JRRD Volume 41, Number 1, Pages 89–94
January/February 2004
Journal of Rehabilitation Research & Development

Common carotid and common femoral arterial dynamics during head-up tilt in persons with spinal cord injury

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Abstract—We examined the effect of the level and completeness of spinal cord injury (SCI), tetraplegia and paraplegia, on common carotid arterial (CCA) and common femoral arterial (CFA) functions supine and during head-up tilt (HUT), compared with able-bodied controls. Subjects (tetraplegia [n = 7], paraplegia [n = 8], and controls [n = 8]) were healthy males between the ages of 19 and 60 years. We used Doppler ultrasound to determine vessel diastolic diameters and flow velocities while supine and at 45° HUT. The results indicated that supine CCA diameter and flow were augmented in the tetraplegia group compared with the paraplegia group ($p < 0.05$); no other group differences were noted. However, CCA_{flow} was significantly reduced from supine to 45° HUT in the tetraplegia group ($p < 0.01$). CFA diameter and flow were significantly reduced in the SCI groups compared with the control group, and CFA_{flow} was reduced from supine to 45° HUT in the tetraplegia group. These results demonstrate that individuals with tetraplegia have increased resting CCA diameters and flows compared with individuals with paraplegia, an adaptation which may contribute to orthostatic tolerance. The significant reduction in CFA_{flow} from supine to 45° HUT in the tetraplegia group may be related to the completeness of lesion rather than the level of lesion.

Key words: common carotid arterial blood flow, common femoral arterial blood flow, orthostasis, spinal cord injury.

INTRODUCTION

During orthostatic provocations, such as head-up tilt (HUT), the carotid baroreceptors immediately sense a fall in systemic pressure and initiate a cascade of events that include autonomic [1–2], hormonal [3–5], and mechanical [1–2] mechanisms. These systems function in concert to produce a compensatory peripheral vasoconstriction to increase systemic blood pressure (BP) and maintain blood flow to the brain. In individuals with spinal cord injury (SCI), neural transmission between the central nervous system and the peripheral vasculature is partially or completely interrupted, and this harmonious interplay is compromised. Subsequently, persons with SCI may be prone to symptoms of hypotension during HUT [6–7].

Evidence in the literature suggests reduced common femoral arterial (CFA) diameter in persons with SCI compared

Abbreviations: ANOVA = analysis of variance, ASIA = American Spinal Injury Association, BP = blood pressure, CCA = common carotid artery, CFA = common femoral artery, ECG = electrocardiogram, HR = heart rate, HUT = head-up tilt, NO = nitric oxide, PWV = pulse wave velocity, SCI = spinal cord injury.

This material was based on work supported by the American Paraplegia Society Seed grant 890 and Department of Veterans Affairs, Rehabilitation Research and Development Center of Excellence B2648C, using the facilities at the Bronx VA Medical Center.

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with able-bodied controls [8–11]. Further, a significant reduction in the response of CFA blood flow (CFA_{flow}) with arm exercise has been reported in individuals with paraplegia compared with controls [11]. There is data to suggest that CFA_{flow} is adversely affected by the completeness of injury [12] and by chronic disruption in sympathetic innervation to the vasculature [13].

Unlike the CFA, vessels above the level of lesion, such as the common carotid artery (CCA), are generally unaffected by SCI. Comparable CCA diameters and CCA_{flow} have been reported to be similar in persons with paraplegia compared with able-bodied controls [9–10,13]. Although Boot et al. distinguished between persons with upper (T_4–T_{12}) and lower lesions (T_{10}–T_{12}) [9], we are unaware of any reports addressing CCA diameter or flow in subjects with higher cord lesions.

The primary objectives of this study were to quantify CFA and CCA diameter and flow responses to HUT in subjects with SCI compared with able-bodied controls and to differentiate the vessel responses dependent on level and completeness of SCI. A secondary objective was to examine the relationship between vessel diameter and flow responses to HUT in the CCA and CFA.

METHODS

Subjects

All subjects (n = 23) were healthy males between the ages of 19 and 60 years, who were without known cardiovascular, pulmonary diseases or diabetes mellitus and were current nonsmokers for a minimum of 1 year before the investigation. Subjects with tetraplegia (n = 7) and paraplegia (n = 8) were healthy outpatients, a minimum of 2 years postinjury, and were able to maintain an independent lifestyle. Using the American Spinal Injury Association (ASIA) classification of neurological impairment, eight individuals were diagnosed with a complete injury (ASIA A) and the other seven with an incomplete injury (ASIA B and C). The able-bodied control subjects (n = 8) were age-, height- and weight-matched to the subjects with SCI. The Institutional Review Board for Human Studies of the Bronx Department of Veterans Affairs Medical Center granted approval for the study, and we obtained informed consent before the investigation.

Protocol Procedures

Subjects reported to the laboratory between 10 a.m. and 1 p.m. and were instructed to be well hydrated and to have avoided caffeine and alcohol for a minimum of 24 hours before testing. The study involved a progressive HUT maneuver from supine to 45° HUT. Upon arrival of the subjects to the laboratory, we transferred or instructed the them to lie on the tilt table. While the subject rested quietly, electrocardiogram (ECG) electrodes were applied to the chest for continuous monitoring of heart rate (HR: 742 Mennen Medical ECG Monitor, Bio-Medical Equipment Service Co. Louisville, Kentucky, USA). We continuously monitored beat-to-beat BP at the wrist using a radial tonometer (Colins 7000, Colins Medical Instruments Corp., San Antonio, Texas, USA), which was stabilized at the height of the heart during the HUT maneuver. For calibration purposes, we took the BP every 5 minutes using a standard manual sphygmomanometer and cuff placed around the brachial artery (W.A. Baum Co., Inc., Copiague, New York). Prior to the HUT, supine Doppler ultrasound measurements (Apogee 800 Ultrasound System Bothell, Washington) of the CCA and CFA were made. The HUT maneuver was then initiated, with a graded, slow progression to 45° (i.e., 10-minute stages at 15°, 25°, 35° and then 45°). HR and BP were continuously monitored during the HUT, and CCA and CFA measurements were acquired at approximately 3 and 5 minutes into the stage of tilt.

Vessel Diameter and Flow

Using Doppler ultrasound technology, we recorded arterial diastolic diameter and blood flow velocities for the CCA and CFA. We used a linear array transducer operating at a frequency of 7.5 MHz to assess diastolic diameter in the two-dimensional (2D) mode with a transverse view. We recorded pulse wave velocity (PWV) with a longitudinal view, making the necessary corrections for the angle of insonation (60°) and sample volume length. All data were downloaded to videotapes for storage and analysis. We analyzed Doppler waveforms using commercially available software installed in the Apogee 800 system to determine vessel diastolic diameter and mean velocity during three successive cardiac cycles. Mean blood flow (mL/min) for the CCA and CFA at supine and 45° of HUT is reported as the product of mean velocity and vessel area * 60 seconds:

$$\text{mean blood flow} = \text{Vel}_{\text{mean}} \times [\pi(\text{diameter} \div 2)^2] * 60 \ .$$

Data Analysis

All continuous variables are reported as mean plus or minus standard deviation (mean ± SD). We used analysis of variance (ANOVA), with a Fischer post hoc, to determine significant group differences for the demographic parameters and for supine and 45° HUT hemodynamic measures. A one-sample-paired analysis was used to determine if changes in vessel diameter and flow from supine to 45° HUT were significantly different from zero and used an ANOVA to determine if these changes were statistically different among the groups. Linear regression analysis was applied to determine the relationship between the change in vessel diameter and flow for the CFA and CCA. Significance was set at the 0.05 alpha level.

RESULTS

No significant differences were found among the groups for age, height, or weight (**Table 1**); however, the tetraplegia group had significantly fewer complete injuries ($p < 0.0001$) and tended to have a longer duration of injury ($p = 0.15$) than the paraplegia group.

Common Carotid Artery

CCA diameter did not differ between the SCI groups and the able-bodied group; however, supine CCA diameter was significantly increased in the tetraplegia compared with the paraplegia group ($p < 0.01$) (**Table 2**). Supine CCA_{flow} did not differ between the SCI groups and the able-bodied group; however, supine CCA_{flow} was significantly increased in the tetraplegia compared with the paraplegia group ($p < 0.05$). No significant effects of completeness of lesion were found on CCA diameter or flow in subjects with SCI.

At 45° HUT, no significant group differences were found for CCA diameter or CCA_{flow} (**Table 2**). CCA diameter was significantly reduced in the tetraplegia group from supine to 45° HUT (–0.84 ± 0.66 mm, $p < 0.01$) and this reduction was significantly larger than in the paraplegia and able-bodied groups ($p < 0.05$). CCA_{flow} was significantly reduced from supine to 45° HUT in the tetraplegia group (–185 ± 122 mL/min, $p < 0.001$), and this reduction was significantly greater than in the able-bodied (–0.2 ± 123, $p < 0.002$) and paraplegia (–11 ± 107, $p < 0.003$) groups. The relationship between the change in vessel diameter and in flow for the CCA correlated moderately in all three groups ($R = 0.54$, $p < 0.01$) (**Figure (a)**).

Common Femoral Artery

CFA diameter was significantly reduced in both SCI groups compared with the able-bodied group while supine and at 45° HUT ($p < 0.01$) (**Table 2**). CFA_{flow} was significantly reduced in the paraplegia compared with the able bodied group while supine ($p < 0.01$) and at 45° HUT ($p < 0.05$). In persons with incomplete compared to complete SCI, CFA diameter (7.9 ± 1.1 versus 7.0 ± 0.8, $p < 0.002$, respectively) and flow (282 ± 147 versus 202 ± 75, $p < 0.05$, respectively) were significantly increased. No significant differences were found in CFA_{flow} supine or at 45° HUT between the tetraplegia and the able-bodied groups.

CFA diameter was significantly increased from supine to 45° HUT in the paraplegia group (0.56 ± 0.75 mm, $p < 0.05$), and this increase was significantly greater than in the tetraplegia group ($p < 0.05$). CFA_{flow} was significantly reduced in the tetraplegia group (–77 ± 126 mL/min, $p < 0.05$) from supine to 45° HUT; however, this reduction was not statistically different from the other groups. The relationship between the change in vessel diameter and flow for the CFA was highly significantly in the groups combined ($R = 0.8$, $p < 0.0001$) (**Figure (b)**). Further, this relationship was significant in persons with incomplete lesions ($r = 0.72$, $p < 0.01$) and not complete lesions ($r = 0.5$, nonsignificant [ns]).

Table 1.
Characteristics of subjects.

Variable	Tetraplegia (n = 7)	Paraplegia (n = 8)	Able-Bodied (n = 8)
Age (yr)	41 ± 12	34 ± 8	32 ± 9
Height (cm)	172 ± 11	175 ± 6	176 ± 11
Weight (kg)	70 ± 9	80 ± 15	77 ± 14
BMI	23.6 ± 3.3	26.1 ± 5.2	24.6 ± 2.3
DOI (yr)	17 ± 13	9 ± 5	—
Complete Injury (%)	29*	75	—

Note: Data presented as mean ± SD.
BMI = body mass index
DOI = duration of injury
*$p < 0.0001$ versus paraplegia group

Table 2.
CCA and CFA dynamics.

Group	Diameter (mm)			Flow (mL/min)		
	BL	45	% Change	BL	45	% Change
Common Carotid						
Tetraplegia	8.1 ± 0.7*	7.4 ± 0.4	[10] ± 8†‡	537 ± 162†	369 ± 133	[33] ± 19†‡
Paraplegia	7.3 ± 0.6	7.1 ± 0.9	[2] ± 11	406 ± 98	359 ± 119	[1] ± 29
Able-Bodied	7.6 ± 1.1	7.3 ± 0.9	[3] ± 9	455 ± 187	455 ± 212	0.5 ± 27
Common Femoral						
Tetraplegia	7.8 ± 1.1§	7.7 ± 1.0‡	[1] ± 8†	305 ± 166	242 ± 113	[18] ± 34
Paraplegia	7.0 ± 0.6§	7.6 ± 1.2‡	8 ± 10	216 ± 95§	191 ± 55‡	9 ± 42
Able-Bodied	9.5 ± 1.8	9.8 ± 2.1	4 ± 14	414 ± 203	305 ± 162	[17] ± 53

Note: Data presented as mean ± SD.
*$p < 0.01$ versus paraplegia
†$p < 0.05$ versus paraplegia
‡$p < 0.05$ versus able-bodied
§$p < 0.01$ versus able-bodied
[ ] = negative number
BL = baseline data

DISCUSSION

We found that CCA diastolic diameter and flow were not significantly different in persons with SCI, compared with able-bodied individuals, as previously reported [9,10,13]. Arterial dynamics above the level of spinal lesion would not be expected to be reduced compared with able-bodied subjects, since neural control is intact. In fact, in paraplegic athletes, Huonker et al. reported significantly increased subclavian diameters compared with sedentary individuals with paraplegia and sedentary controls [14]. Interestingly, we found that supine CCA diameter and flow were significantly increased in the tetraplegia compared with the paraplegia group. However, no significant effects of completeness of SCI were found on CCA diameters and flow. Increased supine diastolic diameter may relate to greater wall shear stress from increased flow [9], but it may also result from higher elasticity caused by reduced resting HR or an increased basal nitric oxide (NO) production [15]. An up-regulation in NO syntheses (NOS) has been reported in an animal model of immobilization and may play a role in chronic dilatation of the vasculature reported in persons with tetraplegia [16]. However, future investigation is necessary to determine the associations among CCA diameter, NOS up-regulation, and cardiovascular integrity in persons with chronic tetraplegia.

At 45° HUT, CCA diameter or flow did not differ among the groups. In the tetraplegia group however, the percent change from supine was significantly different from zero and was greater than the other two groups. The dynamic function of the CCA during HUT has not been previously reported in subjects with SCI. Several reports have examined midcerebral blood flow in persons with tetraplegia during upright postures and have suggested that it is maintained independent of systemic pressure and peripheral sympathetic control [7,17,18]. This may not be the case for the CCA. Based on the results reported herein, CCA_{flow} at 45° HUT was "normalized" in subjects with tetraplegia compared with the other groups and perhaps augmented baseline CCA_{flow} is a precautionary adaptation to the loss of peripheral sympathetic vasomotor tone in this group. During the HUT maneuver, blood flow to the CCA should be maintained regardless of alterations in systemic hemodynamic. The relationship between CCA_{flow} and diastolic diameter was only moderately significant in the three groups (**Figure (a)**) and was driven by the large fluctuations in diameter and flow in the tetraplegia group alone. Whereas, no association was found between CCA diameter and flow in the able-bodied and paraplegia groups combined.

CFA diameter and flow were significantly reduced in the SCI groups compared with the able-bodied group, as previously reported [8–11,14]. It has been suggested that differences in CFA diameter and flow between individuals with SCI and able-bodied controls may reflect skeletal muscle atrophy and consequential reductions in muscle blood flow demands. In accordance, Olive et al. reported

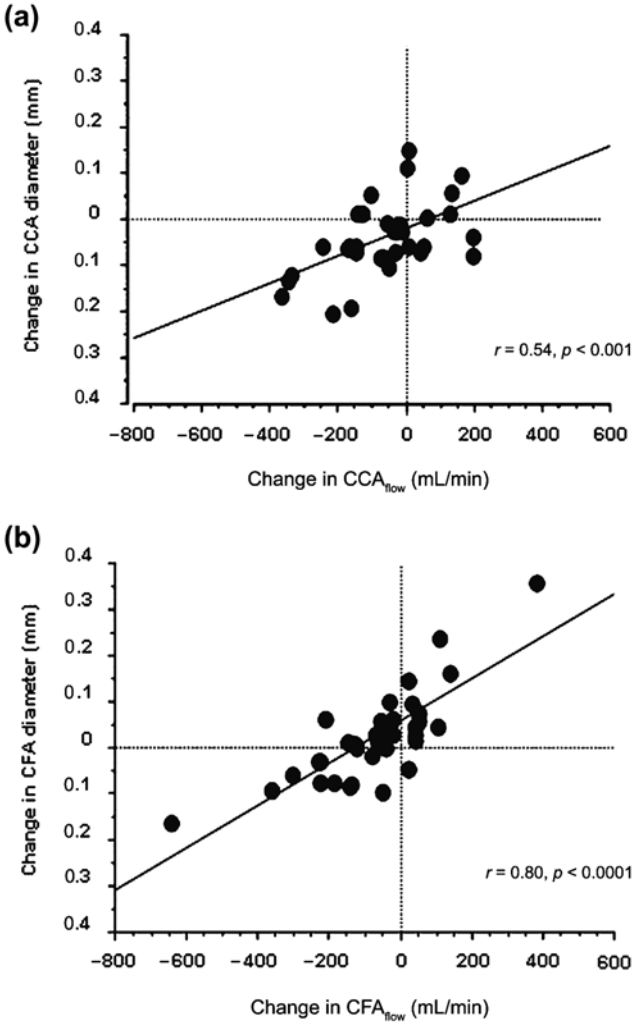

Figure.
Relationship between change in diameter and flow from supine to 45° HUT in **(a)** CCA and **(b)** CFA.

the absence of group differences between SCI and able-bodied controls for CFA diameter when normalized for thigh volume [8]. Limb volume was not measured herein, but is appreciated to be reduced following SCI and may be partially responsible for the reduced CFA dynamics reported herein. However, group differences in CFA dynamics between the SCI and able-bodied controls remained significant when corrected for body surface area (BSA). It has been suggested that completeness of lesion rather than level of lesion may play a more prominent role in CFA dynamics [9–11]. This study has demonstrated significantly increased CFA diameter and flow in subjects with incomplete compared with complete SCI. Whereas, no significant differences were found in CFA diameter and flow between individuals with tetraplegia compared with paraplegia. Future investigation is warranted to determine the contribution of level and completeness of lesion to lower-limb blood flow and arterial function.

The relationship between changes in diastolic diameter to changes in flow was highly significant in the CFA, with much less variation from the line of identity (**Figure (b)**). This relationship was most significant in the able-bodied group and was only moderately significant in the SCI groups combined. Further, the relationship between diameter and flow for the CFA was significant in persons with incomplete lesions and not significant in persons with complete lesions.

CONCLUSION

The results of this investigation demonstrate that persons with SCI, regardless of level of lesion, have relatively normal common carotid hemodynamics similar to that reported in able-bodied controls. Although, individuals with tetraplegia have augmented resting CCA diameters and flows compared with individuals with paraplegia, an adaptation that may contribute to orthostatic tolerance. As previously reported, CFA diameters and flows are significantly reduced in persons with SCI compared with able-bodied controls and are most pronounced in those with complete versus incomplete injury.

REFERENCES

1. Borst C, van Brederode JFM, Wieling W, van Montfrans GA, Dunning AJ. Mechanisms of initial blood pressure response to postural change. Clin Sci 1984;67:321–27.
2. Borst C, Wieling W, van Brederode JFM, Hond A, de Rijk LG, Dunning AJ. Mechanisms of initial heart rate response to postural change. Am J Physiol Heart Circ Physiol 1982; 243:H676–81.
3. Guttmann L, Munro AF, Robinson R, Walsh JJ. Effect of tilting on the cardiovascular responses and plasma catecholamine levels in spinal man. Paraplegia 1960;104:4–18.
4. Mathias CJ, Christensen NJ, Corbett JL, Frankel HL, Goodwin TJ, Peart WS. Plasma catecholamine, plasma renin activity and plasma aldosterone in tetraplegia man horizontal and tilted. Clin Sci Mol Med 1975;49:291–99.
5. Mathias CJ, Christensen NJ, Frankel HL, Peart WS. Renin release during head-up tilt occurs independently of

sympathetic nervous activity in tetraplegia man. Clin Sci 1980; 59:251–56.
6. Blackmer J. Orthostatic hypotension in spinal cord injured patients. J Spinal Cord Med 1997;20:212–17.
7. Gonzalez F, Chang JY, Banovac K, Messina D, Martinez-Arizala A, Kelly RE. Autoregulation of cerebral blood flow in patients with orthostatic hypotension after spinal cord injury. Paraplegia 1991;29:1–7.
8. Olive JL, Dudley GA, McCully KK. Vascular remodeling after spinal cord injury. Med Sci Sports Exerc 2003;35(6): 901–7.
9. Boot CRL, Groothuis JT, VanLangen H, Hopman MTE. Shear stress levels in paralyzed legs of spinal cord injured individuals with and without nerve damage. J Appl Physiol 2002;92:2335–40.
10. Boot CRL, van Langen H, Hopman MTE. Arterial vascular properties in individuals with spina bifida. Spinal Cord 2003;41:242–46.
11. Hopman MTE, Nommensen H, Van Asten WNJ, Oeseburg B, Binkhorst RA. Changes in blood flow in the common femoral artery related to inactivity and muscle atrophy in individuals with long-standing paraplegia. Adv Exp Biol 1996;388:379–83.
12. Olive JL, McCully KK, Dudley GA. Blood flow responses in individuals with incomplete spinal cord injuries. Spinal Cord 2002;40:639–45.
13. Schmidt-Truckass A, Schmid A, Brunner C, Scherer N, Zach G, Keul J, Huonker M. Arterial properties of the carotid and femoral artery in endurance-trained and paraplegic subjects. J Appl Physiol 2000;89:1956–63.
14. Huonker M, Schmid A, Sorichter S, Schmidt-Trucksab A, Mrosek P, Keul J. Cardiovascular differences between sedentary and wheelchair-trained subjects with paraplegia. Med Sci Sports Exerc 1998;30(4):609–13.
15. Sa Cunha R, Pannire B, Benetos A, Siche JP, London GM, Mallion JM, Safar ME. Association between high heart rates and high arterial rigidity in normotensive and hypertensive subjects. J Hypertens 1997;15(12 Pt 1):1423–30.
16. Vaziri ND, Ding Y, Sangha DS, Purdy RE. Upregulation of NOS by simulated microgravity, potential cause of orthostatic intolerance. J Appl Physiol 2000;89:338–44.
17. Nanda RN, Wyper DJ, Harper AM, Johnson RH. Cerebral blood flow in paraplegia. Paraplegia 1974;12:212–18.
18. Houtman S, Colier WNJM, Oeseburg B, Hopman MTE. Systemic circulation and cerebral oxygenation during head-up tilt in spinal cord injured individuals. Spinal Cord 2000;38:158–63.

Submitted for publication July 22, 2003. Accepted in revised form November 12, 2003.