Sudden cardiac death continues to be the leading cause of mortality in the developed world, claiming 400,000 lives annually in the United States alone. The vast majority of these deaths are attributed to ventricular tachycardia/ fibrillation (VT/VF). Currently, electrical defibrillation remains the only effective treatment for this otherwise fatal rhythm. Large-scale clinical trials have demonstrated that for out-of-hospital and intensive care unit cardiac arrests, the sooner defibrillation occurs, the greater the chance of patient survival. It is in this context that the idioms “time is brain” or “time is myocardium” seem quite apt. The perioperative period constitutes an ideal setting to deliver quick defibrillation given the available monitoring and trained personnel. Nevertheless, perioperative patients constitute a complex and heterogeneous patient population quite different from the myocardial infarction patient found down in the field, and thus, the effectiveness of early defibrillation in this setting remains unproven. In this issue of Anesthesiology, Mhyre et al. report that delayed defibrillation occurs in approximately one in seven patients with identifiable VT/VF in the perioperative settings. Predictably, delayed defibrillation in the “off-site perioperative areas” is associated with reduced patient survival when compared with early defibrillation (31% vs. 62%). Surprisingly, no such association is seen for defibrillation in the operating room. To put such findings into context, one must first understand the pathophysiology of VT/VF and the unique attributes of cardiac arrests in the operating room environment.

VT/VF is created as a result of multiple reentrant waves of electrical excitation that meander erratically through the ventricular muscle. Continued breakup of these electrical waves caused by dispersion of refractoriness in the myocardium leads to the disordered state of conduction that is VF. Factors that perpetuate wavebreak include preexisting myocardial tissue heterogeneity (fibrosis, ischemia/infarcts, myofiber orientation) and intrinsic electrophysiology properties of the ion channels and exchangers that influence wave stability. In diseased state or under abnormal electrophysiological conditions, VT/VF can become self-sustaining. Thus, with unstable hemodynamics, defibrillation becomes essential to stabilize the electrical waves and restore normal conduction quickly.

The latest work by Mhyre et al. sheds new light on the clinical effectiveness of perioperative defibrillation both inside and outside of the operating room. Their effort to analyze complex patient data in the National Registry of Cardiopulmonary Resuscitation should be commended. The results unfortunately demonstrate that even in highly monitored perioperative areas, delays in defibrillation are not uncommon, although substantially less frequent than delays observed in “nonperioperative” in-hospital patients (14% vs. 30%). Lack of an association between time to defibrillation in the operating room and patient survival is interesting and may be explained by differences between arrests inside and outside of the operating room. For one thing, the cardioprotective and neuroprotective effects of anesthetics may confer improved survival when defibrillation is delayed. Furthermore, intraoperative monitoring may improve detection of evolving problems, allowing for actions that may mitigate the consequences of pulseless VT/VF. Such is generally not the case with arrests that occur in other areas of the hospital, likely explaining the much worse survival outcomes.

The lack of demonstrable association between early defibrillation in the operating room and survival may also represent insurmountable study design hurdles. First, the rarity of documented intraoperative VT/VF arrests may have translated to insufficient statistical power. Second, it is conceivable that fast and successful intraoperative defibrillation interventions failed to trigger a “code blue” event and thus were not recorded in the medical record, creating a selection bias. Furthermore, the type of shock (biphasic vs. monophasic) and the exact energy of the therapeutic shock were not standardized, nor was the mechanism for measuring precise time to defibrillation. These study design limitations caused by varying clinical practices among participating centers can impact results and are addressed concisely in the manuscript. However, there exist methodological hurdles that do require further attention. For instance, although Mhyre et al. ana-

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