Creatine Kinase and Pressor Response to Orthostatic Tolerance

To the Editor:

In a recent publication, Okada et al. reported that elderly blacks have a blunted sympathetic neural responsiveness, but greater pressor response to orthostasis than elderly whites. The authors conclude that elderly blacks had a greater response in total peripheral resistance for a given change in total muscle sympathetic nerve activity during tilting. The authors suggested that an augmented sympathetic vascular transduction or enhanced nonadrenergic vasoconstrictor mechanisms are involved in this enhanced pressor response. In an earlier article, greater orthostatic tolerance was also found in black women. Commenting on their suggestions, we offer a possible mechanistic contribution.

We have proposed that black individuals have higher vascular contractility because of high creatine kinase (CK) activity. CK rapidly provides ATP for highly energy-demanding cellular responses and may increase ATP-buffering capacity leading to enhanced vascular contractility. At subcellular locations with high energy demands, CK rapidly regenerates ATP, near Na⁺/K⁺–ATPase and Ca²⁺–ATPase at membranes, as well as near myosin light chain kinase and myosin ATPase at the contractile proteins. Even a small increase in the contractility of vascular smooth muscle could have a profound effect on resistance of flow and hence arterial pressure, as expressed in the Poiseuille–Hagen formula, blood flow and resistance in vivo are markedly affected by small changes in the caliber of the vessel.

High CK, as found in men, obese, and black individuals, is thus thought to increase orthostatic tolerance. In line with this, we found that those with low serum CK, as a measure of tissue CK, had a higher lifetime cumulative incidence of fainting. Moreover, high CK activity is associated with predominance of high CK type 2 fibers and capillary rarefaction, which might reduce venous pooling of blood on standing. Thus, we propose that high vascular CK, as found in black individuals, may contribute to the enhanced orthostatic tolerance found in this population subgroup. Further studies are needed to confirm the potential role of CK and ATP buffer capacity in resistance arteries, and its effect on orthostatic tolerance.

Disclosures

None.

Fares A. Karamat
Gert A. van Montfrans
Lizzy M. Brewster
Departments of Internal and Vascular Medicine
Academic Medical Center
University of Amsterdam
The Netherlands

Creatine Kinase and Pressor Response to Orthostatic Tolerance
Fares A. Karamat, Gert A. van Montfrans and Lizzy M. Brewster

*Hypertension*. 2013;61:e24
doi: 10.1161/HYPERTENSIONAHA.111.00701

*Hypertension* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2013 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://hyper.ahajournals.org/content/61/2/e24

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Hypertension* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to *Hypertension* is online at:
http://hyper.ahajournals.org//subscriptions/