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An 84-year-old woman with hypertension and type 2 diabetes mellitus is brought to the emergency department (ED) after an episode of near syncope. When emergency medical service personnel initially assessed her, blood pressure was 96/60 mm Hg and heart rate was "slow"; however, she had no symptoms. In the ED, the patient is awake and comfortable. She has no chest pain, nausea, or diaphoresis. Temperature is 37.2°C (98.9°F); heart rate, 78 beats per minute; respiration rate, 24 breaths per minute; blood pressure, 100/58 mm Hg; and oxygen saturation, 95% on room air. Slight jugular venous distention is noted. Rales are audible at both lung bases, and heart rate is regular, without murmurs, gallops, or rubs. The abdomen and lower extremities are normal. The patient's initial ECG is shown here. The computer interprets the tracing as "normal sinus rhythm; low-voltage QRS; septal infarct, age undetermined." Which of the following conditions is best supported by the ECG findings?

A. Pulmonary embolism.
B. Hypothyroidism.
C. Acute myocardial infarction.
D. Pericardial effusion with possible tamponade.
E. Myopericarditis.

THE COMPUTER ISN'T ALWAYS RIGHT

The diagnostic performance of 9 computer programs for ECG interpretation was analyzed, with mixed results. Although the median performance of the programs across a number of diagnoses was strong (91% correctly classified) and only slightly below that of the cardiologists who served as the gold standard (96% correctly classified), the computer interpretations were notably unsatisfactory in cases of MI, especially inferior MI (median number of cases correctly classified, 59%). Another study looked at computer ECG interpretation specifically as it pertained to acute MI. The positive predictive value of computer interpretations of the ECG (94%) exceeded that of the physicians tested (86%); however, the negative predictive values of both were lower (computer interpretations, 81%; physicians, 85%). This is a key point, given that the ECG is used primarily as a screening tool and thus requires high sensitivity. ECGs are also used for point-in-time exclusion of a diagnosis (eg, acute MI, especially in patients who need emergent percutaneous coronary intervention or fibrinolysis)—another setting in which a high negative predictive value is desirable. Doubtless most clinicians would have admitted this patient and proceeded to "rule out" MI with serial ECGs and measurements of cardiac serum markers. Thus, the diagnosis of acute MI would eventually have been made. However, the opportunity to reduce morbidity and mortality through early revascularization, or even aspirin and heparin therapy, might have been missed. Meanwhile, the diagnosis was available all along on the ECG. It would be unwise to ignore a computer printout that blares "acute MI" across the top of the tracing (even though other diagnoses, such as myopericarditis, ventricular hypertrophy with repolarization abnormality, benign early repolarization, and ventricular aneurysm, should also be seriously considered when that computer diagnosis appears). However, it is also unwise to be reassured by the absence of a computer diagnosis of MI.

INTERPRETING THE LOW-VOLTAGE ECG

Diminished amplitude of the waveforms on the ECG, or "low voltage," is generally defined as a QRS amplitude of less than 0.5 mV (5 mm) in all limb leads and less than 1.0 mV (10 mm) in all precordial leads. Low voltage is a nonspecific finding: it is associated with various causes of fluid retention (myxedema, congestive heart failure, nephrotic syndrome, and anasarca), pericardial effusion, pleural effusion, pneumothorax, chronic obstructive pulmonary disease, obesity, and infiltrative cardiomyopathies. In short, anything that comes between the ECG electrode and the heart--distance, air, fat, fluid, etc--may diminish the amplitude of the waveforms on the ECG. Thus, a finding of low voltage should prompt a search to exclude the above-mentioned syndromes. A previous ECG is often invaluable in establishing the newness of the process. Results of thyroid studies were normal in this woman, which ruled out hypothyroidism (choice B). No pericardial effusion (choice D) was seen on an echocardiogram, and a chest radiograph showed no evidence of...
pneumothorax, pulmonary edema, or pleural effusion. The finding of low voltage on her ECG was likely caused by her body habitus. Low voltage also affects the degree of ST-segment elevation/depression and Q-wave amplitude. Typically, ST-segment elevation is considered significant only when it is greater than 1 mm in the limb leads. In a patient such as this woman, Q-wave duration/amplitude would be considered significant only if it were 0.03 second or greater in lead II or lead aVF or 0.04 second or greater in lead III (or if the Q-wave depth were more than 25% of that of the corresponding R wave). However, when R-wave amplitude is low, ST-segment elevation is correspondingly low. Thus, the degree of ST-segment elevation regarded as significant needs to be modulated when an ECG shows low voltage. In addition, wave width is now thought to be more important than depth in the determination of whether Q waves are pathologic. Although myopericarditis (choice E) could produce the degree of ST-segment elevation seen here, this diagnosis is unlikely for several reasons:

- The regional (rather than diffuse) nature of the ST-segment elevation.
- The absence of PR-segment depression.
- The presence of reciprocal changes (the ST-segment depression seen in lead aVL), which increases the specificity of the ECG findings for a diagnosis of acute inferior wall MI.
- The absence of clinical features of pericarditis (pericardial friction rub and pleuritic chest pain that worsens with supine positioning).

**CLINICAL DIAGNOSIS AND THE ECG**

In a patient who has had an episode of syncope or near-syncope, give early consideration to cardiac causes of ischemia and dysrhythmia. (Although it is less likely, also consider outflow obstruction, which could result from aortic stenosis, pericardial tamponade, or other conditions.) Consider as well other catastrophic causes of syncope, such as pulmonary embolism (choice A) and aortic dissection (which may be painless, especially in the elderly). Because of this woman's history of transient low heart rate and low blood pressure following her near-syncope episode, the diagnosis of acute MI—and in particular, acute inferior MI—moved up in the list of differential diagnoses. Thus, the ECG was examined with special attention to rhythm disturbances (any degree of atrioventricular heart block, sinus or junctional bradycardias) and inferior ischemic changes. Reciprocal ST-segment depression (seen here in lead aVL [see Figure 1]) adds specificity to an ECG diagnosis of acute ST-segment elevation MI. Lead aVL is the key lead to examine for this finding when acute inferior infarction is suspected; it lies roughly opposite (150 degrees in the opposite direction) to lead III in terms of the QRS frontal plane axis vector. Finally, the fact that the ST-segment elevation was greater in lead III than in lead II was a clue to the anatomy of the lesion; such a finding indicates that the right coronary artery is likely the culprit vessel. (ST-segment elevation in lead II equal to that in lead III, on the other hand, is a strong predictor of left circumflex coronary artery occlusion.) An easy way to remember this is to recall that lead III is more rightward in its vector than lead II, which is more leftward.) Another reason to consider a diagnosis of MI early on (when warranted by the clinical picture and ECG) is that ECG findings are not only subtle at times but also can be transient. Note the changes—or lack thereof—in this patient's second tracing, obtained just 20 minutes after the first one (Figure 2). The ST segments are less elevated in the inferior leads, and the ST-segment depression in lead aVL is resolving.

**References:**


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