

who received certification before 2000 to participate in its Maintenance of Certification in Anesthesiology process. She suggests that this approach may contribute to skepticism related to maintenance of life-long learning and the board certification process. I would suggest that clinical competency is primarily an assessment best performed locally in health facilities and systems. Although board certification and participation in maintenance of certification processes provide local facilities and systems with valuable information and help assure them that participants have met minimal but important requirements related to medical knowledge, performance reporting, and licensure, these processes do not necessarily reflect clinical competence. The ABA and its parent organization, the American Board of Medical Specialties (ABMS), have appropriately recognized during the past several decades that diplomates who were issued nontime-limited board certificates should not retroactively have time restrictions placed on them. Instead, the ABA and other ABMS member boards have encouraged voluntary participation in maintenance of certification processes for their diplomates who hold nontime-limited certificates. It is a decision best left to local health facilities and systems as to whether they wish to require participation in maintenance of certification processes by physicians who hold nontime-limited certificates as one measure within a continuum of methods of evaluating clinical competence.

Eger provocatively suggests that one potential factor associated with the findings of Tessler *et al.*¹ may be that older anesthesiologists as a population are generally less competent physicians than younger anesthesiologists. Given the context, it appears that he is directing his comments at Canadian anesthesiologists. However, I presume he means to suggest that his postulated factor applies to the United States as well, since he quotes John Lundy, formerly of my own institution. Interesting thought, yet clearly not provable or disprovable. What we do know is that pass rates of ABA written exams during the 50-yr period of 1960–2010 do not vary significantly. These exams do not measure clinical competence, but they do reflect minimal knowledge acceptable to the ABA for physicians who took these examinations annually during this period. Whether or not this information would alter Eger's thinking rests solely with Eger.

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(Accepted for publication July 20, 2012.)

Unresponsiveness versus Unconsciousness

To the Editor:

The review article¹ writes about connectedness as an aspect of anesthesia. This is a major insight into what we mean by the term anesthesia. In 1986, I had defined anesthesia as paralysis defined as nonmovement, attenuation of the stress response, and unconsciousness.² Unconsciousness I divided into amnesia and hypnosis, consistent with the classic terms used for anesthesia. The article was written as a protest to the arguments as to what is a real anesthetic at that time. Connectedness better defines a problem that I described in that only amnesia was typically used to prove that we had unconsciousness. The review clearly shows that amnesia should not be our only goal. Like most good theories, connectedness opens the door to more questions. Connected to what?

If we cut a nerve, it will respond. Is connectedness lost if it never gets past the spinal nerves, as in spinal anesthesia? If the impulse gets through the spine, where in the brain must it be unconnected for us to consider that we have fulfilled the criteria of our job as anesthesiologists? From chronic pain patients, there is a general understanding that a memory exists in nerve tissue, not just the higher centers. Should our goal be to make all nerve tissue unresponsive?

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(Accepted for publication July 27, 2012.)

In Reply:

We are grateful to Dr. Pinsker for his complimentary letter and also to the insightful question posed. We specifically use the term “connectedness” to define the potential that an external stimulus will trigger an experience.¹ All experience is primarily internally generated; it may be independent of external events (such as a dream), or triggered – or modified – by external stimuli. We use “connectedness” to imply a connection between the patient's internally manufactured consciousness and the environment so that an external event may trigger an experience. We primarily have focused our discussion on general anesthesia because of the widespread assumption that these subjects should be unconscious. We propose that, at a minimum, they should be disconnected and thus unaware of surgery.¹ However, we agree there are multiple ways by which

Supported by a Director's Pioneer Award (to Dr. Tononi) from the National Institutes of Health (Bethesda, Maryland).

connectedness to different stimuli may be manipulated. This may include spinal anesthesia to negate the pain of peripheral surgery. Of course, this may be insufficient as, of the multiple causes for distress under anesthesia, pain is not the commonest.² Similarly, we have proposed that reinforcement of a “thalamic” gate may be helpful, but because activation of the cortex is common during surgery,¹ we propose that targeting connectedness at a cortical level (perhaps a final pathway for all stimuli) may prove the most beneficial. We also agree that if a signal enters the central nervous system, this may leave a trace, as discussed in many studies of implicit learning.^{3,4} We do not define this as connectedness (unless there is a change in the subject’s conscious experience), but acknowledge that this remains an important area of research and are grateful for Dr. Pinsker for highlighting this.

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(Accepted for publication July 27, 2012.)

Use of Risperidone in Cardiac Surgery Patients with Subsyndromal Delirium

To the Editor:

We read with great interest the recent study by Hakim *et al.*, which presented data suggesting that the use of risperidone in cardiac surgery patients with symptoms of subsyndromal delirium may prevent the progression to delirium.¹ We believe additional information is necessary to interpret the clinical significance of their observations.

Screenings for subsyndromal delirium occurred every 8 h in the intensive care unit, with the initial screening 4 h after extubation. The timing of development of subsyndromal delirium is important, as symptoms developing shortly after extubation may be because of residual anesthetics (particularly benzodiazepines and narcotics used in the operating room), and treatment with antipsychotic agents at this point may not have been prudent. Indeed, this point is particularly important with the observation that neither

intensive care unit length of stay, nor the duration of clinical delirium, was significantly shortened in the risperidone group. It would also be helpful to know if the intensive care unit and ward in which the study was conducted already practiced risk-factor management techniques shown to decrease delirium incidence in hospitalized elderly patients.²

Although prevention of postoperative delirium may be important, it is also important to distinguish between symptoms directly related to residual anesthetics that would improve on their own, and those that require antipsychotic therapy.

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(Accepted for publication August 14, 2012.)

In Reply:

I would like to thank Raiten and Gutsche for their thoughtful comments on our article.¹

Despite uncertainty on the clinical course of subsyndromal delirium (SSD), there is evidence that it may herald manifest delirium.^{2,3} Nevertheless, although SSD may be viewed as a penumbra between normal mentation and clinical delirium,⁴ the transition from a normal mental status to SSD may not be distinct temporally and is often missed clinically. Accordingly, we presumed that early identification and treatment of SSD might halt its progression to full-fledged delirium. In other words, targeted delirium prophylaxis was actually the main theme of our trial, which aimed at rationalizing pharmacological prophylaxis by offering it to those at assumedly particular risk for delirium. This approach stands in contradistinction to the conventional pharmacological approach to prophylaxis, which counts on the arbitrary commencement of antipsychotic prophylaxis just before⁵ or shortly after⁶ surgery. In this respect, we believe that a fundamental implication for targeted prophylaxis is that administration of antipsychotics should be commenced as early at the inception of SSD as possible. To achieve this, we started to screen our patients for SSD as soon as they were deemed eligible for assessment using our screening tool, the Intensive Care Delirium Screening Checklist. The time frame of 4 h after extubation, however, may not be regarded as premature in view of the prevailing practice of having the patients extubated within 12 h of surgery unless otherwise indicated,⁷ which is also the regular