

A further contribution to the herpetic inflammations of the geniculate ganglion : a syndrome characterized by herpes zoster oticus, facialis, or occipitocollaris, with facial palsy and auditory symptoms / by J. Ramsay Hunt.

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Publication/Creation

[Philadelphia] : [Lea Bros.], 1908.

Persistent URL

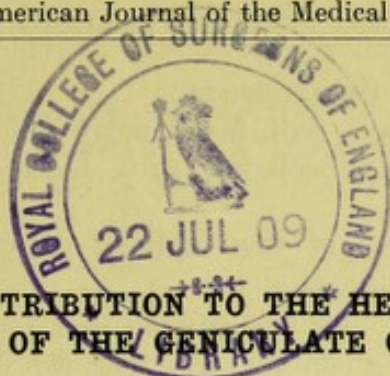
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A FURTHER CONTRIBUTION TO THE HERPETIC INFLAMMATIONS OF THE GENICULATE GANGLION.

A SYNDROME CHARACTERIZED BY HERPES ZOSTER OTICUS, FACIALIS, OR OCCIPITOCOLLARIS, WITH FACIAL PALSY AND AUDITORY SYMPTOMS.

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THIS communication is a further elaboration of a syndrome presented at the American Neurological Association, in June, 1906.¹ At that time, three well-recognized clinical types of herpes zoster of the face and neck, with facial palsy and acoustic symptoms, were fused into a single group. The characteristic symptoms were attributed to a specific, herpetic inflammation (*posterior poliomyelitis*) of the geniculate ganglion of the facial nerve; and the neural complications, to an extension of the inflammatory process to the adjacent seventh and eighth nerves (Fig. 1). The zoster zone for the geniculate ganglion was thought to be intercalated between the zones of the Gasserian in front and the cervical ganglia behind, and situated on the tympanum, auditory canal, and interior parts of the auricle.

The distinctive features of the clinical picture are as follows: Herpes facialis, herpes oticus, or herpes occipitocollaris, with facial palsy; and in some of the cases irritative and paralytic symptoms referable to the acoustic nerve.

The subject matter of the paper may be conveniently considered under the following headings:

- I. The syndrome and its clinical types.
 - A. Herpes oticus.
 - B. Herpes oticus, with facial palsy.
 - C. Herpes oticus, with facial palsy and hypo-acousis.
 - D. Herpes oticus, with facial palsy and Ménière's complex.

¹ Herpetic Inflammations of the Geniculate Ganglion: A New Syndrome and its Complications, Trans. Amer. Neur. Assoc., 1906; Jour. of Nerv. and Ment. Dis., February, 1907; Archives of Otology, August, 1907.

II. Sub-groups of the syndrome.

Herpes facialis, with facial palsy and auditory symptoms.

Herpes occipitocollaris, with facial palsy and acoustic symptoms.

Herpes zoster of the cephalic extremity, with auditory nerve complications.

III. Multiple involvement of ganglia in herpes zoster.

IV. The cranial nerve ganglia and their relation to herpes zoster.

V. The zoster zone of the geniculate ganglion.

VI. Sensory symptoms in the trigeminal and cervical zones in herpes zoster oticus.

VII. The ganglia of the glossopharyngeal and vagus nerves and their probable role in zona.

VIII. Concluding remarks.

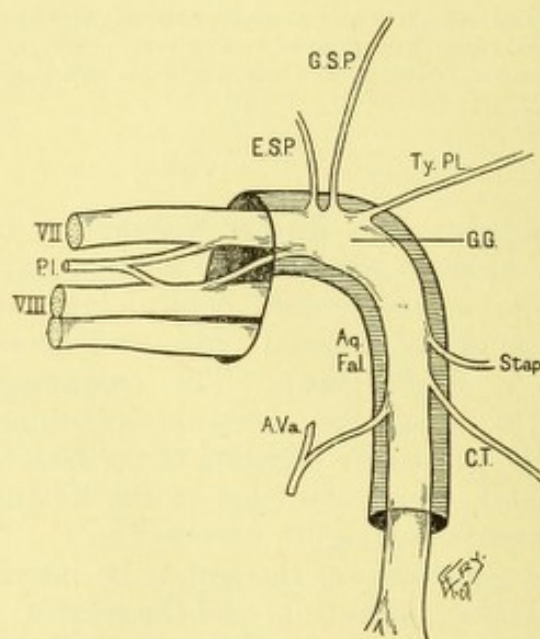


FIG. 1.—The geniculate ganglion, its relations to the facial and auditory nerves: VII, facial nerve; P.I., pars intermedia; VIII, auditory nerve; Aq. Fal., aqueduct of Fallopius; G.G., geniculate ganglion; E.S.P., external superficial petrosal; G.S.P., great superficial petrosal; Ty. PL., branch to the tympanic plexus. (Cunningham's Anatomy.)

I. THE SYNDROME AND ITS CLINICAL TYPES. *Group A. Herpes Oticus.* This, the simplest expression of the affection, is dependent upon an herpetic inflammation (*posterior poliomyelitis*) of the geniculate ganglion. It may be preceded by the mild prodromal symptoms of herpes zoster, with preherpetic pains localized in the ear and mastoid region (otalgia). The eruption, which varies considerably in extent, is distributed over the tympanum, external auditory canal, concha, tragus, antitragus, helix, antihelix, and occasionally overlaps into an adjacent marginal zone. In this cone-shaped area is the ganglionic representation and zoster zone of the geniculate ganglion.

In some cases the auricle may be considerably swollen, red, and tender, standing out somewhat from the side of the head. Often the meatus and canal are so reduced in size as to render the introduction of a speculum difficult and painful. Naturally, when the conduction mechanism of the external ear is infiltrated and swollen tinnitus and some temporary deafness may result. This must be carefully distinguished from neural deafness, which will be described in other groups of the affection. The inflammatory swelling subsides rapidly, the eruption desiccating and disappearing in about a fortnight. Postherpetic otalgia, in some cases, may persist for a considerable time.

Group B. Herpes Oticus, with Facial Palsy. In addition to herpes oticus there is an associated peripheral facial palsy on the corresponding side. The paralysis is complete, involving all branches of the nerve, making its appearance with or soon after the eruption becomes manifest. All degrees of severity have been observed, from a very transient form, lasting only a few days, to that which leaves a permanent weakness and contracture. In this group the adjacent facial nerve has been involved in the inflammatory process.

Group C. Herpes Oticus, with Facial Palsy and Hypo-acousis. In this group, with herpes oticus and facial palsy, there is an associated disturbance of hearing. This consists of a diminution of the acuity of audition with or without tinnitus aurium, and is a milder type of acoustic disturbance. It may be quite transient. It is to be ascribed to an extension of inflammatory products to the auditory nerve which is close by.

Group D. Herpes Oticus with Facial Palsy and Ménière's Complex. This is a very severe manifestation and has associated with the foregoing symptoms all the characteristics of Ménière's syndrome: tinnitus aurium, deafness, vertigo, nystagmus, vomiting, and disturbances of the equilibrium.

This group may also be explained by the extension of inflammatory products along the nerve sheaths to the terminations of the auditory nerve. (It is in my opinion very probable that the *ganglia* of the auditory nerve may be primarily involved in the specific inflammations of zona.)

REMARKS. The four groups of cases just outlined are all dependent upon specific herpetic inflammations of the geniculate ganglion, representing different degrees of severity. The eruption is confined to the geniculate ganglion zone; the other zoster zones of head and neck are free. The neural complications result from the proximity of the inflamed ganglion to the seventh and eighth nerves.

II. SUB-GROUPS OF THE SYNDROME. *Herpes Facialis with Facial Palsy and Acoustic Symptoms.* In this clinical type the eruption makes its appearance upon the face, an area represented by the Gasserian ganglion; the zoster zone of the geniculate is

free. Facial palsy may be the sole neural complication, as in herpes oticus (Group B), or there may be associated disturbances of the auditory nerve.

Herpes Occipitocollaris, with Facial Palsy and Acoustic Symptoms. The herpetic eruption in this group is distributed over the occiput, the side of the neck, the inner surface and the posterior half of the outer surface of the auricle, and a patch corresponding to the angle of the jaw (herpes occipitocollaris). This skin area represents the zoster zones for the second, third, and fourth cervical ganglia. The zones for the geniculate and Gasserian ganglia are free. The neural complications do not differ from those described in the previous group.

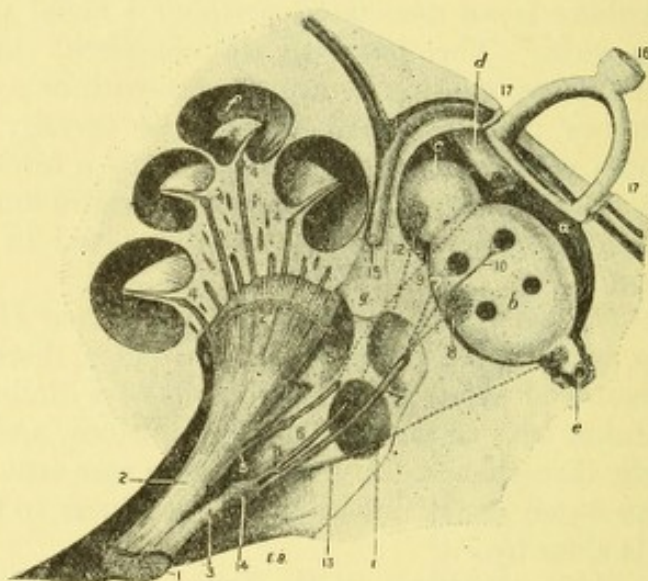


FIG. 2.—The ganglia and terminations of the auditory nerve: 1, auditory nerve; 2, cochlear branch; 3, vestibular branch; 4, ganglion of Corti; 6, ganglion of Boettcher; 14, ganglion of Scarpa; 15, facial nerve. (Testut's Anatomy.)

Herpes Zoster of the Cephalic Extremity, with Auditory Complications. The occurrence of auditory complications in connection with herpes facialis, occipitocollaris, and herpes oticus, have already been mentioned. With the auditory symptoms there has been associated a peripheral facial paralysis. In my first paper this involvement of the auditory nerve was ascribed to an extension of the inflammatory process from the geniculate to the auditory nerve and its terminations, the envelopment of the facial, the nerve of Wrisberg, and the acoustic in a common sheath in the depths of the internal auditory canal favoring such an extension.

The great severity, however, of the acoustic disturbances in some of these cases, and their persistence, has suggested the thought that the acoustic ganglia themselves may be the seat of a primary inflammatory involvement (Fig. 2). It will be recalled that the cells of the ganglion of Scarpa and ganglion of Corti take their origin from the neural ridge, an embryonic structure from which the other

ganglia liable to zoster are derived. Clinical confirmation of this theory is found in the occurrence of acoustic symptoms in herpes zoster of the face, without an associated facial paralysis. In cases of this type, if the acoustic nerve were involved by reason of its proximity to the geniculate ganglion, the facial nerve could hardly escape a simultaneous involvement.

The following cases show that auditory symptoms may accompany herpes facialis and herpes oticus, without a corresponding facial palsy:

Casse.² Observation I. A typical herpes zoster of the third division of the fifth nerve, in a woman, aged fifty-eight years. Accompanying these manifestations in the trigeminal distribution, there were pains in the auditory canal, *roaring in the ear*, and *diminution of hearing*. The face was not paralyzed. Two months later there was still tinnitus aurium and slight diminution of hearing. The examinations of the ear, throat, and nose were negative.

Burin-Desrozieres.³ Observation II. A man, aged thirty-one years. Exposure to cold October 7, 1904, followed by neuralgic pain in the right side of the face; on October 14 an eruption of herpes zoster developed on the right side of the face. On October 18 the neuralgic pain had practically disappeared, but there persisted a certain "*hardness of hearing*" on the right side. Facial palsy was not observed.

Gruber,⁴ in recording two cases of herpes zoster, limited to the external auditory canal (herpes oticus), states: "In both cases there was hardness of hearing and tinnitus, which was out of proportion to the lesion in the external auditory canal. It would seem as if, in these cases, the auditory nerve was involved perhaps reflexly. The opening of the vesicles did not relieve the hearing, which only returned with the disappearance of the other symptoms."

It seems to me, therefore, very probable that the acoustic ganglia may be primarily involved in the specific inflammation of posterior poliomyelitis.

REMARKS. In the chief group of the syndrome, it will be observed that the eruption is situated in the geniculate area (herpes oticus). In the sub-group it is distributed, either in the zone of the Gasserian ganglion (herpes facialis), or in the zones of the upper cervical ganglia (herpes occipitocollaris). In all, the neural complications, if present, are identical in sequence and symptomatology. I would also emphasize the group with herpetic eruption and auditory symptoms, without facial palsy. These cases may depend upon a primary involvement of the ganglia of the acoustic nerve, as it would seem very improbable that an inflammation could extend from the geniculate to the auditory nerve, without at the same time com-

² Zona de la troisième branche du trijumeau, Thèse de Paris, 1902.

³ Ibid., 1904.

⁴ Herpes Auricularis, Monatssch. f. Ohrenheilk., 1875, Nr. 5.

promising the facial. The possibility of slight or transient facial palsies escaping observation, must, however, be kept in mind.

In my previous paper I collected 60 cases of herpes zoster of the cephalic extremity, with facial palsy; in 19 of these cases auditory complications were also present. The eruption in this series of cases had the following distribution:

	Case ¹ .
Herpes oticus	12
Herpes facialis	12
Herpes occipitocollaris	32
Herpes oticus and occipitocollaris (combined)	3
Herpes facialis and occipitocollaris (combined)	1

I would here remark that in the literature I found no recorded case of facial palsy and acoustic disturbance, complicating herpes zoster, in which the eruption was not situated on the cephalic extremity of the body. The question as to the occurrence of neural complications in herpes facialis and herpes occipitocollaris, the zoster zone for the geniculate being free from any eruption, I will consider under the next heading.

III. MULTIPLE INVOLVEMENT OF THE GANGLIA IN ZONA. Herpes zoster is an acute, specific, infectious disease, characterized by an inflammatory reaction in ganglia containing the so-called *spinal* or *unipolar type* of cells. Ganglia of this type are the spinal ganglia of the posterior roots, the Gasserian and geniculate ganglia of the cranial nerves. The localization of a specific inflammatory process in the ganglia of the *spinal type* was termed Posterior Poliomyelitis by Head and Campbell, and the similarity of the pathological changes to those occurring in anterior poliomyelitis was pointed out. The fact was also emphasized that the essential pathological lesion is generally limited to a single ganglion, which corresponds to the eruption of zoster.

That more than one ganglion may be involved is shown clinically by the occurrence of double, triple, bilateral, and alternate forms of zona, the eruption appearing in more than one zoster zone. This tendency to involvement of more than one ganglion is especially frequent about the head and neck. I would call special attention to the fact that clinical evidence exists showing that some ganglia may be implicated in zona, without the occurrence of an eruption in their respective zones.

Symptoms indicating such involvement are to be found in the neuralgic pains and mild sensory disturbances (*hypalgesia* and *hypesthesia*) occasionally observed in zones adjacent to the chief or eruptive focus.^{5 6} It is also possible that the so-called vesicles aberrantes⁷ may be regarded in the same light. In zona the

⁵ Champion, *Manifestation à distance dans la Zona*, Thèse de Paris, 1900.

⁶ Weisenburg, *Herpetic Inflammation of the Cervical and Thoracic Nerves*, Jour. Nerv. and Ment. Dis., 1907, p. 726.

⁷ Tenneson, *Vesicules aberrantes du zona*, Bull. de la Soc. méd. des hôp., 1898.

ganglion which corresponds to the eruption is to be regarded as the chief or *eruptive focus*. The inflammatory reaction is not confined solely to this ganglion, but inflammatory changes of a milder grade may also be present in adjacent ganglia, above and below. In other words, there may be a serial involvement of ganglia, the extent and intensity of which depends upon the severity of the infection. Such a theory of inflammatory reaction in a chain of ganglia, which shades off from the chief or eruptive focus, is also more in harmony with the acute, specific, toxic nature of herpes zoster, and the analogy which it bears to the anterior poliomyelitis, the chief or eruptive focus of the one corresponding to the permanent paralytic focus of the other, while the transient and mild sensory manifestations of herpes are the sensory equivalent of mild and transient palsies. I attach a special importance to this inflammatory involvement of a series of ganglia, in the relation which it bears to the sub-groups of the syndrome—the *herpes facialis* and *herpes occipitocollaris* with neural complications.

The Gasserian, geniculate, and upper cervical ganglia may be regarded anatomically as representing a more or less continuous ganglionic chain. Hence, if the chief inflammatory or eruptive focus were situated in any one, a milder, non-eruptive, inflammatory reaction might occur in other ganglia of the group. In herpes zoster of the face, with neural complications, the chief or eruptive focus is situated in the Gasserian ganglion; there has, however, been a coincident inflammatory reaction in the geniculate, sufficient to disturb the function of the adjacent nerves, without producing an eruption in the geniculate area. In the same manner, I would explain those cases in which, with neural complications, the eruption is confined to the zones of the second, third, and fourth cervical ganglia.

The neural complications (seventh and eighth nerves) observed in herpes zoster of the cephalic extremity may, therefore, be regarded pathologically as depending upon an herpetic inflammation of the geniculate ganglion.

Pathological Evidences of Multiple Involvement of Ganglia. The elaborate pathological researches of Head and Campbell⁸ are now so well known that I will consider this phase of the subject only in its relation to multiple or serial involvement of ganglia; and its bearing upon the existence of lesions of slight degree in ganglia near the chief or eruptive focus.

Histological studies in cases of zona show that the ganglion corresponding to the eruption is the seat of well-marked inflammation and hemorrhages. In the acute stage the ganglion is swollen and cedematous, and is the seat of extensive round-cell infiltration and hemorrhages; the cells are in various stages of degeneration;

⁸ Pathology of Herpes Zoster, Brain, 1905.

later, as the inflammation subsides, there is connective tissue proliferation, and in severe cases a permanent scar results. In both acute and chronic stages secondary changes are demonstrable in the corresponding posterior root, as well as in the peripheral nerves.

I will here briefly analyze the pathological evidence existing at the present time of serial ganglion involvement, which, in my opinion, furnishes a pathological basis for the mild sensory symptoms already mentioned as manifesting themselves in the adjacent non-eruptive zones.

In order to demonstrate the presence of a mild inflammatory reaction in ganglia adjacent to the chief or eruptive focus, quite recent cases would be the most favorable, if not essential. In the event of the process being older, such changes would be more readily demonstrable in the posterior roots than in the ganglia themselves.

In the descriptions of Head and Campbell it would appear that the inflammatory changes in zoster are limited to a single ganglion—that which corresponds to the eruption. Essentially this is true, although the presence of milder changes, if present, should receive due weight. Their statement as to multiple involvement is as follows: "It is probable that two ganglia are occasionally affected together. From clinical observation, this seems particularly liable to occur with the second, third, and fourth cervical. Unfortunately, no such case is included in our series of postmortem examinations."

It will be observed, however, from the following abstracts of some of their observations, that mild pathological changes were noted in ganglia adjacent to the eruptive focus and yet apparently having no corresponding zoster manifestations.

CASE I.—Herpes zoster in first lumbar distribution of left side; three days duration. Left first lumbar ganglion—typical inflammatory changes present. Right first lumbar—the cells do not stain well. Left and right twelfth dorsal ganglia—bloodvessels are much engorged. Left and right second lumbar ganglia—neither show any marked change.

CASE IV.—Herpes zoster in twelfth dorsal distribution of right side; three days duration. Right twelfth dorsal ganglion—typical inflammatory changes present. Left twelfth dorsal—right and left eleventh dorsal were free from noteworthy alterations.

CASE V.—Herpes zoster, eleventh dorsal distribution of left side; fourteen days duration. Left eleventh dorsal ganglion—typical inflammatory changes were present; the remaining ganglia were normal. There were marked secondary degenerations in the posterior root of the eleventh dorsal on the left side. *A few definitely degenerated fibers were seen in the left twelfth dorsal root.*

CASE VI.—Herpes zoster in left third cervical distribution; fifty-seven days duration. In left third cervical ganglion and left third

cervical posterior root the typical changes were present. *Some bundles of the fourth root on the left side also seemed to have undergone change, but this was a less certain change than that of the third root.*

CASE VII.—Herpes zoster in left second dorsal region; ninety-six days duration. Typical changes in left second dorsal ganglion and posterior root. *In first dorsal root a few degenerated fibers could be recognized.*

It will thus be seen that in Cases V, VI, and VII there were distinct, although slight, evidences of ganglionic changes, indicated by degenerations in the posterior root.

Pathological observations published since the appearance of their work are even more convincing. Ballet,⁹ in a case of herpes zoster occipitocervicalis of three weeks' duration, reports as follows: "The ganglia were not examined; marked degenerations are present in the third cervical posterior root. In the second cervical root it is with difficulty that degenerations are found, although they are distinctly present."

Armand-Delille and Camus,¹⁰ in a case of herpes occipitocollaris of one month's duration, found marked chromatolysis in the cells of the left third cervical ganglion. Similar cellular degenerations were also present in the left second cervical ganglia, but less marked. There were corresponding degenerations in the second and third posterior roots. (This case is of especial interest pathologically, in that the ganglionic lesion was a cellular degeneration without the massive inflammatory reactions usually found.)

In a personal observation of herpes occipitocollaris with facial palsy—autopsy eighty-seven days after the appearance of the eruption—there were found well-marked degenerative changes not only in the third cervical posterior root, but also in the posterior root of the geniculate ganglion, the *pars intermedia* of Wrisberg.¹¹

A most interesting observation is that of Hedinger.¹² A herpes zoster in the eleventh dorsal distribution of the left side; death occurred nineteen days after the appearance of the eruption. There was typical hemorrhagic inflammation in the left eleventh dorsal ganglion; in the left tenth and left twelfth dorsal ganglia similar changes are present, but much less severe. All the lumbar ganglia on the left side showed lymphocytic infiltration, diminishing in intensity downward, but especially well marked in the last lumbar. In the right lumbar ganglia only slight and insignificant changes were present. Similar changes were present and somewhat more marked in the right eleventh and twelfth dorsal ganglia.

In this case there seems to be no doubt as to the effect of the

⁹ Lesions nerveuses dans un cas de zona cervicale, Soc. des hôp., 1900, p. 706.

¹⁰ Soc. de neur. de Paris, February, 1903.

¹¹ Jour. of Nerv. and Ment. Dis., February, 1907.

¹² Deut. Zeit. f. Nervenheil., Band xxiv, S. 305.

specific toxin of herpes zoster on a series of ganglia, although the eruptive manifestations were limited to the eleventh dorsal zoster zone. It will thus be apparent, from the more recent pathological studies of herpes zoster, that the inflammatory reaction in the ganglia is not so limited in its extent as a study of the eruption phenomena alone might lead us to infer, in this respect confirming the clinical observation of mild sensory disturbances—neuralgic pains, hypalgesia, hypesthesia—in zones free from the eruption, and also explaining the occurrence of facial palsy in herpes zoster of the face and neck, in which the geniculate zone is free from eruption.

IV. THE CRANIAL NERVE GANGLIA AND THEIR RELATION TO HERPES ZOSTER. I will now mention, briefly, certain facts relating to the anatomy and histology of the cranial nerve ganglia, and the probable role which these structures play in herpes zoster.

At the time of the presentation of my paper, June, 1906, the only recognized cranial nerve localization of zoster was that of the Gasserian ganglion, an inflammation of this ganglion giving rise to the well-known herpes facialis, concerning which, an abundant literature has grown up. Barnes,¹³ however, in 1903, in the report of a case of herpes of the neck associated with facial palsy, made the following statement: "Hence this association of symptoms strongly suggests that the patient is suffering from the effects of an inflammation of three ganglia—one motor (the geniculate of the facial nerve) and two sensory (the third and fourth right cervical root ganglia)."

Vail¹⁴ also, in his paper on herpes zoster auris, mentions the otic ganglion as a possible localization, in the following words: "Politzer and others have observed facial paralysis on the affected side. This would seem to indicate involvement of the otic ganglion, which I believe has a branch of the facial entering it."

The otic ganglion, or ganglion of Arnold, is not regarded by histologists as belonging to the spinal type. It is grouped with the sympathetic system of ganglia, in which the cell type is multipolar, and not unipolar. Therefore, according to the present pathological conceptions of the disease, it should be excluded from the realm of herpes zoster.

As regards the geniculate ganglion, it must be emphasized that this structure is not *motor*; histologically it belongs to the sensory ganglia, composed of the *unipolar* type of cells, and is, therefore, the homologue of the spinal and Gasserian ganglia. With its afferent fibers, the pars intermedia of Wrisberg, and its efferent fibers, the petrosal nerves, it forms the sensory system of the facial nerve. In other words, we have here a ganglionic structure manifesting the same susceptibility to zona as the Gasserian and spinal ganglia.

¹³ Trans. Clin. Soc., 1903, vol. xxxvi.

¹⁴ Laryngology and Rhinology, 1906.

If the Gasserian and geniculate ganglia, by virtue of the unipolar cell type, belong to that series of ganglia liable to the inflammation of zona, the same would also be true of the ganglia of the glossopharyngeal nerve (ganglion of Andersch and ganglion of Ehrenritter) and of the vagus nerve (ganglion jugulare and ganglion plexiforme). These ganglia are also regarded by histologists as belonging to the *unipolar* or *spinal type*, and therefore cannot be excluded from the realm of herpes zoster.

I would also lay especial emphasis upon the possible role played by the ganglia of the acoustic nerve in this affection. All the posterior spinal ganglia, as well as the ganglia of the fifth, seventh (geniculate), ninth, and tenth cranial nerves, are developed from outgrowths of the embryonic structure known as the neural ridge. These ganglia are all of the so-called unipolar or spinal type. The ganglion of the acoustic nerve (ganglion acousticum) is also an outgrowth of the neural ridge, later becoming differentiated into the ganglion spirale of the cochlear nerve and the ganglion of Scarpa of the vestibular nerve. The cells of the acoustic ganglia are not, however, unipolar, but retain the primitive bipolar character (in fishes the cells of the spinal ganglia are bipolar, and it is only in higher types that the unipolar cells are found).

While the ganglia of the auditory nerve retain their bipolar character, their original source is the neural ridge, and it would appear very probable that they, as well as the other ganglia taking their origin from this structure, may be liable to the specific action of the toxin of zoster. This theory is confirmed clinically by the occurrence of very severe auditory nerve symptoms in zona of the face, ear, and neck. From the developmental and histological standpoint, I believe that we are justified in receiving the ganglia of the glossopharyngeal, vagus, and acoustic nerves into the realm of herpes zoster.

V. THE ZOSTER ZONE FOR THE GENICULATE GANGLION (HERPES OTICUS). In order to determine the exact extent and boundary of this zoster zone, accurate clinical observations in the future will be necessary—observations in which the whole extent of the eruption has been carefully noted and indicated on the various anatomical landmarks of the ear. There are, however, at the present time a sufficient number of accurately recorded cases of herpes oticus, by means of which a fair idea of the extent and probable boundaries of this zone may be determined, and which may serve as a basis for future investigations. The difficulties encountered in the interpretation of this area are considerably increased by the very complex innervation of the parts and the tendency to variation and overlapping of the zoster zones on the extremities of the body. It will also be recalled that the zones of the Gasserian and cervical ganglia are partially represented on the auricle, the herpes occipitocollaris encroaching on the auricle posteriorly, and the herpes facialis anter-

riorly. The zone for the geniculate is intercalated between these two areas. For the purpose of determining this zone, I have analyzed and tabulated seventeen cases of primary isolated herpes zoster oticus. It is, however, only the area in which the eruption is distributed to which I will call attention. This distribution corresponds in extent to the following anatomical landmarks: The tympanic membrane, the external auditory canal and meatus, the concha, tragus, antitragus, lobe of the ear (external surface), anthelix, and the fossa of the anthelix.

We may, therefore, assume that the geniculate ganglion is represented in an irregularly cone-shaped area, the apex corresponding to the tympanic membrane, the base situated on the external surface of the auricle. I do not believe that this entire area is represented by the geniculate ganglion alone. Allowance must be made for the overlapping of the Gasserian in front, and the cervical nerves behind. It is in my opinion, also, not improbable that future investigations will show that the ganglia of the glossopharyngeal and vagus nerves are also partially represented in this area.

CASES OF HERPES OTICUS.	THE DISTRIBUTION OF THE ERUPTION ON THE ANATOMICAL LANDMARKS OF THE EAR BY X.											
	Auricle.	Anthelix.	Fossa of helix.	Lobe.	Tragus.	Antitragus.	Concha.	External meatus.	External auditory canal.	Anterior wall.	Posterior wall.	Tympanum.
1. Berger, Neur. Central, 1905	X	X			
2. Chavanne (Bilateral case). Right	X	X	X					
3. Ann. Mal. de L'Oreille, 1906. Left	X	..	X				
4. Gruber, Case 1	X	..	X
5. Monatsschr. f. Ohrenheil., 1875. Case 2	X	X	
6. Gruber, Zeitsch. f. Ohrenheil., vol. xlv	X	X	X				
7. Hasslauer, Deut. Mil. Aerzt. Zeit., 1905	X	X	X			
8. Hammerschlag, Arch. f. Ohrenheil., 1901	X	X	X							
9. Hammerschlag, Arch. f. Ohrenheil., 1898	X	X	X							
10. Sommers, Amer. Med. and Surg. Bull., 1896	X	X			
11. Seral, Zeit. f. Ohrenheil., vol. xlv	X					
12. Szenes, Inter. Central. f. Ohrenheil., 1902	X
13. Tomka, Arch. f. Ohrenheil., 1900	X											
14. J. R. Hunt, Jour. Nerv. and Ment. Dis., 1907	X	X				
15. Personal Observation	X	X	X
16. Personal Observation	X	X	X	X	X
17. Personal Observation	X	..	X	X				

VI. SENSORY SYMPTOMS IN THE TRIGEMINAL AND CERVICAL ZONES IN HERPES ZOSTER OTICUS. In some cases of herpes zoster oticus, symptoms referable to the trigeminal distribution have been noted.

Tearing and shooting pains in one or more of the three divisions of the fifth nerve are mentioned (Hasslauer,¹⁵ Anstie,¹⁶ Hammerschlag¹⁷). The points of exit of the trigeminus were found tender on pressure (Berger¹⁸ and Hammerschlag). Diminution or absence of the corneal and conjunctival reflexes (Berger, Hammerschlag, Ramsay Hunt¹⁹), hypesthesia and hypalgesia of the face on the affected side have been observed. These fifth nerve symptoms are sensory in nature, and unaccompanied by a corresponding eruption of herpes. I find an explanation for their presence in a mild inflammatory reaction of the Gasserian ganglion, but not of sufficient severity to produce the eruption. It must be admitted, however, that the neural connection between the geniculate ganglion and the second and third divisions of the fifth nerve (great and small superficial petrosal nerves) may give rise to a referred pain.

Mild sensory symptoms of a similar nature may, likewise, be present in the distribution of the cervical nerves in cases of herpes oticus. In one of my cases a typical example of herpetic inflammation of the geniculate ganglion, with herpes zoster oticus, facial palsy, and deafness, severe lancinations were present in the occipital region and neck for several days. In addition there were distinct hypalgesia and hypesthesia on the left side of the face, head, and neck for two weeks. This sensory disturbance was not present below the level of the neck, and there were no stigmata of hysteria.

VII. THE GANGLIA OF THE GLOSSOPHARYNGEAL AND VAGUS NERVES AND THEIR PROBABLE ROLE IN ZOSTER. These ganglia, composed of unipolar cells, taking their origin from the neural ridge, are the homologues of the posterior spinal ganglia (Fig. 3). Theoretically there is no reason why they should not be involved in cases of posterior poliomyelitis. From our knowledge gained by the dissection method alone, the ganglia of the vagus presumably have a skin representation on the posterior wall of the auditory canal, the posterior portion of the tympanic membrane, concha, and posteromesial surface of the auricle (*auricular branch of the vagus*). The glossopharyngeal ganglia through their communications with the vagus and facial nerves may also be represented in the same area. In an analysis of many cases of zona of the cephalic extremity, I have had constantly in mind the possibility of symptoms which might be ascribed to a disturbance of the vagus function. The following case, reported by Buys²⁰ is the only one coming under my notice, which might bear this interpretation:

"A girl, aged seventeen years. Onset with headache and vomiting. Severe headaches continued with *stiffness of the neck*, and *frequent*

¹⁵ Deut. Mil.-Aerzt. Zeit., 1905, vol. xxxv.

¹⁷ Arch. f. Ohrenheil., 1901, s. 16; 1898, s. 1.

¹⁹ Jour. of Nerv. and Ment. Dis., 1907.

²⁰ Bull. de la Soc. Belge d'otol., de laryng. et de rhin., 1898. Eruption herpetique du pavillon precedé de phenomenes nerveux considerables.

¹⁶ The Practitioner, 1871, p. 198.

¹⁸ Neur. Centralblatt, 1905, s. 844.

vomiting. Photophobia, no fever or delirium. *The pulse was slow and irregular*. On the fourth day, the acuteness of the pain subsided and settled in the mastoid region of the right side. The mastoid was tender, as was the canal on the introduction of the otoscope. Hearing was diminished. On the fifth day herpetic vesicles made their appearance on the antitragus and lobule of the ear. The headache and neck pains are less severe. On the sixth day a fresh crop of vesicles made their appearance on the mesial surface of the pinna and the lobule. All pain disappeared in the course of a few days, and the hearing was restored. In fifteen days, the eruption had vanished."

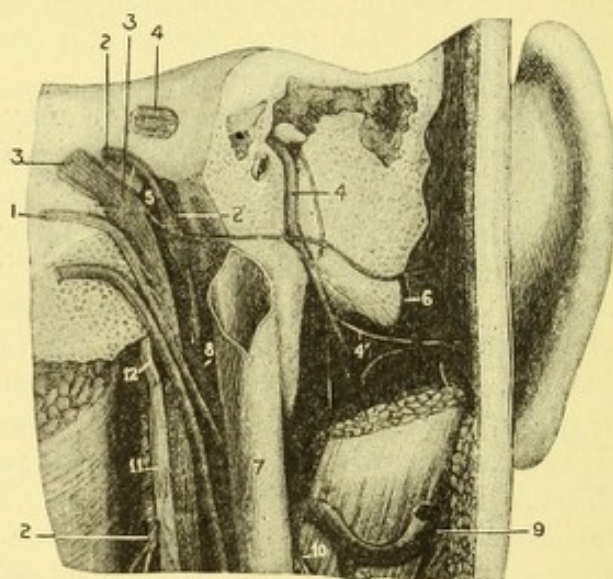


FIG. 3.—The ganglia of the ninth and tenth nerves, and the auricular branch of the vagus: 1, spinal accessory nerve; 2, glossopharyngeal; 2', ganglion of Andersch; 3, vagus nerve; 3', ganglion jugulare; 4, facial; 4', auricular branch of facial; 5, auricular branch of vagus; 6, anastomosis between the auricular branches of the facial and vagus nerves. (Testut's Anatomy.)

The frequent vomiting and the slow irregular pulse in this case are strongly suggestive of a vagus disturbance. I would also emphasize the area of distribution of the eruption corresponding to the cutaneous filaments of the vagus. It will also be observed, in this case, that the hearing was affected, without any associated evidences of facial palsy, also suggesting a primary involvement of the acoustic ganglia.²⁰

VIII. CONCLUDING REMARKS. The fusion of these clinical types into a single large group, with a common pathological basis, constitutes a well-defined clinical entity. It may be summarized as follows: A peripheral facial palsy complicating an eruption of herpes zoster on the cephalic extremity of the body. The eruption may be limited to the face, the head and neck, or the ear, or combinations of these (*herpes facialis*, *herpes occipitocollaris*, *herpes oticus*). The term *herpes oticus* I would confine to that group of cases in which the

eruption is restricted to the cone-shaped zoster zone of the geniculate ganglion (the tympanum, auditory canal, concha, and an adjacent marginal area on the external surface of the auricle). With the facial palsy there may be associated symptoms referable to the auditory nerve. These are of two types—hypo-acousis merely, or a severe complex of symptoms similar to Ménière's syndrome, consisting of tinnitus aurium, deafness, vertigo, vomiting, nystagmus, and disturbances of equilibrium.

The underlying pathology is the posterior poliomyelitis, a specific inflammation of ganglia of the spinal type. The characteristic features are given to this chapter of zona by the proximity of the geniculate ganglion to the facial and the terminal divisions of the auditory nerve, the inflammation in the geniculate ganglion extending to the adjacent nerve trunks by contiguity of structure. The pressure effects would be still further increased by the enclosure of these structures in a common sheath, situated in a narrow osseous canal.

I also believe that the ganglia of the acoustic nerve may be primarily involved, and I have given embryonal, histological, and clinical grounds for advancing this theory. I have also suggested the possibility of the ganglia of the glossopharyngeal and vagus nerves playing a role in the symptomatology of the syndrome, and emphasized the great importance, clinically, of vagus symptoms in this relation.

A particular emphasis has also been given to the occurrence of mild inflammatory reactions in ganglia above or below the chief focus, or that giving rise to the eruption of herpes zoster (*eruptive focus*). The symptoms present in the non-eruptive zones are mild and transitory in nature, and consist of pains and slight objective sensory disturbances. I regard the Gasserian, geniculate, acoustic, glossopharyngeal, vagus, second, third, and fourth cervical ganglia as representing a continuous ganglionic series or chain, all having the same embryonal origin (*neural ridge*), and all, with the exception of the acoustic ganglia, having the same cell type (unipolar), and therefore belonging to the realm of posterior poliomyelitis. This serial chain of ganglia is concerned in the production of the syndrome. A hemorrhagic inflammation in one of these ganglia is followed by the usual manifestation of herpes zoster, an eruption appearing in its respective zone.

Depending upon the severity of the infection, other ganglia of the group may be the seat of an inflammatory reaction as well, but not sufficient in degree to produce an eruption. This milder involvement is manifested clinically by pains and slight objective sensory disturbances, and in the case of the geniculate and acoustic ganglia, by their respective neural symptoms.

