patients with this syndrome, a multidisciplinary discussion regarding the risks and benefits of any image-guided procedure is necessary before an appropriate peri-procedural transfusion management strategy is determined.

Cardiovascular Disease and Arrhythmias

Patients with nonvalvular atrial fibrillation can be expected to receive long-term anticoagulation to reduce the risk of stroke and systemic embolization (8). In addition, patients with cardiac stents or those with a history of acute coronary syndrome will likely receive antiplatelet therapy. Current guidelines (121,122) recommend a minimum of 1 month of DAPT for patients receiving a bare metal stent and 6–12 months of DAPT for patients receiving drug-eluting stents. For patients with a history of acute coronary syndrome, irrespective of whether a percutaneous coronary intervention with or without stent placement was performed, the recommendation is for 12 months of DAPT (123). Similar considerations may exist after peripheral intervention and, most significantly, after carotid intervention.

As a result, management of patients with known cardiovascular disease and/or arrhythmias can be particularly challenging, as the interventionalist has to consider the patient’s risk of stent thrombosis or major adverse cardiovascular and/or cerebrovascular events. Importantly, premature discontinuation of antiplatelet therapy has been shown to be the most important predictor of stent thrombosis (hazard ratio, 89.78; 95% CI, 29.90–269.60; P < .001) in a prospective observation cohort study of 2,229 patients (124). The premature discontinuation of antiplatelet therapy can be associated with a 6% risk of periprocedural stent thrombosis and a 45% mortality rate for periprocedural myocardial infarction secondary to stent thrombosis (124). Therefore, given the complexities inherent in the management of patients with cardiac stents or acute coronary syndrome, particularly if the stent implantation or cardiac event occurred within 1 year, it is recommended that a cardiology or vascular or internal medicine consultation be obtained for patients who are being considered for procedures that require the discontinuation of antiplatelet therapy, and that a discussion of the risks with the patient be documented in the medical record.

CONCLUSIONS

The development of the appropriate management approach to the patient undergoing interventional procedures requires an understanding of the coagulation cascade and how it can be affected by common clinical conditions and antiplatelet or anticoagulation medications. Familiarity with the basic pharmacologic properties and appropriate methods of reversal for each medication class is essential.

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REFERENCES


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