Electrophysiology of motor pathways for sphincter control in multiple sclerosis

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Abstract

The central and peripheral motor pathways serving striated sphincter muscle function were studied using cortical and lumbar transcutaneous electrical stimulation, pudendal nerve stimulation and sphincter electromyography in 23 patients with multiple sclerosis (MS), and sphincter disturbance, including incontinence of urine or faeces, urinary voiding dysfunction, or constipation. The central motor conduction time was significantly increased in the MS group compared to controls ($p < 0.05$). Damage to both the upper and lower motor neuron pathways can contribute to sphincter disturbance in MS. The latter may be due to coexisting pathology or to involvement of the conus medullaris by MS.

Abnormalities of sphincter function are common, socially disabling features of multiple sclerosis (MS). Bladder dysfunction occurs in up to 80% of cases and may be the presenting complaint. The severity of bladder symptoms has been correlated with the degree of corticospinal tract involvement.¹ Faecal incontinence is a less frequent symptom, probably because constipation is so common in MS. Nonetheless, these symptoms are relatively rarely expressed in neurological consultation. Swash *et al*² found faecal incontinence a problem in patients with MS, and noted that it seemed related, in part, to chronic partial denervation of the striated anorectal sphincter muscles, associated with childbirth induced damage to the pudendal nerves. Colonic motility may also be impaired in MS with slowing of gut transit³ and absent post prandial colonic motor responses.⁴

The afferent and efferent nerve pathways serving micturition and defaecation were shown by Nathan and Smith to lie in the lateral columns throughout the length of the human spinal cord.⁵⁻⁷ Autoradiographic tracing techniques have delineated motor pathways in primates⁸ extending from the motor cortex to synapse with neurons in the Onuf's nucleus⁹ in the anterior horn of the sacral spinal cord segments, which innervate the striated sphincter muscles. That the central motor pathways to the sphincteric sacral motor neurons in humans conduct at velocities comparable to other corticospinal tract fibres has been shown using transcortical and spinal electrical stimulation.¹⁰

Slowing of conduction in these spinal pathways can also be detected in specific disease states associated with sphincter disturbance, such as multiple sclerosis and radiation myelopathy.¹¹ The lower motor neuron pathway to the striated sphincter musculature has been extensively studied, in incontinent patients, using translumbar electrical stimulation,¹² pudendal nerve stimulation¹³ and striated sphincter electromyography.^{14,15} Both urinary and faecal incontinence have been correlated with damage to these sphincter lower motor neuron pathways, at proximal and distal sites, using these electrophysiological techniques.

The neural lesions underlying sphincter disturbance in MS are incompletely understood, and difficult to define clinically. Yet such information is important for the design of treatment and in assessing its effects. We have addressed this problem in an electrophysiological investigation of the central and peripheral motor nerve pathways to the striated sphincter musculature in a group of 23 patients with multiple sclerosis and sphincter disturbance.

Patients

Twenty three patients with clinically definite MS¹⁶ and symptoms of sphincter dysfunction were recruited from the neurology service of The London Hospital, and through the outpatient practice of The Sir Alan Park's Physiology Unit, St Mark's Hospital, London. A full general and neurological clinical assessment was made in each patient. The clinical data are shown in table 1. Six neurologically normal subjects acted as controls for central motor conduction time evaluation. These six patients had been admitted to hospital for surgical assessment of gastroenterological problems not associated with incontinence. All patients and controls gave their informed consent to the studies, which were approved by the Ethical Committees of both hospitals.

Methods

The electrophysiological studies were designed to assess the integrity of the corticospinal and sacral motor nerve pathways to the striated sphincter musculature. Anal manometry was performed using a station/pull-through technique to quantify resting anal pressure (mainly a function of the smooth muscle internal anal sphincter) and maximum squeeze pressure

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Table 1 Clinical features of MS patients

Case No	Age (yrs)	Sex	Parity	Kurtzke scale	Pyramidal signs (legs)	Saddle sensation	Sphincter disturbance
1	54	F	1	4	+	N	UI intractable constipation
2	62	F	2	5	+	N	Voiding dysfunction, intractable constipation
3	51	F	1	6	+++	ABN	Occasional DI intractable constipation
4	48	F	3	7	+++	N	DI urinary hesitancy
5	45	F	0	7	+++	N	UI mild constipation
6	52	F	4	5	+	ABN	DI urinary hesitancy (lumbar disc prolapse)
7	63	F	1	7	+++	N	UR UI mild constipation
8	32	F	1	5	+	N	DI urinary hesitancy mild constipation
9	30	F	0	8	+++	N	DI
10	61	F	2	2	+	N	Urinary urgency, anal pain
11	49	F	3	8	+++	N	UR constipation
12	37	F	3	5	+	N	DI mild hesitancy and constipation
13	42	F	0	2	+	N	UI mild hesitancy and constipation
14	39	F	3	5	+	ABN	DI mild hesitancy (lumbar disc prolapse)
15	45	M		5	++	N	DI mild hesitancy
16	46	M		5	+	N	DI mild hesitancy (lumbar canal stenosis)
17	20	M		2	+	N	Urinary voiding dysfunction mild constipation
18	26	M		5	+	ABN	DI mild hesitancy (diabetes mellitus)
19	26	M		7	+++	N	FI ileal conduit for UR/UI
20	36	M		5	++	N	Urinary urgency
21	33	M		5	0	ABN	DI mild constipation
22	39	M		7	+++	ABN	Intractable constipation
23	47	M		8	+++	ABN	DI intractable constipation

Kurtzke Disability Scale.³¹

Pyramidal signs in lower limbs: + mild tone and/or reflex changes without weakness; ++ spastic paraparesis causing significant gait disturbance; +++ disabling weakness and spasticity; wheelchair dependent.

Saddle sensation: N—normal; ABN—abnormal.

UI—urinary incontinence; FI—faecal incontinence; DI—double incontinence; UR—urinary retention. Other relevant diagnoses are shown in brackets.

(reflecting voluntary activity in the striated external anal sphincter muscle).^{17,18}

CORTICAL STIMULATION

Following the technique described by Merton and Morton,¹⁰ the motor cortex was excited transcutaneously using a Digitimer D180 electrical stimulator, which provides single capacitor discharges up to a maximum output of 750V and decaying over 50µs. Two 1 cm diameter, saline-soaked pad electrodes were placed on the scalp in the median sagittal plane with the anode over the vertex and the cathode 5 cm anteriorly. The earth electrode was strapped to the patient's upper thigh. The cortex was stimulated with the patient lying in the flexed left-lateral position, and the motor response facilitated by asking the patient to weakly contract the muscle under study. Evoked motor potentials were recorded from the urinary striated sphincter muscle (USSM) by a catheter-mounted bipolar ring electrode (Dantec 21L11). Simultaneously, recordings were made from the puborectalis (PR) or external anal sphincter (EAS) muscles using glove or anal plug mounted surface bipolar electrodes, respectively. Stimulus intensity was adjusted to ensure supramaximal stimulation and the shortest, reproducible latency measured. Urinary catheterisation was not carried out in patients without urinary symptoms or where there was existing urinary infection. A past history of epilepsy was considered a contraindication to electrical cortical stimulation.

TRANSLUMBAR CAUDA EQUINA STIMULATION

Using the stimulating and recording apparatus described above, transcutaneous spinal stimulation was performed with the anode at the L1 lumbar level and the cathode 5 cm cranially in the midline. The shortest L1 spinal motor latencies to the striated sphincter muscles were recorded following supramaximal stimulation.

CENTRAL MOTOR CONDUCTION TIME (CMCT)

This was calculated by subtracting the L1 spinal latency from the cortical latency to the striated sphincter muscles.

PUDENDAL NERVE STIMULATION

The pudendal nerves in the pelvis were stimulated transrectally at the level of the sciatic notch using a glove mounted electrode array, (St Mark's Pudendal Electrode: Dantec 13L40), following the technique described previously.¹³ By recording the evoked motor response in the EAS or USS muscle, the distal motor latency was calculated for each pudendal (PNTML) and perineal (PerNTML) nerve, respectively.

SINGLE FIBRE ELECTROMYOGRAPHY (SFEMG)

The fibre density (FD) was calculated as the mean number of components in 20 consecutive motor unit potentials recorded using a single fibre needle electrode (Dantec 13K87) to sample both sides of the EAS muscle ring. Standard single fibre EMG filter settings were used.¹⁴

CONTROL DATA

Cortical and L1 spinal stimulation was performed, using the same procedures, in six neurologically normal patients, recording the surface evoked potential in the EAS muscle. The normal values for the other study parameters have previously been obtained in our laboratory, and these are given in table 4.¹⁸

STATISTICAL METHODS

The CMCT results in the patients and in control subjects were compared using Student's *t* test. Results are expressed as mean, standard deviation.

Results

The clinical features of the 23 patients are

Table 2 MS patients: electrophysiological results

Case No	Squeeze	PN TML	PerN TML	FD EAS	Spinal latency (ms)			Cortical latency (ms)		
					L1-EAS	L1-PR	L1-USSM	Cortex—EAS	Cortex—PR	Cortex—USSM
1	N	N	ABN	—	—	4.2	5.0	—	NR	31.5
2	ABN	N	—	1.5	—	6.4	—	—	31.2	—
3	N	N	N	1.7	—	6.8	—	—	NR	NR
4	ABN	N	N	2.6	NR	NR	NR	NR	NR	NR
5	ABN	N	N	3.0	—	9.0	7.7	—	NR	NR
6	ABN	N	ABN	—	—	4.2	5.5	—	NR	17.6
7	ABN	N	N	2.7	—	—	—	—	—	—
8	N	ABN	NR	—	—	6.5	6.0	—	32.5	NR
9	ABN	N	N	1.7	—	6.0	5.4	—	NR	NR
10	N	N	—	2.0	—	6.7	—	—	NR	—
11	ABN	—	—	2.7	4.9	—	5.3	NR	—	32.0
12	N	ABN	ABN	2.3	5.6	—	4.4	28.8	—	28.8
13	N	N	N	1.9	4.8	—	5.0	30.8	—	32.8
14	N	ABN	N	2.0	4.4	—	5.7	20.4	—	20.0
15	N	N	N	—	NR	—	NR	NR	—	NR
16	N	N	N	1.6	—	4.9	5.4	—	NR	29.4
17	N	—	—	—	3.9	—	—	50.4	—	—
18	N	N	N	1.8	—	5.8	5.3	—	34.8	NR
19	ABN	N	—	1.5	—	4.9	—	—	—	—
20	ABN	N	—	1.7	6.0	—	—	NR	—	—
21	N	N	N	2.0	3.6	—	4.4	—	—	17.8
22	ABN	N	—	—	5.4	—	—	34.0	—	—
23	ABN	N	N	1.8	5.2	—	6.0	NR	—	NR

Squeeze—voluntary anal squeeze pressure during anal manometry. N—normal; ABN—abnormal ($> \text{mean} + 2 \text{ SD}$); (—)—not tested; NR—no response. Other abbreviations, see text.

summarised in table 1 and the electrophysiological results appear in tables 2 and 3. Sphincter symptoms were not confined to the more severely disabled patients, but spread fairly evenly throughout the group.

Figure 1 Cortical latency to EAS muscle in normal subject.

Figure 2 Normal cortical latency to USSM in MS patient.

Figure 3 Abnormal cortical latency to EAS in MS patient.

ELECTROPHYSIOLOGICAL RESULTS

The CMCT to the striated anal sphincter muscle in control subjects was 15.3 ± 3.5 ms. All patients in the control group had recordable cortical latencies. CMCT to the sphincter sacral cord segments was determined in 13 MS patients and no response could be recorded from any of the sphincter muscles in a further eight patients. In the MS patient group, the mean CMCT for the striated anal sphincter muscles (EAS + PR) was 27.5 ms and for USSM 21.2 ms. The mean (SD) of the shortest CMCT value measured to any of the striated sphincter muscles in individual MS patients was $24.7 (8.4)$ ms ($p < 0.05$ compared with control values). Cortical stimulation was not performed in two cases. Eleven of 13 patients in whom an evoked sphincter muscle response was present were still independently ambulant, compared to 0/8 of those where the response was unobtainable. The severity of corticospinal tract involvement as assessed by

Table 3 Control patients: central motor conduction time

Case	Age	Sex	Spinal latency L1-EAS (ms)	Cortical latency Cortex-EAS (ms)	CMCT-EAS (ms)
1	37	F	5.2	26.4	21.2
2	33	F	4.8	18.2	13.4
3	20	M	4.3	19.8	15.5
4	20	M	4.4	18.4	14.0
5	43	M	4.5	22.2	17.7
6	35	M	5.1	15.2	10.1

Mean (SD) $15.3 (3.5)$
CMCT—central motor conduction time (ms).

Table 4 Electrophysiological tests: normal data¹⁸

SFEMG	1.32 (0.1) (< 30 years)*
	1.5 (0.16) (< 65 years)
PNTML (ms)	2.0 (0.2)*
PerNTML (ms)	2.4 (0.2)*
L1-EAS (ms)	5.5 (0.4)*
L1-PR (ms)	4.8 (0.4)*
L1-USSM (ms)	4.8 (0.3)*

*Mean (SD).

upper motor neuron signs in the lower limbs did not correlate well with the values obtained for CMCT to the sphincter muscles.

Sixteen (3 male, 13 female) patients had evidence of damage to the lower motor neuron pathways to the striated sphincter musculature, including the three patients with normal CMCT values: raised fibre density in EAS—10 patients (two male, eight female), increased L1 spinal latency—10 patients (two male, eight female), increased PNTML—three parous women, increased PerNTML—three parous women. Seven patients showed more than one abnormality. Possible reasons for these abnormalities other than MS were present in 12 cases (parity 10, lumbar intervertebral disc disease three, diabetes mellitus one).

CORRELATION BETWEEN ELECTROPHYSIOLOGICAL FINDINGS AND PATTERN OF SPHINCTER DISTURBANCE

1 Urinary incontinence

Seventeen patients had urinary urgency and incontinence (11 female, 6 male). Eight were parous women and all had some evidence of damage to the sphincter muscle lower motor neuron innervation, including five with slowing of distal motor conduction in the pudendal nerve or its perineal branch. Three nulliparous women and six men also suffered from urinary incontinence. None of these patients showed any distal pudendal nerve motor conduction slowing although the fibre density in the EAS muscle was elevated in 4/8. Fifteen of the 17 patients with urinary incontinence had cortical and spinal stimulation and CMCT was abnormal in 12. Three patients had CMCT to the striated sphincter muscles within the normal range: two parous women, and one male whose initial clinical presentation was consistent with a lesion of the conus medullaris.

2 Urinary voiding dysfunction

Fourteen patients complained of difficulty with voluntary bladder emptying. Nine (three male, six female) had mild and intermittent hesitancy of micturition associated with more prominent symptoms of urinary urgency and incontinence. Evidence of lower motor neuron damage was present in all six of these women (five parous) and one man. CMCT studies were abnormal in seven (five delayed, two absent).

Five patients had more significant urinary retention, requiring catheterisation in two cases and an ileal conduit in one. Two were independently ambulant and three wheelchair bound. They described less incontinence than those with occasional hesitancy, and tended to show less electrophysiological evidence of damage to the lower motor neuron pathway. Two parous women had a raised fibre density in the EAS muscle. CMCT was increased to one or both sphincter muscles in all three patients studied.

3 Faecal incontinence

Thirteen patients had recurring episodes of faecal incontinence (six male, seven female—one nulliparous). All had normal resting anal tone

on manometry. Five had very poor or absent voluntary anal sphincter contraction (two male, three female). CMCT studies recording from the striated anal sphincter muscles were abnormal in 10 cases (delayed three, absent seven). Eight patients (three male, five female) had evidence of damage to lower motor neuron pathways to the striated anal sphincter muscles in addition to abnormal central motor conduction.

4 Constipation

Thirteen patients had experienced a change in bowel habit since the onset of MS. Eight patients had a mild and variable tendency to constipation which was responsive to dietary measures or occasional oral laxative medication. None of these patients described chronic straining at stool. As a group they tended to have more evidence of lower motor neuron pathway damage to the striated anal sphincter muscles (raised fibre density in EAS—6/6, increased PNTML—2/7, and increased spinal latency to EAS or PR—2/7) than those with severe constipation. CMCT was increased in five, absent in one and normal in one case.

The five other cases (two male, three parous women) had intractable constipation, usually accompanied by tenesmus, chronic straining and abdominal bloating. Three patients had been admitted to hospital because of obstructive bowel symptoms. Two were freely ambulant, two had a marked spastic paraparesis and could walk only a few steps with assistance and one was wheelchair dependent. Voluntary anal squeeze pressure was reduced in three cases, and all showed paradoxical increase in striated sphincter muscle activity instead of the normal inhibition when asked to perform a defaecation strain.¹⁹

This disorganisation of the normal pattern of muscle activity during defaecation straining was confirmed in three cases by defaecating proctography.²⁰ Paradoxical upward movement of the pelvic floor was seen, consistent with muscle contraction, and poor opening of the anal canal obstructed the expulsion of barium from the rectum. Four of these five patients described alteration of bladder or anorectal sensation, and three had subjective alteration of pinprick sensation in the saddle area. All five had abnormal or absent cortical latencies to the sphincter muscles. Pudendal latencies were normal in all five patients.

Discussion

The pathways for neural control of the bladder and bowel sphincter mechanisms traverse the whole neuraxis, and are therefore frequently involved in a multifocal condition such as MS. Our results show that conduction in the upper motor neuron pathways to the Onuf's nucleus is usually, but not always, slowed in patients with MS and sphincter disturbance. CMCT measurements were not specific for the pattern of sphincter disturbance with incontinence versus voiding dysfunction. Since the electrode placement used for transcortical stimulation probably excite the parasagittal motor cortex

bilaterally, and the recording electrodes are arranged bilaterally as catheter-mounted ring electrodes, or anal plug electrodes, the latency of the motor response in the striated sphincter muscle will reflect conduction in the fastest of the preserved corticospinal fibres from both hemispheres. Therefore, unilateral or lesser degrees of damage to these pathways will not easily be detected by this technique. It seems likely that our patients with sphincter symptoms and abnormal cortical motor latencies to the sphincter muscles, had bilateral lesions affecting these pathways, though not necessarily at the same level on each side.

Although the CMCT values to the sphincteric sacral segments in the patient group are significantly greater than in the controls, most abnormal values did not show the two to three fold increase which has been reported for other skeletal muscles studied in MS patients.²¹⁻²³ This and the number of unrecordable responses in our series probably reflects the greater difficulty in recording low amplitude, dispersed potentials from the relatively small striated sphincter muscles, especially in the presence of lower motor pathway dysfunction and denervation atrophy. It also suggests that sphincter motor control may be significantly disrupted by relatively minor changes in motor pathway conduction. When repeated stimulation is required to establish the latency of small, dispersed motor potentials, transcutaneous magnetic stimulation may be better tolerated by patients.²³ However, electrical excitation allows greater localisation of the stimulus which is important for the interpretation of spinal stimulation results.

Translumbal spinal stimulation at the level of the conus medullaris probably excites the proximal motor axons rather than the nerve cell bodies, but the precise location is unresolved. Maertens de Noordhout *et al*²⁴ localised the point of activation to within 3.5 cm of the cell body but commented that it may be as proximal as the axon hillock. The peripheral component in the CMCT is therefore likely to be small.

Lower motor neuron abnormalities were frequently present in sphincter pathways. Most commonly this was found in association with parity, as was noted by Swash *et al*.² Only parous women patients with MS had increased terminal motor latencies in the pudendal nerve or its perineal branch. Although theoretically, any lower motor neuron lesion causing significant Wallerian degeneration within the pudendal nerve could result in an increased terminal motor latency, injury to these nerves during childbirth is the most likely pathogenesis of these abnormalities. Although six out of eight parous MS patients tested had a raised fibre density in the EAS muscle on single fibre EMG, this abnormality was also found in one male and two nulliparous female patients with no known coexisting lower motor neuron pathology. Involvement of the conus medullaris by MS, with secondary axonal atrophy in the sphincter motor neurons may best account for these changes in these three patients, and could contribute to the lower motor neuron disturbance found in the others.

From the example of patients with complete spinal cord transection, it is clear that suprasacral control centres are important for the voluntary initiation and deferment of micturition and defaecation. Without this input, the anorectum and bladder tend to function automatically, emptying reflexly in response to filling or to cutaneous stimuli applied within the sacral dermatomes. Many patients with spinal cord disease have difficulty establishing even this reflex bladder emptying because of vesico-urethral dysfunction, especially detrusor-sphincter dyssynergia.²⁵ This suggests failure of integration between the visceral and somatic motor systems once the sacral cord is isolated from higher control centres. In the same way, striated pelvic floor muscle dysfunction can contribute to constipation in MS patients by causing a functional obstruction at the pelvic outlet. The five patients in our series with intractable constipation all showed a paradoxical increase in striated sphincter muscle activity on attempted defaecation straining. Paradoxical striated pelvic floor muscle contraction indicates a disturbance of the normal pattern of recruitment and inhibition of striated muscle during the voluntary motor task of defaecation straining.²⁶ It is uncertain whether this represents dysfunction in the descending pyramidal or non-pyramidal influences on segmental spinal activity. Patients with severe urinary and faecal voiding dysfunction had, in general, less evidence of disruption to lower motor neuron innervation of sphincter muscles than patients with mild voiding disorders associated with incontinence. However, without the use of urodynamic studies, it is not possible to determine the relative contributions of detrusor hyperreflexia and sphincter muscle weakness to the pathogenesis of urinary incontinence in each case. It is presumed that patients with paradoxical anal sphincter muscle activity during defaecation straining are more likely to achieve some degree of evacuation when the sphincter muscle is weakened by partial denervation. Indeed sphincterotomy is the usual surgical management of idiopathic voiding disorders.²⁷

These electrophysiological investigations allow the identification of upper and lower motor neuron components to the sphincter disturbance in MS. Increased CMCT values to the striated sphincter muscles can signal the presence of an upper motor neuron abnormality where MS coexists with other pathologies such as diabetes mellitus or lumbar intervertebral disc prolapse (Cases 18 and 16). Lower motor neuron dysfunction may also be due to involvement of the conus medullaris by MS,²⁸ and our results reaffirm the previously reported contribution of distal pudendal nerve damage to incontinence in parous women with MS.² Pelvic floor surgery can ameliorate incontinence due to weakened, partially denervated pelvic sphincter muscles²⁹ and may be appropriate in some incontinent patients with MS. However, coexisting voiding or defaecatory dysfunction may worsen if the pelvic floor mechanism is made functionally more competent in this way.

Although we have demonstrated motor pathway abnormalities in all our patients with MS and sphincter disturbance, other neuronal systems with relevance to sphincter control may also be affected. We know from animal studies that the sacral parasympathetic neurons and the cells of Onuf's nucleus receive dual innervation from higher autonomic and somatic neural centres which may be an anatomical substrate for functional integration.³⁰ Nathan and Smith showed that spinal pathways for both conscious and unconscious vesicourethral and anorectal control lie close together in the spinal cord.⁵⁻⁷ Nevertheless, we cannot directly infer autonomic nervous system involvement from our studies of somatic nerve pathways. Impairment of frontal lobe function and involvement of sensory and extrapyramidal pathways also contribute to the complexity of this problem and may limit the analysis of electrophysiological studies of sphincter symptoms in patients with MS.

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