

## VERTIGO

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In this preliminary paper we shall endeavor (1) to outline, necessarily very briefly, a conception of vertigo which appears to us to be based on anatomic and physiologic data, and (2) to point out how this conception aids in the diagnosis and treatment of cases of vertigo. No attempt will be made to enumerate the diseases in which vertigo is a more or less conspicuous symptom, for it appears to us that with a clear conception of what vertigo is there will be less necessity for any such elaborate and detached enumeration. It is unnecessary to apologize for discussing this subject, for the significance of vertiginous attacks offers difficulties both to the general practitioner and to the specialist in different branches of medicine.

Vertigo, when considered from the point of view of etiology, has been and still is a subject of great complexity. Its frequency in so many diverse diseases offers a fruitful field for speculation. The importance of its interpretation from the point of view of diagnosis need not be insisted on. We all know that it is most elusive in response to treatment. One difficulty in regard to its interpretation lies in the fact that most of us have no clear conception of what vertigo is. Subjectively, we all have had experience with it. But few would be prepared offhand or even after considerable thought to define it in terms conveying any conception of its anatomic or physiologic significance. Yet without such a clear conception its diagnostic significance must be largely lost and its treatment must be haphazard.

The term "vertigo" is commonly applied to a sensation of rotation of surrounding objects or of one's self. While true that rotation is a conception of vertigo in its most pronounced form, still there are other forms in which the sensation is not that of rotation, but of a feeling of instability either of one's self or of surrounding objects. There may be the feeling, not that either we or the objects around are rotating, but that we are falling or swaying, or that the objects around us are sinking or rising.

Not all its modes of manifestation, however, can be expressed clinically in terms of instability. We are here dealing with a subjective phenomenon, and every one recognizes how difficult it is to get accurate data on subjective sensations. We all know how the same feeling may be expressed by different persons in different ways, apart from the fact that the sensation may be more marked in one than in another. Investigation has shown that from the clinical point of view we must include under the term "vertigo" sensations of many kinds: a blurring vision, a feeling of discomfort or of anxiety, fulness in the head, as after smoking; a feeling of weakness, a disturbance of consciousness; in short, a variety of sensations which, if they persist, tend sooner or later to become more pronounced as disturbances of equilibrium.

While vertigo may be traceable most frequently to some pathologic condition disturbing, either directly or indirectly, the mechanism concerned with equilibrium,

there are occasions when obviously no such pathologic condition exists. For instance, it may be traceable to the possibility that the surrounding circumstances are such that should a disturbance of equilibrium result, a fall of some considerable magnitude might follow; or there may be simply the fear that, given certain eventualities, a fall would occur; for instance, looking over a bridge into a ravine, or standing on the parapet of a tower with thick mists obscuring the view. That we know the sensation to be irrational does not seem to have much weight.

When vertigo has once arisen under certain conditions, as of fear arising on our being in a dangerous position, it may easily be aroused again under conditions similar in kind though less rational in reality — probably dependent on the fact that anatomic paths once traveled are more easily traveled again.

In the monograph "Der Schwindel," Ewald defines vertigo as a depression of our static consciousness, and Wollenberg as a perplexity in regard to our condition in space, both of which definitions, as Ewald says, amount to the same in the end. We venture to suggest that such statements are so obscurely based on anatomic or physiologic data that they do not help toward a clear, satisfactory definition.

All are agreed that vertigo is related in some way to disturbances of equilibrium. In order to get a clear conception of what vertigo is, and where the disturbance which causes it may arise, it is necessary to have a conception of the paths along which impulses which maintain equilibrium may pass. To preserve equilibrium impulses are constantly coming to the brain from four chief peripheral sources: (1) the ear, (2) the eye, (3) impulses from muscles, tendons and joints, and (4) impulses from the skin, touch and pressure. These impulses and probably others as well, for example, tactile hairs,<sup>1</sup> result in muscle coordination whose most important function is to preserve equilibrium.

We are all agreed that vertigo bears some relationship to a disturbance of equilibrium. But extensive nerve lesions as in ataxia can make difficult or impossible standing or walking without the help of the eyes, without, however, causing vertigo. The tabetic with his eyes shut has instability. He then may have the sensation of vertigo, but he does not fall because he has vertigo. He falls because he experiences a sudden unexpected loss of afferent impulses from the eyes on which he has been depending to reinforce the other impulses which, unaided, are insufficient to maintain his equilibrium. He has vertigo because the sudden closing of the eyes has intensified the mental confusion resulting from the inadequacy of the impulses from the muscle, tendon, joint and skin, which inadequacy has been compensated for by the visual impulses. In the tabetic the muscular incoordination which is the primary factor may appear without vertigo. In the labyrinthine irritation, the instability is the outward manifestation of the vertigo, which vertigo is, or has been, present and may precede the instability.

Vertigo arises from a confusion or dissociation of the impulses on which the subject bases his conception of his position in space. By far the most important of these impulses concerned with the conception of the position in space, or change of position in space, arise from sense organs in the head segment, the otic labyrinth and the muscular and retinal portions of the

1. Vincent, S. B.: Jour. Comp. Neurol., 1913, xxiii, 25.

eyes. In the tabetic, the mechanism of the head segment is not primarily disturbed, but the proprioceptive (muscle, tendon, joint) and the skin sensations are disturbed.

Ataxia may lead to disturbance of equilibrium, but we may have ataxia without vertigo, or vertigo without ataxia. Both ataxia of a certain sort and vertigo are due to disturbances of afferent impulses concerned with equilibrium, but the ataxia is due to a loss in a group of afferent impulses, while vertigo is due to a disturbance of the normal association of certain groups of afferent impulses, especially those from the head segment. An animal after double labyrinthine removal may have its objective signs of vertigo reduced by throwing out other afferent impulses and so reducing the factors which cause the disharmony. Although for purposes of brevity, we are retaining the term "ataxia" in the present discussion because of its universal use, we wish to point out here that it is a very inexact term at present and should be more clearly defined.

It will, then, be noted that what we insist on is that it is not instability alone which constitutes vertigo on its subjective side; it is the rising into consciousness of the sensation of instability; it sometimes is the fear that such an instability may occur, or a fall be possible. There may be no outward objective symptom of vertigo; or there may be incoordination, and the consequent muscular endeavor to maintain equilibrium.

Normally, the afferent impulses which maintain equilibrium are harmonized subconsciously, and it is only when disorganized that they ordinarily attract our attention. The explanation of this is obvious. They are so essential to man's existence and so constant in their activities that Nature has arranged that they work subconsciously; otherwise their insistence on recognition and conscious adjustment would be a serious detriment to man's mental development. But, as in many other subconscious activities, consciousness may enter in either as a controlling or disturbing element, for example, through memory and emotions. Such a disturbing influence from a higher mechanism may cause disharmony, and so there arises psychic vertigo.

In the cerebrum these constant afferent impulses are, under ordinary conditions, associated, or to use a better term, harmonized or integrated, to preserve the equilibrium. If for any reason they become dissociated or disharmonized or disintegrated, the conscious confusion that results is vertigo. The motor incoordination which forms the objective symptoms of vertigo is a result of this dissociation.

Vertigo for the present may be considered as the confusion resulting from the coming into consciousness of afferent impulses concerned with equilibrium which ordinarily are associated (integrated), but now for some reason have become dissociated. Of the impulses coming in from the peripheral organs mentioned, the most important is the ear, and any slight disturbance of the labyrinth will produce severe vertigo. On the other hand, a gross lesion of the third and fourth groups mentioned above may exist without vertigo. Disturbances of the eye are midway between the two; and probably lesions here are largely influenced by the close association of eye movements with the labyrinth. We should elaborate this point, as it is fundamental to a conception of our views. The physiologic action of the labyrinth is primarily to preserve the knowledge

of the position of the head in space and reflexly to maintain equilibrium. To do this the labyrinth is closely related to the musculature of the body, especially the muscles of the head, and the relationship is more clearly discernible in its action on the freely movable muscles of the eyes. An observable outcome of this relationship is that, no matter how the head is turned, the eyes involuntarily are readjusted. Otherwise, the outside world would move with each movement of the head and endless confusion result. Mechanical irritation of the labyrinth with the head at rest, for instance, with hot water, results in a message from a receptor which, normally interpreted, indicates that the body is rotating and calls for certain movements of readjustment. But in this instance the inflowing messages from Groups 2, 3 and 4 are uninfluenced by this irritation and contradict the messages sent in from the labyrinth and resist the demand for readjustment. So there results a confusion in the integrating mechanism arising from this dissociation of peripheral messages; impulses which normally work together are no longer in harmony; and the person, becoming conscious of the disharmony, experiences the sensation which we call vertigo.

Up to a certain point rotation may be carried on, as in a swing, and compensatory movements occur by which means the subconscious impulses are able to adapt themselves to these impulses and no vertigo results. But let the rotation be so great that the impulses from the last three groups no longer fall into line with those arising from rotation, and the controlling power of the cerebrum no longer suffices—then vertigo results. Practice and age are in this an important factor.

Visual vertigo does not so often come under the observation of the clinician as labyrinthine. It can often be produced if we look at a large mirror which is moving without our knowledge. Double vision or errors of refraction cause vertigo because they give false impressions of position in space. The eye muscles, partly because of their close relationship to the labyrinth, play a greater part in vertigo than the other muscles of the body.<sup>2</sup> Their incoordination arising from any cause or the nystagmus which arises when the labyrinth is stimulated is a manifestation of this.

Not only does vertigo arise from disturbance of the peripheral sense organs, but lesions involving their central paths will also produce it. So disturbance of the complex vestibular path, or of the great coordinating mechanism for the unconscious sensations from the muscles—the cerebellum—will give vertigo. One thing that has been impressed on us by experiment is the importance of pressure on the eighth nerve or in the posterior fossa as a cause of vertigo. Clinicians have paid too little attention to the effect of pressure, which need not be very great to produce signs of vertigo.

So far as the areas in which this integration takes place are cerebral, cerebral lesions will cause vertigo, and there is sufficient evidence to show that vertigo does arise from lesions of the cortical mechanisms. So a tumor of the cerebrum may set up an excitation giving a sensation of position in space at variance with impulses from the eye or ear, and vertigo results.

Again, it may occur in a hyperexcitability of the nervous system. So in neurasthenia, in which we have

2. Wilson and Pike: *Proc. Soc. for Exper. Biol. and Med.*, 1913, x, 81.

probably a decreased neural resistance in certain paths and systems due to disturbed metabolic processes, we may have vertigo. Here it may follow that the impulses, say from the two eyes or from the two ears, vary in intensity, and vertigo results. Ordinarily, these slight differences in the intensity of the impulses from the two sides may not rise above the threshold value of the reflex arc, and no symptoms follow.

We may therefore have vertigo due to: (1) organic lesions, or (2) functional disturbance.

Now as the diagnosis of a functional disturbance implies the consideration and elimination of organic lesions, in all cases of vertigo we have to examine at least the four sources mentioned above. And as of these the labyrinth is the most important, the condition of the ear must always be considered. Very slight irritation of the sensitive labyrinth will produce vertigo and muscular incoordination. Even when the muscular phenomena are manifest in the eyes alone more or less vertigo is present. It must be noted that in slight cases we can inhibit the vertigo just as we can inhibit a labyrinthine nystagmus. In such cases the dissociation of afferent impulses is reassociated through conscious processes, and the objective as well as subjective phenomena are controlled. The instability is the outward manifestation of the vertigo or rather of the subconscious state from which the vertigo arises. But the otologic aspect of the problem is not confined to the internal ear. It extends to the vestibular nerve and its central connections, some of which are known, but the greater part are still in doubt.

As equilibrium is a function of the labyrinth, so vertigo is one of the most important symptoms of its disease. We may distinguish: (a) direct vertigo, due to lesion of the vestibular apparatus, and (b) indirect vertigo—disturbance of the vestibular tract, for instance, from gastro-intestinal disturbance.

In both of these, the sickness and vomiting may be due to the spread of vestibular irritation to a neighboring mechanism or, in the indirect vertigo, from the stomach itself. It is not the intention at this time to elaborate these points, but rather to indicate how the otologist attacks the problem. This we can best do by outlining briefly a few of the cases that have come under observation—our remarks being directed toward methods of diagnosis and of treatment.

**CASE 1.—History.**—W. G. K., man, first seen December, 1912, complains of repeated attacks of vertigo with nausea and vomiting during the last four years. These are increasing in severity and frequency so that now he is frequently incapacitated for work. There is a constant feeling of uneasiness. He is very subject to "colds in the head," and when these occur an attack often comes on. He has had a course of bromids for over a year. There is no history of syphilis.

When first examined he had occasional subjective sensations of movement of rotation, but no nystagmus was observed. During a severe attack objects go to right; the patient feels himself going to right but falls to left. If he lies on right side vertigo is diminished. Observation during attacks showed: (1) nystagmus, slow to left; (2) turning eyes or head to left, vertigo less; to right, vertigo increased; (3) skin cold.

Examination during interval when no pronounced vertigo, showed: Romberg to left, no nystagmus; hearing, right, normal; left, markedly diminished, diminution of tones at both ends of scale but especially for lower tones. Rinne, negative; Weber, to right; bone conduction diminished. Both tympanic membranes slightly thickened and retracted; left slightly congested. Nasopharynx, markedly congested;

numerous swollen follicles; synechiae in recessus pharyngeus (Rosenmuelleri). Left eustachian tube swollen and opened with difficulty; right normal.

**Diagnosis, Treatment and Results.**—Nasopharyngitis; tubal congestion on left side; left labyrinth affected. Nasopharynx and tube treated, synechiae broken down, follicles destroyed and zinc chlorid (from 2 to 5 per cent.) applied. Catheterization. Sodium bromid,  $7\frac{1}{2}$  grains, twice a day. The treatment calls for no particular remarks. The sodium bromid was stopped after a fortnight. The nasopharynx rapidly improved and the tube became less congested. With this improvement the vertigo disappeared and the hearing improved.

**Present Condition, June, 1914:** The patient has received no treatment for several months. Hearing is improved in left ear. Both tubes are equally patent. There have been no attacks of vertigo; once or twice when patient had cold in the head there was a feeling that it might come on.

The symptoms in this case point to a combination of internal and middle ear deafness. We believe that there is here a hypersensitive labyrinth probably due to vascular changes similar to those found in the nasopharynx and middle ear. Increase of extra labyrinthine pressure on a susceptible labyrinthine mechanism may produce vertigo. Slight increase of pressure may not do so; the pressure must bear a definite relation to the threshold value for stimulation of the vestibular nerve.

The following points are worthy of emphasis:

1. Patient falls to the left: In labyrinthine disease the patient falls to the side of the lesion. Bárány has stated certain laws in regard to the relation of the quick phase to falling. But as we have pointed out elsewhere,<sup>2</sup> this general rule holds: falling occurs in direction of slow phase of nystagmus.

2. Apparent movement of self to right: Nystagmus during attack slow to left. This is in agreement with the rotation law that apparent rotation of one's self is always in direction of rapid phase of nystagmus.<sup>3</sup> As a corollary, if patient's history show the subjective feeling of movement of self to right and on examination no nystagmus be present, we can assume that the nystagmus would correspond to this law, that is, be slow to left. This is an indication of the ear affected—subjective rotation to right—ear affected left. One may here note how difficult it is to get a distinct history of subjective sensations even from intelligent patients, and further, how much less reliable are the subjective sensations of apparent movement of external objects.

3. Sensations of apparent movement or rotation can persist after or without nystagmus.

4. Turning eyes to left, direction of slow phase of nystagmus, vertigo less: Quick phase of nystagmus alone can be inhibited and inhibition of quick phase inhibits rotatory sensations of self, of external objects and diminishes vertigo.<sup>4</sup>

5. In disease of the labyrinth the patient lies on the sound side; turning to the diseased side makes the vertigo worse.

The importance of pressure or variations of pressure on the labyrinth or on the vestibular nerve from whatever source as a cause of vertigo is not fully appreciated even though not uncommon, for example, after catheterization or Politzerization. We find that Högyes<sup>5</sup> more than a quarter of a century ago pointed

3. Bárány, quoted by Holt: *Ocular Nystagmus and the Localization of Sensory Data During Dizziness*, Psychol. Rev., 1909, xvi, 391.

4. Bárány: *Monatsschr. f. Ohrenh.*, 1906, xl, 193.

5. Högyes: *Arch. f. d. ges. Physiol.*, 1881, xxvi, 558.

out the association and explanation of vertigo from pressure in the ear, and we find that such an aurist as Politzer<sup>6</sup> and such a neurologist as Babinski<sup>7</sup> have noticed the effect of such pressure and the relief following its removal. More recently Bárány<sup>8</sup> has pointed out the possibility of its occurrence. The reason of this non-appreciation of the importance of pressure is not far to seek. The modern aurist still clings to the idea that the stimulation of the nerve endings in the semicircular canals is due to endolymphatic currents, even though Mach and Crum Brown long ago pointed out that such currents were inconceivable in such minute canals.

**CASE 2.—History.**—Man, aged 55, admitted to Wesley Hospital, November, 1912, complaining of vertigo. The first attack was three years ago, at which time he was dizzy and felt faint. He was then treated for left ear deafness by middle ear inflation with eustachian catheter and air pressure. After two weeks of treatment the vertigo left. He was free of discomfort for seven or eight months; then came a second attack which was less severe. About one year ago he had a third attack, less severe than the second. He was first seen by one of us in the present fourth attack, which came on about four weeks ago. He ascribes this attack to excessive fatigue. In the evening while in his office he was suddenly seized with dizziness; the room appeared to rotate and he was compelled to lie down. The severe vertigo lasted about three minutes, during which time he felt as if he were being turned round and round, but the direction of apparent rotation could not be accurately ascertained. This was the first time he had had a distinct sensation of rotation.

Since then he has been more or less dizzy all the time with occasional violent attacks during which a distinct sensation of apparent rotation of self and external objects is present and tinnitus may be increased. Vertigo is so constant and so subject to increase that he has had to abandon his practice. The symptoms are more marked in the evening. At night in the dark he often walks with a staggering gait. When lying down with his head braced he is relieved to some extent, but movement seems to increase his trouble. Reading or writing aggravates the attacks. When the dizziness is severe he feels unable to locate the position of the head, for instance, the angle at which it is; this he describes as inability with eyes shut to locate the head in space.

**Present Condition.**—Right ear normal for low voice and whisper; watch heard at arm's length; Rinné positive; high tones normal; membrana tympani normal; tube patent. Left ear: hearing deficient, traced to an explosion ten years ago; watch heard at 12 inches; Rinné positive; high tones deficient; bone conduction diminished; membrana tympani normal; tube normally patent. Romberg to left; no nystagmus; no increase in blood-pressure; eyes and other organs normal.

**Comment.**—The symptom of more or less constant vertigo is frequently associated with irritation of some part of the vestibular tract. In this case we believe that the vertiginous state from its association with auditory impairment was due to varying pressure on the left vestibular division of the eighth nerve or its terminations probably from disturbances of circulation. In such conditions good results are obtained from pilocarpin followed, if necessary, by iodids. The tendency recently has been to decry the value of pilocarpin, but there are many competent observers who have found it very serviceable. It is not our intention here to discuss its action, but among recent publications reference may be made to the work of Dixon and Halliburton.<sup>9</sup>

**Treatment and Results.**—Pilocarpin 1/20 increasing to 1/6 grain to obtain the physiologic action of the drug. The results were satisfactory. The violent attacks completely disappeared and the patient went home very much improved and

was soon able to resume his work. One of us has kept in touch with this patient since, although he lives at some distance. The reports have been satisfactory until this recent communication:

July 7, 1914: "For the last six weeks the vertigo has returned in a mild form, appearing before dinner or just after and lasting until retiring. The postnasal catarrhal symptoms, manifested mostly by hawking and spitting with which I have been troubled almost all my life, do not appear any worse than usual. I do not believe the catarrh has anything to do with the symptoms of vertigo. The ear inflation you remember, before I came to you, made the vertigo worse, but the first and second attacks, years ago, seemed to be relieved by the middle ear inflation treatment. My general health for summer is as usual. I have thought of calling on you again should the vertigo continue, but as I have said, it is mild."

It would be easy to multiply such cases. The following case exemplifies another variety:

**CASE 3.—History.**—H. W., man, aged 35, admitted to Wesley Hospital, Nov. 24, 1913, complained of vertigo and headache, which he has had for three months. The headaches are chiefly frontal. The vertigo is so severe that he is compelled to sit or lie down and hold on. An attack comes on at least once a day, lasts for an hour and is accompanied by nausea and vomiting. During the attack objects apparently go round in a circle (to right?). Up to ten days ago he had difficulty in swallowing and liquid food frequently returned through the nose.

Patient had syphilis three years ago; he has had mercurial treatment for six months, and recently one injection of salvarsan.

**Present Condition.**—The patient's walk is unsteady, but there is no marked ataxia. Romberg is questionable though with eyes shut he sways considerably. During an attack with his eyes shut he falls to the right and slightly back. There is a spontaneous nystagmus on looking to the left. With eyes shut and with palm down his right hand pointed to the right. He touches point of nose and other parts of body with fair accuracy. Slight paralysis of right facial and of right side of soft palate. Taste impaired. Left vocal cord paralyzed. Tongue deviates to left. Pupils react to light and accommodation. Hearing good in both ears and range unimpaired as tested by forks.

Nystagmus: Cold water in right ear for twenty seconds gave marked nystagmus slow to right, lasting three minutes. Vertigo is so marked that with eyes shut the patient feels himself falling and holds on to chair.

Cold water in left ear for forty seconds gave slight nystagmus lasting one minute. After the cold water in left ear he pointed accurately with his right hand, to the left with his left hand.

Both in blood and cerebrospinal fluid, Wassermann was strongly positive.

**Diagnosis and Treatment.**—Basilar syphilis involving the right seventh and right vestibular and right tenth nerves, and left tenth and twelfth nerves. Mercurial injections and inunctions were given.

The vertigo disappeared and the patient left the hospital December 9, improved.

**The Physician as a Sanitary Policeman.**—The present day physician wants to know not only what is ailing his patient, but also how his patient acquired the disease, whether any other persons are exposed to contagion from the same source, whether the patient himself is a danger to his friends and relatives, and whether the disease will spread throughout the entire community or can be confined to the single case in hand. He owes a responsibility not only to his patient but to the community. He is not only the caretaker and medical adviser of the sick. He is also the guardian and protector of the well. Each practicing physician is or should be a sanitary policeman for the protection of the community against contagion.—Frederick R. Green, *Northwest Medicine*.

6. Politzer: Diseases of Ear, New York, 1909, p. 735.

7. Babinski: Ann. de mal. de l'oreille, 1904.

8. Bárány: Lewandowsky's Handbuch der Neurologie, 1910, p. 935;

Tr. Internat. Cong. of Med., Otol. Section, 1913, p. 571.

9. Dixon and Halliburton: Jour. Physiol., 1914, xlviii, 128.