generally avoids the pretense of omniscience that many patients and families find unsavory. I further explained that one likely scenario was for her to recover to the point of a vegetative state, with possible feeding and tracheostomy tubes, and risks of complications associated with prolonged hospitalization. “Mom would not want to be taken off the ventilator—we think that God will work His miracle.” If she died spontaneously, however, they would let her go without cardiopulmonary resuscitation.

I left the meeting satisfied that we had helped the family in the process of beginning to accept the loss of a loved one. A nurse approached me in front of the coffee machine. “Did you know that (the patient’s) son-in-law, sitting to the right of you in the family meeting, was a patient in this ICU 2 years ago and that you told his wife, sitting to the left of you, that he was unlikely to leave the hospital?” I see hundreds of patients and their families each year. I am really bad with names and faces, beside the fact that I rarely get to see successes—walking, talking, fully functional people who have survived critical illness. “No,” I replied, “but now my opinion probably doesn’t impress them too much!”

The day following our family conference, the patient began to rally. Each day brought a new tiny step in neurologic improvement. One week later, she was opening her eyes. Her repeat EEG showed remarkable improvement. She was extubated and went on to a long recovery (still in the hospital 2 months later). She did not return to the vibrant lady she had been, but she recognized her family and was able to spend quality time with them.

I saw her daughter and son-in-law in the hallway recently—I had gone off service just as she improved significantly and did not have a chance to speak with them about it since the initial family meeting. To my surprise, they seemed very grateful. I acknowledged that I had been wrong in both cases—and that her mother’s case was remarkable. I admitted to learning an important lesson as a result of caring for her. Seeing them sparked a curiosity—could I have been so wrong, so coincidentally? I visited medical records to see what I had documented in the case of the son-in-law. I often write a note when I think care of a patient is approaching futility and document such conversations with families. The son-in-law had had severe multisystem organ failure and sepsis. Although I sensed urgency in my notes, I never documented that I thought care was hopeless. However, I often prepare families for the worst if patients are doing poorly, and likely started that process in his case. The wife had taken away from our discussions that I did not think her husband would survive. In her eyes, I was now 0/2. I was happy that I was wrong, but worried by this degree of imprecision of my prognostic skills. As the Chief of our ICUs, I am expected to be the most knowledgeable, the least likely to so grossly goof.

I read recently in the New York Times Magazine that a neurologist was working with patients in prolonged vegetative states—that he would perform a manipulation and they would suddenly awaken and become communicative. After stopping his “maneuver,” patients would fall back into the abyss. On first consideration, it sounded like hooey to me. Or maybe not.

A fellow countryman once suggested that “a wise man is one who does not pretend to know what he does not know.” Irritated Athenians made him drink hemlock “for corrupting the young.” I often ponder how many patients whose loved ones are told “there is no chance” would have survived to full recovery with best care. This conversation happens countless times each day, in countless hospitals, but infrequently provides the consolation of certainty. My wife, a surgeon, says that to do this job we must do it confidently, with compassion, and to the best of our ability. No one can ask for more. But to stand in the rent between here and there, humility comes in mighty handy. Too much is as-yet undiscovered or unknowable.

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Why Did the Barber Acquire Asbestos?

To the Editor:

The index case had worked from 1964 to 1993 as maintenance fitter in an asbestos factory. He dusted down machines, repaired them, and swept up afterwards. He worked all over the factory. “Asbestos dust lay thick everywhere” was his comment. Danger from asbestos produced no concern in the 1960s, in that no factory precautions were taken. Thus, no masks or caps were worn, machines were not washed down, and there were no extractor fans. There was asbestos-laden white dust outside the showers and changing rooms. A shower would be like “taking a dust bath,” he said. Following legislation in the early 1970s, working conditions at the factory had improved, and the firm began phasing out asbestos products. The fitter was then provided with a cap and mask, but recalls no warning being given to avoid contamination with any dust about the factory, and no one was told that the dust was dangerous. In May 1993, he became ill and has not worked since then. Symptoms and findings confirmed the diagnosis of asbestosis, following employment in the asbestos factory from 1964 to 1993, and he began drawing compensation backdated to 1987.

The special interest arises out of people who were not employed at the factory but who had contact with the index case and coworkers. The fitter took his overalls home, where his daughter shook them free from dust and laundered them. Twenty years later, she was said to have been found to have pleural plaques, presumed related to asbestos exposure. He knew of several men at the factory whose wives had laundered their overalls at home and had had “chest trouble,” but he knew no details.

However, the indirect exposure associated with the most serious consequence related to the barber. Men from the factory often went to the village barber during the lunch break, for a haircut. The barber had worked over forty years locally until retirement in 1987 at the age of 63 years. An account from his assistant employed for his final 18 years said, “The men were often covered with dust, you combed it out of their hair, you worked at hair level, and shook the cloth afterwards.” At no time had the barber worked inside the asbestos factory premises, and (as far as could be determined) any asbestos exposure arose only in his barber shop. He died in hospital in 1992 of pneumonia and mesothelioma (of the left lung), which the coroner decided was asbestos related. The case illustrates the potential for serious asbestos exposure in people (such as the barber) closely associated with workers who were themselves exposed when control measures and risk awareness were largely nonexistent.

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To the Editor:

A recent discussion with resident physicians regarding the relative benefits of various VTE prophylaxis regimens prompted a review of the article in CHEST by Goldhaber et al1 entitled, “New Onset of Venous Thromboembolism Among Hospitalized Patients at Brigham and Women’s Hospital Is Caused More Often by Prophylaxis Failure Than by Withholding Treatment” (December 2000). To my knowledge, there has been no specific correspondence regarding this study, which concluded with the statement that “most deaths from pulmonary embolism (PE) among patients hospitalized for other conditions occurred in the setting of failed prophylaxis rather than omitted prophylaxis.” Several explorations would seem warranted regarding this conclusion.

Goldhaber et al2 described 384 patients who developed venous thromboembolism (VTE) either in the hospital (211 patients) or within 30 days of prior hospital discharge (173 patients). Among this pooled set of patients, 201 had received some form of VTE prophylaxis prior to diagnosis. Details other than the type of modality employed (eg, the dose of unfractionated heparin or the duration of prophylaxis prior to diagnosis) are not provided.

By the definitions used for failure, an assumption is made that the patients who presented within 30 days of hospital discharge (173 patients) would have developed deep vein thrombosis (DVT)/VTE while they were hospitalized and receiving some sort of prophylaxis. If this assumption is rejected, then fully 45% of the patients described cannot be considered as having failed. It is more conceivable that the 173 patients who developed VTE after hospital discharge simply presented with a manifestation of their underlying disease processes while no longer receiving VTE prophylaxis.2

The main conclusion itself, that “most deaths from pulmonary embolism among patients hospitalized for other conditions occurred in the setting of failed prophylaxis rather than omitted prophylaxis” is intriguing but bears closer scrutiny. The authors themselves state that, because the patients they described represented < 0.5% of all hospital admissions, VTE prophylaxis was “quite possibly almost always successful.” Since the most important failure of VTE prophylaxis should be considered fatal PE, an attempt to critically understand the conditions surrounding such failures would be useful. However, some of the data that could contribute to such an understanding are confusing:

1. Among the patients whose death was attributed to PE and “failure of prophylaxis,” it is not clear that these patients were receiving adequate prophylaxis. Inadequate application of an accepted regimen should not be interpreted as having failed.2

2. It is not clear exactly how the course to diagnosis of VTE proceeded in the patients who died, as follows:
   - “...112 patients are described as having PE (62 alone and 50 in association with DVT)....” (in “Results” section of article).
   - “PE was diagnosed primarily by high-probability lung scan (38 patients), intermediate lung scan with high clinical suspicion (50 patients) using revised criteria of the Prospective Investigation of Pulmonary Embolism Diagnosis, or positive pulmonary angiography (33 patients)” (total, 121 patients [in “Materials and Methods” section]).
   - In the 13 patients whose deaths were attributed to PE, that diagnosis was made at autopsy in 8 patients, clinically alone in 2 patients, and by a combination of clinical findings and ultrasonography in 3 patients (in Table 6 of the article by Goldhaber et al1). These data would seem to imply that in none of the fatalities was the diagnosis suspected sufficiently to employ one of the three primary diagnostic modalities.

3. Seven percent of the patients in whom VTE was diagnosed (26 of 384 patients) in this study did not receive any treatment for the disorder. It is not clear that the patients who died were not overly represented within this group that did not receive treatment.

4. It is not possible to discern from the data presented whether, in the cases of fatal PE, any of the patients who failed VTE prophylaxis received diagnoses tardily and/or were treated inappropriately once the suspicion of PE was raised. Because hospitalized patients are likely to be diagnosed and treated promptly for any acute clinical deterioration, this was probably not the case. However, such reassurance is not provided.

It is generally accepted that, once diagnosed, acute nonfatal VTE is at best an uncommon cause of death, recurring in only a small minority of patients, with most deaths actually attributable to underlying diseases.3,4 Therefore, absent the data mentioned, I wonder whether standard VTE prophylaxis, while not 100% successful in preventing all cases of VTE, might actually come quite close to that percentage in preventing fatal PE. Indeed, a more recent article by Arnold et al5 suggests this may well be the case. In that article, the majority of nonpreventable thromboses (ie, thromboses that occurred despite adequate prophylactic regimens) were lone distal DVT.

In summary, the following three points deriving from a critical appraisal of the study by Goldhaber et al1 are likely to be important when considering the concept of VTE prophylaxis failure:

1. It does not follow that patients who go without VTE prophylaxis for any time should be considered as having failed VTE prophylaxis. It is not clear that pooling patients, as was done in the study by Goldhaber et al1, is valid. Further illustrative of this point, as an example, is data showing that extended (ie, out-of-hospital) VTE prophylaxis is useful in hip surgery patients but not necessarily in those undergoing knee surgery.6 Such a distinction further underscores the importance of unique patient characteristics or disease-associated alterations in coagulability.

2. Unique and possibly peculiar individual patient characteristics may be important factors with respect to our evolving understanding of VTE prophylaxis failure. Clinical signs and symptoms, exact methods of prophylaxis, and time to diagnosis and treatment would be useful information that should be available for hospitalized patients. Because hospitalized patients are likely to be diagnosed and treated promptly for any acute clinical deterioration, it is possible that routine standard VTE prophylaxis may be close to 100% effective in the prevention of fatal PE in the hospital.

3. I believe there is persistent contention regarding whether subcutaneous unfractionated heparin, administered two or three times daily in doses of either 5,000 or 7,500 U, is adequate prophylaxis in any population of hospitalized patients.

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