

“We can’t kill your mother!”

and Other Stories of Intensive Care

Medical and Ethical Challenges in the ICU

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“Shock Him!” in *The Saturday Evening Post*, June 1996

DEDICATION:

TO ALL THE PEOPLE WHO WORKED
WITH ME AT MT. SINAI HOSPITAL --
YOU DID A GREAT JOB.

Part 1- (pages 1-111)

“We Can’t Kill Your Mother!” and Other Stories of Intensive Care

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PREFACE

Who is this book for?

In the early 1980s I wrote a story about an extremely ill patient cared for in our medical intensive care unit (MICU) at Mt. Sinai Hospital. Called “A Case For Intensive Care,” the story was for a general audience and appeared in a local college literary magazine. Until then all my published writing had been for doctors only, and I wanted to explain a complicated medical case in a way that anyone could understand.

In the ensuing years I wrote many other patient-centered stories, intended for a general audience; all the published stories, plus several as yet unpublished ones, are included in this book. Writing stories is an enjoyable diversion from medical papers and textbooks, with their dry, leaden prose. The stories allow a latitude of style and expression not possible in scientific prose.

So this book is for the general reader -- no special background or medical expertise necessary. In fact the only ‘requirement’ is an interest in humanity. Illness and medicine are universal and everyone has some familiarity with hospitals, if only from the position of consumer. Most people have, at some point, either been hospitalized or visited a family member in the hospital. These stories take you inside the medical intensive care unit, a major part of every acute care hospital. That’s the setting, but the subject is people and their serious (and sometimes strange) afflictions.

Who are the patients?

The stories are based on real patients cared for in the medical intensive care unit of Mt. Sinai Hospital, at one time a major Cleveland teaching hospital. For many years our hospital had the best statistics in the region for ICU mortality. Sadly, the hospital closed its doors in February 2000, a victim of changing demographics and other factors. However, all these stories were written when the hospital was thriving, and thus frequent use is made of the present tense. To preserve patient anonymity all names have been changed as well as some of the descriptive details.

The first chapter gives an overview of intensive care rounds and how the MICU operates. Succeeding chapters are devoted to one or two patients and the challenges they present. Like Harold Switek, too ill to leave MICU, too psychotic to stay. And Willie the Yellow Man, whose love affair with alcohol exceeded anything you’ve ever seen. You’ll meet a young socialite hospitalized with rapid onset of total paralysis and wonder — as we did — will she ever hug her kids again? And another woman about to have her baby

during a terrifying asthma attack. Then there's the young accountant who slept in a coma – for six months! Another story relates the strange saga of a man who claimed to be coughing up blood, only that wasn't his real problem.

As in most hospitals today, in Mt. Sinai the *medical* ICU was separate from the *surgical* intensive care unit, the latter staffed for post-operative patients and trauma victims. MICU is also separate from the *coronary* care unit, where patients are sent with heart attack and other cardiac emergencies. Whatever the physical arrangement, every sizable hospital handles the same problems and encounters the same ethical dilemmas as presented by our patients. Like elderly, senile Mr. Zigson, who is trying to die a natural death. Only problem: he has no family. Should the doctors leave him alone or 'do everything'? And the nursing home patient who is the subject of the title story; she is awake and alert, but can only live connected to a breathing machine. Her daughter demands that the ventilator be disconnected so "mother can die." Can doctors honor such a request? Can they *ignore* it?

Should a physician write about his or her patients?

Emphatically, yes, if he or she is so inclined, and provided that privacy is maintained. I am not the first, and will certainly not be the last, medical professional to write about his or her patients. In a literary sense doctors and nurses are privileged; what we see in our daily jobs is more than enough to fill many interesting books. We just have to find the time and inclination to tell others about what we do, and to make the work seem as interesting in print as it is in real life.

Lawrence Martin, M.D.
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1. ROUNDS

I greeted the two new interns. “Welcome to MICU. I’m Dr. Martin. I run the unit and will be rounding with you this month. How does it feel to be starting your internship?”

“Scary,” said Deborah Hafly, a petite and energetic woman who came to Mt. Sinai with top recommendations. She and her partner on this rotation, Michael Highland, were both excellent students and promised to be good interns.

It was July 1, the beginning of another academic year at Mt. Sinai Medical Center. As Director of Mt. Sinai’s medical intensive care unit, it’s my job to supervise and teach house staff and help manage patients admitted to our 8-bed ward.

“You’ve met the medical resident Jerry Clark, and been assigned your patients?”

“Yes,” said Deborah. “He assigned us our patients this morning. We each have four.”

“Good. Well, let’s make rounds.”

MICU occupies a large rectangular space on the second floor of the hospital. The “unit,” as it is often referred to, consists of eight single-bed rooms arranged in a broad-based ‘U’ shape, in the center of which is the nursing station. On either side of the nursing station double doors lead to the hallway and family waiting area.

All the patient rooms are fronted by sliding glass doors that can be ‘broken’ open for quick access; drapes across the doors provide privacy when necessary. Each patient can be ‘wired’ so that his or her cardiac rhythm is continuously displayed on monitors at the nursing station.

The nurse to patient ratio in MICU is as high as one-to-one when every patient is critically ill. Despite the appellation ‘intensive’ not all MICU patients are critical. On average, when the unit is full, five nurses per shift can provide excellent care. On the regular hospital wards at Mt. Sinai, the average ratio is one registered nurse per eight patients.

MICU rounds are open to anyone on the staff who may have something to contribute. Besides myself (or another staff physician) rounds include two interns, the supervising resident, one or more nurses, and a respiratory therapist. Also participating, on occasion, are medical and nursing students, various consultants and private attending physicians, and a social worker.

If Mt. Sinai was not a teaching hospital my job would be much more difficult, perhaps impossible. Most MICU patients require constant management, something not easily done over the phone, or even during brief hospital visits. Physicians must always be available to order medications,

adjust ventilator settings, put in catheters, talk to families, examine and treat new admissions, and transfer patients to the regular wards. Private, office-based physicians who send their patients to MICU are thankful for the housestaff and round-the-clock physician coverage. Without interns and residents we could not provide the excellent care Mt. Sinai's MICU is known for.

House staff, although licensed MDs, are in training and not certified in any specialty. They must be supervised throughout their three or more years of hospital internship and residency. Interns, fresh out of medical school, are supervised by the junior resident, he or she by the senior resident, and all the housestaff by the chief resident, full-time staff and visiting physicians.

Physician training is a dynamic, patient-centered process. Lectures occupy no more than an hour a day of house staff training. Most of the learning comes from supervised, hands-on patient care, supplemented by reading journals and textbooks.

* * *

I took the new interns over to room #1 where we met Dr. Clark and the MICU head nurse.

"Let me introduce you to Marsha Ligner, MICU's head nurse," I said. "Marsha, this is Deborah Hafly and Michael Highland, our two new interns."

"Welcome to MICU," she said. "Glad to have you aboard." Turning toward me, Marsha continued, "Dr. Martin, who can we transfer out this morning?"

"I don't know. Do we need a bed right away?"

"Yes. The ER just called. They have an overdose that needs to come up."

I turned to Dr. Clark. "Who can go out?"

"I just put Mr. Jones up for transfer. As soon as a bed's ready upstairs he can go."

"OK. Marsha, find us a bed for Mr. Jones. And please ask Patient Placement not to drag their feet. I know there are empty beds on the wards." Marsha nodded. She would make one or two phone calls and Mr. Jones would soon be transferred.

"Is the ER patient intubated?" I asked.

"Not as far as I know," said Dr. Clark.

"OK. Well, let's start rounds. We'll see the new patient as soon as he arrives. Or is it she?"

"A twenty-year-old woman. She OD'd on tricyclics."

I stood with my back to the sliding glass doors of Room 1, chart rack and housestaff before me. We were also joined by two of the MICU nurses and a respiratory therapist.

"Has everyone met Dr. Hafly and Dr. Highland?" Everyone had. I

addressed the two interns, the only new people on the team. “We round at ten each morning. You should be up to date on your patients by the time rounds begin. Today is an exception of course. Also, we require that you write a chart note on each of your patients every day. You need to list all their medical problems, all drugs they are receiving, and all the tubes entering or exiting their body. Dr. Clark already went over this requirement with you, didn’t he?” They nodded yes.

“Good. Jerry, why don’t you briefly present each patient as we go around.” Jerry Clark, 28, had been the MICU resident in June and was staying another day to orient the new interns. He knew all the patients.

“OK. In room one we have Mr. Hewlett Jones. He’s a sixty-seven-year-old man admitted June twenty seventh, with a CVA.”

“What’s a CVA?” I asked Deborah.

“Cerebrovascular accident,” she answered matter-of-factly.

“Jerry, did Mr. Jones have an accident?” I wanted to send a signal early in the month: avoid jargon if possible.

Dr. Clark showed a knowing smile. He had been through this routine with me before. In the spirit of the new year he played it straight – almost.

“No, Dr. Martin,” he said, with a trace of sarcasm. “He had a stroke. There was no accident.”

“I see. Then why do you call it a ‘cerebrovascular *accident*?’ Why didn’t you – why don’t we – just say Mr. Jones had a stroke and be done with it?”

The interns stared in mild disbelief. What kind of rounds were these? English 101? Every new doctor has heard the term ‘CVA’ a hundred times, always indicating a stroke of some sort. Drs. Hafly and Highland had never before heard anyone *question* the term.

“I don’t know,” admitted Dr. Clark. “That’s just what everyone calls it. I know it makes no sense.”

“I agree. It’s just one of those terms that gets introduced into medicine, and no one ever questions. OK, go on.”

“Well, he had a stroke, a spontaneously-occurring blood clot that blocked his left middle cerebral artery. The clot paralyzed his right side and left him aphasic, but I think he’s getting better. Neurology’s following him and he’s ready for transfer.”

We went in to Mr. Jones’s room to say good by. Reflecting the crossover of nerve pathways, the right side of his body was limp from a blockage in the left side of his brain. Since the speech center is on the left Mr. Jones couldn’t talk, but he recognized us and understood conversation. I explained that he was being transferred out of MICU, that he was improving and with continued physical therapy had an excellent prognosis for recovery. He understood. We left Mr. Jones and rolled the chart rack over to Room 2, stopping in front of the closed glass doors.

“This is Mr. Boykin,” said Dr. Clark. “He’s a thirty-four-year-old man

admitted June twenty ninth with a severe asthma attack. He has improved but we want to continue IV steroids and inhaled bronchodilators another day. His peak flow is up to one forty.”

Through the glass we saw a young man in mild respiratory distress, a state made apparent by a fast breathing rate.

“Who’s got Mr. Boykin?”

“Deborah.”

“OK. Deborah, did you learn about peak flow at your medical school?”

“I didn’t have that much experience managing asthma patients. I think I only had one asthmatic on my medicine clerkship.”

“Well, you’ll become an expert here. Peak flow is the best single breathing test to follow the progress of an asthmatic. It takes only a few seconds and if done properly the test is fairly reproducible.”

I asked Greg, our respiratory therapist, to get the peak flow meter so I could demonstrate the test. He went into Mr. Boykin’s room and brought back a round, metal instrument the size of a small kitchen clock. A handle on the side of the peak flow meter allows the patient to hold the instrument horizontal while blowing into a mouthpiece situated above the handle. A long needle on the face of the meter deflects when air is blown into the mouthpiece; the harder the blow the greater the deflection. A slight ‘puff’ into the mouthpiece by a normal adult will register at least 150 liters/minute peak flow. A maximal effort will register at least 400 liters/minute.

I inserted a cardboard mouthpiece into the meter and handed it to Deborah. “Deborah, just put your lips around the mouth-piece and give a little puff.” She did as instructed and the needle deflected to 180.

“Now reset the needle and take a deep breath, then blow out with all your strength.” The needle went to 495.

“OK. Now let’s go see Mr. Boykin.” He sat in bed, a strong, virile patient humbled by his asthma.

“How do you feel? Are you any better since you’ve come in?”

“Oh, much better,” he said, with conviction. But how much better? You can be fooled by patients. A 30% improvement from a severe asthma attack can make the patient feel like a million bucks, at least at rest. He was still breathing faster than normal and I heard wheezing on exam.

I inserted a fresh mouthpiece and asked Mr. Boykin to do the peak flow maneuver. He took in a deep breath and blew as hard as he could: 170. I asked him to repeat the effort. The needle went to 168.

“Well, you still have a way to go. Your peak flow is still reduced. We’re going to keep you here today and continue the intravenous medication. You might be able to go upstairs tomorrow.” I thanked him and we stepped outside.

“What do you think?” I asked the interns.

“I’m surprised,” said Dr. Highland. He doesn’t look that short of breath.”

“I agree. I think he may have chronic asthma. The only way to gauge severity of asthma is with the peak flow or some similar measurement. Despite maximal effort he couldn’t get above 170 on the peak flow.” I looked at Deborah. “You did better than that with almost no effort. He looks strong but if he ran a race with you right now it would be no contest.”

“What’s going to happen to him?” asked Deborah.

“Too soon to tell,” I said. He’s much better than yesterday, that’s for sure. If he can’t reach a higher peak flow despite several more days of IV therapy, then his impairment is chronic. He used to smoke heavily so that may be contributing. Anyway, it’s too early to say. We’ll watch him in MICU one more day, then send him upstairs if he remains stable.”

We moved to room 3. “This is Mr. Denton Smith,” said Dr. Clark. “He came in last night with a gastrointestinal hemorrhage. He’s a heavy alcoholic. GI’s already ‘scoped’ him.”

“What’d they find?” The gastroenterology service is good at putting ‘scopes,’ long flexible tubes with a light on the end and a channel through the middle, into the stomach of bleeding patients.

“A large duodenal ulcer. Here’s a picture.”

Dr. Clark opened up the chart. Taped in the middle of a progress note was an amazingly sharp, digitized photo of an intestinal ulcer. In the middle of a normal stomach lining sat a white dime-sized patch, and in the middle of that, a tiny dab of red. I read the handwritten legend under the photo.

“Eroding gastric ulcer with bleeding vessel as shown. Vessel cauterized.”

“Has his bleeding stopped?”

“Yes, but GI wants us to observe him for another day. We’ve given him a total of three units of blood.”

“OK. I see in the note that he has continued to drink. Was he drunk when he came in?”

“No, he says he hasn’t had anything to drink in three days. So we’re also going to watch for DT’s [delirium tremens].”

“Didn’t anyone ever tell him to quit drinking?” I asked. It was a rhetorical question. “What’s his hematocrit?”

“It was 23% on admission. After the three units it’s up to 30.”

Suddenly two nurses from the station began running toward room 7. The first to arrive punched open the door and the other one hauled in the red crash cart. We arrived seconds later. The patient was Mrs. Waldstein, a 76-year-old woman with end-stage kidney and heart disease. The day before she had received a shunt in her right arm for kidney dialysis. We had spent considerable time with her and her family discussing such issues as quality of life, what she could expect with dialysis, possible therapy without it, and so on. In the end she said she wasn’t ready to die or become a vegetable, and accepted dialysis.

Even so, her kidney doctor was concerned about whether her heart was strong enough to withstand three times a week dialysis. She had suffered

two heart attacks in the past year. Two days earlier she was admitted to MICU with pulmonary edema – excess fluid in the lungs from kidney and heart failure. Now her heart had suddenly stopped beating altogether. If we did nothing in the next two minutes she would be dead.

“Ambu bag!”

“Epinephrine!”

“Call anesthesiology!”

“They’re called.”

“Get the EKG. Let’s get a rhythm strip.”

Dr. Clark positioned himself at the head of the bed and began ventilating her with an Ambu bag while I took up chest compressions. One of the nurses began infusing epinephrine into an arm vein while another stuck Mrs. Waldstein’s femoral artery for a blood gas sample.

“Any heart beat?”

“Stop pumping for a second.” My pumping was creating an artificial heart beat which could mask the patient’s own.

“I’ll give you three seconds,” I said. “Anything more and she’s lost.”

“Still flat line.”

“Give an amp of calcium.” The nurse handling drugs infused the calcium.

“Give an amp of bicarbonate. She’s acidotic from her renal failure. Somebody please listen to her chest.” Deborah complied.

“Good breath sounds when you’re bagging,” Dr. Martin, we’ve got a rhythm. Looks idioventricular.”

“Any pulse?”

“Only when you pump. Can you stop for a minute?”

“Impossible. A minute is eternity.” I stopped for four seconds.

“I feel something. Let me check her blood pressure.”

The anesthesiologist arrived. Good, I thought, it’s Josh; he’s one of the best. Anesthesiologists are expert at intubating patients, so we always call them for a cardiopulmonary arrest. By now there were at least seven people in the room.

Josh went to the head of her bed.

“Hold off intubating for a second, Josh,” I said. “Let me see what her rhythm is.”

“If she’s got a pressure you still want her intubated?” he asked.

“Yes. I want to make sure she’s adequately oxygenated and ventilated. This can happen again. What’s her blood pressure?”

“I’m getting 90 by palpation,” said one of the nurses. Do you still want epi to run in?”

“Yes. Now, let’s get her intubated.”

Josh expertly slipped the foot-long tube into Mrs. Waldstein’s throat while I held my pumping. Seconds later I resumed chest compressions. Dr. Clark continued bagging her, only now he was pumping fresh air through the

endotracheal tube, a direct conduit into her lungs.

“Looks like a nodal rhythm, Dr. Martin.”

“Good. I’ll stop pumping.” I felt for a pulse in her groin and felt a repetitive thump against my finger tips. “Look’s like she’s gonna make it. Let’s get a ventilator hooked up and also another blood gas. Stop the epinephrine.”

We stayed in Mrs. Waldstein’s room another twenty minutes, to make sure she was stable. The nurses’ initial response to the cardiac arrest was so quick I doubted she had suffered any brain damage.

* * *

On leaving Mrs. Waldstein’s room I noted that Deborah and Michael were staring rather idly at the cardiac monitor, Michael with hands in his pockets. They felt insecure in the midst of this emergency and I sought to reassure them.

“Don’t worry. By the end of the month you’ll know exactly what to do. I promise.” Affecting nonchalance I added, “Let’s resume rounds.” We moved on to room 4.

“This is John Popola,” said Dr. Clark. “He’s seventy-two, with end-stage Alzheimer’s. He was sent here for pneumonia and respiratory failure. His sputum culture’s growing *pseudomonas aeruginosa*. We have him on gentamicin and piperacillin. We can’t get him off the ventilator until his pneumonia clears. He’s DNR.” Before us was a man looking perhaps ten years older, white hair, face grizzly, eyes sunken in. He was not awake, an effect of sedation given to relieve respiratory distress.

“You two know what DNR means, I assume.”

“Do not resuscitate,” said Deborah.

“Right. So why’s he connected to a life support ventilator?” I asked.

There was no answer.

“Jerry, if he’s ‘Do Not Resuscitate,’ why the ventilator? Isn’t that a form of resuscitation?”

“He wasn’t DNR until after he was intubated. Then the family decided they didn’t want any more heroics. They don’t want him resuscitated again if his heart stops or he crashes. So we made him DNR.”

“What family?”

“His wife is deceased. We talked to a sister and his son. They both agreed.”

“Michael, how does being DNR affect the care of a ventilator patient?”

This was not a fair question for the first day of internship, but I wanted the interns to think about it anyway.

“I don’t know,” he said.

“It just means we don’t add more life support,” I explained. “Otherwise it doesn’t affect the care at all. We’ll treat Mr. Popola’s pneumonia in the

usual way, and will do our best to get him safely off the ventilator. In some circumstances no treatment may be given a DNR patient, but that's not the case with Mr. Popola."

"Does the family have to sign for DNR status at Mt. Sinai?" Michael asked.

"No. You just have to write a note in the chart documenting that you talked to the patient if he's competent, or to the family if the patient is not."

We reviewed the ventilator settings and blood gases, then all of Mr. Popola's medications. There was still a way to go before he could breathe unassisted by the machine. We moved on.

"In room five we have Elsie McKnight," Dr. Clark said. "She's a tylenol overdose."

"Looks like a young woman to me," I said. At that, Dr. Clark rolled his eyes and made a here-we-go-again face. I ignored him.

"Suppose a patient pointed to you and said, 'There's a stethoscope,' or 'here we have a reflex hammer.'"

"OK, OK," Dr. Clark said, in a manner of 'enough, enough.' Actually he took my comments good-naturedly. They were really intended to impress the interns and, perhaps, change in some minuscule way the language of medicine. Doctors already well into their post-graduate training, like Dr. Clark, were usually beyond my message.

"Miss McKnight is a twenty-five-year-old woman who *took* an overdose of Tylenol tablets. When she came to the ER they measured her acetaminophen [tylenol] level; it was twenty-four."

"Michael, do you have any question about that? What would you want to know at that point?"

"Was she breathing?"

"No, I don't mean about her vital signs. Obviously you want to know if a patient is breathing, if her heart is beating, and so forth. I mean, given that she took tylenol and you have a blood level of the drug, what specific question should you ask?"

Jerry thought for a moment. "What else did she take?"

"Well, that's important too, but let's assume it's only tylenol. It is, as far as we know, isn't it Jerry?" Jerry nodded yes.

"OK, it's only tylenol. What specific question do you need to ask?"

"I'm not sure what you're getting at, Dr. Martin."

I turned to the other intern. "Deborah?"

"When did she take the pills?"

"EXACTLY. You need to know *when* she took the pills because treatment depends on that information *and* the blood level. What's the story, Jerry?"

"The blood tylenol level was drawn about six hours after she took the pills."

"OK. What would you do?" I addressed both interns. Deborah spoke

up first.

“At that level I would definitely give acetylcystein.”

“Right. We gave it to her,” I said. “Otherwise, what can happen?”

“Severe liver toxicity.”

“Right.” Clearly, of the two new interns Deborah was the sharper one.

* * *

We went in to see Ms. McKnight. Not the most pleasant person, she so far had refused to talk to anyone. A tall, thin, flat-chested young woman, she sat up in bed with arms crossed and glared straight ahead. Based on a suicide note and the number of pills she took, her attempt was no gesture. She was angry because we saved her life.

“How do you feel?” I asked. She looked at me, then away, and did not answer. The cardiac monitor above her bed showed normal vital signs. By now her risk for liver toxicity was minimal because of the treatment she received from acetylcystein. This drug which prevents tylenol from forming a toxic metabolite. As soon as she could be evaluated by psychiatry she would be transferred from MICU. I saw no point in spending more time in her room. We moved on to room 6.

“Here we have the strangest case of all. Everyone meet ‘Jane Doe,’” said Dr. Clark.

“That’s not her real name, is it?” asked Deborah.

“No. They found her in a parking lot. Comatose, no identification. She’s been here since yesterday morning. I’m told she was intubated in front of a Cadillac. Anyway she has severe aspiration pneumonia and is on the ventilator with one hundred percent oxygen. Right now, except for Mrs. Waldstein, she’s our sickest patient. She’s got a chance to make it. Nothing here that’s irreversible.”

“Jerry, did you call the police?”

“Yes, I called and asked if there is a missing persons report on someone of her description. A black woman about sixty years old. They haven’t got back to me yet.”

As we talked the interns took notes. They seemed over-whelmed, but in less than two days they would know everyone in detail, including any new arrivals.

We stopped in room 7 to see Mrs. Waldstein again. Her cardiac rhythm and blood pressure were holding steady. Arterial blood gases were adequate, albeit with artificial ventilation. She seemed in no immediate danger so we moved on to room 8.

“Last but not least, room eight, Marie Jackson. Very sad case,” said Dr. Clark. Before us lay an 80-year-old woman completely comatose and connected to a ventilator.

Jerry opened her chart and pointed to the top of one page. “What do you

see there?" he asked the interns.

"A date."

"What's the date?"

"May third."

"Two months. She came to Mt. Sinai on May third for a dementia workup. Her private [physician] ordered all the right tests, but nobody ever asked her or her family about what to do if she needed resuscitation. Well, she went for a CAT scan of her brain and guess what happened?"

"She arrested?"

"Right on the table. It was a mess, trying to get her intubated. To make a long story short she must have been apneic for a good five to ten minutes. After her cardiac arrest, which was on May sixth, she developed every complication. Pneumonia, kidney failure, sepsis. We've treated everything. Family won't let go. Neurology agrees she has severe hypoxic encephalopathy, with almost zero chance for meaningful recovery. Actually they said that on June 1. Here we are a month later."

Ms. Jackson – tube in throat, life supported by machine – had her eyes open but demonstrated no awareness of us or of her surroundings. She just stared past us. Periodically there was a twitching, writhing movement of her face and mouth, an indication of partially suppressed seizure activity.

"Why does she need a ventilator?" asked Deborah.

"Good question," I said. "Hypoxic brain damage by itself doesn't usually require artificial ventilation. Unfortunately, her pneumonia was so severe that her lungs became permanently damaged. She probably has also some emphysema, from years of smoking. Anyway we can't get her off the machine.

"What happens when you try to wean her?" Deborah asked.

"We tried once. She lasted a day and then developed respiratory distress. We gave her family the option of not connecting her back to the ventilator, but they couldn't agree. Some relatives said yes, some said no. Finally guilt prevailed. They asked us to reconnect her. So we are not even trying to wean her from the ventilator. She would probably arrest again and it would be a bad scene."

"There's also the problem that it happened in the hospital," Dr. Clark added.

"Yes," I said. "But the family's not talking lawsuit or anything. It's just that because it happened here everyone is skittish about pushing them to let her go. I'd love to get Mrs. Jackson out of MICU but the ward isn't ready for her just yet."

The interns just shook their heads. It would take time to adjust to this reality of modern medicine: with all our machines we sometimes do more harm than good.

"Well, let's go look at x-rays. Afterwards you can come back and get to know your patients in more detail." We went to the x-ray viewing room

across the hall from MICU. About ten minutes later the phone rang in the viewing room. One of the house officers answered the phone and took a message from MICU, then relayed the information to the rest of us.

“The overdose is here.”

Comment

In these stories dialogue is presented pretty much as it is spoken on rounds. You are right to be offended if you ever hear patients referred to as a diagnosis or organ; phrases like “this overdose,” “that gallbladder,” and “the heart,” when referring to specific patients, are abhorrent. Unfortunately, doctors and nurses are incorrigible users of jargon and it is not an easy habit to break. I apologize for any conversation that may offend. Despite the way some doctors and nurses may occasionally communicate with one another, in my experience they invariably speak to patients and families in a manner that is most respectful.

-- END --

2. OVERDOSE

Judy Bilowitz was only 20 when she came to MICU but this was not her first hospital admission. She was diagnosed as a “depressed personality” shortly after puberty. As a teenager she spent two long periods in Weathergill Pavilion, the state’s top psychiatric hospital. Judy came from a prosperous family and could afford the best care.

With the aid of expensive tutoring Judy made it through a private girls’ prep school, graduating at 19. Unlike most everyone else in her class she did not go to college or take time off for travel. Instead she stayed home with her parents and 15-year-old brother, an out-going and mentally healthy sophomore.

Judy’s father owned a scrap metal company and her mother was on the board of several important charities. The parents’ financial and social success only heightened the pain of Judy’s illness; their older child simply held no promise. She had no interest in college and was too withdrawn to find and keep a job.

Judy also had little interest in boys, nor they in her. Though attractive physically — she possessed a slim, well-proportioned body, fair complexion and features that made for a pretty face, with straight brown hair — her inattention and blunted affect tended to repel the opposite sex. Boys unaware of her psychiatric illness usually considered her ‘screwed up,’ or ‘weird.’

She was not a virgin. At 15 she became pregnant and had an abortion in her eighth week. She was in Weathergill at the time of conception and the offender was thought to be another patient. Tightened supervision during her second hospital stay, at age 17, prevented another sexual liaison. As far as her parents knew Judy used no birth control.

To the outside world Judy at 20 didn’t seem to care much about anything. She was incapable of relating to others and had few identifiable interests. Despite every material advantage there was little to occupy her time. She stared at TV much of the day, sometimes read or pretended to read (all her books had pictures), and occasionally worked in the garden.

She had been under the care of three psychiatrists since puberty. Her current therapist was Dr. Erasmus Cohen, a medical school faculty member in his late 30’s and, at the time of Judy’s MICU admission, considered the ablest psychiatrist on Mt. Sinai’s staff.

Dr. Cohen’s assessment was that Judy suffered from ‘schizo-affective disorder associated with depression,’ a form of psychosis usually treated

with medication. In Dr. Cohen's best clinical judgment she stood to benefit from Triavil, a combination of the antidepressant *amitriptyline* (also marketed alone as Elavil) and the anti-anxiety drug *perphenazine*.

Triavil comes in various dosages; the dose Dr. Cohen prescribed for Judy was 2-25, twice a day. Each 2-25 tablet contains 2 milligrams (mg) of perphenazine and 25 mg of Elavil. Judy's prescription began in late March, a little over three months before she ended up in MICU.

Judy faithfully took the Triavil and seemed to improve. She became more talkative, took trips with her parents, and joined a local gardening society. Because of the favorable response Dr. Cohen renewed the drug monthly; her last prescription for 60 tablets was filled June 20.

On July 1 Judy didn't come to breakfast as usual. At about 8:30 the maid went to Judy's room and found her unconscious on the bed, the empty Triavil container beside her. There was no suicide note. From this information it was deduced that she took about 40 of the Triavil tablets.

Judy Bilowitz was Mt. Sinai Medical Center's first overdose of the new academic year.

* * *

Overdoses can be classified as intentional or accidental. Accidental overdose occurs when too much of a drug is taken by mistake; this occurs mainly among children and the confused elderly.

People who intentionally overdose are usually suicidal, although occasionally an excess of drugs is taken "just to get some sleep," or "to cure my headache." Most of the overdose patients admitted to MICU are, like Judy Bilowitz, intentional and suicidal. Only about half leave behind a suicide note.

Would-be suicides may choose either prescription or over-the-counter drugs. OTC drugs such as aspirin and tylenol are, of course, toxic in large doses and can be lethal. Prescription drugs commonly overdosed, besides Triavil, include Elavil and other single-agent antidepressants, Valium and other anti-anxiety medications, lithium (used in manic states), dilantin (seizures), theophylline (asthma), and Darvon (pain).

Elavil belongs to the group of 'tricyclic antidepressants' (TCAs), so called because of their three-ring chemical configuration. TCAs are indicated for "endogenous" depression, a state unrelated to external events. People depressed over real-life problems such as loss of job or divorce generally do not benefit from medication.

The usual Elavil dose for outpatients is 50 to 75 mg a day, although twice this amount can be prescribed in some cases. For inpatients, as much as 300 mg a day can be used.

A combination drug like Triavil is particularly helpful in some psychotic patients. The tricyclic fights the depression while the tranquilizer combats

any tendency to agitation.

Tricyclics by themselves are also used for other conditions. Imipramine, the first available TCA, is now widely used for childhood enuresis (bed wetting). Elavil, the most frequently prescribed TCA, is also used for chronic pain syndromes.

It is for endogenous depression, however, that TCAs are most often prescribed and that use of the drug presents the greatest hazard. The hazard exists because depressed patients are often suicidal and TCAs are lethal in large doses. The widespread use and potential toxicity of TCAs, plus the nature of the patients, account for three sobering statistics: an estimated 500,000 Americans overdose on TCAs each year; TCAs are the number one cause of fatal overdose; over 70% of successful TCA suicides are pronounced dead *before* reaching the hospital.

It must be accepted that the risk of suicide exists in any severely depressed patient. If the treatment of choice for depression was bottled water we would no doubt see patients suffering water intoxication. They would be bloated but few, if any, would die. Instead the treatment for many patients is a drug that is potentially lethal in large amounts.

The manufacturer's Product Information Guide to Elavil states:

High doses may cause temporary confusion, disturbed concentration, or transient visual hallucinations. Overdosage may cause drowsiness; hypothermia; tachycardia and other arrhythmic abnormalities; congestive heart failure; dilated pupils; disorders of ocular motility; convulsions; severe hypotension; stupor; and coma.

* * *

Judy Bilowitz arrived at Mt. Sinai's emergency room at 8:55 a.m., comatose. While one physician took a history from the parents others intubated her and began artificial ventilation. Her stomach was evacuated with a large bore nasogastric tube and then aspirated to remove any residual pill fragments. Activated charcoal, a drug absorbent, was put down the tube to bind any tablets not yet absorbed into the blood.

The ER physicians placed her suicide attempt between 11 p.m. June 30 and 7 a.m. July 1. Had she swallowed the pills after 7 a.m. she would not likely have been in coma 90 minutes later.

It took the ER doctors about two hours to stabilize Judy and transfer her to MICU. I saw her for the first time as she was wheeled into room one. My first impression was that she looked like a true suicidal patient: young; comatose; pale skin without makeup; hair all frizzed; face distorted by two tubes, one in the mouth and the other in the nose. From just visual inspection I surmised Judy's overdose was no "gesture."

The clear plastic stomach tube, inserted through her left nostril, was now jet black from the charcoal absorbent. This tube would stay in place at least two days so she could receive additional charcoal every 6 hours. The endotracheal tube stuck out from the left side of her mouth and was secured with white adhesive tape that circled her head; it would remain until she could safely breathe on her own.

The physical exam, which I did with the intern Deborah Hafly, can be summarized as follows:

Body temperature: 97.6 degrees.

Pulse: 116/minute (increased)

Blood pressure: 123/82 (normal)

Respiratory rate: 16 (provided by ventilator)

Head: no bruises or any sign of trauma.

Eyes: closed; no eye makeup; pupils dilated equally and reactive to light; retinal exam normal.

Ears: normal.

Neck: no stiffness; carotid pulses equal and strong.

Heart: normal, except for fast heart rate of 120 beats per minute.

Abdomen: no tenderness or swelling.

Arms and legs: normal; no needle marks or scratches.

Genital area: Bladder catheter (inserted in emergency room) draining clear yellow urine.

Nervous system: in deep coma; no response to her name but responsive to arm pinching by withdrawing her limb; tendon reflexes equal and hyperactive in all extremities. Eye reflexes and eye movements appropriate, indicating no structural brain damage.

* * *

After our exam we checked results of tests obtained in the emergency room. Her EKG showed no arrhythmias but the 'QRS' wave pattern generated by the heart's conduction system showed slight widening, a common finding in TCA overdose. We would watch this closely since further widening could signal impending cardiac arrest.

Her chest x-ray was clear, ruling out pulmonary complications such as aspiration pneumonia and pulmonary edema. An arterial blood gas drawn shortly after intubation showed adequate oxygen and carbon dioxide levels. Other blood tests showed no electrolyte imbalance or disease of the kidneys, liver, or pancreas. Her pregnancy test was negative.

"Well," I said to Dr. Hafly, "she looks stable for now. She'll need an arterial line, which the resident can help you with. We'll need to check her blood gases throughout the night. Our job is to support her ventilation and watch for cardiac or neurologic complications. She took about 40 tablets.

That's enough Elavil to kill her."

"What about the other ingredient, the perphenazine?"

"I'm not so worried about that. Perphenazine's probably contributing to her coma but it doesn't have the same lethal cardiac and neurologic effects as the tricyclics, at least not in the dose she ingested. By the way, is there an antidote for Elavil overdose?"

Dr. Hafly thought for a moment. "I don't know. I guess not, or she would have received it by now."

"Right. But if a patient develops life threatening side effects, particularly cardiac, we sometimes use IV physostigmine. It blocks the stimulatory effects of the Elavil."

"Why don't we use it now? Her QRS [EKG wave form] is slightly widened."

"Physostigmine has its own side effects and can be dangerous. Besides, it's not an antidote, just another drug to block Elavil's nastier side effects. The EKG should improve as the drug leaves her body. After you put in the A-line, read the review on TCA overdose. A copy's at the nursing station."

I gave her the exact reference and went to the waiting area to meet Judy's parents.

* * *

Her parents impressed me as decent, hard working people, upper middle class or perhaps even wealthy but not at all pretentious. Of course no one is pretentious when their child is hospitalized, but I have seen parents whose life style and ostentatious behavior seemed to explain their child's psychopathic behavior. I didn't feel that way about Judy's mother or father. As a parent myself I felt sorry for them. Sorry that they had such a burden of a daughter, that they were denied the pleasure of watching her grow and mature normally and, far worse, that they might lose her to a fatal overdose. I tried to stay professional and show empathy at the same time.

"Right now she's stable," I said. "She's listed as critical but her blood pressure and heart are holding up and all her vital organs are working except for her breathing. The machine will breathe for her until she comes out of coma."

"What are her chances, Doctor?" The questions came from Judy's mother.

"Well, I can't give a definite percentage but I'd say they're better than 50-50. The fact that she reached the hospital alive is a good sign."

"What do you mean?"

"About 70% of people who overdose on drugs like Triavil die before they reach a hospital. It can be a very dangerous drug in the dose Judy took. On the other hand most of the patients who reach the intensive care unit survive the overdose."

heart was still beating fast at one sixty. “And give the physostigmine,” I added.

With the seizure under control I reconnected the endotracheal tube to the ventilator. Next we checked an arterial blood gas and electro-cardiogram. The cardiac conduction complex was still slightly widened but not worse than before, and her heart rate was coming down – 135. Blood pressure and urine output were good and there was no apparent organ damage. Her brain might have suffered damage from the seizure but it was too early to tell, especially with all the Valium on board. With Dilantin infusing into her arm vein there was nothing more to do, at least for the moment. I went to write some chart notes.

Thirty minutes later Judy seized again. Her second spasm was not as violent as the first one. We again bagged her manually and gave another 10 mg of IV Valium. This time her seizure stopped spontaneously, just as the Valium was injected.

ABC. A and B were secure but now C was a problem. Her blood pressure fell from 118/68 before the seizure to a life-threatening 80/40 and her heart rate was back up to 160. Low blood pressure – hypotension – is an ominous sign in TCA overdose. It often precedes cardiac arrest. A critical moment. “Infuse normal saline, wide open. And give two amps of bicarbonate. Also another milligram of physostigmine.”

Saline to expand her intravascular blood volume and raise the blood pressure. Bicarbonate to alkalinize her blood and decrease the amount of active TCA. Physostigmine to lower her fast heart rate. If these maneuvers didn’t work the next step would be ‘pressors,’ drugs that have a direct effect on raising blood pressure. Unfortunately pressors also raise the heart rate and Judy’s was already sky high.

It was now 1 p.m. Since arriving to MICU Judy had received, all intravenously:

- 2 mg physostigmine
- 1000 mg Dilantin
- 20 mg Valium
- 2 ampules of sodium bicarbonate
- 500 cc saline, with more running in rapidly.

My mind was racing. What else to do? I couldn’t think of anything. We continued with manual ventilation. The ventilator might make things worse, perhaps lower her blood pressure by forcing air in too quickly. I kept an eye on the overhead monitor, which gave a continuous readout of heart rate, cardiac rhythm, and blood pressure.

“What about dialysis?” Dr. Hafly asked. “Can it be used to remove TCAs?”

“No,” I explained. “Dialysis does nothing to speed removal of Elavil.

The drug is mostly bound to large protein molecules which aren't removed with dialysis. It just doesn't work for tricyclics. If she makes it through this crisis we'll continue pumping her stomach with activated charcoal. Otherwise we just have to wait for her body to metabolize the drug – and try to keep her pressure up.”

A nurse cut in. “Pressure’s ninety over sixty, Dr. Martin. Heart rate one forty.”

“Good. Deborah, please listen to her lungs again. All this fluid can throw her into pulmonary edema.”

“Her lungs are still clear.”

I looked at the monitor. Blood pressure 100/65. Heart rate 135. I relaxed a little. I wouldn't have to tell Judy's parents the worst news imaginable.

* * *

Over the next half hour the threat of cardiac arrest diminished as Judy's pressure and heart rate stabilized. We reconnected the ventilator and checked another arterial blood gas. Oxygen and carbon dioxide levels were adequate, thanks in part to the enriched oxygen provided by the ventilator.

Continued coma and need for artificial ventilation meant she was still critical, that she still needed one-on-one nursing care. Every hour Judy's nurses charted blood pressure, respiratory rate (from the ventilator), pulse and cardiac rhythm, urine output, fluid intake, and the level of her coma. Every 4-6 hours they charted arterial blood gases, body temperature, a cardiac rhythm strip, list of medications delivered, and a detailed clinical assessment. Besides keeping meticulous records the nurses administered drugs and fluids, turned and cleaned her, and changed her bed sheets at least once every eight hours. Judy's care exemplified one immutable fact: no matter how sophisticated the technology a critically ill patient needs constant *human* attention. Patients like Judy live or die on nursing care. Doctors may direct the show but nurses give the care, and Judy had the best.

* * *

Judy remained comatose for 36 hours. The evening of the second day she showed the first signs of waking up. Only then did I feel confident she would survive the overdose. Seizures were under control and her blood pressure was steady at 120/74. Her young heart had withstood a massive tricyclic overdose.

As the coma lifted her brain's respiratory center also recovered. By the morning of July 3 she was breathing entirely on her own, so we removed the endotracheal tube. A few minutes later we removed the still-black nasogastric tube.

With both tubes out Judy was on her way to full physical, if not mental, recovery. Neurologic exam showed no defects and her heart rate was a healthy 86 per minute. Her affect was flat but that didn't concern us; it is the norm for patients awakening after a severe overdose. Because of possible delayed cardiac side effects we planned to watch her in MICU until at least July 5.

July 4 was a hospital holiday. We made rounds as usual but all non-emergency tests and procedures were put on hold until the next day. As if in recognition of the holiday Judy was well behaved. She ate some soup and other liquids and made no demands on the staff. We pulled out the bladder catheter and got her out of bed. Her parents were in and out most of the day, obviously grateful for her survival, also worried about what might come next.

On the morning of July 5, now that Judy was awake and could at least respond verbally, Dr. Cohen re-entered the picture. He visited her bedside for about 15 minutes, then recommended a transfer to the hospital's psychiatry ward after her discharge from MICU. There he would reassess her need for antidepressant medication.

"Is she still suicidal?" I asked, and immediately felt foolish over the question. Given her history when would Judy Bilowitz *not* be suicidal?

"She doesn't want to talk about the overdose right now," Dr. Cohen said. "I spent most of the time talking about other aspects of her life. She's still pretty numb. I think she'll open up more on Psychiatry." We agreed she could be transferred the next day.

* * *

That afternoon, suddenly and without any threat or warning, Judy began screaming. I was in MICU at the time. My initial reaction was that she hurt herself; perhaps she fell out of bed or hit her head. We rushed to her room. She was in bed, eyes half closed, screaming a high pitched

AAAAYYYYYYYYYYYYYEEEEEEEEEEEEEEEE!

It was a wail to wake the dead.

We found no evidence for any injury. I shook her and she quieted down momentarily. Then she looked at us – at me, two nurses, and Dr. Hafly standing at the bedside – closed her eyes and turned away.

"GO AWAAAAAY!" she yelled.

LEAVE ME ALONNNNNNE!"

"Judy, what's the matter?," I asked. "What's wrong?"

"Where's GREGORY?" she asked, in a tone as if to demand we release someone named Gregory.

"Who's Gregory?" I asked one of the nurses, assuming there was some

history I had missed.

“Beats me,” the nurse said. “I don’t know.”

Dr. Hafly didn’t know either.

“Judy,” I asked. “Who’s Gregory? Is he your brother?”

“AAAAAAAAYYYYYYYYYYYYYYYYYYYYYYYYEEEEEEEEEEEE
EEEEEEEEEE!”

“I don’t think Gregory’s her brother,” said the nurse. “I met him and his name is Jimmy. Should we give her something, Dr. Martin?”

“No. Her vital signs are stable. Let’s just watch her so she doesn’t hurt herself. I’ll call Dr. Cohen.”

I put in a call to his office but he was unavailable. I told his answering service it was important and to have him call back as soon as possible. Judy was in no physical danger but we could not keep her in MICU like this. At the least her wailing was disruptive. More important, it was probably a sign that she needed to go back on psychiatric medication.

One of the nurses tried to calm Judy but it was no use. Judy wasn’t seeking reassurance or a kind word. She didn’t want anything except ‘Gregory,’ and it wasn’t even clear that she wanted him – if Gregory was a him.

Dr. Cohen finally called back about an hour later. I told him what had taken place.

“Gregory is a male nurse she knew at Weathergill,” he explained. “She brings him up during times of extreme stress. He was a calming influence during her worst periods there. I imagine she realized for the first time that she’s in a hospital and called to him for help. I agree she probably should be started back on medication. It would be best if she can be moved to Psychiatry. Are you ready to release her from MICU?”

I was and I wasn’t. I wanted her to go but I also wanted to monitor her heart another night, and cardiac monitoring is not available on the psychiatry ward.

Dr. Cohen recommended we begin Haldol, a major tranquilizer, and that she go to Psychiatry in the morning. I wrote the order: 1 mg Haldol intramuscularly every 12 hours.

Haldol worked and Judy calmed down. There was no more screaming and the following morning, July 6, she was transferred to Psychiatry.

Followup

Triavil was re-started on the Psychiatry ward but at a higher dose: 4-25. Judy’s psychosis improved once again on medication and everyone was encouraged. Mrs. Bilowitz agreed to administer the medication at home and to keep the container locked away. Under this condition Judy was released at the end of July.

At home either her mother or the maid gave out the pill, twice a day, and

watched Judy swallow it. Then the container was secured, a precaution taken only to prevent an impulsive drug overdose. There were of course other ways for someone in Judy's situation to attempt suicide. The new dose worked well and Judy seemed to get better. She sometimes went shopping with her mother and on occasion took one-day trips with both parents. She even attended two meetings of the gardening society in the home of a family friend.

For most of August Judy was watched like a fragile child, but as she improved her parents' concern over another suicide attempt lessened. Dr. Cohen saw Judy regularly and did not find her overtly suicidal.

Judy mother and father so desperately wanted her to live a normal life. As time passed they more and more treated her like a responsible adult. There was even talk with Dr. Cohen of allowing Judy to self-administer the Triavil, though no decision had yet been made.

Judy must have been aware of this change in attitude. Was she just waiting and plotting another attempt? Or was her mind too disordered to make such plans? Whatever the thought processes in her head, one day in December of that same year Judy found the almost-full Triavil container sitting on the kitchen counter. Why and how it came to be left out, and for how long, we never learned. If Judy hesitated or pondered or fought the temptation, we'll never know. We do know that she opened the bottle and swallowed all the tablets, then threw the empty container in the trash and went to lie on her bed. This all happened about 11 a.m. Her mother was out shopping and the maid had the day off.

By supper time Judy Bilowitz was dead.

– END –

3. Call NASA!

“You asked them WHAT?” My voice was raised in mock anger, to show the house staff on morning rounds that Dr. Howard Stine’s question was unacceptable. Shortly after admitting an 88 year-old man with pneumonia the intern had posed a certain question to the patient’s son and daughter.

“I asked them,” Dr Stine repeated, ‘Do you want us to do everything for your father?’”

“And what did they say?”

“They said yes.”

“I see.” I waited a few seconds for someone to offer comment. Dr. Stine was far from the first neophyte physician to ask such a silly question, and he surely wouldn’t be the last. Instead of responding, everyone on rounds – two interns, a senior resident, two nurses – looked past me in the direction of the patient, Jack Smilovsky. We were standing outside his room and sliding glass doors made him easily visible.

I knew what the house staff were thinking. ‘Well, it’s probably not a good idea for Mr. Smilovsky to receive too much heroics, but if that’s what his family wants, what are you going to do?’

“Barbara, what do you think of Howard’s question?” Barbara Milo, the resident on the case, had rounded with me many times and knew my feeling about this recurring problem. Barbara is quick-witted and able to display the right amount of sarcasm when appropriate. Turning to Howard, with her head cocked slightly toward the patient, she went right to it.

“Do they want him dialyzed if his kidneys fail? Do we intubate him if his brain slips between his vertebrae? Do we send him for a heart transplant if he doesn’t respond to drugs? Do we give platelet transfusions if his bone marrow goes zippo? Do we...”

“OK, Barbara, thank you.” I looked toward Howard but spoke to everyone. “WE don’t know everything that can be done. How can you ask a patient’s family if THEY want everything done? Medical technology is endless, infinite!

“Soon we’ll be sending patients into space, for god sakes, to treat them with zero gravity! Do the Smilovsky children want us to send their father into space? Did you ask them?” Everyone laughed. At least I had their attention. Dr. Stine was buried by the laughter but he managed to speak up.

“Dr. Martin, what should I have asked his family?”

“Not a zero option question. With what you asked, either they could say

yes – as they did – or ‘no, don’t do everything.’ But saying no puts them on an instant guilt trip. Maybe their understanding of ‘don’t do everything’ is that we’d let him die for want of an aspirin or a bedpan. Who knows? You didn’t give them any realistic options. It was like you came out and said: ‘OK, we’ve got your father – does he live or die?’ What *could* they say?

“Now that they’ve said yes, you’ve left Mr. Smilovsky open for mega technology. Maybe not the space shuttle – yet – but just about everything else. Look at him.”

I slid open the doors and we went in and surrounded his bed. It was a pathetic scene. Mr. Smilovsky looked his age plus another ten years: gaunt, wasted, emaciated, out of touch with what was happening. His eyes were half closed and sunken into their orbits. His open mouth showed only toothless gums and a tongue that moved ceaselessly back and forth, without purpose. He might be suffering, but nothing in his eyes or hands or mouth communicated any feeling. This was not a human being so much as a heart and pair of lungs inside an ancient body. Mr. Smilovsky deserved antibiotics, a warm bed, and kindness. He did not deserve – because he would not benefit by – artificial life support.

At the start of rounds we learned that Mr. Smilovsky had been in Mt. Zion Nursing home for four years, the last two completely bedridden and demented from longstanding Alzheimer’s disease. When he developed fever and shortness of breath he was sent to Mt. Sinai’s emergency room. Lacking any clear directive about use of heroics the ER doctors sent him directly to MICU, where questions about how far to go were (apparently) first asked.

Now, at the seeming behest of his children, neither of whom, to be sure, had demanded anything in particular, we were obligated to do anything and everything to keep him alive. The medical diagnoses were pneumonia, sepsis, and dementia. Treatment was with antibiotics, fluids, and nasogastric feeding.

Mr. Smilovsky’s condition was tenuous. Any hour he might ‘need’ an artificial ventilator for respiratory failure. An hour after that he might ‘need’ infusion of dopamine to support his blood pressure. Then he might ‘need’ a pacemaker, kidney dialysis, and a host of other readily available medical technologies.

For the moment however, his care looked reasonable; he had not yet entered the realm of high tech. I was ready to move on but the other intern on rounds, Pier Simpson, asked: “Aren’t we obligated to do what’s necessary? I mean, isn’t that why he’s here?”

Blessed be the intern who bids me to continue. Professional life would be lonely without someone to TEACH.

“Pier, how many people die each year in this country?”

The intern shrugged.

“Well, how many people are *in* this country. You need to know that piece of trivia first.” After a few seconds of silence from Pier I said,

“Anybody?”

“About two hundred and fifty million,” said Molly, one of the nurses.”

“Right. Now how many people die each year, of all causes?”

“About a million?”

“No, actually it’s about two million. Now, excluding those who die before they can get to a hospital, and children, and accident victims, let’s say that about one million mostly elderly people die of chronic disease or end-stage illness or old age. Furthermore, let’s say they all end up in hospitals like Mt. Sinai, and that they all get connected to life support machines just as they are about to die.”

“Dr. Martin,” Molly interrupted. “Shouldn’t we discuss this outside his room?” Actually, it didn’t matter to Mr. Smilovsky, I was sure of that. But our proximity to the patient bothered Molly so I agreed to continue outside. We walked out and closed the sliding doors behind us.

“For the sake of argument, let’s say that life support therapy prolongs each patient’s dying by an average of two weeks. They will all die soon anyway, because that’s the premise, but we’re going to interfere with nature a little by instituting artificial ventilation, pressors, and any other life support deemed medically necessary. Furthermore, this is all going to be done in intensive care units like ours. How much are we talking about?”

As soon as conversation on rounds turns to economics everyone perks up and listens. It never fails. I now had their undiluted attention.

“It depends on what each hospital bill is,” said one of the interns.

“Right. Let’s say two weeks of therapy before each patient surrenders to nature – that seems about the average length of time ventilators can keep them going. Remember, they are destined to die anyway. Now the basic room rate is a thousand a day. Added to that are charges for antibiotics, respiratory care services, x-rays – tons of x-rays – and other odds and ends, roughly twelve hundred a day above the basic room rate. Twenty-two hundred a day is the average hospital charge for a MICU ventilator patient. It’s a lot more if the patient is dialyzed or gets a pacemaker or has any surgery. Anyway, at fourteen days for the typical terminal ventilator patient, we’re talking about a little over thirty thousand dollars each, and that’s not counting professional fees.

“Some patients will last longer than two weeks, others will go quicker no matter what you do, but thirty thousand is probably a nice, conservative figure. Now, what’s thirty thousand times one million?” A pocket calculator was brought out.

“Thirty billion dollars,” said Dr. Stine, with some awe in his voice. The others just raised their eyebrows in acknowledgment. One nurse muttered “wow.”

“That’s right. Thirty billion dollars. Not the national budget, but not a small amount either. And for what? To prolong dying two weeks? It seems ridiculous, doesn’t it? Fortunately, those one million patients don’t all end

up in our ICU or anyone else's. Many die at home, or in the nursing home, or even in the hospital, in a quiet room with their family and no tubes or machines."

"But you would save some of those patients," said Dr. Simpson. "How can you decide ahead of time when it's inappropriate to be heroic?"

This intern is a jewel, I thought. He must round with me more often.

"That's right. Statistically some of them might *not* die. They might go on and *live* on the machines. Like Mrs. Jackson."

"Who is Mrs. Jackson?" asked Dr. Stine.

"Barbara, tell him."

"Mrs. Jackson is an eighty-year-old demented woman who has been here about six months. She was only in MICU for two of those months. Now she's up on Tower North. We can't get her off the ventilator. And no nursing home will take her *and* her machine."

The two interns remained silent.

"Look, the whole point is," I continued, "doctors have to make decisions. Ideally, life and death decisions should be made with the input of the patient and family. Mrs. Jackson's family, as I recall, was never given an option about what to do. Now they're stuck and we're stuck. And Mrs. Jackson's stuck. Ask her if she's happy. She doesn't even know what planet she's on. Should we have to spend thirty billion dollars to keep one elderly, otherwise dying patient artificially alive?"

There was no answer. It was time to change course.

"Pier, would you intubate Mr. Smilovsky if he were ninety-eight?"

"Well, it depends on the circumstances." A beautiful hedge. No answer at all.

"How about a hundred and eight?"

"Maybe." Now I had Dr. Simpson on the defensive, and losing ground.

"How about if he was one hundred and ten years old, and you had documented evidence of metastatic cancer to every major organ."

"No, of course not, Dr. Martin."

"So, you're willing to draw the line somewhere. But you feel uncomfortable about Mr. Smilovsky because he doesn't have widespread cancer. Only pneumonia and sepsis, theoretically treatable conditions. I understand that. At least you admit there is a line to draw."

Dr. Stine spoke up. "Dr. Martin, how would you have handled Mr. Smilovsky's family?"

"First of all, I wouldn't call it 'handling.' Explaining the situation is what you really want to do, in terms they can appreciate. I've never met relatives that want their mom or dad or sister or brother kept alive as a vegetable on a machine. Well, I take that back. We had one a few years ago, but the daughter had a few loose bolts, so that doesn't count; she could never understand the difference between near-brain-dead and sleeping. Sensible families *don't* want relatives kept alive on machines, with no hope

of return to humanity. You have to discuss the situation in these terms.

“We should simply tell them the truth. ‘Your father is eighty-eight. He’s at life’s end. Nothing we do will restore his mind or body to anything better than he was last week. The most we can hope to accomplish is a return to his former state, demented and bed-confined. If his breathing stops we’re legally obligated to use machines to keep him going, unless you tell us otherwise. We’d obviously like to know his own wishes, but that’s not possible, so I’m afraid it’s up to you.’

“You’ll be amazed at how often they will say, ‘No, Dad wouldn’t want that. Do what you can to make him comfortable, but no life-support machines.’ Or, they might ask you for more information: ‘What are his chances of getting off the machine once he’s connected? What do you recommend? What would you do if this was your father?’ The point is, you’ve established a dialogue and given the family realistic options. You can easily take it from there.

“If you get a clear sense that heroics and artificial life support are not to be used, you can order ‘Do Not Resuscitate’; then, at least, everyone knows how far to go or not to go. If he’s made ‘DNR’ we don’t end up with a social service disaster like Mrs. Jackson.”

* * *

Fortunately Mr. Smilovsky did not need artificial life support. He survived pneumonia and was sent back to his nursing home. But the man in the next room didn’t fare as well.

Mr. Zigson was 82 when he came to our ICU. For Mr. Zigson everything *was* being done. By default. He received medicine’s top technology not because he requested it, or because his family demanded it, but because there was no one to call a halt.

Mr. Z had no relatives anyone knew about, only a legal guardian. In the nursing home he was thoroughly demented though not as bedridden as Mr. Smilovsky. He could sit and walk with assistance but had to be fed by nurses’ aides.

Mr. Z was sent to Mt. Sinai Medical Center for impending kidney, heart and lung failure – about to die, in other words. His mind was too far gone to give insight into his own wishes for treatment. Having no family around (there were rumors of relatives in far away places, but we never saw any and no one ever called), we had to rely on his legal guardian, an attorney long ago appointed by the court.

The attorney’s knowledge of Mr. Z’s medical condition was close to zero. He preferred it this way “in order to stay objective and not bias myself.” To help preserve his state of blissful ignorance he never visited Mr. Z in the hospital. When confronted over the phone, on the day of Mr. Z’s admission, with the important questions – How far should we go? Can we

make him DNR? What would Mr. Zigson want if he could tell us? – the attorney was non-committal: “Do what you have to do.”

What did we have to do? Was it right to assault Mr. Z’s body with tubes, needles, monitors, a ventilator and other assorted devices? Was it right to take over the function of his heart, lungs, kidneys, pancreas, and stomach without his consent? Was it right to feed his gut with food that never touched his tongue and replenish his blood with scarce blood products? Was it right *not* to do these things?

The ethical questions were weighty but the practical answer to all was simple: protect yourself against the threat of litigation. In the current state of affairs no one could legally fault you for trying to save his life, only for letting him die.

Having no one around to call a halt, physicians often must do things they feel are ethically or morally or socially wrong. If we let Mr. Z die a natural death (God forbid!) and his estate came up for probate, some long lost relative might come zinging out of the West to pursue an award commensurate with the charges – wrongful death, medical malpractice, mercy killing, *euthanasia*. (It’s happened before.) Yet if he had a third cousin (or legal guardian) willing to say ‘No, don’t do this,’ and that decision was consistent with our own sense of medical propriety, we could have stopped.

Mr. Z had no one, so we practiced medicine by default. And the default mode is *everything*. No one caring for Mr. Z thought it proper to begin artificial ventilation when his breathing failed. Artificial ventilation was begun without hesitation. No one felt comfortable about starting hemodialysis when his kidneys failed. He was dialyzed. And no one felt good about giving blood transfusions when his gut began to ooze the vital fluid. He was transfused.

When we consulted a surgeon to advise about the intestinal bleeding she decided not to operate, but not because it was inappropriate (the guardian would have given permission), but because we were finally able to stem the bleeding with more conservative measures. “Call me if the bleeding resumes,” she noted on the chart. She *would* have operated had it been necessary.

And when Mr. Z’s heart began acting in strange ways – here a beat, there a beat, and frequently no beats for many seconds – the cardiologist didn’t hesitate to place a pacemaker, “considering that everything possible is being done for this gentleman.”

By day 14 of Mr. Z’s stay in MICU – the same day I chided Dr. Stine for his ‘do everything’ question to the Smilovsky family – Mr. Z was fully hooked up, a human form who had long since lost his humanity, artificially maintained by the best that medicine had to offer.

Through his mouth entered a foot-long, clear plastic endotracheal tube. Three-fourth’s of the hollow tube lay in his mouth and trachea; the other

quarter dangled outside his face and connected via plastic piping to the ventilator. Twenty-two times a minute the machine went Whoosh! (air in), swishhh (air out). Whoosh!-Swishhh, Whoosh!-Swishhh...

Another machine monitored blood pressure continuously via a thin plastic catheter sitting in his radial artery. A larger catheter invaded one of his neck veins, and through this an infusion of dopamine helped maintain his blood pressure sufficient to perfuse kidneys and brain. A nasogastric feeding tube entered his nose and ended in the small intestine; liquid nutrients fed through this tube could sustain his metabolic needs forever.

A bladder catheter exiting his penis allowed urine to flow freely into a bag for easy collection. The latest and most expensive antibiotics went through another thin plastic catheter in his right forearm. A rectal tube helped funnel his diarrhea – a side effect of the antibiotics – into yet another plastic bag.

On rounds we discussed Mr. Z from head to toe, noting tubes, machines, drugs, diagnoses. I was satisfied that things were being handled correctly, and had very little to add. What more could one say? Everything was being done, practically every organ was supported by some drug or device. Altogether we counted seven tubes and catheters (tracheal, rectal, two intravenous, arterial, bladder, plus one for hemodialysis), three life support machines (ventilator, kidney dialysis, cardiac pacemaker), and 10 different drugs, including the blood-pressure-supporting dopamine. Whatever the outcome, the house staff were on top of Mr. Z's care and the training program was being served.

Or was it? Each device or drug we used for Mr. Z is a major advance in medical therapy. Each, in its own way, has done wonders for patients. You can find many articles that show how each therapy has improved survival by some significant percentage. For example, thousands of people are gainfully employed because of kidney dialysis, without which they would have died. Other thousands live a normal life made possible by a cardiac pacemaker.

But were all these therapies *in toto* appropriate for Mr. Z? What is known about using multiple life support devices in elderly, demented patients. Do they help? Does there not come a point of no benefit, when the second or third or fourth machine will add nothing but expense and patient suffering?

Molly called it. "Dr. Martin, why are we doing all this to Mr. Z?" Not *for*, but *to*.

"We're caught, Molly. We have no choice. No one is taking responsibility for decisions about Mr. Z. We are in a medical-legal conundrum."

The nurses and house staff looked at me as if *I* should be responsible, and I became a little defensive.

"*I* didn't order hemodialysis. Dr. G. started it." They knew this, of course; I was just pointing out the obvious.

“Look,” I said. “Dr. G. doesn’t want to go on record as withholding a life-saving therapy. We asked his opinion about what to do for the kidney failure. What else could he say? Dialyze! Ditto the cardiologist and everyone else. Individually, everyone is doing what’s medically correct. But when you stand back and look at the big picture, that’s when you see it’s wrong. As a result, we’re *all* responsible.” There was a general nod of agreement to this analysis.

Two more observations and I would be through with morning rounds on Mr. Zigson. I pointed toward his monitor, with its erratic heart rhythm, and proposed a favorite non-sequitur in such situations:

“We’re like physiologic voyeurs. We’re charting an inevitable, natural process with our machines. He’s dying and we’re recording it all, down to the last electrolyte and heart beat. It’s absurd, but at least there’s one consolation to all this.”

“What’s that?”

“My second law of intensive care.”

“Second law? What’s your first law?”

“Don’t you know?” They did not. Occasionally I kick around pseudo “laws” to make some sense out of what we do.

“Ok, the first law: If it happens here it happens everywhere. This kind of thing is going all over the country, every day.”

“What’s the second law?”

“Money not spent on dying patients will not go to feed hungry children.”

* * *

The medical and moral travesty continued for another week. After that the dopamine infusion no longer worked and Mr. Z’s blood pressure bottomed out. There was nothing else all our machines and chemicals could do. Nature won.

Mr. Z’s total hospital bill came to \$73,475.37. Not counting professional fees.

-- END --

4. A Strange Pneumonia

One day in March 1985 – the year is important – we received a patient from another hospital: Reginald Herbert III, a 46-year-old oil company executive. In April of that year Mr. Herbert was hospitalized at a non-teaching suburban hospital with an undiagnosed and progressive lung infection. For two weeks his doctors tried in vain to diagnose the cause of infection, finally concluding that he needed an open lung biopsy.

An ‘open’ biopsy is a major operation, literally an opening up of the chest cavity to remove a piece of lung tissue. Mr. Herbert was transferred to our care mainly because Mt. Sinai Medical Center is better equipped for this procedure. He arrived on a Friday afternoon, in preparation for surgery the following Monday. On exam he was short of breath, breathing rapidly, and had a temperature of 102. He was “acutely ill” and could only talk with great effort because of respiratory distress. We obtained most of his medical history from the transfer records and Mrs. Herbert, a trim, fortyish woman who answered our questions but never went out of her way to volunteer more than was asked.

The facts were meager. Mr. Herbert had been well until three months before hospitalization, when he first noted onset of fatigue and a dry cough. Two seven-day courses of antibiotics, taken as an outpatient, did not change his symptoms.

Initially his outpatient chest x-ray was read as normal so pneumonia was not a tenable diagnosis. He soon became quite ill with high fever, sweats and persistent dry cough. At that point he entered the suburban hospital where he underwent a battery of tests, including fiberoptic bronchoscopy. All results were non-diagnostic. His white blood cell count was elevated and he had fever, cough and an abnormal chest exam, so pneumonia seemed a good bet. Sure enough, a few days prior to transfer a shadow appeared on the chest x-ray and his physicians came to recommend the open lung biopsy.

Mr. Herbert’s travel history fueled speculation of an exotic infection. As an oil company executive he had traveled to many countries, mostly to South America. Mrs. Herbert said he had spent about half of the previous two years abroad, in blocks of 6 to 8 weeks. She never went with him because they had two teenage kids in school.

Did he ever have pneumonia before? No, she said. Did he become ill on any of his overseas trips? Nothing that she knew about. Had either she or her children experienced any febrile illnesses in the previous year? No.

What an interesting case! Middle-aged executive with fever and pneumonia of uncommon cause. We ran through the “differential” of possible diagnoses and why he might not have responded to empiric

antibiotic therapy. By the time of transfer Mr. Herbert had already received five different antibiotics, two as an outpatient and three more in the suburban hospital. Any common or typical infection should have been clobbered.

There was no evidence for the usual causes of pneumonia, such as Legionnaire's disease, mycoplasma, streptococcus, staphylococcus, and hemophilus. The one other infection that always has to be considered, tuberculosis, seemed unlikely; tests at the suburban hospital were negative for TB infection, both the TB skin test and a special bone marrow exam.

There is a 'waste basket' category of 'viral' pneumonia, often diagnosed only by excluding everything else. Viral infections are often hard to diagnose. They usually require checking blood samples weeks apart, and the blood has to be sent to a special state lab. We were not ready to call this a viral pneumonia.

There are many other diseases in the world alien to most Americans: malaria, schistosomiasis, blastomycosis, aspergillosis, tularemia, filariasis. Could Mr. Herbert have one of these exotic illnesses? Very possibly.

We also considered non-infectious problems like cancer, lupus erythematosus, and rheumatoid disease but these all seemed highly unlikely. Even AIDS was considered, though there was no history of homosexuality or drug abuse, the only associated risk factors known at the time. Even so, an AIDS antibody test was sent out prior to his transfer.

Whatever Mr. Herbert's diagnosis, we agreed with the need for the open lung biopsy; it just might reveal an unusual and treatable infection. Perhaps one out of a thousand pneumonia patients have to be taken this far to make a diagnosis.

* * *

Mr. Herbert went rapidly downhill. By Sunday evening his respiratory rate was very rapid, he was a bit confusional, and his blood oxygen tension was uncomfortably low. All signs pointed to impending respiratory failure. We intubated him and began artificial ventilation. Unless the lung biopsy showed a treatable pneumonia his prognosis for survival seemed poor.

He went to surgery Monday morning in critical condition: ventilator-dependent, breathing 100% oxygen, semi-comatose. The surgeon quickly opened his chest and excised a small piece of left lung. One half of the biopsy specimen went into a bottle of formalin for fixation and staining; thin slices of this piece would be examined under the microscope. The other piece went into a bottle of saline, for culture of bacteria and fungi.

Twenty minutes after the biopsy Mr. Herbert's chest was closed, save for a drainage tube that is routinely left in place. Mr. Herbert went to recovery and then, an hour later, was returned to MICU.

Unfortunately the effects of the operation, added to his debilitated state, made it impossible for him to breathe without the ventilator. We could not

remove the endotracheal tube until he improved, and improvement would require a treatable diagnosis.

* * *

On Tuesday morning we had the answer. Mr. Herbert was suffering from *pneumocystis carinii* pneumonia, the commonest type of pneumonia in AIDS patients. Coincidentally, his AIDS antibody test returned from the lab the same day. It was positive.

The pieces fell rapidly into place. Our patient had a classic presentation for AIDS but his physicians, myself included, were somewhat fooled by his social background and our own inexperience with the disease at the time. How could an upper middle class executive have AIDS? In 1985?

But he did. And that is why none of the antibiotics had helped Mr. Herbert; he had not received drugs that attack *pneumocystis carinii*. We immediately began the appropriate antibiotic, trimethoprim-sulfamethoxazole.

Because Mr. Herbert was in MICU, and I was available when the diagnosis became established, it fell to me to tell his wife. How to do it? There was no point in being coy or misleading, as that approach invariably backfires. I had told many patients and families terrible news but never, I realized, had a diagnosis of AIDS been that news.

I met Mrs. Herbert in the ICU on Tuesday afternoon as she was coming out of her husband's room. She was alone.

"We have the results of the biopsy," I said, matter-of-factly. She looked at me without responding, waiting for me to continue. "It looks like it's (the truth wasn't so easy after all) the type of infection seen in AIDS patients."

I expected some expression of disbelief, or denial, or even anger, but she only said, "I see." If she was surprised at the diagnosis she didn't show it. This woman seemed like a rock. I hesitated to continue the discussion but continued.

"Also, the blood test sent out last week has just come back. It is positive for antibody to the AIDS virus."

In a most *uninquisitive* tone she responded: "I wonder how he got AIDS?"

I wondered with her. "Did he receive any blood transfusions in the last 5 years? The blood of AIDS carriers can infect otherwise healthy people."

"No, not to my knowledge," she said.

"He didn't use any drugs, did he?" I hoped she was not offended by this question.

"No, of course not," she said in a monotone, showing no offense but also no emotion. I thought it peculiar that she seemed so much more certain about drug abuse than about blood transfusions.

Suddenly it dawned on me. She *did* know how he got AIDS and she

wasn't surprised at all. It now seemed awkward to ask the obvious question so I let it pass. In truth I was embarrassed to ask if Reginald Herbert III, father of her two children, was homosexual.

“Do you wish to be tested?” I asked.

“No, I don't think so. It's not necessary.”

* * *

Over the next several days Mr. Herbert continued to do poorly. He did not respond to the antibiotic for *pneumocystis*, probably because it was begun too late or his infection was too far advanced. He remained ventilator-dependent and unresponsive.

Meanwhile, Mr. Herbert's family physician, who had followed him from the beginning of the illness, learned more about the Herberts and their marital relationship. The Herberts had not had sex together for over 18 months. His lack of interest, and some pictures of men Mrs. Herbert found in his suitcase, made her strongly suspect homosexuality. She presumed that much of his homosexual activity took place abroad. Mrs. Herbert denied all sexual activity since he left her bed. And she repeatedly declined to be tested for the AIDS virus.

Mr. Herbert continued to deteriorate and died seven days after the operation, of sepsis and respiratory failure. Mrs. Herbert did not grant permission for an autopsy.

Comment

By the end of 1981, the year AIDS was first reported, there were 281 known cases in the United States. Two decades later the world wide statistics are appalling – over 20 million dead worldwide, and many more millions infected.

Fortunately, newer techniques for diagnosing *pneumocystis carinii* pneumonia have obviated the need for open lung biopsy. Now the diagnosis of PCP can usually be made right away, with relatively little risk to the patient. The diagnosis is often established by instilling some saline into the lungs through a flexible, fiberoptic bronchoscope, and suctioning the saline out a few seconds later. When these 'saline washings' are carefully examined under the microscope they will almost always reveal the *pneumocystis* organisms in patients who have PCP.

Pneumocystis carinii is still a common cause of pneumonia in AIDS patients. With early diagnosis and aggressive antibiotic therapy most AIDS patients recover from their first infection of PCP. It is recurrent infections from opportunistic organisms like *pneumocystis* that have caused most of the AIDS mortality.

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5. Asthma in the Last Trimester

About five percent of the population in industrialized countries suffers from asthma. An asthma condition can range from minimal symptoms, with little or no impact on daily activities, to severe and life-threatening disability. Asthma can develop at any age. I have often seen asthma develop *for the first time over age 60* – and in people with and without an allergic history.

In an asthma attack smooth muscles lining the bronchial tubes, the airways of the lungs, contract or tighten. This contraction (also called bronchospasm) leads to narrowing of the airways. At the same time the bronchial walls become inflamed and secrete thick mucous into the airway, causing further narrowing. To gain some idea of what it feels like during a severe asthma attack, try breathing through a straw with your nose plugged. As you breathe gradually pinch the middle of the straw until it closes about half way. Now jog in place.

In people with asthma a variety of stimuli can bring on an attack of bronchospasm, including allergic reactions, upper respiratory infection (including the common cold), exercise, climatic changes, cigarette smoke, and emotional distress. Whatever the precipitating event the result is the same: obstruction to air flow, wheezing, and a feeling of air hunger.

In a desperate attempt to bring in more air the asthmatic recruits ‘accessory’ breathing muscles (mainly in the neck and shoulders) and breathes faster. At the height of an asthma attack the patient looks like he (or she) just ran a marathon race (keep breathing through that pinched straw and you will too).

By definition asthma is *reversible* airways obstruction. The bronchospasm and excess mucous usually abate with appropriate medication. The operative word is ‘usually.’ Sometimes asthmatics don’t respond to treatment, or respond so slowly that their condition requires hospitalization. Fortunately only a small percentage of asthmatics ever reach this stage.

* * *

Delores Buchanan was 24 when she came to the medical intensive care unit (MICU). Diagnosis: severe asthma, complicated by a 36-week pregnancy.

As a child Delores suffered from hay fever but not asthma. She received allergy desensitization shots from age 12 to 15. At 18, just out of high

school, she first developed symptoms meriting the label of asthma: some wheezing and shortness of breath on exercise. These symptoms were controlled with oral and inhaled medication and over the next three years she never required hospitalization or emergency room treatment.

After a year of secretarial school she married and shortly afterwards became pregnant. There were no complications of her first pregnancy and at age 20 she delivered a healthy baby boy.

At 21 Delores suffered her first bad asthma attack. It started with a sinus infection. Nasal congestion progressed to mucous in her throat, cough, wheezing, dyspnea, more cough, more shortness of breath, and finally a trip to the doctor. Antibiotics and an asthma inhaler did not provide relief. Her shortness of breath progressed and she came to Mt. Sinai's emergency department. The severity of her symptoms mandated hospitalization for intravenous therapy, but she did not require intensive care. She gradually improved and was able to go home four days later, on a regimen of asthma inhalers and pills.

Her asthma remained quiescent for a while. At 22 she became pregnant again and delivered a healthy girl; she experienced no significant asthma symptoms, either during pregnancy or after delivery. While in the hospital she asked for and received one or two extra inhalation treatments but did not need intravenous asthma therapy.

After discharge Delores underwent thorough allergy evaluation. Multiple skin tests showed allergic responses to nothing more specific than dust and common mold. Desensitization shots were not recommended. She continued regular visits to the allergy clinic and her asthma remained under control, with intermittent use of inhalers, for the next 18 months.

In 1987 she developed a severe asthma attack a few days after a head cold and was again hospitalized on the regular ward. After four days' therapy with intravenous medication she was discharged on a tapering course of prednisone (a corticosteroid, the most powerful anti-asthmatic medication). She continued to do well with outpatient therapy.

In the summer of 1988 Delores became pregnant for the third time. The first two trimesters were uneventful; she used an asthma inhaler as needed, about once every few days, and did not need prednisone. At 34 weeks gestation her asthma symptoms inexplicably worsened. She soon began inhaling asthma medication almost every day until things got so bad her husband brought her down to the hospital. Shortly after she arrived in the ER my beeper went off. I answered right away.

"Dr. Martin? Hi. This is Michael Highland, in the emergency room. I believe you know a patient who's down here, Delores Buchanan?" I had not seen her since the last hospital admission in 1987.

"I remember her. She's a young woman with asthma. What's happening now?"

"She's 24 years-old and 34 weeks pregnant, with one fairly severe

asthma attack. I think she should be in MICU. She's not intubated but she's working very hard to breathe. We started her on methylprednisolone [an intravenous corticosteroid] and IV aminophylline [a bronchodilator]."

"Do you have a peak flow or blood gas?" I asked.

"Her peak flow is only about one hundred ten [liters/minute]. Let's see, I have her blood gas right here. PO₂ is seventy-two, PCO₂ thirty-eight, PH seven point four three. That's on room air." These results showed adequate oxygenation and ventilation, not a life-threatening situation. At least not yet. But then there was the fetus to worry about.

"Is her OB going to see her?"

"Yes. Dr. Senior's her obstetrician. His resident examined her and doesn't think she's in labor. They'll follow her in MICU but they think she's too sick for the OB floor."

"OK. Send her right up."

There is a rule about the course of asthma in pregnant patients: one third will improve, one third will have no significant change in symptoms, and one third will worsen. Unfortunately there is no way to predict which patient will end up in which group. Experience with an earlier pregnancy also doesn't predict what will happen.

There is also a simple rule for treating the pregnant asthmatic: treat the mother and the baby will be taken care of. If you don't treat the mother fully, for fear of harming the fetus, it can actually suffer from distress and hypoxemia. Certain drugs, of course, should not be used during pregnancy. They include tetracycline (an antibiotic that stains fetal teeth); coumadin (a blood thinner that crosses the placenta); most newly-released drugs (not enough information to know about teratogenic effects); and any drug that might cause uterine contractions. This proscription still leaves available virtually all asthma drugs including corticosteroids, the most potent.

Mrs. Buchanan was put in room 5. After the nurses checked her weight and vital signs I went in. "Hello. I'm Dr. Martin." She nodded hello, smiled, and tried to look comfortable, but rapid breathing and contraction of neck muscles with each breath showed distress. Her face, full from pregnancy, displayed the fear and apprehension of severe asthma. Sweat covered her brow. Nostrils flared with each inspiration. Medication begun in the ER had helped a little but her respiratory rate remained fast, about 30 per minute. Also apparent were a distended belly poking up from beneath the bed sheet, legs swollen with edema fluid, and wheezing – audible without a stethoscope. In situations like this you don't walk in and ask, "How are you?" "Well," I said, "your asthma is beginning to respond to the medication. Just to be safe we'll keep you here until your asthma is better, then you can have that baby of yours."

"Good. . .I'm looking forward. . .to that." She could not speak more than a few words without pausing to catch her breath.

I ordered a chest x-ray to make sure we didn't miss pneumonia or some

other acute problem. (A chest x-ray is safe, especially in the third trimester; as an extra precaution the mother's abdominal area is routinely shielded with a lead apron).

We inserted a catheter in her radial artery for frequent blood gas monitoring. An hour after arrival to MICU her respirations were still labored and arterial blood gases showed no major change. Not a bad sign, but not good either. Her chest x-ray was negative.

She received three basic asthma drugs: intravenous infusion of aminophylline and methylprednisolone, and inhaled albuterol via a nebulizer device every three hours. She also received oxygen through a nasal cannula. A cardiac monitor displayed her heart rate and rhythm, and a separate fetal heart monitor recorded her baby's heart rate. Mother's heart rate: 130 per minute, baby's 160. Both acceptable.

* * *

You're not supposed to die from asthma. After all, it's a reversible syndrome, there's good medication for it, every general physician is familiar with the symptoms and therapy. Yet year after year several thousand Americans do die from asthma *and the number is rising yearly*. Reasons for asthma deaths are varied. Over- and under-medication have both been blamed. Some patients are at fault for not seeking prompt medical attention. And, sadly, physicians are sometimes culpable for sending the patient home when he or she should be admitted to hospital. For about 15% of patients there is no explanation; they come for therapy on time, they get treated appropriately, but they just don't respond. Still, why the increase in deaths, especially with newer and more effective medications? No one knows for sure.

At Mt. Sinai we see about one death a year from pure asthma. In the case of Johnny Morgan, "see" is the appropriate word. He was young, poor, and unemployed. And he smoked. It was his additional misfortune to have bad asthma from the age of 15, asthma which, by the age of 21, had landed him in the hospital twelve times, four of them in intensive care.

The pity of Johnny Morgan's asthma is that each attack was probably preventable, at least in its severity. And, until his last attack, each episode was also reversible. After a week of in-hospital treatment his lung function would return to normal or near normal. Then he would go home, smoke, catch a cold or develop bronchitis, and land in the emergency room. Sixty-seven emergency room visits in six years, not counting the twelve that got him admitted to hospital.

Johnny was given countless regimens of tapering steroids, numberless asthma inhalers, and hours of instruction on how and when to use the medication. He was admonished about smoking so often that "tobacco addiction" came to be included in his list of discharge diagnoses.

The outcome was foretold in Mr. Morgan's outpatient clinic record, which included as many stamps of "NO SHOW" as followup notes by his clinic doctor. Two or three consecutive NO SHOW entries were followed by a cryptic "Adm Hosp - See Hosp Chart" [Admit to hospital - see hospital chart].

In the middle of his 21st year, in June, Johnny developed progressive bronchospasm. His ever-present asthma inhaler provided relief for a while but after several hours, perhaps longer, he began to feel that symptom of relentless suffocation that had brought him to our ER so often. A friend drove him to the hospital, about four miles distance on city streets. The ride over, as we learned later, was a nightmare for driver and patient. Johnny rapidly grew more distressed and his friend drove faster, running through red lights and stop signs, finally attracting a police car about half mile from the hospital. Johnny's friend stopped in front of the emergency room (he was familiar with the facility), got out of the car and ran to the officer who had pulled up behind. "My friend's not breathing!!"

The officer ran to the car, saw a slumped-over young man and began CPR in the front seat. The friend ran inside and yelled for help. Within seconds a first class, battle-ready, A-1 trauma team was hard at work on Johnny Morgan, his body spread out on the parking lot pavement.

The ER team got him defibrillated, intubated, IV'd, infused, catheterized and medicated, but they couldn't get him ventilated. They were about two blocks too late. Johnny Morgan's lungs were beyond resuscitation and he died right there, 25 steps from doors marked "Mt. Sinai Hospital Emergency Department."

Autopsy revealed no illicit drugs or alcohol in his body, just the stiffest, most plugged-up lungs we have ever seen. A pure asthma death.

* * *

Whether from stress of pregnancy or severity of the attack, or both, Mrs. Buchanan did not improve. Her peak flow stayed between 80 and 150 liters per minute, about 20 to 30% of the predicted value. Arterial PO₂ remained safe at 70 to 90 but her CO₂ showed an ominous climb upward, to the mid-40s; in an acute asthma attack the higher the CO₂ the greater the severity.

The most severe asthmatics – patients with profound respiratory failure who are in imminent danger of asphyxiating – end up intubated and connected to a breathing machine (if they get to the hospital in time). Mrs. Buchanan was not at this stage but she was also not moving in the right direction.

On hospital day two we doubled her methylprednisolone dose (treat the mother) and started an antibiotic, erythromycin. We continued monitoring both maternal and fetal heart rate. Mother: 120; baby: 162.

The obstetricians saw Mrs. Buchanan twice a day. Their notes were terse and not generally helpful: "Not in labor. Baby OK. Continue current management."

"The baby's fine," Dr. Senior told me 48 hours into Mrs. Buchanan's unremitting asthma attack. "If you get her over this attack we'll let her go to term. Her due date is almost six weeks away. Just in case, I obtained permission for a C-section. How's she doing?"

"Not so great," I said. "The attack hasn't relented. What if she doesn't improve or gets worse? When would you want to do a C-section?"

"It's hard to say. There's a risk at thirty-four weeks. Also, if she suffered any hypoxemia during intubation it could be a real problem for the baby." He was right, of course. Surgery during a severe asthma attack can be a nightmare for mother and child. In addition, the baby would be born premature. Two strikes is no way to start out life.

By the afternoon of the third day she was clearly tiring out. Incessant dyspnea, muscle fatigue, and lack of sleep were taking their toll. Her peak flow was now consistently *below* 100 l/minute and the CO₂ level was almost 50! Blood oxygen was still adequate but everything else pointed to one inescapable conclusion: Mrs. Buchanan's asthma attack would not relent until she was delivered of child. I called Dr. Senior and gave him my assessment.

"You could be right," he said. I knew I was right but he'd have to make the final decision. "I'll be over in a few minutes to see her."

Mrs. Buchanan didn't wait. Five minutes later she became more fatigued and then suddenly *unarousable*. A 'stat' blood gas confirmed the worst; she was severely acidotic from a buildup of carbon dioxide. Treat the mother!

An anesthesiologist was called and within minutes Mrs. Buchanan was intubated and connected to an artificial ventilator. We had to give some IV sedation to coordinate her breathing with the machine; it was that or risk losing the baby to maternal distress.

Minutes after the intubation Dr. Senior arrived. He saw what had transpired and immediately focused his concern on the baby. "What happened to the fetal heart rate when she was intubated?" he asked.

"Here, Dr. Senior." One of the nurse's handed him a long paper strip, a continuous recording of the baby's heart rate. He scanned the strip from beginning to end. For about 30 seconds, just at the time of intubation, the baby's heart rate had fallen to 130 beats per minute, signifying a slight amount of fetal distress. Now it was back up to 150. Sedation had apparently not caused any major problem.

Turning toward me he said, "What do you think?"

"I think the baby's got to come out. We've given her everything and she's going nowhere. Her lungs are tighter than on the day of admission. She's in profound respiratory failure. I think we got to her just in time with

the ventilator. I recommend C-section if it's at all possible." I knew it was possible, I just wanted him to make the decision. I don't like to force the hand of any surgeon.

"It's possible, just risky."

I pointed to Mrs. Buchanan. "True. But this is riskier. Besides, shouldn't a C-section be safer now that we can control her breathing?"

"Sure, but the baby will be born premature and could suffer more distress. Of course there's also the risk of leaving him in there. What's the mother's PO2?" he asked.

"One twenty. That's on forty percent oxygen."

He thought for a few seconds then announced. "We'll do it. You'll take her back afterwards?"

"No problem. She belongs here. You get to keep the baby."

We went out together to speak with Mr. Buchanan about the emergency C-section. A factory worker in his late 20s, he still had on his blue work clothes and steel-toed shoes. We explained the situation, the risks both ways, the worst that could happen if we did and didn't operate. He was in a state of bewilderment and left the decision in our hands, essentially reaffirming his earlier permission for the C-section.

Dr. Senior next placed a call to the head of neonatology, activating the hospital's protocol for high risk delivery. An hour later Mrs. Buchanan left MICU en route to Labor and Delivery, a floor above MICU. Safe transport required a team of four nurses and technicians: two to handle the cart, one to breathe her manually with an Ambu bag, and one watch the maternal and cardiac monitors.

Once in the delivery suite Dr. Senior and the anesthesiologist became responsible for her care. I went along as an observer, and to help in any way necessary.

* * *

"This lady is tight," said Dr. Kazeem, the anesthesiologist, "she's requiring a lot of ventilator pressure." I. T. Kazeem, a short, balding man in his 50s whom I have seldom seen dressed in anything but green surgical, has probably given more high risk anesthetics than anyone in the city. Seeing him in L&D did a lot to ease my own anxiety. His comment was also a question: were we doing everything possible for Mrs. Buchanan's asthma?

"I know," I said. "She's on maximal steroids, the works. The baby's got to come out before she'll improve."

Dr. Kazeem turned his attention to the surgeon. "How quick are you, Dr. Senior?" A gentle reminder that speed was of the essence.

"Can you give me ten minutes of good anesthesia?" Dr. Senior replied.

"I can if her blood pressure holds up. I'm going to give her one hundred percent oxygen."

“The baby’ll love it,” responded Dr. Senior. “He’s already high on aminophylline. Fetal heart rate?”

“One sixty and holding steady,” replied a nurse.

“Scalpel.”

“Retractors.”

“Move that light a little bit down her abdomen. That’s better. Hold it.”

The dialogue sounded like some sort of Grade B movie. Only it was authentic, the patient’s life *was* in balance, and our sweat under the hot ceiling lights was all too real.

“Mother’s pressure is one hundred systolic. How’re you doing with her belly?” Dr. Kazeem was sitting at Mrs. Buchanan’s head and could not easily view the operation. I, on the other hand, could see both Dr. Kazeem and the operative field.

“I’m getting there,” Dr. Senior replied. He was assisted by a third-year obstetrics resident and two scrub nurses. Between them I could see the incision. A wide low swath above her pubic bone exposed the distended uterus. Next Dr. Senior had to cut the uterine muscle. Slice. Slice. So easy in skilled hands. Be careful, a baby’s in there! He cut some more and the abdominal muscles parted. Two hands disappeared into the cavernous sac. Ten seconds later the hands came out holding a pink baby – a girl. With one free hand Dr. Senior suctioned her mouth.

“Whaaaaaaa! Whaaaaaaa!”

Success. He handed her over to the neonatologist.

“How’s mother doing?” Dr. Senior asked, ever so calmly.

“Holding steady. Sew her up,” said Dr. Kazeem, relieved that one of his two patients was free and clear. I shared the relief. We all did. Whatever happened to Mrs. Buchanan, her premature infant entered life with an excellent chance of survival.

“She weighs twenty-two hundred grams. A little slow in reflexes,” said the neonatologist. “Apgar is seven. I think she’ll be all right. We’ll keep her in NICU [neonatal intensive care unit] for a few days.”

I walked over to view the crying infant. She looked fine to me, just a little small. I returned to the operating table and slipped my stethoscope under the drapes, to auscultate Mrs. Buchanan’s mechanically-ventilated lungs. Still wheezing, still tight.

* * *

I was right. Despite the pain of a hysterectomy incision Mrs. Buchanan began to respond to our drugs. Rapidly. Within 36 hours we were able to extubate and disconnect her from the ventilator. She still wheezed, and her asthma attack was far from over, but recovery was now just a matter of time.

Her first post-extubation words: “When can I see my baby?” “Soon, very soon. We can’t bring Abigale to MICU because she might catch

something. And you can't go to her until we're sure you're stable. We'll watch you a few more hours, then take you to her room." Mother and baby were reunited that evening. Breast feeding was out of the question but with some help Mrs. Buchanan bottle-fed and changed a diaper. The next day we transferred her out of MICU, to the OB ward, where her asthma continued to improve. As for Abigale, despite prematurity she developed no major complications. She went home seven days after birth, in the arms of her mother.

Comment

Mrs. Buchanan decided to have no more children and underwent tubal ligation. Her asthma continues to be easily controlled with pills and inhaled medication. As for Abigale Buchanan, she is growing normally and shows no signs of asthma.

-- END --

6. "We can't kill your mother!"

As an intensive care doctor I've dealt with many ethical dilemmas, most involving decisions to start or stop artificial ventilation. One of the most difficult was that of Mrs. Virginia Tyson, an 80-year-old nursing home resident admitted to the medical intensive care unit April 27, 1989.

A month earlier she had fallen and fractured her right hip. She underwent a hip repair and returned to the nursing home, but had not walked since. Additional diagnoses were rheumatoid arthritis, emphysema, and cardiac disease. Dehydration, on top of her bed-confined state and chronic lung disease, led to acute respiratory failure. Shortly after arrival to MICU we had to place an endotracheal tube through her mouth and begin artificial ventilation.

The severity of her condition made it impossible to remove the tube and discontinue the machine ventilation. Although her acute medical problems were eventually corrected she could not be disconnected from the ventilator; each attempt led to severe shortness of breath. On May 4 she underwent tracheostomy, a procedure that places a short plastic breathing tube through an opening in the neck, allowing the larger and more uncomfortable mouth tube to be removed.

With tracheostomy a patient can eat while receiving artificial ventilation. The 'trach' tube is also much easier to care for than a mouth tube, and can remain in place indefinitely.

Mrs. Tyson's need for artificial ventilation did not improve after tracheostomy. She simply did not have the strength to sustain breathing without the machine.

Her closest family was two daughters, one of whom lived in a nearby state and the other far away, in Seattle. The nearby daughter stayed in town and visited her mother daily, and was in phone contact with her sister.

Mrs. Tyson remained mentally alert. She could not talk with the tracheostomy but was able to communicate with a pad and pencil. SIT ME UP. IS MY DAUGHTER HERE? MY THROAT IS SORE.

Reflecting a trait I've noted among the elderly in MICU, Mrs. Tyson never asked about her disease or prognosis. "We're trying to get you off the breathing machine," we told her often; she always nodded in appreciation but didn't raise any questions. She also never objected to the care we gave her.

I met with the daughter almost daily. A thin, pleasant woman, she was quite understanding about her mother's lack of progress. She never challenged what we were doing, and only asked that "mother be made comfortable." I felt no communication barrier between us, and also saw none between her and the MICU nurses.

Considering Mrs. Tyson's age, chronic illnesses, and ventilator-dependency, we raised the question of additional life support should other organs fail. Mrs. Tyson and her daughter requested no further life support or resuscitative measures, and a 'Do Not Resuscitate' order was entered in her chart.

No other organs failed and her lung condition did not improve. She remained stable, albeit ventilator-dependent. Our hospital allows for stable ventilator patients to go to a regular ward, so on May 9 we transferred Mrs. Tyson out of MICU.

At the request of Mrs. Tyson's internist I continued to follow her on the ward, and made another attempt to wean her from the ventilator shortly after transfer. When this failed I suggested that social service look for a nursing home that would take ventilator-dependent patients. Mrs. Tyson's original nursing home could not accept her back with the ventilator.

No matter how routine ventilators become for a hospital they are never routine anywhere else. In 1989 only two nursing homes in our metropolitan area accepted ventilator-dependent patients and they were both full. Still, we had no choice but to look for placement of Mrs. Tyson in a chronic care facility. She could not come off the machine without dying.

Attached to the machine she could in theory live many more years, although a sudden event, such as pulmonary embolism (blood clot in the lungs), could also end her life quickly. Her DNR status meant that we would

not intervene if another bodily system failed, but it did not change the day-to-day care she needed and received. I felt sorry for Mrs. Tyson and her daughter, but there was nothing more we could do except continue medical care and attempt placement.

On May 15 I got a call from the resident caring for Mrs. Tyson. “Her other daughter is here and wants us to turn the ventilator off. She says her mother wants to die.”

“I’ll be right up.”

In a few minutes I met this daughter, standing alone outside her mother’s room. The older of the two children, she appeared to be in her late 40’s, physically similar but more aristocratic in bearing than her sister. As to temperament they seemed totally different. The Seattle daughter had only arrived that day, but wasted no time in getting down to business: “Dr. Martin, I want Mother disconnected from the ventilator.”

“What? Your mother can’t live off the ventilator.”

“I know that. I know what I’m asking.”

She had already learned about our failure to remove the ventilator. Calmly, I expressed amazement at her demand.

“Why all of a sudden?”

“Doctor it’s *not* all of a sudden. Mother has always expressed her wish to die instead of being connected to a machine.”

“But I’ve cared for your mother for three weeks. Neither she nor your sister said anything about turning the machine off.”

“Did you ever ask them?”

“No, it never came up. We made her DNR, that was your mother’s wish. But you’re asking something totally different. If your mother had refused artificial ventilation before we began, it could have been withheld. Mentally competent patients have every right to make such decisions. But neither your mother or sister ever made that decision. What you’re asking now doesn’t make sense.”

“I’m telling you what Mother wishes. My sister was just too timid to bring it up.”

‘Wow!’ I thought. There’s something strange going on here. What, I didn’t know, but it got stranger.

“Let’s go ask Mother now,” she said. In disbelief I followed her into Mrs. Tyson’s room.

There was no introduction to the subject. Not even, ‘Mom, I brought the doctor in to discuss this matter.’ Instead:

“Mother. Do you want to die?”

Mrs. Tyson nodded yes. There was no emotion to the nod. Just a dutiful yes.

“See,” said her daughter. “Now will you disconnect the ventilator?”

It was time to be more forceful. I had a tough and determined woman on my hands. “Could I speak to you outside?” She agreed and we left Mrs.

Tyson's room.

"I'm sorry but I cannot disconnect the machine. I respect your wishes and am not going to ignore your request. But I'm not the only one caring for your mother. This will have to be discussed with other physicians and the hospital's attorney. You have to understand, what you're asking has never been done before in this hospital. There is no way any single physician or nurse can just go in and disconnect a ventilator. We can't just walk in and kill your mother!"

"I'm *not* asking you to kill my mother. I'm only asking you to let her die a natural death."

"I know that's the way you see it. But you have to look at it from our perspective."

"When can you call these other physicians and the attorney? I want to get this resolved as soon as possible. Mother's suffered enough."

"How long will you be here this afternoon?" It was one o'clock. "I'll stay as long as necessary."

"OK. I'll make some phone calls and see what I can arrange."

I called the chairman of our ethics committee (of which I am also a member) and explained the situation; he agreed to meet the next day, at noon. I then called the hospital attorney and outlined my understanding of the legal issues; she also agreed to the meeting. The nurses, social worker, and medical housestaff caring for Mrs. Tyson were also informed, and by 3 p.m. I had everything lined up. I returned to the ward. This time both daughters were there. Somewhat to my surprise, the younger daughter expressed total agreement with her Seattle sibling but also admitted to being "not very good at verbalizing Mother's wishes." Verbalizing? She hadn't ever *suggested* what now seemed written in stone.

"Are you sure your mother wants to die?"

"Oh, yes. Mother told me that many times. She's said that for years. She just never got a chance to say it here." Her tone was unsettling, almost accusatory. I was glad a meeting was arranged for the next day. Let others hear this.

"Now," challenged the lady from Washington state, "will you disconnect Mother from the machine?"

"I already told you that's impossible for me to do. I've spent the last two hours arranging a meeting for tomorrow, at noon. Our lawyer and the chairman of the ethics committee will be there. Is that time OK?"

"Yes. We'll bring our family lawyer."

* * *

The next morning I came to see Mrs. Tyson on rounds and was immediately confronted by the head nurse.

"Mrs. Tyson's daughters were here last night. They were asking the

night shift why Mrs. Tyson was being tortured, and why she can't be disconnected from the ventilator. Mrs. Tyson even wrote a note asking to be disconnected. The nurses are really feeling strained by their attitude. What are you going to do?"

I reminded her of the meeting and left it at that. Clearly, this issue had to be resolved quickly. I took some comfort in realizing that, having called the meeting, the decision was now out of my hands.

There were 12 people at the noon meeting, held in a library off the ward: nine from the hospital staff, plus Mrs. Tyson's daughters and their attorney. I adopted the role of moderator, to both provide medical background and make sure the daughters' wish was fairly presented. It was also important that everyone understand the background against which such an extraordinary request was being made.

After introductions I briefly presented Mrs. Tyson's medical history, emphasizing that at no time did she or her younger daughter ask for the ventilator to be turned off. I explained how she was made DNR, and that this did not translate into disconnecting the ventilator in an awake patient under any circumstances.

I also explained how, because of emphysema and other medical problems, her lungs were damaged beyond repair and that I saw no prospect for her living without the ventilator. I offered my best medical judgment that without the machine she would die within 24 hours.

Then, looking at the older daughter I commented that "we were all surprised when you showed up and asked to have her machine disconnected."

I was not asking for a response but she volunteered one: "I'm truly sorry I didn't come earlier, but it was impossible. I am now here to see that Mother's wish is granted."

Looking toward the younger daughter I remarked, "I understand you are in agreement with this request?"

"Not only am I in agreement, it's what Mother wants. It's what we want. It's what should be done!" What conviction. Where had she been the last three weeks?

The floor was open for discussion. The night nurse on the ward spoke first. "Mrs. Tyson wrote me this note last night." The note was passed around. I had not seen it before. The ethics chairman suggested I read it out loud.

'PLEASE LET ME DIE. I DON'T WANT TO GO ON LIVING THIS WAY. Virginia Tyson.' Was either daughter present when she wrote this note?" I asked.

"No," said the head nurse. "Both had left the hospital."

The older daughter spoke up. "Look, I know this must seem strange to all of you, but you people don't *know* my mother. She never wanted to live like this. Mr. Barnes, the family lawyer, has known Mom for 40 years." She

turned to the elderly gentleman, at least as old as Mrs. Tyson.

“That’s true,” he affirmed in a creaky and barely audible voice. “She told me many times not to let this happen.” (Why had none of this been made clear prior to admission?)

The ethicist spoke up. “I’m Dr. Knowles. I was asked to come because I head the hospital’s ethics committee. I don’t know your mother and have not cared for her, but I’m a physician and have cared for many patients in similar circumstances, that is, elderly patients connected to a ventilator.

“I think I understand what you’re asking. One problem we’re all having – at least something that bothers me – is how this has developed. You are absolutely correct. The patient has a right to determine her destiny. The problem, from a purely ethical and moral perspective is, what is her real wish? I don’t doubt for an instant your sincerity. It’s just that I’m having trouble separating your mother’s true desire from what she may be expressing out of guilt, perhaps for what her illness is doing to the two of you.”

There! He said what we were all only wondering. Was Mrs. Tyson asking to die so as not to be a burden on her daughters – especially the older one, for whom the constraints of time, if not distance, seemed more of a problem than the younger daughter? Or were the sisters merely conveying what was truly their mother’s wish from the very beginning?

Mr. Barnes objected. “That’s not true. Mrs. Tyson has always said she didn’t want to live like this.”

Dr. Knowles responded. “I don’t doubt that, Mr. Barnes. And I’m truly respectful of the awful situation she’s in. I don’t think any of us would want to live under these circumstances. I’m just expressing why we’re all so surprised, and why it’s difficult to accept what you are asking. If she had made this wish clear from day one, and the family agreed, then I think ethically there would be less confusion on the issue. I’m not saying we’d take her off the machine even then. It’s just that, from an ethical viewpoint, the request would seem less unreasonable.”

“Are you saying you won’t take my mother off the machine?” asked the older daughter indignantly. Good, I thought; let everyone see what I’ve been up against.

It was our lawyer’s turn to speak. A former RN, she was compassionate and direct. “First, let me say that I understand your request. I really do. I know your mother has no chance of getting off the ventilator, and I accept that it may be her wish to die rather than go on living this way. The truth is, under state law we can’t disconnect the ventilator. Your mother is awake and alert, and we can’t do anything that will lead directly to her death.”

“You mean I’ll need a court order to stop the machine?”

“Yes, I’m afraid so. I must warn you, though, that no court in this state has ever granted such a request on an awake patient. And if one did, we’d appeal it. Also, I don’t think there’s anyone in this room who would

personally disconnect your mother's machine."

"Speaking for myself," I said, "as one of her physicians I could not disconnect the machine and watch her die, court order or not. Would any of the nurses be able to do it?"

The three nurses in the room quickly shook their heads and the discussion was over. Mrs. Tyson's daughters had presented their demand, entirely reasonable in their eyes, but unreasonable from a legal and ethical perspective. The daughters had lost the first round but they were prepared for the outcome.

Without missing a beat the older daughter said: "Then we'll take mother home."

* * *

Over the next several days prodigious arrangements were made to transfer Mrs. Tyson to the home of a local relative. We made it clear to the daughters that she could not be released until we felt assured she would receive adequate care. Sending ventilator-dependent patients home is not impossible and we have done it before. It just takes a lot of planning and some commitment on the part of the family.

The hospital's attorney felt that we could not block Mrs. Tyson's discharge unless we had evidence that the daughters might harm her. We had no such evidence. In fact both daughters were accepting of our decision not to disconnect the ventilator, coming as it did from such a powerful show of force and determination. They cooperated with all the people involved in the discharge, including respiratory therapists, visiting nurses, and Mrs. Tyson's social worker. Arrangements to send a ventilator patient home ordinarily take at least a week, and they did not try to rush us.

On the evening of May 21, four days after our meeting, Mrs. Tyson was found dead in bed. Her daughters had not been there for several hours and the previous nurse check, only an hour earlier, had found the patient weak but otherwise stable. Her death was deemed due to natural causes. No autopsy was performed.

-- END --

7. The Yellow Man

Willie Duncan's body was a mess, the end result of a pint of whisky a day for God knows how many years. He was only 38 but looked 60.

When most people think of end stage alcoholics the visual image is probably one portrayed by Hollywood: a drunk stumbling down the street, or sleeping on a bar stool, or beating his wife, or abandoning her children for the bottle. The theater is also a source of impressions, for example the character of Eugene O'Neill's alcoholic mother in his autobiographical "Long Day's Journey Into Night."

Stage or screen, the portrayal is usually focused on the alcoholic's *behavior*: out of control, irrational, or self-destructive. But what about the *body*? Has Hollywood ever presented for our entertainment the physical persona of the terminal alcoholic? Hardly, and with good reason.

Maybe you'll see a closeup of a face just before the drunk slips into alcoholic stupor: grizzly beard, red lips, bloodshot eyes. And perhaps you'll hear some well rehearsed lines: "I don't care – BURP! – if you leave me – BURP! – Give me a drink – BURP!" ...fade away.

Movies – and television, for that matter, should not be faulted for glossing over the true clinical picture. In many cases it's too unpleasant, too *gory*. The kids might love it if the alcoholic was also a werewolf or vampire, but kids aren't interested in the last throes of an ordinary drunk.

You could go into any teaching hospital on any given day and find a patient like our Willie Duncan. He's yellow, head to toe; the eyeballs give it away. Normally white, Willie's eyes were a deep, obvious yellow, the result of liver failure and accumulation of yellow-tinged bile.

End-stage alcoholics have arms and legs thinned by malnutrition. Examine the skin of the forearms and chest and you'll likely see purple blotches, the result of fragile capillaries and easy bruising. Take the bed covers off and you may find a distended, tense belly, like that of a child with kwashiorkor. The physiology is the same – massive fluid buildup due to lack of protein. Protein holds water inside the blood vessels. Victims of kwashiorkor are starved for protein. In contrast, victims of alcoholism like Willie Duncan have a destroyed liver, and so can no longer manufacture water-holding protein.

Looking up from the beach ball belly you will often find a chest covered with red "spiders," superficial blood vessels in a star burst pattern about an inch in diameter. Press the center of one spider vessel and it will blanch; let go and it will fill quickly with blood from the center outward. These abnormal vessel clusters are another sign of severe liver failure.

The mind of the typical yellow man will likely be slipping away. Our Willie Duncan was confused and disoriented. He did not know the day, the

date, or the president (end-stage alcoholics older than Willie often answer “Roosevelt” or “Eisenhower”). Even when you think the terminal alcoholic is lucid, he is not. Ask him to count back from a hundred by sevens (100...93...86... 79...); he cannot do it.

There’s more. Search carefully and you’ll discover that the patient is bleeding internally. As the liver shrinks from the effects of alcohol the veins inside the esophagus – the tube connecting mouth to stomach – become engorged with blood. Normally, esophageal veins route their blood through the liver. The more the liver shrinks the more these veins distend until, like a balloon continuously filled with air, they burst open and spill blood into the esophagus. The dark red blood is both vomited up and passed in the stool, where it appears as mahogany-colored diarrhea.

Even if you’ve seen the Willie Duncans of America before, you will stare in amazement each time another yellow, bloated, bleeding patient comes to the hospital. It is a startling spectacle.

Not that our Willie Duncan wasn’t forewarned. He was now in for his seventh hospital admission in as many years. Long before alcohol turned his liver into a rock hard lump of scar tissue Willie was told what to expect if he didn’t stop drinking. During every hospitalization he was advised, cajoled, threatened: stop drinking.

In the early days he had a wife, a brother and sister; his relatives were told to get Willie to stop drinking or he would die from liver failure. Eventually his wife left him (or him her, it isn’t clear which). Willie’s brother was killed in a fight, leaving only a sister as immediate family. He had no children.

Nothing worked for Willie. Like most terminal alcoholics, Willie was poor. He had once held a steady job as a cab driver but was now on welfare and living with his sister. There was certainly no money for an expensive alcohol treatment program even if he were motivated to join one. His sister once persuaded him to enter Alcoholics Anonymous; he attended meetings only a few months before the bottle lured him away.

Willie’s case reflected a sad fact: it is far easier to convince patients to go on a diet, exercise, or quit smoking, than to stop drinking. Alcoholism is a certifiable and often incurable *disease*. Except for a few specialized centers, most of which are expensive and closed to the uninsured, medical treatment is relegated to the *effects* of alcohol and not to the addiction itself.

In truth, Willie Duncan’s alcoholism was as untreatable as terminal cancer. Willie Duncan came to MICU to die. The method chosen by his body was exsanguination. He was admitted one day in June with massive gastrointestinal bleeding.

In MICU we estimated that his engorged esophageal veins oozed blood at a rate of about 200 cc’s an hour. Without some intervention his body would be empty of blood in 24 short hours. Long before that he would die of shock.

Everyone in MICU knew Willie was terminally ill. But only 38 years old! How could we let him just bleed out? Doctors don't like to watch people bleed to death, even if therapy appears futile. We had to try something. We tried everything.

First we replaced his blood. In the first 48 hours he received eight units of blood and two units of fresh frozen plasma. Shortly after admission, during his first blood transfusion, gastroenterologists passed a flexible scope (a procedure called 'endoscopy') into Willie's gut to identify the sites of bleeding.

As expected, the gastroenterologists found bleeding esophageal veins. (In addition to engorged esophageal veins, alcoholics can also bleed from stomach and intestinal ulcers. Ulcer bleeding occurs from erosion into an *artery*. Esophageal bleeding is from a leaky or ruptured *vein*. The two causes of bleeding are treated differently.) His bleeding veins were injected, through the endoscope, with a latex material.

Endoscopic latex injection stops hemorrhage by changing the free-flowing venous blood into a harmless clot, but the technique works only about 60% of the time. Initially it seemed to work for Willie. Then, just five hours later he vomited up about 100 cc's of dark red blood.

The next therapy tried was intravenous injection of 'vasopressin', a potent vasoconstrictor. Vasopressin has long been used to control bleeding from the gastrointestinal tract. For stomach and intestinal bleeding the vasopressin must be infused directly into the bleeding artery. This *intra-arterial* infusion requires threading a long catheter through the thigh artery and then into the bleeding vessel itself, a highly invasive and specialized technique.

For bleeding esophageal veins – alone among causes of bleeding within the gastrointestinal tract – vasopressin works just as well when infused through a peripheral arm vein. We began Willie on 0.4 units of vasopressin per minute through an arm vein.

For the next 12 hours he was stable: no bleeding. Then he retched, leaned over the side of the bed and vomited up several hundred cc's of dark blood.

The gastroenterologists were summoned. What to do now? Surgery was not a viable option. No good operation exists for bleeding esophageal veins and Willie would not have survived surgery in any case. There seemed but one more thing to try: a Sengstaken-Blakemore tube. This three-foot long, hollow tube was introduced in the 1950s and named after the inventing surgeons. Once the main form of treatment for massive esophageal bleeding, the S-B tube is now used only rarely and as a last resort, when latex injection and vasopressin infusion fail.

The S-B tube is affixed at one end with two inflatable balloons, one for the esophagus and a smaller balloon for the stomach. When inflated with air, the esophageal balloon forces pressure against the hemorrhaging veins,

much as you might stop bleeding from a cut by applying pressure. The esophageal balloon is basically a mechanical technique whose success depends greatly on proper positioning and the right amount of pressure.

To prevent the inflated balloons from moving and dislodging, the outside end of the S-B tube must be attached to a fixed object. Years ago the outside part of the S-B tube was taped to the patient's forehead but the tape always peeled off when the patient was moved. Then someone had a brilliant idea: use a football helmet. An S-B tube tied to the face mask of a football helmet works beautifully. End-stage alcoholics with esophageal bleeding all wear football helmets in the ICU!

An S-B tube was placed into Willie's stomach and the balloons inflated. Amazingly, the bleeding stopped. But what a sight! A thin, emaciated, disoriented yellow man with giant belly. Wearing a football helmet emblazoned with the emblem of our city's professional team. For two days Willie Duncan lay like a wounded linebacker, the bleeding stemmed by fragile balloons pressed against his gut.

Jokes are inevitable about such patients. "Willie's trying out for the team – at the rate they're going, he'll make first string." "I just got a call from [the coach]. He wants to see Willie right away. Let's clean him up." "Willie should try out; he won't hurt their chances for the superbowl." That year, our team and Willie Duncan were losers.

(Jokes are a necessary valve for ICU staff. We never really laugh at patients. We laugh at situations, at circumstances, at the human condition, and at ourselves. It is a healthy laughter.)

Six different doctors became involved in Willie's care this time around. If you asked each doctor what he or she honestly thought about his chances, none would admit to any optimism. I don't believe anyone held out much hope for Willie. I know I didn't, but still we tried. Was it inappropriate to do so? Should we have quit because Willie was an incorrigible alcoholic?

Given his age, I don't think so. You never want to let a patient's lifestyle influence your care. It would be ethically wrong to limit care *because* he was an alcoholic. Once you start making that type of judgment you are on a very slippery slope. It is then only a small step to withholding care because someone smokes cigarettes, or eats too much, or doesn't get enough exercise, or drives a foreign car.

The only justification for holding back is if what you have to offer will not help the patient. It is as morally wrong to offer treatment that cannot benefit the patient as it is to withhold treatment that may. Not being a specialist in esophageal bleeding and liver failure, I didn't want to make any life and death decision about Willie Duncan. If the gastroenterologists wanted to try multiple latex injections and ten S-B tubes (or surgery, for that matter) I could not legitimately object. Only the most experienced physicians should say there is nothing more to offer a dying patient.

Two days after the S-B tube was placed Willie's vessels opened up.

Blood gushed everywhere. Dark red blood oozed out and around the S-B tube. Beneath his buttocks lay a puddle of mahogany-colored diarrheal stool. Before another unit of fresh blood could be infused Willie's heart stopped beating. Willie Duncan bled to death.

Comment

We obtained permission for an autopsy from Mr. Duncan's sister. At autopsy his liver was markedly shrunken to only about half its normal weight. His esophageal veins were enormous, over two inches across. One vein had a large rent down the side, the site of his fatal bleed. There was no evidence for cancer or infection.

Chronic liver disease is the ninth leading cause of death, claiming about 26,000 lives yearly. Many of these liver disease victims – like Willie Duncan – succumbed to the effects of chronic alcoholism.

– END –

8. Adult Respiratory Distress

Joe Woodbury, 35 years old, was healthy until January 11, 1982, when he developed a scratchy sore throat, no different in character or intensity from what many of us suffer every year. Even in retrospect, those early symptoms suggested nothing more sinister than a minor upper respiratory infection. Two aspirin gave some relief, but that evening he also developed a slight cough, fever of 100 degrees and a general achy feeling. His wife called their family physician, Dr. Levinson, who reviewed the symptoms over the phone. Everything certainly sounded like the classic flu syndrome, which usually gets better in three to seven days. Dr. Levinson reinforced the need for aspirin and asked to be called in two days if there was no improvement.

The next morning Mr. Woodbury was worse. He ached all over and cough was painful, so he went to see Dr. Levinson, who listened to his heart and lungs and heard nothing unusual. Eyes, ears, nose and throat were normal except for a flushing of the mucous membranes. On the outside chance that this was a bacterial infection, he prescribed an antibiotic, erythromycin, to be taken four times a day. He reassured Mr. Woodbury and asked him to call the next day if he was no better.

That evening Mr. Woodbury developed a mild sensation of shortness of breath, what physicians call dyspnea. Partly for this reason he had a restless night and the next morning, January 13, was back in Dr. Levinson's office. Now there was also a new physical finding, cyanosis, a slight bluish skin color indicating insufficient oxygen in the blood. Dr. Levinson also found Mr. Woodbury's breathing heavier and deeper than normal. Everything pointed to a lung problem, so a chest x-ray was taken immediately. It was not normal. In the right lung was a grapefruit-sized, irregular white shadow just above the diaphragm, consistent with some type of pneumonia. Thirty minutes later Mr. Woodbury was admitted to the Medical Center's ICU (intensive care unit) with a presumed diagnosis of viral pneumonia.

The pace is fast in the ICU. Within the hour Mr. Woodbury gave a brief history to two doctors and a sputum sample to one of them, had a physical examination, several blood tests, and another chest x-ray, and began receiving intravenous fluids. One of the blood tests, known as arterial blood gas, showed his oxygen pressure dangerously low at 37 millimeters of mercury. Normal PO_2 , as the test is abbreviated, is 85 to 100. To help counteract his hypoxia, an oxygen mask was set up to deliver 60% oxygen — almost triple the amount in room air.

I saw Mr. Woodbury soon after admission. His overall appearance can

be described as “acutely ill” – an observation based mainly on his dusky skin color, sweat over his brow and obvious breathing difficulty. Also, his muscular build and full, round face made it certain he hadn’t been ill for very long. Despite being short-winded he was alert and cooperative. He also seemed strangely optimistic for someone so precipitously admitted to hospital, as if he perceived his condition to be easily curable by medical science. He had no particular reason to be cheerful, so I sensed this was his way of reassuring Mrs. Woodbury, who had just seen him and was now out in the waiting room.

I learned something of their family situation. The Woodburys had two children, ages four and seven. He worked as a foreman at the Ford Motor plant and his wife held a part-time secretarial job in the mornings while their younger child was in day care. Neither his children nor Mrs. Woodbury had been ill recently. Mr. Woodbury had not been hospitalized before, in fact had never been very sick, and did not smoke. He also had not been recently exposed to noxious fumes, chemicals or dusts.

I went to see Mrs. Woodbury, a petite and pretty woman in her mid-30's. She was scared, which considering the circumstances was an appropriate reaction. Before I could explain his problem she wanted to know just how sick he was and “would he make it?” She might have sensed something in my demeanor, or my lack of smile or the way I held my head. (Some ICU physicians believe in laying out all the worst possibilities from the very beginning, at least to the patient’s family. This is called “hanging crepe,” referring to the black fabric displayed at wakes or funerals. Once this approach is taken, anything bad that happens will have been expected; anything good will make the physician look like a hero. The truth is, I had seen many patients similar to Mr. Woodbury and “guarded” was an optimistic prognosis. Patients with such a rapidly progressive pneumonia can be dead a few days after their first symptoms. Still, except in the most obvious cases of brain anoxia, to emphasize only the worst possibilities is not fair to the family and could even be self-fulfilling.)

I told Mrs. Woodbury that her husband had severe viral or bacterial pneumonia and it was seriously interfering with oxygen delivery into his blood. His course appeared so rapid that if he did not begin to recover soon he would need artificial ventilation and even that would provide only temporary support. We would order specific diagnostic tests, such as blood cultures and microscopic examination of his sputum, and continue treatment with oxygen and antibiotics. He would either respond or not and we would know in 24 to 48 hours. There was a reasonable possibility he would improve but I could give no odds. She accepted this, which is to say all of her immediate questions were answered. I also called Dr. Levinson at his office and told him of the situation. He agreed with our approach.

The initial tests revealed increased numbers of infection-fighting white cells, both in his blood and sputum. We also found his sputum devoid of any

bacteria – a finding which could be explained by his already brief use of erythromycin, which might have suppressed their growth. Alternatively, he could be infected with organisms that don't show up on routine sputum examination – this includes all viruses and many bacteria as well. In any case, the chest x-ray, white cell count and sputum exam all suggested a diagnosis of pneumonia, but did not reveal what specific type. Dozens of different organisms – including many species of virus, bacteria, fungi and protozoa – could be responsible.

(By January 1982 AIDS had just been described and most physicians had never seen a case. Almost all the cases reported up to that time were from California or New York. Therefore AIDS was never a consideration in Mr. Woodbury, although we did look for organisms now commonly associated with AIDS infections.)

We started Mr. Woodbury on two intravenous antibiotics, erythromycin and oxacillin. It was decided to continue the erythromycin because that is the best treatment for Legionnaire's disease, which we had not ruled out, and also for mycoplasma pneumonia. The bacteria responsible for mycoplasma pneumonia (*mycoplasma pneumoniae*) and Legionnaire's disease (*legionella pneumophila*) share certain characteristics: both are difficult to diagnose in the first few days of illness; neither can be seen under the microscope using conventional laboratory methods; infections caused by both usually respond to erythromycin. Thus the drug seemed a logical choice for Mr. Woodbury. Oxacillin, a relative of penicillin, was chosen because it is excellent against staphylococci. Staphylococci are a much more virulent group of bacteria than either *mycoplasma* or *legionella*; whenever a serious "staph" infection is suspected, treatment is begun immediately, without waiting for confirmation.

Mr. Woodbury did not respond. A few hours later he was still dyspneic and cyanotic and his PO₂ was only 45. An oxygen pressure this low – especially while receiving extra oxygen – is always life-threatening. Improving oxygenation at this point would require a major change in management since the oxygen mask was ineffective. Mr. Woodbury needed artificial ventilation, which meant placing a tube in his trachea (the throat), a procedure called endotracheal intubation. We asked the anesthesiologist on call to come up to the ICU and intubate Mr. Woodbury.

Normal breathing, which involves inhaling and exhaling 10 to 16 times a minute, is silent, automatic and effortless. It is also not obvious to the observer. Mr. Woodbury was now breathing 40 times a minute and working very hard at it. From across the room anyone could see his neck muscles rise and fall, a sure sign of increased work of breathing – yet each breath was ineffectual and their sum not enough to sustain life.

Despite severe respiratory distress he remained alert, so I carefully explained what was about to happen. I told him intubation was necessary (the ventilator could not be effective otherwise), that he would have to be

sedated for the procedure and that even when awake he would not be able to talk or eat. The ventilator would take over the work of breathing for him. He understood and asked me to call his wife, who had since gone home. I said I would call her afterwards. Fortunately the intubation was quick and successful; he required 10 milligrams of intravenous Valium for sedation.

The ventilator, a machine about the size of a dishwasher, was hooked up to his endotracheal tube via plastic hoses about two inches wide. The dials were set to deliver 14 breaths per minute, with the volume of each breath quadruple the amount he was breathing on his own. To make it easier for him to tolerate the endotracheal tube and not “fight” the ventilator we gave him another 10 milligrams of Valium.

A chest x-ray was taken with a portable machine; it now showed an abnormal whitish haze in both lungs. (An x-ray film is like the photographic negative of a black-and-white print. A white haze or shadow appears whenever pneumonia or some other abnormality blocks the x-ray beams. Bony structures such as ribs also block the beams and appear white on x-rays. Normal lung, which is mostly thin tissue surrounded by air, lets the x-rays through to develop that part of the film, which then appears black between white ribs.)

At this point the admitting diagnosis seemed correct – viral pneumonia. His sputum revealed no bacteria and it was too early for culture reports to confirm any other diagnosis. But Mr. Woodbury’s distress was due to more than just viral pneumonia. In just a few hours once-healthy lung tissue had come apart, allowing plasma to leak out and flood spaces where only air should be. His chest x-ray showed the progression clearly. This flooding of the lungs – the medical term is acute pulmonary edema – is not what you see in the usual case of viral (or any other) pneumonia. The problem had progressed beyond simple pulmonary infection.

* * *

Mr. Woodbury now displayed all the classic features of ARDS, the adult respiratory distress syndrome: acute onset of severe respiratory distress; bilateral “white-out” on the chest x-ray; life-threatening hypoxia. ARDS is one of those medical entities that has been around for a long time, but only recently recognized and defined. ARDS was first characterized in a 1967 article published in THE LANCET, England’s famous medical weekly. (The authors were all from the University of Colorado Medical Center – it is not unusual for Americans to publish first descriptions of disease in THE LANCET.) Drs. David Ashbaugh, D. Boyd Bigelow, Thomas Petty and Bernard Levine described twelve patients admitted to the Rocky Mountain Regional ICU, all of whom had symptoms and clinical findings similar to each other and to Mr. Woodbury: respiratory distress, severe hypoxia, leakage of fluid into both lungs and a need for artificial ventilation. These

symptoms in the twelve patients were precipitated by several different events, including viral pneumonia, pancreatitis and trauma to the chest or abdomen. A few of the patients were in shock prior to the onset of their respiratory distress. (“Shock lung” is sometimes used to mean ARDS; the latter is a much broader term, as shock is only one of many conditions that can precede ARDS.)

Five of the twelve patients died, a percentage which has remained about the same to this day. An autopsy of their lungs were a deep-reddish purple, much heavier and darker than normal, air-filled lungs. This reflected the tremendous inflammation and edema characteristic of ARDS. ARDS was certainly not new in 1967. The type of patient with severe lung leakage has been described increasingly in the medical literature since World War I, when injured soldiers were often observed to die in fulminant (sudden) respiratory failure. Autopsy studies in the 1930s and 1940s showed that many patients dying of shock had heavy, beefy lungs. In the 1950s and early 1960s severe respiratory distress was described after such disparate conditions as open-heart surgery, compound leg fractures and viral pneumonia. In 1966 the term “DaNang lung” was coined to describe the respiratory complications of soldiers critically wounded in Vietnam. In retrospect most – if not all – of these patients exemplified the adult respiratory distress syndrome.

The importance of the 1967 paper was in recognizing a pattern of injury not unique to one cause but the final pathway for multiple causes, and in describing the clinical features of ARDS. This subsequently allowed many patients with ARDS to be diagnosed, studied and treated in a rational, uniform fashion.

Precise statistics are unavailable, but when all the causes are considered ARDS turns out to be a common problem. In this country an estimated 75,000 to 100,000 new cases of ARDS occur each year, with about half of the patients surviving. A true incidence is difficult to come by since most ARDS cases end up classified in some other disease category, such as “viral pneumonia” or “multiple trauma.” Most ARDS patients are under the age of 65 with no prior history of lung disease. ARDS is not due to heart failure, although a rapidly failing heart can sometimes cause a similar clinical picture. (Some physicians prefer the terms “cardiac pulmonary edema” for severe heart failure and “non-cardiac pulmonary edema” for ARDS.)

ARDS is distinct from infant respiratory distress syndrome, also known as hyaline membrane disease – a condition that claimed the life of John and Jacqueline Kennedy’s son on August 9, 1963, two days after birth. Infant ARDS is due solely to premature birth and lack of a normal lung chemical called surfactant. While lack of surfactant plays a role in ARDS, it is not the primary cause.

Little has been learned about the pathophysiology of ARDS since 1967. Basically it involves the leakage of plasma and protein out of pulmonary

capillaries, flooding millions of tiny airspaces. (Our lungs are a collection of 300,000,000 or so microscopic airspaces, called alveoli; each alveolus is surrounded by numerous capillaries that take in oxygen and give off carbon dioxide. Normally the barrier between the airspaces and capillaries allows gases to exchange but nothing else, certainly not plasma and large protein molecules.) Why plasma and proteins leak out of the capillaries is not known. There are many theories but none is universally or even widely accepted.

One problem in understanding ARDS is that most patients suffering the commonly-associated medical conditions, such as shock or viral pneumonia, do not end up with leaky lungs. It is for unknown reasons that some victims manifest florid pulmonary edema while others, in a similar clinical situation, maintain fluid-free lungs.

Though there have been some advances in the management of ARDS, none is considered specific therapy; in the jargon of intensive care they are referred to as “supportive,” as opposed to “curative.” One general advance is the ICU itself, a development dating from the 1930s when polio victims were first concentrated for better care. The modern ICU, with electronic monitoring, artificial ventilators and specially trained staff, evolved in the 1960s and spread nationwide in the 1970s. Today every hospital of more than minuscule size has at least one area where modern monitoring technology and skilled nursing are concentrated. This is the true meaning of intensive care. (Though the concept of the ICU makes a lot of sense intuitively, the benefit of ICUs in reducing mortality has not been proved for most illnesses, including ARDS. Given the critical condition of these patients and the fact that only half may survive the illness, it is not a study anyone cares to perform; no one wants to give any ARDS patient less care than that available in the modern ICU.)

Another advance is the artificial ventilator, which has become more compact and reliable over the years. Unlike the old iron lungs, which hampered nursing contact with the patient’s, today’s artificial ventilators stand at the side of the bed and are connected to the patient through flexible tubing. They pump air into the lungs, rather than suck air from around the chest cage, which is what the iron lungs did. And modern ventilators can work round the clock for weeks with no more than routine bedside maintenance. It is seldom that any patient dies today from inability to be machine-ventilated.

Today’s ventilators are also able, at the twist of a knob, to deliver positive end-expiratory pressure, known worldwide as “PEEP” (pronounced to rhyme with beep). This extra airway pressure-maintained at the end of each breath – helps keep the alveoli open longer so more oxygen can enter the blood. PEEP for ARDS was first reported in the 1967 paper, although its roots go back much further. In the late 1940s pioneering jet aircraft pilots used positive-pressure face masks to increase their oxygen pressure at high

altitudes.

Clinical use of PEEP was only conjecture until the Colorado physicians placed five of their ARDS patients on positive airway pressure. Three of the five lived whereas only two of the seven non-PEEPed patients survived. Because of the small numbers of patients these results were, in the parlance of medical investigation, merely anecdotal, but they were enough to place PEEP in the pantheon of ICU techniques. PEEP's main use is to increase oxygen pressure in the blood without using extremely high, and potentially toxic, concentrations of inhaled oxygen.

PEEP is not without hazards; because of the increase in positive airway pressure the lungs can sometimes "blow out" like a burst tire. PEEP can also prevent the normal flow of blood into and out of the heart and cause heart failure. Those complications are manageable – and to a large extent preventable – with careful monitoring.

Machines that easily and rapidly measure blood oxygen and carbon dioxide tensions (the "blood gases") are another evolutionary development. Various methods for blood gas measurement have been available for decades, but technically easy and rapid measurements only since the late 1950s when new types of gas electrodes were introduced. Today, caring for critically-ill patients without blood gas measurements would seem like driving fast in a dense fog. You might make it but you would probably crash. Before the blood gas test was widely available physicians literally guessed at the blood oxygen and carbon dioxide tensions. It was a real crapshoot. Doctors now appreciate the unreliability, for sick patients, of clinically assessing blood gases.

Yet another advance has been a special type of cardiac catheter – a long, thin tube about 1/16 inch wide with a tiny, inflatable balloon on one end. The catheter is trademarked "Swan-Ganz" (Edwards Laboratories, Inc., Santa Ana, California) after H.J.C. Swan and William Ganz, two Los Angeles cardiologists. Drs. Swan and Ganz (along with four others) published their now classic article in a 1970 issue of the NEW ENGLAND JOURNAL OF MEDICINE, "Catheterization of the Heart in Many with use of a Flow-Directed Balloon-Tipped Catheter." Since then "Swan-Ganz" catheterization has taken ICU's by storm. ICU staff speak of inserting a "Swan-Ganz," "Swanning" the patient or of the patient being "swanned." No other medical proprietary name has become so universalized in recent memory. (In fact in the early 1990s there is something of a backlash; some physicians feel the catheters are used too freely and when less invasive diagnostic methods would be just as good.)

Most patients who develop ARDS (or one of several other critical heart-lung problems) will sooner or later have a Swan-Ganz catheter placed in their heart. The single major advantage over any previously used catheter is its ability to be accurately placed from the bedside. The patient does not have to be removed to a catheterization lab and, furthermore, bedside

measurements can now be obtained on a continuous basis.

(The Swan-Ganz catheter modifies an old technique. Credit for the first cardiac catheterization is given to the German physician Werner Forssmann, whose story is now legend. In 1929, as a recently-graduated doctor working at Augusta-Viktoria Hospital in Eberswalde, near Berlin, Dr. Forssmann conceived the idea of threading a long, thin catheter through an arm vein and into one of the heart's right-sided chambers. His superior refused permission for such a daring study, so Dr. Forssmann clandestinely did it anyway – using himself as subject! He confirmed the catheter's placement with a chest x-ray and published the results in *Klinische Wochenschrift* later that year. The medical possibilities lay dormant until the 1940s when Dickinson W. Richards, Jr., and Andre Cournand, working at Bellevue Hospital, began a systematic study of heart catheterization. Their work revolutionized cardiac diagnosis and paved the way for open heart surgery. In 1956 Drs. Forssmann, Richards and Cournand shared the Nobel Prize for Physiology or Medicine.)

One of many measurements taken via the Swan-Ganz catheter can tell if a patient has heart failure and serve as a guide to rational fluid management. Patients intubated and artificially ventilated can easily become dehydrated or over-hydrated – normal mechanisms for regulating thirst obviously do not operate. Without these measurements it is often impossible to know how much intravenous fluid to give; the physical examination, chest x-ray and routine lab studies are totally unreliable for this purpose.

Despite these and other advances, and even though we surely manage the syndrome more rationally, have more measurements and understand the physiology much better than a generation ago, the mortality rate in ARDS is still very high -- but improving. From 1967, when it was first described, through the 1980s, mortality hovered between 50 and 60%. Now, in the 21st century, it is less than 50%, closer to 40-45%. A significant improvement to be sure, but not comforting to the patients (and their families) who still succumb to this most complicated of illnesses. But survivors of ARDS, now in the majority, can thank their lives to modern intensive care.

The key to treatment is supporting the patient long enough for the lungs to heal. Once lungs fail in their capacity to deliver oxygen either they must recover in a short time or the patient dies. Artificial ventilation for lung failure is measured in days or weeks, not months or years. There is no long-term “dialysis” as exists for kidney failure. If the patient is not destined to recover lung function, no amount of support can keep him alive indefinitely.

Patients who don't recover usually succumb to sepsis, cardiac arrhythmia, internal bleeding or some other catastrophe. One of the saddest spectacles in medicine is to see a patient die with progressive respiratory failure despite the panoply of state-of-the-art, intensive-care technology. Amazingly, those who recover from ARDS, which is surely one of the most serious insults to afflict any organ, end up with normal or nearly normal lung

function. ARDS seems to be an all-or-nothing phenomenon.

* * *

On the ventilator Mr. Woodbury's PO_2 went up to 84 millimeters of mercury. This was adequate but far from normal, since he was inhaling 80% oxygen – almost four times the normal concentration. An expected PO_2 under these conditions is over 400. He was also receiving PEEP at 10 centimeters of water pressure, a moderate amount.

We gave him two grams of an intravenous corticosteroid, a powerful anti-inflammatory hormone often given empirically for ARDS in the early 1980s (today the drug is used much more sparingly in ARDS patients). Through a neck vein we inserted a Swan-Ganz catheter and threaded it into his heart. Unfortunately the catheter did not work at first, or rather the measurements did not make sense, as sometimes happens.

A chest x-ray revealed the problem. The catheter tip was coiled inside Mr. Woodbury's chest. After pulling back and reinserting the catheter we were able to get the measurements needed to guide fluid therapy.

This was the scene about 7 p.m., some nine hours after Mr. Woodbury's admission. A young, previously healthy man lay semi-conscious in bed, sedated with Valium. In addition to the endotracheal tube, now firmly taped to his face so as not to slip out, four other tubes violated his body: the Swan-Ganz catheter, through a neck vein; an intravenous catheter, through an arm vein; a special arterial catheter, previously inserted into the radial artery in his right wrist and used for drawing blood gases and monitoring blood pressure; and a soft rubber bladder tube, earlier placed through his penis so that urine output could be measured.

Clear plastic tubing connected the several bags of intravenous fluid to Mr. Woodbury's body; in the aggregate all the tubes looked like vines of some science-fiction forest. Interspersed between the vines were several pieces of electronic monitoring equipment: one to measure heart rate and rhythm, another to display the Swan-Ganz readings and a third to show his blood pressure. Just to the right of his head was the ventilator with its reassuring "whoosh" of air being pumped into his lungs, a sound repeated 14 times every minute.

(At a glance, scenes like that surrounding Mr. Woodbury may appear unreal, certainly not the picture of human care. Doctors and nurses sometimes have to remind themselves that ICU patients like Mr. Woodbury are in fact human, possessing normal capacity to live and love. When we forget this we are dealing not with patients as much as "heart-lung preparations," and the outcome is no more important than an interesting experiment. Intensive care can certainly give the appearance of an exercise in gross physiology. In truth, like some technologic fail-safe mechanism, internal reminders of the patient's humanity constantly arise and help guide

us.)

After discussing the next twelve hours' care with the nurses and resident staff I left for the evening. Mr. Woodbury was one of five intensive care patients in our ICU at the time, yet by far the sickest. No major changes in therapy were planned and we hoped for an uneventful night.

The next morning, January 14, Mr. Woodbury was not much better. During ICU rounds we reviewed the accumulated data: several chest x-rays, many blood test results, microbiology reports of his sputum, vital sign sheets. We double-checked the medication records and nursing reports. He was receiving the drugs on time and in the correct amount. Throughout the night he had been suctioned frequently and turned in his bed as recommended (lying in one place for prolonged periods is bad for any patient, especially those with ARDS). There was no doubt in anyone's mind – Mr. Woodbury was receiving superb nursing and doctor care, yet he was not improving.

The information so far pointed to an infectious pneumonia as the initial event. Was he on the right antibiotics? The lack of definitive culture reports (still too early for many organisms to grow) and absence of bacteria in his sputum suggested a virus or one of the difficult-to-diagnose bacteria. Could he instead have an unusual fungal or parasitic infection, also difficult to uncover and requiring altogether different antibiotics? And if so, how did he get it? Mr. Woodbury had no history of a compromised immune system, the common setting for "opportunistic infections" (so-called because ordinary fungi and parasites take the opportunity to invade a weakened host). Unusual infections can also occur in bird-handlers, pigeon-breeders and farmers, occupations remote from anything in his experience.

We decided to ask for help and called Dr. Dumont of the Infectious Disease Service. Since Dr. Dumont also ran the microbiology lab he already knew about Mr. Woodbury, at least about all his negative lab results. He sent his clinical fellow to the ICU and an hour later Dr. Dumont himself appeared, fully armed with all the data and his tentative conclusions. He didn't waste any time.

"You've got to treat him for Legionnaire's and pneumocystis." We had continued the erythromycin because of possible Legionnaire's disease and had considered pneumocystis, but thought it a highly unlikely infection in Mr. Woodbury. Pneumocystis is a protozoan that occasionally invades kidney-transplant recipients and immunologically-compromised infants, but rarely healthy adults. Until Mr. Woodbury became suddenly ill, he was a healthy adult.

"I don't think it's pneumocystis but we can't be sure," he continued. "Let's stop the oxacillin and add Bactrim. If he doesn't respond in 48 hours we'll consider an open lung biopsy. Meantime, up his steroids to four grams a day and send a serum sample to the lab for fungal titers."

Bactrim – a trade name for trimethoprim-sulfamethoxazole – is the one

of two major drugs for *pneumocystis carinii* pneumonia (the same pneumonia common in AIDS patients). Bactrim is effective and at the same time relatively free of major side effects. Since there was no reason not to follow his recommendations we ordered the Bactrim and increased the steroid dose.

As for open-lung biopsy, this is major surgery and used for diagnosis only as a last resort. There was also no assurance that a piece of Mr. Woodbury's lung, under the microscope, would in fact yield an answer. He was not ready for an open-lung biopsy.

In the afternoon I met with Mrs. Woodbury. She came without the children, mainly because ICU policy does not permit children to visit. I told her about Dr. Dumont and our suppositions. She was not discouraged but also not encouraged. It was just too early to know which way her husband was headed.

Dr. Levinson also came by and we discussed the case. He reassured me that nothing important was missed in Mr. Woodbury's past history and that whatever precipitated this crisis was acute and probably infectious. (Although private family physicians frequently will follow their patients in the ICU, it is not feasible to manage any critically ill patient from an outside office. It's no reflection on primary-care physicians, in this case Dr. Levinson, to have their ICU patients under the care of full time, hospital-based doctors. It is simply the best arrangement for the patient, a fact most office-based physicians well appreciate.)

The rest of the day Mr. Woodbury maintained a PO₂ in the 60s on 10 centimeters of PEEP and 60% oxygen. On the evening of January 14, his second day in the hospital, his PO₂ suddenly dropped to 35; a portable chest x-ray showed that his endotracheal tube had slipped into his right lung, effectively bypassing his left lung which was now collapsed. The tube was pulled back, restoring breathing to both lungs and pushing the PO₂ back up to 59 – still a low level but not life-threatening. A check of his electrocardiogram, urine output and blood pressure uncovered no damage from the transient hypoxia.

On January 15 we received the preliminary culture, microbiology and toxicology reports from specimens taken on admission. (On the outside chance some toxic chemical was the culprit a complete "toxic screen" of his urine and blood had been ordered the day of admission.) Everything so far was negative: there was no obvious or easily-diagnosed bacterial infection or toxin. This did not exclude the possibility of Legionnaire's, *mycoplasma* or a viral infection – all organisms which usually take weeks to diagnose because they require a convalescent blood specimen. (Antibody levels in the convalescent specimen are compared with levels when the patient is acutely ill; a diagnosis is made when antibody to the infecting organism increases four-fold in the interval.)

So forty-eight hours after admission we had no way of knowing what

Mr. Woodbury had or if our treatment was effective. Whether or not he would improve seemed as likely to depend on the natural course of his illness as on any treatment. On Dr. Dumont's advice we continued the erythromycin and Bactrim. Throughout January 15 his temperature hovered between 101 and 102 degrees.

There was still no improvement on January 16. His PO_2 ranged between 50 and 60, on 60% oxygen and 10 centimeters of PEEP; the chest x-ray continued to display a "whiteout" in both lungs; and we continued to use small doses of Valium to sedate him and allow the machine to keep him alive. The steroids had not only caused his body to become puffy, a predictable side effect, but had also produced diabetes, a not uncommon result when massive doses are used; his blood sugar went to over 400 milligrams percent (normal is less than 100) and required insulin injections to control.

Now the picture was bleak, not because he was worse but because he was not improving. Patients who don't improve invariably die; ARDS is not a chronic condition anyone can live with. In desperation we began to consider an open-lung biopsy. What would a lung biopsy offer? Not much, unless he had some weird infection we had otherwise missed. What were the risks? General anesthesia and major – albeit technically not difficult – surgery in a critically ill patient. Perhaps an operative mortality rate of one or two percent. We discussed the procedure with his wife, with Dr. Dumont and, tentatively, with the thoracic surgeon. Somewhat reluctantly – since doubted it would be revelatory – we scheduled a lung biopsy for January 18.

He showed the first sign of improvement on January 17. You wouldn't know it unless you had been following his blood gases. He certainly looked no different and his x-ray still showed the diffuse haze in both lungs. But his PO_2 was now up to 95 on the same concentration of oxygen. This indicated some microscopic clearing of his lungs, not yet visible on the x-ray. In retrospect this was a dramatic turnaround.

We lowered his oxygen concentration a little and still his PO_2 held. On 50% oxygen, four hours later, his PO_2 was 92. We left things there. Something was working, probably the natural healing process we had been hoping for. We had no way of knowing if the steroids or antibiotics or PEEP or all three (or none) had helped turn his course. As often happens in ARDS, there was no indication of why he was suddenly improving. We canceled the lung biopsy.

On January 18, breathing 40% oxygen, his PO_2 was 123. The pattern was now one of definite and sustained improvement. Except for the side effects of the steroids and the discomfort of the various tubes, Mr. Woodbury was doing well. We had stopped the sedatives and he was alert. His x-ray also began to show some clearing, the white areas melting away to reveal normal or clear lung fields. We removed the Swan-Ganz and arterial catheters and his urine tube.

As suddenly as he had deteriorated, he got better. Miraculously, we were able to disconnect the ventilator on the afternoon of January 18. The endotracheal tube was left in place another two hours, just in case he became worse, and he received humidified oxygen through the tube. There was no problem and he was able to breathe on his own, through the tube.

About 4 p.m. that afternoon the endotracheal tube was pulled out of his throat.

Followup

Mr. Woodbury was discharged from the ICU on January 20 and from the hospital on January 24. He returned to work February 15. I saw him as an outpatient the following week, at which time breathing tests, including an arterial blood gas, were near normal. Convalescent antibody titers were drawn. At a followup visit in June he was still doing well. Except for some slight decrease in exercise tolerance he has suffered no noticeable aftereffects.

He doesn't remember much of his ICU experience. He does remember going to the hospital and can recall the physical setting in the ICU, but most details, even from a patient's point of view, remain a blur.

We never made a specific diagnosis. All the antibody titers were non-diagnostic. In retrospect, considering all the negative laboratory results, a viral infection seems most likely, both for his early symptoms and the subsequent picture of ARDS. However, so many strains of virus can cause pneumonia that when only one person is infected (as opposed to an epidemic, like influenza), the responsible virus usually goes undetected.

Most viral illnesses are self-limiting and this seemed to fit Mr. Woodbury's course, except that he developed ARDS and became critically ill. With the aid of intensive-care support — particularly artificial ventilation and round-the-clock care by nurses and doctors — he proceeded to get better on his own. Without this support he would surely have died.

-- END --

9. Too Much Sugar, Too Little History

On morning rounds Peter Mance, one of the interns in MICU, presented a 30-year-old woman admitted the night before with diabetic coma and ketoacidosis. The patient had developed gastro-enteritis two days earlier and stopped taking her daily insulin injections.

Dr. Mance had stayed up with his dehydrated, acidotic patient most of the night, balancing her blood glucose and acid levels with the proper amount of insulin and fluids. Before the insulin era she would probably not have survived hospitalization. Now modern medicine – and a conscientious intern – had changed her condition from critical to stable in less than 12 hours.

In diabetes the body's normal supply of insulin is either absent or deficient. Without insulin, a hormone made by the pancreas, glucose cannot enter the cells and be used as energy. Without insulin glucose accumulates 'outside' the cells, in the blood. Depending on the severity of diabetes the blood glucose level may range from normal to over 10 times normal.

Ketoacidosis, the most extreme state of uncontrolled diabetes, results from a sudden and severe lack of insulin; glucose builds up rapidly in the blood, to dangerously high levels. To forestall starvation the cells turn to abundant fat as an alternative fuel. Metabolism of fat, less efficient than that of glucose, causes a buildup of harmful acid products called ketoacids – hence the term diabetic ketoacidosis, or DKA. The hallmark of DKA is an excess of acids and glucose in the blood.

DKA patients are very dehydrated because the extra blood glucose spills into the urine along with a large amount of the body's water. Glucose, a type of sugar, is not normally present in urine. 'Tasting the urine' was an early way to diagnose diabetes (*not* a test of modern medicine). The full name of the disease is from an early description in Latin that referred to the urine: diabetes (to pass through) mellitus (honeyed).

* * *

Dr. Mance's patient was a 'textbook' case of DKA. Not every MICU patient has to have triple organ failure or present ungodly ethical dilemmas. This patient, at least, presented a straight-forward problem amenable to therapy. She also provided an opportunity to teach some medical history.

We went in to see her, a pleasant Puerto Rican woman named Carlita Gomez. Mrs. Gomez looked and acted normal, which in itself was remarkable. From just her appearance you would not know how sick she was a day earlier.

“How do you feel?” I asked.

“Much better, doctor.”

“What happened that you had to come to the hospital? Did you forget to take your insulin?”

“No. Two days ago I began vomiting and was sick to my stomach. I didn’t eat so I didn’t take my insulin yesterday or the day before.”

“Why didn’t you come to the hospital right away, when this all started?”

“I thought it would get better, like a stomach virus or something.

Yesterday I got much worse and came to the emergency room.”

“What made you finally decide to come yesterday?”

“I felt very dizzy and sick. My boyfriend said I looked very bad and he brought me.”

“Peter,” I said, “can you describe what she looked like on admission?”

“I saw her about twenty minutes after she arrived to the emergency room. She was very somnolent. I had to arouse her to answer questions, but she was lethargic and couldn’t give me any information except her name.”

“Was she short of breath?”

“Well, she didn’t look like an acute asthmatic or anything, but her breathing was rapid and deep.”

Rapid and deep, the classic breathing pattern in DKA, is the body’s attempt to balance the excess ketoacids by hyperventilation or ‘blowing them off.’ The other classic symptom, excessive urination, causes great thirst and water ingestion.

“Mrs. Gomez, were you very thirsty before you came to the hospital?”

“Yes, I must have drank twenty glasses of water yesterday and the day before. I couldn’t seem to get enough water.”

“I bet you went to the bathroom a lot, too.”

“Yes.”

“Has this ever happened to you before, that you had to come to the hospital for your diabetes?”

“I once came to the emergency room with the flu and they said my blood sugar was high, but I didn’t have to stay in the hospital.”

“How long have you had diabetes?”

“Since I was twenty-one.”

“Have you taken insulin that long, for ten years?”

“Yes.”

“Does anybody else in your family have diabetes?”

“My mother, but she just uses [anti-diabetic] pills. She doesn’t need insulin.”

“Who’s at home with you?”

“My two kids. I’m divorced.”

“How old are they?”

“I have two, a boy seven and a girl ten.”

“Did you have any problems with the pregnancies?”

“No. They were both heavy babies, though. The doctors said that was because of my diabetes.”

“Who’s taking care of the children while you’re in the hospital?”

“My mother.”

“Do your kids have diabetes?”

“No, not as far as I know. They seem to be fine.”

Further history revealed that Mrs. Gomez was followed in our hospital’s diabetes clinic, that she was compliant with her insulin therapy and that she did not suffer from the potential ravages of the disease, such as kidney failure and vascular disease. She supported herself with income from the state’s Aid to Dependent Children program and some help from her mother.

We attributed her exacerbation to non-specific gastroenteritis, an inflammation of the stomach and intestines probably caused by a virus. She stopped taking insulin because she couldn’t eat, but this only accelerated the vicious cycle leading to high blood sugar and DKA.

I reviewed Dr. Mance’s detailed charting of insulin dose and blood test results. In the emergency room Mrs. Gomez’s blood glucose was 948 milligrams%, over nine times the normal value of 80 to 100 milligrams%. Her bicarbonate level was only six, about a quarter normal and indicative of severe acidosis (the lower the bicarbonate, the more acid is in the blood). Dr. Mance’s meticulous chart showed that both glucose and bicarbonate levels were returning toward normal, a direct result of insulin and fluid therapy.

“Let’s see. She’s now receiving two units of insulin an hour, right?”

“Right,” said Dr. Mance.

“And her blood glucose is down to two hundred forty and bicarbonate up to nineteen?”

“Yes, those are the latest values,” he affirmed.

“Well, it looks like she’s on her way to complete recovery. You’ve done a good job, Peter.” Turning to Mrs. Gomez, I asked, “How’s your stomach? Do you think you can eat now?”

“I feel much better,” she said. “I think I’d like to eat something.”

“Peter, let’s start her on a soft diet and advance to regular food if she tolerates that.”

I thanked Mrs. Gomez and we stepped outside her room to continue the discussion.

“We should be able to switch her to subcutaneous insulin later today [the method of insulin delivery she used at home]. Let’s keep her in MICU until she’s off the intravenous insulin.”

“OK.”

I turned to address the medical student, Sarah Miles, a bright and energetic woman about five years younger than Mrs. Gomez.

“A fairly typical case of DKA, don’t you think, Sarah? Have you had a chance to read about her problem?”

“Yes. I read about it last night.”

“Good. You worked her up with Dr. Mance, right?”

“Yes.” Sarah was on call the night before and had stayed up to help manage Mrs. Gomez.

“What did you read?”

“I read that DKA patients typically present with hyperventilation and stupor, and that reversal is fairly rapid with treatment, which always consists of fluids and insulin.”

“That’s right. What did doctors do with these patients before insulin was available? Anybody know?”

There was no answer.

“Starvation,” I said. “Doctors prescribed starvation diets to their diabetic patients. Unfortunately, without insulin, almost everyone with ketoacidosis died. How long has insulin been available? Did your book mention that bit of history, Sarah?”

“No, I didn’t see that,” she replied.

“Well, how old are you, if you don’t mind my asking?”

“Twenty-four.”

“Was insulin around when you were born, in nineteen sixty-five?”

“I think so,” she said, perhaps wondering what the question had to do with our patient.

“Well, was insulin around in nineteen *fifty-five*?”

Bill Sedgwick, the other intern on rounds, spoke up. “I think that’s when insulin was introduced. Nineteen fifty-five.” He spoke with assurance, as if he really knew the answer.

“Bill,” I said in a congratulatory tone, “do you play Trivial Pursuit?”

“Sometimes,” he said.

“Anybody disagree with Bill?” I asked. No one answered. Everyone seemed to agree 1955 was when insulin became available. If so, they were only off by three decades.

Ignorance about this seminal event in medicine wasn’t their fault, if it could even be called a fault. ‘History of medicine’ as a subject is almost never taught in American medical schools. Even the historical aspects of everyday diagnosis and therapy are usually omitted. One principal reason is that the teachers are themselves often ignorant or uninterested, and that’s too bad.

Doctors interested in history know that it helps provide perspective and humility in daily practice. It teaches us that today’s “correct” therapy may be viewed as wrong-headed nonsense by future generations, much as we view primitive therapies of the 19th century and earlier.

Knowledge of historical events can also be of practical importance. For example, anti-tuberculous drug therapy was only introduced in the late 1940s. Anyone who contracted TB before then could not possibly have received effective drug therapy, yet on more than one occasion I've been told on rounds, by some well-meaning medical resident, that a patient received "TB drugs" in the 1930's or early 1940's.

Another time a house officer related a patient's treatment for Legionnaire's disease to a year before the disease was first described (1976)! Even local history is important. A patient could not have had a CAT scan before the machine was available (in our hospital, 1978), yet I have been presented with the verbal reports of such phantom scans.

Like any history, of course, medicine's is far more than dates. The pioneering work of Banting, Best, Macleod and Collip in insulin, of Koch in tuberculosis, of Pasteur in rabies, and of Enders and Salk in polio vaccine, cannot be taught by dates alone. The stories behind major medical breakthroughs are invariably full of scientific excitement and human drama (and are well-told in numerous books, such as Paul DeKruif's *Microbe Hunters*, Michael Bliss's *The Discovery of Insulin* and James Watson's *The Double Helix*). Still, it would be helpful if young physicians knew a few important dates.

"Nineteen twenty-two," I said. "Insulin was introduced into clinical medicine in nineteen twenty-two."

"Oh well," said Bill Sedgwick, suggesting by his tone that it really didn't matter whether it was 1922 or 1955.

"The first patient to receive insulin was a thirteen-year-old diabetic boy named Leonard Thompson. In what city?" I asked. If they didn't know the date they also would not know the location of this medical milestone, but I was having fun and they didn't seem to mind.

"Boston," guessed Dr. Sedgwick.

"No."

"New York," said Dr. Miles.

"London?" said one of the nurses, in a final desperate stab.

"No. Everyone give up?"

All did.

"Toronto. The Toronto General Hospital. January eleventh, nineteen twenty-two. And who received a Nobel Prize for the discovery of insulin?"

"Banting," said Dr. Sedgwick, who was still out to win the trivia prize.

"You're half right," I said. "Banting who?"

"That's his last name. I don't remember his first name."

"Did anybody else get the Nobel Prize for work on insulin?"

No answer.

"How about Best? Banting and Best, does that ring a bell?" The two names, of course, are forever linked with the discovery of insulin. Even many grade school students have heard of Banting and Best.

“That’s right,” said Dr. Sedgwick. “Banting and Best. I remember now.”

“Yes,” said Dr. Mance. “I remember too. Banting and Best, but I don’t remember their first names or anything about them.”

“Actually, they are names you probably heard before you entered medical school. Like Salk and Sabin. Probably because of the Nobel Prize, I bet. Salk and Sabin got the Nobel Prize for the polio vaccine, and Banting and Best got it for insulin. Right?”

There was general murmur of agreement.

“Does anyone disagree?” If my questions seemed obnoxious no one complained. In any case I felt compelled to continue, especially since no one had disagreed with my purposely erroneous statement about Salk, Sabin, and Best.

“Well, for starters, Sabin and Salk did *not* win the Nobel Prize.”

“No? Sure they did,” said Dr. Sedgwick. “That can’t be.”

“Sure they *didn’t*. They developed and introduced the polio vaccines but never got a Nobel Prize for their work. The Prize was given for earlier work on culturing the polio virus in monkey kidney cells, work that was published in 1949. So who got the Prize for work on polio?”

Now they were baffled. I was asking a bit of trivia they must have missed in school. No one answered so I continued.

“The Nobel Prize was awarded to three Americans, Drs. Enders, Robbins, and Weller, in nineteen fifty-four, before the polio vaccine was even released. They were the ones who developed a method to culture the polio virus. Without their work there would have been no vaccine.”

“Really?”

“Really. And it was in Boston, too.”

“Are you sure Salk and Sabin never got the Nobel Prize?” asked Dr. Sedgwick, still unbelieving.

“I’m sure. They got just about every other prize, but not the one from Stockholm.”

“What about Banting and Best? Didn’t they get it for discovering insulin?”

“Yes and no. That’s an interesting story,” I said. “Anybody want to hear it?”

I wasn’t sure anyone did, but I wanted to tell it. It is one of the more fascinating stories of medical discovery.

“Yes,” said one of the nurses. “Tell us.”

“Right,” agreed Dr. Mance.

Sensing a trace of sarcasm, I suddenly felt a need to defend the subject of medical history. Looking toward the patient I said, “There’s more to a case like this than just glucose and bicarbonate levels. Sometimes we take these modern miracles too much for granted. Since you insist, I’ll tell you about the discovery of insulin.” And I did.

* * *

Before 1922 young diabetics were treated with low-carbohydrate, starvation diets. Even so, patients with DKA invariably died in coma. William Osler, in his 1892 *The Principles and Practice of Medicine*, a standard textbook of the era, wrote:

In children the disease [diabetes] is rapidly progressive, and may prove fatal in a few days...as a general rule, the older the patient at the time of onset the slower the course...In true diabetes instances of cure are rare...Our injunctions today are those of Sydenham [an earlier physician]: 'Let the patient eat food of easy digestion, such as veal, mutton, and the like, and abstain from all sorts of fruit and garden stuff.' The carbohydrates in the food should be reduced to a minimum.

Reflecting medical practice of the day, Osler's text devotes almost a full page to specific dietary recommendations, listing those items which the diabetic may take and those which were prohibited. Among the latter was "ordinary bread of all sorts."

Osler's comment on "Medicinal treatment" of diabetes was an understatement:

This is most unsatisfactory, and no one drug appears to have a directly curative influence.

In fact, no drug was even indirectly curative. On the subject of diabetic coma, Osler wrote:

The *coma* is an almost hopeless complication.

Frederick Banting was a 30-year-old surgeon from London, Ontario who had the idea of isolating the specific secretion of the pancreas that lowers glucose in the blood. By the end of World War I scientists knew some substance in the pancreas acted to lower glucose, but no one had been able to isolate it or use pancreatic extract successfully in diabetic patients. Banting moved to Toronto in 1921 to work in the lab of Dr. John Macleod, a Scottish physiologist who was an expert in the field of carbohydrate metabolism.

Banting was aided in his work by Charles Best, a 22-year-old medical student. The two of them succeeded in preparing an extract of pancreas that did lower blood sugar in dogs. This extract, of course, contained insulin. Under their direction the first human trial of insulin took place in January 1922. (History records earlier animal trials of crude pancreatic extract, most notably by a Georg Ludwig Zuelzer in Berlin, but nothing came of that

research.)

Leonard Thompson weighed only 64 pounds when he was admitted to Toronto General in December, 1921. His diagnosis: diabetic ketoacidosis. He was initially given a diet consisting of only vegetables [see “A Case of Diabetes Mellitus,” *New England Journal of Medicine*, February 11, 1922]. Before he received insulin Thompson’s blood sugar ranged from 350 to 560 milli-grams%.

Thompson was still in the hospital when Banting and Best were ready with their extract in January 1922, so he was the first human to receive it. According to the hospital record, Thompson’s blood sugar fell from 470 to 320 milligrams% six hours after the injection.

It took Banting and Best two weeks to get more of the extract and Thompson didn’t receive his next injection until January 23. His blood sugar responded by falling each time. Thompson continued to receive insulin and was sent home on May 15, 1922, weighing 67 pounds.

Almost from the very beginning the discovery of insulin sparked controversy. Up until early 1922 Macleod had played no direct role in the discovery. Although he had acted as advisor on a number of the experiments, and Banting and Best used his lab, Macleod was actually out of the country in the summer of 1921 when most of the important dog experiments were accomplished.

The earliest scientific reports listed only Banting and Best as authors. In fact, Banting later wrote that he was discouraged in his work by Macleod, and that Macleod told him that “negative results would be of great physiological value.” When it became clear that an important discovery had been made Macleod got involved and orchestrated the production of insulin and further research into its use.

At the time insulin was considered a cure for diabetes so the discovery was quite a sensation. And no wonder. Patients near death were miraculously resurrected after only a few injections of the vital hormone. As expected, the discovery led to the Nobel Prize for Physiology or Medicine. It was awarded in the fall of 1923 to Banting and . . . Macleod!

Upon hearing of the award Banting was furious. In his view, Macleod had done none of the original work but only impeded his own brilliant research. Banting thought Charles Best should have shared the Nobel Prize.

Since Best was only a student at the time, and did not present any of the research at meetings, as did Banting and Macleod, his role in the discovery was not prominently featured. The Nobel committee was aware of Best (he was co-author on the original papers) but was more impressed by Macleod’s senior status and the fact that he presented much of the research at scientific gatherings, one of which was attended by a Nobel committeeman. Also, by intention the Nobel committee wanted to honor only two recipients (it was not until 1945 that three people would share a Nobel Prize in Physiology or Medicine). To the Nobel committee in Sweden Banting and Macleod

seemed the logical choices.

Banting tried to correct the injustice by publicly giving half of his award to Best (the total monetary award in 1922 was \$24,000). As it turns out another scientist was also overlooked by the Nobel committee, a Toronto biochemist named J.D. Collip. Collip was responsible for purifying insulin far beyond what Banting and Best had achieved with their crude extract. In fact Collip had purified all the injections given the Thompson boy except the first one. After being apprised of Banting's gesture Macleod shared half of *his* prize money with Collip.

For the next two decades there was bitter (and private) feuding among all four men over priorities and who did what. Banting continued to believe that Macleod had hogged the limelight and deserved no credit for the discovery of insulin. Macleod thought Banting was an ungrateful young doctor who didn't appreciate how much he, the senior professor, had contributed to the discovery. Best, of course, was truly slighted by the Nobel Committee and many contemporaries felt he should have been named a recipient of the Prize.

At least Best's contribution has been fully recognized by history, not to mention the University of Toronto. He outlived everyone in the story, dying in 1978 after a distinguished career as a professor of physiology. Collip also achieved long-lasting recognition for his pioneering work on purifying insulin. Macleod died in 1935, in Scotland. As for Banting, he died in a plane crash in Newfoundland in 1941.

* * *

"Well, that's enough about the discovery of insulin," I said. "Anybody have any questions?"

"What happened to the boy who got the insulin?" asked Dr. Sedgwick.

"Oh, glad you asked that. He lived another thirteen years, dying in nineteen-thirty-five at age twenty-seven, of severe bronchopneumonia. They did an autopsy and found the ravages of diabetes, including a shrunken pancreas."

"Any more questions?" There were none.

"OK," I said, "let's go see the next patient."

-- END --

10. Crusade

Harlan Tembo has chronic bronchitis, a result of smoking cigarettes since he was 15. At age 57 Mr. Tembo was brought to Mt. Sinai Medical Center's emergency room one Monday in respiratory distress, blue, semi-conscious, and about to die. His diagnosis: exacerbation of bronchitis and acute respiratory failure. He was intubated, connected to a ventilator and sent up to MICU.

His hospital care reflected a quiet revolution in medicine. Before ventilators were available for patients like Mr. Tembo – that is, before the 1960's – he would probably have died. Now patients with acute and otherwise fatal lung failure are often saved by just a few days of 'ventilator support.' Even chronic patients with end-stage lung disease can benefit if their respiratory failure is acute and reversible. One of Mt. Sinai's chronic lung disease patients has been intubated at least nine times for acute respiratory failure. For Mr. Tembo, this was his first time intubated.

As late as 1967 there was debate about the utility of artificial ventilation in respiratory failure. Back then ventilators were much scarcer; now they are commonplace in every hospital. The only real debate over ventilator use today involves ethical issues, not medical ones.

After four days of artificial ventilation in MICU, Mr. Tembo could breathe on his own, unassisted by machine. It was time to begin my no-smoking crusade.

Every physician should mount a crusade when patients are most vulnerable, which is usually when they are ill and especially when hospitalized. It is not wrong to choose this time; it is wrong not to.

I am amazed how often physicians treat serious illness related to alcohol or tobacco, and do little (or nothing) to educate the patient. Why? Do doctors think patients automatically make the connection between habit and illness? In fact patients all too often fail to make any connection unless directly and specifically told what it is.

Cynics may say I am wasting my time but evidence from various studies suggests otherwise. There is a positive influence on individual patients when *their* doctor gives advice in a forceful and direct manner. This is especially true about smoking.

The advice has to be more than mouthing a few words. The doctor (or nurse, for that matter) has to demonstrate genuine concern for the patient. Sometimes simple repetition will do it. Occasionally a threat works, as in: "You'll need to find another doctor if you don't quit smoking."

But how do you communicate to someone you may never see again, or someone with only a grade school education, or someone who is a borderline psychotic? Sometimes reason, logic, and common sense are not enough.

The message must always be personal and tailored to the patient's level of understanding. The important thing is to try.

* * *

"Let's extubate him," I said.

"Do you think he's ready?" asked one of the interns.

"Sure. He's awake, he's writing notes, he wants the tube out. Right Mr. Tembo?" He gave a vigorous nod. The nurse went to get the few items needed for extubation.

You know you can never smoke again, Mr. Tembo. We found cigarette smoke in your blood and that's why you ended up in intensive care." He nodded again, as if agreeing just so I would be sure to take the tube out of his throat.

"Once we get this tube out, if you go back to smoking the tube will have to go right back in. Do you understand?" He shook his head, meaning he wasn't going to smoke again.

The nurse returned with scissors and an oxygen face mask. I cut the tape securing the tube to his face, deflated the cuff inside his throat and pulled the tube out. Extubation is always a pleasure for patients, providing they are not gasping for air in the first place (as was Mr. Tembo on admission). If you can imagine a foot long plastic tube stuck in your throat, making it impossible to speak or swallow or move your mouth, you can imagine the relief when it comes out.

"How do you feel, Mr. Tembo?"

"Better." His voice was hoarse but understandable.

"Do you know where you are?"

"In the hospital."

"When did you come in?"

"Last night."

"What day is it?"

"Tuesday?"

He had lost track of time, not uncommon in patients who receive artificial ventilation for more than a day.

"No. It's Thursday. You came in on Monday. You've been here for four days already. Do you know why?"

"No."

"Your lungs stopped working."

"Really? I guess I'm lucky to be here. Can I eat something?"

"Not right away. We'll let you have a little water, and then some soup." It's best not to give solid food right after a patient has just come off the ventilator.

"I'll be back later to talk to you about that cigarette smoke in your blood." Having finished our immediate task we went to see the rest of our

patients.

The next morning Mr. Tembo was like a new man. He had eaten his first regular meal and was well enough to leave MICU.

“Well,” I said. “You’re much better. You know you were quite sick a few days ago. A machine had to take over your breathing for four days.”

“That’s what they tell me.”

“Do you know why?”

“Bronchitis, I guess.”

“Well, that’s true. You almost *died*. You came this close to being completely dead.” I held up my forefinger and thumb an inch apart. “Do you know how you got that way?”

“No.” He really didn’t know or understand. But, sensing that the preacher was about to strike and remembering the conversation from the day before, he answered, “Cigarettes?”

“That’s right, Mr. Tembo. You almost died from cigarettes. Your blood was filled up with cigarette smoke!” (Actually, his blood showed an excess of carbon monoxide, one of the toxic components of cigarette smoke).

“Well, I’m not going to smoke again.”

I acted skeptical. “I’ve heard that before. Should we save this bed for you just in case?”

“Naw, you can give it to someone else. You won’t see me back.”

Time to stop badgering? Believe him? No. The physician has to make some *impression* on the patient, even to the point of looking silly or sounding obnoxious. Besides, I rationalized, I’m not only communicating with Mr. Tembo, I’m also teaching the housestaff. Teaching them what? That they must do more than offer perfunctory advice about smoking.

“Mr. Tembo, I’m afraid your next cigarette could be your last.” Then I delivered a non-sequitur: “Do you live near a funeral home?”

“Yes, why?”

“After you get home and decide to start smoking again, go to the funeral home before you light up. That way, if you die right away they won’t have to bring your body to the hospital.” (Actually this is not true. Even if you drop dead in the lobby of a morgue, the police will come and remove your body to the closest hospital to be pronounced. After this was pointed out to me I changed “funeral home” to “emergency room.”)

“Well I’m not going to smoke again, you can bet on that.”

“I hope not. But we’ll keep a bed open just in case.”

Mr. Tembo was discharged after a week in the hospital. He was admitted a year later for another exacerbation of chronic lung disease, but it was not as bad as the episode recounted above. He did not require intubation or care in MICU. And his blood carbon monoxide level was normal, so he had not resumed smoking.

“Why not?” I asked him.

“Well, Doctor Martin,” he said, “I remember what you told me last year. So I just gave it up. And I don’t miss it at all.”

* * *

I don’t know how many times I’ve taken cigarette packs from a patient’s bed stand (always with their permission) or used my funeral home (emergency room) plea to get the message across. Probably a hundred times over the years. If it worked only once it was worth it.

For Amanda Wiggins though, none of my usual messages got through. She has chronic lung disease and always complains of being “short winded.” That was her constant complaint during one hospitalization on the medical ward. (On that occasion she did not need artificial ventilation or care in MICU.) Neither fear of funeral homes nor emergency rooms nor ventilators nor lung cancer nor skin wrinkles made any dent in Mrs. Wiggins’ smoking addiction. She was incorrigible. She continued to smoke in her hospital room and in the ward lounge even as we treated her for smoking-related chronic lung disease. (The episode recounted herein occurred before our hospital became a smoke-free institution).

Psychiatrically Mrs. Wiggins was a “borderline” personality disorder – not psychotic, but with a tendency in that direction. Her anchor in life seemed to be the bible and fundamentalist religion. I saw this belief as a wedge to change her smoking behavior.

One day on the ward, as she complained about her shortness of breath, I auditioned my new message. I made sure her nurse was there; no one would probably believe me if Mrs. Wiggins really did quit smoking.

“Amanda, we can’t get you better if you continue to smoke.”

“I’ll quit,” she said, in a manner which conveyed just the opposite intention.

“You’ve got to quit.”

“I’ll quit. I want to get better.”

“You’re gonna die!”

“Don’t say that, Dr. Martin. If I quit will I get better?”

“How are you going to quit? You’ve promised me a hundred times and you always go back to smoking.”

“Well, I’ll quit now.”

“Can I have your cigarettes?” I knew her supply was endless; taking them would be like trying to cut off the flow of cocaine with a single arrest. Still, it would be a step in the right direction.

“Take ’em Dr. Martin,” she said confidently. I opened her drawer and took out two unopened packs of cigarettes.

“Can I have the others?”

“I don’t have any more. That’s all I have,” she said.

I knew this woman. She was not about to quit smoking so easily.

“Now you’ve got to swear you’ll quit smoking.”

“I’ll swear,” she said, with no emotion.

“Then swear.”

“I swear.” Still no emotion. I reached over and picked up her bible.

“Swear on this,” I commanded.

“Why do I have to swear on the bible?” Now her voice was rising. “I said I wouldn’t smoke. Don’t you believe me?”

I knew it. She had no intention of quitting. Unless I could get her to swear on the bible she would never take her promise seriously. “Mrs. Wiggins you’ve got to swear on the bible. Otherwise God won’t believe you’re sincere.”

She hesitated and her body began to shake. She looked at me, then the bible, then at me again.

“Dr. Martin,” she said indignantly, her voice trembling a little, “that’s the word of the Lord! You want me to swear on the Bible?”

“Swear!” I was transforming my demeanor into that of a medical evangelist. “SWEAR!”

“I can’t do that!”

“Then you don’t intend to quit. You lied to me.”

“But I will quit, Dr. Martin. I promise!”

“Then SWEAR ON THE BIBLE!”

Slowly, hesitatingly, she placed her right hand on the bible. I could sense the adrenalin flow. I was about to make my first convert.

“Repeat after me. I, Amanda Wiggins...”

She hesitated and looked at the nurse. Not wanting to break the spell with another voice, the nurse merely nodded her head, affirming my command. Then Mrs. Wiggins looked back at me. My eyes were fixed on hers. There was no way out. She repeated my preamble.

“I, Amanda Wiggins...”

“Do swear before God in Heaven...”

“Do swear before God in Heaven...”

“That I will never touch or smoke cigarettes again.”

“Oh, Dr. Martin!”

I repeated the command with raised voice. “THAT I WILL NEVER TOUCH OR SMOKE CIGARETTES AGAIN. SWEAR, MRS. WIGGINS!”

“That I will never touch or smoke cigarettes again,” she echoed.

“SO HELP ME GOD!” I bellowed.

“So help me God,” she responded. With the last word her whole body shook and she began crying. I checked her pulse and listened to her lungs. No acute problem. She was not having an asthma attack, just a religious experience. Had I reached her? I left Ms. Wiggins sobbing quietly on the bed. The nurse watching the scene promised to check her every half hour.

Feeling quite smug about my effort, I went to see other patients. I remember thinking: to get a patient to quit smoking you must learn to

communicate on their level, to search out that part of their psyche that listens to the doctor. Why aren't all physicians this creative with their advice?

A half hour later the nurse called me to come to the ward lounge. "You won't believe this," she said. The tone in her voice punctured my balloon. 'Oh yes I will,' I thought. There in the lounge sat Ms. Wiggins, smoking a cigarette. Relaxed and calm, she looked up at me with not the least hint of anxiety.

"What happened?" I asked, feigning a hurt incredulity. "You PROMISED me you would quit. You promised GOD! You swore on the Bible!"

"I just had to have a cigarette," she said with an innocent smile.

That was the first and last time I tried religion to break a patient's habit.

Comment

In 1964, when the first surgeon general's report on Smoking and Health was published, 40% of adult Americans smoked regularly. Today the figure is 25%, or about 50 million people. Of the millions who have quit smoking in the past generation, an estimated 90% did so 'cold turkey,' without the aid of any therapy or drugs or behavior modification. Although nicotine in cigarettes can be addicting, once the mental decision is made to quit it is not all that difficult.

Still, for some people like Mrs. Wiggins and the patient in the next story, quitting is out of the question.

-- END --

11. “Just give me a cigarette!”

Harold Switek has two major diseases – emphysema and paranoid schizophrenia. His lung disease comes from smoking two packs a day for 35 years. Cigarettes did not cause his psychosis, of course. Though “tobaccoism” is a form of addiction, and is now recognized as such by the medical profession, mental illness is not a known complication.

Smoking is distressingly common among hospitalized psychiatric patients. Smoking helps to allay their anxiety, and any attempt to withhold tobacco tends to make for a bad situation. It is almost impossible to get psychotic patients to quit smoking (schizophrenia is one type of psychosis) and psychiatrists are reluctant to badger them about the harmful effects of tobacco. As a result, although in-hospital psychiatric patients are forbidden to keep matches, smoking is not generally discouraged by the psychiatry staff. To allow smoking but not matches, many psychiatry wards install a permanent cigarette lighter at the nurse’s station.

I knew Mr. Switek’s case would be difficult the moment I heard from Dr. Janis, his psychiatrist.

“Larry? Hi. This is Nancy Janis. How are you? Good. I wonder if you could do me a favor.”

“Sure Nancy, what is it?”

“I have a 50-year-old man in Weathergill [Psychiatric Pavilion] who’s been there two weeks. He’s developed a breathing problem and I can’t keep him there any longer. They have no medical ward and I’m afraid he needs to be transferred for medical care. Would you be able to take him on MICU?”

“Sure. How bad is he? Does he need a ventilator?”

“I don’t think so. He has chronic emphysema and we have him on oral meds but he’s getting worse. We can’t give IV therapy at Weathergill.”

“What medication is he taking? Anything that might suppress his breathing?”

“He’s had schizophrenia for decades. Poor Harold. He’s been in and out of hospitals. I’ve only had him as a patient the past year. The only thing I can control him with is Thorazine. He’s up to 800 milligrams a day but I think that’s a safe dose. He might be able to go without it for a while, but he’ll eventually need it.”

“Oh,” I said. “Well, we’ll see what he’s like. That much Thorazine probably can suppress breathing in someone with severe lung disease. I may

have to stop it for a short time.”

“What’s happening is his emphysema is getting worse. I think he needs IV therapy.”

“No problem. When will he arrive?”

“I’ll call an ambulance now. He should be there within an hour. I’ll make sure they send a copy of his medical records with him.”

“What’s the situation with relatives? Is he married?”

“No. He lives with his 80-year-old mother. She’s all he has. I’ll call and let her know.”

“OK. We’ll call you back if we have questions about his psychiatric management.”

“Good. I’ll stop by tomorrow and see him anyway.”

* * *

Mr. Switek arrived on schedule. Since everything was prearranged he came right up to MICU, bypassing the emergency room. My first impression was that he didn’t look all that bad. My second impression was that he didn’t look all that crazy. I was wrong on both counts.

The MICU nurses put him in bed, took his vital signs, and began setting up an intravenous line. I went in with the intern, Dr. Solomon, to begin our examination. Wearing only shorts and a hospital gown open in the front, he appeared short and stocky, with large thighs and arms. His barrel-shaped chest was covered with hair and his abdomen protruded with about 25 excess pounds. The transfer record listed him at 185 pounds, 5 feet 7 inches.

His face was big and round, with a crew cut on top, a feature I’ve always found incongruous in a middle-aged-man. Mr. Switek did not appear particularly anxious or short of breath, but one can be fooled by patients at rest, especially those with chronic lung disease. I have seen patients comfortable in bed who could not walk across the room without gasping for air.

On closer observation his breathing problem was more apparent. Dusky skin and lips signified oxygen deficiency, all the more remarkable because he was receiving oxygen through a nasal cannula. Rapid respiratory rate (34 breaths a minute) and contracting neck muscles signified his dyspnea. His blood pressure was normal, heart rate increased, and temperature up slightly to 100 degrees.

Probably because of his psychiatric history I was also cognizant of some subtle features. His eyes were open wider than normal and he gazed through rather than at you. He did not make eye contact for more than a fraction of a second. Also, his speech was rapid, much faster than the typical patient with severe lung disease.

“Hello, I’m Dr. Martin and this is Dr. Solomon. How are you?”

“I want to call my mother. Is she here? What’s your name? What are

you going to do? Is my mother here? Where are my shoes? Why can't I have my shoes?"

"She's been called, Mr. Switek. She knows you're here. And you won't need your shoes while you're in MICU."

"She wants me home. I want to go home. Where are my shoes? Hey, can I have a cigarette? Where are my cigarettes?" With this last question he began turning his head from side to side and up to the ceiling, as if we were hiding his cigarettes close by, somewhere in the ICU cubicle.

"You can't go home now, Mr. Switek. You were sent here because of your breathing."

"Hey Doc, there's nothing wrong with my breathing. I just want a cigarette. Can I have a cigarette? Where are my cigarettes? I'll just take one."

"That's impossible, Mr. Switek. You're in the intensive care unit! There's oxygen all around you."

"I'll go outside. Can I have a cigarette?"

"Can I listen to your lungs?"

"OK."

Dr. Solomon and I went right to his lungs and heart, bypassing areas customarily examined first: no telling when he might decide not to cooperate. Facing each other behind Mr. Switek's hairy back, we each placed a stethoscope up and down his chest, listening to him breathe. Our eyes met as our ears heard the same thing: diffuse and severe wheezing, sometimes described as "tight breathing" because it signifies air squeezing through constricted passages.

"Better check his blood gases," I said, and Dr. Solomon left to get a syringe for the arterial blood sample. I came around the side of the bed to face Mr. Switek.

"Now can I have a cigarette?" he asked, as soon as he saw me.

"You can't smoke!" I gesticulated, flapping my hands up and down for effect. "You'll blow up if you smoke!" (Not true, but I thought this would get to him.)

"Naaah," he said, without missing a beat. "I won't blow up. You're just saying that. Please get me my cigarettes. Where are they?"

"First let's see what our tests show," I said.

"OK. But I can have a cigarette first?"

MICU could handle any type of lung problem. Could we handle a psychotic patient with a lung problem?

Thirty minutes later we had results of arterial blood gas analysis, chest x-ray, and electrocardiogram. Mr. Switek's oxygen level was dangerously low, only 48. The cardiogram showed a heart strain from the low oxygen, and his cardiac rhythm showed skipped beats and other ominous irregularities. Fortunately his chest x-ray was clear — no pneumonia.

Our diagnosis was acute bronchial infection on top of emphysema, with

impending respiratory failure. He was almost at the point of needing intubation and artificial ventilation. Perhaps with an increase in inhaled oxygen, plus antibiotics, steroids and other medication, he could get by without intubation. I went in to tell him the news, realizing he probably wouldn't hear a thing I had to say.

"We have the results of the tests, Mr. Switek. Your lungs are really bad. We're starting treatment with some powerful drugs. If they work you'll recover from this breathing attack. If not, we might have to sedate you and put a tube in your throat. Under no circumstances can you smoke. Do you understand?"

"OK. When can I get my cigarettes?"

He was incapable of understanding. It was like talking to a child.

"Look, I'll make a deal with you. Let us treat you for a few days. When you get better you can have a cigarette." Never before had I made such a deal.

"OK," he said.

We discontinued Thorazine since the drug was probably depressing his respiratory drive, and provided him with supplemental oxygen through a nasal cannula. At this point improving his breathing had a higher priority than treating his psychosis.

Over the next 24 hours his blood oxygen level came up a little but his heart beat still danced around the monitor and his wheezing was as tight as before. We vacillated about whether to intubate him and begin artificial ventilation. There are no numbers to go by for such an important decision, only careful bedside observation coupled with the results from arterial blood gases.

During his first 24 hours in MICU Mr. Switek's breathing became so labored that he actually ceased asking for cigarettes. In the middle of the second day his agitation increased, a probable result of not receiving Thorazine. At Dr. Janis's suggestion we restarted the drug at a much lower dose than she had used before, only 100 milligrams twice a day.

On the evening of the second day Mr. Switek deteriorated rapidly. His breathing became more labored, nostrils flared with each breath, and he looked like a fish out of water. Coupled with cardiac irregularities and poor oxygenation, all signs pointed to the need for artificial ventilation. We called anesthesiology.

The anesthesiologist came and injected him with a quick-acting paralyzing agent; without complete paralysis Mr. Switek would have been too difficult to intubate. His neck was too fat and he was too agitated. Once intubated, the ventilator took over his breathing. We then placed an arterial catheter in his arm so frequent blood samples could be obtained for oxygen and carbon dioxide measurements.

Over the next few hours the ventilator corrected his oxygen and carbon dioxide levels. As paralysis wore off we began sedative medication so he

would sleep; he had not had much sleep since arriving to MICU. We held Thorazine while he was under sedation.

With sleep, adequate oxygenation and antibiotics Mr. Switek's condition improved. After two days of artificial ventilation his lungs sounded clear. We stopped the sedation and tested his lung function with the ventilator temporarily disconnected: it was much better. We pulled the tube from his throat.

For the next eight hours Mr. Switek was docile: no complaints, no agitation. Then, towards the evening he asked his nurse for a cigarette. She explained that he couldn't smoke.

"Just give me a cigarette," he insisted.

"Harold, you know you can't smoke. You just came off the ventilator," she reasoned.

He did not argue the point. Instead, he waited for her to leave the room. Then he got out of bed to go search for tobacco. This was not so easy in his condition. Mr. Switek had been in bed for four days, two of them unconscious, narcotized. Anyone in his state should get out of bed slowly and always with assistance.

Incredibly, standing up for the first time in several days didn't phase him. He didn't faint or even wobble. However, he still had an arterial catheter in one arm, connected by delicate tubing to a monitor, and a venous catheter in the other arm, through which he received antibiotics. As he moved away from the bed each catheter disconnected from its extension tubing. The venous catheter dripped dark red blood. The arterial catheter, being in a high pressure vessel, spurted blood of a brighter hue.

"Harold, where are you going?" his nurse called out. Harold didn't answer.

"You'll bleed to death," she exclaimed. This was not an idle threat, as arterial blood continued to spurt from the catheter in his arm.

"Naaah. I'll be OK. I need a cigarette." He headed toward MICU's double doors and their EXIT sign.

The nurse quickly realized he was not about to get back in bed. "Let me pull those tubes out of you," she said bravely. He let her. That done, he felt free to roam. Although blood still oozed from the puncture sites at least he would not exsanguinate.

"I want a cigarette," he said to the night clerk at the nurses' station. She got up and walked away.

Two nurses implored him to get back to bed. "OK. Give me a cigarette, OK?"

"We can't, Harold," they pleaded. "Please get back into bed." He moved on, a sight to behold, walking around the MICU nurses' station wearing only a hospital night shirt and no underwear, with blood from each arm dripping to the floor. Our schizophrenic patient was unconcerned with his appearance or health or the scene he was causing. He was possessed

with a single idea – CIGARETTE! The nurses kept their distance. One of them called Security. Then me.

Mr. Switek again headed for the swinging doors that separate MICU from the waiting area. No one tried to stop him. He left MICU and walked down the hallway toward the elevators, where he was met by a security team of two large men.

“Where are you going, Sir?”

“I’m looking for a cigarette machine.” (There is none in the hospital).

“You have to get back to your room, Mr. Switek.” He asked the security guard for a cigarette. They proceeded to escort him back to MICU. As he did not offer any resistance, he may have thought they were taking him to a cigarette machine.

I arrived in MICU just as he was being escorted back by the officers. At the entrance to his room it dawned on him that he wasn’t being lead to a cigarette machine and he turned around rapidly to go the other way.

At this point the scene became physical. He had to be restrained to prevent injury to himself (he would have walked in front of a moving car to get a cigarette). The two guards with the help of a male nurse grabbed his arms and pulled him toward the bed. Harold began thrashing around. Leather restraints were yelled for. He was literally lifted up and thrown onto the bed. One arm, then the other arm, then both legs were tied securely to the bed frame.

Within minutes Mr. Switek was in full leather restraints, thrashing around the bed, screaming and crying. It was impossible to keep a sheet on the bed or an intravenous line in his arm. Sedation was out of the question because of his lung condition. And the last thing I wanted to do was intubate him again.

He looked at me and yelled, “You can’t do this to me! You promised me a cigarette.” (He remembered!)

“Mr. Switek, we have to do this,” I apologized. “You’re going to hurt yourself if we don’t keep you here.”

He began to sob, the sob of a child, a whimpering for candy denied. I left the room and called Dr. Janis. I needed help with this man-child, this schizophrenic, tobacco-addicted, emphysema-riddled bull of a patient. I had to keep him from killing himself.

Dr. Janis recommended trying Haldol, a drug less sedating than Thorazine that can be given by intramuscular injection. I agreed. Controlling his psychosis now took priority over his breathing, which was much improved anyway.

Mr. Switek stayed in leather restraints another 36 hours, until the Haldol took effect. Fortunately his breathing remained stable and he was able to take his other medications orally. On the sixth hospital day the leather restraints came off. Miraculously, he had calmed down and was no longer a threat to himself or others.

“Call my mother,” he requested.

His mother, old and infirm, had not been able to visit him in MICU. I wanted to speak with her also, so I brought a phone into his room and dialed the number. I handed over the receiver as soon as it began ringing.

“Ma? This is Harold. I’m still in the hospital. They’re trying to kill me, ma. Yea. They had me all tied up...Yea, they tried to kill me...Bring my cigarettes, OK Ma? I’m coming home now...I don’t know, here’s the doctor.”

I took the receiver from Harold and talked to his mother, a very pleasant lady who, I imagined, had been through some hellish times with her son. She was quite reasonable on the phone. I explained the situation and why her son had to stay in the hospital a little longer and why he could not smoke. She understood and thanked me, adding that she was ready for Harold “whenever you and Dr. Janis say he’s ready to come home.” We said good bye and I hung up the phone.

“OK, Harold. Your mother says she can’t come to see you now but knows you’re coming home soon.”

He started to cry again. I tried to console him, to reason with him, but it was no use. Mr. Switek was ill in ways I didn’t understand and I felt powerless to help. Since he was not in imminent physical danger I thought it best to just leave him alone.

* * *

The next few days went more smoothly than I expected. We gradually weaned down the Haldol dose and switched him back to Thorazine. An arterial blood gas test showed adequate oxygen and carbon dioxide levels. We allowed him to walk outside his room as long as he stayed within the confines of MICU.

By this time a mentally normal patient would have been transferred to a regular ward, but Mr. Switek’s psychosis required that he either remain in MICU or go to a psychiatric ward. On the eighth day of hospitalization he returned to Weathergill. During the entire 170 hours in MICU he did not have a single cigarette.

Followup

This episode took place in the late 1980s. I kept in touch with Mr. Switek through his psychiatrist for about 10 years, then lost track of him. During that period he remained on Thorazine and continued to smoke heavily. He did not need further hospitalization for his lung problem.

-- END --

12. Pickwickian

I knew Gloria Fallows for about two years before she was admitted to Intensive Care. I first saw her as an outpatient in 1992 when she was 63. Even then she was enormous – 275 pounds, five feet two inches – and had trouble breathing. Her chief complaint was “shortness of my breath.”

“Oh Dr. Martin!” she exclaimed back then. “I can’t walk from here to there without struggling.” She pointed to a wall of my office about ten feet away.

“How long Mrs. Fallows? How long’s it been this bad?”

“Only the past few months. But it seems to get worse each day.”

Weight was her problem. Imagine carrying a hundred-pound sack of potatoes packed around your abdomen and rib cage. Just like movement itself, your breathing would be *restricted*. Gloria breathed this way all the time. Each of her breaths was limited, too shallow to do a proper job of gas exchange.

We normally take in about half a quart of air with each breath. Gloria could only manage one-fourth of a quart. She needed more air than her chest cage, overburdened with largess, could oblige; as a result, her blood carbon dioxide level was too high and oxygen level too low. Gloria was comfortable at rest but exhausted - “I’m wiped out, Dr. Martin” - on walking any distance or climbing stairs.

Breathing problems also interrupted her sleep. She awoke each morning exhausted from incomplete slumber. To compensate, she frequently napped during the day, and at the worst times. She’d been in two car accidents after falling asleep at the wheel. No one was seriously hurt, including Gloria, but at age 62 she had to quit driving.

After that first visit I diagnosed Gloria’s problem as typical of Pickwickian syndrome. Like all syndromes, Pickwickian is not a specific disease as much as a collection of abnormal findings. To most physicians the appellation ‘Pickwickian’ connotes a fat, sleepy patient who has some difficulty breathing. A more precise definition is any patient with obesity, excessive daytime sleepiness and elevated blood carbon dioxide pressure (PCO₂). A high PCO₂ in the blood signifies inadequate breathing or, in medical parlance, “hypoventilation.”

Many syndromes are called by the name of the doctor who first described the malady; less commonly, the name comes from that of the original patient. Pickwickian is unique, for there is no Doctor or patient Pickwick, but instead a literary pedigree. The term is traced to a

common-named character in Charles Dickens' first novel, *Pickwick Papers* (published 1837). At the end of Chapter 53 Dickens introduces a scene involving the fat boy Joe:

A most violent and startling knocking was heard at the door; it was not an ordinary double knock, but a constant and uninterrupted succession of the loudest single raps, as if the knocker were endowed with the perpetual motion, or the person outside had forgotten to leave off. . .

The object that presented itself to the eyes of the astonished clerk, was a boy - a wonderfully fat boy - habited as a serving lad, standing upright on the mat, with his eyes closed as if in sleep. He had never seen such a fat boy, in or out of a travelling caravan; and this, coupled with the calmness and repose of his appearance, so very different from what was reasonably to have been expected in the inflicter of such knock, smote him with wonder.

"What's the matter" inquired the clerk.

The extraordinary boy replied not a word; but he nodded once, and seemed, to the clerk's imagination, to snore feebly.

"Where do you come from?" inquired the clerk.

The boy made no sign. He breathed heavily, but in all other respects was motionless.

The clerk repeated the question thrice, and receiving no answer, prepared to shut the door, when the boy suddenly opened his eyes, winked several times, sneezed once, and raised his hand as if to repeat the knocking. Finding the door open, he stared about him with astonishment, and at length fixed his eyes on Mr. Lowten's face.

"What the devil do you knock in that way for?" inquired the clerk, angrily.

"Which way?" said the boy, in a slow and sleepy voice.

"Why, like forty hackney-coachmen," replied the clerk.

"Because master said, I wasn't to leave off knocking till they opened the door, for fear I should go to sleep," said the boy.

This 19th century portrayal lay medically dormant for over a century, until 1956 when Dr. C.S. Burwell and colleagues published a medical case report, "Extreme Obesity Associated With Alveolar Hypoventilation – a Pickwickian Syndrome." After quoting Dickens' description of the corpulent Joe, the authors went on to describe their patient, a 51-year-old business executive who stood 5 feet 5 inches and weighed over 260 pounds:

[He] entered the hospital because of obesity, fatigue and

somnolence. . .The patient was accustomed to eating well but did not gain weight progressively until about one year before admission. . .As the patient gained weight his symptoms appeared and became worse. . .he had often fallen asleep while carrying on his daily routine. . .on several occasions he suffered brief episodes of syncope [fainting]. Persistent edema of the ankles developed. . .Finally an experience which indicated the severity of his disability led him to seek hospital care. The patient was accustomed to playing poker once a week and on this crucial occasion he was dealt a hand of three aces and two kings. According to Hoyle this hand is called a "full house." *Because he had dropped off to sleep he failed to take advantage of this opportunity.* [Italics original]. A few days later he entered. . .hospital.

. . .Therapy consisted chiefly of enforced weight reduction by means of an 800-calory diet. On this regimen the patient's weight fell from 121.4 to 103.6 kg [267 to 228 pounds] in a period of three weeks. As he lost weight his somnolence, twitching, periodic respiration, dyspnea and edema gradually subsided and his physical condition became essentially normal.

Since that first medical paper thousands of patients have been diagnosed with sleep disorders. The spectrum of problems ranges from occasional insomnia to sleep walking to the far more serious (and potentially life-threatening) Pickwickian syndrome. Today many hospitals run "sleep labs," secluded rooms replete with bed and exotic monitoring equipment for charting physiology during sleep.

Gloria Fallows needed such an evaluation. More important, she needed to lose weight. Even if her sleep pattern tested normal, which I knew it wouldn't, her weight was a serious health problem.

Gloria's wedding picture at 23 showed a woman of 140 lbs, solid and attractive. By age 50 she tipped the scales at 200 lbs but had no (known) medical problems. At 60 she weighed 240 lbs and was under treatment for high blood pressure and diabetes. She added another 35 pounds over the next three years. Fortunately Gloria did not smoke (it is likely the combination of cigarettes and morbid obesity would have been fatal well before I ever saw her). As it was, she could barely manage.

Why did she eat all that food?

"I don't eat that much." she said. "Honest I don't, Dr. Martin."

Doctors used to discount this oft-heard claim of the morbidly obese, but to a certain extent it may be true. Body metabolism plummets in late middle age and a reduced caloric intake may not bring about any weight loss, at least not without the addition of exercise. But daily, aerobic-type exercise for people like Gloria Fallows is seldom feasible. The only solution for most

massively obese people is such a drastic decrease in calories that medical supervision becomes necessary.

Gloria had been on diets before but they always failed. “No will power,” she confessed. But she had never been in a medically-supervised weight loss program.

“Gloria,” I said, “you need two things. First, you need to lose weight in a special program so doctors can follow your metabolism. And we need to study your breathing to see why your oxygen is so low. The only way to do both is to put you in the hospital.”

“Hospital? It’s that bad?”

“Yes,” I insisted.

“Will my insurance cover it?”

I checked. Her insurance plan did not recognize hospitalization for obesity alone, so I admitted her for “respiratory failure, chronic.” Unfortunately for Gloria this was a legitimate diagnosis, confirmed by the elevated carbon dioxide in her blood.

We did a battery of tests to check organ function: of her heart, lungs, liver, kidneys, pancreas and adrenal glands. Surprisingly, all tests were normal or near-normal except her lung function. She had reduced lung volumes, confirming our clinical impression of restricted breathing. As a result of not being able to take deeper breaths, her blood oxygen pressure was only 55 mm Hg (millimeters of mercury; normal is above 80) and carbon dioxide pressure 53 mm Hg (normal is between 36 and 44, with an average of 40). Some physicians jokingly refer to patients like Gloria as belonging to the “50-50 club,” signifying the abnormal blood oxygen and carbon dioxide levels. Membership is definitely not desirable.

By the evening of day three our routine tests were completed. We sent her to another wing of the hospital for a sleep study, technically known as ‘polysomnography’, the recording of many (poly) records (graphy) during sleep (somno). The study is conducted in a windowless room with the subject asleep on a queen-sized bed, reinforced to sustain the heaviest patients. A technician hooked Gloria’s head, nose, ear, chest, and extremities to multiple wires emanating from sundry monitoring devices.

I went to observe the beginning of the sleep study. Laying in bed, wired up, surrounded by all kinds of electronic boxes, Gloria looked like a character in a sci-fi thriller. I thought of taking a picture and sending it to one of the tabloids. The headline would blare FAT WOMAN ZAPPED INTO THIN BEAUTY or something like that. My picture would be the ‘before’ pose; the tabloid would find a beautiful model with a similar face for the after shot. (I didn’t do it, of course, but just remember where the idea originated.)

Twenty minutes later Gloria was asleep, so I left her to the technician and his monitoring devices. She slept from 10 p.m. to 6:30 a.m., when she was wheeled back to her regular ward bed. I saw her on morning rounds a

few hours later.

“Well, Mrs. Fallows, did you sleep last night?”

“Off and on, Dr. Martin. Off and on. They sure had me wired up.”

“How do you feel now?”

“OK. A little tired I guess.”

I went to the sleep lab to check the results. Gloria’s polysomnogram showed three things. She snored a lot; her throat tended to close and block her upper airway during sleep (called sleep apnea); and her blood oxygen level fell, at one point to a level incompatible with any longevity. During sleep Gloria Fallows was at risk for *sudden death*.

I prescribed a night-time breathing machine, called “Bi-PAP” for Bi-level Positive Airway Pressure. A Bi-PAP machine is essentially a watered-down version of the full scale life-support ventilator, the kind used routinely in intensive care units. About a foot square in size, the Bi-PAP machine sits on a table or night stand, and is connected to the patient via a long hose. At the end of the hose sits a small, nose-shaped mask, made out of soft rubber. With the aid of head straps, the mask can fit tightly over the patient’s nose, so air will not escape as it is pushed through the nostrils (older mask versions covered both mouth and nose, but they proved too uncomfortable for night time use).

During sleep (the most vulnerable time for air flow obstruction) the Bi-PAP machine pushes air into the nose, upper airway and lungs. This “pushed” air enters under increased pressure, and in this manner helps prevent the upper airway from collapsing while the patient sleeps. The air pressure is highest on inspiration, when the machine does all of its work. The patient exhales passively and the air pressure falls, but is still elevated above normal (hence the two levels of air pressure). Better ventilation helps transfer oxygen into the lungs and carbon dioxide out, ameliorating the Pickwickian’s gas exchange problem.

When the Bi-PAP machine works, it works well. Gloria tried Bi-PAP only two nights before rejecting it. “It’s like sleeping in an air vent,” she said. (I could not argue, having never slept with the device. For the record, however, many patients gladly accept the machine’s incessant WHOOSH-whoosh, WHOOSH-whoosh, in exchange for a night without sleep apnea. Spouses often have a harder time.)

I prescribed a more comfortable nasal cannula for use during sleep; through it, extra oxygen enters the nostrils and lungs, but at no increase in air pressure. Though not as effective as Bi-PAP, nasal oxygen at least kept her O₂ level from hitting rock bottom during sleep.

In the middle of week two Gloria started a liquid protein supplement diet. All solid food was taken away and she drank only the liquid meal, several times a day. The supplement allows the body to burn mainly carbohydrate and fat during what amounts to semi-starvation. For patients who stick to the supplement there is often remarkable – and safe – weight

loss.

After three weeks in hospital Gloria went home weighing 255 pounds. A 20-pound weight loss was not bad in such a short period, but the first 20 are the easiest. Now all she had to do was continue the diet, plus use her nasal oxygen at night.

At first all was success. A month after discharge she weighed 240 pounds and her oxygen level was up. In another two months she weighed 230 pounds, a satisfying drop of 45 pounds in only three months. She looked and felt better and had improved blood gases as well.

Unexpectedly, she quit attending the clinic. Since that was the only place to get the protein supplement, she quit dieting as well. About a month afterwards I received a card from the weight-loss clinic: "Your patient, GLORIA FALLOWS, has dropped out of the Liquid Protein Diet Program. Please let us know if we can be of any further help in her weight control."

I called her. "Mrs. Fallows, what happened? Why did you quit going to the weight clinic?"

"Oh, Dr. Martin. I couldn't get a ride anymore. And it was just too far by bus."

People who lose weight in the best of programs frequently gain it back. Reasons for sliding are varied, but Gloria's was a common one – inability to keep up clinic appointments. She could have found other transportation but didn't make the effort.

On the phone she admitted to gaining weight and having more trouble breathing. I saw her the next day, in my office at the hospital. She weighed 262 pounds and had much leg edema (swelling from excess fluid). A chest x-ray confirmed early congestive heart failure. I admitted her to the hospital and began diuretics to mobilize the fluid. She did not need the intensive care unit on this admission.

Our specialist in morbid obesity saw her in consultation. He didn't mince words, writing in the chart: "Given the severity of her problem and recent failure on the liquid protein diet, I suggest consideration for gastric stapling. Please contact Surgery."

First you try dieting without supervision. That seldom works. Then you try supervised dieting. That is sometimes successful. When it fails you have a range of procedures to choose from, all disappointing in their long term results. Gastric stapling, literally stapling the stomach into a smaller pouch for receiving food, was at one time a popular operation for the massively obese. (The 'stapled' stomach was supposed to make the patient feel satiated with less food. Success with the operation was limited, however, and is seldom done anymore.)

A surgeon visited Gloria to explain the operation and the risks. "Let me think about it," she said. She thought about the procedure for two days and decided against it. "I'll lose weight with the diet," she said.

"Gloria," I remarked on learning of her decision, "you failed the diet."

You gave up.”

“Oh, Dr. Martin! I won’t quit next time. I promise.”

“It’s up to you, Gloria.”

“Let me try again.”

She was accepted back into the diet program. We also made special arrangements for transportation if she couldn’t find a ride to the clinic. Most patients are given only two chances in the program; this was Gloria’s second.

She left the hospital in a week, weighing 253 pounds.

* * *

Gloria quit the diet three months later, this time with the excuse that “it just wasn’t for me.” Compliance is everything in weight reduction and there was nothing more the diet clinic could do.

She continued using the nasal oxygen and diuretic medication, and her weight did not go down. It didn’t go up either, but age was against her. What the 40- or 50-year-old-body can tolerate, the 64- or 65-year-old can find unbearable.

I followed Gloria, along with her internist, but we could not correct her underlying medical problems. Her oxygen and carbon dioxide levels remained grossly out of balance. She was – I told her several times – a ticking time bomb. It was a question of when, not if.

The bomb went off in late March, 1994, just after she turned 65. I was called from the emergency department. “Dr. Martin, this is Dr. Thompson. I understand you know Gloria Fallows? Her internist asked that I contact you.”

“Yes, yes. What happened?”

“Mrs. Fallows rolled in about an hour ago, almost apneic. We intubated her and will be sending her up to MICU [Medical Intensive Care Unit].”

“I was afraid this would happen. What caused her to fail?”

“We don’t know. Chest x-ray’s clear and her cardiogram shows no acute changes. She apparently collapsed at home and EMS [Emergency Medical Service] was called. When she got here her PCO₂ was ninety-six and PO₂ only thirty-five.”

“Wow! Sounds like she was on her way out.”

“Yes. We had real trouble intubating her. Finally had to put the tube through her nose. Her blood gases are improving on the ventilator and she’s stable enough to be moved. Do you have a bed available now?”

“Sure. Send her right up.”

I *thought* I knew Gloria but the person rolled into our intensive care unit was much larger than what I remembered. She must have gained at least another 50 pounds. Three nurses and two doctors lifted her from the transport stretcher to hospital bed. HEAVY.

Her MICU bed rested on a scale so that additional weight could be accurately recorded. She weighed 318 lbs. and looked it. Her belly was enormous. How could anyone breathe with all that fat pressing on the lungs?

About 30 minutes later, after things were squared away with our patient, I went to speak with Mr. Fallows in the family waiting area. A thin, balding man in his mid-60s, he had just recently retired from a job with the post office. I knew from previous visits that their marriage was a good one and that Mr. Fallows was devoted to her care. Unfortunately there was little he could do without her cooperation.

"She's stable now, Mr. Fallows," I said. "But she was in a lot of trouble when she arrived. What happened to her? Look's like she's gained over 60 pounds since I last saw her."

"I don't know, Doctor Martin. She just lays around at home and doesn't do much. For the last few days she's been kind of mooney. Today I couldn't even get her out of bed."

"Why didn't you call us before?"

"She didn't want to come to the hospital. She told me not to call the ambulance. Said she was fine, just wanted to be left alone."

I affirmed that she was critically ill and could die any time.

"Well, I have faith. Just do what you can for her, Dr. Martin."

Our initial tests did not reveal any acute infection or other explanation for her deterioration. She seemed to have worsened just from increase in both age and weight. That left only weight to correct. Until we took off a couple dozen pounds – or redistributed them – she would likely need the ventilator.

The next morning on rounds we found her awake, laying in bed with her head raised slightly on one pillow. She looked pachydermish with the endotracheal tube coming out of her right nostril, a giant, thick neck, and a mountain of fat south of that. Her legs were huge, rounded limbs of hardened tissue, the result of years of waxing and waning edema. The skin around her ankles was bluish-red and scaly.

I arranged the sheets to expose her abdominal protuberance. We've had heavier patients before (one of 550 lbs), but Gloria's short stature and Jabba-the-Hut appearance made her look more grotesque than the others. Clinical knowledge and professionalism aside, very large patients always elicit a bit of voyeurism. So it was on teaching rounds with the interns and residents. Everyone stared at Gloria's belly.

Placing my hands on her huge abdomen, I pronounced: "*This* is the problem." Mrs. Fallows nodded in agreement. "I'm not going to minimize the situation, Mrs. Fallows. Your weight is killing you. It's got to come off." She nodded again.

"We'll do what we can to get that tube out of your nose. But when we do, you have to lose some weight. No, a *lot* of weight. Or you'll be right back here." I pointed to her bed and she nodded.

I reviewed the ventilator settings and blood gases with the house staff, examined Gloria's lungs and heart, noted her fluid intake and urine output. She was stable but not ready to come off the ventilator. I led the house staff out of her room, to resume discussion near the nursing station.

"What would you do now?" I asked Sherry, one of the senior residents. A petite young woman, she was perhaps the brightest of the group.

"I don't know. Can we keep her on the ventilator while she loses weight? I guess that's one way to stop her from eating."

"Is there any alternative?" I asked the group. "Can we get her off the ventilator the way she is now?"

They were stumped.

"What's her major problem?" I asked.

"Her weight."

"Right. Any other problem?"

"Well, hypertension."

"Right, that's a problem too, but it's not what I'm thinking of. Is there any other reason she could be in respiratory failure besides the weight?"

"She doesn't smoke and has no asthma. Her chest X-ray is clear. What are you getting at, Dr. Martin?"

"Suppose you studied blood gases [measurements of PO₂ and PCO₂] and breathing capacity in 20 non-smokers, all under 5 feet 3 inches and weighing over 300 pounds. What do you think you would find?"

"I don't know," said Sherry. "Did you do that study?"

"No, but others have. Breathing abnormalities were found in most of them, but not enough to seriously impair gas exchange. In other words, they did not have frank respiratory failure like Mrs. Fallows, even in those over age 60. The point is, weight by itself is not the only problem. There has to be some other factor or factors to explain *her* respiratory problem. I've been following Mrs. Fallows for two years. Even when she weighed two-fifty she had hypoxemia [low oxygen level] and CO₂ retention.

"Most likely patients like Mrs. Fallows have an abnormal brainstem respiratory center. For some reason, her brain won't let her do the extra work of breathing all that extra weight requires. Many obese people *are* able to do the extra work and maintain decent oxygen and carbon dioxide levels. She can't do the extra work necessary for deeper breaths, so her blood gases become abnormal and stay that way. It's just a theory, but it does help explain why most very obese patients don't have her problem.

"Would respiratory stimulants help?" asked Sherry.

"You mean some kind of pill to stimulate her breathing?"

"Yes, something like that."

"A few have been tried, particularly progesterone. They generally don't work, and if they do it's only over the long term. Certainly that's not going to help in the short term. We've got to get her off the ventilator very soon. Any other ideas?"

“What about a fat-ectomy?” someone else asked. (Literally, cutting off the excess weight.) The question generated snippets of laughter.

“What about wiring her jaw shut?” queried another, eliciting more laughter.

“You laugh, but those are possibilities. Still, you’re not really answering my question. How can we safely get her off the ventilator in the next few days? She’s not going to lose enough weight to make a big difference in a few days. How are we going to do it?”

“Diuretics,” someone said.

“Diuretics will help mobilize excess water, but probably won’t make much of a dent in her belly. Anyway, she’s already on Lasix [a potent diuretic]. Any other ideas?”

No answer.

“Well, there’s one way,” I said. “A therapy too little used in intensive care. What is it?”

They were stumped by my guess-what-I’m-thinking question.

“I’ll give you a hint. It’s not a drug and not a medical device.”

“What else is there?” asked Sherry.

“I’ll give you another hint. It’s an elemental force of nature. One of the four primary forces.”

Ohhhhhh,” swooned one of the heretofore silent residents, a quiet chap who had been listening intently.

“Yes?”

“Isaac Newton.”

“Yes! That’s right. We’re going to use *gravity*. It’s free and every room is equipped. If we don’t get that tube out of her throat soon she’s bound to have a major complication. Infection or airway damage. A tracheostomy on Mrs. Fallows would be very difficult. She has no neck. A surgeon looking to place a hole in her trachea could get lost.” The house staff glanced back at Gloria, visible through the glass door, and nodded in agreement.

“We’ve got to get her off the ventilator,” I said. “The easiest way to take advantage of gravity is with . . . an anti-gravity bed.”

“What’s that?”

“A bed that will allow her to sit up without sliding. Look at her. She’s in the anti-breathing position, all squinched up in bed. With the typical hospital bed like this one, you can’t keep her abdomen from attacking her chest. How can anyone breathe with all that weight squeezing the lungs? If we can just unload her lungs a bit I think we can get her off the ventilator.”

“Marsha,” I said to our head nurse, “can we get her one of those Big Boy beds? You know, the kind we used for that five-hundred pounder?”

“Sure, Dr. Martin. I’ll see what I can do.”

* * *

Unlike a conventional hospital bed, the Big Boy is constructed in four bendable sections. Each section can be adjusted to place the patient in practically any desired position. The Big Boy made it possible to care for Gloria in a semi-sitting position, night and day, effectively shifting her massive abdomen *downward*, away from her chest. By using gravity to lower the abdominal mass, her lungs had more room to expand with each breath. She remained in this posture (with slight variation) for the next two days while we gradually turned down the ventilator settings.

On her third day in MICU day we disconnected the ventilator. At that point she breathed on her own, but still through the endotracheal tube. Her arterial blood gases remained about the same as baseline values: PO₂ 64 mm Hg, PCO₂ 59 mm Hg. I decided to take a chance and remove the endotracheal tube. We could always put it back in.

After extubation Gloria's PO₂ remained low, PCO₂ high, but not deranged enough to require re-intubation. Still, she wasn't ready to leave intensive care. She remained in that precarious state I call 'ventilator limbo': almost-but-not-quite needing artificial ventilation, almost-but-not-quite ready to leave MICU for a regular ward bed.

When not sleeping Gloria just lay in bed. She made few demands on the nurses and generally seemed unconcerned about her situation. There was no hint of desire to get better and leave MICU. Had she given up? Or was her moodiness just a result of deranged blood gases? We found no evidence for neurologic damage. She was oriented and conversant but simply unmotivated, listless really. Overall, a bad sign.

Gloria's fifth day in hospital, April 1, was also the day some of our house staff changed rotations. Gloria got a new intern, Roger Bailey, a 29-year-old with career interest in one of the surgical specialties. Roger was in his ninth month of internship (the academic year begins in July) and had already spent a month in our ICU the previous October. He knew his way around. What he didn't know, unfortunately, was much about pulmonary physiology.

Interns and residents cannot choose their patients. They have to take whoever is assigned on the rotation; if Dr. Bailey had any choice it would not be Mrs. Fallows, for he was not sympathetic to her medical problems. As far as he was concerned, if she just took deeper breaths she could get out of the ICU and off his service. He did not like obese, slothful patients.

During morning rounds on April 3 he asked, "Dr. Martin, can we transfer her out of MICU today? I don't think we're doing much for her here."

"What are her blood gases this morning?" I asked.

"About the same. PO₂ fifty-six, PCO₂ is seventy-two, on nasal oxygen."

I reviewed all the blood gases measured since extubation. They did not

show much variation: low oxygen and high carbon dioxide. We had tried everything to increase her ventilation, including the Big Boy bed, deep breathing exercises, adjustments to diet, and diuretics to mobilize edema fluid.

“I don’t know,” I said wistfully. “I wish she could just take deeper breaths and lower her CO2 level.”

At my remark Dr. Bailey’s eyes opened wide. He must have been looking for the proper opening and I provided it. “I think I have an explanation for her failure to improve,” he said, rather professionally.

“Really? What?”

“Mrs. Fallows just doesn’t want to breathe more. It’s her personality. She needs motivation.” Translation: let’s get a psychiatry consult and transfer her out of intensive care. This may seem, to the lay mind, a not unreasonable suggestion; after all, much illness can be related to depression, poor motivation, low self esteem. But even assuming psychiatry had something to offer by way of remedy, Dr. Bailey’s explanation and understanding of her situation were naive, wrong-headed. I decided to respond with mock incredulity.

“WHAT? What did you say? The problem’s her personality?”

“Yes,” he replied. “Some patients just don’t want to breathe deeply. She’s just lazy and wants to be this way. Maybe psychiatry can help her.”

How to answer this medical delusion? He was so scientifically confused I was actually amused. Personality and attitude have nothing to do with why patients under breathe or have a low oxygen level. First-year-medical-school physiology teaches that one cannot raise carbon dioxide or lower oxygen levels by any aspect of *will or mind control* (transcendental or other forms of meditation notwithstanding). That is why children who hold their breath to gain attention cannot really stop breathing. The brain’s breathing control center won’t let a child (or an adult) voluntarily slow down ventilation to a dangerous level. As soon as the control center senses the slightest buildup of CO2, *you will breathe*. It’s that powerful a stimulus.

I knew this, but did Dr. Bailey? I decided to give him the benefit of doubt. Perhaps I misunderstood his explanation. “You mean because she eats a lot that her breathing is affected, and that psychiatry could help motivate her to lose weight. Is that what you mean?”

“Well, that too,” said Roger. “But you see this kind of breathing in lazy people. It’s just her basic personality.”

‘Wow!’ I thought. This is getting out of hand. Was he serious or just being playful?

“Roger, do you have a reference for that?”

“Well, I read that somewhere,” he said lamely.

“Where?”

“I don’t remember. Somewhere.”

Time to attack.

“Roger, suppose you measured the PCO₂ of everybody with any kind of psychiatric disorder, excluding people on massive amounts of anti-psychotic medication. What would be the average PCO₂ of all these people? Would it be high, low or normal?”

“Oh, I don’t know,” he said.

“Anybody?”

“Forty?” asked Sherry (she was still on the MICU rotation).

“Forty point oh oh,” I said. “In other words, normal. And suppose you measured PCO₂ of people who are, say, just depressed and mopey, but not with a bona fide psychiatric diagnosis? What would you find, on average?”

No one answered, so I did, “forty point oh oh.” No comment, so I continued.

“And, what’s the average PCO₂ of all the people who are nasty, mean, uncivilized, unwashed, and – or – just plain LAZY?”

This time I waited for someone else to answer. A short pause, then Sherry spoke up once again: “Forty?”

“Right. Very good, Sherry. “Forty point oh oh. Any other bad traits you want to know about as far as the associated PCO₂?” It was all in good fun. No one seemed offended and most of house staff, at least, were enjoying the repartee.

“Roger,” I continued, “you clearly don’t understand what can make someone under ventilate. Fat can do it. She’s fat. Some kind of structural lesion in the brainstem can do it. That’s a theoretical possibility here, but there’s no way to prove it. Long term smoking can do it by causing severe lung damage, like emphysema, but Mrs. Fallows doesn’t smoke. Some drugs can slow breathing, particularly sleeping pills and narcotics, but she’s not on any of those. Weakness of the chest cage muscles can do it, and she may have that problem to some extent. But personality? Never.”

No one said anything. Time for the repetition routine. If you can’t help the patient, at least teach the house staff. Make a point they likely won’t forget, even if you have to act overbearing while doing so.

“Sherry, can a patient’s personality explain an elevated CO₂ level?”

At first Sherry looked startled, but she quickly caught on. “No, Dr. Martin. Personality can’t be blamed.”

“Roger, can the nature of a patient’s personality explain an elevated PCO₂?”

He took my teasing repetition good-naturedly. “No, Dr. Martin, personality can’t be blamed.”

“OK. Anybody have any questions?” No one did.

“Well,” I said, “I think we’ve done everything possible for Mrs. Fallows in MICU. Why don’t we transfer her out.”

* * *

I wish I had a happy ending, but the outcome for the morbidly obese patient with respiratory failure is never happy. Mrs. Fallows stayed in the hospital another two weeks. Her attitude improved along with a return to ambulation. On the day of discharge her weight was down 45 pounds and arterial blood gases, though better than in MICU, were still far from normal.

She was given a rare third chance in the special diet program. She stayed with the diet for a few months, then stopped a third time. Two months later she collapsed at home. Mr. Fallows called Emergency Medical Service and paramedics reached the house within minutes. They found her apneic and began CPR, which was continued during the ten minute ambulance ride to the hospital. On arrival in the emergency department she had no pulse except that provided by external chest compressions. Another half hour of CPR failed to restore her heart beat and she was pronounced dead. No autopsy was performed.

-- END --

13. Coma

There is coma and there is COMA. Jack Wilkerson, a 35-year-old accountant, was out for six months and we never knew why. His case was the stuff of tabloids (**MAN SLEEPS HALF-YEAR – DOCS BAFFLED**) and also the kind that ends up in medical journals ('Prolonged coma of unknown etiology: report of a case and review of the literature').

It happened this way. One day in July I got a call from the emergency room. "Dr. Martin, we have a thirty-five-year-old man who may have encephalitis. He's confused and febrile to one hundred point two. His brother says it began with a headache last night and this morning he didn't know where he was."

"Has he been tapped?" I asked.

"Yes, his spinal tap is clear, so we don't think he has meningitis. Anyway, he's going for a CT scan and then to MICU."

Apart from the fact that encephalitis is always a serious medical problem, there was nothing particularly unique or startling about this message. We see encephalitis all the time. Because of the potential for disaster, such as respiratory failure, everyone with this diagnosis is first admitted to the medical intensive care unit.

On arrival to MICU Jack Wilkerson did not look ill at all, just a bit drowsy and confused. Of average height and stocky, he had a day's growth of beard and a sweet, round face that tended to stare off into space, both eyes wide open. He was arousable by shaking, and actually answered some questions appropriately, but for others his answers made no sense.

"What's your name?"

"Jack."

"Jack what?"

"Jack."

"Where are you, Jack?"

"My brother."

"Anything hurt you, Jack?"

"Yes."

"What?"

"Yes."

And so it went. All we knew was that Mr. Wilkerson had been healthy two days earlier, and that along with his confusional state he had a fever but no evidence for meningitis (inflammation of membranes surrounding the brain). Instead, the clinical picture suggested encephalitis or inflammation *within* the brain. His vital signs were stable and otherwise he seemed in good physical health.

Additional medical history provided by his brother was not especially

helpful. The patient had never been sick before: tonsils out at age eleven, occasional flu syndrome, but no illnesses of any type for about a year. He was a certified public accountant and lived alone. He was unmarried but had no history of homosexuality. Nor was there any history of drug abuse or alcoholism or travel outside the country. In short, nothing to suggest a diagnosis more specific than “encephalitis of unknown cause.”

The CT scan of his brain was negative: no evidence for stroke, hemorrhage, or tumor. We ordered blood cultures and many other blood tests; all eventually came back normal or negative. A ‘toxic screen’ of his blood – a test for about a dozen common poisons and drugs people overdose on – turned up nothing except aspirin, and at a level expected in someone using the drug for headache. Urine and feces were examined for infecting organisms; none were found. Blood was sent to the state lab for viral titers; these results would be inconclusive until repeat testing could be accomplished in three weeks to check for a diagnostic rise in titer.

Six hours after admission to MICU Jack became less responsive. His level of breathing remained adequate but his eyes were now closed, a sign of developing coma. A plastic catheter was inserted into his radial artery so arterial blood could be drawn to monitor oxygen and carbon dioxide. Another tube was inserted through his penis into his bladder, to monitor urine output.

Mr. Wilkerson deteriorated rapidly. Twelve hours after he first presented his breathing became shallow and his blood carbon dioxide level began to rise. Respiratory failure set in. To provide artificial ventilation doctors inserted a foot-long hollow plastic endotracheal tube into his windpipe, and connected it to the ventilator. Until coma reversed the patient would have to be breathed by machine.

Another tube was inserted through his nose and into the stomach, for feeding. Twenty-four hours after hospital admission Jack Wilkerson had five plastic tubes in his body and was completely comatose.

We called in two consultants, Neurology and Infectious Disease. “Stage IV coma – possible encephalitis,” opined the former. “Recommend continued support. Check titers for possible viral etiology.”

The ID specialist was no more helpful. “Puzzling case, not typical of encephalitis in this area. No indication for antibiotic coverage.” (Note to medical readers; this case pre-dated current antibiotic therapy for some types of viral encephalitis).

Mr. Wilkerson’s coma continued. More doctors came by, to ponder or wonder or offer their advice. It all amounted to the same thing: wait and watch.

The second week came and went, without improvement. The toll was especially hard on Jack’s brother. Tom Wilkerson, 38, owned a cabinet shop, had a wife and two kids, and was as close to his brother as any adult sibling could be. Tom’s business suffered as he spent hours at Jack’s

bedside. “When do you think my brother will wake up? Anything new?” Always the same good questions and always the same answer – ‘We don’t know.’

The longer someone is in coma the worse the prognosis. This is not necessarily because the brain is damaged, but because the rest of the body tends to wither from disuse, particularly the arm and leg muscles. Also, bed sores develop quickly unless the patient is regularly turned from side to side.

The many plastic tubes necessary for medical care can also cause problems. The urinary tract becomes ripe for infection with a catheter in the bladder. The endotracheal tube can damage the vocal cords and trachea. Catheters in the arteries and veins tend to cause inflammation and infection if not meticulously cared for and changed frequently. Also, because of inadequate nutrition body weight almost always falls in the comatose patient; a feeding tube is no substitute for the healthy appetite.

A completely comatose patient requires a small army of nurses and therapists to prevent complications of lying still for so long. Around the clock people had to turn Mr. Wilkerson from side to side, massage his muscles, change his catheters, oil his skin, feed his stomach. It would do no good to have him wake up only to be crippled by muscle contractions, or missing a limb from hospital-acquired infection, or suffering a giant decubitus ulcer (these things have happened).

Jack’s body was well maintained. As for his brain, it had its own agenda. He did not wake up. There was not even a glimmer of improvement. A brain wave test – electroencephalogram – was carried out once a week but it’s pattern never varied: “...slow waves consistent with encephalopathy.” No diagnosis, really.

At Tom’s suggestion, and with our ready acquiescence, a consultant neurologist was called in from another hospital, someone no more qualified than our own staff neurologist but perhaps able to offer a fresh perspective. His conclusion after two hours with the patient and the chart: “Agree with current management. Continue what you are doing.”

Mr. Wilkerson’s case became part of the hospital’s culture. I was no longer met in the hallway with the perfunctory ‘How are you doing?’ but with ‘How’s Mr. Wilkerson doing?’ “The same,” I found myself replying each time. “Still sleeping.”

By the middle of the third week, when it became clear Mr. Wilkerson was not going to wake up soon, he went for tracheostomy. This operation places a short breathing tube directly through the neck into the trachea, and is routine for patients who require prolonged artificial ventilation. It frees up the mouth and provides for much better oral hygiene. Also, if the patient is awake he or she can eat while being breathed by machine.

During the fourth week we took out Jack’s nasogastric feeding tube. To provide a better conduit for food surgeons placed a rubber feeding tube directly through his abdominal skin into the stomach, a procedure called

percutaneous endoscopic gastrostomy (PEG). With both the endotracheal and feeding tubes removed Mr. Wilkerson looked much more comfortable. Whether he felt that way was impossible to know.

By the end of a month all his tubes had been changed at least once, some as many as five times. His weight was down from 180 pounds on admission to 147, and holding steady. Every-other-day blood tests showed no deterioration of any vital organ. In short, Mr. Wilkerson was stable, just not recovering from coma.

Repeat viral titers were sent to the state lab. To our surprise, all tested negative; there was no rise in titer of any virus known to cause encephalitis in our state.

Mr. Wilkerson's case was explored in conference after conference at the hospital. The ID people presented his problem in a seminar on difficult-to-diagnose encephalitis. The neurologists found his length and depth of coma worthy of academic discourse. Lung specialists waxed over the subject of prolonged artificial ventilation. Nurses discussed basic care of the unconscious patient, and the physical therapists highlighted the importance of maintaining muscle tone by passive range of motion. The nutritionists talked about feeding the comatose patient and assured us (as we already knew) that, food-wise, Jack could be kept alive "forever."

A month elapsed and still there was no improvement. Emboldened by his survival yet dismayed by the lack of progress, we decided it was time to experiment. Perhaps something in our patient's blood was causing coma that we could not detect with all our tests. A method is available for 'washing' the blood of unwanted proteins, called 'plasmapheresis.' Had plasmapheresis ever been used before in prolonged coma? Well, no, at least not at our hospital. But it is used successfully in other afflictions of the nervous system, including Guillain Barré syndrome and myasthenia gravis. True, these are disorders of the *peripheral nervous system*, not the brain, but what did we have to lose? What did Jack Wilkerson have to lose? Because the procedure was somewhat experimental, at least in this case, we asked for and readily received permission from his brother.

Three times a week the plasmapheresis device, a bulky machine the size of a console TV, was wheeled into his room and connected via tubing to his arm veins. Blood from one vein entered the machine to be "pheresed," or washed, and a fresh plasma-like solution was instilled back into another vein. Each washing session lasted about two hours. From experience with other diseases a beneficial effect from pheresis might not be seen for weeks.

We continued plasmapheresis for a whole month, longer than usual in other diseases. Mr. Wilkerson didn't get worse and he didn't get better. Essentially nothing happened.

At the end of the plasmapheresis trial – after two months in the hospital – I noticed a perceptible shift in attitude among the hospital staff. Many now felt that Mr. Wilkerson would never wake up, that he wasn't going to

live. This attitude found its way into the hospital chart, among notes of the several consultants still involved in his care: “Prognosis appears hopeless” and “At this point doubt meaningful recovery” and “Poor outlook. Discussed with brother.” No one actually gave up, of course. He continued to receive excellent care. But the clinical and intellectual excitement of the first two months was no longer present, and other patients and other problems took center stage. Jack Wilkerson’s coma became more or less an accepted fact and his problem was pushed ‘to the back’ of the ICU. We continued daily bedside rounds but his blood was now drawn only once a week, a chest x-ray taken only every ten days or so, and the chart notes became less wordy and appeared less often.

“About the same.”

“No change today.”

“Vital signs stable.”

“Agree with ongoing management.”

Only the intern, newly assigned each month, wrote comprehensive notes. Everyone else sort of backed off. This change in intensity of care was entirely appropriate. We had done virtually everything feasible and, apart from supporting his ventilation and monitoring bodily functions, as physicians we seemed to have no therapeutic role. If Mr. Wilkerson was going to wake up it would be on his brain’s own timetable.

Tom Wilkerson never gave up. He was at his brother’s side daily, talking to him, reading letters, playing the radio. It is a matter of controversy whether comatose patients benefit from stimulation by familiar sounds. Tom had discussed the issue with several laymen and was convinced of the value of audio stimulation. We were more than happy to go along with this and made sure that Jack “listened” to music via headphones at least six hours a day.

Fall came and went. Discussions were held about sending Mr. Wilkerson to a chronic care facility but his brother was against it. He felt Jack’s only chance was to remain in the hospital, in the ICU.

The new year came. We had no plans to move him or change anything, just to continue administering food, fluids, artificial ventilation, and good nursing care. No one expected anything more.

* * *

One day in mid January, as suddenly as it had begun, Jack’s coma lifted. He opened his eyes and looked at a nurse washing his face. I was not there but heard that she cried out, “Jack, you’re awake!”

Because of the tracheostomy tube he could not talk even if he remembered how, but he was evidently coherent. He looked around, as if to ask “Where am I?” Everyone came to see and stare. One nurse remarked, not inappropriately, that it was like seeing a dead man wake up.

Suddenly the chart notes blossomed. “Mr. Wilkerson appears to have

spontaneously recovered from prolonged coma,” wrote the neurologist. “Will repeat EEG.”

“Spontaneous remission of ?non-infectious encephalitis,” wrote the ID specialist.

“Remarkable improvement,” wrote the intern in her daily note. “Praise God,” said Jack’s brother, not usually taken to religious commentary. “What made him come out of it?”

“I don’t know. No one knows!” I exclaimed. “Let’s hope he doesn’t relapse.” That is always a possibility with encephalitis.

It took another week to get Mr. Wilkerson off the breathing machine, then several more days to get him to the point of standing at the bedside. Remarkably, amazingly, he continued to improve, haltingly at first, then steadily, with each passing day.

After two weeks of aggressive physical therapy he learned to walk again. The whole hospital turned out to observe. And to enjoy.

* * *

One day in early February Jack was wheeled down to the lobby where his brother’s car was waiting to take him home. Using a cane for balance and helped by his brother and a hospital aid, but walking under his own power, Jack entered the front seat of the car and the door closed. He had been hospitalized seven months and three days.

Extensive neurologic testing in April revealed no significant deficits. His EEG was read as normal and residual muscle weakness we attributed to prolonged disuse rather than any nerve damage. He returned to his accounting job and reported no problems with memory or calculating ability. By May Jack Wilkerson was back working a 40-hour week.

Several years after admission for prolonged coma, Jack Wilkerson is healthy and well.

Comment

Jack Wilkerson’s recall of seven months in the hospital *begins* with his first trip to physical therapy, about a week after he woke up. The entire hospital stay until that point is a blank. He feels lucky to be alive and, like the rest of us, has no idea what happened or why.

Many people cite his case as an example of why doctors should never give up on a patient in coma, but it is often a comparison of apples to oranges. Mr. Wilkerson’s problem was coma of unknown cause in an otherwise healthy body. The outlook is very different for many known causes of coma, such as with the patient in the next story, “Cocaine Wins”.

– END –