SPINE Volume 20, Number 16, p 1803

The Pathophysiology of Painful Lumbar Disorder Symposium

Introduction

The following symposium, *The Pathophysiology of Painful Lumbar Disorders*, was presented at the North American Spine Society Annual Session in Boston, Massachusetts, in 1992. Its intent was to present current research in the areas of inflammation, vascular compromise, and neurophysiology as they related to the pathophysiology of painful lumbar disorders.

The fundamental questions of what makes a lumbar spine painful remain open to debate. In the recent past, the principle focus of spinal pain research has been primarily structural and biomechanical. The spine was viewed as a structural entity capable of suffering from a variety of mechanical perturbations. Disc herniation has been viewed as causing compression of neural structures, spinal stenosis as causing a compressive lesion of the nerve root canals, and degenerative disc disease as manifesting itself as segmental instability. The structural paradigm fostered structural intervention designed to relieve compression and stabilize instability. Biomechanical testing was used to evaluate structural integrity.

The paradigm appeared to be simple and complete. However, the validation has begun to fall apart. Large, but asymptomatic, disc herniations that display structural signs of neural compression have been shown. Symptom changes after discectomy or chemonucleolysis have not been correlated with the abolition of the compressive lesion. Patients with excellent relief after the procedure may continue to show a high degree of mass effect, whereas others with persistent symptoms may show the absence of postinterventional compressive mass effect. Similarly, patients with severe nerve root canal narrowing may be asymptomatic, whereas others have severe pain. The patient response after decompression is mixed and cannot be entirely correlated with the degree of compressive relief.

The structural paradigm has not met the challenge. A paradigm shift is clearly necessary to fill in the blanks and move forward. Immunology, biochemistry, and neurophysiology have the capability of filling this void. Inflammation, immunologic, and vascular mechanisms as discussed in this symposium present a new paradigm. Although the presenters of this symposia have not collaborated on their manuscripts, the reader will clearly see a thread that blends them together. Inflammation of some type appears to be the central mechanism that activates the myriad pathways of biochemical perturbation. Efficient interruption of this process should yield beneficial effects. This chemical event will require a biochemical intervention, not necessarily a structural one. This may explain why attempts at structural solutions to the lumbar spine have not been entirely satisfactory.

The future will continue to reveal the complex interactions between inflammatory enzymes, degratory enzymes, neurotransmitters, and vascular responses. This should lead to a new array of therapeutic strategies that will address these issues on a chemical and perhaps cellular level.

In my opinion, the "better mousetrap" will emerge from the basic science laboratory. I believe the reader will find this symposium provocative and instructive.

Jeffrey A. Saal, MD
1992 NASS Program Chairman
Sports, Orthopedic and Rehabilitation
Medicine Associates
Menlo Park, California