

# Causes of complications from cervical spine manipulation

Timothy Mann<sup>1</sup> and Kathryn M. Refshaug<sup>2</sup>

<sup>1</sup>Private Practice, New South Wales <sup>2</sup>The University of Sydney

Cervical manipulation occasionally causes serious vertebrobasilar complications. The usual cause is vertebral artery dissection, however in some cases there has been no obvious arterial injury. The present paper reviews the mechanisms by which complications occur, particularly when the applied force is trivial or there is no injury to the vertebral arteries, and the factors that increase risk of complications. In addition, implications are drawn for use of the recently revised Australian Physiotherapy Association (APA) guidelines.

In the absence of vertebral artery rupture, complications are proposed to arise from vasospasm, haemostasis, endothelial injury or turbulent flow. These mechanisms have a sound scientific basis but have yet to be demonstrated as specifically causing vertebrobasilar complications.

The most important risk factors for vertebrobasilar complications appear to be prior trauma to the vertebral arteries and symptoms of vertebrobasilar ischaemia from previous manipulation. There is weak evidence that hypoplasia of the vertebral arteries also increases the risk of complications. Neither general vascular factors nor pre-existing degenerative conditions of the cervical spine increase risk of vertebrobasilar complications.

The procedures described in the APA guidelines test adequacy of total cerebral perfusion during cervical movements rather than patency of the vertebral arteries or their susceptibility to injury. The guidelines may therefore indicate potential for surviving a complication from manipulation. They may also identify patients at risk of complications from minor trauma.

It is recommended that the procedures described in the APA guidelines be applied prior to every manipulation, and that manipulation be avoided in the presence of any signs of vertebrobasilar insufficiency. [Mann T and Refshaug K (2001): Causes of complications from cervical spine manipulation. *Australian Journal of Physiotherapy* 47: 255-266]

Key words: Cervical Vertebrae; Manipulation, Orthopedic; Vertebral Artery; Vertebrobasilar Insufficiency

## Introduction

The risk of vertebrobasilar complications contributes significantly to a therapist's decision on whether to perform cervical manipulation. Complications can be serious because the vertebral and basilar arteries provide up to 20% of total cerebral blood flow (Lord 1973), supplying brainstem and cerebellar structures (Crawford et al 1984, Lord 1986, Schmitt 1991, Williams and Warwick 1980). Compromise of vertebrobasilar circulation therefore results in ischaemia of any of these structures, causing symptoms primarily of dizziness, but also of nystagmus and altered trigeminal sensation or, in extreme cases, stroke or death (Fujita et al 1995, Lord 1986). Such complications arise not only from cervical manipulation, but also from spontaneous head turning and sustained postures involving rotation, extension or traction, without obvious injury to the vertebrobasilar system (Endo et al 2000, Frumkin and Baloh 1990, George and Laurian 1987, Okawarra and Nibbelink 1974, Parkin et al 1978). This paper focuses particularly on potential mechanisms of injury when there is no observable damage to the vertebral artery.

The reported incidence of severe adverse events varies widely from one in 20,000 patients receiving cervical

manipulation to one per million manipulative procedures (Vikers and Zollman 1999), but may be as high as 1:4,500 (Dunne et al 2000). It is often suggested that the incidence is probably seriously underestimated because many, or even most, adverse events are not reported in the public domain (Krueger and Okazaki 1980, Robertson 1981, Sinel and Smith 1993). This is confirmed by confidential data from recent Australian court cases and insurance companies (personal communication). Physiotherapists should be cognisant that these recent data involve the profession to a far greater extent than is often appreciated.

The potential severity of complications from manipulation led the Australian Physiotherapy Association (APA) to advocate use of a protocol to test adequacy of vertebral artery blood flow as a possible predictor of risk of poor outcome from manipulation (APA 1988). The physical component of the current guidelines has recently been revised (Magarey et al 2000). The current guidelines assess symptoms during sustained rotation of the cervical spine held at the end of range, but do not replicate the rapidly applied thrusting force of manipulation. It is possible, however, that the guidelines may identify risks from manipulation not associated with major arterial damage, such as the development of haemostasis or vasospasm (Schmitt 1991).

The purpose of this paper is to examine causes of serious complications arising from manipulation of the cervical spine with and without injury to the vertebral arteries. This analysis is followed by discussion of the guidelines and their use for determination of risks associated with manipulation. Knowledge of the anatomy and physical properties of the vertebral arteries is central to understanding the risks, and therefore is presented first for contextual relevance. A search of the MEDLINE, PubMed, EMBASE and CINAHL databases from 1966 was conducted, although the volume of published work precludes citation of any but key articles. For example, more than 1100 publications about blood flow in the vertebral arteries were located.

### **Anatomical structure of the cervical spine and the vertebral arteries**

The course of the vertebral arteries is thought to render them particularly vulnerable to injury from mechanical trauma (Dunne et al 1987, Schmitt 1991). However, the marked variability in vertebrobasilar anatomy among individuals is likely to result in vulnerability that varies in magnitude between individuals (Guiffre and Sherkat 1999, Johnson et al 2000, Macchi et al 1996). In general, from their entry at approximately C6, the vertebral arteries ascend within the transverse foramina of the cervical vertebrae, where they are enclosed and relatively fixed. They are thus protected for most of their extracranial course until their exit from C2. Injury from cervical manipulation rarely, if ever, occurs in this part of the vertebral artery, damage to this part of the vertebral artery requiring large traumatic forces.

The artery passes through the C2 intervertebral foramen to the more laterally located intervertebral foramen of C1. At their exit from C2, the vertebral arteries are exposed to large shear and tensile forces during cervical spine movement, in particular rotation and lateral flexion (Braun et al 1983, Caplan 1991, Goel et al 1988, Nibu et al 1997, Schwartz et al 1991). From C1 (the third section of the artery) the vertebral artery usually passes medially across the lateral process of C1 through a groove, often beneath a bony ring towards the foramen magnum (George and Laurian 1987, Thiel 1991), although frequently it passes directly cephalad to the foramen magnum (Johnson et al 2000). Thus, the vertebral artery is fixed at several sites: the C1 transverse foramen, the groove on the superior aspect of C1, and at the dura intracranially (Johnson et al 2000, Macchi et al 1996). The vertebral arteries are frequently injured by manipulation at these sites of fixation (Frumkin and Baloh 1990, George and Laurian 1987, Sim et al 1993, Sullivan 1992). The intracranial portion of the vertebral arteries is rarely injured from cervical manipulation (Mechler 1988, Pollanen et al 1992).

### **Collateral circulation of the vertebral artery and basilar artery and its relationship to complications**

The branches of the vertebral and basilar arteries (eg posterior inferior cerebellar artery) are important sources

of collateral circulation that can prevent ischaemia in cases of injury to the vertebrobasilar system. The posterior inferior cerebellar artery is the most common site of occlusion from propagating thrombus or embolism caused by injury to the third section of the vertebral artery (Mechler 1988, Nagler 1973), resulting in lateral syndrome of Wallenberg, the most common form of stroke from cervical manipulation (Caplan 1986).

### **Relationship between structure of the vertebral arteries and failure**

The walls of the vertebral artery, like all human blood vessels, consist of three layers: the tunica intima, tunica media and adventitia (Pollanen et al 1992, Silver 1987). The composition of the vertebral artery varies both among its layers and along its length (Bevan 1979), probably in response to the different demands along its course (Silver 1987). The different stiffness of each of the three layers may contribute to the complications from minor trauma as well as from manipulation.

The most important determinants of stiffness of all arterial walls are collagen, smooth muscle, and particularly elastin (Dobrin 1978). Stiffness of arterial tissue alters under various conditions; stiffness increases with stretch (Johnson et al 2000) and ageing (Nagasawa et al 1982). Throughout life, the collagen content increases, the amount of smooth muscle decreases, and the lumen diameter decreases, resulting in stiffer arteries (Nagasawa et al 1982, Piffer and Zorzetto 1980) and a decline in total cerebral blood flow (Dorfler et al 2000), suggesting that older patients may be at greater risk of complications from manipulation. However, arterial stiffness is also modulated by other factors, such as contraction of the smooth muscle which, in small arteries including the vertebral artery, leads to arterial constriction and decreased stiffness of arterial walls (Berczi et al 1990). Furthermore, there is great variability in vertebral artery stiffness within and between individuals, consistent with the variability in anatomical structure (Johnson et al 2000, Lord 1986).

The elasticity of the vertebral arteries varies with the differing composition of each of the layers. The thick innermost layer, the *tunica intima*, is the most elastic, being composed of a single layer of endothelial cells and a network of elastic fibres with a predominantly longitudinal orientation (Piffer and Zorzetto 1980). The tunica intima is susceptible to rupture from rapidly applied tensile forces (Caplan 1986) such as occur during manipulation, suggesting that either speed or magnitude of the applied force may be factors involved in complications.

The thinner *tunica media* contains a few collagen fibres, smooth muscle that is mainly arranged circumferentially, and is sparsely interposed with some elastic fibres (Piffer and Zorzetto 1980) and is therefore less elastic than the tunica intima. Unlike most other human arteries there are also some longitudinally arranged muscle fibres (Piffer and Zorzetto 1980). It is likely that these longitudinal fibres resist longitudinal tensile forces and the circumferentially

arranged fibres, in addition to resisting radial forces, resist rotational forces. Although lesions of the tunica media can occur directly from trauma, they occur more frequently from release of enzymes associated with the trauma (Pollanen et al 1992).

The thick outermost layer, the *tunica adventitia*, the stiffest layer, is comprised mainly of collagen fibres arranged longitudinally in the inner aspect, and circumferentially in the external layer, and is interspersed with some elastin fibres (Piffer and Zorzetto 1980). Interestingly, the adventitia is infrequently injured during manipulation of the cervical spine (Blaauw et al 1976, Dunne et al 1987).

*Longitudinal variations:* The composition, and therefore stiffness, of the vertebral artery varies along its length as its major function changes. The extracranial vertebral artery resists tensile forces, whereas the intracranial section maintains transmitted blood pressure (Wilkinson 1972). Reflecting the change in primary function, there is an abrupt decrease in the elastic elements in the adventitia and tunica media at the transition from extracranial to intracranial vertebral artery, the amount of elastin remaining being negligible ~0.5cm distal to dural perforation (Bevan 1979, George and Laurian 1987, Wilkinson 1972). The intracranial vertebral artery is therefore substantially stiffer than the extracranial vertebral artery (Nagasawa et al 1982).

*Failure of the vertebral artery:* During physiological movements in cadavers, the vertebral artery elongated an average of 5.8mm in side flexion and 4.7mm in rotation, with no appreciable elongation during flexion and extension movements (Nibu et al 1997, Sim et al 2000). The vertebral artery was also shown to exceed its physiological range by one millimetre in response to accelerations as low as 24.5m/s<sup>2</sup> (Nibu et al 1997) and to fail when elongated 16%-20% (Johnson et al 2000) ie ~6-14mm (Nibu et al 1997). In addition, acceleration of the applied load was significantly and positively correlated with vertebral artery elongation (Nibu et al 1997). Since elongation of an artery before failure is a function of the rate of application of the load, and because the viscoelastic artery becomes stiffer with increased loading rate (Westerhof and Noordegraaf 1970) it is likely that the vertebral arteries would fail earlier with forces applied at higher speeds.

### **Manipulation: Which features account for the risk?**

The characteristics that distinguish a manipulation from other passive manual techniques are the thrusting force and the speed of the manoeuvre. Interestingly, however, the force and amplitude, more than the speed of the manipulative thrust, are generally thought to be responsible for vertebral artery injury (Frisoni and Anzola 1991, Smith and Estridge 1962). The direction of the manipulation has also been implicated, the general consensus being that rotation (with or without extension) holds the greatest risk (Dunne 2000, Schmitt 1991). However, there is evidence that manipulations with a lateral flexion component, or in

fact any type of "rapid jerking movement" (Haldeman et al 1999, p. 790) can also cause injury. The force and duration of the manipulative thrust have been evaluated, but little is known about the transfer of the externally applied manipulative forces to the vertebral arteries or the speed or the amplitude of the manipulation.

*Force:* The forces applied during manipulation have been evaluated indirectly by measuring forces generated at the skin rather than forces resisted at the spinal joints (Herzog et al 1993, Kawchuk et al 1992). The mean peak force used in the cervical spine was 118 N, compared with 399 N in the thoracic spine and 328 N at the sacroiliac joint (Conway et al 1993, Herzog et al 1993, Kawchuk and Herzog 1993, Kawchuk et al 1992). That is, the force necessary to elicit the "click" associated with manipulation was much less in the cervical spine than in other spinal regions. However, the force transferred to the vertebral arteries has not been determined, and consequently the torque applied to the vertebral arteries that must be resisted by the arterial walls is unknown, but it could be assumed that use of higher forces is associated with larger amplitude of manipulation, and possible arterial failure through elongation.

*Speed:* Because the vertebral arteries are viscoelastic, the high speed (velocity or acceleration) of the manipulative thrust is thought to cause dissection of the vertebral artery at lower forces and smaller elongation than slowly applied techniques (Goldstein 1982, Mechler 1988, Meier 1981). Although the speed of the thrust may be of major concern (Schmitt 1991), it is the duration of the manipulation, rather than the speed of the thrust that has been measured. The duration of the manipulative thrust applied to the cervical spine ranges between 92ms and 32ms, compared with ~150ms in the thoracic spine (Herzog et al 1993, Kawchuk et al 1992, Kawchuk and Herzog 1993), and has not been measured in other spinal regions. The substantially shorter duration of manipulation performed in the cervical spine than in the thoracic spine could suggest that the thrust is faster, although lack of information about thrust amplitude prevents definitive conclusions. Because of the viscoelastic properties of the vertebral arteries, speed of the manipulation should be assumed to be associated with the risk of vertebrobasilar complications until further evidence is available.

*Amplitude:* The reason that the C1/C2 segment of the vertebral artery is most frequently injured is thought to relate to the great mobility of the C1/C2 intervertebral joint that causes considerable elongation of the vertebral artery in this region (Horn 1983, Nibu et al 1997, Schneider et al 1972, Simeone and Goldberg 1968). Cervical rotation elongates both the contralateral and ipsilateral vertebral arteries: the contralateral vertebral artery elongates by 10% and kinks at ~30 degrees rotation as it exits the C2 transverse foramen (Braakman and Penning 1971), the ipsilateral vertebral artery kinks and elongates at 45 degrees rotation (Selecki 1969), although more recent evidence suggests that the ipsilateral vertebral artery undergoes shortening rather than elongation during rotation and side flexion (Nibu et al 1997). Elongation of

the vertebral artery reduces lumen diameter (Learoyd and Taylor 1966), causing altered haemodynamics such as turbulence or haemostasis (Johnson et al 2000). Turbulence can cause minor trauma to the vertebral artery, including damage to the endothelium, and both turbulence and haemostasis can cause thrombosis (Johnson et al 2000, Lord 1986, Vaccaro et al 1998). Thus, manipulations in positions of less than 30 degrees rotation may cause less tensile and radial stress within the vertebral artery and avoid reduction in lumen diameter, but as yet there is no evidence of this decreased risk. It should be assumed however, in the absence of further evidence, that the combination of large amplitude at end of range and high speed is likely to carry more risk to the viscoelastic tunica intima and media than a small amplitude, slower manoeuvre performed near the beginning of range with little force.

### **Can reflex muscle action decrease the risk of vertebral artery damage from manipulation?**

The role of muscles, apart from absorbing some of the applied forces, is unclear. It is often suggested that the speed of the manipulation precludes reflex contraction of the cervical muscles (eg Schmitt 1991). Indeed, Herzog et al (1999) reported that reflex responses to high speed, low amplitude thrusts in the cervical spine using surface EMG commenced 50-100ms after the thrust commenced. However, it is theoretically possible that such responses could occur during manipulation. The duration of a cervical manipulation is ~100ms (Herzog et al 1993, Kawchuk et al 1992) and the conduction velocity of the large diameter axons is ~70ms (Kandel et al 1991). Given the proximity of the cervical muscles to the exit of the nerve roots (likely to be < 2cm), the reflex latency of the cervical muscles is probably less than a few milliseconds. Therefore, EMG activity in response to the manipulation could occur within the duration of the manipulation, although force generation (ie muscle contraction) is likely to take longer. However, if positioning the cervical spine in preparation for the manipulation stimulated EMG activity, reflex muscle contraction could occur within the duration of the manipulation. Such a contraction may reduce the applied force and amplitude of the manipulation and/or induce muscle injury, but this has yet to be investigated.

### **How do vertebrobasilar complications occur?**

In the majority of cases where damage to the vertebral arteries results from manipulation, frank injuries to the artery can be identified, commonly in the third section (Patiijn 1991). The primary lesion is generally a mural tear which causes dissection (Caplan and Sergay 1991), usually between the tunica media and tunica intima (Terrett 1987) or the tunica adventitia and tunica media (Dunne et al 1987). The dissection is filled by haemorrhage from the vasa vasorum and the resultant swelling causes obstruction of the vessel lumen (Dunne et al 1987). The reduction of lumen diameter is thought to alter the haemodynamic flow to the point of thrombogenesis (Caplan and Sergay 1991). Such alteration of the haemodynamic flow could also

injure the endothelium (Pollanen et al 1992). Prognosis for acute occlusion in the vertebrobasilar system is poor, with mortality as high as 86% (Becker 1997).

On many occasions, major arterial injury has been assumed even when investigations such as autopsy, Doppler ultrasound or angiography have not been conducted. However, there are also reports of complications, including death, where damage to the vertebral artery was minor or absent (Daneshmend et al 1984, Easton and Sherman 1977, Katirji et al 1985, Parkin et al 1978, Schmitt 1991). These reports were confirmed by negative findings on angiogram, Doppler ultrasound and at autopsy, verifying the lack of arterial damage (Schmitt 1991). In addition, vertebral artery injuries can occur from normal physiological movements of the cervical spine such as spontaneous or sustained rotation, extension or flexion, that is in the absence of applied external forces or trauma (Easton and Sherman 1977, Hanus et al 1977, Nagler 1973, Okawara and Nibbelink 1974, Sherman et al 1992). These cases indicate that vertebrobasilar complications can occur from minor damage or minor trauma.

A number of mechanisms of vertebrobasilar insufficiency have been proposed to explain those cases where no vertebral artery injury was evident, excessive external force was not used, or when damage appeared to be minor or "subclinical". These mechanisms include vasospasm (Schmitt 1991), haemostasis from interruption of blood flow, and "subclinical" endothelial injury from either contusion of the vertebral artery wall, ie subintimal haematoma or turbulent flow (Fast et al 1987, Farag et al 1988, Lord 1986, Parent et al 1992).

*Vasospasm* from cervical manipulation, as a hypothesised mechanism of vertebral artery occlusion, is often suggested (Easton and Sherman 1977, Fast et al 1987, Horn 1983, Kanshepolki et al 1972, Schmitt 1991) but is usually diagnosed by exclusion of other mechanisms (Braakman and Penning 1971, Jentzen et al 1987). Vasospasm in the vertebral arteries has been observed following cervical spine trauma (Sceric et al 2000) and anterior scalene syndrome (Apsatarov et al 1988, Nagasawa et al 1982) and is known to produce cerebral ischaemia in humans (Nagasawa et al 1982, Vaccaro et al 1998). It has also been induced experimentally in the coronary artery of pigs (Kuga et al 1993) and human cerebral and vertebral arteries post-mortem (Charpie et al 1994). The experimentally-induced spasm caused intramural haematoma and dissection of sufficient intensity and duration to occlude the artery and produce ischaemic myocardial infarction in pigs (Kuga et al 1993). Gentle irritation of the endothelium has also been shown to induce vertebral artery spasm that can occlude the vertebral artery and cause thrombosis in humans (Charpie et al 1994).

In addition, vasospasm can be caused by potent vasoconstricting substances released from blood elements, vessel walls and nerves during trauma and ischaemic episodes (Pollanen et al 1992), and can cause turbulence that results in thrombosis (Fuster et al 1988). Permanent

symptoms can result from the effects of vasospasm initiated by transient compression or stretch of the vertebral artery (Vaccaro et al 1998). Despite removal of the deforming stimulus, the diameter of the vertebral artery lumen may remain reduced from the initial vasospasm and cause slowing or turbulence of blood flow and ultimately thrombosis (Smith and Esteridge 1962). Thus, although currently unproven in vivo, vasospasm following cervical manipulation can theoretically cause complications and, if collateral circulation is inadequate, infarction of the hindbrain can result (Caplan 1986).

*Haemostasis*, a temporary cessation or slowing of blood flow, has been proposed as a cause of thrombosis. The implication of the slower blood flow is that the haemostasis would cause ischaemia and, in turn permanent damage in brain tissue. Blood exhibits viscous behaviour as a consequence of there being a suspension of corpuscles (Chong et al 1994). The corpuscular nature becomes important when the diameter of the artery is small compared with the size of the corpuscles. The vertebral artery is commonly a narrow artery, and the diameter is further decreased when stretched, such as during cervical rotation (Seric et al 2000). Furthermore, blood viscosity increases as blood flow decreases, as may occur with narrowing of the vertebral artery, thereby promoting haemostasis (Seric et al 2000). During haemostasis, platelets aggregate and adhere to the vessel wall (Buonanno and Toole 1981) and this is presumed to cause thrombosis (Caplan and Sergay 1991, Gutowski et al 1992), although this process has not been demonstrated in vivo (Mueller and Sahs 1976).

Based on observations at surgery and of ischaemia induced by application of a sphygmomanometer, arterial blood flow can be interrupted for prolonged periods (> 3 hours) without formation of thrombi (Klenerman et al 1982). Such interruptions are far in excess of the average 102ms duration of a cervical spine manipulation, suggesting that the movement during manipulation could not interrupt blood flow for sufficiently long periods for thrombus propagation. The sustained position of the cervical spine in preparation for the manipulation interrupts flow for longer periods but not for prolonged periods of more than three hours. Thus perfusion is unlikely to be threatened.

*Endothelial injury* may result from minor trauma to the vertebral artery. Contusion from compression or kinking of the vertebral artery during manipulation is possible, resulting in denuding of the endothelium and exposure of the highly thrombogenic subendothelial cells, which then promote thrombosis (Dunne et al 1987).

#### **Risk factors for vertebrobasilar complications from manipulation and non-traumatic events**

It is clear that vertebrobasilar complications with or without obvious pathology can arise from cervical manipulation and other non-traumatic events. The problem facing physiotherapists is identification of those individuals at greatest risk. The factors believed to increase

the risk include: anatomical anomalies of the vertebral arteries; prior vertebral artery injury; previous ischaemic episodes; and other factors such as smoking or use of oral contraceptives (Haldeman et al 1999).

*Anatomical anomalies of the vertebral artery* The anatomy of the vertebral arteries and the adjacent structures varies widely between and within individuals (Fisher et al 1961, Johnson et al 2000, Schneider and Schemm 1961). There are a number of anomalies thought to increase the risk of complication from cervical manipulation. Such anomalies include those that increase mechanical deformation of the vertebral artery or render deformation critical, such as hypoplastic vertebral arteries, and anomalies in the course of the vertebral arteries such as a posterior origin from the subclavian artery or absence of connecting arteries in the Circle of Willis (Lord 1986).

*Hypoplasia of the vertebral arteries:* Asymmetry of the vertebral arteries was considered to be a major predisposing factor for vertebrobasilar complication from cervical manipulation (Jentzen et al 1987, Lyness and Wagman 1974). However, the vertebral arteries were also considered to be normally asymmetrical (~92% of subjects: Stopford 1916, Lord 1986), although recent work on larger samples suggests that they are more commonly asymmetrical in females (Macchi et al 1996) or more specifically in white females (Mitchell and McKay 1995). It is more likely, therefore, that hypoplasia, rather than asymmetry as such, increases the risk of complications. Hypoplasia, defined as a vertebral artery diameter of  $\leq$  degrees 2mm in the neutral head position, is very uncommon, being present in 2% (Lovrencic-Huzjan et al 1999) to 6% of the population (Lord 1986, Simeone and Lyness 1976).

The internal diameter of the vertebral artery is normally decreased by cervical rotation (Learoyd and Taylor 1966) causing a reduction in blood flow in healthy controls (eg Licht et al 1998, Refshauge 1994, Stevens 1991), and subjects with symptoms of vertebrobasilar insufficiency (Kuetter et al 1997, Rivett et al 1999). Adequate cerebral perfusion is usually maintained (Rivett et al 2000), however, probably because a large decrease in blood flow (70-80%) is necessary to materially reduce cerebral perfusion, and because collateral circulation is generally adequate (May et al 1963). Hypoplastic vertebral arteries are of concern, because a contralateral hypoplastic vertebral artery may be completely occluded during rotation or an ipsilateral hypoplastic vertebral artery may be unable to provide adequate collateral flow in the case of contralateral vertebral artery occlusion. This critical reduction may occur more readily in individuals with hypoplastic or small vertebral arteries (Barton and Margolis 1975, Selecki 1969). Importantly, in several reports of vertebral artery damage following cervical manipulation, the uninjured vertebral artery was hypoplastic or poorly filling (Parkin et al 1978, Schmitt 1991), suggesting an association between vertebrobasilar complications and inadequate collateral circulation.

*Prior vertebral artery injury* Occasionally, complications arise from manipulation after many previously uneventful manipulations, possibly due to prior vertebral artery trauma. If the tunica intima or media of the vertebral artery are traumatically compromised, they are thought to be more susceptible to further injury (Frumkin and Baloh 1990, Krueger and Okazaki 1980). The evidence for this proposal is indirect: at autopsy following fatal vertebrobasilar complications from manipulation, both old and new thrombi were found in the vertebral arteries (Frumkin and Baloh 1990). The old thrombus was thought to have formed from an earlier dissection, arising either spontaneously or from previous manipulation, and the new thrombus from the recent fatal manipulation (Laterra et al 1988). This could indicate that a previous vertebral artery injury increases the risk of serious vertebrobasilar complications from even minor trauma, and also that injury following manipulation could be subclinical and remain undetected.

One difficulty for physiotherapists in recognising vertebral artery injury such as dissection is that the signs and symptoms are similar to those for mechanical non-specific neck pain, that is, symptoms of headache and pain and stiffness in the cervical spine (Frumkin and Baloh 1990, Silbert et al 1995, Schievink 2001). For such a clinical presentation, intervention with spinal manual therapy might appear to be appropriate. Currently available clinical tests cannot clearly differentiate between these conditions (Silbert et al 1995, Schievink 2001). It is strongly recommended, therefore, that if any doubt exists about the nature of a clinical presentation, vigorous manual procedures should be avoided until either the diagnosis is definitive or gentle manual therapy has proven effective. The therapist must be certain that the presentation is clearly and unambiguously mechanical non-specific neck pain or cervicogenic headache before proceeding with such procedures. Since diagnosis cannot be established with certainty at the first treatment, it is probably prudent to demonstrate improvement with gentle techniques over one or two treatment sessions before progressing to more vigorous procedures such as manipulation. Provocation of symptoms of vertebrobasilar insufficiency following manipulation could indicate that trauma to the vertebrobasilar system has occurred.

*Previous ischaemic symptoms:* Previous ischaemic symptoms such as dizziness from head movements or manipulation may indicate marginal hindbrain perfusion and are strongly associated with subsequent stroke from manipulation, (Kanshepolksi et al 1972). Fifty per cent of subjects who sustained vertebrobasilar complications, particularly hindbrain stroke from manipulation, reported previous ischaemic symptoms (Miller and Burton 1974, Patijn 1991, Sullivan 1992). A history of ischaemic symptoms therefore appears to be a predictor of risk and an unequivocal contra-indication against further manipulation, unless the dizziness can be attributed with certainty to an unrelated benign disorder such as postural hypotension.

*Summary:* The factors that appear to predispose

individuals to risk of vertebrobasilar complications from either manipulation or minor trauma include hypoplastic vertebral arteries, previous ischaemic symptoms and prior vertebral artery injury. There is no evidence that hypermobility of the cervical spine, decreased vessel wall compliance or other factors, such as decreased compliance of arterial walls, increases the risk of complications. Other factors believed to increase the risk of complications, including hypertension, consumption of oral contraceptives and smoking, have been shown not to increase the risk (Haldeman et al 1999).

### **Do the Clinical Guidelines for Pre-Manipulative Procedures for the Cervical Spine identify individuals at risk?**

The Clinical Guidelines for Pre-Manipulative Procedures for the Cervical Spine (Magarey et al 2000) aim firstly, to determine the cause of symptoms potentially associated with vertebrobasilar insufficiency and secondly, to identify patients at risk of complications from manipulation. The guidelines therefore recommend screening any patient with symptoms of vertebrobasilar insufficiency, and also every patient prior to each manipulation. The guidelines recommend taking a history about the symptoms of vertebrobasilar insufficiency and testing, at a minimum, sustained end of range rotation to both sides and the position or movement that provokes the patient's symptoms.

The guidelines can partially fulfil these two aims. Differential diagnosis of most symptoms of vertebrobasilar insufficiency is made largely on the basis of the behaviour of the symptoms and other findings in the history. Few diagnoses require further definitive testing, such as audiology for Meniere's disease or magnetic resonance imaging for acoustic neuroma. The physical tests can add information to assist diagnosis, although little is known about the sensitivity and specificity of rotation for most of the differential diagnoses.

The history recommended in the guidelines may also assist in the identification of patients at risk from manipulation, if information about prior vertebral artery injury and previous ischaemic symptoms is weighted appropriately by the therapist. However, the physical tests recommended in the guidelines cannot identify the likelihood of major vertebral artery injury from the force or speed used in a manipulative thrust, although they may be able determine the adequacy of blood supply to the brainstem and may identify risk from minor trauma. Rotation with or without extension of the cervical spine tests total blood flow to the hindbrain from all the extracranial vessels, not the vertebral arteries in isolation, and thus assesses whether the total flow is marginal in the test positions. Since markedly reduced blood flow in the vertebral arteries during rotation and extension is almost assured (16 of 20 studies showed a decrease with rotation with or without extension: Table 1), these movements test whether collateral circulation could maintain hindbrain perfusion in the event of vertebral artery occlusion during manipulation. It should be noted

**Table 1.** Effect of the test positions used in the APA pre-manipulative test protocol on vertebral artery blood flow.

Test Movement	Effect on flow in VAs	Population	Authors
ipsilateral and contralateral rotation	↓↓ or absent		Stevens 1984
contralateral rotation	↓ in 62% of subjects ↓ in 20% of subjects		Stevens 1991
contralateral rotation	ISQ		Weingart & Bischoff 1992
contralateral rotation	↓↓	2 subjects with VBI	Brautaset 1992
ipsilateral and contralateral rotation	↓ in basilar artery	subjects with hypoplasia	Hedera et al 1993
contralateral rotation	↑ at 45° rotation ↓ at end of range	healthy controls	Refshauge 1994
ipsilateral and contralateral rotation	ISQ at 30° and 60° rotation	healthy controls	Simon et al 1994
extension + contralateral rotation	↓ (L) VA ↓ (R) VA The ↓ in (L) VA significantly greater in dizzy subjects than healthy controls. ↓ in (R) VA same in dizzy and control subjects.	i) subjects with symptoms of neurovascular ischaemia ii) healthy controls	Theil 1994
ipsilateral and contralateral rotation	↓	subjects with degenerative cervical spine changes	Olszewski et al 1994
contralateral rotation	↓	subjects with neck-related symptoms	Haynes 1995
ipsilateral and contralateral rotation	↓	healthy controls	Rossiti & Volkmann 1995
contralateral rotation	↓↓ or absent. (Also absent at 45° in 2 subjects)	subjects with neck-related symptoms	Haynes 1996
contralateral side flexion	↓ in 5%, ISQ in 95%		
extension + rotation	↓↓ or absent (same effect in healthy and dizzy patients)	i) subjects with symptoms of neurovascular ischaemia ii) healthy controls	Coté et al 1996
contralateral rotation	↓↓	subjects with symptoms of VBI	Kuether et al 1997
neutral position after lateral flexion manipulation to C1-C5	ISQ	young subjects with stiff cervical segments	Licht et al 1998
contralateral rotation	↓	healthy controls	Licht et al 1998
ipsilateral rotation	↑		
ipsilateral and contralateral rotation after manipulation	volume blood flow ISQ volume blood flow ISQ	young subjects with stiff cervical segments	Licht et al 1999
ipsilateral rotation	↓	i) subjects with symptoms of neurovascular ischaemia	Rivett et al, 1999
contralateral rotation	↓	ii) healthy controls	
extension	↑ (same effect in healthy and dizzy patients)		
ipsilateral and contralateral rotation	↓↓	healthy young and elderly controls	Li et al 1999
extension	↓↓ (right VA ↓ > left)		
extension + rotation	↓↓ (contralateral ↓ > ipsilateral)		
contralateral rotation	↓	subjects with neck-related symptoms	Haynes 2000

Rotation significantly decreased velocity of blood flow in the vertebral arteries (VAs) in 13 of the 15 studies to investigate this position. Extension increased velocity in one study and decreased it in another. The combination of extension and rotation markedly decreased blood flow in all three studies that investigated this position. Arrows denote direction of altered velocity (increase or decrease). No change is denoted by ISQ (in status quo). All studies measured velocity of blood flow except Licht et al (1999) who measured volume of blood flow, ie quantity of blood rather than velocity.

that although both increased and decreased velocity indicate narrowing of the artery (Seric et al 2000), cerebral haemodynamics are more complicated and are influenced by arterial pressure, intracranial pressure, blood viscosity, vessel stenosis, collateral circulation and cerebral autoregulation (Licht et al 1999, Seric et al 2000). In fact, Rivett et al (2000) presented preliminary evidence that decreased or absent flow is not highly correlated with presence of symptoms of vertebrobasilar insufficiency.

The guidelines could also indicate the likelihood of complications arising from mechanisms other than those caused by major vertebral artery trauma. The mechanisms of minor endothelial injury, haemostasis and vasospasm might not require substantial force or speed, being provoked instead by the gentler stress of sustained positions of the cervical spine with consequent haemodynamic changes. The guidelines may identify these individuals, although the tests should be performed with great care, being especially vigilant for provocation of symptoms of vertebrobasilar insufficiency.

It should be noted that since sustained rotation or extension of the cervical spine can cause vertebrobasilar complications (Parkin et al 1978), the guidelines themselves may not be without risk. However, the positions are sustained for only 10 seconds, and it appears that occlusion needs to be for substantially longer periods to cause ischaemic damage (Klenerman et al 1982), and therefore the risk is probably small. Careful monitoring of symptoms during performance of the tests should minimise the risks.

## Conclusion

The importance to physiotherapists of vertebrobasilar complications arising from minor trauma, or in the absence of injury to the vertebral arteries, is that episodes can potentially occur with forces smaller than those used in gentle manipulation. This emphasises the caution that should be applied during cervical manipulation to minimise the chance of vertebral artery injury.

*Examination:* It is not currently possible to predict every individual at risk of vertebrobasilar complication from manipulation (Terrett 1987), and there is no guarantee that negative test results from performance of the current guidelines preclude vertebral artery injury (Hildebrandt 1979, Rivett 1998, Rivett et al 2000). The guidelines only test the ability of the vertebrobasilar system to maintain adequate hindbrain perfusion in the positions of cervical rotation and extension and this may be an indicator of the patient's likelihood of survival if cervical manipulation injures the vertebral artery. We therefore recommend that the physical tests in the guidelines be performed prior to every manipulation, but therapists must be aware that this is for reasons other than risk assessment. In addition, we strongly agree with the new guidelines that manipulation be avoided in the presence of dizziness or if other risk factors such as a history of previous ischaemic episodes exist.

*Performance of manipulation:* Although complications can occur spontaneously or from the use of trivial force, they are also commonly associated with manipulation. Of particular note is that the combination of speed and force of the manipulation appears to provide the greatest risk. Thus, to minimise risk, it would be prudent for practitioners to employ the smallest force required to achieve the therapeutic objective, as far from end of range as is achievable, and as a progression of effective manual treatment.

**Authors** Kathryn Refshauge, School of Physiotherapy, The University of Sydney, PO Box 170, Lidcombe, New South Wales 1825. E-mail: k.refshauge@cchs.usyd.edu.au (for correspondence). Timothy Mann, Corrimal Physiotherapy, 55 Railway St, Corrimal, New South Wales 2518.

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