Letter by Tomczak and Haykowsky
Regarding Article, “Discrepancy Between Cardiac and Physical Functional Reserves in Stroke”

To the Editor:

Jakovljevic et al1 recently reported that peak cardiac output was not significantly different between patients with stroke and healthy control subjects despite a 31% lower peak oxygen uptake (VO₂) in patients with stroke. The authors1 attributed the latter finding to a reduction in muscle function and further suggested that rehabilitation interventions should target such dysfunction. A reduction in peak VO₂ is typical in chronic stroke, and thus improving VO₂ should notably be of interest for stroke rehabilitation.

A number of methodological and interpretive concerns need to be discussed in the context of the authors’ study1 and in comparison with the only previous work in this area.2

First, contrary to the authors’ interpretation of our previous publication,2 we assessed peak cardiac output in 7 of 10 subjects, not 4 as the authors stated. The authors question our finding that cardiac output truly was different at peak exercise between patients with stroke and control subjects. Further corroborating our finding that cardiac output was lower in patients with stroke, we found that a lower peak VO₂ was associated with slower postexercise VO₂ recovery kinetics (R = −0.72, P < 0.001), which supports the likelihood that an O₂ availability limitation existed in our patient group. Moreover, heart rate reserve was lower by 31 beats/min in our patient group and likely reflects some degree of β-receptor downregulation that is typical in cardiac deconditioning/dysfunction.3 Based on the Fick principle, heart rate contributes substantially to cardiac output. Thus, even if stroke volume reserve was preserved in patients with stroke (which we found it was not), our finding of a significantly lower peak and reserve heart rate2 would conceivably still cause a substantial reduction in peak and reserve cardiac output.

Lastly, our finding of a 41% reduction in minute ventilation reserve that was attributable to impaired tidal volume reserve in our patient group should not be ignored2 and further highlights that both cardiac and ventilatory mechanisms contribute to the reduction in peak VO₂ reported in patients with stroke.

Although the authors indicated their study limitations related to different exercise modalities and cardiac output estimation methods used between groups,1 the effect that these differences have on the strength of the authors’ data interpretation and subsequent conclusions cannot be understated.

In the authors’ study,1 patients with stroke and control subjects completed peak exercise differently (ie, cycle ergometry versus treadmill). Furthermore, data that could provide additional insight about how “maximal” the exercise actually was for control subjects were not provided. We found a greater reduction (43% lower) in peak VO₂ for patients with stroke2 than the authors report (31%); does this reflect a difference in exercise capacity for participants between studies or is it an effect of the different exercise protocols used by Jakovljevic et al?1 We suspect it is the latter.

The authors estimated cardiac output with different methods between the groups (ie, bioreactance versus CO₂ rebreathe).1 Despite the inherent error of each method, the study design “benefit” of systematic error from using only one method that would be present in both groups is lost. The authors’ findings and conclusions are confounded by using different methods for cardiac output estimation. Subsequently, the authors’ recommendation for rehabilitation strategies “targeting muscle oxygen uptake” in light of their statement that “cardiac function and pumping capability are maintained”1 warrants caution, because we have found that peak and reserve cardiovascular function may indeed be impaired in patients with chronic stroke.2

We strongly suggest that stroke rehabilitation should integrate exercise therapy that targets cardiorespiratory, peripheral vascular, and skeletal muscle function (not just skeletal muscle function as the authors’ suggest)1 to improve peak VO₂ and functional ability.

Disclosures

None.

Corey R. Tomczak, PhD
Department of Speech Pathology and Audiology
University of Alberta
Edmonton, Alberta, Canada

Mark J. Haykowsky, PhD
Faculty of Rehabilitation Medicine
University of Alberta
Edmonton, Alberta, Canada

Corey R. Tomczak and Mark J. Haykowsky

Letter by Tomczak and Haykowsky Regarding Article, "Discrepancy Between Cardiac and Physical Functional Reserves in Stroke"

*Stroke*. 2012;43:e91; originally published online June 28, 2012;
doi: 10.1161/STROKEAHA.112.660787

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/43/9/e91

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Stroke* 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 *Stroke* is online at:
http://stroke.ahajournals.org/subscriptions/