the apex of right lung and bronchial glands; pulmonary edema; chronic and acute passive hyperemia of the liver and spleen; acute exudative and degenerative nephritis.

This case is of interest because of the unusual location of the lesion and because of the organism causing it. The diagnosis of lesions of the right heart are always difficult, but are dwelt on sufficiently in the various text-books, and will not be made the subject of remark here. Cultures from the vegetations were made on blood-agar, and the organism found to be the Micrococcus endocarditidis rugatus. This organism was described by Weichselbaum in 1889. Inoculation experiments made on dogs cause an endocarditis, while in rabbits and guinea-pigs local lesions only occur.

The writer had the opportunity of working with this organism while assistant to Professor Leary, of Tufts College Medical School. The organism was obtained from a fatal case of acute endocarditis, and various cultural tests and animal inoculations were made, though little was added to the knowledge of the organism as supplied by Weichselbaum. It was found to undergo autolysis rather easily, and resembled in this respect the Gram-negative group of cocci of which the meningococcus and gonococcus are members. The organism grows slowly on blood-agar, adhering to the medium in spreading, raised, tenacious colonies, of a dirty yellowish-white color with darker centres.

Smears stained by Gram’s method, from the lesions, showed most of the cocci to be Gram-positive. In smears from culture the young cocci retain the stain while many of the older organisms do not, and there are numerous large involution forms produced. Old cultures kept below 37.5° C. are especially rich in large Gram-positive involution forms.

HERPES ZOSTER OF THE CEPHALIC EXTREMITY, WITH A SPECIAL REFERENCE TO THE GENICULATE, AUDITORY, GLOSSOPHARYNGEAL, AND VAGAL SYNDROMES.

By Norman Sharpe, M.D., New York.

INTRODUCTION. That herpetic inflammations attack the sensory ganglia of the cephalic extremity other than the ganglion of Gasser, and constitute distinct clinical entities, has been known for the past several years; yet, judging from the reports that have appeared in recent literature of herpes zoster of the face, ears, or neck, with or without motor-nerve involvement, this fact has not been generally recognized.
Herpes zoster, as an independent disease, has long been known. It was the zona of the Greeks and the cingula of the Romans. The etiology is still in doubt, but the consensus of opinion is that it is due to a specific agent. In 1863 von Büren sprung\(^1\) first showed the lesion to be in the sensory ganglia on the posterior spinal roots; but more recently Head and Campbell developed the pathology of herpes zoster and placed it on a firm basis. Their observations were, however, confined to the ganglia of the spinal roots and the Gasserian ganglion. Still more recently Ramsay Hunt, entering a hitherto unexplored field, in a series of convincing articles, has shown that the sensory ganglion of the facial nerve (the geniculate ganglion), the peripheral ganglia of the acoustic, of the glossopharyngeal, and the vagus nerves, may also be the seat of herpetic inflammation, and has outlined a definite syndrome and zoster zones for each ganglionic distribution. These ganglia have been shown to be of the same anatomical construction and embryological derivation as the spinal ganglia and the ganglion of Gasser.

Beyond a few remarks on the etiology and pathology of herpes zoster in general, this paper will be confined to a consideration of the herpetic inflammation of the sensory ganglia of the cephalic extremity, omitting, however, the Gasserian ganglion, which has already been well and abundantly studied.

As stated above, nothing is positively known concerning the etiology of zona, but as Head and Campbell pointed out, and as others have emphasized since, the inflammatory lesion in the sensory ganglia is similar to that attacking the anterior horn cells in acute anterior poliomyelitis. It is most likely an acute infectious condition caused by a specific agent, as only those nerve structures are attacked which contain unipolar cells, of which the spinal ganglia and the Gasserian ganglion are types. Hunt's\(^2\) work in demonstrating that the peripheral ganglia of the seventh, eighth, ninth and tenth nerves, which contain unipolar cells and, like the spinal ganglia, are developed from the neural ridge and subject to the inflammation of herpes zoster, has strengthened the belief that zona is an acute infectious condition caused by a specific agent.

On account of the similarity of this condition to that of anterior poliomyelitis, Head and Campbell gave to herpes zoster the name of posterior poliomyelitis, a rather misleading term, and one which should be restricted to designating inflammatory conditions of the posterior horn.

PATHOLOGY. The pathology consists of an acute inflammation of the sensory ganglion on the posterior root, in whose skin zones

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\(^1\) Beiträge zur Kenntnisse des Herpes Zoster, Charité Annalen, 1863.

the vesicles are found, as Head and Campbell\(^3\) have shown in their twenty-one autopsies, the herpetic eruption having appeared from three days to seven hundred and ninety days previous to death. In those cases in which the eruption appeared but a few day before death the affected ganglion was found swollen and edematous, the vessels intensely engorged, and small foci of hemorrhage scattered throughout the ganglion. This was accompanied by degeneration and destruction of some of the nerve cells. They also found degeneration of fibers in the posterior root central to the ganglion, and in some cases degeneration of afferent fibers in the peripheral nerve leading to the affected ganglion. In those cases in which weeks or months had elapsed between the herpetic inflammation and death there was found replacement of the degenerated cells in the ganglion by scar tissue.

Though these observers stated that the inflammatory changes were confined to the ganglion (including afferent and efferent nerve fibers) in whose skin zone the eruption was found, yet their own data show, and as Hunt has demonstrated in a case of his own, the adjacent ganglia, both above and below, show evidences of inflammation similar to that of the affected ganglion, though in a lesser degree. Therefore, although the specific inflammation attacks chiefly one sensory ganglion, with eruption in its skin zone, the adjacent ganglia do not escape involvement, though the inflammation is rarely severe enough to cause herpes in their respective skin zones. This is important to remember, especially when dealing with herpes zoster of the cephalic extremity, where the different ganglia are closely associated, as it explains those cases in which the eruption is in the zone of one ganglion, and there are complications which can be attributed only to the involvement of other ganglia.

Zona usually attacks the ganglia of one side only, and the eruption and neuralgic pain are strictly confined to the skin zone governed by the affected ganglion.

Owing to the specific nature of the affection a second attack of herpes zoster is rare, and in fact is less common than a second attack of measles. Head and Campbell found but four such cases in over four hundred. In some cases reported recently this fact seems to have been overlooked, as the reports spoke of previous attacks, and in other respects did not correspond to true herpes zoster. As Dabney\(^4\) says, symptomatic herpes, accompanying or following caries, arsenical or other chemical poisoning, and seen in certain brain diseases, must be differentiated from true herpes zoster.

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\(^3\) The pathology of Herpes Zoster and its Bearing on Sensory Localization. Brain, 1900, xxiii, 353.

Formerly herpetic eruptions in the auricle were ascribed to inflammation of the Gasserican ganglion or of the ganglia of the second and third cervical ganglia; but Cushing's\(^4\) studies of the anesthesia following extirpation of the Gasserican ganglion show that the fibers of the fifth nerve do not innervate the area in which the eruption of herpes zoster oticus is found. The sensory innervation of the ear by the fifth nerve includes the tragus, the anterior wall of the canal, and the anterior half of the tympanum. The sensory representation of the second and third cervical nerves upon the auricle is the area posterior to a line drawn from the middle of the pinna down along the edge of the helix, under the antitragus, and forward, including the larger part of the lobe. Thus there is a part of the ear, composed of the posterior half of the tympanum, the posterior wall of the canal, the concha, the antitragus, and the fossa of the antihelix which is innervated by neither the fifth nerve nor the second and third cervical. This area is innervated by fibers from the seventh, the ninth, and tenth nerves, and it is in this area that the eruption in herpes zoster oticus is found.

Hunt,\(^6\) in his work on the pathology of herpes zoster oticus, with or without facial palsy and acoustic symptoms, and in his later work on localizing the lesions of the peripheral ganglia of the eight, ninth, and tenth nerves, and mapping out their skin zones in the external ear and in the buccal cavity, has rendered comprehensible the various symptoms presented by herpes zoster in these localities.

**Symptomatology.** Following Hunt's\(^7\) classification we group herpes zoster of the cephalic extremity as follows:

1. Herpetic inflammation of the geniculate ganglion (herpes zoster oticus).
2. Herpetic inflammation of the geniculate ganglion with facial palsy and acoustic symptoms.
3. Herpetic inflammation of the auditory ganglia with acoustic symptoms.
4. Herpetic inflammation of the glosso-pharyngeal and vagal ganglia.
5. Herpes zoster facialis or occipitocollaris with facial palsy and auditory symptoms, alone or in combination.

1. *Herpetic Inflammation of the Geniculate Ganglion.* It has long been known that the facial nerve is a mixed nerve, having a ganglion, the geniculate, a sensory root, the pars intermedia of Wrisberg, and afferent sensory fibers in the superficial petrosal

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\(^6\) A Further Contribution to the Herpetic Inflammations of the Geniculate Ganglion, AMER. JOUR. MED. SCI., August, 1908.

\(^7\) The Symptom Complex of the Acute Posterior Poliomyelitis of the Geniculate, Auditory Glosso-pharyngeal and Pneumogastric Ganglia, Archiv Int. Med., 1910, v, 631. (Full bibliography to date.)
nerves and the chorda tympani. The geniculate ganglion is situated on the seventh nerve just at the opening of the Fallopian aqueduct (Fig. 1). Anatomically it corresponds to the sensory ganglia of the spinal roots, like them being developed from the neural ridge and being composed of unipolar cells. In this situation in the aqueduct the ganglion, the facial, and the auditory nerves are closely associated, being contained in a common sheath. When the specific inflammation attacks the geniculate there is preherpetic pain, otalgia, more or less severe, and following this, usually two to

Fig. 1.

eight days, the characteristic vesicular eruption in the zoster zone of the ganglion. This zone, which is somewhat cone-shaped includes the posterior half of the tympanic membrane, exterior auditory canal, the concha, the antihelix and its fossa, the antitragus, and a portion of the lobule. If the inflammation in the geniculate extends to the facial and auditory nerves, as it is prone to do because of their close association in the internal canal, there will appear facial palsy and auditory symptoms. Dejerine,\(^8\) who

has accepted Hunt’s geniculate syndrome, reports a case of herpes zoster oticus accompanied by facial impairment. In his case the eruption, which was more widespread than that shown in Fig. 2, occupied a large part of the geniculate zone, being placed on the concha, the fossa of the antihelix, and the lobe of the ear.

2. Herpetic Inflammation of the Geniculate with Facial Palsy and Acoustic Symptoms. In the course of zona of the geniculate ganglion, if the seventh nerve is involved, facial palsy supervenes. The paralysis, which is always complete, involving all three branches of the facial nerve, may come on at the time the eruption appears or not until from five to twelve days later, showing that the disease had not reached its height with the appearance of the eruption. Hennebert\textsuperscript{9} has reported a case in which the eruption on the concha and on the mastoid was followed in five days by facial palsy. The auditory symptoms, which are due either to an extension of the inflammation from the geniculate to the eighth nerve in the auditory canal, or to a simultaneous inflammation of the auditory ganglia, vary from hypacusis with tinnitus to severe expressions of Ménière’s syndrome (nystagmus, vertigo, nausea, and vomiting).

Muck\textsuperscript{10} reports a case of herpes zoster of the geniculate (herpes

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\textsuperscript{9} Zona Otique, Archiv internat. de laryngol d’otol. et de rhinol., 1913, xxxv, 365.

\textsuperscript{10} Neuritis des Trigeminus, des Fazialis und des Akustikus als symptomenkomplex eines Herpes Zoster Oticus, Ztschr. f. Ohrenh., Weisb., 1912, lixiv, 217.
zoster oticus) in which, following the eruption on the auricle, there
was involvement of the fifth, seventh, and eighth nerves, as shown
by the presence of hypesthesia, facial impairment, and disturbances
of hearing.

Dombrowski,\textsuperscript{11} who discusses at length the various symptom-
complexes of herpetic inflammations of the cephalic extremity,
also states that a similar combination may occur.

In a recent article\textsuperscript{12} Leonard Kidd, although he does not reject
in toto the Hunt Syndrome, denies that the seventh nerve has a
sensory cutaneous zone, and says that herpes zoster oticus is not
due to an inflammation of the geniculate ganglion.

He states: "Now, if we recall the fact that the geniculate ganglion
of man is a swelling on the course of the facial nerve, we must
conclude at once that there could be no such thing as a true isolated
geniculate herpes, without, at any rate, marked paresis of the
facial nerve. It is certain, then, that all those cases of herpes auris,
which are unaccompanied by facial palsy or paresis are of non-
geniculate origin."

If this statement were to be accepted, we could not have a
limited involvement of the Gasserian ganglion—as in those cases,
not uncommon, in which the zoster eruption is strictly limited
to the first, second or third division of the fifth nerve; also, according
to this hypothesis, in cases of herpes frontalis, which is a very
common and strictly limited Gasserian lesion, we should expect
evidences of sensory involvement in the other distributions of this
ganglion. This is, of course, by no means the case, and while it may
occur, it by no means necessarily follows. Also, according to this
theory of Dr. Kidd's, the motor roots of the spinal nerves, and the
motor branch of the fifth nerve, should show, to say the least,
frequent involvement; but on the contrary, the clinical facts
indicate this to be extremely rare. Therefore the clinical evidence
is all in favor of the possible occurrence of a limited selective inflam-

In reviewing the evidence furnished by comparative anatomy,
Kidd recognizes the existence of a cutaneous facial component
in cyclostomes, but says that this has yet to be demonstrated in
the higher mammals. This may be true, yet the fact that it has
existed in types as high as the cyclostomes, is strong presumptive
evidence of its vestigial persistence in man. And a vestigial cutane-
ous representation is all that Hunt has claimed for this ganglionic
distribution. Kidd admits that the facial nerve may carry fibers

\textsuperscript{11} Contribution à l'Étude de la Paralysie Faciale Zostérienne. Syndrome de l'In-
flammation Herpétique des Ganglion Geniculé, Thèse de Paris, Jouve and Cie., Paris,
1912.

\textsuperscript{12} The Alleged Sensory Cutaneous Zone of the Facial Nerve of Man, Rev. of
Ner. and Psych., Edinburgh, September, 1914, 393.
from the tympanic membrane and the mastoid cells. Why, however, he should admit this much of the geniculate distribution, and deny its cutaneous vestigial remnant, is rather difficult to understand. Kidd also states "If there were any facial cutaneous fibers in man, we should find in every case of Bell's palsy as complete a cutaneous anesthesia of its alleged auricular zone as any clinician ever found on the trigemino-cutaneous area, after a complete Gasserectomy."

In this statement, Kidd shows his complete unfamiliarity with the geniculate zoster zone, its outline, size and relations with its neighboring zones. He ignores the fact that the ninth and tenth nerve have representation in part of this area. He also overlooks the fact that section or inflammation of one posterior spinal root, will not cause anesthesia in its cutaneous zone. This applies with a special force to this part of the auricle, where the sensory supply of the seventh, ninth and tenth nerves is vestigial in character, and the different zones are small, and to a certain extent, overlap. Kidd does not appear to be familiar with, as he does not quote from it, Hunt's comprehensive study of the zoster zones of the seventh, ninth, and tenth nerves. 13

So that this rather spirited paper of Kidd's, who has approached his subject entirely from the critical and controversial side, contains no personal contribution except a discussion of the evidence. And even then does not present all the evidence, but omits entirely a citation of the most important contribution to the subject which he is discussing.

3. Herpetic Inflammation of the Auditory Ganglia. Herpes zoster attacking the eighth nerve ganglia (ganglion of Corti on the cochlear branch, and the ganglion of Scarpa on the vestibular branch) causes symptoms varying in severity from hypacusis with tinnitus to Ménière's syndrome (vertigo, nausea and vomiting, and prostration). That these symptoms are due to an inflammation of the auditory ganglia and not to an extension of inflammation from the geniculate to the auditory nerve can not be doubted. As proof of this, there are the cases in which the above symptoms occur, without an involvement of the seventh nerve, which, if involved, would be evidenced by facial impairment or paralysis. Considering the anatomical arrangement in the internal auditory canal, where the facial and auditory nerves are closely associated, being contained in a common sheath, and the geniculate ganglion seated upon the facial nerve, it is not conceivable that the facial would escape were the above symptoms due to zona of the geniculate ganglion.

4. Herpetic Inflammation of the Glossopharyngeal and Vagal Ganglia. The zoster zones of the peripheral ganglia of the glossopharyngeal nerve (ganglion petrosum and ganglion of Ehrenritter) and of the peripheral ganglia of the vagus nerve (ganglion jugulare and ganglion plexiforme) are represented upon the auricle and also

intra-orally. On the ear their zones are placed upon the posterior half of the tympanum, posterior wall of canal, and posterosmesial surface of the auricle and adjacent mastoid region. Thus the ninth and tenth nerves occupy part of the geniculate zone. As the auricular branches of the ninth and tenth nerves unite it is impossible to separate their zoster zones. Of course, where several nerves have representation in such a small area, some allowance must be made for variation and overlapping. Intra-orally it is possible to separate the zones of the ninth and tenth nerves. The zoster zone of the glossopharyngeal is placed on the posterior surface of the tongue, pillars of the fauces, and the tonsil. Zona in this region (herpes zoster pharyngis) is difficult to diagnose, for usually there are but few vesicles, and they disappear rapidly. The eruption is strictly unilateral, may be accompanied by paralysis of the soft palate, and at times facial palsy due to simultaneous involvement of the geniculate. Zona in this locality must be distinguished from pseudo-herpes, which are bilateral, are found on the soft palate, uvula, and pharynx, and are due to blocking of gland follicles. The intra-oral zone of the vagal ganglia is placed more posteriorly, at the root of the tongue, at the entrance of the larynx, and the adjacent pharyngeal region. Herpes zoster of these ganglia, with eruption in this region, may be accompanied by brachycardia, singultus, nausea and vomiting due to involvement of fibers of the vagus by the inflamed ganglia.

5. Herpes Zoster Facialis or Occipitocollaris with Facial Palsy and Acoustic Symptoms (Alone or in Combination). In herpes facialis or occipitocollaris, facial palsy and auditory disturbances are sometimes seen. This is due to the fact that in cephalic zona the tendency is for invasion of more than one ganglion. In this type of zoster, although the chief focus of inflammation is in the Gasserian ganglion, with eruption in its skin zone (herpes zoster facialis), or in the ganglia of the second and third cervical nerves, with eruption on the neck or occiput (herpes zoster occipitocollaris), yet the geniculate or auditory ganglia are also involved, though in a less degree, facial palsy, or auditory symptoms, occurring without an eruption in the geniculate zone. Happel\textsuperscript{14} reports a case of herpes occipitocollaris with facial palsy, though the true connection between the inflammation in the cervical ganglia and the facial palsy was evidently not recognized. In his patient the eruption appeared twenty-four hours after the preherpetic pain, and twelve days later paralysis of the facial nerve occurred. A month later the palsy still persisted, though the eruption had long since disappeared.

Motor nerve paralysis in herpes zoster occurs in other parts of the body but is rather uncommon. This is because the spinal ganglia and the ganglion of Gasser possess fibrous capsules which separate

\textsuperscript{14} Report of a Case of Herpes Zoster Complicated by Bell's Palsy, Episcopal Hospital Reports, Philadelphia, 1913, i, 215.
them from the motor roots. But the ganglia of the seventh, eighth, ninth, and tenth nerves have no fibrous capsules, and are in close relationship with the nerve fibers; hence, slight inflammations of the ganglia tend to involve the nerve fibers, though not always.

If the examiner be alert the diagnosis of herpes zoster of the cephalic extremity with its various symptom-complexes is usually not a matter of great difficulty. But if the true condition is not recognized much harm may be done. The pain may at times be very severe and ear drums have been incised in the belief that the condition was one of middle-ear infection, or mastoid operations have been contemplated, even in the presence of an herpetic eruption. Occasionally there are but few vesicles present, and these situated on the tympanic membrane, and they are not found unless careful search is made.

Herpes zoster is not dangerous to life, though there is a belief among the laity that if the eruption extends around the body the patient will die. It is conceivable that if a bilateral involvement of the vagal-ganglia should occur it might end fatally. The darting pains that often follow an attack of herpes zoster may be quite acute and persistent and cause great annoyance (Hunt\(^{15}\)).

TREATMENT. In the treatment of herpes zoster many remedies are advised. Zinc phosphide and quinin internally and applications of soothing ointments are most in favor at present. When the nature of the condition is considered it is difficult to see how they can do any good, and they probably have none but a mental effect. Fortunately herpes zoster is a self-limiting disease. Opening the vesicles is futile and may give rise to infection. Hot applications, and at times cold, seem to give as much relief as any other form of treatment. In severe pain anodyunes may be necessary.

Report of Cases. The following case is of interest, belonging to the type most commonly seen, herpes zoster of the geniculate with facial palsy, and also demonstrating the error that is generally made, that of attributing the symptoms to middle-ear or mastoid disease.

CASE I.—L. S., female, aged sixteen years. Had never had previous attack of herpes zoster. Onset November 18, 1913, with severe pain in depths of right ear and in the auricle. At the height of the pain, which was intermittent, her physician suggested mastoid or middle-ear disease, and contemplated operation. Pain markedly lessened at end of two days, at which time paralysis of the right facial nerve occurred, complete in all three branches; with onset of paralysis vesicles were found in the auricle, as noted in Fig. 2. There was no discharge from the ear, no sore throat, no nausea or vomiting.

\(^{15}\) Otalgia Considered as an Affection of the Seventh Cranial Nerve, Archiv Otol., 1907, xxxvi. 371.
January 13, 1914. Came to the Vanderbilt Clinic because of the facial palsy. Examination of the right ear revealed several small pigmented scars. They were situated on the concha and also just within the canal, as noted in Fig. 2; none behind ear. She complained of sticking pains in the right ear and slight tinnitus in both ears; no hypacusis. Facial palsy was complete with R. D. Palate was normal. She said taste sense was lost on the right side of the tongue for one month following the onset of the trouble. At time of examination taste sense was normal. The corneal and conjunctival reflexes on right side were diminished. The pupils were equal and reacted to light and accommodations.

March 16. Complete paralysis of middle and lower branches of right facial. Upper branch had partially recovered. Had occasional faint pain in depths of right auditory canal.

April 27. Upper branch of facial almost entirely recovered. Middle and lower branches partially recovered. Electric reactions K C C > A C C. The occasional sticking pains had disappeared. Scars on concha still visible.

CASE II.—Herpes Oticus with Facial Palsy. Mrs. A. J., aged thirty-two years; widow. Personal and family history negative. Five weeks before present illness had a "cold," but had recovered. Felt well and was at work when present trouble began.

December 24, 1913, while at work, a paralysis of the right side of the face developed. Two days later had pain in canal of ear and behind ear, and a small ulcer appeared just behind antitragus. Had no tinnitus, no sore throat, no vomiting. Complains of a burning taste in the mouth which she refers to whole of tongue.
and roof of mouth. No constitutional symptoms, and the only discomfort was pain at entrance of canal and tenderness of auricle, which lasted about three days.

*Status.* Pupils equal and react; hearing acute and equal on both sides; arm-jerks, knee- and ankle-jerks normal; pulse regular. Right seventh nerve paralysis, complete (can, however, close eye). Palate symmetrical; taste lost in right fifth nerve distribution. Auricle reddened and slightly swollen; in the concha, just behind the antitragus is a healing ulcer. Sense of smell normal. Other cranial nerves normal. Tactile and pain sense equal on both side of face.

January 12, 1914. No pain, and the auricle was not swollen. For several days has had considerable itching behind right ear in post-auricular (vagus) distribution. Facial innervation is almost normal in all branches. Tongue still feels rough and queer on right side.

**Case III.—Herpes, Occipitocollaris with Facial Palsy.** R. M. L., male, aged sixty years. In the early part of February, 1914, he had pain in the neck and lower part of the face on left side. His neck felt as if there was a "kink" in it. Two days later an eruption of herpes appeared in the left side of the neck and over the angle of the jaw. The eruption was rather widespread, there being eighteen vesicles present. Three days after the appearance of the eruption, a left facial paralysis occurred. Hearing was slightly diminished on left side. No tinnitus. Had keen preherpetic pain, which lessened with appearance of facial palsy. Taste not impaired; no sore throat. Seen at Vanderbilt Clinic two weeks after onset of pain.

*Status.* General neurological examination negative. Complete left seventh nerve palsy; no nystagmus; corneal reflex present. No discharge from ear; no vesicles on or in the ear. Tactile and pain sense diminished over face and neck on left side.

**Summary.** The disease herpes zoster, in addition to its clinical, spinal, and Gasserian ganglia manifestations may involve other sensory ganglia of the cranial nerves. It may involve the ganglion of the facial nerve (the geniculate), the ganglia of the ninth nerve (ganglion petrosum and ganglion of Ehrenritter), and of the vagus nerve (ganglion jugulare and ganglion plexiforme). There are strong reasons for the supposition that the auditory ganglia (ganglion of Corti and ganglion of Scarpa) may be similarly involved. These ganglia may be involved singly or in combination.

The syndrome of the geniculate ganglion involvement is herpes zoster oticus, alone or with facial and auditory complications.

Herpetic inflammations of the ninth and tenth nerve ganglia occur, with herpes zoster oticus, herpes zoster pharyngis and laryngis, with pharyngeal and laryngeal palsies, occasionally with nausea and vomiting, brachycardia, hiccuphanging, and other symptoms of vagal irritation.
Herpetic inflammations of the eighth nerve ganglia is indicated by symptoms referable to the vestibular and cochlear nerves; deafness, tinnitus aurium, nystagmus, nausea and vomiting, disturbances of equilibrium, the fully developed picture resembling a severe type of Ménière's disease.

The neural symptoms may be very slight, often clearing up in a few days or weeks, or they may be quite severe, leaving permanent disturbances of function.

In conclusion, emphasis is laid upon the fact that the clinical picture is by no means always limited to involvement of a single ganglion, and that multiple involvement of these ganglia is not infrequent, thus producing a great variety of clinical combinations which are readily interpreted if the fundamental pathological conceptions are borne in mind. It is by the careful study and recording of these cases that we shall achieve a definite knowledge of the sensory distribution (zoster zones) of these small and obscure sensory ganglia.

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PILOUS CEREBRAL ADIPOSITY: A NEW SYNDROME.¹

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Many instances of genital dystrophy associated with obesity have been described since Froelich² first separated the syndrome from the general class of obesities, but we have not found any account of a class of cases complicated by an anomalous condition such as has been found in the following case. This condition is a marked increase of the body-hair, accompanied by a lack of the dry skin characteristic of the classical Froelich syndrome.

The case was in the service of Dr. Alexander Lambert in the medical wards of Bellevue Hospital, and it is through his kindness that I have been enabled to investigate it, and through his courtesy that I publish it.

CASE.—A white man, aged thirty-one years; watchman; father living, and is alcoholic. Mother died of nephritis following childbirth. One brother died in infancy of unknown cause. One sister living and well. Other family history negative. The patient has been married since 1906. His wife is of an enteroptotic constitution, and has asthma. She has had one miscarriage and four normally born children. Three of these have died in infancy, one at twenty

¹ Presented at the meeting of the New York Neurological Society on November 10, 1914.
² Fall von Tumor der Hypophysis Cerebri ohne Akromegalie, Wiener klin. Rundschau, 1881.