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Comment on the paper of Nibu K et al (1997) Dynamic elongation of the vertebral artery during an in vitro whiplash simulation. Eur Spine J 6:286–289

This interesting study experimentally established that in a whiplash injury model hyperelongation of the vertebral artery (VA) occurs, being proportional to horizontal sled acceleration. The VA elongation is related to an S-shaped curvature of the cervical spine, forming 50–70 ms after the impact, when the head is translated posteriorly prior to being rotated into extension.

As the experiment only allowed measurement of VA elongation as a whole, from C6 to the occiput, it did not identify where the elongation took place. Anticipating future research by the authors to identify the strain distribution along the VA and establish the sites vulnerable to lesion, I should like to present a marginal note.

The course of the VA in lateral radiographs images is projected near the average instantaneous centres of flexion-extension motion, not only in the C4–C6 region, but also in the occiput–C2 region [1]. As a consequence, the projected VA path between C6 and the occiput is expected hardly to change in length at all during physiological flexion-extension of head and neck (Fig. 1). The authors have indeed found that there is rela-

tively little stretch of the VA during flexibility testing, where pure flexion or extension moments are applied.

The authors did not investigate changes in VA length during the so-

Fig. 1 Lateral radiographic projection of cervical spine from the occiput to C7, with superimposition of vertebral artery (1) and average instantaneous centres of flexion-extension motion (2; C6/7; 4: C5/6; 6: C4/5; 7: C3/4; 9: C2/3; 14: C1/2; 17: occiput/C1). The artery, passing through the costovertebral foramina (3: C6; 5: C5; 8: C4; 10: C3; 11: C2; 12: C1) and the VA sulcus of C1 (16), runs close to these centres. 12: First vertebral artery loop (overlength for C1/2 rotation); 15: second vertebral artery loop (probable device for damping pulse waves)

called chin-in/chin-out manoeuvre, in which the head translates backward and forward with respect to the trunk, without extension or flexion. During this manoeuvre the upper and lower parts of the cervical spine move in opposite directions [2]. As these opposite motions follow normal flexion or extension patterns in each of the moving segments, it may be expected that the chin-in/chin-out manoeuvre likewise will not produce significant stretch of the VA if pure forward and backward translation moments are applied.

At the end of the chin-in manoeuvre (as the head has been maximally translated backward with respect to the trunk), the upper cervical spine is maximally flexed, and the lower cervical spine and cervicothoracic region extended, although not maximally [2]. As the physiological range of backward translation of the head is relatively small, maximal occiput–C2 flexion is reached relatively soon.

Further (traumatic) backward translation of the head, therefore, is likely to produce hyperflexion/subluxation injury of the craniovertebral region. As the lower cervical spine is not maximally extended at the end of the chin-in manoeuvre, traumatic backward translation of the head is presumed not to have a damaging effect upon this region, at least not during the initial stage.

The mechanism of whiplash injury, defined as sudden backward ac-

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Fig. 2 Initial stage of whiplash injury. The head is forcibly translated backward beyond the physiological range (1) causing hyperflexion of the craniovertebral region (3) with kyphotic curve of the spinal canal (2), and extension of the lower cervical region (4) with lordotic curve (5)

celeration of the head with respect to the trunk, has generally been presented and understood as *hyperextension* of the head and neck, at least during the initial stage of the injury [3]. However, study of animal sled acceleration experiments [4] and autopsy results of acceleration injury victims [5] lead me to the surprising

conclusion [6] that during the initial stage of whiplash injury the head translates backward (as in the chin-in manoeuvre), causing not hyperextension but *hyperflexion* of the occiput-C2 region, as shown in Fig. 2.

The so-called S-shaped curve, observed by the authors during the initial stage of their simulated whiplash experiments, in fact reflects the predicted result of backward hypertranslation of the head, causing hyperflexion of the upper cervical spine with a markedly kyphotic curve and (normal) extension of the lower cervical region with a lordotic curve. Although my prediction was based on extrapolation of cervical spine motion during normal backward translation of the head [6], the experimental results of the authors now seem to bear out that hyperflexion of the upper cervical spine is indeed the initial pathological event in whiplash injury.

The hyperflexion/subluxation of the upper cervical spine, combined with extension of the lower cervical spine remaining within normal limits, suggests that the location of the experimentally proven hyperelongation of the VA has to be primarily sought at the craniovertebral region.

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