

Nocturnal penile tumescence and rigidity (NPTR) findings in spinal cord injured men with erectile dysfunction

DM Schmid^{1,2*}, D Hauri² and B Schurch¹

¹Department of Neurourology, Spinal Cord Injury Centre, Balgrist University Hospital, Zurich, Switzerland; and

²Department of Urology, University Hospital Zurich, Zurich, Switzerland

This prospective study aimed at determining whether nocturnal penile tumescence and rigidity (NPTR) findings correlate to the neurologic disorders in spinal cord injured (SCI) patients suffering from erectile dysfunction (ED). A total of 25 acute SCI male patients with post-traumatic ED underwent neurological, electrophysiological and urodynamic examinations, respectively, as well as NPTR recordings. The mean value for rigidity (R), tumescence (T) and duration (D) during NPTR tests were 83.3%, 3.3 cm, 6.4 min in patients with a complete lesion above the sacral (S2–S4) spinal cord ($n=10$), 46.1%, 1.6 cm, 5.5 min in patients with a complete lesion involving the sacral metamer (S5) ($n=5$) and 89.8%, 3.8 cm, 29 min in patients with an incomplete suprasacral lesion ($n=7$). The differences among these groups were statistically significant ($P<0.05$). Patients with lesions involving both sacral and thoracolumbar spinal cord showed no erections ($n=3$). We found four NPTR patterns: (1) normal R and T, short D; (2) weak R and T, short D; (3) normal R, T and D; and (4) no erections, which can be assigned to different levels and completeness of spinal cord lesions. Nocturnal erections of normal quality need preservation of thoracolumbar and sacral neuronal control as well as partially intact connections of the spinal erection centres with brain areas responsible for sexual arousal.

International Journal of Impotence Research (2004) 16, 433–440. doi:10.1038/sj.ijir.3901188

Published online 11 March 2004

Keywords: erectile dysfunction; spinal cord injury; nocturnal penile tumescence; electrophysiology; neuronal erectile control

Introduction

Although examinations of the neurogenic voiding disorders of spinal cord injury patients are well standardized, there is less agreement on how to assess neurogenic impotence. Basic physiological knowledge of the neurogenic control of human male sexual function is mainly extrapolated from animal studies¹ or based on the clinical observation of sexual disorders following a spinal cord lesion.² Some of the diagnostic methods that have been suggested to assess erectile dysfunction (ED) as nocturnal penile tumescence recordings³ (NPTR),

intracavernous injections⁴ and corpus cavernosum EMG⁵ are not sufficiently standardized or delicate to perform. Moreover, most of these tests examine only one component (vascular, psychogenic or neurogenic) of the complex erection phenomenon, whereas others, especially NPTR, are controversial regarding their diagnostic value.^{3,6} Up to now, hardly any study has been published dealing with NPTR findings in spinal cord injured (SCI) men with a defined neurogenic lesion, which had no ED before trauma. SCI men often suffer from post-traumatic neurogenic ED, depending on the level and completeness of the spinal lesion: There are three nervous systems, that is, the parasympathetic, sympathetic and the somatic, which are involved in triggering erections and which could be damaged after a spinal trauma. With regard to the assessment of neurogenic ED and for future checking of results after treatment, we investigated whether NPTR findings in SCI patients may correlate to different types of erectile deficits.

*Correspondence: DM Schmid, MD. Current address: Department of Urology, University Hospital Zurich, Frauenklinikstrasse 10, CH-8091 Zurich, Switzerland.
E-mail: daniel.max.schmid@usz.ch

Received 18 June 2003; revised 3 November 2003; accepted 20 November 2003

Population and methods

Patients

Between January 2001 and June 2002, 25 male SCI patients aged 18–57 y (average: 35 y) admitted to our spinal cord unit were examined prospectively. All but one presented a traumatic spine lesion. All 25 were in the acute (ie less than 12 months after injury but out of spinal shock phase): 18 were paraplegic and seven tetraplegic patients. The spinal cord lesion was incomplete in 12 cases and complete in 13 cases. All patients had normal preaccidental sexual function, as reported by the patient himself or by his sexual partner. None of the patients presented peripheral neuropathy, brain damage or endocrinal diseases, which could have influenced their sexual function. The hormonal status (FSH, LH, prolactine, testosterone) was normal in all cases. The projected study received the full approval of the Local Ethics Committee.

Anamnesis

Sexual anamnesis aimed at estimating pre- and post-accidental erectile function was always obtained by patient's self-report and partner interview. Special attention was given to the presence or absence of reflexive and/or psychogenic erections.

Considering the traditional dichotomy of the reflexive-psychogenic erection mechanisms, we defined 'reflexive erections' (RE) as those elicited by direct penile/perineal somesthetic stimulation and mediated by sacral spinal segments.^{7,8} On the other hand, 'psychogenic erections' (PE) were defined as those resulting from any extrinsic nonsomesthetic stimuli (visual, auditory, chemosensory) received via the cranial nerves or elicited by erotic thoughts, fantasy or memory.

Clinical, neurophysiological and urodynamical examination

All patients underwent a complete neurological examination in accordance with the ASIA scores.⁹ Special attention was given to the loss or preservation of voluntary contraction of the external anal sphincter, to the bulbocavernous reflex (BCR) and to the sensory impairment of the sacral dermatomes S2–S5.

To define level and completeness of the spinal lesion, electrophysiological examinations, comprising BCR (tests integrity of the sacral conus and the spinal sacral reflex volley), perineal sympathetic skin responses (SSR: examine spinal efferent

sympathetic pathways to the genitalia) and pudendal somatosensory evoked potentials (pSSEP: assess spinal afferent somatosensory nervous pathways arising from the genitalia) were recorded regularly in addition to clinical tests. Finally, all underwent urodynamics in order to assess their neurogenic bladder type (ie detrusor hyper-reflexia, areflexia or normal micturition), according to the recommendations of the ICS.¹⁰

Four patient groups were defined (Figure 2): Group A ($n=10$): level/completeness of the lesion suprasacral/complete (C4–T8); group B ($n=5$): level/completeness of the lesion lumbosacral/complete (L2–S3); group C ($n=7$): level/completeness of the lesion incomplete (C4–S2) and group D ($n=3$): lesion/completeness of the lesion thoracolumbar and sacral/complete (T8–S5: myelomalacia).

NPTR and audiovisual sexual stimulation

Erections were recorded noninvasively with a conventional Rigiscan[®] plus device (Dacomed Cooperation, Minneapolis, MN, USA) provided with a loop placed at the base and another at the tip of the penis (Figure 1). All medications that could have influenced the recordings were stopped at least 4 days prior to measurement. If used, urine condom was removed during the recording period, the bladder having been emptied by clean-intermittent catheterization before placing the device. Nocturnal tumescence recordings were obtained in 25 patients. In four cases, nocturnal tumescence recordings were completed by the recordings obtained during psychogenic stimulation, the patients lying comfortably on a bed watching an erotic movie and having been asked not to masturbate.¹¹ Normal NPTRs were defined as: increase in rigidity >80%, changes in the penile circumference of >1.5 cm (distal) and >2.5 cm (proximal), duration >10 min (plateau >5 min) and frequency of >3 erections/8 h recording time.¹²

Testing of RE and PE

In addition to patient's self-reports, RE capacity was tested using a vibrator device (Elstar Elektronik AG, Basel, CH) and PE potency was assessed with audiovisual sexual stimulation (AVSS) using erotic movies or literature.¹¹

PGE1 intracavernous injection-test

In all patients, an intracavernous injection (ICI)-test with 5–10 µg PGE1 was performed to exclude any

major vascular disease. To avoid a possible negative influence of the fear of injection and a sympathetic induced detumescence, two consecutive tests were obtained at 1-day intervals.



Figure 1 The Rigiscan® device (Dacomed Cooperation, Minneapolis, MN, USA) for NPTR recordings.

Statistical analysis

Statistical evaluation was performed using a SAS software package. A variance analysis and subsequently the Scheffe's test (level of significance $P < 0.05$) for the variables rigidity, tumescence and duration was performed for paired comparison of the four groups A–D.

Results

The results of the NPTR examinations are presented in Table 1 and Figures 2 and 3.

Level and completeness of SCI and erection type

According to vibratory and AVSS test results, 15 subjects had either reflexive erections ($n = 10$, group A) or psychogenic erections ($n = 5$, group B), whereas seven had both types (group C) and three were complete impotent (group D). All 17 patients with complete or incomplete SCI above the conus medullaris had RE potency. Of these patients, 10 with complete lesions (above both sacral and thoracolumbar segments) had no PE, whereas the seven with incomplete lesions preserved PE. All five patients with complete lesion below the TL level and conus/cauda equina injury presented with PE but absent RE.

ICI-tests

All patients showed prompt (within 5 min), and strong ($> 80\%$ rigidity; > 3 cm tumescence) erections

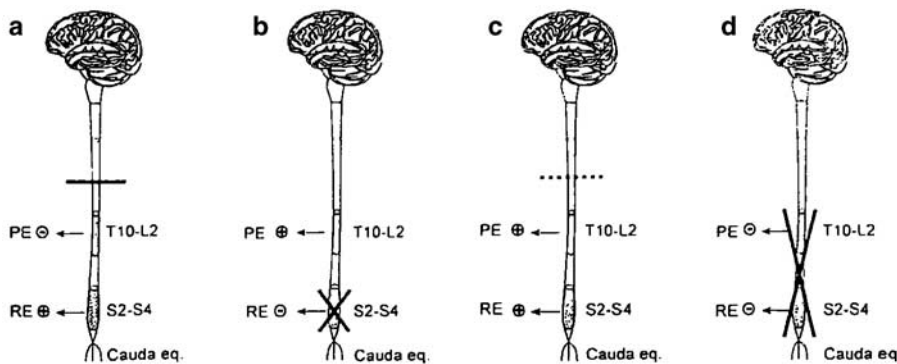


Figure 2 Neurogenic ED depends on the level and severity of the spinal lesion: sympathetic spinal erection centre involves metameres T10–L2 and parasympathetic spinal erection centre involves metameres S2–S4. (a) Complete lesion above both thoracolumbar and sacral erection centres. (b) Complete lesion on the level of the sacral erection centre. (c) Incomplete lesion above or between thoracolumbar and sacral erection centres. (d) Complete lesion of both thoracolumbar and sacral erection centre (myelomalacia).

Table 1 Neurological and urodynamic findings and NPTR recording results in 25 SCI male patients

SCI pat. ♂	n	Spinal lesion level and severity	Electro-physiology	Urodynamic recordings	Rigidity (%)	Tumescence (cm)	Duration (min)	Erection type
Group A	10	C4–T8 Complete suprasacral	SSR neg. BCR pos. pSSEP neg.	Complete detrusor-hyper-reflexia	83.3 ± 8.5 s.d.	3.3 ± 0.9 s.d.	6.4 ± 3.7 s.d.	RE
Group B	5	L2–S3 Complete lumbosacral	SSR pos. BCR pos./neg. pSSEP neg.	Complete detrusor-areflexia	46.1 ± 13.4 s.d.	1.6 ± 0.6 s.d.	5.5 ± 2.7 s.d.	PE
Group C	7	C4–S2 Incomplete suprasacral	SSR pos. BCR pos. pSSEP pos.	Incomplete detrusor-hyper-/areflexia or normal voiding	89.7 ± 7.7 s.d.	3.8 ± 0.4 s.d.	29.1 ± 8.5 s.d.	RE/PE
Group D	3	T8–S5 Complete thoracolumbar & sacral	SSR neg. BCR neg. pSSEP neg.	Complete detrusor-areflexia, bladderneck-incompetence	0	0	0	0 erections

SSR = sympathetic skin responses; BCR = bulbocavernous reflex; pSSEP = pudendal somatosensory evoked potentials; RE = reflexive erections; PE = psychogenic erections.

of at least a duration of half an hour, indicating good relaxation of cavernous tissue.

NPTR findings

In all 10 SCI patients of group A, mean penile rigidity and tumescence measured during NPTR were normal ($83.3 \pm 8.5\%$ and 3.3 ± 0.9 cm, respectively), whereas erection duration was too short (6.4 ± 3.7 min). In all these cases, preserved NPTR contrasted with absence of psychogenic erections. Urodynamics showed detrusor hyper-reflexia. All five patients of group B showed impaired NPTR; however, they revealed psychogenic diurnal erections by absence of RE. In these cases, erections were always of short duration (5.5 ± 2.7 min) with reduced rigidity ($46.1 \pm 13.4\%$) and tumescence (1.6 ± 0.6 cm). Three of these patients could not perform sexual intercourse unless they were under oral treatment. Urodynamically, they had detrusor areflexia. NPTR were of normal quality (mean rigidity $89.9 \pm 7.7\%$, mean tumescence 3.8 ± 0.4 cm and mean duration 29.1 ± 8.5 min) only in the seven motor and sensory incomplete SCI patients of group C with preserved psychogenic as well as reflexive erections. In all these cases, the electrophysiological recordings were normal or only slightly altered and urodynamics revealed normal or less disturbed micturition. Finally, the three patients of group D with complete injury of T8–S5 spinal segments (myelomalacia) showed no measurable NPTR and neither RE nor PE could be elicited. Urodynamic recordings showed detrusor areflexia and bladder neck incompetence.

Discussion

Neurogenic sexual dysfunction of SCI male patients depends on the level and completeness of their lesion. *Reflexive erections* occur in patients with intact sacral conus and cauda equina, independent of its central afferent connections to forebrain areas, by a somatosensory pudendal afferent → autonomic pelvic efferent spinal reflex volley. However, *psychogenic erections* are observed in patients with preserved sacral (S2–S4) erection centre and partially intact central efferent connections or, in the case of injured or disconnected conus, intact thoracolumbar (T10–L2) segments, the lesion being below L2.^{13,14}

The neurological and urodynamic examinations combined with patient's anamnesis might indicate the type of expected sexual disorders. However, in many cases (about 30%) discordance between patient's self-report and clinical examination exists, as patients often underestimate their erectile

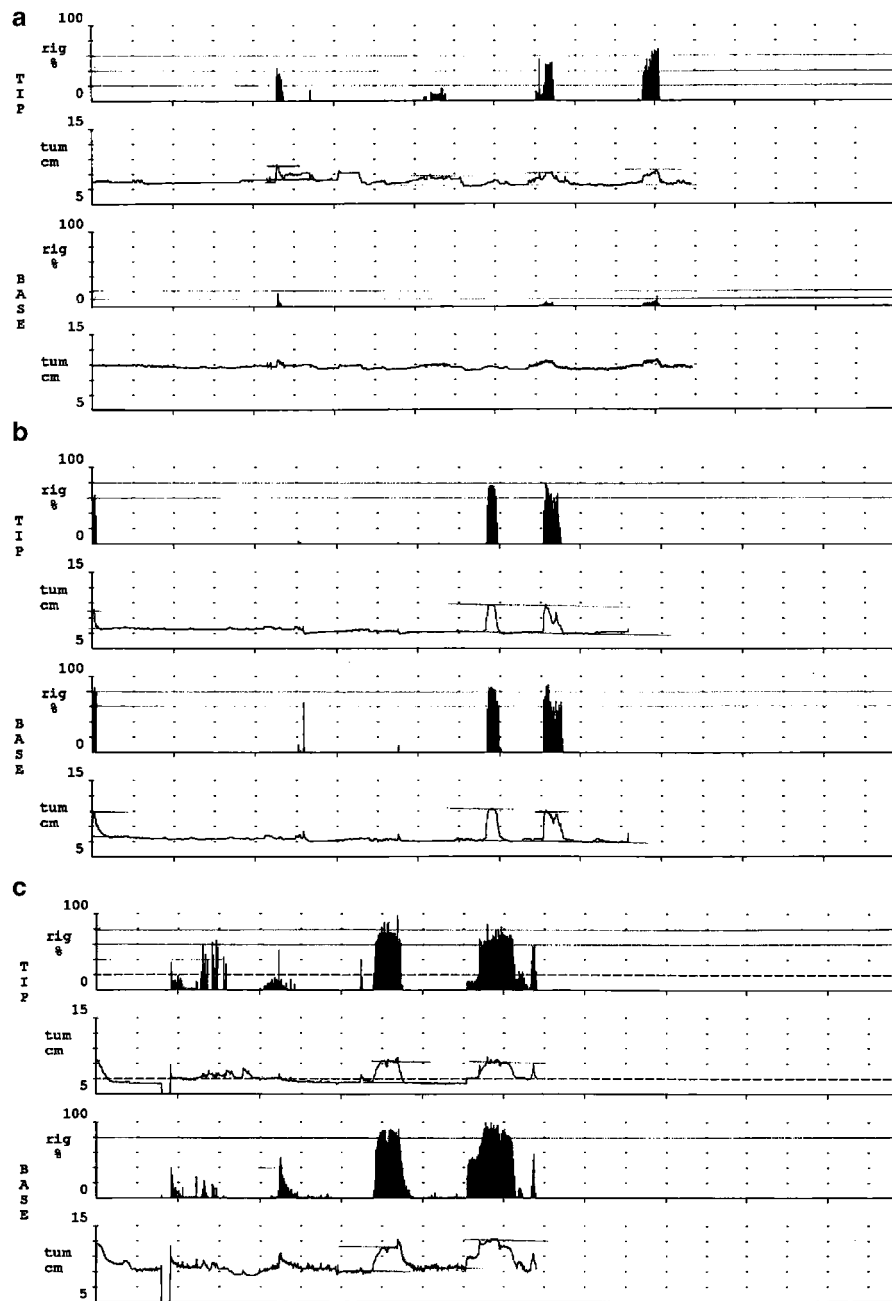


Figure 3 Three types of NPTR patterns. (a) NPTR pattern in patients with complete lesion on the level of the sacral erection centre (Group B). (b) NPTR pattern in patients with complete lesion above both thoracolumbar and sacral erection centres (Group A). (c) NPTR pattern in patients with incomplete lesion above or between thoracolumbar and sacral erection centres (Group C). Normal NPTR pattern could only be found in cases of partially preserved thoracolumbar *and* sacral erection centres, both of which still have connections to higher brain centres (group C).

capacity. The purpose of this study was to assess the value of the NPTR measurement in SCI men with neurogenic sexual dysfunction in order to get more insight into the origin of different erection types and to plan individual sexual therapy better.

We found four typical patterns of NPTR curves in our SCI patients with statistically different values for tumescence, rigidity and duration

(Scheffe's test, $P < 0.05$), which can be assigned to different levels and completeness of spinal cord lesions. The pattern clearly depends on the integrity of the thoracolumbar (sympathetic: T10–L2) and the sacral (parasympathetic: S2–S4) spinal erection centres, respectively, and on their connections to forebrain centres by autonomic supraspinal efferents (descending in the intermediolateral column) as

well as by pudendal-mediated somatosensory afferent pathways (ascending in the posterior spinal column). Patients with a complete lesion above the sympathetic (T10–L2)/parasympathetic (S2–S4) spinal erection centres had strong but short nocturnal erections despite the absence of waking psychogenic erections (group A). This finding might be explained by a centrally independent, uninhibited spinal erection generator on sacral level, even if a direct reflexive response to the Rigiscan[®] recording loops cannot completely be excluded. Patients with preserved reflex erections but impaired or missing perineal/penile sensibility, substantiated by strongly disturbed or absent pSSEP, had erections of too short duration, which disappeared as soon as the stimulus (vibration) had stopped. This finding was in accordance with strong, but short nocturnal erections. This fact underlines the importance of an intact afferent somatosensory nervous pathway projecting from pudendal sensory fibres up to hypothalamic areas involved in sexual arousal. The start of an erection is governed by the autonomic nervous system via vasomotor control. Erection duration then is maintained by somatosensory afferent nerve impulses released by tactile stimulation of the glans penis during intercourse. These peripheral impulses seem to influence forebrain centres modulating sexual arousal, which in return stimulate spinal erection centres.^{15–18} The influence of penile sensory disorders on ED has already been described by Bemelmans *et al*¹⁶ in 58 patients with complaints of too short and weak erections, which had at least one abnormal neurophysiological measurement of their pudendal nerve.

However, patients with preservation of the sympathetic thoracolumbar spinal centre (T10–L2) but complete injury of the parasympathetic and somatic sacral spinal centres (S2–S4) had poor and short nocturnal tumescence and rigidity, despite diurnal psychogenic erections (group B). For a long time, the cerebrospinal pathway projecting on the sacral erection centre was considered to mediate solely psychogenic as well as nocturnal erections. If we assume that in SCI patients with completely destroyed reflexogenic sacral erection centre (S2–S4), their thoracolumbar sympathetic erection centre (T10–L2) is not only responsible for detumescence (via adrenergic transmitters^{19,20}), but also becomes a proerectile function (via a still unknown pathway), then our results underline the theory of a sympathetic dichotomy, which was already described by other authors in animal models and from clinical observations in SCI patients.^{21–25} Accordingly, our SCI patients with destroyed sacral conus and preserved thoracolumbar metameremes were still able to get erections after audiovisual stimulation; direct tactile stimulation of the glans penis never resulted in erections in these cases. Whereas in healthy men, the parasympathetic sacral erection centre seems to play a major role in producing

reflexive *and* psychogenic erections as well as nocturnal erections, our NPTR findings corroborate the observation that many men with lesion of the sacral conus maintain sympathetic induced psychogenic and nocturnal erections.^{11,13,26,27} Whether this phenomenon is due to a neuronal plasticity process in a still intact thoracolumbar centre, which then assumes a compensatory proerectile function, remains to be proven. Transection and stimulation experiments of the hypogastric nerve (HGN) in animals showed controversial results.^{21–25}

Nearly normal NPTRs could be observed only in patients with incomplete lesions above or between both erection centres, presenting with normal or slightly disturbed electrophysiological (SSR, pSSEP, BCR) and urodynamic recordings (group C). This may be explained by the fact that both spinal erection centres and their somatosensory afferent/autonomic efferent connections to brain centres are left more or less intact, allowing nearly normal induction of erections. As was expected, patients with a spinal myelomalacia involving lesions of both the thoracolumbar as well as the sacral erection centres lost any type of erections in clinical tests as well as in NPTR measurements (group D).

The origin of *nocturnal erections (sleep-related erections)*, occurring during REM phases of sleep, is still a controversial topic. For this reason, extensive discussion has been undertaken in relation to NPTRs and their validity in the assessment of neurogenic function disorders.^{3,15,28,29} Recordings do not always correlate with patient's self-reports about their own sexual capacity. This may be due to the fact that erections recorded by NPTRs occur during sleep under conditions that do not correspond to waking erectile function. It seems that nocturnal erections and waking PE/RE are generated by different neuronal mechanisms. To our knowledge, there is only one experimental approach in a rat model dealing with the neurophysiology of REM sleep-related erections by Schmidt *et al*.^{30–32} The authors elucidated by neural transections the effects of paraplegia on REM sleep-related erections and determined the brain level at which the mechanisms underlying erectile activity are generated. It has to be mentioned that Schmidt's group investigated primarily the influence of high spinal cord and forebrain transections to erection function in rats,³⁰ whereas we targeted the effects of different spinal lesion levels and completeness to human erectile function. In their model, spinal transections virtually eliminated REM sleep-related erections. In our study, complete spinal cord injury above both thoracolumbar and sacral level significantly disturbed nocturnal erection activity, but could never eliminate it. We have already discussed above, whether a centrally inhibited sacral spinal erection generator might be the explanation for this phenomenon: Indeed, spinal transections in rats shortened the latency to reflex erection induction, whereas

mesencephalic transections increased this latency to reflex erection induction. Schmidt concluded that forebrain structures (rostral to the mesencephalon) are essential for the maintenance and integrity of REM sleep-related erections, whereas the brainstem is not. Spinal transection removes tonic descending inhibition of erections occurring from brainstem, whereas mesencephalic transection enhance such inhibition. He suggested that from these data, the forebrain plays a facilitatory role in erectile context through disinhibition of brainstem tonic antierecile mechanisms.³¹ Our SCI patients with incomplete cord lesions, that is, partially intact connections to forebrain centres, were the only ones with nearly normal erection duration during REM sleep, corroborating Schmidt's suggestions of a proerecile role of the lateral preoptic area.³² However, waking-state erections remained unchanged after neurotoxic lesions of this forebrain area.³² These data show that higher erectile mechanisms appear to be context-specific, as we concluded from our clinical observation, that quality of nocturnal erections does not always reflect waking erectile function. Whereas abnormal NPTRs imply—almost without exception—waking ED (except sleep disturbances), a normal NPTR result in patients with neurological disorders only suggests intact erectile mechanism during sleep, not warranting waking erections, which could be used for sexual intercourse.¹⁵

NPTR tests dynamically assess the functional aspect of erections; overall, they reveal at least some typical patterns hinting at the injury/disruption of spinal erection centres of a male patient with known spinal lesion. On the other hand, we cannot draw reliable conclusions about the aetiology of ED (ie vasculogenic, psychogenic, hormonal or neurogenic) from the base of NPTR curves alone without having the information of a defined and assessed spinal lesion. Nevertheless, NPTRs may help clinicians to better inform their SCI patients about remaining erectile function in their life after the trauma.

Conclusions

Our study demonstrates that ED in SCI men can be reliably assessed and characterized using NPTR measurement combined with clinical examination, electrophysiological recordings and urodynamic evaluation. Quality of erections correlates with the lesion's level and its completeness in SCI men. Patients with lesion of the sacral erection centre showed the most disturbed NPTR pattern. Normal erection function depends on preserved thoracolumbar and sacral efferent nervous outflow and on intact connections of spinal erection centres to forebrain areas, the latter being important for maintaining sufficient erection duration.

Acknowledgements

This work was partially funded by the International Institute for Research in Paraplegia (P17/97-2000) and the Swiss National Science Foundation (32-52562.97).

References

- 1 De Groat WC, Booth AM. Neural control of penile erection. In: Maggi CA (ed) *The Autonomic Nervous System*. Academic Publishers: London, UK, 1993, pp 467–524.
- 2 Courtois FJ *et al*. Clinical approach to erectile dysfunction in spinal cord injured men. A review of clinical and experimental data. *Paraplegia* 1995; **33**: 628–635.
- 3 Morales A, Condra M, Reid K. The role of nocturnal penile tumescence monitoring in the diagnosis of impotence: a review. *J Urol* 1990; **143**: 441–446.
- 4 Wyndaele JJ. Correlation between clinical neurological data and urodynamic function in spinal cord injured patients. *Spinal Cord* 1997; **35**: 213–216.
- 5 Wagner G, Gerstenberg T, Levin RJ. Electrical activity of corpus cavernosum during flaccidity and erection of the human penis: a new diagnostic method? *J Urol* 1989; **142**: 723–725.
- 6 Fowler CJ, Ali Z, Kitby RS, Pryor JP. The value of testing for unmyelinated fibre sensory neuropathy in diabetic impotence. *Br J Urol* 1988; **61**: 63–67.
- 7 Sachs BD. Placing erection in context: the reflexogenic-psychogenic dichotomy reconsidered. *Neurosci Biobehav* 1995; **19**: 211–224.
- 8 Bernabe J, Rampin O, Sachs BD, Giuliano F. Intracavernous pressure during erection in rats: an integrative approach based on telemetric recording. *Am J Physiol* 1999; **276**: R441–R449.
- 9 Maynard FM *et al*. International standards for neurological and functional classification of spinal cord injury. *Spinal Cord* 1997; **35**: 266–274.
- 10 Stöhrer M *et al*. The standardization of terminology in neurogenic lower urinary tract dysfunction: with suggestions for diagnostic procedures. International Continence Society Standardization Committee. *NeuroUrol Urodyn* 1999; **18**: 139–158.
- 11 Courtois FJ, Goulet MC, Charvier KF, Leriche A. Posttraumatic erectile potential of spinal cord injured men: how physiologic recordings supplement subjective reports. *Arch Phys Med Rehabil* 1999; **80**: 1268–1272.
- 12 Colombo F, Fenice O, Austoni E. NPT—Test di tumescenza peniena notturna. *Arch Ital Urol* 1994; **66**: 159–164.
- 13 Bors E, Comarr AE. Neurological disturbances of sexual function with special reference to 529 patients with spinal cord injury. *Urol Surv* 1960; **10**: 191–222.
- 14 Comarr AE. Sexual function among patients with spinal cord injury. *Urol Int* 1970; **25**: 134–168.
- 15 Van Nueten J, Verheyden B, Van Camp K. Role of penile nocturnal tumescence and rigidity measurement in the diagnosis of erectile impotence. *Eur Urol* 1992; **22**: 119–122.
- 16 Bemelmans BLH *et al*. Penile sensory disorders in erectile dysfunction: results of a comprehensive neuro-urological diagnostic evaluation in 123 patients. *J Urol* 1991; **146**: 777–782.
- 17 Herbert J. The role of dorsal nerves of the penis in the sexual behaviour of the male rhesus monkey. *Physiol Behav* 1973; **10**: 293.
- 18 Padma-Nathan H. Neurologic evaluation of erectile dysfunction. *Urol Clin North Am* 1988; **15**: 77.

- 19 Andersson KE, Stief CG. Neurotransmission and the contraction and relaxation of penile erectile tissues. *World J Urol* 1997; **15**: 14–20.
- 20 Traish A, Kim NN, Moreland RB, Goldstein I. Role of alpha adrenergic receptors in erectile function. *Int J Impot Res* 2000; **12**(Suppl 1): S48–S63.
- 21 Courtois FJ, Macdougall JC, Sachs BD. Erectile mechanism in paraplegia. *Physiol Behav* 1993; **53**: 721–726.
- 22 Dail WG, Walton G, Olmsted MP. Penile erection in the rat: stimulation of the hypogastric nerve elicits increases in penile pressure after chronic interruption of the sacral parasympathetic outflow. *J Auton Nerv Syst* 1989; **28**: 251–257.
- 23 Root WS, Bard P. The mediation of feline erection through sympathetic pathways with some remarks on sexual behavior after deafferentation of the genitalia. *Am J Physiol* 1947; **151**: 80–90.
- 24 Semens JH, Longworthy OR. Observation on the neurophysiology of sexual function in the male cat. *J Urol* 1938; **40**: 836–846.
- 25 Cruz MR *et al*. Peripheral nerves mediating penile erection in the rat. *J Auton Nerv Syst* 1999; **76**: 15–27.
- 26 Chapelle PA, Durand J, Lacert P. Penile erection following complete spinal cord injury in man. *Br J Urol* 1980; **52**: 216–219.
- 27 Courtois FJ *et al*. Sympathetic skin responses and psychogenic erections in spinal cord injured men. *Spinal Cord* 1998; **36**: 125–131.
- 28 Marshall P, Surrive D, Delva N. The role of nocturnal penile tumescence in differentiating between organic and psychogenic impotence: the first stage of validation. *Arch Sex Behav* 1981; **10**: 1–11.
- 29 Thase ME *et al*. N.T.M. in depressed men. *J Psychiatry* 1987; **144**: 89.
- 30 Schmidt MH *et al*. Experimental evidence of penile erections during paradoxical sleep in the rat. *Neuroreport* 1994; **5**: 561–564.
- 31 Schmidt MH, Sakai K, Valatx JL, Jouvet M. The effects of spinal or mesencephalic transections on sleep-related erections and ex-copula penile reflexes in the rat. *Sleep* 1999; **22**: 409–418.
- 32 Schmidt MH *et al*. Role of the lateral preoptic area in sleep-related erectile mechanisms and sleep generation in the rat. *J Neurosci* 2000; **20**: 6640–6647.