DEGENERATIVE DISEASE OF THE ADULT LUMBAR SPINE

A Methodological Approach to the Diagnosis and Surgical Treatment of Back Pain

Back pain and sciatica secondary to degenerative disc disease of the adult lumbar spine are incredibly common medical complaints heard repeatedly by physicians. In spite of this mundane complaint most physicians, including both neurosurgeons and orthopaedic surgeons, continue to be perplexed by this entity, which exasperatingly remains fraught with confusion, controversy, and obfuscating terminology. Even its basic cause remains a mystery to most. The public is perplexed and frustrated by an expensive and seemingly haphazard series of oftentimes trendy and ineffective treatments. New ideas and concepts continually embellished by technology and jargon persistently rise up succeeding to further mystify and surround this ubiquitous medical entity. A quick demonstration of this latter point is made when one considers the relatively recent re-introduction of posterior spinal fusion and anterior spinal fusion techniques supplemented by a maddening and expensive array of hardware; the ubiquitous, confusing, and inappropriate use of MRI; and the never-ending rush to develop an expanding and diverse assortment of minimally invasive surgical techniques. Amidst all of this the very source of back pain remains controversial and not universally defined. The terminology and nomenclature used to describe back pain and the various medical syndromes associated with degenerative disc disease are imprecise, misleading, and often inaccurately applied. Inaccurately ascribing the clinical situation to muscle pulls, muscle cramps, low potassium, arthritis, fibromyalgia, dysfunctional psychic states, etc. illustrates this point. Frequently the clinical problem is inappropriately ascribed to muscle pulls, muscle cramps, low potassium, arthritis, fibromyalgia, and a variety of other maladies. The list is endless. Lacking universal definition and precise nomenclature one is led further afield as the confusion and frustration are heightened.

This entire disarray is further fueled by the expanding variety and ubiquitous use of diagnostic modalities presently available, most notably MRI, but also including including EMG’s/NCV’s, myelography, CAT scans, and discograms. Each of these modalities has its advocates, who are often not clear as to what the test seeks to find, what has been found after the test is performed, and what to do with the information after it has been collected. Hence, the assembled information is often misinterpreted and misapplied, and instead of shedding illumination upon this enigmatic entity, back pain becomes further immersed in pseudo-science, controversy and mysticism. The final result is often inadequate medical treatment and/or failed surgical results. Out of this morass continue to spring various medical practitioners, theorists, and cultists, each advocating their own diagnostic and therapeutic modalities, often based on nothing more than convention, prejudice, untested hypotheses, and politics. (McGinty)

A non-exhaustive list of non-surgical treatments and procedures currently available includes mobilization and/or immobilization (read “rest”), physical therapy, William’s exercises, non-steroidal anti-inflammatory drugs (NSAIDs), and epidural steroid injections. The ever expanding and diverse array of surgical procedures currently in vogue are as controversial as they are varied and include: “minimally invasive surgery”, which itself includes partial, microscopic, percutaneous, endoscopic, and laser discectomy; complete discectomy; laminotomy; laminectomy; and an expanding collection of anterior and/or posterior fusion techniques. include: “minimally invasive surgery”, which itself includes partial, microscopic, percutaneous, endoscopic, and laser discectomy;

A historical perspective helps to understand this situation and serves as an insight suggesting caution. Historically spine fusion as a surgical procedure had experienced a general waxing and waning in favor, but in recent times, most notably posterior spinal fusion, it has undergone a resurgence, but this time supplemented by a maddening array of hardware devices and pedicle screw fixation systems, which are theoretically designed to enhance the spine to fuse. Unfortunately this has led to a paradigm whereby the primary goal of the practitioner is the successful fusion of the spine and not the successful relief of back pain. Fusion of the spine evolved from the concept of fusion of other arthritic joints such as the knee or the hip and finds its intellectual support in the mechanical paradigm of back pain and its ambiguous spinal corollary of “instability”. The idea is that some form of aberrant biomechanical motion, i.e., instability, is the fundamental cause of back pain. Armed with this idea, it appears logical to many practitioners that if this motion can be stopped by fusion, then the pain should go away. Moreover, if the first fusion procedure doesn’t work, then a second fusion procedure, perhaps more extensive than the first should be the goal of surgery. It appears that if the relief of back pain is not the result, then the goal should be to make the spine fuse ever more rigidly regardless of the conceptual inadequacies of the technique and its implications for the morbidity of the patient. If the relief of back pain is not the associated result, the practitioner and the manufacturer of the hardware seem to think it is not the fault of the procedure but of other less defined and poorly conceived parameters. The spine fusion advocates are apparently on a never-ending quest to make the spine “fuse”. Now in vogue with some practitioners of the art of spinal surgery is the combination technique of posterior spine fusion followed by anterior spine fusion on the same patient, both supplemented with hardware. These developments have been given impetus by enhanced metallurgical knowledge and the technological explosion in medicine, which together generate continuous design modifications. The bone grafts utilized to create the fusion have also demonstrated a great deal of diversity, the results of which have not always been favorable. These grafts run the gamut from autograft (from the same person) to bovine heterograft (from a different species) and include biological as well as non-biological “osseous-inductive” (designed to induce bone fusion) materials. Unfortunately, these grafts often fail to fuse, stimulate immune reactions, or become infected.

The author’s observations of this situation, supplemented by his own study and capable clinical experience, have provoked him to formulate an approach to disc disease, which combines sound medical basics, modern medical science, and sound empirical observations into a logical approach to the treatment of back due to degenerative disc disease. Armed with this insight it becomes obvious that the mechanical paradigm, although historical medical dogma in America, is simply inadequate to explain back pain and serve as the rational basis for its treatment. For example, fusion procedures have not being scientifically validated and have not been shown to be associated with a relief of back pain even when technically successful. Quite astonishingly, they continue to be performed without a clear demonstration of their benefit or specific delineation of their indications as scientifically based and verifiable outcome data are totally lacking. There is not even a universal standard by which to judge such outcomes. The reports discussing these procedures, which must be judged quasi-scientific at best “contain precious little objective information to substantiate such approaches, particularly in the difficult patient population on which these operations are performed”. (Hanley). In the United States more of these spinal procedures are performed than the whole rest of the world combined, and doubtlessly, this recent escalation in fusion surgery is propelled not only by the technological explosion in medicine, but also by the concomitant increase in the number of “trained spine surgeons”—a title not necessarily synonymous with “spine physician”. Needless to say the obvious economic implications of this situation are immense.

Finally, there begins to emerge in this abysmal sea murmurings scientific evidence that the source of discogenic back pain is something other than aberrant biomechanics, i.e., “instability”, but rather can be found in the chemical mediators of an inflammatory reaction. Therein, lies the very
challenge to the notion of the mechanical paradigm of back pain as the intellectual basis for spine fusion as the treatment of back pain and sciatica due to degenerative disc disease. Unfortunately the mechanical paradigm has insidiously and fallaciously clouded and restricted practitioners’ thinking while at the same time providing an impetus to non-scientific modalities such as spine fusion in the treatment of back pain. In order for the physician to escape from the mechanical paradigm of back pain in which he finds himself, he must understand the inflammatory process as the fundamental source of degenerative disc disease and forego the notion that the use of hardware and metal devices somehow enhances bones to heal, stops motion, and thereby stops pain. Indubitably, this is not the case and this false concept has led to devastating results and numerous human tragedies.

As a result Dr. Coniglio has come to the realization that inflammation and not aberrant motion is the ultimate source of back pain. Derived from the fundamental principles described above and based on a meticulous and thorough approach to the patient, the judicious use of imaging studies, and coupled with his training and experience, he has developed an approach to the treatment of back pain due to degenerative disc disease, and, when appropriate, a conservative, repeatable, and reasonable surgical technique. This same fundamental surgical technique is used in all cases of disc herniation, spinal stenosis, failed back surgical syndrome with or without previous spinal fusion, spondylolisthesis, and chronic back pain, because the basic cause of the pain is the same no matter what the clinical syndrome. Because of its repeatability, simplicity and logic the surgical procedure demonstrates consistently low morbidity and cost.

The purpose of this report is to educate the reader as to the nature of back pain due to degenerative disc disease, to illustrate the pre-operative evaluation, demonstrate and discuss the surgical technique, and to present a series of cases utilizing this approach with an emphasis on the chronological implications. Thus, armed with this foundation a process of thinking can be derived, which will benefit both the patient and the physician.

Methods and Approach

Definitions and Fundamental Concepts.

A. The Morphology of Degenerative Disc Disease; or, “What is degenerative disc disease of the lumbar spine”?

1. Degenerative Disc Disease: “Degenerative disc disease of the adult spine” [hereinafter DDD] is a generic term meant to include all those other terms and processes that have as their initiating event an injury or alteration to the disc annulus and the subsequent disc space and associated facet joint changes that inevitably result from these instigating events. Contrary to much historical speculation and erroneous thinking the fundamental element in the initiation and production of DDD is inflammation and not abnormal motion euphemistically termed “instability”. It is true that traumatic events in about 1/3 of the cases seem to herald the onset of low back pain. Nevertheless, if a meticulous history is derived from the patient by an adequate interview it is usually seen that the patient had some back pain prior to the supposed traumatic event. Furthermore, even if there was no precedent traumatic event, the chronic nature of back pain due to DDD is certainly due to inflammatory changes in the disc itself.

Pathologically DDD is simply arthritis, and the changes that occur in the spine secondary to DDD are the exact same changes that occur in any other joint in the body secondary to degenerative osteoarthritis in any other joint and are solely due to inflammation. It is
fallacious, misguided and confusing to imply that these processes are any different merely because they occur in different locations in the body. After the initial event in the annulus fibrosus of the disc, whether it be traumatic or otherwise, the disc and its respective pair of synovial facet joints undergo bony hypertrophy, subchondral sclerosis, disc space (or joint) narrowing, and osteophyte (“spur”) formation. The ultimate patho-anatomic result of this process is circumferential narrowing of the spinal canal due to a combination of anterior disc bulging or herniation and posterior bony thickening of the synovial facet joints. Nerve root compression, back pain and/or leg pain and associated disability are the inevitable results.

2. Instability: Physicians have often used the term “instability” to indicate the presence of back pain due to DDD, but this approach and terminology is to be condemned for there is no scientific explanation of this concept nor definition as to what this term means on a patho-anatomic basis. There are as many different definitions of this term as there are practitioners engaging in the business of treating low back pain. Instead, it has come to represent an undefined abnormal motion pattern, which, without explanation or demonstration, is generally presumed to exist by spinal surgeons and serves to rationalize the performance of spinal fusions. Astonishingly, at various times different practitioners have implied that “instability” could mean increased motion or decreased motion.

3. The Natural History of Degenerative Disc Disease: After the concepts of inflammation and instability as they relate to DDD are recognized, the connection between “disc herniation” and “spinal stenosis” can be easily appreciated for these two terms simply imply the opposite patho-chronological ends of the same disease process. Ideally, disc herniation is the disease of the very young and spinal stenosis is the disease of the very old. It is important to realize that the disc injury for whatever reason and no matter what the form is the initiating event. It is followed by development of long term arthritic changes in the facet joints, which anatomically bound the lateral recesses through which the nerve roots traverse the spinal canal for a distance of about 2.5 cms. before they exit the spinal canal via their respective nerve root foramina. These arthritic changes of the synovial facet joints most prominently consist of bony hypertrophy (enlargement) and sclerosis (hardening) entrapping the nerve roots in the lateral recesses producing the clinical phenomenon of spinal stenosis. Spinal stenosis develops over a long period of time. Since most people are somewhere between these two extremes, it is logical to assume that any one individual with DDD has components of both pathological stages. In fact most people don’t have a pure herniated disc syndrome or a pure spinal stenosis syndrome. Therefore, intuitively, when considering the design of a surgical procedure, one does not have to consider a separate procedure for each of these entities, for it is more preferable to develop a single surgical concept that simultaneously deals with all the affected components.

4. Patient Evaluation – History, Physical Examination, and Diagnostic Studies:

   History: As in the evaluation of any patient, a precise and meticulous history and physical examination must be performed. Much about the pathological process can be ascertained by careful and precise use of these simple, basic, and non-expensive hands on techniques. Generally, back pain due to DDD is easily recognized and its presence readily confirmed by the experienced physician. No esoteric and complicated tests need be done. Moreover, much information can be obtained by careful interviewing of the patient. Patients with a disc problem usually have pain with sitting and lifting; those with spinal stenosis generally have pain with walking, sleeping, and standing. For example, patients with L5 radiculopathy emanating from the L4-5 interspace usually have pain/numbness and tingling involving the great toe, dorsal surface of the foot, and weakness of dorsiflexion of the great toe; those with
S1 involvement usually have L5-S1 interspace dysfunction and have pain/numbness and tingling of the lateral side of the foot, weakness of plantar flexion of the foot, and decrease of the S1 ankle reflex. Plain x-rays are confirmatory and no more studies need be ordered at this stage. The treatment at this early state is non-steroidal anti-inflammatory drugs, bed rest, physical therapy, and possibly epidural steroid injections.

Diagnostic and imaging studies are meant to corroborate and confirm the clinical impression when necessary, not to preempt it. Generally, the MRI is an overused tool that is used too early and often necessarily. It provides too much unimportant, confusing, and unnecessary information and precious little significant information. For example, if surgery is not the immediate concern it is unnecessary; the bony detail is not nearly as good as that of the CAT scan; and, when surgery is contemplated, it lacks the dynamic flow characteristics of the myelogram/CAT scan, which show subtle pressure changes of the nerve roots as they traverse the bony lateral recesses prior to their exit from the spinal canal. Because of these factors, myelography and simultaneous CAT scan is the preferred pre-surgical diagnostic examination.

*M Myelography and simultaneous CAT scan* in spinal stenosis is the quintessential diagnostic spinal test, there being no pre-surgical study that can evaluate the spinal canal, nerve roots and spinal cord as completely and with such detail as the water soluble myelogram followed by the CAT scan. The myelogram/CAT scan shows the gamut of changes in the lumbar spine including compressive phenomena, bony pathology and disc alterations. The myelogram/CAT, e.g., often shows no anterior disc bulge, but rather can demonstrate impingement of the nerve roots in the lateral recesses by the posterior bony elements with associated nerve root swelling. In pure herniated disc syndrome an anterior disc bulge is generally seen with little or no lateral recess stenosis depending on how long the patient has had symptoms. On the other hand in a patient who has had long standing degenerative disc disease a complete myelographic block to the flow of myelographic dye is sometimes seen due to lateral and posterior bony hypertrophy. There is no other test that shows the subtle pressure alterations in the spinal canal and lateral recesses and is due to the flow characteristics of the low – viscosity myelographic dye.

*Discography* is the test that is done when the diagnosis is uncertain. For example, often people have back pain and little or no leg pain and the diagnostic tests are equivocal. A discogram done with the patient sedated by injection of radiographic dye into the discs can show an abnormal picture of the injured disc and/or reproduce the patient’s symptoms. Both of these results reveal the presence of an injured disc when no other test has been informative. The presence of such a disc injury with no herniation to produce sciatica is the classic description of the Internal Disc Disruption [hereinafter the IDD] as opposed to the herniated (bulging, extruded, etc.) disc. The surgical answer is not to perform a fusion, but rather to remove the diseased disc.

5. **Pain Level and Interpretation:** Confounding the evaluation of the treatments for DDD is the almost unbelievable realization that even today there is no standardized method or criteria to communicate the pain level of patients’ back pain. Therefore, mild pain in the view of one practitioner or patient can be severe in the view of another. A satisfactory surgical result for one doctor can be a poor result for another. Therefore, because it is necessary to understand what one is talking about when he attempts to rationally communicate with another regarding back pain, and the relief of back pain is the purported goal of those who treat DDD, then this practitioner has established empirical parameters to delineate different degrees of back pain. They are indicated by the criteria as follows:
a. *Slight Pain*: The pain has no effect on work or recreational activities; the patient takes no medication stronger than aspirin or non-steroidal anti-inflammatory drugs [hereinafter NSAIDs]
b. *Moderate Pain*: The pain occasionally causes the patient to miss work or the patient can’t perform some desired recreational activities; and/or the patient persistently takes medication such as narcotics, sedatives, muscle relaxants, or NSAIDs.
c. *Moderately Severe*: The pain is moderate, the pain is present at all times. Pain pills and muscle relaxants often don’t provide relief; the patient repeatedly misses work, and/or cannot perform most desired recreational activities.
d. *Severe*: The pain is so severe that the patient can’t work or perform any recreational activities; medications, physical therapy, rest, or epidural steroid injections provide no relief. The patient is effectively completely disabled.

6. **Surgical Procedure**: The surgical procedure was the natural outgrowth of the intellectual exercise by which the spectrum of DDD syndromes are conjoined and correlated. Because the patho-anatomic components of DDD include both disc and facet space abnormalities, then both the disc space and facet joints must be taken into consideration when the surgical procedure is planned. Since the disc space nucleus is an inflammatory pain-producing lesion it must be adequately removed, otherwise it will continue to be painful and continue to extrude disc material. The hypertrophied synovial facet joints must be sufficiently resected to free the nerve roots from pressure in the lateral recesses and allow free excursion as they proceed on their intraspinal course to exit the spinal canal at the nerve root foramina.

**RESULTS**

The author of this paper has approximately 300 surgical cases he used as empirical evidence of the principles stated herein. Moreover, these cases were surgically treated in the specific fashion described in this paper. The results of these cases are being tabulated and formatted and will be soon be properly inserted into this space.

**DISCUSSION**

Degenerative Disc Disease of the adult lumber spine is not an inert, adynamic entity wholly represented by plain x-ray plain films or completely and straightforwardly interpreted from static, albeit highly technical and sophisticated, imaging paraphernalia such as MRI. Rather it is a complex, fascinating, and ever changing “disease process”. When understood much of this disease progression can be ascertained by astute clinical judgment in combination with relatively simple diagnostic tools. The perplexing and continued failure of medicine to collectively grasp and deal efficiently with the back pain imbroglio remains a baffling quandary. But certainly it is related to medicine’s general failure to embrace the inflammatory paradigm and function accordingly. In order to understand the overall situation and thereby appreciate one’s own back pain predicament it is helpful to break down the entire dilemma of back pain into its components and study the problems related to these components individually. Then one may disregard redundant and/or false information and spurious ideas and then reassemble his concepts into a solid grasp of back pain. Armed with this comprehension one may make an educated decision as regards his own problem and its most efficacious treatment.
The terminology used to describe DDD is varied, non-standard, and frankly, often erroneous. The improper use of this inappropriate terminology is replete throughout the medical community, the insurance industry, the lay press, and the public. Such use creates confusion amongst medical care providers, consternation amongst patients, and frustrating medico-legal difficulties. One often hears ambiguous and inexact terms to describe back pain syndromes such as “muscle sprain”, “low back sprain”, “disc syndrome”, “low back muscle pull”, “slipped disc”, “bulging disc”, etc. Also commonly heard are the even more non-specific “fibrositis”, “neuritis”, “rheumatism”, “fibromyalgia”, “dysmorphic pain syndrome”, the recently rediscovered “reflex sympathetic dystrophy”, etc. The list is endless. Other non-scientific and imprecise mechanical qualifiers with absolutely no medical definition, such as “significant…” and “non-significant degree of bulging”, leave one groping and often frozen trying to figure out what the problem exactly is and what is clinically important. Frustration and consternation is increased by radiologists, who seem to take refuge in these equivocal terms.

Words and phrases have meaning and impart cognizance and comprehension to conditions from which concepts are derived. Thus, the ubiquitous use of imprecise and glib terminology together with diverse and misapplied theories becomes a serious obstacle to the management of DDD and demands clarification. Thus, the term degenerative disc disease of the lumbar spine is a generic term, and as such, does not connote a particular pain syndrome, but rather refers to altered x-ray findings caused by chemical changes in the discs and facet joints whether they were induced by inflammatory or mechanical factors. These alterations consist of physiological and osseous changes in the disc, facet joints, and vertebral bodies, which lead to the loss of the disc’s structural and functional integrity and the narrowing of the spinal canal by bony hypertrophy. The clinical syndromes associated with DDD are Internal Disc Disruption [hereinafter IDD], Herniated Nucleus Pulposus [hereinafter HNP], Spinal Stenosis [hereinafter SS] and the more anatomically precise Lateral Recess Stenosis [hereinafter LRS] (also known as “intermittent cauda equina syndrome”) and degenerative spondylolisthesis. Each of these titles indicates a clinical syndrome, which is then associated with and correlated with a clinical syndrome. This is a very important point, because precise terminology and nomenclature impart improved understanding and better communication and treatment. This is vital as government, industry, and the insurance system, in their zeal for cost control, continue to analyze and deal with medicine from the “manufacturing systems” approach of Edward Deming.

Hence, it is evident that these syndromes are not disjointed entities, each deserving a completely divergent view and independent therapeutic approach. Rather, they are different clinical manifestations of symptomatic degenerative disc disease. So in order to comprehend DDD it is vital to envision degenerative disc disease as a process having a natural history with a beginning and endpoint. The terminology quandary in combination with nebulous and scientifically inadequate theories has led to practice patterns and modalities that are now based on nothing more than tradition and are often ineffective and costly. Herein, an attempt has been made to use terminology that is precise, consistent, and scientifically established.

The term “herniation” deserves further elucidation in order to keep it in proper perspective and enhance its utilization by the medical profession, the legal profession, the insurance industry, and the lay press, which by itself is a significant source of obfuscation. For example, a disc does not need to

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“rupture”, i.e., extrude its contents into the spinal canal or reach an arbitrary degree of “bulging” to qualify as a “herniation”. Pathologically speaking, a “herniation” is “the protrusion of an organ or part through connective tissue or through a wall of the cavity in which it is normally enclosed…” Therefore, even if a disc is only “bulging”, but at the same time inflamed, swollen, and painful, it qualifies as a “herniation”. In such a case the nucleus “protrudes” or bulges through the stretched and deformed fibers of the fibro-cartilaginous connective tissue of the annulus fibrosus and fulfills the criteria for herniation. 6

THE SOURCE OF LOW BACK PAIN

There remains no generally agreed upon theory explaining the source of back and leg pain in DDD, and therein lies the greater part of the controversy surrounding back pain. As discussed, historically most have dealt with back pain according to the “mechanical paradigm”, which in America continues to form the general framework for its understanding despite its lack of scientific veracity and contrary empirical evidence. 7 From the mechanical paradigm are derived the two basic components pain production, which are “compressive radiculopathy”, or sciatica due to disc herniation, and “instability”, or pain due to aberrant biomechanical motion. These two concepts have become such dogmatic axioms in the lexicon of DDD that to imply something else as the cause of pain is seen as irreverent by some and frankly blasphemous by others. So be it for that is exactly what is being done here.

These ideas are simple and straightforward, however they are simply inadequate to explain discogenic back pain and its associated radiculopathy. 8 The mechanical paradigm dictates that the only source of sciatica or “radiculopathy” is compression of the nerve by disc herniation. Moreover, the mechanical paradigm directly spawns the vague and insidious concept of “instability”, which connotes some sort of aberrant biomechanical motion as the source of back pain and the HNP syndrome. These ideas are uncomplicated and straightforward, but unfortunately, they are simply inadequate to explain discogenic pain and its associated radiculopathy.

The concept of instability is particularly insidious for it rationalizes fusion procedures, but astoundingly, there exists nowhere in the medical literature a scientific and consistent description or definition of instability and its relationship to back pain, which tells the practitioner how to define it, recognize it, interpret it, and correlate it with an anatomical source on imaging studies or clinical examination. Nevertheless, in spite of this lack of scientific and/or empirical proof that spine fusions have any good effect for back pain, thousands of spine fusions are done in the US each year.

Physiological loads are those forces exerted during normal activities of daily living. Physiological forces on a normal nerve produce numbness and tingling. But physiological forces against an inflamed nerve produce pain. The medical literature today demonstrates a very noteworthy and voluminous increase in scientific information, data, and evidence that inflammation and its chemical mediators such as prostaglandins found in large concentrations in a deranged discs are necessarily involved in producing pain in DDD.

Ironically these ideas are not new as years ago others suggested inflammation as the source of pain in DDD. Charnley first noted and described the “swollen” or fluid ingested state of the painful disc, implying inflammation as a source of these changes and the degenerative process. Seen by Charnley this swelling is a precipitating component of the series of physiological events leading ultimately to disc

herniation. Interestingly Charnley further postulated that the provocative discogenic pain mechanism may be “internal” spinal pressure spine acting on an inflamed disc, rather than “external”, i.e., mechanical, forces acting on a degenerative disc. Years ago Hirsch identified granulation tissue in the disc and suggested it as a possible source of pain and/or healing in the degenerate disc, thereby further implicating inflammation as an essential component of degenerative disc disease. Wiberg suggested that the vascular changes in the adjacent vertebral body are more likely the cause of spinal back pain than the disc itself. The lack of nerves in the disc itself and the plethora of nerves in the bone gives a great deal of credibility to this theory. Moreover, Arnoldi demonstrated an increase in bone pressure in the vertebral bodies adjacent to a degenerate disc; a concept that repeatedly continues to surface as a probable cause for back pain. Hungerford has also recognized the increase in internal bone pressure as a component of degenerative arthritis in peripheral joints, a patho-physiological phenomenon in no way different from degenerative disc disease of the spine. It is postulated that “microfractures” of subchondral bone along with their associated pressure effects can have an initiating and/or propagating effect in the development of osteoarthritis and pain.

There are some interesting clinical parallels. It is recognized that the pain relieving effects of the “high tibial”, or Coventry, osteotomy is likely due to the pressure relieving effect of the bony cut rather than any type of “spurious mechanical concept”. Similarly core decompression of the neck of the femur in a patient with avascular necrosis has been noted to have a pain relieving effect. It is theorized that the beneficial effects of these procedures is probably due to the opening and simultaneous decompression of the “venous hypertension” of the of the vascular sinusoids in the medullary bone cavities. Moreover, it is postulated that “microfractures” of subchondral bone along with their associated pressure effects have an initiating and/or propagating effect in the development of osteoarthritis and pain. Thus, we are left with the inevitable realization that inflammation and its effects appear to be a fundamentally necessary patho-physiological component of spinal back pain and sciatica.

Scientific data and empirical evidence, which contradict the mechanical paradigm and support inflammation as the source of discogenic back pain include the following:

1. A large number of asymptomatic individuals have been shown to have disc herniations, protrusions, or otherwise abnormal radiological studies; and the reverse is also true;
2. Post-surgical mechanical (pictorial) representations of the size of the residual disc herniation do not correlate precisely with post surgical pain relief or physical signs;
3. The treatment of DDD syndromes with epidural corticosteroid injections has been quite successful despite the continued presence of disc herniations;
4. Discography demonstrates the internal disc morphology (anatomy) as opposed to the external morphology shown by myelography and MRI’s and can simultaneously reproduce the patient’s clinical pain oftentimes in spite of their being no herniation; this contradicts the

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concept of external disc herniation and its presumed compressive effects as the only possible pain producing mechanism;\(^\text{(16)}\)

(5) moreover, discography often reproduces pain in a pathological disc underneath a solid fusion, contradicting the notion that a solid fusion stops motion and so stops pain;

(6) pain in a sitting or supine position is obviously indicative of a cause other than aberrant motion mechanics;

(7) age related increase in x-ray findings of disc degeneration, which does not correlate with the incidence of pain.

(8) Size of the disc herniation is not necessarily correlated with the degree of pain that a patient has indicating that there must be other factors involved.

### THE NATURAL HISTORY

The Natural history of a disease is the sequence of events that occur between the beginning and the end of the disease process in the usual and routine case without intervention by man and his therapies. It is necessary to understand the natural history of a disease in order to manage it more rationally and intervene more effectively. Back pain and DDD, conceptualized in this fashion, allows the treating physician to visualize it as a “process” and apply predetermined “pathway” mechanics that are in current vogue among third party payers and the Health Care Finance Administration (HCFA).\(^\text{(17)}\) As discussed above DDD syndromes, as they are encountered on a clinical basis, are not truly separate and diverse entities, but rather they are different points on a natural history time line; i.e., a disease pathway. When this systems approach approach is applied to DDD as it is being done with other medical syndromes and diagnoses, the “manufacturing process” criteria of statistical exactitude and quality improvement driven by scientific principles and reproducible data can be utilized enabling outcome data to be more readily obtainable.\(^\text{(18)}\)

The relationship of DDD syndromes along a patho-chronological time line has been well described with incredible erudition by Kirdaldy-Willis and, again, as has been pointed out above, further illustrates their common etiology. With this concept of commonality in place one derives a more clear notion of reasonable treatment at appropriate intervention points. Fundamental to this mode of thinking is the perception of Crock’s concept of disc degeneration and his use of the term Internal Disc Degeneration [hereinafter IDD]. Understanding the IDD is fundamental, because it provides the necessary insight into the disc as the initial source of pathological changes and pain due to disc degeneration. Additionally, from a more general perspective, the term IDD connotes all the pathological processes and pain producing syndromes, which are clinically evident and emanate from and/are contained within the disc prior to herniation; i.e., without clinical radiculopathy. The IDD, then, is the HNP without sciatica.

The IDD and/or the HNP, depending on the initial presentation, are the clinical beginning of Kirkaldy-Willis’ time line. At the same time these two phenomena are anterior column entities. As the patient ages and the time line progresses, the patho-physiological changes initiated in the disc simultaneously stimulate secondary changes inflammatory and degenerative changes within the associated facet joints and respective neural arch (i.e., lamina, pars, ligamentum flavum). Thus, the pathological process of DDD soon encompasses the entire “three joint complex”. Hence, the pathological processes and clinical manifestations chronologically proceed from the anterior column

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18 Id.
disc to the posterior column facet joints. These changes include sclerosis, hypertrophy, osteophyte formation, and nerve root foraminal encroachment inevitably resulting in Spinal Stenosis (SS) or Lateral Recess Stenosis (LRS) as the nerves become entrapped. The patient’s symptoms and clinical syndrome are reflective of this progression transition. Ultimately, if the process continues to its final conclusion the discs and the facet joints undergo spontaneous ankylosis, and the patient’s clinical problem changes from predominately back pain to predominately leg pain.

THE CLINICAL PERSPECTIVE AND THE ITS
“PAIN” DILEMMA

A patient with DDD presents to the physician with pain. The problem for the patient is pain and the dilemma for the physician is to decide how he must deal with it. Therefore, the standard criterion against which the clinical success of any treatment protocol is pain relief. In analyzing pain it must be quantified in order to determine the type of modalities the physician should employ. Generally the quality is assessed according to the type of clinical syndrome the patient has. However, after one understands the natural history and the commonality of the clinical syndromes, it becomes less necessary to try and separate them and more important to decide the appropriate treatment according to the severity of the pain pattern. However, it is true that the younger patient is more likely to have the IDD/HNP syndrome and the older patient the SS syndrome. The youngest patient in this series was 17; the oldest 82. Satisfactory results were obtained in 74% of the HNP category; 80% in the combined HNP/LRS category; and 81% in the LRS category. This data can be helpful, because, for example, epidural steroid injections are generally more helpful in the older patient with spinal stenosis. Fortuitously, for the astute physician who understands biomechanical factors, the clinical syndrome can be easily confirmed by a meticulous history and physical examination without the need for expensive diagnostic examinations. 19

When the physician appreciates that the IDD is the basic pathological substrate, he can then understand how the majority of patient who develop the HNP and sciatica give no history of a preceding traumatic event for pathological changes can occur in the disc long before clinical symptoms become evident. 20 As anterior column pathology the IDD/HNP is recognized by pain with forward bending, lifting, prolonged sitting, and variably with lying down and other static situations. In contrast, LRS has a reversed symptom complex: back pain and leg pain are increased with walking, standing, and backward extension and sleep is often disturbed; while sitting, lifting and forward flexion gradually become less painful and even provide relief. Furthermore, SS/LRS is generally a clinical diagnosis based on symptoms and signs and often these patients have no demonstrable neuromuscular deficit at all. For example, aggravation of pain in an elderly individual is often ignored or misinterpreted for it is often produced by venous engorgement of the bony vertebral body sinusoids without obvious clinical signs. 21, 22

It is necessary to understand spinal biomechanics in order to diagnose the patient’s syndrome. This helps to correlate the clinical problem with the imaging study rather than using the imaging study in a frustrating attempt to obtain a diagnosis. Typically the physician is met by a patient who has an apparently inconsistent, bizarre, or mixed pattern of symptoms and signs with sciatica and no imaging correlation; 23 or appears to have multiple nerve roots involved from a distant area. Accounting for these

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observations are the following dynamics: the inflammatory process may exist over more than one local area and all neurological structures in this milieu can manifest clinical symptoms of radiculopathy; any one disc and its respective motion segment can be at one place on the natural history time line, while a second, and even a third disc, at other places; since at any one time most patients that the physician sees are middle aged and so at some point in the middle of the natural history time line, DDD will likely involve both the anterior and posterior columns at the same time; LRS most commonly occurs unevenly so that its symptom severity is usually asymmetrical. Incidentally, an isolated “facet syndrome” invalidated as an isolated entity by some, did not occur without an associated motion segment IDD or HNP as indicated by discography, and so as far as this practitioner also is concerned, does not occur as an isolated entity. Also, most patients have neuromuscular evidence on the physical exam of bilateral involvement, which will be found if it is looked for (table).

Hence the physician is rarely ever presented with a neat clinical problem, but so long as he is cognizant of the above factors, DDD syndromes can be recognized, categorized, and treated without expensive, excessive, and often confusing tests. For example, it is helpful for the physician to be aware that only 1/6 of those patients who are found at surgery to have nerve entrapment in the lateral recess have a pure disc herniation (i.e., HNP); i.e., 5/6 of those with radiculopathy have at least a minimal element of LRS. Expensive and/or invasive imaging studies are not routinely necessary to determine the clinical syndrome. In this series the patients, assisted only with plain x-rays, were divided into clinical syndromes based on their clinical history and physical examination (table), which were HNP, the combination HNP/LRS, and the relatively pure LRS. Testifying to the reasonableness of this concept is the fact that the surgical outcomes of these individual clinical groupings generally parallel the overall results.

The above leads the astute observer to conclude the following fundamental factors: disc herniation and other spinal DDD syndromes are asymmetric, multiple level, clinically evident states of the degenerative disc process rather than solitary pathological events isolated in time and brought on by a mere mechanical event. Consequently imaging studies should not be routinely seen as indicative of static events, but rather they represent pathological points along the patho-chronological time line. When the imaging studies are used to define the disease process and establish clinical syndromes, then the imaging study serves to define the disease, and one is led afield seeking individual, varied, and often inappropriate treatment protocols. Moreover since DDD is an inflammatory condition it is associated with dessication, chemical alterations in the glucosaminoglycan substrate, inflammatory cell infiltrate, and invasion by granulation tissue. Over time this leads to spontaneous auto-fusion and/or fibro-osseous ankylosis of the disc space indicated on the plain x-ray by progressive disc space narrowing, subchondral sclerosis, and osteophyte formation. Mechanical factors are superimposed upon the inflamed anatomic structures and serve to aggravate the clinical symptomatology and make it clinically evident. Hence, since radiculopathy has a fundamentally inflammatory basis, it can often be relieved by epidural steroid injections as seen in this series.

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It must be understood that the ankylosis (narrowing and fibrosis) of the disc space on plain x-ray represents healing of the disc space.\textsuperscript{31} Ironically, these are the exact opposite conditions of swelling and early inflammation, which would predispose to “acute disc herniation”.\textsuperscript{32} Thus, DDD as seen on plain x-rays is not necessarily associated with a painful state, for in reality, it represents just the opposite; i.e., a pain relieving or healing phase of disc degeneration.\textsuperscript{33} It now becomes readily understandable why there is no statistical correlation between pain and x-ray evidence of DDD.

**THE SURGICAL PROBLEM**

**I. The Surgical Dilemma**

Since there is neither a consensus as to the cause of low back pain nor a commonly agreed upon definition and meaning of the term “instability”, it is obvious there can be no agreement regarding the ideal surgical procedure. And there isn’t. This bewildering muddle is due to the mechanical paradigm that ascribes DDD and its clinical symptoms to biomechanical aberrations rather than inflammatory changes. This fundamental flaw in current thinking inevitably leads to surgical failures. Implicit in surgical failure is the neglecting of surgery to “focus on the chemical aspect of back discomfort”.\textsuperscript{34} Fundamental logic tells one that there will be failures if the surgical procedure is designed to correct the wrong problem. Moreover, the factors associated with surgical failure and repeat surgery are well known and established and are corroborated with the observations in this series.\textsuperscript{35} In summary these factors are: the mechanical paradigm as the foundation for the surgical procedure and its consequential derivative, spine fusion; partial (read: micro-) discectomy; failure to deal with the total number of pathological discs; and the misinterpretation of imaging studies.

In order to understand the proper surgical approach is it necessary to understand and be cognizant of the reasons for surgical failure. When analyzed, these factors individually further illustrate the reasons for low back pain as already discussed.\textsuperscript{36, 37, 38} As the first step in this comprehension it is necessary to fully grasp the patho-anatomy of lateral recess stenosis. The term LRS does not just signify “bony impingement” deep to the superior facets in the lateral recesses, but has a collective connotation, which includes disc herniation, facet joint hypertrophy, and the entrapment of the nerve root in scar. Charles Burton, MD states, “…neural compression within the intervertebral foramen is rarely the result of any one single phenomenon, but usually results from the interplay of a spectrum of pathologic changes. These (hyperplastic) changes are usually secondary to progressive degeneration of the three-joint complex”.\textsuperscript{39} It is difficult and probably not helpful to classify any one factor as being any more common or important in continued post-surgical pain, because all of them are usually found together and whenever they are encountered, must be dealt with compositely to ensure inevitable surgical success. Furthermore, facet joint hypertrophy and entrapment of the nerve roots in scar can be a fusion induced form of LRS and collectively are termed iatrogenic spinal stenosis.\textsuperscript{40}

\begin{thebibliography}{9}
\textsuperscript{33} Osti OL, Vernon-Roberts B, Fraser RD. Annulus tears and intervertebral disc degeneration. Spine 1994;15/8:763-7.
\textsuperscript{35} White and Panjabi, Biomechanics, p. 159.
\textsuperscript{39} Supra; at 33.
\textsuperscript{40} Verbiest
\end{thebibliography}
Partial discectomy (e.g., micro-discectomy) is fundamentally inadequate to “deal with (all) the pathology”. 41 Retained disc material and/or an annular disc opening made or enlarged at surgery without complete disc removal inadvertently serve as iatrogenically enhanced egresses for inflammatory disc exudates and subsequent nerve root scarring. 42, 43 The disc remnant serves as a source of continued source of spondylogenic (spinal) pain ready to “herniate” again another day. 44 In this series, without exception, surgical re-exploration after micro-discectomy failure always demonstrated retained disc material, facet joint hypertrophy (LRS), and nerve root scarring. Revision surgery consisting of extensive and complete nuclear disc excision, meticulous neurolysis, and adequate bony laminotomy and medial facetectomy were important factors in the successful surgical revision of these unfortunate people. In spite of these genuine factors and legitimate observations we see today an endless increase in the number and technical variations of micro- and partial/limited discectomies, sometimes even combined with endoscopically aided fusions. 45

The failure to deal with the total number of pathological disc levels is also a significant cause of surgical failure (table). Of the primary cases in this series 66 involved surgery on more than one level with 75% of the 2 level and 78% of the 3 level cases showing satisfactory results. Similarly the revision cases showed similarly satisfactory results for both 2 level (78%) and 3 level revisions (100%). (see table). These results strongly imply that unrecognized pathology at disc levels not previously operated on in the primary procedure can be a significant source of post-surgical pain. Discography was indispensable in analyzing these pathological and painful levels, which indicates that modern spine surgery cannot be practiced without it. 46

Ultimately spine fusion is the most devastating and dreadful cause of surgical failure for it and its routinely used hardware cause innumerable complications and morbid results. Patients are consistently left with stiffness, worsened pain, neurological injuries and routinely can’t work or even perform normal activities of daily living as well after this surgery as before. Fusion has long been employed to treat painful peripheral joint conditions. From this experience together with the facile mechanical paradigm and its co-derivative theory of spinal “instability” orthopaedic surgeons have conjured the seemingly logical concept of spine fusion. 47 Unfortunately for the concept of spine fusion and those that receive this highly morbid procedure there is no scientific evidence at all that this procedure has any positive effect whatsoever on the relief of back pain due to DDD. 48 Instability is a particularly insidious idea for it has stimulated an untold number and variety of arthrodesis procedures designed to correct this undefined “condition”. There remains today no universal definition of “instability”, no ability to correlate any construed instability concept with back pain, and no mechanism to show a relationship with any surgical treatment of instability with relief of back pain. It is an entirely specious concept as it relates to the treatment of back pain due to DDD. Amazingly most evidence indicates that it has a

deleterious effect on back pain yet it continues to be used at astounding rates! Moreover, “motion studies”, when done to substantiate “instability” provide inconclusive and confusing results and one is left groping trying to figure out if the patient truly has more pain with increased or decreased motion. Such studies were not done in this series, because aberrant motion studies of the spine are incapable of demonstrating painful discs. Painful discs can only be demonstrated by discography. There is no test to demonstrate instability.

Rolander has demonstrated that the motion inhibiting theory behind posterior spinal fusion is groundless for even when a solid posterior fusion is obtained inhibition of motion is not complete. This is because the axis of motion is in front of the posterior arch where the posterior arthrodesis is performed and the anterior moment arm is just too great to inhibit motion even when a posterior arthrodesis is obtained. Moreover, in any event only an anterior interbody lumbar fusion is a load bearing construct and can stop motion in the sagittal plane. Finally, posterior fusion does nothing to address a pathological and inflamed concept. Spinal fusion based on such spurious concepts should be condemned.

“Pseudoarthroses” (“false joints”; i.e., failure to fuse) were not found to be the cause of the “failed back syndrome”. Theoretically, according to the mechanical paradigm, the presence of a pseudoarthrosis would explain the continued presence of back pain after a failed spine fusion. This thinking inevitably leads to one spine fusion after another. It is not surprising that in the revision cases in this series pseudoarthroses were not found to be the cause of back pain, because there are as many reports claiming the resolution of back pain with the presence of pseudoarthroses as there are those in the presence of solid fusion where pseudoarthroses are not found. This fact, of course, again belies the concept of instability as a producer of back pain. Quite likely the fortuitous outcomes in the presence of solid fusion described in the literature were as a result of associated procedures serendipitously done at the same time as the spine fusion rather than the spine fusion itself.

Herein, revision procedures were assessed in the same fashion and according to the same criteria as primary procedures. The theory utilized was that if the procedure was not successful the first time, then it made little sense to consider a different procedure. Making much more sense was to do the procedure correctly the second time—so long as the procedure made sense. Discography was an extremely important source of diagnostic data. As a consequence, the overall results of the revision procedures were as good as the primary procedures. (table). There was no deterioration in results with the total number of previous surgical procedures as is the usual supposition. In fact, there was often dramatic improvement. There were several patients having had 3 procedures and ( ) as many as 5 and 7 previous procedures. These satisfactory results are unusual in view of the generally pessimistic view of “revision” lumbar spine surgery, but nevertheless point out that the causes of continued pain in the face of surgical failure are the same as the original causes of pain to begin with. When the “disease” was “not recognized” the first time, there is no reason not to recognize it the second time and, instead, to embark on a subsequent surgical misadventure; e.g., a spinal fusion.

Psychiatric/compensation cases in this series had similarly sub-optimal results both in the primary and revision cases. The male/female categories within the revision group had almost the same statistical results as the overall collective results (tables I and II). Analysis of tables I and II indicates a satisfactory result in 86% of the primary male group and 89% of the revision group—an actual

50 White and Panjabi. Biomechanics of the lumbar spine.
improvement in this generally thought to be hopeless group! Inspection of the similar female totals demonstrates satisfactory results in 70% and 69% of the primary and revision groups respectively. The other data are quite comparable. These observations lend credibility to the methodology used herein for the results are consistent with generally accepted results. At the same time, the results point out that women did more poorly, because they tend more commonly to be the victims of depressive illness and overmedication. Perhaps medicine should reassess its viewpoint and techniques in dealing with this group of patients.

Hence, satisfactory outcomes in these revision cases underscore the premise that the syndromes causing back pain due to DDD have a common pathogenesis and so logically necessitate a common viewpoint and a consistent approach. Therein, the possibilities of beneficial clinical and surgical results are maximized.

**II. The Surgical Goal**

Although it would appear obvious that the surgical procedure should be designed to alleviate the patient’s pain this is not true as regards disc surgery. In the case of DDD, as regards spine fusion, the stated goal of the practitioner is to create a “solid” spine fusion. Hence, curiously in the context of spinal fusion, practitioners generally consider success to be the attainment of a technical goal; i.e., a successful spine fusion, and not the relief of the patient’s pain. This practitioner considers such thinking to be, at best, unusual. Physicians are charged with both doing no evil and curing the patient’s pain. A spine fusion is a technical surgical exercise, which accomplishes neither stated goal. This practitioner considers such thinking irresponsible. The idea that the expressed goal of surgery that would signal success is a solid arthrodesis regardless of the patient’s pain relief, is considered by this practitioner to be bizarre. That a practitioner would subject a patient to such a highly morbid technical exercise, curiously without any proven efficacy and with well-known complications familiar to all, is considered by this practitioner to be a peculiar twist of medical science and ethical values. Ideally, since physicians are charged with the relief of pain and not the successful performance of surgical procedures the goal of the surgical cases presented herein was the relief of the patient’s pain. Ideally the procedure should be designed to deal with the inflammatory condition, which is the fundamental source of back pain itself, and the commonality of all DDD syndromes. Then the surgical approach should deal collectively, concurrently, and completely with all the components of the deranged pathological motion segment (remember: the disc and its two associated facet joints) and definitively with the pain emanating therefrom. Intuitively, the surgical technique should recognize the potential sources of surgical failure as described above and be designed with inhibiting such outcomes. Specifically, the goals of surgery should always be kept in mind and can be summarized as follows:

a) relieve the patient’s pain
b) leave the patient with a functional spine.

(Note: the above enumerated goals do not include a solid fusion.)

Equipped with the above knowledge and data a surgical procedure was designed and utilized on every disc level operated on; in primary and revision cases; regardless of the cause of the previous surgical failure. This makes sense, because the reasons for failure are always the same and will be reiterated again: (1) failure to remove completely the offensive disc; (2) failure to deal completely with lateral recess stenosis; (2); and iatrogenic (physician induced) spinal stenosis due to inappropriate

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spinal fusion. So contrived and performed, the surgical procedure does not act to supplant and counteract the natural history of DDD but rather acts in concert with it. Simultaneously the procedure induces the continued natural healing process, which DDD is; provokes the continuation of spontaneous (i.e., natural) interbody ankylosis (fusion), which is the final healed end point of disc degeneration (ref). Therefore, the surgical stimulation of spondyloarthrosis, which in this series was associated with pain relief, was viewed as a desirable outcome of surgery (figure). This opinion is in sharp contradistinction to those who have viewed the development of post-surgical spondyloarthrosis as less than an optimal result (figure, x-rays). Notwithstanding the latter viewpoint, it is inexplicable to this observer, that such a process when seen in its natural context as a pain relieving mechanism and evidence of disc healing, should be viewed by some in a less favorable light when seen post-surgically.

III. The Surgical Procedure

Since the inflammatory paradigm allows the disc to be envisioned as a “sterile abscess”, i.e., the progenitor of pain and the central component of the deranged motion segment, then it naturally follows that the ideal surgical procedure to accomplish pain relief should be to attack the inflamed disc nucleus. In this series total nuclear extirpation and vigorous curettement of the cartilaginous end plates covering the vertebral bodies was the procedure performed in all of the cases in this series. This procedure concurrently decompresses the pathological disc space and the hypertensive and painful venous engorgement of the vertebral sinusoids. Thus simultaneously all the painful mediators emanating from the disc and the vertebral bone are treated. Additionally, as has been seen time and again by this practitioner, as the procedure eliminates the inflammatory environment it also eliminates radicular or sciatic pain.

The components of the surgical procedure used in this series consisted of a bilateral stripping of the paraspinous muscles, a laminotomy, or the approach component of the procedure, and a partial medial facetectomy. The laminotomy is the “approach” component to the disc and nerve and is done with osteotomes. It consists of a partial excision of the distal portion of the proximal lamina and the proximal portion of the distal lamina. The partial medial facetectomy is also done with osteotomes and along with the laminotomy opens the lateral recess and gives excellent exposure to the lateral side of the nerve deep in the lateral recess along with its associated disc. Microscopes are not necessary and serve only to deleteriously narrow the field of vision. The soft tissues are stripped from the medial aspect of the enlarged facet joints with small angled curettes, and the ligamentum flavum, spinous processes and interspinous ligaments are left intact as are left intact. Since the pathology is usually found lateral in the deep to the facet joints in the lateral recesses there is no reason to operate in the midline and take a chance on injuring the dura and its contained nerve elements. Once the nerve is retracted toward the midline and bleeding controlled a small rectangular opening is made in the disc annulus, through which the degenerate disc, under pressure, proceeds to bulge forth. Next the pain relieving component of the procedure is carried out: a bilateral complete nuclear discectomy. The disc nucleus and cartilage endplates are removed with pituitary rongeurs after scraping and freeing the disc material with an angled disc curette. The curette is rotated in the disc space creating some morsellized cancellous bone, which simultaneously opens and decompresses the vertebral bone. This renders a confluence of the blood supply between the adjacent vertebral bodies by creating a hematoma and capillary in-growth. The net result is the elimination of the producer of discogenic pain by complete extirpation of the disc nucleus.

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Generally, fibro-osseous, or even osseous, ankylosis of the vertebral bodies occurs secondarily. Since solid fusion is inconsequential for pain relief, and bone graft harvesting and insertion are painful and dangerous procedures, there is no reason to subject a patient to the complications of bone grafting techniques. Moreover, solid fusion is even less desirable than fibrous ankylosis for fibrous ankylosis may leave the disc space with a minor degree of desirable motion. Therefore, no hardware or remote bone graft was utilized in the procedures presented in this series. Formerly, local bone graft was placed in the disc space to serve as a stimulus for bone formation. This is no longer done, and such bone grafting has been gradually ceased completely, since it has been determined that it is unnecessary for pain relief.

Purposely the mid-line structures, which include the spinous processes, the interspinous ligaments, the supraspinous ligament, and the ligamentum flavum intact, are left alone and intact for they as a rule are not involved in the production of clinical symptoms. Moreover this helps to decrease the chance of a post-surgical spondylolisthesis. This makes sense for in the vast majority of instances the extradural L4, L5, and S1 nerve roots are involved, not the intradural midline S2, S3, and S4 nerve roots, which innervate the bladder, genitalia, and rectum. Furthermore, it makes better sense not to operate in this area, because it is the area most commonly involved in dural scarring especially in revision cases. Hence, surgery in the mid-line has the greatest propensity to create dural tears and their potentially disastrous complications. The procedure is done exactingly, and purposely the ligamentum flavum is left in place as a protective mechanism from intra-operative dural injury and as a physiological barrier from post-surgical scarring.

Generally, the meticulous physical examination done prior to surgery demonstrated the presence of bilateral weakness and/or sensory changes in about 70% of patients. Moreover, the literature states that many patients post-surgically develop subsequent pain and evidence of radiculopathy on the opposite side. For these reasons, a bilateral procedure is usually performed.

A midline incision is made about 8-10 cm. long. No attempt is made to perform a “microdisectomy” and make an unreasonably short incision, because the procedure requires a “macroscopic” (i.e., easily visualized with the naked eye) technique. The overall dimensions of the patho-anatomic field must be seen. The soft tissue and paraspinous muscles are bilaterally stripped out to the facet joints exposing the pathological disc space and adjacent disc space on either end. A self-retaining retractor is placed to allow continuous visualization of the surgical field and free the assistant so he can be of help controlling bleeding and retracting the nerve root once it is exposed. The nerve roots are easily visualized with the naked eye. Moreover, in order to visualize the disc anatomy, perceive the enlarged facet joint sometimes encroaching almost to the midline, and determine the specific area of nerve entrapment within the lateral recess (an area at least 2.5-3 cm long), a gross visualization of these structures with the eye is required. Although allowing for the enhanced visualization of small bleeders and details of the nerve roots themselves, a microscopic perception is generally unnecessary, counterproductive, and itself accounts for many surgical failures and intra-operative complications, because the anatomy can’t fully be seen at one time. The disc space is also a macroscopic structure and the instruments used to clean it of disc material are macroscopic in dimension. A microscope can be dangerous, because it narrows and confines the visual field and prevents its overall perception in its entirety. It is functionally the equivalent of peering through the tiny end of an inverted cone. Thus, areas of disc herniation and lateral recess nerve impingement by disc fragments deep in the lateral recesses and far laterally are often not seen with the confined visual field of a microscope. Without a microscope the entire dimensions of the pathological field can be seen. When the surgeon is equipped with experience and technical expertise bleeding can generally be controlled with cottonoid patties placed proximally in the axilla (angle) of the nerve root above and distally on the shoulder of the nerve root below and judicious use of bipolar electro-cautery. Moreover, this practitioner feels it is absolutely
unreasonable to perform a micro-discectomy in the face of a previous surgical failure. In such a case it vital to see the entire intra-spinal area that the nerve root traverses after its exit from the dura and before its exit from the spine through the respective foramen.

The inter-laminar area is visualized and cleaned of all soft tissue except the ligamentum flavum. A long angled curetted with an adequately small cup is used as a tissue elevator to strip soft tissue from the bone. The angled cup is inserted deep to the superior lamina between it and the underlying ligamentum flavum, it is swept in a lateral and distal direction on the medial aspect of the enlarged facet joint stripping the attached soft tissue and ligamentum flavum from the bone, while taking care to maintain the heel of the curette facing in a medial direction to protect the vulnerable midline dura. The distal lamina and approximately the medial ½ of the facet joint are osteotomized with an bone osteotome and removed back to the level of scoring with a Kerrison rongeur, again taking care to always maintain the heel of the instrument facing in a medial direction and the biting portion facing in a lateral (i.e., safe) direction. Thus, during this delicate procedure there is no chance that the dura might be injured. All this time the midline structures and ligamentum flavum are left intact and the nerve root lying in the lateral recess can be easily seen. Hemostasis is maintained with cottonoid patties proximally and distally and bipolar electrocautery as is necessary.

Much of the rest of the procedure is done by 3 dimensional knowledge of the anatomic field and general perception of the pathology, which is required vital knowledge before the surgeon becomes entangled in these procedures. A nerve hook is used to identify the bony pedicle of the distal vertebral body, knowing that the respective nerve is lying immediately medial to the pedicle and the disc space is just proximal to it. The nerve hook is used to sweep between the underlying disc space and the overlying nerve, thereby freeing all adhesions commonly found between these structures especially in revision cases. The nerve root is then retracted to the midline with the help of the assistant and the entire lateral surface of the disc is seen. Hopefully, the field is now reasonably bloodless. A number 11 surgical blade is inserted into the disc and a small rectangular window is made. Depending on the pathological stage of the disc it will often bulge forth from the disc space and any immediately extruded fragments can be grasped with a pituitary rongeur. A specially made angled disc curette can then be inserted into the disc space and the friable disc material can be loosened and removed by the pituitary rongeur. The angled disc curette can then be reintroduced and the cartilage endplates on either surface of the disc space on the adjacent vertebral bodies can be loosened. All this loose material can then be removed by reinsertion of pituitary rongeurs. The disc nucleus should be extirpated entirely. Of course the annulus is left intact. The angled curette can be rotated in the disc space breaking into the surface of the vertebral bone. This has the desirable effect of opening, draining and decompressing the hypertensive vascular sinusoids of the vertebral bodies. Post-operatively the patient should awake in the recovery room with the sciatic pain completely gone and his strength and sensation totally restored. His back pain should be converted to merely post-surgical incisional pain. Generally, he should be up that evening and out of the hospital the next in one or two days.

In summary, the entire procedure has the effect of removing all of the offensive disc nucleus, decompressing the vertebral bodies, and draining the inflamed disc, and thus eliminates the disc as an inflammatory focus. If the results are not as described the procedure will not be successful.

**AUTONOMIC NERVOUS SYSTEM DYSFUNCTION**

**I. Genitourniary Symptoms**

In this series the incidence of urinary tract symptoms was sought as surgical back patients often pre-surgically complained of frequency, dysuria, and urgency. These symptoms are generally much
more common than is thought and in this series an incredibly high percentage of patients, (see table) had improvement in these symptoms post-op. Routinely these patients prior to surgery never had their symptoms properly diagnosed as caused by low back dysfunction and had sought treatment elsewhere for their symptoms, some even having undergone surgery on various other organ systems with generally unsuccessful results.\textsuperscript{60} Such observations cannot be taken lightly or easily discounted when one considers the inflammatory nature of radiculopathy and the altered chemical environment in which the entire cauda equina lives. \textsuperscript{61} Others have alluded to this problem and/or observed similar outcomes.\textsuperscript{62}

In this series as the patient questioning became more specific regarding involvement of other organ systems other symptomatology such as constipation, diarrhea, or gynecological discomfort was noted to abate post-surgically. Some of these individuals had been told that they had prostate dysfunction, endometriosis, “irritable bowel syndrome”, etc. as a cause of their problems. No effort was made to categorize this myriad of symptoms as it was too multifarious, but it is obvious that within the context of autonomic nerve dysfunction of the lumbar spine secondary to degenerative disc disease, there is much opportunity for research on this topic. Moreover, therein may lie the claim of various practitioners who practice manipulation that complaints and/or diseases other than just back pain can be helped by manipulative techniques.

II. Reflex Sympathetic Dystrophy

In the light of present day techniques and the associated large number of surgical failures some elucidation of the disease state known as Reflex Sympathetic Dystrophy [hereinafter RSD] may be helpful. RSD is a term applied to a disease state marked with the following neurologically based implications:

a. **Symptoms**:

(1) **Pain**: inexorable, relentless, burning, knifelike, or stinging type of pain completely out of proportion to all physical signs and other clinical indicators; aggravation of the pain with motion leading to inevitable decrease and or complete elimination of motion at the afflicted joint or spinal segment; an inevitable spreading of the pain throughout the anatomical distribution of the nerve; the production of severe and excruciation pain and paresthesias with any attempted motion; production of these symptoms by even light stroking of the skin; tenderness throughout the distribution of the nerve; positive Tinel’s sign at the site of the impingement (in the case of involvement of a spinal nerve usually at a joint)

(2) **Swelling**: swelling leading to “brawny” edema often leading to mechanical limitation of motion; leading to periartricular (tissues around the joints) thickening and fibrous tendon adhesions, again leading to an inexorable cycle of decreased joint motion.

(3) **Stiffness**: early the stiffness is due to pain and edema, but then later it is due to pain + fibrosis, tendon adhesions and ineslasticity of periartricular joint structures. Inevitably there are joint contractures.

(4) **Discoloration**: early the disease is manifested by some type of increased discoloration such as redness or blueness giving way to pallor. This is due to abnormal vascular vasoconstrictive reflexes.


Sudomotor: hyperhidrosis or increased sweating is usually seen early, but later there may be dryness. Sometimes the sweating may be so great that it causes secondarily skin changes associated with increased sweating.

Temperature: With increased redness there is an increase in temperature; when the skin is pale there is a decrease in temperature.

Osteoporosis: there is demineralization far out of proportion to that seen in any other condition, and it occurs so fast and severely that it in and of itself is a very dramatic finding.

Trophic changes: the skin becomes thin, tight, and shiny and the underlying affected tissue becomes atrophic.

b. Diathesis: this term applies to personality changes that seem to occur secondary to the disease or are felt to be associated with a type of personality that seems to engender the disease. This seems to be a personality or psychological makeup that is described by psychiatrists as “fearful, suspicious, emotionally labile, inadequate personality, chronic complainers, dependent personality, insecure and unstable personality”. This at times seems to lead to situations on the part of practitioners that these patients are malingering.

c. History: The history of this disease is informative and illustrative. Of course most diseases have been present throughout history, but many such as RSD have been only recently discovered, i.e., recognized and described relatively in recent times. The history of RSD begins after the American Civil War. Because of the nature of this war, the type of injuries seen, the medical treatment offered to the wounded for the first time, and the survival of these wounded, this disease was first recognized and described. There were a large number of wounded in the Civil War who ended up with amputations and peripherally associated neurological injuries associated with these amputations. Also present at that time was the most highly developed and aggressive military medical delivery system seen up to that time together with a bureaucracy dedicated to systematically categorizing and treating these injuries. These factors caused RSD to be described for the first time. After the passing of the Civil War veterans RSD was not heard from much again until after World War II when it was again discussed with renewed interest, again due to a large number of traumatic injuries together with renewed scientific interest. Again curiosity with this entity in the medical literature subsided again until the late 1980’s and 1990’s.

During this period we see a renewed interest in RSD, but this time the stimulus was not due to the injuries of war. One can only speculate as to why this new interest, but it appears that it might be due to the large number of surgical failures that occurred with a dramatic increase in back surgery. It appears that the diagnosis has been given renewed interest, because it suggests a cause of symptomatology other than that of the surgeon’s technique. This seems to be for 2 reasons:

(1) The term “autonomic” connotes symptoms that are “automatic” or reflexive and outside the patient’s, and therefore, outside of the doctor’s control. In other words the symptoms are monitored through the spinal cord in a subconscious way without the patient or the doctor being unable to do anything about it. The use of the term RSD seems to imply that the treating doctor had nothing to do with the production of the patient’s symptoms.

Nevertheless, even if the symptoms are monitored via a subconscious reflex arc in the spinal cord, they can still be generated and propagated an irritation to the peripheral
nerve at any point along the length of the nerve. This includes irritation to the nerve in the lateral recess, which at that location is a peripheral nerve. Just because the reflex arc makes its connection in the spinal cord, the symptoms are propagated through the autonomic nervous system, and occur on a subconscious and involuntary level, does not mean that the treating doctor did not have something to do with their production. Iatrogenically by producing an injury to the respectively involved nerve somewhere along its length, i.e., in the lateral recess, the symptoms can be produced and manifest themselves clinically. They can also be corrected by properly done revision surgery.

(2) The term *diathesis* implies that the patient has a psychological propensity, for whatever reason, to develop RSD and the apparently abnormally heightened pain response. Whether this is true or not, invariably in the context of a less than optimal surgical result it is invariably the actions of the treating surgeon, who was responsible for the problem.

**IN SUMMARY**

It has become clear that the biomechanical principles derived from the mechanical paradigm and which heretofore have served as the intellectual substrate for the understanding and treatment of back pain, DDD, and its surgical implications are woefully inadequate to explain this disease and rationalize its treatment, surgical or otherwise. The solution for this costly and frustrating medical situation includes a general recognition and acceptance of the inflammatory process and its chemical mediators as the cause of low back pain, the establishment of clear and distinct non-surgical treatment protocols, the establishment of precise surgical indications, and the elimination of spine fusion as a technique to be utilized in the presence of back pain due to DDD. Spine fusion simply has no fundamental role in the primary treatment of discogenic back pain. Therefore, it is obvious that it would be for the greatest benefit medicine is pleased to serve, if each of the medical specialties acting in isolation and directed by its own particular vision and intellectual dimensions, would place its self-interest in abeyance and act in concert to find a clinical solution to the debilitating problem of back pain, while simultaneously offering consistent and minimally morbid surgical intervention.

In the series of surgical cases presented herein, this was specifically done. Needless spine fusion, unnecessary bone harvesting techniques, the insertion of hardware and their associated complications were avoided. The routinized surgical approach paralleled the natural history of the disease process and treated the patient’s pain by resection of a painful and pathological organ. Results comparable with the best claimed in medicine were obtained inexpensively, safely, and with minimal morbidity. Within this presentation are attestations of unsuccessful results as well as caveats that will hopefully help to prevent less than an optimal result.

Clearly, much of medical practice to this day is not governed by rational theory and scientifically proven outcomes research. Much of what we do is an effort to solve problems, while using available resources. Nevertheless, such empirical efforts should be performed methodically and driven by logic and available scientific data. Furthermore, they should be engendered in a historical context for ignorance of history and a lack of knowledge obtained in the past leads to a vicious cycle of similar results obtained using different methods from experiments similar to those of others. This leads to the same mistakes and ideas being perpetuated unchanged. Lastly, as physicians we must always remember what we are, what we are supposed to be, and be guided by ethical and intellectually honest principles. Upon that we will inevitably be judged.
It behooves one to remember the cryptic words of Josh Billings:

“It ain’t what a man don’t know that makes him a fool, but what he does know that ain’t so”.

Gerald A. Coniglio, MD