

AGORAPHOBIC VERTIGO

With a Consideration of the Nature of Ménière Vertigo

J. J. LOPEZ-IBOR*

Recently the problem of vertigo has been mainly considered from the neurological point of view. The methods for investigating vertigo due to a lesion in the labyrinth or in its central or peripheral connections have been greatly improved and there is a better understanding of its pathogenesis. On the other hand, the study of a very common form of giddiness or vertigo without any known organic cause has been neglected. There is also a tendency to limit the diagnosis of vertigo of psychic origin to the Ménière forms (mild forms of the true vertigo of Ménière's syndrome). Even if justified from various viewpoints this tendency gives rise, however, to many diagnostic errors. I remember a patient who came to my hospital clinic after his affliction was diagnosed as Ménière vertigo. He had undergone many treatments, including surgical section of the VIIIth cranial nerve without relief. The deep depression he was in was considered a reaction to his illness. A detailed analysis of the case enabled us to demonstrate that his depressive state was primary, not secondary, and that his vertigo was closely associated with the anxiety accompanying his depression. An adequate therapy, concerning which we shall presently discuss, allowed us to solve the problem.

The first to speak of agoraphobic vertigo was Westphal (19). He had observed that the crises overcame the patients on crossing open spaces such as a square. In his original work, Westphal mentions 3 cases and it is interesting to learn that one of these patients suffered from convulsive crises while another had migrainous paresthesia. Westphal was inclined to regard the condition as of psychic origin since he found the cases linked to determining situations. Benedict's opinion (2) that the cause of vertigo could be traced to disturbances in ocular convergence was readily demonstrated to be erroneous. The attention of Cordes was attracted to the presence of other symptoms and some neuropathic disturbances in the patients. Amusingly, he expressed the attraction of the patients to the music of Wagner. Numerous other interpretations arose. Nevertheless the theory considering these ailments purely psychic and the vertigo of open space as another phobia among the other possible ones, gained ground. Freud (7) gives

*From the University of Madrid, Spain.

us a description of the symptoms of anxiety neurosis in an attempt to separate from neurasthenia a certain number of symptoms he groups under anxiety neurosis. Freud describes it in the following way:

"The vertigo of anxiety neurosis is not a rotatory vertigo; it does not arise as the vertigo of Ménière from distinct planes and directions. It is a locomotor vertigo similar to the vertigo due to paralysis of the ocular muscles. It consists of a specific malaise accompanied by sensations such as an undulant floor or heavy lead-like limbs that wobble. This kind of vertigo never causes a fall. I would suggest that such vertigo can also be represented by an attack of profound syncope. Other states simulating a syncope of anxiety neurosis can be due to a cardiac disturbance. An attack of vertigo is frequently accompanied by the worst kind of anxiety commonly combined with cardiac or respiratory disturbances. The vertigo felt when at high altitudes and on looking down precipices is also found in anxiety neurosis. I do not know if the vertigo due to gastric disturbance would also be recognized."

As I have intended to show elsewhere, in the so-called anxiety neurosis there is, in the great majority of cases, a biological factor which constitutes the basis of its symptomatology. This biological factor is similar to the one appearing in melancholia. In the same way that melancholia consists in the presentation of a phase of vital sadness, in the anxiety neurosis a phase of vital anxiety takes place. The word "vital" points to the fact that the origin of the anxiety has to be found not in certain psychic conflicts or in repressed complexes, as the psychoanalyst wants, but in the disturbance of vitality (vital feelings and coenesthesia). Freud himself thought this in his first work on the biological genesis of anxiety, when he said that the morbid anxiety crisis is due to a retention of the libido. Later, he came to establish as his doctrine, a theory on the exclusive psychological origin of anxiety, a theory which I consider untenable.

Considering then that the anxiety present in the great majority of these patients is vital and not psychic, I propose to call this group by the name of "anxiety timopathy," and I mean by timopathy the common denominator for all the affective disturbances.

According to my experience, 53 per cent of the patients diagnosed in the latter group complain of vertigo ("vertiginous crisis"). The symptoms of the anxiety neurosis or better, anxious timopathy, appear in three planes: a) mental, which crystallizes round the anxiety and the phobias; b) neurological, and here the fundamental symptoms are vertigo and headache, and we could also add certain alterations of the body schema, paresthesias, tremors and various algias of which the more frequent ones involve the back of the head and the subscapular

region; and c) visceral, with disturbance of the respiratory, digestive, and other apparatuses.

Presentation of the symptomatology follows as a rule these two norms: i) a frequently phasic character, and ii) interchangeability of symptoms in the different phases.

The neurological projection of this disorder is no less rich than the psychic one. Among the symptoms, vertiginous crisis is the most outstanding. The subject feels that he is falling, that his legs flex, that the floor is giving way, or he has other similar sensations. At times he may not actually experience such sensations but only the dread that they might occur. Sometimes the phenomenon presents itself purely, so to say, with no relation to any special circumstance. The crisis appears suddenly, like a flash of lightning in a quiet night, but at other times it is related to the circumstance of place, as an open square, the summit of a hill, etc.

The interpretation of the crisis of vertigo as equivalent to anxiety has several reasons. It is very important to note in the clinical history the link between the two crises as one supplanting the other, depending on the phase at the time the patient is seen by the physician.

G. A., 59 years old. After the age of 25, the illness started as a sensation of emptiness when he found himself in an open space. When in the midst of a square, for instance, he felt as though he were falling and in order to avoid such a fall he had to follow a circuitous path by the sidewalk, for the neighboring houses seemed to support him.

This symptom subsequently disappeared and he was able to travel and to carry on a normal life. At 28 he was deeply moved by the death of his father and the crisis reappeared, but only when he left the city where he lived, even if only on an excursion to the fields. The sensation that he now had was not one of giddiness, but anxiety. Gradually the sensation became more intense and the patient limited the perimeter of his activity until he could not get further than 100 metres away from his house. On returning home the anxiety disappeared.

At times the similarity between the crisis of anguish and vertigo is manifest because of their respective symptomatological overlap. For instance, a female patient observes that the crisis begins as an alteration in visual perception, "as if the things before my eyes were in a merry-go-round." Suddenly the merry-go-round becomes faster and faster until everything disappears from sight. And then still more abruptly the third and more dramatic act ensues with a feeling of anxiety "as if my heart is being tightened." In some episodes the patient feels transported for a few seconds and on coming back to herself she continues "feeling an oppression in the chest and wanting to cry." Another

patient has frequent bouts of vertigo occurring either during the day or at night. Sometimes she had the crisis even while sitting down. She then stood up and started to run as if going "amuck." Despite the fears of dizziness she never fell down.

Obviously the web of symptoms in these crises is so complicated that at most we can come to an artificial differentiation of one from the other.

Patients speak of giddiness, but if we urge them to describe the sensation with more precision they do not know whether to assign it to the plane of anxiety or to the level of vertigo. At times one can understand them only when they resort to a description of the sensations felt in the head or those felt in the epigastric region (a feeling of emptiness in the stomach). The vegetative symptomatology is found inlaid in the thymopathic vertigo. The patients speak of a vague feeling of malaise, of dread of the break-down, at any moment, of their state of equilibrium, of a vague fear of something going to happen. Together with this state of "emotive drunkenness" (*ivresse émotive*, as Féré described it), there is a sensation of flaccidity and wobbling of the legs, emptiness of the stomach, perspiration of the hands, palpitations, etc. . .

There is no objective disturbance in gait; at most we see that the subject widens the base, or he is reluctant to lengthen his steps. The patient can even be made to walk with his eyes closed, but a sudden violent gesticulation announces the crisis. Some patients dislike walking on a very smooth floor; they prefer one that gives a sensation of stickiness, or firmness. It is interesting to note the relations of these crises with the digestive system. There is no doubt that some of the vertigos described as due to perverted stomach (Trousseau) belong to this type. Many patients feel better after meals. It is possible to explain such well-being as related to postprandial vagotony. In Westphal's first case (19), the patient felt worse in the morning before breakfast, and better after meals or after having ingested a small amount of wine or beer. A hearty meal or excessive drinking, however, provoked or aggravated the crises.

Since vertigo has no relation to the organic regulation of equilibrium, it is not strange that the patient suffering from vertigo can ride a bicycle perfectly. The bicycle acts as a prop or support just like a cane or a companion. In French literature the relation between anxiety and vertigo is recognized to such a point that this syndrome is described as "anxiety of equilibrium."

Chavany says that even though compensatory or inhibitory mus- from true vertigo because in the former the patient never falls. Further- cular reactions are present, agoraphobic vertigo can be differentiated

more it lacks the dominant feature of true vertigo, namely disorientation in space of the body and the objects surrounding it.

This distinction by Chavany (5) is not altogether exact. In thymopathic vertigo (agoraphobic) there can also be an apparent displacement of objects. It is distinguished, however, by the fact that the displacement is more confusing and disorderly. Furthermore the subject is always capable of marking the difference between his own perception of the movement of objects and his judgment that the objects are not in motion, explaining the common expression "as if the objects were moving to and fro." This to and fro motion is never organized.

Some patients are bothered more by this disturbance of visual perception while others suffer from the instability and quaking of the ground that seems to sink at every step. These two groups of sensations can substitute each other for when one predominates the other diminishes. It is not true either, as Chavany says, that the patients never fall during the vertiginous crises. Some patients do fall. These cases span the transition between thymopathic vertigos and vasovagal synopes and even those cases of psychogenic etiology. In some rare cases the crisis of vertigo is accompanied by facial paresthesias, as has been printed by Bonnier. He explained this coexistence as a propagation of the irritation of the VIIIth cranial nerve to the Vth, in accordance with the physiopathologic explanation of vertiginous crises which he established. The paresthesias are more frequent than the algias, as the following example demonstrates:

O.G.G., a 45-year-old veterinarian, of a pyknic build with thymopathic ancestors. For about a year he has been suffering from dizziness that appears mainly on walking. During the last two months it has become more intense. He develops a falling sensation, though he has never fallen. The face becomes red with a sensation of numbness and stretching of the cheeks. He cannot continue walking with the head erect. He has to fix his gaze toward the floor. There is an accompanying feeling of anxiety similar to that felt before an examination. Since the start of the dizziness he has been depressed and fatigued in the mornings but he feels better in the afternoons. He is more easily annoyed and vaguely sad. He never feels dizzy at home even when he walks about for a long time. On the other hand, he easily gets dizzy in the streets. *E.N.T.* as well as physical and neurological examinations are completely negative. There seems to be a slight tactile hyposthesia in the region where he claims numbness. We cannot, however, exclude a factor of psychogenic fixation in this respect.

The vertigo experienced at high altitudes is a phenomenon which can be considered normal. It is interesting to point out that there can be vertigo when one is on a balcony, on top of a tower or overlooking a

precipice and yet one may not feel giddy on peeping out of the window of an airplane. The influence of imagination in the vertigo at high altitudes is obvious. A patient of mine had vertigo inside the theatre when a precipice or the ascent or descent of an airplane was projected, but not when the plane was in uniform flight. In the phobia for elevators we find a combination of a vertiginous experience and claustrophobia.

Thymopathic vertigos are of extraordinary interest clinically. From my point of view they constitute the most frequent form of vertigo and the most common source of error. The problem is so evident and at the same time interesting that in the recent book by Friedman (8) devoted to the study of the functional cardiovascular diseases, vertigo was pointed out as one of the primary symptoms. Vertigo, he explained, is due to cerebral anemia but there are no objective data presented to convince us of the presence of such anemia. On the other hand an undue importance is laid on the abnormality in the size of the pulmonary artery. But these two reasons are not convincing enough. And as we shall see further on, one deals with a chain of symptoms that independently can substitute each other. Judging from the very subjective description of the patient, there is no reason why the subjective cardiac or vertiginous disturbance should be more prominent than anxiety (found in neurocirculatory asthenia).

On studying the physiopathology of thymopathic vertigo we shall examine its relationship with the vertigo of Ménière. Thymopathic vertigo does not absorb the vertigo of Ménière, which has a clearly defined symptomatology. However it does include many of the diagnoses erroneously attributed to the vertigo of Ménière.

The subjective sensations of stretching of the neck, or the nuchal region "as if I cannot sustain my head," "as if my neck would collapse," etc. . . . are probably related to the crises of vertigo. Some patients complain of similar widespread sensations throughout the body "as if it were creeping up," "as if it were creeping down." Studied more closely these sensations are analogous to those experiences confusedly described by the patients when they are getting dizzy.

We could also establish a fine scale in the somatic plane, especially that of muscular restlessness. The patients complain that they cannot keep still, that they have to keep moving or at least shift their legs. They feel uncomfortable in a state of immobility; thus they have to make gestures or grimaces. Some cases make us recall acatisia, even though the present use of this term is limited to the phenomenon of ceaseless activity brought about by certain extrapyramidal affections (postencephalitic parkinsonism and Parkinson's disease). We must

bear in mind that Haskovec used the term for the first time in describing some hysterical personalities which probably belonged to this type.

The feeling of restless limbs was described as "muscular impatience" by Brissaud. One deals with an almost painful need to move, particularly the legs. This appears at bedtime and is relieved only by a change of position. Von Weizsaecker (17, 18), who studied this phenomenon, briefly said that they are generally found among persons of middle or old age who cannot be classified as neurasthenics. Excluding the participation of a vascular factor, he considered the phenomenon to be a consequence of paresthesias of proprioceptive sensibility, perhaps from the muscles.

Experience has taught me that this phenomenon, which is so difficult to classify clinically (even von Weizsaecker refrained from classifying it), belongs to the symptomatology of anxiety thymopathy. It is often found in association with the feeling of "restless expectation," giving the impression that it belongs to the very same syndrome within the general symptomatology of the thymopathies. Muscular restlessness increases indoors, thus making it akin to claustrophobia.

J.M.S.—"At times on taking coffee I feel as if I float in the air. At other times I am simply in an anxious state and I find it difficult to focus my attention on anything. It is similar to my feelings of "restless expectation." At night in bed my legs jerk and I cannot keep them still. Keeping them still seems to produce pain although there is actually no real pain. I feel a drive to move them or to change posture and I am momentarily relieved after satisfying the impulse. The phenomenon presents itself when I lie down, and it lasts for some time, delaying my sleep."

In this case "restless expectation" is a special form of the vertiginous crisis accompanied by anxiety. The muscular restlessness is a secondary but equally disturbing phenomenon. It is a projection of restless anxiety on the lower extremities. I believe that its origin is central rather than peripheral, as von Weizsaecker claims.

The evolution of the concept of Ménière's syndrome is well known. In 1861 Ménière (14) described it as due to hemorrhage in the labyrinth. Its present use includes all paroxysmal diseases of the VIIIth cranial nerve (13). Initially the presence of objective signs (nystagmus, deafness, etc.) were required. Today, disappearance of nystagmus between the attacks is accepted. The absence of cochlear disturbances is not a safe criterion either, for deafness may exist unrecognized unless a careful audiometric measurement is performed. In the audiogram of patients suffering from Ménière's syndrome we find that diminution of hearing in one ear is followed by a decrease in the other. The deaf-

ness is for high tones. Crowe, Thornelle and Azoy insist on the fluctuating character of deafness in these cases. From the original concept of hemorrhage in the labyrinth there was a time, particularly starting with Kobrak, when every vertigo was attributed to angioneurosis. Waltman speaks of an endolymphatic edema. The picture of Ménière's vertigo is, little by little, becoming more ill-defined. The criterion for its diagnosis has loosened. Among the authors responsible for the disjointed scope of this diagnosis, Watkyn-Thomas stands out prominently, considering Ménière's vertigo as a syndrome caused by various factors acting not only on the labyrinth but on its central connections or on its blood supply.

Against this disjointing tendency are the recent studies that have attempted to make the clinical concept of the vertigo of Ménière more precise. Crowe and Wright insist on the possibility of finding certain signs and symptoms of cochlear and vestibular disorders. These objective findings together with the onset and peculiarities of the course, enable us to consider labyrinthine vertigo as a separate entity. Hallpike and Cairns (9) further support these criteria by their histological findings in the temporal bones of 2 cases. In both cases, the affected labyrinth showed marked dilatation of the endolymphatic system. The saccule was dilated to the point of filling up the vestibular cavity. Reissner's membrane was found pushed to the osseous wall, protruding through the helicotrema to the point of obliterating the perilymphatic space. In a third milder case, the necropsy findings were the same although less marked. According to these authors, the anatomic basis of the disease is the obstructive distention of the endolymphatic system. Rollin (16) has obtained similar findings in 6 other cases. From the clinical standpoint, Cawthorne, Fitzgerald and Hallpike (4) have made a detailed investigation of a group of 50 patients suffering from Ménière's vertigo. In these cases they have found evident signs of cochlear deficit. In the majority (86 per cent) of the cases, the deficit was bilateral though more accentuated in one ear than in the other. The deafness was of an internal type. The caloric reactions showed the presence of a vestibular lesion in the great majority of cases (47 of 50), localizing the lesion in the more defective ear in almost all the cases. The types with unilateral lesions found were: paretic lesions of the reticulum (10 cases), paretic lesions of the external canal (29 cases) and combined paretic lesions of the reticulum and external canal (8 cases).

Lyman as well as others expressed the confusion associated with Ménière's syndrome. Originally, the term was applied to those cases in which deafness and intense vertigo appeared paroxysmally. In the course of time the scope of this designation has been expanded up to a

point where it is indistinctly used for vertiginous states of central or peripheral origin. This indiscriminate use rendered the need for a more precise diagnosis unimportant in clinical practice. Yet, the need still exists and the syndrome of Ménière is at present not limited to the pure cases (deafness-vertigo), but includes disturbances in the functions of the labyrinth due to various causes. Those cases of central vertigo, be they organic or functional, are thus excluded. Finally Ménière's syndrome is due to an edema of the labyrinth. In an excellent work, Mygind and Dederding (15) state: "Ménière's syndrome is not an affection sui generis, but the reaction of an ear exposed to the obnoxious effects of various exogenous as well as endogenous factors through vasomotor alterations especially of the capillaries."

Apart from Ménière's vertigo there are many cases diagnosed as ménièriform vertigo, which are vertigos of central origin, and probably due to a dysfunction of the central pathways for the regulation of equilibrium. In this group of central vertigos one should place a) the vertigos that are a sequel of cerebral concussion, and b) the agoraphobic vertigos.

In my opinion, the agoraphobic vertigos are vertigos either of functional character or of central origin, although they may be both when they appear with certain psychic situations. Their character is as follows: According to Hitzig's classification (12) they are nonsystematic vertigos. This author distinguishes two kinds of vertigo: systematic and nonsystematic. In the systematic the false sensation of sliding of space is felt in a definite direction, while in the nonsystematic the subject's relations with space are altered in an undefined and vague way.

If we analyze carefully the patient's descriptions of these states, we find that during the crises the patient pays very little attention to the external space surrounding him. He feels giddy not because he has an erroneous perception of his situation in space. The cause of his giddiness is internal; he feels that something fails and breaks down within him. This is why he often describes this state in the following terms: "I felt as if I were going to fall." It is a feeling of internal uncertainty preceding the external sensation (described "as though everything swayed before me") associated with vegetative symptoms. Schematically we can consider three different parts in a vertiginous crisis: a) altered perception of space, b) disturbance in the subject's normal way of experiencing his being in space, and c) accompanying vegetative symptoms. These three different features can stand out more or less according to the type of vertigo. In organic vertigos the first is more marked; in agoraphobic vertigos it is the second, and in seasickness the third.

Anyone who has felt seasick can tell us that he suffered mainly from the vegetative sensations and not from any disturbance of the perception and feeling of space, which are unaffected except in very severe cases. Of course this division of the vertiginous crisis into three parts is a bit artificial. Nevertheless there are certain grades or features which enable us to make these distinctions. The sensation the subject experiences during the agoraphobic crisis is so ill-defined and vaguely outlined, that the patient himself refers to it as "mental confusion."

Herz uses a similar term when he defines vertigo as a particular state of confusion produced by the perception of a too rapid chain of images, which cannot be separated by the natural interval which should lie between one another. We can state that giddiness in these cases is equivalent to anxiety.

Anxiety can be substituted by giddiness, and vice versa. If we force the patients to dwell more carefully on the analysis of their sensations, the identity of the two symptoms stands out clearly. From their initial description of giddiness they pass on gradually to expressions of their anxiety. Often they include cardiac oppression and wobbly legs.

These sensations are not at the core of such states. If we probe deeper into the analysis we find two expressions which the patients use indistinctly, giving them nearly the same meaning. The patient generally tells us: "I felt as if I were going to fall" or "I felt as if I were going to lose consciousness, to faint." The sensation of the agoraphobic is not at all the same as the one felt in ordinary fainting, in which dizziness appears gradually and there can be a feeling of "well-being."

In agoraphobia this fear of losing consciousness is full of anxiety. In certain instances it can be felt as pure anxiety.

As I have stated in other papers, the root of anxiety is an internal fear of losing consciousness. Consciousness in the patient's language means an awareness of the self (I), of its being there. The very being of the person is menaced by the surging anxiety. During sleep this awareness of the self seems to vanish also, but the similarity is only superficial. During anxiety the patient fears a dissolution of the unity and continuity of the awareness of the self. A feeling of emptiness, of not being there, replaces this continuity.

Whenever a patient is threatened by vertigo, he feels it as a wave of emptiness surging into his head. This is exactly the same experience at the root of anxiety. Subsequently it can appear as vertigo or anxiety, depending on the way the subject develops this primary experience.

In anxiety, the basic experience develops on the psychological level as a "fear of going off one's head," and on the vegetative level as palpitation, precordial oppression, clammy perspiration. The feeling that

one is going to faint, to disappear, that one cannot keep up a moment longer, is ever present.

We shall now analyse how this primary experience develops into vertigo. Giddiness is present, but here we have it expressed in a different form. It disturbs the relations between the subject and the world.

In man we can consider two different forms of space: the psychological space and the physical one. In the latter, we can stand before, behind, to the right or to the left of something. There are still more important ways of being in space, such as floating or resting, under cover or out in the open air.

There always exist an intercourse and coordination between the subject and his world. When these physical relations are altered we have an organic, or a Ménière's, vertigo. It is systematic or nearly systematic and is due to a lack of coordination between the subject and the external physical world.

Let us take the example of a subject placed in a rotating chamber: If the chamber rotates at full speed, or very, very slowly, no giddiness is experienced. Helmholtz observed many years ago that when a subject is rotated very slowly his visual perception of space remains unaffected, but when at full speed, no perception is possible; thus to feel giddy he must turn neither too fast nor too slow. The vertigo appears when there is a crucial state of disequilibrium between the subject and the surrounding space.

Space does not appear before us in a uniform manner. An object placed below another in the same geometrical plane, seems nearer to us than the one above. If we study closely the works of primitive painters we find that they lack real perspective. Perspective is achieved by placing those figures meant to be in the background in the upper part of the painting. Many experiments prove how space is valued and weighed by man in these different manners.

Systematic vertigo is closely related to the perception of the physical circumstances in which the human body is set. We can state as well that agoraphobic vertigo is related to the psychologic circumstances. In the former, what is really important is the situation, at times the crossing of a square, other times on ascending the steps of an altar, or when a small hill is surmounted and the horizon broadens before the subject. It can also appear when the subject goes outdoors without an umbrella or parasol as a protecting roof.

It is erroneous to think we can deduce the genesis of vertigo from its circumstances. There are merely data in the life history of the patient. Psychoanalytic studies endeavour to grasp the primary circumstances which brought about the vertigo, but they will never get to the real

nucleus of this problem because the process is exactly the opposite. A crisis of dizziness appears at a certain moment, without any particular motive or cause. There is no reason for its appearance. Man is always in need of finding out the way of everything that happens to him, especially when the internal events are tainted by anxiety. Thus he tries to attribute his vertigo to the particular circumstances under which it appeared.

When the patient finds himself again in the same circumstances he fears that a new crisis of vertigo might appear. These particular circumstances, which we may call vital circumstances would be the analogue of movement or acceleration in space of organic vertigo.

From the clinical point of view, post-traumatic vertigos resemble more closely the agoraphobic vertigos. When we compare the experiences of the patients in these two cases, we find a certain parallelism, although they are not the same. The psychological and historical circumstances play a more important role in agoraphobia than in post-traumatic vertigo. Therefore we can consider these experiences as only relatively similar.

Post-traumatic giddiness is a vertigo of central origin. It has been well studied by Hebel through the use of the rotating chamber.

When the rotating chamber is stopped abruptly, the endolymph continues moving in the same direction, giving the subject the impression that he is turning to the left. If we then open the window of the chamber, his visual perceptions counterbalance this imaginary sensation. Therefore we can say that these two sensations act a check to each other. The one that leaves the last impression is more predominant. In the aforementioned case, the visual sensations dominate, as they appear on the field of perception when the impressions produced by the labyrinth are fading away.

The phenomena, with their many possible variations, observed in the experiments prove the existence of a fine adaptation and coordination among the different senses. Thus the relations between subject and world (subject and object) are firmly established. Whenever a new situation arises the subject adapts himself to it. This active balance links previous sensations to the present one, forming thus a continuity. Fundamentally this is the vital act (17, 18).

In a healthy person this co-ordination is smoothly achieved; it is almost automatic. In pathological states it is altered. The continuity of the physiological process is only possible when the previous sensation has vanished permitting the new one to act.

When the link between the past and the present is severed, a gap appears, however small this separation between past and present may be.

The mist that gathers before the subject's eyes is produced by defective co-ordination between the endolymphatic movements and the optical images. On the other hand the change from one sensation to the other is achieved with great difficulty, hence the appearance of the pathological crisis. An analysis of automatic muscular co-ordination when vision is blurring, shows it to be disturbed during the crisis.

Hebel places the subject on a scale, with each foot resting on a plate. Then he asks the subject to look at a revolving drum (similar to that used for provoking optokinetic nystagmus). The patient suffers a crisis. The shaking of the legs is registered by the scale. We find a lack of balance on the left or on the right, depending on the direction of the drum's rotation. The more abruptly this crisis of sensorial perception is brought about, the more intense the motor disturbances appear.

According to von Weizsäcker's school, the term "nonsystematic vertigo" means a disturbance in the co-ordination of the various senses. The vague perception cannot be definitely ascribed to one organ or another. The duration of the crisis is a direct expression of the central disturbance.

Agoraphobic vertigo can also be considered as due to a disturbance of the co-ordination between the different senses, caused by a lack of central regulation.

Briefly then, from my point of view, agoraphobic vertigo is not psychogenic but functional. It is linked to an endogenous disturbance of the central control of equilibrium, found in relation to other central regulations that intervene in the crisis of anxiety. I have described in the foregoing the reasons for which this anxiety, because of its phasic character, should be named "vital anxiety" in order to distinguish it from reactive anxiety. In a similar way I propose a somatic therapy for vital anxiety and I consider psychotherapy as a complementary therapy. The same happens to the vertiginous patients. My observations demonstrate the phasic course of this type of vertigo and the good therapeutic results obtained with vegetative shocks through the use of acetylcholine, dilvasene, and analogous preparations. In some cases I have obtained brilliant results with convulsive therapy, as in the first case I mentioned, of a patient who had been previously treated for alleged *ménièreiform* vertigo. Of course, the results achieved by this therapeutic measure are much better than those obtained purely by psychotherapy or with the usual medicaments employed in the vertigos of *Ménière*.

REFERENCES

- (1) Altmann, F., and Fowler, E. P., Jr.: Histological findings in Ménière's symptom complex. *Ann. Otol., Rhin. & Laryng.*, 52: 52-80, 1943.
- (2) Benedict: Cited by Westphal (19, 20).
- (3) Cawthorne, T.: Vestibular injuries. *Proc. Roy. Soc. Med.*, 39: 270-273, 1945.
- (4) Cawthorne, T. E., Fitzgerald, G., and Hallpike, C. S.: Studies in human vestibular function: III. Observations on the clinical features of Ménière's disease: with especial reference to the results of the caloric tests. *Brain*, 65: 161-180, 1942.
- (5) Chavany, J. A.: L'anxiété d'équilibration. *Presse méd.*, 2: 1017-1019, 1941.
- (6) Féré, C.: *La pathologie des émotions. Études physiologique et cliniques*. Paris, F. Alcan, 1892; pp. 1-605.
- (7) Freud, S.: *Gesammelte Schriften*. Bd. I. Wien, Internat. Psychoanal. Verl., 1927-28.
- (8) Friedman, M.: *Functional Cardiovascular Disease*. Baltimore, Williams & Wilkins, 1947.
- (9) Hallpike, C. S., and Cairns, H.: Observations on the pathology of Ménière's syndrome. *J. Laryngol. & Otol.*, 53: 625-655, 1938.
- (10) Hebel, K.: Die Relativität in der Wahrnehmung von Ruhe und Bewegung. *Ztsch. f. Sinnesphysiol.*, 70: 75, 1943.
- (11) —: Experimentelle Untersuchungen zum Verständnis des zentralen Schwindels nach Schädeltrauma. *Deutsche Ztschr. f. Nervenhe.*, 156: 14-63, 1944.
- (12) Hitzig, E.: *Der Schwindel (Vertigo)*. (Herausgegeben und bearbeitet von J. R. Ewald u. R. Wollenberg). Wien u. Leipzig, A. Hölder, 1911.
- (13) Klestadt and Rotter: Ein Beitrag zum spontanen Vorbeizeigen als Kleinhirnsymptom. *Arch. f. Psychiat.*, 84: 93-115, 1928.
- (14) Ménière, P.: Memoire sur des lésions de l'oreille interne donnant lieu à des symptômes de congestion cérébrale apoplectiforme. *Gaz. Méd. Paris*, 16: 597-601, 1861.
- (15) Mygind, S. H., and Dederding, D.: Studies on some cutaneous and subcutaneous phenomena and their relation to labyrinthal alterations in Mb. (Morbus) Ménière. *Acta Otolaryng.*, 13: 474-488, 1929.
- (16) Rollin, H.: Zur Kenntnis des Labyrinthhydrops und des durch ihn bedingten Ménière. *Hals-Nasen-Ohrenarzt*, 31: 73-109, 1940.
- (17) Weizsäcker, V. von: Über Ohr und Nervensystem. *Ztschr. f. d. ges. Neurol. u. Psychiat.*, 165: 132-148, 1939.
- (18) —: *Der Gestaltkreis. Theorie der Einheit von Wahrnehmen und Bewegen*. Leipzig, Thieme, 1940.
- (19) Westphal, C.: Die Agoraphobie, eine neuropathische Erscheinung. *Arch. f. Psychiat.*, 3: 138-161, 1871.
- (20) —: Ueber Platzfurcht. *Arch. f. Psychiat.*, 7: 377-383, 1877.

