Median Nerves Electrical Activation Reduces Ipsilateral Brachial Arteries Blood Flow and Diameter – Commentary

This interesting article on the changes to the brachial artery flow velocity following ipsilateral and contralateral median nerve stimulation is an eye-opener to a new range of opportunities provided by bedside Doppler sonography. The autonomic nervous system comprises a vast expanse of neural tissue that spans from the hypothalamus to the sympathetic, parasympathetic, and sudomotor nerves. As there is no single test to monitor all autonomic functions, the quest for a reliable bedside, noninvasive diagnostic method has a priority in the hierarchy of tests for autonomic dysfunction.[1,2] Some tests have a risk of precipitating cardiac arrhythmias or hypotension in subjects with dysautonomia, and hence simple and quantitative bedside tests that are performed by specialists are reliable in patients suspected to have autonomic dysfunction.[3-5]

The difference between ipsilateral and contralateral brachial artery flow is a novel bedside procedure that can be conducted in outpatient clinics or at the patient's bedside with an accurate estimate of the sympathetic function.[3]

There have been recent reports of brachial artery flow-mediated responses to pharmacological and physiological interventions.[3,4] The vasoconstriction of the brachial artery in response to electrical stimulation of the ipsilateral median nerve is an index of vasomotor function mediated through the peripheral nerves.[3,4]

Going through the published literature, a study was conducted by O Kaczi *et al*.[3] utilized Doppler flow studies in 31 healthy volunteers and detected significant reduction of brachial arterial velocity following stimulation of peripheral sympathetic nerves. Here, the utility of Doppler sonography was expanded beyond its realm into a reliable bedside procedure that measured arterial flow velocity, flow rate, and flow direction to quantitate peripheral sympathetic function.[3]

Dyson *et al*.[4] recorded that the flow-mediated dilatation of the brachial artery was impaired by sympathetic activation in healthy subjects. While Harris *et al*.[5] reported that mental stress released catecholamines that caused vasoconstriction of the brachial arteries in Doppler studies.

The present study on alteration of brachial artery diameter and blood flow in response to median nerve stimulation was conducted in 62 healthy volunteers with an age range of 19–61 years. The decreased volumetric flows were consistent and reliable parameters in all subjects. The test was conducted by experienced sonologists and the response was documented by a Doppler transducer placed over the right brachial artery. A week later, the test was repeated on the contralateral arm to the right median nerve stimulation, and there were no changes in flow dynamics. This method thus evaluated the peripheral sympathetic nerve function in these healthy subjects independent of the adrenal activity and body position, which can interfere with sympathetic function. There was a significant decrease in the median nerve diameter following stimulation of the ipsilateral side, thus confirming that the sympathetic activation was through the same peripheral nerve.

The points in favor of this method were the test was noninvasive, cost-effective, and easy to perform bedside procedures that could be used in clinics or hospitals. The procedure reliably estimated the sympathetic function, and the response was the same when performed by different operators like physicians, sonologists, or neurologists. The results could be obtained immediately. Sympathetic nervous system dysfunction could be easily detected without an added risk of vasovagal attacks or cardiac dysfunction, as in Head-up Tilt table test. This method has been tested in a wide range of ages varying from 19 to 61 years, and the results were similar and quantifiable using Doppler Ultrasonography. The vasoconstriction of the Brachial Artery lasted for 2–5 seconds and was caused by activation of the sympathetic nervous system with a reduction in volumetric flows, which can be monitored by sonography. This test was independent of the adrenal function and was not altered in hypermetabolic conditions.

The points against were that the tests were done in healthy young subjects, and the normative data were restricted to the physiological changes of the brachial artery and ipsilateral median nerve. As of date, there have been no similar studies in patients with autonomic neuropathy. These tests have not been validated in elderly subjects with sympathetic dysfunction. These tests have not been used in patients with neurodegenerative diseases. This test does not record parasympathetic or sudomotor dysfunction. Preparation of the subject was needed four hours before the procedure by restricting physical activity, caffeine, nicotine, and alcohol.

Conclusion

Comparison of this technique with contemporary studies and application of these methods to hospitalized subjects will help in improving this diagnostic technique for large-scale use. Interobserver variability and operator reliability are crucial for accurate results. We would recommend that the authors recruit more subjects in their study group and compare their results with patients suffering from disorders like carpal tunnel syndrome, diabetic autonomic neuropathy, primary or secondary dysautonomia, parkinson's disease, or multi system degeneration. The clinical benefit of the test will depend on its utility in patients with autonomic dysfunction rather than healthy young volunteers. This test has an advantage over many

other tests of dysautonomia in that it is a bedside, noninvasive, and cost-effective test with reliable and easily reproducible methods. We hope Doppler ultrasonography will be used in more centers to assess the vasoconstrictor response to peripheral nerve stimulation. The benefits of these large-scale studies could be compared simultaneously for better results.

Laxmi Khanna

Consultant Neurologist and Neurophysiologist, Sir Ganga Ram Hospital, New Delhi, India

Address for correspondence: Dr. Laxmi Khanna, Consultant Neurologist and Neurophysiologist, Sir Ganga Ram Hospital, New Delhi, India.
E-mail: laxmikhanna@hotmail.com

References

1. Assessment: Clinical autonomic testing report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. Neurology 1996;46:873-80.
2. Cheshire WP, Freeman R, Gibbon CH, Cortelli P. Corrigendum to "Electrodiagnostic assessment of the autonomic nervous system: A consensus statement endorsed by the American Autonomic Society, American Academy of Neurology, and the International Federation of Clinical Neurophysiology. Clin Neurophysiol 2021;132:666-82.
3. Kaczi O, Ege F. Evaluation of sympathetic vasomotor activity of the brachial arteries using Doppler ultrasound. Med Sci Monit 2023;29:e939352. doi: 10.12659/MSM.939352.
4. Dyson KS, Shoemaker JK, Hughson RL. Effect of acute sympathetic nervous system activation on flow-mediated dilation of brachial artery. Am J Physiol Heart Circ Physiol 2006;290:H1446-53.
5. Harris CW, Edwards JL, Baruch A, Riley WA, Pusser BE, Rejeski WJ, *et al*. Effects of mental stress on brachial artery flow-mediated vasodilation in healthy normal individuals. Am Heart J 2000;139:405-11.

Submitted: 15-Jul-2023 **Accepted:** 08-Aug-2023
Published: 31-Oct-2023

DOI: 10.4103/aian.aian_628_23