MÉNIÈRE'S DISEASE.

A Review of the Literature with reference to the present status of Pathology and Treatment, including a report on a series of Cases treated by Alcohol Injection of the Labyrinth.

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C O N T E N T S.

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Historical Review.

Although the symptoms of the disease, which bears his name, were described by Ménière in 1861, they were known for approximately forty years previous to his report, in which reference is made to the work of Itard (1825), who included cases of hysteria, epilepsy and other conditions with those of aural vertigo, classifying all of them as "cerebral apoplexy".

Ménière first suspected the aural origin of the symptoms, his belief in the site of the disease being based on the experiments of extirpation of the semicircular canals conducted by Flourens in 1842. The report of Ménière's classical case antedated the acceptance of the theory of organismal infection as the cause of disease, and in the light of present knowledge it is evident that he linked his clinical findings with the pathological findings of a case of acute labyrinthitis.

His reports give a clear picture of the attack of vertigo of sudden onset, without preceding illness, the recurrence of the attacks, the extreme discomfort of the patient during an attack and increased dizziness on movement. He describes the periods of spontaneous remission, the relationship of tinnitus to the attacks and remissions, and the onset of deafness which is progressive.

Nine cases of the typical symptom complex were subsequently reported by Ménière. The helplessness of all forms of treatment was admitted by Charcot (1874), who demonstrated many cases, observing that when deafness became complete the disease stopped spontaneously, an observation which has not been confirmed by later authors.

Terminology.

The literature on Ménière's disease contains many apparently divergent theories interspersed with experimental attempts to establish facts. Terminology has given rise to much confusion. The terms "Ménière's syndrome" and "Ménière's symptom complex" have been used to include labyrinthine disturbances of vascular, toxic or other origin as well as the idiopathic type of vertigo which has been shown to be a clinical entity now generally designated Ménière's disease or, more recently, Hydrops labyrinthi. Although the causation and pathogenesis remain obscure, the disease has been shown fairly conclusively to be based on hydrops of the labyrinth.

Most of the early knowledge of the disease was derived from empirical attempts to treat the symptoms and many of the papers studied refer clearly to a disorder of toxic or vascular origin. Thus, Dandy (1937), in a paper on the pathological changes in Ménière's disease, is of the opinion that only lesions of the sensory root of the acoustic nerve (vestibular division) can

cause the disease. His report is obviously not concerned with Ménière's symptom complex of idiopathic aetiology, but with structural pathological changes in the cerebello-pontine angle causing attacks in which the symptoms are not entirely of Ménière character.

Aetiology.

Many early observers suggested chronic catarrh of the middle ear as a cause. Woakes (1883) advanced the theory of increased intra-labyrinthine tension. In 1897 Cheatle speculated on increased tension of endolymph or perilymph due to excessive secretion or obstruction to outflow. This theory was also adopted by Ferreri, Aboulker and Portmann. Escat in 1906 suggested vascular spasm, elaborated by Lermoyez (1919) stating that angiospasm of the cochlear or vestibular branches of the auditory artery could account for some instances of the symptom complex.

Duke (1923) reported cases attributable to allergy. Proetz (1931) also considers allergy of the middle or inner ear an aetiological factor. Williams (1945) states that the symptom complex may be regarded as an allergy belonging to the larger syndrome of physical or intrinsic allergy as it involves the head

The first biochemical approach to the problem was made by Mygind and Dederding (1924 et seq.) adopting the premise that the condition was one of glaucoma or hydrops of the labyrinth. As a result of their experiments on water metabolism in patients

suffering from Mènière's disease, they came to the conclusion that the aetiology and pathology is based on an extra-cellular oedema resulting from a deficient vitality, and an increased permeability of the cells of the organ of Corti, the deficient vitality being due to a deficient capillary function in a "predisposed" labyrinth. Little is known of the extra-cellular oedema postulated by the authors, who quote the experiments of Falbe-Hansen in which oedema of the organ of Corti was produced in guinea pigs by administering large intraperitoneal injections of distilled water.

Furstenberg, Lathrop and Lashmet in 1934, accepting the conclusions of Mygind and Dederding with reservations, pointed out that oedema is not water alone but solutions of electrolytes, namely sodium salts, which are lost with water in diuresis since they are complementary in maintaining isotonicity. Therefore relief of symptoms may be due to loss of sodium salts. In their experiments a careful water exchange investigation was carried out on a patient with a history of typical Mènière attacks for ten years. Their results suggested that the symptoms of Mènière's disease are due to retention of sodium by the body and that the tissues involved have either an increased avidity for or unusual sensitivity to sodium.

Talbot and Brown investigated the blood of forty-eight patients before and after treatment by the Furstenberg regime.

In all except four cases, where serum potassium was raised and serum sodium lowered, the concentrations of Sodium Potassium, Calcium, Chlorides, Carbon Dioxide, Phosphate, Protein and Non-protein Nitrogen were found to be normal. If the symptoms are produced by gross retention of water or sodium chloride, there should be variations in the levels of sodium, water and protein content. Such a change, namely an increase in the sodium and total fixed base concentration with an unchanged water and protein content, was produced in four patients by Talbot and Brown without inducing an acute vertiginous attack.

Since, in the Furstenberg diet a relative increase in potassium is unavoidable, Talbot and Brown tried a normal diet with extra potassium, the diuretic effect of potassium causing increased excretion of sodium and water, thus reproducing the condition obtained by a low sodium diet and administration of ammonium chloride.

These metabolic theories are therefore not supported by examination of the blood and it is difficult to believe that a disturbance of general metabolism can produce trouble limited to the ear.

Pathology.

A significant advance in establishing the pathology of Ménière's disease was made in 1938 when Hallpike and Cairns reported their findings in two cases in which death occurred following operation for section of the auditory nerve. Since

their report, twelve more cases have been described. Of the fourteen cases published, nine showed unilateral and five bilateral involvement, thus contradicting Rollins' statement that the disease is nearly always unilateral.

The essential feature in the pathology of these cases is dilatation of the endolymphatic system. In all of them the cochlear duct was dilated, the saccule in most of them and the utricle in many of them, but in none were the semicircular canals dilated. Lindsay reported cases with a herniation of the saccule into the inner extremity of the lateral semicircular canal; in others the utricle protruded into the perilymphatic space of one or several canals. The organ of Corti, the maculae and cristae showed post-mortem changes but no definite pathological changes. In some cases degenerative changes were present in the cells of the spiral ganglion in the basal coil. The cases of Hallpike and Cairns showed gross dilatation of the scala media with displacement of Reissner's membrane on to the wall of the scala vestibuli with obliteration of its perilymph space. At the helicotrema, Reissner's membrane was displaced into the apex of the scala tympani. In Lindsay's case (1944) and in the case reported by Hallpike and Wright, there was a lesser degree of obliteration of the scala vestibuli, which suggests that its complete obliteration is not an essential factor in disturbance of function.

If dilatation of the endolymph system is accepted as the essential change, two cases reported before 1938 belong to this group.

In Brunner's case, which showed Paget's disease of both temporal bones, there was marked dilatation of the left inner ear with herniation and dilatation of the saccule and medial part of the utricle. Corti's organ was degenerated and the stria vascularis atrophic. Atrophy of the spiral ganglion and cochlear fibres was most marked at the base of the cochlea. The cochlear aqueduct was filled with reticulated tissue which contained concretions. The endolymphatic duct was patent. On the right side dilatation was confined to the medial part of the utricle. The cochlear aqueduct was patent, otherwise changes were similar to those on the left.

Wittmaack's case showed a neuroma deriving from the ramifications of the cochlear nerve in the basal turn. It had grown into the scala tympani andthen into the lumen of the cochlear duct, which was dilated. Corti's organ was in a state of "post-hydropic degeneration". The cochlear aqueduct contained concretions in the narrowest part of its lumen.

The pathological changes in these cases were believed to be a serous labyrinthitis, but the dilatation of the endolymph system first discovered by Hallpike and Cairns was greater in degree than that usually found in serous labyrinthitis.

Other similar reports have been made by Berggren, Alexander, Manasse and Videbach, but either details are insufficient or the clinical picture did not correspond with that of the typical Ménière disease.

In the three temporal bones described by Hallpike and Cairns the connective tissue surrounding the endolymphatic sac, described by Guild as the normal absorption area of the endolymph, but also lost by diffusion through Reissner's membrane into the scala vestibuli, was absent. The same condition was present in the unaffected ear of the first case and in two out of thirteen normal cases examined. It is suggested that this condition may be a normal variation yet a predisposing factor in endolymph system dilatation. The absence of perisaccular connective tissue or presence of perisaccular fibrous tissue cannot, on the available evidence, be regarded as proof of resorptive mechanism failure.

The primary condition may be an increased production of endolymph itself or, as suggested by Crowe, some alteration in physico-chemical constitution. It seems possible that the continued secretion of endolymph of abnormally high ionic concentration might lead to dilatation by osmotic attraction of water molecules through the membranous walls from the surrounding perilymph.

Pathogenesis.

Despite the unanimity which has been attained in descriptions

of the histo-pathology, many varied hypotheses have been presented to correlate this picture with the clinical condition.

Hallpike and Cairns believe that there is a chronic condition of lowered function of the affected labyrinth due to increased endolymphatic pressure and resultant anoxaemia of its end organs; that the sudden attacks are due to "rapidly initiated bouts of asphyxia of the labyrinthine end organs brought about by extremely rapid rises of fluid pressure in response to relatively very small volume increases in the endolymph".

To accept this theory it is necessary to believe that the pressure is sufficient to reduce vascular supply but insufficient to interfere with the secretory activity of the stria vascularis, or that the end organs and stria vascularis vary in sensitivity to a certain level of anoxaemia. On the other hand it has been pointed out that vascular supply of the stria vascularis is supplied anatomically at shorter range and higher pressure than to the maculae and cristae, where there is a rich capillary network closely related to overlying epithelium. Thus a greater degree of anoxaemia may exist in the end organs whilst the secretory activity of the stria vascularis is unimpaired.

Lindsay opposes this theory on the grounds that the relatively small increase of pressure possible in the endolymph system can only exceed that of the cerebro-spinal fluid by the

minute amount necessary to overcome elasticity of the limiting membranes sufficiently to maintain equilibrium. Such pressures must be infinitesimal compared with pressure changes to which labyrinthine fluids are known to be exposed without loss of function. He explains the vertiginous attacks as being caused by the herniation of the saccule or utricle into the semicircular canals with distortion of the walls of the ampullae. The distortion interferes with the function of the cupula and underlying hair cells, thus causing the attacks of vertigo and depression of caloric excitability.

It is believed by many workers that in the initial stages the involved labyrinth is hyperexcitable, but as the disease progresses it gradually becomes hypoexcitable. Hallpike and Cairns believe in a chronic state of hypo-excitability of the involved labyrinth and particularly during the attacks, the resulting vestibular symptoms being due to an imbalance as a result of the predominance of the healthy or less involved labyrinth.

Using hot and cold caloric tests, Cawthorne, Fitzgerald and Hallpike endeavour to explain the abnormal vestibular reactions which they state are present in 90% of cases. The most frequent finding is a lowered vestibular response for hot and cold stimuli alike, leading to a preponderance of the normal labyrinth. It is explained on the basis of an impaired function of the affected lateral semi-circular canal. The second type of response is

characterised by the fact that nystagmus to one side, no matter from which ear it is induced, shows longer duration than the nystagmus induced by the same stimulus to the opposite side and is explained by a paresis of the utricle on the affected side. The third group constitutes a combination of one-sided preponderance and directional preponderance of the induced nystagmus.

In attempting to explain loss of cochlear function it has been concluded that tissue respiration of the organ of Corti may be conducted via the endolymph but in the early stages of the disease increased endolymph pressure alone would not lead to anoxaemia of the organ. There may,however, be an increased impedance in the vibratory mechanism. Generalised stretching of the basilar membrane could be expected to give rise to changes in the normal frequency of its components. Lindsay has drawn attention to the distortion of the normal column of perilymph from oval window to round window by the dilated saccule and cochlear duct at the expense of the perilymphatic cistern and scala vestibuli. Therefore it seems reasonable to suppose that fluctuation in hearing may be related to temporary variations in the degree of distortion of the membranous system. This seems a more reasonable explanation than that of Rollins, who ascribes the disturbance in hearing to the fact that the dilated saccule is pushed towards the stapes footplate and annular ligament, interfering with its motility.

In many cases tinnitus is evidently caused by irritative changes in the nerve endings due to increased endolymphatic pressure, but in a proportion of cases tinnitus persists after section of the acoustic nerve and is attributed to unknown changes in the central cochlear neurones.

The therapeutic response to certain drugs has given rise to the belief that the attacks are the result of circulatory disturbances in the labyrinthine vessels which cause an overproduction of endolymph with increased pressure.

Atkinson assumes that attacks in the younger age group may be due to vascular spasm produced by some unknown local vascular fault, when discussing the aetiology of the vasoconstrictor or histamine insensitive group which he has described. The division of the disease into an allergic or primary vasodilator group and a larger vasoconstrictor group on the basis of the histamine sensitivity test has not received general acceptance. Brunner finds it difficult to believe that two antagonistic mechanisms could produce identical symptoms and Williams has found skin tests of little value either for diagnostic purposes or an index to therapy. Horton likewise does not think it possible to distinguish between histamine sensitive and insensitive cases.

Lindsay refutes the theory that Ménière's disease may be on an allergic basis since allergic manifestations are rare in Ménière's disease and attacks of vertigo are not usual in allergy,

but is of the opinion that vasomotor instability may be the basis for attacks in most cases of the idiopathic Ménière syndrome.

On the other hand, Williams, in a recent paper on the "Intrinsic Allergy Syndrome", draws attention to the findings of Hallpike and Cairns, Hallpike and Wright, Lindsay and others, where the pathological picture is one of a non-inflammatory distension of the endolymphatic system. Since it is clear from the clinical picture that the pressures within the labyrinth must fluctuate, it is suggested that this fluctuating oedema must have an allergic aetiology.

Duke (1923) pointed out that Ménière's disease might be due to an antigen-antibody type of allergy and cited two cases where attacks were alleviated by withdrawal of the causative foods. Not more than twenty such cases have been recorded. Therefore allergy of this type can only be considered in a relatively small group of cases.

The term intrinsic or physical allergy, also introduced by Duke, denotes the reaction, which is not an immunological one similar to the antigen-antibody reaction, in response to physical stimulus such as heat, light, cold, trauma and emotional disturbance. According to Williams such a stimulus acting through the autonomic nervous system affects certain cells, which have an inherited abnormality, resulting in their disruption with release of histamine and a change in the permeability of the cell

membranes. Such a disorganisation is an injury response of the cell, giving rise to vasodilatation and pain. This theory is supported by considerable evidence, which shows that many phenomena believed to be allergic are produced by cellular injury with release of histamine, which is a constituent of most tissues. When released into the tissue spaces it acts on the arterioles, causing constriction and capillary dilatation. With increased capillary permeability localised oedema occurs.

Summary.

In summing up the present status of knowledge concerning the pathology and pathogenesis of Ménière's disease it can be stated: (1) there is acceptance of the view that there is a state of Hydrolabyrinth; (2) the source and chemical nature of the excessive fluid is unknown; (3) the disease may resolve without injury to the internal ear unless attacks are prolonged or severe, when permanent changes occur, namely, a fixed dilatation of a varying part of the inferior portion of the internal ear and an acute or gradual destruction of the sensory cells in the internal ear; (4) the histo-pathology favours the vascular theories of aetiology whether due to hypersecretion of endolymph or increased capillary permeability; (5) absence of morphological changes cannot be regarded as proof of normal function.

Treatment.

Where preservation of the function of the inner ear as well as prevention of vertiginous attacks is aimed at, treatment is

still in the experimental stage, though much can be done to reduce the patient's burden.

In attempting to evaluate therapeutic measures, the tendency to spontaneous remissions must not be forgotten. Thus in examining a series of cases the duration of treatment and observation is of primary importance. The discomfort caused by tinnitus does not appear to be sufficiently stressed in the assessment of results.

Treatment is divided into medical or surgical. It is generally recognised that the former method should be given a trial in most cases. Factors to be taken into consideration are the patient's occupation, intelligence and social status. If the occupation makes attacks of dizziness dangerous, or if economic circumstances do not permit loss of time from work, or prolonged medical treatment, operation should be advised.

Early medical treatment was obviously a groping in the darkness, being chiefly sedative in character. A wide variety of drugs, ranging from quinine (Charcot), strychnine, pilocarpine, atropine, adrenaline, parathyroid extract and calcium (Thornval), to ovarian preparations, have been employed. In addition, many types of physio-therapy, such as pine-needle baths, sulphur baths, carbonic acid baths, ultra-short-wave therapy and massage are recommended.

Dehydration Therapy.

The dehydration treatment of Mygind and Dederding yielded a series of results which were an improvement on previous methods.

In outline the treatment consists of a restricted and balanced fluid intake, with adequate caloric requirements, vitamins and calcium. The peripheral circulation and functions of kidneys and lungs are assisted by graduated exercises and massage. The patient's habits are carefully supervised, e.g. alcohol, coffee, tobacco are restricted. In addition, several cases have been treated by routine Eustachian inflation.

Their series of 157 cases, observed over a period of three years showed a relief of vertigo in 43%, a decrease in severity of vertigo in 52% and relief of deafness in 17.4% of cases. No assessment of results on relief of tinnitus is given.

Furstenberg Régime.

This method of therapy has been largely replaced by the dietary régime of Furstenberg, Lashmet and Lathrop, following their experiments with a low sodium intake. Articles of diet are divided into three groups - those which may be taken daily, those to be avoided, and those which may be taken twice weekly. Water is unrestricted but not excessive. Sodium chloride is replaced by ammonium chloride in a dosage of three grammes with each meal for three days followed by an interval of two days. The régime is continued for six weeks and if the patient is asymptomatic the ammonium chloride is stopped; otherwise it is continued for a further six weeks.

Review of their series of cases with a period of observation up to seven years shows relief of vertigo in 57%, a decrease in

severity of vertigo in 26% and relief of deafness in 9%. Tinnitus was relieved in 51% of cases.

Although the authors advised hospitalisation for an average period of thirty days., it has been found practicable to prescribe the Furstenberg dietary in many cases without such supervision.

Following the work of Talbot and Brown, the Furstenberg régime was continued by Walsh and Adson, substituting potassium nitrate for ammonium chloride in a dosage of nine grammes daily, taken as enteric coated pills of half a gramme each after meals. They found that more patients with unilateral deafness improved under the Furstenberg régime than patients with bilateral deafness. In a series of one hundred and fifty two cases observed for four years, 34% obtained complete relief of vertigo, in 28% vertigo was diminished and in a similar percentage of cases deafness was relieved.

The preceding forms of therapy have attacked the problem from the metabolic angle, but more recent methods involve the use of drugs affecting vasomotor tone of peripheral vessels and hence the degree of capillary permeability.

Histamine Therapy.

Following the successful use of histamine in the treatment of erythromelalgia of the head, Shelden and Horton introduced similar therapy in treating fifteen patients suffering from Ménière's disease. In November 1939 their first patient was treated by the intravenous administration of 1.9 mgms. of

histamine phosphate dissolved in 250 cc.s of physiological saline, the solution being administered over a period of one and a half hours. This patient was immediately relieved of dizziness and unsteadiness. They concluded that histamine is of undoubted value in the treatment of selected cases of Ménière's disease, but in many of the severer cases it is of no value. The intravenous use of histamine is of value in an acute attack, but during a remission treatment is started by the subcutaneous route. Using ampoules of histamine phosphate containing one milligram per cc., treatment is started by diluting this concentrated solution with sterile normal saline, 1:10, and giving subcutaneous injections twice daily, commencing with 0.1 cc. On the second day, 0.15 cc. is given night and morning and the dose increased by 0.1 cc. daily until the 0.9 cc. dose is reached. The action is maintained by giving 0.3-0.4 cc. of the concentrated vial once weekly for an indefinite period.

Lillie, Horton and Thornell have reported a series of twenty-five patients in which improvement in hearing was noted in twelve cases, improvement in tinnitus immediately following treatment was noted in fourteen and relief of vertigo in twenty-one cases.

Nicotinic Acid Therapy.

Treatment by Nicotinic acid was introduced in 1940 by Harris and Moore, following the observation that many pellagrins have rotational nystagmus. This drug has a powerful vaso-dilator

effect on the peripheral circulation and can be given over long periods without harm or without producing tolerance. It has been extensively used by Atkinson in the vaso-constrictor or histamine insensitive group. In an acute attack the drug may be administered intravenously after an intra-muscular dose of thirty mgms. to test the patient's reaction. The course of therapy may be started with 25 mgms., repeated daily or every second day until six to eight doses have been given, increasing by 5 mgms. each day to a tolerance dose of 50 mgms. but sometimes as much as 75 mgms. After a few days, oral administration is started, giving 50 mgms. twice daily on days of injection and thrice daily on other days.

Following the intravenous course, intra-muscular injection of the maximum dose is started daily for one to three months, then if symptoms are controlled, they are gradually diminished to once weekly and eventually discontinued. At the same time oral treatment of 100 to 150 mgms. is given daily.

Atkinson has reported a series of one hundred and ten cases with a "follow up" ranging from six months to three years. Relief of vertigo was obtained in 38% of cases, improvement of vertigo in 46%, of deafness in 23% and of tinnitus in 52% of cases.

Atkinson also reports success in a number of cases in which vaso-constriction was assumed to be the cause of the attacks, but

Williams found that the response to treatment of all types of the physical allergy syndrome was equally good with histamine or nicotinic acid.

From this summary of medical treatment and results one can say that medical measures are of proved value and should be tried before operation is contemplated.

Surgery.

Surgical treatment was classified by Ramadier (1933) into conservative and radical groups. The former group included decompression measures such as lumbar puncture, trephination of the cerebellar fossa and puncture of the endolymphatic duct, whilst radical measures comprised destructive trephination of the inner ear and section of the auditory nerve.

Removal of the ossicles was an early form of treatment. Burnett (1899) removed the incus and Crockett (1903) removed the stapes in two cases with relief of vertigo.

Frazier (1912), following a suggestion by Mills (1908), divided the acoustic nerve intracranially in one patient, but dizziness was not relieved. The diagnosis in this case appears to be doubtful. This operation, variously attributed to Charcot (1874) and Ballance (1894) has been developed by Dandy, who has performed four hundred and one operations, with one death due to meningitis. In 1937 he advocated partial section of the acoustic nerve, where there is reason to save hearing. Usually three fourths of the anterior part of the acoustic nerve is sectioned

and even half to four-fifths of the cochlear branch may be divided with practically no loss in hearing. Dandy is of the opinion that medical treatment is of little value. He reports complete disappearance of tinnitus in 50% of his cases.

Subtotal section of the acoustic nerve is regarded as the operation of choice where substantial hearing remains or where there is early involvement of the other inner ear. Walsh and Adson report twenty cases in which total unilateral section was done in thirteen cases and subtotal section in seven. In the first group nine obtained complete relief of vertigo and tinnitus disappeared in two cases. Of the second group, four were completely relieved, three greatly relieved and hearing maintained in these three cases. Tinnitus disappeared in two cases. Fifteen stated that they were able to resume their regular work.

Altmann believes that the Portmann operation for drainage of the saccus endolymphaticus is a rational approach to the problem, but his results in three cases and Cawthorne's in two cases have proved disappointing. Musgrave Woodman reports eleven cases of which eight were completely cured of vertigo. Tinnitus ceased in two cases and hearing was almost restored to normal in one case. The period of observation is not recorded.

When useful hearing is lost and it is desired to relieve tinnitus to a maximal extent, total section of the acoustic nerve

has been advised, but the less extensive procedure of alcohol injection of the labyrinth is regarded as sufficient in these cases. This operation has been employed by Mollison, through an opening in the lateral semicircular canal; by Wright and Peacock through the oval window and through the promontory into the basal cochlear coil by Berggren.

Wright (1944) reports the results of treatment in sixty cases. Vertigo was improved in 55 and not improved in five patients. Tinnitus was improved in 33, not improved in 16 and not recorded in nine cases. Complications were two cases of facial palsy and one case of meningitis.

Original Report.

Alcohol injection of the labyrinth as a radical measure has been employed in the Royal Infirmary of Edinburgh. The operative procedure adopted follows that of the Schwartze mastoidectomy with good exposure of the antrum and enlargement of the aditus until the lateral semicircular canal is clearly visible. The canal is opened by electric burr just on to the roof of the vestibule. The membranous canal is exposed and a few minims of endolymph withdrawn into a syringe through a fine needle, then three minims of absolute alcohol are injected. The opening is loosely filled with bone chips and the edges burred over. The wound is then closed with drainage.

Seventeen cases have been treated in this manner during the past three years, without complications. In a recent review of

these cases fifteen patients have been traced and the following results recorded. Vertigo has been completely relieved in nine cases. In six cases the acute attacks of vertigo have been relieved but there is dizziness on rapid movements, involving a change of direction, or on bending forward. Tinnitus has been completely relieved in six cases, diminished in two cases, unrelieved in five cases, and absent before and after operation in two cases. Twelve patients stated that they had been able to resume their regular employment.

Conclusion.

The introduction of chemotherapy, with consequent reduction in post-operative infection dangers, has stimulated interest in labyrinthine surgery, and it seems probable that operation on the labyrinth will replace intra-cranial section of the eighth nerve.

The final method of treatment, whether medical or surgical, depends on a greater knowledge of the physics and chemistry of the endolymphatic system, its vascular supply and response to stimuli. Though that method requires further observation and experiment for its evolution, much can now be done to relieve the distress of an individual suffering from a vertiginous attack or living in fear of an impending one.

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