Case Report

Electrocardiogram voltage attenuation and shortening of the duration of P-waves, QRS complexes, and QT intervals

John E. Madias\textsuperscript{a,b,*}

\textsuperscript{a}The Icahn School of Medicine at Mount Sinai of the New York University, NY, USA
\textsuperscript{b}The Division of Cardiology, Elmhurst Hospital Center, Elmhurst, NY, USA

\textbf{ABSTRACT}

Multiple pathologies in concert may lead to attenuation of the electrocardiogram (ECG) voltage. A case of a patient illustrating the above is presented, who showed marked attenuation of the ECG voltage. Automated values of the amplitude of the ECG QRS complexes, P-waves, and T-waves (in mm), duration of the QRS complexes, P-waves, and QT intervals (in ms), in 2 ECGs were compared. The patient was a 64-year-old woman who developed in the setting of a fatal illness, pleural and pericardial effusions, pneumomediastinum, pneumoperitoneum, subcutaneous emphysema in the neck and chest, peripheral edema with weight gain of 43.4 lbs, marked hypoalbuminemia, abnormal liver tests, and renal failure. All the above pathologies led to a marked attenuation of the ECG voltage, and shortening of the mean P-wave, QRS complexes, and QTc interval durations. The postulated mechanism of the observed ECG phenomena is discussed.

\textbf{1. Introduction}

There are many factors which singly, on in concert, can influence the electrocardiogram (ECG) voltage and lead to attenuation of the P-waves, QRS complexes, and T-waves. The resultant effects are extracardiac in nature. What is not appreciated is the resultant shortening of the P-wave, QRS complex and QT intervals. The case of a patient presented herein illustrates all the above ECG changes resulting from multiple intrathoracic and systemic pathologies, which the patient suffered.

\textbf{2. Case presentation}

A 64-year-old woman, with a history of hypertension, failure to thrive, weight loss of undefined etiology worked up at another hospital, was admitted to our facility with dyspnea, hypoxemia, and lung infiltrates, for which she was intubated, and placed on broad spectrum antibiotics and vasopressors. She abruptly decompensated, and was found to have pleural and pericardial effusions, pneumomediastinum, pneumoperitoneum, subcutaneous emphysema in the neck and chest, and peripheral edema with weight gain of 43.4 lbs (Fig. 1).
Exploratory laparotomy did not reveal any intestinal perforation in the abdomen, and the tip of an endotracheal tube was seen proximal to the carina; a short axis parasternal view of the echocardiogram on day 6 of hospitalization showed normal left ventricular size and thickness, with a large pericardial space which contained a large amount of “organized” echo-dense material, adherent to the epicardium and a pericardial effusion; an axial view of the thoracic computerized tomographic angiography on day 2 of hospitalization revealed a prominent right pleural effusion, no parenchymal mass lesions, mediastinal and subcutaneous emphysema, right lower lobe infiltrate/atelectasis, pericardial effusion, suggestion of possible pneumatic pericardium, but no pleural effusion. Subcutaneous emphysema in the neck resolved but her respiratory failure worsened and she developed adult respiratory distress syndrome, shock with hypotension, tachycardia and peripheral hypoperfusion, marked hypoalbuminemia, abnormal liver tests, and renal failure. The family opted for extubation and no resuscitation, and the patient succumbed on day 7 of hospitalization.

Comparison of the 2 ECGs recorded 7 days apart (Fig. 2) (Table 1), revealed minor changes in heart rate (118–119 beats/min), P-wave frontal axis (55°–53°), QRS frontal axis (−14° to −5°), and T-wave frontal axis (96°–73°), while sums of peak-to-peak QRS complexes of all 12 leads decreased by 75% (81.68–20.44 mm), sums of peak-to-peak P-waves by 55.0% (11.0–4.95 mm), and sums of peak-to-peak T-waves by 58% (17.48–7.28 mm). This was associated with shortening of the mean P-wave duration by 26.5% (90–66.18 ms), QRS duration by 19.4% (72–58 ms), QT interval by 8.5% (328–300 ms), and QTc by 8% (460–423 ms).

3. Discussion

What precipitated the ECG amplitude changes was the combination of pneumonia, acute respiratory distress syndrome, pericardial effusion, pleural effusion, mediastinal and subcutaneous emphysema, hypoalbuminemia, and peripheral edema. All these pathologies either enhance the water content (thereby resulting in reduction of electrical impedance) or produce marked disparities in the boundary of layers of the passive volume conductor (e.g., emphysema) leading to attenuation of all components of the ECG curve (extracardiac phenomenon). What is not as much appreciated is that such attenuation results in shortening of the durations of P-waves, QRS complexes and T-waves. The postulated mechanism for
this shortening is that the overall decrease in the amplitude of the P-waves, QRS complexes and T-waves introduces a measurement error of a portion after the onset and before the offset of these components of the ECG curve, which become as low as the underlying noise amplitude, and thus they are lost in the automated ECG measurement.\textsuperscript{5,6} All the above have significant diagnostic, monitoring, and prognostic implications in the setting of lung infections, lung congestion, pericardial and pleural effusions, emphysema in any location, hypoalbuminemia, and peripheral edematous states, encountered in clinical practice or research. Consequently knowledge about the association of these pathologies and ECG amplitudes of complexes, waves, and durations of intervals, for all clinicians caring for patients, is contributory to correct ECG-based diagnosis. However the above should be considered with caution when interpreting changes in the P-wave duration and dispersion (i.e., difference between the longest and the shortest P-wave duration among the 12 ECG leads, in ms) in the setting of development and amelioration of increased water content in any extracardiac body compartment. Indeed, mean P-wave duration P-wave dispersion did not change in patients who developed peripheral edema during a variety of critical illnesses, and their stability was attributed to the offsetting of the electrophysiologically-mediated real changes (due to hemodynamic perturbations), by opposite apparent changes, imparted by the effect of the peripheral edema, as described above.\textsuperscript{7} Another matter of practical importance is that such changes in voltages/duration of ECG QRS complexes, exerted by extracardiac influences, may carry special bearing in patients with ischemic and nonischemic cardiomyopathies and heart failure, who are being considered for implantable cardioverter-defibrillation implantation, or cardiac resynchronization therapy, since such decisions often are based, among other parameters, on the duration of the QRS complexes.\textsuperscript{8}

\textbf{Conflicts of interest}

The author has none to declare.

\textbf{REFERENCES}

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