Radiologic Evaluation of Chronic Neck Pain
American Family Physician

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FROM ABSTRACT:

This article summarizes the American College of Radiology Appropriateness Criteria for chronic neck pain.

Imaging plays an important role in evaluating patients with chronic neck pain.

Five radiographic views (anteroposterior, lateral, open-mouth, and both oblique views) are recommended for all patients with chronic neck pain with or without a history of trauma.

Magnetic resonance imaging should be performed in patients with chronic neurologic signs or symptoms, regardless of radiographic findings.

Patients with normal radiographic findings and no neurologic signs or symptoms, or patients with radiographic evidence of spondylosis and no neurologic findings, need no further imaging studies.

THIS AUTHOR ALSO NOTES:

1) This study considers two etiologies of chronic neck pain:

A)) Posttraumatic: includes gross injuries and whiplash syndrome.

B)) Degenerative: includes spondylosis, degenerative disk disease, and acute disk herniation; degeneration may also be secondary to previous injury.

2) Spondylosis is radiologically diagnosed when osteophytes, disk space narrowing, or facet disease are present.

3) The American College of Radiology Appropriateness Criteria has produced the optimal imaging study for every clinical scenario.

4) There is "little correlation between the presence of cervical spondylosis or degenerative disk disease and the severity or duration of symptoms."

5) "Although spondylosis and disk disease increase with age and are usually asymptomatic, whiplash can accelerate these processes and lead to symptoms."

6) A long-term (minimum 10 years follow-up) study in 2009 demonstrated that progressive degenerative changes on MRI are not associated with clinical symptoms, and "the authors concluded that there is no statistically significant association between MRI findings and changes in clinical symptoms." **[Important]**

7) MRI is useful in documenting disk herniations, canal encroachment by osteophytes, tumor, infection, fractures, and posttraumatic ligament ruptures of the lower cervical column.

8) "Although MRI does not always detect the cause of chronic neck pain, particularly at the craniocervical junction, it is the preferred method for making most diagnoses."

9) Guidelines from the American College of Radiology Appropriateness Criteria for the evaluation of patients with chronic neck pain (regardless of the etiology) include the following:

- A five-view radiographic examination (i.e., anteroposterior, lateral, open-mouth, and both oblique views) should initially be performed in patients of any age with chronic neck pain with or without a history of remote trauma, with a history of malignancy, or with a history of neck surgery in the remote past.

- Patients with chronic neck pain and normal radiographic findings, and no neurologic signs or symptoms need no further imaging.

- Patients with chronic neck pain and normal radiographic findings, and neurologic signs or symptoms should undergo MRI.

- Patients with chronic neck pain and whiplash-associated disorders should "undergo MRI to evaluate for disk herniations, spur encroachment of the vertebral canal, or ligament abnormalities of the lower cervical region."

- If MRI is contraindicated (e.g., in patients with a cardiac pacemaker or severe claustrophobia), CT myelography with multiplanar reconstruction is recommended.

- "Patients with radiologic evidence of cervical spondylosis or a previous trauma without neurologic signs or symptoms need no further imaging."

- "Patients with radiographic evidence of cervical spondylosis or previous trauma and neurologic signs and symptoms should undergo MRI. If MRI is contraindicated, CT myelography is recommended."

- "Patients with radiographic evidence of bone or disk margin destruction should undergo MRI. If an epidural abscess is suspected, the examination should be performed with intravenous contrast media. CT is indicated only if MRI cannot be performed."

- "Facet injections and arthrography are useful for patients with multilevel disease diagnosed by any imaging modality to identify the specific disk level that is producing symptoms."

- "Diskography is not recommended in patients with chronic neck pain."

KEY POINTS FROM DAN MURPHY:

1) All chronic neck pain patients, with or without a history of trauma, should have 5 radiographic views (anteroposterior, lateral, open-mouth, and both oblique views).

2) All chronic neck pain patients with neurologic signs or symptoms should have an MRI, regardless of radiographic findings.

3) It is inappropriate to ascribe a whiplash-injured patient's chronic neck symptoms to pre-accident degenerative disease (spondylosis).

4) Depending upon the circumstances, if one did not follow these Guidelines from the American College of Radiology Appropriateness Criteria for the evaluation of patients with chronic neck pain, it is possible that one might be accused of practicing below the standard.

5) On the other hand, if an insurance company, reviewer, or examining board claimed that radiographs were not indicated in a chronic neck pain patient, one could use this study to argue for their necessity.

6) [Additionally, I would suggest that all acute traumatic neck pain patients have x-rays to rule-out fracture. I also suggest that all acute traumatic and chronic neck pain patients have maximum flexion-extension x-rays to evaluate for segmental instability].

Kinematic Cervical Spine Magnetic Resonance Imaging in Low-Impact Trauma Assessment

Seminars in Ultrasound, CT, and MRI
June 2009; Volume 30; Number 3; pp. 168-173

Vincenzo Giuliano, MD, Antonio Pinto, MD, PhD, and Mariano Scaglione, MD
Dr. Giuliano is from Nova Southeastern University, College of Medicine, Winter Springs, Flordia.

FROM ABSTRACT

Kinematic magnetic resonance imaging can be implemented as a noninvasive adjunct examination for injuries in the cervical spine in the clinical assessment of ligamentous, disk, and soft-tissue injuries, as a basis for determining medical vs. surgical management, and in establishing the degree of functional clinical impairment.

THESE AUTHORS ALSO NOTE:

"The cervical spine is particularly susceptible to acceleration and deceleration injuries resulting from impact trauma."

"Low-impact collisions result in acceleration and deceleration of the head and neck, also known as whiplash."

In the biomechanics of whiplash the cervical spine forms an S-shaped curve, with hyperflexion in the upper cervical spinal segments, and simultaneous hyperextension in the lower cervical spinal segments.

"Approximately 60% of whiplash injuries are occult to magnetic resonance imaging (MRI) and include occult soft-tissue, intervertebral disk, and ligamentous injuries, accounting for approximately 90% of injuries missed by MRI."

"Cervical instability is defined as angular motion greater than 11 degrees, or translation of greater than 3 mm, for contiguous spinal segments."

"The optimal period for performing evaluation of the cervical spine using kinematic MRI methods is 12 weeks post-injury, following resolution of muscle spasm."

"Initial radiographic series should include the anteroposterior and lateral flexion/extension views. The most common finding is straightening of the cervical spine, with either loss or reversal of the normal lordotic curve."

"MRI is clinically indicated in the setting of persistent arm pain, neurologic deficits, and clinical signs of nerve root compression."

"MRI offers the best noninvasive and detailed evaluation of the intervertebral disks, soft-tissue structures, and spinal cord but is considered unreliable in the detection of subtle annular disk tears."

"Hyperflexion injuries can evade radiologic detection."

"Kinematic MRI provides the most optimal means of detecting subtle hyperflexion injuries and annular disk tears, in addition to evaluating segmental spinal motion and cervical lordosis patterns."

"Kinematic MRI, in contradistinction to other imaging methods, such as lateral flexion/extension radiographs and videofluoroscopy, provides accurate assessment of spinal canal stenosis."

"Clinical criteria for kinematic MRI evaluations include the persistence of signs and symptoms during the subacute period, including localized neck pain and radiculopathy, despite clinically resolved muscle spasm.

"The kinematic MRI evaluation is typically coordinated with manipulative therapy and rehabilitation programs. **[Important]**

The kinematic MRI protocol should be performed as an additional sequence following the static cervical MRI examination.

"The sagittal T2 fast-spin-echo (FSE) scan sequence is the most optimal imaging parameter and provides the most accurate and reliable diagnostic information in distinguishing soft-tissue contrast between aqueous structures, such as nucleus pulposus and cerebral spinal fluid, from ligamentous structures."

Typical normal, non-injured findings with kinematic cervical spine MRI:

1) A stepwise segmental motion starting at C1-C2 and extending to the lower cervical spinal segments in a coordinated and orderly pattern.

2) A lordotic cervical curve.

3) "Hypolordosis with normal segmental motion is generally observed in 4%-7% of cases, representing a normal variant."

4) A fanlike and unrestricted motion of the spinous processes is.

5) Between 45-60 degrees of cervical spine flexion.

6) Between 50-70 degrees of cervical spin extension.

7) Small asymptomatic bulging disks in 2% of patients.

Kinematic cervical spine MRI evaluations in injured subjects usually reveal:

1) Injury to the joint capsule, interspinous/supraspinous ligaments, and ventral annulus fibrosus.

2) The posterior longitudinal ligament is intact.

3) "Hypolordosis is invariably present, with notable segmental motion restriction characterized by an absence of the normal fanlike movements of the spinous processes of C4 through C7."

4) "Flexion appears disproportionally restricted compared to extension, with exacerbation of symptoms, including headache, arm pain, and arm numbness."

The majority of whiplash-injured patients improve within 8-12 weeks."

20%-70% of whiplash-injured patients remain symptomatic at 6 months.

When imaging findings include disk herniations, spinal stenosis that can be increased with flexion, hypolordosis and segmental motion restriction and fixation, the recovery period is longer, with the majority achieving maximum improvement 36 weeks following injury. **[9 months]**

"Maximum medical improvement of all whiplash injuries is generally achieved within 2 years."

CONCLUSIONS

"Kinematic MRI evaluations of the cervical spine can provide a valuable adjunct method to the standard static cervical spine MR examination."

"Kinematic MRI is clinically indicated in patients with whiplash injuries with 1 or more persistent neurologic deficits or clinical signs and symptoms beyond the normal and expected recovery period, generally within 8-12 weeks."

KEY POINTS FROM DAN MURPHY

1) Kinematic magnetic resonance imaging can be implemented as a noninvasive adjunct examination for injuries in the cervical spine in the clinical assessment of ligamentous, disk, and soft-tissue injuries.

2) "The cervical spine is particularly susceptible to acceleration and deceleration injuries resulting from impact trauma."

3) "Low-impact collisions result in acceleration and deceleration of the head and neck, also known as whiplash."

4) "Approximately 60% of whiplash injuries are occult to magnetic resonance imaging (MRI) and include occult soft-tissue, intervertebral disk, and ligamentous injuries, accounting for approximately 90% of injuries missed by MR."

5) "Cervical instability is defined as angular motion greater than 11 degrees, or translation of greater than 3 mm, for contiguous spinal segments."

6) "The optimal period for performing evaluation of the cervical spine using kinematic MRI methods is 12 weeks post-injury, following resolution of muscle spasm."

7) "Initial radiographic series should include the anteroposterior and lateral flexion/extension views. The most common finding is straightening of the cervical spine, with either loss or reversal of the normal lordotic curve."

8) "MRI is clinically indicated in the setting of persistent arm pain, neurologic deficits, and clinical signs of nerve root compression." "Clinical criteria for kinematic MRI evaluations include the persistence of signs and symptoms during the subacute period, including localized neck pain and radiculopathy, despite clinically resolved muscle spasm.

9) "MRI offers the best noninvasive and detailed evaluation of the intervertebral disks, soft-tissue structures, and spinal cord but is considered unreliable in the detection of subtle annular disk tears."

10) "Kinematic MRI provides the most optimal means of detecting subtle hyperflexion injuries and annular disk tears, in addition to evaluating segmental spinal motion and cervical lordosis patterns."

11) Kinematic MRI provides accurate assessment of spinal canal stenosis.

12) "The kinematic MRI evaluation is typically coordinated with manipulative therapy and rehabilitation programs. **[Important: they advocate manipulation]**

13) The kinematic MRI protocol should be performed as an additional sequence following the static cervical MRI examination.

14) Typical normal, non-injured findings with kinematic cervical spine MRI:

A)) A stepwise segmental motion starting at C1-C2 and extending to the lower cervical spinal segments in a coordinated and orderly pattern.

B)) A lordotic cervical curve.

C)) "Hypolordosis with normal segmental motion is generally observed in 4%-7% of cases, representing a normal variant."

D)) A fanlike and unrestricted motion of the spinous processes is.

E)) Between 45-60 degrees of cervical spine flexion.

F)) Between 50-70 degrees of cervical spine extension.

G)) Small asymptomatic bulging disks in 2% of patients.

15) Kinematic cervical spine MRI evaluations in injured subjects usually reveal:

A)) Injury to the joint capsule, interspinous/supraspinous ligaments, and ventral annulus fibrosus.

B)) The posterior longitudinal ligament is intact.

C) "Hypolordosis is invariably present, with notable segmental motion restriction characterized by an absence of the normal fanlike movements of the spinous processes of C4 through C7."

D) "Flexion appears disproportionally restricted compared to extension, with exacerbation of symptoms, including headache, arm pain, and arm numbness."

16) The majority of whiplash-injured patients improve within 8-12 weeks."

17) 20%-70% of whiplash-injured patients remain symptomatic at 6 months.

18) When imaging findings include disk herniations, spinal stenosis that can be increased with flexion, hypolordosis and segmental motion restriction and fixation, the recovery period is longer, with the majority achieving maximum improvement 36 weeks following injury. **[9 months]**

19) "Maximum medical improvement of all whiplash injuries is generally achieved within 2 years."

20) "Kinematic MRI evaluations of the cervical spine can provide a valuable adjunct method to the standard static cervical spine MR examination."

21) "Kinematic MRI is clinically indicated in patients with whiplash injuries with 1 or more persistent neurologic deficits or clinical signs and symptoms beyond the normal and expected recovery period, generally within 8-12 weeks."

A case-control study of cerebellar tonsillar ectopia (Chiari) and head/neck trauma (whiplash)

Brain Injury
July 2010; 24(7–8): 988–994

Michael D. Freeman, Scott Rosa, David Harshfield, Francis Smith, Robert Bennett, Chris J. Centeno, Ezriel Kornel, Ake Nystrom, Dan Heffez, Sean S. Kohles

FROM ABSTRACT

Primary objective: Chiari malformation is defined as herniation of the cerebellar tonsils through the foramen magnum, also known as cerebellar tonsillar ectopia.

Cerebellar tonsillar ectopia may become symptomatic following whiplash trauma. The purpose of the present study was to assess the frequency of cerebellar tonsillar ectopia in traumatic vs non-traumatic populations.

Methods and procedures: Cervical MRI scans for 1200 neck pain patients were reviewed; 600 trauma [whiplash] (cases) and 600 non-trauma (controls). Half of the groups were scanned in a recumbent position and half were scanned in an upright position.

Cerebellar tonsillar ectopia was found in 5.7% and 5.3% in the recumbent and upright non-trauma groups vs 9.8% and 23.3% in the recumbent and upright trauma groups.

Conclusions: The results described in the present investigation are first to demonstrate a neuroradiographic difference between neck pain patients with and without a recent history of whiplash trauma.

The results of prior research on psychosocial causes of chronic pain following whiplash are likely confounded because of a failure to account for a possible neuropathologic basis for the symptoms.

KEY POINTS FROM DAN MURPHY

1) Chiari malformation is defined as herniation of the cerebellar tonsils through the foramen magnum, also known as cerebellar tonsillar ectopia.

2) Chiari type I malformation is a caudal herniation of the cerebellar tonsils through the foramen magnum (tonsillar ectopia). It can be acquired.

3) Typical Chiari type I malformation symptoms include occipital headache, neck pain, upper extremity numbness and paresthesias and weakness; occasionally there may be lower extremity weakness and signs of cerebellar dysfunction.

4) "Previously quiescent Chiari Type I malformations can become symptomatic as a result of exposure to traumatic injury." Minor head and neck trauma can cause an asymptomatic Chiari type I malformation into becoming symptomatic.

5) Patients with a history of motor vehicle crash-associated neck pain have a "substantially higher frequency" of cerebellar tonsillar ectopia than non-traumatic subjects; 4-times greater when evaluated with an upright MRI scan.

	600 Chronic Controls	**600 Chronic Whiplash Patients**
<u>Supine</u> MR # cerebellar tonsillar ectopia	5.3%	9.3%
<u>Upright</u> MR # cerebellar tonsillar ectopia	5.7%	23.3%

6) "Cerebellar tonsillar ectopia is substantially more prevalent in whiplash-injured neck pain patients than in neck pain patients with no recent history of trauma."

7) In the trauma group, cerebellar tonsillar ectopia was found 2.5-times more often in the upright MR scan vs the recumbent MR scan. "Upright position MR imaging appears to increase the sensitivity to cerebellar tonsillar ectopia over recumbent MR imaging by 2.5 times."

8) It is well established that Chiari type I can be acquired, and this study led the authors to suggest that the increased incidence of cerebellar tonsillar ectopia was caused by the whiplash trauma.

9) The incidence of cerebellar tonsillar ectopia was nearly identical (5.3% v 5.7%) in the control group in both the supine and upright MRI; but the incidence of cerebellar tonsillar ectopia was significantly greater (23.3 v 9.3) in the whiplash-injured group with the upright MRI. This can be explained by reduced cerebral spinal fluid (CSF) as a consequence of a trauma induced leak.

10) "There is clinical evidence that dural leaks are associated with whiplash trauma and chronic symptoms."

11) 56% of chronic whiplash patients with headache, memory loss, dizziness and neck pain, had cerebral spinal fluid leaks, primarily in the lumbar spine at the dural sleeves. 88% of these patients enjoy substantial improvement in chronic whiplash symptoms with an epidural blood patch to seal the leak. [Epidural blood patch is an the dural leak.]

12) The best method to document cerebral spinal fluid leak is using radioisotope cisternography.

13) Studies show that there is a substantial and rapid increase in cerebral spinal fluid pressure during simulated whiplash trauma.

14) In this study, neuroradiographic abnormality (cerebellar tonsillar ectopia) was found in approximately 25% of upright whiplash trauma cases. This unrecognized definable pathology may account for a patient's chronic pain complaints. This suggests that in these cases, chronic whiplash symptoms may not be ascribable to psychosocial factors or litigation status, but rather to organic neurological injury.

15) "Nearly half of the population with chronic neck pain attribute the onset of their pain to a whiplash trauma-associated injury."

16) Patients with fibromyalgia syndrome also have a higher than expected frequency of Chiari type I malformations. Thus, "cerebellar tonsillar ectopia has been found to be associated with both a history of whiplash trauma and fibromyalgia syndrome." Therefore, both fibromyalgia syndrome and chronic whiplash injury may be secondary to cerebellar tonsillar ectopia, possibly secondary to dural leak.

17) "Clinicians may want to consider evaluating patients for cerebellar tonsillar ectopia (i.e. upright MRI of the neck and head) when there is a history of whiplash trauma and persisting suboccipital headache in combination with headache worsened by cough or bilateral sensory or motor deficits in the upper extremities."

18) "In cerebellar tonsillar ectopia patients with headache that is relieved when supine it also may be appropriate to consider radionuclide cisternography to evaluate for the presence of a dural leak."

COMMENTS FROM DAN MURPHY

In 2005, Tomlinson, Gargan, and Bannister prospectively evaluated whiplash-injured patients for 7.5 years. They found that 23% continued to suffer from intrusive and/or disabling symptoms that required ongoing treatment and investigations, 7.5 years after being whiplash-injured. This present study found 23.3% of the whiplash-injured group showed cerebellar tonsillar ectopia with upright MRI scans. These authors suggest that the 23% incidence in both groups may not be coincidental. This suggests that all chronic whiplash-injured patients should be examined with upright MRI scan to evaluate cerebellar tonsillar ectopia. [PJ Tomlinson, MF Gargan and GC Bannister. The fluctuation in recovery following whiplash injury: 7.5-year prospective review; Injury; Volume 36, Issue 6, June 2005, Pages 758-761].

Dynamic kine magnetic resonance imaging in whiplash patients

Pain Research and Management
2009 Nov-Dec 2009;Vol. 14, No. 6; pp. 427-32

Lindgren KA, Kettunen JA, Paatelma M, Mikkonen RH.

KEY POINTS FROM DAN MURPHY

1) On average, 30% (range 11% to 42%) of people with acute whiplash develop chronic whiplash symptoms.

2) "Injury to the alar ligaments associated with neck sprain could be a cause of pain and disability among these [chronic whiplash] patients."

3) Whiplash injury to the upper cervical spine can cause balance disturbance, dizziness, visual problems and jaw problems.

4) The stability of the cranial-cervical junction is primarily provided by the alar and transverse ligaments.

5) "The alar ligaments restrain rotation of the upper cervical spine."

6) "The alar ligaments may be irreversibly overstretched or even ruptured in unexpected rear-end collisions."

7) Alar ligament integrity can be assessed using high-resolution proton density-weighted dynamic MRI.

8) Chronic whiplash patient symptoms attributable to Occiput -C1-C2, include:
Neck pain
Headache
Upper limb symptoms
Lower limb symptoms
Loss of balance
Some tongue numbness

9) This study found:

	Normal Controls	**Chronic Whiplash Patients**
Abnormal Alar Ligament	24%	92%
Abnormality of Dens Movement	20%	56%
Unstable Occiput-C1 Joint	3%	23%

Transverse Ligament/Medulla Contact	Zero	7%

10) 95% of dens movement abnormalities found were "no movement of the dens." **[Significant chiropractic applications]**

11) "Because of the lack of a disc and the horizontal nature of the facet joints, the stability of the atlanto-axial complex depends mainly on the ligaments and muscles."

12) "The most important function of the alar ligaments is to limit axial rotation of the head."

13) 55% of the rotation of the cervical spine occurs at the C1-C2 joint

14) 5% of the rotation of the cervical spine occurs at the Occiput-C1 joint.

15) 40% of the rotation of the cervical spine occurs at C2-C7.

16) Alar ligaments can be visualized using proton density weighted MRI with slice distances not exceeding 2mm.

17) Chronic whiplash patients have more abnormal signals from the alar ligaments and more movement disturbances at Occiput-C1-C2 in dMRI than control subjects.

18) Studies that use 4mm slice distances often miss alar ligament abnormalities. Slice distances must be no more than 2mm.

19) "Contact between the transverse ligament and the medulla can only be seen during rotation using [dMRI]." Contact between the transverse ligaments and the medulla during rotation "is abnormal."

20) "Symptoms and complaints among WAD patients can be linked with structural abnormalities of the ligaments and membranes of the upper cervical spine, particularly the alar ligaments."

21) "We found that many whiplash patients with persisting disabilities had alar ligament abnormalities, and disturbed function Occiput-C1-C2 complex."

22) "Abnormalities in proprioception and in the upper neural coordination centers produce symptoms similar to those seen in our whiplash patients." Upper cervical spine ligaments could contribute to these symptoms.

What We Have Learned About Vitamin D Dosing?

Integrative Medicine
Vol. 9, No. 1, Feb/Mar 2010

Joseph Pizzorno, ND, Editor in Chief

BACKGROUND FROM DAN MURPHY

The world standard uses nmol/l, while US standard uses mg/dl.

For vitamin D, to convert mg/dl to nmol/l, divide the mg/dl by 2.5.

For vitamin D, to convert nmol/l to mg/dl, just multiply by 2.5.

KEY POINTS FROM THIS ARTICLE:

1) "Over the past several years, the surprising prevalence of vitamin D deficiency has become broadly recognized."

2) Vitamin D deficiency is linked to:
Osteoporosis
Cardiovascular disease
Cancer
Autoimmune diseases
Multiple sclerosis
Pain
Loss of Cognitive function
Decreased strength
Increased rate of all-cause mortality

3) "Deficiency of vitamin D is now recognized as a pandemic, with more than half of the world's population at risk."

4) Approximately 50% of the healthy North American population and more than 80% of those with chronic diseases are vitamin D deficient.

5) 80% of healthy Caucasian infants are vitamin D deficient. [And the rate of vitamin D deficiency tends to be greater in African American and Hispanic children].

6) Those with vitamin D deficiency experience 39% higher annual healthcare costs than those with normal levels of vitamin D.

7) Suggested levels of vitamin D as measured by 25(OH)D3 is:

Caucasians	125 – 175 nmol/l	=	50 - 70 mg/dl
Hispanics	100 – 150 nmol/l	=	40 - 60 mg/dl
African Americans	80 – 120 nmol/l	=	32- 48 mg/dl

8) The minimum blood levels of vitamin D [25(OH)D3] is 80 nmol/l (32 mg/dl).

9) Prolonged intake of 10,000 IU of supplemental vitamin D3 "is likely to pose no risk of adverse effects in almost all individuals."

10) The maximum safe levels for vitamin 25(OH)D3 in the blood is 275 nmol/l (100 mg/dl).

11) Sarcoidosis patients (and other granulomatous diseases) should not supplement with vitamin D because it increases granuloma production increasing the risk of hypercalcemia.

12) A loading dose of supplemental vitamin D3 of 10,000 IU/day for 3 months and maintenance dose of 5,000 IU/day "is not enough for most people in northern climes."

13) The loading dose of supplemental vitamin D3 should be about 20,000 IU/day for 3 – 6 months with a maintenance dose of 5,000 IU/day. Those taking this amount of supplemental vitamin D3 should periodically have their serum 25(OH)D3 levels measured.

COMMENTS FROM DAN MURPHY

The lab we use to test blood vitamin D3 [25(OH)D3] uses a finger prick analysis:
ZRT Laboratory
8605 SW Creekside Pl
Beaverton, OR 97008
866-600-1636
www.zrtlab.com
Vitamin D Testing Finger prick

The vitamin D3 my family takes is ***Complete Hi D3***, from Nutri-West (5,000 IU):
800-443-3333
The primary researcher on this product was Don Bellgrau, PhD. Dr. Bellgrau is a tenured Professor of Immunology and Medicine at the University of Colorado, Denver, where he is a Program Leader in Immunology and Immunotherapy at the Cancer Center on vitamin D3 supplementation. Dr. Bellgrau has conducted experiments with nutrients/vitamin D and immune cells. He has published in over 100 peer-reviewed articles, including the Journal of Neurooncology, Nature, Clinical Immunology, Cancer Research, Cancer Immunology and Immunotherapy, and Cell Transplantation.

Osteoarthritis
Epidemiology, Risk Factors, and Pathophysiology

American Journal of Physical Medicine and Rehabilitation
November 2006, Vol. 85, No. 11, pp. S2-S11

Susan V Garstand, MD and Todd P Stitik, MD
From the University of Medicine and Dentistry of New Jersey

THESE AUTHORS NOTE:

"Osteoarthritis (OA) is the most prevalent form of arthritis and a major cause of disability in people aged 65 and older." OA affects the majority of adults over age 55.

58% of those older than 70 years have symptomatic OA.

10-30% of those with OA have significant pain and disability.

OA is "the clinical and pathologic outcome of a range of disorders that results in structural and functional failure of synovial joints. OA occurs when the dynamic equilibrium between the breakdown and repair of joint tissues is overwhelmed."

The risk of OA has 2 major categories: systemic factors and local factors:

1) Systemic Factors:
A)) Ethnicity
B)) Age: "The presence of radiographic OA rises with age at all joint sites."
C)) Gender
D)) Hormonal Status
E)) Genetic Factors
OA has a major genetic component
F)) Bone Density
G)) Nutritional Factors
There is evidence that OA is linked to free radicals, and that high dietary antioxidants (especially vitamins C and D) are protective against the development of OA. "Chondrocyte senescence is thought to be the result of chronic oxidative stress."

2) Local Factors:
Local factors "result in abnormal biomechanical loading of affected joints."
A)) Obesity
B)) "Altered joint biomechanics"
- ligamentous laxity
- malalignment

- impaired proprioception
  With aging, there is a decline in proprioception, causing decreased neurologic responses, impairing proprioceptive joint-protective mechanisms. Consequently, reduced proprioception advances OA.
- muscle weakness

C)) Prior joint injuries
D)) Occupational Factors
E)) Effects of sports and physical activities
F)) Developmental abnormalities

"If systemic factors are in place, the joint may be thought of as vulnerable, and thus local biomechanical factors will have more of an impact on joint degeneration."

"Injuries that alter mechanical or joint alignment may also predispose individuals to OA at other sites."

"Other risk factors for posttraumatic arthritis include high body mass, high level of activity, and residual joint instability or malalignment."

Obesity increases the risk of OA. Importantly, the increased risk includes joints that are not weight bearing, like hand OA. This suggests that "obesity may predispose to OA, perhaps via an inflammatory or metabolic intermediary." [I suggest prostaglandin E2 (PGE2)]. "This means that obesity plays a role not only as a local process but systemically as well."

Repetitive occupational stresses increase OA.

In the absence of systemic factors, moderate exercise, such as running, does not cause joint degeneration. However, there is increased OA in male runners who exceed more then 20 miles per week.

High-intensity direct joint impact or torsional loading can increase the risk of OA in the affected joint.

Loss of normal joint biomechanics result in increased joint vulnerability to OA.

Joint malalignment, or proprioceptive deficits predispose the joint to the development of OA.

"Impaired proprioception has been seen in patients with OA compared with age-matched controls, which may also indicate that proprioceptive loss preceded disease development."

"Joint immobilization has been shown to be detrimental, reducing cartilage thickness and proteoglycan content."

Intense exercise, especially in the elderly, can accelerate cartilage breakdown and OA.

Muscle weakness predisposes individuals to the development of OA because greater stress loads are borne by the joints, accelerating joint damage.

"Adequate muscle strength and bulk are protective to the joint."

"Cartilage is avascular, and therefore chondrocytes receive nutrients and eliminate waste by diffusion through the synovial fluid and by facilitated imbibition."

Osteoarthritis of a joint typically involves all of these tissues of the synovial joint, including:
1) Articular cartilage
2) Subchondral bone
3) Synovial tissue
4) Ligaments
5) Joint capsules
6) Muscles that cross the joint

A decreased range of joint motion leads to muscle atrophy and loss of joint protection, increasing the risk of OA.

Although OA is considered to be a non-inflammatory arthritis, as cartilage destruction proceeds, mild to moderate inflammatory reactions are found in the synovial membranes.

As the OA catabolic process progresses, the synoviocytes begin to make and release the pro-inflammatory eicosanoid hormone prostaglandin E2 (PGE2). **[Recall that PGE2 is derived from the omega-6 fatty acid arachidonic acid]**

KEY POINTS FROM DAN MURPHY

1) "Osteoarthritis (OA) is the most prevalent form of arthritis and a major cause of disability in people aged 65 and older." OA affects the majority of adults over age 55.

2) OA is "the clinical and pathologic outcome of a range of disorders that results in structural and functional failure of synovial joints. OA occurs when the dynamic equilibrium between the breakdown and repair of joint tissues is overwhelmed."

3) Both systemic factors and local factors will increase the risk of osteoarthritis.

4) Systemic Factors:
A)) Ethnicity
B)) Age: "The presence of radiographic OA rises with age at all joint sites."
C)) Gender

D)) Hormonal Status
E)) Genetic Factors
OA has a major genetic component
F)) Bone Density
G)) Nutritional Factors
There is evidence that OA is linked to free radicals, and that high dietary antioxidants (especially vitamins C and D) are protective against the development of OA. "Chondrocyte senescence is thought to be the result of chronic oxidative stress."

5) Local Factors:
Local factors "result in abnormal biomechanical loading of affected joints."
A)) Obesity
B)) "Altered joint biomechanics"
- •• ligamentous laxity
- •• malalignment
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  With aging, there is a decline in proprioception, causing decreased neurologic responses, impairing proprioceptive joint-protective mechanisms. Consequently, reduced proprioception advances OA.
- •• muscle weakness

C)) Prior joint injuries
D)) Occupational Factors
E)) Effects of sports and physical activities
F)) Developmental abnormalities

5) "If systemic factors are in place, the joint may be thought of as vulnerable, and thus local biomechanical factors will have more of an impact on joint degeneration."

6) "Injuries that alter mechanical or joint alignment may also predispose individuals to OA at other sites."
[Altered alignment or mechanics predispose joints to osteoarthritis]

7) "Other risk factors for posttraumatic arthritis include high body mass, high level of activity, and residual joint instability or malalignment."
[Important: joint instability and malalignment increase risk of OA]

8) Obesity increases the risk of OA in both weight-bearing and non weight-bearing joints. This suggests that "obesity may predispose to OA, perhaps via an inflammatory or metabolic intermediary." **[I suggest prostaglandin E2 (PGE2)]**. "This means that obesity plays a role not only as a local process but systemically as well."

9) Repetitive occupational stresses increase osteoarthritis.

10) High-intensity direct joint impact or torsional loading can increase the risk of OA in the affected joint.

11) Loss of normal joint biomechanics result in increased joint vulnerability to osteoarthritis. **[Important]**

12) Proprioceptive deficits predispose the joint to the development of osteoarthritis.
[This is important because the subluxation is not only a mechanical alignment lesion, but also has associated aberrant proprioception]

13) "Impaired proprioception has been seen in patients with osteoarthritis compared with age-matched controls, which may also indicate that proprioceptive loss preceded disease development."

14) Muscle weakness predisposes individuals to the development of OA because greater stress loads are borne by the joints, accelerating joint damage. "Adequate muscle strength and bulk are protective to the joint."

15) "Cartilage is avascular, and therefore chondrocytes receive nutrients and eliminate waste by diffusion through the synovial fluid and by facilitated imbibition."
[Important: reduced motion impairs joint nutrition, accelerating OA]

16) Both immobilization and decreased range of joint motion leads to muscle atrophy and loss of joint protection, increasing the risk of osteoarthritis.

17) Although OA is considered to be a non-inflammatory arthritis, as cartilage destruction proceeds, mild to moderate inflammatory reactions are found in the synovial membranes.

18) As the OA catabolic process progresses, the synoviocytes begin to make and release the pro-inflammatory eicosanoid hormone prostaglandin E2 (PGE2).
[Recall that PGE2 is derived from the omega-6 fatty acid arachidonic acid]

COMMENTS FROM DAN MURPHY

For decades, at least since the Renaissance seminars in the 1970s (Flesia and Riekeman), chiropractors have maintained that the spinal subluxation accelerated spinal joint degeneration and osteoarthritis. Components of the subluxation include altered alignment, altered movement, muscle atrophy, reduced range of joint motion and aberrant proprioception. These components of the subluxation are the same factors that this article associates with an increased risk of osteoarthritis. This supports the teachings of the Renaissance seminars of the 1970s, and the phases of subluxation degeneration. It supports the contention that uncorrected subluxations predispose those joints to osteoarthritis.

Whiplash Injury
30-Year follow-up of a single series

**Journal of Bone and Joint Surgery - British Volume,
Volume 92-B, Issue 6, pp. 853-855**

J. Rooker, M. Bannister, R. Amirfeyz, B. Squires, M. Gargan, G. Bannister

KEY POINTS FROM DAN MURPHY

1) This is the longest study of whiplash-injured patients that I have seen in the PubMed database: a 30-year follow-up study. The results are:

Group A	Group B	Group C	Group D
Asymptomatic	Mild symptoms Not interfering with work or leisure activities	Intrusive Symptoms Handicapping work and leisure activities Patient using drugs, physical therapy, orthoses for symptoms	Severe Symptoms Causing patients to lose their jobs and rely continually on drugs, orthoses and repeated medical consultations
45%	40.9%	9.1%	4.6%

2) Between 15.5 year and 30 years, neck disability had improved in 45.5% of patients, remained the same in 45.5% of patients, and deteriorated in 9.1% of patients.

3) 45.5% of whiplash-injured patients had made a full recovery 30 years after being injured.

4) About 15% of whiplash-injured patients have significant symptoms and impairments 30 years after being injured.

5) Although most whiplash-injured patient will have reached maximum improvement by 2 years after injury, this study shows that some (9.1%) are continuing to deteriorate 30 years after being injured.

6) At two years after injury, about 50% of whiplash-injured patients are completely recovered, and about 50% will have ongoing symptoms. 4.5% of whiplash-injured patients will suffer from severe symptoms 2 years after injury.

7) Whiplash-injured patients with a disability often develop an abnormal psychological profile.

8) Age related spinal degeneration advances with age. In this study, 30 years after being injured, 91% of the whiplash-injured patients either improved or remained the same between 15 and 30 years after being injured. This suggests that chronic whiplash symptoms are not linked to age-related advancing of spinal degeneration.

COMMENTS FROM DAN MURPHY

Once again, this study shows that a significant number of those injured in whiplash trauma will suffer with chronic symptoms. Thirty years after being injured:
45% are completely recovered
40% retain nuisance symptoms
15% have significant symptoms and impairments, requiring ongoing treatment

Additionally:
Psychological distress is common in the chronic group.
It is not unusual for maximum improvement to take 2 years.
Most patients with chronic symptoms at 2 years will continue to have chronic symptoms 30 years later.

Chiropractic Manual Intervention in Chronic Adult Dyspepsia

European Journal of Gastroenterology & Hepatology
April 2009, 21:482–486

Martin F. Young, Peter W. McCarthy and Susan King,
Martin Young is a chiropractor from the UK

THESE AUTHORS NOTE:

"Your readership may be interested in the results of a pilot study investigating traditional but unproven chiropractic intervention in patients presenting with symptoms of functional dyspepsia, opening the possibility that manual therapy (MT) may have a role to play in the management of this condition."

This prospective cohort study involved 83 consecutive patients with symptoms of digestive pain in the central chest or epigastric area of more than 2 years' duration.

"Patients were managed conservatively using spinal manipulative therapy and soft tissue techniques. Exercise and other home-based treatment elements were not included in treatment protocols."

71% of the patients reported an improvement in the average severity of their symptoms.
29% of the patients remained unchanged.
No patients reported any worsening of symptoms.
45% reduced their use of dyspepsia drugs.

This study "seem to indicate that chiropractic management can have a highly significant positive impact on gastro-esophageal reflux disorder symptoms, with the majority of patients reporting decreased frequency and severity of symptoms and many being able to reduce or eliminate their requirement for medication."

DISCUSSION

Chronic dyspepsia has a low incidence of self-resolution and a natural history of deterioration.

"This pilot study demonstrates that patients with a clinical complaint of dyspepsia might benefit from conservative chiropractic management in terms of both symptomatic relief and decreased use of palliative pharmacological interventions."

COMMENTS FROM DAN MURPHY: This study adds to the evidence of chiropractic benefit in the management of non-musculoskeletal visceral disorders.

"Whiplash" Injury of the 2nd Cervical Ganglion and Nerve

Canadian Journal of Neurological Sciences
1986, Vol. 13, pp. 133-137

William S Keith
From the Department of Neurosurgery, Toronto Western Hospital

FROM ABSTRACT:

Amongst the many patients with persisting neck pain and headache following cervical injuries are a small number in whom the mechanism is compression of the second cervical nerve root and ganglion.

The main features are unilateral pain in the upper cervical and occipital region, tenderness in the suboccipital region, and diminished sensation in the C2 dermatome.

KEY CONCEPTS FROM THIS AUTHOR:

1) Extension injuries of the neck following motor vehicle accidents are common even with the universal extension of the seat back to protect the neck.

2) 10-15% of whiplash extension injured patients "continue to have persisting symptoms after time intervals which should be sufficient for recovery and after litigation settlements have been completed."

3) It has been known since 1949 that the C2 nerve root is vulnerable to compression between the lamina of the atlas and the axis, especially in extension.

4) Crushing injury of the C2 nerve and/or ganglion is a common cause of persisting occipital and posterior cervical pain following neck injuries.

5) In reviewing 14 patients with this syndrome, this author reports:

- On average, it takes 20 months after injury before the correct diagnosis made.
- 14% of patients with this injury will be permanently disabled.
- The pain is confined to the ipsilateral upper neck and occipital region.
- Some patients may additionally have pain around the eye and face.
- Pain is often aggravated by unguarded or sudden movements of the head.

- Described sensations at the time of injury include dazed, shaken up, disoriented, dizzy, and a blinding or explosive feeling.

- All patients had marked tenderness on deep palpation of the suboccipital region.

- All patients had diminished sensation to pin prick and touch in the C2 dermatome. [A paradox: decreased superficial sensation in the same area that has increased pain to deep digital pressure]

- Patients rarely complain of numbness.

6) The author reviews dissections from clinical anatomist (and physician) Nikoli Bogduk, and includes his own photographs of dry specimens of articulated C1-C2, to make these points:

- The C2 dorsal root ganglion (DRG) and nerve root lie against the C1-C2 joint capsule.

- The most vulnerable part of the C2 nerve root to compression is the dorsal root ganglion because it is the "thickest neural structure."

- It is nearly impossible to compress the C2 DRG in extension unless there is also rotation.

- It is impossible to compress the LEFT C2 DRG if the head is rotated to the LEFT prior to extension mechanism. When the head is rotated LEFT, the space between the lamina of C1 and the lamina of C2 on the LEFT is actually increased, offering more protection for the C2 DRG and root.

- Consequently, when the head is rotated LEFT, the vulnerable C2 DRG and nerve root is on the RIGHT. In LEFT rotation, "with sudden unexpected hyperextension in this position of the head on the neck, the C2 ganglion and nerve may be crushed on the right side, but not on the left."

7) This C2 DRG or root compression syndrome has been called:
- Occipital neuralgia
- Occipital neuritis
- Cervical migraine
- Occipital migraine

8) A review of 200 whiplash injury cases showed:
- Women have more whiplash neck symptoms than men.
- Many patients continue to have symptoms after their legal cases have ended.

9) "If the C2 ganglion [DRG] is contused there may be sufficient scarring to account for continuing symptoms over a longer period of time." **[Important: post-traumatic scar tissue to a nerve root can cause chronic symptoms]**

10) If the injury to the C2 DRG is slight, "there are no symptoms during quiescent periods, but pain is provoked by the slightest insult." **[Important]**

11) The anatomical studies of Bogduk "showing the relationship of the atlas to axis in various positions, clearly demonstrates the vulnerability of the C2 nerve root and ganglion to hyperextension injury."

12) Some patients with this syndrome will require a surgical decompression by root avulsion or laminectomy or adhesion resection.

COMMENT FROM DAN MURPHY:

In my experience, many patients with this syndrome are completely or greatly improved with upper cervical chiropractic care.

Benign paroxysmal positional vertigo following whiplash injury: a myth or a reality?

American Journal of Otolaryngology
September 9, 2010 [epub]

Francesco Dispenza MD, Alessandro De Stefano MD, Navneet Mathur MS, Adelchi Croce MD and Salvatore Gallina MD, PhD

FROM ABSTRACT

The aim of the study was to evaluate the true incidence, diagnosis, and treatment of benign paroxysmal positional vertigo (BPPV) arising after whiplash injury and to distinguish this type of posttraumatic vertigo from other types of dizziness complained after trauma.

Eighteen whiplash patients who had BPPV were evaluated. In 16 cases [89%], the posterior semicircular canal was involved; the lateral semicircular canal was involved in 2 cases [11%].

BPPV was the cause of vertigo in 34% of total whiplash patients.

The Dizziness Handicap Inventory score improved in all patients treated with canalith repositioning maneuvers.

The diagnosis of posttraumatic BPPV is not different from the idiopathic form, but the treatment may require more maneuvers to achieve satisfactory results.

KEY POINTS FROM AUTHORS:

1) Equilibrium is the result of a perfect integration of input from eyesight, cervical spine proprioceptive receptors, and labyrinths.

2) "Balance problems affect 5% to 50% of patients of whiplash injury."

3) 15% to 20% of whiplash-injured patients develop late whiplash syndrome with chronic complaints including headache, vertigo, instability, nausea, and tinnitus.

4) Cervical trauma may increase the discharge of neck muscles' proprioceptors, interfering with normal afferent input into the vestibular system, resulting in cervicogenic vertigo. **[These patients will not have a positive Dix-Hallpike test and may experience vertigo without moving the head]**.

5) Whiplash trauma causes labyrinthine vertigo in 25% of subjects.

6) Whiplash trauma causes auditory disturbances in 17% of subjects.

7) "The incidence of dizziness with even mild head injury ranges from 15% to 78%."

8) Benign paroxysmal positional vertigo (BPPV) is the most frequent cause of peripheral vertigo, accounting for 24% of all cases.

9) Most cases of BPPV are idiopathic; however trauma is a known cause.

10) Most BPPV involves the posterior semicircular canal.

11) Classic BPPV is "set off" by moving the head, causing dizziness, rotating vertigo with nausea and vomiting.

12) The classic clinical vestibular tests used for BPPV are the Dix-Hallpike and McClure-Pagnini tests (nystagmus observed in a supine position while turning the head to the left and right).

13) The standard canalith-repositioning maneuver for posterior semicircular canal BPPV is either the Epley maneuver or the Semont maneuver.

14) The standard canalith-repositioning maneuver for lateral semicircular canal BPPV is the Gufoni maneuver.

15)"Comparing these results with the treatment of idiopathic BPPV, we noted that the posttraumatic variant requires more maneuvers to reach curative repositioning of otoliths."

	Idiopathic BPPV	**Post-traumatic Whiplash BPPV**
Asymptomatic in 1 maneuver	81%	56%
Asymptomatic in 2 maneuvers	17%	33%
Asymptomatic in 3 maneuvers	2%	11%

16) Significant injuries can occur following low-speed motor vehicle collision.

17) "Simulated accidents have shown that a 5-mph rear-end car crash can result in a positive acceleration of 8.2 G of the head."

18) Typical acute symptoms after whiplash injury includes neck pain, headache, paraesthesia of upper cervical dermatomes, dizziness or imbalance, and tinnitus.

19) "Pathophysiologically, there is central nervous system weakness following a whiplash injury."

20) Whiplash injury can injure the inner ear through a number of mechanisms, including:
A)) Transient ischemia by vertebral artery compression
B)) Hemorrhage into labyrinth
C)) Direct labyrinthine concussion

21) Whiplash injury is a direct cause of BPPV, especially when head trauma is involved. Following the trauma, the otoliths are detached and displaced within the labyrinth.

22) Classically, BPPV patients experience severe vertigo when rolling in one particular direction in bed. When dizziness occurs at times other than in bed, cervicogenic vertigo must be considered after a whiplash trauma.

23) Posttraumatic BPPV accounts for 15% to 20% of all cases.

24) The diagnosis of BPPV is by the Dix-Hallpike positional tests.

25) BPPV is easily treated with simple canalith repositioning maneuvers (CRM).

26) About 80% of patients with posterior canal *idiopathic* BPPV become free of symptoms and signs following a single canalith-repositioning maneuver.

27) Whiplash injury that causes a disorder of neck proprioceptors can cause static labyrinthine stimulation and vertigo that manifests without changing head position.

28) Dizziness can be the main complaint following a whiplash injury.

29) "Posttraumatic BPPV is not different from the idiopathic form, but the treatment may require more canalith repositioning maneuvers to achieve satisfactory results."

Does Discography Cause Accelerated Progression of Degeneration Changes in the Lumbar Disc: A Ten-Year Matched Cohort Study

Spine
October 1, 2009, Volume 34, Number 21, pp 2338–2345
2009 ISSLS Prize Winner (International Society for the Study of the Lumbar Spine)
This study was done at Stanford University School of Medicine

Eugene J. Carragee, MD, Angus S. Don, FRACS, Eric L. Hurwitz, DC, PhD, Jason M. Cuellar, MD, PhD John Carrino, MD, and Richard Herzog, MD

KEY POINTS FROM DAN MURPHY

1) The objective of this study was to compare progression of common degenerative findings between lumbar discs injected 10 years earlier with those same disc levels in matched subjects not exposed to discography. The study used 102 subjects, 50 who had discography and 52 who were matched controls.

2) "In all graded or measured parameters, discs that had been exposed to puncture and injection had greater progression of degenerative findings compared to control (noninjected) discs."

3) "Small bore needle puncture and limited pressure injection, can clearly cause an increase in progression of degenerative [disc] findings."

4) Injecting normal discs even with small gauge needles appears to increase the rate of degeneration in these discs over time.

5) Intradiscal therapeutic strategies, like injecting steroids, sclerosing agents, etc, may also have detrimental consequences as a consequence of the injection procedure itself.

6) The accelerated disc degeneration caused by discography is secondary to mechanical injury to the annulus and to secondary biochemical cellular processes.

7) "Disc puncture with even a small gauge needle and limited injection pressures appears to be associated with accelerated disc degenerative processes, disc herniation, loss of disc height and signal and the development of reactive endplate changes compared to match-controls."

COMMENT FROM DAN MURPHY

This study indicates that disrupting the integrity of the annular ring of the disc with a needle, either for diagnostics or for treatment, tends to accelerate disc degenerative disease. These procedures should not be performed without considering these risks.

Is compensation "bad for health"? A systematic meta-review

Injury
January 8, 2010

Natalie M. Spearing and Luke B. Connelly

KEY POINTS FROM DAN MURPHY

1) This study is the first to objectively examine the quality of systematic reviews on the topic of compensation and health status.
2) These authors used 11 studies that met their stringent inclusion criteria:

A)) One review was unable to come up with a conclusion.

B)) 9 of the 11 reviews concluded that health outcomes are poorer among people seeking or receiving compensation compared to uncompensated individuals.

However, all 9 were of low quality and suffered from a number of methodological flaws.

C)) One review concluded there is no evidence of an association between compensation and health outcomes.
[Scholten-Peeters G, Verhagen A, Bekkering G, van der Windt DA, Barnsley L, Oostendorp RA. Prognostic factors of whiplash-associated disorders: a systemic review of prospective cohort studies. Pain, 2003, 104: 303-22]

This study was judged by the authors to be the highest quality study in their review.

3) Three of the systematic reviews pertained to whiplash injuries.

A)) Two of these reviews [Carroll] [Cote] relied on many of the same studies and had similar conclusions: compensation results in poorer health outcomes; in fact, three of the authors are the same in each article. Interestingly, two of the shared authors are chiropractors. These same two whiplash studies used "claim duration as a proxy for recovery." This is illogical and weakens their conclusions. "There is debate about whether proxy measures of health such as return to work and time-to-claim closure are suitable, given that other factors may influence decisions about absence from work and the duration of compensation claims."

B)) The third whiplash systematic review was considered to be the best quality study in this review. [Scholten-Peeters, Pain, 2003]
It "specifically limited their focus to measures of symptoms and disability (i.e., health outcomes) and found strong evidence of no association between the legal process of litigation and recovery from whiplash."

"The review considered to provide internally and externally valid results found no evidence that 'compensation', or more specifically, litigation, is 'bad for health'."

4) "There is evidence from one well-conducted systematic review (focusing on one legal process and on health outcome measures) of no association between litigation and poor health outcomes among people with whiplash, contradicting the hypothesis that such an approach contributes to poorer health status." [Scholten-Peeters, Pain, 2003]

"Notwithstanding the limitations of the research in this field, one higher quality review examining a single compensation process and relying on primary studies using health outcome (rather than proxy) measures found strong evidence of no association between litigation and poor health following whiplash, challenging the general belief that legal processes have a negative impact on health status."

5) "While there is apparent interest in linking compensation with poor health status, the evidence for such an association is equivocal, conflicting, and suffering from methodological limitations."

6) "Moves to alter scheme design and limit access to compensation on the basis that it is 'bad for health' are therefore premature."

7) "This meta-review demonstrates that calls to change scheme design or to otherwise alter the balance between the cost and availability of injury compensation on the basis that compensation is 'bad for health' must be viewed with caution by decision makers in the health, law, and insurance fields, and by consumers."

8) "Based on the current research, the question of whether injury compensation is associated with poor health outcomes among subjects with verifiable and non-verifiable injuries remains unanswered because the research in this field is hampered by methodological limitations, the intervention is heterogeneous and complicated to measure, and data on health outcomes are not routinely collected."

COMMENTS FROM DAN MURPHY

This is an important article for those who treat whiplash-injured patients. The authors note that two of the systematic reviews that concluded that compensation adversely influences health outcomes following whiplash injury suffer from methodological flaws (Cote, Spine, 2001; Carroll, Spine, 2008). Interestingly, both studies have the same chiropractic authors. The authors of this current study are critical of Core/Carroll for the use of "claim duration as a proxy for recovery."

In contrast, the study that these authors judged to be the best quality (Scholten-Peeters, Pain, 2003) found no association between compensation and whiplash recovery.

Omega-3 PUFA: Good or bad for prostate cancer?

Prostaglandins, Leukotrienes and Essential Fatty Acids
September-November 2008;79(3-5):97-9

Ingeborg A. Brouwer

FROM ABSTRACT:

The objective of this meta-analysis was to estimate quantitatively the associations between intake or status of omega-3 polyunsaturated (omega-3 PUFA) fatty acids and occurrence of prostate cancer in observational studies in humans.

Methods
We combined risk estimates across studies using random-effects models.

Results
The combined estimate showed an increased risk of prostate cancer in men with a high intake or blood level of alpha-linolenic acid (ALA) (36% increased risk).

The association is stronger in the case-control studies (84% increased risk) than in the prospective studies (10% increased risk).

Eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) were not significantly associated with prostate cancer.

Discussion
The association between high intake of ALA and prostate cancer is of concern and needs further study.

THIS AUTHOR ALSO NOTES:

"Prostate cancer is the second most common cancer in men in the World."

This author reviewed the MEDLINE literature to determine the effect of different omega-fatty acids on the incidence of prostate cancer. Thirteen observational studies were found (7 were prospective studies and 6 were case-control studies).

"The combined estimate of all observational studies showed an increased risk of prostate cancer in men with a high intake or blood level of ALA" by 36%.

The association between ALA intake and prostate cancer was stronger in the case-control studies (84% increased risk) than in the prospective studies (10% increased risk).

The author found 8 observational studies on EPA intake or blood concentrations and prostate cancer: 5 studies were prospective and 3 were case-control studies.

Combined, EPA reduced the risk of prostate cancer by 10%.

The author found 7 observational studies on DHA intake or blood concentration and prostate cancer: 4 of these were prospective studies and 3 were case-control studies.

Combined, DHA reduced the risk of prostate cancer by 9%.

DISCUSSION

This meta-analysis shows a combined increased risk for prostate cancer of 36% for men with a relatively high intake of ALA.

"In conclusion, intake or status of the very long-chain omega-3 fatty acids EPA and DHA do not seem to be associated with risk of prostate cancer."

"Association between high intake of ALA and prostate cancer is of concern and needs further study."

KEY POINTS FROM DAN MURPHY

Alpha-linolenic acid (ALA) is an 18-carbon long plant omega-3 fatty acid. Sources include flax seed oil, hemp oil and walnut oil.

Eicosapentaenoic acid (EPA) is a 20-carbon long omega-3 fatty acid found in fish oil.

Docosahexaenoic acid (DHA) is a 22-carbon long omega-3 fatty acid found primarily in fish oil; there are algae sources of DHA.

In this meta-analysis study, the authors found 24 studies assessing the risk of prostate cancer as related to the intake of ALA, EPA, and DHA

1) "Prostate cancer is the second most common cancer in men in the World."

2) Combined, higher intake or blood levels of ALA increased the risk of prostate cancer by 36%.

3) Combined, higher intake or blood levels of EPA reduced the risk of prostate cancer by 10%.

4) Combined, higher intake or blood levels of DHA reduced the risk of prostate cancer by 9%.

Deceleration during 'real life' motor vehicle collisions: A sensitive predictor for the risk of sustaining a cervical spine injury?

Patient Safety in Surgery
March 8, 2009; Volume 3; Number 1

Martin Elbel, Michael Kramer, Markus Huber-Lang, Erich Hartwig, Christoph Dehner

FROM ABSTRACT:

Background: The predictive value of trauma impact for the severity of whiplash injuries has mainly been investigated in sled- and crash-test studies. However, very little data exist for real-life accidents.

Therefore, the predictive value of the trauma impact as assessed by the change in velocity of the car due to the collision (ΔV) for the resulting cervical spine injuries were investigated in 57 cases after real-life car accidents.

Methods: ΔV was determined for every car and clinical findings related to the cervical spine were assessed and classified according to the Quebec Task Force (QTF).

Results:
32 (56%) subjects did not complain about symptoms and were therefore classified as QTF grade 0.
25 (44%) patients complained of neck pain:

- 8 (14%) were classified as QTF grade I
- 6 (10%) as QTF grade II
- 11 (19%) as QTF grade IV.

Only a slight correlation was found between the reported pain and ΔV.

No relevant correlation was found between ΔV and the neck disability index and between ΔV and the QTF grade for any of the collision types.

There was no ΔV threshold associated with acceptable sensitivity and specificity for the prognosis of a cervical spine injury.

Conclusion: The results of this study indicate that ΔV is not a conclusive predictor for cervical spine injury in real-life motor vehicle accidents.

This is of importance for surgeons involved in medicolegal expertise jobs as well as patients who suffer from whiplash-associated disorders (WADs) after motor vehicle accidents.

THESE AUTHORS ALSO NOTE:

"The economic damage caused by whiplash amounts to some 10 billion Euros a year in Europe and 29 billion US Dollars a year in the USA."

The delta v (ΔV), which describes the velocity change of a motor vehicle during a collision with another vehicle, has become a widely accepted criterion for the energy that acts on the vehicle during a collision. When the energy (ΔV) is below a defined threshold it is considered to be harmless.

"ΔV threshold values were adopted very early in the history of insurance law as a criterion to accept or deny the claim settlement for whiplash-associated disorders (WADs)."

Volunteer crash tests do not replicate real-left collisions for two reasons:

1) The subjects maintained an upright body and head position while waiting for impact.
2) The headrest was optimally adjusted.

These factors have been shown to influence injury, and are completely independent of the collision ΔV:

1) The seat angle and springiness
2) Headrest height
3) The distance between head and headrest
4) Head rotation
5) The collision type

This study analyzes the correlation between ΔV and cervical spine injuries in real-life accidents and questions whether ΔV is a valid predictor for cervical spine injuries following whiplash.

The 57 patients in this study were thoroughly evaluated with:

- Visual analog scale (VAS)
- The neck disability index (NDI)
- Physical examination (cranial nerves as well as of the motor and sensory function of spinal nerves C5–C8, cervical range of motion)
- X-ray examination
- A CT scan was taken if pathological findings were noted

"In addition to the clinical findings, the ΔVs of their respective accident vehicles were determined for all patients. The damage on all vehicles involved in the accidents was examined by a certified engineer who was experienced in the assessment of such damage. The ΔV and the collision type (frontal, rear-end, side

collision, multiple collisions, rollovers) were determined on the basis of the damage sustained by the vehicles."

"For all collision types it was impossible to define a ΔV value that excluded the occurrence of cervical spine injury with acceptable sensitivity while simultaneously predicting the occurrence of cervical spine injury with acceptable specificity."

DISCUSSION

"This study provides evidence that, in real-life accidents, cervical spine injuries may occur at low ΔV values, while it is possible to escape unscathed from collisions with high ΔV values." **[Very Important]**

"The correlation between ΔV and the occurrence of WADs was very low for any of the collision types."

"It is impossible to make meaningful statements about the existence of WAD based solely on assessment of the ΔV value."

"Diagnostic and therapeutic management should not be based solely on information related to trauma impact."

"Multiple factors may influence the risk of injury in each individual case. Due to the additive effects of various protective factors, high-energy impacts may be absorbed without injury, while the additive effects of unfavorable factors could explain injuries sustained in low-energy impacts."

Factors known to influence the risk of injury include:

- Sex
- Head position
- Sitting position
- Distance between head and headrest
- Seat construction
- The duration of the crash pulse

"The current data exclude the assumption of a linear correlation between ΔV and the risk of suffering a whiplash injury."

"It can be concluded that ΔV is an irrelevant predictive value for cervical spine injury after a MVA."

"The ΔV value as measured in the trauma impact does not represent a conclusive predictor for cervical spine injury in real-life motor vehicle accidents. This could be important for surgeons and patients in their medicolegal assessment of WADs."

BACKGROUND:
Quebec Task Force clinical classification of whiplash-associated disorders:

0 No complaint about the neck, no physical signs
I Neck complaint of pain, stiffness or tenderness only, no physical signs
II Neck complaint and musculoskeletal signs, including decreased range of motion and point tenderness
III Neck complaint and neurological signs, including decreased or absent deep tendon reflexes, weakness and sensory deficits
IV Neck complaint and fracture or dislocation

"Symptoms and disorders that can be manifest in all grades include deafness, dizziness, tinnitus, headache, memory loss, dysphagia and temporomandibular joint pain."

KEY POINTS FROM DAN MURPHY

1) The delta v (ΔV), which describes the velocity change of a motor vehicle during a collision with another vehicle, has become a widely accepted criterion for the energy that acts on the vehicle during a collision. When the energy (ΔV) is below a defined threshold it is considered to be harmless.

2) "ΔV threshold values were adopted very early in the history of insurance law as a criterion to accept or deny the claim settlement for whiplash-associated disorders (WADs)."

3) Volunteer crash tests do not replicate real-left collisions for two reasons:

A)) The subjects maintained an upright body and head position while waiting for impact.
B)) The headrest was optimally adjusted.

4) These factors have been shown to influence injury, and are completely independent of the collision ΔV:

A)) The seat angle and springiness
B)) Headrest height
C)) The distance between head and headrest
D)) Head rotation
E)) The collision type

5) Our examination of the whiplash-injured patient should probably include:

- Visual analog scale (VAS)
- The neck disability index (NDI)
- Physical examination (cranial nerves as well as of the motor and sensory function of spinal nerves C5–C8, cervical range of motion)
- X-ray examination
- A CT scan was taken if pathological findings were noted

6) "For all collision types it was impossible to define a ΔV value that excluded the occurrence of cervical spine injury with acceptable sensitivity while simultaneously predicting the occurrence of cervical spine injury with acceptable specificity."

7) "This study provides evidence that, in real-life accidents, cervical spine injuries may occur at low ΔV values, while it is possible to escape unscathed from collisions with high ΔV values." **[Very Important]**

8) "The correlation between ΔV and the occurrence of WADs was very low for any of the collision types."

9) "It is impossible to make meaningful statements about the existence of WAD based solely on assessment of the ΔV value."

10) "Diagnostic and therapeutic management should not be based solely on information related to trauma impact."

11) "Multiple factors may influence the risk of injury in each individual case. Due to the additive effects of various protective factors, high-energy impacts may be absorbed without injury, while the additive effects of unfavorable factors could explain injuries sustained in low-energy impacts."

12) No relevant correlation was found between ΔV and the neck disability index and between ΔV and the QTF grade for any of the collision types.

13) There was no ΔV threshold associated with acceptable sensitivity and specificity for the prognosis of a cervical spine injury.

14) The results of this study indicate that ΔV is not a conclusive predictor for cervical spine injury in real-life motor vehicle accidents.

15) "The current data exclude the assumption of a linear correlation between ΔV and the risk of suffering a whiplash injury."

16) "It can be concluded that ΔV is an irrelevant predictive value for cervical spine injury after a MVA."

17) "The ΔV value as measured in the trauma impact does not represent a conclusive predictor for cervical spine injury in real-life motor vehicle accidents."

Orofacial Injuries Due to Trauma Following Motor Vehicle Collisions: Temporomandibular Disorders

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KEY POINTS FROM THIS ARTICLE:

1) Temporomandibular disorders are "a collective term that embraces a number of clinical problems that involve the masticatory muscles, the TMJ [temporomandibular joint], and associated structures."

2) Most literature strongly supports the association between motor vehicle collisions, temporomandibular disorders, facial pain and headache.

3) Whiplash trauma may cause temporomandibular disorders via two mechanisms:
A)) Direct orofacial trauma
B)) Indirect or [inertial] injury; [this occurs without direct contact to the jaw]

4) "TMDs have been clearly documented following an MVC involving direct orofacial trauma and in a subset of WAD patients where no direct orofacial trauma is recognized."

5) Temporomandibular disorders may not be identified at the time of first assessment of the whiplash-injured patient, but may develop weeks or more after the collision. **[This delay in the development of temporomandibular symptoms following motor vehicle collisions is quite important].**

6) "TMDs may not necessarily be diagnosed during a first assessment, but may manifest weeks or months after an MVC."

7) Temporomandibular disorders in whiplash-injured patients occur predominantly in women.

8) Temporomandibular disorders in whiplash-injured patients may be associated with regional or widespread pain. **[Important: some whiplash-injured patients, especially those with temporomandibular disorders, develop widespread pain syndrome].**

9) Temporomandibular disorders following motor vehicle collisions may respond poorly to independent therapy and may be best managed using multidisciplinary approaches.

10) Approximately 33% of those in a motor vehicle collision develop whiplash-associated disorders.

11) Whiplash-injured patients who also develop TMD have a measurably worse recovery prognosis than those who do not also develop TMD.

12) Temporomandibular disorders associated with whiplash injuries include:
- Jaw pain or dysfunction
- Headache
- Dizziness
- Hearing disturbances
- Neck pain and dysfunction
- Reduced or painful jaw movement

13) Temporomandibular disorders often include TMJ sounds (clicking, crepitus) and catching or locking with opening or closing.

14) There is a "risk of delayed onset of temporomandibular disorders following a motor vehicle collision." Of whiplash-injured patients, approximately 4 times more patients have temporomandibular disorders at 1 year compared to the first evaluation following the collision.

15) "The potential delay in onset of TMDs following an MVC raises concerns about diagnosis, prognosis, management and medico-legal issues."

16) "Regional and widespread physical symptoms as well as psychological disturbances are common in motor vehicle collision patients."

17) Air bag deployment injuries include:
- TMJ injury
- Maxillofacial fractures
- Burns
- Eyes injuries
- Ear injuries
- Cranial VII paresis
- Neuropathic facial pain (Cranial V injury)
- Basal skull fractures
- Transection of the internal carotid artery
- Atlanto-occipital dislocation
- Spinal cord injuries

18) "Approximately 15–40% of patients with acute whiplash associated disorders develop chronic symptoms."

19) "TMDs in WAD are more common in females and can be associated with regional or widespread pain that may reflect central, systemic and psychological effects."

Motor Vehicle Accidents: The Most Common Cause of Traumatic Vertebrobasilar Ischemia

Canadian Journal of Neurological Sciences
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FROM ABSTRACT:
Background:
Recent media exposure of strokes from chiropractic manipulation have focused attention on traumatic vertebrobasilar ischemia. However, chiropractic manipulation, while the easiest cause to recognize, is probably not the most common cause of this condition.

Methods:
We reviewed all consecutive cases of traumatic vertebrobasilar ischemia referred to a single neurovascular practice over 20 years.

Results:
There were 80 patients whose vertebrobasilar ischemia was attributed to neck trauma. Five were diagnosed as due to chiropractic manipulation, but the commonest attributed cause was motor vehicle accidents (MVAs), which accounted for 70 cases; one was a sports injury, and five were industrial accidents.

In some cases neck pain from an MVA led to chiropractic manipulation, so the cause may have been compounded.

In most vehicular cases the diagnosis had been missed, even denied, by the neurologists and neurosurgeons initially involved.

The longest delay between the injury and the onset of delayed symptoms was five years.

Conclusions:
Traumatic vertebrobasilar ischemia is most often due to MVAs; the diagnosis is often missed, in part because of the delay between injury and onset of symptoms and, in part, we hypothesize, because of reluctance of doctors to be involved in medicolegal cases.

SIGNIFICANT POINTS FROM THIS ARTICLE:

1) Recent neck trauma may cause extracranial carotid and vertebral dissection.

2) Genetic abnormalities of collagen, elastin and other supporting elements in the blood vessel wall may predispose the patient to carotid and vertebral artery dissection with even minor trauma such as sneezing or Valsalva maneuvers.

3) When symptoms are delayed after trauma, the connection between trauma and vascular events are difficult to recognize. The longest described (in the literature) delay between trauma and onset of vertebrobasilar symptoms is two months.

4) "Delays of 7 weeks after injury and 37 days after a motor vehicle accident (MVA) have been reported for the vertebrobasilar distribution, and delay of many years has been described in the carotid artery distribution."

5) When vertigo is experienced following vertebral artery dissection, it is "often difficult for the attending physician to think of cerebral vascular problem as opposed to a vestibular problem, particularly in a young patient."

6) Migraine can produce visual symptoms indistinguishable from those due to transient ischemia in the posterior cerebral artery territory, and migraine can also be triggered by trauma.

7) In this study, all of these were attributed to vertebrobasilar ischemia:
70% showed loss of consciousness
64% had some difficulty with short-term memory or episodes of transient global amnesia
2.5% had sleep disturbances (narcolepsy, sleep-walking)

Mechanisms of Injury (80 Cases)

Number	**Percentage**	**Mechanism**
1	1%	Struck by Swinging Car Door
5	6%	Chiropractic Manipulation
5	6%	Industrial Injury
7	9%	Pedestrian Struck by Vehicle
62	78%	Motor Vehicle Collisions

Motor Vehicle Mechanism (62 Collisions)

3	4%	Single Vehicle Collision
4	7%	Head-on Collision
24	39%	Rear-end Collision
31	50%	Side-impact Collision

8) "Several patients had a motor vehicle accident, and then had further vascular injury by neck manipulation." [This suggests that absent the initial MVA, the chiropractic manipulation would not have been associated with the ischemia].

9) "The severity of the initial trauma was substantial for some patients, but was mild for many."

10) "We have been struck by how often neurologists and neurosurgeons miss or refuse to recognize the diagnosis, and suspect that it is often dismissed because of an aversion to medicolegal cases and legal practitioners." "The unwillingness to diagnose this condition of traumatic vertebrobasilar ischemia in the setting of MVAs is in marked contrast to the willingness, even eagerness, to diagnose it in the setting of chiropractic manipulation, often by the same physicians who are unwilling to recognize it in MVA cases."

11) The cases of sleep disturbance noted (sleepwalking and narcolepsy) were attributed to ischemia of the brainstem reticular formation.

12) "The diagnosis is often missed, in many cases because the neck injury is a minor event such as a chiropractic manipulation."

13) Another reason for failure to consider the association between the neck injury and the subsequent vertebrobasilar ischemia is the delay in time, up to nearly five years.

14) "With extension of the neck, especially when adding a rotational component, the vertebral artery is stretched and/or compressed in the foramen transversarium of the atlas or before it pierces the atlanto-occipital ligament."

15) Trivial neck turning while looking backward while backing up a car or during swimming, yoga or archery, or a Valsalsva maneuver during birthing, may initiate vertebral artery dissection.

16) Neck extension during surgical intubation has been shown to injure the vertebral artery.

17) Vertebrobasilar ischemia may occur after extension of the neck over the edge of a hairdresser's sink while having hair shampooed.

18) 72% of people have an asymmetric circulation, usually having one hypoplastic vertebral artery.

19) Rotation and extension of the neck obstructs flow in a dominant vertebral artery, which may predispose the patient to problems when the collateral circulation is poor.

20) Rarely, osteophytes may compress a vertebral artery causing recurrent transient ischemic attack events.

21) Instability of the alar or other atlanto-axial ligaments may cause episodes of vertebrobasilar ischemia provoked by turning of the head.

22) Arterial spasm may lead to ischemic symptoms, and pre-traumatic spasm increases the incidence of dissection.

23) Migraine may cause ischemia symptoms similar or identical to artery dissection.

24) Artery dissections are best shown with either MRA (magnetic resonance angiogram) or ultrasound.

25) "Traumatic vertebrobasilar ischemia may present up to four and nearly five years after the neck injury. It is, therefore, probably much more common than is currently suspected."

26) "Though chiropractic manipulation is perhaps the best-known cause, it is important to recognize that MVAs are a much more common cause, which is often missed."

COMMENTS FROM DAN MURPHY

- All chiropractors should be aware that motor vehicle collisions are a prime (and possibly the primary), trigger of vertebral artery dissections.

- The symptoms of post-traumatic vertebral artery dissections can be delayed for hours, days, weeks, months and even years.

- The primary mechanism for vertebral artery dissection is a combination of cervical extension and rotation.

- There appears to be an increased risk of dissections in patients with migraines.

Late Sequelae of Whiplash Injury with Dissection of Cervical Arteries

European Neurology
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FROM ABSTRACT

Background/Aims:
The objective of our study was to estimate the incidence of posttraumatic dissections of cervical arteries in patients with whiplash injury acquired in a car accident.

Methods and Patients:
We performed a retrospective analysis of medical records of 500 patients with whiplash injury acquired in car accidents between 1996 and 2005 and searched for dissections of cervical arteries occurring within 12 months after injury.

Results:
Eight cases of cervical arterial dissection occurred within 12 months following whiplash injury:

Age	Sex	Artery	Time Delay	Collision Type	Speed
21	F	Vertebral	2 wks	Head-on	High
31	M	Carotid	Minutes	Head-on	Low
32	F	Carotid	6.5 mo	Rear-end	?
32	M	Carotid	8 mo	Head-on	Low
38	M	Middle Cerebral (off Carotid)	Minutes	Head-on	High
44	M	Carotid	6 days	Rear-end	Low
45	M	Vertebral	4 mo	Rear-end	?
45	F	Carotid	Hours	Rear-end	Low

The incidence of posttraumatic dissections after whiplash injuries **[1,600/100,000]** was much higher **[about 400 times higher]** than the overall incidence of cervical arterial dissections in the general population **[4.1/100,000]**.

The risk of cerebrovascular events was still increased 4–12 months after whiplash injury (600/100,000 from whiplash v. 3/100,000 in the general population). **[200 times greater from whiplash v. the general population].**

Conclusions:
There is an increased risk of posttraumatic dissection and cerebrovascular events within 12 months after whiplash injury.

Car accidents are an important risk factor for arterial dissections.

The victims of car accidents should be screened for arterial dissections.

KEY POINTS FROM THIS ARTICLE:

1) "Cervical arterial dissection is one of the main causes of ischemic stroke in young adults."

2) Cervical arterial dissections can be categorized as traumatic or spontaneous.

3) Cervical artery dissections "occur when a tear forms in the tunica intima and blood enters into the space between intima and media. This can lead to a complete occlusion of the vessel lumen, which is mostly followed by recanalization after several months."

4) "Approximately 2/3 of dissections of cervical arteries are spontaneous and 1/3 of them are posttraumatic."

5) The overall annual incidence of spontaneous and posttraumatic dissections of the carotid artery is 26 / 1 million."

6) The incidence of vertebral arterial dissection is 15 / 1 million.

7) The overall incidence of cervical arterial dissections is 41 / 1 million (26 / million carotid + 15 / million vertebral).

8) These authors "retrospectively analyzed the data on 500 consecutive patients with whiplash injury acquired in a car accident, and revealed 8 cases with cervical arterial dissection. The incidence of cervical arterial dissections in patients with whiplash injury was much higher than the overall incidence of cervical arterial dissections in the general population. Therefore, we assume a causal relationship between arterial dissection and cervical spine distortion injury."

9) "Cervical arterial dissection can become symptomatic months after a whiplash injury." In this study, 37.5% occurred between 4 -12 months post whiplash injury.

10) "Whiplash trauma in a road traffic accident can lead to cervical arterial dissection, which initially is asymptomatic."

11) "Most clinicians are not aware that patients with arterial dissections are still at risk of cerebrovascular events months after the dissection."

12) "Dissections of cervical arteries following car accidents are often not recognized by clinical examination."

13) "Arterial dissections following car accidents can become symptomatic months after whiplash injury."

14) "Many dissections of cervical arteries remain clinically asymptomatic, and the association with a car accident is not recognized."

15) There is an increased risk of posttraumatic cervical artery dissection within 12 months after whiplash injury (by about 400 times).

16) Motor vehicle collisions should be considered as a risk factor for cervical arterial dissections.

17) "The clinical implementation of this finding should be that the patients with whiplash injury acquired in a car accident are screened for arterial dissections. In case of clinically suspected cervical arterial dissection, each patient should receive Doppler sonography."

18) "Initial MRI of the cervical spine and follow-up investigations after 1–3 months should be considered in patients with whiplash trauma in order to detect vascular, osseous, ligamentous and nerve injuries."

19) Car accidents are an important risk factor for arterial dissections.

20) The victims of car accidents should be screened for arterial dissections.

21) "There is an association between whiplash injury with arterial dissection and delayed cerebrovascular events occurring months after a car accident."

22) MECHANISM OF CERVICAL ARTERY DISSECTION AND SUBSEQUENT SYMPTOMS:

A)) Cervical Artery Dissection forms an intraluminal blood clot that occludes the vessel. This occlusion cannot be washed out by the blood stream.

B)) "The occlusion of the vertebral artery is compensated by a collateral blood supply through the contralateral vertebral artery and does not become symptomatic."

C)) The occlusion of the carotid artery can be compensated through the collaterals of the Circle of Willis.

D)) Therefore, if arterial dissection does not cause a hemodynamic infarction and is adequately compensated for, "it remains asymptomatic."

E)) Recanalization of the occluded vessel can occur weeks and even months (max. 24 months) after the initial arterial dissection.

F)) The recanalization can wash out the intraluminal blood clot, causing a down stream embolism. This embolism is the probable mechanism responsible for delayed cerebrovascular events following whiplash injury. "Generally, secondary thromboembolism can occur within 12 months following whiplash trauma."

IN THIS STUDY:

23) Head-on collision and rear-end collisions were equally likely to produce a cervical artery dissection.

24) Low speed collisions were just as likely as higher speed collisions to create a post-traumatic cervical artery dissection.

25) 25% of the whiplash artery dissections were the vertebral artery, while 75% were from the carotid or its branches.