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# Shorter Radial compression Time: Is Chemistry the Solution?

In this issue of the journal, Haq et al. [1] report the results of their randomized trial comparing two strategies of hemostatic compression in patient who had undergone transradial access for cardiac procedures. The authors found that when using a hemostasis-promoting disc in conjunction with a mechanical compression device, a significant reduction in time to hemostasis was observed. In the control group, the duration of compression was a mean of 2 h and 46 min, and in the hemostasis promoting disc group it was 1 h and 20 min. Radial artery occlusion (RAO) rates were similar between the groups. Although re-bleeding requiring extension of compression duration was not different, hematoma formation was numerically and statistically significantly higher in the disc group. Similar occurrence of higher incidence of rebound bleeding has been observed in previous reports [2]. This is likely related to the impact on clot stabilization that duration has, which is likely not replaced by the prothrombotic agents that have been evaluated. The radial artery also has unique anatomic disadvantages. Its lumen-to-wall thickness ratio makes it very susceptible to lumen collapse while compressing, hence making it vulnerable to occlusion, although the very same characteristic makes it easier to achieve hemostasis. Also, the lack of large tissue space between the artery and the skin, as well as lack of anchoring anatomy such as a fibrous sheath or canal that houses, it makes it relatively mobile under the skin and, hence, increases the possibility of shearing off the hemostatic plug.

The pursuit of accelerated hemostasis has obsessed interventionalists regardless of access site and has yielded modest results at best despite innovation. This lack of unequivocal efficacy and safety is likely driven by the lack of full understanding of the complexity of the process of hemostasis in a tubular structure exposed to pulsatile flow and its resultant stresses, and a myriad of intrinsic biologic processes triggered simultaneously with multiple extrinsic influences modifying the balance. Use of prothrombotic material applied externally likely accelerates the formation of the hemostatic plug, but upon removal of the reinforcing external compressive force, both mechanically and biochemically facilitates its dislodgement. The dominance of the thrombolytic processes, created by suppression of the intrinsic prothrombotic processes caused by residual heparin effect, likely has a large role to play.

Whether these findings were a mere effect of additional mechanical reinforcement during hemostatic compression also needs to be evaluated. To separate the mechanical reinforcing effect of the disc from its biologic effects will require an adequately sampled trial where a biologically inert disc is directly compared to the procoagulant accessory with similar mechanical features.

The effort to find a better solution to maximize radial artery preservation after transradial access, while improving throughput as well as patient comfort, should continue to further refine this elegant procedure.

## References

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