Literature has supported the efficacy of venous stenting, and several published studies, including recent investigational device exemption studies, suggest excellent long-term outcomes in patients with venous outflow obstruction. Nonetheless, particularly in the context of chronic postthrombotic patients, there is a significant loss of patency after stenting. This is not correctable in some patients, and therefore, “getting it right the first time” is important.

Patency loss after stenting may not be considered a complication—indeed, in our practice, many patients with extensive disease consent to the likelihood of multiple interventions, especially if there is significant common femoral vein (CFV) disease. Despite this, the ultimate aim of intervention is to perform a procedure once, get a successful result, and return the patient to normal life.

Failure of stenting can be broadly divided into three categories: technical, hematologic, and flow-related. It is the perfect union of all three aspects that results in excellent outcomes (Figure 1), and when considering why a “good” stent has gone “bad,” this serves as a framework to allow for correction and ultimately achieve the goal of long-term patency. The principle of stenting in the nitinol era is to recognize that the “rules” established with the use of Wallstent (Boston Scientific Corporation) need to be unlearned.

**Figure 1.** Factors affecting stent outcome. This presents a schema for considering why stents may have failed and factors that may need to be addressed.

**TECHNICAL REASONS FOR FAILURE**

Technical failure is likely the single largest cause of early stent failure. In the majority of cases, failure is due to inadequate treatment of inflow by not extending the stent caudally enough to manage disease in the CFV or not extending the stents cranially enough to treat outflow disease (Figure 2). There are several consistent bony landmarks for ensuring that stents are placed correctly.
The cranial landmark for the confluence of the iliac veins, and therefore a marker for the position of the proximal extent of stenting in left-sided disease, is almost invariably the spinous process when the spine is viewed anteroposteriorly. To ensure accuracy, it is important to correct for rotation. The outer border of the spinal body serves as a good marker for the contralateral inferior vena cava wall. Ensuring the stent extends beyond the left edge of the spinous process when viewed anteroposteriorly is a good indicator that the stent is beyond the compression point of the left common iliac artery. Similarly, the lesser trochanter serves as a reliable landmark for the confluence of the profunda femoral vein. The lesser trochanter is a reliable landmark for the typical confluence point.

Fracture
Stent fracture may complicate extension of the stents beyond the inguinal ligament into the CFV. Typical fractures are approximately 1 cm beyond the line of the ligament and correspond to the head of the femur. Many hypotheses have been suggested regarding the cause of fracture, including compression by the ligament or against the bony prominence of the pubis. Fracture may complicate extension of the stents beyond the inguinal ligament into the CFV. Typical fractures are approximately 1 cm beyond the line of the ligament and correspond to the head of the femur. Many hypotheses have been suggested regarding the cause of fracture, including compression by the ligament or against the bony prominence of the pubis. Fracture may complicate extension of the stents beyond the inguinal ligament into the CFV. Typical fractures are approximately 1 cm beyond the line of the ligament and correspond to the head of the femur. Many hypotheses have been suggested regarding the cause of fracture, including compression by the ligament or against the bony prominence of the pubis.

Migration of stents is a clinical disaster because once a stent moves from the common iliac vein, the typical end point is the right side of the heart, likely lodging in the tricuspid valve. This can be life-threatening but more frequently results in open heart surgery to retrieve the stent and repair the tricuspid valve. Stent migration is much more likely to occur in nonthrombotic iliac vein lesions where the normal vein on either side of the compressive lesion allows for less “gripping” of the stent than that seen in chronic postthrombotic occlusive disease. Migration is prevented by ensuring the stent is an adequate diameter and length. In practice, this means...
avoiding short-length stents where the temptation is to place a stent just across the compressive lesion. A Valsalva maneuver postprocedure almost always results in some stent movement with this strategy. Placement of longer-length stents with extension into the EIV will mitigate this problem significantly.

**Back Pain**

Back pain is a sequela of placing venous stents and resolves after 2 to 3 weeks in most patients. It is much more probable that it will occur when larger stent diameters are placed, and for this reason, it is important to ensure that nitinol stents are not oversized. Back pain may also occur due to compression of the nerves arising from the lower lumbar and upper sacral region as the stent traverses the spine and sacrum to the pelvis. Persistent back pain has been well described, and explantation of the stent in the most severe cases may be the only alternative.

**HEMATOLOGIC REASONS FOR FAILURE**

Anticoagulation strategies are vital after stent placement to reduce the risk of early stent thrombosis. This is especially important in the context of acute and chronic postthrombotic disease. Although the importance of anticoagulation is reduced in patients with nonthrombotic iliac vein lesions, early stent thrombosis has still been reported, and thus it cannot be totally ignored. Animal studies have indicated that it takes approximately 56 days for the newly placed stents to epithelialize, and the data on stent thrombosis support that the risk of thrombosis is higher in the first 6 weeks after stent placement, although it may still occur later.

In our practice, we place all patients on 2 weeks of low-molecular-weight heparin after stent placement—a full treatment given as a twice daily dose. This is designed to “flatten” the dosing and prevent extreme troughs. After 2 weeks, we convert patients to warfarin for the first 6 months and then consider a direct oral anticoagulant if longer-term anticoagulation is needed.

We have observed no difference in outcomes between patients with or without a defined thrombophilia; however, this is only achieved if the anticoagulation strategies are closely followed.

In our patients who have stent thrombosis with no technical or flow issues, the majority have been secondary to patient compliance. This is partly the rationale for warfarin. The requirement for monitoring and the ability to check the international normalized ratio help to exclude this as a cause.

Additional issues have arisen from underdosing low-molecular-weight heparin in patients > 100 kg, and we have therefore begun routine testing of antifactor Xa levels in these patients with appropriate dose adjustment. Additional groups at risk are patients with triple-positive antiphospholipid syndrome who are maintained on warfarin after 6 months with no use of direct oral anticoagulants.

Patient education on anticoagulation is vital. For example, few patients understand that rivaroxaban needs to be taken with food and at a consistent time of day. In patients on a single daily dose of rivaroxaban, it is preferable to give this dose with an evening meal rather than breakfast to avoid late-night troughs when the patient is typically immobile and asleep.

A multidisciplinary team including a hematologist with an interest in thrombosis is a vital component of managing complex patients.

**FAILURE DUE TO FLOW**

Flow is the single most difficult factor to account for, and in many respects, this becomes more an issue of patient selection. It is increasingly apparent from data analysis that patients with normal inflow to the CFV
and good flow into the stents will likely do well regardless of the length of the stent cranial to this. In our practice, cumulative stent patency in patients with normal inflow and stents below the ligament is 90%, and the patency drops to 70% in those with single or, particularly, multiple diseased inflow segments. Although good results are clearly still achievable, close attention needs to be paid to the quality of the inflow vessels. In patients with extensive CFV disease, this may necessitate endophlebectomy and fistulas, but the outcomes for these patients have not been shown to be good in the long term, with significant short-term wound-related and infective complications.

Selecting a good target vessel for inflow at the index procedure is vital; however, if a stent thromboses and it is clear that the previously outlined technical issues are corrected and the anticoagulation strategy is appropriate, then flow is the issue (Figure 4). In some patients, persistent balloon dilation of the dominant inflow vessel (whether the femoral vein or profunda femoris vein) may improve the flow to the extent that a good outcome is achieved. If the inflow is not improved and a single target vessel is identified, extension of the stent into either the femoral vein or profunda femoris vein may be appropriate on occasion, but it is rare that this achieves lasting results. Improvement of inflow remains the biggest challenge in venous stenting procedure.

**CONCLUSION**

Venous stenting achieves good results in most patients, with well-established long-term benefits. However, it is clear that attention to detail is important to achieve lasting results and reduce the need for reintervention over the long term in this patient population. A simple practice for addressing the potential issues that may cause stent failure—ideally done by a methodologic approach at the time of stent implantation with clear peri- and postprocedural protocols—minimizes these failures and provides a framework for reintervention when patients do present with problems.

In the longer term, the absence of a standard set of outcomes and classification of disease pretreatment continues to compound our understanding of factors that may enable better patient selection and improve outcomes. It is beholden upon the venous community to come together to address these issues.


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