

Chiropractic Philosophy & Clinical Technique

Chiropractic manipulation and cervical artery dissection

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Millions of cervical spine manipulations (CSMs) are performed each year in the United States, yet only a handful of associated cervical artery dissections (CADs) are reported. In Canada, for instance, only 23 cases of stroke or vertebral artery (VA) dissection were reported to be associated with CSM during a 10-year period in which approximately 135 million procedures were performed. Estimates of the incidence of manipulation-related CAD are for the most part based on retrospective analyses and vary widely, ranging from 1 in 400,000 to 1 in 5.85 million cervical manipulations.^{1,2} Probably the most commonly quoted statistic is 1 in 1 million.³ Precise estimates are not available and would be nearly impossible to obtain, because it would require a prospective trial involving millions of patients receiving CSM over an extended period of time to make an accurate estimate.

There is a great deal of controversy surrounding the etiology of CAD in general, and in particular whether the relationship between CSM and CAD is causal. Some assert that patients often go to doctors of chiropractic with CADs that are already in progress for treatment of associated neck pain and headache.⁴ The evolving stroke signs and symptoms that develop subsequent to CSM might have occurred anyway as part of the natural course of the pathology. In contrast, others assert “. . . there is no doubt that chiropractic neck manipulation can result in dissec-

tion of the carotid or vertebral arteries leading to stroke.”⁵

The purpose of this overview is to explore the relationship between CSM and CADs and to suggest appropriate patient management strategies for those who present for CSM treatment and have signs of CAD, or who develop such signs following CSM.

Proposed Mechanism of Arterial Dissection

Arterial dissection is an uncommon vascular wall condition that usually involves a tear in the artery's lining and the formation of an intimal flap, which allows blood to penetrate into the muscular layer of the vessel wall. Blood flowing between the layers of the torn blood vessel may cause the layers to separate, resulting in arterial narrowing or even complete obstruction of the lumen. Pulsatile pressure may damage the muscular layer, resulting in a splitting or dissection of the intimal and medial layers that may extend along the artery variable distances, typically in the direction of blood flow.^{6,7} (See Fig. 1)

Subintimal hematoma develops after the intimal lining separates from the media and blood begins to accumulate in the vessel wall. The accrued blood soon develops into a thrombus and deforms the intima into the arterial lumen. Blood flow in the cervical arteries can be obstructed directly by the subintimal hematoma, or emboli may detach from the thrombus and travel distally to obstruct the progressively smaller vessels in the

brain, which results in brain ischemia (stroke). Most commonly, it is the release of emboli that causes brain ischemia secondary to CAD.⁸ (See Fig. 2)

There are many reports in the literature of CAD occurring subsequent to a variety of traumas.⁹ CADs have also occurred, however, following very minor events and even everyday activities that most people would consider non-traumatic.¹⁰ In some cases, no potentially triggering activity could be identified. The latter variety of CADs would be considered spontaneous.¹¹ A review that considered CAD studies that involved 5 or more patients during the 10-year period between 1994 and 2003 reported that 61% of them were classified as spontaneous.¹²

The VA seems most susceptible to strain in the atlantal segment as it traverses the C1-C2 articulation,¹³ where it appears most at risk during rotational movements.¹⁴ The proposed mechanism of arterial strain during neck rotation involves the ipsilateral C1-C2 joint, while the contralateral side of C1 is pushed forward, compressing and stretching the artery. (See Fig. 3)

The internal carotid artery (ICA) is most susceptible to strain in the upper cervical region when the head and neck are in rotation or lateral flexion combined with hyperextension.^{15,16} This position has been theorized to fix the otherwise freely moveable ICA against the anterior surface of C1 or C2, making it susceptible to injury when sufficient force is applied.¹⁷ (See Fig. 4)

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The exact pathogenesis of CAD is uncertain, especially in cases that occur spontaneously, or are related to trivial trauma. Many researchers think that an underlying arterial abnormality must be present for dissection to occur.¹⁸⁻²⁰ Its etiology is probably multifactorial and related to a variety of arteriopathies that are produced by an assortment of genetic and environmental factors.^{21,22}

The outcome of CAD is unpredictable, with some patients obtaining a full recovery, some suffering permanent neurological deficits, and a small number of patient deaths. For instance, a series of 126 CAD patients revealed that after a maximum of 6 months, 88 patients made an excellent recovery, 22 had a mild to moderate handicap, 15 were severely disabled, and 1 died⁹. Common long-term neurological problems in CAD patients include incoordination, numbness, speech or swallowing dysfunction, and visual disturbances.²³

CAD Occurring in Association with Manipulation

All studies that have investigated the relationship between CSM and CAD have used research designs that are not capable of determining causation. The same can be said about other CAD risk factors (e.g., connective tissue abnormalities,¹⁹ recent infection,²⁴ hyperhomocysteinemia,^{22,25} hypertension,²⁶ etc.). What is known about CSM and CAD, however, is that the 2 events are, on rare occasions, chronologically associated. That is to say, a patient is manipulated and afterward develops dissection-related signs or symptoms. What is unknown about this association is whether it is causal. This is because, among other things, affected patients do not live in a vacuum and are typically exposed to other potentially causal events before and

after the manipulation. For instance, it is common for people to turn their heads to back up cars, have their hair washed at a beauty shop, practice yoga, sleep on their stomachs, get angry, or experience some other event that has also been temporally linked with CAD.¹⁰ In addition, since most CADs develop in the absence of any discernable mechanical event, how can one ever say that manipulation caused a particular stroke?

Notwithstanding the lack of a clear cause-and-effect relationship, nearly 10% of CADs that were reported in the literature from 1994-2003 occurred following CSM.¹² This association warrants closer investigation by the chiropractic profession in an effort to minimize, if possible, the likelihood of these sometimes catastrophic incidents. In keeping with this goal, a variety of strategies have been proposed. Two primary ones include better identification of potential CAD patients and the development of improved manipulative techniques. That being said, a study that considered 64 CAD cases that were temporally associated with cervical spine manipulation found that CAD-related strokes followed any form of standard cervical manipulation, including rotation, extension, lateral flexion, neutral position, and even non-force manipulations. The authors concluded that CAD should be considered a random and unpredictable complication of any neck movement including cervical manipulation. Some of the strokes in this study occurred after just 1 manipulation, while others occurred only after many manipulations; thus, no dose-response relationship was observed.²⁷ The authors also pointed out that 92% of the patients sought treatment because of headache and neck pain; symptoms which, at least in some cases, may actually have been caused by a

CAD in progress. Subsequent manipulation may have had nothing to do with the development of ischemic neurological signs or it may have served as a trigger to provoke the condition. Nevertheless, the other 8% in this study who did not have head or neck pain, yet still developed post-manipulation CAD, are unexplained by this rationale.

Some have theorized that increased peak blood flow velocity in the VAs following CSM may be a reasonable explanation for the rare instances where CAD subsequently develops. To test this hypothesis, a randomized, controlled, and observer-blinded study investigated blood flow changes in the VA following cervical manipulation. Researchers measured VA peak flow velocity both before and after cervical manipulation using Doppler ultrasound. No significant differences in VA peak flow velocity, however, were found following manipulation. The authors concluded that increased peak blood flow velocity was not affected by uncomplicated cervical manipulation.²⁸

Peak VA blood flow changes were noted by Seric, et al.²⁹ following minor whiplash injury. Blood flow velocities were either increased or decreased in 68% of 47 whiplash patients 1 month post-injury, which was still present in 50% of the subjects at follow-up 6 months later. The authors concluded that blood flow in the VAs could be affected by cervical spine lesions and produce symptoms of vertebrobasilar insufficiency. One would think that if blood flow irregularities were capable of producing spontaneous or trivial trauma-related CAD, the condition would be seen regularly in whiplash patients. Whiplash is rarely cited, however, as a trigger of spontaneous CAD, although it is com-

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monly associated with the traumatic variety.³⁰

The contrast between the findings of these studies leads to an enigma about whether cervical manipulation is even capable of causing CAD, and lends support to the viewpoint that patients who develop CAD post-manipulation may actually have had the condition prior to the intervention. As stated in a previous review on this issue, it is doubtful that a healthy VA is at risk from contemporary CSM performed by a licensed doctor of chiropractic.¹⁸ Furthermore, a commentary by Rubenstein, et al.³¹ suggested that stroke following manipulation is unlikely in a healthy person who does not have prerequisite risk factors. Considering all of the etiologic possibilities of CAD, it is the inherent fragility of the arterial wall, rather than any trauma associated with manipulation, that is the major culprit in the causation of CADs.³²

Patients Presenting to DCs with Musculoskeletal Symptoms of CAD

Approximately 700,000 strokes occur every year in the U.S., with 88% of them classified as ischemic. Researchers have estimated that 2% of the ischemic variety of strokes are caused by CAD³³, which means that the annual incidence of CAD in the U.S. may be around 12,000 cases. Another way to ascertain the annual incidence of CAD is derived from the combined incidence rates of VA and ICA dissection, which totals about 4 cases per 100,000 persons per year. This rate is an average of the reported incidence rates of ICA dissection, which is 2.6 to 2.9 per 100,000,^{33,34} and VA dissection, which is 1 to 1.5 per 100,000.³⁵ At this rate, the same value of approximately 12,000 cases per year results. Some of

these people, who often have neck pain and headache as the early symptoms of CAD,³⁶ will undoubtedly seek chiropractic care. If signs of cerebral ischemia develop following CSM in these patients, it does not necessarily mean that manipulation caused the dissection. Moreover, since CAD frequently deteriorates on its own, without associated mechanical triggers or risk factors, manipulation may have nothing to do with the condition.

Differentiating Musculoskeletal from Dissection-Related Symptoms

There are, unfortunately, no valid functional vascular screening tests that are capable of detecting patients who are likely to develop manipulation-related CAD.⁴ Indeed, a review of 64 medicolegal cases of manipulation-related stroke found that a pre-manipulative provocative screening test was utilized, but was negative, in 27 of the cases.²⁷ In other words, it failed to detect which patients would develop stroke following cervical manipulation 100% of the time.

Thiel and Rix³⁷ maintain that pre-manipulative provocative tests fail to satisfy the necessary requirements of a screening test because they are not capable of identifying the pre-symptomatic condition they were designed to detect. The authors made some observations and recommendations on this topic that are especially relevant to chiropractic practice. They are as follows:

1. Practitioners must assess the patient thoroughly, through careful history taking and physical examination, for the possibility of VA dissection. It is important to note that CAD may present as pain only, and may not be associated with symptoms and signs of brainstem ischemia.

2. If there is a strong likelihood of CAD, provocative pre-manipulation tests should not be performed, and the patient must be referred appropriately.

3. In the patient presenting with symptoms of brainstem ischemia due to non-dissection stenotic VA pathologies, provocative testing is very unlikely to provide any useful additional diagnostic information.

4. In the patient with unapparent VA pathology, where manipulation is considered as the treatment of choice, provocative testing is very unlikely to provide any useful information in assessing the probability of CSM-induced VA injury.

5. Practitioners may well now consider whether provocative testing provides any real benefit to any of these patient populations.

Because of the poor validity of functional vascular screening tests, the optimal pre-cervical manipulation patient management strategy is to identify high-risk patients at the time of the initial evaluation, especially those that exhibit signs or symptoms of cerebral ischemia, and then either modify chiropractic techniques/therapies or refer the patient to an appropriate medical provider,⁴ DCs may employ the following strategies to prevent manipulation-related CADs or to improve outcomes in the rare case they do occur:

1. Identify high-risk patients before treatment and modify or avoid CSM (e.g., patients with heritable connective tissue disorders, atypical migraine syndromes or hypertension).

2. Identify patients with frank signs or symptoms of cerebral ischemia. Do not perform CSM, but refer them for appropriate medical care, in some cases via emergency transport.

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3. Identify patients who develop CAD post-manipulation and take immediate action as described in step 2.

A sudden and severe onset of neck pain and headache frequently described as different from previous experience, especially when it affects the sub-occipital region, may be indicative of a CAD in progress.³⁸ Patients presenting with these symptoms require extra consideration if cervical manipulation is to be utilized. In addition to sudden pain, Terrett³⁹ described 9 signs and symptoms of vertebro-basilar ischemia, termed the 5 Ds and 3 Ns, which every doctor of chiropractic should be very familiar with. They are as follows:

1. **D**iplopia – not only double vision, but infrequently may involve other vision problems.

2. **D**izziness – may range in severity from unexplained light-headedness to frank vertigo.

3. **D**rop attacks – sudden weakness of the face, arm or leg, which may result in a sudden, unexplained fall.

4. **D**ysarthria – consisting of impaired speech or hoarseness.

5. **D**ysphagia – difficulty swallowing.

Ataxia – difficulty walking, falling or veering to the side.

1. **N**ausea – may range in severity from queasiness to vomiting.

2. **N**umbness – loss of sensation on one side of the body.

3. **N**ystagmus – involuntary rhythmic eye movements.

Although manipulation-related VA dissection is reported much more commonly, the incidence of ICA dissection is actually higher than VA dissection in the general

population.⁶ DCs should therefore be familiar with its presenting signs and symptoms and be able to recognize affected patients. In addition to neck pain and headache, which are the most common presenting symptoms, these patients may have transient ischemic attacks that affect the cerebral cortex, transient monocular loss of vision (known as amaurosis fugax), or an incomplete Horner's Syndrome that includes miosis and ptosis, but not anhidrosis. Indeed, a patient with Horner's Syndrome that is painful and of sudden onset is strongly suggestive of ICA dissection. Other possible symptoms include subjective bruit, distorted sense of taste (known as dysgeusia), and visual scintillations.⁴⁰

CAD can be very difficult to diagnose, even under the best circumstances. As a result, it is often overlooked in clinical practice, especially in its early stages before ischemic signs are evident.⁴¹ Cases that only cause mild symptoms may actually go undiagnosed.⁴² Asymptomatic CADs have also been reported, which were only detected by chance during imaging studies of the neck that were prompted by other suspected conditions. This suggests that some cases are never discovered and probably heal spontaneously. At any rate, DCs should be attentive to the subtle clues manifested in these patients to more effectively identify those with CADs, and then refer them for appropriate treatment.

Conclusion

Several trivial events, including cervical spine manipulation, have been associated with dissection of the cervical arteries. This association is confusing because people normally encounter innumerable

trivial events during their lifetime without ever experiencing cervical artery dissection. In fact, the overall incidence of CADs is extremely low.

Most CADs occur spontaneously and only 9% have been reported to be associated with manipulation. No cause-and-effect relationship has been established between CSM and CAD. There is anecdotal evidence suggesting that cervical manipulation may be capable of triggering dissection in a susceptible patient or contributing to the evolution of an already existing CAD.

Several risk factors described in the literature are thought to contribute to the development of CAD. On the other hand, some patients experience CAD without any of them. The most compelling risk factors have to do with connective-tissue abnormalities, which may contribute to a weakening of the vascular wall, making it more susceptible to tearing. In spite of the many risk factors that have been proposed as possible causes of CAD, it is still unknown which of them, if any, actually predispose patients to CAD following CSM. Thus, it is not possible at this time to accurately identify patients at risk of CAD prior to CSM.

Although the frequency is unknown, patients occasionally present to chiropractic offices with early symptoms of CAD or have risk factors that may make them vulnerable to dissection. The association between CAD and CSM is so rare that, on average, 3 out of 4 DCs will go through their entire professional careers without ever being connected with such an event. Attention to possible red flags may serve to lower this ratio still further. ■

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Fig. 1.

A) Intimal tear and formation of an intimal flap. B) Blood penetrates into the muscular layer of the torn blood vessel and causes the layers to separate.

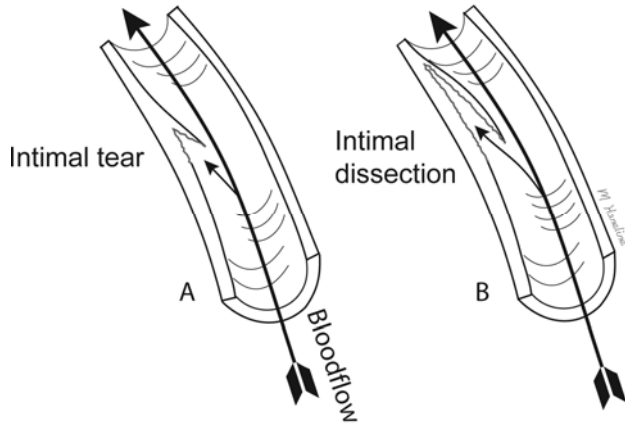


Fig. 2. A) Blood develops into a thrombus, deforming the intima into the arterial lumen. B) Emboli may detach from the thrombus and travel distally to obstruct the progressively smaller vessels in the brain.

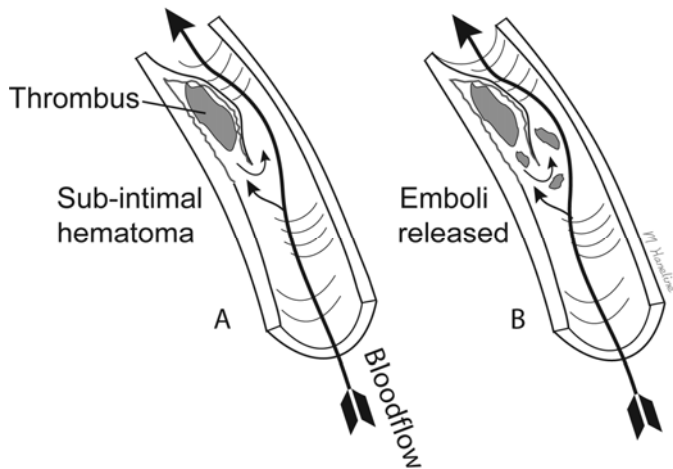
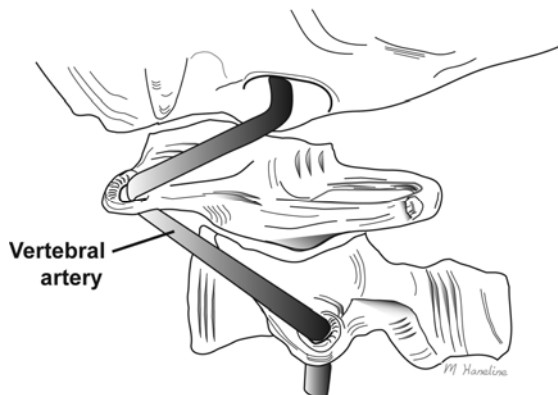
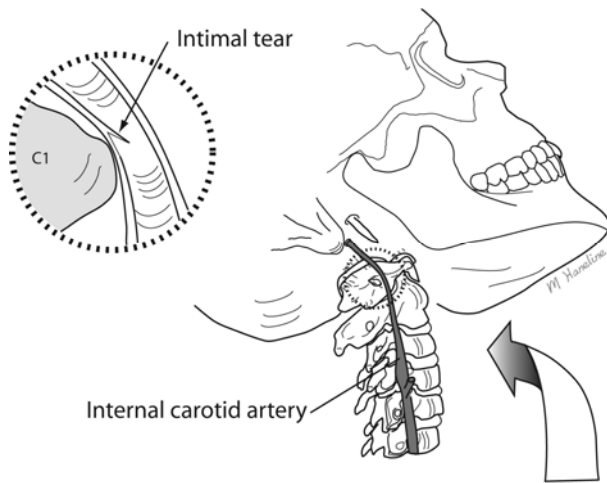


Fig. 3. During neck rotation, the ipsilateral C1-C2 joint is fixed, while the contralateral side of C1 is propelled forward, which compresses and stretches the vertebral artery.



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Fig. 4. The internal carotid artery ICA is most susceptible to strain in the upper cervical region when the head and neck are in combined rotation or lateral flexion and simultaneous hyperextension.



References

1. Haldeman S, Carey P, Townsend M, Papadopoulos C. Arterial dissections following cervical manipulation: the chiropractic experience. *CMAJ* 2001 Oct 2;165(7):905-906.
2. Dvorak J, Orelli FV. How dangerous is manipulation to the cervical spine? *Manual Med* 1985;2(1):1-4.
3. Hosek RS, Schram SB, Silverman H, Myers JB, Williams SE. Cervical manipulation. *JAMA* 1981 Mar 6;245(9):922.
4. Triano J, Kawchuk GN, eds. *Current Concepts in Spinal Manipulation and Cervical Arterial Incidents*. West Des Moines, IA: NCMIC Group Inc; 2005.
5. Norris JW, Beletsky V, Nadareishvili ZG. Sudden neck movement and cervical artery dissection. The Canadian Stroke Consortium. *CMAJ* 2000 Jul 11;163(1):38-40.
6. Mokri B, Sundt TM, Jr., Houser OW, Piepgras DG. Spontaneous dissection of the cervical internal carotid artery. *Ann Neurol* 1986 Feb;19(2):126-138.
7. Zetterling M, Carlstrom C, Konrad P. Internal carotid artery dissection. *Acta Neurol Scand* 2000 Jan;101(1):1-7.
8. Benninger DH, Georgiadis D, Kremer C, Studer A, Nedeltchev K, Baumgartner RW. Mechanism of Ischemic Infarct in Spontaneous Carotid Dissection. *Stroke* 2004 Feb 1;2004;35(2):482-485.
9. Dziewas R, Konrad C, Drager B, et al. Cervical artery dissection--clinical features, risk factors, therapy and outcome in 126 patients. *J Neurol* 2003 Oct;250(10):1179-1184.
10. Rome P. Perspectives: an overview of comparative considerations of cerebrovascular accidents. *Chiropr J Aust* 1999;29:87-102.
11. Kochan JP, Kanamalla US. Carotid and Vertebral Artery Dissection. *eMedicine.com* [Web page]. June 23, 2006. Available at www.emedicine.com/radio/topic132.htm. Accessed August 12, 2006.
12. Haneline MT, Lewkovich GN. An analysis of the etiology of cervical artery dissections: 1994 to 2003. *J Manipulative Physiol Ther* 2005 Oct;28(8):617-622.
13. Hufnagel A, Hammers A, Schonle PW, Bohm KD, Leonhardt G. Stroke following chiropractic manipulation of the cervical spine. *J Neurol* 1999 Aug;246(8):683-688.
14. Davis JM, Zimmerman RA. Injury of the carotid and vertebral arteries. *Neuroradiol* 1983;25(2):55-69.
15. Stringer WL, Kelly DL, Jr. Traumatic dissection of the extracranial internal carotid artery. *Neurosurg* 1980 Feb;6(2):123-130.
16. Fabian TC, et al. Blunt carotid injury. Importance of early diagnosis and anticoagulant therapy. *Ann Surg* 1996 May;223(5):513-522; discussion 522-515.
17. Lepojarvi M, Tarkka M, Leinonen A, Kallanranta T. Spontaneous dissection of the internal carotid artery. *Acta Chir Scand* 1988 Oct;154(10):559-566.

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18. Haneline M, Triano J. Cervical artery dissection. A comparison of highly dynamic mechanisms: manipulation versus motor vehicle collision. *J Manipulative Physiol Ther* 2005 Jan;28(1):57-63.
19. Brandt T, Orberk E, Weber R, et al. Pathogenesis of cervical artery dissections: Association with connective tissue abnormalities. *Neurology* 2001 July 10;57(1):24-30.
20. Schievink WI, Mokri B, Piepgras DG. Spontaneous dissections of cervicocephalic arteries in childhood and adolescence. *Neurology* 1994 Sep;44(9):1607-1612.
21. Brandt T, Grond-Ginsbach C. Spontaneous cervical artery dissection: from risk factors toward pathogenesis. *Stroke* 2002 Mar;33(3):657-658.
22. Gallai V, Caso V, Paciaroni M, et al. Mild Hyperhomocyst(e)inemia : A Possible Risk Factor for Cervical Artery Dissection. *Stroke* 2001 Mar 1;2001;32(3):714-718.
23. Haldeman S, Kohlbeck FJ, McGregor M. Unpredictability of cerebrovascular ischemia associated with cervical spine manipulation therapy: a review of sixty-four cases after cervical spine manipulation. *Spine* 2002 Jan 1;27(1):49-55.
24. Guillon B, Berthet K, Benslamia L, Bertrand M, Bousser MG, Tzourio C. Infection and the risk of spontaneous cervical artery dissection: a case-control study. *Stroke* 2003 Jul;34(7):e79-81.
25. Pezzini A, et al. Hyperhomocysteinemia A potential risk factor for cervical artery dissection following chiropractic manipulation of the cervical spine. *J Neurol* 2002 Oct;249(10):1401-1403.
26. Pezzini A, Caso V, Zanferrari C, et al. Arterial hypertension as risk factor for spontaneous cervical artery dissection. A case-control study. *J Neurol Neurosurg Psychiatry* 2006 Jan 1;77(1):95-97.
27. Haldeman S, Kohlbeck FJ, McGregor M. Stroke, cerebral artery dissection, and cervical spine manipulation therapy. *J Neurol* 2002 Jul;249(8):1098-1104.
28. Licht PB, et al. Vertebral artery flow and spinal manipulation: a randomized, controlled and observer-blinded study. *J Manipulative Physiol Ther* 1998 Mar-Apr;21(3):141-144.
29. Seric V, Blazic-Cop N, Demarin V. Haemodynamic changes in patients with whiplash injury measured by transcranial Doppler sonography (TCD). *Coll Antropol* 2000 Jun;24(1):197-204.
30. Beaudry M, Spence JD. Motor vehicle accidents: the most common cause of traumatic vertebrobasilar ischemia. *Can J Neurol Sci* 2003 Nov;30(4):320-325.
31. Rubinstein SM, Haldeman S, van Tulder MW. An etiologic model to help explain the pathogenesis of cervical artery dissection: implications for cervical manipulation. *J Manipulative Physiol Ther* 2006 May;29(4):336-338.
32. Rosner A. Risks of cerebrovascular accidents in perspective. *Manuelle Medizin* 2003 Jun;41(3):215-223.
33. Schievink WI, Mokri B, Whisnant JP. Internal carotid artery dissection in a community. Rochester, Minnesota, 1987-1992. *Stroke* 1993 Nov;24(11):1678-1680.
34. Giroud M, Fayolle H, Andre N, et al. Incidence of internal carotid artery dissection in the community of Dijon. *J Neurol Neurosurg Psychiatry* 1994 Nov;57(11):1443.
35. Schievink WI. Spontaneous dissection of the carotid and vertebral arteries. *NEJM* 2001 Mar 22;344(12):898-906.
36. Arnold M, Cumurciuc R, Stapf C, Favrole P, Berthet K, Bousser M-G. Pain as the only symptom of cervical artery dissection. *J Neurol Neurosurg Psychiatry* 2006 Sep 1;77(9):1021-1024.
37. Thiel H, Rix G. Is it time to stop functional pre-manipulation testing of the cervical spine? *Man Ther* 2005 May;10(2):154-158.
38. Sturzenegger M. Headache and neck pain: the warning symptoms of vertebral artery dissection. *Headache* 1994 Apr;34(4):187-193.
39. Terrett AGJ. *Current concepts: Vertebrobasilar complications following spinal manipulation*. West Des Moines: NCMIC Group, Inc.; 2001.
40. Desfontaines P, Despland PA. Dissection of the internal carotid artery: aetiology, symptomatology, clinical and neurosonological follow-up, and treatment in 60 consecutive cases. *Acta Neurol Belg* 1995 Dec;95(4):226-234.
41. Treiman GS, Treiman RL, Foran RF, et al. Spontaneous dissection of the internal carotid artery: a nineteen-year clinical experience. *J Vasc Surg* 1996 Oct;24(4):597-605; discussion 605-597.
42. Mokri B. Traumatic and spontaneous extracranial internal carotid artery dissections. *J Neurol* 1990 Oct;237(6):356-361.