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0196-0644/$-see front matter

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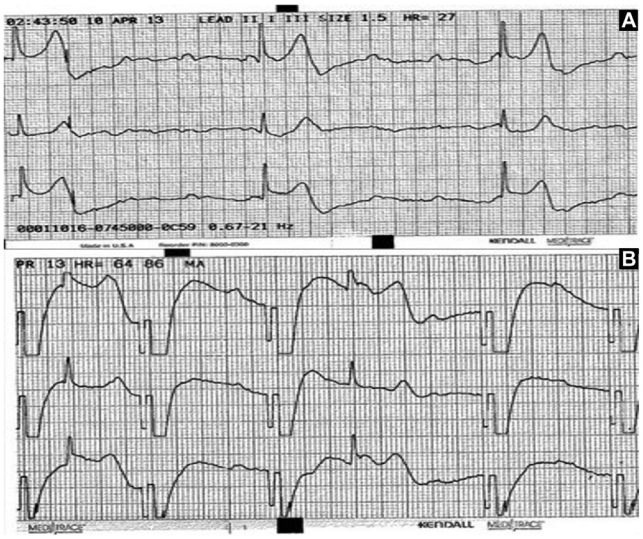

Figure 1. Three-lead rhythm strips (leads order: II, I, and III): A, on presentation; B, postexternal pacing.

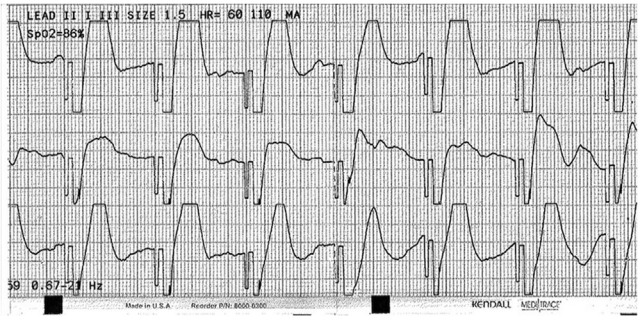

Figure 2. Three-lead rhythm strip (leads order: II, I, and III) after pacemaker output was increased from 86 MA to 110 MA.

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A 65-year-old man called emergency medical service (EMS) because of chest pain and diaphoresis. The EMS team found him hypotensive and bradycardic. His field 3-lead rhythm strip showed inferior ST-elevation myocardial infarction (STEMI) with heart block (Figure 1A). Intravenous atropine was given with no response; therefore, external transcutaneous pacing was initiated, and he was taken to the hospital (Figure 1B). However, he remained hypotensive en route.

For the diagnosis and teaching points, see page 74.
To view the entire collection of ECG of the Month, visit www.annemergmed.com

ECG OF THE MONTH

(continued from p. 73)

CLINICAL QUESTION

What went wrong? How could it have been corrected?

DIAGNOSIS:

ECG INTERPRETATION

In Figure 1A, the tracing shows inferior STEMI with heart block (P wave rate of 100 bpm with dissociated, narrow QRS complexes at a rate of 27 bpm).

In Figure 1B, there are two groups of complexes (best seen in leads I and III): the first group is prominent, wide negative complexes following each pacing spike, and the second group is less prominent, narrow positive complexes (the first is seen immediately after the first large negative deflection, the second one is seen after the third large negative deflection, and the next one is expected to coincide with the last wide deflection, hence will not be visible). These narrow complexes are similar (including associated ST-T waves) and rate (27 bpm) to the presenting beats (Figure 1A). Hence, they likely represent the persistence of the underlying presenting rhythm marching through uninterrupted pacing.

This lack of native rhythm interruption questions the effectiveness of pacing in capturing myocardium and taking over, or at least resetting, the underlying rhythm. Therefore, wide negative QRS complexes are unlikely to represent true myocardial activation.

Furthermore, the occurrence of the first native narrow beat shortly (<160 milliseconds) after the first wide complex makes it physiologically implausible to assume that this wide complex represents myocardial activation. These wide complexes may be generated by system saturation after large pacing spikes or skeletal muscle capture but not by actual myocardial capture. The lack of hemodynamic response to this pacing strip further supports this assumption.

This lack of myocardial capture is likely secondary to insufficient output (86 mA shown on the top-left part of Figure 1B).

In Figure 2, the pacemaker output was increased to 110 mA (top-left part of tracing), leading to actual myocardial capture. The tracing now shows continuous pacing with wide complexes at a rate of 60 bpm. Two important clues suggesting myocardial capture are noted: the elimination of underlying dissociated native slow, narrow complexes noted previously, and the presence of very tall and distinct T waves following the wide paced complexes, suggesting ventricular repolarization. These tall T waves were not seen in Figure 1B when the pacemaker did not activate the myocardium.

These electrocardiographic findings of myocardial capture were supported clinically by paramedics feeling the patient's pulse and led to significant improvement in the patient's hemodynamics upon arrival to the hospital.

CLINICAL COURSE

The patient underwent successful stenting of the right coronary artery with the resolution of symptoms. He was discharged home 48 hours later without needing a permanent pacemaker.

DISCUSSION

Transcutaneous pacing is a quick way to support a slow pulse rate in emergency situations. The high output delivered by external pacemakers typically inflects large complexes on the ECG at the paced rate. However, these complexes do not necessarily represent actual myocardial activation and may give the rescuer a false sense of security by distracting attention from the underlying, uninterrupted slow heart rhythm. Thus, the hemodynamic response to ventricular capture should be confirmed clinically by evaluating the patient's pulse.

Rescuers should be vigilant and look specifically for confirmation of actual myocardial capture. Consistent ventricular capture should take over and eliminate any underlying dissociated slower native rhythm and is typically

associated with discrete T waves following these large complexes. Therefore, rhythm strips should be searched carefully for evidence of dissociated native beats, which can be subtle and masked by these large artifacts.

When ventricular capture is uncertain, the pacemaker output should be dialed up gradually until capture is confirmed electrocardiographically and clinically. However, increasing the pacemaker output may not be tolerated by patients who are awake; hence, sedation to control pain should be considered.

TEACHING POINTS

- Electro-hemodynamic correlation by confirming a pulse that corresponds with a pacing impulse is the most definitive way to confirm myocardial capture by external pacemaker.
- When using external pacemaker, the mere presence of pacing spikes followed by large complexes at the paced rate does not necessarily mean that the heart is being paced at that rate. Rescuers should specifically look for confirmation of myocardial capture as described above. Placing monitor leads away from pacing pads may minimize the confusing artifacts in these leads.[1]
- Low pacemaker output selection (typically driven by concerns about the patient's discomfort) may not capture the heart. Therefore, enough sedation is crucial to deliver enough output that captures the myocardium.
- Placing pacing pads close to the heart (eg, the anteroposterior position) can potentially enhance ventricular capture at a lower output.[2]

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