The Perception of a Tactile Illusion in Central and Peripheral Nervous System Injury

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Abstract

Neuropathic pain patients often have lemniscal-mediated symptoms such as tactile allodynia. A novel method and device for testing lemniscal pathway integrity is described using a tactile illusion (TI) in patients with neuropathic pain resulting from peripheral nervous system (PNS) or central nervous system (CNS) injury. An unexpected enhanced perception of the TI was found in some PNS injured pain patients with mononeuropathies, in the majority of cervical spinal cord injured and in nearly half of brainstem injured central pain patients. A disinhibition of lemniscal pathway mediated TI was found in these patients. A subset of thalamic pain patients have a unique TI perseveration or persistent after-sensation despite reduced TI perception on their affected side compared to their normal side. The results of these studies indicate a unique form of sensory disinhibition not previously described.

Introduction.

Sensory illusions occur in the tactile, auditory and visual systems [1]. The tactile illusion (TI) of movement includes the phi phenomenon and is defined as the perceived movement between 2 or more fixed alternating points of vibration on the skin [2]. The TI perception is dependent on intact peripheral and central nervous system pathways including lemniscal pathways. The lemniscal pathway to the cerebral cortex is necessary for tactile discrimination. Cortical integration of peripheral stimulation is needed to form the TI of perceived movement between points of vibration that form symbols such as numbers "written" on the skin. This is distinguished from graphesthesia, defined as the ability to recognize numbers traced lightly on the skin with a stylus or pencil. Both lemniscal (pressure, touch, and vibration) and reticular (pain and temperature) neural pathways can be affected by a disease of the peripheral and central nervous system. This is due to the close anatomic proximity of these pathways so that pathologic injuries, such as diabetic neuropathies, traumatic nerve and spinal cord injuries, strokes to the brain, etc., primarily affecting one pathway can often affect the other. Injury to a single peripheral nerve can result in a perversion of the tactile sense, called allodynia, in which normally innocuous light touch (mediated by lemniscal pathways) is perceived as noxious or painful. Noxious stimuli are normally only mediated by reticular pathways. Disease or injury to the central nervous system (CNS) may produce similar distortions of lemniscal and reticular pathway-mediated sensations.

In these studies patients with CNS and peripheral nervous system (PNS) injury were tested using a device that produced the TI of numbers "drawn" on the skin across several dermatomes. These studies are described below and details have been recently published [3, 4].

Methods.

In the 2 studies described, patients were identified from the authors' [GRG] clinical practice, Mayo Clinic, Scottsdale, Arizona, U.S.A. An institutional review board protocol was submitted and approved for these investigations, and informed consent was obtained from all patients and controls. A room that was free of distractions was used in testing all subjects in both studies. The tactile device holds solenoids that vibrate and stimulate discreet cutaneous points and produce the illusion of "drawing" complex symbols (i.e. numbers) on the skin where the device is applied (see Figure 1).

In the first study, 27 patients with unilateral central pain resulting from a CNS injury to the cervical spinal cord, brain stem, lateral thalamus, or suprathalamic brain regions were studied using this device. The device is applied to the volar wrist on the unaffected, normal side first and then to the affected, painful side. In a second study, 8 subjects with PNS injury were tested using the same study procedures. The magnitude of the learning effect of delivering sequential stimuli was determined by testing the device on 10 normal, testing-naïve individuals. The learning effect, \(L\), was expressed as \(L = (T_1 / T_2) \times 100\), where \(T_1\) is the total number of attempts to identify the 10 random digits presented on the first body side tested and \(T_2\) is the total number of attempts to identify the 10 random digits presented on the second body side tested. The first side was tested immediately after testing the second side.
Results. In measuring the learning effect, 9 of the 10 normal subjects improved in that they required fewer trials to identify correctly the 10 randomly delivered numbers on the second side tested. The median learning effect, $L$, was 1.23%. This constant, $L$, was then applied to the number of trials on the affected side of the patients with pain from CNS and PNS injury to yield the Adjusted-for-Learning values $= 1.23x$, where $x$=the number of trials on the affected side.

As expected, in the first study, 8 of 10 patients with thalamic and suprathalamic central pain had decreased TI perception on the affected side. Unexpectedly, 7 of 10 patients with cervical spinal cord central pain, and 3 of 7 patients with brainstem central pain had better TI perception on their affected side. In other words, fewer number of trials were needed for them to correctly identify the number presentation when their abnormal side was compared to the normal side. The enhancement of the perception of the TI in the majority of patients with cervical spinal cord central lesions and in nearly half of the brainstem-injured patients was unexpected. Another observation from this study was that 2 of 4 patients with thalamic injury had a unique phenomenon of TI perseveration, despite a decreased TI perception on the affected side. This perseverance was maintained and more than one number could be felt simultaneously even after subsequent stimulation to that body part was discontinued [3].

In the second study, performed on 8 patients with PNS injury and mononeuropathy with tactile allodynia, 3 of 8 patients unexpectedly had better TI perception on the affected side compared to their unaffected side [4].

Discussion. Lesions within the PNS and CNS that cause pain are often associated with lemniscal pathway injury as well. For example, a mononeuropathy caused by a traumatic injury to the ulnar nerve will often injure motor and sensory fibers. The injured sensory fibers are destined for both the pain- and temperature-mediating reticular, spinothalamic pathways as well as touch-, pressure-, and vibration-mediating lemniscal, posterior column pathways in the spinal cord. Lesions in the CNS, in particular those above the spinal cord level, are associated with both reticular and lemniscal pathway injury due to the close proximity of the pathways in brainstem, lateral thalamic, and suprathalamic regions. Spinal cord lemniscal pathways are located in the posterior spinal cord whereas reticular pathways are located primarily in the anterior spinal cord. None-the-less spinal cord injuries from conditions such as infarcts, trauma, multiple sclerosis plaques and others will often times produce injury to both sensory pathways.

Even though spinal cord, brainstem and suprathalamic lesions can cause central pain without thalamic injury, central pain is dependent upon thalamic modulation [5]. The accentuated perception of the TI in some PNS and CNS neuropathic pain patients are consistent with a proposed central disinhibition theory. This theory posits that "tonic activation from net descending facilitation of supraspinal sites" [4], results in improved cortical
processing of the TI [5, 6]. In the thalamic injured central pain patients with TI perseveration, the possible mechanism of this phenomenon may include TI aftersensations released from the disinhibited cortex or thalamus, similar to the tactile movement aftereffect mechanism proposed by Hollins and Favorov [7].

The device in Figure 1 produced cutaneous stimulation over several dermatomes. Theoretically a device that would stimulate a single dermatome will provide more precise determination of peripheral inputs needed to produce this illusion, which is ultimately caused by brain cortical activity. Newer devices in development will activate a smaller area of skin on the “tactile rosette” of the index finger, in a single dermatome, and will spatially reduce the area of CNS somatotopic activation (see Figures 2A and 2B).

The index fingertip is also known to have a higher density of Pacinian corpuscles than more proximal cutaneous sites and has a greater primary, and possibly secondary, parietal cortical representation than more proximal cutaneous dermatomes. Localization using a single dermatome TI device on the fingertip will assist in the interpretation of CNS imaging using functional magnetic resonance imaging (fMRI). Therefore, this type of device will be helpful in determining factors that may modulate TI discrimination as well as injury associated sensitivity changes of the lemniscal system. This may also help define whether the newly described observation of TI perseveration in subsets of patients with central pain is dependent on multiple peripheral nerve or dermatomal activation, or spatial summation in a single nerve or dermatomal distribution.

References.


