Chapter

Spasticity: Diagnosis and Treatment

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Abstract

This chapter presents the technology of spasticity treatment—from diagnosis and treatment to quality control of treatment and rehabilitation. The diagnosis is based on methods of manual testing and differential diagnosis of spastic muscles, methods of quantitative assessment of spasticity on the basis of the Tardieu scale. The methodical development of the Tardieu scale with variants of its full and reduced use is presented. The basic patterns of spasticity of the upper and lower limbs are given. Schemes of management of patients with spasticity with indication of control points for application of methods of an assessment that shows efficiency of treatment and rehabilitation are presented. The methodology of spasticity treatment using botulinum neurotoxin (BoNT), including ultrasonic navigation, orientation of intramuscular motor endpoint of muscles (IME), is described. IME location diagrams and ultrasound picture of muscles are presented. Scales are proposed to assess the effect of spasticity on the functions of the upper and lower limbs. In conclusion, a variant of complex treatment of spasticity and original patient models are proposed, the use of which makes it possible to calculate the cost of BoNT.

Keywords: spasticity, patterns of spasticity, testing of spastic muscles, modified Tardieu scale (MTS), botulinum neurotoxin (BoNT), ultrasonic navigation, intramuscular motor endpoint (IME), rehabilitation

1. Introduction

Spasticity as the most important component of the defeat syndrome of the upper motor neuron is a motor disorder characterized by a speed-dependent increase in tonic stretching reflexes (muscle tone) with increased tendon reflexes, due to hyperexcitability of the stretching reflex, as one of the components of the syndrome [1]. It is detected in more than 12 million people in the world and is the cause of disability in 12–27% of them [2, 3]. The list of nosological forms in which the structure of the injury syndrome in the *upper motor neuron lesion* (UMNL) observed spastic hypertonicity is significant. It is determined in approximately 20–40% of stroke survivors, 14% of traumatic brain injury survivors, 65–78% of patients with spinal cord lesions, and 85% of patients with multiple sclerosis [4, 5].

Spasticity is a major obstacle to the recovery of the patients who have suffered brain damage. The work of restoring movement becomes impossible with the unresolved issue of spasticity, the treatment of which expands the frame of "the rehabilitation window" and defines the rehabilitation terms. In addition, in the absence of spasticity treatment, the risk of paresis complications increases: contractures, bedsores, limb deformities, pain, muscle spasms, etc. [6].

The development of spasticity is directly related to the initial stages of recovery of movement in paretic limbs. In 1–3 weeks after UMNL, the connections of the cerebral cortex with the structures of the extrapyramidal system (EPS) begin to recover. Recovery time varies depending on the degree of damage: from 2 weeks to 3 months [7].

During this period, the initiation of movements, in accordance with their image in the associative cortex, is able to be realized through intact EPS pathways. This efferent flow, reaching α-small motor neurons (MN), increases the tone in the muscles innervated by them (1st movement phase) for subsequent implementation of the dynamic phase (2-phase motion). However, the latter does not receive a sufficiently powerful efferentation due to the continuing defective functioning of the pyramid path. The result of this is the lack of inclusion of a sufficient number of inhibitory cells (Renshaw), which could inhibit α-small MN and lower muscle tone [7, 8].

Spasticity in strokes is usually formed in the first 3 months after the vascular accident. Its first signs usually begin to appear by the beginning of the 3rd week. The process of spasticity development is quite dynamic and variable. The terms of spasticity development from the first signs of tonus increase to the formed pattern in 2–5 weeks [9–11].

The development of spastic syndrome depends on many reasons: pain, stress, violation of proprioception, violation of the image of the body scheme and balance, tension and phobias with instability in a sitting or walking position.

Pain syndrome has a special place in the development of spasticity. For example, pain in the shoulder joint is directly associated with the development of spasticity. The absence of pain or its temporary relief in 83% of cases reduces the severity of spasticity.

Instability and uncertainty when walking, as well as stress, significantly affect the development of spasticity in the upper limb. So, spasticity in the hand, often develops in patients with severe weakness of the lower limb who retained the ability to move. During the period of yet unformed pattern of spasticity, these patients begin to strain, bend, and bring paretic arm to maintain balance while walking, which in 3–4 weeks causes the formed pattern of spasticity of the upper limb [12–14].

An important role in the development of spasticity plays the violation of proprioception, which leads to the violation of the image of the body scheme. Lack of afferentation triggers mechanisms that should increase the power of information from the tendons and joints receptors. In the case of spasticity, when dynamic muscle contraction is impossible due to paresis, it leads to activation of spinal and supraspinal mechanisms of tonus enhancement [15, 16].

Thus, the main directions in the rehabilitation of the consequences injury to the upper motor neuron and in the treatment of spasticity are:

- creating conditions precluding the need for the injury. For this purpose, analgesics, anxiolytics, as well as special styling, exercise therapy and hardware techniques are used, which activate proprioception, forming an associative image of the body scheme;

- the start of the physiological mechanisms of spasticity by activating muscle antagonists (taping, magnetic, electric stimulation, etc.) and initiating the mechanism of reciprocal interaction (kinesitherapy and other techniques);

- the use of methods that enable balancing the activity phases of the movement by reducing the activity in the 1st phase, up to severity 2nd (BoNT injections, stretching).

The most common complications of spasticity are contractures. They, together with spasticity, serve as the main obstacle to rehabilitation measures. The most common are the contractures of the shoulder (86%) and ankle (19%) joints. Adaptogenes of these contractures is different. Ankle contracture occurs among patients with late motor activity, severe paresis (70%), low motivation (81%), and incorrect treatment (23%). In contrast, shoulder contracture in 71% of cases is caused by the activity and verticalization of the patient in the first 14 days after the stroke. The shoulder joint, having the largest volume of movements, completely assumes all weight of the upper limb. With paresis and initial hypotension, often occurring after a stroke, the entire weight of the arm (4–7 kg) falls on the ligamentous apparatus of the joint, its articular bag, causing their trauma and pain. This, in turn, leads to the progressing severity of spasticity, as well as periarticular and articular changes [13].

By 6 months after the stroke, 90% of cases of spasticity, 36% of joint contractures, and 57% of tendon-muscle contractures (internal spasticity) are formed. At the same time, of all cases of spasticity, the manifestation of its signs in 77.3% of patients falls on the first 2–3 weeks. As a rule, the severity of spasticity in the first 2 weeks does not exceed 1–2 points on the Modified Ashworth scale (MAS). Increased spasticity by 1-point MAS increases the consumption of botulinum neurotoxin (BoNT) by 100–200 Units, which significantly increases the cost of treatment. Thus, early detection and treatment of spasticity reduces the cost of therapeutic rehabilitation measures [13, 14].

In the muscles involved in the spasticity pattern, diffuse muscle changes (DMC) develop over time in a form of connecting tissue substitution. There are no clear time criteria for the development of DMC. Among many patients with a disease duration of more than 4 years, the muscle structure is preserved. Some DMC develop within 6 months. Clinical signs of DMC are the following: low tissue turgor, decreased muscle volume, and significant restriction of movement, up to its total absence. While ultrasounding such muscles, in addition to reducing the volume, a hyperechogenic signal is registered (**Figure 1**) [17].

Currently, a classification has been adopted in which generalized, regional, and focal forms of spasticity are distinguished. There are also patterns of spasticity characteristic of different joints and muscles of the upper and lower limbs. Depending on the form and pattern of spasticity, the strategy and tactics of its treatment are determined [10]:

- generalized spasticity with pain—central muscle relaxants;

- focal and segmental spasticity—drugs BoNT;

- spasticity with marked para- or tetraparesis—intrathecal baclofen [15].

For effective treatment of spasticity with BoNT drugs, it is necessary to determine the spasticity pattern with verification of the muscles that form it; and then their correct introduction into the target muscle.

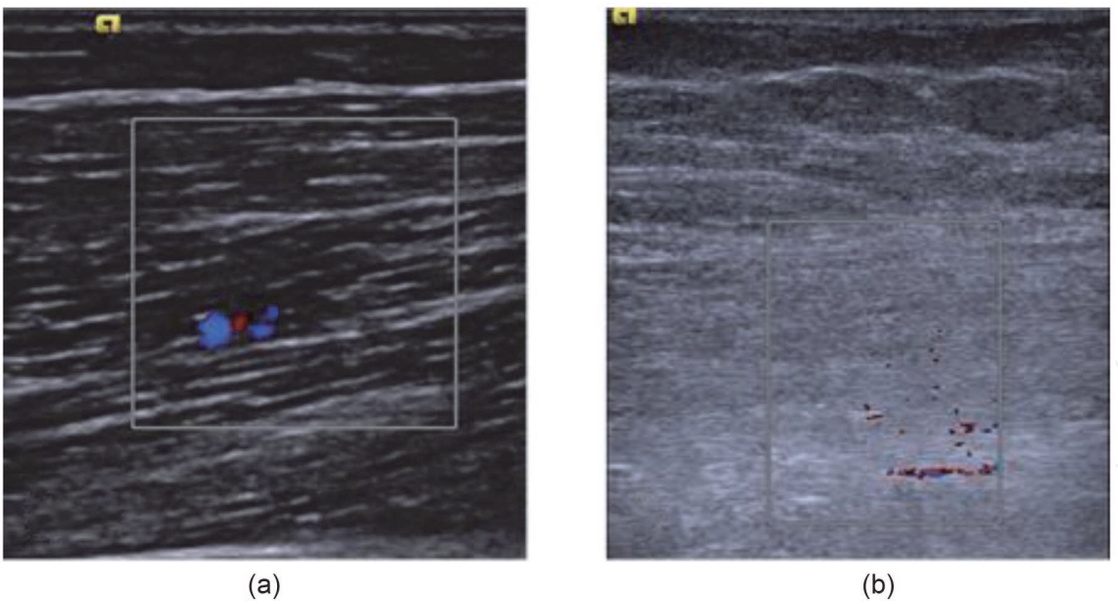

Figure 1.
Ultrasound picture of normal muscle tissue (a) and muscles with hyperechogenic ultrasonic signal due to DMC (b).

Twelve main clinical patterns of spasticity for the upper (5) and lower (7) limbs have been identified. Spasticity patterns for the hands include various, mainly flexor, variants of flexion in the joints: retracting the shoulder, elbow flexion, forearm pronation, wrist flexion, and finger flexion [18]. Spasticity patterns for the leg consist of hip reduction, knee flexion, knee extension, plantar flexion of the foot, equinovarus positioning of the foot, flexion of the fingers, and extension of the thumb [19].

2. Clinical diagnostic of spasticity

2.1 Manual testing of muscles for spasticity

Muscle testing is required to identify specific muscles involved in the spasticity pattern. All spastic muscle testing methods are based on two principles:

1. Identification and activation of exclusive and additional functions of the studied muscle.

2. Differentiation of movements for muscles with the same function, but passing through a different number of joints.

For spasticity patterns in the arm, an anatomical description is used with a representation of all the muscles that could perform a given movement in the joints (**Figure 2**). For differentiation of compensatory inclusions of muscles, it is necessary after definition of type of a pattern of spasticity in a hand to carry out manual testing.

2.1.1 Upper limb muscle testing

Bringing and pronation of the shoulder. At the heart of the restriction of movements in the shoulder with spasticity are problems with the withdrawal,

Figure 2.
Types of upper limb spasticity patterns [16].

Figure 3.
M. Subscapularis spasticity testing.

lifting of the shoulder, and its supination. Almost all the muscles of the shoulder girdle can influence these movement vectors, but most often it is done by the following: *m. Pectoralis* major (PM) (90%), *m. Subscapularis* (S/s) (20%), and *m. Laissimus dorsi* (LD) (5%).

Spasticity PM and LD is diagnosed when the shoulder is raised and withdrawn with the express tension of their lateral edge (the anterior and posterior walls of the axillary cavity, respectively). Diagnosis is done visually and by palpation.

S/s retracts the shoulder and rotates it inward. Spasticity is diagnosed by lifting, retraction and supination of the shoulder by visual and palpatory control of the lower angle and medial edge of the scapula. Normally, the shoulder is withdrawn without moving the blade to the horizontal level (80–90°). Thus, if the movement of the blade begins before reaching this level, there is an increase in tone in S/s. There is individual variability, so it is necessary to compare this movement with the intact hand (**Figure 3**).

Elbow flexors. The main muscles flexing the elbow are mm. Brachioradialis (BR), Brachialis (Br), and Biceps brachii (BB) (muscles are listed by importance in the spasticity pattern). BB is also a powerful arch support of the forearm. The muscles are tested when provoking a stretch reflex (muscle stretching reflex, mitotic reflex) at different rates of extension in the elbow joint. The reaction of BR and BB is evaluated visually and by palpation (**Figures 4** and **5**). The reaction of Br can only be assessed by palpation, gripping, with your own fingers upon humerus

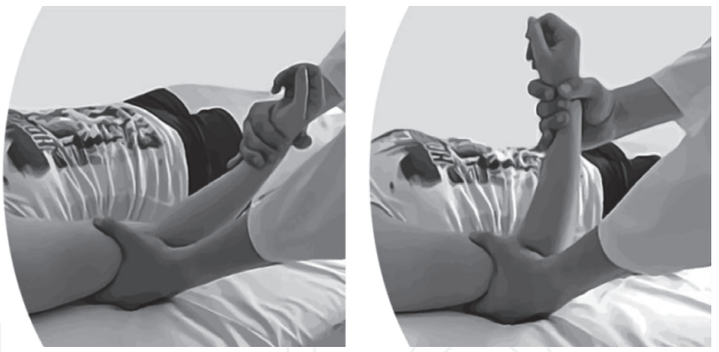

Figure 4.
M. Biceps brachii spasticity testing.

Figure 5.
M. Brachialis spasticity testing.

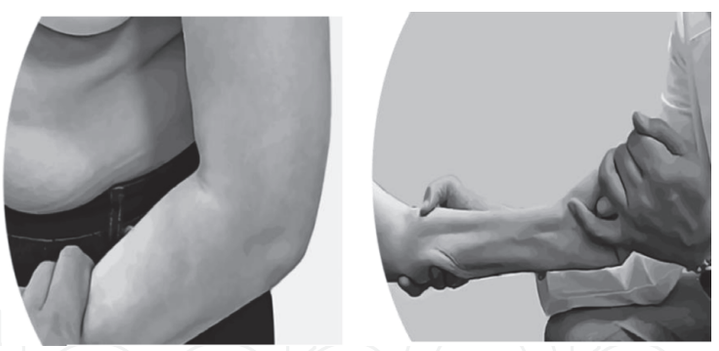

Figure 6.
M. Brachioradialis spasticity testing.

from below (rear). Win case of spasticity in response to a sharp extension in the elbow joint under the fingertips you feel tension (**Figure 6**).

Forearm pronators. Forearm pronates three muscles: mm. Pronator teres (PT) and Pronator quadratus (PQ) and m. Flexor carpi radialis (FCR). PT and FCR in pronation act as a single functional unit. In some cases, with ultrasound of these muscles, you can find the lack of a clear boundary between them, which once again confirms the generality of their function. Testing these two muscles is based on provoking the stretch reflex in response to rapid supination. The presence of spasticity in FCR is manifested visually and confirmed by palpation. Spasticity in PT can be determined only by palpation, putting your fingers on the middle line of the forearm 2–4 cm below the elbow bend and producing supination. Sometimes when assessing pronator spasticity, FCR is more stressful than PT.

Flexors of the hand and fingers. Testing is performed by back flexion in the wrist joint. If no significant resistance is felt during this movement, and the fingers

are progressively flexed as the hand is extended, this means that neither of the two flexors of the hand (m. Flexor carpi radialis and m. Flexor carpi ulnaris) participates in its flexion. Pathological flexion of the hand in this case is due to spasticity m. Flexor digitorum profundus (FDP) and m. Flexor digitorum superficialis (FDS) (**Figure 7**).

In order to distinguish spasticity in FDS and FDP, you can extend the wrist joint. In this movement, it is necessary to pay attention to the proximal and distal interphalangeal joints of the fingers. If it is found that the distal interphalangeal joints are bent to a greater extent, this indicates the spasticity of the deep flexor of the fingers. If the proximal interphalangeal joints-superficial flexor fingers (**Figure 8**). If this bends the distal phalanx of the thumb—this indicates spasticity m. Flexor pollicis longus (**Figure 9**).

If manipulations in the wrist joint did not have any effect on the position of the thumb, it means that the muscles of the hand are responsible for its position: mm. Flexor pollicis brevis, Opponens pollicis Adductor pollicis (**Figure 10**).

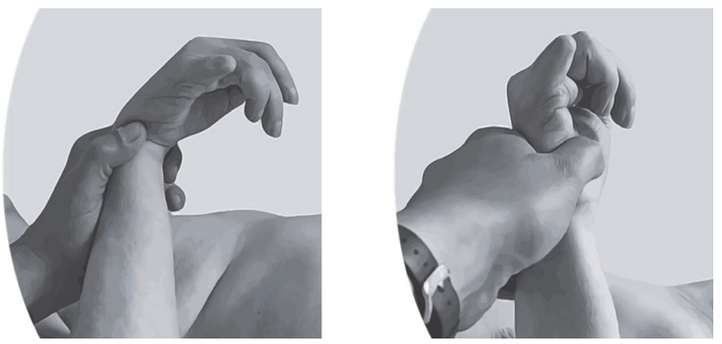

Figure 7.
M. Flexor digitorum profundus u m. Flexor digitorum superficialis spasticity testing.

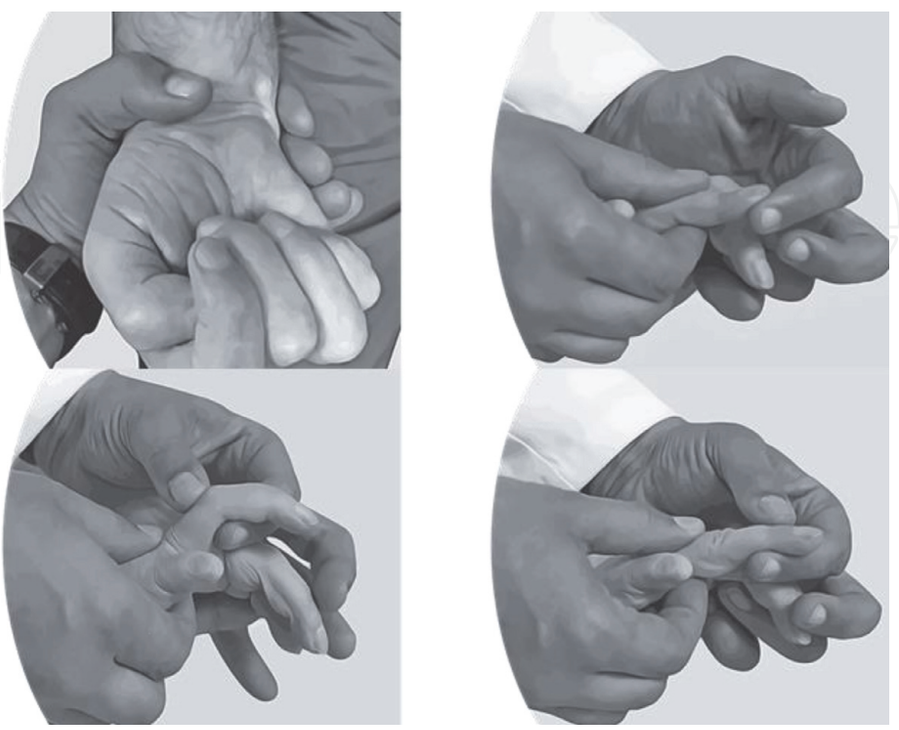

Figure 8.
Differential diagnosis of spasticity m. Flexor digitorum profundus and m. Flexor digitorum superficialis.

Figure 9.
M. Flexor pollicis longus spasticity testing.

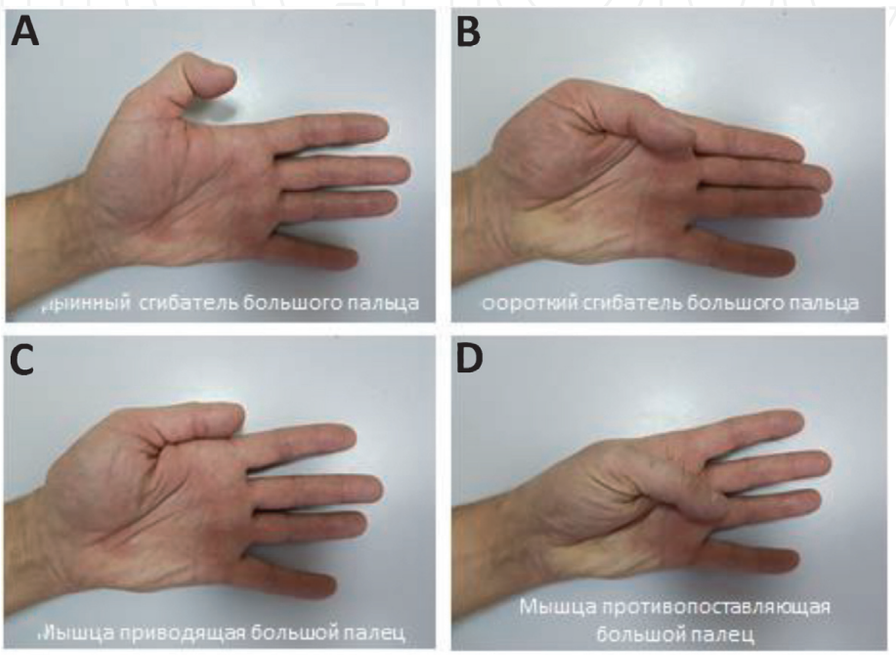

Figure 10.
Patterns in spasticity of the muscles of the hand. (A) m. Flexor pollicis longus, (B) m. Flexor pollicis brevis, (C) m. Adductor pollicis, and (D) m. Opponens pollicis.

2.1.2 Lower limb muscle testing

While a professional consensus has been formed on the upper limb spasticity patterns based on the work of H. Hefter, a consolidated opinion on the lower limb spasticity patterns has not been developed, and the question of determining target muscles and BoNT dosages still remains open [11].

The peculiarity of the lower limb patterns is the ambiguity of their distribution among clinical nosological forms. This fact and the remaining unclear features of the pathogenesis of spasticity is that it does not allow you to choose and apply to the patterns of the lower limb a single classification principle. We can only say about the predominant frequency of occurrence of a particular pattern in any nosology.

Thus, we can distinguish:

1. Spasticity patterns in cerebral palsy.

    - Flexion of hip and knee joints.
    - Internal rotation and the tibia adducci.
    - equinus.

- Valgus or varus.
- Exterior turn stop.
- Adduction.

2. Patterns of spasticity in multiple sclerosis.

- Adduction.
- Flexion or extension of the hip.
- Of equinus.
- Flexion or extension of the knee.

3. Spasticity patterns in severe brain injury, encephalitis, and spinal injury.

- "Triple flexion".
- Flexion of the ankle joints.
- Adduction.
- Flexion of the toes.

4. Patterns of spasticity in stroke and brain injury.

- Dynamic pattern.
- Static pattern (equinovarus)
- their combination with flexion of the toes.

Testing of the muscles of the lower limb is carried out in the supine position, on both limbs, consistently comparing flexion in the joints (to identify poor muscle extensibility not associated with spasticity) [18].

There are two main types of lower limb spasticity patterns in patients undergoing stroke: dynamic pattern (DP) and static pattern (SP), as well as their possible combinations with flexion of the toes and hip reduction. Patterns are proposed based on the principle of visual assessment of the limb position at rest and when walking [19].

In case DP manifestations of spasticity are determined only in the process of movement. In the resting position, the legs do not differ from healthy and its normal statics is kept (limb are visually full length, the joints are in the middle position, and toes are separated), the position of the fingers most often corresponds with the finger of these intact side. Walking is characterized by a peculiar pattern, in which in the phase of hip transfer and knee extension, before lowering the foot to the surface, there are oscillatory movements of the shin from side to side with possible flexion of the fingers. The cause of DP is increased tone and muscle-tendon contraction in the muscles of the back of the thigh (hamstrings), – mm. Semitendinosus (S/t), Semimembranosus (S/m), Biceps femoris (BF) and in *M. gracilis* (G).

Gait peculiarity in this type of spasticity is associated with the phylogenetic foundations of neurophysiology of movement in providing the act of walking and is

realized through segmental connections, leading, in part, to its automatism [20–22]. As a result, the paretic limb, which tends to step of the same characteristics as the intact one, encounters hamstrings contraction, which leads to a push of the hip and knee backward and medially, stopping the inertia of the limb forward, shortening the step and, sometimes, bringing the hip and shin oscillatory movements [23–25].

SP is characterized primarily by equinecom and equinovarus that can be observed in standing and lying down. There may also be curvature of the pelvic position due to changes in limb length, and/or knee flexion. But more often there is flexion in the ankle joint with a possible compensatory tension of the anterior muscle group of the thigh. The gait in this case becomes circulatory with a slope contralateral to the paresis. This result toning any of the four muscles: m. Soleus (S), m. Gastrocnemius (G/c), m. Tibialis posterior (TP), m. Tibialis anterior (TA).

An additional phenomenon in SP and DP can be flexion of the toes, which is responsible for spasticity mm. Flexor digitorum longus (FDL), Flexor hallucis longus (FHL), Flexor digitorum brevis (FDB), and Flexor hallucis brevis (FHB).

1. Assessment of DP (**Figure 11**):

   A. For the differential diagnosis of posterior thigh muscle hypertonicity, the patient's straight leg is bent at the hip joint. At the limit of extensibility of spastic muscles, involuntary flexion of the leg in the knee joint is fixed. Fixing the leg at this level, use palpation and visually determine tense muscles of the back of the thigh. In difficult cases, for verification of spastic muscles, ultrasound diagnosis should be carried out.

   B. To test spasticity in the inner thigh muscles (adductors, G and m. Sartorius (Srt)), the leg is retracted to the tensile limit. After that spastic muscles are examined by palpation, visually, and optionally using ultrasonic equipment (**Figure 12**). To differentiate the tone increase in adductors and G, a gracilis test was also performed (**Figure 13**). To identify limitations in the diversion of the leg, it is made bending at the knee (the test must be performed on the edge of the couch). The ability to perform further hip abduction after flexion-indicates spasticity in G. The Lack of response to knee flexion indicates an increase in tone in the adductors.

2. Assessment of SP:

   A. To differentiate spasticity in m. Soleus (S) and in the heads of m. Gastrocnemius (G/c), a Silfverskiold test should be performed (**Figure 14**) [26].

Figure 11.
Spasticity testing in posterior thigh muscles (mm. Semitendinosus, Semimembranosus, Biceps femoris, and Gracilis).

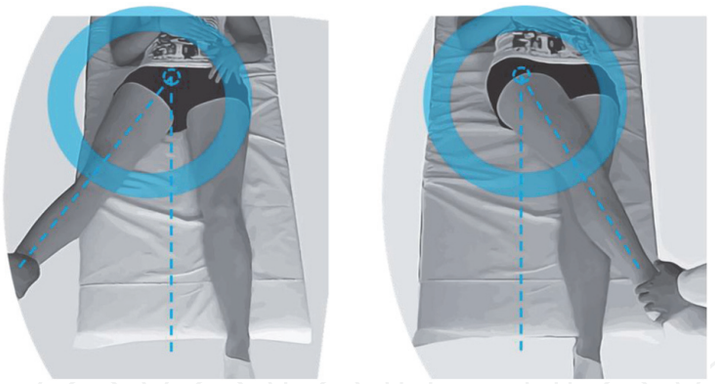

Figure 12.
Testing of spasticity in the hip adductor muscles.

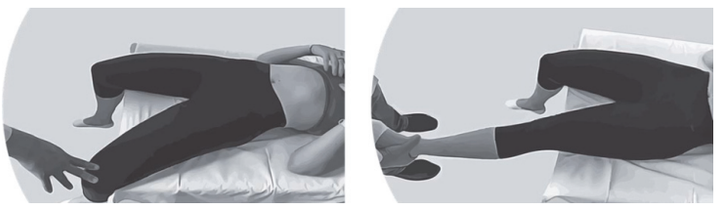

Figure 13.
Testing of spasticity in m. Gracilis (gracilis test).

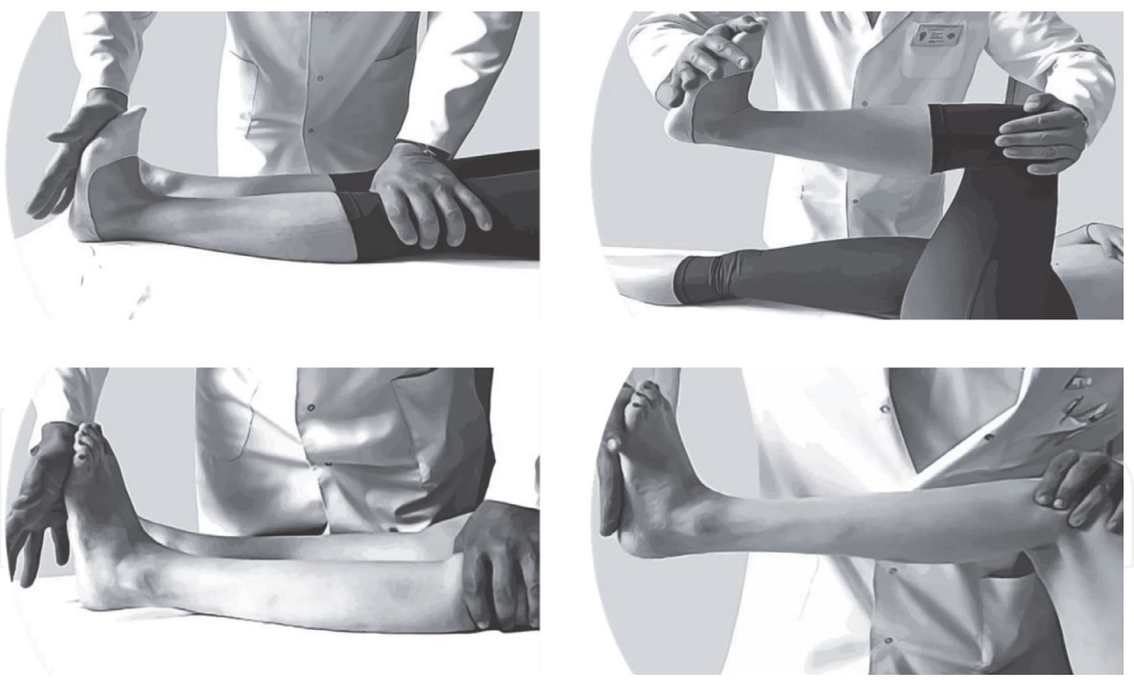

Figure 14.
Sequential test execution Silverskiold's to identify spasticity in m. Gastrocnemius and m. Soleus.

During the test, the angle of flexion of the foot is consistently determined with the leg straightened and bent at the knee. No change in the position of the ankle joint in response to flexion of the knee and the foot clonus testify the increase of tone in S. Marked decrease of the bending angle up to 80° or less while you straighten the legs indicates spasticity in G/c. Supination of the foot of any severity in the last phase of the rectification of the foot manifests the increased tone only in the medial head G/c. As an additional test when the leg is straightened, should be a passive extension of the foot

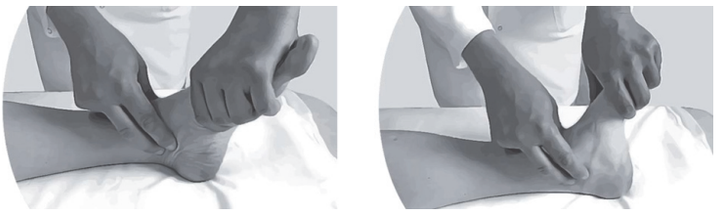

Figure 15.
Testing of spasticity in m. Tibialis posterior.

with simultaneous sharp proanation. Visually and by palpation fixed tension of medial head G/c confirms the increase of its tone. The presence of spasticity only in the medial head G/c leads to tension of the medial part of the Achilles tendon, which is manifested by pulling the heel bone back and up with a turn inward.

B. To determine the increase in tone *m. Tibialis* anterior (TA), it is necessary to make a rapid flexion of the foot followed by pronation. This maneuver provokes a stretch reflex in TA. The detected tension TA (visually and by palpation) and the contour of the tendon at the back of the foot is the evidence of increase of its tone.

C. To test spasticity in *m. Tibialis* posterior (TP), it is necessary to perform a rapid extension of the foot followed by pronation. This triggers the stretch reflex to TP. The muscle is not visually controllable, but its tendon runs along the lower edge of the medial ankle. Palpation recorded muscle tension and tendon contouring indicates an increase in its tone (**Figure 15**).

D. Diagnosis of spasticity in the flexors of the toes is made by performing sequential passive flexion and extension in the ankle joint:

- if there is simultaneous flexion of the toes during the extension of the foot, this indicates an increase in tone in m. Flexor digitorum longus (FDL) and/or m. Flexor hallucis longus (FHL);
- maintaining your posture of flexion, regardless of movements in the ankle joint demonstrates increased tone in m. Flexor digitorum brevis (FDB) and/or m. Flexor hallucis brevis (FHB). Tension in them can also be seen with palpation of the arch of the foot.

3. Scales for assessing spasticity and disorders of activity and participation

3.1 Rating scale for evaluating the condition of muscles

The main scales to assess the condition of the muscles are: the scale of muscle contraction force and volume of voluntary movements (MRCS), modified Ashworth scale (MAS) and the Tardieu scale (MTS) [27–30].

The scale of muscle contraction strength and volume of voluntary movements (Medical Research Council Scale (Oxford Scale), MRCS) allows to estimate the strength of muscle contraction and amplitude of active movements in the limb.

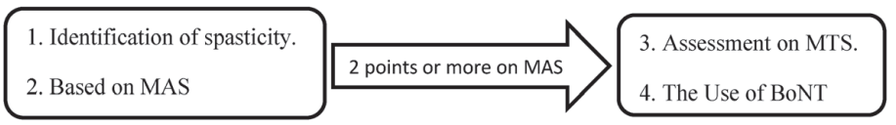

Figure 16.
Algorithm of diagnosis and treatment of spasticity.

The modified Ashworth Scale (MAS) serves to objectify muscle tone and is the most used to evaluate the effectiveness of treatment of spasticity of BoNT [31].

MAS allows, without resorting to special measuring tools and calculations, to assess the degree of mobility of the joints, associated with increased muscle tone when performing passive movement. At the same time, MAS does not reveal the nuances of spasticity, such as muscle reactivity, the dependence of its contraction on the rate of tendon stretching [13].

The modified Tardieu Scale (MTS) [29, 30, 32] allows the most complete assessment of all manifestations of spasticity: tone, stretch reflex (reaction to tendon stretching), and spastic co-contraction (inclusion of muscles antagonists to movement).

The capabilities inherent in the MTS assessment system allow not only to verify spasticity in more detail, but also to quantify muscle weakness, fatigue, and the state of deep sensitivity [33].

The following algorithm is used to diagnose and treat spasticity (**Figure 16**) [11]:

The measurement system incorporated in MTS is performed by a goniometer and must be performed at the same time of the day, and the tested limb must be placed in the same position during repeated testing (**Figure 17**) [11].

The peculiarity of MTS application is the assessment of changes in muscle tone and angles of movement in the joint in response to the provocation of spastic co-contraction (activation of muscles antagonists to movement) and stretch reflex (reaction to tendon stretching) obtained at different speeds of passive movement in the joint.

The speeds are selected according to the following characteristics:

- as slowly as possible (V1);
- speed equal to the speed of fall of a limb moving under the action of gravity (V2);
- as soon as possible (V3) (faster than the speed of natural fall of the limb segment under gravity).

In recent years, in the professional community, there is a refusal to assess the rate V2 [31, 33], which leaves two fundamental indicators (**Figure 17**):

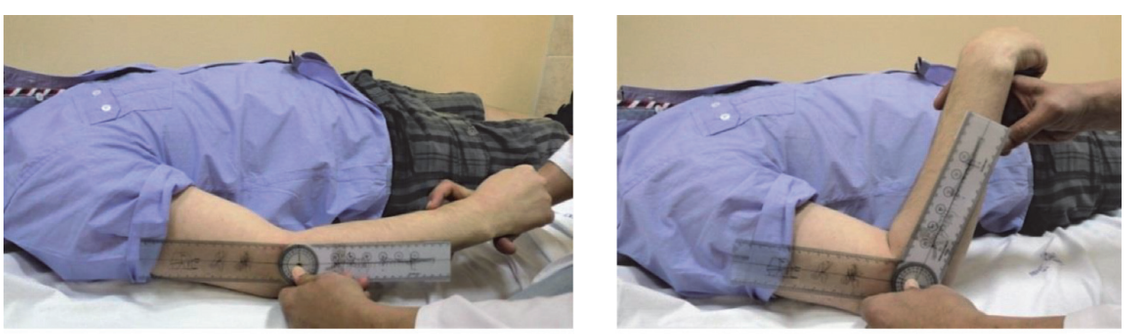

Figure 17.
(A) Slow passive extension, X_{V1} and (B) fast passive extension, X_{V3}.

X_{V1} – angle measured at speed V1.
X_{V3} – the angle measured at the speed V3.

The Tardieu scale offers a flexible evaluation system that allows you to use different approaches in the diagnosis of spasticity, opens the possibility of choosing evaluation parameters, provides options for both rapid evaluations based on one or two parameters, and a full-scale study of spasticity and paresis with the calculation of indices and coefficients, which makes it possible to register the minimum rehabilitation dynamics.

There are two main ways to use MTS. The first of them involves taking into account the score, which reflects the characteristics of the reaction of muscles and tendons in response to their stretching, the other option is based on taking into account the angle of the movement end, without a special assessment of the nuances of the muscle reaction. It is also possible to combine the use of both options or the use of separate elements from each of them.

In the 1st variant of MTS application, the assessment is based on two parameters: the degree of muscle reaction (Y) in points and the angle at which the muscle reaction (X) in degrees is achieved.

To score the degree of muscle reaction (Y), a table of scores and their interpretations is used (**Table 1**).

The estimated home value of 1st version of evaluation is **Index Tardieu** (IT)—the ratio Y (point) to X (degrees) achieved at different speeds of movement in the joint:

$$\mathrm{IT}\ (\mathrm{V1}, \mathrm{V2}, \mathrm{V3}) = \mathrm{Y}\ (\mathrm{score})/\mathrm{X}\ (\mathrm{angle\ in\ degrees}). \quad (1)$$

Thus, if the angle at which the reaction occurs and/or the score varies with the velocity, we get three results. For example, in the elbow joint:

1. $\mathrm{IT}_{V1} = 1/180 = 0.005$.

2. $\mathrm{IT}_{V2} = 2/90 = 0.022$.

3. $\mathrm{IT}_{V3} = 3/90 = 0.033$.

IT_{V1} characterizes increased muscle tone outside the reaction to the stretch reflex and demonstrates the degree of shortening of the muscle. The results obtained at V2 and V3 rates characterize the degree of muscle reaction to the tendon stretching rate and are different degrees of stretch reflex provocation.

A significant difference between $\mathrm{IT}_{V1–V3}$ (2.5 times or more) may indicate the degree of dynamism of the muscle-tendon contracture or its absence, which allows us to count on a good result when using BoNT. A slight difference in the values of it

Points	Interpretation
0	No resistance throughout passive movement
1	Slight resistance throughout with no clear catch at a precise angle
2	Clear catch at a precise angle, followed by release
3	Fatigable clonus (10 s) occurring at a precise angle
4	Unfatigable clonus (>10 s) occurring at a precise angle
5	Joint immobile

Table 1.
Quality of muscle reaction.

(1.5–2 times or less), with severe limitation of movement in the joint indicates a worse prognosis and the need for active exercises on muscle stretching or diagnosis of joint contracture [13].

The 2nd variant of application of MTS actually does not assume the use of the table of a point estimation and is based on variety of measurements of angles of movement in a joint and change of a scope of movements depending on manifestations of spasticity, register:

- X_{V1}—angle range of passive movement of a limb at a slow speed (angle arrest);
- X_{V3}—angle stop movement of the limbs at high speed (angle catch);
- X_A—angle muscle power (corner of the active movement in the joint by working the antagonist muscles spasticity (active motion));
- X_{A15}—angle fatigue of the muscle (measured after 15 s of working the antagonist muscles spasticity).

The main calculated value in the 2nd variant of MTS application is the spasticity angle (X_S):

$$X_S = X_{V1} - X_{V3}. \tag{2}$$

As part of the diagnosis and treatment, it is also necessary to know the X_N – angle of the normal volume of movement in a particular joint. This is necessary not only to understand the degree of spasticity but also to calculate the coefficients proposed in the scale and characterizing the state of the muscles. Such factors are: (1) velocity factor (C shorting) muscle C_{SH}, (2) ratio of spasticity (spasticity C) C_S; (3) the coefficient of weakness (weakness C) muscle C_W; and (4) coefficient of fatigue (fatigue C) C_F:

1. $C_{SH} = (X_N - X_{V1})/X_N$.
2. $C_S = (X_{V1} - X_{V3})/X_{V3}$.
3. $C_W = (X_{V1} - X_A)/X_{V1}$.
4. $C_F = (X_A - X_{A15})/X_A$.

Measuring X_A and X_{A15} and calculating C_F and C_W fill another important gap in neurology—the ability to fully quantify paresis, thereby significantly complementing the use of MRCS [33].

There is also a muscle reaction angle (x): measured as the difference between the forced position of the joint and the angle of the normal anatomical position of the limb and its segments (applies to all joints except the hip) [32, 34].

In a complete evaluation system for the Tardieu scale includes not only the motion estimation but also sensitivity. Verification of deep sensitivity disorders is achieved by measuring the proprioception angle (X_P). Normally, the brain fixes the angular displacement in the joints to 2–3°.

At neurological examination, as a rule, it is considered sufficient to reveal only the fact of violation of muscular-articular feeling. But, for the prognosis of spasticity, qualitative diagnosis of proprioception disorders and evaluation of

rehabilitation dynamics is not enough. This provision has a pathophysiological justification. One version of the pathogenesis of spasticity is the activation of muscle contraction in response to afferentation insufficiency [7]. Obtaining information about the degree of proprioception impairment allows us to predict the subsequent development of spasticity, suggesting the effectiveness and assessing the dynamics of rehabilitation. That in turn makes it possible to talk about such a definition as "rehabilitation potential" and plan the volume, structure and timing of rehabilitation of the patient.

Full use of all the features of the Tardieu scale actually allows you to make a "passport" of a certain muscle. In cases where we cannot isolate the function of a single muscle, differentiating it from the synergists, we identify the effect of spasticity of movement in the joint as a whole. An example of this is the work of the muscles of the back of the thigh, where we cannot separate the function of m. Semitendinosus and m. Semimembranosusand assess the degree of their isolated effect on movement in the joint [35].

The "passport" of spastic muscles or restrictions of movements in the joint can be presented in the form of a table:

Muscle/joint	X_{V1}	X_{V3}	X_S	X_A	X_{A15}	X_P	C_{SH}	C_S	C_W	C_F

Especially important for the use of the Tardieu evaluation system is the understanding of the principles of measuring motion in the joint, in particular, the selection of the reference point of the angle of motion. In this case, the measurement system is different from that adopted in orthopedic practice. The reference point is the point is opposite to the studied movement or, in other words, the measurement is carried out from the extreme points of flexion/extension, reduction/withdrawal, pronation/supination, that is, the points to which the co-contraction tends [11]. The point is selected along the axis of the limb segment under study, regardless of whether the segment reaches this point or not. The main task is to make a movement from the point of greatest muscle relaxation to the point of maximum muscle stretching. A good example of this is the study of the movement during the extension of the ankle joint with spasticity in its flexors (**Figure 18**) [12].

Angles are measured from the point lying on the continuation of the axis line of the shin outside the limits of possible flexion in the ankle joint, that is, the count of extension in the joint a priori begins with 45°. The entire range of extension in the joint measured from the line of continuation of the Shin, that is, to the angle of 115°, is estimated. Thus, the movement is carried out in the direction of maximum stretching of the flexors of the joint. In the presented example (**Figure 18**), the

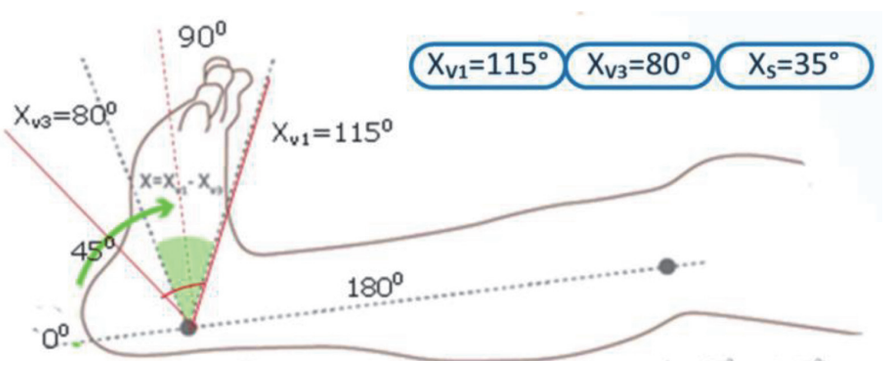

Figure 18.
Measurement of the volume movements of the MTS with increasing tonus of flexors of the ankle joint.

slow-speed stop (V1) occurs at the extreme point of extension in the joint 115°, which indicates the absence of muscle contractures. Stopping at a fast speed (V3) occurs at 80°, which characterizes the stretch reflex and co-contraction of the ankle flexor muscles. The calculated spasticity angle in this case will be 35°.

In the case where it is necessary to evaluate the extensor muscles of the joint, the movement is carried out in the opposite direction-toward their maximum stretching and flexion of the joint. The starting point is the point lying on the shin axis, but since the maximum extension of the foot reaches 115°, the movement begins only from the position 65°. The entire range of flexion in the joint is estimated, that is, up to 135°.

Most often, spasticity limits the following movements: flexion and retraction in the shoulder joint, extension in the elbow, wrist and wrist joints, supination of the forearm, extension/flexion of the hip and knee joints, hip abduction, extension and pronation of the foot, and extension of the toes [19, 35]. Accordingly, the reference point for measuring the volume of these movements will be located at the point of maximum contraction of the muscles that prevent this movement (**Figures 19–25**) [11].

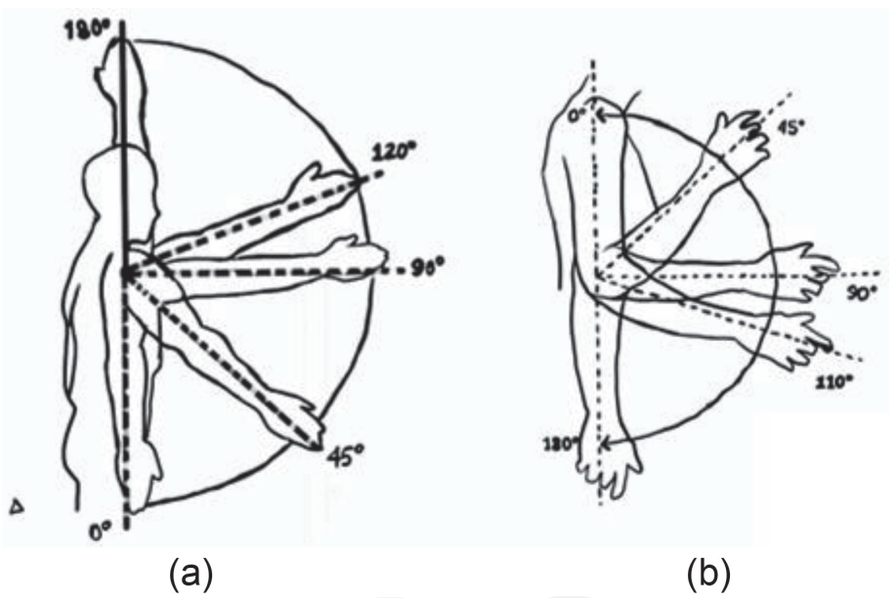

Figure 19.
Measurement of MTS range of motion in the shoulder (a) and elbow (b) joints in typical spasticity patterns.

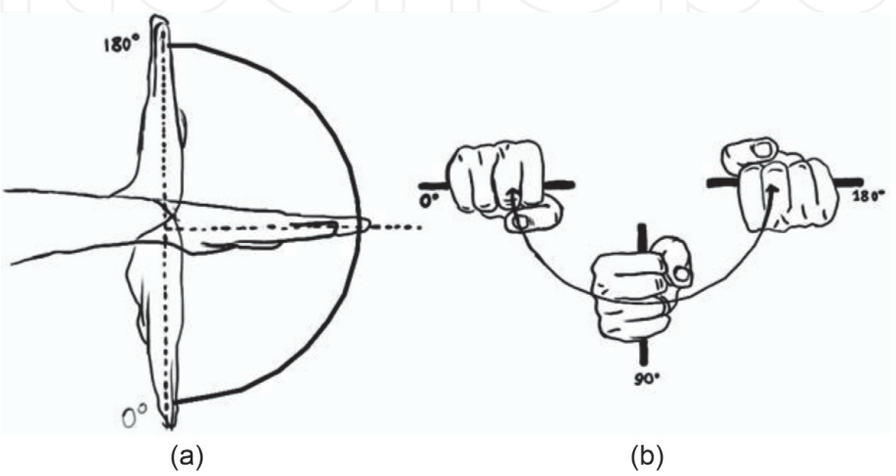

Figure 20.
Measurement of range of motion of MTS in the wrist joint in extension of wrist (a) and supination of the forearm (b) when the typical patterns of spasticity.

Considering the treatment of spasticity as part of the rehabilitation process and, given that the therapeutic effect on spasticity has the ultimate goal of normalizing the life and activities of the patient, Graces recommends the following step-by-step strategy for the use of the tardier scale [33].

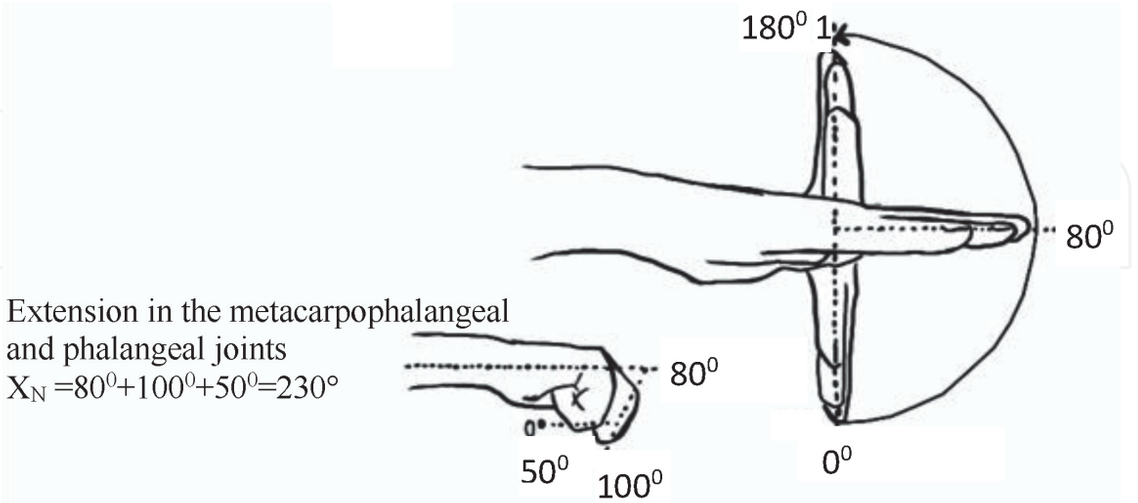

Figure 21.
Measuring the volume of movements by MTS in typical spasticity patterns in the joints of the hand.

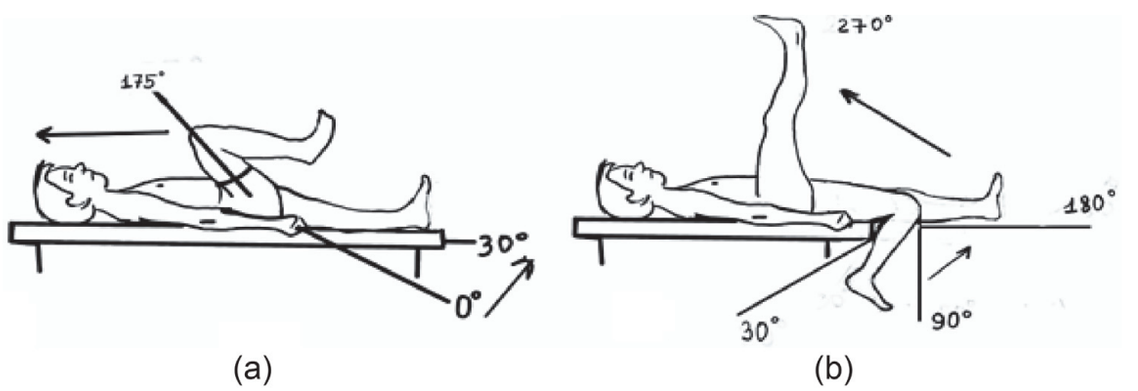

Figure 22.
Measurement of MTS range of motion in the hip and knee joints with gluteus maximus (a) and hamstrings (b) spasticity.

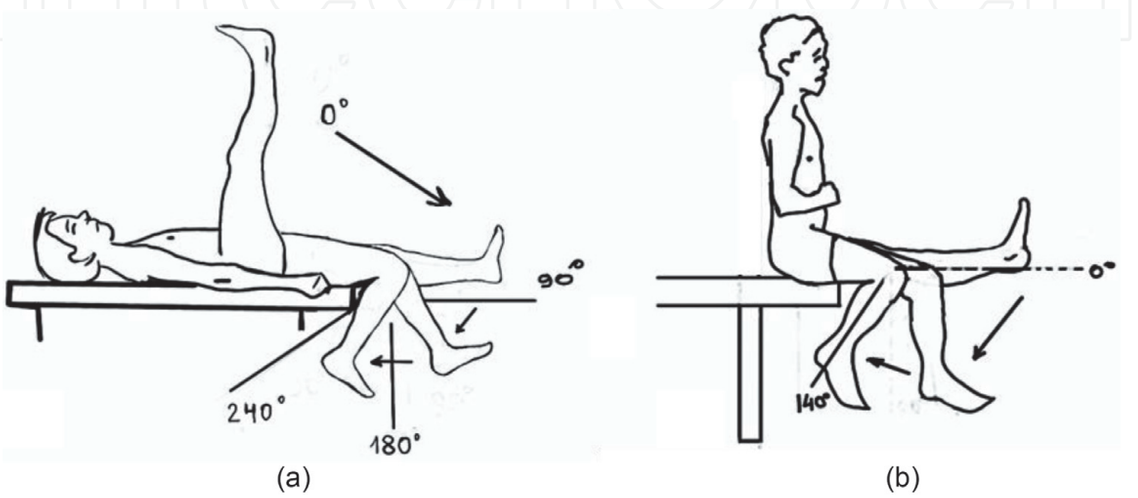

Figure 23.
Measurement of range of motion at the MTS when spasticity in the rectus (a) and lateral vastus, and medial, intermediomedialis et lateralis of m. Quadriceps femoris (b).

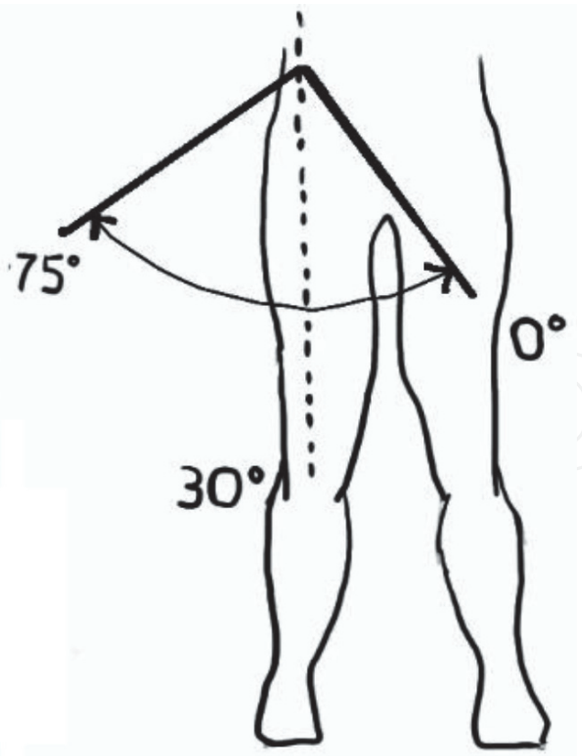

Figure 24.
Measuring the volume of movements by MTS in adductor spasticity.

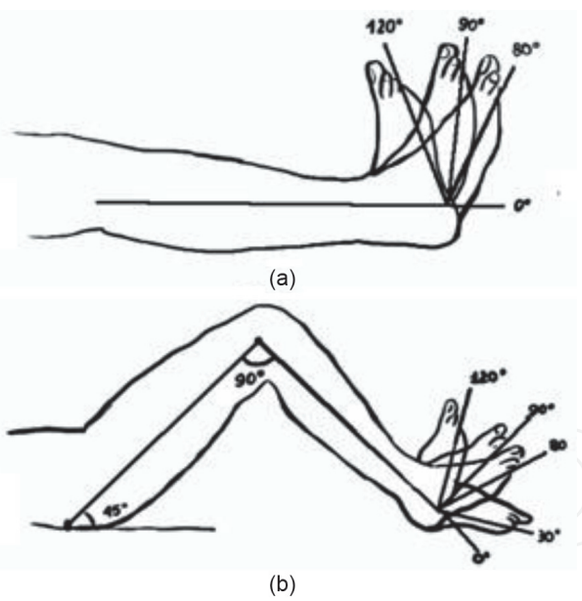

Figure 25.
Measurement of the volume of movements by MTS in the ankle joint with spasticity of Gastrocnemius (a) and Soleus (b).

Step 1: Maximum volume of passive movement (PROM-passive range of motion) in the joint at slow speed, assessment of the degree of muscle shortening (angle arest) = X_{V1}.

Step 2: Passive movement in the joint at fast speed, evaluation of spasticity (Y and/or angle catch) = X_{V3}.

Step 3: Active joint movement, strength score = X_A.

Step 4: Active and rapid movement in the joint for 15 s. With the subsequent angle measurement, assessment of fatigue = X_{A15}.

Step 5: Assessment of limb function and human activity (various activity and participation tests) (LASIS, Frenchay, 10 m walk test, etc.) = F (limb function).

The place and role of MTS in rehabilitation is demonstrated in the following algorithm of rehabilitation approach to patients with spastic paresis (**Figure 26**) [34]. It involves testing the patient, identifying the problem, selecting the rehabilitation goal, developing an intervention plan, and then analyzing the outcome with the selection of the new rehabilitation goal.

It is possible to use MTS in the most limited form. It is enough to measure V1 and V3 and calculate X_S. Registering these three parameters and their changes will

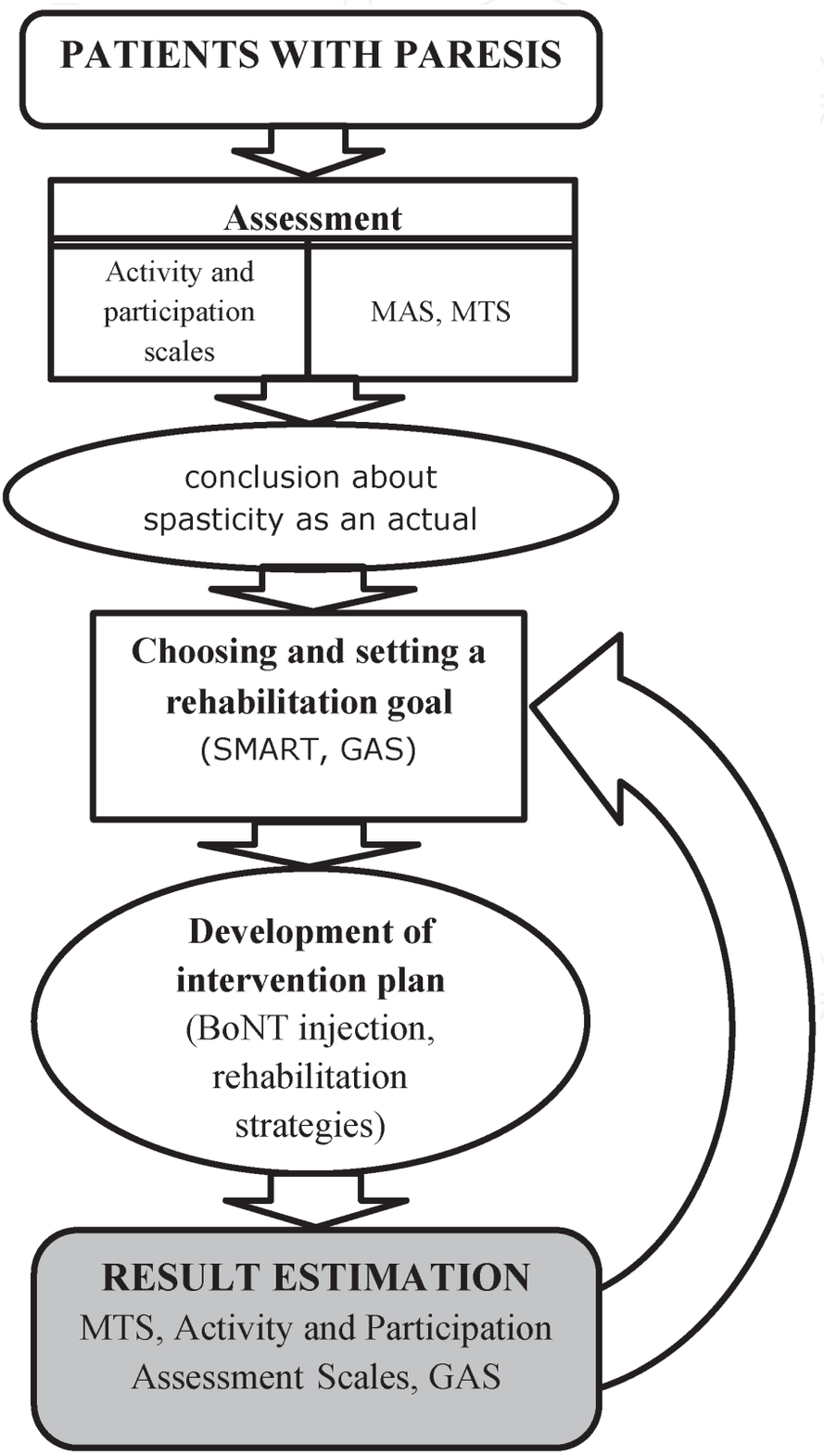

Figure 26.
Algorithm of diagnosis and treatment of a patient with spastic paresis.

allow to sufficiently assess the effectiveness of botulinum therapy and rehabilitation dynamics.

In the treatment of spasticity, the use of MTS allows us to conclude how the introduction of BoNT had an impact on the rehabilitation of the patient. The choice of a specialist drug BoNT for the treatment of spasticity is based on the experience and analysis of many factors, among which one of the most significant is pharmacoeconomics. On average, the duration of various drugs BoNT in patients with spasticity reaches 12–14 weeks [11].

Thus, the stages of application of MTS in the treatment of spasticity BoNT are:

1. testing before injection;

2. testing 3–4 weeks after injection, which demonstrates the effectiveness of BoNT;

3. testing 12–20 weeks after injection of BoNT. Evaluation at this time interval shows the effectiveness of rehabilitation, as well as being a baseline assessment for deciding on the next injection session.

Thus, the modified Tardieu scale (MTS) is convenient for full-scale diagnosis of the main elements of the clinical picture of the central nervous system damage, such as paresis, spasticity, proprioception disorders and allows to qualitatively and quantitatively assess the dynamics of treatment and rehabilitation.

3.2 Activity and participation scales

Spasticity has an extremely negative impact on daily and professional activities, severely restricts independent movement, self-service, reduces the role of the individual in the family and society. The therapeutic effect on spasticity has the ultimate goal of normalizing the life and activity of the patient and with a favorable outcome provides the return to the original standard of living [36].

The Hauser walking index (HAI) and the Rivermead mobility index (RMI) are used to verify self-mobility and self-service violations. Since these scales are not sensitive enough to small changes in mobility, they should be supplemented by a quantitative test to assess walking: distance, time, and independence [36–38].

The concept of self-service includes not only movement but also its assessment should be supplemented by the analysis of the degree of influence of spasticity in the hand on daily activities. The most convenient and informative tool for this analysis is the Leeds scale of influence of spasticity of the hand on the activity (LASIS) [34, 39, 40].

3.2.1 Movement assessment

The Hauser Ambulation Index (HAI) includes the ranking of patients by 10 gradations depending on the need for external assistance, the use of devices for movement, and the time of passing the test distance [34, 36].

Index of Rivermead mobility (Rivermead Mobility Index, RMI) [37, 38]. The value of the mobility index, developed at the Rivermead center, Cambridge University, is the sum of the points: 1 point for each positive response.

The range of values of the scale of the index can vary from 0 points (the inability to perform any of these actions on their own) to 15 points (the ability to run 10 m), which corresponds to normal human mobility. Of particular value for assessing the impact of spasticity on human activity, this test acquires due to the fact that it includes tasks similar to those performed by a person in everyday practice (that is, it

has a high "environmental friendliness"). Also, this test can be used to assess the effectiveness of the use of BoNT in relation to the improvement of movement [41].

Walking assessment tests. A common feature of these tests is the lack of assessment of walking quality. Unfortunately, walking quality cannot be reliably assessed without the use of laboratory gait analysis techniques. But it must be borne in mind that it will always be more important for the patient to be able to reach the object he needs safely and quickly than to walk "beautifully." Therefore, the above scales and tests do not lose their relevance in clinical practice, despite the development of instrumental methods for diagnosing walking disorders.

3.2.2 Evaluation of hand productivity

For a quick (less than 10 min) evaluation of the possibility of manipulation (capture, lifting and transfer) objects of different sizes, you must use the "Test with nine pegs" (objects with a diameter of about 1 cm), the test "Box and blocks" (box and Block Test) (cube edge 2.5 cm), the test ARAT (Action Research Arm Test) (manipulation of objects with different sizes, shapes, and weights), Frenchay test (evaluation of functional movements: fixing the ruler, manipulation of objects of different sizes, pinch grip, as well as the ability to touch the top of the head), and Leeds Arm Spasticity Impact Scale (LASIS) [42–45].

This scale was developed at the University of Leeds to measure the effect of spasticity on the functionality and care procedures for paresis of the hand [44]. The daily activities of the patient or the caregiver during the preceding 7 days shall be taken into account.

In each case, the respondent, the patient and/or the caregiver are asked if the action is feasible or not. The difficulty is evaluated on the scale from 0 to 4.

Modified scale Frenchay (Modified Frenchay Test MFS) allows to estimate the functional state of hands. The concept of "functional state" indicates how the sick hand is adapted to everyday life and participates in it [46].

4. Treatment of spasticity

4.1 Methods of injection of BoNT in spasticity

The effectiveness of treatment of spasticity BoNT depends on the accuracy of the introduction into the target muscle and thus is directly related to the skill of a particular specialist and possession of his methods of navigation [47].

4.1.1 Ultrasonic navigation in the treatment of spasticity

Representations of the injection point and depth of the target muscle, based on the knowledge of anatomy, are often incorrect. The location of muscles and bones relatively to each other, their volume is individual, and the presence of vessels and nerves at the injection site is unpredictable. Only 15–20% of individual anatomical structure corresponds to that presented in the relevant atlases. Any pathology, associated with the distortion of movement, exacerbates the differences in the relative position and volume of muscles. Thus, the orientation to the generally accepted anatomical guidelines in the introduction of BoNT makes the treatment of spasticity extremely ineffective. Ultrasound scanning is the main navigation method for BoNT injections in the treatment of spasticity [17].

Ultrasound scanner. To navigate the muscles in botulinum therapy, it is enough to have a black and white ultrasound scanner. The use of the Doppler effect and

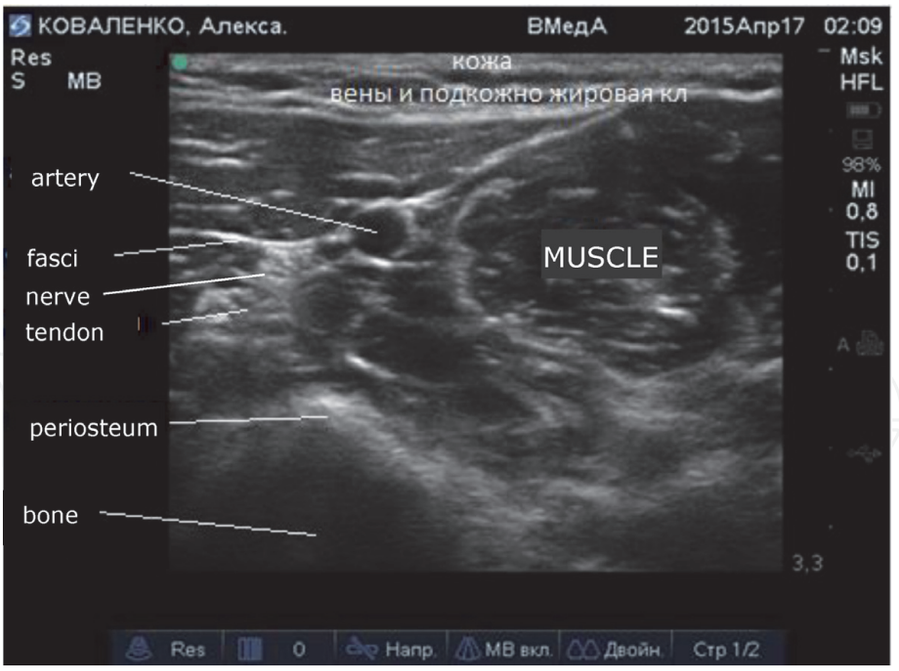

Figure 27.
Indicative ultrasound picture with symbols. Screen view of Edge (FujiFilm SonoSite Inc.'s handheld ultrasound machine).

color staining is effective, but practically not required for work on the limbs and in fact is only necessary for the ultrasound of tissues on the body and neck.

Ultrasonic sensor. Optimal, generally accepted and convenient for muscle visualization is a linear sensor with a width of about 38–50 mm and an operating frequency of 3–16 MHz. Sensors of smaller width narrow the ultrasound picture, thereby reducing the orientation space, some key points fall out of sight. This is especially noticeable when working on large muscle arrays, such as the thigh muscles.

Ultrasound picture of tissues (**Figure 27**).

1. Skin and bones represent the most superficial and deepest layers obtained by ultrasound navigation. Due to their acoustic properties, they tend to be hyperechogenic, that is, look light, bright. Tendons look almost the same.

2. Tendons are hyperechogenic. They have a characteristic fibrillar striated structure in the longitudinal and granular in the transverse section.

3. Arteries and veins. Anechoic (black) tubular structures. Arteries are pulsating and round, veins can be round or oval. A distinctive feature of veins is their easy compression when pressed by the sensor.

4. Muscle tissue is hypoechoic, compared to bones or tendons; its structure is flooded with bright spots. If these points are traced along the muscle, you can see how they come together and form tendons.

5. Nerves. With good resolution, you can see the structure of the nerve. Due to the presence of nerve fibers, its cut is similar to a honeycomb. The nerve, as a rule, is located next to the blood vessel and is considered as part of the neurovascular bundle.

Workplace organization. It is necessary to pay serious attention to the workplace, achieving its convenience, the correct location of the elements necessary for work.

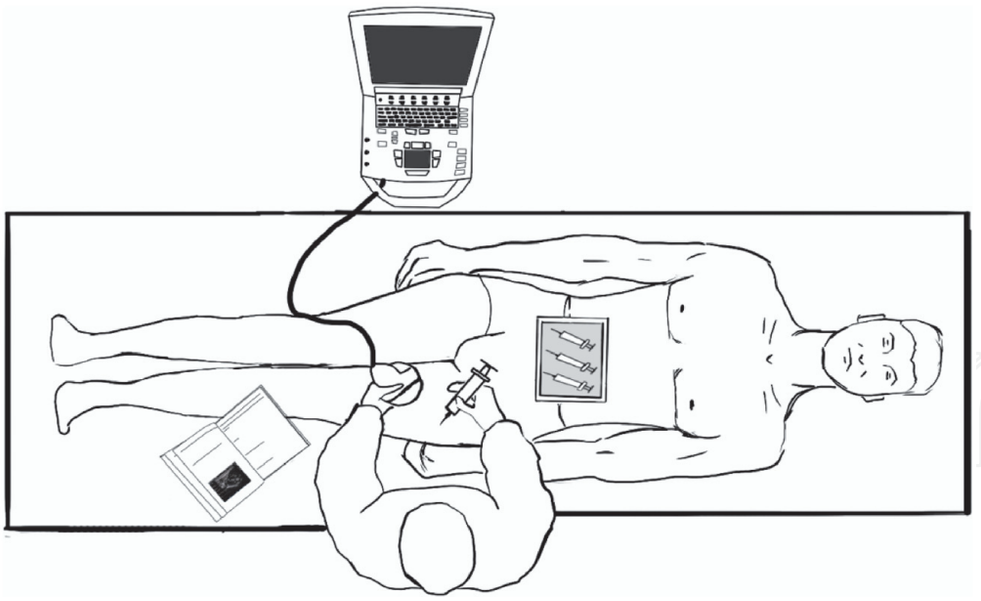

Figure 28.
Example of workplace organization for injection under ultrasound control.

The task of the doctor is not just to locate and verify the muscle, but also to make an injection. It should be borne in mind that the positioning of the limbs may be difficult due to spasticity and disturbance of the patient's consciousness. Picturing the muscles can be distorted by muscle contraction, etc. Therefore, it is preferable to use portable ultrasound scanner that allows you to easily move the machine around the patient. The most convenient location of the patient is between the doctor and the screen, the doctor does not have to turn around in order to study the ultrasound picture, and he can place all the necessary tools directly in front of him/her (**Figure 28**).

4.1.2 Operating procedure on the ultrasound machine

Pairing and orientation of ultrasound image and sensor. Coordination of needle and sensor movements, verification of the resulting image and orientation in the tissues of the body are developed over time. With the necessary experience, no problems in the orientation of the image will arise. For beginners, determining the coincidence of the sides of the sensor and the image on the monitor is an elementary but mandatory rule to get started. To do this, different simple methods are used: palpation of tissues, tapping on the edge of the working surface of the sensor, and positioning the label on the basis of the sensor.

Work with instrument settings. The skill of working with the parameters of the device also plays an important role. If for examining of some muscles (quadriceps femoris), special settings are not required, and then when scanning some other – the quality of the settings can affect the effectiveness of the injection. Additional image adjustment may require the location of the long extensors of the thumb and toes, the posterior tibial muscle, as well as the study of the muscles of the foot.

There are several basic settings for ultrasound imaging. Depending on the instrument, adjustments can be made manually or partially automatically.

The main adjustment parameters include:

1. Imaging modes:

    - B-mode – the main imaging mode in which anatomical tissues and organs are displayed in real time.

- Musculoskeletal (Msk) mode, optimal for muscle examination.

2. Depth in most cases, the optimal depth is greater than the depth at which the target muscle is located. This is because when scanning, it is necessary to focus on the surrounding markers—vessel, bone, tendon, etc. for examination of the upper limbs in adults; the average depth are the following:

   - Muscles of the shoulder girdle-up to 4 cm.
   - Shoulder muscles-up to 4–6 cm.
   - Upper third of the forearm 3.3–4 cm.
   - The middle and lower third of the forearm-3.3 cm.
   - Muscles of the hand-up to 2 cm.

3. Frequency (frequency/THI) – the wave frequency is directly related to the ability to penetrate into tissue. It should be remembered that the higher the frequency, the faster the tissue absorption and shallower penetration of the signal, the lower the frequency, the greater the signal immersion. On average, the optimal frequency for the muscles of the shoulder girdle, shoulder, and forearm is 7–8 MHz, for the muscles of the hand from 10 MHz.

4. Focus (focus). Focus on a specific object from the overall scan pattern, allowing for higher contrast and resolution of the object.

5. Brightness (gain). This is the ability to amplify all signals from the entire image. It is perceived as the increased brightness of the picture. It should be noted that with excessive amplification, tissue boundaries may be fuzzy and interference may occur.

In addition to the basic adjustments, there are additional ones that can be used to change the power of the ultrasonic wave, improve the quality and overview of the image, change its profile, remove image interference, etc.

4.1.3 Types and methods of needle insertion under ultrasound control

1. Way to № 1. Transversely to the ultrasound beam.

   The needle is inserted at an angle to the plane of the sensor and, accordingly, transversely to the plane of the ultrasonic beam. The thickness of the ultrasound beam is 2–3 mm. Therefore, when moving the needle, the researcher sees only the displacement of tissues from it and only that part of it, or the slice that passed through the beam (**Figure 29**). This method, despite the limitations of needle visibility, is convenient, easy to learn and most often used in practice.

2. Way to № 2. In the plane of the ultrasonic beam (longitudinally). Introduction of the needle from the end of the working surface of the sensor at an angle. In this case, the entire needle is in the plane of the beam and is fully visible (**Figure 30**).

   This method has some limitations: even a slight change in the angle of the sensor relatively to the skin or its displacement leads to the loss of the needle

from the plane of the beam and, accordingly, its image on the screen. In addition, it creates the need for the passage of the needle through the adjacent muscles and other formations.

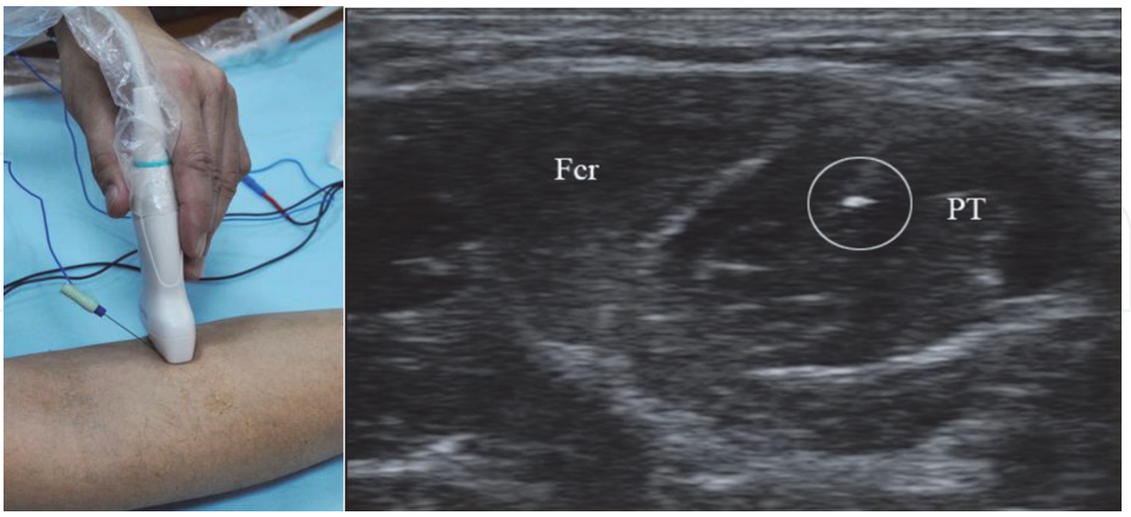

Figure 29.
Relative positions of the needle and the sensor introduction in a transverse slice of the needle as a point in the round pronator.

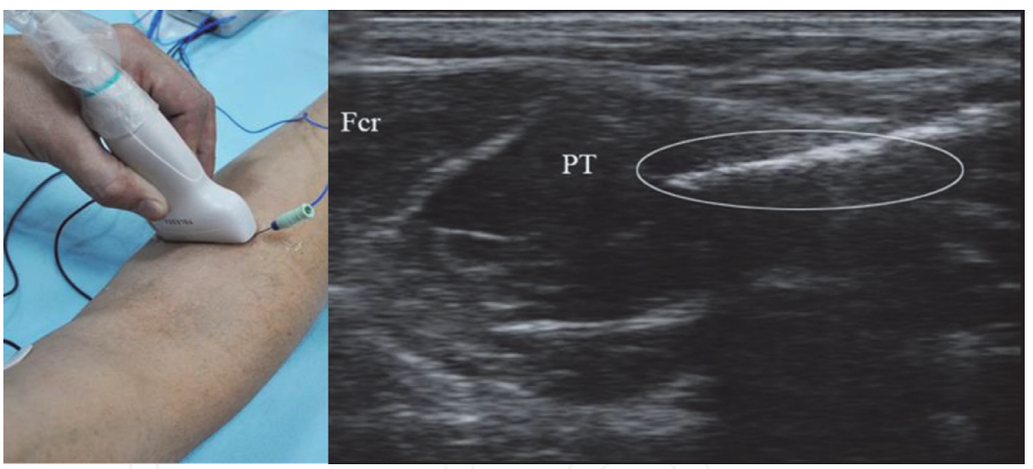

Figure 30.
The relative position of the needle and sensor in the longitudinal introduction and the needle along the ultrasound beam in a circular pronator.

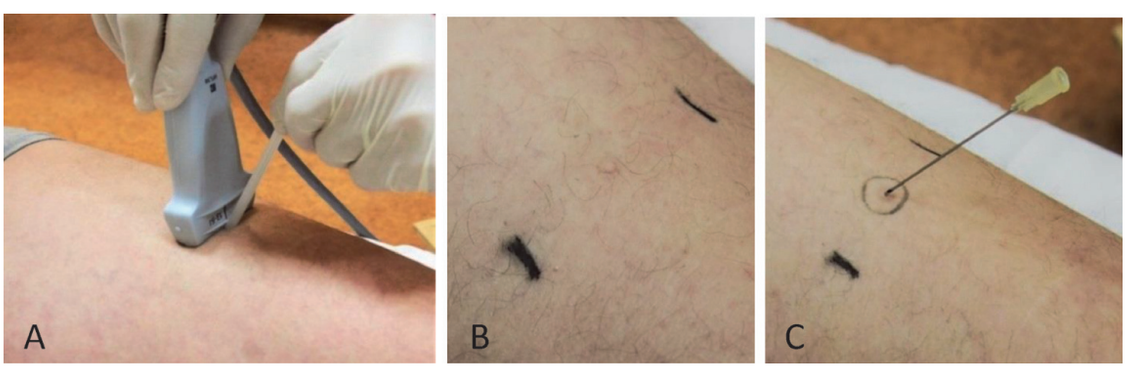

Figure 31.
(A) Tissue marking, (B) needle cap pressure mark on skin, and (C) inserting the needle into the center of the marking.

3. Way to № 3. Needle insertion after ultrasound control.

In a situation where it is impossible to simultaneously hold the sensor and insert the needle into the tissue, you can use the following method:

Determine the sensor object and depth of injection. Without removing the sensor, put a mark on the edge of the sensor. It is very convenient to use a sterile tip of the cap from the injection needle as a marker (**Figure 31A**). When you press the cap, a clear imprint in the form of a circle remains on the skin (**Figure 31B**). The needle is inserted into the circle on the skin to a measured depth (**Figure 31C**).

Tissue traction. Sometimes, for various reasons, it is impossible to get a full image of the needle on the screen of the ultrasound machine. In this case, you should focus on the tissue traction that occurs when the needle passes. This effect can be enhanced by light oscillating movements of the needle. This technique allows with a certain degree of error to understand at what depth and in what place on the screen the end of the needle is.

Aseptica. Introduction of drugs under ultrasound control requires compliance with the rules of asepsis. To do this, there are several different methods of treatment and protection: sterile gloves for the performer, sterile covers for the sensor, sterilizer for the sensor, sterile gel, aseptic solutions for the sensor, and the patient's skin.

For practical execution of the procedure, the scanner sensor can be protected by a sterile disposable cover, which has an adhesive base inside for fixing to the working part of the sensor. The adhesive base itself in this case also replaces the gel for ultrasound. Sterile cover can be replaced with a sterile glove, and instead of the adhesive base, you can use usual gel, which is applied to the working part of the sensor and the inner surface of the glove. Fixation of the glove on the handle of the sensor is performed using a patch (**Figure 32**).

Gel. Sterile ultrasonic gel is used for invasive manipulations under ultrasound control. Release form is sachets of 15 g, so when conducting the therapy even on one limb, you must have a few packages.

Treatment of the injection field. The patient's skin should be treated with a solution of 0.015% chlorhexidine.

Sensor processing. Treatment of the sensor with alcohol is undesirable. This causes damage to the rubber coating of the work surface and premature failure of the sensor. To sterilize the sensor, special solutions of the Sani-Cloth series are used and chlorhexidine can be used.

Figure 32.
Sensor in sterile case.

4.1.4 Methods of administration of BoNT in the intramuscular motor endpoint

Neuromuscular transmission is carried out by axon terminals in limited areas of intramuscular motor endpoint (IME). Accurate introduction to IME makes botulinum therapy more effective. The distribution of IME in the left and right limbs is identical; it does not depend on gender and age. The number of muscle motor points depends on the complexity of its functions and does not depend on its mass [47].

After finding a muscle using ultrasound navigation to orient the IME projection of the corresponding muscles on the human body, use the location map or find them using electroneuromyography (EMG). A cutaneous bipolar stimulating electrode is used to search for muscle IME. The study is carried out at a current strength of 5–10 mA and a frequency of 2 Hz [47–49]. The use of location maps in combination with ultrasound navigation significantly increases the effectiveness of treatment (**Figures 33–40**) [47].

4.1.4.1 Complex treatment of spasticity

Given the timing of the development of spasticity and the risk of complications, which in the future significantly reduces the effectiveness of rehabilitation and increases the cost of treatment, treatment of spasticity should be started when just its first signs appear. The period requiring special attention for early diagnosis and treatment is between 3 and 12 weeks after brain damage. In severe paresis, the period of occurrence of spasticity may coincide with the first signs of muscle strength and purposeful movement [50, 51].

Basically, all the drugs BoNT produced in the world are standardized to the 100-unit equivalent of Botox. The only drug that stands out from this series is Dysport. All drugs, except for Dysport, are similar in dosage of introduction to the relevant muscles and multiples of 100 Units. The drug Dysport is 500-unit drug and is

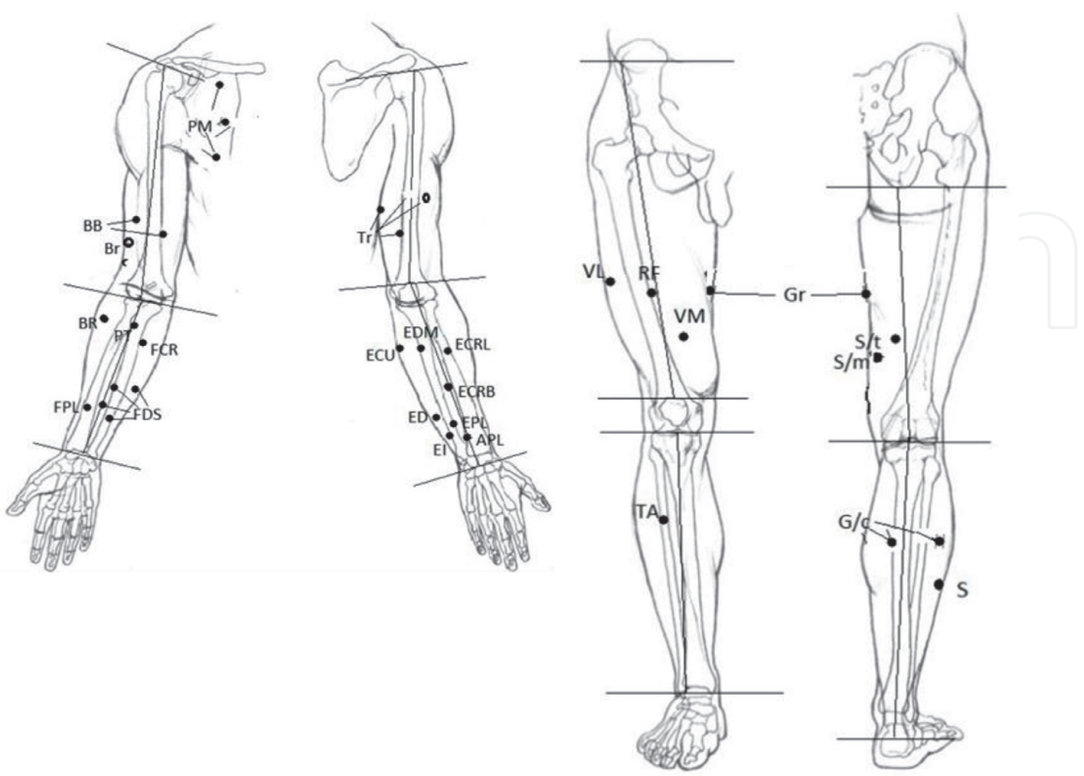

Figure 33.
Location map of muscle motor points for botulinum toxin injections in the treatment of spasticity.

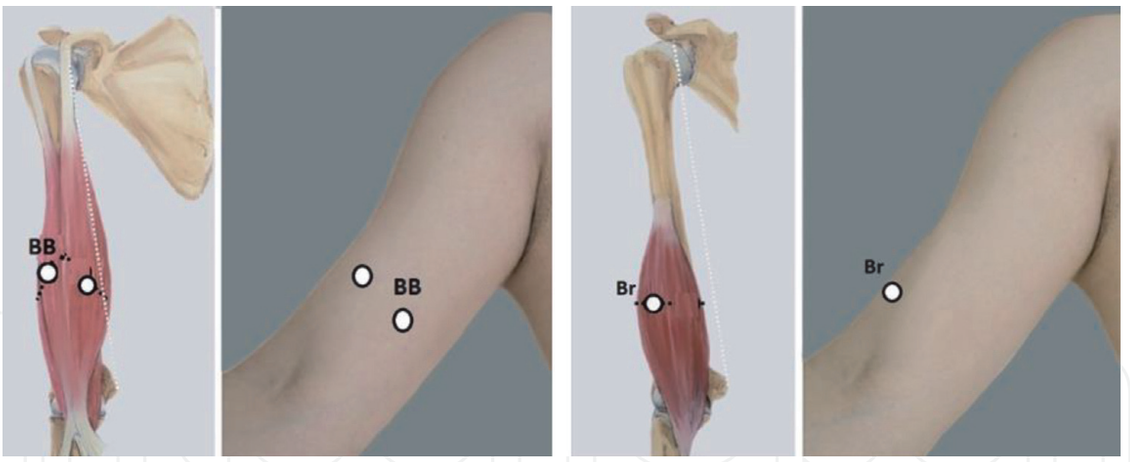

Figure 34.
Image of anatomy m. Biceps brachii (BB) and m. Brachialis (Br) and projections of their IME on the surface of the body.

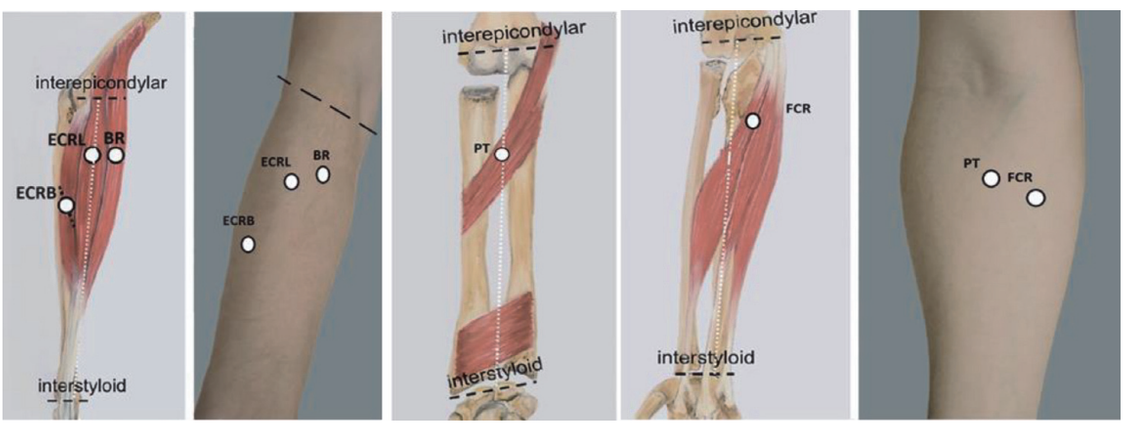

Figure 35.
Image of anatomy of m. Brachioradialis (BR), Extensor carpi radialis longus (ECRL), Extensor carpi radialis brevis (ECRB), Flexor carpi radialis (FCR), m. Pronator teres (PT), and projections of their IME on the surface of the body.

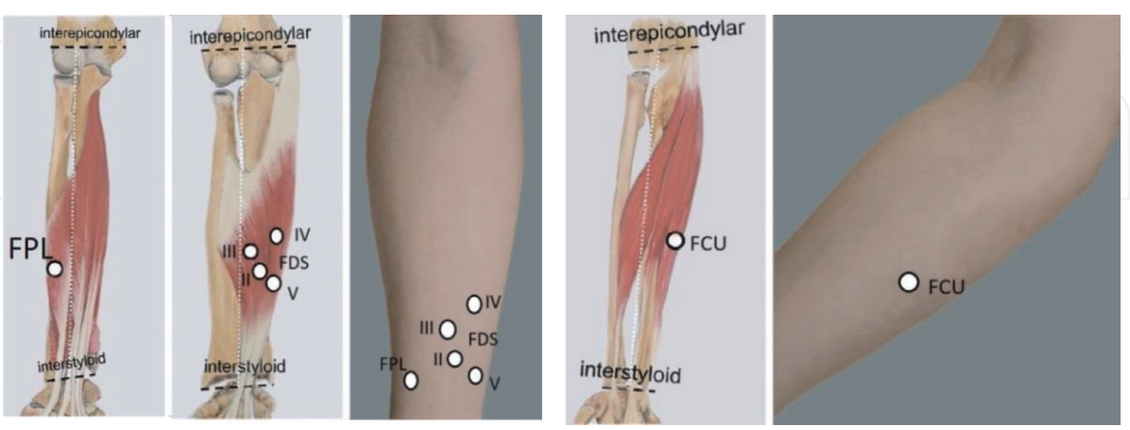

Figure 36.
Image of anatomy of m. Flexor pollicis longus (FPL), m. Flexor digitorum superficialis (FDS), m. Flexor carpi ulnaris (FCU), and projections of their IME on the surface of the body.

significantly different from the 100 individual drugs, dosage of the injection in the muscle (**Tables 2** and **3**) [7, 50–54].

To optimize the calculation of drug consumption and prognosis of needs, it is advisable to use models of patients based on the frequency of participation in the

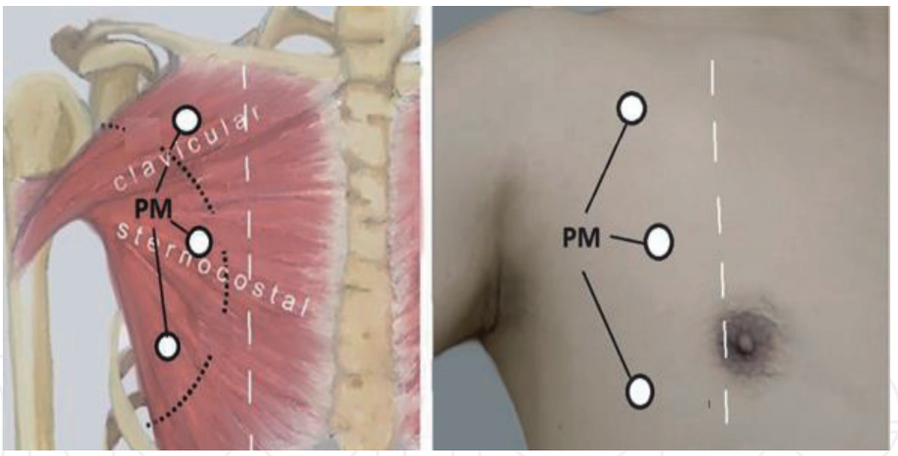

Figure 37.
Image of anatomy m. Pectoralis major (PM) and projections of their IME on the surface of the body.

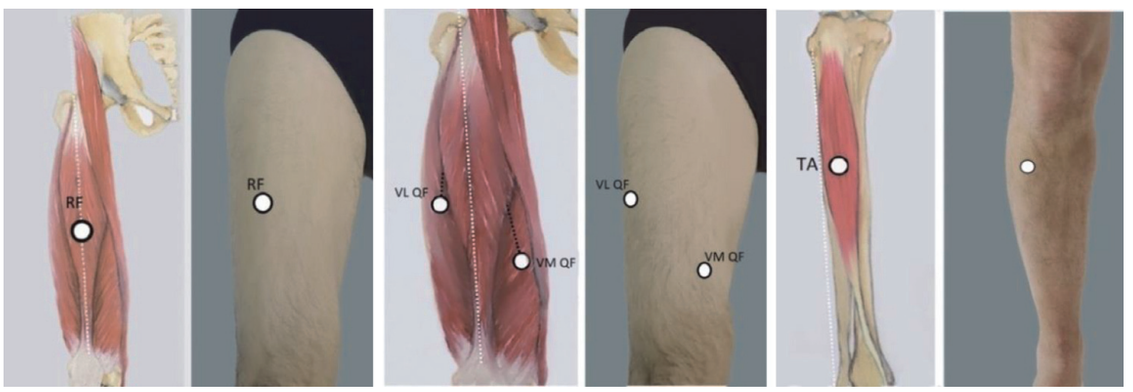

Figure 38.
Image of anatomy mm. Vastus lateralis (VL), Vastus medialis (VM), m. Rectus femoris (RF), m. Tibialis anterior (TA), and projections of their IME on the surface of the body.

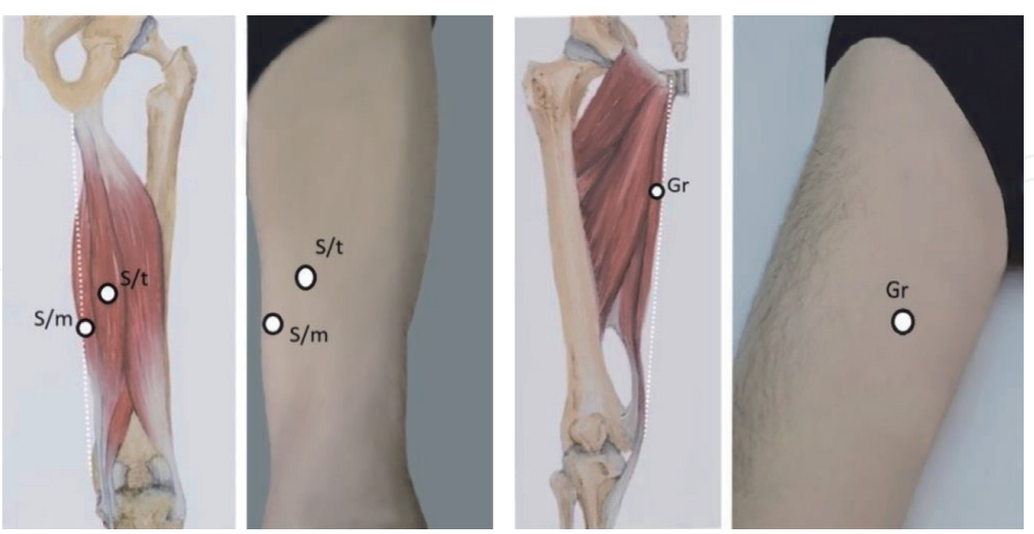

Figure 39.
Image of the anatomy of m. Semimembranosus (S/m), m. Semitendinosus (S/t) m. Gracilis (Gr), and projections of their IME on the surface of the body.

formation of a pattern of specific muscles (**Tables 2** and **3**). The use of these models allows you to accurately determine the required amount of the drug and the cost of treatment of spasticity.

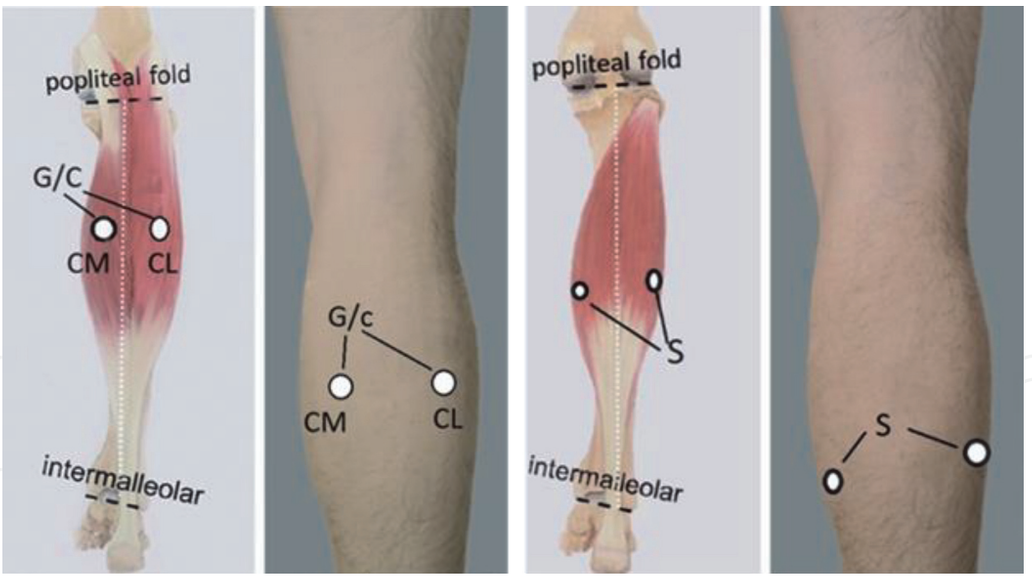

Figure 40.
Image of anatomy mm. Gastrocnemius (G/c), Soleus (S), and projections of their IME on the surface of the body.

Model	Pattern of spasticity	Muscles	100 Units of the BoNT, U	Dysport, U
1A	Flexion of the wrist, fingers and thumb	Flexor digitorum superfacialis	60	200
		Flexor digitorum profundus	60	200
		Flexor pollicis longus	20	60
			140	**460**
2A	Flexion of the wrist, fingers and thumb	Flexor digitorum superfacialis	60	200
		Flexor digitorum profundus	60	200
		Flexor pollicis longus	20	60
	Pronation of the forearm	Flexor carpi radialis *Sometimes one of two*	60	200
		Pronator teres	30	80
			230	**740**
3A	Flexion of the wrist, fingers and thumb	Flexor digitorum superfacialis	60	200
		Flexor digitorum profundus	60	200
		Flexor pollicis longus	20	60
	Pronation of the forearm	Flexor carpi radialis *Sometimes one of two*	60	200
		Pronator teres	30	80
	Elbow flexion	Brachialis *More often one-two from three*	80	300
		Brachioradialis *(BB less often than others)*	100	400
		Biceps brachii (BB)	100	400
		Very rarely are all muscles involved, so most often the average dosage	**410**	**1600**
4A	Flexion of the wrist, fingers and thumb	Flexor digitorum superfacialis	60	200
		Flexor digitorum profundus	60	200
		Flexor pollicis longus	20	60
	Pronation of the forearm	Flexor carpi radialis *Sometimes one of two*	60	200
		Pronator teres	30	80
	Elbow flexion	Brachialis *More often one-two from three*	80	300
		Brachioradialis *(BB less often than others)*	100	400
		Biceps brachii (BB)	100	400
	Impossibility of shoulder retraction and arm extension	Pectoralis major	100	400
		Very rarely are all muscles involved, so most often the average dosage	**530**	**1600**

Table 2.
Models of patients with spasticity in the upper limb.

Model	Pattern of spasticity	Muscles		100 Units of the BoNT, U	Dysport, U
1L	Dynamic	Semitendinosus Semimembranosus	*Almost always*	80 100	300 400
		Gracilis Biceps femoris	*Often* *Very rarely*	80 140	200 500
		Very rarely are all muscles involved, so most often the average dosage		**250**	**800**
2L	Static	Gastrocnemius caput mediale (G/c c.m.)	*Almost always*	100	400
		Tibialis posterior Soleus Tibialis anterior	*Most often one of the muscles in combination with G/c c. m.*	100 80 80	400 300 300
		Very rarely are all muscles involved, so most often the average dosage		**200**	**700**
3L	Dynamic + Static	Semitendinosus Semimembranosus	*Almost always*	80 100	300 400
		Gracilis Biceps femoris	*Often* *Very rarely*	80 140	200 500
		Gastrocnemius caput mediale (G/c c.m.) Tibialis posterior Soleus Tibialis anterior	*Almost always* *Most often one of the muscles in combination with G/c c. m.*	100 100 80 80	400 400 300 300
		All muscles are never involved, so the average dosage is		**450**	**1500**
4L	Static + Flexion of fingers and big toe	Gastrocnemius caput mediale (G/c c.m.) Tibialis posterior Soleus Tibialis anterior	*Almost always* *Most often one of the muscles in combination with G/c c. m.*	100 100 80 80	400 400 300 300
		Flexor digitorum longus (FDL) Flexor halucis longus (FHL) Flexor digitorum brevis (FDB) Flexor halucis brevis (FHB)	*FDL and FHL are more common than FDB and FHL, and FDL is more common than FHL.* *A rare combination of long and short flexors of the fingers.*	40 40 100 30	140 140 400 100
		All muscles are never involved, so the average dosage is		**300**	**1000**
5L	Dinamic+ Static + Flexion of fingers and big toe	Semitendinosus Semimembranosus	*Almost always*	80 100	300 400
		Gracilis Biceps femoris	*Often* *Very rarely*	80 140	200 500
		Gastrocnemius caput mediale (G/c c.m.) Tibialis posterior Soleus Tibialis anterior	*Almost always* *Most often one of the muscles in combination with G/c c. m.*	100 100 80 80	400 400 300 300

Model	Pattern of spasticity	Muscles		100 Units of the BoNT, U	Dysport, U
		Flexor digitorum longus (FDL)	*FDL and FHL are more common than FDB and FHL, and FDL is more common than FHL.*	40	140
				40	140
		Flexor halucis longus (FHL)		100	400
		Flexor digitorum brevis	*A rare combination of long and short flexors of the fingers.*	30	100
		Flexor halucis brevis			
		All muscles are never involved, so the average dosage is		**500**	**1500**

Table 3.
Models of patients with spasticity in the low limb.

The treatment scheme of spasticity with the complex use of peripheral muscle relaxants (BoNT) and central muscle relaxants (baclofen) action may also be effective. Baclofen should be prescribed 25 ± 3 days after the introduction of BoNT. This treatment scheme provides a sufficient clinical effect for 110 ± 10 days after the injection session, which is 14–25 days longer than the action of BoNT in monotherapy. With an average spasticity treatment time of 2 years, this combination reduces the number of injection sessions from 7 to 5.

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