Continuous monitoring of vertebrobasilar hemodynamics utilizing TCDS transducer holder Sonopod during postural changes

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Abstract

Background: The objective was to evaluate continuous monitoring in the vertebrobasilar arteries (VBA), utilizing the transducer holder Sonopod for transcranial color duplex sonography (TCDS), vertebrobasilar hemodynamics and autoregulation, during postural changes.

Methods: Subjects were five normal controls and seven patients: two patients with arterial hypertension, three with dizziness (peripheral neuropathy, hepatic cirrhosis, and unknown), one with lacunar infarction and diabetes mellitus (LI/DM), and one with spino-cerebellar degeneration (SCD). TCDS utilizing the transducer holder Sonopod was used to continuously monitor the intracranial VA and BA. Blood pressure (BP), heart and respiration rates were also monitored. During two series of postural changes (supine or sitting to/from standing), a) clinical symptoms, b) BP: systolic, mean, and diastolic pressures (SBP, MBP, and DBP), c) TCDS: time-averaged maximum velocity (Vmax) and pulsatility index (PI), estimated cerebrovascular resistance (eCVR) = MBP/Vmax, and autoregulation index (ARI) = %ΔeCVR/%ΔMBP, were all calculated on the basis of maximum and minimum values during both series and of separate values from sitting to standing.

Results: a) Severe dizziness resulted in an inability to remain standing in two patients (LI/DM and SCD). b) BP: 1) ΔDBP >10mmHg in all cases. 2) ΔSBP>20mmHg in 2 controls and all but one patient (LI/DM). c) TCDS: 1) ΔPI and ΔeCVR tended to increase in the two severe dizziness patients. 2) ARI for both normal control subjects and patients fluctuated in all series and during individual standing.

Conclusion: Continuous TCDS monitoring in the VBA during postural changes is capable of evaluating vertebrobasilar autoregulation associated with autonomic regulation.

Keywords: Transcranial color duplex sonography, Transducer holder sonopod, Vertebrobasilar artery, Autoregulation, Postural changes.
Introduction

Orthostatic intolerance is associated with various symptoms caused by hypotension during postural changes due to autonomic dysregulation [1]. The hypothesis is that these symptoms result from cerebral dysautoregulation in the vertebrobasilar artery (VBA) system. Autoregulation (AR) in the VBA has been studied in normal subjects utilizing a hand-held probe with conventional transcranial Doppler sonography (TCD) [2-5].

Recently, continuous suboccipital monitoring with a transducer fixation device has been introduced for the evaluation of vasoreactivity [6, 7] and detection of high intensity transient signals [8]. We have introduced and improved a transducer holder, named the Sonopod, for transcranial color duplex sonography (TCDS) monitoring via both temporal/suboccipital windows [9, 10]. However, no AR study has yet been carried out in the VBA utilizing a transducer holder for TCDS.

The objective of this study is to clarify the significance of continuous monitoring in the VBA, utilizing the transducer holder Sonopod for TCDS, and in this way vertebrobasilar hemodynamics and autoregulation were evaluated during postural changes.

Methods

Subjects were five normal controls and seven patients (aged 23-75, mean 53 years); two patients had hypertension, three had dizziness (peripheral neuropathy, liver cirrhosis, and unknown), one had a lacunar infarction and diabetes mellitus (LI/DM), and one had spino-cerebellar degeneration (SCD). TCDS utilizing the transducer holder Sonopod has monitored continuously the intracranial vertebral artery (VA) and basilar artery (BA). Blood pressure (BP), heart and respiratory rates were also monitored. During two series of postural changes (supine or sitting for 3-5 minutes to/from standing for 3-5 minutes), it was registered a) clinical symptoms, b) BP: systolic, mean, and diastolic pressures (SBP, MBP, and DBP), c) TCDS: time-averaged maximum velocity (Vmax) and pulsatility index (PI), estimated cerebrovascular resistance (eCVR) = MBP/Vmax. Autoregulation index (ARI) = %ΔeCVR/%ΔMBP were calculated on the basis of maximum and minimum values during both series: %ΔeCVR = (eCVR maximum - eCVR minimum)/eCVR minimum and %ΔBP = (BP maximum - BP minimum)/BP minimum.

Also individual ARIs during two series of standing (= %ΔeCVR/%ΔMBP) were based on separate values from sitting (or supine) to standing: %ΔeCVR = (eCVR standing - eCVR sitting or supine)/eCVR sitting or supine and %ΔBP = (BP standing - BP sitting or supine)/BP sitting or supine.

Results

a) Clinical symptoms: Severe dizziness resulted in an inability to remain standing in two patients (LI/DM and SCD). No symptoms during postural changes were observed in the remaining 5 patients or 5 normal control subjects.

b) BP (Table 1): DBP increased at least 10mmHg in all cases. SBP increased at least 20 mmHg in 2 normal controls and in all but one patient (LI/DM). Hypotension during standing was remarkable only in the SCD patient.

c) TCDS (Table 1): ΔPI and ΔeCVR tended to increase in the two severe dizziness patients. ARIs in both normal control subjects and patients fluctuated in all series and during individual standing.

Table 1. Demographic characteristics of the study population.

<table>
<thead>
<tr>
<th>Case</th>
<th>Age/ Sex</th>
<th>Diagnosis</th>
<th>Artery</th>
<th>ΔSBP (mmHg)</th>
<th>ΔMBP (mmHg)</th>
<th>ΔDBP (mmHg)</th>
<th>ΔVmax (cm/s)</th>
<th>ΔPI</th>
<th>ΔeCVR</th>
<th>%ΔeCVR</th>
<th>%ΔMBP</th>
<th>ARI</th>
<th>ARList1</th>
<th>ARList2</th>
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<td>DY</td>
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<td>Normal</td>
<td>BA</td>
<td>19</td>
<td>21</td>
<td>10</td>
<td>5.3</td>
<td>0.33</td>
<td>1.00</td>
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<td>0.33</td>
<td>1.70</td>
<td>1.62</td>
<td>-0.05</td>
</tr>
<tr>
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<td>24F</td>
<td>Normal</td>
<td>BA</td>
<td>11</td>
<td>10</td>
<td>16</td>
<td>11.2</td>
<td>0.75</td>
<td>0.49</td>
<td>0.37</td>
<td>0.12</td>
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<td>2.21</td>
<td>-9.54</td>
</tr>
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<td>LVA</td>
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<td>31</td>
<td>13</td>
<td>8.2</td>
<td>0.61</td>
<td>1.55</td>
<td>0.66</td>
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<td>2.18</td>
<td>1.94</td>
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<td>Normal</td>
<td>BA</td>
<td>34</td>
<td>32</td>
<td>18</td>
<td>22.5</td>
<td>0.50</td>
<td>1.60</td>
<td>0.98</td>
<td>0.39</td>
<td>2.51</td>
<td>3.39</td>
<td>2.50</td>
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<tr>
<td>YN</td>
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<td>23</td>
<td>10</td>
<td>16.0</td>
<td>0.40</td>
<td>0.44</td>
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<td>1.20</td>
<td>0.95</td>
<td>1.47</td>
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<tr>
<td>MN</td>
<td>65F</td>
<td>HT</td>
<td>HT</td>
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<td>1.23</td>
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<tr>
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<td>60M</td>
<td>HT</td>
<td>RVA</td>
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<td>18</td>
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<td>BA</td>
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<td>75M</td>
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<td>1.73</td>
<td>0.24</td>
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<td>ND</td>
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<td>SCD</td>
<td>RVA</td>
<td>40</td>
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<td>13</td>
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<td>dizziness/ PN</td>
<td>RVA</td>
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<td>5.0</td>
<td>0.30</td>
<td>0.74</td>
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<tr>
<td>HI</td>
<td>36M</td>
<td>SCD</td>
<td>RVA</td>
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<td>14</td>
<td>9.0</td>
<td>0.49</td>
<td>0.34</td>
<td>0.21</td>
<td>0.29</td>
<td>0.72</td>
<td>0.60</td>
<td>4.95</td>
</tr>
</tbody>
</table>

HT = Hypertension; SCD = Spino-cerebellar degeneration; LI = Lacunar infarction; DM = Diabetes mellitus; LC = Liver cirrhosis; PN = Peripheral neuropathy; BA = Basilar artery; L = Left; R = Right; VA = Vertebral artery; SBP = Systolic blood pressure; MBP = Mean blood pressure; DBP = Diastolic blood pressure; Vmax = Time-averaged maximum velocity; PI = Pulsatility index; eCVR = Estimated cerebrovascular resistance; Δ = Maximum-minimum; %ΔeCVR = (eCVR maximum - eCVR minimum)/eCVR minimum; %ΔMBP = (MBP maximum - MBP minimum)/MBP minimum; ARI = Autoregulation Index (%ΔeCVR, %ΔMBP); ARList1 = ARI during standing (Vmax sitting or supine/Vmax standing-BP sitting or supine)/BP standing/IT- BP sitting or supine/BP standing); ND = No data
Falling BP and Vmax and increasing PI and eCVR during two series of standing were distinctive in the SCD patient (Figure 1). Calculated ARI on the basis of maximum and minimum values was 2.88 during all series. However, ARIs during individual standing was -12.3 and -1.47, respectively.

**Discussion**

**Continuous suboccipital monitoring and transducer fixation device**

Evaluation of cerebral vasomotor reactivity, such as AR, in the BA, via the suboccipital window, has been evaluated by conventional TCD with a labor-intensive hand-held probe utilizing thigh cuffs [2], phenylephrine infusion [4], and change in position from supine to sitting [5]. In order to perform accurate and reproducible evaluation, continuous monitoring utilizing a transducer fixation device is needed. Recently, continuous suboccipital monitoring with a transducer fixation device has been introduced for evaluation of CO₂ reactivity [6], autoregulation in migraines [7], and detection of high intensity transient signals [8].

Compared to conventional TCD, TCDS is able to measure much more accurately on the basis of angle-collected velocities in the intracranial major vessels via both the temporal and suboccipital bone windows. We have introduced and improved a transducer holder, named Sonopod, for TCDS monitoring via both temporal/suboccipital bone windows [9, 10]. In this study, transducer displacement was not problematic in all cases despite position changes from supine or sitting to standing. However, in order to avoid transducer displacement, it is necessary to use a semi-lateral, lateral or sitting position instead of a pure supine position. Furthermore, in this study, we had to monitor in the VA in 4 out of 12 cases due to a limitation of fixation angle. Future improvement of the Sonopod is necessary in this matter.

**Static and dynamic autoregulation**

Calculation of static ARI has been performed as changes of estimated cerebrovascular resistance (eCVR) in relation to the changes in BP: eCVR = BP/Vmax and ARI = %ΔeCVR/%ΔBP with %ΔeCVR = (eCVR2 - eCVR1)/eCVR1 and %ΔBP = (BP2-BP1)/BP1 [11]. Alternatively, static ARI can be calculated as follows: ARI = (initial Vmax/initial Vmin - initial BP/final BP)/ (1 - initial BP/final BP) [12]. Our calculation of ARI during two series of standing and sitting (or supine) was based on maximum and minimum values: %ΔeCVR = (eCVR maximum - eCVR minimum)/eCVR minimum and %ΔBP = (BP maximum-BP minimum)/BP minimum. Additionally, individual ARIs during standing was based on values from standing to sitting (or supine): %ΔeCVR = (eCVR standing- eCVR sitting or supine)/eCVR sitting or supine and %ΔBP = (BP standing-BP sitting or supine)/BP sitting or supine.

Normal static ARI values in children were reported as 0.95±0.05 [3] and 0.96±0.09 [5] in the middle cerebral artery (MCA), and 0.94±0.10 [3] and 0.94±0.12 [5] in the BA. Static ARI in normal orthopedic adult patients during sevoflurane anesthesia [4] were much lower, 0.66±0.2 in the MCA and 0.72±0.2 in the BA, than that found in children. It has been considered that static ARI is a dimensionless value ranging between 0-1 [4]. Dynamic ARI is ranged from 0-9 and 5±1 as normal [11].

However, our calculated ARI were variable from -12.3 to 16.20 during position changes based on the previous two equations (Table 1 and Figure 1) [11, 12]. A demonstrated patient with SCD (Figure 1) showed that MBP decrease (68 mmHg to 64 mmHg), Vmax decrease (22.5 cm/s to 12.3 cm/s), and calculated CVR increase (MVP/Vmax) (3.02 to 5.20) resulted in -12.3 ARI (ΔeCVR/ΔBP) during her first standing. During her second standing, MBP decrease (79 mmHg to 63 mmHg), Vmax decrease (24.1 cm/s to 14.8 cm/s), and calculated CVR increase (MVP/Vmax) (3.28 to 4.26) resulted in -1.47 ARI (ΔeCVR/ΔBP).

Our data sampling occurred every 1 minute during all series. The previous study utilizing change in position (from supine to sitting) took five minute intervals between position changes before data collection [5]. Other static AR studies utilizing phenylephrine infusion probably required a much longer period of data sampling, in the period between before and after BP increase [4, 11]. In contrast, dynamic AR studies utilizing the thigh cuff method evaluated the data every second, resulting in higher ARI [2, 11, 13] in comparison with static ARI (Table 2). Dynamic AR utilizing spontaneous transient pressor and depressor changes [14] in normal control subjects was much higher, as high as 6.3±1.1 [15]. Data sampling time is probably affecting the ARI results.
Vertebrobasilar autoregulation during postural changes

Our data of first and second standing were also variable, there was no definite tendency between series. This variability should be clarified in following analysis.

Orthostatic hypotension (OH) and autoregulation in the VBA

Our patients of SCD and LI/DM complained of severe dizziness resulting in an inability to remain standing for three minutes. Both patients during the series showed DBP ≥ 10, and there was a SPB ≥ 20 in the SCD patient. The SCD patient showed decreased BP and Vmax during standing, fulfilling the criteria of OH on the basis of the recommendations of active standing [16]. The SCD patient showed ΔPI and ΔeCVR increases and probably disturbed AR in the BA. Older subjects displayed greater vulnerability to reduced perfusion in the posterior cerebral artery (PCA) during orthostatic stress [17]. The head-up tilt table tests indicated that the static AR in the PCA tended to be worse than in the MCA [18]. Further studies in the VBA AR are recommended for those patients with autonomic dysregulation.

In conclusion, continuous TCDS monitoring in the VBA during postural changes is capable of evaluating pathophysiology of vertebrobasilar hemodynamics and autoregulation associated with autonomic regulation.

Abbreviations
AR: Autoregulation; ARI: Autoregulation index; BA: Basilar artery; BP: Blood pressure; DM: Diabetes mellitus; eCVR: Estimated cerebrovascular resistance; DBP: Diastolic blood pressure; LI: Lacunar infarction; MBP: Mean blood pressure; MCA: Middle cerebral artery; OH: Orthostatic hypotension; PCA: Posterior cerebral artery; PI: Pulsatility index; SBP: Systolic blood pressure; SCD: Spino-cerebellar degeneration; TCD: Transcranial Doppler sonography; TCDS: Transcranial color duplex sonography; VA: Vertebral artery; VBA: Vertebrobasilar artery

Competing interests
The authors declare no conflict of interest.

References

Table 2. Normal autoregulation index (ARI) in the basilar and middle cerebral arteries (BA and MCA).

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year published</th>
<th>Age (range)</th>
<th>n</th>
<th>Dynamic or Static AR</th>
<th>Methods</th>
<th>BA</th>
<th>MCA*</th>
</tr>
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<tr>
<td>Tiecks et al.</td>
<td>1995</td>
<td>35±10 (27-54)</td>
<td>10</td>
<td>Dynamic</td>
<td>Propofol/thigh cuff deflation</td>
<td>4.9±1L</td>
<td>4.6±0.9R</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Static</td>
<td>Propofol/prenylinephrine infusion</td>
<td>–</td>
<td>0.87±0.2L</td>
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<td>Dynamic</td>
<td>Isoflurane/thigh cuff deflation</td>
<td>–</td>
<td>2.2±1.1L</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Static</td>
<td>Isoflurane/prenylinephrine infusion</td>
<td>–</td>
<td>0.33±0.26L</td>
</tr>
<tr>
<td>White and Markus</td>
<td>1997</td>
<td>69±7 (51-81)</td>
<td>69</td>
<td>Dynamic</td>
<td>Spontaneous pressor and depressor changes</td>
<td>–</td>
<td>6.3±1.1</td>
</tr>
<tr>
<td>Vavilala et al.</td>
<td>2002</td>
<td>25-45</td>
<td>9</td>
<td>Dynamic</td>
<td>Thigh cuff deflation</td>
<td>–</td>
<td>5.3±0.8</td>
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<tr>
<td></td>
<td></td>
<td>12-17</td>
<td>8</td>
<td></td>
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<td>Spontaneous depressor changes</td>
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<td>2003</td>
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<td>4.77±1.23</td>
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<td>Vavilala et al.</td>
<td>2005</td>
<td>Boys:12.9±1.7 (10-16)</td>
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<td>Change in position (from supine to sitting)</td>
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<td>Girls:12±1.4 (10-16)</td>
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<td>0.97±0.06</td>
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<td>0.66±0.2</td>
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<tr>
<td>Tontisirin et al.</td>
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<td>6±2 (4-8)</td>
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<td>Static</td>
<td>Change in position (from supine to sitting)</td>
<td>0.94±0.12</td>
<td>0.96±0.09</td>
</tr>
</tbody>
</table>

* L = left; R = right

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