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LETTER

Attenuated ECG Voltage in Patients after Cardiac Surgery

To the Editor:

I enjoyed the thorough analysis of the pre- and postoperative ECGs, provided by Do, Tran, and Schweitzer of the patient who underwent aortic valve replacement and single-vessel coronary bypass.[1] I agree with all the diagnostic considerations discussed by the authors, whose primary objective was to focus on the underlying postoperative cardiac arrhythmia and its pathophysiological underpinnings, causes and management. I would like to draw attention to an additional finding in the post-operative ECG, which occurs frequently and rarely receives a commentary, ie, attenuated voltage. When commenting upon this, reference is usually made to the attenuated QRS complexes in the cardiac postoperative setting and is attributed to pericardial effusion, pleural effusion, hydromediastinum, pneumothorax, pneumopericardium, pneumomediastinum, or other "nonspecific" postoperative influences. Although the above cited causes are often at the root of the problem, a frequently present alternative mechanism for such ECG voltage attenuation is peripheral edema of varying etiology.[2]

Looking at Figure 2 of the patient's postoperative ECG,[1] one appreciates that the QRS complexes of the conducted atrial ectopic beats in the limb leads are at least 2 1/2 times lower in amplitude than on the preoperative ECG (Figure 1). The same and even more applies to the precordial ECG leads, even accounting for the half standardization of these leads in the Figure 2 (compare the QRS amplitude of beats #6 and #10 of ECG in Figure 2 [after doubling the amplitude] with the amplitude of the QRS complexes of the corresponding leads of Figure 1).[1] Often such QRS attenuations lead to elimination of the diagnosis of ECG-based diagnosis of left ventricular hypertophy (LVH),[3] as indeed happened in the case of the patient under discussion, where diagnosis of LVH cannot be made in the ECG in Figure 2 either by the Sokolow-Lyon or Cornell voltage criteria.

Since the mechanism of the body fluid overload-based ECG voltage attenuation is extracardiac in origin,[1] not only the QRS complexes but the P-waves also are affected.[4] Accordingly if one scrutinizes the 2 ECGs, and even if one accounts for the half standardization of the ECG in Figure 2, the ectopic P-waves have become attenuated and almost imperceptible in the ECG of Figure 2. Indeed, the attenuation is so intense that often an erroneous diagnosis of "junctional rhythm" is made in patients who are shown to have sinus P-waves when using intracardiac electrograms.[5] Many other changes are imparted on the ECG as a result of this peripheral edema-derived attenuation of the ECG voltage that cannot be discussed here in the space allotted to a letter, but are amply documented in the literature of the author of this communication.

The mechanism of ECG attenuation is a short-circuiting of the epicardial ECG potentials, as they are transferred to the body surface, and it is due to the decreased impedance of the passive volume conductor imparted by the low resistivity of the retained fluids.[1] In the postoperative setting this could be due to congestive heart failure, renal failure, or simply administration of excessive fluid loads, even in patients with normal hearts.[6] What was the mechanism in this patient?

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