Disc herniation into the intervertebral foramen (IVF) or osteophytic outgrowths from the margins of the apophyseal joints that project into the IVF may compress the neural structures, but in this cadaveric study of 160 lumbar foramen (age range, 35-91 years), the authors have found that they were much more commonly associated with compression and distortion of the large venous plexus within the IVF. In the absence of direct nerve compression (seen in only eight specimens), the most severe neural changes were associated with compression, congestion, and resultant dilatation of foraminal veins. Pathologic changes within and around the nerve root complex included peri- and intrafascicular fibrosis, edema of nerve roots, and focal demyelination. Inflammatory cells were notably absent. Vascular changes within the thicken fibrous sheath about damaged nerves, namely, basement membrane thickening, suggestive of endothelial cell injury also were observed. The association between vascular compression, tissue fibrosis, and endothelial injury distant from the compression may be causal—probably due to ischemia as a result of reduced venous outflow. Such observations have led the authors to propose that venous obstruction may be an important pathogenic mechanism in the development of perineural and intraneural fibrosis. [Key words: lumbar spine, intervertebral foramen, pathology, venous obstruction, ischemia, perineural and intraneural fibrosis]

It has been recognized for many years that the phenomenon of disc degeneration is closely associated with abnormalities in the anatomy and physiology of the adjacent nerve and peri- and intraneural fibrosis. In a cadaveric study, Holt and Yates[16] were able to correlate the histology of cervical disc degeneration with adjacent spinal nerve root fibrosis. Lindahl and Rexed[21] demonstrated intraneural fibrosis in 78% of dorsal nerve root biopsies taken at the time of surgery in patients operated for herniated intervertebral discs.

Based on this association between disc degeneration and neural fibrosis, several hypotheses have emerged regarding the pathogenesis of perineural and intraneural fibrosis. Marshall and Trehewie[26] and others[27] have suggested that leakage of breakdown products from the degenerating nuclei pulposi may induce a "chemical radiculitis." Some investigators[15,12] have proposed that an autoimmune response to discal material may lead to inflammation and a reactive fibrosis about adjacent nerves. Inflammation has frequently been implicated in the pathogenesis of perineural and intraneural fibrosis. However, an examination of the literature reveals that the presence of inflammation is more often presumed than proven. Many investigators have suggested inflammation, although no cell infiltrate was noted.[15,21] Recently, Jayson and colleagues[13,15,19,20] have found a defect in fibrinolytic activity correlating with the presence of chronic back pain and suggest that this enzyme abnormality may play a role in the perpetuation of chronic inflammation and scarring about nerve roots.

In this article, we present the results of studies of the pathology of the structures within and bordering the intervertebral foramen (IVF), in which we have found an association between neural fibrosis and venous obstruction and dilatation. This has led us to propose that venous obstruction is an important pathogenic mechanism in the development of perineural and intraneural fibrosis.

MATERIALS

Intact lumbar spines were removed at autopsy from 46 unselected patients 6 to 12 hours after death. Each specimen consisted of the bodies, discs, and neural arches of the lumbar spine and a small portion of the sacrum. The clinical record (name, age, sex, cause of death, and any history of back pain) was documented. A questionnaire ascertaining any history of back pain, nature, and duration was sent to the patients' general medical practitioners.

METHODS

Lumbar spines removed from unselected cadavers at autopsy were divided in the sagittal plane using a band saw. After noting the internal features of the spinal canal, each half of the spine was radiographed in the lateral plane. Both halves were fixed in formal saline. After fixation, each half-spine was cut into blocks incorporating the IVF at each level of the vertebral column, ie, L1-2, L2-3, L3-4, L4-5, L5-S1 foramen. From each spine, five fora-
ral ischemia (from whatever cause), there is intraneural microves- sel damage as well as localized demyelination. Microvascular damage results in an increased permeability, and hence endoneur- nal edema may develop. In lumbar nerve roots, in the absence of a good lymphatic drainage, the edema is cleared slowly, thus prolonging contact with the tissue and predisposing the nerve to damage and reactive fibrosis. It has long been recognized that pain, paresthesias, sensory deficits, and motor weakness are clinical manifestations of neural ischemia with or without mechanical deformation, and that the natural response to such injury in time is neural fibrosis.

Because of the nature of cadaveric studies, we have not been able to correlate the observed changes adequately with a history of back pain. As far as can be established from the patients' hospital records and the results of a questionnaire sent to the patients' general medical practitioners, all of the patients with documented back pain had both disc herniation and neural fibrosis in the absence of nerve compression. Of patients with no documented history of back problems, some had no evidence of these changes, whereas others had both disc herniation and nerve root fibrosis.

In summary, we believe that pathologic changes observed around and within the nervous tissue in the IVF are indirectly associated with disc herniation through disc protrusion causing compression of veins, leading to venous stasis and ischemia. These observations may be of therapeutic importance, as they suggest that improving venous drainage, by whatever method, may prevent neural fibrosis.

REFERENCES


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