Depression Caused by Chronic Pain

NELSON HENDLER, M.D., M.S.

The chronic pain patient presents a difficult diagnostic and management challenge. The relationships between depression and chronic pain, and the stages of chronic pain with their associated psychiatric symptoms, are reviewed. The methods of validating pain and the most commonly missed causes of chronic pain are described. It is clear that depression associated with chronic pain is a complex problem necessitating a careful multidisciplinary approach if misdiagnosis and inappropriate or insufficient treatment are to be avoided.
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Almost all patients with chronic pain are depressed. In the majority, pain is the source of depression: in a few, chronic pain is a manifestation of depression and anxiety. Because pain is a totally subjective experience, the patient with chronic pain presents a difficult diagnostic challenge. Costly and protracted efforts may be made to establish an organic cause for the pain. Often, these efforts are unsuccessful, not because an organic basis for the pain is lacking, but because there are only a few measurable physiologic changes that correlate with pain. Inappropriate treatment of chronic pain further entrenches learned pain behavior, creating a host of medical and social problems. Patients find they must prove that their pain is real, and they use the health care system to do this. Many chronic pain patients are addicted to narcotics and misuse other drugs, usually tranquilizers, in ways that actually worsen pain and related symptoms.

The term "chronic pain" is often applied rather loosely to subsets of pain patients who, in fact, have differing and distinctive characteristics. In the context of this discussion, chronic pain refers to back and limb pain, as opposed to headache, gastrointestinal distress, genital pain, and cancer pain. The distinction is important, because each type of pain entails different psychological implications and has different meanings to patients. Chronicity of pain is really the only common feature of chronic back pain and chronic headache, for example. However, different kinds of pain tend to be associated with certain specific fears. Cancer patients fear dying; patients with genital pain fear sexual loss; and so on. This observation, along with the rest of this presentation, is based on an analysis of 358 inpatient admissions to Mensana Clinic, roughly 1,200 outpatient visits to the same clinic during the past 5½ years, and on my 8 years' experience as a psychiatric consultant to the Chronic Pain Treatment Center when it was part of the Department of Neurosurgery at Johns Hopkins Hospital.

From the Mensana Clinic and Johns Hopkins Hospital.
Reprint requests to: Nelson Hendler, M.D., Mensana Clinic, Greenspring Valley Road, Stevenson, MD 21153.

"PSYCHOGENIC" PAIN

Much confusion about chronic pain arises from the mistaken belief that, absent underlying organic disease, such pain is largely imaginary. In our practice, we do not see the psychological, psychogenic, or psychosomatic pain so often discussed. Indeed, it is my belief that the psychogenic pain diagnosis is made far too freely, often without reference to the patient's actual psychiatric status and history.

Frequently, this label is applied by professionals other than psychiatrists or psychologists — nurses, neurosurgeons, orthopedic surgeons, etc. — either because the patient fails to respond to medical or surgical treatment or because the patient is extremely difficult to manage. In the mid-sixties, Eugene Meyer analyzed the reasons for psychiatric referral of medical and surgical inpatients in a large city hospital. He found that in many cases, the referral was triggered by difficulty in managing the patient on the floor, rather than by any objective evidence of a mental disorder.[1]

It is also my experience that when orthopedic surgery, especially disc surgery, fails to clear pain symptoms, the psychogenic label is often applied. The patient is told, "The pain is in your head," rather than, "The surgery didn't give the good result we hoped for." The patient, of course, knows the pain is real, and he or she is infuriated to have someone suggest that there is a mental problem. The therapeutic alliance that may have existed between patient and physician is weakened or destroyed. And, if the pain persists, management of the depression that will inevitably result may become more difficult because of the "mental problem" stigma.

It is easy to be misled, of course. Depression and other psychiatric disorders are frequently manifested in somatic complaints, resulting in misdiagnosis. The clinician needs to be ever vigilant to the possibility that the patient's pain and other physical complaints may have a psychological origin. At the same time, he or she must recognize that the reverse is often true: Psychiatric problems — especially depression — can arise from physical disease.

DEPRESSION AND CHRONIC PAIN

Chronic pain and depression are closely linked. Chronic pain almost always leads to depression: This is normal — a point we emphasize to our patients. However, depression is rarely manifested by chronic pain. The first question we ask

depressed chronic pain patients is which came first, the persistent pain or the depressed mood. In most of the patients we see with back and limb pain, pain is the precursor of depression. Do patients who develop chronic pain syndromes arising from real organic causes have some intrinsic predilection to depression? There is no objective evidence to support this notion. However, patients with poor premorbid, prepain adjustment readily learn pain behaviors, which raises the possibility that there is more involved than just that first slipped disk. It is imperative, therefore, to gain a good understanding of the patient's prepain adjustment status.

The Stages of Pain

By way of example, let us consider a well-adjusted individual who injures his or her back cleaning out the garage. By well-adjusted, I mean someone who is married, has a good job, relates well to spouse and children, and has no history of alcohol or drug abuse.

When the injury produces chronic pain in such a person, he or she typically progresses through a four-stage response to pain[2] that is remarkably similar to stages of dying as described by Kubler-Ross. These four stages are described in the following sections.

The acute stage (0-2 months). The first stage of chronic pain is the acute stage, which lasts for up to 2 months. During this period, the patient normally expects to get well. He or she has sustained an injury, gone to the doctor, and possibly the hospital, and has been treated (presumably successfully). The patient anticipates a full recovery with few, if any, residual effects. So does the physician! The injury and resulting pain may have been quite severe. The pain may still be severe, but during the early phase, the patient believes it is only transient, and he or she is not clinically depressed. If the Minnesota Multiphasic Personality Inventory (MMPI) is administered to this previously well-adjusted patient during the initial pain stage, scores on all scales will invariably be normal.

The subacute stage (2-6 months). During the second pain stage, which lasts from 2 to 6 months, so-called hypochondriacal concerns begin to emerge. However, these concerns are not truly hypochondriacal; they have a legitimate basis. Contrary to expectations, the patient still hurts, and he or she wonders why. Is something wrong? Was there a misdiagnosis, or an error in the treatment? Was a break set incorrectly? Was a cast too tight? Was an X ray misread? The patient is telling the truth when he or she claims to have more body pain than other people. A positive answer to the "body pain" and similar questions on the MMPI generates elevated scores for hypochondriasis and hysteria. However, for these patients, the test falsely points to psychiatric pathology. It erroneously indicates that these behavior patterns reflect intrinsic personality traits rather than a transient response to real, continuing pain.

The chronic stage (6 months-8 years). When chronic pain persists past 6 months in our once well-adjusted patient, he or she moves into the third stage of pain — the chronic pain stage. The patient may remain at this stage for the next 3 to 8 years — or more. Marked depression, triggered by the realization that the pain appears to be permanent, is the rule during this stage. This is usually a full-blown depression, with sexual dysfunction, sleep disturbance, loss of self-esteem, guilt, increased suicide risk — in short, all the symptoms of a major affective disorder. At this point, on the MMPI profile, the patient has elevated hypochondriasis, hysteria, and depression scales, with the depression scale the most elevated of these. Patients with this profile are sometimes unjustly labeled with pejorative, even insulting, terms such as "pain prone," "low back loser," "pain neurotic." Such language is inaccurate and damaging to the patient and should be discarded.

The subchronic stage (3-12 years). In the final, subchronic pain stage, the patient becomes reconciled to the situation and starts to adjust to it. Acceptance and accommodation are the motifs of this stage, which occurs anywhere from 3 to 12 years after the initial injury. The patient realizes that the pain will probably persist for life, no matter what medical intervention is attempted. He or she is not happy with the situation, but nevertheless begins devising strategies for dealing with the pain and functioning despite it, rather than fighting it. In most cases, depressive symptoms decline significantly or disappear entirely. When an MMPI is given to patients at this stage, the depression scale is usually low, although the hypochondriasis and hysteria scales are generally still elevated and thus subject to dangerous misinterpretation.

Here again, the test findings are often misinterpreted due to a fundamental misconception about what happens to the chronic pain patient. In consequence, the patient may be described as having a "conversion reaction" or a "hysterical neurosis" because of the "conversion V" seen on the MMPI.

The Physician's Role

The physician can play an important role in helping the patient deal with the pain and learn to function in spite of it. However, I recommend that patients never be told that they have to "live with the pain." To be sure, they must accept the pain and work with it, but they do not have to settle for the negative, hopeless state that this expression conveys. Chronic pain patients must readjust their goals, and recognize that there are some things they may never be able to do again. Because it maximizes the probability of success and minimizes the possibility of failure, realistic goal setting is of critical importance in managing chronic pain.

I want to stress the potentially disastrous consequences of looking at the results of the MMPI and other psychological status screens in the absence of a thorough historical perspective on the patient. Such results *must* be considered in the context of the other clinical findings noted in tracing our chronic pain patient. I recall years ago sending a young woman with total deafness in one ear and a 50% hearing

loss in the other to a psychologist for an MMPI. His diagnosis was paranoid schizophrenia, which I did not expect at all. I asked why he thought the patient was schizophrenic. It turned out that my patient had answered "yes" to the question, "Do you sometimes hear voices without knowing where they are coming from?" After a brief discourse on the need for biaural hearing for echo location, we revised the diagnosis.

EVALUATING PAIN

In evaluating chronic pain patients, the relationship of the pain to the underlying triggering event is critical. Patients who follow the four-stage progression just described generally have a significant degree of *objective* organic pathology (i.e., damage to nerves, bone, blood vessels, or muscle) that is demonstrable on EMG, nerve conduction studies, thermography, CAT scan, X ray, or myelogram. (Unfortunately, as there is no objective test for directly measuring pain, clinicians must rely on measurements of physiologic factors generally known to have a high correlation with the presence of pain but which vary considerably from individual to individual.) In contrast to these individuals with objective pain is the exaggerating pain patient (Figure 1). This is an individual who may not have been well-adjusted before the onset of the pain. He or she may have been married several times, have an alcohol and/or drug abuse history, and may present evidence of a prepain personality disorder. There is a real organic basis for experiencing pain, but the severity and persistence of the pain exceed the relatively minor nature of the measurable damage, such as the kind of intermittent lower back problems that plague millions of people. The exaggerating pain patient is devastated by such a problem: He or she cannot function. The pain is not imagined or faked: It is psychologically exaggerated. In my experience, the pain exaggerator is rarely depressed by the pain. To the contrary, the pain is used with great skill to manipulate the patient's environment.

It is clearly necessary to distinguish between the objective and the exaggerating pain patient in order to plan treatment and to predict outcome. This distinction can determine the response to treatment or surgery and help to predict compliance to treatment regimens, as well as the litigiousness of the patient. How exactly do you assess the person's chronic pain? What precisely are you looking for? In most instances, the first concern is defining the validity of the pain complaint. As a consultant, the question I am asked is, "Is the pain real? Is the patient faking it?" Unfortunately, this question is often impossible to answer, especially in the absence of clear-cut organic pathology underlying the pain complaint. As I have observed, pain is a purely subjective experience that defies direct measurement. One can only measure the *impact* of pain on the patient's life.

Outright fabrication of chronic pain is rare. To the patient, the pain is almost always real, although unconscious exaggeration or conscious amplification may play a part. What the patient says directly about his or her condition is totally subjective and must not be challenged. In any case, subjective severity is of little value in validating the pain. The clinician must depend on the history, physical findings, and appropriate psychological testing to determine the etiology of the pain and to develop treatment that will diminish or eliminate it.

Figure 1. Objective vs. exaggerating pain patients.

OBJECTIVE PAIN PATIENT
• Good premorbid adjustment
• Objective basis for complaint of pain
• Normal response through the four stages of pain
EXAGGERATING PAIN PATIENT
• Real etiology of pain, but minor damage
• Poor premorbid (prepain) adjustment
• Little or no depression
• High secondary gain

Psychological Tests

The MMPI. In terms of pain validation, the MMPI is useless. The MMPI results tell you about personality traits and may suggest whether the individual is deliberately or unconsciously exaggerating his symptoms, but they will not indicate whether the pain is "real." Several studies suggest that different MMPI scales are useful in predicting outcomes in certain kinds of orthopedic surgery, while others find no predictive value. Certainly, the MMPI cannot evaluate the validity of the complaint of pain.

The Hendler Screen. We have devised the Hendler Screening Test for back pain patients to assess pain validity regardless of the presence of preexisting personality disorders.[4] This is important because hysterics *do* get disc disease, and Briquet syndrome patients *do* get sympathetic dystrophy. Our test is analogous to Eysenck's concept that personality traits can exist on an X axis, while psychotic symptoms can exist on a Y axis, and that a patient may have severe psychosis without neurosis, or vice versa. The Hendler Screening Test is based on the following concepts:

1. Severe physical pain alters certain patterns of living, regardless of preexisting personality traits.
2. One must examine prepain behavior to determine the impact of pain on someone's life.
3. A physician can determine a pathologic (exaggerated) response to pain only by longitudinally studying a normal patient's response to pain and comparing the two responses.
4. Regardless of preexisting personality traits, if a patient develops severe, debilitating physical pain, there will be a predictable pattern of response to this pain.

The test is structured around groups of simple, unambiguous questions that cumulatively reveal actual pain response patterns. For example, it is important to find out if the pain is causing sleep difficulties. The first question in this area is, "Do you have trouble falling asleep at night?"

If the answer is "yes," then one asks "Why?" The response "The pain keeps me awake" tells you a lot about the pain: So does the answer, "My teenage boy stays out late and I worry about him driving at night." Another question is: "If you had three wishes for anything in the world, what would they be?" One patient may respond, "I want my husband to stop drinking, my son to get off drugs and my daughter to stop fooling around," while another may say, "The only thing I want is to get rid of the pain." These responses are very revealing.

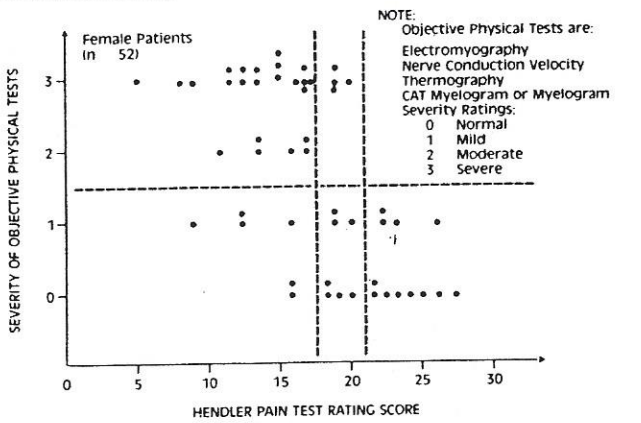
We ask the same kind of questions in other areas such as sexual activity and financial status, and eventually acquire insight into how much the pain actually affects the patient's day-to-day life. There is no way to measure chronic pain directly, but, by asking very specific questions, one may get an idea of the impact of the pain on the patient's life. And, in our experience, the degree of impact (which can be expressed quantitatively in a standard matrix applicable to all patients) fairly accurately reflects the validity of the pain.

There are 15 questions in our test, which takes about 10 minutes to administer. How good is our test? In an attempt to find out, we looked at the objective measures of organic pathology (EMG, nerve conduction, thermography, CAT scan, myelogram, and X ray) in 82 male and female patients and compared these findings with the results of the MMPI and the Hendler test on these individuals. We reviewed the objective findings blindly, without any foreknowledge of the psychological test scores, ranking each on a scale from 0 (normal) to 4 (most severe). We then correlated the objective data with each scale on the MMPI and Hendler test. We found that none of the MMPI scales could predict the presence or absence of physical abnormalities as indicated by the objective measuring procedures. In other words, the MMPI was totally unable to predict the presence of organic disease.

In contrast, the Hendler test was generally reliable in predicting the presence or absence of organic abnormalities. Of patients scoring 17 or less on the test, 70% had objective abnormalities on at least one of the six physiologic tests mentioned above (see Figure 2). Of the patients who scored 21 or more on the test, *none* had measurable organic pathology, as predicted. With further testing this extremely high accuracy rate will no doubt decrease.

The Stress Vector Analysis. The Stress Vector Analysis, an amalgam of eight commonly used psychiatric tests, measures and compares stress in three areas — environmental, personality/psychological, and physical — and can be helpful in validating chronic pain, since stress and chronic pain are often associated. The SVA normally integrates the Holmes-Rahe Scale, the SCL-90, the Health Scale, the Somatic Stressor Scale, the Life Stressor Scale, the Type-A Behavior Scale, and the Hendler Screening Test. In our application of the SVA, for reasons I have already mentioned, we have replaced the MMPI with the Hubbard-Staats Validity Scale, a test that indicates the reliability of the subject's responses (i.e., whether the patient is answering truthfully). The SVA defines the sources of stress in a patient's life and compares findings against appropriate group norms.

Figure 2. Scattergram of Hendler Pain Test score versus severity of objective physical tests.



COMMONLY MISSED CAUSES OF CHRONIC PAIN

As I have emphasized, chronic pain usually has a legitimate organic basis, although the extent of pain, severity of disability, and the degree of organic pathology may be poorly correlated. In some chronic pain patients, the underlying organic problem may be overlooked or obscured, possibly leading to a wrongful designation of hysterical pain or imaginary pain. It is appropriate, therefore, to review some of the commonly missed causes of chronic pain, which include myofascial syndrome, sympathetic dystrophy, facet syndrome, and temporo-mandibular joint syndrome.

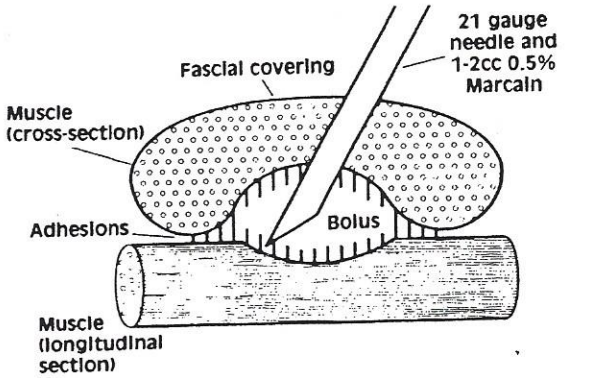
Myofascial Syndrome

Myofascial syndrome, also called fibromyositis, myositis, and fibromyalgia, is usually a residual consequence of soft tissue injury. The injury causes extravasation of extracellular fluid between the fascial covering of muscles and causes adhesions that prevent these muscles from gliding smoothly over one another in the normal fashion.

In the past, it has been difficult to diagnose myofascial syndrome, and some clinicians argue that the condition is nonexistent. One method of documenting the presence of this disorder is through the use of surface electrodes to measure muscle tension, using EMG biofeedback equipment as a recording device.[5] The advent of color thermography has made it possible to pinpoint discrete lesions within muscle groups. These show up as either hot or cold spots, depending on the chronicity of the symptoms. Clinically, one can palpate discrete nodules (called trigger points) in an affected muscle group. Multiple trigger points may be found in a single large muscle.

We use trigger point injections to treat myofascial syndrome. This is controversial, of course (if the syndrome does not exist, then what are you treating?), and there are

Figure 3. Trigger point injections in myofascial syndrome.



numerous theories about what constitutes a trigger point within a muscle. Some people postulate that the trigger point relates to the accumulation of mucopolysaccharides. Others contend that trigger points arise from microinfarcts. In my opinion, the most logical explanation is that trigger points are caused by fibrosis between fascial planes, arising, in turn, from soft tissue injury.

As shown in Figure 3, we insert the needle between the fascial planes where the lesions are located, i.e., into the nodule (area of localized pain). The mechanical lysis of the trigger point is the basis of the therapeutic effect, regardless of what substance is injected. Water or saline will work, but we usually use 0.5% bupivacaine, which minimizes discomfort to the patient. The injection either blows apart the microinfarct or separates the fascial planes, allowing the muscles to move freely without causing microspasms. Sometimes, simply injecting subcutaneously into areas of chronic pain or chronic muscle spasm gives relief. However they are done, injections should be followed by muscle stretching to break up additional adhesions. In our experience, the sooner myofascial syndrome is diagnosed and treatment is started, the greater the likelihood of long-term benefit.

Sympathetic Dystrophy

Sympathetic dystrophy is another chronic pain-producing syndrome that is frequently overlooked, although it has been discussed in the literature for more than a century. Clinically, the condition is characterized by burning, pins and needles, and coldness in one or more extremity. There is a positive response to a light touch, but not to deep pressure. The early presentation of sympathetic dystrophy may occur without the hair loss, osteoporitic changes in the distal phalanges, and marked temperature difference between affected and unaffected extremities that are described in textbooks as classic manifestations.

At one time, diagnosis of such conditions depended on the ability of the physician to perceive, by touch, temperature differences in affected and unaffected limbs — a patently unreliable procedure given the human animal's relatively limited sensitivity to temperature differential. Today, thermography is highly accurate in the early detection of sympathetic dystrophy, causalgia, and other conditions that compromise blood flow and perfusion into the extremities and other areas of the body.[6] Sympathetic dystrophy is treated with repetitive sympathetic blocks, and if indicated, sympathectomy.

Facet Syndrome

Perhaps the most commonly missed source of chronic back pain is facet syndrome. The condition usually results from injury (due to twisting or bending) to the capsule located between the upper and lower facets of adjoining vertebra and to a small sensory nerve that supplies the area (the recurrent nerve of Luschka). There may be inflammation of the capsule; arthritis may develop in the facet area. Bony tissue may start to press on the small sensory nerve, causing radiating pain to the lower back, buttocks, and upper legs, but usually not below the knees.

Because some of these symptoms are similar to those of disc herniation, diagnosing facet syndrome can be tricky. A myelogram, of course, will be negative, and one may be tempted to believe that the patient is faking. However, if the patient tells you the pain does not go below the knees, is made worse by movement, bending, and lifting, and is *not* made worse by coughing or sneezing, irritation of the facet joint is strongly suggested. By simply asking the patient about the relationship of the pain to position, one may avoid a protracted and unsuccessful progression from specialist to specialist. The primary treatment of facet syndrome is nerve block at the joint and then burning out the nerve.

Temporomandibular Joint Syndrome and Bruxism

Temporomandibular joint syndrome (TMJ) is usually due to persistent grinding of the teeth. Involvement of the temporalis and masseter muscles produces bitemporal pain, which may radiate to the eyes and neck. The jaw clicks on opening and cannot open more than 30 to 35 mm. Temporomandibular joint damage may or may not be present, so an X ray will not necessarily rule in or rule out TMJ syndrome. However, worn cusps of molars, bicuspids, and tricuspids may suggest bruxism as a cause of bitemporal headache. Bruxism is treated with injections into the external pterygoid muscles, oral nonsteroidal anti-inflammatory drugs, muscle relaxants, bite plates, and EMG biofeedback.

THE NEUROCHEMICAL BASIS OF CHRONIC PAIN

The patients we see with chronic back or limb pain typically go through a protracted period (years in most instances) during which they have sleep difficulties, depression, anxiety and, of course, constant pain. An understanding of what specifically occurs at the neurochemical level is helpful in planning a treatment regimen for these individuals.

Figure 4. Effects of neurotransmitters on sleep, depression, and pain.

	↑ Sleep	↓ Depression	↓ Pain
Serotonin	↑	↑	↑
Norepinephrine	↓	↑	↓
Location	Reticular Activating System	Limbic System	Periaqua-ductal gray

Basic Neuroanatomy

The mechanism of central nervous system (CNS) activity, of course, depends on many different neurosynaptic transmitters. Thirty percent of CNS neurotransmitters use GABA (gamma-aminobutyric acid), and nearly all of these neurons are found in the cortex. Another 10% of CNS neurotransmitters use acetylcholine and most of these are in the limbic system. The biogenic amines, believed to play an important role in the mediation of pain and emotional perception, account for only 2%-5% of CNS neurotransmitters. They include catecholamines, such as epinephrine, norepinephrine, dopamine, and L-dopa, and the indoleamines, such as serotonin. Ninety percent of the biogenic amines are found in the limbic system — the hypothalamus, the median forebrain bundle, the periventricular area of the hypothalamus, and the reticular activating system of the medulla — areas of the CNS that control emotion, vegetative functions, and sleep.

The enkephalins, endorphins, and dynorphins also concentrate in the limbic system and have primarily a neurosynaptic effect; beta-endorphin may also have a hormonal effect. There is considerable neuroanatomic overlap between the biogenic amines and enkephalins and beta endorphin, which suggests an intimate relationship of function as well.

Neurochemistry of Sleep, Depression, and Pain

Neurochemically, the accumulation of serotonin in the dorsal median raphe nuclei of the medulla's reticular activating system increases natural sleep. Sleep and wakefulness are governed by the ratio between serotonin and norepinephrine (see Figure 4). When serotonin is increased and norepinephrine decreased, natural sleep occurs.

Increasing serotonin also reduces certain kinds of depression and, most importantly in the context of chronic pain, reduces the perception of pain. This has been demonstrated in studies by Hososuchi et al.,[7] who found that putting serotonin in the cerebrospinal fluid of animals enhanced the natural process thought to be mediated by enkephalins released by electrically stimulating the periventricular areas, which results in analgesia. In other words, pain perception can be diminished by enhancing the action of either endogenous opiates or exogenously administered morphine-like substances by the addition of serotonin. When the process is reversed and norepinephrine is introduced exogenously, pain perception increases.

The ratio between serotonin and norepinephrine can be altered through pharmacologic modification of synaptic transmission. The administration of L-tryptophan encourages presynaptic buildup of serotonin, thus increasing the availability of the substance. Likewise, the same effect can be achieved by blocking presynaptic reuptake of the neurotransmitter with a tricyclic antidepressant. This is an important neurochemical consideration, since the activity of almost every neurosynaptic transmitter except acetylcholine is terminated by presynaptic reuptake.

TRICYCLICS IN CHRONIC PAIN

A number of tricyclics have been found to be effective in treating both depression and pain in chronic pain patients. The tertiary amines (doxepin, clomipramine, imipramine, amitriptyline, and nortriptyline) have all been found to block serotonin reuptake more than they block norepinephrine reuptake. Two of these, doxepin and amitriptyline, appear to be especially useful in treating mixed pain and depression. Both block serotonin reuptake more effectively than norepinephrine reuptake. Ward has reported in this issue on a controlled study demonstrating the effectiveness of doxepin in managing chronic back pain patients with concomitant depression. Heretofore, it was thought that tricyclics decreased pain perception exclusively by increasing serotonin availability. However, recent work in Europe indicates that some of these agents are able to inhibit the action of enkephalin hydralyzing enzyme in the postmitochrondrial fraction of the cortex, thus "protecting" enkephalin from degradation.[5] This may prove to be another mechanism by which the tricyclic antidepressants increase pain tolerance.

CONCLUSIONS

The diagnosis and management of chronic pain demands knowledge of many fields, including basic biochemistry, pharmacology, neuroanatomy, orthopedics, neurosurgery, rheumatology, psychiatry, neurodiagnostic studies, and even sociology and the law. Each of these areas contributes to the evaluation of the patient with pain. For this reason, chronic pain is best diagnosed and treated in a multidisciplinary setting.

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