the patient's word for it, as the temperature showed nothing, but there was no reason why he should have complained of more pain just at the time the drugs were altered.

CASE VII.—H. K., aged 68. This case, very similar to the above, was suffering from chronic rheumatism and sciatica. He was admitted into the Metropolitan Hospital on January 15th, 1895. There was a history of having suffered for nine years. Pains were now chiefly in left knee and hip. He also was put on salicylate, and improved considerably, but he also later on got bronchitis, and on March 20th was given the same ammonia mixture as in the other case. On February 23rd the note says: "Can get about better and pain is, comparatively speaking, gone." On March 20th he got some bronchitis. The salicylate was stopped, and mixt. senegæ cum ammonia ordered. On March 26th the pains were decidedly worse again, especially in the knee. He was ordered to have the salicylate mixture as well as the ammonia, and the pains soon got better.

I may explain that urate of ammonium is one of the more insoluble urates, and that ammonium raises the acidity of the urine and increases the hourly excretion of acid. It would therefore increase gouty or rheumatic pains when given alone, but would not interfere with the action of salicylates; on the contrary would aid it.

I could easily give a number of similar cases; indeed, a week never passes without my seeing one or more of them. Almost every time a uric acid headache is cured some joint pains are produced, and in gouty and rheumatic cases the well-known effects of changes of weather can almost always be translated into changes in the alkalinity of the blood, secondary to the effects of the weather on the excretion of the skin.

Thus a patient recently seen gives me the following history of an attack of gout. He lives in India, but occasionally comes home to England, and after a previous visit to England, where he had been "feeding up," he felt the heat very much in the Red Sea on the way out, and then in Bombay was exposed to comparatively cold March winds, and had a violent attack of gout. Can anything be more simply and easily explained than this history? We have (1) the ingestion of a lot of uric acid, (2) its solution in the blood (uricæmia) as the result of heat, and (3) its precipitation on the joints by the rising acidity produced by cold winds.⁹

Similarly in patients who suffer from mental depression, pains in the joints are often looked upon as the harbingers of better things; and in women, when menstruation with its depressing influences causes a rise in the alkalinity of the blood and an excess of uric acid in that and the urine, mental depression or headache are generally worse, while rheumatic pains are to some extent relieved.

It is not my object in this paper to lay any great stress upon the cases quoted above, which I have been able to collect in a few months, but I hope, by bringing them to the notice of clinical observers, to induce them to regard their cases of arthritis and endocarditis in the light of what is now known about the solubility of uric acid; and if I am successful in doing so, I feel sure that they will very soon see hundreds and hundreds of similar cases, which only the mist of an erroneous pathology has prevented their seeing before, and they will not only see, but will be able to prevent and control, these disease processes to an extent which was previously impossible.

Disease by the visitation of God, or the work of that Jupiter Tonans of modern pathology, the nervous system, will give place to disease the result of folly in diet; the straightforward and easily verified chemistry of uric acid will replace the hypothetical microbe, and gout and rheumatism will be regarded as mere modifications of one and the same disease—arthritis due to uric acid.

⁹ See BRITISH MEDICAL JOURNAL, 1895, previous reference.

THE MEDICAL PROFESSION IN HUNGARY.—According to a statistical report recently issued by the Hungarian Minister of the Interior, the total number of medical practitioners and lower grade Surgeons (*Wundärzte*) in Hungary in 1894 was 4,280, being an increase of 106 as compared with the previous year. The distribution of practitioners throughout the country is very unequal: thus there are counties where 20 per cent. of the population are without a doctor. The average proportion of medical practitioners to population in the towns in 1894 was about 11.1 in 10,000. In Buda-Pesth there was 1 doctor to every 545 inhabitants, the lowest proportion in any town being at Hód-Mező-Vasárhely, where it was 1 in 3,460. In rural districts there was on an average 1 medical man to every 4,992 inhabitants.

OBSERVATIONS ON THE PATHOLOGY OF ENLARGED OR HYPERTROPHIED PROSTATE.

By REGINALD HARRISON, F.R.C.S.,
Surgeon, St. Peter's Hospital.

IN advocating what I would speak of as the muscular theory in explanation of hypertrophy of the prostate I am not aware I ever denied that the prostate was dissociated from the genital function. I have referred to it as "a muscle containing a tolerably large proportion of glandular or secreting tissue embedded in it," and though failing to see evidence showing that it is engaged in elaborating the secretion of the testicles I adopt the view of Dr. Handfield Jones¹ expressed in the following words: Its function relative to the sexual act is "in supplying a vehicle which enables the fecundating fluid to act with greater certainty over a larger area whilst at the same time it supplies a muscular buttress against which the ejaculatory muscles of the urethra may advantageously act in the emission of the semen." It is therefore, I believe, to this extent a compound organ, and I am not aware that anyone has brought forward arguments contravening this limitation of duality.

I regard senile enlargement of the prostate as an example of a muscular hypertrophy analogous to other similar kinds of overgrowth, and arising out of the muscular functions in which the part is unceasingly engaged. It is of the first importance that its muscular action should be clearly established, for unless this can be done the whole of my argument necessarily falls to the ground. The difficulty that has hitherto arisen in fully recognising this explanation is in some measure due to the isolated manner in which the prostate has been studied.

It has been urged that though the prostate is in some measure involved in the process of micturition, the fact that it is used for this purpose half a dozen times or so in the twenty-four hours is insufficient to account for its overgrowth. This, I would emphasise, is merely the casual part the organ plays in what we generally regard as a voluntary act. Such a limited view as this implies is in a large measure due to the habit we have acquired of taking our ideas of the living organ from the condition it presents after death when removed from the body in a more or less contracted state, somewhat resembling a Spanish chestnut in shape. This is no more like the living prostate than the dead heart resembles the organ in full vigour and activity. The prostate assumes, I believe, no such appearance during life except on the rare occasions when the bladder is absolutely empty, but, on the contrary, the muscular fibres of which it is so largely composed are spread out like a funnel so as to furnish a contractile support for the bladder and its varying contents. This disposition is probably best appreciated by examination by the rectum when the patient is in the erect and semi-erect positions with the bladder empty as well as in various degrees of repletion. Though the existence of a prostate is not limited to man, I am not aware that in any animal it undergoes a change analogous to the senile enlargement observed in the human species. Hence, though enlargement of the prostate and the habit of maintaining the erect position may be mere coincidences, this fact cannot be allowed to pass unnoticed.

As the normal prostate is in this funnel-shaped manner spread out, so is it when it is enlarged. We are often surprised to find after death, or by a suprapubic incision, how much smaller the prostate turns out to be as compared with what it was when examined during its functional activity by the finger in the rectum. Under an anæsthetic an enlarged prostate usually ceases to oppose the introduction of an instrument into the bladder. Unless this continuous muscular action of the prostate in supporting the contents of the bladder in accordance with the degree required, as well as in minimising the effects of shock applied to this part of the body, are fully appreciated, it is impossible to understand how the functions of the viscus can be discharged without some other provision than that afforded by a mere fibrous floor.

It was not until I recognised the funnel-shaped manner in

¹ London Medical Gazette, vol. v, 1947.

which during life the prostate was disposed in contradistinction to the contracted mass presented after death that I could find an explanation for certain results following different lesions of the part, as for instance in the various perineal incisions made for the removal of stone by lithotomy. Why an incision into the prostate, radiating from the urethra passing through it, and not as a rule dividing more than one-third of the solid mass presented by the dead prostate, should be followed by absolute incontinence of urine for some days seemed difficult to understand when so much of the circumference remained intact. Such a result, however, at once became intelligible when we recognised that the incision, though limited, absolutely destroyed, until repair took place, the capability of a cone-shaped muscle to hold fluid.

The male bladder, in its mechanical arrangements, has little in common with that of the opposite sex. In the latter the process of micturition and the axis of urine pressure relative to the pelvic outlet are different, whilst there is an absence of provision for ejaculation of semen or even any direct connection with the genital function. In the female the manner in which the bladder is supported when distended has a resemblance to what is found in some quadrupeds.

Further, the surgery of the part furnishes evidence in various ways of the muscular power of the prostate. Thus openings may be made into the urethra in any part of its course as high up as what we term the apex of the prostate without incontinence of urine following. After lithotomy by the median operation patients often retain full control over the bladder during the whole period of their convalescence, in spite of the dilatation to which the prostate has been subjected by the introduction of the finger and the extraction of the stone. Directly, however, the knife impinges to any appreciable extent on the prostate, as in the lateral operation for stone, incontinence from that moment takes place; the patient has no command over his urine, he can neither collect nor expel it; and in this condition he remains until the healing process has made considerable advance. Some instances I have examined in which permanent incontinence of urine followed the operation of lateral lithotomy appeared to have been connected with the complete division of the prostatic circumference by too free an incision. I can hardly see how such a consequence as this could follow if the prostate during life presented the appearance of the contracted mass [we are accustomed to look at after death.

In cases of extroversion of the bladder, in which there is no receptacle for the urine, the prostate is only met with in a rudimentary form. In advanced life, so far as I have been able to ascertain, hypertrophy never occurs in these malformations, though sexual desires are often vigorous. I have recorded the case of a boy² in which incontinence of urine appeared to be associated with an arrest in the development of the prostate. This, however, may merely be a coincidence, as at this period of life this part only exists in a very rudimentary form. In an instance³ of removal of the prostate I performed for malignant disease, although the patient recovered completely from the operation, and lived over fourteen months afterwards, during the greater portion of which time he followed an active and laborious occupation, control over the bladder was lost. To provide against the incontinence the patient was fitted with a sort of truss, which, by exercising pressure on the urethra below the arch of the pubes, enabled him to prevent the involuntary escape of urine and go about his work. Before proceeding to point out the steps by which prostatic enlargement seems to me to be brought about, I would mention one or two points which are generally admitted in connection with the natural history of his growth. In the first place there can be no doubt that though a considerable proportion of elderly males develop it, only a minority suffer from any ill-effects on the urinary apparatus it may produce. If such a growth serves no useful purpose, it is difficult to understand how this can be, and why we should draw our conclusions as to the process being a morbid one from the lesser number of instances of it than the greater. It is a matter of common observation to find persons with largely hypertrophied prostates, and

yet showing no other structural defect either in the capacity of the bladder to contain or to expel fully the urine for which it acts as a reservoir. Nor with proper safeguards is it necessary that persons so situated should develop any prostatic trouble calculated to shorten their lives. Another point is also worthy of notice. The process of hypertrophy involves no structural substitution or the importation of tissue foreign to the part other than those degenerations, such as the fibrous, to which the human body is liable. Hence we are narrowed down to offering an explanation as to the purpose for which this excess of normal structure is called into existence.

In studying pathological lesions, more particularly in relation to function, instances can be found in the human body where defects may call into existence such compensatory changes as eventually themselves constitute disease. A very small lesion, for instance in the mechanism of the heart, if it happens at the right spot, is capable of producing a hypertrophy which, though first compensatory, by-and-by proves to be a source of disorder. Thus the prostate in the course of its growth, so as to form a buttress or support for the most dependent portion of the bladder, tends to project in directions in which the resistance is least, and to form, by the fibrous degeneration these portions undergo, those obstructing masses with which we are familiar. Nor, though the whole gland is eventually more or less involved in the hypertrophy, can we fail to observe that in these changes the posterior segment, where it exists, is usually primarily and principally involved.

I have already laid stress on the importance of not regarding the normal prostate merely in the light of an individual organ, but as forming a part of the genito-urinary system. This holds good with its pathology. In the study of instances of enlarged prostate in the *post-mortem* room it is impossible not to be struck by the coincident changes that are taking place in the adjacent parts. For some reason or other there is a concentration of hypertrophied tissue in the form of buttresses or supports about the perpendicular axis of urine pressure at the base of the viscus. This is seen in the development of the inter-ureteral bar, the growth of the prostate, the gradual approximation and consolidation of these two structures, and the restriction of the natural trigonal area.

The trigone or floor of the bladder, in addition to being a highly sensitive part, is peculiar in that it contains the minimum amount of muscular fibre as compared with the rest of the viscus; muscle in abundance may be found as low as a transverse line drawn between the openings of the ureters marking the superior boundary of the trigone, and below in the prostate, but between these two points the power of muscular contraction can hardly be said to exist. Assuming, as I have stated, that from any cause, such as the long retention of urine, habit, position of the body, or the debility connected with advancing years, the floor of the bladder sinks lower within the pelvis relatively to the prostate, so as to offer some difficulty in expelling the last portion of urine, the effect will be frequently repeated efforts in all the muscles immediately adjacent to a part of the bladder which by reason of its connections and structure has but little power of contracting. It is suggested that in this way quantity is substituted for quality, and that as age advances structural deterioration and incapacity are in a measure provided against by superabundant tissue.

I have said that although hypertrophy usually includes the entire gland, the posterior segment or that in relation with the rectum is principally involved. When the part which was originally described by Sir Everard Home⁴ as the third lobe, but subsequently shown by Sir Henry Thompson⁵ to have no independent or isolated existence, is imperfectly or not at all developed, as is sometimes the case, it is interesting to notice that hypertrophy of the inter-ureteral bar may be observed taking place independently, and thus provision is made by a buttress of this kind for the support of the posterior wall of the bladder.

An inability to empty the bladder and the discomfort, or rather the consciousness of an incompleteness, of the act of micturition, is a common symptom in connection with the

² *Surgical Disorders of the Urinary Organs*, fourth edition, 1893, Churchill.

³ *Ibid.*

⁴ *Philosophical Trans.*, 1808.

⁵ *Diseases of the Prostate*, 1886.

early form of prostatic enlargement. This is due, I believe, not to atony or paresis of the bladder in the ordinary acceptance of the term, but to a sinking or tendency to prolapse of the posterior wall.

Though it is difficult to demonstrate these changes after death by measurements or castings accurately representing the previous shape and relations of the part, there are signs existing during life which tend to corroborate this view. By the catheter and by examination through the rectum we are usually able to convince ourselves of this. That the bladder alters its position relative to the pelvic outlet during life there can, I think, be no doubt. In early adult existence it may be regarded as an abdominal rather than as a pelvic organ; as years advance it gradually sinks within the pelvis, whilst still later it will be found to have become further depressed within the pelvic area. In this way a prominence is sometimes given to the floor of the prostate, which is due not in the first instance to the development of more prostatic tissue, but to the subsidence of the posterior wall of the bladder. This mode of forming a prostatic bar may readily be imitated, and is, I believe, the initial lesion in the hypertrophic changes which follow.

The most important objections urged against my views as to the muscular origin of the hypertrophied prostate are based on the statement that this organ is essentially a genital gland, and that the muscular fibre it includes, both intrinsic and extrinsic, is for the most part occupied in this function. This view has been supported by Mr. Joseph Griffiths, of Cambridge, and is based upon a series of observations,⁶ both in human and in comparative anatomy, which are deserving of careful consideration. Though in no way taking exception to the histology of the parts investigated, I cannot accept all the conclusions arrived at by this author relative to the disposition and function of the prostate.

As I have already pointed out, I believe we have formed a wrong conception of the arrangement of the prostate during life, and that to this is due in a large measure the difficulty we have hitherto experienced in recognising the extent and limitations of its true functions. My views are much more in accordance with those of Professor Viner Ellis,⁷ who speaks of the prostate as "essentially a muscular body."

The fact, however, that the hypertrophied organ contains a considerable amount of gland tissue of an inferior quality, yielding a secretion which Mr. Griffiths refers to as "scanty, thin, and watery" as compared with the normal exudation, is in no way opposed to the views I have advanced in reference to the circumstances under which the conglomerate growth is called into existence. The degenerated character of the gland tissue is in keeping with a function which at this period of life is on the wane, and not in that state of activity which in some of the lower animals at certain periods is attended with a large and more than usually developed gland. There can be no analogy between the large prostate of a rutting animal and that of a septuagenarian male.

Still more recently it has been put forward, in consonance, I presume, with the genital view of the function of the prostate, that the operation of complete castration is likely to prove of service in connection particularly with the treatment of the more advanced forms of prostatic obstruction. In a paper of considerable interest Dr. J. W. White, of Philadelphia,⁸ summarises the somewhat scattered evidence that exists in favour of the view that in man removal of the testes is followed by atrophy of the normal prostate, and thus remarks on the suggestion as to its practical value as a means of treatment:

As to the possibility of employing castration as a therapeutic method in prostatic hypertrophy, I imagine that the final answer must be left to our patients. Of one thing I am convinced, however, that if we even reach a point in certainty of knowledge in this direction comparable to that already attained in regard to oophorectomy in relation to uterine fibroids, and can promise equivalent results, there will be no lack of cases willing to submit to an operation almost painless, with a low mortality, and followed by no such unpleasant conditions as accompany persistent fistulous tracts, either suprapubic or perineal, even although the operation carries with it the certainty of sacrificing whatever sexual power has survived the sufferings of such patients.

In a subsequent communication⁹ Dr. White reviews further evidence he adduces tending to show that shrinkage of the

enlarged prostate has followed castration. As a contribution to the discussion which these papers elicited on this subject I narrated the particulars of a case which incidentally came under my notice some years ago, where, with the same object in view, and under considerable pressure, I had divided the vasa deferentia of a man with a large and troublesome prostate. The proceeding and its effects were summed up in the following words:¹⁰ "This operation was readily done, first on one side and then on the other, with a tenotomy at a few days' interval, and my patient left me in the course of a short time, alleging that he had already derived benefit from it. Six or seven years afterwards, when I was in America, I ascertained that he was alive and well, but as I had no opportunity of testing the case I thought nothing further of it until reading Dr. White's interesting lecture."

From a recent paper by Dr. Ewing Mears¹¹ it seems not unlikely that interference with other constituents of the spermatic cord may be found to lead to atrophy in the prostate by first inducing changes in the nutrition of the testicle. Dr. Mears more particularly refers to subcutaneous ligation of the vessels of the cord for varicocele, in which he has noticed more or less atrophy of the testes following. The accidental division of a vas deferens under somewhat similar circumstances has also resulted in a like consequence. These are observations which I think other surgeons can corroborate.¹² I refer to these collateral points requiring further consideration and testing, but without in any way wishing to detract from the credit due to Dr. White in first bringing this matter forward.

Shrinkage of the prostate has followed other measures than castration. Some years ago I published¹³ the particulars of a case in which for this condition in its most advanced and distressing form I punctured the bladder from the perineum through the enlarged prostate, and retained the cannula in this position for six weeks. This process has since been described as tunnelling the prostate. In the instance referred to the patient not only entirely recovered his power of normal micturition, but on the removal of the cannula it was discovered that the prostate had undergone a diminution in size, in fact, it had almost returned to its natural shape. This patient died at the age of 90, eight years after the operation, without any recurrence of his ailment or the necessity for again using a catheter.

Mr. Teale,¹⁴ of Leeds, has recently added testimony as to the value of physiological rest and the importation of some amount of scar tissue into the prostate in bringing about its shrinkage. In former days, when lateral lithotomy was more generally practised than now, I have seen in several instances where it has been necessary to divide a large prostate with some freedom to permit of the withdrawal of the stone, atrophy of the gland follow, and prostatic symptoms cease. More recently I have observed the same effects after a prostatotomy for bladder drainage, and in instances where comparatively small portions of the prostate have been removed.

Assuming, on the evidence of some cases which have been recently recorded, that shrinkage of the prostate follows upon castration, I do not see that such a conclusion is either at variance with the views I have advanced relative to its pathology when enlarged, or proves it to be essentially—that is to say, exclusively—a genital gland. That it secretes in association with the genital act no one, I think, will deny. That some atrophy must follow the extinction of this function by removing the testicles is equally clear and logical.

My object in this paper is to show: (1) That the prostate in connection with its associated parts has an arrangement and muscular function which are not sufficiently recognised; (2) that its hypertrophy is to be regarded as a provision against structural dilapidations in adjacent parts, arising for the most part out of senile degenerations; (3) that these

¹⁰ *Ibid.*, September 23rd, 1893.

¹¹ *Philadelphia Academy of Surgery*, November 5th, 1894.

¹² Mr. Birkett, in referring to a patient of the late Mr. Hilton, observes (*Holmes's System of Surgery*, vol. ii, p. 740) that "in a case dissected by him he accidentally found one testis excessively atrophied and the vas deferens of the same gland ruptured and closed at both ends; the ends were at least 2 inches apart. The vesicula seminalis on the same side was smaller than that on the opposite."

¹³ *Surgical Disorders of the Urinary Organs*, fourth edition, 1893.

¹⁴ *Lancet*, October 29th, 1894.

⁶ *Journal of Anatomy and Physiology*, vols. xxiii, xxiv.
⁷ *Med.-Chir. Trans.*, vol. xxii.

⁸ *BRITISH MEDICAL JOURNAL*, September 9th, 1893.

⁹ *BRITISH MEDICAL JOURNAL*, June 23rd, 1894.

changes are mainly compensatory, whilst in others they are excessive and hurtful; (4) that in the latter respects it resembles other provisional hypertrophies.

GIDDINESS AND STAGGERING IN EAR DISEASE.

By THOMAS BARR, M.D. GLAS.,

Lecturer on Diseases of the Ear, Glasgow University; Aural Surgeon, Glasgow Western Infirmary; Surgeon, Glasgow Ear Hospital; Honorary Aurist, Glasgow Sick Children's Hospital.

MÉNIÈRE's classical paper on aural giddiness in the *Gazette Médicale de Paris* for February 9th, 1861, marked an epoch in the history of medicine. Founding his conclusions partly on the experiments of Flourens in 1828-42, but chiefly upon very carefully observed cases, he demonstrated that a sudden attack of giddiness, with nausea and vomiting, with noises in the ear and deafness, has frequently its pathological basis in the interior of the ear. The view previously held that such an attack meant a lesion in the brain involved serious mistakes in prognosis and treatment. Paralysis, imbecility, and death itself were the gloomy prospects held out to such a patient; while blood-letting, mercurials, and vesicants were the usual remedies employed. All this is now changed. In the light of Ménière's discovery and of subsequent investigations the patient who is the subject of such a seizure may now, in the majority of cases, be assured that the ear and not the brain is the seat and source of the mischief; that the danger to life is very slight; that the dread of mental disease may be dismissed, and that, by simple and rational treatment, the symptoms will partially, if not entirely, disappear. It must be admitted, therefore, that the knowledge of the connection of these symptoms with the organ of hearing has been a great gain to medical science and fraught with valuable practical results. How desirable it is that every medical practitioner should be fully alive to the far-reaching influences of Ménière's discovery.

FREQUENCY OF GIDDINESS IN EAR DISEASE.

Since Ménière's discovery the profession has become gradually impressed with the importance of giddiness as a symptom of ear disease, and most practitioners now think of the ear when a case of giddiness presents itself. As a sign of ear disease it is by no means a rare phenomenon, occurring in all degrees of intensity from that of a slight and temporary lightness in the head to so violent a disturbance of the equilibrium as necessitates the lying posture. From my own experience, gained both in private and hospital practice, I am prepared to say that in a marked form it is far from being rare, while in a comparatively mild degree it is a very common concomitant of ear disease. At the Glasgow Ear Hospital a scheme is in use in which the existence of giddiness is included among the questions put to every patient of sufficient age, and the answer recorded. Examination of the scheme shows that in 1,276 consecutive cases, occurring in hospital practice, there were 73 cases, or 5½ per cent., of marked giddiness; while in 237, or 18 per cent., there was slighter or occasional giddiness. In 1,025 consecutive cases, occurring in private practice, there were 63 cases, or 6 per cent., in which giddiness was a distinct and indubitable feature. In the case of hospital patients we cannot, of course, vouch for the accuracy of all the replies. Excluding slight and occasional giddiness observed in hospital practice from our calculation, these being possibly in many instances connected purely with gastric or hepatic disturbance, the proportion in the severer forms is pretty much the same in hospital as in private practice.

Considering the importance and frequency of giddiness as a symptom of ear disease, it is remarkable that it seems to have eluded such able observers as Wilde and Toynbee. Neither in Wilde's volume on *Aural Surgery*, published in 1853, nor in Toynbee's treatise on *Diseases of the Ear*, published in 1860, do we find any definite reference to giddiness as a symptom of ear disease. In the supplement to the 1868 edition of Toynbee's work, however, written by James Hinton, there is reference to the symptoms of Ménière.

VARIOUS FORMS OF GIDDINESS DUE TO EAR DISEASE.

Ménière's discovery naturally stimulated much investigation, with the result, not only of still more widely connecting the symptom of giddiness with disease in the ear, but also at the same time of modifying some of his views. While he believed that these symptoms had always as their basis a lesion, usually hæmorrhagic, in the labyrinth or semicircular canals, further investigation and experience have modified and enlarged this view, and it is now well known that giddiness may be associated with almost any form of ear disease. In the light of the fuller experience of recent years aural giddiness may now be conveniently described as manifesting itself in four distinct forms.

I. The typical form, or true Ménière's disease (sometimes termed apoplectic deafness), due to the presence of exudation in the labyrinth. In a person with either previously normal hearing, or who may have suffered from a chronic affection of the middle ear, the four great symptoms—namely, sickness and vomiting, giddiness and staggering, subjective sounds in the ear and deafness—occur as a sudden seizure. In this class entire disappearance of all the symptoms is rare.

II. This form of aural giddiness is due to pressure upon the walls of the middle ear of air, liquid, or inflammatory products. Here the symptoms are somewhat less severe in character, the sickness and vomiting being occasionally absent. The vertigo is usually preceded by other symptoms of auditory disturbance. Disappearance of the symptoms, however, is usually effected by suitable treatment.

III. The giddiness is in this class due to pressure upon the walls of the external auditory canal or outer surface of the tympanic membrane, especially by ceruminous masses. Here, also, the giddiness is usually preceded by disturbance of the hearing, and here also entire recovery is often brought about by treatment.

IV. In the fourth class of cases, the giddiness is due to irritation of the auditory nerve, chiefly through chronic processes in the middle ear leading to pressure upon the labyrinth at the fenestral openings, or to vasomotor disturbance in the labyrinth. The vertigo of this class is that usually termed by writers *vertigo ab aure laesa*, and may occur in almost any affection of the ear, but usually in the course of a chronic inflammatory affection of the middle ear. The giddiness is generally preceded, accompanied, and followed by subjective sounds and deafness, but it is frequently without nausea or vomiting. Recurrences of the giddiness are common from time to time. In most cases the giddiness eventually disappears, but the deafness and noises in the ear remain permanent.

I.—LABYRINTHINE GIDDINESS.

This is true Ménière's disease or apoplectic deafness, and is a form of aural giddiness, which is most apt to be confounded with cerebral or cerebellar disease. It is perhaps best illustrated by the description of an actual case, such as the following:

CASE I. *Typical Ménière's Disease*.—A married lady, aged 32, was never, up till the time of seizure, conscious of any impairment of hearing or affection of the ear. On a spring forenoon, while walking in the garden, she became suddenly conscious of a loud sound in the left ear and left side of head, like steam blowing off, accompanied by deafness in the same ear. Almost immediately afterwards she became extremely giddy, staggering with difficulty into the house, where she became very sick and vomited freely. After resting in the dining room for two or three hours she was helped up stairs to bed, still extremely giddy. Next morning, however, the vertigo had, to a great extent, disappeared, and, with the exception of slight transient attacks, has never returned. The nausea and vomiting did not return after the first day, but the noises in the ear and deafness continued. A few days after the attack I examined both ears. In the left, or affected, side there was no sign of disease in the external or middle ear. The hearing by air conduction was practically gone, while bone conduction, as tested by Rinne's and Weber's tests was very defective. Inflation of the middle ear by Politzer's method and the catheter had no appreciable effect upon the deafness or on the subjective sounds. The right ear was perfectly healthy, with normal hearing. Seven years after the seizure the condition of the left ear was unchanged. Neither tick of watch nor speech was heard, and the bone conduction was defective. No abnormal change in external or middle ear. The subjective sounds still continued pretty much the same, but there had been no return of the vertigo. This patient had given birth to a child prematurely a few months previous to the attack, and a year afterwards a child—born at full time—had to be treated with anti-specific remedies. The husband, some years prior to his marriage, had a specific affection, for which he was treated for a long time. In the case of the lady a course of pilocarpin, employed subcutaneously, as well as long-continued mercurial treatment, made no impression upon the ear affection.