



Phys Med Rehabil Clin N Am
14 (2003) 455–472

**Physical Medicine
and Rehabilitation
Clinics of
North America**

The anatomy and pathophysiology of neck pain

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This article is more than an anatomy lesson, but it is an anatomy lesson on neck pain. It is more than a treatise on pathophysiology of neck pain, but it is a treatise on pathophysiology of neck pain. Dr. Bogduk has carefully itemized the various anatomic structures that can invoke neck pain. He provides an extensive review of the literature, including but not limited to the outstanding contributions he has made to that literature and to the understanding of the basic and the aging-elderly anatomy and pathophysiology of musculoskeletal, and, in particular, neck pain. He puts in perspective what clinicians know, what they assume, and what they need to understand better in terms of neck pain and neck-referred pain. His critique of many of the accepted items in the difficult diagnosis of neck pain is brilliant and crucial to an understanding and ability to implement appropriate therapies.

—RLS

In preparation for considering the pathophysiology of neck pain, a critical distinction must be made. The neck is not the upper limb. The upper limb is not the neck. By the same token, pain in the neck is not pain in the upper limb, and vice-versa.

For these reasons, neck pain should not, and must not, be confused with cervical radicular pain. Neck pain is perceived in the neck, and its causes, mechanisms, investigation, and treatment are different from those of cervical radicular pain. Reciprocally, cervical radicular pain is perceived in the upper limb, and its causes, mechanisms, investigation, and treatment are different from those of neck pain. Equating the two conditions, or confusing them, results in misdiagnosis, inappropriate investigations, and inappropriate treatment that is destined to fail.

Confusion arises because neck pain and cervical radicular pain are both caused by disorders of the cervical spine, but this common site of pathology

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does not constitute a basis for equating the two conditions. In all other respects the two conditions are totally different.

So critical is difference that pedagogically it is unwise to include the two topics in the same book, let alone in the same chapter. Doing so, as has been the tradition, risks readers' remaining confused and applying to neck pain the interpretations, investigations, and treatment that apply to radicular pain. Traditions and expectations are hard to break. In deference to habit, this article addresses both entities but does so by underplaying cervical radicular pain so as to retain the emphasis on neck pain. Cervical radicular pain has been covered comprehensively elsewhere [1].

Radicular pain

Perhaps surprisingly, little is known about the causes and mechanisms of cervical radicular pain. In the literature, cervical radicular pain has conventionally been addressed in the context of cervical radiculopathy, but that condition, too, is not synonymous with cervical radicular pain.

Cervical radiculopathy is a neurologic condition characterized by objective signs of loss of neurologic function, that is, some combination of sensory loss, motor loss, or impaired reflexes, in a segmental distribution. None of these features constitutes pain.

Many causes of cervical radiculopathy have been reported (Table 1). They share the common feature of compressing or otherwise compromising

Table 1
Possible causes of cervical radiculopathy, listed by structure and pathology

Structure	Pathologic condition	Structure	Pathologic condition
Intervertebral disk	protrusion	Meninges	cysts
	herniation		meningioma
	osteophytes		dermoid cyst
Zygapophysial joint	osteophytes	Blood vessels	epidermoid cyst
	ganglion		epidural abscess
	tumor	Nerve sheath	epidural haematoma
	rheumatoid arthritis		angioma
	gout		arteritis
	ankylosing spondylitis		neurofibroma
Vertebral body	fracture	Nerve	schwannoma
	tumor		neuroblastoma
	Paget's disease		ganglioneuroma
	fracture		
	osteomyelitis		
	hydatid		
	hyperparathyroidism		

a cervical spinal nerve or its roots. The axons of these nerves are either compressed directly or are rendered ischemic by compression of their blood supply. Symptoms of sensory loss or motor loss arise as a result of blockage of conduction along the affected axons. The features of cervical radiculopathy, therefore, are essentially negative in nature—they reflect loss of function. In contrast, pain is a positive feature not caused by loss of nerve function.

For this reason cervical radicular pain cannot be summarily attributed to the same causes as those of radiculopathy. Compression of axons does not elicit pain. If compression is to be invoked as a mechanism for pain, it must explicitly relate to compression of a dorsal root ganglion.

Laboratory experiments on lumbar nerve roots have shown that mechanical compression of nerve roots does not elicit activity in nociceptive afferent fibers [2,3]. Therefore, compression of nerve roots cannot be held to be the mechanism of radicular pain. Compression of a dorsal root ganglion does evoke sustained activity in afferent fibers, but that activity occurs in A β fibers as well as in C fibers [2,3]. Therefore, the activity is something more than simply nociceptive activity. This understanding underlies and underscores the particular nature of radicular pain. Radicular pain is shooting, stabbing, or electric in nature, traveling distally into the affected limb, consistent with a massive discharge from multiple affected axons. Radicular pain is commonly associated with paresthesia, which is consistent with A β fibers being included in the discharge.

There are growing contentions that cervical radicular pain may be caused by inflammation of the cervical nerve roots. This mechanism might be applicable to radicular pain caused by disk protrusions, because inflammatory exudates have now been isolated from cervical disk material [4,5]. Inflammation, however, cannot be invoked as the mechanism of radicular pain caused by noninflammatory lesions such as tumors, cysts, and osteophytes. For these conditions, compression of the dorsal root ganglion is the only mechanism for which experimental evidence is available.

None of these considerations, however, bears on the causes and mechanisms of neck pain. Whatever its cause, and whatever its mechanism, cervical radicular pain is perceived in the upper limb. This manifestation has been clearly shown in experiments in which cervical spinal nerves have been deliberately provoked with needles [6]. The subjects report pain spreading throughout the length of the upper limb. Unlike the sensory loss of cervical radiculopathy, the pattern of cervical radicular pain is not dermatomal. Radicular pain is perceived deeply, through the shoulder girdle and into the upper limb proper. Radicular pain from C5 tends to remain in the arm, but pain from C6, C7, and C8 extends into the forearm and hand. These patterns of distribution indicate that the pain is not restricted to cutaneous afferents. It involves afferents from deep tissues, such as muscles and joints, as well. Because the segmental innervation of deep tissues is not the same as that of skin, radicular pain cannot be, and is not, dermatomal in distribution.

In particular, muscles of the shoulder girdle are innervated by C6 and C7, well away from the dermatomes of these nerves. If anything, the segmental innervation of muscles is a much better guide to the distribution of radicular pain than are the dermatomes. Dermatomes are nonetheless relevant for the distribution of the neurologic signs of radiculopathy, but this distribution of neurologic signs has nothing to do with the distribution of pain.

Neck pain

By definition, neck pain is pain perceived as arising in a region bounded superiorly by the superior nuchal line, laterally by the lateral margins of the neck, and inferiorly by an imaginary transverse line through the T1 spinous process [7]. This definition does not presuppose, nor does it imply, that the cause of pain lies within this area. It defines neck pain simply on the basis of where the patient feels the pain. An objective of clinical practice is to determine exactly the source and cause of this pain and then to implement measures to stop it.

Sources of neck pain

The notion of source of pain is different from that of the cause of pain. A source is defined in anatomic terms and pertains to the site from which pain seems to be arising, without reference to its actual cause.

Potential sources

For a structure to be a potential source of pain, it must be innervated. In this regard, there is abundant information concerning the cervical spine.

The posterior neck muscles and the cervical zygapophysial joints are innervated by the cervical dorsal rami [8]. The lateral atlanto-axial joint is innervated by the C2 ventral ramus [9], and the atlanto-occipital joint is supplied by the C1 ventral ramus [9]. The median atlanto-axial joint and its ligaments are supplied by the sinuvertebral nerves of C1, C2, and C3 [10]. These nerves also supply the dura mater of the cervical spinal cord [10,11]. The innervation of the prevertebral and lateral muscles of the neck has not been studied in modern times, but textbooks of anatomy affirm that these muscles are innervated by branches of the cervical ventral rami [12].

The cervical intervertebral disks receive an innervation from multiple sources. Posteriorly they receive branches from a posterior vertebral plexus that lies on the floor of the vertebral canal and which is formed by the cervical sinuvertebral nerves [13–15]. Anteriorly they receive branches from an anterior vertebral plexus that is formed by the cervical sympathetic trunks [13]. Laterally they receive branches from the vertebral nerve [14].

The vertebral nerve is formed by branches of the cervical gray rami communicantes, and accompanies the vertebral artery [16]. In addition to

giving rise to the sinuvertebral nerves, the vertebral nerve provides a somatic (sensory) innervation to the vertebral artery [17].

Because they are innervated, all of the muscles, synovial joints, and intervertebral disks of the neck are potential sources of neck pain, along with the cervical dura mater and the vertebral artery. Innervation alone, however, is insufficient grounds to credit that these structures can be, or actually are, sources of neck pain. For a structure to be credited as a potential source of pain, physiologic evidence of its potential is required.

In that regard, sources of neck pain have been studied in two ways. In normal volunteers, various structures have been studied experimentally to determine if possibly they can, and therefore could, produce neck pain. In patients suffering from neck pain, the same sites have been anesthetized to determine if doing so relieves the pain.

Normal volunteers

Classic experiments involved the noxious stimulation of posterior midline structures with injections of hypertonic saline [18–20]. These experiments showed that such stimulation produces local neck pain and also somatic referred pain. The distribution of referred pain related to the segment stimulated. Accordingly, stimulation of upper cervical segments produced referred pain into the head; stimulation of lower cervical segments produced referred pain into the shoulder girdle and upper limb.

These experiments were important because they demonstrated the phenomenon of somatic referred pain. They showed that disorders of the cervical spine could produce headache and that they could produce pain in the upper limb. In both instances the mechanism did not involve irritation of nerve roots. The mechanism involves convergence. Nociceptive afferents from the cervical spine converge with afferents from distal sites, on second-order neurons in the spinal cord. Under these conditions, spinal pain can be perceived as also arising from those distal sites.

What these experiments did not show is what the sources of neck pain could be in patients. The experiments targeted the interspinous ligaments as the site of stimulation. Interspinous ligaments, however, are lacking in the cervical spine. The interspinous spaces are filled with fascia and the interspinous muscles. There are no substantive ligaments. Therefore, with respect to source of pain, the experiments must be recast as studying pain from interspinous fascia or interspinous muscles. There is no serious contention that neck pain is caused by some peculiar disorder of these interspinous structures. Nevertheless, the classic experiments paved the way for pertinent experiments.

It has been shown that noxious stimulation of the cervical zygapophysial joints causes neck pain and referred pain. The observations have been corroborated using a variety of stimuli. One series of experiments used a mechanical stimulus, in the form of an injection of contrast medium to

distend the target joint [21,22]. Another used the same mechanical stimulus but also used electrical stimulation of the nerves that innervated the target joint [23]. Both approaches found the same outcomes.

Pain from the cervical zygapophysial joints tends to follow relatively constant and recognizable segments patterns (Fig. 1). From the C2-3 level pain is referred rostrally to the head. From C3-4, and C4-5, it is located over the posterior neck. From C5-6 it spreads over the supraspinous fossa of the scapula. From C6-7 it spreads further caudally over the scapula.

Essentially similar patterns of pain have been produced by mechanical stimulation of cervical intervertebral disks [24–26]. This fact underscores the rule that it is not the structure that determines the pattern of pain stemming from it; rather, the pattern of pain is determined by the nerve supply of the structure. Thus, any structure innervated by the same cervical segmental nerves will have the same distribution of pain. Clinically, discogenic pain cannot be distinguished from zygapophysial joint pain, but the distribution of pain serves as a reasonable guide to the most likely segmental location of its source.

In principle, this rule would also apply to neck muscles. Pain from muscles innervated by a particular segment should be perceived in the same location as pain from articular structures innervated by the same segment. There have been no systematic studies of neck pain from muscles in normal volunteers (save for the classic experiments involving the interspinous muscles). The only study involving neck muscles showed that stimulation of upper cervical muscles could produce pain in the head [27].

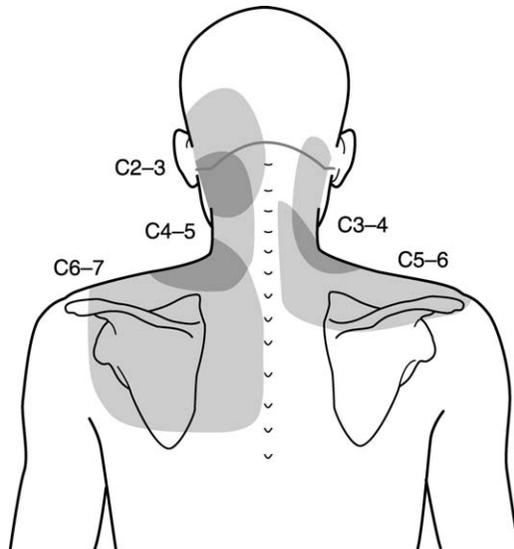


Fig. 1. The distribution of pain following stimulation of the zygapophysial joints indicated.

Other structures that have been shown to be able to produce neck pain and headache in normal volunteers are the atlanto-occipital and the lateral atlanto-axial joints [28]. Pain from these structures does not occur in a unique distribution. Along with the C2-3 joints, these structures all produce pain in the suboccipital region.

Clinical studies

As a complement to the studies in normal volunteers, clinical studies have provided evidence of the sources of pain in patients with neck pain. They involved either the anesthetization or the provocation of pain.

Several studies have shown that anesthetizing the cervical zygapophysial joints can relieve neck pain [29–33]. The most powerful of these studies used controlled, diagnostic blocks, either comparative local anesthetic blocks or placebo-controlled blocks, each on a double-blind basis.

Other studies have used provocation discography to implicate the cervical intervertebral disks as sources of neck pain [25,26,34]. Discography, however, is a capricious test. Even if performed carefully, with attention to testing control levels, it can be subject to false-positive responses [34]. Moreover, in patients with neck pain, it is uncommon to find a single disk that seems to be painful. If all cervical disks are tested, two, three, or more can be found to be painful. Under those conditions, it is difficult to determine whether multiple disks are truly multiple, simultaneous sources of neck pain or whether the patient is simply expressing hyperalgesia. Nevertheless, the clinical data are consistent with observations in normal volunteers that the cervical disks are possible sources of neck pain.

Several studies have demonstrated that the C2-3 zygapophysial joint can be the source of pain in many patients with headache [35,36]. Anesthetizing the joint completely abolishes the headache in these patients. Other studies have reported the same results following anesthetization of the lateral atlanto-axial joints [37–39].

Other tissues, such as the posterior neck muscles, the cervical dura mater, the median atlanto-axial joint and its ligaments, and the vertebral artery, are potential sources of neck pain, because they are all innervated, but they have not been subjected to study either in normal volunteers or in patients. That they could be sources of pain is a credible proposition, but formal evidence is lacking.

Implications

The experimental data, from normal volunteers and from patients, indicate the synovial joints and intervertebral disks of the neck are potential sources of neck pain. Other tissues such as muscles, ligaments, the dura mater, and the vertebral artery, are in theory potential sources of pain. For a structure to be promoted from a potential to an actual source of pain, it must be affected by a disorder capable of causing pain.

Causes of neck pain

Hearsay and imaging have been the traditional basis for listing causes of neck pain. Particular conditions have been regarded as causes of neck pain simply because someone one said they were, or because they can be seen on a radiograph. Both are weak arguments subject to large errors.

Hearsay allows any conjecture to be raised as a possible cause of neck pain, but when these conjectures are listed in textbooks, they tend to assume an undeserved status of veracity. Once a condition is listed, readers tend to accept that it is a possible cause of neck pain, and proponents are excused the responsibility of providing corroborating evidence.

The necessary evidence is some objective test that confirms the presence of the condition and that can be used to show both that the condition occurs in patients with neck pain and that it does not occur in patients without neck pain. For some conditions, the objective test may be a radiograph, but other conditions are not visible on radiographs. For these latter conditions, some other form of evidence is required.

For most entities, an objective test is not available or has not been applied. Consequently, there is no evidence that these conditions cause neck pain; that they do is no more than a conjecture. In some instances, applying objective tests has resulted in certain, sometimes hallowed, entities being refuted as causes of neck pain.

Typical of lists of purported causes of neck pain are those published in leading textbooks of rheumatology (Table 2). The lists are not identical, but there is considerable agreement about several conditions.

These purported causes of neck pain can be grouped in three ways.

- They can be grouped according to clinical significance into serious or nonthreatening conditions.
- They can be grouped into common and uncommon conditions.
- They can also be grouped into valid and nonvalid causes.

Serious but rare conditions are the neoplasms and infections. No one seriously doubts the legitimacy of such conditions as actual causes of neck pain because, by and large, they can be diagnosed by medical imaging and by biopsy, if required. These conditions are, however, rare. In population studies of patients presenting with neck pain, unsuspected tumors and infections have never been disclosed [43,44]. Given the size of these studies, calculation of 95% confidence intervals reveals that serious conditions account for fewer than 0.4% of cases of neck pain.

Overlooked by contemporary textbooks is the importance of vascular disorders in the diagnosis of neck pain. Although headache is the most common presenting feature of internal carotid artery dissection, neck pain has been the sole presenting feature in some 6% of cases [45,46]. In 17% of patients headache may occur in combination with neck pain [46]. Neck pain has been the initial presenting feature in 50% to 90% of patients with

Table 2

Causes of neck pain as listed in three major textbooks of rheumatology, with concordance between sources indicated

Causes	Hardin and		
	Nakano [40]	Halla [41]	Binder [42]
Serious but rare			
Vertebral tumors	++	++	++
Discitis	++	++	
Septic arthritis	++	++	
Osteomyelitis	++	++	++
Meningitis	++	++	
Valid but rare or unusual			
Rheumatoid arthritis	++	++	++
Ankylosing spondylitis	++	++	
Crystal arthropathies including gout	++	++	++
Polymyalgia rheumatica		++	++
Longus colli tendinitis		++	
Fractures		++	
Miscellaneous			
Torticollis	++	++	++
Detectable but of questionable validity			
Diffuse idiopathic skeletal hyperostosis	++	++	++
Ossification of the posterior longitudinal ligament			++
Paget's disease	++	++	++
Spondylosis/degenerative disease	++	++	++
Osteoarthritis	++		
Synovial cyst		++	
Neurologic			
Thoracic outlet syndrome	++	++	++
Spinal cord tumors	++		
Nerve injuries	++		
Myelopathy			++
Radiculopathy			++
Spurious or vague			
Soft tissue injuries		++	
Whiplash			++
Cervical strain	++		
Psychogenic pain			++
Postural disorders	++		++
Fibrositis, myofascial pain	++	++	++
Hyoid bone syndrome		++	
Sternocleidomastoid tendinitis	++		
Fibromyalgia		++	

vertebral artery dissection but is usually also accompanied by headache, typically although not exclusively in the occipital region [45,47]. Although the typical features of dissecting aneurysms of the aorta are chest pain and cardiovascular distress, neck pain has been reported as the presenting feature in 6% of cases [48,49]. All these vascular conditions, however, are

unlikely to be causes of persistent neck pain, because in due course, and sometimes rapidly, they all develop additional clinical features that implicate a vascular disorder.

Less serious conditions are the inflammatory arthropathies. The validity of these conditions as a cause of neck pain is not questioned, because the pathologic condition can be detected by imaging, and these conditions are accepted, recognized causes of joint pain when they affect the joints of the appendicular skeleton. These conditions, however, typically affect the neck in patients with evidence of systemic distribution of arthropathy. They rarely cause neck pain alone.

Polymyalgia rheumatica is a valid entity, but it should not be listed as a cause of neck pain. It is a condition that may involve the neck, but, by definition, it is a systemic disorder that affects other regions of the body as well. It does not present with isolated neck pain. Similar comments apply to fibromyalgia. Regardless of whether one accepts fibromyalgia as a valid entity, it is, by definition, a widespread disorder and not one that enters the differential diagnosis of neck pain as an isolated symptom.

Longus colli tendinitis is a misnomer for a condition better known as retropharyngeal tendinitis [50–57], because the condition involves more than just the tendons of the prevertebral muscles. It involves inflammation and edema of the upper portions of the longus colli (not just its tendons), from the level of C1 to C4 and even to C6 [50–52]. It is a rare condition but can be diagnosed by plain radiography and, most accurately, by MR imaging [52].

Fractures are an accepted cause of pain, although not all fractures are necessarily painful. As causes of neck pain, however, fractures are rare or unusual. Like tumors, unsuspected fractures proved to have zero prevalence in large population surveys [43,44], placing their prevalence at less than 0.4%. Even among patients presenting to emergency rooms with suspected or possible cervical trauma, fractures are uncommon [58–66]. A prevalence figure of 3.5% (\pm 0.5%) is representative.

Synovial cyst is a spurious cause of neck pain. There are no reports of this condition simply causing neck pain. When symptomatic, these cysts cause radiculopathy or radicular pain [67–69]. Accordingly, they do not constitute a differential diagnosis of neck pain.

Torticollis is a clinical syndrome; it is not a specific cause of neck pain. It is characterized by fixed rotation of the head and cervical spine. The neck may or may not be painful, but the presentation does not define the cause, or even the source, of pain. In adults, known causes include basal ganglion disorders, subluxation of the lateral atlanto-axial joint [70–74], and epidural abscess [75]. Speculative causes include subluxations of the zygapophysial joints and extrapment of their meniscoids [76].

Several listed conditions constitute detectable disorders but are questionable sources of neck pain. Diffuse idiopathic skeletal hyperostosis is vividly demonstrable on radiographs of affected regions of the spine, but it is often asymptomatic [41,42]. When symptomatic it causes stiffness and dysphagia

rather than neck pain [41,42]. Similarly, ossification of the posterior longitudinal ligament can be asymptomatic [41,42]. This condition is more likely to present with myelopathy rather than neck pain [41,42].

Paget's disease is, as a general rule, an accepted cause of pain in the body regions affected. Technically, therefore, it is an acceptable cause of neck pain if detected in the cervical spine. One large survey, however, found that Paget's disease is often painless and that patients with cervical spine involvement had no pain complaints referable to that region [77]. This finding gives reason to doubt that Paget's disease is ever a cause of neck pain. When diagnosed radiologically, Paget's disease in the cervical spine may be no more than an incidental finding.

Spondylosis and osteoarthritis are perhaps the most commonly applied diagnoses in patients with neck pain with demonstrable radiographic changes. Neither diagnosis, however, is valid. The radiographic features of cervical spondylosis occur with increasing frequency with increasing age in asymptomatic individuals [78,79]. This finding indicates that these features are age-related changes. Most commonly they affect the C5-6 and C6-7 segments. These changes, however, are weakly, if at all, associated with pain. In some studies, cervical spondylosis occurs somewhat more commonly in symptomatic individuals than in asymptomatic individuals [43,80], but the odds ratios for disk degeneration or osteoarthritis as predictors of neck pain are only 1.1 and 0.97, respectively for women and 1.7 and 1.8 for men [80]. In other studies, the prevalence of disk degeneration at individual segments of the neck is not significantly different between symptomatic patients and asymptomatic controls [81]. Indeed, uncovertebral osteophytes and osteoarthritis were found to be less prevalent in symptomatic individuals [81]. Consequently, finding spondylosis or osteoarthritis on a radiograph does not constitute making a diagnosis or finding the source of pain.

The various neurologic conditions listed in Table 2 are, by definition, not causes of neck pain. They cause symptoms, not in the neck, but in the upper limb. Furthermore, they cause loss of neurologic function rather than pain.

The remaining, listed causes of neck pain are little more than spurious labels. These labels, however, are often applied to patients with neck pain.

Soft-tissue injury means nothing more than something has been injured but there has not been a fracture. Whiplash describes the possible origin of the pain but not its cause or its source. Cervical strain is a totally ambiguous term that implies no more than something went wrong with the neck to produce pain.

Psychogenic pain is a dated, but often abused, term. It is not admitted by the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM IV)* [82]. Unless an alternative, specific psychiatric diagnosis is proffered, psychogenic pain is a euphemism for "I don't know what's wrong" or for malingering.

Although sometimes invoked as a diagnosis, postural disorders may be secondary to neck pain. There is no evidence that some sort of habitual

abnormal posture causes pain. The prospective, longitudinal, long-term data that are available indicate that abnormal posture does not lead to a greater incidence of pain [83].

Although commonly held to be a cause of neck pain, myofascial disorders fail on several counts. The cardinal diagnostic feature for myofascial pain is the detection of a trigger point. There is no evidence that examiners can reliably detect trigger points in the neck; furthermore, the classic trigger points of the neck do not satisfy the prescribed criteria for a trigger point [84]. Indeed, the features of cervical trigger points seem better to describe a tender, underlying zygapophysial joint, and the pain associated with those trigger points is identical in distribution to the pain that would arise from the underlying joint [85].

Hyoid bone syndrome is a poorly studied condition. Its features are said to be tenderness over the greater cornu of the hyoid bone. As such, hyoid syndrome might be included in the differential diagnosis of anterior neck pain, but it cannot be confused with posterior neck pain. The diagnostic criterion is said to be relief of pain upon anesthetizing the cornu [86], but no studies have tested this criterion under controlled conditions.

Beyond its being listed recurrently in textbooks, there is little literature on sternocleidomastoid tendinitis. The cardinal diagnostic feature would seem to be tenderness over the tendons of the muscle, but this manifestation has not been distinguished from random hyperalgesia in patients with neck pain.

Implications

A sober review of the purported causes of neck pain reveals that the most readily diagnosed and serious conditions are rare and do not account for most cases. Meanwhile, the most commonly applied diagnoses lack validity. They have either been disproved by epidemiologic studies or have defied testing. Other entities are descriptive terms but are not proper diagnoses. There are no data on the cause of common, uncomplicated neck pain.

Discussion

For the management of acute neck pain, there is little need for a knowledge of the sources, causes, or mechanisms of pain. Tumors and infections are rare and should be associated with alerting clues from the patient's history. Otherwise, the natural history of acute neck pain is such that most cases will recover regardless and even despite treatment. Indeed, for the treatment of acute neck pain after whiplash, two studies have shown that no more sophisticated intervention is required than a schedule of home exercises [87–89]. Another study has shown that advice to resume normal activities is all that is required [90].

Nevertheless, despite the favorable natural history of acute neck pain, a proportion of patients will develop chronic neck pain. The magnitude of

that proportion is not certain, but 10% to 30% seems a reasonable estimate. For those patients knowledge of the sources and possible causes of pain becomes pertinent, because that knowledge can determine what measures are taken to investigate and to treat the chronic pain.

At present, the only valid data pertain to cervical zygapophysial joint pain. Several studies have now shown that these joints are a common source of chronic neck pain [30–33]. Indeed, three studies have indicated that zygapophysial joint pain is the single most common basis for chronic neck pain after whiplash. It accounts for at least 50% of cases [31,32] and occurs in up to 80% of victims of high-speed collisions [91]. Comparable studies in patients with no history of whiplash have been undertaken.

The cause of zygapophysial joint pain, however, is not known. Post-mortem studies implicate subchondral fractures and contusions of the intra-articular meniscoids [92–94]. Such lesions are consistent with the biomechanics of whiplash [95], but they have defied detection *in vivo*. Contemporary techniques of medical imaging simply do not have the resolution to identify these lesions. High-resolution CT scanning might be able to identify subchondral fractures, if the joints were subjected to serial 1-mm sections, but no one has undertaken such studies. Reports of fractures to the zygapophysial joints have been limited to occasional case studies or small case series [96–100].

Biomechanics studies predict that anterior tears of the annulus fibrosus could also be lesions resulting from whiplash [95], but these, too, have defied detection *in vivo*. Although one MR imaging study reported a rim lesion in a cervical disk in a patient with a whiplash-associated disorder [101], subsequent studies have failed to corroborate this finding [102–107].

More vexatious is providing an explanation for chronic neck pain not related to trauma. Such neck pain cannot be attributed to spondylosis. Even if that label were accepted, it does not provide a mechanism for the pain or its source. There is no known mechanism whereby an ageing disk should spontaneously become painful.

Perhaps attractive is the proposition that osteoarthritis of the zygapophysial joint is basis for atraumatic neck pain. This diagnosis cannot be made on the basis of radiographic findings, however. It requires some other objective test, such as performing controlled, diagnostic blocks of the suspected, painful joint. Such studies have yet to be conducted.

Similarly, for any other purported cause of neck pain, appropriate studies have yet to be conducted. There are not yet compelling data from controlled studies showing that muscles, ligaments, or other cervical structures are the source of chronic neck pain.

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