

Anesthesiologists' Management of Simulated Critical Incidents

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Human error is believed to contribute to the majority of negative anesthesia outcomes. Because retrospective analysis of critical incidents has several shortcomings and prospective studies are limited by the low frequency of critical incidents, an anesthesia simulator was used to evaluate the management of simulated emergency situations by ten anesthesia residents, ten faculty anesthesiologists, and ten anesthesiologists in private practice in order to identify specific patterns of errors in diagnosis and treatment. The simulator is a computer program that presents the patient, monitors, and management choices in a graphical display on an IBM or compatible personal computer. Many errors were observed in the management of these emergency situations, and even anesthesiologists with years of experience made serious errors. Although all experienced anesthesiologists correctly diagnosed simulated esophageal intubation, two residents misinterpreted the lack of end-tidal carbon dioxide. Only 40% of subjects correctly diagnosed simulated anaphylactic reaction; 27% adequately treated simulated myocardial ischemia; and 30% managed a simulated cardiac arrest according to Advanced Cardiac Life Support (ACLS) guidelines. Problems with continuous infusions of vasoactive agents were common. Fixation errors or failure to revise a plan in the presence of inconsistent cues were made by 63% of subjects. The subjects that gathered more information during simulated anaphylaxis made the correct diagnosis more often and made fewer treatment errors. The time since the last ACLS training was found to be an important predictor of correct management of simulated cardiac arrest. Whereas 71% of those trained within the last 6 months managed simulated resuscitations successfully, successful management was decreased to about 30% by those whose ACLS training occurred from 6 months to 2 yr earlier, and no subject who had trained in ACLS longer than 2 yr prior to evaluation successfully followed ACLS guidelines. Based on the retention of ACLS protocols during the management of simulated cardiac arrest, anesthesiologists should review the management of emergency situations such as cardiac arrest, anaphylaxis, myocardial ischemia, and malignant hyperthermia every 6 months to maintain the appropriate skill level. (Key words: Complications: critical incidents. Computer simulation: decision-making. Education.)

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COOPER ESTIMATED that human error is responsible for at least 70% of anesthesia events that lead to adverse outcome.¹ This estimate was based on adaptations of the critical incident analysis method,² which involves voluntary self-reporting of the anesthesiologist's recollection of anesthesia-related human errors and equipment failures. This technique is limited by difficulty analyzing the response to the critical incident due to poor record-keeping during the crisis, missed information, inaccurate observations, and incorrect sense of time.^{3,4}

Because of the low frequency of critical incidents in the operating room, prospective evaluation of the response of a large number of anesthesiologists to these events is not practical. In an effort to model the clinical setting we used an anesthesia simulator to create critical incidents in order to observe how anesthesiologists diagnose and treat these situations. This approach provides an opportunity to look for patterns of errors in the management. The purpose of this study is to assess the ability of anesthesiologists to recognize diagnostic clues, to make the diagnosis rapidly, to effect treatment, and to evaluate the patient's response during simulated critical incidents.

Materials and Methods

The Anesthesia Simulator Consultant (ASC) is a computer program that reproduces many of the events associated with general anesthesia. ASC is an expanded version of the Anesthesia Simulator-Recorder previously described.⁵ In these programs, a graphic interface displays the simulated patient and monitors and allows the anesthesiologist to examine the patient, administer drugs, control the airway, ventilate, and administer fluids using mouse-controlled input. Mathematical models of physiology and the pharmacologic effects of 70 drugs predict the simulated patient's responses. A variety of patient scenarios and critical incidents can be reproduced with the simulator. The patient's vital signs and all management decisions are automatically recorded for review after the case.

With the approval of the University of Washington Human Subjects Committee, 30 anesthesiologists (ten residents, ten faculty anesthesiologists, and ten anesthesiologists in private practice) were evaluated on their management of six simulated cases. The residents had a minimum of 1 yr of anesthesia training. The faculty anesthesiologists had an average of 7.6 (range 4-18) yr ex-

perience, and the private practice anesthesiologists had an average of 8.5 (range 3–16) yr experience. The six cases were 1) a healthy patient with a full stomach; 2) an elderly, dehydrated patient; 3) an esophageal intubation; 4) a patient who developed myocardial ischemia intraoperatively; 5) an anaphylactic reaction; and 6) a cardiac arrest.

The subjects were each presented with the first five cases in the order listed above in a single session lasting 2–3 h. If a cardiac arrest did not occur in these cases, the subject was given a sixth case in which a cardiac arrest would occur regardless of the initial management. None of the subjects knew the type of problem they would confront at the start of the simulation. The subjects were encouraged to vocalize their thoughts during the case; these comments were recorded manually, and the individual's actions using the simulator were recorded by the program.

The first two cases did not include preprogrammed critical incidents and provided the subject the opportunity to become familiar with use of the simulator. The authors were present throughout all of the simulations to assist subjects in making diagnostic and therapeutic manipulations with the program. The first simulated patient was healthy but was at high risk for aspiration of gastric contents due to recent ingestion of a large meal. Preoperative administration of histamine receptor-type 2 blocking agents, nonparticulate antacids, and metoclopramide, and nasogastric suctioning demonstrated concern for prevention of acid aspiration, but rapid-sequence induction with cricoid pressure or awake intubation was required for correct management of this case. The second patient was elderly, dehydrated, and had little myocardial reserve. Optimal management included rehydration prior to induction and careful titration of anesthetic agents with minimal myocardial depressive effects. After these two cases, the subjects were permitted to obtain additional practice on the simulator before attempting to manage the critical incidents.

The first critical incident (third case) involved an esophageal intubation. Breath sounds were abnormal and end-tidal carbon dioxide decreased to zero a few breaths after the intubation. Depending on the duration of oxygen breathing prior to induction, arterial hemoglobin oxygen desaturation would occur after several minutes. Dysrhythmias would occur if the hypoxemia was not corrected in a few minutes. The correct response to the esophageal intubation was to establish a patent airway and to ventilate the patient prior to arterial hemoglobin oxygen desaturation.

In the fourth case, the patient had a history of coronary artery disease with stable angina. Regardless of the anesthetic management, the simulated patient developed ST-segment depression caused by acute coronary vaso-

spasm. In the model, coronary resistance was increased until myocardial oxygen supply was less than 90% of demand. During ischemia, the simulated patient had decreased left ventricular contractility and diastolic compliance resulting in decreased cardiac output and blood pressure and increased left ventricular filling pressure. Hypotension resulted in reflex tachycardia. If the ischemia was not improved in 10 min, the patient's rhythm evolved to ventricular fibrillation. A previously described model of myocardial oxygen supply and demand was used to predict changes in myocardial oxygenation.[‡] In this model, tachycardia, hypotension, and increased filling pressures worsen myocardial oxygenation. Administration of vasoactive agents in the simulator alter heart rate, contractility, and vasomotor tone, which lead to changes in blood pressure, filling pressures, cardiac output, and myocardial oxygen supply and demand. The correct response was to titrate vasoactive agents to improve myocardial oxygen balance so that myocardial oxygen supply was greater than 90% of demand in less than 10 min. Many combinations of agents could be used to increase blood pressure, decrease heart rate, decrease filling pressure, and promote coronary vasodilation.

The simulated anaphylactic reaction was severe. Histamine release occurred a few minutes after the first drug administration, resulting in tachycardia, severe vasodilation, and hypotension. The blood pressure reached its nadir within about 3 min. During this time a skin rash appeared. By 5 min, wheezing, elevated airway pressures, and delayed upstroke of the carbon dioxide waveform could be noted. Correct diagnosis was counted if the subject stated during or after the simulation that anaphylaxis was the likely etiology. The simulated case was programmed to change the heart rhythm to ventricular fibrillation if the systolic blood pressure was less than 30 mmHg for more than 3 min. Treatment of the hypotension with fluids and vasoconstrictors increased the blood pressure enough to avoid a cardiac arrest (systolic pressure > 30 mmHg), but optimal management included assurance of adequate oxygenation, discontinuation of the anesthetic, aggressive fluid administration, and administration of epinephrine (2–3 µg/kg initially). Excessive doses of epinephrine (> 10 µg/kg) led to ventricular tachycardia.

Each subject was tested on the management of cardiac arrest. Resuscitation according to ACLS guidelines for ventricular fibrillation ensured a successful result, but the simulator does not require strict adherence to the exact sequence of steps of the ACLS protocol. Conversion to sinus rhythm occurred in the simulator only when defibrillation with ≥ 300 J followed epinephrine (minimum

‡ Schwid HA, Buffington CW, Strum DP: Computer simulation of the hemodynamic determinants of myocardial oxygen supply and demand. *J Cardiothor Anesth* 4:5-18, 1990.

1 mg intravenous total) and lidocaine (minimum 1 mg/kg total) in simulated patients with arterial pH greater than 7.20 and arterial oxygen partial pressure greater than 50 mmHg.

Hypotheses concerning the management and outcome were analyzed using the one-tailed Z test.⁶

Results

The esophageal intubation was recognized by most subjects either by lack of carbon dioxide on the capnogram or by distant breath sounds with carbon dioxide confirmation. Two residents missed the diagnosis. The first noted the lack of end-tidal carbon dioxide and stated that "this must be really bad bronchospasm." The next several minutes were spent administering a variety of bronchodilators while the simulated patient's condition deteriorated. The initial impression of bronchospasm was never questioned. The other resident noted the lack of carbon dioxide and stated "the carbon dioxide monitor stopped working so I would ask a technician to bring a new one." Two minutes later the correct diagnosis was made.

Although every subject noted the ST-segment changes within 60 s, myocardial ischemia was often inadequately treated (table 1). Tachycardia and hypotension were recognized but were frequently left untreated (table 2). In a few instances, inappropriate drugs were administered, including labetalol and sodium thiopental to treat tachycardia despite hypotension. Continuous infusions of vasoactive agents were especially troublesome. Typical therapeutic dose ranges were not known for a selected agent (nitroglycerin, lidocaine, or esmolol) by 30% of subjects, and 50% had difficulty calculating the correct infusion rate in drops per minute for the desired microgram per kilogram per minute dosage.

In the case of simulated anaphylactic reaction, the diagnosis of anaphylaxis was missed 60% of the time (table 1). The most frequent incorrect or incomplete diagnoses were supraventricular tachycardia (17%), pneumothorax (17%), and electromechanical dissociation (10%). Pulmonary embolism, pericardial tamponade, malignant hyperthermia, and vaporizer malfunction were also consid-

TABLE 1. Management of Simulated Critical Incidents

Incident	Anesthesia Residents	Anesthesia Attending	Anesthesiologists in Practice
Diagnosis of esophageal intubation	80%	100%	100%
Treatment of myocardial ischemia	20%	40%	20%
Diagnosis of anaphylaxis	20%	60%	40%
Treatment of cardiac arrest	40%	30%	20%

See text for correct management criteria.

TABLE 2. Failures in Management of Myocardial Ischemia

Incident	Anesthesia Residents	Anesthesia Attending	Anesthesiologists in Practice
Untreated tachycardia	30%	50%	70%
Untreated hypotension	40%	60%	20%
Inappropriate drug	20%	10%	0%
Unable to recall infusion dose	50%	20%	10%
Unable to calculate infusion rate	70%	40%	40%

See text for grading criteria.

ered. Observation of the subject during the emergency and review of the comments made during and after the event confirmed that an incorrect initial diagnosis was not questioned despite lack of patient response to treatment or other evidence that was not consistent with the incorrect diagnosis.

Eight of the 11 subjects who were able to make the diagnosis of anaphylaxis did so by associating tachycardia, hypotension, and bronchospasm with anaphylaxis. Only 30% (8 of 27) of subjects who did not observe the skin rash made the diagnosis, whereas 100% (3 of 3) of those who noted the skin rash made the diagnosis. This statistically significant difference ($P < 0.01$) implies that noting the skin rash increased the likelihood of making the diagnosis.

Generic treatment of the severe hypotension associated with anaphylaxis to systolic blood pressure greater than 30 mmHg with fluids and vasopressors will prevent a cardiac arrest for this simulated patient. Cardiac arrest was prevented 70% of the time by resident management, 100% of the time by faculty, and 40% of the time by anesthesiologists in private practice. While cardiac arrest occurred in 30% of case simulations, 47% (9 of 19) of arrests were without the correct diagnosis, and no patient (0 of 11) had a cardiac arrest if the diagnosis was made. This statistically significant difference ($P < 0.01$) indicates that making the correct diagnosis improves outcome, even though only fluids and vasopressors were required to prevent the cardiac arrest.

In the simulated anaphylactic reaction, the tachycardia was usually noted first on the electrocardiogram or pulse oximeter. Because of the delay in cycling of the automated blood pressure monitor, the decrease in blood pressure was not identified as rapidly. Almost half of the subjects (14 of 30) initially assumed that the tachycardia was caused by inadequate anesthesia and administered additional anesthetic agents despite the hypotension. In the simulator, the default measurement interval for the noninvasive blood pressure monitor is 3 min. It can be adjusted to measure blood pressure every 1 min, every 2 min, or at longer intervals. A manual reading that takes about 20 s

can be obtained at any time. Fifteen of the 30 subjects (50%) measured the blood pressure only every 3 min during the anaphylactic reaction. Twelve of these subjects (80%) administered agents that could contribute to the cardiovascular collapse, such as thiopental, labetalol, or propranolol. Only 13% (2 of 15) made this error if the blood pressure was measured more frequently. This difference is statistically significant ($P < 0.01$).

Most subjects were not able to recall the initial steps of the ACLS algorithm for the treatment of ventricular fibrillation (table 1). The most common errors involved incorrect dosages of medications (43%), incorrect defibrillator use (40%), failure to hyperventilate (30%), and failure to turn off the vaporizer (23%). Incorrect drug doses included epinephrine (0.1 mg), lidocaine ($0.4 \text{ mg} \cdot \text{kg}^{-1}$), and atropine (0.2 mg). Incorrect defibrillator use included administering inadequate power (50, 100, or 200 watt-seconds) and not defibrillating as often as recommended. A clear relationship was seen between management of the cardiac arrest and the time since the last ACLS training (fig. 1). Seventy-one percent (5 of 7) of anesthesiologists trained in the previous 6 months managed the arrest according to ACLS guidelines. This number decreased to 33% (1 of 3), 28% (2 of 7), and 25% (1 of 4) for those anesthesiologists with ACLS training in the prior 7–12 months, 13–18 months, and 19–24 months, respectively. No anesthesiologist (0 of 5) who had ACLS training more than 24 months prior to evaluation or who never had ACLS training (0 of 4) was able to follow the first few steps of the ACLS protocol for management of ventricular fibrillation.

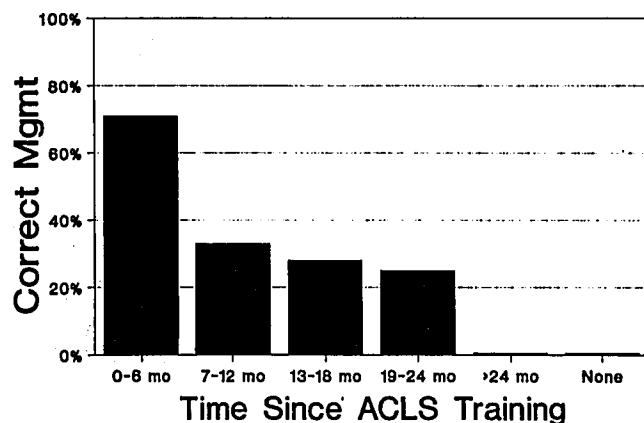


FIG. 1. Anesthesiologists' ability to follow Advanced Cardiac Life Support (ACLS) guidelines for the treatment of ventricular fibrillation is strongly related to the period of time since ACLS training. Subjects with ACLS training within 6 months of evaluation did fairly well, whereas those with ACLS training more than 6 months prior to evaluation had markedly worse management. No anesthesiologist with ACLS training more than 2 yr prior to evaluation and no anesthesiologist without previous ACLS training followed the recommended algorithm.

Discussion

Many management errors were observed in this study of simulated critical incidents. All three groups of anesthesiologists made significant management errors. Our purpose was not to compare the responses of one group of anesthesiologists to the others, but to determine patterns of management errors for experienced and inexperienced anesthesiologists. The results are reported by groups only to illustrate that experienced anesthesiologists were not immune to errors.

Problems were observed in the diagnosis of the critical incidents. Absence of carbon dioxide in the capnogram was interpreted as bronchospasm or a malfunctioning monitor. Similarly, for anaphylaxis, many incorrect diagnoses were made. While waiting for improvement in the patient's condition, many subjects stopped searching for diagnostic information. An incorrect initial diagnosis was not questioned despite lack of response to the treatment by the simulated patient. This type of error is classified as a fixation error.⁷ Fixation errors are so pervasive that several studies even suggest that the best predictor of correct diagnosis is the inclusion of that diagnosis in the initial list of possible etiologies.⁸

The diagnosis of anaphylaxis was especially difficult. Many subjects did not consider anaphylaxis when their patient had tachycardia, hypotension, and bronchospasm. It is noteworthy that when they were asked how anaphylaxis presents, they listed tachycardia, hypotension, and bronchospasm. They could infer from disease to presenting symptoms, but not from presenting symptoms to disease. Furthermore, only 27% (7 of 26) of United States-trained anesthesiologists diagnosed anaphylaxis, whereas 100% (4 of 4) of the anesthesiologists trained outside the United States made this diagnosis. This difference is statistically significant ($P < 0.01$) and could be due to training differences or previous exposure to anaphylactic reactions.

Misuse of the noninvasive blood pressure monitor was common. Although the facsimile of the monitor on the screen-based simulator clearly displayed that the pressure was measured every few minutes, several subjects noted tachycardia but missed hypotension because the monitor displayed a normal blood pressure reading that was several minutes old. This error was observed during both simulated myocardial ischemia and anaphylaxis.

In the event of an unknown problem, the anesthesiologist should gather more information by examining the patient and monitors, and ordering laboratory studies. An initial diagnosis should be considered from a differential or precompiled list, and treatment should be initiated. The information should be continuously updated and reevaluated to avoid errors in diagnosis and treatment. Breakdowns in this process were observed at every

step by experienced and less experienced anesthesiologists. Nineteen of the 30 subjects (63%) made one or more fixation errors in which an initial faulty diagnosis was not reassessed despite information that was not consistent with the diagnosis. The diagnosis should be based on complete information, but only 10% of the subjects looked for a skin rash during severe hypotension even though this is an important physical finding. It is not unrealistic to require the anesthesiologist to look actively for a skin rash on the simulator, since that is the only way it would be observed in a draped patient in the operating room. Noting the presence of the skin rash significantly increased the probability of making the correct diagnosis. Initial diagnoses such as bronchospasm in the case of esophageal intubation or supraventricular tachycardia in the case of anaphylaxis should be reevaluated, especially when the patient is unresponsive to conventional therapy.

In some cases treatment should be initiated before a diagnosis is established. With severe hypotension, for example, the anesthesiologist should immediately discontinue or decrease the concentration of the anesthetic, ventilate the lungs with 100% oxygen (if hemoglobin oxygen saturation by pulse oximetry is decreasing), administer fluids and pressors, and then try to make a specific diagnosis. Several subjects (9 of 30), even experienced clinicians, undertreated severe hypotension while trying to find the etiology. This behavior, an example of cognitive lock-up, has been observed in the operating room.⁹ Frequent reassessment was found to reduce management errors. Anesthesiologists who reevaluated blood pressure at 3-min intervals made significantly more therapeutic errors than those who measured blood pressure more frequently.

Other problems were also observed in the treatment of the critical incidents. Continuous infusions proved to be a particularly troublesome area. Many subjects did not know therapeutic doses, and others found it difficult to calculate correct drip rates during the emergency. While many simply started the infusion at a low dose and increased the infusion rate to achieve the desired effect, it is inefficient to start the rate at one tenth or less of a typical therapeutic dose and not have the ability to compare the administered dose to the typical dose.

Even a simple, well-rehearsed protocol, such as the ACLS algorithm for cardiac arrest, is hard to remember during a crisis unless it is refreshed in memory periodically. Performance was good (71% had correct management) for those anesthesiologists with ACLS training within the past 6 months, but declined significantly (about 30% had correct management) for those trained in ACLS more than 6 months prior to evaluation. No subject trained in ACLS more than 24 months prior to evaluation followed the protocol correctly. These results agree with previous studies that demonstrated poor retention of

ACLS protocols by residents called to a simulated cardiac arrest.^{10,11} The American Heart Association recommends that the ACLS course should be repeated every 2 yr, but based on our observations this period of accreditation should be considered the maximum time between training, rather than a period of competency. Frequent updates are recommended since Stross¹² demonstrated a significant improvement in ACLS skill retention 1 yr following the course with quarterly review using patient management problems or mailed periodic reprints.

Gaba and DeAnda studied the response of experienced anesthesiologists¹³ and trainees¹⁴ to simulated critical incidents on a full-scale operating room simulator.¹⁵ As in the present study, diagnostic and therapeutic errors were commonly made by both anesthesia trainees and experienced anesthesiologists. Gaba reported from these studies that the anesthesiologist's primary diagnostic process involves matching one or more abnormalities to known patterns, rather than abstract causal reasoning. In agreement, we also found that anesthesiologists made the diagnosis by observing an abnormality and selecting possible etiologies from a precompiled list. In addition, Gaba and DeAnda stated that the primary treatment pattern was to recall precompiled sets of actions. This pattern was observed in the present study for the treatment of anaphylaxis and ventricular fibrillation. The anesthesiologists who aggressively treated the myocardial ischemia listed increasing the blood pressure, decreasing the heart rate, and coronary vasodilation as treatment goals, although they did not appear to be constrained to a specific set of actions.

LIMITATIONS

Ideally, evaluation of anesthesiologists' management of critical incidents would be based on observations in operating rooms while caring for actual patients. The conclusions would not be tempered by evaluation in an artificial environment. Of course, it could take years to be present at 30 unplanned esophageal intubations, 30 cases of myocardial ischemia, 30 anaphylactic reactions, and 30 cardiac arrests. Even if the events could be witnessed, the observer would be ethically obligated to intervene if errors were perceived. Videotape and automated record-keeping systems offer the possibility of reproducing details of large numbers of cases, but the critical incidents cannot be controlled or duplicated as in a simulator.

Performance in a simulator is different from performance in the real situation. In some cases performance will be better in the simulator, and in some cases it will be better in the real world. Response in a nuclear power plant simulator was found to be superior to that of field tests because the subject had a heightened state of vigilance and anticipated problems in the simulator.¹⁶

Unrealistic simulation of the workplace may have a negative impact on performance. Even in a full-scale simulator, fidelity of the mannequin has limitations. The real operating room environment is admittedly very different than the screen-based simulator: mathematical models predict patient responses; analog and digital information normally presented on physiologic monitors are displayed on facsimiles of the monitors; the anesthesia machine and circle system are displayed graphically; and physical signs are displayed as text messages.

In order to determine the utility of the screen-based simulator as a training and evaluation device, it was tested by 44 anesthesiologists at seven different training centers. They reported that the models produced clinically realistic predictions of patient behavior, that mouse-driven input was easy to use, and that the graphical display clearly presented the simulated patient's condition.⁵ In a recent survey of anesthesiology residents at the University of Washington, 92% (25 of 27) stated that the simulator was realistic.

The performance impact of the artificial nature of the screen-based simulator was also controlled by familiarizing all subjects with the simulator through practice cases prior to critical incident management. The subjects worked through two practice cases to become familiar with the operation of the simulator and were given the opportunity for further practice before beginning the critical incidents. The authors were immediately available for consultation to ensure that there were no difficulties operating the simulator.

It is certainly possible that anesthesiologists' responses to real critical incidents in an operating room could be different than reactions in response to this screen-based simulator, but we believe that the observations concerning management of simulated critical incidents are valid despite the differences between the screen-based simulator and the real situation. Graphical simulation does not account for misinterpretation of absent carbon dioxide in the capnogram after intubation, difficulty calculating infusion rates, or inability to recall ACLS protocols. There is no reason to believe that these errors would not have occurred in the operating room under similar circumstances. It is possible that treating tachycardia without determining the current blood pressure using the non-invasive blood pressure monitor may be an artifact of the screen-based simulator. This issue should be reevaluated in a full-scale simulator.

Every subject evaluated in this study made at least one potentially dangerous error. We infer that as a specialty, our management of emergency situations is not yet instinctive and flawless. We need to improve anesthesiologists' training for the management of critical incidents. We must develop methods to avoid fixation errors. It may be helpful to have simple charts in the anesthesia machine

for differential diagnosis and tables for continuous infusions of medications.

Based on the poor retention of ACLS protocols 6 months after training, we propose that anesthesiologists review the management of critical incidents at least every 6 months. In addition to cardiac arrest, anesthesiologists should be prepared for anaphylaxis, difficulty ventilating an intubated patient, malignant hyperthermia, pneumothorax, pulmonary embolism, equipment failures, and other emergencies. The preparation can consist of reading and reviewing an organized approach (algorithm) or refresher courses covering these incidents. Anesthesia simulators may become an important way to train and retrain for critical incidents. Anesthesiologists can now rehearse the management of these problems with a graphic simulator on a personal computer; in the next few years, full-scale operating room simulators may become accessible. Graphic simulators operating on personal computers, also known as microsimulators,⁸ have proven to be highly effective training devices for radar,¹⁷ sonar,¹⁸ weapons systems,¹⁹ and nuclear power.²⁰ Further studies will examine the role of graphic and full-scale simulators in training and retraining for critical incidents in anesthesia.

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