The Challenge of Pain

Ronald Melzack
and Patrick D.Wall

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method of treatment for these pains appears to be the modulation of the sensory input by either decreasing or increasing it. Phantom limb pain is sometimes relieved by successive local anaesthetic blocks of tender areas, peripheral nerves or sympathetic ganglia. It may also be relieved by vigorous vibration, transcutaneous electrical nerve stimulation, or by pain-producing injections of hypertonic saline into the stump or low-back interspinous tissues. Causalgia and the neuralgias can similarly be helped by anaesthetic blocks that temporarily decrease inputs from the affected areas, or by increased stimulation such as vigorous massage. These observations have given rise to new therapeutic methods that hold great promise for the relief of pain without producing irrevocable damage to the peripheral or central nervous systems.

These properties and their implications provide valuable clues towards an understanding of pain. They represent parts of a puzzle which, together with those obtained from psychology and physiology, will reveal the solution to a perplexing, urgent problem. Any satisfactory theory of pain must be able to explain the properties of these syndromes. If our theories do not lead eventually to effective treatment, they have failed, no matter how elegant or compelling they may seem. The clinical problems of pain, in other words, represent the ultimate test of our knowledge.

Part Two
The Physiology of Pain

'I was brought up in a medical generation in which . . . pain was [considered to be] a primary sensation dependent upon the stimulation of a specific sensory ending by a stimulus of a certain intensity, and conducted along a fixed pathway to ring a special bell in consciousness. Pain was as simple as that . . . The idea that anything might happen to sensory impulses within the central nervous system to alter their character, destination, or the sensation they registered in consciousness was utterly foreign to my concept. But in practice I found that it was incredibly difficult to make this concept consistent with clinical observations.'

William K. Livingston, 1943

perception all point to abnormal activity in the central nervous system in addition to any peripheral pathology. Changes in peripheral nerves certainly exist in these syndromes. However, these changes cannot be the whole story: surgical section of the appropriate nerves right up to the point of entry into the spinal cord or brain frequently fails to relieve the pain. Rather, the data suggest that changes in central nervous system activity, perhaps initiated by peripheral factors, may underlie the summation, delays, persistence and spread of pain.

Implications of the clinical evidence

The implications of the pathological pain syndromes described above are the following:

1 *Summation.* Gentle touch, warmth, and other non-noxious somatic stimuli can trigger excruciating pain. The fact that repetitive or prolonged stimulation is usually necessary to elicit pain, together with the fact that referred pain can often be triggered by mild stimulation of normal skin, makes it unlikely that the pain can be explained by postulating hypersensitive 'pain receptors'. A more reasonable explanation is that abnormal information processing in the central nervous system allows these remarkable summation phenomena to occur.

2 *Multiple contributions.* The pain, in these syndromes, cannot be attributed to any single cause. There are, instead, multiple contributions. The cutaneous input from the affected part of the body obviously plays an important role. However, inputs that result from sympathetic activity are also important. So too are inputs from the auditory and visual systems. All of these inputs appear to act on structures in the central nervous system that summate the total activity to produce nerve impulse patterns that ultimately give rise to pain. Anxiety, emotional disturbance, anticipation and other cognitive activities of the brain also contribute to the neural processes underlying these pains. They may facilitate or inhibit the afferent input and thereby modulate the quality and severity of perceived pain.

3 *Delays.* Pain from hyperalgesic skin areas often occurs after long

delays and continues long after removal of the stimulus. Gentle rubbing, repeated pinpricks, or the application of a warm test-tube may produce sudden, severe pain after delays as long as forty-five seconds. Such delays cannot be attributed simply to conduction in slowly conducting fibres; rather, they imply a remarkable temporal and spatial summation of inputs in the production of these pain states.

4 *Persistence.* The durations of these pain states often exceed the time taken for tissues to heal or for injured nerve fibres to regenerate. Causalgia tends to disappear as regeneration occurs, but sometimes it persists for years, as does neuralgic or phantom limb pain. Furthermore, in all of these syndromes pain may occur spontaneously for long periods without any apparent stimulus. These considerations – together with the observation that pain in the phantom limb frequently occurs at the same site as it occurred in the diseased limb prior to amputation – suggest the possibility of a memory-like mechanism in pain.

5 *Spread.* The pains and trigger zones may spread to unrelated parts of the body where no pathology exists. This is further evidence that the central neural mechanisms involved in pain receive inputs from multiple sources. The organization of these mechanisms does not reflect the precise dermatomal (or segmental) innervation of the body by the somatic nerves. (This is immediately evident when Figures 6 and 7 on pp.77 and 81 are compared.) Instead, the mechanisms appear to be more widespread and receive inputs from all parts of the body.

6 *Resistance to surgical control.* The widespread distribution of the neural mechanisms associated with these pain states is also indicated by the frequent failure to abolish pain by surgical methods. Surgical lesions of the peripheral and central nervous systems have been singularly unsuccessful in abolishing these pains permanently, although the lesions have been made at almost every level from receptors to sensory cortex. Even after such operations, pain can often still be elicited by stimulation below the level of section and may be more severe than before the operation.

7 *Relief by modulation of the sensory input.* The most promising

Therefore the left side of the spinal cord has more tissue to innervate and it is reasonable that the pain should be referred to the left. The uppermost thoracic segments also play a part in the development of the arms, so that some spinal cells receive converging signals from the heart as well as the arm. This convergence appears to be the reason why pain is referred to the left arm.

There is more to referred pain than just a mislocation by the patient. If you touch the left arm of a patient during an angina attack, you find that it is tender, although the right arm is not. This even applies when he is on the verge of having the attack. This is strange because there is no disease in his left arm. It is clear, then, that in addition to mislocation there is a summation of impulses from both sources. The tenderness of the arm suggests that nerve impulses from the heart and from the region where the pain is referred must converge and summate and thereby increase the pain. There is a very simple way of testing that idea. By using local anaesthesia it is possible to eliminate one of the sources of nerve impulses. In the case of the arm, it is possible to infiltrate the brachial plexus, the massed bundle of nerves at the root of the upper arm. If this is done, the arm becomes numb and it is found that the patient suffering from angina can do more exercise than normal before he triggers his pain. This suggests that the pain is triggered by two sources of nerve impulses: a major one from the heart and a minor one from the arm. These two add together. If one source is removed, it becomes more difficult for the other to trigger the feeling of pain. This applies as a general rule to referred pains but even more universally it applies to all pains. It will be seen that summation – the excitatory effects of converging inputs – provides important clues to understanding the causes and treatment of these pains.

Toothache

Sufferers and their dentists often have problems in locating the origin of a toothache, which is usually evoked by bacterial infection in the pulp of a tooth. Patients sometimes report that they have an earache when, in fact, the problem is not the ear but decay of the back upper teeth. In the case of front teeth, the patient frequently points to the wrong tooth, missing by one or two on either side of the culprit. The dentist knows he must

search carefully. He therefore examines, probes, X-rays and adds local stimuli to each tooth to detect where he can add a stimulus and enhance the pain.

In some cases, if the tooth infection is neglected, the pain increases as the pulpitis gets worse. Eventually the infection may leak out of the root of the tooth and begin to affect the gum. Now there is an instant and dramatic change: the patient accurately points to the exact area of trouble. The damage now involves superficial tissue with its ability to signal the true location of the injury. We have here, in a small area, a repetition of the changes in pain during appendicitis. In the initial stages, damage is limited to deep tissue and is incorrectly located. Later, superficial structures become involved and then the area in which the pain is felt coincides with the location of the damage.

evidence of a herniated lumbar disc, but the results fail to show that fusion is beneficial. In fact it may be deleterious. Loeser (1980) and Sweet (1980) urge strongly against continuation of the procedure in cases of disc protrusion.

In short, even when physical causes are clearly present, low back pain remains a problem after surgery for a substantial number of patients. And we are still confronted with the high proportion of people who have no obvious physical signs and still suffer agony.

Low back pain usually has a particularly unpleasant quality. It is deep, aching and burning and sometimes immobilizes the patient who is terrified of moving and triggering a severe bout of pain. Often the pains radiate down the leg and are called sciatic pain because they follow the innervation pattern of the sciatic nerve. For patients with minor physical signs such as curvature of the spine, or 'normal' disc disease that occurs with aging, surgery is rarely effective. Such patients, then, in the desperate search for relief of their pain, sometimes prevail on surgeons to carry out successive operations. After orthopaedic operations fail, these patients may undergo section of sensory roots (rhizotomy), of the spinal cord (cordotomy) and other operations; few of them help.

A variety of forms of physical therapy may help low back pain. The most effective is a regimen of special exercises that develop the back muscles. Transcutaneous electrical nerve stimulation, ice massage, and acupuncture may all help some patients. Injections of trigger points may be effective as well. Recently, it has been shown (Brena *et al.*, 1980) that injection of a long-lasting anaesthetic (bupivacaine) into the sympathetic ganglia relieves pain in a substantial number of people. But so does injection of saline, indicating that the mere hyperstimulation of the ganglia can bring about changes in the nervous system.

It is possible that the major culprit in many cases of low back pain is abnormal activity in nerve-root fibres due to minor changes in the surrounding vertebrae and tissues. The roots may be affected by compression caused by degenerated disc material (which commonly occurs during normal aging), interference with the blood supply, stress on ligaments and joints that surround the nerve, and so forth. These 'minor' irritations may be cumulative and eventually produce symptoms of 'low back sprain' (Gunn and Milbrandt, 1978). This can be the beginning of a 'vicious circle', because later pain would enhance the autonomic effects, produce spasm, pain, and progressive deterioration of a situation that began 'harmlessly' with normal aging processes (or possibly due to relatively minor physical trauma in younger people). Whatever the reason, the ensuing mechanisms are complex.

The actual neural mechanisms that are involved in back pain, even when disc herniation has occurred, are not clear. Evidently, either an increase or a decrease in input may be the basis of pain. Howe, Loeser, and Calvin (1977) found that chronically scarred axons tend to fire repetitively after mechanical compression and thereby produce an abnormal, high-frequency input through the dorsal roots, which could be the basis of low back pain and sciatica. On the other hand, prolonged compression of a nerve root may have the opposite effect – it may produce a marked decrease (rather than the expected increase) in firing in the root fibres (Wall, Waxman and Basbaum, 1974). The decrease could, of course, remove inhibitory influences and produce hyperactive spinal cells, which would tend to 'open the gate' and produce more pain.

As a result of the persistence of low back pain despite orthopaedic surgery, neurosurgery and countless drugs – most fail to work and some, such as tranquillizers, increase depression – it is not surprising that psychological therapy has become an important new approach to the problem. Indeed, anti-depressants (such as the tricyclic drugs) are sometimes remarkably effective in relieving the pain as well as the patient's depression. The various kinds of therapy that are effective are behaviour modification, progressive relaxation, hypnosis in its various modes of use, biofeedback to help learn to relax muscles, and so forth. All of these, it has been shown, help *some* patients. But no one of them is more effective than the others. In fact, clinics that employ several procedures at the same time get the best results. One group (Swanson *et al.*, 1976) found that patients with several syndromes, but mostly low back pain, were helped by a combination of techniques; about 80% of patients received marked to moderate improvement after treatment, and 50% claimed they were still

improved 3 to 6 months later. Interestingly, most patients reported that the pain was unchanged but they were able to work, to live with their pain, and lead more normal lives. Another study, specifically on chronic low back pain (Gottlieb *et al.*, 1977), also used a battery of techniques and found that about 60% of patients were able to resume a normal life style. The therapy in this study, it should be noted, required an average hospitalization period of 45 days. At 6 months after the programme, about 80% of the successful patients contacted still reported that they were living a normal life style.

The multiplicity of causes and treatments

The evidence on low back pain permits two important conclusions: (1) low back pain is not a single syndrome produced by a single causal agent; and (2) the most effective approach to pain relief and return to a normal life style is to use multiple convergent procedures.

It has been seen that disc disease and vertebral arthritis play a role in only a relatively small proportion of patients. However, other physical factors may play a role. Many patients with low back pain have tense muscles and many have clearcut trigger points which evoke severe pain when they are mechanically activated. Furthermore, many patients become depressed by their disability, lose their self-esteem, become obsessed with their health and are anxious. Finally, it has become clear that in a proportion of patients, low back pain is referred as a result of disease in another part of the body, especially in the pelvis (Loeser, 1980). Pain may be referred to the lower back as a result of a variety of visceral diseases that have gone undetected. Jones (1938), in a remarkable study, showed that inflating a balloon at various levels of the digestive system sometimes produces pain felt in the back. In other people, the pain is felt at the site of a scar of an earlier operation. Surgery of the back, then, can leave a scar that may potentially become the site of referred pain.

Therapists must look for trigger points, evidence of excessive sympathetic and muscle activity, and other physical contributions which can be helped by any one of the variety of sensory-modulation procedures. In addition, psychological assessment is essential to determine the psychological contributions – tension, anxiety, fear, and especially depression. The psychological methods described above can all potentially help to some degree. Finally, the patient, who has been terrorized by the pain and sometimes victimized unintentionally by health care professionals who do not understand the complexity of the problem, must be guided back to a normal life style. Behaviour modification methods, particularly those that recognize the patients' capacity to understand the problem and their need for satisfactory coping strategies, appear to be useful. The studies which report impressive relief of low back pain (Swanson *et al.*, 1976; Gottlieb *et al.*, 1977) have utilized virtually all of the above procedures at the same time.

The puzzle of pain, as we have seen, is far from solved. Increased research on pain is clearly needed. The number of scientists who work on the problem is small in comparison with the magnitude of its importance. We have a remarkable capacity to forget pains that we have suffered in the past, and it is often difficult to comprehend the suffering of another person. Research time and money are devoted to many problems of obvious clinical significance, but pain, often considered the symptom and not the disease, receives far less attention. Yet, there are few problems more worthy of human endeavour than the relief of pain and suffering.

But even when we acknowledge the priority of the problem and provide the funds for research, new problems arise that are the concern of all humane people – the ethics of research on pain.

The ethics of research with humans and animals

In recent years, there has been a rising tide of disquiet about the use of animals or people in experiments. This problem is especially serious for scientists involved in research on pain.

Experiments on healthy human subjects

Let us first consider the question of experiments on people, since most societies and individuals have debated this issue thoroughly and have reached fairly definite conclusions. The horrible experiments carried out on innocent people (who were considered

Pain without injury

Lesch-Nyhan disease is a rare congenital disorder which seems to be the opposite of congenital insensitivity to pain. The children appear normal at birth but they fail to thrive, both mentally and physically. They begin to exhibit self-mutilation, which is the major characteristic of the disease. With cries and appearances of great anguish, the child suddenly and viciously attacks some part of himself, acting as though it was the source of intolerable pain. It is necessary to restrain these children for their own protection. They do not attack others. Postmortem examination of their brains shows no anatomical abnormalities. The disease is tentatively explained as due to a failure to develop one of the essential enzymes which regulate metabolism (enzymeopathy). These children are particularly affected by a class of chemicals – the xanthines – which includes caffeine. In experiments designed to test the chronic effects of extremely large doses of caffeine in rats, it was found that the animals would start to bite viciously at their feet and limbs as though they felt that they were injured.

Pain disproportionate to severity of injury

Those who have experienced the passing of a kidney stone describe it as painful beyond any expectation that pain can reach such an intensity. The kidney may, under certain conditions, concentrate some components in the urine so that these compounds precipitate out and form small kidney stones (renal calculi). Small pieces of these stones break off and pass into the ureter that leads from the kidney to the bladder. In size, they are often not more than twice the size of the normal diameter of the ureter. Pressure of urine builds up behind the plug formed by the stone, tending to drive it into the ureter. As a result, the muscle in the ureter wall goes into localized strong contraction. This band of contraction moves down the ureter to produce peristaltic waves to drive the stone down. During this process, agonizing spasms of pain sweep over the patient so that the toughest and most stoical of characters usually collapses. The patient is pale, with a racing pulse, knees drawn up with a rigid abdominal wall and

motionless. Even crying out is restrained because all movement exaggerates the pain. As the stone passes into the bladder, there is immediate and complete relief, leaving a dazed and exhausted patient. The reason for describing this condition here is that in mechanical terms it is a rather trivial event. Furthermore it occurs in a structure which is poorly innervated when compared to any equal volume of skin. In spite of the minor nature of the actual event and the relatively small number of nerve impulses which are sent to the spinal cord, the effect in terms of pain is gigantic.

Pain after healing of an injury

Nerve injury of the shoulder is becoming increasingly common because motorcycles are widely accessible and all too often their power is far greater than the skill of their riders. On hitting an obstruction, the rider is catapulted forwards and hits the road at about the speed the bike was travelling. The wearing of crash helmets has effectively decreased head injuries; but the next vulnerable point to hit the road is often the shoulder, which is violently wrenched down and back. The arm is supplied by a network of nerves – the brachial plexus – which leaves the spinal cord at the level of the lower neck and upper chest, and funnels into the arms.

In the most severe of these injuries, the spinal roots are avulsed – that is, ripped out of the spinal cord – and no repair is possible. In 1979, well over a hundred brachial plexus avulsions occurred in England alone. C.A., aged twenty-five, an air-force pilot, suffered such an accident. After eight months he had completely recovered from the cuts, bruises and fractures of his accident. There had been no head injury and he was alert, intelligent and busy as a student shaping a new career for himself. His right arm was completely paralysed from the shoulder down and the muscles of the arm were thin. In addition, the limp arm was totally anaesthetic, so that he had no sensation of any stimuli applied to it. On being questioned, he stated that he could sense very clearly an entire arm, but it had no relationship to his real arm. This 'phantom' arm seemed to him to be placed across his

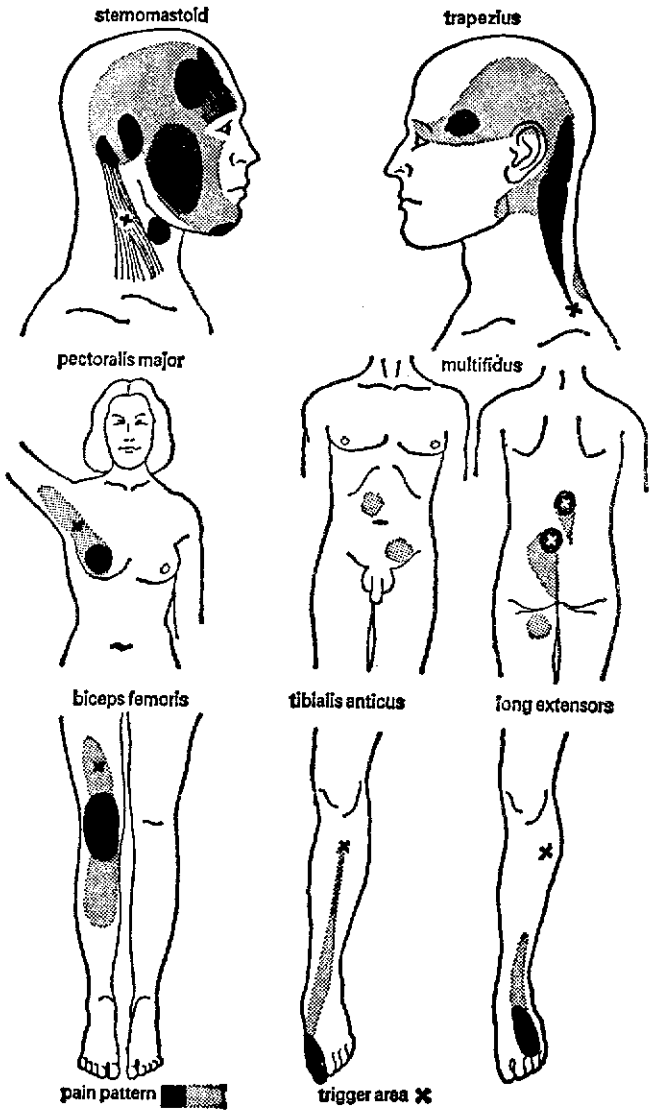

Figure 36. Typical myofascial pain patterns and their related trigger areas reported by Travell and Rinzler (1952, p.425). When the referred pain pattern of a muscle is known, it can be used to locate the muscle that is the source of pain. The name of the muscle associated with each pain pattern is shown.

sudomotor (sweating) and other changes. One effect could well be to produce local muscle spasm. Indeed, a recording electrode inserted into a trigger point elicits a sharp, severe, persistent pain and abnormal, high-frequency discharges in the muscle that persist for thirty minutes or more. No such activity is seen in normal muscle.

The kinds of trauma that initiate these activities are not fully documented, but it is clear that there is a variety of them (Simons, 1975, 1976). Loss of local blood flow (ischemia) due to a sudden sprain, unusual mechanical pressure, toxins, extreme cold or heat, and fever-producing diseases appear to be some of the causes. Persistent pressure on nerves at exit points, including nerve entrapment (Kopell and Thompson, 1976), is another. Scar tissue remaining from an earlier injury or even surgery may be yet another trauma that initiates the sequence of events that produces trigger points or larger trigger zones. Some of these causes, such as muscular stresses and strains, would produce patterns of trigger points common to most people, while others, such as scars, would vary from person to person. As Glyn (1971) notes, such 'insults' cause painful lesions which tend to heal spontaneously; but in the elderly, and in others in whom there may be a constitutional chemical abnormality in muscle or connective tissue, these trivial lesions perpetuate themselves until they become chronic.

Yet it is astonishing how easily many of these trigger point diseases are relieved (Travell and Rinzler 1952; Simons, 1975, 1976). Injection of an anaesthetic or saline into the trigger point, or simply needling the area with a dry needle may produce total relief of pain. It is essential (Bonica, 1957) to find *the* trigger point; that is, it is often highly localized, and stimulation must occur precisely at the point to produce the sharp pain and the sudden or gradual cessation of the vicious cycle of changes that produced it. Simons (1976) observes that the precise point is often found by the characteristic sharp pain and by a localized muscle spasm (not seen in normal muscle) which grips the tip of the needle as it penetrates. Why these simple procedures work is not clear. Possibly, the sharp, severe pain may activate descending inhibitory mechanisms which temporarily decrease or abolish the pain signals. This, in turn, may decrease the sympathetic outflow that appears to be an essential component of the whole cycle

(Glyn, 1971). The simplicity of these procedures is especially startling when we consider the severity and crippling nature of many of these myofascial pains, such as muscular rheumatism and low back pain (Glyn, 1971), torticollis ('stiff neck'), and a variety of other disabling myofascial syndromes.

Trigger points may involve only myofascial structures (Travell and Rinzler, 1952) or may become associated with pathological visceral activity (Simons, 1975, 1976). It is reasonable to assume that trigger points produce a continuous input into the central nervous system. Diseased viscera, then, may evoke an input which summates with the input from the trigger points to produce pain referred to the larger skin areas which surround the trigger points. Conversely, stimulation of the trigger points may evoke volleys of impulses that summate with low-level inputs from the diseased visceral structure, which would produce pain that is felt in both areas. These phenomena of referred pain, then, point to summation mechanisms which can be understood in terms of the gate-control theory.

Two types of mechanisms may play a role. The first involves the spread of pain to adjacent body areas. The T cell has a restricted receptive field which dominates its 'normal activities'; in addition, however, it is also affected by electrical stimulation of afferent nerves that cover a much larger body surface (Mendell and Wall, 1965; Devor, Merrill and Wall, 1977). This diffuse input is normally inhibited by gate mechanisms, but may trigger firing in the T cell if input is sufficiently intense or if the gate is opened. Anaesthesia of the area to which the pain has spread, which blocks the spontaneous impulses from the area, is sufficient to reduce the bombardment of the cell below the threshold level for pain. The discovery that the small visceral afferents project directly or indirectly onto lamina 5 cells (Pomeranz, Wall and Weber, 1968) provides the gate-control theory with still further power in explaining referred pain.

The second mechanism to explain referred pain involves the spread of pain and trigger zones to regions at a considerable distance, including visceral structures as well as cutaneous and myofascial areas. These referred pains suggest that the gate can be opened by activities in distant body areas. This possibility is consistent with the gate model, since the substantia gelatinosa at

any level receives inputs from both sides of the body and (by way of Lissauer's tract) from the substantia gelatinosa in neighbouring body segments. Mechanisms such as these may explain the observations that anginal pain, or pressure on other body areas such as the back of the head may trigger pain in the phantom limb.

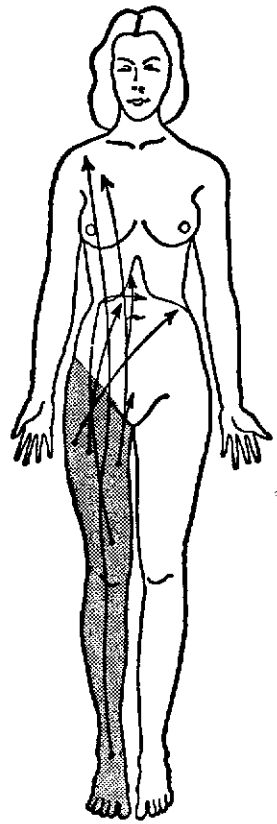

Figure 37. Patterns of referred sensation after cordotomy. The stippled area shows the region of analgesia produced by cordotomy in this woman. Heavy pressure applied to the analgesic skin produced 'an unpleasant form of tingling' that was felt at a non-analgesic part of the body. The sites of stimulation are indicated by dots, and the arrow from each dot indicates the point to which sensation was referred.
(from Nathan, 1956, p.88)

of many ways to produce analgesia by an intense sensory input which may be labelled generally as 'hyperstimulation analgesia'.

Hyperstimulation analgesia

Intense transcutaneous electrical stimulation

The concept that acupuncture is only one of many ways to deliver intense stimulation led Melzack and his colleagues to administer transcutaneous electrical stimulation the same way as acupuncture – for brief periods of time at moderate-to-high stimulation intensities. Consequently, they carried out a series of studies to determine whether acupuncture and transcutaneous electrical stimulation are comparable procedures.

The first study (Melzack, 1975b) examined the effects of brief, intense transcutaneous electrical stimulation at trigger points or acupuncture points on severe clinical pain. The data indicated that the procedure provides a powerful method for the control of several forms of pathological pain. The duration of relief frequently outlasted the twenty-minute period of stimulation by several hours, occasionally for days or weeks. Different patterns of the amount and duration of pain relief were observed. Daily stimulation carried out at home by the patient sometimes provided gradually increasing relief over periods of weeks or months. That these effects were not due to placebo phenomena was demonstrated in a double-blind study (Jeans, 1979).

Having established the effectiveness of brief periods of intense transcutaneous electrical stimulation, a study (Fox and Melzack, 1976) was then carried out to compare the relative effectiveness of transcutaneous stimulation and acupuncture on low back pain. The results showed that both forms of stimulation at the same points produce substantial decreases in pain intensity but neither procedure is statistically more effective than the other. Most patients were relieved of pain for several hours, and some for one or more days. Statistical analysis also failed to reveal any differences in the duration of pain relief between the two procedures. Interestingly, an almost identical study was carried out independently in Finland at the same time (Laitinen, 1976) and

also found that the two procedures were equally effective in relieving low back pain.

These findings have important practical implications. The chief advantage of acupuncture is that the procedure is of short duration – at intense levels, stimulation may sometimes last only a few minutes. The method, however, is invasive, and requires licensed practitioners with specialized training. Transcutaneous electrical stimulation, on the other hand, is non-invasive, and once the appropriate points are located, it can be administered by paramedical personnel. Furthermore, once the procedure is found to be effective for a given patient, it can be self-administered by the patient with supervision by the physician.

Our understanding of hyperstimulation analgesia is further enhanced by studies which show that the distribution of acupuncture points is similar to that of trigger points (see p.249) and motor points (the points which produce maximal contraction of muscles and have been shown to lie above the area of highest density of innervating motor neurons on the muscle). When acupuncture needles are inserted into sites that reduce pain, they produce a deep, aching feeling when they are twirled manually or electrically stimulated. This is reminiscent of the deep, aching feeling reported by patients when a trigger point is stimulated by the pressure of a finger pushing on it. This similarity led Melzack, Stillwell and Fox (1977) to examine the correlation between trigger points and acupuncture points for pain. The results of their analysis showed that every trigger point reported in the western medical literature has a corresponding acupuncture point. Furthermore, there is a close correspondence – 71% – between the pain syndromes associated with the two kinds of points. This close correlation suggests that trigger points and acupuncture points for pain, though discovered independently and labelled differently, represent the same phenomenon and can be explained in terms of similar underlying neural mechanisms.

A comparable study (Liu *et al.*, 1975) investigated the relationship between motor points and acupuncture loci and also found a remarkably high correspondence. This shows that there are sensitive sites on the body which produce a deep, aching feeling when they are palpated or needled, that they are intimately

relationship to the agent injected, but is 'related to the intensity of pain produced at the trigger zone, and to the precision with which the site of maximal tenderness was located by the needle' (Lewit, 1979, p.83). The needle, in short, must penetrate at the point of maximum pain. While this sounds like torture, the brief shot of pain produced by the needle resulted in striking relief of pain in 86.8% of cases and persistent relief for months or even permanently in about 50% of the cases.

Physiological basis of hyperstimulation analgesia

There are three major properties of hyperstimulation analgesia: (1) a moderate-to-intense sensory input is applied to the body to alleviate pain; (2) the sensory input is sometimes applied to a site distant from the site of pain; and (3) the sensory input, which is usually of brief duration (ranging from a few seconds to 20 or 30 minutes) may relieve chronic pain for days, weeks, sometimes permanently.

The first property can be explained by the brainstem mechanisms, described in Chapter 7, that exert a descending inhibitory control over transmission through the dorsal horns as well as at

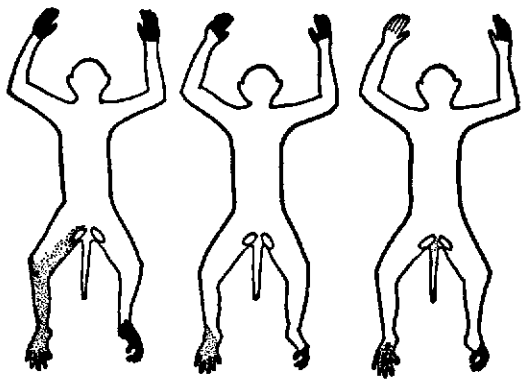

Figure 39. Excitatory and inhibitory receptive fields of dorsal horn cells in the monkey. The excitatory fields of three typical cells are indicated by the stippled areas. The inhibitory fields are shown in black. Large, medium, and small excitatory fields are illustrated from left to right. The inhibition of spontaneous or evoked activity was produced only by *intense* stimulation in the inhibitory fields, and persisted for as long as 1.2 seconds after stimulation was stopped. (from Wagman and Price, 1969, p.803)

higher levels in the somatic projection system. Intense somatic stimuli, of almost any kind, would produce pain but would also activate the brainstem mechanisms which could exert an inhibitory effect on the gate (Le Bars, Dickenson, and Besson, 1979a and b). However, this does not preclude direct inhibition at the level of the spinal cord. Wagman and Price (1969) found that the spontaneous or evoked activity of cells in lamina 5, whose receptive fields cover part or all of one of the legs, can be inhibited by intense stimulation of the opposite leg or even the hands (Figure 39). The short latencies of onset of the effect suggest that it may occur entirely by means of connections in the spinal cord. It is most likely, therefore, that both spinal and supraspinal mechanisms mediate the complex effects of intense stimulation on pain.

The same spinal and supraspinal mechanisms can also explain the second property – relief of pain by intense stimulation at a distant site. It is clear in Figure 39 that spinal cells excited by stimulation of a leg are inhibited by intense stimulation of areas as distant as the other leg or either hand. The brainstem mechanisms which exert a powerful descending inhibitory control provide an additional basis for inhibition of pain signals by intense stimulation at distant sites. These brainstem areas may be conceptualized as a 'central biasing mechanism' (Melzack, 1971, 1973) which acts as an inhibitory feedback system (Figure 40). Cells in the reticular formation and periaqueductal grey which respond to noxious stimuli exhibit a gross somatotopic organization characterized by large receptive fields (Groves, Miller, Parker and Rebec, 1973). Within the periaqueductal grey matter, Liebeskind and Mayer (1971) recorded responses evoked by noxious stimuli and found a somatotopic organization in which the face and forepaws are represented in the rostral portion whereas the hindpaws and tail are represented more caudally. Furthermore, when the periaqueductal area is electrically stimulated to produce analgesia, a complex somatotopic organization is revealed – there is a dorsoventral organization in which the face is represented dorsally and the more caudal parts of the body become analgesic as the electrode tip is moved ventrally (Soper, 1979). This basic organization exists throughout the rostrocaudal extent of the midbrain. It appears, then, that particular body