

# MANAGEMENT OF VERTEBRAL ARTERY INJURIES FOLLOWING NON-PENETRATING CERVICAL TRAUMA

## RECOMMENDATIONS

### Diagnostic

Standards: There is insufficient evidence to recommend diagnostic standards.

Guidelines: There is insufficient evidence to recommend diagnostic guidelines.

Options: Conventional angiography or magnetic resonance angiography (MRA) is recommended for the diagnosis of vertebral artery injury (VAI) after nonpenetrating cervical trauma in patients who have complete cervical spinal cord injuries, fracture through the foramen transversarium, facet dislocation, and/or vertebral subluxation.

### Treatment

Standards: There is insufficient evidence to recommend treatment standards.

Guidelines: There is insufficient evidence to recommend treatment guidelines.

Options:

- Anticoagulation with intravenous heparin is recommended for patients with vertebral artery injury who have evidence of posterior circulation stroke.
- Either observation or treatment with anticoagulation in patients with vertebral artery injuries and evidence of posterior circulation ischemia is recommended.
- Observation in patients with vertebral artery injuries and no evidence of posterior circulation ischemia is recommended.

## RATIONALE

The association of cerebrovascular insufficiency and cervical fracture was first described by Suechting et al in a patient with Wallenburg's syndrome occurring four days after a C5-C6

fracture-dislocation injury.(13) Although Schneider et al implicated vertebral artery injury at the site of cervical fracture dislocation as a cause of posterior circulation cerebral ischemia (9), Gurdjian et al suggested that unilateral vertebral artery occlusions might be asymptomatic.(4) Subsequent articles described larger series of patients with asymptomatic vertebral artery injuries (VAI) after blunt cervical spinal trauma.(2,18) However, Biffl et al, in the largest prospective series consisting of 38 patients with VAI diagnosed by angiography, reported more frequent strokes in patients not treated initially with intravenous heparin anticoagulation despite an asymptomatic VAI.(1) Fractures through the foramen transversarium, facet fracture-dislocation, or vertebral subluxation are almost always seen in patients with VAI.(1,2,3,5,17,18,19) A cadaveric study demonstrated progressive vertebral occlusion with greater degrees of flexion-distraction injury, confirming this clinical observation.(11) In order to develop diagnostic and treatment recommendations for VAI after blunt cervical trauma, an analysis of the articles examining its management is undertaken in this report. Specific questions that were addressed include: the clinical and radiographic criteria used to prompt diagnostic evaluation, appropriate diagnostic tests for identifying VAI, and the management of VAI (observation versus anticoagulation with heparin).

## **SEARCH CRITERIA**

A National Library of Medicine computerized literature search of publications from 1966 to 2001 was performed using the following headings: vertebral artery, cervical vertebrae, dislocation, and wounds and injuries. An exploded search of the headings led to 6,447, 15,667, 24,174 and 459,759 citations respectively. The first two headings were combined, and a subset of 61 citations was also contained in the third heading. A subset of 239 citations was also

contained in the fourth heading. Abstracts were reviewed and only articles in English containing three or more human subjects with vertebral artery injury after blunt cervical trauma were included. Fourteen articles, including eight articles that were prospective studies (1,2,3,5,10,16,17,18), met these selection criteria, and provided data on 122 patients for this report. These articles are summarized in Evidentiary Table format.

## **SCIENTIFIC FOUNDATION**

### **Diagnosis**

The diagnosis of VAI can be made with a variety of radiographic studies. Angiography is the traditional imaging technique utilized to diagnose VAI and was used for all patients in seven of the studies reviewed (1,5,7,14,15,18,19) and in combination with other modalities in two additional studies.(6,8) Three studies prospectively applied conventional angiography to patients sustaining nonpenetrating cervical trauma that met certain inclusion criteria.(1,5,18) Similarly, three studies prospectively applied magnetic resonance angiography to patients sustaining nonpenetrating cervical trauma that met certain inclusion criteria.(2,16,17)

Biffel et al reported the largest prospective study using angiography, selecting patients from 7205 blunt trauma victims using clinical and radiographic criteria.(1) “Symptomatic” patients were selected for angiography if they had facial hemorrhage (bleeding from mouth, nose, ears), cervical bruit (in those younger than 50 years of age), expanding cervical hematoma, cerebral infarction by computed tomography (CT), or lateralizing neurological deficit. “Asymptomatic” patients were selected for angiography if they had cervical hyperextension/rotation or hyperflexion injuries, closed head injury with diffuse axonal injury, near hanging, seat belt or other soft tissue injuries to the neck, basilar skull fractures extending

into the carotid canal, and cervical vertebral body fractures or distraction injuries. Between 350 and 400 angiograms were performed, identifying 38 patients with VAI. However, neither the exact number of angiograms performed nor the number of patients meeting the various criteria without VAI were reported. As a result, sensitivity, specificity, positive predictive value, and negative predictive value of the selection criteria could not be determined. Cervical spine injuries were observed in 27 of 38 patients with VAI, including fractures through the foramen transversarium in four, dislocations in six, vertebral subluxations in two, and more than one of these injuries in two. Twenty-nine patients had unilateral VAI (18 left, 11 right); nine had bilateral VAI. A vascular injury scale was used to stratify patients into five categories:

- Grade I - arterial dissections less than 25% luminal narrowing,
- Grade II - arterial dissections with more than 25% luminal narrowing,
- Grade III - pseudoaneurysm of the vertebral artery,
- Grade IV - occlusion of the vertebral artery,
- Grade V - vertebral artery transection.

Seven patients died; five had bilateral VAI (Grade I), while two had unilateral VAI (one Grade I, one Grade IV). Three patients with either no neurological deficit or mild deficit had bilateral VAI. The authors concluded that stroke incidence and neurological outcome appeared independent of the grade of vertebral artery injury.

Another prospective study by Willis et al identified 30 patients with midcervical fractures and/or dislocation for angiography.(18) However, only 26 patients meeting the criteria agreed to proceed with angiography. Twelve patients sustained VAI demonstrated by angiography (six left occlusion, three right occlusion, one left intimal flap, one left pseudoaneurysm, one left dissection). The authors provided sufficient data regarding the presence of foramen

transversarium fracture, facet dislocation, and subluxation to determine the utility of these radiographic findings in identifying patients with VAI. The calculated sensitivity of foramen transversarium fracture as a criterion for identifying VAI in this study was 58%. The specificity was 36%. The positive predictive value of foramen transversarium fracture was 44%. The negative predictive value was 50%. The calculated sensitivity of facet dislocation as a criterion for identifying VAI was 42%. The specificity was 57%. The positive predictive value of facet dislocation was 45%; the negative predictive value was 53%. The calculated sensitivity of subluxation as a criterion for identifying VAI was 67%; the specificity was 29%. The positive predictive value of subluxation was 80%; the negative predictive value was 50%. Any combination of foramen transversarium fracture, facet dislocation, and/or vertebral subluxation revealed a calculated sensitivity for identifying VAI of 92% and a specificity of 0%. The positive predictive value of the presence of any of the three criteria and VAI was 44%. The negative predictive value was 0%.

A third prospective study by Louw et al examined 12 consecutive patients with cervical spine facet dislocations with digital subtraction angiography.(5) Five of seven patients with bilateral facet dislocations had vertebral artery occlusion (one bilaterally), and four of five patients with unilateral facet dislocations had unilateral vertebral artery occlusion. Angiography was not performed in blunt cervical trauma patients without facet dislocation. In a retrospective study by Woodring et al (19), seven of eight patients with transverse process fractures undergoing angiography had VAI (two right occlusions, two left occlusions, two right dissections, one left dissection). Seventy-eight percent of transverse process fractures extended into the foramen transversarium. Angiography was not performed in 44 other patients with transverse process fractures. Alternatively, magnetic resonance angiography (MRA) has been

used to diagnose VAI noninvasively. Weller et al prospectively examined twelve patients with nonpenetrating cervical trauma who sustained fractures through the foramen transversarium.(17) Three patients had unilateral vertebral artery occlusion and one had focal narrowing, all at the site of fracture. MRA was not performed on the twenty-six patients without these fractures. Giacobetti et al prospectively evaluated all patients admitted with cervical spine injuries with MRA.(3) Twelve of 61 patients had vertebral artery occlusion demonstrated by MRA and all injuries were unilateral (six left, six right). Although seven of twelve patients with VAI had flexion-distraction injuries with facet dislocations, the types of cervical spinal injuries sustained by the 49 patients with normal MRA were not reported. Since none of these four articles provided sufficient information regarding the types of injury and results of vertebral artery imaging in the entire population of patients studied, sensitivity, specificity, positive predictive value, and negative predictive value of the injury types could not be determined. (3,5,17,19)

Friedman et al prospectively examined 37 patients admitted with “major” blunt cervical spine injuries using MRA and compared these patients with a size-matched control group of patients without a history of cervical trauma.(2) Nine patients had VAI (six unilateral occlusion, two narrow, one bilateral injury). Both vertebral arteries were visualized in all 37 control subjects. Complete spinal cord injuries were observed in 12 of 37 patients with cervical trauma, six of whom had VAI ( $p < 0.02$ ; chi-square test). More than three millimeters of subluxation was observed in 13 of 37 patients, five of whom had VAI ( $p < 0.14$ ; chi-square test). The calculated sensitivity of complete spinal cord injury as a criterion for identifying VAI in this study was 67%, whereas the specificity was 79%. The positive predictive value of complete spinal cord injury was 50%. The negative predictive value was 88%. The calculated sensitivity of

subluxation as a criterion for identifying VAI was 56%. The specificity was 71%. The positive predictive value of subluxation was 38%, whereas the negative predictive value was 83%.

Other diagnostic modalities have also been used to identify of VAI. CT with intravenous contrast demonstrated a unilateral vertebral artery occlusion in one patient with a Jefferson fracture, which was subsequently confirmed by angiography.(12) Duplex sonography has also been used to diagnose of VAI.(8,10,14) Angiography has occasionally been used to confirm the results of MRA or ultrasonography, but there has not been a study comparing MRA or ultrasonography with angiography in the diagnosis of VAI.

## **Treatment**

After diagnosis of VAI, treatment options examined by the various studies have included observation alone or anticoagulation with either intravenous heparin or oral antiplatelet agents. Some authors treated asymptomatic patients (1,2,18,19); others did not treat symptomatic patients. (5,7)

Several articles retrospectively identified patients with neurological complications of VAI. Schellinger et al described four patients with VAI who had delayed onset of neurological dysfunction.(8) Two patients awoke after surgery with altered consciousness and posterior circulation stroke. One of these patients with vertebrobasilar occlusion diagnosed by ultrasound died; the other with vertebral artery dissection and pseudoaneurysm improved. Neither patient was anticoagulated. Two other patients developed delayed symptoms; one 12 hours after injury, the other three weeks after injury. The first patient died from basilar artery thrombosis confirmed by CT. The second patient developed vertigo and nystagmus due to a dissection diagnosed by ultrasound and angiography. This patient was successfully treated with

intravenous heparin anticoagulation and recovered within several weeks. Thibodeaux et al reported one patient who incurred immediate blindness after left vertebral artery dissection.(14) No infarction was seen on CT. Treatment included three months of anticoagulation with sustained improvement at four-year follow-up. A second patient with ataxia and dizziness two days after VAI had occipital infarction by CT and bilateral vertebral artery dissections, recovered with six months of anticoagulation therapy. One patient with dissection remained asymptomatic without anticoagulation. Prahbu et al reported three patients of five patients with VAI who experienced sudden loss of consciousness three hours after injury.(6) All were anticoagulated. One patient with vertebrobasilar thrombosis died; one patient with bilateral vertebral occlusion improved. The third patient with vertebral artery stenosis improved, although anticoagulation was stopped several days later secondary to a rectus sheath hematoma. Two patients with asymptomatic vertebral artery occlusion were not treated and remained asymptomatic. Tulyapronchote et al reported three patients with delayed onset of symptoms two weeks to three months after VAI, including syncope, visual disturbance, dysarthria, dysphagia, and vertigo.(15) Two patients had vertebral artery occlusions and one had a dissection. No treatment was reported for any of these patients. Woodring et al reported two strokes in nine patients with transverse process fractures after blunt cervical trauma.(19) All nine were studied with vertebral angiography; seven studies were abnormal. One patient with vertebral artery occlusion was treated with anticoagulation and improved. One with dissection improved without treatment. Three of five asymptomatic patients were anticoagulated (intravenous heparin converted to Coumadin, total treatment of six weeks); two patients were not treated. All five remained asymptomatic. Schwarz et al reported four patients with ischemic vertebrobasilar symptoms after non-penetrating cervical trauma.(7) Two patients with atlantoaxial injuries recovered

following atlantoaxial stabilization (one treated with halo immobilization, one operatively). Treatment of one patient with delayed symptoms after unrecognized facet dislocation was not reported. Streptokinase infusion was used in a patient with vertebral artery occlusion who became comatose shortly after reduction of a facet dislocation injury. This strategy failed to achieve complete thrombolysis and the patient died days later.

Six prospective studies examining the diagnosis of VAI provided the incidence of neurological complications related to VAI. Biffi et al (1) reported the highest frequency of posterior circulation stroke, occurring in nine of 38 patients (24%). Stroke occurred eight hours to twelve days after injury in these patients, with 78% occurring more than 48 hours after injury. Three of 21 asymptomatic patients treated with intravenous heparin subsequently developed stroke; one died and two had mild residual deficits. In contrast, six of 17 asymptomatic patients not initially treated with intravenous heparin developed stroke; two died, three had mild deficits, and one had a severe deficit. Neither of the two patients who died were treated with anticoagulation when stroke developed nine to twelve days after VAI (one Grade IV, one bilateral Grade I which progressed to right Grade II and left Grade III). All three asymptomatic patients who subsequently developed stroke and were then treated with heparin had mild residual deficits (one Grade I, one Grade II, one Grade IV). A final patient who developed stroke and was treated with antiplatelet agents had severe residual deficits (Grade I). Two of nine patients with stroke who were treated with heparin developed hemorrhagic infarction. Two of the 38 patients had dominant vertebral arteries; both had injuries to the dominant artery and neither developed a stroke.

Weller et al (17) treated all four patients in their report with vertebral artery injuries with aspirin. Three patients with vertebral artery occlusion remained asymptomatic; one with

vertebral artery narrowing developed syncope 17 days after injury. This patient was treated with intravenous heparin followed by aspirin without recurrent symptoms. Giacobetti et al (3) described three of twelve patients with vertebral artery occlusion who developed transient blurred vision; all three were treated with three months of anticoagulation (intravenous heparin followed by Coumadin), and none had recurrent symptoms. Friedman et al (2) reported one patient with bilateral vertebral artery injuries who died after a massive right cerebellar infarct. One patient with vertebral occlusion and concurrent carotid occlusion remained asymptomatic on heparin. The remaining seven patients remained asymptomatic without treatment (five occlusions, two stenosis). Willis et al (18) reported no symptoms in twelve patients with VAI, none of whom were treated with anticoagulation. Louw et al (5) reported two of nine patients with vertebral artery occlusion with symptoms including blurring of vision which spontaneously resolved without anticoagulation.

The management and outcome of 106 patients with VAI after nonpenetrating cervical injury could be determined from the articles reviewed. Twelve patients had radiographic evidence of a posterior circulation stroke as their first symptom. Four patients were treated with intravenous heparin; one died and three improved.(1,19) The remaining eight patients were not treated with intravenous heparin; five died, two improved, and one had a severe neurological deficit.(1,2,8,19) Fifteen patients developed symptoms of posterior circulation ischemia without stroke before treatment was instituted.(3,5,6,7,8,14,17) Eleven of 15 patients were treated with intravenous heparin; two died (both had strokes) and nine improved (one developed a stroke). The remaining four patients were not treated with intravenous heparin; all improved without developing a posterior circulation stroke. Twenty-seven asymptomatic patients were prophylactically treated with intravenous heparin.(1,2,18,19) Three patients developed posterior

circulation stroke; twenty-seven patients remained asymptomatic. Finally, fifty-two asymptomatic patients were not prophylactically treated with heparin (eight were treated with aspirin and one with embolization). Three patients died from injuries unrelated to the VAI. (1,18) Four patients had severe deficits (three were treated with aspirin and one with embolization) and three had mild deficits (one was treated with aspirin); all were related to the associated spinal cord injury.(1) The remaining 42 patients (three of whom were treated with aspirin) remained asymptomatic.(1,2,3,5,6,14,17,18,19)

The articles reviewed did not specifically address the risk of progressive spinal cord hemorrhage worsening in patients with VAI and an associated spinal cord injury treated with heparin. One patient with a complete cervical spinal cord injury and hematomyelia was placed on intravenous heparin prophylactically for left carotid and vertebral artery occlusion; no neurological changes occurred with treatment.(2)

At least 13 of 42 patients treated with intravenous heparin had complications during their treatment, six of which were significant (14%). Six patients (two of whom died) developed posterior circulation strokes after treatment with intravenous heparin was initiated.(1,6,7,14) Two patients developed hemorrhagic posterior circulation strokes; the timing of intravenous heparin relative to the development of the posterior circulation stroke was not reported.(1) Intravenous heparin was discontinued in three patients. One symptomatic patient developed a rectus sheath hematoma (6); the patient's symptoms stabilized after intravenous heparin treatment was discontinued. Two asymptomatic patients had progression in the grade of vertebral artery injury; both remained asymptomatic after intravenous heparin was discontinued. (18) Four other patients had progression in the grade of vertebral artery injury; intravenous

heparin was continued and none of the patients developed deficits related to the progression of injury grade.(1)

In several studies patients were re-imaged to determine whether disease progression or resolution occurred after vertebral artery injury. Biffi et al reported follow-up angiography on 21 patients.(1) Of 16 patients treated with heparin, two improved to a lesser grade of vascular injury and four worsened to a poorer grade. Of five patients not receiving heparin, one improved and three had worse vascular injury grades. Vaccaro et al found reconstitution in one of six vertebral artery injuries by MRA twelve days after the original diagnosis; this patient was not treated with anticoagulation.(16) The remaining five still had vertebral artery occlusion more than one year later, including two treated with anticoagulation. Willis et al described the results of follow-up angiography in three patients with VAI.(18) One patient with a pseudoaneurysm received one week of intravenous heparin followed by aspirin; the pseudoaneurysm had slightly enlarged seven days after treatment was begun, but had disappeared on angiography performed six weeks later. One patient treated with intravenous heparin for a vertebral artery dissection had an asymptomatic occlusion of the artery demonstrated by angiography two days later; the heparin was discontinued. The third patient was treated with intravenous heparin for a vertebral artery intimal flap; the patient had a normal vertebral angiogram ten days later. Thibodeaux et al found a patent vertebral artery six months after dissection was diagnosed; this patient was not anticoagulated.(14) Sim et al reported delayed Duplex sonography in eleven patients with a history of facet dislocation, but unknown vertebral artery status at the time of the original cervical spine injury.(10) Two of these studies demonstrated VAI: one with persistent dislocation had vertebral occlusion, and one patient with a reduced injury had vertebral artery stenosis.

## SUMMARY

The incidence of vertebral artery injury may be as high as 11% after non-penetrating cervical spinal trauma in patients with specific clinical criteria, including facial hemorrhage (bleeding from mouth, nose, ears), cervical bruit in those younger than 50 years of age, expanding cervical hematoma, cerebral infarction by CT, lateralizing neurological deficit, cervical hyper-extension/rotation or hyperflexion injuries, closed head injury with diffuse axonal injury, near hanging, seat belt or other soft tissue injuries to the neck, basilar skull fractures extending into the carotid canal, and cervical vertebral body fractures or distraction injuries. Many patients with VAI have complete spinal cord injuries, fracture through the foramen transversarium, facet dislocation, and/or vertebral subluxation, but many patients with these injuries have normal vertebral arteries when imaged, thus compromising specificity of these injury criteria. Many patients with VAI are asymptomatic, including those with vertebral artery occlusion or dissection. The literature reviewed indicates that patients with posterior circulation stroke and VAI have a better outcome when treated with intravenous heparin compared to those who are not. However, others have reported improvement among patients without anticoagulation.(8,19) The outcome of patients who develop symptoms of posterior circulation ischemia without stroke treated with intravenous heparin (3,6,8,14,17) is similar to those patients receiving no treatment.(5,7) Although the largest prospective study suggested a trend toward less frequent stroke in asymptomatic patients treated with heparin (1), others have not reported similar observations.(2,3,5,6,14,18,19) Since the risk of significant complications related to anticoagulation is approximately 14% in these studies, there is insufficient evidence to recommend anticoagulation in asymptomatic patients.

## **KEY ISSUES FOR FUTURE INVESTIGATION**

Although several prospective studies have been reported examining patients at risk for VAI, most articles did not report sufficient data about the characteristics of the patients with normal vertebral arteries to examine clinical or radiographic characteristics to predict which patients may have VAI. A prospective study comparing MRA with conventional angiography in nonpenetrating cervical spine trauma may define the role of non-invasive imaging studies in these patients. A multicenter, randomized, prospective study comparing anticoagulation with intravenous heparin with observation in asymptomatic patients and in symptomatic patients with posterior circulation ischemia but without stroke is recommended to determine whether anticoagulation of these patients is justified.

## EVIDENTIARY TABLES

First Author Reference	Description of Study	Data Class	Conclusions
Schellinger PD et al, 2001, <i>Spine</i>	Retrospective review of 4 patients with cervical spine injury of 27 patients with vertebral artery dissection diagnosed with various imaging studies	Class III	2 with motor complete cord injuries awoke after cervical surgery with stupor and posterior circulation infarcts; one with basilar occlusion by Doppler died and one with dissection by DSA partially recovered. One with C5 foramen trans. fx became comatose 12 hrs after admission and had fatal basilar thrombosis by CT hrs later. One with treated C2 fx had vertigo & nystagmus 3 wks later; dissection by angio; sx's resolved with Coumadin followed by ASA.
Biffi WL et al, 2000, <i>Ann Surg</i>	Prospective angiography screening in blunt craniovertebral injury identified 38 pts with vertebral injuries	Class III	38 pts with 47 VI out of approx 350 angios done. 27 of 38 had cervical fxs. 6 of 27 had fxs thru foramen transv. 9 of 38 post circ stroke. Stroke not related to occlusion v. stenosis 8hr-12 day delay, most beyond 48 hr 3 of 21 asymptomatic patients treated with heparin had stroke vs. 6 of 17 w/o heparin had stroke (p=0.13) 1 of 6 died w/ heparin. 2 of 3 died w/o heparin. 2 treated with heparin had hemorrhagic strokes.
Weller SJ et al, 1999, <i>J Trauma</i>	Prospective MRA in 12 pts with for. transv. fx	Class III	3 of 12 had VA occlusion; all remained asymptomatic on ASA. 1 of 12 with stenosis had delayed syncope on ASA, resolved with brief IV heparin followed by ASA.
Vaccaro AR et al, 1998, <i>Spine</i>	Prospective study with F/U MRA in 6/12 pts (1 excluded) previously reported by Giacobetti FB et al (3)	Class III	1 of 6 treated w/o heparin reconstituted by 12 days. 5 of 6 remained occluded (2 of 5 w/ heparin) >1 yr later.
Giacobetti FB et al, 1997, <i>Spine</i>	Prospective study with MRA in 61 pts with cervical injuries found 12 pts with vert occlusion	Class III	1 of 4 with transv fx had occl, 6 of 15 with facet disloc had occl. 3 of 12 with transient blurred vision resolved with 3mo anticoagulation.
Thibodeaux et al, 1997, <i>Br J Surg</i>	Retrospective review of 3 pts with vertebral dissection on angiography	Class III	1 with blindness but no infarct improved with anticoagulation. 1 with C4-5 facet fx was asymptomatic w/o treatment. 1 with ataxia/dizziness 2 days later with occipital infarct recovered with 6 mo anticoagulation.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Prahbu V et al, 1996, <i>J Trauma</i>	Retrospective review of 5 symptomatic pts with 4 vertebral artery occlusion and 1 stenosis on MRA and/or angiography	Class III	3 with fxs delayed coma at 3 hrs, 2 occl/1 narrow, 1 died with stroke, 2 improved with anticoag but 1 d/ced 2° rectus hematoma. 1 with fx delayed confusion/aphasia with multiple MCA strokes improved w/o anticoag 1 with fx asymptomatic w/o anticoagulation.
Friedman D et al, 1995, <i>Am J Roentgen</i>	Prospective study of 37 pts with nonpenetrating cervical trauma found 9 vertebral injuries by MRA	Class II	50% patients w complete cord injuries had vert injury vs 12% pts w incomplete cord injuries (p<0.02). 5 of 13 patients w >3mm subluc had vertebral injuries vs 4 of 24 patients w <3mm subluc. 1 w bilat vert injuries died of large cerebellar infarct (bilat for. trans. fx) 8 asymptomatic (1/8 anticoag also had carotid occlusion).
Tulyapronchote R et al, 1994, <i>Neurology</i>	Retrospective study of 3 pts with VB ischemia >2 wks after occult C2 fractures diagnosed by angiography	Class III	2 occluded, 1 narrow. Sxs included syncope, vertigo, dysphagia, dysarthria, facial numbness, blurred vision. Treatment not reported.
Willis BK et al, 1994, <i>Neurosurgery</i>	Prospective study of 26 pts with cervical facet dislocation or facet fx thru for transv. fx revealed 12 with vertebral injuries on angiography	Class II	9 of 14 with normal angio had for. transv. fx vs. 7 of 12 with abnormal angio. 1 of 9 with occlusion w/o anticoag died from unrelated injuries. 1 dissection became an occlusion on heparin. 1 intimal flap/1 pseudoaneurysm healed with heparin in 7-10 days.
Sim E et al, 1993, <i>Acta Orthop Scand</i>	Prospective delayed Duplex sonography of 11 pts with previously locked facets	Class III	1 of 11 had occlusion (persistent locked facet). 1 of 11 had a narrow vertebral artery.
Woodring JH et al, 1993, <i>J Trauma</i>	Retrospective study of 216 pts with cervical fxs showed 52 with TP fxs, 8 had angiography	Class III	78% of TP fxs extended into for. transv. 4 of 8 had occlusion, 3 of 8 had dissection, 1 of each had stroke that improved with anticoag. 3 asymptomatic treated with anticoagulation.
Schwarz N et al, 1991, <i>J Trauma</i>	Retrospective review of 4 pts with symptomatic vertebral injuries by angiography	Class III	4 delayed sxs (3 occlusions, 1 pseudoaneurysm). 1 with coma and stroke 20 min after reduction of C4-5 dislocation received Streptokinase/heparin for occlusion but died.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Louw JA et al, 1990, <i>J Bone Joint Surg</i>	Prospective study of 12 pts with facet dislocations with digital subtraction angiography	Class III	4 of 5 unilateral and 5 of 7 bilateral dislocations had occluded vert (1 bilat). 3 occluded at level, 5 within 2 cm. 2 of 9 symptomatic patients had bilat C5-6 facet disloc and improved w/o treatment.

## REFERENCES

1. Biffl WL, Moore EE, Elliot JP, Ray C, Offner PJ, Franciose RJ, Brega KE, Burch JM. Ann Surg 231:672-681, 2000.
2. Friedman D, Flanders A, Thomas C, Millar W. Vertebral artery injury after acute cervical spine trauma: rate of occurrence as detected by MR angiography and assessment of clinical consequences. Am J Roentgen 164:443-447, 1995.
3. Giacobetti FB, Vaccaro AR, Bos-Giacobetti MA, Deeley DM, Albert TJ, Farmer JC, Cotler JM. Vertebral artery occlusion associated with cervical spine trauma. A prospective analysis. Spine 22:188-192, 1997.
4. Gurdjian ES, Hardy WG, Lindner DW, Thomas LM. Closed cervical trauma associated with involvement of carotid and vertebral arteries. J Neurosurg 20:418-427, 1963.
5. Louw JA, Mafoyan NA, Small B, Naser CP. Occlusion of the vertebral artery in cervical spine dislocations. J Bone Joint Surg 72B:679-681, 1990.
6. Prabhu V, Kizer J, Pail A, Hellbusch L, Taylon C, Leibrock L. Vertebrobasilar thrombosis associated with nonpenetrating cervical spine trauma. J Trauma 40:130-137, 1996.
7. Schwarz N, Buchinger W, Gaudernak T, Russe F, Zechner W. Injuries to the cervical spine causing vertebral artery trauma: case reports. J Trauma 31:127-133, 1991.
8. Schellinger PD, Schwab S, Krieger D, Fiebach JB, Steiner T, Hund EF, Hacke W, Meinck HM. Masking of vertebral artery dissection by severe trauma to the cervical spine. Spine 26:314-319, 2001.
9. Schneider RC, Crosby EC, Russo RH, Gosch HK. Traumatic spinal cord syndromes and their management. Clin Neurosurg 20:424-492, 1973.

10. Sim E, Schwarz N, Biowski-Fasching I, Biowski P. Color-coded duplex sonography of vertebral arteries. *Acta Orthop Scand* 64:133-137, 1993.
11. Sim E, Vaccaro AR, Berzlanovich A, Pienar S. The effects of staged static cervical flexion-distraction deformities on the patency of the vertebral arterial vasculature. *Spine* 25:2180-2186, 2000.
12. Song WS, Chiang YH, Chen CY, Lin SZ, Liu MY. A simple method for diagnosing traumatic occlusion of the vertebral artery at the craniovertebral junction. *Spine* 19:837-839, 1994.
13. Suechting RL, French LA. Posterior inferior cerebellar artery syndrome following a fracture of the cervical vertebra. *J Neurosurg* 12: 187-189, 1955.
14. Thibodeaux LC, Hearn AT, Peschiera JL, Deshmukh RM, Kerlakian GM, Welling RE, Nyswonger GD. Extracranial vertebral artery dissection after trauma: a five year review. *Br J Surg* 84:94, 1997.
15. Tulyapronchote R, Selhorst JB, Malkoff MD, Gomez CR. Delayed sequelae of vertebral artery dissection and occult cervical fractures. *Neurology* 44:1397-1399, 1994.
16. Vaccaro AR, Klein GR, Flanders AE, Albert TJ, Balderston RA, Cotler JM. Long-term evaluation of vertebral artery injuries following cervical spine trauma using magnetic resonance angiography. *Spine* 23:789-795, 1998.
17. Weller SJ, Rossitch E, Malek AM. Detection of vertebral artery injury after cervical spine trauma using magnetic resonance angiography. *J Trauma* 46:660-666, 1999.
18. Willis BK, Greiner F, Orrison WW, Benzel EC. The incidence of vertebral artery injury after midcervical spine fracture or subluxation. *Neurosurg* 34:435-442, 1994.

19. Woodring JH, Lee C, Duncan V. Transverse process fractures of the cervical vertebrae: are they insignificant? J Trauma 34:797-802, 1993.