



Hand Thermography: A Novel Approach to Evaluate Hand Function After Transradial Access ^{☆,☆☆}

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Thermoregulatory control of skin temperature involves both central reflexes facilitated by sympathetic vasoconstrictors and vasodilator nerves, along with a variety of local thermal control interactions mediated by several vasoactive pathways [1]. These reflexes interact with both the nutritional capillaries and intrinsic arteriovenous shunts to change the vascular flow. Transradial cardiac catheterization almost universally results in endothelial damage from access sheath trauma [2] and may, therefore, damage the thermoregulatory control to the hand as an unintended consequence of the transradial approach. This question of whether transradial catheterization may be associated with changes in hand blood flow was explored in a paper by Maki et al. [3], in this issue, using serial hand thermography.

The investigators used ultrasound technology and a thermal imaging camera system to evaluate hand temperature both regionally, and overall in both the catheterized extremity and non-instrumented extremity. These measurements were done at baseline before the catheterization, and then 30-days later with 158 subjects completing the full follow-up. Ultrasound demonstrated a single radial occlusion but otherwise no significant differences in radial diameters or velocities either pre- or post-catheterization in either hand [3]. This would suggest that radial arteries used for catheterization remained adequate as conduits and mechanical changes in arterial size or lumen should not be affecting hand temperature.

Hand thermography before and after the procedure did show changes. Overall, hand warmth was increased at 30-days in both hands compared to baseline. With occlusion of the ulnar artery, both sides before the procedures showed a reduction in temperature and also at 30-days afterward. This ulnar induced temperature drop was attenuated in both arms at follow-up [3]. On one hand, this preservation of vascular size and downstream warmth in the side that underwent radial catheterization is reassuring, but other questions are raised by the apparent systemic effect seen with the non-instrumented hand also responding to catheterization procedure. What is happening here, and can we explain these findings?

Use of hand thermography to evaluate hand flow after transradial catheterization is a relatively novel approach but what do the results imply? With the physical size of the radial arteries unchanged based on ultrasound, simple mechanical changes cannot be playing a role, or

explain the differences in the non-instrumented hand. There must be a system explanation. Perhaps this is just an example of exposure attenuation. The subjects were tested just before cardiac catheterization. They may have been tense and undoubtedly unfamiliar with the test protocol much less other concerns about the catheterization procedure itself. At 30-days, the subjects were familiar with the testing procedures, and the catheterization was now in the past, resulting in a reduction of their anxiety and therefore alteration in their net hand warmth. Testing the subjects undergoing a stress test with thermography before and after 30-days might be one way to check whether exposure attenuation might be at play using stress testing as the stressor but without the trauma of the catheterization.

Another alternative explanation not suggested by the authors were the effects of medication changes that may have occurred between the initial testing and 30-day follow-up. Many patients have medication adjustment as a result of the findings from the cardiac catheterization. These changes, usually result in an increase in vasoactive drugs such as nitrates or calcium channel blockers. Since we don't have an account of these changes in the subjects, this does raise the possibility that these systemic effects are pharmacologic effects. Likewise, the subject's sympathetic tone may have been reduced if angina had been successfully treated by either pharmacologic changes or percutaneous revascularization with stenting accomplished during the index procedure.

The authors did speculate that ischemic conditioning and neuronal-humoral signal transduction [4] maybe another explanation as to why hand temperatures increased by 30-days. There are direct observations such as the skin finding of livedo racemose (a form of livedo reticularis) that is associated with alterations in microvascular blood flow and can be seen after transradial catheterization [5]. Also, alterations in nerve conduction in the forearm have also been noted after transradial catheterization [6]. Altered or injured microvascular control might explain local changes in the skin thermoregulatory system. But, systemic or bilateral changes implies that there exists central or spinal cross-talk between the reflex controls such that the contra-lateral hand is also affected. Such cross-talk has been described before with remote ischemic preconditioning associated with effects beyond the region of ischemic conditioning [7]. Whether transradial catheterization is associated with enough ischemia to induce pre-conditioning is unclear. Lactate production during radial catheterization is minimal in the hand and only rises 10–20% over baseline during hemostasis [8]. The subjects in the present study had a very low rate of radial occlusion (<1%) and most likely underwent patent hemostasis [9] which would have further reduced ischemic zones during hemostasis. Given the limited amount of ischemic stimulus available from radial artery catheterization, the

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support for this hypothesis as the underpinning of increased hand temperature appears tenuous. Likewise, there are several other candidates that might act as stimulants of a remote response during a transradial procedure. These include microembolization, pain induced from access, spasm, and medications used during the procedure. None of these appear as obvious inducing agents with their limited dosing and transient biologic half-lives that would diminish any stimulating role.

While this novel thermography study raises more questions than answers, there is some good news. There was no evidence that iatrogenic narrowing of the radial arteries occurred in this 157 subject-cohort beyond the one subject with radial occlusion. The low radial occlusion rate reported further exemplifies that at 30-days minimal residual trauma may exist in the radial artery after modern hemostasis. The question that remains is why did the hand temperatures increase in both the instrumented and control hands. Further testing and data can probably tease out the answer, but for now one can only speculate.

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