AFFECTIONS OF THE SPHENOPALATINE GANGLION

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A knowledge of the sphenopalatine or Meckel's ganglion and its affections has gained considerable importance since Sluder[1] in 1908 discovered the relationship that certain headaches and neuralgic sensations have to disturbances of this nerve-centre. In the past a great variety of ailments, ranging from neuralgia of the head to sciatica, have been treated by applications to or injection of this ganglion. It seems apparent that overenthusiasm has been responsible for many claims of pain relief without due consideration being given the neurological aspect or the transient psychic benefit that may result from an injection of this ganglion. When relief for such conditions as lumbago, sciatica, dysmenorrhœa and gastric pain are claimed without adequate explanation of the nerve connections involved, it naturally leads to a certain amount of skepticism in the minds of many, and tends to place a therapeutic procedure that does have a certain limited usefulness in considerable disrepute. This review will give consideration to only those neuralgic conditions that appear to have an anatomical and neurological basis for symptoms existing.

Anatomical Considerations.—Space will not be taken for review of the detailed anatomy of the ganglion, its branches and roots, as this can be obtained from any anatomical text-book. It would be well, however, to relate briefly its outlying connections.

The sphenopalatine ganglion may be considered as a nerve-centre acting as a terminus for the trigeminal, facial and sympathetic systems. The ganglion mass receives sensory impulses from practically all the lining mucosa of the nose, the maxillary, posterior ethmoidal and sphenoidal sinuses, the roof of the mouth, the upper two-thirds of the tonsils, the soft palate, the greater part of the pharynx and nasopharynx, and, in addition, taste fibres from the anterior two-thirds of the tongue. These sensory fibres pass for the most part through the superficial portion of the ganglion mass, not being in relation to the ganglionic cells, on their way to join the maxillary divisions of the fifth nerve. The ganglion is connected with the facial nerve by means of the superficial petrosal nerve, its motor root. A rather wide motor connection is given the ganglion when we consider that the facial nerve either directly or indirectly is connected with the following nerves: the acoustic, the otic ganglion, the sympathetic on the middle meningeal artery, the auricular branch of the vagus, the great auricular, the auriculotemporal, the lesser occipital, the trigeminal and the cutaneous cervical nerves. In addition, connections of the sphenopalatine ganglion to the sympathetic system by means of the great deep petrosal nerve open up possibilities for a great variety of indirect nerve associations.

Etiology and Pathology of Sphenopalatine Ganglion Disturbances.—The

disturbances of the sphenopalatine ganglion may be considered due to an irritation or inflammation of it which is usually secondary to inflammation of adjacent structures, namely, the posterior ethmoidal air-cells, the sphenoidal sinuses, the nasal mucous membrane and probably rarely the maxillary sinuses. Some cases are without doubt of systemic toxic origin without any pathological conditions of the nose or sinuses having been present.

The pathology of the tissues adjacent to the ganglion that sets up disturbances in its structure may be only an acute rhinitis that transmits its influence through the tissues of the sphenopalatine foramen to the ganglion situated often as close as one millemetre from the nasal mucous membrane. The ganglion is probably more often affected by inflammation in the posterior ethmoidal or sphenoidal sinuses. This inflammation may be suppurative or hyperplastic in character and affects the ganglion by an extension of the disease process or by a transmission of toxic products through the thin separating bony walls.

In addition to these exciting causes there must also be considered the factor of heredity as constituting a latent cause. This factor may show the primary instability to be in the endocrine system or in the autonomic nervous system.

Symptomatology.—The various connections of the sphenopalatine ganglion by means of its sensory, motor and sympathetic roots open up possibilities for a great variety of symptoms. The sensory distribution to the nose, throat and sinuses gives characteristic indications in these regions. An irritation of the motor root can produce neuralgias of the face and neck by its connection with the facial and the lesser occipital and cutaneous cervical nerves; can account for disturbances in the eye and mandibular regions by its connections with the ciliary and otic ganglion; can cause reflex otalgia by its connection with the tympanic plexus; and may account for a variety of visceral symptoms by its connection with the vagus. In addition, there may be motor phenomena of the soft palate from involvement of the motor fibres to the levator palati and azygos uvulæ muscles. The connections of the ganglion to the sympathetic system may give a great variety of symptoms largely of glandular and vascular structures, but also of involuntary muscular structures, and possibly also remote parts of the body which may send sensory impulses through the sympathetic by paths that are but little understood. Thus such conditions as hyperæsthetic rhinitis, hay fever, asthma, cardiac pains, angina pectoris, gastric pains, nausea and vomiting and, in addition, symptoms in remote structures of the body which cannot be explained except by attributing to the sympathetic system functions which have not as yet been scientifically proved, may possibly be caused by disturbances of the ganglion.

Sensory fibres in the sympathetic nervous system have been definitely stated to be dendrites of neurones which have their cell bodies in the posterior root ganglions; but they are transmitted by the sympathetic root only as a pathway. The sympathetic system in some way appears to be capable of transmitting afferent pain impulses, but to explain some of the phenomena seen after anæsthetizing the nasal ganglion, it is necessary to attribute to the sym-

pathetic system the power of transmitting afferent impulses which are not painful sensory ones. Asthma, as a nasal reflex, may be explained in such a way.

The affections of the ganglion can be considered as neuroses or neuralgias and give rise to several syndromes of symptoms depending on the part of the ganglion involved and severity and duration of the exciting cause. Due to the variation in the position of the ganglion and the differences in the shape and size of the adjacent sinuses, all parts of the ganglion are not equally affected by adjacent pathology. One part alone may be involved giving one series of symptoms, or several parts may be affected giving a combination of symptoms. It is quite possible for any one of the sensory, motor or sympathetic roots to be involved alone or in any combination, thereby giving a series of symptoms easily traceable to the particular root involved, or in the case of affection of several roots giving a distribution of symptoms of such variety and vagueness as to suggest a general neurosis. At times, mingled with the symptoms of ganglion involvement, may be indications of affection of other nervous structures due to the variation in position and size of these nervous structures and the sinuses. The great variation in size and shape of the sphenoid sinus brings it at times in close relation with other nerves besides those connected with the sphenopalatine ganglion. The cavernous sinus containing the third, fourth and sixth cranial nerves is situated just external to and above the body of the sphenoid bone. These may be involved by pathology in the sphenoid sinus. The first, second and sometimes the third division of the fifth nerve are in relation to the sphenoid sinus laterally and the optic nerve passes over the sinus and is often separated from it by only a thin layer of bone. A large sphenoidal sinus extending outward may encroach on the foramen rotundum containing the maxillary nerve or the foramen ovale containing the mandibular nerve, or the sphenoid fissure containing the ophthalmic division, and also the third, fourth and sixth cranial nerves. A prolongation downward of the sinus brings it in very close relation to the vidian nerve and a further prolongation downward brings it in close touch with the eustachian tube.

The symptomatology of sphenopalatine ganglion disturbances can therefore be divided into four syndromes: (1) A trigeminal maxillary syndrome. (2) A sensory facial nerve syndrome. (3) A sympathetic syndrome. (4) A local sphenopalatine ganglion syndrome.

The trigeminal maxillary syndrome gives symptoms referable to the distribution of this nerve such as pain at the root of the nose or inside the nose, pain in the upper jaw and teeth, itching of the hard palate or throat or stiffness or aching in the throat or sense of foreign body, feeling that the teeth are too long, and diminished sensation of the soft palate, pharynx and tonsils of the affected side.

The facial nerve syndrome shows itself in such symptoms as a diminished and delayed perception of taste on the affected side and certain motor signs, such as a suggestion of flattening of the face due to involvement of the innervation of the muscles of facial expression and a higher palatine arch on the

affected side, deflection of uvula from the affected side, and a deflection of the median raphé from the affected side in the act of swallowing.

The sympathetic syndrome gives symptoms of much wider distribution in its ordinary capacity as controlling vasomotor and secretory phenomena and also in its power of transmitting afferent sensory impulses through sympathetic connections with remote parts of the body. Its vasomotor and secretory influence is shown typically in attacks of hyperæsthetic rhinitis. This condition is apt to come on at any time of the year, is irregular in its duration and severity, and is not associated with the inhalation of the various pollens. The symptoms in such attacks are similar to those of hay fever but are apt to be more transient in character. At times there may be a persistant salivation.

Involvement of the sympathetic elements in the ganglion may also show itself in such symptoms as pain in, behind and about the eye; pain in the lower jaw and teeth; pain in the ear and mastoid extending back of the mastoid into the occiput, neck, shoulder, breast, axilla, and even down the upper extremity into the fingers; sometimes pain in the brow and upper part of the head and muscular weakness of the arm and shoulder; itching of the upper extremity, and tic douloureux.

While such a classification into syndromes is convenient for purposes of description, it is unusual to find any patient with symptoms of one type alone or all the symptoms of any one type. There is practically always a combination of symptoms belonging to two or more of these syndromes.

The typical picture of sphenopalatine ganglion neuralgia is pain beginning at the root of the nose or in or around the eye, extending over the maxilla and backward to the mastoid process to become most severe at a point five centimetres posterior to its tip; from here the pain extends backward into the neck, shoulder, scapular region and sometimes into the axilla and breast. In severe attacks it may go down into the arm and forearm and even to the fingers. The attacks may occur at any time without warning and may last from a few minutes to several weeks or even be more or less constant. In mild cases there may be only slight sensory disturbances as a sensation of tightness or drawing, of the teeth being too long, stuffiness of the ears, aching throat, itching of the hard palate, or stiffness or rheumatism of the neck and shoulders. In severe cases the pain may be like a ball of fire or a knife cutting or boring into a nerve. As isolated symptoms of ganglion neurosis there has also been mentioned glossodynia, otalgia, nausea, paraguesia, vertigo, scotomata, photophobia and asthma.

When once the nerve-centre has been affected, it apparently remains vulnerable for an indefinite time for the symptoms may be rekindled by fatigue, anxiety, apprehension, tribulation, anger, mental shock, strain or systemic toxæmias. Many of the cases are of long standing and have finally had an imbalance in their whole nervous mechanism becoming neurotic and going from one doctor to another seeking relief.

Diagnosis.—A diagnosis of sphenopalatine ganglion neurosis may be of great importance to the health and life of a patient. Many cases with ganglion

symptoms have been treated by the ordinary local therapeutic measures without relief when an injection of the ganglion may have given quick relief. On the other hand, being overly anxious to find the ganglion at fault has resulted in many cases being diagnosed and treated as ganglion neuralgia when there was present some other very decided cause for the existing symptoms, such as an impacted molar tooth, infected tonsils, hypertrophied turbinates, sinusitis, deviated septum, *etc.*

Practically all the symptoms of sphenopalatine ganglion neuralgia may be produced by lesions of the nerve-trunks supplying the ganglion, namely, the maxillary and vidian nerves. These nerves often lie in very close association with the sphenoid sinus or a posterior ethmoidal cell occupying the body of the sphenoid bone. When infection of these sinuses occurs it may readily involve these nerve-trunks at a point central to the ganglion and have no influence whatever on the ganglion itself. Therefore it is necessary to differentiate between a lesion of the ganglion itself and a lesion of the nerve-trunks involved in suppurative or hyperplastic inflammation in these sinuses. This differentiation can be made by a therapeutic test. Cocainization of the ganglion will stop the pain caused by a lesion of the ganglion and will have no effect if the lesion is involving the nerve-trunks central to the ganglion. On the other hand, intrasphenoid application of cocaine in the latter cases may stop the pain.

In cases with symptoms simulating hay fever it should be determined if the patient is sensitive to pollens or other proteins before making a diagnosis of ganglion neuralgia.

Another diagnostic point may be found in the appearance of the nasal mucosa about the site of the sphenopalatine foramen. When the ganglion is at fault there is sometimes found a congestion and thickening of the mucous membrane in this region. This appearance is too infrequent to be relied upon as a diagnostic sign.

Another condition to be taken into consideration is that of endocrine imbalance. Headaches of pituitary origin may be greatly benefited by pituitary extract. Pituitary insufficiency may also cause symptoms of hyperæsthetic rhinitis which may be controlled by the use of glandular products. The other endocrine glands must also be considered before definitely making the diagnosis of ganglion neuralgia.

Treatment.—Treatment of sphenopalatine ganglion disturbances consists in the local application to or injection of this nerve-centre, with chemical agents. This should be preceded by a diligent search for the cause of the ganglion disturbance and elimination of the causative factor if possible. In many cases, however, no definite cause of the condition can be found. These are probably due to an irritation or inflammation remaining in the ganglion region after the sphenoiditis or ethmoiditis which originally causes the disturbance has been cured. In these cases application to or injection of the ganglion alone have completely relieved pain.

A completely satisfactory explanation of how a single application of 20 to 40 per cent. cocaine solution to the mucous membrane of the nose overlying the ganglion can permanently cure a ganglion neuralgia has not as yet been made. It has been stated that the effect of the application or injection may in some way bring about an involution of the hyperplastic process surrounding the ganglion. If this be true, although there may be an immediate cessation of symptoms, there is apt to be an interval of several weeks before a permanent cure is obtained.

A number of different chemical agents have been used for application to or injection of the ganglion. The most widely used drug for local application is cocaine, 20 per cent. to a saturated solution. Silver nitrate, from 2 to 75 per cent. and a 0.4 per cent. gaseous solution of formaldehyde have also been used.

For injections of the ganglion, cocaine or procaine solutions are sometimes used but generally either a 5 per cent. phenol in 95 per cent. grain alcohol or absolute alcohol is employed. The addition of the phenol to the alcohol prevents much of the pain experienced in the injection of alcohol alone.

In the treatment of mild cases showing the neuralgic or sympathetic syndrome it has been found that the local applications to the ganglion region in the nose of a 2 per cent. solution of silver nitrate one or two times weekly for from two to four weeks has given permanent relief in some cases. In such cases I prefer to use a 20 per cent. cocaine solution repeated two to three times at three-day intervals. The area in the nose to which the solution is applied is located on the outer wall of the nasal cavity at a point just posterior to and above the attachment to the nasal wall of the posterior end of the middle turbinate. I have obtained better results by a rather brisk rubbing with the applicator at this area rather than simply allowing the applicator to remain quietly in position. Three or four minutes of rubbing should suffice for anæsthetization, but if there is no massaging, the applicator should be left in position for fifteen minutes.

In more severe or long-standing cases of ganglion involvement, more complete and lasting benefit will be obtained by the injection of alcohol into the ganglion. For this purpose from 0.5 to 1 cubic centimetre of alcohol can be used, and if accurately placed within the ganglion substance, should give relief from four months to permanently. If, as is probably the case in most instances, the injection should be in the tissues surrounding the ganglion rather than in the ganglion structure, the relief is apt to be less complete and of shorter duration. In most cases I have found it better to make a series of three injections at intervals of two or three days.

The injection of the ganglion can be made by three different routes: Through the nasal mucous membrane, through the pterygomaxillary fissure, and through the posterior palatine canal. The injection through the nasal mucous membrane was first used by Sluder and for years thereafter was the route commonly employed. Abnormalities in the nose, however, make injecting by this route at times difficult, uncertain and sometimes dangerous.

The method of injection through the pterygomaxillary fissure has been commonly used by dentists. It avoids the possible difficulties of the intranasal route but there is still the uncertainty of exactly placing the solution in the ganglion.

The use of the posterior palatine canal as an injection route was first advocated, in 1921, by Carrea, a dental surgeon of Buenos Aires. He used this route for anæsthetizing the maxillary nerve. Since 1925, this route has been the one of choice for injections of the ganglion because of its features of accessibility, accuracy and certainty of result. However, there were still present certain factors that made it at times difficult, uncertain and occasionally impossible to reach the ganglion by this route. This led me,[2] a few years ago, to make a study of dissected specimens and skulls in an attempt to develop a technic of injection that would attain greater accuracy.

The following technic was adopted by me as the one giving the surest method of approach to the ganglion: First palpate the hard palate in an attempt to find a depression corresponding to the location of the foramen. If this cannot be felt, the needle is inserted first so that the shaft comes opposite the anterior edge of the first molar tooth, close up to the alveolar edge, pressing the needle upward and slightly backward at an angle of about 45°. If the needle does not enter the canal it is removed and inserted again a millimetre or two directly posterior to the first insertion. In this way it may be necessary to insert the needle several times, but if the palate has been previously rubbed with a 10 per cent. cocaine solution and painted with an antiseptic solution, it causes no pain and gives very little chance for infection. When the molar teeth are absent the posterior edge of the hard palate and especially the posterior end of the pterygoid process should be palpated and the needle first inserted about two centimetres anterior to the edge of the pterygoid process. The point of the needle should be inserted to a depth of 3.3 to 3.8 centimetres, being directed somewhat internally to slide up along the inner wall of the canal. To prevent as far as possible the pain of an alcoholic injection, it is wise as a preliminary measure to massage the ganglion region in the nose for a few minutes with a 10 to 20 per cent. cocaine solution. Before making an injection, an estimate of the size of the skull should be made. If the skull appears to be smaller than the average, the needle should be inserted to a lesser depth, and if the skull is large, the point of the needle should be carried to a greater depth. In spite of the most skillful technic there is occasionally found a patient in whom an injection of the ganglion by this route cannot be made, due to extreme narrowness of the canal or a tortuosity that would prevent the entrance of the needle. In such cases injection by one of the other two routes will be necessary. In rare instances it may be found wise or necessary to attempt an extirpation of the ganglion. Frazier[3] in 1921, described a surgical approach to the ganglion that would appear to give satisfactory results in the hands of the experienced and skillful surgeon.

Prognosis and Complications.—The prognosis in treating cases of spheno-

palatine ganglion neuroses is very uncertain. A larger proportion of improvement is obtained in the sympathetic than in the neuralgic type.

When symptoms are typical of ganglion involvement much can be promised as a result of treatment, but the typical case is rare. The great majority of patients are among those cases of atypical neuralgia which are but little benefited by any one procedure of treatment. The typical neuralgic symptom-complex can usually be relieved by the application of cocaine to the ganglion region in the nose. If this is found to give some relief, a series of alcoholic injections are apt to give excellent results. Patients that show no improvement after three alcoholic injections can probably be considered hopeless so far as that method of treatment is concerned.

Some cases are not relieved by local applications but are relieved or cured by alcoholic injections. Others seem to be relieved for some time and then recur. In the latter case re-injections are indicated. Sometimes a failure to establish a lasting cure is due to the failure to follow up the injections with the proper surgical procedure to remove the cause of the condition primarily such as sinusitis, or to correct an imbalance in the endocrine system.

Probably the most frequent complication that may follow the attempt to inject the sphenopalatine ganglion is the escape of the alcoholic solution down the pharyngeal wall into the throat or larynx. This would give an unpleasant but not dangerous experience. Sometimes a temporary swelling and ecchymosis in the cellular tissue beneath the lower eyelid has occured, causing the eye to close. Swelling and pain in the temporal and parietal regions of the head are common and transient following the injection. Excruciating pain in the head is sometimes experienced which may last for several days and require sedatives. Primary hæmorrhage has occurred rarely, probably due to the penetration of the internal maxillary artery. Secondary hæmorrhage is somewhat more common, coming on five or six days after the injection. This is probably due to the erosion by the alcohol of the sphenopalatine or descending palatine arteries. A few cases of paresis of the abducens nerve have been reported, causing a diplopia. This occurs shortly after the injection and is due to the passage of the injecting fluid through the sphenomaxillary fissure into the orbital tissues. In these cases the paresis and diplopia have cleared up within a few weeks. It seems possible also for blindness to occur if the injected alcohol reaches the optic nerve but this nerve is well out of reach, unless the needle is inserted to an unwarranted depth.

Cases of neuralgia for treatment by injection of the sphenopalatine ganglion should be selected with care. Injection of all atypical neuralgias is apt to bring discredit on a therapeutic procedure that does have a limited field of usefulness. In typical conditions of ganglion neurosis brilliant results can be obtained but in the majority of neuralgic cases little should be promised. However, in many cases, it seems perfectly justifiable to employ this procedure for the purpose of eliminating the ganglion as a source of the neuralgic condition present. If there is failure to relieve the pain other methods of therapeusis may give the desired results.

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