

Recently, our group reported a health care redesign project for colorectal surgery patients where median LOS was reduced from 4.24 to 3.32 days ($P < .01$). This reduction in LOS was associated with a 17% cost reduction.² Showing incremental improvement even as partial bed days is important in proving benefit from care redesign projects such as the PSH. In busy hospital systems, even a reduction of 0.5 days may result in an afternoon discharge turning into a morning discharge, improving throughput throughout the entire institution.

Because the American Society of Anesthesiologists is embracing the PSH as core to the future of our specialty,³ it is imperative that we publish meaningful literature by providing data supporting this new and expanded role outside the operating room theater. We would ask that all future publications include all outcome data so that practitioners and payers alike can make informed judgments.

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An App to Calculate Bedside Stewart Acid–Base Equations

To the Editor

Dr. Story is to be commended for his recent description¹ of a clinically useful approach to blood gas interpretation that combines the base excess and Stewart models, and that does so using only 4 simple equations. He expresses the hope that readers will try this method for “the next ten patients ... requiring arterial blood gas analysis.”

Because calculating even 4 equations can be daunting in the midst of complex patient management, I have created a calculator app to do this part of the work. It is a Web app that can

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be used on any device (smart phone, tablet, or computer) that is capable of accessing the Internet. To open it, point your Web browser to the following address: www.abgst.altervista.org. It is my hope that the availability of this easy-to-use cognitive aid will encourage practitioners to try Dr. Story’s approach.

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Disruption of Memory Consolidation May Explain Patterns of Memory Better Than Emotion-Induced Retrograde Amnesia in Study by Chen et al

To the Editor

We read with interest the article by Chen et al,¹ which focused on the significant number of patients who did not remember locations where they had been prior to midazolam administration. The authors suggest that one explanation for these memory deficits could be emotion-induced retrograde amnesia. However, we feel a more likely explanation of the presented memory deficits is γ -aminobutyric acid (GABA)-mediated disruption of memory consolidation.

A major determinant of memory formation is memory consolidation, a series of neural processes that occur after memory encoding that strengthen and stabilize memory representations in the long term.² Consolidation processes begin immediately after an event is encoded, but they continue over a longer time course. Consolidation is an especially vulnerable time for memory formation, and processes that interfere with consolidation can result in subsequent forgetting of those memories. Notably, previous work has shown that agonism of GABA receptors interferes with memory consolidation, resulting in impaired memory.³ In the current study, participants were given the GABA agonist midazolam, which may have interfered with the consolidation of memories of events in the preoperative holding area and operating room (OR) experienced before midazolam administration. The presented pattern of memory deficits is consistent with GABA-mediated disruption of memory consolidation because patients who had more time to consolidate memories of the locations where they were given midazolam were subsequently more likely to remember those locations. The authors show that 24.9% of patients remembered the preoperative holding area, where they may have spent hours awaiting surgery, at Washington University in St Louis and the University of Chicago (sites where midazolam was given in that location). However, only 15.4% of patients remembered the OR, where patients typically only spend minutes before the induction of anesthesia, at the University of Manitoba (where midazolam was only administered in the OR). The

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disruption of the consolidation process was more critical for information that was weakly encoded (ie, the operating room) than for information that had the chance to be more strongly encoded (ie, the preoperative holding area).

We further support a mechanism of GABA-mediated disruption of memory consolidation over emotion-induced retrograde amnesia because previous research has shown that emotional events can actually enhance memory consolidation.² Finally, and perhaps most important, because consolidation processes can vary depending on the content of memories (ie, the patients' interactions with their anesthesiologists versus details of a location), it is unknown whether patients would have difficulty remembering their anesthesiologists simply because they did not remember the preoperative holding area. As suggested by Chen et al, more research regarding memory and the perioperative period is needed to appreciate the implications that retrograde amnesia may have on patient compliance and satisfaction.

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In Response

Midazolam Is Unlikely to Be Responsible for Retrograde Amnesia

We thank Dr McHugh and colleagues for their interest in our article and for their erudite letter in which they hypothesize that γ -aminobutyric acid (GABA)-mediated disruption of memory consolidation, rather than emotion-induced retrograde amnesia, might account for few patients in our study who did not remember being in the preoperative holding area before the surgery. Before addressing this question directly, it is important to emphasize that according to the results of our study, most patients (~84%) did in fact remember being in the preoperative holding area. Therefore, even if memory consolidation was disrupted for whatever reason, our study suggests that this was relatively uncommon.

All anesthetic and sedative drugs, including midazolam, impair memory formation after their administration, albeit by different mechanisms. Although there are isolated reports of GABAergic drugs, including benzodiazepines, being associated with transient global amnesia, a rare syndrome of uncertain etiology, systematic investigations have consistently failed to demonstrate any retrograde memory effect in humans. These studies assess memory for words or images presented immediately before administration of the GABAergic drug, and they have been performed in the clinical setting with midazolam, propofol, and barbiturates, with pediatric patients, and in controlled studies using human volunteers.¹ In a relevant small randomized controlled trial, midazolam administered before surgery was not associated with even immediate (up until the time of drug administration) retrograde amnesia.² In fact, rather than causing retrograde amnesia, benzodiazepines have been demonstrated to cause enhancement of memory for material presented immediately before drug administration, a phenomenon termed retrograde facilitation. The mechanistic explanation is that benzodiazepines block the induction of new consolidation sequences, protecting recent, partially consolidated memories from the competitive interference that normally occurs.³ The GABAergic drugs appear to have no discernible effect on ongoing consolidation processes in humans. Finally, with recent data showing 5.6% of the US adult population (>13,000,000 individuals) filling at least one prescription for benzodiazepines annually, any common effects on retrograde memory should have been readily detected and reported.

Our study was not able to assess the basis for possible retrograde amnesia, and the effects of emotion, dissociation, and divided attention represent no more than possibilities that require further investigation. However, we think it is unlikely that retrograde amnesia resulting from midazolam administration plays a significant role in explaining the observation.

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