Introduction

A recent Canadian study highlighted chronic neuropathic pain as highly prevalent and clinically significant [1]. It affects an estimated one in six adults and is associated with greater functional restriction and lower quality of life than standard chronic pain without neuropathic symptoms. Despite being common and important, a study of 1230 physicians revealed that a majority feel that the diagnosis and treatment of various neuropathic pain syndromes is challenging. Indeed, evaluation of patients does require a detailed history of pain and neuropathic features as well as careful physical examination of associated findings.

Defining neuropathic pain

Prior to 2008, the International Association for the Study of Pain (IASP) defined neuropathic pain as "pain initiated or caused by a primary lesion or dysfunction in the nervous system." The word dysfunction allows for a diagnosis based on symptoms even when a clear lesion is not identified. In 2008 the definition of neuropathic pain was revised by NeuPSIG, the Special Interest Group on Neuropathic Pain of IASP, to be "pain arising as a direct consequence of a lesion or disease affecting the somatosensory system" [2]. A further grading system of "definite", "probable," or "possible" neuropathic pain was proposed based on the likelihood of showing a lesion or disease [3]. Under such a scheme, patients presenting with post-herpetic neuralgia (PHN) – long considered a classic example of neuropathic pain – would merit only a grade of probable neuropathic pain in clinical situations because confirmation of a lesion or disease is not routinely made.

In practice, the diagnosis of neuropathic pain is made on history and physical examination. This chapter will summarize a standard approach to identifying neuropathic pain for the clinician.

History

A comprehensive history to be elicited from patients with chronic neuropathic pain involves focusing not only on the description of pain, but also the constellation of frequently associated changes in mood, sleep, and functioning. Like many conditions in medicine, the majority of neuropathic pain syndromes are diagnosed on history while examination is used for subsequent confirmation.

Patients with chronic pain often feel that their complaint of pain is minimized by healthcare providers and not appreciated or understood by their families. For this reason, it is best to initiate the medical history by permitting the patient to fully describe their pain experience as they understand it. Once a history is obtained, then the mnemonic OPQRST can be used to ensure that all important components to a standard pain history are obtained (Table 1.1).

For neuropathic pain, and for the condition of complex regional pain syndrome (CRPS) especially, the six Ss (Table 1.2) should be queried when obtaining details regarding the affected region.

The measure of pain severity, both during the initial history and subsequent follow-up visits, is purely by subjective patient reporting. A numerical rating scale (NRS) from 0–10 is frequently employed for convenience in doctor–patient discussions. Studies have shown it is less reliable than a Visual Analog Scale (VAS) [4]. A VAS measurement of pain is considered especially important in pediatric...
populations and recommended as preferred to NRS in both children and adults.

Chronic pain is usually defined as pain persisting for at least 3–6 months. However, chronic pain may be neuropathic, non-neuropathic (due to tissue injury or inflammation), due to an abnormality of pain processing or a mixture of all of these. In some cases, this is not obvious and for this reason a short screening questionnaire such as the Douleur Neuropathique (DN4) can help distinguish neuropathic pain in less than one minute. A DN4 score of 4 or greater suggests neuropathic pain with high sensitivity and specificity [5]. The DN4 questionnaire can be downloaded free of charge from http://dn4.ca/en/splash/.

Pain diagrams
A useful tool to rapidly and accurately localize sources of chronic pain and assist in the diagnosis of causes of neuropathic pain is a pain diagram (see Figure 1.1 and case vignettes). Patients are instructed to color or shade where they have pain, sometimes with simple word descriptors. The distribution of pain tends to be quite well defined in trigeminal neuralgia, PHN, stump pain, entrapment neuropathy, radiculopathy due to disc disease, and diabetic neuropathy. Unilateral whole body pain diagrams are consistent with post-stroke pain, while entire body pain is seen with fibromyalgia (which can have neuropathic features).

Table 1.1 The OPQRST approach to identifying important historical factors for the neuropathic pain patient.

| O: | Onset of the pain: sudden or gradual and when it first began, e.g. history of trauma or surgery |
| P: | Provoking and palliating factors: position or activities that makes the pain worse or better. Note: chronic neuropathic pain-specific symptoms of sensory avoidance such as specific clothing to avoid contact with skin, wearing dark glasses in the examination room, poor oral hygiene in patients with mouth pain, etc. may occur. |
| Q: | Quality of the pain: descriptors like sharp or dull, burning or cold, intermittent or constant. |
| R: | Region where the pain is primarily felt and Radiation, if any, where the pain may seem to spread toward. |
| S: | Severity of pain on a Visual Analog Scale of 0–10, where higher numbers represent greater pain |
| T: | Time duration of pain, especially how long the pain has been the way it presents now |

Table 1.2 The six Ss with respect to identifying important factors for recognition of chronic regional pain syndrome.

| S: | Skin color changes |
| S: | Swelling – intermittent or constant |
| S: | Sweating |
| S: | Spasms and dystonia |
| S: | Shaking such as focal tremors, myoclonus, and tics |
| S: | Self: affected area or limb neglected as foreign |

Past medical history
Patients’ medical conditions often lead directly to the diagnosis of neuropathic pain when it is generated in the peripheral nervous system (PNS). In patients with polyneuropathy, a history of diabetes, chemotherapy, or HIV points to pain due to each causative agent, respectively. In cases of a mononeuropathy, a history of trauma, surgery, or disc disease must be inquired about, with the latter presenting as a radiculopathy pain. Presenting similarly to a mononeuropathy is PHN, which involves both the peripheral and central nervous system, but nevertheless patients tend to initially complain of pain in a nerve root distribution. Past medical history also is
Critical in patients with pain due to direct injury to the CNS. A recent history of spinal cord injury, stroke, or multiple sclerosis generally provides the clinician with the cause of pain.

**Nutrition history**

Low levels of folate, vitamin B1, vitamin B6, and vitamin B12 can cause peripheral polyneuropathy [6]. Therefore, it is essential to record a family or personal history of possible malabsorption such as that seen in pernicious anemia, celiac disease and short bowel syndrome. Any of these conditions can affect vitamin B12 levels, as can the use of common medications such as proton pump inhibitors and certain antiepileptic drugs. A vegan diet can also be associated with low vitamin B12 levels due to reduced intake. Furthermore, the supplementation with vitamin B12 can reduce polyneuropathy-associated pain. It is important to ask about multivitamin use in general and B vitamin supplementation in particular.

**The constellation of chronic pain syndromes**

Chronic pain is more than simply pain that persists for a long duration of time. Instead, chronic pain is a syndrome with a constellation of symptoms including depression, insomnia, fatigue, and decreased functioning [7,8]. Each of these comorbidities contributes to a line of questioning that is as important as the pain itself. The comorbidity of depression is high in the chronic pain population [9], necessitating a discussion of mood in all chronic pain patients. In some cases, this can be examined using screening questionnaires while in other cases, a more detailed and directed history may be appropriate. The Beck Depression Inventory (BDI-II) is more detailed than shorter questionnaires such as the Patient Health Questionnaire (PHQ-9) [10]. Beyond questionnaires on history, patient mood is often inferred by looking for common themes of anger after an accident, feelings of abandonment by family members and lack of dreams and goals for the future. Anhedonia is especially common and a good gauge for quality of life. Suicidal ideation can occur in the context of chronic pain and patients’ response to questioning must be documented.

Pain can interfere with sleep in many ways, from increasing sleep latency to leading to frequent awakenings and decreased slow-wave sleep. It is important to ask specifically about the need for daytime naps, typical sleep environments, and the requirement for sleep aids. Insomnia is very prevalent in chronic pain populations [11]; it is also important to know the patient’s previous sleep habits prior to the onset of chronic pain. Determination of habits such as poor sleep hygiene (using the bedroom as a place of entertainment, eating, as well as sleeping) and afternoon napping can be important to help guide recommendations for proper sleep hygiene.

Patients with chronic pain for many years usually experience a gradual decline in overall and daily functioning. Patients may grow accustomed to this, so not all functional limitations may be reported unless specifically inquired for. Important questions include: “How long can you sit or stand?” “How far can you walk without resting?” “Do you have lifting restrictions?” In addition to directed questioning, questionnaires can determine a summary of how chronic pain is affecting overall functioning, such as with the short Pain Disability Index (PDI) questionnaire [12].

**Chronic pain coping mechanisms**

Finally, no history of patients with chronic pain would be complete without inquiring into both successful and unsuccessful coping mechanisms. The burden of living with pain and associated symptoms is heavy, leading many patients to eventually attempt methods such as alcohol, illicit substances, or unsuccessful coping mechanisms such as denial or escapism. Common coping strategies are listed in Table 1.3 [13].
Neurological exam for pain

The examination of a chronic pain patient should start with an appropriate and directed general examination including a neurological examination. The goal of the examination is to determine the presence of other pathological processes capable of causing pain (i.e. infection, inflammation, trauma) and determining which level of the neuroaxis may be involved, i.e. peripheral, spinal cord, or brainstem(brain). Most neuropathic pain disorders are characterized by stimulus-evoked positive sensory phenomena (i.e. pain or just the sense that the sensation is somehow increased when the skin is touched with a brush, a pinprick, or something cold or hot) and negative sensory phenomena (i.e. perception that the skin feels a brush, pinprick, or something hot or cold less or not at all when compared with similar normal areas). The clinician will need to identify the area of abnormality (hemibody loss suggesting spinal or brain cord localization vs. glove and stocking pattern suggesting a diffuse peripheral nerve condition as a cause for pain).

It will help to know which sensations are altered. A small-fiber neuropathy, a common cause of painful diabetic neuropathy, will present with pain and temperature sensory changes with preserved vibration and light touch in a glove and stocking distribution.

Definition of terms

A sensory threshold is the lowest point at which a stimulus begins to produce a sensation. This is relatively consistent but has some variability depending on age, sex, and body site tested. Pain tolerance is the greatest level of pain that any given person can tolerate at any given time and varies widely from person to person and in one person over time. A nerve fiber is an axon and the Schwann cell that ensheaths it. An unmyelinated fiber is one or more unmyelinated axons ensheathed by a single Schwann cell (i.e. a Remak bundle). There are three types of afferent fibers that originate in the periphery. The larger the fiber and the thicker the myelin coating the faster the nerve transmission, thus the fastest are the large myelinated A alpha and A beta fibers that conduct non-painful sensory information regarding light touch and vibration. When stimulated they also recruit inhibitory interneurons in the spinal cord which will inhibit nociceptive (painful) input at the same level. (That is why rubbing your elbow after you painfully bang your elbow helps.) Information is transmitted rapidly at 35–75 m/s. They use specialized nerve endings or sensory organs (e.g. Pacinian corpuscles). Changes in these large sensory (A alpha, A beta) fibers can be examined using nerve conduction studies (via electromyography studies) and somatosensory-evoked potentials (SSEP).

There are two fibers that transmit painful stimuli, the A delta and C fibers. A delta fibers are thinly myelinated small fibers that have faster (10–40 m/s) velocity conduction than do unmyelinated C fibers. If you burn your finger these fibers transmit the first sharp pain and are responsible for the withdrawal reflex. They transmit information about pinprick (punctate) and cold threshold sensation. The C fibers are unmyelinated fibers with slow conduction velocity (0.5–2 m/s), transmitting information about heat, heat pain, cold pain, and pinprick. These slower fibers are responsible for the second pain, for instance, after a finger burn – they transmit the deeper, more diffuse pain. These fibers are also responsible for the sensation of itch and paradoxically in the perception of pleasant touch. They are not associated with specialized nerve endings, although some have transient receptor potential vanilloid (TRPV) receptors and are sensitive to capsaicin. A small fiber neuropathy is a neuropathy in which C and A delta fibers are predominantly affected.

A recent study evaluating patients with painful diabetic neuropathy examined capsaicin application to identify functioning C fibers. If the capsaicin generated pain, then the C fibers were thought to still be functioning and thus susceptible to a topical application of clonidine.

Pain over a localized area may have multiple different contributing factors including pain from tissue damage or inflammation, from a neurologic injury or from abnormalities of pain processing. It is important to identify all of them and when possible, treat each individually. For example, a patient 3 years after a traumatic elbow injury may present with neuropathic pain (shooting and burning pain in the ulnar distribution), and sensory changes, such as tingling and numbness, affecting the ulnar nerve distribution. They may have nociceptive pain from secondary muscle weakness and arthritis, and a component of central sensitization which may manifest with symptoms such as fatigue, memory and sleep disturbance, spreading pain beyond the usual boundaries for each of these pathologies (deep aching pain in the shoulder and the entire forearm and wrist) with diffuse sensitivity to light touch and prolonged pain after palpation.
Pain behaviors

During the initiation of the exam, the health professional should provide attention to patient behaviors that are consistent with pain such as an antalgic gait or frequent positional changes during the interview. Patients with piriformis syndrome will often sit exclusively on one buttock whereas patients with coccygeal injuries will shift from one buttock to the other. Document bracing or splinting and if patients adopt particular protective positioning of painful body parts. They may wear extra clothing to prevent exacerbation from cold. Many patients with neuropathic pain and brush allodynia in their feet will wear tight boots to prevent their feet moving and brushing the inside of the shoe and wear socks to bed to prevent sheets from brushing over them at night. Patients with neuropathic facial pain related to the trigeminal nerve will sometimes relieve their pain by compressing the painful area with a thumb. There may be giveaway weakness from pain. Patients with CRPS may have difficulties with movement and may describe motor incoordination rather than weakness (some have described it as feeling the same as a limb that has just emerged from a cast) or tremors.

Body diagrams

A body diagram (Figure 1.2) is a picture of a body, front and back. All patients presenting with pain should be asked to complete one. The patient is asked to draw their pain. It is an opportunity for the patient to tell their story with pictures rather than words. When color is used, for instance red for burning pain, blue for numbness, green for tingling, yellow for deep ache and black for stabbing pain, the picture can also provide important clues to the diagnosis. (Imagine how blue, green, and red drawn from the knees distally to both feet could suggest a painful peripheral neuropathy.) The diagram also provides information on areas of potential sensory abnormalities and which areas may be normal, important information when performing a sensory examination.

Clinical vignette 1. The value of a pain diagram

A 58-year-old female developed a rash over her abdomen. After consulting with a friend, she determined that it may be shingles. Since she knew this should be treated promptly, she attended a walk-in clinic. The physician there diagnosed a staphylococcus infection and prescribed a topical antibiotic. The rash subsequently cleared and 4 weeks later, the patient developed right upper quadrant abdominal pain. She saw her family physician but never mentioned the (now healed) rash. The pain became severe and she was referred to the local emergency for assessment. She was admitted and underwent multiple investigations that included an ultrasound, CT scan, endoscopy, and colonoscopy. A lung CT was ordered and when that too was normal she was referred for a chronic pain evaluation. Her pain diagram suggested a dermatomal pattern (Figure 1.2). Sensory mapping confirmed a typical sloping dermatomal distribution of sensory changes and clinical quantitative sensory testing demonstrated positive phenomena (unexpected pain from pinprick and light touch) present at the distal end of the dermatome. Faint rash scars over her right upper abdomen accompanied by a normal thoracic MRI excluding other causes led to a diagnosis of post-herpetic neuralgia (PHN). Her pain diagram revealed changes over a single dermatome.

The history should be reviewed along with the pain diagram to formulate a list of possible diagnoses. Then, the neurological examination will supplement additional information that will help formulate the diagnosis or differential diagnosis.

During a recent presentation [18], Drs. Maija Haanpa and Michael Rowbotham reminded their audience of clinicians that pain resulting from CNS lesions may be very poorly localized with vague boundaries, whereas peripheral nerve lesions generally produce a deficit that can be mapped out quite precisely. Trying to determine the precise boundary of a dermatome (defined as the area of skin supplied by sensory neurons arising from a spinal nerve ganglia) underemphasizes how much overlap exists between two ganglia. A wide differential diagnosis should be formulated along with keeping an open mind to different possible conditions when evaluating the sensory maps. The health professional should not become excessively concerned if the lines do not perfectly resemble published diagrams of an innervated territory. It is known, for example, that patients with PHN may sometimes have bordering dermatomes affected, and there is great variance in actual nerve distributions between individuals.
Section 1: The Clinical Presentation of Neuropathic Pain

Please color the areas where you experience pain. Use one of these five coloring pens to shade the specific type of pain that you are experiencing. Then circle with a pen all areas of pain and starting with the worst, number the areas in order of severity.

Red - burning
Green - tingling
Blue - numbness
Yellow - Yellow
Black - Black

If you have other pain sensations name them here and color as black or yellow

Name _____________________________________ Date ______________________

Figure 1.2 The pain diagram for the patient in case vignette #1 demonstrates a right T9 dermatomal pattern consistent with that of shingles and later post-herpetic neuralgia. This figure is presented in color in the color plate section.

Clinical vignette 2. The value of additional neurological examination

An elderly patient with mild dementia presented with left arm pain. The nursing notes documented pain when they touched the forearm but noted that her upper arm was normal. It was also recorded that she had stopped using the left arm. An X-ray of the left arm was reported as normal. When assessed, she was asked about her sore arm, and surprisingly, she presented her right arm. Her right arm was normal to examination. Attempts to examine the left arm elicited cries of pain, leading to her withdrawing the left arm. After some cautious inspection, there were not any obvious abnormalities aside from tenderness over her entire left forearm. When she was asked again to indicate her painful arm, again she presented her right arm. The examiner held onto her right arm and she was asked to show her other arm. She looked confused and didn’t appear to “see” her other arm. This additional information helped to identify the neurological level of abnormality, since the picture of visuospatial neglect and diffuse pain in the same arm were suspicious for central mechanisms. An MRI scan (Figure 1.3) confirmed a right middle cerebral artery ischemic stroke.
Case vignette 3. Sensory mapping

Mapping of sensation can be performed with a brush or an unwound paperclip. The latter tool provides a stimulus of greater amplitude than a brush but less than that of a pin. It can be quickly dragged from normal skin to the described area of changes and will stimulate both small and large sensory fibers. An effective method is to start by establishing an area of normal touch sensation and then drag the paperclip towards the abnormal area in a radial pattern, establishing the boundaries of the sensory change.

Concurrently, a pen can be used to draw reported sensory changes, easing documentation. Some clinicians will use photography to document the markings for later comparison and to aid in information transfer to other interested parties.

The sensory map illustrated (Figure 1.4) is for a patient referred for assessment of low back and leg pain with sensory changes over the right thigh, thought to represent a right L2 radiculopathy. However, the pain diagram suggested a lesion of the lateral femoral cutaneous nerve of the thigh (meralgia paresthetica) due to the appearance of an oval patch at the anterolateral thigh inconsistent with that of the L2 dermatome. Sensory abnormalities were limited to that specific area, while further testing revealed that light brush sensation was reduced (brush hypoesthesia), pinprick was more painful than anticipated (pinprick hyperalgesia), and temperature sensations were described as delayed but normal in intensity. The remainder of the neurological exam confirmed a normal motor exam, normoreflexia (including at the left knee jerk) and unremarkable straight leg testing.

An ultrasound-guided block of the lateral femoral cutaneous nerve abolished the pain, helping to confirm the diagnosis. The pain subsequently resolved spontaneously. In this case, conservative measures included advising the patient to avoid prolonged crouching or kneeling, avoidance of tight belts or clothing and weight loss to limit compression of the lateral femoral cutaneous nerve of the thigh.

Once the area of sensory abnormalities has been mapped out, clinical quantitative sensory testing can be used to determine the areas of sensory normality and the presence of positive and negative sensory phenomena as an aid to diagnosis (to diagnose a specific mononeuropathy or dermatomal involvement in radiculopathy, for example) and to determine...
the extent of a known abnormality (how proximal does the sensory polyneuropathy affecting a patient’s feet extend?). This approach can assist with documenting progression or recession or a lesion-inducing sensory change. Also, if a topical treatment was planned, the clinician may not want to attempt this should the affected area be significantly large.

Bedside method for quantitative sensory pain testing

Improving outcomes involves understanding disease risk factors and mechanisms, determining which are relevant, developing accurate and standardized measurements, and then developing and evaluating treatment interventions that address as many of the relevant contributing factors as possible. The evolution of assessment and treatment of hypertension is a model of this paradigm. One of the earliest developments that facilitated this evolution was the ability to accurately measure blood pressure. It began with the introduction of a standardized tool, the sphygmomanometer blood pressure cuff. Over time it became clear that the method of using this tool (seating the patient for 5 minutes before measuring and ensuring proper positioning of the patient’s arm) was as important as the tool itself. Accurate and reproducible blood pressure measurement then allowed the development of standardized normal values. From there, deviations from normal could be quantified and tracked over time and as research developed new therapeutic interventions measuring treatment success became quantifiable. The ability to reliably reproduce these measurements was a crucial link between understanding disease mechanisms and improving treatment outcomes.

In chronic pain states, the degree of peripheral damage or inflammation does not correlate well with pain severity. This initially led to a focus on the psychosocial aspects of pain to explain a discrepancy but over the last few years research has also focused on identifying the many different biological mechanisms that may contribute to chronic pain. This information is changing how chronic pain is characterized but documentation of sensory abnormalities will help confirm or deny the presence of neuropathic pain [20]. The article stated “A careful bedside examination of somatosensory functions is recommended, including touch/vibration, cold, warmth and pain sensibility” for patients presenting with possible neuropathic pain. Sensory testing alone cannot determine the neuroaxial level of pathology, but documentation of sensory abnormalities will help to confirm or deny the presence of neuropathic pain.” This has been most useful in patients presenting with possible PHN, painful polyneuropathy, complex regional pain syndrome, spinal cord injury pain, and post-stroke pain.

We will describe a method that was initially developed by Dr. Misha Backonja [21]. Quantitative sensory testing (QST), as defined by the Neuropathic Pain Research Consortium (NPRC) [22], does not intend to determine pain thresholds, but instead is designed to measure subjective experience (loss or gain of sensation) in response to particular thermal, mechanical, or vibratory stimuli. It also seeks to provide indirect information used to evaluate underlying sensory function abnormalities using only small, portable tools and with less time requirement than protocols developed by the German Neuropathic Research Network [23, 24]. Both protocols are psychophysical methods utilizing specific physical stimuli (pinprick, touch, vibration, heat, cold) to activate sensory receptors. Both protocols also require active participation and directed attention on behalf of the patient. Together, the examiner and the patient require instruction and training in the testing procedures of QST.

The clinical role of bedside pain sensory testing in the diagnosis of neuropathic pain

The Neuropathic Pain Special Interest Group of the International Association for the Study of Pain recently published guidelines on the assessment of neuropathic pain [20]. The article stated “A careful bedside examination of somatosensory functions is recommended, including touch/vibration, cold, warmth and pain sensibility” for patients presenting with possible neuropathic pain. Sensory testing alone cannot determine the neuroaxial level of pathology, but documentation of sensory abnormalities will help to confirm or deny the presence of neuropathic pain.” This has been most useful in patients presenting with possible PHN, painful polyneuropathy, complex regional pain syndrome, spinal cord injury pain, and post-stroke pain.

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The following standard set of verbal instructions and procedures is intended to guide the clinical examination based on commonly available equipment. Feedback to the authors is encouraged to improve the face validity of the procedures. Begin by using the pain diagram with pain descriptors to identify affected areas to direct the physical exam (see Figure 1.4). Establish a control site where the patient does not describe any sensory abnormalities or pain and briefly examine to confirm the expected findings. A site that is either contralateral or diagonal to the most painful areas (e.g. if the affected area is an arm, use the opposite arm if unaffected or the opposite leg as normal) is best. Test only in the area of worst pain. If there are several sites that are painful, limit testing to two areas. If there are areas where sensation seems reduced or lost and others where there is hypersensitivity, ensure that you test at least one area that represents sensory deficit and one area that represents hypersensitivity. Begin a basic screening examination by testing touch (to evaluate the large A beta fibers) and then pinprick (to evaluate the small A delta fibers) to avoid sensitizing the skin. If these are normal, then vibration (which is also A beta) and temperature sensation (which is a mix of A delta and C fibers depending on the temperature tested) should be tested before you declare the sensory examination is normal. The sensory findings, using tools as outlined in Table 1.4, should be documented regarding the reported responses of the patient as the same, increased, or decreased, as compared with the normal area. Have patients rate the change on a scale of 0–100.

For instance, if the patient rates the stimulus to brush in the right hand as normal but reduced in the left hand, brush the normal right hand again and say, “If this is worth a dollar, (then brush the abnormal left hand) how much is this worth?” Patient responses “I feel it less” can then be quantified as I would rate that as 10 cents or 97 cents giving the clinician a much cleared idea of the sensory experience of the patient. Likewise, a patient who reports a pinprick stimulus as 30/100 in the normal side and 80/100 on the affected side is also providing more information than “I feel it more.” If multiple modalities are assessed, then try to perform them in this order to avoid sensitizing the skin: light touch, vibration, cool and warm, pinprick, cold and hot, pressure, and then summation testing (Table 1.5). In order to test these different sensory modalities, there are different options for the performance of bedside QST [21,22,25].

After concentrating upon the sensory examination, it is important to complete the neurological examination with assessments of cranial nerves, motor assessment, deep tendon reflexes, muscle tone, coordination, gait, and specific testing such as with straight leg raising, Tinel’s test, or Adson’s test for thoracic outlet syndrome.

It is important to identify associated abnormalities. These include temperature differences between affected and unaffected areas which can be documented with a laser temperature probe. Swelling, such as with neurogenic edema associated with CRPS, should be documented. In some cases, this may be transient and intermittent – having the patient provide a photograph may be valuable, if necessary. Look for skin lesions, i.e. scarring from varicella zoster, foot ulcers with diabetic neuropathy, and color changes, such as with mottling or with erythromelalgia where erythematous skin flushing may occur. There may be differences in sweating in an affected body part or trophic changes such as with loss of hair, thinning skin, cracked dry skin, or altered nails. Secondary changes associated with chronic denervation, such as with Charcot neuropathic foot destruction with necrotic arthropathy and chronic ulcers on the plantar surface, should also be documented.

The role of neural plasticity

Injury to the nervous system results in maladaptive plasticity which can alter function at multiple levels of the somatosensory system including the peripheral
nerves (where they can produce spontaneous discharges and alter nerve transduction to create, for instance, cold allodynia), the dorsal horn (where changes enhance transmission of nociceptive information), and the cortex [18]. Peripheral nerve injury can lead to increased neuronal activity throughout the central nervous system, resulting in increased responses to noxious and non-noxious stimuli. Sunburned shoulders are an example of normal, adaptive central sensitization not due to direct nerve injury, and anyone experiencing this will recall features of warm and pressure allodynia as they stood in a shower, for example. Under normal circumstances, this is a temporary phenomenon that may resolve as tissues heal. In some circumstances, however, either affected tissues fail to heal or the mechanisms evolve and, despite tissue healing, neuronal hyperexcitability persists, thus pain is no longer coupled to ongoing tissue damage. Documentation of this phenomenon is clinically important to provide (to patients, their family, insurance companies, and the courts), in the differential diagnosis, a physiological basis that may explain some of their symptoms. Diagnosis of peripheral sensitization relies on a history that has features consistent with neuropathic pain. Spontaneous pain may be present. Research has demonstrated some clinical findings that characterize plasticity at different levels or by different mechanisms but it can be difficult to impossible to separate peripheral from central mechanisms. In some cases this is because both mechanisms contribute to a particular clinical finding. For example, abnormally increased pain following a noxious cold stimulus (cold hyperalgesia) is mediated by peripheral sensitization in addition to reduced inhibition and central sensitization. In other cases peripheral input may be driving central sensitization so both will be present. Clinically, especially in the setting of a brief bedside examination, it can be difficult to document findings that would allow distinction between these two mechanisms. The bedside examination should focus on simply documenting signs consistent with the presence of sensitization. Interpretation of these signs must take into account caveats described at the end of this chapter.

<table>
<thead>
<tr>
<th>Modality</th>
<th>Axon</th>
<th>Increased and not painful</th>
<th>Abnormally painful (0 to 10)</th>
<th>Summation and after pain</th>
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<tr>
<td>Light brush</td>
<td>A beta and some C</td>
<td>Dysesthesia</td>
<td>Allodynia CS</td>
<td>CS</td>
</tr>
<tr>
<td>Vibration</td>
<td>A beta</td>
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<td>Cool</td>
<td>A delta</td>
<td>Dysesthesia</td>
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<td>Warm</td>
<td>C fiber</td>
<td>Dysesthesia</td>
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<tr>
<td>Punctate</td>
<td>A beta and some A delta</td>
<td>Dysesthesia</td>
<td>Allodynia CS</td>
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<td>Pin prick</td>
<td>A delta</td>
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<td>Cold pain</td>
<td>C/some A delta</td>
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<td>Deep pressure</td>
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CD, central disinhibition; CS, central sensitization; PS, peripheral sensitization. Loss or reduction in sensation is generally due to sensory pathway damage or degeneration; SPSA, superficial peripheral sensitization allodynia; SPD, superficial peripheral dysesthesia.