**Supplemental Text Box 5**

**Is Catatonic Stupor an Expression of Tonic Immobility?**

Some writers have hypothesized that catatonic stupor may be an expression of tonic immobility (for review, see Moskowitz [2004]).1-4 Although catatonic stupor and tonic immobility have numerous behavioral similarities—immobility, decreased vocalization, analgesia, waxy flexibility, and evidence of alertness—no current evidence suggests that catatonic stupor is mediated by the circuits within the amygdala, hypothalamus, or periaqueductal gray (PAG), and the evidence for basal ganglia involvement is increasing.3,5 In addition, the autonomic profile of individuals experiencing catatonic stupor (increases of heart rate, temperature, blood pressure, and oxygen consumption)6,7 is the opposite of that seen in the animal model of tonic immobility (see Supplemental Text Box 3). Two possible explanations exist. Since activation of different areas within the ventrolateral PAG mediate different types of immobility—waxy immobility coupled with bradycardia in one area versus atonic immobility coupled with bradycardia in another8—catatonic stupor could theoretically be mediated by activation of a specific area of the ventrolateral PAG that mediates waxy immobility coupled with a different pattern of autonomic activation. An alternate explanation is that catatonic stupor and tonic immobility involve different pathways to the basal ganglia, with the motor component of catatonic stupor—the immobility and waxy flexibility common to both conditions—being mediated by a final shared pathway in the basal ganglia.

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