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Vestibular Dysfunction Followed by Panic Disorder with Agoraphobia

The association between vestibular dysfunction and anxiety disorders, including panic disorder, first reported by Pratt and McKenzie (1958), has received renewed attention (Hamlin, 1988; Jacob et al., 1985). It can be explained by a somatopsychic model (Jacob, 1988): panic attacks are triggered by unexplained physiological symptoms, such as dizziness, which vulnerable individuals may misinterpret as indicative of anxiety. This may set in motion a "fear of fear" cycle in which anxiety feeds on itself, culminating in a panic attack (Jacob, 1988). Others (*e.g.*, Clark, 1986) have proposed that this cycle is cognitively mediated, and that some individuals misinterpret certain unexpected physiological sensations as presaging catastrophic outcomes (*e.g.*, a heart attack).

Alternative explanations include a psychosomatic model, in which panic symptoms can produce vestibular dysfunction, and a noncausal model (Jacob, 1988). In support of the former is the finding that increased arousal and hyperventilation can affect parameters of vestibular functioning (Furman et al., 1981; Theunissen et al., 1986). We present here a case of vestibular dysfunction preceding the onset of panic disorder with agoraphobia that supports the somatopsychic model.

Case Report

Mr. K., a 39-year-old married white male sales manager, experienced a sudden onset of disequilibrium while walking, which he likened to "walking on a plush carpet." This initial episode, apparently preceded by a viral infection, was accompanied by a sense of weakness in his legs. He reported no previous psychiatric history and no major psychosocial stressors immediately before his attack.

Several days later, Mr. K. experienced his first panic attack, following sensations of dizziness triggered by leaning back in his office chair. Over the next 6 months, he had four more panic attacks, consisting of dizziness, vertigo, palpitations, paresthesias, dyspnea, diaphoresis, nausea, and fears that he might have a brain tumor, heart attack, or stroke. Between attacks, he reported anticipatory anxiety about future attacks, occasional mild left ear pain, fullness and mild tinnitus in both ears, and dizziness while standing. Following the onset of his imbalance, he developed fear—and in some cases avoidance—of situations in which he felt "trapped," such as parties, theaters, crowded churches, and heavy traffic, a pattern typical for agoraphobics. However, he also experienced anxiety in various situations atypical for agoraphobics (see *Discussion*).

A glucose tolerance test and CT and MRI scans were negative. Vestibular testing revealed normal ocular-motor findings, negative Hallpike's maneuvers, and normal responses to alternate binaural caloric irrigations. Static positional testing, however, revealed a low amplitude left-beating nystagmus in all positions. Rotational testing showed normal gain and phase lead at .05 Hz, but there was a left directional preponderance of 19% (Normal = 2.8% ± 2.6%). These nonspecific and nonlocalizing findings are unequivocal evidence of vestibulo-ocular dysfunction of moderate severity.

Because of his agoraphobic symptoms, he was referred to an outpatient clinic specializing in anxiety disorders and evaluated with a semistructured interview, the Initial Evaluation Form (Mezzich et al., 1981; see Turner et al., 1986, for reliability data). His DSM-III-R diagnosis was panic disorder with agoraphobia (mild avoidance, mild attacks). Both the Initial Evaluation Form and a second evaluation by a psychiatrist (R. G. J.) revealed no preexisting psychopathology. His mean scores of 1.55 and 1.35 on the "when alone" and "when accompanied" subscales of the Mobility Inventory (Chambless et al., 1985) confirmed the presence of mild but clinically significant agoraphobic avoidance (normal mean scores are 1.25 ± .24 and 1.07 ± .08, respectively).

The patient's anxiety symptoms decreased markedly in intensity after he learned that his dizziness was of vestibular origin. For dizziness, he was prescribed diazepam, 2 mg p.r.n., which he took only rarely. During the next 6 months, he experienced no panic attacks and had minimal agoraphobic avoidance. Nevertheless, his disequilibrium did not abate, and he generally avoided vigorous activities such as basketball and dancing.

Discussion

The temporal sequence of Mr. K.'s symptoms implicates vestibular dysfunction and is consistent with the somatopsychic model of panic. Further support for a vestibular origin is the fact that he experienced anxiety in situations characterized by what we have elsewhere called space and motion phobic stimuli (Jacob et al., 1989). We have tentatively separated these stimuli into three categories: a) excessive vestibular stimulation, b) incongruent or complex movements of both the visual surround and self, and c) paucity of visual cues for orientation in space. Regarding the first category, the patient frequently experienced anxiety following abrupt head movements (e.g., during dancing, while exercising, and while changing speed during driving). Regarding the second category, he experienced anxiety in situations characterized by a mismatch between vestibular and visual information (*e.g.*, riding in moving, as opposed to stationery, subway cars and elevators) and by complex movement of both himself and the visual surroundings (*e.g.*, looking at lights at the sides of a tunnel while driving and looking at items on the shelves while walking down a supermarket aisle). This "supermarket syndrome" has been observed previously in patients with vestibular disorders (McCabe, 1978). Regarding the third category, Mr. K. became anxious while looking up at tall buildings, although this symptom may have

been triggered by excessive vestibular stimulation. The stimuli in this category are similar to those eliciting anxiety among the "space-phobic" patients with neurological and vestibular disorders studied by Marks and Bebbington (1976), whose term we have adapted.

Finally, the patient's anxiety diminished substantially when he learned the pathogenesis of his symptoms, although his infrequent use of diazepam is a confounding factor in explaining his improvement. An implication of the cognitive model of panic (Clark, 1986) is that educating patients about the etiology of their attacks should lessen the intensity of their anxiety. Rapee (1987), for example, found that, when panic disorder patients were made to experience unusual physical sensations by inhaling a carbon dioxide/oxygen mixture, those informed of the cause of these sensations reported less severe panic than did uninformed patients. Similarly, providing patients with information regarding the vestibular origin of their dizziness may foster more veridical and thus more benign attributions, thereby alleviating anxiety symptoms.

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