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Original Articles

Iatrogenic Tendinopathy Associated with Levaquin (levofloxacin)

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Abstract

Fluoroquinolone antibiotics, Levaquin (levofloxacin), ciprofloxacin (Cipro), enoxacin (Penetrex), lomefloxacin (Maxaquin), norfloxacin (Noroxin), ofloxacin (Floxin), sparfloxacin (Zagam), cinoxacin (Cinobac), and nalidixic acid (NegGram)) are increasingly implicated as a cause of tendinopathy and particularly of Achilles tendinopathy and rupture. Reported here is the case of a 52-year old man who developed unilateral Achilles tendinopathy, without rupture, approximately 10 days following commencement of Levaquin therapy. Tendinopathy management focused on conservative, non-invasive procedures. Recovery was incomplete, with refractory weakness and muscle strength issues, as well as range of motion changes. This report includes a review of the current literature on Fluoroquinolone associated tendinopathies and of the various methods of tendon and soft tissue management.

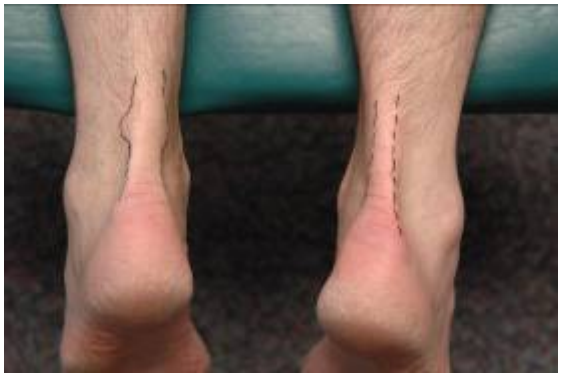
Keywords: Achilles tendinopathy, Achilles tendon MRI, angiofibroblastic hyperplasia, eccentric calf muscle exercise, heel lifts, Kinesio taping, Levaquin, magnesium sulfate, Quinolone side effects, quinolone tenotoxicity, Tendinitis, Tendinopathy

Outline

Case Report

A 52-year old male elite-level body builder, runner, and athlete, with no known co-morbidities, was prescribed a five-day course of Levaquin (levofloxacin), 750 mg per day, by his General Practitioner, for acute pneumonia. He had no other regular medication. Ten days after commencing the primary dosing, and in the absence of trauma, the patient developed an acute, persistent sharp pain in his left lower leg and at the Achilles tendon insertion on the heel. The patient developed a limp, as well as notable swelling of the Achilles tendon. (**Fig. 1**) Dorsiflexion of the foot was painful, worse on stairs or incline surfaces. Two weeks after ending primary dosing, he reported to the ICON Whole Health facility for chiropractic orthopedic evaluation. Physical and orthopedic examination confirmed left Achilles tendinopathy of probable iatrogenic quinolone-therapy origin. The tendon remained intact, and advanced imaging was deferred.

Figure 1. Localized swelling at the 3-6 cm level (from the calcaneal insertion) in the left Achilles tendon.



The patient underwent conservative management of the Achilles tendinopathy, which included: **1**) rest and non-weight bearing of the part, **2**) applications of Achilles paratenon, gastrocnemius, and soleus musculature low watts pulsed ultrasound, (**Fig. 2**) (although indicated for combined application with ultrasound in this condition, treating physicians delayed cross-frictional massage for the subacute phase). (**Fig. 3)** Conservative treatment should be used as first-line management when treating Achilles tendinopathy, with the expectation of significant improvement after a 12-week treatment program[1]. A 12-week eccentric exercise program appears to produce a superior functional outcome when used in addition to ultrasound and deep frictional massage, versus ultrasound and deep frictional massage alone.

Figure 2. Ultrasound application (low watts) to Achilles tendon and peritendinous tissue.

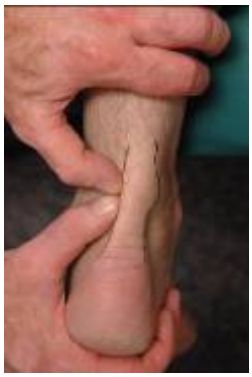


Figure 3. Cross-frictional massage for Achilles Tendinopathy, commonly utilized in subacute phase of disorder, often combined with applications of ultrasound.

3) Application of a 6mm supportive heel lift. Although heel lifts place the ankle in a more plantar-flexed position during quiet standing, walking and running, this does not result in a decrease in the strain placed upon the Achilles tendon during locomotion[2]. Heel lifts cannot be used as the sole therapeutic in Achilles Tendinopathy. **4**) Application of Kinesio taping of the Gastrocnemius-Soleus muscle mechanism (**Fig. 4**).

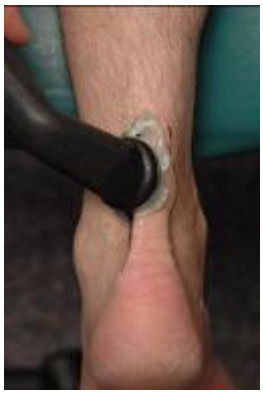


Figure 4. Kinesio taping for left Achilles Tendinopathy.

The application of tape has been widely used for many years by physiotherapists as a useful adjunct to prophylactic or rehabilitation programs in the management of a wide variety of described include: neuromusculoskeletal disorders. The effects of taping which are most commonly synergists, inhibition of overactive synergists or antagonists, facilitation of underactive movement unloading of promotion of proprioception, optimization of joint alignment, pain reduction and sulfate irritable neural tissue[3]. **5**) Applications of analgesic/antiinflammatory magnesium foods, drugs, and hydrotherapy. Although magnesium can be absorbed through the digestive tract, many Therefore, medical conditions can interfere with the effectiveness of this delivery method. making magnesium hydrotherapy using a magnesium sulfate solution is one of the most effective means of of 1% Epsom salts readily available[4]. Significant rise in plasma magnesium and sulfate is seen at a level liters, with maximal concentration in water. This equates to 1g MgS04/100ml water; 600g Epsom salts/60 Epsom salts each benefit reached by using the hydrotherapy 2 or 3 times/ week, prepared with 500-600g time[4]. **6**) Rehabilitative ROM, and strengthening exercises. Imaging studies demonstrate that some of the pathophysiological changes identified in symptomatic tendons reverse following a course of eccentric loading exercises. Following a 12-week course of eccentric loading exercises, the majorities of patients become asymptomatic and demonstrate a return to normalized Achilles tendon structure, and a corresponding reverse in neovascularization. The high torques associated with eccentric loading in comparison with

concentric loading may cause interfibrillar disruption and stimulate a healing response. Matrix remodeling and collagen realignment are perhaps a response to the increased demands placed upon the tendon during sustained eccentric loading[5].

Acute care continued for six weeks, with sub-acute rehabilitative management continuing for another six weeks. The patient demonstrated good return to function, but incomplete recovery of the Achilles peritendinous tissues. The patient returned to running, with protective taping of the part, primarily on flat or low incline terrain. Strengthening exercises and ROM exercises continue. He continues to experience post inertial dyskinesia, limited to the tendon and peritenon tissues, which dissipates with activity. Morning stiffness and soreness persists. Granulomatous tissue persists and complicates return to elite-level athletic training activities.

Discussion

Fluoroquinolone

Fluoroquinolone induced tendinopathy has been most commonly described after the administration of ciprofloxacin, although more recently, cases from levofloxacin are emerging. Achilles tendinitis or rupture are among the most serious side effects of Fluoroquinolone and have been increasingly reported since 1983[6]. As will be discussed, literature (clinical reports, histopathologic findings, and experimental studies) demonstrates a causal relationship between fluoroquinolone administration and tendinopathy/tendon rupture.

Fluoroquinolones are characterized by a good safety and tolerability profile, good bioavailability, a strong antibacterial effect with low minimal inhibitory concentrations, and a broad antibacterial spectrum. Commonly recognized side effects consist of gastrointestinal tract symptoms, central nervous system toxicity, cardiotoxicity with QT prolongation, and phototoxicity. Additionally, but less frequently clinically correlated, fluoroquinolones can cause damage to connective tissues; cartilage in young individuals (chondrotoxicity), tendons in adults (tenotoxicity)[7].

The incidence of tendon injury associated with fluoroquinolone use is low in a healthy population, 0.14% to 0.40%, but increases in patients who are older than 60 years, who have renal dysfunction, who are under corticotherapy, who are undergoing hemodialysis, or who have received renal transplants[8-10]. For Quinolone associated Achilles tendon ruptures, the following statistics are supported in literature: An average age of 64 years; 2:1, Male/Female ratio and 27% incidence of bilateral involvement. Half the ruptures occurred within one week of quinolone administration with symptoms occurring within two hours in one patient, 85% presented in less than one month with 20% on oral corticosteroids. Ciprofloxacin was responsible for over half the cases, most often being prescribed for respiratory infections and over half of patients had other associated risk factors for Achilles tendon rupture[11]. Complete ruptures accounted for three quarter with good functional recovery following appropriate diagnosis and management[6]. The relative risk of tendinitis from quinolones is 3.7 compared to other antibiotics[9]. However, the relative risk increases to 8.0 for tendinitis and rupture in lung transplant patients prescribed ciprofloxacin[12]. In a World Health Organization survey in Australia of the quinolone-related tendon disorders, ciprofloxacin was found to be the causal agent in 90% of the patients[13].

Levaquin

Levaquin (levofloxacin) is a member of the fluoroquinolone family, which is a relatively widely used group of broad-spectrum antibiotics. Levofloxacin has enhanced activity against gram-positive aerobic organisms, including penicillin-resistant pneumococci. In published comparative trials involving commonly used treatment regimens, levofloxacin had equivalent if not greater activity in the treatment of community-acquired pneumonia, acute bacterial exacerbations of chronic bronchitis, acute bacterial sinusitis, acute pyelonephritis, and complicated urinary tract infection[14]. As frequently reported by national media entities, resistance to antibiotics has become a major public-health concern. This is mainly caused by national and international spread of multiresistant bacterial clones, and the declining interest from the pharmaceutical industry in research and development of new antibacterial drugs[15]. Quinolone therapy is supplanting penicillin and penicillin-derivative therapy, and with increasing frequency[16].

Achilles Tendinopathy

Chronic painful conditions in the midportion of the Achilles tendon, often referred to as Achilles tendinopathy, are relatively common, especially among recreational athletes in the age group between 30 and 60. In the athletic population, the etiology is associated with overuse of the aging tendon, but scientifically, the etiology and pathogenesis are unclear. In general, Achilles tendinopathy includes an inflammatory component, and the nomenclature often used (tendinitis) implies this. Recent research using micro dialysis technique and gene technology, however, clarifies that there is no chemical inflammation in chronic midportion Achilles tendinosis (tendinopathy with ultrasonographically verified tendon changes). Interestingly, the neurotransmitter glutamate and its NMDAR1

receptor were found for the first time in human tendons by using micro dialysis and immunohistochemical techniques. The concentrations of glutamate were significantly higher in painful tendons with tendinosis than in normal, pain-free tendons.

There are several possible differential diagnoses, such as os trigonum syndrome, tenosynovitis or dislocation of the peroneal tendons, tenosynovitis of the plantar flexors, an accessory soleus muscle, tumors of the Achilles tendon (xanthomas), and neuroma of the suralis nerve, that must be considered in differentiating Achilles Tendinopathy [17].

Most commonly, the patients with Achilles tendinopathy present a history of gradual onset of pain during tendon loading, and morning stiffness in the tendon. The common findings during clinical examination are an area of tendon/paratenon tenderness, and often, but not always, a localized swelling in the tendon. In evidence-based clinical investigations, an accurate clinical examination (**Fig. 1**), ultrasonography, magnetic resonance imaging (MRI) (**Fig. 5**), and biopsy are helpful tools for a correct diagnosis.

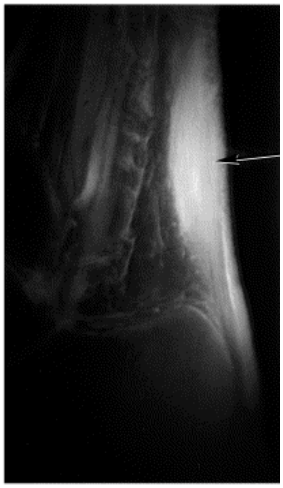


Figure 5. Chronic tendinopathy in a woman aged 57 years, as demonstrated on FUTE, fat-suppressed ultrashort echo time MRI. *Adapted from Robson MD, Benjamin M, Gishen P et al. Magnetic resonance imaging of the Achilles tendon using ultrashort TE (UTE) pulse sequences. Clinical Radiology 2004; 59 (8):727-35.*

Fluoroquinolone-induced tendinopathy

Fluoroquinolone-induced tendinopathy differs from other forms of tendinopathy by its acute onset pain that occurs while using the affected extremity, especially during or immediately following fluoroquinolone therapy (up to six months following therapy). Typically, fluoroquinolone associated tendon symptoms, including rupture, occur within the first few weeks after therapy is started. Other symptoms include swelling, warmth, tenderness, erythema or itchiness over tendon sites and functional disability. Bilateral involvement is common[9, 18, 19]. The diagnosis should always be considered for a patient undergoing fluoroquinolone therapy and experiencing acute onset, tendon related pain. If prudent in the management of the primary infective disorder, fluoroquinolones should be discontinued at the first sign of tendon inflammation to reduce the risk of subsequent rupture[20, 21]. The latency period between the start of fluoroquinolone treatment and symptom ranges from a few hours to a few weeks, with a mean of 6–10 days[18]. Usually, the fluoroquinolone-induced tendinopathy recovers in the course of weeks after cessation of fluoroquinolone therapy. However, such recovery may be incomplete, and may only represent cessation of the cytotoxic process in the tendon, rather than true tissue repair.

The mechanism by which ciprofloxacin (fluoroquinolones, Levaquin, etc.) predisposes certain people to tendon injury is unclear. Clinical evidence suggests a direct interaction between fluoroquinolone antibiotics and tendinous tissues. Tendon rupture may occur due to a vasculitic phenomenon leading to ischemia[22]. Further, it is possible that the tendinopathy observed in certain patients treated with fluoroquinolones is secondary to an alteration in fibroblast cell homeostasis that results in structural compromise of the tendon[23].

Achilles Tendinopathy Management Options

Treatment options include rest and splinting of the part for 6-8 weeks. This is best for elderly patients and patients unfit for surgical intervention (for repair of full rupture) or unsuitable candidates for surgery due to delayed presentation. Achilles tendinopathy management also includes applications of low wattage ultrasound; cross-frictional massage; elevation of the calcaneus to achieve plantar flexion of the foot; analgesia with cryotherapy or hydrotherapy (MgSO4) depending on the clinical stage; and rehabilitative strengthening/ROM exercises. A 12% re-rupture rate can be expected. Surgical methodology includes both percutaneous and open procedures. Percutaneous procedures cause less wound breakdown, but have a higher re-rupture rate and demonstrate sural nerve damage in 13% of the cases. Open resection and repair is best for younger patients with early presentation but also carries a higher risk of tissue breakdown[11]. (**Fig. 6**)

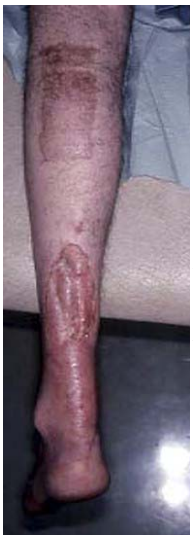


Figure 6. Patient with a one-week course of Ciprofloxacin, 250 mg twice daily for lower respiratory tract infection. One-year post-operative photo shows an intact flap, donor defect and skin graft donor site on the left. *Adapted from Akali AU, Niranjan NS.*

Other Considerations

Fluoroquinolone-associated tendinopathy and tendon rupture most commonly occur in the Achilles tendon[9]. Other structures, such as the long head of biceps, rotator cuff, supraspinatus, extensor pollicis longus, peroneus brevis, patellar, and quadriceps tendons, are also occasionally affected by fluoroquinolone drugs[21]. Lateral epicondylitis has also been reported with fluoroquinolone therapy[24]. Tendinopathy of tibialis anterior tendon associated with fluoroquinolone therapy has been previously reported[25].

Epicondylitis of the Elbow

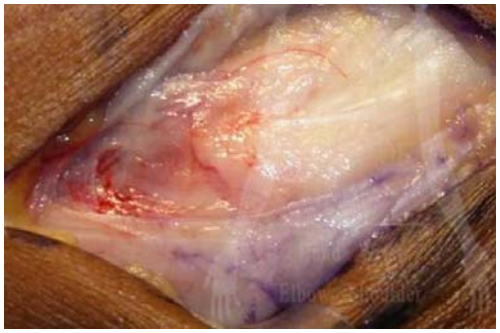


Figure 7. Angiofibroblastic hyperplasia, as seen at the time of debridement of the origin of the elbow extensor tendon with decompression of bone. *Adapted from Gupta, V, Upper Extremity Clinic of India*

Histologic evaluation of fluoroquinolone-induced elbow tendinopathy demonstrates *angio fibroblastic tendinosis* (**Fig. 7**) and is likely the result of a degenerative and avascular process. The use of fluoroquinolone antibiotics (Levaquin, ciprofloxacin, ofloxacin, norfloxacin, enoxacin, lomefloxacin) has been implicated in epicondylitis. The intense pain at the epicondyle appears very shortly after administration of the first dose of the drug. Ultrasonography reveals extensive inflammatory pannus with pseudo necrotic areas. MRI confirms the lesions and demonstrates subclinical abnormality of the adjoining tendons.

Hand

Fluoroquinolone toxicity on cartilages and tendons has been well established since the early 1980's. By the late 1990's a case of rupture of extensor tendons of the hand in an elderly woman treated by fluoroquinolones was reported[26]. Histological examination of tendon injuries was possible after surgical treatment, demonstrating classical tissue descriptions but had specific features consistent with vascular changes as well as direct cell toxicity. A most recent case report describes a 42-year-old female presenting with acute onset of generalized pain several hours after taking ciprofloxacin for presumed urinary tract infection[18]. She was diagnosed with ciprofloxacin-related tendinopathy, and treated with ibuprofen and graded exercises. She after 3 weeks and the condition resolved after 4 months.

Tibialis Anterior

Rupture of the tibialis anterior tendon is uncommon. When it does occur, it is typically attributable to a laceration or sudden force. More uncommon is spontaneous rupture secondary to an underlying degenerative process. (**Fig. 8**)

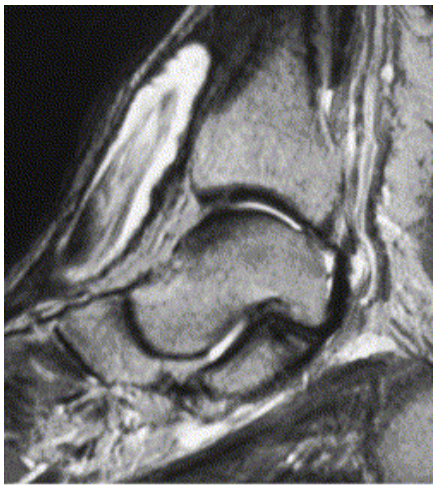


Figure 8. T2-weighted MRI shows fusiform thickening of the anterior tibial tendon at it is distal and linear high signal striations compatible with a partial rupture. There was also fluid in the tendon sheath. *Adapted from Kosucu P, Koktener A, Sahin G et al. Rupture of anterior tibial tendon associated with ciprofloxacin therapy. European Journal of Radiology Extra 2003; 48 (1):31-4.*

Other etiologies for an underlying degeneration of the tibialis anterior include impingement, inflammatory arthritis, diabetes mellitus, infection, chronic microtrauma, ischemia, hyperparathyroidism, systemic lupus erythematosus, gout, psoriasis, obesity, and oral or local steroid therapy, and more recently recognized Fluoroquinolone therapy[23, 25, 27]. Such spontaneous rupture occurs usually in men

in their sixth to seventh decades. Symptoms and signs of anterior tibial tendinopathy are generally mild and include pain, weakness of foot dorsiflexion, swelling at the dorsum of the foot and ankle, limp, and foot-drop. If there is rupture, palpation may reveal a tendon defect or nodule. The nodule represents tendon stump retraction. Spontaneous rupture of the anterior tibial tendon can occur approximately 0.5–3 cm proximal to its bony insertion, corresponding to the inferior half of the avascular zone[27].

Statin Therapy

Tendinopathy may be a yet unreported side effect of statin therapy. Cases of tendinopathy in patients on statin therapy (atorvastatin and fluvastatin, respectively), are beginning to emerge in literature[28]. Clinical examples include a 50-year-old female with bilateral patellar tendinopathy 8 months into atorvastatin treatment, and a male with Achilles tendinopathy 8 weeks after initiation of fluvastatin therapy. Both recovered with discontinuation of the statin.

Practical Implications

Fluoroquinolone-induced tendinopathy differs from other forms of tendinopathy by its acute onset pain that occurs while using the affected extremity, during or immediately following fluoroquinolone therapy. The diagnosis should always be considered for a patient undergoing fluoroquinolone therapy and experiencing acute onset, tendon related pain. If prudent in the management of the primary infective disorder, fluoroquinolones should be discontinued at the first sign of tendon inflammation to reduce the risk of subsequent rupture.[18] Usually, the fluoroquinolone-induced tendinopathy usually recovers in the course of weeks after cessation of fluoroquinolone therapy. However, such recovery may be incomplete, and may represent cessation of the cytotoxic process in the tendon, rather than true tissue repair.

The mechanism by which ciprofloxacin (fluoroquinolones, Levaquin, etc.) predisposes certain people to tendon injury is unclear, but several theories have been proposed such as an immuno allergic mechanism mediated by cytokines, toxic effects directly to the tendon and an ischemic mechanism in the less vascularized portion of the tendon or mechanical stress. Certainly, literature supports caution with elderly patients in the treatment of community acquired chest infection combining steroids with Levofloxacin. This is especially true in those patients who have other increased risk of tendon disease such as those with advanced age, renal failure, hemodialysis, corticosteroid use, peripheral vasculopathy, and strenuous physical activity in a previously healthy person.

Conclusions

Fluoroquinolone antibiotics are increasingly being recognized as a cause of tendinopathy, particularly of Achilles tendinopathy and rupture. Fluoroquinolone induced tendinopathy has been most commonly described after the administration of ciprofloxacin although more recently, cases from levofloxacin are recognized. Fluoroquinolones can cause damage to connective tissues (cartilage in young individuals (chondrotoxicity), tendons in adults (tenotoxicity)). The incidence of tendon injury associated with fluoroquinolone use is low in a healthy population but increases in patients who are older than 60 years, who have renal dysfunction, who are under corticotherapy, who are undergoing hemodialysis, or who have received renal transplants.

This patient, an elite-level body builder, runner, and athlete, with no known co-morbidities, was prescribed a five-day course of Levaquin (levofloxacin), 750 mg per day, by his General Practitioner, for acute pneumonia. Ten days after commencing the primary dosing, and in the absence of trauma, he developed an acute, persistent sharp pain in his left lower leg and at the Achilles tendon insertion on the heel. Patients most often complain of constant pain of the tendon (39%), worsening after physical activity, and improving with rest (61%). In 43% of cases, complaints are reduced with the passage of time, but in 36%, they increase[28]. The key to correct diagnosis is a careful history. The character of first onset of symptoms (acute or gradual) is an essential indicator.

This patient's Achilles Tendinopathy is principally due to treatment with Levofloxacin because of the close temporal relationship between the fluoroquinolone therapy and his symptoms. The patient underwent conservative management of the Achilles tendinopathy, which included: 1) rest and non-weight bearing of the part, 2) Applications of Achilles paratenon, gastrocnemius, and soleus musculature low watts pulsed ultrasound, 3) Application of a 6mm supportive heel lifts, 4) Application of Kinesio taping of the Gastrocnemius-Soleus muscle mechanism, 5) Applications of magnesium sulfate hydrotherapy, and 6) Rehabilitative ROM and strengthening exercises. Acute care continued for six weeks, with sub-acute rehabilitative management continuing for another six weeks, with good return to function, but incomplete recovery of the Achilles peritendinous tissues. Granulomatous tissue persists and complicates rapid return to elite-level athletic training activities.

Acknowledgments

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Competing Interests

The author declares that there is no conflict of interest with respect to the work described in this case report.

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Reprints

LBP Guidelines 2008

Diagnosis and Treatment of Low Back Pain: A Joint Clinical Practice Guideline from the American College of Physicians and the American Pain Society. *What's New? What's Different?*

By: Paul Shekelle, MD, PhD

See the related ACP/APS Guideline Summary.

In October of last year (2007), the American College of Physicians (ACP) and the American Pain Society (APS) released a new clinical practice guideline on the diagnosis and treatment of low back pain. (1) This is the most comprehensive, national-level guideline to be released on this topic since the seminal Agency for Health Care Policy and Research (AHCPR) Clinical Practice Guideline #14 "Acute Low Back Problems in Adults" was released in 1994. (2) (**Disclaimer: I am a co-author on the new guideline in my role as a member of the ACP guideline committee**). Since the 1994 guideline, there has been a vast increase in the research on back pain, including a Patient Outcome Research Team, and the numerous systematic reviews and meta-analyses sponsored by the Cochrane Back Group. So the time seemed ripe for a re-examination of the evidence. What should clinicians make of this new guideline? What's new or different compared to the AHCPR guideline, and can clinicians trust it?

First, there is much to commend about the methods used to develop this new guideline. Although it was sponsored by an interested specialty society (the APS), the society wisely chose to both partner with the American College of Physicians and to turn over control of major portions of the process to an independent group — the Oregon Evidence-based Practice Center (EPC). The Agency for Healthcare Research and Quality (AHRQ) is the successor to AHCPR, and the EPCs are the flagships of AHRQ's program to synthesize evidence. The Oregon EPC conducted extensive literature syntheses on pharmacologic and nonpharmacologic therapies for acute and chronic low back pain — reviews which are published with the guideline in the October 2, 2007 edition of the *Annals of Internal Medicine*. For clinicians, this means the evidence basis for the guidelines has been synthesized by an independent, state-of-the-art group and the results are publicly available for all to see.

Since published evidence is never sufficient to construct a comprehensive guideline, expert opinion is always needed to interpret the existing evidence and help "fill in the gaps." Patients with back pain see many types of providers, so it makes the most sense to have an expert panel that is multidisciplinary. For this guideline, the panel had experts that were general internists/primary care physicians, back surgeons, physical therapists, rheumatologists, neurologists, rehabilitation physicians, nurses, pain specialists, social scientists expert in back

pain, osteopathy, and chiropractic. So diversity of expertise was well represented in the panel that considered the evidence and crafted the guidelines.

Third, the guideline statements are graded both for the strength of the evidence and the strength of the recommendations, letting clinicians know how strongly the guideline panel felt about each statement.

Fourth, the guideline is presented in a fashion that makes it accessible to clinicians. The flow charts (Figures 1 and 2 in the document) present the essence of the guideline in a compact, easy to follow manner.

So what's new or different? The following table compares the "principal conclusions" of the 1994 guideline with the seven statements in the new guideline. Empty cells mean no direct match was present:

2007 ACP/APS Guideline Statements	1994 AHCPR Guideline
Recommendation 1: Clinicians should conduct a focused history and physical examination to help place patients with low back pain into 1 of 3 broad categories: nonspecific low back pain, back pain potentially associated with radiculopathy or spinal stenosis, or back pain potentially associated with another specific spinal cause. The history should include assessment of psychosocial risk factors, which predict risk for chronic disabling back pain (**strong recommendation, moderate-quality evidence**).	• The initial assessment of patients with acute low back problems focuses on the detection of "red flags" (indicators of potentially serious spinal pathology or other nonspinal pathology). • Nonphysical factors (such as psychological or socioeconomic problems) may be addressed in the context of discussing reasonable expectations for recovery.
Recommendation 2: Clinicians should not routinely obtain imaging or other diagnostic tests in patients with nonspecific low back pain (**strong recommendation, moderate-quality evidence**).	In the absence of red flags, imaging studies and further testing of patients are not usually helpful during the first 4 weeks of low back symptoms.
Recommendation 3: Clinicians should perform diagnostic imaging and testing for patients with low back pain when severe or progressive neurologic deficits are present or when serious underlying conditions are suspected on the basis of history and physical examination (**strong recommendation, moderate-quality evidence**).	
Recommendation 4: Clinicians should evaluate patients with persistent low back pain and signs or symptoms of radiculopathy or spinal stenosis with magnetic resonance imaging (preferred) or computed tomography only if they are potential candidates for surgery or epidural steroid injection (for suspected radiculopathy) (**strong recommendation, moderate-quality evidence**).	• If low back symptoms persist, further evaluation may be indicated. • Patients with sciatica may recover more slowly, but further evaluation can also be safely delayed. • With or without surgery, 80 percent of patients with sciatica recover eventually.

2007 ACP/APS Guideline Statements	1994 AHCPR Guideline
Recommendation 5: Clinicians should provide patients with evidence-based information on low back pain with regard to their expected course, advise patients to remain active, and provide information about effective self-care options (**strong recommendation, moderate-quality evidence**).	• While some activity modification may be necessary during the acute phase, bed rest >4 days is not helpful and may further debilitate the patient. • Patients recovering from acute low back problems are encouraged to return to work or their normal daily activities as soon as possible.
Recommendation 6: For patients with low back pain, clinicians should consider the use of medications with proven benefits in conjunction with back care information and self-care. Clinicians should assess severity of baseline pain and functional deficits, potential benefits, risks, and relative lack of long-term efficacy and safety data before initiating therapy (**strong recommendation, moderate-quality evidence**). For most patients, first-line medication options are acetaminophen or nonsteroidal anti-inflammatory drugs.	Relief of discomfort can be accomplished most safely with nonprescription medication and/or spinal manipulation.
Recommendation 7: For patients who do not improve with self-care options, clinicians should consider the addition of nonpharmacologic therapy with proven benefits — for acute low back pain, spinal manipulation; for chronic or subacute low back pain, intensive interdisciplinary rehabilitation, exercise therapy, acupuncture, massage therapy, spinal manipulation, yoga, cognitive-behavioral therapy, or progressive relaxation (**weak recommendation, moderate-quality evidence**).	Relief of discomfort can be accomplished most safely with nonprescription medication and/or spinal manipulation.
	Low-stress aerobic activities can be safely started in the first 2 weeks of symptoms to help avoid debilitation; exercises to condition trunk muscles are commonly delayed at least 2 weeks.
	Within the first 3 months of low back symptoms, only patients with evidence of serious spinal pathology or severe, debilitating symptoms of sciatica, and physiologic evidence of specific nerve root compromise corroborated on imaging studies can be expected to benefit from surgery.

In summary, the new ACP/APS guideline as compared to the old AHCPR guideline:

- Covers a broader spectrum of patients with back pain, including acute and chronic back pain
- Reinforces the statement about using brief history and physical examination findings to categorize patients into those requiring more evaluation to search for a potential other, serious cause of back pain, and those patients in whom this can be delayed or foregone completely
- Is a bit stronger on emphasizing the need for psychosocial assessment to help predict potentially delayed recovery
- Is similarly cautious about the use of plain x-ray imaging, but now more strongly supported by the availability of randomized trials showing no benefit for early x-ray imaging
- Is more forceful about the need to avoid specialized diagnostic imaging such as magnetic resonance imaging (MRI) or computed tomography (CT) without a clear rationale for doing so
- Is similarly strong about the need to stay active
- Is broader in the number of pharmacologic and nonpharmacologic options that may be offered to patients requiring them for symptom control

So what's missing? Most of the money, and most of the controversy, concerns the use of surgical procedures (fusion, artificial discs) and non-surgical invasive interventions (mostly needling procedures, but also including radiofrequency denervation and intradiscal electrothermal therapy) for patients with chronic back pain and no evidence of a herniated disc or spinal stenosis (commonly classified as "discogenic disease"). For many of these conditions, the evidence is either scant or poor quality or both. The 1994 AHCPR guideline dealt with surgery and needling interventions, but only in the context of acute low back pain, and most procedures are not done in patients with acute low back pain. The new ACP/APS guideline does not deal with surgery or other invasive interventions. Clinicians and health plans are still at a loss for the best guidance for the small proportion of patients considering these options. The APS and the Oregon EPC are continuing work on such a guideline — stay tuned to www.guideline.gov!

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Disclaimer

Potential Financial Conflicts of Interest

Dr. Shekelle is a Co-Chair for the NGC/NQMC Editorial Board and is paid for his work in connection with this project. Dr. Shekelle had declared no potential financial conflicts of interest with respect to this Expert Commentary.

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Letters to the Editor:

In reading these guidelines, I am reminded of a quote from a fellow DACBR, Susan L. Vlasuk, DC: "Chiropractors, who routinely utilize a therapeutic procedure that involves manual skeletal forces, have a higher order of necessity to see their "territory" than does a physician who sits across a desk from a patient and writes a prescription."

I think a distinction needs to be made that whilst Dr. Shekelle's guidelines I am sure apply very well to medical practice where a GP MD is providing a prescription for medication for low back pain, they do not apply nearly as well to chiropractic management of low back pain, nor most likely to management by an orthopedic surgeon or a physical medicine specialist.

Rick Corbett DC DACBR DABCO

Editor (Priest) Comment
Recommendation 4: Clinicians should evaluate patients with persistent low back pain and signs or symptoms of radiculopathy or spinal stenosis with magnetic resonance imaging (preferred) or computed tomography only if they are potential candidates for surgery or epidural steroid injection (for suspected radiculopathy) (strong recommendation, moderate-quality evidence).

The use of the word 'only' in this recommendation is my primary concern – I have no problem whatsoever with ordering imaging for these patients as part of their workup when they fail conservative treatment, etc., but one of the main reasons I do so is to help ascertain whether or not they are in fact surgical candidates...I cannot determine before the imaging whether they are or not, yet this recommendation requires that I do so...in other words, how is one to determine if patients 'are potential candidates for surgery' unless one obtains the MRI or CT (many patients with radicular symptoms do not have surgically-responsive pathology)? In fact, when it comes to spinal stenosis and radiculopathy, I know of no surgeon that would make a decision as to whether or not a patient is a candidate for surgery without MRI and/or CT. So...if, as Recommendation 4 is to be followed algorithmically, the physician is placed in the untenable position of having to determine if the patient is a candidate for surgery before ordering the imaging study (to determine if the patient is a candidate for surgery!)...what am I missing here?
More later as time permits,

Gregory Priest, DC, DABCO

Abstracts & Literature Review

Risk of Vertebrobasilar Stroke and Chiropractic Care Results of a Population-Based Case-Control and Case-Crossover Study

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Yaohua He, MD, PhD,* Sheilah Hogg-Johnson, PhD,†§ Frank L. Silver, MD, FRCPC,¶
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SPINE Volume 33, Number 4S, pp S176–S183 February 15, 2008, Volume 33, Issue 4S Supplement

Abstract:

Study Design. Population-based, case-control and case-crossover study. Objective. To investigate associations between chiropractic visits and vertebrobasilar artery (VBA) stroke and
to contrast this with primary care physician (PCP) visits and VBA stroke. Summary of Background Data. Chiropractic care is popular for neck pain and headache, but may increase the
risk for VBA dissection and stroke. Neck pain and headache are common symptoms of VBA dissection, which commonly precedes VBA stroke. Methods. Cases included eligible incident VBA strokes admitted to Ontario hospitals from April 1, 1993 to March 31, 2002. Four controls were age and gender matched to each case. Case and control exposures to chiropractors and PCPs were determined from health billing records in the year before the stroke date. In the case-crossover analysis, cases acted as their own controls. Results. There were 818 VBA strokes hospitalized in a population of more than 100 million person-years. In those aged 45 years, cases were about three times more likely to see a chiropractor or a PCP before their stroke than controls. Results were similar in the case control and case crossover analyses. There was no increased association between chiropractic visits and VBA stroke in those older than 45 years. Positive associations were found between PCP visits and VBA stroke in all age groups. Practitioner visits billed for headache and neck complaints were highly associated with subsequent VBA stroke. Conclusion. VBA stroke is a very rare event in the population. The increased risks of VBA stroke associated with chiropractic and PCP visits is likely due to patients with headache and neck pain from VBA dissection seeking care before their stroke. We found no evidence of excess risk of VBA stroke associated chiropractic care compared to primary care.

Editor (Jahn) comments

This is a landmark study whose results are very robust with 109,020,875 person-years of observation over 9 years. This article has some interesting comments that the chiropractic orthopedist may want to take note of.

It is also possible that patients presenting to hospital with neurologic symptoms who have recently seen a chiropractor might be subjected to a more vigorous diagnostic workup focused on VBA stroke (i.e., differential misclassification)

Our results should be interpreted cautiously and placed into clinical perspective. **We have not ruled out neck manipulation as a potential cause of some VBA strokes.** On the other hand, it is unlikely to be a major cause of these rare events. Our results suggest that the association between chiropractic care and VBA stroke found in previous studies is likely explained by presenting symptoms attributable to vertebral artery dissection. It might also be possible that chiropractic manipulation, or even simple range of motion examination by any practitioner, could result in a thromboembolic event in a patient with a pre-existing vertebral dissection.

Unfortunately, **there is no acceptable screening procedure** to identify patients with neck pain at risk of VBA stroke. These events are so rare and difficult to diagnose that future studies would need to be multicentered and have unbiased ascertainment of all potential exposures. Given our current state of knowledge, the decision of how to treat patients with neck pain and/or headache should be driven by effectiveness and patient preference.

Omega-3 fatty acids (fish oil) as an anti-inflammatory: an alternative to nonsteroidal anti-inflammatory drugs for discogenic pain.

Maroon JC, Bost JW. Surg Neurol. 2006 Apr;65(4):326-31.

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BACKGROUND: The use of NSAID medications is a well-established effective therapy for both acute and chronic nonspecific neck and back pain. Extreme complications, including gastric ulcers, bleeding, myocardial infarction, and even deaths, are associated with their use. An alternative treatment with fewer side effects that also reduces the inflammatory response and thereby reduces pain is believed to be omega-3 EFAs found in fish oil. We report our experience in a neurosurgical practice using fish oil supplements for pain relief.

METHODS: From March to June 2004, 250 patients who had been seen by a neurosurgeon and were found to have nonsurgical neck or back pain were asked to take a total of 1200 mg per day of omega-3 EFAs (eicosapentaenoic acid and decosahexaenoic acid) found in fish oil supplements. A questionnaire was sent approximately 1 month after starting the supplement.

RESULTS: Of the 250 patients, 125 returned the questionnaire at an average of 75 days on fish oil. Seventy-eight percent were taking 1200 mg and 22% were taking 2400 mg of EFAs. Fifty-nine percent discontinued to take their prescription NSAID medications for pain. Sixty percent stated that their overall pain was improved, and 60% stated that their joint pain had improved. Eighty percent stated they were satisfied with their improvement, and 88% stated they would continue to take the fish oil. There were no significant side effects reported.

CONCLUSIONS: Our results mirror other controlled studies that compared ibuprofen and Omega-3 EFAs demonstrating equivalent effect in reducing arthritic pain. omega-3 EFA fish oil supplements appear to be a safer alternative to NSAIDs for treatment of nonsurgical neck or back pain in this selective group.

Case History

Post-Traumatic Upper Cervical Subluxation Visualized by MRI: A Case Report

Adapted from: Demetrious J: *Chiropractic & Osteopathy* 2007, 15:20.
Full text available at: http://www.chiroandosteo.com/content/15/1/20.

As defined by the Association of Chiropractic Colleges, a subluxation is a complex of functional and/or structural and/or pathological articular changes that compromise neural integrity and may influence organ system function and general health. A subluxation is evaluated, diagnosed, and managed through the use of chiropractic procedures based on the best available rational and empirical evidence [1].

Specific to spinal trauma, evidence of injury can be appreciated via MRI. Ligamentous disruption can produce inflammation that can be readily visualized [2,3]. Annular tears and rim lesions described in cadaveric studies by Taylor and Twomey [4] can be visualized as High-Intensity Zones (HIZ) on MRI [5]. Recent studies have evaluated mechanobiologic issues and diffusion patterns that provide marvellous glimpses of the affect of endplate damage and subsequent disc desiccation/degeneration [6].

Images produced by MRI also provide physiologic assessments of adaptability following injury. Intermediate and late stage adverse effects of biomechanic flaws can be readily visualized utilizing MRI. Vertebral body marrow degeneration has been described and classified by Modic [7]. Atrophic changes of paraspinal musculature are readily visualized using MRI [8,9].

In our case study, MRI provided objective evidence of upper cervical ligamentous injury and components of chiropractic subluxation were demonstrated that led to a refined approach and a favourable outcome (Figure 1).

In this case, chiropractic care was carefully applied and led to the resolution of acute neck pain, associated headache and dizziness secondary to post-traumatic subluxation.

Clinicians must realize that typical cervical spine MRI protocols may not include adequate visualization of CO/C1/C2. Ligamentous injuries may be missed if imaging is not requested of the upper cervical spine. Clinicians should consider requesting additional high-resolution MRI protocols that include occiput-C3. Additional coronal views may provide improved visualization of these structures.

More study to evaluate issues related to the stability/instability of C0/C1/C2 could be undertaken utilizing an upright MRI scanner during cervical flexion, extension, rotation and lateral flexion. Through careful correlation of MRI findings to clinical manifestations, perhaps improved clinical relevance of presumed ligamentous injury can be achieved.

Further scientific investigation is needed to evaluate the role of MRI in chiropractic practice. This unique technology may have the capability of visualizing diagnostic considerations and restorative processes of healing inherent to chiropractic intervention.

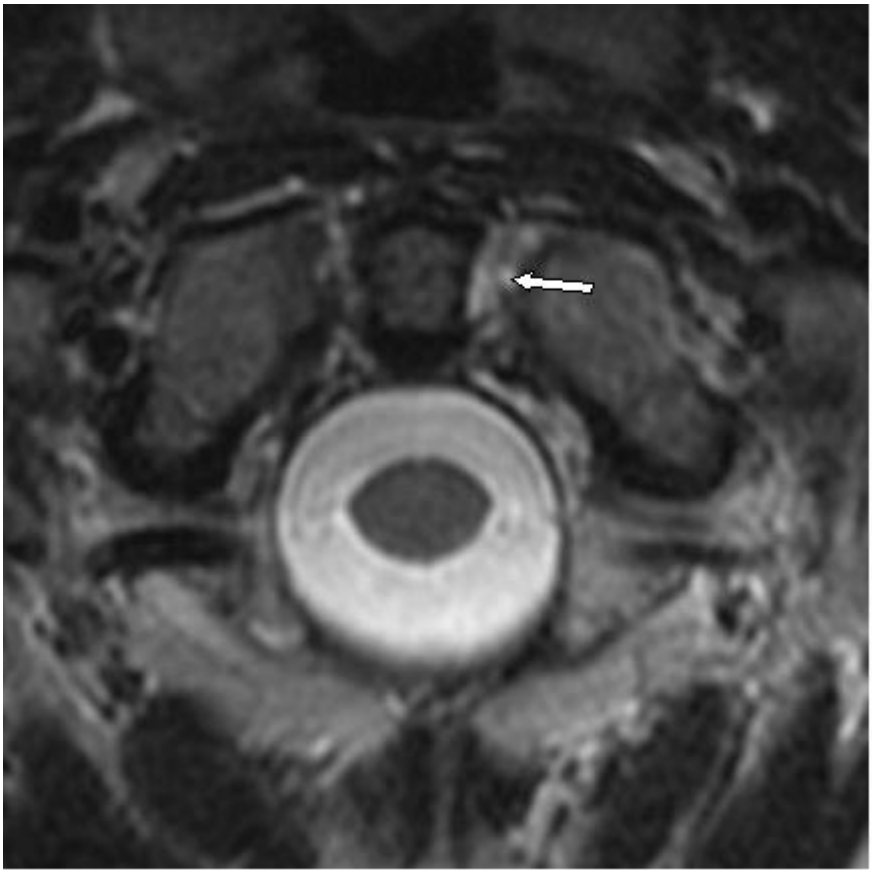

Figure 1. Increased signal intensity (white arrow) on T2WI reveals region of alar ligament disruption.

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The full article text is available at: **http://www.chiroandosteo.com/content/15/1/20.**

Clinical Pearl

Commentary by Philip Van Campen, DC, FACO

V A D

Vertebral Artery Dissection

Office Procedure Protocol

Philip Van Campen, DC, FACO

Up to 50% of patients with VAD "in progress" exhibit symptoms of musculoskeletal complaints, ie: neck pain, decreased range of motion, sub occipital headache, etc. when presenting to a chiropractic office .

"In the presence of a patient who experiences non-traumatic or post – whiplash like neck pain as a new chief complaint, or who refers to this pain as ….unlike anything they have ever had before…, and / or who is experiencing other neurological symptoms, referral for evaluation of possible VAD *prior to* chiropractic manipulation is strongly recommended." [1]

It is entirely possible that a patient may be experiencing a vertebral arterial dissection in progress when initially seeking chiropractic care.

NEUROLOGICAL STATUS

If any of the below neurological symptoms are noted by the physician, the level of suspicion of VAD must be heightened:

Dizziness Diplopia Dysphasia Dysarthria Drop Attacks Ataxia Nystagmus Numbness Nausea

If any the above symptoms are significant in intensity, or present in constellation, the patient must be evaluated to determine whether or not the neurological response that initially drew the doctors attention persists, is progressive, or does not resolve within a few minutes.

DO NOT PERFORM ARTERIAL STRESS TESTING

Provocative testing designed to induce strain on the structures involved should not be undertaken. Recent studies have shown that tests such as George's Test or DeKlynes test may actually cause the condition to worsen. At the present time, there are no safe and reliable tests that will rule out a VAD in progress or identify a patient at risk for VAD and there are no clear cut predisposing factors suggesting a patient at risk for VAD.

CALL 911 IMMEDIATELY

Suspicion of Vertebral Artery Dissection is cause for concern for both the patient and the physician. Proper management of the patient and prompt medical care following VAD may serve to limit the extent of the neurological compromise and embolic ischemia that may occur during such an event. If the symptoms are mild, transient, limited, and resolve quickly, "watchful waiting" may be a prudent approach.

PATHOGNOMONIC PHRASES

If a patient states that they are experiencing a… pain unlike they have ever had before" or a headache that feels like an "explosion" or "thunderclap", be highly suspicious of VAD.

DO NOT RE-MANIPULATE THE PATIENT

Re-manipulation of the cervical spine following an event of VAD may become a complicating factor that might cause the situation to significantly worsen and can turn an unfortunate situation into a terrible one.

DO NOT LEAVE THE PATIENT ALONE

If VAD is suspected, following the 911 call, the chiropractor should stay with the patient as much as possible to monitor any change in neurological status and to obtain baseline vital

signs. Watch for persistent, progressive neurological symptoms and note any change in the degree of mental state or confusion.

INFORM EMERGENCY PERSONNEL THAT YOU SUSPECT VAD

When emergency personnel arrive, direct communication with the responders is needed to portray the urgency of the situation. Description of the doctors suspicion of VAD with expectation of prompt neurological evaluation at the hospital is imperative. Provide them with description of your neurological evaluation.

TIME IS OF THE ESSENCE

Emergency pharmaceutical administration of tPA (Tissue Plasminogen Activator) is most effective if given to the patient within the initial 90 minutes following an infarct. tPA is only moderately effective when administered within 3 hours and is only possibly effective if administered within 6 hours of infarct.

DOCUMENTATION

Comprehensive documentation is essential whenever an event such as this occurs. Honest, straightforward recordkeeping of what transpired during the course of events leading up to this occurrence is crucial in cases where allegations of malpractice are brought forth.

EDUCATE YOUR STAFF

Because the onset of symptoms of VAD may not present themselves immediately following an adjustment, your staff should be aware of any unusual symptoms patients might exhibit whenever they interact with them.

Educate your staff to watch for patients that, if, upon entering or leaving your office, or when calling the office, say, for an appointment, exhibits anything unusual, they need to take measures to inform the doctor of the situation immediately. These unusual signs or behaviors can include such
things as altered or slurred speech, mental confusion, unusual emotional reactions to situations such as inappropriate laughter or sadness, dizziness, unsteady gait or incoordination, or relates a symptom described as "….unlike anything they have ever had before" etc.,

It has been shown in the literature that trivial circumstances such as looking over ones shoulder while backing their car, sneezing, painting a ceiling, nose blowing, getting a shampoo, sexual activity, oral contraceptive use, judo, yoga, etc have all been implicated as a cause of VAD. The evidence is inconclusive, however, to support the claim that VAD *results* from cervical manipulation. Nonetheless, the literature shows that along with major and minor

trauma as a risk factor, the mechanism of VAD is still *associated* with cervical manipulation , although it occurs very rarely. The incidence of VAD temporal to cervical spine manipulation has been shown to be between 1 in 100,000 to 1 in 5.85 million.

The risk of morbidity and mortality following cervical spinal manipulation is "low", and most of the reported cases of injury following cervical spinal manipulation have been performed by non chiropractic practitioners.

Whether a patient experiences VAD temporal to or secondary to chiropractic care is inconsequential. Proper patient management is essential in minimizing morbidity and mortality to the patient.

Editorial Comments

Current Events

Spring 2008 Academy Diplomate Examination

The Academy Orthopedic Diplomate Examination will be held May 3, 2008, at Texas College of Chiropractic in Pasadena, Texas.

The candidate who successfully completes the Academy Orthopedic Certifying Examination will receive the designation *Diplomate of the Academy of Chiropractic Orthopedists* (**DACO)**

Applications are available from the: Academy at www.dcorthoacademy.com

If you wish to participate please contact Cheryl at the Academy office at 515-981-9654.

Special Announcement:

Prior to the AMA Guides 4th Edition, range of motion assessment served as the foundation for defining impairment. With the 4th, published in 1993, the Injury (Diagnosis-Related Estimates - DRE) model was introduced and served as the primary rating method. Most impairments were categorized on the basis of clinical findings resulting in a fixed impairment number for each category; surgery did not modify the original impairment. The 5th Edition, published in 2000, uses both the Diagnosis-Related Estimates (DRE) and the Range of Motion methods dependent on the condition. Categorization was based typically on findings at maximal medical improvement, definitions for the categories were revised, and four whole person permanent impairment values were provided for each category. The 6th Edition, which was released in December, 2008, uses a methodology based on the International Classification of Functioning, Disability and Health (ICF) and ratings are based primarily on a specific diagnosis, which results in assignment to an impairment class (IC), using grids designed for this purpose. The impairment value within a class is further refined by considering information related to functional status, physical examination findings, and the results of clinical testing. Range of motion is no longer used as a basis for defining impairment since current evidence does not support this as a reliable indicator of specific pathology or permanent functional status.

The College on Forensic Sciences (CFS), a CCO and ACA affiliate, has added the AMA's *Guides to the*

Evaluation of Permanent Impairment (6th edition) to the list of impairment rating products sold via its website. CFS offers these products to assist with their organizational objectives of providing, delivering and conducting educational and training opportunities for professionals seeking training in forensics, disability, impairment rating and federal programs leading to board or subspecialty certification.

Order ($20 off the AMA suggested list price) a copy now by going to http://www.forensic-sciences.org. Click on the products button and then impairment rating publications.

Attribution

Ed Payne, FCER,