Letters to the Editor

Cardiac Arrest During Neuraxial Anesthesia: Are All Databases Comparable?

To the Editor:

In a recent article, Kopp et al. (1) claim the following: “The survival rate among these patients was similar to the overall rate for our series and suggests that neither the patient population nor the neurological outcome of patients included in the ASA Closed Claims Project is representative of those who arrest during neuraxial block.” This makes little sense because:

1. The Closed Claims Database (CCDB) (2,3) is 60 times larger than Kopp’s.
2. The outcome at the Mayo Clinic should be better than at the varied hospitals represented in the CCDB, where fewer resources are available compared with a tertiary care facility.
3. The distribution of serious outcomes in the CCDB does not necessarily match those from a single institution’s sample.
4. Because the CCDB arose from malpractice claims, there can be no meaningful statistical comparisons with the Mayo Clinic’s, where cases not involving a malpractice claim are equally likely to be included as those that do.

If all cardiac arrests owing to neuraxial anesthesia occurred in a hospital like the Mayo Clinic, the outcomes might be more favorable than reported by the CCDB. However, the CCDB appears sound and is probably more representative of what happens in the general population.

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References

Is Local Anesthetic Overdose the Most Common Cause of Neuraxial Cardiac Arrest?

To the Editor:

Kopp et al. (1) should provide more details which could help to prevent or more effectively treat cardiac arrest after neuraxial anesthesia. Preventing these arrests may require smaller doses of the local anesthetic. Many patients received excessive doses of local anesthetic before their arrests (40 mg of spinal tetracaine, 840 mg of chloroprocaine for caudal anesthesia, and 610 mg epidural lidocaine). Because smaller doses are recommended for elderly patients and for lower body procedures, there were a number of other overdoses in this series, including overdoses from a mixture of procaine and tetracaine for spinal anesthesia. A similar combination (Neocaine) was available in the 1930s, and larger overdoses were utilized in half of the deaths in this early series (2).

We also need to learn more from the resuscitations. Why did 50% of the patients that received epinephrine die? Was epinephrine given too late, or are these failures related to epinephrine itself (3)? Successful resuscitation may require a combination of intravascular fluids, vasopressor, and vagolytics. Were patients hypovolemic at the time of arrest? Was atropine associated with a more frequent rate of successful resuscitations than epinephrine?

Could more of the arrests be related to the neuraxial anesthetic? For example, when an elderly patient had a cardiac arrest after receiving 40 mg of spinal tetracaine was this at least partially related to anesthetic overdose? Much of this information could be provided by including in the response letter a summary like Table 5 from Biboulet et al. (4). Providing this information could highlight the need for more limited dosing of the local anesthetic, emphasize the importance of adequate fluid replacement, or help confirm or refute the value of atropine in this setting. We must learn all that we can from a series of this size and improve on current practice if we want to reduce the current 25% to 30% mortality rate from cardiac arrest after neuraxial anesthesia.

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References

Bradycardia and Asystole During Neuraxial Anesthesia

To the Editor:

Kopp et al.’s recent study (1) adds to the body of evidence (1–5) that proves that respiratory depression, deep sedation, high block, and high vagal tone are not prerequisites for brady cardiac arrest during neuraxial anesthesia. The remaining confounding aspect of the problem is the suddenness of onset. This suddenness poses the greatest hazard to patients and the greatest challenge to the clinician. As Caplan put it (6), “all of these cardiac arrests seemed to evolve with unexpected speed....”

In a letter, Brown et al. (7) claimed that it was “sudden recognition” rather than sudden bradycardia that was the problem. Subsequent case reports, however, even one co-authored by Brown et al. (5), included documentation, with rhythm strips (2), of truly abrupt severe bradycardia and asystole occurring in seconds, even describing cases of sudden unconsciousness while the patient was conversing with their anesthesiologist (2–3). Not all cases occur abruptly but some certainly do.

I would suggest that loss of compensatory vasoconstriction is a feature frequently neglected in the practice of spinal and epidural anesthesia. This compensation that we are so used to taking advantage of during ordinary states of bradycardia or hypovolemia is absent during neuraxial anesthesia. Thus, hypotension occurs at higher heart rates than ordinarily expected, even as high as the 50s, even in healthy young men with low baseline heart rates. My experience with spinal anesthesia is that when BP begins to drop, the rate of the usual HR slowing begins to accelerate. At that point,
progression to bradycardic arrest with pulselessness and unconsciousness may progress with a rapidity on the order of seconds.

Unfortunately, few of the authors of recent studies have addressed the issue of the hemodynamic pattern in the 60–120 s preceding bradycardic arrest/asytole. Even Lesser et al. (8), who published an account of their cases as recorded by automated anesthesia record keepers, did not provide the above information. Granted, this would be difficult without continuous invasive arterial blood pressure monitoring.

The focus needs to shift to detailed analysis of the hemodynamics in the minute or two leading up to bradycardic arrest and asystole during neuraxial anesthesia. This is a time frame more than adequate in which to intervene to prevent calamities.

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References

In Response:

We would like to thank Drs. Lambert, Pollard, and Stemp for their interest in our recent article (1). It is important to note that the aim of our study was to evaluate the association of preexisting medical conditions and peri-arrest events with survival after cardiac arrest during neuraxial anesthesia. Our series, which includes only the cases of cardiac arrest and does not characterize the ~35,000 patients who uneventfully received spinal or epidural block during the study period, is not able to determine factors associated with the risk of cardiac arrest itself. Thus, we are unable to definitively determine whether a smaller dose or lower sensory level would decrease the frequency of cardiac arrest during neuraxial anesthesia, as proposed by Dr. Pollard. However, Dr. Pollard’s hypothesis is not supported by the 82% survival rate among patients who had a sensory level of T6 or above, which is higher than our overall survival rate of 65%.

Cardiac arrest during neuraxial block has often been described as sudden and without predisposing factors. After reviewing 14 cases of cardiac arrest during spinal anesthesia in healthy patients, Caplan et al. (2) concluded “spinal blockade—conducted under routine conditions and in a standard manner—carries a poorly understood potential for unexpected cardiac arrest and severe brain injury.” Both Drs. Pollard and Stemp reflect this concern and discuss possible predisposing factors to cardiac arrest during neuraxial block, specifically the combined effects of loss of compensatory vasoconstriction, hypotension, and bradycardia. Once again, this is speculative. Despite ongoing and comprehensive reviews of cardiac arrests associated with neuraxial block, the precise mechanism(s) remain undefined and continue to be classified simply as “cardiovascular” in origin (2,3). Furthermore, preventive measures that facilitate early detection of hypotension and bradycardia as well as resuscitative techniques that reverse these hemodynamic effects do not guarantee a good outcome (3).

Unlike cases included in the American Society of Anesthesiologists Closed Claims Project (2), in over half of the patients in our series (1), the primary cause of the cardiac arrest was a specific surgical event (12 patients), preexisting cardiac condition (2 patients), vagally mediated response (2 patients), or sedation leading to respiratory depression (3 patients). In the remaining 10 of 26 (39%) patients, no other etiology was identified and the precipitating cause of the cardiac arrest was assumed to be the neuraxial block. However, it is equally important to note that the etiology of the cardiac arrest during general anesthesia is often unknown. Sprung et al. (4) attributed a “cardiac cause” as the probable source of cardiac arrest in 98 of 223 (44%) patients who arrested during noncardiac surgery; 207 of the 223 arrests were associated with a general anesthetic. Therefore, the primary cause of cardiac arrest during either neuraxial or general anesthesia is “cardiovascular” in approximately 40% of cases (and the contributing factors/mechanisms remain elusive).

Drs. Pollard and Stemp also request more details regarding the peri-arrest events and resuscitation of these patients. Much of the information requested is included in Tables 2–5. However, our study also provided these data for the patients who arrested during general anesthesia while undergoing similar surgical procedures. We point out that comparing the predisposing factors associated with survival for arrests that occur under equivalent circumstances (but different anesthetics) are the most critical results. In our series, patients who arrested under general anesthesia were more likely to have experienced hypotension than those who arrested during neuraxial block. The presenting cardiac rhythm and resuscitative efforts did not vary between groups. Importantly, cardiac arrest that occurred during neuraxial anesthesia was associated with a likelihood of survival equal to or better than an arrest during general anesthesia. We cannot determine why 50% of patients who received epinephrine did not survive despite prompt and aggressive interventions, although this has also been noted in other series (3). Autopsy diagnoses (severe coronary artery disease, massive fat, or amniotic fluid embolism) support the difficulty encountered during resuscitation.

Finally, Dr. Lambert states that successful resuscitation may not be possible in other (non-academic) centers. Existing data indirectly support this hypothesis. In their series, Sprung et al. (4) reported that arrests during nonregular working hours had worse outcomes, indicating that availability of personnel may influence survival. Likewise, the ASA Closed Claims Database continues to accrue cases of cardiac arrest that occur in unrecommended settings (obstetric ward) and/or the diagnosis is delayed (3). Although much remains to be illuminated, the available data suggest several recurring principles: cardiac arrest during neuraxial anesthesia may not always be preventable but, if promptly recognized, it is often treatable (1,3). Thus, perioperative monitoring and vigilance remain crucial to patients undergoing neuraxial anesthesia (3).

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References

Possible Air Embolism During Eye Surgery

To the Editor:

The case report “Possible Air Embolism During Eye Surgery” (1) appears on the cover index as “Severe Eye Embolism During Eye Surgery” and the title is misleading. Air can embolize to any organ system, not just the eye, and eye surgery carries the potential for air embolism. The case report “Possible Air Embolism During Eye Surgery” (1) has been reviewed and accepted for publication in the American Society of Anesthesiologists Closed Claims Project (2), which confirms the hypothesis that air embolism is a significant hazard during eye surgery. The authors have been informed of the error and have been asked to submit a corrected version of the title and abstract for the next issue of the journal. The corrected version of the title and abstract will be published in the next issue of the journal. The authors have also been asked to submit a corrected version of the article for the next issue of the journal. The corrected version of the article will be published in the next issue of the journal.

References

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