

A Perspective on Social Anxiety Disorder in Adults

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PERSPECTIVE

A persistent and overwhelming fear of one or more social or performance settings characterises social anxiety, which is a prevalent disorder. Behavioral inhibition is one of the early indications of social anxiety, which can lead to the formation of maladaptive cognitive biases and a specific personality structure (low extraversion and high neuroticism) later in life. Despite the fact that there are various successful psycho- and pharmacological choices, many patients do not gain significantly from these treatments. Above and beyond self-report data, brain and neuroendocrine studies might help unravel the molecular basis of social anxiety and perhaps give signs, or "biomarkers," that may be instructive for early disease detection or treatment response. Social anxiety affects several large-scale brain networks related to emotion, motivation, cognitive control, and self-referential processing. Increased cortisol response and reduced testosterone levels are also signs of social anxiety.

Alterations in connection patterns, such as diminished amygdala-prefrontal coupling, are also linked to these neuroendocrine systems. However, much more research is needed to fully understand the relationships between neuroendocrine function and large-scale brain networks. More contemporary therapeutic procedures are currently being used to supplement traditional well-established therapies for social anxiety disorder (SAD). The goal of this review is to describe the various treatments for SAD and to explain recent evidence-based insights on how to manage this illness. Psychotherapy, particularly cognitive-behavioral therapy (CBT), and medication are recommended as first-line therapies for individuals with SAD in recent guidelines; however there is no obvious advantage of one approach over the other. CBT incorporates traditional procedures like in-person social situations exposure and cognitive therapy, but new modalities and techniques have lately emerged, including third-wave approaches, internet-delivered therapy, virtual reality exposure, and cognitive bias correction. Selective serotonin reuptake inhibitors and serotonin-norepinephrine reuptake inhibitors have also been widely researched and proved to be useful in the treatment of SAD. To treat SAD with poor results, two alternative techniques have been devised. D-cycloserine was used during exposure sessions to help with cognitive bias correction and pharmaceutical augmentation

of psychotherapy. SAD patients now have access to personalised therapy.

CBT and SSRIs can be replaced by innovative tactics such as online psychotherapy and virtual reality exposure. Attention bias adjustment and pharmaceutical augmentation of psychotherapy may see further development and optimization in the future. Adults with social anxiety disorder (SAD) have a tendency to anticipate bad social performance and to think adversely on their performance after a social encounter, according to the cognitive theory of SAD. While several studies with socially anxious adults have shown the involvement of poor performance prediction and post-event rumination in SAD, just a few investigations with children with SAD have been conducted. Successful social problem resolution necessitates both a flexible approach to the issue and the ability to produce relevant and effective solutions. Few researches have looked at social problem solving in the setting of social anxiety, which is surprising. We compared the social problem-solving abilities of 38 people with social anxiety disorder (SAD) to 30 healthy control (HC) participants who had no prior anxiety issues. Participants rated their problem-solving attitudes and talents (problem orientation) before coming up with solutions to hypothetical interpersonal problems from both their own and an objective other's perspective. The effectiveness and relevance of these solutions, as well as the degree to which they were active versus passive, were all coded. SAD participants were more negative in their problem orientation than HC controls.

Furthermore, while there were no general differences in the production of relevant and effective solutions to interpersonal difficulties between SAD and HC individuals, using a personal perspective helped the generation of more active solutions for HC participants. The findings point to new study avenues in social problem solving in social anxiety, as well as implications for intervention. According to age-of-onset data, adolescence is a developmentally sensitive phase for the genesis of the illness, as it is during this time that peer groups become increasingly prominent. Social anxiety in adolescence is linked to significant damage that lasts throughout adulthood. Delivering effective interventions during adolescence has strong potential benefits. However, information on the specific efficacy of existing therapy is sparse. Adults, on the other hand, benefit from treatments with extremely

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precise therapy outcomes. Individual cognitive therapy is one such treatment. The cognitive model of social anxiety presented by Clark and Wells is the foundation for cognitive therapy.

The current study looks at how this adult cognitive model could be used to better understand teenage social anxiety, as well as other adolescent-specific characteristics that must be taken into account. Adolescents' treatment outcomes may be improved if the cognitive model of social anxiety disorder is adopted in a developmentally relevant manner. Fear of positive evaluation (FPE) is described as distress and avoidance of positive feedback in more contemporary descriptions of SAD, such as the Bivalent Fear of Evaluation Model. There is currently no specific evaluation of FPE's incremental validity in distinguishing SAD patients from controls - in addition to its explanatory capacity - because well-controlled laboratory tests with positive and negative social stimuli in this patient group are uncommon. To close this gap, we showed 35 patients with SAD and healthy controls (HCs) short social-evaluative video clips with actors expressing negative, positive, and neutral statements while measuring reactivity on experiential measures (valence, arousal, and approval ratings), as well as facial electromyography and electrocardiography.

In the presence of end-organ damage, hypertensive crises can present with non-specific symptoms as well as identifiable and severe symptoms. Hypertensive emergency, also known as hypertensive crisis with end-organ damage, is characterised by more severe symptoms and can result in irreversible organ damage. As a result, it's critical to conduct a comprehensive workup on any paediatric kid suspected of having a hypertensive emergency while treating the raised blood pressure in a progressive manner. Hypertensive crisis treatment is determined by the existence of end-organ damage and can range from fast-acting intravenous therapy to oral medication in less severe situations. Treatment for such conditions necessitates

a delicate balancing act between gradually lowering blood pressure while avoiding end-organ damage.

Protocols for the treatment of hypertensive crises have been created in specific situations, such as in the presence of endocrinologic neoplasms, monogenic sources of hypertension, renal illnesses, and cardiac disease. Clinicians can now expand their reach of care to emergency situations and assist emergency medical service (EMS) providers in real time thanks to the introduction of telehealth. Furthermore, new drug development and updates on the expanding topic of hypertension in the paediatric population continue to enhance outcomes and efficiency in hypertension diagnosis and management. When there is evidence of end-organ toxicity, children with hypertension should be evaluated for emergency therapy. Extreme hypertension complications can be exceedingly serious, even life-threatening, with long-term consequences. Damage to the central nervous system is the most serious.

Treatment for hypertensive emergencies should focus on reducing blood pressure to the level required to decrease toxicity while avoiding hypoperfusion of important organs. In general, blood pressure decrease in an intensive care unit should be closely monitored, with special emphasis paid to central nervous system, cardiac, and renal function. Intravenous agents are recommended in these situations since they are easier to control blood pressure with. A continuous infusion of nicardipine or sodium nitroprusside is recommended in the absence of certain contraindications. A bolus injection of intravenous labetalol followed by a continuous infusion can also be employed. Oral medications should only be used in cases where end-of-life symptoms are present. Because general paediatricians have limited experience managing hypertension emergencies, it is recommended that patients consult with hypertensive emergency specialists as possible.