Discrepancy Between Cardiac and Physical Functional Reserves in Stroke

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Background and Purpose—Understanding the physiological limitations to exercise after stroke will assist the development of targeted therapies to improve everyday function. This study defines (1) whether exercise capacity is limited by the cardiovascular system (oxygen supply) or skeletal muscles (oxygen utilization); and (2) cardiac function and pumping capability in people with stroke.

Methods—Twenty-eight male participants with mild ischemic stroke (70±6 years of age, 18±20 months poststroke) and 25 male, age-matched healthy control subjects performed a graded cardiopulmonary exercise test with gas exchange and noninvasive hemodynamic measurements. Maximal oxygen extraction was calculated as the ratio between peak oxygen consumption and peak cardiac output. Cardiac function and pumping capability were assessed by peak exercise cardiac power output (expressed in watts) and cardiac output.

Results—Peak oxygen consumption (18.4±4.6 versus 26.8±5.5 mL/kg/min, P<0.01) and arterial–venous O2 difference (9.3±2.5 versus 12.6±1.9 mL O2/100 mL of blood, P<0.01) were both reduced in stroke participants compared with healthy control subjects. In contrast, peak exercise cardiac power output (4.79±0.79 versus 4.51±0.96 W, P=0.49), cardiac output (16.4±3.1 versus 17.1±2.5 L/min, P=0.41), and the pressure-generating capacity of the heart (127±11 versus 125±14 mm Hg, P=0.97) were similar between stroke participants and healthy control subjects.

Conclusions—The ability of skeletal muscles to extract oxygen is diminished after stroke. However, cardiac function and pumping capability are maintained. Appropriate therapies targeting muscle oxygen uptake such as exercise rehabilitation may improve exercise capacity after stroke. (Stroke. 2012;43:1422-1425.)

Key Words: cardiac function ■ cardiac power ■ exercise performance ■ oxygen consumption

To develop appropriate therapies to improve everyday function and physical fitness, it is essential to understand the physiological limitations to exercise after stroke. Peak oxygen consumption (VO2) is the product of the capacity of the cardiovascular system to supply oxygen (ie, cardiac output [QT]) and the capacity of the skeletal muscles to use oxygen (ie, arterial–venous oxygen difference). In stroke survivors, peak oxygen consumption has found to be reduced by approximately 50%.1

Cerebrovascular disease can alter cardiovascular and autonomic regulation and compromise left ventricular performance.2,3 To date, limited information is available on measures of cardiac function and performance during exercise in stroke survivors.4 Cardiac power output, a direct and integrative measure of overall cardiac function (ie, cardiac pumping capability) that incorporates both pressure and flow domains of the cardiovascular system,5 has not been reported in stroke survivors yet. The aims of this study were to define (1) whether maximal aerobic capacity is limited by the cardiovascular system to supply oxygen or by the capacity of the skeletal muscles to use oxygen in these individuals; and (2) cardiac pumping capability in individuals after stroke.

Subjects and Methods

Twenty-eight male stroke survivors were enrolled in 2010 (Table). The study was approved by Durham and Tees Valley Research Ethics Committee and participants provided written informed consent.

Expired gases (Metalyzer 3B; Cortex, Leipzig, Germany) and bioreactance QT (NICOM; Cheetah Medical) were collected at rest for 5 minutes followed by a maximal progressive exercise test using an electromagnetically controlled recumbent bicycle ergometer (Corival; Lode, Groningen, The Netherlands). The exercise protocol included a warm-up, cycling at 20 W for 3 minutes,
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Independent-sample t tests were used for between-group comparison. The Pearson coefficient of correlation demonstrated relationships between variables. Statistical significance was indicated if \( P<0.05 \). All data are presented as means \( \pm \) SD unless otherwise indicated. Post hoc power analysis for the sample size of 28 demonstrated high power of the study (\( \beta=0.95 \)).

Results

Stroke participants completed the maximal exercise testing with no adverse events and they achieved 87% of their age-predicted maximal heart rate. The mean peak exercise respiratory exchange ratio and the Borg scale scores were 1.10 \( \pm \) 0.10 (range, 1.02–1.18) and 19.5 \( \pm \) 1.2, suggesting that the patients demonstrated a high level of exertion.

Peak \( \text{VO}_2 \) was 31% lower in stroke participants (18.4 \( \pm \) 4.6 versus 26.8 \( \pm \) 5.5 mL/kg/min, \( P<0.01 \); Figure 1A) and was accompanied by a reduction in peak exercise arterial–venous oxygen difference of 26% (9.3 \( \pm \) 2.5 versus 12.6 \( \pm \) 1.9 mL \( \text{O}_2/100 \) mL of blood, \( P<0.01 \); Figure 1B). Peak cardiac power output was not significantly different (\( P=0.49 \)) between control and stroke participants (Figure 1D) as were not \( Q_T \) (16.4 \( \pm \) 3.1 versus 17.1 \( \pm \) 2.5 L/min, \( P=0.41 \); Figure 1C) and mean arterial pressure (127 \( \pm \) 11 versus 125 \( \pm \) 14 mm Hg; \( P=0.97 \)).

Peak \( \text{VO}_2 \) highly correlated with \( Q_T \) in healthy subjects (\( r=0.84, P<0.01 \)) and only moderately in stroke participants (\( r=0.42, P<0.05 \); Figure 2).

Discussion

The 2 major findings of the present study suggest that in stroke participants (1) the ability of skeletal muscles to extract oxygen is diminished; and (2) cardiac pumping capability is maintained.

Our finding that peak exercise \( \text{VO}_2 \) is severely compromised in stroke survivors is in line with previous reports.\(^{3,8} \) The underlying physiological limitations of this finding have not been well defined, restricting the ability to target therapies. Some explanations include bedrest-induced deconditioning, concomitant left ventricular dysfunction, the associated severity of neurological involvement, and the increased aerobic requirements of walking.\(^{1} \) Cardiac function, measured at rest, has been found to be impaired after stroke\(^ {9} \) and cardiac dysfunction appears to be associated with severity of stroke.\(^ {3} \) Resting central hemodynamics, however, do not reflect peak \( \text{VO}_2 \) and pumping capability of the heart because they do not account for the reserve of cardiac function.\(^ {10} \) Only 1 previous study has assessed \( Q_T \) during exercise in people with stroke.\(^ {4} \) The authors argued that reduced peak \( \text{VO}_2 \) is secondary to a decline in peak and reserve \( Q_T \), which was assessed in only 4 patients and therefore needs to be considered with caution. The present study, however, suggests that peak cardiac function is not diminished, whereas maximal arterial–venous oxygen difference is reduced by 26% in stroke participants. This suggests that maximal aerobic capacity in people with stroke is likely due to reduced ability of the working muscles to extract oxygen. Only a moderate relationship...
between VO₂ and QT suggests that factors other than cardiac (ie, muscle) play an important role in the determination of maximal VO₂ in patients with stroke.

This study has several limitations. Patients with mild stroke were recruited and it remains to be elucidated whether cardiac pumping capability is preserved in those with severe residual disability. In contrast to our stroke participants, healthy control subjects performed a treadmill exercise test with CO₂ rebreathing cardiac output measurement. This may limit findings of the present study bearing in mind that the treadmill may elicit a 10% to 15% higher physiological response than cycling. However, this does not compromise our findings because peak VO₂ was reduced in patients with stroke by 31%.

In conclusion, the ability of skeletal muscles to extract oxygen is diminished after stroke but cardiac function and pumping capability are maintained. Appropriate therapies targeting muscle oxygen uptake such as exercise rehabilitation may improve exercise capacity after stroke.

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Disclosures

None.

References


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