

## Social phobia with sudden onset—Post-panic social phobia?

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Received 19 February 2007; received in revised form 25 June 2007; accepted 3 July 2007

### Abstract

Overlap between social phobia (SP) and panic disorder (PD) has been observed in epidemiological, family, and challenge studies. One possible explanation is that some cases of SP develop as a consequence of a panic attack in a social situation. By definition, these cases of SP have sudden onset. It is hypothesized that patients with SP with sudden onset are more similar to patients with comorbid SP and PD than to patients with SP without sudden onset regarding age of onset, extraversion, and prevalence of anxiety symptoms. One hundred and eighty-two patients with a lifetime diagnosis of PD and/or SP were recruited as part of an etiological study. Patients with SP with sudden onset did, as hypothesized, differ from patients with SP without sudden onset with regard to age of onset and extraversion, but not with regard to symptoms. They did not differ markedly from patients with comorbid SP and PD. The concept of post-panic SP is discussed.

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*Keywords:* Classification; Diagnosis; Comorbidity; Panic attacks; Age of onset; Extraversion; Signs and symptoms

Panic disorder (PD) and social phobia (SP) are common disorders that have a serious impact on the lives of those affected. PD is characterized by recurrent, spontaneous panic attacks, whereas the distinctive features of SP are fear and avoidance of being the focus of attention or being humiliated in social situations. The two disorders are considered separate diagnostic entities and are to a certain degree also treated differently.

Overlap between PD and SP has, however, been observed in epidemiological surveys, family studies, and challenge studies. Large comorbidity between PD and SP has been found in both clinical samples (Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Rodriguez et al., 2004) and epidemiological studies

(Alonso et al., 2004; Goodwin & Hamilton, 2001; Schneier, Johnson, Hornig, Liebowitz, & Weissman, 1992).

Family and twin studies have found significant familial aggregation for PD and SP, respectively (Fyer, Mannuzza, Chapman, Martin, & Klein, 1995; Hettema, Neale, & Kendler, 2001; Stein et al., 1998). Some family studies have also investigated the comorbidity between PD and SP and found indications that the comorbidity between PD and SP may be non-familial or that one of the disorders could be causing the other (Merikangas, Lieb, Wittchen, & Avenevoli, 2003).

Challenge studies using Pentagastrin (McCann, Slate, Geraci, Roscow-Terrill, & Uhde, 1997), 35% CO<sub>2</sub> (Caldirola, Perna, Arancio, Bertani, & Bellodi, 1997; Gorman et al., 1990), hyperventilation (Nardi, Valenca, Nascimento, Mezzasalma, & Zin, 2001) and caffeine (Tancer, Stein, & Uhde, 1994) have found that patients with SP, like patients with PD, have a

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heightened sensitivity to these substances compared to controls, indicating a shared neurobiology in SP and PD. Some of the results, though, have indicated that fewer patients with SP than with PD responded to the substances.

In light of the overlap found in these areas of research, it is relevant to investigate possible reasons for the association between PD and SP.

We propose a possible explanation for the overlap between PD and SP. During diagnostic interviews, some patients with SP have described that the condition begun very suddenly. These patients were able to recall the first time they experienced anxiety symptoms in a social situation very similarly to the way patients with PD most often remember their first panic attack. On the basis of this clinical observation, we suggest a possible route to the development of SP: a sudden unexpected panic attack in a social situation may initiate fear of similar social situations. If so, SP begins very suddenly in these patients.

The idea parallels the common learning theory of agoraphobia with PD, i.e. agoraphobic avoidance is conditioned by panic attacks experienced in agoraphobic situations. Prior studies have suggested the importance of the context in which the first panic attack occurred in the development of subsequent agoraphobic avoidance behavior (Faravelli, Pallanti, Biondi, Paterniti, & Scarpato, 1992; Lelliott, Marks, McNamee, & Tobena, 1989). The unpleasant experience was attributed to the specific type of situation in which it occurred. Furthermore, if a person has experienced an initial panic attack with uncontrollable anxiety symptoms, such as shaking, sweating, and blushing, in a social situation, it may also have triggered a fear of other people noticing the anxiety symptoms.

McNally and Lukach (1992) have found that in a minority of cases, panic attacks could lead to posttraumatic stress disorder (PTSD) with symptoms such as intrusive thoughts and startle response, although at a lower rate than following prototypical traumatic stressors. The subjective experience of a panic attack may have mirrored that of a trauma for a number of reasons.

First, panic attacks have often led to a subjective experience of catastrophe and fear of dying. The severity of PTSD has been thought to be a function of the perceived threat, and patients with PD have certainly often perceived their panic attacks, especially the initial panic attack, as life threatening (McNally & Lukach, 1992).

Second, by definition a panic attack has happened suddenly, “out of nowhere”, which is also the defining feature of SP with sudden onset. Brown and Kulik, who

coined the phrase “flashbulb memories” to refer to the vivid and detailed memories of the personal context for the reception of surprising news, have claimed that in relation to forming strong emotional memories, “the registration of surprise and unexpectedness in the central nervous system is the first step and the sine qua non of all else” (Brown & Kulik, 1977, p. 84).

Third, both encoding and long-term memory are highly affected by the level of arousal present (Bradley, Greenwald, Petry, & Lang, 1992; Cahill & Alkire, 2003). The more arousing or stressing the stimuli, the better the memory of it. Although a panic attack may not have been a traumatic event in the traditional sense of the word, it has involved a surge of autonomic arousal and may therefore in large part be responsible for the vivid memory of the situation in which the patient first experienced a panic attack.

## 1. Purpose and hypotheses

The purpose of this study was firstly to describe the phenomenon of SP with sudden onset. Secondly, it was hypothesized that if SP with sudden onset was post-panic SP it would be more similar to comorbid SP and PD than it would be to SP without sudden onset.

Comorbid SP and PD has unfortunately not been the focus of enough studies to make it possible to draw hypotheses about expected differences between patients with SP and patients with comorbid SP and PD. Instead, hypotheses about differences between the diagnostic categories in the present study were based on prior research indicating areas with robust differences between patients with PD and patients with SP: age of onset, personality, and prevalence of specific anxiety symptoms.

SP typically starts in childhood or during the teenage years, whereas PD typically begins in the early to mid 20s (Regier, Rae, Narrow, Kaelber, & Schatzberg, 1998).

A number of prior studies have used different versions of the NEO personality questionnaire (Costa & McCrae, 1992) to compare the personality of patients with PD or SP with that of controls without anxiety disorders (Cuijpers, van Straten, & Donker, 2005; Katon et al., 1995; Trull & Sher, 1994). These studies have consistently found that patients with both anxiety disorders had higher scores on neuroticism than controls, whereas patients with SP had lower scores on extraversion than controls.

Regarding the prevalence of specific anxiety symptoms, several studies have found differences between PD and SP but as the findings have not been entirely consistent, only findings that have been

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