Sudden Cardiac Arrest during Epidural Anesthesia: Venous Air Embolism?

To the Editor:—We read the report by Gild and Crilley with interest. While agreeing with their conclusion that this may be a case of unexplained cardiac arrest during epidural anesthesia, we think that a more likely explanation is that the patient suffered an acute venous air embolism that produced the cardiac arrest.

Venous air embolism occurs when there is open access to the venous circulation above the level of the heart, creating a pressure gradient leading to the right atrium. Although this is most often associated with intracranial surgery with the patient in the sitting position, it also has been described during radical hysterectomy and hip arthroplasty.

In the situation described by Gild and Crilley, we postulate that air was entrained through the bone marrow sinuses from the bone-harvesting entry point, which we presume was above the level of the right atrium. In addition, spontaneous respiration creates a negative intrathoracic pressure, which would have increased the pressure gradient and the possibility of air embolism.

The first line of treatment of venous air embolism is prevention of further air entrainment by closing the entry point or increasing the venous pressure. In this case, this was achieved by turning the patient onto her back and administering positive pressure ventilation and cardiac massage. Her quick response to these measures and the absence of subsequent cardiovascular sequelae (indicating that she had no cardiac pathology) may also corroborate our contention that the cardiac arrest was due to an acute temporary event such as venous air embolism.

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How Best to Monitor for Detection of Myocardial Ischemia?

To the Editor:—In their previous studies, Slogoff and Kears have provided convincing evidence that in patients having coronary artery bypass surgery, myocardial ischemia prior to cardiopulmonary bypass is associated with increased risk of postoperative myocardial infarction. In their recent report comparing different electrocardiographic (ECG) systems for detection of myocardial ischemia, Slogoff et al. suggest that ECG monitoring with narrower-bandwidth filters provides higher sensitivity without loss of specificity in detecting myocardial ischemia, as compared with a standard diagnostic ECG.

We do not believe that their data support their conclusions. Their data show that, in patients known to be at risk for myocardial ischemia, ST-segment depression on the Spacelabs monitor in monitor mode (SL-MON) (0.5 Hz) was consistently more negative than on a standard ECG (0.05 Hz). The assumption that all 1.0-mm ST-segment depression observed on the SL-MON system represented myocardial ischemia requires confirmation with an independent measure of ischemia. Indeed, the authors noted that the SL-MON displayed up to 0.25-mm ST-segment depression at all heart rates from a simulator-generated isoelectric ST-segment signal. By definition, these ECG changes are artifactual, yet the authors conclude that the ECG changes detected using the SL-MON system were “not artifactual since specificity of the observation remains high.” However, none of the data in the current or previous studies allows calculation of the specificity of the SL-MON system. By recommending the SL-MON system for diagnosing intraoperative ischemia, the authors neglect the dangers of false positive or artifactual results.

The authors' implication that the SL-MON system may be appropriate, with “cautious interpretation,” for detecting myocardial ischemia in patients without coronary artery disease (CAD) is especially troubling. It is easy to envision the iatrogenic cascade of costs and complications when a patient is erroneously labeled as having had intraoperative myocardial ischemia and arrives in the recovery room with a nitroglycerin infusion, receives a cardiac consultation and overnight stay in the coronary care unit, and so forth. It is surprising that Dr. Kears, an outspoken critic of monitoring without demonstration of efficacy, has concurred in the recommendation that the SL-MON-type system replace standard diagnostic-bandwidth ECG for patients with known CAD in the operating room. We continue to believe that standard (0.05 Hz) ECG filtering in the operating room is more appropriate for detection of myocardial ischemia in all patients, and we encourage manufacturers to incorporate diagnostic-mode ECG as the default setting in operating room ECG monitors.

Richard A. Steinbrook, M.D.
Donald B. Goldman, B.S., C.B.E.T.
Jonathan B. Mark, M.D.
James H. Philip, M.D.
Daniel B. Raemer, Ph.D.
Department of Anesthesia
Brigham and Women's Hospital
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In Reply.—We appreciate the opportunity to clarify some apparent confusion between what we demonstrated and what we then speculated. We demonstrated that in a population of patients with documented coronary artery disease, myocardial ischemia occurring before cardiopulmonary bypass (diagnosed as ≥1.0 mm of ST segment depression on a narrow-bandwidth SL-MON ECG system) was associated with an increased risk of postoperative myocardial infarction.¹² We next demonstrated that the ST segment recorded by the SL-MON narrow-bandwidth ECG system was consistently more negative than the standard diagnostic-bandwidth ECG (ECG-100) and as a consequence led to a diagnosis of myocardial ischemia twice as frequently as did the ECG-100.³ These observations were supported by abundant data.

From these we speculated that myocardial ischemia diagnosed by the narrow-bandwidth SL-MON system had high sensitivity (more frequent diagnosis of ischemia) and high specificity, because: 1) the incidence of ST-segment displacement was directly related to heart rate; 2) ST-segment displacement responded to treatment by propranolol or nitroglycerin; 3) ST-segment displacement was a significant independent predictor of postoperative myocardial infarction; and 4) the magnitude of ST depression (including 1.0 mm) was directly related to incidence of postoperative myocardial infarction. We claim high specificity on this basis because there is no independent measure of myocardial ischemia applicable to this clinical situation for use as a “gold standard”. Neither wall motion abnormalities nor coronary sinus lactate extraction possesses high specificity for myocardial ischemia. Without some “absolute” measure of ischemia, specificity of any of these indices cannot be calculated, but must be inferred.

We then speculated that the ECG-100 system and ≥1.0-mm ST depression criterion, standards adopted for primary diagnosis of ischemic heart disease by exercise testing, may be too stringent for the ECG diagnosis of myocardial ischemia in patients with documented coronary artery disease in the perioperative setting. If tested, a criterion of less than 1.0-mm ST depression using either the ECG-100 or the SL-MON in these patients and this setting may prove to be diagnostic of myocardial ischemia with high specificity by the specificity criteria listed above.

Conversely, we also speculated that when an ECG system with the narrow bandwidth of SL-MON is used for the diagnosis of myocardial ischemia in patients not known to have coronary artery disease (as reported for patients undergoing cesarean section), displacements of ≥1.0 mm may not be diagnostic of myocardial ischemia (false positive) because of all the physical and patient factors influencing the position of the ST segment described in our discussion.³ These factors will always generate some unpredictable false positive results in some patients at some time.

Finally, we did not recommend that the SL-MON replace the ECG-100 in operating rooms. We merely noted that the reason for a narrow-bandwidth ECG in the operating rooms still existed (electrical noise), and when used for patients with documented coronary artery disease, high specificity for the diagnosis of myocardial ischemia could be expected.

All of these speculations require confirmation or refutation by data directed specifically to each issue. The senior author remains quite consistently “an outspoken critic of monitoring without demonstration of efficacy”.

Stephen Slogoff, M.D.
Arthur S. Keats, M.D.
Department of Cardiovascular Anesthesiology
Texas Heart Institute
P. O. Box 20345
Houston, Texas 77225-0345

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