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Cervical Arterial Dysfunction: Knowledge and Reasoning for Manual Physical Therapists

Adverse events associated with manual-therapy interventions have been the concern of clinicians for many decades. Arguably, the most reported and discussed adverse event is that related to cerebrovascular accident as a result of vertebrobasilar insufficiency (VBI). VBI is a specific and rare occurrence occasionally attributed to certain manual therapy techniques of the cervical spine. Guidelines for practice have been available for the last 2 decades to assist clinical decision making with regard to VBI and manipulation techniques.[32,53] This paper takes the perspective that VBI is only 1 example of a number of pathologies affecting the arterial structures of the cervical region. Pathologies may range from transient mechanical occlusions, intimal tearing (dissections), to frank atherosclerotic thromboembolic events leading to a stroke. Such pathologies are evident in both the posterior arterial system perfusing the hind-brain (vertebrobasilar), and the anterior arterial system supplying blood to the cerebral hemispheres and eye (internal carotid arteries). As such, the term *cervical arterial dysfunction* (CAD) is used to represent the anatomical and pathological spectrum of events.

Furthermore, this paper proposes that the issues around this clinical topic are wider than simply considering the chance of serious event following manipulative thrust. Restricting focus to this single clinical question, "Will there be a neurovascular event following the manipulation?" prevents proper evidence-informed analysis of the patient presentation. We approach the topic within a different paradigm: the consideration of clinical and experimental literature related to cervical arterial disease (pathology and presentation), and how this knowledge can be utilized in manual-therapy clinical decision making. A consistent theme within this literature is how both vertebrobasilar and internal carotid dysfunction may, at an early stage, present with very few signs and symptoms other than head and neck pain. Thus, an interesting clinical challenge is offered to the manual therapist—vascular differential diagnosis of head and neck pain.

The main focus of this paper therefore concerns what information can be gathered by the clinician to determine a potential vascular cause of head and neck pain. It is also proposed that the presence of signs and symptoms indicative of vascular disease need not necessarily be a contraindication to treatment. A reasoned risk-benefit analysis needs to be undertaken for the best clinical decision to be made. These issues are discussed within the context of 2 case reports.

The first case scenario represents misdiagnosis of a serious vascular trauma.

• **SYNOPSIS:** This clinical commentary provides evidence-based information regarding adverse cerebrovascular events in the context of manual therapy assessment and management of the cervical spine. Its aim is to facilitate clinical decision making during diagnosis and treatment of patients presenting to the therapist with cervicocranial pain. Rather than focusing on a traditional view of premanipulative testing as the cornerstone for decision making, we present information concerning the clinical presentation of specific vascular conditions. Additionally, we discuss the assessment and management of musculoskeletal pain in the presence of risk factors for cerebrovascular accident. It is proposed that vascular "red flag" presentations mimic neuromusculoskeletal cervicocranial syndromes. Invariably, the 2 conditions coexist. This reasoning presupposes that some patients who have poor clinical outcomes, or a serious adverse response to treatment, may be those who actually present with undiagnosed vascular pathology. We use 2 case reports to demonstrate how incorporating vascular knowledge into clinical reasoning processes may influence clinical decision making.

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The second case focuses on the integration of vascular pathology knowledge in the decision-making process for the manual therapy assessment and management of a patient with significant risk factors for stroke. It should be noted that the first case report is not an exemplar of standard of care. Rather, this case is used as a vehicle to highlight classically underconsidered areas within manual therapy. Conversely, the second case report represents a good example of how the clinician was able to develop an evidence-informed risk-benefit analysis. This second case is used to demonstrate how information regarding risk needs to be contextualized by the potential benefits of an intervention for the best clinical decision to be made. The 2 case reports are included in this paper to present relevant information regarding CAD in a meaningful manner. It is hoped that these cases can be used as learning tools and narratives that will benefit future clinical experiences.

It must be considered that the area of CAD (including VBI) beholds a paucity of absolutes in terms of guidelines. Each clinical decision should embrace this uncertainty and the clinician should aim to make the *best* decision, based on a balance of probabilities, rather than aiming to make the "right" decision. With this in mind, it must be remembered that although the exact prevalence of these cases (ie, patients presenting to manual therapists with CAD) is unknown, it is likely to be very low. As such, initially, the chance of a patient's head and neck pain being caused by frank arterial dysfunction is very low. This low probability may or may not be affected as a result of unfolding information derived from the history intake and physical examination.

CASE REPORT 1

This case presents an unfortunate episode of misdiagnosis. Superficial analysis of the clinical presentation may suggest a benign musculoskeletal dysfunction. However, by considering knowledge of vascular pathology, it may have been possible for the therapist to recognize an alternative cause of signs and symptoms. The case is described below, and discussion on pathology and presentation of the vertebrobasilar system follows.

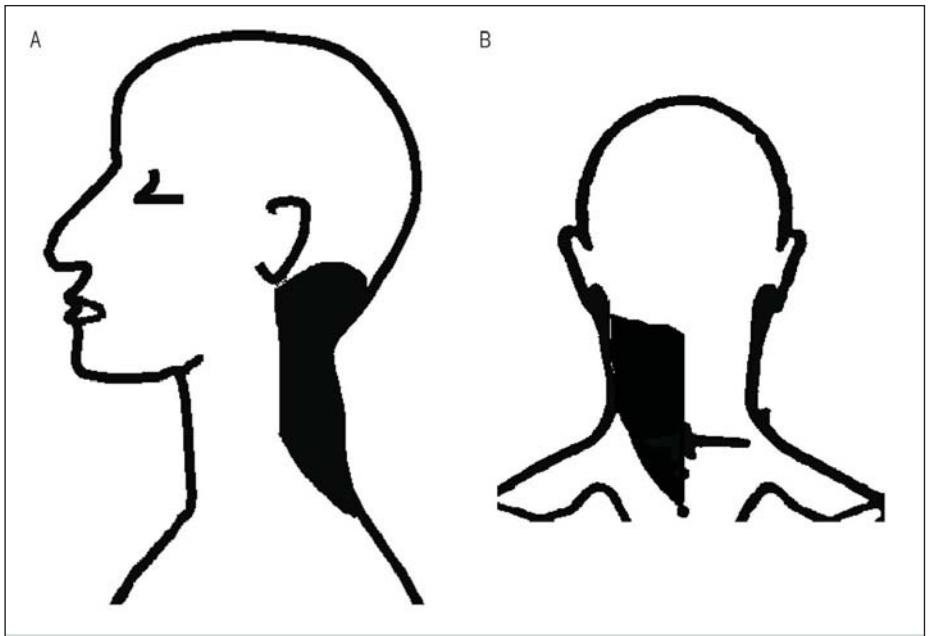

FIGURE 1. Typical pain distribution relating to extracranial vertebral artery dissection: ipsilateral posterior upper cervical pain and occipital headache. Reprinted from *Manual Therapy*, 11(4), Kerry and Taylor, Cervical arterial dysfunction assessment and manual therapy, 243-253. Copyright 2006, with permission from Elsevier.

A 40-year-old female clothing factory worker presented to Physiotherapy with left-sided head and neck pain (**FIGURE 1**). She reported a 3-day history of the symptoms, which began when she quickly rotated her neck to the right at work. The symptoms progressively worsened. The pain was described as a dull constant ache, rising to a sharp pain of 7/10 severity on a numerical rating scale with neck rotation to the right. Cervical spine extension worsened the symptoms mildly. The pain was eased by rest and Paracetemol. The patient reported 2 road traffic accidents in the last 18 months, resulting in prolonged periods of head and neck pain, successfully managed by Physiotherapy with complete resolution of symptoms. The present pain was described as "unlike previous episodes." Past medical history included periods of stress resulting in time off work and high blood pressure. She reported no classic signs and symptoms of either upper cervical segmental instability or VBI (eg, dizziness, diplopia, drop attacks, dysarthria, dysphagia, facial numbness, nystagmus, nausea). Her present use of medication included Paracetemol, as needed, and statins.

Physical examination revealed no neurological deficit; however, mechanical movement dysfunction involving the upper- and mid-left cervical spine was noted. Formal VBI testing (functional positional testing)[53] was not performed, as the clinician did not intend to perform manipulative techniques, nor had the patient reported any subjective indicators for VBI. Over 6 days the patient was treated on 3 occasions, with a combination of passive mobilization techniques, electrotherapy modalities, and exercise prescription (no end-of-physiological-range manual-therapy techniques or exercises were performed).

At the start of the last therapy session, the patient reported no improvement in original symptoms and an onset of new symptoms, including "feels like might be sick," "throaty," "feels faint," especially after performing prescribed exercises. Prior to treatment on this final session, a VBI positional test was

performed (10-second end-range positioning in left and right rotation), meeting the minimum requirements of the Australian Physiotherapy Association Guidelines,[53] which did not cause any symptoms. Therapeutic ultrasound therapy was provided, together with a review of home exercises. The patient failed to attend her next appointment and was discharged after a further 4 weeks of no contact with the Department. Upon longer-term follow-up inquiry it was revealed that the day following her last therapy session she had suffered an acute stroke as a result of left cerebella infarction related to a left vertebral artery dissection between the C1 and C2 vertebral levels (V2-V3 portion of the vertebral artery).

Discussion of First Case Report

This case highlights the misdiagnosis and subsequent mismanagement of a patient who most likely presented with a vascular pathology at initial consultation. It is argued herewith that, with the consideration of vascular anatomy and pathology, a more detailed examination would have resulted in more accurate clinical decisions. It is unlikely that a definitive diagnosis could have been made at initial assessment. However, the lack of response to treatment, contextualized by cardiovascular background and presentation of the patient, could have alerted a re-examination and possible referral to a specialist (neurologist or vascular specialist) for further investigation. We also suggest that the clinical issue is one of misdiagnosis and not causation (the stroke was not caused by physiotherapy interventions).

Pattern Recognition and Symptomatology

In the early stages, vertebral artery pathologies commonly present as neuromusculoskeletal symptoms, often isolated neck and head pain without commonly described VBI symptoms.[3,40,63] Upper cervical pain accompanied with occipital headache has been reported as a common initial presentation of structural trauma to the vertebral arteries.[3,5,11,59,65] On rare occasions, cervical nerve root impairment (usually C2 to C6) can be present as a result of local neural ischemia.[14] Migraine-type headache has been reported as a strong predictor of vertebral artery injury.[4,58] It is also known that cervical spine rotation stresses the contralateral vertebral artery, and this is a potential mechanism of vertebral artery injury.[2,20,31,38,39,47,49,51,56] While this presentation and mechanism of injury is a common manifestation of injury to numerous other cervical structures, given the above knowledge, and the awareness of the serious potential sequelae of vascular trauma, it is reasonable to suspect such a presentation as a vascular injury. The initial presentation in this case is representative of what is known about vertebral artery injury.

As stated above, the initial presentation of vertebral artery injury is commonly head and neck pain. This phase of injury is described as a nonischemic phase (ie, local, somatic responses[3]) and may last from a few minutes to a few weeks. If the injurious process were to continue, the pathology would develop into an ischemic phase. Classically, the signs and symptoms related to hindbrain ischemia are considered as the "five Ds" (dizziness, diplopia, dysarthria, dysphagia, and drop attacks) and "three Ns" (nausea, numbness, nystagmus) of Coman.[12] Unreasoned adherence to these cardinal "classic" signs and symptoms can, however, be misleading and result in an incomplete understanding of patient presentations. More detailed evidence from medical literature shows that the typical presentation of vertebrobasilar dysfunction is not always in line with this classical picture. Although these classic indicators may be present, there are further signs and symptoms to consider (**TABLE 1**).[3,50,59]

It is rare for CAD to manifest in only 1 sign or symptom, and isolated dizziness or transient loss of consciousness are often misattributed to posterior circulation ischemia.[59] Dizziness is often reported as being one of the most common symptoms of VBI.[13] However, there have been cases reported when dizziness was not present. The nature of dizziness can be a differentiating factor in establishing a vascular versus nonvascular cause. Typically, dizziness secondary to posterior circulation dysfunction does not present as frank vertigo, although some authors have suggested this could occur.[59] Vascular dizziness occurs as a result of neck rotation, and does not improve with repeated movement. This pattern differs from nonvascular vestibular dizziness, which often has a short latency and can improve with repeated movement. Nine days posttrauma, this patient presented with new symptoms indicative of hindbrain ischemia.

TABLE 1 Presentations of Vertebral Artery Dissection*

Nonischemic (Local) Signs and Symptoms
• Ipsilateral posterior neck pain/occipital headache
• C2-C6 cervical root impairment (rare)
Ischemic Signs/Symptoms
• Hind-brain TIE (dizziness, diplopia, dysarthria, dysphagia, drop attacks, nausea, nystagmus, facial numbness, ataxia, vomiting, hoarseness, loss of short-term memory, vagueness, hypotonia/limb weakness (arm or leg), anhidrosis (lack of facial sweating, hearing disturbances, malaise, perioral dysthesia, photophobia, papillary changes, clumsiness and agitation)
• Hind-brain stroke (eg, Wallenberg's syndrome, locked-in syndrome)

Abbreviation: TIE, transient ischemic event.
** Nonischemic symptoms can precede ischemic events by a few days to several weeks. Reprinted from* Manual Therapy, *11(4), Kerry and Taylor, Cervical arterial dysfunction assessment and manual therapy, 243-253. Copyright 2006, with permission from Elsevier.*

Predisposing Factors and Physical Examination

The authors of a small number of manual therapy reports have suggested that vertebral artery injury related to manual therapy is a random, unpredictable event with no predictive indicators.[18,21] It should be noted that "manual therapy" is very rarely defined in many papers and may refer to any technique from soft tissue massage to high-velocity thrusts. However, other authors suggest that prediction of cerebrovascular events can be made. Specifically for manual therapy-related adverse events, several predictors have been reported, including aortic root diameter, migraine, relative common carotid artery diameter change during the cardiac cycle, and trivial trauma (ie, associated with cervical spine manual therapy), plasma homocysteine, and recent infections.[58] Vertebral artery accidents have been reported to be 5 times more likely to occur if a patient received manual therapy within 1 week of the stroke, and individuals with a vertebral artery related stroke are 5 times more likely to have had received manual therapy than the control group.[57] Other reports have concluded that arterial dissection is 5 times more likely to occur if manual therapy had been administered within 30 days prior to, and twice as likely to have had increased neck/head pain preceding, the stroke.[60] These studies concluded that vertebral artery dissection was independently associated with stroke and increased neck/head pain.

Manual therapy specific studies are, however, scarce, have small subject numbers, and possess numerous limitations that make forming clear judgments about predisposing factors and predictors of adverse events difficult and inconclusive.[8] A wider, more holistic review of the literature helps to facilitate knowledge and understanding of CAD. Trauma to cervical blood vessels is generally classified as either dissection (intimal tearing), resulting from direct trauma to the vessel, or localized thrombogenesis and embolus formation in response to endothelial damage.[9] Either pathological state may lead to stroke.[22,44,55] Arterial dissection may occur after trivial trauma to the vessel or spontaneously. This may be related to pre-existing congenital weakness of the vessel wall or acquired vascular pathology (atherosclerosis). Atherosclerosis is considered to be a fundamental predisposition for cervicogenic stroke.[8,27,28,36,37] Atherosclerosis is an inflammatory process associated with a number of factors, including hypertension, hypercholesterolemia, hyperlipidemia, hyperhomocysteinemia, diabetes mellitus, genetic clotting disorders, infections, smoking, free radicals, direct vessel trauma, and iatrogenic causes (surgery, medical interventions).[22,23,36,37,55]

It is important for the clinician to appreciate that hypertension is positively correlated to disease and dysfunction of the cervical arteries.[16,24,34,43,62] Consequently, this may indicate that recognition of hypertension by the clinician could be important when assessing the likelihood of potential CAD. The history of the patient in this case report indicated several risk factors for vascular disease: a state of hypertension (subjectively reported), hypercholesterolemia (she reported that she was taking statins in her drug history), possible trauma to vessel wall (mechanism of injury being 2 road traffic accidents), and headache. Further questioning could have been directed towards gaining information about other known predisposing factors for vascular disease.

TABLE 2 Diagnostic Utility of the Vertebrobasilar Insufficiency (VBI) Test*

Author	Sensitivity	Specificity	LR+	LR–
Cote et al 1996	0.00	0.86	0.00	1.16
Rivett et al 2000	0.10	0.39	0.16	2.30
Kerry et al 2003	0.31	0.48	0.59	1.44
Kerry 2006	0.10	0.44	0.16	2.30

** LR+ is the likelihood ratio for a positive test. LR– is the likelihood ratio for a negative test. The further away from 1 (on a scale of 0.001 to 1000) the LR is (LR+, above 1; LR–, below 1), the better the test at ruling the condition in or out. Above 10 would be considered a good LR+, and below 0.01 would be considered a good LR–. All readings from the studies in the table would indicate poor and inconsistent findings for the diagnostic utility of the VBI test.*

Functional positional tests of the cervical spine are commonly used to identify the presence of VBI.[19,53] The purpose of establishing whether or not a patient has VBI is obviously of great importance to health professionals from whom a patient has sought help for cervical pain. The underlying mechanical principle of these tests has been the subject of a number of research reports. Many blood flow studies have demonstrated a reduction in blood flow in the contralateral vertebral artery during rotation.[2,20,31,39,38,47,48,51,56,67,68] Most of this work has been undertaken on asymptomatic subjects. Some authors have used these studies to support the validity of screening tests. In other words, these studies demonstrate that rotation changes blood flow; therefore, the test is valid. The tests may be valid in that they may alter blood flow, but there is little consistent evidence relating these changes to alterations in symptoms (eg, a patient could have significant reduction in blood flow but no VBI symptoms, and vice versa). This makes the specificity and sensitivity of these tests poor and variable, affecting their diagnostic utility and, therefore, clinical usefulness (**TABLE 2**).[13,25,26,52]

In this case report, even though the patient was reporting signs of possible hind-brain ischemia (9 days posttrauma), the response to functional positioning testing was still negative. Clinically, a negative response does not rule out the presence of the pathology.

Based on available information, it would be justifiable to reconsider the tra-

ditional approach to physical examination for vertebral artery pathology. Based on the atherosclerotic theory, measurement of blood pressure in the clinic is a clear way to gather useful information about the possible hypertensive state of the patient. Blood pressure testing can be used as either a measure of regular state of hypertension for a patient or as an indicator for vascular trauma, which may result in acute alterations in pressure.[20,33,41]

It is important to appreciate that, although hypertension is undoubtedly a strong predictor of cardiovascular disease, interpretation of readings must be in context of other findings and sound logical reasoning. Vascular disease is an interplay between various factors, of which high blood pressure is just one (albeit a consistently important one). Blood pressure is a graduated, continuous measure and, as such, cannot have a threshold.[41] The clinician should bear these points in mind during clinical decision making. It is, therefore, unreasonable to state that all patients with hypertension and neck pain should be referred to a medical specialist. Hypertension and neck pain are only 2 of the many factors that influence the decision on probability of vascular pathology. The concept and terminology of hypertension are contentious.[33] But, as a general guide, hypertension is usually indicated by a systolic blood pressure greater than 140 mmHg and a diastolic blood pressure greater than 90 mmHg.[17] Data regarding scaled risk is equally as clinically useful. There is a positive correlation between increased systolic and diastolic pressure and risk of stroke (the higher the pressure, the greater the risk).[1] This would mean that a patient with blood pressure of say 190/100 mmHg is at greater risk than a patient with that of 160/95 mmHg. Thus, the risk is different, even though they are both hypertensive. However, to reiterate, the actual utility of these data in isolation is limited, as the true clinical risk is dependent on additional coexisting factors.

Many of the symptoms related to hindbrain ischemia are related to dysfunction of cranial nerves, whose origins are within the territory of the vertebrobasilar system (except cranial nerves I and II). Being sensitive to changes in blood supply, it is likely that the cranial nerve dysfunction will manifest early on in the ischemic phase. Cranial nerve testing is, therefore, proposed as a firm part of the clinical examination for vertebral artery dysfunction.

In summary, there were indicators in this patient's history and background to warrant the inclusion of a vascular hypothesis in the diagnostic reasoning process. Standard of care in this instance would have included the focused testing of such a hypothesis. This should include further questioning (cardiovascular profiling) and understanding the clinical presentation in the context of vascular pathology. Additionally, informed interpretation of a small number of clinical tests would help in forming a judgment on the chance of vascular pathology being present. To reiterate a critical point, an absolute diagnosis cannot be made by the physiotherapist—a clinical decision based on the balance of probabilities would be the aim of this standard of care.

CASE REPORT 2

This case represents a good reasoning process whereby a clinical decision is reached to continue with treatment in the presence of high cardiovascular risk factors. The point of this case is to highlight that the potential risks of interventions should be judged in the context of potential benefits. Careful examination and consideration of risk factors are, of course, essential. Equally necessary is the consideration of the potential negative impact on quality of life of withholding treatment.

A 79-year-old female presented for consultation with a 20-year history of left arm pain resulting from surgery for removal of a malignant histiocytoma. She reported a 7-year history of neck pain, which she believed was related to the arm pain. For 6 years she had received intermittent episodes of cervical manual therapy when her pain worsened. These episodes of therapy would dramatically reduce her severe neck and arm symptoms. A local change in resource policy had resulted in this treatment option stopping. Because she had stopped receiving treatment, the patient reported a severe and disabling increase in symptoms. She now described a very poor quality of life, including inability to sleep and numerous side effects from ineffective medications prescribed to relieve her pain. The patient was referred for third-party specialist consultation as part of an appeal by the patient and her family to have the manual therapy treatment regime reinstated. Upon consultation, the patient revealed that she had 2 episodes of cerebrovascular accident (stroke) 8 and 9 years ago. Vascular examination at the time of the last stroke demonstrated 70% occlusive disease of her left internal carotid artery (abnormal). On examination, her blood pressure was 186/75 (hypertensive), although the patient reported significantly lower home readings (around 126/76 [normotensive]). Doppler examination revealed triphasic flow (normal) in both the left and right internal carotid arteries, and the left and right vertebral arteries. The flow in the left internal carotid artery was significantly more turbulent (indicative of abnormal flow which would relate to the known occlusive disorder). Flow in all 4 vessels was unaffected by cervical extension or rotation, although the amount of range of motion available was markedly limited. There were no positive findings on neurological testing (cranial or peripheral). Despite the apparent significant cerebrovascular risk factors, a decision to reinstate manual therapy was made. The reasoning for this decision is discussed below.

Discussion of Second Case Report

This case represents a challenging clinical decision-making process. The patient may gain significant benefit from the re-

TABLE 3 Clinical Features of Internal Carotid Artery Dissection*

Nonischemic (local) signs/symptoms
• Horner's syndrome
• Pulsatile tinnitus
• Cranial nerve (CN) palsies (most commonly CN IX to XII)
Less common local signs and symptoms include:
• Ipsilateral carotid bruit
• Scalp tenderness
• Neck swelling
• CN VI palsy
• Orbital pain
• Anhidrosis (facial dryness)
Ischemic (cerebral or retinal) signs/symptoms
• Transient ischemic attack (TIA)
• Ischemic stroke (usually middle cerebral artery territory)
• Retinal infarction
• Amaurosis fugax

** Nonischemic signs and symptoms may precede cerebral/retinal ischemia by anything from a few days to over a month. Reprinted from Manual Therapy, 11(4), Kerry and Taylor, Cervical arterial dysfunction assessment and manual therapy, 243-253. Copyright 2006, with permission from Elsevier*

FIGURE 2. Typical pain distribution relating to dissection of internal carotid artery: ipsilateral front-temporal headache, and uppercervical/midcervical pain. Reprinted from *Manual Therapy*, 11(4), Kerry and Taylor, Cervical arterial dysfunction assessment and manual therapy, 243-253. Copyright 2006, with permission from Elsevier.

instatement of manual therapy intervention. However, she has a strong history of cervical vascular disease and has demonstrable internal carotid artery disease. Superficially, she presents as a high risk for cerebrovascular accident. The clinical reasoning challenge is one of risk-benefit analysis. The fact that she has received previous treatment with no adverse effect might be an initial line of support for continuing manual therapy. However, this inductive inference is by no means a secure argument for ensuring that no adverse events will occur in the future. Detailed analysis and examination, utilizing evidence and information, is needed to provide an optimum reasoned basis for the best decision.

Pattern Recognition and Symptomatology

With a history of stroke, an initial question is whether or not the patient presents at this point with signs and symptoms of stroke, or impending stroke. The internal carotid artery supplies the brain and the retina. The natural onset and progress of internal carotid artery dissection begins with local arterial trauma, which, like vertebral artery trauma discussed above, can manifest as nonischemic signs and symptoms (somatic pain related to local injury). As in vertebral artery pathology, these local signs and symptoms can precede cerebral ischemia (transient ischemic attack, stroke, or retinal ischemia) by anything from less than a week to beyond 30 days.[7,69] There is, therefore, a period when a patient with internal carotid artery dissection may present to the manual therapist with signs and symptoms that may mimic a neuromusculoskeletal presentation.[63] **TABLE 3** shows the classic nonischemic and ischemic manifestations of internal carotid artery dissection.

Thus, in the early stages of the pathology, headache and/or cervical pain can be the sole presentations of internal carotid artery dysfunction.[42,54,63] **FIGURE 2** shows a typical pain distribution associated with dissection of the internal carotid artery. The frontotemporal headaches are often described as cluster-like, thunder-clap, migraine without aura, hemicrania continua, or simply "different from previous headaches." The upper cervical or anterolateral neck pain, facial pain, and/or facial sensitivity are described as carotidynia.

Cranial nerve palsies and Horner's syndrome are often pathognomonic of internal carotid artery pathology, especially if the onset is acute. The hypoglossal nerve (CN XII) is the most commonly affected followed by the glossopharangeal (CN IX), vagus (CN X), or accessory (CN XI) nerves.[3,69] However, all cranial nerves (except the olfactory nerve) can be affected.[69] If the dissection extends into the cavernous sinus, the oculomotor (CN III), trochlear (CN IV), or abducens (CN VI) nerves can be affected.[29,69] The 2 most likely mechanisms for these cranial nerve palsies are (1) ischemia to the nerve via the vasa nevorum (comparable to peripheral neurodynamic theory) and (2) direct compression of the nerve axon by the enlarged vessel.[3,29,69]

Horner's syndrome has been found to be present in up to 82% of patients with known internal carotid artery dissection.[10] Most commonly, this syndrome occurs with head, neck, or facial pain. Carotid induced Horner's syndrome

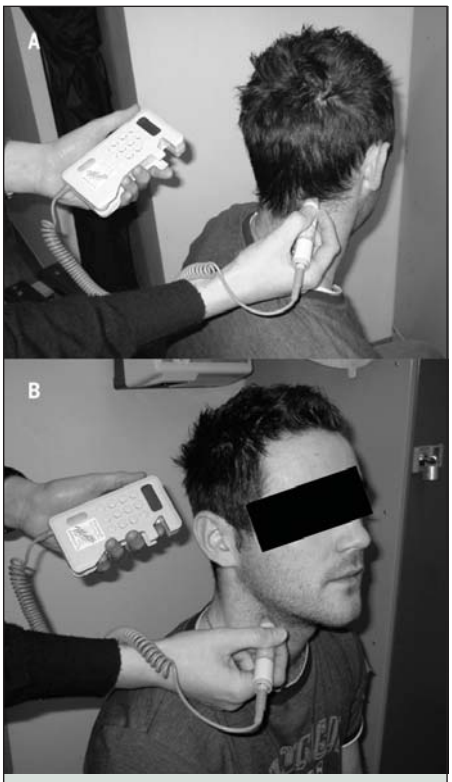

FIGURE 3. Blood flow examination with handheld ultrasound Doppler of (a) vertebral artery at the suboccipital site, and (b) internal carotid artery at the carotid sinus. The examination assesses velocity, quality, and presence/absence of blood flow in a specific vessel. The technique can be used to assess patency of flow during movement of the neck and head.

manifests as a drooping eyelid (ptosis), sunken eye (enophthalmia), a small and constricted pupil (miosis), and facial dryness (anhidrosis), due to the overbalance of parasympathetic activity in the eye. The syndrome is a result of interruption to the sympathetic nerve fibers supplying the eye. In the case of carotid induced Horner's syndrome, the pathology is classified as postganglionic. The superior cervical sympathetic ganglion lies in the posterior wall of the carotid sheath, and the postganglionic fibers follow the course of the carotid artery before making their way deep towards the eye through the cavernous sinus. Compression or ischemia as a result of internal carotid artery dysfunction will occur at the ganglion or distal to it. Some postganglionic sympathetic fibers that follow the course of the external carotid artery control facial sweating, accounting for the presence of anhidrosis in postganglionic Horner's syndrome.

In this second case report, the patient describes longstanding symptoms that do not relate to the classic pattern and symptomatology of cervical vascular disease. There were no subjective descriptions of potential cranial nerve palsy. At this stage, it was unlikely that the actual present features were indicative of vascular disease.

Predisposing Factors and Physical Examination

This patient reported a significant history of cervical vascular and, specifically, internal carotid artery disease. This in itself is considered evidence of poor cardiovascular status and a potential predictor of future stroke. The patient had known internal carotid artery occlusive disease. It is reported that internal carotid artery disease correlates to stroke, particularly in younger patients with diabetes.[15,35,45,46] Although the patient presents as an apparent high risk for adverse cerebrovascular event (internal carotid artery occlusion), her age and nondiabetic status may contribute towards lessening the judgment on chance of stroke.

As hypertension has been consistently reported to be associated with cerebrovascular events (as mentioned earlier), blood pressure measurements were taken and found to be high. The patient reported much lower home readings. Blood pressure monitoring is known to be influenced by several factors,[33] including the immediate environment, and, although care was taken with the testing, this reading could still be misleading. Ultrasound Doppler examination was undertaken to assess the patency of all 4 vessels during cervical spine movement (**FIGURE 3**). This examination technique has become a focus of interest for manual therapists in recent years and may be considered as a useful addition to physical examination for patients with suspected vascular pathologies.[64,66]

In this case, observable flow in both internal carotid arteries and vertebral arteries was maintained through all positions of cervical movement. During this procedure, there was no reproduction of signs and symptoms indicative of either hind-brain or internal carotid artery territory ischemia. As anticipated, cranial nerve examination revealed no remarkable findings. Neuromusculoskeletal examination revealed a reliable relation of the patient's pain to a specific stiff cervical joint complex, and local muscular dysfunction.

In summary, this patient, although presenting as apparently high risk for cerebrovascular event, demonstrated patent flow in 4 vessels during cervical movement, with no indication of transient brain ischemia. Her pain pattern was musculoskeletal in nature, and she did not present as classic vascular trauma indicative of impending stroke. Although there were obvious indicators of stroke risk (previous stroke, internal carotid artery disease), an informed decision could be made to reinstate manual therapy, supported by the fact the she derived great benefit from treatment. Manual therapy in this case would be subject to a number of caveats: the avoidance of end-of-range movements known to stress the 4 cervical vessels (primarily rotation and extension); monitoring of new, unexpected signs and symptoms indicative of vascular trauma; reassessment of blood pressure and cranial nerve function if vascular trauma is suspected; communication and/or referral with vascular or neurology clinic in the event of any suspect symptomatology; and emergency referral in event of acute onset symptomatology. Medical referrals should be supported with objective data from assessment procedures (blood pressure, cranial nerve examination, eye examination).

SUMMARY

This paper has presented contemporaneous evidence and information regarding cervical arterial dysfunction and manual therapy. These 2 case reports were used to illustrate how this

TABLE 4 Summary of Key Objective Examination Procedures for Differentiating Vasculogenic Head and Neck Pain*

Test	Purpose	Evidence Status	Limitations and Advantages
Functional positional test, cervical rotation	Affects flow in contralateral vertebral artery. Limited effect on internal carotid artery.	Poor sensitivity, variable specificity. Blood flow studies support effect on vertebral artery flow.	Only assesses posterior circulation. Results should be interpreted with caution. Recommended by existing protocols. Cannot predict propensity for injury.
Functional positional test, cervical extension	Affects flow in internal carotid arteries. Limited effect on vertebral arteries.	No specific diagnostic utility evidence available. Blood flow studies support effect on internal carotid artery flow.	Primarily assesses anterior circulation.
Blood pressure examination	Measure of cardiovascular health.	Correlates to cervical arterial atherosclerotic pathology.	Reliability dependent on equipment, environment, and experience. Continuous, not categorical, measure.
Cranial nerve examination	Identifies specific cranial nerve dysfunction resulting from ischemia or vessel compression.	No specific diagnostic utility evidence available.	Reliability dependent on experience.
Eye examination	Assists in diagnosis of possible neural deficit related to internal carotid artery dysfunction.	No specific diagnostic utility evidence available.	Eye symptoms may be early warning of serious underlying pathology.
Handheld Doppler ultrasound	Direct assessment of blood flow velocity.	Limited manual therapy specific evidence. Existing studies suggest good to excellent reliability. Validity requires further study.	Reliability dependent on equipment, environment, and experience.

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knowledge can be integrated into clinical decision making. Case report 1 revealed misdiagnosis in the presence of classic vertebral artery trauma. Closer attention to the literature in this area would have facilitated the clinician's understanding of vascular presentation and its relationship to head and neck pain. Case report 2 demonstrated decision making informed by knowledge of vascular dysfunction and how treatment could be provided to a patient with known arterial disease.

We acknowledge that these cases show a divergence from classically taught arterial pathology assessment (premanipulative screening) and accept that the literature is used within a different paradigm. We recommend that manual therapists involved in the assessment and management of cervical spine dysfunction include what is known about cervical arterial dysfunction in their daily clinical decision making. **TABLE 4** summarizes information on clinical utility for a number of the testing procedures referred to in this paper.

Specifically, the following recommendations, although not intended as definitive guidance, will assist advancement of practice and clinical reasoning based on the emerging evidence.

Recommendations

1. Develop a high index of suspicion for cervical vascular pathology, particularly in cases of cervical trauma. Note that, although motor vehicle accident has been reported as one of the most common causes of CAD,[6] as stated earlier, the actual prevalence of CAD posttrauma (although unknown) is likely to be extremely low. The clinician should be constantly aware that chronic-pain issues and psychological factors are major factors in this patient group[61] and should, therefore, be sensitive to the possible impact of reinforcing biomedical beliefs about a chronic-pain episode.
2. Develop increased awareness that neck pain and headache may be precursors to potential posterior circulation ischemia.
3. Expand manual therapy theory to encompass the whole cervical vascular system, including the carotid arteries.
4. Expand manual therapy theory and practice to include hemodynamic principles and their relationship to movement anatomy and biomechanics.
5. Develop an awareness of the limitations of current objective tests and enhance the knowledge that reliance on objective testing alone represents incomplete clinical reasoning.
6. Enhance subjective/objective examination by including vascular risk factors such as hypertension and procedures such as cranial nerve and simple eye examination.
7. Consider new advances in the objective assessment of cervical arteries.
8. In cases of acute onset headache "unlike any other," conservative treatment techniques are recommended in the early stages.
9. Where frank arterial injury is suspected prior to or following treatment, immediate triage to an appropriate emergency center is recommended, together with a report on any treatment methods undertaken.

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