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INTRODUCTION

The hospital in Hornbæk (Fysiurgisk hospital) was established November 1952 in the previously fashionable seaside hotel with the aim to treat children paralysed from poliomyelitis. When the polio-epidemic came to an end, other patient categories with severe physical disabilities were admitted.

In the early days up to 200, primarily children, were admitted. As the hospital increasingly admitted adults, who were wheelchair bound, the need for a thorough renovation of the building increased. September 1988 this modernization of the hospital was completed, and today the hospital is staffed to receive 42 patients with spinal cord lesions.

Initially the hospital was bought and ran by the Society and Home for the Disabled (Samfundet og Hjemmet for Vanføre), but in 1980 it was taken over by the State, and in 1995 the Copenhagen Hospital Cooperation (H:S), and established as a department in the University hospital Rigshospitalet. Therefore in 1980 it became possible to create an extensive cooperation with the other departments in Rigshospitalet working with the many challenges individuals with spinal cord lesions face. Apart from the service departments (Clinical Biochemistry, Microbiology and Neurophysiology, Radiology, and Clinical physiology/Nuclear medicine) it is the clinics for Neurosurgery, Urology, Plastic surgery, Gastroenterology, Orthopaedic surgery including Hand surgery, Gynaecology and Obstetrics, Neurology, Neuroanaesthesia including the Centre for Respiratory Disabled and the Pain Clinic, Paediatrics including the Neuropaediatric Clinic, Psychiatry including the Clinic for Sexology, and others. In addition, over the years the hospital has established cooperation with many other hospitals and research institutions within as well as outside Denmark. Further the Clinic cooperates closely with the Patient Associations.

Within the Clinic for Para- and Tetraplegia, as it was renamed in 2000, as a consequence of the changed patient clientele, the treatment and rehabilitation of the spinal cord lesioned persons has always been accomplished within the Multi-disciplinary team, consisting of nursing staff, physiotherapists, occupational therapists, social workers, psychologists, and doctors, supported by the secretaries, as well as all the other staff members in the cleaning and maintenance departments and the kitchen.

This 50-year Anniversary Book gives among other information a State of the Art insight to some of the many areas, which may challenge the spinal cord lesioned individuals during their lives.

On behalf of the staff at the Clinic for Para- and Tetraplegia

Hanne Gregersen and Fin Biering-Sørensen

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INCIDENCE OF SPINAL CORD LESIONS IN EUROPE

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SUMMARY

This is a survey on spinal cord lesions (SCL) in Europe. Postal questionnaires were mailed to representatives of the European Network of country representatives of the International Medical Society of Paraplegia (IMSOP).

Twenty-one representatives representing a population of 415.7 million responded. The average annual incidence of traumatic SCL was 17.2 per million inhabitants. The incidence for Spina Bifida (SB) was 6.6/mill./year in average. For the other non-traumatic SCL the incidence was reported to be 8.0 in average.

The number of hospitals/units treating acute traumatic SCL per million inhabitants ranged from 3.9 to 0.16. The number of beds available for this function was 7.7 in median per million inhabitants. The rehabilitation of patients with SCL generally seems centralised to fewer institutions than the acute treatment, the median being 0.5 rehabilitation facilities per million inhabitants, and the number of beds for rehabilitation of SCL patients was in median 13.5 per million inhabitants.

Although the information is mostly subjective estimates the figures gives a picture of the present situation in Europe, in particular regarding facilities available for treatment and rehabilitation of individuals with SCL.

The annual incidence of traumatic spinal cord lesions (SCL) has in various studies varied from 8 to 60 per million inhabitants (1-13). A recent study

from Mississippi found even an incidence for patients in hospitals and pre-hospital fatal cases of 77 per million. The rate for patients in hospitals was 59 per million (14). Most persons with traumatic SCL, are males (3-5 to 1) in their late teens or twenties, but they seems in recent years to become older (15). Most often they are injured in traffic accidents, followed by falls or violence, depending on the part of the world in focus.

McKinley et al. (16) found in a 5-year prospective study of 220 SCL admissions, 39% to be non-traumatic in aetiology (spinal stenosis 54%; tumour 26%). They were significantly older and more likely married, female, and retired than the traumatic SCL individuals. In addition, more had paraplegia and incomplete lesions. In another study (4) the same proportion of admissions were non-traumatic, the aetiology being tumours in 44%, infections in 19%, vascular in 16%, and Spina Bifida in 5%.

The literature on Spina Bifida gives the incidences in cases per 10,000 births. The incidences reported vary from 0.9 to 36 (17-31). This large variation includes a significant decline from the seventies and eighties to the nineties in certain countries, further there are differences associated with inclusion of stillbirth and preterm terminations of pregnancies. In addition, there are of varying patterns of neural tube defects in different populations (18,19,21-23,26-28,32,33), and the use of folate (28,34).

The aim of this paper is to give the results of a survey on incidence of SCL

in Europe, together with information about hospitals, rehabilitation facilities and beds available for these patients.

MATERIAL AND METHODS

All 34 national representatives of the European Network of the International Medical Society of Paraplegia (IMSOP) were sent a questionnaire including questions about: Number of inhabitants in your country (population size). Estimated number of new traumatic spinal cord injuries per year. Estimated number of new Spina Bifida cases with spinal cord lesion per year. Estimated number of other new non-traumatic spinal cord lesions per year (due to prolapsed disc, spinal stenosis, spondylitis, epidural abscesses, tuberculosis and other infectious causes, benign tumours etc. But not due to progressive causes like multiple sclerosis or cancer). How many hospitals/units in your country treat acute spinal cord injuries. Estimated total number of beds for this function. How many hospitals/units/centres/facilities in your country carry out rehabilitation of individuals with spinal cord lesion. Estimated total number of beds for this function.

The received answers were collected, and circulated to all the responders for their approval. Here the data from Israel, Kyrgyzstan and Turkey have been excluded, as these countries geographically are entirely or mostly outside Europe.

RESULTS

Twenty-one country representatives representing a population of 415.7 million inhabitants of Europe responded to the questionnaire.

In Table 1 the annual incidences per million inhabitants are given.

Table 1	Traumatic SCL	Spina Bifida With SCL	Other non-traumatic
Country			
Austria		5.4	
Belgium	12.0	2.0	25.0
Denmark	11.5	3.1	11.5
England	16.7		
Estonia	33.3		
Finland	10.8	2.9	3.9
France	16.7	5.0	5.0
Germany	10.1	10.1	5.1
Hungary	22.5		
Iceland	14.8	3.7	
Ireland	10.7		5.3
Italy	21.4		10.7
Netherlands	9.6		9.6
Norway	15.6		11.1
Romania	41.3		
Slovenia	20.0	1.5	20.0
Spain	17.6	2.4	8.1
Sweden	15.7	6.2	5.6
Switzerland	26.7	2.7	13.3
Yugoslavia	12.1	17.3	8.0
Total	17.2	6.6	8.0

The median frequency of traumatic SCL was 15.7 per million per year, for SB was the median 3.4, and for the other non-traumatic SCL the median was 8.9.

For those 14 countries reporting incidences for traumatic as well as other non-traumatic SCL the median proportion traumatic/non-traumatic was 1.99 (range 0.5-3.3).

Table 2 gives the number per million inhabitants of hospitals/units treating

Table 2	Hospitals treating acute	Beds for acute treatment	Hospitals Rehab SCL	Beds for rehab of SCL
Country	Number/mill.	Number/mill.	Number/mill.	Number/mill.
Austria	1.00	6.25	0.38	22.50
Belgium			0.60	12.00
Croatia	0.40	10.00	0.20	10.00
Denmark	0.58	1.54	0.38	13.46
England	0.17	7.50	0.17	7.50
Estonia	1.33	66.67	2.00	83.33
Finland	3.92		0.20	4.90
France	0.42	1.67	0.50	13.33
Germany	0.71		0.27	13.98
Hungary	0.80	10.00	0.90	
Iceland	3.70	3.70	11.11	29.63
Ireland	1.07	1.60	0.27	13.33
Italy	0.16	3.21	0.89	6.25
Netherlands	0.96		0.51	12.74
Norway	1.33		0.67	11.56
Romania	1.52			
Slovenia	1.50	14.50	0.50	17.50
Spain	0.30	14.19	0.30	14.19
Sweden	0.67	7.87	3.37	21.35
Switzerland	1.33		0.53	33.33
Yugoslavia	0.94	107.36	0.75	13.77
Total	0.60	10.50	0.52	12.46

acute SCL, beds for this function, hospitals/units/centres/facilities carrying out rehabilitation of individuals with SCL, and beds for this function as well. The most decentralised system for the acute treatment of traumatic SCL seems to be Finland with 3.92 hospitals per million inhabitants. Nearly the same is true for Iceland, but they receive only these patients in one hospital, but have the high ratio because of a numeric very little population. The

most centralised treatment in the acute phase was reported for England and Italy with 0.16-0.17 acute hospitals per million. The number of beds available for this function was 7.7 in median.

Except from the situation in Sweden, Estonia and Iceland, the rehabilitation of patients with SCL generally seems centralised to fewer institutions than the acute treatment of the traumatic SCL, the median being 0.5 rehabilitation facilities per million inhabitants.

Table 3 First author and year published	Country	Study year(s)	Incidence of traumatic SCL per mill. Per year	Comments
Gehrig 1968 (35)	Switzerland	1960-67	Approximately 15	Alive at hospital admission
O'Gorman 1974, cited from (39)	Ireland	1973	19.8	Adjusted for 72% coverage of hospital admissions
Minaire 1978-79 (40)	France – Rhône-Alpe	1970-75	12.7	Alive at hospital admission
Gjone 1978-79 (36)	Norway	1974-75	6.5	Alive at hospital admission
Knútsdóttir 1993 (37)	Iceland	1973-89	24 (1973-82) 18 (1983-89)	
Biering-Sørensen (2)	Denmark	1975-84	9.2	Surviving the 1990 acute phase. Have SCL of some severity. Excl. ventilator dependent
Garcia-Reneses 1991(4)	Spain – selected hospitals	1984-85	8	Incomplete data collection and delimitation
Köning 1989 (38)	Federal Republic of Germany	19853	5.7	Alive at hospital admission. indirect calculation
Martins 1998 (8)	Portugal – central region	1989-92	57.8, incl. deaths 25.4 for those surviving	16% dead upon arrival at hospital. 40% died before release
Soopramanien 1995 (41)	Romania – Bucharest	1992	At least 28.5	Extrapolation to all Romania
Asbeck 2000 (1)	The Netherlands	1994	10.4	Surviving the acute phase. Have persisting symptoms

The median number of beds for rehabilitation of SCL patients per million inhabitants was 13.5.

DISCUSSION

The data presented here has to be interpreted with caution, as the information

are collected on the basis of a postal questionnaire, with only very few explanations. Previous reported figures on incidences of traumatic SCL from Europe are very much in the same range (Table 3):

The incidence of traumatic spinal cord injuries was in average 17.2/mill./year. As pointed out in the literature incidence figures for traumatic SCL may vary very much, depending on the way the information is collected. The incidence will become higher if death at the scene of the accident or during the early hospitalisation is included (8,9,14).

It seems that most countries in Europe do count those who reached the rehabilitation only, which imply a possibility for lower incidences (1,2). Other criteria which may influence the magnitude of the incidence is the period of persistence and the seriousness of the symptoms, which each representative have accepted for inclusion in the report (1,2), such criteria will lower the estimated frequency.

The reported incidences of non-progressive non-traumatic SCL apart from SB varied somewhat. One of the reasons for this variation may be a different tradition in treatment and rehabilitation of these patients. In particular for many traditional Spinal Units this group of patients is relatively new, and several do not have the necessary beds to take up this challenge in full (42). In addition these patients may be treated in several other departments than those, which usually take care of the traumatic SCL patients, therefore some of the estimates may be too low as the awareness of these patients can be low. The proportion between the incidences of traumatic and other non-traumatic SCL are in median similar to those previously reported (4,16).

There may also be differences in the interpretation of traumatic and non-traumatic SCL. This problem is particular prevalent when observing a case of a light trauma in a person with a spinal stenosis. Is this considered being a traumatic or non-traumatic incidence? This can in particular be a problem when considering the geriatric population (43). The incidence rates in the literature on Spina Bifida is given as cases per 10,000 births, and they vary considerably, i.e. at a magnitude of up to 40 times (17-31). Also in Europe there are well known differences between various populations (21,26,28, 32). Some of the variation observed in SB frequencies may be associated with the possible difference in inclusion of stillbirth and preterm terminations of the pregnancies.

The information on the number of hospitals treating acute traumatic spinal cord injuries seem to show that the acute treatment is reasonable centralised in the reporting European countries. Regarding the number of available beds for this function, the very high figures are most probably due to a different interpretation of the question. Thus it might be understood as all beds in the departments, and not just the number of beds in use for the SCL patients. Furthermore the number may also vary according to the use of the beds, in the sense that they may in some departments only be active for a short acute period, while other units use the "same beds" from the early acute treatment and until the discharge from the rehabilitation.

Apart from few exceptions it seems that the rehabilitation of the SCL patients is even more centralised than the acute treatment. For the number of beds available for rehabilitation some of the same precautions as mentioned

Although the information included in this presentation is mostly subjective estimates it is found that the figures give an interesting picture of the present situation in Europe.

REFERENCES

1. Asbeck FWA van, Post MWM, Pangalila RF. An epidemiological description of spinal cord injuries in The Netherlands in 1994. *Spinal Cord* 2000;38:420-4.
2. Biering-Sørensen F, Pedersen V, Clausen S. Epidemiology of spinal cord lesions in Denmark. *Paraplegia* 1990;28:105-18.
3. Chen H-Y, Chiu W-T, Chen S-S et al. A nationwide epidemiological study of spinal cord injuries in Taiwan from July 1992 to June 1996. *Neurol Res* 1997;19:617-22.
4. Garcia-Reneses J, Herruzo-Cabrera R, Martinez-Moreno M. Epidemiological study of spinal cord injury in Spain 1984-1985. *Paraplegia* 1991;28:180-90.
5. Karacan I, Koyuncu H, Pekel Ö et al. Traumatic spinal cord injuries in Turkey: a nationwide epidemiological study. *Spinal Cord* 2000;38:697-701.
6. Karamehmetoglu SS, Nas K, Karacan I, et al. Traumatic spinal cord injuries in Southeast Turkey: an epidemiological study. *Spinal Cord* 1997;35:531-3.
7. Maharaj JC. Epidemiology of spinal cord paralysis in Fiji: 1985-1994. *Spinal Cord* 1996;34:549-59.
8. Martins F, Freitas F, Martins L, Dartigues JF, Barat M. Spinal cord injuries – epidemiology in Portugal's central region. *Spinal Cord* 1998;36:574-8.
9. National Spinal Cord Injury Statistical Centre: Spinal cord injury: Facts and figures at a glance. *J Spinal Cord Med* 2000;23:51-3.
10. Otom AS, Doughan AM, Kawar JS, Hattar EZ. Traumatic spinal cord injuries in Jordan – an epidemiological study. *Spinal Cord* 1997;35:253-5.
11. Price C, Makintubee S, Herndon W, Istre GR. Epidemiology of traumatic spinal cord injury and acute hospitalization and rehabilitation changes for spinal cord injuries in Oklahoma, 1988-1990. *Am J Epidemiol* 1994;139:37-47.
12. Shingu H, Ikata T, Katoh S, Akatsu T. Spinal cord injuries in Japan: a nationwide epidemiological survey 1990. *Paraplegia* 1994;32:3-8.
13. Silberstein B, Rabinovich S. Epidemiology of spinal cord injuries in Novosibirsk, Russia. *Paraplegia* 1995;33:322-5.
14. Surkin J, Gilbert BJ, Harkey HL 3rd, Sniezek J, Currier M. Spinal cord injury in Mississippi. Findings and evaluation, 1992-1994. *Spine* 2000;25:716-21.
15. Nobunaga AI, Go BK, Karunas RB. Recent demographic and injury trends in people served by the Model Spinal Injury Care Systems. *Arch Phys Med Rehabil* 1999;80:1372-82.
16. McKinley WO, Seel RT, Hardman JT. Nontraumatic spinal cord injury: Incidence, epidemiology, and functional outcome. *Arch Phys Med Rehabil* 1999;80:619-23.
17. Alembik Y, Dott B, Roth MP, Stoll C. Prevalence of neural tube defects in Northeastern France, 1979-94 Impact of prenatal diagnosis. *Ann Génét* 1997;40:69-71.
18. Borman B, Cryer C. The prevalence of anencephalus and Spina Bifida in New Zealand. *J Paediatr Child Health* 1993;29:282-8.
19. Buccimazza SS, Molteno CD, Dunne TT, Viljoen DL. Prevalence of neural tube defects in Cape Town, South Africa. *Teratology* 1994;50:194-9.
20. Chan A, Robertson EF, Haan EA, Keane RJ, Ranieri E, Carney A. Prevalence of neural tube defects in South Australia, 1966-91: effectiveness and impact of prenatal diagnosis. *BMJ* 1993;307:703-6.
21. The EUROCAT Working Group. Prevalence of neural tube defects in 16 regions of Europe, 1980-1983. *Int J Epidemiol* 1987;16:246-51.

22. Feuchtbaum LB, Currier RJ, Riggle S, Roberson M, Lorey FW, Cunningham GC. Neural tube defects prevalence in California (1990-1994): eliciting patterns by type of defect and maternal race/ethnicity. *Genet Test* 1999;3:265-72.
23. Hendricks KA, Simpson JS, Larsen RD. Neural tube defects along the Texas-Mexico border, 1993-1995. *Am J Epidemiol* 1999;149:1119-27.
24. Himmetoglu O, Tiras MB, Gursoy R, Karabacak O, Sahin I, Onan A. The incidence of congenital malformations in a Turkish population. *Int J Gyn Obst* 1996;55:117-21.
25. Koch M, Fuhrmann W. Epidemiology of neural tube defects in Gernamy. *Hum Genet* 1984;68:97-103.
26. Kristensen P, Irgens LM, Andersen A, Bye AS, Sundheim L. Birth defects among offspring of Norwegian farmers, 1967-1991. *Epidemiology* 1997;8:537-44.
27. Lian Z-H, Yang H-Y, Li Z. Neural tube defects in Beijing-Tianjin area of China. Urban-rural distribution and some other epidemiological characteristics. *J Epidemiol Community Health* 1987;41:259-62.
28. McDonnell RJ, Johnson Z, Delaney V, Dack P. East Ireland 1980-1994: epidemiology of neural tube defects. *J Epidemiol Community Health* 1999;53:782-8.
29. Rankin J, Glinianaia S, Brown R, Renwick M. The changing prevalence of neural tube defects: a population based study in the North of England, 1984-96. *Paediatric Perinatal Epidem* 2000;14:104-10.
30. Stevenson RE, Allen WP, Pai GS et al. Decline in prevalence of neural tube defects in a high-risk region of the United States. *Paediatrics* 2000;106:677-83.
31. Tuncbilek E, Boduroglu K, Alikasifoglu M. Neural tube defects in Turkey: prevalence, distribution and risk factors. *Turk J Pediatr* 1999;41:299-305.
32. Dolk H, De Wals P, Gillerot Y et al. Heterogeneity of neural tube defects in Europe: the significance of site of defect and presence of other major anomalies in relation to geographic differences in prevalence. *Teratology* 1991;44:547-59.
33. Moore CA, Li S, Li Z et al. Elevated rates of severe neural tube defects in a high-prevalence area in Northern China. *Am J Med Genet* 1997;73:113-8.
34. Rosano A, Smithells D, Cacciani L et al. Time trends in neural tube defects prevalence in relation to preventive strategies: an international study. *J Epidemiol Community Health* 1999;53:630-5.
35. Gehrig R, Michaelis LS. Statistics of acute paraplegia and tetraplegia on a national scale. *Paraplegia* 1968;6:93-5.
36. Gjone R, Nordlie L. Incidence of traumatic paraplegia and tetraplegia in Norway: a statistical survey of the years 1974 and 1975. *Paraplegia* 1978-79;16:88-93.
37. Knútsdóttir S. Spinal cord injuries in Iceland 1973-1989. A follow-up study. *Paraplegia* 1993;31:68-72.
38. Köning W, Frowein RA. Incidence of spinal cord injury in the Federal Republic of Germany. *Neurosurg Rev* 1989;12:562-6.
39. Kurtzke JF. Epidemiology of spinal cord injury. *Exper Neurol* 1975;48:163-236.
40. Minaire P, Castanier M, Girard R, Berard E, Deidier C, Bourret J. Epidemiology of spinal cord injury in the Rhône-Alpes region, France, 1970-75. *Paraplegia* 1978-79;16:76-87.
41. Soopramanien A. Epidemiology of spinal injuries in Romania. *Paraplegia* 1994;32:715-22.
42. Exner G, Meinecke F-W. Trends in the treatment of patients with spinal cord lesions seen within a period of 20 years in German Centres. *Spinal Cord* 1997;35:415-9.
43. Chen H-Y, Chen S-S, Chiu W-T et al. A nationwide epidemiological study of spinal cord injury in geriatric patients in Taiwan. *Neuroepidemiology* 1997;16:241-7.



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BLADDER MANAGEMENT

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SUMMARY

Spinal cord lesioned persons suffer a significant risk for developing urinary tract complications, thus regular surveillance of bladder and kidney function is highly indicated. Investigations should include ultrasound of the kidney and urinary tract or i.v. pyelography, renography and Cr-EDTA-clearance, and ultrasound for residual urine (RU). RU should preferably be below 50 mL. Urodynamic investigation are performed, and a leakage pressure below 40 cm H₂O, and a maximal pressure below 60 cm H₂O are aimed at. With continuing bladder dysfunction the urinary tract should be investigated at least every second year. Bacteriuria is only to be treated with antibiotics if symptoms on urinary tract infection occur. Intermittent catheterisation is the preferred method of bladder emptying. Permanent catheter should be avoided whenever possible. Pharmacological treatment includes parasympatholytics for detrusor hyperreflexia, adrenerge α -receptor blocking agents are used to decrease the urethral resistance, including detrusor sphincter dyssynergia. Botulinum toxin injected into the external urethral sphincter and the bladder wall has been successfully applied for treatment of detrusor sphincter dyssynergia and detrusor hyperreflexia. Various operations are possible, but irreversible procedures should be avoided when possible due to development of new treatment options including neuromodulation.

Until the mid 1970s renal failure caused between 5% and 58% (mean 30%) of the deaths in spinal cord lesioned (SCL) persons, and renal failure and other urinary tract complications were the overall most frequent causes of death in these patients (1,2). However, the treatment modalities of the urinary tract problems have improved, and the mortality due to related complications consequently declined; yet problems associated with the urinary tract, including urinary tract infection (UTI) and incontinence, are still of importance for many SCL individuals, and may frequently lead to significant morbidity.

BACKGROUND

Neurogenic bladder dysfunction can be divided into two main types (3): *Suprasacral neurogenic bladder*, which imply, that the reflex arc between the spinal cord segments S2-4 and the bladder is intact (parasympathetic innervation). Therefore the bladder can have active detrusor contraction, which can be elicited by reflex activity, however hyperreflexia with incontinence may often occur. At the same time there is a risk for detrusor sphincter dyssynergia (DSD) giving rise to infravesical obstruction and high intravesical pressure. *Infravesical neurogenic bladder* implies that the connection between the spinal cord segments S2-4 and the bladder are interrupted. Then the detrusor cannot contract, and is flaccid with a risk for over distension.

INVESTIGATIONS

During the initial admission either ultrasound of kidney and the urinary tract or i.v. pyelography should be performed for evaluation of the kidneys and the upper urinary tract morphology and possible pathology. For evaluation of the individual kidney function, and the outflow renography is carried out. Cr-EDTA-clearance is performed for evaluation of the total kidney function (4). Ultrasound for residual urine (RU) is a quick investigation. The RU should preferably be below 50 mL, although the absolute magnitude is not critical as long as the upper urinary tract is normal and there are no bladder emptying problems or infection.

Urodynamic investigation is performed for evaluation of the bladder and urethral function. The leakage pressure should be below 40 cm H₂O, and the maximal pressure below 60 cm H₂O (5). With continuing bladder dysfunction the urinary tract should be investigated at least every second year, due to the risk of development of urinary stones and high bladder pressures. Lifelong follow-up of the urinary tract is necessary, as the SCL person due to impaired sensibility do not have the same possibilities to perceive symptoms, which - in neurologically intact persons - might indicate a possible deleterious state. The investigations may consist of ultrasound of the kidneys and urinary tract or X-ray of the urinary tract to exclude urinary stones, and renography to evaluate kidney and outlet function. If only one kidney is functioning or an artificial bladder-reservoir or other urinary diversion has been performed, the investigations should be carried out every year and a Cr-EDTA-clearance added. Other investigations are performed when needed (4).

THE INITIAL PERIOD

If there is not normal bladder emptying, the bladder should if possible be emptied by sterile catheterisation at intervals securing bladder-fillings of no more than 500 mL (6,7). The fluid intake should be regulated corresponding to 4-5 times of bladder emptying per day. There may be a requirement for measuring of the diureses or the patient may have polyuria, and a permanent catheter may be needed.

Bacteriuria is common with SCL. It is therefore important that antibiotics are only used when symptoms on UTI occur, e.g. fever, increased incontinence, spasticity or reflex sweating, difficulty in bladder emptying, or unclear or malodorous urine (3, 6).

REHABILITATION PERIOD

BLADDER EMPTYING METHOD

Intermittent catheterisation is the preferred method for emptying, because it gives a high assurance of complete bladder emptying with a modest risk of complications (7,8). Provided the emptying is sufficient, this method will often secure that the bladder pressure is sufficiently low in individuals with suprasacral lesions, and incontinence due to hyperreflexia may be avoided. Likewise, for those with infrasacral lesions, overdistension can be prevented.

In a hospital facility sterile intermittent catheterisation (SIC) should be preferred over a nonsterile procedure, as long as the staff is doing the catheterisations, as fewer cases of UTI occur (6). Provided the SCL person has the necessary hand function one should change to clean intermittent self catheterisation (CIC), and in general all SCL persons with bladder emptying

problems should be able to do CIC before discharge.

Reflex bladder emptying may be utilised by individuals with suprasacral lesions, and is typically elicited by suprapubic tapping, but may also be initiated by dilatation of the anal sphincter (7,8). Due to the risk of high intravesical pressure during reflex bladder emptying, it is important to secure acceptable pressure during emptying, and sufficient emptying. If not, CIC should usually be advised.

Abdominal pressure or Credé manoeuvre are used primarily by SCL individuals with infrasacral lesions (7). This emptying method is discouraged due to the risk that the applied pressure over the bladder may result in vesico-ureteral reflux.

Permanent catheter should be avoided due to the risk of developing UTI (3,7-9). The use of an indwelling catheter, either urethral or suprapubic, is virtually always associated with bacteriuria within two weeks (6,7). In addition it predisposes to urinary calculi and bladder carcinoma, and a urethral catheter further increases the risk of local complications, including fistula, epididymitis and scrotal abscess (3,6,7,10). Thus, if it is not possible within a few weeks to avoid a permanent catheter, a suprapubic catheter should be inserted, in order to decrease the risk of urethral complications (3). In some situations permanent catheter may be accepted due to social, physical or psychological reasons (3).

PHARMACOLOGICAL TREATMENT

Detrusor hyperreflexia may be reduced by parasympatolytics, and newer drugs including tolterodine and oxybutynine exhibit a higher uroselectivity, and a more potent treatment (3,7,11). Furthermore, there has been good experience with parasympatoly-

tics for intravesical administration (12). Imipramine can decrease incontinence, partly because of a parasympatolytic effect, though the mechanism is not fully understood (11).

Adrenerge a-receptor blocking agents can decrease the urethral resistance and may be used when the bladder emptying is incomplete, including cases with DSD (3, 7).

If the outlet obstruction may be due to spasticity in the perineal striated muscles spasmolytics like baclofene, tizanidine or dantrolene may be indicated (7).

Intratechal infusion of bachlofene can reduce bladder hyperreflexia and DSD. Likewise intrathecal administration of clonidine seems to reduce detrusor hyperreflexia (13,14).

Botulinum toxin injected into the external urethral sphincter may reduce DSD and decrease the urethral resistance and improve the bladder emptying (13). Botulinum injected into the bladder wall can be used for detrusor hyperreflexia (15).

SURGICAL TREATMENT

Bladder enlargement operations may be indicated in carefully selected cases of severe intractable hyperreflexia with urine incontinence and vesico-ureteral reflux. The procedures are frequently performed as an augmentation enterocystoplasty, where the bladder is opened at the top, and subsequently patched by a detubularized intestinal segment in order to increase the bladder capacity as well as to decrease its ability to contract (16,17). Similar results may be obtained by performing an auto augmentation of the bladder, where a myomectomy of part of the detrusor is performed (7,17).

Rarely, individuals with SCL experiencing insufficient bladder emptying, yet not able to perform catheterisation

per urethra, may benefit from supravesical urinary diversion. A number of different techniques may be applied, thus depending on the indication as well as the capabilities of the patient, continent reservoir or incontinent conduit procedures should be carefully selected (17,18). No matter which type of diversion is chosen, these procedures imply ureteral implantation into a bowel segment, which is excluded from the intestinal continuity. The bowel may thus serve as a simple conduit ending in a wet ostomy (e.g. Bricker conduit), or the bowel segment is used to construct a reservoir, which is connected to a dry ostomy via a continent valve mechanism (e.g. Indiana pouch). A reservoir is emptied by CIC. Between the emptyings a little plaster may be placed over the opening. This method is today found more cosmetic acceptable than a Bricker conduit. The continent reservoirs may give rise to disturbances of the electrolytes with metabolic acidosis and stone-formation.

Vesico-appendico-cutaneostomia is an operation where the appendix is used to make a connection between the bladder and the abdominal skin, e.g. in the umbilicus. The SCL individual will then be able to use CIC through this canal. This operation is often combined with a bladder enlargement and sometimes with simultaneous closure of the urethra (7). This solution may for example be the choice for women who have difficulty in CIC per urethram due to adiposity or immobility (19,20). In case of hyperreflexia with DSD and high intravesical pressure, which cannot be lowered satisfactorily by pharmacological treatment, surgical relief of an increased outlet resistance may be indicated. This has commonly been obtained by a sphincterotomy, how-ever

there is a significant incidence of treatment failure and recurrence, and the complications following the procedure is not negligible (6). Furthermore, the procedure is irreversible. Alternatively, the use of a permanent stent to bypass the sphincteric area has been introduced, and this procedure seems as efficient as the conventional external sphincterotomy, and in addition is reversible (7,21).



SCL individual controlling bladder emptying with a sacral anterior root stimulator.

Sacral-anterior-root-stimulation may be considered in SCL individuals with preserved detrusor-function, high intravesical pressure, large RU and recurrent UTI. This technique implies implantation of electrodes at the anterior S₂₋₄ nerve roots and tunnelling of the cables to the abdomen, where a radio receiver is implanted subcutaneously (7). The SCL person will then with an external transmitter be able to control the detrusor contractions and hereby the bladder emptyings.

Artificial sphincter can in selected individuals be a possibility against incontinence (7).

INCONTINENCE AIDS

Incontinence aids for SCL persons are of great importance as the incontinence can be a significant barrier for social interaction (22,23). However, the men-

tioned treatments often have to be supplemented with one or more aids. Diapers are available in many sizes and forms to be used for different purposes.

For men penile-bags of various types, including small diaper-bags for less severe incontinence, and urisheaths (condom catheter) connected to a leg-bag for larger leakage, are available. Use of urisheaths implies a risk for ulcer formation, why the skin should be inspected.

Women have unfortunately no effective urine collecting aid, but some with less incontinence may use mechanically aids to close the urethra (24). However, if hyperreflexia is present these devices are frequently insufficient.

TREATMENT COMPLIANCE

Impaired bladder function caused by SCL may impact multiple areas of quality of life, including an individual's ability to create and maintain social relationships (23). Major factors which may impair the persons individual technique of bladder management may be worry about incontinence, or the devices ability to control voiding, difficulties and increased time demand for bladder care and complications. Consequently, attention should also be addressed to the SCL person's compliance with bladder management, and it is mandatory, that a carefully tailored bladder management program be implemented for each individual (22,23).

COMPLICATIONS

The kidney function should be followed closely and, if deteriorating, additional investigations of the bladder, in particular the intravesical pressure and the outlet conditions, should be performed.

UTI is the most frequent complication. In case of recurrent infections it is important to assure, that there is no significant RU after bladder emptying or urinary stones as background for the infections. On the same time the urine in individuals who perform CIC will in approx. 50% of the instances show bacteriuria. Therefore one should only treat with antibiotics if there are symptoms. When antibiotic treatment is necessary antibiotics with little or no influence on the normal flora should be used. Single agent therapy - in accordance with anti-microbial susceptibility test - is preferred. It is advised that the treatment is extended to at least 5 days, and in case of re-infection or relapsing UTI at least 7-14 days, depending on the severity of the infection (3,6).

Regarding prevention of UTI general cleanliness and local hygiene is important. With recurrent UTI without treatable structural or functional causes, i.e. urinary stones, outlet difficulties, RU, or high bladder pressure, may long-term low-dose antibiotics be the solution. This option should not be used for SCL persons with permanent catheter, due to the risk of developing resistant bacteria. Permanent catheter is the greatest risk for UTI in SCL individuals (6). It seems that cranberry products may prevent UTI, but there is a need for appropriate placebo-controlled trials to confirm this (25).

Urinary stones in the bladder as well as in the kidneys occur in 5-10% of SCL individuals over a period of 5-10 years (26). Treatment of these and structural risk factors follows general urological principles from non-SCL patients

FUTURE

At the present time promising developments are underway in the area of

neuromodulation, including experiments with electrical stimulation and penile vibration to decrease the bladder hyperreflexia and increase the bladder capacity (12-14,27). These new possibilities may have great importance for many SCL persons, who suffer from detrusor hyperreflexia with incontinence. Irreversible procedures thus seems to be inappropriate in most patients for the time being, due to the possibilities for multimodal treatment including local (intravesical or intrathecal) and oral administration of drugs in combination with mechanical (urethral stent) and electrical or other means of neuromodulation (13).

REFERENCES

1. Hartkopp A, Brønnum-Hansen H, Seidenschner A-M, Biering-Sørensen F. Survival and cause of death after traumatic spinal cord injury. A long-term epidemiological survey from Denmark. *Spinal Cord* 1997;35:76-85. [Corrigendum *Spinal Cord* 1997;35: 862-864].
2. Selzman AA, Hampel N. Urologic complications of spinal cord injury. *Urol clin NA* 1993;20:453-464.
3. Burns AS, Rivas DA, Ditunno JF. The management of neurogenic bladder and sexual dysfunction after spinal cord injury. *Spine* 2001; 26;suppl 24:S129-36.
4. Linsenmeyer TA, Culkin D. APS recommendations for the urological evaluation of patients with spinal cord injury. *J Spinal Cord Med* 1999;22:139-42.
5. McGuire EJ, Woodside JR, Borden TA, Weiss RM. Prognostic value of urodynamic testing in myelodysplastic patients. *J Urol* 1981;126:205-9.
6. Biering-Sørensen F, Bagi P, Høiby N. Urinary tract infections with spinal cord lesions: Treatment and prevention. *Drugs* 2001;61:1275-87.
7. Jamil F. Towards a catheter free status in neurogenic bladder dysfunction: a review of bladder management options in spinal cord injury (SCI). *Spinal Cord* 2001;39:355-61.
8. Weld KJ, Wall BM, Mangold TA, Steere EI, Dmochowski RR. Influences on renal function in chronic spinal cord injured patients. *J Urol* 2000;164:1490-3.
9. Weld KJ, Graney MJ, Dmochowski RG. Differences in bladder compliance with time and associations of bladder management with compliance in spinal cord injured patients. *J Urol* 2000;163:1228-33.
10. West DA, Cummings JM, Longo WE, Virgo KS, Johnson FE, Parra RO. Role of chronic catheterization in the development of bladder cancer in patients with spinal cord injury. *Urology* 1999;53:292-7.
11. Wein AJ, Rovner ES. Pharmacological management of the overactive bladder. *Contemp Urol* 2001;13:suppl 6:22-35
12. Sullivan J, Abrams P. The overactive bladder: neuropharmacological basis of clinical management. *Curr Opin Obstet Gynecol* 1999;11:477-
13. Yoshimura N, Smith CP, Chancellor MB, de Groat WC. Pharmacologic and potential biologic interventions to restore bladder function after spinal cord injury. *Curr Opin Neurol* 2000;13:677-83.
14. Madersbacher HG. Neurogenic bladder dysfunction. *Curr Opin Urol* 1999;9:303-7.
15. Schurch B, Stöhrer M, Kramer G, Schmid DM, Gaul G, Hauri D. Botulinium-A toxin for treating detrusor hyperreflexia in spinal cord injured patients: A new alternative to anticholinergic drugs? Preliminary results. *J Urol* 2000;164:692-7.
16. Nomura S, Ishido T, Tanaka K, Komiya A. Augmentation ileocystoplasty in patients with neurogenic bladder due to spinal cord injury or Spina Bifida. *Spinal Cord* 2002;40:30-3.
17. Wein AJ. Neuromuscular dysfunction of lower urinary tract. In: Walsh PC, Retik AB, Darracott Vaughan Jr E, et al., eds. *Campbell's Urology*. Philadelphia: Saunders, 1998: 953-1006.

18. McDougal WS. Use of intestinal segments and urinary diversion. In: Walsh PC, Retik AB, Darracott Vaughan Jr E, et al., eds. *Campbell's Urology*. Philadelphia: Saunders, 1998:3121-61.
19. Hakenberg OW, Ebermayer J, Manseck A, Wirth MP. Application of the Mitrofanoff principle for intermittent self-catheterization in quadriplegic patients. *Urology* 2001;58:38-42.
20. English SF, Pisters LL, McGuire EJ. The use of appendix as a continent catheterizable stoma. *J Urol* 1998;157:747-9.
21. Chancellor MB, Bennett C, Simoneau AR, Finocchiaro MV, Kline C, Bennett JK, et al. Sphincteric stent versus external sphincterotomy in spinal cord injured men: Prospective randomized multicentre trial. *J Urol* 1999;189:3-8.
22. Yavuzer G, Gök H, Soygür T, Arikan N, Arasil T. Compliance with bladder management in spinal cord injury. *Spinal Cord* 2000;38:762-5.
23. Hicken BL, Putzke JD, Richards JS. Bladder management and quality of life after spinal cord injury. *Am J Phys Med Rehabil* 2001;80:916-22.
24. Thyssen H, Bidmead J, Lose G, Møller Bek K, Dwyer P, Cardozo L. A new intravaginal device for stress incontinence in women. *BJU Int* 2001;88:889-92.
25. Biering-Sørensen F. Urinary tract infection in individuals with spinal cord lesion. *Curr Opin Urol* 2002;12:45-9.
26. Biering-Sørensen F, Nielans H-M, Dørflinger T, Sørensen B. Urologic situation five years after spinal cord injury. *Scand J Urol Nephrol* 1999;33:157-61.
27. Læssøe L, Sønksen J, Bagi P, Biering-Sørensen F, Ohl DA, McGuire EJ, Kristensen JK. Effects of ejaculation by penile vibratory stimulation on bladder reflex activity in a spinal cord injured man. *J Urol* 2001;166:627.

BOWEL MANAGEMENT

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SUMMARY

Colorectal dysfunction is reported by nearly 40% of spinal cord lesioned (SCL) individuals. Faecal incontinence and constipation are major issues. In the acute phase there is an increased risk of paralytic ileus. Whenever possible continuous enteral nutrition should be maintained. Initial intestine is atonic and digital evacuation and enema may be necessary for bowel evacuation. Due to the risk of constipation the use of morphine etc. should be avoided. It is often necessary to add laxatives, which soften the stool, e.g. lactulose and magnesiumoxide, or increase the volume, e.g. fleawort seed. Laxatives with effect on the motility may in case of constipation, e.g. bisacodyl and natriumpicosulfate. With more severe constipation enterokinetic drugs may be used, e.g. Cisapride. Digital stimulation of the anal canal or digital evacuation is used regularly. Rectal laxatives include the lubricating and softening enemas, which are used hours before bowel evacuation, and contact-laxatives, as suppositories or enemas they stimulate the defecation reflex after 5-20 minutes. Retrograde colonic wash-out may be performed with an enema continence catheter. Malone antegrade continence enema is another possibility. Colostomy may be the choice of surgical procedure. Haemorrhoids and mega colon are frequent complications.

Among 424 spinal cord lesioned (SCL) individuals 39% reported that colorec-

tal dysfunction caused some or major restrictions on social activities or on their quality of life, and 30% regarded colorectal complaints to be worse than both bladder and sexual dysfunction. In addition, faecal incontinence was experienced by 75%, but the majority only reported a few episodes each month (15%) or each year (56%) (1). Another study reported that incontinence affected the quality of life for 62% of SCL individuals, compared to 8% in the control population (2). Furthermore only 19% felt a normal desire to defecate, whereas the remaining felt no desire to defecate (38%) or a combination of abdominal discomfort (37%) and headache, physical uneasiness, and perspiration (25%) (1).

ACUTE MANAGEMENT

In the acute phase of a spinal cord injury, the patient has an increased risk of paralytic ileus. It is important whenever possible to maintain continuous enteral nutrition. This seems to improve the integrity of the intestinal mucosa and may decrease the risk of septicaemia and reduce the morbidity and mortality. Intravenous nutrition may have to be used.

As long as the intestine is atonic digital evacuation and enema may be necessary for the bowel evacuation. Otherwise there should be initiated a regular bowel management program as soon as possible.

Due to the risk of constipation the use of morphine and similar drugs should be restricted as much as possible.

BOWEL TYPES

In SCL individuals with upper motor neuron bowel or reflex-bowel, the reflex arc between the sacral spinal cord and the bowel is intact (3,4). Therefore the bowel evacuation may be initiated reflexogenic, e.g. by stretching of the anal sphincter digitally, or by 'tapping' over the bowel abdominally. The striated external anal sphincter will usually be tightly closed with these spinal cord lesions due to spasticity of the pelvic floor. The spastic sphincter may contribute to the occurrence of faecal retention.

In SCL individuals with a lower motor neuron bowel, including cauda equina lesions the colon is 'relaxed' without spinal cord mediated reflex peristaltic, which may imply development of constipation (3,4). Furthermore there is a flaccid anal sphincter, resulting in a risk of faecal incontinence. Therefore the bowel content should not be too soft. SCL individuals with these lesions on the other hand have the possibility to make use of abdominal pressure or Valsalva manoeuvre during the bowel evacuation.

In a study comparing SCL individuals with upper and lower motor neuron bowels it was found, that those with upper motor neuron bowel demonstrated increased frequency of defecation, increased frequency of faecal incontinence, increased use of oral medications for bowel care, increased required time for defecation and more diet modification than those with lower motor neuron bowel. However, there was no difference in the subjective difficulty of bowel care (5).

BOWEL EVACUATION

The procedures for bowel evacuation should aim for, that no faecal incontinence appears, simultaneously it

should not be too time-consuming and constipation should be avoided. Constipation may be difficult to avoid due to the missing desire to defecate, the general immobilisation and the paralysis of the abdominal and perineal muscles. The colonic transit time is increased in SCL individuals, in particular if the lesion is above T1 (6). Therefore one should also be aware of diarrhoea, which may be paradoxical and be caused by the constipation. Furthermore the SCL often imply that the voluntary ability to contract the external anal sphincter is abolished, which in combination with missing anorectal sensibility increases the risk of incontinence.

To avoid constipation the faeces should be voluminous and wet. It is therefore important from the beginning to make sure that the SCL individual has a diet rich on fibres (fruit, vegetables, and coarse bread) and sufficient liquid. It has also to be observed that increasing dietary fibre in SCL individuals not necessary has the same effect on the bowel function as in normally functioning bowels. Indeed the effect may be the opposite to that desired (7).

It is often necessary to add laxatives. Also, it may be for some SCL individuals an advantage to regulate the bowel evacuation to appear according to a scheduled time plan, not least if help is needed for the bowel management. The most optimal time will often be in the morning shortly after breakfast, as this stimulates the gastro colic reflex, which causes increased colon motility about 30 min. after the meal. In some patients colonic massage from the ascending colon towards the rectum may be of help. Others may use digital evacuation.

Either digital stimulation of the anal

canal before defecation or digital evacuation of the rectum was used regularly by 65% (1).

LAXATIVES

In a large questionnaire study laxatives were used by 39% of SCL individuals, compared to 4% of normal controls (2).

ORAL AGENTS

Laxatives can be divided in those primarily functioning on the bowel content, i.e. its consistency and volume, and those with primary effect on the intestinal motility.

Laxatives with function on the bowel content include agents, which soften the stool, e.g. lactulose and magnesium oxide, and those, which increase the volume, e.g. fleawort seed. By increasing the volume of the bowel content the peristalsis is stimulated. This will imply evacuation of more loose faeces.

Laxatives with primary effect on the intestinal motility should first of all be utilized in case of acute constipation. If the constipation is chronic they may be used in smaller doses distributed over the day. This could be, e.g. bisacodyl and natriumpicosulfate. In patients with more severe constipation treatment with enterokinetic drugs may be used. The effect of Cisapride has been investigated in two studies (8,9). Cisapride appears to shorten transit time in the left sided colon and about 50 % of the patients reported a subjective improvement in constipation. In a recent study a newly developed prokinetic drug Prucalopride was investigated in a controlled study (10) and a significant decrease in constipation symptoms and colonic transit time was found. Prokinetic drugs may become an important adjunctive for the treatment of constipation in SLC individuals.

RECTAL AGENTS

Rectal laxatives include the *lubricating and softening enemas*, which are used hours before bowel evacuation with the purpose of lubricating the accumulated stools in the rectum, and *contact-laxatives*, which as suppositories or enemas by contact with the mucosa stimulate the defecation reflex after 5-20 minutes.

RETROGRADE COLONIC

WASH-OUT

Retrograde colonic wash-out may be performed with an enema continence catheter positioned in the rectum and held there by a balloon filled with air. The catheter is through a tube connected with a bag containing ~1 L lukewarm water. When the bag is elevated the water runs into the bowel, where it stays for approximately a quarter of an hour. Thereafter the balloon is deflated and the catheter is removed and the bowel is emptied for the instilled water.

This method has proven to be effective in selected adult individuals with neurogenic bowel dysfunction, particular to prevent faecal incontinence while constipation was less successfully treated (11). In children with Spina Bifida the success rate seems even higher. In the adult SCL individuals there are major problems with retaining the balloon in the rectum when instilling the water.

Pulsed irrigation evacuation has been tried for the clearing of faecal impaction. It was found to be safe and effective (12), and may in the future become a useful addition to other methods in the management of the neurogenic bowel.

SURGICAL TREATMENT

Malone antegrade continence enema is a

procedure where the appendix vermiformis is lead to the abdominal wall where a small stoma is made. A tube connected with a bag or a large syringe can now be inserted in the stoma and the colon may be flushed in the antegrade direction with ~1 L lukewarm water. The procedure takes all together approximately half an hour. It is enough to cover the stoma with a plaster between the wash-outs. The surgical procedure is minor and reversible, and the procedure is thus a suitable alternative to more extensive procedures (11)

Colostomy. The choice of surgical procedure should reflect the clinical problem. Patients with colon inertia will do better with an ileostomy and segmental resection of the colon should be avoided. SCL individuals with rectal or sigmoid dysfunction and pelvic dysfunction may do very well by diversion of the descending colon. Thus, in a study of 26 SCL individuals with colostomy and 26 without, those with colostomy were no worse off in regard to quality of life, than those without. The inference is that perhaps a colostomy should be considered earlier in SCL individuals with major bowel dysfunction following SCL (13), which is supported by another study, where 83% said their independence had increased after colostomy, and 92% wished colostomy had been offered earlier, and none wanted their colostomy reversed (14).

Sacral Anterior Root Stimulator has showed in a large segment of the SCL individuals implanted that it is used for bowel evacuation with success. (15). Therefore at the present time a development work is in progress to improve this technique for bowel evacuation.

FUNCTIONAL MAGNETIC STIMULATION

Recently functional magnetic stimulation (FMS) as a non-invasive method to stimulate the colon in SCL individuals has been evaluated. It was found that FMS is able to stimulate the colon and reduce the colonic transit time, thus this method may in the future be one possible option (16).

COMPLICATIONS

Beside constipation, and the prolonged time needed for bowel evacuation, haemorrhoids may be a significant and recurring problem for SCL individuals. They should be treated in the usual way. Haemorrhoidectomy has been found to be more common in SCL individuals compared to the normal population, 9% vs. 1.5%, particularly among those requiring manual evacuation (2). A feeling of abdominal distension is not unusual. One of the reasons is that many SCL individuals not are able to evacuate flatus in the normal way as they are sitting very heavy most of the day. In addition they don't have the normal feeling or desire to evacuate flatus. In such instances it may be advantageous to have bowel evacuation daily.

Megacolon was found in 73% of 128 consecutively admitted SCL inpatients, and in 52% of these associated radiological constipation was found. Independent correlates of megacolon were more than 10 years elapsed since acute injury, age over 50 years, and use of ≥ 4 laxative doses per month (17).

REFERENCES

1. Krogh K, Nielsen J, Djurhuus JC, Mosdal C, Sabroe S, Laurberg S. Colorectal function in patients with spinal cord lesions. *Dis Colon Rectum* 1997;40:1233-9.

2. Lynch AC, Wong C, Anthony A, Dobbs BR, Frizelle FA. Bowel dysfunction following spinal cord injury: a description of bowel function in a spinal cord-injured population and comparison with age and gender matched controls. *Spinal Cord* 2000;38:717-23.
3. Clinical practice guidelines: Neurogenic bowel management in adults with spinal cord injury. Spinal Cord Medicine Consortium. *J Spinal Cord Med* 1998;21:248-93. www.pva.org, under publications.
4. Stiens SA, Bergman SB, Goetz LL. Neurogenic bowel dysfunction after spinal cord injury: Clinical evaluation and rehabilitative management. Focused review. *Arch Phys Med Rehabil* 1997;78:S-86-102.
5. Yim SY, Yoon SH, Lee IY, Rah EW, Moon HW. A comparison of bowel care patterns with spinal cord injury: upper motor neuron bowel vs lower motor neuron bowel. *Spinal Cord* 2001;39:204-7.
6. Krogh K, Mosdal C, Laurberg S. Gastrointestinal and segmental colonic transit times in patients with acute and chronic cord lesions. *Spinal Cord* 2000;38:615-21.
7. Cameron KJ, Nyukasi IB, Collier GR, Brown DJ. Assessment of the effect of increased dietary fibre intake on bowel function in patients with spinal cord injury. *Spinal Cord* 1996;34:277-83.
8. Geders JM, Gaing A, Bauman WA, Korsten MA. The effect of cisapride on segmental colonic transit time in patients with spinal cord injury. *Am J Gastroenterol* 1995;90:285-9.
9. Longo WE, Woolsey RM, Vernava AM, Virgo KS, McKirgan L, Johnson FE. Cisapride for constipation in spinal cord injured patients: a preliminary report. *J Spinal Cord Med* 1995; 18:240-4.
10. Krogh K, Jensen MB, Gandrup P, Laurberg S, Nilsson J, Kerstens R, De Pauw M. Efficacy and tolerability of prucalopride in patients with constipation due to spinal cord injury. *Scand J Gastroenterol* 2002;37:431-6.
11. Christensen P, Kvitzau B, Krogh K, Buntzen S, Laurberg S. Neurogenic colorectal dysfunction – use of new antegrade and retrograde colonic wash-out methods. *Spinal Cord* 2000;38:255-61.
12. Puet TA, Jackson H, Amy S. Use of pulsed irrigation evacuation in the management of the neuropathic bowel. *Spinal Cord* 1997;35:694-9.
13. Randell N, Lynch AC, Anthony A, Dobbs BR, Roake JA, Frizelle FA. Does a colostomy alter quality of life in patients with spinal cord injury? A controlled study. *Spinal cord* 2001;39:279-82.
14. Kelly SR, Shashidharan M, Borwell B, Tromans AM, Finnis D, Grundy DJ. The role of intestinal stoma in patients with spinal cord injury. *Spinal Cord* 1999;37:211-4.
15. Creasey GH, Grill JH, Korsten M, Sang U H, Betz R, Anderson R, Walter J. An implantable neuroprosthesis for restoring bladder and bowel control to patients with spinal cord injuries: A multicenter trial. *Arch Phys Med Rehabil* 2001;1512-9.
16. Lin VW, Nino-Murcia M, Frost F, Wolfe F, Hsiao I, Perkas I. Functional magnetic stimulation of the colon in persons with spinal cord injury. *Arch Phys Med Rehabil* 2001;82:167-73.
17. Harari D, Minaker KL. Megacolon in patients with chronic spinal cord injury. *Spinal Cord* 2000;38:331-9.

SKIN - DECUBITUS

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SUMMARY

Spinal cord lesioned individuals comprise a group of persons with the highest risk of pressure ulcer development. The single most important etiological factor is excessive and/or prolonged pressure. Other factors include incontinence, inferior nutritional status, alcohol and drug abuse and other psychosocial factors. Prevention, through identification and elimination of risk factors, positioning and pressure relief is therefore of paramount importance. Following pressure ulcer development, treatment can be either conservative or surgical. The generally complex medical nature and needs of these patients and the high recurrence rate of pressure ulcer, emphasizes the need of a vigorous multidisciplinary team approach in treatment and post-operative rehabilitation.

PREVALENCE

Due to the neurological deficit with impaired or lost sensibility and mobility and to certain psychosocial circumstances, spinal cord lesioned (SCL) individuals comprise a group of individuals with the highest risk for development of pressure ulcers (1,2,5).

Studies from the United States have reported a 30-40% prevalence of pressure ulcers during the acute care phase and rehabilitation period following Spinal Cord Injury (1).

Treatment initiated in state-of-the-art care centres (Model System Centres) within 72 hours of injury was associated with a significantly lower preva-

lence - and with development of less severe pressure ulcers than treatment in Non-System Centres (1).

In community resident SCL individuals, one third were found to have pressure ulcers (2). Based on a study of 4,065 SCL individuals from the National Spinal Cord Injury Statistical Centre, 15% had a pressure ulcer at their first annual follow-up examination; 20% at year 5; 23 % at year 10; 24% at year 15, and 29% at year 20 (1).

The recurrence rate of pressure ulcers, irrespective of medical or surgical treatment, has been found to be around 31-35% (1,5).

Amongst SCL individuals 7-8% will die from pressure ulcer-related complications (3,4).

GRADING

The table below shows the clinical stages of pressure ulcers.

PATHOPHYSIOLOGY

Forces between the points of contact with the environment generate stress, designated *pressure* when forces are perpendicular to, and *shear* when they are tangential to the surface. The response of the tissue to pressure or shear is determined by the viscoelastic and microvascular properties of the tissue (1).

Spinal cord injury produces changes in the structure and physiology of the skin before even the appearance of ulcers. There is evidence of an increase in collagen degradation in skin and muscle, as well as an imbalance in collagen biosynthesis following spinal

Stage	Characteristics
Stage 0	Reactive hyperaemia. Redness disappears on pressure. Circulation intact. The condition is reversible with pressure relief.
Stage 1	Hyperaemia. Redness does not disappear with pressure. Classic inflammatory signs. Initial tissue damage with aseptic inflammatory reaction. The condition is reversible with pressure relief.
Stage 2	Blister formation, eventually ulceration. The lesion does not penetrate dermis. The condition is reversible with pressure relief.
Stage 3	The ulcer extends into the subcutaneous tissue. Necroses, may be undermining or fistulae. The lesion does not penetrate the fascia. Needs active treatment to heal.
Stage 4	The ulcer has penetrated the fascia and down into the muscle and may be the bone. Necroses, undermining and fistulae. Often infected. Needs intensive treatment to heal.

cord injury, rendering these structures highly vulnerable to pressure (1). Compression of skin and muscle (which is highly sensitive to oxygen depletion), compression and deformation of vessels and capillaries, initiates a cascade of events, which ultimately leads to ischemia and tissue necrosis. Also, imbalance in internal interstitial pressure gradients due to repeated stress also induces damage to the colla-

gen network and cellular structures, resulting in deficient metabolism and lack of circulating nutrients. Pressure ulcers usually develop over bony prominences, where tissue pressure is highest. Thus the most obvious exposed pressure points in the supine position are the sacrum, iliac crests, trochanters, elbows, shoulders, shoulder blades, knees, malleolar bones and heels. In the sitting position the exposed sites are the ischial tuberosities, crena ani and the spinal processes. Pressure may also be caused by heterotopic calcifications, and may be influenced by external factors like braces, shoes, folds in the support surface or in the clothing, mode of repositioning (shear), type of wheel chair, psychosocial assistance etc. Due to loss of sensibility, it is also important to be aware of the risk for ulcer formation from contact with warm objects, i.e. bathwater, warm taps, coffee cups, cigarette embers, heating pad, and radiator.

RISK INDICATORS

Various studies have given contradicting information regarding risk indicators for pressure ulcer development in SCL individuals. Demographic factors have included the young as well as the old age groups, male gender, single marital status, and lower educational level. Among the SCL related factors, pressure ulcers have been found more frequently in individuals with more extensive paralyses and completeness of the SCL, longer duration of the SCL, and less functional independence. Physical and psychosocial aspects include lack of responsibility for skin care, poor nutrition, use of tobacco and alcohol, drug abuse, low self-esteem and feeling of dissatisfaction with life and one's activities (1,5).

PREVENTION

Assessment of risk indicators for pressure ulcers in SCL persons increases awareness among professional health care workers for the necessity of prevention strategies and thus, improves clinical practice (1).

It is extremely important to regularly assess and document risk indicators, once these have been identified, as the SCL individual's situation can deteriorate or improve. Risk assessment tools can be applied to support clinical evaluation. Several tests, like the Brandon -, Salzberg -, Norton scales, and the Waterlow Pressure Sore Risk Calculator have been used. However, the predictive value of these tools are imprecise in the SCL population, as certain risk factors known to be of scientific importance are not included (1).

Prevention strategies are aimed at appropriate positioning, relieving pressure exposed sites at regular intervals to allow blood supply to the area, instituting pressure relief on appropriate surfaces and at avoiding prolonged immobilisation.

In wheelchair positioning, postural alignment, balance, weight distribution and pressure reduction capabilities are evaluated. Weight shifts several times every hour should be encouraged. Likewise in the supine position, turns and changing of position should be



Figure 1. Clinitron bed

performed every second hour. Positioning directly on bony prominences or on pressure ulcers should be avoided (1).

Pressure relief should be obtained by using an optimal mattress and cushion for each individual SCL person. With regard to mattresses, a static mattress may often be sufficient.

Alternatively, a dynamic mattress may be chosen when positioning becomes difficult. For high-risk SCL individuals: tetraplegics, simultaneous presence of several ulcers or other injuries, low-air-loss and air-fluidized beds (Clinitron bed – Figure 1) may be employed. Static- and dynamic wheelchair cushions are also available. Bed- and wheelchair support surfaces have to be individually adjusted.

Furthermore, it is important to be attentive of possible pressure exposure from bath chair, toilet seat, lift-sail etc. Foam-padding and special designs may be necessary. Moist skin or soaking, due to moist clothes, sweat, incontinence from urine or faeces, decreases the resistance to pressure, friction damage and bacteria-invasion and should therefore be avoided. Maintaining adequate protein, calorie, and fluid intake to prevent negative nitrogen balance and dehydration is essential (1).

MEDICAL TREATMENT

Following actual development of a pressure ulcer, assessment of the individual with the pressure ulcer is performed, with identification of risk factors and determination of the nutritional status. In addition, the pressure ulcer is assessed, describing anatomic location, the extent of the lesion, margin, the presence of undermining, sinus tracts, necrosis, infection, and bone and joint involvement. Generally, stage 1 and 2 pressure ulcers can be

As the most important etiological factor is unrelieved pressure, the primary treatment is aimed at total relief of the ulcer area, on an appropriate pressure relief mattress, to obtain healing and avoid surgery. Treatment in the community thus implies, that considerable help in the home may be necessary, as the SCL individual often has to lie in bed continuously. The wound should initially be cleaned and dressed at least once daily, depending on the amount of exudation. This should be performed cautiously with gauze or a sponge and normal saline. Necrotic tissue should also be debrided daily. There are innumerable different dressings on the market and the choice of products will vary according to the condition and stage of the pressure ulcer and the preferences of the particular institution. It is important to apply a dressing, which can establish and maintain a moist environment in the ulcer with concomitant dry surroundings and also to monitor that the dressings are satisfactorily placed at all times. Assessment of ulcer healing should be performed weekly and if progress is not confirmed within 2-4 weeks, the reason for this must be analysed and alternative treatment regime should be considered (1).

Several investigations have shown that electrical stimulation around the ulcer can accelerate wound healing. There is however, no scientific evidence that hyperbaric oxygen treatment, sub-atmospheric pressure therapy, laser therapy, ultrasound or application of growth factors has a favourable effect on wound healing (1).

Complications to medical treatment of pressure ulcers include *cellulitis*, *osteomyelitis* and *sepsis*. Common causes of sepsis include *Bacteriodes fragilis*, *Staphylococcus aureus* and gram-neg-

ative rods. Diagnosis can be attained partly clinically, partly by tissue or bone biopsies. These complications should be treated with antibiotics (1).

SURGICAL TREATMENT

In stage 3 and in particular stage 4 ulcers (see table above), surgical intervention is often necessary to shorten wound healing, to prevent osteomyelitis, sepsis, protein loss, secondary amyloidosis and renal failure, *Marjolin ulcer* (development of carcinoma in long-standing ulcers - Figure 2) and to improve hygiene (1,6).



Figure 2. Marjolin ulcer - sacral

Preoperative evaluation, including identification of risk factors (i.e. white blood count, erythrocyte sedimentation rate, haemoglobin, nutritional status, incontinence, medical conditions, smoking, spasms, contractures) and wound care is of paramount importance to ensure success of the operation. Local wound - and urinary tract infections should be treated. Scintigraphy and X-rays and in some cases a CT or MRI scan should be performed when osteomyelitis and heterotopic calcifications or fistulas are suspected. The purpose of surgery is to excise all necrotic tissue and also in most cases, to reconstruct the defect in order to

Figure 3.



Large defect over left ischial tuberosity in a 18 year old paraplegic patient.



A large Hamstring flap has been prepared to cover the superficial defect and a pedicled Gracilis muscle flap for eliminating dead space.



The final result.

obtain sufficient soft tissue covering over the underlying bony prominences. A fascio-cutaneous flap occasionally combined with an underlying muscle flap or preferably a myocutaneous flap, from a neighbouring area, is moved to cover the ulcer with maintenance of the blood supply through a pedicle (Figure 3). Re-section of bony prominences may be indicated. Skin grafts or direct wound closure over pressure bearing areas should be avoided.

The operation with reconstruction comprises revision of the ulcer and closure with a flap either in one procedure or with 4–7 days interval. The ulcer is excised including scar tissue, heterotopic calcifications, bursa, necrotic tissue and bone. Tissue and bone cultures are obtained. The defect is covered with a large pedicled muscle and or fascio-cutaneous flap to eliminate dead space and ensure vascularity for optimising wound healing. Antibiotic treatment is continued for 6–12 weeks in case of osteomyelitis.

Postoperatively, it is necessary with pressure relief to secure healing of the tissue. Usually total pressure relief is maintained for 2-3 weeks and at least 6-8 weeks must be expected before the area can be fully load bearing. If only revision has been performed, total pressure relief has to be maintained until the wound has healed, which can take many months. In the postoperative period it is an advantage if the patient can be placed on a dynamic air-fluidized bed (Clinitron bed – Figure 1). This makes it possible for the patient to lie directly on the flap without compromising wound healing. This is especially important if several flaps are present or for tetraplegics. Rigorous postoperative rehabilitation and an educational programme to teach methods to prevent recurrence is essential following pressure ulcer treatment.

The most common postoperative surgical complication was wound dehiscence (31%) usually caused by deep infection/abscesses or haematoma.

Eleven percent required re-operation, the remainder healed secondary or were grafted with split skin. Recurrent ulcer in the same position was seen in 31% of SCL individuals (5).

REFERENCES

1. Pressure ulcer prevention and treatment following spinal cord injury: A clinical practice guideline for health-care professionals. Consortium for Spinal Cord Medicine. Paralyzed Veterans of America 2000, pp.1-80. www.pva.org, under publications.

2. Fuhrer MJ, Garbor SL, Rintala DH et al. Pressure ulcers in community-resident persons with

spinal cord injury: Prevalence and risk factors. *Arch Phys Med Rehabil* 1993; 74: 1172-7.

3. Yarkony GM. Pressure ulcers: A review. *Arch Phys Med Rehabil* 1994; 75:908-17.

4. Byrne DW, Salzberg CA. Major risk factors for pressure ulcers in the spinal cord disabled: a literature review. *Spinal Cord* 1996; 34:255-63.

5. Schryvers OI, Strane MF, Nance PW. Surgical treatment of pressure ulcers: 20-year experience. *Arch Phys Med Rehabil* 2000; 81: 1556-62.

6. Ellitsgaard N, Hauge EN, Biering-Soerensen. Pressure sore carcinoma in spinal cord injury patients. *Ugeskr for Laeger* 1993; 155: 1473-4.

SEXUAL FUNCTION IN WOMEN; PARTNERSHIP, PREGNANCY AND DELIVERY

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SUMMARY

The majority of spinal cord lesioned (SCL) women had sexual intercourse after the lesion, but describe practical problems. Many of the women are capable of achieving orgasm. SCL women are normally fertile. At pregnancy there is an increased risk of urinary tract infections. The babies are usually delivered vaginally. In case of prolonged labour or severe autonomic dysreflexia caesarean section may be necessary.

It has previously been stated, that there is a lack of studies, comparing the sexuality and sexual functioning of spinal cord lesioned (SCL) women with able-bodied women (1). In spite of this, in recent years, studies have given information on these areas in SCL women.

SEXUAL FUNCTION

In a questionnaire investigation including 472 SCL women with an average age of 32 years at their SCL, 87% reported, that they had had sexual intercourse before their SCL while 67% had had it after the SCL (2). With increasing duration of the injury the number of women reporting to have had sexual intercourse increased from 49% within the first year to 76% for SCL women who were 11 or more years after their SCL. This is in accordance with a study by White et al. (3) where women who had been involved in a physical relationship or intercourse since injury were younger at the time of injury, and those involved in a

physical relationship had been injured for a longer time than those who had not had such a relationship.

Sipski and Alexander (4) found that although intercourse had been the favoured activity pre-injury, kissing, hugging, and touching were the favoured activities post-injury, as given in the following table:

Kissing and hugging	76%
Penis-vaginal intercourse	68%
Touching	68%
Oral sex	56%
Manual stimulation	40%
Vibrator stimulation	24%
Anal intercourse	12%

Depending of the level of the SCL 62%, 70% and 82% respectively, reported to have had sexual inter-course for cervical, thoracic, and lumbosacral SCL. Those women, who had had sexual intercourse, reported the problems given in the table below in relation to intercourse (n=315) (2):

Lack of enjoyment	45,1%
Difficulty with positioning	41,9%
Increased spasticity	26,3%
Lack of lubrication	23,2%
Bladder incontinence	16,5%
Autonomic dysreflexia	11,4%
Problems with Foley catheter	6,7%
Other	8,9%

As reason for not having sexual intercourse since the SCL 155 women reported one or more of the following causes: Not married (16,4%), uncomfortable with their body (16,6%), no opportunities (31,8%), not important (53,6%), and other causes (28,8%).

Although sexual activity continues post-injury, the frequency of sexual activity decreases with only around 50% having sexual activity at least once a week (4,5).

Women that were injured during childhood or early adolescence, i.e. before they have had any sexual experience are usually not described separately, but they constitute a group, which need special attention (6).

ORGASM

Before the SCL 79,1% of the women had experienced orgasm, while 37,3% of the women reported orgasmic ability after the SCL (2). This depended on the time post injury, i.e. 18,0% achieved orgasm within the first year, compared to 49,7% 11 or more years post injury. Among women with cervical SCL 31,7% had achieved orgasm, while the corresponding frequencies were 40,5% and 51,5% for women with thoracic and lumbosacral SCL. Similar results were found by Westgren et al. (7).

When comparing to able-bodied women SCL women had lower levels of sexual satisfaction and sexual drive and higher levels of psychological symptoms and negative affect. In SCL women, sexual satisfaction decreased with age, while it increased with age in able-bodied women (1).

Sipski et al. (8) found, that less than 50% of investigated women with SCL were able to achieve orgasm, compared to 100% of able-bodied women. Only 17% of women with complete infrasacral lesion were able to achieve

orgasm, compared with 59% of those with another SCL. The time to achieve orgasm was considerable longer for women with SCL compared to normal women.

Although physical disabled women compared to able-bodied may have lower level of sexual activity, response, and satisfaction, they have the same sexual desire (9). Furthermore, their description of the orgasm is indistinguishable from that of the able-bodied (8).

Sildenafil citrate (Viagra[®]) in combination with sexual stimulation increased both subjective and physiological parameters for sexual arousal in SCL women compared to sexual stimulation alone (10). Other drugs like prostaglandin E1 and phentolamine may also be of interest (11).

The neurophysiology of the orgasm is not fully elucidated. An intact sacral reflex arc seems to be important (8), but not a necessity. Sensory pass ways that bypass the spinal cord are likely and indicates that there might be different responses to clitoral versus vaginal/cervical stimulation (12).

VAGINAL LUBRICATION

Women with SCL, in particular if they have a complete lesion, should be aware of their vaginal lubrication when sexually aroused. If the vagina is dry it is recommended to use a water-based sliding-crème to avoid irritation or ulcer of the vaginal mucosa during sexual intercourse.

PELVIC FLOOR

Pareses of the pelvic floor due to SCL may be trained using pelvic floor exercises or another voluntary contraction of the musculature. If the SCL has caused paresis and not paralysis of the pelvic floor muscles, then the part

under voluntary control can be trained normally. For SCL women our knowledge about the pelvic floor function is little, but it may be anticipated, that a well functioning pelvic floor is of significance for the sexual function, pregnancy, delivery and prevention of genital prolapse.

MENSTRUATION AND FERTILITY

Usually a SCL does not affect the menstruation cycle. But approximately 60% has post-traumatic amenorrhoea up to 1/2 year (5). The fertility is normal in SCL women (2).

MENOPAUSE

In SCL women the menopause usually occurs earlier than in able-bodied women (2), but only 26% of the post menopausal SCL women were on hormone replacement therapy. After the menopause the frequency of sexual dysfunction is increased. At least some of this is due to atrophy of the vulva and the vagina and can be reversed by local or systemic oestrogen.

PREVENTION

If SCL women want to use prevention, 2nd generation prevention pills with a low oestrogen content are recommended. SCL women have an increased risk of deep venous thromboses, and because p-pills also increase this risk the choice of prevention is adjusted individually. If the frequency of sexual intercourse is low, condom should be considered, and if the women have had the children she wished sterilisation may be an option.

Due to the risk for undiscovered uterus perforation intrauterine devices (IUDs) have generally been used with caution in SCL women. But the LevoNova IUD may be a possibility as it has a high

safety (0.1 pregnancies per 100 women years), menstruation bleedings are reduced and the hormone dose is little. The risk of perforation is reported to 0.1-0.5% of the IUD users.

PARTNERSHIP

In a review it was found that divorce rates after SCL have been reported to be from 8% to 48%, but it appears that the rates tend to decline to the normal for the general population after the initial high-risk period. It was also shown that a SCL person who strives to minimise the impact of the disability on a potential partner makes a more attractive candidate for a long-term relationship than an individual who has come to rely on others (13).

Married SCL women have been found to be no less sexually satisfied than able-bodied women. Partner availability may influence levels of sexual satisfaction (1). It has also been shown that women with disabilities who lived with a significant other reported greater sexual satisfaction and had a higher level of sexual activity (9).

But on the same time women with a disability were more likely to be single and much less likely to bear children than able-bodied women (9).

PREGNANCY

During pregnancy SCL women have common pregnancy complications with the same frequency as other women, but there is an increased risk of urinary tract infections (46%) (2). Furthermore pregnancy in SCL women implies a risk of autonomic dysreflexia (12%), development of pressure ulcer (6%), increased spasticity (12%), problems with transfer towards the end of the pregnancy (11%), etc. (2).

DELIVERY

With complete SCL above Th₁₀ the woman is unable to feel pain during labour, but there may be malaise in the abdomen, reflex sweating, spasms or autonomic dysreflexia (2). Labour may begin without the SCL woman being aware of it. If the lesion is below Th₁₂, there will be normal sensibility for labour.

Premature delivery may occur due to vigorous spasms in the abdominal muscles or in severe cases of autonomic dysreflexia.

The delivery can usually take place vaginally, but should be led by an experienced midwife for the sake of spare the pelvic floor and the sphincters. If the second stage proceeds slowly, there may be a need for assisted delivery by vacuum extraction.

When the delivery does not progress or severe autonomic dysreflexia is present caesarean incision may be necessary, and is performed more frequently in SCL than non-SCL women (2).

If anaesthesia is necessary during the delivery epidural or spinal anaesthesia is primarily used (2). Epidural block may in addition to the pain relieving effect moderate autonomic dysreflexia, spasms and pelvic floor dyssynergy.

LACTATION

Women with SCL are less likely to breast feed their baby post-injury compared to pre-injury (2). The reason for this is unclear, but the process is important and mothers should be encouraged breast feed their babies in order to strengthen the mother-baby relationship. Furthermore the benefits of mothers' milk have been proven beyond doubt.

CHILDREN

It has been verified that there is no dif-

ferences between mothers with SCL and able-bodied mothers, and no difference between children raised in families with mothers with SCL and children raised in families with able-bodied mothers. In addition, no differences were found in dyadic or family functioning with mothers with SCL or able-bodied mothers (14).

INFORMATION

It is to be noticed, that Westgren et al. (7) found, that less than 10% of the women were offered information about sexual function before discharge and none of the partners were offered information. This seems to be an important area to be taken up by all departments working with SCL women.

REFERENCES

1. Black K, Sipski ML, Strauss SS. Sexual satisfaction and sexual drive in spinal cord injured women. *J Spinal Cord Med* 1998;21:240-4.
2. Jackson AB, Wadley V. A multicenter study of women's self-reported reproductive health after spinal cord injury. *Arch Phys Med Rehabil* 1999;80:1420-8.
3. White M, Rintala D, Hart KA, Fuhrer MJ. Sexual activities, concerns and interests of women with spinal cord injury living in the community. *Amer J phys Med Rehabil* 1993;72:372-8. Ref. from Black et al. 1998.
4. Sipski ML, Alexander CJ. Sexual activities, response and satisfaction in women pre- and post-spinal cord injuries. *Arch Phys Med Rehabil* 1993;74:1025-9.
5. Carlifue SW, Gerhart KA, Menter RR, et al. Sexual issues of women with spinal cord injuries. *Paraplegia* 1992;30:192-9.
6. Harrison J, Glass CA, Owens RG, Soni BM. Factors associated with sexual functioning in women following spinal cord injury. *Paraplegia* 1995;33:687-92.

7. Westgren N, Hultling C, Levi R, Seiger Å, Westgren M. Sexuality in women with traumatic spinal cord injury. *Acta Obstet Gynaecol Scand* 1997; 76:977-83.
8. Sipski ML, Alexander CJ, Rosen R. Sexual arousal and orgasm in women: Effects of spinal cord injury. *Ann Neurol* 2001;49:35-44.
9. Nosek MA, Rintala DH, Young ME, et al. Sexual functioning among women with physical disabilities. *Arch Phys Med Rehabil* 1996;77:107-15.
10. Sipski ML, Rosen RC, Alexander CJ, Hamer RM. Sildenafil effects on sexual and cardiovascular response in women with spinal cord injury. *Urology*. 2000; 55 (5) 812-5.
11. Berman JR, Berman LA, Werbin TJ, Goldstein I. Female sexual dysfunction: anatomy, physiology, evaluation and treatment options. *Curr Opinion Urol* 1999;9:563-8.
12. Whipple B, Komisaruk BR. Sexuality and women with complete spinal cord injury. *Spinal Cord* 1997; 35: 136-138
13. Kreuter M. Spinal cord injury and partner relationship. Review. *Spinal Cord* 2000;38:2-6.
14. Alexander CJ, Hwang K, Sipski ML. Mothers with spinal cord injuries: Impact on marital, family, and children's adjustment. *Arch Phys Med Rehabil* 2002;83:24-30.

SEXUAL FUNCTION AND FERTILITY IN MEN

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SUMMARY

We recommend in spinal cord lesioned (SCL) men with erectile dysfunction: If it is possible to obtain a satisfactory erection but of insufficient duration, then try to use a venous constrictor band to find out if this is sufficient to maintain the erection. Otherwise we recommend sildenafil but on the same time to avoid nitrates. If sildenafil is not satisfactory then use intracavernous injection with prostaglandine E1. Some SCL men may prefer cutaneous or intraurethral application. We discourage the implantation of penile prosthesis for the sole purpose of erection. For management of ejaculatory dysfunction we recommend penile vibratory stimulation (PVS) to induce ejaculation as first treatment choice. If PVS fails electro ejaculation (EEJ) is the possibility. Together these options give almost 100% success.

Impaired semen profiles with low motility rates are seen in the majority of SCL men. However, home insemination with semen obtained by PVS and introduced intravaginally in order to achieve successful pregnancies may be an option for some SCL men and their partners. The majority will further enhance their fertility potential when using either PVS or EEJ combined with assisted reproduction techniques such as intrauterine insemination or in vitro fertilization with or without intracytoplasmic sperm injection.

For several decades it was a general

belief that individuals with SCL and cauda equina lesion were permanently and completely impotent and sterile. Fortunately, over time many investigations have proved that this is wrong.

ERECTION

The frequency of erection has been found to be between 54 and 95%, and successful coitus reported obtained in 5-75% (1). Generally is the possibility to obtain erection higher among SCL men with incomplete than complete lesions.

PHYSICAL MANAGEMENT

Erection can be prolonged by use of a venous constrictive band (pubic ring, elastic band, retention band, rubber ring) carefully placed at the root of the penis to trap blood, and thereby maintain penile rigidity for a longer period. It is important to instruct SCL men not to use the band for more than 30 minutes at a time, to avoid ischemic damage.

Artificial penile appliances of plastic material can be of help to some individuals with no or insufficient erection.

Vacuum erection devices in the form of vacuum pumps, either manual or electric, have been used for many years. The device consists of a plastic cylinder into which the flaccid penis is placed and subsequently engorged with blood by producing a vacuum within the cylinder. When adequate erection has been obtained, a venous constrictor band is slid from the base of the device onto the base of the penile shaft to maintain the erection. The cylinder is

then removed from the penis and sexual intercourse may be initiated. As advised above should the venous constrictor band be removed after 30 minutes, and the skin should be closely monitored. SCL individuals in anticoagulation therapy or those with a bleeding disorder should not use this system, as common, but normally insignificant adverse effects include bruises, petechiae and abrasions as well as skin oedema (2).

PHARMACOLOGICAL MANAGEMENT

Sildenafil citrate (Viagra™) promotes erection by inducing smooth muscle relaxation in the corpus cavernosum (3,4). It has been found that the efficacy depends on sparing of either sacral (S₂₋₄) or thoracolumbar (T_{10-L2}) spinal segments (5). This mean absence of both psychogenic (nonsomesthetic supraspinally elicited) and reflexogenic (somesthetic spinally elicited) erections seemed to exclude a successful treatment. On the other hand SCL men with preserved function of at least one component of the erection phenomenon (psychogenic/reflexogenic) responded well to sildenafil (5). Sildenafil should be taken about 20 to 60 minutes prior to intercourse (4).

The first published double-blind, placebo-controlled trials (6,7) have shown good erectile response in 75% of SCL men. In addition, significant improved satisfaction with sex life was reported (6,8,9).

Adverse events seem in SCL men most often to include headache and flushing, but dyspepsia, dizziness, abnormal vision, rhinitis/respiratory tract infections/disorders, diarrhoea and rash have also been reported. These adverse events were generally transient and mild to moderate. None of

the SCL men experienced priapism (5,7). No autonomic dysreflexia symptoms were reported (5).

Co-administration of sildenafil and nitrate-containing medications (nitroglycerine, isosorbide dinitrate, amyl nitrate, etc.) is contraindicated and potentially lethal, because they synergistically potentiate vasodilation and may cause excessive reductions in blood pressure (3,4). This is of particular importance since some SCL individuals may use nitrate-containing medications to treat autonomic dysreflexia. They must be warned about this danger and advised to use nifedipine in stead.

Apomorphine hydrochloride (Upri-*ma*), a dopamin-like molecule, is the first centrally acting agent for erectile dysfunction. The sublingual formulation of apomorphine results in rapid action on erectile dysfunction by enhancement of the natural erectile signals from the brain through the spinal cord. Whether this new agent will be useful in SCL men remain to be examined.

Intracavernous injection of vasoactive drugs like papaverine, phentolamine, and more recent prostaglandine E1 has been used to produce erection (3). In SCL men it is the general experience that the denervated tissues are super-sensitive to the drugs used for intracavernous injection. Therefore the time of erection is longer and the quality of erection is better in men with neurogenic impotence compared with men with vascular impotence. A success rate up to 95% is not uncommon in SCL men. The erection will usually be obtained around 10 minutes after the injection and last for 1/2-6 hours, on the average 2 hours depending on the dose.

To avoid sustained erection (priap-

ism), the SCL men should begin with low doses (papaverine 2-5 mg or prostaglandine E₁ 1-2 µg) and gradually increase the dose if necessary. The SCL men must be instructed to see his doctor or hospital when erection lasts for more than 4-6 hours, to have the erection reversed. Sustained erection may require aspiration of blood from the corpora cavernosa and intracavernosal injection of an α-adrenergic agent. If the male partner is unable to perform the injection himself because of motoric problems, the female partner may be taught the intracavernous injection technique. Disadvantages with injection therapy are the risk of prolonged erection and fibrosis in the corporal tissue.

Cutaneous application. Transdermal nitro-glycerine and topical application of papaverine and prostaglandin E₁ gel to the penis, scrotum and perineum has shown some effect (3).

Intraurethral prostaglandin E₁ (alprostadil, MUSE) showed dissatisfying results in SCL men. Adverse reactions may be pain in the penis, with burning sensation in the urethra and occasionally bleeding from the urethra (10).

SURGICAL MANAGEMENT

Penile prosthesis surgically inserted has been performed in SCL men for a number of years. However, complications such as infection, tissue breakdown, and extrusion of the prosthesis occur in up to half of men with SCL (3). Because of the high risk of complications and the many other possibilities available to obtain erection in SCL men we don't find indication for this kind of surgery today for the purpose of erection only.

Sacral anterior root stimulation (SARS). Brindley and co-workers (11) have demonstrated that SARS for bladder control can also be used to obtain erection. The best root is usually S₂

Induction of erection may not be an indication for this implant in SCL men, but when it is in place it is possible to use it for erection.

EJACULATION

The ability to procreate naturally is lost in the majority of SCL males due to ejaculatory dysfunction and abnormal semen characteristics. The reported ability to ejaculate during sexual stimulation or masturbation range from 0 to 55% (median 15%) (12). The characteristics of semen obtained by several different methods of assisted ejaculation in men with chronic SCL have been consistently reported below normal levels (13), in particular, the sperm motility rates.

In order to solve the problems of ejaculatory dysfunction in men with SCL several methods of assisted ejaculation have been used:

CHOLINESTERASE INHIBITORS

Intrathecal injection and subcutaneous administration of cholinesterase inhibitors may result in ejaculation, but due to many adverse effects, including death due to malignant hypertension and cerebral haemorrhage, with the alternative procedures available it is no more recommended (3).

ELECTROEJACULATION (EEJ)

Rectal EEJ has been used for treatment of ejaculatory dysfunction in SCL men for many years (14). EEJ is carried out with an electrical probe, which is inserted rectally and placed in contact with the rectal wall in the area of the prostate gland and the seminal vesicles. The procedure may require anaesthesia before treatment.

EEJ may be successful in obtaining ejaculate from men with all types of SCL. It is possible to induce ejaculation with EEJ in 80-100% of all SCL men (3).

PENILE VIBRATORY STIMULATION (PVS)

The PVS procedure is performed with the patient placed in the supine or sitting position. The PVS activate the ejaculatory reflex by application of a vibrating disc primary against the frenulum penis for periods of 3 minutes or until antegrade ejaculation occurs. If no ejaculation has occurred the stimulation period is followed by a rest period of 1-2 minutes and stimulation begins again.



FERTI CARE® , Personal vibrator for PVS.

The required time to induce ejaculation by PVS ranges from 10 seconds to 45 minutes (3). The highest rates of ejaculation (antegrade plus retrograde) were seen with a vibrator amplitude level of 2.5 mm and a frequency of 100 Hz. Antegrade ejaculation was seen only in men with cord lesions above T₁₀ (15).

SIDE-EFFECTS

All procedures of assisted ejaculation in SCL men with lesions above T₆ may provoke an acute episode of autonomic dysreflexia, if they are prone to this syndrome. To prevent an episode of autonomic dysreflexia nifedipine is given sublingually 10 to 15 minutes prior to PVS or EEJ in men with SCL above T₆. In general, significant com-

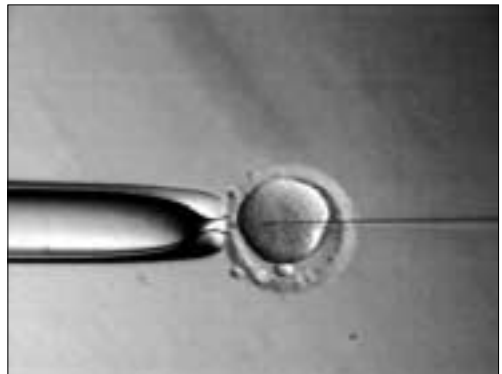
plications from PVS and EEJ are rare. In PVS, local skin abrasion is a common finding but no treatment has been necessary other than a short rest period. In EEJ, there is a potential risk of rectal injury (3).

SURGICAL SPERM RETRIEVAL

If assisted ejaculation procedures fails surgical procedures of sperm retrieval are indicated (13). Successful sperm retrieval from vas deferens and from implanted sperm reservoirs have been reported. Ejaculate through direct stimulation of the hypogastric nerve using an implanted nerve stimulator, and direct spermatozoa aspirated from the testicle or epididymis have also been used. Although the surgical techniques are relatively easy to perform, an effective non-surgical method must be preferable (3).

FERTILITY

Despite the fact that nearly all SCL men are able to produce an ejaculate with PVS or EEJ the ability to procreate is still impaired due to abnormal semen characteristics (12). However, successful pregnancies in partners of SCL men have been reported using either vaginal home insemination or various methods of assisted reproduction techniques (16).



Intracytoplasmic sperm injection (ICSI).

SEMEN CHARACTERISTICS

Nearly all data concerning semen analyses in SCL men are from ejaculates induced by PVS or EEJ and the semen is, in general, characterized by normal to high sperm count and low sperm motility rates. The cause of the problem is not yet determined (3).

PREGNANCIES

HOME INSEMINATION

PVS and vaginal self-insemination performed by the couple at home is a viable option for those SCL men with adequate semen parameters. Most studies reported, that multiple ovulation cycles were used to achieve the home pregnancies and the overall pregnancy rate per couple is 25-61 %. The unique advantage of PVS is the possibility of home use. Furthermore, it will allow the majority of SCL couples to perform the PVS procedure themselves at the hospital when a specimen is required in connection with assisted reproduction techniques (3).

Several successful pregnancies have been reported (3, 16) using spermatozoa obtained by PVS or EEJ combined with assisted reproduction techniques such as intrauterine insemination or in-vitro fertilization with or without intracytoplasmic sperm injection. The overall pregnancy rate per cycle from those studies is about 25%, and this rate is similar to the pregnancy rate per cycle during natural procreation in healthy couples wanting to become pregnant (3).

Several fertility treatment options are available to enhance the reproduction prospects in SCL men and their partners. The proper choice of treatment should be made through coordinated efforts of different specialities, which may involve urology, gynaecology, andrology and rehabilitation. It is also

of importance to inform the couples about possible side effects from hormonal ovulation induction as well as problems related to multiple births.

REFERENCES

1. Biering-Sørensen F, Sønksen J. Penile erection in men with spinal cord or cauda equina lesions. A review. *Seminars in Neurology* 1992;12:98-105.
2. Denil J, Ohl DA, Smythe C. Vacuum erection device in spinal cord injured men: patient and partner satisfaction. *Arch Phys Med Rehabil* 1996;750-3.
3. Biering-Sørensen F, Sønksen J. Sexual function in spinal cord lesioned men. Scientific review. *Spinal Cord* 2001;39:455-70.
4. Monga M, Bernie J, Rajasekaran M. Male infertility and erectile dysfunction in spinal cord injury: A review. *Arch Phys Med Rehabil* 1999;80:1331-9.
5. Schmid DM, Schurch B, Hauri D. Sildenafil in the treatment of sexual dysfunction in spinal cord-injured male patients. *Eur Urol* 2000;38:184-93.
6. Derry FA, Dinsmore WW, Fraser M, Gardner BP, Glass CA, Maytom MC, Smith MD. Efficacy and safety of oral Sildenafil (Viagra) in men with erectile dysfunction caused by spinal cord injury. *Neurology* 1998;51:1629-33.
7. Giuliano F, Hultling C, El Masry WS, et al. Randomized trial of sildenafil for the treatment of erectile dysfunction in spinal cord injury. *Annals of Neurology* 1999;46:15-21.
8. Hultling C, Giuliano F, Quirk F, Peña B, Mishra A, Smith MD. Quality of life in patients with spinal cord injury receiving VIAGRA® (Sildenafil citrate) for the treatment of erectile dysfunction. *Spinal Cord* 2000;38:363-70.
9. Ramos AS, Vidal J, Jáuregui ML, Barrera M, Recio C, Giner M, et al. Efficacy, safety and predictive factors of therapeutic success with sildenafil for erectile dysfunction in patients with different spinal cord injuries. *Spinal Cord* 2001;39:637-43.

10. Bodner DR, Haas CA, Krueger B, Seftel AD. Intraurethral alprostadil for treatment of erectile dysfunction in patients with spinal cord injury. *Urology* 1999;53:199-202.
11. Brindley GS, Rushton DN. Long-term follow-up of patients with sacral anterior root stimulator implants. *Paraplegia* 1990;28:469-75.
12. Sønksen J, Biering-Sørensen F. Fertility in men with spinal cord or cauda equina lesions. A review. *Seminars in Neurology* 1992;12:-106-14.
13. Lundberg PO, Brackett NL, Denys P, Chartier-Kastler E, Sønksen J, Vodusek DB. Neurological disorders: Erectile and Ejaculatory Dysfunction. In Jardin A, Wagner G, Khoury S, Giuliano F, Padma-Nathan H, Rosen R, eds.: *Erectile Dysfunction*. Health Publication Ltd, UK, pp 591-645, 2000.
14. Halstead LS, VerVoort S, Seager SWJ. Rectal probe electrostimulation in the treatment of anejaculatory spinal cord injured men. *Paraplegia* 1987;-25:120-9.
15. Sønksen J, Biering-Sørensen F, Kvist Kristensen J. Ejaculation induced by penile vibratory stimulation in men with spinal cord injuries. The importance of the vibratory amplitude. *Paraplegia* 1994;32:651-60.
16. Sønksen J, Sommer P, Biering-Sørensen F, Ziebe S, Lindhard A, Loft A, Nyboe Andersen A, Kvist Kristensen J. Pregnancies after assisted ejaculation procedures in men with spinal cord injuries. *Arch Phys Med Rehabil* 1997;78:1059-61.

RESPIRATION

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SUMMARY

Respiratory problems and an inability to cough are common in patients with spinal cord lesions (SCL), and the most common cause of death for SCL persons is pulmonary disease, in particular pneumonia. In the initial stage intubation and mechanical ventilation may be necessary. Chest physiotherapy and Continuous Positive Airway Pressure is to be started soon after admission. Tetraplegics with high lesions may need continuing mechanical ventilation or phrenic nerve pacing. Kyphoscoliosis, respiratory exhaustion and sleep problems are complications, which should be looked for.

Life expectancy amongst individuals with spinal cord lesions (SCL) has improved considerably over the last decades. The most common cause of death today for SCL persons is pulmonary disease, in particular pneumonia (1). This illustrates the importance of respiratory function in this population, not only during the early period after SCL, but throughout life.

The more cranial the SCL, the more respiratory muscles – abdominal and thoracic – are paralysed. If the lesion is located in the cervical region then the diaphragm is the only muscle left for respiration. When the cervical lesion is above C₅ the innervation of the diaphragm is also compromised and permanent ventilator support may be necessary.

It has been found that forced vital capacity (FVC), forced expiratory vol-

ume in 1 s (FEV₁) and inspiratory capacity (IC) decrease with ascending SCL from T₁₀. FVC and FEV₁ tended to be larger in the supine than seated position down to T₁. Below this level they were less than when erect. IC was higher with descending injury level. Expiratory reserve volume (ERV) was generally smaller in the supine position and reached highest values in both positions at the T₁₀ level (2).

THE INITIAL PHASE

INTUBATION AND MECHANICAL VENTILATION

The SCL patient must be admitted to a facility where he can be monitored for respiratory exhaustion/complications. Patients not intubated at the scene of accident may need intubation and mechanical ventilation at a later point. Intubation is mandatory when the vital capacity (VC) falls below 10 mL/kg body weight, respiratory rate (RR) rises above 35 or a rise in PaCO₂ occurs with a concomitant fall in blood pH. We have recently shown in 2 patients that intubation of the trachea can be avoided by non-invasive mechanical ventilation via a facemask for up to 5 weeks (Personal observation).

For the anaesthesiologist involved in such patients there are specific concerns: The airway may be difficult to handle because of a stiff neck collar, the presence of full stomach contents, autonomic hemodynamic instability and if several days have passed there is a high risk of fatal cardiac arrhythmias

due to hyperkalemia after the use of suxamethonium.

If mechanical ventilation is to be continued for more than one week, a tracheostomy should be performed. The presence of a recent surgical wound from an anterior stabilising cervical spine operation warrants a delay in performing a tracheostomy due to the risk of infection of the osteosynthesis material.

Mechanical ventilation may in the early stages also be necessary for paraplegics with more caudal SCL. Respiratory muscle paralysis carries a great risk for accumulation of bronchial secretions because of insufficient ability to cough. Therefore, respiratory complications such as atelectasis and pneumonia may develop at any time. Patients at risk for acute respiratory failure are those whose VC is decreasing over time (hours), particularly when VC falls below 1.5 L or if RR is rising and exceeds 30 per minute. At increased RR the patient may still have normal P_aO_2 and P_aCO_2 values for some time in spite of clinical signs of respiratory distress. Therefore, arterial blood gas values should not be used as the only parameter when evaluating impending respiratory failure. It is important to institute treatment, e.g. respiratory support, before changes in blood gas values occur.

VC should be measured as early as possible after admission in all SCL patients with lesions above T₆. Prophylactic chest physiotherapy (CPT) is important until the SCL patient can be mobilised. The rationale behind this is: When the diaphragm contracts and descends in a tetraplegic individual, the chest wall retracts rather than expands. The FVC and the maximal inspiratory force (MIF) are thus acutely decreased by about 70%. As the

spinal shock resolves, paralysed intercostal muscles become spastic and hence the chest wall becomes rigid improving ventilatory function. Five months after injury, the FVC and MIF stabilise at about 60%, while maximum expiratory force remains at 33% of pre-SCL level (3).

CPT should be carried out before insufficient cough and retained secretions are present. Prophylactic use of CPT and CPAP (Continuous Positive Airway Pressure) or PEP (Positive Expiratory Pressure) mask treatment should be initiated as soon as possible after admission, and continued 4-6 times per day – around the clock - to all patients with lesions above T₆, in particular in patients who were heavy smokers before the accident. Also, the presence of rib fractures constitute a major risk for pulmonary complications adding to the inability to cough due to pain or difficulties in titrating effective pain treatment.

WEANING FROM MECHANICAL VENTILATION

The majority of SCL patients on mechanical ventilation will subsequently be extubated and decannulated. In 600 SCL patients, it was found that 11.2% had a tracheostomy performed (4). The median time to decannulation was 31 days. More than 50% had no sequelae, 28% complained about the look of the scar and 21% complained about difficulty in swallowing possibly related to the tracheostomy. Only one patient reported problems that could be related to a possible tracheal stenosis, formerly a dreaded complication of intubation and tracheostomy. Patients with high cervical lesions may need permanent tracheostomy and mechanical ventilation or phrenic nerve stimulation.

THE CHRONIC PHASE

Respiratory problems have a great impact on daily life of many SCL patients. For many tetraplegics the diaphragm is the only functioning respiratory muscle. The VC is generally decreased by approximately one third. Rehabilitation should focus on improvement of the VC and thus cough function.

VC can be improved if the tetraplegics exercise with an inspiratory resistance using a respiratory mask. Employing an expiratory resistance may further increase peak flow, which is possibly of importance for improving cough (5).



Respiratory muscle training mask may improve peak expiratory flow.

This respiratory exercise should be initiated as soon as possible after the SCL and continue life-long.

Paralyses of truncal muscles compromise cough. This implies a high risk for mucous plugging of airways leading to atelectasis and/or pneumonia. It is important that the personnel - and later on the relatives - are taught how to support the cough of a

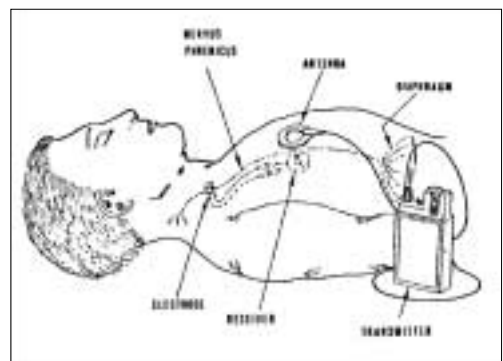
tetraplegic person. This “assisted cough” is performed through the assistance of a helper who uses both hands to apply a sudden pressure inwards and upwards at the upper part of the abdomen. Simultaneously, the tetraplegic person coordinates the breathing by closing and opening the vocal cords in order to use the applied pressure optimally. This technique increases peak flow during the cough and thus helps clearing the airway of secretions.

ELECTRICAL STIMULATION OF RESPIRATORY MUSCLES

Electrical stimulation of the abdominal muscles improves FVC, FEV1, and peak expiratory flow (6,7). Skin electrodes over the paralysed but excitable abdominal muscles are employed. It has been demonstrated that an electrically stimulated cough is not as powerful as a manually assisted cough, as measured by peak flow. On the other hand the SCL patient can independently control the electrically stimulated cough. Multicentre studies of electrical or magnetic stimulation of cough after periods of training of abdominal muscles are underway.

PHRENIC NERVE PACING

Tetraplegics with SCL above C₅ may need respiratory support. Some of these patients are eligible for diaphragmatic pacing using phrenic nerve electrical stimulation (8).



The prerequisite for diaphragmatic pacing is the presence of normal phrenic nerves, diaphragm and lungs. Electrodes are placed around the phrenic nerves, and subsequently tunneled to a radio receiver implanted subcutaneously in the pectoral region. An external, battery-driven transmitter emits coded radio signals via an antenna to the radio receiver. The transmitter controls contractions of the diaphragm and thereby tidal volume and RR. Diaphragmatic pacing increases quality of life. Daily life becomes easier because the patient becomes less dependent on daytime mechanical ventilation. Thus, the tetraplegic patient is able to get around without the need for a respirator on his wheelchair. The psychological impact of such an intervention is obvious. Worldwide, approximately 1500 patients have phrenic pacers implanted. Some patients use pacing day and night, while others prefer to maintain the tracheostomy using a ventilator at night as this may give a more quiet sleep.

HOME VENTILATOR THERAPY

A few SCL individuals having high cervical lesions may go on to permanent ventilatory assistance – some during night only – some for 24 hours a day. In Denmark SCL individuals can be cared for in a private setting. Helpers are hired and trained to do all the chores needed to maintain them in their homes. There are two respiratory care centres in Denmark dedicated to this purpose. Helpers are – without prior education or nursing skills – trained to perform suction of the airways, maintain ventilator equipment and monitor them during sleep. Patients on daytime ventilation have a respirator fitted to the electrical wheelchair. By using uncuffed tracheostomy

tubes they are able to speak freely during ventilator treatment.

LATE RESPIRATORY COMPLICATIONS AND PREVENTIVE MEASURES

Some SCL individuals develop *kyphoscoliosis*, because of paralysis of the trunc muscles, causing significant restrictive lung problems. In turn, this may contribute to the impairment of the ability to cough and thus to recurrent lung infections. Persons at risk should be monitored with regard to this to allow relevant respiratory measures as well as orthopaedic surgical straightening of the deformity to be carried out in time.

Some tetraplegics having used their diaphragm as the sole means to breathe for many years, are found to become *respiratory exhausted* with time – particularly at night – developing a need for respiratory support, preferably by non-invasive ventilation with BiPAP (Biphasic Airway Pressure).

Significantly more *sleep problems* are seen in SCL patients than in the normal population (9). Most patients suffering from *OSAS (Obstructive Sleep Apnea Syndrome)* can be treated with nasal or full mask CPAP at night (10). Sleep disordered breathing in tetraplegics is not always diagnosed by ordinary screening for irregular sleep, daytime drowsiness and lack of ability to concentrate. In this respect SCL individuals differ from other persons with OSAS and thus many SCL individuals with OSAS may remain undiagnosed (11). Night sleep studies should therefore be performed regularly every 2-3 years in tetraplegics – preferably with transcutaneous CO₂ tension measurements to diagnose nightly hypoventilation – in addition to continuous oxygen saturation measurements.

Finally, it is advisable to vaccinate all tetraplegic individuals against the flu every year.

REFERENCES

1. Hartkopp A, Brønnum-Hansen H, Seidenschnur AM, Biering-Sørensen F. Survival and cause of death after traumatic spinal cord injury. A long-term epidemiologic survey from Denmark. *Spinal Cord* 1997;35: 76-85. Corrigendum: *Spinal Cord* 1997;35:862-4.
2. Baydur A, Adkins RH, Milic-Emili J. Lung mechanics in individuals with spinal cord injury: effects of injury level and posture. *J Appl Physiol* 2001;90:405-11.
3. Ball PA. Critical care of spinal cord injury. *SPINE* 2001;26:S27-S30.
4. Biering-Sørensen M & Biering-Sørensen F. Trach-eostomy in spinal cord injured: frequency and follow up. *Paraplegia* 1992;30:656-660.
5. Biering-Sørensen F, Knudsen JL, Schmidt A, Bundgaard A, Christensen I. Effect of respiratory training with a mouth-nose-mask in tetraplegics. *Paraplegia* 1991;29:113-9.
6. Langbein WE, Maloney C, Kandare F, Stanic U, Nemchausky B, Jaeger RJ. Pulmonary function testing in spinal cord injury: effects of abdominal muscle stimulation. *J Rehabil Res Dev* 2001;38:591-7.
7. Estenne M, Pinet C, de Troyer A. Abdominal muscle strength in patients with tetraplegia. *Am J Respir Crit Care Med* 2000; 161: 707-12.
8. Elefteriades JA, Quin JA. Diaphragm pacing. *Chest Surg Clinics NA* 1998;8:331-57.
9. Biering-Sørensen F, Biering-Sørensen M. Sleep disturbances in spinal cord injured. An epidemiological questionnaire investigation, including a normal population. *Spinal Cord* 2001; 39:505-13.
10. Biering-Sørensen M, Norup PW, Jacobsen E, Biering-Sørensen F. Treatment of sleep apnoe in spinal cord injured. *Paraplegia* 1995;33:271-3.
11. Stockhammer E, Tobon A, Michel F, Eser P, Scheuler W, Bauer W, Baumberger M, Müller W, Kakebeke TH, Knecht H, Zäck GA. Characteristics of sleep apnea syndrome in tetraplegic patients. *Spinal Cord* 2002;40:286-94.

UPPER LIMB FUNCTION

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SUMMARY

Today the rehabilitation of the tetraplegic with C₄-C₇ lesions offers the possibilities for improved upper limb function. A professional team approach is critical, and the team should include rehabilitation medicine specialists, physical and occupational therapists as well as a hand surgeon. It is essential that the patient as soon as possible after the injury receive intensive physio- and occupational therapy to prevent contractures.

The first hand surgery evaluation of the patient should be at the latest 6 – 12 months following SCL. The main goal of surgery of the tetraplegic patient is to restore the function of locomotion and grip because these patients are totally dependent on their upper limbs.

There are a number of well-established surgical procedures applicable for improving upper limb function in tetraplegia. Functional neuromuscular stimulation is another option.

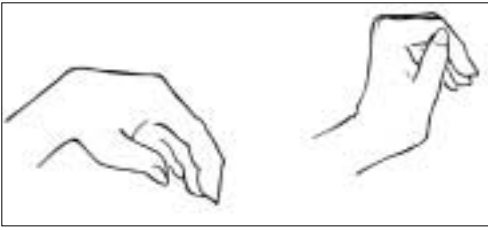
Tetraplegic individuals with C₄-C₇ lesions will often, even when the lesion is incomplete, have impaired function of the wrist and finger muscles. In spite of this situation it is possible for many tetraplegics to utilize special mechanical conditions in the hand to obtain manual abilities, both without or with aids. In addition, the development has further increased the possibilities for improved hand function through electrical stimulation etc.

OCCUPATIONAL THERAPY

In the acute phase of a spinal cord lesion (SCL) it is important to prevent contractures of the finger and hand muscles. Functional training of the impaired function in the upper limb is started when the patient's condition allows it. This includes training of the decreased muscle strength, joint range of movement, coordination, but also to teach the persons to compensate for impaired sensibility etc. This may imply restoration of the muscle balance around the shoulder, improve the quality of existing functions, including stem, or to obtain new functions.

For the tetraplegic individuals it is important to obtain as much hand function as possible. This includes evaluation of which grasps are possible. Depending on the level of lesion and the functions present there will be a large variation regarding the grasps each single individual can obtain. It may be one- or two-hand grasps. Using the tenodesis function many tetraplegics may beside the keypinch (lateral pinch/grasp) be able to have a palmar grasp, which is formed by the thumb, the palm, and the other 4 fingers.

The tenodesis function can be obtained in tetraplegics with C₆ lesions with some function in the extensor carpi radialis muscles, but paralyzes of the finger and hand muscles. It consists of a passive grasp (the key pinch), which is formed between the thumb and the index finger when the wrist is dorsiflexed:



The tenodesis function. From Gregersen H et al. Ergoterapeuten 1978;8:275-80.

Two-hand grasps may be as a support grasp, where the one hand is used as support for a palmar grasp of the other hand, or it may be as a double grasp obtained by the base of the two hands. There may be a need for a bandage, training of the muscle strength, and exercising the use of specific trick grasps.

It is often necessary to adapt splints individually. E.g. a spiral splint, which stabilizes the wrist, and on the same time make it possible for the tetraplegic himself to carry out activities like eating, writing on a PC, drive an electrical wheelchair etc.



A spiral splint.

If the shoulder function is seriously impaired it may be necessary to supply the individual with moveable armrest (feeders), which may give the possibility of performing some functions independently.

ELECTRICAL STIMULATION

Electrical surface stimulation of arm, hand and finger muscles is increasingly used to strengthening the muscles. It seems to facilitate a better and stronger grasp function. This is supported by findings done with use of the functional electrical stimulation device the Bionic Glove. It also resulted in an increase of the range of movements, and most manual tasks tested improved significantly with the use of the assistive system, but on the same time those who retained some dexterity without the system hesitated to use it when manipulating small objects (1).

A specially designed splint with electrical stimulation (Handmaster), which can open and close a key and a palmar grasp, has been developed. It may be used therapeutically, as the functional benefit seems limited to tetraplegics (2).

ADL TRAINING

Functional training is the basis for the ADL (Activities of Daily Living) training. The aim is, that each individual according to his or her possibilities obtain as many skills as possible and hereby gain maximal independence. This should be done considering, that the goals for the training must be realistic and not too ambitious to avoid overuse injuries in the upper limbs the short as well as the in the long run. Therefore it is important to discuss the ADL goals with the SCL person. How far each individual may reach regarding ADL skills depend primarily on the functions remaining according to the neurological lesion, but also on age, height, weight, and not least motivation and psychological condition. In addition, conditions like contractures, spasticity, pain etc. may

influence the final result. One has to be open towards the use of hoist and electrical wheelchair instead of manual transfer and manual wheelchair to decrease deterioration of the remaining upper limb function.

The ADL training in eating, personal hygiene, bathing, and dressing and undressing is carried out in a cooperative effort between the occupational therapists and the nursing staff.

Due to the impaired hand function it is essential to have the necessary aids produced to make each SCL person as independent as possible. It may be standard aids, individually adapted aids or special technical aids.

BOTULINUM TOXIN

The focal injection of botulinum toxin in muscles to alleviate spasticity in the forearm of incomplete tetraplegic SCL individuals may give functional benefits (3). The injections may have to be repeated within some months.

HAND SURGERY

The tetraplegic individuals will lose control of most or all of the 37 muscles, which normally are acting on the wrist and hand. Furthermore is the sensibility of the hand and the fingers in these situations impaired or absent. Surgical treatment of the hand in the tetraplegic patient is more than 50 years old. Later Moberg from Sweden (4), Zancolli from Argentina (5), Freehafer from USA (6) and Lamb and Landry from Scotland (7) evaluated the basic principles in the hand surgery treatments of the tetraplegic patient. The main goal of this surgery is to obtain an efficient grasp or extend the working field for the hand, to increase the skills and hereby the independence. On the same time the need for splints or aids may be diminished.

Hand surgery in the tetraplegic patient has to be part of a team approach. The team must consist of a rehabilitation doctor, an occupational therapist, a physiotherapist and a hand surgeon. It is essential that the patient as soon as possible after the injury receive intensive physio- and occupational therapy to prevent contractures. Often there is a need for splints - especially in the nights.

The first hand surgery evaluation of the patient should be at the latest 6 – 12 months following SCL provided adequate therapy has been instituted (6,8). A good timing of the hand surgeons' first evaluation of the patient would be at the time when the patient is mobilised in a wheelchair and all the other functions are stabilised. In a group of men with SCL 76% ranked the loss of arm and hand function highest – above the loss of leg, bladder and bowel, or sexual function (9).

The options for surgical reconstruction of the tetraplegic upper limb are clarified by an *international classification* of each upper limb independently, based upon the lowest functioning key muscle and residual sensation (8):

<i>Motor grouping</i>	
0	No muscle below elbow suitable for transfer
1	Brachioradialis grade 4 at least
2	Extensor carpi radialis longus plus above
3	Extensor carpi radialis brevis plus above
4	Pronator teres plus above
5	Flexor carpi radialis plus above
6	Finger extensors plus above
7	Thumb extensors plus above
8	Digital flexors plus above
9	Lacks intrinsics only
X	Exceptions

<i>Sensory grouping</i>	
O	If 2 point discrimination is greater than 10 mm on the thumb and the index finger
Cu	If 2 point discrimination is less than 10 mm on the thumb and the index finger

This classification is only a guide for the forearm and the hand. However, the classification is useful in determination of the possibility for hand surgery at the wrist and hand in the tetraplegic patient. The classification does not include the shoulder, and thus the need and possibility for activating of the triceps must be evaluated separately.

TENDON TRANSFERS

In evaluation of the available muscles for tendon transfers, the muscles must have an adequate strength. The muscles, which are going to be used in tendon transfers, must have strength at grade 4. Operating on the upper extremity of the tetraplegic patient, one should always address the proximal problems first. If the patient does not have any function of the brachial triceps muscle, this will be the first muscle to activate. For activation of the brachial triceps muscle the posterior part of the deltoid muscle can be used (10). This muscle is connected to the brachial triceps tendon by the use of a free tendon graft from the tibialis anterior tendon or the fascia lata. The biceps tendon can also be used to activate the brachial triceps. This transfer should be considered if the patient has an extension defect in the elbow exceeding 30 degrees. Stabilisation of the elbow activating the brachial triceps is very important for improving the results of the more distal tendon transfer - especially when using the

brachioradialis muscle for transfer, due to the fact that brachioradialis and triceps works as antagonist. The triceps activation also improves the overhead activities, transferring and pressure lifts.

Functional reconstruction of the hand has two main stages: restoration of wrist extension and construction of the key grip. The restoration of the wrist extension is only necessary in group 1 of the international classification; these patients present a very weak wrist extension. The only muscle they have available for transfer is brachioradialis which can be used to activate extensor carpi radialis longus and brevis so the patient get an active dorsal extension in the wrist. This will improve the tenodesis grip (10).

If the patient has an active dorsal flexion of the wrist e.g. group 3 to 4 is it possible to provide him with an active key pinch using brachioradialis to activate flexor pollicis longus, at the same time securing a tenodesis at the interphalangeal joint (10).

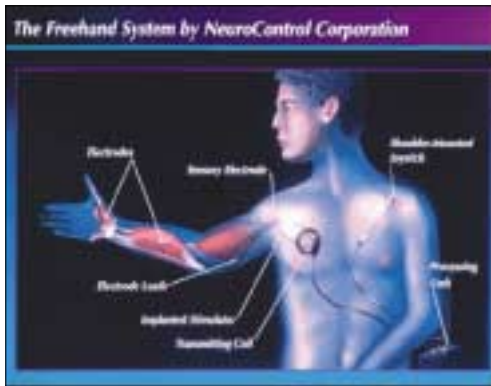
There are numerous other tendon transfers available according to the available muscles. However, only few of these have been described with an acceptable follow-up.

Following hand surgery procedures, the patients generally need to be immobilised in a bandage for 3 to 6 weeks. This period is often very troublesome for the patient being more dependent than before the operation. Therefore, it is important carefully to discuss the operation and follow-up treatment with the patient.

IMPLANTED ELECTRODES

In Cleveland, Ohio, USA an implanted system FES (Freehand system) is developed to facilitate a key pinch and a palmar grasp in C₅₋₆ tetraplegics.

The system includes implantation of



epimyseal electrodes in up to 8 muscles with hand and finger function. These are selected after testing with electrical surface stimulation and verification during surgery. There will usually concurrently be performed tendon transfers. The grasps and their opening and closing are controlled with a shoulder transducer on the opposite side. Today the system is implanted in about 300 tetraplegics worldwide, and a multicentre investigation of 51 adult tetraplegics has shown that the grasping ability provided by the neuroprosthesis is substantial and lasting, and offers improved independence (11).

This system is under continuous development, and a new advanced neuroprosthesis provides additional control of the forearm pronation and elbow extension. The implanted components include a 10-channel stimulator and a joint angle transducer. The shoulder control is changed to a control by using re-tained voluntary wrist extension (12).

COMPLICATIONS

Shoulder problems constitute a special challenge for many SCL persons. In a prospective study of 53 SCL individuals until 15 years post-injury 72%

demonstrated radiological evidence of degenerative changes, but only 11% complained of pain in the shoulders. The study found correlation between development of *degenerative changes* in the shoulders and higher level of wheelchair activity, higher age, and female gender (13). This may be due to the sometime extreme load they are exposed to in their daily life, during use of manual wheelchair, transfers to/from bed, toilet, car etc. A shoulder exercise program may be used for prevention.

The risk of shoulder impingement syndrome in wheelchair athletes, not least elite tetraplegic wheelchair rugby players is stressed (14).

There is also an increased risk of nerve entrapment in SCL individuals, both to the median nerve at the carpal tunnel and of the ulnar nerve at the elbow, not least due to the use of manual wheelchair. It has been shown the weight of the SCL person and the push rim biomechanics is related to the median nerve function, and this should be used prophylactic in manual wheelchair users (15).

Neuropathy of the shoulder (16) and elbow is a risk due to the impaired or absent sensation.

Reflex sympathetic dystrophy may be seen in tetraplegics, and should be managed in the same way as in other patients.

CONTINUOUS FOLLOW-UP

All tetraplegics should, in addition to the initial evaluation be seen by a hand surgeon if functional changes occur.

REFERENCES

1. Popovic D, Stojanovic A, Pjanovic A, Radosavljevic S, Popovic M, Jovic S, Vulovic D. Clinical evaluation of the bionic glove. Arch Phys Med Rehabil 1999;80:299-304.

2. Snoek GJ, IJzerman MJ, in't Groen FACG, Stoffers TS, Zilvold G. Use of the NESS Handmaster in tetraplegia: clinical experience in ten patients. *Spinal Cord* 2000;38:244-9.
3. Richardson D, Edwards S, Sheean GL, Greenwood RJ, Thompson A. The effects of botulinum toxin on hand function after incomplete spinal cord injury at the level of C5/6: a case report. *Clin Rehabil* 1997;11:288-92.
4. Moberg E. The upper limb in tetraplegia. A new approach to surgical rehabilitation. Stuttgart: Georg Thieme, 1978.
5. Zancolli EA. Structural and dynamic bases of hand surgery, 2nd ed. Philadelphia: JB Lippincott, 1979
6. Freehafer AA. Tendon transfers in tetraplegic patients: the Cleveland experience. *Spinal Cord* 1998;36:186-9.
7. Lamb DW, Landry RM. Hand in quadriplegia. *Paraplegia* 1971;9:204-12.
8. Johnston BR, Jordan CJ, Buntine JA. A review of surgical rehabilitation of the upper limb in quadriplegia. *Paraplegia* 1988;26:317-39.
9. Hanson RW, Franklin MR. Sexual loss in relation to other functional losses for spinal cord injured males. *Arch Phys Med Rehabil* 1976; 57:291-3.
10. Waters RL, Sie IH, Gellman H, Tognella M. Functional hand surgery following tetraplegia. Review article. *Arch Phys Med Rehabil* 1996; 77:86-94.
11. Peckham PH, Keith MW, Kilgore KL, et al. Efficacy of an implanted neuroprosthesis for restoring hand grasp in tetraplegia: a multi-center study. *Arch Phys Med Rehabil* 2001;82: 1380-8.
12. Peckham PH, Kilgore KL, Keith MW, Bryden AM, Bhadra N, Montague FW. An advanced neuroprosthesis for restoration of hand and upper arm control using an implantable controller. *J Hand Surg* 2002;27A:265-76.
13. Lal S. Premature degenerative shoulder changes in spinal cord injury patients. *Spinal Cord* 1998;36:186-9.
14. Miyahara M, Sleivert GG, Gerrard DF. The relationship of strength and muscle balance to shoulder pain and impingement syndrome in elite quadriplegic wheelchair rugby players. *Int J Sports Med* 1998;19:210-4.
15. Boninger ML, Cooper RA, Baldwin MA, Shimada SD, Koontz A. Wheelchair pushrim kinetics: body weight and median nerve function. *Arch Phys Med Rehabil* 1999;80:910-5.
16. Barber DB, Janus RB, Wade WH. Neuroarthropathy: an overuse injury of the shoulder in quadriplegia. *J Spinal Cord Med* 1996;19:9-11.

LATE COMPLICATIONS

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SUMMARY

As life expectancy is increasing in the population with spinal cord lesions (SCL), the emphasis of late complications is becoming more important. These include multiple challenges within most body functions: Respiratory with pneumonia, atelectasis, respiratory exhaustion, and sleep problems. Cardiovascular with low blood pressure, autonomic dysreflexia, thromboembolism, and swollen legs. Urinary with incontinence, residual urine, urinary tract infection and dilatation, urinary stone, decreased kidney function, and bladder cancer. Bowel with constipation, haemorrhoids, air in the intestines, and megacolon. Skin with pressure ulcers with osteomyelitis and Majolain cancer, and erysipelas. Impaired temperature regulation and reflex sweating. Sexual function with impaired orgasm, vaginal lubrication, erection, ejaculation, and fertility. Muscle-skeletal with contractures, spasticity, degenerative change, shoulder impingements, nerve entrapment, overuse injury, osteoporosis, fractures, heterotopic ossifications, and spinal deformities. Pain. Endocrine and metabolic changes. Neurological deterioration. Psychological with depression and suicide.

The survival after a traumatic spinal cord lesion (SCL) has improved. Before the 2nd World War died 60-80% within the first year post-injury. In the 1960'ies the mortality decreased to 30%, in the 1970'ies to 15% and in the 1980'ies down to 6% (1). It has been

shown that the probability to survive 25 years after a SCL is 60.6% and 66.7% for SCL men and women respectively. The corresponding figures for the general population of the same age are 66.9% and 76.5%. Correspondingly 70% of the SCL men and women will still be alive after 19.5 and nearly 22 years respectively. The corresponding figures for the general population of the same age are 23.5 and 29 years. The most common causes of death for the SCL individuals were lung diseases, in particular pneumonia, cardiovascular diseases, and suicide (2).

Those early as well as late complications SCL individuals experience are due to the functional changes the SCL cause in every system below the level of lesion.

Because some of the issues are elaborated upon in other chapters they will only be mentioned briefly in this chapter to give the overall picture of the late complications that may occur in SCL individuals.

RESPIRATION

The most common cause of death is pulmonary disease, in particular pneumonia (2). Due to decreased cough function with secrete stagnation atelectasis is common.

Due to paralyse of the trunk muscles kyphoscoliosis may develop. In severe cases it can cause significant restrictive lung problems. Some tetraplegic individuals are found to be respiratory exhausted within time and may develop a need for respiratory support.

Significant more sleep problems have

been observed in SCL individuals than in the normal population. See the chapter on RESPIRATION.

CARDIO-VASCULAR FUNCTION

LOW PRESSURE AND SLOW PULSE

With a SCL the blood vessels below the level of the cord lesion will dilate due to the decreased tone of the sympathetic nervous system. Because of this change the blood pressure decreases. This is more pronounced the more cranial the cord lesion is located, i.e. in tetraplegics a systolic pressure of 90-110 mmHg is normal. The low pressure, and the often prolonged bed rest can give *orthostatic hypotension*, which may be relieved by gradual mobilisation to upright position, e.g. by use of a tilt-table.



Orthostatic hypotension may be relieved by gradual mobilisation to upright position by use of a tilttable

The heart is innervated by the proximal part of the sympathetic nervous system (above T5), which for more cranial cord lesions means that the pulse will become slower.

AUTONOMIC DYSREFLEXIA

Autonomic dysreflexia is a condition, which may occur in the acute stage and during the rest of the life for SCL individuals with tetraplegia and paraplegia with levels of lesion at or above T₆ (3). The condition may vary from a mild to a life-threatening situation. Autonomic dysreflexia is due to hyperactivity of the thoracolumbar sympathetic nervous system, which is without the normal supraspinal control. This hyperactivity is caused by visceral (from e.g. bladder, rectum, uterus) and other somatic afferent impulses from below the level of the SCL. This means that a reflexogenic vasoconstriction with blood pressure increase may be triggered, which via the baroreceptors (in the aortic arc or carotic sinus) trigger a vagal bradycardia, but without a coordinated regulation of the peripheral resistance due to the SCL. The greatest risk is severe hypertension with potential danger of cerebro-vascular haemorrhage.

Clinically the cause may be distension, or instrumentation of the bladder or bowel, urinary tract infection, kidney or bladder stones, pressure ulcer, burns, fracture, uterus contractions or spasms during menstruation, sexual activity, stomach ulcer, appendicitis etc.

One or more of the following symptoms may occur: Pounding headache, flushing, sweating proximal to the level of lesion, nausea, goose flesh without fever, anxiety, and cardiac arrhythmia. There is slow pulse and blood pressure up to 300/160 mmHg.

Treatment include:

- 1) Calm down the person. Sit up in a comfortable position. Remove compressing stockings, and loosen any clothing or constrictive devices.
- 2) If catheterisation or any other manipulation of the patient is ongoing it should be stopped until the symptoms have declined.
- 3) Bladder distension is the most common cause: Drainage systems should be checked for possible obstructions (clamp, kinks, filled urinary bag, obstructed catheter), and a large bladder without catheter should be emptied by catheter. Avoid manually compressing or tapping on the bladder.
- 4) Other treatable causes: Bowel distension due to faecal impaction: Apply local anaesthetic jelly in the rectum, but wait with the evacuation approximately 5 minutes.
- 5) Localised inflammation, ingrown toenails, ulcer, fractures etc.
- 6) Pharmacological treatment:
 - a) Acute: Nifedipine 10 mg sublingually, glycerylnitrate 0.25mg - 0.5mg sublingually (no nitrates if the person used sildenafil within the last 24 hours), or clorpromazine 1-2 mg i.v. All may be repeated.
 - b) Less acute: Clorpromazine 12-25 mg i.m./100 mg rectal, fentolamine 5-10 mg i.m./slowly i.v., or clonidine 0.15 mg s.c./i.m./slowly i.v. Dihydralazine 2.5 mg i.v., may be repeated with increasing doses up to 20 mg. Max. dose of 77.5 mg.
 - c) Preventive: If repeated attacks, t.b.l. prazosine -1 mg may be tried and can be increas

THROMBOEMBOLISM

Because of high risk of deep vein thrombosis and hereby lung embolus, in particular within the first 12 weeks it is advised to use anticoagulant prophylaxis with e.g. low molecular

weight heparin, and individually adjusted compression hose. Reinstitution of prophylactic measures should be considered in chronic SCL individuals if they are immobilised for a prolonged period. With documented deep vein thrombosis mobilisation and exercise of the lower extremities should be withheld 48 to 72 hours until appropriate medical therapy is implemented (4).

SWOLLEN LEGS

Due to the paralyse of the lower limbs the normal muscle pump doesn't function. This implies a risk for swollen legs and feet. The most important is to keep the legs elevated whenever possible. Compressive stockings may also help.

URINARY TRACT AND KIDNEY

Incontinence and residual urine due to impaired bladder emptying, with risk for development of urinary tract infections (UTI) are common. Permanent catheter is the greatest risk for UTI, and additional a risk for development of bladder cancer.

Urinary stones occur in 5-10% of SCL persons during 5-10 years.

Upper urinary tract dilatation and decreased kidney function may occur, not least due to high intravesical pressure, and the outlet conditions should be checked.

See the chapter on BLADDER MANAGEMENT.

BOWEL

Faecal incontinence and constipation are major challenges.

Haemorrhoids are another significant and recurring problem.

Air in the intestines and feeling of distension is common.

Megacolon has been found in 73% of admitted SCL in-patients.

See the chapter on BOWEL MANAGEMENT.

SKIN

Pressure ulcer is the complication, which cause most days out of normal function for SCL individuals. The risk of osteomyelitis and malignant transformation (Majolain cancer) in chronic ulcers are important to observe. Erysipelas is common.

See the chapter on SKIN AND DECUBITUS.

TEMPERATURE REGULATION

The temperature regulation is compromised. Afferent information to the hypothalamic temperature centre is abolished. The denervation causes that the SCL individual can't sweat or shiver below the level of the SCL. Therefore the body is not able to adjust to either low or high temperatures in the environment, but will become relatively poikilothermic.

This implies that in particular tetraplegics may have problems at low as well as high temperatures. In cold environment it is important for tetraplegics to have sufficient cloths on, and in addition to drink warm liquid. In warm environments it is necessary to stay in the shade, drink cold liquid, and have the possibility of a cold bath etc.

In addition, tetraplegics may not always have fever even with serious conditions.

REFLEX SWEATING

Above one third of SCL individuals reports more or less frequent reflex sweating, and for the majority this is annoying. Tetraplegics seem to have most problems with hyperhidrosis, while it is seldom in SCL with lumbar lesions. Its cause is not well understood, but it is usually thought to be due to the compromised temperature

regulation and autonomic dysfunction. Probably reflex sweating is provoked through the same mechanisms as autonomic dysreflexia. Therefore before treatment it is important to exclude any treatable cause, such as urine retention, constipation, decubitus, infection, etc., as well as post-traumatic syringomyelia. The efficacy of various drugs has only to a limited extend been tried out in controlled studies. α -receptor blockers, anticholinergics, and painkillers may be used.

SEXUAL FUNCTION

Many SCL individuals may not obtain orgasm. Due to physical limitations there may be positioning problems when having sex.

WOMEN

The vaginal lubrication may be impaired. At pregnancy there is an increased risk of UTI. Delivery can be performed vaginally, but caesarean section may be necessary.

See the chapter on SEXUAL FUNCTION IN WOMEN, PARTNERSHIP, PREGNANCY AND DELIVERY.

MEN

SCL men may have erectile as well as ejaculatory dysfunction.

Impaired semen profiles are seen in the majority of SCL men.

See the chapter on SEXUAL FUNCTION AND FERTILITY IN MEN.

MUSCULO-SKELETAL CONTRACTURES

Contractures may limit the functional capability of the person very much, and should therefore be avoided. Maintenance training at a physiotherapist who counteracts the contractures by stretching of the muscles is important.

It is important to prevent contractures wherever possible. This could for in-

stance be by lying at the belly during the night to avoid contractures of the hip flexors. Likewise the SCL person can prevent the development of contractures of the foot plantar flexors by use of a standing frame or similar.

SPASTICITY

Spasticity and spasms are serious complications to SCL.

See the chapter on SPASTICITY.

UPPER LIMB

Shoulder problems constitute a challenge, and the development of degenerative changes in the shoulders is correlated to higher level of wheelchair activity. Neuroarthropathy of the shoulder and elbow is a risk due to the impaired or absent sensation. There is also an increased risk of shoulder impingement and nerve entrapment both to the median nerve at the carpal tunnel and of the ulnar nerve.

See the chapter on UPPER LIMB FUNCTION.

LOWER LIMB

Hip and knee problems may develop in SCL individuals with incomplete lesions who are walking. These include degenerative changes in the knees due to overextension.

OVERUSE INJURY

Viewing changes in ADL it is important to find out if there are activities, which may cause overuse injury, and accordingly to find modifications to avoid the situation causing the problem. There has been an increased awareness of the possible long-term side effects of a high ambition level. This implies a larger openness for the use of e.g. hoists and electrical wheelchair to decrease the demand to the upper limb during transfer and propelling the manual wheelchair.

It is likewise important to be aware of the negative implications of a weight increase.

OSTEOPOROSIS AND FRACTURES

Bone mineral may be reduced to 50% at the level of the knee with high risk of bone fractures.

See the chapter on OSTEOPOROSIS.

HETEROTOPIC OSSIFICATIONS (HO)

Posttraumatic neurogenic HO may develop around the large joints below the level of the SCL in 8-30%, and up to 35% will eventually have significantly limited joint mobility (5), which may reduce ADL, transfers and the sitting position.



CT-scanning showing bilateral hip HO.

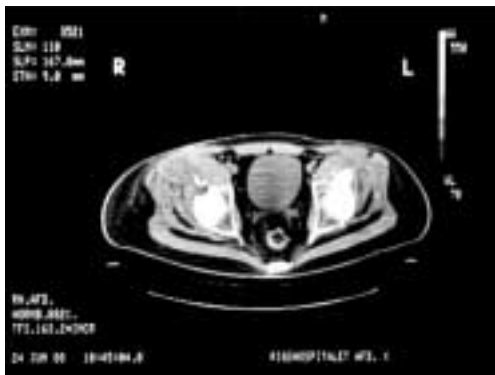
The cause of HO is unknown, but the pathogenesis has been suggested to be a transformation of primitive cells of mesenchymal origin into osteogenic cells (5).

The diagnosis in the early HO with fever, swelling, and erythema may be difficult to distinguish from cellulitis, osteomyelitis, and thrombophlebitis (5).

Alkaline phosphatase level increase from two weeks after HO. Before the HO can be visualised by X-ray, bone scanning is the investigation of choice. Complications of HO include peripheral nerve entrapment and pressure ulcers (5).

The use of diphosphonates, non-steroidal anti-inflammatory drugs and

radiation for prevention and treatment of HO remain controversial. Limitations in ADL may indicate surgical resection of HO, but recurrences are common.



CT scan of the same patient as above – after surgery.

SPINAL DEFORMITY

After a spinal fracture there is a risk of non-union, implant failure, Charcot spine, and technical error. The overall outcome of surgical management of these post-traumatic deformities is better if treated earlier as opposed to later (6). Spinal deformities like scoliosis, severe lumbar lordosis, and spinal kyphosis may develop due to the paralysis, contractures and spasticity, not least in those who had their SCL during childhood. Some of these deformities may cause



Paralytic scoliosis before corrective spinal surgery to the left, and after the surgery to the right.



pulmonary problems and affect the ADL. Therefore they should be monitored and surgical spinal intervention may be indicated.

PAIN

Pain is experienced by a majority of SCL individuals.

See the chapter on PAIN.

ENDOCRINE AND METABOLIC FUNCTIONS

The paralysis cause muscle atrophy. This and diminished physical activity implies insulin resistance, changes in the blood lipid, increased regional fat deposition, and increased blood pressure. These conditions predispose to diabetes and cardiovascular diseases. To prevent these problems the SCL individual may e.g. stop smoking, and do physical activity using the arms or electrical stimulation of the lower limbs.

See the chapter on EXERCISE AND PHYSICAL TRAINING MODALITIES FOR THE SPINAL CORD INJURED INDIVIDUAL: POSSIBLE CLINICAL IMPLICATIONS TO COUNTERACT LIFE-STYLE DISORDERS.

NEUROLOGICAL DETERIORATION

Post-traumatic syringomyelia has been reported at incidences of 20-50%, and seen as early as 2-3 months after injury. See the chapter on SYRINGOMYELIA.

PSYCHOLOGICAL

There is a high prevalence of depression among SCL individuals. Individual and family education is needed, and psychotherapy should be provided. If indicated selective serotonin reuptake inhibitors (e.g. citalopram, fluoxetine, paroxetine and sertraline)

are safer and better tolerated than the tricyclic antidepressants and monoamine oxidase inhibitors. If efficient during the acute treatment phase it should be continued for at least 6 months (7).

Suicide was found up to 4.9 times more common in SCL than in the general population. The rate among marginally disabled was nearly twice as high as in complete tetraplegics. These situations should be given special attention during rehabilitation and follow-up (8).

LIFE-LONG FOLLOW-UP

Due to the risk for multiple late complications it is important that SCL individuals are followed life-long in designated centres familiar with this large spectrum.

REFERENCES

1. Ducker TB. Treatment of spinal cord injury. [Editorial]. *New Engl J Med* 1990; 322(20):1459-61.
2. Hartkopp A, Brønnum-Hansen H, Seidenschnur AM, Biering-Sørensen F. Survival and cause of death after traumatic spinal cord injury. A long-term epidemiologic survey from Denmark. *Spinal Cord* 1997;35:76-85. [Corrigendum. *Spinal Cord* 1997;35:862-4].
3. Clinical practice guidelines: Acute management of autonomic dysreflexia. *J Spinal Cord Med* 1997;20:284-308. www.pva.org, under publications.
4. Clinical practice guidelines: Prevention of thromboembolism in spinal cord injury. *J Spinal Cord Med* 1997;20:259-83. www.pva.org, under publications.
5. Shehab D, Elgazzar AH, Collier BD. Heterotopic ossification. *J Nucl Med* 2002;43:346-53.
6. Vaccaro AR, Silber JS. Post-traumatic spinal deformity. *SPINE* 2001;26(24S):S111-8.
7. Depression following spinal cord injury: A clinical practice guideline for primary care physicians. Consortium for Spinal Cord Medicine, Paralyzed Veterans of America, 1998. www.pva.org, under publications.
8. Hartkopp A, Brønnum-Hansen H, Seidenschnur AM, Biering-Sørensen F. Suicide in a spinal cord injured population in relation to functional status. *Arch Phys Med Rehabil* 1998;79:1356-61.

SPASTICITY

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SUMMARY

About 70% of persons with spinal cord lesions are spastic 1 year after injury, although not all need treatment. For clinical assessment the modified Ashworth and Penn scales may be used. More precise evaluation may be obtained by isokinetic measurements. Before treatment of spasticity is initiated otherwise treatable factors should be excluded. Physical treatment including positioning, stretching, application of cold, topical anaes-thesia, and electrical stimulation may diminish tone, but the effects are generally short-lived and to some extent debateable. Oral antispastic medication is commonly used, in particular baclofen, and tizanidine. Less commonly used drugs are diazepam, dantrolene and clonidine. Intrathecal medication, in particular baclofen, is very efficient. The effect of cannabinoids seems doubtful. Injection of Botulinum toxin into motorpoints is effective in relieving spasticity in specific muscles. Most destructive surgical procedures are rarely indicated today.

Spasticity may be defined as a motor disorder characterised by a velocity-dependent increase in tonic stretch reflexes that results from abnormal intraspinal processing of primary afferent input (1). Clinically this may imply enhanced tendon reflexes, extended reflex zones, clonus, increased muscle tone and increased

flexor-reflexes. *Spasms* are sudden involuntary muscle-contractions, which are characteristic for many spinal cord lesioned (SCL) individuals, in particular corresponding to knee-extension and hip-flexion.

Spasticity is one of the most common and potentially disabling complications affecting SCL individuals. Roughly 70% of persons with SCL are spastic 1 year after injury, although not all require treatment (2).

PATHOPHYSIOLOGY

Spasticity is caused by adaptational changes in transmission in the spinal networks distal to a lesion of descending motor pathways. Selective lesion of the pyramidal tract does not lead to spasticity, whereas lesion of descending pathways from the brainstem as well as the cortical control of these pathways does. In SCL individuals impaired transmission in different spinal inhibitory pathways, such as reciprocal inhibition and presynaptic inhibition has been found. This leads to increased excitability of spinal motoneurons, exaggeration of reflex activity and increased muscle tone. Likely, physiological changes in the signalling properties of the spinal neurons as well as more structural changes such as sprouting are involved. Changes in the mechanical properties of muscles in SCL individuals have also been found. Such changes may clinically be mistaken as signs of spasticity.

Spasticity is less severe in SCL individuals with complete spinal cord lesions, and is more severe in those with minimal sparing of voluntary movement (2).

ASSESSMENT

The degree of spasticity varies from insignificant to very severe disability.

BIOMECHANICAL METHODS

Objective biomechanical techniques, such as isokinetic dynamometers, are of great value when a precise objective and reproducible measure of spasticity is necessary, such as in relation to research projects and drug evaluation, but they play no or only a minor role in the daily clinical evaluation of spasticity. The various techniques are based on the application of well-defined passive movements of specific joints (mostly

Modified Ashworth scale

0	No increased tone
1	Slight increase in tone, manifested by a catch and release or by minimal resistance at the end range of motion (ROM) when the part is moved in flexion or extension / abduction or adduction etc.
2	Slight increase in tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM
3	More marked increase in tone through most of the ROM, but the affected part is easily moved
4	Considerable increase in tone, passive movement is difficult
5	Affected part is rigid in flexion or extension, etc.

Penn scale

0	No spasms
1	Mild spasms at stimulation
2	Irregular strong spasms less than 1 time/hour
3	Spasms more often than 1 time/hour
4	Spasms more than 10 times/hour

knee and ankle joint) and measurement of the resistance to these movements. This permits that the velocity-dependency of the muscle resistance (which is central to the definition of spasticity; cf. above) and contributions from intrinsic muscle resistance and stretch-evoked muscle resistance may be determined (3,4).

CLINICAL METHODS (5)

The most frequently used clinical methods are the Modified Ashworth Scale (6) and the Penn scale (7) for spasticity and spasm frequency evaluation, respectively.

WHEN TO TREAT

Before treating spasticity otherwise treatable factors should be excluded, e.g. urinary tract infection, pressure sore, deep vein thrombosis, heterotopic ossification, stool impaction, unsuspected fracture, ingrown toenails, markedly worsens the symptoms and signs of spinal spasticity. In addition possible syringomyelia as cause of increased spasticity should be ruled out. Stress, emotional or physical, may also have an impact on the degree of spasticity present. Similarly, drugs that are being taken for other co-morbid disorders may adversely affect spasticity, e.g. some antidepressants, including fluoxetine, trazadone, and sertraline (1, 2).

When deciding to treat spasticity one must have realistic goals. The motor disability is most likely the result of paresis, not spasticity itself, and relief of spasticity may reduce pain and discomfort, prevent or decrease medical complications (decubitus or contractures), improve sleep, and facilitate mobility, nursing care or hygiene, but is unlikely to restore function (1, 2).

It should be considered that the neuronal elements, which show exaggerated activity in spasticity, are important also for the central control of normal voluntary movements. This makes it sometimes difficult to treat spasticity without also affecting the functional ability of the SCL individuals.

PHYSICAL TREATMENT

PHYSICAL AND OCCUPATIONAL THERAPY

The most common physical measures include proper positioning and stretching. Proper bed positioning early after SCL may reduce long-term spasticity and contractures. Stretching once to twice daily diminishes tone, although the effect is relatively shortlived (1,2). Stretching should be performed gently to avoid haematomas in the muscles or fractures.

Standing in tilt table or standing frame has been shown to decrease spasticity, and splints can improve range of motion that develops as a result of spasticity and contracture (1,2).

Modalities such as cold and topical anaesthesia may decrease tendon reflex excitability, reduce clonus, and increase range of motion. A cold pack should be used for roughly 20 minutes for maximal effect, but its duration of effectiveness is less than 1 hour (2).

ELECTRICAL STIMULATION

Various techniques of electrical stimulation have been shown to be effective in

the treatment of spasticity. This includes stimulation to the antagonist muscle to achieve reciprocal inhibition, or tetanic contraction to the spastic muscle. Likewise functional electrical stimulation has shown to decrease spasticity in SCL individuals (2). A disadvantage may be that the muscles stimulated become stronger, and this may cause stronger spasms. It should be noted that there is not universal acceptance of the effect of electrical stimulation on spasticity.

Electroejaculation using a rectal probe has also shown to reduce paraplegic spasticity and spasms for 3 to 24 hours (1).

VIBRATORY STIMULATION

Likewise there have been indications that penile vibratory stimulation for ejaculation can reduce spasticity and spasms (8).

ORAL MEDICATION

If medical treatment is found indicated one should always evaluate if the treatment increase the paresis and eventual compromise a standing or walking function.

A Cochrane review found that 9 out of 53 studies met the inclusion criteria, and they found only tizanidine to have significant effect, while for other drugs (gabapentine, clonidine, diazepam, amyral and oral baclofen) the results do not provide evidence for a clinical significant effectiveness. Therefore there is insufficient evidence to assist clinicians in a rational approach to antispastic treatment for SCL individuals (9).

Therefore it is not surprising that in daily clinical practice the use of oral medications is often a question of trial and error. Most commonly used medications (2):

Medication	Daily dose
Baclofen	10→200 mg
Tizanidine	2→36 mg
Diazepam	4→60 mg
Dantrolene	25→400 mg
Clonidine	0.05→0.4 mg

All these drugs have potentially serious side effects, which should be carefully considered before administration. They should only be continued if there is a clear beneficial effect. Many other drugs are used (10).

The possible use of cannabinoid CB1 receptor agonists for suppression of muscle spasm and spasticity in SCL individuals is currently discussed, but oral tetrahydrocannabinol seems to undergo variable absorption and to have a narrow therapeutic window, making it difficult to predict an oral dose that will be both effective and tolerable (11). A current randomised placebo-controlled trial in 16 multiple sclerosis patients showed no effect in reducing spasticity (12).

INJECTION TECHNIQUES

This is effective for treating local spasticity and facilitating functional goals, with minimal systemic side effects and no effect on cognition. Peripheral blocks include motor point and nerve blocks.

Botulinum toxin is injected into motor-points and will reduce or abolish the release of acetylcholine from presynaptic motor axons and thereby weaken the muscle. This chemical denervation develops over the course of a few days and lasts for 3 to 8 months. The usefulness depends on treating a few particularly crucial muscles (1,2).

Peripheral nerve blocks with alcohol or phenol may damage the nerves, cause

pain at the injection site, and are physically destructive, producing scarring and fibrosis. The effects last 8-12 months and are not fully reversible (1,2).

INTRATHECAL MEDICATION

Intrathecal baclofen is indicated for SCL individuals who do not respond or have intolerable side effects from oral medication. Intrathecal baclofen has shown to decrease pain, improve sleep, ADL, bladder and bowel program, and decrease caregiver time. Objectively, there is a reduction in Ashworth and spasm scores (2). It is even found effective for treating upper extremity hypertonia (13). If the decision for administration of intrathecal baclofen has been made, a lumbar puncture is performed and a trial dose of baclofen is administered. If satisfactory effect is observed, a reservoir-pump may be surgically positioned in a subcutaneous pocket at the abdomen and connected with an intrathecal catheter. The reservoir is filled every month or every second month with baclofen in an appropriate concentration. The pump releases the baclofen continuously at a preset amount. The treatment is very efficient, and the SCL individuals avoid the side effects of oral treatment, in particular the drowsiness.

Intrathecal clonidine and morphine may play a role, when baclofen is inefficient or there is increasing tolerance to baclofen (2).

SURGICAL PROCEDURES

Peripheral ablative procedures include rhizotomy and peripheral neurectomy, and central ablative procedures cordectomy, myelotomy and stereotactic procedures (14). The destructive therapies are today rarely indicated (1,2). Spinal cord stimulation

of the upper lumbar segments can be effective in the treatment of lower extremity hypertonia in SCL individuals (15).

In intractable cases *tendon elongations and myotomies* may be necessary due to contractures.

REFERENCES

1. Young RR. Spasticity: A review. *Neurology* 1994;44 (Suppl. 9):S12-S20.
2. Kirshblum S. Treatment for spinal cord injury related spasticity. *J Spinal Cord Med* 1999;22: 199-217.
3. Akman MN, Bengi R, Karatas M, Kilinc S, Sozay S, Ozker R. Assessment of spasticity using isokinetic dynamometry in patients with spinal cord injury. *Spinal Cord*. 1999 37:638-43
4. Franzoi AC, Castro C, Cardone C. Isokinetic assessment of spasticity in subjects with traumatic spinal cord injury (ASIA A). *Spinal Cord*. 1999; 37:416-20.
5. Pierson SH. Outcome patterns of clinical motor dysfunction. *Muscle and Nerve* 1997;20 (Suppl.6):S36-S60.
6. Bohannon RW, Smith MB. Interrater reliability of a Modified Ashworth Scale of muscle spasticity. *Physical Therapy* 1987;67:206-7.
7. Penn RD, Savoy SM, Corcos D, et al. Intrathecal baclofen for severe spinal spasticity. *N Engl J Med* 1989;320:1517-54.
8. Sønksen J, Biering-Sørensen, F, Kristensen, JK. Ejaculation induced by penile vibratory stimulation in men with spinal cord injuries. The importance of the vibratory amplitude. *Paraplegia* 1994;32:651-60.
9. Taricco M, Adone R, Pagliacci C, Telaro E. Pharmacological interventions for spasticity following spinal cord injury (Cochrane Review). In: *Cochrane Library*, 4, 2001. Oxford: Update Software.
10. Gracies J-M, Nance P, Elovic E, McGuire J, Simpson DM. Traditional pharmacological treatments for spasticity Part II: General and regional treatments. *Muscle and Nerve* 1997;20 (Suppl.6):S92-S120.
11. Pertwee RG. Cannabis and cannabinoids: Pharmacology and rationale for clinical use. *Forsch Komplementärmed* 1999;6(Suppl.3):12-5.
12. Killestein J, Hoogervorst ELJ, Reif M, Kalkers NF, van Loenen AC, Staats PGM, Gorter RW, Uitdehaag BMJ, Polman CH. Safety, tolerability, and efficacy of orally administered cannabinoids in MS. *Neurology* 2002;58:1404-7.
13. Burns AS, Meythaler JM. Intrathecal baclofen in tetraplegia of spinal origin: efficacy for upper extremity hypertonia. *Spinal Cord* 2001;39:413-9.
14. Smyth MD, Peacock WJ. The surgical treatment of spasticity. *Muscle and Nerve* 2000;23: 153-63.
15. Pinter MM, Gerstenbrand F, Dimitrijevic MR. Epidural electrical stimulation of posterior structures of the human lumbosacral cord: 3. Control of spasticity. *Spinal Cord* 2000;38:524-31.

PAIN

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SUMMARY

Musculoskeletal, visceral, and neuropathic pain represent a major burden in chronic spinal cord lesioned (SCL) patients. Treatment of musculoskeletal pain involves a careful analysis of underlying mechanisms and treatment of inflammation, muscle weakness, instability, stiffness, posture, and obesity. NSAID and paracetamol may be used for shorter periods. Neuropathic pain presents itself as spontaneous and evoked pain (e.g. allodynia) and may be accompanied by unpleasant abnormal sensations (dysesthesias). Neuropathic pain often have a delayed onset, it is long lasting and difficult to treat. Drugs usually provide only partial pain relief. For management of neuropathic pain we recommend pharmacological treatment with tricyclic antidepressants (e.g. imipramine) or anti-epileptic drugs (e.g. gabapentin or lamotrigine). Opioids (e.g. tramadol) should be used with care because of their obvious side effects.

The average reported frequency of chronic pain in patients with spinal cord lesion (SCL) is about 65%, with one-third of those affected reporting severe pain (1). The Spinal Cord Injury Pain Task Force of the International association of the study of pain (IASP) broadly classifies SCL pain into nociceptive (musculoskeletal and visceral) and neuropathic pain (1).

MUSCULOSKELETAL PAIN

Shoulder pain, with a reported preva-

lence ranging from 30 to 60%, is most common in tetraplegics. Common shoulder pathologies in SCL patients include shoulder joint degeneration and chronic impingement syndrome with rotator cuff tears, subacromial bursitis, or supraspinatus tendinopathy (2,3).

Musculoskeletal pain in elbow, wrist, and hand related to extreme joint postures, high mechanical stresses, and repetitive movements during wheelchair propulsion is also frequently reported (review in (3)).

Neck pain in SCL patients has been attributed to overuse, inflammatory processes, mechanical spinal instability, and hypoperfusion of tonically active neck muscles. A recent study found patients with orthostatic hypotension to have a higher frequency of neck pain, which was made worse by assuming the upright posture and exercise and relieved by lying flat (4). A "steal" effect was proposed to explain it, and strategies to treat orthostatic hypotension may be helpful.

Vertebral column pain due to secondary changes following fractures, dislocations, and fixation, scoliosis, mechanical instability, and osteoporosis may also occur (3).

Other types of musculoskeletal pain conditions include headache and heterotopic ossification.

MANAGEMENT

Controlled studies and studies comparing different treatments strategies are lacking. Chronic musculoskeletal pain is often best treated by non-surgical means and should be directed at

treating pain, inflammation, weakness, instability, stiffness, posture, and obesity (3). *Physiotherapy*, well balanced exercise programs with strengthening and stretching exercises, wheelchair and home/work modifications, and *education* in basic biochemical principles and posture control may be helpful in prevention and treatment of musculoskeletal pain (3). Prevention is important and therefore these strategies should be incorporated in early rehabilitation.

Reports on the use of *TENS and acupuncture* in SCL patients are limited and controlled trials are not available (5,6). Patients with muscle pain may experience pain relief. It has been recommended that the use of TENS should be relatively contraindicated in tetraplegics in the first two years post injury since postural detrusor-sphincter dyssynergia have been reported (7).

Pain due to muscle spasms is best relieved by *spasmolytics*.

VISCERAL PAIN

Visceral pain usually presents as dull or cramping abdominal uncomfortable and painful sensations, which may be associated with nausea and autonomic reactions. The pain may be related to secondary bowel, bladder or kidney problems, but it is likely that visceral pain may occur in the absence of any abdominal organ dysfunction. There is little or no information on the mechanisms and treatment of visceral pain after SCL.

NEUROPATHIC PAIN

Neuropathic pain is divided into "above-level", "at-level", and "below-level" types, where "level" refers to the level of the spinal cord that was injured (1). Above level neuropathic pain

includes pain due to compressive mononeuropathies (particularly carpal tunnel syndrome) and complex regional pain syndromes. At level pain may be caused by spinal cord trauma, nerve root compression, or syringomyelia. Below level pain is caused by the spinal cord trauma. Neuropathic pain is seen in an area with sensory deficits and may be accompanied by dysaesthesia (unpleasant abnormal sensations e.g. tingling, pricking, and burning), allodynia (the elicitation of pain in the affected area by non-noxious stimulation with light touch or innocuous cold or warmth), and hyperalgesia (increased response to a painful stimulus). Neuropathic pain may have a late onset (up to a year or more after SCL), and it is often long lasting with a low tendency to spontaneous recovery. Concurrent infections or other illnesses may aggravate the pain. The pathophysiology underlying at and below level neuropathic pain is unclear. Proposed mechanisms include loss of intraspinal or descending inhibitory mechanisms, neuronal hyperexcitability and spontaneous activity of spinal or thalamic neurons, loss of balance between different sensory channels, and structural changes in spinal and higher deafferented neurons (8).

PHARMACOLOGICAL MANAGEMENT

Treatment of neuropathic pain is usually difficult and there is at present no established treatment algorithm for neuropathic SCL pain (9). Presently, there is little evidence for targeting specific pain characters with specific drugs. Randomised controlled trials (RCT) on pharmacological treatment after SCL are summarised in the Table. Because of the different pain mechanisms that seem to be involved in SCL

pain, pharmacological treatment is often only able to reduce pain partially, and combination of different therapies may be appropriate.

Antiepileptic drugs

Lamotrigine stabilizes sodium channels and suppresses glutamate release. Lamotrigine 400 mg daily showed no effect on neuropathic pain in SCL patients, although this RCT indicated that lamotrigine might have an effect on spontaneous pain in patients who also have allodynia or hyperalgesia (10). Adverse events include rash, dizziness, somnolence, and other CNS-related symptoms. Slow dose escalation (initially increasing the dose with 25 mg every two weeks, later with 50 mg every week to 400 mg daily) is recommended to minimize the risk of serious hypersensitivity reactions. Gabapentin, which blocks a subtype of calcium channels and has a partial blocking action on sodium channels, is frequently used in neuropathic pain conditions. The most common adverse events are dizziness, somnolence, confusion and ataxia. Gabapentin is slowly increased by 300 mg every to every other day up to 2400-3600 mg daily administered in three divided doses with reduced doses in patients with decreased renal function.

Antidepressants

Tricyclic antidepressants (TCA) have a wide range of pharmacological actions, but the main mechanism by which TCAs relieve neuropathic pain is thought to be by blocking the reuptake of norepinephrine and serotonin. Side effects are mainly attributed to sedative and anticholinergic actions (drowsiness, dry mouth, urinary retention, and constipation). TCAs are contraindicated in patients with heart failure and glaucoma and ECG should be monitored. More selective antide-

pressants (e.g. selective serotonin reuptake inhibitors (SSRI)) have fewer side effects, but they seem to be less effective than TCAs with the full range of actions (11). Both TCAs and SSRIs have been reported to exacerbate spasticity (12,13). Although the two RCTs of antidepressants in the treatment of pain associated with SCL were negative, TCAs (e.g. imipramine) are still considered to be the first drug of choice for many neuropathic pain conditions (11). However, side effects often limit the use of these agents. Starting with 25 mg daily, imipramine is increased by 25 mg every two weeks usually up to 150 mg daily in one to two divided doses, but effective doses vary among patients.

Monitoring plasma drug levels may be useful (optimal plasma levels of imipramine plus desipramine is 300-750 nM).

Opioids

Studies have shown efficacy of opioids in different neuropathic pain states (11), but their use is controversial for chronic non-malignant pain conditions. Only intravenous administration of opioids have been evaluated in SCL patients with conflicting results (see Table).

Cannabinoids

The cannabinoid system, which is very similar to the opioid system, might be a future target for therapy of neurological disorders including pain. Presently, it is not well documented that cannabinoids have an effect on chronic pain.

NMDA receptor antagonists and sodium channel blockers

N-methyl-D-aspartate (NMDA) receptors antagonists and sodium channel blockers have been used to treat different neuropathic pain states, although the clinical use of these

Randomized controlled trials of pharmacological treatment of pain in SCL

Study	Active drug	Dose and study duration	No. total / SCL	Outcome
Davidoff et al. 1987 (15)	Trazodone hydrochloride	6 weeks: 150 mg/day	18*	Tra = pla
Chiou-Tan et al. 1996 (16)	Mexiletine	2 x 4 weeks: 450 mg/day	11**	Mex = pla
Drewes et al. 1994 (17)	Valproate	2 x 3 weeks: final dose 600-2400 mg/day	20**	Val = pla
Finnerup et al. 2002 (10)	Lamotrigine	2 x 9 weeks, final dose 200-400 mg/day	22**	Ltg = pla [Incomplete: Ltg > pla]
Cardenas et al. 2002 (13)	Amitriptyline	6 weeks	84*	Ami = pla
Attal et al. 2000 (18)	Lidocaine i.v.	5 mg/kg (over 30 minutes)	16/10**	Lid > pla
Canavero et al. 1995 (19)	Propofol i.v.	bolus 0.2 mg/kg	32/8**	Pro > pla
Attal et al. 2002 (20)	Morphine i.v.	9-30 mg infusion	15/9**	Morp = pla
Eide et al. 1995 (21)	Ketamine i.v. or alfentanil i.v.	Ket: bolus 60 µg/kg + 6 µg/kg/min (17-20 min) Alf: bolus 7 µg/kg + 0.6 µg/kg/min (17-20 min)	9**	Ket > pla Alf > pla Ket = alf
Loubser et al. 1991 (22)	Lidocaine s.a.	50-100 mg (titrated every 5 minutes in 25 mg aliquots)	21**	Lid > pla
Siddall et al. 2000 (23)	Morphine i.t., clonidine i.t., or the combination	Mor: bolus 0.2-1.5 mg Clo: bolus 50-100 µg or 300-500 µg (over 6 hours) Comb: half of each dose	15**	Mor = pla Clo = pla Mor+clo > pla
Herman et al. 1992 (24)	Baclofen i.t.	bolus 50 µg	7**	Bac > pla

Abbreviations: i.v. = intravenous; i.t. = intrathecal; s.a. = subarachnoidal; pla = placebo. * parallel study design; ** cross-over study design.

agents have been limited because of unwanted psychotropic effects.

SURGICAL PROCEDURES

There is no evidence for long-term efficacy of deep brain stimulation and

spinal cord stimulation for SCL pain (5). Ablative procedures including dorsal root entry zone (DREZ) lesions may have some effect on those with at-level and unilateral pain (5). These proce-

dures carry the risk for exacerbation of weakness and sensory loss and new or increased neuropathic pain. A new method of computer assisted DREZ microcoagulation which records and ablates areas of focal hyperactivity in several segments of the spinal cord is suggested to have a higher success rate than standard DREZ (14), but the long term efficacy and side-effects of this method needs to be evaluated.

PSYCHOSOCIAL PROBLEMS

Chronic pain syndromes may cause increased disability, social limitations, cognitive, affective, and depressive symptoms; and these factors can contribute to increased pain. Careful evaluation of these aspects is important, and non-pharmacological types of treatment such as physical therapy, psychological support with cognitive behavioural treatment etc. are important. High levels of activity should be encouraged.

REFERENCES

1. Siddall PJ, Yeziarski RP, Loeser JD. Pain following spinal cord injury: Clinical features, prevalence, and taxonomy. *IASP newsletter* 2000;3:3-7.
2. Lal S. Premature degenerative shoulder changes in spinal cord injury patients. *Spinal Cord* 1998;36:186-9.
3. Goldstein B. Musculoskeletal conditions after spinal cord injury. *Phys Med Rehabil Clin N Am* 2000;11:91-108.
4. Cariga P, Ahmed S, Mathias CJ, Gardner BP. The prevalence and association of neck (coat-hanger) pain and orthostatic (postural) hypotension in human spinal cord injury. *Spinal Cord* 2002;40:77-82.
5. Finnerup NB, Yeziarski RP, Sang CN, Burchiel KJ, Jensen TS. Treatment of spinal cord injury pain. *Pain Clinical Updates* 2001;9: 1-6.
6. Nayak S, Shiflett SC, Schoenberger NE, Agostinelli S, Kirshblum S, Averill A, Cotter AC. Is acupuncture effective in treating chronic pain after spinal cord injury? *Arch Phys Med Rehabil* 2001;82:1578-86.
7. Leyson JF, Stefaniwsky L, Martin BF. Effects of transcutaneous nerve stimulation on the vesicourethral function in spinal cord injury patients. *J Urol* 1979;121:635-9.
8. Vierck CJ, Siddall P, Yeziarski RP. Pain following spinal cord injury: Animal studies and mechanistic studies. *Pain* 2000;89:1-5.
9. Finnerup NB, Johannesen IL, Sindrup EH, Bach FW, Jensen TS. Pharmacological Treatment of Spinal Cord Injury Pain. In: Burchiel KJ, Yeziarski RP, eds. *Spinal Cord Injury Pain: Assessment, Mechanisms, Management. Progress in Pain Research and Management.* Seattle: IASP Press, 2002: 341-351.
10. Finnerup NB, Sindrup SH, Bach FW, Johannesen IL, Jensen TS. Lamotrigine in spinal cord injury pain: a randomized controlled trial. *Pain* 2002;96:375-83.
11. Sindrup SH, Jensen TS. Efficacy of pharmacological treatments of neuropathic pain: an update and effect related to mechanism of drug action. *Pain* 1999;83:389-400.
12. Stolp-Smith KA, Wainberg MC. Antidepressant exacerbation of spasticity. *Arch Phys Med Rehabil* 1999;80:339-42.
13. Cardenas DD, Warms CA, Turner JA, Marshall H, Brooke MM, Loeser JD. Efficacy of amitriptyline for relief of pain in spinal cord injury: results of a randomized controlled trial. *Pain* 2002;96:365-73.
14. Edgar RE, Best LG, Quail PA, Obert AD. Computer-assisted DREZ microcoagulation: posttraumatic spinal deafferentation pain. *J Spinal Disord* 1993;6:48-56.
15. Davidoff G, Guarracini M, Roth E, Sliwa J, Yarkony G. Trazodone hydrochloride in the treatment of dysesthetic pain in traumatic myelopathy: a randomized, double-blind, placebo-controlled study. *Pain* 1987;29:151-61.

16. Chiu-Tan FY, Tuel SM, Johnson JC, Priebe MM, Hirsh DD, Strayer JR. Effect of mexiletine on spinal cord injury dysesthetic pain. *Am J Phys Med Rehabil* 1996;75: 84-7.
17. Drewes AM, Andreassen A, Poulsen LH. Valproate for treatment of chronic central pain after spinal cord injury. A double-blind cross-over study. *Paraplegia* 1994;32:565-9.
18. Attal N, Gaude V, Brasseur L, Dupuy M, Guirimand F, Parker F, Bouhassira D. Intravenous lidocaine in central pain: a double-blind, placebo-controlled, psychophysical study. *Neurology* 2000;54:564-74.
19. Canavero S, Bonicalzi V, Pagni CA, Castellano G, Merante R, Gentile S, Bradac GB, Bergui M, Benna P, Vighetti S, et al. Propofol analgesia in central pain: preliminary clinical observations. *J Neurol* 1995;242:561-7.
20. Attal N, Guirimand F, Brasseur L, Gaude V, Chauvin M, Bouhassira D. Effects of IV morphine in central pain: A randomized placebo-controlled study. *Neurology* 2002;58:554-63.
21. Eide PK, Stubhaug A, Stenehjem AE. Central dysesthesia pain after traumatic spinal cord injury is dependent on N-methyl-D-aspartate receptor activation. *Neurosurgery* 1995;37:1080-7.
22. Loubser PG, Donovan WH. Diagnostic spinal anaesthesia in chronic spinal cord injury pain. *Paraplegia* 1991;29:25-36.
23. Siddall PJ, Molloy AR, Walker S, Rutkowski SB. The efficacy of intrathecal morphine and clonidine in the treatment of pain after spinal cord injury. *Anesth Analg* 2000;91:1-6.
24. Herman RM, D'Luzansky SC, Ippolito R. Intrathecal baclofen suppresses central pain in patients with spinal lesions. A pilot study. *Clin J Pain* 1992;8:338-45.

POSTTRAUMATIC SYRINGOMYELIA

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SUMMARY

A posttraumatic syringomyelia (PTS) is a cystic expansion in the tissue of the spinal cord seen after a traumatic spinal cord injury. The incidence of PTS is reported at 20-50%. Symptoms may vary considerably, with new or increasing sensibility disturbances or pareses, increased spasticity, pain or hypersensitivity, autonomic dysreflexia, new bladder- or bowel-management problems, or hyperhidrosis. The diagnosis may be difficult and the symptoms have often been misinterpreted. There have been reported cases with PTS diagnosed from 3 weeks to 33 years after injury. The pathophysiological mechanism is not known. But a two-stage process may include an initial cavitation and a secondary extension of the cavity. The treatment is still controversial. In the acute stage alignment of the vertebral bodies should be achieved to avoid spinal canal stenosis. If a PTS develop correction of the obstruction of the subarachnoid space and thereby opening of the CSF pathways is preferred. Efforts should be directed to achieve lysis of adhesions tethering the spinal cord. The results of surgical intervention have so far not been very encouraging. There seems to be a general agreement about the importance of early treatment to be able to stop the further development of the syrinx with even more deterioration of the patients' neurology or abilities. A regular and frequent follow up of the patient with spinal cord injury is the best way to ensure that PTS is dia-

gnosed and managed early in order to avoid further disability.

DEFINITION

Syringomyelia and hydromelia are both cavitations in the spinal cord. *Hydromyelia* is a cystic expansion of the central canal of the spinal cord. If the cystic expansion is situated in the tissue of the spinal cord it is a *syringomyelia* (1), which can be seen following intraspinal tumours, arachnoiditis after infections, radiation necroses and vascular malformations, and not least after a traumatic spinal cord injury.

There is a certain agreement to define *posttraumatic syringomyelia (PTS)*, also called progressive post-traumatic cystic myelopathy, as a cystic expansion in the spinal cord with an extension corresponding to at least two vertebrae, and which has the signal intensity as liquor, i.e. low on T1 weighted and high on T2 weighted images. Cavitations in the spinal cord with an extension of less than the height of two vertebrae are called cysts. The cavitation is often decentral in the spinal cord but may communicate with the central canal (2,3).

INCIDENCE

A case of PTS was first reported in 1943. The incidence of PTS was prior to the introduction of MRI usually given as 0.3-3.5%. After the more widespread use of MRI a marked increase of this diagnosis has been experienced with PTS incidences reported at 20-50%,

when counting symptomatic as well as asymptomatic cases.

SYMPTOMS

Clinical symptoms related to the development of PTS may vary considerably in individuals who often have neurological deficits. In many instances symptoms never develop. When symptoms are experienced it is often with pain, which not always are localised to specific dermatomes or nerve roots. New or increasing sensibility disturbances, also dissociated, development of new pareses, increased spasticity, pain or hypersensitivity, autonomic dysreflexia, new bladder- or bowel-management problems, or hyperhidrosis at various parts of the body are reported (4,5,6).

DIAGNOSIS

The diagnosis may be difficult and the symptoms have often been misinterpreted. One of the reasons is the large variation in symptoms, and the varying time post injury symptoms are observed. There have been reported cases with PTS diagnosed from 3 weeks to 33 years after a spinal cord injury (7, 8). The problem is not least seen when patients after an incomplete spinal cord injury is discharged with normal or nearly normal function (ASIA E), but several years later deteriorate to end up with e.g. complete tetraplegia (9,10).

PATHOPHYSIOLOGY

The pathophysiological mechanism for the development of PTS is not known. But the most attractive theory is still the one suggested by Williams et al. (11), and later supported by other investigators (2, 5,12,13). This two stage process include an initial cavita-

tion either by liquefaction of hematomyelia, myelomalacic necrosis, release of lysosomal enzymes, or by ischemia and phenomena of tethered cord at the origin of traction-distension produced by arachnoiditis. The secondary extension of the cavity is thought to be a consequence of transmission of ongoing pressure pulses from the epidural venous system. Subarachnoid scarring and adhesions around the syrinx may cause blockage of the normal cerebrospinal fluid (CSF) wave, which is being created by the epidural venous pulsations. The pulse pressure is transmitted to the PTS cavity. This repetitive 'sloshing' and 'sucking' of CSF may cause the extension of the syrinx in rostral and caudal direction. Therefore activities, which increase intrathoracic or abdominal pressure, like cough, straining, and weightlifting exercises, may expand the cyst.

During the later years there have been an increasing focus on a chronic mechanical stress to the spinal cord in the development of PTS. Perrouin-Verbe et al. (2) showed the occurrence of a syrinx was significantly correlated with spinal canal stenosis. Abel et al. (14) likewise found there were indications that patients with more than 15 degrees of posttraumatic kyphosis and more than 25% of stenosis were twice as likely to develop PTS.

TREATMENT

Due to the lack of a precise pathophysiological mechanism the treatment is still controversial.

In the acute stage when the spinal cord injured patient is admitted to a hospital a plain x-ray and a MRI (CT-scan) of the spine should be taken. In case of instable fractures and/or luxations, this should be attended at once so that alignment of the vertebral bodies is

achieved whereby stenosis of the spinal canal is avoided.

The initial treatment of the vertebral injury could be the first step in the prevention of the development of PTS.

If *at a later time* a PTS develop several surgical procedures have been used in the last four decades, including drain from the syrinx to the subarachnoidal, pleural, or peritoneal space; spinal cord untethering; duroplasty (pseudomeningocele); myelotomy; cord transection; omental grafting; and combinations of the above procedures.

Treatment has evolved over the years. Drainage-procedures were preferred earlier but seem to have little or no place in surgical treatment of PTS today (15). Omental grafting has been abandoned (16).

Modern surgical treatment of PTS has come to reflect the treatment of hindbrain related syringomyelia with correction of the obstruction of the subarachnoid space and thereby opening of the CSF pathways. Efforts should be directed to achieve lysis of adhesions tethering the spinal cord (2,9,16,17).

The results of surgical intervention have so far not been very encouraging. Only seldom are improvements seen in the patients' neurological deficits or their general activities of daily living. However, there seems to be a general agreement about the importance of early treatment to be able to stop the further development of the syrinx with even more deterioration of the patients' neurology or abilities (2,4,6,9,17,18,19,20,21).

FUTURE

In the initial stage of a spinal cord injury with a fracture it seems important to make a sufficient reduction of the vertebral fracture to avoid spinal

canal stenosis, which may be part of the genesis for PTS (2).

Whether obliteration of the PTS with neural tissue transplantation is going to be a treatment of choice remain to be determined (21,22). Maybe grafting of gene-modified autologous neural tissue will be a possibility.

In addition, Biyani and Masry (12) state in their literature review, that the regular and frequent follow up of the patient with spinal cord injury for this complication, as well as other complications, is the best way to ensure that PTS is diagnosed and managed early in order to avoid further disability. This could imply MRI at regular intervals, more often in the first years post-injury, and more seldom later on if no signs of PTS are observed.

REFERENCES

1. Milhorat TH, Johnson RW, Milhorat RH, Capocelli AL, Pevsner PH. Clinicopathological correlation in syringomyelia using axial magnetic imaging. *Neurosurgery* 1995;35:206-13.
2. Perrouin-Verbe B, Lenne-Aurier K, Robert R, et al. Posttraumatic syringomyelia and posttraumatic spinal canal stenosis: A direct relationship: Review of 75 patients with spinal cord injury. *Spinal Cord* 1998;36:137-43.
3. Wang D, Bodley R, Sett P, Gardner B, Frankel H. A clinical magnetic resonance imaging study of the traumatised spinal cord more than 20 years following injury. *Paraplegia* 1996;34:64-81.
4. Falci SP, Lammertse DP, Best L et al. Surgical treatment of posttraumatic cystic and tethered spinal cords. *J Spinal Cord Med* 1999;22:173-81.
5. Kramer KM, Levine AM. Posttraumatic syringomyelia. A review of 21 cases. *Ci Lkin Orthop Rel Res* 1997;334:190-9.
6. Lee TT, Alameda GJ, Gromelski EB, Green BA. Outcome after surgical treatment of progressive posttraumatic cystic myelopathy. *J Neurosurg (Spine 2)* 2000;92:149-154.

7. Bains RS, Althausen PL, Gitlin GN, Gupta MC, Benson DR. The role of acute decompression and restoration of spinal alignment in the prevention of post-traumatic syringomyelia. Case report and review of recent literature. *Spine* 2001;26:E399-E402.
8. Hida K, Iwasaki Y, Imamura H, Abe H. Post-traumatic syringomyelia: Its characteristic magnetic resonance imaging findings and surgical management. *Neurosurgery* 1994;35:886.
9. Nielsen OA, Biering-Sørensen F, Bötzel U et al. Clinical case of the month. Posttraumatic syringomyelia. *Spinal Cord* 1999;37:680-4.
10. Terré R, Vallès M, Vidal J. Post-traumatic syringomyelia following complete neurological recovery. *Spinal Cord* 2000;38:567-70.
11. Williams B, Terry AF, Jones HWF, McSweeney T. Syringomyelia as a sequel to traumatic paraplegia. *Paraplegia* 1981;19:67-80.
12. Biyani A, El Masry WS. Post-traumatic syringomyelia: a review of the literature. *Paraplegia* 1994;32:723-31.
13. Schurch B, Wichmann W, Rossier AB. Post-traumatic syringomyelia (cystic myelopathy): a prospective study of 449 patients with spinal cord injury. *J Neurol Neurosurg Psychiatry* 1996;60:61-7.
14. Abel R, Gerner HJ, Smit C, Meiners T. Residual deformity of the spinal canal in patients with traumatic paraplegia and secondary changes of the spinal cord. *Spinal Cord* 1999;37:14-9.
15. Backe HA, Betz RR, Mesgarzadeh M, Beck T, Clancy M. Post-traumatic spinal cord cysts evaluated by magnetic resonance imaging. *Paraplegia* 1991; 29: 607-12.
16. Sgouros S, Williams B. Management and outcome of posttraumatic syringomyelia. *J Neurosurg* 1996;85:197-205.
17. Batzdorf U, Klekamp J, Johnson JP. A critical appraisal of syrinx cavity shunting procedures. *J Neurosurg* 1998;89:382-8.
18. Klekamp J, Batzdorf U, Samii M, Bothe HW. Treatment of syringomyelia associated with arachnoid scarring caused by arachnoiditis or trauma. *J Neurosurg* 1997;86:233-40.
19. Ronen J, Catz A, Spasser R, Gepstein R. The dilemma in post-traumatic syringomyelia. *Disabil Rehab* 1999;21:455-7.
20. Schaan M, Jaksche H. Comparison of different operative modalities in post-traumatic syringomyelia: preliminary report. *Eur Spine J* 2001;10:135-40.
21. Wirth III ED, Reier PJ, Fessler RG, et al. Feasibility and safety of neural tissue transplantation in patients with syringomyelia. *J Neurotrauma* 2001;18:911-29.
22. Falci S, Holtz A, Akeson E, Azizi M, Ertzgaard P, Hultling C, Kjaeldgaard A, Levi R, Ringden O, Westgren M, Lammertse D, Seiger A. Obliteration of a posttraumatic spinal cord cyst with solid human embryonic spinal cord grafts: First clinical attempt. *J Neurotrauma* 1997;14:875-84.

OSTEOPOROSIS

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SUMMARY

Spinal cord injury (SCI) patients display a specific form of demineralization typified by an exclusively sublesional topography. The demineralisation is the same in both tetraplegics and paraplegics. This demineralization predominates on the long bones with a partial preservation of the trabecular bone of the lumbar spine. The most affected is the trabecular metaphyseal-epiphyseal areas of the distal femur and the proximal tibia.

Low bone mass is a major and the best characterized determinant of fractures occurrence. Currently, the best method of measuring low bone mass (bone density) is Dual-energy X-ray Absorptiometry (DXA).

Risk factors for osteoporosis includes cigarette smoking, excess alcohol consumption and vitamin D deficiency, among others.

The types of interventions to improve bone density in patients with SCI that have been reported have involved either electrical stimulation of muscles or pharmacologic targeting of bone metabolism with bisphosphonates.

Any intervention to prevent the occurrence of osteoporosis should be initiated as soon as possible after injury, before bone density loss is detectable.

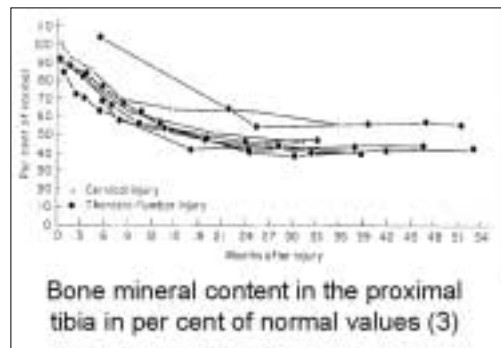
Fractures of the lower extremity should be treated with interlocking, intramedullary nails or splints, depending on site of fracture.

Osteoporosis is well known in spinal

cord-injured (SCI) patients. Although the terms *immobilization* and *disuse* have been applied to describe these changes in bone density, the pathophysiology is likely to be complex. In the acute injury period, there is an increased urinary excretion of calcium and hydroxyproline and accelerated bone remodelling. The increase in urinary calcium cannot be attributed solely to lack of exercise or to prolonged inactivity because a greater degree of hypercalciuria is found in SCI patients than in age-matched controls after prolonged bed rest.

This bone demineralisation has an important functional consequence due to risk of pathologic fractures of the lower limbs. The preferential site of these fractures is the distal femur (lower third) and the proximal tibia (upper third) (1). A highly increased risk for femur fractures (RR = 23.4, $p < 0.001$), and lower leg fractures (RR = 5.2, $p < 0.001$) compared to controls has been found (2).

Previous absorptiometric studies have shown an important decrease of bone mineral density. The average amount of bone loss during the first year after



injury is about 40 % to 70 %, depending on the site and age of the patient (3,4).

PATHOPHYSIOLOGY

Low bone mass is the most important predisposing factor for osteoporotic fractures. Bone mass is affected by peak bone mass and the degree of subsequent bone loss. These two processes are regulated at the level of the bone remodelling units, which in turn are responsive to an interaction between genetic and environmental factors.

BONE REMODELLING

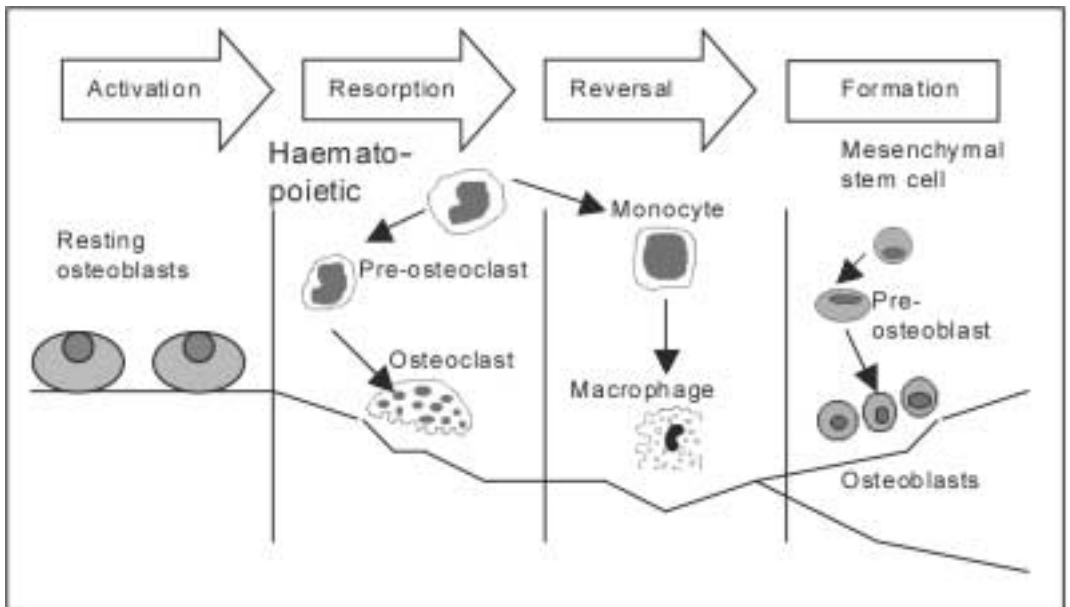
A knowledge of the process of bone remodelling is important in order to understand how bone mass can be altered by heritable and environmental influences. Skeletal remodelling predominates once longitudinal growth ceases. The cyclical process occurs in microscopic elements called “remodelling” units (Figure). Bone remodelling begins with bone resorption and ends

with new bone formation. Cells called osteoclasts, which originate from the monocyte-macrophage lineage, carry out bone resorption. New bone is formed by osteoblasts, cells of the fibroblast-stromal lineage that produce several bone matrix proteins and synthesize a lattice for subsequent mineralization.

Each remodelling cycle is balanced – resorption equals formation – and last between 90 and 130 days. Maintenance of bone mass during remodelling ensures a ready source of calcium for the body, and a persistent reservoir of stored calcium. However, remodelling cycles can become imbalanced, and over several cycles this can result in significant bone loss.

RISK FACTORS FOR OSTEOPOROSIS

A number of factors have been identified that increase the likelihood of developing osteopenia (low bone mass).



- Age
- Female gender
- Hypogonadism (incl. Premature menopause)
- Glucocorticoid therapy
- Previous fragility fracture
- Low body weight
- Cigarette smoking
- Excess alcohol consumption
- Low dietary calcium intake
- Vitamin D deficiency
- Late menarche
- Physical inactivity
- High caffeine intake
- Maternal history of hip fracture

Osteopenia is a major and the best characterized determinant of fractures occurrence. Therefore, the risk factors noted for osteopenia also increase risk for fractures. Falls and propensity to fall are other major considerations with respect to fracture risk (5).

METHODS OF MEASURING BONE DENSITY

A number of techniques are now available to measure bone mass at various skeletal sites. The values obtained from these measurements represent the bone mineral density (BMD). Currently, these provide the best assessment of fracture risk and have an established role in clinical practice.

Dual-energy X-ray Absorptiometry (DXA) is widely regarded as the diagnostic method of choice. In this test, differential absorption of two X-ray frequencies by soft tissue and bone enables bone mass to be calculated. It is applied to both the axial and appendicular skeleton, with the femoral neck, lumbar spine and radius being the sites most commonly assessed. The measurements can take as little as 1-2 minutes and are achieved at a very low dose of radiation.

Quantitative computed tomography

(QCT), though available in few centres only, can be used to measure bone mass both in the axial and appendicular skeleton. The radiation dose is significantly higher than for DXA.

Quantitative ultrasound (QUS) is most commonly applied to the os calcis and reflects both bone mass and architecture. It is portable, relatively cheap and free of ionizing radiation.

Radiography is an insensitive method for assessing bone loss but plays a major role in the diagnosis of fracture.

Biochemical markers of bone turnover are a number of products of collagen breakdown or of bone cells. These can be measured in the serum or urine and provide information about the rate of bone loss at the time of measurement. At present, however, their use is mainly restricted to research applications. The wide variation in levels of markers within and between individuals means that they are unsuitable as diagnostic tools.

PREVENTION OF OSTEOPOROSIS AND FRACTURES

Osteoporosis prevention should take place from early childhood into old age. Many of the risk factors listed above are relevant. Avoidance of smoking and excessive alcohol consumption is among these. Later in life vitamin D supplementation has shown to reduce fracture incidence in some elderly populations. The types of interventions to improve bone density in patients with SCI that have been reported have involved tricyclic stimulated cycling exercise training for 12 months (3 days per week) in 10 motor-complete SCI subjects increased the BMD of the proximal tibia by 10%, but those improvements were lost after 6 more months of reduced-frequency exercise (one per week). No changes

were observed in the lumbar spine or in the femoral neck. The lack of effect of electrically induced exercise upon the regions about the hip, as well as the positive effects about the knee is consistent in several studies.



Electrically stimulated cycling exercise training for spinal cord lesioned individual

The effect of standing on bone has been reported in several other studies, but verticalization and the gate with orthosis are insufficient means of prevention.

Treatment with bisphosphonates is known to be powerful inhibitors of bone resorption by osteoclasts. The bisphosphonates have the ability to reach bone directly with almost no extraskeletal side-effects. Several studies have shown decrease in bone loss using different kind of bisphosphonates. A study using alendronate 10 mg per day orally in 3 years, improved femoral neck density 10-18% in some subjects (unpublished data).

Side-effects due to oral alendronate are limited to the gastro-oesophageal tract and alendronate should therefore be swallowed with 180–250 mL of water

in the morning, and the patient should stay up for at least 30 minutes and until breakfast has been eaten. Due to poor bioavailability alendronate is given on an empty stomach with water only.

TREATMENT OF OSTEOPOROTIC FRACTURES

Patients with established SCI have numerous altered physiologic factors that affect the management of long-bone fractures. Significant spasticity may preclude use of non-operative management for shaft fractures. Cast may cause skin breakdown and splints may not provide adequate immobilization, and osteoporosis may prevent adequate fixation.

In displaced femoral neck fractures prosthetic replacement may be necessary, although dislocation after surgery may occur as well as late subluxation and dislocation.

In intertrochanteric fractures an intramedullary hip fixation is currently the preferred operative method.

The treatment of choice for displaced femoral fractures or non-displaced fracture, which lose reduction during non-operative methods, is interlocking, intramedullary rodding. Locking is necessary because the femoral canals



Femur fracture in a spinal cord lesioned individual.

are wide and rotational deformities as well as non-union cannot be prevented with standard nails.

Distal femur/proximal tibia fractures are best treated with well-padded splints or a knee immobilizer. Long leg casts are not recommended.

Tibial shaft fractures can be treated non-operatively if minimally or non-displaced. Displaced fractures require an interlocking, intramedullary nail.

Distal tibia, ankle, and foot fractures respond to splinting (7).

REFERENCES

1. Keating JF, Kerr M, Delargy M. Minimal trauma causing fractures in patients with spinal cord injury. *Disabil Rehabil* 1992;14:108-9.
2. Vestergaard P, Krogh K, Rejnmark L, Mosekilde L. Fracture rates and risk factors for fractures in patients with spinal cord injury. *Spinal Cord* 1998;36:790-6

3. Biering-Sørensen F, Bohr H, Schaadt O. Longitudinal study of bone mineral content in the lumbar spine, the forearm and the lower extremities after spinal cord injury. *Eur J Clin Invest* 1990;20:330-5

4. Dauty M, Perrouin-Verbe B, Maugars Y, Dubois C, Mathe JF. Supralesional and sublesional bone mineral density in spinal cord-injured patients. *Bone* 2000;27:305-9

5. Wark JD. Osteoporotic fractures: background and prevention strategies. *Maturitas* 1996;23:193-207

6. Mohr T, Pødenphant J, Biering-Sørensen F, Galbo H, Thamsborg G, Kjær M. Increased bone mineral density after prolonged electrically induced cycle training of paralysed limbs in spinal cord injured man. *Calcif Tissue Int* 1997;61:22-5

7. Garland D, Shokes L. Management of long-bone fractures in patients with SCI. In Eltorai IM, Schmitt JK eds.: *Emergencies in chronic spinal cord injury patients*. Eastern Paralyzed Veterans Association, pp163-71, 2001

EXERCISE AND PHYSICAL TRAINING MODALITIES FOR THE SPINAL CORD INJURED INDIVIDUAL: POSSIBLE CLINICAL IMPLICATIONS TO COUNTER- ACT LIFE-STYLE DISORDERS

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SUMMARY

Spinal cord injury (SCI) causes dramatic neuromuscular changes in the paralysed muscle. These changes include reduction in muscular force and in cross-sectional area of skeletal muscle as well as increased intramuscular connective tissue and adipose tissue. In addition, the muscular content of oxidative enzymes as well as glucose transporter protein, together with insulin sensitivity decreases dramatically. Electrical stimulation (ES) can beneficially increase glycolytic and oxidative enzymes in muscle and cause a muscle fibre type shift towards slower and more oxidative fibres. This is accompanied by improved insulin sensitivity and enhanced aerobic capacity of the whole body. This contributes to counteract the risk for inactivity associated diseases (diabetes, adiposity and cardiovascular disease) in individuals with SCI. The cross-sectional area of muscle fibres and the whole muscle improves with ES and may contribute to counteract development of decubitus and bone fractures.

Exercise for the spinal cord injured (SCI) individual serves many purposes; the most important being the ability to reduce the morbidity and mortality rates evolving from the secondary medical sequelae after disrup-

tion to the spinal cord. Improvements in cardiovascular fitness have been limited by the use of the smaller upper body musculature (1), the inability for blood redistribution below the level of the injury (1,2) and a dysfunctional autonomic system (3,4).

ES of the paralysed lower limbs has gained acceptance as a valid exercise modality for the SCI individual. Stimulation of the lower limbs can increase muscle strength and muscle oxidative capacity (5-7), improve cardiovascular conditioning (8,9), enhance bone mass (10), reduce the incidence of decubitus ulcers (11) and improve self image and quality of life (12).

LOWER PARALYSED LIMBS AND ELECTRICAL STIMULATION

MUSCLE HYPERTROPHY

Muscle atrophy is one of the most dramatic changes in skeletal muscle after a SCI. Following a SCI, there is a progressive muscle fibre atrophy of all fibre types accompanied by the proliferation of fibrous interstitial tissue and fatty infiltration. Dark angular atrophic fibres, target-targetoid fibres and proliferative changes of the sarcoplasmic reticulum have also been reported during this time (13).

ES training can significantly reduce the amount of atrophy observed in indi-

viduals with SCI, although this has not been shown in all studies. Studies involving ES training for three (14) and six months (15) have reported no significant increase in fibre cross-sectional area, while increases of 294% and 142% have been noted mainly in the type II fibres after ten weeks and eight months of ES training, respectively (7,16). In addition, ES training when introduced within the first four weeks of injury has been shown to reduce the level of disuse atrophy experienced within the paralysed muscles (6).

MUSCLE CONTRACTILE CHARACTERISTICS

The twitch and tetanus from human muscle paralysed for at least three years have been shown to have contractile properties during repetitive activation that is characteristic of a fast-fatigable whole muscle (17). The outcome however is not consistent across studies (18). Neurophysiological measurements after ES training demonstrate that the contractile properties of paralyzed human muscle revert toward the normal values seen in slow muscles, with slower contraction times (19) and greater resistance to fatigue (18,20).

MUSCLE FIBRE TYPE TRANSFORMATION

The vastus lateralis muscle shows a heterogeneous fibre type population in sedentary able-bodied individuals, however within 12 months of a SCI, the percentage of type IIX fibres can increase from 20% pre-injury to 100% (6,21). Similarly, the myosin heavy chain composition of individual fibres shows a significant alteration in the paralysed muscle with 41% of fibres co-expressing MHC IIa and MHC IIb in the vastus lateralis muscle after six months of injury (22).

ES has been shown to return skeletal

muscle fibre types to within ranges seen in able-bodied controls (6,7,15), however the alteration seen in the paralysed muscle is not always consistent between individuals and is not shown in all studies (14). Importantly, ES training has also been shown maintain the heterogeneity of the fibre type population if applied during the acute phase of the injury (6).

METABOLIC ENZYME ACTIVITY

Only limited data exists on the effect of ES training on paralyzed human skeletal muscle enzymes. Oxidative enzymatic capacity in paralyzed muscle is well below the level seen in a sedentary able-bodied group of subjects (21,23). It has been shown that early after the onset of training, the gene expression and formation of glycolytic and oxidative enzymes increases, as does the content of glucose transporter (GLUT-4) and the insulin sensitivity of the muscle (24,25). After four and ten weeks of ES training, the concentration of oxidative enzymes can increase by up to 160%, returning to the limits expected in an able-bodied population (7,26). A similar up-regulation of the glycolytic enzyme capacity has also been demonstrated after ten weeks of ES training, although this is been shown to be inconsistent (7,24).

METABOLIC RESPONSES TO ELECTRICAL STIMULATION

Both motor center activity and feedback from working muscle is important for the magnitude of substrate mobilisation of glucose and fatty acids. Involuntary exercise in SCI individuals showed that mobilisation of glucose from the liver was impaired resulting in a gradual drop in plasma glucose during exercise (4), indicating that neural mechanisms are crucial for the matching of glucose mobilisation to peripheral glucose uptake during exer-

cise. During ES exercise in the SCI individual, the energy is primarily glycolysis and high levels of lactate have been found both in muscle and blood, and a relative low fat combustion and fatty acid uptake is seen in contracting muscle of the SCI individual (27).

CARDIOVASCULAR RESPONSES TO ELECTRICAL STIMULATION

Circulation has been studied recently in SCI patients. Interestingly, the rise in cardiac output with increasing exercise workload follows the linear curve of healthy able-bodied individuals (28). Thus, the correlation between oxygen uptake and cardiac output is unaltered in SCI during electrically induced leg exercise. This indicates that even in the absence of intact neural pathways, cardiac function is coupled to metabolic needs via blood born mechanisms. Despite a difference in obtainable maximal heart rate between para- (170 bpm) and tetraplegics (110 bpm), both can reach similar levels of maximal oxygen uptake and cardiac output. Thus, at maximal exercise, paraplegics only have a minor increase in stroke volume and a large heart rate increase, whereas tetraplegics compensate for the limited heart rate reserve by preferentially increasing the stroke volume. Furthermore, it was shown that ES cycling with cuffing both legs did not result in any significant increase in heart rate (29). Interestingly, when the cuffs were released in the resting state, the heart rate rose to the levels seen during exercise without the cuff. The results indicate that either substance in the blood released with exercise (norepinephrine or CGRP) or the blood itself is the signal towards increased heart rate and cardiac output during exercise in this situation. Determination of sympathetic nerve activity

as a norepinephrine spillover from contracting muscle confirms a markedly impaired norepinephrine response in the tetraplegic versus paraplegic individuals. At this stage the most likely explanation for heart rate regulation during exercise in SCI individuals, is the increased venous return that follows with muscular contraction.

CLINICAL BENEFITS OF ELECTRICAL STIMULATION EXERCISE

SPASM

ES techniques, duration, frequency of stimulation and electrode placement, all appear to have different clinical outcomes in regards to the frequency and intensity of spasticity. A significant reduction in the intensity of spasm may last up to 24 hours, with eight hours being the average time of relief, after an acute bout of ES in approximately half the SCI population (30,31). The efficacy of ES has been shown to be directly proportional to the pretreatment clonus that an individual experiences (31).

DEEP VEIN THROMBOSES

During the first three months of injury, deep vein thrombosis (DVT) is clinically prevalent in 16 – 75% of all SCI individuals (32). In combination, anticoagulant medications and pneumatic pressure have been shown to decrease the occurrence of DVT from 40% with pneumatic stockings alone to 25% with the combination (33). While, the application of ES to the lower limbs in combination with low dose heparin reduced the incidence of DVT to 0% (32). In one pilot study an improved fibrinolytic activity was shown up to 100 mins after ES was applied to the calf muscle, accompanied with a mild to moderate improvement in venous blood flow (34).

BONE DEMINERALIZATION

Bone mass loss of up to 50% in the proximal tibia and femoral neck is seen (9). Excretion of markers of increased bone metabolism reaches a peak one to six months after injury (35). Etiology for the increase in bone resorption has been linked to reduced gravitational loading, reduced blood flow and a decrease in the protein matrix that prevents recalcification (36). Data suggests that ES commenced within two weeks of a SCI, may reduce the bone demineralization (37).

The effects of ES training in individuals with a chronic SCI are still unclear. One study showed no improvement in bone mineral density (BMD) of the femoral neck, trochanter or Ward triangle after six months of ES cycling (38) and a second reported a 10% improvement in the proximal tibia after 12 months of ES cycle training (9). A further study found that the increases if observed in BMD are site specific at the insertion site of the bone (39).

DECUBITIS ULCERS

The integumentary system is particularly vulnerable damage following a SCI. Although theoretically preventable, decubitus ulcers develop in 50% of patients with a complete SCI (40). The application of ES may reduce the number of decubitus ulcer that result after a SCI by altering the pressure of the seating interface and by increasing tissue blood flow (41,42). The application of ES may also reduce the healing time once a decubitus ulcer develops (40,43).

CONCLUSION

Exercise for any individual when performed in moderation provides many benefits both physiological and psychological. Exercise for the SCI individual is not only a leisure time activi-

ty but also a way of minimising the secondary medical complications that exist after a disruption to the spinal cord. While upper body training is important for improving wheelchair locomotion particularly in the early phases of rehabilitation, the ability to exercise the lower limbs with ES has provided a means of whole body exercise.

REFERENCES

1. Glaser RM. Functional neuromuscular stimulation. Exercise conditioning of spinal cord injured patients. *Int J Sports Med* 15:142-148, 1994.
2. Raymond J and Crameri RM. Electrical Stimulation-Induced Leg Exercise for Individuals with Spinal Cord Injury. *Am J Med Sports*. 3: 209-222, 2001.
3. Kjær M, Perko G, Secher NH, Boushel R, Beyer N, Pollack SF, Horn A, Fernandes A, Mohr T, Lewis SF, Galbo H. Cardiovascular and ventilatory responses to electrically induced cycling with epidural anesthesia in humans. *Acta Physiol Scand* 151: 199-207, 1994.
4. Kjær M, Pollack SF, Weiss H, Gleim GW, Nicolaisen T, Galbo H, Ragnarsson KT. Regulation of glucose turnover and hormonal responses during electrical cycling in tetraplegic humans. *Am J Physiol*. 271: R191-R199, 1996.
5. Hjeltnes N, Aksnes A-K, Birkeland KI, et al. Improved body composition after 8 wk of electrically stimulated leg cycling in tetraplegic patients. *Am J Physiol*, 273:R1072-R1079, 1997.
6. Crameri RM, Weston, AR, Rutkowski S, Middleton J, Davis GM and Sutton JR. Effects of electrical stimulation leg training during the acute phase of spinal cord injury. *Eur J App Physiol* 83(4 -5), 409-15, 2000.
7. Crameri RM, Weston, AR, Climstein M, Davis GM and Sutton JR. Effects of electrical stimulation-induced leg training on skeletal muscle adaptability in spinal cord injury. *Scand J Med Sci Sports* 12: 1-7, 2002.

8. Hooker SP, Scremin E, Mutton DL, et al. Peak and submaximal physiologic responses following electrical stimulation leg cycle ergometer training. *J Rehabil Res Dev*, 32:361-366, 1995.
9. Mohr T, Andersen JL, Biering-Sørensen F, Galbo H, Wagner A, Kjær M. Long term adaptation to electrically induced cycle training in severe spinal cord injured individuals. *Spinal Cord*, 35:1-16, 1997.
10. Pacy P, Hesp R, Halliday D, Katz D, Cameron G and Reeve J. Muscle and bone in paraplegic patients, and the effect of functional electrical stimulation. *Clin Sci* 75: 481 - 487, 1988.
11. Laskin J, Ashley E, Olenik L, Burnham R, Cummingham D, Steadward R and Wheeler G. Electrical stimulation-assisted rowing exercise in spinal cord injured people. *Paraplegia* 31: 534 - 541, 1993.
12. Sipski ML, Delisa JA and Schweer S. Functional electrical stimulation bicycle ergometry: patient perceptions. *Amer J Phys Med Rehabil*, 68:147-149, 1989.
13. Scelsi R, Marchetti C, Poggi P, Lotta S and Lommi G. Muscle fibre type morphology and distribution in paraplegic patients with traumatic cord lesion. *Acta Neuropathol* 57: 243 - 248, 1982.
14. Greve J., Muszkat R., Schmidt B., Chiovatto J., Barros T. and Batistella L. Functional electrical stimulation (FES): muscle histo-chemical analysis. *Paraplegia* 31: 764 - 770, 1993.
15. Martin T, Stein R, Hoepfner P and Reid D. Influence of electrical stimulation on the morphological and metabolic properties of paralysed muscle. *J Appl Physiol* 72(4): 1401 - 1406, 1992.
16. Neumayer C, Happak W, Kern H and Gruber H. Hypertrophy and transformation of muscle fibres in paraplegic patients. *Artif Organs* 21(3): 188 - 190, 1997.
17. Shields R, Freylaw L, Reiling B, Sass K and Wilwert J. Effects of electrically induced fatigue on the twitch and tetanus of paralysed soleus muscle in humans. *J Appl Physiol* 82(5): 1499 - 1507, 1997.
18. Stein R, Gordon T, Jefferson J, Sharfenberger A, Yang J, Totsy De Zepetnek J and Belanger M. Optimal stimulation of paralysed muscle after human spinal cord injury. *J Appl Physiol* 72(4): 1393 - 1400, 1992.
19. Peckham P, Mortimer J and Marsolais E. Alteration in force and fatigability of skeletal muscle in quadriplegic humans following exercise induced by chronic electrical stimulation. *Clin Orthop* 114: 326 - 334, 1976.
20. Rochester L, Chandler C, Johnson M, Sutton R and Miller S. Influence of electrical stimulation of the tibialis anterior muscle in paraplegic subjects. 1. Contractile properties. *Paraplegia* 33: 437 - 449, 1995.
21. Grimby G, Broberg C, Krotkiewska I and Krotkiewski M. Muscle fibre composition in patients with traumatic cord lesion. *Scand J Rehab Med* 8: 37 - 42, 1976.
22. Andersen JL, Mohr T, Biering-Sørensen F, Galbo H, Kjær M. Myosin heavy chain isoform transformation in single fibres from m. vastus lateralis in spinal cord injured individuals: effects of long-term functional electrical stimulation (FES). *Pflügers Arch* 431: 513-518, 1996.
23. Castro M.J, Apple DF, Staron RS, Campos GE and Dudley GA. Influence of complete spinal cord injury on skeletal muscle within 6 months of injury. *J Appl Physiol* 86(1): 350 - 358, 1999.
24. Hjeltnes N, Galuska D, Bjørnholm M, Zierath J. Exercise-induced overexpression of key regulatory proteins involved in glucose uptake and metabolism in tetraplegic persons: molecular mechanisms for improved glucose homeostasis. *FASEB J* 12: 1701-1712, 1998.
25. Mohr T, Dela F, Galbo H, Biering-Sørensen F, Kjær M. Enhanced whole body insulin action in spinal cord injured subjects after 12 months of electrically induced cycling. *Med.Sci.Sports Exerc.* 33: 1247-1252, 2001
26. Kjær M, Mohr T, Biering-Sørensen F, Bangsbo J. Muscle enzyme adaptation to training and tapering in spinal cord injured individuals. *Eur.J.Appl.Physiol.* 84: 482-486, 2001.
27. Kjær M, Dela F, Biering-Sørensen F, Secher NH, Bangsbo J, Mohr T, Galbo H. Fatty acid

- kinetics and carbohydrate metabolism during electrical exercise in spinal-cord injured humans. *Am.J.Physiol.* 281: R1492-R1498, 2001.
28. Kjær M, Mohr T, Dela F, Secher N, Galbo H, Olesen HL, Biering-Sørensen F, Shifter S. Leg uptake of calcitonin gene related peptide during exercise in spinal cord injured humans. *Clin Physiol.* 21: 32-38, 2001.
29. Kjær M, Pott F, Mohr T, Linkis P, Tornøe P, Secher NH. Heart rate during exercise with leg vascular occlusion in spinal cord injured humans. *J Appl Physiol* 86 806-811, 1999.
30. Robinson CJ, Kett NA and Bolam JM. Spasticity in spinal cord injured patients: 1. Short-term effects of surface electrical stimulation. *Arch Phys Med Rehab* 69(8): 598-604, 1988.
31. Seib TP, Price R, Reyes MR and Lehmann JF. The quantitative measurement of spasticity: effect of cutaneous electrical stimulation. *Arch Phys Med Rehab* 75(7): 746-50, 1994.
32. Merli GJ, Crabbe S, Paluzzi RG and Fritz D. Etiology, incidence, and prevention of deep vein thrombosis in acute spinal cord injury. *Arch Phys Med Rehab* 74(11): 1199-1205, 1993.
33. Green D, Lee MY, Lim AC, Chmiel JS, Vetter M, Pang T, Chen D, Fenton L, Yarkony GM, Meyer PR Jr. Prevention of thromboembolism after spinal cord injury using low-molecular-weight heparin. *Ann Intern Med* 113(8):571-574, 1990.
34. Katz RT, Green D, Sullivan T, Yarkony G. Functional electrical stimulation to enhance systemic fibrinolytic activity in spinal cord injury patients. *Arch.Phys.Med.Rehabil.* 68: 423-426, 1987.
35. Roberts D, Lee W, Cuneo RC, Wittmann J, Ward G, Flatman R, McWhinney B and Hickman PE. Longitudinal study of bone turnover after acute spinal cord injury. *J Clin Endo Metab* 83(2): 415-422, 1998.
36. Mazess RB, Barden HS, Ettinger M. Radial and spinal bone mineral density in a patient population. *Arthritis Rheum.* 31: 891-897, 1988.
37. Hangartner TN, Rodgers MM, Glaser RM and Barre PS. Tibial bone density loss in spinal cord injured patients: effects of FES exercise. *J Rehab Res Dev* 31(1):50-61, 1994.
38. Leeds EM, Klose KJ, Ganz W, Serafini A and Green BA. Bone mineral density after bicycle ergometry training. *Arch Phys Med Rehab.* 71(3):207- 209, 1990.
39. Bloomfield SA, Mysiw WJ, Jackson RD. Bone mass and endocrine adaptations to training in spinal cord injured individuals. *Bone* 19: 61-68, 1996.
40. Griffin JW, Tooms RE, Mendius RA, Clift JK, Vander Zwaag R, el-Zeky F. Efficacy of high voltage pulsed current for healing of pressure ulcers in patients with spinal cord injury. *Phys Ther* 71(6):433- 442, 1991.
41. Levine SP, Kett RL, Cederna PS and Brooks SV. Electric muscle stimulation for pressure sore prevention: tissue shape variation. *Arch Phys Med Rehab* 71(3): 210-5, 1990.
42. Mawson AR, Siddiqui FH and Biundo JJ Jr. Enhancing host resistance to pressure ulcers: a new approach to prevention. *Prevent Med* 22(3): 433-50, 1993.
43. Stefanovska A, Vodovnik L, Benko H and Turk R. Treatment of chronic wounds by means of electric and electromagnetic fields. Part 2. Value of FES parameters for pressure sore treatment. *Med Biol Eng Comp.* 31(3):213-20, 1993.

MULTIDISCIPLINARY ELECTRONIC PATIENT RECORD AND CLINICAL DATA BASE IN ONE FOR SPINAL CORD LESIONED INDIVIDUALS (SCIBase).

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SUMMARY

A multidisciplinary patient-record and database in one (SCIBase) has been created. The aims are to strengthening the multidisciplinary teamwork, to improve the quality of the daily documentation, to allow the record to be read on computers several places at the same time, and to make quality assurance easier.

SCIBase is used by doctors, nurses, physiotherapists, occupational therapists, social workers, psychologists, and secretaries, and is approved by the Danish Data Protection Agency.

The programming is performed in PowerBuilder, and data are stored in a Sybase database. The system is functioning under Windows, and the printing is in Microsoft Word. The structure is flexible to allow changes to be made when the need arise with the development. Data are online transferred from the patient administrative system.

The SCIBase structure is based on checklists for each professional group delivering data to the system. Except from the nurses record is all written clinical documentation electronically available.

Data form the database can in anonymous form be transferred to Microsoft Access, for further analyses or data-export.

The Clinic for Para- and Tetraplegia introduced 1999 a multidisciplinary electronic patient record (ERP), which on the same time was a clinical database, i.e. the data are only typed into the system once, and can thereafter be reused in other connections. The system is named SCIBase (SCI = Spinal Cord Injury).

The use of personal computers (PC) is now the routine among all professional groups taking part in the clinical management of the spinal cord lesioned (SCL) individuals, i.e. they all provide their respective inputs to SCIBase.

AIMS

The major aims for introducing SCIBase was:

1. To strengthening the multidisciplinary teamwork at the Clinic.
2. To ensure high quality of information in the patient records of the SCL individuals treated, rehabilitated and followed-up in the Clinic. This includes the use of checklists for basic information by all the professional groups, doctors, nurses, physiotherapists, occupational therapists, social workers, and psychologists. In this way it also secured that newly employed will have the information included. On the same time data need only to be written once.
3. To make it possible for all personnel

taking part in the treatment, rehabilitation and follow-up of the SCL individuals to review the information in the record from all clinical professionals at the PC. This means the paper record is not necessary, and many individuals may simultaneously review the same record.

4. To make quality control easier. This includes using the outcome measures, which are part of the clinical database.

BACKGROUND

The Clinic has an uptake area corresponding to East-Denmark, Greenland and the Faroe Island. This covers a population of approximately 2.5 million. The Clinic receives traumatic as well as non-traumatic SCL patients. Every year is approximately 60 patients admitted with new lesions – approximately half are non-traumatic. Furthermore the Clinic has over 200 admissions related to complication treatment and specific rehabilitation tasks. At the outpatient clinic are 300 SCL individuals seen yearly. Geographically the admissions to the Clinic are in Hornbæk, 50 km north of Copenhagen, and the outpatients are received in the hospital in Copenhagen. The doctors are the same.

It is the hope in the future that SCI-Base shall cover all Denmark, i.e. West-Denmark as well, at the Para-plegia-function, Viborg hospital.

RECORD INFORMATION

SCI-Base is built up on the basis of structured profession specific checklists. These checklists were developed to make sure that the most important information related to the SCL individuals is collected. The clinical personnel are responsible for collection of their respective information to the record.

Doctors fill in checklists at *admission*,

during admission, at *discharge*, and at any later *follow-up* control.

The admission checklist is filled in when the SCL patient is admitted to the Clinic for his or her primary rehabilitation after the SCL. This checklist includes information as any other patient record, i.e. information about previous diseases and symptoms, including possible operations and hospitalisations, known allergy, tobacco and alcohol habit. Then it includes information related to the SCL: date and type of injury, possible associated injuries, treatments performed, respiratory and urological status, etc.

The *discharge* checklist is filled in when the SCL person is going to be discharged from the Clinic after the primary rehabilitation period. It includes information of possible diseases and complications during the rehabilitation period, more detailed information about the sexual function, spasticity, pain, etc. and its treatment, urological investigations performed etc.

The *follow-up* checklist is filled in every time the SCL individual come for control in the Clinic, or when relevant during the admission. It includes information about symptoms, complications, and diseases, and possible hospital admissions and treatments since the last contact to the Clinic. Furthermore detailed information about the bladder and bowel emptying, the sexual function, spasticity, pain, etc. and its treatment, urological investigations performed etc. In addition it includes basic data on activities of daily living (ADL), social and psychological situation, etc.

Nurses fill in a checklist at *discharge from the primary rehabilitation*. This includes detailed data about the bladder and bowel emptying method at discharge. Furthermore information

about the skin condition and pressure ulcers at admittance, developed *during* the stay in the Clinic, and at discharge. In addition they during admission fill in information of height and weight, for the calculation of body mass index (BMI), residual urine, and blood pressure.

Physiotherapists fill in checklists *during admission as well as at discharge* covering muscle strength, all kinds of skills related to transfers, mobility, and various ADL.

Occupational therapists fill in checklist *during admission* as well as at discharge including muscle strength of the upper extremity, a range of ADL, together with detailed information about tetraplegic individuals hand function.

Social workers fill in a checklist at *discharge* with data related to pre-injury civil status, housing conditions, educational level, work situation, and information about post-injury compensation possibilities, applications submitted regarding new housing, education, pension, etc. Furthermore a follow-up checklist to keep record on which of the applications have been successful is used.

Psychologists fill in a checklist at *discharge* with data about psychological crises pre-injury, the use of psychological or psychiatric assistance during acute and the rehabilitation phase post-injury. In addition information about their coping is included.

In addition to the above mentioned checklists are the following classifications made available for use at various time points, both during the acute and primary rehabilitation phase, and at later control visits to the Clinic:

ASIA/IMSOP (*American Spinal Injury Association/International Medical Society of Paraplegia*) international neurological classification for spinal cord

injury (1). The physician fills in this classification, at least twice, around admission and discharge for primary rehabilitation.

FIM (Functional Independence Measure) (2) is filled in at least 3 times: at admission, midway during rehabilitation, and at discharge from the primary rehabilitation. The treating nurse, physiotherapist and occupational therapist in co-operation perform the scoring.

Physicians as well as physiotherapists use Modified Ashworth Scale for spasticity when relevant.

Much of the information in the checklists is primarily entered by ticking boxes and similar. After finishing entering data the information is transferred to the record. For the doctors and nurses record a transformation to normal text is performed. The data in the other checklists are printed out more directly as they have been entered on the PC.

Continuations for free text is available for day-to-day information, which is to be communicated in the record. These are not made as checklists, and the data therefore cannot be retrieved as easy as the previously described information.

The doctor's record is made as a common record, in the sense that free text, i.e. conclusions or other important extracts, from the physiotherapist's, occupational therapist's, social worker's, and psychologist's records can be copied from their own record into the common record, to make it easier to have a total overview for the particular patient. On the same time it is always possible to have the more detailed information by looking up the particular professions' record.

Finally, several other schemes are available in SCIBase, including many research data sheets. This includes a data sheet

for everyone in the SCIBase who have died. Data related to the death may be retained in SCIBase for 75 years.

Updated review including previous diseases, admissions and urological investigations can always be generated in SCIBase.

Discharge letter from the primary rehabilitation is made semi-automatically on the basis of the information collected in the mentioned checklists and classifications. Multidisciplinary information in the discharge letters is secured.

In addition, all schemes etc. for each single SCL individual can be retrieved as they are arranged according to the date they were entered.

MANUALS

Manuals are developed for each professional group for the filling in of the various data sheets. In these are described how the fields should be filled in, and in particular information are given regarding how the various answer possibilities shall be interpreted. This because many of the collected data are stored according to international classifications (1-6), and guidelines used in other similar databases for SCL individuals (7).

SECURITY

All information in the database is secured with an unique Central Person Register Number for each patient.

Every access to the system has to be performed with a password, and each person is approved for specific writing and reading rights according to their function in the Clinic. The system entrances are logged by name of the person entering the database and what he/she has been doing.

SCIBase is approved by the Danish Data Protection Agency.

TECHNICAL SOLUTION

The programming of SCIBase database is made in PowerBuilder, and the data are stored in a Sybase database. The system is running under Windows. The printout to the record is made in Microsoft Word.

Anonymous analyses of the data and data export can be carried out in Microsoft Access.

The structure is flexible to allow changes to be made when the need arise with the development, e.g. new investigations or treatments.

When a new patient is entered with his or her Central Person Register number demographic data and information on the general practitioner etc. are online transferred from the patient administrative database system. Then Central Person Register number and name of the patient will automatically occur on every printout from SCIBase. This minimise the risk for misplacing paper record pages.

The doctors', nurses', physiotherapists', occupational therapists', social workers' and psychologists' records can be read directly on any PC in the inpatient facility and the outpatient clinic.

RESULTS

In the daily treatment, rehabilitation and follow-up the SCIBase facilitate the multidisciplinary work around each SCL individual. This not least because the various personnel categories get more daily insight to the others work.

Some of the experiences gained during the first year's use of SCIBase in the multidisciplinary team are summarized in the table. In addition, it was reported that the use of common classifications in different professional groups and international standards

ADVANTAGES	DISADVANTAGES
<p>The record is always available to all treating personnel Easy to use</p>	<p>Too few computers, creating waiting time Time spend at the PC is increase The system goes down</p>
<p>Take in demographic data directly from the Administrative System The records look very alike The checklists secure that the most important information are documented More details are documented Gives faster overview – better structure The logging gives security</p> <p>The discharge letter become very thorough and is multidisciplinary Summaries and draft for discharge letters can be generated Greater consciousness about what is documented Higher level of information</p>	<p>Some data cannot be transferred from the Administrative System The records look very alike Dependence of the checklists can be inhibiting Much text is generated The overview may disappear The logging makes it more difficult to change mistakes The discharge letter become very long</p> <p>More paper is used</p>

gave an extra insight and awareness. Further it was pointed out that SCIBase implies a possibility for creation of better quality indicators and evaluation methods. With this purpose SCIBase has already delivered data to and international project (8). SCIBase in general increase the possibility for research. Finally the present SCIBase is found to be an appetizer for further development.

DISCUSSION

Avoidance of redundant entering and reuse of data are possible due to the stringent structure of SCIBase, in comparison to many EPRs, which primarily function as word processor patient charts (9). Because of the structure it is also possible to validate many of the data in SCIBase, e.g. only specific answer possibilities are available, and fields with numbers can be defined. In our experience it is decisive that

each user/discipline find their respective checklists meaningful. It is not the ambition at the present time to abandon the paper record, not least because many information are available in paper only. Further, not yet it was found meaningful to use resources for scanning and indexing of the additional information. In the future we wish all information to be available in a multimedia EPR (10).

REFERENCES

1. American Spinal Injury Association/ and International Medical Society of Paraplegia ASIA/IMSOP. International Standards for Neurological Classification of Spinal Cord Injury. American Spinal Injury Association, Chicago, Illinois, revised 2000.
2. Keith RA, Granger CV, Hamilton BB, Sherwin FS. The functional independence measure: A new tool for rehabilitation. In Eisenberg MG, Grzesiakrc (eds.): Advances in Clinical Rehabilitation. New York, Springer Publishing Co., 1987, vol.1, pp.6-18.

3. Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth scale of muscle spasticity. *Phys Ther* 1987;67:206-7.
4. Penn RD, Savoy SM, Corcos D, Latash M, Gottlieb G, Parke B, Kroin JS. Intrathecal baclofen for severe spinal spasticity. *N Engl J Med* 1989;320:1517-21.
5. Daniel RK, Hall EJ, MacLeod MK. Pressure sores – a reappraisal. *Ann Plast Surg* 1979;3:53-63.
6. Sollerman C, Ejeskär A. Sollerman hand function test. *Scand J Plast Reconstr Surg* 1995;29:167-76.
7. Levi R, Ertzgaard P. Quality indicators in spinal cord injury care: a Swedish collaborative project. The Swedish Spinal Cord Injury Council 1998. *Scand J Rehabil Med Suppl.* 1998;38:1-80.
8. Haigh R, Tennant A, Biering-Sørensen F, Grimby G, Marinček C, Phillips S, Ring H, Tesio L, Thonnard J-L. The use of outcome measures in physical medicine and rehabilitation within Europe. *J Rehabil Med* 2001;33:273-8.
9. Sujansky WV. The benefits and challenges of an electronic medical record: Much more than a "word-processor" patient chart. *Western J Med* 1998;169:176-83.
10. Lowe HJ. Multimedia electronic medical record systems. *Academic Med* 1999;74:146-52.

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PhD defended at The Danish Technical University 22.02.1997.

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PhD defended at The Danish Technical University 03.02.1998.

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PhD student 1999-.

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PUBLICATIONS ON SPINAL CORD LESIONS FROM THE CLINIC

1969

Eskesen B. Follow-up on a group of patients with para- and quadriplegia. *Acta Orthop Scand* 1969;40:667-8.

1970

Eskesen BE. Spinal paraplegi. En efterundersøgelse af medicinsk og social revalidering. *Ugeskr Læger* 1970; 132: 849-54.

Glahn BE. Manual provocation of mictrurition contraction in neurogenic bladders. *Scand J Urol Nephrol* 1970; 4: 25-36.

1972

Ranløv P, Rossing N, Schwartz M, Eskesen B, Rasmussen G. Late hypoalbuminaemia and albumin metabolism in paraplegic and tetraplegic patients. *Clin Sci* 1972;43:639-44.

1974

Glahn BE. Neurogenic bladder in spinal cord injury. Management of patients in Hornbaek, Denmark. *Urologic clinics of North America* 1974;1: 163-73.

1977

Eskesen B. Siddesår helet eller udngået med gelépude. *Sygeplejersken* 1977;77:16-7,22.

1978

Eskesen B. Hvorledes skal behandling af para- og tetraplegiker tilrettelægges. *Ugeskr Læger* 1978;140:747-8

Gregersen H, Hansen B, Schmidt L, Møller K. Om tetraplegikernes gribefunktion. *Ergoterapeuten* 1978;Nr. 8:275-80.

1982

Knudsen L, Sølvhøj L, Christensen B. The use of a haemodialysate in the treatment of decubital ulcer: A doubleblind randomized clinical study. *Curr Ther Res.* 1982; 32: 498-504.

Knudsen L, Lundberg D, Ericsson G. Myositis ossificans circumscripta in para-/tetraplegics. *Scand J Rheumatol* 1982; 11: 27-31.

Skagen K, Jensen K, Henriksen O, Knudsen L. Sympathetic reflex control of subcutaneous blood flow in tetraplegic man during postural changes. *Clin Sci* 1982; 62: 243-5.

1983

Biering-Sørensen F. International Medical Society of Paraplegia. Stoke Mandeville, 18. - 20.V.1983. *Ugeskr Læger* 1983;145:2665-6.

Biering-Sørensen F. The classification system of ISOD (International sports Organisation for the Disabled): In: Hoerberigs JH, Vorsteveld H (eds.). *Proceedings of the workshop on disabled and sports* 1983; pp.97-105.

Biering-Sørensen F. Problems of the ISOD classification system. In: Hoerberigs JH, Vorsteveld H (eds.). *Proceedings for the workshop on disabled and sports* 1983; pp. 106-10.

1984

Biering-Sørensen F, Munk L, Reerslev P. Sexualforhold. In: Riishave H, Hansen CE, Tanghøj P (eds.). *Håndbog om para- og tetraplegi*. 1984. Paraplegikerkredsen, København pp.194-228.

Biering-Sørensen F. First Euro-Mediterranean meeting on paraplegia. Hyeres, 13. - 14.I.1984. *Ugeskr Læger* 1984;146:1147-8.

Biering-Sørensen F. International Medical Society of Paraplegia. Denver, 20. - 22.VI.1984. *Ugeskr Læger* 1984;146:2638-9.

1985

Sonne M, Friis J. Myositis ossificans localisata, behandlet med difosfonat. *Ugeskr Læger* 1985; 147: 4007-8.

Pedersen W. Spinale læsioner som følge af sport- og fritidsulykker 1965-84. *Ugeskr Læger* 1985; 147: 4236-8.

1986

Biering-Sørensen F. International Medical Society of Paraplegia. Edinburgh 2.-5.IX.1985. *Ugeskr Læger* 1986;148:262.

Biering-Sørensen F, Mosdal C, Mortensen A. Spinal cord injuries: Management and recent developments. Stoke Mandeville 8. - 18.IX.1985. Ugeskr Læger 1986;148:263.

Andersen EB, Boesen F, Henriksen O, Sonne M. Blood flow in skeletal muscle of tetraplegic man during postural changes. Clin Sci 1986; 70: 321-5.

1987

Pedersen W, Clausen S, Kriegbaum NJ. Spinal lesions in patients with ankylosing spondylitis. Scand J Rheumatol 1987; 16: 381-2.

Pedersen V, Biering-Sørensen F. International medical society of paraplegia. Annual scientific meeting, Stoke Mandeville, England. 13. - 15.V.1987. Ugeskr Læger 1987;149:1821-2.

Schrøder I, Thiele V. "Rejen" - et godt hjælpemiddel, hvis forflytninger er svære. Ergoterapeuten 1987; Nr. 21:944.

Hagenbøl L, Biering-Sørensen F. Sexual aspects of spinal cord injury. Stoke Mandeville, 16.V.1987. Ugeskr Læger 1987;149:1958-9.

Biering-Sørensen F. Handicapidræt. Månedsskr Prakt Lægegern 1987;okt:795-706.

1988

Pedersen W, Clausen S, Biering-Sørensen F. Prognosen ved traumatisk cervikal rygmarvslæsion. Ugeskr Læger 1988;150:1162-6.

Pedersen W, Müller PG, Biering-Sørensen F. Traumatiske rygmarvslæsioner opstået i Grønland 1965-86. Ugeskr Læger 1988;150:1220-2.

Biering-Sørensen F, Bohr H, Schaadt O. Bone mineral content of the lumbar spine and lower extremities years after spinal cord lesion. Paraplegia 1988;26:293-301.

Biering-Sørensen F, Holst-Nielsen F. Tetraplegi-håndkirurgi. Hvad hjælper håndkirurgi tetraplegikere? Paraplegi 1988;10:16-8.

Biering-Sørensen F, Friis J. Fysiurgisk hospital, Hornbæk er blevet moderniseret. Ugeskr Læger 1988;150:2474.

Biering-Sørensen F. Book review of Illis LS (ed).

Spinal cord dysfunction: assessment. Ugeskr Læger 1988;150:2763.

Biering-Sørensen F. Klasseinddeling af idrætssudøvere ved OL i Seoul. Handicap idræt 1988;12:37-9.

1989

Müller PG. Actinomycosis as a cause of spinal cord compression: A case report and review. Paraplegia 1989; 27: 390-3.

Mosdal C, Biering-Sørensen F. Organisation og funktion af et paraplegiafsnit. Ugeskr Læger 1989;12:770-3.

Pedersen V, Müller PG, Biering-Sørensen F. Traumatic spinal cord injuries in Greenland 1965 - 86. Paraplegia 1989;27:345-9.

Biering-Sørensen F. Handicapidræt. In: Krohn L, Bugge PM, Kristensen JH, Vinterberg H (eds.). Idrætsreumatologi. Symposium 1989, pp. 88-103.

Biering-Sørensen F, Haraldsen T, Kissmeyer T & Paraplegikerkredsen. Patienten er rygmarvsskadet - en oplysningspjece for praktiserende læger og hjemmesygeplejersker (Information pamphlet for general practitioners and home nurses). Paraplegikerkredsen 1989. Revised edition 1994.

1990

Pedersen SS, Hørbov S, Biering-Sørensen F, Høiby N. Peroral treatment with ciprofloxacin of patients with spinal cord lesion and bacteriuria caused by multiply resistant bacteria. Paraplegia 1990;28:41-7.

Biering-Sørensen F, Jacobsen E, Hjelms E, Fodstad H, Trojborg W. Diafragma pacing ved elektrisk stimulation af nn. phrenici. Ugeskr Læger 1990;152:1143-5.

Biering-Sørensen F, Ryde H, Bojsen-Møller F, Lyquist E. Shock absorbing material on the shoes of long leg braces for paraplegic walking. Prosthetics and Orthotics International 1990; 14:27-32.

Biering-Sørensen F, Børgesen SE, Tøndevold E. Akut operativ behandling af columnafraktur

med medullær påvirkning. Ugeskr Læger 1990;152:1143-5.

Falktoft C, Biering-Sørensen F. Vandrutsjebane og traumatisk tetraplegi. Ugeskr Læger 1990;152:1910-1.

Biering-Sørensen F, Bohr HH, Schaadt OP. Longitudinal study of bone mineral content in the lumbar spine, the forearm and the lower extremities after spinal cord injury. Eur J Clin Invest 1990;20:330-5.

Sønksen J, Hansen EF, Biering-Sørensen F, Colstrup H. Intrakavernøs selvinjektion til behandling af erektil dysfunktion hos rygmarsvskadede. Ugeskr Læger 1990;152:3006-9.

Biering-Sørensen F, Pedersen V, Clausen S. Epidemiology of spinal cord lesions in Denmark. Paraplegia 1990;28:105-18.

Biering-Sørensen F, Halkier KI. Denmark. In: Williams T (ed.). Research and perspectives on Adapted Physical Activity in Europe. European Association for Research into Adapted Physical Activity 1990, pp. 9-11.

Biering-Sørensen F. Spinal cord injury: Neurological & functional recovery. Philadelphia, USA, 18. - 19.1.90. Ugeskr Læger 1990;152:929-30.

Biering-Sørensen F. Neural transplantation to spinal cord injuries. Århus, 30.8-2.9.1990. Ugeskr Læger 1990;152:3716-7.

Biering-Sørensen F. Mindstehandicap. Handicapidræt 1990;14:33 & 35.

1991

Christensen FL, Jensen TS. Sanseforstyrrelser og smerter ved rygmarsvslæsion. Ugeskr Læger 1991;153:3466-9.

Biering-Sørensen F, Knudsen JL, Schmidt A, Bundgaard A, Christensen I. Effect of respiratory training with a mouth-nose-mask in tetraplegics. Paraplegia 1991;29:113-9.

Dalsgaard B. Krydderi på samlivet. Artikel om seksualitet og hjælpemidler for rygmarsvskadede. Ergoterapeuten 1991; Nr. 9: 4-7.

Langhoff-Roos J, Biering-Sørensen, Bock JE. Graviditet og fødsel hos rygmarsvskadede. Ugeskr Læger 1991;153:1286-7.

Sønksen JOR, Drewes AM, Biering-Sørensen F, Giwerzman AJ. Vibrationsfremkaldt refleksejakulation hos rygmarsvskadede. Ugeskr Læger 1991;153:2888-90.

Haxthausen E-U, Biering-Sørensen F, Dahl K, Hansen SD, Andersen OT. Restoration of key grip in SCI patients. An attempt using FES controlled by EMG from the stimulated muscle. In: Proceeding of the topical workshop: New theoretical and applied approaches in the restoration of impaired motor control. Thomas Sinkjær ed. 1991, pp.71-81.

Biering-Sørensen F, Mosdal C. Den akutte behandling. In: Kommer vi til at gå....? - en bog om rygmarsvskader og mulighederne for at reparere. 1991 pp.35-50.

Biering-Sørensen F, Drewes A. Statistiske oplysninger. In: Kommer vi til at gå....? - en bog om rygmarsvskader og mulighederne for at reparere. 1991 pp.89-99.

Sønksen JOR, Biering-Sørensen F. The first world congress on electroejaculation. Pasadena, Californien, 4.-6.2.1991. Ugeskr Læger 1991;153:1733-4.

Biering-Sørensen F. 30th annual scientific meeting of the international medical society of paraplegia. Stoke Mandeville, England, 15.-17.5.1991. Ugeskr Læger 1991;153:2241.

Biering-Sørensen F. Elektrisk stimulering i behandlingen og rehabiliteringen af rygmarsvskadede personer. viaDATCH 1991, Nr.5:6-8.

1992

Sindrup JH, Wroblewski H, Kastrup J, Biering-Sørensen F. Nocturnal variations in lower leg subcutaneous blood flow in paraplegic men. Clinical Science 1992;82:47-54.

Biering-Sørensen F, Pedersen W, Müller PG. Spinal cord injury due to suicidal attempts. Paraplegia 1992;30:139-44.

Andersen LS, Biering-Sørensen F, Müller PG, Jensen IL, Aggerbeck B. Dextropropoxyphene

hydrochloride for treatment of hyperhidrosis in spinal cord injured, and prevalence of hyperhidrosis. *Paraplegia* 1992;30:184-91.

Winther K, Gleeurup G, Snorrason K, Biering-Sørensen F. Platelet function and fibrinolytic activity in cervical spinal cord injured patients. *Thrombosis Research* 1992;65:469-74.

Biering-Sørensen, Sønksen J. Penile erection in men with spinal cord or cauda equina lesions. *Seminars in Neurology* 1992;12:98-105.

Sønksen J, Biering-Sørensen F. Fertility in men with spinal cord or cauda equina lesions. *Seminars in Neurology* 1992;12:-106-14.

Sønksen J, Biering-Sørensen F. Transcutaneous nitroglycerin in the treatment of erectile dysfunction in spinal cord injured. *Paraplegia* 1992;30:554-7.

Biering-Sørensen M, Biering-Sørensen F. Tracheostomy in spinal cord injured. Frequency and follow-up. *Paraplegia* 1992;30:656-60.

Haxthausen E-U, Biering-Sørensen F, Dahl K, Hansen SD, Andersen OT. Restoration of key grip in SCI patients. An attempt using FES controlled by EMG from the stimulated muscle. In: Restoration of walking for paraplegics. Recent advancements and trends. Pedotti A & Ferrarin M eds. Commission of the European Communities COMAC BME Concerted Action "Mobility restoration for paralyzed persons" IOS Press 1992, pp.291-9.

1993

Sønksen JOR, Biering-Sørensen F, Kristensen JK, Seager SWJ, Giwerzman AJ. Elektrostimulation til frembringelse af ejakulation hos rygmarvsskadede mænd. *Ugeskr Læger* 1993;155:176-9.

Ellitsgaard N, Hauge EN, Biering-Sørensen F. Udvikling af karcinom i kroniske tryksår hos rygmarvsskadede patienter. *Ugeskr Læger* 1993;155:1473-4.

Hamamci N, Hawran S, Biering-Sørensen F. Achondroplasia and spinal cord lesion. *Paraplegia* 1993;31:375-9.

Biering-Sørensen F, Tøndevold E. Indomethacin

and disodium etidronate for the prevention of recurrence of heterotopic ossification after surgical resection. Two case reports. *Paraplegia* 1993;31:513-5.

Hamamci N, Biering-Sørensen F. Acute management of spinal cord injured patients. *Yeni Tip Dergisi (New J of Medicine)* 1993;10(2):-38-45.

Biering-Sørensen F, Pedersen W, Müller PG. Rygmarvsskade forårsaget af selvmordsforsøg. *Ugeskr Læger* 1993;155:2881-2884.

Drewes AM, Biering-Sørensen F, Andreasen A. "Mini 12-metre" boats. A sport and recreational activity for persons with spinal cord injuries. *Palaestra* 1993;9:36-9.

Svendsen L, Drewes AM, Biering-Sørensen F, Lønnberg F. Kroniske smerter efter rygmarvslæsion - en deskriptiv undersøgelse. *Ugeskr Læger* 1993;155:3118-22.

Nielsen J, Petersen N, Ballegaard M, Biering-Sørensen F, Kiehn O. H-reflexes are less depressed following muscle stretch in spastic spinal cord injured patients than in healthy subjects. *Experimental Brain Research* 1993;97:173-6.

Hamamci N, Biering-Sørensen F. Sexual function and fertility in spinal cord injured men. *Yeni Tip Dergisi* 1993;10(6):44-50.

Biering-Sørensen F. & Hansen C. Idrætsskader. Rapport fra VISTA'93. *Handicapidræt Nr.5:31*, September 1993.

Biering-Sørensen F. & Hansen C. Integration. Rapport fra VISTA'93. *Handicapidræt Nr.6:23*, October 1993.

Biering-Sørensen F. & Hansen C. Doping. Rapport fra VISTA'93. *Handicapidræt Nr.6:25*, October 1993.

Biering-Sørensen F. & Hansen C. Klassificering. Rapport fra VISTA'93. *Handicapidræt Nr.7:25*, november 1993.

1994

Biering-Sørensen F, Højby N, Nordenbo A, Ravnborg M, Bruun B, Rahm V. Ciprofloxacin as

prophylaxis for urinary tract infection. Prospective, randomized, cross-over, placebo controlled study in patients with spinal cord lesion. *J Urol* 1994;151:105-8.[Erratum *J Urol* 1994;151:1032].

Sønksen J, Biering-Sørensen F. Semen quality before and after spinal cord injury. *Paraplegia* 1994;32:117-9.

Sørensen JL, Hauge EN, Wroblewski H, Biering-Sørensen F. Cutaneous and subcutaneous blood flow rates in paraplegic man investigated by ¹³³Xenon washout. Methodological considerations. *Clinical Physiol* 1994;14:281-9.

Sønksen J, Biering-Sørensen F, Kristensen JK. Ejaculation induced by penile vibratory stimulation in men with spinal cord injuries. The importance of the vibratory amplitude. *Paraplegia* 1994;32:651-60.

Biering-Sørensen F, Biering-Sørensen M, Hilden J. Reproducibility of Nordic sleep questionnaire. *Paraplegia* 1994;32:780-6.

Biering-Sørensen F. Minimal disability in sport competitions for disabled. In: *VISTA '93 - The Outlook*. Eds. Steadward, R.D, Nelson, E.R. and Wheeler, G.D. Publisher: Rick Hansen Centre, Edmonton, Alberta, Canada, 1994, pp.378-390.

Biering-Sørensen F. EMG signals from paretic muscles controlling electrical stimulation of the same muscle. Project 1083 - EPCES. In: *Technology initiative for disabled and elderly people. TIDE. Bridge phase - synopses*. European Commission, Directorate General XIII. December 1994, pp.70-71.

Paraplegikerkredsen, m.fl.: Biering-Sørensen F, Bock JE. Patienten er rygmærskadet - en oplysningspjece om graviditet og fødsel. (Information brochure on pregnancy and delivery) Published by Paraplegiker-kredsen October 1994.

1995

Biering-Sørensen M, Norup PW, Jacobsen E, Biering-Sørensen F. Treatment of sleepapnoe in spinal cord injured. *Paraplegia* 1995;33:271-3.

Ødum L, Sønksen J, Biering-Sørensen F. Seminal somatostatin in men with spinal cord injury. *Paraplegia* 1995;33:374-6.

Biering-Sørensen F, Hartkopp A. Trauma and Rehabilitation. *Spinal Cord Lesions. Current Opinion in Neurology* 1995;8:451-5.

Mohr T, Tornøe P, Biering-Sørensen F, Secher NH, Kjær M. Measurement of heart rate during exercise induced by cyclic functional electrical stimulation in spinal cord injured individuals. Methodological problems. In: *Proceedings from 5th Vienna Inter-national workshop on Functional Electrostimulation. Basics, Technology, Clinical application*. Vienna, Austria, August 17-19, 1995. pp.91-94.

Sennels S, Thorsen R, Biering-Sørensen F, Hansen SD, Andersen OT. EMG-controlled wrist extension. In: *Proceedings from 5th Vienna International workshop on Functional Electrostimulation. Basics, Technology, Clinical application*. Vienna, Austria, August 17-19, 1995. pp.417-420.

1996

Andersen JL, Mohr T, Biering-Sørensen F, Galbo H, Kjær M. Myosin heavy chain isoform transformation in single fibres from m.vastus lateralis in spinal cord injured individuals: Effects of long-term functional electrical stimulation (FES). *Pflügers Arch - European Journal of Physiology* 1996;431:513-8.

Sønksen J, Ohl DA, Giwerzman A, Biering-Sørensen F, Kristensen JK. Quality of semen obtained by penile vibratory stimulation in men with spinal cord injuries: Observations and predictors. *Urology* 1996;48:453-7.

Hawran S, Biering-Sørensen F. Paraplegics use of long leg calipers. A follow-up study of patients discharged 1973-82. *Spinal Cord* 1996;34:666-8.

Nielsen KT, Agersten J, Biering-Sørensen F. Electronic Patient Record SCIRH. In: *Medical Informatics Europe '96. Human Facets in Information Technologies*. Eds. J Brender, JP Christensen, Scherrer J-R, P McNair. IOS Press 1996, pp.443-447.

Biering-Sørensen F. Rehabilitation of para- and tetraplegia. In: *Proceedings from European Association of Neurosurgical Societies Course in Rebuild-Aalborg, Denmark, September 8th-13th, 1996*. (pp.1-8).

Thorsen R, Biering-Sørensen F, Hansen SD, Andersen OT. Myoelectric signals from paretic wrist extensor controlling electrical stimulation of the same muscle. In: A.Pedotti, M.Ferrarin, J.Quintern, R.Reiner (eds.): *Neuroprosthetics - from basic research to clinical application*. Springer Verlag, Berlin, Heidelberg 1996, pp.373-376.

Mohr T, Tornøe P, Biering-Sørensen F, Pødenphant J, Andersen J, Wagner A, Galbo H, Kjær M. Long term electrically induced cycling in SCI - A pilot study. In: A.Pedotti, M.Ferrarin, J.Quintern, R.Reiner (eds.): *Neuroprosthetics - from basic research to clinical application*. Springer Verlag, Berlin, Heidelberg 1996, pp. 569-577.

Hartkopp A, Harridge SDR, Mizuno M, Ratkevicius A, Gregersen H, Hagen E, Kjær M, Quistorff B, Biering-Sørensen F. Preliminary results of muscle strength, endurance and metabolism in the wrist extensors of tetraplegics following 12 weeks of electrically induced training. In: A.Pedotti, M.Ferrarin, J.Quintern, R.Reiner (eds.): *Neuroprosthetics - from basic research to clinical application*. Springer Verlag, Berlin, Heidelberg 1996, pp. 579-590.

Sennels S, Biering-Sørensen F, Hansen SD, Andersen OT. Adaptive filters for muscle response suppression. In: *Proceedings from IEEE engineering in Medicine & Biology, Amsterdam 1996*, on CD-ROM.

Biering-Sørensen F. Bodil Eskesen, 8.9.1912-31.8.1996. *Ugeskr Læg* 1996;158:7136.

1997

Mohr T, Andersen JL, Biering-Sørensen F, Galbo H, Bangsbo J, Wagner A, Kjær M. Long term adaptation to electrical induced cycle training in spinal cord injured individuals. *Spinal Cord* 1997;35:1-16.

Hartkopp A, Brønnum-Hansen H, Seidenschmur AM, Biering-Sørensen F. Survival and cause of death after traumatic spinal cord injury. A longterm epidemiologic survey from Denmark. *Spinal Cord* 1997;35: 76-85. [Corrigendum. *Spinal Cord* 1997;35:862-4].

Karlsson A-K, Elam M, Friberg P, Biering-Sørensen F, Sullivan L, Lönnroth P. Regulation

of lipolysis by the sympathetic nervous system: A microdialysis study in normal and spinalcord injured subjects. *Metabolism* 1997;46:388-94.

Sennels S, Biering-Sørensen F, Andersen OT, Hansen SD. Functional neuromuscular stimulation controlled by surface electromyographic signals produced by volitional activation of the same muscle: Adaptive removal of the muscle response from the recorded EMG-signal. *IEEE Transactions on Rehabilitation Engineering* 1997;5(2):195-206.

Mohr T, Pødenphant J, Biering-Sørensen F, Galbo H, Thamsborg G, Kjær M. Increased bone mineral density after prolonged electrically induced cycle training of paralyzed limbs in spinal cord injured man. *Calcified Tissue International* 1997;61:22-5.

Biering-Sørensen F, Sørensen JL, Barros TEP, Monteiro AA, Nuisebeh I, Shenaq SM, Shibasaki K. Clinical case of the month. Pressure ulcer treatment. *Spinal Cord* 1997;35:641-6.

Pedersen IS, Jensen BS. Hornbæk anvender FIM testen. *Ergoterapeuten* 1997;Nr.9:22-3

Sønksen J, Sommer P, Biering-Sørensen F, Ziebe S, Linhard A, Loft A, Andersen AN, Kristensen JK. Pregnancies after assisted ejaculation procedures in men with spinal cord injury. *Arch Phys Med Rehabil* 1997;78:1059-61.

Granat MH, Maxwell DJ, Hermens HJ, Baardman G, Heller BW, Biering-Sørensen F, Juan FJ, Veltink PH. Clinical rehabilitation using electrical stimulation via telematics (CREST). In: D.Popovic (ed.). *Proceedings from Second annual conference: International Functional Electrical Stimulation Society, and Fifth triennial conference: Neural prosthesis: Motor systems V*. Simon Fraser University, Burnaby, British Columbia, Canada, 16-21 August, 1997, pp.263-264.

Biering-Sørensen F, Sønksen J. Potens og fertilitet hos rygmarvsskadede mænd. In: C Graugaard, P Hertoft, B Møhl (eds.). *Hjerne og seksualitet. Aspekter af teori & klinik*. Munksgaard 1997, Kapitel 16, pp.235-256.

Biering-Sørensen F. Dr. Thomas Mohr. Winner of the Lars Sullivan Spinalis Prize 1996. *Spinal Cord* 1997;35:192.

Biering-Sørensen F. Obituary. Dr. Bodil Eskesen. *Spinal Cord* 1997;35:193.

1998

Ohry A, Weingarden H, Kiwerski J, Otom A, El-Masry WS, Kovindha A, Biering-Sørensen F. Clinical case of the month. Spinal injury rehabilitation complicated by psycho-social problems. *Spinal Cord* 1998;36:262-5.

Vaidyanathan S, Soni BM, Biering-Sørensen F, Bagi P, Wallberg AH, Vidal J, Borau A, Singh G, Sett P, Krishnan KR. Clinical case of the month. Recurrent bilateral renal calculi in a tetraplegic patient. *Spinal Cord* 1998;36:454-62.

Moser C, Kriegbaum NJ, Larsen SO, Høiby N, Biering-Sørensen F. Antibodies to urinary tract pathogens in patients with spinal cord lesions. *Spinal Cord* 1998;36:613-6.

Hartkopp A, Murphy RJL, Mohr T, Kjær M, Biering-Sørensen F. Bone fracture during electrical stimulation of the quadriceps in a spinal cord injured subject. *Arch Phys Med Rehabil* 1998;79:1133-6.

Hartkopp A, Brønnum-Hansen H, Seidenschur AM, Biering-Sørensen F. Overlevelse og traumatisk rygmærskade. Et epidemiologisk langtidstudie. *Ugeskr Læger* 1998;160:6207-10.

Brasso K, Sønksen J, Sommer P, Ødum L, Biering-Sørensen F, Iversen P, Kristensen JK. Seminal plasma PSA in spinal cord injured men: a preliminary report. *Spinal Cord* 1998;36:771-3.

Hartkopp A, Brønnum-Hansen H, Seidenschur AM, Biering-Sørensen F. Suicide in a spinal cord injured population in relation to functional status. *Arch Phys Med Rehabil* 1998;79:1356-61.

Gregersen H. FES – funktionel elektrisk stimulation og Freehand system. *Centernerven* 1998; Nr.21:4.

Nielsen K, Biering-Sørensen, Hansen HV. Estimation of total number of epithelial cells on the surface of hydrophilic catheters used for intermittent urinary bladder catheterization by use of fractionator method. *Acta Stereol* 1998; 17(1):83-85.

Biering-Sørensen. Functional electrical stimulation: Clinical experience and perspectives. International Symposium on Spinal Cord Injury and Repair II. Odense, Denmark. *J Neurologic Rehabil* 1998;12(4):184-5.

Mohr T, Van Soeren M, Graham TE, Kjaer M. Caffeine ingestion and metabolic responses of tetraplegic humans during electrical cycling. *J Appl Physiol* 1998; 85(3): 979-85.

Pilegaard H, Mohr T, Kjaer M, Juel C. Lactate/H⁺ transport in skeletal muscle from spina-cord-injured patients. *Scand J Med Sci Sports* 1998; 8(2): 98-101.

Klokke M, Mohr T, Kjær M, Galbo H, Pedersen BK. The natural killer cell response to exercise in spinal cord injured individuals. *Eur J Appl Physiol* 1998;79:106-9.

1999

Bülöw PM, Biering-Sørensen F. Paraplegia, a severe complication to epidural analgesia. *Acta Anaesthesiologica Scandinavica* 1999;43:233-5.

Sønksen J, Ohl DA, Momose H, Rocha FT, Barros TEP, Biering-Sørensen F. Clinical Case of the Month. Treatment of infertility. *Spinal Cord* 1999;37:89-95.

Barros TEP, Bohlman HH, Capen DA, Cotler J, Dons K, Biering-Sørensen F, Marchesi DG, Zigler JA. Clinical case of the month. Traumatic spondylolisthesis of the axis. Analysis of management. *Spinal Cord* 1999;37:166-71.

Sønksen J, Ohl DA, Giwerzman A, Biering-Sørensen F, Skakkebæk NE, Kristensen JK. Effect of repeated ejaculation on semen quality in spinal cord injured men. *J Urology* 1999; 161:1163-1165.

Biering-Sørensen F, Nielsen K, Hansen HV. Urethral epithelial cells on the surface on hydrophilic catheters after intermittent catheterization: cross-over study with two catheters. *Spinal Cord* 1999;37:299-300.

Biering-Sørensen F, Nielans H-M, Dørflinger T, Sørensen B. Urologic situation five years after spinal cord injury. *Scand J Urol Nephrol* 1999;33:157-61.

Nielsen OA, Biering-Sørensen F, Bötzel U, Gardner BP, Little J, Ohta H, Shrosbee R, Melwill R. Clinical Case of the Month. Posttraumatic syringomyelia. Spinal cord 1999;37:680-4.

Hartkopp A, Andersen JL, Harridge S, Crone C, Gruschy-Knudsen T, Kjær M, Mizuno M, Ratkevicius A, Quistorff B, Zhou S, Biering-Sørensen F. High expression of MHC I in the tibialis anterior muscle of a paraplegic patient. Muscle Nerve 1999;22:1731-7.

Tennant A, Grimby G, Marincek C, Phillips C, Ring H, Biering-Sørensen F, Tesio L, Thonnard J-L. Standardising outcome measurement in Physical Medicine and Rehabilitation across Europe. Eurorhab 1999;3-4:178-80.

Biering-Sørensen F. Treatment and rehabilitation of spinal cord injured patients. Danish approach and results. Annals Universitatis Mariae Curie-Sklodowska. Lublin, Poland, 1999;LIII(Sectio D, Medicina),Suppl.V:132-40.

Paraplegikerkredsen, m.fl.: Biering-Sørensen F, Bock JE. Patienten er rygmarvsskadet - en oplysningspjece om graviditet og fødsel. (Information brochure on pregnancy and delivery) Published by Paraplegiker-kredsen Revised edition December 1999.

Kjaer M, Pott F, Mohr T, Linkis P, Tornøe P, Secher NH. Heart rate during exercise with leg vascular occlusion in spinal cord-injured humans. J App Physiol 1999; 86(3): 806-11.

Murphy RJL, Hartkopp A, Gardiner PF, Kjær M, Béliveau L. Salbutamol Effect in Spinal Cord Injured Individuals Undergoing Functional Electrical Stimulation Training. Arch Phys Med Rehabil 1999;80:1264-7.

2000

Biering-Sørensen F, Schröder AK, Wilhelmson M, Lomberg B, Nielsen H, Højby N. Bacterial contamination of bath-water from spinal cord lesioned patients with pressure ulcers exercising in the water. Spinal Cord 2000;38:100-5

Faarvang KL, Müller P, Lomberg B, Biering-Sørensen F. Screening for bacteriuria in patients with spinal cord lesion. Dipstick test, micro-

scopic examination and urine culture. Spinal Cord 2000;38:106-8.

Mohr T. Elektrisk stimuleret muskeltræning af underkølestremiteterne hos rygmarvsskadede personer. Ugeskr Læger 2000;162:2190-4.

Biering-Sørensen F, Gregersen H, Hagen E, Haugland M, Keith M, Larsen CF, Leicht P, Nielsen FH, Rabischong E, Sinkjær T. Forbedret håndfunktion hos tetraplegikere ved elektrisk stimulering via implanterede elektroder. Ugeskr Læg 2000;162:2195-8.

Koskinen SOA, Kjær M, Mohr T, Biering-Sørensen F, Suuronen T, Takala TES. Type IV collagen and its degradation in paralysed muscle: Effect of functional electrical stimulation. Muscle Nerve 2000;23:580-9.

Dela F, Stallknecht B, Biering-Sørensen F. An intact central nervous system is not necessary for insulin-mediated increases in leg blood flow in humans. Pflügers Arch - Eur J Physiol 2000;441:241-50.

Hansen NL, Hansen S, Crone C, Christensen LOD, Petersen N, Nielsen JE, Biering-Sørensen F, Nielsen JB. Synchronization of lower limb motor units in spastic patients. Clinical Neurophysiology 2000;53(Suppl.):178-86.

Biering-Sørensen F. Rehabilitation of para- and tetraplegics. In: Proceedings from European Association of Neurosurgical Societies (EANS) Course in Opio-Nice, France, September 17th-21st, 2000.

Heller BW, Granat MH, Hermens HJ, Baardman G, Biering-Sørensen F, Marqués MA, Veltink PH. Clinical rehabilitation using electrical stimulation via telematics (CREST). Proceedings of the IFESS'2000 (International Functional Electrical Stimulation Society) meeting in Aalborg, Denmark.

Hasler JP, Molin M, Lentz K, Gerkens V, Østergaard S, Scott E, Vazquez SL, del Rio IN, Cloostermans JThMM. Observational gait analysis for evaluating the effectiveness of functional electrical stimulation enhanced gait. Proceedings of the IFESS'2000 (International Functional Electrical Stimulation Society) meeting in Aalborg, Denmark.

Biering-Sørensen F, Maxwell DJ, Heller BW, Marqués AM, Cloostermans JThMM, Hermens HJ and the CREST-group. Surface Functional Electrical Stimulation (FES) for Walking in Incomplete Spinal Cord Lesioned (SCL) Individuals. A Multicentre Trial. Proceedings of the IFESS'2000 (International Functional Electrical Stimulation Society) meeting in Aalborg, Denmark. CD-ROM conference proceedings 1996-2001, June 2001. Release 3.0. Editor Paul Meadows.

Biering-Sørensen F. Rehabilitation research in Denmark. *Rehab Arcticus* 2000;Nr.29:19-20.

2001

Gam AN, Hviid I, Rasmussen GG, Rasmussen JM, Biering-Sørensen F, Pedersen BK. Bevægeapparatets arti. Editorial. *Ugeskr Læger* 2001;163:271.

Østergård S, Gerkens V, Molin M, Lentz K. Ny behandlingsmetode testes. *Fysioterapeuten* 2001;Nr.2:14-6.

Østergård S, Gerkens V, Molin M, Lentz K. Besværlige skridt. *Fysioterapeuten* 2001;Nr.2:16-9.

Biering-Sørensen F, Haigh R, Holgersson MH, Ravnborg MH. Brugen af effektmål i fysiurgisk/reumatologisk rehabilitering. Resultat af en spørgeskema-undersøgelse. *Ugeskr Læg* 2001;163:612-6.

Kjær M, Mohr T, Dela F, Secher N, Galbo H, Olesen HL, Biering-Sørensen F, Schifter S. Leg uptake of calcitonin gene-related peptide during exercise in spinal cord injured humans. *Clin Physiol* 2001;21:32-8.

Biering-Sørensen B, Egebart J, Hilden J, Biering-Sørensen F. Reproducibility and validity of a questionnaire filled in by spinal cord lesioned individuals before regular follow-up. *Spinal Cord* 2001;39:161-7.

Biering-Sørensen F. Rygmarvsskade - den moderne behandling. *Ugeskr Læg* 2001;163:2766-9.

Biering-Sørensen F, Gregersen H, Hansen HV, Nielsen L, Ranneries P, Rølsager K, Østergård S. Tværfaglig elektronisk patientjournal og kliniks database i ét. SCIBase. *Ugeskr Læger* 2001;163:3207-12.

Biering-Sørensen F, Bagi P, Høiby N. Urinary tract infections in patients with spinal cord lesions. Treatment and prevention. *Drugs* 2001;61:1275-87.

Læssøe L, Sønksen J, Bagi P, Biering-Sørensen F, Ohl DA, McGuire EJ, Kristensen JK. Effects of ejaculation by penile vibratory stimulation on bladder reflex activity in a spinal cord injured man. *J Urol* 2001;166:627.

Kjær M, Mohr T, Biering-Sørensen F, Bangsbo J. Muscle enzyme adaptation to training and tapering-off in spinal-cord-injured humans. *Eur J Appl Physiol* 2001;84:482-6.

Mohr T, Dela F, Handberg A, Biering-Sørensen F, Galbo H, Kjær M. Insulin action and long-term electrically induced training in individuals with spinal cord injuries. *Med Sci Sports Exerc* 2001;33:1247-52.

Biering-Sørensen F, Walter S. Urologisk behandling og kontrol af rygmarvsskadede personer. *Ugeskr Læg* 2001;163:5168-71.

Jensen G, Schiødt AV, Sanders S, Nordenbo A, Fischer-Rasmussen W, Lose G, Biering-Sørensen F, Andersen JT, Nielsen JB, Walter S. Vandladningsforstyrrelser ved neurologisk sygdom. Vejledning for neurologer. *Ugeskr Læg* 163, Klaringsrapport Nr.11, 2001, pp.1-8.

Biering-Sørensen F, Sønksen J. Sexual function in spinal cord lesioned men. Scientific review. *Spinal Cord* 2001;39:455-470.

Inmann A, Haugland M, Haase J, Biering-Sørensen F, Sinkjær T. Signals from skin mechanoreceptors used in control of a hand grasp neuroprosthesis. *Neuroreport* 2001;12:2817-20.

Kjær M, Dela F, Biering-Sørensen F, Secher NH, Bangsbo J, Mohr T, Galbo H. Fatty acid kinetics and carbohydrate metabolism during electrical exercise in spinal cord-injured humans. *Am J Physiol Regulatory Integrative Comp Physiol* 2001;81:R1492-8.

Stallknecht B, Lorentsen J, Enevoldsen LH, Bülow J, Biering-Sørensen F, Galbo H, Kjær M. Role of the sympathoadrenergic system in adipose tissue metabolism during exercise in humans. *J Physiol* 2001;536:283-94..

Biering-Sørensen F, Biering-Sørensen M. Sleep disturbances in spinal cord injured. An epidemiological questionnaire investigation, including a normal population. *Spinal Cord* 2001;39:505-13.

Haigh R, Tennant A, Biering-Sørensen F, Grimby G, Marincek C, Phillips S, Ring H, Tesio L, Thonnard J-L. The use of outcome measures in physical medicine and rehabilitation within Europe. *J Rehabil Med* 2001;33:273-8.

Biering-Sørensen F. Para- og tetraplegi.. In: J.Friis, P.Junker, C.Manniche, J.Petersen, K.Stengaard-Pedersen (eds.). *Reumatologi. FADL's Forlag* 2001, Kapitel 36, pp.629-46.

Biering-Sørensen F. Book review: A Guide and Ressource Directory to Male Fertility following Spinal Cord Injury/Dysfunction. Maria J. Amador, Charles M. Lynne, Nancy L. Brackett. Published by: The Miami Project to Cure Paralysis, University of Miami 2000, 44 pp. *Spinal Cord* 2001;39:503.

Paraplegikerkredsen, Several authors from the Clinic: SEX – en guide for rygmarsvskadede og deres partnere. (SEX – a guide for spinal cord lesioned and their partners). Paraplegikerkredsen 2001.

2002

Biering-Sørensen F. Urinary tract infection in individuals with spinal cord lesion. *Curr Opin Urol* 2002;12:45-9.

Pelck R, Stenbygaard LE, Biering-Sørensen F. Strålemyelopati. En beskrivelse af fire tilfælde. *Ugeskr Læger* 2002;164:2048-9.

Harridge SD, Andersen JL, Hartkopp A, Zhou S, Biering-Sørensen F, Sandri C, Kjær M. Training of low-frequency stimulation of tibialis anterior in spinal cord-injured men. *Muscle Nerve* 2002;25:685-94.

Biering-Sørensen F. Rehabilitation of patients with para- and tetraplegia. In: Course Book from European Association of Neurosurgical Societies (EANS) European Training Course in Rome, Italy, February 3rd-7th, 2002.

Biering-Sørensen F. Rygmarsvskadedes vandladning. *LoFric Dialogen*. 2002; Nr.5:3-4.

Biering-Sørensen F. Book review: Rehabiliteringsmedicin. 4.udgave. Höök O, ed. Stockholm: LIBER AB, 2001. *Ugeskr Læg* 2002;164:2633-4.

Biering-Sørensen F. Kvalitetssikring af rehabilitering af rygmarsvskadede. In: Kvalitet i rehabilitering – sådan kommer vi videre. MarselisborgCentrets nationale konference 19.november 2001, pp.40-3. Published 2002.

RYK - Rygmarsvskadede i Danmark, Several authors from the Clinic. Para- og Tetraplegi – Handbook for spinal cord lesioned. Dansk Handicap Forbund 2002, 215 pp.

Biering-Sørensen F. Rygmarsvskadede og inkontinens. *Kontinensnyt*. 2002, Nr.2:6-7.

LEADING POSITIONS AT THE CLINIC OVER THE YEARS

Administrators:

Edith Gervig, 1952 to 1954
Karen Margrethe Jensen, 1954 to 1979
Knud Børge Nielsen, 1979 to 1987
Preben Christoffersen, 1987 to 1989
Anne Mette Jensen, 1990 to 1994
Nina Dencker, 1994
Hanne Gregersen, since 1995

Chief physiotherapists:

Wind, 1952 to 1954
Karen Møller, 1954 to 1978
Lissen Henriksen, 1978 to 1979
Lene Lundsted, 1979 to 1996
Susanne Østergaard, since 1996

Head occupation therapists:

Lis Kai Nielsen, 1952 to 1956
Ulla Ryø, 1956 to 1962
Line Schmidt, 1962 to 1985
Hanne Gregersen, since 1985

Head nurses:

Mary Madsen, 1952 to 1975
Orla Olsen, 1975 to 1993
Anne Mertz, 1994 to 1995
Susanne Lorentzen, 1995 to 1997
Birgitte Pontoppidan, 1997 to 1998
Hanne Hagelin, since 1998
Mette Nistrup, since 1999

Chief physicians:

Ole Remvig, 1952 to 1977
Bodil Eskesen, 1960 to 1972
Claus Helleesen, 1973 to 1977
Johannes Friis, 1978 to 1992
Fin Biering-Sørensen, since 1986

