Phenol as a neurolithic agent in the treatment of focal and multifocal spasticity

O fenol como agente neurolítico no tratamento da espasticidade focal e multifocal

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ABSTRACT

The pyramidal syndrome occurs in central nervous system injuries that affect the corticospinal pathways and are defined by the triad of muscular weakness, increased myotatic reflexes and spasticity, which is defined by the involuntary increase in resistance to passive movement that varies in intensity according to the velocity of joint movement. When spasticity interferes with the patient's functioning, making it difficult to actively move, causing pain, or making it difficult to receive care from others, treatment must be stated. For the treatment of generalized spasticity or for large portions of the body, drug intervention may be used either orally or intrathecally, depending on available resources, but undesirable and variable intensity side effects may occur, especially impairment of attention or awareness, which further delays the rehabilitation process. Focal treatment of spasticity utilizes botulinum toxin or nerve blocks with phenol or alcohol. This article reviews the literature on the most suitable techniques for performing phenol neurolysis. Neuromuscular blockade with phenol is an effective treatment, with immediate action, low cost, prolonged duration and few adverse effects when the regular care of application is respected.

Keywords: Phenol, Muscle Spasticity, Nerve Block, Therapeutics

RESUMO

A síndrome piramidal ocorre nas lesões do sistema nervoso central que afetam as vias corticoespinhais e são definidas pela tríade de fraqueza muscular, aumento dos reflexos miotáticos e espasticidade, que é definida pelo aumento involuntário da resistência ao movimento passivo cuja intensidade varia com a velocidade do movimento. Quando a espasticidade interfere na funcionalidade do paciente, dificultando o movimento ativo, causando dor ou dificultando a prestação de cuidados por terceiros, há necessidade de iniciar seu tratamento. Para o tratamento da espasticidade generalizada ou de grandes porções do corpo, a intervenção medicamentosa ocorre por via oral ou intratecal, conforme os recursos disponíveis, mas efeitos colaterais de intensidade variável e indesejáveis podem ocorrer, especialmente o comprometimento da atenção ou da consciência, que prejudicam o processo de reabilitação. O tratamento focal da espasticidade utiliza a toxina botulínica ou os bloqueios nervosos com fenol ou álcool. Este artigo revisa a literatura sobre as técnicas mais adequadas para realizar a neurólise com fenol. Os bloqueios neuromusculares com fenol são um tratamento efetivo, de ação imediata, baixo custo, duração prolongada e de poucos efeitos adversos quando são respeitados os cuidados regulares de aplicação.

Palavras-chave: Fenol, Espasticidade Muscular, Bloqueio Nervoso, Terapêutica

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INTRODUCTION

Spasticity is a motor control disorder resulting from injury to the upper motor neuron that presents as involuntary muscle activation. Several common diseases in our environment can cause lesions in the upper motor neuron and, consequently, spasticity. Thus, the management of spasticity is important in the daily clinical practice of physiatrists, neurologists, physiotherapists, occupational therapists and all health professionals involved in the rehabilitation of neurological patients.

Clinically, spasticity is characterized by increased speed-dependent muscle tone and is accompanied by other signs of upper motor neuron syndrome such as clonus, spasms or simultaneous contractions of antagonistic groups.

The clinical sign characteristic of the physical examination is the "pocketknife signal", in which the body segment that is passively moved with fast angular velocity exhibits greater resistance at the beginning of the range of motion and abrupt reduction of endurance at the end.[1]

Spasticity can deteriorate the original clinical presentation of the underlying disease and negatively impact patient functioning as it can reduce range of motion, cause pain, disrupt sleep, and reduce functional capacity.

In more severe cases, patients evolve with muscle shortening, tendon retractions, orthopedic deformities and the need for surgical approaches, which increases morbidity and treatment costs. However, not all patients require treatment for spasticity.

Some patients use tonus augmentation to develop compensatory mechanisms and facilitate some functional activities, such as orthostatic posture and transfer, which makes it necessary to evaluate the real need of treatment.

Therefore, evaluation should be individualized, based on the functional objective agreed upon and prioritized by the patient and caregivers.[2]

The treatment of spasticity can be multimodal and combine several techniques: oral medications, chemical blocks (botulinum toxin and / or phenol), intrathecal baclofen pump, surgeries to correct deformities. It is essential to associate non-pharmacological measures such as physical therapy, occupational therapy and the use of orthoses.[3]

The choice of treatment modality depends on the characteristics of spasticity, especially if it is generalized, focal or multifocal. In generalized spasticity, the use of drugs with muscle relaxation effect is frequent; while in focal or multifocal spasticity, neuroleptic blockades, with phenol, alcohol or botulinum toxin, are the main treatment option.

This article highlights the neuroleptic blockade with phenol as an effective and inexpensive option for the treatment of focal and multifocal spasticity, especially in patients with large numbers of affected muscles, in whom treatment with botulinum toxin can become quite costly or exceed the maximum recommended dose.

It also discusses the chemical characteristics, usage history, mechanism of action, dose and techniques of the main application sites.[4] Indications for the treatment of spasticity are summarized in Chart 1.[5]

Chart 1. Indications for the treatment of spasticity[5]

Indication	*Examples*
Improvement of active range of motion	*Improvement of velocity and precision of movement; improvement of linear gait parameters and security or wheelchair propulsion. Better agility for transfers. Improvement in dexterity to reach and grab objects; more favorable conditions for sexual intercourse.*
Relief of symptoms and subjective aspects	*Reduction in pain intensity and sensation of spasticity associated stiffness. Improvement in self image.*
Better posture and control of involuntary movements	*Prevention of the installation and worsening of muscular contractures and deformities. Adequacy of periarticular muscle balance. Reduction of muscle spasms.*
Reduction of need of care	*Reduction of need of care for hygiene and selfcare, wearing na orthosis or assistive device, mobilization and positioning.*
Improvement of future responses rehabilitation	*Prevention of contractures, facilitation of active movement, better adherence to rehabilitation exercises and recommendation, improvement of self image.*

Chemical properties of phenol

Phenol is a chemical compound that contains carbolic acid, phenolic acid, phenyl acid, phenyl hydroxide, hydroxybenzene and oxybenzene.[6]

It is a very soluble substance in organic solvents such as alcohol or glycerol. Usually, aqueous solutions of phenol are used with concentrations between 3,0% to 7,0% (25% phenol in 60% glycerol solution diluted in 5% sterile water).[7]

History of clinical use of phenol

The earliest historical reports of the use of phenol in healthcare date back to the 1860s, when it was used as an antiseptic agent during surgical procedures. Later, at the beginning of the 20th century, the first applications of nerves for the treatment of neuralgias were performed. It was only in 1919 that phenol was first used for the treatment of spasticity by direct muscular application; then came the publications regarding intrathecal use and, finally, neurolyses of peripheral nerves.[8]

The use of the intrathecal route to block the anterior roots of the spinal nerves was discouraged over the years due to the significant complications reported:[9] nerve root damage, motor paralysis, spinal cord infarction, loss of sensitivity, meningitis, painful paresthesia and death.[10] In In view of this, in 1966, an alternative route of administration with motor point blocks was described, reporting an improvement in spasticity in 39 patients in the long term.[11]

Phenol blocks were widely used for the treatment of spasticity in the 1970s and 1980s; However, compared with botulinum toxin type A (BONT-A), phenol has been less widely used in the last decades, although it is effective as a denervation chemical agent, has immediate onset, low cost and often a duration equivalent to the effect of botulinum toxin type A (BONT-A).[10,12]

The loss of popularity of neurochemical blockade with phenol may be related to the lack of knowledge by many physicians about the techniques for its administration, the fear associated with its adverse effects and the simpler application of BONT-A.

It is now common sense that dysesthesia is very rare when nerves with predominance of motor fibers are blocked and, when painful symptoms arise, the alternatives for drug therapies are wider, phenol treatment in the form of a mixed procedure with toxin in cases of multifocal spasticity resurfaced. This offers the opportunity to treat more muscles in a single session without exceeding the doses of both agents.[13]

The combined use of phenol block was chosen as a possible treatment alternative by 16 out of 49 Brazilian medical doctors, from which 73% reported its use in 1 to 5 patients a week. The isolated use of phenolic block was described by 55% of the medical doctors, whereas 45% of them reported its use combined with botulinum toxin.

The most commonly blocked nerves are the anterior branch of obturator nerve (67.2% of the patients), motor branch of the sciatic nerve (35%), musculocutaneous nerve (20.6%), femoral nerve (18%), motor branch of the tibialis nerve (15%) and motor points in 10%.[14]

Mechanism of action of phenol

Phenol appears to have a dual mechanism of action. On the one hand, the drug acts as a local anesthetic on the gamma fibers and, on the other, produces chemical axoniotomesis, that is, it disorganizes the structure of the axons, although it still maintains the endoneural tubes.[9] It occurs proteolysis, injury to the lipid component of the cell membrane and separation of the myelin sheath from the axons, with consequent interruption of efferent signals from the hyperexcitable cells of the anterior horn of the spinal cord, through induced necrosis.[7,13,15]

The result is nerve interruption of the reflex arc and, consequently, decrease of muscle tone.[9] The effects of chemical neurolysis with phenol are not permanent, since functional reinnervation may occur in a period of months or years.[9,16,17]

Indication of the use of phenol

The indication of chemical neurolysis is the existence of spasticity with functional or painful impairment, which deranges the quality of the motor act or results in joint deformities.[18]

The treatment of spasticity significantly modifies the rehabilitation process since, whatever its topography, presence of severe pain to joint mobilization prevents effective rehabilitation. In patients with focal and multifocal spasticity, some studies indicate that phenol blockade would have a superior effect on traumatic brain injury and spinal cord injuries than in patients with stroke due to longer recovery times.[9]

Dose

The amount applied ranges from 0.6ml to 3.0ml per point.[19,20] Generally, it starts at low doses, with further increases to obtain more suitable effects. However, the patient and nerve to be treated should be individualized. The estimated lethal dose ranges from 8.5 g - 15.0 g and it is not recommended to administer more than 1 g in 24 hours, ie 20.0 ml of 5% phenol.[17,21,22]

Adverse effects

The acidity of the phenol may cause local inflammatory effects. In addition, it has a low potential for tissue diffusion and should therefore be injected as close as possible to the target nerve for better results and less local adverse effects.[7]

If phenol is injected near sensory fibers, it can cause dysesthesia and neuropathic pain, which occurs in 2-32% of procedures in adult patients.[23,24]

Such an adverse effect may last up to 4 months and affect the relationship of the body segments, implying changes in posture and may compromise the gains obtained by the procedure in the daily activities of the patients.

Therefore, phenol neurolysis for the treatment of spasticity should be performed on nerves with exclusive or predominantly motor function. Procedures guided by electrostimulation may reduce the risk of involvement of the sensory fibers.

Other adverse effects include edema, cutaneous erythema, deep venous thrombosis and local infection.[17,22,25]

Immediately after application, patients may experience headache, feeling of intoxication, alcoholic breath, nausea or vomiting. These side effects usually remit spontaneously in less than one hour.[7,26]

Materials and equipment

In order to have a greater effect of reducing spasticity and pain, as well as reducing the occurrence of adverse effects, neuromuscular blockade with phenol requires that the application of the drug be located more precisely with the target nerve.

The procedure should be guided by an imaging resource, usually ultrasonography, or by electrostimulation, which will be the focus of the following material guidelines:

- Teflon coated needle for plexus anesthesia, gauge 22 to 28, the length of which should be based on the procedure site (1.2 to 3.0 inches);
- 5ml syringe with needle extension connector - extension needed for better needle stabilization during drug aspiration and injection;
- Electrostimulator with 1 millisecond electrical stimulus and square wave pulse;
- Electrode gel for connection of the positive pole of the electrostimulator;
- Gauze and alcohol 70% for hygiene of the procedure site;
- 5% Phenol for injection.

Technique

In the next paragraphs, we described four widely discussed neurolysis techniques of peripheral nerves: the ansa pectoralis, the musculocutaneous nerve, the obturatory nerve and the motor branches of the tibial nerve. Because the median nerve has a large sensory component and the risk of secondary dysesthesia is significant, it will not be addressed in this review.[21]

Despite the usual recommendation in the most recent literature of techniques use ultrasound and electrostimulation simultaneously to better location the nerve to be addressed, since it offers a direct visualization of the nerve and other vascular structures to be avoided. This combination also promotes the reduction of the required volume of phenol, once precise nerve localization can be achieved.[16,27,28]

However, in view of the need for equipment and ultrasound skills, this combined location is not part of the scope of this review, which is to describe a more practical and accessible approach. In addition, the anatomical approach based on surface milestones combined with electrostimulation was shown to be as effective as the combined approach.[28]

After proper cleaning of puncture site with 70% alcohol, the electrostimulator is connected to the plexus anesthesia needle and the electrode is attached to the patient's body, preferably close to the nearest bony prominences. The syringe with 5% phenol content should be connected to the needle, filling the entire route with the the drug.

The needle is inserted according with surface anatomy and directed to nerve location. The electrostimulator is adjusted to achieve maximum contraction of the desired muscles with minimum current. This process start with a more intense current, around 3.0 to 4.0 mA, which causes both direct stimulation of muscles and nerves.

The progressive mobilization of the tip of needle and reduction of the electric current define the position of the needle. The needle is considered correctly placed if the necessary rheobase, which is the current intensity that can cause muscle contraction, is 1.0 mA or less; a higher rheobase indicate incorrect location, and the needle was moved slightly in all directions, until the proper rheobase was obtained.

Phenol volume to be injected ranges from 0.6 to 3.0 mL,[7] indicating the effectiveness of neurolysis.[17,18,29] However, the response should be verified and, if necessary, a larger dose can be injected, given that the effect of the phenol is dose-dependent.[7]

Not all nerves should be approached with phenol in patients with full sensitivity, preferring those with exclusive motor component or

with little sensitive component. In patients with complete spinal cord injury, for example, the unlikely occurrence of neuropathic pain as an adverse effect permits the blockage of nerves with a much larger sensitive component. When phenol is administered, there is a virtually instantaneous decrease in muscle tone.

Phenol has a long period of action ranging from 6 months to 1 year, or even up to a year and a half. Effect duration varies according with the concentration of the drug, the volume used, technique used and concomitant therapies.[27,30]

Anatomical ocations

Obturator nerve

The anterior branch of the obturator nerve innervates the long and short adductor and gracile muscles, while the posterior branch innervates the adductor magnum and external obturator muscles. This is probably the nerve most commonly addressed in chemical neurolysis, resulting in reduced spasticity of adductors.[31]

In this article, we will describe positive and negative aspects of three different techniques addressing this nerve. The use of ultrasound to guide the procedure implies success rates of 90% to 100%, but no comparative studies with other localization techniques have been performed.[32]

Pubic or simplified classical approach

Initially described by Labat in 1922, this technique was simplified by Park in 1967,[33] which used only anatomical landmarks. In 1984, Gasparich et al.[34] included electrostimulation to the technique for better localization of the obturator nerve.

For this procedure, the patient is placed in the classical lithotomy position, that is lying supine with slightly abducted and externally rotated thigh.[35]

The needle is inserted at 2.0 cm lateral and 2.0 cm caudal of the pubic tubercle, with a small cephalic deviation towards the upper branch of the pubic bone. Then, the needle slides to the inferior margin of the pubic bone and is deepened posterior and superiorly for 2.0 cm. At this point the electrostimulator is used for more accurate localization of the obturator nerve.

In this technique, the obturator nerve is reached before the origin of anterior and posterior branches. In the traditional approach, a puncture is performed near the pubic tubercle, which can cause pain on contact with the periosteum, bone and ther is a risk of injury to organs such as the bladder or vagina,[19] so other techniques have been studied to address the anterior and posterior branches separately and distally to the inguinal area.

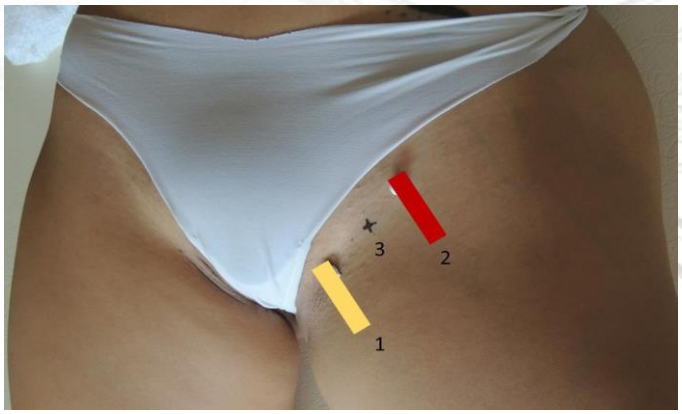
Inguinal approach

Choque et al.[35] described this technique to locate the anterior and posterior branches separately. They performed a previous test with fresh cadavers and later performed blockages in patients with the following procedure: with the patient placed in the supine position and the thighs slightly abducted and externally rotated, the tendon of the long adductor muscle is sought (the palpable tendon more superficial) and the femoral artery pulse on the inguinal skin fold as reference points.

The midpoint between these points is marked on the inguinal fold, which corresponds to the center of an easily palpable groove between the artery and the long adductor muscle.

The needle is inserted at this point at an angle of 30° in the cephalic direction, with electrostimulation, until the contractions of the long adductor and gracilis muscle, which indicates the anterior obturator nerve branch.

After blocking, the needle may be slightly deepened, with a slight lateral deviation until contraction of the adductor muscle is observed (probable site of the posterior branch of the obturator nerve) (Figure 1).



1) Tendon of the long adductor muscle (the most superficial palpable tendon), 2) Pulse of the femoral artery, 3) Place where the needle is inserted at an angle of 30 ° in the cephalic direction

Figure 1. Location of the anterior branch of the obturator nerve

This technique is faster and causes less discomfort for the patient compared to the classic technique, in addition to obtaining the same quality in the block.[35]

Interactors approach

The technique described by Wassef in 1993[36] consists of the interadductors approach of the anterior branch of the obturator nerve and was an easier and more accurate approach compared to the classical technique, presenting the same success rate in the block.[6,37,38]

It comprises of the insertion of the needle 1.0 cm below the tendon of the long adductor muscle and 2.0 cm lateral to the pubic arch with the patient in a lithotomy position (Figure 2). The needle should be inserted with a slightly posterior inclination.

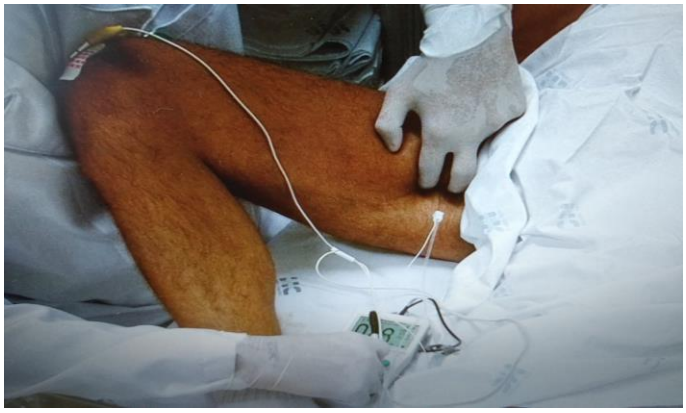


Figura 2. Positioning of the electrostimulation needle between long and magnus adductor muscles

Tibial nerve

It is the second most commonly blocked nerve used to treat spasticity of the calf.[6] The tibial nerve innervates the gastrocnemius, soleus, popliteal, extensor digitorum longus, extensor longus hallux, and several intrinsic foot muscles.

Blockade of this nerve has been used to treat the equinovarus foot, which is mainly caused by spasticity of solear, gastrocnemius and tibialis posterior,[29] resulting in improvement in orthostatism and gait, reducing plantar flexion and varus.[39,40]

However, the main trunk of the tibial nerve is not exclusively motor, due to the presence of sensory fibers that innervate the sole of the foot[29] and there may be adverse effects of pain and dysesthesia,[28]

however a recent series of patients treated with this approach presented neuropathic pain in only 2.2%.[41]

These recent studies have focused on the location of motor branches for greater selectivity and fewer adverse effects.[29,39]

Branches from the tibial nerve to the gastrocnemius

These motor branches originate obliquely from the trunk of the tibial nerve at a average distance of 3.3 ± 1.2 cm above the horizontal line of the upper margin of the fibular head.

The needle should be inserted at the lower border of these branches (located by electrostimulation), 1.0 cm medial and lateral to the tibial nerve above the horizontal line formed by the upper margin of the fibular head, at a depth of approximately 1.0 to 3.0 cm[39] (Figure 3).

The tibial nerve can be located using the electrostimulator or its position defined approximately by a line that goes from the Achilles tendon to the midpoint of the horizontal line formed by the head of the fibula.

Motor branches of tibial nerve to soleus and posterior tibial muscles

There are few studies of selective blocks of these branches in the literature since they are very small and further hinders the localization.

Deltombe et al.[29] located these branches by tomography and defined the coordinates that were later used for blockade with the aid of an electrical stimulator in 12 patients with lower limb spasticity.

The solear muscle motor branch was located 10.0 ± 5.0 mm below the horizontal line running through the fibula head, 17.0 ± 9.0 mm lateral to a midline in the calf that goes from the midpoint of the popliteal fossa to the Achilles tendon and 47.0 ± 4.0 mm deep (Figure 4).

The motor branch of the posterior tibial muscle was located at 45.0 ± 6.0 mm inferior to the fibular head, 17.0 ± 8.0 mm lateral to a midline in the calf from the midpoint of the popliteal fossa to the Achilles tendon and 47.0 ± 4.0 depth (Figure 4).

Ansa pectoralis

The ansa pectoralis corresponds to the medial and lateral pectoral nerves, both branches of the brachial plexus,[18] which innervate the pectoralis major and minor muscles.

The spasticity of the pectoralis major produces a movement toward adduction and internal rotation of the shoulder, which limitats abduction of the arm and causes regional pain[18,42,43] as well as limitation for hygiene and the change of clothes.

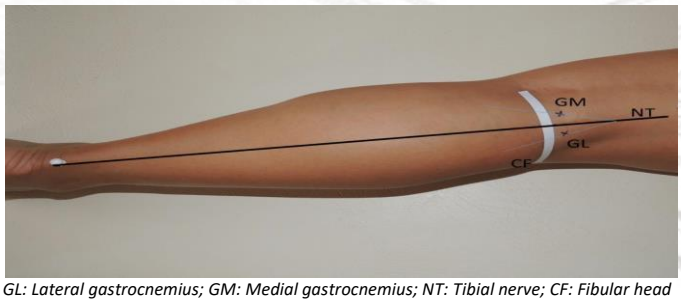
The point of insertion of the needle corresponds to the union of the middle third to the external third of a line that connects the upper part of the axilla to the sternoclavicular joint[18] (Figure 5), which is reasonably close to the proposal of Özel et al.[43] with a direct approach to the nerves medial and lateral pectorals from a cadaveric study.[31]

The results of the neurological block show a significant reduction of spasticity and shoulder pain.[18]

Musculocutaneus nerve

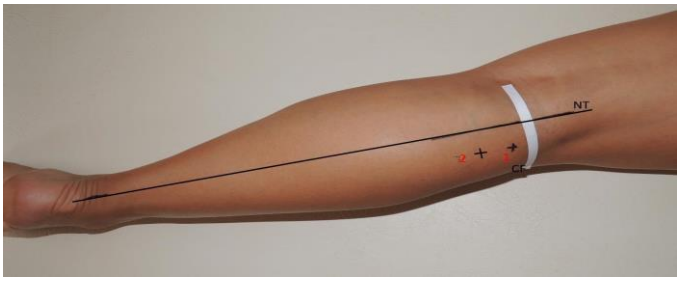
The musculocutaneus nerve is responsible for the innervation of the three muscles in the anterior aspect of the arm: the coracobrachialis, the biceps brachii and the brachialis, which are the most potent flexors of the elbow. This nerve can be identified to 2 digital pulps distally to the insertion of the pectoralis major muscle in the upper arm.

The brachial artery is palpated and the needle is inserted 1.0 cm anterior to the artery and directed anterolaterally until the biceps contraction occurs[22,44] (Figure 6).



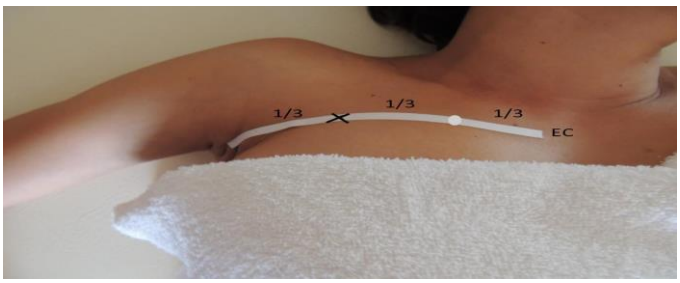
GL: Lateral gastrocnemius; GM: Medial gastrocnemius; NT: Tibial nerve; CF: Fibular head

Figure 3. Localization of motor branches of the tibial nerve to the medial and lateral



1) Cutaneus localization to motor branch of the tibial nerve to the soleus muscle, 2) Cutaneus localization to motor branch of the tibial nerve to the tibialis posterior muscle. NT: Tibial nerve; CF: Fibular head

Figure 4. Motor branches to the soleus and tibialis posterior muscles



EC: sternoclavicular joint

Figura 5. Ansa pectoralis. Union of the middle to external third of a line from the upper border of the axila and the sternoclavicular joint

Figure 6. Musculocutaneus nerve. Two digital pulps distally from the insersion of pectoralis major, anterior to the braquialis artery

The nerve is immediately posterior to the biceps braquii. It is advisable to be aware of the contraction pattern produced by electrical stimulation, since the stimulation of the median nerve stimulus can contract the flexor muscles of the fingers and the carpus, producing some degree of elbow flexion, given the origin of these muscles in the medial epicondyle of the humerus. In this way, both the stimulation of the musculocutaneous nerve and the median nerve can cause elbow flexion.

The stimulation of the musculocutaneous nerve, however, does not result in flexion of the wrist or fingers, which serves to differentiate it from the stimulation of the median nerve.

Inadvertent blockage of the median nerve can be very problematic, since its sensory component is very pronounced, and complaints of dysesthesia and pain may prevail.

In the event of perceiving the rhythmic contraction of the wrist and fingers in front of the electrical stimulation, the physician performing this procedure should drawback the tip of the needle to a more superficial position and point it anteriorly until the elbow flexors are contracted vigorously.

CONCLUSION

Phenol neurolysis may be an alternative for the treatment of spasticity when the use of BoNT-A is limitated by doses or cost. It is an effective, immediate-acting, low-cost, long-term effective treatment and few adverse events occur when simple care with nerve selection is observed.

Ultrasound-guided nerve blocks have been increasingly used,[38] however, in view of the technical and financial limitations of these new methods, the use of surface anatomy to guide the localization of nerve blocks, associated with electrostimulation, still is a useful and effective technique.

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