

SPHENOPALATINE GANGLION NEURALGIA

WATT W. EAGLE, M.D.

DURHAM, N. C.

In the early part of this century the late Greenfield Sluder first directed attention to a syndrome which he called sphenopalatine ganglion neuralgia, or lower half headache. The novelty of this disease drew considerable attention, and the enthusiasm continued for several years, reaching a maximum in the early twenties. Then the interest subsided. The main reason, apparently, was that various bodily ailments, many quite remote, were attributed to irritation of the sphenopalatine ganglion. The syndrome had become a "dumping ground" for many otherwise undiagnosed conditions. This statement is easily substantiated by a glance at the literature of that period. As a result, many clinicians began to doubt the existence of the disease; and to this day some internists, neurologists and specialists in various other fields refuse to recognize the disease and to accept it as a reality. Textbooks more or less ignore the disease. Less than half of the otolaryngologic texts give any appreciable space to the syndrome, and the others barely mention it. Neurologic texts also give it the minimum space.

It is the purpose of this paper to discuss those unilateral facial pains which are considered to be justifiably diagnosed as sphenopalatine ganglion neuralgia. It is essential to review briefly the anatomic aspects of the ganglion itself.

The sphenopalatine ganglion (also called Meckel's ganglion and, in the old nomenclature, nasal ganglion) lies suspended, so to speak, from the maxillary branch of the trigeminal nerve (fig. 1). It is medial to the maxillary nerve and just distal to the semilunar ganglion. It transmits motor, sensory and sympathetic fibers, together with a few sensory neurons. A root known as the vidian nerve is formed by the union of the great superficial petrosal nerve and the great deep petrosal nerve. The fibers of the former, or great superficial petrosal nerve, have their origin in the medulla and are transmitted through the geniculate ganglion of the seventh nerve. The fibers of the great deep petrosal nerve are mainly sympathetic in nature and are simply extensions of fibers of the carotid plexus. The vidian nerve traverses the pterygoid canal and enters the sphenopalatine fossa.

From Duke University Hospital and School of Medicine.

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The second root of the ganglion is the so-called sensory root and is composed of two or three trunks originating in the maxillary branch of the trigeminal nerve. A few motor neurons are also thought to be included in these sensory trunks. All of these fibers are distributed to the nasal membranes, to the soft palate and to other portions of the pharynx and are associated with gustatory and other sensations.

The ganglion lies in the sphenopalatine fossa, which is quite adjacent to the sphenoid and posterior ethmoid paranasal sinuses, and it may vary slightly in its position. However, the ganglion always lies just behind the posterior end of the middle turbinate, regardless of its relation to the other structures. The sphenopalatine foramen is large in most instances. This facilitates local application of cocaine during diagnostic procedures or treatment.

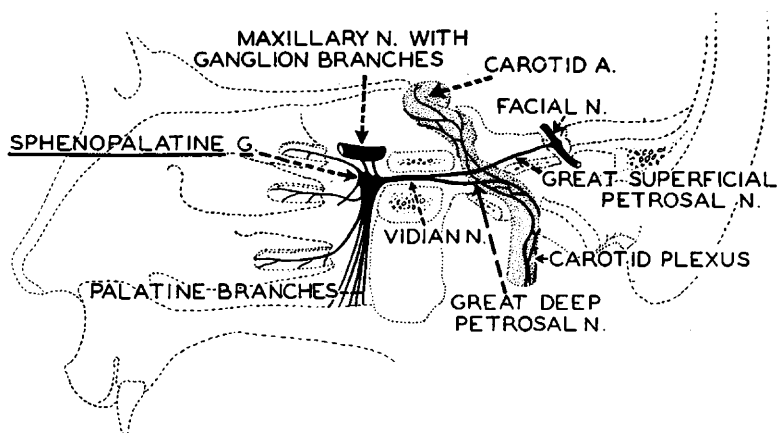


Fig. 1.—Schematic illustration of the right sphenopalatine ganglion and its branches.

*Incidence of Sphenopalatine Ganglion Neuralgia in One Hundred and Fifty-Nine Cases **

Age	Per Cent
20 to 30	20
30 to 40	38
40 to 50	20

* This neuralgia occurs in females in the ratio of 2:1. It occurs almost entirely in members of the white race.

INCIDENCE

There seems to be a preponderance of this disease in patients of the ages between 30 and 40. Of 159 patients observed in a five year period, from 1934 to 1939, 38 per cent were in this age group, with 20 per cent each in the twenties and in the forties (table). There was further incidence of the disease in patients varying in age from the later teens to the seventies. The disease occurred in females in a ratio of

2:1, there being 108 female and 51 male patients. There seems to be no definite seasonal incidence, although a study of this series of 159 cases showed May and September to be the months when my associates and I first encountered the greatest number (fig. 2). There seems to be no definite association with allergy, which is borne out by the fact that we saw very few patients who were allergic. The menstrual cycle also seems to have no association; however, menopausal symptoms may be associated with this condition.

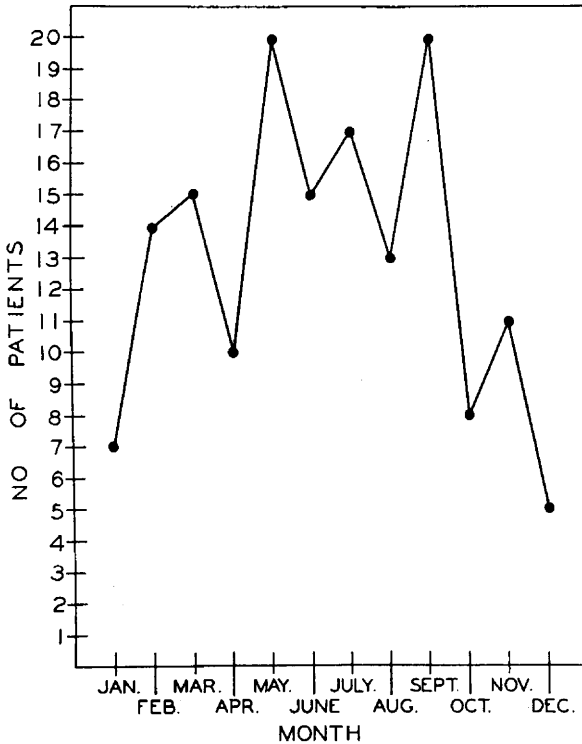


Fig. 2.—Seasonal incidence of neuralgia of the sphenopalatine ganglion in 159 cases (based on the time when the patient was first observed).

Sphenopalatine ganglion neuralgia seems to be almost entirely restricted to the white race. Our group of patients contains only 2 members of the colored race. We had no patients of foreign extraction, but this statement bears no real information, since in North Carolina one sees patients of foreign birth rarely. The disease occurs in persons in the upper strata of social life, the so-called white collar group. Only 5 patients out of the group observed were seen in the dispensary. Our records show that 129 were married, while 30 were single. Practically all of the latter were females.

ETIOLOGY

In the past, practically all irritations of the sphenopalatine ganglion were attributed to an infection or hyperplasia of the lining membrane of the sphenoid or the posterior ethmoid paranasal sinuses. In analyzing our cases, we note that intumescence of the nasal membrane is the immediate cause of the stimulation of the ganglion, and we recognize three primary etiologic factors for this intumescence: first, intranasal deformities; second, systemic disorders, including toxemia, anemia and fatigue, and third, primary neurosis, hysteria and other emotional manifestations.

The first and most common of these exciting factors is that of intranasal deformities. These deformities include deviations of the nasal

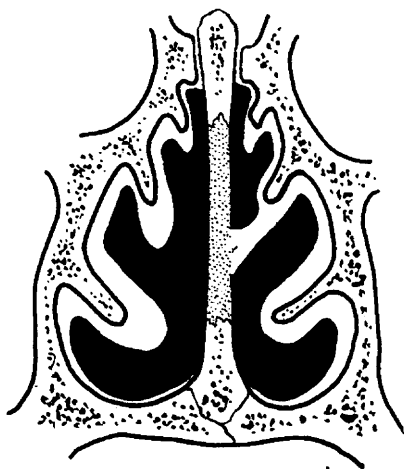


Fig. 3.—Schematic drawing of an adhesion between the left middle turbinate and the nasal septum. Such a condition usually follows trauma or is postoperative. The adhesion causes more intense symptoms if it is located on the posterior portion of the middle turbinate.

septum, ledges, spurs, prominent turbinates, adhesions (fig. 3) and osteomas (fig. 4). Actual contact of the septum and the middle turbinate have been seen often, either when there is a prominent middle turbinate or when the septum deviates against the middle turbinate (figs. 5 and 6). More frequently, however, a ledge has been found, originating at the vomer inferiorly, extending up and back and ending in a prominence or spur which projects into the middle meatus. This spur invariably touches the middle turbinate (fig. 7). One may find a spur which has divided the middle turbinate into two portions, causing the turbinate to resemble a peanut hull. It has long been recognized that spurs and other nasal deformities are likely to cause frequent and

severe infections of the upper respiratory tract. Eighty per cent of our 159 patients were found to have an intranasal deformity involving contact with the middle turbinate. The nasal bones and cartilage cease

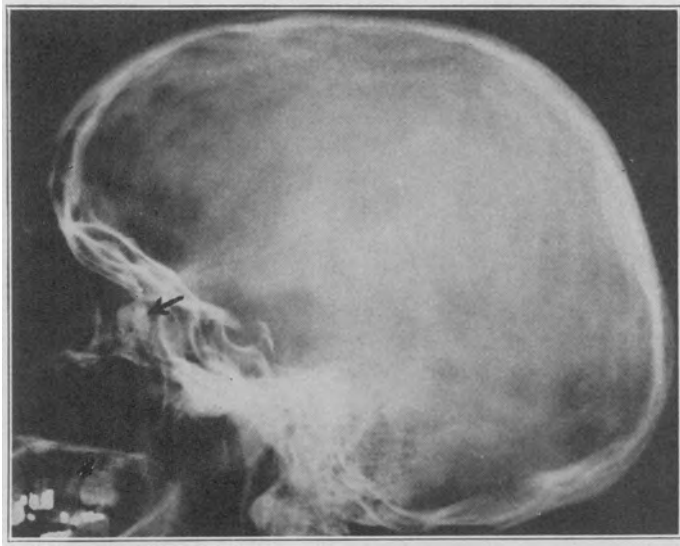


Fig. 4 (case 3).—Osteoma in the left posterior ethmoid sinuses. This patient had excruciating pain for three years and was relieved completely on removal of the osteoma.

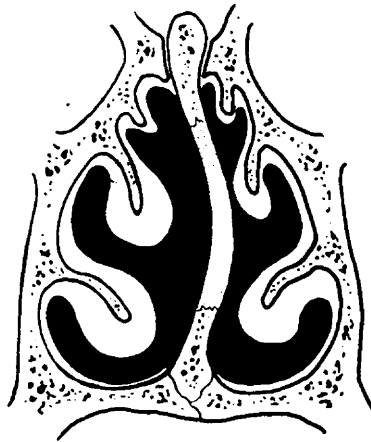


Fig. 5.—Schematic illustration of a septal deviation meeting the middle turbinate. Approximation of soft tissue probably does not cause irritation of the sphenopalatine ganglion, but bony structures touching are likely to incite symptoms.

growth and adjustment at about the age of 16, and it is difficult to explain why symptoms do not appear until years later.

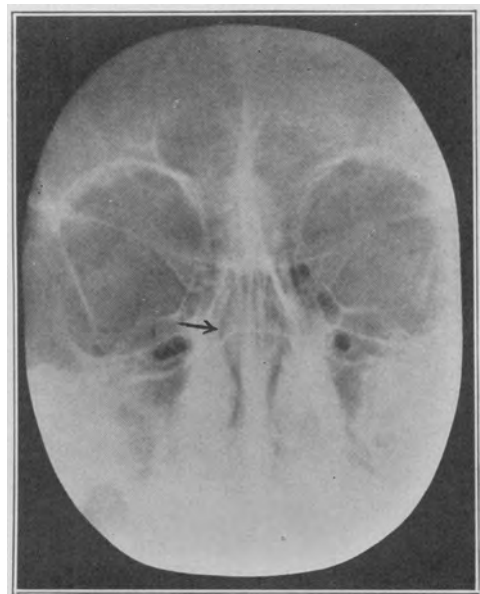


Fig. 6.—Roentgenogram, showing an ordinary septal deviation with pressure phenomena caused by pressure against the right middle turbinate in its posterior portion. Also shown is a cartilaginous deviation to the left anteriorly, which caused no symptoms whatever.

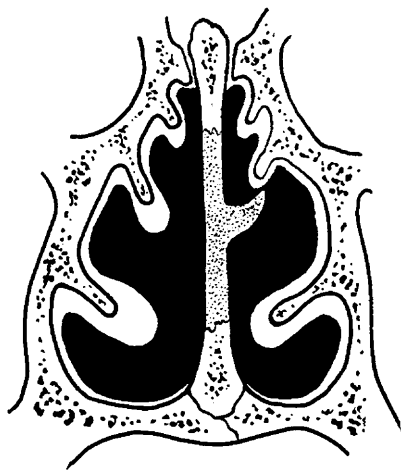


Fig. 7.—Schematic illustration of a septal ledge ending in a spur rather far back in the middle meatus and causing contact with the middle turbinate in its posterior portion. This condition is one of the more commonly found causes of irritation to the sphenopalatine ganglion.

One must recall that the respiratory function of the nose entails the contraction and swelling of the membranes of the turbinates and septum, especially those of the turbinates. This explains why patients may note the onset of sphenopalatine ganglion neuralgia with sudden changes of weather and humidity and with the passing from cold or chilly rooms to warm or well heated rooms or vice versa. The onset may be traced to the presence of acid fumes, dust, pollen, smoke, soot and grime in the air. All of these conditions are likely to cause intumescence of the nasal membranes, thus causing intranasal pressure symptoms. Emotional stimulation also may cause swelling of the intranasal membranes. An osteoma of a paranasal sinus may cause irritation of the sphenopalatine ganglion. Foreign bodies have not been observed.

The mechanism of excitement of the sphenopalatine ganglion has not been determined fully, but most probably the condition is due to irritation of branches of the ganglion supplying the middle turbinate. This is thought to be true, especially since in the majority of cases of intranasal deformity the middle turbinate is involved. Excitement of the ganglion also occurs when a too radical surgical operation on the middle turbinate has been performed. In persons who have had such operations, there is a replacement of the ciliated mucosa with considerable scar tissue, and there is a scarcity of mucosa of a secretory nature. Apparently, the nerves from this ganglion are included in scar tissue as healing occurs, and pronounced neuralgia of the ganglion ensues. Some observers consider ischemia of the nasal membranes, due to any irritation, as the cause of the pain. If this were true, allergic patients should be very susceptible to the pain, but, as stated previously, few patients with allergic disease were found to have sphenopalatine ganglion neuralgia.

Certain persons who have no intranasal deformity demonstrate a pronounced neuralgia of the sphenopalatine ganglion; and furthermore, these persons are quite stable emotionally. The neuralgia probably has persisted only a short time and followed an infection of the upper respiratory tract with subsequent infection of the sphenoid or the posterior ethmoid sinuses. As pointed out, the ganglion is adjacent to these paranasal sinuses, and by contiguity, edema of the membranes and spread of the toxins may occur. More numerous are the cases in which the posterior ethmoid and the sphenoid sinuses are seen to be perfectly clear by clinical and roentgen examinations and there have been no symptoms relative to these sinuses. Such cases demand further search for infections producing the intoxication. One must suspect the tonsils, teeth, gallbladder, intestinal tract and other structures as foci of infection until each is proved innocent. One, however, is rather likely during the search to find a definite infection. Extraction of abscessed teeth may cause an immediate and considerable increase in the severity

of the facial pains. Removal of the tonsils or stirring up of any other focus of infection is likely to increase symptoms for a short period, after which a gradual but complete restoration of normal nasal function and sensation occurs. Anemia, endocrine disorders and other generalized debilities in which the patient easily becomes fatigued may result in susceptibility to attacks of the disease. Overwork, excessive reading or reading under poor illumination and various excesses may bring on attacks.

The third cause of the disease is illustrated in all those patients who demonstrate psychoneurotic or hysterical tendencies and are otherwise emotionally unstable. At times the patients in this group may give clearcut information, and the examiner is led to believe that they may have an organic basis for their complaints. They have visited numerous physicians. They like to read medical books and journals, and they are well versed in medical terms. The examiner must obtain the confidence of these patients, and soon he may suspect some difficulty in their personal and domestic affairs. Otherwise, he may continue the search for the cause of the trouble over long periods. Rarely do these patients have an attack of pain during a visit to the physician. There are numerous conditions which may lead to their emotional instability, such as worry over health and wealth, fear of pregnancy, domestic incompatibility, jealousy and depressive reactions. These patients should be referred to a psychiatrist.

SYMPTOMS

The most important point of all to remember regarding sphenopalatine ganglion neuralgia is that it presents a unilateral facial pain and not a headache. As the patients relate the symptoms, they frequently speak of a headache, but on questioning they will in each case admit that they are experiencing a pain rather than a headache.

A typical history of sphenopalatine ganglion neuralgia includes all or part of the following: There is a unilateral pain in the face, which never extends above the level of the ear. The pain is usually lancinating and continuous; it may last from a period of a few minutes up to several days at a time, recurring after no special interval and always more or less repeating the previous episode. (In females no increase of symptoms is noted during the menstrual cycle.) There are usually two main points of maximum pain, one in the region of the orbit and the root of the nose and the other in the region just posterior to the mastoid process in the temporal bone. In some patients only one of these points of maximum tenderness is present, that more likely being the region of the orbit and the root of the nose. If both points of intense pain are present, the pain seems to originate in the root of the nose and to involve the orbit, causing extreme soreness in the eyeball, and

to extend back through the eye into the region of the ear, frequently causing earache, a sensation of fullness in the ear and tinnitus. From there the pain extends straight back to a point about 5 cm. behind the auditory canal and mastoid process. Occasionally, the pain then extends on into the neck, sometimes involving the shoulder blade but more often the top of the shoulder. In extreme cases the pain extends from the top of the shoulder to the elbow and in a few cases on into the finger tips, involving usually the middle and index fingers. During the attacks vasomotor changes have occurred intranasally, producing swelling of the nasal membranes on the involved and painful side, in addition to the outpouring of mucoid and serous nasal secretions. Naturally, there is impairment of the breathing space on the involved side of the nose. Tinnitus with dizziness, indicating obstruction of the eustachian tube, is infrequently described. Occasionally, there may be pharyngeal symptoms, to some extent resembling glossopharyngeal or ninth nerve neuralgia. All the upper teeth on the affected side may become sensitive, and there may be some discomfort of the lower teeth. In addition to facial pain, burning, stinging and tingling sensations have been noticed, especially in the skin over the lower part of the jaw. Nausea and vomiting are notably absent but if present are probably due to increased postnasal secretions in those patients who have hyperactive gag reflexes and in whom gastritis develops easily.

DIAGNOSIS

The diagnosis is conclusive if the symptoms are relieved within one to three minutes by cocainization of the sphenopalatine ganglion on the affected side. When periods of cocainization of more than three minutes fail to alleviate the pain, the presence of sphenopalatine ganglion neuralgia is not likely. The application of 10 per cent cocaine directly on the mucosa over the sphenopalatine ganglion is easily done in all cases unless some extreme intranasal deformity prevents. However, applicators can usually be so curved as to get around these abnormalities. Unless cocaine can be applied to the region of the ganglion and the pain thereby relieved, a diagnosis is not certain. Headaches and pains of central origin or due to a neural lesion medial to the ganglion will not be relieved by the application of cocaine. Pains from migraine, brain tumors and cervical arthritis are not relieved by cocainization of the ganglion; nor is relief obtained for headache due to alcoholic cephalgia or to gastrointestinal disturbances, such as disease of the gallbladder, gastritis and constipation. The ocular fundi are normal. Laboratory studies give little information and yield negative results except in those cases in which irritation is due to causes of the second type—toxemia and other forms of infection. In no case did my associates and I find

a positive Wassermann or Kahn reaction, indicating syphilis, which is not a probable factor. Roentgenograms may demonstrate intranasal deformity or dental and sinus infection.

Differential Diagnosis.—It is to be remembered that in neuralgia of the sphenopalatine ganglion one is dealing with a unilateral disease. Therefore, the differential diagnosis is much easier, and there is not a large list of differentials. Migraine naturally is easily confused in some respects, but it is a unilateral disease in which the patient suffers actual headache, while in neuralgia of the sphenopalatine ganglion the patient suffers pain. Furthermore, a history of a familial tendency is obtained frequently from patients with migraine, and the disease is characterized by more or less periodic headaches, at which time the patient notes prodromal symptoms, such as halos about objects looked at and scintillating effects in the field of vision with considerable scotoma and blurring of vision, as if a semitransparent screen were drawn over the field of vision. Frequently, the patient suddenly sees only half of the object looked at. There may be nausea and vomiting. The headache is of the throbbing type. The patient is more or less incapacitated in every respect, while sphenopalatine ganglion neuralgia is not in most cases so incapacitating. The use of gynergen is essential in the diagnosis of migraine. While it will have no effect whatever on sphenopalatine ganglion neuralgia, this product is used efficiently in treating migraine. Thiamine hydrochloride has not been used extensively for this condition, and so far reports have varied as to the results produced. Our observation of a very few patients has failed to show any benefit; however, avitaminosis may yet be proved a causative agent of sphenopalatine ganglion neuralgia.

Trifacial neuralgia, involving mainly the middle (or maxillary) branch, should not be confused with sphenopalatine ganglion neuralgia, because in the former condition there is usually a trigger point for the origin of the symptoms, and the pains, although extremely excruciating, are lightning-like in duration. In case the ophthalmic or mandibular branches are involved, the diagnosis is clearcut, since the region involved is not that frequently observed in sphenopalatine ganglion neuralgia.

Dental abscesses, impactions and pulp stones may cause some difficulty in a differential diagnosis, because frequently unerupted teeth or infections involving the gingival margins or even the alveolar processes do not localize pain definitely at any one point and therefore may cause pain throughout the entire maxillary region. Roentgenograms should give conclusive evidence of any dental involvement.

Dental extractions have frequently and erroneously been performed in an effort to alleviate sphenopalatine ganglion neuralgia because it was suspected that the teeth were abscessed, just as extractions have

occurred myriads of times in cases of trifacial neuralgia. Because of these extractions some patients demonstrate the temporomandibular joint syndrome in addition to sphenopalatine ganglion neuralgia.

Acute infection of the maxillary or ethmoid sinuses may cause considerable irritation of the sensory nerves of that region, and this is especially true when the infection is in the hyperemic stage, previous to the onset of the purulent disease. As the purulent stage of the disease progresses, one finds the patient to suffer more headache than pain, and the headache is localized in the region of the sinuses involved. Furthermore, the headache is of a throbbing type and can easily be increased in severity by percussion over the involved sinuses or by movement in which jarring occurs and, finally, by leaning the head forward, thereby increasing the flow of blood to the inflamed tissues.

Intracranial tumors and other lesions may cause unilateral pain and headache and may offer considerable difficulty in a differential diagnosis. This is especially true of an intracranial aneurysm in the anterior portion of the circle of Willis, which causes marked pain in the orbit and the postorbital regions. Cocainization of the sphenopalatine ganglion does not relieve symptoms in any of these intracranial conditions and helps to rule out sphenopalatine ganglion neuralgia.

The temporomandibular joint syndrome may be confused with sphenopalatine ganglion neuralgia in that a greater portion of the symptoms are localized in the ear, posterior to the ear or perhaps anterior to the ear. In addition, there may be burning sensations and pains in the cheek of the affected side. However, in the temporomandibular joint syndrome there is usually a history of dental extractions in the molar region about the time of the onset of the symptoms. The symptoms can be alleviated by preventing the rotation of the mandible in the temporomandibular joint by the use of corks, disks or short pieces of rubber tubing, which will hold the lower jaw as far apart from the upper jaw as it was previous to the extractions; and the pains may be permanently relieved by building up the molar regions of dental plates.

Elongation of the styloid processes may cause some confusion in a differential diagnosis, but here again most of the symptoms are referred to the aural region and the lateral wall of the pharynx rather than to any region about the orbit and bridge of the nose. Palpation in the tonsillar fossa or beneath the tonsil, together with lateral roentgenograms of the neck, to demonstrate an elongation of the styloid process, should clear the diagnosis completely. Elongation of the styloid processes usually causes symptoms after tonsillectomy has been done rather than before. Shortening of the elongated styloid process clears up these symptoms.

Ocular disorders of all types should offer little difficulty in differential diagnosis following ophthalmologic consultation. Neuralgia of the otic

ganglion, ciliary ganglion and geniculate ganglion is so rare that it need not be considered in the differential diagnosis. Sensitivity to histamine may offer a diagnostic problem in future years, but at present little is known of this subject for differential purposes.

PRETREATMENT SUGGESTIONS

There may be systemic disturbances which must be treated or eradicated before any attempt is made to alleviate neuralgia of the sphenopalatine ganglion. In recent years numerous medicaments of proprietary nature for the relief of headache have appeared on the market. The public, as well as members of the medical profession, has unwisely made extensive and frequent use of these agents. Most of these preparations contain large amounts not only of bromides but also of acetanilid and other similar drugs. As a result, it is not uncommon to find patients who have headaches and facial pains, together with some cerebral disorientation, which are due to nothing more than acetanilid poisoning and brominism. The acetanilid poisoning produces cyanosis, and the patient has headaches due to anoxemia. It is necessary to free these patients of their acetanilid poisoning and brominism before giving any treatment related to the sphenopalatine ganglion; otherwise, the headaches, which obscure, and sometimes exceed, the symptoms referable to the nasal ganglion, will continue. A number of these patients have been given narcotics, and it is well to have the patient perfectly free of the use of narcotics before proceeding with any intranasal surgical treatment.

Patients who have anemia or a blood dyscrasia of any type producing anoxemia or ischemia should be relieved of these conditions before any surgical procedure is anticipated. In cases in which an infection of the paranasal sinuses is concomitant with preexisting sphenopalatine ganglion neuralgia caused by a septal deviation or other intranasal abnormality, it is necessary to clean out the involved sinuses thoroughly at the time the necessary intranasal surgical procedure is carried out. It happens not infrequently that the patient has arthritic, asthmatic or other generalized symptoms due to the infection of the paranasal sinuses. These symptoms will also be alleviated by the thorough eradication of the sinus infection.

TREATMENT

Regardless of the cause, duration or severity of sphenopalatine ganglion neuralgia, the treatment should always be most conservative. Frequently, palliative intranasal treatment suffices. If surgical intervention is required, the simplest procedure provides the best results. One must not forget that surgical procedures produce fibrosis and that the best of intranasal surgical treatment may be defeated by scarring.

There are several phases of treatment for the irritation which produces neuralgia of the sphenopalatine ganglion. Attacks of the disease may be alleviated permanently, or at least for a few hours, by the application of 10 per cent cocaine to the area of the ganglion, which, as mentioned earlier, lies just posterior to the middle turbinate. One may find it necessary to spray the nose with a 1 per cent saline solution of ephedrine sulfate in order to shrink the mucosa so that an applicator may be directed past a spur or ledge, to the ganglion. Cocaine is most easily applied by curving the tip of a wire applicator, wrapped in cotton (which has been flamed to eliminate lint), so that as one proceeds along the floor of the nose with the applicator, the tip will fit into the area

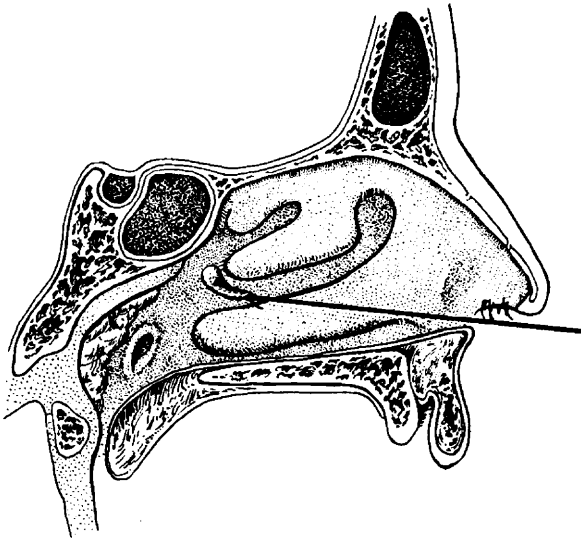


Fig. 8.—Region to which cocaine is applied in the treatment of neuralgia of the sphenopalatine ganglion.

behind the middle turbinate, as desired (fig. 8). One application of cocaine may give permanent relief. However, there may be recurrence at any time from a few hours to weeks later. For a patient who has a recurrence of pain during the night or who for some reason cannot be seen in the office, spraying with a 1 per cent to 3 per cent solution of butyn sulfate frequently alleviates the pain. Butyn sulfate is a rather mild and relatively nontoxic local anesthetic. It is also helpful to have the patient spray the nose with 1 per cent saline solution of ephedrine sulfate previous to the spraying with butyn sulfate, in order that the butyn sulfate may have full access to the desired area.

Silver nitrate, in a 2 per cent solution, and even up to a 50 per cent solution, has been used by some in the treatment of this disease, but

this is likely to produce a further irritation of the ganglion rather than quiescence of symptoms.

When symptoms of considerable degree continue after the former, more conservative, methods of treatment, one finds it necessary to resort to the more radical surgical procedures, which in reality are not radical. This is necessary for that group of patients who have an intranasal deformity of some type or a focus of infection that is to be eliminated. The simplest type of surgical procedure designed to eliminate the symptoms is that of fracturing and displacing laterally a middle turbinate that has projected too far out to the center of the nose and lies adjacent to the nasal septum. This is easily done by the application of cocaine along the line of insertion of the turbinate. Then by lateral pressure with any blunt instrument the turbinate is easily fractured. It is even more desirable to crush the bony structure in the turbinate, so that there will be no tendency for it to expand back to its original position. When a septal deviation or a spur or ledge of the nasal septum is involved and is thought to be the cause of the irritation of the sphenopalatine ganglion, it is necessary to perform a submucous resection of the nasal septum, which, if well done and complete, includes the removal of the spur or ledge, as the case may be. Should the disease be considered toxic in origin and due to an infection of the posterior ethmoid or sphenoid sinuses, these paranasal sinuses and any others which are diseased should be opened thoroughly and the diseased mucosa removed. My associates and I have found very few cases, however, in which the posterior ethmoid and sphenoid sinuses were infected or were thought to be the cause of the disease. Abscessed teeth should be extracted, and diseased tonsils should be removed. Disease of the gallbladder, constipation and other constitutional disorders should be treated as indicated. If these procedures should not suffice to relieve symptoms, one may attempt to inject into the sphenopalatine ganglion 0.5 cc. of 5 per cent phenol in 95 per cent alcohol. Originally, we made injections into the sphenopalatine ganglion in 5 patients. The majority of these had submitted to a submucous resection. However, we have found in 53 instances in which submucous resection was done only 1 in which symptoms were not alleviated by the submucous resection itself. Thus, injection into the ganglion is considered superfluous and is not without danger.

Sluder recommended injection into the ganglion in all cases as his treatment of choice. It is difficult to make injections into the ganglion either with a straight or a curved needle in most cases, because of intranasal deformities. A submucous resection of the nasal septum is certainly

indicated, and since the operation itself relieves the symptoms, the injection is not necessary. If symptoms should continue after all of the aforementioned procedures, it would then be necessary to eviscerate the entire sphenopalatine ganglion. Happily, we have not found this necessary in any instance. This is an extremely radical procedure, which requires the greatest of skill and probably would be best handled by a neurosurgeon in cooperation with the otolaryngologist. We have observed only 1 patient who has not been relieved by the previously mentioned procedures, and this patient has not yet reached the stage at which extirpation of the ganglion is deemed necessary.

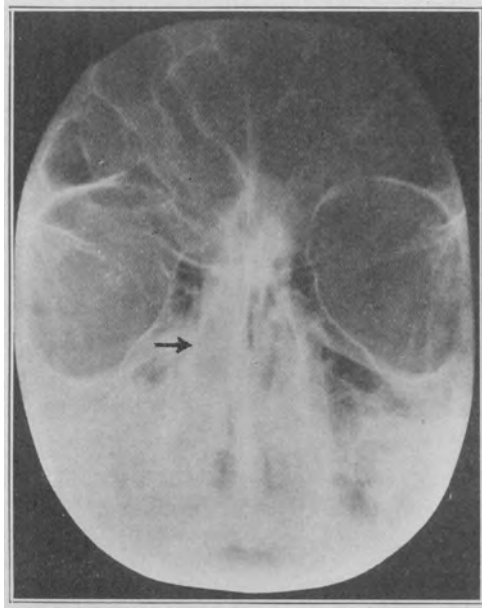


Fig. 9.—Roentgenograms illustrating a large middle turbinate projecting against the nasal septum on the right. Eventually, chronic ethmoid infection had caused necrosis of the right middle turbinate with abscess formation, on account of the fact that there were ethmoid cells in the middle turbinate.

RESULTS

It has been emphasized throughout the discussion of this disease that complete relief of symptoms due to sphenopalatine ganglion neuralgia is anticipated by the methods enumerated in the treatment of the disease. If the conservative methods control the disease, surgical intervention is not indicated. However, if surgical treatment is indicated, it likely will call for a correcting of septal deviations, spurs and ledges or a prominent middle turbinate. Nothing short of the correction of

these deformities will suffice. Operations to remove these obvious deformities have provided excellent results.

Of this group of 159 patients, 119, or 80 per cent, were found to require some type of intranasal correction, and there were 7 additional patients who had intranasal adhesions following previous operation. Fifty-three of the patients returned for operation, and probably a large number appeared elsewhere for the operation advised, because the symptoms in most cases were so severe that the patient could not tolerate them much longer. All except 1 of the 53 patients operated on were relieved of their symptoms. According to our records, therefore, a submucous resection of the nasal septum was found to be the most efficient method of treating sphenopalatine ganglion neuralgia in the largest number of cases.

A report of a few typical cases, in which the various etiologic factors were involved, follows:

REPORT OF CASES

CASE 1 (nasal deformity type—deviated septum).—Mrs. A. M., then aged 28, was seen on Dec. 8, 1931, complaining of pain over the left side of the face—most intense around the left eye, the root of the nose and just behind the mastoid on the left side—and of earache and stiffness and pain in the left jaw. These symptoms had been present for at least one year. There was difficulty in breathing through the left naris on account of a narrowing in the region of the middle turbinate due to septal deviation. Cocainization of the sphenopalatine ganglion relieved the pain immediately. There was no evidence of infection of the paranasal sinuses.

Six days later a submucous resection of the nasal septum was done. The patient was relieved of the facial pains on the left side and when seen again six years later, in 1937, she was complaining of stiffness in the left jaw and some pain in the ear, similar to that which she had complained of in 1931. An elongation of the styloid process on the left side was found to be the cause of these symptoms, and after operation for shortening the left styloid process her aural symptoms were relieved completely. There has been no recurrence of the sphenopalatine ganglion neuralgia.

CASE 2 (nasal deformity type—septal spur).—Mrs. M. O., aged 62, was seen on Sept. 11, 1939. She had two separate pathologic conditions. Since childhood she had been subject to a generalized headache, with scotomas and halos around objects looked at. In addition, she had a facial pain on the left side, of at least twenty years' duration, which was located in the region of the left eye, causing pain in the nose and extending back into the ear and the region behind the left mastoid. Frequently, there had been pains running into the left shoulder and into the left arm. There was some confusion about the pain in the arm, because of arthritic symptoms. (She had suffered from arthritis for many years.) Throughout her life she had experienced difficulty in breathing through the left side of the nose. For the headache and the facial pain on the left side she had been treated with vaccines and intranasal packs without benefit for many years. Her tonsils had been removed ten years previously. All her teeth had been extracted many years before.

The examination revealed, in addition to generalized infectious arthritis, a marked anterior septal deviation to the left, with a large ledge ending in a spur in the left middle meatus. The spur was as large as a thorn on a rose bush, and the tip of this spur could not be seen. Attempts to cocainize the sphenopalatine ganglion were not successful, on account of the septal deformities. Butyn sulfate sprayed into the nose relieved the pain in the left side of the face to a slight extent.

This patient's nasal septum was straightened on the following day, and she was completely relieved for the first time in twenty or more years of the severe facial pains on the left side. She thought the generalized headaches were also somewhat alleviated. To date there has been no recurrence of the facial pains on the left side.

CASE 3 (nasal deformity type—osteoma).—Mrs. R. W., aged 32, was seen on July 2, 1937. At the age of 12 she had sustained an injury over the left side of her face from a baseball bat, and there had been swelling of that region ever since. At the age of 20 she had an intranasal operation for the removal of turbinate tissue. For the past three years she had had excruciating pain, which occurred almost daily throughout the entire left side of the head. These symptoms were most severe medial to the left eye and behind the left mastoid region. If the scalp anywhere over the entire left side was touched, this caused extreme and unbearable pain.

The examination showed entirely normal conditions except that a portion of the middle turbinate on the left side had been removed. The area was free of hyperemia, and there was no discharge. There was pain on the left side from all the branches of the fifth nerve, in addition to the symptoms referable to the nasal ganglion. Roentgenograms revealed an osteoma in one of the left posterior ethmoid sinuses (fig. 4).

After an operation for removal of the osteoma, on July 10, 1937, this patient had complete relief from the pain on the left side. No active infection was found, and her symptoms were thought to be due to pressure by the ethmoid osteoma directly on the branches of the sphenopalatine ganglion.

CASE 4 (toxic type).—Dr. H. F., aged 37 had recurrent infections of the upper respiratory tract of all types, of several years' duration. There had been much postnasal and pharyngeal discharge. He had a series of roentgen ray treatments, to eradicate the lymphoid tissue of the pharynx, and the uvula had been shortened. Eventually, it was found necessary to perform a submucous resection and to remove a cyst from the left maxillary sinus. The operations were done in May 1939, and the cyst, containing about 6 cc. of straw-colored fluid, was removed from the floor of the left antrum by the Caldwell-Luc operation. Immediately after the operation extreme angioneuroedema developed. This not only involved the left side of the face but extended to the right side. Both eyes were swollen completely shut. The upper lip became enormous.

On the second day the patient began to have excruciating pain on the left side, typical of sphenopalatine ganglion neuralgia. Application of a 1 per cent saline solution of ephedrine sulfate and 10 per cent cocaine reduced to a great extent the swelling in the left side of the nose. Within ten minutes he was able to open his eyes slightly. The edema and the pain, however, recurred daily for about five days and were controlled by the application of cocaine to the sphenopalatine ganglion.

This case is mentioned as one of toxic stimulation of the left sphenopalatine ganglion, since the operation performed did not involve the area near the sphenopalatine ganglion but involved the floor of the antrum only. Furthermore, the angioneuroedema was evidence of an intoxication.

CASE 5 (toxic type).—Mrs. L. D., aged 55, was seen on May 10, 1939. For seven weeks there had been symptoms typical of sphenopalatine ganglion neuralgia on the left side, during which time there had been dental extractions. The most excruciating pain had occurred after the extraction of lower teeth on the left side five days before. The teeth were extracted because of root abscesses. The symptoms in this case indicated toxic irritation of dental origin. Complete and permanent relief was obtained by cocainization of the sphenopalatine ganglion.

CASE 6 (neurotic type).—A married woman aged 25 was seen in November 1938. She had complained for some months of neuralgic pains on both sides of the head. (Note that she had pains on both sides, which is unusual in true sphenopalatine ganglion neuralgia.) She had been married six months previously. She complained of dizziness and fainting and soon became emotionally very unstable. Eventually, she developed into a highly neurotic patient. After she had heard on the radio a dramatized story which concerned a case of brain tumor, extreme intracranial headaches developed. Domestic difficulties were pronounced. There was some sexual maladjustment, with fear of pregnancy. An examination revealed slight deviation of the nasal septum to the right, but certainly not enough to justify operative procedure. Usually, the local application of 10 per cent cocaine relieved the symptoms for only one hour. She was not permanently benefited by any medical treatment but has been benefited by proper psychiatric therapy.

SUMMARY

A clinical entity known as sphenopalatine ganglion neuralgia was recognized in the early part of the twentieth century. Immediately it came into great prominence but by the late twenties had lost much of its prestige. It is a neglected disease, not only because it is often not recognized but also because it is not accepted. The characteristics of the disease have been described. No attempt should be made to include under the diagnosis any condition which does not present unilateral facial pain. The inclusion of questionable and remote diseases seems to have caused the nonacceptance of the syndrome in the not too distant past. It appears that the syndrome is not often caused by an infection of the sphenoid or posterior ethmoid sinuses, as was indicated by Sluder. On the contrary, it seems proper to attribute the disease to intranasal swelling due to three main causes: first, intranasal deformities, including septal deviations, spurs, ledges and prominent turbinates, adhesions, osteomas and scarring due to too radical intranasal surgical procedure in the region of the sphenopalatine ganglion; second, toxicity due to systemic disorders, including infections of the paranasal sinuses, dental abscesses, infections of the tonsils, disease of the gallbladder, general debility and endocrine disturbances, and, last, emotional instability, hysteria and psychoneuroses.

The various methods of treatment have been enumerated. This disease has been improperly treated because it has not been correctly diagnosed. The treatment recommended in all instances is that which is most conservative for the patient under consideration. Local application of 10 per cent cocaine to the ganglion, the spraying of the region with 1 per cent butyn sulfate, the application of silver nitrate to the ganglion and the injection of phenol and alcohol into the ganglion have been described.

My associates and I have found 80 per cent of the patients to have an intranasal deformity, and in most of these cases a submucous resection of the nasal septum has been required, to eliminate contact of the intranasal structures. This operation has proved our best weapon among the various methods of treatment. We wish to stress the point that injections into the ganglion are not necessary and that patients are relieved of their symptoms after the nasal septum has been straightened or when all points of intranasal pressure have otherwise been eliminated. Several cases have been cited, to illustrate various origins of the disease.

Finally, the two points to be remembered are: first, sphenopalatine ganglion neuralgia causes a unilateral facial pain and not a headache (this should facilitate the differential diagnosis); and second, the simple elimination of contact between intranasal structures will eliminate symptoms in most instances.