Vibratory Response in Congenital Scoliosis

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Summary: Vibratory studies were performed on 10 adolescent subjects being followed for congenital scoliosis. The average age was 12 years 4 months and the average curve was 32°. Twenty-one age-matched controls underwent identical studies. Threshold to detection of vibratory stimuli in the upper and lower extremities was quantified. Results showed that the scoliotic subjects were consistently less sensitive than the controls in the lower

but not the upper extremity. The occurrence of hyposensitivity in only the lower extremity of persons with congenital scoliosis implies that the sensory deficit is secondary to the spinal deformity itself rather than a primary etiologic factor. Key Words: Congenital scoliosis— Neurologic testing—Posterior column—Vibratory stimuli.

Much evidence accumulated in recent years implicates neurologic dysfunction as an etiology for adolescent idiopathic scoliosis (AIS). Dysfunction of the nervous system at virtually any level can induce axial skeletal deformity during periods of rapid growth (11). Disease processes such as syringomyelia or Friedreich's ataxia which display dorsal column system dysfunction in growing children are consistently associated with an idiopathic curve pattern (5,7,17). Posterior column system dysfunction as an etiology of AIS has been implicated in both animal experiments and clinical studies.

Experimental section of the dorsal root ganglia induced lateral spinal curvature in young rabbits (8,10). Pathologic findings have been documented in dorsal root ganglia of human scoliotic spines. Scoliosis has been induced in animals through selective destruction of the posterior column at the spinal cord level, the brainstem, and the posterior hypothalamus.

Clinical studies assessing posterior column function have consistently shown significant differences between scoliotic subjects and controls. Numerous studies have shown that patients with AIS display abnormal postural equilibrium (1,2,15,16,19,20). Proprioceptive input, a prominent component of postural equilibrium, has been hypothesized to play a significant role in the etiology of AIS (9). Clinical studies have supported this theory (3). Furthermore, studies of patients with AIS have shown that this group displays hypersensitivity to vibratory input (18). Since proprioceptive and vibratory input are conducted through the dorsal column system, the finding of significant differences in response between patients with AIS and normal age-matched controls implies dysfunction at some level of the pathway. That these differences have been observed in both the upper and lower extremity implies a lesion proximal to the cervical spine and one that is primary rather than secondary.

A fundamental weakness of clinical trials which seek to examine the etiology of AIS is that assessment of whether observed differences in sensory testing are the primary etiologic factors or are secondary to the spinal deformity is often difficult. The purpose of this investigation was to evaluate the response to vibratory stimuli of patients with a scoliosis of known etiology. In congenital scoliosis, lateral spinal curvature results from a vertebral malformation. Aberrant neurologic findings believed to be etiologic factors in AIS would not be expected in this group. From this investigation, the effect of lateral spinal curvature on vibratory sensitivity may

be determined.

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