



ORIGINAL RESEARCH

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

Toshiyuki Shiogai¹, Mayumi Yamamoto¹, Yuka Arima¹, Daichi Yamasaka², Kenji Yoshikawa³, Toshiki Mizuno⁴, and Masanori Nakagawa⁴

Special Issue on Neurosonology and Cerebral Hemodynamics

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) = $\% \Delta eCVR / \% \Delta MBP$, 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) $\Delta DBP > 10 \text{ mmHg}$ in all cases. 2) $\Delta SBP > 20 \text{ mmHg}$ in 2 controls and all but one patient (LI/DM). c) TCDS: 1) ΔPI and $\Delta eCVR$ 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.

¹Department of Clinical Neurosciences, Kyoto Takeda Hospital, Kyoto, Japan

²Department of Radiology, Kyoto Takeda Hospital, Kyoto, Japan

³Department of Stroke Medicine, Hoshigaoka Kouseinenkin Hospital, Osaka, Japan

⁴Department of Neurology, Kyoto Prefectural University of Medicine, Kyoto, Japan

Citation: Shiogai et al. Continuous monitoring of vertebrobasilar hemodynamics utilizing TCDS transducer holder Sonopod during postural changes. IJCNMH 2014; 1(Suppl. 1):S21

Received: 08 Sep 2013; Accepted: 29 Nov 2013; Published: 09 May 2014

Correspondence: Toshiyuki Shiogai
Departments of Clinical Neurosciences, Kyoto Takeda Hospital
Minamikinuta-cho 11, Nishinanajo, Shimogyo-ku, Kyoto 600-8884, Japan
Email address: shiogait@pop11.odn.ne.jp



Open Access Publication Available at <http://ijcnmh.arc-publishing.org>

© 2014 Shiogai et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.



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 (TCDS) [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 moni-

tored continuously the intracranial vertebral artery (VA) and basilar artery (BA). Blood pressure (BP), heart and respiration 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) = $\% \Delta eCVR / \% \Delta MBP$ were calculated on the basis of maximum and minimum values during both series: $\% \Delta eCVR = (eCVR \text{ maximum} - eCVR \text{ minimum}) / eCVR \text{ minimum}$ and $\% \Delta BP = (BP \text{ maximum} - BP \text{ minimum}) / BP \text{ minimum}$.

Also individual AR1st during two series of standing (= $\% \Delta eCVR / \% \Delta MBP$) were based on separate values from sitting (or supine) to standing: $\% \Delta eCVR = (eCVR \text{ standing} - eCVR \text{ sitting or supine}) / eCVR \text{ sitting or supine}$ and $\% \Delta BP = (BP \text{ standing} - BP \text{ sitting or supine}) / BP \text{ sitting or supine}$.

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 $\Delta eCVR$ 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.

Case	Age/ Sex	Diagnosis	Artery	ΔSBP (mmHg)	ΔMBP (mmHg)	ΔDBP (mmHg)	ΔV_{max} (cm/s)	ΔPI	$\Delta eCVR$	$\% \Delta eCVR$	$\% \Delta MBP$	ARI	AR1st	AR1st2
DY	23M	Normal	BA	19	21	10	5.3	0.33	1.00	0.56	0.33	1.70	1.62	-0.05
MY	24F	Normal	BA	11	10	16	11.2	0.75	0.49	0.37	0.12	3.08	2.21	-9.54
RS	68M	Normal	LVA	11	31	13	8.2	0.61	1.55	0.66	0.39	1.69	2.18	1.94
KI	23M	Normal	BA	34	32	18	22.5	0.50	1.60	0.98	0.39	2.51	3.39	2.50
YN	60M	Normal	BA	20	23	10	16.0	0.40	0.44	0.30	0.25	1.20	-9.05	1.47
MN	65F	HT	BA	22	24	23	6.2	0.40	0.77	0.37	0.30	1.23	0.66	2.17
TS	60M	HT	RVA	25	18	17	6.1	0.26	1.03	0.31	0.20	1.55	5.46	16.20
MY	67F	SCD	BA	20	16	14	11.8	1.16	2.18	0.72	0.25	2.88	-12.30	-1.47
MK	75M	LI/DM	RVA	15	9	13	1.4	1.22	1.73	0.24	0.16	1.50	0.09	ND
KN	63M	dizziness/ LC	BA	40	25	13	8.0	0.39	1.15	0.73	0.32	2.28	0.24	0.63
SM	66M	dizziness	BA	21	14	10	5.0	0.30	0.74	0.28	0.16	1.75	2.62	0.81
HI	36M	dizziness/ PN	RVA	25	22	14	9.0	0.49	0.34	0.21	0.29	0.72	0.60	4.95

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; $\% \Delta eCVR = (eCVR \text{ maximum} - eCVR \text{ minimum}) / eCVR \text{ minimum}$; $\% \Delta MBP = (MBP \text{ maximum} - MBP \text{ minimum}) / MBP \text{ minimum}$; ARI = Autoregulation Index ($\% \Delta eCVR / \% \Delta MBP$); AR1st = ARI during standing (Vmax sitting or supine/Vmax standing-BP sitting or supine)/BP sitting/(1- BP sitting or supine/BP standing); ND = No data

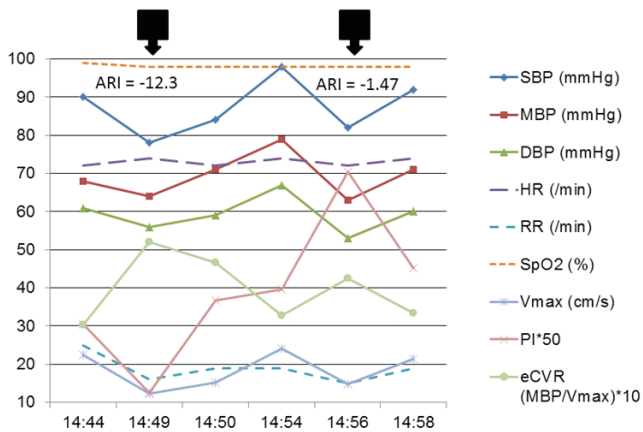


Figure 1. Parameters demonstrated during two series of standing (indicated by arrows) in the MY patient with spinocerebellar degeneration. The patient complained of dizziness during standing and could not remain standing. Falling BP and Vmax and increasing PI and eCVR during standing were distinctive. Calculated ARIst during individual standing was -12.3 and -1.47, respectively. However, calculated ARI on the basis of maximum and minimum values during all series was 2.88.

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, ARIst 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 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: $eCVR = BP/V_{max}$ and $ARI = \% \Delta eCVR / \% \Delta BP$ with $\% \Delta eCVR = (eCVR_2 - eCVR_1) / eCVR_1$ and $\% \Delta BP = (BP_2 - BP_1) / BP_1$ [11]. Alternatively, static ARI can be calculated as follows: $ARI = (initial V_{max} / final V_{max} - initial BP / final BP) / (1 - initial BP / final BP)$ [12]. Our calculation of ARI during two series of standing and sitting (or supine) was based on maximum and minimum values: $\% \Delta eCVR = (eCVR_{maximum} - eCVR_{minimum}) / eCVR_{minimum}$ and $\% \Delta BP = (BP_{maximum} - BP_{minimum}) / BP_{minimum}$. Additionally, individual ARIst during standing was based on values from standing to sitting (or supine): $\% \Delta eCVR = (eCVR_{standing} - eCVR_{sitting \text{ or } supine}) / eCVR_{sitting \text{ or } supine}$ and $\% \Delta BP = (BP_{standing} - BP_{sitting \text{ or } supine}) / BP_{sitting \text{ or } supine}$.

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 ($\Delta eCVR / \Delta BP$) 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 ($\Delta eCVR / \Delta BP$).

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.

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

Authors	Year published	Age (range)	n	Dynamic or Static AR	Methods	ARI	
						BA	MCA*
Tiecks et al.	1995	35±10 (27-54)	10	Dynamic	Propofol /thigh cuff deflation	–	4.9±1L 4.6±0.9R
				Static	Propofol/ pnylephrine infusion	–	0.87±0.2L 0.82±0.1R
				Dynamic	Isoflurane/ thigh cuff deflation	–	2.2±1.1L 2.1±1.1R
				Static	Isoflurane/ pnylephrine infusion	–	0.33±0.26L 0.31±0.27R
White and Markus	1997	69±7 (51-81)	69	Dynamic	Spontaneous pressor and depressor changes	–	6.3±1.1
Vavilala et al.	2002	25-45 12-17	9 8	Dynamic	Thigh cuff deflation	–	5.3±0.8 3.9±2.1
Eames et al.	2002	69±7 (51-81)	48	Dynamic	Spontaneous pressor changes	–	4.5±2
				Dynamic	Spontaneous depressor changes	–	4.7±2.2
Park et al.	2003	27.4±8.5 (19-46)	15	Dynamic	Thigh cuff deflation	4.62±1.26	4.77±1.23
Vavilala et al.	2005	Boys:12.9±1.7 (10-16) Girls:12±1.4 (10-16)	13 13	Static	Change in position (from supine to sitting)	0.92±0.12	0.98±0.03
						0.97±0.06	0.92±0.1
Rozet et al.	2006	30±9 (22-47)	9	Static	Sevoflurane/ pnylephrine infusion	0.72±0.2	0.66±0.2
Tontisirin et al.	2007	6±2 (4-8)	48	Static	Change in position (from supine to sitting)	0.94±0.12	0.96±0.09

* L = left; R = right

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 \geq 10, and there was a SPB \geq 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 Δ PI and Δ eCVR 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

- Naschitz JE, Rosner I. Orthostatic hypotension: framework of the syndrome. *Postgrad Med J* 2007; 83(983):568-574.
- Park CW, Sturzenegger M, Douville CM, Aaslid R, Newell DW. Autoregulatory response and CO₂ reactivity of the basilar artery. *Stroke* 2003; 34:34-39.
- Vavilala MS, Kincaid MS, Muangman SL, Suz P, Rozet I, Lam AM. Gender differences in cerebral blood flow velocity and autoregulation between the anterior and posterior circulations in healthy children. *Pediatr Res* 2005; 58:574-578.
- Rozet I, Vavilala MS, Lindley AM, Visco E, Treggiari M, Lam AM. Cerebral autoregulation and CO₂ reactivity in anterior and posterior cerebral circulation during sevoflurane anesthesia. *Anesth Analg* 2006; 102:560-564.

5. Tontisirin N, Muangman SL, Suz P, Pihoker C, Fisk D, Moore A, Lam AM, Vavilala MS. Early childhood gender differences in anterior and posterior cerebral blood flow velocity and autoregulation. *Pediatrics* 2007; 119:e610-615.
6. Hong JM, Joo IS, Huh K, Sheen SS. Simultaneous vasomotor reactivity testing in the middle cerebral and basilar artery with suboccipital probe fixation device. *J Neuroimaging* 2010; 20:83-86.
7. Reinhard M, Schork J, Allignol A, Weiller C, Kaube H. Cerebellar and cerebral autoregulation in migraine. *Stroke* 2012; 43:987-993.
8. Yamaoka Y, Ichikawa Y, Kimura T, Sameshima T, Ochiai C, Morita A. A Novel Method for Transcranial Doppler Microembolic Signal Monitoring at the Vertebrobasilar Junction in Vertebral Artery Dissection Patients. *J Neuroimaging* 2012; 20:1-4.
9. Shiogai T, Koyama M, Yamamoto M, Yoshikawa K, Mizuno T, Nakagawa M: Brain tissue perfusion monitoring using Sonopod for transcranial color duplex sonography. *Perspectives in Medicine* 2012; 1: 34–38.
10. Shiogai T, Koyama M, Yamamoto M, Hashimoto H, Yoshikawa K, Nakagawa M: Monitoring of brain tissue perfusion utilizing a transducer holder (Sonopod) for transcranial color duplex sonography. *Acta Neurochir (Suppl)* 2013; 118: 229-233.
11. Tiecks FP, Lam AM, Aaslid R, Newell DW. Comparison of static and dynamic cerebral autoregulation measurements. *Stroke* 1995; 26:1014-1019.
12. Strebel S, Lam AM, Matta B, Mayberg TS, Aaslid R, Newell DW. Dynamic and static cerebral autoregulation during isoflurane, desflurane, and propofol anesthesia. *Anesthesiology* 1995; 83:66-76.
13. Vavilala MS, Newell DW, Junger E, Douville CM, Aaslid R, Rivara FP, Lam AM. Dynamic cerebral autoregulation in healthy adolescents. *Acta Anaesthesiol Scand* 2002; 46:393-397.
14. Eames PJ, Blake MJ, Dawson SL, Panerai RB, Potter JF. Dynamic cerebral autoregulation and beat to beat blood pressure control are impaired in acute ischaemic stroke. *J Neurol Neurosurg Psychiatry* 2002; 72:467-472.
15. White RP, Markus HS. Impaired dynamic cerebral autoregulation in carotid artery stenosis. *Stroke* 1997; 28:1340-1344.
16. Task Force for the Diagnosis and Management of Syncope; European Society of Cardiology (ESC); European Heart Rhythm Association (EHRA); Heart Failure Association (HFA); Heart Rhythm Society (HRS); Moya A, Sutton R, Ammirati F, Blanc JJ, Brignole M, Dahm JB, Deharo JC, Gajek J, Gjesdal K, Krahn A, Massin M, Pepi M, Pezawas T, Ruiz Granell R, Sarasin F, Ungar A, van Dijk JG, Walma EP, Wieling W. Guidelines for the diagnosis and management of syncope (version 2009). *Eur Heart J* 2009; 30:2631-2671.
17. Sorond FA, Khavari R, Serrador JM, Lipsitz LA. Regional cerebral autoregulation during orthostatic stress: age-related differences. *J Gerontol A Biol Sci Med Sci* 2005; 60: 1484-1487.
18. Wang YJ, Chao AC, Chung CP, Huang YJ, Hu HH. Different cerebral hemodynamic responses between sexes and various vessels in orthostatic stress tests. *J Ultrasound Med* 2010; 29: 1299-1304.