ATLAS OF ULTRASOUND-GUIDED NERVE-TARGETED PROCEDURES FOR SPASTICITY

Maximizing outcomes for the patient with spasticity through optimal muscle selection

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Abbreviations

In the text the following abbreviations are used:

AFO ankle foot orthosis
AIN anterior interosseous nerve
ASP arm spastic position
BI Barthel Index
BoNT botulinum neurotoxin
CNS central nervous system
CRPS complex regional pain syndrome
DBUN deep branch of ulnar nerve
DIO dorsal interosseus
DNB diagnostic nerve block
ECRB extensor carpi radialis brevis
ECRL extensor carpi radialis longus
ECU extensor carpi ulnaris
EDC extensor diigtorum communis
EDB extensor digitorum brevis
EI echo intensity
EMG electromyography
EPB extensor pollicis brevis
EPL extensor policis longus
FCR flexor carpi radialis
FIM Functional Independence Measure
FPB flexor pollicis brevis
FDP flexor digitorum profundus
FDS flexor digitorium superficialis
FPL flexor pollicis longus
HSN hyperselective neurectomy
HSP hemiplegic shoulder pain
LPN lateral pectoral nerve
MAS Modified Ashworth Scale
MSCN musculocutaneous nerve
MPN medial pectoral nerve
PMM pectoralis major muscle
PSS post-stroke spasticity
ROM range of motion
SCI spinal cord injury
SEMLC single-event multilevel chemoneurolysis
SEVF spastic equinovarus foot
SMD spastic movement disorder
SMO spastic muscle overactivity
TBI traumatic brain injury
TEL transepicondylar line
TIP thumb-in-palm
UMN upper motor neuron
UMNS upper motor neuron syndrome
US ultrasound

1.3 How to use this altas

Paul Winston

The **Atlas of Botulinum Toxin Injection**, by Professor Wolfgang Jost, was first published in 2008 and offered a detailed anatomical guide to targeting botulinum neurotoxin (BoNT) injections for spastic muscles. Further editions have included key clinical pearls and more recently, ultrasound (US) images of muscles. The Atlas has become a clinical staple. Using the familiar images and principles was a natural option for this new Atlas. The Atlas remains the standard for localization for BoNT injections.

Spasticity requires multimodal intervention. This includes pharmacologic agents from oral to BoNT and phenol/alcohol. However, surgical interventions with neurotomy/neurectomy, tendon lengthening, casting bracing, and newer innovations such as percutaneous cryoneurolysis are gaining attention. To maximize outcomes, it is necessary to understand the different manifestations of spastic muscle overactivity. Spastic postures may be attributed to a fully reducible deformity. This means that the joint can be ranged through the full range passively or even actively. Typically, the slow passive phase of movement will allow for this, while a fast movement will result in a catch followed by reduced ease of movement and a possible hard end point. A musculo-tendinous retraction is the other end of the spectrum, wherein the muscle cannot be extended any further to achieve full range of motion. While we attribute the term contracture to the second state, physical examination alone will not establish whether a joint is fully reducible. Spastic muscle overactivity may simply prevent the muscle from going to its full length. Patients may note their hand opens in the morning or at night but cannot be forced open in the day. This Atlas offers the techniques to perform anesthetic motor diagnostic nerve blocks (DNBs). The DNB is used to block muscle activity, thus overriding spastic muscle activity and allowing for the differentiation between true reducibility and contracture. Spastic dystonia is an additional pattern that must be explored when treating spasticity. The most common spastic positions of the limbs are described by Dr Jörg Wissel in this introduction section. Prof Anand D. Pandyan and Prof Jens Bo Nielsen review the challenges of diagnosing spasticity and the inadequacy of most of our measures of assessment and outcomes.

Most of the literature on the DNB is from French-speaking countries and relied on surface anatomical localization and percutaneous e-stimulation (e-stim). This Atlas provides the framework to perform targeted US-guided DNB in an efficient manner. The Atlas provides the surface location and landmarks and images of the nerves and their branches. The Atlas will demonstrate the close proximity of nerves with vascular structures that provide rapid identification of the targeted nerves. Color Doppler US can augment the experience and help avoid vascular puncture. It is important to recognize that traditional line drawings do not reflect the spatial relationship of the fascicular arrangement of nerves, nor demonstrate the proximity of nerves branches, nor the wide variation of nerve branches in vivo. We therefore provide three-dimensional (3D) nerve reconstructions by Dr Axel Schramm and surgical images by Dr Caroline Leclerq and Dr Mark A. Mahan.

The DNB can be seen as the new cornerstone for spasticity management. For example, performing a DNB to the brachialis branch demonstrates that only this muscle is required to target for BoNT therapy since the patient can achieve full active range of motion with this muscle paralyzed. The management of spasticity elbow is covered in the Atlas by Dr François Genêt and Dr Paul Winston. Similarly, a DNB to the tibial branch to the gastrocnemius or soleus can show which muscles are implicated. This is provided in detail by Dr Thierry Deltombe with the US localization by Dr Alessandro Picelli. Therefore, the DNB can maximize the use of BoNT or phenol through accurate targeting of the key muscles involved in spasticity. The DNB will also suggest if a more definitive procedure such as neurotomy/neurectomy of cryoneurolysis could be offered for longer duration and more significant blockade. Dr Caroline Leclercq, renowned orthopedic surgeon will cover the role of nerve-targeted surgery. Dr Heakyung Kim and Dr Michael Munin will demonstrate the role of phenol in spasticity and Dr Sheng Li will demonstrate the targeting of the nerves to the wrist and fingers with phenol. Many experts from multiple countries have contributed to the content, a culmination of many decades of experience. Challenges in US technique and muscle fibrosis will be explored by Dr Rajiv Reebye.

Whether a seasoned expert in nerve-targeted procedures or a novice, this Atlas will offer the guidance, demonstration, clinical scenarios, treatment options and accompanying videos to improve outcomes. We hope it will highlight that complex patient presentations require a multimodal intervention and collaboration between experts. This Atlas is an initiative of members that represent both CANOSC and Symposium International en Neuro-Orthopédie à Versailles (SNOV) to offer advanced accessible education for spasticity interventions on a global scale.

CANOSC
Canadian Advances in Neuro-Orthopedics for Spasticity Congress

SNOV - 2021
Neuro-Orthopedics
Versailles

The Atlas was conceived through a collaboration of CANOSC and SNOV physicians.

1.4 Changing the approach to the clinical measurement of spasticity

Anand D. Pandyan & Jens Bo Nielsen

Introduction

Spasticity is one of those ubiquitous terms in neurologic rehabilitation that permeates both practice and research.[1–3] A significant proportion of the literature (30%) does not provide a definition when the term is used. When a definition is provided, there is a lack of consistency in these definitions.[3,4] The challenges of definition have been previously discussed and will not be revisited;[3–5] however, instead of a definition let us present a first principle argument that may help with establishing a framework for measurement and, therefore, for treatment.

What is spasticity?

Before we attempt to explore the term spasticity it may be worth our while to refresh our memory on the evolution of normal motor control. We are born with very little voluntary control or purposeful movement. This is gained over time and the acquisition of this control is underpinned by a combination of **excitation** of essential muscles and the **inhibition** of non essential muscles. The normal tendency is for an individual to **turn on** more muscles than that normally needed when a movement is being learnt for the first time and then as competency is gained the individual learns to **turn off** the muscles that are not required for efficient movement (e.g. learning to dance or a martial art). The process of both **excitation** and **inhibition** are active processes within the central nervous system (CNS) and motor control evolves with the level of practice, the individual's capacity (e.g. strength, fitness), and the environment.

For the purposes of this chapter, we will focus on injuries to the (upper) motor neuron pathways within the CNS. The CNS is surprisingly resilient and the signs and symptoms after an injury are a reflection of not just the site and size of the injury but also the ability of the CNS to compensate for the primary injury. After an injury, a person loses both the ability for **excitation** and **inhibition**. The **loss of excitation**, if severe, presents as paralysis and, in milder forms, as a combination of weakness and a loss of dexterity. The **loss of inhibition** primarily presents as an involuntary activation of the alpha-motor neuron **(spasticity)** to an external (e.g. cold) or internal (e.g. pain) stimulus. If one had to be technical and accurate, the best way to describe the initial presentation of an injury to the CNS is **spastic-paralysis.**[5] Following this theoretical argument, one can infer that **most adult patients who have an injury to the CNS are likely to show signs of spasticity (i.e. muscles being active when they should normally be silent).**

Clinical manifestations of spasticity and contractures and their time course of development

Since a majority of the literature studying the clinical manifestation of spasticity and its time course has drawn on unvalidated, unreliable, and confounded clinical scales for the measurement of spasticity, there is not much we can learn from this.[4,5] Based on the studies that have used direct measures, one can summarize the clinical presentations as shown below (see reference 5 for a full description):

- Muscle activity at rest (spastic dystonia).
- Muscle activity when a resting muscle is stretched — this activity can present as velocity-dependent[1] and/or position-dependent[2] (stretch-induced muscle activity).[3]
- This can also present as a clasp knife presentation, that is, a buildup of resistance that stops the movement; however, if one were to continue with the movement a sudden release is felt.
- Spasms and/or clonus.
- Secondary changes in muscles, tendons, joints, and connective tissue leading to increased passive stiffness and eventually contractures. **(It can be argued whether these should be separated from spasticity, possibly in their own right as spastic myotonia or as here included under the spasticity heading as a reflection of the difficulty in separating these changes clinically from other signs and symptoms of spasticity.)**

It is difficult to comment on whether the abnormal movement patterns and co-contractions, during an active, purposeful, or effortful movement result from spasticity because this could be a normal physiologic response in people with weakness.

There is a profound difference in how frequently spasticity is reported in studies using direct measures rather than clinical eval-

1 As the passive stretching velocity is increased, the angle at which muscle activity occurs earlier in the range of motion and/or the magnitude of the muscle activity increase.
2 Muscle activity increases proportionally as the muscle is passively lengthened/stretched.
3 This phenomenon is consistent with the Lance definition of spasticity.[5]

suffering from arm spasticity after stroke or traumatic brain injury.[2] The data show that spastic postures of the arm are associated with reduced functional independence. The association of spastic pattern and FIM was strongest for arm spastic position (ASP) I of Hefter's classification (see below).[1,2]

In the context of functional independence, a differentiation between 'spasticity' and 'disabling spasticity' is of relevant importance. Disabling spasticity or spastic movement disorder (SMD) needs medical treatment, additionally to physical treatment to overcome functional limitations.[3] To improve the management and prognosis of SMD, especially in case of disabling spasticity, several studies focused on focal medical interventions additionally to physical treatment. Those interventions include intramuscular BoNT A injections, phenol injections in spastic muscles, or neurotomies of relevant peripheral nerves. Appropriate intervention, possibly in combination, should be chosen individually according to the different spastic patterns. Therefore, in clinical routine, it is important to start with a detailed analysis of the individual spastic positions of proximal and distal limbs in different body positions (supine, sitting, walking). It should be followed by the pattern classification of the spastic body parts. An accurate and precise muscle selection is then both the basis and essential requirement for an effective focal medical therapeutic intervention to adequately manage of the SMD. An exact and detailed analysis and classification of spastic patterns of the upper and the lower limbs improves and facilitates the goal-setting process and therapeutic schedule of rehabilitation to attain the required goals.

Therefore, we introduce both published information and our own view on major typical positions of the spastic upper and lower limbs in SMD.

Spastic patterns of the upper limb

Arm spasticity often disables the function of the upper limb and impairs the patient's functional independence. In most cases all or a combination of elbow, wrist, and shoulder are involved in SMD in patients suffering from post-stroke spasticity (PSS).[4–7] In the chronic phase after a stroke, a pronounced disabling spasticity affects both gross motor function and fine-tuned motor skills (Table 2-1).[4–7] Spasticity of the shoulder girdle restricts the degree of movement of the upper limb joints (passive range of motion), impairs active movements and upper-limb tasks, measured by validated motor tests.[8–10]

In the following sections we describe diverse spastic movement patterns of the upper limb according to Hefter's classification system.[11]

Hefter's classification (ASP I–V)

Hefter et al.[1] analyzed the postures and spastic movement patterns of the upper limb in 665 patients with post-stroke spasticity. In 94% of the cohort, 5 typical patterns of arm positions during walking, standing, or sitting were found (Fig 2.3).

Shoulder muscles	Elevation	Depression	Protraction	Flexion	Extension	Internal rotation	External rotation
Supraspinatus							(+)
Deltoid				(+)	(+)	(+)	(+)
Pectoralis major		+		+		+	
Subscapularis						+	
Latissimus dorsi		+			+	+	
Teres major					+	+	
Teres minor							+
Infraspinatus							+
Trapezius	+						
Serratus anterior		(+)	+				
Pectoralis minor			+				

Table 2-1 Contribution of shoulder girdle muscles to shoulder positions.

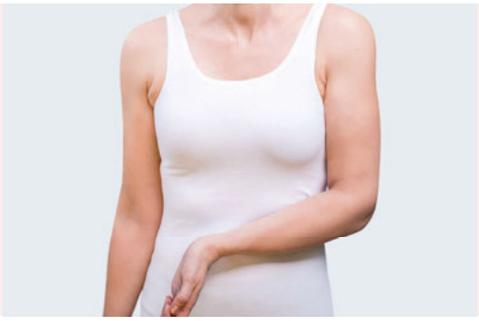
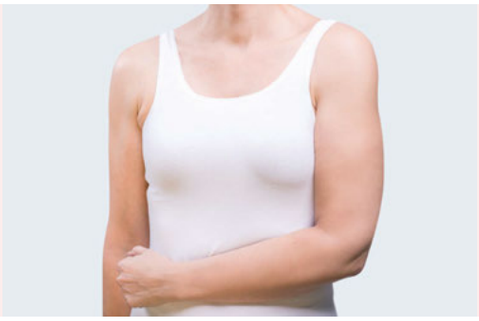
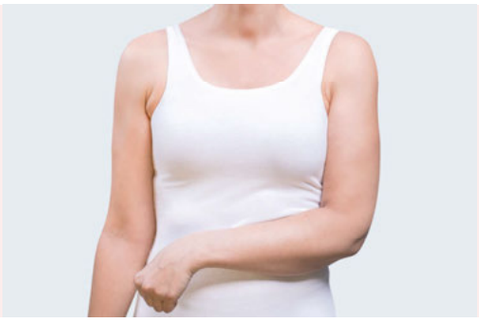
	Shoulder	Elbow	Forearm	Wrist
	Internal rotation/ adduction	Flexion	Supination	Flexion
	Internal rotation/ adduction	Flexion	Supination	Extension
	Internal rotation/ adduction	Flexion	Neutral	Neutral
	Internal rotation/ adduction	Flexion	Pronation	Flexion
	Internal rotation/ retroversion	Flexion	Pronation	Flexion

Fig 2.3 The classification of arm spastic patterns (Hefter et al.)[1]. Upper-limb spasticity pattern. Note: All five upper-limb patterns could be combined with any spastic hand and finger position (e.g. claw, spastic, flexed, intrinsic, lumbrical).

ASP I–V:

ASP I: internal rotation/adduction of shoulder, elbow flexion, forearm supination, wrist flexion (24.8%[1], 3.4[12], 34%[13]).

ASP II: internal rotation/adduction of shoulder, elbow flexion, forearm supination, wrist extension (5.3%[1], 9.4%[12], 3%[13]).

ASP III: internal rotation/adduction of shoulder, elbow flexion with neutral positioning of forearm and wrist (41.8%[1], 52.3%[12], 36%[13]).

ASP IV: internal rotation/adduction of shoulder, elbow flexion, forearm pronation, wrist flexion (18.9%[1], 19.5%[12], 23%[13]).

ASP V: internal rotation/retroversion of shoulder, elbow extension, forearm pronation, wrist flexion (3.6%[1], 3.4%[12], 3%[13]).

Clinical studies focusing on the frequency of each pattern showed that ASP III was by far the predominant pattern (52.3%), followed by ASP I and IV.[1,12] Another international multicenter study, which included 409 patient suffering from PSS, found that even 99% of patients showed 1 out of 5 Hefter's spastic arm patterns (pattern III 36%, I 34%, and IV 23%).[13]

In principle, the treatment of spasticity by injection of BoNT is recommended to follow the classification system; however, the last decision on dosing and which muscles to target is made by the physician. The mean doses of BoNT A (Dysport) according to ASP I–V were 707 IU for ASP I, 575 IU for ASP II, 711 IU for ASP III, 799 IU for ASP IV, and 747 IU for V. The total mean dose was 728 IU (range 100–2300 IU).[13]

In this study, 84% of patients achieved their individual goals; 91.3% were rated as improved by the physician's assessment. Of note, BoNT injections were performed mostly by the use of technical guidance techniques such as electromyography, e-stimulation, or US, well known as being more effective in targeting the muscles chosen for treatment than manual palpation only.[13]

In Table 2-2, we show an assignment of muscles recommended for injection for each category of the ASP I–V classification based on consideration of functional anatomy and recent publications.

Spastic positions of the distal upper limb are not part of Hefter's classification system. That includes spastic positions of all hand, thumb, and fingers due to overactivity of extrinsic and intrinsic flexors, resulting in malpositions such as 'claw', 'clenched fist', 'intrinsic lumbrical hand', and 'thumb in palm'. Neither their occurrence nor the detailed pattern of the distal spasticity is systematically related to shoulder/arm spastic patterns. However, for therapeutic purposes, the distal patterns are easy to combine with the proximal pattern provided by Hefter et al.[1]

Spastic patterns of hand, thumb, and fingers

Spastic positions of hand, thumb, and fingers are one of the major topics in hand rehabilitation after stroke and TBI. They are related to progressive shrinkage of fasciae and tendons and thereby to a disturbed function of fine-tuned motor skills. There are at least six classification systems for spastic postures of hand, fingers, and thumb, assessed in two conditions at rest or in function:

Zancolli et al. classification of wrist and finger positions (Zancolli et al.)[14]

Level 0: No flexion spasticity
Level 1: Minimal flexion spasticity
Level 2a: Moderate flexion spasticity with the ability to extend the wrist
Level 2b: Moderate flexion spasticity with the inability to extend the wrist
Level 3: Severe flexion spasticity

House thumb-in-palm (TIP) classification (House et al. 1981)[15]

There are four types for spastic thumb positions, assessed while grasping large or small objects (Fig 2.4B).

Type I: Metacarpal adduction
Type Ia: Neutral interphalangeal joint
Type Ib: Interphalangeal hyperextension
Type Ic: Interphalangeal hyperflexion
Type II: Metacarpal adduction + metacarpophalangeal flexion
Type IIa: Neutral interphalangeal joint
Type IIb: Interphalangeal hyperextension
Type III: Metacarpal adduction + metacarpophalangeal hyperextension
Type IV: Metacarpal adduction + metacarpophalangeal flexion + interphalangeal flexion.

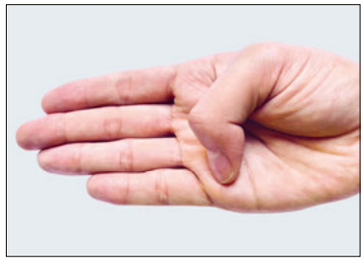


Fig 2.4A Corry classification.[16]

The **Corry classification**[16] for thumb extension (five levels, Fig 2.4A and thumb abduction (three levels) and the **Sakellarides thumb classification** (four levels)[17] provides added value for the clinician compared to the House TIP classification. The **Forearm deformity classification** (four levels, Gschwind & Tonkin[18]) has been rarely used in stroke patients.

	First recommendation	Additional recommendation
1. Upper limb		
Shoulder and arm (ASP I–V)[1]		
ASP I	PM, biceps, FCR, FCU	Br, Pm, BR, TM, Sup
ASP II	PM, biceps	Br, Pm, BR, TM
ASP III	PM, biceps, Br, BR	Pm, PT
ASP IV	PM, biceps, BR, PT, FCR, FCU	Br, Pm, TM
ASP V	Triceps, LD, TM, PT, FCR, FCU	Deltoideus, SS, IS
Hand and finger positioning		
Wrist flexion (Zancolli[14] level >1)	FCR, FCU	PL
Claw	FCR, FCU, FDP, FDS	
Clenched fist	FDP, FDS	FPL
Intrinsic lumbrical	Lumbricalis	
HTIP[13] FPL, FPB, AP		
Ia	AP	
Ib	AP	EPL
Ic	AP, FPL	
IIa	AP, FPB	
IIb	AP, FPB	EPL
III	AP, EPB	
IV	AP, FPL	FPB
2. Lower limb		
Hip flexion	Iliacus and/or psoas major	Pectineus (external roation/flexion)
Hip adduction/internal rotation	AM, AL, sartorius, gracilis	AB, ST/SM
Knee flexion	BF	SM/ST
Knee extension (stiff knee)/overextension	Quadriceps (mostly RF), GCM/GCL	
Equinus (drop foot)	Soleus	GCM/GCL
Equinovarus	TP, soleus	GCM/GCL
Hyperextension of big toe	EHL	
Claw toes	FDL	FDB

Table 2-2 ASP; arm spastic position; PM, pectoralis major; Br, brachialis; Pm, pectoralis minor; BR, brachioradialis; TM, teres major; Sup, supinator; PT, pronator tere; FCR, flexor carpi radialis; FCU, flexor carpi ulnaris; LD, latissimus dorsi; SS, supraspinatus; IS, infraspinatus; FDS, Flexor digitorum superficialis; FDP, flexor digitorum profundus; PL, palmaris longus; FPL, flexor pollicis longus; FPB, flexor pollicis brevis; AP, adductor pollicis; HTIP, House TIP classification; AM, adductor magnus; AL, adductor longus; AB, adductor brevis; ST, semimebranosus; SM, semitendionsus; BF, biceps femoris; RF, rectus femoris; TP, tibialis posterior; EPL, extensor pollicis longus; EPB, extensor pollicis brevis; EHL, extensor hallucis longus; FDL, flexor digitorumlongus; FDB, flexor digitorum brevis

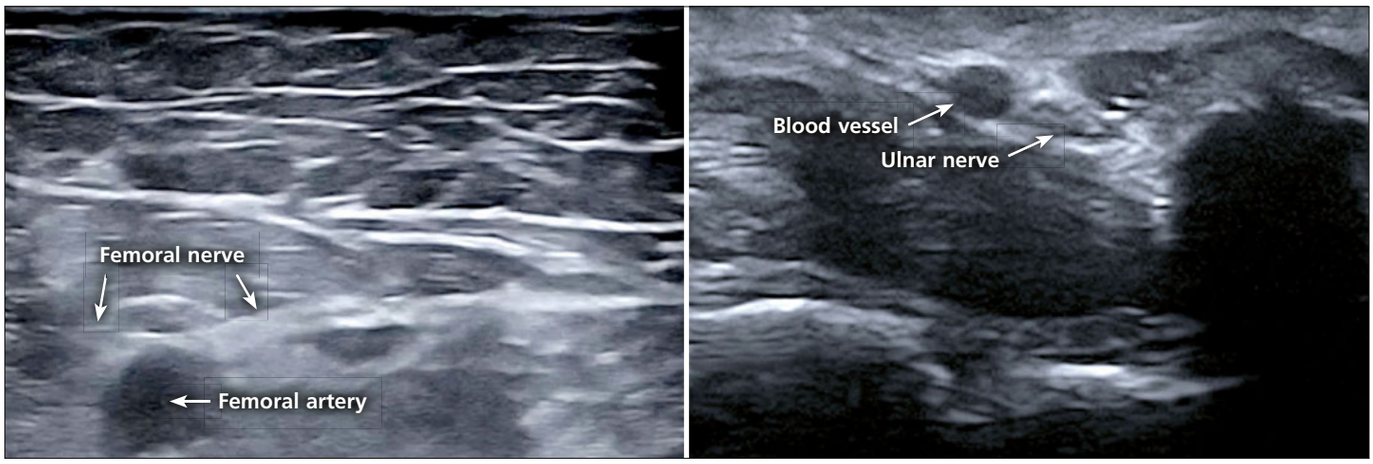


Fig 3.10 The ulnar nerve in the palm is seen adjacent to the structure in black.

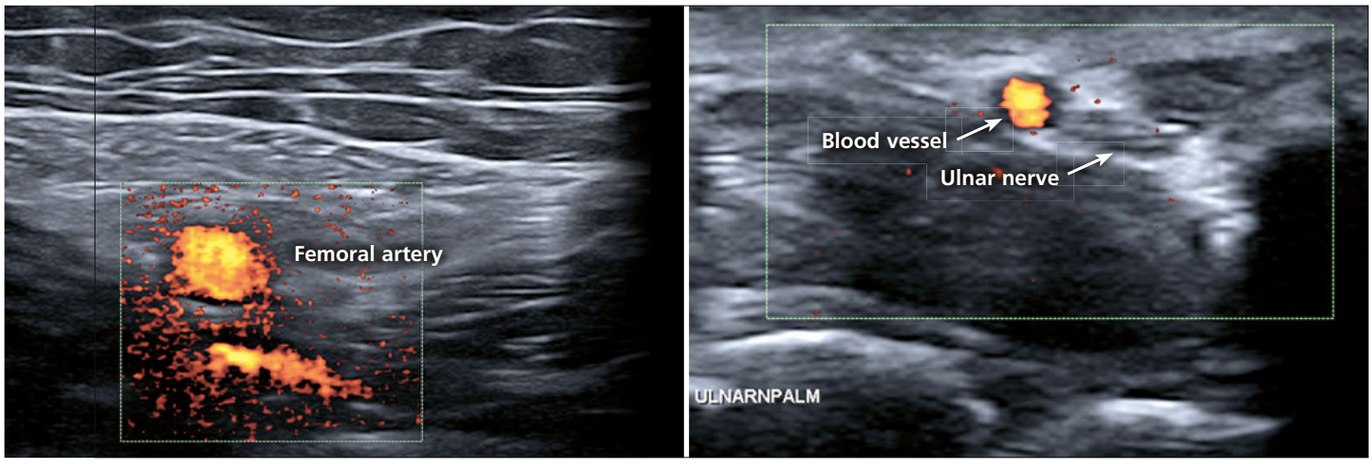


Fig 3.11 The color Doppler allows for faster identification and assists with avoiding vascular puncture.

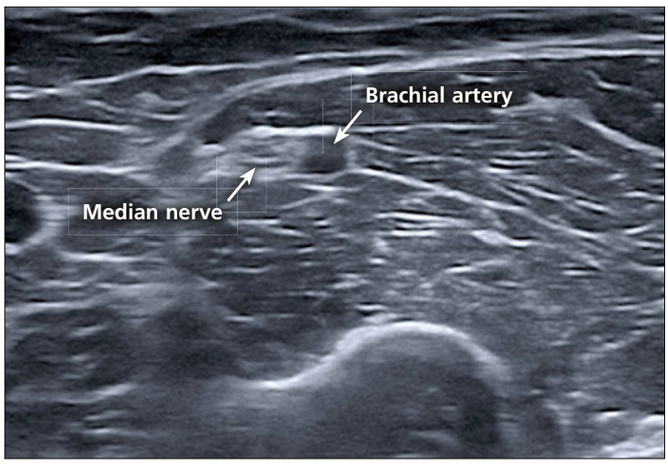


Fig 3.12 Median nerve above the elbow that includes the pronator teres.

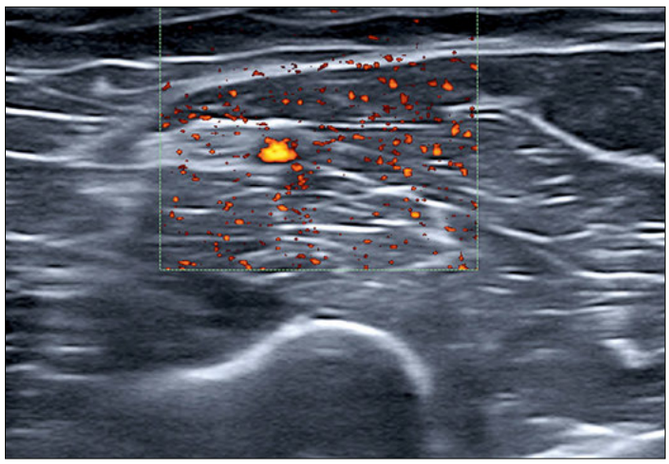


Fig 3.13 The color Doppler image shows the close relationship of the nerve trunk and artery.

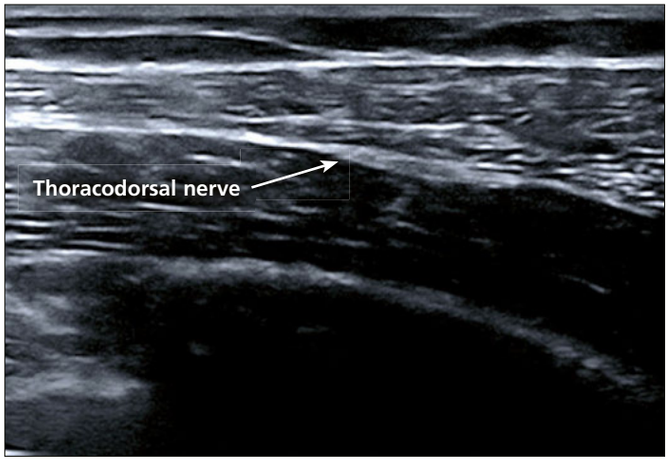


Fig 3.14 Thoracodorsal nerve to the latissimus dorsi. A highly consistent nerve that may be confused with the fascial planes.

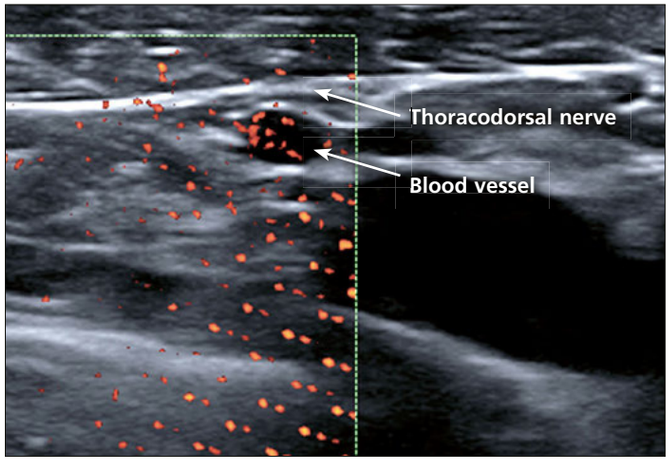


Fig 3.15 The color Doppler image alerts to the risk of puncture and confirms the position of the nerve.

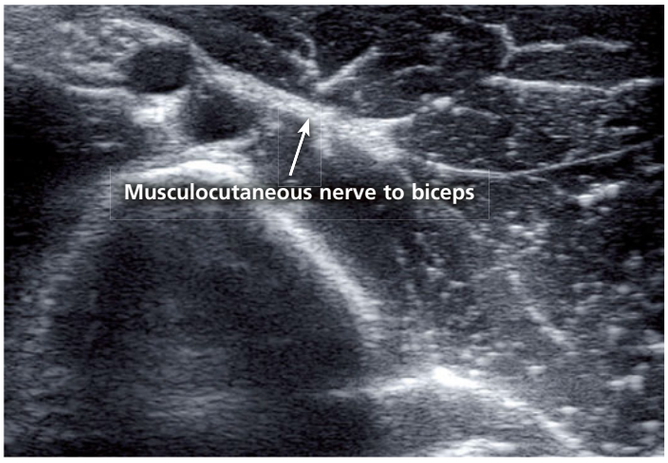


Fig 3.16 Musculocutaneous nerve to the biceps.

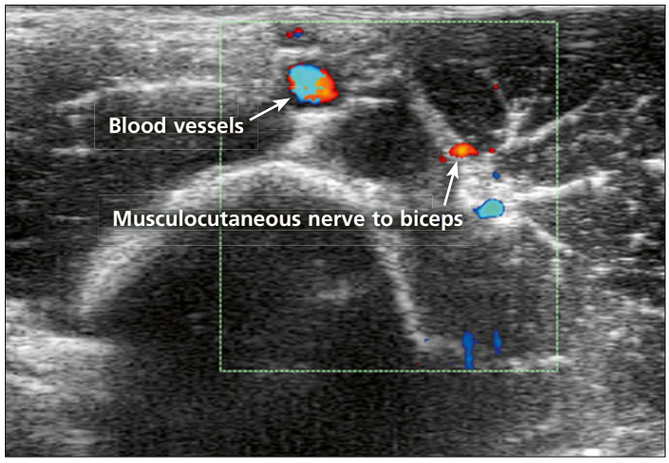


Fig 3.17 Color Doppler images show the appearance of the vessels wrapped with nerve.

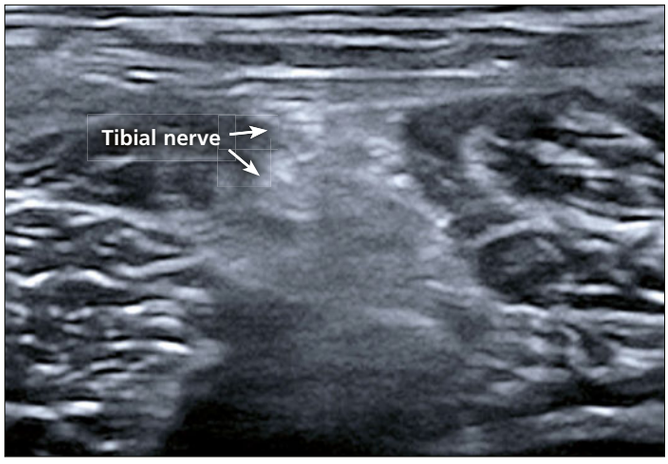


Fig 3.18 Tibial nerve trunk at the popliteal fossa.

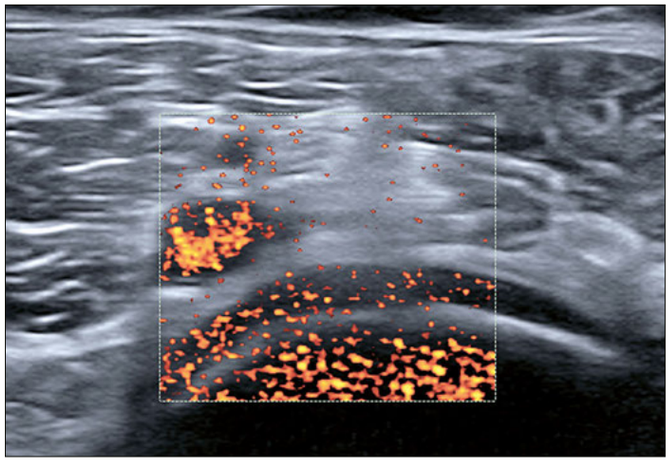
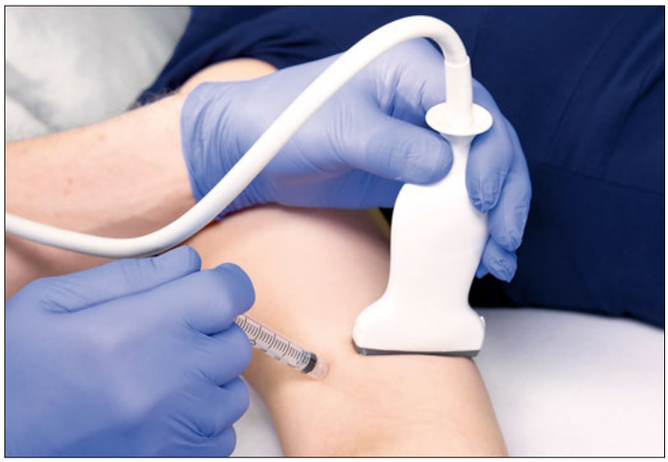


Fig 3.19 The color Doppler may reveal the nerve adjacent or above the tibial artery.

3.8 Tracking a nerve proximal to distal: median nerve from pronator teres to the intrinsic muscles

Before any DNB or procedure, it is important to familiarize oneself with the relationship of the nerve and vascular structures. This is made possible by tracking the US probe and down to nerve. There is high variability in nerve branches and innervation and the appearance of muscle architecture. Nerve stimulation to the target muscle(s) is key to ensure accuracy of the location.

Median nerve tracking



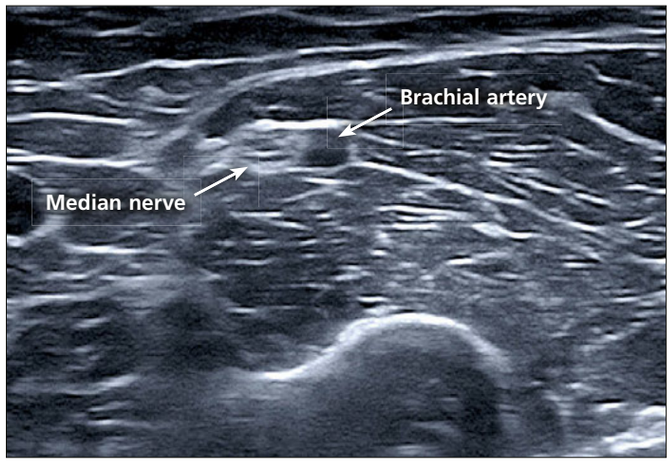
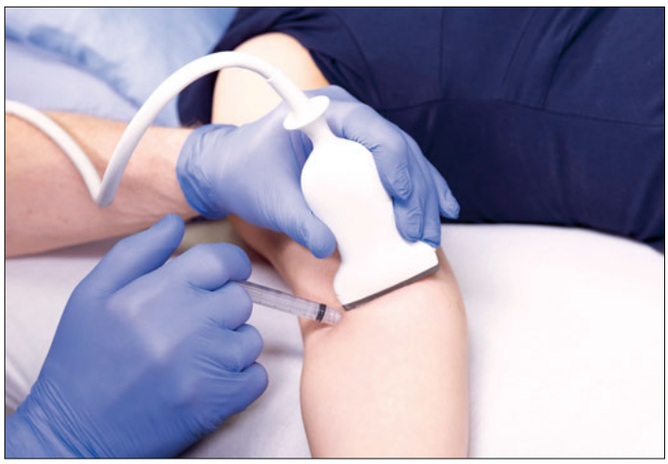


Fig 3.36 Above elbow pronator teres and distal muscles.



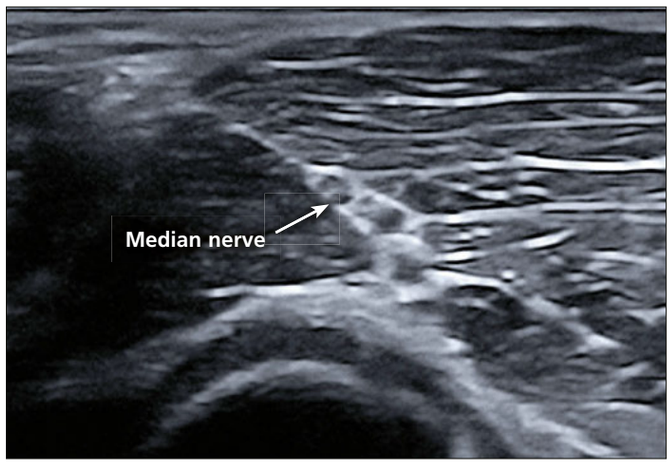
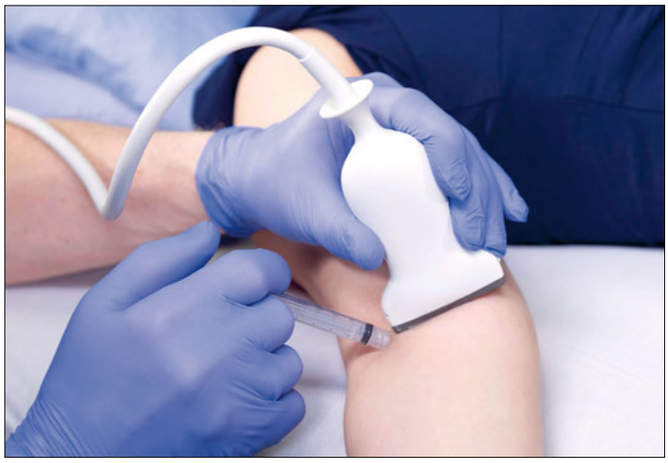


Fig 3.37 Below elbow level for the flexor carpi radialis.



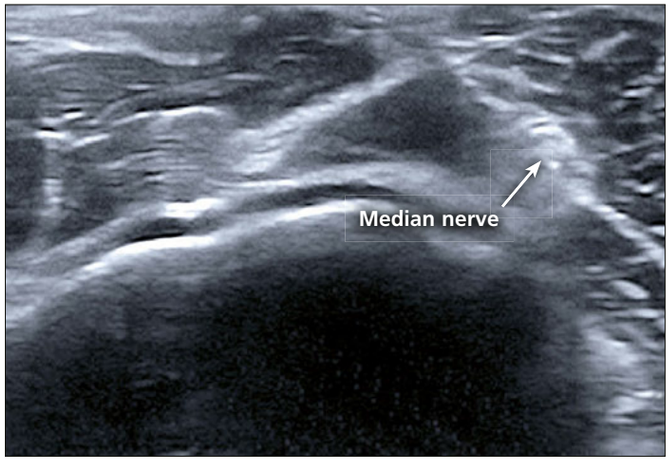
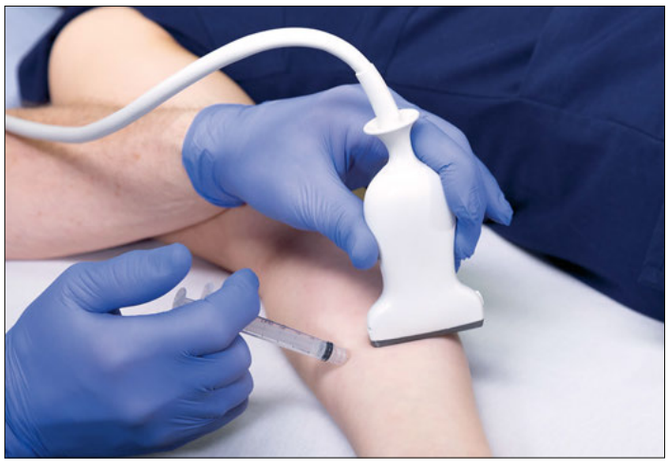


Fig 3.38 Below elbow to FDS. It is possible to stimulate each fascicle.



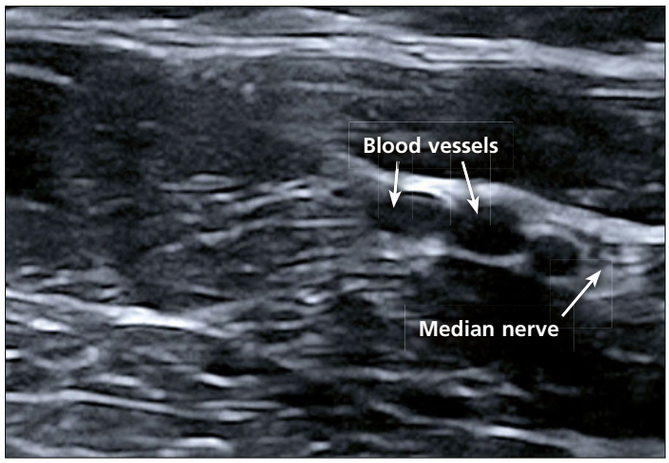
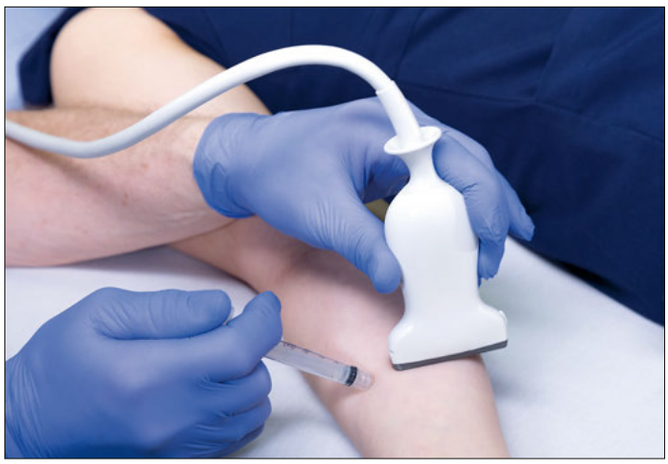


Fig 3.39 Below elbow at level for flexor digitorum profundus. Note this is a branch of the anterior interosseous nerve. Must stimulate to find the branch.



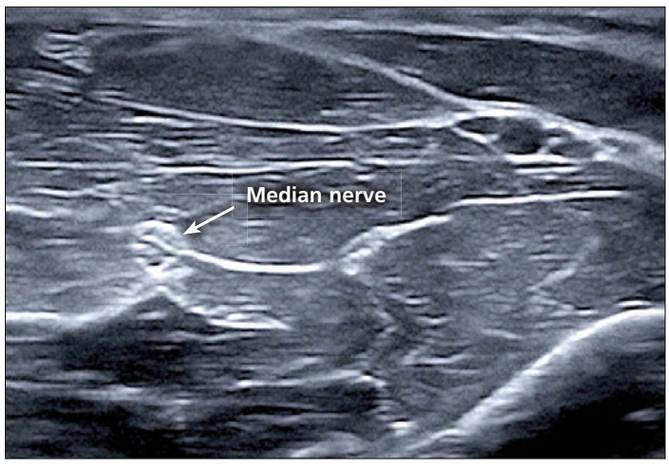


Fig 3.40 The median nerve at the level of flexor pollicis longus. Must stimulate to differentiate the anterior interosseous nerve from the median nerve. A more nerve proximal stimulation may be necessary.

4.2 Cryoneurolysis – a new percutaneous minimally invasive therapy for the treatment of the spastic limb

Daniel Vincent, Eve Boissonnault & Paul Winston

The principles of diagnostic nerve blocks (DNBs) can be utilized to determine if longer-lasting minimally invasive percutaneous neurolysis if feasible. For example, phenol and alcohol are used for partial or segmental neurolysis to selected muscles. It is possible to perform neurolysis without chemical agents, as demonstrated in the practice of pain medicine using radiofrequency ablation or with cryoneurolysis. Lloyd first used liquid nitrogen in 1961 to cool a probe and coined the term cryoanalgesia in 1976 when they noted the absence of neuritis and/or neuralgia with their procedures.[1] Cryoneurolysis has decades of established efficacy in the analgesic literature. Compared to the extensive literature on surgical neurolysis, there are historically few mentions of percutaneous ablative neurolysis for spasticity. There was one published case report of using percutaneous radiofrequency ablation on a motor nerve for spasticity[2] and one for cryoneurolysis before 2019.[3] An additional published abstract documented improvements in elbow spasticity using cryoneurolysis to the musculocutaneous nerve (MSCN).[4]

Diagnostic nerve blocks are first performed to isolate the motor nerve(s) responsible for the observed spasticity; this can then be followed by cryoneurolysis, which is performed using a small 1.2–1.3-mm diameter cryoprobe that is inserted percutaneously to targeted peripheral nerves. It has an established history over more than 50 years for lasting pain relief from months to years, when used for sensory nerves and neuromas.[5,6] Cryoneurolysis is possible due to the process of throttling a gas through an orifice from high to low pressure resulting in a rapid expansion of the gas and a drop in temperature, known as the Joule–Thomson effect.[1] The rapid cooling generates an ice ball or oval between 3.5 mm and 18 mm that is formed at the tip of the cryoprobe using a closed-circuit system with compressed CO_2 or N_2O (Fig 4.1). The temperature is determined by the boiling point of the gas, typically between –60° C and –88° C (the boiling point of N_2O[1]). The ball or oval, creates a targeted zone of axon and myelin disruption. The lesion is classified as a Sunderland Nerve Classification injury second degree. This is an axonotmesis of the nerve, with resulting loss of axon continuity due to Wallerian degeneration of the targeted nerve extending outward from the lesion over a limited distance. However, the basal lamina, epineurium, and perineurium of the targeted nerve remain intact and serve as a conduit or tube for neural regeneration. Unlike alcohol or phenol chemodenervation, there is little risk of damage to surrounding structures and resulting perineural and muscular fibrosis, which makes it difficult for subsequent ultrasound (US) visualization. With cryoneurolysis, there is no wide dispersion of fluid and minimal release of neurotropin, which is implicated in the formation of painful neuromas.[7]

Cryoneurolysis of the upper extremity

Cryoneurolysis of the whole tibial nerve

We believe that the cryoneurolysis delivered via a small cryoprobe (e.g. 20 gauge), can be safely used in a new manner to perform selective thermal lesions of peripheral motor nerve branches for the management of limb spasticity.[8,9] We recommend the use of US guidance (Fig 4.2) combined with electrostimulation (e-stim) to selectively identify the appropriate nerve(s). For e-stim, the threshold for muscle twitches should be less than 0.8 mA. The use of cryoneurolysis produces an immediate effect on sensory and motor nerves mimicking that of the diagnostic anesthetic nerve block such that the target muscle can be easily identified. This results from the time- and temperature-dependent thermal disruption of neural conduction. Since the physiologic extent of neurolysis is dependent on temperature and time for each lesion generated, the process can be immediately aborted should any sensory disturbance be noted, reducing the risk of permanent neural damage. This would not be the case with chemoneurolysis (alcohol or phenol), radiofrequency neurolysis, or surgical neurotomies. Pulsed radiofrequency neuromodulation may afford the same result as cryoneurolysis but has not been sufficiently studied to recommend its use.

Concerning spasticity, our clinical experience has documented gradual improvement in muscle tone and range of motion, which is most notable at 1–3 months post-cryoneurolysis. With passive stretching, active movements, therapies, and bracing, further improvements have been documented over periods of several months.[7,8] Rehabilitation of the antagonistic muscles is also recommended. The patient may now experience an increase in unopposed movements. For example, the patient may be able to actively dorsiflex the ankle, extend the wrist and fingers, or abduct the shoulder after the flexors are overcome with cryoneurolysis. Physical therapies, bracing at full length, and e-stim of the antagonists can also be helpful. Further, considerations may be necessary such as adjusting orthoses to improve range of motion are noted. Due to the location of the targeted nerve immediately alongside blood vessels it is notable that a review by Gage found that 'The large blood vessels were remarkably resistant to structural change after freezing and their function as a conduit of blood was not impaired.'[10]

Cryoneurolysis is primarily focused on targeting motor nerves; however, sensory branches and mixed nerves can also be treated as is the case in pain management. This includes the suprascapular nerve for shoulder pain, and the median and ulnar nerve trunks for the painful fisted hand and wrist. The tibial trunk will also have a sensory blockade. Intramuscular cryoneurolysis to small nerve branches are also possible to avoid sensory disruption or spare distal muscle targets. For example, the pronator teres, FCR, FCU, FDS or rectus femoris.

Selecting the patient

Targeted diagnostic nerve blocks serve to isolate problematic spastic muscle groups. Typically, these spastic muscle groups have received various combinations of BoNT, chemoneurolysis (alcohol or phenol), physical therapies, and bracing but have reached a therapeutic plateau. However, cryoneurolysis can be the initial treatment provided the DNB identifies a reducible deformity and not a contracture. Although not all nerves involved in spasticity have been published we suggest that the following motor nerves are most easily targeted:

Shoulder girdle
- Lateral pectoral nerve to the pectoral major muscle[11]
- Medial pectoral nerve is harder to image and deeper but possible
- Thoracodorsal nerve to latissimus dorsi
- Suprascapular nerve for reducing pain, which may increase range of motion

Elbow flexors
- Musculocutaneous to brachialis and biceps brachii
- Radial to brachioradialis

Wrist and fingers

It is imperative to assure no sensory dysesthesia in this group
- Median nerve if no sensory dysesthesia with a DNB
- Ulnar nerve if no sensory dysesthesia with a DNB
- Radial nerve for finger extensors

Hip girdle
- Obturator to anterior and posterior adductor groups

Knee
- Femoral nerve to the rectus femoris
- Sciatic nerve for hamstrings (will likely affect all distal muscles)

Foot and ankle
- Tibial nerve to medial and lateral gastrocnemius, soleus, tibialis posterior, and flexor digitorum longus: it is to ensure no sensory dysesthesia.
- Tibial at the ankle for the foot intrinsic muscles

Standardized outcome measures should be used to capture outcomes of the DNB to ensure improvements. We recommend using the Modified Tardieu Scale, Motor Assessment Scale and video capture to compare before and after. We found video capture to be very helpful in documenting nerve block efficacy and motor control. For functional outcomes, the Goal Attainment Scale, the Disability of the Arm, Shoulder and Hand, Box and Block Test, and grip strength are among the outcome measures that can track outcomes. For the adductors, distance between the knees, is used. For lower-extremity procedures, numerous gait assessments can be used in addition to active and passive movements.

A proposed guidance for cryoneurolysis

It is imperative that a DNB is performed to target and select the desired neve or nerve branch using a small amount of a short-acting local anesthetic such as lidocaine (1–1.5 ml of 2% solution). It may be necessary to confirm with a second DNB. The DNB will simulate the expected clinical benefits. The DNB may predict the potential of adverse outcomes or events such as loss of function or sensory impairment. This is particularly important for the tibial nerve and ability to weight bear and walk, or for hand function. A nerve stimulator with a digital display and easily adjusted mA output is essential in identifying the target motor nerve. It should have either an internal nerve stimulator or an external stimulator attached, which can conduct electricity along the exploring needle or probe.

Our clinical procedure entails:

1. The skin is prepped with sufficient antibacterial precautions, for examplee, use of a sterile or bacteriostatic (2–4% chlorhexidine) US gel.
2. A small dose of local anesthesia for cutaneous and subcutaneous anesthesia is used to reduce pain at the percutaneous entry site; less than 0.5 ml of 1% lidocaine to avoid diffusion and anesthetizing the nerve.
3. Specialized probes that allow for deeper injections without need for insulation and should be used according to the manufacturer's guidance. If not available, then either a thermal insulating catheter (no. 16 or no. 18 angiocath) or similar gauge needle larger than the probe to serve as a guide and offer protection from cutaneous frostbite. The use of the catheter will act as an additional insulator to provide optimal low-dose e-stim and assist in cryoprobe positioning.
4. Color Doppler can be enabled to ensure localization of the targeted nerve in the neurovascular bundle. We recommend an in-plane US approach for cryoneurolysis. This ensures the large needle is visualized as it approaches the targeted nerve, to minimize unnecessary repositioning. The probe is guided until it is juxtaposed to the motor nerve branch, with

the tip of the probe clearly visualized and away from any vein or artery.

5. The stimulator should be engaged before needle or probe insertion to ensure that the muscle is responding at a low amperage of between 0.5 and 0.8 mA. Using US and e-stim, observe for the desired muscle movement and adjust the needle and probe depth to obtain the best muscle stimulation. Avoid excessive or repeated entries and if in doubt reexamine the US imagery to confirm the best point of entry and depth. If there is a painful sensory stimulation along the sensory branch, the tip is radjusted.
6. If the patient experiences a painful sensation along the nerve pathway during cryoneurolysis, the device is immediately turned off, the cryoprobe is allowed to defrost and the probe tip is then readjusted before resuming a subsequent lesion.
7. To ensure that complete neurolysis occurs, we suggest that two lesions are performed along the same nerve branch at least 1 cm apart or from opposite sides of a nerve. Each device will have a different protocol. At a temperature of −60°C to −88°C, we recommend 2–3 lesions per large nerve for a total of 3–3.5 min. It is possible to do multiple nerves; one nerve in one setting, each with 2–3 lesions.
8. In the upper extremity, we find that starting with a suprascapular nerve block can reduce shoulder pain and allow for better positioning for abduction. The lateral pectoral nerve can be done next. This will also allow for shoulder external rotation, which will allow for better access to the MSCN, median and ulnar nerves.
9. After completion of the procedure, the percutaneous entry point is stabilized with pressure and an occlusive dressing such as a Band-Aid. Skin glue can be applied if needed.
10. No specific activity restrictions are necessary.
11. Patients on systemic anticoagulants may proceed with DNB or cryoneurolysis. However, if there is a potential risk of bleeding, the anticoagulant may have to be held till after the procedure is complete. The risk of holding the anticoagulant must be weighed against the benefits of the procedure. For patients with a history of embolic phenomenon, a consultation is prudent with the family physician, internist, or cardiologist. An anticoagulant bridging technique may be used in consultation with the internist or hematologist.

Side effects include rare risk of infection and bleeding and sensory dysesthesia or pain from the sensory branches; Unwanted numbness is reduced by ensuring that the DNB does not elicit this. Complex regional pain syndrome type II or neuritis is possible but is self-limiting and treated using conservative measures. Venipuncture risk is mitigated with US guidance. Frostbite is possible. The use of an angiocath is used to protect the skin.

Cryoneurolysis of the MSCN to the brachialis

Cryoneurolysis of the median nerve above the elbow

Cryoneurolysis of the anterior division of the obturator nerve

Cryoneurolysis of the anterior division obturator

Cryoneurolysis of the lateral pectoral nerve

Demonstration of hyperselective intramuscular cryoneurolysis

Cryoneurolysis with the IOVERA system (Pacira)

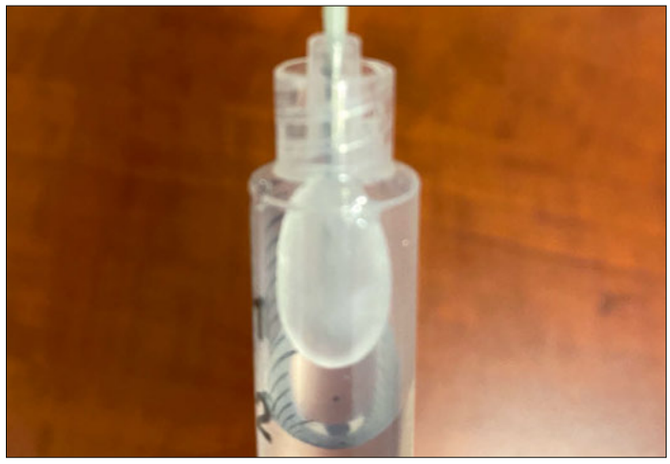


Fig 4.1 Ice ball on IOVERA device (0.6 mm × 10 mm).

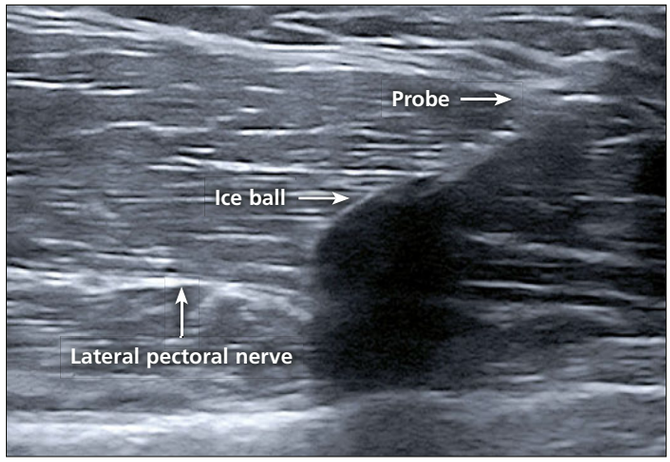


Fig 4.2 Ice ball formation as it grows alongside the nerve.

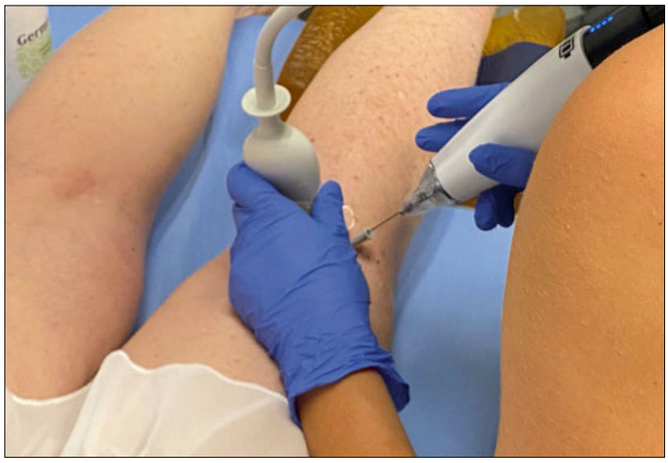


Fig 4.3 Cryoneurolysis of the tibial nerve through no. 16 angiocath.

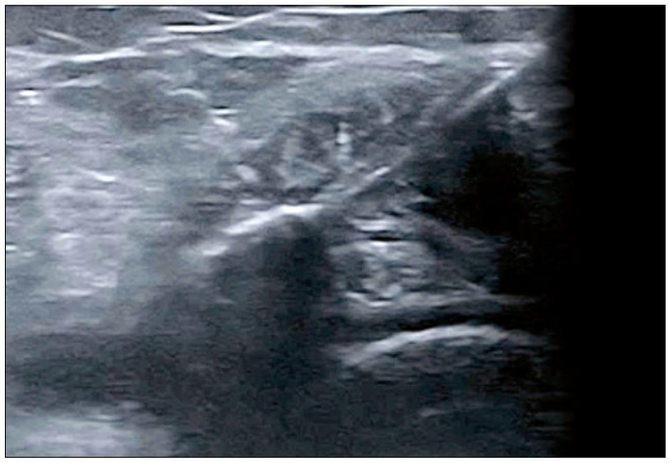


Fig 4.4 Tibial nerve ice ball seen touching the nerve trunk.

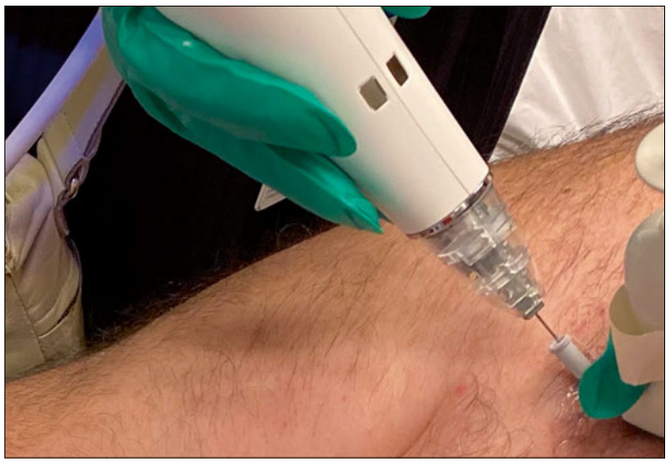


Fig 4.5 Cryoneurolysis of the lateral pectoral nerve through a no. 16 angiocath.

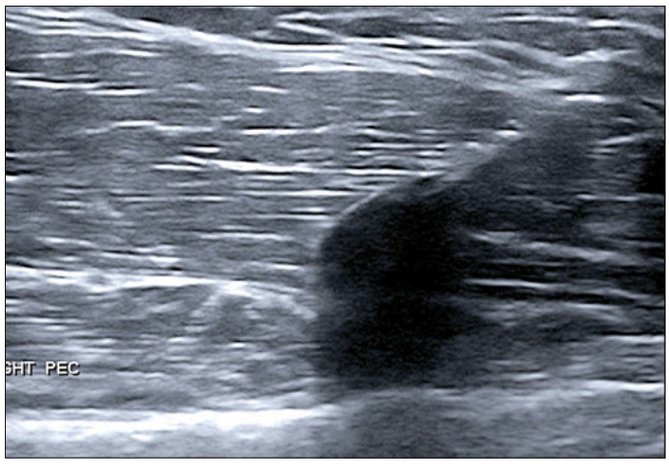


Fig 4.6 Lateral pectoral nerve ice ball.

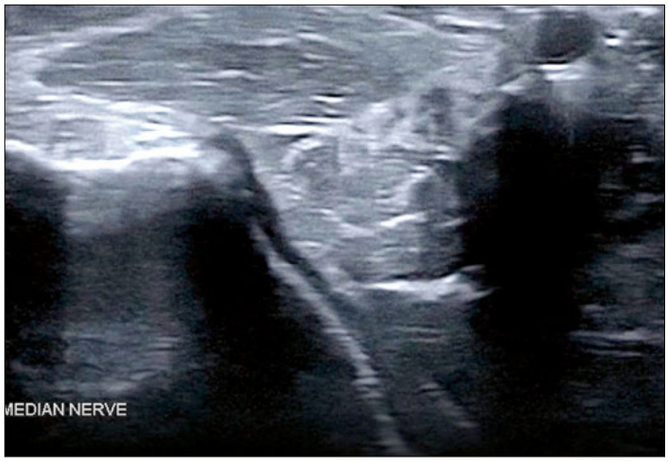


Fig 4.7 Median nerve ice ball. Extensive shadow under the vascular structures.

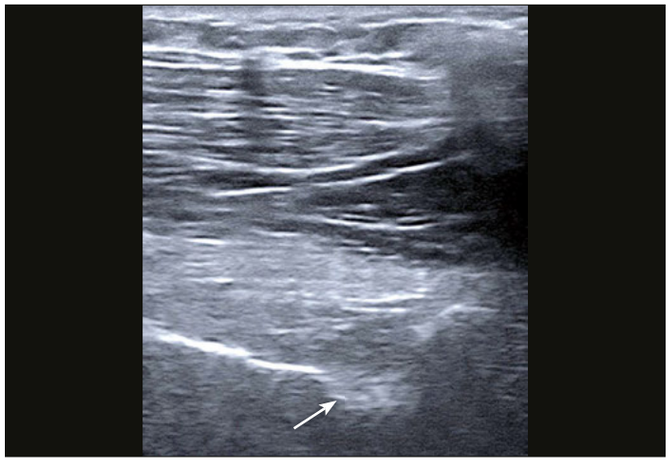
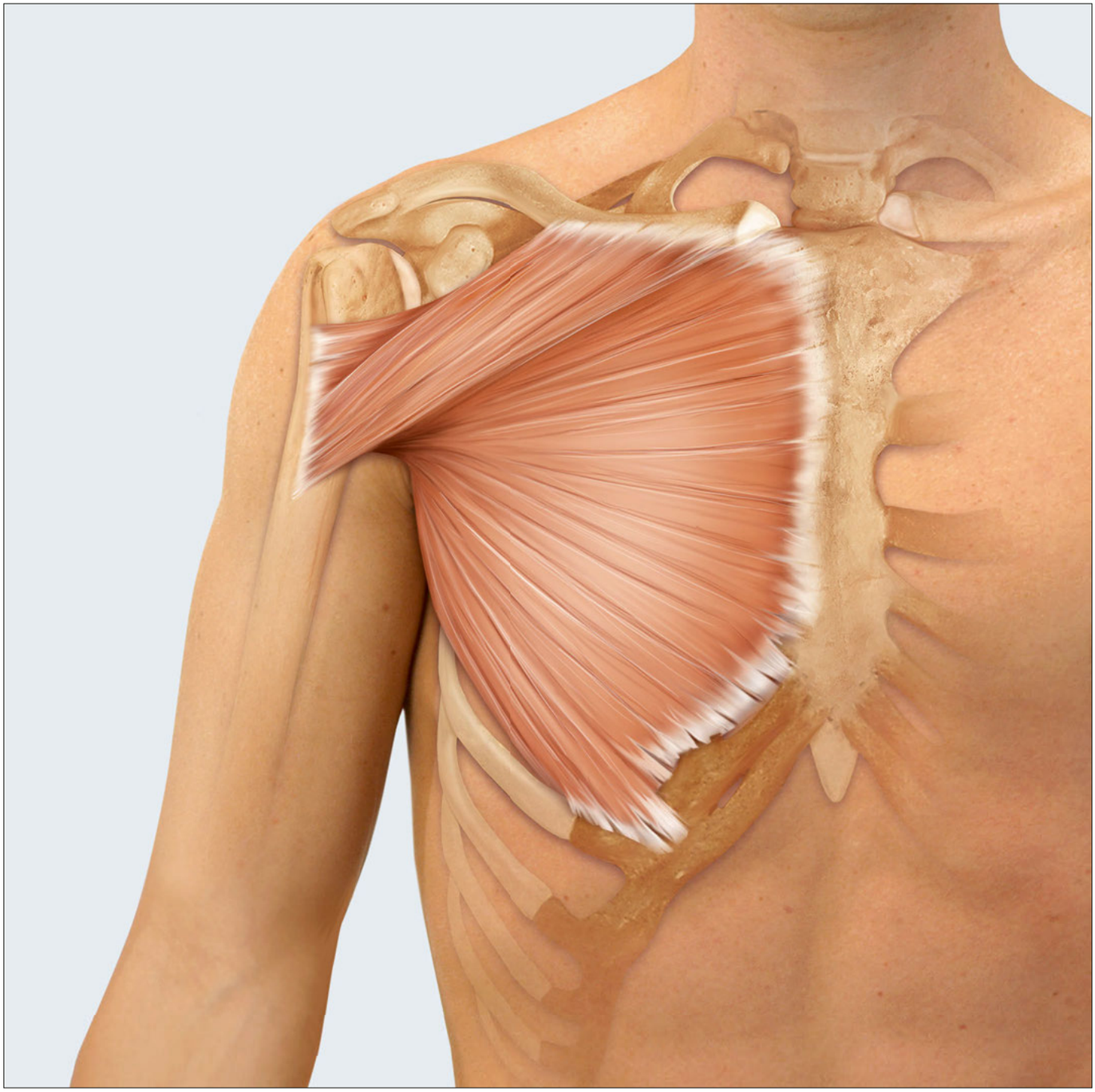


Fig 4.8 Suprascapular nerve. Out of plane approach for pain relief.

5.3 Lateral pectoral nerve: pectoralis major muscle



Nerve supply
Lateral pectoral nerve dominant innervation. The clavicular head receives C5–C6. Sternal head is C7–T1. Some innervation from the medial pectoral nerve C8–T1.

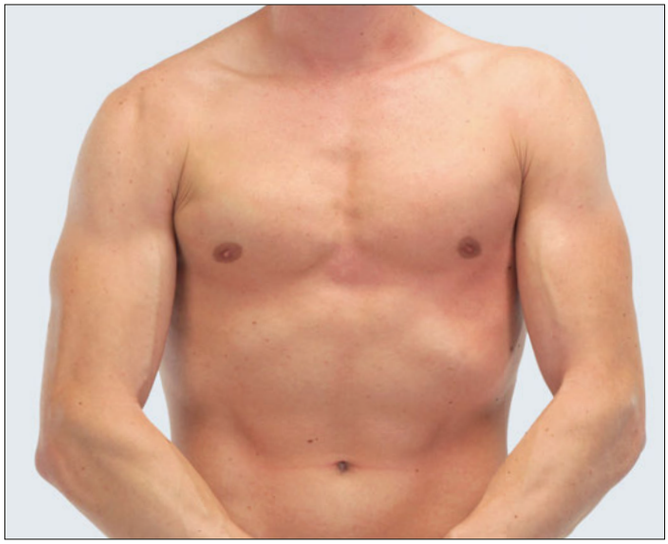
Origin

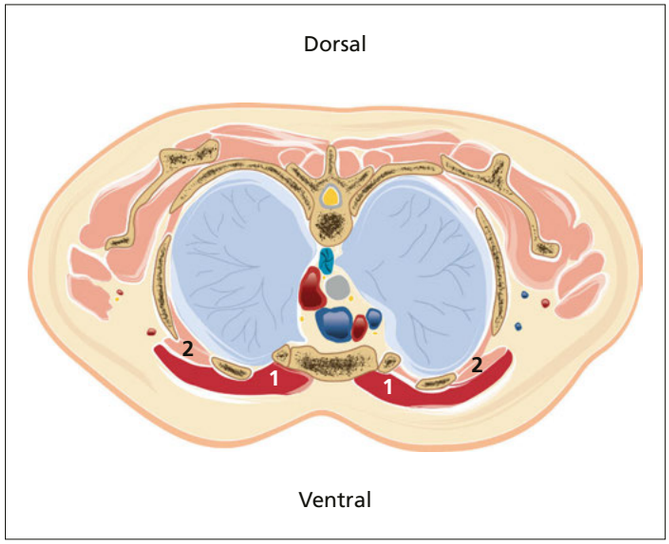
Clavicular atachment:	medial half of the anterior surface of the clavicle
Sternocostal attachment:	ventral surface of sternum, cartilage of sixth to seventh ribs, aponeurosis of the external oblique muscle of the abdomen
Abdominal attachment:	anterior lamina of the rectus sheath

Insertion
Crest of the greater tubercle of the humerus

Key learning points
Due to the highly variable course of the medial pectoral nerve, we recommend the lateral pectoral nerve in this book for the initial pectoral nerve block or procedure.

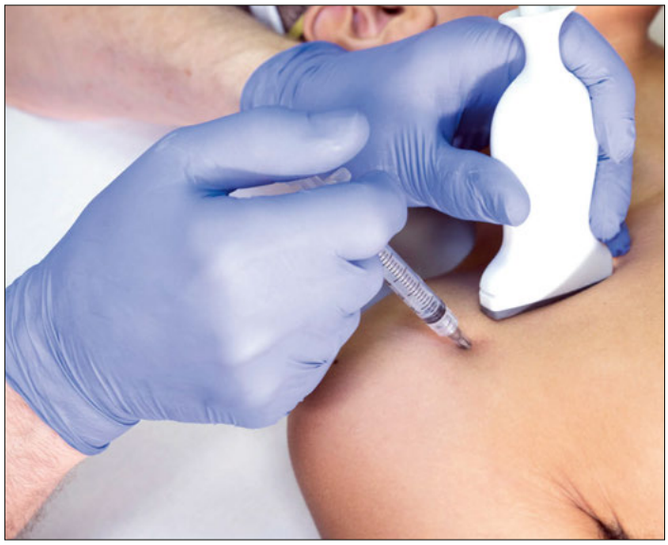
Lateral pectoral nerve stimulation to pectoralis major



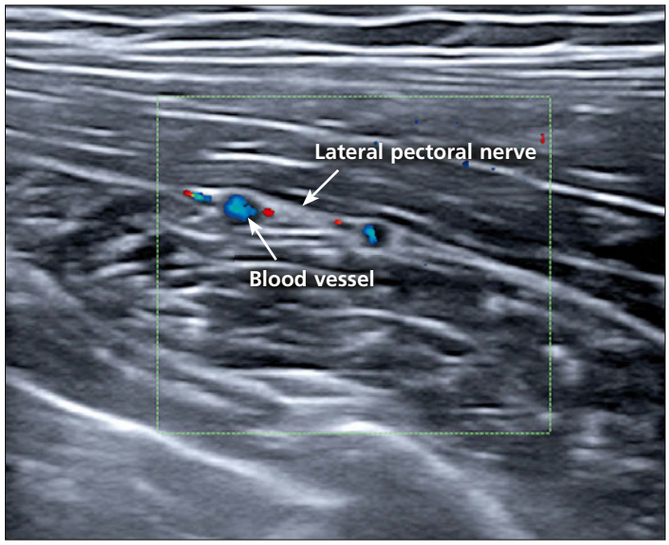


Action
The inferior and middle parts of the pectoral muscle (abdominal and sternocostal attachments) adduct and medially rotate the arm.
In addition to this, the isolated contraction of the clavicular fibers flexes at the shoulder joint (anteversion).

Topographic indication
(1) Pectoralis major muscle
(2) Pectoralis minor muscle



Injection technique
One-third of the distance between the sternoclavicular and acromioclavicular joint just inferior (0.5–2 cm) to the inferior border of the clavicle.



Key US principles
The lateral pectoral nerve is highly consistent. It lies along the inferior border of the pectoralis major and is found easily by locating the thoracoacromial blood vessels.

5.16 Median nerve: anterior interosseous nerve to flexor digitorum profundus muscle

Nerve supply

Lateral half: Anterior interosseous branch of median nerve (C8–T1)

Medial half: Ulnar nerve, C8–T1

Origin

Proximal anterior shaft of ulna

Forearm fascia

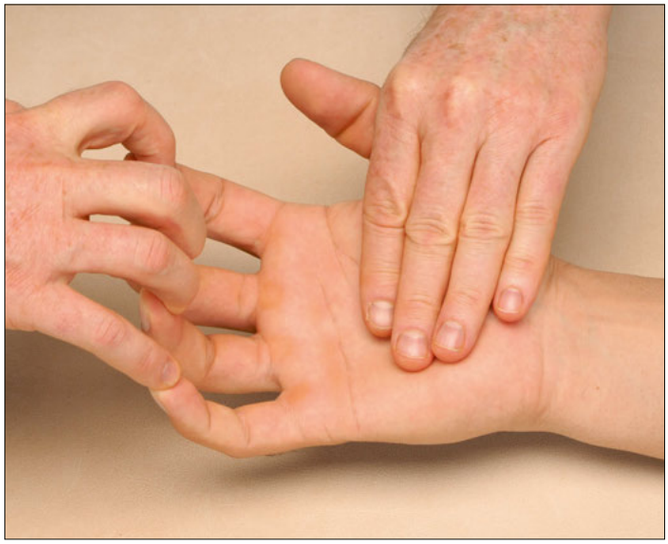
Interosseous membrane

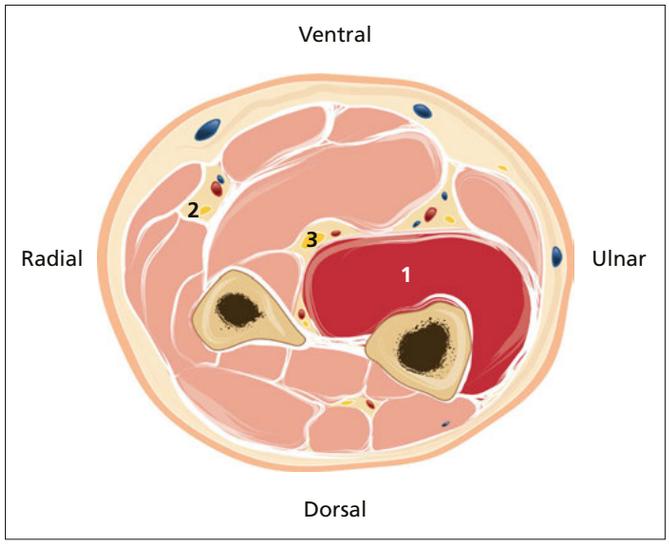
Insertion

Four tendons pierce those of the flexor digitorum superficialis and insert into the anterior surface of the base of the distal phalanges.

Key learning points

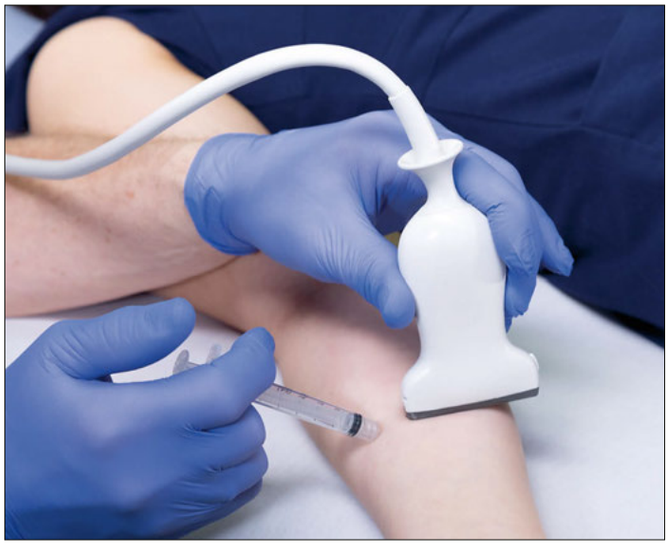
The flexor digitorum profundus (FDP) muscle fibers for the second and third digits are innervated by the anterior interosseous nerve (AIN). Most blocks at this level are done to the median nerve and all muscles distal.

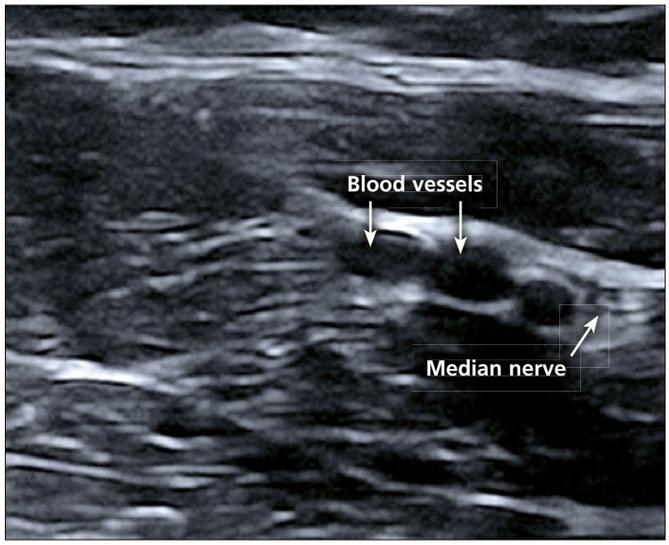




Action
The FDP flexes the metacarpophalangeal joints II–V and the respective proximal and distal interphalangeal joints. It is the only flexor of the distal interphalangeal joints. It is most powerful when the wrist is fixed by the extensors; otherwise, the FDP also flexes at the wrist joint.

Topographic indication
(1) FDP muscle
(2) Radial nerve, pars superficialis
(3) Median nerve

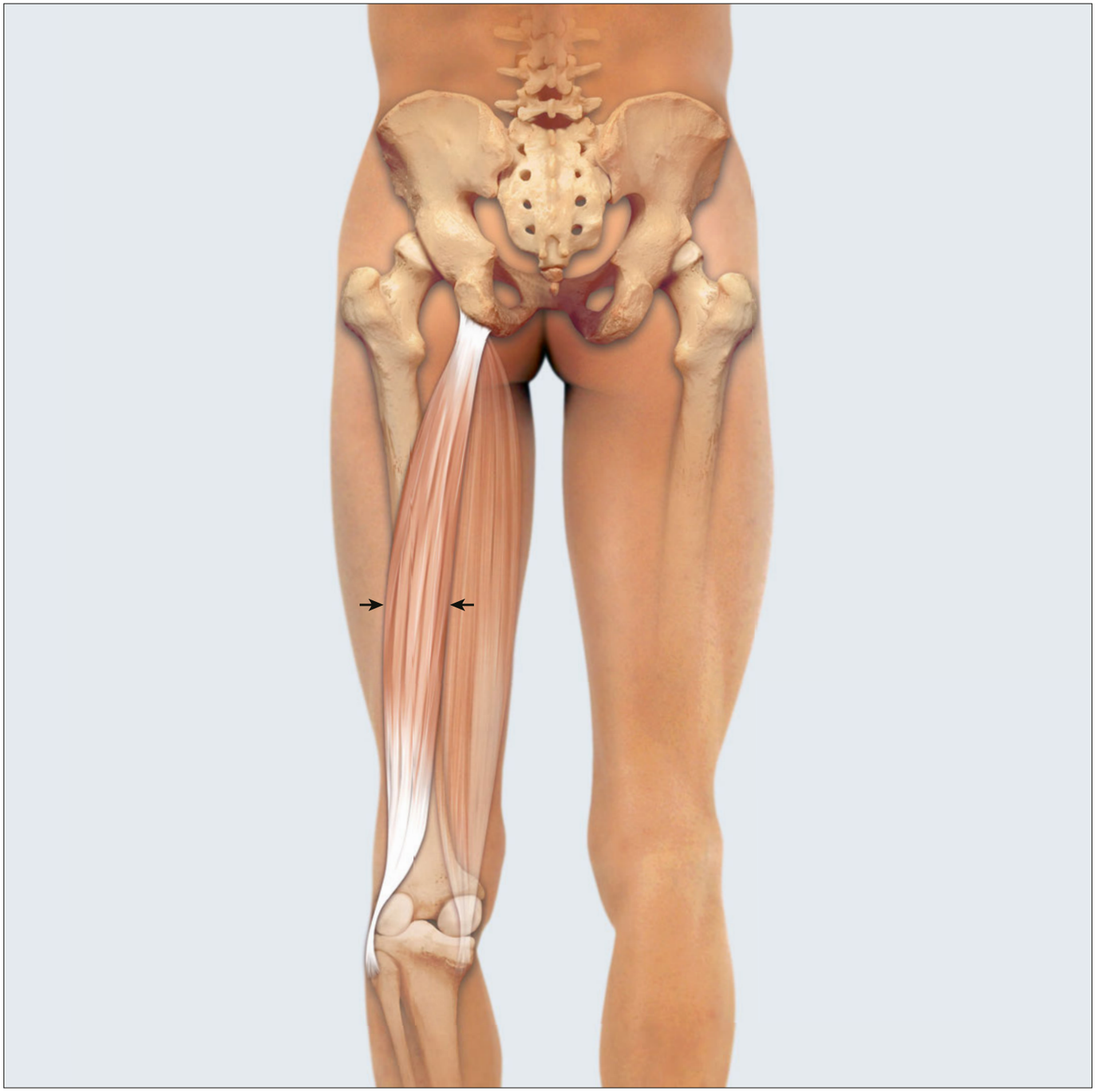




Injection technique
The location is found distal to the flexor digitorum superficialis (FDS) injection site and stimulation and is used to try to isolate the FDP to preserve FDS function.

Key US principles
The median nerve before its branch to the AIN is the typical target, seen next to the vascular bundle.

6.10 Sciatic nerve: biceps femoris muscle



Nerve supply

Long head: sciatic nerve, tibial part L5–S2

Short head: sciatic nerve, peroneal (fibular) part L5–S2

Origin

Long head: ischial tuberosity, sacrotuberous ligament

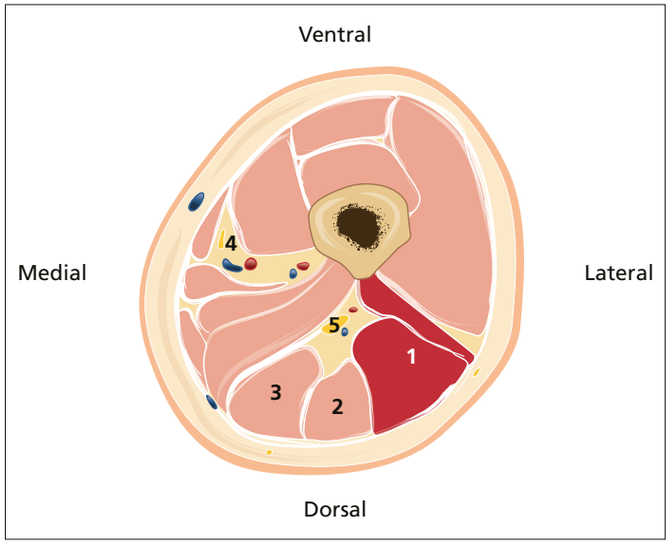
Short head: linea aspera, lateral intermuscular septum

Insertion

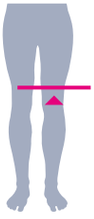
Lateral side of the head of the fibula and lateral condyle of the tibia

Key learning points

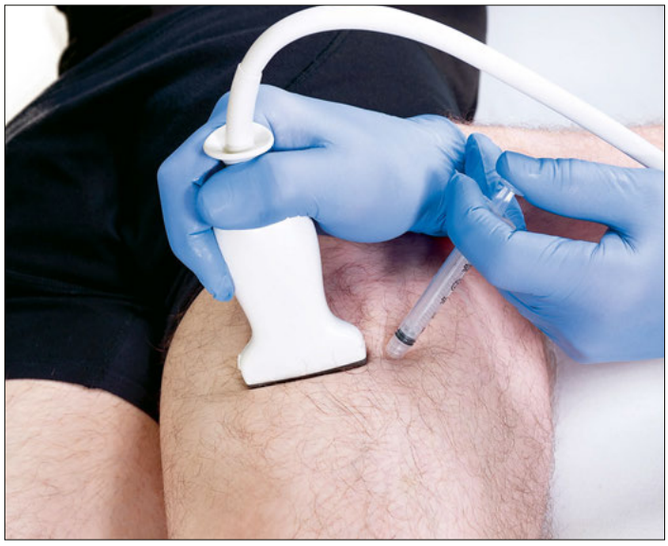
Not recommended for an ambulatory patient to test gait. For assessing degree of spasticity or contracture only.

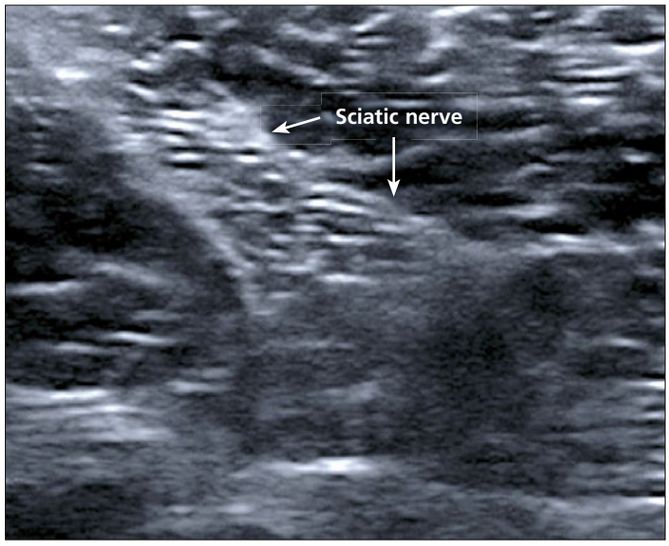


Action
The biceps femoris extends the hip joint and rotates the thigh laterally. It flexes the extended knee powerfully and can rotate the flexed knee laterally. The biceps femoris can bring the trunk into an upright position out of a forward-flexed position, indirectly flattening the lumbar lordosis.



Topographic indication
(1) Biceps femoris muscle
(2) Semitendinosus muscle
(3) Semimembranosus muscle
(4) Saphenous nerve
(5) Sciatic nerve





Injection technique
5–10 cm below the subgluteal fold.

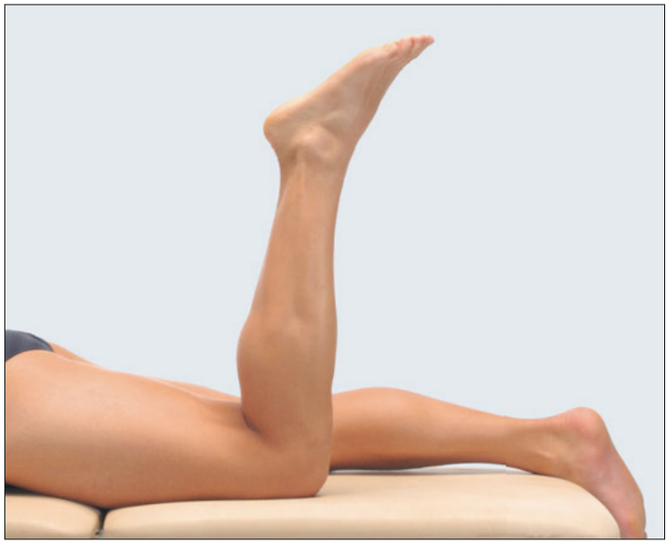
Key US principles
This block will affect the tibial and peroneal muscles distally.

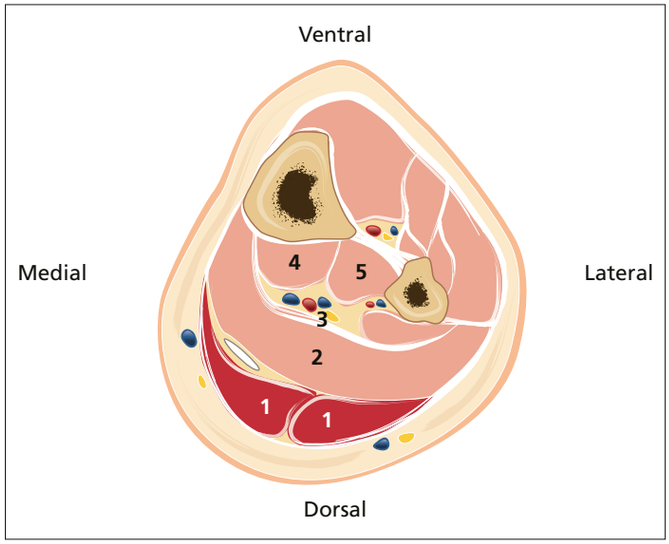
6.17 Tibial nerve: gastrocnemius muscle

Nerve supply
Tibial nerve, S1–S2
Origin
Medial head: popliteal surface of femur, medial condyle of femur
Lateral head: popliteal surface of femur lateral condyle of femur
Insertion
Calcaneal tuberosity via calcaneal tendon (Achilles tendon)

Key learning points
The medial and lateral branches can be individually targeted. The diagnostic nerve block to this location is targeted to avoid a sensory disturbance. The key choice when the ankle is fully reducible with the knee bent but not when extended.

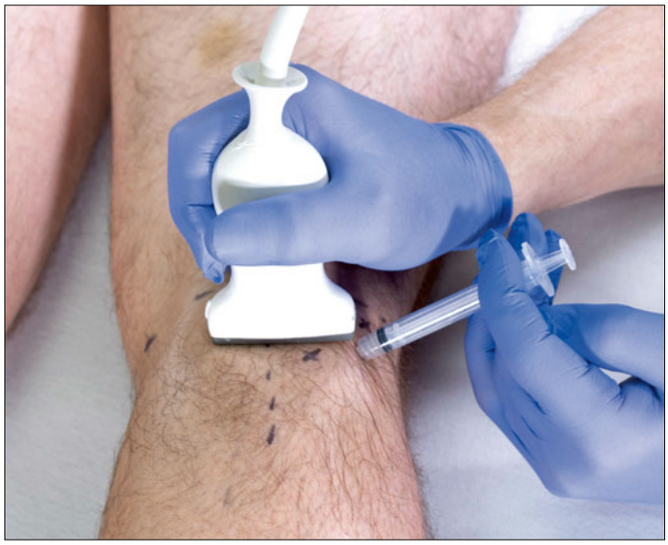
Tibial nerve branch to the lateral gastrocnemius head

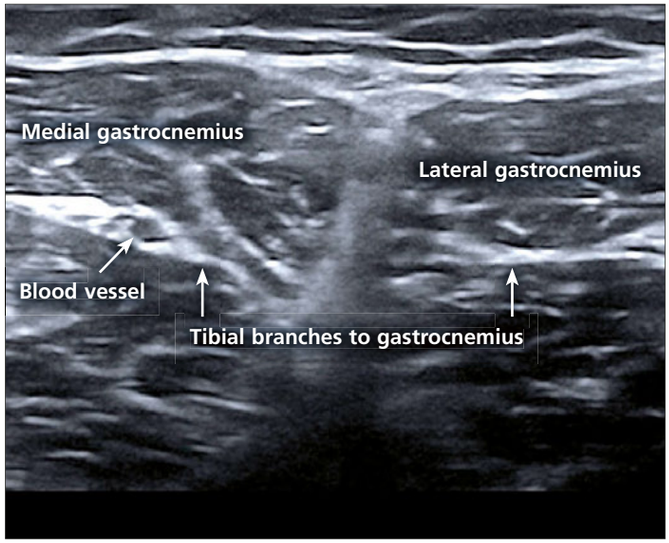




Action
The gastrocnemius is a powerful flexor both at the knee and ankle joints. It plays an important role in producing the required propulsion during the role-through and toe-off phases of walking. It inverts (supinates) the foot at the talotarsal joint, lifting the medial side of the foot as it flexes in the ankle joint.

Topographic indication
(1) Gastrocnemius muscle
(2) Soleus muscle
(3) Nerves and vessels (tibial nerve, posterior tibial artery and vein)
(4) Flexor digitorum longus muscle
(5) Tibialis posterior muscle





Injection technique
The tibial branches are best found by locating the trunk of the tibial nerve along side the artery and sliding distally to find the two branches

Key US principles
The injection is done in two parts; the lateral gastrocnemius is made with stimulation of the nerve for plantar flexion. The injection side and probe are made from a new medial injection site.