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A Civilian Experience With Causalgia

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The case histories of 32 patients treated in a University hospital for "minor causalgia during a recent ten-year period have been reviewed. The inciting causes were varied and could rarely be directly attributed to nerve injury. Patients with minor causalgia presented for treatment much later following injury than patients with "major" causalgia seen in the same hospital, and they exhibited varied physical signs. Osteoporosis was present in over half of the involved extremities examined radiographically. Because of the less severe nature of the pain, the remoteness of the injury, and the varied clinical presentation, over half of these patients had been treated unsuccessfully on the basis of other diagnoses and many were considered psychoneurotic. Sympathetic block proved to be the most accurate diagnostic aid and in four cases was of therapeutic benefit. Sympathectomy produced gratifying relief in 24 of 27 patients, and all patients who responded favorably to sympathetic block benefited from surgery. Follow-up in 15 patients, 2 to 17 years after surgery, revealed lasting relief in 13. In civilian practice, minor causalgia is more common than major causalgia, and, because it is so readily treated, early recognition is of great clinical significance.

"Minor" causalgia or "reflex sympathetic dystrophy" is probably one of the most poorly understood and frequently misdiagnosed entities encountered in clinical practice. This is all the more unfortunate because most of those afflicted with this condition can be relieved by relatively simple measures. The term "causalgia" was coined from two Greek words meaning "burning pain" by Mitchell et al in the first comprehensive description of this pain syndrome in 1864.[1] However, since that time it has been identified by a number of other terms, such as reflex sympathetic dystrophy, posttraumatic sympathetic dystrophy, sympathetic neurovascular dystrophy, posttraumatic neurovascular pain syndrome, and Sudeck's syndrome. While many of these terms are more descriptive, they have been too long for popular adoption and only the terms causalgia and reflex sympathetic dystrophy have achieved general usage.

Most of the basic information on causalgia was derived from military, war time experiences. This disabling, burning pain, associated with incomplete penetrating nerve injuries, occurred with a frequency which ranged from 2% to 5%.[2-4] Following the report of Homans[5] in 1940, there has been increased recognition of the fact that this classic form of causalgia represents only one part of a spectrum of painful conditions involving reflexes mediated in part by the sympathetic nervous system. Far more frequently in civilian practice, one encounters a somewhat less disabling but otherwise similar pain in which there is no obvious or demonstrable nerve injury. This has been labelled minor causalgia. In an attempt to clarify the apparent continued uncertainty which surrounds this condition, 32 documented instances of minor causalgia taken from the records of the Johns Hopkins Hospital over a recent ten-year period were reviewed.

Clinical Material

A review of the medical records of the Johns Hopkins Hospital for a recent ten-year period produced 32 cases which fulfilled the clinical criteria of minor causalgia as described by Homans[5] and which were sufficiently well documented to be included in this study. During this same period, four cases which fulfilled the criteria for "major" causalgia, described by Mitchell et al,[1] were located in hospital records. There were 22 women and ten men

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Table 1.—Inciting Cause in 32 Cases of Minor Causalgia

Inciting Cause	No.
Fracture	10
Surgery	5
Sprain	4
Crush injury	4
Contusion	2
Subcutaneous injection of thiopental sodium (Pentothal)	1
Intramuscular injection	1
Third-degree burn with nerve palsy	1
Nontraumatic causes	4
Total	32

Table 2.—Presenting Signs and Symptoms in 32 Cases of Minor Causalgia*

Symptom or Sign	No.
Burning pain	23
Edema	10
Increased sensitivity to touch	9
Increased sensitivity to pressure	8
Motor function impaired	8
Muscle atrophy	8
Increased sensitivity to cold	7
Aching	7
Sensory function loss	7
Cyanosis	5
Hyperhidrosis	5
Pain on movement of extremity	2
Sharp pain	2
Cold sensation of involved extremity	1
Stiffness of involved extremity	1

* Some symptoms occurred in more than one patient.

Table 3.—Treatment for Patients at Johns Hopkins Hospital

Therapy	Beneficial Effect	No Effect
Neurolysis	1	0
Sympathetic block*	19 (temporary)† 3 (lasting)	0
Sympathectomy	24	3
Total cases (32)	29	3

* 18 patients with temporary relief are also included in the sympathectomy group.

† Prolonged in one case.

in this minor causalgia group, their ages ranging from 21 to 73 years with a median age of 46 years. The inciting events which were presumed to have led to the development of the causalgic pain are outlined in Table 1. The symptoms were referable to the upper extremity in nine cases and to the lower extremity in 23. The median delay between onset and time of treatment was two years, but it varied widely from eight days to 28 years. The signs and symptoms with which these patients presented are listed in Table 2. In addition, osteoporosis was evident in seven of the 12 patients in whom x-ray examination of the involved extremity was performed.

Nineteen of the 32 patients had already received some form of therapy prior to referral to the Johns Hopkins Hospital. Physiotherapy had been employed in seven cases, local nerve block in two, and in three instances, although stabilization was not indicated for the primary injury, immobilizing casts were applied. Two patients had undergone ligation and stripping of varicose veins without relief of their pain. Other individuals had been subjected to the following treatments without relief: excision of a lumbar disk, ankle fusion, excision of the greater trochanter of the femur, excision of a "metatarsal neuroma," anterior cervical fusion, evacuation of a hematoma, peripheral neurectomy, peripheral neurolysis, local cortisone acetate injections and treatment with phenylbutazone and diuretics.

The treatment employed after admission of the patients to the Johns Hopkins Hospital included neurolysis, sympathetic block, and sympathectomy (Table 3). Neurolysis had a lasting effect in the one case in which it was employed. Sympathetic block was performed in 22 patients, four of whom were not subjected to sympathectomy because the sympathetic block afforded lasting relief in three and a prolonged temporary relief in one. In all, 27 patients were subjected to sympathectomy and only in three of these was the procedure not effective in bringing about a gratifying relief of their pain. Two of these three patients had apparently been relieved by a prior sympathetic block. Sixteen of the 24 patients relieved by sympathectomy had had prior sympathetic block which brought relief, and in no instance of successful sympathectomy had sympathetic block been ineffective in providing relief.

The long-term response to sympathectomy was evaluated by means of a questionnaire to which 15 patients responded. The average period of follow-up in the responders was five years and seven months (range, 2 to 17 years). Of the 15 patients responding to the questionnaire, 13 had obtained either complete relief or gratifying improvement from their preoperative pain. Eight of these patients continued to enjoy the same degree of relief, whereas five had had some return of or increase in residual symptoms which began from three months to three years after sympathectomy. The residual symptoms consisted of tenderness, swelling, or color changes which were characterized as either mild or infrequent and responded to the occasional use of proprietary analgesics. However, all 13 patients still considered themselves significantly improved. Of the 15 patients answering the questionnaire, the two patients who had not been satisfactorily relieved by sympathectomy responded that their symptoms had continued unabated. However, the symptoms they described bore little resemblance to their original causalgic pain as described in the hospital chart. One claimed that the pain and swelling in his extremity was relieved by elevation and diuretics. The other described arthritic-like pain and had obtained some relief from indomethacin (Indocin) and aspirin. Surprisingly, all six cases in which compensation was known to have been involved were relieved by sympathectomy. Twelve of our 32 patients were considered to have an underlying psychiatric disturbance and yet only two of these had persisting emotional problems after their pain was relieved.

Comment

Numerous theories have been proposed to explain causalgia but none have been universally accepted. Most of these were developed to explain the causalgic pain associated with nerve injury. The most popular theory is probably that of "artificial synapses" occurring at the site of the nerve injury as first proposed by Doupe et al.[5] According to this theory, a "short circuit" occur-

at the point of partial nerve interruption or demyelinization, which allows efferent sympathetic impulses to be relayed back along afferent somatic fibers. Such an artificial synapse has been demonstrated experimentally in crushed nerves,[7] and the interruption of sympathetic efferent impulses may explain the warm, red, and dry extremity seen initially in cases of major causalgia. It has also been demonstrated that stimuli to a sensory nerve along its course make the nerve more sensitive to the usual types of sensory stimulus.[4] The work of Walker and Nulsen[8] in the human suggests the presence of this artificial synapse. Stimulation of the postganglionic sympathetics after upper thoracic preganglionic sympathectomy reproduced the causalgic pain for which the surgery was performed. No such pain was produced, however, in patients in whom the sympathectomy was performed for conditions other than causalgia. These findings have recently been confirmed by White and Sweet.[9]

One disturbing piece of evidence against this theory was the demonstration that nerve block beyond the site of nerve injury and presumably beyond this artificial synapse not infrequently afforded relief.[10] However, proponents of this theory have suggested that the efferent sympathetic impulses which are "short circuited" at the site of injury may not always be strong enough by themselves to cause a retrograde propagation of impulses and summation of these impulses together with other afferent somatic impulses may be necessary. It has also been suggested by Barnes[11] that impulses at sympathetic-sensory fiber short circuits may travel both proximally and distally. Impulses traveling proximally would then cause pain, and those traveling distally would release "antidromic" substances demonstrated by Chapman et al.[12] Release of these "antidromic" substances has been shown to lower the threshold for sensory stimuli, thus further increasing the sensory input.

While these theories are plausible enough in cases with demonstrable nerve injuries, obvious difficulties arise in extending them to explain the similar pain experienced in minor causalgia. They do not explain the sympathetic overactivity often seen in the latter stages of this condition, the relief of pain associated with intra-arterial injections of a peripheral adrenergic blocking agent, such as tolazoline hydrochloride, or the fact that in early cases the relief of causalgic pain not infrequently lasts beyond the duration of a sympathetic block by anesthetic agents. The appropriate hypothesis for the mechanism of minor causalgia must also explain the modification of pain by emotional and sensory stimuli and the relief of pain by contralateral sympathectomy after failure of an apparently adequate ipsilateral sympathectomy.[13] The hypothesis must also be compatible with the relief of pain by spinal anesthesia below the sympathetic-lumbar outlet and before sympathetic blockade response,[11] and finally it must explain the failure of sympathectomy in some of the long-standing cases.

In the late 1930's, Livingston[14] proposed that there was a "vicious cycle of reflexes" consisting of three components in causalgia. These were (1) chronic irritation of a peripheral sensory nerve with increasingly frequent afferent impulses, (2) abnormal (heightened) activity in the "internuncial pool" in the anterior horn of the spinal cord, and (3) increase in efferent (sympathetic) activity. This theory was supported experimentally by Toennie's[15] demonstration that individual stimulation of over one third of the afferent fibers of a cat's saphenous nerve resulted not only in relayed impulses cephalad from the spinal center, but impulses back down efferent fibers, including sympathetics. This theory, therefore, explains a number of characteristics of minor causalgia which cannot be explained by the "artificial synapse" theory of Doupe et al.[6] In particular it explains the high incidence of "sympathetic overactivity" in these patients, and the effect of emotional or sensory stimuli, all of which could exert their influence by heightening the background activity of this internuncial pool. It would suggest that anything that broke this vicious cycle, whether it be interruption of sympathetic efferents by spinal anesthesia or interruption of somatic nerve conduction, would relieve pain.

This latter theory enjoyed only a brief wave of enthusiasm, however, probably because it did not conform to the classic concepts of sensory perception as originally proposed by von Frey. According to von Frey, individual receptors existed for pain, touch, warmth, and cold, and these sensations involved simple transmission of a sensory impulse up a modality-specific peripheral nerve fiber, followed by relay from the spinal center to the brain via the spinophthalmic tract.[16] In recent years considerable evidence has been accumulated against this modality-specific pain transmission mechanism. Recently Melzack and Wall[17] have advanced a similar but more sophisticated explanation of pain transmission. They suggest that there are cells in the substantia gelatinosa of the dorsal horn of the spinal cord which act as a "gate control system" in modulating the transmission of afferent sensory input patterns to the neurons which are responsible for relaying patterns of impulses to the brain. These authors emphasize a pattern of impulses rather than single impulses because they feel that only a code-selection process can explain all the intricacies of sensory experience. The most important part of their concept, as it applies to the mechanism of minor causalgia, is their contention that impulses along large myelinated fibers inhibit or "close the gate," whereas tonic background impulses transmitted along small fibers (which may be afferent sympathetic fibers) tend to "open the gate" and facilitate transmission and thus increase the rate of firing of the transmission neurons in the spinal center. These authors feel that the transmission of im-

pulses above a certain critical rate is interpreted centrally as pain regardless of the initiating stimulus. Furthermore, they propose a "central control trigger" which can influence afferent conduction from the somesthetic system. Such a mechanism offers an explanation of the exacerbation of causalgic pain observed in "sympathetic overreactors" and emotionally labile individuals.[9,18-21] This theory also explains the burning pain associated with peripheral neuropathies in the process of demyelinization, in which larger fibers are diminished or absent, and it explains most of the unusual features of minor causalgia, as well as the poorly understood individual variations in pain thresholds.

The differences between major and minor causalgia may well be one of degree. It was once thought that major causalgia was primarily associated with overt penetrating nerve injury and that minor causalgia was not associated with such injuries. However, in a retrospective study of 1,500 cases of peripheral nerve injuries sustained in military combat, Echlin et al[2] found a significant incidence of minor causalgia. The overall incidence of causalgia requiring treatment was 2% in this series. From this series, of 310 consecutive patients questioned specifically about causalgia, however, 19% had symptoms typical of major causalgia at some point during their convalescence while 8% experienced transient symptoms of minor causalgia.[1] Nathan,[22] also, in a review of 160 peripheral nerve injuries found evidence of minor causalgia in 14%. In addition to those cases in which nerve injury is readily identified, minor causalgia has been reported in association with a myriad of civilian traumatic, vascular and inflammatory conditions. Animal bites, thermal injuries, including frostbite, and peripheral vascular disorders, such as phlebitis or acute arterial occlusion, have all been associated with the development of causalgia.[23,24] Thus, it is common in civilian practice to find no clearly demonstrable nerve injury.

There has been considerable discussion regarding why some patients with the same inciting causes or injuries suffer from causalgia while others do not. It has been long suspected that these patients are in some way intrinsically different. Patients with causalgic pain frequently give a history of sympathetic overactivity and careful questioning will often reveal that they have always noticed increased extremity sweating and relative intolerance to cold. Owens[20] was able to identify by history almost 70% of causalgic sufferers in his series as sympathetic overreactors, and Evans[21] likewise suggests that vasomotor instability is a common predisposing factor.

One of the most important characteristics of patients with minor causalgia is a disparity between the severity of the inciting cause or injury and the degree of pain experienced. This frequently leads physicians to suspect that the patient is a malingerer, psychoneurotic, or emotionally unstable. In reviewing the medical records of our patients, it was not unusual to encounter such impressions or more diplomatic comments. These suggested that there was "a strong psychic component" to the patient's pain or that the physician was "unable to explain the patient's complaints on an organic basis." It would be wrong, however, to exclude psychologic factors entirely as they have been shown to modify and enhance causalgic pain.[9,19] Furthermore, many of these cases were not only initially misdiagnosed or maligned, but they were frequently mistreated, as evident by the misdirected therapeutic endeavors, several involving major surgery, to which patients had been previously subjected without relief.

The need to improve the ability of most clinicians in recognizing minor causalgia seems apparent. Many painful conditions of the extremities cannot properly be called causalgia, although frequently they are confused with this syndrome. Pain secondary to herpes and other forms of neuritis, spinal cord lesions such as poliomyelitis, tabes, combined degeneration and syringomyelia, thalamic pain, cortical lesions, Morton's toe neuroma, glomus tumor, cauda equina injury, postsympathectomy neuralgia, arachnoiditis, arterial insufficiency, phantom-limb pain, pain associated with Pancoast's syndrome, periphlebitis, and hand-shoulder-arm syndrome must be distinguished from causalgia if appropriate therapy is to be selected.[9,10,25] The similarity of the presenting symptoms in these conditions and minor causalgia renders diagnosis both more difficult and more important.

Certain common, although not universal features of minor causalgia, however, should be kept in mind in this regard because once this diagnosis is entertained it can be confirmed readily. In the most severe and prolonged cases, the pain may be localized poorly and even spread up the extremity, but initially it is well localized by the patient and is described as a superficial skin pain rather than a deep pain. When localized, the pain often falls within the peripheral distribution of a single sensory nerve, although it rarely involves its entire distribution. The hyperesthesia is very characteristic. Even stroking the skin or gentle pressure in the involved area may elicit severe discomfort. This hyperesthesia and the absence of demonstrable neurologic deficit are in sharp contrast to most peripheral neuropathies presenting with burning pain. Furthermore, whereas ischemic rest pain may have a similar burning character, initially it occurs only at night and is relieved by dependency. Causalgic pain is also more constant, even though its severity may be modified by emotional factors and by thermal or almost any other sensory stimulus. Eventually signs of autonomic imbalance in the involved area will become evident as either sympathetic overactivity or underactivity. It was formerly thought that the extremity affected with major causalgia was warm, red, and dry and that the extremity affected with minor causalgia was cool, pale or cyanotic, and moist. These conclusions ap-

pear to have been derived from observation of major and minor causalgia at different times following injury, since major causalgia is typically diagnosed and treated earlier than minor causalgia. This temporal difference in presentation has been observed in our series. The delay prior to diagnosis and treatment of four cases of major causalgia was three months, but in our 32 cases of minor causalgia the average delay was two years.

Associated trophic changes also vary in time of appearance and further serve to preclude a uniform clinical presentation for causalgia. Drucker et al[23] have suggested a time course for these vascular and trophic changes involving three stages. In the "initial" stage he described increased blood flow and temperature, "soft" edema, sweating, and disuse atrophy. In the second or "dystrophic" stage, coolness, pallor or cyanosis, "brawny" edema, hair loss, and brittle nails were characteristically observed. The changes in these first two stages were considered reversible. Finally, some patients reached an "atrophic" stage with irreversible changes, including decreased dermal flow, atrophic, shiny skin, and loss of subcutaneous fat. Although this may represent an oversimplification, the pattern outlined may be helpful in appreciating the variation in clinical signs which may be encountered in extremities involved with causalgia. Drucker also pointed out that in the final stage, the pain, like the trophic changes, might be irreversible.

Sudeck described a patchy osteoporosis in association with inflammatory changes around injured joints at the turn of the century and until 1938[26,27] maintained that this osteoporosis was caused by inflammatory changes, rather than the trauma itself or increased vascularity. Osteoporotic changes were confirmed by x-ray film examination in seven out of the 12 cases studied in this series and the reported incidence of this feature varies considerably on either side of 50%.[19,19,21] These changes cannot always be explained on the basis of disuse or immobilization. Good evidence has been presented, however, to show that the same vascular changes which are so obvious in the skin may also be responsible for the bony abnormalities.[19]

From a therapeutic point of view, the most important consideration is that the pain of minor causalgia can be relieved by pharmacologic or surgical sympathetic block. Owens[20] has observed that the intra-arterial injection of a small amount of tolazoline hydrochloride (10 mg) usually relieves causalgic pain in the extremity served by that vessel, and, in his experience, this has proven to be a useful screening test. He noted that in 34 of 38 cases relief of pain was obtained by this procedure. Furthermore, in four patients, pain was permanently relieved by this measure. Only two out of 29 cases, however, could be managed by oral administration of tolazoline alone. Others have reported favorable therapeutic results with trigger-point injection, rhizotomy, cordotomy, and resection and resuture of the involved nerve.[14,28-30] Neurolysis has been found to be effective in selected instances, but sympathetic block together with sympathectomy is generally the recommended form of treatment.[9,31] Although it has not been our experience in civilian practice, physical therapy and exercise have also been shown to bring relief from causalgia in significant numbers of military personnel.[32]

In our experience, paravertebral sympathetic block has been found to be the most reliable diagnostic test. White and Sweet[9] believe that in cases of sympathetic dystrophy (minor causalgia) a diagnostic sympathetic block is extremely useful. In patients who are overly suggestive or in whom the diagnosis is suspect, a control block with normal saline solution may clarify the diagnosis. In earlier, mild cases of minor causalgia, the relief may last beyond the duration of the block, and if this occurs, one should persist with intermittent blocks until either the pain is relieved or the frequency of need for such blocks is seen to be static or increasing. Relief was obtained in this fashion in 10% of our cases, and others have had similar success.[9,18,21] This is particularly true in early cases. In cases in which the duration of the relief of pain is equivalent to that of the sympathetic block, or where the need for sympathetic block is static or increasing, one should proceed with sympathectomy. This should only be done, however, if the symptoms are sufficiently debilitating and the inciting factor is static. It is important, however, not to wait too long, for fixed pain patterns and trophic changes may become established and refractory even to sympathectomy.

The results of sympathectomy for major causalgia have been excellent, possibly because these cases have been diagnosed and treated early. Mayfield,[33] from his World War II experience, reported that only two of 75 patients with major causalgia had a return of significant pain, initially. Of the 73 patients who were relieved of pain, 63% remained free of pain for five years and the other 37% had minor enough residual complaints to be considered significantly improved. Other authors report similar experiences.[2,3,24,25] The results of sympathectomy for minor causalgia have been almost as good. Twenty-six of 31 in Drucker et al's[23] series, 32 of 34 in Owens'[20] series, 10 of 12 in Hardy et al's[21] series, 3 of 4 in White and Sweet's[9] series, and 24 of the 27 patients in this series who were subjected to sympathectomy for minor causalgia were either totally relieved or significantly improved. In our experience, the late return of symptoms, which occurs in approximately one third of patients, does not significantly detract from the overall satisfactory result. In general, failure to achieve satisfactory long-term relief from causalgia can be attributed to one of three factors: (1) incorrect diagnosis, (2) a long delay between the onset and treatment, or (3) either incomplete sympathectomy or collateral reinnervation.

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