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Skin potential recordings during cystometry in spinal cord injured patients

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In order to investigate autonomic mechanisms associated with bladder filling and bladder contraction, skin potentials from the hands and the feet of 32 spinal cord injured patients were recorded during cystometry. All had a complete clinical loss of motor and sensory function below the lesion, but in 3 patients, the autonomic lesion was electrophysiologically assessed as incomplete. In patients with a complete autonomic lesion, any rise in intravesical pressure associated with bladder hyperreflexia induced SP responses below the level of the lesion. SP responses were never obtained during bladder filling, as the intravesical pressure remained low. These results tend to confirm those of Guttmann and Whitteridge, but differ in so far as SP responses at the foot were a regular finding in all paraplegic and in most tetraplegic patients. Furthermore, bladder contraction failed to elicit SP responses below the level of the lesion in patients with an incomplete autonomic lesion. This study emphasises the importance of assessing the integrity of the autonomic nervous pathways when dealing with autonomic mechanisms in spinal cord injured patients. The possible relation between SP responses and bladder neck dysfunction is further discussed.

Keywords: skin potentials; spinal cord injury; bladder hyperreflexia; autonomic nervous system; bladder neck dysfunction.

Introduction

Malfunctioning sympathetic mechanisms are seen in most spinal cord injured (SCI) patients who are seriously injured. Inappropriate inactivity occurs during postural change causing orthostatic hypotension while overactivity occurs when spinal sympathetic reflexes are activated, which may result in the syndrome of autonomic dysreflexia.[1] Bladder distension is the most frequent stimulus in the production of this sympathetic overactivity.[2]

Sweating represents an outstanding component in the symptomatology elicited by bladder distension. It was first described by Head and Riddoch in 1917,[3] and later extensively studied by Guttman and Whitteridge, using the quinizarin test.[4] Other techniques were used to monitor the sudomotor activity in SCI patients such as galvanic skin responses during percutaneous electrical stimulation[5] or microneurographic recordings in response to bladder pressure and other stimuli.[6] However, for several reasons, these techniques are not routinely available in a clinical setting.

Skin potentials (SPs) were first described by Tarchanoff in 1890. These electrical potentials are generated by a change in the skin resistance which is attributed to a large extent to sudomotor activity.[7,8] This technique has been well studied in the 1980s, and SPs are now currently used in the assessment of autonomic dysfunction in polyneuropathy or impotence.[9–11] In a former paper, we showed that SP recordings are a reliable and convenient procedure to evaluate the sympathetic manifestations of SCI patients in response to various stimuli.[12]

In order to investigate autonomic mechanisms associated with bladder filling and

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bladder contraction after spinal cord injuries, we recorded SPs from the extremities of 32 SCI patients during cystometry.

Material and methods

Thirty-two patients with chronic spinal cord injury were studied. The clinical data of the participating patients are presented in Table I. All had a complete clinical loss of sensory and motor function below the level of the lesion. The integrity of their autonomic pathways was also analysed: complete or incomplete lesion of the autonomic nervous system was diagnosed when heavy sound or mental arithmetic respectively failed to elicit (29 patients) or elicited (3 paraplegic patients) SP responses below the lesion.[12–14] All but 4 patients were on antispastic drugs (baclofen). Four patients were on anticholinergic drug (oxybutinine hydrochloride) at the time of the assessment.

Urodynamic studies were performed in all 32 patients, with simultaneous SP recordings from hand and foot in all paraplegic and in 13 tetraplegic patients. Recordings were limited to the hand in the remaining 10 tetraplegic patients. Water cystometry with a filling rate of 10 cc/min with physiological sodium chloride at room temperature was performed. The intravesical pressure and the intraurethral pressure at the site of the maximum pressure were measured by a water-filled infused catheter (filling rate of 3 cc/min) which was inserted through the urethra.

Small electrodes were applied to the skin of the palm and sole to record the SPs. Anodes were placed on the dorsum of the hand and the instep of the foot. A ground plate surrounded the waist.

Pressures and SPs were recorded with a Gould apparatus (sweep speed 1 sec/mm). For the SPs, we used a band pass of 1 Hz to 5 kHz and an amplification sensitivity between 100 and 1000 μV/cm.

The sympathetic outflow to the extremities originates from the spinal cord at the upper thoracic segments (T3–T7) for the upper limbs and at the thoracolumbar segments (T10–L2) for the lower limbs (Fig 1).[15] In the following, the term 'SPs below the lesion' refers to SP responses obtained in areas where the sympathetic innervation is mediated by fibres originating below the level of the lesion. This includes hands and feet of tetraplegics, and feet of paraplegics. These areas are deprived from central control when the autonomic lesion is complete. In contrast, 'SPs above the lesion' designates SP responses obtained in areas innervated by sympathetic fibres originating above the level of the lesion, as is the case in hands of paraplegics.

Results

Urodynamic findings

All patients presented detrusor hyperreflexia. The intravesical pressure ranged between 30 and 100 cm H_2O, the cystometric capacity between 50 and 400 cc and the values of the maximum voiding pressure between 40 and 120 cm H_2O. Maximum urethral pressure during bladder filling ranged between 30 and 90 cm H_2O.

Table I Basic data for participating patients

	Number of patients	Upper level of the lesion	Completeness of the lesion	
			sensory-motor	autonomic
Tetraplegics	23	C5–C8/T1	all complete	all complete
Paraplegics	9	T5–T10	all complete	3 incomplete

Mean age of patients (years) = 40 (17–67)
Mean age of lesion (months) = 72 (6–144)
Sex ratio = 5 women, 27 men

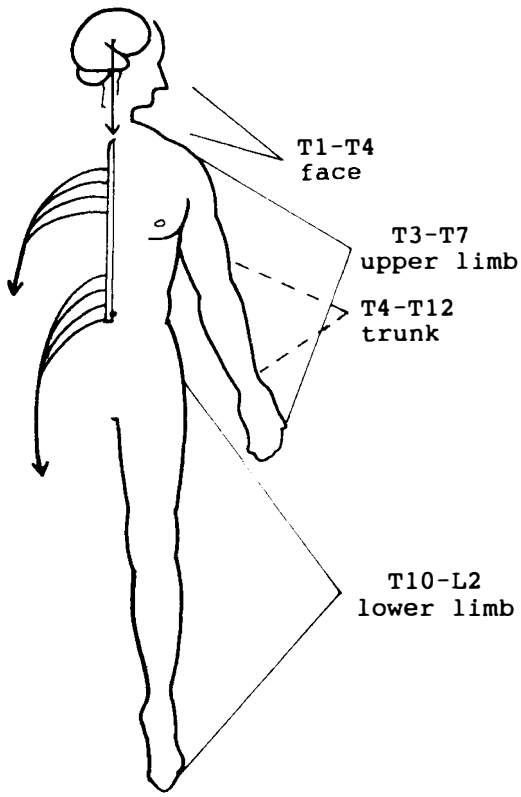

Figure 1 Schematic metameric sympathetic cutaneous innervation of a normal subject.

SPs below the lesion

At rest, we recorded a spontaneous activity in all patients, called SP base level. This consists of continuous slow and irregular undulation of the base line, with an amplitude ranging from 0.1 to 0.3 mV in 27 patients, but higher than 0.5 mV in 5.

During bladder filling, the activity recorded did not differ from the SP base level, as long as the intravesical pressure remained low. Reflex bladder contractions elicited SP responses in all 29 patients with complete autonomic lesion, with amplitudes ranging from 0.5 mV to more than 5 mV. In these patients, SPs were elicited at the onset of the rise of intravesical pressure (Fig 2), during the course of the contraction (Fig 3) or at the peak pressure. When present, SPs could last from a few seconds up to the entire time of the bladder contraction (Figs 2, 3). In the tetraplegic patients, SP responses were constant at hand level whereas SP responses at the foot were found in 10 of the 13. In these cases, the amplitude of the responses was always higher in hands.

In the 3 paraplegic patients with incomplete autonomic lesion, no SP responses were observed during bladder contraction.

SPs above the lesion

In the paraplegic patients not taking any anticholinergic drug, the SP base level recorded at rest was similar to that below the lesion. However, the SP responses were obtained during psychological stimuli such as mental arithmetic or loud noise, and also during cutaneous stimuli such as pain, similar to that of normal subjects. This was not the case in the patients on anticholinergic drug as no SP responses were obtainable regardless of the stimulus used. During bladder filling or bladder contraction, we did not observe any further SP responses in the patients.

Discussion

The sympathetic nerve supply to the extremities originates in cells in the lateral grey column of the spinal cord from the level of C8 to L2. These pathways are controlled by supraspinal descending systems (Fig 1).[15] Two different types of sympathetic outflow have been recognised by microneurographic recordings in man. Muscle nerve sympathetic activity is essentially composed of vasoconstrictor impulses, which are controlled by arterial baroreflexes whereas skin nerve sympathetic activity is composed of vasoconstrictor and sudomotor impulses without cardiac rhythmicity. Sudomotor activity is affected by thermal stimuli, but any unexpected arousal stimulus (such as a sudden sound, touch or pain stimulus . . .) usually evokes reflex sudomotor activity as well.[16,17] These 'encephalic sudomotor reflexes'[15] occur in parallel in hands and feet.[9,18]

A complete spinal cord transection removes the supraspinal control of the sweat glands below the lesion which are not involved in the thermoregulation of these patients. After the state of spinal shock,

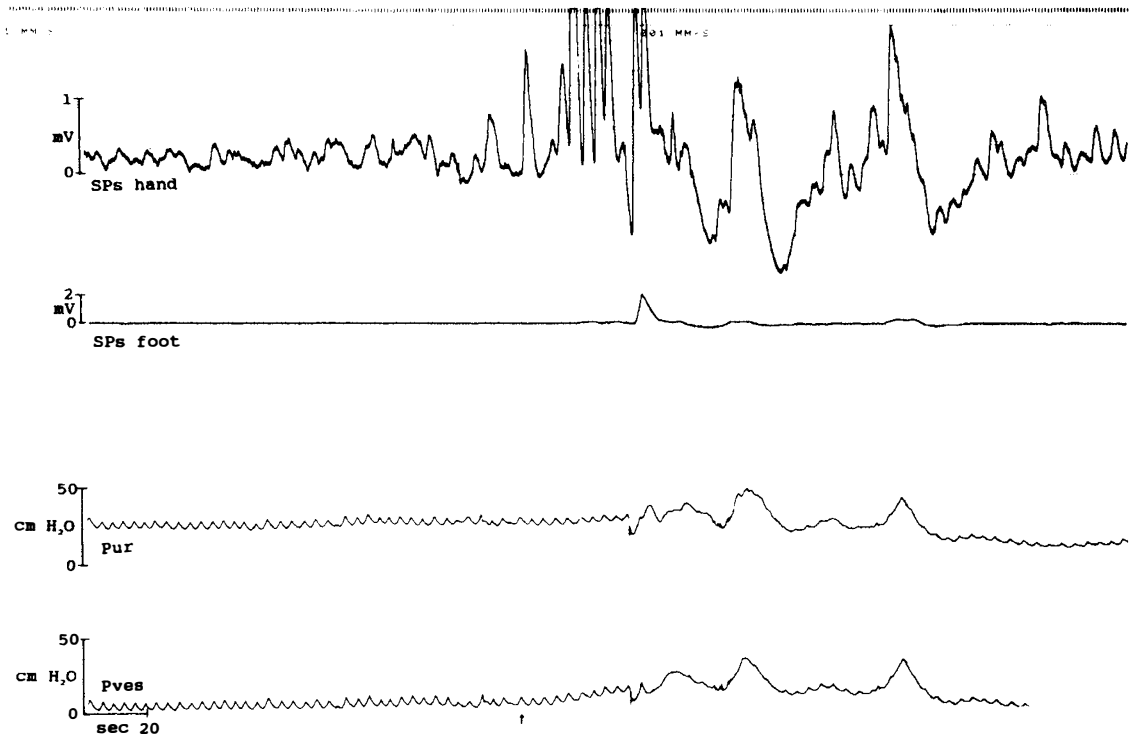

Figure 2 2 SP recordings from the hand and the foot during bladder filling and bladder contraction in one tetraplegic patient (C7) with complete autonomic lesion. Arrow: onset of the rise in intravesical pressure. Pves: intravesical pressure; Pur: urethral pressure.

sympathetic sudomotor reflexes caudal to the spinal cord lesion are still elicitable, though stronger stimuli are needed to elicit these responses:[6] sweating,[3,4,15] electrodermal activity (galvanic skin responses[5] or skin potentials[12,19]), sympathetic sudomotor impulses (microneurographic recordings)[6] in response to bladder distension, electrical shocks or other stimuli below the level of the lesion have been reported. In these cases, it is obvious that the sympathetic activity is mainly of spinal origin.[6,20]

Afferent pelvic nerve activity generated by bladder filling elicits segmental and intersegmental sympathetic neural responses (vesicosympathetic reflexes). Sympathetic outflow can be found in the hypogastric nerve,[21–23] but also in the splanchnic,[24] renal and splenic nerves,[25] and in the upper thoracic neurons.[26] This sympathetic outflow can influence the motility of the bladder as well as the cardiovascular system. Cardiovascular effects include vasoconstriction and rise in blood pressure which are present in normal animals and humans[24,27,28] but can dominate after spinal transection at or above T5, causing autonomic dysreflexia.[1–4,14,24,29–31]

As far as we know, Prout is the only author who dealt with sudomotor effects in relation to bladder distension in normal humans. In 4 subjects, he observed an inconstant depression of the amplitude of the galvanic skin response.[32] We repeated Prout's test on 8 normal subjects, and 2 others underwent a cystometry. During bladder filling, or at maximum bladder distension, we did not observe any SP responses. The SP activity recorded was variable, but not different from that during resting activity. However, during micturition, an extinction of SP activity was observed in all (Fig 4).

Head and Riddoch in 1917,[3] and later

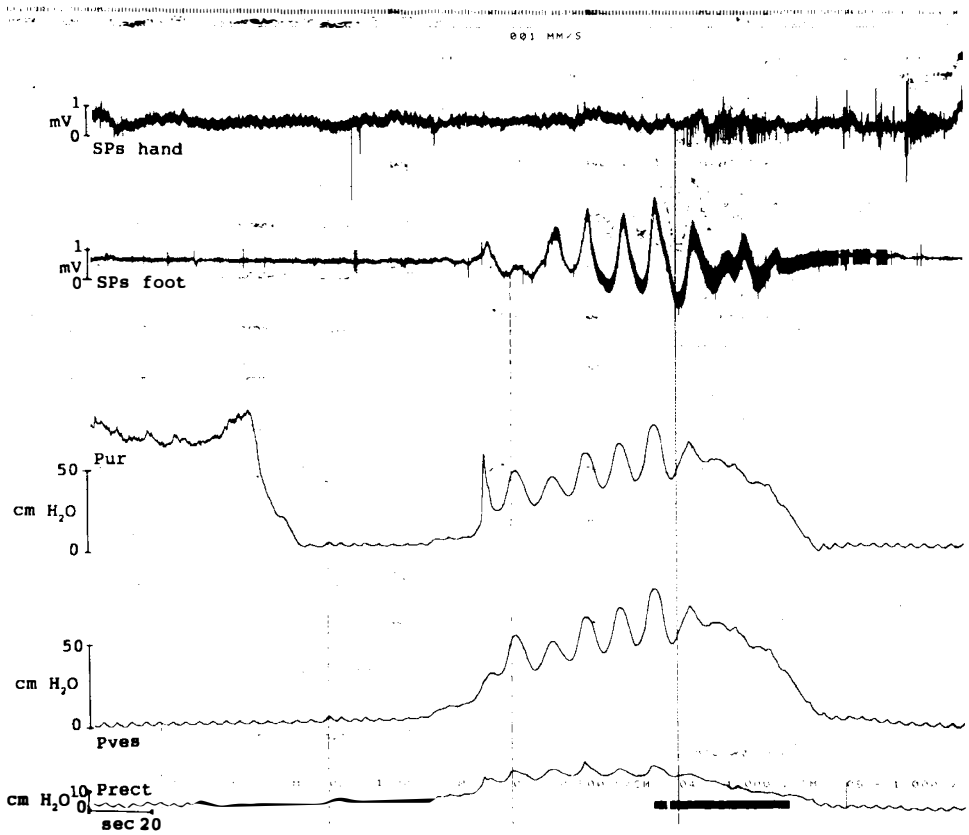

Figure 3 SP recordings from the hand and the foot at the end of bladder filling and during bladder contraction in one paraplegic patient (T8) with complete autonomic lesion. Pves: intravesical pressure; Pur: urethral pressure; Prect: intrarectal pressure.

Guttmann and Whitteridge with the quinizarin method,[4] were able to demonstrate sweating to be one of the prominent effects of bladder distension in SCI patients. In upper thoracic and cervical lesions, sweating is a regular finding, involving face, neck, upper limbs and chest, and to a lesser degree the trunk. The lower limbs remained dry. In patients with midthoracic lesions, sweating response was not found to be a regular component of the autonomic response.[4] In our SCI patients with complete autonomic lesion and not taking any anticholinergic drug, there is a clear relation between SP responses below the lesion and the rise of intravesical pressure. During bladder filling, in the absence of bladder contraction, we were unable to demonstrate any SP responses. The intravesical pressure always remained low (below 15 cm H_2O) in all patients, and we never reached a state of hypertonic bladder, such as described by Guttmann. In contrast, any sharp rise in intravesical pressure (always associated with reflex bladder contraction with or without micturition), elicited regular SP responses below the lesion in all patients. These results complete those of Guttmann and Whitteridge. The prominent role of intravesical pressure in the afferent part of the vesicosympathetic reflex is thus emphasised here.[33]

In the 3 paraplegic patients with incomplete autonomic lesion just as in the 10 normal controls, bladder contraction never succeeded in eliciting SP responses. These results suggest that persistent supraspinal pathways may inhibit the SP responses. On

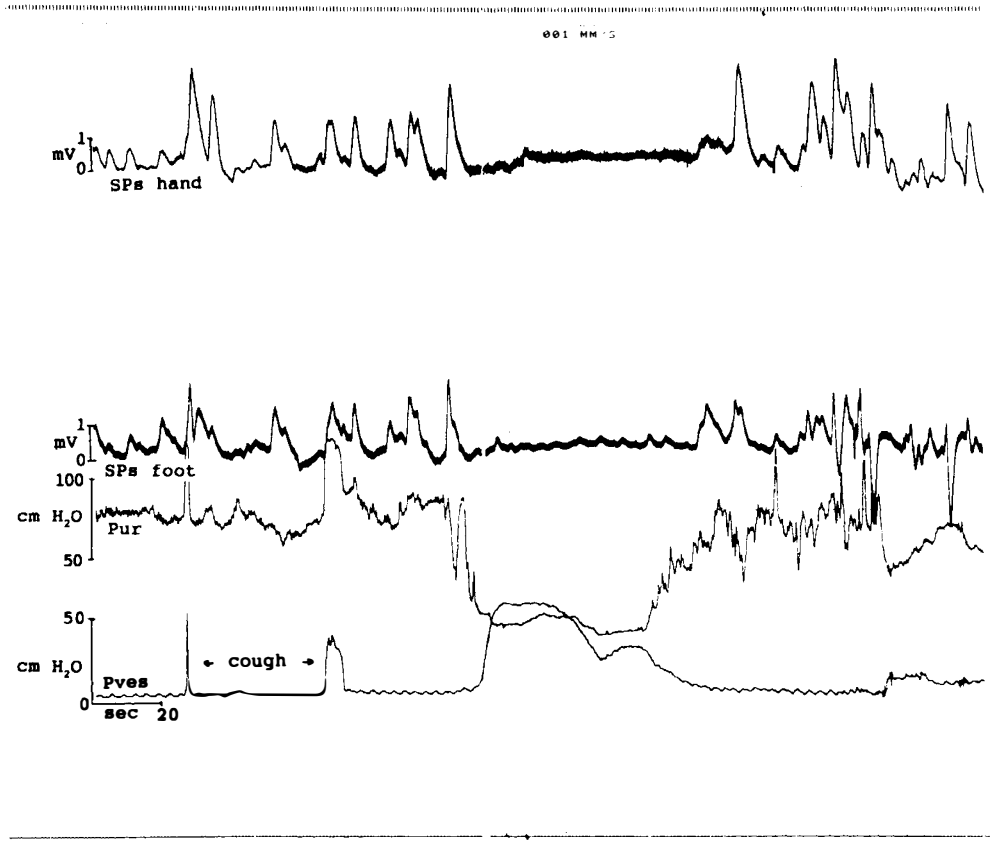

Figure 4 SP recordings from the hand and the foot at the end of bladder filling and during bladder contraction in one normal subject. Pves: intravesical pressure; Pur: urethral pressure.

the other hand the complete destruction of these pathways allows sudomotor overactivity to occur.

Atropine is known to block the cholinergic activation of sweat glands.[10,34] Four paraplegic patients with complete autonomic lesion were on an anticholinergic drug (oxybutinine hydrochloride) prescribed for bladder hyperreflexia at the time of the investigation. However, the efficacy of the medication was limited in 2 patients, as they still presented incontinence between intermittent catheterisation. Interestingly enough, the SP responses below the lesion were still elicitable during bladder contraction in these patients, whereas they were not in the two continent patients. This suggests that persistence of SP responses below the lesion could be an indicator of the limited efficacy of the anticholinergic drug. Further studies in this domain would be needed to verify these observations.

Dealing with tetraplegic patients, it must be remembered that the face is innervated by sympathetic fibres originating well below the level of the lesion (Fig 1). Sweating above the lesion in the tetraplegic does not occur, whatever part of the body is considered (Table II). The same applies to patients with a high thoracic lesion.[4] In patients with midthoracic lesions, there is a general agreement that sweating below the lesion in response to bladder distension is the main finding (Table II).[3,4,15] However there have been reports of sweating above the lesion as well.[3,4] In such cases, sweating

Table II Respective localisation of encephalic and spinal reflex sweating in SCI patients according to the level of the lesion

	Encephalic reflexes	Spinal reflexes	
	André-Thomas[15]	André-Thomas[15]	Guttmann[4]
Cervical	Absent	No observation	On the head, neck, thorax, upper limbs, trunk to T11 but never on the lower limbs
T1–T2	Weak at head and neck	On head and neck, especially on trunk and limbs	
T3–T4	Normal at head and neck, weak at upper limbs and trunk to T7	On lower limbs, trunk up to T3 and upper limbs	
T6–T7	Normal at head, neck, upper limbs and trunk to T10	On lower limbs, trunk up to T8	Inconstant on lower limbs and trunk
T8–T9	Normal down to the iliac crest	On lower limbs up to the iliac crest	
T10–T11	Normal for the whole body	On lower limbs	No reflex sweating

can be explained as part of the thermoregulatory response mediated by efferent fibres arising from above the spinal cord lesion.[4]

Contrary to Guttmann's study, we observed SP foot responses in all paraplegic and in most tetraplegic patients as well. The differences in experimental procedure may explain this discrepancy. The quinizarin test used by Guttmann measures sweating only, whereas SP responses are elicited by the activation of sweat glands but probably by nonsudorific mechanisms as well.[7,8,35] Some authors have pointed out the poor correlation observed between the amplitude of the SPs and sweat response,[35] and the lack of a linear relationship between SPs and sweat output.[36]

Prominent SP responses of the hands of tetraplegics were already observed by Fuhrer (with electrical stimulation)[5] and by Guttmann (with bladder distension).[4] This may be explained by higher regional concentration of sweat glands.[5] It may also depend on the distance of the spinal lesion from the cervicothoracic junction.[4]

Spontaneous electrodermal activity in SCI patients has been described by Fuhrer.[5] However, microneurographic recordings showed that the sudomotor activity is sparse in the SCI patients.[6] In our patients, this activity could eventually be explained by irritation in relation with the urethral catheter.

de Groat *et al.* have stressed the importance of the autonomic system in the regulation of the vesical function. During continence, the vesicosympathetic reflexes allow the bladder to accommodate the larger volumes. During micturition these reflexes are probably depressed by supraspinal controls, thereby allowing the bladder to empty completely.[37,38] In SCI patients, persistence of sympathetic outflow during bladder contraction, causing inappropriate bladder neck contraction, could be a major factor in the etiology of bladder neck dysfunction.[1,38,39] If spinal sympathetic reflexes of SCI patients are 'true mass responses', that is, if they also extend to visceral sympathetic nerves,[20] then a possible positive relation between the presence and importance of the SP responses and bladder neck dysfunction could be hypothesised. We are verifying this with further studies.

In conclusion, SP recordings during cystometry in SCI patients allow monitoring of sympathetic reflexes during bladder filling and bladder contraction. The extent of the autonomic lesion is an important factor to be taken in account as different patterns of

SP responses are found. Our results suggest that SP recordings could be used to follow the efficacy of an anticholinergic treatment. We also stress the possible positive relation between SP recordings and bladder neck dysfunction.

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