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

To the Editor:

Jakovljevic et al 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 (VO$_2$) in patients with stroke. The authors attributed the latter finding to a reduction in muscle function and further suggested that rehabilitation interventions should target such dysfunction. A reduction in peak VO$_2$ is typical in chronic stroke, and thus improving VO$_2$ should notably be of interest for stroke rehabilitation.

First, contrary to the authors’ interpretation of our previous publication, 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 VO$_2$ was associated with slower postexercise VO$_2$ recovery kinetics ($R=-0.72$, $P<0.001$), which supports the likelihood that an O$_2$ 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 $\beta$-receptor downregulation that is typical in cardiac deconditioning/dysfunction.

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 rate 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 ignored and further highlights that both cardiac and ventilatory mechanisms contribute to the reduction in peak VO$_2$ 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, the effect that these differences have on the strength of the authors’ data interpretation and subsequent conclusions cannot be understated. In the authors’ study, 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 VO$_2$ for patients with stroke 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? We suspect it is the latter.

The authors estimated cardiac output with different methods between the groups (ie, bioreactance versus CO$_2$ rebreathe). 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” warrants caution, because we have found that peak and reserve cardiovascular function may indeed be impaired in patients with chronic stroke.

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) to improve peak VO$_2$ 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


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Stroke. 2012;43:e91; originally published online June 28, 2012;
doi: 10.1161/STROKEAHA.112.660787
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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