Conclusions—The impaired peak $\dot{V}O_2$ in SP is secondary to a decline in peak and reserve cardiac output and ventilation.

Impact peak $\dot{V}O_2$ in addition to the recovery of $\dot{V}O_2$ after has not been evaluated. Circulatory impairments adversely compromised in acute SP, cardiac function during exercise, yet this relationship has not been established in SP. Additionally, it is not known whether impairments in the rate of muscle $O_2$ use. (Stroke. 2008;39:3102-3106.)

Key Words: cardiac output ■ exercise ■ oxygen uptake kinetics ■ stroke
where Y is the parameter at any time (t), \( \text{peak} \) is the greatest 30-s value of Y, and \( \text{rest} \) is the value of Y over 1 minute before exercise onset. A is the amplitude change in Y, \( T_{\text{d}} \) is the time to reach a 63% change in Y, and TD is the time delay before the exponential change in Y.

Phase II \( \dot{V}\text{O}_2, \dot{V}\text{CO}_2, \text{and } \dot{V}\text{E on-kinetics were determined and HR on-kinetics were measured from exercise onset. The best curve fit was defined by minimization of the residual sum of squares (Origin 7.5; OriginLab Corp, Northampton, Mass).}

Analysis of variance was used for between-group comparisons and correlation regression to determine variable relationships. Data are mean±SEM and \( P<0.05 \) was significant.

**Results**

Peak \( \dot{V}\text{O}_2, \dot{V}\text{CO}_2, \text{VE}, \text{HR, stroke volume (SV), Q, systemic vascular resistance, and power output were lower in SP**
The major new findings of this investigation are that (1) the power output was lower at the ventilatory threshold in SP (45 ± 4 versus 96 ± 5 W; P < 0.001).

VO₂ and VE off-kinetics were prolonged in SP (Figure A). VO₂ and VE off-kinetics were correlated (R = 0.80, P < 0.001) and VO₂ off-kinetics related to peak VO₂ (R = −0.72, P < 0.001) and peak Q (R = −0.75, P < 0.001).

The power output was lower during low-level exercise (26 ± 3 versus 59 ± 5 W; P < 0.001) and VO₂ on-kinetics slower in SP (Figure B). VO₂ (66 ± 10 versus 62 ± 6 s; P = 0.800), VE (63 ± 11 versus 66 ± 6 s; P = 0.764), and HR (45 ± 9 versus 32 ± 8 s; P = 0.307) on-kinetics were not different between SP and healthy control subjects, respectively. There was no relation between time poststroke and VO₂ on-kinetics (R = −0.60, P = 0.064).

### Discussion

The major new findings of this investigation are that (1) the reduction in peak and reserve VO₂ in SP is secondary to an impaired peak and reserve Q and VE; (2) prolonged VO₂ off-kinetics in SP are related to a reduced peak VO₂ and Q; and (3) SP have slower VO₂ on-kinetics during low-level exercise.

We demonstrate that impaired VO₂ in SP is secondary to a lower peak and reserve Q. The impaired Q reserve was secondary to an impaired HR reserve, because SV reserve was not different between groups (Table 2). Our finding of a similar SV reserve despite a higher systemic vascular resistance in SP (Table 2) is likely due to an enhanced preload or contractile reserve. This is plausible given that peak exercise end systolic volume likely increased secondary to an increased afterload. Additionally, systolic peak velocity is inversely related to afterload. Thus, the lower peak and reserve HR (Table 2) in our SP would lead to a greater diastolic filling time, preserving preload and SV reserve.

Peak and reserve VE was lower in SP secondary to an impaired peak and reserve VT, because peak and reserve breathing frequency were not different between groups (Table 2). Electromyographic activity is reduced in ventilatory muscles contralateral to the brain lesion side in SP and may be exacerbated during increases in VT.¹ Lanini and colleagues⁴ reported no asymmetrical effect of resting breathing on VT, which is consistent with our findings (Table 2). However, VT of the paretic chest wall may be reduced during hyperventilation.⁴ Peak and reserve VT was lower in our SP, suggesting the normal increase in breathing frequency throughout exercise exacerbated asymmetrical ventilation, thus contributing to an overall lower peak and reserve VT.

VO₂ remained elevated throughout recovery in SP (Figure A), reflecting a slower rate of skeletal muscle energy restitution. This is determined in part by the oxidative capacity and maximal rate of oxidative adenosine triphosphate synthesis of skeletal muscle, which may be mediated by aerobic fitness and the availability of O₂ for phosphocreatine regeneration.³ Indeed, a lower peak VO₂ was associated with prolonged VO₂ off-kinetics in our SP. Consistent with the hypothesis that O₂ availability may be rate-limiting for VO₂ recovery, peak Q and VO₂ off-kinetics were well correlated.

VCO₂ and VE off-kinetics were prolonged in SP (Figure A). Prolonged VCO₂ kinetics are explained by a greater CO₂ tension in the skeletal muscle secondary to buffering of exercise-induced lactate. This is consistent with the ventilatory threshold occurring at a lower exercise intensity in SP, suggesting a more immediate reliance on anaerobic glycolysis. The lower Q may also have exacerbated the delay in recovery VCO₂. Resulting CO₂ levels likely contributed to the

### Table 2. Pulmonary Gas Exchange, Ventilation, and Cardiovascular Parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Rest</th>
<th>Control</th>
<th>P Value</th>
<th>Peak Exercise</th>
<th>Control</th>
<th>P Value</th>
<th>Reserve</th>
<th>Control</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Power output, W</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>VO₂, mL/kg/min</td>
<td>3.2±0.3</td>
<td>3.7±0.2</td>
<td>0.149</td>
<td>16.0±1.2</td>
<td>28.1±2.0</td>
<td>&lt;0.001</td>
<td>12.8±1.2</td>
<td>24.4±2.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>VE, L/min</td>
<td>0.25±0.02</td>
<td>0.27±0.02</td>
<td>0.460</td>
<td>1.33±0.16</td>
<td>2.06±0.14</td>
<td>0.003</td>
<td>1.08±0.15</td>
<td>1.79±0.14</td>
<td>0.003</td>
</tr>
<tr>
<td>VCO₂, mL/kg/min</td>
<td>2.8±0.2</td>
<td>3.2±0.2</td>
<td>0.195</td>
<td>17.7±1.9</td>
<td>32.7±2.6</td>
<td>&lt;0.001</td>
<td>14.9±1.9</td>
<td>29.6±2.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>VE, L/min</td>
<td>9.6±0.6</td>
<td>9.5±0.7</td>
<td>0.934</td>
<td>49.4±6.4</td>
<td>76.5±7.9</td>
<td>0.016</td>
<td>39.8±6.2</td>
<td>67.0±7.6</td>
<td>0.013</td>
</tr>
<tr>
<td>Breathing frequency (breaths/min)</td>
<td>18±2</td>
<td>15±2</td>
<td>0.208</td>
<td>36±3</td>
<td>35±3</td>
<td>0.897</td>
<td>17±2</td>
<td>20±3</td>
<td>0.498</td>
</tr>
<tr>
<td>VT (L)</td>
<td>0.55±0.05</td>
<td>0.66±0.05</td>
<td>0.147</td>
<td>1.43±0.21</td>
<td>2.16±0.15</td>
<td>0.012</td>
<td>0.88±0.17</td>
<td>1.50±0.12</td>
<td>0.009</td>
</tr>
<tr>
<td>VO₂/VO₂</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>74±3</td>
<td>67±2</td>
<td>0.098</td>
<td>133±6</td>
<td>157±4</td>
<td>0.003</td>
<td>59±7</td>
<td>90±4</td>
<td>0.001</td>
</tr>
<tr>
<td>Stroke volume, mL/beat</td>
<td>59±3</td>
<td>69±2</td>
<td>0.008</td>
<td>79±3</td>
<td>94±2</td>
<td>0.001</td>
<td>19±2</td>
<td>25±3</td>
<td>0.128</td>
</tr>
<tr>
<td>Cardiac output, L/min</td>
<td>4.4±0.4</td>
<td>4.6±0.2</td>
<td>0.556</td>
<td>10.4±0.8</td>
<td>14.8±0.6</td>
<td>&lt;0.001</td>
<td>6.0±0.7</td>
<td>10.2±0.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>a-VO₂abs, mL/100 mL</td>
<td>6±1</td>
<td>6±0.5</td>
<td>0.629</td>
<td>11±1</td>
<td>14±1</td>
<td>0.106</td>
<td>6±1</td>
<td>8±1</td>
<td>0.152</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>123±10</td>
<td>123±4</td>
<td>0.972</td>
<td>165±16</td>
<td>191±9</td>
<td>0.143</td>
<td>42±15</td>
<td>68±8</td>
<td>0.117</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>78±4</td>
<td>76±3</td>
<td>0.751</td>
<td>81±4</td>
<td>93±5</td>
<td>0.123</td>
<td>3±5</td>
<td>15±4</td>
<td>0.109</td>
</tr>
<tr>
<td>Mean arterial pressure, mm Hg</td>
<td>93±5</td>
<td>92±3</td>
<td>0.870</td>
<td>109±7</td>
<td>126±6</td>
<td>0.098</td>
<td>16±8</td>
<td>32±5</td>
<td>0.092</td>
</tr>
<tr>
<td>SVR, dynes/cm²</td>
<td>1824±138</td>
<td>1607±82</td>
<td>0.172</td>
<td>906±85</td>
<td>687±36</td>
<td>0.016</td>
<td>−918±73</td>
<td>−933±69</td>
<td>0.886</td>
</tr>
</tbody>
</table>

*Data not obtained in 3 stroke subjects because of technical difficulties. Reserve values were calculated as peak exercise—rest. Data are mean±SEM. a-VO₂abs indicates systemic arterial–venous O₂ difference.
high VE, which is consistent with the well-correlated V˙CO₂ and V˙E off-kinetics.

V˙O₂ on-kinetics were slower in SP (Figure B), which indicates a slower rate of muscle O₂ consumption. Unfavorable changes in skeletal muscle function related to stroke may account for this observation, because Type I and II muscle fibers may be atrophied in SP. A consequence of the alteration in the Type I muscle fibers is an associated decline in mitochondrial density and oxidative capacity, which may account for the slower V˙O₂ on-kinetics.

Slower V˙O₂ on-kinetics may have been due to a decline in vascular function. Given that endothelial function may be impaired in SP, coupled with findings that VO₂ kinetics and blood flow become impaired with peripheral arterial disease, supports this hypothesis. Further investigation is required to establish the role of blood flow dynamics and mitochondrial function on muscle O₂ consumption in SP to further clarify the mechanisms responsible for our findings.

A limitation of our investigation is that comorbidities such as diabetes, hypertension, and chronic obstructive pulmonary disease may also prolong VO₂ kinetics. Additionally, the years poststroke may account for the slower VO₂ kinetics simply because of deconditioning secondary to inactivity. However, we matched SP and healthy control subjects on activity levels and the time poststroke and VO₂ kinetics were not related. Future investigations are required to establish the impact of comorbid conditions and stroke type on exercise cardiopulmonary function and VO₂ kinetics in SP.

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**Disclosures**
None.

**References**
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