LETTERS TO THE EDITOR

Increase of Electrical Impedance Following Hemodialysis Is Not the Reason for QRS Augmentation

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Astan et al.¹ studied the effect of hemodialysis on the electrocardiographic (ECG) parameters. They found that the P wave and the QRS amplitudes and durations become significantly greater while T wave decreases in posthemodialysis (post-HD) patients. We confirmed the QRS and T wave changes in our study.² In addition, we introduced the so-called HD-induced "silent ischemia" manifested by increased ST-segment deviation.

We would like to debate the cause of the observed results. According to Astan et al.¹ and other authors, they are provoked by the post-HD increase of body fluids resistance. Still, we must mention the final statement of Astan et al.¹: "the cause of the QRS increase remains unclear."

We claim that the change of the body fluids resistance cannot reflect on the ECG waveforms since:

- The body resistance and its variation will cause none or minimal shift in amplitudes, since it is in order lower than the skin impedance and the electrodes resistance connected in series between the signal generator and the ECG amplifier.
- Post-HD body resistance change will shift all ECG waves in one direction, but T wave shifts in the opposite direction.^{1,2}
- Changes in ECG amplitudes during the systole are observed in hypertonic patients too,³ with no change in body resistance.

The authors claim as a whole that these ECG changes "are of extra-cardiac origin." Considering the

cardiac action potentials as a specific signal generator, we speculate that these are heart-intrinsic changes, a reaction to the heart-preload, and related to the interaction of blood pressure, ventricular contraction velocity, and excitation wave propagation.

Decrease of fluid leads to reduction of heart volume and relaxation of the heart-preload during the supraventricular (P wave) and ventricular (QRS-complex) systole. This is confirmed by the post-HD decrease of the systolic blood pressure.² The T wave represents the cardiac depolarization and is not affected by the heart-preload. Its decrease can be related to the change of sodium (Na) and potassium (K) concentration during HD.^{1,2}

In conclusion, we may say that the post-HD increase of electrical impedance is not the reason for QRS and P wave augmentation. We speculate that their amplitude shift is heart-intrinsically caused and inversely correlated to the change of the heart-preload.

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RESPONSE OF THE AUTHOR

Theories and Controversies on Mechanism of Electrocardiographic Changes during Hemodialysis: Reply to Christov et al.

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To the Editor,

We would like to thank the authors for their valuable comments. The procedure of hemodialysis (HD) exerts significant acute and chronic stress upon the cardiovascular system.^{1,2} Lung congestion and pericardial and pleural effusions can cause attenuation in the electrocardiogram potentials; therefore, the HD has been associated with an increase in the amplitudes of P wave and QRS complexes but not T waves.^{3,4} However, the mechanisms of these changes continue to be elusive, and whether they represent cardiac and/or extracardiac influences has not as yet been unraveled.^{5,6} Although Christov et al. speculated that post-HD increase in electrical impedance is not the reason for QRS and P-wave augmentation, Madias et al. reported that these changes are only apparent (not electrophysiologically real), representing an extracardiac phenomenon mediated by an increased electrical resistance of the passive body volume conductor, resulting in the measurement of augmented QRS complexes after fluid removal^{6–12} with subject-specific dynamic profiles during HD.¹³ This thesis is supported by theoretical work, animal experimentation, and clinical evidence.^{9–12} Unlike the other waves, decreased T-wave amplitudes after HD might be caused by decreased blood potassium levels after HD as T-wave amplitudes decrease with decreasing blood potassium.³ However, many studies have demonstrated silent ST-segment changes occurring during dialysis reporting the occurrence of dialysis-

induced ST depression at rates that vary between 15% and 40%.¹ Although Christov et al. speculated that these amplitude changes inversely correlated to the change of the heart-preload; an interesting study¹⁴ using volumetric hemodynamic monitoring evaluated the changes in extravascular lung water index (EVLWI) and intrathoracic blood volume index (ITBVI) that were measured immediately before and after HD in addition to hemodynamic- and oxygenation-related parameters; and found that although the EVLWI decreased significantly with fluid removal, ITBVI remained stable. Significant changes were also observed in stroke volume variation, cardiac index, and mean arterial pressure. EVLWI reduction with dialysis reflects the removal of excess body fluid, whereas preservation of cardiac preload is indicated by ITBVI stability.¹⁴ All these mechanisms in HD patients may explain the variable results in the literature, and further studies are required to better investigate the both short-term and long-term HD effects on human electrophysiology and to elucidate the pathophysiology involved and identify and select optimal therapies.

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