

Low QRS voltage and its causes

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Abstract

Electrocardiographic low QRS voltage (LQRSV) has many causes, which can be differentiated into those due to the heart's generated potentials (cardiac) and those due to influences of the passive body volume conductor (extracardiac). Peripheral edema of any conceivable etiology induces reversible LQRSV, reduces the amplitude of the P waves and T waves, decreases the duration of P waves, QRS complexes, and QT intervals, and alters in turn the measurements of the signal-averaged electrocardiogram and T wave alternans, all with enormous clinical implications.

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Low electrocardiographic QRS voltage (LQRSV) is traditionally defined by zenith-to-nadir QRS amplitudes of the QRS complexes of less than 0.5 mV in all the frontal leads and less than 1.0 mV in all the precordial leads.¹ However, the remarks that follow pertain both to electrocardiograms (ECGs) with LQRSV and to any attenuation of the QRS voltage based on the comparison of at least 2 ECGs, even if none of them satisfy the above-cited criteria for LQRSV. Occasionally, LQRSV in the ECG may not have an apparent explanation; and thus, in normal subjects, it is considered a normal variant. Low ECG QRS voltage may be noted only in the limb leads (a frequent encounter), the precordial leads, or both. Low ECG QRS voltage in limb leads with normal QRS precordial amplitudes,² or LQRSV in limb leads with high QRS complexes in the precordial leads with poor R-wave progression (“Goldberger triad”)³ have been described in patients with dilated cardiomyopathy. There are many causes of LQRSV, and they can be differentiated into those due to the deficient heart's generated potentials (cardiac causes) and those due to the attenuating influences of the pericardial space and pericardium, or the passive body volume conductor, enveloping the heart (extracardiac causes). Pericardial pathology, particularly pericardial effusion, can be categorized among the “cardiac” causes of LQRSV, considering that the visceral and parietal

pericardium is part of the heart, or the “extracardiac” causes because the site of generation of the cardiac potentials are inside the locus of pericardial fluid accumulation. Occasionally, both cardiac and extracardiac causes of LQRSV are simultaneously in operation in the same patient.

Cardiac causes of LQRSV

Multiple myocardial infarctions may lead to LQRSV because of cancellations and diminished electromotive force generation; LQRSV and QRS notches are seen in conjunction with severe post-myocardial infarction dysrhythmia.⁴ Infiltrative cardiomyopathies, a prototypical example being amyloidosis, may lead to LQRSV involving both the limb and the precordial leads,⁵ which occurs despite the marked cardiac hypertrophy or dilatation. Other infiltrative cardiomyopathies are reputed to be associated with LQRSV, but literature review does not provide relevant information. Myocarditis is associated with LQRSV attributed to the ailing myocytes,⁶ although extracardiac influences may also contribute to the LQRSV.⁷ Reduction of QRS voltage (not necessarily LQRSV) follows reduction of cardiac volumes due to various pathologies, hemorrhage, or hypovolemia (“Brody effect”).⁸ This is probably the mechanism for the LQRSV in patients with Addison's disease, although pulmonary congestion and/or peripheral edema (PERED) may contribute to LQRSV (vide infra).⁹ Extensive skin burns may lead to hypovolemia and cause LQRSV, although associated hypoalbuminemia may contribute (vide infra). An increased hematocrit leads to

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LQRSV because of the reduction in disparity of electrical resistivity of the intracardiac blood mass and the surrounding myocardium, which attenuates the radial depolarization forces, as per the Brody effect.^{8,10} Intracardiac injection of contrast media leads to some attenuated QRS voltage¹¹; however, the authors attributed this to 3-vessel coronary artery disease. The voluminous literature on the topic focuses on QTc and other repolarization changes.¹² This author suspects that the LQRSV may be due to increased resistivity of the contrast media,¹³ inducing the Brody effect; and it must be different for ionic, nonionic, hyperosmolar, isoosmolar, and hypoosmolar contrast media. Although LQRSV is rarely found in hypothyroidism in the absence of pericardial effusion, it has been suggested that hypothyroidism per se contributes to LQRSV.¹⁴

Pericardial causes of LQRSV

Pericardial effusion leads to LQRSV, the mechanism purported to be that of a short-circuiting of the heart's potentials as they are transmitted to the body surface; however, the mechanism may be more complex¹⁵ and may include even the intrapericardial pressure, like in tamponade, as the primary reason, along with the inflammation.^{16,17} The delays in recovery of LQRSV after pericardiocentesis or alleviation of tamponade suggest that the effects on the ECG in pericarditis/pericardial effusion/tamponade are multifactorial. This author believes that the occasional stability of the QRS amplitudes before and after pericardiocentesis of hemopericardium, in contrast to hydropericardium, is due to an interplay of changes in the position of the heart and the heart/chest wall distance, and the increased resistivity of the blood surrounding the heart (unpublished data). Electrical alternans, in the presence of a large pericardial effusion, often with impending tamponade, is attributed to a swinging motion of the heart, with a 2-beat¹⁸ or varying periodicity.¹⁹ Thickening of the pericardium in constrictive pericarditis is associated with LQRSV²⁰; however, pericardiectomy only partially restores the QRS amplitude, suggesting that the underlying myocardium is contributing to the LQRSV.²¹

Extracardiac causes of LQRSV

It has been long realized that the pathology of the organs and tissues surrounding the heart impacts the transfer of heart's potentials to the body surface with resultant LQRSV.¹ Analysis of theoretical models and of relevant animal and clinical work has elucidated the influences of variation in resistivity of various body tissues and geometrical considerations of the heart/thorax on the transformation of what is generated at the epicardial surface and what is recorded at the body surface.^{8,13,22,23} Accordingly, a host of clinical conditions is associated with LQRSV. Patients with chronic obstructive lung disease may show LQRSV, particularly in the limb leads,²⁴ because of an increased heart/chest wall distance from lung hyperinflation, which, if not offset, would be expected to augment QRS potentials by way of an increased electrical impedance. For the same reason,

pneumopericardium,²⁵ pneumomediastinum,²⁶ and pneumothorax, particularly left sided,²⁷ are associated with LQRSV. Pulmonary edema²⁸ and bronchopulmonary “lavage”²⁹ result in LQRSV because of decreased lung impedance by way of increased water content. Pneumonia, with extensive pulmonary infiltrates, and adult respiratory distress syndrome (“wet lung”) are expected to lead to similar ECG findings, although nothing to this effect has been described heretofore. Pleural effusion, particularly left sided and in the absence of congestive heart failure, causes LQRSV with an inverse relation between the extent of the effusion and the amplitude of QRS complexes.³⁰ Extensive subcutaneous emphysema, with retroperitoneum and mediastinal emphysema, is associated with LQRSV.³¹ Change in the position of the heart and its relationship with the chest wall, and certain ECG leads³² must be influential in LQRSV in some of the above-discussed conditions. Obesity is linked to reversible LQRSV.³³

In the past few years, it has been observed that PERED of any conceivable etiology is predictably linked to LQRSV.³⁴ Thus, PERED seen in association with sepsis,³⁴ certain drugs (eg, nonsteroidal anti-inflammatory drugs³⁵ and the antidiabetic thiazolidinediones [unpublished data]), cor pulmonale,³⁴ perioperative fluid load administration, even in the presence of normal left ventricular function,³⁶ chronic renal failure, particularly during the predialytic state,³⁷ congestive heart failure,³⁸ hepatic cirrhosis,³⁹ and numerous other conditions result in reversible LQRSV. In some of these conditions (eg, cirrhosis), hypoalbuminemia may exacerbate the PERED and LQRSV.^{40,41} Hypoalbuminemia and resultant PERED must be the mechanism of LQRSV noted invariably in children with kwashiorkor, rather than “myocardial atrophy,”⁴² although low cardiac volumes and decreased cardiac thickness and mass may contribute to the LQRSV.⁴³ Peripheral edema also decreases the amplitude of P waves⁴⁴ and T waves,⁴⁵ and the duration of P waves,⁴⁶ QRS complexes,⁴⁷ and QT intervals⁴⁸; such changes have enormous clinical implications for the diagnosis, management, and follow-up of patients with broad categories of cardiac and noncardiac diseases and, in addition, impact the signal-averaged electrocardiogram⁴⁹ and T-wave alternans,⁵⁰ with major consequences on the reproducibility and clinical relevance of such measurements.

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