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THE SPHENO-PALATINE GANGLION.

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I. Introduction.

THE spheno-palatine ganglion has gained considerable prominence during recent years, largely on account of the researches of American and French rhinologists, who consider it to be the site of many obscure deeply-seated headaches. It is blamed also for cases of congestion and hypersecretion of the nose and adjoining cavities. In these conditions it is possible to establish a fairly plausible connection between the ganglion and the pathological phenomena of which it is supposed to be the cause. There are, however, other conditions, in which a claim for cure or alleviation has been made by treatment of the spheno-palatine ganglion, where the connections between cause and effect are difficult to trace. Examples of such conditions are sciatica, hiccough, œsophageal spasm and diarrhœa.

It would appear from allusions in later papers that there was, at the end of the nineteenth century in France, a phase of spheno-palatine ganglionectomy for all sorts of lesions. This craze died out on account of the dangers and difficulties of the operation, and has no connection with the modern cult which was started by Sluder in 1908. In this discussion only the work since the first paper of Sluder will be considered.

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II. Historical Review.

Sluder was a pioneer in the differentiation and treatment of the nasal ganglion neuralgias. In his first paper, in 1908,[1] he described a group of neuralgia cases of unknown origin. In the second part of the paper he presented an account, after Sobotta, of the spheno-palatine ganglion, which gave him the idea that this structure might be the cause of the pain. He therefore attempted to anæsthetise the ganglion from the nasal cavity and found that in some cases all the symptoms disappeared; in others, however, only partial or no relief was obtained. He also noted that the application of 2 per cent. silver nitrate or 0·5 per cent. formol to the nasal mucous membrane over the ganglion often cured or relieved the neuralgia.

In a later paper (1910)[2] Sluder formulated a syndrome due to spheno-palatine neurosis. The cause of the condition is a coryza of the nose, which may be so slight that the patient does not notice it; examination of the mucus, however, shows the presence of inflammation. The neuralgia begins with an attack of deep-seated pain near the root of the nose, the orbit, the upper jaw and teeth; in certain cases the area will include the lower jaw and teeth. From this area the pain spreads into the temporal region, the ear and the mastoid process, and is often severest about 5 cm. behind that process. This is the group of symptoms which Sluder calls "lower half headache." From here the pain may extend for varying distances into the occiput, neck, shoulder, arm, forearm, hand and fingers. Between the attacks a feeling of soreness or stiffness may remain. Other symptoms may be found such as raising of the soft palate on the affected side, pain or itching of the nose or throat, and more rarely a metallic taste in the mouth, scotoma scintillans, or salivation.

Later (1913-15)[3,4] Sluder added a second or sympathetic syndrome which, although it may occur separately, is closely associated with the neuralgic syndrome. It is characterised by a very severe and prolonged sneezing attack, rhinorrhœa and congestion of the nose, which may become red on the outer side. There may be lachrymation, conjunctival congestion, wide separation of the eyelids, photophobia and symptoms of asthma with dyspnœa and dry râles in the chest. If the condition becomes chronic there will be sneezing in the morning or rhinitis nervosa.

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The two syndromes may occur together, or isolated symptoms such as glossodynia, otalgia, photophobia and rhinorrhœa may be present independently.

A complete account of the two syndromes will be found in Sluder's book (*Headaches and Nasal Neurology*).

Sluder's views have been widely discussed, and there appears to be a fair amount of agreement that the syndromes are definite clinical entities. When, however, these writers come to consider the cause, there is considerable divergence of opinion.

Duverger and Dutheillet de Lamolle (1923)[5] divide Sluder's syndrome into three groups of symptoms: (*a*) vasomotor, with swelling of the nasal mucous membrane; (*b*) secretory, with copious nasal discharge; (*c*) sensory, with characteristic deeply-seated neuralgic pains. The first and second groups may be of an anaphylactic nature, but they state that the third is always due to the nasal ganglion and invariably begins with nasal infection. Examination of the nose is often negative and the diagnosis depends on the response to cocainisation of the ganglion.

Boebinger (1923),[6] on the other hand, believes that treatment of the ganglion is more successful with the sympathetic than the neuralgic symptoms.

Williams (1924)[7] thinks that two factors are concerned in spheno-palatine ganglion neurosis: (1) infection of the ganglion; (2) hereditary involvement of the endocrine organs and the sympathetic nervous system.

Worms (1924)[8] considers that the migrainous headaches are often due less to involvement of the ganglion than to reflex irritation from lesions of the nasal mucosa.

Bourquet (1925)[9] goes further than Worms and states that the causes of the whole condition are reflex impulses from the nasal mucosa.

III. Anatomy and Physiology of the Spheno-palatine Ganglion.

(*a*) **Relations.**—The spheno-palatine ganglion is a small triangular body placed deeply within the spheno-maxillary fossa. The nasal cavity lies to its inner side, and as the outer wall of the nose at this point is more or less deficient of bone, on account of the spheno-palatine foramen, the ganglion is

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therefore only separated from the cavity by the mucous membrane and some fatty tissue (Sluder, 1-9 mm.) which varies in amount according to the depth of the ganglion in the fossa. The sphenoidal sinus postero-superiorly and the posterior ethmoidal cells antero-superiorly have varying relationships to the ganglion and the spheno-maxillary fossa. The maxillary sinus is placed antero-inferiorly, but it is normally not so closely related to the fossa as the other sinuses.

The close relationship of the nasal cavity, the sphenoidal and the posterior ethmoidal sinuses to the spheno-maxillary fossa makes its contents very liable to irritation, when one of those spaces is inflamed, and has led to the opening of the sinuses in cases of obscure headache. The variable results obtained have been considered to be due to the varying distances of the sinuses from the fossa and ganglion.

(*b*) **Connections.**—It used to be customary to ascribe three roots to the ganglia associated with the trigeminal nerve. In the spheno-palatine ganglion the sensory roots were the spheno-palatine branches from the superior maxillary nerve. The motor root was the great superficial petrosal nerve and the sympathetic root the great deep petrosal nerve from the plexus on the internal carotid artery. These two roots unite and run to the spheno-palatine ganglion as the vidian nerve.

The modern conception is different. It is true that the three nerves, just described, run in close relationship to the ganglion; but the word *root* is now reserved for those fibres which have cell stations in the ganglion. The ganglia connected with the trigeminal nerve are known to be cell stations of the cranial parasympathetic outflow (cranial autonomic), therefore the parasympathetic part of the great superficial petrosal nerve is the only true root of the spheno-palatine ganglion.

There is another objection to the terms "sensory," "motor" and "sympathetic" being given to these nerves, as two of them, at least, contain fibres with different functions.

The great superficial petrosal nerve macroscopically connects the geniculate ganglion and the spheno-palatine ganglion, and contains two distinct groups of fibres, parasympathetic and sensory. The parasympathetic fibres run from the brain in the pars intermedia, pass through the geniculate ganglion and end in the spheno-palatine ganglion, where the post-ganglionic fibres arise. These fibres run mainly to the lachrymal gland via the orbital and temporo-malar nerves. There is, however,

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evidence that they also supply secretory fibres to the glands of the nasal mucosa, for Jung, Tagand and Chavanne (1926)[10] stimulated the vidian nerve in the dog and obtained a copious nasal secretion. They also found that section of the nerve diminished the amount of the secretion.

As the function of the parasympathetic system is normally antagonistic to that of the sympathetic, one might expect to find vasodilator fibres in the great superficial petrosal nerve. In support of this view there is the evidence of Brubaker (1916)[11] that stimulation of the peripheral part of the divided great superficial nerve with induced electric currents causes dilatation of the blood vessels and secretion of the glands of the nasal mucosa and its associated structures. Stimulation of the spheno-palatine ganglion and its branches gives similar results. It will be observed that this work of Brubaker agrees with the findings of Jung, Tagand and Chavanne that the great superficial petrosal nerve contains secretory fibres to the nasal glands.

The sensory fibres of the great superficial petrosal nerve arise within the geniculate ganglion in a manner similar to the sensory nerves in the posterior root ganglia of the spinal cord. The fibres, corresponding to the inflowing fibres of the posterior roots of the spinal nerves, run to the brain in the pars intermedia. *The peripheral fibres* course outwards in the great superficial petrosal nerve, *pass through the spheno-palatine ganglion without being interrupted*, into the descending palatine nerves, to end in the taste buds of the palate. They presumably carry gustatory impulses from the palate to the brain.

Rusken (1926)[12] claims, on clinical grounds, that the great superficial petrosal nerve supplies taste fibres to the tongue, but the evidence of Olmsted (1922)[13] appears to prove conclusively that such cannot be the case. This investigator cut the chorda tympani nerve in the dog and found that the taste buds in the anterior part of the tongue atrophied. He also mentions that Vintrelgau and Honigschmied had shown that a similar change occurs in the circumvallate papillæ when the glosso-pharyngeal nerve is divided. It is therefore clear that taste fibres to the tongue cannot be present in the great superficial petrosal nerve.

Mills (1910)[14] states that the chorda tympani nerve supplies taste fibres to the soft palate, but does not give any evidence for this statement.

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The great deep petrosal nerve consists of post ganglionic sympathetic fibres arising in the superior cervical ganglion. They pass through the spheno-palatine ganglion without interruption, and then run in the various nerves which are given off from the ganglion. Parsons Schaeffer believes that a few of the sympathetic fibres in the great deep petrosal nerve may be pre-ganglionic and have their cell stations in the spheno-palatine ganglion.

The above account of the vidian nerve is the one which is given by the modern British and American authorities. In the French literature three roots are described for the vidian nerve :—

(1) The great superficial petrosal nerve is the same.
(2) The great deep petrosal nerve is considered to be parasympathetic, its fibres coming from the glosso-pharyngeal nerve through the nerve of Jacobsen.
(3) The sympathetic fibres come from the cavernous plexus.

The sensory fibres come from the superior maxillary nerve via the spheno-palatine nerves. Some of the fibres pass through the spheno-palatine ganglion without interruption, while others either pass in front of, or external to, the ganglion. They run in the various nerves given off from the ganglion and carry impulses of common sensibility.

There are also a few post-ganglionic sympathetic fibres in the spheno-palatine nerves.

IV. Discussion.

Anatomy and Physiology: Sluder's Syndrome.—In this section it will be considered how far anatomical and physiological observations support the hypotheses of Sluder and other workers, whose results have been largely based on clinical experience.

It is clear that Sluder believes that the symptoms of his syndrome are due solely to irritation of the ganglion. It was shown in the last section that the spheno-palatine ganglion is a cell station for those parasympathetic fibres which serve as secretory fibres to the lachrymal gland, and probably secretory and vasodilator fibres to the nasal mucosa. We would expect, therefore, lachrymation and hypersecretion and congestion of the nasal mucous membrane to be prominent symptoms in

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cases of spheno-palatine ganglion irritation; if the vasodilator fibres do have such a wide distribution, which is uncertain, we could add congestion of the conjunctiva and the skin of the nose. These are the main features of Sluder's sympathetic syndrome, so that there appears to be fair anatomical and physiological support for his views, although it is a pity that he did not use the term "parasympathetic syndrome." It must be remembered, however, that irritation of the parasympathetic fibres themselves would cause the identical symptoms. Also it is difficult to imagine any cause that set up irritation in the ganglion which would not at the same time set up irritation in the fibres of the parasympathetic nerves. The evidence from treatment is no more convincing, as drugs applied to the nasal mucous membrane are just as likely to act on the parasympathetic fibres as on the ganglion cells. We are therefore brought to the conclusion that there is not sufficient evidence to show that Sluder's sympathetic syndrome is due to the spheno-palatine ganglion.

When an inquiry is made into the hypothesis that the "neuralgic" syndrome of Sluder is also caused by irritation of the spheno-palatine ganglion, it is found that anatomically and physiologically there are no grounds for such a view. How is it possible to suggest that an essentially efferent structure as the spheno-palatine ganglion can evoke such marked afferent symptoms as the deep-seated pains found in this condition? That the spheno-palatine ganglion, though closely associated with sensory nerves, is a part of the efferent nervous system cannot be too strongly emphasised. Writers on this subject have been misled time and time again on this point. The explanation of the pain, as pointed out in the article from Cushing in 1920, is that any pathological lesion which may set up irritation in the spheno-palatine ganglion is just as likely to irritate the main trunk of the superior maxillary nerve or its branches. The same objections to the evidence from treatment which were mentioned in discussing the sympathetic syndrome hold here, for the drug is just as likely to act upon the sensory nerves within the spheno-maxillary fossa as on the ganglion. Byrd (1930)[15] and others have denied this and stated that there are few or no signs of anæsthetisation of the superior maxillary nerve after the application of cocain or the injection of alcohol. So far as can be gathered from the histories of their cases, little or no attempt has been made to

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investigate the problem by the more modern neurological methods. The authors are at present carrying out an investigation on this point and hope to publish full reports at an early date. It can be stated, however, the results which have been obtained, up to the present, tend to show that the sensory nerves are actually anæsthetised.

Besides the "half headache" Sluder describes pain and itching of the throat, metallic taste, salivation, scotoma scintillans, and raising of the soft palate as symptoms which may occur in irritation of the spheno-palatine ganglion. Little need be said about most of these. What has already been stated about the other pain symptoms applies here. The metallic taste may well be due to irritation of the afferent fibres of the great superficial petrosal nerve. It is not possible to give any explanation for the scotoma.

The motor phenomena must be examined a little more closely. To explain the raising of the soft palate it must be assumed that the nerve supply to some of the palatal muscles pass through the ganglion or the nerves of the spheno-maxillary fossa. A reference to the standard text-books of anatomy shows that they all agree that the tensor veli palati muscle receives its nerve supply from the mandibular branch of the trigeminal nerve via the otic ganglion, while the remainder are innervated either by the spinal accessory or vagus nerves via the pharyngeal plexus.

The experiments of Brubaker (1916)[11] on stimulating the great superficial petrosal nerve show that there is no evidence that it contains any fibres for the supply of the palatal muscles.

Rich (1920)[16] investigated this question by observing the individual muscles while the intracranial parts of the various nerves were stimulated, and found that the inferior maxillary nerve supplied the tensor muscle and the bulbar portion of the 11th cranial nerve the levator.

There is therefore no anatomical evidence that any neurological structure in the spheno-maxillary fossa innervates the muscles of the palate. On the other hand there is the definite statement of a capable observer that such a condition can be observed. It is impossible at present to reconcile the two statements, and the solution of the problem must wait until further research has been done.

Ozæna.—A number of workers in France have claimed that irritation of the spheno-palatine ganglion is an important

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cause of ozæna. These claims are mainly supported by clinical observations and have been disputed by experimental workers.

Jung, Tagond and Chavanne (1926)[10] removed the spheno-palatine ganglion in animals and found no trophic changes in the nasal mucous membrane three to four months after. In a later paper they state that if the sympathetic be removed also, no changes take place either in the nasal bones or the mucosa. They favour the view that the changes in ozæna are due to destruction of the nerves and vessels of the mucosa by local suppuration.

Moulonguet and Collin (1929)[17] removed one of the nasal ganglia in the dog. After an interval they inoculated both nasal cavities with ozæna crusts. They then killed the animal and made a histological examination of the mucous membrane. At no stage did they see any difference between the two sides. They therefore concluded that removal of the ganglion was insufficient to cause trophic changes in the nasal mucosa.

Schatz (1929)[18] studied two patients in whom the spheno-palatine ganglion had been excised. One had also had a gasserectomy and in the other the superior cervical sympathetic ganglion had been removed. In both cases the nasal mucosa was unchanged five months after operation.

Against this strong evidence there is little to support the idea that the spheno-palatine ganglion causes ozæna except the clinical cases. There is, however, an interesting observation of Vogel (1929).[19] He was able to study the microscopic appearance of the various ganglia in a young female sufferer from ozæna who died under a general anæsthetic. The spheno-palatine ganglion was very atrophied, with great increase of connective tissue, round-celled infiltration near the veins and degenerative changes in the nerve cells. The gasserian ganglion showed an increase of the periganglionic cells. The other ganglia were normal. The author found that spheno-palatine ganglia removed from patients suffering from ozæna showed similar changes, whilst in a case of syphilitic rhinitis all the ganglia were affected. He therefore argues that there must be a close connection between ozæna and the spheno-palatine ganglion atrophy. He does not suggest which of the conditions is primary and which secondary. On account of the close anatomical relationship between the two structures we would expect to find changes in the spheno-palatine ganglion following the serious trouble in the mucous membrane.

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We may therefore conclude that there is little anatomical or physiological evidence in favour of the idea that trouble in the spheno-palatine ganglion is the primary cause of ozæna.

When the more distant symptoms are considered, it can be said definitely that they cannot be explained on any anatomical or physiological grounds. Various suggestions have been made by different writers and these will now be considered.

Sluder points out that there is little doubt that there are afferent fibres in the sympathetic. Now although nothing is known of the effects of a ganglion on the fibres which pass through it without interruption, he suggests, tentatively, that it is not difficult to imagine that irritation in the spheno-palatine ganglion might set up impulses in the afferent fibres of the sympathetic. The impulses might run back into the cervical sympathetic ganglia and there set up reflex secretory or sensory phenomena within the area of supply of a large part of the sympathetic. Many of the disconnected symptoms such as asthma or œsophageal spasm could be accounted for in this way. This suggestion would, of course, be a satisfactory explanation if it were proved, but at present it is purely hypothetical.

Rusken (1926) believes that the spheno-palatine ganglion can affect practically every region treated by the oto-rhino-laryngologist. He considers that the fibres of the great superficial petrosal nerve end in that ganglion, and that the nerve is a branch of the chorda tympani nerve, which is the sensory part of the facial nerve, which establishes connections with many other nerves. From this he argues that inflammation of the nasal ganglion would cause irritation in the terminal fibres of the great superficial petrosal nerve and so set up referred pain in the wide distribution and connections of the facial nerve.

In criticism of this statement it must be repeated that only the efferent fibres of the great superficial petrosal nerve end in the ganglion, the afferent ones passing through it from the peripheral nerves. Inflammation of the ganglion cannot therefore irritate the nerve endings of the sensory part of the great superficial petrosal nerves. Let it be granted that the pars intermedia, from which the chorda tympani and great superficial petrosal nerves arise, is in part the sensory root of the facial nerve. This does not prove that sensory fibres of the pars intermedia are to be found in all the branches of the 7th, and far less does it prove that the many connections between the motor part of the facial nerve and the other cranial nerves

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are also connections of the pars intermedia. Such an assumption cannot be made—indeed all the evidence shows that these afferent fibres are limited to carrying taste impulses from the tongue and palate, and in their peripheral path do not run with the facial nerve at all, but with the trigeminal.

In an article in 1928[20] Rusken states that the cranial autonomic fibres, besides going to the nasal ganglion, also run in the various branches of the facial. He therefore suggests that irritation of the trigeminal twigs of the autonomic nerves may set up reflex impulses which will cause vasomotor spasm in the region of distribution of the 7th nerve, and as this spasm will interfere with the metabolism of the muscle, local symptoms will occur. He suggests that this mechanism would explain the good results obtained in cases of shoulder pain, lumbago and sciatica. There are grave objections to this hypothesis in that there is no evidence that the peripheral branches of the facial nerve contain cranial autonomic fibres. There is the further difficulty that there is no evidence that the cranial autonomic nerves are vaso-constrictors—indeed we have already shown that they are probably vaso-dilators.

Rusken also thinks that spastic states of the gastro-intestinal tract may be caused by impulses from the spheno-palatine ganglion depressing the vagus nerve. A somewhat similar view is held by Heitger (1926),[21] who wrote on the subject of brachial neuritis due to the spheno-palatine ganglion. He suggests that the path of the impulse is from the spheno-palatine ganglion, the gasserian ganglion, the spinal sensory root of the trigeminal, and then by radiations to the sensory nerves in the cervical cord.

It appears to the authors that if the spheno-palatine ganglion is responsible for trouble in remote parts of the body, that the explanation will have to be sought in some central connections, as is suggested in the last paragraph.

Clinical Review.

Neuralgia.	F.	M.	Unknown.	Total.
Sluder's syndrome under various names .	42	12	2	56
Sluder's syndrome and other symptoms .	17	6	0	23
Total . .	59	18	2	79

Names of Diseases.—Lower half headache, migraine, spheno-palatine neuralgia, neurosis or syndrome, tic-douloureux, neuralgia, sympathetic pain, maxillary and vidian neuralgia, headaches named after different regions of head.

One author does not stick to one name.

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	Age 20-29	30-39	40-49	50-59	60-69	70-79
F.	9	19	11	5	4	1
M.	4	4	4	3	0	1
Total	13	23	15	8	8	2

Treatment.—Cocainisation, 43 cases all relieved. Alcoholic injection of phenol alcohol, 30. Complete relief eight months and more, 15. Incomplete relief, 6. No relief, 9. Dangerous bleeding, 2.

Bonain's fluid	Relief, 1	
Endocrines	,, 4	
Silver nitrate	,, 1	
0·4 per cent. formol	,, 3	
Opening sphenoid	,, 1	Failure, 1
Extraction tooth	,, 1	
Salvarsan	,, 1	
Submucous resection and cocainisation		Cure, 1
Injection and tonsillectomy		,, 1

	F.	M.	Unknown.	Total.
Secretory and vascular disturbances nose	12	4	3	19
Secretory and vascular disturbances nose and other symptoms	9	1	0	10
Total	21	5	3	29

Names of diseases included in group:—Vasomotor rhinitis, hyperæsthetic rhinitis, hay fever, nasal discharge, coryza, hydrorrhœa, secretory spheno-palatine neurosis, sneezing.

	Age 20-29	30-39	40-49	50-59	60-69
F.	6	6	2	2	0
M.	0	1	0	1	1
Total	6	7	2	3	1

Treatment.—Cocainisation ganglion, 14 cases all relieved. Injection ganglion, 14 cases—complete relief, 12; incomplete, 2. Bonain's fluid relief, 1. Corpora lutea, 1. Silver nitrate to ganglion—cure, 4; marked relief, 1; partial relief, 1. Anæsthetisation ganglion and submucous resection—cure, 1.

	F.	M.	Total.
Asthma	4	1	5
Asthma and other symptoms	5	0	5
Total	9	1	10

	Age 20-29	30-39	40-49	50-59	60-69
F.	1	3	2	1	1
M.	1	0	0	0	0
Total	2	3	2	1	1

Treatment—Cocainisation, 7 all relieved. Injection, 5 cases complete relief, 1 partial relief. One case treatment of ganglion gave no result. The condition was cured by opening the sphenoids. Removal turbinates and 8 per cent. Ag_2O, 1 case cured.

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Spheno-Palatine Ganglion

Disease.	Female.	Male.	Not stated.	Cocain.			Injection.			Other Treatment applied to Ganglion Region and Remarks.
				Relief.	Partial.	Nil.	Relief.	Partial.	Nil.	
Chorea	0	1	0	...	...	...	1	...	...	.....
Diarrhœa	1	0	0	1	...	...	...	...	...	.....
Earache and Itching	13	4	0	11	2	2	3	...	...	Cocain and ag. nit. cure.
Eczema	1	0	0	1	...	...	1	...	...	.....
Eye, Blepharospasm	1	2	0	1	...	...	1	1	1	.....
,, Conjunctivitis	0	1	0	...	...	...	...	...	...	Trichloracetic and relief.
,, Episcleritis	0	1	0	1	...	...	...	...	...	Ag. nit. cured condition.
,, Iritis	1	1	0	2	...	...	...	...	...	1 case ag. nit. cure.
,, Pain	1	0	0	1	...	...	...	...	...	1 case carbolised oil cure.
,, Ptosis	1	...	...	1	...	...	...	...	...	.....
,, Photophobia	1	1	0	2	...	...	...	...	...	.....
,, Scotoma	1	1	0	...	...	...	1	...	1	.....
,, Glaucoma	5	4	1	6	3	...	2	...	...	.....
Glossodynia	6	1	0	5	...	...	2	...	2	.....
Herpes, Intercostal	1	0	0	1	...	...	...	...	...	Relieved pain only.
,, Lingual	1	0	0	1	...	...	...	...	...	,, ,, ,, Ag. nit. cure.
,, Oticus	1	0	0	1	...	...	...	...	...	,, ,, ,,
,, Neck and Upper Arm	0	1	0	1	...	...	...	...	...	,, ,, ,,
,, Frontal, etc.	0	2	0	...	...	...	...	...	...	1 case cocain in sphenoid sinus. Relief. 1 case nasal nerve relief.
Hiccough	1	2	0	1	...	2	1	1	1	.....
Lumbago	0	1	0	1	...	...	1	...	...	.....
Otosclerosis	2	2	2	...	...	...	...	...	...	Cocainisation gave varying relief.
Nausea	4	...	...	2	...	...	...	...	...	Other 2 not stated.
Pain, Parietal	1	...	...	...	...	...	...	...	...	Ag. nit. per cent. cure.
,, General	1	0	0	1	...	...	...	...	...	.....
,, Intercostal	2	0	0	1	...	...	...	...	...	.....
,, Lower Limb	0	1	0	...	...	1	...	...	...	Spasm associated was relieved by cocain.
,, Arm	2	1	...	2	...	...	...	...	...	.....
,, Middle Finger	...	1	...	1	...	...	...	...	...	.....
,, Sciatica	2	4	0	2	2	2	...	...	...	The females were the cases relieved.
,, Throat	1	1	0	2	...	...	1	...	...	.....
,, Tooth	1	0	0	1	...	...	...	...	...	.....
,, T. B. Laryngitis	1	1	0	2	...	...	...	...	...	.....
Parageusia	2	1	0	3	...	...	1	...	...	.....
Paralysis, Contracted Ulnar	0	1	0	...	1	...	...	...	...	.....
Sensation F.B. Throat	1	...	...	1	...	...	...	...	...	.....
Spasm, Œsophagus	1	0	0	1	...	...	...	...	...	.....
Stiffness, Shoulder	0	1	0	1	...	...	1	...	...	.....
Stiffness, Neck	1	...	...	1	...	...	...	...	...	.....
Stomach, Ache	1	0	0	1	...	...	...	...	...	.....
Tinnitus	1	0	0	...	1	...	...	...	...	.....
Vertigo	4	1	0	3	0	0	...	...	...	Corpora lutea, etc., relieved 1 cure.
Weakness, Arm	0	1	0	1	...	...	...	...	...	.....
Writer's Cramp	0	1	0	1	...	...	...	...	...	.....

The appended list of diseases and symptoms which have benefited by thorough treatment of the spheno-palatine ganglion is impressive. So impressive, perhaps, as to make one

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suspicious, there being seemingly no region of the body which is free from the influence of this ganglion.

There can be no doubt that the lower half headache is a very real clinical entity, but there is much room for doubt, on purely anatomical grounds, as to its cause being as stated by Sluder, as we have already stressed. We find on looking at the statistical return that an overwhelming percentage of the cases are "females," and that the third and fourth decades of life hold the greatest number of reported cases. It is reasonable, therefore, to wonder how many of these cases are functional and how many organic? The subsequent history of many of the "cured" cases (regardless of the method of cure) are missing and leaves one still uncertain as to the permanent value of direct treatment to the ganglion.

The likelihood of benefit resulting from transmural injection of the spheno-maxillary fossa being due to diffusion of the fixative to the branches of the superior maxillary nerve is stressed by Cushing,[22] but in the same paper the author admits that there are cases of neuralgia in the area of the maxillary nerve which derive no benefit from the neurectomy.

We have seen that the widespread nature of the pain has been attributed to the connection which the great superficial petrosal nerve has with the facial through the pars intermedia, but anatomists are not willing to admit that the sensory fibres of the facial nerve are as widespread as are those of the motor fibres, yet on clinical grounds there is at any rate reason to give a richer sensory supply to the facial nerve than anatomists are wont to do. Souques and Hartman[23] found that after resection of the retrogasserian portion of the 5th nerve deep sensibility is still present. This they say is either a sympathetic or facial nerve function, and quote a clinical case to support their belief in the latter explanation. A woman had a complete left facial paralysis except for the orbicularis palpebrarum muscle following the removal of a parotid tumour. Later it was necessary to perform a retrogasserian resection of the left 5th nerve, after which it was found that deep sensibility was lost over the left side of the face except over the orbicularis, where deep sensibility persisted.

The more widespread distribution of pain to the neck and upper limb has been explained by the fact that there is anatomical continuity of the sympathetic nervous system from the nasal ganglion, the carotid plexus and the superior cervical

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ganglion, and finally by the grey rami to the spinal cord and the spinal nerves to the extremities. From the anatomical continuity, without much justification to our mind, observers have explained this widespread control which the spheno-palatine ganglion is given credit for exercising over pain of all kinds.

Glasser[24] reviews 143 cases of atypical neuralgia, and his arguments are interesting. "As a class," he writes, "the victims of these irregular and atypical neuralgias are a pathetic lot. They often date their discomfort back many years, and they insist that the pain is unbearable and difficult of description, yet they seldom show any evidence of the intense suffering of which they complain."

Glasser admits that he is nonplussed by these cases, and finds that no local remedies or direct treatment to structures within the territory of the pain, especially teeth and the sinuses, will give the slightest relief. In ten of the cases which he found to be suffering from the sympathetic syndrome (or what we prefer to call the parasympathetic syndrome) Frazier resected the superior cervical ganglion, but this, like other treatments, failed entirely.

Dr Byrd, an American observer, is perhaps the most ardent worshipper at the shrine of the nasal ganglion, and his theory of "pathological impulses" in relation to the ganglion is worth quoting.[25] The following is an extract of the author's summary:—

1. Pathological impulses are, within the meaning of this article, real entities.
2. Pathological impulses may originate in either nasal bloc (or possibly elsewhere), from whence they are, or may be, transmitted to the remotest parts of the body.
3. Pathological impulses, it is conceivable, of such low amperage as to produce no appreciable effect, may be abroad in the body.
4. Pathological impulses are attracted to pathological loci, wherever such loci occur on the circuit of the impulse.
5. A pathological impulse attracted to a pathological locus co-operates with the latter in establishing a vicious circuit.

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6. It is conceivable that a pathological impulse may take advantage of a slight impairment, such as fatigue, or momentary chilling of a part, or slight strain, to insinuate itself in and thus set up a vicious circuit.

7. A pathological impulse originating in either nasal bloc is not transmissible to its fellow of the opposite side, but is transmissible to both shoulders, arms and trunk, and to both lower extremities.

8. Pathological impulses have, in my experience, been definitely connected with a large number of phenomena.

This is the article in which Byrd elaborates his hypothesis and is possibly therefore worth reading. It appears to have been accepted by many American rhinologists as a satisfactory explanation of the cures obtained by cocainising the nasal ganglion. We do not believe any physiologist or neurologist would give it a moment's consideration. If the facts about results of cocainising the nasal ganglion are true, which requires proof, it will be necessary to form a more plausible hypothesis.

In a late paper [26] the same author recommends the application of cocain to all obscure cases. "It matters not," the author continues, "what the subjective symptom, or where it may be, that brings doctor and patient together, the nasal ganglion test, whether the results be positive or negative, always yields valuable information." Apparently if the symptom is controlled by the test it can be followed by cocain or alcohol as a therapeutic measure.

The following cases, quoted by H. Byrd,[27] give some idea of the author's unfailing belief in the ganglion's powers:—

CASE I.—"I developed a sensitiveness at the second joint of the middle finger of the right hand. This sensitiveness remained with me constantly . . . for a period of about nine months. Then I discovered that cocainising the left nasal ganglion would intercept the current, giving complete relief, for a period of several hours (after a few cocainisations of the nasal ganglion it recovered entirely)."

CASE II.—"A girl, about 20, developed a sore spot at the lower border of the ribs just posterior to the right lateral aspect. She dated it to the cranking of a Ford. . . . It remained with her constantly . . . for a period of four years. At this time it was found that

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cocainisation of the left nasal ganglion relieved it entirely and . . . it never returned."

The author argues that in these cases a state of chronic irritation was set up by a pathological current. Now suppose this chronic irritation has been set up by the current in an area susceptible to cancer, is there not a possibility that it would have set up cancer?

There are many cases quoted in the list which we have compiled from the literature at our disposal which we must be content to accept at their face value, as the clinical notes are scanty and reveal obvious flaws in the way they have been investigated. In some cases the major credit has been given to the spheno-palatine ganglion treatment, where it has been a part only of the treatment directed to the condition.

Most striking are some of the cases of glaucoma where eserine has also been used, and yet the therapeutic value of this drug appears to have been dwarfed in the minds of the observers in their regard for the nasal ganglion.

That otosclerosis, as we understand it on this side of the Atlantic, can be cured, or even the deafness alleviated by cocainising the nasal ganglion, is difficult to understand. In fact, where fixation of the stapes in the oval window is present there can surely be no question of this comparatively simple treatment doing any good at all. We note that one writer says that the application of cocain to the ganglion causes contraction of the tensor tympani and thus movements may be transmitted to the ossicular chain, and he makes this one form of treatment for deafness.

Authors' Conclusions.

1. There exists a neuralgic type of pain of widespread distribution about the head and neck which differs from the true tic-doloureux, and that its cause is probably due to nasal infection but its underlying neurological nature is still obscure.

2. The control of the so-called sympathetic syndrome can be understood though the mechanism may not be as described by Sluder.

3. The trophic influence which the spheno-palatine ganglion is said to exert over the nasal mucosa (*e.g.* in cases of ozæna) is not in keeping with experimental evidence.

4. Any phenomena obtained as a result of treatment to

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the region of the spheno-palatine cannot be separated from phenomena resulting from involvement of the nerves of the spheno-maxillary region as a whole.

5. The anatomical explanation offered by the supporters of the ganglion theory are not founded on the conception of the anatomy of the region as understood by pure anatomists.

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Die Verfasser beschreiben die Arbeit seit Sluder's erstem Blatt.

Nachdem man die historische Anschauung geschildert hat werden die kleinsten anatomischen Verbindungen des Ganglien besprochen.

Darauf besprechen sie ausführlich wie weit die anatomische und physiologische Beobachtungen die Hypothesis von Sluder und von den anderen aufrecht erhalten.

Es gibt eine clinische Abteilung welche zur Beschreibung der Fälle und der verschiedenen Methoden und derer Behandlung gewidmet ist.

Les auteurs discutent l'étude sur le ganglion sphénopalatin depuis le premier article de Sluder.

Ils font un aperçu historique ; puis ils entrent dans les minutieuses liaisons anatomiques du ganglion.

Puis ils considèrent enfin à quel point les observations anatomiques et physiologiques viennent à l'appui de l'hypothèse de Sluder et des autres practiciens.

Il y a une section clinique consacrée à l'exposé des cas et des méthodes varieés de traitments qui ont été employés.

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Spheno-Palatine Ganglion

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