


### Anasarca and Low Electrocardiogram Voltage

In a recent article in the Journal, Madias et al. (1) reported an inverse relationship between electrocardiogram (ECG) voltage and changes in body weight due to fluid loss or retention in patients with anasarca. The investigators could find no reference in the literature to a link between low ECG voltage and anasarca. A direct correlation has been reported, however, between ECG voltage and the concentration of serum albumin (2). Anasarca is frequently associated with a lowered serum albumin concentration (3). To ascertain how much the Madias et al. (1) study expands existing knowledge, it would be necessary to correlate the observed changes in ECG voltage with concomitant changes in serum albumin. Are the investigators able to do this?

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**REFERENCES**


**REPLY**

I am grateful to Dr. Brennan for directing me to literature describing a direct correlation of albumin and amplitude of QRS complexes (1). To respond to his inquiry I have modeled an analysis of our data after the study of Dr. Heaf (1). Our patients were critically ill, were followed for a long time (34.5 ± 36.7 days) (2) and inevitably experienced gradual reduction of albumin, despite our concerted efforts to use enteral and/or parenteral feeding. We had not measured albumin daily, but frequently. Thus, I was able to employ values that were the closest to the admission and peak weight albumin values of our patients. The albumin levels available for my analysis were checked on days 0.61 ± 1.71 for admission, and on days 0.43 ± 1.79 of peak weight time point.

Correlation of the sums of all 12 QRS leads (ΣQRS) (2) from admission with the corresponding albumin values revealed an r = 0.057 and a p = 0.77. A similar analysis, carried out for the ΣQRS obtained on the day of the peak weight and the corresponding albumin values, showed an r = 0.38 and a p = 0.04. Correlation of the percent change in the ΣQRS between admission and points of the peak body weights, with the percent change in the corresponding albumin values, revealed an r = 0.37 and a p = 0.053.

The difference in our results from the admission time point and the time of peak weight is hard to explain. If there was a real relationship between QRS amplitude and albumin, such a discrepancy should not have been encountered. Moreover, I find it odd to correlate absolute values of albumin with absolute values of amplitude of QRS complexes, taking into consideration that the latter can vary widely among both healthy subjects and patients, while the normal range of the former is 3.3 to 5.3 g/dl in our laboratory.

I find the correlation of change in each of the two correlates more meaningful scientifically, and in our data we found an almost significant association between the drop of albumin and loss of QRS amplitudes. The correlation coefficient was much smaller than the one we reported (r = 0.61) (2) for the association of weight gain and ΣQRS drop. However, some association may exist between albumin and QRS amplitudes, and may be confirmed in patients in whom both daily electrocardiograms and albumin measurements are implemented.

Does this mean that the hypoalbuminemia was causally related to the drop of ΣQRS in our patients, or was this biochemical abnormality “an innocent bystander”? Innocent it is not, for certainly it contributes to the extent of water retention; however, the causative mechanism for the drop in ΣQRS must have been the weight gain (or fluid retention). Correlation of % weight gain and % albumin drop in our 28 patients was poor (r = 0.02, p = 0.9; also, correlation of the % weight loss and % albumin rise in 9 patients was poor (r = 0.5, p = 0.1). It would have been very interesting to have data on weight change in the patients reported by Heaf (1).

There is evidence that edema fluid has low resistivity (3); thus, its effect on the transfer of cardiac potentials from the heart to the body surface can be explained by Ohm’s law. I am not aware of any work ascribing any mechanistic role for albumin per se in modulating the ECG voltage. Finally, the fact that patient 26 (2) gained 122.4 lb and lost 68.3% of his ΣQRS, while his albumin increased from 1.8 g/dl to 2.7 g/dl, and subsequently lost 77.4 lb and gained 185.9% of his ΣQRS (Fig. 6 of Madias et al. [2]) while his albumin increased merely from 2.9 to 3.0 g/dl, casts serious doubt on the contention that a causative relation between albumin and QRS amplitudes really exists.
Furthermore, Muro et al. (11) have shown that negative values of the augmentation index imply smaller or more diffuse reflections than other patterns. The results of the Nakayama et al. (1) study for PPH are thus inconsistent with the numerous reports demonstrating increased wave reflections in PPH (7–10).

In conclusion, the study of Nakayama et al. (1) is similar to previous studies demonstrating markedly increased wave reflection in CPTE (2–4) and suggesting that the timing and extent of wave reflection might be useful in the differential diagnosis of CPTE and PPH (2,3). The numerical values of the reflection indices calculated from fluid-filled catheters require careful scrutiny, whereas high-fidelity catheters must be preferred when attempting to obtain a reliable insight into pulmonary artery pathophysiology.

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REFERENCES