The 27th Ravenstine Lecture: Neuroanesthesia and the Achievement of Professional Respect

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I AM HONORED and privileged to be chosen to present this, the 27th Ravenstine Lecture. I am of that generation who did not know Dr. Ravenstine personally. However, having heard most of my predecessors at this podium, many of whom did know him, I feel as if I also knew him—at least in spirit. Were he present today, I believe he would be proud, for the most part, of the specialty which he so influenced in uncounted ways during its early formative years. I'm sure too that he would have been disappointed in some of our failings. His influence obviously continues on by way of the many anesthesiologists whom he trained and who have been, for a number of us, role models in academic anesthesiology. Dr. Ravenstine was among the first of the truly academic anesthesiologists, and it is this heritage that he and others passed on which we must continue to nurture and allow to grow. We cannot forget, of course, that anesthesiology as a medical specialty is responsible for clinical service, but we must never allow the clinical pressures for delivering service to choke off academic pursuits. In too many departments across the country, this has been allowed to occur. To the degree that we permit it to occur, we shall never fully gain the professional respect we seek from our colleagues in the other medical specialties.

I intend in this presentation to consider aspects of the process and some of the pitfalls in the achievement of professional respect using examples taken from my chosen subspecialty: neuroanesthesia. As an aside, you might be interested in knowing how I believe this subspecialty came to be so named. In 1968, I submitted an invited review article authored by myself and Drs. Gerald Gronert and Kai Rehder to the then Editor-in-Chief of Anesthesiology, Dr. Leroy Vandam, with a covering letter that included the following: "Regarding the title 'Neuroanesthesia,' I recognize it is not a generally accepted term.

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III anesthesia. The literature was largely barren of any information. There were a few exceptions: by 1960, Dr. Harvey Slocum had introduced the use of controlled hyperventilation for brain tumor patients in his military practice at Walter Reed Hospital arguing that it resulted in a more relaxed brain. At the Mayo Clinic, Drs. Edward Daw and Howard Terry were in the process of attempting to introduce controlled hyperventilation, while several of our neurosurgeons remained opposed. In truth, none of us really knew what we were doing or why; we were literally groping in the darkness.

This changed within a few years when the group at the University of Pennsylvania, including Drs. Harry Wollman, Craighead Alexander, and Peter Cohen, began to report their findings regarding the effects of anesthetics on cerebral blood flow and cerebral metabolism in volunteers; while the group in Glasgow, including Drs. Gordon McDowall and Murray Harper, began to report their findings regarding the effects of anesthetics on cerebral blood flow and metabolism in experimental animals. Stimulated by these early reports, I was soon encouraged and accommodated by Dr. Richard Theye in 1964 to pursue similar studies in his laboratory. I jumped at the opportunity and have been involved in such activities ever since, with the generous support of both the Mayo Clinic and the National Institutes of Health. By 1969, most of the basic knowledge regarding the effects of anesthetic agents on cerebral blood flow, cerebral metabolism, and intracranial pressure was in the literature, and much of it came from the three laboratories I have mentioned. This, then, was a major achievement and was accomplished almost exclusively by academic anesthesiologists. We had moved from a state of profound ignorance to one of sophisticated understanding. We could discuss in depth what each of our drugs or interventions and their combinations might do to cerebral blood flow, cerebral metabolism, and intracranial pressure.

And then, in my opinion, something went wrong. It began in 1969, when a report in *Lancet* summarized the Glasgow group’s findings regarding the effects of certain volatile anesthetics on intracranial pressure in patients with brain tumors. It was followed by an anonymous editorial in the *British Journal of Anaesthesia*. The report in *Lancet* demonstrated that with halothane, trichlorethylene, and methoxyflurane, intracranial pressure increased immediately upon introduction of the agent. Furthermore, it was stated that this increase in ICP was not prevented by “hypocarbia.” However, this was not true hypocarbia—the patients were intended to be studied at normocarbia but, because of inadvertent temperature decreases, the corrected PaCO₂ resulted in values below 35 mmHg. If you believe, as I do, that physiologically such correction is wrong, then, in fact, none of their patients were truly hypocarbic. As an aside, I might add that one of the most fascinating lectures I have ever heard was that given by the renowned physiologist, Dr. Hermann Rahn, concerning his work in poikilotherms which provided the basic evidence against temperature correction for PaCO₂ and pH values. Interestingly, that lecture happened to be the 7th Ravenstine lecture given in 1968, 1 year before the Glasgow work was published. In any case, the anonymous editorial in the *British Journal of Anaesthesia* utterly condemned the use of any of these volatile anesthetics in neurosurgical patients at risk and concluded that even hyperventilation is without effect. I recently discussed this work with two of Dr. McDowall’s early coworkers, Drs. William Fitch and John Barker. They pointed out that in none of their publications did they ever arrive at a clinical recommendation that anesthetics such as halothane were absolutely contraindicated in these patients. Rather, it was those who had not done the work that arrived at these conclusions.

But the die was cast. Never mind that halothane had been the most commonly used anesthetic in neurosurgery for almost 10 years the world around at that time. Never mind that up to that time no neurosurgeon had made known his suspicions that halothane caused a tight brain. Never mind that at the Mayo Clinic alone we had anesthetized more than 2000 patients undergoing craniotomy using halothane, and neither anesthetist nor surgeon even recognized an intracranial pressure problem. Certainly the latter group, i.e., the neurosurgeons, are not noted for their reluctance to identify such problems.

In response to this sudden condemnation of volatile anesthetics, we and others did studies to further elucidate the effects of halothane. In our study, as reported by Adams et al., we clearly demonstrated that by the simple expedient of prior hyperventilation (i.e., producing true hypocarbia at normothermia), clinically significant increases in intracranial pressure do not occur upon introducing 1% halothane in patients at risk. Almost reluctantly, it seemed, most agreed that, yes, you can use halothane, but you should avoid it if you can. To this day, I do not know why I should avoid it. We also showed in that study why the potential effects of halothane on intracranial pressure had never previously been appreciated. In those patients studied without prior hyperventilation, about one-third did experience some increase in intracranial pressure following simultaneous introduction of halothane and hyperventilation. But even in these, the increase lasted less than 30 minutes followed by spontaneous return usually to below baseline. Thus, by the time the craniotomy was completed, any effect of halothane on intracranial pressure or brain mass was dissipated.

But the pattern had been established and remains to this day. In short, any intervention or drug which can be shown to cause any increase in ICP provides a license for condemnation, at least by the pseudoacademicians. Never
mind that the reported increase in intracranial pressure may only be of the magnitude of 10 or 15 mmHg (or less). A favorite gambit by these pundits is to report increases in intracranial pressure as a percentage of control to perhaps as much as 200–400% or even more! Shocking! Until one notes that the increase is from 5 mmHg to perhaps 10 or 20 mmHg. Why should such increases concern us? Daily, we allow mean arterial pressure to decrease or increase by this magnitude without any concern. So surely it is not the effect on cerebral perfusion pressure that causes us to worry about such ICP increases. Are we concerned about herniation of brain tissue through fixed structures? Yes, of course. But, again, not with only modest increases in intracranial pressure. Indeed, I challenge any of you to find a single case report of an anesthetically induced intracranial disaster, such as herniation, since the early days of ketamine when it was incorrectly used for neuromodiagnostic procedures, or since the days when nitrous oxide was used incorrectly during air contrast studies. Granted, halothane and the other volatile anesthetics have the potential to induce such a disaster, but, if used correctly, they will not do so.

The list of currently popular agents condemned by our pseudoacademic associates is mind-boggling: halothane, enflurane, isoflurane, nitrous oxide, ketamine, d-tubocurarine, atracurium, pancuronium, succinylcholine, nitroglycerin, and nitropressure are ones that come immediately to mind; I’m sure there are others. All share in common that, under the proper circumstances, it is possible to induce some increase in intracranial pressure with these drugs. Take d-tubocurarine. One study from Finland showed that, when you give a large bolus dose of 0.6 mg/kg to spontaneously breathing patients, you will produce a modest transient increase in intracranial pressure from 10 to 18 mmHg for a short period of time of less than 2 minutes, possibly due to histamine release. Therefore, condemn it? Nonsense! The increase itself was probably unimportant, and if you want to avoid that, simply don’t give large bolus doses. Take isoflurane. In two human studies, including one from my institution, it was concluded that isoflurane plus hyperventilation was safe in patients with intracranial mass lesions. But, in a third study, the authors concluded differently, even though eight of 14 of their patients had no increase in intracranial pressure, while, in their remaining six patients, the increase in intracranial pressure was so modest that there was no need to change the protocol or to discontinue the isoflurane. What, then, was the basis for their conclusion that “Isoflurane may not be a benign anesthetic in patients known to be at risk for increases in intracranial pressure”? To extrapolate clinically insignificant measurements of increased intracranial pressure to potential intracranial disasters is without foundation and should be ignored. Take nitropressure. Yes, it can cause modest increases in intracranial pressure in patients with brain tumors. But to recommend instead that an unreliable hypotensive agent such as trimethaphan should be used because it’s perhaps less likely to induce a modest increase in intracranial pressure is clinical nonsense. In patients with aneurysms, for example, immediate reliable control of systemic blood pressure is essential. Do not replace nitropressure with trimethaphan for such patients. To do so is accepting ill-directed advice.

Why the eagerness to condemn? Is it some form of demagoguery? Does it imbue the condemner with some sense of power? I am struck that more often than not the ones who condemn are not the ones who did the work; more commonly, it is the hangers-on, the pseudoacademician. In my opinion, concern about intracranial pressure has become a fetish among self-appointed so-called “expert” neuroanesthetists. They have promoted the unimportant to the status of importance, possibly as a means of gaining self-importance. Those who promote such fashions for whatever reason should be challenged to provide logical support for their position backed up by hard scientific data. Without such, their opinions should not be respected and they should be ignored. Neither is this problem unique to neuroanesthesia. I would only point to the recent furor regarding isoflurane’s potential for producing coronary steal as another example of probable premature condemnation with only marginal scientific foundation.

A second scientific area wherein our specialty might take pride is that work dealing with the potential for protecting and/or resuscitating the brain. Since the mid 1950s, anesthesiologists, along with neurosurgeons, cardiac surgeons, physiologists, and neuroscientists, have made major contributions to our understanding of the neuroprotective effects of hypothermia. That this is now an established and reasonably well understood protective intervention is certainly in part due to the many valuable contributions made by a number of academic anesthesiologists.

With the explosion of information in the mid to late 1960s regarding the effects of anesthetics on cerebral blood flow and metabolism, it was natural to explore the potential for possible neuroprotective effects of some of these anesthetics and other drugs. Throughout the 1970s, attention was focused largely on the potential of barbiturates for brain protection or resuscitation. Much of this work came from anesthesiology laboratories, but by no means all of it; neurologists, neurosurgeons, and basic neuroscientists have also been deeply involved in this field. Among the anesthesiology laboratories that have made major contributions, I would include Dr. Peter Safar’s, Dr. Harvey Shapiro’s, and, perhaps immodestly, my own. Throughout the 1970s, the topic was controversial and often heated. This controversy was, of itself, good in that
it stimulated considerable interest and a great deal of work. As a result, we have today a fairly good grasp of the circumstances during which barbiturates might protect the brain—namely, during incomplete ischemia or partial hypoxia. We also know that barbiturates will neither protect the brain during complete ischemia nor offer any potential for resuscitating the brain following complete ischemia (i.e., cardiac arrest).¹¹ We also know that, although barbiturates have the capacity to decrease intracranial pressure, they do not improve prognosis in head-injury patients. For the most part, we have a reasonable understanding of at least some of the mechanisms that account for these barbiturate effects. The methodologies learned in dissecting out the barbiturate story during the 1970s are now being applied to a host of other pharmacologic interventions that might provide brain protection or resuscitation. These include isoflurane, calcium entry blockers, free-radical scavengers, iron chelators, and excitatory amino acid antagonists. One of these, the calcium entry blocker nimodipine, has already been shown in primates in my laboratory to impact favorably on brain resuscitation following complete global ischemia as reported by Steen et al.¹² In humans, nimodipine has been reported to improve outcome in cardiac arrest patients when compared to a retrospective historical group¹³ and the drug is now undergoing trial in a randomized prospective study in Europe. In addition, nimodipine has been shown in humans to improve outcome in acute ischemic stroke¹⁴ and to decrease the morbidity secondary to cerebral vasospasm in aneurysm patients.¹⁵-¹⁷ The potential for an exciting breakthrough in this field is a real one, and our specialty will have played a major role in this development should it come about.

So there is reason to be proud. But have we forgotten the uncounted thousands of patients who were subjected to prolonged barbiturate-induced coma during the mid to late 1970s and into the early 1980s? Anesthesiology must take the blame for that as well, and, as such, we must lose some of the respect that was so arduously gained. How many patients were hurt by such an intervention? No one knows, but certainly none were helped. How did this come about? Largely as the result of a single positive primate study,¹⁸ which later proved to be irreproducible,¹⁹ an enthusiastic group of proponents, and a plethora of physicians who were grasping for anything, simply anything, that might help. It would have been different, perhaps, if barbiturates, like steroids, were associated with little down-side risk, but the hemodynamic and CNS effects of a high dose of barbiturates alone represent major interventions that should not be introduced lightly. It is to be hoped that we have learned from this rather sorry experience—perhaps we have overlearned.

Thus, despite the unchallenged demonstration in primates that nimodipine improves outcome following complete cerebral ischemia and despite the fact that the drug is virtually innocuous in humans as regards systemic or other CNS effects, it has been argued by some of our peers in this country that more animal studies are needed before we dare give it to patients. Fortunately, the Europeans have not seen it this way, and the appropriate studies are ongoing in patients. Similarly, the demonstration in patients by Nussmeier et al.²⁰ that high-dose barbiturates during cardiopulmonary bypass reduces neuropsychiatric complications, although not challenged, has not apparently had any major impact as yet on anesthetic practices. Granted, high-dose barbiturate therapy during cardiopulmonary bypass is not an innocuous intervention for the reasons already considered and perhaps practice should not change, but I suspect the original embarrassing experience with barbiturates following cardiac arrest has raised a red flag of caution of such proportion that all are hesitant to walk that path again. Yes, we may be proud as a specialty of our contributions, but not our applications in so far as brain protection and resuscitation are concerned.

A third area to which neuroanesthesiologists have made major and almost exclusive contributions is that of diagnosing and managing venous air embolism. When I first began practice as a consultant in 1961, the complication of venous air embolism in patients operated while in the sitting position for posterior fossa surgery was considered to be rare—probably less than 1%. At the same time, it was recognized that, when the complication did occur, the mortality was high—usually over 50%.²¹ Diagnosis of air embolism was dependent on recognizing the so-called “mill wheel” murmur with either a precordial or esophageal stethoscope. We now know that the “mill wheel” murmur is a late sign indicative of a large volume of intracardiac air and is audible only if the heart is still able to contract with some vigor.

All of this began to change as the result of two repetitive serendipitous events that occurred at the Mayo Clinic in the early to mid 1960s. For reasons I’ve long since forgotten, I was interested in measuring central venous pressure in patients operated on while in the sitting position. Catheters were inserted somewhere in the superior vena cava for this purpose without precise localization. On one occasion, a patient being so monitored developed signs that were at least suspicious of air embolism: hypotension, premature ventricular contractions, and a low grade systolic murmur on the esophageal stethoscope. An associate, I believe Dr. Edward Daw, suggested that I aspirate the catheter. Upon doing so, we were amazed that over 100 ml of air were aspirated over the next several minutes and all of the patient’s signs of air embolism disappeared. We thought this a unique experience that likely would never be repeated. We did not change our practice, we did not report the case. But within a few months in an-
other patient being monitored with a catheter in the superior vena cava, the experience repeated itself almost exactly. Now we did change our practice and began to insert catheters in all patients being operated in the sitting position. The first problem was where to localize the tip of the catheter. Pulmonary artery catheters were not available at that time, so this was not even a consideration. Ironically, at this very same time, Dr. Jeremy Swan of eventual Swan—Ganz catheter fame was walking the halls just one floor above us working in his cardiac catheterization laboratory. We attempted placement of the catheter in the right ventricle, but this resulted in a high incidence of premature ventricular contractions and difficulty in aspirating the catheter. Thus, by elimination, we selected the right atrium, assuming it would be more effective than superior vena caval placement. Thereafter, we began to catalogue the early signs of air embolism consisting primarily of a characteristic low grade systolic murmur detectable by the esophageal stethoscope and confirmed by aspiration of air bubbles.

In 1969, we reported the "true" incidence of air embolism, proven by aspiration of air bubbles, to be about 7% with no morbidity or mortality. From this modest beginning, our knowledge of venous air embolism grew exponentially. Probably the most important contribution during this time was the introduction of the Doppler in 1969 by a neurosurgeon, Dr. Joseph Maroon, and an English anesthetist, Dr. John Edmonds-Seal. They reported a remarkably high incidence of air embolism of over 50% in a small group of only seven patients operated on while in the sitting position for posterior fossa pathology. In disbelief, we adapted a Doppler designed for fetal monitoring and, much to our surprise, confirmed the Maroon report by aspirating air bubbles in over 40% of our patients when the Doppler developed characteristic sounds of air.

Since that breakthrough, most of the progress represents fine tuning of our understanding of the complication and of our monitoring techniques. Mortality from venous air embolism in neurosurgery is now almost unheard of. Morbidity is likewise negligible. The one exception is that rare complication of a symptomatic episode of paradoxical air embolism, i.e., air crossing to the systemic arterial circulation resulting in either stroke or coronary embolization. Even that complication can now be diagnosed intraoperatively using a transesophageal echocardiograph, as was reported by Cucchiara et al. We also now know, thanks to the work of Bunegin et al. that placement of the catheter tip is probably best just above the junction of the superior vena cava with the right atrium and that air retrieval is further improved by the use of catheters with multiple orifices.

This, then, is a story of steady remarkable progress whereby academic anesthesiology has converted our perception of air embolism in patients operated in the sitting position from that of a rare complication with a high mortality rate to that of a common complication with a minimal mortality rate. The key, of course, has been early recognition by highly sensitive accurate monitors. We, as a specialty, can and should be proud of this story.

But, once again, I believe we have tripped over our own successes and thereby have diminished the respect otherwise gained. There are those who contend it is malpractice and negligent to operate on a patient in the sitting position without a central venous catheter. In my practice, we have done so on occasion for all these years and continue to do so. Granted, we always try to insert such a catheter and we usually succeed. But if a reasonable effort is without success, we will proceed after appropriate consultation with the attending neurosurgeon and an appropriate note in the chart. Rarely, if ever, would we cancel such a case. I believe it analogous to the circumstance of failing to insert a pulmonary artery catheter in a situation where one is indicated. Most would not cancel such a case, but would proceed with, perhaps, a degree more anxiety, but certainly not negligently.

There is another group of our peers that contends that central venous catheters for air embolism should be inserted in all patients undergoing craniotomy, regardless of position. True, there is a measurable incidence of air embolism during craniotomy in the horizontal position but, unless the body is to be placed in a distinct head-up position, the risk of a major embolic event is virtually nil and no catheters are required. The logic of those who argue the opposite could be extended, such that all patients undergoing surgery of any kind in the horizontal position should have a central line inserted for air embolism. I reject such arguments as being in the same category as those concerned with unimportant increases in intracranial pressure—a fetish without scientific foundation.

Finally, many of our peers have taken what I consider to be our triumphant experience in conquering most of the problems of the sitting position and have converted it to an argument for condemning the sitting position. They again use words such as malpractice and negligence. But where is the evidence for harm due to the use of the sitting position? Two recent retrospective studies revealed that there are minimum complications, while, in a third study from the Mayo Clinic, Black et al. did a retrospective comparison over the same time period of patients operated in the horizontal versus the sitting position for posterior fossa pathology and reported no important differences, while those few differences that did exist tended to favor the sitting position.

Choice of patient position is a surgical decision. As the responsible anesthesiologist, we may disagree with the surgeon and, if we do, we should express that opinion.
the surgeon insists, we have the choice to either agree or
to withdraw from the case. We do not have the obligation
to "educate" the neurosurgeon regarding the dangers of
the sitting position. Rather, if we accept the case, we have
the obligation to render the use of that position as safe
as possible for the patient. The evidence is clear that we
have the means to do so. We need not continually prove
how prescient that remarkable 15th century painter,
Bosch, was when he painted "The Cure of Folly" depict-
ing intracranial surgery performed on a sitting subject
(fig. 1). Bosch was big on symbols: according to art his-
torians, the funnel identifies a deceitful person or false
doctor, while the closed book symbolizes the futility of
knowledge in dealing with human stupidity. So much for
Bosch and the sitting position.

In my opening remarks, I made reference to the im-
portance of earning the respect of our medical colleagues.
I sometimes think that God had intended Rodney Dang-
erfield to be an anesthesiologist rather than a stand-up
comic. His repetitive lament that "I don't get no respect"
is one that I have heard from anesthesiologists since I
began my residency in 1958. It is true that, when you
embark on a career in anesthesia, you do not auto-
matically gain the respect of your medical peers, your
patients, the lay press, or even of your own family. I re-
member with clarity my mother in 1958 saying, upon
announcing my intentions, "Anesthesia? But I thought
only nurses did that;" and my medical advisor in the navy,
a crusty old four-striper, saying "But lieutenant, you're
good enough to be a real doctor." Things have changed
a lot, and for the better, since 1958, but we will never
have the glamour and the automatic aura of respect that
is conferred upon the brain surgeon, the heart surgeon,
or the transplant surgeon. Neither have we yet gained
the automatic respect conferred upon internists for their
perceived pursuit of the intellectual aspects of medicine.
We are, instead, like that financial institution which ad-
vertises on T.V.—Smith Barney—as regards respect; we
must gain it the old fashioned way—we must earn it. And,
in my opinion, that's fair enough. We gain the respect of
our medical peers and of our patients by demonstrating
our professional skills, our knowledge, and our reliability
on a day-to-day basis while working in the trenches.

As we are required to earn the respect of our associates,
so, too, may we lose any opportunity to gain that respect.
Certainly it is true in the academic world that an individual
who attempts to promote his or her career by taking short
cuts, by plagiarizing the work of others, by fragmenting
a set of data into multiple reports, or perhaps even by
creating false data is quickly recognized by his or her peers
and is shunned. That person is correctly labelled a liar, a
cheat, and a coward, for he or she was without the courage
or fortitude to do what must be done to achieve profes-
sional respect legitimately.

In addition to earning respect in the trenches of the
operating room, the delivery suite, the pain clinic, the
emergency room, or the intensive care unit, we must con-
tinue to strive for respect in the academics of our specialty.
I have highlighted but three areas of my chosen subs-
siclty of neuroanaesthesia wherein academic anesthesia
played a major role in advancing our knowledge and
hence improving patient care. There are, of course, many
other such examples in my own and the other subs-
siclities. I have said that, despite occasional abuses, we can
and should take pride as a specialty in these achievements,
for it is these types of achievement that gain the respect
of our colleagues. However, we must not simply rest on
our laurels. It is my opinion that most of the progress
that has been made has been the result of phenomeno-
logical research. That is, we have explored successfully
the "whats" of the problems and not the "whys." There
is nothing wrong with this. Indeed, it is necessary to first
recognize and describe the phenomenon before one can
explore its causes. I am not ashamed to admit that I am
largely a phenomenologist as regards the nature of my
research, and so too are most of my peers. But now we
need the next generation, the next step. We need indi-
viduals with backgrounds in basic science who can explore
the mechanisms behind the phenomena. Once mecha-
nisms can be clarified, then interventions can be designed
to alter the phenomena in a way favorable to the patients
we care for. The future of academic anesthesia is bright,
but it must now incorporate the basic scientist, as well
as the clinically oriented academic anesthesiologist.
We must replace the pseudoacademicians with true academicians.

In closing, I would like to paraphrase one of my heroes: Sir Winston Churchill. When he was confronted in his waning years with a critic who charged, based upon his highly celebrated habits of beginning the day with a large cigar and cognac and continuing in that vein until the early hours of the next morning, that probably he, Sir Winston, had not drawn an entirely sober breath since his youth, the great man supposedly responded: "Yes, that's probably true, but I believe I've gotten much more out of alcohol than alcohol ever got out of me."

Now, regardless of how one may view such use, or possibly even addictive abuse, of alcohol, the typical pithy symmetry of that Churchillian phraseology struck a chord with me as I contemplated how to bring this to an end. Presumably, I was asked to give this, the 27th Ravenstine lecture, because in the opinion of at least some, I have made a few contributions to the specialty during the past 30 years. I sincerely hope so. But, regardless of how you may perceive any such contributions, I can say this to you and with genuine humility, gratitude, and even apology: although I may be addicted to this specialty, and as such have invested an enormous amount of time and effort in it, I have gotten much more out of anesthesiology than anesthesiology ever got out of me.

Thank you.

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