The Gonstead Disc Model
Advancing It into the 21st Century

for the Gonstead Clinical Studies Society
Meeting of the Minds III
Cleveland College of Chiropractic – Kansas City, Missouri
by Steven T. Tanaka, D.C.
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The Gonstead Disc Model is a product of the 1960s, but the model largely carries forward to today. Like all clinical sciences, it must continually incorporate current science. The technique requires it; the profession demands it; and society is best served by it.

Some features of the model are controversial and may have to be modified. Much of the model can be explained in greater detail with what science has been uncovering with the explosion in disc research since the 1960s to better explain features of the model which the science at the time did not have the tools to delineate. This on-going project for the Gonstead Clinical Studies Society is to bring the Gonstead Disc Model into the 21st Century.

This paper is a preliminary draft for parts of upcoming sections in the Updating the Gonstead Disc Model series, of which, three parts have been published in the GCSS *The 'G' Note*. Below, I am posing and discussing 6 questions or statements.

1. In the Gonstead Disc Model, the radiographically observed *parallel disc* is considered normal, and when present, the adjacent vertebrae are said to be in *optimal relationship*. Disc wedging as seen on the lateral film – re: the opposing vertebral body endplates diverge anteriorly and converge posteriorly – is considered to be abnormal.

This is probably the most controversial aspect of the Gonstead technique. Is a parallel disc always normal? Is a "wedged" disc always abnormal? In my opinion, a wedged disc is not always abnormal. Endplate bulging and other changes to the shape of the endplates can influence the shape of the disc. In the cervical and lumbar spine, at least, the nucleus pulposus is located slightly posterior to the center of the disc. How this affects the appearance of the disc as viewed laterally is not known. It is also known that the anular laminae are more numerous and thicker in the anterior than the posterior. In the cervical spine, there are few anular layers in the posterior aspect and its duty appears to be, to a major extent, substituted by the posterior longitudinal ligament and uncinate processes.

Other physiological influences that may affect the appearance of the disc on the lateral film. These include locations of the axes of instantaneous rotation and the location and size of the neutral zone.

An instance where a *parallel disc* is abnormal is in a D5 or D6 disc wherein the both the anterior and posterior regions of the disc have been resorbed to such an extend that the disc appears parallel but is very thin.

It is probable that a small amount of disc wedging wherein the opposing endplates diverge anteriorly as seen from the lateral view is normal, at least in the lumbar spine. It is probably less normal in the cervical and thoracic spine due to their structures.

2. Is disc wedging caused by the nucleus pulposus protruding into the anulus fibrosus and raising the region of the disc that it protrudes into?

As we now know, nuclear material tends to protrude and herniate posteriorward or posterolateralward. Fluid or semi-fluids favor areas of less pressure. When the inner anular layers adjacent to the nucleus is compromised, nuclear material, which is under considerable compressive force, will migrate to the weakened anular regions. The damaged region where the nuclear material protrudes into the anulus and the region of

the nucleus where the material originated would not be able to sustain the compressive forces. The forces will compress that region of the disc. The density of proteoglycans would be diminished in the region which will in turn, reduce the water content there. Much of the resistance to compression comes from the water content in the disc. By this time, there is fibrotic infiltration into the disc. Fibrotic tissue is not hydrophilic and cannot withstand compression, or for that matter, other forces on it. The remaining matrix structure opposite the protrusion may temporarily maintain some semblance of disc height where the nuclear material remains. Rather than raising the disc, the region where the nuclear material protrudes into the anulus is lowered, because there is less resistant to compressive forces due to the loss of the hydrophilic proteoglycans and other elements in the extracellular matrix.

3. A vertebra (C2 to sacrum) goes posterior in subluxation.

The orientation of the facet joints tends to cause the vertebral to move posteriorward and is usually coupled with movement in other secondary directions as well – as we know, structurally, the vertebra cannot translate anteriorly unless there is trauma or deformation of the structure. When the disc is compromised, the weakness would allow the vertebra to misalign. Because of the orientation of the facets, a component or vector of the misalignment would be posterior. In the Gonstead System, posteriority is considered to be the primary direction of misalignment. It would be difficult to have misalignment without a component of posteriority. This is readily apparent in the cervical and lumbar spine, less so in the thoracic spine. Even in the thoracic spine where the orientation of the facets tends to be coronal, the superior facet is the posterior element and changes in position would tend to cause posteriority, although the costal attachments complicate its movements and position.

4. In acute disc injury, the disc can swell up in what White and Panjabi call disc ingestion. We call this the D1 disc or acute, swollen disc.

According to White and Panjabi, *disc ingestion* (one author reportedly calls it “glaucoma of the disc”) can occur following trauma. The mechanism that causes it is not known, but the ingestion swells the disc. As we know, proteoglycans can attract a considerable amount of water, but it is finite. A different mechanism must be involved. The influx of fluids increases the intradiscal pressure which set the stage for internal derangement of the disc, particularly rupture of anular fibers. (Naylor 1976, White & Panjabi 1990) It also alters the biochemical balance in the nucleus that may lead to disc degeneration. (White & Panjabi 1990) It is thought that discogenic pain may be induced by stimulation of the nerve receptors on the periphery of the outer anulus of the swollen disc. (White & Panjabi 1990)

When the disc is acute and swollen following an injury, it would be logical to restore more ideal function to the disc. This would reduce the additional strain on the already injured structures. Restoring function to the motion unit by careful, highly specific spinal adjustments seems to be a rational choice, although there is no research to confirm this.

5. During disc degeneration, the disc begins to reduce in height as pathological or degenerative changes that cause complete internal disruption within the disc. In the Gonstead System, we have the radiographically visualized concept of D1 to D6 disc degeneration that describes, radiographically, this progression.

An entire book can be written on this statement. Kirkaldy-Willis divides up what occurs physiologically into 3 stages. The following is a common scenario. 1) Following injury to the disc, circumferential tears may form in the outer anulus fibrosus. Further injury can cause enlargement of the tear and further tears. These tend to occur in the posterior aspect of the disc. With further injury and time, the circumferential tears may unite and form radial tears. Radial tears progressively expand and may eventually breach the innermost anular layers and expose a path for nuclear material to protrude into. (Kirkaldy-Willis) Damaged

fibers are also being replaced with cross-linked fibrous tissue which is less elastic than the original tissue. 2) There is internal disruption wherein, over time, the anulus and nucleus become indistinguishable and as the disc is being resorbed adjacent vertebral body bone in the region of the endplates become dense and sclerotic which compromises nutrient transport. 3) In the final stage, the body attempts to stabilize the spinal unit with osteophytes, and occasionally, ankylosis. (Kirkaldy-Willis) We need to fill in the details, i.e., “put some flesh on the bones” as stage 2 is the phases of D2 to D6 disc. Stage 3 is the later stages of D6.

From a high of up to 90% in the new born, the water content is about 70% in the adult due to the loss of proteoglycans and an increase in fibrotic/collagen cells. (Oishi et al 2000) With both degeneration and aging, the water content can continue to diminish substantially.

Simultaneously with the anular disruption, changes may occur in the nucleus pulposus. In both degeneration and aging, type II collagen begins to be replaced by type I fibers in the inner anulus and nucleus at an early age. (Adam & Roughley 2006) This may, in part, be associated with apoptosis or programmed cell death. The hows and whys of apoptosis are largely unknown, although, it is necessary a specific stages in development and the maintenance of homeostasis. (Gruber & Hanley 1998)

The nutrient transport system may become disturbed. Within the first two years of life, there is a substantial reduction in the number of vascular channels in the osseous vertebral endplates. With time and injuries, microfracturing, cracks, and calcification may occur in the endplates and subchondral bone sclerosis. (Roberts et al 2006) Metabolic waste material has difficulty being removed, and nutrients and water may have difficultly entering the nucleus. This would affect the synthesis of proteoglycans and other constituents of the nucleus.

Injury also brings in fibrotic tissue. Disruption of the normal milieu in the nucleus occurs. Herbst states that the disc becomes more turgid. (Herbst 1970) We now have a better idea what contributes to this turgidity. With the loss of proteoglycans and therefore the loss of water and increase in fibrous cells, the nucleus, become more turgid. There is a proliferation of cytokines and matrix metalloproteinases (MMP) in the degenerating disc. (Adams & Roughley 2006, Wang et al 2006) MMPs, in particular, stromelysin, cause breakdown of the core proteins in the proteoglycans. (Kang et al 1997) There is also a proliferation of Fas/ FasL which leads to apoptosis of many cells types; IL-1α which causes cells in the nucleus to synthesize inflammatory mediators such as interleulin-6 (IL-6), prostaglandin E2 (PGE2), and nitric oxide; and TNF-α which produce a local inflammatory response and inhibit prostaglandin synthesis. (Kang et el 1997; Wang et al 2006) Nerve fibers, such as in the outer anulus, dorsal root ganglion, and spinal nerve roots, when exposed to these inflammatory mediators, may stimulate a pain response. (Kang et al 1997)

Examinations of discs from all ages has found that the semi-gelatinous consistency of the nucleus begins to disappear in the second decade of life. (Haefeli 2006) In addition, there is proliferation of fibrotic tissue in the disc. (Haefeli 2006) Scar tissue does not have the orderly orientation of the original anular tissue and are cross-linked and adhere to adjacent layers. Roberts et al note that changes to the endplate precede changes to the nucleus pulposus. (Roberts et al 2006)

There is controversy in regards to whether many of the above described changes are part of the aging process of the disc or is due to degeneration. Probably both, but these are likely to be mainly features of degeneration. Adams and Roughley opine that structural failure in disc degeneration may be the key difference between changes in the disc undergoing aging or degeneration. In degeneration, there is breakdown of tissues with tears or fissures and complete loss of differentiation between tissues. In aging, there is a gradual change in the

composition of the tissues. (Adams & Roughley 2006)

Eventually, fibrotic changes occur both in the anulus fibrosus and nucleus pulposus to the point where there is considerable internal disruption such that the anulus and nucleus become indistinguishable from one another. Synthesis of the normal elements in the disc and the water content are progressively diminishing. The matrix of the nucleus and the anular laminae disappear. The overall disc height reduces to the point where the disc is solely fibrotic tissue with no hydrophilic properties. Around the periphery, osteophytic activity is attempting to stabilize the injured and degenerating spinal motion unit. One is headed to D6 stage disc degeneration and complete or near complete stabilization of the injured spinal motion unit. Adams and Roughley state that “gross injuries to the disc never fully heal.”(Adam & Roughley 2006)

In a normal disc, nerve fibers are only found in the outermost anulus and the periphery. In a degenerating or injured disc, nerve fibers and vascular vessels have been found to enter the damaged anulus which has undergone granulation and can be found in even in damaged inner anular layers and the nucleus pulposus. (Adams & Roughley 2006, Freemont et al 1997, Johnson et al 2001, Inoue et al 2006, Osti et al 1996, Palmgren et al 1996, Palmgren et al 1999) The purpose of these fibers, or whether they can stimulate a pain response, is unknown. It is thought that inflammation of the disc may promote growth of nerve fibers, in particular, afferent nerve fibers, towards the centrum of the disc. It is hypothesized that some of the fibers might induce discogenic pain. (Inoue et al 2006)

NOTES on the Biochemistry/Histology & Structure of the Disc

The anulus fibrosus is composed of concentric layers of primarily Type I collagen in the outer region and Type II in the inner region. It surrounds and contains the nucleus pulposus, which begins as a semi-gelatinous structure that is high in Type II collagen. There is not a distinct line between the anulus and nucleus as the innermost anulus takes on more of the characteristics of the nucleus with its high percentage of Type II collagen, proteoglycan, and water. (Skaggs et al 1994) The nucleus has a high water content due to the hydrophilic proteoglycans. Quite early in life, fibrochondrocyte-type cells begin to displace other cells within the nucleus pulposus. (Kluba et al 2005)

Proteoglycans: These are formed by chains of polysaccharides, primarily karatan sulfate and chondrotin sulfate, that are bound covalently to a central protein core. These are attached by the protein core like Christmas lights to a hyaluronic acid chain and forms the proteoglycan aggrecan with 8 to 18 proteoglycans attached to the chain. (McDevitt 1988, Urban & Roberts 2004) Groups of aggrecans form the nuclear extracellular matrix along with other cells. (Urban & Roberts 2004) There are a greater proportion of unaggregated proteoglycans in the nucleus that in the anulus. (McDevitt 1988) The proteoglycans in the extracellular matrix have a negative charge. This attracts water but limits diffusion outwards. (Hukins 1988)

Collagen: These are macromolecules that are major constituents of connective tissues. They form the fibrous framework of the disc. (Eyre 1988) The polypeptides in collagen are composed of the amino acids glycine, proline, hydroxyproline, and alanine. (Stathopoulus & Cramer 1994) There are at least 11 types of collagen described. (Eyre 1988, Stathopoulus & Cramer 1994 The primary collagen in the discs are types I and II. Many of the other collagen types are found to a varying degree in the disc.

Type I collagen fibers: rope-like fibrils that consist of 2 alpha-1 chains and 1 alpha-2 chains. They are resistant to tensile stresses. (Stathopoulus & Cramer 1994) They are the most abundant collagen in the anulus.

Type II collagen fibers: small, banded fibrils that are a constituent of hyaline cartilage. It is found in the hyaline cartilaginous vertebral endplates, nucleus pulposus, and inner anulus. (Skaggs et al

1994, Stathopoulus & Cramer 1994) They resist intermittent pressure. ((Stathopoulus & Cramer 1994)

Glycosaminoglycan (GAG): These are carbohydrate polymers that bind onto a protein core to form proteoglycans. GAGs in the proteoglycans of the disc include keratan sulfate and chondrotin sulfate. (McDevitt 1988)

Outside of water, there are numerous other elements in the composition of the disc, in particular, glycoproteins. Some of these are not found in the normal disc but are found in the injured or degenerating disc. Fibronectin is one of the glycoproteins found in the extracellular matrix. It is a large protein that provides binding sites for collagen and other glycoproteins. (Chung et al 2003, Stathopoulus & Cramer 1994). The proportion of intact and fragmentary fibronectin increases in disc degeneration where fragmentary fibronectin is thought to suppress proteoglycan and DNA synthesis in the nucleus pulposus but increase both in the anulus fibrosus. (Chung et al 2003) Elastin is found in small numbers in the anulus, but it is thought to be important in the elasticity of the anulus as it is found between laminae. (Roberts et al 2006) Some of the elements found in injured or degenerating discs are described above in the text.

6. Maintaining optimal disc and spinal health.

Biomechanics of the spine and disc are largely missing from this paper. I will conclude with a statement on optimal spinal biomechanics. Wang, et al state the “both static and dynamic forces are critical in maintaining normal position and function of the cervical spine.” The static restraints are the bones and ligaments [which includes the disc]. The dynamic restraints are the “muscles forces that are transmitted across the spine.” (Wang et al 2006) In other words, biomechanically, we have an extremely complex structure – the spine – that requires substantial stability due to the forces placed upon it and the upper body and head which it must support, and it must also offer a high degree of mobility and flexibility. It has these two very different missions that it must perform perfectly. On top of that, even under ideal conditions, *wear and tear* and time take their toll.

Outside of its mechanical duties, the spine also protects an important component of the nervous system, the spinal cord, and allows the passage of nerves from the spinal cord to the periphery. It has duties providing passage of blood vessels and helps to anchor organs. It is a complex and delicate balancing act to maintain function and prevent degenerative changes.

The normal disc has distinct anulus fibrosus laminae and a semi-viscous nucleus pulposus that the anulus and the vertebral endplates must effectively contain. With aging, the anular laminae become thicker and there are more interrupted or incomplete laminae. (Roberts et al 2006) The normal nucleus pulposus of a child is gelatin-like. In the second decade of life, it shows signs of fibrotic cells. This appears to be a “normal” progression, although it is difficult to have a life without injuries to the disc. The displacement of aggrecans (aggregates of proteoglycans) by fibrotic cells reduces the ability of the disc to absorb water. This and changes to other components of the extracellular matrix reduce the ability of the disc to withstand compressive forces. The disc, zygapophyseal joints, and surrounding soft tissue help the spine withstand rotatory and shear forces to some degree. Optimal alignment and joint motion are required to resist the various forces on the spine and absorb forces efficiently, to a point. The vertebral endplates receive protection by the efficient absorption of forces on the disc. This would prevent or reduce endplate damage from forces transmitted from the disc and reduce the opportunity for damage and calcification that would reduce the nutrient transport through the endplates.

With its lack of direct vascular supply and the tremendous physical forces placed on it, the disc has a life *on the edge*. It’s function must be impeccable to maintain homeostasis and to avoid the off-ramp onto the road of degeneration. This is purely my opinion on maintaining optimal disc and spine function. First, of course, is to maintain overall fitness.

Research is finding that physical fitness is necessary for genetic expression in addition to improving overall function to all systems. In chiropractic, our philosophy is that a well-maintained spine in good alignment and proper coordination of function of the various structures minimizes the opportunity for degeneration and may slow the aging process in the disc. This would be logical from biomechanical, biochemical, and neurological standpoints. In aging, we know that the synthesis of proteoglycans diminishes over time. This would make the disc more susceptible to injury. No matter how one lives life, there are always mishaps and accidents, most quite minor, but their impact can accumulate over time. Obviously, a spine functioning optimally can maximize the ability of the spine to compensate for the effects of minor injuries and allow the nervous system to function as intended.

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