Chapter 7
How Does Spasticity Affect Patients with Disorders of Consciousness?

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Abstract Spasticity is a frequent issue encountered by brain-damaged patients, arising from an anarchic reorganization of the central nervous system that may significantly alter motor function. While it is well described in patients with a lesion of the descending corticospinal system, little is known about the occurrence and physiopathology of this disorder in patients with more complex brain lesions and disorders of consciousness (coma, unresponsive wakefulness syndrome, and minimally conscious state). Most of the time, these patients are bedridden and lack voluntary motor command which favors spasticity to occur and may lead to complications including pain, loss in range of motion, or bed sores. Given the inability for many of these patients to express their pain or discomfort and knowing that spastic syndromes may restrain them to express signs of consciousness, the multimodal treatment of this spasticity is crucial for their management. In the present chapter, we describe the physiopathology and the current available treatments of spasticity in this specific population of severe brain-injured patients with disorders of consciousness.

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List of Abbreviations

CNS	Central nervous system
DOC	Disorders of consciousness
EMG	Electromyogram
MAS	Modified Ashworth Scale
MCS	Minimally conscious state
MTS	Modified Tardieu Scale
ROM	Range of motion
TBI	Traumatic brain injury
UMN	Upper motor neuron
UWS	Unresponsive wakefulness syndrome

Introduction

Spasticity is a motor disorder occurring after a lesion of the central nervous system (CNS) such as stroke, spinal cord injury, multiple sclerosis, or traumatic brain injury [1]. This trouble is commonly defined as *a motor disorder, characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyper-excitability of the stretch reflex as one component of the upper motor neuron (UMN) syndrome* [2]. The UMN syndrome classically shows positive (e.g., increased tendon reflexes, clonus, positive Babinski sign) and negative signs (e.g., muscle weakness, loss of dexterity, fatigability) [3]. A more recent definition describes spasticity as *a disordered sensori-motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscles* [3]. Actually, there are many and various definitions of spasticity [4, 5] which shows there is no any consensus yet on its specific meaning. Nonetheless, notions of increased hypertonia and hyperreflexia are widely accepted through the different definitions of spasticity [1, 6, 7]. However, a specific definition of spasticity accepted by all still remains to be determined.

This disorder occurs in about one-third of patients who suffered from a stroke [8] or a traumatic brain injury (TBI) [9] and can reach up to 89% in chronic patients with disorders of consciousness (DOC) [10]. These altered states of consciousness include coma, unresponsive wakefulness syndrome (UWS), and minimally conscious state (MCS). While coma is characterized by the complete loss of both wakefulness and awareness [11], the UWS means that the patient has recovered sleep-wake cycles but without any sign of awareness of self, nor the environment [12, 13]. A patient in MCS shows inconsistent but clearly discernible behavioral signs of consciousness (e.g., response to command, visual pursuit, object manipulation, or verbalization) [14]. However, he/she is unable to functionally communicate yet.

Only a few studies have investigated spasticity and its side effects or consequences in patients with DOC, whereas these patients often raise serious issues

about their daily therapeutic management. Therefore, it is of a high importance to take care of this disorder in this population of noncommunicative brain-injured patients.

Clinically, spasticity is demonstrated through exaggerated tendon tap reflexes associated with an increased and velocity-related resistance of a muscle when passively stretched [15]. Yet, patients suffering from spasticity may also present, in addition to hyperexcitability of the stretch reflex, spastic dystonia (muscle constriction at rest) or spastic cocontractions (contraction of both agonist and antagonist muscles during the volitional movement) [10]. Spasticity arises from a dissociation of sensory input (e.g., passive movement) from the motor responses, resulting in hyperexcitability of these latter by increased segmental CNS processing [16, 17]. In chronic paralyzed patients, spasticity is often associated with ankylosis of the joints, or even joint fixation. In this case, the velocity-related resistance of a muscle when passively stretched is more difficult to assess.

Spasticity has to be objectively assessed, in order to follow its evolution over time. To this end, several scales have been developed and validated [18]. The most commonly used are the Modified Ashworth Scale (MAS) and the Modified Tardieu Scale (MTS) (Table 7.1). The MAS measures the level of resistance to a passive movement. This scale is widely used in both research and clinical practice since it is fast and easy to use, but validation studies showed "poor" to "moderate" inter-rater reliability despite a "moderate" to "good" intra-rater reliability [19, 20]. The investigator assessing spasticity should thus always be the same person.

The MAS does not take into account the influence of the velocity of the passive movement, whereas its importance is specified in several definitions. On the other hand, the MTS does take into account this parameter using three different velocities (low, normal, and fast), and it includes the angle of contraction outbreak as well. However, its validity still needs to be proven [21].

Other clinical tools are used to quantify spasticity in a more objective way. For instance, electromyography (EMG) is a commonly used neurophysiological method assessing muscles' response to mechanical or electrical stimuli. The electrical signals preceding mechanical muscle activity can provide information about muscle properties and neuromuscular control. Therefore, neurophysiological assessments are often employed to investigate the effects of therapeutic interventions on spasticity, as well as to understand the different pathways involved [22]. Biomechanical standardized methods involving isokinetic dynamometers are other options to evaluate spasticity in an objective manner [23, 24]. An "optimal" evaluation would reside in a combination of electrophysiological and biomechanical techniques to see if the mechanical response of the muscle is proportional to the electrical signal. Indeed, while the EMG measurement only allows determining the stretch reflex threshold, adding a biomechanical assessment would permit evaluating the relationship between stretch velocity and the evoked stretch reflex-mediated torque generated from the stretched muscle [25]. Assessing patient's spasticity using reliable and sensitive tools is crucial in order to develop and adjust patients' most effective antispastic treatment.

Table 7.1 The Modified Ashworth Scale (MAS) and the Modified Tardieu Scale (MTS) [10]

Modified Ashworth Scale	
0	No increase in muscle tone
1	Slight increase in muscle tone, manifested by a catch or by minimal resistance at the end of the range of motion (ROM)when the affected part(s) is (are) moved in flexion or extension
1+	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM
2	More marked increase in muscle tone through most of the ROM, but affected part(s) is (are) easily moved
3	Considerable increase in muscle tone, passive movement difficult
4	Affected part(s) is (are) rigid in flexion or extension
Modified Tardieu Scale	
X: Quality of movement mobilization	
0	No resistance throughout the course of the passive movement
1	Slight resistance throughout the course of passive movement, no clear catch at a precise angle
2	Clear catch at a precise angle, interrupting the passive movement, followed by release
3	Fatigable clonus with less than 10 s when maintaining the pressure and appearing at the precise angle
4	Unfatigable clonus with more than 10 s when maintaining the pressure and appearing at a precise angle
5	Joint is fixed
V: Measurements take place in three different velocities	
V1	As slow as possible
V2	Speed of limb segment falling under gravity
V3	As fast as possible
Y: Angle of catching (muscle reaction)	

Regarding treatments available to manage spasticity, the most commonly used are pharmacological drugs. Most pharmacological treatments are targeting the reduction of reflex activity by decreasing the release of excitatory neurotransmitters (glutamate, monoamines) or by potentiating the activity of inhibitory neurotransmitters (GABA, glycine) [26]. Baclofen, a GABAb receptor agonist, is one of the most widely used oral antispastic drug [27]. It reduces spasticity by enhancing presynaptic inhibition at the spinal level [15]. Some drugs act by increasing the affinity of GABA to its receptor complex (diazepam, clonazepam) or miming the GABA structure (gabapentin), while others take action at the muscular level (dantrolene, phenol, botulinum toxin) [10]. Intrathecal baclofen is another type of treatment against spasticity. In this case, baclofen is delivered by an implantable pump directly into the spinal fluid, aiming to have more direct effects while less side effects (e.g., sleepiness). Moreover, it appears to improve the level of consciousness and not only on a motor side but also regarding visual pursuit, object-related eye movements, and verbalization attempts [28, 29]. However, no controlled clinical

trials have been done to assess the effect of baclofen in a large cohort of patients with DOC, and the mechanisms underlying these behavioral effects have to be further investigated.

Beside pharmacological treatments, various non-pharmacological treatments exist, including physical therapy (especially stretching) [30], occupational therapy [31], orthoses [32], transcutaneous electrical nerve stimulation [33], cortical activation by thalamic stimulation [34], and surgical interventions [10]. However, all these treatments tend to reduce the symptoms of spasticity and not the source of spasticity itself. This can be explained by the lack of understanding regarding the exact pathophysiology of spasticity.

Spasticity in Patients with Disorders of Consciousness (DOC)

Pathophysiology

Processes underlying spasticity have not been fully understood yet, and its pathophysiology is multifactorial. Physiologically, muscle overactivity and pathological reflex responses to peripheral inputs such as cutaneous stimuli or muscle stretch might be the consequence of an anarchic reorganization of the CNS after a brain lesion [35, 36].

It is also known that central motor lesions are associated with loss of supraspinal control leading to impaired patterns of spinal reflexes [1]. On the other hand, intramuscular changes such as alterations of the collagen tissue, change in muscle fiber type, or loss of sarcomeres result in a spastic muscle pattern [37, 38]. Based on experimental animal models, spasticity would result from an imbalance between the inhibitory lateral reticulospinal tract and the excitatory medial reticulospinal and vestibulospinal tracts [39].

The location of the lesion(s) plays, of course, a determining role [40]. Damage of the cerebral cortex, cerebellum, and basal ganglia likely results in the abnormal muscle tone and motor patterns in patients with DOC [41]. The clinical signs of spasticity are due to a lesion of the UMNs (as part of the UMN syndrome) which include supraspinal inhibitory and excitatory fibers controlling spinal reflex activity. These UMNs are motor neurons starting in the motor cortical regions (Brodmann areas 4 and 6) or in the brain stem. However, spasticity emerges not only as a consequence of pyramidal tract lesion but due to parapyramidal fiber (e.g., dorsal/lateral reticulospinal tract) damages as well [35].

Due to these central lesions, patients may suffer from paralysis or paresis defined as *decreased voluntary motor unit recruitment* [42]. Beside the lesion itself, spasticity may be influenced by other non-neurological factors. Indeed, patients with DOC are mostly confined to bed, and many of them have lost voluntary motor movements. We observe immobilization in the one hand and disuse in the other hand.

While immobilization is a peripheral situation of lack of passive or active movement around a joint, disuse is a central situation of lack of voluntary command to this joint. These two different phenomena tend to occur together and have devastating motor consequences.

Patients are thus immobilized with their muscles in a shortened position which causes a reduction in longitudinal tension. This way, the muscles will lose mass and sarcomeres while accumulating connective tissue and fat [43, 44] leading to an exacerbation of muscle contracture. In addition, the loss of gravity effects (weight-bearing and counter-resistance activity) will major these phenomena [45] and reduce bone mineralization by stimulating catabolic responses of the musculoskeletal system [46]. Muscles which are maintained in a shorter position adapt to this resting length, and the amount of sarcomeres decreases and reorganizes in order to develop maximal tension at this new reduced length [47]. Not only the muscle is involved but the myotendinous junction endures a decrease in its tensile strength as well as due to a reduction in local vascular density and degenerative changes [48, 49]. This may also occur in patients encountering immobilization without neurological lesion (e.g., cast, burn). In patients with a CNS lesion, after a few weeks, the emergence of muscle overactivity becomes then an additional mechanism which will aggravate the contractures (defined as *loss of range of motion in a joint to a degree that impedes activities of daily living* [50]). If not treated, this decreasing passive muscle extensibility will lead to an acquired loss of ROM up to a permanent joint fixation [7, 51].

Afterward, progressive supraspinal and spinal rearrangements will give rise to muscle overactivity (defined by "increased involuntary motor unit recruitment" [36]). It progressively appears when the central execution of voluntary command is disrupted and represents another aggravating factor. Spastic overactivity as defined by Gracies [36] includes spasticity, spastic dystonia, and spastic cocontraction which are distinguished by their primary triggering factor (i.e., phasic muscle stretch, tonic muscle stretch, and volitional command, respectively). The precise pathophysiology of these excessive responses is still incompletely understood [5]. Logically, all these phenomena (paresis, contracture, and muscle overactivity) never present a symmetrical distribution between agonist and antagonist muscles, leading to torque imbalances around joints and deformities. As patients with DOC present a paresis that is aggravated by disuse due to the lack of voluntary command, the therapeutic challenge is to break the vicious cycle paresis—disuse—further paresis [42] (Fig. 7.1).

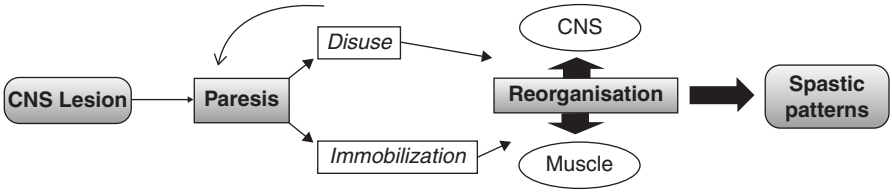

Fig. 7.1 Pathophysiology of spastic patterns (spasticity, spastic dystonia, spastic cocontractions)

Clinical Picture

In patients suffering from spasticity, the stretch reflexes are preserved and potentially accentuated [38]. Indeed, for a given velocity, stretch responses are increased and appear at a lower threshold compared to healthy subjects [39]. In some extreme cases, muscle contracture can be permanent, leading to a complete joint fixation. Regarding patients with DOC, they seldom fit in one particular clinical setting.

The first issue is to distinguish spasticity from rigidity. Rigidity is a form of plastic hypertonia arising from remodeling occurring in the basal ganglia [52]. It does not depend upon the speed of the muscle stretch and is not associated with other positive UMN signs such as hyperreflexia or spasms as spasticity does [35]. Therefore, it may be difficult to differentiate these two clinical entities since patients with DOC often show lesions involving extended areas responsible for the emergence of various forms of hypertonia. In addition, spasticity may often be associated with dystonia, which are sustained abnormal postures while the subject is at rest, due to basal ganglia lesions as well [53, 54]. Thus, spasticity is not necessarily the only dysfunctional motor pattern in patients with DOC. It is therefore difficult to assess it electively since it can be occulted by rigidity and/or dystonia.

Since these patients rarely demonstrate voluntary movements, an adaptive muscle shortening occurs as a consequence of this immobilization. This maladaptation contributes to a decrease in passive muscle extensibility, and, thus, passive movement will demonstrate a resistance. If muscle shortening is not treated, a permanent loss in ROM may occur and can further lead to joint deformation. Consequently, patients may have higher difficulties to initiate a movement and thereby to demonstrate a sign of consciousness [42].

This loss in ROM will in turn lead to joint retraction, due also to many other phenomena occurring together in the involved articular structures (e.g., adherence of fibrofatty connective tissue to cartilage surfaces, atrophy of cartilage, or regional osteoporosis) [55, 56]. The most often affected joints of brain injury survivors include the elbows, wrists, hips, knees, and ankles [57] (Fig. 7.2). Two stereotypic

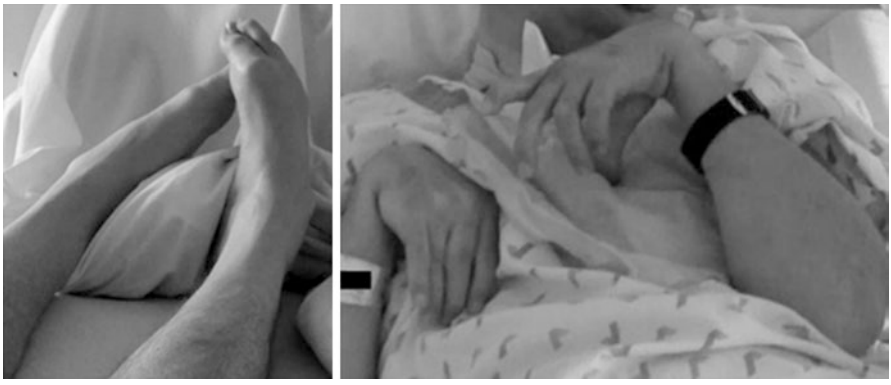

Fig. 7.2 Equinovarus feet (*left*) and decorticate spastic pattern (*right*) (from Thibaut et al. [10])

motor patterns due to different lesions' localizations are frequently observed. The first one is the decortication spastic pattern, due to subcortical lesions, which consists of a flexion of the upper limb and an extension of the lower limbs. The second one is the decerebration spastic pattern, due to brainstem lesions, which consists of an extension of both upper and lower limbs. However, the spastic patterns observed in patients with DOC are quite heterogeneous. While some suffer from spasticity in one side of the body, others have lower limbs bilaterally affected. There is no such "typical" pattern observed with patients with DOC as in poststroke hemiplegic patients (e.g., upper limb in internal rotation and adduction of the shoulder coupled with flexion of the wrist and the fingers [58]).

Muscle contractures are likely to appear especially if spasticity affects a joint in a shortened position [59]. Yet, while some authors allege that spasticity will lead to contractures [60, 61], in some patients, contractures may actually potentiate spasticity by amplifying the stretch reflex [62]. This hypothesis is based on the fact that the muscle shortening (due to the contracture) would alter the stretching effects [7]. While the thigh relationship between these two motor dysfunctions is clear, the exact interaction between them remains to be determined.

Considering the important difficulties for patients with DOC to express potential pain and their almost permanent immobile position in bed, which may increase side effects, prevention and treatment of spasticity need to be a critical part in their daily management [63], in order to reduce muscle tone, improve ROM and joint positioning, and thus facilitate the rehabilitation [26], as well as avoiding joint deformity and pain.

Patients with DOC Versus Stroke or Moderate TBI

In some clinical situations, spasticity is not problematic but may help hemiplegic patients to conserve their walking ability. Sometimes, indeed, it helps the patient to supply other weak muscles and allows to stand, to grab something, or to walk [1, 6]. Nevertheless, spasticity in patients with DOC, due to sustained immobilization [42] and lack of voluntary movements and communication, is a serious issue that needs to be managed as it is detected. Indeed, a loss in ROM is frequently observed, with various levels, in patients in UWS or MCS [64]. As mentioned above, the consequences related to spasticity (e.g., retractions, pain, and movement's limitation) may negatively impact patients' rehabilitation and quality of life. Besides, the presence of spasticity and thus the alteration of the motor function may potentially unable the patient to show subtle signs of consciousness, which will alter the diagnosis [65, 66].

One main difference between patients with DOC and patients who have suffered from a stroke is the lesion's location and extent. Indeed, while patients with stroke suffer from a typical and focal lesion, patients with DOC usually have far more extensive lesions, involving both cortical and deeper brain regions as well as subcortical areas [67]. Taking this into account, it may be difficult to

allocate some specific areas to the emergence of spasticity because of the intricacies of the lesions and the fact that some cortical lesions may be occulted by deeper regions' damages. As said above, from an anatomical point of view, lesions of the pyramidal tract are thought to be involved in the development of spasticity [68]. This neural pathway classically includes the brainstem, the cortex of the primary, secondary, and supplementary motor area, and the spinal cord. However, the pyramidal tract is far from being the only area of interest involved in the UMN syndrome which can be involved in the development of spasticity. The parapyramidal fibers have an important role as well since they pass very closely with the upper motor neurons and include inhibitory and excitatory pathways afferent on the spinal reflexes [35].

Patients suffering from a chronic stroke demonstrate a combination of spastic muscle hypertonia and an excess in muscle activity measured by EMG [69]. This can be measured by the responses to electrical (Hoffman reflex or H-reflex) or mechanical stimuli (Tendon reflex or T-reflex). The H-reflex allows assessing the alpha motoneuron's excitability and is often higher in patients presenting spasticity [10]. However, patients with DOC may not necessarily show this association of hyperactivity and hypertonia in the muscles since the EMG responses (e.g., the H-reflex) reflect the damage of focal lesions (such as in stroke) which may be occulted by more diffuse lesions (e.g., cortical and subcortical lesions) in patients with DOC. The pathophysiology of spasticity in DOC has to be further investigated, with the help of magnetic resonance imaging (MRI), for instance, in order to assess which specific structural lesions are often observed.

Regarding patients who had a spinal cord lesion, they will classically show the most severe signs of spasticity as compare to patients who had a stroke or other supraspinal lesions [16]. In the case of a spinal cord lesion, both inhibitory and excitatory pathways are abolished. Supraspinal lesion only withdraws cortical facilitation of the inhibitory pathways, which leads to mildly reduced inhibitory drive and less severe clinical signs [35]. Overall, cortical lesions observed in patients with DOC or stroke result in some degree of spasticity, hyperreflexia, and clonus, but these symptoms are less severe as compared to what is seen after a spinal cord lesion where the Wallerian degeneration after the anatomical disruption of the motor neurons leads to a major abnormal motor pattern [16, 70].

How to Treat Spasticity in Patients with DOC

While it is known that the care and rehabilitation of patients with DOC are time-demanding and can be expensive, there is still a lack of scientific evidence to guide their rehabilitation [15, 71]. Most of the time, physiotherapy treatment of spasticity includes passive range of motion and passive muscle stretch. Schmit and colleagues [72] investigated the effects on spasticity of repeated passive movements in flexion and extension of the elbow. They found out that the stretch reflex torque and EMG

responses were significantly reduced after 20–30 sequential flexion-extension movements (at constant velocity with a 10-s hold between flexion and extension) showing the potential positive impact of mobilization associated with stretching on muscle spasticity. Passive muscle stretch includes several modalities in order to hold the muscle in a lengthened position (e.g., by a therapist, a splint or orthosis, a cast, and a standing frame). Three studies showed that passive muscle stretch from 10 to 35 min seems effective on adults with cerebrovascular accident (CVA) and TBI and on children with cerebral palsy [73–75]. Short passive stretching exercises (20–60 s) in children with cerebral palsy who suffered from spasticity also seem to be effective for reducing knee flexion contractures [76]. However, according to a recent Cochrane Review, the effectiveness of stretch for the management of contractures in UWS or MCS patients is not well established yet [77].

Other studies demonstrated the effectiveness of serial casting (process of applying and removing corrective casts in succession [57]) by an increase of joint ROM and a reduction of spasticity following the placement of gradual corrective casts for 4–6 weeks, 24 h a day [78]. According to Mortenson and Eng [79], the use of casts would be the best technique to improve passive ROM. However, it should be noted that this conclusion is not shared by all the researchers [80, 81] due, in part, to the lack off randomized clinical trials on patients with CNS disorders. Besides, it is recommended to correctly position the joint, approximately 5° less than maximal endpoint of ROM, in order to avoid triggering a reflexive increase in muscle hypertonicity [57]. Yet, chronic patients with DOC usually present severe reduction of ROM. Furthermore, they often are at highest risk, as compared to other populations of patients with neurological condition, for skin problems, such as irritation or bedsores [82], and, thus, casting seems less likely to be adapted for this specific population of patients.

Many reviews and studies emphasized the lack of clinical effectiveness of passive muscle stretch [78, 83], and none of them actually suggest new rehabilitation approaches. The evidence of the efficacy of these interventions remains poor although they are used in a daily routine by caregivers since patient's collaboration is not required.

Rigid splints are widely used for patients with poststroke spasticity [84, 85] and for children with cerebral palsy [86] even if their tolerability for long periods is not optimal [32]. Patients in UWS or MCS often show severe signs of spasticity with significant contractures; therefore, they need a more appropriate way to stretch and relax their spastic muscles without risking them to be injured. A recent study [87] proposed an alternative by applying hand-rolled soft splints on the upper limb of chronic spastic patients with DOC (Fig. 7.3). The aim was to decrease spasticity, improve hand opening, and compare the effectiveness of manual stretching against soft splints on upper limb spasticity. Both manual stretching and soft splints showed positive impact on patients' spasticity, while only soft splints were able to increase hand opening, which is significant for patients' hygiene, to avoid maceration or even injuries.

As in previous studies [73, 78], the effects seemed to be transient by lasting more or less 30 min but disappeared after 60 min. The main advantages of soft splints

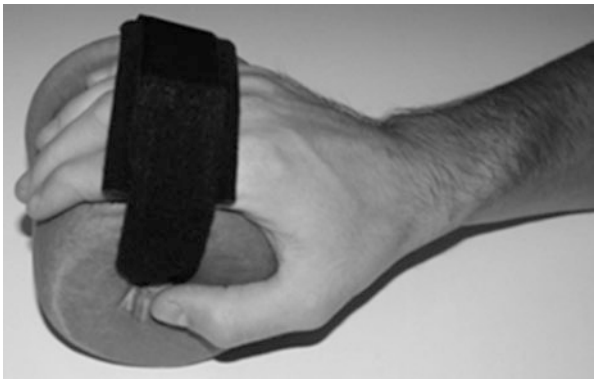

Fig. 7.3 Example of soft splint (from Thibaut et al. [87])

compared to rigid splints are their easy application and the low risk of causing pain or injuries since they are flexible and allow muscle contraction and grasping reflex. Therefore, they might be a valuable option for chronic patients with DOC suffering from upper limb spasticity.

A more invasive perspective is the botulinum toxin injection. National clinical guidelines identified the benefits of botulinum toxin with a program of stretching and physiotherapy (including splinting) for adult patients suffering from poststroke spasticity [88]. Regarding its use for patients with DOC, Belgian researchers investigated the effects of botulinum toxin type A (BTX-A) for the management of spasticity in children with an acquired brain injury [89]. One of the three subgroups consisted of young children with severe spastic quadriparesis and impaired consciousness. In this group, bilateral intramuscular BTX-A injections (~4.19 IU/kg) in the hip adductors, knee, and plantar flexors showed improvement in spasticity and ROM (average 1.75 point decrease in MAS and +7° goniometry), with the higher effects at 3 months posttreatment. The authors concluded that intramuscular BTX-A injections in combination with orthotic devices may be considered as an effective treatment to manage severe spasticity in chronic patients with severe acquired brain injuries. These results are in line with those of Yablon and colleagues [90] who showed that BTX-A significantly improves spasticity and ROM in the distal upper limb of both acute (less than 1 year after the lesion) and chronic patients (more than 1 year after the lesion) with moderate or severe TBI. However, botulinum toxin requires careful use because of its toxicity [91]. It is not manageable to perform injections in a high amount of muscles; only a few can be targeted which restricts the efficiency of this method.

Further randomized clinical trials investigating both pharmacological and non-pharmacological treatment have to be performed in this specific population of patients with chronic DOC. Naturally, there are as many clinical settings as the amount of patients suffering from spasticity. Therefore, the best clinical practice for patients with DOC is naturally individualized, multidisciplinary, and patient centered [92]. However, providing evidence-based guidelines based on randomized clinical trials is a difficult task since this population of patients is highly heterogeneous

which makes the comparison between groups very difficult. Crossover designs and single-subject designs seem to be a more appropriate option for patients with DOC [78].

Clinical Recommendations

Since the process of muscle contracture initiating muscular atrophy is acute (occurring within the first 6 h of immobilization) [42, 93], early mobilization (passive ROM) is crucial in order to avoid premature complications. Although the main objective in the intensive care unit is to keep a sufficient lung function, the motor issue should not be put aside. Actually, any type of treatment, pharmacological or non-pharmacological, should be started early, as soon as muscle overactivity is distributed diffusely and causes clinical disability, in order to prevent permanent articular deformities or muscle contractures. Afterward, even if it is often unknown in the acute setting if the patients will or not fully recover, an early post-acute care should be provided in a specialized rehabilitation center where patients can be properly assessed, and a suitable care program can be established. Further, at the chronic stage, using soft and comfortable splints to decrease spasticity should be recommended [87], in addition to the existent treatments, since patients with DOC are not able to communicate their pain feelings.

Physiotherapists will have a key role to play although little is known about the effect of intensity of physiotherapy on motor outcomes [15]. They are strongly involved in the patient's care and rehabilitation by seeing them nearly every day for respiratory physiotherapy, stretching, positioning, multisensory stimulation programs, and much more, in order to enhance the comfort and stimulate patients' arousal. Regarding spasticity in particular, physiotherapists have to position and stretch them right for several hours a day in order to manage the muscular tone, to avoid any contracture and to maintain the skin integrity [94–96]. In order to effectively improve the function and comfort of patients with DOC, the physiotherapist's interventions have to be frequent and of prolonged duration. However, the cost for the social security is high. Future studies have to clarify the minimal but sufficient amount of physiotherapy in acute and chronic situations and establish evidence-based guidelines for the spasticity management.

Naturally, a multidisciplinary approach combining physical, pharmacological, and surgical treatment interventions is needed to manage this spasticity properly [82]. Not only the therapists but the families have a significant role to play [97], and they should be encouraged to take part in the care by gently stretching, massaging, and stimulating their relatives every day, beside physical therapy or oral therapy sessions. At a prolonged chronic stage, the emphasis is more on maintaining quality of life than preserving function toward the expectation of future recovery since the chances of recovery are less likely to occur [94].

Conclusion

Patients with DOC usually need important and specific management (e.g., medical care, nursing, rehabilitation, and speech therapy). They may require up to 7 h of care per day due to their (almost) entire dependency [98]. As said above, spasticity concerns a majority of these patients and does not only induce pain but may also lead to severe orthopedic deformities which increase the care difficulty. In addition to the induction of pain, ankylosis, and muscle weakness, the presence of spasticity and thus the alteration of the motor function may not only interfere with rehabilitation but potentially unable the patient to show several signs of consciousness as well, which can lead to misdiagnosis [99]. Appropriate treatment and careful follow-up are thus required in order to enhance patients' daily life. Over time, spasticity management may become more comfort care and less curative, but it has to stay in the medical care priorities.

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