Different blood flow responses to dynamic exercise between internal carotid and vertebral arteries in women

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METHODS

Ten healthy young women [22 ± 2 yr (mean ± SD), 164 ± 6 cm, 58 ± 4 kg, and peak oxygen uptake (\(V_{\text{O2peak}}\)): 38.3 ± 5.1 ml·kg\(^{-1}\)·min\(^{-1}\)] participated in this study. All procedures and protocols confirmed to Declaration of Helsinki and were approved by the Institutional Review Board at the Japan Women’s College of Physical Education. Following a detailed verbal explanation of the intended experimental measures and procedures, each subject gave informed, written consent before participation. The subjects were not performing endurance training on a regular basis. In addition, they were free of any known cardiovascular and pulmonary disorders and were not using prescribed or over-the-counter medications. Before the experiment, each subject visited the laboratory for familiarization with the CBF measurement by Doppler ultrasound and dynamic exercise protocol.

Aerobic power. The \(V_{\text{O2peak}}\) was determined by an incremental protocol on a cycle ergometer (Aerobike 800, Combi) 2 wk before the experiments. Subjects were exposed to an initial work rate of 30 W at a pace of 60 cycles/min. The subjects were told to maintain the frequency of pedaling, and work rate was increased 10–15 W every minute until volitional exhaustion. Respiratory variables were determined by breath by breath, and gas flows were analyzed by a mass spectrometer (ARCO-1000, Arco System), while expired gas volume was measured by a Fleisch pneumotachometer (WLCU-5201, We-
The highest value obtained for oxygen uptake ($\dot{V}O_2$) over 30 s was taken as $V_\text{O2peak}$.

**Exercise and experimental protocol.** The subjects were seated on a semisupine cycle ergometer (Cateye-Ergociser EC-3700, Cateye) with a backrest inclination of $\sim 40-50^\circ$. The upper body of the subject was held by shoulder straps and a waist belt to the cycle frame, and head and neck were also held in a stable position by a padded head rest (Fig. 1). The procedure included a 5-min baseline period, followed by exercise with loads of 30, 50, and 70% of $V_\text{O2peak}$, with each stage lasting 5 min. This graded dynamic exercise was followed by a further 3 min of recovery period in a constant position.

**Cerebral blood flow.** The measurements of CBF in this study were carried out during the rest (for 2 min between the 2nd and 4th min), the exercise stage (for 1 min between the 4th and 5th min) and the recovery (for 1 min between the 2nd and 3rd min). The representative values of CBF at each period were the average of three recordings taken.

The $Q_{\text{ICA}}$ was measured with a high-resolution ultrasound system (Vivid 7 Pro, GE Yokogawa Medical Systems) equipped with a 10-MHz linear transducer. Measurements were performed $\sim 1.0-1.5$ cm distal to the carotid bifurcation on the right ICA, while the subject’s chin was slightly elevated (Fig. 2). We first used brightness mode to measure the mean vessel diameter of ICA ($D_{\text{ICA}}$) in the longitudinal section, and, thereafter, the Doppler velocity spectrum was identified by pulsed wave Doppler mode. The systolic and diastolic diameters of the ICA were measured, and the $D_{\text{ICA}}$ was calculated in relation to the blood pressure curve: $D_{\text{ICA}} = \text{systolic diameter} \times 1/3 + \text{diastolic diameter} \times 2/3$. Moreover, the time-averaged mean flow velocity obtained by the pulsed wave Doppler mode was defined as the mean blood flow velocity ($V_{\text{ICA}}$; m/s). The recordings of the $V_{\text{ICA}}$ were taken from the average of $\sim 10$ cardiac cycles to eliminate the effects caused by the breathing cycle. In $V_{\text{ICA}}$ measurement, care was taken to ensure that the probe position was stable, that the insonation angle did not vary (in most cases, $60^\circ$), and that the sample volume was positioned in the center of the vessel and adjusted to cover the width of the vessel diameter. Three data of $D_{\text{ICA}}$ and $V_{\text{ICA}}$ were obtained for rest and for the last 1 min of exercise with each workload and for the last 1 min of the recovery period, and then the average of three data was defined as the representative value of $D_{\text{ICA}}$ and $V_{\text{ICA}}$ in the individual period. $Q_{\text{ICA}}$ was calculated by multiplying the cross-sectional area [$\pi \times (D_{\text{ICA}}/2)^2$] with $V_{\text{ICA}}$: $Q_{\text{ICA}} = V_{\text{ICA}} \times \text{area} \times 60$ (ml/min).

The $Q_{\text{VA}}$ was measured with a similar Doppler ultrasound systems (Vivid e, GE Yokogawa Medical Systems) equipped with a 10-MHz linear transducer. Measurements were mainly performed between the transverse processes of the C4 and C5 vertebrae on the left side, and the $Q_{\text{VA}}$ was calculated as described for $Q_{\text{ICA}}$. To avoid the ultrasound interference, we chose the right ICA and left VA for CBF measurement. In a pilot study, we confirmed no significant differences in blood flow volume in the left and right side of ICA, whereas the left VA tended to have a larger blood flow than the right VA (31). All of CBF measurements were performed by the same two experienced operators (28, 29).

The coefficients of variation (CV) in $Q_{\text{ICA}}$ and $Q_{\text{VA}}$ were 5.3 $\pm$ 1.2 and 5.8 $\pm$ 1.0% at rest, 6.1 $\pm$ 0.9 and 6.3 $\pm$ 1.2% at 30% $V_{\text{O2peak}}$, 5.9 $\pm$ 0.7 and 5.8 $\pm$ 1.0% at 50% $V_{\text{O2peak}}$, and 5.3 $\pm$ 0.7 and 6.3 $\pm$ 0.6% at 70% $V_{\text{O2peak}}$, respectively. Moreover, we carried out a test-retest experiment to confirm the reproducibility of $Q_{\text{ICA}}$ and $Q_{\text{VA}}$ measurement at rest and during dynamic exercise.

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**Fig. 1.** Position of the subject while semisupine cycle exercising (A), and the head position for cerebral blood flow measurement (B).

**Fig. 2.** Ultrasound Doppler screen while the internal carotid (ICA; A) and vertebral artery (VA; C) blood flow are measured. B: probe and head placement.
exercise in the pilot study \( (n = 6) \). Such determinations of Q_{ICA} and Q_{VA} are made with an average CV of 4.6 ± 10% for Q_{ICA} and 5.3 ± 13% for Q_{VA} at rest, 6.3 ± 0.9 and 6.2 ± 1.3% at 30% VO_{2peak}, 5.0 ± 0.9 and 4.9 ± 1.2% at 50% VO_{2peak}, and 6.4 ± 0.8 and 6.5 ± 1.1% at 70% VO_{2peak}, respectively. The intraclass correlation coefficient of the repeated measurements of Q_{ICA} was 0.94, and that in Q_{VA} was 0.96, respectively. The CVs in Q_{ICA} and Q_{VA} at rest and during dynamic exercise were within the range of reported values at rest and all workloads \( (11, 13, 14, 31) \).

Cardiorespiratory responses. Heart rate was continuously monitored using a three-lead electrocardiogram \( (\text{OEC-6401, Nihon Koden}) \). Beat-to-beat blood pressure was measured using finger photoplethysmography obtained from the middle or index finger of the nondominant hand \( \text{(Finometer, Finapres Medical Systems)} \). These methods of blood pressure measurement have been validated for use both at rest and during low to moderate level of exercise \( (5) \). Furthermore, stroke volume and CO were determined from the blood pressure waveform obtained from the middle or index finger of the nondominant hand \( \text{(Finometer, Finapres Medical Systems)} \). This method provides a reliable estimate of change in stroke volume and CO in healthy humans at rest and during moderate exercise \( (15, 16, 36) \). Respiratory variables were determined as described for VO_{2peak}, and end-tidal partial pressure of CO_{2} (PETCO_{2}) was measured. The cardiorespiratory responses at rest were analyzed over 2 min that ended 1 min before the onset of exercise. During exercise, these parameters were analyzed within the last 1 min of each workload. Furthermore, the data of the last 1 min of the recovery period were analyzed.

Data processing and statistics. The ratio of mean arterial pressure \( (\text{MAP}) \) at ICA level to Q_{ICA} and the ratio of MAP at VA level to Q_{VA} were, respectively, taken as indexes of cerebrovascular resistance \( \text{(CVR}_{\text{ICA}} \text{ and CVR}_{\text{VA}}) \). The MAP at ICA level or VA level took into consideration the vertical distance from the fourth intercostal space in the midclavicular line \( \text{(heart level)} \) to the Doppler probe \( \text{(i.e., hydrostatic pressure = the vertical distance \times 0.77 mmHg/cm)} \) \( (25) \). The gCBF was calculated as the sum of volume flow in ICA and VA \( \left[ (Q_{\text{ICA}} + Q_{\text{VA}}) \times 2 \right] \text{ (ml/min)} \) \( (7) \). The distribution of CO to brain was expressed as gCBF/CO \( \times 100 \) (%). The relative contribution of Q_{ICA} and Q_{VA} to gCBF was estimated as \( Q_{\text{ICA}}/\text{gCBF} \times 100 \) (%) and \( Q_{\text{VA}}/\text{gCBF} \times 100 \) (%), respectively.

The cerebrovascular and cardiorespiratory responses at rest were analyzed over 2 min that ended 1 min before the onset of exercise. During exercise, these parameters were analyzed from the last 1 min of each workload and also expressed relative to rest. In addition, the data of the last 1 min of recovery period were analyzed.

Values are expressed as means ± SE, and differences between values at rest, exercise, and recovery were evaluated by ANOVA with repeated measures and Dunnett post hoc test. To compare differences between changes in the cerebrovascular responses in the ICA and VA, two-way repeated-measures ANOVA were used. If the data were normally distributed, a two sample \( t \)-test was performed. Otherwise, Wilcoxon signed-rank test was used \( (\text{SPSS12.0, SPSS}) \), and \( P < 0.05 \) was considered to indicate a significant difference.

**RESULTS**

The resting values, the change in the cardio-respiratory and cerebrovascular responses to graded exercise, and the recovery are shown in Table 1. VO_{2}, MAP, heart rate, and CO increased with workload \( (P < 0.01) \) and also PETCO_{2} was higher than at rest \( (P < 0.01) \). Yet, at the 70% VO_{2peak} workload, PETCO_{2} was lower than at 50% VO_{2peak} \( (\text{from 45.3 ± 1.4 to 43.2 ± 1.6 Torr}; P < 0.05) \). All cardio-respiratory variables in the recovery were lower than during exercise, but they remained higher than at rest.

We confirmed whether metabolism had reached steady state at each workload by difference in VO_{2} between the 4th or 5th min of each exercise stage. According to this results, there were no significant differences in VO_{2} between the 4th or 5th min of exercise during 30 and 50% VO_{2peak}, suggesting that a steady-state VO_{2} was achieved during 30 and 50% VO_{2peak}. However, 70% VO_{2peak} did not show a steady-state VO_{2}.

With exercise, Q_{ICA} increased \( (P < 0.01; \text{Table 1 and Fig. 3A}) \). At 30% VO_{2peak}, the increase was by 11.6 ± 1.5%, and it then

Table 1. Cardiorespiratory and cerebrovascular variables at rest, during dynamic exercise, and at recovery

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rest</th>
<th>30% VO_{2peak}</th>
<th>50% VO_{2peak}</th>
<th>70% VO_{2peak}</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO_{2}, ml/min</td>
<td>236 ± 2</td>
<td>1335 ± 68*</td>
<td>1728 ± 77*</td>
<td>377 ± 27*</td>
<td></td>
</tr>
<tr>
<td>MAP, mmHg</td>
<td>77 ± 2</td>
<td>102 ± 3*</td>
<td>114 ± 2*</td>
<td>83 ± 2</td>
<td></td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>60 ± 2</td>
<td>127 ± 3*</td>
<td>159 ± 3*</td>
<td>79 ± 3*</td>
<td></td>
</tr>
<tr>
<td>CO, 1/min</td>
<td>4.4 ± 0.5</td>
<td>11.5 ± 0.4*</td>
<td>16.1 ± 0.6*</td>
<td>6.5 ± 0.2*</td>
<td></td>
</tr>
<tr>
<td>PETCO_{2}, Torr</td>
<td>40.3 ± 0.9</td>
<td>45.3 ± 1.4*</td>
<td>43.2 ± 1.6*</td>
<td>42.7 ± 2.6*</td>
<td></td>
</tr>
<tr>
<td>Q_{ICA}, ml/min</td>
<td>295 ± 20</td>
<td>349 ± 25*</td>
<td>344 ± 21*</td>
<td>311 ± 23*</td>
<td></td>
</tr>
<tr>
<td>Change from rest, %</td>
<td>0</td>
<td>18.4 ± 2.7</td>
<td>17.2 ± 2.0</td>
<td>5.1 ± 2.2</td>
<td></td>
</tr>
<tr>
<td>D_{ICA}, cm</td>
<td>0.48 ± 0.02</td>
<td>0.49 ± 0.02</td>
<td>0.49 ± 0.02</td>
<td>0.48 ± 0.02</td>
<td></td>
</tr>
<tr>
<td>V_{ICA}, cm/s</td>
<td>28.0 ± 2.5</td>
<td>31.1 ± 2.8*</td>
<td>31.7 ± 2.7*</td>
<td>29.6 ± 2.8</td>
<td></td>
</tr>
<tr>
<td>Q_{VA}, ml/min</td>
<td>95 ± 5</td>
<td>126 ± 7*</td>
<td>122 ± 8*</td>
<td>109 ± 7*</td>
<td></td>
</tr>
<tr>
<td>Change from rest, %</td>
<td>0</td>
<td>32.8 ± 3.6</td>
<td>39.5 ± 3.4</td>
<td>15.8 ± 4.0</td>
<td></td>
</tr>
<tr>
<td>D_{VA}, cm</td>
<td>0.32 ± 0.01*</td>
<td>0.33 ± 0.01*</td>
<td>0.33 ± 0.01*</td>
<td>0.33 ± 0.01*</td>
<td></td>
</tr>
<tr>
<td>V_{VA}, cm/s</td>
<td>19.9 ± 0.9</td>
<td>24.5 ± 1.0*</td>
<td>25.3 ± 1.0*</td>
<td>21.2 ± 0.7</td>
<td></td>
</tr>
<tr>
<td>CVR_{ICA}, mmHg/ml^{-1} \cdot min^{-1}</td>
<td>0.26 ± 0.02</td>
<td>0.30 ± 0.03*</td>
<td>0.34 ± 0.03*</td>
<td>0.27 ± 0.02</td>
<td></td>
</tr>
<tr>
<td>CVR_{VA}, mmHg/ml^{-1} \cdot min^{-1}</td>
<td>0.81 ± 0.05</td>
<td>0.82 ± 0.05</td>
<td>0.87 ± 0.05</td>
<td>0.76 ± 0.05</td>
<td></td>
</tr>
<tr>
<td>gCBF, ml/min</td>
<td>779 ± 40</td>
<td>950 ± 52*</td>
<td>952 ± 45*</td>
<td>840 ± 47*</td>
<td></td>
</tr>
<tr>
<td>Change from rest, %</td>
<td>0</td>
<td>12.9 ± 1.5</td>
<td>21.9 ± 2.5</td>
<td>7.6 ± 1.6</td>
<td></td>
</tr>
<tr>
<td>gCBF/CO \times 100, %</td>
<td>17.7 ± 12</td>
<td>8.4 ± 0.6*</td>
<td>6.4 ± 0.4*</td>
<td>13.0 ± 0.8*</td>
<td></td>
</tr>
<tr>
<td>Q_{ICA}/gCBF, %</td>
<td>75.2 ± 18</td>
<td>73.0 ± 2.0</td>
<td>71.9 ± 1.8*</td>
<td>73.4 ± 2.0</td>
<td></td>
</tr>
<tr>
<td>Q_{VA}/gCBF, %</td>
<td>24.8 ± 18</td>
<td>25.7 ± 2.0</td>
<td>27.0 ± 2.0</td>
<td>28.1 ± 1.8*</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SE, VO_{2}, oxygen uptake; VO_{2peak}, peak VO_{2}; MAP, mean arterial pressure; HR, heart rate; CO, cardiac output; PETCO_{2}, end-tidal partial pressure of CO_{2}; Q_{ICA} and Q_{VA}, blood flow in internal carotid arteries \( \text{(ICA)} \) and vertebral arteries \( \text{(VA)} \), respectively; V_{ICA} and V_{VA}, mean blood flow velocities in the ICA and VA, respectively; D_{ICA} and D_{VA}, mean diameter in ICA and VA, respectively; CVR_{ICA} and CVR_{VA}, index of cerebrovascular resistance in the ICA and VA, respectively; gCBF, global cerebral blood flow \( \left[ (Q_{\text{ICA}} + Q_{\text{VA}}) \times 2 \right] \text{ gCBF/CO} \times 100 \) (%), the distribution of CO to brain; Q_{ICA}/gCBF (%), the relative contribution of Q_{ICA} to gCBF; Q_{VA}/gCBF (%), the relative contribution of Q_{VA} to gCBF. *Different from rest \( (P < 0.05) \).
increased further to 18.4 ± 2.7% above rest at 50% \( \dot{V}O_2\text{peak} \). However, at 70% \( \dot{V}O_2\text{peak} \), QICA leveled off (17.2 ± 2.0%). There were no significant changes in the gCBF in the recovery remained above the resting value by 1.5%. At 30% \( \dot{V}O_2\text{peak} \), gCBF was elevated by 21.9 ± 2.5%, but at 70% \( \dot{V}O_2\text{peak} \), the increase in gCBF leveled off (22.5 ± 2.0%) (Table 1). gCBF in the recovery remained above the resting value by 7.6 ± 1.6% (\( P < 0.01 \)). At rest, gCBF accounted for 17.7 ± 1.2% of CO, but, during exercise, a progressive decrease was observed: from 10.3 ± 0.7% at 30% \( \dot{V}O_2\text{peak} \) to 6.4 ± 0.4% at 70% \( \dot{V}O_2\text{peak} \), and 13.0 ± 0.8% in the recovery. The QVA/gCBF was 24.8 ± 1.8% at rest, 26.7 ± 2.0% at 30% \( \dot{V}O_2\text{peak} \), 27.0 ± 2.0% at 50% \( \dot{V}O_2\text{peak} \), 28.1 ± 1.8% at 70% \( \dot{V}O_2\text{peak} \), and 29.6 ± 2.0% at recovery, and QVA/gCBF at 70% \( \dot{V}O_2\text{peak} \) was significantly different from that at rest (\( P < 0.05 \)).

**DISCUSSION**

Using Doppler ultrasound, we examined the simultaneous blood flow responses in ICA and VA during graded dynamic exercise. The major findings of the present study were that the increase in QICA leveled off over the intensity of 50% \( \dot{V}O_2\text{peak} \), whereas the continuous increase in QVA occurred until an intensity of 70% \( \dot{V}O_2\text{peak} \), and that the CVRICA increased with increasing exercise load, whereas CVRVA remained stable throughout the graded intensities. These results confirmed our hypothesis that the cerebrovascular responses to dynamic exercise are different between ICA and VA systems. Furthermore, we found that gCBF was elevated ~20% during dynamic exercise, and that the relative contribution of ICA and VA systems to the gCBF varied during dynamic exercise.

**Differential responses between the QICA and QVA during dynamic exercise.** The increase in QICA leveled off over an intensity of 50% \( \dot{V}O_2\text{peak} \) during cycling exercise, which was consistent with the previous report (11). However, the QVA increased progressively with graded intensities of exercise up to 70% \( \dot{V}O_2\text{peak} \). These different cerebrovascular responses between ICA and VA during dynamic exercise are probably mediated by several factors and/or mechanisms. The first possible explanation is that the neurormetabolic demand in the brain was regionally different between the territories covered by the ICA system and the territories covered by the VA system and thereby resulted in different cerebrovascular responses between ICA and VA. The previous animal studies support this explanation (4, 8). Delp et al. (4) reported that the blood flow in the cortical areas showed less increase to maximal exercise than that in the brain stem, spinal cord, and the cerebellum. A second possibility is that anatomic differences might exist between ICA and VA systems. In line with this explanation, the histological studies of Edvinsson et al. (6) have shown regional differences concerning density of \( \beta \)-adrenergic, cholinergic, and serotoninergic innervation of the intracerebral vessels, which may have different influences on the cerebrovascular resistance. In addition, pharmacological studies have suggested regional differences in the sensitivity to vasoactive substances, e.g., noradrenaline (9). The third factor is the difference in cerebral CO2 reactivity (3, 18, 35) and/or autoregulatory control (10, 21) between ICA and VA systems in humans. The present study indicates that cerebral CO2 reactivity during moderate exercise is reduced in the VA system compared with the ICA system (35). In addition, previous studies have demonstrated impaired autoregulation in the VA system compared with the ICA system (10). Thus several factors probably contributed to the different cerebrovascular responses observed between the ICA and VA systems during dynamic exercise in the present study. Further research is required to clarify the detail mechanisms.

**gCBF responses during dynamic exercise.** We found that gCBF, calculated as the sum of QICA and QVA, elevated ~20%
during dynamic exercise, despite different contribution of ICA and VA to the increased gCBF during exercise. Our results are consistent with the previous findings that gCBF increased by \(\sim 20-25\%\) during moderate intensity of \(\sim 50-60\%\ V_{O_2\text{peak}}\), but not during a higher intensity over \(70\% V_{O_2\text{peak}}\) (11, 17, 22, 26, 33). The increase in gCBF during dynamic exercise reflected \(Q_{ICA}\) leveling off at moderate exercise intensity and is attributed to hyperventilation-induced decreases in \(PETCO_2\) (11, 17, 22, 26, 33). In contrast to our findings, studies using the Kety-Schmidt method to express gCBF as the internal jugular venous flow found no change in dynamic exercise (17, 20, 26, 33, 34). This discrepancy, most likely, reflects that the internal jugular vein is collapsed in the upright position used in human exercise studies (2), and blood is transmitted to an alternative venous pathway (i.e., the spinal veins) (37). In addition, evaluation of CBF by the Kety-Schmidt method is complicated by the asymmetry of the venous drainage from the brain (33).

Although arterial blood supply at rest appears to be balanced between the ICA and VA systems in humans (32), our new finding was that the relative contribution of blood to gCBF via the two systems may vary from rest to dynamic exercise. Furthermore, we observed that the decrease in the distribution of CO to the brain (gCBF/CO) from rest (\(\sim 18\%\)) to moderate exercise (\(\sim 6\%\)) and these changes depended on the exercise intensity (27). CO is an important factor that can influence CBF during dynamic exercise (15, 16, 23, 24, 38). However, CO influence on CBF was more pronounced at rest than during dynamic exercise (23), and our results regarding the distribution of CO to brain might be associated with these observations.

**Limitations.**

The present study has several limitations. First, we did not measure the CBF up to maximal exercise (100\% \(V_{O_2\text{peak}}\)), because the CBF measurements during maximal exercise showed a large variation due to changes in the probe position and the insonation angle of the ultrasound beam during body movements. However, the CV and test-retest reproducibility in our CBF measurements indicated that the data obtained at rest and during submaximal exercise were reliable in this study. Second, the subjects in the present study were only 10 women. Thus, if data from male subjects were added, the cerebrovascular responses in ICA and VA systems during exercise could be generalized. Although there was no sex differences in the \(Q_{ICA}\) and \(Q_{VA}\) at rest (30, 32), further investigations are required in both systems during dynamic exercise. Third, we used photoplethysmography and Modelflow methods to estimate MAP and CO responses to exercise. Although these methods were validated in previous studies for an estimation of MAP and CO at rest and during dynamic exercise (5, 15, 16, 36), these techniques have some limitations (1, 12).

In summary, during dynamic exercise, the increase in the \(Q_{ICA}\) leveled off at moderate dynamic exercise over the intensity of 50\% \(V_{O_2\text{peak}}\), whereas the \(Q_{VA}\) progressively increased up to the intensity of 70\% \(V_{O_2\text{peak}}\). These results indicated that the cerebrovascular responses to dynamic exercise are different between ICA and VA systems. Moreover, the relative contribution of \(Q_{ICA}\) decreased and \(Q_{VA}\) increased to gCBF as exercise intensity increased, yet \(Q_{ICA}\) still accounted for the majority of cerebral perfusion.

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**DISCLOSURES**

No conflicts of interest, financial or otherwise, are declared by the author(s).

**REFERENCES**


