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Somatosensory Evoked Potentials in Cerebral Palsy After Partial Dorsal Root Rhizotomy

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• Somatosensory evoked potentials (SEPs) were studied in 20 children with cerebral palsy and severe lower extremity spasticity before and after selective partial dorsal root rhizotomy of the lumbosacral cord. The potentials from stimulating nerves in the lower extremity were abnormal in two thirds of the children before the operation, whereas the potentials were generally normal from upper extremity nerves. Dorsal root rhizotomies caused an attenuation of nerve root entry volleys recorded over the lumbar cord but did not change SEPs recorded over the cortex. The exception to this was that the incidence of abnormal sural nerve SEPs decreased postoperatively. Lumbar cord functions measured by H-reflexes or by tendon jerks were depressed following the operation. These results indicate a significant degree of abnormality of somatosensory transmission from the lower extremity in a group of cerebral palsied children with severe spasticity. Moreover, selective sectioning of approximately 50% of the dorsal root fibers in the lumbosacral cord had little influence on cortical evoked potentials.

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Spastic diplegia, one form of cerebral palsy, has been attributed to lesions involving the motor pathways in the cerebral hemisphere¹ without

having clinical signs of an accompanying involvement of the nearby sensory pathways. Somatosensory evoked potentials (SEPs), which provide an objective noninvasive way to test the integrity of these sensory pathways, have not been studied systematically in this group of patients.

We have had the opportunity to study SEPs in a group of cerebral palsied patients with severe spastic diplegia before and after partial selective sectioning of the dorsal roots in the lumbosacral region. The nerves that were stimulated to evoke SEPs arose in the lower extremities and passed to the lumbosacral cord through the roots that were to be partially sectioned. Quantification was made of the potentials evoked from the dorsal roots and spinal cord in the lumbar region and over the somatosensory cortex in the midline scalp. The effects of the rhizotomy on spinal cord monosynaptic reflexes were also determined. Surprisingly, we found a significant number of these patients had abnormal SEPs before the operation, a result that was not anticipated. We report on these findings and the modifications of SEPs and monosynaptic reflexes that follow partial selective dorsal root rhizotomies.

PATIENTS AND METHODS

We studied 20 children with spastic diplegia without dystonia before and after partial lumbar rhizotomy. Their ages ranged from 2½ to 9½ years. There were nine girls and 11 boys; four had been full-term infants and 16 had been born prematurely. Selective posterior rhizotomy

was performed as described by Fasano et al² and modified by Peacock and Arens.³ Using general anesthesia, a laminectomy was performed from L-2 to the upper sacrum. After opening the dura mater, the dorsal roots were examined with an operating microscope, separated from the ventral root, and divided into rootlets. Usually four to ten rootlets were dissected and identified in each root. Each rootlet was placed over a bipolar stimulating electrode and the threshold current for eliciting a movement of the leg identified using single pulses of 0.3-ms duration. Repetitive stimuli at this threshold at a rate of 50/s were then applied and the motor responses observed and classified as a contraction that was restricted to the appropriate myotome of the stimulated leg and was brief in duration, or a contraction that was sustained and spread to other myotomes of the same or even the opposite leg. The rootlets giving rise to the latter responses were sectioned. Approximately 50% of the dorsal nerve rootlets from L-2 to S-1 were sectioned bilaterally in these patients.

Somatosensory evoked potentials were recorded from stimulation of peripheral nerves in the legs (posterior tibial at the ankle, peroneal at the knee, and sural at the ankle) and the median nerve at the wrist in the arms. The nerves on each side of the body were tested in sequence. In addition, H-reflexes and F waves were tested in eight of the patients.

The children were sedated with chloral hydrate (50 mg/kg orally). The nerve to be studied was stimulated percutaneously with bipolar electrodes (separation of 2.5 cm) at 4.8/s with a 300-μs square-wave pulse at an intensity sufficient to cause minimal contraction of the muscles innervated by that nerve. In the case of the sural nerve, the intensity was raised to the patient's tolerance but not more than 15 mA.

Scalp recordings were made by placing

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Fig 1.—Somatosensory evoked potentials from stimulating posterior tibial nerve in subjects with cerebral palsy and severe spasticity. Lumbar root entry volleys are to left of each cortical tracing. Patients have been divided into two groups, normal and abnormal. Note that abnormality is restricted to cortical potentials and consists of their absence or marked delay of initial vertex positive component occurring at approximately 40 ms. This component is marked by filled circle above traces. Traces represent sum of three separate averages of 200 to 600 trials each.

Table 1.—Cortical SEPs in Cerebral Palsy*

	Preoperative		Postoperative	
	No. of Nerves Tested	Abnormal Tests (%)	No. of Nerves Tested	Abnormal Tests (%)
Lower extremity				
Sural	37	14 (38)	37	5 (14)†
Posterior tibial	38	11 (29)	38	10 (26)
Peroneal	30	8 (27)	30	8 (27)
Sum	105	33 (31)	105	23 (22)
Upper extremity				
Median	28	3 (11)‡	28	2 (7)‡

* SEP indicates somatosensory evoked potential.

† $P < .05$ compared with preoperative values.

‡ $P < .05$ compared with lower extremity sum.

electrodes over C_3 , C_2 , and C_4 referenced to F_{pz} for lower extremity stimulation and C_3' and C_4' referenced to F_{pz} for upper extremity stimulation. In addition, the ascending volleys for the posterior tibial nerve were recorded between surface electrodes over L-1 referenced to L-5. For the upper extremities, recordings were made between electrodes over the ipsilateral Erb's point referenced to F_{pz} and C-7 referenced to C-1, and C3' or C4' referenced to F_{pz} .

The potentials were amplified and filtered between 5 and 500 Hz for the lower extremity and between 30 and 1500 Hz for the upper extremity. A total of approximately 200 to 600 stimuli composed each average and up to three averages were

made to stimulation of each nerve.

The H reflex was obtained by stimulating the posterior tibial nerve behind the knee while recording the surface electromyogram from the calf. The maximum H and M responses (H_{max} and M_{max}) were obtained and the H_{max}/M_{max} ratio calculated.

The F wave was obtained by stimulation of the posterior tibial and peroneal nerves with a supramaximal current at a 0.5-Hz rate for 16 trials. The amplitude of the F waves obtained on these trials was defined and the mean calculated.

The latency of the components of the SEPs was defined at the peak of the component of interest. For the potentials

evoked by stimulation of lower extremity nerves, P40 latency was derived from either C_2 or the ipsilateral parasagittal electrode site. For the lumbar potentials, both the latency and amplitude (defined as the peak-to-peak value of the negative component [N22] to the following positivity) was made. For the potentials evoked by stimulating the nerves in the upper extremity, latency was defined by the peak value of the initial negative components (N8-10 at Erb's point, N14-20 at the cortex). These measures were made from the grand average of the two to three separate averages collected for each nerve tested. The evoked potentials were classified as abnormal if the cortical components (P40 from the lower extremities or N19 from the upper extremities) were absent (no reproducible component on two of the three replications) or if the latency of the cortical components from the lower extremity had been greater than 50 ms (see Zhu et al⁴ for normal values) and more than 25 ms for the upper extremity. We used χ^2 to test the significance of the effects of dorsal rhizotomy on these measures of evoked potentials.

RESULTS

Preoperative

Only five of the 16 patients tested with SEPs from all six lower extremity nerves were normal. The frequency of abnormality in the 11 affected patients involved one or two nerves in seven patients and four or more nerves in four patients (see Fig 1 for examples of normal and abnormal SEPs from posterior tibial nerve stimulation). Data from these 16 patients, when combined with those derived from the other four patients who had fewer than six nerves tested, showed that the probability of defining an abnormal SEP was slightly greater with sural nerve (14/37 instances [38%]) compared with posterior tibial and peroneal nerve (11/38 [29%], and 8/30 [27%], respectively; Table 1). The abnormality of the cortical components of the SEP occurred even though lumbar nerve root potentials were usually present and of normal latency. Thus, in children with spasticity and the diagnosis of cerebral palsy, the probability of detecting abnormalities of the P40 component of cortical SEPs from stimulating nerves in the lower extremities was approximately 30%.

We examined median nerve SEPs in 14 of these patients to ascertain whether the deficit of somatosensory processing was widespread or was restricted to the input from the lower extremities. Eight of the nine patients with abnormal posterior tibial SEPs had normal median SEPs. Furthermore, of the 28 median nerves tested, only three were abnormal. Thus, in

Table 2.—Spinal Root Volley: Incidence of Detection and Amplitude*

	Preoperative	Postoperative
Detection*		
Sural	37 (81)	36 (39)
Posterior tibial	36 (100)	33 (88)
Peroneal	32 (69)	28 (25)
Sum	105 (84)	97 (52)†
Amplitude		
Sural	0.99 ± 0.39	0.87 ± 0.54
Posterior tibial	2.14 ± 1.28	0.78 ± 0.42†
Peroneal	1.44 ± 0.67	0.74 ± 0.56†

*Detection values are number of nerves tested (percentage of nerves tested showing a spinal root volley). Amplitude values are mean ± SD.

†P < .05 compared with preoperative values.

this group of patients, the incidence of abnormal SEPs is significantly greater from stimulation of nerves in the lower extremity than from stimulation of nerves in the upper extremity (Table 1; $P < .05$).

Postoperative

The probability of detecting the nerve root volley over the lumbar region from stimulating the lower extremity nerves was reduced following the operation to 52% compared with the 84% preoperative detection rate (Table 2; $P < .05$). The effect was most striking with sural and peroneal nerves. When the lumbar volley was present postoperatively, its latency was normal. However, the amplitude of the remaining lumbar volleys to posterior tibial and peroneal nerve stimulation was clearly attenuated compared with preoperative values, whereas the lumbar volleys to sural nerve stimulation were essentially unchanged (Table 2). Thus, sectioning of approximately 50% of the dorsal roots L-2 to S-1 was accompanied by (1) a decrease in the probability of detecting a lumbar root volley from stimulating the posterior tibial, peroneal, and sural nerves, whereas (2) an attenuation in the amplitude of the remaining volleys was detected only with posterior tibial and peroneal nerve stimulation.

The overall incidence of abnormality of the cortical evoked potentials was not significantly reduced following the operation: from 33 (31%) of the 105 nerves tested preoperatively to 23 (22%) of the 105 nerves tested postoperatively. However, when the data from each nerve were analyzed separately, the SEPs from the sural nerve showed a significant normalization following the operation, changing from a 38% incidence of abnormality preoperatively to a 13% incidence postoperatively. The frequency of abnormal SEPs from stimulating pos-

terior tibial or peroneal nerve was essentially unchanged by the operative procedure (Table 1). Evoked potentials to median nerve were changed postoperatively in only one of 28 nerves tested.

H and F Responses

There were eight patients tested preoperatively and postoperatively. All had an H reflex preoperatively, with the ratio of H_{max}/M_{max} varying from 10% to 70%. Postoperatively, the H-reflex became unobtainable in five of these patients, with the other three showing shifts of the ratio, two a decrease and one an increase (Fig 2). F wave amplitudes for the 11 patients tested for peroneal and posterior tibial nerves did not show any consistent pattern as a result of the operation.

In summary, these patients with cerebral palsy and severe spasticity show a 31% incidence of abnormality of cortical somatosensory potentials evoked from stimulating mixed and sensory nerves from the lower extremities, whereas the cortical SEPs to median nerve stimulation were only occasionally (11%) abnormal. The discrepancy between the SEPs evoked by stimulating upper and lower limbs in these patients reveals a deficit of sensory processing predominantly affecting the lower extremities. The sensory processing deficit is within the central somatosensory pathways, since nerve root volleys recorded over the lumbar cord were normal in these patients.

COMMENT

The results of this study define an abnormality of cortical SEPs from stimulating nerves in the lower extremities in children with spasticity and the clinical diagnosis of cerebral palsy. Two thirds of the patients had an abnormal SEP from stimulating one or more of the lower extremity nerves. Moreover, abnormal SEPs

Fig 2.—Change in H reflexes as result of partial dorsal root rhizotomy in eight patients. Note loss or marked attenuation of H reflex postoperatively in seven of eight patients. H_{max} and M_{max} indicate maximum H and M responses; solid bars, preoperative values; and hatched bars, postoperative values.

were obtained from approximately one third of the nerves tested. We had the impression that the likelihood of defining abnormalities of SEPs was greater with the older compared with the younger subjects. We believe that this high incidence of abnormality of SEPs from the lower extremities in patients with cerebral palsy has not received attention before the present observation. The patients in our study group were highly selected from among those children carrying the diagnosis of cerebral palsy because of their severe lower extremity spasticity requiring a surgical procedure on their posterior spinal roots from L-2 to S-1 to improve their gait. It is likely that the neuropathological basis for their severe spasticity also accounts for the abnormality of SEPs obtained from stimulating nerves in the lower extremities.

We also examined median nerve SEPs in 14 of these patients to define how generalized was the abnormality of somatosensory function. We found that the SEPs from the upper extremity median nerve was normal in all but a few instances. Thus, the results suggest two major alternatives: (1)

the locus of the somatosensory disorder is in the spinal cord below the cervical region, thus affecting only the ascending pathways from the legs; or (2) there is a selective involvement of the lower extremity somatosensory pathways in the cerebral hemispheres. A distinction between these two alternatives might be made by defining whether the potentials over the cervical cord to stimulation of the lower extremity nerves were normal or abnormal. The finding of abnormal potentials would favor a spinal cord site as the basis for the selective abnormality of lower extremity SEPs in these children. A cerebral site for the lesion producing spasticity was proposed by Little,⁵ who asserted that, in this condition, there was a particular involvement of the fibers in the periventricular region controlling the lower extremities. The results from the present study can exclude the peripheral nerve as a source of abnormality contributing to the SEP findings, since the latency and amplitude of the spinal root and lumbar cord volleys were within normal limits for these patients.

It came as a surprise that there were so few changes in the cortical SEPs following sectioning of approximately 50% of the dorsal root fibers from L-2 to S-1 bilaterally. In contrast, the potentials representing the entering dorsal root volleys recorded from over the low back were affected with both the probability of their detection and their amplitudes being diminished. The only significant

change in cortical SEPs following the operation was to sural nerve stimulation, with the incidence of abnormality being reduced from 33% preoperatively to 13% postoperatively. One mechanism that might account for the postoperative improvement of sural nerve SEPs is based on a change in the interaction between the various somatosensory inputs in the spinal cord.⁶ The selective character of the rhizotomy could have affected the balance between the different types of sensory inputs, thereby altering the types of interaction taking place in the cord and the resulting SEPs. This suggestion is supported in part by the clear evidence that the rhizotomy produced an impairment of muscle spindle afferent input (loss of H-reflexes and depression of deep tendon reflexes) without clinical evidence of altered cutaneous sensibility.

The remarkable preservation of cortical SEPs following section of the dorsal rootlets supports prior clinical observations⁷ of the preservation of cortical SEPs even after considerable damage to peripheral nerves. The results from the present study provide a quantitative definition of this phenomenon such that with even 50% of the dorsal root fibers sectioned over several segments, cortical SEPs are little affected.

There were changes of spinal cord function following the rhizotomies as evidenced by the loss or diminution of H-reflexes in most of the patients tested. Clinically, these patients also showed a reduction in tendon reflexes

in the lower extremities, and they were deemed to be less spastic postoperatively. F-wave analysis, which measures changes in motor neuron pool excitability, were too variable to allow definition of significant postoperative changes.⁸ It may be of interest, in future cases, to assess the long-loop reflexes as a way of measuring the effects of the operation on their spasticity.

One important lesson to be gleaned from this study is that there can be a marked reduction in the number of axons conveying sensory input through the dorsal roots without significant changes in the SEPs from peripheral nerve stimulation. This finding is relevant for understanding the utility of SEPs in root lesions such as disk herniations, which, in our experience, are normal. A measure of somatosensory function employing stimulation of the whole nerve by electrical means obviously has limits in assessing such lesions, and alternative strategies of natural activation⁹ of the somatosensory periphery by touch, muscle stretch, and temperature need to be developed to test the effects of such partial lesions. In contrast, SEPs to electrical stimulation of peripheral nerves are quite sensitive for revealing lesions of the central pathways, as evidenced in this group of patients without sensory complaints but with clear evidence of altered somatosensory transmission.

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