Proving the Existence of Chronic Pain

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§ 5.1 Introduction

Proving the existence of chronic pain is a challenging task, given the wide variety of patients who present clinically with chronic pain, the large volume of medical literature, and the controversies among professionals. The more that is learned about chronic pain, the more the controversy seems to intensify. This chapter interprets the subject based on published information and the author's professional experience, with the understanding that there are other facts and viewpoints. The objectives of this chapter are: to describe the methods of identifying and treating patients who present with chronic pain; to offer insight into clinical methods of assessing chronic pain behavior; and to discuss recent research findings on biochemical factors in chronic pain.

§ 5.2 Definitions of Pain

Pain can be generally described as "an unpleasant sensory and emotional experience."[1] Webster's Dictionary defines pain as "a basic bodily sensation induced by a noxious stimulus, received by naked nerve endings, characterized by physical discomfort (as pricking, throbbing, or aching), and typically leading to evasive action."[2]

Pain is perceived differently by each person. A stimulus that greatly affects one person may provoke no pain response in another. Although pain is experienced differently, its common denominator is physical and mental suffering. Moreover, because pain typically motivates people to seek treatment, the success of treatment is usually determined by the degree of pain relief, whether it is actual or imagined.

When considering pain and its interrelated determinants, it is important for health care providers to treat the patient, not merely the symptoms. "The minutes . . . spent to operate on a patient, not a spine, may save years of coping with the human wreckage caused by ill-considered surgery on the lumbar discs."[3] As British medical pioneer Sir William Osier stated, "It is not nearly as important what illness a patient has, as what patient has the illness."[4]

§ 5.3 Anatomy and Physiology of Pain Reception

Many anatomical pathways relay information from the periphery (skin) to the higher center (brain). Lesions in or around many pathways yield particular findings, which health care providers can detect by neurological examination to identify the anatomical region that has been injured. Unfortunately, not all neurological pathways or regions have examination procedures that are specific to one area, which can confuse the process of making a diagnosis. This chapter limits its scope to the neurological pathways or regions that can be identified by a routine neurological examination.

Pain typically begins at the skin, where pain receptors are very numerous (particularly over the head, hands, and feet). This concentration can be represented by the homuncular patterns on the sensory cerebral cortex of the brain. To experience any sensation, the following structures must be intact and functioning:

1. Sensory receptors
2. Sensory-conveying organs
3. Sense-interpreting centers in the brain
4. Associative memory centers in the brain.

For example, when the skin is cut by a knife, the pain is initially sharp and the perception of the pain depends on the integrity of the surrounding tissue. For example, an area on the skin previously scarred by a burn loses the free nerve endings and their nociceptive (pain-relaying) function,

TABLE 5–1. NEUROLOGICAL FUNCTIONS FOLLOWING INJURY

Anatomical Pathway or Structure	Function
1. Pain receptor (nociceptor)	Receives the initial harmful stimuli, such as a cut.
2. Dorsal horn (spinal cord)	Receives incoming information from injured tissue via a peripheral nerve.
3. Ascending spinal cord tract (spinothalamic tract)	Carries information from the cord to the sensory cortex of the brain.
4. Sensory cortex of the brain	Receives and interprets incoming information.
5. Motor Cortex of the brain	Receives messages from the sensory cortex and sends information down the corticospinal tract.
6. Descending spinal cord tract (Corticospinal tract)	Sends information from motor cortex to the spinal cord level where the information originated.
7. Muscle	Receives motor orders from the brain via the peripheral nerves, causing the muscle to contract or splint to protect the area.

TABLE 5–2. TESTS FOR ASSESSSING FUNCTION OF PARTS OF THE NERVOUS SYSTEM

1. Pain receptor	Sensibility testing: Pinwheel, light touch, deep pressure, vibration, Two-point differentiation, hot, cold, smell, sight, hearing, taste
2. Peripheral nerve	Sensory: Same as #1 plus nerve conduction velocity test (NCV) Motor: Deep tendon reflexes, muscle strength tests, measurement of circumference
3. Dorsal horn (spinal cord)	Same as #1 and #2 sensory tests; cannot be isolated
4. Ascending spinal cord tract	Spinothalamic tracts: pain (lateral ST), 2-point (ventral ST) Dorsal columns: Vibration, position sense, posture and gait
5. Sensory cortext of the brain	Electroencephalogram, somatosensory evoked potential (SSEP)
5. Motor cortex of the brain	Same as #4 and #6
6. Descending spinal cord tract (corticospinal tract)	Deep tendon reflex exaggeration, clonus, spastic paralysis, pathological reflexes
7. Muscle	Range of motion (active, passive, and resisted), measurement of circumference, electromyography

because the neuroreceptors (nociceptors) that detect pain have been damaged or destroyed by the bum and subsequent scar tissue formation. Separate types of receptors detect touch, temperature, pain, vibration, smell, taste, sight, and hearing, and the ability of the brain to receive incoming sensory information from one of these receptors depends on the integrity of the receptor and the peripheral nerve. For the purposes of this chapter, it will be assumed that the receptors and peripheral nerves that carry received information to and from the spinal cord and brain are intact. For pain to be perceived, the structures described in **Table 5–1** must be intact.

The nerve impulse enters the sensory area of the spinal cord called the dorsal horn. Some information travels up the spinal cord via the spinothalamic or lateral tract (which propagates pain signals) to the sensory cortex of the cerebrum. Incoming signals travel directly to the ventral or motor horn, and outgoing signals are received by muscles; causing a movement or retraction of the limb away from the painful stimulus. This protective reflex reaction (the quick withdrawal from the harmful object or noxious stimuli) minimizes injury.

This sequence of events is a simplification of the order of neurological functions that occurs in response to an injury. This sequence can repeat, establishing a cycle of pain until something interrupts it, such as an ice pack, anti-inflammatory agent, or another form of treatment.

§ 5.4 The Pain Cycle

The pain cycle is perpetuated by failure to adequately apply the treatment principles suggested by the mnemonic PRICE; protect rest, ice, compress. and elevate the injured part. Because healing (analogous to the formation of a scab over a cut) begins immediately after an injury, the PRICE principles should be implemented in the first few days post-injury. Failure to do so delays healing, results in poorly organized scar tissue, and may lead to chronic, permanent lesions, especially if the injury occurs in a body region that is highly functional, such as the neck or cervical spine. Ongoing pain often results in pain behavior, which is manifested by fear, avoidant behavior, catastrophization, and loss of coping strategies.[5] Although the pain cycle is well accepted in many manual therapies, its validity is unproven.[6] However, the literature indicates that intense and/or

prolonged pain can lead to both psychologic and physiologic consequences, such as abnormal illness behavior and dorsal horn sensitization.[7] Evoked potentials of chronic pain patients show lower pain thresholds and higher than normal evoked responses than those found in a normal comparison population.[8] Similarly, magnetoencephalographic studies in chronic back pain patients reveal a higher than normal pain-evoked magnetic field.[9] Either or both of these responses are thought to be at the heart of the transition from acute to chronic pain.[10]

Pain has been reported for as long as records have been kept. For example, Plato described pain as the effect of disturbed elements of the air, earth, fire, and water on the soul; Aristotle described the heart as the pain processing center.[11] Even though the central nervous system was first described in 300 B.C., it was not until the 1800s that a rational explanation of pain was offered. Melzack and Wall's 1965 *pain gate theory* remains a landmark study in the understanding of pain and its relationship to the nervous system.[12] Essentially, the pain gate theory asserts that various forms of stimulation of faster nerve fibers (Type A nerve fibers) create a gate in the dorsal horn cells of the substantia gelatinosa and inhibit the nerve transmission from the slower sensory nerve fibers (Type C) that transmit pain signals to the brain. Although the validity of this theory has been questioned, the discovery of the pain gate theory was pivotal in reaching the current level of understanding of the neural pathways and mechanisms of pain.

Just as a wide, gaping cut heals slower than a thin, well-approximated cut, a third-degree or severe sprain (tear) of a ligament takes longer to heal than a mild sprain or pulled muscle. Hence, the intensity of treatment in the initial stage of healing depends on the amount of damaged tissue. For a successful treatment outcome, one needs to stay within the physiological limits of the injured tissue.

§ 5.5 Clinical Examination

Table 5–2 lists examination procedures unique to addressing different parts of the anatomy. Keep in mind that no test is 100 percent sensitive at detecting true positives or 100 percent specific at detecting true negatives. Unless a great deal of damage has occurred, the test used to evaluate a structure's function may reveal no sign of abnormality Therefore, an examination that fails to reveal adequate information about a damaged region may be due to the test's inadequate sensitivity or specificity or to the examiner's lack of skill in applying and interpreting the test.

The nervous system can be divided into two broad categories: the upper motor neuron (UMN) and the lower motor neuron (LMN). The LMN essentially includes all neurological structures from the anterior horn cells (motor horn) to the muscle, and the UMN includes the brain and spinal cord. A diagnostic approach using various physical examination tests is shown in **Table 5–3**.

§ 5.6 Subjective and Objective Pain

Pain can be classified into two broad headings: objective and subjective.[13] Objective pain follows anatomical pathways and arises from some foreign agent or known condition Objective pain can be further classified into peripheral and central objective pain. *Peripheral objective pain* has a known cause outside the central nervous system, and *central objective pain* has no peripheral cause. Subjective pain in contrast does not follow any known anatomical path and has no organic cause. Nonorganic pain is a hallmark of chronic pain behavior and is the result of the patient's perception that pain exists; even though it cannot be substantiated by objective findings or tests. This is not the same as malingering; which is a conscious attempt or preconceived intention to mimic a disease or disorder. For a detailed discussion of the problem of identifying malingering; see **Chapter 6**.

The discussion of pain becomes significantly more complex when factors other than anatomy are considered. For example; the perception or interpretation of pain differs significantly between genders; ages; levels of formal education varying fatigue levels; and other factors. **Table 5–4** describes some of the factors that influence pain thresholds.

§ 5.7 Fibromyalgia

Recent research on fibromyalgia is shedding new light on our understanding of chronic pain.[15] Fibromyalgia is a common condition characterized by diffuse musculoskeletal pain and fatigue. This syndrome is defined by the presence of pain on at least 11 of 18 tender points when palpated with approximately four kilograms of pressure. These tender points are located throughout the body.[16] See **Figure 5–1**. Other symptoms include headache, ocular and vestibular complaints, paresthesias, esophageal dysmotility, allergic

TABLE 5–3. TESTS FOR ASSESSSING FUNCTION OF PARTS OF THE NERVOUS SYSTEM

Examination	Upper Motor Neuron (Central)	Lower Motor Neuron (Peripheral)
Reflexes (e.g., deep tendon reflex)	Increased	Decreased
Pathological reflexes (e.g., Babinsky's sign)	Present	Absent
Paralysis	Spastic	Flaccid
Clonus	Present	Absent
Degeneration	Absent	Present

TABLE 5–4. FACTORS AFFECTING PAIN THRESHOLD[14]

Factor	Effect on Pain Threshold
Age	Rises with age.
Anxiety	Lowers with fear of pain, domestic distress, and other anxiety states.
Distraction	Rises with external distraction (e.g., noise)
Fatigue	Mental fatigue often lowers the threshold. Physical fatigue does not appear to influence pain threshold.
Laterality	Lowers on dominant side for physical pain. Reports differ whether psychic pain is increased on the non-dominant side.
Lifestyle	Lowers in patients confined to bed or home with little to occupy themselves.
Pain elsewhere	Hippocrates and recent investigators alike note that when pain is produced simultaneously in two places, the lesser pain tends to be obliterated by the greater pain.
Pathology	Lowers if tissue damage is present at the site of measurement. Such a site should not be used to test general pain threshold.
Personality	Lower with a history of severe, prolonged childhood pain (e.g., child abuse).
Placebos and direct suggestion	Increases.
Race	Lower among blacks, Semitics, and Mediterraneans. Higher among East Indians and Northern Europeans.
Gender	Lower for electrical stimuli in women. Reports conflict with heat and mechanical pressure.
Skin temperature	Lowers when skin temperature is warmed.
Miscellaneous conditions	Rises with carbon dioxide retention, impaired judgment, peripheral vasoconstriction, and respiratory depression.

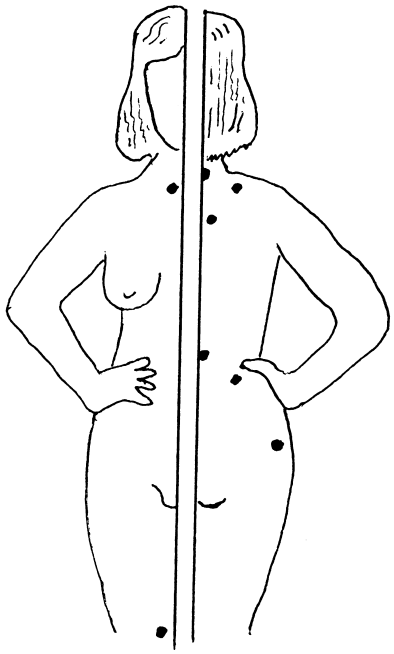

Figure 5–1 Pain-sensitive points. Reprinted from F. Wolfe et al., *American College of Rheumatology 1990 Criteria for the Classification of Fibromyalgia: Report of the Multi-Center Criteria Committee*, 33 Arthritis & Rheum. 162 (1990).

symptoms, irritable bowel syndrome, genitourinary symptoms, and affective disorders. Research has identified objective biochemical, hormonal, and neurotransmitter abnormalities in patients with fibromyalgia.

In this author's experience, diffuse tenderness is more common than tenderness restricted to only 11 of 18 tender points. Normal (nonfibromyalgic) people have an average of three positive tender points.[17] Women are more likely than men to be afflicted with fibromyalgia, and compared to men, women have a lower pain threshold. The mean age of fibromyalgia patients is 55 to 60, and pain tends to increase linearly with age in both men and women.[18] Clinical features include:

Pain and tender points exist.

Pain tends to migrate and wax and wane.

Morning stiffness is common.

Pain is increased by weather changes, physical activity, stress, and menstruation.

No objective evidence of swelling or synovitis is apparent on examination.

An overall lower pain threshold or increased pain sensitivity is noted throughout the body, not only in the periphery.

Increase in visceral or referred pain (for example, irritable bowel syndrome).[19]

Nociception (pain perception) is also influenced by age, gender, aerobic fitness, poor sleep, and depression.[20] Fatigue is a common complaint, but it is not required for a

diagnosis. A careful sleep history must be obtained, especially in men, because data suggests that men with fibromyalgia may have an underlying sleep apnea,[21] which may be a stage 4 deep sleep disorder.[22]

Neurological symptoms include a higher incidence of both tension and migraine headaches. Numbness and tingling sensations are also common and often occur randomly on the body. (Eighty-four percent of people with fibromyalgia complain of these paresthesias.[23]) Hearing and ocular vestibular abnormalities, such as lower tolerance to sound, exaggerated or slow eye movements, and low frequency sensorineural hearing loss have been noted.[24] Cognitive complaints include difficulty in concentration and short-term memory impairment.

Standard neurological examination, nerve conduction tests, and diagnostic imaging are typically normal; but more subtle tests, such as evoked responses and functional assessments, may show abnormalities. Because expensive tests are usually not helpful in assessing fibromyalgia, a neurological assessment should be given only when objective abnormalities are noted on clinical examination.

Allergic symptoms are common in the fibromyalgic population. A wide range of symptoms to adverse drug and environmental stimuli has been noted, sometimes meeting criteria for multiple chemical sensitivity syndrome. For a detailed discussion of this syndrome, see **Chapter 6**. In addition, there is a higher than expected incidence of rhinitis, nasal congestion, and Sower respiratory symptoms.[25] It is unlikely that these are true allergic reactions; instead, these symptoms may be the result of central nervous system activation seen in fibromyalgia.

Cardiac, pulmonary, and gastrointestinal symptoms are also common in patients with fibromyalgia. Functional disorders of the visceral organs that have been linked to fibromyalgia include recurrent noncardiac chest pain, heartburn, heart palpitation, and irritable bowel syndrome. Recent studies of randomly selected patients with fibromyalgia have detected objective abnormalities in visceral organs, including a 75 percent incidence of echocardiographic evidence of mitral valve prolapse.[26] Forty to 70 percent had esophageal dysmotility and diminished static inspiratory and expiratory pressures on pulmonary function tests.[27] These studies suggest that the symptoms associated with fibromyalgia have a physiological basis that is probably centrally mediated through the nervous system.

Genitourinary symptoms noted in fibromyalgia patients include higher than normal incidences of dysmenorrhea and frequency and urgency of urination.[28]

Fibromyalgia patients have a higher incidence of affective and other psychiatric disorders, with 20 percent reporting current depression and 50 percent reporting at least one major bout of depression. Significant controversy surrounds the presence of psychiatric conditions and concurrent physical symptoms. Those who consider fibromyalgia to be a psychiatric condition believe that the symptoms result from somatization. Others think that psychological problems are primarily the consequences of the chronic pain, fatigue, and disability associated with fibromyalgia. This debate becomes less important when one considers psychiatric disturbances in the same perspective as physical symptoms. A common neurotransmitter or hormonal imbalance is responsible for both and occur more frequently among patients with fibromyalgia.

Because its clinical manifestations can vary considerably, establishing a diagnosis of fibromyalgia is difficult. Fibromyalgia can be broadly classified into two types: Primary fibromyalgia is isolated and not associated with other disorders, whereas secondary fibromyalgia is associated with other disorders. A triggering event can usually be identified, such as a physical or emotional trauma or an infection. A premorbid history often suggests a high lifetime incidence of related conditions and links to major events in childhood. In addition, a family tendency toward fibromyalgia and associated syndromes supports a possible genetic link to the condition.[29] A laboratory screening might include a complete blood count, differential white blood count, chemistry and thyroid panels, and erythrocyte sedimentation rate (ESR). If positive results are noted on the ESR, further tests may include a rheumatoid panel, including antinuclear antibody and Lyme disease tests.

Magnetic resonance imaging (MRI) may be useful, especially if a co-existing condition is strongly suspected. However, MRI commonly gives false positive results, because so much anatomy is displayed that it can be difficult to determine clinically significant findings from those that are insignificant. A patient's syndrome should not be attributed to a co-existing disorder because fibromyalgia may still be a major factor despite any abnormalities found on tests. If fibromyalgia is responsible for the majority of the symptoms, surgery may be a highly inappropriate form of treatment

§ 5.8 —Pathophysiology of Fibromyalgia

Researchers have identified several hormonal, neurotransmitter, and biochemical abnormalities in fibromyalgia patients, including the following:

Insulin-like growth factor (IGF). Patients with fibromyalgia typically have low levels of IGF-I/Somatomedin C, a hormone produced by the liver primarily in response to growth hormone. This test has good sensitivity and specificity in detecting fibromyalgia-30

Substance P. Substance P is a neuropeptide stored in the secretory granules of sensory nerves and released by axonal stimulation. Cerebral spinal fluid (CSF) levels of substance P appear quite high in patients with fibromyalgia.

Serotonin. Levels of serotonin and its precursor tryptophan are low in people with fibromyalgia.[31] This finding is intriguing, because migraine headaches, irritable

bowel syndrome, and other associated affective disorders are known or thought to be due to low levels of serotonin. This biochemical explanation could cause both symptoms of fibromyalgia and organic symptoms.

Magnesium. Low tissue levels of magnesium have been found in fibromyalgia patients, even in those with normal serum or blood levels.[32]

Hormonal abnormalities. Abnormalities of the hypothalamic-pituitary-adrenal axis are seen in patients with fibromyalgia. These abnormalities are probably responsible for the low IGFs noted, but they are not unique to fibromyalgia so their significance is uncertain.[33] Therefore, hormone tests should not be used for screening purposes, but only as a secondary or tertiary level of testing. These tests have about an 80 percent level of sensitivity and specificity, which is comparable to the sensitivity and specificity of the commonly used test for rheumatoid factor.

§ 5.9 — Management of Fibromyalgia

Because of its often multiple co-existing conditions, management of fibromyalgia must take into account many factors, including:

Education. Patients need to be reassured that death is not a consequence of fibromyalgia. Patients need to take an active role in their treatment and not rely on passive therapies alone.

Behavior modification. Patients need to get adequate sleep. They should avoid consumption of caffeine and alcohol near bedtime since deep sleep can be impaired. Emotional stress should also be minimized.

Drug therapies. Tricyclic compounds, such as amitryptyline (Elavil) and cyclobenzaprine (Flexeril), may be prescribed» Patients should be warned that nightmares and hung-over feelings are initially common, but will subside once the dosage is regulated. Improvement from tricyclics may not be noted for four to six weeks. Nonsteroidal anti-inflammatory drugs can be used, but they should be discontinued if little benefit is noted. Selective serotonin uptake inhibitors (Prozac, Zoloft, Paxil, and Effexor) may be of some benefit in selected patients, especially if depression is also present. Narcotics and benzodiazepines should be avoided because of the addictive potential for fibromyalgic patients as well as the detrimental effect these drugs have on deep sleep. Zolpidem (Ambien) may be the most appropriate hypnotic agent because it impairs deep sleep less than other narcotics.

Exercise. Low-impact and water aerobic exercises, active stretching, and stationary bicycles, rowing machines, and similar machines are helpful to the fibromyalgia patient. Exercise should initially be restricted so only mild tenderness occurs, and gradually increased to more strenuous levels. A significant benefit of exercise is that it actively involves the patient, in contrast to the prolonged passive care in chiropractic and physical therapy.

Other modalities. Biofeedback, massage therapy, spinal manipulation, acupuncture. and injection of trigger points with topical anesthesia are all known to benefit selected patients.

Although fibromyalgia has no known cure, prompt recognition and proper management can substantially alleviate its symptoms and allow the patient to engage in a less chronic learned helplessness behavior.

§ 5.10 Post-Herpetic Neuralgia

Post-herpetic neuralgia (PHN) is another example of a condition that can result in chronic pain. It is an almost purely neuropathic pain syndrome, and its unique mechanism of causation has been clearly identified. PHN is a common complication of herpes zoster (HZ) and is frequently resistant to treatment. It is the leading cause of intractable, debilitating pain among the elderly and a major cause of suicide in chronic pain patients over age 70.[34] PHN has variably been defined as pain persisting more than one to six months after resolution of the vesicular eruption of HZ.[35] The time frame of three months is commonly accepted.

HZ begins with the reactivation of latent varicella-zoster viral nucleic acid in dorsal root ganglia. Viral particles are transported via sensory nerves to the skin in the corresponding dermatome, where the cutaneous nerves undergo an inflammatory process. Adjacent soft tissues hemorrhage and can become necrotic, manifesting as a characteristic vesicular eruption. Pain is probably derived from destruction of one or more sites, which may include the dorsal root ganglia, peripheral nerves, nervi nervorum, and soft tissue.

The incidence and duration of PHN increases with age. Nearly half of PHN patients are over 60. PHN may afflict as many as 160,000 individuals, with chronic sufferers accounting for 10 to 15 percent. PHN gradually improves over time but may persist for years in the elderly. Pain duration greater than one year is often seen in the age range of 80 to 89. Improvement may occur even in cases of longer duration, but longer duration tends to result in a poorer outcome. The outcome is not otherwise affected by age, gender or region of involvement.[36]

Characteristically, there is a history for a vesicular and painful cutaneous rash roughly in a dermatomal distribution. The most common sites are the mid-thoracic dermatomes (torso) and the ophthalmic division of the trigeminal nerve (near the eye). The persisting pain of PHN may be of three types: (1) constant deep aching, (2) spontaneous paroxysmal shooting or “electric” pain, and (3) allodynia or touch-evoked, sharp, burning pain. On examination, scarring or loss of normal pigmentation may be seen in a dermatomal distribution. Hyperoesthesia, hypoesthesia, and other sensory deficits are often found.[37] Associated symptoms can include sleep disturbance,

lassitude, anorexia, constipation, inactivity, social withdrawal, and secondary myofascial pain.

There are many treatment options. The best supported through clinical study are the tricyclic antidepressants. Opioid pain medications and topical applications are gaining in use. Anti-epileptics, although frequently tried, have not been clearly demonstrated to be of value. Sympathetic or somatic blockade is variably successful. Ablative techniques appear to have lost favor. It is not clear whether the incidence or course of PHN can be influenced by specific treatment regimes for HZ or by early and aggressive treatment of PHN.

§ 5.11 Complex Regional Pain Syndrome

Complex regional pain syndrome (CRPS) is a relatively rare, moderately to severely disabling neuropathic pain syndrome. Reflex sympathetic dystrophy (RSD), an older term for CRPS, is a complex disorder or group of disorders that may develop as a consequence of trauma affecting the limbs, with or without obvious nerve lesions. RSD or CRPS sometimes develops after visceral diseases or central nervous system lesions or, rarely, without an obvious antecedent event. It consists of pain and related sensory abnormalities, abnormal blood flow and sweating, abnormalities in the motor system, and changes in structure of both superficial and deep tissues. It is not necessary for all components to be present.[38] Because RSD is not necessarily a sympathetically mediated pain syndrome, and "dystrophy" is not an entirely accurate description, the International Association for the Study of Pain coined the newer term "complex regional pain syndrome" to avoid semantic confusion.

There are two distinctly different types of CRPS. *Type I* is what was formerly known as RSD — a syndrome that usually develops after an initiating noxious event, which is not limited to the distribution of a single peripheral nerve and which appears disproportionate to the inciting event. It is associated with edema, changes in the skin blood flow, abnormal sweating in the region of the pain, allodynia, or hyperalgesia. *Type II* is classical causalgia — burning pain, allodynia, and hyperpathia, usually in the hand or foot after partial injury of a nerve or one of its major branches. Some authorities recognize RSD as distinct from sympathetically maintained pain (SMP), but the International Association for the Study of Pain does not.

Although trauma frequently precedes the onset of Type I CRPS, the trauma can be as innocuous as spraining an ankle. Shortly after injury, a complex of symptoms develops, demonstrating involvement of the autonomic, motor, and sensory systems. Because of altered blood flow, the skin can become marbled, erythematous, or pallid. Skin temperature is frequently warmer or colder than the opposite limb. Sweating may be noticeably increased or decreased in the affected region. Less commonly, trophic changes are seen, such as altered nail and hair growth, thinning and glossiness of the skin, and in late stages, the development of osteoporosis. Muscular strength diminishes and active range of motion is typically reduced. Tremor may be seen and, rarely, a limb becomes dystonic. Diminished sensation may occur, but increased sensation to stimuli is more commonly experienced. Pain is typically out of proportion to the injury and is often described as burning and deep aching, and sometimes as paroxysmal. These symptoms do not follow nerve pathways, but often adopt a stocking or glove distribution and are not necessarily located at the site of injury. Usually, only one limb is affected but, for some individuals, symptoms may spread to the opposite limb and, rarely, to the entire body.

There has been an attempt to divide the development of CRPS into stages. The first stage (acute) is said to exhibit edema, warmth, and erythema; the second stage (dystrophic) is characterized by trophic changes and pallid, cold skin; the third stage (atrophic) is marked by muscular and bony atrophy and contractures. However, this staging system is unreliable. Individuals with CRPS do not predictably progress through these stages, the time spent in any stage is highly variable, and there is no symptomatic specificity for these stages. Symptoms tend to be durable, lasting years, and sometimes are permanent. Some individuals experience recurring CRPS, and there are occasionally spontaneous remissions.

The peak onset of CRPS is around age 50, although very young and very old people can also be affected. Women are affected more often than men.[39] The incidence of CRPS is about 1 in 5000.

There is no "gold standard" for diagnosing CRPS. Sympatholytic blocks with local anesthetic injected to the stellate ganglion or the lumbar paravertebral sympathetic ganglia may result in temporary relief of pain, which suggests a sympathetically mediated component. Similarly, intravenous guanethidine or phentolamine can indicate sympathetic mediation. X-ray examination may demonstrate bony demineralization as a later development. Triple-phase bone scans sometimes exhibit characteristic uptake patterns. Testing sudomotor function (the nerves that activate the sweat glands) can uncover side-to-side asymmetry.

The etiology of CRPS is not understood. Hypotheses include sensitization of peripheral small-diameter nerve fibers, changes in the modulation of sensory processing at the level of the spinal cord, altered control of blood vessels by local pain nerve fibers, and coupling of sympathetic neurons with pain nerves, resulting in abnormal firing of the nerves.[40]

Treatment often attempts to address the hypothesis of sympathetic mediation utilizing medications, typically sympatholytics like phenoxybenzamine, that decrease the action of sympathetic nerve fibers or circulating sympathetic neurotransmitters. Other adjuvant pain

medications are given in the hope that enhancing or diminishing other pathways will decrease pain. For example, tricyclic antidepressant medications offer at least two different mechanisms by which CRPS may be favorably modulated. Anti-seizure medications with a local anesthetic-like action sometimes work. Opioid pain medications, although less effective for CRPS than for other neuropathic syndromes, can nevertheless provide a significant reduction in pain. Physical therapy or a home exercise program is necessary to maintain strength and range of motion.

Desensitization techniques can be tried as a means of diminishing allodynia; typically, a gradient of tactile stimulation is applied to the affected area up to the limit of the individual's tolerance. TENS is frequently irritating but occasionally beneficial. Another important part of treatment is psychological counseling to address underlying psychological issues, develop coping techniques, and enhance sleep. Vocational counseling is of benefit for patients who are functional enough to return to some form of employment.

§ 5.12 Documentation of Chronic Pain Patients

A wide variety of pen and paper instruments can be used to measure patients' perceptions of pain. Commonly utilized tools for assessing the quantity and intensity of pain include the Visual Analogue Scale (VAS) and the Numerical Rating Scale (NRS). These tools use a 10-point pain rating scale on which 0 indicates no pain and 10 represents maximum pain. On the NRS, the patient manually numbers or verbally grades the pain level. On the VAS, however, the scores are kept from the patient. A "triple VAS" grades pain at three times: current, on the average, and at its worst.[41] When using the triple VAS, pain levels recorded over months are considered appropriate for obtaining an average pain level. The final VAS score is calculated by averaging the three pain level grades and multiplying by 100. A score lower than 50 is considered low intensity, and a score greater than 50 is high intensity. Some studies indicate that blinding the numbers from the subject is a more valid and reliable method. Other studies suggest that the VAS and the NRS are comparable, whereas still others assert that the NRS is more reliable.[42]

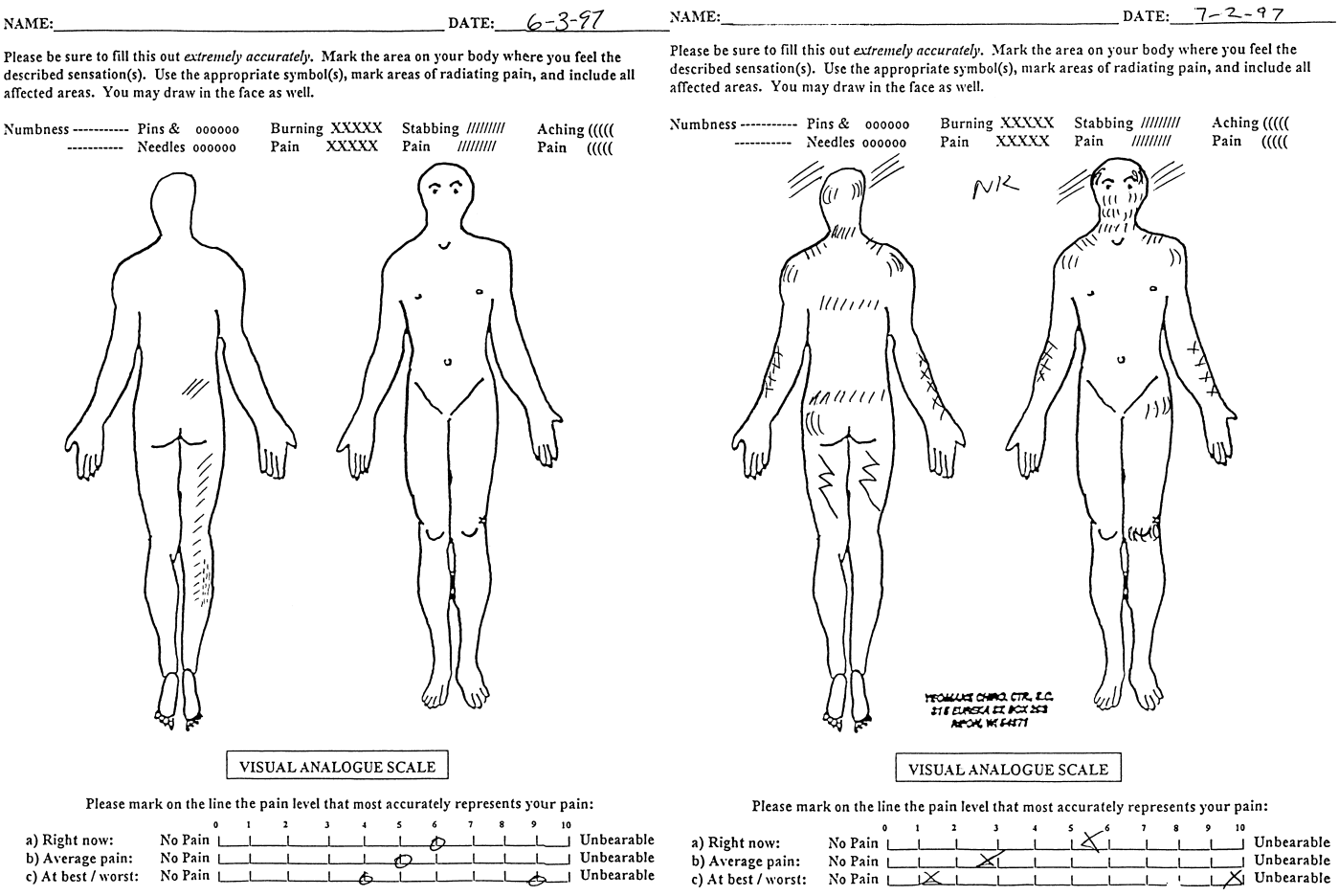

Figure 5–2. Pain drawings. (a) A normal, physiological pain drawing by a patient with low back pan and sciatica. (b) A drawing by a chronic low back pain patient, showing a nonanaomical pattern and markings outside the body.

Information regarding the quality of pain is best obtained from a pain drawing, which is a map drawn by the patient. Different marks are placed on an outline of the body to indicate the pain qualities felt in various affected body parts. Examples of pain quality include burning, aching, throbbing, stabbing, pins and needles, and numbness. A quick glance at a pain drawing can often help to decide if the presenting complaints are organically based. This decision may be verified by comparing the patient's history to the pain drawings See **Figure 5–2**, The pain drawing can also be scored by using a template with grid lines, adding up points for the number of marks outside the body and the number of body parts drawn on, noting pain traveling from the neck or low back into a limb, and other factors.[43]

The frequency and duration of pain are also important to determine. Frequency may be either constant or intermittent, and duration applies only to an intermittent pain pattern. This information can be quantified while taking the patient's history: "During what percentage of your waking hours do you have pain?" If pain is described as constant in frequency (100 percent of waking hours), further information on duration can be gathered by asking: "During what percentage of your daily waking hours is your pain at the maximum pain level? The minimum pain level?" These questions enhance the ability to track improvement

Each of the 31 spinal segments gives rise to a pair of mixed spinal nerves. Each pair of spinal nerves innervates a specific region of the body. Comparing the pain drawing with charts that map the body into dermatomes, myotomes, and sclerotomes can be helpful in determining whether there is an anatomic basis for the pain. Although this may sound simple, the line between organic and nonorganic pain is far from well defined, so all aspects of a physical examination are necessary to complete this difficult task.

From a clinical standpoint, patients typically have their history taken, then the physical examination is completed, from which an assessment is made and treatment rendered. The mnemonic SOAP (subjective, objective, assessment, plan) is often used to ensure that an important step is not missed.

§ 5.13 — Subjective Factors of Chronic Pain

Subjective or historical data is helpful in establishing a diagnosis of chronic pain. Historical and subjective factors to consider include the eight D's listed in **Table 5–5**. The presence of any two of the D's supports a presumptive diagnosis of chronic pain syndrome.

Other kinds of historical data that may support chronic pain behavior include:

Vague history, confused chronology, or information given that has nothing to do with the injury or symptoms

Expression of resentment toward prior caretakers due to mismanagement or neglect

Dramatic descriptions of the symptoms and the patient's

Table 5–5. THE EIGHT D's: CHARACTERISTICS OF CHRONIC PAIN PATIENTS [44]

CHARACTERISTICS	DEFINITIONS
Duration	The criterion used to be six months. The current opinion is that chronic pain can be diagnosed as early as two to four weeks. Prompt evaluation, recognition, and treatment are essential.
Dramatization	Unusual behavior, such as using affective, exaggerated, or emotionally charged words, and dramatic moaning, groaning, gasping, grimacing, posturing, or pantomiming.
Diagnostic Dilemma	Extensive history of evaluations by multiple physicians with repeated diagnostic tests. Clinical impressions tend to be vague, inconsistent, and inaccurate.
Drugs	Substance dependence and abuse of drugs and/or alcohol is frequent. Multiple drug therapy can lead to adverse interactions. An excessive amount of prescribed drugs may be consumed.
Dependence	Dependency on physicians, spouses, and families; excessive medical care; passive physical therapies with only short-term benefit; and relinquishment of domestic and social responsibilities.
Depression	Emotional upheaval is a hallmark. Psychological test results suggest depression, hypochondriasis, or hysteria. Cognitive aberrations give way to unhappiness, depression, despair, apprehension, irritability, and hostility. Coping mechanisms are severely impaired. Low self-esteem results in impaired self-reliance and increased dependence on other.
Disuse	Secondary pain occurs from prolonged, excessive immobilization. Health care providers' directives to be cautious can result in self-imposed splinting and cause progressive muscular dysfunction and generalized deconditioning. This perpetuates the pain cycle and illness behavior.
Dysfunction	Progressive loss of coping strategies and skills results in a gradual withdraw from the social milieu, including work, recreational endeavors; friendships; and family With increased isolation, activities are restricted to the bare essentials of life. "Bereft of social contacts, rebuffed by the medical system, and deprived of adequate financial means, the patient becomes an invalid in the broadest sense: physical, emotional, social, and economic."[45]

reaction to them Difficulty in localizing the symptoms or a complaint about many areas Failure of prior appropriate care to provide significant pain relief

Neurotic symptoms, such as anxiety, insomnia, irritability, pressure headaches, depression, crying spells, chronic fatigue, acute or chronic anxiety attacks, and/or neurotic gaits consistent with hysteria[46]

Psychological and physical factors are interrelated "in virtually every case of chronic back pain"[47] However, the U.S. Department of Health and Human Services commission on the evaluation of pain concluded in 1985 that chronic pain is not a psychiatric disorder.[48]

The Agency for Health Care Policy and Research (AHCPR) published guidelines for treatment of low back pain. These guidelines propose a diagnostic triage between mechanical nerve root involvement and red flags, such as tumor, infection, fracture, and cauda equina syndrome.[49] Other clinical factors are equally important in determining the prognosis of low back pain cases. These predictors of chronicity help health care providers and insurers identify potentially difficult to manage patients, often even before treatment has commenced. **Table 5–6** lists some of these prognostic indicators, which can help differentiate chronic pain patients with low back pain from other patients with fewer factors of chronicity.

Outcome assessment questionnaires can also contribute important information to the assessment of chronic pain patients. These questionnaires are completed by the patient and are usually placed in the history or subjective portion of the patient's record. For example; the Health Status Questionnaire (SF-36) poses 36 questions on general health issues-^ Responses are scored on eight general health scales and a depression scale:

General Health Scales
Health Perception
Physical Function
Role-Physical
Role-Emotional
Social Functioning
Bodily Pain
Mental health
Energy fatigue

Depression Screen
Dysthymia (major depression)

Affirmative responses in the depression screen may suggest the use of a more formal instrument, such as the Symptom Checklist (SCL-90-R), Beck's Depression Index, or the Zung depression questionnaire. In this author's experience, the scores on the numerical rating scale and condition-specific questionnaires, such as the Oswestry Low Back Disability Questionnaire,[57] are higher than the objective findings of either perceived or real activity intolerance. Therefore, equal weight should be given to the subjective risk factors to differentiate between organic and nonorganic portions of chronic pain syndrome.

Table 5–6. Predictors of Chronicity

Goert, M. (1990)[50]
1. Very heavy job classification.
2. Spasm and/or abnormal deep tendon reflexes.
3. Pain below the knee.
4. Lost time from work.

Burton, AK, Tilloptson, KM, Troub, DJ. (1989, 1991)[51]
1. Increasing age
2. A long initial spell
3. Initial onset early in life

Morris R. (1983)[52]
1. Roland Morris score of 14 or greater and/or worsening pain reported after four weeks
2. Positive straight leg raise less than 60 degrees
3. Gradual onset of pain
4. Duration of pain greater than one week before consultation
5. Absence from work for greater than two weeks

British Guidelines (1996)[53]
1. Loss of work in past year
2. Radiating leg pain
3. Positive straight leg raise (root tension signs)
4. Signs of nerve root involvement
5. Reduced trunk strength endurance[81, 82]
6. Poor physical fitness
7. Poor self-rated health

Cats-Baril, W, Frymoyer, JW. (1987, 1991)[54]
1. Characteristics of patient's job
2. Perception of fault
3. Compensable injury
4. Past hospitalization
5. Low educational level

Mercy Center Conference Guides (1993)[55]
1. History of more than four episodes
2. Symptoms lasting longer than one week
3. Severe pain intensity (more than 50 percent on VAS)
4. Pre-existing structural pathology related to symptoms

§ 5.14 — Objective Factors of Chronic Pain

The physical examination contains several objective tests designed to determine if nonorganic signs are present. One classic objective approach to assessing chronic low back pain is the Waddell test for signs of nonorganic low back pain.58 The concept is to test the patient in a manner that should not provoke a pain response. A response is considered positive if the patient reports pain, even though the test is carefully applied not to create a pain response. Nonorganic

low back pain is defined by positive responses on three or more of the five signs. In his article, Dr. Waddell specifically cautioned against overinterpreting these signs because false positives are common if the signs are not carefully assessed. The five signs include eight total tests (three categories contain two tests):

1. Pain: Two tests (superficial stimulation and nonanatomic location).
2. Simulation: Two tests (axial loading and trunk rotation).
3. Distraction: The patient reports pain on a supine straight leg raise (SLR) test, but does not report pain when a sitting SLR is performed later in the examination while the patient is momentarily distracted. A positive finding means that pain was reported in the supine undistracted test, but not in the sitting distracted SLR test.
4. Regional neurological exam: Two tests (sensory and motor neurological examinations). A positive finding means that the reported responses do not follow an explainable anatomical path.
5. Exaggeration: To be noted at any time during an examination.

Korbon introduced a quantifiable method of assessment using many of the Waddell signs.[59] In fact, however, there is no clear delineation between disability levels. After a discussion with Dr. Waddell, Korbon stressed the need for caution in interpreting these tests and argued against trying to force the signs into a grading scheme.

Standard orthopedic and neurological examinations are crucial in distinguishing organic from nonorganic responses. The presence or absence of nonorganic pain should be revealed by a detailed history and a careful physical examination. When the objective findings do not support the level of subjective complaints, the presence of chronic pain should be considered.

Some provocative objective tests, which are considered standard examination protocol, are frustratingly insensitive. Their limitations become apparent when their results are negative or equivocal, but the patient's complaints and the review of other health care provider records indicate ongoing dysfunction and disability. Measuring loss of function is typically accomplished in a low-tech manner by using an inclinometer to assess range of motion and muscle length, a stop watch to test trunk strength endurance, and counting repetitions. Trying to reproduce pain with provocative tests results in more useful information, especially at the end-stage of care. For example, a standard provocative orthopedic test, such as a straight leg raise test, is positive when sciatic nerve compression or nerve tension signs are present. However, in mechanical low back pain without nerve entrapment, the test is typically recorded simply as negative, which yields very little information. This qualitative objective test can easily be converted to a quantitative test by using an inclinometer to measure the degree of movement to determine hamstring length, which is often short in patients with mechanical low back pain.

Quantitative physical performance tests are especially useful in assessing soft tissue injuries when neurological function is normal, but the patient reports significant disability that is supported in the records. Nonorganic elements must be considered in chronic cases involving significant disability of intolerance of activity. Many fear-avoidance behaviors and pain personality traits result from a combination of physical and psychosocial elements.[60] Therefore, a quantitative functional capacity examination (QFCE) should be performed when the patient plateaus at a level short of full resolution. The QFCE establishes baseline data that can later be repeated to assess changes in function resulting from treatment or rehabilitation. It also helps in the final evaluation of permanent impairment, as the patient's data can be compared to normative data, and a percent loss of function can be assessed.

Of all the functional tests, only the static back endurance test has been found to predict the patients who are at risk for developing future low back pain (LBP). In one study, after adjusting for age, sex, and occupation, patients with poor performance were 3.4 times as likely to have another episode of LBP compared to patients with medium or good performance.[61] Hence, this test is useful not only in assessing current function but may also serve as a predictor of future LBP subjects.

The tests that make up the QFCE have been described as valid and reliable in peer-reviewed journals.[62] The following physical performance tests measure functional deficits. They should be considered whenever a patient plateaus short of full resolution. Unless further special testing is indicated, the patient may be enrolled in an active care rehabilitation program or simply discharged with permanent residual symptoms.

Muscle Length and Joint Range of Motion Tests

- Gastrocnemius/ankle dorsiflexion test (knee straight)
- Soleus/ankle dorsiflexion test (knee flexed)
- Modified Thomas test/hip extension test (iliopsoas flexibility)
- SLR (hamstring flexibility) test
- Knee flexion test/Nachlas (quadriceps femoris flexibility)
- Hip rotation range of motion (internal and external rotation)

Spinal Range of Motion

- Lumbar and cervical measurements

Waddell Nonorganic Low Back Pain Signs

- Pain (superficial stimulation and nonanatomical pain or tenderness)
- Simulation

- Axial loading: low back pain is reported on downward pressure on the skull
- Rotation: low back pain is reported on passive pelvic-shoulder rotation in the absence of nerve root pain
- Distraction
- Regional neurology (motor and sensory)
- Exaggeration or overreaction
- Waddell scores: 0 to 2 = within normal physiological (organic) limits;
- 3 to 5 = abnormal or positive

Strength Tests

- Repetitive arch-up test
- Repetitive sit-up
- Repetitive squat
- Static back endurance test
- Grip strength dynamometry

Balance/Proprioception Test

- One leg standing test

§ 5.15 — Mechanisms of Soft Tissue Injury in Motor Vehicle Accidents

Motor vehicle accidents (MVAs) are commonly associated with chronic soft tissue injuries. Their mechanisms of injury, pathogenesis, diagnosis, and treatment approaches can serve as a model for chronic pain syndrome. Residual complaints associated with MVAs represent a great clinical challenge because care is protracted, chronicity issues are involved, and litigation is often pending. There are many other causes of soft tissue injuries, such as falls, repetitive motion injuries, and exacerbation of pre-existing conditions. It is beyond the scope of this chapter to discuss every mechanism of injury that can result in soft tissue injury and chronic pain syndrome.

The classic whiplash, or cervical acceleration/deceleration (CAD), injury is often a Grade 2 sprain or strain in which ligament or muscle is partially torn. Little if any X-ray evidence may support the presence of a significant or permanent soft tissue lesion. The degree of injury depends on many factors. Common to all rear-end collisions is the delayed forward acceleration of the head and neck in comparison to the shoulders and trunk. Immediately after impact, as the vehicle, shoulders, and trunk are thrust forward, the head and neck remain stationary for approximately the first 100 milliseconds. As the trunk and shoulders are propelled forward and the head and neck remain stationary, the muscles in the anterior portion of the cervical spine are stretched. Depending on the position of the head restraint and its initial distance from the back of the head, the stiffness of the seat back, the angle of the seat, and the size of the person's neck, the stretched muscles act more or less like rubber bands, propelling the extended head and cervical spine forward at a faster rate than the torso. This acceleration compresses the anterior structures and stretches the posterior soft tissues. A second "crack the whip" phenomenon occurs when the stretched posterior muscles also act as rubber bands, pulling the flexed head and cervical spine backward. This basic mechanism of injury was discovered in the 1950s through experiments on anthropomorphic dummies and human volunteers.[63] An eight-mile per-hour rear-end collision produced a two-G acceleration of the vehicle and a five-G acceleration of the head within 300 milliseconds. (One G is the normal force of gravity.) The "crack the whip" phenomenon occurs because the head remains stationary at the time of impact and does not reach its peak of acceleration until approximately 300 milliseconds after impact. See **Figure 5-3**. Although reflex muscle contraction takes only 120 milliseconds and voluntary muscle contraction takes approximately 500 milliseconds, the degree to which muscle contraction attenuates acceleration forces is not sufficient to significantly mitigate injury.[64] This fact becomes more significant with age. Cervical range of motion decreases by approximately 40 percent, cervical muscle reflexes slow by 23 percent and voluntary strength capability diminishes by 25 percent[65]

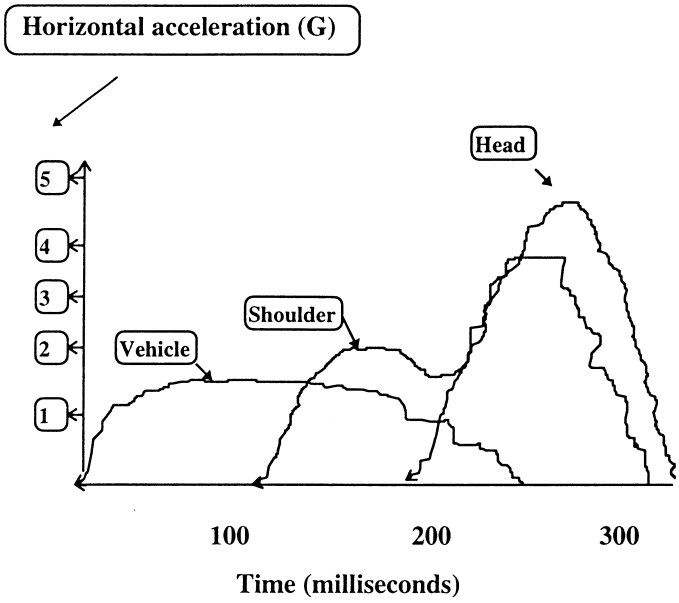

Figure 5–3. Sequence of events following rear impact. Adapted from D.M. Severy, J.H. Mathewson & C.O, Bechtol, *Controlled Automobile Rear-End Collisions: An Investigation of Related Engineering and Medical Phenomena*, *in* Medical Aspects of Traffic Accidents, Proceedings of the Montreal Conference 152-84 (1955).

The following factors contribute to occupant injury by allowing increased forward acceleration of the struck vehicle in a rear-end collision:

- Small struck vehicle
- Large striking vehicle
- Wet or icy road conditions
- Struck vehicle is moving at time of impact
- Brakes are not applied
- Automatic transmission in struck vehicle,

"A 6 to 8 kph (3.7 to 5 mph) rear-end impact, which subjects the cervical spine to as much as 4.5 G forces, constitutes the threshold for mild cervical strain injury."[66]

Two aspects of deformation contribute to the degree of injury. *Plastic deformation* refers to the increased external property damage as a vehicle dissipates the force of the impact by deforming and transmitting forces outward, thereby absorbing the energy of impact. This is why accidents in modern race cars, which are lighter than those built 20 years ago, result in less serious driver injury from rollovers and front-end impacts. *Elastic deformation* means that there is no external property damage to the vehicle, so energy during collision is transferred to occupants within the vehicle. It is commonly thought that cars that are "built like tanks" are safest in a collision, but in fact, they cause more injury to occupants than cars designed to break apart on impact. Transportation vehicles are not designed to plastically deform, so the energy of a collision is transferred to the occupants. As a result, vehicles that are not damaged in low-speed impacts can produce higher dynamic loading on the occupants than vehicles that plastically deform under the same impact conditions. Hence, the amount of damage to the automobile may bear little relationship to the injuries sustained in the cervical spine.[67]

§ 5.16 — Seat Belts and Head Restraints

The relationship of seat belts to soft tissue injury is still controversial. Seat belts may be responsible for more injuries than any other contact source within a vehicle as a result of the belt system operating in a manner in which it is designed, that is, preventing an occupant from contacting other structures within the vehicle.[68] The majority of seat belt injuries occur in the pelvic area and in the anterior superior iliac spine. Wearing a seat belt may be a risk factor for whiplash. Three-point seat belt systems in particular can create trunk rotation and prevent torso rebounding, thus increasing the flexion moment of the cervical spine. The single-shoulder restraint is most likely to induce rotation of the torso and neck when the unrestrained shoulder moves forward.[69]

Head restraints are effective only when properly positioned. Only 25 percent of adjustable head restraints are properly positioned, and only short distances between the headrest and the occupant's head will reduce injury.[70] The headrest should be positioned at least as high as the level of the occupant's ears. If the head is more than two inches from the head restraint, the ability of the head restraint to protect against neck injury sharply declines. If properly positioned, the headrest can reduce the incidence of cervical acceleration-deceleration injury.[71] However, an improperly positioned headrest that is set too low may increase the seriousness of neck injury by acting as a fulcrum for the head.[72] Injuries are further complicated by *ramping*, which is the upward movement of the occupant of the struck vehicle occurring immediately after impact. Ramping is caused by the angle of the seat back. The 5-to-10 degree angle in a seat designed to allow for a controlled backward collapse would provide fluid damping and reduce the G-forces on the head and neck.[73] Automobile seats can rebound with a velocity of 150 percent of the initial velocity.[74] Low- to moderate-speed rear-end crashes are more likely to cause neck injury than high-speed collisions because in high-speed crashes the seat back usually breaks and reduces hyperextension.[75] The stiffer the spring, the safer the seat.

If the head is in slight rotation, a rear-end impact will force the head into further rotation prior to extension. Cervical rotation prestresses various cervical structures, including the facet capsules, discs, and alar ligament of the upper cervical spine, making them more susceptible to injury. When the direction of force is from the side, or when a frontal or rear force occurs while the head is turned to one side, the spine is less flexible, and the force is increased on the facet pillar where the small bony elements may be fractured. Rotation also increases stress in certain soft tissue structures that reach their limits of motion sooner, resulting in more severe injury with less application of force.

§ 5.17 — Factors Affecting Severity of Injury

Severity of injury depends on six factors:

1. Force of impact.

2. Occupant awareness of impending collision. Injury to the neck is due to the inability of muscles to contract rapidly enough to compensate for the rapid movements of the head, neck, and torso resulting from the acceleration of impact. This is particularly true when the impact is unexpected and the victim is unable to brace for it.[76] "Three features of accident mechanisms were associated with more severe symptoms: An unprepared occupant; rear-end collision, with or without subsequent frontal impact; and rotated or inclined head position at the moment of impact."[77]

3. Position of occupant's head at moment of impact.

4. Gender and physical build of the occupant. Men tend to suffer more severe injuries, but women have a 40 percent higher risk of whiplash injury following a rear-end collision.[78] Children are also at higher risk than men.[79] Women are more likely than men to suffer cervical injuries because of their weaker neck muscles, longer necks, and lower body weight. Taller occupants are also at greater risk

for neck injury.

5. Age of occupant. Tolerance to impact decreases after age 40.[80] Injuries to occupants over age 65 have a poor prognosis because of decreased bone strength, decreased muscle mass, decreased brain weight, decreased intracellular fluid volume, decreases in nerve conduction velocity, and decreases in blood vessel flexibility.[81] Among the elderly, neck injury can be very serious because degenerative changes make the spine stiffer, so that it behaves like a single long bone rather than a set of articulating structures. Deforming forces are less evenly dissipated, and more damage results.[82]

6. Pre-existing conditions. A nonsymptomatic, pre-existing condition is not the cause of post-traumatic symptoms. However, "the vulnerability of the cervical discs to rupture increases as degeneration, annular fissuring, and nucleus pulposus desiccation progress, resulting in a situation in which a trivial trauma may cause disc rupture."[83]

Congenital anomalies that may complicate the effects of a whiplash injury include congenital anomalies of the spine, prior spinal surgery, prior spinal injuries, osteoporosis, osteoarthritis, rheumatic disorders, metabolic disorders affecting bone, primary or metastatic neoplasm, and bone infection. In a study of patients with similar injuries with pre-existing degenerative changes in the neck, and after an average of seven years post-injury, 39 percent had residual symptoms, and 55 percent showed X-ray evidence of new degenerative change at another level of the spine.[84]

§ 5.18 — Factors Affecting Prognosis

Risk factors influencing the prognosis following motor vehicle accident injuries include the following:

1. Symptoms persisting beyond six months
2. Significant ligament, disc, nerve, or joint capsule injury
3. Delay in initiating treatment
4. Need to resume treatment for more than one flare-up of pain
5. Occupant's age is over 65
6. Head restraint more than 2 inches from occupant's head
7. Occupant in a small car
8. Alcohol intoxication at time of MVA
9. Pre-existing radiographic degenerative changes
10. Prior whiplash injury
11. Prior cervical spine fusion
12. Female gender
13. Initial symptoms of radicular pain
14. Collar use for more than two weeks.[85]

Immediate symptoms usually indicate more serious lesions, such as disc or ligament rupture or end plate fractured Low back pain is also reported following motor vehicle accident collisions. Studies conducted in 1955, 1975, and 1991 found low back pain in 34 to 42 percent of whiplash victims.[87]

§ 5.19 — Delay in Reporting Symptoms Following Motor Vehicle Accidents

There are many reasons why patients delay reporting to a health care provider following injury related to a MVA:

- The patient may initially try to resolve the symptoms with pain medication
- Family members, friends, or physicians may have said the injury would take care of itself
- The patient may suffer from post-traumatic anxiety
- Time may be spent on car repairs, insurance company reports, or other more pressing priorities
- The patient may think the injury is insignificant.

The onset of symptoms may also be delayed following a rear-end motor vehicle collision Twenty-one percent of whiplash subjects did not appear to be injured at the scene of the collision.[88] Another 14 percent reported onset of symptoms 24 hours to one week post-accident.[89] Twenty-two percent of collision subjects with neck injuries did not have onset of neck pain until 12 hours or more after the collision, and 35 percent had onset ofradicular symptoms more than three months after the crash.[90] Onset of pain may occur within 12 hours of the accident,[91] or it may be delayed several days or even weeks after the injury.[92]

Particular neurological symptoms and signs (such as the early onset of intense headaches and neck pain) and certain accident features may indicate more severe injury. A short latency interval of symptoms appears to reflect more severe injury as well.[93] Delayed instability is also quite common.[94]

§ 5.20 Predictors of Chronic Pain Behavior

Many factors may be significant in predicting chronic pain behavior. The British Medical Association's guidelines discuss predictive factors in treatment of low back pain. These factors are listed in **Table 5–7**, along with the location where each factor may be documented.

In addition, the Mercy Center Conference Guides on practice parameters for the chiropractic profession discuss factors that may extend the period of treatment by a factor of 1.5 or more. These factors are listed in **Table 5–8**.

A profile of the disability-prone patient includes the following factors:

- Symptom magnification
- Pain avoidance behavior
- Psychological distress
- Job dissatisfaction
- Anxiety
- Treatment dependency
- Catastrophizing as a coping strategy
- Pending litigation

Methods of identifying disability-prone patients are discussed in §§ **5.13** and **5.14** on the subjective and objective factors related to chronic pain, and are summarized in **Tables 6–5** and **6–6**. Other methods worthy

Table 5–7. BRITISH MEDICAL ASSOCIATION FACTORS PREDICTING CHRONICITY[95]

1. Work loss in past year	Occupational history
2. Radiating leg pain	History and pain diagram
3. Positive straight leg raising test	Orthopedic and neurological exam
4. Signs of nerve root involvement	Orthopedic and neurological exam
5. Reduced trunk strength endurance	Physical performance testing (e.g., QFCE)
6. Poor physical fitness	Aerobic capacity tests (e.g., 3-minute step test)
7. Poor self-rated health	General health questionnaire (e.g., SF-36)

Table 5–8. MERCY CENTER CONFERENCE FACTORS PREDICTING CHRONICITY[96]

Predictive Factors of Chronicity	Documentation
1. History of more than four episodes	History
2. More than one week of symptoms before visit to doctor	History
3. Severe pain intensity	History (more than 50 percent on a triple VAS, or more than six positive responses on a single VAS)
4. Pre-existing structural pathology related to symptoms	History

of consideration include the following:

- Poor lumbar extensor strength or endurance
- Modified Work APGAR
- Vermont Disability Prediction Questionnaire
- Correlation with other outcome assessment tools
    - Pain drawing and VAS
    - Condition-specific questionnaires, such as the Oswestry Low Back Disability Questionnaire, the Neck Disability Index, the Headache Disability Index, and others.
    - Depression and Mental Health Scales of the SF-36
    - Symptom Checklist SCL-90-R

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