Disc “Ingestion”

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White and Panjabi have brought forth the concept that acute, post-traumatic, lower back pain and muscle spasms may be due to a rapid ingress of fluid into the nucleus pulposus that may induce pain by irritating the outer anular fibers and thereby eliciting a pain response to the peridiscal and discal nociceptors. This describes the Gonstead acute D1 “swollen” disc.

The influx of fluids increases the intradiscal pressure and may result in internal derangement of the disc, in particular, rupture of anular fibers. (Naylor 1976, White & Panjabi 1990) Studies by Pezowicz et al found that an influx of fluid into the disc and the resulting increase in hydrostatic pressure in the nucleus pulposus leads to clefts in the poorly organized inner anular layers. Continued pressure can cause further cleft formation into the middle and outer anular layers and the progression of material from the nucleus pulposus into the damaged areas. (Pezowicz et al 2006) Material in the nucleus is known to have a noxious effect on other tissues. It is not stated how the fluids enter the disc, i.e., whether through the periphery of the disc or the endplates or both.

Proteoglycan solutions in varying concentrations regulate the degree of resistance to fluid flow and the transport of solutes. (Uei et al 2006) Injury to the disc may cause a derangement of this proteoglycan solution that results in the matrix bringing in more fluids. This rapid swelling of the nucleus pulposus is thought to precipitate biochemical changes which can lead to disc degeneration. (White & Panjabi 1990) If true, this could be the mechanism that initiates the Gonstead “Stages of Disc Degeneration” that begins with the acute, swollen Gonstead D1 disc.

How do the proinflammatory mediators and cytokines that are being found in the disc enhance the fluid build-up in the disc? Their presence would obviously increase fluid levels in the disc. The degree that they contribute to disc swelling has not been stated. Studies on the mediators and cytokines, such as, interleukin-6, interleukin-8 and prostaglandin E2, appear to still be in the preliminary stages with much focus on their possible role in discogenic pain. These agents have also been found in degenerating disc. (Burke et al 2002)

In this scenario, quick restoration of function of the affected spinal functional unit would undoubtedly have a highly beneficial effect. It is obviously important to quickly restore optimal biomechanical function to the motor unit to reduce damage to the disc and other motion unit structures. Restoration of optimal neurological function of the nerve supply to the disc, zygapophyseal joints, and other structures that have been affected by the increased disc height and altered motion of the area is necessary for tissue repair. Any previously injured, inelastic and fibrotic tissues, are susceptible to further injury by this expansion of the disc space due to increased fluid content and high hydrostatic pressure. v

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