Effect of Polyunsaturated Fatty Acids on Baroreflex Function in Heart Failure Patients

Recently in the Journal, Radaelli et al. (1) reported that 4 months of dietary polyunsaturated fatty acid (PUFA) supplementation enhanced baroreflex control of the circulation in heart failure patients. Augmented cardiac–vagal baroreflex control was suggested from increased bradycardic responses to neck suction as well as enhanced cardiac period fluctuations during spontaneous blood pressure oscillations after PUFA supplementation. These effects of PUFAs on the cardiac–vagal arm of the baroreflex in heart failure patients were impressive and may be of clinical importance.

Based on the detrimental role sympathoexcitation is believed to exert in heart failure, a separate but equally important question is the effect that PUFAs exert on both basal and reflexive measures of sympathetic outflow. To address this question, Radaelli et al. (1) used several indirect indices. First, increased low frequency heart rate variability after PUFA supplementation was used as evidence of reduced sympathetic activity at rest (1). Second, a greater depressor response to neck suction, which likely is mediated via both cardiac (vagal and sympathetic) and peripheral (sympathetic) effects, after PUFA supplementation, was suggested to indicate enhanced sympathoinhibitory baroreflex function (1). As pointed out, these effects of PUFAs in heart failure patients may need to be confirmed using more robust measures of sympathetic outflow, which should also include responses to sympathoexcitatory stimuli (2).

To date, only a single study performed in humans has determined the effect of PUFA supplementation exerts on a direct measure of sympathetic outflow (muscle sympathetic nerve activity [MSNA]) (3). In that study we demonstrated that PUFA supplementation had no effect on MSNA at rest (against our hypothesis that it would reduce it). Furthermore, we observed augmented (not depressed as we hypothesized) increases in MSNA during several distinct sympathoexcitatory stressors (exercise and cold stress) in young healthy adults (3). Because sympathetic outflow at rest is increased and reflex responses to baroreflex activation/deactivation may be impaired in heart failure patients, it is unclear whether similar effects of PUFAs on MSNA would be observed in heart failure patients. Accordingly, it appears that studies employing more robust methodologies are needed to more definitively determine the effect that PUFAs exert on basal and reflexive (both sympathoinhibitory and sympathoexcitatory during baroreflex and nonbaroreflex stimuli) regulation of sympathetic outflow in heart failure patients.

*Kevin D. Monahan, PhD
Chester A. Ray, PhD

*Penn State Heart and Vascular Institute
The Milton S. Hershey Medical Center
Campus Box H047
500 University Drive
Hershey, Pennsylvania 17033-2390
E-mail: kmonahan@psu.edu
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REFERENCES


Reply

We appreciate the comments by Drs. Monahan and Ray concerning our suggestion that, in heart failure patients, polyunsaturated fatty acid (PUFA) supplementation may attenuate efferent sympathetic nerve overactivity (SNA) (1). We entirely agree that this issue is of potential clinical relevance and merits direct experimental testing, as indicated in the discussion of our own study and reinforced by Floras and Bagai in their accompanying editorial (2).

This question is made even more interesting because, as appropriately recalled by our colleagues, they previously observed (3) that PUFA supplementation failed to modify resting muscle SNA in healthy individuals. Conversely, we all perfectly realize that the two possibilities (no change in the healthy subject, reduction in the heart failure patient) are by no means mutually exclusive considering that regulation of autonomic outflows is significantly altered in cardiac systolic dysfunction, and may thus be differentially affected by PUFA supplementation compared to the normal condition.

While awaiting more direct evidence (cardiac norepinephrine spillover? peroneal microneurography? metaiodobenzylguanidine cardiac scans?), all we can do is to contemplate the most reasonable interpretation of the indirect evidence emerging from our study, namely that PUFA 1) increased both low- and high-frequency powers of the RR interval; 2) potentiated cardiovascular inhibitory control by the baroreflex; 3) fell short of significantly slowing heart rate; and 4) markedly enhanced overall RR interval variability. Each of the previously-cited effects points to a PUFA-related reduction in sympathetic (and probably an enhanced vagal) nerve activity.

*Alberto U. Ferrari, MD
Giuseppe Mancia, MD
Alberto Radaelli, MD
We congratulate Hendel et al. [1] on their detailed documentation of appropriateness criteria for cardiac computed tomography (CT) and cardiac magnetic resonance imaging, which they published in the October 3, 2006, issue of the Journal. We are, however, concerned regarding the apparent recommendation for use of non-electrocardiographic (ECG)-gated CT angiography in the evaluation of potential aortic dissection (Tables 8 and 10 in Hendel et al. [1]).

The aortic root and ascending thoracic aorta move in concert with the left ventricle and have the greatest motion during systole. It is well documented that motion artifacts from aortic wall motion and to reduce motion artifacts when compared to results of non-ECG-gated studies [6,7]. Furthermore, the application of ECG gating by adequately trained technologists has no impact on the workflow of the CT examination [7]. In our own practice, we prefer the use of prospective ECG gating to minimize radiation exposure to our patients.

Therefore, we believe that ECG gating should be mandatory for thoracic aortic CT angiograms performed to detect potential aortic dissection. We hope that the investigators agree and will promptly make this critical and appropriate correction.

Benjamin Cheong, MD, MRCP (UK), FACC
Scott D. Flamm, MD

*Cardiovascular Imaging, Hb-6
Cleveland Clinic Foundation
9500 Euclid Avenue
Cleveland, Ohio 44195
E-mail: flamms@ccf.org

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