Title: The Diagnosis and Treatment of the Symptoms Associated with Cervical Spondylosis

Definition and Terminology
Spondylosis of the cervical spine is a specific term used for the progressive results of Osteoarthritis and other degenerative processes with contiguous tissues in that area. Osteoarthritis, the most common joint disease, is characterized by a progressive loss of articular cartilage, a production of appositional new bone in the subchondral trabeculae, and a formation of new cartilage and new bone at the joint margins (osteophytes). To apply the generalized bone changes not necessarily connected to the joints, we find that Cervical Spondylosis (CS) is a process of that same degeneration in all contiguous tissues.

The term spondylosis categorizes these degenerative changes of the vertebral column. CS is specific to the ligaments of the spinal canal and the structures that define the neural foramen: the discs, the pedicles, the end-plates, the joints of Luschka, and the apophyseal or facet joints. Spondylosis is a common occurrence. It involves all these structures and becomes ubiquitous in the later decades. Most imaging techniques are reflected in progressive restriction in range of motion. Therefore, the result is that the specificity of all forms of cervical spondylosis for a particular episode of regional neck pain is so low, and attempts at a specific diagnosis is futile. In this case, aggressive imaging is meaningless at best. Cervical osteophytosis is occasionally considered to cause dysphagia.

Neck pain rarely localizes in the neck. Unusual radiations to the shoulder, suboccioital, and inter- scapular regions are common and overshadow the pain in the neck. This panoply of discomfort must reflect the complex and redundant neuroanatomy of the cervical spine. Even the vertebral arteries are susceptible to impingement and compromise from various osteophytes as they traverse their bony foramina, which result in the rare but distinctive vertebral artery syndrome. This syndrome is identified by symptoms of dizziness, tinnitus, occasional retroorbital headaches, and fleeting blurred vision associated with neck motion. Finally, there are the torticollis or wryneck syndromes. Usually, this is associated with pain and reflects guarded motion in the setting of regional musculoskeletal disease. From what has been observed, the age groups for painless torticollis varies, mimicking the movement disorder expected of phenothiazine toxicity. The etiology is hotly debated with some advocating pharmacological intervention for a neuromuscular disease and others for a psychiatric disorder.

With the observation about the architecture of neural foramina and the likelihood of spondylotic changes in the anatomic components, it is remarkable that more of us do not suffer radiculopathies. It is even more remarkable that most radiculopathies are
intermittent and remittent illnesses. This implies that some components of the spondylotic process, quite likely in associated soft tissues, are reversible.

Two major reasons for provide a consideration to view radiculopathy as a separate clinical issue: one includes benefits the physician and the other for the patient. For the intellectual satisfaction of the physician, there is localization. The pathophysiology may not be definable, but at least we are aware of its location with some reliability. In the view of the patient, radiculopathy portends peripheral damage, provokes a special anxiety since the experience of referred pain appears illogical, and offers some specific therapeutic options.

Table 1 presents the traditional signs and symptoms associated with the compromise of each cervical root. The categorization is considered to be clinically useful, but not necessarily valid or reliable. Generalizations of symptoms and signs, beyond a single root, is common and most likely reflects some multiplicity of innervations peripherally and dispersion of input at the level of the cord.

Table 1  Signs and symptoms of cervical radiculopathies

<table>
<thead>
<tr>
<th>Root</th>
<th>Pain Numbness</th>
<th>Sensory Loss</th>
<th>Motor Loss</th>
<th>Reflex Loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>C3</td>
<td>Occipital Region</td>
<td>Occiput</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>C4</td>
<td>Back of Neck</td>
<td>Back of neck</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>C5</td>
<td>Neck to outer shoulder and arm</td>
<td>Over shoulder</td>
<td>Deltoid</td>
<td>Biceps supinator</td>
</tr>
<tr>
<td>C6</td>
<td>Outer arm to thumb and</td>
<td>Thumb and index fingers</td>
<td>Biceps (triceps)</td>
<td>Triceps supinator</td>
</tr>
<tr>
<td></td>
<td>Index fingers</td>
<td></td>
<td>and wrist extensors</td>
<td>biceps</td>
</tr>
<tr>
<td>C7</td>
<td>Outer arm to middle finger</td>
<td>Index and middle fingers</td>
<td>Triceps</td>
<td>Triceps</td>
</tr>
<tr>
<td>C8</td>
<td>Inner arm to fourth and</td>
<td>Fourth and fifth fingers</td>
<td>Intrinsic,</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>fifth fingers</td>
<td></td>
<td>Extrinsic</td>
<td></td>
</tr>
</tbody>
</table>

A satisfactory explanation of disk nutrition is that the nutritive fluids are imbibed by “sponge like” action of the disk contracting and expanding. This motion is due to alterations of gravity and muscular action. The fluids brought to the disk are further imbibed by colloidal chemical action. This reasoning would partially explain the disk degeneration observed in individuals who are chronically emotionally tense, experience persistent occupational tension, posturally tense, or whose general physical inflexibility decreases their circulatory integrity. This interesting concept provides reason to advocate exercise, and periodic antigravity rest as well as traction.

Degeneration begins in the annulus with slight breaks in the concentric annular fibers and dehydration and fragmentation of the nuclear material. The early stage includes softening of the nucleus and beginning fragmentation of the relatively firm yet elastic gel. Although there is in this initial stage some fragmentation of the annulus, the contents of the disk remain completely encapsulated with no invasion of the annulus.

With a progression of degenerative changes, there is increasing nuclear fragmentation with invasion outward through the rents in the annulus. This sequence continues until the nuclear material is against and held by the longitudinal ligament. In a later stage, either
the longitudinal ligament stretches and pulls away from its attachments, or the nuclear material herniates through this weakened ligament.

As the disk degenerates, the intradiskal pressure decreases, the annulus bulges, and the vertebral endplates approximate. This approximation is enhanced by muscle tone and muscular action as well as by gravity since the opposing intradiskal pressure is diminished. The annulus fibers cease to experience distension stress, therefore, further protrusion of nucleus decreases; but what has protruded now works its way between the ligamentous attachment site and the lax ligament. At this point there are at least three results: this exuded material may be a “soft” disk; if accompanied by annular tissue, it may be a “hard” disk; or there may be osteophytic formation of the raw periosteal site of ligamentous attachment plus calcification of the extruded disk material that forms an osteophyte.

Evolutionary stages of disk degeneration. A) Young, intact disk with elastic annular fibers and a well hydrated nucleus. B) Early stages of degeneration reveal fibrillation of the annulus, some fragmentation, and beginning dehydration of the annulus. C) Moderate stages show a furtherance of B with early invasion of the annulus by fragments of the nucleus. D) Advanced stage of degeneration is that of marked nuclear dehydration and fragmentation with invasion of the shredded annulus permitting nuclear fragments to reach the periphery of the disk where only the ligamentous structure remains.

The vertebral bodies also approximate posterolateral protrusions called the uncovertebral joints of von Luschka. The true anatomy and physiology of these “joints” is, at this point, controversial. In their function as joints with constant irritation and friction, they also become the site of osteocyte formation. These hypertrophic changes of “spondylosis” ultimately affect the entire periphery of the involved vertebrae. The central bulging from the osteophytosis beneath the longitudinal ligament and the thickening of the uncovertebral joints may form an osteophytic bridge or spur completely across the rim of the vertebra and encroach into the spinal canal.

Mechanism of spondylosis. A) Normal anterior portion of functional unit with an intact disk, normal interspace, and a taut posterior longitudinal ligament that is totally adherent to the vertebral body periosteum. B) Disk degeneration permits approximation of the two vertebrae, causing a slack in the posterior longitudinal ligament. The intradiskal pressure dissects the ligament away from the periosteum, and disk material intervenes. C) Extruded disk material becomes fibrous, A, then calcifies into a “spur”, B.

Disk degeneration with formation of “spondylosis”. The vertebral bodies approximate; the uncovertebral joints thicken, roughen, and distort; the foramina deform; and the facets thicken and also deform. There are additional soft tissue changes such as thickening of the longitudinal ligaments and thickening and curling of the ligamentum flavum. The facet capsules also thicken. All these soft tissue changes plus the body changes narrow the intervertebral foramina and the interspinal canal.
Degenerative changes in the cervical spine evolving from the disk changes deform the intervertebral foramina as well as the size and shape of the spinal canal. Degenerative changes make these variations greater and potentially compressive to the tissues in the foramina.

The predominant areas of the osteophyte formation in the entire spine are at the summits of concavity, at the points farthest from the center of gravity. These sites are at C4-5, and C5-6 in the cervical region, at T8 in the thoracic spine, and at L3-4 in the lumbar lordosis. These findings point to the concept that ostophytes develop as a defense mechanism and thus are a repair process instead of a disease state. The osteophytes are ultimately composed of more compact, strong bone than is the rest of the vertebral body. The intended repair function is marred by their potential damage from protrusion into the spinal canal and the intervertebral foramina.

Normal nutrition and lubrication of the posterior articulation (facet). A) This cervical joint is a diarthroidal joint containing a capsule, synovium, joint space containing fluid, and two articular cartilages; and it is supplied by its unique vascular bed. Diffusion cycle to cartilage nutrition. The arterial supply separates into a capillary bed to bone, and a capillary bed to the synovium. Cartilage nutrition is by diffusion through the cartilage, from both capillary beds by “sponge like” compression and expansion of the cartilage. Mechanism of cartilage nutrition. A) No imbibition with joint at rest. B) flow from relaxation or joint extension. C) “Squeezing” out of synovial fluid by cartilage compression. D) Creation of lubrication layer between surfaces by motion between two incongruent surfaces.

The most disk degeneration appears in the lordotic curves. This would form as a result of man’s erect posture. These curves have the greatest static stress; since these areas (lordosis) also have the greatest movement, they are the sites of maximum kinetic stress as well. Trauma, in this respect, undoubtedly plays a part in disk degeneration and in its repair mechanism.

Anterior osteophytes occur most often in the thoracic spine, whereas posterior osteophytes prevail in the cervical (and lumbar) spine. This is consistent with the concept that there is greater pressure on the concave side of the curve.

The existence of faulty posture can accelerate degenerative changes. Poor posture, in which the dorsal kyphosis is accentuated and the compensatory superincumbent cervical curve has increased (extended), brings in all the factors that influence osteophyte formation.

Elastic disks, resilient ligaments, elastic capsule tissue, and openings for nerve and blood vessels that have a good margin of safety adequately handle trauma to a normal spine. Trauma to a degenerated spine finds less compressibility in the disk, less reversible distortion of the disk, weakened or less resilient ligaments, and foramen already narrowed to the point of a smaller margin of safety. The pre-existence of degeneration may have

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been quiescent in that no symptoms were noted, but now minor trauma may “decompensate” the safety margin and symptoms may occur.

Mechanism of osteoarthritic changes in facet joints. The articular cartilage consists of three layers: (1) superficial tangential layer of collagen fibers; (2) an intermediate spongy, shock-absorbing layer; and (3) a deep, calcified, basal layer that is firmly bound to the subchondral bone. Wear and tear causes new bone formation of the subchondral plate and thickening of the calcified basal layer with lengthening of the bone. Peripheral lateral growth widens the end of the bone, and finally, the ligaments ossify.

Encroachment into the intervertebral foramen from osteoarthritis.

Foraminal opening variations. Normal open intervertebral foramen with the neck in a neutral, slightly flexed position and not rotated or laterally tilted to either side. Flexion, which occurs below C3 by forward gliding of the upper upon the lower vertebra, maintains full opening. Extension, by backward gliding of the upper upon the lower vertebra normally narrows the foraminal aperture. Degenerated disk and osteophyte formation from the joints of von Luschka (not considering the soft tissue components).

The slow development of spondylosis allows the body to adjust without experiencing symptoms. This fact is evident by marked osteophytic changes in the X-ray studies, significant narrowing of intervertebral foramina with no nerve root involvement, no painful limitation of movement, and no functional disability. Trauma intervenes in the form of mechanical injury, physical stress, or emotional strain. As a result, the adaption is overwhelmed, the defense is overcome, and symptoms occur. A good compensatory equilibrium needs moderate to large stress to be overcome; if the equilibrium is tenuous, a minor stress will have great repercussions.

**Review of Current Literature**

A review of the recently published works in this subject reveals considerable interest. The studies seem to center on surgical and chemical treatment, however and little is written in the realm of physical medicine. There is one study that focused on the efficacy of home cervical traction therapy that revealed some evidence of short-term pain reduction in only a small group of patients. Studies on provocative range of motion testing and on rehabilitation are scarce but present. Little effect was reported in each. No studies were found using adjusting or manipulation of the spine as a treatment modality.

**Treatment Goals**

The incidence of the symptoms varies in each patient but appears to be cyclic as often as weekly according to Yochum and Rowe. The State of Main has set the Medicare audit level at 52 based on this weekly symptom pattern and need for palliative treatment. With this in mind, routine visits are contraindicated. Visits based on initiation of symptoms and objective signs could be necessary as often as weekly. Relief of the immediate pain, inflammation and dysfunction is the goal.
Management Protocols
The most important part of treatment is protection of the joints from overuse and excessive weight bearing. Weight reduction is a valuable adjunctive therapy. Weight reducing devices such as canes may be helpful in advanced cases. Remediation of the symptoms on a temporary basis may be accomplished by application of heat modalities such as diathermy, ultrasound, interferential current, sine wave currents, infrared lamp and hot pack fomentation. This may augment general mobilizations of the joint under distraction as well as specific articular adjustment. Acute cervical spine symptoms benefit from the use of a collar and traction, particularly when nerve root pain is prominent.

Nutritional Management Protocols
The typical allopathic management for this disorder is analgesic agents such as acetaminophen on a regular basis. The nonsteroidal anti-inflammatory drugs are very popular now but share a wide variety of side effects such as rash, gastrointestinal upset, peptic ulceration, occasional vague psychic reactions and tinnitus. Adrenal corticosteroids are not indicated.

The use of glucose amine sulphate has attracted a great deal of attention. Recently, studies have shown that the use of glucosamine in several forms has produced a remission of symptoms in patients with CS.

Patient Information Sheet
Your condition is called Cervical Spondylosis. That means that the surfaces of the bones and joints that endure stress, bare weight or deal with excessive movement are starting to cause pain because they are beginning wearing out. This happens at different speeds, in different locations and with different types of symptom patterns with every human being who walks uprightly. Everyone is different and will respond differently to the treatment. And there is an effective treatment for the symptoms.

Heat is the best thing for the pain because it brings more blood to the area that is worn. More blood helps relieve the pain and helps the body repair the worn out tissue. It must be understood, however, that the treatment is designed to make things feel better, not to cure them. There is no known cure for the disease at this time. The long-term use of glucosamine in its varied forms has shown a great deal of promise in helping the cartilage regenerate itself.

Things that we do for you in the office include spinal adjustment; this helps to reduce the stress on the joints of the body, helps reduce blood pressure while increasing blow flow, helps increase the nerve activity to injured or diseased joints and affects the metabolic rate of injured or diseased tissues as well. Heat modalities such as hot packs, ultrasound, whirlpool, diathermy and paraffin baths help to reduce the pain and inflammation of the joint surfaces and thus make you feel better. Dietary considerations are also important, as a reduction of refined carbohydrates has shown to reduce the frequency of the symptoms. This may be due to the secondary affect of weight loss that is always helpful to CS.
Things you can do at home include any kind of stress reduction, resting, weight loss, improved diet and mild, non-weight bearing exercises such as swimming, cycling and cross country machine.

You can expect to reduce your symptom severity and frequency if you follow these instructions knowing that with the style of life and stress involved, some symptoms on a periodic basis are inevitable.

References


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QUESTIONS KEY is A for each

1. Which of the following statements best describes the term spondylosis?
   a. A general term categorizing the degenerative changes of the vertebral column.
   b. A general term categorizing the degenerative changes of the vertebral body.
   c. A specific term defining the degenerative changes of the vertebral ligaments.
   d. A specific term defining the degenerative changes of all vertebrae.

2. Which of the following represent the signs and symptoms of a typical radiculopathy of the 5th cervical nerve?
a. Pain in the neck, outer shoulder, sensory loss over the shoulder, motor loss to the deltoid and loss of biceps reflex.
b. Pain in the occipital region, sensory loss to the occiput, no motor loss and no reflex loss.
c. Pain in the back of the neck, sensory loss to the back of the neck, no motor loss and no sensory loss.
d. Pain in the inner arm and fourth finger, sensory loss to the fourth and fifth finger, motor loss to the extrinsic muscles and no reflex loss.

3. What is the treatment goal for the patient suffering from symptoms of cervical spondylosis?
   a. Relief of the immediate pain.
   b. Reduction of the objective findings.
   c. Restoration of range of motion.
   d. Remission of the pathological process.