The ease of achieving hemostasis after radial artery access and the significant decrease in access site complications are probably the main reasons that make the transradial approach attractive. Radial artery compression is well tolerated and easy to perform in view of the absence of large neurovascular structures in the vicinity of radial artery, extensive collateralization at the level of the hand, and the hard surface of radius bone upon which the radial artery lies. These attributes allow for the application of liberal compression and frequently excessive compression at the radial access site. The small lumen and thicker wall of the radial artery lead to obliteration of the radial artery lumen and a resultant cessation of radial artery flow when liberal compression is applied. Although hemostatic compression at the radial access site is very effective in providing hemostasis, this article discusses its relationship with radial artery occlusion and the importance of the finer details of radial hemostasis.

**RADIAL ARTERY OCCLUSION**

Radial artery occlusion (RAO) occurs in 2% to 10% of patients after transradial catheterization. Extensive macro- and microcollateralization protects the hand from ischemic complications at rest, although occasional digital ischemia/gangrene has been reported, which is likely due to embolization of digital end arteries. Local inflammatory symptoms occur in some patients, and frequently resolve spontaneously. In the majority of patients, recanalization of the occluded radial artery occurs during the following month with re-establishment of radial artery patency. In some patients with acute occlusion in whom recanalization does not occur, permanent obliteration of the radial artery lumen occurs as a result of fibrotic organization of the thrombus.

**BY SAMIR B. PANCHOLY, MD, FACP, FACC, FSCAI**
Administration of anticoagulants, such as unfractionated heparin, enoxaparin, and bivalirudin, has been shown to reduce the incidence of RAO. It appears to be a systemic effect as evidenced by the comparative efficacy of intravenously administered anticoagulant agents. Although some operators administer unfractionated heparin intra-arterially, no difference is observed in the efficacy of RAO prevention between intra-arterial and intravenous routes of administration. Another factor related to the occurrence of RAO is a high sheath-to-artery ratio, likely related to intimal trauma caused by oversized catheters. The occurrence of RAO, although free of major symptomatic consequence, leads to loss of a preferred access site because ipsilateral transradial access in an occluded radial artery is difficult, if not impossible.

The mechanism of acute RAO after radial access appears to be transmural thrombosis, with rapid organization causing fibrotic obliteration of the lumen. Intimal injury from catheters and spasm-related flow cessation in the radial artery initiate the process of thrombus formation. The subsequent hemostatic compression-related flow interruption reinforces the process of local thrombus formation and propagation, leading to transmural thrombosis and occlusion. RAO has been observed to be significantly more prevalent in patients with radial artery flow interruption during hemostasis. Hence, optimization of the radial artery hemostasis technique offers an opportunity to prevent RAO.

Active pursuit of radial artery patency during hemostatic compression, without compromising hemostasis, has been shown to be effective in lowering the incidence of RAO. Using the “patent hemostasis” technique, the combination of radial artery hemostasis and patency during compression can be achieved in more than 75% of patients. Despite the lower compression pressures used with patent hemostasis, no significant increase in hemorrhagic complications has been noted.

**PATENT HEMOSTASIS TECHNIQUE**

Patent hemostasis, as the name implies, aims at achieving radial artery hemostasis with maintenance of radial artery patency. This can be achieved in the following fashion:

- **Step 1:** Apply the radial hemostasis device to the radial artery puncture site (Figure 1).
- **Step 2:** Tighten or inflate the device to apply pressure at the arterial puncture site. Of note, it is important to center the device at the point of arterial entry, and not skin entry, to prevent subcutaneous hemorrhage (Figure 2).
- **Step 3:** Remove the introducer sheath from under the compression device. It is optimal to open the stopcock and let the side arm of the introducer “bleed” in order to remove the prethrombotic material and thrombi from the radial artery lumen (Figure 3).
- **Step 4:** Loosen or deflate the compression device until visible and continuous leakage of blood from the access site is noted. Once “bleeding” is seen, gently tighten or reinflate the device until com-
Complete cessation of bleeding occurs. Apply the least amount of pressure necessary to eliminate active bleeding (Figure 4).

- **Step 5**: Perform a reverse Barbeau’s test by using plethysmography, with ipsilateral ulnar artery compression, to assess radial artery flow. If radial artery flow is present, you have achieved both endpoints, and periodic monitoring of adequacy of hemostasis and radial artery patency is recommended (evaluation every 15 minutes has been our practice) (Figure 5).

- **Step 6**: If radial artery patency is not observed, repeat the process of decreasing hemostatic compression pressure to maintain hemostasis. In the first 15 minutes of compression, the balance of radial patency and hemostasis changes frequently due to changes in patients’ blood pressure, and hence frequently, even if patency is not achieved on the initial attempt, it can be established with subsequent attempts.

**OTHER FINER DETAILS**

A longer duration of compression is associated with a higher incidence of RAO compared to a shorter duration of compression. 

A compression duration of 2 hours has been shown to have a significantly lower incidence of RAO compared to a 6-hour duration of compression. This is likely a result of a higher probability of flow cessation with longer duration of compression. It may also be related to the absence of the anticoagulant effect of heparin after 4 hours of administration, increasing the probability of thrombus formation with duration of compression longer than 4 hours. The data comparing different compression devices have been conflicting, with some suggesting that devices that “loosen up” over time may inherently help prevent RAO by reducing compression pressure. An ideal hemostatic device would have a programmed decay in compression pressure, facilitating flow maintenance and preventing RAO without compromising hemostasis.

It is of paramount importance not to apply tight compressive dressings after removal of the radial hemostatic device because doing so frequently occludes the radial artery lumen and defeats the purpose of patent hemostasis.

In patients in whom patent hemostasis cannot be achieved, hemostasis being the primary intention, necessary compression needs to be applied to prevent blood loss or formation of hematoma. Recanalization of an occluded radial artery after removal of compression is seen in a fraction of patients. If radial artery patency is not observed after removal of the compression device, occlusive compression of the ipsilateral ulnar artery has been shown to facilitate re-establishment of radial artery patency.
Patency of the radial artery during the process of hemostatic compression appears to exert the most potent influence on postprocedural radial artery patency. Although systemic anticoagulation is a must in any transradial procedure, patency during hemostasis, if maintained fastidiously, may obviate the need for administration of anticoagulation in patients with bleeding diathesis, as shown in a recent prospective randomized, proof-of-concept trial.  

**SUMMARY**

Hemostasis after transradial access, although effective and uneventful, needs an “active” approach, with careful attention to prevent RAO. Incorporation of a patent hemostasis technique in postprocedural protocol with an interdisciplinary team approach will maximize radial artery patency, making future ipsilateral transradial access feasible.

Samir B. Pancholy, MD, FACP, FACC, FSCAI, is Program Director, Fellowship in Cardiovascular Diseases, The Wright Center for Graduate Medical Education; and Associate Professor of Medicine, The Commonwealth Medical College in Scranton, Pennsylvania. He has disclosed that he receives speaking honoraria from Medtronic, Inc., Terumo Interventional Systems, Inc., and The Medicines Company. Dr. Pancholy may be reached at (570) 587-7817; pancholys@gmail.com.