

Cervical artery dissection: a biomechanical perspective

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Introduction

Although there has been a putative link between cervical spinal manipulative treatment (cSMT) and cervical artery dissection (CAD) ever since Thornton's report in the literature in 1934¹, recent evidence suggests that this is an association rather than a causal relationship. Since 2008, several studies published by Cassidy and co-workers²⁻⁴ have attributed the association between cSMT and CAD to patients seeking chiropractic care for neck pain and headaches during the prodrome of a stroke.

Most reviews in the literature now generally report that there are no convincing data, either to prove or disprove, any causality between cSMT and CAD.⁵ However, case reports and case series still accumulate that identify chiropractic as the sole cause of CAD.⁶⁻⁷ Furthermore, Tuchin⁸ recently tested the causality between CAD and SMT using Hill's criteria, and concluded that there is no evidence that SMT is causally related to stroke. Nevertheless, some authors continue to claim that cSMT causes CAD.

Rather than using an epidemiologic approach to assess the risk of whether cSMT can cause CAD, another approach is to investigate the mechanism(s) of how cSMT can cause CAD. Since 2002⁹, our laboratory has focused on the latter strategy. Using cadaveric vertebral arteries (VAs) as a model for the *in vivo* neck, we have measured the strains experienced by VAs using ultrasonography to dynamically measure the changes in VA segment lengths during manipulative procedures. The details of the experimental procedures have been described elsewhere.⁹⁻¹¹ We have now replicated these experiments on a total of 16 VAs obtained from 10 cadavers⁹⁻¹¹ in 3 different papers.

Table 1:

Vertebral Artery (VA) and Internal Carotid Artery (ICA) strains obtained during Diversified-style cervical manipulation. For VA, results of the V3 and V1 segments are given. Strains during range of motion (ROM) are also given for reference, as are the strains at which gross mechanical failure was first observed (Fail). ND indicates that no data are available. Note that the strains for the cervical manipulation trials are typically less than 50% of those obtained during normal ROM testing.

	VA V3	VA V1	ICA	ROM	Fail
Symons et al. ⁹	5.2%	2.7%	ND	12.5%	62%
Wuest et al. ¹⁰	2.6%	ND	ND	10.8%	ND
Herzog et al. ¹¹	3.8%	0.9%	ND	12.2%	58%
Herzog et al. ¹²	ND	ND	2.0%	7.1%	59%

Please note that we were unable to pool these data due to statistical considerations and advances in the experimental procedure.

More recently, we have extended these experiments to investigate the strains experienced by the internal carotid artery (ICA) during cSMT utilizing essentially the same experimental protocol.¹²

Table 1 shows a summary of the data obtained thus far. The mechanical engineering strains experienced by the V3 segment of the VA, the segment which exits from the C2 transverse foramen and loops around C1 into the foramen magnum, during a Diversified-style cervical manipu-

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lation at C2-C4 vertebral levels ranged from 2.6 to 5.0%. In other words, the V3 segment stretches by roughly 3-5% of its resting length during SMT. In comparison, passive range of motion (ROM) testing exerted a peak 11-13% strain. Typically, contralateral cervical rotation caused the greatest strain values, and hence the ROM testing generally constitutes a test of tolerance to contralateral rotation. The VA underwent gross mechanical failure at 58-62% strain.

When these experiments were replicated on the ICA, similar trends were observed; the ICA underwent 2% strain during cSMT, stretched to 7% strain during contralateral rotation, and failed mechanically at 59% strain.

It is clear from these data that Diversified-style cSMT exerts roughly half or less of the strain that the vessel (VA or ICA) undergoes during ROM movements of the neck, such as shoulder-checking while driving. If the putative mechanism of injury is that forceful SMT causes excessive stretching on the VA or ICA, thus tearing the vessel wall and causing a dissection, then this is highly unlikely to occur in the absence of a pre-existing injury (such as prodromal dissection) or pre-existing condition (such as collagen disease). If cSMT can indeed precipitate a CAD, then any similar movement of the neck should be a sufficient cause, since the biomechanics of cSMT have now been studied in detail.¹³

Although most of the literature has focused on the VA rather than the ICA for the association between cSMT and stroke, there have been several reports on ICA dissection linked to cSMT.^{14,15} However, these reports are rare, and there is no clear association between ICA dissection and cSMT.¹⁶

Armed with the epidemiologic studies from Cassidy and co-workers plus the biomechanical evidence described above, it is reasonable that most clinicians tend to discount any association between CAD and cSMT. Furthermore, Murphy¹⁷ suggested that with the recent evidence pointing towards a temporal association rather than a causal association, chiropractors should shift their focus from managing CAD as a complication of SMT to recognizing patients who are in the prodromal phases of CAD. However, case reports on chiropractors causing CAD still continue to accumulate despite the growing body of basic research and epidemiology to the contrary. Researchers in this area need to continue their investigations into the association between CAD and cSMT.

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