Commentary: A “shoot first (with anti-psychotics) and ask questions later” approach is not appropriate for the management of delirium after cardiac surgery

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One of the earlier reports of delirium following cardiac surgery was described in 1964 by Blachy and Starr1 entitled “Post-Cardiotomy Delirium.” Now, delirium remains an important modifier of short- and long-term outcomes in at least 1 in 5 patients after cardiac surgery.2 Still, the mechanisms that underlie this syndrome are relatively poorly understood. Among the many prevailing theories that subsume the onset of delirium are derangements of neurotransmitter systems within the brain.3 Antipsychotics are therefore an attractive class of pharmacologic agents to mitigate the incidence and duration of delirium in the intensive care unit. The MIND-USA trial by Girard and colleagues4 is the most recent trial to examine the use of antipsychotic agents for this purpose (see also the Effect of Intravenous Haloperidol on the Duration of Delirium and Coma in Critically Ill Patients [HOPE-ICU]3 and The Prophylactic Haloperidol Use for Delirium in Intensive Care Unit Patients at High Risk for Delirium [REDUCE]5 trials).

The editorial in this issue of the Journal by Cha and Brown6 is an excellent overview of the relevance of the The Modifying the Impact of Intensive Care Unit–Associated Neurological Dysfunction–USA (MIND–USA) trial to the postoperative cardiac surgical patient. First, antipsychotic medications reduce neither delirium-free, coma-free days nor 90-day mortality risk, specifically in hypoactive delirium, which is highly prevalent among cardiac surgical patients.7 By way of analogy, just as the use of furosemide does not treat acute kidney injury, the use of haloperidol or ziprasidone does not “fix” delirium. Nonetheless, despite published guidelines, the use of antipsychotic medications remains in cardiac surgery.8 Second, best practices with the intensive care unit liberation bundle (ie, “ABCDE[F]” bundle) was a key element in caring for patients enrolled in the MIND–USA study (see also Ali and colleagues9 and Engelman and associates10). Third, Cha and Brown emphasize as essential an understanding of modifyable risk factors (ie, the potential mechanism) that contribute to the onset of postoperative delirium. The idea of “shooting first” with antipsychotics and then searching for reasons why hypoactive delirium has occurred thus can no longer be considered best practice. Here, we agree that regular systematic screening to mitigate modifyable delirium risk factors is needed.

The use of tools at the bedside that are capable of identifying the spectrum of delirium presentation, from hypoactive to hyperactive delirium, needs to be emphasized. Although the Society of Thoracic Surgeons Adult Cardiac Surgery Database does capture rates of delirium, it is limited by its definition of the hyperactive form (“marked by an acute course of illness, confusion, and cerebral excitement”). Arenson and colleagues11 have demonstrated that implementing a systematic delirium screening tool in the cardiovascular intensive care unit (such as the Confusion Assessment Method for the ICU [CAM–ICU] used in the MIND–USA trial) resulted in a significant increase in postoperative delirium after cardiac surgery. As such, the potential underdiagnosis of delirium by the Society of Thoracic

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Central Message
The perioperative team needs to identify and treat the mechanism of delirium, not just treat the symptoms with antipsychotic medications.

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Surgeons Adult Cardiac Surgery Database definition may result in under recognition of end-organ brain dysfunction diagnosis in postoperative cardiac surgical patients. The ability of a systematic screening tool to improve risk prediction of poor outcomes after cardiac surgery, however, requires further study. Whether the institution of an interdisciplinary, multicomponent, nonpharmacologic program will mitigate delirium and its associated risks is a motivating area of inquiry.

References