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Intervertebral Discs as a Source of Pain: Is Our Current Model Valid?

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Intervertebral discs have no innervation - or so we were taught back in the 1970s (and even earlier). As we developed better staining methods, researchers discovered that discs did, in fact, have innervation. The corrected notion then became: Discs that have begun to degenerate, or that have been traumatized, may become innervated as part of a pathological process. That thinking served to preserve what we had invested in our paradigm of the time: essentially, that discs are normally insensate and only cause clinical mischief when they herniate or bulge into thecal sacs, spinal cords or nerve roots. This was the basis for the renaissance of the intervertebral disc that began in 1938, when Mixter and Barr performed the very first discectomy. Since then, the "dynasty of the disc" has captured the imagination of surgeons, instrument-makers and inventors of imaging modalities and software.

Also notable among the apparatus of this old paradigm is that discs, once herniated or bulging, cannot naturally regress. Therefore, surgery is the only definitive resort if the severity of symptoms warrant it. This thinking has served the dynasty of the disc well for many years, although forward thinkers and renegades of the profession, such as Alf Nachemson, called for moratoriums on certain forms of disc surgery, arguing that they had not been validated as effective. Indeed, for some conditions, such as degenerative disc disease - for which some 200,000 operations are performed each year in the U.S. - there is still poor evidence of the efficacy of surgical versus nonsurgical management. One expects, at least these days, that class-one evidence (randomized, controlled, double-blind trials) should be the *sine qua non* in support of any highly invasive, anatomically destructive and irreversible surgical procedure. To date, none of this evidence exists in support of discectomy and fusion for that condition, or most other conditions involving the discs.

To some extent, spine surgeons reasonably could dodge the validation issue by arguing that blinded surgery is problematic (although it has been applied recently to knee surgery) and that double-blinding would be

entirely infeasible. The most successful loophole, however, has been that it would be unethical to deny members of a randomly selected control group the surgery that would likely prevent a progression of neurological deterioration (e.g., from radiculopathy or myelopathy), let alone the unnecessary suffering that might be expected from denial of surgical treatment. At face value, it appears a plausible argument, although at least one study has demonstrated that patients electing to forego such surgery fared as well, generally, as those who had the surgery. In fact, careful research of the spine surgery literature does not leave as flattering an impression as spine surgeons would probably like, suggesting that the dynasty of the disc may have reached its zenith years ago.

My surgeon colleagues might take some offense here, but I would remind them that the commonly rather sanguine outcomes reported in this literature (in general, 60 percent to 90 percent of outcomes achieve "good success") should be considered in light of their study designs, which are generally less-than-rigorous. Those that are retrospective in nature offer weaker evidence than longitudinal designs. In many cases, the surgeons themselves rate the outcomes in direct interviews with patients, resulting in a classic form of bias. Perhaps most significantly, few studies have compared surgery to nonsurgical approaches. In most cases, varying periods of physical therapy or other conservative measures have been instituted; however, in other instances, the indicators for surgery prompt early abandonment of conservative measures or a more thorough exploration of other, as-yet-untried methods. The yardstick here is elastic, and these indications vary among surgeons and include clear signs of radiculopathy or myelopathy (or some progression of them); intractable pain beyond some arbitrary time interval; or simply a degree of pain the patient is unwilling to tolerate. Such designs throw doubt into the very efficacy of surgery, and few studies follow subjects for more than a year or two. However, it is known that the consequences of surgical fusion in the cervical spine are accelerated degenerative changes and recurrence of radiculopathy or myelopathy at adjacent levels in the ensuing years^{1,2} - consequences not comprehensively accounted for in studies with follow-up periods of under 10 years.

In the past decade, we have learned that discs do, in fact, regress, and to no insignificant degree (70 percent or more),³⁻⁵ but surprisingly, clinical improvement does not generally correlate with regression, indicating that our notion of the "anatomical compression as cause" paradigm might need some theoretical overhaul. We have learned that discs normally are innervated by branches of the ventral ramus, and that (at least in the cervical spine) they also possess Golgi tendon organ-like mechanoreceptors,⁶ suggesting a possible biomechanical function, or perhaps new, more complicated avenues of pain generation. Studies using

discography, in which the discs are injected under some pressure as a means of provoking the patient's typical clinical pain patterns to implicate the offending disc, have demonstrated a number of things that shake the old dynasty of the disc to its firmament. Discs often can be symptomatic (i.e., they can be the cause of classical pain patterns) even when not bulging, herniated, or vertically compromised, and some cases, the MRI appearance of these offending discs is completely unremarkable. We also have come to realize that discs as low as C3-C4 can cause headache pain, and in some cases, headaches with no neck pain.⁷ In light of this knowledge, it seems rather arrogant to continue to believe we can rule out the disc as the source of pain or other symptoms merely on the basis of the appearance of the spine on MRI, although this remains a common practice.

Another limitation of static anatomical imaging is imposed by the process itself. The subject lies on his or her back in the CT or MRI scanner, with the hips and knees flexed. This is done to stabilize the subject, and also to make the patient as comfortable as possible so the procedure can be tolerated. Interestingly, Nachemson and Elfstrom⁸ studied intradiscal pressure in a group of medical students by placing pressure transducers into the lumbar discs and having the students assume various positions. In this now-classic study, the authors reported that the highest intradiscal pressure was found in the sitting and forward-leaning posture, and the lowest in the supine position, with the hips and knees flexed. Conceivably, standing and sitting subjects the discs to load paths that produce dynamic anatomical changes, which are opaque to the standard CT or MRI scan, leaving clinicians to conjecture whether a 3-mm bulge seen on the conventional MRI might not become, for example, a 6-mm bulge in the standing position. Years ago, I opined that in the cervical spine, extension would result in posterior bulging of those discs.⁹ This was later demonstrated by Epstein,¹⁰ and since then, I have always ordered flexion/extension MRI studies of the necks of patients, rather than neutral-only studies. It is important to remember that the spine is a dynamic structure: Its canal diameter changes with flexion and extension, and discs and ligaments have the potential to bulge, irritating and displacing sensitive tissues. It is encouraging to see that weight-bearing MRI has emerged in the marketplace. Better late than never!

Another source of pain emanating from the spine is the endplate. Fractures of the endplate can allow otherwise sequestered antibodies contained within the *nucleus pulposus* to mix with the peripheral circulation, and animal studies support the potential for local autoimmune reactions, which can produce inflammatory pain. Such injuries also result in dessication of the nuclear material, with resulting change in disc load distribution¹¹ imposed by the loss of the nucleus. This can have the short-term effect of altering

spinal biomechanics, and the long-term effect of accelerating disc degeneration, as a result of the shift toward peripheral disc-loading paths adjacent to vertebral rims composed of inflexible cortical bone. Another consequence of endplate fracture is a disruption of the normal, passive diffusion of nutrients between the vertebral circulation and the disc, which takes place across the endplate. After fracture, scarring occurs, which can diminish or prevent this diffusion. The result is a gradual loss of the disc's component cell population and vertical dimension. Again, the long-term effects are spondylosis and its clinical consequences.

How does one diagnose endplate fracture? In most cases, standard radiographic procedures will not disclose the fracture. Some have suggested that fractures of this type may be visible on MRI in the early stages, but this has not been well-documented. We studied the problem using single photon emission computed tomography (SPECT) and reported a high number of probable endplate fractures in a group of subjects suffering from the late consequences of whiplash.¹² (I caution, however, that the average VAS score in this group of patients was 7.8, so they may not be representative of most long-term sufferers. Moreover, using SPECT, we can only make a presumptive diagnosis of endplate fracture.)

Recently, I served as a consultant on a "rollover" case, in which the patient sustained a pedicle fracture that was diagnosed at the hospital on the day of the crash. She did not subsequently respond to conservative medical care and was eventually examined by videofluoroscopy, which did demonstrate some ligamentous subfailure. However, on two occasions, many months apart, she underwent MRI scanning of her cervical spine, both of which were read as normal. Given the nature of the trauma, the index of suspicion for endplate fracture remained high in my mind, and a SPECT was recommended. It was "hot" from C2 through C7, indicating a widespread active process, but again, its appearance offered no definitive explanation for her continued pain. Still suspecting endplate fracture, I recommended a CT scan with a beam strength midway between the standard bone window and soft-tissue window, hoping to delineate the endplate. I also specifically asked the radiologist to look for this type of lesion. This study confirmed endplate fractures at two spinal levels which, in concert with the healed pedicle fracture, no doubt contributed to the SPECT appearance.

In conclusion, it has long been evident that discs can herniate or bulge, and thereby produce clinical effects by compressing neural or other paraspinal structures. This is the well-known and undisputed anatomical effect of disc pathology. It also has become evident that discs possess pain-sensitive nerves and (in the cervical spine at least) a small number of mechanoreceptors whose function is currently unclear, but may

involve active biomechanical participation. Moreover, disc bulges and herniations - once thought to be permanent consequences of trauma or degenerative changes - often undergo some degree of regression, albeit poorly correlated with clinical recovery.

Static anatomical imaging modalities, such as MRI or CT, probably do not allow us to assess the full extent of disc pathology (that might occur within the dynamic loading boundaries of active range of motion under weight-bearing conditions) as a result of the relative unloading of the discs imposed by traditional non-weight-bearing and neutral positioning techniques common in their acquisition. (One wonders what further effect might be imposed by conditions of everyday activities, such as heavy lifting.) Moreover, discography studies have demonstrated that even mid-cervical discs can cause headaches - even in the absence of spinal pain - and that discs can cause spinal pain patterns even though no abnormalities (morphological or signal appearances) can be appreciated using standard MRI techniques. Disruptions of endplates, another diagnostic dilemma, can also lead to spinal pain via a local autoimmune reaction, and are mentioned here because of their intimate relationship with the discs.

Collectively, this knowledge challenges our older, simpler notions about anatomical models of discogenic pain, which traditionally have prompted radiologists to search chiefly for herniations and bulges as a source of trouble, and which beg for the development of a new disc paradigm. Naturally, this new knowledge also necessitates a new paradigm for therapy and questions the reasonableness of surgery in some (and perhaps many) cases. Medicolegal questions also will be on the periphery of this debate. For example, if it has been shown that conservative care may be equally effective in the management of some forms of disc lesions, and such care can be provided with less invasiveness and risk (and at lower cost), is a spine surgeon broaching some ethical boundary by failing to offer or recommend this form of care - say by a chiropractic physician - prior to moving on to a riskier and more final surgical approach? In my opinion, the clear answer is "yes." That is, after all, one of the pillars of informed consent - but that is the subject of another discussion. In the meantime, it is time for a shift in thinking about discs and what to do about them.

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