

# Cervical Discs as a Source of Neck Pain. An Analysis of the Evidence

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## Abstract

**Objectives.** To determine the extent and strength of evidence that supports the belief that cervical intervertebral discs are a source of neck pain. **Design.** The evidence from anatomical, laboratory, experimental, diagnostic, and treatment studies was summarized and analyzed for concept validity, face validity, content validity, and construct validity. **Results.** Evidence from basic sciences shows that cervical discs have a nociceptive innervation, and experimental studies show that they are capable of producing neck pain. Disc stimulation has been developed as a diagnostic test but has rarely been used in a disciplined fashion. The prevalence of cervical disc pain has not been properly established but appears to be low. No treatment has been established that reliably achieves complete relief of neck pain in substantial proportions of patients. **Conclusions.** Basic science evidence supports the concept of cervical disc pain, but epidemiologic and clinical evidence to vindicate the clinical application of the concept is poor or lacking.

**Key Words:** Cervical Intervertebral Disc; Neck Pain

## Introduction

Serious causes of neck pain, such as cancer or infections, are rare. Two population studies, using plain radiography of the cervical spine, each involving >1,000 patients, reported not detecting any serious disorder that was not otherwise suspected from the patient's history [1,2]. These data imply a prevalence of less than 0.4%. Nor are fractures a common cause of neck pain. Even in patients attending emergency departments with suspected fractures, fractures are evident in only about 4% [3].

In patients with chronic neck pain after whiplash, some 55% can be found to have cervical zygapophysial joint pain [4]. For this condition, the mechanism of injury is understood, the pathology that causes pain has been demonstrated, and a treatment that eliminates pain is available [4]. However, for the remaining 45% of patients with chronic neck pain after whiplash, a cause of pain has not been found.

For idiopathic neck pain, the taxonomy of the International Association for the Study of Pain offers a variety of rubrics, such as myofascial pain, trigger point,

muscle strain, and segmental dysfunction [5]. Although attractive to some physicians, and although widely used in clinical practice, these entities lack a scientific foundation because no tests have been validated for their diagnosis, and no studies have shown how commonly such conditions are the cause of neck pain.

A final contender for the source of neck pain is cervical intervertebral discs.

These discs are purported to be a source of pain when affected by so-called degenerative changes; the condition is referred to as degenerative disc disease. Belief in cervical disc pain is common among surgeons who have the option of operating on the supposedly painful disc. For pain physicians, the concept of cervical disc pain is less immediately relevant, for there is no proven intervention that they might offer. However, this might change if emerging, selective, intradiscal therapies prove effective [6].

What should be of concern to physicians, whether they rely on surgeons to treat their patients' neck pain or treat it themselves, is the validity of the concept of cervical disc pain. If patients are to be submitted to invasive

treatments that remove or irreversibly damage tissues, physicians need to be confident that the condition being treated is genuine and likely to be the cause of pain, and not just a spurious idea.

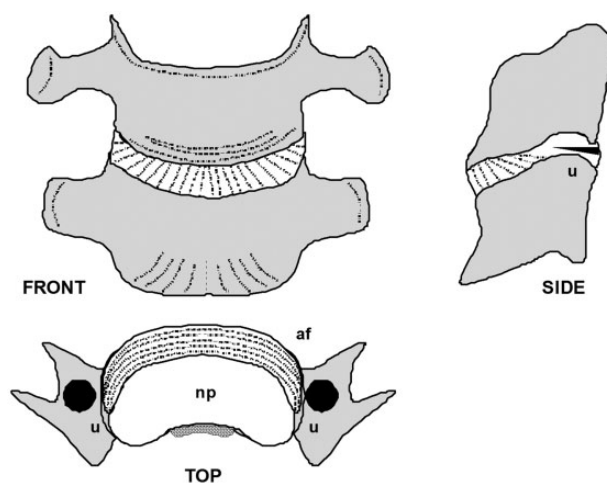
For this reason, the present review was conducted to examine the evidence for cervical disc pain. The review explicitly addresses cervical discs as a source of neck pain. It does not encompass cervical radicular pain caused by disc herniation or foraminal stenosis as this is a separate entity, characterized by pain in the upper limb, not pain restricted to the cervical region.

The available evidence can be stratified into basic sciences covering the anatomy, neurology, and pathology of cervical discs; experimental studies covering pain elicited from the discs; and clinical studies of the diagnosis and treatment cervical disc pain.

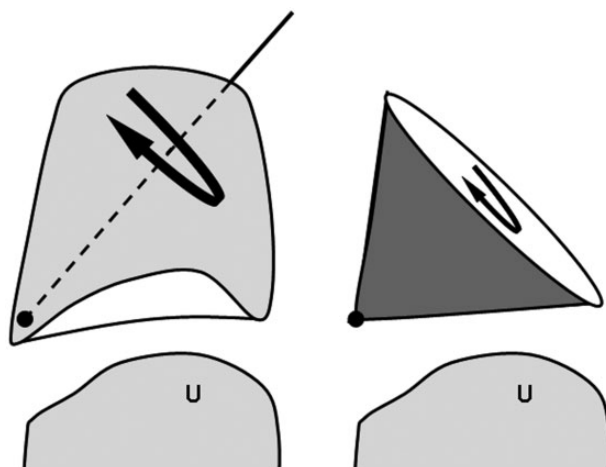
### Anatomical Studies

The intrinsic structure of cervical intervertebral discs is unlike that of lumbar discs and differs with age [7,8]. The nucleus pulposus of cervical discs is gelatinous only in children and young adults. By the age of 30, it dries out to form a firm, fibrocartilaginous plate [7]. Moreover, the nucleus is not surrounded by concentric lamellae of the anulus fibrosus [8]. The anulus fibrosus is largely deficient posteriorly, where it consists of a thin, paramedian band of collagen fibers that run longitudinally between the vertebral bodies (Figure 1). Posterolaterally, the nucleus is covered by the posterior longitudinal ligament, rather than by anulus fibrosus. In axial views, the anulus fibrosus is crescentic in shape and thin posteriorly near the uncinete processes, but thicker anteriorly toward the midline. All its collagen fibers pass in a similar direction: cephalad and medially, effectively aiming at a median point on the lower anterior surface of the vertebral body above (Figure 1). This configuration endows the anulus fibrosus with the structure of a thick interosseous ligament that binds the anterior edges of consecutive vertebral bodies.

This structure of the cervical discs underpins the movements of the cervical interbody joints. In the sagittal plane, flexion is achieved by a combination of anterior translation and anterior rotation of the moving vertebra, across the anterior anulus and the nucleus pulposus [9]. Opposite movements occur during extension. For movements in the axial and coronal planes, the interbody joint operates like a modified saddle joint [9,10]. The anterior end of the moving vertebral body is held in place by the anterior anulus, whereas its posterior end rotates and slides across the ellipsoid concavity between the uncinete processes of the vertebra below (Figure 2). These kinematics can be likened to the movement of a toppled cone, whose apex is fixed but whose base can spin around the longitudinal axis of the cone (Figure 2). In the cervical spine, that axis runs in a plane perpendicular to the plane of the zygapophysial joints.



**Figure 1.** Sketches of the structure of a cervical intervertebral disc in front, side, and top views. Anteriorly, all fibers of the anulus fibrosus run toward a common point on the lower anterior surface of the vertebral body above. In side view, a fissure is evident above the uncinete process (u). In top view, the anulus fibrosus (af) is crescentic: thin posteriorly at the uncinete processes (u) but thick anteriorly. There are minimal longitudinally orientated fibers near the midline, behind the nucleus pulposus (np).



**Figure 2.** Sketches of the movements of a cervical interbody joint in side view. The anterior end of the upper vertebral body is held in place by the anterior anulus fibrosus. The posterior end of the upper vertebral body has a transversely convex lower surface that rotates above and between the uncinete processes (u), around an axis that passes cephalad, and posteriorly, perpendicular to the planes of the zygapophysial joints. This movement can be likened to that of a toppled cone, whose apex is fixed, but whose base is free to spin around the long axis of the cone.

To allow these latter movements, cervical discs must lack a posterior anulus, and transverse fissures must be present across the posterior end of the disc. These fissures are absent at birth but start to develop around the age of nine years [7,11]. They start as clefts in the anulus fibrosus immediately above the uncinete processes and progressively extend medially across the disc, until the clefts

from each side join and form a transverse fissure. This process is completed by the age of 30 [7,11]. The transverse fissure is not horizontal but concave upwards and forwards, parallel to the curvature of the internal surfaces of the uncinete processes. This curvature of the transverse fissure endows the posterior end of the intervertebral disc with an ellipsoid, false joint space, across which the posterior inferior end of the vertebral body can slide and spin.

Understanding the structure and biomechanics of cervical discs is critical to understanding, and not misrepresenting, the possible pathologies that affect them. In the first instance, posterolateral and transverse fissures are normal features; they are not signs of injury or degeneration. Perhaps foremost, the cervical discs are not susceptible to internal disc disruption, as are the lumbar discs [12], because the cervical discs lack a gelatinous nucleus, because their nucleus is not constrained by a circumferential anulus fibrosus, and because cervical discs are not loaded by 50% or so of body weight, as are the lumbar discs. Conversely, because of their essentially ligamentous structure, cervical discs might be more susceptible than lumbar discs to the equivalent of ligament sprains.

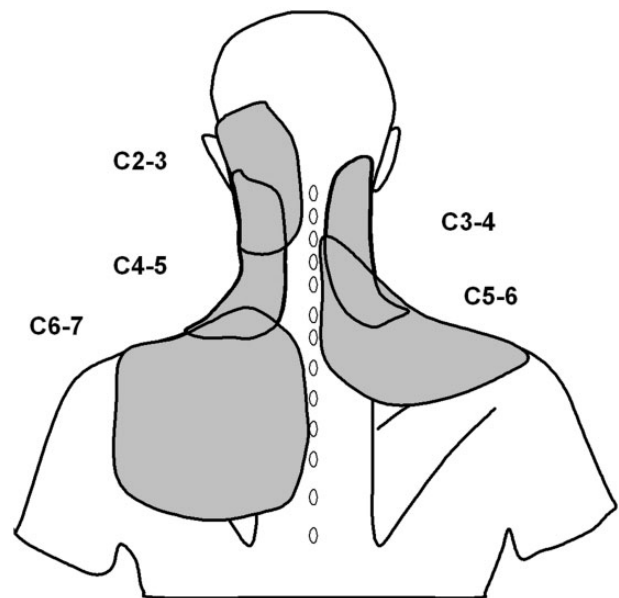
### Innervation

The cervical intervertebral discs receive an innervation posteriorly from the cervical sinuvertebral nerves, laterally from the vertebral nerve, and anteriorly from branches of the cervical sympathetic trunks [13,14]. These nerves feed an anterior plexus that covers the entire length of the anterior and lateral surfaces of the cervical vertebral column and a posterior plexus that covers the entire posterior longitudinal ligament [15]. From the anterior plexus, nerve fibers enter the cervical discs and terminate with free nerve endings in the outer layers of the anulus fibrosus [13,14,16,17]. These fibers are positive for nerve growth factor and for substance P, which is a marker for nociceptive nerves [18]. Other fibers terminate in the anulus and in the anterior longitudinal ligament as Ruffini corpuscles [17].

These anatomic data show that the cervical discs are endowed with the appropriate apparatus for nociception. Therefore, in principle, the discs could be a source of neck pain. There is a suggestion in the data that patients with neck pain attributed to cervical spondylosis have a greater density of free nerve endings, and free nerve endings penetrate deeper into the annulus [17], but technical issues, such as comparing cadaveric samples with operative samples, and small sample sizes, preclude this from being a firm conclusion [19].

### Experimental Studies

In volunteer patients, Cloward [20] exposed the anterior surfaces of cervical discs and stimulated them mechanically by probing and with electrical stimuli. These stimuli



**Figure 3.** Patterns of referred pain from the cervical zygapophysial joints or the intervertebral discs at the segments indicated.

evoked neck pain and referred pain to the medial scapular region. Similar patterns of pain were evoked by stimulating the back of the discs with a hooked probe inserted through trephined holes in the vertebral bodies. These observations established a proof of principle that cervical discs could be a source of neck pain and referred pain.

In asymptomatic volunteers, Schellhas et al. [21], and later Slipman et al. [22], stimulated cervical discs with injections of contrast medium. These injections evoked neck pain referred to various regions, and thereby corroborated the proof of principle established by Cloward [20] that cervical discs could be a source of pain.

The patterns of referred pain from cervical discs (Figure 3) were essentially the same as those found in a separate study in which the cervical zygapophysial joints of normal volunteers were stimulated with injections of contrast medium [23]. This similarity shows that the region in which pain is perceived reflects not the structure that is the source of pain, but the neurological segments that innervate the source. Thus, referred pain from the C5-6 disc is perceived in a location similar to that of referred pain from a C5-6 zygapophysial joint, as the two structures share a similar segmental innervation.

### Pathology

The molecular, histologic, and macroscopic features of so-called disc degeneration do not define a particular or unique disease. These features are expressions of the response of the disc to disturbances or insults to its metabolism [24]. These disturbances may be genetic, in the form of mutated genes for disc proteoglycans or collagen. They can be metabolic, as in deposition of homogentisic

acid or impaired mucopolysaccharide syntheses in diabetes mellitus. Chronic or past infection can induce degenerative changes, as can prolonged exposure to vibration and overt injuries to the disc.

The most common association with degenerative changes is aging. So, in one sense, degenerative changes are a normal age change, not a disease. On the other hand, degenerative changes with age may have a subtle cause that has not yet been elucidated, such as a metabolic deficiency, exposure to carboxyhemoglobin, or cumulative microtrauma [24].

However, degenerative changes are not radiographic signs of pain. Both in the lumbar spine and in the cervical spine, degenerative changes seen on medical imaging have no clinically significant association with pain [25]. They are only marginally more common, if at all, in patients with pain than in asymptomatic subjects, which reflects the fact that degenerative changes are a normal feature of age. If degenerative changes become painful, some process must operate in addition to those features visible on medical imaging. That process has not been established. Therefore, in the meantime, degenerative disc disease, simply seen on medical imaging, cannot be held as a valid diagnosis of neck pain because it cannot be distinguished from asymptomatic age changes.

Postmortem studies have shown that cervical discs can be injured in motor vehicle accidents [26–29]. The lesions demonstrated are hemorrhages in the posterior disc and so-called rim lesions in the anterior anulus. Rim lesions amount to tears or avulsions of the collagen fibers of the anulus. These lesions are not detectable on plain radiographs [26]. There is one report of detecting rim lesions in magnetic resonance images of patients with neck pain [30], but this detection has not been corroborated in any subsequent imaging study [31–35].

## Laboratory Studies

Studies conducted on the *lumbar* discs of laboratory animals provide a model for the molecular mechanisms for disc pain [36]. This model is readily applicable to pain resulting from disc injury. It is less immediately applicable to disc pain in the absence of overt injury, unless the injury is deemed to be subtle, such as microtrauma or a metabolic insult to the disc.

The small-diameter nociceptive fibers in the disc are endowed with neurotrophin (NT) receptors [37], which can initiate a variety of responses: increased production of substance P, sensitization of nociceptors, and nerve fiber proliferation [36]. Release of substance P causes neurogenic inflammation, whereas sensitization of the neural membrane results in hyperalgesia. NT receptors can be activated by a variety of neurotrophins, prominent among which is nerve growth factor (NGF). NGF can be produced by local cells in the nucleus pulposus and anulus fibrosus and by migrating mast cells, macrophages, lymphocytes, and eosinophils [36]. The production of

NGF is triggered by cytokines, such as interleukins (IL-1 $\beta$ , IL-4), tumor necrosis factor- $\alpha$ , platelet-derived growth factor, and transforming growth factor- $\beta$  [36].

When disc tissue is damaged, the affected cells release cytokines, which stimulate the production of NGF, resulting in proliferation and in-growth of nerve fibers, neurogenic inflammation, and sensitization of nerve endings. Nerve in-growth is inhibited by normal aggrecans in the disc matrix but is permitted if those aggrecans are degraded [38]. Painful discs and damaged discs in the lumbar spine exhibit neo-innervation, with a higher density of nerve fibers extending deeper than normal into the disc [39–41], with increased levels of NGF, substance P, and cytokines [38,41–43].

Few studies have explored these mechanisms in cervical discs, but the available evidence is consistent with this model. Free nerve fibers positive for NGF and substance P, together with enhanced expression of NGF, have been detected in human samples of extruded disc material [18]. In rats, experimental injury of cervical discs induces the expression of calcitonin gene-related peptide, which is suppressed by intradiscal administration of anti-NGF antibody [44].

## Diagnosis

No clinical features in the history or on physical examination have been shown to be indicative, let alone diagnostic, of cervical disc pain. There is no evidence that the quality of disc pain is distinguishable from pain from other sources, such as the cervical zygapophysial joints. No-one has shown that movements that aggravate disc pain are different from those that aggravate other sources of pain. The distribution of pain can provide a clue as to the segmental origin of neck pain but does not implicate any particular source.

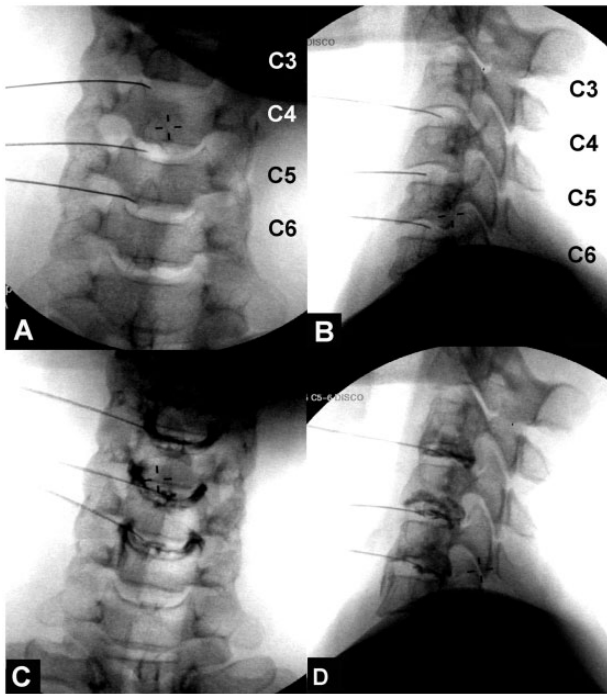
No features on medical imaging indicate that a particular disc, or discs in general, is the source of neck pain. Greater degrees of degeneration do not implicate the affected disc as the source of pain because such changes are common in asymptomatic subjects [23].

The only diagnostic test that has been developed for identifying a painful cervical disc is cervical disc stimulation, previously known as cervical discography [45]. This revised nomenclature evolved because the operant feature is the patient's response to stimulation, not the morphologic appearance of the disc.

Cervical disc stimulation involves inserting spinal needles into the discs to be tested. These include the disc or discs suspected of being the source of pain, or potentially the source of pain, and adjacent discs that serve as controls (Figure 4). In sequence, each disc is stimulated with an injection of a small quantity of contrast medium, and the patient's response is monitored.

The operational criteria for a positive response are that stimulation of a particular disc reproduces the patient's neck pain at an intensity of 7/10 or more on a





**Figure 4.** Fluoroscopy images of stages in cervical disc stimulation. A) Anterior view of needles inserted into the C4, C5, and C6 intervertebral discs. B) Lateral view of needles inserted. C) Anterior view after injection of contrast medium into each of the intervertebral discs. D) Lateral view after injection of contrast medium. Reproduced with permission from Bogduk [45].

pain rating scale, but provided that stimulation of adjacent discs does not reproduce the patient's pain and provided that zygapophysial joint pain has previously been excluded [45]. This last criterion is pivotal, as 67% of positive responses to disc stimulation are false because of zygapophysial joint pain at the same segmental level [46]. A pain intensity of 7/10 or higher is the chosen criterion because in a comparative study [20] the intensity of pain evoked by disc stimulation in asymptomatic volunteers was significantly lower ( $P < 0.0000$ ) than that evoked in patients with neck pain, and an intensity of 7/10 effectively distinguished the two populations.

Another liability of cervical disc stimulation arises in how it is practiced in patients with neck pain. A common practice is to test only the lower three discs—C4-5, C5-6, and C6-7—with the expectation that C5-6 or C6-7 will be positive and the other two discs will be asymptomatic controls. A study has shown that this can lead to false inferences [47]. When all cervical discs are tested, symptomatic discs can be found in addition to, or instead of, the ones at C4-5, C5-6, or C6-7. Consequently, if only three levels are tested and one of these is found positive, the test may be false-positive or incomplete if there is another positive disc that has not been tested. Alternatively, if only three levels are tested and found negative, the test may be false-negative if there is a positive disc at another level that was not tested. Consequently, all discs need to be tested, not just

the habitual three, in order to avoid missing additional or alternatively symptomatic discs.

In theory, this problem might be overcome by closer attention to the patient's pain map, as headache invites consideration of higher discs whereas scapular pain invites targeting only lower discs [22]. However, this conjecture has not been evaluated empirically to determine if reference to pain maps minimizes the false-positive rate of cervical discography when it is performed at only a small number of segmental levels.

In 1976, Roth [48] proposed that painful cervical discs could be diagnosed by anesthetizing the disc with an intradiscal injection of local anesthetic. Curiously, this test was not adopted by others, and its application has not been pursued in the literature. In principle, relieving pain temporarily would constitute a consummate diagnostic test. However, the test would need to be refined to achieve target specificity. It would need to be performed in such a way that the local anesthetic was restricted to the disc and did not leak onto other structures such as the adjacent nerve roots. Furthermore, the test would need to be controlled for false-positive responses using placebo injections or comparative local anesthetic blocks.

When contrast medium is injected into cervical discs, it will often reveal transverse fissures. On frontal views, contrast medium in these crescentic fissures produces an appearance that can be likened to that of a smile, referred to by some exponents of discography as the “smiley” (Figure 4C). These fissures are not signs of pathology; they are normal age changes. Nor is so-called “extravasation” of contrast medium over the uncinate processes a sign of either pathology or the cause of pain. The transverse fissures are normally open over the uncinate process to allow the vertebral bodies to rotate across the disc [9].

### Prevalence

In some circles, cervical disc pain might seem to be a common disorder, particularly among surgeons to whom patients with neck pain are referred for surgical intervention, but no population studies have determined the prevalence of cervical disc pain either in surgeon samples or in the general population. The cardinal reason for this is that a simple diagnostic test for disc pain is not available, and cervical disc stimulation is too invasive a test to be used for epidemiologic research purposes.

The one study that has provided quantitative data in this regard investigated patients with neck pain referred to a private pain practice [49]. Most of the patients had a history of some sort of injury to the neck, including whiplash; only 11% had idiopathic neck pain. In that sample, among patients who completed investigations, 55% had zygapophysial joint pain and 16% had disc pain. These figures show that cervical disc pain does occur in patients presenting with neck pain, but they suggest that its prevalence may be much lower than commonly believed. No

study has produced data that either corroborate or contradict this suggestion.

## Treatment

A favorable response to treatment that specifically targeted a painful cervical disc would constitute compelling evidence to support the concept of cervical disc pain. Such a response would also constitute a powerful criterion standard against which to test the validity of diagnostic tests for disc pain.

There is no conservative treatment designed specifically to treat cervical disc pain, nor is there any minimally invasive treatment. The only such treatment is surgical, in the form of anterior discectomy and fusion (ACDF). In some circles, ACDF is being supplanted by cervical disc arthroplasty in the treatment of cervical radiculopathy and myelopathy, but arthroplasty has not been studied in patients whose sole indication is neck pain. In contrast, ACDF for neck pain has been studied.

There is much literature on ACDF for the treatment of cervical radicular pain and radiculopathy caused by cervical degenerative disc disease or spondylosis, but this condition is not the same as neck pain. Few studies have been published in which neck pain—not radiculopathy or arm pain—was the indication for surgery. Indeed, some surgeons maintain that neck pain alone should never be an indication for surgical treatment [50]. Nevertheless, surgery for neck pain alone has been described.

It is difficult to find an explicit statement of the rationale for ACDF for neck pain. Surgeons seem to have assumed that because this operation is reputed to work for radicular pain caused by degenerative disc disease, it should work equally well for neck pain caused by degenerative disc disease.

A traditional rationale is that ACDF works by immobilizing the affected segment; that is, if neck pain is aggravated by movement, eliminating movement by arthrodesis should relieve the neck pain. However, this rationale is refuted by two lines of evidence. First, it has been shown that, for the relief of neck pain in patients with radiculopathy, cervical disc arthroplasty—which preserves motion—is as effective as ACDF, if not marginally superior [51–53]. Second, anterior discectomy alone, without fusion, is no less effective for neck pain than ACDF [54]. Consequently, the active component of ACDF cannot be immobilization. Therefore, the rationale for ACDF must be that removal of the offending disc removes the source of pain. A competing rationale is that denervation of the disc in the course of its excision disconnects it as a source of pain.

The published studies on ACDF for neck pain are of poor quality because most were published before the modern demand for rigor in reporting outcome studies. The studies are barely more than anecdotal in nature. The authors reported success rates that they claimed to

have achieved, for various definitions of success, but provided little or no corroborating data in terms of pain scores, disability, or use of other health care, either before or after treatment. Most did not use an independent assessor, or they relied on fellow surgeons in the same study assessing one another's patients. In terms of the GRADE system for evaluating the quality of evidence [55], this body of evidence rates as low because it relies entirely on observational studies, and is downgraded to very low because of the imprecision of measuring effects, the risk of observer bias, and the risk of publication bias. An evidence-based review noted such limitations, among others, and concluded that there is low evidence suggesting that patients with axial neck pain without radicular or myelopathic symptoms may receive some improvement in pain and function following ACDF [56].

Table 1 summarizes the outcomes reported in the literature and stratifies the studies according to whether discography was used to determine which disc to treat. The data show that the success rates are low for achieving complete relief of pain. The reputation of ACDF as an effective treatment for neck pain relies on the additional success rates for achieving partial relief of pain, which was variously described in the studies as persisting “discomfort,” “occasional pain,” or pain being “much better.”

Some proponents of discography argue that the success rates of surgery are greater when patients are selected by discography than when discography is not used [68]. The data in Table 1 vindicate this view but only in one respect. The weighted mean success rate after discography ( $75\% \pm 4\%$ ) is, indeed, significantly greater than that when discography is not used ( $55\% \pm 7\%$ ), but this applies only if success is based on combining excellent and good outcomes. It does not apply for obtaining complete relief of pain. For this latter outcome, the reported success rates without discography are patently at least equal to those after discography, or marginally better, but the sample sizes are too small to provide a meaningful statistical comparison.

The success rates of ACDF for neck pain may or may not be sufficiently attractive to pain physicians to justify surgery as a treatment for neck pain. Some have argued that they are not good enough for what amounts to a permanent, irreversible treatment [69]. However, the success rates are too low, and too inconsistent, for ACDF to be a criterion standard for the concept of cervical disc pain.

An impressive criterion standard would be complete relief of pain in a substantial majority of patients. Then it could be claimed that the source of pain had been correctly detected and thoroughly treated. With respect to ACDF, this occurs in only 20–30% of patients. Something else happens in the remaining patients. Their persistent pain is incompatible with the source of pain having been found and successfully eliminated.

The leading possibility is inaccurate diagnosis and, therefore, incorrect patient selection. The disc treated

**Table 1.** Reported success rates for the relief of neck pain after anterior cervical discectomy and fusion, according to whether discography was used to plan surgery

Study	Outcome	Total	Total
<b>No Discography</b>			
Dohn 1966 [57]	No symptoms	35%	61%
	Some symptoms	26%	
Williams et al. 1968 [58]	No symptoms	7%	27%
	Some symptoms	20%	
White et al. 1973 [59]	No symptoms	29%	58%
	Some symptoms	29%	
Algers et al. 1987 [60]	No pain	20%	50%
	Occasional	30%	
Kikuchi et al. 1981 [61]	No pain	40%	40%
Garvey et al. 2002 [62]	No pain or much better		86%
Nyström et al. 2016 [63]	No pain	22%	49%
	Much better	27%	
<b>Discography Used</b>			
Simmons and Bhalla 1969 [64]	No symptoms or some		97%
Kikuchi et al. 1981 [61]	No pain or no interference		80%
Simmons and Segil 1975 [65]	No symptoms	33%	72%
	Some symptoms	39%	
Whitecloud and Seago 1987 [66]	No symptoms	25%	57%
	Some symptoms	32%	
Siebenrock and Aebi 1994 [67]	No symptoms	12%	74%
	Some symptoms	62%	
Palit et al. 1999 [68]	Met expectations	47%	79%
	Not as much as I hoped	32%	
Garvey et al. 2002 [62]	No pain or much better		91%

may not have been symptomatic, or there may have been another source of pain. Discography, when used, may have been false positive. In that regard, none of the published studies excluded zygapophysial joint pain before discography and before treatment, which is a critical prerequisite for cervical discography [45,46]. Other possibilities include that the source of pain was not completely removed or not completely denervated.

## Discussion

Some physicians might be satisfied with concept validity; that is, when the concept sounds plausible, they choose to believe it. However, the fallibility of such faith arises when the concept fails to deliver what it promises. If the concept promises a diagnosis, belief in it becomes flawed if there is no reliable and valid means of making the diagnosis, or if the discipline required to make a reliable and valid diagnosis is not applied. If the concept promises a successful outcome from treatment, belief in the concept becomes flawed if relief of pain is not achieved.

Cervical disc pain is an attractive concept. It provides an explanation for why patients suffer neck pain. The

concept has face validity. Anatomical studies have shown that cervical discs are endowed with the necessary neural apparatus to be sources of pain. Experimental studies have shown that pain can be evoked from cervical discs in normal volunteers. Laboratory studies have provided a model of the complex mechanisms involved in producing nociception from the disc. However, this strong evidence for the concept of cervical disc pain is limited to what effectively amounts to basic sciences.

Unknown is the pathology that renders cervical discs painful. Spondylosis or disc degeneration is not an explanation because it is so common in people who do not have pain. Injuries to the disc fit the proposed mechanisms for nociception from the disc, and postmortem studies have revealed possible injuries, but such injuries remain invisible to contemporary methods of medical imaging. There is no convenient, passive method by which to make a diagnosis of a painful cervical disc.

Cervical disc stimulation has been developed as a means of establishing a diagnosis, but precautions need to be taken to reduce the possibility of false-positive results of this diagnostic test. Steps need to be taken to avoid failing to detect symptomatic discs at segmental levels that are not conventionally studied. Zygapophysial joint pain has to be excluded before attempting disc stimulation.

Disc stimulation has not been systematically applied to determine dependably the prevalence of cervical disc pain. So we do not know if it is common or uncommon. In studies of treatment, in which disc stimulation has been used, it has not been used in a rigorous, disciplined manner. Consequently, we do not know if cervical disc pain was wrongly diagnosed or overdiagnosed in those studies.

Most disappointingly, no convincingly successful treatment has been validated for cervical disc pain. Although surgery might be regarded as the ultimate solution for cervical disc pain, the published evidence fails to vindicate this faith. Some one in five patients are successfully cured. These few patients vindicate the concept of cervical disc pain. A painful disc was diagnosed, that disc was treated, and neck pain was abolished. However, the problem lies with the other four out of five patients. The published evidence does not show if patients with partial relief of pain have their function sufficiently restored to resume a normal life; the evidence does not show if the burden of illness is removed for these patients, in that they no longer require continuing care for their neck pain. Consequently, the clinical significance of incomplete relief of pain remains questionable.

Whereas the basic science evidence invites belief in the concept of cervical disc pain, the clinical evidence falls short of vindicating the belief. Evidence is still required concerning the prevalence of cervical disc pain, its causative pathology, its rigorous diagnosis, and its effective treatment. Consequently, in scientific and philosophical terms, the concept of cervical disc pain remains a worthy

conjecture, but despite its popularity to date, it has not achieved the status of a proven, detectable, and treatable entity.

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