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1. Introduction

Contractures limit free joint movement and are a common consequence of traumatic brain injury. They interfere with activities of daily living and can cause pain, pressure areas, and result in unsightly deformities [1 - 4], affecting patient quality of life and increasing institutionalization rates. Contractures also cause significant secondary impairment which ultimately interferes with the rehabilitation process. Their treatment is therefore an integral part of physical recovery. Before effective intervention can take place, therapists must first determine both the primary cause as well as the specific structures involved. Several different therapeutic modalities exist to treat them, and choice of which to apply will depend on each individual case [5].

The aim of this chapter therefore is to review currently available physical therapy techniques for the treatment of contractures and for prevention of deformity development, in subjects suffering traumatic brain injury (TBI).

2. Generalities

Contractures are a common complication of traumatic brain injury and may occur in up to 84% of cases [4, 6]. The most commonly affected joints are: the hip, shoulder, ankle, elbow and knee, with a significant percentage of patients developing contractures in five or more joints [4].
For purposes of this chapter, we define contracture as any degree of loss in joint range of motion restricting activities of daily living[4]. Movement restriction is not limited only to joints, but will also affect many other body structures including skin, subcutaneous tissue, muscles, tendons, ligaments, joint capsules, vessels and nerves [7].

Contractures are characterized by reduced range of motion (ROM) and increased stiffness. The increased resistance to stretch caused by changes in the mechanical properties of tissues is due to both neurally and non-neurally mediated factors [8, 9]. Non-neural factors include changes in mechanical properties of tissue resulting from stress deprivation, and may be secondary to orthopedic injury, heterotopic ossification, use of a splint or plaster, pain, paralysis, severe spasticity or any disorder that restricts movement. [10]. Contractures also produce structural changes within muscles; myofibril shortening and loss of sarcomeres are often observed, as well as relative increase in connective tissue causing loss of elasticity.

Neural factors are of central origin and cause muscle overactivity. They generate spasticity, increasing interdigitation between actin and myosin, thus reducing muscular concentric contraction range and producing rigidity, as a result of the absence of monosynaptic reflex inhibition [9, 11].

3. Evaluation and diagnosis

Evaluation of patients with head trauma includes examination of joint range of motion, looking for possible contractures. Prior to physical exam, it is important to record general patient posture (decortication, decerebration, etc.) and clinical condition, and especially to register any hyperexcitability such as spasms or clonus triggered by intense stimulation in patients with sensory impairment.

Joints should be observed at rest in order to establish whether muscle overactivity or flaccidity is present; next, voluntary movement should be examined, and presence of dystonia when changing positions recorded, if elicited [12]. These findings are important because contracture development in patients with TBI is related to presence of spasticity and/or dystonia[13]. Joint examination is performed systematically, mobilizing each joint manually and passively. In patients able to follow verbal commands and perform active movements, it is important to encourage them to move each segment to the widest possible range.

Once manual examination has been completed, findings in individual joints should be recorded including quality of resistance and end feel, which refers to examiner perception of a barrier to further motion when exerting passive ROM. For better diagnosis or evaluation, passive range of motion may be measured using an instrument such as a goniometer or applying other objective methods. The universal goniometer (UG) is simple and easy-to-use. Result reliability is acceptable, but only after appropriate user training and always applying standardized measuring methods [14, 15].
When loss of range of motion is detected, differential diagnosis needs to be established between lack of range due to motor deficit (muscle overactivity) or resulting from structural alterations. Clinical assessment of hypertonia can be conducted using a well-established rating scale, the Modified Ashworth Scale. However therapists should be aware that this scale does not distinguish between soft tissue and neural contributions to hypertonia [16]. To make this differential diagnosis, the contracture segments need to be re-mobilized at different speeds in order to unmask muscle overactivity, which if present, can be rated using the Modified Tardieu Scale. This scale is widely used because it renders both quantitative and qualitative measurement of spasticity. Its validity has been studied and has demonstrated high intra and inter-rater reliability when using a standardized protocol. It is highly reliable for assessing spasticity in hamstrings, rectus femoris, gastrocnemius, soleus and tibialis anterior muscles in adults with neurological injury. It has also shown very good intra rater reliability for elbow flexors [17 - 18].

Other important factors that need to be considered in the individual patient and factored into the therapeutic decision-making process include: periods of prolonged bedrest, presence of subcortical lesions, and decreased levels of awareness or lack of voluntary activity in the presence of muscle overactivity, as they are all indicators of greater lesional complexity. Yarkony et al. postulate contracture as clinically evident when coma extends beyond three weeks [4]. Severity of contracture is also generally more pronounced in patients with brainstem lesions and treatment usually begins later and will require more time to achieve gains in ROM [19].

Therefore, one can expect coma patients to suffer greater structural changes in muscle tissues both because of worse neurological status as well as absence of voluntary movement countering effects of immobility.

Motor control and presence of spasticity are important. For example, if no underlying motor control is present and spasticity is not reduced after contracture treatment, probability of sustaining improvement is poor [19]. In contrast, presence of voluntary motor control in muscles antagonist to the contracture, increase the likelihood of maintaining any ROM gain [20].

Cognitive impairment will also influence rehabilitation therapy choice. Sometimes, cognitive impairment may interfere with patient ability to cooperate and interpret pain [19].

Similarly, patients presenting preserved cognitive status, voluntary motor control and minimal or no muscle overactivity, will attain better treatment outcomes.

Once a thorough evaluation has been completed, a treatment plan can be individually designed and the most appropriate technique chosen to treat or prevent contracture. Treatment should be specifically adapted to each patient and will require constant reassessment in light of changes occurring in the course of recovering from head injuries. See table 1.
4. Therapeutic modalities

Stretch is one of the most widely used techniques for treatment and prevention of contractures. Its aim is to increase joint mobility and it can be self-administered or applied manually by therapists. Splints, positioning programs or casts changed at regular intervals (serial casting) can also be used. All methods involve mechanical elongation of soft tissues during varying lengths of time. Some can only be applied for short periods, such as manually applied elongations, performed for only a few minutes at a time. Others, such as splints and plasters are used to stretch muscles for longer periods, and sometimes to provide uninterrupted elongation for days or even weeks.

5. Stretching

Research in physiotherapy and neurophysiology has led to the development of some alternatives to passive stretching. Cherry[10] described four approaches to reduce contractures,
namely: activating or strengthening the weak agonist, local inhibition, general inhibition and passive lengthening, some of which may be used simultaneously.

The first approach involves activating or strengthening the weak agonist opposing the tight muscle. If tissue innervation is intact and the agonist has the ability to function at all, a double benefit will be gained by improving its ability to contract. If the agonist becomes stronger, it will be able to counter the contracture of the antagonist and pull the joint through more complete range. Also, the contracted muscle (antagonist) will be reciprocally inhibited, allowing itself to be stretched because the stretch reflex is also inhibited.

If the weak agonist can be activated and strengthened, better muscle balance around the joint may result, reducing the potential for myostatic contracture recurrence.

Selection of a technique to strengthen the weak muscle will depend on the nature of the problem and the ability of the patient to cooperate. Effective strengthening methods that employ resistance or load on muscle include maximal resistance in diagonal spiral patterns and progressive resistance exercises. Methods that use unconscious automatic responses to activate the weak agonist require eliciting righting reactions and equilibrium responses.

The second approach may be used when an agonist is unable to contract at all, or the antagonist muscle may be so tight that attempts to strengthen the agonist fail. Also, even if the agonist can be activated, it will move through more complete range of motion if the stretch reflex of the tight antagonist can be inhibited. In this approach, inhibition of the tight muscle is considered. The proprioceptive neuromuscular facilitation techniques, such as contraction-relaxation or hold-relax may be used to inhibit the tight muscle selectively, so it will tolerate being stretched without immediate activation of the stretch reflex.

Local inhibition is useful for localized tightness, especially within one muscle group at a single joint, such as after plaster immobilization following injury or surgery. Techniques generating inhibition to the contracted muscle include vibration of the opposite muscle group, prolonged icing and hold-relax, used in traditional rehabilitation.

A third approach to reduce hypertonus, in a single limb or throughout the whole body, consists in allowing tight spastic muscle groups to relax and be lengthened. Some concepts as neurodevelopmental treatment (NDT) consider that movement control is achieved in an integrated way when the nervous system works cooperatively. For example the organization of postural systems require interaction between external forces (gravity), body mechanics and kinetics, multisensory inputs, and adaptative responses to voluntary movements. Therefore, inhibition of hypertonus is best when the patient participates actively.

The fourth approach is passive lengthening. If passive lengthening is selected as the appropriate alternative, there are two techniques that may be applied. One involves manual passive stretching and the other prolonged holding of the desired position at the point of maximum tolerated length of the contracted muscle.

Passive stretching is likely to be most effective in individuals whose stretch reflex is inhibited by cortical effect or by peripheral nerve injury.
Adaptative equipment may enable an individual to function more readily in certain positions, for example use of a prone standing board for hip and knee extension, to align lower limb joints in weight bearing patients with lower limb flexor problems. Also, positions for sitting, sleeping, and other daily activities, useful in correcting contracture, can be adopted and held for prolonged periods.

Other techniques of passive lengthening include standing, orthosis, splints and casts with prolonged holding. All require close monitoring (see below). Generally, patients with head trauma present severe injuries. It is very common to find extremely weak agonist muscles to work with, or to attempt to generate antagonist tone inhibition on. When these techniques are employed it is important to consider that any increase in range obtained by forced motion will be lost unless it is maintained by active motion or by use of supportive devices.

6. Standing

TBI patients can be supported while standing using a tilt table, standing frame and/or knee extension splints. Regular standing in the correct position will maintain sufficient range of dorsal flexion of the ankle joint for walking when the patient reaches that stage but, if he has not been made to get out of bed from the beginning; his Achilles tendon may have already shortened [21]. In our experience, the first option for patients with more severe motor deficits or disorders of consciousness is the tilt table. Special attention must be paid to the knee and ankle. If the knee has full range, it should be positioned in slight flexion or in a neutral stance. The ankle must be able to bear sufficient weight on the rearfoot without causing tissue damage or increasing muscle overactivity.

The second option is the standing frame. Patient can be supported in upright position by a solid frame with padded struts in front of his knees to keep them extended and a broad strap behind his hips to prevent them from flexing. Using the standing frame may be useful when the patient has regained consciousness and is able to extend his trunk actively, because it is then possible for him to stand for longer periods.

The third option is standing assisted by the therapist, recommended for patients with better functional status and trunk control. If necessary, extension knee splints can be used to prevent lower limbs from giving way due to lack of motor activity. Knee extension splints should reach from about 8 cm below the ischial tuberosity to 4 cm above the malleoli. Made of a firm posterior shell of plaster of Paris or some other hard material, knee-extension splints will need to be bandaged in place with two 10 cm wide crepe bandages to give adequate support and maintain optimal leg positioning. Patient should be lying in supine position while therapist bandages on the splints, to be able to correct any inward or outward rotation of the limb [21].

When the patient has marked spasticity in the plantar flexors of the foot, standing upright is often the only way in which therapists can maintain range of ankle dorsiflexion and prevent shortening of the Achilles tendon [21].
In case of equinus deformity that does not allow the heel to contact the ground, a posting on the foot, AFO (ankle-foot orthosis) or shoe is needed. Posting entails construction of an interface between the aligned foot and the ground to optimize heel loading and hip and knee alignment while standing or walking (filling in the space under the heel when the ankle is positioned in plantarflexion) [22].

When standing the patient up, one must consider alignment of the pelvis in the frontal plane, since use of a post will be shorten contra-lateral limb length. This must be compensated with an additional pad under the contra-lateral heel, high enough to correct pelvic alignment.

There is evidence to support placing patients in the standing position to prevent loss of calf muscle length. In severe brain trauma cases where little functional recovery is expected, maintaining patient physical condition is challenging. In this context, long term effects of supported standing to maintain muscle length appear more important. Although often prescribed, effectiveness of standing programmes carried out over months or even years remains unknown. Standing may prevent small losses of ankle dorsiflexion, but clinical importance of these effects is uncertain. Future studies should investigate standing in a wider range of settings. Evaluating potential multidimensional effects of standing using standardized measures would provide greater insight and be more effective than studies focused on a single outcome [23].

7. Splinting – Orthosis

Use of splinting for both prevention and reduction of contractures is recommended following traumatic brain injury [24]. Alternatively, orthosis also reduces contractures through prolonged low-load stretch, maintaining joints lengthened [25]. Therapists who apply a biomechanical treatment rationale recommend splinting both to prevent as well as manage length-associated changes in muscles and connective tissue.

7.1. Types of splints and outcome

Splints of various forms, often combined with other passive and/or active tissue ‘stretching’ procedures (e.g. passive movements, positioning, weight-bearing), are the treatment of choice for physiotherapists. General goals of therapy seek to inhibit/reduce increased muscle tone and/or elongate shortened soft tissues. In the case of some splints, an additional goal may be improving/maintaining appropriate biomechanical limb positioning during (later) functional activity retraining, such as walking [24].

Elongation effects obtained can be sustained by using splints overnight. Advantages to wearing a night splint are: overnight intervention which allows therapy time to be spent on active retraining of everyday tasks, ease of application, and perhaps most importantly, continued long term use after discharge from hospital. The main disadvantage is the risk of pressure sores, particularly when patients have poor vascular supply and/or sensation in the affected areas [26]. It is important for the therapist to monitor splint use with the interdisci-
plinary team. When indicating a night orthosis, patient tolerance and adherence to use should be closely controlled. In the presence of adverse effects the splint should be removed immediately and its indication reassessed.

A published case report illustrating lower limb orthosis implementation described use of a static adjustable ankle orthosis, placed after administration of a phenol nerve block in conjunction with stretching, strengthening and functional mobility training. The adjustable orthosis was applied to provide low-load prolonged stretch of the ankle and address apparent soft tissue shortening, and the phenol nerve block was administered to address ROM limitations secondary to muscle spasm [27]. Positive results reported in this case would support orthosis as an addition to standard physical therapy stretching regimens in patients with brain injury.

Other published evidence indicates that overnight splinting of an affected ankle in subjects with stroke appears to be as effective as standing on a tilt table in preventing contracture at the ankle [26].

In our experience an alternative intervention to prevent ankle contractures is use of an overnight splint. Although the ankle is not usually positioned in maximum dorsiflexion, the extra time spent in this position helps avoid contracture development.

Indication for upper limb splint placement is common, mostly at rest to keep the joint in extended position, thus halting further shortening.

According to Lannin [27], night splinting the hand in the functional resting position does not produce clinically useful effects in adults with acquired brain impairment who receive a daily stretching programme. In his discussion, the author postulates absence of effect after splint use may have been because routine motor training and upper limb stretch were maintaining muscle length. Therefore, additional stretching provided by the night splint may have been redundant.

Sometimes, therapists indicate additional electric stimulation and splinting for contracture management, but it is not clear if it is more effective than splinting alone after acquired brain injury [28].

### 7.2. Evidence and conclusion

There is insufficient evidence to either support or refute effectiveness of orthotic devices for contracture treatment or prevention. Further research is needed for better understanding of the influence of elongation on soft tissue, as well as with respect to timing, length of application and efficacy of splinting programmes. In conclusion, splinting options should be carefully analyzed in TBI patients as they may represent a clinically effective strategy when used appropriately.

### 8. Casting

Casts are a viable option for treating contractures after upper motor neuron injury in adults [19, 30]. These are non-removable external devices, made of plaster or casting tape, applied
with intention to change structural or functional characteristics of the neuromuscular system [31]. Casting to control hypertonus (understood as increased resistance to passive movement as a result of spasticity and/or changes in muscular and connective tissue characteristics) was first described in the 1960’s in the treatment of children with cerebral palsy [20, 32]. Later, casts were applied to adults with acquired brain injury with different therapeutic objectives. These goals include preventing loss of joint range, helping to cure pressure ulcers associated with severe spasticity, restoration of articular range (ROM) and muscle length, and inhibition of hyperexcitability [20].

Different theories have been proposed to explain underlying neurophysiological and mechanical mechanisms behind the positive effects of plasters, on hyperexcitability reflexes and mechanical changes [32].

The first hypothesis relates to neurophysiology. Plasters prevent changes in muscle length by eliminating excitatory input to muscle receptors, which in turn reduces spasticity. Prolonged elongation stimulates Golgi tendon organs and subsequently afferent Ib fibers, creating an inhibitory response in alpha motor neurons. Type II muscle afferents have also been postulated as inhibitors of alpha motor neurons following prolonged elongation [32]. Casts may also reduce spastic muscle tone through alleviation or reduction of tactile input, proprioceptive input, and temperature receptors. Uninterrupted contact provided by the cast, including the neutral pressure and heat generated, reduces excitability of alpha and gamma motor neurons at the spinal cord level [30 - 32].

A second hypothesis is based on biomechanics. It postulates that casts achieve low load elongation during prolonged periods, able to prevent and correct contractures. Casts are usually applied at the end of joint range of motion because prolonged stretch with low load, generating permanent changes in soft tissues. It has been well established that with prolonged immobilization, specific physiologic changes occur involving connective tissue and muscle remodeling. This remodeling is mediated by fibroblasts in response to physical forces. When a muscle is immobilized in a stretched position, the number and length of sarcomeres in series increases, thus changing the muscle tension / length ratio. Connective tissue is also extended through a process of disorganization of the fibrous matrix [30].

The third hypothesis refers to motor learning. It proposes that casts provide adequate support to proximal joints, until patient gains enough distal control [31]. The external stability provided by the cast to the limb is presumed to allow the patient to receive normal sensory input with appropriate weight bearing and normal reflex patterns, helping to develop normal movement and accommodation in the central nervous system [32].

8.1. Types of casts

Different casts used in TBI patient rehabilitation and described in the literature include: serial casts, inhibitive cast, drop out casts and bivalved casts [30]. Specific types of cast are important in treating contractures and indications will depend on patient characteristics and level of functional recovery.
Serial casting involves application and removal of a series of casts resulting in progressive range of motion increase with the introduction of each cast. Serial casting allows 24 hour a day elongation, with casts changed regularly to maintain gain as the joint becomes more mobile [33].

Inhibition cast. The purpose of an inhibitory cast application is to maintain a position, to reduce spasticity and facilitate improvements in motor function. Inhibitory casts seek to provide stability and inhibition to the treated joint. The inhibition may be achieved by normalizing proprioceptive input, joint alignment, and weight load. Effects of these casts do not seem to last long after they are removed; for this reason, inhibitory casts resulting in a positive impact on functional performance need to be followed by use of an inhibitory splint during a prolonged period [30].

Functional drop out casts are the combination of serial casting and inhibitory casts. When making a functional cast, a portion of a cylindrical cast is removed to allow the involved joint to move beyond the desired range, preventing the joint from pushing back toward the contracted position. This allows passive or active movement in the desired direction and allows the user to gain additional active range while using a cast. These casts also allow application of electrical stimulation or other facilitation techniques [30].

Bivalved casts are casts cut in two halves (front and back), which are then filled and padded at the edges to allow reapplication. Usually this is done when the patient has achieved the desired range and needs to maintain the new position. This is required when tone in the involved limb remains high and there is doubt whether a traditional splint will resist and keep the muscle in the desired position. Bivalved casts provide full contact and have the advantage that they can be removed for hygiene, inspection, active movement, and other dynamic activities [30].

The main indications for use of any type of plaster in general, and of serial casting in particular, is permanent limitation of range of motion [19], or immobility during muscle function only. If other disorders such as muscle weakness or overactivity of some kind are present, these will influence both plastering technique as well as length of treatment.

8.2. Application procedure

The cast-making process follows a basic preparation which adapts to any plastering technique, and consists first in the placement of soft materials such stockinette, foam and cast padding, to protect the skin and bony prominences and avoid friction and pressure points, followed by application of a plaster bandage (fiberglass and/or plaster cast) to achieve the necessary hardness to fix the limb in the desired position.

There two basic objectives sought after by the procedure: to place tension on the muscle and maintain the pressure during a pre established time period.

Tension levels will vary depending on which theory the therapy is based on. If we consider the biomechanics theory, plasters are applied on final range in order to gain maximum length. Neurophysiology-based theory considers tension levels should stretch the joint to within 5
degrees of final existing range. Variability in studies on angle used to make the cast range from 5° and 10° under full range, to neutral, to end of available range. This last option is the one with higher levels of evidence to support it [31]. In our experience, we prefer to use end of range for plaster application. The force applied to elongate the muscle is usually limited by patient pain threshold [31].

With respect to duration of application, it is highly variable, and will depend on the treatment objective and on how much time is needed to achieve the desired effect. There is significant discrepancy in the literature over total length of treatment between published studies [30]. Some publications suggest shorter implementation generates fewer complications and similar results [31].

Significant variation also exists in relation to length of time between cast changes. Duration of casting generally should reflect the pace at which the individual is making progress and the goals of the casting. Tardieu and Tardieu recommend lengthening via casting should be very gradual because careless application may result in muscle fiber break down, but they do not give specific time frames or guidelines. If the patient is experiencing slower progress or is at lower risk for breakdown or complications, casts can be left on longer between changes; this is particularly true if a cast is difficult to apply because of time constraints, cost, patient agitation, or patient need for medication during the switch. If the individual seems to be progressing rapidly or is at increased risk for skin breakdown, if the cast is damaged or wet, or if there is any other concern, it should be changed more frequently [30].

In a serial casting program, we start by placing an initial plaster at rest. This is applied with the extremity positioned at the end of the movement range, but easily reached without applying additional tension. Generally, we use 3 or 4 progressive casts [19]. Some authors such as Davies recommend 6 changes on average [21].

Authors suggest mobilizing passively through the entire range of motion available to maintain full mobility of all immobilized joints and then apply a new cast at a greater angle [23]. In contrast, Davies [21] does not mobilize in between casts, to avoid any chance on losing the range gained. Currently, there is no agreement on whether the extremity should or should not be mobilized between each change of plaster.

The last cast in the series is the supporting or positioning cast. It is cut in two halves making it bivalved, like an anterior-posterior splint [19]. This splint is used in the resting position for as long as deemed necessary until the patient improves and gradually stops using the equipment [19, 20]. If the patient’s muscles are still weak and present overactivity, it is important to continue using the device overnight [19].

Cast progression should be discontinued when there is no further gain of range. Although there is no consensus between authors on this point, some suggest that progression can be interrupted if no quantifiable gain is achieved after two consecutive applications. Others suggest stopping if there is no measurable gain (<5° measured with goniometer) in maximum range after three consecutive plaster changes. [20] If the team decides to end the application, it is important the patient be examined by a physician to consider the possibility of orthopedic surgery [20].
Therefore, application, duration and frequency of change will need to be individualized, defining for each protocol: timing, whether it will be a single plaster or several, how often casts will be changed, when joint range will be progressively increased and finally, total duration of single or serial casting [31].

8.3. Precautions, complications and contraindications

There are a number of precautions to consider before placing a cast. With respect to the patient it is important to take into account skin integrity, presence of fluctuating edema, decreased sensitivity, cognitive impairment and agitation [19, 30]. Therapist experience will contribute to establish level of existing complications and whether these can be managed and still continue with the cast. For some authors contracture treatment has priority over any skin condition, because they argue that it can help heal skin lesions [21]. Fluctuating edema and sensitivity disorders can be managed with frequent controls after application and if necessary the plaster may be removed at any time.

In cases of cognitive disorders and agitation, sedation or use of restraints have been described in the literature, so that the patient does not hurt himself or others during the placing of the plaster and is careful while wearing it [21].

Casting contraindications include: uncontrolled hypertension and/or elevated intracranial pressure, open wounds, external fixation or unresolved fractures, ligament injuries, need for access to check vital signs, recent episodes of autonomic dysreflexia, circulatory disorders such as deep vein thrombosis (DVT), acute inflammation, heterotopic ossification [19, 30, 33, 34], tone fluctuation or any unstable medical condition [31]. Some authors also exclude pregnant patients [34].

Adverse effects and/or complications should be considered. Close monitoring, assessing sensitivity, motion, blood flow, skin indemnity and the presence of inflammation is always recommended, checking for presence of vasoconstriction or discoloration of fingers or toes [33]. Temporary discoloration is quite common, but if it persists for more than 20 or 30 seconds, plaster should be removed and a new one applied [19]. It should be noted that patients often report less adverse effects than therapists [33].

In a study by Moseley, adverse effects reported included skin irritation, skin breakdown, pain, inflammation and dysautonomic events [31]. Inflammation and pain can be relieved by limb elevation, applying plaster with less tension and use of analgesia. Irritation and breakdown of skin are serious complications, and often require discontinuation. To avoid complications, patient selection is important together with careful monitor and regular plaster change (once to twice weekly)[33]. If patient develops pain cast may also need to be changed.

8.4. Complementary treatment

Casts are never applied as isolated intervention. Patients who are treated with casts usually also participate in multidisciplinary rehabilitation programs [33].
Techniques complementing treatment with casts include: mobilization, stretching, electrical stimulation, neurodynamics[21], mobility and strengthening exercises and any neuro-rehabilitation techniques such as NDT or PNF (propioceptive neuromuscular facilitation).

It is important to actively or passively mobilize joints adjacent to the cast as a routine practice, as many times a day as the therapist deems necessary.

Electrostimulation is mostly used with dropout techniques, and once the plaster is cut and bivalved can also be used to prevent atrophy or enhance voluntary motor activity.

Exercise plans are designed for each patient, to stimulate activity in paralyzed muscles and encourage voluntary motor control improvement [33].

For upper limb casts we recommend simultaneous exercises such as weight bearing activities, bilateral functional skill development, motor control training on muscles on the periphery of the cast, and voluntary isometric contraction of muscles included in the cast which can be contracted.

For lower limb casts, repetitive exercises are indicated to activate weak muscles in task-related training and improve strength and coordination. It is important that patients be helped to stand with supporting equipment, which will vary depending on motor reserve (tilt table, standing frame, or assisted by the therapist). In this manner patient is forced to use the affected limb. If the patient retains a good level of standing balance, exercises involving weight bearing on the affected limb can be considered, while other limbs perform swings or reaching for objects in the case of the upper extremity.

For subjects who respond minimally, treatment may focus on following simple commands to induce muscle activity in addition to the passive modalities mentioned before [33].

Physical therapists must consider a program of stretching, positioning and indication of night splints, or even use of splints during the day after removal of final cast [31].

Maintaining range gained with cast progression is difficult when muscle overactivity persists as a result of CNS injury and lack of motor control, or of voluntary activity in muscles antagonist to the contracture.

Another common combination is the application of progressive casts with botulinum toxin for muscle overactivity management. Toxin is often used as first line drug therapy for focal spasticity [34]. However, authors disagree on whether toxin use promotes or enhances cast effect [35]. One study attempted to determine whether serial casting combined with botulinum toxin reduced contracture development in calf muscles after severe head injury, and concluded cast alone was sufficient [34].

In the presence of severe or persistent dystonia pharmacological intervention may be required. Neurosurgery is another option to maintain gains obtained with serial casting [20].

The effectiveness of plasters has been compared to that of other therapeutic modalities in the TBI population. According to Moseley, casts are more effective in the short term than positioning for one hour a day to reduce elbow flexion contracture in patients with TBI, observing
that treatment difference was not maintained over time [33]. Therefore, although serial casting is effective in reducing contracture deformity after acquired brain injury, and serial casting induces transient increase in range of motion, these effects are not maintained longterm [33].

8.5. Evidence and conclusion

There is insufficient evidence to support or refute the effectiveness of plasters in the upper limbs after an acquired brain injury. Although more evidence has been published for the lower limbs, systematic reviews on the use of casts also conclude there is lack of strong and consistent evidence to support their use in the lower limb [31].

Casts to improve passive joint range have a grade B level of recommendation, and for treatment of spasticity, a grade C. There is also lack of firm evidence therefore to recommend their use to improve function [32]. The development of clinical practice guidelines is limited because most studies are case series. One of the major limitations of research is the method selected for measuring results, very few studies use ROM measurement with controlled torque. For all the reasons mentioned above, authors disagree on when to prescribe casts, and on which approach to use and what underlying theory supports the indication. Currently, decisions on whether to use casts in patients with motor disorders due to CNS injury are based more on clinical judgment than on scientific evidence [31].

Finally, casts should be placed by experienced therapists in patients at early stages of their rehabilitation program. Associated complication rates will decrease as therapists get better at using the technique [19, 30, 34].

This treatment modality should always be part of a global and comprehensive rehabilitation program with a multifactorial vision of patient’s deficits, in particular motor control, in order to maximize functional recovery [19, 30].

9. Case studies

Case 1: Patient with Traumatic Brain Injury and Bilateral Equinus

MC, a 47 year old male diagnosed with multiple trauma and severe TBI after a motor vehicle accident was admitted to the rehabilitation center 137 days post event.

Patient was conscious, oriented in time and space and able to communicate effectively. Physical examination showed mild quadriparesis and loss of trunk control. Superficial and deep sensitivity was preserved with generalized hyperreflexia and normal muscle tone.

Passive mobilization of the soleous and calf showed tension, shortening was observed in both lower limbs, and plantar flexion and contracture of both ankles detected. No signs of muscle overactivity (under the modified Tardieu scale) were found, ruling out presence of spasticity. Voluntary movement was also present in both ankles and feet.
Goniometric measurement with knees extended indicated a right ankle limitation of -24° to reach neutral right-angle position and -34° on the left ankle. Ankles remained in plantar flexion even during weight-bearing while standing.

Given the absence of spasticity and the limited range of motion in both ankles, as a result of prolonged immobilization during the acute stage of treatment for multiple complications, the therapeutic team interpreted patient contracture responded to non-neural factors (muscle shortening, structural changes in connective tissue) and implemented the following therapeutic strategies:

- Daily stretching in the standing position for 40-50 minutes, first on the tilt table and then using a standing frame.

- Electrical muscle stimulation (EMS) on gastronemius-soleus and pretibial muscles as adjunctive treatment, in order to generate elongation by reciprocal inhibition of both muscle groups, this was implemented while patient was standing and for 20 minutes in each leg.

- Manual passive stretching.

Patient quickly recovered skills such as sitting and standing independently, so the team decided to apply four serial casts (Fig. 1). These were changed every 7 to 14 days for a total program duration of two months.

Figure 1. Independent standing with the first of the series of four casts.
Patient was allowed to rest for 2 days between casts, during which time he continued to stand using the standing frame with therapist assistance and received electro-therapy (Fig. 2).

While wearing the casts, patient performed lower limb weight-bearing exercises, balance and gait rehabilitation, with and without walking aids.

Serial cast result was positive with a ROM increase of -4° in the right ankle dorsi-flexion limitation, and of -10° in left ankle. Functional improvement was also achieved and patient was able to stand and walk unassisted, although use of a posting on both heels was required to compensate limited ankle range of motion (Fig. 3).

Case 2: Young adult with Traumatic Brain Injury and Multiple Contractures

CT, a 27 year-old man who suffered a traumatic brain injury as a result of a motor vehicle accident was referred to our rehabilitation center 53 days later. Imaging studies showed frontal and occipital hemorrhagic contusions had been treated with biparietal cranioplasty.

At time of admission patient was tracheostomized, evaluation of cognitive functioning showed level 3 function according to the Rancho Los Amigos Scale (localized response) and a score of 9/23 on the revised Coma Recovery Scale with visual pursuit, indicating a minimally conscious state. During physical examination, flexed global body posture and spastic flexor pattern was observed in all four limbs, with marked limitation in passive ROM and multiple contractures.

Goniometric measurement of right elbow movement indicated limitation in free range of motion from 10° to full flexion and in left elbow from 70° to full flexion. ROM could not been measured on wrists or hands because patient experienced pain during passive examination.
Right wrist was extended and fingers flexed. Left wrist was in extreme flexion with metacarpophalangeal (MCP) joints in extension.

In the right lower limb severe hip limitation was present with free range of motion of 30° (from 60° to 90° of flexion), knee had short range of motion, only from 120° to full flexion and ankle presented plantar flexion with a 30° angle to reach right-angle neutral position.

In the left lower limb, hip presented severe contracture with range of motion limited to 45° (from 45° to 90° of flexion), knee had reduced range from 130° to full flexion and ankle did not reach the neutral position of (plantar flexion of 40°).

Muscle tone evaluated with the modified Ashworth scale showed a score of 3/4 for elbow flexors and hamstrings, although examination was difficult because of great lost of ROM and presence of heterotopic ossification in the right hip. Voluntary movement was present only in the right shoulder. Patient was admitted to the coma program to increase his level of awareness and receive multi-sensorial treatment (auditory, visual, etc.) to stimulate voluntary motor responses.

To manage contractures we developed the following treatment plan:

- Patient positioning, both in wheelchair and in bed. In the supine position a triangular foam cushion was used to hold legs in maximum possible knee extension and to prevent hip rotation (Fig. 4).
Figure 4. Positioning on bed.

- Manual passive stretching,
- Daily stretching postures achieved through use of the tilt-table, and prone positioning with adaptative equipment because of lack of range in both cases (Fig. 5).
- Knee extension splints were applied for night positioning and standing during therapy.

After two months, patient emerged from minimal consciousness and started a rehabilitation programme. Functional status showed trunk control with independent sitting and voluntary movement in lower limbs performed by imitation and procedural activities.

At this stage, patient muscle contractures were reassessed and a Baclofen pump test performed because of generalized muscle overactivity, with negative results. No changes were observed before or after the test. Given the favorable motor recovery a serial casting program was started for both knees, particularly because absence of spasticity and presence of voluntary movement favored a good outcome.

- Three progressive plasters were applied (changed every 4 to 6 days) on his left knee. The last cast was then bivalved. Initial goniometric measurement for the joint was 65º of flexion, and after final cast reached 40º of flexion. For right knee, initial measurement was 95º of flexion and reached 40º of flexion after six serial casts (lasting on average 6-8 days each). The fourth cast was a dynamic drop out cast. Three months after applying the last plaster, new ROM measurements showed further improvement, attaining 35º of flexion.
- During cast application, patient continued standing and carrying out prone activities. During the drop-out, he also worked on passive and active knee extension (Fig. 6 y 7).
Electric stimulation was applied to the right quadriceps.

The cast allowed sufficient range to the knees to permit activity performance while standing, training of balance and for assisted walking with armrest support walker. Patient continued rehabilitation with favorable outcome (Fig. 8).

Figure 5. Standing on tilt table with adaptative equipment.

Figure 6. Prone position and exercise with Drop out cast in left knee.
The right hip range limitation was solved surgically several months later. Currently, patient walks independently, requiring supervision for cognitive deficit. As for the upper limbs, right elbow regained normal mobility with conventional therapy and left elbow and wrist flexors required a tendon lengthening surgery.
10. Conclusion

The high prevalence of contractures in patients with head trauma limits functional therapy and presence of multiple complications often increases difficulty of rehabilitation implementation.

Contractures originate as a result of both neurologic disorders specific to central nervous system injuries and of non-neural disorders. These factors alter many anatomical structures that contribute to joint range loss.

While neurologic patients are always included in a comprehensive rehabilitation program, treatment of contractures requires special attention. The goals of the latter are to improve patient quality of life, reduce hospital stay and secondary complications, and promote better mobility and alignment, in order to recover maximum functional potential.

Several physiotherapy procedures exist for specific treatment of contractures, including stretching, positioning, splints and casts. All of them, to greater or lesser degree, involve placing anatomical structures in a position that respects or maintains normal joint range with varying duration of application. The techniques are usually combined with other therapeutic interventions to improve neurological dysfunction in its different presentations (weakness, paresis or paralysis and muscle overactivity). The variety of techniques available for treatment of contractures challenges physical therapists to gain better understanding of the principles on which the techniques are based and to develop skills in their application.

No single method renders final or full solution to contractures and all need further research to establish which procedures are most effective. All existing possibilities are commonly used in any rehabilitation program and represent the current tools until new procedures are developed. Research may also lead to the development of entirely new treatment modalities.

It is very important to address contracture management through a multidisciplinary team approach, and to train therapists for early detection as well as to assess degree of contracture. Treatment of contractures remains an active process conducted under constant monitoring, where team members can make use of different therapeutic procedures at appropriate times and adapt them to changes in clinical responses of each particular patient.

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