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Relying on your H&P

ARE WE LOSING THE ART OF CLINICAL MEDICINE TO TECHNOLOGY?

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I presented a lecture entitled "Critical Complaints" at the 2006 EMS Today Conference in Baltimore. This primer lecture on differential diagnosis had been well received for many years past; however, 10 minutes into my lecture, I was asked by an attendee, "Why do we need to know this? We see X, we do Y. That's it."

Totally stunned, I spent the next 10 minutes explaining why knowing the "why" of medicine is sometimes more important than blindly doing the "what" of medicine.

Let's look at three actual field cases. In the first case, proper care depends on patient history. In the second case, appropriate care depends on physical examination findings. The third case illustrates that dependence on a "medical technology diagnosis" can result in inappropriate, and usually perilous, patient care. The crews in the following cases depend on solid clinical skills to direct appropriate care over the disparate input of medical technology.

CASE 1: PATIENT HISTORY

A call is dispatched for a 27-year-old male with a chief complaint of "generalized weakness." On EMS arrival, his history of present illness is malaise, non-productive cough and myalgia increasing over the past two days. He was working at his desk when his office colleagues noted he wasn't feeling well. He was healthy prior to the onset of these symptoms. He denies significant past medical history, takes no medications and has no known drug allergies. Initial vital signs by automated medical technology devices are as follows: heart rate 96/minute, blood pressure 110/85 mm/Hg, respiratory rate 20/minute, pulse oximetry 96% on room air.

On primary examination, the patient is in no acute distress, lungs are clear, no jugular venous distention (JVD), regular rate and rhythm, soft and non-tender, A+Ox4 and afebrile. The patient states he probably has a virus "like everyone else in the office," and he just wants to go home and rest. The crew discusses follow-up options and jokingly tells the patient, "Now, don't go using your sick days to fly off to warmer climates and leave the rest of us here in the cold." All on scene laugh at this jovial comment. The patient assures the medics he was going to go home and rest. "And besides," the patient says, "I just got back from a three-day business trip to sunny Southern California."

This last "incidental" tidbit of patient history peaks the interest of the senior medic on scene. The medic then completes the secondary survey and notes the patient's left leg was swollen and slightly erythematous from mid-thigh distally. The patient was apparently unaware of this. On further evaluation, the pulses are globally equal, but there's some tenderness along the medial thigh in the distribution of the left saphenous vein, and the left calf is larger than the right and slightly tender.

The medic reviews the case and realizes, taking one "automated medical technology number" at a time, the

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readings are essentially within normal limits; however, the patient's heart rate is almost tachycardic, and his blood pressure is slightly low for age and gender, with a slightly narrow pulse pressure. His respiratory rate is 20/minute when the adult average respiratory rate at rest should be 14/minute. He has no fever common of viral illness, and his room air pulse oximeter level is 96% but should be 98–100%. The physical findings of a tender swollen left leg and a recent history of prolonged immobility (i.e., a round-trip transcontinental airline flight over a three-day period), suggests "venous stasis."

The medic now realizes that this "simple" treat-and-release viral-syndrome patient actually has a textbook example of signs and symptoms, history and physical conditions consistent with deep-vein thrombosis (DVT) and pulmonary embolism (PE). If the medics were to rely on the cold numbers of medical technology noted above, the vital signs could essentially be dismissed as normal. The chief complaint could also be dismissed as viral syndrome, especially with the primary survey being normal. Based on the "numbers," the patient should be in no acute danger.

VENOUS STASIS is one of Virchow's triad for predisposition to DVT/PE, the other two being hypercoagualability and endothelial damage. Only one of the three is needed to raise suspicion for DVT/PE.

The medic discusses these findings and his suspicions with the patient and urges emergency department (ED) evaluation. The patient is transported to the ED and admitted to the intensive case unit (ICU) with left iliofemoral saphenous DVT and multiple bilateral sub-segmental pulmonary perfusion defects (i.e., PE).

In this case, if the medic didn't have his "medical-history radar" on, the connection between the cross-country airline flights, venous stasis and the patient's signs and symptoms wouldn't have been made. The patient may have gone home and continued to release emboli with consequential increased morbidity, if not mortality.

Even if the patient's complaints are minimal, and they have relatively normal vital signs and a benign cardio-pulmonary exam, we must remember that for an otherwise healthy young person to be symptomatic due to PE equates to significant pulmonary dysfunction; therefore, we're dealing with a patient with considerable loss of pulmonary perfusion. The patient is bordering his limit for normal physiological compensation to maintain homeostasis. It takes a significant insult to any single bodily system to produce signs and symptoms in an otherwise healthy patient.

CASE 2: PHYSICAL EXAM

On a hot summer morning an ambulance is dispatched to a tennis court at a retirement village for a female with a chief complaint of "dizziness." On arrival, the crew finds a 70-year-old female sitting against the tennis court fence. Her history of present illness is fatigue, shortness of breath and chest tightness for the past 20 minutes. She states she had been playing tennis for about a half-hour without difficulty when the symptoms began.

She has a past medical history of hypertension treated with 25 mg of hydrochlorothiazide QD and takes 82 mg of ASA. She has no known drug allergies. She has no other significant history. Initial "automated" vital signs are as follows: heart rate 70/minute, blood pressure 100/70 mm/Hg, respiratory rate 18/minute, pulse oximetry 98% on room air. Initial exam reveals lungs are clear, regular rate and rhythm, abdomen soft and non-tender, pulses equal, no JVD, no pitting edema, A+O x 4.

They follow their protocol for chest pain and to rule out myocardial infarction (MI). Oxygen by 50% mask is applied; IV 0.9 NS KVO is begun. The Lead II rhythm strip reveals normal sinus rhythm (NSR) at 75 bpm with 1.5 mm horizontal ST elevation.

The patient is placed on the trundle at a 45° angle to prepare for transport and the crew prepares to follow protocol and place a second IV KVO line, administer a 1/150 SL nitroglycerin (NTG) and place 1/2" NTP on her chest wall (providing that systolic blood pressure (SBP) is greater than 90 mm/Hg). The protocol also calls for 2 mg morphine sulfate if needed per patient response.

The patient is placed in the unit. As the medic adjusts the O_2 mask, he notes that the patient appears to now have JVD. His first explanation is that she was at 90° for the primary examination and is now being evaluated at 45°. He repeats the chest exam, while his partner prepares the SL NTG and NTP. Lungs are still clear to exam, but now, being closer to the patient, it's obvious there's JVD.

Right after inspiration, the JVD rises to about two-thirds above the clavicle toward the angle of the jaw and falls to one-third between breaths. Protocol didn't require a call-in to the base station to discuss therapy at this point; however, the medic decides to contact the base-station physician before continuing with the protocol of SL NTG, NTP and MS.

The medic specifically wants to discuss if an NS (normal saline) bolus is warranted—even though the patient has JVD—before continuing protocol to avoid dropping the patient's blood pressure further and causing LOC.

The medic concisely presents the case to the base-station physician. The physician agrees with the bolus of 250 cc NS, and then re-assesses vital signs and chest exam. They're told to continue with protocol and call back for change of status. A 250 cc NS bolus is given.

At completion of the bolus, the vital signs were heart rate 70, blood pressure 115/75, respiratory rate 18, pulse oximetry 100% on 50% mask. The patient states the dizziness is resolved. Chest Pain/AMI protocol continues with SL NTG and 1/2" of NTP to chest wall, and in three minutes, the chest tightness has decreased. Blood pressure is now 105/65. The patient is admitted through the ED to the critical care unit (CCU) with acute inferior-wall and right-ventricular infarct (RVI).


PHOTO: KEVIN LINK

An automated blood pressure may give reliable SBP/DBP, but it won't indicate clinically diagnostic abnormalities.

In this case, the medic was concerned about the patient's relative hypotension. Remember, she's supposed to be a hypertensive patient. Although his protocols could have been justifiably followed without base-station consult, he was worried about the finding of JVD, clear lungs, no peripheral edema and a CNS complaint of dizziness. Had the protocol been followed without the bolus of NS prior to NTG/NTP therapy, the patient's blood pressure would have dropped precipitously. The medic was describing Kussmaul's sign to the base-station physi-

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cian.

The rise of JVD after inspiration is caused, in this case, by right-ventricle (RV) dysfunction due to right ventricular myocardial infarction (RVMI). As the patient inspires, the now low pressure in the thoracic cavity increases SVC and IVC return. Because the RV has lost its normal contractile ability, the RV is unable to handle this overload. The JVD worsens until it normalizes during exhalation, and the RV can eject the blood volume already present.

They were dealing with an acute myocardial infarction patient who also had CNS complaints; she was dizzy from relative hypotension. If they were to drop her preload to the right side of the heart by using NTG, NTP and MS, her blood pressure would have dropped much further, along with her cerebral perfusion pressure. Now they'd likely be dealing with an unconscious 70-year-old with an MI.

In addition, afterload may also decrease and, because the coronary arteries fill during diastole, cardiac perfusion pressure may also fall, thereby worsening oxygen delivery to the myocardium. You must remain concerned about a patient with a history of hypertension who's now relatively hypotensive and symptomatic. The human body can handle many large insults to its proper physiological function if they occur over time. Acute dysfunction doesn't allow the body to compensate adequately.

KUSSMAUL'S SIGN is the occurrence of increased jugular venous pressure (JVD), and appearance of JVD, with inspiration. It can be indicative of right ventricular contractility deficiency. Other usual findings are arterial hypotension and clear lungs. Normal physiology would find the JVD falling with inspiration due to reduced intrathoracic pressure. Kussmaul's sign can indicate venous return overload of the right ventricle due to failure of adequate systolic ejection. Other causes of Kussmaul's sign include constrictive pericarditis, pericardial effusion and restrictive cardiomyopathy.

CASE 3: TECHNOLOGY CAN FOOL YOU

A medic unit is dispatched for a 60-year-old male with a chief complaint of syncope. On arrival, they find the patient sitting in a chair. His wife is nearby. History of present illness reveals that the couple was eating breakfast when the syncope occurred. The patient had been well until this morning when, upon awakening, he complained of feeling dizzy and nauseated before breakfast. The patient's wife states he had been eating breakfast with her when he failed to respond to a question. When she looked up, the patient had slumped in his chair and was unresponsive. She went to his side to assist him and called 9-1-1.

The patient became alert spontaneously in less than one minute. He had apparently returned to his general baseline health, except for not being able to recall the event and his chief complaints of still feeling nauseated and dizzy. He responds to the medics' questions appropriately and is fully able to cooperate with the questions and commands for the medical history and physical exam.

The patient's wife reports no seizure-like activity. He had no significant past history, no known drug allergies and has been compliant with taking Toprol for hypertension for about two years. He states he had just seen his primary-care physician for his physical last week and was found to be "doing fine."

The patient is placed on the stretcher in the

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PHOTO: KEVIN LINK

Observe for ease or difficulty, regular or irregular, or patterned respiration in addition to rate.

kitchen with his head elevated at 45° so the evaluation, per protocol, could continue as he was readied for transport. Initial "automated" vital signs are as follows: heart rate 90/minute, blood pressure 120/75 mm/Hg, respiratory rate 16/minute, pulse oximetry 98% on room air. Primary exam reveals pupils equal and reactive to light, regular rate and rhythm, clear to auscultation, abdomen soft and non-tender, pulses equal and regular, deep tendon reflexes normal and equal, motor function normal and equal.

The medics begin protocol for syncope/LOC. Treatment includes O_2 6L NC, I/V KVO, cardiac monitor and finger-stick blood glucose, which equals 115. There's no obvious cause at this point for the patient's signs and symptoms. A 12-lead ECG is performed per protocol of syncope in a non-trauma adult.

The 12-lead ECG states "atrial fibrillation." The medics change their treatment protocol to new onset and symptomatic atrial fibrillation. Per this protocol, because the patient had a SBP greater than 90 mm/Hg and was currently alert, cardioversion wasn't indicated.

The medics place a second line and contact the base station as required for continuation of protocol pending administration of diltiazem or verapamil. The case is presented to the base-station physician. The medic discusses the patient's chief complaint of status post loss of consciousness with preceding and residual nausea and dizziness, history of present illness, past history including hypertension for two years treated with Toprol, vital signs, physical findings, normal blood-glucose reading and 12-lead ECG diagnosis of atrial fibrillation.

The medic tells the base physician, "We're following the atrial fibrillation protocol; however, cardioversion isn't indicated at present. Request administration of I/V diltiazem per protocol." The medic's partner repeats automated vital signs, which are essentially unchanged and stable, and prepares the diltiazem for administration. This data is relayed to the base physician.

During the medics' primary assessment, no dosage is given for the patient's current Toprol dose. The base physician, wanting to complete his own mental picture of the patient they're treating, calls back on the radio and asks the medics to determine the patient's current Toprol dose. They ask the patient what Toprol dose he's taking, and his wife hands them his prescription bottle. The prescription is written for "Toprol XL 100 mg, one by mouth every night after eating dinner."

The medic notes the bottle is almost full and the filling date was five days ago. The medic asks the patient if his physician had just refilled this prescription during his physical last week. The patient replies, "Yes, but when I see her again, I will tell her that I always felt fine

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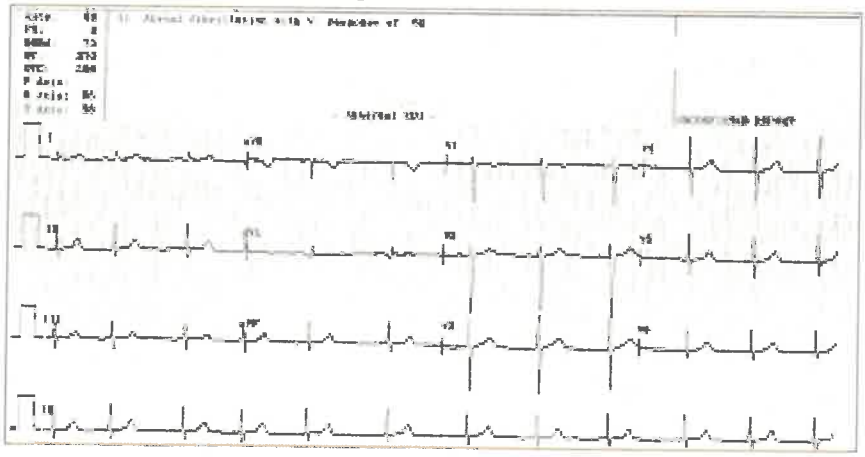
with the 50 mg pills, but she said I would feel even better with these 100 mg pills."

The medic calls the base physician and discusses the recent increase in Toprol dose from 50 mg to 100 mg. The medic states the patient is still stable and requests they hold treatment for atrial fibrillation, and transport and monitor the patient for an additional diagnosis of beta-blocker toxicity. The physician agrees.

The patient arrives in the ED, and the base physician takes report from the medics. They discuss the clinical issues of this interesting case that progressed from a diagnosis of "syncope" to a diagnosis of "new onset atrial fibrillation" to a diagnosis of "drug toxicity." They discuss the obvious danger of treating atrial fibrillation with a calcium-channel blocker in a patient that already has beta-blocker toxicity.

As the discussion continues, the base physician is organizing the EMS data sheets, the ED intake sheets and the 12-lead ECG with it's computer reading of "atrial fibrillation with V-response of 69" and "abnormal ECG." The ECG catches his eye because of the relatively slow regular rhythm. The base-station physician evaluates the 12-lead ECG more closely

Figure 1: Clinically Interpreted Diagnosis of 12-Lead ECG = NSR



(see Figure 1, above). The physician realizes that not only was a patient with beta-blocker toxicity almost treated with a calcium-channel blocker—which assuredly would have worsened the patient's condition—but they also came close to treating a patient for a diagnosis of atrial fibrillation, which he did not have.

The correct clinical interpretation of the 12-lead ECG is normal sinus rhythm (NSR) with ventricular response of 69 bpm. There appears to be some slight sinus arrhythmia/respiratory rate change, but there are clearly conducted "P" waves.

This patient had a fairly straightforward drug reaction due to a recently increased dose of Toprol. This new dosage decreased his blood pressure too rapidly. Additionally, because Toprol is a beta-blocker, it reduced his ability to compensate for a symptomatic decrease in blood pressure.

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The normal physiological compensation to symptomatic decreased blood pressure is an increased heart rate. This reflexive increase in heart rate is intended to maintain his cardiac output and cerebral perfusion pressure. The patient's inability to physiologically compensate for decreased blood pressure caused his nausea, dizziness and syncope.

CASE RESULTS

The above cases demonstrate, as advanced as medial technology and devices have become over the past 30 years, their "numbers" don't tell the entire story. To begin with, these medical devices don't perform a medical history for us. Traditionally, the patient's history gives us more than 75% of our diagnostic data.

In the first case, the patient's correct diagnosis was determined by an alert medic based on a piece of new, albeit accidentally derived, patient history. In the second case, the proper care of the patient was dependent on the medic's re-evaluation of his physical findings; he didn't automatically proceed with a potentially harmful protocol. In the third case, the medic felt that, although there were multiple new diagnoses, the patient appeared otherwise stable. At that point, he felt it was in the patient's best interest to transport to avoid further complications. Also, in this third case, we see that the medical device, the prehospital 12-lead ECG with its computerized diagnosis, rendered erroneous data, which almost led to unneeded and potentially harmful treatment.

The cold output of medical-device data has numerous limitations: The heart rate on a monitor doesn't equal pulse rate, which implies we actually touched the patient. We not only palpate for quantity of pulsations but also quality. Many diagnostic clues are gained by characterizing the palpated pulses. Blood pressure by automated device may give us reliable SBP/DBP, but it won't tell us if there was any evidence of pulsus paradoxus or any other type of clinically diagnostic abnormality. Respiratory rate calculated by various current medical devices isn't the same as observing the patient during respiration. Observing respirations for ease or difficulty, labored or not, regular or irregular, or the presence of patterned respiration, such as Cheyne-Stokes respiration, is clinically significant. The data from our devices is important to modern medical care standards; however, as clinicians, we must use this data appropriately and be ready and able to clinically validate, or invalidate, any data gained.

A working knowledge of anatomy, physiology and pathophysiology are essential foundations for your history, physical and diagnostic skills. *DeGowin's Diagnostic Examination* and *Bates' Guide to Physical Examination* are excellent references for diagnostic skills that will expand and enhance your clinical acumen.

Our patients place their trust in our clinical skills to help them if we can, and certainly to avoid harming them in the process. We must be willing to continue honing our clinical skills with education, training and experience, with the goal of learning something from every patient encounter. We're in the serious business of reducing pain and suffering, and decreasing morbidity and mortality in patients we've never met before. I don't know of a heavier responsibility. JEMS

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