Unexpected Deaths during Anesthesia: Wherein Lies the Cause?

That death may follow exposure to anesthesia and operation is well known to physicians. Because of exposure to television, the press, and popular novels such as COMA, there is an increasing awareness of this possibility among lay persons. When the patient’s disease is dramatic and profound, such as major trauma, sudden spontaneous subarachnoid hemorrhage or impending myocardial infarction, and where prompt anesthesia and operation may be lifesaving, the potential for a fatal outcome is recognized and accepted by all involved. Under such circumstances, deaths during anesthesia are rarely challenged. However, when death or serious disability occurs under more ordinary circumstances of anesthesia and operation, a reaction is commonly set into motion, usually by patients or relatives but with increasing frequency by other physicians. The possible causes of the death or disability are explored (at least in theory), and when no plausible explanation is forthcoming, it is often concluded that physician error was the cause. It is reasoned that because so many patients each year undergo exposure to anesthetic drugs without serious problem, when an unexpected death or serious disability does occur and no other obvious explanation emerges, the anesthetist must be to blame. These are circumstances that anesthetists have come to recognize and abhor.

In this issue of Anesthesiology, Dr. Arthur Keats considers the question of anesthetic mortality. In spite of many broadly based covering statements, I fear a strong and dangerous implication—carefully never concisely stated—is that drugs per se are responsible for an important number of anesthetic deaths.

As expected from one with Dr. Keats’ background and talents, there is much correct in his protestation, and we may reach that happy compromise of differing in degree only. In my view, Dr. Keats correctly identifies some errors in the utterances of pundits and commissions. They did overstate their case. He properly emphasizes the fallibility of establishing firm conclusions without proper and complete information. I share his dislike for equating departure from current practice with error, as little will be gained without such departure. He wisely implores us to take a fresh look at anesthetic mortality—and try as I might, no quarrel with this plea is forthcoming.

Beyond this, however, I think him incorrect in two very basic aspects of his presentation. First, he has erred in his assessment of the profession’s posture regarding the cause of anesthetic mortality. Second, he has erred by overemphasizing the role of drugs alone in anesthetic mortality. Additionally, I identify some specific incorrect statements forwarded in this presentation. I know of no member of our specialty who would glibly state values for anesthetic mortality—far from it. The experts claim that we do not know the incidence of anesthetic mortality, and have urged that this issue deserves major attention and funding. Unless and until we can firmly document the frequency and causes (including unknown) of unexpected anesthetic mortality by improved record keeping and reporting systems, the true incidence and causes are not likely to be known.

An important thrust of Dr. Keats’ article is that the
error bias has paralyzed our thinking towards considering any explanation other than pilot error. He states "the error bias precludes any new knowledge concerning mechanisms of death attributable to anesthesia." If true, why did we carry out investigations of halothane hepatitis, succinylcholine-induced potassium flux, and methoxyflurane renal toxicity? Dr. Keats concludes that these discoveries did not result from studies directed to anesthetic mortality by review of death reports. Again, we differ. These investigations did result from reviews of death reports; perhaps not from formally constituted commissions but from clinicians who, when serious events were brought to their attention, suspected drug effects and conducted research to elicit answers. Before the mechanisms were discovered, no anesthetist was found guilty of malpractice in these areas. The risk/benefit concept is widely recognized in the specialty. I have not heard it doubted or even challenged. Dr. Keats and I have shared many hours on hunting trips and I know him to be a far better marksman than I. It is therefore especially difficult for me to perceive his existing with the above facts in full view and concluding that we exonerate drugs and ignore developments in modern clinical pharmacology. He has created a strawman for purposes of debate and overstated his case as strongly as his self-appointed adversaries overstated theirs.

The most profound implication of Dr. Keats' article is that drugs per se may be a major cause of anesthetic mortality, irrespective of the skills of the anesthetist. This is not a new idea. My recollection—not nearly exactly the same time span as Dr. Keats'—indicates that drugs and patient diseases were given most of the blame for anesthetic misadventures. Thiopental caused laryngospasm; cyclopropane caused morbid ventricular arrhythmias; patients died suddenly of status thymolymphaticus; alcoholics "took a poor anesthetic"; a history of difficult anesthesia meant avoiding that particular agent at all costs. Diseases were blamed to such an extent that they became rather absolute contraindications to anesthesia. Even today, we encounter many patients who are told that their unhappy anesthetic experience in the dental chair was due to a "drug reaction." More commonly, we are still admonished by colleagues to avoid specific drugs in particular situations as if the choice of these drugs were specifically determinable, sacred and certain. Careful review, introspection and, yes, even self-flagellation have altered this concept to a large extent. We know that proper administration of thiopental can actually avoid laryngospasm, status thymo-whatever is not known to today's students, and diseases that were contraindications are now indications for our most major anesthetic ventures.

From this earlier posture of blaming drugs, we have increasingly recognized the importance of human error and blamed the drugs less and less. I cannot accept Dr. Keats' statement that this development arises from an earlier concept of chemical inertness of anesthetic agents. The reluctance to blame our drugs for catastrophic reactions came from the observations of millions of exposures without the unhappy event in question's being experienced. When thiopental is used successfully for an overwhelming majority of anesthetics given every day throughout the world, a death from this drug is not very likely. When a drug has been administered successfully for an hour or two to a patient, sudden death from an adverse reaction—or drug interaction—is most improbable. To blame an undiscovered or unexplained acute toxic effect of a drug, an idiosyncratic reaction, or divine intervention, as would appear to be Dr. Keats' thrust, is very questionable when we know that physician errors can result in mortality. We know that treating a patient's complaint of dyspnea during spinal anesthesia with sedation can result in death. We don't know that any adverse drug reaction or idiosyncrasy or drug interaction can cause death as a result of spinal anesthesia per se. We know that leaving the operating room during anesthesia makes recognition and management of ventilator disconnections or cardiac arrhythmias unlikely, and that death may result. We don't know of a drug idiosyncrasy or interaction that would produce sudden death in the middle of an otherwise stable operative procedure. We know that induction of general anesthesia in patients who have unrecognized airway compromise will have a measurably high incidence of serious complications. We know that failure to identify preoperative renal failure may result in relative drug overdose, and failure to take into account preoperative pulmonary disease may allow dangerous levels of hypoxia to develop during and after anesthesia. Such results from idiosyncratic responses to drugs remain entirely in the realm of supposition and theory.

The case for "unavoidable" adverse actions of drugs as an important cause of death would be stronger if Dr. Keats followed the advice he admires from Dr. Snow's article "—he conducts his own experiments to verify that the reported events could lead to death."9

There is presented no such verification in the current article, and what evidence is elsewhere available points to the extreme rarity and uncertainty of idiosyncrasies and adverse drug actions as causes of death.

There is no doubt that there are variations in responses to average doses of drugs. A competent anesthetist expects, prepares for, recognizes and treats these variations properly and promptly. This is the essence of the practice of anesthesia, and it is an
important area in which anesthesia differs from other specialties. Failure to recognize these differences in response, and to recognize them in time to recover from either error or varying response, is rarely the drug’s fault.

There is little doubt that drugs with such powerful effects on vital functions as anesthetic agents possess will result in harmful effects on occasion even in the best of hands. It must be admitted that idiosyncratic reaction can occur, and with the myriad drugs now used, serious drug interactions must remain a possibility. The important controversy, then, is the relative role of drugs as causative elements in anesthetic mortality, as opposed to the role of management error. Cliches may be used to support either view. On one hand it is said “we don’t debate safe versus dangerous anesthetic agents—only which is the least dangerous”; on the other “There are not bad anesthetics, only bad anaesthetists.” While it is mathematically imprudent to conclude that mortality or near-catastrophe occurs only as a result of misuse or error, we must consider probabilities, especially when they are overwhelming. Practically, it boils down to the statement in Dr. Keats’ text “... it is important to know whether anesthetic deaths attributable to error amount to 10 or 90 per cent.” In my view, error is near the 90 per cent end.

Dr. Keats’ article will probably be most popular. It offers salve to our conscience and provides at least a glimmer of hope and defense in malpractice situations.

Life is easier when our problems, especially the serious ones, can be blamed on the tools of our trade, our patients, the gods,—or anything else that has an aura of unavoidability. What is Dr. Keats’ final message? To the extent that he says “don’t make decisions without proper basis,” I agree wholeheartedly. To the extent that his theme is that drugs per se are numerically an important cause of anesthetic mortality, I disagree strongly. This opinion is based on the statistical probabilities, or improbabilities of drug behavior. It is further based on experience that has repeatedly seen error produce predictable result. This view in no way states that drugs can never be causative, or even suggests that all error is avoidable, or that benefits are not achieved without certain definite risk. There is room for all these to coexist.

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Reference

Dr. Starling and the “Ventilator” Kidney

Once, a time few can remember, the pattern of breathing was described in simple terms: fast or slow, shallow or deep, quiet or labored. Later, the stethoscope and the trained ear added a new dimension to our interpretation of the sounds of breathing. Unfortunately, during progress from less art to more science, we have improved the instruments but forgotten the music. In fact, our penchant for innovation has promoted the use of a language without grammar and a branch of science best characterized as the physiology of iatrogenesis. “To breathe” is no longer enough; it is now ventilation, controlled, mechanical or not, intermittent, mandatory or not, with or without PEEP, CPAP, and many more. The juggernaut of abbreviations threatens to gain momentum; so does the complexity of iatrogenic abnormalities and their grotesque description in terms such as “respirator” lung, “respirator” brain, and many more. One shudders to contemplate the possibility that acute pancreatitis, developed during mechanical ventilation, may someday be described as “respirator” pancreas!

Now we have moved in a new direction which, for lack of more elegant terminology, I propose to call the natural history of the “ventilator” kidney. In this issue, Marquez et al. provide nourishment for thought and an opportunity to consider the qualitative differences in the responses of the lung, normal or otherwise, the heart, normal or otherwise, and, as we have long suspected, the kidney, normal or otherwise, to breathing with added airway pressure. Their study raises two questions: one minor, the other major,

Key words: Kidney; blood flow; function. Ventilation: mechanical.