Letters to the Editor

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## Adrenergic Mediation of Dissociative Symptoms in Posttraumatic Stress Disorder

**To the Editor:** Liu-Barbaro and Stein's recent case report<sup>1</sup> 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

## It is illegal to post this copyrighted PDF on any website. successful treatment of dissociative symptoms with selective the current case report as well as with the aforementioned rigorous

serotonin reuptake inhibitors, I shall focus on adrenergic mechanisms in this regard.

Southwick and collaborators<sup>2</sup> were able to produce dissociative flashbacks after double-blind administration of intravenous infusions of the  $\alpha_2$ -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,<sup>2</sup> I have, for more than 20 years, prescribed the a2-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.<sup>1</sup> 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.<sup>2</sup> To my knowledge, the only previous psychobiological theoretical article<sup>3</sup> 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.

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