Clinical Teaching Scripts for Inpatient Medicine

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❖ Hypokalemia

Purpose

Potassium disorders are a frequent occurrence in the inpatient setting. The frequency of intended or accidental diuresis (occurring with, for example, heart failure, diabetes, and alcoholism) makes hypokalemia prevalent; the frequency of renal failure makes hyperkalemia equally prevalent. Both disorders pose significant cardiac arrhythmogenic risks, increasing their importance. Hypokalemia has a special importance as a teaching topic—unlike other electrolyte abnormalities, there is rarely the luxury of quickly correcting the abnormality. The arrhythmogenic risks of repleting potassium too quickly create a scenario in which the best strategy is to prevent having the problem in the first place. An admonishment to "keep the potassium in the normal range" is rarely effective. The attending can be much more effective in her coaching by teaching the physiology that underlies the development of hypokalemia (diseases characterized by diuresis, either intrinsically or by their management) and the physiology that underlies the arrhythmogenic complications of hypokalemia.

This is also an opportunity to teach the general lesson that internal medicine is much more like a game of chess, where every move has to be contemplated with respect to its consequences several moves

down the game, than it is like checkers, where the player merely responds to his opponent's move.

Setting, Cast of Characters, and Abbreviations

The team is post-call, and Paul, the medical student, is presenting a patient who has hypokalemia on the problem list. Phaedrus, the attending; Stef, the intern; and Moni, the resident, watch on. It is clear from Paul's presentation that he sees hypokalemia as merely a number to be corrected. He fails to address what got the patient to that point in the first place and the fact that the disorder causing the hypokalemia will persist unless it is addressed.

The following abbreviations are used in this dialogue: ATPase = adenosine triphosphatase; DKA = diabetic acidosis; EKG = electrocardiogram.

Dialogue

"So problem number five is hypokalemia. We will give him 40 milliquivalents of potassium. Problem number six is…"

Phaedrus interrupts. "Paul, let me stop you there. Have we thought through why he has hypokalemia?"

"Well, not really. His potassium is 2.8, so we thought we would replace the potassium."

"Okay, what are you going to replace it with?" Phaedrus replies. Paul looks stunned. Phaedrus continues. "Do you mean replete it?"

"Yes. Replete it."

"Okay, just yanking your chain a little bit, Paul. It might seem petty, but I want you to sound sharp. From sharp-sounding people come sharp-performing physicians. And remember, sloppiness metastasizes.… But tell me, do you have an approach for diagnosing hypokalemia?"

"Well, no. I know that people who are on diuretics waste potassium. But after that, no.… no approach."

"That's okay. Let me give you one." Phaedrus draws out the picture of a nephron (Figure w-1) and points to the distal convoluted tubule. "The body handles potassium in three ways, Paul. And these three ways should formulate your approach. The first is aldosterone, which you'll remember activates the sodium-potassium ATPase pump on the proximal side of the tubule. This pumps sodium into the body from the renal tubular cells, by exchanging

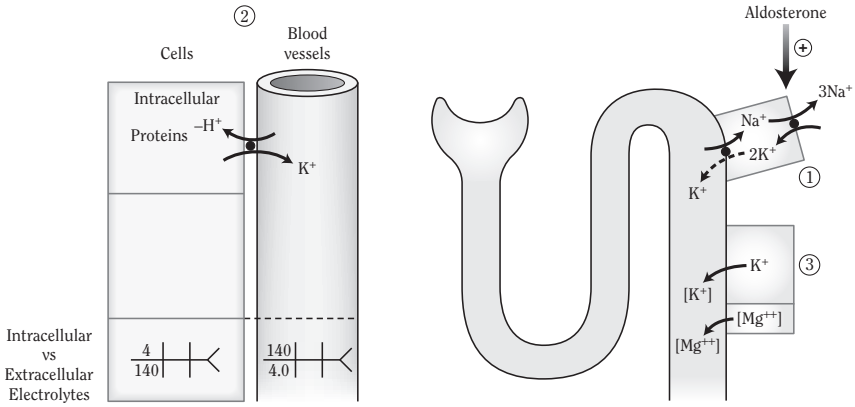

Figure w-1 The kidney and potassium management. 1 = aldosterone-mediated potassium reabsorption; 2 = intracellular shift of potassium; 3 = potassium lost in the distal tubule due to dilute urine.

it for potassium." Phaedrus draws a number "1" on the diagram in Figure w-1 and then continues. "We'll talk more about that when we get to metabolic alkalosis.… Paul, remind me to go through that with you." Paul writes it in his book. "But for now, it will suffice to say that the renal tubular cells will be loaded with potassium when the aldosterone is high … and this leads to potassium literally falling out of the cells into the urine, where it is lost from the body. So that is cause number one." Phaedrus pauses. The whole team is writing this down.

"Okay, cause number two: cellular shifts. At a later point, I'm going to talk to you about acidosis, but it's a much bigger talk than we have time for here. For now, Paul, I want you to write down these principles. I'll come back and explain them later. Principle number one: Potassium is a predominately intracellular ion, and sodium is a predominately extracellular ion. Stef, what is the normal sodium concentration in the blood?"

The first step in teaching potassium is to outline the three primary causes of potassium disorders. Students will probably focus on aldosterone but will forget the other two causes unless the method directs them to these causes.

"140," Stef replies.

"Indeed. And the normal potassium concentration in the blood is?"

"4."

"So in a yin-yang sort of way—should things balance out in the universe, I mean—what do you suppose the sodium level is inside the cells?"

"Based on the yin-yang, I think … 4?" Stef responds.

"And the potassium?" Phaedrus asks.

"140?"

"Right. Though in actuality, the numbers aren't exactly that, but it does make the point. Principle number one: potassium is an intracellular ion. Now principle number two: proteins are negatively charged."

By establishing potassium's predominant intracellular role, the attending sets up the discussion of cell shifts as a major player in potassium management.

"Huh?"

"Yeah, I know, it seems tangential. But think of it, where are most of your proteins? Floating around in the blood, or inside the cells doing things?"

"I'm guessing doing things," says Paul.

"Right, and if I suddenly dumped a lot of acid in your body, you would want to buffer that acid—those 'H-pluses,' I mean—with some negative ion, right?"

"Right," says Stef.

"So that's what happens, and that's the second cause of low potassium disorders, at least eventually. When an acidosis occurs, the hydrogen ions go into the cells to be buffered by the negatively charged proteins." Phaedrus draws the cell/blood diagram to the left of Figure w-1 and labels it "2." "But because the hydrogen ions are positively charged, they push out the also positively charged potassium ions. You've seen that, haven't you, Moni? In the setting of a patient with diabetic ketoacidosis, the initial potassium is usually…?"

"Well, initially, it's very high because of that cell shift," says Moni.

The attending involves the resident both because diabetic ketoacidosis is far above the student's level right now and because it brings the resident into the discussion. For all learners, this principle of cell shift will set up the discussion of diabetic ketoacidosis when a patient with this disease arrives.

"Right. And think of it, Paul. With the potassium concentration in the blood being very high to start, what will be the amount of potassium filtered across the glomerulus? I mean, if it's very high in the blood, the filtered amount will be…"

"Well, if there's a lot in the blood, a lot should be filtered."

"Correct, Paul. And over time, potassium is lost from the body, causing the hypokalemia."

Moni jumps back in. "Phaedrus, is that why we're told to not panic if the potassium is high in DKA, but to really panic if it's low?"

"Well, the first rule is that the EKG will tell us whether to panic or not … I'll get to that in a minute … but yes, in the setting of DKA, assuming the EKG is normal, a high potassium is not necessarily a bad thing—once we correct the acidosis, we can expect that some of that high potassium concentration will go back into the cells as the hydrogen ions leave. If it's low, then it tells you that the patient has lost a LOT of potassium and that you are going to have a lot of ground to cover to make it up. Which leads to the next principle before we address the final cause of hypokalemia." Phaedrus pauses.

"Okay, here's the rule," he continues. "As potassium is lost from the body, the body will equilibrate by shifting some intracellular potassium into the blood to keep the blood level normal. So the first phase of the loss, Paul, you will barely see. Maybe a potassium concentration that declines from 4.0 to 3.5. The serum change is little, but the cells are being robbed. In the next phase of potassium loss, more intracellular potassium is shifted from the cells to the blood, and the cells are robbed even more. You'll start to feel this drop, with a potassium level of 3.5 to 3.0. But be warned, at the 3.0 point, the cells have more or less exhausted their ability to contribute potassium to the serum to keep it normalized. Thereafter, any subsequent potassium loss from the body will lead to precipitous drops in the serum potassium, down to 2.5 or 2.0, and it is these drops that kill people."

Aside from the cell-shift phenomenon in acidosis, it is very important that learners appreciate the slow drain on intracellular potassium stores as the cause of the hypokalemia persists. Once they understand this, it is much easier to emphasize the urgency of correcting hypokalemia before it gets to the 2.0-2.5 level; once at these levels, potassium repletion becomes much more onerous and lengthy.

"Kill people, really?" asks Paul.

"Indeed, and I'll tell you why in a minute. For now, though, remember our DP/DT rule: it's not the magnitude of the value that kills people, it's how fast the value changes that kills people. And the rate of the shift, as you said, Moni, is assessed by looking at the EKG." Phaedrus pauses. He has set up the next part of the talk, but it is time to finish with the last piece of this part.

An important principle of internal medicine is the DP/DT: The rate of change over time is what is most dangerous to patients. The attending sets up the EKG as the measure of assessing this rate of change with respect to potassium, allowing him to return to it at the end of the talk.

Phaedrus continues. "Okay, so the last cause is this, and I'll tell you that this accounts for most of the serious hypokalemia cases that you will see. Paul, Stef, Moni, look at the distal collecting tubule here." Phaedrus points to the distal tubule in Figure w-1 and labels it "3." "There is a potassium concentration in the lumen, and there is a potassium concentration in the tubular cells. Now, a lot of intracellular machinery goes into this, but let's keep it simple: potassium is going to flow down its concentration gradient. If the potassium concentration is very high in the cells, or very low in the urine, potassium will move from the cells to the urine … and that's how you lose it. So, Paul, let me ask you, without changing the total ***amount*** *of potassium in the tubular cell, how could you make the potassium* ***concentration*** *in the lumen lower. Think carefully about what the denominator is for a 'concentration.'"*

Paul smiles, appreciating being helped along. "Well, I guess more water to the distal tubule would decrease the potassium concentration."

"Right! So, Paul, why is it that patients on diuretics often have hypokalemia?"

"Ah, the diuretics prevent sodium reabsorption, so more sodium, and thus more water, goes to the distal tubule."

"Indeed. As a simple rule, patients who urinate a lot, lose a lot … of potassium, that is." Phaedrus pauses. "Stef, what other patients urinate a lot?"

"Well, diabetic patients. And I suppose alcoholics?"

"Indeed. And do these patients have low potassium levels?"

Moni jumps in. "If their diabetes is out of control, they do. Especially if they have polyuria… And yes, come to think of it, alcoholics often have very low potassium levels, too."

"Correct. So just think of it this way. If a patient is urinating a lot, you need to be thinking ahead and anticipating that he might have or develop hypokalemia…" Phaedrus pauses, "… and the converse is true as well. If a patient has hypokalemia, you need to be thinking about why he is urinating a lot."

Part of potassium management is anticipating what the potassium level will be. This is the "chess player's" proactive mentality—thinking down the game and anticipating problems before they happen. The maxim of "high urination, low potassium; low urination, high potassium" enables the learners to anticipate what the potassium levels will be in their patients as they make management decisions.

"Wow. But why is this such a big deal, Phaedrus?" asks Moni. "I mean, I know that it is ... but why?"

"Great question, Moni. This is an involved topic, and at a later point, I'm going to address EKGs and arrhythmias with you. But here's the short take, which is much more at Moni's level, Paul, so if you don't get all of this, that's okay. We'll spend more time on it later." Phaedrus pauses. "Okay, Moni, you remember that the cardiac membrane potential is made possible by the sodium-potassium ATPase pumps, right?" Moni nods as Phaedrus draws out a square with a sodium-potassium ATPase pump attached to it (Figure w-2). "And that this exchanges three sodiums out of the cell for two potassiums into the cell, making it a little bit negative inside." Moni nods again as Phaedrus points to point A in Figure w-2. "But you'll also remember that the biggest part of repolarization—that is, making the cell intracellularly negative—is the potassium being pushed out of the cell by its chemical gradient: that is, the big potassium concentration in the cell pushing potassium out of the 'side door,' called the potassium rectifier channel." Phaedrus points to point B on Figure w-2.

It's fine for the student to hear this, but calling out that this is high-level content takes the pressure off of the student, enabling him to watch, but not struggle with the content so much that it confuses him.

"Yes, I do remember that," Moni says.

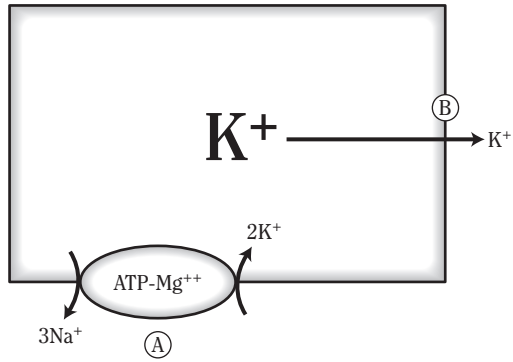

Figure w-2 Repolarization of a cell. A = The sodium-potassium adenosine triphosphatase pump pumps three sodiums out of the cell for every two it pumps in, making the cell slightly negative; B = the high intracellular concentration of potassium causes potassium to be pushed out of the cell through the potassium-rectifier channel, making the cell much more negative. ATP = adenosine triphosphate.

"Okay, when the potassium in the extracellular fluid…" Phaedrus points to the small "k" sitting outside of and to the right of the box in the figure, "…is small, we can say, based upon our previous discussion, that the intracellular potassium is really low … at least compared to its normal high intracellular value. Can you buy that for a quarter?"

"Umm, sure I'll buy that, even for a quarter."

"Okay, so I'll ask you, which cell takes longer to repolarize: a cell with a very high intracellular potassium concentration, or a cell with a very low intracellular potassium concentration?" Phaedrus places his thumb over the small "k" sitting outside of the box in Figure w-2 and continues. "Forget the extracellular potassium concentration for now … since it's small, it plays a very small role in repolarization … focus on the intracellular potassium concentration. Which cell will have the 'oomph' to push potassium out quickly? The big intracellular concentration or the low intracellular concentration?"

Learners will get confused on this point, thinking that if the extracellular potassium is low, then the cell will repolarize faster because there is less potassium on the outside to oppose the efflux of potassium from the cell to the serum. Having begun the talk with how significant hypokalemia drains the intracellular stores, the attending is able to emphasize that it is the *intracellular* potassium concentration, seriously depleted in severe hypokalemia, that leads to the long QT interval and risk for torsades de pointes.

"Well, the cell with a large intracellular potassium concentration will repolarize quicker," says Moni.

"Correct, and when on the EKG does this happen? When does repolarization happen?"

"The T wave." Moni stops cold. "Hey, is that why you get the long QT with hypokalemia?"

"Indeed, and …"

Moni interrupts, "AND the peaked T wave … a short repolarization time … with hyperkalemia?"

"Exactly. Do you see why this is a big deal now?"

"Absolutely. Thanks, this makes sense now."

"One last point, and Paul, Stef, I do want you to hear this part … at least the conclusion." Phaedrus pauses. "Moni, look again at this repolarization of the cardiac cell." Moni does as she is instructed. "What would happen if I suddenly increased the extracellular potassium to 10-fold its current level? What would happen to the efflux of potassium out of the cell?" Phaedrus replaces the small "k" on the outside of the box in Figure w-2 with a very large "K."

"Well, potassium moving out of the cardiac cell is because of its concentration gradient … so if you suddenly increased the extracellular concentration, then potassium would stop moving out of the cell," Moni replies.

"And what would that do the cell's repolarization?"

"It would stop it completely…" Moni pauses. "I get it. That's why we can't give potassium quickly."

"Not unless you want to lethally inject someone." Phaedrus takes the point and drives it home with the entire team. "Paul, Stef, did you catch Moni's point? It's critical that you do. You cannot give potassium quickly via an intravenous line. If you do, you'll bathe the heart in a sudden increase in the potassium concentration, and that will effectively stop repolarization of the heart, inducing a heart 'seizure,' if you will." Phaedrus pauses. "This, Paul, is why we take hypokalemia so seriously. If you let the body's stores drain down too low, then it is a long and arduous road to get it back, since we do not have the luxury of rapid correction. Do you see that?"

"I do now," Paul says.

The discussion of the cell shift, and of the EKG physiology, sets up the dramatic learning point: Rapid potassium delivery kills patients. By investing in the preceding discussion, the attending is afforded the luxury of explaining this concept as opposed to just admonishing the team with another rule of clinical medicine.

Phaedrus continues. "Okay, a couple of other points. First, people who urinate a lot should make you think of…"

Paul replies, "Hypokalemia."

"Right, and so people that don't urinate at all … say renal failure … should make you think of?"

"Hyperkalemia?"

"Exactly, and that's the differential for hyperkalemia—renal failure. And add on acidosis and cell damage with a sudden release of potassium into the blood."

"Wow, very helpful."

"And final point, since you have been so patient—magnesium. Magnesium is handled in exactly the same way as potassium. It's all about the concentrations of magnesium in the renal lumen and the concentrations in the renal tubular cells. So if a patient has hypokalemia from urinating a lot, and since magnesium and potassium are handled in the same way, what do you suppose the magnesium concentration in the blood will be?"

Discussions of magnesium should always be linked with discussions of potassium, not only because the body's handling of both is very similar (such that patients who have a disorder of one usually have a disorder of the other) but also because of the key management point (magnesium before potassium repletion) discussed below.

"It should be low, too, I would guess," says Paul. "Hey, Mr. Lawrence has a low magnesium as well."

"Imagine that." Phaedrus smiles. "And listen, I'll teach you about magnesium and what it does when we talk about the EKG, but for now, I want you to remember this simple rule: Anytime you see 'ATPase,' you are going to need magnesium as a cofactor. That's more or less its sole purpose in the body, to spin the ATPase pumps." Phaedrus pauses. "So, no magnesium, no pumps. Hey Stef, once you replete his potassium, how are you going to restore the intracellular potassium stores? That is, through which pump will potassium be pumped into the cells?"

"Hmm … through the sodium-potassium ATPase pumps, I guess."

"So what will happen if you don't give the magnesium first? I mean, if there is no magnesium, then those pumps won't work, right?"

"That's right. No magnesium, no pumps," Stef repeats.

"So without those pumps, what will happen to all of that potassium you are going to put into his blood?"

"I guess he would just pee it out."

"Okay, then, lesson learned. Rule one: if you see hypokalemia, suspect that hypomagnesemia will also be present, since they are both handled by the kidney in the same way. And rule two: you have to replete the magnesium first, or you will just be spinning your wheels with the potassium."

The high-level insight, not discussed here, is that when you see hypokalemia in the absence of hypomagnesemia, it is probably due to one of the other non–urinary flow causes of hypokalemia: high aldosterone or cell shifts.

Essential Components of Teaching Potassium Disorders

- Discussions of hypokalemia should begin with the pathophysiology (cell shifts, high aldosterone, and tubular loss) that causes it. This will enable the team to anticipate which patients are likely to have potassium disorders.
- Before proceeding to the arrhythmogenic risks of hypokalemia, learners should be made aware that serum hypokalemia will gradually drain the cells of their intracellular stores. This will enable them to see why very low potassium levels (2.5 vs. 3.5) take a very long time to correct, which is part of the impetus to think ahead to prevent hypokalemia. It will also enable them to understand the next part of the talk: why hypokalemia puts them at arrhythmogenic risk.
- Once learners understand that severe hypokalemia represents depleted intracellular stores, they should then be taught how the depleted intracellular potassium concentration (a smaller gradient pushing potassium out of the cells) slows the repolarization of the membrane, increasing the QT interval and increasing the arrhythmogenic risk.
- The talk should end with a brief discussion of magnesium because few learners will understand its links to potassium.

❖ Dyspnea

Purpose

Dyspnea is one of the most common diagnoses encountered on the clinical wards. It is also one of the most mismanaged; considerable mortality results from learners "shooting from the hip" in making a quick diagnosis in lieu of having a disciplined approach to its assessment. This dialogue illustrates two important points: first, an organ-by-organ approach to the problem, and second, a method that ends with emphasizing the importance of obtaining the three necessary tests for all cases of dyspnea.

Setting, Cast of Characters, and Abbreviations

The intern, Stef, and the student, Paul, are seeing a patient together on the afternoon of the call day and are about to discuss a patient with Phaedrus, the attending.

The following abbreviations are used in this dialogue: ABG = arterial blood gas; ARDS = acute respiratory distress syndrome; ATP = adenosine triphosphate; CHF = congestive heart failure; COPD = chronic obstructive pulmonary disease; CT = computed tomography; EKG = electrocardiogram; ER = emergency room; MI = myocardial infarction; Pao_2 = partial pressure of arterial oxygen; PE = pulmonary embolism.

Dialogue

"Phaedrus, I have a patient to present to you."

"All right, Paul. What do you have?"

Being around the wards on the on-call day can give a new insight into a learner's methods of approaching problem. The attending should not infringe on the team's autonomy in decision-making because early learners may use the physician's presence to defer their own decision-making.

"Well, he's a 48-year-old man with shortness of breath. I think he has a PE!"

"Really? How did you come to that conclusion?"

"Well, honestly, I can't take credit for diagnosing it. The ER doctor said it."

"Hmmm … she may be right. But in the interest of staying true to our method, let's go through it and make sure. Let me hear your method for approaching shortness of breath, that is, dyspnea. Have you seen that done before?"

It's tempting for the attending to "bash" the ER physician at this point. But the attending shows restraint in skipping past the "ER physician commentary" and focuses instead on the patients' problem. In doing so, he models good "team-based" care by not sharing any bias or skepticism about the diagnosis, and thereby demeaning a colleague, even though he has a strong suspicion that it might not be a PE. The attending further models appropriate behavior by not prematurely dismissing the diagnosis (just because he didn't make it), but also by insisting on a methodical evaluation of the problem before agreeing to the diagnosis (that is, not succumbing to the anchoring heuristic).

"Honestly, Dr. Phaedrus, I've never had a patient with dyspnea. I've heard it talked about quite a bit, but I don't really have an approach."

"That's okay, Paul. I appreciate the honesty. Let me talk you through it. Do you have a piece of paper?" Phaedrus takes the paper and draws a pyramid on the back of the sheet (Figure w-3). "Okay, I've drawn a pyramid with four levels to it. Each level represents an organ system that might, if it failed, result in dyspnea. But before we get to that, remind me of Equation 1. Remember the first step is getting oxygen from the environment to the alveoli."

Although the dyspnea pyramid will be used to organize the student's approach to dyspnea, the attending still keeps the physiologic approach to diagnosis at the forefront.

Paul thinks for a second and then begins. "Well, let's see … the PAO_2 … the oxygen in the alveoli … that is the oxygen that is

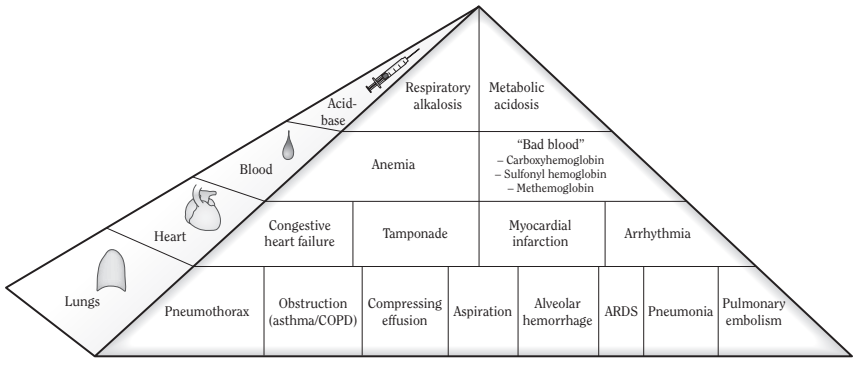

Figure w-3 The dyspnea pyramid. ARDS = acute respiratory distress syndrome; COPD = chronic obstructive pulmonary disease.

pushed in to the alveoli … the barometric pressure, minus the water vapor in the air … times the percentage of oxygen in the air." Paul draws out the first part of the equation.

"Yes … and what other 'roommate' shares that 'apartment' we call the alveoli?"

"The CO_2, which we can estimate by looking at the CO_2 in the blood. So you subtract the CO_2 in the blood times 1.25."

"Well done, Paul." Phaedrus writes out Equation 1 as Paul has laid it out. "Now, how do you tell if there is enough oxygen in the blood?"

"You get an ABG and check the Pao_2."

"Good, and what if there is insufficient oxygen in the alveoli and the blood ?"

"Then the patient isn't breathing. That's when we have to intubate the patient."

"Good. And what if there is sufficient oxygen in the alveoli, but insufficient oxygen in the blood?"

"That's the A-a gradient. That means something is disrupting the diffusion of oxygen across the membrane," Paul says proudly.

"Nice." Phaedrus responds. "And what is Equation 2? Remember that one? The diffusion of a gas across a membrane?"

"Yeah … diffusion is proportional to the pressure gradient times the area of diffusion … all of that divided by the wall thickness."

Phaedrus writes out Equation 2 as Paul speaks. "And in this case, what is the pressure gradient refer to?"

"Again, that's the A-a gradient."

"Good, so if there is a large A-a gradient, then that's not the problem, which leaves only two variables to explain why oxygen isn't diffusing across the membrane: decreased surface area or increased wall thickness. What are the causes of decreased area in the lung?" As Phaedrus speaks, he writes in the "lung" symbol at the base of the pyramid, adding in the diagnoses as Paul continues.

"Well, from outside in …"

"You're thinking methodically, Paul. I like that."

This is a method that the attending did not provide to the student, but it's a good one all the same. The attending rewards the student for developing his own rational ("outside-in") method for decreased lung area.

"… pneumothorax, a large pleural effusion, and COPD … as you taught me, due to a few alveoli compressing the others, thereby functionally decreasing area." Paul looks for approval. Phaedrus nods. "… and pneumonia."

"Great, work Paul." Phaedrus looks to Stef, who raises his eyebrows and nods his head in approval. Phaedrus continues. "I'll add alveolar hemorrhage to the list. What about increased wall thickness?"

"Well, CHF, ARDS, interstitial pneumonia, and PE?"

"PE? You sure?"

"Phaedrus, you taught me this. PE is the release of platelet-derived mediators from the small clot that causes extravasation of fluid into the interstitial space. It acts more like ARDS and shunt than it does a decrease in area."

Although the test for pulmonary embolism is a ventilation–perfusion scan, and technically it is a ventilation–perfusion mismatch, the physiology is more "shunt" than it is nonperfused alveoli (as evidenced by the decreased CO_2 and decreased O_2). Students are likely to mislabel, and eventually mistreat, pulmonary embolism unless this point is established.

"Just testing you, Paul. Thanks for paying attention." Phaedrus extends a hand for a handshake.

Blind obedience can be a real problem on the wards, with the student agreeing to everything the attending says. Intermittently, learners should be challenged to defend their statements, even when the attending knows that they are correct.

Phaedrus continues. "All right, Paul. Now that oxygen is in the blood, how does it get to the brain stem? Remember Equation 3?"

"Equation 3 … let's see … that's the one with the trucks, isn't it?"

"Indeed it is. What are the trucks?"

"Hemoglobin … and the speed of the trucks is the cardiac output … and the saturation of the trucks is the oxygen saturation."

"And we've already dealt with the causes of poor oxygen saturation in Equations 1 and 2. And the low trucks would be anemia or bad blood." Phaedrus draws the symbol for "blood" at the third level of the pyramid and completes the squares with "no blood" and "bad blood." He then continues. "What are the causes of poor cardiac output? Do you remember Equations 5 and 6?"

"Well, cardiac output is the stroke volume times the number of strokes … that is, the heart rate. That's Equation 5. And the stroke volume is how much volume comes into the heart … the preload … times the strength of the ventricle's contraction …the contractility."

Phaedrus continues to write in the diagnoses for the second level of the pyramid. "So help me fill in level two here … cardiac causes of dyspnea."

"So, CHF and MI, I guess."

"Great. Stef, anything to add?"

"Well, tamponade … since it limits the preload … and arrhythmia, since that limits contractility."

"Nice work, Stef. Good team-based play. And now for the final layer. Once the oxygen gets to the brain stem, or the tissues in general, what happens to it, Paul?"

"The cells use it to make ATP."

"Great, consuming glucose, right?" Paul nods. "And what is the byproduct of that reaction?"

"CO_2," Paul responds.

"Which we've established is the equivalent of 'acid' in the blood, right?" Paul nods—Phaedrus had taken him through the acid-base dialogue the day before. "So the top layer is acid-base disorders. Metabolic acidosis from the elevated CO_2, and of course respiratory alkalosis, since when you have respiratory alkalosis, you breathe fast and heavy … that is, dyspnea." Phaedrus pauses to let Paul take it in. "So that's your method, Paul. And of course, your history is going to narrow this list down and set some pretest probabilities. But let me give you a challenge. Let's say that the history doesn't help much in distinguishing the cases. What are the three tests that can exclude everything on this pyramid? But choose carefully … see if you can only use three tests."

This is the disciplined approach to new dyspnea, highlighting importance of diagnosing "protean" diagnoses such as pulmonary embolism by a method of deduction.

"Well, a chest x-ray."

"Absolutely." Phaedrus crosses off the diagnoses as he speaks. "So that would evaluate and potentially exclude pneumonia, ARDS, alveolar hemorrhage, pneumothorax, a large pleural effusion, CHF, and tamponade. Just remember to have your method in hand such that you think about these diagnoses as your read the chest x-ray. Otherwise, you might miss them. Okay, what else?"

"An EKG?"

"Indeed, that would help to exclude MI and arrhythmia. What else?"

"An ABG?"

"Yes. That would exclude metabolic acidosis, respiratory alkalosis, and, since the ABG comes with hemoglobin data to normalize the calculations, it will also exclude 'no blood,' that is, anemia. And 'bad blood,' that is, the carboxyhemoglobins, sulfohemoglobins, et cetera." Phaedrus pauses. "And that leaves you with COPD/asthma, which I'm sure your physical exam could detect … and…?"

"PE."

"Yes, Paul. And that's how you diagnose PE … after excluding everything else on your pyramid, you then embark on the evaluation of PE. PE is what we call one of the 'protean' diagnoses. Do you know what I mean by that?"

"No, not really."

"'Protean' means 'coming in many different shapes and sizes.' Some diagnoses, in fact most, present with a fairly regular and consistent pattern. Pneumonia, for example, usually comes with a cough, sputum production, fever, and an elevated white cell count. CHF usually comes with dyspnea, elevated neck veins, crackles, and edema. Not always, but usually." Phaedrus pauses. "Other diagnoses … the 'protean' diagnoses … usually present in irregular ways. As you continue to learn about medicine, you'll build your list of 'protean' diagnoses. But for now, let me give you the standard approach to these types of diseases. When you are considering a protean diagnosis, the best method is to 'back into' the diagnosis. Or in other words, use deductive reasoning to estab-

lish the diagnosis by excluding all of the nonprotean diagnoses first. This will take the pretest probability you had given to those diagnoses and load it onto the protean diagnosis."

The attending validates phase 2 clinical reasoning: syndromes that are linked to diseases. The discussion then moves to phase 3 by establishing that there may be more than one disease, either by typical or atypical presentation, that could cause the syndrome; hence the differential diagnosis. The phase 4 clinical reasoning will be emphasized at the conclusion of this dialogue, when the theoretical approach moves back to the actual patient, where prevalence based on age and presenting history will help rank the diagnoses.

Phaedrus continues. "With the pretest probability now loaded onto the protean diagnosis, you will now have enough rational weight to go the distance in doing the multiple tests that are usually required to establish the protean diagnosis as being present. It's just like taking a multiple-choice test. By excluding A, C, and D, you make B the answer, even if it doesn't look 'classically correct.'" Phaedrus pauses to let Paul think through it. "In this case, excluding the remainder of the pyramid before jumping to the conclusion that it is a PE can also save a life."

"Really, how?"

"This method will keep you from ordering a spiral CT on everyone, Paul. And remember, no test is noninvasive. All tests are 'preinvasive.' If you order enough of them, you'll eventually end up with a patient with the 'equivalent result,' which of course prompts the next test … which is invariably more invasive—in this case, the angiogram. And some percentage of patients who receive the angiogram will have a bad outcome. You can mitigate that risk by selectively choosing who gets the spiral CT to begin with. Plus, Paul, it's pretty dark in radiology. Have you been down there? Not exactly the place you want to have your patient who is having an MI or an arrhythmia." Phaedrus pauses again for effect. "So let me ask you, Paul. This patient of yours, have you looked at the chest x-ray, ABG, and EKG?"

"Umm, no. Not yet," Paul replies.

"Okay, then. Might be worth doing that." Stef is busy writing orders. The two start to leave, excited, but anxious to see the results. "And Paul…"

"Yes, Dr. Phaedrus?"

"Don't ever forget this method. For every patient you are called to see with dyspnea, make sure you order these three tests. If you deviate from the method, you will deviate at your peril. It's very important to me that you remember this."

"I won't forget it. I'll call you after I see those test results."

Most discussion of judicious use of testing is couched in cost savings. The attending should be attuned to the fact that novice physicians see tests as noninvasive, as evidenced by the frequent retort, "Well, what could it hurt?" They do not realize that positive test results lead to more tests. Students also fail to see the potential complications associated with patient transportation to unmonitored areas of the hospital. Although small, these are unnecessary risks if the test is not indicated.

Essential Components of Teaching Dyspnea

- The attending should anticipate that learners will see dyspnea as only hypoxia, neglecting hypercarbia and pressure in the chest as additional causes.
- The predilection will be for learners to focus solely on the lungs. Providing an anatomic approach to dyspnea (lungs, heart, blood, acid-base) allows the learner to organize his thoughts, and ensures that he will not neglect considering an organ system in his evaluation.
- The dyspnea pyramid method ensures considering all organs but also directs the learner to the key diagnostic tests that need to be ordered for all cases of dyspnea (ABG, EKG, chest x-ray).
- Linking the pathophysiology of dyspnea (Equations 1, 2, and 8) enables the learner to interpret the diagnostic tests in establishing the diagnosis.
- Pulmonary embolism is a protean diagnosis, meaning that it comes in variable presentations. As with all protean diagnoses, the best diagnostic strategy is to use deductive reasoning to exclude diagnoses that have classic presentations. If pretest probability for the protean diagnosis remains (in this case, pulmonary embolism), then the diagnostic evaluation for that diagnosis should proceed.

❖ Abdominal Pain

Purpose

Abdominal pain is a frequent presenting symptom in hospitalized patients. The cause of the abdominal pain, however, may not be evident. Thus, students must be trained to have a disciplined, organ-based approach to building a differential diagnosis. The attending should emphasize that this is especially true for patients who have unreliable localization of abdominal pain due to being immunocompromised or having neuropathy (for example, patients with HIV infection, elderly patients, diabetic patients). Because of space limitations, this dialogue addresses only the overall approach to abdominal pain, enabling learners to establish a comprehensive differential diagnosis. Over time, the attending should have in her repertoire a teaching script for addressing each of the major subcategories of abdominal pain (such as pain with diarrhea, epigastric abdominal pain, right-upper-quadrant abdominal pain).

Setting, Cast of Characters, and Abbreviations

The team is rounding as a team post-call. The attending can see by the team members' faces that the call night was not excessively intense but that there were several patients. The team capped. To be time efficient, the attending, Phaedrus, has taken the post-call rounds out of the conference room and to each patient's bedside. Moni, the resident, is leading rounds; Stef, the intern, is observing because this patient has not been assigned to him. Paul, the student, is joined by Phaedrus. The team is in the hallway just outside of the patient's room.

The following abbreviations are used in this dialogue: GERD = gastroesophageal reflux disease; MI = myocardial infarction.

Dialogue

"So, who do we have here, Paul?" Phaedrus asks.

"This is Mrs. Guidry. She's a 56-year-old woman who presented early this morning with abdominal pain…"

"Paul, let me stop you for a second. Is there any information that is sensitive … that is, sensitive enough that we can't talk about it in from of Mrs. Guidry?"

Paul looks through his notes. "Umm … I don't think so."

"Okay, then..." Phaedrus knocks on the door … and then reaches for the Purell dispenser. He enters the room as he continues to rub his hands. The remainder of the team follows suit. Phaedrus

introduces himself to Mrs. Guidry. "Hello there. I'm Dr. Phaedrus, the attending physician with your other doctors here. What is your name?"

The attending models three important patient safety and quality techniques. First, moving the discussion to the bedside instead of talking in the hall not only saves time (one discussion per patient instead of two), it also establishes a "patient-focused" discussion and ensures better compliance with the Health Insurance Portability and Accountability Act. Second, the attending washes his hands as he enters; the team follows his lead. Finally, the attending refrains from assuming that the patient is Mrs. Guidry, asking instead to have the patient introduce herself. Further, as shown below, he then asks the patient for her understanding of why she is here. This establishes that she is the correct patient. Although not critical in this case, it models the habit he wants his team to emulate with future patients; it will be critical for patients who need procedures.

"My name is Margeaux Guidry."

"And what problem has caused you to come to the hospital?"

"Well, I started having the abdominal pains."

Phaedrus is satisfied that this is the right patient. He continues. "Well, I've heard a lot about you from your doctors here. They've been working very hard to figure out what the problem is and to try to fix it. Have they been taking good care of you?"

The attending communicates to the patient and to his team that although he is the supervising physician, the patient's doctors are the residents and students. This clarifies the roles of the team to the patient, but it also preserves the patient–doctor bond with the team. This is important for maintaining team accountability and responsibility and for ensuring that the team is not hamstrung by the patient reserving information only for the attending physician.

"I know they have … they're very good," says Mrs. Guidry.

"You are about to really know," Phaedrus thinks. "Excellent. If it's okay, I am going to ask the student doctor to tell me about your case. I want you to jump in at any time if we get something wrong. Paul, maybe I could ask you to stand over there." Phaedrus positions Paul to the left side of the patient's bed, toward the head. He positions himself to the right side of the patient, halfway down the bed.

Good bedside rounds begins with the proper position of the team members and the patient. With the presenter at the head of the bed and the attending on the opposite side, the attending can observe both the presenter and the patient's face simultaneously, while still being able to examine the patient during the presentation if needed. Symbolically, the closest bond is still between the presenter and the patient, and the attending has the luxury of seeing the patient's facial expressions during the presentation. Facial expressions can be a clue that the patient disagrees with the information as presented or becomes frightened, concerned, or confused as the information is presented.

"Okay, Paul, tell me what you've learned."

Paul begins his presentation, a little nervous at first. It is clear that this is the first time he has been asked to present at the bedside. But he successfully delivers the first paragraph, characterizing the chief complaint and describing the time course. As he moves to the past medical history, it is very clear to Phaedrus that Paul doesn't have a method for abdominal pain. He has skipped the second paragraph.

Phaedrus interjects. "Mrs. Guidry, is it all right if I teach the student doctors, here?"

"Sure."

"Now, don't be alarmed, because we are going to talk in general terms here. It doesn't mean that you have any of the diagnoses or diseases I might talk about. We're just talking in general. Okay?"

The attending's dialogue captures the second and third principles of bedside rounds. The attending communicates the sacredness of the patient's bedside by asking her permission to teach the team; this is her space, not ours. Second, the attending proactively tells the patient that what will be discussed may not be directed to her case. Without this, patients may misinterpret the conversation and become unnecessarily alarmed.

"Okay," replies Mrs. Guidry.

"Paul, let me give you a method for approaching abdominal pain. Do you have a watch?"

"Yes." Paul looks at his watch to make sure.

"Does it have numbers on it … or is it one of those fancy digital calculator sort of rigs?"

"No, it's the old-fashioned type."

"Perfect. Okay, imagine the watch face superimposed on the patient's abdomen. At each hour of the clock, I want you to think about the organ that might be located at that spot on the abdomen. That has to be your method, Paul. Abdominal pain can be sneaky. So you have to be methodical in your approach. Organ-by-organ is how I want you to approach it." Phaedrus takes the time to draw out a picture of a clock face overlaid on the abdomen (Figure w-4). As he finishes, he looks again at Paul. "This is very important to me, Paul."

Nothing is as powerful in emphasizing the importance of a method as making the admonishment personal. The attending provides a rationale (that is, "abdominal pain can be very sneaky") to ensure that the personal admonishment does not come off as merely a style preference.

"Okay," Paul replies.

"All right. Start at 12 noon. What organs are at the epigastrium?" Phaedrus points to the patient's abdomen to keep the method patient-focused. This is how Phaedrus would want Paul to see it later.

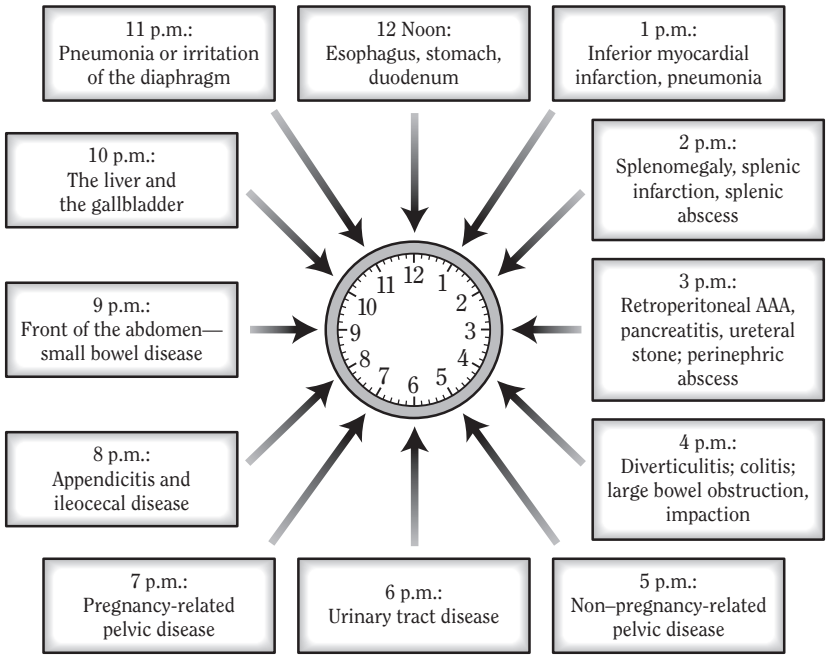

Figure w-4 The abdominal pain clock (…it's ticking). AAA = abdominal aortic aneurysm.

"Well, the esophagus and the stomach."

Moni adds, "And the duodenum."

"Correct. So I'll give you the short list of diagnoses to consider, but I want you to read about these later. Esophageal rupture, esophagitis, GERD, gastritis, gastric ulcer, gastric cancer if obstructive or erosive, and gastroparesis. For the duodenum, it's duodenitis and duodenal perforation. Paul, where does the word 'duo-denum' come from?" Phaedrus emphasizes the break in the word.

"No idea," Paul replies.

"The German word is 'Zwolf-finger-darm'... twelve fingers long … and that's how long it is … twelve fingers long." Phaedrus pauses; clearly this is cooler to him that Paul finds it. "Well, it's not really that important, but the 'twelve' will help you remember to think of it at 12 p.m. on your method. Okay, how about 1 p.m.?"

"The spleen?"

"Well, too early for the spleen just yet. At 1 p.m., the first thing I want you think about is myocardial infarction, especially the referred pain via the vagus nerve of an interior MI. Without the method, you'll miss it, Paul." Paul nods. "All right, Paul … Now 2 p.m., what do you say?"

This is an important point. Without a disciplined method, novice physicians will not think of supradiaphragmatic causes of abdominal pain. To a lesser extent, this is also true of retroperitoneal causes of abdominal pain (see the following discussion of 3 p.m.).

"I'll go with splenomegaly again."

"And this time you are correct. I'll lay out the causes of splenomegaly later, but for now, I want your mind to default to the 'two' default causes of splenomegaly: leukemia and lymphoma." Phaedrus pauses. "Now, Mrs. Guidry, I'm positive you don't have leukemia or lymphoma … this discussion is just to prepare the doctors for the patients that they will someday see who do."

"That's okay. I'm kind of enjoying this," says Mrs. Guidry.

While bedside rounds seem invasive to the patient, most patients will agree with Mrs. Guidry. Most patients enjoy bedside rounds, appreciating

the "intellectual capital" that is being invested in their care and the honest discussion that involves them.

"Me too." Phaedrus acknowledges. "All right, Paul, since you got the last one correct, I'll give you the next one for free. At 3 p.m., I want you to think of the three organs that are retroperitoneal: the pancreas, the kidneys, and the aorta. Without this mental stop at 3 p.m., Paul, you'll miss the aortic aneurysm, I promise you." Phaedrus pauses to emphasize the point. "So stop and think at 3 p.m. about pancreatitis, pyelonephritis, renal stones, and the aortic aneurysm. Okay, on to 4 p.m. ... what do you think? Stef, how about you?"

"Well, the colon, I guess." Stef responds. Following Phaedrus's earlier exam of starting with the organ and then elucidating a differential, he continues. "It could be colitis due to infection, Crohn's, or ulcerative colitis."

"Great approach, Stef. It's also worth thinking about diverticulitis, volvulus, and impaction. So colitis plus these three makes four diseases to think about at 4 p.m." Phaedrus pauses for effect. "How about 5 p.m.?"

Stef continues, "Well, that's in the pelvis, so in a woman, I would think pelvic inflammatory disease."

"Great work. At 5 p.m., I want you to think of the five causes of nonpregnancy pelvic disease. Mentally work backward from the uterus to the pelvic cavity. So… endometritis, salpingitis, tuboovarian abscess, ovarian cyst/rupture, and then pelvic inflammatory disease. How about 6 p.m.?"

Paul jumps back in. "Urinary tract disease."

"Great. So working from the tip of the urethra backwards: urethritis, prostatitis, cystitis, orchitis, and add on the conduits … hernias." Phaedrus pauses again. "Now, 7 p.m.?"

Even within each "hour of the clock" are mini-methods to build the differential diagnosis. In this example, the methods are anatomic (for example, "starting at the urethra and working backward").

"Appendicitis?" asks Paul.

"Well, that's on the list, but a bit early for appendicitis. Think about the equivalent of 5 p.m."

"5 p.m. … hmmm…" Paul continues to think.

Phaedrus takes the opportunity to reinvolve the patient, "Mrs. Guidry, are you doing okay?"

"Oh, I'm fine. They are doing such a good job."

"Yes, they are. If you want us to stop just let us know."

"I'm fine. Please continue," says Mrs. Guidry.

The attending intermittently involves the patient in the discussion, lest the team forget that she is still there. This also allows the patient the opportunity to interrupt the discussion should she feel ill or tired or otherwise wishes to see it come to a close.

By this point, Paul has found the answer. "I know now … the pregnancy-related causes of pelvic pain. So … intrauterine pregnancy, I guess, and of course ectopic pregnancy."

"Well done, Paul. Both 5 p.m. and 7 p.m. should remind you of the importance of doing a pelvic exam on all women with abdominal pain. If a full pelvic exam cannot be performed, then at least a bimanual examination." Phaedrus pauses. "Okay now, Paul … appendicitis at 8 p.m., but add in the other causes of ileocecal disease: Crohn's and ulcerative colitis, but also the causes that mimic appendicitis. Moni, this is probably at your level, but remember mesenteric adenitis … that is, infections that get into the lymph nodes of the mesentery. The specifics you can read about, Paul. The big point is to remember to think of the right colon and the appendix. So how about 9 p.m.?"

"I have no idea."

"An honest answer, Paul. I appreciate that. It's okay not to know … just not okay to continue to not to know. Well, at 3 p.m. we swung our focus to the back of the abdomen, the retroperitoneum. On the other side of the clock at 9 p.m., we'll swing our focus to the front of the abdomen, namely, small bowel that overlies all of the organs. In particular, think of ischemic bowel, a Meckel's diverticulum, Crohn's again, and a small bowel obstruction. Especially if the patient has had prior surgery on the abdomen or is at the age where there is a reasonable risk for an intraperitoneal cancer."

"Why is that?"

"Remember that a small bowel obstruction needs something about which to twist … a fulcrum if you will. Usually this is either a cancer metastasis that has lodged on the small bowel wall or a fibrous adhesion from a previous surgery. All right… now for 10 p.m. What do you think, Stef?"

"The liver and the gallbladder … so I would think of hepatitis, cholecystitis or choledocholithiasis, and cholangitis."

"Fantastic. We'll talk more later about all of the causes of liver disease and gall bladder disease. How about 11 p.m.? Moni, this might be one for you."

"I would guess the equivalent of the referred heart pain at 1 p.m. … so pneumonia or irritation of the diaphragm, such as with pleuritis."

"Well done, Moni. And even though we are using '11 o'clock,' that is, the right diaphragm, to remind us of supra-diaphragmatic causes of abdominal pain, remember that a pneumonia on the left side could cause it as well."

"Wow, that's a pretty good method," says Paul.

"I agree," says Mrs. Guidry.

Phaedrus laughs. "Okay, four other rules before I give you the clincher on this method. First, remember that abdominal pain from a 'solid' organ, such as the pancreas, the liver, or the spleen, will be constant, since the stretch of the organ's capsule is what causes the pain, and the stretch is constant. Pain from an obstructed hollow tube, otherwise known as a viscous, will be intermittent, as the pain only occurs when the muscular tube squeezes against the obstruction. So the pain of a bowel obstruction or renal stone, for example, will be intermittent … that is, colic. Second rule: Abdominal pain, especially for hollow organs, begins at the embryologic origin of the affected organ. Hence, small bowel disease such as appendicitis begins at the umbilicus … your 'small bowel' nutrients as a fetus came from the umbilical cord. The large bowel's origin is in the lower pelvis, hence we talk about large bowel at 4 and 8 p.m. on our method, even though the large bowel overlies the abdomen at the midpoint. Once the affected organ begins to rub against neighboring structures, the pain localizes to its actual location. Hence, the pain of appendicitis begins at the umbilicus and then moves to the right lower quadrant.

"Okay, rule number 3 … an obstructed tube begins with anorexia and nausea, then vomiting, then the pain. See the anorexia and nausea as a built-in mechanism by which the body is preventing further material—that is, food—from entering the abdomen to make the obstruction worse. And finally, rule number four … peritonitis. Once a tube has ruptured, spilling the bacterial contents in the abdomen, the peritoneal lining will become inflamed. I'm sure you remember the peritoneal signs from your physical diagnosis course, but if you don't, let me know, and we'll go through them together. Just remember the image of the patient lying perfectly still so as to prevent further irritation of an already irritated peritoneal lining." Phaedrus pauses for effect. "And now for the clincher of the method. Paul, are you ready?"

"Yeah."

"Me too," says Mrs. Guidry.

Phaedrus smiles. "Once the abdominal pain starts … especially if it is peritonitis … the clock is ticking … either you discover the cause and fix it, or the time expires on the patient. It is a medical urgency that you have to take seriously."

"Oh dear," sighs Mrs. Guidry.

"Don't worry, Mrs. Guidry. You're in good hands with this group of doctors, and I think I know what is causing your pain."

"You do?" asks Paul.

"I think so. Mrs. Guidry, I understand that the pain is right here at the top of your abdomen, and that it is a burning pain. Is that right?" Phaedrus touches the epigastrium lightly; there is no guarding.

"Yes, that's about it."

"And you've had one episode of vomiting, but no blood in the vomitus. Is that correct?"

"Yes."

"And I see that your joints are very swollen, particularly the knees and the tips of your fingers and thumb. Do you have osteoarthritis?"

The attending teaches the importance of using the full physical examination to evaluate the cause of the abdominal pain.

"Yes, I've had it for years. Something fierce of late … the weather change, I think. I've been taking lots of my pain pills, but nothing seems to work."

"Is the pain pill ibuprofen?"

"Yes, I believe they are. I take 10 or so a day, but nothing works."

Moni rolls her eyes and shrugs her shoulders as she looks at Phaedrus.

"So one last question. Have you noticed any blood in the stool or black stools?"

"No. None of that."

With the diagnosis reasonably well established, the attending teaches the value of using the history and physical examination to assess the next level of clinical reasoning: assessing the severity of the diagnosis (bleeding ulcer versus gastritis). This dialogue is not outlined here, but will clearly be the subject of the team's questions as they leave the patient's room.

Phaedrus looks at the patient's conjunctiva. "Looks pink—that's a good sign. Okay, Mrs. Guidry, I think that all of your pain pills have caused some irritation of your stomach lining. It could be an ulcer. If it's okay with you, I'm going to have the gastroenterology specialists come and talk with you about looking at the inside of your stomach just to make sure that it isn't an ulcer that needs more attention. We'll get you some medicine to help with both the stomach pain, as well as the joint pain. Can I come back and talk with your later?"

"Sure."

"Is there anything I can do for you right now?"

"No, I think that's about it for now."

Phaedrus begins to leave the room, and then pauses to ask, "Do you want the TV on or off?"

"You can leave it off, that's okay," replies Mrs. Guidry.

"Okay, Mrs. Guidry, I'll see you later on today."

The attending models the behavior of staying patient-focused. The diagnosis was important, but so was treating the patient's pain. The attending also models the behavior of assuring a second visit during the day; the practice is assuring to the patient, but also time saving in that it relieves the pressure, put on the patient to try to get all questions in during the morning visit, that inspires patient verboseness. Finally, the attending ends

the bedside rounds by giving the patient a choice. Although it is a small choice (having the TV on or off), it shows respect, conveys that the patient is in charge of her space, and conveys a sense of empowerment to the patient. Psychologically, one of the toughest parts of being hospitalized is losing control over one's environment. The physician can lessen this burden by re-establishing a sense of control and, in doing so, models a valuable lesson for his team. Once the team reaches the hallway, Phaedrus will make this teaching point.

Essential Components of Teaching Abdominal Pain

- The approach to abdominal pain requires a systematic method. The referral patterns of abdominal pain, and the proximity of the organs, makes diagnosis based on the history and examination alone difficult. It does not matter if the learner uses the preceding "clock" method or a similar method, as long as he uses a method that ensures a systematic consideration of each organ in the body.
- The attending should emphasize that at its core, diagnosing abdominal pain is like diagnosing a disease that is in "a big black box" (that is, the abdomen). The learner has to be methodical in considering all diagnoses, at least initially, before jumping to a diagnosis. The "clock" advanced organizer helps the student establish this methodical approach.
- The teaching script should include the general patterns of abdominal pain (for example, hollow tubes = colic; solid organs = constant pain), and should include supradiaphragmatic causes of abdominal pain (such as heart and lungs).
- While not addressed here, the attending should have teaching scripts for approaching each of the "hours of the clock" subsections (such as how to approach right-upper-quadrant pain).

❖ Weakness

Purpose

Weakness is one of the most common clinical symptoms encountered in the hospital, varying from weakness that prompts a decision for admission to new-onset weakness that develops in the hospital. The weakness may be focal (usually neurologic in origin) or diffuse (usually metabolic or primary muscle disease).

Yet for the number of times that it occurs, few students and residents have an organized method for approaching weakness. Without a method,

students are prone to premature closure, locking onto the first diagnosis that seems close to fitting the clinical presentation, without systematically considering other diagnoses. The following teaching script outlines one sample method.

Setting, Cast of Characters, and Abbreviations

The clinical setting for this dialogue is post-call. The student, Paul, has approached the attending physician, Phaedrus, to present his patient.

The following abbreviations are used in this dialogue: ALS = amyotrophic lateral sclerosis; ATPase = adenosine triphosphatase; CT = computed tomography; MRI = magnetic resonance imaging.

Dialogue

"Dr. Phaedrus, I have a patient to present to you."

"Great…" Phaedrus prepares his data cards to record the information as Paul speaks. "Okay, tell me about him."

"Well, the patient is a 68-year-old man who presents with diffuse weakness. He was well until1 week ago, when he began to experience weakness while walking. The weakness has been constant but has steadily progressed over the last 7 days. It is not associated with pain, but just a feeling of 'I feel weak in my legs.' Today on the way to his front door, he slumped to his knees and could not stand up. He did not pass out, however. He then called 911 and they brought him to the emergency department." Paul is silent for a few seconds. He flips through his papers. "His exam seemed normal…"

"Paul, let me stop you for just a minute. Do you have a method for approaching weakness?"

"Well, no, not really."

"Okay, let me see if I can give you one sample method. You don't have to use this method, but at least you'll have a method to start, and then you can either build upon it, or you can find another method. Either way is okay, but you have to have a method for approaching each problem."

Listening to the presentation, it is clear that the student has mastered the first part of obtaining a history and delivering an oral case presentation. The first paragraph contains all of the "FAR COLDER" components (see chapter 4 of this book), but the quick jump from the characterization/time course of the chief complaint (paragraph 1) to the past medical history

indicates that the student is stuck in the "reporter" mode. The skipping past the part of the history where questions would have been targeted to evaluate the differential diagnosis being considered (paragraph 2) suggests the lack of "interpretation" of the information. The most common cause for failing to build an early differential diagnosis is the failure to have a method for approaching the problem. Phaedrus does the right thing by stopping the student's presentation and asking Paul if he has a method.

"Do you watch gymnastics, Paul?"

"Umm, during Olympic years, I guess."

"Well, perhaps you've seen what the gymnasts call the 'iron cross,' where a gymnast on the rings has to support his weight by holding his arms straight out to the side. Have you seen that?"

"Yeah, I've seen that."

"Well, we can agree that that is the furthest thing from weakness. So what I'm going to propose is that you think about that cross when you think about weakness. Here, let me draw it out for you." Phaedrus draws out Figure w-5. "Okay, look at the left bar. Ischemic muscles are weak; nonischemic muscles are strong. What differentiates an ischemic muscle from a nonischemic muscle?"

"Oxygen," Paul notes as he smiles.

The questioning in this exchange is following the Socratic method. The questions are asked not to test the student's knowledge (that is, to see whether he comes up with the answer or not) but rather to lead the student along a line of reasoning. The "ischemic muscles are weak, nonischemic muscles are strong" introduction ensures that the student will make the connection between a lack of oxygen and weak muscles. This sets up the first quarter of the iron cross method.

"Okay, great. So you'll recognize Equation 3 from the 10 Equations when I write it out." Phaedrus writes out Equation 3 on the left bar of the iron cross. "What is this equation?"

"That's the delivery of oxygen equation," Paul notes.

"Exactly, and if the three components in that equation were compromised, would the muscle get oxygen?"

"No. So, it would be weak."

"Excellent. You are a quarter of the way home. After we are through here, I'm going to ask you to build a differential diagnosis for your

patient's weakness, so I want you to remember the three things in that Equation 3: oxygen saturation, cardiac output, and hemoglobin concentration. But before we move on, let me ask you this. If a patient had weakness due to one of these three systemic causes, would you expect the weakness to be focal (for example, just the right arm and the right leg)? Or diffuse, that is, all extremities?"

Paul smiles again. "Well, if they were systemic, all muscles would be weak."

"Exactly. Nice work. Let's keep going with the iron cross method."

The way in which a question is asked is as important as the question itself. The voice inflection that emphasizes the word *systemic* gives the answer away, which is what Phaedrus wants. He wants the student to feel empowered in making the connection between oxygen/hemoglobin/cardiac output and "systemic weakness." This question will set up the teachable

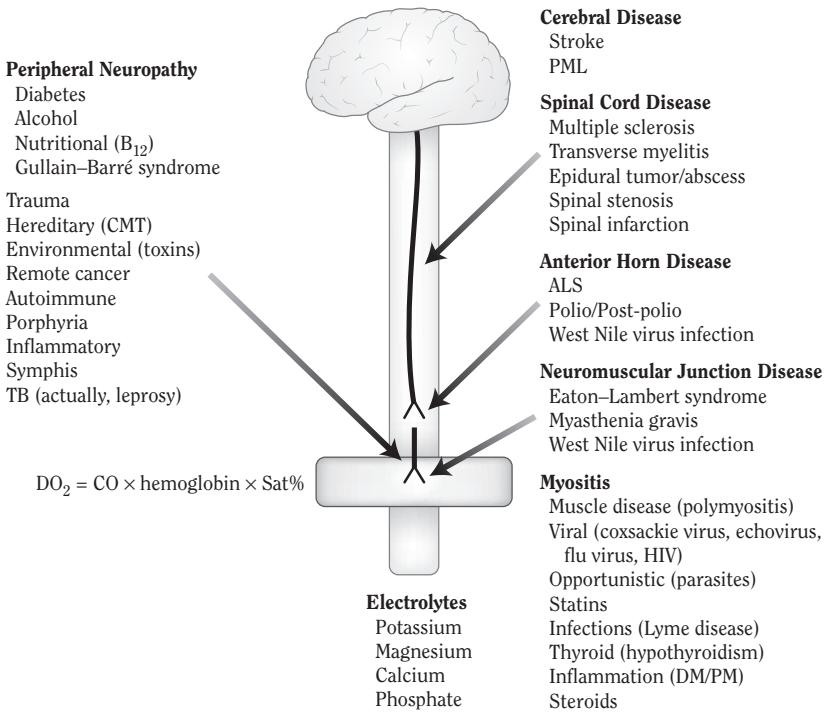

Figure w-5 The iron cross of weakness. ALS = amyotrophic lateral sclerosis; CMT = Charcot-Marie-Tooth; CO = cardiac output; DM/PM = dermatomyositis/polymyositis; DO_2 = oxygen delivery; PML = progressive multifocal leukoencephalopathy; TB = tuberculosis.

moment in using the pattern of the weakness to distinguish between the four bars of the iron cross at the conclusion of the lecture.

"Paul, I think we can agree that a muscle that can't contract is weak. Is that right?" Paul nods. "So what causes a muscle to contract?"

"A nerve stimulates it."

"Okay, we'll come to that in a moment, but let me first ask you this question. When the nerve does stimulate it, what causes the muscle to contract?"

The attending keeps the order of the method intact by keeping the student on track in sequentially considering each of the four components of the iron cross. Allowing the student to jump to the most obvious bar (that is, the top bar, nerve dysfunction) too early will prevent him from sequentially considering each of the four components later.

"Well, if I remember correctly, calcium moves into the cell and that causes it to contract," says Paul.

"That's right. But what causes the calcium to move into the cell?"

Paul looks puzzled. Phaedrus waits 5 seconds and then continues.

"Okay, let me walk you through this. Remember that the cell has to be polarized, which means that the inside of the cell is more negative than the outside. Once it is polarized, the cell is ready to entertain the stimulus. Once it is stimulated, channels open, and calcium—which is positively charged—rushes in. As you noted, Paul, this calcium is what causes the cell to contract. But here's the question." Phaedrus pauses. "What causes the cell to be polarized—that is, what causes it to be more negative on the inside versus the outside?"

Paul begins to speak, but stops.

"I think you know this, Paul. Just take a stab at it."

"Um, okay … Well, the sodium-potassium pump?"

"That's it! Well done, Paul. But let me add one adjective to your answer. How about the sodium-potassium ATPase pump? As a pump, it does, after all, require energy."

"I do remember that. The sodium-potassium, ATPase pump."

"Okay, now I'm going to ask you, Paul—and before you answer this, I want you to remember that disorders of sodium are not dis-

orders of sodium at all … they are disorders of water—based upon what you have told me, what electrolytes might be important for maintaining strength?"

It would be easier to just list the electrolytes that cause weakness, but the effect would be short-lived: The student would merely memorize the list using short-term memory and then forget it later. The attending takes the time to walk through the action potential of a muscle. This will be important later in enabling the student to recreate the information. This one-time investment will be important in facilitating an easier understanding of the cardiac electropotential and thus an easier time in teaching the electrocardiogram (see the following dialogue).

Phaedrus also spurs a wrong answer (sodium) even before the student can integrate it into his thinking. Remember, the way that a student lays down the knowledge in his brain is the way it will be recalled, especially when reasoning is being used to lay that foundation.

"Well, potassium, and calcium … and…"

"How about that ATPase?" Phaedrus asks, with a few extra decibels on the "P."

"Phosphate!" Paul responds.

"That's it … and you may or may not know this, but remember that every ATPase pump needs magnesium as a cofactor. So look at that bottom bar on our 'iron cross' of weakness. This is to remind you to think of the electrolytes important for muscle contraction. By your list: that is, potassium, calcium, phosphate, and magnesium."

Paul interjects, "You know, I think he's taking a diuretic. So he probably has low potassium. We can check some electrolytes, and…"

"Great, Paul, now you're thinking. But let me stop you before we go there, because I don't want you to jump to one diagnosis before we have finished the whole method." Paul nods in acceptance.

The student's enthusiasm is a good sign, but the enthusiasm is getting the best of him, leading to "premature closure." It's tempting to let the student go, but after recognizing and encouraging the enthusiasm, Phaedrus redirects the student to the importance of considering the whole method before jumping to a conclusion.

"Okay, on the far right bar … Let me ask you this, Paul. Will damaged muscles contract with as much force as nondamaged muscles?"

"Of course not."

"Okay, I don't expect you to know this, but I'm going to give you a mnemonic to remember some of the causes of muscle damage. This is just to hold you over until you better know these diseases. The mnemonic is MYOSITIS." Phaedrus writes out the mnemonic in Figure w-5, and Paul begins to copy the list.

"Don't worry, Paul. When we are done here, I'm going to give this paper to you." Phaedrus pauses. "Now, let me ask you this, Paul. For all three of these bars, would you expect these systemic diseases to cause weakness in only one part of the body, or all parts of the body?"

"All parts, I suppose."

"Well, done, Paul. The truth of the matter is that there are some exceptions to that, and I'll tell you about those later. But as a general rule, the systemic diseases that comprise these three bars cause systemic or general weakness."

The attending's line of questioning is truly Socratic. Instead of quizzing the student on what he knows and doesn't know, he asks a series of leading questions (that is, "systemic" and "all"), and in doing so, he creates a thought process in the student's mind that consolidates the association of "systemic diseases and diffuse weakness." The attending is quick to note that there are exceptions, but emphasizes that the exceptions will be easier to remember if the general rules are understood first.

"So, Paul, let's see what you remember about neuroanatomy."

Paul looks flushed. Phaedrus intervenes.

"Okay, let me ask you a series of questions. When you decide to move your finger, where does the neural impulse begin?"

"In the brain … umm, in the motor cortex."

"Good, and where do those nerves go next?"

"To the spinal cord," Paul replies.

"Great. You're on a roll. Do you remember where those spinal cord nerves end?"

"Umm, no."

"All right, they end in the anterior horn."

"Yes, I remember that. And then they go to the peripheral nerve."

"Absolutely, and the peripheral nerve ends at the neuromuscular junction. So, Paul, I want you to start at the neuromuscular junction and work your way up until you get back to the brain. So let's start with the neuromuscular junction. Paul, let me ask you this. Do you like college basketball?"

"Sure. Duke's my favorite team."

"Great. Well, let me tell you what I know about college basketball teams. There are teams like your favorite, Duke, that have a deep roster of great players, and then there are smaller college teams, like, say, Gonzaga, that have a starting five players that are great, but a much thinner roster of backup players. So let me ask you this. When a player fouls out of the game, which of these two types of teams feels the hit worse? The big college team that has several equally great players sitting on the bench or the smaller college team that doesn't have as many great players sitting on their bench?"

"Well, the smaller college team of course."

"Okay, so which of the following two muscles will feel the effect—that is, show signs of weakness—when neuromuscular junctions start to 'foul out.' Big muscles like the arms and legs, or small muscles, like the extraocular muscles?"

"I would guess the small muscles." Paul pauses and thinks about it. "Wow, so maybe that explains why people with myasthenia gravis have double vision. I learned that on neurology, but I didn't know why."

"That's it, Paul. There are two big diseases that cause neuromuscular junction disease, Eaton-Lambert and myasthenia gravis. The presenting feature with both is weakness with extraocular muscle dysfunction. And that's what you need to know about these two diseases." Phaedrus pauses. "But one last question, will there be sensory abnormalities if only the motor neuromuscular junctions are diseased?

"Well, no. Sensory function should be normal."

"Great. Remember that … for neuromuscular disease, small muscles are affected, with no sensory deficits. What I'm going to show you as we go up this neuropathway is how we can use historical questions and the physical examination to give us clues as to where the damage in the system took place."

Once again, it would be easier just to list the symptoms that are associated with neuromuscular junction disease, but the attending takes the time to find an analogy that will help the student understand *why* the disease causes the symptoms. This will be important for improving recall later. In this case, Phaedrus had the advantage, via some of the small talk that started the attending month, to know that Paul liked basketball. This made the analogy effective; a different analogy might have been chosen depending on a different student's interests.

"Okay, working backwards from the neuromuscular junction, what is next?"

"The peripheral nerve."

"Yes. And the peripheral nerve carries nerve impulses that are motor, sensory, or both?" Phaedrus asks.

"Both," Paul replies.

"So what physical abnormalities might you expect to find on the examination?"

"Sensory deficits and motor deficits," Paul answers.

"Absolutely. Now, I'll tell you that sometimes it is one or the other, depending upon the disease. On a different day, we'll devote time to understanding how the diseases that cause peripheral neuropathy do so, and what the exceptions are. I want to make sure that we get through this full method, so for now, here is another mnemonic to help you remember the causes of peripheral neuropathy [Figure w-5] until we have time to show how each disease causes the neuropathy. But for now, I want you to think of peripheral neuropathy as presenting with both sensory and motor deficits. Because both of these impulses are necessary for a reflex, we can expect that a peripheral neuropathy will have decreased or absent reflexes."

The attending exercises discipline in choosing to give the mnemonic for peripheral neuropathy, knowing that he has limited time and that this could be a major tangent that would prevent the student from having a full

perspective of the method. Remember, a student, like a person carrying an armload of packages, can handle only so much in one sitting. After that, things start to get dropped.

"Okay, Paul. You told me that the peripheral nerve—at least the motor component, since we are talking about weakness—originates at the anterior horn." Phaedrus pauses. "The nice part of anterior horn disease is that there are only two or three diseases that cause it. Do you know what those are?"

"No, I'm sorry, I don't."

"That's okay, Paul. It's okay not to know, it's just not okay to continue to not know. So I'll tell you that they are polio, ALS, and West Nile myelopathy. Later on, I'm sure we'll have the occasion to talk about these diseases, but in the event that we don't, I want you to read about these diseases. For now, I'll ask you this question. Is there any sensory component to the anterior horns?"

"No, they are pure motor."

"Absolutely. So should we expect any sensory deficits if the weakness is due to anterior horn disease?"

"No," Paul says.

"That's right. And since the anterior horns are the connecting node that enables the reflex arc, what should we expect the reflexes to be?"

"They should be absent."

"Perfect, Paul. So remember this: anterior horn disease presents with loss of reflexes with preserved sensory function." Phaedrus pauses as Paul writes the axiom in his book. "Okay, before we move up the spinal cord, let me ask you a few questions to see if 'learning took place here.' What are the physical findings we would expect to see with neuromuscular disease?"

"Well, small muscles will be affected more, so cranial nerve dysfunction like diplopia and trouble swallowing.… Oh, and sensory function will be preserved."

"Great, Paul. How about peripheral nerve disease?"

"Well, since it should affect both sensory and motor fibers, there should be loss of both …with a loss of reflexes. But you did say there were some exceptions to that."

"Yes, I did, but that's what I want you to know for now." Phaedrus pauses. "How about anterior horn disease?"

"Just the motor function will be lost … and the reflexes, of course."

The attending takes the time to do some feedback and evaluation of his teaching. This is a nicely chosen break because it groups all of the "lower motor neuron diseases" together. The student answers the questions correctly, giving the attending the feedback that he needs: assurance that the student has mastered the teaching to this point. Only with this assurance can Phaedrus continue on to the next level. Failure to answer these questions correctly would require that Phaedrus return to this material and reconsolidate the teaching until the student can demonstrate competency. This also sends a hidden message to the student going forward; it will be important to pay attention and invest energy in learning it now, for failure to do so will stop the lecture until he has done so.

"Great. Paul, you are really picking this up. Just a couple more steps. Before we talk about the spinal cord, I want to remind you that the spinal cord carries motor fibers from the brain down to the anterior horns, but it also carries inhibitory fibers that help regulate the sensory input … from the peripheral sensory fibers… into the anterior horn. That's what helps the body regulate its fine motor responses. Do you remember that?"

"Sure."

"Okay, so what would happen if you cut the spinal cord?"

"Well, you would lose the motor fibers, so the patient would be weak below the cut in the cord."

"Yes, what about those inhibitory fibers?"

"You would lose those, too. So I suppose whatever input came into the cord to the anterior horn would be unregulated."

"And what would the reflexes be?"

"The reflexes would be hyperactive."

"Great, Paul. I want you to remember that. Weakness, with increased reflexes … that's the definition of a spinal cord disease. Indeed, it's the definition of both cerebral and brain cord disease. This is why we lump these two diseases together as 'upper motor

neuron diseases.' Phaedrus pauses. "Do you have this? Upper motor neuron diseases are those with weakness with increased reflexes."

"Got it. Upper motor neuron diseases are those with weakness with increased reflexes."

"Again, we'll talk in greater detail about the specific diseases that cause spinal cord and cerebral disease, but the most likely causes are stroke, multiple sclerosis, metastatic disease to the spine, and spinal cord abscesses. There are many others, but these are the big ones."

In true Socratic fashion, the attending gives the student the information he needs; he hands the student the necessary information regarding inhibitory neurons in the spine before teaching the signature physical findings that go with the disease.

"Okay, Paul, here's what I would like for you to do. Go back to your patient, and reinterview and examine him. But before you do so, let me ask you this. What is your differential for weakness?"

The attending ends the session by consolidating, asking questions to evaluate the student's understanding of the method.

"Well, if it's diffuse weakness, it could be due to oxygen delivery to the muscles, such as anemia, hypoxia, or heart failure."

"Good. And what questions would you ask to evaluate these diagnoses?"

"I would ask if he has had bleeding or blood in his stools. I would ask if he has a history of lung disease, if he smokes, and whether he has been short of breath … and I would ask if he has had a history of heart disease, heart attacks, chest pain, or signs of heart failure, such as shortness of breath or edema."

"Nice work, Paul. What else is on your differential diagnosis?"

"Well, it could be electrolyte abnormalities."

"What would you ask?" Phaedrus replies.

"Well, I would ask about things that might cause loss of these electrolytes, such as diet, diuretics, vomiting, diarrhea, or a history of kidney disease."

"Good work, Paul. What else?"

"Well, there are causes of muscle disease. I don't know much about those yet, but I guess I would ask if he had muscle pains, was taking any medications, or had a history of thyroid disease."

"Nice, Paul. We'll discuss those in greater detail later. But as you learn about each of those myositis diseases, you'll know what to look for as you read … you'll be thinking about what signs and symptoms suggest each."

"Yeah, I'm going to read about those tonight."

"Great, just what I hoped. Hey, how about if the weakness was focal and not diffuse?" Phaedrus asks.

"Well, I would think it would be more likely that it was neurologic in origin. I would ask about diplopia and swallowing difficulties to see if it were neuromuscular disease. I would probably just go through the list on the DANG THERAPIST list and ask what I know about those diseases … I'm not sure I know what I would ask about the anterior horn or spinal cord disease. I would have to read more. But I could do an examination to get some clues."

"Now you're on to it Paul. So tell me, what will you do on that physical examination?"

"Well, I'll listen to the lungs and to the heart to make sure that it's not the left bar. I'll then see if the weakness is in part of the body or the whole body."

"Why are you going to do that?" Phaedrus asks.

"Because if it's diffuse, then it makes it more likely that it's the bottom three bars on the cross, and not neurologic in origin."

"Great. What else on the examination will you do?"

"Well, I'll test his cranial nerves … and his motor strength …and his sensory function …and his reflexes."

"I think you've got it, Paul. We'll talk about the laboratory values when you are finished."

"I guess it will depend upon what I find, but I think I already know what I'll need to order."

"Really? What is that?"

"Well, on the left bar, I'll want to know the oxygen saturation and the hemoglobin. If there are signs of heart failure, perhaps an

echocardiogram. I'll probably want to order electrolytes to see the potassium, magnesium, calcium, and phosphate. And on the right bar, probably a creatine kinase level."

"Hmm, anything else?"

"Well, if it's focal weakness, it's probably the top bar. So either a CT or MRI of the brain or spinal cord, and maybe a nerve conduction study."

"Nicely, done. Paul. I knew there was a great physician in there somewhere. Let me know what you diagnose."

No doubt, there were multiple times during this series when the attending became frustrated with the student's lack of knowledge. But the attending held the frustration to himself, exercising patience and looking for signs that warranted encouragement for the student. In the end, clinical coaching that leaves the student encouraged and empowered inspires the desire to return for more. The attending leaves the session by explicitly referring to the student as a physician. He'll be back.

Essential Components of Teaching Weakness

- Because the presentation of weakness is frequently amorphous, the approach to weakness requires a systematic method that will prompt the learner to consider diagnoses that will be missed if she is relying solely on pattern recognition.
- The attending should begin by establishing the four broad categories of weakness, each corresponding to one of the four bars on the "cross of weakness": oxygen delivery, electrolytes, primary muscle disease, and neurologic dysfunction.
- After the four causes of weakness are established, the attending should train the learner to ask the question: Is this focal weakness (in which case the top bar, neurologic causes, becomes more probable), or is this diffuse weakness (in which case, the remaining three bars become more probable)?
- When working through the top bar, it is useful for the attending to emphasize what physical findings are associated with each step in the neuronal pathway from the brain to the muscle. This will help the learner integrate the method into his physical examination of the patient.

❖ Electrocardiograms

Purpose

The electrocardiogram (EKG) is one of the most common tests interpreted by learners on the hospital medicine service. The temptation on the part of both attending physicians and learners is to defer reading the EKG in lieu of consulting to cardiology or using the computer interpretation for diagnosis and subsequent management. This is an opportunity lost, because although consultation can be helpful, most learners will encounter a time when immediate interpretation of the EKG is required for diagnosis and management (clinical performance). Moreover, the EKG provides a framework for practicing the general skill of methodical thought in approaching diagnostic tests and for understanding the pathophysiology that causes cardiac disease.

Attendings should see the EKG as this teaching opportunity: an opportunity to enable learners to understand the disease that causes the abnormalities and how the disease will subsequently behave (potential complications, such as arrhythmias after a myocardial infarction), not merely identifying the abnormalities on the EKG.

This dialogue is a high-level script and may not be appropriate for beginning attendings. But as time elapses and the attending becomes more facile with the preceding teaching scripts, her repertoire will grow. As the attending expands into new teaching scripts, she will discover the joy of designing scripts that make challenging topics appear simple. For the reader who is at this stage of her career, this dialogue has been included as an example of a more challenging, higher-level teaching script. To this end, the first four "teaching blocks" of EKG instruction have been skipped (on rate, rhythm, axis, hypertrophy), and the dialogue begins with an upper-level discussion of ST/T-wave abnormalities.

Setting, Cast of Characters, and Abbreviations

The dialogue begins on a non-call day of the rotation, on the wards, as the attending is making his afternoon personal rounds to complete chart documentation. The cast includes the attending, Phaedrus; the resident, Moni; and the intern, Stef. The medical student, Paul, is in class. With the team split, the attending has a great opportunity to take the teaching to a higher level.

The following abbreviations are used in this dialogue: ATP = adenosine triphosphate; ATPase = adenosine triphosphatase; AV = atrioventricular; EKG = electrocardiogram; MI = myocardial infarction; PVC = premature ventricular contraction; SA = sinoatrial; VT = ventricular tachycardia.

Dialogue

"Dr. Phaedrus, you know Ms. Johnson, the patient we admitted with an ST elevation MI?" asks Stef.

"Yes, how is she doing?"

"Great. I wonder if you have some time to talk me through the EKGs… the ST and T waves? On rounds, you said we would when we had more time."

"Sure," Phaedrus says. "Call Moni. I know she wanted to hear this as well.

Minutes later, Moni, arrives.

"Is Paul here?" Phaedrus says.

"No, he's in class," replies Moni.

"Okay, well, that might work out for the best. This is pretty high-level content. But here's the opportunity, Moni … you too, Stef. You'll never really know something until you can teach it. So what I'm going to do is teach you this skill. I think you'll like it. But then I want you to turn around and teach it to Paul in the next few days. Next week, I'll find a patient with an EKG and ask Paul some questions, just to see how you did. How's that sound?"

Stef replies first. "I'm not sure I'm as good a teacher as you."

"Of course not, Stef. I've been doing this for years, and that's how you become a great teacher … in the same way you become a great golfer: you hit a lot of golf balls. But I will say that I think you have greater potential, Stef, and I believe that you can do it."

Stef replies, "Thank, Dr. Phaedrus. I'll do my best."

When the team is split, there is an opportunity to take one of three roads: 1) choose content that would be beneath other members of the team (if the lower-level learner remains with the attending), 2) choose higher-level content (if the higher-level learner remains with the attending), or 3) teach the higher-level learners and ask them to teach the lower-level learners. The third strategy not only serves as a measure of feedback and evaluation of the attending's coaching efficacy but also serves to consolidate the lessons in the learner (soon to be teacher).

"I know you will. So, Stef, do you have a piece of paper?" Stef hands Phaedrus a sheet of paper, and Phaedrus begins to draw on

the back (Figure w-6). "But before we get to this, let me make sure that we are all on the same page with respect to the method up until this point. Stef, give me your approach to reading an EKG."

"Okay. I start with the name on the EKG and the date, just to make sure it's the right patient."

"Good. Then what?"

"I check the calibration. On the left, the square waves should be 10 millimeters tall, and the paper speed, on the bottom, should be 25 millimeters per second. Then I look at aVR, since all three of the waveforms should be pointing down if the leads have been placed correctly … since all of the electrical current points away from the aVR."

The temptation in reading EKGs is to jump to the most obvious abnormality. Teaching the EKG provides an opportunity to emphasize and reiterate the importance of discipline in interpreting diagnostic test results, particularly the patient safety issue of double-checking that the test result interpreted is for the correct patient.

"Very good, Stef. What's next?" Phaedrus says.

"Next is the rate. I divide 300 by the number of big boxes between the RR intervals.

"Indeed. Well done. What's next?"

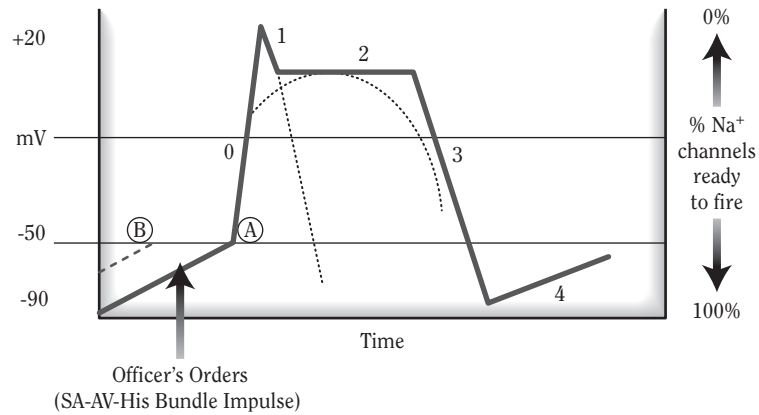

Figure w-6 Action potential of the heart. AV = atrioventricular; SA = sinoatrial.

"Then I look at the rhythm. As you taught me, it should look like a really good prom: everyone has a date, with no one going stag." Stef pauses for Phaedrus's approval, which he receives from Phaedrus's smile. "So every P is paired with a QRS, and every QRS is paired with a P."

"Nice, and remember, the intervals between the two should be consistent. And where do you look to determine the rhythm, Stef?"

"The leads on the bottom of the sheet… V1, II, and V5."

"Correct. Remind me why we use those leads."

A learner's predilection with EKGs is to have memorized steps and patterns. Questions such as "why do we use those leads" reveal that there is a purpose for why we do what we do, and refocus the student to try to understand the physiology being revealed.

"These are the leads that cross the atria, and are best at showing the P waves," replies Stef.

"Yes, and the P waves are important in determining the rhythm. Well done. Now, it's my turn to review the cardinal rules of the EKG. Rule number one: The EKG is like any other test … you need some pretest probability before you look at it. Rule number two: electricity traveling toward a lead will cause an upward or 'north' deflection; current away from a lead will cause a downward or 'south' deflection. Rule number three: Each lead is like a 'spy plane' performing reconnaissance on one part of the heart. Rule number four: the greater the muscle mass of the 'meat,' the greater the electricity will be conducted, provided the meat is healthy, of course. And finally, the x-axis is time, so the greater the width of any waveform or interval on the EKG, the slower the time is required to complete that wave. Do you have those five rules?"

Do not underestimate the lack of understanding in how an EKG works. The attending uses the physiology-based rules to refocus the discussion away from mere "pattern recognition" to understanding why the wave forms look the way they do.

Phaedrus pauses. He then asks Stef to read the five rules back to him, which he does. Convinced he has it, Phaedrus continues. "All right, and how about the axis?"

"Well, I remember that the normal axis is a vector, meaning that it is a balance of two forces: one pulling to the left—the lateral free wall of the ventricle…" Stef pauses, "…and one pulling to the right, the medial or septal wall of the left ventricle. Splitting the difference gives us the axis that is normally in the left lower quadrant, between negative 30 and 90 degrees."

"Excellent, Stef. Great work. And how do you assess the axis?"

"Well, if the current is moving toward the left lower quadrant, it must be moving in the direction of lead I. So I'll look at lead I and ensure that the QRS is upright, since current toward a lead should cause an 'upward' or 'north' deflection."

"Fantastic. It's clear that you've learned from our last session. What else?"

Stef continues. "In the same way, if it's moving to the left lower quadrant, then the QRS should be upright in lead F…" Paul pauses. "But you said that up to a negative 30-degree axis was normal, so the better lead to look at would be lead II."

"Excellent," Phaedrus replies. "And Stef, what are the causes of, say, a left axis deviation?"

Moni looks on, clearly impressed that Stef is going to attempt this. Stef starts in, "Well, if there is more muscle on one side of the left ventricle … since it's the left ventricle that largely contributes to the axis, then the current will be pulled to that side. So I suppose asymmetric left ventricular hypertrophy—in this case, the left lateral wall. Or a loss of meat … I mean, muscle … on the other side. So, an inferior myocardial infarction."

"Fantastic, Stef. I'll fill in the rest, and we can talk about those later. The other causes are a shift in the electrical wiring of the heart, such that one part of the left ventricle depolarizes before the other side. In specific, that's Wolff-Parkinson-White and fascicular blocks."

The attending has already spent time with the team talking about axis, but he takes the time to review the topic both to ensure that the learner performance is where it needs to be and to consolidate the lesson that axis needs to be addressed sequentially in the EKG interpretation before jumping to the "dramatic part," that is, the ST/T waves. This also reaffirms to the learner the critical component to reading EKGs: Current toward a lead is a

"north" deflection; current away from a lead is a "south" deflection. This will be important for the learner to understand the next block of teaching.

Phaedrus begins, "Okay, we are ready to talk about ST/T waves, then. Stef, are you a sports fan?"

"Sure."

"Have you ever wondered why football players cramp at the end of the game? And usually only during the summer months?"

"Yeah, I guess so. Hadn't really thought about it, but that's true."

"And Moni, you remember your OB rotation days, right?"

"Vaguely. But we do consults on those patients."

"Indeed. Did you ever wonder why we give oxytocin to make the baby come out, and magnesium to keep him in?"

"That's just what we do … I don't know why. I thought we were going to talk about MI and arrhythmias."

"I assure you, Moni, we already are." Phaedrus has finished the diagram he began drawing earlier in the dialogue. "Okay, Stef, you're closer to this stuff than we are. Let's see what you remember. This is an action potential for a cardiac muscle. Do you remember the phases?" Phaedrus draws out Figure w-6.

Stef is stretched a bit, but he finally identifies phases 0, 1, 2, 3, and 4 on the diagram.

"Well, done. See, all of those hours in the lecture hall paid off. And you'll remember that the y-axis on this chart is millivolts, from negative 90 to, let's say, positive 20." Phaedrus pauses as he draws in the numbers on the diagram. "… and the x-axis, of course, as always … is time." Phaedrus pauses as the two look at the diagram. "Okay, so Stef … what causes phase 0 and phase 1?"

"Sodium rushing into the cell."

"Correct, and through what voltage-gated, time-gated channels does sodium pass?"

"Ummm, through sodium channels?" Stef says as he raises his eyebrows.

"Correct again, but let me hear you say it all together: voltage-gated, time-gated sodium channels."

Stef and Moni respond in unison, "Voltage-gated, time-gated sodium channels." Both laugh as they finish saying it.

"Wise-acres. Thanks for that, but I meant the words 'all together.'" Stef and Moni laugh again. Phaedrus continues. "Okay, and the y-axis is?"

"Millivolts.… Oh, I get it, that's the 'voltage-gated' part," Moni says.

"I think you are starting to. Look at the z-axis over here." Phaedrus pauses and points to the far right part of Figure w-6. "I'm going to put a spectrum from 0% at the top to 100% at the bottom." Phaedrus draws in the numbers. "Stef, what's the difference between a musket and an automatic weapon?"

"A musket only fires once at a time and then you have to reload it; the automatic weapon fires multiple times."

"Indeed, Stef. I want you to see these voltage-gated, time-gated sodium channels as muskets. Each one has to be cocked in order to fire. And what cocks these voltage-gated, time-gated sodium channels?" Phaedrus asks, emphasizing the word "voltage-gated."

"Umm, the volts, I guess."

"Smart you are. Look at the y-axis and compare it to the z-axis." Stef and Moni lean in. Phaedrus continues. "And would you look at that! When the membrane potential is negative 90 millivolts, 100% of the voltage-gated, time-gated sodium channels are cocked and ready to fire. And when the membrane potential is plus 20, 0% of the voltage-gated, time-gated sodium channels are cocked and ready to fire."

"That's because they are refractory," Stef adds.

"Indeed, Stef … the two terms are the same. 'Refractory' is the same thing as 'not having any of your muskets'—that is, the voltage-gated, time-gated sodium channels—cocked and ready to fire." Phaedrus pauses to let it sink in. "So Stef, at say negative 50 millivolts, what percentage of the voltage-gated, time-gated sodium channels are cocked and ready to fire?"

"Umm … well, let's see…"

Phaedrus can see Stef is overthinking it. "That's right, 'somewhere between 0 and 100%.'" Stef nods in appreciation for letting him off

the hook this time. Phaedrus continues. "The greater the membrane potential—that is, the more negative it is, the greater the percentage of the muskets that are cocked and ready to fire." Phaedrus pauses to highlight the compare-and-contrast moment, "And the more positive the membrane potential, the fewer the number of muskets cocked and ready to fire." Phaedrus emphasizes the key words "negative, greater," and "positive, fewer" as he speaks. "Okay, let me direct you to this picture." Phaedrus draws out the figure of a cell (see Figure w-2). What causes the membrane potential of a cell to be negative 90 millivolts? That is, more negative inside the cell than out? Stef?"

There is a big investment of time and basic science physiology for this teaching script, but it will be worth it later—not only for the rewards of this mini-lecture but for other mini-lectures as well. This not only validates the learner's previous learning in the preclinical years, but also communicates that internal medicine is more than just protocols; it is about the science that enables understanding and predicting disease.

"The sodium-potassium pump," Stef says.

"Fantastic, but don't sell the pump short. It's the sodium-potassium ATPase pump. If it's a pump, it has to have energy to make it work … this sodium-potassium ATPase pump is very important not only to the heart but also the kidney and the muscles. So the sooner you make this guy your friend, the easier medicine will become." Phaedrus pauses. "So I'll walk you though this part. Moni, tell me if I get it wrong." Moni looks appreciative of the validation. Phaedrus continues, emphasizing the word "ATPase" as he speaks. "Okay, the sodium-potassium ATPase turns, and that moves three sodiums out of the cell, and two potassiums into the cell. So already we're net-negative inside the cell since more positives are moving out than in. AND … since the chemical concentration of potassium inside of the cell is so high, whatever potassium is pumped into the cell gets immediately expelled out the 'side door'—the potassium rectifier channel—making the cell even more negative. Moni, did I get that right?"

"Yes, I believe so." Moni shows hints of pride for having been asked.

"Okay, Stef, are you ready?"

"I guess so."

"All right." Phaedrus draws the diagram in Figure w-7. "This is a heart. I've taken this part of the heart and enlarged it to give you some greater detail." Phaedrus points to the small box in the diagram, then directs the group's attention to the coronary artery. "Let's say a plaque ruptures … a thrombus forms on top of the plaque … and blood flow through the coronary artery stops." Phaedrus draws in an obstruction in the coronary artery. "Stef, based upon what you know about Equation 3—the delivery of oxygen equation—what happens to the delivery of oxygen to this part of the heart?" Phaedrus points to the hatched area of the heart in the diagram.

"It stops …. Uh, there is no oxygen delivered."

"Correct. And with no oxygen to the heart muscle, what happens to the ATP?"

"It goes down," Stef replies.

"Indeed … and with no ATP, what happens to the activity of the sodium-potassium ATPase pump?"

"It goes down," Stef says quickly.

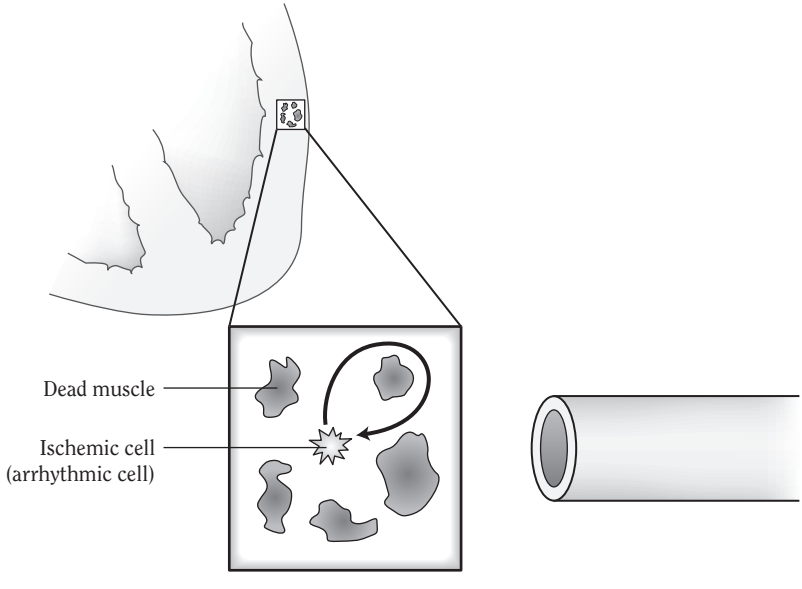

Figure w-7 The scarred heart.

"Right again. This question is a little tougher, Stef, but I think you can do it. What happens to the membrane potential as the activity of the sodium-potassium ATPase goes down?"

"It goes up … or, rather, the membrane potential becomes less negative."

"Yes! And what happens to the percentage of muskets that are cocked and ready to fire if the membrane potential goes up?" Phaedrus points to the z-axis in Figure w-6.

"There are less of them."

"Indeed. Now, hold that in your left hand … keep your right hand mentally free for what comes next … we'll return to the implications of having less muskets in a minute." Phaedrus pauses. "But for now, look at the y-axis. With the membrane potential becoming less negative, where does phase 4…" Phaedrus points to the y intercept of phase 4 on the diagram, and then continues. "… where does phase 4 start?"

"Well, as you have it drawn here, it looks like it starts higher up on the y-axis."

"Correct. And with the same slope, how much time does it take for phase 4 to reach its threshold…" Phaedrus draws in the new "ischemic" phase 4 slope (line B in Figure w-6) and continues, "… relative to the normal phase 4 slope?"

"It reaches its threshold earlier," Stef says.

"Absolutely. Now Moni, I'm going to turn to you. Here's what you need to know … indeed, what you probably already know. The heart is like an army, with each cell being a 'private' solider in that army. When the army is synchronized, all soldiers acting as one, it is effective. Leading the heart is a general—the SA node. It barks orders to the colonel, the AV node, who in return passes along the orders to the three sergeants: the three fascicles of the right and left bundles. These in return pass the orders to the privates—the muscle cells—and the muscle cells contract in unison. The time it takes for the orders to get from the general to the colonel is the P-R interval … the SA node to the AV node." Phaedrus pauses to allow the two to think about the metaphor, and then continues. "The time it takes for the orders to get from the colonel to the sergeants, and then to the privates—the AV node to the bundles to the cells—is the QRS." Phaedrus pauses again. "This is the officer's line of commu-

nication, or the 'macro current' that is running through the heart." Another pause. "Once the soldiers—the muscle cells—receive the orders, then each depolarizes. Their collective depolarization occurs during the ST segment, that is, just after they have received their orders. The depolarization of each cell is the 'micro current.' Ordinarily, we do not see the summation of these 'micro currents' on the EKG because all of the cells are depolarizing at the same time, which is why the ST segment is flat."

Again, talking about the SA-AV-His current seems like a big tangent, but it has a very important purpose. The first block of the dialogue was to establish that ischemic cells reach their threshold early; this part of the block is to enable learners to compare normal method of depolarizing the cells (the SA-AV-His current) to the "new" method of depolarizing, that is, the ischemic cells getting to their threshold first. The big lesson is that ischemic cells are arrhythmic cells since they reach their threshold and depolarize before the SA-AV-His current reaches the cells. This is the outcome the learner needs to focus upon in managing ischemic heart disease. The EKG is being used as the vehicle for teaching that lesson.

"I'm not sure I get that last part." Stef says. "Why is the ST segment flat if they are depolarizing?"

"Hold that in your left hand, Stef. I'll return to it in a moment. But for now, let me ask you … Stef, you said you like movies. Did you see that movie Platoon*? You know the one, with Charlie Sheen and Willem Defoe?"*

"Yeah, I remember that. Cool movie." Stef shows some additional enthusiasm at the mere mention of a movie.

"I agree, cool flick … okay, then you'll know the answer to this question. What happens when you let a platoon of privates go without orders from above for a prolonged time?"

"Well, in Platoon*, they eventually just made their own orders. They did their own thing," Stef notes with authority.*

"Indeed." Phaedrus points to the spot where phase 4 crosses the threshold on Figure w-6. "I want you to see each patch of muscles as a platoon of soldiers waiting for orders from on-high, slowly growing impatient because they haven't yet received orders from their 'officers' and rising to the point where the platoon makes its own orders." Phaedrus traces along phase 4 in Figure w-6 to the threshold point. "If they have to wait too long, they reach the

threshold point, and they fire on their own." Phaedrus pauses for effect. "So the race is between contestant number one … the general-colonel-sergeant's orders getting to the cell…" Phaedrus draws in the vertical line on the figure and labels it 'officers' orders.' "versus contestant number two, the platoon's slowly rising slope to get to their own order point." Phaedrus pauses to let Stef and Moni see the juxtaposition of the two contestants. "So Stef, look at these two lines. Which of these two lines reaches its threshold first, line A—the normal heart—or line B—the ischemic heart?"

"Line B, of course."

"And which of these two lines has the better chance of beating the officer's orders?" Phaedrus points to the spot where the vertical line labeled "officer's orders" crosses the threshold.

"Again, Line B," Stef says.

"Exactly. And here's the point. It's sad that muscle cells might die from an MI, but the real risk to the patient is not the cell death, but the residual 'ischemic' muscle cells, those muscle cells with a higher membrane potential … close to their threshold. Those residual cells that will lead a revolt to cause the arrhythmia that kills the 'army' and eventually the patient."

The time it takes to get the learners to this critical point (ischemia = arrhythmia) is proportional to its importance. Learners see the EKG as a diagnostic test, but fail to see its prognostic significance (that is, its ability to show who is at risk for sudden death).

Stef points to Figure w-7. "But so what if the 'platoon' reaches its threshold … isn't that just a PVC?"

"You ever been camping, Stef?" Phaedrus replies.

"No," Stef says.

"Seen it on TV?" Phaedrus responds.

"Yeah, sure."

"Then you know the first rule of camping: You don't have to be faster than the bear—you just have to be faster than the slowest person in your camping group." Stef laughs, but it is clear by his expression that he wonders where this is going. Phaedrus continues. "It seems intuitive that if you shut off oxygen to a part of the

heart that all cells would die at the same rate. But like a camping group, there is heterogeneity, and some cells are stronger than others … that is, some cells die before others." Phaedrus completes the diagram in Figure w-7. "I've shaded in the cells that die first—the slow campers. It is unfortunate that they're dead, but these cells are of little consequence now, since dead cells can't start the electricity needed for an arrhythmia. But look at the platoon of cells in the middle here." Phaedrus points to the middle of Figure w-7. "Stef, are these cells dead, fully alive, or, to quote The Princess Bride*, mostly dead all day?"*

Stef perks up again with the second movie reference. "I would guess 'mostly dead all day'— they're ischemic."

"Correct. These cells are mostly dead—that is, ischemic—but still capable of an action potential. So as you said, Stef, these cells have decreased oxygen, decreased ATP, decreased activity of the sodium-potassium ATPase pump, and an increased membrane potential … thus, they are close to their threshold. Note, too, that the myocardial scar surrounding the cells…" Phaedrus points to the shaded patches in Figure w-7. "… slows the communication of the 'general-colonel-sergeants' orders from getting to the platoon." Phaedrus draws the line bouncing around the scar in Figure w-7. "This platoon is going to revolt, both because it is closer to its threshold and because the orders from on-high are delayed in getting to it. The revolt will, as Stef noted, result in a PVC. And if that PVC spins right back on itself…" Phaedrus points to the far part of the phase 2 on the action potential in Figure w-6. "… what will happen, Stef?"

"Nothing. The cell is refractory. Uh, it has no muskets cocked and ready to fire." Stef looks for approval at remembering the metaphor.

Phaedrus obliges with a smile and continues. "But what if the action potential—the PVC—went out and around the scar…" Phaedrus draws the circuitous path of an action potential traveling out from the interior of the scar, bouncing around the scar patches, and then returning to its origin (Figure w-7). "… returning to its original origin?"

Stef agrees. "Well, then it could stimulate itself … the cell would be repolarized by then."

"And what would you have if that circuit happened five times?"

Stef responds quickly, "A five-beat run of VT."

"And 10 times?"

"A 10-beat run of VT."

"So here's the learning point, Moni. Whenever you see a five- or 10-beat run of VT, I want you to visualize this picture." Phaedrus points to the diagram of the scar again. "The patient is telling you that he has a high probability of a scarred myocardium, and by implication, a high probability of VT. This will be useful as you try to determine which of those 'rule-out MIs' is the real deal."

This is the learning objective reiterated (ischemia = arrhythmia), but it is moved one step closer to objective indicators of risk (PVCs).

Stef, playing his role as the intern, asks, "But what do I do about a five-beat run of VT?"

"Good question, Stef. The answer, Stef, is nothing. If the 10-beat run stops, then it's done. There is need for lidocaine at that point. But I want you to have the gestalt. If there's a 10-beat run of monomorphic VT—and monomorphic it will be, because it's coming from one, or a 'mono' source—then there is a high probability that a bigger run could or will arise."

"Hmm." Stef says, obviously contemplating past patients.

"Now, let me ask you, Moni. What would happen if it became constant: multiple cycles of stimulation-restimulation?"

"That would be true VT," says Moni.

"Indeed … that's the lesson. The way your MI patient will die is not from the heart pooping out but instead by arrhythmia. For all cardiac patients, this is the 'ball' that I want you to keep your eye on." Moni nods. Phaedrus continues, "Now think carefully about what started it all. What is the most powerful antiarrhythmic known? How would you fix this?"

"Beta-blocker?" Stef responds with a question.

"Well, not the answer I was looking for, but it is a correct one. How would that help?"

"I don't know, but it is in the protocol."

Moni jumps in, "I think I know this. It flattens, or lessens, the slope of phase 4. Catecholamines raise the slope … that's the rea-

son we advise against caffeine, stimulants, and stress in the post-chest pain period, and the reason patients who have used cocaine not only have chest pain but also arrhythmias."

"Well said, Moni." Phaedrus responds. "Beta-blockers are anticatecholamines, so they flatten the slope, giving more time for the SA-AV node impulse to get to the cells before the platoon hits its threshold. Now Stef, let me ask my question another way: why is it we hospitalize someone for 48 hours after an MI?"

Correct answers, even if they are not what the attending was looking for, should be validated. Even though tangential to the original flow of "ischemia causes arrhythmia," the tangent of "beta-blockers in arrhythmias" in this example is brief and links back into the original flow of the conversation. If a "right answer" is very tangential, or not easily linked back into the original flow, a validating but deferring tactic should be evoked to avoid confusing the learners with a half-done discussion. An example of this tactic might include, "Great point. Take a note of that and we'll come back to it when we are finished here."

"Because that's how long it takes to get a cath at Charity?"

"So cynical, Stef." Phaedrus smiles. "But the real reason is that 90% of post-MI arrhythmias occur within 48 hours of the MI."

"Why's that?" Stef asks.

"Because this renegade platoon of soldiers can't remain 'mostly dead all day' forever. At 48 hours, it has to either regain oxygen and the slope returns to normal, or it dies, in which case it is no longer capable of starting an arrhythmia. So final question, Stef, what is the most powerful antiarrhythmic? And as you prepare to answer, think about what started this whole thing: high slope, high membrane potential, no ATPase activity…"

"Oxygen. That's what started it. The lack of oxygen."

"Yes! Even if little contractility remains, revascularization is the right answer, just as aspirin and enoxaparin are the right answers to open the thrombus to resume oxygen to the muscles and prevent the arrhythmia. The sooner you re-deliver oxygen, the lower the mortality."

Moni looks on with a contemplative face. "But what about the ST/T-wave changes? How does that play in here?"

"All right, Moni. I think you've earned this. You've worked hard this month." Phaedrus draws Figure w-8. "Now I've taken this part of the myocardium and expanded it into a larger diagram. Do you see how the normal heart is on the left and the ischemic area is on the right?"

"Sure."

"Now, I'm going to draw a line through the middle. The top part will be the extracellular—that is, the electrolytes on the surface of the cells. The lower part will be intracellular—that is, the electrolytes on the inside of the cells. Have you ever wondered why

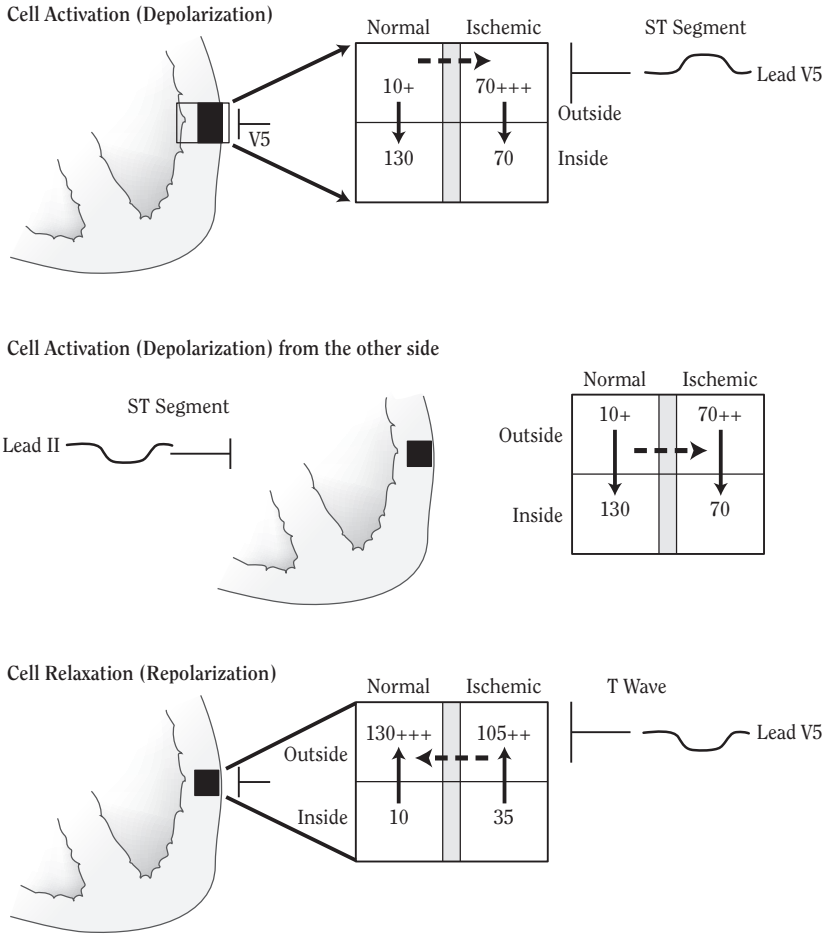

Figure w-8 The micro-current and ST segments.

they are called 'electro-lytes'?" Phaedrus asks, emphasizing "electro" as he speaks.

"I'm starting to get it," Stef says.

"Now, I'm going to draw a lead … let's say it's V5." Phaedrus draws the horizontal T to the right of the exploded diagram. "Let me ask you, Stef—the lead will pick up current toward and away from it, right?"

"Yes. And current toward the lead will be 'north' and current away from it will be 'south,'" Stef responds.

"Indeed. Can the lead see what is inside the cells … insulated by that lipid bilayer?"

"I'm guessing no."

"Correct. Now at the start of it all, how much sodium is setting outside of each patch of cells, if the patient's serum sodium concentration was, say, 140."

"Well, 140."

"Right. Now the nonischemic cells on the left have 100% oxygen delivery. Right?" Stef nods. "… which means 100% ATP … and 100% activity of the sodium-potassium ATPase pumps … and thus a negative 90-millivolt membrane potential. So, Stef, what percentage of the muskets are cocked and ready to fire … the voltage-gated, time-gated sodium channels?"

"100%," Stef replies.

"Good. So how much of the sodium will rush into the cell when stimulated?"

"All of it," Stef replies.

"Correct. For illustration purposes, let's say almost all of it, say 130 of it. And you'll agree that if 130 of it rushes into the cells, only 10 sodiums will remain?"

"Sure," Stef replies.

"Okay, good. Now, let's look at the ischemic path of cells. Let's say they only have 50% oxygen delivery. Moni, walk through it for me. What will happen to the ATP, the ATPase pumps, and the membrane potential and the number of muskets cocked and ready to fire?"

"Well, with half the oxygen delivery, there will be half the ATP. And with half the ATP, there will be half the activity of the ATPase pumps, and that will lead to a less negative membrane potential." Phaedrus points to the z-axis on the action potential figure as Moni speaks (see Figure w-6). "And that will lead to less voltage-gated, time-gated sodium channels available."

After the learners have been through a Socratic method enough times such that the attending is confident of their competence, they should be given the opportunity to prove even additional competence by running through the method on their own.

"Great, and if that's the case, how much of the sodium will rush into the cells when they are stimulated?"

"About half, I guess."

"Okay, so if 70 rushes in, how much is left outside of the cells?"

"70," Moni says.

"Great. Now look at this, and keep in mind that all of this is happening just after the cells have been stimulated—that is, just after the QRS."

"Yeah, during the ST segment." Stef notes.

Phaedrus continues. "Correct. Now there are 10 'pluses' … that is 10 sodiums, sitting out side of the normal cells. There are 70 'pluses' … that is 70 sodiums sitting outside of the ischemic cells. If you were an electron, being negative and all, where would you head, assuming Paula Abdul's wisdom is correct and 'Opposites Attract'?"

"Toward the 70 pluses, of course."

"And as we have it drawn, would that be toward or away from the lead?" Phaedrus draws the dashed line in the top figure in Figure w-8.

"Toward the lead," Moni says.

"So would there be a deflection in the waveform? And if so, which waveform, and it what direction?"

Moni looks at it and then suddenly gets it. "There would be a deflection, and it would be the ST segment heading north since it is toward the lead. ST elevation!"

"That's pretty cool," Stef adds.

"Indeed it is. And what if I had drawn the lead on the other side of these cells, say lead II?" Phaedrus points to the left of the diagram (the middle level of Figure w-8). "What would the ST segment look like in that lead?"

Stef answers, "Well, the current would be going away from that lead, so it would be ST depression."

"Reciprocal change," says Moni. "ST segment elevation on one side of the heart has ST depression on the opposite side … I agree, that's pretty cool."

"Indeed it is." Phaedrus says. "And now for the really cool part. Look at these cells again, Stef. When they go to repolarize, what will make that happen?"

"The sodium-potassium ATPase pump."

"And the normal cells … with normal oxygen delivery—normal ATP—will have what percentage of their sodium-potassium ATPase pumps working?"

"All of them," says Stef.

"How much of the intracellular sodium," Phaedrus points to the "130" inside the cells, "will be pumped out of the cells?"

"Almost all of it."

"Good, so we can say that all but … say, 10 sodiums … are pumped out. So 130 sodiums are on the outside of the cells." Phaedrus points to the bottom layer in Figure w-8. Stef nods. Phaedrus continues. "Now, how about the ischemic cells with half the oxygen delivery, half the ATP, half the activity of the sodium-potassium ATPase pump? How much of the 70 sodiums inside the cell will be pumped out?"

"Half, I guess," Stef responds.

"Good. So we can say that the 35 that are pumped out will join the 70 that were left outside … so 105 sodiums … or 105 'pluses.'" Phaedrus pauses. "So 130 pluses outside of the normal cells, and 105 pluses outside of the ischemic cells … where will the electrons travel?"

"Toward the 130 pluses … toward the normal cells, or away from the lead."

"And this is happening during what part of the EKG, this repolarization activity?"

"During the T wave," Stef notes. And then he gets it. "Wow, so that's T wave inversion."

"Well, there you go, team. All you need to know about how ST and T waves relate to what's actually happening at the cells. The QRS measures the conduction of the officer's orders. But it's the ST and T wave that tell you what's going on with the platoons. And hopefully, this has linked what was previously just 'another test' to you to the real issue in taking care of these patients: assessing the risk for, and preventing, arrhythmias."

The link is complete: ischemic cells are arrhythmic cells, and ST changes, T-wave changes, blocks, and PVCs are all EKG indicators that the probability of arrhythmias has increased.

The team nods in approval. Then Stef interjects, "But why do athletes cramp in the fourth quarter?"

"Oh, indeed. I forgot about that," says Phaedrus. "Well, quickly, Stef … during strenuous exercise, especially when it is hot, your body sweats to keep you from getting too overheated. By the end of the game, again, especially when it is hot, all this sweating has most likely decreased your intravascular volume. Am I right?"

"Yeah, sounds right."

"And with decreased volume, what happens to the preload to your heart?"

"It decreases." Stef replies.

"And what happens to the cardiac output to the kidneys?"

"Well, it decreases as well."

"Good. And with decreased blood flow to the kidneys, the glomerular filtration rate will go down. What will happen to the aldosterone level?" Phaedrus asks.

"The aldosterone level will go up." Stef replies.

"Right. And do you remember how aldosterone works at the cellular level?" Phaedrus waits 5 seconds, and with no answer, he draws out the renal tubular cell with the aldosterone pathway and continues. "So you'll remember that aldosterone is a steroid, and

like all steroids, it turns on transcription for pumps. In this case, it is our old friend, the sodium-potassium ATPase pump." Phaedrus points to the sodium-potassium ATPase pump. "Now, here's something that you might not know. Your sweat glands also have aldosterone receptors, which means that when this cycle of dehydration-renin-aldo, more dehydration-renin-aldo has gone on and on, the aldo goes to the sweat glands, reabsorbing sodium from there too. The solute that goes out with the sweat is now potassium."

"So potassium causes the cramp?"

"No, not really, it's just a side effect. But remember this, Stef, and this is important as you think about causes of 'back side arrhythmias'—that is, arrhythmias that occur during phase 3 repolarization—you know, torsades." Phaedrus pauses. "Okay, here's the important point. Any time you see ATPase pumps, you have to have two things to make those pumps work. You need ATP, of course, but you also need magnesium. Both ATP and magnesium will accelerate the activity of those pumps, and the absence of either significantly impairs the activity of those pumps."

Moni interjects, "Hmm … I see where the OB thing might come in."

"Indeed, Moni. But hold that for a second, and I'll finish with the athlete's cramps … then we'll turn to the uterine cramps." Phaedrus pauses. "So now I want you to think of the calf muscle cell. Its membrane potential is a little different than the cardiac muscle cell action potential, but the principles are the same. And for the purpose of what we are doing here, follow along with the figures that we used for the cardiac action potential." Phaedrus points to Figure w-6. "Okay. So the cell contracts, and the membrane potential becomes positive as sodium and then calcium enters the cell. But let me ask you, and stay with our action potential here, what cases repolarization of the cell?" Phaedrus points to Phase 3 in Figure w-6.

"Well, the sodium-potassium ATPase pump," Stef notes.

"And that ATPase pump needs oxygen to make the ATP, and magnesium, right?"

"Yes," Stef agrees.

"Well, okay then. Here's what happens. The aldosterone level gets high, stimulating the 'millions of little nephrons on your skin'

known as sweat glands. What do you suppose happens to the magnesium?"

"It goes to all of those sweat glands."

"Indeed. And with all of the magnesium tied up in the periphery, how much magnesium is left for the muscles?"

"None." Stef pauses. He has figured it out. "Oh, so with no magnesium, there is no activity of the sodium-potassium ATPase pump, and the cell doesn't repolarize—that's the cramp!"

"Indeed, Stef. Add to that the decreased venous volume, which decreases the cardiac output, which in turn decreases the delivery of oxygen to the tissues … remember Equations 3, 5, and 6?" Stef nods. "So if you were going to fix that, what would you advise the athlete?"

"Well, to drink some magnesium and potassium containing-fluids. Wait, that's Gatorade."

"Pretty cool, huh?" Stef nods. "Now for the OB lesson. And Moni, think about this if you are asked to consult on that service." Moni nods. "What would you give to a pregnant patient if you want the uterus to contract … you know, like you were going to induce the delivery?"

"Well, oxytocin. And prostaglandins."

"Indeed. And remember that most prostaglandins keep vessels and tubes open; but this prostaglandin causes vasoconstriction to the uterus. And of course oxytocin is only two amino acids different from vasopressin, so it will cause vasoconstriction as well. So based on Equation 3, what will the oxygen delivery to the uterine muscle be?"

"Decreased."

"And how much ATP will be available for the uterine cells?"

"Less … oh, I get it. With no ATP, there will be no activity of the sodium-potassium ATPase, no repolarization of the muscle, and the muscle will contract and cramp."

"And what would you give if you wanted the uterine muscle to not contract … say for preterm contractions?"

"Magnesium."

"Good. Tell me why," Phaedrus says.

Moni has connected the dots. "Well, because magnesium will increase the activity of the sodium-potassium ATPase, and that will drive the membrane potential below its threshold, so no contraction."

"Indeed, and talk me through the complications of high-dose magnesium therapy."

"Well, I remember that the complications are … let's see … decreased reflexes … that must be due to the weakness of the skeletal muscles because they are hyperpolarized as well!"

"Well done, Moni. And how about the smooth muscles that maintain vascular tone? Any effect on those?"

"The hypotension."

The presumption of phase 2 clinical reasoning (that is, one syndrome equals one disease) is that each disease is its unique entity. Teaching the pathophysiology that underlies various diseases allows crossing over from one organ to the next (for example, from the heart, to the kidney, to the uterus, to the smooth muscle), and this reveals the underlying theme that explains medicine.

"Doesn't she sound smart, Stef?" Stef nods. "But just so I don't lose you to OB/Gyn, let me bring this back to internal medicine and the 'backside arrhythmias.'" Phaedrus points at the phase 3 on the cardiac action potential (Figure w-9)."Let's say that we stimulate the cell at this point." Phaedrus points to the very beginning of phase 3 (Arrow [a] in the top part of Figure w-9). "What would happen, Stef? And tell me why."

"Nothing. The cell would be refractory … there would be no muskets cocked and ready to fire."

"Correct," says Phaedrus. "Now, let's say that we stimulate the cell here." Phaedrus points at the very end of phase 3 and the beginning of phase 4 (arrow [b] in top part of Figure w-9). "What would happen? And tell me why."

"It would stimulate the cell. There would be another action potential because all of the muskets are cocked and ready to fire."

"Indeed … no great shakes, though. It would just be an early depolarization." Phaedrus pauses. "But let's say I stimulated the

cell here—maybe another cell somewhere in the heart hit its action potential, and the current hit right at this time." Phaedrus points at the midpoint of phase 3 (arrow [b] in the top part of in Figure w-9). "What would happen then?" Moni's gaze goes to the z-axis. She is measuring up the number of muskets cocked and ready to fire. Phaedrus recognizes this and continues. "And I'll give you a clue. There are 51% of the muskets cocked and ready to fire, just enough to entertain an impulse."

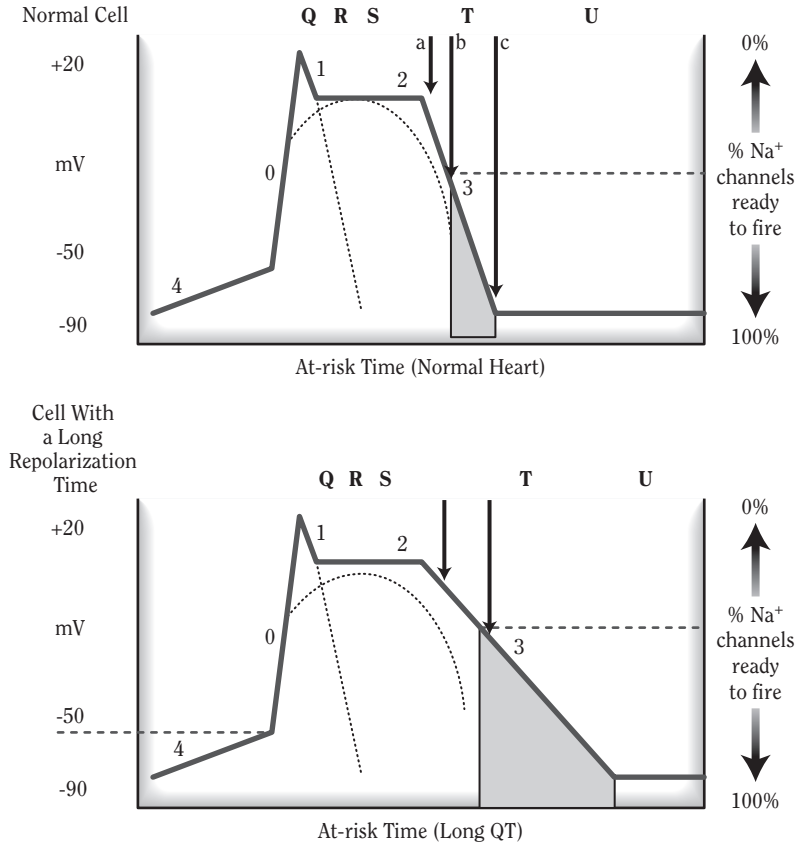

Figure w-9 The "at-risk time" due to long QT. Phase 0 = Sodium rushes into the stimulated cell through voltage-gated, time-gated sodium channels; phase 1: the time-gated sodium channels close, causing a decrease in the membrane potential; phase 2: calcium enters the cell, buoying up the membrane potential; phase 3 = the sodium-potassium adenosine triphosphatase pump activates, making the membrane potential of the cell more negative; phase 4 = the hyperpolarized cell slowly loses its gradient, with a membrane potential that gradually rises.

"Well," says Moni. "I would say that there would be an action potential then."

"Okay," says Phaedrus, "But I want you to look at where this cell is in reference to its threshold." He draws a line from that point over to the y-axis.

Moni compares the lines and responds, "It's already over its threshold."

"Correct. This cell is 'supercharged' with sodium, a literal time-bomb waiting to go off. And where are the other cells in the heart at this point?"

"At the same point, I guess."

"Indeed. And when that cell goes off, with all neighboring cells already above their threshold—supercharged—a wave of current is born, circling around the heart, and then turning to go the other direction, then back and forth again. And when the wave goes toward and away from the lead, you get … Stef, what does the QRS look like?"

"North and south."

"And when the wave goes side to side, neither toward nor away … Moni, what does the QRS look like?"

"Well, I guess flat."

"And that twisting appearance, sometimes north/south, sometimes flat, that is…"

"Torsades!" Stef announces proudly. "Wow, I knew the pattern, but I didn't know why."

Moni interjects, "That's cool. But are you saying that anyone who had an action potential that fell at exactly that point could have torsades? I thought it was only for people with a long QT."

"That's exactly what I'm saying, Moni. Life is fragile." Phaedrus pauses. "Have you heard of commotio cordis?" Moni has a quizzical look on her face. "Well, it is the syndrome of sudden death that occurs when a baseball, or a judo kick, or something like that, hits the chest at exactly the right time." Phaedrus pauses to point at arrow b in the top part of Figure w-9, and then continues, "… with enough energy to cause the action potential to kick off the torsades." Stef looks frightened. "Don't worry, Stef, it's rare. But

what is not rare is the long QT, and Moni is correct … a long QT does increase your risk. At a later point, I'll show you why. But for now, let me show you this…" Phaedrus redraws Figure w-9 with a flatter phase 3 (the bottom part of Figure w-9). He then draws a vertical line from the point on phase 3 where "51% of the VG-TG sodium channels are cocked and ready to fire" (the dashed line) down to the x-axis. He draws another line from the cell threshold to the x-axis, and shades in the area between these two lines (the shaded area in Figure w-9). "The difference between these two points is the 'at-risk time.'" "Now tell me, Stef, which of these two 'at-risk times' is bigger?"

"The second one."

"And what part of the EKG does phase 3—repolarization—correspond to?"

"Well, repolarization is the T wave.… So I would say the T wave," responds Stef.

"Indeed, so what would the T wave look like with a longer phase 3 … remember the x-axis is time." Phaedrus points to the second phase 3 line with the flatter slope.

"Well, it would be a longer T," says Moni.

"Indeed, Moni. And that's the long QT … corresponding to the longer phase 3 slope, and the bigger 'at risk time' for torsades." Phaedrus pauses. "Now, knowing what you know about how to hyperrepolarize the uterine muscle, how would you sharply decrease the slope of phase 3? Because if you did that, you could cut the 'at risk time' in half."

"Magnesium," Moni responds. "… would increase the sodium-potassium ATPase pump, and that would repolarize the cells faster."

"That's right, Moni. And that's the treatment for torsades, magnesium."

"Wow." Moni thinks for a little bit. "But why does potassium cause the long QT?"

"That, Moni, you already know … you just don't know that you know it, yet. Think about it—we'll talk about it later when we address potassium."

Essential Components of Teaching Electrocardiograms

- The EKG is one of the most common tests in clinical medicine. Because it so common, however, the attending should be aware that many learners will come to see the test as an exercise in "art history," trying to memorize visual images. This method does not work reliably, however, because the number of variables in the EKG (infarcts, blocks, rhythms, hypertrophy, axis, and anatomic variations) creates multiple visual patterns.
- By linking the interpretation of the EKG to the pathophysiology that underlies the normal and abnormal tracings, the attending can liberate the learner from the "art history" mentality, and enable the learner to interpret subsequent EKGs that may have multiple abnormalities. It also enables the learner to interpret the same abnormality (that is, ST-segment elevation) in different diseases (such as myocardial injury due to ischemia vs. pericarditis).
- Trying to do all of the EKG in one session is information overload. The natural break points between rate/rhythm and axis, and again between axis and ST/T waves, enable bite-sized chunks during which students can reflect on what they have learned.
- The attending should emphasize the importance of having a method of interpretation that is routinely performed. Because teaching of the EKG will probably take several sessions, the attending should begin each session by quickly reviewing the prior teaching lessons (rate, then rhythm, then axis, then hypertrophy, then ST/T analysis). This will consolidate the importance of using the method for each EKG, which by deliberate practice standards improves learner performance.