A SYSTEM FOR THE ELECTROPHYSIOLOGICAL MONITORING OF 
THE SPINAL CORD DURING OPERATIONS FOR SCOLIOSIS

S. J. JONES, M. A. EDGAR, A. O. RANSFORD, N. P. THOMAS

From the Royal National Orthopaedic Hospital, London

An electrophysiological system for monitoring the spinal cord during operations for scoliosis is described. During the development of the technique the recording of cortical somatosensory evoked potentials from the scalp and spinal somatosensory evoked potentials from the laminae or spines was superseded by the positioning of recording electrodes in the epidural space cephalad to the area to be fused. All recordings were made in response to stimulation of the posterior tibial nerve at the knee. Results in 138 patients are presented and the findings in three patients who exhibited neurological deficits after operation are described. It is concluded that spinal somatosensory evoked potentials are sensitive to minor spinal cord impairment, possibly due to ischaemia, and that these changes may be reversed when the cause is quickly remedied. The monitoring system interferes minimally with anaesthetic and surgical procedures and is now performed as a routine.

The surgical correction of spinal deformities with a distraction force, as in Harrington instrumentation, carries a small but significant risk of neurological complications due to lesions of the spinal cord (MacEwen, Bunnell and Siram 1975). In order to reduce the incidence of such problems several methods of evaluating spinal cord function have been introduced, with the aim of detecting impairment at an early and reversible stage.

One method of intra-operative assessment is the "wake-up" test (Vauzelle, Stagnara and Jouvinroux 1973; Hall, Levine and Sudhir 1978). This provides only a qualitative assessment of motor function at a particular moment, whereas an ideal system would be both quantifiable and continuous. In addition anaesthetic difficulties have been noted.

When a brief electrical stimulus is applied transcutaneously to a large nerve trunk in the leg, somatosensory evoked potentials (SEPs) can be recorded from the scalp overlying the sensorimotor area of the cerebral cortex (Tsumoto et al. 1972). The afferent nerve volley can also be detected in the epidural space adjacent to the spinal cord (Shimoji et al. 1972). Both types of response are naturally dependent on the integrity of intervening segments of the spinal cord. Nash et al. (1977) and Engler et al. (1978) have described the use of cortical recordings of SEPs to monitor spinal cord function during Harrington instrumentation. Tsuyama et al. (1978) and Tamaki et al. (1981) recorded ascending and descending activity in the spinal cord peroperatively, by placing both stimulating and recording electrodes in the epidural space.

This report describes the development of an epidural monitoring technique and its use in 138 patients undergoing Harrington instrumentation. The findings in three patients who exhibited neurological deficits after the operation are considered in more detail.

MATERIAL AND METHODS

One hundred and thirty-eight patients underwent Harrington instrumentation for the correction of scoliosis. The mean age was 15 years (range 8 to 42 years) and 94 patients were female. The breakdown of diagnoses is shown in Table I.

After induction of anaesthesia, bipolar stimulating electrodes were strapped to each leg over the posterior tibial nerve in the popliteal fossa, and an earthing electrode was secured to one thigh. The stimulus was

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic scoliosis</td>
<td></td>
</tr>
<tr>
<td>adolescent</td>
<td>88</td>
</tr>
<tr>
<td>juvenile</td>
<td>6</td>
</tr>
<tr>
<td>infantile</td>
<td>1</td>
</tr>
<tr>
<td>Congenital scoliosis and kyphoscoliosis</td>
<td>22</td>
</tr>
<tr>
<td>Neurofibromatosis</td>
<td>6</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>15</td>
</tr>
</tbody>
</table>

Table I. Diagnosis in 138 patients undergoing Harrington instrumentation
provided by a square-wave electrical impulse of duration 0.2 milliseconds and intensity between 25 and 150 volts (usually 100 volts), sufficient to produce a direct motor response in the foot. This was delivered at a rate of 2, 10 or 20 per second, according to the nature of the response to be recorded.

In the first three patients disc electrodes (silver plated with silver chloride) were attached to the scalp overlying the lower leg and foot region of the sensorimotor cortex (on the midline two centimetres posterior to the vertex) and at a midfrontal position 12 centimetres above the nasion (reference electrode). Signals recorded in the first 100 or 200 milliseconds following the delivery of each stimulus (presented at two per second) were amplified using a Medelec MS6 electromyograph recorder (frequency response approximately 100 hertz to 1.5 kilohertz), and fed into a signal-averaging module consisting of 100 memory addresses. This computed an average response, usually to 100 stimuli, with a resolution of one or two milliseconds.

In a further 20 patients the spinal cord potentials were recorded from the posterior elements of the spine cephalad to the area to be fused. Various recording devices were used, including a Michel clip fixed to the spinous process and a stainless-steel screw inserted into the base of the proximal spinous process. A more satisfactory technique was to attach the lead to an extra Harrington hook which was located in a facet joint. The reference electrode (a stainless-steel needle) was inserted into muscle at the same vertebral level. Signals occurring up to 25 milliseconds after each stimulus were amplified with a bandwidth of 100 hertz to 1.5 kilohertz, and 200 to 400 responses were averaged with a stimulation frequency of 10 per second and a resolution of 0.2 milliseconds.

A further 115 patients were monitored by recording spinal SEPs from the epidural space at low cervical or upper thoracic level. The recording electrode was a blunted length of stainless-steel needle, 3 millimetres long by 0.9 millimetres in diameter, at the end of a lead of the same external diameter. This was introduced into the dorsal epidural space at the midline by means of a 16-gauge catheter, and advanced by a few centimetres. The reference needle was inserted into muscle at a similar level. Initially the stimulation, amplification and signal-averaging indices were as previously stated, but for the last 52 cases (using a Medelec MS91 electromyograph recorder) the frequency response was from 200 hertz to 2 kilohertz, the stimulation frequency was 20 per second and 200 to 1000 responses were averaged with a resolution of 0.033 milliseconds.

Cortical or spinal SEPs were recorded in response to stimulation of either leg, first of all just before the application of corrective forces and then at regular intervals until shortly before closure of the wound. A permanent record was kept on photographic or heat-sensitive paper.

RESULTS

Cortical SEPs (three patients). Well-formed responses, consisting of at least two positive and two negative deflections, were recorded while the patients were conscious. The initial positivity had a latency of onset of about 30 milliseconds, with the peak occurring approximately 35 milliseconds after the stimulus. After induction of anaesthesia only this first positive component was consistently identifiable, with unchanged latency but greatly reduced amplitude. Throughout the course of the operation the response showed wide fluctuations in amplitude, apparently related to minor changes in blood pressure and the depth of anaesthesia. Although in these three cases there were no neurological complications after operation, the spurious amplitude fluctuations were likely to have masked any small changes that might have been caused by impaired spinal cord conduction.

Vertebral spinal SEPs (20 patients). After induction of anaesthesia and exposure of the upper thoracic vertebrae the response recorded from posterior elements of the spine consisted of two or three negative deflections, occurring 14 to 22 milliseconds after the stimulus. The waveform did not appear to be affected by small fluctuations in blood pressure or anaesthetic level, but considerable interference was encountered due to surgical manipulation and to instruments and blood coming into contact with the electrodes. Marked changes of up to 60 per cent of amplitude were frequently observed after application of distraction, but since the electrode was sometimes displaced and no patients developed neurological symptoms, this was thought to have been artificial. The method was therefore abandoned in favour of the epidural recording technique.

Epidural spinal SEPs (115 patients). The epidural recording electrode was found to be easy to insert, and the leads were unobtrusive to the surgeon. The incidence of technical failure was initially quite high, but fell to approximately five per cent after acquisition of more modern equipment. No responses could be recorded from two partially paraplegic patients (both had spina bifida), one patient with Friedreich's ataxia and one with peroneal muscular atrophy. However, the remaining 111 patients were successfully monitored at upper thoracic levels (C6 to T3; 102 cases) or midthoracic levels (T3 to T7; nine patients).

The waveform and characteristics of the spinal SEP have been described elsewhere (Jones, Edgar and Ransford 1982). At low cervical or upper thoracic levels at least three negative deflections could be resolved, occurring approximately 15, 17 and 19 milliseconds after the stimulus (Fig. 1). The amplitude of the response was between 0.3 and 7 microvolts, with a mean of 2.4 microvolts. By recording at more than one vertebral level in 11 cases it was shown that the three major components could be attributed to activity in different fibre groups or tracts, with conduction velocities ranging from 45 to 80
metres per second. The response was not affected by small fluctuations in anaesthetic level or blood pressure, nor by varying the stimulation frequency between 2 and 20 per second. Although for brief periods it was not possible to record, owing to high-amplitude diathermy and movement artifacts, for the greater part of the operation good quality recordings could be obtained with a feed-back time of as little as 10 seconds.

Table II compares initial recordings with those obtained just prior to closure of the wound. There was less than 10 per cent change in the amplitude of the response for 118 out of 212 limbs stimulated. For 22 limbs there was a significant amplitude increase. The largest amplitude increase was in the order of 130 per cent. An amplitude reduction was seen for 71 limbs and was more than 50 per cent in only one of these; in this patient (already partially paraplegic on account of an intramedullary tumour) the final response was attenuated by about 80 per cent on one side and no response could be recorded at any time on the other, but there was no increase in the severity of neurological symptoms after operation. The reduction of amplitude occurred unilaterally in 16 cases. In one patient (with reliable recordings for one leg only) no response could be identified in the final record. This was one of three patients who developed mild neurological symptoms after operation; all three are described in detail below. Comparing 81 idiopathic with 30 non-idiopathic cases, there was little difference in the overall incidence of amplitude reductions (32 per cent of limbs compared with 40 per cent).

Table II. Number of limbs showing SEP alteration (epidural response) between initial and final recordings. stimulation of 212 limbs in 111 cases

<table>
<thead>
<tr>
<th>Change in amplitude (per cent)</th>
<th>Number of limbs</th>
<th>Change in latency (milliseconds)</th>
<th>Number of limbs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increase</td>
<td></td>
<td>&gt;0.9</td>
<td>11</td>
</tr>
<tr>
<td>&gt;50</td>
<td>7</td>
<td>0.6-0.9</td>
<td>22</td>
</tr>
<tr>
<td>31-50</td>
<td>4</td>
<td>0.2-0.5</td>
<td>24</td>
</tr>
<tr>
<td>11-30</td>
<td>11</td>
<td>(±0.1)</td>
<td>80</td>
</tr>
<tr>
<td>No change</td>
<td>(±10)</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Decrease</td>
<td></td>
<td>0.2-0.5</td>
<td>2</td>
</tr>
<tr>
<td>11-30</td>
<td>54</td>
<td>0.6-0.9</td>
<td>2</td>
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<tr>
<td>31-50</td>
<td>16</td>
<td>&gt;0.9</td>
<td>1</td>
</tr>
<tr>
<td>&gt;50</td>
<td>1</td>
<td>Absent response</td>
<td>1</td>
</tr>
<tr>
<td>Absent response</td>
<td></td>
<td></td>
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</tbody>
</table>

Case 2. Figure 1—Adolescent idiopathic scoliosis. (A) Initial responses recorded from the epidural space at C7 level following stimulation of the posterior tibial nerves. The waveform is resolved into three or four components (negative polarity up). (B) Absent responses recorded 20 minutes after spinal distraction. Note unchanged stimulus artifact at the start of each trace. (C) Partially restored responses with highly abnormal waveform recorded after release of distraction (see text for details). Figure 2—Epidural responses recorded at various levels after loss of the response at C7. Although present, the responses recorded at T6 and T4 are of low amplitude and abnormal waveform (not resolved into distinct components).
The latency of the response varied by no more than 0.1 milliseconds for 80 limbs, but was increased in 127 limbs (Table II), the greatest change being 1.2 milliseconds. A unilateral increase in latency was observed in eight cases. A reduction in latency was noted for four limbs (three patients) but in all of these the position of the tip of the electrode had been slightly displaced.

Transient changes in SEPs, partially or completely recovering before closure of the wound, were noted for nine limbs (six patients). For four limbs (three patients) there was a general amplitude reduction of between 31 and 50 per cent, but in one of these the change occurred and recovered before the application of distraction. In the other two the response deteriorated shortly after the force was applied, but recovered spontaneously in less than 20 minutes.

In one patient there was a unilateral flattening of the waveform after distraction, while the third component was lost on the other side. Both responses recovered fully when instrumentation was removed and the halo-tibial weights were reduced by 70 per cent, but deteriorated again when the distraction force was reapplied. The Harrington rod was finally laid into position without distraction. This produced no significant impairment of the response, and there were no neurological symptoms or signs after operation.

The last two patients in whom transient SEP changes occurred exhibited neurological deficits after operation, and are therefore described in more detail along with one patient in whom the final recording was flat.

PATIENTS DEVELOPING NEUROLOGICAL COMPLICATIONS

Case 1. A boy aged 15 years had thoracic scoliosis which was diagnosed as Noonan's syndrome. There were no abnormal neurological signs before operation. Technical problems were encountered in this relatively early case such that little credence could be given to recordings obtained in response to stimulation of the right leg. Recordings from the left leg showed the waveform flattening 15 minutes after the initial Harrington rod distraction, but recovering when the distractive force was reduced. No further distraction was applied, but the response was lost once more just before closure of the wound. The patient was soon roused and motor function appeared to be normal; however, some spasticity was subsequently noted in the lower limbs, resolving in about 24 hours.

Case 2. A girl aged 16 years had adolescent idiopathic scoliosis. Halo-tibial traction was applied before operation, with 6 kilograms of force present during the operation. Approximately 20 minutes after Harrington rod distraction the waveform became flat on both sides (Fig. 1). It was noted that the amplitude of the background noise and the stimulus artifact were unchanged, suggesting that there had been no technical breakdown. A "wake-up" test was performed, and although the patient was able to respond to instructions with either hand, no voluntary movement of the feet could be elicited.

The patient was then reanaesthetised, the halo-tibial weights were reduced by 50 per cent and the Harrington rod and bone graft removed. This produced no immediate improvement in the SEP waveform. The electrode was then relocated at a number of spinal levels. Responses were present at T4, T6, T10 and L3, but at the first two sites the waveform was unusual in that only a broad biphasic wave was present (Fig. 2). The electrode was then replaced at C7 level and a bilateral laminectomy performed at T4 to T5 and T5 to T6, during which no pressure on the spinal cord was noted.

After laminectomy a response was again recorded at C7, although the waveform was highly abnormal (see C of Fig. 1). This showed no signs of further change while bone grafting was undertaken without instrumentation. On recovery the patient was able to move both feet, but later noticed paraesthesiae in the right leg, particularly over the lateral aspect of the foot, and a band of numbness around the chest at the level of T4 to T6. Catheterisation was initially required, but after its removal the patient suffered episodes of incontinence. The paraesthesiae and incontinence resolved in approximately two weeks, but truncal numbness was residually present nine months later.

Case 3. Figure 3—Juvenile idiopathic scoliosis. Sequential recordings (left and right leg) showing reduction of amplitude and loss of the first component in the second recording on the left (28) after mild trauma to the conus medullaris. The response is fully recovered by the seventh recording (38) approximately 30 minutes later, while that from the right leg is unchanged throughout.
Case 3. A girl aged 14 years had juvenile idiopathic scoliosis. During Luque intersegmental wiring of the lumbar vertebrae both legs were seen to jump once when a wire loop came into contact with the conus medullaris. Immediately after this the overall amplitude of the response to stimulation of the left leg was reduced by about 30 per cent, while the first component was completely abolished (Fig. 3). On the right the waveform was unchanged. During the next 30 minutes the amplitude gradually recovered and the first component reappeared. No further deterioration occurred while instrumentation was completed. After operation the patient noticed paraesthesiae and numbness of the lateral aspect of the left thigh; this resolved in three days.

**DISCUSSION**

The relevant experimental work in animals has been directed mainly at three questions. First, can the degradation of spinal and cortical SEPs be taken as a sensitive indicator of spinal cord dysfunction, particularly in view of the fact that the anterior motor tracts are likely to be more at risk than the sensory posterior columns? Secondly, at what point do SEP changes and associated neurological deficits become irreversible? Lastly, what is the likely mechanism of spinal cord damage caused by excessive distraction?

Several groups have studied the electrophysiological and neurological effects of experimental spinal cord compression (for example, Croft, Brodkey and Nilsen 1972; Kojima et al. 1979) but these reports are not, perhaps, directly relevant to the present study. Nordwall et al. (1979), however, applied destructive force to two adjacent lumbar vertebrae in cats and looked for any correlation between changes in the spinal SEP and impairment of motor function, assessed by a "wake-up" test. No significant motor deficit was observed when the distraction produced up to 50 per cent attenuation of the response, but when further distraction resulted in an amplitude loss of more than 75 per cent a severe impairment was seen. If the displacement of the vertebrae was not further increased the response recovered to a considerable degree in the 30 minutes after removal of the apparatus, and the motor deficits were not so severe as those seen while distraction was still in force.

Dolan et al. (1980) employed similar distraction and recording techniques, stretching the lumbar spine by 0.25 centimetres every 10 minutes until there was significant degradation of the spinal SEP (usually abolition of late components). At this critical point there was found to be a 50 per cent reduction of blood flow in the spinal cord within and immediately caudal to the distraction zone, although the gross histological appearance of the cord was normal. When distraction was continued for an additional 0.5 centimetres after the SEP was completely abolished there was virtually no blood flow up to two centimetres caudal to the distraction zone, yet microscopic examination of the cord revealed only a few small haemorrhages. This suggested that damage to the spinal cord incurred during distraction is likely to be due to ischaemia rather than to mechanical deformation of neuronal tracts.

Also significant in the context of operations for scoliosis are the results of Brodkey et al. (1972), who found that a lesser degree of cord compression was necessary to produce conduction block (attenuation of cortical SEPs) when applied in the presence of moderate ischaemia. Kobrine, Evans and Rizzoli (1979) reported that generalised ischaemia alone might cause abolition of spinal SEPs only after approximately 10 minutes, and that after reinfusion of blood a similar interval might elapse before the response recovered. So far, therefore, the experimental evidence is somewhat incomplete, but it does appear that SEPs may be used as an early indicator of spinal cord dysfunction and that the deficits incurred may not be permanent, provided the force and duration of distraction are not above critical levels.

In Case 2 the mild but definite symptoms after operation confirmed that the loss of the SEP was not merely a false alarm, and it was believed that early detection of the conduction block may have prevented more serious symptoms. In addition to the three patients in whom major degradation of the response was correlated with the signs after operation, there were a large number of cases in whom minor SEP changes were not associated with any neurological deficits. It is not clear whether an amplitude reduction of, for example, 30 per cent signified a partial blockage, or whether the decrement could be accounted for by trivial factors such as movement of the tip of the electrode relative to the spinal cord. As with the animal studies already described, however, it does appear that a general amplitude reduction of more than 50 per cent or complete loss of one component can be regarded as signifying a high likelihood of spinal cord impairment.

In the present study the spinal SEPs recorded from the epidural space were considered more useful than vertebral or cortical responses, on account of their greater electrical stability and lesser susceptibility to anaesthetic agents and fluctuations in blood pressure. Spinal responses can be recorded at much faster stimulation rates than cortical responses, with a corresponding improvement in feedback time. There is also evidence that distinct components of the spinal SEP may be generated in different tracts of the spinal cord (Tsuyama et al. 1978), whereas the cortical response is likely to depend on the posterior columns alone (Halliday and Wakefield 1963).

In previous studies where epidural SEPs were used for monitoring the spinal cord the stimulating electrodes were also located in the spinal canal, rather than over a peripheral nerve trunk (Tsuyama et al. 1978; Tamaki et al. 1981). This has the advantage that the response is of much larger amplitude, but with the aid of modern signal-averaging devices the small amplitude of epidural potentials evoked by peripheral nerve stimulation is not a major problem. In the study by Tsuyama et al. (1978) the potentials recorded cephalad and caudal to the stimulation site were of similar waveform, suggesting they were probably reflecting orthodromic and antidromic activity in the same tracts. Only two major components were identifiable, and there was no evidence
that either was likely to arise in anterior motor tracts. Lesion studies in cats suggested that the faster activity probably originated in the lateral white matter and the slower polyphasic waves in the posterior columns. This may also be the case for the first and last components of the peripherally evoked response, since conduction velocities within the spinal cord were very similar (Jones et al. 1982). One may conclude from this that the only real advantage of the epidural stimulation technique is the larger amplitude of the response, while the peripheral stimulus is potentially less hazardous, less likely to give rise to unmanageable stimulus artifacts, and requires only one pair of electrodes to be manipulated by the surgeon.

With regard to the future of monitoring the spinal cord, it is now possible to justify the recording of epidural SEPs throughout any operation where there is significant risk of neuronal damage, and it is hoped that the technique may become widely adopted in the operative treatment of scoliosis.

This project was greatly assisted by an equipment grant from Action Research for the Crippled Child. We are also indebted to Mr J. Pitman for the design of electrodes, and to Ms L. Carter for technical assistance.

REFERENCES


