THE membership of the Society of Interventional Radiology (SIR) Standards of Practice Committee represents experts in a broad spectrum of interventional procedures from both the private and academic sectors of medicine. In general, Standards of Practice Committee members dedicate the vast majority of their professional time to performing interventional procedures; as such they represent a valid broad expert constituency of the subject matter under consideration for standards production.

Technical documents specifying the exact consensus and literature review methodologies as well as the institutional affiliations and professional credentials of the authors of this document are available upon request from SIR, 3975 Fair Ridge Dr, Ste 400 North, Fairfax, VA 22033.

METHODOLOGY

SIR produces its Standards of Practice documents by using the following process. Standards documents of relevance and timeliness are conceptualized by the Standards of Practice Committee members. A recognized expert is identified to serve as the principal author for the standard. Additional authors may be assigned dependent upon the magnitude of the project. An in-depth literature search is performed by using electronic medical literature databases. Then, a critical review of peer-reviewed articles is performed with regard to the study methodology, results, and conclusions. The qualitative weight of these articles is assembled into an evidence table, which is used to write the document such that it contains evidence-based data with respect to content, complication rates, outcomes, and thresholds for prompting quality assurance reviews.

When the evidence of literature is weak, conflicting, or contradictory, consensus for the parameter is reached by a minimum of 12 Standards of Practice Committee members by using a Modified Delphi Consensus Method (Appendix) (1). For purposes of these documents, consensus is defined as 80% Delphi participant agreement on a value or parameter.

The draft document is critically reviewed by the Standards of Practice Committee members, either by telephone conference calling or face-to-face meeting. The finalized draft from the Committee is sent to the SIR membership for further input/criticism during a 30-day comment period. These comments are discussed by the Standards of Practice Committee, and appropriate revisions are made to create the finished standards document. Before its publication, the
document is endorsed by the SIR Executive Council.

INTRODUCTION AND BACKGROUND

The hematologic management of the patient undergoing percutaneous image-guided intervention is complex due to the wide range of procedures and equally wide range of patient demographics and co-morbidities. A concurrent increase in the use of both short- and long-term anticoagulation, as well as the increasing use of anti-platelet agents, further complicates the preprocedural management of these patients. Despite the continuing increase in the volume of percutaneous imaging-guided procedures, there is a general paucity of data regarding the periprocedural management of the patient with abnormal coagulation parameters. In the absence of data, clinicians may respond to the patient with abnormal coagulation parameters by canceling or postponing the procedure, altering an otherwise indicated procedure, or infusing blood products such as fresh-frozen plasma (FFP) or platelets. Recommendations from open surgical experience may not be applicable to interventional procedures because of direct visualization and the ability to obtain prompt vascular control in open cases. Finally, medicolegal factors may influence the management of the patient, as clinicians feel the need to “correct” an abnormal coagulation factor, despite the fact that studies of bleeding complications in percutaneous procedures have not shown correlation between mild to moderate abnormality of preprocedural coagulation parameters and a higher incidence of bleeding complications.

The coagulation status of patients undergoing imaging-guided interventions should be assessed whenever the procedure involves direct entry into the arterial or venous system as an anticipated part of the procedure or whenever there is a possibility of inadvertent entry into the arterial or venous system with significant sized interventional devices or tools. Patients are at increased risk for delayed detection of postprocedural hemorrhage when the site of the intervention is not easily assessed and poorly controllable (eg, percutaneous intraperitoneal procedures). Coagulation status is complex; components of the intrinsic and extrinsic coagulation cascade and platelet function figure integrally into human hemostasis. The components of coagulation are evaluated by multiple tests of hemostasis. These tests and the component of coagulation function they assess are described below and summarized in Table 1, along with normal values for each test.

DEFINITIONS

Coagulation Parameters

*Prothrombin time (PT).—*The PT test measures the clotting time upon activation of the extrinsic coagulation pathway. It is used for monitoring oral anticoagulant therapy and is now widely reported as an international normalized ratio (INR). The degree of prolongation of the clotting time correlates with the degree of deficiency or inhibition of extrinsic or common pathway clotting factors I (fibrinogen), II (prothrombin), V, VII, and X, which are synthesized by the liver. When any of these factors is deficient, the PT is prolonged and the INR is elevated. The PT in a healthy adult is approximately 11–14 seconds. There is variation depending on the reagent used in the test (2).

*INR.—*The INR is an expression of the results of a PT in a standardized testing environment. It is calculated by using an international standard that corrects for laboratory variation. The INR allows for universal standardization of anticoagulant therapy. In the following calculation, the ISI is the International Sensitivity Index of the thromboplastin reagent used in the assay: INR = (patient PT/control PT)^ISI.

In this test, the patient’s plasma is mixed with a PT reagent containing thromboplastin and calcium chloride. The time to clot formation is measured. The degree of prolongation of the clotting time correlates with the degree of deficiency or inhibition of extrinsic or common pathway clotting factors I (fibrinogen), II (prothrombin), V, VII, and X, which are synthesized by the liver. When any of these factors is deficient or inhibited, the PT is prolonged and the INR is elevated. The INR in a normal patient not undergoing warfarin therapy is 0.9–1.1.

A prolonged PT and elevated INR occur with vitamin K deficiency, lupus anticoagulants, extrinsic pathway coagulation factor deficiencies, liver disease, disseminated intravascular coagulation bile duct obstruction, malabsorption, and other conditions. Hirudin, argatroban, and heparin may prolong the PT.

The degree of prolongation of the clotting time correlates with the degree of deficiency or inhibition of extrinsic or common pathway clotting factors I (fibrinogen), II (prothrombin), V, VII, and X, which are synthesized by the liver. When any of these factors is deficient, the PT is prolonged and the INR is elevated. The coagulation factors are synthesized in the liver, and the PT is elevated with severe liver failure and acute liver injury (3,4).

*Activated partial thromboplastin time (PTT).—*The activated PTT measures the clotting time upon activation of the intrinsic coagulation pathways. In this test, the patient’s plasma is mixed with reagent containing an activator, phos-
pholipid, and calcium chloride. The time to clot formation is measured.

A normal activated PTT in an adult is approximately 25–35 seconds. A therapeutic ratio of 1.5–2.5 times the control value is frequently employed in heparin therapy; however, this range varies depending on the reagent.

A prolonged PTT occurs with factor deficiencies (especially of factors VIII, IX, XI, and/or XII), inhibitors (lupus anticoagulants), liver disease, disseminated intravascular coagulation, vitamin K deficiency, or therapeutic anticoagulants such as heparin, hirudin, or argatroban). The PTT is not useful in monitoring warfarin therapy (5).

**Thrombin time.**—Thrombin time provides an assay for fibrinogen concentration indirectly by measuring exogenous thrombin-activated clotting times (6).

**Bleeding time.**—Originally introduced in 1901 by Milian, the bleeding time has been used to diagnose platelet disorders, assess patients for clinically significant bleeding tendencies before invasive procedures, and assess the effects of various therapies on bleeding tendencies and platelet function. The bleeding time has largely fallen out of favor in modern clinical practice as an assessment for bleeding tendencies because of conflicting data on its usefulness (7).

**Platelet count.**—The platelet count is generally measured as a standard part of the complete blood count. It is used to diagnose and follow bleeding disorders, thrombocytopenia, drug-induced thrombocytopenia, disseminated intravascular coagulation, and neoplastic disorders and to evaluate the response to platelet transfusions. A normal adult platelet count is approximately 150,000–450,000 platelets per microliter of blood. A platelet count of less than 20,000/μL is a life-threatening event in which spontaneous bleeding may occur.

A small number of patients receiving heparin (including low-dose heparin) develop thrombocytopения. Drugs and chemicals associated with thrombocytopenia include chemotherapeutic agents, chloramphenicol, colchicine, H2 blocking agents, heparin, hydralazine, indomethacin, isoniazid, quindine, streptomycin, sulfonamide, thiazide diuretic, and tolbutamide. Estrogen and oral contraceptives may cause elevated platelet levels.

### Anticoagulants

**Warfarin.**—Warfarin (Coumadin; Bristol-Myers Squibb, New York, New York) antagonizes the production of the vitamin K–dependent clotting factors (II, VII, IX, X) in the liver. The clinical effect is measured with the INR, which reflects antagonism of factor VII, which has the shortest half-life of approximately 6 hours. Therapeutic INR values may vary by indication for anticoagulation but most often range from 2.2 to 2.8. Patient co-morbidities may significantly alter the effect of Coumadin. Congestive heart failure, malignancy, malnutrition, diarrhea, unsuspected vitamin K deficiency, and concomitant antibiotic use may all enhance the response to Coumadin.

**Heparin (unfractionated).**—Unfractionated heparin potentiates the action of antithrombin III, is dosed according to weight, and is administered by means of continuous intravenous infusion. Therapeutic response is monitored by activated PTT, which is targeted at 1.5–2.5 times normal.

**Platelet count is monitored after the administration of heparin for the possibility of heparin-induced thrombocytopenia, which is defined as a platelet count of less than 150,000/μL or a 50% decrease in platelet count within 5–10 days of the start of therapy. There are two types of thrombocytopenia: type I is a benign self-limited disorder where the platelet count is rarely less than 100,000/μL and type II is a possible life-threatening disorder with platelet counts often less than 75,000/μL and often seen in association with acute arterial and/or venous platelet rich “white” thrombi.

**Low-molecular-weight heparin.**—Low-molecular-weight heparin is administered subcutaneously and often dosed by weight. It does not affect the values of the INR or activated PTT. Therapeutic dosing (ie, treatment of acute deep venous thrombosis) is at 12-hour intervals, whereas prophylactic dosing (ie, postoperative deep venous thrombosis prophylaxis) is at 24-hour intervals.

### Hemostatic Agents

**FFP.**—The effect of FFP is variable due to the variable concentration of vitamin K dependent clotting factors. On average, at least 10 mL/kg is needed to effectively raise plasma protein levels. Common dose ranges from 15–30 mL/kg. In practice, in the patient with an INR in the 2.5 range, 2 units of FFP may be effective in reversing the effect of Coumadin. Patients with higher INR levels should be dosed accordingly, with possible concomitant use of vitamin K (8–11).

**Platelets.**—Fractionated blood product used in the setting of thrombocytopenia or platelet dysfunction. Often dosed as 4–6 units (random donors) or as one single donor unit (12).

**Protamine.**—Protamine may be used in emergency situations when rapid reversal of heparin is needed before a procedure or when heparin reversal is desired before the removal of arterial catheters or sheaths. Protamine has a rapid onset of action within 10 minutes after administration. Its half-life, however, is short and ranges from 5 to 7.5 minutes, which can lead to “paradoxic” re-anticoagulation after protamine administration. Protamine dosing strategies vary considerably. A “neutralizing dose” of protamine is 2 mg/kg. Protamine may also be dosed according to the amount of heparin given, on a 1-mg protamine to 100-unit heparin ratio. To reverse commonly given intraprocedural doses of heparin (3,000–5,000 IU), we often give a total of 50-mg protamine in an adult. Protamine should be given by means of slow intravenous push or infusion over 5–10 minutes. Side effects include hypotension, bradycardia, pulmonary arterial hypertension, decreased oxygen consumption, and anaphylactoid reactions (13–17).

**Vitamin K (phytonadione).**—Vitamin K may be given orally, intravenously, or subcutaneously depending on the value of the INR and the desired timeframe for anticoagulant reversal. The American College of Chest Physicians has published evidence-based guidelines for the use of vitamin K in managing elevated INRs and/or clinical bleeding in patients receiving oral anticoagulation (18). In stable, elective patients without active bleeding, oral administration (5–10 mg in adults) is preferred. Although intravenous administration of vitamin K is associated with a risk of anaphylactoid reaction, it has a more rapid effect than the subcutaneous route and may be more effective in the truly emergent patient. The U.S. Food and Drug Ad-
administration has issued a black box warning for the subcutaneous, intravenous, and intramuscular routes of administration due to reports of severe reactions, including fatalities (19,20).

Cryoprecipitate.—Cryoprecipitate is used in acquired or hereditary deficiencies of fibrinogen. Ten bags will typically increase the fibrinogen level by 75 mg/dL in a 70-kg patient (21).

Recombinant factor VIIa.—Recombinant factor VIIa is used in hemophilia in patients with inhibitors to factor 8 or in severe, nonhemophilia-related bleeding such as acute trauma (22).

Desmopressin.—Desmopressin (1-deamino-8-D-arginine vasopressin [DDAVP]) is a synthetic analogue of antidiuretic hormone. DDAVP acts through an unclear mechanism to enhance the plasma levels of factor VIII and von Willebrand factor (23). A dose of 0.3 μg/kg is given intravenously, usually diluted in 100 mL of normal saline and infused over 20–30 minutes. A single dose can be expected to raise the factor VIII level 3–6 fold. Adverse effects may include mild hypotremia. Tachyphylaxis has been reported in patients who have received multiple treatments. There are case reports of vascular thrombosis and myocardial ischemia after intravenous administration (24).

DDAVP may be indicated before image-guided procedures in patients with hemophilia, von Willebrand disease, and acquired platelet disorders due to uremia, liver disease, or anti-platelet agents (25).

Transfusion Management

FFP.—The most common intervention before imaging-guided procedures is transfusion of FFP. In the United States, more than 3 million units of FFP are transfused each year. Dzik and Rao (26) reported in a 3-month audit of FFP usage at the Massachusetts General Hospital that the most common reason for prescribing FFP was to prepare the patient with an elevated INR for an invasive procedure. This indication accounted for one-third of all requests for FFP. Stanworth et al (27) reported a review of 57 randomized controlled trials investigating the efficacy of FFP to prevent hemorrhagic complications over a wide variety of indications and clinical settings, including cardiac surgery. They found the data insufficient to recommend or refute the prophylactic use of FFP. Due to the lack of data, percutaneous procedures were not included in this comprehensive review. There is a clear need for additional investigation of the use of FFP with imaging-guided procedures (27).

Segal and Dzik (28) recently reported an analysis of 25 studies analyzing the ability of abnormal coagulation parameters to predict bleeding associated with invasive bedside or image-guided procedures. Of the 25 studies available for analysis, one was a clinical trial (comparison of transjugular liver biopsy to percutaneous biopsy with tract plugging [29]). The remaining studies were case series. The studies included patients undergoing bronchoscopy with biopsy, central vein cannulation, femoral angiography, liver biopsy, kidney biopsy, paracentesis, thoracentesis, and lumbar puncture. Overall, the authors concluded that elevated coagulation parameters provide little to no predictive value for bleeding complications from imaging-guided interventions. They assert that, in the absence of randomized, controlled studies, mild to moderate elevation of coagulation times should neither be assumed to represent an increased risk for periprocedural bleeding nor be used as an indication for transfusion of FFP or clotting factor concentrates. Literature data on preprocedural coagulation testing for specific procedures are summarized below.

Angiography.—In a prospective study of 1,000 patients undergoing arteriography via common femoral artery access, Darcy et al (30) identified 85 patients with abnormal coagulation parameters, defined as a PT greater than 15 seconds (range, 15–20.8 seconds; normal, 13 seconds). Major bleeding, defined as a groin hematoma larger than 4 cm, was found in 1.2% (one of 85) of patients with abnormal coagulation parameters and 1.6% (15 of 915) of patients with normal coagulation parameters. Most procedures were performed with 5-F catheters (72%) or 6–7-F catheters (23%). There was, however, a correlation of a higher incidence of hematoma with a platelet count less than 100,000/μL (P = .002). The study concluded that, in the absence of an overt history of bleeding and an expected PT of less than 18 seconds, preprocedural testing with PT and activated PTT is not warranted (30).

Liver biopsy.—In a laparoscopic study, Ewe (31) was able to directly visualize the liver biopsy site for bleeding. He found that 4.3% of patients with a PT longer than 13.5 seconds bled for more than 12 minutes after biopsy, in comparison to 4.6% of patients with normal coagulation parameters. He was unable to draw any correlation between the degree of abnormality of preprocedural coagulation parameters and the length of observed bleeding.

Central venous catheter placement.—Fisher and Mutimer (32) evaluated 580 patients with an INR greater than 1.5 undergoing central venous catheterization. All procedures were performed with a 16- or 18-gauge needle. Most patients (83%) had a platelet count less than 150,000/μL. One patient (0.2%) had major bleeding due to inadvertent puncture of the carotid artery. The authors concluded that central venous access can be performed safely by experienced physicians in the presence of abnormal coagulation parameters. Other studies have supported these results (33,34).

Morado et al (35) reported a case series of 15 patients with hemophilia and inhibitors who underwent a total of 34 catheter insertions. The mean patient age was 8.8 years (range, 16 months to 39 years); all patients had factor VIII/IX inhibitors. Percatheter bleeding was seen in seven catheter insertions in six patients and required substantive treatment for several days.

Central Venous Catheter Removal

There is some controversy and lack of consensus over the management of patients undergoing the removal of tunneled catheters. There is no evidence of the value of pre-removal coagulation parameters or platelet count in the management of these patients. Stecker et al (36) reported a study of 180 patients with tunneled cuffed central venous catheters. Time to hemostasis was 5 minutes for 166 patients, with 14 patients requiring more than 5 minutes of manual compression at the insertion site (range, 10–35 minutes). Only one patient required more than 15 minutes of pressure. In the 14 pa-
patients with prolonged (>5 minutes) time to hemostasis, statistically significant factors included the use of antiplatelet agents, renal failure, the use of high-flow hemodialysis catheter, and operator experience. They concluded that pre-removal laboratory evaluation was not warranted and that platelet dysfunction was a more important factor than platelet number in prolonging time to hemostasis, but that the degree of prolongation was unlikely to be clinically relevant.

Nephrostomy Tube Placement

Martin et al (37) reported a series of 160 patients who underwent percutaneous nephrostomy tube placement by an experienced operator. One hundred fifty-three patients had normal coagulation tests; seven patients had an abnormal PT or activated PTT (mean PT, 13.9 seconds; mean activated PTT, 30.3 seconds). No patients in the study experienced bleeding complications associated with nephrostomy tube placement. The authors concluded that screening coagulation studies are not necessary in patients before nephrostomy tube placement (37,38).

PLATELET TRANSFUSIONS

Severe thrombocytopenia may result in an increased bleeding risk with imaging-guided interventions and open surgery, although the recommended threshold for platelet transfusion varies between procedures. As with the use of FFP, the literature data for platelet use are mostly from case series, retrospective case reviews, and consensus data (39–43). There are many etiologies of thrombocytopenia and significant variation in platelet function associated with patient comorbidities and medication use. The decision for platelet transfusion before percutaneous procedures, therefore, is based on multiple variables, including platelet count, etiology of thrombocytopenia, platelet function, type of procedure, operator expertise and experience, and concurrent coagulopathies and other co-morbidities.

Ewe (31) reported a series of 200 consecutive patients in whom liver biopsy was performed. Liver biopsy after percutaneous biopsy. Liver biopsy time and patient outcomes did not correlate with preprocedural coagulation parameters or platelet count. The author concluded that standard measures of evaluation of coagulation and platelet count were not useful in determining bleeding risk associated with percutaneous liver biopsy.

Shiffer et al (44) reported recommendations for platelet transfusions in cancer patients. With respect to periprocedural management, the authors concluded that, on the basis of several consensus statements, in the absence of coexisting coagulation abnormalities a platelet count of 40,000–50,000/μL is sufficient for the safe performance of major invasive procedures. The consensus statements included data from both major operative procedures such as laparotomy or craniotomy to more minor invasive procedures such as central line placement, transbronchial biopsy, and bone marrow biopsies. They noted that certain procedures, such as bone marrow aspiration and biopsy, are routinely performed safely at platelet levels of 20,000/μL or less. There were some data suggesting that coexisting coagulopathies resulted in higher periprocedural blood loss in the setting of thrombocytopenia. They noted the relative lack of studies evaluating the safety and efficacy of invasive procedures in thrombocytopenic patients. Posttransfusion platelet counts were strongly recommended in all patients receiving platelet transfusions before invasive procedures (44).

With respect to percutaneous transabdominal liver biopsy, McVay and Toy (45) reported an incidence of clinically significant bleeding of 3.4% in 291 consecutive patients with mild thrombocytopenia, as defined by platelet counts between 50,000 and 99,000/μL. There was no difference in bleeding incidence in comparison with patients with normal platelet counts. Underlying malignancy appeared to be an independent risk factor for bleeding. Wallace et al (46) reported a series of 50 patients with hematologic malignancies and moderate to severe thrombocytopenia who underwent transjugular liver biopsy. The mean preprocedure, pretransfusion platelet count was 17,000/μL. Patients received prophylactic platelet transfusions before the procedure, with posttransfusion platelet counts ranging from 5,000 to 105,000/μL (mean 38,000/μL). The platelet count was less than 30,000/μL in 24 patients. Patients received a mean of 11 units of platelets. No clinically significant bleeding was encountered. The authors concluded that a platelet count of 30,000/μL represents a safe level for transjugular liver biopsy (46).

Multiple studies have evaluated the risk of bleeding with the placement of central venous access devices in the setting of thrombocytopenia. In a prospective study of 105 patients, Ray and Shenoy (47) evaluated the effect of various levels of thrombocytopenia on the incidence of bleeding complications necessitating intervention. Patients were divided into three groups: (a) those with moderate thrombocytopenia (platelet count <50,000/μL), (b) those with mild thrombocytopenia (platelet count 50,000–100,000/μL), and (c) those with platelet counts greater than 100,000/μL. Patients in the first group received platelet transfusions during the procedure, although the mean increase in platelet count was only 11,500/μL. There were no significant bleeding complications requiring intervention in patients with thrombocytopenia (47). Other studies have shown similar results in patients with both thrombocytopenia and an elevated INR, with a low incidence of bleeding complications (1%–2%) and no deaths due to periprocedural bleeding complications (21).

Multiple studies have investigated the incidence of bleeding complications with lumbar puncture in the setting of thrombocytopenia. In 1974, Edelson et al (48) reported spinal subdural hematomas after lumbar puncture in eight patients with thrombocytopenia, five of whom had platelet counts of less than 20,000/μL. In 1982, Breuer et al (49) reported significant spinal subarachnoid hematomas in two of 13 patients with platelet counts less than 20,000/μL who did not undergo preprocedural platelet transfusion. None of the seven patients with this degree of thrombocytopenia who received platelet transfusions developed significant bleeding complications. In 2000, Howard et al (50) retrospectively reviewed the results of 4,309 lumbar punctures performed in 959 children with acute lymphocytic leukemia. Three hundred seventy-eight procedures were performed in patients with platelet counts of less
than 25,000/μL. There were no significant bleeding complications in any patients, although a higher incidence of traumatic lumbar puncture appeared to be associated with worsening thrombocytopenia. Vavricka et al (51) reported the results of 195 lumbar puncture procedures in 66 adult patients with acute leukemia. There were no significant bleeding complications, although the authors reported a statistically significant trend in the occurrence of traumatic procedures in patients with the lowest platelet counts. Each of the above authors recommended a threshold level of 20,000/μL for platelet transfusion before lumbar puncture, and most noted the effect of increased operator experience in reducing hemorrhagic complications and traumatic procedures.

Many of these authors have expressed the opinion that severe coagulopathies and severe thrombocytopenia should be corrected before percutaneous nephrostomy tube placement, although specific recommendations for threshold values vary or many times are not specified.

ANTIPLATELET AGENT MANAGEMENT

Antiplatelet therapy has been shown to be effective in patients with coronary artery disease. Due to the prevalence of coronary artery disease, antiplatelet agents are commonly on the list of medications of patients presenting for noncardiac image-guided procedures. Two of the most commonly prescribed medications include aspirin and clopidogrel (Plavix; Bristol-Myers Squibb). Fox et al (52) reported the results of the Clopidogrel in Unstable Angina to Prevent Recurrent Ischemic Events (CURE) trial, in which 12,562 patients with unstable angina were randomized to clopidogrel or placebo. Of the study patients undergoing bypass surgery (n = 1,015), the rates of life-threatening hemorrhage were 4.2% in the placebo group and 5.6% in the clopidogrel group (relative risk = 1.30, not significant). The authors reported an excess of seven patients requiring transfusion and a trend for four patients to experience a life-threatening hemorrhage in the clopidogrel group. They recommended stopping clopidogrel for 5 days before coronary artery bypass graft placement for nonemergent surgery (52). There have been reports of serious, and in one case, fatal bleeding following lumbar sympathetic block in patients undergoing lumbar block procedures (53). Hussain et al (54) reported a case control study of 40 patients undergoing endoscopic sphincterotomy. Most of their study patients who were receiving antiplatelet therapy were taking aspirin. The authors concluded that, after adjustment for elevated INR and cholangitis, antiplatelet agents do not increase the risk of clinically significant bleeding associated with endoscopic sphincterotomy.

Both clopidogrel and aspirin result in irreversible platelet inhibition. In patients with normal bone marrow function and reserve, the platelet lifespan is approximately 10 days. Taking into account variabilities in drug clearance, withholding antiplatelet agents for 5 days will, therefore, result in approximately 30%–50% of platelets at the time of the procedure to have normal function.

Nonsteroidal anti-inflammatory drugs (NSAIDs) can inhibit platelet aggregation. The effect of NSAIDs on platelet aggregation, unlike aspirin, is reversible and will decay along with clearance of drug levels from the circulation. In general, NSAIDs do not cause significant bleeding problems except in patients with existing coagulopathies such as hemophilia, von Willebrand disease, or severe thrombocytopenia. Paradoxically, NSAIDs tend to diminish the antiplatelet effect of aspirin when given concomitantly and, therefore, should not be given to patients receiving aspirin therapy for cardiovascular disease (55,56).

RECOMMENDATIONS FOR PREPROCEDURE TESTING AND MANAGEMENT

Assessment and preparation of the patient before imaging-guided procedures will vary according to the procedure to be performed in conjunction with a comprehensive assessment of the patient’s comorbidities. Although image guidance is likely to make minimally invasive procedures more accurate, for example, in their ability to target lesions or to put effector devices such as needles or catheters in optimal position, by their very nature these procedures preclude the operator from direct visualization of postprocedure bleeding. The lack of available randomized, controlled studies specific to imaging-guided percutaneous procedures has resulted in considerable variety in clinical practice. Furthermore, it is doubtless that one can extrapolate the results from open surgical procedures to minimally invasive procedures due to the aforementioned separation of the operator from direct assessment of bleeding (and the associated ability to control it) at the site of the procedure.

Recommendations for patient evaluation and general indications for the use of blood products and other hemostatic agents are outlined in Tables 2–4. Where reliable data was lacking, recommendations were derived by Delphi consensus of a panel of expert practitioners. Tables 2–4 represent the results of the Delphi consensus panel, which were derived for the management of a patient with a single hemostatic defect. A total of 18 Certificate of Added Qualification–certified interventional radiologists participated in a four-round Delphi process. Although representative procedures were placed into one of three categories of risk, as outlined in Tables 2–4, the panel believed there was significant potential variability in risk from procedure to procedure within each category, depending on the individual patient comorbidities and possible multiple concomitant hemostatic defects. It must be stressed, therefore, that specific assessment of bleeding risk and considerations for the use of blood products or other hemostatic agents must be individualized to the patient at the total discretion of the performing physician, who must, at the time of the procedure, make clinical decisions on the basis of an often complex array of patient variables, co-morbidities, and concomitant hemostatic defects. With respect to the categories in Tables 2–4, any individual procedure might possibly be treated at a higher risk level, depending on these individual patient factors. In addition, for the purposes of this document, the Delphi consensus panel treated the procedures as elective, with a single hemostatic defect. Emergency indications, multiple concomitant hemostatic defects, and the use of topical or intravascular/perivascular closure devices were not specifically addressed.
Emergency or highly urgent procedures, where the risk of procedural delay may outweigh the potential hemorrhagic risk, may not afford the time for equivalent correction of hemostatic defects as may be achieved in elective procedures. The physician must take into account pathophyslogic, psychosocial, medicolegal, and religious variables in coming to an overall assessment of the patient. For example, periprocedural management for percutaneous liver biopsy may vary significantly between one patient with an INR of 1.7 with no co-morbidities and a second patient with an INR of 1.7 and concomitant renal failure and cirrhosis. Although the recommendations given in the tables attempt to divide the spectrum of imaging-guided procedures into categories of risk, only the practicing physician can adequately assess risk at the point of care and, therefore, the potential need for treatment in the individual patient.

Because there is no evidence to support the use of bleeding times before...
CONCLUSION

In this document, we attempt to summarize some of the available literature regarding periprocedural surveillance and management of hemostatic defects in patients undergoing percutaneous, imaging-guided procedures. Because of the lack of randomized, controlled studies or other high-level evidence on this topic, a Delphi panel of experts constructed a set of consensus guidelines to hopefully serve as a reference for the practicing interventionalist in constructing their individual practice guidelines. Although it is likely that individual practice parameters will vary from this document, each practitioner should monitor outcomes and look for trends, both positive and negative, that may suggest modifications or adjustments to these parameters. Outlining bleeding complication rates for specific procedures is beyond the scope of this document and, in many cases, may be difficult or impossible to accurately accomplish due to the lack of high-level data. Where external benchmarks are not available, practitioners may choose to benchmark against their own historical data as part of an overall quality improvement program.

The periprocedural management of patients undergoing imaging-guided procedures is a continually evolving paradigm. Local factors such as procedure types and patient selection will influence management. In addition, advances in technology and image guidance may have a significant effect on periprocedural management. The use of closure devices, smaller gauge catheters and biopsy devices, adjunct hemostatic measures such as postbiopsy tract plugging, and color flow ultrasonography or computed tomographic fluoroscopy all have the potential to effect the incidence of periprocedural bleeding complications, although further studies will be needed to accurately assess their impact.

Acknowledgments: Dr. Patrick C. Malloy authored the first draft of this document and served as topic leader during the subsequent revisions of the draft. Dr. Clement J. Grassi was chair of the SIR Standards of Practice Committee during the development of this document. Dr. Sanjoy Kundu is now chair. Dr. John F. Cardella is Councilor of the SIR Standards Division. Other members of the Standards of Practice Committee and SIR who participated in the development of this clinical practice guideline are (listed alphabetically): John “Fritz” Angle, MD, Ganesh Annamalai, MD, Curtis W. Bakal, MD, Stephen Balter, PhD, Daniel B. Brown, MD, Danny Chan, MD, Timothy W.I. Clark, MD, MSc, Bairbre L. Connolly, MD, Horacio D’Agostino, MD, Brian D. Davison, MD, Peter Drescher, MD, S. Nahum Goldberg, MD, Manraj K.S. Heran, MD, Maxim Itkin, MD, Sanjeeva P. Kalva, MD, Arshad Ahmed Khan, MD, Neil M. Khilnani, MD, Curtis A. Lewis, MD, MBA, JD, J. Kevin McGraw, MD, Tim McSorley, BS, RN, CRN, Philip M. Meyers, MD, Steven F. Millward, MD, Charles A. Owens, MD, Anne C. Roberts, MD, Steven C. Rose, MD, Tarun Sabharwal, MD, Cindy Kaiser Saiter, NP, Nasir H. Siddiqu, MD, LeAnn Stokes, MD, Timothy L. Swan, MD, Patricia E. Thorpe, MD, Richard Towbin, MD, Aradhana Venkatesan, MD, Louis K. Wagner, PhD, Prof, Michael J. Wallace, MD, Brent N. Wiechmann, MD, Joan Wojak, MD, and Kenneth S. Rholl, MD.

APPENDIX: SIR STANDARDS OF PRACTICE COMMITTEE
Consensus Methodology

Consensus guidelines reported in this document were obtained by using a modified Delphi technique (1). Eighteen CAQ-certified members of the SIR Standards of Practice Committee participated through four rounds of the Delphi to reach consensus as reported.

References

Table 4: Procedures with Significant Bleeding Risk, Difficult to Detect or Control

<table>
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<th>Management</th>
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<td>INR: Correct above 1.5 (95% consensus)</td>
</tr>
<tr>
<td>Transjugular intrahepatic porto-systemic shunt</td>
<td>Activated PTT: Routinely recommended in patients receiving intravenous unfractionated heparin infusion. No consensus on patients not receiving heparin</td>
<td>Activated PTT: Stop or reverse heparin for values &gt;1.5 times control</td>
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<tr>
<td>Nonvascular</td>
<td>Platelet count: Routinely recommended</td>
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<td>Renal biopsy</td>
<td>Hematocrit: Routinely recommended</td>
<td>Hematocrit: No recommended threshold for transfusion</td>
</tr>
<tr>
<td>Biliary interventions (new tract)</td>
<td></td>
<td>Plavix: Withhold for 5 d before procedure</td>
</tr>
<tr>
<td>Nephrostomy tube placement</td>
<td></td>
<td>Aspirin: Withhold for 5 d</td>
</tr>
<tr>
<td>Radiofrequency ablation: complex</td>
<td></td>
<td>Fractionated heparin: withhold for 24 h or up to two doses</td>
</tr>
</tbody>
</table>

There was an 80% consensus on each of these recommendations unless otherwise stated. The management recommendations for each coagulation defect and drug assume that no other coagulation defect is present and that no other drug that might affect coagulation status has been administered.

SIR DISCLAIMER

The clinical practice guidelines of the Society of Interventional Radiology attempt to define practice principles that generally should assist in producing high quality medical care. These guidelines are voluntary and are not rules. A physician may deviate from these guidelines, as necessitated by the individual patient and available resources. These practice guidelines should not be deemed inclusive of all proper methods of care or exclusive of other methods of care that are reasonably directed towards the same result. Other sources of information may be used in conjunction with these principles to produce a process leading to high quality medical care. The ultimate judgment regarding the conduct of any specific procedure or course of management must be made by the physician, who should consider all circumstances relevant to the individual clinical situation. Adherence to the SIR Quality Improvement Program will not assure a successful outcome in every situation. It is prudent to document the rationale for any deviation from the suggested practice guidelines in the department policies and procedure manual or in the patient’s medical record.