Remission of Agoraphobia without Panic after Surgical Correction of a Cardiac Arrhythmia

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Phobias are psychiatric syndromes of excessive or completely irrational fears, e.g., fear of dogs, driving, enclosed spaces, thunderstorms, etc. The most disabling type of phobia is agoraphobia, which is a generalized fear of clusters of stimuli, typically travel, crowds, and leaving the house alone. Panic attacks, unlike phobias, are intense episodes of sudden, unprovoked fear (i.e., without a stimulus). They too are considered a psychiatric disorder.

The etiology of agoraphobia remains obscure. In recent decades it was proposed that agoraphobia shares a specific relationship with panic attacks, particularly because agoraphobia generally follows the appearance of panic attacks, but also on the basis of genetic studies and response to lactic acid challenge [1,2]. Indeed, whether agoraphobia even exists without panic attacks is being questioned. The American Psychiatric Association's Diagnostic and Statistical Manual [3] suggests that agoraphobia is in fact a fear of being in situations from which escape might be difficult or help unavailable in the event of a panic attack.

If agoraphobia in fact stands in a special relationship to panic attacks, what is the nature of the relationship? Is it cognitive-behavioral as the APA suggests (patients learn to fear situations they believe promote or exacerbate panic), or innate-biological (common genes, common neurochemistry)? We report on a patient who developed classic agoraphobic symptoms following the onset of a supraventricular cardiac arrhythmia. Following surgical correction, the agoraphobia remitted without specific treatment.

APA = American Psychiatric Association

Patient Description

A 52 year old man, married with three children and without a previous psychiatric history, presented at the psychiatric outpatient department, complaining of a decline in his general health. A detailed history revealed that for the past 3 years he had experienced two to three attacks a week. These episodes manifested as palpitations associated with diaphoresis, shortness of breath, chest pain, nausea, light-headedness, and concern that his illness might be fatal. The attacks resolved within 10–15 minutes. He gradually developed agoraphobia, manifested as fear and avoidance of driving, theaters, restaurants, and being alone. After 2.5 years he underwent Holter examination and electrophysiologic study, which revealed paroxysmal supraventricular tachycardia due to reentry at the atrioventricular node. He was also referred to psychiatric consultation. Radiofrequency catheter ablation of the bypass tract was performed, and the attacks ceased immediately. Within 2 months the agoraphobia resolved without any additional treatment.

Comment

This case raises thorny questions about the etiology and psychopathology of agoraphobia. The fact that it follows attacks of tachycardia and anxiety of cardiac origin appears to support cognitive theories of phobiogenesis. Tachycardia secondary to panic of central nervous system origin would hardly share common brain biochemistry or predisposing genes with tachycardia of cardiac origin. In other words, it is the response to the cognitive inferences that patients draw about tachycardia that are responsible for their fears of driving, being alone, etc. So, might propranolol before panic attacks prevent agoraphobia? Could cardiac pacing, without panic (and without a cognitive explanation), induce it? However, the notion that agoraphobia should follow any frightening experience accompanied by tachycardia may be too simplistic. Clinicians familiar with agoraphobia know that the symptoms are quite distinctive. Simple phobics do not complain of difficulties in the middle of three lanes of traffic, in the theater, or when showering with the bathroom door closed. What is common to these situations may be the difficulty of escaping from them quickly. These particular symptoms do not result from every fear-inducing stimulus, even when they are accompanied by tachycardia. The traumatic predisposing event (assault, traffic accident) in post-traumatic stress disorder is also sudden, terrifying, and accompanied by symptoms of peripheral arousal, but it does not lead on to agoraphobia. The cognitive theorist would therefore suggest that agoraphobia will follow only certain kinds of frightening experiences, e.g., those that pose particular risks in crowds (perhaps

feelings of dyspnea) and when alone (a life-threatening arrhythmia).

A related theory, the "peripheral" theory of emotions, suggests that anxiety is actually caused by the cognitive response to peripheral symptoms such as tachycardia and trembling, which are held to be primary [4]. However, this is inconsistent with reports that several biochemical provocations induce quite severe physical symptoms in panic patients, without causing panic [5].

Although our patient's episodes of tachycardia, anxiety, etc., satisfy most criteria for panic attacks, they lack the absence of an "organic" etiology (anxiety during a myocardial infarction or during severe hypoglycemia) or external cause (e.g., terrorist attack). Acute anxiety in these situations is normal, not a panic attack. The onset well into middle age is also more consistent with a medical than with a primarily psychiatric approach.

Until now we were content to assume that the distinctive nature of agoraphobia would ultimately have to be explained in the context of a special relationship – cognitive and/or biochemical – with panic attacks. This patient challenges that assumption.

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