CASE REPORT

Amplitude of the Electrocardiographic QRS Complexes during and after Severe Pulmonary Edema

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There is recent evidence that anasarca peripheral edema, irrespective of its etiology, attenuates ECG QRS potentials. Pulmonary edema (PE) also is thought to cause reduction in the amplitude of QRS complexes. The case reported herein is of a patient with severe PE, hypertension, left ventricular diastolic dysfunction, and no peripheral edema who did not show changes in the QRS complexes with the management of her pulmonary edema. Thus it appears that PE does not attenuate the amplitude of QRS complexes, and alleviation of this condition does not cause augmentation of QRS voltage. This is in contrast to alterations of peripheral edematous states in the setting of congestive heart failure, which result in changes in the QRS amplitude, as shown previously.

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ECG; anasarca pulmonary edema; peripheral edema; congestive heart failure; QRS complexes;
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Changes in the geometry and the conductivity of the different organs and tissues interspersed between the heart and the body surface (volume conductor) have a profound effect on the transfer of the epicardial potentials to what is recorded as the standard 12-lead ECG.^{1,2} The conductive properties of the lungs exert a major influence,³ and elegant work by Rudy and Plonsey employing a theoretical "eccentric spheres heart and torso" model, predicted reduced surface potentials for both high-lung conductivities (as seen in the setting of chronic obstructive lung disease and emphysema) and low lung conductivities (as found in the presence of pulmonary edema (PE)).⁴ Furthermore, these workers and their colleagues have shown, by theoretical simulations with their model and experience with bronchopulmonary lavage in three patients (one with cystic fibrosis and two with alveolar proteinosis) in whom filling of one of the lungs at a time with normal saline was undertaken, that the resultant decreased lung conductivities led to attenuated surface QRS potentials.⁵

By contrast in other work,⁶ management of PE in four patients did not produce the anticipated increase in the amplitude of QRS complexes. Also in four other patients treated for congestive heart failure (CHF),^{7,8} management of PE, which was also present early on admission in three of these four patients did not have any influence on the amplitude of QRS potentials; however, the effective diuresis implemented over the course of hospitalization led to subsequent significant augmentation of the amplitude of QRS complexes, which could only be traced to the loss of weight that these patients experienced from the resolution of their fluid overload. Ishikawa et al. described increase in the QRS potentials with management of CHF,⁹ which reluctantly, by their admission, they attributed to the change in the cardiac volumes due to therapy. However, decrease of cardiac volumes with effective management of CHF is expected to produce the opposite effect (i.e., decrease in the QRS voltage) than the one observed,^{10,11} prompting Rudy et al. to attribute the initial lower QRS potentials

prior to therapy in that study⁹ to an interplay of two opposing forces, one (the increase of cardiac volume) causing augmentation of such potentials, and the other (PE) producing attenuation of voltages, with the latter predominating.⁵

Certainly this interplay increases the complexity of the matter, and hinders mechanistic coronary care unit, with *just* severe transient PE, *without* evidence of peripheral edema, dilated cardiomyopathy, or significant loss of weight during hospitalization provided the opportunity to study the impact of *only* PE on the QRS potentials.

CASE REPORT

The patient was a 51-year-old obese black woman admitted with severe dyspnea following a low-impact automobile accident, in which she was involved as a restrained driver. She had a history of hypertension, diabetes mellitus type 2, and hyper-

cholesterolemia, and she took furosemide, a K⁺ supplement, quinapril, clonidine, and metformin, although there was a question as to her compliance with her medication. She did not complain of chest pain, and a myocardial infarction was ruled out by ECG and enzymatic testing. Some mild neck discomfort due to the accident was subsequently evaluated by CT scan, which was found to be negative. Auscultation on admission revealed pulmonary rales bilaterally, accentuation of the S2 at the base of the heart, and an S4 sound at the lower left sternal border; jugular venous pressure and pulse could not be evaluated due to obesity of the neck; peripheral pulses were felt bilaterally, and no peripheral edema could be demonstrated by deep palpation. Blood pressure was 210/120 mmHg, heart rate was 111 beats/min, and oxygen saturation was 70% by pulse oxymetry. An ECG (Fig. 1) showed sinus tachycardia, left ventricular hypertrophy and left atrial abnormality.

Standard ECGs on days #1, #2, and #7

Figure 1. Standard 12-lead ECGs are displayed, with the corresponding Σ QRSs in mm, inscribed on the ECGs. The six Xs denote a superimposed artifact during the recording of this ECG, which could be easily separated from the real QRS complexes during measurements in the original tracing.

Chest radiographs on day #1 and day #2 of hospitalization

Figure 2. Chest radiographs done on admission and day 2. Selected leads from the corresponding ECGs with measurements of QRS amplitudes are displayed. The Σ QRSs from admission and day 2 ECGs were 245.5 mm and 207.5 mm, correspondingly.

A chest radiograph (Fig. 2) obtained less than 1 hour after the first ECG, showed dense PE resembling the radiological picture of "white lung," seen in cases of severe acute respiratory distress syndrome. The patient was intubated, received intravenous nitroglycerin, furosemide, artificial ventilation with 100% FiO₂ oxygen, and she improved rapidly, showing 99% oxygen saturation, blood pressure 165/92, and heart rate of 115 beats/min, with further drop of these last two parameters over the ensuing hours. On the next day the patient was extubated, diuresis with intravenous furosemide was continued, and quinapril and clonidine were added; she also required insulin for the management of her diabetes. Beta-blockers were avoided, since the patient allegedly had a history of asthma, which we could not confirm. The blood pressure on the day after admission was 140/60. Over the course of 7 days the patient lost 2.2 kg from her weight of 104.0 kg obtained on the second day of her hospitalization.

The patient had daily chest radiographs for the initial 5 days; also a total of nine ECGs (two, on day

1 and 5) were recorded. The amplitude of the QRS complexes (from zenith to the nadir) in mm was measured to the nearest 0.5 mm of the 12 leads of all ECGs, using a magnified lens and calipers under strong overhead light, and the sum of all the measurements (Σ QRS) was used in the comparisons. Of particular interest was the initial drop of the Σ QRS over the course of the first 7½ hours, when a second ECG was recorded, with subsequent stability over the course of hospitalization. The chest radiograph of day 1 showed massive PE, while the study of the next day (21½ hours later) showed almost complete clearing (Fig. 2), with subsequent three radiographs being completely normal, without evidence of congestion, infiltrates, or pleural effusions. The heart size was normal in all radiographs, particularly if one considers that the studies were portable antero-posterior films, which as a rule magnify the heart size. However, one could read some underlying cardiomegaly in the first radiograph (Fig. 2), although the presence of severe PE prevented accurate detection of the heart's borders. Σ QRS from the nine ECGs were 245.5 (first ECG), 205.0 (ECG

6½ hours later), 207.5 (ECG of day 2), 204.0, 206.0, 195.5, 206.5, 209.0, 215.5, and 206.0 mm (Figs. 1 and 2). The mainly inverted P wave in lead V1 of the ECG of days 1 and 2 was replaced by a positive/negative P wave, and finally a mainly positive P wave was inscribed, suggestive of decreasing left atrial and pulmonary venous pressure or left atrial size.^{12,13} Differentiation between change in left atrial pressure and left atrial size is not feasible electrocardiographically. Since Rudy et al.⁵ described that the antero-posterior vector and the scalar Z lead were those that showed maximal decrease with the lung lavage, leads V1 and V2 would be expected to be the ECG leads with the most pronounced amplitude augmentation with elimination of PE. The QRS complexes of V1 from the nine ECGs measured 24.0, 19.0, 25.0, 23.5, 27.0, 27.5, 25.5, 23.5, and 25.0 mm. The corresponding values for V2 were 30.0, 29.0, 32.5, 30.0, 31.5, 34.0, 32.5, 32.5, and 24.0 mm. Thus for both these two leads there was no change after elimination of PE, by comparison of the first ECG with the second or third ECG, encompassing the first 24 hours of hospitalization. An echocardiogram, done on day 3, showed normal left ventricular size, left ventricular hypertrophy (septum was 20 mm, and in the posterior wall 17 mm), hyperdynamic left ventricular function with an estimated ejection fraction of ~75%, diastolic dysfunction, and mild left atrial enlargement, (left atrial dimension 40 mm, with a left atrial area being 22.5 cm²). The patient was discharged on day 7, to be followed at the Cardiology and Diabetes Clinics.

DISCUSSION

Severe PE and subsequent resolution in our patient were associated with an initial 16.5% drop of the Σ QRS, with subsequent stability. The stable course of Σ QRSs corroborates our previous experience with a total of seven patients treated for PE, who did not show changes of QRS potentials with management of this condition.^{6–8} What differentiates this patient from those seven others except that she showed an initial drop of Σ QRS, is that her PE was the most severe encountered, and that she did not have associated peripheral edema; those seven patients had radiological evidence of pulmonary congestion, but they were not critically decompensated from the clinical or oxygen saturation points of view. Also data for those patients included serial daily ECGs and chest radiograms

routinely done, while in the present case the first chest radiograph and ECG were obtained in temporal proximity, and a second ECG was recorded a few hours later when the patient had improved markedly. All those seven patients, and an additional one who did not have PE on admission had CHF,^{6–8} revealed augmentation of QRS complexes in the days after the resolution of PE, in the process of their management as they experienced receding of their peripheral edema, and loss of weight. Moreover, the response of the ECG with loss of weight in those patients was not unlike what was observed in patients with anasarca due to a variety of illnesses,⁶ or in patients with end stage renal failure following hemodialysis.^{14–18} Finally, the stability of the Σ QRS of the present patient during 7 days of observation, save for the initial drop, is in keeping with the absence of peripheral edema during physical examination, and the loss of only 2.1% of her admission weight during therapy.

Previous work in patients with CHF, which documented augmentation of QRS voltage,⁹ did not include details of the presence of peripheral edema or weight changes, or the presence and severity of PE, or the underlying cardiac pathology (dilated cardiomyopathy vs diastolic dysfunction/normal systolic function) to enable one to differentiate between the effect of the resolution of PE early in the clinical course, from the subsequent alleviation of peripheral edema, on the amplitude of QRS potentials. "Lumping" together these two consequences of left and/or right heart failure under the rubric of "CHF" certainly prevents understanding of the complex PE/peripheral edema/ECG voltage interactions. Thus patients presenting in CHF without PE, but with peripheral edema should be considered different than the ones who have both, and these two should be differentiated from the patients with just PE, as the patient presented herein, who constitute a subgroup with diastolic dysfunction, and hyperdynamic left ventricular function, described previously.¹⁹

More relevant to the present communication is the previous work with bronchopulmonary lavage,⁵ although this cannot be equated with clinical PE; thus (1) the lung in that study was filled with normal saline, while in PE the alveolar space fluid composition is a mixture of fluid and respiratory gasses; (2) in PE there is extravasated fluid present in the interstitial spaces (producing the radiological picture of interstitial edema), and blood-based vascular engorgement of pulmonary veins, venules,

capillaries, arterioles, and arteries; (3) finally, both lungs are involved in PE, while one lung is filled at a time in bronchopulmonary lavage. All the above are expected to produce a different "aggregate" impedance of the lungs in patients with PE,²⁰ as compared with the status of undergoing lung lavage. Also, experience with monitoring of patients in critical care areas, or postoperatively with impedance cardiography²¹ suggests that the status of the lung water content affects the thoracic electrical properties, and although in that work ECGs were not implemented, PE would be expected to produce changes in the ECG voltage. We have also made use of body electrical properties/amplitude of the QRS complexes relation in monitoring patients treated for CHF or undergoing hemodialysis.^{8,18} The difference in our implementation is only that it is implicitly based on changes in the total body impedance, while impedance cardiography²¹ monitors changes in the thoracic impedance. Thus the parallel is that increase in the ECG voltage with alleviation of CHF reflects reduction of water in the body volume conductor, while similar changes in the ECG with correction of PE implies reduction of the lung water content.

Based on the above, alleviation of the PE in our patient should have produced increase in the Σ QRS; not only this was not observed, but some initial decrease was shown, with subsequent stability of QRS potentials. Why was this so? First, it is possible after all that clinical PE does not decrease the amplitude of ECG potentials, and thus correction of this condition is not expected to produce increase in the QRS voltage. This is in keeping with our previous work, but is contradicted by the work with lung lavage and impedance cardiography data. The decrease of Σ QRS in the beginning of therapy of PE in our patient is even more difficult to explain. One way to reconcile these discrepancies could be that as suggested⁵ the effect of increase in conductivity of the lung tissue due to PE, which would be expected to produce attenuation of QRS potentials is counteracted by changes in the heart's dimensions, which in the setting of CHF would be expected to produce an augmentation of such potentials. Thus a cancellation of these two opposing forces would leave the QRS potentials unaltered. These two factors are expected to be present in patients with dilated cardiomyopathy, and probably have led to the stable QRS amplitudes noted by us previously.⁶⁻⁸ However, our present case was a patient with diastolic dysfunction and hypercon-

tractile left ventricle, theoretically not prone to dilation. In reality though even a heart not showing dilation at baseline could show such change during a bout of PE. Accordingly one wonders whether there was a phase of left ventricular dilation at the time that afterload was increased, and although we documented systolic blood pressure of 210 mmHg, it is conceivable that it was even higher at the time of her accident when she started experiencing severe dyspnea. This is supported by the appearance of the heart on the first chest radiograph (Fig. 2). Also, the initial drop of Σ QRS noted would be due to decreasing heart dimensions as the blood pressure decreased, as documented, while the PE persisted. In such a combination, an initial decrease of QRS potentials would be expected with subsequent stability, as the PE was corrected and the heart's size gradually returned to the baseline. This mechanism, therefore, is expected to produce unchanged QRS potentials in patients with CHF treated for PE, as we have previously documented. Confirmation of such a postulate would require treating patients with PE and no peripheral edema, like the case presented herein, with echocardiographically measured heart volumes prior and after therapy of PE. Until such work is implemented we can only be confident about the relationship of alleviation of peripheral edema and increase in QRS potentials in patients with CHF.⁷⁻⁹ The role of PE per se, and at the exclusion of concomitant changes in heart dimensions, on the ECG QRS voltage continues to be incompletely characterized.

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