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Editorial: Management of Spasticity in Multiple Sclerosis

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Spasticity in multiple sclerosis (MS) can be a blessing or a curse. It is a blessing in that it adds strength to the support muscles that hold us erect against gravity when those muscles have been weakened due to loss of input from the upper motor neurons. When we look at strength in a spastic leg, we find that the weakest muscles are those that pick up the leg (ie, the hip and knee flexors and the ankle dorsiflexors). The strongest muscles are the quadriceps, which extend the knees; the glutei, which extend the hip; and the gastroc-solei, which plantar flex the ankle. Picking up the leg is typically much more difficult than standing. One aspect of spasticity is loss of the precision of muscle control. It is hard to partially relax a spastic muscle; consequently, going down stairs is typically more difficult for patients than going up stairs. The muscle is strong enough to lift the body's weight, but when an attempt is made to bend the hip and knee, the muscle is likely to give way because the gradations in contraction necessary to hold one's weight with the knee bent cannot be accomplished.

The resulting stiffness and abnormal pull on joints from spasticity can cause many problems, including abnormal stresses on joints, particularly the knees; muscle pain and soreness; and flexor and extensor spasms. Flexor spasms, in which the knees pull up to the chest periodically or with a minimal stimulus, can be troublesome and make sitting in a chair difficult. Extensor spasms, which cause the body and legs to extend, can push an individual out of his or her chair or cause the individual to tip over backwards. Spasticity in the adductor muscles of the legs makes perineal hygiene difficult, interferes with sexual activity, and can cause pressure problems where the knees rub together.

Management of spasticity can be helpful, but considerable care must be taken. Available medical treatments include baclofen, tizanidine, dantrolene, and diazepam. All of these are sedating at therapeutic doses, and many patients cannot get adequate muscle relaxation without too much sedation. Additionally, all of these medications cause weakness, so that adequate control of spasticity almost invariably causes some weakness in addition to some sedation. The dose of a muscle relaxant used to manage spasticity is "enough and not too much." The goal is to reduce spasticity without causing too much weakness.

In those patients who cannot get adequate muscle relaxation without too much sedation, intrathecal baclofen may be a useful option. By providing a high local concentration in the lumbar cord with a very low systemic concentration, muscle relaxation can be accomplished without sedation.

As indicated in the paper by Jarrett et al, a goal-oriented approach is important. Individuals who are to undergo intrathecal baclofen therapy need to understand what the therapy can and cannot do. It will not improve the tradeoff between spasticity and weakness. Added weakness in the legs may make standing more difficult or impossible while improving other problems caused by the spasticity. However, with a clear understanding of what can be accomplished, most patients in whom intrathecal therapy is indicated will be happy with the results.

Managing Spasticity in People With Multiple Sclerosis A Goal-Oriented Approach to Intrathecal Baclofen Therapy

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Abstract

The effectiveness of intrathecal baclofen therapy (ITB) in the management of severe spasticity in people with multiple sclerosis (MS) was reviewed retrospectively. The multidisciplinary team reviewed the medical, therapy, and nursing notes of 19 people with MS who were treated with ITB. The audited information included surgical procedures, postoperative complications, medical side effects, dose requirements, and multidisciplinary input.

Seventeen people were included in the audit. A total of 23 problems and 34 functional goals as objects for ITB treatment were recorded. Eighty-seven percent of the patients had sustained improvement in at least one problem, and 79% in at least one goal. Only two patients had no sustained improvement in any problem or goal. These results suggest that ITB can be an effective intervention in people with severe spasticity in MS. However, this approach requires careful patient selection, a dynamic goal-oriented approach, expert implantation, and ongoing evaluation of individual responses to treatment over time.

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Intrathecal baclofen (ITB) is an effective treatment option in the management of severe spasticity of either cerebral^{1,2} or spinal origin,^{3,4} and benefit is sustained over a period of time.^{5,6} The use of ITB, however, is not without risk. To assure that complications are

minimized while therapeutic benefit is maximized, a coordinated approach by an experienced team including a neurologist, a neurosurgeon, physiotherapists, nurses, and occupational therapists is advisable.^{7,8}

Baclofen (Lioresal[®]) acts by binding to gamma-aminobutyric acid (GABA) receptors. It has a presynaptic inhibitory effect on the release of excitatory neurotransmitters.⁹ Postsynaptically it decreases the firing of motor neurons.¹⁰ This results in inhibition of monosynaptic and polysynaptic spinal reflexes,¹¹ with associated reductions in spasm, clonus, and pain. Delivering baclofen intrathecally accentuates its antispasticity effect while minimizing the troublesome systemic side effects associated with oral intake.

Baclofen can be administered intrathecally via a subcutaneously implanted electronic pump with a reservoir and a catheter (SynchroMed Infusion System, Medtronic Ltd.) with the tip placed at L2/L3 or higher. This system is externally programmed using a computer and telemetry, allowing different dose regimes to be delivered. A 24-hour dose can have up to 10 steps, each prescribing the dose, rate, and duration, allowing the delivery of complex regimes. The two pumps currently available are of identical diameter (70.4 mm) and battery life (five to seven years) but differ in reservoir size (10 mL and 18 mL). It should be noted, however, that our recommendations are based on our experience with previous models, which had a battery life of three to five years.

This paper is a retrospective audit of one unit's experience of using ITB during a five-year period. It provides evidence of efficacy as recorded in clinical practice, demonstrating the need for a comprehensive, multidisciplinary management strategy, incorporating clearly defined goals and responsive to changing clinical needs.

Methods

The medical, therapy, and nursing notes were retrospectively reviewed for 19 people with MS who had ITB pumps implanted for management of severe spasticity. The review panel consisted of two neurologists, two nurse specialists, and one physiotherapist involved in the subjects' care. The problems and goals agreed upon before treatment were identified from the notes for each individual. Problems related to impairment or symptoms, such as spasms. Goals—which had been set from the patient perspective and had to be realistic and potentially achievable—concerned improvement of function or comfort, such as improved sitting posture. To evaluate treatment outcome, the sustained improvement in the problems and the level of achievement of the goals over time were identified and graded by the multidisciplinary team. Achievement had to last for at least three months. The following grading system was used: nil (not improved or achieved), mild, moderate, or marked improvement or achievement. Transient response was also recorded.

Surgical procedures, postoperative complications, medical side effects, dose requirements, and multidisciplinary involvement were noted. Outcome measures used to assess spasticity were documented, but these varied during the five years, making comparison difficult. Therefore, they were not included in the data.

Surgical Technique

Certain aspects of the surgical technique were modified to make the procedure more effective. These include where the Touhy needle is inserted and the position of both the lumbar and abdominal incisions. Current practice is summarized as follows:

- The body of the patient is flexed and placed in the lateral position with the upper arm elevated on an arm support, thus exposing the abdomen for the pump placement, but also ensuring the true upright position. This eases the insertion of the lumbar catheter.
- A 4- to 5-cm horizontal incision over the area of lumbar vertebrae 3 and 4 has proved sufficient. Therefore, a longitudinal incision has been abandoned, because it makes it more difficult to secure the catheter.
- The Touhy needle (SIMS Portex Ltd, Kent, UK) is inserted off the midline to pass through the muscle of erector spinae rather than through the interspinous ligament. This reduces cerebrospinal fluid (CSF) from flowing back, perhaps because the muscle makes a more permanent seal.
- The needle is passed directly through to the CSF rather than using the standard air injection method. Once the dura is penetrated and clear CSF flow is demonstrated, the stylet of the needle is replaced to minimize leakage.
- The catheter is inserted with the guidewire cephalad to about 10 cm or between the third and fourth marks. If it will not pass initially, the needle is withdrawn a few millimeters, which usually unblocks the tip opening, although CSF flow must be reconfirmed. First the needle, then the guidewire, are removed, the latter by holding the catheter at the entry point to the spine and pulling the guidewire at the loose end. A curved channel is the best way to anchor the catheter as this takes strain off the entry point and directs the catheter toward the pump.
- The abdominal pocket is fashioned at the same time by a second surgeon, either in the left or right abdominal quadrant. An oblique 10- to 12-cm incision is sufficient. This prevents producing a relatively avascular flap and avoids an incision line that lies over the pump itself. Both of these modifications can aid in reducing infection. The pocket is then fashioned medial to this incision. A dummy pump is useful for determining the correct size.
- While the first surgeon is still at work creating the anchorage for the catheter, the pocket can be packed with a povidone-iodine-soaked swab both to minimize oozing and reduce infection.
- Any tunneling device can be used to connect the two incisions. Once the pump is prepared, it can be connected to the suitably shortened catheter, leaving enough slack to allow some movement from subsequent spasms. The pump is inserted with the catheter lying behind it, and the incisions are closed.

Results

Nineteen sets of notes were reviewed: 17 were included in the audit, one patient's care had been transferred locally, and one patient had died (unrelated to ITB therapy). The review group consisted of six men and 11 women with a mean age of 49 years (range, 38 to 67 years). The mean duration of therapy was 43 months (range, four to 79 months). Twenty-seven pumps (25 electronic, two manual) were implanted; eight were replacements—four due to expiration of batteries and three were due to malfunction (one electronic and two manual). One pump was replaced and relocated onto the other side of the abdomen because of an infection at the pump site. Five catheters were replaced, and one catheter was resutured.

Problems and Goals

Twenty-three problems were identified—10 related to painful spasms, eight to non-painful spasms, four to spasticity, and one to pain not associated with spasticity. Thirty-four treatment goals relating to spasticity management were set (Table 1). The most common goals were improvement of transfers and pain relief.

Table 1. Type and Number of Goals Relating to Spasticity Management.

Goals identified	No.
Improve transfers	9
Relieve pain	8
Improve sitting	6
Use standing equipment	4
Improve perineal access	3
Improve sleep	2
Reduce systemic toxicity	1
Improve scoliosis	1
Total	34

Sustained improvement to varying degrees was seen in 87% of the problems, with the largest number of patients showing moderate improvement (44%, n = 10) (Figure 1). Although three patients showed no sustained improvement, all three problems demonstrated a transient improvement: moderate in two patients, and marked improvement in the third patient.

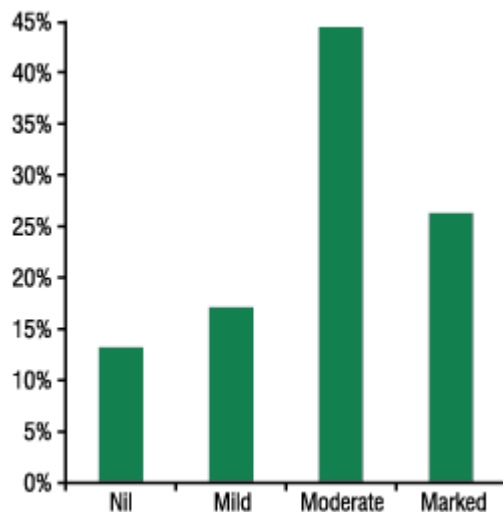


Figure 1: Percentage of problems with sustained improvement following ITB therapy.

The patients attained sustained goal achievement in 79% of the goals they had set (Figure 2). Of the 21% of goals for which achievement was not sustained, 15% (n = 5) showed transient achievement: mild, 6% (n = 2); moderate, 3% (n = 1); and marked, 6% (n = 2).

Fifteen patients (88%) had sustained improvement in at least one problem and one goal. Only two patients (12%) had no sustained benefit for any problem or goal.

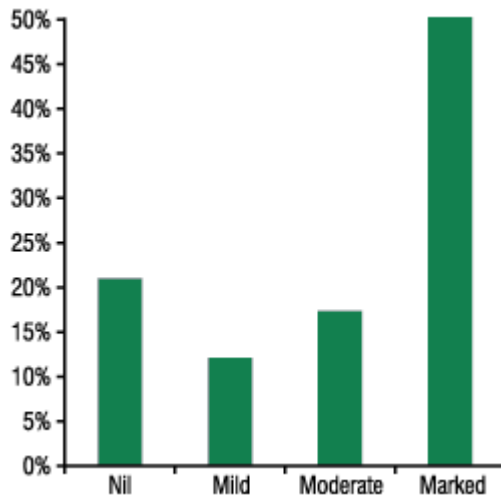


Figure 2: Percentage of goal achievement following ITB therapy.

Postoperative and Follow-up Complications

Table 2 summarizes the postoperative complications and medical side effects experienced by this group. Transient, low-grade fever was the most common postoperative complication; it tended to resolve in 24 to 48 hours. The main medical side effect is excessive weakness, predominantly truncal. This required frequent dose titration, balancing reduction in tone against exposing underlying weakness to avoid worsening upper body function and wheelchair posture.

Table 2. Type and Number of Postoperative Complications and Medical Side Effects.

Postoperative complications	No.
Transient fever	11
Excessive weakness	5
Headache	4
Drowsiness	3
Respiratory depression	3
Lumbar wound complications	2
Abdominal wound complications	2
Neck stiffness and pain	2
Other	8
Total	40

Five patients reported weight gain following use of ITB. This has the potential of causing difficulties refilling the reservoir, even when using a template, and on one occasion an x-ray was required to facilitate refilling.

Four radiopaque studies have been carried out to assess the patency of systems demonstrating suboptimal performance. One study was complicated by a baclofen overdose. The person was ventilated overnight until the effects of the baclofen had worn off and subsequently made a complete recovery.

Dose Adjustments

In response to clinical need, from 0 to 10 dose adjustments were required per patient per year. Currently, 12 people are being administered a continuous infusion of a specific dose throughout 24 hours. Five people have a repetitive sequence of between two and four steps. The dose during 24 hours ranges from 3 µg to 625 µg, with a mean of 283.2 µg. The length of time needed to reach an optimal therapeutic dose is extremely variable and is an ongoing process based on the clinical status of the person. Follow-up of individual patients ranged from one to six months, which was dependent upon how well the spasticity/pain was being controlled and the dose and concentration of drug used. In addition, one patient experienced temperature-related changes in tone: during the hot summer months less baclofen was required for spasm control without weakness. This variation is now managed by twice-yearly dose adjustments. Four patients described a "wearing off effect" within one month before requiring a pump refill, which resolved 24 hours after having their pump refilled.

Multidisciplinary Input

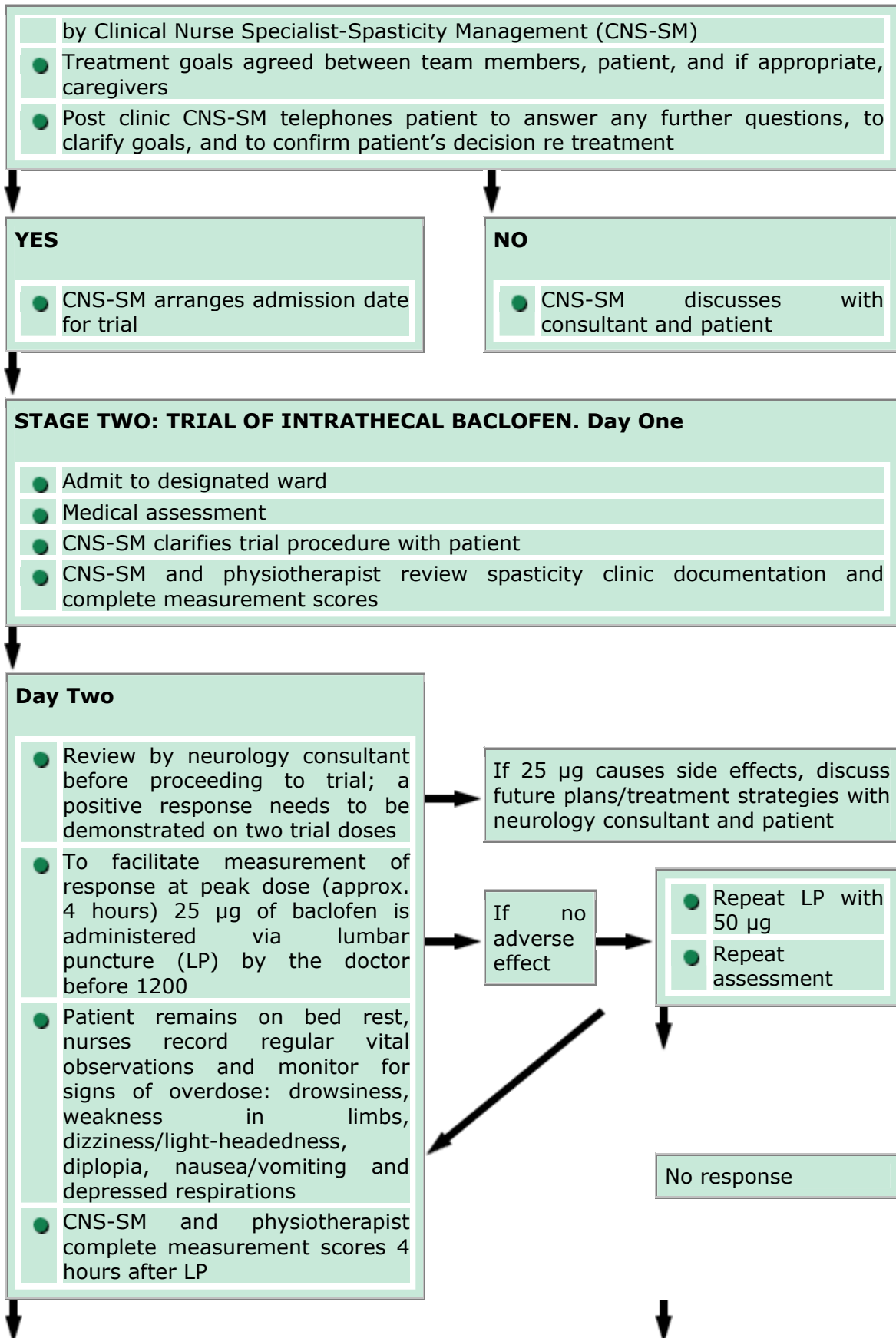
Following implantation, 12 patients had their wheelchair posture and seating reassessed and revised. The adjustments improved posture and comfort when sitting. Twelve patients made gains with targeted therapy input as an outpatient, and seven benefited from a focused inpatient rehabilitation admission. Following neurophysiotherapy, four patients used standing equipment who previously had been unable to use it.

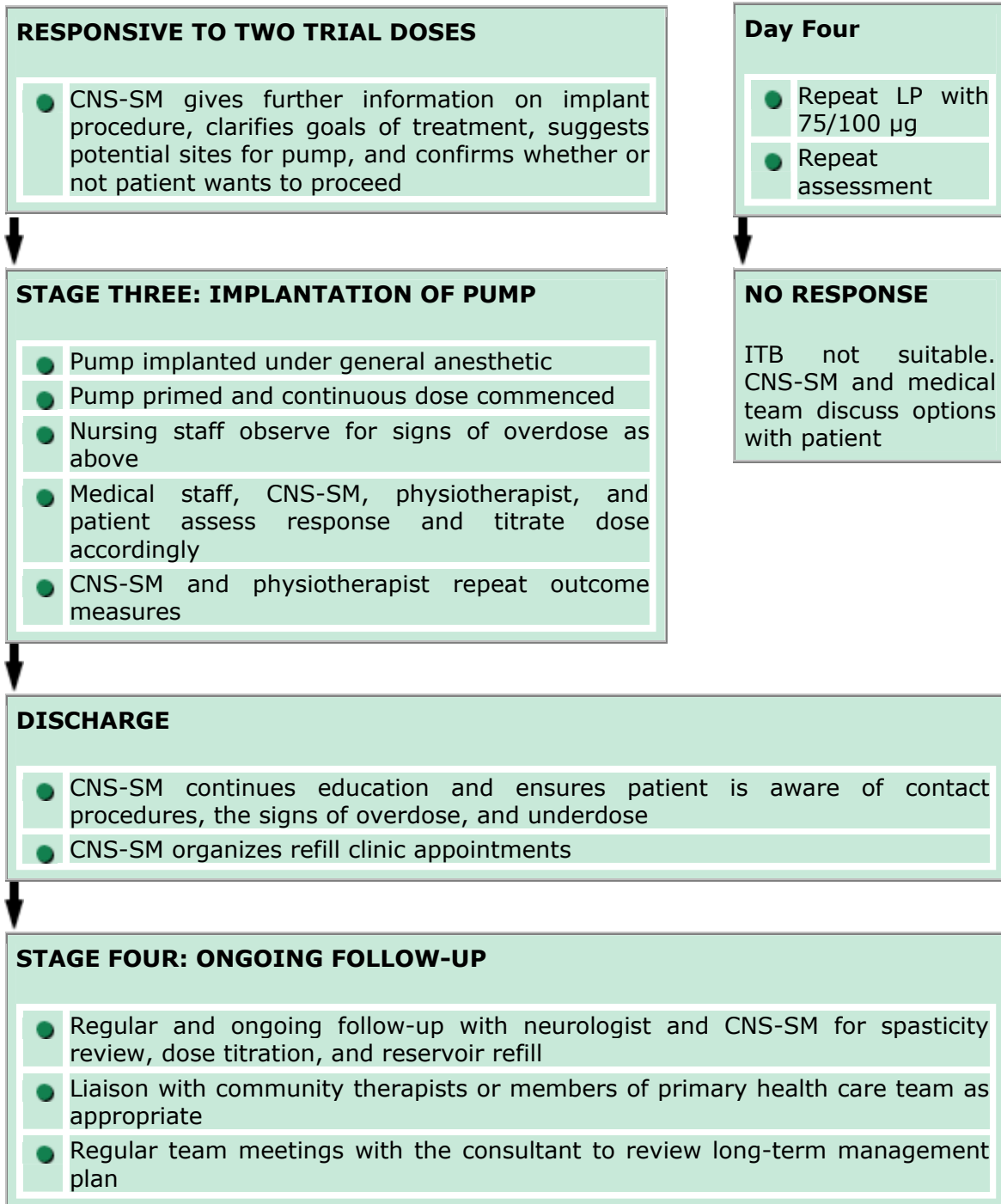
Discussion

Intrathecal baclofen is not without complications, but in carefully selected patients it can be an effective treatment for spasticity in MS. To maximize efficacy, it should be only one part of a comprehensive multidisciplinary management strategy that incorporates clearly defined goals that are responsive to changing needs. The key points to consider when using ITB include providing an infrastructure to ensure a coordinated multidisciplinary service, identifying problems and goals to guide patient management, and being alert to any practical difficulties possibly unique to this treatment. The audit demonstrated that a successful ITB service requires collaboration between the patient and a number of health care professionals, including neurologists, neurosurgeons, therapists, and nurses at all four stages of the treatment process: assessment, trial, implant, and ongoing follow-up (Table 3).

Table 3. Algorithm for using ITB in the management of severe spasticity.

STAGE ONE: MULTIDISCIPLINARY ASSESSMENT IN SPASTICITY CLINIC	
●	Trial of intrathecal baclofen chosen as treatment option
●	Verbal and written information given to patient by team and ongoing education





Assessment

Skilled multidisciplinary assessment allows accurate selection of individuals who may benefit from ITB therapy and the identification of achievable goals. Education of patients at each stage is key to informing them about the process and, most important, being clear about what ITB can and cannot achieve. The results show that identifying problems and goals amenable to treatment provides a useful focus for the patient and the multidisciplinary team to assess effectiveness.

Trial

The trial stage is important, as it allows the patient to experience the effect of ITB, and it indicates whether a person will respond to the therapy. However, it does not provide an indication of the appropriate therapeutic dose. This is best achieved by multidisciplinary assessment of response and careful post-implant dose titration.

Implant

A consultant neurosurgeon implanted all the pumps; this led over time to the modification of the surgical technique to optimize the functioning of the implanted system. Although it is usually a straightforward neurosurgical procedure, pump implantation is intricate. Patients with spasticity can be thin and often have unusual fixed deformities, which can lead to neurosurgical complications. These include leaking back of cerebrospinal fluid around the catheter track, damage to or dislodging of the catheter, and infection and breakdown of the abdominal wound. For these reasons, it is suggested that the procedure be carried out by an experienced surgeon. This differs from other common neurosurgical procedures, such as the implantation of lumboperitoneal shunts, for which senior trainees have the lowest complication rate.¹²

Postsurgical complications and catheter problems have been previously documented^{5, 13-16} and were evident in this review. A one-piece new catheter design (rather than a two-piece design) has recently been introduced to try to minimize catheter revisions. It is easier to implant and reduces the need for dye studies to check the patency of systems. Such studies require the pump to be stopped, emptied, and filled with sterile saline. Because such a procedure carries the risk of drug overdose, an inpatient admission is required, adding to the cost of running the service.

Careful consideration needs to be given regarding the size of pump to be implanted. A smaller pump may be preferred in a patient with a low body weight or little muscle bulk, but the relatively small advantages in the dimensions of the pump need to be outweighed against losing 8 mL of reservoir volume, which can mean frequent refilling of the pump. One patient in our review with a 10-mL reservoir required refilling on a monthly cycle. This cannot be counteracted by using a higher concentration of baclofen, as it is not currently available in a stable form above 3,000 µg/mL.

Ongoing Management

Skilled assessment and treatment by a neurophysiotherapist and occupational therapist is pivotal to spasticity management and continues to be so when using ITB. Postural tone and spasticity can be affected by an individual's position in lying, standing, and sitting. Selecting appropriate physical treatment strategies will help to manage tone and to minimize secondary soft tissue changes. Standing (with or without supportive devices) with good limb and body alignment can help to improve tone.¹⁷ Symmetrical posture and alignment in a wheelchair prevents joints and muscles from becoming fixed in abnormal and often painful positions and can promote functional use of the upper limbs.

Adequate time and personnel to carry out safe, effective follow-up need to be incorporated into delivering an ITB service, without which the benefits of ITB may not be realized. In this audit, monthly refill clinics were run by a neurologist and a nurse practitioner, during which a spasticity management review was completed, the pump refilled, dose adjustments made, and any further education with regard to tone management was discussed. If required, liaison with members of their primary health care team or outpatient therapists helped to maximize an individual's spasticity management.

Patients who experienced a "wearing off effect" may be sensitive to changes in the stability of baclofen. To prevent this in practice, the pumps of these patients are refilled earlier than indicated by their low reservoir alarm date.

A backup system to respond to emergencies out of hours also needs to be established. One method is to designate a ward where the nursing staff are trained in certain aspects of ITB and have access to detailed protocols. In the event of a complication out of hours they can offer support or put the patient or any member of their primary health care team in contact with a neurologist. A further advantage of this system is that the nurse practitioner (under the supervision of a neurologist) is able to forewarn the ward nurses and doctors of specific patients who are experiencing problems and may require advice or admission outside of normal office hours. This has facilitated smoother admissions into the hospital.

Conclusion

This review of data on 17 patients with MS who use ITB demonstrates that it can be an effective treatment for improving problems relating to spasticity and for achieving specific treatment goals. The service requires a multidisciplinary, coordinated approach during the assessment, selection, implantation phase, and long-term follow-up. A further study to examine the cost implications of this treatment would be timely.

References

1. Albright AL, Cervi A, Singletary J. Intrathecal baclofen for spasticity in cerebral palsy. *JAMA*. 1991;265:1418-1422.
2. Meythaler JM, McCary A, Hadley MN. Prospective assessment of continuous intrathecal infusion of baclofen for spasticity caused by acquired brain injury: a preliminary report. *J Neurosurg*. 1997;87:415-419.
3. Penn RD, Savoy SM, Corcos D, et al. Intrathecal baclofen for severe spinal spasticity. *N Engl J Med*. 1989;320:1517-1521.
4. Loubser PG, Narayan RK, Sandin KJ, et al. Continuous infusion of intrathecal baclofen: long-term effects on spasticity in spinal cord injury. *Paraplegia*. 1991;29:48-64.
5. Coffey JR, Cahill D, Steers W, et al. Intrathecal baclofen for intractable spasticity of spinal origin: results of a long-term multicenter study. *J Neurosurg*. 1993;78:226-232.
6. Ochs G, Struppler A, Meyerson BA, et al. Intrathecal baclofen for long-term treatment of spasticity: a multi-centre study. *J Neurol Neurosurg Psychiatry*. 1989;52:933-939.
7. Becker WJ, Harris CJ, Long ML, et al. Long-term intrathecal baclofen therapy in patients with intractable spasticity. *Can J Neurol Sci*. 1995;22:208-217.
8. Gardner B, Jamous A, Teddy P, et al. Intrathecal baclofen—a multicentre clinical comparison of the Medtronic Programmable, Cordis Secor and Constant Infusion Infusaid drug delivery systems. *Paraplegia*. 1995;33:551-554.

9. Fox S, Krnjevic K, Morris ME, et al. Action of baclofen on mammalian synaptic transmission. *Neuroscience*. 1978;3:495-515.
10. Davies J. Selective depression of synaptic excitation in cat spinal neurones by baclofen: an iontophoretic study. *Br J Pharmacol*. 1981;72:373-384.
11. Davidoff RA, Sears ES. The effects of Lioresal on synaptic activity in the isolated spinal cord. *Neurology*. 1974;24:957-963.
12. Renier D, Lacombe J, Pierre-Kahn A, et al. Factors causing acute shunt infection. Computer analysis of 1174 operations. *J Neurosurg*. 1984;61:1072-1078.
13. Porter B. A review of intrathecal baclofen in the management of spasticity. *Br J Nurs*. 1997;6:253-260,262.
14. McLean BN. Intrathecal baclofen in severe spasticity. *Br J Hosp Med*. 1993;49: 262-267.
15. Penn RD. Intrathecal baclofen for spasticity of spinal origin: seven years of experience. *J Neurosurg*. 1992;77:236-240.
16. Teddy P, Jamous A, Gardner B, et al. Complications of intrathecal baclofen delivery. *Br J Neurosurg*. 1992;6:115-118.
17. Edwards S. Physiotherapy management of established spasticity. In: Sheean G, Barnes MP, eds. *Spasticity Rehabilitation*. London, Great Britain: Churchill Communications Europe Ltd; 1998:71-89.

Sweat Response During Submaximal Aerobic Exercise in Persons With Multiple Sclerosis

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Abstract

The relationship between the metabolic, thermal load of exercise, sudomotor (sweat) response, and core temperature was examined in 20 individuals with multiple sclerosis (MS) and eight non-MS control subjects. Participants performed an incremental bicycle test of maximal aerobic power (VO₂peak) from which 50% of VO₂peak was calculated. A 50% VO₂peak endurance test was performed while subject wore a full-body water garment. Initial circulating water temperature of 27°C was increased 2°C every three minutes to enhance the thermal load. Core, skin, and circulating water temperatures were monitored. Onset of sweating was measured at five sites. Results showed that 50% of the subjects with MS had an abnormally low sweat response in spite of relatively high skin temperatures (> 35°C) at several sites. The thermal load (kJ/kg) of these subjects at test termination was 89% higher than that of the MS subjects with a normal sweat response. Clinicians should be aware that sweat response may be blunted or absent in many patients with MS. Guidance for ways to enhance heat dissipation under adverse climatic conditions or during activities that increase whole body metabolism should be provided to patients with MS. Perhaps of even greater importance was the finding that increased core and skin temperature during exercise did not result in the appearance of any MS-related symptom in 95% of our sample. This finding is in direct conflict with much of the clinical literature.

Suggested citation: Sweat Response During Submaximal Aerobic Exercise in Persons With Multiple Sclerosis. Mulcare, JA, et al. *Int J MS Care* [Serial on-line]. December 2001;3(4).

In performing aerobic exercise, the body's primary mechanism for eliminating metabolic heat generated from contracting muscles is the sudomotor (sweat) response. This response is initiated when skin temperature reaches approximately 35°C. Sweating is controlled from the preoptic area of the hypothalamus, which receives input from peripheral thermosensory nerves, the final portions of which are myelinated fibers in the spinal cord and the lower brain. The entire efferent path is composed of myelinated nerves, hence subject to damage by lesions such as those seen in multiple sclerosis (MS). In earlier research involving patients with MS, the sudomotor response had been reported to be abnormal in a high percentage of individuals (42% to 60%).^{1,2} It is unknown whether decreased sweating in some patients is due to central lesions affecting the autonomic pathways for sweating—that is, from the hypothalamus—or whether the reported inability to sweat is a matter of patients not exerting themselves sufficiently to elicit the sweat response. It also has been reported that the higher the degree of

disability (eg, higher scores on the Kurtzke Expanded Disability Status Scale [EDSS]), the more extensive the loss of sudomotor responses.¹

In reviewing earlier studies, there are two distinct methodological problems that need to be addressed to properly examine this phenomenon. First, sudomotor response must be examined in the context of a valid measurement of core temperature (T_c). In previous research, core temperature often has been measured orally,^{1,2} which is not a reliable measure of T_c for research purposes.³ Previous research has shown that a valid, minimally invasive technique that approximates true core temperature can be obtained using the rectal probe method.^{4,5} The second methodological concern has been the examination of sudomotor response in conjunction with conditions that would simulate a dynamic thermal phenomenon, and not merely during quiet rest (eg, light physical activity or exercise). Previous research has not examined thermoregulation in this population under exercise conditions,^{1,2} nor has actual skin temperature (T_{sk}) been measured.

One of the main problems in measuring thermoregulation in this type of population under exercise conditions is that even minimally impaired individuals with MS (patients with an EDSS score of 1 to 4), when asked to exercise at a moderate intensity (eg, 50% to 65% of maximal aerobic capacity—VO₂max), would be able to exercise only at an absolute intensity equivalent to approximately 50 to 60 watts (W).⁶ Based upon maximal exercise power outputs reported for more severely impaired persons (with EDSS scores > 4.5),⁷ moderate exercise would be performed only at 35 to 50 W. These metabolic (thermal) loads are far below an absolute level and duration that would be expected to cause an increase in core temperature sufficient to elicit sweating in the presence of normal sudomotor responses. Therefore, to address this issue, the current study used a full-body, water-circulating garment to assist in imposing a controlled, ambient, thermal load during moderate exercise to simulate a greater workload. At this simulated higher workload, sweat response could then be examined.

The primary purpose of this study was to document thermoregulatory responses of sweating and core temperature during moderate aerobic exercise with an additional thermal load and to compare the response of subjects with MS with that of healthy, non-MS controls. In addition, we examined the relationship of thermoregulatory responses to the level of disability related to the MS.

Methods

Subjects:

Twenty persons who met the criteria for diagnosis of MS and eight healthy, non-MS control subjects participated. The experimental subjects (MS) were full to semi-ambulatory (EDSS scores between 1.0 and 6.5).⁸ Eligibility requirements for the MS group included: 1) a diagnosis of MS, with laboratory and clinical confirmation; 2) the disease currently in remission; 3) ambulatory with minimal use of assistive devices (ie, cane, crutches, walker, wheelchair); and 4) no other chronic health conditions present (ie, type 2 diabetes, coronary artery disease, thyroid or endocrine disorder, pulmonary disease, body mass index > 25% above recommended standards, or non-MS related orthopedic problems). A summary of physical characteristics and neurologic status is presented in Table 1. Eight non-MS control subjects matched for physical characteristics, age, and sex were recruited; their data are presented in Table 2.

Table 1. Summary of physical characteristics, neurologic status, and maximal aerobic performance variables of the experimental (MS) subjects.

Subject	Sex	Age (yr)	Height (cm)	Weight (kg)	EDSS	VO ₂ max (mL/kg/min)	PO _{max} (w)
1	M	50	168.0	78.0	6.0	18.8	50
2	M	38	174.0	77.5	6.0	22.8	75
3	F	35	164.0	66.5	2.5	14.3	90
4	M	78	169.5	76.0	5.0	21.2	85
5	M	55	195.0	98.5	1.0	28.0	187
6	F	51	171.0	62.0	2.0	23.1	100
7	F	51	157.5	76.0	0	23.6	100
8	M	58	182.0	89.0	1.0	32.3	175
9	M	51	176.0	80.5	3.5	35.0	150
10	F	50	166.0	78.0	3.0	19.0	125
11	F	47	155.0	51.0	2.0	26.8	75
12	F	48	166.0	62.0	5.5	24.0	100
13	F	54	163.5	77.0	1.0	23.8	147
14	F	50	163.0	58.0	2.0	32.2	100
15	F	43	157.5	66.5	6.5	20.5	30
16	F	50	165.5	66.5	4.0	25.7	100
17	M	53	185.0	103.0	6.0	17.7	50
18	M	43	181.5	83.0	3.0	20.8	150
19	F	52	157.5	82.5	6.0	11.0	40
20	M	45	177.0	67.0	6.0	29.0	100
Mean	-	50.1	169.7	74.9	3.6	23.5	100
±SD	-	8.6	10.5	12.9	2.1	6.0	44

Table 2. Summary of physical characteristics and maximal aerobic performance variables of the control (non-MS) subjects.

Subject	Sex	Age (yr)	Height (cm)	Weight (kg)	VO ₂ max (mL/kg/min)	PO _{max} (w)
1	M	57	164.5	96.0	33.0	250
2	F	58	160.5	62.5	23.2	125
3	M	59	163.1	69.0	32.9	125
4	F	52	160.0	76.5	26.4	125
5	M	47	185.0	133.0	24.8	225
6	F	24	157.0	52.0	46.1	150
7	F	47	166.2	67.5	26.8	100
8	F	54	156.0	81.2	21.3	100
Mean	-	49.8	164.0	79.7	29.3	150
±SD	-	11.4	9.2	25.2	8.0	57

Preliminary Screening

Neurologic Examination:

All MS patients received a standard neurologic examination to ensure that they met the diagnostic criteria for MS. Based on the outcome of this examination, subjects were rated on the Functional Systems Scale and the EDSS.⁸ A 12-lead resting electrocardiogram was administered and interpreted to provide clearance for patients to perform moderate exercise.

Subjects were also asked to complete a short medical history form containing questions regarding past and current health. Height and weight were also measured. Prior to any exercise, the subjects were asked whether they experienced sweating under any climatic or physical exertion conditions. In addition, notation was made regarding general symptomatology experienced during increased thermal load (ie, internal or external). Informed consent approved by the local Institutional Review Board was obtained prior to all testing.

Maximal Aerobic Power (VO₂peak) Exercise Test:

To generate equivalent workloads for the submaximal sweating protocol, subjects performed a test of maximal aerobic power (VO₂peak) using an upright stationary bicycle (Monark, model 818e). The test began with a three-minute rest period, followed by three submaximal stages (0, 25, and 50 W), each lasting four minutes. Following these three stages, a continuous phase began with exercise resistance increased each minute until volitional fatigue. For most participants, this final stage lasted a maximum of three to four minutes. During testing, metabolic (ie, oxygen uptake [VO₂], carbon dioxide production [VCO₂]) and cardiopulmonary (ie, minute ventilation [VE], systolic and diastolic arterial blood pressure, and heart rate) data were collected continuously. Perceived level of exertion was solicited at the end of the test in three categories: central, peripheral, and integrated. These categories represent anchors for perceived stress related to cardiovascular, local muscle, and overall general stress, respectively.⁹ Test termination was based on the following criteria: 1) volitional fatigue; 2) predetermined cardiopulmonary indicators (appearance of a plateau of oxygen uptake for two consecutive 15-second samples with a concomitant increase in minute ventilation); or 3) the appearance of MS-related symptoms (eg, dizziness, double vision, spasticity). Again, this test was performed to establish an index from which to estimate a moderate level of exercise (eg, 50% VO₂peak) that each subject would be able to maintain for a period of 30 minutes during the sweat response protocol.

Sweat Response During Endurance Cycling:

Subjects exercised continuously at a moderate intensity (approximately 50% of VO₂peak) for a maximum of 30 minutes using the same Monark 818e bicycle ergometer. The test began with a three-minute rest period to establish baseline measurements for all physiological parameters previously described. Based upon the results of the VO₂peak test, a workload that would elicit a metabolic response equal to 50% of each subject's VO₂peak was established and the subject was asked to exercise at that intensity until one of the following criteria was met: 1) volitional fatigue; 2) sweat appearance on all five sites; or 3) 30-minute time limit. Once the test was terminated, the subject remained seated while skin and core temperatures were continuously monitored. Monitoring continued until the T_c reading stabilized.

Thermal Cooling/Heating Garment:

In previous research in this laboratory, it was observed that many individuals with MS have such a low maximum work capacity that exercising at 50% would not provide sufficient internal metabolic load to elicit either an increase in core temperature or raise skin temperature to a level that would stimulate a sudomotor (sweat) response. Even if the external workload were sufficient, it would take approximately 50 to 70 minutes of exercise to increase rectal

temperature even slightly.¹⁰ To reduce the time and amount of effort required by the subject, a controllable external heat load was added by using a tubing garment similar to one previously used for cooling patients.^{7,10} Before beginning the endurance test, the subject donned a two-piece full-body garment (Figure 1) consisting of micro-tubing (internal diameter, 3/32 inch) imbedded within stretchable lycra material, with 2 centimeters separating each line of tubing. This distance changed based upon the size of the subject and the amount of stretch imposed on the garment. The limbs and torso were wrapped with Ace[®] bandage to improve the contact of the suit tubing with the surface of the skin. It should be noted that the head cover depicted in this illustration was not used in the present study. To accommodate our need to access sweating sites, small openings were constructed in the garment to expose the sites for monitoring sweating. Initially during the rest period, 27°C water was circulated through the suit at a flow rate of 1.5 liters per minute. Once the exercise protocol began, the water temperature was increased 2°C every three minutes to a maximum of 41°C. The exercise test was terminated for any of the following reasons: 1) volitional fatigue; 2) appearance of MS-related symptoms; or 3) the appearance of sweating at all five sites. Using the suit in this manner “clamps” skin temperature to allow for controlled measurement of heat transfer.¹¹⁻¹⁴ Once the test was terminated, 12°C water was immediately circulated through the garment to reverse the effect of the heating.



Figure 1. Full-body water garment (Carleton Technologies, Inc., Tampa, Florida).

Core and Skin Temperature and Sweat Measurement:

Core temperature was measured using a rectal thermistor (YSI 400 series, Yellow Springs, Ohio) inserted 10 centimeters beyond the anal sphincter and secured with adhesive tape. Skin temperatures were monitored with small surface thermistor probes, all on the right side of the body at the following nine sites: forehead, anterior chest, abdomen, upper back, lower back, forearm, anterior thigh, posterior thigh, and calf. An area-weighted skin temperature was calculated from these sites and recorded every minute along with the individual skin temperatures. The thermistor sensors were mounted in plastic rings and worn beneath the tubing garment. This isolated the sensor from the cooling tubes and allowed air circulation over the point of contact.

Sweating was detected by the starch-iodine method.^{11,15,16} This commonly used method relies upon the combination of iodine and wet starch as an indication of the presence of sweat. A small area of the skin adjacent to the site of skin temperature measurement on the forehead, upper arm, upper anterior chest, abdomen, and anterior femur regions was swabbed with tincture of iodine. As long as the skin was dry (ie, free of sweat), applying a piece of starch paper to the area would elicit no chemical reaction on the paper. The onset of sweating appears as a change in color (blue or brown) at the point of contact with the surface of the skin adjacent to active sweat glands. This method was not intended to quantify sweat rate, but merely to indicate the onset of sweating. The usual areas observed for sweat onset are the forehead and chest. These two sites were observed, in addition to the arm, lower abdomen, and thigh, since neurologic damage from MS could affect central pathways for sweating in many areas.¹⁷

Heat Storage:

The amount of heat stored during exercise was derived from the temperature of the water entering the tubing suit (T_{wi}), mean T_{sk} , external workload, and metabolic heat production. This method was based on previously published calibration data.¹⁸⁻²⁰ In the current experiments, during the three minutes that preceded exercise, there was heat loss to the suit and to the room that exceeded metabolic heat production. T_{wi} was lower than T_{sk} by $5 (\pm 1)^\circ\text{C}$. Heat loss continued until this gradient was reduced to 3°C , when losses equaled gains, and metabolic heat no longer escaped and was stored. The gradient from T_{wi} to mean T_{sk} diminished to zero after about 10 minutes of exercise. The cumulative heat stored until this time was near zero. Thereafter, metabolic heat, plus external heating, was fully stored. As T_{wi} rose above mean skin temperature, the heat stored was the cumulative sum of heat added by the garment, plus the metabolic heat, less external work. For each degree that T_{wi} exceeded mean skin temperature, heat transfer into the subject was 35 W. A typical recording of T_{wi} , mean T_{sk} , and heat storage for a subject during the sweating protocol is illustrated in Figure 2.

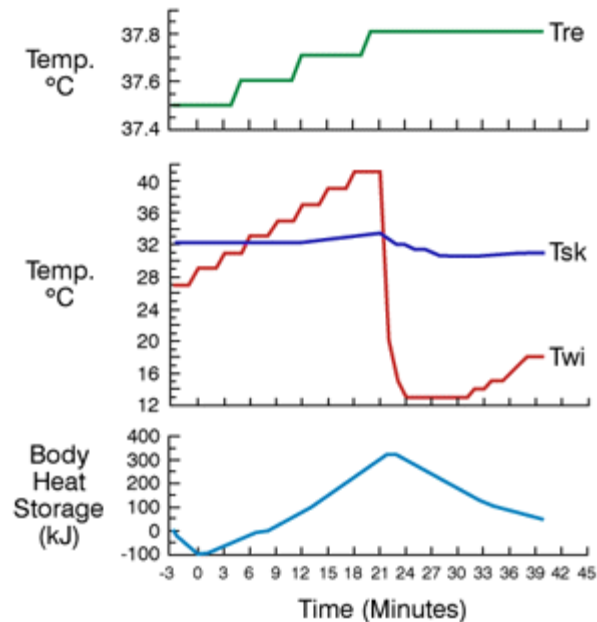


Figure 2. Typical data from an MS subject. The center panel shows how water temperature entering the suit (T_{wi}) changes from a comfortable level (27°C) to heating, until detection of sweat onset in all five body sites at 21 minutes. This is followed by rapid cooling. The same panel shows the course of mean skin temperature (T_{sk}). Rectal temperature (T_{re}) is shown in the upper panel and cumulative body heat storage in the lower panel.

Statistical Procedures:

The nature of this study is descriptive research. Measures of central tendency (mean, median, mode) and variance (standard deviation) were calculated for all variables. An independent t-test comparing the experimental (MS) and control groups was used for all dependent variables, $P < .05$ maintained for the Type I error rate. Correlation analysis was also used to examine the relationship among several dependent variables.

Results

The results of the test of maximal aerobic power (PO_{max}) used to derive a submaximal workload for the sweating protocol are presented for the experimental and control groups in Tables 1 and 2, respectively. PO_{max} for the MS subjects ranged from a low of 30 W to a high of 187 W. Seventy percent of the MS subjects were capable only of a $PO_{max} < 100$ W. Based on these performance criteria, submaximal exercise at a moderate level (ie, 50% to 65% PO_{max}) would be at an insufficient external work level to promote a metabolic drive under which to examine the sweat response.^{6,7} These data support earlier published findings.^{6,7}

One of the key questions that directed this research was, "Given a sufficient metabolic (ie, thermal) load, would the sweat response in persons with MS occur in a normal manner—that is, at or before a maximum T_{sk} of 35°C ?" The results of the endurance cycling test revealed that 50% of subjects with MS exhibited normal sweating (MS-NS) in all sites within the 30-minute maximum, and 50% showed abnormal sweat (MS-AS) patterns, with sweating occurring in at least four, but not all five sites. These latter subjects, despite relatively high skin temperatures ($33.9 \pm 1.10\text{C}$), failed to show sweating at the fifth site before exercise was terminated. All

control subjects exhibited normal sweat response within the allotted time, and the heat load for the control group was significantly lower than that for the MS-AS group ($P < .05$). The mean length of time required by the MS-NS group to exhibit sweating at all five sites was 13.8 minutes (range eight to 30 minutes), with 90% of the group sweating in less than 20 minutes. These results were similar to those for the non-MS control group, with a mean (\pm SD) time of 14.3 (\pm 6.0) minutes. Sixty-two percent of the control group achieved sweating at all five sites in 12 minutes or less (range, six to 12 minutes), with the remainder achieving the criterion within 21 minutes.

Figure 3 illustrates the total heat load (kJ/kg) for the MS-NS, MS-AS, and control groups during the endurance test. The total heat load for the group that did not exhibit normal sweating was significantly higher than that for the 10 MS subjects with normal sweating, as well as that for the control group ($P < .05$). The higher total heat load experienced by the MS-AS group was a direct reflection of heat being stored by these subjects and a "blunting" of the sweat response. Surprisingly, this higher heat storage did not result in a significantly higher mean T_c or T_{sk} for the MS-AS group. In addition, the magnitude of the "blunting" appears to vary greatly among individuals. Two individuals in the MS-AS group had a fairly high mean T_{sk} ($> 35.0^\circ\text{C}$), with three of the four sites measuring higher than 35°C . This would be the upper limit at which sweating would be expected to occur.

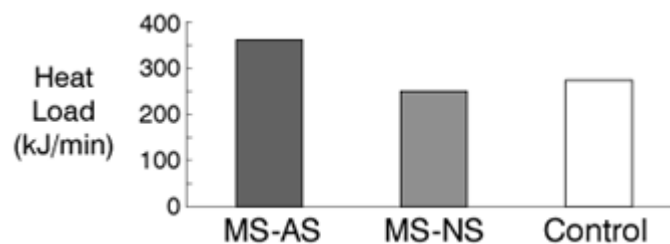


Figure 3. Total heat load (kJ/kg) during the sweating protocol for MS subjects with a normal sweat response (MS-NS, $n = 10$), an abnormal sweat response (MS-AS, $n = 10$), and non-MS subjects (controls, $n = 8$) during endurance cycling. The MS-AS is significantly different from the other two groups ($P < .05$).

An analysis of the data in terms of disability level found a nonsignificant difference in the mean EDSS rating between the MS-NS and MS-AS groups, with 50% of the abnormal sweat response group having a rating of greater than or equal to 5.0 and only 30% of the subjects in the normal sweat response group having EDSS rating greater than or equal to 5.0. The result of a Spearman Rho correlation found only a modest relationship between EDSS and the onset of a sweat response ($+ 0.44$, $P < .05$), indicating that the EDSS scale is probably incapable of predicting whether an individual would have a normal or abnormal sweat response. This was not surprising since the EDSS rating is heavily weighted on ambulatory ability, which would be unrelated to the autonomic nervous system function of sweating.

Discussion

The nature of descriptive research is to observe a phenomenon with the purpose of generating further research of either a descriptive or "experimental" design. Demonstration of the "sweat response" phenomenon in persons with MS under controlled laboratory conditions provides a foundation for future inquiry that should incorporate a larger, more diverse sample. It must be noted that, based upon the small sample of this study, the generalizability of these findings is limited.

Nearly 30 years ago, Noronha and colleagues¹ reported that 58% of their MS sample demonstrated abnormal sweating. These findings are strikingly similar to the 50% incidence rate in the current study. The earlier study also suggested that people with an abnormal sweat response were probably more disabled; however, the authors offered no statistical support for their conclusion. In the present study, there were more individuals with a higher EDSS in the group exhibiting abnormal sweating; however, results of correlation analysis failed to support a relationship between disability level (ie, EDSS) and the presence of abnormal sweating.

It is interesting to note that 70% of the individuals with MS who participated in the study were able to correctly predict whether or not they would sweat. The remaining 30% were incorrect in their prediction. These findings suggest that most people with MS would be correct in predicting whether they would sweat if conditions warranted. However, there still remains the question as to why the remaining 30% have an apparent "lack of understanding" of their own physiological responses. One hypothesis is that many of these individuals avoid situations under which the sweat response would be elicited (eg, staying indoors during high humidity and temperature or avoiding exercise or strenuous physical activity). Interestingly, the subject from the control group with the lowest fitness level also predicted she would not sweat. An in-depth interview with this subject revealed a conscious and consistent avoidance of physical activities and climatic conditions that would elicit a sweat response. This behavior is similar to that often expressed by many of the MS subjects.

Noronha et al¹ suggested the incidence of abnormal sweat response to be higher in patients with more severe and advanced MS, which appears to be somewhat supported by the current findings. Noronha's "normal sweat response" group had a mean disability rating of 2.4 while the "abnormal sweat response" group had a mean disability rating of 3.8. In the current study the mean (\pm SD) EDSS for the normal sweat group was 3.0 (\pm 2), and that for the abnormal sweat group was 4.2 (\pm 2).

Since the onset of sweating can be altered through training and/or an individual's aerobic fitness level, we examined the relationship between fitness level (VO₂max mL/kg/min) with time to onset of sweating during the endurance test. Although the range of scores showed a fair amount of variability, the correlation between fitness level and onset of sweat was low (-0.35 , $P > .05$). Categorization of fitness level²¹ for both groups is presented in Figure 4. If we remove the one outlier in the control group, it appears that the fitness levels of the majority of the MS group are similar to those of the controls. However, a more in-depth analysis of fitness level as it relates to sweat response is illustrated in Figure 5. This analysis revealed that only those subjects with abnormal sweating were significantly less fit ($P < .05$) than those in the control group. Part of the reason for these findings could be related to the greater percentage of subjects in the abnormal sweating group with a higher EDSS rating. Previous research has shown that VO₂peak lowers as EDSS increases.²² Certainly, future research as to whether exercise training could enhance and/or normalize sweat response in our experimental population would be of great interest if it could improve exercise tolerance.

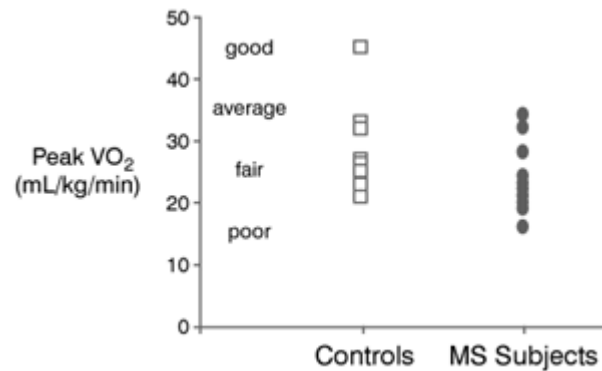


Figure 4. Fitness categories of control and MS subjects based upon performance on the test of maximal aerobic power.

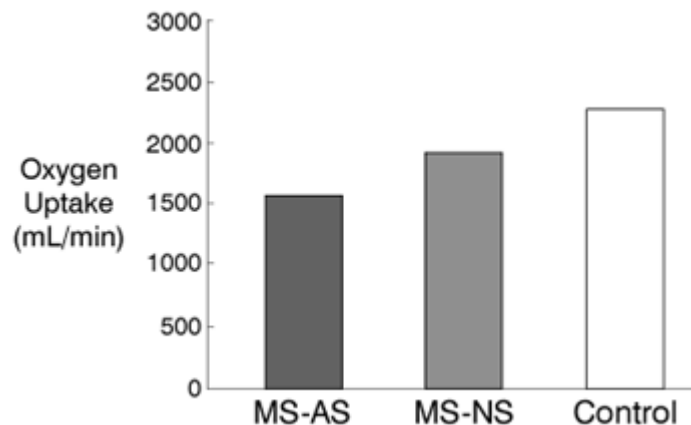


Figure 5. Comparison of VO₂peak among controls ($n = 8$), MS subjects with normal sweat response (MS-NS, $n = 10$), and MS subjects with abnormal sweat response (MS-AS, $n = 10$) during an incremental bicycle exercise. The control group was statistically different from the MS-AS group only ($P = < .05$).

A final, yet important question addressed by this research concerns the relationship between sweat response, core temperature, and perceived level of stress in persons with MS. Examination of the relationship between the overall rating of perceived level of exertion showed a strong positive relationship with T_c ($r = +0.86$). This means that a subject's perception of the overall level of stress during the exercise protocol increased proportional to his or her increase in core temperature. The peripheral rating of perceived exertion, which cues the person to consider stress related to the musculature that is performing the exercise, showed moderate relationships with mean skin temperature ($r = +0.64$) and the magnitude of the work intensity ($r = +0.66$). This indicates that the "peripheral" stress anchor is probably driven by local mechanoreceptors as well as by peripheral sensory and temperature receptors. All other relationships among any of the dependent variables examined fell below $r = \pm 0.20$.

It has often been reported that persons with MS should avoid excessive heat generated through physical exertion or from outside climatic conditions for fear of eliciting an MS symptom. In the current investigation, participants were subjected to a large thermal load (as great as 0.8°C in

Tc and skin temperature greater than 35°C for some individuals), greater than what most could generate simply through physical activity or what might be absorbed from environmental surroundings. Even so, only one person with MS reported the appearance of an MS symptom during or immediately following the endurance exercise protocol. The single symptom experienced by one subject was of an optic nature (cobweb-like fuzziness in vision), which also occurred whenever this person felt hot due to other reasons (eg, taking a hot shower). Similar symptoms of an optic nature have been previously reported as being common.

Current guidelines for prescribing exercise to persons with MS published by the American College of Sports Medicine²³ indicate that some individuals may have an abnormal sweat response. The current study supports this statement. As a result, even for moderate-intensity aerobic exercise, room temperature should be kept neutral (21° to 24°C). Fans may also be used for added heat dissipation when normal sweating is not present.

Based upon the percentages from the present study, we might anticipate that 50% of any given sample of subjects might have a problem with normal sweating. Furthermore, if individuals with MS indicate that they do not sweat, there is a 70% likelihood that they are correct in their observation. As such, when counseling this type of individual regarding participation in physical activity, leisure pursuits, exercise, and employment options, strategies for maintaining a cool environment should be discussed. These might include the following:

- Participating in outdoor activities early in the morning or later in the day, substituting indoor activities on days when the heat index is in the unhealthy zone
- Maintaining hydration during all physical activities
- Working and exercising in an air-conditioned atmosphere or one where fans are available
- Limiting activity to short intervals spaced throughout the day and applying cool compresses to the skin when feeling hot (eg, wrapping a wet scarf around the neck or running cold water on the wrists)
- Using caution when applying ice directly to the surface of the skin (this will cause peripheral vasoconstriction and counteract the reflex vasodilation by the body to dissipate metabolic heat)
- Pre-exercise cooling can significantly enhance performance and reduce subjective feelings of fatigue.²⁴ It has been recommended that immersion of the lower extremities into a tepid bath to which cool water has been added for 20 to 30 minutes prior to exercise can help prevent an increase in core temperature throughout a 40-minute bout and subsequent recovery period.²⁴

Conclusion

From these pilot data we can conclude the following:

- Many persons with MS in this study were incapable of sustaining a high enough work intensity for a sufficient length of time to elicit a large enough metabolic (internal thermal) load to increase core temperature to any degree of clinical significance. This conclusion is based upon the results of the VO₂peak test, which revealed maximal power output for all MS subjects to be quite low. Since metabolic load is directly related to the absolute power output of the exercise being performed, it is safe to conclude that a moderate intensity of exercise (50% to 65% VO₂max) could not be sustained long enough to incur a sufficiently large internal thermal load to cause an increase in core temperature. This would be true even in the complete absence of any type of thermoregulatory response to dissipate the additional metabolic heat.

- The thermal load generated during moderate physical activity, when compounded by an external thermal load such as that provided by the water garment, is not sufficient to elicit MS-related symptoms in most persons in our sample.
- Most persons with MS who believed they would not sweat were correct in their assumption. This phenomenon may result from a neurologic deficit or, in many cases, from an avoidance of exertion, especially in heat.
- Abnormal sweat response may occur in as much as 50% of the MS population who rate below 6.5 on the EDSS.

Acknowledgment

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References

1. Noronha MJ, Vas CJ, Aziz H. Autonomic dysfunction (sweating responses) in multiple sclerosis. *J Neurol Neurosurg Psychiatry*. 1968;31:19-22.
2. Vas CJ. Sexual impotence and some autonomic disturbances in men with multiple sclerosis. *Acta Neurol Scand*. 1969;45:166-182.
3. Ryan M, Pompei F. Core body temperature via the tympanic membrane. A practical method using the Exergen OTO-TEMP 3000 Infrared Tympanic Temperature Scanner. Exergen Corporation. 1990.
4. Nash HL. Treating thermal injury: disagreement heats up. *Phys Sports Med*. 1985;13:134-144.
5. Nadel ER, Horvath SM. Comparison of tympanic membrane and deep body temperatures in men. *Life Sci I*. 1970;9:869-975.
6. Ponichtera-Mulcare JA, Glaser RM, Mathews T, Camaione DN. Maximal aerobic exercise in persons with multiple sclerosis. *Clin Kinesiol*. 1992;46:12-21.
7. Mulcare JA, Webb P, Mathews T, et al. The effect of body cooling on the aerobic endurance of persons with multiple sclerosis following a 3-month aerobic training program. *Med Sci Sport Exerc*. 1997;29(5)suppl:S83.
8. Kurtzke JF. Rating neurological impairment in multiple sclerosis: an expanded disability status scale (EDSS). *Neurology*. 1983;33:1444-1452.
9. Noble BJ, Borg GA, Jacobs I, et al. A category-ratio perceived exertion scale: relationship to blood and muscle lactates and heart rate. *Med Sci Sports Exerc*. 1983;15:523-528.
10. Mulcare JA, Webb P, Barrett PJ, Mathews T. Exercise endurance during body cooling in patients with multiple sclerosis. *Multiple Sclerosis*. 1996;2:2.
11. Webb P, Garlington LN, Schwarz JJ. Insensible weight loss at high skin temperatures. *J Appl Physiol*. 1957;11:41-44.

12. Nunnely SA. Water cooled garments: a review. *Space Life Sci.* 1970;2:335-360.
13. Webb P, Troutman SJ Jr, Annis JF. Automatic cooling in water cooled space suits. *Aerosp Med.* 1970;41:269-277.
14. Troutman S, Annis JF, Webb P. Controlling human heat content – method and application. *NAECON.* 1977:259-266.
15. Leithead CS, Lind AR. Heat Stress and Heat Disorders. Philadelphia: FA Davis; 1964:192.
16. Webb P. Temperature of skin, subcutaneous tissue, muscle and core in resting men in cold, comfortable and hot conditions. *Eur J Appl Physiol.* 1992;64:471-476.
17. Kaeser HE, Lambert EH. Nerve function studies in experimental polyneuritis. *Electroencephalo Clin Neurophysiol.* 1962;22(suppl):29-35.
18. Hambraeus L, Sjodin A, Webb P, et al. A suit calorimeter for energy balance studies on humans during heavy exercise. *Eur J Appl Physiol.* 1994;68:68-73.
19. Webb P. Human Calorimeters. New York, NY: Praeger; 1985.
20. Webb P, Annis JF, Troutman SJ Jr. Human calorimetry with a water-cooled garment. *J Appl Physiol.* 1972;32:412-418.
21. Nieman DC. Fitness and Sports Medicine: An Introduction. Palo Alto, Calif: Bull Publishing Company; 1990:504.
22. Ponichtera-Mulcare JA, Mathews T, Barrett PJ, Gupta SC. Change in aerobic fitness of patients with multiple sclerosis during a 6-month training program. *Sports Med Training and Rehab.* 1997;7:265-272.
23. Mulcare JA. Multiple sclerosis. In: Durstine JL, ed. ACSM's Exercise Management for Persons with Chronic Diseases and Disabilities. Champaign, Ill: *Human Kinetics*; 1997:189-193.
24. Petajan JH, White AT. Recommendations for physical activity in patients with multiple sclerosis. *Sport Med.* 1999;27(3):179-191.

Book Review

The Editors of the *International Journal of MS Care* are constantly evaluating reference books that can be of assistance to MS care providers around the world. If you have questions on any of the books reviewed here, or have any suggestions on additional books to be reviewed, please contact us at IJMSC@partmedcomm.com.

Wheelchair Selection and Configuration

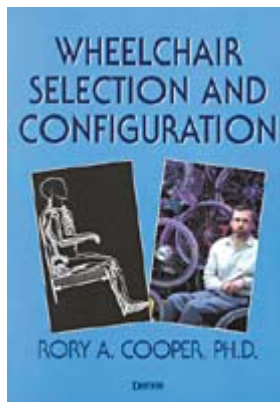
Rory A. Cooper, PhD

424 pp. New York

Demos Medical Publishing, Inc; 1998

\$34.95 US

ISBN 1-888799-18-8



This book provides a comprehensive description of the wheelchair from historical, sociological, psychological, functional, mechanical, and medical perspectives. The target audience is students and professionals in the fields of occupational therapy, physical therapy, rehabilitation sciences, and rehabilitation engineering. Because of the inclusion of content on US legislation and international standards for wheelchairs, as well as wheelchair repair and maintenance, this book may also be a resource for wheelchair users to better assess quality and maintain their wheelchairs.

The first chapter describes the meaning, symbolism, societal constructs, and regulations related to the wheelchair. It also touches on the social development of a wheelchair user at school, work, and recreation. The chapter is sensitive to the emotional, psychological, and social adjustments required of the user. For example, a child born with a disability requires no transition, while an adult with a sudden onset of disability experiences a dramatic adjustment to his or her sense of autonomy and is faced with the abrupt challenge of learning new skills. For an adult with a gradual onset of disability, such as patients with multiple sclerosis (MS) who require wheeled mobility, the wheelchair may symbolize acquiescence to the disease. The price of obtaining increased mobility and function may be exacted at great emotional cost. Finally, the importance of the environment and cognitive impairments of the individual are noted, though their implications are minimally discussed.

The contents of chapters 2 through 5 are in-depth discussions of physical measurements, as well as the engineering, biomechanic, ergonomic, and electronic fundamentals of wheelchairs. These chapters provide detailed commentary augmented by diagrams and photographs. The charts included in these sections are clinically very useful. While there is little doubt that chapters 3 and 5, as well as parts of chapter 4, are for the intellectual appetite of the engineer and those studying for the Rehabilitation Engineering and Assistive Technology Society of North America (RESNA) credentialing exams, the principles of physics and mathematics are presented in an understandable way for the therapist. Likewise, issues related to human factors, such as the diversity of wheelchair users and uses, are relayed as tangible considerations for the engineer. It should be noted, however, that the example at the end of

chapter 2 describing someone with MS is relatively atypical. The first choice of mobility for the majority of those with MS is a scooter. A manual wheelchair is infrequently chosen due to the physical energy required to operate it and the presence of fatigue, upper extremity weakness, sensory anomalies, and movement disorders.

There is an excellent discussion in chapter 6 about wheelchair standards and testing. Compliance with the 22 standards established by the International Standards Organization is voluntary, so the testing of wheelchairs by manufacturers is described in detail. The various tests carried out for strength, fatigue, range, and safety are indicated, and the three classes of "wheelchair failures" are reviewed. This chapter also distinguishes "rehabilitation" from "depot" manual wheelchairs and provides a pointed discussion about the difference in longevity between these and its subsequent economic implications.

Chapters 7 through 10 give thorough treatment to the types of wheelchairs. Each chapter is dedicated to one of the following: manual, power, specialized, or sport (recreational) wheelchairs. For each type of wheelchair, the subtypes are presented, along with descriptions of numerous components and accessories. For practitioners working with people with MS, there are a couple of pages in chapter 8 that directly address scooters.

In chapters 11 and 12, Dr. Cooper turns his attention to accessories for wheelchairs. Chapter 11 discusses issues related to cushions, including measurement, properties, stresses on tissue, and types of cushions. Seat bases, seat base-positioning equipment, and computerized shape-measurement are also addressed. Postural support systems are the subject of chapter 12. The variations and customized options available for complex seating needs are clearly described. The issues addressed in this chapter may also be relevant for some persons in advanced stages of MS.

Theoretical and clinical perspectives on assessment and intervention are outlined in chapter 13. Philosophic approaches discussed include the human activity assistive technology (HAAT) model, the "allocation of functions" approach, the decision-making process for implementing continuous quality improvement, and a framework for disability: an individual and societal relationship. Clinical areas addressed are interviewing, observation, measuring performance, team assessment, computerized assessment, service delivery systems, funding, and documentation.

Chapter 14, the final chapter, deals with the important topic of wheelchair adjustment and maintenance. Topics include adjusting wheel camber and alignment; center of gravity; and seat, back, and leg-rest positions. Also discussed are user maintenance: cleaning, scheduled inspections, repairs, and replacements. There is also a section on manufacturing and modification.

Wheelchair Selection and Configuration serves as an excellent resource for those learning about the "ins and outs" of wheelchairs, or as a refresher for those not regularly involved in recommending wheeled mobility. This book would be a superb adjunct to print or electronic materials about products (wheelchairs and accessories) currently available.

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