Adrenergic Mediation of Dissociative Symptoms in Posttraumatic Stress Disorder

To the Editor: Liu-Barbaro and Stein's recent case report[1] raises the more general question of adrenergic mediation of dissociative reactions among individuals with posttraumatic stress disorder (PTSD). In their report, a combination of sertraline and prazosin produced significant amelioration of both PTSD and dissociative symptoms. Since the authors reviewed the mixed results regarding

successful treatment of dissociative symptoms with selective serotonin reuptake inhibitors, I shall focus on adrenergic mechanisms in this regard.

Southwick and collaborators[2] were able to produce dissociative flashbacks after double-blind administration of intravenous infusions of the α_2-adrenergic antagonist yohimbine among Vietnam veterans with PTSD. In comparison to healthy controls who had little response to yohimbine-induced adrenergic activity, 70% of the PTSD patients experienced panic attacks while 40% reported frank dissociative flashbacks.

On the basis of their findings,[2] I have, for more than 20 years, prescribed the α_2-adrenergic agonists clonidine and guanfacine for PTSD patients who have reported dissociative reactions. Patients whom I select for this treatment consistently report time lapses, sometimes lasting several hours, as did the Ethiopian refugee in the Liu-Barbaro and Stein report.[1] It is also noteworthy that my patients typically report experiencing intense arousal prior to the onset of such dissociative episodes, suggesting that, for these patients, dissociation "kicks in" when their arousal/panic exceeds a certain threshold. I always start my patients on small doses of clonidine to make sure they won't become hypotensive, and they generally achieve complete remission of dissociative symptoms at a clonidine dosage of 0.1 mg 2 to 3 times daily, although I have occasionally had to double this dose. Sometimes, when patients begin to develop tolerance to clonidine, I switch to guanfacine, to which tolerance does not develop, probably because of its longer half-life.

I recognize that I have provided little more than anecdotes about clinical success. However, my clinical experience is consistent with the current case report as well as with the aforementioned rigorous yohimbine infusion data.[2] To my knowledge, the only previous psychobiological theoretical article[3] on the pathophysiology of dissociation has described observations after ketamine infusion and has focused exclusively on glutamatergic neurotransmission. It seems to me that adrenergic mechanisms also deserve attention as a mediator of dissociative symptoms.

References

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