SOCIETIES' PROCEEDINGS

ROYAL SOCIETY OF MEDICINE—SECTION OF OTOLOGY

February 5th, 1937

President—DOUGLAS GUTHRIE, M.D.

Some Observations on the Function of the Labyrinth

By I. A. TUMARKIN

IN his latest work on the labyrinth Professor Rigaud makes the following statement :—

" Although vestibular examinations have been in widespread use for over twenty years, yet we are still arguing about the nature of the nystagmic tests ; about their correct performance and their diagnostic value. To-day this domain has become the private property of a few initiates who argue among themselves, whilst usually the clinician is quite unable to derive any benefit from the results of their obscure researches."

That is a very crushing indictment but we must agree that it is not unfounded. Neurologists and physicians in general attach little importance to the labyrinth tests, and even otologists are usually content to know whether a labyrinth is alive or dead. The explanation of this is that no one has ever discovered a simple method of measuring the labyrinth sensitivity. In other words, our tests are qualitative but not quantitative. This would not be so bad, provided we kept our limitations in mind. Unfortunately one constantly sees both clinical and research reports in which the labyrinth is found to be, shall we say, slightly hyper- or hypo-sensitive.

Before we discuss the efforts which are being constantly made to give this mathematical precision to the labyrinth tests, let us recall some of the sources of error which are inherent in the standard simple method. I shall confine myself to the rotation tests. The caloric and galvanic tests have their own errors, and do not come within the scope of this work.

Here then are the main sources of error which I propose to discuss in detail.

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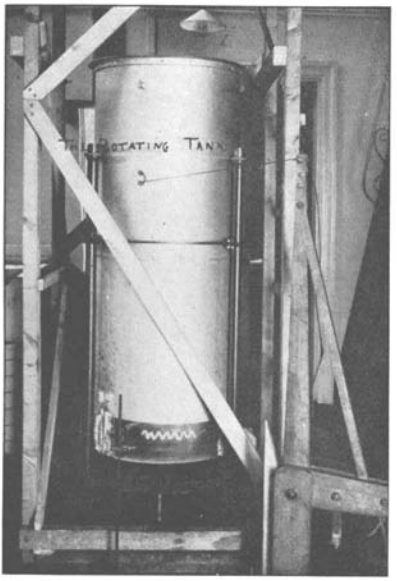


Fig. 1.

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SOURCES OF ERROR IN ROTATION TEST.

(1) Excessive stimulus.
(2) Visual influences.
(3) Personal bias of examiner.
(4) Confusion of post-rotary nystagmus with
    (*a*) Per-rotary nystagmus.
    (*b*) Secondary nystagmus.

Take first the actual method of rotation. The patient is turned by hand ten times in about twenty seconds and then suddenly stopped. This stop probably takes no more than one-tenth of a second. Consequently we have retarded him at the rate of 1,800 degrees per sec. per sec. This is a severe jolt, as many of our patients know to their cost, and as a basis of comparison I attempted to find the threshold for rotary sensation, that is, the feeblest acceleration capable of giving a sense of rotation. References to this threshold in the literature are very scanty. Buys gives a value of 1-2 degrees per sec. per sec., others as much as 8 degrees per sec. per sec. I should like to stress at this point that the specific stimulation for the semi-circular canals is acceleration and not velocity.

The experimental method to be adopted seemed obvious, and comparatively simple. We must rotate our patient smoothly with a gradually increasing acceleration, and ask him to report as soon as he feels the movement. Further, in order to get a pure labyrinthine sensation, we must eliminate all visual, auditory, and kinæsthetic impulses, i.e. the body sensations from the buttocks, the soles of the feet, and so on.

In order to deprive our subject of the pressure sensations on the soles of the feet and on the buttocks we floated him in a tank (Fig. 1) filled with warm water, his eyes blindfolded, and his ears filled with plasticine. As we were dealing with remarkably small velocities it was essential to avoid the slightest suspicion of jerking. This was achieved by means of ball-bearings, rotating contacts, and so on. The motive power was also very difficult to control. Electric motors, dropping weights, geared handles, and so on, were far too crude and jerky. Eventually we used the viscosity motor. This works on the principle that a piston with a small valve in it will drop at a uniform speed through a cylinder of oil. The same principle is utilized, for instance, in dropping a slide through the camera of an electro-cardiograph. By gradually opening the valve we were able to increase the speed of the tank at any rate required.

Recording was quite simple. A waistcoat of smoked paper around the tank, and a $\frac{1}{2}$ sec. time marker, gave us the record. In the beginning rotation was so slow as to be almost unnoticeable. The $\frac{1}{2}$ sec. marks were separated out as the tank gathered speed;

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a cross marked the moment at which the subject reported a sensation of rotation.

A series of results such as these was taken and analysed graphically (Fig. 2). Horizontally we have *time*, and vertically, *velocity*. Acceleration, being the rate at which velocity increases, is represented by the steepness with which the graph rises. The line A B, for instance, is not rising at all—it represents a constant velocity of 2 degrees per second. The line O X is rising steadily. It represents a regular increase in velocity, i.e. a constant acceleration of $\frac{1}{2}$ degree per sec. per sec. The curves represent the various

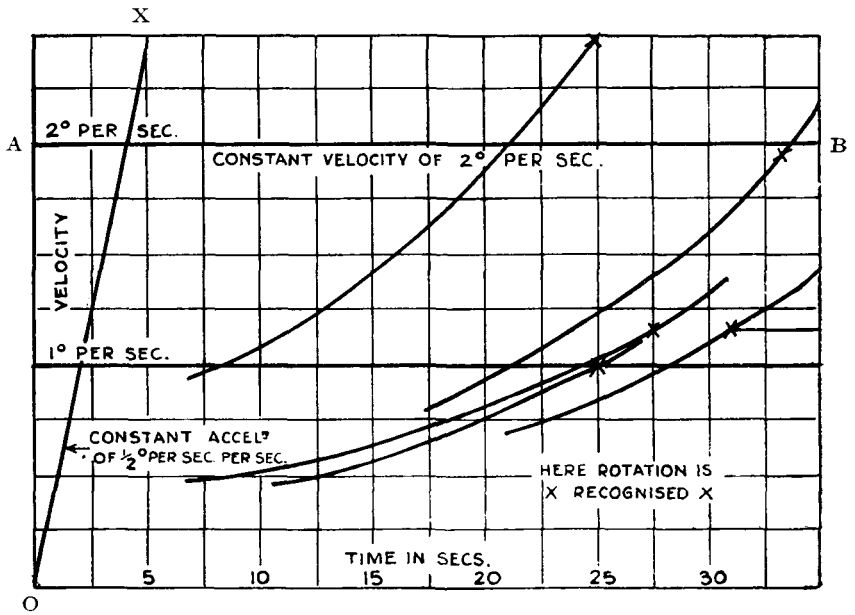


FIG. 2.

flights of the tank. Here we have a very gradual acceleration which increases with the tank speeding up. The cross again indicates the moment at which the rotation was recognized.

It is clear that the acceleration is recognized long before it has reached $\frac{1}{2}$ degree per sec. per sec. These curves are nothing like so steep as the comparison line, as is shown by the angle at X. The actual value lies somewhere about $\frac{1}{5}$ degree per sec. per sec. In other words, the Bárány shock of some 1,800 degrees per sec. per sec., is 9,000 times the threshold—rather like testing knee-jerks with a sledge-hammer. This seems to me a serious disadvantage, but it is by no means the only one.

Consider, for instance, the optokinetic effect. We know that, as a result of the environment swirling past the patient, there is

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set up a form of optical nystagmus which has nothing to do with the labyrinthine reaction. It is a purely visual phenomenon and is easily eliminated by instructing the patient to keep his eyes closed, yet it is surprising how often this simple precaution is forgotten.

The optostatic effects are more serious. The examiner usually observes the eye movements by asking his patient to look at some fixed point, e.g. his finger. Now this very act of visual fixation must have a braking or checking effect on the nystagmus. It will vary from one individual to another and introduces a quite uncontrollable source of error. The use of Bartel's glasses and similar devices is unsatisfactory. We need an apparatus, such as the

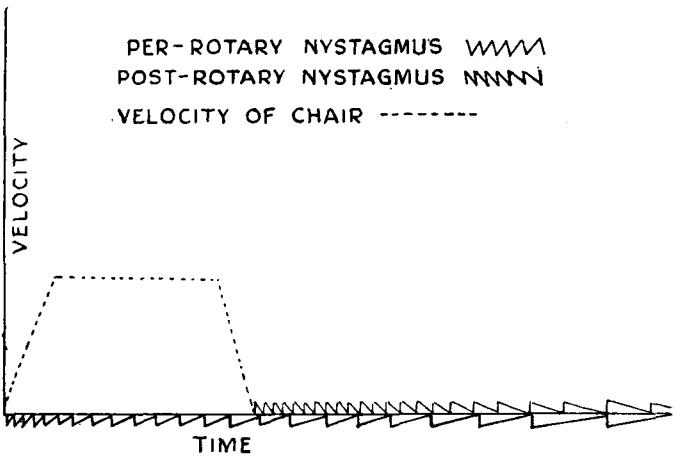


FIG. 3.

nystagmograph, which will record nystagmus whilst the eyes are closed.

Such an apparatus has the further advantage of providing a permanent record which can be scrutinized at leisure and compared with standards. Furthermore, it considerably reduces the error from the personal bias of the examiner. Everyone knows how difficult it is to be certain that nystagmus has ended. The terminal flickers are small, irregular, and spaced out at long intervals. It is only too easy, if one is expecting a short response, to ignore these terminal flickers and, conversely, if one is expecting a long response, to include indefinite eye-jerks of dubious origin.

Further complications are produced by the so-called per-rotary nystagmus. Post-rotary nystagmus is what we usually measure. This is produced by the sudden stopping of the chair. It was naturally suspected that the starting of the chair would also produce a nystagmus, which should be in the opposite direction.

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Its existence, however, was difficult to prove until graphic records were utilized by Buys and others. Such a record is represented by this diagram (Fig. 3). The important thing to note is that the per-rotary nystagmus opposes the post-rotary, and so confuses our result. I shall show later how nystagmography enables us to overcome this difficulty.

Lastly I must refer briefly to the phenomenon of secondary nystagmus. This was originally described by Wodak and Fischer (Fig. 4).

Suppose we have induced a nystagmus in our patient, as at A. After a time it dies down and the eyes become stationary. If, however, we wait a little longer we observe a fresh outbreak of

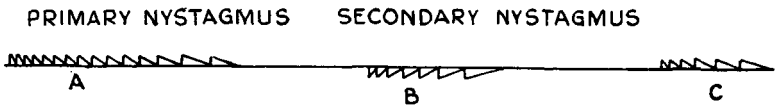


FIG. 4.

nystagmus, as at B, which is in the opposite direction. As a matter of fact, under favourable conditions, a third (C), and even a fourth series of jerks can be observed, the direction in each case being reversed, and there can be no doubt that a really strong stimulus can so affect the labyrinth as to vitiate any other test for several hours. This chaotic mixture of per-, post-, and secondary nystagmus, evoked by the crudest of hand rotations, is the material on which we attempt to base our estimate of labyrinthine sensitivity. Is it any wonder, then, that results are contradictory and clinicians sceptical ?

Suppose, now, that we were in a position to improve our testing technique, what modifications would we adopt. As I have pointed out, visual influences will be eliminated by recording off closed eyes. The patient must not see any environment or fixed point. Confusion with post-rotary nystagmus will be avoided if we confine our analysis to the per-rotary nystagmus itself.

Finally, we must avoid the colossal shocks which are normally used. When I began this work I went to the opposite extreme and attempted to determine the minimum acceleration which was capable of evoking nystagmus, just as I had already investigated the minimum acceleration which was capable of evoking a sensation of rotation.

That this is quite feasible I have not the slightest doubt. On the other hand, it must be confessed that we are as yet far from having available a simple apparatus suitable for ordinary clinical testing.

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A disadvantage of some apparatus, such as those of Kuilman and Dohlman, is the necessity of cocainizing the eye, so as to apply to the cornea a small rubber cap bearing the necessary levers. Such a method would not be popular with patients, especially if repeated tests were required. I have no intention of giving a review of the work which has been done on nystagmography. The briefest account must include mention of Buys, who did so much pioneer work, using pneumatic methods, with rubber tambours applied to the closed eyelids. Galvanic methods for measuring the currents of action in the ocular muscles have not been very helpful, nor has the cinematograph.

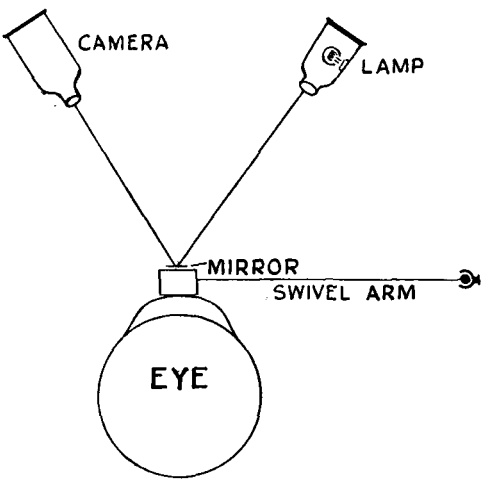


FIG. 5.

My own instrument is a modification of one designed and used by Professor Dodge, of Yale University, some years ago (Fig. 5). His apparatus consisted of a robust spectacle-frame, complete with swivel arms. These carried small blocks of wood on which mirrors were mounted. The swivel arm is rotated so that the wooden block rests lightly on the closed lid. A powerful beam of light directed on to the mirror, is reflected by it and photographed by a special camera which has to be held firmly in a wooden frame between the subject's teeth. The limitations of such an apparatus are obvious, and I was able to improve it by utilizing the properties of the photo-electric cell.

Suppose we set up two cells, A and B, side by side, and direct a beam of light equally on them; their two effects will cancel out and the galvanometer will not record any current. Now let the

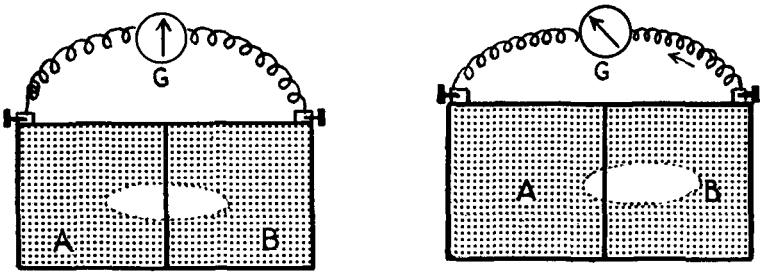
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beam wander towards B. Obviously a current will flow from B to A (Fig. 5a).

The apparatus now to be described utilizes this principle. A robust spectacle frame is adjusted to the subject's head. Two small struts projecting anteriorly carry the double cell A B in a position about 4 in. in front of one eye (Fig. 6.)

A delicate swivel arm carries a bronchoscopic lamp which is mounted in a tiny ebonite base (Fig. 7). This ebonite base is lightly adjusted against the closed eyelid. Any nystagmic jerks will displace the lamp base and so alter the position of the beam of light on the cell. The current set up is led through an electro-cardiograph. It is quite as large as the heart-muscle current of action, and is photographed in the same way.

Let us consider to what extent it complies with the criteria which I set out before. To begin with, it provides a permanent record which is independent of the personal bias of the examiner.



Central beam.
No current.

Excentric beam.
Current from B to A.

FIG. 5a.
The photo-electric cell.

It eliminates all visual influences, as the patient's eyes are closed. Indeed, the test can be done in pitch darkness.

Again, it enables us to record the per-rotary nystagmus. Fig. 8 shows per-rotary nystagmus during one quarter of a turn. The $\frac{1}{2}$ sec. marks above, and the automatic record of the chair velocity below, enable us to estimate that this acceleration was something like 20 degrees per sec. per sec. The details of the nystagmus are easily recognizable.

Lastly, the instrument is surprisingly sensitive. This sensitivity encouraged me to persevere for many months in efforts to determine the minimum acceleration capable of evoking nystagmus. Eventually, however, for various reasons, I came to the conclusion that the method was not practicable. It was not easy to rotate the chair with the required minimal acceleration. Recording the actual speed was troublesome—much more so than with the tank. Above

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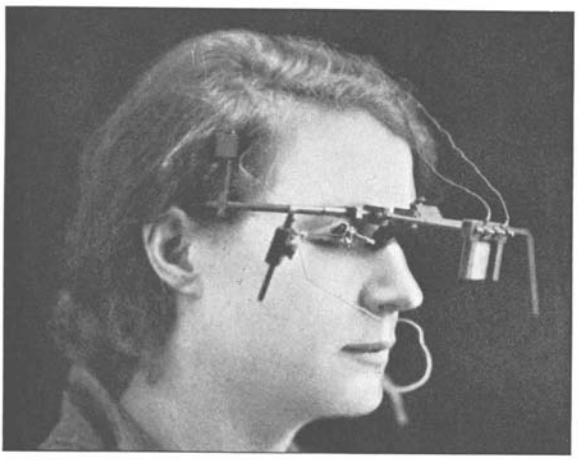


Fig. 6.
Nystagmograph in position.

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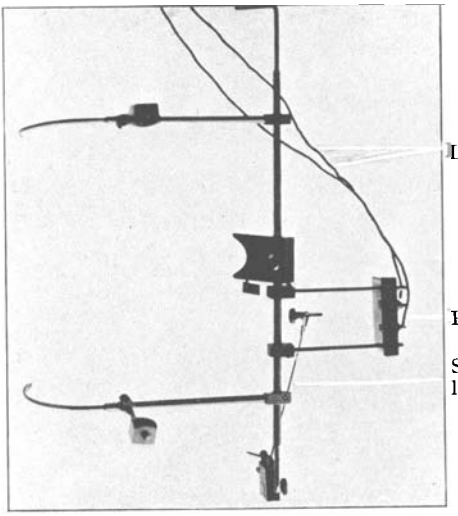


FIG. 7.
Nystagmograph.

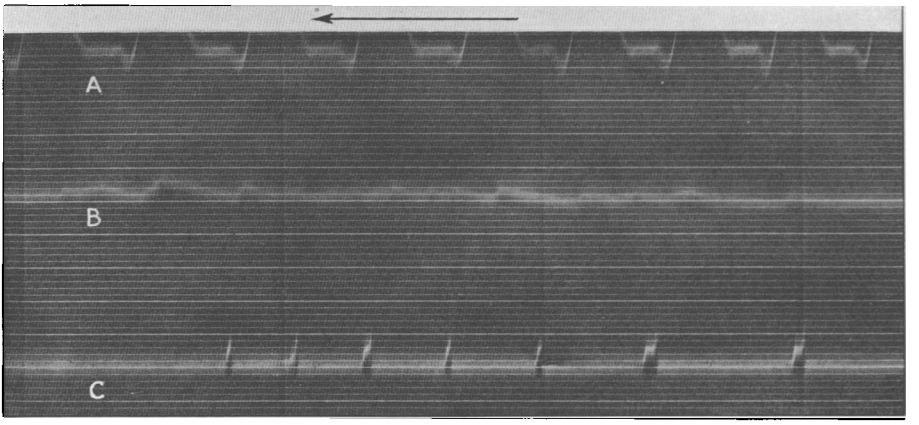


FIG. 8.
A = Time record, $\frac{1}{2}$ seconds.
B = Nystagmus.
C = Chair velocity.

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all, however, tests were liable to be vitiated by blinking, which gives jerks very similar to those of true nystagmus. Under a powerful rotary stimulation the nystagmic reflex is pre-eminent and suppresses the blink reflex ; furthermore, the sequence of true nystagmic jerks is absolutely unmistakable. Under conditions of minimal rotation, however, the converse obtains, and our nystagmograms are no longer reliable, on account of uncontrollable blinking. Naturally, only a few jerks are produced, and one cannot be sure whether they are nystagmus or blinks. These difficulties could probably be obviated by utilizing a technique which I have had under consideration for some time, namely, the determination of the per-rotary nystagmus which can be evoked by a definite acceleration, say, of 20 degrees per sec. per sec. (Fig. 9).

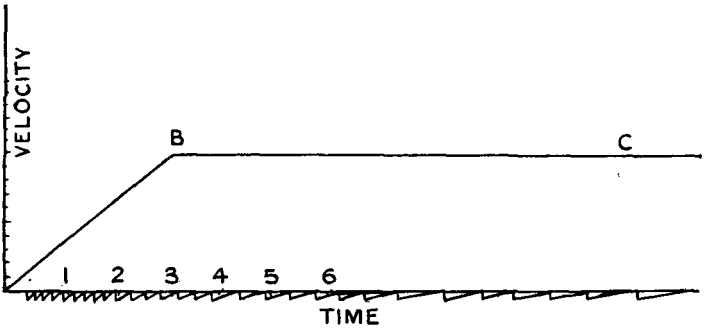


FIG. 9.

Proposed method for determination of per-rotary nystagmus.

The graph shows a steady acceleration of 20 degrees per sec. per sec. for three seconds. This is quite sufficient to give us a definite and recognizable nystagmus. At B the velocity is 60 degrees per sec. We must not stop the chair, otherwise we shall get a post-rotary nystagmus. Instead we keep it revolving steadily as at B C until the per-rotary response has died down. For comparison I have drawn graphs illustrating the standard method and also the Buys-Fischer method which is being adopted by investigators on the Continent (Fig. 10). In the standard method a rapid irregular start is followed by a roughly uniform velocity for 20 sec. which is then brought up to a dead stop. It is clear that the post-rotary nystagmus is being vitiated by the per-rotary nystagmus and also by its secondary effect.

The Buys-Fischer method attempts to obviate this by rotating the patient at a very low acceleration—so low that per-rotary nystagmus is not set up. Gradually the chair gets up speed and when it is rotating fast enough it is brought to a standstill by a special brake, and the examiner measures the pure post-rotary

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nystagmus thus obtained. Special chairs are built which can be rotated at an acceleration of 1 degree per sec. per sec., which was believed to be below the threshold. Even this low acceleration is, however, not really below the threshold which I have placed at $\frac{1}{5}$ degree per sec. per sec. This method still has many disadvantages. The terminal jerk is still severe and not accurately measurable, and also the optostatic and personal bias factors are present.

This third method, which I have just outlined, has not yet been put to the test, owing to difficulties of time and expense. A special chair, or rotary platform, will have to be constructed. It seems

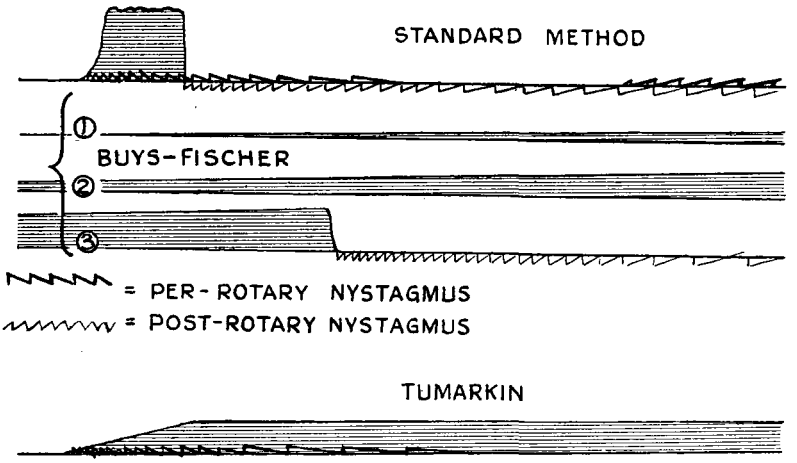


FIG. 10.

to me, however, a reasonable solution for the various problems and difficulties which I have outlined and as such I submit it for your criticisms.

I should like now to turn from this arid and academic subject to something which I hope will be a little more interesting and clinically useful.

Last year I drew attention to the fact that sufferers with Ménière's syndrome presented two entirely different types of attack.

MÉNIÈRE'S SYNDROME.

	Syndrome of Semicircular canals.	Syndrome of Otolithic catastrophe.
Onset	Gradual.	Instantaneous.
Duration	(Hours to days.)	(Minutes or less.)
Nausea, pallor, etc. ..	*Yes.*	*No.*
Disorientation	*Yes.*	*No.*

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The standard type, which I refer to the semicircular canals, is characterized by gradual onset. Although patients may declare that their giddiness comes on suddenly, yet close questioning usually reveals that they do not fall down or hurt themselves. On the contrary they are able to get to a chair or other resting place. The attack may last for hours or even days, and is associated with visceral symptoms: Pallor, nausea, sweating, and so on. Disorientation may be very pronounced.

The second syndrome, which I have termed "the otolithic catastrophe", presents a marked contrast. The onset is instantaneous. One of my patients declared that he was standing at his desk talking to a client and the next second he was flat on his back on the floor. Another man, a furniture-remover, was standing in his van when suddenly he collapsed. His own words were that he thought one of the wheels must have fallen off and that the van had crumpled up. There is no loss of consciousness in these attacks, and my patients have never complained of nausea, faintness, sweating, or other sympathetic symptoms. They do not complain of disorientation; indeed there is hardly time, as the attack lasts only a matter of seconds and ends almost as quickly as it begins.

It might be argued that this second syndrome is nothing more than a severe degree of the first syndrome, but a moment's consideration will suffice to refute that. If it were an ordinary attack it must be a severe one to strike down, as it does, so dramatically and without warning. In that case surely we should expect it to last longer than a minute. Further, we should expect our patient to be violently sick, completely disorientated, and so on. This is not the case, and I suggest that the evidence is quite sufficient to justify us in distinguishing two syndromes which can be ascribed respectively to the semicircular canals and to the otoliths.

Admittedly we know little of the functions of the otoliths. I am well aware that experimenters have triumphantly disproved almost every suggestion which has ever been made about the static labyrinth. Nevertheless, as a common-sense working hypothesis, the theory of muscle-tone control has much to commend it, and accords well with these clinical observations.

There is ample evidence to connect the utricular otolith with the maintenance of flexor and extensor tone. Magnus showed quite definitely that decerebrate rigidity could be systematically altered from maximum to minimum by rotating the animal's head. At one extreme the full weight of the otolith is pressing on its neuro-epithelial bed. In the opposite extreme it hangs upside down and drags on its moorings. Figure 11 shows these two extremes, and the corresponding body postures produced.

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The dramatic collapse is referable to a sudden increase in flexor tone (Fig. 11C). Such a patient will double up like a clasp-knife.

As to the saccule, the probabilities are that the two sides act

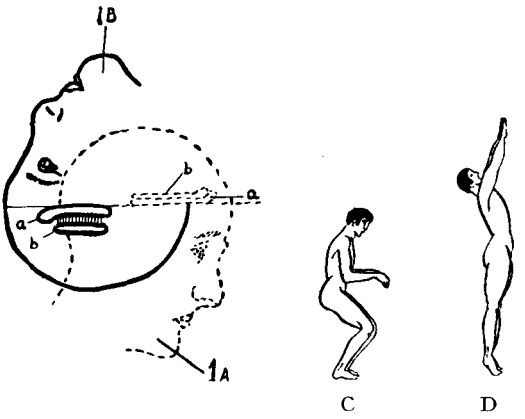


FIG. 11.

In the upright position (1A), *b* is pressing on *a*. In the upside down position (1B) it will drag, and intermediate positions will produce intermediate effects. The right and left lapilli are synergic.

C, D } Show corresponding positions of flexion and extension.

in opposition and affect abduction and adduction (Fig. 12). My second patient, who crashed on his side, is possibly an example of saccular imbalance.

I would point out that this differentiation of the classical-symptoms complex has more than a purely theoretical interest. To begin with, the rare fatal accidents which occur in Ménière's

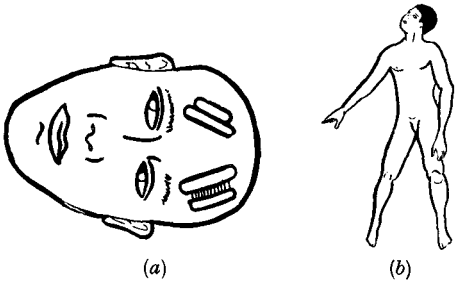


FIG. 12.

The saccule (*a*) is anatomically very similar to the utricle. The chief difference lies in its position in space. The sensitive area (sagitta) lies on the side instead of the floor of its retaining sac; furthermore, the right and left sagittae are roughly at right angles to each other, so that unlike the lapilli they are opponents and not synergists. (*a*) Shows this opposition and (*b*) the resulting posture.

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syndrome are almost certainly due to the otolith catastrophe. The absence of sickness is noteworthy, because it is opposed to Quix's theory that sea-sickness is due to over-stimulation of the otoliths.

Most important of all, however, is the possibility of erroneous diagnosis. Of course, where the patient shows both syndromes, the otolith catastrophe will be accepted as part of the general condition of aural vertigo. But there must be cases in which the otolith effect stands alone. Such a case, in which the patient merely complained of " falling down ", might easily be diagnosed as one of hysteria, minor epilepsy, and so on. One of my patients was even suspected of cerebral abscess. By keeping this possibility in mind, the otologist may possibly preserve his medical or surgical colleagues from falling into an error which would prove troublesome, if not disastrous, to the patient.

In conclusion, I am happy to take this opportunity of expressing my gratitude to Professor Dodge, of Yale University, for sending me a copy of his nystagmograph, out of which my own was evolved, and to Professor Roaf, of Liverpool, for his constant help and advice and for the facilities which he provided in his laboratories, where most of my experiments were performed. I am also deeply indebted to Mr. Perrin, of the Cunard White Star Line, for designing the rotating tank.

Discussion.

Mr. C. S. Hallpike asked if Mr. Tumarkin would give a few more details concerning the inside of the tank he used. He also asked how the threshold of nystagmus shown by the graphic method compared with the threshold shown by the purely optical method, using Bartel's glasses ; he believed there could be no objection to the use of the latter. The advantage of that method was that there was no load on the eyeball, nor was any mechanical system introduced. There seemed no reason why fixation should occur with a highly-magnifying lens in front of the eye.

As to the saccule, any contribution that his colleagues and himself had been able to make might be considered complementary to those of de Kleijn and Versteegh, who showed that the saccule in the rabbit had no relation to equilibrium. They (Ashcroft and Hallpike) had recorded electrically the physiological response of the saccule, and so had obtained positive evidence of what the saccule responded to.

Mr. F. Watkyn-Thomas said that all the effects of the so-called " otolith catastrophe " could frequently be seen in a patient who had a labyrinthine fistula about the torus of the external canal. In two such cases he had seen exactly the picture described, the slightest pressure over the torus fistula bringing forward that crashing effect, an instantaneous one, with practically no after-discomfort or trouble.

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One of his patients brought it on himself while shaving, and found himself in the fireplace ; the fistula was fairly far back on the external canal, and, in that case at all events, the cupola of the external canal must have been affected before the otolith.

Mr. SYDNEY SCOTT said that they would all recognize the two syndromes described, though he himself hesitated to accept Mr. Tumarkin's explanation. Mr. Tumarkin had been dealing with rotatory and post-rotatory phenomena, but he (the speaker) would like to suggest that the collapse was not a relaxation collapse, but was due to strong tonic contractions—a " flinging-down " action, not mere relaxation, thus differing from what happened in a faint.

The PRESIDENT asked to what extent the threshold of labyrinthine irritability varied in normal individuals. In making labyrinth tests he had felt the need of a normal standard for comparison, and it was difficult to postulate such a standard, on account of the existence of variations in normal healthy persons. The unfortunate possessors of a sensitive labyrinth, although they felt most uncomfortable at sea or in the air, might be perfectly happy and healthy under stable conditions. Apparently the boundary between physiology and pathology was ill-defined so far as the labyrinth was concerned.

Mr. TUMARKIN (in reply to Mr. Hallpike's question as to the inside of the tank) said that the point had also occurred to him at the beginning, but it did not prove a real difficulty. The question was : If a person was floated in a tank of water, would he go round with the water when the tank was rotated ? To ensure this a couple of baffles were constructed across the tank, dividing it into three compartments. As it happened, even this was unnecessary as the rotations were so slow. In the experiments the patient was carried round smoothly with the water, there was no surging to and fro.

With regard to the Bartel's glasses, he did not agree that they removed the power of fixation. It was preferable to record off closed eyelids in pitch darkness, as in his own method. He wished to make it quite clear that the string galvanometer and recording camera were *not* rotated with the patient. They could, in fact, be in a different room. It was quite easy, by means of simple standard formulae, to calculate the acceleration of the chair given the time and distance rotated. He was familiar with de Kleijn's experiments which suggested that the saccule did not function in maintaining posture, but it was to be remembered that the results of selective ablation experiments must be received with the greatest caution. His conception of a dual syndrome was put forward on the strength of clinical observations, and accorded well with the standard hypothesis which was accepted for the working of a labyrinth.

As to the patient who suddenly collapsed into the fireplace : It would not be easy to work out mechanically the effect of pneumatic compression of a fistula of the external canal, but it might easily affect the otolith as much as the canal.

The point raised by the President as to the variation of the threshold was a very important one. Possibly it might be the rock on which the whole attack on this problem would founder. If the patient's

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labyrinthine sensitivity varied from day to day, it might be that investigation of the threshold would be of no value. That, in fact, was one of the reasons which had made him abandon that part of the problem.

The suggestion that the sudden collapse was an active phenomenon was very interesting, and he was grateful to Mr. Scott for drawing his attention to it. It was an added factor in the differentiation of the two syndromes. In the canal type there was a flaccid collapse. The patient fell, or rather lay, down in order to increase his base. In the otolith type he was thrown down. The original simile of " collapsing like an empty suit of clothes ", was therefore inaccurate. It might be better to speak of " collapse like the closing of a clasp-knife ".

ABSTRACTS

EAR

Complete Apicectomy. A new technique for the complete exenteration of the Apical Carotid Portion of the Petrous Pyramid. JULIUS LEMPERT. (New York.) (*Archives of Oto-Laryngology*, XXV, 2, February, 1937.)

The confusion which exists in descriptions of operations on the petrous pyramid is partly the result of inadequate detail in anatomical text-books regarding the structure. Lempert suggests that the petrous temporal should be viewed in two portions: (1) the basal labyrinthine portion and (2) the apical carotid portion. He describes each portion in detail. Suppurative peri-labyrinthitis is seldom diagnosed prior to operation, when fistulous tracts leading to infected cells and epidural collections of pus may be found. Suppurative apicitis may appear secondary to acute purulent otitis media without mastoiditis, or it may exist concurrently with mastoiditis or as a sequel to suppurative peri-labyrinthitis appearing after mastoidectomy. The majority of cases of apicitis recover without any surgical intervention, the infection never passing beyond the stage of congestion. Apparently the infection tracks directly through the tympanic cavity into the pyramid in many cases, leaving the mastoid process uninvolved. It is said, however, that 20 per cent. of the patients die of meningitis. Since the first report of Kopetzky and Almour in 1930, " the otological world has become petrositis conscious ". The subject is still controversial. Apicitis does not always become suppurative and only when suppuration is recognized clinically and radiologically does surgical intervention become essential. But there is no middle course in treatment and if suppuration is diagnosed it must be treated surgically. The question is not, " Is operation

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