THE YEAR BOOK of DENTISTRY

(*1960-1961* YEAR BOOK *Series*)

YEAR BOOK MEDICAL PUBLISHERS
INCORPORATED
200 EAST ILLINOIS STREET
CHICAGO 11

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DIAGNOSIS

Facial Neuralgias are discussed by Henry Cohen.[1] Wherever a sensory nerve is stimulated, the resulting pain is referred (localized) to the peripheral distribution most commonly of the same nerve but sometimes of a related nerve. The latter is properly labeled "referred" pain, and it arises because of a shared sensorium. There are only two qualities of pain, one superficial and the other deep. Superficial pain may be "bright" or "pricking" from short stimuli and "burning" on more prolonged stimulation. Deep pain usually has a dull aching quality, is ill localized and is often accompanied by disturbances of pulse and respiration, and nausea. However, all gradations from superficial to deep pain may occur.

The term "neuralgia" should indicate not simply a pain, but pain corresponding to the known anatomic distribution of a nerve. Two types of neuralgia are recognized. The first is due to a gross pathologic lesion, such as neoplasm, scar or infection directly involving a nerve. This type is best termed "symptomatic" or "secondary" neuralgia. Primary neuralgia occurs in the absence of obvious gross disease. The two types can usually be differentiated clinically. Secondary neuralgias commonly show a mixture of superficial and deep pain. Pain is usually continuous. Objective signs of interruption of nerve continuity, such as anesthesia, paresis and muscle wasting, are often noted and tend to involve contiguous areas. Primary neuralgias are associated with paroxysmal pain lasting only seconds, with complete or almost complete remission between spasms. There are no objective signs of nerve involvement, and spread to other areas is rare. Spontaneous remissions are common, and this has given rise to groundless claims for the effectiveness of a host of drugs and other measures.

Trigeminal neuralgia is the most common and important facial neuralgia. It must be recognized early to avoid unnecessary dental treatment. No dental or oral condition accurately simulates trigeminal neuralgia, and it is never permanently relieved by dental treatment. Treatment by

(1) Brit. D. J. 107:9-18, July 7 and 21, 1959.

sensory root section should not be delayed, as in advanced cases the instability of the nerve cells of the higher stations may have markedly increased, so that sensory root section will not cure the pain. Trigeminal neuralgia is almost always accompanied by pain in the tongue, which helps differentiate it from other lower jaw pain.

The atypical neuralgias form a most important group. Psychogenic factors appear to play a major role in etiology. Pain appears to have no organic basis or else there is a gross overreaction to very minor disease, which has long since been corrected. Surgical procedures not only fail to relieve, but invariably aggravate, the pain. Affected patients are usually women over 40 and commonly menopausal. The pain is described in picturesque and extravagant terms. It is never of the tic type but is more constant, dull and boring. It often spreads over large areas, and trigger zones are innumerable. Previous dental treatment or a slight facial injury is usually blamed for the pain. This pain usually incapacitates the patient for business or domestic duties, in contrast with true tic, with which the patient usually carries on his or her occupation. Psychogenic atypical neuralgia must not be the last refuge of the diagnostically destitute. Every effort must be made to exclude an organic basis and the diagnosis of psychogenic atypical neuralgia be accepted only if there are positive signs of mental instability.

► [Cohen has lucidly described the facial neuralgias in a manner that should aid the dentist in diagnosis. The reaction of a given individual to pain is fascinating. Sonis (J. D. Med. 15:85, 1960) has described children who showed no emotional signs when subjected to injuries that produce severe pain and pointed out that others may over-react, as judged by the dentist or the physician. The reaction of the dentist who is requested to extract a tooth without the use of an anesthetic exemplifies our tendency to be "Godlike, omnipotent, all giving and all forgiving, completely understanding and comforting" instead of recognizing ourselves as practicing an art which should include the art of human relations and recognizing oneself as human and not Godlike. Such self-understanding and whole-patient approach is essential in diagnosis of oral and facial pain.—H.B.G.R.]

Atypical Face Pain. Joseph G. Rushton, Joseph A. Gibilisco and Norman P. Goldstein[2] (Mayo Clinic and Found.) studied 100 patients who complained of deep, poorly localized and vaguely described facial pain. Symptoms could not be ascribed to trigeminal, glossopharyngeal or postherpetic neuralgia or to obvious diseases of the teeth, nose, throat,

(2) J.A.M.A. 171:545-548, Oct. 3, 1959.

sinuses, eyes or ears. It was possible to classify all conditions under one of three heads: psychogenic, organic or indeterminate.

In 53 cases the pain was finally ascribed to a psychiatric illness, such as depression, hysteria or schizophrenia. Each of these patients complained primarily of facial pain. This group consisted of 46 women and 7 men. Most were in the 4th and 5th decades. Thirty-three were unable to recognize any cause for the pain. The other 20 blamed dental operations, injuries or infections. These patients had pain of an unusual character and unanatomic distribution and prominent symptoms indicating emotional illness. Evidence of an organic cause was lacking.

In 33 cases an organic cause for the pain was finally determined. This group included 14 men and 19 women. Causes included vasodilating face pain, dental disease, neuritis, neoplasms and miscellaneous causes. Eight patients had pain as a result of dilation of the facial arteries. This pain closely resembled histaminic cephalalgia, but localization was unusual. Injection of ergotamine tartrate relieved the pain. In 8 other patients unrecognized dental disease caused the obscure facial pain. In 5 the final diagnosis was pulpitis, and relief was obtained by extraction. Three had causalgia of the mandibular nerve after extractions. All efforts to relieve this pain failed. In 8 cases neuritis of some portion of the trigeminal nerve was considered to be the etiology. This often followed some injury, and pain was limited to the branch of the nerve presumed injured. Each patient had a slight but definite loss of sensation in the affected region. Three had neoplasms that caused unusual pain. Two patients had adenoid cystic carcinoma, 1 in the parotid and 1 in the antrum. Neither tumor was detected until late, despite repeated examinations. The third patient had a neurofibroma of the acoustic nerve. Six patients had various organic lesions, and facial pain was considered to be an unusual manifestation of the diseases.

In 14 cases neither psychiatric nor physical causes could be identified and etiology remained undetermined. In general, these patients complained of pain of shorter duration than that in the other two groups. They also tended to be older. Remissions were common, and pain could often be

made worse by chewing, rubbing the face or jarring the head. Most obtained substantial relief from ordinary analgesics. There was no reliable evidence to indicate a psychogenic origin, and the character of the pain suggested an organic basis.

The term "atypical face pain" should be used as the name of a symptom in the same manner as the terms "fever" or "convulsion" are used. These pains do not have a common cause. The designation should be used only when a definite diagnosis is impossible. Surgical treatment is not indicated. Cautious conservative treatment and continued observation are the best means for care of these patients.

► [Histaminic cephalalgia which produces severe unilateral headache, facial pain, inflammation of the eye, rhinorrhea and blocking of the nose is described by Kresne (J.A.D.A. 59:447, 1959). It usually occurs when the patient is asleep and pain lasts for 15 minutes to several hours. The dental profession should be aware of the various facial pains and aid in this diagnosis.—H.B.G.R.]

Syndromes of Head and Neck of Dental Origin: I. Pain Caused by Mandibular Dysfunction. Harold Gelb and Godfrey E. Arnold[3] (New York Eye and Ear Infirm.) studied 100 patients with otolaryngologic disorders of dental origin. The ratio of women to men was 3.5:1. Most of the patients complained of pain in the head and neck region. A small group (25%) complained of subjective ear noises, such as tinnitus, crepitus or clicking in the region of the mandibular joint. All these patients were first carefully examined to rule out possible ear, nose or throat disease of local or systemic origin.

The commonest sites for pain were the temporomandibular joints and the internal pterygoid muscles. The pain could usually be traced to certain "trigger areas" in the muscles of mastication. Treatment was focused on physiologic prosthetic management of maxillomandibular dysfunction. Most patients presented with malocclusion. Premature contacts or other traumatogenic factors were relieved. Definite trigger areas for muscle spasms could usually be palpated. These areas were sprayed with ethyl chloride or injected with a local anesthetic or normal saline. Injections were performed with a Luer-Lok syringe using a 21-gauge needle that permitted aspiration before injection.

(3) J. D. Med. 14:201-212, October, 1959.

Temporary mandibular occlusal splints were often used in conjunction with myofunctional therapy. Favorable results were obtained in some patients by simply correcting the occlusion by use of splints or equilibration in conjunction with myofunctional therapy. Others required a combination of ethyl chloride spray or muscle injection plus splinting and myofunctional therapy.

In those who complained of subjective ear noises, jaw relationships were corrected by temporary acrylic splints. About half of these patients noted subjective improvement or became symptom free.

If the patients remained symptom free for several months, they were referred to their dentists for permanent restorations. Although only 7 cases of bruxism were reported, many of these patients clenched, ground or gnashed their teeth. Most of the patients in this series were unduly nervous or tense.

Costen's syndrome does not occur uniformly in temporomandibular joint dysfunction. The various symptoms of this syndrome can occur singly or in groups. Treatment by physiologic prosthetic management gave a high percentage of successful results.

► [This article and that which follows show the value of occlusal adjustment but point out its limitations. Schwartz (New York D. J. 25:391, 465, 1959; Disorders of the temporomandibular joint, Philadelphia: W. B. Saunders Company, 1959) emphasizes the trend toward recognition of temporomandibular joint function as equivalent of that of other joints with the neuromuscular mechanism directing it. He claims that the incidence of organic disease is low in this region but painful spasm of muscles is prominent.—H.B.G.R.]

Mandibular Joint Pain: Survey of 100 Treated Cases is presented by Hamish Thomson[4] (Eastman Dental Hosp., London). A control group of 100 patients who did not have mandibular joint pain was also studied. A detailed history was obtained from and examination performed on each patient. Patients were aged 12-75. In the pain group the ratio of women to men was 3:1 and in the control group, 2:1. In the pain group 40% of the women were aged 18-30.

Mandibular joint pain was manifested by one or more of six symptoms. A dull ache was one of the most common complaints. It was usually unilateral, came and went without known cause and usually lasted 1-2 hours. Other symptoms

(4) Brit. D. J. 107:243-251, Nov. 3, 1959.

included limitation of opening, pain on mastication, pain on wide opening and stiffness in the jaw on awakening. Pain to finger pressure over the condylar regions when the teeth were in occlusion was noted by 63% of the pain patients. Symptoms in this group were predominantly unilateral. Only 13 patients had only one symptom. Most had two or three of these symptoms, and 1 patient experienced all six.

Functional analysis of occlusion was performed in each case. This included determination of the rest position, freeway space, deviations on closure, facets of wear and analysis of the side used in function and of the posterior tooth support.

Treatment of the patients with pain was generally carried out in two stages, initial and permanent. Initial treatment in 61 consisted in diagnostic splints or acrylic resin bite plates. These were designed to restore the occlusal level of contact at 2-4 mm. upward and forward of the rest position. These splints were worn for up to 3 months and were then removed for a week. If pain recurred, the splints were replaced and permanent restorations were planned. Initial treatment in 39 patients consisted of selective grinding, correction of occlusion on existing dentures or construction of new dentures or appliances to prevent grinding habits. Alteration of occlusion was initial treatment in 80%, and 96% of these patients improved. Permanent treatment in 46 patients consisted of fixed bridges, partial dentures, overlay appliances, orthodontia or operative dentistry. Of these 46 treated patients, 22 have shown no recurrence in 4 years. Thirteen patients were symptom free for 6 months but were not seen subsequently. The other 11 had occasional recurrence of pain while continuing to wear the appliances.

In the pain series corrections of defects in occlusal relations was successful in relieving pain in most patients. Many believe this indicates that mandibular joint pain is caused by these occlusal defects. However, in this study displacement of the mandible on closure, tooth-grinding habits and lack of posterior support had a similar incidence in the control group and in the group with mandibular joint pain. It appears that occlusal defects are not the main causative factors in mandibular joint pain, although they may contribute.

Study of the symptoms suggests that the initial lesion is irritation of the tissues directly related to the joint, which