



Cervical artery dissection: Initial asymptomatic phase or healing phase? A counter-hypothesis

Steven Brown^{*}

University of Bridgeport Instructor, Health Sciences Postgraduate Department 126 Park Avenue Bridgeport, CT 06604, United States of America

ARTICLE INFO

Keywords:

cervical artery dissection
asymptomatic dissection
healing phase
cervical manipulation
neck pain
stroke risk

ABSTRACT

It has been proposed that many cervical artery dissections remain undiagnosed because of an initial asymptomatic phase. That is, such dissections may occur asymptotically, with no characteristic neck pain and/or headache at the time of onset. We propose an alternative hypothesis: dissections identified without concurrent painful symptoms more likely represent a later healing phase rather than true silence from the beginning.

The onset of acute arterial dissection is typically a markedly painful event, with headache and/or neck pain reported in 55–100% of cases across studies. The intramural hematoma and associated neck pain and/or headache typically resolve as the vessel heals, yet the structural changes (hematoma or thrombus) remain visible on later imaging. This temporal distinction is analogous to a cutaneous laceration, which is intensely painful at injury but painless once the scab forms and healing progresses.

The hypothesis has direct clinical relevance for manual therapists: a thorough history for recent (weeks to months) neck pain and/or headache is essential before cervical manipulation, even when the patient is currently asymptomatic. This perspective reframes incidentally discovered “asymptomatic” cervical artery dissection and complements calls for greater awareness without contradicting the risk of under-recognition.

Introduction

Grond-Ginsbach and colleagues [1] hypothesized that cervical artery dissection (CeAD) frequently goes undiagnosed due to an initial asymptomatic phase. That is, such dissections may occur asymptotically, with no characteristic neck pain and/or headache at the time of onset. They supported this view with data on recurrent dissections, multivessel dissections, and inverse associations between local signs and ischemia in study samples. Population-based studies have indeed documented a substantial proportion of asymptomatic or incidentally discovered dissections [2,3]. Lee et al. (2006) reported that 6% of cases in their Olmsted County cohort were asymptomatic, while Griffin et al. (2024) found that 15.5% of cases were discovered incidentally. While we agree that under-recognition of CeAD is an important clinical issue [4], we suggest a different hypothesis for many of the “asymptomatic” dissections identified on imaging studies. Grond-Ginsbach et al. interpreted these cases as evidence of silent onset at the beginning of the dissection lifespan. We propose instead that they more often represent a later healing phase at the end of the dissection lifespan. This distinction is particularly relevant for chiropractors, physical therapists, and other

manual practitioners, who frequently encounter patients presenting with current or recent neck pain and/or headache — symptoms that may represent an existing undiagnosed dissection.

The hypothesis

Grond-Ginsbach et al. propose that dissections found without concurrent symptoms have been asymptomatic from the time of onset. However, acute arterial dissection is, by all accounts, a markedly painful event [4–7]. While pain is the most frequent presenting feature, painless or minimally symptomatic dissections do occur and are more likely to be identified incidentally or on follow-up imaging. The sudden tear in the vessel wall typically produces intense neck pain and/or headache at the time of occurrence, often prompting visits to the emergency department. Patients frequently describe the headache as severe, unique, and unusual—or even a thunderclap headache [6]. This is significant because a history of migraine headache is a risk factor for dissection [8,9]. That a migraine patient may experience a headache that is worse than their migraines is an indication of how severe the onset of dissection may be [6].

^{*} Corresponding author.

E-mail address: drbrown@brownchiro.com.

<https://doi.org/10.1016/j.mehy.2026.112074>

Received 19 April 2026; Received in revised form 28 June 2026; Accepted 4 July 2026

Available online 4 July 2026

0306-9877/© 2026 Elsevier Ltd. All rights are reserved, including those for text and data mining, AI training, and similar technologies.

Pathophysiologically, the acute phase of dissection involves formation of an intramural hematoma and a thrombus that is firmly adherent to the vessel wall. As the dissection evolves over days to weeks, the hematoma organizes, the thrombus becomes more organized and less adherent, and local inflammatory and pain-generating processes subside. Consequently, what appears as “asymptomatic” on imaging performed sometime after onset is more likely to represent a formerly symptomatic dissection in its healing phase rather than one that was painless from inception.

This situation is analogous to a skin laceration. The initial tear is painful, yet once the scab forms and healing progresses, the lesion remains visible while no longer being symptomatic. In the case of CeAD, the intramural hematoma or thrombus persists on imaging, as demonstrated by population-based studies documenting substantial rates of asymptomatic or incidentally discovered dissections [2,3].

Evolution of the hypothesis

This counter-hypothesis builds directly on the 2013 observations of Grond-Ginsbach et al. [1] while offering a mechanistic reinterpretation of the same imaging findings. It does not dispute the existence of underdiagnosed CeAD; it simply reframes the “asymptomatic” subset as healed and later in the dissection lifespan, rather than of silent onset and early in the dissection lifespan.

Although minor dissections may occur without pain at onset, and painless or minimally symptomatic dissections are documented in the literature, such minor lesions are likely to heal spontaneously and remain clinically insignificant. This is analogous to a minor skin laceration that may go unnoticed at the time of injury and heal with an insignificant scab (illustrative analogy only).

Prospective imaging data reinforce that these clinically silent events are typically minor lesions with low risk of significant complications. In a systematic MRI follow-up study of 36 consecutive patients with spontaneous cervical artery dissection, Dittrich et al. (2007) detected recurrent polyarterial dissections in 9 patients (25%), mostly within the first 1–4 weeks [10]. Strikingly, 8 of the 9 recurrences (89%) remained asymptomatic or produced only local neck pain; only one patient experienced ischemic stroke. All events occurred while patients were already receiving full antithrombotic therapy (intravenous heparin followed by oral anticoagulation with phenprocoumon for at least 6 months), and the authors concluded that “the prognosis of recurring sCAD appears benign, particularly in patients already receiving antithrombotic therapy.”

Grond-Ginsbach et al. cite these recurrent events as key evidence for frequent asymptomatic dissections in the natural history of CeAD. This represents a clear apples-to-oranges comparison. All the new dissections in Dittrich's cohort developed in the highly artificial setting of full therapeutic anticoagulation, intensive inpatient monitoring, and likely concomitant analgesic therapy. Such conditions are fundamentally different from spontaneous dissections that arise *de novo* in untreated individuals in the community. Consequently, the absence of significant pain cannot be generalized to argue for truly painless onset of a clinically significant dissection in the absence of medical intervention and close surveillance. This misinterpretation risks giving clinicians — particularly those performing cervical spine manipulation — a false sense of security that dissections can be entirely silent from onset through the high-risk embolic phase, potentially undermining the importance of obtaining a thorough history of recent neck pain and/or headache [11].

Such subclinical, clustered recurrences are therefore best viewed as minor intramural lesions that heal spontaneously without clinical sequelae — entirely consistent with the healing-phase interpretation proposed here.

This temporal distinction has direct relevance to clinical practice, particularly for chiropractors and other practitioners performing cervical spine manipulation (CSM). Grond-Ginsbach et al. correctly noted that since many CeADs are asymptomatic, manipulative therapy of the

neck bears a certain risk [1]. A thorough history must be taken to identify risk factors for dissection and any episode of neck pain and/or headache consistent with dissection in the preceding weeks to months. Such a history may signal a recent dissection that is now in a painless healing stage. Specifically, the sudden neck movement from CSM could dislodge a loosely adherent thrombus from an asymptomatic healing dissection, leading to immediate thromboembolic stroke [12].

Consequences of the hypothesis

Implications for Incidence of Cervical Artery Dissection.

In 2013, Grond-Ginsbach et al. hypothesized that due to asymptomatic dissections the incidence of CeAD was higher than estimated. They appear to have been correct. At that time, Lee et al. (2006) had estimated an annual incidence rate of 2.6 per 100,000 population [2]. More recently, Griffin et al. (2024) estimated an increased incidence of CeAD at 8.93 per 100,000 per year [3]. The healing-phase explanation suggests that the true incidence of CeAD may be even higher than estimated by Griffin et al. (2024). Symptomatic dissections may heal, become painless, and therefore never prompt any imaging studies.

Implications for manipulative therapy

Recognition of an asymptomatic healing phase better explains the occurrence of ischemic stroke immediately with CSM in a patient with no current neck pain and/or headache consistent with dissection. Dissection thrombi become more loosely adherent later in the healing process, during the asymptomatic healing stage. It is when a loosely adherent thrombus is present that it could plausibly be embolized by neck manipulation (a hypothetical mechanism) [12]. In earlier stages of dissection, thrombi are more firmly adhered to the artery wall as part of the natural healing process. This is again analogous to the firm adherence of a scab on the skin recently after a cutaneous laceration. It is later in the healing process that such thrombi or scabs become loosely adherent enough to be dislodged.

Therefore, the timeframe within the lifespan of a dissection during which immediate post-manipulative thromboembolic stroke is most likely to occur is the same timeframe in which the dissection may be asymptomatic. This realization has important implications for risk-benefit assessment in manual therapy. Current pre-manipulative screening frameworks for manual therapy practitioners [11] rely heavily on identifying active symptoms; our hypothesis suggests that clinicians must also probe for resolved symptoms in the preceding weeks to months.

In many manual therapy settings, high-volume practice models frequently limit consultation time, which can restrict opportunities for comprehensive history-taking and examination. This constraint may result in less attention to recent or resolved symptoms of cervical artery dissection, thereby increasing the risk of performing cervical manipulation in the presence of a healing dissection and causing thromboembolic stroke. Educational programs on CeAD for manual therapy practitioners should emphasize both the classic painful presentation and the possibility of a painless healing stage [12].

Implications for migraine patients

There is an association between migraine headaches and CeAD [8,9]. Although CSM is not likely to resolve migraines, many migraine patients seek CSM for symptom relief. Such patients, and their manual therapists, may not be able to distinguish the severe headache associated with dissection from their migraines. An episode presumed to be a resolved migraine may represent a painful onset of dissection which progressed to an asymptomatic healing state. CSM would be absolutely contraindicated in such a case.

Based on our hypothesis, a history of migraine headaches (particularly when associated with recent severe or unusual episodes) may

warrant consideration as a relative contraindication to CSM, pending further prospective investigation given the high prevalence of migraine in the general population. Practice guidelines frequently recognize connective tissue disorders such as Ehlers-Danlos syndrome, Marfan syndrome and osteogenesis imperfecta as predisposing to weakness of the arterial wall [11]. However, clinical signs of such connective tissue disease are absent in the majority of CeAD patients [13]. No current practice guidelines recognize migraine as a contraindication to CSM. More research in this area is indicated [14,15].

Limitations of the hypothesis

Our hypothesis specifically concerns dissections severe enough to form an intramural thrombus capable of embolization to the brain—the cases most relevant to clinical practice and the risk of stroke. We acknowledge that the proposed temporal progression from symptomatic onset to painless healing phase remains a hypothesis and would benefit from dedicated longitudinal imaging studies with serial clinical correlation in individual patients.

Summary

Dissections identified without concurrent symptoms are more plausibly in the healing phase than asymptomatic from onset. This counter-hypothesis therefore refines rather than contradicts the original observations of Grond-Ginsbach et al. (2013) and provides a clear, actionable clinical message: a thorough recent-history screen for neck pain and/or headache consistent with cervical artery dissection is essential prior to cervical manipulative therapy, even in currently asymptomatic patients.

Declaration of generative AI and AI-assisted technologies

During the preparation of this work the author used Grok, an AI language model developed by xAI, to assist with drafting, editing, refining language and readability, improving structure and flow, and generating suggested highlights. After using this tool/service, the author reviewed and edited the content as needed and takes full responsibility for the content of the published article.

CRedit authorship contribution statement

Steven Brown: Writing – review & editing, Writing – original draft, Conceptualization.

Ethics statement

This manuscript is a hypothesis-based work and does not include any experiments involving human participants, animals, or identifiable human data. Hence, ethical approval and informed consent were not applicable.

Funding

This work was not funded by grants from any university, company, nonprofit, organization, or agency.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Steven Brown, DC, DIAMA reports a relationship with BCA Professional Services, LLC that includes: consulting or advisory, employment, paid expert testimony, speaking and lecture fees, and travel reimbursement. Steven Brown, DC, DIAMA reports a relationship with American Specialty Health Incorporated that includes: consulting or advisory, speaking and lecture fees, and travel reimbursement. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

- [1] Grond-Ginsbach C, Metso TM, Metso AJ, Pezzini A, Tatlisumak T, Hakimi M, et al. Cervical artery dissection goes frequently undiagnosed. *Med Hypotheses* 2013;80: 787–90. <https://doi.org/10.1016/j.mehy.2013.03.012>.
- [2] Lee VH, Brown RD, Mandrekar JN, Mokri B. Incidence and outcome of cervical artery dissection: a population-based study. *Neurology* 2006;67:1809–12. <https://doi.org/10.1212/01.wnl.0000244486.30455.71>.
- [3] Griffin KJ, Harmsen WS, Mandrekar J, Brown RD, Keser Z. Epidemiology of spontaneous cervical artery dissection: population-based study. *Stroke* 2024;55: 670–7. <https://doi.org/10.1161/STROKEAHA.123.043647>.
- [4] Debette S, Leys D. Cervical-artery dissections: predisposing factors, diagnosis, and outcome. *Lancet Neurol* 2009;8:668–78. [https://doi.org/10.1016/S1474-4422\(09\)70084-5](https://doi.org/10.1016/S1474-4422(09)70084-5).
- [5] Trager RJ, Troutner AM, Pikus HJ, Daniels CJ, Dusek JA. Symptoms of patients with vertebral artery dissection presenting to chiropractors: a systematic review and Meta-analysis. *Cureus* 2023. <https://doi.org/10.7759/cureus.51297>.
- [6] 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; 77:1021–4. <https://doi.org/10.1136/jnnp.2006.094359>.
- [7] Gottesman RF, Sharma P, Robinson KA, Arnan M, Tsui M, Ladha K, et al. Clinical characteristics of symptomatic vertebral artery dissection: a systematic review. *Neurologist* 2012;18:245–54. <https://doi.org/10.1097/NRL.0b013e31826754e1>.
- [8] Daghas I, Sargurupremraj M, Danning R, Gormley P, Malik R, Amouyel P, et al. Migraine, stroke, and cervical arterial dissection: shared genetics for a triad of brain disorders with vascular involvement. *Neurol Genet* 2022;8:e653. <https://doi.org/10.1212/NXG.0000000000000653>.
- [9] Trager RJ, Cupler ZA, Williamson TJ, Chu EC-P. Association between migraine with and without Aura and Cervical artery dissection. *Cureus* 2025. <https://doi.org/10.7759/cureus.87883>.
- [10] Dittrich R, Nassenstein I, Bachmann R, Maintz D, Nabavi DG, Heindel W, et al. Polyarterial clustered recurrence of cervical artery dissection seems to be the rule. *Neurology* 2007;69:180–6. <https://doi.org/10.1212/01.wnl.0000265595.50915.1e>.
- [11] Chaibi A, Russell MB. A risk-benefit assessment strategy to exclude cervical artery dissection in spinal manual-therapy: a comprehensive review. *Ann Med* 2019;51: 118–27. <https://doi.org/10.1080/07853890.2019.1590627>.
- [12] Brown SP. Plausible mechanisms of causation of immediate stroke by cervical spine manipulation: a narrative review. *Cureus J Med Sci* 2024;16:e56565. <https://doi.org/10.7759/cureus.56565>.
- [13] Grond-Ginsbach C, Debette S. The association of connective tissue disorders with cervical artery dissections. *Curr Mol Med* 2009;9:210–4. <https://doi.org/10.2174/156652409787581547>.
- [14] Romozzi M, Garignano G, Gentile A, Vollono C. Status migrainosus as the only manifestation of vertebral artery dissection due to osteopathic neck manipulation. *Neurol Sci* 2025;46:4057–9. <https://doi.org/10.1007/s10072-025-08180-0>.
- [15] Brown S. Selective criticism confounds objective evaluation of case report on spinal manipulation and vertebral artery dissection. *Neurol Sci* 2025. <https://doi.org/10.1007/s10072-025-08524-w>.