Unusual electrocardiogram in a man with chest pain

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A 56-year-old man came to the hospital emergency department with chest pain, and an electrocardiogram (ECG) showed numerous abnormalities (*Figure 1*). A striking one was the extremely low voltage in lead II, indicating essentially no electrical potential difference between the two electrodes. This finding has usually occurred when the right arm and left leg electrodes, the electrodes for lead II, have both been on the legs and, thus, are an equally long distance from the heart. The cause most often has been the right arm lead's being on the right leg and vice versa. The lead malplacements in this case, however, appeared to be more complex. The right arm lead was probably on the right leg, but the right leg lead (the ground) was probably on the left arm. The left

arm lead was probably on the right arm, and the left leg lead was correctly placed. Such placement would have accounted for the negative P waves and QRS complexes in leads I and aVL and the fact that lead III had the appearance of lead II in an ECG with properly placed leads (*Figure 2*). Furthermore, in *Figure 1*, the chest leads appear to have been on the right side of the chest.

With the electrodes placed correctly (*Figure 2*), the ECG had most of the features of acute anterior myocardial infarction due to left anterior descending occlusion proximal to the first major septal perforating branch and the first diagonal branch: ST-segment elevation in lead aVR, ST-segment depression in lead V₅, ST-segment depression in leads II and III and/or aVF

Figure 1. Electrocardiogram recorded in the emergency department in a 56-year-old man with chest pain. See text for explanation.

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Figure 2. Electrocardiogram recorded with the electrodes placed properly. See text for explanation.

≥ 1 mm when $1.0 \text{ mV} = 1 \text{ mm}$, Q waves in lead aVL ≥ 0.03 seconds, and ST-segment elevation in lead aVL ≥ 1 mm.^{1,2} Absent was ST-segment elevation in lead V₃, a virtually universal finding in acute transmural anterior myocardial infarction. The explanation for this was that the infarct was not transmural in all locations.

Coronary arteriography showed total occlusion at the origin of a wraparound left anterior descending with retrograde filling of the left anterior descending back to its origin via collaterals from the right coronary artery. This collateral flow was sufficient to limit the infarct to the subendocardial myocardium in all areas supplied by the left anterior descending except for the high anterolateral wall of the left ventricle where ST-segment elevation was seen. Elsewhere there was ST depression.

The occlusion was uneventfully opened with a balloon and stented. The following day the ECG showed widespread T-wave inversion with less ST depression and no ST-elevation. The only Q waves were the small ones seen in I and aVL in Figure 2.

Electrode malposition, a bane to the electrocardiographer, is common but rarely as egregious as in Figure 1.³ No seasoned ECG technician would have made such an error. This ECG was recorded shortly before midnight when none of the regular ECG technicians were in the hospital, and ECGs at such times have been performed by less-trained personnel. Despite the lead misplacements in Figure 1, evidence of myocardial injury was obvious, as was the need for a properly recorded ECG.

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