Panic Disorder in Otoneurological Experience

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> Abstract: The evaluation of cochleovestibular dysfunction in psychiatric patients often causes a difficult problem in neurootological experience. The authors discuss here the neurobiological basis of panic disorders and cochleovestibular dysfunction. In this multicenter study, we examined 63 patients with vertigo and panic disease. Twenty patients with primary panic disease and consecutive vertigo composed group 1, whereas group 2 comprised 43 vertiginous patients with secondary panic disease. The most interesting question is whether the patients have an organic vestibular lesion, which would explain why vertigo alone is the problematic symptom in these panic patients, whereas in other patients, panic disease can cause other severe symptoms that resemble heart attack, dyspnea, or abdominal crisis. Vertigo has a bidirectional connection with psychiatric disorders. The panic disorder can be superimposed on chronic vertigo, and psychiatric patients with a cochleovestibular lesion have diminished chances for complete recovery. The examination of psychiatric patients with vertigo is very time consuming and requires much more empathy than does examination of vertiginous patients with a normal mental state. Anxiety provokes somatic and behavioral symptoms in most such patients. The treatment of vertigo in panic patients and of the panic disorder in vertiginous patients requires cooperation between neurootologist and psychiatrist.

Key Words: otoneurology: panic disorder; vertigo; vestibular symptoms

The evaluation of cochleovestibular dysfunction in psychiatric patients often causes a difficult problem in neurootological practice. A frequent question in neurootological experience is whether the vertigo of psychiatric patients suffering from panic and anxiety disorders is caused by vestibular dysfunction.

Several possibilities exist in the relationships between psychiatric disorders and otoneurological disease: (1) In dealing with an organic cochleovestibular lesion and a normal mental state, the neurootologist must treat patients according to the cochleovestibular examination results. (2) The main problem in psychiatric patients with normal vestibular function is the psychiatric disease, and so the psychiatrist must treat the patient. (3) Most challenging is the case of a combined lesion: psychiatric disorder and organic cochleovestibular disease. A very interesting question is exactly why vertigo is the main symptom in such patients, whereas panic disease in other patients can cause other severe symptoms resembling heart attack, dyspnea, abdominal crisis. Vestibular neuronitis, benign positional vertigo, and migraine are the most common neurootological conditions, all of which can trigger the anxiety disorder and panic [1].

Several types of these combined problems can be

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observed: (1) organic vestibular dysfunction with consecutive anxiety and panic; (2) established psychiatric disease with a new cochleovestibular lesion; and (3) dizziness due to panic and an independent organic lesion of the vestibular system (e.g., dizziness due to agoraphobia with compensated, previously well-known unilateral loss of function).

This relationship between panic disorder and cochleovestibular dysfunction has been examined from two distinct perspectives: the assessment of vestibular dysfunction in patients with panic disorder, and the evaluation of panic symptomatology in patients with a vestibular disturbance [2]. Subclinical vestibular dysfunction, as identified by clinical tests, may contribute to the phenomenology of panic disorder, particularly to the development of agoraphobia in panic disorder [3].

In previous studies, abnormal posturography scores under the eyes-open phase were related to high anticipatory anxiety, whereas those under the eyes-closed phase were related to phobic avoidance [4]. Many patients with panic disorder have abnormalities in their balance system function as compared with healthy controls [4]. People whose panic symptoms were consistently associated with dizziness reported rates of vertigo higher than those in people with panic unrelated to dizziness and spoke of rates of fainting, agoraphobic behavior, and occupational disability higher than those in either comparison group [5]. A significant correlation was seen between the presence of vestibular symptoms and psychiatric morbidity, which in turn correlated with measures of anxiety, perceived stress, and previous psychiatric illness [6].

An article by Yardley et al. [7] reviewed evidence for three mechanisms whereby psychological factors may aggravate dizziness and retard recovery from balance disorders. A common behavioral response to dizziness is to avoid activities and environments that provoke symptoms; yet, such avoidance deprives affected individuals of the exposure necessary to promote psychological and neurophysiological adaptation. Also, anxiety arousal and hyperventilation may add to, amplify, and disinhibit the somatic symptoms induced by balance disorder. Further, attention and cognitive load may influence the central processing of information required for the perception and control of orientation.

Yardley's findings suggest that dizziness in the community is typically characterized by mild physical disorder accompanied by some psychiatric disturbance. As the combination of minor physical and psychiatric disorders is known to be unusually persistent and handicapping, treatment programs must be provided for dealing with this prevalent syndrome. Perhaps such programs would include a partnership between primarycare and neurootological and psychiatric hospital outpatient clinics with experience and expertise in the diagnosis and management of dizziness and psychiatric disturbance [8].

A review article by Balaban et al. [9] examined neurological bases of links between balance control and anxiety on the basis of neural circuits that are shared by pathways that mediate autonomic control, vestibuloautonomic interactions, and anxiety. The core of this circuitry is a parabrachial nucleus network, consisting of the parabrachial nucleus and its reciprocal relationships with the extended central amygdaloid nucleus, infralimbic cortex, and hypothalamus. Specifically, the parabrachial nucleus is a site of convergence of vestibular information processing and somatic and visceral sensory information processing in pathways that appear to be involved in avoidance conditioning, anxiety, and conditioned fear. Monoaminergic influences on these pathways are potential modulators of both effects of vigilance and anxiety on balance control and the development of anxiety and panic. This neurological schema provides a unifying framework for investigating the neurological bases for comorbidity of balance disorders and anxiety.

Treatment with selective serotonin reuptake inhibitors (SSRI) relieved dizziness in patients with major or minor psychiatric symptoms, including those with peripheral vestibular conditions and migraine headaches. Patients fared far better with SSRI treatment than with treatment with vestibular suppressants or benzodiazepines [10].

PATIENTS AND METHODS

For this study, we selected from six otoneurological departments 63 vertiginous patients with panic disease. A psychiatrist had previously diagnosed these patients' panic disease. The neurootological examination methods were the same in the six departments. In a standardized questionnaire, we asked our patients about their life conditions and about their symptoms of panic and balance disorders.

In every department, we made a detailed case history by standardized questionnaire, pure-tone audiometry, tinnitometry, acousticofacial reflex examination, and brainstem evoked response audiometry. In addition, we performed vestibulospinal tests (Romberg test, Bárány past-pointing test, blind-walking test). Most patients were examined by craniocorpography or posturography, depending on the capabilities of the departments.

In every patient, we performed vestibuloocular reflex examination, pursuit eye movements or optokinetic nystagmus, and a bithermal caloric test by electronystagmography registration. The electronystagmography systems were partially different in the six departments, but the main aspects of analysis were standardized. After data collection and analysis, we divided the patients into two groups retrospectively. Group 1 (N = 20; mean age, 44 years) contained primarily the panic patients with consecutive vertigo, and group 2 (N = 43; mean age, 41 years) contained vertiginous patients with secondary panic disease. In the first group, 75% of patients were female; in the second group, 76.7% of patients were female.

RESULTS

The mean age in both groups was roughly 40 years. Both groups contained many more females than males. In both groups, more patients lived in cities than in rural settings. Of those in group 1, some 65% lived in cities; in group 2, the percentage was 69.7%. Combined psychiatric disorder and cochleovestibular dysfunction occurred more often (55%) in the group of manual laborers than in the white-collar workers (55.8%). We found this fact to apply in both the primarily panic disease group (group 1) and in the primarily vertiginous group (group 2).

More than one-third of the patients listed a family member with a psychiatric disorder, and most reported bad housing conditions. Most patients knew the factor that provoked their panic attacks (Table 1). More than one-half of the patients acknowledged stress and overtaxing situations in their lives.

In everyday practice, we often see the somatic symptoms of anxiety disorder, most important among these being the skin and eye symptoms, cardiopulmonary symptoms, and behavioral changes (i.e., mood swings). During our examination, all these symptoms were observed and scored (from 1 to 5) and then summarized. The results are tabulated in Tables 2 and 3.

In most patients, we observed a characteristic, socalled pessimistic view of their experience. When we told a patient, "Your balance system is normal again," he or she said, "Yes, but vertigo will recur." We often hear this type of response, which signals an anticipatory anxiety disorder.

In both groups, vestibular function was variable, but in all patients it was pathological. Detailed vestibular

Table 1. Provoking Factors of Panic Attacks

Group I (%)	Group 2 (%)
45.0	37.2
70.0	72.1
30.0	41.9
45.0	60.5
15.0	34.9
25.0	14.0
	(%) 45.0 70.0 30.0 45.0 15.0

Table 2. Somatic Symptoms of Anxiety Disorder

Dermographism Dry mucosa, monotone speech Cold sweats Odor due to sweating Opened pupils, tearing eyes, blinking Hyperpnea, sighing Tachycardia Pessimistic view of experience ("Yes, but")

Table 3. Scores of Somatic Anxiety Symptoms

	Group 1	Group 2
Total score (max. 40 points*)	17.6	15.4
Mean value of one symptom	2.2	1.93
Pessimistic view of experience ("Yes, but")	3.4	3.36

Each of eight symptoms (see Table 2) having a possible score of 5 (range, 1-5).

test results in these work groups will be the subject of future studies that we undertake. In brief, most of our patients demonstrated central vestibular dysfunction; this was true of more than one-half of the group 1 patients, in whom central vestibular lesions were present (Table 4).

DISCUSSION

The examination of vertiginous patients with panic disease and panic disease patients experiencing dizziness is sometimes very difficult. The evaluation of psychiatric patients with vertigo is very time consuming and requires much more empathy than does evaluation of vertiginous patients having a normal mental state. Anxiety is associated with somatic and behavioral symptoms in most affected patients. Knowing that panic disease can cause other severe symptoms resembling heart attack, dyspnea, or abdominal crisis, we were curious to learn why vertigo is the main symptom in these patients.

As reported, the complete examination is lengthy and tiring. First, we listen to patients' stories as told in their own words. Sometimes a patient's interpretation is redundant and can be hysterical; thus, we must listen also for the tenor or "mood" of the interpretation. The

Table 4. Vestibular Function Test Results

Patient Group	Peripheral Dysfunction (%)	Central Dysfunction (%)	Combined Dysfunction (%)
Group 1 ($N = 20$)	35	-55	10
Group 2 (N = 43)	37	44	19

next step is to ask in a circumspect fashion about the symptoms of disease. Finally, we must read all of a patient's previous reports to search for an organic lesion: For example, has the patient experienced nystagmus during an attack? Additionally, some patients fear the neurootological examination, and we must convince them to consent.

In most patients, we observe a characteristic, pessimistic opinion of their experience. Even when assured that his or her balance system has returned to normal, a patient is likely to bemoan the "fact" that the vertigo will certainly recur. Such a reply, which is common, signals an anticipatory anxiety disorder.

In our experience, vertigo proved to have a bidirectional connection with psychiatric disorders. The panic disorder can be superimposed on chronic vertigo, and psychiatric patients with a cochleovestibular lesion have a diminished chance for complete recovery. This relationship can be explained as a somatopsychic mechanism by which the vestibular symptoms provoke panic in the patient, who feels that the disease is a lifethreatening catastrophe. Alternatively, the psychosomatic mechanism might operate in such a way that the anxiety and panic increase vestibular responses to positional tests and caloric and rotational provocations. Possibly, a previously compensated and symptom-free vestibular lesion can decompensate owing to panic disorder and thereby cause acute vestibular symptoms.

This bidirectional connection between vertigo and panic disease necessitates parallel treatment. Treatment possibilities include antivertigo drugs and SSRIs, vestibular training, and psychotherapy. Treatment of vertigo in panic patients and in panic disorder of vertiginous patients requires cooperation between neurootologist and psychiatrist.

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