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Dynamic elongation of the vertebral artery during an in vitro whiplash simulation

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Abstract Clinical signs of whiplash are presently not well understood. Vertebral artery (VA) stretch during trauma is a possible pathomechanism that could explain some aspects of the whiplash symptom complex. This study quantified the VA elongation during whiplash simulation using an in vitro model. Seven fresh human cadaveric specimens (occiput to C7 or T1) were carefully dissected, preserving the osteoligamentous structures. The right VA was replaced with a thin nylon-coated flexible cable. This cable was fixed at one end to the occipital bone and at the other end to a specially designed VA transducer. Physiological motion of the occiput and physiological elongation of the VA were measured with a standard flexibility test. Next the specimen was mounted on a specially designed sled and subjected to 2.5, 4.5,

6.5, and 8.5 g (1 g = 9.81 m/s²) horizontal accelerations. Elongation of the VA was continuously recorded from the start of the trauma. The average (standard deviation) physiological VA elongation was 5.8 (1.6) mm in left lateral bending and 4.7 (1.8) mm in left axial rotation. Flexion and extension did not result in any appreciable elongation of the VA. The maximum VA elongation during the whiplash trauma significantly correlated with the horizontal acceleration of the sled ($R^2 = 0.7$, $P < 0.05$). The VA exceeded its physiological range by 1.0 (2.1), 3.1 (2.6), 8.9 (1.6), and 9.0 (5.9) mm in the 2.5-, 4.5-, 6.5-, and 8.5-g trauma classes respectively.

Key words Vertebral artery · Whiplash trauma · Spine · Biomechanics

Introduction

Clinical symptoms identifying a whiplash injury complex are presently not well understood. Vascular deficiency, especially vertebral artery (VA) lesions initiated by its stretch, have been suggested as a partial explanation of the complicated symptoms related to whiplash trauma [9]. Various mechanisms can be hypothesized. The elongation of the VA decreases its diameter due to Poisson's effect, and may decrease the blood flow [5]. The stretch of the artery beyond its physiological range may damage the endothelium and may induce embolism and pseudoaneurism [3, 8]. Some cases of stroke and vertebrobasilar injuries

have been reported following cervical manipulation [1, 10, 14]. Evidently, these accidents may occur in the absence of clinically apparent vascular or cervical spine disease. The resulting symptoms may include nausea, visual disturbance, vomiting, and vertigo, which are often reported by whiplash trauma patients.

More recently, Thiel et al. reported no VA encroachment or blood flow restriction to the head in response to voluntary cervical motion [15]. However, no research has attempted to determine VA stretch in relation to head and cervical spine motion during trauma. The purpose of the present study was to develop a VA transducer and to measure VA stretch under physiological loads and during simulated whiplash trauma using a cadaveric model.

Fig. 1 **A** Lateral view of the vertebral artery transducer (VAT) mounted in the cervical spine specimen. **B** Sketch of the VAT, showing top and lateral views. (A outer bracket, B base plate, C carriage, D Hall effect sensor, E rare earth magnets, F steel cable)

Materials and methods

Specimen preparation

Seven fresh human cadaveric specimens (four specimens occiput to T1 and three specimens occiput to C7) were carefully dissected, preserving the osteoligamentous structures. The right VA was exposed both at C6 and at the occiput. A thin nylon-coated flexible cable was passed through the VA from the occiput to the outlet of the transverse foramen of C6. The cable was fixed at one end to the occipital bone with a screw and at the other end to the carriage of a vertebral artery transducer (VAT) (Fig. 1A). The occiput and C7 or T1 vertebra were mounted in a quick-setting epoxy for the testing.

Vertebral artery transducer design

The VAT consisted of two main parts: an outer bracket A (fixed to a base plate B) and an inner carriage C (Fig. 1B). The carriage moved smoothly in the bracket in the longitudinal direction with minimal friction and play. In this way, the Hall effect sensor D (A3506LU, Allegro Microsystems, Worcester, Mass.) attached to the carriage moved between two rare earth magnets E ($13 \times 13 \times 5$ mm, part no. PR28ES4187B, Dexter Magnetic, Billerica, Mass.) glued with cyanoacrylate to each end of the bracket. The carriage was connected to the VA line F on one end and to a tension spring G on the other end. The A3506LU Hall effect sensor provided an output voltage proportional to the strength of the magnetic field and required no amplification. The VAT was capable of measuring VA length change in the range of 0–40 mm with a root mean square (RMS) error of 0.64 mm. Technical specifications and calibration details can be found elsewhere [4].

Procedures

The cervical spine specimen was first tested with a standard flexibility protocol under physiological loading conditions [11, 12]. Pure moments of flexion/extension, axial torque, and lateral bending, up to a maximum of 1.0 Nm, were applied to the occiput. Physiological motions of the occiput were measured with respect to the C7 vertebra with an Optotrak motion analysis system (Northern Digital, Waterloo, Ontario, Canada) and VA elongation was recorded with the VAT. We defined the physiological loads as the maximum moments that produced no injuries and, thus, resulted in reproducible

Fig. 2 Average elongations of the right VA under physiological loading during the flexibility test. Pure moments of 0.33, 0.66, and 1.0 Nm were applied in flexion/extension, left and right axial torque, and left and right lateral bending

intervertebral motions. Of note, the intervertebral motions observed here were similar to the cervical spine motions observed *in vivo* [16].

Next, a simulation of a whiplash trauma was performed with the specimen mounted on a specially designed sled [13], which was subjected to 2.5, 4.5, 6.5, and 8.5 g ($1 \text{ g} = 9.81 \text{ m/s}^2$) horizontal acceleration. A head surrogate, free to move within the sagittal plane, was attached to the occiput to simulate the inertial forces acting on the cervical spine during the whiplash. The VA elongation was continuously sampled at 1 kHz using an analog-to-digital converter and a personal computer. Data were digitally filtered with a fourth order, dual pass, Butterworth low pass filter at a cut-off frequency of 30 Hz. Residual and Fourier analysis demonstrated that most of the signal power was contained under 20 Hz.

Results

The maximum VA elongation under physiological loading occurred during left lateral bending (contralateral to the

VAT), and its average (standard deviation) was 5.8 (1.6) mm (Fig. 2). Left axial rotation produced 4.7 (1.8) mm elongation, while flexion produced 1.1 (1.0) mm elongation. Right lateral bending and extension motions resulted in shorter lengths of the artificial VA in comparison to the length in neutral spine posture.

In the whiplash simulations, the maximum VA elongation correlated with the maximum horizontal sled acceleration ($R^2 = 0.7$, $P < 0.05$; Fig. 3). Sample data of dynamic VA elongations v.s. time for one specimen are presented in Fig. 4. Based on such data for all specimens, the average maximum dynamic VA elongations exceeded their physiological values by 1.0 (2.1), 3.1 (2.6), 8.9 (1.6), and 9.0 (5.9) mm in the 2.5-, 4.5-, 6.5-, and 8.5-g trauma classes respectively. The sled accelerations above 4 g pro-

duced statistically significant (one factor ANOVA, $P < 0.05$) vertebral artery elongation in comparison to the physiological maxima established in the flexibility tests (Fig. 5). There was no statistically significant difference in VA elongation between the 6.5-g and 8.5-g trauma classes.

Discussion

We developed an accurate transducer system to measure in situ VA elongation during simulations of physiological motions and during a whiplash trauma with cadaveric cervical spine specimens. In order to estimate the severity of VA stretch, the maximum elongation during whiplash was compared to the maximum elongation recorded during flexibility testing. This flexibility testing applied moments that did not produce injury to the spine and for which the resultant intervertebral motions were reproducible. We termed these elongations physiological.

The maximum VA elongations during the simulated whiplash trauma were on average 9.0 mm over the maximum physiological elongation. This is relatively small stretch when compared with the length of the whole artery. However, in the cervical spine, the anatomical path that the VA takes is divided into two parts. One is the posterior atlanto-occipital membrane through the transverse process of the axis, and the other is the lower part (C3–C6). We measured the total length change of the artery from the occiput to C6. It is not possible to determine in our measurement system the strain distribution of the VA throughout its length. Since the lower portion of the VA runs closer to the flexion/extension centers of rotation of each functional spinal unit, it is possible that the VA stretch takes place mostly in the upper cervical spine and

Fig. 3 Maximum vertebral artery elongations during simulated whiplash trauma at various sled accelerations for all specimens

Fig. 4 An example (one specimen) of the VA elongation during simulated whiplash at 2.5, 4.5, 6.5, and 8.5 g of sled acceleration

Fig. 5 Average (one standard deviation) maximum VA elongation during simulated whiplash trauma at various sled accelerations

the occiput junction. If that were the case, the VA stretch of 9.0 mm above its physiological elongation over a much shorter length may be clinically significant.

There was relatively little stretch of the VA during the flexibility testing, where pure flexion or extension moments were applied. In contrast, the whiplash trauma restricted motion to the sagittal plane and caused VA elongation that was over 14 mm greater than that caused by physiological head flexion or extension (Figs. 2, 5). These results suggest that, during whiplash trauma, other mechanisms than simple flexion/extension of the cervical spine cause the artery stretch. Indeed, the maximum elongation of the VA occurred in the initial stage of whiplash trauma

when the cervical spine formed an S-shaped curvature [6]. This curvature is formed 50–75 ms after the impact, when the head is translated posteriorly with respect to C7 prior to it being rotated into extension.

The main limitation of this study is the *in vitro* approach, in which simulation of active muscle function is not as yet possible. However, it may take more than 200 ms to activate the reflex loop and to develop any significant tension in the muscles when the rear end collision is unexpected [2, 7, 17]. Since the mechanical events in whiplash trauma take place in less than the 200-ms time period, we deem the active muscle function to be of a lesser importance. Of note, the head kinematics obtained in our study compare well with other reports utilizing whole cadavers or volunteers [13]. Therefore, even without passive muscle forces, the validity of our bench-top whiplash model is assured.

Conclusions

Clinical symptoms of whiplash are presently not well understood. Vascular deficiency has been suggested to be one of the causes. This study represents the first attempt to quantify the VA stretch during simulated whiplash trauma. Future research should be directed towards identifying the strain distribution along the artery to establish the sites vulnerable to lesion.

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