Electrophysiologic and Ultrasonographic Evaluation of Median and Ulnar Nerves in Spastic Upper Extremities in Chronic Stroke Patients: A review

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Abstract

Stroke is a leading cause of death and long-term disability in adults. In 2013, stroke was reported as the second leading cause of death after ischemic heart disease and the third leading cause of disability. Stroke is considered as a chronic disease. Spasticity is a common symptom after stroke, arising in about 30% of patients, and usually occurs within the first few days or weeks. However, the onset of spasticity is highly variable and can occur in the short, medium, or long-term post-stroke period. In the upper limbs, the most frequent pattern of arm spasticity is internal rotation and adduction of the shoulder coupled with flexion at the elbow, the wrist, and the fingers. In the lower limbs, the most observed pattern is adduction and extension of the knee with equinovarus foot. Peripheral neuropathies and muscle spasticity are among the most important complications to be considered. Use of ambulatory assistive devices, excessive use of non-paretic hand and wrist, edema, nerve traction or compression may cause peripheral neuropathies. Upper motor neuron lesions are also reported to cause some alterations in the lower motor neurons. This study aimed to evaluate the effect of spasticity in chronic hemiplegic upper extremities and electrophysiological characteristics of median and ulnar nerves. In conclusion post-stroke spasticity can affect the results and interpretation of NCS of peripheral nerves. Patients with spasticity may have lower NCV and amplitude, and false-positive or negative findings may occur. Clinicians should be aware of the potential limitations of NCS in patients with spasticity and use additional diagnostic tests as necessary to confirm the diagnosis.

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Introduction:

Stroke is a leading cause of death and long-term disability in adults. In 2013, stroke was reported as the second leading cause of death after ischemic heart disease and the third leading cause of disability. (1) Many complications might develop during stroke recovery that can affect patient's motor, sensory and cognitive functions. (2, 3)

Stroke is considered as a chronic disease. Spasticity is a common symptom after stroke, arising in about 30% of patients, and usually occurs within the first few days or weeks. However, the onset of spasticity is highly variable and can occur in the short-, medium-, or long-term post-stroke period. (4) A study by Wissel et al. (5) showed that 25% of patients with stroke suffer from spasticity within the first 6 weeks of the event. They also observed that spasticity primarily affects the elbow (79% of patients), the wrist (66%) and the ankle (66%). In the upper limbs, the most frequent pattern of arm spasticity is internal rotation and adduction of the shoulder coupled with flexion at the elbow, the wrist and the fingers. In the lower limbs, adduction and extension of the knee with equinovarus foot is the most observed pattern. (6)

Peripheral neuropathies and muscle spasticity are among the most important complications to be considered. Use of ambulatory assistive devices, excessive use of non-paretic hand and wrist, edema, nerve traction or compression may cause peripheral neuropathies. Upper motor neuron lesions are also reported to cause some alterations in the lower motor neurons. (7)(8)

Peripheral nerves of stroke patients have been investigated in many studies and several neurophysiologic alterations on paretic or non-paretic sides of stroke patients were reported .Decreased ulnar nerve compound motor action potential amplitudes

(CMAP) and sensory nerve action potential amplitudes (SNAP), pathologic spontaneous activities, slowing of the ulnar nerve motor and sensory conduction velocities, low M wave amplitudes, and increased number of complex motor unit potentials have been reported previously. (8)

The nerve conduction studies (NCS) of the hemiplegic upper extremity have shown a prolonged motor distal latency, reduced compound muscle action potential (CMAP) amplitude in the paretic limb compared to the non-paretic limb. (8)

In recent years, there has been a growing interest in the effects of spasticity on peripheral nerves in stroke. Median and ulnar nerves can be affected by the postural changes due to flexor spasticity causing flexion of the elbow, pronation of the forearm, flexion of the wrist in the upper extremity. It has been shown that during the flexed wrist and finger positions, the median nerve is exposed to the compression of the flexor tendons within the carpal tunnel. (8)

Ultrasound (US) has become an increasingly popular tool for visualizing soft tissues and peripheral nerves in the last decade. It provides a number of advantages over other imaging modalities since it is quick, dynamic, inexpensive, and non-invasive. There are no known adverse effects with the use of diagnostic US and therefore, no specific restrictions. It has already been found that peripheral nerves may undergo morphological changes in many clinical conditions (especially in carpal tunnel syndrome and polyneuropathies). (8)

However, ultrasonographic features of the peripheral nervous system have not been studied extensively in stroke patients. Certain studies reported that median and sciatic nerve CSA values were found to be smaller on the paretic sides when compared with the non-paretic sides of stroke patients. On contrary to this study other study reported higher median nerve CSA values in the affected extremities of spastic stroke patients. (8)

The purpose of this study is to evaluate the effect of spasticity in chronic hemiplegic upper extremities on electrophysiological measurements using nerve conduction studies and morphological measurements using neuromuscular ultrasound of median and ulnar nerves.

Ultrasonographic evaluation of median and ulnar nerves

High-resolution ultrasonography has rapidly gained acceptance as a first-line modality in the evaluation of peripheral nerves. Advances in scanner and transducer design over the last decade have enabled high-quality peripheral nerve imaging with resolutions equaling or surpassing those of magnetic resonance (MR) imaging. (9)(10)(11)

A study comparing US with MR imaging in the detection of peripheral nerve disease showed that although US and MR imaging had equal specificity (86%), US had greater sensitivity than MR imaging (93% vs 67%) (12). Although many centers routinely perform MR imaging to evaluate peripheral nerve disease, US offers several benefits over MR imaging. US is cheaper and faster to perform than MR imaging, and can also be used to image patients who are not eligible for MR imaging. US enables real-time dynamic evaluation of peripheral nerves when entrapment is suspected. An entire nerve can be quickly evaluated with US, whereas MR imaging is limited by coil and coverage constraints. (13)

Comparison with the contralateral side can easily be made with US. In addition, US has demonstrated clinical utility in patients with suspected peripheral nerve disease by guiding diagnostic and therapeutic decisions as well as confirming electrodiagnostic findings. (13)

Indications

US is useful for the evaluation of all manner of peripheral nerve disease. We are most frequently asked to evaluate for nerve entrapment such as carpal or cubital tunnel syndromes. A common request is for assessment of nerve morphology in the setting of trauma, particularly in cases of suspected laceration. Other indications include evaluation for tumor, infection, autoimmune disease, demyelinating or inflammatory polyneuropathy such as Charcot-Marie-Tooth disease, and diabetic neuropathy. (13)

An experienced sonographer can perform bilateral upper extremity US examinations in approximately 30 minutes, whereas MR imaging of bilateral upper extremities using high-resolution sequences would take 1 hour or more.

Technical considerations

Peripheral nerves should be scanned with high frequency, linear-array transducers. Superficial nerves should be scanned with a 17–5-MHz linear-array transducer; deeper nerves should be scanned with a 12–5 MHz transducer. The most superficial digital nerves can be studied with a

small-footprint 18–8-MHz “hockey stick.” It may be necessary to use lower-frequency transducers in larger patients. It is simplest to find a nerve in the short axis (the transverse plane) at an easily identifiable

anatomic landmark, and then follow the nerve up or down the extremity in the short axis. If disease is present, the abnormality may then be imaged in the long axis. It is useful to follow the same search pattern in every examination, commencing the examination at the

anatomic land-
mark where each nerve is most easily found, and then move proximally or distally from that site. It is helpful to learn the osseous, muscular, and vascular landmarks associated with each nerve. (14)

Electrophysiologic evaluation of median and ulnar nerve

As a fundamental component of the electrophysiological evaluation, nerve conduction study (NCS) is a vital and irreplaceable diagnostic tool to assess the overall condition of the peripheral nervous system and provides valuable quantitative and qualitative insights into neuromuscular function.(15-18) Actually, NCS has been significantly used in the identification and characterization of various neuromuscular disorders, particularly peripheral nerve diseases including nerve roots, peripheral nerves, muscle and neuromuscular junction, and is usually accompanied by a needle electromyography (EMG).(15-18)

Among the electrophysiological studies, NCS is one of the most widely used diagnostic tools, however it must be performed and interpreted with careful attention by either an experienced physician or a technologist under the supervision of an experienced physician. Motor, sensory, or mixed nerve studies can be performed, stimulating the nerve with the recording electrodes placed over a distal muscle, a cutaneous sensory nerve, or the entire mixed nerve, respectively. (17-20) The findings of motor, sensory, and mixed nerve studies often complement one another and yield different types of information associated with distinct patterns of abnormalities, depending on the underlying pathology. (17-20)

The principals and aim of nerve conduction studies

Peripheral nerves contain many nerve fibers of different diameters, degrees of myelination, and afferent or efferent connections. The NCS studies the fastest parts of these nerve fibers and the aim of the investigation is to document focal or continuous abnormalities in the length of the mixed, motor or sensory nerve. (15-18) Technically, a variety of methods may be used for stimulus or recording, however most NCSs currently are performed with cutaneous, or surface, stimulating and recording electrodes. (15-20)

Peripheral nerves may be stimulated through the skin with surface stimulators placed close to a nerve. NCS involves the application of depolarizing electrical waves to the skin over a peripheral nerve, followed by propagated nerve action potential (NAP) recorded at a distant point over the same nerve and a compound muscle action potential (CMAP) arising from the activation of muscle fibers in a target muscle supplied by the nerve. (16-19)

These action potentials may be commonly recorded with surface recording electrodes. Surface recording electrodes are designed to give information about the physiologic data for the time taken for the fastest axons to conduct an impulse to the muscle and the size of the response. (4) Abnormalities of nerve conduction studies may anticipate specific pathologic processes, such as demyelination or axonal loss, and may provide precise localization of focal nerve lesions. More specifically, the conduction velocity, latency and amplitude of reactions are assessed in the NCS. (15-21)

The general preparation for the NCS

The physicians should provide explanation of the examination to the patient so that they understand the examination, and notify the patient that electric stimulation may induce slight pain to help with the completion of the examination in a more cooperative manner. The physicians would help the patient to relax and put them in a comfortable position for the examination. The physicians may measure the skin temperature using a thermometer on the EMG machine or skin thermometer. Motor and sensory nerve conduction velocities are reduced by 1.3-2.4 m/s as the skin temperature decreases by 1°C. (16-23)

Nerve conduction velocities can be appropriately measured when the temperatures of the examination room and the patient's skin are 26°C and 30-36°C, respectively. If the examination site feels cold, skin temperature should be measured prior to the examination, and the appropriate treatment (i.e., fan heater or warm water) should be given
to raise the skin temperature. However, if a skin temperature controller or warm water are unavailable, skin temperature must be accurately measured and recorded so that the NCS results can be better interpreted.

It is crucial to minimize the impedance as much as possible; in fact, the impedance during the examination should remain below 5 kΩ. Typically, gel on the electrode or tape to hold the electrodes on the skin are used. In rare cases, the skin is rubbed with alcohol or acetone, needles, or sandpaper to reduce the impedance between the electrode and the patient's skin. When electrode impedance is at its minimum, there is no interference or noise at 60 Hz. Even if the effects from interference are negligible for motor NCSs, they can be an issue during sensory NCSs or combined NCSs, especially if the sensitivity is set below 5 μV.(16-23)

The ground electrode should be attached to the limbs to be examined, with the ideal position between the stimulation and recording electrodes. Having the current path run through the frontal chest should be avoided if possible. This can occur when performing an NCS on

the left arm and putting the ground electrode on the right arm. In order to minimize noise after stimulation, the ground electrode should be placed in the appropriate position between the stimulation site and recording electrodes. However, changing the position of the ground electrode for different examination sites is inconvenient, and it is, therefore, recommended that the ground electrode is placed at a set location on the arm or leg to be examined. This is the most appropriate method in most NCS cases. Nevertheless, if noise from stimulation hinders clear measurement of complex muscle or nervous action potentials, the ground electrode should be positioned between the stimulation and recording electrodes. Often, infants have small and short limbs, and therefore, specific electrodes for infants should be used. This prevents the stimulation from spreading to other nerves and ensures an adequate distance between the two stimulation areas to calculate nerve conduction velocity. (23)

Unexpected stimulation can also induce sudden movement during the examination, which can sometimes result in the detachment of the electrodes. Therefore, informing the patient prior to stimulation can help the patient to prepare for any pain associated with the stimulation. Additionally, in order to minimize pain, the shortest possible stimulation time and weakest intensity to induce a supramaximal response in the recorded muscle or nerve should be used. The patient can best endure the pain when stimulation is given at a rate of one stimulation per second. In healthy people, 0.05 ms of stimulation is sufficient to induce a supramaximal response. However, certain EMG machines have a minimum stimulation time greater than 0.05 ms, and therefore, the stimulation time should be set accordingly. Patients with neurological conditions often require longer stimulations to induce a supramaximal response. (23)

In sensory and combined NCSs, these patients may not exhibit any increase in the amplitude of compound nerve action potentials even with stimulation duration above 0.2 ms. In motor NCSs, the stimulus duration must be increased up to 1.0 ms, in some cases, to obtain a response. These findings suggest that patients with neurological disorders exhibit a lower level of nervous system activity assuming that the stimulation and recording electrodes are appropriately positioned. (18-23)

The machine settings for NCSs should be fixed to default, unless alterations are needed. It is important to observe overall responses on the oscilloscope monitor. The latency, compound muscle action potentials, and compound nerve action potentials should all be clearly observable. Often, the settings for sensitivity and sectional velocity need to be changed; for example, if the latency, amplitude, and duration of a compound muscle action potential are 40 ms, 1,500 μV, and 40 ms, respectively, the sensitivity and sectional velocity should be respectively changed to 500 μV and 10 ms.The two most important machine settings for NCSs are the stimulus duration and sectional velocity.(18-23)

Supramaximal stimulation should be obtainable after an appropriate adjustment of stimulation duration and intensity. Changing the sectional velocity can result in an inaccurate measurement of latency. Because sensory and combined nervous action potentials have small amplitudes, the patient must be completely relaxed to accurately record these. With the audio monitor on, the patient hears the noise and can, therefore, be more relaxed. (23)

The procedure of motor NCS:

Motor conduction study is performed by electrical stimulation of a nerve, and recording the CMAP from surface electrodes overlying a muscle supplied by a stimulated nerve. The recording electrodes are placed over the skin overlying the target muscle using adhesive conductive skin electrode. The active electrode (known as G1) is placed over the muscle belly and the reference electrode (known as G2) over an electrically inactive site such as usually the muscle tendon. A ground electrode is also placed somewhere between the stimulating and recording electrodes providing a zero-voltage reference point. The stimulator then is placed over the nerve that supplies the muscle, with the cathode placed closet to the recording electrode. In most electrodiagnostic studies, routine motor NCS of the arm include median nerve (recording the abductor pollicis brevis muscle) and ulnar nerve (recording the abductor digiti minimi muscle), and in the leg, the tibial nerve (recording the abductor hallucis muscle) and peroneal nerve (recording the extensor digitorum brevis muscle). (15-19)

Proximal stimulation sites can be added to address specific clinical questions (e.g., stimulating the ulnar nerve above and below the elbow, or common peroneal nerve above and below the fibular head to assess for evidence of demyelination). In many cases these routine studies must be supplemented to adequately assess an area of potential injury. In the arm, motor studies of the radial nerve (recording the brachioradialis, extensor digitorum communis, or extensor indicis proprius muscles), ulnar nerve (recording the first dorsal interosseous muscle), musculocutaneous nerve (recording the biceps muscle), or axillary nerve (recording the deltoid muscle) may be indicated. (24)

The CMAP is the summation of all underlying

individual muscle fiber action potentials. The latency of the CMAP is the time from stimulus to the initial CMAP deflection from the baseline and is a biphasic potential with an initial upward deflection (negativity) followed by a smaller downward deflection (positivity). The CMAP amplitude is measured from baseline to negative peak which is demonstrated by an upward deflection and measured in millivolts (mV) (Fig. 1).(15-23)

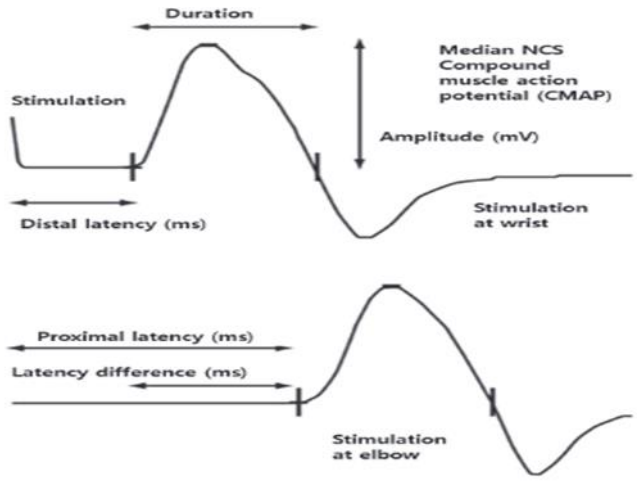

Figure (1) Compound muscle action potential (CMAP) in median motor nerve conduction study (NCS).

The difference in latency represents the time taken for the fastest nerve fibers to conduct between the two stimulation points as all other factors involving neuromuscular transmission and muscle activation are common to both stimulation sites (Fig.1). If one measures the distance between the two sites then the fastest motor nerve conduction velocity (NCV) can be calculated as follows:

NCV (m/s) = distance between stimulation sites (mm) / (proximal latency-distal latency [ms]). (24,25)

The procedure of sensory NCS

The sensory nerve action potential (SNAP) is obtained by electrically stimulating sensory fibers and recording the nerve action potential at a point along the same nerve. In the sensory NCS, technical factors and electrical noise assume more importance, because most sensory responses are very small. And, once again the stimulus must be supramaximal. A pair of recording electrodes (G1 and G2) are placed in line over the nerve at an interelectrode distance of 3 to 4 cm, with the active electrode (G1) placed closest to the stimulation (fig. 2). (25)

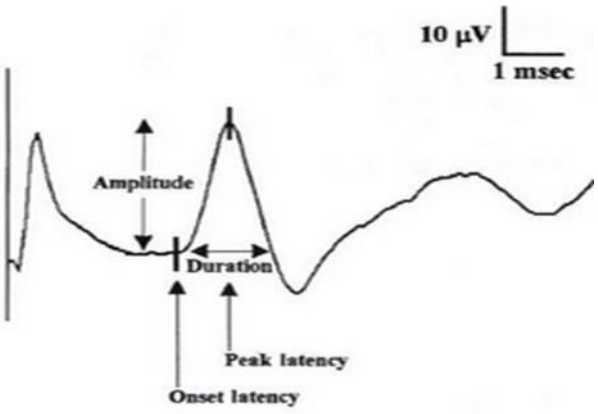

Figure (2) : The sensory nerve action potential (SNAP)

Recording the SNAP orthodromically refers to distal nerve stimulation and recording more proximally; the direction in which physiological sensory conduction occurs. (15-23)

The procedure of F waves

F waves are a type of late motor response that occurs after the CMAP. When a motor nerve axon is electrically stimulated at any point an action potential is propagated in both directions away from the initial stimulation site. (25). The most reliable measure of the F wave is the minimum latency of 10-20 firings. F waves allow testing of proximal segments of nerves that would otherwise be inaccessible to routine nerve conduction studies. The F wave ratio which compares the conduction in the proximal half of the total pathway may be used to determine the site of conduction slowing, particularly, to distinguish a root lesion from a patient with a distal peripheral neuropathy. Furthermore, the circuity of the H reflex involves the Ia muscle spindles as sensory afferents and the alpha motor neurons and their axons as efferents. If a low submaximal stimulus with a long duration is applied to a nerve, it is possible to selectively activate the Ia fibers. The gain must be increased initially to 200 to 500 uV (fig. 3). (25)

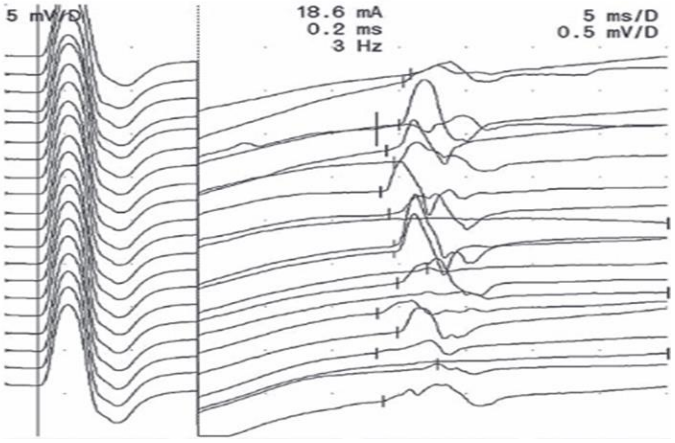

Figure (3): F-waves recorded from the median nerve.. Twenty responses have been recorded. The compound muscle action potential (CMAP) response is seen at the

left of the traces. The F-waves are seen on the right hand side of the traces. These result from the fi ring of different individual motor neurons and the single motor unit that they supply. They therefore show a range of latencies and morphologies, and their amplitudes are also much smaller than the CMAP response

Post Stroke Spasticity

Upper Motor Neuron Syndrome and spasticity after stroke:

Stroke can cause Upper Motor Neuron Syndrome (UMNS) which is manifested in two ways:

A) Loss of motor function such as muscle weakness, limb flaccidity and fatigue, a result of lack of motor activity.

B) Muscular over activity such as spasticity, increased tendon reflexes, clonus and extensor/flexor spasm. (26-29)

Spasticity is more common in younger individuals (30-31) after stroke and mostly impacts upper extremities (32). Spasticity can occur immediately after stroke or as much as one year later (33). It reaches its peak one to three (34) months after onset (35). Spasticity has been defined as "a motor disorder characterized by a velocity dependent increase in the tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome" (36). This definition implies that stretch reflex hyperactivity is the sole cause of spasticity (37). Pandyan (2005) elaborated on Lance's definition explaining that there is not enough evidence to indicate that spasticity results only from stretch reflex hyperexcitability. He proposed that the supraspinal systems and afferent pathways may play a role as well, and advanced the following definition: "a disordered sensori motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscles" (37).

In 2012, Ward proposed that a universal definition (33) was not possible because spasticity was not a "single entity" and that there was considerable clinical manifestation among patients; therefore, there cannot be a universal definition.Spasticity impacts the individuals' function; in a prospective study, Koski et al., (1996) showed that spasticity may lead to impaired balance and gait, falls and bone fractures. Spasticity may cause pain, contracture and skin breakdown (17,18). Welmer et al. (2006) found a moderate correlation between spasticity and quality of life (38).

In one study, it was shown that six months after stroke, 50% of individuals with spasticity develop contracture (39). Usually after stroke, spasticity develops differently in the upper and lower extremities; flexors in the upper limb and extensor muscles in the lower limbs are most typically involved(40).

Prevalence of spasticity:

The prevalence of spasticity is variable and ranges from 19% to 92% (22). The onset of spasticity also varies and could happen in the short, medium or long-term after stroke (33). In the short term, Somerfield and colleagues (2004) showed that in the first week after stroke, 21% have spasticity3. Similarly, Wissel et al. (2010) followed 103 post stroke patients at six days, six weeks and 16 weeks post-stroke; the results indicated that 24.5% developed spasticity within two weeks after stroke (23). For the medium term, Urban et al., (2010) showed that six months after stroke, 43% developed spasticity (41), and in the long term, a year post-stroke, Lundstrom et al. (2008) showed a rate of 21% (31).

The variability in prevalence estimates of spasticity in the post-stroke population may depend on the tools used to measure it. Reported rates of spasticity might be lower than the actual rates because of the low sensitivity of measurement tools (42). Initially, researchers and clinicians relied on clinical measures such as the Modified Ashworth Scale (MAS) to measure spasticity (43).

Neurophysiology of spasticity:

The motor control system encompasses four major components: cerebral cortex, subcortical structures, brainstem, cerebellum and spinal cord. The cerebral cortex is critical for directing signals for preparation and execution of movements through different areas such as premotor and supplementary motor areas which program and plan voluntary movements and the primary motor cortex, which controls execution of movement. Subcortical centres, including basal ganglia are essential for movement coordination and sustaining tone(44).

The brainstem plays an important role in stretch reflex, posture and repetitive movements. The spinal cord is the last pathway for motor movement and it encompasses components of spinal circuitry that coordinates movement and alpha motor neurons that innervate muscles (44). At the spinal level, motor function is controlled by three elements: afferent input from sensory receptors, interneurons, and reflex activity. In stroke patients, two categories could mediate spasticity: abnormal intraspinal control of stretch reflex and/or supraspinal control with abnormal descending pathway (45).

Stretch reflex and intraspinal control

At the spinal level, the stretch reflex is one of the factors that regulate muscle function(40,46). Muscle tone occurs due to the excitation of alpha motorneurons located at the anterior horn of the grey matter and ongoing sensory input about the muscle's length from

each muscle to the spinal cord (47). In the muscle spindles, the sensory receptors in the intrafusal muscle fibers (i.e. muscle spindles) convey information about muscle length to the spine (45).

The efferent fibers include alpha motor neurons which innervate extrafusal fibers and gamma neurons that innervate muscle spindles (48) to help maintain sensitivity to stretch. Stretch reflex continuously generates muscle tone by activating muscle spindles. Muscle tone is a basic muscle function that helps the body to maintain body posture against gravity. When there is a change in muscle length, both gamma and alpha fibers (alpha-gamma co-activation) are excited which leads to the contraction of both extrafusal and intrafusal muscle fibers(44).

Effect of post stroke spasticity on morphological features of peripheral nerves:

Post-stroke spasticity is a common complication of stroke, affecting up to 40% of patients in the chronic phase. It is characterized by involuntary muscle contractions and hyperactive reflexes, leading to stiffness, pain, and impaired motor function. While the pathophysiology of post-stroke spasticity is not fully understood, recent evidence suggests that it may be associated with morphological changes in peripheral nerves. Peripheral nerves are an essential component of the motor system, transmitting motor commands from the central nervous system to the muscles and providing sensory feedback. Morphological changes in peripheral nerves may affect their ability to conduct motor and sensory signals, contributing to the development and persistence of spasticity. Several studies have investigated the effect of post-stroke spasticity on the morphological features of peripheral nerves, with mixed results. (49)

One study by Wang et al. (2018) used high-resolution ultrasound to compare the morphological features of the median nerve in patients with and without post-stroke spasticity. They found that the cross-sectional area of the median nerve was significantly larger in patients with spasticity than in those without, suggesting that spasticity may cause nerve hypertrophy. However, they did not find any differences in nerve echogenicity or fascicular pattern between the two groups. (50)

Another study by Li et al. (2018) used magnetic resonance imaging (MRI) to investigate the morphological changes in the tibial nerve in patients with post-stroke spasticity. They found that the tibial nerve cross-sectional area was significantly larger in patients with spasticity than in healthy controls, and that the nerve morphology was characterized by increased fascicular number and decreased nerve fiber density. These findings suggest that spasticity may cause nerve hyperplasia and fiber degeneration. (51)

In contrast, a study by Kim et al. (2019) used ultrasound to compare the morphological features of the median nerve in patients with and without spasticity and found no significant differences in nerve cross-sectional area, echogenicity, or fascicular pattern between the two groups. (52)

The conflicting results of these studies may be due to differences in the patient population, the severity and duration of spasticity, and the methods used to assess nerve morphology. Further research is needed to clarify the relationship between post-stroke spasticity and morphological changes in peripheral nerves and to explore the potential mechanisms underlying this relationship. (52)

Post-stroke spasticity may be associated with morphological changes in peripheral nerves, including nerve hypertrophy, hyperplasia, and fiber degeneration. However, the evidence regarding the effect of spasticity on nerve morphology is mixed, and further research is needed to elucidate the underlying mechanisms and to develop effective interventions for the prevention and treatment of spasticity-related nerve damage. (52)

Effect of post stroke spasticity on nerve conduction studies of peripheral nerves

Post-stroke spasticity is a common neurological complication of stroke, characterized by increased muscle tone and hyperreflexia. It affects about 30-40% of stroke survivors, and can significantly impair functional recovery and quality of life. The pathophysiology of post-stroke spasticity is complex and involves both central and peripheral mechanisms. Nerve conduction studies (NCS) are a useful diagnostic tool to evaluate the function of peripheral nerves in patients with spasticity. However, the presence of spasticity can affect the results of NCS, leading to false positive or negative findings. (53, 54)

Several studies have investigated the effect of post-stroke spasticity on NCS of peripheral nerves. A study by Kim et al. (2014) found that patients with spasticity had significantly lower motor nerve conduction velocity (NCV) and amplitude compared to patients without spasticity. Similarly, another study by Lee et al. (2015) reported that patients with spasticity had significantly lower sensory NCV and amplitude compared to patients without spasticity. These findings suggest that spasticity can affect both motor and sensory nerve function. (53-55)

The mechanism by which spasticity affects NCS is not fully understood, but several theories have been proposed. One theory suggests that spasticity leads to increased muscle stiffness and resistance, which can cause a decrease in NCV and amplitude. Another theory

proposes that spasticity may affect the integrity of peripheral nerves, leading to axonal degeneration and demyelination. Additionally, spasticity may cause abnormal firing patterns in motor and sensory nerves, which can further affect NCV and amplitude. (53)

In addition to affecting NCS parameters, spasticity can also affect the interpretation of NCS findings. For example, spasticity can lead to false-positive findings of carpal tunnel syndrome (CTS), as patients with spasticity may have increased median nerve conduction slowing due to increased resistance in the wrist flexor muscles. Similarly, spasticity can lead to false-negative findings of peripheral neuropathy, as patients with spasticity may have preserved NCS parameters despite the presence of neuropathic symptoms. (54)

Despite the potential limitations of NCS in patients with spasticity, this diagnostic tool remains an important component of the clinical evaluation of peripheral nerve function. Clinicians should be aware of the potential effects of spasticity on NCS findings and interpret the results in the context of the patient's clinical presentation. In some cases, additional diagnostic tests such as electromyography (EMG) may be necessary to confirm the diagnosis. (54)

In conclusion post-stroke spasticity can affect the results and interpretation of NCS of peripheral nerves. Patients with spasticity may have lower NCV and amplitude, and false-positive or negative findings may occur. Clinicians should be aware of the potential limitations of NCS in patients with spasticity and use additional diagnostic tests as necessary to confirm the diagnosis.

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