the hemorrhage is confined to the cochlea, in which case vertigo is absent. With or immediately following this occur nausea or vomiting, severe tinnitus (in some cases preceding the attack), occasionally profuse perspiration, and impairment of the hearing up to complete deafness. The symptoms thus given constitute what was formerly generally designated as Ménière's disease, and is the only condition to which that name should be given. Amelioration of these symptoms takes place in a short time, the vertigo, hardness of hearing, and tinnitus continuing longest. The hardness of hearing rarely disappears entirely, and the tinnitus is likely to persist, although diminished in intensity. There is always danger of a repetition of the hemorrhage. The functional tests give varying responses according to the locality and the extent of the hemorrhage, but always confirm a diagnosis of involvement of the sound-perceiving apparatus. G. C. Smith, in the Boston *Medical and Surgical Journal* for November 24th, 1898, thus describes a case of so-called Ménière's disease which he observed: "The patient, a man, sixty-two years old, in fair health, while sitting one evening in conversation, suddenly felt as though he was seized by some power and hurled a considerable distance, striking against the left side. He was unconscious for the moment, but arose with assistance. He was unable to walk on account of vertigo, and had to lie down. This was followed by vomiting and vertigo, which continued all night. There was also a hissing tinnitus, and on the following day there was deafness in the left ear. In the following five months he had six seizures similar to the first one. During all this time the noises in the head were continuous. The deafness, though incomplete, never improved after the first seizure."

The symptoms of embolism and thrombosis are presumably similar to those of hemorrhage. Thus Habermann, at the meeting of the German Otological Society held May, 1898, mentions the case of a man, fifty-three years old, who suffered one night from an attack of hemiplegia, which in a few weeks disappeared, with the exception of a slight weakness. Four years later, he had a relapse, with frequent attacks of tinnitus, vomiting, and deafness. The whisper was not heard on the affected side. The field of hearing embraced the tuning-forks from C^1 to C^7. The affection was probably of embolic origin, possibly in the brain along the course of the auditory nerve.

Serous effusions may give rise to the same symptoms, but more fleetingly. Rosenstein was the first to consider the possibility of an œdema of the auditory tracts causing disturbance of hearing in nephritic patients. Other authors also favor the assumption of a transitory œdema as causing transitory functional disturbance in parts of the brain.

Labyrinthitis Hyperplastica.—The most marked form of this inflammation is seen in connection with syphilis, usually as a late manifestation in the acquired variety, or around puberty in the hereditary, and gives rise to deafness, appearing gradually or suddenly, subject to periods of quiescence and exacerbation; it is also commonly accompanied by loud aural tinnitus. Vertiginous attacks and disturbances of equilibrium are usually slight unless the exudative form of inflammation is induced. The disease is likely to be accompanied by very violent headache, often nocturnal when due to syphilis. Both ears are usually affected. The sudden deafness coming on with serous saturation or lymphoid infiltration may disappear almost completely; but usually the hyperplastic formations are causative of a certain amount of permanent deafness. The functional tests leave no doubt as to the seat of the trouble in the sound-perceiving apparatus.

Labyrinthitis Exudativa in its most acute form comes on very suddenly, with perhaps a rigor; it is commonly associated with fever, nausea, or vomiting; with profound deafness, and marked derangement of co-ordination; at times, with stupor or delirium (although usually the mind is clear); with intense tinnitus and vertigo, and, in some cases, with pain. This very acute form occurs with epidemic cerebro-spinal meningitis (Voltolini described this form of inflammation as a primary inflammation, but there is not much doubt that it is an affection secondary to a more or less localized meningitis), with the acute infectious diseases (measles, scarlet fever, diphtheria, etc.), with epidemic parotitis, etc. Most of the symptoms abate or disappear in the course of a few days or a few weeks, but the staggering gait and deafness are more persistent, the latter rarely improving to any great extent. Functionally, the upper-tone limit is greatly lowered; B. C. is markedly reduced throughout the entire musical scale, or is destroyed for a part or for all of it; A. C. > B. C.; the power of equilibrium is much impaired. The less acute forms of exudative inflammation of the labyrinth give rise to vertigo (unless confined to the cochlea), to a sudden loss of hearing power, intense tinnitus, lowering of the upper-tone limit, with B. C. reduced or absent, and A. C. > B. C., and to disturbances of equilibrium.

Escape of Pus into the Inner Ear sometimes occurs in cases of acute suppuration of the middle ear and presents distinct symptoms. If it breaks through into the semicircular canals there will be vertigo, vomiting, disturbances of equilibrium, and nystagmus; if into the cochlea, great deafness. Nystagmus alone justifies the diagnosis. The pus may work its way indirectly into the upper semicircular canal from a subdural abscess. In chronic cases, Jansen states that the point of entrance is invariably the horizontal semicircular canal, but J. Orne Green presented three specimens at the 1898 meeting of the American Otological Society, in all of which the perforation was into the vestibule.

Labyrinthine Necrosis, according to Bezold, follows otitis media purulenta chronica; exceptionally otitis media purulenta acuta. Generally the inner cochleal skeleton, with either the entire first turn, or only a part of it, is the structure most liable to necrosis. There are two ways by means of which the otitis media purulenta may extend to the cochlea: (1) through the small cells which lie at the base of the tympanum and around the commencement of the Eustachian canal, and which surround the lower aspect of the cochlea; (2) into the cochlea through a perforation caused by rupture of the membrane of the round window. The sequestrum formation is very much more extensive in the early years than in later life. Disturbance of equilibrium with simultaneous tinnitus and occasional vomiting is generally present. Vertigo disappears with the extrusion of the sequestrum, and this symptom, taken in connection with the tinnitus and vomiting, may be regarded as an evidence of irritation of the nervous terminals in the ampullæ and sacculi; these symptoms may also be taken as marking the date for the extension of the middle-ear process to the labyrinth. Paresis or paralysis of the facial nerve is a constant symptom of labyrinth necrosis, and usually appears one or several months after the first attack of vertigo. It exists in from eighty to ninety per cent. of the cases, and is permanent in fifty per cent. In the other cases, the paralysis exists only for a short time and disappears before the sequestrum is extruded. Prolonged pain is a constant symptom, and is of such severity as to prevent sleep for weeks or even months; it also produces a poor physical condition and emaciation. This pain comes on from two to nine months before the expulsion of the sequestrum; it is at first felt in the entire half of the head and is accompanied by fever, vomiting, etc., probably due to meningeal irritation. Later, the pain becomes localized in the ear and takes on a more boring character. Preceding or at the beginning of the symptoms the otorrhœa becomes more profuse and is creamy and very fetid. Exuberant polypoid hypertrophies are present.

When *the Nerve Trunk is affected* the most prominent symptom is impairment of hearing. There are also present: tinnitus, vertigo, staggering gait, and excessive functional exhaustibility. Usually unilateral, it may be bilateral, as in tabes dorsalis. Hardness of hearing is generally most pronounced for the tuning-forks of middle

register, perception for high and low notes being fairly well preserved. B. C. is very much impaired.

Word-deafness (sensory aphasia) furnishes the most reliable sign of involvement of *the cortical area*, usually of the left first temporal convolution. The function of both ears is usually impaired; tinnitus is more commonly absent; B. C. is reduced in duration.

Concussion due to Gun-firing occasions alterations chiefly in ears that were not normal before exposure. The perception by bone conduction is abbreviated, owing to over-irritation and subsequent exhaustion of the nervous portions of the ear. The field for tones is not contracted. Tinnitus, vertigo, and headache are not usually complained of, and, in the case of normal ears, very little permanent injury follows ordinary artillery practice. Permanent injury to the hearing is produced when the ear has been to some degree abnormal before the exposure.

Concussion of the Head may provoke symptoms indicating an involvement of the sound-perceiving apparatus, even up to complete deafness, and this latter may be induced without any recognizable changes being necessarily found in the labyrinth on post-mortem examination. The symptoms usually present are: diminution of hearing, tinnitus, vertigo, headache, unconsciousness, in some cases pain, occasionally acoustic hyperæsthesia or alteration in the pitch of certain tones, etc.,—one or all. These symptoms have been explained as due to shock to the acoustic nerve, or to basilar inflammation resulting from a blow, or to hemorrhage at the point of origin of the acoustic nerve.

Fractures of the Petrous Bone, involving the labyrinth, are accompanied by hemorrhage from the external meatus, or, if the tympanic membrane is not ruptured, the blood may pass through the tympanum and the Eustachian tube into the throat. As additional symptoms may be mentioned: a serous-looking discharge in considerable quantity; very pronounced subjective noises; disturbances of equilibrium and vertiginous symptoms; and facial paralysis in fifty-five per cent. of the cases. In both concussion and fracture the functional tests all show an involvement of the sound-perceiving apparatus.

Neurotic Disturbances of the sound-perceiving apparatus, which may be unassociated with pathological anatomical alterations, are by no means uncommon and are described below.

Acoustic Neurasthenia has as symptoms: impairment of hearing, varying from mere slowness of hearing (acoustic torpor or lassitude) to considerable deafness, especially marked under any prolonged strain or confusion of sound; mental anxiety, or extreme physical fatigue; and rapid improvement of the symptoms after rest. Tinnitus may or may not be present, and is increased by fatigue; paræsthesiæ are common, with great fluctuation of the ability to hear; the upper-tone limit is not apt to be impaired, but the duration of B. C. is lowered throughout the musical scale, and there is great functional exhaustibility of the acoustic nerve. Either one or both ears may be involved, though usually both are affected. The patients are generally neurasthenic, and any circumstance which aggravates this condition is the cause of marked decrease in the hearing. This condition is frequently associated with that which is next to be described.

Acoustic Hysteria is usually associated with great deafness which appears suddenly and is not subject to the fluctuations noticeable in neurasthenia; it remains the same throughout the continuance of the attack. Vertigo is never present and tinnitus is not frequent; one or both ears may be affected or the attack may pass from one ear to the other; paræsthesia or anæsthesia of the external auditory canal and the tympanic membrane may be present; functional tests are apt to be contradictory.

For example, D. Grant reports a case of hysterical nerve deafness with spontaneous recovery. A girl, aged eighteen years, was first seen May 27th, 1895. She gave the history of gradually increasing deafness for three years, the hearing having grown much worse after the extraction of some teeth three months before the visit. Only very loud conversation was heard. Watch heard at six inches; Galton up to the mark 3.8. B. C. diminished. Rinné's test positive. Tuning-fork hearing lost for C^2 and C^1, while for the other forks from C up to E^5 the amount of hearing power varied from three to fifteen per cent. Hearing returned, after a complication of ailments, in January, 1897.

Veit also has reported a case of hysterical deaf-mutism in a man of twenty-six years, who awoke a deaf-mute six weeks before he was first subjected to an examination. In writing he could very well make himself understood. Anæsthetic regions and palsies were missing. The deafness was absolute. Attempts to speak were followed only by inarticulate sounds. After letting him read the sentence, written by the physician before him, "You will hear again in a few minutes," he was catheterized with a good deal of difficulty, but hearing and speech returned at once.

Hyperacusis is an over-excitable condition of the acoustic nerve, sometimes even painful, occurring generally periodically in connection with great nervous or mental excitement or with neuralgia, or manifesting itself after the patient has partaken of stimulants. It also occurs in the incipiency of inflammatory affections of the ear.

Paracousis consists in the false perception of the pitch of a sound. *Paracusis loci* is the term used for designating inability to tell the direction from which the sound comes; it depends upon the difference in the acuteness of perception of the two ears. As our judgment of the direction of sound depends upon the binaural hearing, in unilateral deafness the apparent source of the sound will be projected in the direction of the normal-hearing ear.

Diplacousis is a form of paracousis in which a single tone is heard double; either each ear perceives the tone differently and it seems doubled (D. binauricularis), or a double perception of a single tone is got by one ear (D. monauricularis); the two tones differ from each other in time (D. echoica) or in interval (D. harmonica or disharmonica).

"*Nervous Tinnitus*" may exist as a pure neurosis without difficulty of hearing. It is observed as an irritable condition of the nerve in convalescence from severe febrile affections; also in connection with sexual excesses, intemperance, abuse of tobacco, over-fatigue of the auditory nerve, and extreme mental disturbance. It may continue indefinitely, the hearing remaining unimpaired.

Hallucinations of Hearing may occur in ear diseases without a changed condition in the brain.

Color-hearing is a term employed to define that phenomenon by which certain tones always excite in some people the sensation of color; certain sounds invariably inducing the perception of a definite color, which color is always the same for any particular sound. The cases fall into two groups: (1) A crude color sensation, often very beautiful, is associated with certain sounds, such as each of the vowel sounds, musical notes, or particular musical instruments; the appearance being usually that of a transparent colored film, similar to a rainbow, in front of the observer, but not obscuring objects; (2) color sensations are experienced whenever letters or written words (symbols of sound) are spoken or thought of, so that when a word is uttered the subject visualizes the letters, each of which has a distinctive tint.

Deaf-mutism.—One of the most important results of labyrinthitis is the production of deaf-mutism. In the United States there were about 38.2 deaf-mutes to every 100,000 inhabitants, but this proportion is apparently diminishing. They belong, to a great extent, by birth to those classes of society which are least favorably situated economically as well as socially. In the majority, the deaf-mutism develops before the end of the third year, but may develop as late as the eighth year. The pathological seat of the causative process is, almost without exception, in the labyrinth. In connection with the labyrinth, the middle ear is surprisingly often found to be affected—only exceptionally as regards lack of formation, but almost regularly by violent inflammation, generally of a purulent nature. Deaf-mutism occurs more

frequently in the male sex. More than half the cases are due to acquired deafness, and epidemic diseases are probably most often the cause of the deafness. Bezold is probably nearly right in his statement that about forty-three per cent. are totally deaf, and it is the general opinion that among these the acquired cases are more numerous than the congenital. There is noticeably a very frequent occurrence of partial defects in the musical scale, in which sometimes the upper and sometimes the lower limit of tone is absent; sometimes single or multiple gaps or islands are found which show no perception of sound whatever. Only about 8.4 per cent. have hearing power sufficient for intercourse with other people. Disturbances of equilibrium (static or dynamic) are present in fifty per cent., and those showing normal equilibrium are also much more apt to have normal speech. Heredity exerts a great influence, especially in those families in which there are many cases of hardness of hearing, but direct transmission is usually absent, as Mygind found that not a single child of deaf-mute parents was itself deaf and dumb.

This is somewhat at variance with the findings of E. A. Fay, who has studied the marriages of deaf-mutes in America, and states that the marriages of congenital deaf-mutes give birth to more deaf-mute children than marriages of persons with acquired deaf-mutism in the proportion of 12 to 4.2 per cent. The number of deaf-mute children is increased if the deaf-mute's parents are relatives, especially if, besides themselves, there are other deaf-mutes in the families. Consanguineous marriages are causative only when joined to hereditary and other influences (as constitutional disease). Deaf-mutism is especially apt to occur in those families in which many children have been born in rapid succession. Gillespie has drawn attention to the frequency of goitre in deaf-mutism; and Lemcke, to that of affections of the naso-pharyngeal tract, especially adenoid vegetations. The bodily growth keeps pace with that of normal persons, but there is defective brain development; as a rule, however, they are endowed with organic and mental sensitiveness but little inferior to the normal. They do not exhibit a higher mortality than normal individuals living under the same circumstances, but they are especially prone to lung diseases. Nearly half of all deaf-mutes over twenty years of age are obliged to fall back on the help of others for their maintenance. Marriages contracted by deaf-mutes exhibit a very small degree of fertility. (For further information on this subject, consult the articles on *Deaf-Mutism* in the present volume.)

Diagnosis.—The diagnosis of diseases of the auditory nerve has been almost sufficiently indicated in the symptomatology, but there are a few points which it may be well to emphasize further.

In any case of hardness of hearing the first thing to be determined is whether the lesion is located in the sound-conducting or in the sound-perceiving apparatus. The antagonistic reactions to the functional tests may be tabulated as follows:

Diseases of the Sound-Conducting Apparatus.	Diseases of the Sound-Perceiving Apparatus.
Upper-tone limit very little if any lowered.	Upper-tone limit noticeably lowered.
Lower-tone limit by A. C. elevated.	Lower-tone limit by A. C. not elevated.
Absolute duration of perception of B. C. increased throughout the musical scale.	Absolute duration of perception of B. C. diminished or abolished throughout all or in parts of the musical scale.
B. C. > A. C. both in intensity and in duration in the lowest part of the musical scale, and ascending with the gravity of the disease (Rinné).	A. C. > B. C. both in intensity and in duration throughout the musical scale (+ Rinné).
Weber's test heard in the diseased or the harder-hearing ear.	Weber's test heard in the normal or better-hearing ear.
Deeper tones of speech not heard; higher tones well heard.	Deeper tones of speech well heard; higher tones not heard.

In order to bring differing reactions more graphically before the eye, the author has arranged them below in the *schema* originally devised by himself. In this the normal reaction in the healthy ear is given for purposes of comparison; the numerals representing the duration of perception in seconds, the Rinné showing whether the respective forks are heard louder by A. C. or B. C. at the initial point.

In marked disease of the sound-conducting apparatus the reaction will be approximately as given below:

I. Average Normal Ear.

A. C.	A. C.	A. C.	A. C.	A. C.	A. C.	Rinné.
22 / 12	25 / 13	15 / 7½	33 / 13	32 / 13	22 / 14	Schwabach { B.C. / A.C.
C¹	C	C¹	C²	C³	C⁴	Tuning-fork.
Galton 1½.			Weber =			

II. Average Case of Otitis Media Purulenta Recurrens.

B. C.	B. C.	B. C.	B. C.	B. C.	Equal.	Rinné.
0 / 14	8 / 14	8 / 11	13 / 17	15 / 16	13 / 14	Schwabach { B.C. / A.C.
C¹	C	C¹	C²	C³	C⁴	Tuning-fork.
Galton 2.			Weber in the affected ear.			

In disease of the sound-perceiving apparatus the following reactions will serve as a type:

III. Average Case of Otitis Interna.

A. C.	A. C.	A. C.	A. C.	A. C.	A. C.	Rinné.
12 / 4	17 / 6	18 / 7	21 / 6	15 / 4	10 / 2	Schwabach { A.C. / B.C.
C¹	C	C¹	C²	C³	C⁴	
Galton 2.7.			Weber in the better-hearing ear.			

In cases in which there is an affection of both the sound-conducting and the sound-perceiving apparatus, both upper- and lower-tone limits are contracted, the duration of B. C. is impaired, B. C. is better than A. C. (—Rinné) for the lower forks, while A. C. is better than B. C. (+Rinné) for the higher forks, and both the higher and deeper tones of speech are imperfectly heard. The degree in which one or the other apparatus is responsible for the hardness of hearing is indicated by the closeness of the resemblance of the results of the functional tests to the reactions given by the one or the other type of disease.

In the matter of locating the lesion in any particular portion of the sound-perceiving apparatus much has yet to be learned, but the following deductions seem to be well established as the result of post-mortem examinations of cases clinically observed before death:—Word-deafness points to involvement of the cortical areas; lower- and upper-tone limits fairly well preserved, with deafness for forks of middle register and greatly impaired B. C., indicate involvement of the nerve stem; disturbances of equilibrium may occur in the course of any pathological process that causes irritation of the terminal filaments in the vestibule or ampullæ, of the nerve fibres in the auditory nerve stem, or of the central origin of the nerve; pathological processes involving the cochlea alone do not induce vertigo; the cochlea is the only part specialized for the perception of sound, as the retina is for light, and its total destruction is followed by total deafness; it is probable that the lower notes are perceived at the cupola, and the higher notes at the base.

When the attempt is made to establish a diagnosis these deductions should be borne in mind while studying

the results of the functional testing. At the same time it should always be remembered that it is often impossible to determine whether the disease is in the labyrinth, in the nerve trunk, or at some point in the intracerebral course of the nerve.

According to C. H. Burnett, anæmia of the labyrinth, causing deafness, tinnitus, and vertigo, may be diagnosed in two ways, viz.: by the temporary congestion induced by eating, and also by the inhalation of nitrite of amyl. If temporary improvement in all or any of these symptoms ensues after eating or after the inhalation of a few minims of amyl nitrite, the conclusion is that the case is one of chronic anæmia of the labyrinth.

In fracture of the petrous bone, the escape of cerebro-spinal fluid is not essential, and no certain conclusions with respect to the anatomical situation, gravity, or the subsequent behavior of the fracture can be drawn from the external appearances in the ear and from the functional disturbances. In most cases both the internal and the middle ear are affected together. Fracture may occur without loss of hearing if the labyrinth is not involved in the fracture line.

The diplacouses are, in the author's experience, usually due to affections of the middle ear, as in a case recently observed of diplacusis echoica coming on during the acme of an attack of otitis media subacuta.

The neuroses are recognized by their symptoms and by the peculiar constitutional condition of the patient.

If the few remarks made above in connection with the subject of symptomatology are borne in mind, the diagnosis, according to our present knowledge, should not offer insuperable obstacles to the conscientious observer.

Prognosis.—The prognosis is always hopeful in those cases in which there has been no destruction of the anatomical elements, as in anæmia, neurasthenia, etc. On the other hand, it is always unfavorable in those cases in which such destruction has taken place. The condition remaining after a trial of treatment of moderate duration is apt to be the condition that will remain permanently, except in the case of hemorrhage, in which repeated attacks will tend to further impairment of hearing.

Amelioration may and usually does take place in the other symptoms, such as vertigo; but the hearing improves only so far as the anatomical elements develop recuperative power, and when that power is exhausted improvement ceases.

Treatment.—The treatment of anæmia of the labyrinth is in most cases practically that of the treatment of general anæmia, as in the anæmia and oligæmia following gestation and parturition; the condition is often temporarily benefited by doses of trinitrin, gr. $\frac{1}{100}$, two or three times daily. In the local anæmia due to aneurism or atheroma very little can be done; when it is due to pressure brought on by changes in the middle ear, operative interference to relieve that pressure should, if possible, be undertaken.

Hyperæmia, if acute, should be met by local blood-letting, purgation, and rest, while at the same time the diet is limited and stimulants are interdicted. The causative agency should always be searched for and corrected, as far as possible, on general lines. Regulation of the diet and bowels, curtailment or denial of stimulants, and correct ordering of the care of the body and method of life are always indicated and produce the best results.

Hemorrhages into the labyrinth should be treated, until the acute symptoms subside and absorption begins, by complete bodily rest, local blood-letting, purgation, hot foot baths, limitation of diet, and abstinence from the use of all stimulants, alcohol, tobacco, etc. Later, comparative rest and abstention from mental or physical work, with the regulation of the diet and bowels, will do more to favor absorption than the administration of drugs. The use of iodide of potassium in gradually increasing doses has given the best results in the author's hands. It may be employed conjointly with the other measures recommended, or independently, if for any reason they cannot be adopted. Pilocarpine has been strongly advocated by some authors; it should be given either by the mouth or hypodermatically, beginning with gr. $\frac{1}{4}$, two or three times daily, and increasing the dose until the physiological effect of the drug is obtained, when the patient is held to that dosage for a shorter or longer time.

In the hyperplastic form of labyrinthitis, regulation of diet and of the bowels, and denial of stimulants, counter-irritation over the mastoid, and attempts to provoke derivation and, most important, the attempted removal of the cause, are the proper measures to be employed. Resolution may be encouraged by the administration of iodide of potassium or of pilocarpine, if these are not contraindicated by the condition which caused the lesion or by the state of health of the patient.

During the acute stage of the exudative form of labyrinthitis as much should be done as possible to diminish the intensity of the attack and to limit the extension of the process. This in most cases amounts to very little. Rest in bed is imperative, limitation of the diet is advisable, cardiac sedatives and diaphoretics should be prescribed, the bowels should be regulated, and derivatives not contraindicated by the general condition should be employed. After the acute symptoms have subsided, resolution is to be encouraged by the continuance of rest, by the adoption of a light but nourishing diet, by the regulation of the excretory organs, and by the administration of those remedies which are known to have an effect on the pathological deposits and new formations—such, for example, as the iodide of potassium, mercury, pilocarpine, etc.

Jansen holds that when pus penetrates from the middle ear into the semicircular canals, the operative field should be extended so as to effect the removal of the horizontal or even the vertical canal with narrow chisels or with the electro-motor drill.

In labyrinthine necrosis the treatment, according to Bezold, should consist in systematic cleansing, insufflation of boric acid powder, and removal of granulations. A forcible extraction of the sequestrum before it is loose is not advisable. The mastoid process is rarely involved. A properly executed radical operation insures a safer recovery than if the sequestrum is spontaneously eliminated through a narrow canal, especially if signs of general disturbance are present. The result in six out of ten cases in which such an operation was resorted to, was complete recovery with epidermization of the cavity. Recovery probably also occurred in a seventh case. In one case there resulted atresia of the bony canal. One patient died from some complication in the other ear, and one died eleven days after the removal of the sequestrum, presumably of meningitis.

The regeneration of the affected nerve elements may be encouraged by the administration of the various nerve stimulants and nerve foods, such as strychnine, phosphorus, etc.

The principal treatment for concussion and fracture is rest and the meeting of symptoms as they arise.

The treatment of acoustic neurasthenia is, of course, that of the general neurasthenic condition. The general health should be improved by all the means at our command. The author has found that the feeling of well-being produced by the administration of gelsemium is a very important aid in inducing the patient to attempt and to adhere to those regulations which are necessary to the attainment of this much-hoped-for improvement; it should be administered in the form of the fluid extract. Strychnine has produced only a temporary improvement in the author's hands. The general health must be improved if any permanent betterment is to be attained, and gelsemium has the power of stimulating the ambition of the neurasthenic to the extent of accomplishing the tasks necessarily set for this purpose.

Hysteria requires administration of those remedies—pharmaceutical, psychical, and physical—usually recommended for use in the general condition.

The various other neuroses, reflex and otherwise, are to be treated from the standpoint of the cause.

It will be noticed that the subject of the treatment of