

Bleeding Avoidance Strategies

Experts offer their experience with acute coronary syndrome protocol and how they approach access sites and manage bleeding events.

PANELISTS



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*Disclosures: Research grants and honoraria
from Haemonetics; Medtronic, Inc.; Amgen;
Duke Clinical Research Institute; Merck;
National Institutes of Health; Coramed
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What is your institution's acute coronary syndrome (ACS) protocol, and what specific strategies do you recommend from your experience?

Dr. Espinoza: At our institution, we divide our treatment strategies based on the type of ACS. We break it down into the three components, the unstable angina, ST-segment elevation myocardial infarction (STEMI), and non-STEMI (NSTEMI) patient populations and have a slightly different algorithm for each modality.

For the unstable angina population who are not troponin positive, we tend to be a little bit more patient and direct treatment based on the patient's clinical presentation and overall risk analysis. We will usually initiate anticoagulation with unfractionated heparin and aspirin pending anatomic assessment.

With the NSTEMI population, we tend to be more aggressive. When you look at the outcomes for NSTEMI and STEMI patients and you follow them out to 1 year,

both patient populations have a substantially increased risk of recurrent ischemic events. We try to use risk scores (ie, thrombolysis in myocardial infarction [TIMI] risk score) to ascertain not only the patient's in-house score but also what the 30-day and subsequent event risk is going to be over the course of time, and then we use treatment strategies to implement appropriate pharmacologic therapy.

We are big advocates for not only an early treatment strategy, but also for an early invasive strategy. Based on the patient's age, sex, other comorbid conditions, and the calculated score from the risk calculators, we tend to give pharmacotherapy up front. I do differ with some of my colleagues at my institution who tend to prefer to wait. Data are conflicting regarding upstream pharmacology. However, the most contemporary literature suggests about a 10% risk of ACS patients requiring urgent bypass surgery. If patients undergo open heart surgery, some colleagues don't want to extend the patients' hospital stay or predispose them to a possible increase in bleeding. I would argue that leaves 90% of ACS patients untreated with drugs we know reduce ischemic events and mortality.

STEMI patients are a unique patient population but obviously require very aggressive therapy right out of the gate. At our institution, we have an implemented field contact administration of dual antiplatelet therapy when our medical rescue squads essentially touch the patient for the first time. Once it is established that the patient has a STEMI, they are given dual antiplatelet therapy orally at the point of contact before they even arrive in the emergency department (ED). We have a treatment algorithm of either going straight to the cath lab or a brief, temporary stop in the ED and then to the cath lab for treatment. We have had no issues with regard to bradyarrhythmia, dyspnea, acute bleeding, or any other potential concern about this strategy, which is consistent with the ATLANTIC trial results. Even though no clear endpoint benefit is derived, the simple truth is that in the United States, when a patient arrives in the ED, the first thing they are given is dual antiplatelet therapy. We have simply moved the ED into the field.

Dr. Gurbel: Let's address STEMI. First, there are strict time guidelines. For STEMI, we have an emergency call team with strict time limits for staff arrival. If staff cannot make it to the hospital in that time, they must work out coverage with one of their colleagues to make the time cutoff. It is the same for the doctors.

Second, the ED staff and physicians have been educated in administration of antithrombotic therapy.

Patients with STEMI get up-front treatment with aspirin and an oral P2Y12 inhibitor, and the guidelines are followed regarding anticoagulant therapy. Heparin is the anticoagulant. The specific P2Y12 inhibitor is left to the discretion of the treating physician. However, prasugrel and ticagrelor are preferred over clopidogrel. The real aim in these patients is early reperfusion, unless there's a contraindication to emergent catheterization. The duration of time from the call to the time that the catheter is actually in the coronary artery is scrutinized.

For NSTEMI, patients are treated with an anticoagulant, usually heparin and aspirin. The decision to treat with a P2Y12 inhibitor depends on the likelihood of three-vessel coronary artery disease, the potential for needing a coronary artery bypass graft, and the duration of time until catheterization. In the setting of electrical or hemodynamic instability, these patients will go immediately to the cath lab.

Dr. Rao: We have had an ACS protocol for more than 25 years now and it's changed to keep up with the latest data. For our ACS protocol, we separate it out into baseline presenting electrocardiogram (ECG) and STEMI versus NSTEMI. The STEMI population is focused on activating the cath lab, so antiplatelet and antithrombin therapy are recommended. But the real focus is on getting the patient to the cath lab for primary percutaneous coronary intervention (PCI) as quickly as possible.

Like many other institutions, we have a single page that activates the cath lab team, and the patient is quickly brought to the cath lab. Emergency personnel have all been trained to interpret ECGs, and patients can actually bypass the ED and come directly to the cath lab.

The NSTEMI population involves initial therapy to stabilize the patient with a pathway to taking the patient directly to the cath lab if they have refractory chest pain or are clinically unstable. Medical therapy would include antiplatelet therapy (our first line is the newer P2Y12 inhibitors such as ticagrelor); antithrombin therapy, which is primarily unfractionated heparin; and then usually early invasive risk stratification or catheterization within the first 24 hours. Our protocol is very explicit in terms of what we as a group have decided we want to do and how we want to treat ACS patients.

Dr. Yakubov: Our institution's ACS strategy is typically anticoagulation with heparin. We usually use heparin; however, for the patients who become tropo-

nin positive, we may add tirofiban if they are going to be delayed from getting into the cath lab.

Patients who are sent to the cath lab quickly, such as those with STEMI, are treated with oral ticagrelor or prasugrel as soon as possible, if there are no contraindications. The intravenous glycoprotein (GP) IIb/IIIa inhibitors are used if there is inadequate time for oral agents to work or if there is excessive thrombus burden in the cath lab.

Is there consensus among the treating physicians at your facility on these strategies and on this protocol?

Dr. Rao: Yes, there is. I think our ACS protocol, while it's fairly prescriptive, also allows for a little bit of wiggle room in terms of the clinical decision making for the physician who is actually taking care of the patient. We have meetings to discuss changes based on new data, and that's usually vetted across the invasive and noninvasive cardiologists.

Dr. Yakubov: There's relative consensus. There might be individual variability, but there is not much difference. We'd prefer to use the faster-acting oral antiplatelet therapy for patients who are going to the cath lab quickly. We have similar strategies when we're using the intravenous GP IIb/IIIa inhibitors. In most instances, heparin is the antithrombin strategy.

Dr. Gurbel: In terms of speed, yes, in general there is consensus. Most of the cut points for time are mandated by a consensus need. We still leave it up to the treating doctor which antithrombotic or antiplatelet agent to use. For example, some hospitals use ticagrelor and other hospitals use prasugrel, but we do not specify the type of P2Y12 inhibitor in a protocol. Intravenous GP IIb/IIIa inhibitor therapy is often used when there is inadequate time for a full pharmacodynamic effect of oral P2Y12 inhibitors or when there is thrombus burden. We do not yet have cangrelor on the formulary, and since it has not been compared with potent oral P2Y12 inhibitors, its efficacy as compared with the latter is unknown. Finally, GP IIb/IIIa inhibitors are the most potent platelet inhibitors, so when there is concern regarding thrombus, the totality of the evidence, in my opinion, would favor use of these agents.

Dr. Espinoza: It depends on which aspects of the program are being referred to. When you look at some of those aforementioned clinical diagnoses and their intended pathways, the ED physicians at our medical center seem to be very much in line with these specific

clinical approaches. There are other physicians within the system who are not necessarily as aggressive and who tend to be more conservative both with timing and choice of agent. Clopidogrel is an agent still used frequently. In fact, one may argue that it could be used to treat an uncomplicated unstable angina patient. The use of clopidogrel in the STEMI and NSTEMI setting ignores the scientific evidence and current best practices. I think there is tremendous consensus within the STEMI patient population. When you expand outside of the STEMI patient population, there's more wiggle room for physician preference.

With the inherent procedural risk of PCI now so low and bleeding becoming the most common risk for patients undergoing PCI, how has this paradigm change in risk had an impact on your clinical practice?

Dr. Yakubov: One of the biggest factors that has decreased bleeding is switching to the radial approach for our procedures. Since switching, bleeding complications have decreased tremendously. This decrease has allowed us to use more potent agents with more regularity because there is just not as much bleeding because of the access approach.

Dr. Gurbel: I'm not so sure in the real world the inherent risk of PCI is "so low." Data from the registries and clinical trials would suggest that thrombotic complications are alive and well in 2017. Complication rates in the clinical trials are always lower than in the real world. One can get a distorted impression from what you see in the results in a clinical trial versus what is observed in the real world.

But, I also agree that bleeding is a persistent issue. An important factor we are observing is the performance of more complex interventions in elderly patients with advanced disease. The frequency of these patients in the overall PCI population is growing. As the population gets older, the bleed risk of patients undergoing PCI increases. As the age of the patient population increases and the overall level of aggressiveness in interventional cardiology advances, we will see more complications and one will be bleeding. Therefore, I wouldn't say that serious bleeding is the biggest problem we confront, I would say bleeding and the procedural risk of PCI both remain problems.

Dr. Espinoza: That is a great question simply because I remember my days training as a fellow in the 1990s when we were using massive doses of heparin and combining it with GP IIb/IIIa inhibitors. We were using large-

er sheath sizes and larger guiding catheters. Inherently, the bleeding risk was fairly excessive, and it was almost commonplace and considered an acceptable outcome as long as you had an excellent procedural interventional outcome. It was almost an assumed occurrence that patients would have some form of bleeding event, almost invariably access site related, but even at times, conditions as uncommon as alveolar hemorrhage.

Over the past 15 to 20 years, there has been a tremendous evolution in approaches—everything from technology to anticoagulation strategies, marginalization of the dosages, and going to bolus as opposed to continuous prolonged infusions of GP IIb/IIIa inhibitors. Cangrelor seems to be an attractive “on-and-off-switch” agent, but has not been widely assessed to justify the costs.

We’ve appreciated the fact that over the last 15 years, there has been a tremendous reduction in periprocedural events, in particular with elective PCI patients. When you move into the ACS patient population, those patients are inherently at higher risk for bleeding. In general, we have moved into what’s been commonly coined as a bleeding avoidance strategy approach, trying to minimize the most common complications we still encounter both access and nonaccess site related.

Dr. Rao: One obvious change is that we have adopted the radial approach for most of our patients, including for STEMIs. That’s been the biggest change. Another is that we’ve always had a robust involvement of cardiovascular pharmacists in our day-to-day practice, and now that we have electronic health records, we have a lot of safeguards in place to ensure that drugs are appropriately dosed. In addition, drugs that are contraindicated for specific patient situations (eg, the use of prasugrel for previous stroke, transient ischemic attack, low body weight) are flagged in our electronic health records and allow us to minimize bleeding risk.

How do you attempt to reduce the bleeding risk?

Dr. Gurbel: We use the activated clotting time (ACT) to guide the heparin dosing, so we’re very careful with heparin administration. We use an algorithm. We dose the patient according to the recommendations. Many of our interventionists use radial access. These practices have cut back on the bleeding issue.

We are aware of the higher risk of recurrent ischemic events, including stent thrombosis, which has been shown in studies employing bivalirudin as the anticoagulant. Data suggest that use of this therapy instead of heparin is associated with a higher thrombotic event

rate. That’s one of the reasons why the preference for anticoagulant is moving away from bivalirudin and back to heparin.

I think patients who have a high bleed risk and low body weight, such as older women in whom the inherent bleeding risk is higher, may be good candidates for bivalirudin. But the majority of patients in our institution are treated with heparin.

Dr. Yakubov: Bleeding is always one of our biggest concerns; we continually encourage everyone to access patients from the radial approach. Although this isn’t uniform with every physician in our hospital, radial access is done now in a majority of cases. I think that’s the biggest change to decrease bleeding strategies.

Typically, we use heparin, which is associated with higher bleeding rates as opposed to bivalirudin; however, the radial approach has pretty much obviated that increased bleeding risk. There has been a trend in lower ischemic events overall with these strategies.

Dr. Espinoza: Depending on who you ask, you are going to get a wide variety of opinions, most of which are experience based. Amazingly enough, in interventional cardiology, simply because of the number of permutations of possible strategies you can use, we have not honed in on a singular strategy that fits all patients.

My clinical practice changed dramatically back in 2008 at the Transcatheter Cardiovascular Therapeutics meeting in San Francisco. I remember attending Dr. Jeffrey Popma’s transradial session, which was one of the side room sessions, but it was standing room only. I left there, literally picked up the phone, and called my cath lab and told them that we’re immediately starting the transradial approach simply because I realized all the potential benefits, one of which is clearly bleeding.

It would be difficult for any high-volume interventional operator to suggest that, whether evidence based or not, there is not a lesser bleeding risk using a transradial approach compared with a transfemoral approach. The absence of proof is not the proof of absence. I have used the transradial approach as my default strategy for patients, with transfemoral as the fallback. There are myths that it potentially affects door-to-balloon times in STEMI or that there is no tremendous benefit. Again, I think anyone who has morphed their clinical practice to the transradial arena will tell you that their patients’ bleeding complications have been reduced dramatically, if not altogether, at least from an access site standpoint.

Another aspect would be to use modified doses of anticoagulant therapy. The guidelines would tell you if you are not using a GP IIb/IIIa inhibitor, you want the

ACT to be somewhere around 250 and 300 seconds, whereas if you are using a GP IIb/IIIa inhibitor, ACT should be between 200 and 250 seconds. My preference is to use a much lower dose of heparin initially and complete the procedure as quickly as possible. Of course, sometimes procedures take longer than you anticipate, but again maintaining the anticoagulation level, specifically with heparin, seems to be an effective way of avoiding potential complications not only during but also after the procedure.

In addition, judicious and very selective use of GP IIb/IIIa inhibitors can help reduce bleeding risk. We tend to use them more as a bolus for patients who have not received up-front oral antiplatelet therapy. When using a transfemoral approach, you focus on using some other modalities to access the vessel, such as fluoroscopic visualization to identify where the femoral head is. Many operators use ultrasound-guided punctures, which is a simple thing to do, especially for interventionists who do peripheral work and use ultrasound on a regular basis. Micropuncture technique is an excellent technique to mitigate risk of accessory branch laceration and minimize the initial puncture diameter. We like pulling sheaths early and try not to leave them in for extended periods. Some advocate for vascular closure devices, but unfortunately, data have not suggested these devices improve bleeding or access site complications. However, their use enables a patient to ambulate more quickly, but some data suggest that the use of vascular closure devices is associated with higher bleeding.

The most compelling way to minimize bleeding falls into the lap of the physician during the procedure and is when the patient is vetted in advance of the procedure using risk calculators. I've become a big fan of utilizing risk calculators. There are a host of tools available, and while not perfect, I think using them and getting a better clinical assessment is an advisable strategy.

Overall, bleeding avoidance strategies are important. I don't think there is one calculator that is the be-all and end-all. But if you combine all those strategies, you can use a fairly intuitive approach to attempt to minimize bleeding complications periprocedurally.

What are your pharmacologic choices to mitigate bleeding risk?

Dr. Gurbel: It's patient dependent. We use GP IIb/IIIa inhibitors for big thrombus burden and also for STEMI until the full pharmacodynamic effect of the oral P2Y12 inhibitor is reached. I am a proponent of shortening the duration of GP IIb/IIIa infusion enough to allow for

the full pharmacodynamic effect of the P2Y12 inhibitor to occur. With the new P2Y12 inhibitors (ie, ticagrelor and prasugrel) that most of the interventional cardiologists at my institution use, this means a very short duration of GP IIb/IIIa inhibitor therapy. We mostly use low-molecular-weight GP IIb/IIIa inhibitors that have a short half-life, such as eptifibatid and tirofiban when it is back on the formulary.

Appropriate dosing of heparin and monitoring the ACT, using short courses of GP IIb/IIIa inhibitors, using bivalirudin selectively in patients with high bleed risk, and the use of radial artery access are all strategies to mitigate bleeding risk.

Dr. Rao: First and foremost, appropriate dosing and our primary approach for pharmacotherapy is the number one thing that has evolved in our practice over the past few years. We have about 50/50 use of bivalirudin versus heparin. Some operators use bivalirudin, and we tend to reserve that for transfemoral cases. For radial cases, we're still using unfractionated heparin, although some operators will use bivalirudin in this case as well.

I'd say that our pharmacotherapy choice depends largely on our access site choice, but the one strategy that is important regardless of access site choice is appropriate dosing.

Dr. Espinoza: We choose based on bleeding risk, while being thoughtful to issues such as renal impairment, prior history of bleeding, age, and gender, among other factors.

Some institutions have moved toward cangrelor for patients, but again, it is not substantiated in any large clinical trials, especially in STEMI patients. It's not the choice of agent but rather the classification of the patient going into the procedure and then using the appropriate doses of anticoagulation and avoiding overdosing that will likely be most effective.

Dr. Yakubov: For patients who are at high risk of bleeding, we may use bivalirudin rather than heparin in that particular instance. We may shy away from using additional agents unless absolutely necessary. Intravenous GP IIb/IIIa inhibitors would be used only if necessary, such as in patients with visible thrombus burden. I don't believe there's a significant difference, especially for early bleeding risk with the more potent P2Y12 receptor blockers compared with clopidogrel alone, especially when bolus infusion alone is used. We have not yet implemented a strategy for cangrelor.

What new or upcoming drugs do you anticipate will have an impact on your bleeding protocols, if any?

Dr. Rao: The two agents we are considering integrating into the protocols would be cangrelor and tirofiban. Tirofiban is a GP IIb/IIIa inhibitor. It's been around for a long time—there have been some data on shortened duration of infusion, and it's a very cost-effective GP IIb/IIIa inhibitor. We are in active discussion to figure out where that fits in. Similarly, with cangrelor we're trying to figure out exactly where that is going to fit into our ACS protocol. Those are the two drugs that are going to influence the change in our ACS protocol over the next year or so.

Dr. Gurbel: I do not see any drugs with a major impact. However, I do see that the strategy of short-course GP IIb/IIIa inhibitors may have an impact, enough to allow adequate pharmacodynamic effect of an oral P2Y₁₂ inhibitor. With respect to long-term bleeding, there are studies evaluating whether aspirin is necessary in the presence of potent oral P2Y₁₂ blockers. Omission of aspirin may reduce bleeding. Finally, I can tell you about a drug that we're working on for interventional procedures. It is a parenteral platelet thrombin receptor blocker. It has a rapid onset and rapid offset of effect. The drug is called PZ128, and it's in a phase 2 clinical trial right now at my institution and at two other institutions in the United States. But it's really new and hasn't hit the headlines yet because it's early in its development.

Cangrelor has not been vetted against potent oral P2Y₁₂ inhibitors. Most importantly, it has not been vetted against these agents in high-risk patients. It hasn't been compared to GP IIb/IIIa inhibitors. The jury is still out on cangrelor, particularly when you look at the speed at which oral P2Y₁₂ inhibitors achieve their pharmacologic maximum effect and the totality of evidence supporting the antithrombotic benefits of GP IIb/IIIa inhibitors in high-risk situations.

If I had a massive thrombus burden, I don't know if I'd rely on cangrelor to be my savior. We have a lot of data that have preceded this discussion on the efficacy of GP IIb/IIIa inhibitors to bail out patients and to disaggregate platelets. The totality of the evidence, in my opinion, would favor a GP IIb/IIIa inhibitor over cangrelor when there is a real concern for an ongoing major thrombotic problem.

Dr. Espinoza: There is nothing I am involved with from a clinical research perspective. I do know there are some other antithrombin drugs in the works that look to be much more targeted. I would suspect in the future, or at least hope, that we can find some pharmacologic

agents that mitigate bleeding but maintain ischemic risk reduction benefits as well.

Dr. Yakubov: We haven't been a big site for cangrelor, but as the others have mentioned, it's being considered at other institutions. We just haven't gone that route yet.

How do access sites factor into your protocol with regard to access site-related and non-access site-related bleeding?

Dr. Yakubov: My impression is that most of the bleeding that occurs at the time of intervention for patients with ACS is typically related to the access site. Now, the clinical trials don't always bear that out. The only real control I have is around access site bleeding. Therefore, the choice in our lab by almost every physician is to do the procedure through the radial approach in patients at high risk for bleeding. We have found this has tremendously decreased our access site complications, not only for bleeding, but also for pseudoaneurysm development and improved patient comfort. By far, the patients would prefer the radial approach rather than the femoral artery approach. However, if patients are accessed via the femoral approach, micropuncture techniques are always used.

Dr. Espinoza: There is always the potential where an arteriotomy is created, especially in a patient who is either currently anticoagulated or going to be anticoagulated. Mitigation using the transradial approach as a default strategy is imperative. If I were continuing to use the transfemoral approach as my default strategy, I would almost invariably go to either a fluoroscopic ultrasound-guided or a micropuncture needle technique to minimize hematomas, pseudoaneurysms, or retroperitoneal bleeds, and all the other complications that arise from using that access site approach.

As far as nonaccess site-related bleeding, that's where the risk score calculators for a bleeding avoidance strategy come into play. For example, if you have a high-risk patient who experienced a STEMI, was in cardiogenic shock, had a hypothermia protocol, was in the coronary care unit being cared for after the procedure, and is in ventilator-dependent respiratory failure, the use of intravenous proton pump inhibitor therapy is the cornerstone of treatment. Then, the patient would be switched to oral antacid therapy to prevent gastric erosion, gastric ulceration, and gastrointestinal bleeding.

We cannot do a lot about spontaneous hemorrhage; those tend to be more bad luck than anything else. With respect to nonaccess site bleeding, which tends to be the predominance of bleeding that we experience,

good diligence about reducing gastrointestinal bleeding is probably the most compelling issue that we can focus on with antacid therapy.

Dr. Gurbel: Data from clinical trials can give us some help here. A lot of the bleeding that occurs comes from outside of the access site, but that's particularly true with GP IIb/IIIa inhibitors. If you go from the radial site, you may have a bleeding issue from the nonvascular access site. Still, in my experience, most of the bleeding events occur from the access site. Having said that, we do not include access site in a protocol.

Dr. Rao: We're not trying to protocolize any specific access site. We still leave it to the discretion of the operator to choose, primarily because some operators are very comfortable with radial access, while others may still prefer femoral access. In that sense, our adoption of radial access is continuing to increase. For those who are very comfortable with radial access, we'll use a radial-first approach. Some of my colleagues use femoral approach, but they are using strategies to reduce femoral access site complications, such as micropuncture and ultrasound-guided access.

How does it interact with pharmacotherapy and access site bleeding? We're very cognizant of access site bleeding, which is why even our operators using femoral access are using techniques to minimize complications, like micropuncture and ultrasound.

With respect to nonaccess site bleeding, we have stepped away from using routine postprocedure GP IIb/IIIa inhibitors, and that's probably true nationwide. That has had a huge impact. We also try to avoid the use of potent P2Y₁₂ inhibitors in patients who may be at higher risk for bleeding, such as those who are older or who have very low body weight.

What is important to consider in 2017?

Dr. Rao: The biggest thing that warrants discussion is that it's one thing to develop an ACS protocol, but it's another to continue to evolve it. That's where the challenge comes in. It's very difficult to get people on the same page to develop an initial protocol, but then you must keep addressing it as new information comes out. We've empowered a few people to say they're going to own the ACS protocol, that they're going to be the ones to continue to change it and keep it up to date. By empowering them, and it's not just physicians but also ancillary personnel such as practice providers and nurses, it allows for a team-based approach so someone is constantly looking out to make sure that the ACS protocol is up to date. That's an important aspect of developing pathways.

Dr. Gurbel: I think we need more data. I know a lot of people are excited about the radial artery approach for ACS patients, but we still don't have an adequately powered clinical trial to vet that as a superior method of access in a high-risk ACS population. I honestly do not buy into all the proposed benefits of radial access versus femoral access. I think there is definitely a learning curve. Once you've decided to go with the radial artery and you have an issue with access, particularly in STEMI, then you have to switch and go through the femoral artery anyway and you're delaying reperfusion in the patient. When you are talking about putting the patient on a GP IIb/IIIa inhibitor, and you've already entered the blood vessel, then you can have bleeding from the arm. I think that the patient should be carefully selected for the radial approach in high-risk scenarios such as STEMI where delay to reperfusion can have disastrous effects.

Dr. Yakubov: The only other consideration we've had is the more frequent use of preclose techniques for implantation of cardiac assist or balloon pump devices to try to decrease the risk of bleeding if we're using those devices on high-risk patients.

Dr. Espinoza: Bleeding still accounts for a significant amount of morbidity and associated mortality. There should be a greater shift, especially in the era of quality and value-based care, about having institutions focus on bleeding avoidance strategies using calculators and preprocedural assessment of patients and their inherent risks. I truly think radial access is very simple and should be a mandated default strategy for most patients, but clearly not all.

In general, there should be an adaptation, either institutionally or nationally, of some form of best practices. We're always going to have differences in the choice of agents, stents, and guides, but I think there should be either a societal or larger mandate for a best practice that is utilized on an institution-wide basis to reduce bleeding events for no other reason than to augment patient outcomes and ensure that we avoid those complications.

I think there's a lot of potential. There is a tremendous amount of difference from operator to operator, institution to institution, but there are some fundamental principles that we as an interventional society need to begin to implement and accept as global strategies for the benefit of our patients. Of course, this comes with the understanding that to do so we need more clinical trial evidence to support the bleeding avoidance strategies. ■