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Clinical Study

Comparison of somatosensory evoked potentials between adolescent idiopathic scoliosis and congenital scoliosis without neural axis abnormalities

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Abstract

BACKGROUND CONTEXT: Abnormal somatosensory evoked potentials (SEPs) have been documented in patients with adolescent idiopathic scoliosis (AIS) with different cure severity. However, few studies investigated whether abnormal SEPs were the cause or effect of idiopathic scoliosis.

PURPOSE: The purpose of this study was to investigate the significance of abnormal SEPs in patients with AIS, and to explore its effect on the etiopathogenesis of AIS.

STUDY DESIGN/SETTING: This study evaluated SEPs in patients with AIS and congenital scoliosis (CS) with similar curve pattern and severity both in coronal and sagittal planes.

PATIENT SAMPLE: Female patients with AIS and CS in our spine surgery center from 2000 to 2009 were recruited for this study.

OUTCOME MEASURES: Rate of abnormal SEPs.

METHODS: Posterior tibial nerve SEPs (PTN-SEPs) were performed on female patients with AIS and CS. The inclusion criteria were patients with AIS with a Lenke type 1 curve and patients with CS with right thoracic curve (apex between T5 and T12) and normal sagittal profile (kyphosis less than 50° measured from T2 to T12). All patients were evaluated with total spine magnetic resonance imaging, and those with neural axis abnormalities were excluded. The patients with neurological deficits on detailed physical examination were also excluded. Absence of SEPs waveforms or prolongation of peak latency or asymmetrical peak latency were defined as pathological change. The incidence of pathological SEPs and clinical characteristics were compared between patients with AIS and patients with CS.

RESULTS: Forty-six patients with AIS and 33 patients with CS were included in this study. There was no significant difference in coronal and sagittal Cobb angle between the two groups. The rate of abnormal SEPs was 32.6% (15/46) and 12.1% (4/33) in AIS and CS groups, respectively, and the difference was statistically significant ($p<.05$).

CONCLUSION: Somatosensory pathway dysfunction could be found in both AIS and CS without neural axis abnormalities, and the patients with AIS tended to have higher rates of somatosensory disorders than patients with CS with similar scoliosis curve, which indicates that both scoliosis curve and primary etiopathogenic factor contribute to the sensory deficit in patients with AIS.

Keywords: Adolescent idiopathic scoliosis; Somatosensory evoked potentials; Etiopathogenisis

Introduction

Much evidence in recent years has shown neurologic dysfunction to be regarded as an ultimate cause for adolescent idiopathic scoliosis (AIS) [1,2]. In growing children, diseases that alter posterior column function (eg, Chiari I malformation, syringomyelia, and Friedreich ataxia) are consistently

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EVIDENCE & METHODS

Context
Abnormal SEPs have been noted in some patients with AIS. The authors aimed to assess whether this is caused by AIS or a more upstream root reason to have AIS.

Contribution
The investigators compared two groups of patients with scoliosis with similar curve severity and patterns. One had AIS and the other congenital scoliosis. Abnormal SEPs were found more frequently with AIS than with congenital scoliosis.

Implications
The data suggest that a neurological abnormality may be a contributing factor in the development of AIS, though a true causal relationship cannot be conclusively determined.

—The Editors

associated with an idiopathic (rather than a neuromuscular) curve pattern. Experimental resection of the dorsal root ganglia induced lateral spinal curvature in young rabbits [3]. Scoliosis can be induced in animals through selective destruction of the posterior column pathways [4].

Somatosensory evoked potentials (SSEPs) are generally used to reflect posterior column pathways function. The purpose of the current study was to evaluate somatosensory function by SSEPs in patients with AIS and patients with CS with similar curve pattern and severity. 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 somatosensory function may be determined.

Materials and methods

Patients for the study were recruited from the authors' spinal center. The inclusion criteria were as follows: patients with AIS with a Lenke type 1 curve, and patients with CS with right thoracic curve (apex between T5 and T12) and normal sagittal profile (kyphosis less than 50° measured from T2 to T12). All patients were evaluated with total spine magnetic resonance imaging.

For establishing normative values of posterior tibial nerve cortical SSEPs (PTN-SSEPs) in adolescents, healthy controls were recruited from volunteer healthy youth aged from 10 to 18 years. All subjects showed no clinically detectable neurologic deficit, nor had history of trauma or surgery to the brain, spine, or lower extremities.

PTN-SSEP monitoring was performed on all subjects. The tibial SSEPs were elicited by electrical stimulation (square-wave stimulation of 200 seconds at a frequency of 1.9 Hz) at the ankle (using surface electrodes; proximally placed cathodes, the anode placed 2 cm distally) by a conventional electromyography machine (Dantec Keypoint; Medtronic Functional Diagnostics, Skovlunde, Denmark). Parameters analyzed for PTN-SSEPs were absolute latency of P40 (Lat P40), and interside differences in latency. The following criteria from Chiappa [5] were used for defining abnormal pathologic changes in SEPs: (1) absent SEP waveforms, unilateral or bilateral; (2) prolonged latencies: unilateral latency, or bilateral latencies normalized with the body height and longer than 2.5 standard deviations (SDs) over the mean values calculated from the healthy control; and (3) asymmetrical latencies: interside difference of latency or amplitude more than the mean+2.5 SD of healthy controls.

Statistics

Means and SDs of each SSEP parameter were calculated for each group. In the control group, correlation coefficients were calculated for age, body weight, and body height against latency. Bivariate regression analyses were performed using latency as the dependent variable and body height as the independent variable. Abnormal SSEPs were determined according to Chiappa's [5] criteria. The incidence of pathological SSEPs was compared between patients with AIS and patients with CS by χ^2 test. For all analyses, statistical significance was set at $p<.05$. Statistical analysis software (SPSS, ver. 13.0; SPSS, Chicago, IL, USA) was used for all statistical analyses.

Results

Normative values of SSEPs were established from healthy controls. SSEPs were obtainable in all 45 control individuals. The P40 latency and interside differences are shown in Table 1. Correlation analyses showed latency was significantly correlated with body height, but not with age and body weight. Interside difference of latency in all subjects was below 2.0 ms (mean+2.5 SD), and did not correlate with any of the anthropometric variables. Equations of linear regression using latency as the dependent variable and body height as the independent variable were Lat P40=0 .277×body height (BH) (cm)−4.467 (r=0.892). The regression line is shown in the Figure.

Included in this study were 46 patients with Lenke1 type AIS and 33 patients with CS. General information is shown in Table 2. Comparison of the two groups showed no statistically significant differences for age and coronal Cobb

Table 1
Peak latency of PTN-SSEPS ($\bar{x}$±SD, ms)

Peak latency	Left, n=45	Right, n=45	Interside difference (upper limit), n=45	Total, n=90
P40	37.927±2.335	38.007±2.429	0.75±0.50* (2.0)	37.943±2.368
N50	46.486±1.471	46.536±1.618	2.30±1.80* (6.8)	46.497±1.535

PTN-SSEPS, posterior tibial nerve somatosensory evoked potentials.
* No statistical significance, p>.05.