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EDITORIAL COMMENT

Just the Right Pressure to Optimize Post–Radial Access Care*

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Transradial arterial access (TRA) has emerged as the preferred choice to reduce bleeding, access-related complications, and mortality among patients undergoing coronary angiography and percutaneous coronary intervention (PCI). Unlike in many European and Asian countries, transfemoral arterial access remains the predominant mode of arterial access across the United States. However, recent practice trends demonstrate a steady increase in TRA uptake for both coronary angiography and PCI (1).

Complications during and following TRA include radial artery occlusion (RAO), spasm, pseudoaneurysm, arterial perforation, dissection, and eversion of the artery during sheath removal. Of these, RAO is by far the most common, occurring in 8% to 10% of cases, with reported rates as high as 30% with larger sheath sizes in registry studies (2). RAO is thought to occur as a consequence of diminished blood flow and endothelial disruption during arterial sheath insertion. The clinical presentation is often asymptomatic because of the benefit of a dual blood supply with rich collateral circulation that perfuses the hand. However, a small percentage of patients experience pain or paresthesia with RAO, and a minority report reduced limb function. Additionally, downstream consequences of RAO include inability to reaccess the radial artery for future procedures, loss of an arterial conduit for coronary artery bypass grafting, and loss of a site for future arterial-venous fistulas. For these reasons, minimizing RAO remains a clinical priority for interventional cardiologists.

Hemostasis following TRA can be achieved mechanically using a wristband with an inflatable bladder or through manual compression by the operator. In the United States, mechanical compression is used most widely, though the 2 approaches have not previously been comparatively evaluated. In this issue of *JACC: Cardiovascular Interventions*, Petroglou et al. (3) report on a randomized comparison of manual or mechanical radial artery compression in 589 patients from 5 centers following diagnostic coronary angiography using 5-F catheters. Notable exclusions were patients who underwent ad hoc PCI or who previously underwent TRA. The primary endpoint was RAO at 24 h, assessed by color duplex ultrasound and interpreted by blinded physician readers. Secondary endpoints included bleeding, vascular complications, and time to hemostasis. RAO occurred in 12% of patients assigned to manual compression and 8% with mechanical complication (p = NS), and manual compression was associated with a significant reduction (98 min) in time to hemostasis.

SEE PAGE 1050

Bleeding and vascular complication rates were similar between the groups. Overall, accounting for clinical and procedural variables, limitations in study size, and event rates, the analysis did not show a definitive benefit with either manual or mechanical compression.

Using multivariate regression models, the investigators identified a number of independent predictors of RAO at 24 h post-procedure: the number of arterial puncture attempts, activated clotting time, radiation time (as a proxy for length of procedure), and patency of flow in the radial artery during hemostasis ("patent hemostasis"). In this study, patent hemostasis was associated with a 92% reduction in odds for RAO. Among all of these clinical variables,

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patent hemostasis has been consistently identified as among the most important nonpharmacological factors in preventing RAO. In the PROPHET (Prevention of Radial Artery Occlusion—Patent Hemostasis Evaluation) trial, RAO rates at 24 among 436 patients randomized to patent or occlusive hemostasis with the HemoBand were 5% and 12% ($p < 0.05$), respectively (4). The RACOMAP (Radial Compression Guided by Mean Artery Pressure Versus Standard Compression With Pneumatic Device) trial demonstrated even larger differences in RAO with patent versus occlusive hemostasis following TRA with the TR Band (1.2% vs. 12%; $p < 0.0001$) (5). Other factors identified through observational studies as associated with RAO include age, sex, sheath size, heparin dose, radial artery size, and compression time.

Overall, this study highlights several important clinical insights regarding post-TRA outcomes. First, RAO is an important complication following TRA that can be significantly underrecognized in the clinical setting. In this study, RAO occurred in approximately 10% of patients, similar to large observational studies. However, clinical detection primarily relied on Doppler evaluation rather than clinical examination alone. Second, the study findings support the conclusion that mechanical compression appears to be safe compared with meticulous manual compression. Despite a drastic reduction in time to hemostasis, the process of meticulous manual compression is more resource intensive viewed from the standpoint of personnel time, such that the incremental cost of a compression band would be negligible in comparison. Third, patent hemostasis is an important priority in post-TRA management. In this study, patent hemostasis was associated with an astounding 92% reduction in odds for RAO. Approximately 10% of patients did not achieve patent hemostasis despite meticulous technique, such as intermittent ulnar compression and continuous plethysmography to assess arterial patency. Of note, the present study excluded PCI procedures, and it is therefore unknown whether manual compression would significantly reduce time to hemostasis after such procedures, which require significant additional anticoagulation and frequently larger sheath sizes.

Strategies to reduce rates of RAO include adequate anticoagulation at the time angiography. Recommended agents include unfractionated heparin 70 U/kg up to 5,000 U and bivalirudin 0.75 mg/kg bolus followed by 1.75 mg/kg/h during the procedure. Additional strategies include using low-profile sheaths that are smaller than the diameter of the radial artery, avoiding repeated access of the radial artery, and patent hemostasis. Techniques to improve patent hemostasis, as recommended by the Society for Cardiovascular Angiography and Intervention's Transradial Working Group, include allowing mild pulsatile bleeding after application of the hemostatic compression device, followed by tightening to eliminate bleeding and subsequent evaluation for patency using a reverse Barbeau test (6). If compression of the ulnar artery leads to absence a plethysmographic waveform, the band should be loosened to allow antegrade radial artery flow. More recently, the PROPHET-II trial demonstrated additional reduction in 30-day RAO using prophylactic ipsilateral ulnar compression in addition to patent hemostasis (7). It is likely that without attention to such techniques, rates of patent hemostasis are lower, and RAO even higher, than we appreciate. Future studies evaluating responses to lack of patent hemostasis and optimal strategies to monitor radial patency may additionally help improve outcomes following TRA.

Overall, the study by Petroglou et al. (3) highlights several important messages for interventionalists pursuing TRA for coronary angiography and PCI. Chief among these are that RAO is an underrecognized complication following TRA access, with rare but important clinical implications. Patent hemostasis is powerfully associated with reduced risk for RAO. Thus, strategies to ensure patent hemostasis are important quality optimization opportunities. Although complications associated with TRA are, overall, less severe than those associated with transfemoral access, meticulous radial access techniques and post TRA care should be emphasized to optimize clinical results, and support a radial-first approach.

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