ASSOCIATION OF INTERNAL CAROTID ARTERY DISSECTION AND CHIROPRACTIC MANIPULATION

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BACKGROUND– To determine the relationship between chiropractic manipulative therapy (CMT) and internal carotid artery dissection (ICAD), a MEDLINE literature search was performed for the years 1966 through 2000 using the terms *internal carotid dissection.* Literature that included information concerning causation of ICAD, as well as all case studies and series, was selected for review.

REVIEW SUMMARY– In reviewing the cases of internal carotid dissection potentially related to CMT, there were many confounding factors, such as connective tissue aberrations, underlying arteriopathy, or coexistent infection, that obscured any obvious cause-and-effect relationship. To date there are only 13 reported cases of ICAD temporally related to CMT. Most ICADs seem to occur spontaneously and progress from local symptoms of headache and neck pain to cortical ischemic signs. Approximately one third of the reported cases were manipulated by practitioners other than chiropractic physicians, and because of the differential risk related to major differences in training and practice between practitioners who manipulate the spine, it would be inappropriate to compare adverse outcomes between practitioner groups.

CONCLUSIONS– The medical literature does not support a clear causal relationship between CMT and ICAD. Reported cases are exceedingly scarce, and none support clear cause and effect.

KEY WORDS *chiropractic manipulation, internal carotid artery dissection, stroke*

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The medical literature contends that chiropractic manipulative therapy (CMT) applied to the cervical spine may be a cause of internal carotid artery dissection (ICAD) (1–4). The first report attributing ICAD to cervical CMT was by Beatty in 1977 (3), who described a patient who was manipulated by a chiropractic physician for the complaint of neck pain. There were no immediate ill effects reported after the manipulation, and symptomatic relief of neck pain resulted. Five days later, the patient awoke with right arm weakness and difficulty in verbal expression attributable to ischemic effects of ICAD. Beatty ascribed the ICAD to the CMT, asserting that because a tear of the arterial intima was found at surgery, there necessarily must have been trauma, and cervical CMT was the only known trauma before the dissection. More recently, investigators have observed that tearing of the intima is common in cases of spontaneous ICAD with no known associated trauma (5–8). Some state that such tearing is a requisite condition for dissection (8). The Beatty commentary is by far the most frequently cited reference by contemporary authors when connecting CMT with ICAD (1,9–16).

It has been established that vertebral artery dissection (VAD) can be triggered by CMT (17–20), based on the anatomic arrangement of the vertebral artery within the cervical vertebrae, the stress within the artery that takes place in the course of neck rotation, as well as the numerous reports of VAD that are in close temporal relationship to cervical CMT. However, there is still some debate concerning the relationship between VAD and CMT, because pa-

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tients with neck pain may consult a chiropractic physician with an already established dissection (21). Nonetheless, the relationship of VAD to CMT is well recognized and accepted by most within the chiropractic community, whereas the connection between ICAD and cervical CMT is not as clear.

MATERIALS AND METHODS

A MEDLINE literature search was performed for the years 1966 through 2000, using the search terms *internal carotid dissection*. English language articles that included information concerning causation and all case studies and series were selected for review. Initially, 170 items were retrieved from the literature search, and appropriate first-round selections were made. From these selections, 75 articles were accepted as being applicable. Additional references were collected from citations incorporated within the articles.

Anatomy of the Internal Carotid Artery

The internal carotid artery (ICA) arises from the common carotid artery at its bifurcation and has 4 named segments. The cervical segment is situated vertically in the neck, lying posterior to the external carotid artery. The ICA courses below the sternocleidomastoid muscles and is separated from the external carotid artery in its superior portion by the styloglossus and stylopharyngeal muscles. It lies anterior to the longus cervicis muscle and anterior to the transverse processes of the upper 3 or 4 cervical vertebrae (10). At the base of the skull, the ICA becomes the petrous segment as it enters the carotid canal. As the artery courses through the skull, it is referred to as the cavernous segment and ultimately becomes the supraclinoid segment (22). The internal carotid artery has numerous branches supplying blood to major regions of the brain. The middle cerebral artery and the junction between the middle and anterior cerebral arteries are most often affected by ICAD-associated emboli (23). The ICA is freely moveable within its cervical pathway but becomes fixed to the surface of the bone as it enters the carotid canal above the atlas (24).

Pathophysiology of ICAD

Dissection of the ICA may be caused by an initial tear in the intima, followed by blood penetrating into the vessel wall or by a primary intramural hemorrhage of the vasa vasorum that ruptures into the true lumen (7,9,11) (Figure 1). There is subsequent extension of the dissection across varying distances along the length of the artery, typically in the direction of blood flow (6,25). ICAD characteristically originates at a level approximately 2 cm distal to the bifurcation, near the level of the second and third or third and fourth intervertebral spaces (26). The dissection may occur in closer proximity to the intima, termed subintimal dissections, or closer to the adventitia, designated as subadventitial dissections (7). When subintimal dissections occur, the intimal lining is forced away from the tunica media of the artery as circulating blood invades the area. This effectively narrows the arterial lumen. The subintimal layer is always affected by dissection (5). Subadventitial dissections may cause dilation of the artery and are often referred to as dissecting aneurysms or pseudoaneurysms. These develop in 25% to 35% of all ICAD patients (6,9,27). Occasionally, a double lumen is formed when the subintimal hemorrhage ruptures back into the arterial lumen distally (28) (Figure 2). On follow-up, the dissected artery often recanalizes, and aneurysms that may have developed may show regression (27). Based on two series of cases comprising 96 patients, ICAD leads to stroke in 36% to 68% of patients attributable to occlusion of the artery at or near the site of the dissection or distal embolization from a dislodged fragment of thrombus (23,12).

Causation

Extracranial ICAD is classified as being either spontaneous or traumatic (blunt trauma), depending primarily on the occurrence of a premorbid injury (29). Patients with ICAD who have only experienced minimal or trivial trauma are often classified as spontaneous, whereas the traumatic classification is reserved for cases associated with definite, and often severe, trauma. The spontaneous category of ICAD represents the most frequent manifestation of the disease (26).

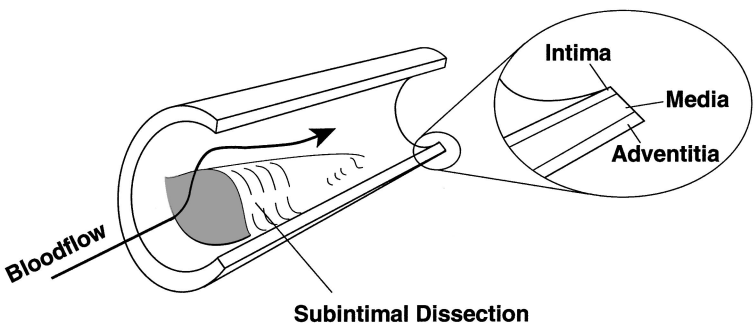

Figure 1.
Internal carotid artery dissection with tearing of the intima and blood penetrating into the vessel wall.

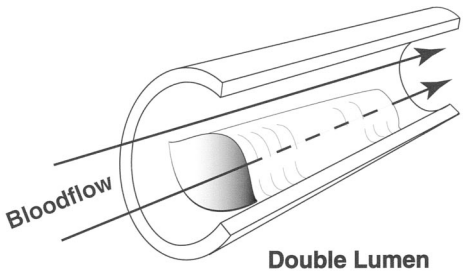

Figure 2.
Double lumen formed by subintimal hemorrhage rupturing back into the arterial lumen distally.

Trivial traumas to the head or neck, such as pushing a car, coughing, vomiting, rhythmic movement of the head and neck to music, chiropractic manipulation, and childbirth, have been considered to be provoking factors in 25% to 41% of patients with ICAD (11,30–32). Some authors consider that any rapid or excessive movement of the head or neck is capable of triggering spontaneous ICAD (2).

Concurrent pathologic conditions frequently exist with ICAD, and many consider them to be predisposing risk factors (Table 1). Hypertension was present in 26% and 36% of patients with ICAD in 2 series, indicating a prevalence higher than in an age-matched population (6,33). Infection in the weeks before ICAD was considered a triggering factor by both Constantinescu (34) and Grau et al (35). Fibromuscular dysplasia, a nonatherosclerotic and noninflammatory vascular disorder affecting primarily the ICA and renal arteries, is present in up to 23% of ICAD patients (36), making it the most frequently reported associated abnormality (23,37–39). Other conditions that have been reported to be related to ICAD include Marfan's syndrome and cystic medial necrosis (6). Several authors have postulated that there is probably a yet to be identified underlying arteriopathy that leads to ICAD (40,41).

Although atheromatous changes are a frequent cause of stroke in young patients (42), such changes are rarely found in the vessels involved in dissection, and when present, the changes are generally mild to moderate (7,43). Barbour et al (44) found significant ICA redundancies, kinks, coils, or loops in 62% of their group of 13 ICAD patients, compared with 19% of 108 arteriograms without dissection. They determined that there was a relationship between ICA redundancy and dissection, particularly if the redundancy occurred bilaterally.

Some authors have speculated that there must be a short-lived underlying arteriopathy present in ICAD because of the fact that multiple arteries are involved in 5% to 28% of patients (6,9,43,45) and recurrence rates are very low 1 month after the dissection (46). Ringel et al (24) described an unusual case of a patient who developed simultaneous acute dissections of both ICAs and both vertebral arteries while skiing, even though there were no significant falls reported. Brandt et al (47) found ultrastructural abnormalities in 68% of 25 cervicocerebral artery dissection patients, resembling Ehlers-Danlos syndrome type II or III. In a study of 26 ICAD cases assessed with ultrasonography, Guillon et al (48) found that there was a relative increase in the diameter of the common carotid compared with matched controls, which they considered might be an indicator of disturbed vessel wall integrity.

The precise pathogenesis of ICAD is not clear in most cases, and a history of a trivial trauma is frequent (40).

Extracranial internal carotid artery dissection is classified as being either spontaneous or traumatic.

Desfontaines and Despland (23) indicated that the cause of ICAD remains speculative, concluding that the cause of ICAD was multifactorial, probably combining an underlying primary arterial disease with repetitive or more violent traumas. Some authors argue against the position that truly spontaneous dissections ever occur and maintain that unrecognized mechanical factors or minor vascular abnormalities must exist (9,49).

Table 1.
Concurrent Pathological Conditions

Condition	Reported Frequency	Significance
Hypertension	26% to 36%	Much more prevalent than in the general population.
Fibromuscular dysplasia	15% to 28%	Most frequent associated abnormality.
ICA redundancies, kinks, coils, or loops	30% to 62%	Compares with 3% to 19% of the general population.
Ehlers-Danlos Syndrome, Marfan's Syndrome, and collagen disorders	Varies	Connective tissue defects seem to be associated with higher incidence.
Infection	Infrequent	A few case reports and reports of higher incidence of ICAD during cold and flu season.

Hart and Easton (41) pointed out that the pathogenetic association of ICAD with inconsequential neck torsion remains a complex and ill-defined issue. They elsewhere stated that if such casual trauma as bowling and coughing is capable of precipitating extracranial carotid dissection, perhaps no dissections are truly spontaneous, and for practical purposes, spontaneous dissection and dissection attributable to trivial trauma may be considered one entity (9). Other authors have indicated that there is a well-known difficulty in determining whether a dissection is spontaneous or secondary to a minor or trivial trauma, because their clinical presentation does not significantly differ (33).

The proposed mechanism of traumatically induced ICAD involves a sudden, severe stretch of the internal carotid artery over the upper cervical spine when the neck is hyperextended and laterally flexed to the opposite side (50) (Figure 3). The above-mentioned neck positioning forces the ICA against the upper cervical vertebrae, allowing it to become fixed in place and thus becoming susceptible to injury (51). The association of hyperextension and rotational forces, rather than lateral flexion, as the causal mechanism has also been proposed (52). The hypothesis of traumatic causation is based on patient histories, as well as pathologic and radiographic observations, but no validating animal or postmortem models exist to substantiate either hypothesis.

The intimal lining, being weaker than the media or adventitia, is more vulnerable to developing tears and is the usual site of injury (50,53). The leading cause of traumatic ICAD is motor vehicle crashes (MVCs), (54) with one study reporting that 72% of their group's traumatically induced ICADs were the result of MVCs (55). Younger patients seem to be more susceptible to ICA injury leading to dissection, because the artery becomes more tortuous in the elderly, which allows the artery to straighten rather than stretch when sudden hyperextension and rotational forces are applied (10).

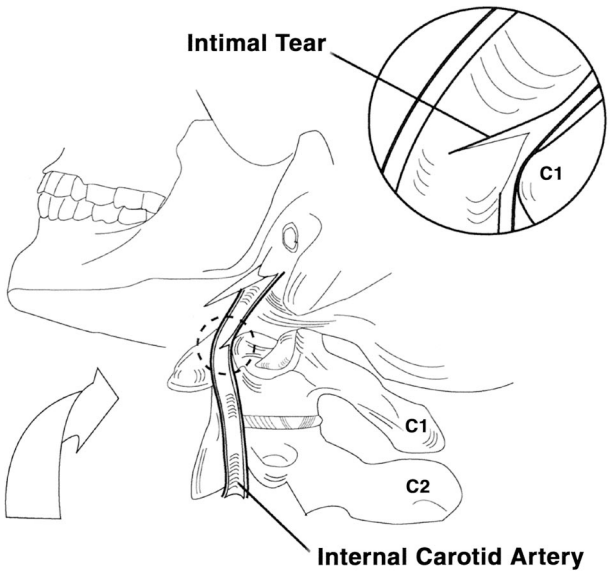

Figure 3.
Proposed mechanism of traumatically induced internal carotid artery dissection, associated with neck hyperextension and lateral flexion to the opposite side.

Epidemiology

Epidemiologic studies have estimated the incidence of spontaneous ICAD as 2.6 and 2.9 cases per 100,000 population yearly, making the condition uncommon but not rare (56,57). Another series of 1200 first strokes found that 2.5% were attributable to spontaneous ICAD (37). The more conservative figure of 2.6 per 100,000 would equate to more than 7000 cases per year in the United States. Only symptomatic cases that resulted in a diagnosis were considered in formulating the above estimates (58). ICAD typically affects relatively young patients under age 50 years, and a few patients are afflicted in childhood or adolescence (40,46,56,59). ICAD is reported to be the cause of 5% to 20% of strokes in those younger than 50 years of age (60). In a group of 135 patients with symptomatic ICAD, it was reported that 49% experienced TIAs or stroke (61). Based on a series of 80 ICAD patients, it was determined that there are no known characteristics of ICAD that would enable clinicians to predict which patients will develop a stroke (33).

In a study comprising 200 cases of cervicocephalic artery dissection reported by Schievink et al (46), 74% were ICAD and 24% were VAD. Other authors have verified that of all the cervical arteries, the ICA is the most common to undergo dissection (6,9,18,31,61–63).

ICAD AND CMT

After a thorough search of the medical literature, we were able to locate 12 cases where the authors reported that ICAD had occurred subsequent to what was referred to as spinal manipulation or chiropractic manipulation.

Beatty's 1977 article is typical of what other reports have conveyed concerning ICAD following CMT. The patient's presenting complaint to the chiropractic physician was neck pain, a well-known early symptom of ICAD. It is possible that the patient was developing the ICAD before CMT, which progressively developed into characteristic ICAD symptoms. However, the patient noticed symptom relief after CMT, which probably points more toward a musculoskeletal condition at that time, because no plausible physiologic explanation can be offered to explain how spinal manipulation might relieve the symptoms of ICAD. It is also possible that the CMT made an early ICAD worse, but Beatty did not mention any other postmanipulation sequelae occurring before the ICAD. As previously mentioned, the study by Beatty was cited considerably more than any other when an author was referring to the relationship of CMT to ICAD.

Chiropractic manipulation was mentioned as being related to one of the ICAD cases in a study by Brandt et al (47); however, in addition to having CMT up to 1 month before

ICAD, the patient was coughing and suffering flu-like symptoms, both regularly mentioned precursors of ICAD. The patient also had a more severe category of connective tissue abnormalities than any of the other 24 patients, which the authors considered to be a possible indication of a primary arteriopathy.

Peters et al (15) described a case of ICAD that followed CMT by 3 hours. The patient had increasing neck pain and 5 episodes of vertigo before the manipulation. She was also a smoker, took oral contraceptives, and had segmental mediolytic arteriopathy with widespread mucoid degeneration. The authors hypothesized that the mediolytic arteriopathy was a predisposing factor for the dissection that followed CMT.

Hufnagel et al (14) reported 10 cases of stroke after CMT, with 2 involving the ICA. However, none of the practitioners were chiropractic physicians. More accurately, they mentioned that the manipulations were performed outside their institution by orthopedists in 7 patients, a physiotherapist in 1, and health practitioners who were not physicians in 2. The outcome of these cases resulted in full recovery in 1 patient, whereas the other maintained a permanent mild neurologic deficit.

Internal carotid artery dissection is reported to be the cause of 5% to 20% of strokes in those younger than 50 years of age.

Murthy and Naidu (64) described an ICA aneurysm purportedly following chiropractic spinal manipulation. However, the practitioner was actually a barber. Furthermore, the origin of the aneurysm was at the origin of the ICA, rather than more distally near C1/C2, where the ICA is reported to be fixed during hyperextension and rotation. Any intimal tear and subsequent aneurysm in relation to blunt trauma would be expected to occur at that level (9). The fact that the patient's symptoms started so soon after the lay manipulation may have been related to a triggering effect acting on an already existing arterial disorder. The patient had a persistent Horner's Syndrome as a result of the aneurysm.

Parenti et al (4) discussed a moderately hypertensive 50-year-old female patient who developed a sudden onset left occipital headache and a posterior circulation stroke within a few minutes after CMT for neck pain. The authors mentioned that MRI disclosed both VA and ICA dissections. However, the VA was intracranial and the ICAD was along its prepetrous tract. The specified location of ICAD described in this case was not in close proximity to the upper cervical vertebrae, which would cause one to question its connection to the manipulation. The ICAD was asymptomatic, and the authors were accordingly of the opinion that this was evidence that its incidence may be underestimated. There is little, if any, evidence to support the notion that intracranial artery dissection can be related to CMT. Indeed, this is the only reported case that we encountered in our review of the literature. There have been reports of intracranial artery dissection after head trauma, but the mechanism of causation by CMT is not present. Pelkonen et al (65) did find one case related to vigorous head movements while dancing, but CMT does not involve similar amplitudes or accelerations of head movement and thus cannot reasonably be compared. The fact that the patient in the study by Parenti et al was diagnosed with multiple dissections also points to an underlying arteriopathy that predisposed her to the condition.

Dragon et al (66) gave an account of an ICAD following manipulation that occurred 2 days after CMT. The patient had severe left neck pain that was ipsilateral to the ICAD and experienced a brief flashing light in the ipsilateral eye, which we inferred was in progress before CMT. The authors did not indicate the type of manipulative practitioner involved. The patient recovered without neurologic deficit. This case of dissection may have already been in process at the time CMT was applied.

Lyness and Wagman (67) described a patient who, within a few moments of being manipulated by an osteopathic physician, became paralyzed in all 4 extremities and was unable to speak or swallow. A subsequent arteriogram disclosed pathology affecting the basilar, right, and left vertebral and right internal carotid arteries. The authors mentioned the presence of a congenital anomaly affecting the vertebral arteries, which might have been a predisposing factor. In addition, multiple artery dissection may indicate the presence of an underlying arteriopathy in this case. The female patient developed lasting locked-in syndrome attributable to damage to the middle and high pontine levels, which was the result of vertebrobasilar compromise. They noted that there were no discernible symptoms related to the ICAD.

Lee et al (1) reported on a survey of California Neurologists concerning strokes and other possible chiropractic-related neurologic problems. They reported that 3 of their stroke cases were caused by CMT-attributed ICAD. However, they admitted that the study had many limitations. Some of these limitations were as follows: they did not in any way verify the results of the survey, the survey's response rate was limited at 36%, only 45% of the cases were verified by angiography, the mechanism was unknown for some of the strokes, and they did not indicate if any of the ICAD cases were verified. Indeed, the study did not reliably represent the relationship of CMT to ICAD; nevertheless, we did count the 3 ICAD cases in the study by Lee et al as part of the twelve that were considered.

Norris et al (2) reported findings of a series of 74 cervical artery dissections that they indicated were related to spinal manipulation in 28% of the cases. They did not provide sufficient data to determine how many, or if in fact any, of

the cases were considered to be ICAD following cervical manipulation. Consequently, we were not able to consider in our tally any of the purported cases of ICAD mentioned by Norris et al.

Taking into account all of the reports of CMT-related ICAD, in 3 instances the practitioner was not a chiropractic physician (in 1 case the manipulator was a barber) (64), and in 3 cases the type of practitioner was not ascertainable. Some regard any form of spinal manipulation as chiropractic manipulation regardless of the licensure or training of the practitioner. The convention of attributing adverse manipulation outcomes to chiropractic practitioners when the procedure was not actually performed by a chiropractic physician has previously been noted by Terrett (68) (Table 2). Errors in attribution may contribute to a distorted perception of the safety for chiropractic spinal manipulative therapy.

CMT has been represented in the literature in the same category as frank trauma, such as MVCs (12,69), indicating a perception that CMT is violent and injurious. This notion is in stark contrast to studies that have looked at the safety of cervical CMT, such as one by Hurwitz et al (70) that estimated the rate of vertebrobasilar accidents or other complication to be 1 per 1,000,000 cervical manipulations.

Only approximately two thirds of the cases of CMT-attributed ICAD, in which the type of practitioner was identified, have been ascribed to chiropractic physicians. Yet they perform 94% of all spinal manipulations (71). This finding indicates the relative safety of chiropractics, especially when compared with medical and lay manipulators who were involved in a substantial number of the reported ICAD cases while only performing approximately 6% of all spinal manipulations.

Dubious implication of trivial trauma related to ICAD was evident in several of these papers, where all unusual neck movements were considered in the month preceding ICAD (11,31,47). A case of VAD described by Mas et al (72) portrayed a young woman who experienced right-sided neck pain, for which she received manipulation and consequently obtained relief. The author went on to state that the patient felt perfectly fine after the manipulation until the dissection occurred. She suffered the dissection 40 days after CMT, yet neck manipulation was identified as a premorbid trauma.

CONCLUSION

As previously mentioned, one can estimate that there are more than 7000 cases of ICAD per year in the United States, and because the primary presentation of a typical ICAD patient is headache and neck pain, it is likely that a proportion of these 7000 patients will consult chiropractic physicians for treatment in the days, weeks, or months before the ultimate ICAD diagnosis. The mere fact that an ICAD develops after CMT does not necessarily create a causal relationship. There may be a causal relationship in cases of head or neck pain, with ensuing ischemic signs that immediately follow or develop within hours after CMT, but because of the widespread uncertainty concerning ICAD etiology (9,23,47), doubt remains.

While discussing cervicocephalic arteries in general, Norris et al (2) remarked that the hallmark of dissection was

Table 2.
Reported Cases of Chiropractic Manipulation Preceding ICAD

	Number of cases	Type of Practitioner	Predisposing Factors	Delay in Onset	Outcome
Beatty	1	DC	None mentioned	5 days	Surgery, full recovery
Brandt	1	Not specified	Severe connective tissue aberrations, coughing	Not specified, up to 4 weeks	Not specified
Peters	1	DC	Widespread arteriopathy, smoker, contraceptives	3 hours	Death
Lee	3	Probably DCs	Not specified	Within 24 hours	Not specified
Hufnagel et al	2	MD, PT, or Nonphysician	One smoker, hypercholesterolemia	Not specified	Full recovery, Mild deficit
Lyness et al	1	DO	Multiple artery dissections, congenitally variant VAs	A few moments	Locked-in syndrome from VAD
Parenti et al	1	DC	Multiple artery dissections, hypertension, VAD	A few minutes	Gradual neurological improvement
Murthy et al	1	Layperson	None mentioned	1 to 2 hours	Mild residual deficit
Dragon et al	1	Not specified	Severe ipsilateral neck pain	2 days	No deficit
Norris et al	Not specified	Not specified	Not specified	Not specified	Not specified

sudden and often severe neck or occipital pain. In cases absent some degree of adverse symptoms in relatively close temporal proximity to CMT, there will always be doubt about a connection with ICAD. Patients who have an associated primary arterial disease, which is often present in ICAD patients (6), are at risk when subjected to virtually any sort of head or neck motion.

Lansley (73) wrote a letter that appeared in *Lancet* in response to a study concerning 4 cases of CMT-attributed VAD. He noted that there could be another explanation of their findings, that the involved patients already had spontaneous dissection, giving rise to symptoms that led them to consult a chiropractor. Mas et al (74) demonstrated pathologically, concerning VAD, that cervical pain that precedes and motivates chiropractic cervical manipulation may be the first symptom of a hitherto unrecognized spontaneous or traumatic dissection.

None of the cases reviewed for the present study suggested a clear causal relationship between CMT and ICAD. There were other contributing factors in most cases, such as connective tissue aberrations, arteriopathy, multiple artery dissections, infection, and bouts of coughing. One third of the cervical manipulations were performed by nonchiropractic practitioners, which cannot realistically be compared with CMT. In some instances there was a lack of a temporal relationship, with the patients experiencing no adverse post-manipulation symptoms that would point to the occurrence of a dissection.

None of the cases reviewed for the present study suggested a clear causal relationship between chiropractic manipulative therapy and internal carotid artery dissection.

Terrett (68) addressed the issue of CMT-related ICAD in his 1996 monograph on the subject of vertebrobasilar stroke. After performing an extensive literature search, he was only able to find four such cases in the English and foreign biomedical literature. Only one of the practitioners involved was a chiropractic physician; the others included a medical practitioner, an osteopath, and a barber. With respect to the relationship between cervical manipulation and the occurrence of carotid artery injury, he noted that "the relationship between manual therapy and the occurrence of reported carotid injury is extremely tenuous at best" (p. 67). Dabbs and Lauretti (75) were unable to locate any literature that provided a plausible anatomic or pathophysiological mechanism for carotid stroke being related to CMT. They concluded that the association between manipulation and carotid stroke was poorly substantiated and speculative.

After investigating the issue of ICAD resulting from CMT, Stapf (5) commented, "A relationship between chiropractic manipulation and carotid dissection is plausible but unproven. Although we have seen cases of carotid dissection in individuals who have undergone cervical manipulation, it is difficult to prove that the manipulation caused the dissection in any individual case" (p. 332).

The relationship between ICAD and CMT remains tenuous, and a causal relationship is not supported by the literature. Furthermore, the notion that trivial trauma may cause ICAD is conjectural and is based entirely on a few case reports (class III evidence) (Table 3). There is no class I or II evidence available, and class III evidence is not sufficient in assigning causation. The existing studies are limited to case reports and small series that, for the most part, suffer from methodological flaws. It is plausible that persons with underlying arteriopathy, or those who are already in the process of dissection before CMT, may have a triggering effect from manipulation that proceeds to ICAD, but similar triggering could just as likely be the result of normal day-to-day movements.

The mechanism of traumatic ICA injury hypothesized by Stringer (50), where the ICA is stretched across the upper cervical vertebrae in response to hyperextension with lateral

Table 3.
Classification of Medical Evidence

Classification	Types of Studies	Quality of Evidence
Class I	Controlled studies published in refereed journals. (ie, randomized clinical trials)	Highest level of evidence, which may be drawn upon to assign cause-and-effect relationships.
Class II	Significant results from uncontrolled studies in refereed journals. (ie, case-control studies)	Less persuasive evidence, which cannot by itself be used to assign cause-and-effect relationships.
Class III	Opinions of experts, anecdote, or by convention. (ie, case reports)	Least persuasive evidence, which cannot be used to assign cause-and-effect relationships.

Table 4.
Risk of CMT-Related ICAD Compared With Other Events

Developing CMT-related ICAD in US/ cervical CMT	1:601,145,000
Death from CMT related ICAD in US/ cervical CMT	0:3,606,870,000
Death by falling aircraft/year	1:10,000,000
Death by lightning strike/year	1:2,000,000
Death by earthquake in California/ year	1:588,000
Death by being struck by an automobile/year	1:20,000
Death related to regular NSAID use/ users	1:4000
Death related to cervical spine surgery/procedures	1:145

Data adapted from References 91, 94, and 95.

flexion, involves severe trauma and should be reserved for such cases. Violent long-lever manipulation, commonly used by untrained manipulators, is not taught in any of chiropractic institutions in the United States. This may be the reason nonchiropractic providers have been responsible for a disproportionate share of reported cases of manipulation following ICAD. CMT is a highly specialized procedure that is the focal point of the educational process of chiropractic physicians.

Some would argue that osteopathic physicians are comparably trained in manipulation, yet, according to the Policy Statement on Spinal Manipulation published by the American Chiropractic Association (76), most osteopathic schools only offer spinal manipulation on an elective basis. This compares with chiropractic education, which devotes approximately 52% of its 4-year education to diagnosis and manipulation.

CMT encompasses short-lever, low-amplitude thrusts that are directed segmentally, and simultaneous neck extension with rotation are minimized, whereas less-formal cervical manipulation may consist of long-lever thrusts, with contact only applied to the patient's head. This positioning may cause strain to the associated cervical tissues. CMT cannot be learned from a book or through weekend courses, which may be the reason the per person incidence of manipulation related injuries is higher outside the chiropractic community.

The issue of ICAD following cervical CMT certainly warrants additional investigation, possibly in an attempt to identify an underlying arteriopathy that is suspected or to better define contributing factors. Because the question of causation is so speculative at this time, researchers, as well as clinicians, should use caution in the assignment of causation in these cases.

The exceedingly low risk of a patient developing ICAD following CMT may be ascertained by calculating the approximate number of cervical manipulations provided by chiropractic physicians in America during the time period covered by the reported cases, then dividing that figure into the number of reported ICADs attributed to CMT that were actually manipulated by chiropractic physicians and occurred in the United States. According to the 2000 American Chiropractic Association Statistical Survey (77), the average chiropractic physician attends to approximately 115 patient visits per week. Cervical manipulation is performed in about 75% of these cases. The yearly total visits for each practitioner can then be projected to be 4300, based on a 50-week year. The population of practicing chiropractic physicians in America was roughly 20,000 in 1977 and 50,000 in 2000, with estimated even incremental growth in the years between. Using these figures, in conjunction with the previously reported incidence of ICAD in America, an approximation of the chance of developing ICAD following CMT would be 0.0000000017 or 1:601,145,000 (Table 4).

This estimate does not take into account unreported cases of CMT-associated ICAD. In fact, the literature may represent only a fraction of the actual number of cases. Therefore, there may be greater risk than discussed herein.

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