to gastric juice of normal acidity and pepsin strength, than do similar lesions in the absence of gastric juice. Gastric and duodenal ulcers are, therefore, locally infected areas of the wall of the digestive tract, irrespective of whether the ulcers themselves are produced by primary infection from the blood stream (Rosenow), by local circulatory disturbances, or by mechanical and chemical factors acting on the mucosa

Fig. 6 (Dog 32, Table 3).—Ulcer in the duodenum (twenty days) exposed to the action of gastric juice.

from the lumen of the intestine. It is well known that the tissues surrounding the ulcers are hyperemic. It is highly probable that in these hyperemic ulcer areas the nerves (sensory and motor) become hyperexcitable, as is the case in all other areas of infection. These hyperexcitable gastric nerves, subjected to stimulation by the normal gastric juice, may thus yield exaggerated responses (motor, inhibitory, and conscious). Carlson suggests that the favorable results from the alkaline and frequent feeding treat-

TABLE 3.—EXPERIMENTS TO DETERMINE THE TIME REQUIRED FOR THE HEALING OF ULCERS IN THE DUODENUM IN THE PRESENCE OF GASTRIC JUICE.

No. of Dog	No. Days Since Production of Ulcer	Stage in Healing of Ulcer
37	10	Active ulcer; no healing
1	13	Active ulcer 1.5 by 1 cm.; no healing
40	15	Active ulcer 1 by 1.5 cm.; no healing
32	20	Ulcer almost healed
33	20	Ulcer completely healed
34	26	Ulcer healed
2	31	Small depressed area in mucosa
22	45	Completely healed

ments in these cases may be due to the removal of this added stimulation of the hyperexcitable nerves rather than the prevention of any digestive or corrosive action of the pepsin hydrochloric acid on the exposed tissues. The degree of reflex disturbance from the acid stimulation of the nerves in the ulcer areas would vary with the individual, depending on the severity and extent of the infection, as well as on the individual stability of the entire nervous organization. We should expect it to be much greater in man than in the lower animals.

The bacteriologic examination of the ulcers is interesting in that it shows that lesions, produced experimentally by chemical means, may subsequently become infected. The bacteria are the same as those isolated from clinical ulcers in man. It seems probable that

TABLE 4.—EXPERIMENTS TO DETERMINE THE TIME REQUIRED FOR THE HEALING OF ULCERS IN THE DUODENUM IN THE ABSENCE OF GASTRIC JUICE

No. of Dog	No. Days Since Production of Ulcer	Stage of Healing of Ulcer
36	5	Perforation
35	7	Active ulcer; no healing
27	9	Active ulcer, 0.5 by 1 cm.; no healing
11	9	Active ulcer, 1.5 cm. dia.; no healing
25	10	Active ulcer, 1 cm. dia.; no healing
38	15	Active ulcer; edges raised; indurated
16	22	Ulcer 0.5 cm. dia.; edges indurated
8	24	Ulcer nearly healed; one small depressed area
39	27	Ulcer healed
20	32	Ulcer healed
14	39	Ulcer healed

the infection is direct from the alimentary tract, since it is not likely that these bacteria would be found in the circulating blood of otherwise healthy dogs.

CONCLUSIONS

1. The digestive activity of gastric juice is not the important factor in the delayed healing of acute ulcers of the stomach and duodenum and the consequent formation of chronic ulcers.

2. Ulcers produced by local injection of solutions of silver nitrate become subsequently infected with organisms probably from the alimentary tract.

THE IMPORTANCE OF AURAL SYMPTOMS IN THE EARLY DIAGNOSIS OF TUMOR OF THE CEREBELLOPONTILE ANGLE*

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The eighth nerve, comprising the auditory and the vestibular, from a diagnostic surgical standpoint, may be divided into three portions: the *peripheral*, comprising the end organs in the labyrinth; the *intradural*, extending from the fundus of the internal auditory meatus to its entrance in the groove between the pons and the medulla, and the *intracerebral* ramifications of its tracts in the brain to its cortical centers in the temporosphenoidal lobe and cerebellum.

The sudden and commanding character of the symptoms in inflammation of the cochlea and semicircular canal are due largely to the small, inelastic walls of the containing bony labyrinth, compelling an immediate paralysis of the function of the delicate nerve structures; their bony case not allowing the gradual development of symptoms from slow increasing pressure on the nervous structure, which frequently occurs in nervous tissue elsewhere. (The greater resistance of the auditory portion in the labyrinth in serous labyrinthitis with the preservation of hearing after the complete abolition of the vestibular function is, however, somewhat of this nature.)

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After the entrance of the eighth nerve into the pons, its proximity to other vital centers generally causes the aural symptoms to be immediately overshadowed. The diagnostic symptomatology of lesions involving the intracerebral portion of the auditory nerve is but rarely recognized, although in my experience such lesions are far from uncommon.¹

In the passage of the eighth nerve, through the small space from the fundus of the internal auditory meatus to the medulla, across the posterior border of the middle peduncle of the cerebellum, the nerve, while intradural, is still extracerebral, and, being in a free space, may escape for a long time a paralyzing pressure from a tumor of the cerebellopontile angle.

Many cerebellopontile angle tumors, especially dural endothelioma and neurinoma,² are of very slow growth, and disturbances of hearing and of the vestibular apparatus of the affected side are frequently the only symptoms of which the patient complains, the aural often preceding other symptoms for months, and sometimes for years.³ On the recognition of the intracranial origin of the aural symptoms may depend the success of surgical intervention, and the reputation of the aurist. I know of many instances in which, after weeks and months of unsuccessful inflammations and local treatments, the development of a papilledema has first called attention to the true cause of the deafness (Fig. 1).

In studying the aural symptoms of cerebellopontile angle tumors, it should be remembered that they are but a modification of the acute aural manifestations so frequent in fracture of the base of the skull, extending through the petrous portion of the temporal bone. The end-result of compression of the eighth nerve, whether from fracture or tumor, is the same, namely, loss of function, deafness and loss of vestibular reactivity. However, in one, the function of the vestibular apparatus of one side is suddenly upset; but, in tumor, the slow pressure on the nerve allows of a readjustment of the vestibular apparatus during the process of compression.

In fracture there are suddenly: (1) tinnitus and deafness; (2) loss of vestibular reactivity, associated with (3) vertigo; (4) spontaneous nystagmus away from the affected side, and (5) spontaneous pointing deviations of both homolateral and contralateral extremities toward the side of the lesion. The deafness and loss of vestibular reactivity are permanent, but the nystagmus, dizziness, and spontaneous pointing deviations rapidly disappear. The transient vestibular manifestations, the dizziness, nystagmus and spontaneous pointing deviations result from a total,

sudden loss of function of the vestibular apparatus of the affected side, and the early abolition of these symptoms is due to a readjustment of the vestibular system, as a whole, to meet the changed condition.

It is through the same readjustment in the vestibular apparatus (of the opposite as well as the affected side) which, in the acute cases, so early abolishes the spontaneous nystagmus, pointing deviations and dizziness, that the slowly growing cerebellopontile angle tumors may first manifest themselves.

We have at our command for the early diagnosis of intracranial disturbances of the eighth nerve: (1) functional examination of the auditory portion, and (2) functional examination of the vestibular portion.

So important is a complete and systematic routine functional examination of the eighth nerve, that all patients presenting themselves with disturbance of the hearing or disturbance of the equilibrium should, because of the time consumed and the part played by the personal equation, be delegated for examination to a specially trained attendant.

This routine examination by an attendant in my office consists, for the auditory portion, in ascertaining: (1) the hearing distance of the (a) watch, (b) whisper, and (c) voice; (2) the lower and upper tone limits and the measuring of their respective durations; (3) the Rinne-positive or negative; (4) reference of the Weber at both vertex and teeth, and (5) the measurement of the duration of the bone conduction.

The routine examination of the vestibular apparatus consists of ascertaining (1) the presence or absence of spontaneous nystagmus. If from the above I suspect labyrinthitis or intracranial involvement, the attendant further (2) examines the spontaneous pointing of both arms and legs, and (3) explains to the patient the caloric test: (a) its inconveniences, such as vertigo, possible nausea and vomiting; (b) the short duration, during induced nystagmus, of the pointing deviation and falling, and (c) its suc-

cessful application, largely depending on the patient resisting the natural tendency to close the eyes. (d) The attendant then proceeds to the repeated rehearsing with the patient of the pointing and falling positions.

When the patient thoroughly understands what is required of him (4) the cold caloric test is applied in my presence, and during it are noted: (a) the induced nystagmus; (b) the rapidity of its induction; (c) whether it is associated with vertigo or a sensation of dizziness; (d) the pointing deviation for both arms and legs during the induced nystagmus, and (e) the falling tendency.

If the patient has been thoroughly drilled by the attendant prior to the application of the caloric (cold) test, it is remarkable what a small amount of time is consumed in making the test, and how much can be ascertained in the few seconds of an induced nystagmus.

Fig. 1.—Large endothelioma springing from dura, causing great compression of cerebellum. Aural symptoms were present for years prior to general symptoms of tumor.

1. Starr: Jour. Nerv. and Ment. Dis., 1910, 37, 401. Guthrie: Jour. Laryngol., Rhinol. and Otol., 1915, 30, 177. Weinland: Arch. Psychiat., 26, 375. Campbell: Histological Studies in Cerebral Localization, London, 1905. Mills: Jour. Nerv. and Ment. Dis., February, 1912.
2. Healy: Jour. Nerv. and Ment. Dis., 1909, 26, 229. Henneberg and Koch: Arch. Psychiat., 36, No. 1.
3. Wimmer: Six Cases of Acoustic Nerve Tumors with Operation, Hospitalstidende, September, 1914, p. 1169. Schmiegelow: Ztschr. f. Ohrenh., July, 1915.

In cerebellopontile angle tumors growing from the dura, endothelioma, or attached to the acoustic nerve, neurinoma or fibroma molluscum, one of the first symptoms complained of is a slight deafness with a distressing tinnitus. Routine examination with the tuning forks may make an early diagnosis of an intracranial origin by (1) a reduction of the duration of the bone conduction of the affected side; (2) the referring of Weber to the unaffected side; (3) a positive Rinne on the affected side, and (4) frequently greater alteration of the tuning fork reactions than is accounted for by the amount of the loss of hearing.⁴

All these are too well known to be discussed. In the vestibular apparatus, however, many problems are still in the realm of speculation.

Spontaneous nystagmus is generally associated with tumor of the cerebellopontile angle when the tumor causes compression of the cerebellar cortex, but its slow growth frequently allows of considerable displacement of the cerebellum without nystagmus; in fact, the slowly progressive deafness has, as a rule, long passed into total deafness before the development of nystagmus, by which time a papilledema from increased intracranial pressure may have also appeared. Spontaneous nystagmus is apt to be a late manifestation of tumor originating in the cerebellopontile angle, in contradistinction to tumors growing into it from the cerebellum.⁵ Spontaneous nystagmus is also present in a certain proportion of tumors situated in the cerebrium,⁶ in which case, however, there will be no disturbance of the auditory apparatus, the hearing and the tuning fork reactions being normal.

Again, in intracerebellar growths, glioma, not involving the cerebellar cortex, spontaneous nystagmus may be entirely absent, although the growth may have attained considerable size; but in these also the tuning fork reactions are apt to be normal, deafness not usually being present.

Spontaneous Pointing Deviations.—Spontaneous deviations are, in my experience, very rare during all stages of cerebellopontile angle tumors or intracerebellar lesions of slow development, in contrast to their presence in the early stages of acute lesions, fractures and abscesses. Especially in those cases not associated with a spontaneous nystagmus are spontaneous deviations uncommon. In cases associated with nystagmus, slight deviations from the normal are frequently present, but the deviations are very irregular and variable in their appearance, my experience confirming Grey's⁷ observations.

Pointing Deviations During Induced Nystagmus from Cold Caloric.—In cerebellar abscess during an induced nystagmus, there is frequently an absence of the normal pointing deviation of the homolateral side, but in cerebellar growths, deviations are variable. I believe that in small tumors (especially those confined to the auditory nerve, as in a case observed by me of fibroma molluscum, causing complete deafness, and three years later involving the opposite ear) no alteration from the normal pointing deviations will be present. In the larger growths of the angle and those originating in the cerebellum and growing into the angle, the interference with the normal pointing deviations will depend on the tumor being extensive enough to interfere permanently with the functioning of the cerebellar cortex or tracts of the different parts.

In increased intracranial pressure from disturbances of the cerebrospinal fluid system, variations in the degree of increase may cause variable pointing deviations, the latter being at one time absent and at another time present (Fig. 2).

Vestibular Reactability of the Unaffected Side.—The diagnostic assistance obtained from the cold caloric of the ear on the opposite side to the lesion has been of the greatest value in the cerebellopontile angle tumor. In cases of partial deafness of one ear, cold caloric applied to the unaffected ear uniformly gave a *greatly diminished reactability*.⁸

After the deafness has become complete and symptoms of increased intracranial pressure, such as a papilledema, present themselves in my experience, it is frequently impossible to induce nystagmus by the syringing of cold water into the ear of the unaffected side; and if a slight nystagmus is induced, it is unaccompanied by vertigo or vomiting, at most a slight sensation of momentary giddiness being complained of. Also in the early cases in which hearing of the affected side is still present, while syringing of cold water in either ear induces a slight nystagmus, this nystagmus is not associated with vomiting or vertigo, but generally with a "floating" or swimming sensation of very transient duration.

The value of the diminution of the vestibular reactability of the unaffected side, as a sign of increased intracranial pressure, was originally stated⁹ in a communication to the American Otological Society in June, 1912. Confirmation of this observation has frequently occurred since that date.

Examination with regard to the reactability of the vestibular apparatus in patients suffering from marked internal hydrocephalus, the result of intracerebellar

Fig. 2.—Small tumor with large secondary cyst of cerebellum causing great hydrocephalus. During life, spontaneous nystagmus was absent. Vestibular reactability was at times present and again absent.

4. Holinger: Tr. Ninth Otol. Cong., p. 494.

5. Weisenburg, T. H., and Work, Philip: The Diagnosis of Tumors in the Posterior Cranial Fossa, THE JOURNAL A. M. A., Oct. 16, 1915, p. 1345.

6. Grey, E. G.: Studies on the Localization of Cerebellar Tumors, THE JOURNAL A. M. A., Oct. 16, 1915, p. 1341.

7. Grey, E. G.: Am. Jour. Med. Sc., 1916, 151, 693.

8. All caloric tests were made with patients seated in an upright position.

9. Eagleton, W. P.: Decompression for the Relief of Disturbances of the Auditory Apparatus of Intracranial Origin. Report of Three Cases with a Previously Undescribed Aural Condition, Laryngoscope, 1912.

tumors, may give very conflicting findings at different examinations.

Figure 2 shows a large intracerebellar glial cyst, with marked secondary hydrocephalus. The child was repeatedly examined during a year and a half prior to death. Spontaneous nystagmus was never present, and the spontaneous pointing deviations observed were too slight and transient to be definite. She was repeatedly subjected to the cold caloric. During a violent headache, doubtless due to a disturbance of the constantly present internal hydrocephalus, it was impossible to induce nystagmus or pointing deviations; a day later, however, cold water in either ear easily induced nystagmus, associated with normal pointing deviations and falling tendencies; but the nystagmus was never associated with a subjective sense of vertigo, or with vomiting.

It would seem, then, that alterations in increased intracranial pressure influences the possibility of the production of an induced nystagmus and of associated pointing deviations, abolishes the vomiting so often associated with the cold caloric, and lessens to a marked degree the subjective discomfort of dizziness.

The absence, following cold caloric of vertigo (the only complaint frequently being a transient giddiness) and vomiting, with a reduction of the vestibular reactivity of the unaffected side, may probably be regarded as symptoms of an increased intracranial pressure.

It would appear, therefore, that the aural manifestations of cerebellopontile angle tumor are: (1) progressive deafness, beginning with a disturbance of the proper relationship between the degree of hearing and the tuning fork reaction, especially the duration of the bone conduction to the degree of deafness, and ending in (2) total deafness; associated with (3) loss of vestibular reactivity of the affected side; and during the time that the vestibular apparatus is still functioning (4) a gradual readjustment of the vestibular apparatus of the contralateral as well as the homolateral side is going on, which is manifested by (a) a reduction or even a temporary abolition of its reactivity to the cold caloric (at least when applied in the upright position); (b) an absence of the vertigo and vomiting which normally accompany the induced nystagmus from the cold caloric, and (c) absence of spontaneous pointing deviations. As the cerebellar cortex becomes affected, however, may be added: (5) spontaneous nystagmus; (6) spontaneous pointing deviations, and (7) absence during an induced nystagmus of the normal pointing deviations of the homolateral side.

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Women in Industry.—In December, the Committee on Labor of the House of Representatives favorably reported a bill creating a woman's division in the Department of Labor to investigate and report on all matters pertaining to the welfare of wage-earning women, and especially to investigate the competitive influence of women in the several industries, the adjustment of modern industrial mechanism and management to the nervous organization of women, and the influence of industrial employments on the subsequent home life of wage-earning women. The chief of this division is to be a woman appointed by the secretary of labor. The division will cooperate with the Bureau of Labor Statistics and with the Children's Bureau, in the same department. The bill has the approval of the secretary of labor.

CLINICAL TYPES OF LABYRINTHITIS

WITH COMMENTS ON TREATMENT *

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Preliminary to a discussion of the clinical types of labyrinthine disease, may I remind you that the hardness of the labyrinthine capsule and its anatomic structure seem to be so arranged by nature that they form an unusually strong barrier against invasion by a purulent process?

It is estimated by Bezold that the labyrinth becomes involved only once in 500 cases of purulent otitis media. Friedrich and Hinsberg, on the other hand, estimate its occurrence once in 100 cases. The latter seems to me more nearly correct. The most vulnerable points in the labyrinthine wall are the horizontal semicircular canal, the fenestra ovalis, the fenestra rotunda, the promontory and, from the cranial side, the internal auditory meatus. The labyrinth may be invaded by a purulent process from three sources: (a) from the middle ear, (b) from the blood currents, (c) from the meninges. Our cases come chiefly from the first-mentioned type, wherein the middle ear spaces become the seat of a purulent lesion, which in turn advances into the bony structure, and during its progress attacks the labyrinthine wall and finally invades the delicate structures within the labyrinthine capsule. This gives rise to our so-called purulent labyrinthitis.

The following description of the various types of labyrinthine inflammation is based largely on clinical data. Thus, we broadly divide internal ear inflammation into circumscribed labyrinthitis and diffuse labyrinthitis. Before entering into a discussion of the subdivisions it may be well to eliminate those cases of chronic suppurative otitis media which have vertiginous attacks, but without disturbance in the functions of the labyrinth. These patients complain of attacks of vertigo, but they have hearing, a normal rotation test, a normal caloric test and no evidence of fistula. As a basis of these attacks of vertigo it is believed that middle ear disease has attacked the bony labyrinthine capsule without eroding the same. This type of labyrinthine disturbance is known as paralabyrinthitis. Probably there should be added to this type of paralabyrinthitis those cases in which the patient suffers for a few days from labyrinthine irritability as a result of irritation caused by injury to the stapes in performing a paracentesis. Such a case was recently reported by Guntzer. The family physician did a paracentesis on a patient who evidently had no infective process, but was suffering from an attack of acute catarrhal otitis media:

There was history of pain in the right ear for two days; no discharge; since the previous evening has been unable to walk; was dizzy and vomited. Examination of the right ear showed the membrana tympani slightly reddened, no bulging, and a pinhead perforation about the center of the superior-posterior quadrant, with a droplet of serous red discharge exuding. The patient had a spontaneous horizontal nystagmus to the left and rotary on looking up; on attempting to rise fell toward the right. Hearing was not impaired, no mastoid tenderness, no fever, pulse 88. Diagnosis of labyrinthine irritation was made, but no assignable cause could be elicited. This condition lasted ten days before the patient was able to leave the bed. At a subsequent office visit the patient confided

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