

SOME CURABLE AFFECTIONS OF THE ACOUSTIC NERVE.*

DR. ALFRED LEWY, CHICAGO.

Under this title it is intended to describe a group of cases differing in etiology but having in common a lessened hearing-power for the spoken or whispered voice; for tuning-forks especially by conduction through the cranial bones; and their difference from actual degeneration of the acoustic nerve in that recovery ensues under appropriate treatment.

It is not the intention to consider in this paper hysterical deafness nor acute anemia of the labyrinth, both of which have won considerable text-book recognition, but more especially a form of hardness of hearing of rather gradual onset, due to general anemia of longer standing; to a general systemic depression, or to neurasthenia. The drug intoxications and the toxemias of the various infectious and constitutional diseases which may cause a disturbance of function of the acoustic nerve, as of others, without necessarily resulting in a permanent lesion, will also be briefly considered.

The references to this class of cases, with the exception of drug intoxications and the toxemias of the infectious diseases, in most text-books are casual, and direct reports of such in the literature are not frequent, so that we may infer either that they are rare, which I do not believe, or that they have not attracted much attention. Perhaps the fact that they recover, and no doubt often spontaneously, accounts somewhat for the lack of attention paid to them.

I am of the opinion that cases of what we may call functional depression or asthenia of the cochlear nerve due to various causes are fairly common, either alone or associated with tubal catarrh. It is reasonable to suppose that within normal limits the hearing as well as other functions is subject to variations. In fact, Edelmann,¹ in experimenting on himself with his normal siren (an improved and accurate acoumeter), found such to be the case. In abnormal degree this form of deafness is reported during convalescence from typhoid.²⁻³ Politzer,⁴ in a foot-note, refers to "Torpor nervi acustici" described by Rohrer as occurring in persons under 50, with otitis media chronica. Urbantschitsch⁵ also mentions torpid-

*Read at the meeting of the Chicago Laryngological Society, May 21, 1912.

ity of the acusticus, a progressive form associated with middle-ear disease, but says nothing as to prognosis. He also describes fatigue of hearing, "Ueberanstrengung," temporary or permanent, believed by him to be due to cramp of the tensor tympani. The tuning-fork findings are not described. Fatigue of hearing is also described by Hammerschlag,⁷ Gelle,⁸ Blake⁹ (occupational), Lannois¹⁰ and Dench.¹¹

Ballenger, treating of hysteria, mentions the fatigue symptom (after perception for the sound of the vibrating tuning-fork has ceased, the tone is again heard if the fork is removed for a moment and replaced without having been struck again.) Under the head "neurasthenia," Phillips¹² describes hyperacusis with tinnitus, but says nothing of depression of function. Barnhill and Wales¹⁴ indicate that the symptoms of nerve-deafness may be found in chronic anemias or after exhausting illness and Dench¹⁵ goes a little more into detail on this subject. Grunert¹⁶ mentions deafness due to sub-acute anemia. Urbantschitsch⁶ mentions deafness in myxedema, responding to thyroid treatment; it is also described in nephritis,¹⁷ increasing and decreasing with the anasarca, in recurrent fever,¹⁸ and periodically in malaria.¹⁹ There is also described vaso-motor deafness of sudden onset, associated with redness of the same side of the face and disappearing just as rapidly.

Can we differentiate these recoverable cases from those of organic changes in the nerve by the clinical examination? I think we can, at least with probability, by careful investigation of the etiology and by careful tuning-fork tests. It may sometimes be necessary to supplement these by a short period of observation. Nerve-degeneration is more apt to occur in men; in those whose occupation subjects them to continuous noise; in both sexes past middle age; in young people as the result of one of the infectious diseases, particularly meningitis, mumps, or hereditary syphilis, while in older people acquired syphilis, and later arterio-sclerosis are factors. Osteomyelitis and leukemia are occasional causes. The loss of hearing is usually severe, unequal in both ears except when very advanced, and the loss of the upper tone-limit as a rule, or of other areas of the scale, is marked. In a series of cases of neuritis described by Zytowich,²² the perception taken all along the scale from C to c⁴, was very variable. I might mention here that in six of seven cases of acoustic nerve-degeneration examined by me the vestibular reaction to rotation was subnormal.

In the simple asthenia or functional depression of the cochlear nerve the etiological factor is apt to be a depressing illness, a

chronic anemia or *nervous stress*; the loss of hearing not so severe (about 5 feet or more for the whispered voice) and not of long standing, both upper and lower tone-limits nearly normal; except the loss of lower tones when tubal catarrh is associated, and with this there is shortened perception for the tuning-fork through the cranial bones by both Schwabach and Rin   tests. In the neurasthenic form the vestibular r  action to rotation is increased,²³ and the fatigue symptom is present. The association of this fatigue symptom and shortened bone-conduction with tubal catarrh lead to the suggestion that in some cases the internal ear-disturbance may be due to over-exertion in the attempt to hear clearly in the presence of defective conduction of sound. (See case of the piano tuner reported below).

Another condition occasionally giving the findings of nerve-deafness with prompt recovery on removal of the cause is impacted cerumen. It is not to be forgotten that genuine nerve-degeneration may be associated with cerumen. This condition is now being investigated by my colleague, Dr. Sonnenschein.

At this point I should like to offer a suggestion, without, however, sufficient clinical evidence to warrant the name of theory: that a purely local lesion of the cochlear nerve, that is a lesion specifically affecting the peripheral neuron, is apt to show diminished hearing especially in certain areas of the tone scale, while a systemic depression, acting through the sensorial center or possibly through the nucleus of the eighth nerve will show diminished hearing of approximately the same degree for all tones, and may be associated with depression or hyperesthesia of other senses.

I present herewith three case-histories in which the internal ear-disturbance appears to have been of the so-called functional variety.

Case 1: B. B., age 39; occupation, piano tuner; has congenital cataracts and ocular nystagmus. Following "a cold" two months before, had dull pain and feeling of fullness in both ears, accompanied by a noise, like running water. Examination, November 8, shows the following:

Membrana tympani: right and left, retracted, dull. Schwabach: A fork 10 seconds and again 10 seconds on replacement; (fatigue symptom); normal for our fork, 28 seconds. Weber: not lateralized. Rin  : right, +12:33; left, +12:35; normal, +15:35. Whisper: right and left, 10 feet. Low limit: right, G_2 (24 v. d.); left, G_2 (24 v. d.). High limit: right and left normal for c^4 .

After inflation the whisper was heard 20 feet in both ears. Fatigue symptom still present. Diagnosis: tubal catarrh: asthenia of

the acoustic nerve. Tubal catarrh because of the marked improvement on inflation, and the loss of low tone limit; asthenia acustici because of the relatively shortened bone-conduction by both Schwabach and Rinné tests, and the fatigue symptom. Treatment: inflation, massage; strychnia.

December 13: Whisper before inflation: right, 18 feet; left, 20 feet; Schwabach, 23 seconds; no fatigue symptom present. Low limit: right, C_2 , 16 v. d.; left, G_{21} , after inflation C_2 .

Case 2: Mrs. M. W., age 25; for several months has suffered from some pelvic disturbance, the exact nature of which could not be learned. For a month or more difficult hearing and tinnitus which she described as throbbing. Examination, October 16:

Membrana tympani: right, negative; left, negative. Whisper: right, "four" at 4 feet, "six" at 8 feet; left, "four" at 4 feet, "six" at 7 feet. Schwabach: variable, average 15 seconds (normal, 28 seconds.) Weber: left, Rinné; right, +8:18; left, +8:18. Low limit: right, C_2 , 16 v. d.; left, C_2 , 16 v. d. High limit: right, 0.3; left, 0.3; (normal, 0.5.)

This test shows Schwabach about 50 per cent of normal, Rinné slightly more than 50 per cent for both bone and air conduction. No loss of either high or low tone limits, in fact, the high limit is slightly increased in sensitiveness. There was no improvement on inflation at this time. The apparent better hearing for sibilants is not entirely consistent with the tuning-fork tests. No evidence of hysteria. Diagnosis: asthenia nervi acustici.

She was referred to her physician for general roborant treatment, and on November 26 another test showed as follows: Whisper: right, 18 feet; left, 14 feet; after inflation, both ears 22 feet; Schwabach varied from 20 to 30 seconds; Rinné: right, +10:23; left, +15:33. Apparently a mild tubal catarrh had supervened; meanwhile a considerable degree of recovery from the asthenia acustici had been obtained.

Case 3: Mrs. H. A. A., age 30; two children; influenza, followed by double frontal sinusitis two months before, from which recovery seems complete. Now complains of whizzing noise in both ears. Examination made with the Reiner set of forks as recommended by Neumann shows the following:

Membrana tympani: right and left normal. Whisper: before inflation, right, 10 feet; left, 8 feet; after inflation, right, 14 feet; left, 12 feet. Schwabach, 10/15 of normal. Rinné, right, +10:25; left, +10:25; normal, 15/60. C, right, 35/70 of normal; left, 22/70 of normal. c^4 : right and left slightly shortened. Diagno-

sis: tubal catarrh with asthenia nervi acustici, probably due to anemia. Returned to her physician for tonic treatment, under which she recovered.

Of the drug intoxications, perhaps quinin is the best known. The destructive effects of this drug are exerted largely upon the ganglion cells of the cochlea. The degeneration of the organ of Corti and of the acoustic nerve are believed to be secondary effects and the hemorrhagic exudations supposed to be due to agonal convulsions.²⁴⁻²⁶ The cases which recover, that is to say, those in which no permanent organic lesion is established, are more frequent, and we can only conjecture that some form of irritation, resulting in depression of function takes place. Probably, judging from the hyperemia of the middle-ear, there is also hyperemia of the labyrinth. It is difficult to say sometimes whether the symptoms are due to the drug or the disease for which the drug is given, but where the disease is not one which produces nerve-deafness, and where the symptoms improve rapidly after withdrawal of the drug, other factors being excluded, one is justified in assuming that the drug has been responsible. Many drugs are reported to have caused deafness with the functional findings of nerve-deafness, and I believe one may safely assume that any of these may cause only temporary deafness when the toxic effect is not too severe.

The frequent use of salvarsan at the present time has brought arsenic preparations to the foreground. The recently reported cases of deafness due to this drug have apparently been cases of permanent injury. Arsenic also affects the ganglion cells²⁷ as do the salicylates.²⁸⁻²⁹

Alcohol and tobacco toxemias often co-exist. The alcoholic form may be part of an alcoholic polyneuritis. Amblyopia may also be present. The pathology of the amblyopia is a degeneration of the papillo-macular bundle, probably an ascending degeneration due to death of the retinal ganglion cells.³⁰ Alcoholic neuritis, perhaps more than others may be associated with a conduction deafness due to a catarrhal process. Zytowich claims to have shown³¹ that characteristic for the alcoholic form of neuritis is a relative loss of low tones, associated with shortened bone-conduction. Degenerative changes found by him were more marked in the upper part of the cochlea. Alexander states³² that in chronic alcohol and nicotine poisoning the vestibular reaction to the usual tests may be either increased or decreased.

Other substances reported to have caused deafness are chloroform,³³ iodine,³⁴ carbon monoxide,³⁵ oil of chenopodium³⁶ mush-

rooms,³⁷ lead,³⁸ mercury,³⁹ phosphorus,⁴⁰ silver,⁴¹ carbon dioxid,⁴² carbon disulphate,⁴³ smoke,⁴⁴ ergot,⁴⁵ morphin.⁴⁶

A case of quinin poisoning seen by me gave the following findings, which are somewhat incomplete on account of loss of the record: The whispered voice is heard 5 feet in either ear; slight improvement on inflation; Schwabach 60 per cent normal; Weber indefinite; Rinné osseous conduction 50 per cent, air 60 per cent of normal; low limit G₂ (24 v. d.); high limit with the Edelman Galton whistle 0.3 (normal 0.5.)

Patient's history follows: D. W.; aged 40, occupation, bartender, alleged moderate use of liquor; had a cold for two weeks, for which he took quinin, quantity unknown; for a week deafness and noise like rushing water in ears. The quinin was withdrawn; after three weeks the tinnitus was relieved, hearing for whisper 18 feet. This patient disappeared from observation.

Many of the infectious diseases are known to have caused nerve-deafness without suppurative lesion. Unfortunately, most of these, at least at the time they usually come under the observation of the otologist, are irreparable. Undoubtedly earlier recognition will lead to better results. Of these diseases mumps as a cause of deafness is perhaps the best known. Typhoid fever, influenza, diphtheria, scarlet fever, measles, syphilis, rheumatism and herpes zoster are also offenders. Siebenmann⁴⁶ and Witmaack⁴⁷ describe acoustic neuritis due to the toxemia of tuberculosis, causing a permanent lesion. It is to be remembered that a nerve-deafness during convalescence from a protracted disease like typhoid⁴⁸⁻⁴⁹ may be due simply to functional depression or *asthenia nervi acustici*. Of the non-infectious diseases, if I may call them all so, carcinoma,⁵⁰ diabetes,⁵¹ leukemia, pernicious anemia, and purpura⁴ should be kept in mind in the etiology of acoustic neuritis.

The so-called rheumatic form of acoustic neuritis, the real etiology of which is in doubt except in the cases associated with polyarthrititis, is described as coming on rapidly with deafness, tinnitus, vertigo, nausea and ataxia (most of these cases were described before accurate observation of nystagmus was practiced) either with or without associated lesions of the facial and other cranial nerves, and with or without herpes. Under this head Hamerschlag reported a series of cases in 1901.⁵² Bing⁵³ reported a case without static disturbances or associated lesions of other cranial nerves. Some of these recovered and some did not. Bezold⁵⁴ in his admirable work, under the general head polynuritis, describes as group D, those presenting the same cochlear and static disturb-

ances, but makes no mention of associated paralyses or herpes, and makes a favorable prognosis. Similar cases are reported by Zyto-wich,⁵⁵ who includes modern methods of testing the vestibular reaction, and finds it sometimes increased, sometimes diminished. Whether these cases are rheumatic, or belong to the group described by Ramsay⁵⁶ as "Acute poliomyelitis posterior of the geniculate, auditory, glosso-pharyngeal and pneumogastric ganglia, that is to say herpes zoster, is a question. As neither the vestibular nor cochlear ganglia govern any nerve terminals in the skin or mucous membrane, affections of these ganglia alone would not cause herpes.

As an example of nerve-deafness (in this case combined with middle-ear trouble), resulting from an infectious disease and responding promptly to treatment, permit me to report the following case of syphilis:

L. H., female, aged 22; deafness and tinnitus of three weeks' duration; tinnitus like bells ringing. Examination, August 10, 1910: Papules in right and left canals. Speech: right, 8 inches; left, 5 inches. Schwabach: A-fork, 70 seconds; (normal, 28.) Weber, left. Rinné right. +10/21; left, indefinite 0/15; (normal, 15/35.) Low limit: left, F# 90 v. d.; right, F# 90 v. d. High limit: left, 5.00; right, 4.00; normal, 0.5.

Initial lesion present on genitals; macular eruption; mucous patches on tonsils. Treatment: intra-muscular injections of mercury.

October 8, 1910: Whisper heard at 6 feet. January 11, 1911: Whisper at 22 feet; c¹ normal; low limit C₂ (24 v. d.). This case is especially interesting because of the early development of an internal ear affection, coincident with the primary lesion and secondary eruption, because of the condition affecting the external, middle and internal ear, and on account of the rapid response to antisiphilic medication, particularly of the internal ear condition. There was no complaint of vertigo or ataxia, and we found no evidence of hysteria.

Politzer has said that the auditory is the most vulnerable of all the cranial nerves. Of 144 cases of deafness due to non-suppurative causes observed on Dr. Norval Pierce's service at the Illinois Charitable Eye and Ear Infirmary, and in patients of sufficient age and intelligence to permit accurate tuning-fork tests, fifty-seven were cases of nerve-degeneration, and thirty-five others showed nerve-deafness as an element. This appears to be a larger proportion than one ordinarily sees in private practice, and, of course,

does not include the cases of tubal catarrh so common in children with adenoid hypertrophy, but it shows the importance of more attention to this phase of deafness.

By careful attention to the cause of nerve-deafness, by systematic and accurate functional tests leading to earlier recognition of this condition, I believe we can learn to distinguish the curable from the non-curable forms, and particularly may we learn to avoid those errors of treatment that arise from inaccurate diagnoses.

In conclusion, I wish to express my thanks to Dr. Norval Pierce who first called my attention to recoverable forms of nerve-deafness and to whom I am further indebted for his teaching and advice, as well as for the opportunity to study patients at his clinic, to Dr. Robert Sonnenschein for assistance in tuning-fork tests, and to Dr. Maximilian Meinhardt, for the reference of interesting cases.

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22 East Washington Street.

Anakinetic Re-education of the Ear by the Electrophonic Method.

O. RAOULT, *Arch. intern. de Laryngol.*, March-June, 1912, p. 413; and *Rev. med. de l'Est*, April 15-May 1, 1912.

Since March, 1911, 71 cases were treated. The case-histories of 20 patients are given upon whom this method was used. In cases of otosclerosis with nasal lesions, the latter should be attended to before the re-education is begun. Cases still in the presclerotic stage are most benefited.

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Phlegmon of Left Orbit Following Injection of Paraffin in Right Nasal Fossa.

PISTRE, *Rev. hebdomadaire de Laryngol.*, June 1, 1912, p. 641.

Girl of 28 years complaining of ozena, received injection of paraffin into left nasal fossa and was greatly improved. Month later injection into right side, followed in one week by phlegmon of left orbit, severe coryza, intense headache, vomiting, fever, lancinating pains and complete ptosis in left eye; vision undisturbed; orbit explored but no pus found; drained. Recovery in three weeks. Author feels that infection possibly took place through blood channels.

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