SPASTICITY: CORRELATE OF FES MOTOR PERFORMANCE IN SPINAL INJURY

Campbell,J., Waters,R.L., Meadows,P.M., Nakai,R., Carter,C.L., Grek,A.E., Miller,L., Gillim,R.L.

Rancho Rehab Engineering Program, Rancho Los Amigos Medical Center
Downey, California, USA
California State University, Long Beach
Long Beach, California, USA

ABSTRACT

Spasticity can interfere with voluntary or electrically stimulated movement. Muscle force generation may be augmented by a spastic response or reduced by involuntary contraction of antagonistic muscles. Compensation for mechanical and reflexive contributions to stretch, passive joint motion (PJM), would permit more accurate tracking of change in muscle performance as a result of volitional strengthening and/or electrical stimulation. The purpose of this paper is to describe a system for the documentation of spasticity and to present preliminary results of passive knee motion in normal (N) and spinal cord injured (SCI) subjects. Twenty N subjects and 8 SCI (15 knees) participated. PJM was controlled by an isokinetic system (LidoActive) which was modified to provide signals to an IBM PC/AT computer for angular position, velocity and moment. Custom software was developed for collection of peak moments (Nm) and resistance work (Nm-deg) during PJM. Surface EMG electrodes recorded EMG activity in the thigh. Gravity compensation at 5 deg/sec was performed prior to testing (10 joint oscillations or cycles) at 60, 90, 120, 180 and 230 deg/sec. Moment versus position plots were assessed to determine the arc of knee motion and the number of cycles to be analyzed. Selection of data points for subsequent analyses excluded the moment arm acceleration to the target velocity in the initial cycles as well as within each cycle at the end of the 0-90 degree arc. Repeated measures ANOVA revealed a velocity dependent relationship in both normal and SCI patients ($p<0.001$). Differences between SCI patients and normal subjects were best detected at 230deg/sec. Resistance work (Nm-deg) correlated with velocity. The results of this study have immediate clinical application in the day-to-day assessment of motor performance of SCI, stroke, head trauma and cerebral palsy patients.

KEY WORDS: Spasticity, Electrical Stimulation, Spinal Cord Injury, Skeletal Muscle, Passive Joint Motion

INTRODUCTION

Involuntary contraction of paretic or paralyzed muscles is a common problem for many patients with central nervous system disorders. Spasticity, or hyperexcitability of the muscle stretch reflex, may augment or interfere with movement as its severity fluctuates throughout the day. Determination of the relative merits of therapies designed to reduce spasticity or to strengthen muscle is confounded by the problem of objective documentation.

One focus of research designed to evaluate the effects of electrical stimulation on spasticity has been an effort to document short term versus prolonged reduction of spasticity in a variety of patient groups including spinal cord injury, stroke, head trauma and Parkinsonism (1-20). The influence of spasticity on electrically stimulated muscle performance has not been adequately described. If electrical stimulation is to successfully simulate normal movements, as in standing and walking, for the paretic or paralyzed individual, additional issues must be addressed. Electrically stimulated muscle performance must be optimized to meet the demands of repetitive, functional activity at 40 to 60 percent of maximal, normal muscle capability. As a part of this optimization process, FES exercise protocols designed to increase both force generation and fatigue resistance will need to be studied. Spasticity of the muscle under study, or its antagonist, may markedly alter day-to-day measurement of change in muscle performance.

The purpose of this paper is to describe a system for the documentation of spasticity, by resistance to PJM (RPJM), and to present preliminary findings in normal subjects and SCI patients.

BACKGROUND

Spasticity is clinically observed as an increased responsiveness of skeletal muscle to stretch. Involuntary muscle contractions, or even sustained oscillating joint motion (clonus), may interfere with safe movement in the patient with a central nervous system disorder. For some patients, who can call upon their heightened stretch reflexes to augment force production in weak or mechanically disadvantaged muscles, spasticity is useful. For many patients, however, spasticity of a muscle, or muscle group, results in a functional weakness of antagonistic muscles (15,21-22). In walking, functional weakness may interfere with the critical events that must occur in each sub-phase of gait. Spasticity of even one of the two-joint hamstring muscles, for example, may limit or prevent hip flexion for limb advancement in the swing phase of gait (22-23).

Spasticity may be more objectively defined as a velocity sensitive resistance to passive movement (24-28). There is disagreement among investigators about the optimal measurement technique(s) to quantify spasticity. Tendon tap responses, demonstration of clonus and the Ashworth scale have been used in the clinic (29). Quantitative mechanical assessments of spasticity have included the pendulum drop test (3-4,9-10,14-15,30-32), sinusoidal joint motion (8,12-13,16,24,33-38) and isokinetic movement of the limb segment (39-41). The contribution of the passive mechanical properties of musculotendinous and periarticular connective tissues versus reflexive muscle contraction to RPJM requires intramuscular electromyographic recordings. Interpretation of mechanical test measurements also mandates a compensation for the effect of gravity on the limb, the inertial effects of the limb as limb motion changes direction, and the inertial and frictional effects of the measurement system. It is also essential to consider the range of joint motion tested in relation to the patient's functionally required arc of motion, and the range of test velocities required to not only elicit spasticity, if it is present, but to approximate the velocities normally seen by the joint in daily function (23,34-35).

Resistance to passive joint motion, in the absence of electromyographic activity of the stretched muscles, has been documented during slow joint motion in normal individuals (16,34,42). The differences in type of motion (sinusoidal versus linear or constant velocity with acceleration and deceleration components) and the relative arc of joint motion in various upper and lower limb joints makes comparison of data and determination of the threshold for muscle stretch reflex difficult. It has been suggested that linear stretching movements are probably of greater use in quantitative studies of reflexive response to PJM and that sinusoidal stretching movements may be preferable to demonstrate phase relationships of the reflex contraction to the degree of muscle stretch (34).

Increased RPJM has been demonstrated in a variety of patient groups including stroke, cerebral palsy and spinal cord injury (1-20). Contributions to increased RPJM would be expected from both hyperreflexia and greater stiffness of connective tissues. Reflexive EMG was found in response to stretch of the spastic muscle at its maximum length by some authors (24), while other investigators have described differences in length/spasticity relationships between upper and lower limb muscles as well as among muscles in the same extremity (37,43) pointing to a need for a clinically relevant arc of motion during PJM test procedures. The velocity threshold for detection of spasticity has been reported to range from 5 to 193 deg/sec (34). Knutsson, et al, have indicated that the velocity threshold for spasticity during voluntary movement is quite variable and therefore testing should simulate normal joint velocities (40).

Testimonial, case study and objective reports on the effectiveness of electrical stimulation in reducing spasticity have appeared in the literature since the 1800's (6). Although earlier studies reflected subjective measurement techniques, more recent studies have attempted to quantify joint and/or muscle response to joint perturbation (1,3,8,12-15,18,44-45). Electrical stimulation has been shown to temporarily reduce spasticity in hemiplegic, spinal injured and cerebral palsied patients, and trends toward a 24-hour per day effect of chronic electrical stimulation on spasticity have been reported (10,13,46). Once again, protocols for stimulation as well as for evaluation have varied and it is difficult to synthesize the available information. Subjects, or patients, have been diverse in the genesis of their motor paresis or paralysis and the extent of residual sensation. Activation of reciprocal inhibition mechanisms or spinal inhibitory interneurons (Renshaw cells) in response to electrical stimulation may differ in patients with complete versus partial paralysis (26,40,47-50).

Assessment of electrically stimulated muscle performance is confounded by day-to-day variability in spasticity, as well as by increased stiffness of periarticular and intramuscular connective tissues in the neuromuscular patient (15,21-23). In order to accurately track changes in muscle performance (peak moments and work performed) as a result of an FES training protocol, it is necessary to sort out changes due to modification of connective tissues as well as spasticity. When muscle performance is controlled by electrical stimulation, as in FES systems to provide functional movement for the paralyzed individual, day-to-day changes in the recruitment properties secondary to fluctuations in spasticity will need to be compensated in the stimulation protocol.

METHODS

SUBJECTS: Twenty healthy, normal subjects between the ages of 21 and 32 years were studied and compared to 8 SCI patients (15 knees in 3 quadriplegic and 5 paraplegic). Both normal and SCI subjects demonstrated full knee range of motion and were free of any history of musculoskeletal or neurological disorder. SCI subjects retained no volitional control of knee muscles.

INSTRUMENTATION: Passive joint motion (PJM) was controlled by an isokinetic multi-joint system (LidoActive, Loredan Biomedical, Davis, CA 95617). The Lido controller was modified to provide analog signals to an IBM PC/AT computer for angular position, velocity, and torque or moment and limb cuff radial displacement. Raw voltages were sampled at 100 Hz each by a Data Translation DT-2821 analog-to-digital converter board controlled by the IBM PC/AT computer under custom software modelled after Carter (39). Prior to data acquisition, calibration and then gravity compensation (5 deg/sec) procedures were performed to eliminate the effect of gravity on limb and mechanical arm components. Data acquisition and management were carried out by the IBM.

Silver disc electrodes (9mm) placed over the quadriceps recorded electromyographic signals at 50μV/div and 10 msec/div (Cadwell Laboratories, Model 5200A, Kennewick, WA 99336) during PJM.

PROCEDURE: Prior to data collection, calibration of angular moment, velocity and position was performed and a gravity compensation trial was completed. Normal subjects were instructed to extend and flex the knee to demonstrate the electromyographic (EMG) activity recorded by the electrodes placed over the quadriceps. Subjects were instructed to relax and to avoid producing EMG activity, if possible. Ten joint oscillations (0-90 degrees of knee flexion with a return to full extension) were performed at each test velocity (60, 90, 120, 180 and 230 deg/sec). Moment and position were recorded for an additional 30 seconds after the cessation of the PJM.

DATA ANALYSIS: RPJM was reported as peak moment and work (the area within the moment versus position plot, determined by a simple trapezoidal rule for each sampling instant) for every cycle as well as for each direction of joint motion (i.e., knee flexion or extension). Compensated data were software filtered using a low-pass Blackman window with cutoff frequencies of 30 and 15 Hz for position and moment respectively. The cutoff frequencies were chosen from Fast Fourier Transform plots of moment and position data to ensure high data integrity. The full range of joint motion (0-90 deg) was analyzed and compared to mid-range values (i.e., 15-75 deg) in order to avoid the inertial effects observed at the end ranges as the Lido moment arm reversed direction and accelerated to constant velocity (Fig. 1). The position ranges of the inertial effects, which were dependent upon velocity, were determined by running the system empty and with an inert load to simulate a human limb. Moment and work values were corrected for the values measured when the system was run empty (Fig. 1). Cycles one and two were compared to other cycles, and cycles three through eight were averaged for comparison across velocities.

The BMDP statistical package was used for all analyses. Repeated measures analysis of variance was employed to identify differences in moment and work due to velocity of joint motion. Paired-t tests with a Bonferonni adjustment were used to locate the differences. Analysis of variance was used to identify differences between SCI patients and normal subjects. Correlation and regression analyses demonstrated the velocity-work relationship in normal versus SCI individuals.

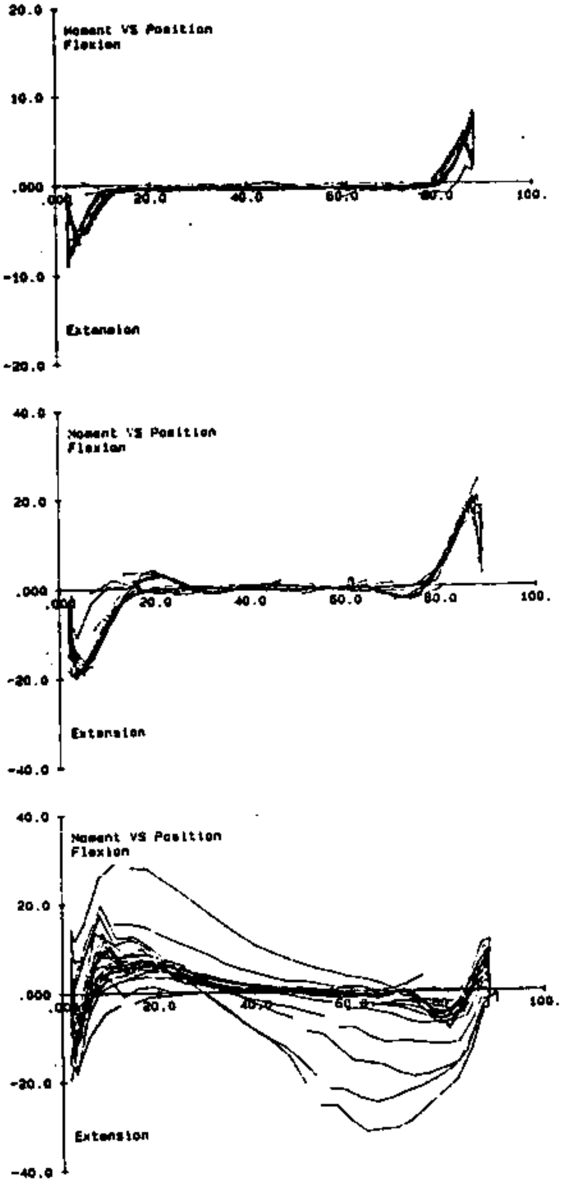

Figure 1. RPJM (Nm) versus knee position; No subject; Normal subject; and SCI subject. (60 Deg/Sec, Cycles 1-10, 0-90 Degrees arc.)

RESULTS

A velocity dependent RPJM was observed in normal and in SCI subjects ($p<0.001$) (Fig. 2). In normal subjects, the total work (Nm-deg), as well as the work during each direction of knee movement (through a 15-75 degree arc), increased from 60 to 230 deg/sec ($p<0.001$). Significant differences in work between pairs of velocities were most evident when 60 deg/sec was compared to 180 deg/sec ($p<0.01$) and to 230 deg/sec ($p<0.001$). RPJM, reported as peak moment and work, was similar at 60 and 90 as well as at 90 and 120 deg/sec. The use of peak moments, rather than work, to document RPJM significantly reduced the ability to predict resistance from velocity in normal subjects ($r=0.80$ for work and velocity versus $r=0.56$ for peak moment and velocity). Electromyographic activity was not evident during PJM in normal subjects at any of the test velocities. Two subjects who demonstrated minimal EMG during the 60 deg/sec trial were instructed to relax and the EMG was silent on repeated testing.

The increase in RPJM (Nm-deg) was greater in SCI than in normal subjects ($p<0.001$) (Fig. 2). Post hoc testing indicated that differences between normal and spastic individuals could be best demonstrated at 230 deg/sec, although some SCI patients demonstrated exaggerated responses at the slower velocities (Fig. 1). At 180 deg/sec, statistical distinction between normal and SCI subjects was reduced (total work and resistance to knee extension) or absent (Table 1). Even though the variability of RPJM was greater in SCI than in normal individuals, velocity remained more closely correlated with work ($r=0.62$) than with peak moments ($r=0.31$ and 0.13). Muscle activity was apparent during PJM in the patient group and, although EMG was not quantified, muscle contraction of the quadriceps and hamstrings was more evident at the higher velocities.

Table 1. Differences between normal and SCI subjects in Knee RPJM at 180 Deg/Sec and 230 Deg/Sec. (15-75 degree arc, cycles 3 through 8)

	180 Deg/Sec			230 Deg/Sec		
	Normal		SCI	Normal		SCI
Total Work [Nm-Deg]	164.5 +/-29.5	vs	286.5^{++} +/-138.9	127.3 +/-40.2	vs	474.6^{**} +/-134.6
Work During Extension [Nm-Deg]	105.8 +/-55.5	vs	205.9^{+} +/-136.1	82.8 +/-54.1	vs	251.9^{*} +/-89.9
Work During Flexion [Nm-Deg]	58.7 +/-28.1	vs	80.6 +/-57.6	44.5 +/-20.3	vs	222.7^{*} +/-110.2
Peak Moment During Extension [Nm]	9.7 +/-1.3	vs	9.9 +/-2.2	5.0 +/-2.1	vs	7.0 +/-2.3
Peak Moment During Flexion [Nm]	7.5 +/-2.3	vs	6.9 +/-4.1	3.8 +/-2.2	vs	4.3 +/-4.0

$^{+}$ $p<0.05$, $^{++}$ $p<0.01$, * $p<0.001$, ** $p<0.0001$

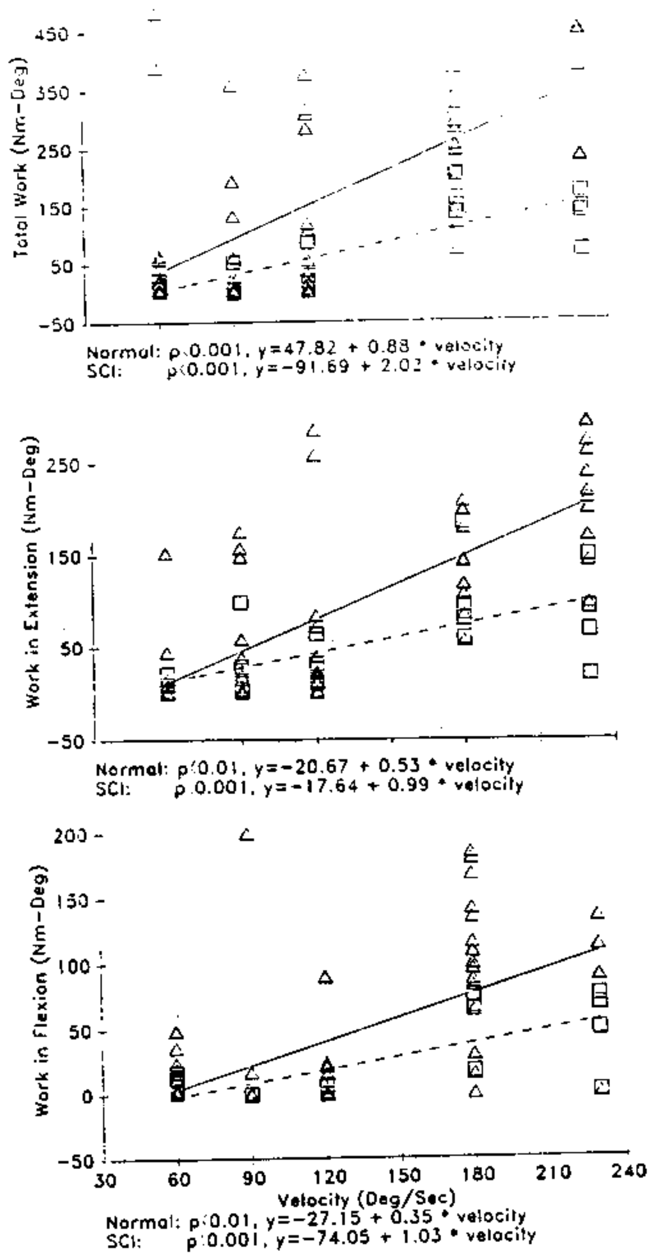

Figure 2. RPJM (Nm-Deg) in Normal versus spinal injured subjects at 60, 90, 120,180 and 230 Deg/Sec.

Differences in peak moments and work were present when the first cycle was compared to the second and third cycles, as the Lido moment arm accelerated to the selected velocity. In the normal individuals, cycles 3 through 8 were similar. Patients demonstrated more variability than normal subjects in the RPJM among all cycles (Table 1). In some knees, RPJM was greater in the first few cycles than in cycles 4 through 10 as demonstrated by the spastic patient in Figure 1.

DISCUSSION

The methodology employed in this study provides a means to detect velocity-dependent RPJM and to quantify responses for comparison over time. The use of RPJM (at velocities above the threshold for hyperreflexia) as a covariate in the analysis of electrically stimulated muscle performance offers the potential to smooth the day-to-day variability in muscle force generation and work accomplished. For example, strength-amplitude curves appear to change in concert with patients' subjective reports of their spasticity. Repetition of the same stimulus intensity (amplitude/pulse width) may result in significantly greater muscle moments on days when spasticity is considered more severe by the patient. Spastic patients who lose joint stability or experience functional muscle weakness, on the other hand, could benefit from objective documentation of their change in performance in order to justify reassessment of medication or therapeutic regimen.

Electrically stimulated muscle moments have decreased in the initial week(s) of an electrical stimulation protocol prior to the onset of improved moment and fatigue resistance. This has been observed in both complete SCI and in incomplete paralysis secondary to SCI, stroke or traumatic brain injury (22). It is possible that a reduction in spasticity, as a result of FES, may contribute to this early decrement in electrically stimulated moments. Correction of moment and fatigue values for RPJM would improve estimates of change in muscle force generation capability.

To resolve differences between subject groups (ie patient versus normal) and between sequential test sessions, critical instrumentation and procedural issues must be considered. For example, although selected patients demonstrated exaggerated resistance to PJM at slower velocities (60 and 90 deg/sec) the statistical differentiation of patients from normal individuals was possible only at 180 and 230 deg/sec. The voltages generated by the exercise device must be accessible to the investigator and calibration must be performed with weights appropriate to the range of expected moments at the test velocities. Gravity compensation at 5 deg/sec minimized the proportion of RPJM (contributed by hyperreflexia in the patient group) that was subtracted from the moment and work measurements. Removal of the resistance observed with the system operating empty, or without a subject, improved the validity of measurements, especially at the faster velocities.

The selection of cycles and/or arcs of motion for analysis allowed the rejection of mechanical artifact, and documentation of velocity-related RPJM. Variation among the first few PJM cycles in SCI patients warrant further study. The differences between normal and SCI subjects may become more apparent in these early cycles when the acceleration artifact is removed. It is possible that the present method of analysis underestimates the normal versus SCI differences.

In the future, spastic subjects might be further classified by the relative nature of the RPJM as well as the location of resistances within the arc of motion tested. This approach would be augmented by intramuscular electrodes placed in the muscles acting upon the test joint. The previously described limitations of EMG surface electrodes (51) were recognized in this study, and EMG was graded as present or absent in view of the volume conducted EMG to the quadriceps electrodes during voluntary knee flexion. Intramuscular EMG of the quadriceps and hamstrings in this study would have helped to explain the greater work during knee extension than during knee flexion (ie greater resistance to stretching of the hamstring muscles) (Table 1). Differences between the quadriceps and hamstring in RPJM have been attributed to a higher stretch reflex threshold in the quadriceps (34). Joint position could provide an event marker for the onset of muscle activity and quantification of the signal (normalized for each electrode insertion site) would permit better resolution of change in muscle response over time. Non-linear regression analysis of RPJM and intramuscular EMG can be expected to improve detection of the velocity threshold for hyperreflexia and mechanical RPJM could be better distinguished from RPJM secondary to hyperreflexia with this analysis. Increased RPJM throughout the range, or "elastic contracture," is potentially as detrimental to both neurological and musculoskeletal patients as a fixed joint contracture. Reduction in joint velocity of motion as a result of increased stiffness can significantly slow, or even prevent, functional activities such as walking (23).

Repeatability of RPJM, as described in this study, remains to be established in both normal and SCI subjects. Pilot testing of normal subjects indicates a test-retest correlation of 0.90 and 0.88 for peak moment and work values, respectively. Preliminary data indicate that patients with complete paralysis will potentially vary to a greater extent than normals and so medication administration (dose and schedule), bowel and bladder status and general health must be optimally controlled during test-retest procedures. Patients with partial paralysis may present different findings as voluntary muscle activation modulates spasticity as well as responses to FES (26,40,47-50).

In summary, the RPJM protocol described in this study can be used as a tool to document spasticity in SCI. The findings have immediate clinical application to the ongoing documentation of progress in patients with central nervous system disorders. The findings also have implications for research protocols designed to evaluate change in and/or improve muscle performance as a result of cutaneous or implanted electrical stimulation systems. In order to be useful, FES systems must produce safe and repeatable motor performance. It is suggested that electrically stimulated motor performance may be improved by the incorporation of RPJM into the evaluation of research FES system utility and patient performance when using such a system in everyday life.

REFERENCES

1. V. Alfieri, "Electrical treatment of spasticity," Scand. J. Rehab. Med., vol. 14, pp. 177-182, 1982.

2. B. J. Andrews, T. Bajd and R. H. Roxendale, "TENS treatment of spinal spasticity," Scand J. Rehab. Med. Suppl, vol. 17, p. 155, 1988.

3. B. Bajd, M. Gregorič, L. Vodovnik and H. Benko, "Electrical stimulation in treating spasticity resulting from spinal cord injury," Arch. Phys. Med. Rehabil., vol. 33, pp. 515-517, 1985.

4. B. R. Bowman and T. Bajd, "Influence of electrical stimulation on skeletal muscle spasticity," Proc. Int. Symp. on External Control of Human Extremities, Belgrade, Yugoslav Committee for Electronics and Automation, pp. 561-576, 1981.

5. A. Franek, B. Turczynski and J. Opara, "Treatment of spinal spasticity by electrical stimulation," J. Biomed. Eng., vol. 10, pp. 266-270, 1988.

6. M. G. Levine, M. Knott and H. Kabat, "Relaxation of spasticity by electrical stimulation of antagonist muscles," Arch. Phys. Med. Rehabil., vol. 33, pp. 668-673, 1952.

7. D. R. McNeal and W. K. Wilemon, "Treatment of spasticity through functional electrical stimulation," in **Neuroelectric Research**, D. V. Reynolds and A. E. Siebert, Eds. Springfield: Charles C. Thomas, 1971, pp. 65-71.

8. S. Reberšek, A. Stefanovska, N. Gros and L. Vodovnik, "The influence of electrical stimulation and passive movements on spastic ankle joint in hemiplegia," Proc. 5th Annual RESNA Conf., Houston, Texas, p. 56, 1982.

9. C. J. Robinson, N. A. Kett and J. M. Bolam, "Spasticity in spinal cord injured patients: 1. Short-term effects of surface electrical stimulation," Arch. Phys. Med. Rehabil., vol. 69, pp. 598-604, 1988.

10. C. J. Robinson, N. A. Kett and J. M. Bolam, "Spasticity in spinal cord injured patients: 2. Initial measures and long-term effects of surface electrical stimulation," Arch. Phys. Med. Rehabil., vol. 69, pp. 862-868, 1988.

11. M. Schlecht, G. Snoek, H. E. v.d. Aa, R. Kleissen and G. Zilvold, "Quantitative evaluation of the effect of spinal cord stimulation on motor function," **Advances External Control of Human Extremities IX**, pp. 405, 413, 1987.

12. A. Stefanovska, T. Bajd, L. Vodovnik and N. Gros, "Spasticity evaluation by ambulatory EMG," Dermatome stimulation: Differences in paraplegic and hemiplegic spasticity," Proc. VIII Congress of EMG and Related Clinical Neurophysiology. Shannon, Ireland: Elsevier Scientific Publishers, Ltd., vol. 66, pp. S100, 1987.

13. A. Stefanovska, N. Gros, L. Vodovnik, S. Reberšek and R. Acimovic- Janezic, "Chronic electrical stimulation for the modification of spasticity in hemiplegic patients," Scand. J. Rehab. Med. Suppl., vol 17, pp. 115-121, 1988.

14. L. Vodovnik, A. Stefanovska and T. Bajd, "Effects of stimulation parameters on modification of spinal spasticity," Med. & Biol. Eng. & Comput., vol. 25, pp. 439-442, 1987.

15. L. Vodovnik, S. Reberšek, A. Stefanovska, J. Zidar, R. Acimovic and N. Gross, "Electrical stimulation for control of paralysis and therapy of abnormal movements," Scand. J. Rehab. Med. Suppl., vol. 17, pp. 91-97, 1988.

16. A. W. Weigner and R. L. Watts, "Elastic properties of muscle measured at the elbows in man: I. Normal controls," J. Neurol. Neurosurg. & Psychiat., vol. 49, pp. 1171-1176, 1986.

17. R. R. Richardson and D. C. McLone, "Percutaneous epidural neurostimulation for paraplegic spasticity," Surg. Neurol., vol. 9, pp. 153- 155, 1978.

18. G. Barcolat-Romana, J. B. Myklebust, D. C. Hemmy, M. Myklebust and W. Wenninger, "Immediate effects of spinal cord stimulation in spinal spasticity," J. Neurosurg., vol. 62, pp. 558-562, 1985.

19. V. Mooney, E. Wilemon and D. R. McNeal, "Stimulator reduces spastic activity," JAMA, vol. 207, pp. 2199-2200, 1969.

20. R. L. Watts, A. W. Wiegner and R. R. Young, "Elastic properties of muscles measured at the elbow in man: II. Patients with Parkinsonian rigidity," J. Neurol. Neurosurg. Psychiat., vol. 49, pp. 1177-1181, 1986.

21. W. M. Landau, "Spasticity: What is it? What is it not?" in Spasticity: Disordered motor control, R. G. Feldman, R. R. Young and W. P. Koella, Eds. Chicago: Year Book Publishers, 1980, p. 17.

22. R. L. Waters, J. M. Campbell and R. Nakai, "Therapeutic electrical stimulation of the lower limb by epimysial electrodes," Clin. Orthop., vol. 233, pp. 44-52, 1988.

23. J. Perry, "Normal and pathological gait," in Atlas of Orthopaedic Biomedical Principles and Applications, Second Edition, Am. Acad. Orthop. Surgeons., Ed. St. Louis: C. V. Mosby, 1985, pp. 76-111.

24. P. Ashby and D. Burke, "Stretch reflexes in the upper limb of spastic man," J. Neurol. Neurosurg. Psychiat., vol. 34, pp. 765-771, 1971.

25. M. R. Dimitrijević and A. M. Sherwood, "Spasticity: Medical and surgical treatment," Neurol., vol. 30, pp. 19-27, 1980.

26. M. R. Dimitrijević, "Neurophysiology in spinal cord injury," Paraplegia, vol. 25, pp. 205-208, 1987.

27. R. R. Young and A. W. Wiegner, "Spasticity," Clin. Orthop., vol. 219, pp. 50-62, 1987.

28. J. W. Lance, "Symposium synopsis," in **Spasticity Disordered Motor Control,** R. G. Feldman, R. R. Young and W. P. Koella, Eds. Chicago: Year Book, 1980, pp. 485-494.

29. B. Ashworth, "Preliminary trial of carisoprodol in multiple sclerosis," Practitioner, vol. 192, pp. 540-542, 1964.

30. T. Bajd and L. Vodovnik, "Pendulum testing of spasticity," J. Biomed. Eng., vol. 6, pp. 9-16, 1984.

31. J. Kawamura, M. Ise, M. Matsuya, M. Tagami, S. Ezaki and K. Nishihara, "Clinical use of electrical stimulation for reducing spinal spasticity," in **Advances In External Control of Human Extremities IX**, 1987, pp. 353-362.

32. L. Vodovnik, U. Stanič, A. Kralj, F. Gračanin and P. Strojnik, "Functional electrical stimulation for control of locomotor systems," CRC Critical Reviews in Bioengineering, vol. 6, pp. 63-131, 1981.

33. G. C. Agarwal and G. L. Gottlieb, "Oscillation of the human ankle joint in response to applied sinusoidal torque on the foot," J. Physiol., vol. 268, pp. 151-176, 1977.

34. D. Burke, C. J. Andrews and J. D. Gillies, "The reflex response to sinusoidal stretching in spastic man," Brain, vol. 94, pp. 455-470. 1971.

35. G. L. Gottlieb, G. C. Agarwal and R. Penn, "Sinusoidal oscillation of the ankle as a means of evaluating the spastic patient," J. Neurol. Neurosurg. Psychiat., vol. 41, pp. 32-39, 1978.

36. J. F. Lehmann, R. Price, B. J. deLateur, S. Hinderer and C. Traynor, "Spasticity: Quantitative measures as a basis for assessing effectiveness of therapeutic intervention," Arch. Phys. Med. Rehabil., vol. 70, pp. 6-15, 1989.

37. S. Reberšek, A. Stefanovska, L. Vodovnik and N. Gros, "Some properties of spastic ankle joint muscles in hemiplegia," Med. & Biol. Eng. & Comput., vol. 24, pp. 19-26, 1986.

38. A. Stefanovska, L. Vodovnik, N. Gros, S. Reberšek and R. Acimovic- Janezic, "FES and spasticity," IEEE Trans. BME, vol. 36, pp. 738-745, 1989.

39. C. L. Carter, "Elastic properties of muscle measured at the elbow in man," J. Neurol. Neurosurg. Psychiat., vol. 63, p. 1019, 1989.

40. E. Knutsson, "Analysis of gait and isokinetic movements for evaluation of antispastic drugs or physical therapies," Adv. Neurol., vol. 39, pp. 1013- 1034, 1983.

41. Y. Okawa and S. Ueda, "The development of a testing device for the quantitative evaluation of muscle tone and strength," Scand. J. Rehab. Med. Suppl., vol. 17, p. 163, 1988.

42. K. C. Hayes and H. Hatze, "Passive visco-elastic properties of the structures spanning the human elbow joint," Europ. J. Appl. Physiol., vol. 37, pp. 265-274, 1977.

43. D. Burke, "Spasticity as an adaptation to pyramidal tract injury," Adv. Neurol., vol. 47, pp. 401-423, 1988.

44. B. R. Bowman and D. R. McNeal, "Response of single alpha motoneurons to high-frequency pulse trains," Appl. Neurophysiol., vol. 49, pp. 121-138, 1986.

45. B. Carnstam, L. Larsson and T. Prevec, "Improvement of gait following functional electrical stimulation. 1. Investigations on changes in voluntary strength and proprioceptive reflexes," Scand. J. Rehab. Med., vol. 9, pp. 7-13, 1977.

46. B. S. Nashold, J. Grimes, H. Friedman, J. Semans and R. Avery, "Electrical stimulation of the conus medullaris in the paraplegic," Appl. Neurophysiol., vol. 40, pp. 192-207, 1977.

47. M. R. Dimitrijević, "Residual motor functions in spinal cord injury," *Adv. Neurol., vol. 47, pp. 139-155, 1988.*

48. J. Kimura, "Recurrent inhibition of motoneurons during the silent period in man," in **Motor Control Mechanisms in Health and Disease**, J. E. Desmedt, Ed. New York: Raven Press, 1983, pp. 459-465.

49. E. Pierrot-Deseilligny, R. Katz and H. Hultborn, "Functional organization of recurrent inhibition in man: Changes preceding and accompanying voluntary movements," in **Motor Control Mechanisms in Health and Disease**, J. E. Desmedt, Ed. New York: Raven Press, 1983, pp. 443-457.

50. R. Tanaka, "Reciprocal Ia inhibitory pathway in normal man and in patients with motor disorders," in **Motor Control Mechanisms in Health and Disease**, J. E. Desmedt, Ed. New York: Raven Press, 1983, pp. 433-441.

51. J. Perry, C. S. Easterday and D. Antonelli, "Surface vs. intramuscular electrodes for EMG of superficial and deep muscles," Phys. Ther., vol. 61, pp. 7-16, 1981.

49. E. Pierrot-Deseilligny, R. Katz and H. Hultborn, "Functional organization of recurrent inhibition in man: Changes preceding and accompanying voluntary movements," in **Motor Control Mechanisms in Health and Disease**, J. E. Desmedt, Ed. New York: Raven Press, 1983, pp. 443-457.

50. R. Tanaka, "Reciprocal Ia inhibitory pathway in normal man and in patients with motor disorders," in **Motor Control Mechanisms in Health and Disease**, J. E. Desmedt, Ed. New York: Raven Press, 1983, pp. 433-441.

51. J. Perry, C. S. Easterday and D. Antonelli, "Surface vs. intramuscular electrodes for EMG of superficial and deep muscles," Phys. Ther., vol. 61, pp. 7-16, 1981.