



Masterclass

A 'system based' approach to risk assessment of the cervical spine prior to manual therapy

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ABSTRACT

This paper presents a clinical overview and update of cervical arterial dysfunction (CAD) for osteopaths and other clinicians who treat patients presenting with cervical pain and headache syndromes. An overview of a 'system based' approach to the concept of vertebrobasilar arterial insufficiency (VBI) is covered, with reference to assessment procedures recommended by commonly used guidelines. We suggest that the evidence supporting contemporary practice remains limited and present a more holistic approach to considering cervical arterial dysfunction. This 'system based' approach considers typical pain patterns and clinical progressions of both vertebrobasilar, and internal carotid arterial pathologies. Attention to the risk factors, pathomechanics and haemodynamics of arterial dysfunction is also given. We suggest that consideration of the information provided in this updated 'Masterclass' will enhance clinical reasoning with regard to differential diagnosis of cervical pain syndromes and prediction of serious adverse reactions to treatment.

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1. Introduction

This paper provides an updated overview of current thought; practice and research in the field of cervical arterial dysfunction and pre-treatment risk assessment and builds on a previous 'Masterclass' published in *Manual Therapy*,⁷⁷ in the light of new emerging research. Guidelines for screening patients for the risk of neurovascular complication post-manual therapy have been available for clinical use for a number of years.^{5–7,9} However, several authors have recently questioned the utility of such guidelines.^{4,15,31,55,72} These authors suggest that current practice based on available guidelines and information may be limited by a number of factors including: validity and reliability of the guidelines;⁴ validity and reliability of physical tests used for pre-treatment screening;^{55,72} uncertainty associated with clinical decision making;¹⁵ uncertainty of risks of treatment, an unsubstantiated knowledge base, a questionable evidence base to guidelines, and discomfort among the profession regarding medico-legal issues.^{4,31}

The main aim of this paper is to provide a framework for manual therapists to broaden their clinical approach to the understanding

and assessment of cervical arterial dysfunction. A more holistic approach can be achieved by considering recent advances in the evidence base, together with a change in thinking with regard to movement, and the resulting haemodynamics of the cervical spine. The paper is divided into two linked clinical summaries;

- 1) Vertebrobasilar arterial system (posterior system).
- 2) Internal carotid arteries (anterior system).

Risk factors and mechanisms of CAD are then presented, followed by an indication of possible directions for future approaches to clinical assessment.

2. Vertebrobasilar arterial system

Both traditional and contemporary thinking in manual therapy has been concerned with blood flow problems related to the vertebrobasilar arterial (VBA) system. The term 'vertebrobasilar insufficiency' (VBI) is a familiar term with all therapists and attempts have been made throughout the years to find the best way to identify patients with posterior circulation ischemia (e.g.^{37,7}) A brief review of the posterior vascular anatomy will help appreciate what is meant by the term VBI.

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2.1. The vertebrobasilar arterial system and vertebrobasilar insufficiency

The VBA system provides blood flow to the hind-brain (i.e. brain-stem, Medulla Oblongata, Pons, Cerebellum, and Vestibular apparatus). The left and right vertebral arteries arise from the subclavian arteries and pass through the transverse foramina of cervical vertebral levels 6 to 1 – see Fig. 1. When they exit the atlas, the vessels make a sharp postero-medial turn to pass along the posterior mass of the atlas. They then enter the skull through the foramen magnum of the occiput. The vessels are ‘tethered’ at various points along this route: namely C2 transverse foramina, C1 transverse foramina, and at the atlanto-occipital membrane. It is this tethering, combined with the convoluted route of the vessels around C2/C1 and the occiput, that have been a cause of concern for therapists. Considering this anatomy of the upper cervical spine it is easy to appreciate how, during rotation, the contralateral vessel may be stretched therefore potentially affecting flow (Fig. 2). This is the basis for the ‘VBI Tests’ that have commonly been advocated for VBI screening.

Once inside the skull, the two vertebral arteries join each other to form the basilar artery, which in turn feeds into the circle of Willis. When there is a reduction of blood supply to specific parts of the hind-brain, certain signs and symptoms are displayed. This is what can be referred to as VBI.

2.1.1. Vertebrobasilar insufficiency – signs and symptoms

Classically, the signs and symptoms related to hind-brain ischemia are considered as the “5 Ds and 3 Ns” of Coman.¹⁶ These signs and symptoms are presented in Table 1 (together with a ninth ‘classic’ sign – ataxia), along with the associated neuro-anatomical site of insult.

Unreasoned adherence to these cardinal ‘classic’ signs and symptoms can, however, be misleading and result in an incomplete understanding of patient presentations. A closer look at contemporary evidence from the medical, ophthalmic and neurological literature shows that the typical presentation of vertebrobasilar dysfunction is not always in line with this classical picture. The haemodynamic presentations of VBI can be better understood if the symptomology is divided into non-ischemic (i.e. local, somatic causes) and ischemic (i.e. symptoms of hind-brain ischemia) manifestations (see Table 2).

VBI may be a result of arterial dissection. This is a tearing of the intimal wall which may lead to severe stenotic lesions or embolisation this process has been well documented by a number of recent papers.^{25,26} The non-ischemic presentation of vertebral dissection is typically ipsilateral posterior neck pain and/or occipital headache alone – Figure 3 (e.g.^{2,8,15,59,71}) Very rarely cervical root impairment (usually C5/6) can be present as a result of local neural ischemia.¹⁸ These clinical features may then be followed by the ischemic events associated with vertebrobasilar dysfunction. These may also include some of the classic 5Ds and 3Ns as stated above, but may also include many other symptoms (see Table 2).^{2,55,59} It is rare for posterior dysfunction to manifest in only one sign or symptom, and isolated dizziness or transient loss of consciousness are often mis-attributed to posterior circulation ischemia.⁵⁹

Dizziness is often reported as being one of the most common symptoms of VBI.¹⁷ However, there have been cases reported when dizziness has not been present. The nature of dizziness can be a differentiating factor in establishing a vascular versus non-vascular cause. Typically, posterior circulation dizziness does not present as frank vertigo, although some authors have suggested this could occur (e.g. Savitz et al.⁵⁹) Vascular dizziness occurs as an effect of neck rotation, and does not improve with continued

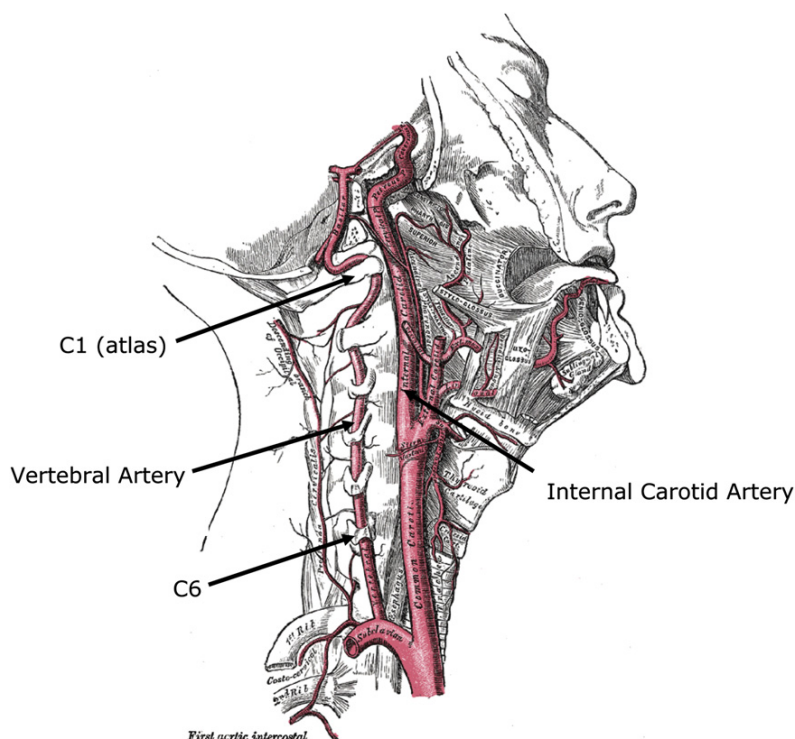


Fig. 1. Course of the vertebral and internal carotid arteries through the cervical spine. (adapted with permission from Elsevier Ltd, Drake et al. Gray's Anatomy for Students, www.studentconsult.com).

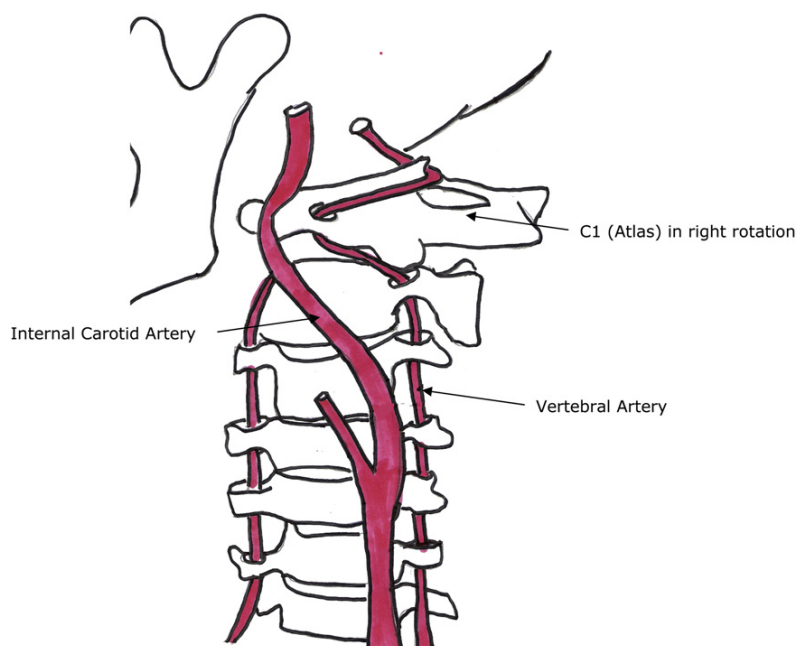


Fig. 2. Vertebral and Internal Carotid arteries during upper cervical rotation (Reprinted with the permission of NCMIC Group, Inc. No further reproduction is allowed without the express permission of NCMIC).

movement. This pattern differs from non-vascular vestibular dizziness (see below) which often has a short latency to it, and can improve with repeated movement.

2.2. VBI testing

2.2.1. Functional positioning tests

Functional positional tests of the cervical spine have been traditionally used by manual therapists to identify the presence of VBI.^{23,7} The purpose of establishing whether a patient has VBI is of obvious great importance to health professionals to whom a patient has sought help for their cervical pain. The reason for undertaking these tests is based on the principle that some treatment interventions commonly used to help patients with neck pain hold inherent risks if applied in the presence of VBI. It would seem, therefore, to be necessary to identify whether or not VBI was present. The primary risk associated with VBI (i.e. the longer term sequelae of these transient events) is one of neurovascular accident (i.e. stroke) as a result of further insult to an already compromised (insufficient) blood supply to the brain.

Functional positioning tests are based on the principle of compromising flow in the vertebral arteries by passively sustaining the cervical spine in a particular position. Positions can include extension, combined extension and rotation, a pre-manipulation position, or most commonly, rotation alone. The APA pre-manipulative guidelines suggest a 10 s sustained hold of rotation as a minimum requirement to establish whether or not VBI is present⁷ – Fig. 4. The purpose of these tests is to monitor for reproduction of symptoms associated with VBI during the sustained hold. Reproduction of symptoms during the test is classed as a positive test result and contraindicates certain treatment interventions.⁷

The underlying mechanical principle of these tests has been the subject of a number of research reports focusing on the clinical question of ‘does rotation of the neck affect blood flow?’ Many blood flow studies have demonstrated a reduction in blood flow in the contralateral vertebral artery during rotation (e.g.^{1,34,35,41,43,51,53,54,58}) Most of this work has been undertaken on asymptomatic subjects. Some authors have used these studies to support the validity of the ‘VBI’ test; in other words these studies demonstrate that rotation changes blood flow, therefore the test is valid. The test however, is for VBI and that is seldom if ever

Table 1

Classic signs and symptoms of vertebrobasilar insufficiency (VBI) with associated neuroanatomy. See text for the limitations of only considering these features for potential VBI.

Sign or Symptom	Associated Neuroanatomy
Dizziness (vertigo, giddiness, lightheadedness)	Lower vestibular nuclei (vestibular ganglion = nuclei of CN VIII vestibular branch)
Drop attacks (loss of consciousness)	Reticular formation of midbrain Rostral Pons
Diplopia (amaurosis fugax; corneal reflex)	Descending spinal tract, descending sympathetic tracts (Horner's syndrome); CN V nucleus (trigeminal ganglion)
Dysarthria (speech difficulties)	CN XII nucleus (Medulla, trigeminal ganglion)
Dysphagia (+hoarseness/hiccups)	Nucleus ambiguus of CN IX and X, Medulla
Ataxia	Inferior cerebellar peduncle
Nausea	Lower vestibular nuclei
Numbness (unilateral)	Ipsilateral face: descending spinal tract and CN V Contralateral body: ascending spinothalamic tract
Nystagmus	Lower vestibular nuclei + various other sites depending on type of nystagmus (at least 20 types)

Table 2

Presentations of vertebral artery dissection. Non-ischemic symptoms can precede ischemic events by a few days to several weeks.

Non-ischemic (local) signs and symptoms	Ischemic signs/symptoms
• Ipsilateral posterior neck pain/occipital headache • C5/6 cervical root impairment (rare)	• Hind-brain TIA (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 dyesthesia, photophobia, papillary changes, clumsiness and agitation) • Hind-brain stroke (e.g. Wallenberg's syndrome, Locked-In syndrome)

reproduced during such blood flow studies e.g. 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, and this has been mathematically demonstrated in diagnostic utility calculations.^{24,32,52} In a recent meta-analysis⁴² of VA blood flow velocity changes associated with cervical spine rotation. The author suggested that VA flow was found to be more compromised in patients than healthy individuals during contralateral rotation and more commonly in the intra-cranial portion of the artery during a sitting rotational test. 'VBI' symptoms were not reported by the subjects within the analysis, despite the significant changes in flow which were demonstrated.

2.2.2. Limitations of VBI and differentiation testing

On the basis of the inconsistency of the evidence, there have been recent propositions regarding cessation of the use of functional pre-screening tests.^{72,55} Despite some of the above mentioned tests being advocated in published guidelines for the assessment of VBI, and other tests being often quoted in textbooks (e.g. Hautant's test etc. in³⁸) it is essential that the clinician is aware of the limitations of using information gained from these tests in their diagnostic, clinical decision making. As stated above, the functional positional tests have poor diagnostic utility i.e. a positive test response does not necessarily mean that the condition (VBI) exists, and a negative test response does not necessarily mean the

condition does not exist. This phenomenon has been highlighted in a number of case reports and studies which have documented either patients having adverse neurovascular effects in the absence of a positive test (i.e. false-negative^{53,74}) or no identifiable vascular dysfunction despite a positive test result (i.e. false-positive³⁶).

With these limitations in mind, it is necessary to explore other possible approaches to the assessment of cervical arterial dysfunction. Below is a brief overview of the anterior cervical arterial system (the internal carotid artery) which appears to be a neglected source of diagnostic information within manual therapy literature and education.

3. The internal carotid arteries

Due to its perceived anatomical vulnerability, the posterior cervical arterial system has traditionally been the focus of attention for manual therapists. In order to enhance clinical reasoning and facilitate diagnostic decisions and judgments, it is necessary to consider an approach which incorporates the *anterior* cervical arterial system; i.e. the internal carotid arteries (ICA). Knowledge of the ICA is important for manual therapists because;

1. The ICA's provide the most significant proportion of blood to the brain.^{21,62}
2. Pathological changes of the ICA are very common.³
3. Blood flow in the ICA is known to be influenced by movement of the neck.^{54,60,61}

3.1. The internal carotid arteries and related pathologies

The ICAs carry for the majority of blood flow to the brain – around 80% – compared to 20% through the posterior system. It is primarily increased flow through the ICA which helps maintain brain perfusion in the presence of reduced flow through the

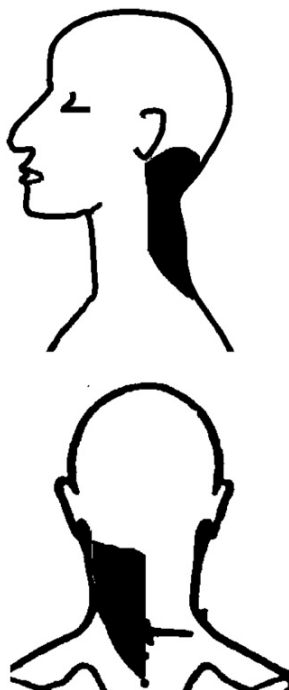


Fig. 3. Typical pain distribution relating to extra-cranial vertebral artery dissection – ipsilateral posterior upper cervical pain and occipital headache.



Fig. 4. Functional positional testing of the vertebral artery (rotation). The patient's head is passively rotated and held for 10 s. Reproduction of symptoms associated with vertebrobasilar insufficiency result in a positive test.

vertebral arteries. The ICA arise from around the C3 level of the cervical spine where they bifurcate (with the External Carotid Artery) from the Common Carotid Artery (see Figs. 1 and 2). The course of the ICA takes them through a number of contractile structures such as the sternocleidomastoid, longus capitis, stylohyoid, omohyoid, and diaphragm muscles. In the upper cervical spine, they pass by the anterior body of C1, to which they are tethered. The ICA enters the skull through the carotid canal in the petrous temporal bone, where it continues intra-cranially to join the Circle of Willis. Extra-cranially, the flow through the ICA is influenced by movement of the cervical spine – primarily extension, and less so, rotation.^{54,60}

3.2. Internal carotid artery (anterior) dissection

The ICA supplies the brain and the retina. The natural onset and progress of ICA dissection begins with local arterial trauma (the dissection event itself). This dissection event can manifest in a number of signs and symptoms which, like early vertebral artery dissection, are *non-ischemic* (i.e. somatic pain related to local injury). These local signs and symptoms can precede cerebral ischemia (TIA or stroke) or retinal ischemia by anything from less than a week, to beyond 30 days.^{12,76} There is, therefore, a period of time when a patient with ICA dissection may present to the manual therapist with signs and symptoms which may mimic a neuromusculoskeletal presentation.⁶⁹ Table 3 shows the classic ICA non-ischemic and ischemic manifestations of ICA dissection.

It is important to appreciate that most commonly, particularly in the early stages of the pathology, headache and/or cervical pain can be the sole presentations of internal carotid artery dysfunction.^{47,56,69} Fig. 5 shows a typical pain distribution associated with dissection of the ICA. The fronto-temporal headaches are often described as cluster-like, thunder-clap, migraine without aura, hemispheric continua, or simply “different from previous headaches”.^{2,13,56,63,69} The upper cervical or antero-lateral neck pain, facial pain and/or facial sensitivity are described in medical literature as “carotidynia”.

The local pain mechanisms involved with the internal carotid artery are likely to be related to either deformation of nerve-endings in the tunica-adventitia, or direct compression on local somatic structures.⁴⁴ Specifically, the terminal nerve-endings in the carotid wall are supplied by the trigeminal nerve, which accounts for instances of facial pain and carotidynia. Stimulation of the trigemino-vascular system may account for this carotid induced pain.⁴⁵

Cranial nerve palsies and Horner's syndrome are phenomena which are often indicative of internal carotid artery pathology, especially if the onset is acute. The hypoglossal nerve is the most



Fig. 5. Typical pain distribution relating to dissection of Internal Carotid Artery – ipsilateral front-temporal headache, and upper/mid cervical pain.

commonly affected followed by the glossopharyngeal, vagus, or accessory.^{76,2} However, all cranial nerves (except the olfactory nerve) can be affected.⁷⁶ If the dissection extends into the cavernous sinus, the oculomotor, trochlear, or abducens can be affected.^{33,76}

The two most likely mechanisms for these cranial nerve palsies are:

1. Ischemia to the nerve via the vasa nervorum (comparable to peripheral neurodynamic theory).
2. Direct compression of the nerve axon by the enlarged vessel.^{2,33,76}

Identification of the early stages of ICA dissection may be facilitated by testing the cranial nerves and observing the eyes. Cranial nerve and eye examination should therefore be an integral and important component of manual therapists' assessment procedures. Previous authors have also highlighted the importance of neurological examination with regard to cervical arterial dysfunction.^{50,15}

Horner's syndrome has been found to be present in up to 82% of patients with known internal carotid dissection.¹⁴ Most commonly, this syndrome occurs with head, neck, or facial pain. Carotid induced Horner's syndrome manifests as a drooping eyelid (ptosis), sunken eye (enophthalmia), a small, constricted pupil (miosis), and facial dryness (anhidrosis). The syndrome is the result of interruption to the sympathetic nerve fibres supplying the eye. In the case of carotid Horner's syndrome, the pathology is classed as post-ganglionic. The superior cervical sympathetic ganglion lies in the posterior wall of the carotid sheath, and the post-ganglionic fibres 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 dysfunction will occur at the ganglion or distal to it.

In addition to the above early signs, it is important for the manual therapist to be aware of signs and symptoms related to cerebral, and retinal ischemia. It is unlikely that a patient with full stage cerebral ischemic stroke will present to the manual therapist, but the more subtle presentation of retinal ischemia might, which makes simple eye examination a key part of assessment. The internal carotid artery supplies (via the ophthalmic artery) the retina, and emboli from the ICA can result in retinal ischemic dysfunction. Symptoms include a painless episodic loss of vision, or blackout (amauris fugax), and localized/patchy blurring of vision

Table 3

Clinical features of ICA dissection. Non-ischemic signs and symptoms may precede cerebral/retinal ischemia by anything from a few days to over a month.

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

(scintillating scotomas). Orbital ischemia syndrome, as a result of ophthalmic artery occlusion, presents as weakness of the ocular muscles (ophthalmoparesis); protrusion of the eye due to weakness of extrinsic eye muscles (proptosis); swelling of the eye or conjunctiva (chemosis).^{2,19,76}

4. Aetiology of cervico-cranial arterial dysfunction

Whilst the exact mechanism of arterial dissection remains unexplained, vertebral and internal carotid artery disease and dysfunction are intrinsically associated with two inter-related principles:

- 1) Underlying pathology (including atherosclerosis) which may predispose a vessel to dissection.
- 2) Mechanical forces generated as a result of movement or biomechanics, which results in altered haemodynamics.

Both of the above may be linked to trauma to the blood vessels. Atherosclerosis is an inflammatory process associated with a number of factors including^{29,40,57}

- hypertension
- hypercholesterolemia
- hyperlipidemia
- hyperhomocysteinemia
- diabetes mellitus
- genetic clotting disorders
- infections
- smoking
- free radicals
- direct vessel trauma
- iatrogenic causes (surgery, medical interventions).

It is important for the clinician to appreciate that hypertension (indicated by measurement of blood pressure) is positively related to disease and dysfunction of the carotid arteries.^{20,30,39,49,67} Consequently, this may indicate that recognition of hypertension by the clinician could be important when assessing the likelihood of potential cervico-cranial neurovascular dysfunction.

4.1. Mechanisms of cervico-cranial dysfunction

Important mechanisms in the pathogenesis of localized vascular pathology for clinicians to consider are;

- I) Spontaneous arterial dissection is known to occur in certain individuals and is often related to *innocuous* day to day movements such as turning to reverse the car or visiting the hairdresser.¹³ The pathogenesis of such events remains unknown but is considered by some to be due to inherent vessel wall weakness linked to connective tissue abnormalities.^{46,11}
- II) Intimal trauma (intimal dissection/injury) is known to occur as a result of blood flow changes and/or vessel wall pathology due to *frank trauma*, i.e. extreme neck movement, sustained neck movement, or repeated neck movement (e.g. whiplash injury, domestic violence, sport, medical interventions, intuition, manual therapies etc.^{2,25}
- III) Localised endothelial inflammatory events (i.e. atherosclerosis)^{57,29} linked to abnormal flow in vessels due to biomechanical factors such as kinking/looping or localized obstructions (e.g. 1st rib and subclavian artery).
- IV) Endothelial inflammatory disease – e.g. temporal arteritis. Giant cell arteritis of the Temporal Artery (extra-cranial

branch of the External Carotid Artery) can present as unilateral headache and/or temple soreness, sore neck, and jaw soreness. The medium-term sequelae of this disease is potential blindness as a result of ischemia to the optic nerve, thus making early recognition critical.⁶⁴ Temporal arteritis has also been associated with ICA and VBA disease.⁴⁸

- V) Upper cervical instability has been associated with localized atherosclerotic changes in the cervical vessels.^{75,22} The mechanism of injury is possibly associated with repetitive micro-trauma to the VA and ICA secondary to increased upper cervical vertebral movement and/or the presence of connective tissue inflammatory disease. Consideration should be given to patients with known rheumatoid arthritis and acute whiplash injury.

5. Directions for the future

It is becoming progressively clear that the current manual therapy knowledge base does not equip therapists with the information required to make valid risk assessment prior to treatment. The alert clinician requires not only the vast neuromusculoskeletal knowledge base but also integration of the basic functional anatomy of the arterial system. Knowledge of haemodynamic principles, pathophysiology, risk factors of arterial dysfunction and *above all an awareness of classical vascular clinical presentations* is paramount.

The integration of such knowledge will allow the manual therapist to make the best informed decisions when assessing and treating patients presenting with head and neck symptoms. It is important for the clinician to understand that *headache/neck pain may be the early presentation of an underlying vascular pathology*.

The task for the therapist is to differentiate the symptoms by:

1. Having a high index of suspicion
2. Testing the vascular hypothesis

This should take place at an early point in the assessment process – i.e. soon into the history taking. The symptomology and history of patients suffering vascular pathology is what may reveal the alert clinician to an underlying problem.

Reliance solely on objective clinical tests i.e. so called “vertebral artery tests” which have poor validity and reliability,^{70,72} should be avoided.

As movement of the neck, particularly rotation and extension movements, can be a potential risk factor for vascular events in itself, identification of patients with other pre-existing vascular risk factors (especially hypertension) should also be of great importance to the therapist before manual therapy interventions are undertaken. Careful monitoring of patients' signs and symptoms after treatment is also necessary, especially acute post-treatment onset of localized upper cervical pain, or headache, which is worsening. Furthermore, where post-treatment pain or “treatment soreness” is encountered (i.e. an apparent response to joint or soft tissue techniques), the therapist should consider carefully whether there has been a vascular or haemodynamic response to treatment. For example, a recent survey of Irish manipulative therapists⁶⁸ revealed a range of reported adverse events (26% of clinicians reported an adverse event in the 2 years studied) including drop attacks and transient ischemic attack, both of which occurred during non high velocity thrust techniques. Numerous reports suggest that such presentations may be the manifestation of a traumatically (treatment) induced arterial trauma or dissection.⁶⁵

A high index of suspicion of cervical vascular involvement is required in cases of acute onset neck/head pain described as “unlike any other”. Observation and conservative treatment may well be

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 VA 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 ICA flow.	Only assesses anterior circulation.
Blood pressure examination	Measure of cardiovascular health.	Correlates to ICA atherosclerotic pathology.	Reliability dependent on equipment, environment, and experience.
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 ICA dysfunction	No specific diagnostic utility evidence available.	Eye symptoms may be early warning of serious underlying pathology.
Hand-held Doppler ultrasound	Direct assessment of blood flow velocity	Limited manual therapy specific evidence. Debate within literature re; reliability, validity, clinical utility and interpretation of findings.	Reliability dependent on equipment, environment and experience.
Therapist administered Questionnaire screening	Questionnaire assessment of events related to movement and 'VBI'/Cranial nerve symptoms.	No specific diagnostic utility evidence available. Requires further study.	Reliant on patients' interpretation of events.

advised in such cases in the early stages of treatment, unless frank arterial injury is suspected (especially in the presence of posterior circulation ischemia). In this case, the appropriate action is triage to an emergency or suitable diagnostic centre as a matter of urgency, particularly in the case of a deteriorating patient. Vascular testing such as Duplex ultrasound, magnetic resonance arteriography and computerized tomographic angiography are increasingly sophisticated methods of vascular diagnosis with increasing reliability. The key maxim for the clinician is as always DO NO HARM.

Medical evidence suggests that the diagnosis of carotid and vertebral arterial dissections is on the increase, as awareness develops and diagnostic imaging becomes more reliable and less expensive. The causes of arterial dissection remain largely unknown, but are thought to involve a combination of genetic predisposition and environmental factors such as trauma. Early diagnosis is essential to prevent the potential sequelae of stroke. Manual therapists may be exposed to patients presenting with the early signs of stroke (i.e. neck pain/headache) and as such need both knowledge and awareness of the mechanisms involved. A basic understand of vascular anatomy, haemodynamics, and the pathogenesis of arterial dysfunction may help the clinician differentiate vascular head and neck pain from a musculoskeletal cause. It is apparent, however, that dissemination of knowledge and further work is necessary in establishing the best way to identify patients who may present as, or be at risk of neurovascular accident as a result of treatment.

One interesting and ongoing focus of ongoing clinical research is the use of simple hand-held ultrasound Doppler units to objectively assist in identifying flow dysfunction.^{27,55,73} This method has been the subject of a recent academic debate in *Manual Therapy* following the publication of an article by Thomas et al.⁷³ which cast doubt on the validity and reliability of such testing methods. The authors of this article raise a number of key points which add to the dialectic.

1. The alterations in flow detected by the Doppler velocimetry studies may well be an artefact of normal movement, similar to that found in the lower limb during cycling¹⁰ and the upper limb at end range positions.⁶⁶
2. None of the subjects studied⁷³ appear to have suffered any 'VBI' symptoms, despite 'revealing 30% more patients with altered blood flow than would have been detected by pre-treatment positional testing'. This raises the question; does the use of Doppler velocimetry give us any further information than VBI

testing? Assuming a clinician discovers (via the medium of velocimetry) that VA flow is significantly reduced, but in the absence of 'VBI' symptoms, how does that information direct the clinical decision making process? The suggestion being therefore, under those circumstances that the system has adequately compensated for the reduced flow.

3. Considering the high cost of the equipment required, the high level of training and the reported poor validity/reliability, does Doppler velocimetry *really* add any further value for the clinician?
4. The authors beg the question of whether the use of a patient questionnaire similar to that devised by,²⁸ may prove to be as clinically effective and a much cheaper, pre-treatment screening tool, providing it is combined with sound haemodynamic knowledge and clinical reasoning skills.

Table 4 gives a summary of the objective examination procedures referred to so far.

6. Summary

Attempts have recently been made to provide guidelines for the effective screening of patients who may be at risk of neurovascular accident post-manual therapy. However, current evidence questions the validity and utility of such guidelines. It is therefore necessary to re-consider the clinical approach towards assessment of potential cervical arterial dysfunction. Based on the existing evidence base, the authors suggest manual therapists consider the following recommendations;

1. Expand manual therapy theory to encompass a 'systems based' approach, incorporating the whole cervical vascular system, including the carotid arteries.
2. Expand manual therapy theory and practice to include haemodynamic principals and their relationship to movement anatomy and biomechanics.
3. Develop a high index of suspicion for cervical vascular pathology, particularly in cases of acute trauma.
4. Develop increased awareness that neck pain and headache may be precursors to potential posterior circulation ischemia.
5. Enhance subjective/objective examination by including vascular risk factors such as hypertension, and procedures such as cranial nerve and simple eye examination.
6. Consider new advances in the subjective assessment of cervical arteries such as questionnaire screening.²⁸

7. Develop an awareness of the limitations of current objective tests such as pre-treatment movement testing and the proposed use of hand-held Doppler ultrasound. This should enhance the knowledge that reliance on objective testing alone, represents incomplete clinical reasoning.
8. In cases of acute onset headache “unlike any other” couples with ambiguous examination findings, retain an index of suspicion and use conservative or gentle treatment techniques in the early stages of management.
9. Where frank arterial injury is suspected prior to or following a treatment intervention, immediate triage to an appropriate emergency centre is recommended, together with a report on any treatment methods undertaken.

The summarised points above are not intended as definitive guidance – rather an advancement of theory, practice and clinical reasoning, based on the constantly emerging evidence base.

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