

SECOND EDITION

SPASTICITY

DIAGNOSIS AND MANAGEMENT



Allison Brashear



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Spasticity

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Diagnosis and Management

SECOND EDITION

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*We dedicate this book to our families for their unconditional support,
and to our professors, colleagues, students and patients
who continue to humble us with their strength and challenge
us to improve the care of those with spasticity.*

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Preface

Spasticity: Diagnosis and Management is the first book solely dedicated to the diagnosis and treatment of spasticity. This second edition has been substantially revised to reflect the significant advances in the treatment of spasticity since the first edition. Our objectives in the development of this second edition were to outline the still-evolving process for the diagnosis of spasticity and the basic science behind its pathophysiology, and to provide updated information on both the measurement tools used for spasticity evaluation and the newest available treatment options. This book remains the most comprehensive guide to diagnosis and management of spasticity.

Over the past 5 years, the focus of spasticity management has moved from interventions on tone to the impact of the spasticity on the lives of patients and caregivers. Additional drugs, including new forms of botulinum toxin, have been reported in large clinical trials and are changing or will, in the future, change treatment paradigms. Comprehensive programs in spasticity management increasingly focus on special populations including children, cancer survivors, and patients in long-term care programs. As a result, this edition addresses new treatment pathways, outcomes, and economics of spasticity care within the larger context of the rapidly changing health care environment.

Divided into four sections, this book is intended to provide both clinicians and researchers up-to-date access on the latest comprehensive treatment of spasticity. Part I includes a general overview with four chapters highlighting why spasticity is important, epidemiology of spasticity and other signs of the upper motor neuron syndrome, and finally ancillary findings associated with caring for the patient with spasticity.

Part II focuses on the assessment tools in diagnosis and management of spasticity. Five chapters include an outline of general overview measurement tools, specific techniques and scales, assessment of the upper and lower extremity, and setting realistic goals for treatment. The revised chapter, "Measurement Tools

and Treatment Outcomes in Patients With Spasticity," includes the Goal Attainment Scale, which is specifically designed to focus on patient-specific outcomes. The newly added chapter, "Techniques and Scales for Measuring Spastic Paresis," details the use of scales such as the Tardieu. The use of such scales is more common in both patient care and clinical trials. These chapters provide details on the administration of these scales. Taken together, these five chapters provide a comprehensive review of assessment and measurement of spasticity.

Part III provides 11 comprehensive chapters on treatment of spasticity. New chapters include the role of the physical and occupational therapist in spasticity management, the use of ultrasound in guidance of botulinum toxin management, and emerging technologies in the treatment of spasticity. Part III is designed to highlight the changes in the field in the past 5 years.

The final section, Part IV, is devoted to individual diseases involving spasticity and treatment within the context of these conditions. In addition to updated chapters on evaluation, genetics, and spasticity in adults and children with spinal cord injury, multiple sclerosis, stroke, traumatic brain injury, and cerebral palsy, we have added new chapters on more specialized areas including spasticity in patients with cancer, treatment of spasticity in patients in long-term care facilities, and the economics of spasticity treatment.

With the development of effective therapies for spasticity, we originally sought to address the diagnosis and treatment of spasticity in an integrated, clinically useful text. This revised second edition builds on that foundation and integrates recent advances in the field for diagnosis, treatment, and outcomes. The real focus of this book is on providing the most up-to-date, effective, comprehensive, and economical therapy for patients with spasticity. We invite you to explore these pages and join us in our mission to improve the care for our patients with spasticity.

Allison Brashear, MD, MBA

Acknowledgments

Thank you to our patients, their families, our colleagues and staff, and our families for their many contributions to this text. This second edition challenges us to improve the diagnosis and care of spasticity in our patients. Thank you to you, the reader, for

joining us on this journey. We hope this book inspires you to continue to improve the diagnosis and management of spasticity.

Allison Brashear, MD, MBA

Share

Spasticity: Diagnosis and Management, Second Edition



General Overview

Why Is Spasticity Treatment Important?

Allison Brashear and Elie Elovic

Spasticity treatment is important because the increased tone may interfere with the physical functioning of patients. The overarching goal of spasticity management should be to improve the ability of patients to perform active and passive ranges of motion and improve the ability of caregivers to assist patients with disabilities. Increased tone or spasticity is the tightness that patients and/or caregivers report with passive movement of the limb. In more scientific language, spasticity is a motor disorder characterized by a velocity-dependent increase in the tonic stretch reflex. A clinical finding on the neurologic examination, spasticity, together with increased tone, brisk reflexes with incoordination, and weakness, represents the upper motor neuron syndrome.

Regardless of the cause, spasticity causes significant disability. An estimated 4 million individuals are stroke survivors in the United States, and as many as one third may have spasticity with sufficient disability to require treatment. According to the Centers for Disease Control and Prevention, 1.4 million people in the United States sustain a traumatic brain injury each year, and additional patients develop spasticity after spinal cord injury. The result of any brain or spinal cord injury is a variable pattern of increased tone with weakness and discoordination that leads to significant disability in many patients.

The treatment of spasticity relies on the physician's assessment of the individual together with conversations with the caregiver. Patients' inability to perform simple activities of daily living for themselves and the adverse effects on the caregiver drive physicians to find ways to decrease tone, build strength, and improve coordination. The team approach is a cornerstone of a successful treatment, and interaction of the patient, the caregiver, the therapist, and the physicians works best to provide a care plan that addresses functional impairment and plots a course to treat the problems.

Spasticity is a clinically relevant medical problem when it interferes with function or care of patients. The evolution of upper motor neuron syndrome may take days to months after a central nervous system injury. Moreover, the presentation in one patient may differ from that of another despite both having similar central nervous system lesions. The lesion alone does not predict the amount or impact of the spasticity. Other factors such as medications, stress, medical illness, timing of therapy, and so on impact the clinical presentation. As a result, each patient must be assessed individually with his or her caregiver, noting the concerns that impair the performance of activities of daily living or other deficits. No matter how much we learn about stroke, traumatic brain injury, multiple sclerosis, and spinal cord injury, the assessment of spasticity and the effect of tone on function will remain unique to each individual patient's circumstance.

Although neurologic examination is essential for the diagnosis of spasticity, the management of spasticity has many paths for treatment depending on the disability and goals of the patient and caregiver. One patient may benefit from a combination of tools for spasticity, including interventions such as botulinum toxin injections and intrathecal baclofen, whereas others may require a more conservative route such as splinting or oral medications. The informed physician should know how to assess the amount of spasticity, determine the functional limitations it creates, and then be able to develop a management plan for that individual patient.

How to assess the complicated picture of spasticity and when to intervene are the focus of this text. Our coauthors define for you why spasticity is important and detail the diagnosis and management options, but the goal is to provide the reader with the best options for the physician's individual patient. As editors, we aim to explore the diagnosis and management of the

many different types of patients with spasticity and to open the door to the different treatment paradigms for patients with spasticity. This second edition has been updated to reflect the newest assessments and treatments.

So why is spasticity important? The answer is because it often causes disability and impairs function in our patients. The goal of this book is to provide the foundation for excellent care of our patients facing these disabilities.

Epidemiology of Spasticity in the Adult and Child

John R. McGuire

Despite the extensive work done to develop improved treatments for muscle overactivity in patients with upper motor neuron (UMN) lesions, there is only a limited number of studies on the incidence and prevalence of spasticity. Most likely, this is due to the lack of consistent definitions and reliable measures of spastic hypertonia. Compounding the difficulties in the literature is that the few prevalence studies that have been performed rely primarily on patient surveys or clinical measures of spasticity, which lack sensitivity for quantifying abnormal muscle activation (1). More importantly, there are fewer studies on the prevalence of problematic or significant spasticity. A final issue is that different authors use dissimilar definitions for the condition. Some of the descriptions have included spasticity that requires medication or physiotherapy (2–4), causes pain, (5) interferes with activities of daily living (ADL) (6–10), or has an Ashworth score of 2 or higher (11,12).

The actual incidence of spasticity depends on the cause of the UMN lesion. After damage to central motor pathways above T12, there is initial paralysis followed by adaptive changes in the brain and spinal cord that develop over time, which result in a complex set of motor behaviors (13–17). Paresis, soft tissue contracture, and muscle overactivity are the three major mechanisms of motor impairment (18). Although spasticity is often used as an umbrella term, it is just one component of the muscle overactivity that contributes to the upper motor neuron syndrome (UMNS) (16,19,20). Reliable assessments are complicated by the fact that spasticity can vary throughout the day, change with different positions, and increase with any noxious stimulus, such as pressure sores, urinary tract infection, deep venous thrombosis, ingrown toenails, joint pain, or constipation (15,16,21).

In a large population-based study initiated by the Christopher & Dana Reeve Foundation Paralysis Resource Center (PRC) and researchers at the University of New Mexico's Center for Development and Disability that was performed from 2006 to 2008, more than 33,000 households across the country were surveyed for any disability (22). From this review, they estimated that nearly 1 in 50 people or approximately 6 million people in the United States are living with paralysis. The leading etiologies for this condition were stroke, spinal cord injury (SCI), multiple sclerosis (MS), and cerebral palsy (CP; Table 2.1) (22). It should be noted that the prevalence of paralysis noted in people with SCI and MS from this survey is significantly higher than those found previously.

Additional important questions that need to be answered are: what are the conditions that cause problematic spasticity and what is the number of people who require treatment? Data collected from the adult spasticity management clinic at the Medical College of Wisconsin (MCW) during a 6-month period in 2008 may give some answers to these questions. The number and diagnosis of the patients treated with intrathecal baclofen (ITB) or botulinum neurotoxin (BoNT) are shown in Table 2.2. SCI, CP, and MS were the most common diagnoses treated with ITB, whereas stroke, CP, and SCI were the most common conditions treated with BoNT. This suggests that patients with these conditions may have the highest prevalence of problematic spasticity.

When discussing the conditions in children, data from the work of Hutchison et al (23) may shed some light. The most common causes of spasticity in 341 children seen at some of the clinics at the Royal Children's Hospital in Melbourne, Australia, in 1998 were CP (79%), traumatic brain injury (TBI; 6%), spina bifida (5%), and SCI (2%).

TABLE 2.1

PREVALENCE OF PARALYSIS IN THE UNITED STATES (<i>N</i> = 5,596,000)		
Cause	<i>n</i>	%
Stroke	1,608,000	29
SCI	1,275,000	23
MS	939,000	17
CP	412,000	7
Postpolio syndrome	272,000	5
TBI	242,000	4
Neurofibromatosis	212,000	4
Other	636,000	11

CP, cerebral palsy; MS, multiple sclerosis; SCI, spinal cord injury; TBI, traumatic brain injury.

Source: Adapted from Ref. (22). Paralysis Resource Center, Christopher & Dana Reeve Foundation, 2015.

TABLE 2.2

PATIENTS TREATED FOR SPASTICITY AT THE MEDICAL COLLEGE OF WISCONSIN WITH ITB OR BoNT FROM JANUARY 2008 TO JULY 2008		
Diagnosis	ITB Patients	BoNT Patients
SCI	43 (30%)	50 (14%)
CP	34 (24%)	74 (20%)
MS	30 (21%)	23 (6%)
TBI	17 (12%)	37 (10%)
Stroke	6 (4%)	133 (37%)
Anoxic encephalopathy	3 (2%)	43 (12%)
Other	11 (7%)	3 (1%)
Total	144	363

BoNT, botulinum neurotoxin; CP, cerebral palsy; ITB, intrathecal baclofen; MS, multiple sclerosis; SCI, spinal cord injury; TBI, traumatic brain injury.

STROKE

Each year, approximately 795,000 people in the United States experience a new (about 610,000) or recurrent (185,000) stroke (24). The prevalence of stroke in the United States is 2.6 million for men and

3.9 million for women (24), with an annual incidence rate of 183 per 100,000 (25). There are significant differences in the prevalence of stroke by race/ethnicity, education level, and state/area of residence (26). Blacks have a higher incidence of stroke than whites, especially among the young, and the rate increases with age regardless of race (24). In Europe, the annual standardized incidence for stroke is 113 per 100,000 per year (27). In Sweden, with a population of 9 million, about 30,000 patients are hospitalized every year because of stroke, of whom 20,000 experience a first-ever stroke (28).

Four studies evaluated the prevalence of spasticity after a stroke and are summarized in Table 2.3. They are all from Europe, with the prevalence of spasticity ranging from 17% to 38%. Each identified the arm and leg spasticity using the Modified Ashworth Scale (MAS) score (29) and used the Barthel Index (BI) (30,31) as the functional measures. In a cross-sectional survey 1 year poststroke, Lundström et al (2) identified 140 people with their first event from a national stroke registry. Arm and leg spasticity was measured using the MAS, and disability was measured with the modified Rankin Scale (32) and the BI. Disabling spasticity (DS) was defined as spasticity in need of an intervention, for example, intensive physiotherapy, orthosis, or pharmacological treatment. The observed prevalence of any spasticity was 17% and of DS 4%. Patients with DS scored significantly worse on the modified Rankin Scale and the BI than those with no DS. DS was more frequent in the upper extremity and correlated positively with other indices of motor impairment and inversely with age. Although the prevalence of DS after a first-ever stroke from this study was low, in the context of the large number of stroke survivors, the number became more significant.

In a Swedish cohort study, Sommerfeld et al (33) evaluated 95 patients with a first-ever stroke within 1 week of their stroke (mean, 5.4 days) and 3 months after their event. The authors measured spasticity by obtaining the MAS for the arm and leg, as well as self-reported muscle stiffness, tendon reflexes, several motor impairment measures, and the BI as a disability measure. Of the 95 patients studied, 64 were hemiparetic, 18 were spastic, 6 reported muscle stiffness, and 18 had increased tendon reflexes 3 months after stroke. Nonspastic patients (77 total) had statistically significantly better motor and activity scores than spastic patients (18). However, the correlations between muscle tone and disability scores were low, and severe disabilities were seen in almost the same number of nonspastic and spastic patients. They concluded that severe disabilities were seen in almost the same number of nonspastic and spastic patients

TABLE 2.3

PREVALENCE OF SPASTICITY AFTER FIRST STROKE						
Study	No. of Patients	Time Poststroke	Spasticity Diagnosis	Location	Prevalence of Spasticity (%)	Problematic Spasticity (%)
Lundström et al 2008 (2)	140	1 year	MAS	Sweden	17	4 ^a
Welmer et al 2006 (34)	66	18 months	MAS	Sweden	20	NR
Sommerfeld et al 2004 (33)	95	<1 week 3 months	MAS	Sweden	21 19	NR
Watkins et al 2002 (10)	106	12 months	MAS- elbow TAS combined	UK	27 36 38	67 ^b

^aSpasticity that requires an intervention, for example, physiotherapy, orthosis, pharmacologic.

^bPatients with arm and leg spasticity (67%) had 50% lower Barthel score than patients with no spasticity.

MAS, Modified Ashworth Scale; NR, not reported; TAS, Tone Assessment Scale.

and suggested that the importance of spasticity may be overstated. There were several limitations to this study including the small number of participants and the investigators' reliance on the use of the MAS as the only means of identifying if a person has spasticity. As a result, they may have missed patients with spasticity or other components of the UMNS (10). In addition, the sample of patients only had a limited amount of motor deficits because 67% were hemiparetic at 3 months. Of this group, 28% had spasticity (33). In an 18-month follow-up study with the same cohort of patients, Welmer et al (34) evaluated the frequency of spasticity and its association with functioning and health-related quality of life (HRQL) (35). Of the 66 patients studied, 38 were hemiparetic; of these, 13 displayed spasticity, 12 had increased tendon reflexes, and 7 reported muscle stiffness 18 months after stroke. Although there was a weak correlation between spasticity and HRQL, the hemiparetic patients without spasticity had significantly better BI functioning scores and significantly better HRQL scales than patients with spasticity. This follow-up study suggests that spasticity may have a negative impact in the long-term on functional improvement in patients who have had stroke.

Watkins et al (10) evaluated 106 consecutive community-dwelling stroke survivors in Liverpool, UK, who were 12 months poststroke. They measured spasticity at the elbow using the MAS and at several joints and in the arms and legs using the Tone Assessment Scale (TAS) (36); they also assessed disability using the BI. The prevalence of spasticity in

their study depended on the metric used. Using the MAS, 29 (27%) of the 106 patients had spasticity, whereas 38 (36%) were identified as spastic using the TAS. Forty (38%) were spastic when including those who were identified as having tone by either metric. Those with spasticity had significantly lower BI scores at 12 months, whereas those with arm and leg involvement had a BI 50% of those without spasticity.

Of the four studies that addressed the prevalence of spasticity in stroke survivors, three suggest that it is associated with greater motor impairments and has a negative impact on functional capabilities. The low prevalence of spasticity in these reports is most likely due to the lack of sensitivity of the measures used to assess it and the mild motor impairments of the samples studied. The study of more involved patients can be undertaken by looking at prevalence of spasticity from an inpatient rehabilitation unit. Francisco (4) performed this type of study when he presented a retrospective review of 204 stroke admissions to a free-standing rehabilitation hospital in 2002. The mean duration of stroke to admission was 5.76 days (range, 1.2–48 months), and 78% of the patients had hemorrhagic strokes. Seventy percent had spastic hypertonia (MAS \geq 1), and 50% had clinically significant spasticity that required treatment. The larger prevalence of problematic spasticity in this group supports the notion that more severe spasticity is associated with greater impairments, as many of the patients included in this investigation also had severe motor, language, and cognitive impairments.

Two studies used electrophysiologic measures to evaluate the prevalence of spasticity after a stroke. O'Dwyer et al (37) evaluated 24 hemiparetic stroke patients 1 to 13 months (mean, 5.3 months) after their event for upper limb spasticity and contracture. The motor impairment was graded mild to severe based on item 6 of the Motor Assessment Scale (38). They studied stretch-induced electromyographic activity of the biceps muscle at different velocities of stretch and found tonic stretch reflexes in 5 patients (21%). Of the 24 patients, 13 had a flexion contracture from 2° to 22°, suggesting that contracture may be more important than spasticity in this population. Although this study had similar prevalence data to the studies in Table 2.3, there were a limited number of patients in this study and they only tested one muscle for spasticity. In a larger study, Malhotra et al (1) evaluated wrist spasticity in 100 patients 1 to 6 weeks (mean, 3 weeks) after their first stroke with severe weakness (scored 0 in the grasp section of the Action Research Arm Test) (39). Spasticity was evaluated using the MAS and biomechanical and neurophysiologic measures. The MAS was abnormal in 44 patients, and 87 patients had abnormal involuntary muscle activation using a novel portable device with an electrogoniometer, force transducer, and surface bipolar electromyographic electrodes. This suggests that neurophysiologic measures for spasticity are more sensitive than clinical ones and that assessing prevalence with clinical metrics may result in an underestimate. Additional studies with more objective measures of spasticity are needed to more accurately determine the prevalence of spasticity in patients who have had stroke.

SPINAL CORD INJURY

The estimated annual incidence of SCI in the United States, not including those who die at the scene of the accident, is approximately 40 cases per million or approximately 12,000 new cases each year (40). The estimated prevalence of SCI in the United States for 2008 was approximately 259,000 persons, with studies reporting within a range of 229,000 to 306,000 persons (40). The PRC reports a much higher estimate of SCI prevalence of approximately 1,275,000 people in the United States, with the most common cause of SCI being motor vehicle accidents followed by falls and acts of violence (22). Sports-related SCIs occur more commonly in children and teenagers, whereas work-related injuries are more common in adults. Most people with SCI are in their teens or 20s, and 78% are male (41). The male preponderance of SCI decreases after age 65 years, at which point, the

most common mechanism of SCI is falls. More than half of all SCI occur at the cervical level, almost a third in the thoracic level, and the remainder in the lumbar area (41).

Table 2.4 summarizes the studies that assessed the prevalence of spasticity in patients with SCI. Of the seven studies reviewed, three of the studies used clinical assessments to identify patients with spasticity, whereas three used patient questionnaires. The prevalence of spasticity ranged from 40% to 78% (average, 68%), with the higher prevalence noted in the studies that used a clinical scale. The prevalence of problematic spasticity was addressed in five of the studies. The criteria used to define it was if the patient required medication for treatment and if their spasticity interfered with ADL, was painful, or both. Using these measures, the prevalence of problematic spasticity ranged from 12% to 49%, with an average of 33%.

In the first of two epidemiological studies, Maynard et al (3) evaluated the occurrence of spasticity and its severity in 96 patients at one SCI center. Spasticity was considered present if the patient had increased deep tendon reflexes, muscle tone during passive movements, or involuntary muscle spasms. Severity of spasticity was determined if patients were taking antispasticity medication and if they had satisfactory treatment. Treatment was indicated if the spasticity was interfering with ADL and sleep or caused pain that prevented or interfered with activities. By this definition, 67% of the patients had spasticity at the time of their discharge (average, 118 days) and 37% were taking anti-spasticity medication. The incidence of spasticity was higher among groups with cervical and upper thoracic levels of injury compared with groups with other levels of injury. At their 1-year follow-up, the percent of patients with spasticity increased to 78% and 49% of them required medication. The second part of the study analyzed the presence of spasticity severe enough to require treatment in 466 subjects with SCI from 13 different SCI centers. From this patient population, 26% of the patients received anti-spasticity treatment at the time of discharge (average, 105 days), and the percentage increased to 46% at their 1-year follow-up. Spasticity treatment was more common in cervical and upper thoracic patients with incomplete injuries. The percentage of patients requiring spasticity treatment with Frankel grades B (sensory incomplete, motor complete) and C (motor incomplete, nonfunctional) was 50% and 52% (42), respectively, whereas the percentage of patients requiring spasticity treatment with Frankel grades A (sensory and motor complete) and D (motor incomplete, functional) was 27% and 29%, respectively. Little et al (43) reported similar

TABLE 2.4

PREVALENCE OF SPASTICITY AFTER SCI						
Study	No. of Patients	Time Postinjury	Spasticity Diagnosis	Location, Duration	Prevalence of Spasticity (%)	Problematic Spasticity (%)
Maynard et al 1990 (3)	96	DC	CS	Michigan, 1985–1988	67	37 ^a
Study 1		1 year			78	49
Maynard et al 1990 (3)	466	DC	CS	USA	NR	26
Study 2		1 year				46
Anson et al 1996 (44)	191	1 to >15 years	NR	Atlanta	62	12 ^b
Johnson et al 1998 (45)	853	1 year 3 years 5 years	PSR	Colorado	NR	35 ^c 32 28
Sköld et al 1999 (5)	354	12 months	MAS	Sweden, 1997	65	30 ^d
Noreau et al 2000 (46)	482	12 months	PSR ^e	Quebec	40	NR
Walter et al 2002 (47)	99	NR	PSR ^f	Chicago Hines VA	53	40 ^a

^aSpasticity that required medication.

^bSpasticity that interfered with ADLs.

^cProblematic spasticity.

^dSpasticity that was painful, restricting ADLs, or both.

^ePSR: "Over the past 12 months have you developed or suffered from spasticity?"

^fPSR: "Are you having a problem with spasticity?"

ADL, activities of daily living; CS, Clinical Scale (spasticity present if deep tendon reflexes increased, increased muscle tone during passive movements, or involuntary muscle spasms); DC, discharge from hospital; MAS, Modified Ashworth Scale; NR, not reported; PSR, patient self-report.

findings in 26 patients with SCI, where the patients with Frankel grade C had greater flexor withdrawal responses and extensor spasms, more pain, and interference with sleep than those with Frankel grades A and D. These findings suggest that increased time after injury and motor incompleteness of SCI may contribute to the increased severity of spasticity.

Johnson et al (45) investigated the frequency of both medical and nonmedical complications reported to the Colorado Spinal Cord Injury Early Notification System for patients with SCI. They interviewed each patient by telephone at 1, 3, and 5 years after injury. They noted a decrease in the prevalence of spasticity from year 1 (35%) to year 5 (28%), which may have been due to reduced sample size at year 5 (50% of year 1). They also noted that spasticity had a variable

impact on quality of life and productivity measures. They recommended that follow-up needs to be longer than 5 years (decades rather than years) to gauge the full impact of each SCI complication. Using both physical examination and patient self-report, Sköld et al (5) found abnormal MAS in only 60% of the patients reporting significant spasticity, whereas 97% of patients with abnormal MAS reported spasticity. This study underscores the importance of using both clinical measures and patient self-report when evaluating problematic spasticity. The other studies using patient self-report, which are summarized in Table 2.4, support the need for patient questionnaires to ask sufficient questions to determine the full impact of spasticity and the other components of UMNS on the patient's daily activities (44,46,47).