Vestibular-evoked muscle responses in patients with spinal cord injury

J. F. Iles, 1 Alima S. Ali 1 and Gordana Savic 2

1 Department of Zoology, University of Oxford, Oxford and 2 National Spinal Injuries Centre, Stoke Mandeville Hospital, Aylesbury, UK

Correspondence to: J.F. Iles, Department of Zoology, South Parks Road, Oxford OX1 3PS, UK
E-mail: john.iles@zoo.ox.ac.uk

Summary
The vestibular system was activated by galvanic electrical stimulation in 22 patients with spinal cord injury. Three patients were studied standing and all were studied sitting. Electromyographic responses recorded in soleus (standing patients) and the erectors spinae (all patients) were compared with data from 18 control subjects. The vestibular stimulus polarity and head position were arranged so as to produce excitatory medium latency muscle responses in the controls. Responses in the patient group were present bilaterally, present unilaterally or absent below the level of injury. The amplitude of response recorded in erectors spinae at lumbar levels below the lesion in 21 patients (left and right side responses summed) and five control subjects was positively correlated with American Spinal Injuries Association (ASIA) grade: the smallest amplitudes were found in patients with the most severe impairment (Spearman rank correlation coefficient \( r_s = 0.59; P = 0.002 \), two-tailed). The latency of response (averaged for both sides) was negatively correlated with ASIA grade in 21 patients: the longest latencies were found in patients with the most severe impairment (\( r_s = -0.57; P < 0.01 \), two-tailed). Amplitude and latency were negatively correlated (\( r_s = -0.72, P < 0.002 \), two-tailed). The latencies of responses recorded in the erectors spinae at different vertebral levels were linearly related to the vertical distance from the inion to the recording site in both patient and control groups. The conduction velocities of the spinal pathways activated by vestibular stimulation were 4.6 and 10.4 m/s in patient (recording below lesion) and control groups, respectively. Both clinical status (patients recording below lesion, patients recording above lesion and controls) and distance were significant predictors of latency (general linear model, \( P < 0.0005 \)). It is concluded that measurement of vestibular-evoked responses could provide information on the level and density of spinal cord lesions.

Keywords: vestibular system; spinal cord; spinal cord injury

Abbreviations: ASIA = American Spinal Injury Association; MEPs = motor evoked potentials; ML = medium latency; SCI = spinal cord injury; TMS = transcranial magnetic stimulation


Introduction
A number of physical techniques have been used to monitor injury in the spinal cord (McDonald and Sadowsky, 2002). Amongst them (McKay et al., 1997) is transcranial magnetic stimulation (TMS). TMS applied over the motor cortex activates corticospinal neurons. The corticospinal system excites spinal motoneurons and causes motor evoked potentials (MEPs) in particular muscle groups depending upon the area of cortex stimulated. In cases of spinal cord injury (SCI), MEPs can be absent, or if present show a higher than normal threshold and occur at longer latency than in controls. TMS has also been used in longitudinal studies after injury (Smith et al., 2000). The lateral corticospinal tracts are located in the posterior and lateral spinal cord, extending slightly anterior to the central canal (Nathan et al., 1990). The results of TMS presumably reflect the integrity of those regions. Motor pathways originating in the brainstem (reticulospinal and vestibulospinal), which have an important role in postural control (Lawrence and Kuypers, 1968), are located in the anterior and lateral parts of the spinal cord (Nathan et al., 1996). We have studied postural control pathways in cases of SCI in order to develop procedures analogous to those using TMS, but which could monitor the integrity of a different part of the spinal cord. We have used galvanic electrical stimulation of the vestibular apparatus to...
activate the brainstem pathways. This induces medium latency (ML) EMG responses in limb muscles of standing control subjects. Most patients with SCI cannot stand and these responses disappear if the subject sits. However, we have confirmed recently that ML responses can be obtained in paravertebral (erectores spinae) muscles in sitting control subjects (Ali et al., 2003). Study of the erectores spinae muscles therefore has the double advantage of being applicable to sitting patients and of monitoring almost all segmental levels.

**Methods**

**Subjects**

Twenty-two patients, 19 male and three female, between the ages of 21 and 71 years (median age 34.5 years; height 1.79 ± 0.09 m, mean ± SD; see Table 1), were recruited from in-patients and out-patients of the National Spinal Injuries Centre. Seventeen patients had experienced traumatic SCI, at or below C4 motor level. In two cases, the lesion resulted from infection, in two cases from a tumour and in one case from a vascular accident. Lesions occurred from 9 to 1380 weeks before the experiment and the patients were stable at the time of the experiment. Patients representing all the clinical grades of injury were recruited from American Spinal Injury Association (ASIA) grade A (complete, i.e. no motor or sensory function preserved at S4–S5) to D (incomplete, i.e. motor function preserved below the neurological level, and at least half of the key muscles below the neurological level with a muscle grade of 3, i.e. active movement against gravity, or more), as defined by the ASIA (Frankel et al., 1969; Maynard et al., 1997). Eighteen subjects with no history of neurological disorder, six male and 12 female, between the ages of 18 and 59 years (median age 24 years; height 1.67 ± 0.09 m, mean ± SD) from a previous investigation (Ali et al., 2003), were used as controls. All subjects gave informed, written consent. Procedures conformed to the Declaration of Helsinki and were approved by the Aylesbury Vale Local (study number: C01.057) and Central Oxfordshire (study number: C01.057) Research Ethics Committees.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Height (m)</th>
<th>Weeks from injury</th>
<th>SCI motor level</th>
<th>ASIA grade*</th>
<th>Stim. (mA)</th>
<th>Response below lesion left</th>
<th>Response below lesion right</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>51</td>
<td>1.85</td>
<td>157</td>
<td>T12 D**</td>
<td>2</td>
<td>Present L1?*</td>
<td>Present L1* and soleus</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>66</td>
<td>1.78</td>
<td>139</td>
<td>T12 D**</td>
<td>2</td>
<td>Present L3?‡‡</td>
<td>Present L3 and soleus‡‡</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>33</td>
<td>1.70</td>
<td>37</td>
<td>T7 A</td>
<td>1</td>
<td>Absent L3</td>
<td>Absent L3</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>31</td>
<td>1.85</td>
<td>37</td>
<td>C7 D**</td>
<td>1.25</td>
<td>Present L3 and T8</td>
<td>Present L3 and T8</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>36</td>
<td>1.79</td>
<td>19</td>
<td>C4 A</td>
<td>1.5</td>
<td>Present L3 and soleus</td>
<td>Present L3</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>31</td>
<td>1.85</td>
<td>32</td>
<td>T2 A</td>
<td>2</td>
<td>Absent T9</td>
<td>Absent T9</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>57</td>
<td>1.75</td>
<td>27</td>
<td>T9 B</td>
<td>1</td>
<td>Not recorded</td>
<td>Not recorded</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>29</td>
<td>1.80</td>
<td>103</td>
<td>T6 A</td>
<td>2.5</td>
<td>Absent T12</td>
<td>Absent T12</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>F</td>
<td>30</td>
<td>1.65</td>
<td>20</td>
<td>T9 A</td>
<td>2</td>
<td>Present T12</td>
<td>Present T12</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>52</td>
<td>1.57</td>
<td>9</td>
<td>L1 C</td>
<td>1.5</td>
<td>Present L4‡‡</td>
<td>Present L4‡‡</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>M</td>
<td>25</td>
<td>1.75</td>
<td>321</td>
<td>C7 C</td>
<td>0.8</td>
<td>Present L2 and T7</td>
<td>Present L2 and T7</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>M</td>
<td>43</td>
<td>1.80</td>
<td>34</td>
<td>T6 A</td>
<td>2</td>
<td>Present L2</td>
<td>Present L2</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>F</td>
<td>47</td>
<td>1.70</td>
<td>32</td>
<td>T4 D‡</td>
<td>2</td>
<td>Absent L2</td>
<td>Absent L2</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>M</td>
<td>58</td>
<td>1.87</td>
<td>1380</td>
<td>C5 D*</td>
<td>2</td>
<td>Present L3 and T7</td>
<td>Present L3 and T7</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>M</td>
<td>25</td>
<td>1.85</td>
<td>44</td>
<td>T3/4 C*</td>
<td>1.5</td>
<td>Absent L3</td>
<td>Present L3‡‡</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>M</td>
<td>21</td>
<td>1.85</td>
<td>21</td>
<td>C6 B</td>
<td>2</td>
<td>Present L3</td>
<td>Present L3</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>M</td>
<td>26</td>
<td>1.78</td>
<td>11</td>
<td>T2/3 A</td>
<td>2</td>
<td>Absent L3</td>
<td>Absent L3</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>M</td>
<td>42</td>
<td>1.77</td>
<td>194</td>
<td>T6 C</td>
<td>2</td>
<td>Present L3‡‡</td>
<td>Present L3‡‡</td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>M</td>
<td>25</td>
<td>1.80</td>
<td>13</td>
<td>T3/4 A</td>
<td>1.5</td>
<td>Absent L3</td>
<td>Absent L3</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>M</td>
<td>39</td>
<td>1.73</td>
<td>52</td>
<td>T3/4 A</td>
<td>2</td>
<td>Absent L3</td>
<td>Absent L3</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>M</td>
<td>71</td>
<td>2.00</td>
<td>32</td>
<td>T9 A</td>
<td>1.5</td>
<td>Absent L3</td>
<td>Absent L3</td>
<td></td>
</tr>
<tr>
<td>22</td>
<td>M</td>
<td>29</td>
<td>1.83</td>
<td>28</td>
<td>C5 C</td>
<td>2.5</td>
<td>Present L3</td>
<td>Present L3</td>
<td></td>
</tr>
</tbody>
</table>

*Frankel et al. (1969); Maynard et al. (1997); **could stand without support; †no responses to TMS; ‡measured with head facing forward; §§responses did not reverse with electrode polarity; ¶could stand only with support.
the mastoid processes. Stimulus duration was 0.4 s. In control
subjects, we routinely used a stimulus of 2 mA. Nine of the 22
patients complained of an unpleasant feeling of disorientation and
were studied with weaker stimuli (Table 1).

In the first two patients, we also activated the corticospinal tract by
TMS of the motor cortex at the vertex. The results are included in
Table 1. This part of the investigation was not continued because
examination of both corticospinal and vestibular responses made the
experimental session uncomfortably long for the subjects.

Surface EMG records were taken bilaterally from soleus and
tibialis anterior in the three standing patients. EMG records were
taken from the erectors spinae muscle at one or two levels on both
sides in all patients, using electrodes aligned along the muscle 45 mm
lateral to the midline. At least 40 rectified and integrated records
were averaged for each tested combination of stimulus polarity
(anode on right or left mastoid process) and head position (turned
right, turned left or facing forward). In the patients, it was originally
our aim to record both above and below the lesion, but not to know
the clinical details when assessing whether responses were present or
not. To this end, the Oxford authors recorded and assessed EMG
responses in patients 1–11. The clinical assessment made indepen-
dently at Stoke Mandeville was then revealed. In seven patients, we
proved to have obtained records from above the lesion and in 10
from below the lesion. Eleven further patients were then recruited
and the clinical evidence on the injury level was revealed before the
experiment to ensure that we recorded below the lesion.

Results
Responses to vestibular stimulation recorded from the erectors spinae
Control subjects
In control subjects, the responses recorded in the erectors spinae
depend upon the polarity of the vestibular stimulating electrodes, head position and task (Ali et al., 2003). When the
head is turned to left or right and the anode is placed on the
posterior mastoid process, an ML excitatory response (latency 60–80 ms) is recorded bilaterally in the erectors
spinae in both sitting (Fig. 1A) and standing subjects. The ML
response is sometimes preceded by an inhibitory response,
but with the rather weak stimuli (~2 mA) chosen for these
experiments the short latency responses are small and have
been ignored. Stimulation causes a backward sway of the
trunk in the sagittal plane. Changing the direction of head
rotation, or electrode polarity, produces inhibitory ML
responses and the subject sways forward. When the head
faces forward, vestibular stimulation causes the trunk to sway
in the coronal plane towards the anode side. An excitatory
ML response is recorded in the erector spinae on the anode
side, and an inhibitory response is recorded on the cathode
side. These patterns of response were observed in all of 18
control subjects. Because responses are largest in the head
turned, sagittal sway condition, we used this when response
amplitudes were measured (excepting responses on the left
side of patients 2 and 18, and the right side of patient 15
which were measured in the coronal condition where the time
of ML response onset was clearest: Table 1).

Patients: recording above the clinically
determined lesion
We recorded above the lesion on both sides of each of seven
patients. With one exception (patient 10 described below),
responses could be observed that showed the same depen-
dence on electrode polarity and head position as did the ML
responses in the controls, though in some patients the
restricted ability to rotate the head meant that the erector
spinae was always activated most effectively on the anode
side. These responses have therefore been classified as ML
responses, even though their latencies were often longer than
in controls (see below).

Patients: recording below the clinically
determined lesion
We made recordings below the lesion in 21 patients. In eight
patients, no responses were detected below the lesion
(Fig. 1C). In three patients, a response was detected on one
side only, and a fourth patient had predominantly unilateral
responses (Fig. 2). The other patients had bilateral responses.
With the exception of patient 10, the responses had the same
features as control subject ML responses.

An unusual response pattern
In patient 10, excitatory responses were recorded on the
anode side with the head facing forward, where control
subjects also showed excitatory ML responses. However, the
responses on the cathode side were also excitatory, whereas in
control subjects and the other patients the ML responses were
inhibitory. Because of the uncertain status of these responses,
subsequent analyses have been performed both with and
without data from patient 10.

Relationship between response amplitude
recorded below the lesion and clinical (ASIA)
grade
The peak amplitude of the response was measured on each
side below the lesion in 21 patients and the responses from the
two sides were summed. Where we classified the response as
absent, an amplitude of zero was entered. The summed
amplitudes were then ranked. The ASIA grade (A–D) was
converted to an ordinal numerical scale (1–4) and the patients
were ranked. The Spearman rank correlation coefficient
(Siegel, 1956) was calculated. The coefficient was positive:
the smallest amplitudes were found in patients with the most
severe impairment. The values of the correlation coefficient
$r_s = 0.442$ and 0.436 (patient 10 included and excluded
respectively) are significant: $P = 0.05$, two-tailed (Neave,
1978). However, exceptions were seen: bilateral responses
were recorded in patient 12 who was ASIA grade A complete,
and no responses were recorded in patient 13 who was ASIA
grade D.
Response amplitudes measured under the same conditions at L3 in five control subjects (ranked for this purpose as ASIA grade E normal, ordinal scale 5) were then added to the data set. The values of $r_s = 0.585$ and 0.605 (patient 10 included and excluded respectively) are significant ($P = 0.002$, two-tailed).

Vestibular-evoked responses are larger on average in female subjects than males (Welgampola and Colebatch, 2001). In order to correct for any bias introduced by this factor, the amplitudes in female subjects were scaled down by their published average ratio of male to female amplitudes. The rank correlations were unchanged.

There was no significant correlation between response amplitude and level of lesion.

**Relationship between response latency recorded below the lesion and clinical (ASIA) grade**

Response latency was measured at the most caudal recording position below the lesion on each side of nine patients. The

---

**Fig. 1** EMG recordings made bilaterally from the erector spinae in sitting subjects. The head was turned to the right and the stimulating anode was placed on the right (posterior) mastoid process. Galvanic vestibular stimulation was applied at time 0 and lasted for the duration of the recording. This activates the left and right erector spinae with a latency of ~60 ms (at L3) and induces backward sway in the sagittal plane in control subjects (A). A baseline has been drawn through the data recorded during the period of 0.1 s preceding the stimulus. A vertical line has been drawn at time 0 to indicate the stimulus onset. Excitatory responses to vestibular stimulation are present bilaterally close to the lesion level in patient 3 (motor level T7; ASIA grade A) at a longer latency of ~200 ms, preceded by small inhibitory responses (B). No responses are present below the lesion in patient 3 (C). The signal recorded at the more rostral position is still visible in the recordings from below the lesion, but at a much reduced amplitude, consistent with recording at a distance (volume conduction).
values for the left and right sides were averaged and ranked. The patients with purely unilateral responses were ranked as the next longest latency, and the eight patients with no responses below the lesion were ranked at equal longest latency. The Spearman rank correlation coefficient between ASIA grade and response latency was calculated. The

Fig. 2 EMG recordings made bilaterally from the erectors spinae above and below the lesion and from left and right soleus in patient 1 (motor level T12; ASIA grade D), while standing. The head was turned to the side with the anode placed over the posterior mastoid process (A). Excitatory responses to vestibular stimulation are present on the right side at latencies of ~70, 90 and 150 ms at T4, L1 and soleus, respectively (A–D). On the left side, no response was seen in soleus (D). No response was found in the left erector spinae at L1 when the head was turned to the right with the anode on the right (B), but a small response was present at L1 when the head was turned to the left with the anode on the left (C). The excitatory responses are preceded by very variable short latency inhibitory responses.
coefficient was negative: the longest latencies were found in patients with the most severe impairment. The values of $r_s = -0.57$ and $-0.58$ (patient 10 included and excluded, respectively) are significant ($P < 0.01$, two-tailed).

There was no significant correlation between response latency and level of lesion.

**Baseline EMG levels**

Although the posture of the subjects was adjusted to produce a background EMG of $\approx 10 \mu$V, there was nevertheless some variation. However, an analysis using the general linear model (Iles et al., 2000; Grafen and Hails, 2002) confirmed that ASIA grade was a powerful predictor of both ML response amplitude and latency ($P < 0.002$), whereas baseline EMG level was not ($P > 0.16$).

**Relationship between response amplitude and latency**

Response amplitude and latency were significantly negatively correlated ($r_s = -0.72$ and $-0.75$; $P < 0.002$, two-tailed; patient 10 included and excluded, respectively).

**Relationship between response latency and intraspinal conduction path in patients and control subjects**

The intraspinal conduction path of the brainstem motor tracts was approximated as the vertical distance from the inion to the more rostral recording electrode of each pair. Twenty-six measurements of response latency and conduction distance were made below the lesion in 13 patients (the left and right sides were treated as independent). Fourteen measurements were made for recording sites one segment below, at or above the lesion in seven patients. Fifty-four measurements were made on one side only in 18 control subjects (from C6 to L4).

The relationship between latency and conduction distance is illustrated in Fig. 3. The small non-linearities previously described in control subjects (Ali et al., 2003) were ignored and linear regression lines were fitted. The slopes of the regression lines were $0.22$ and $0.097$ ms/mm ($R^2 = 0.22$ and $0.56$), in patients (recording from below the lesion) and controls, respectively. The smaller $R^2$ value for the patient regression presumably reflects the patients’ varied clinical status. The conduction velocities for the spinal pathways activated by vestibular stimulation, calculated as the reciprocals of the slopes, were 4.6 and 10.4 m/s in patient and control groups, respectively. Statistical significance was examined using the general linear model. The model treated latency as the response, clinical status as a categorical predictor and intraspinal conduction distance as a covariate (model formula: latency = status|distance). With three levels of clinical status (patient group recording above the lesion, patient group recording below the lesion and control group), there was a significant interaction of clinical status and distance ($P < 0.0005$), i.e. both clinical status and conduction distance were significant predictors of latency, and the slope of the relationship between latency and distance depends upon clinical status. The data were analysed twice with two levels of clinical status (patient group recording above or below the lesion, versus the control group). Latency was significantly longer in both the patient groups than in the control group ($P < 0.0005$ and $P < 0.037$). There was no significant interaction of clinical status and distance when comparing recordings made above and below the lesion ($P = 0.14$).

**Discussion**

**The nature of the responses recorded in patients with SCI**

We have related the responses recorded in the patients to the vestibular-evoked ML responses that have been described by many authors in control subjects (Ali et al., 2003), despite the fact that the response latencies in patients are longer. Instead of the criterion of latency, we have used the criterion that the responses vary with stimulus polarity and head position in the same way as in control subjects. In some patients, there was reduced head mobility but, in practice, where responses were detected at all, it was possible to establish the normal effects of stimulus polarity and head position and to confirm the asymmetry previously observed in control subjects where erectors spinae muscle responses are largest on the anode side (Ali et al., 2003).

In one patient, the responses to vestibular stimulation were not modulated by stimulus polarity and head position. This
might represent an unusual variant of the control response. Incomplete reversal of responses during reversal of a strong (4 mA) stimulus has been reported in controls on a compliant surface (Welgampola and Colebatch, 2001). Alternatively, reorganization of the brainstem motor systems proximal to the SCI could occur. The interval between injury and experiment (9 weeks) in this patient was the shortest of the group. By analogy, there is some (disputed) evidence for changes within the motor cortical area controlling muscles proximal to a lesion (Topka et al., 1991; Streletz et al., 1995; Brouwer and Hopkins-Rosseel, 1997; Bruehlmeier et al., 1998; Davey et al., 1999).

**ML response amplitude in patients and controls**

The amplitudes of vestibular-evoked responses in the erector spinae at lumbar levels below the lesion in patients were significantly correlated with the clinical motor evaluation (ASIA grade A–D). When data from five control subjects (ranked as ASIA grade E) were included, there was a more significant positive correlation (amplitudes were smallest in patients with the most severe impairment and largest in controls). This observation probably reflects damage to the brainstem motor pathways as they traverse the region of cord injury.

There was some variation in the level of muscle activation in both patients and controls. However, baseline EMG level was a poor predictor of ML response amplitude (or latency). Another difficulty with the analysis was that some patients preferred smaller vestibular stimuli than the other patients and the controls. The average stimulus amplitude in patients was 1.75 mA, compared with 2 mA in controls. We reanalysed the data with response amplitudes all normalized to the same stimulus amplitude, using the linear relationship between response and stimulus which has been described for lower limb EMG responses (Iles and Pisini, 1992) and for sway in the coronal plane (Wardman et al., 2003). This procedure did not alter the conclusions of the analysis or their statistical significance.

Delayed, reduced and altered postural responses to perturbation have been reported in subjects with spinal cord injury (Dietz and Berger, 1984; Seelen et al., 1997, 1998; Kamper et al., 1999; Potten et al., 1999). These observations on posture are consistent with the reduced vestibulospinal actions reported here.

In studies of the corticospinal system, increased thresholds for responses to TMS have been described below the lesion (Cariga et al., 2002). The increased thresholds of responses to TMS and the decreased amplitudes of responses to a fixed strength of vestibular stimulation show here probably both result from impaired transmission through the region of injury of the respective motor systems.

Responses to TMS have been reported several segments below clinically complete lesions (Cariga et al., 2002), though not by all authors (Ertekin et al., 1998). In seven of our patients with clinically complete lesions (ASIA grade A), no vestibular-evoked responses were found below the lesion. In patient 9, responses were detected three segments below the lesion. This could result from multisegmental innervation or long muscle fibres in the erector spinae. In patient 12, responses were detected eight segments below the lesion.

Our patient group was older than the control group. ML responses in soleus show a tendency to increase in amplitude with age (Welgampola and Colebatch, 2002). Therefore, age differences are unlikely to be responsible for the decreased amplitudes of responses in the erector spinae of patients reported here.

**Response latency and conduction velocity**

The latencies of responses recorded below the lesion in the patient group were correlated with the clinical motor evaluation (ASIA grade). The longest latencies were found in patients with the most severe impairment. One problem with this analysis is that in controls, latencies are prolonged when the vestibular stimulus is weaker than 1.5 mA (Ali et al., 2003), and some patients preferred weaker stimuli. In order to correct for any such effect in the patient sample, we removed patients 4 and 11 from the data set. The conclusions remained unchanged.

Prolonged latencies of responses to TMS in cases of traumatic SCI have been widely reported (Dvorak et al., 1990; Brouwer et al., 1992; Clarke et al., 1994; Bondurant and Haghhighi, 1997; Alexeeva et al., 1998; Davey et al., 1998; Ertekin et al., 1998; Chen, 2000; Smith et al., 2000). The average latency increase at lumbar or lower limb levels in these published TMS experiments is 11.4 ms. This value is very different from the increases of $\geq 50$ ms seen in the current experiments with vestibular stimulation. However, this is what we should expect because the conduction velocity is much smaller in brainstem pathways than in the corticospinal tract (Ali et al., 2003). For the same reason, a larger increase in latency for slower conducting axons has been reported during cooling peripheral nerves (Paintal, 1965).

The difference can be illustrated by a simple calculation. The plots of vestibular response latency against conduction distance illustrated in Fig. 3 indicate conduction velocities of 4.6 and 10.4/ms in brainstem motor pathways of patient and control groups, respectively. If we assume a conduction distance of 650 mm from the brainstem to the lumbar cord, then an average 79 ms difference in latency between patients and controls is predicted. We can assume a conduction velocity of 55/ms for the corticospinal tract in control subjects (Ali et al., 2003) and the same proportional decrease in conduction velocity produced by injury (to 24/ms). If the conduction distance from the primary motor cortex to the lumbar cord is 750 mm, then a 17 ms difference in latencies is predicted. Other things being equal, slower conducting axons will show the largest latency increases.
A reduced conduction velocity in the vestibulospinal tract has been reported recently in cases of multiple sclerosis (Sartucci and Logi, 2002). The data illustrated in Fig. 3 suggest that the conduction velocity in the brainstem motor tracts is decreased both above and below the lesion. The same suggestion has been made for the corticospinal tract (Cariga et al., 2002). This could result from some retrograde action of injury or from a selective loss of large fibres as described in a contusion model in the cat spinal cord (Blight, 1983) and after ischaemic injury in the monkey (Branston et al., 1988). The latter explanation would be supported by the inverse relationship between amplitude and latency found in the present experiments. However, it must be noted that in some experiments using TMS, there is evidence for a delay in transmission at the region of injury and normal conduction velocity below (Calancie et al., 1999).

Using galvanic vestibular stimulation to monitor SCI

The present experiments show that vestibular-evoked EMG responses, similar to the ML responses of control subjects, can be recorded in the erectors spinae in some seated patients with SCI. Physiological measures such as vestibular-evoked response amplitude or latency are correlated with the clinical assessment, even though the clinical tests may depend more on the integrity of the corticospinal motor pathway and sensory pathways. The responses to vestibular stimulation can provide some information about the level and density of lesions. A combination of TMS and vestibular-evoked responses could provide more information about the location of lesions in the coronal plane because the corticospinal and brainstem motor tracts travel in separate parts of the cord. Both techniques could be used in longitudinal studies during the assessment of new therapies.

Acknowledgements


References


Welgampola MS, Colebatch JG. Vestibulospinal reflexes: quantitative effects of sensory feedback and postural task. Exp Brain Res 2001; 139: 345–53.