REVIEW ARTICLE

R-Peak Time: An Electrocardiographic Parameter with Multiple Clinical Applications

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Abstract: In the 12-lead electrocardiogram (ECG), the time from the onset of the QRS complex (Q or R wave) to the apex or peak of R or to R' (when present), using indirect or semidirect surface unipolar precordial leads, bipolar limb leads or unipolar limb leads, is called ventricular activation time (VAT), R wave peak time (RWPT), R-peak time or intrinsicoid deflection (ID). The R-peak time in a specific ECG lead is the interval from the earliest onset of the QRS complex, preferably determined from multiple simultaneously recorded leads, to the peak (maximum) of the R wave or R' if present. Irrespective of the relative height of the R and R' waves, the R-peak time is measured to the second peak. The parameter corresponds to the time of the electrical activation occurring from the endocardium to the epicardium as reflected by the recording electrode located at a variable distance on the body surface, depending on the lead type: a unipolar precordial lead, a bipolar or unipolar limb lead. In normal conditions, the R-peak time for the thinner-walled right ventricle is measured from lead V₁ or V₂ and its upper limit of normal is 35 ms. The R-peak time for the left ventricle (LV) is measured from leads V₅ to V₆ and 45 ms is considered the upper limit of normal. In this manuscript, we review the clinical applications of this parameter.

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R-peak time; R wave peak time; ventricular activation time; intrinsicoid deflection

Initially, the term *intrinsicoid deflection* meant the time from the onset of the earliest Q or R wave to the peak of the R wave in the unipolar lateral precordial leads V₅–V₆, and was considered as the instant at which the area of cardiac muscle immediately below a unipolar epicardial electrode was completely depolarized. This term was coined by MacLeod, Wilson, and Barker in 1930.¹ to differentiate it from the term intrinsic deflection coined in 1914 by Sir Thomas Lewis experimentally in dog's hearts.² because the transition from a positive deflection registered from an electrode placed on the precordial body

surface (semidirect unipolar lead) is less abrupt than from an electrode placed experimentally directly on the epicardium. The validity of this concept was supported experimentally by Dower.³ He summarizes: "*The intrinsic deflection of Lewis has been variously defined and has been rejected by some as an indication of activity beneath an electrode on the surface of the heart. The use of intracellular electrodes, a surrounding wire loop electrode and fast recording speeds has, however, shown that the surface electrocardiogram does, in fact, present a negative deflection that is produced by immediately subjacent cells and which may, therefore, correctly be termed*

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intrinsic. It is more marked on the left than on the right due to the electric field generated by the heart as a whole. The importance of this finding relates to the possibility of the existence of appreciable intrinsicoid deflections in precordial leads."

This also makes the deflection less rapid and distinct. Because, by definition intrinsicoid deflection can only be measured from unipolar precordial leads,⁴ the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society (AHA/ACCF/HRS) recommended (Part III: Intraventricular Conduction Disturbances) to use the term R wave peak time instead.⁵ However, the term has also been applied to the limb leads. For practical purposes, the term "R-peak time" is used in both the limb (unipolar and bipolar) and precordial leads. The definition follows the recommendations given in the Minnesota Code.⁶

Conditions Where the Measurement of the R-Peak Time Is Useful

Volume overload, eccentric or diastolic left ventricular hypertrophy and hypertrophic cardiomyopathy

It is generally recognized that chronic left ventricle (LV) volume overload is characterized by chamber enlargement with an eccentric pattern of hypertrophy. In these patients, the LV end-diastolic pressure is consistently higher than normal. Increased resting (diastolic) wall tension results in gradual myocardial fiber elongation, which improves the efficiency of the ventricular chamber; however, this comes at the price of diastolic wall stress. In eccentric LV hypertrophy (LVH) with diastolic overload, there is an increased LV mass, while the measured wall thickness is normal. In clinical context, eccentric LVH is seen in mitral and aortic regurgitation, ventricular septal defect, patent ductus arteriosus and hypertrophic cardiomyopathy.^{7,8} The time interval from the onset of QRS to the peak of R ≥ 50 ms in V_5 and V_6 is a criterion for LVH due to diastolic overload. Prolongation of the R-peak time is frequently associated with diastolic dysfunction in patients with newly diagnosed untreated hypertension. There was a significant correlation between R-peak time and early diastolic velocity measured by tissue Doppler imaging, ratio of early and late diastolic velocities, transmitral Doppler (TMD) E/A ratio (early peak filling rate, and early deceleration

peak), and ratio of early diastolic mitral inflow and early diastolic tissue Doppler velocities (E/e').⁹

ECG manifestations of systolic and diastolic overload of the left ventricle

In electro-vectorcardiography, we have for quite a long time been using the term diastolic LVH, when the blood volume entering the LV in diastole is increased (diastolic, eccentric or volumetric overload).^{10,11} The term pressure, systolic or strain pattern overload refers to situations with deficiencies during ventricular systole (with normal or abnormal contraction). Diastolic and systolic overload may coexist. Table 1 and Figure 1 depict the most important differences between the two main hemodynamic modalities of LVH.

The ECG correlation for concentric as compared with eccentric LVH is a shorter R-peak time, significant ST-segment depression and inverted T waves in the antero-lateral leads. In patients with advanced acquired heart disease, severe LV dilatation and hypertrophy, the correlation between hemodynamic and ECG parameters is poor, while it is better in congenital heart diseases.¹²

As indicated above, the normal value of R-peak time is <50 ms in leads V_5 or V_6 . This parameter is characteristically prolonged in diastolic, volumetric, or eccentric LVH. R-peak time is shorter in systolic or concentric than in diastolic LVH. "Clean" (nonfragmented) deep (≥ 2 mm) and narrow (<40 ms) Q waves are registered in the left lateral leads I, aVL, V_5 - V_6 and eventually in the inferior leads; consequently the initial 10-20 ms first middle septal vector (I_{AM} or antero-medial vector) is increased.¹³

QRS duration is frequently increased in LVH (between 110 and 120 ms). QRS duration greater than 110 ms in subjects older than 16 years of age is considered abnormal. This is manifested by a general increase in QRS duration or, more specifically, an increase of R-peak time in the lateral precordial leads V_5 and/or V_6 (≥ 50 ms). The increased QRS duration may be attributed to the increased thickness of the LV wall and to intramural fibrosis, which distorts and prolongs the transmural impulse propagation from endo- to epicardium. The hypertrophied ventricle is thicker than normal; the distance the activation wave must travel to reach the epicardium is increased. Thus, onset of the R-peak time or the appearance of the

Table 1. Differences between Systolic and Diastolic Overload

	Systolic Overload	Diastolic Overload
Main causes	Systemic hypertension Aortic stenosis Coarctation of the aorta	Aortic valve insufficiency Mitral valve insufficiency Ventricular septal defect Patent ductus arteriosus
Initial R wave in V ₁ lead	Absent or minimal	Greater R wave
Initial Q waves in left leads	Absent	Present
R peak time in V ₅ –V ₆	Shorter	Longer (≥50 ms)

Figure 1. ECG/VCG patterns in two main modalities of left ventricular hypertrophy in the horizontal plane (only leads V₁ and V₆). (A) Systolic or concentric LVH: Absent or minimal initial r wave in lead V₁, absence of Q waves in the left leads, shorter R-peak time in left lateral leads V₅–V₆. (B) Diastolic, volumetric or eccentric LVH: Greater R wave in lead V₁, presence of initial “clean” deep Q waves in the left leads, concordant QRS-ST/T relationship and prolonged R-peak time in leads V₅–V₆. The R-peak time is prolonged in the left leads in case of diastolic LVH. “Clean” deep (≥2 mm) and narrow (<40 ms) Q waves are recorded because the first middle septal initial 10 to 20 ms vector or I_{AM} vector (anteroseptal) is increased and directed away from the left leads.

maximal vector in a lead facing the hypertrophied ventricle is delayed. The greater muscle mass of the hypertrophied ventricle also causes an overall lengthening of the time required for ventricular activation, as evidenced by an increased duration of the QRS interval. In LVH diastolic, eccentric or volumetric overload, the augmentation of the first vector responsible for the initial increase in R wave amplitude in the right precordial leads and a concomitant initial deep Q wave in the left lateral precordial leads are responsible for the R-peak time increment. When the LVH pattern with widened QRS is present, there may be loss of the septal Q wave, sometimes with slurred R wave upstroke diagnosed as incomplete left bundle branch block (LBBB) always associated with LVH.

In hypertrophic cardiomyopathy the most frequent ECG abnormalities are a prolonged R-peak time in the leads corresponding to the affected region and (more or less) deep and clean Q waves in leads aVF, aVL, V₅, and V₆.¹⁴

Right ventricular hypertrophy

In right ventricular hypertrophy (RVH) a prolonged R-peak time ≥40 ms is frequently observed in leads V₁–V₂ (normal 15–35 ms). R-peak time in lead V₆ is not prolonged. In mitral stenosis, as an expression of the systemic hemodynamic alteration of the left ventricle (LV), the decrease in time of inscription of the R-peak time in lead V₆ becomes evident when the mitral area is decreased or when

Figure 2. Case presentation.

Clinical diagnosis: Female patient, 35 years old with severe mitral stenosis. ECG description: Left atrial enlargement. Triphasic QRS complexes (rsR') in leads V₁ and V₂ and negative T waves from leads V₁ to V₅: RVH. R-peak time is prolonged in the right precordial leads and lesser in lead V₆.

the mean pressure of the pulmonary artery starts to rise.^{15–18} (Fig. 2).

The R-peak time was delayed in the right precordial leads (V₁/V₂) in all age groups of asthmatic children; however, no significant difference was demonstrated in the left precordial leads (V₅/V₆).¹⁹

Right bundle branch block

In right bundle branch block (RBBB) the R-peak time is prolonged in leads V₁–V₂ (≥ 50 ms) and normal in the left precordial leads V₅ and V₆ (Fig. 3). The time difference existing between both R-peak times (left and right) is consequence that the last electrical activity is directed to the right, or toward lead V₁. In the right-hand side, the stimulus must activate sequentially the left side of the interventricular septum; the LV free wall from endo to epicardium and the slow trans-septal conduction until it reaches the right ventricle epicardium. On the other hand, in the left side (V₅/V₆).

Left septal fascicular block

In left septal fascicular block (LSFB), the R-peak time is prolonged in the right precordial leads V₁–V₃.²⁰ (Fig. 4). The supraventricular

stimulus reaches the LV endocardium by two non-blocked fascicles (left anterior fascicle and left posterior fascicle) originating two vectors of opposite directions that tend to functionally cancel each other. The vector dependent on the left septal fascicle (1_{AM} antero-medial vector) normally heading to the front and the right, does not manifest by block, which is translated by an absence of initial q wave in the left leads and minimum delayed in the R-peak time in leads V₁–V₃ (>35 ms).

Complete left bundle branch block

In complete LBBB the R-peak time is greater than 60 ms in leads V₅ and V₆ but normal in leads V₁, V₂, and V₃; small initial r waves can be discerned in these leads. In LBBB, an important delay in the R-peak time is observed in the left precordial leads I, aVL, V₅, and V₆.²¹ The end of the R-peak time in the left leads I and V₆ coincides with the second notched at the peak of R wave or with the end of the slurred plateau-like peak of the R wave in these leads. Because of the delayed onset of left ventricular activation and the delayed appearance of the maximal instantaneous vector, the onset of the R-peak time in lead V₆ is likewise delayed to

Figure 3. Complete RBBB.

Note prolonged R-peak time in lead V₁ and normal R-peak time in lead V₆.

Figure 4. Case presentation.

Clinical diagnosis: Caucasian, 67-year-old man, with controlled hypertension and coronary artery disease. Coronary angiography: Proximal critical obstruction of the LAD before the first septal branch.

ECG description: Sinus rhythm, HR = 75 bpm, QRS axis + 110°, rS in leads I and aVL, qR, qR pattern in leads III, aVF and II, RIII > RII, prominent QRS anterior forces with significant increased R-peak time in leads V₁ and V₂ (80 ms), small (embryonic) initial q wave in leads V₂–V₃, R wave voltage in lead V₁ ≥ than 5 mm, R wave in leads V₂ and V₃ > 15 mm, sharp-pointed R wave in leads V₂–V₃ leads, R/S ratio in lead V₂ > 2, prolonged R-peak time in lead V₂, R wave amplitude "in crescendo" from lead V₁ through V₃ and decreasing from V₅ to V₆, absence of q wave in the left precordial leads V₅–V₆ by absence of the first middle septal vector or vector 1_{AM} (anteromedial).

The ECG shows a typical example of LSFB with prolonged R wave peak time in the right precordial leads.

LAD = left artery descendant; LSFB = left septal fascicular block.

80–100 ms after the beginning of the QRS interval. The activation spreads from fiber to fiber through the left ventricular myocardium along a broad activation front. The produced electrical forces are therefore of increased magnitude. The trabeculated nature of the endocardium results in the varying directions of the initial activation wave front. The activation wave spreads concentrically from each of these trabeculations, resulting in activation of

the inner muscle layers normally occurring in too many directions to give rise to measurable electric forces. The posterobasal wall of the LV is activated before the anterolateral wall. Thus, the thickest portion of the left ventricle wall is the first undergoing depolarization, with the result that the forces generated at this time are larger and directed more posteriorly and superiorly than those that follow (Fig. 5).

Figure 5. Complete LBBB.

Note significant prolongation of the R-peak time in lead V_6 and lesser R-peak time in the right precordial leads V_1 – V_2 .

Incomplete left bundle branch block

In incomplete LBBB the R-peak time is ≥ 60 ms in leads V_4 , V_5 , and V_6 .²² There is slurring or notching of the QRS complexes such as seen in complete left bundle branch block. The principal point of distinction (arbitrary though it might be) that has been made between complete and incomplete LBBB is that the QRS duration is ≥ 120 ms in complete LBBB and less than 120 ms in incomplete LBBB. A QRS duration between 110 and 119 ms in adults, between 90 and 100 ms in children 8–16 years of age, and between 80 and 90 ms in children less than 8 years of age. There is always LVH and absence (or diminutive) initial q wave in leads I, V_5 , and V_6 (Fig. 6).

Left anterior fascicular block

In left anterior fascicular block (LAFB), the R-peak time in lead aVL is prolonged (≥ 45 ms), while is normal in V_6 .²³ (R-peak time in $V_6 < \text{aVL}$). Supraventricular impulses are conducted to the LV via the left posterior fascicle, which inserts into the infero-septal wall of the LV along its endocardial surface (postero-medial mitral papillary muscle). On reaching the LV, the initial 10 to 20 ms electrical vector is therefore directed downwards

and rightwards (as excitation spreads outwards from the endo- to the epicardium), producing small initial r waves in the inferior leads (II, III, aVF) and small initial q waves in I and aVL. The major wave of depolarization then spreads upwards and leftwards, producing large positive voltages (tall R waves) in the left-sided leads and large negative voltages (deep S waves) in the inferior leads with $\text{SIII} > \text{SII}$. This process takes about 20 ms longer than simultaneous conduction via both fascicles, resulting in a slight widening of the QRS. The impulse reaches the left-sided leads later than normal, resulting in an increased R-peak time in aVL. Horwitz et al.²⁴ evaluated two criteria for the diagnosis of LAFB: extreme left axis deviation (LAD) and the delay in the time of inscription of the R-peak time in lead aVL asynchronous to V_6 . From 400 ECGs with left axis deviation (LAD) of $\geq -30^\circ$, 62% showed asynchronous activation of the LV. There was only a general relationship between the degree of LAD and delayed R-peak time in aVL. The incidence of delayed R-peak time in aVL was as follows: 2% with a mean frontal QRS axis at 0° ; 9% at -15° ; 41% -30° ; 69% at -45° ; 82% at -60° ; and 100% at -75° or greater. The lack of correlation between both criteria in many instances questions their validity. The LAD alone should not be considered a synonymous of LAFB. Recognition of delayed inscription of the R-peak time in aVL is a useful supplemental criterion for the diagnosis of LAFB²⁵ (Fig. 7).

Alboni and Malacarne²⁶ studied 12 patients with complete LBBB with extreme left axis deviation (LAD) (beyond -45°). The LAD may be connected to an asynchrony of LV activation, due to delayed excitation of the antero-superior LV wall fascicle. This asynchrony should be evident in the ECG by a more delayed R-peak time in aVL (high lateral lead) than in V_6 (low lateral lead), as occurs in isolated LAFB.

Left posterior fascicular block

The diagnosis of LPFB based on ECG and vectorcardiography (VCG) tracings alone, is possible when the ECG and VCG indicate asynchronous left ventricular activation: R-peak time in leads aVF or $V_6 > \text{aVL}$ by early initial activation of the high lateral leads (aVL and I) related to the low leads such as aVF and V_6 .²⁷ Since the left posterior fascicle of the left bundle branch when interrupted the activation of the LV proceeds superiorly and to

Figure 6. Incomplete LBBB. R-peak time is prolonged at 60 ms in leads V₄, V₅, and V₆ with a QRS duration of 110 ms.

Figure 7. Left anterior fascicular block (LAFB).

ECG diagnosis: S⁺QRS near -60°; qR pattern without s wave in I and no r' in II. S wave of III <15 mm aVR near isodiphasic complex in aVR. Additionally, R-peak time in V₆ < aVL. Clear difference of duration of R-peak time in low lateral free wall (V₆) related to high lateral LV wall (aVL), because the lower parts of the lateral wall of the LV are activated earlier than the high ones (R-peak time V₆ < aVL). Conclusion: LAFB.

the left. This is due to the fact that the antero-superior portion of the LV is initially activated. Therefore, a small 20 ms q wave in leads aVF-V₆ is inscribed before the high lateral lead (aVL) (Fig. 8). This explains that R-peak time aVF or V₆ > aVL.

Differential diagnosis of wide QRS complex tachycardia

Wide QRS complex tachycardia still presents a diagnostic challenge when the clinician is confronted with a 12-lead ECG. The differential

diagnosis includes ventricular tachycardia (VT) vs. supraventricular tachycardia with functional aberrancy (SVT-A), preexisting bundle branch block, preexcitation, electrolyte disturbance or drug effects, such as tricyclic antidepressant (TCA) abuse or class IA or IC antiarrhythmic intoxication (sodium-channel blocker toxicity). Despite all available criteria, wide complex tachycardias are still misdiagnosed or may remain undiagnosed. In patients with wide QRS complex tachycardia a value of R-peak time in lead II ≥50 ms is diagnostic of VT (Fig. 9).

Figure 8. Left posterior fascicular block (LPFB).

Classical ECG/VCG pattern in the frontal plane of LPFB associated with complete RBBB. Note that R-peak time in aVL < aVF because of the earlier activation of the high lateral wall of the LV related to the low portion of this ventricle. Additionally, the final portion of QRS loop shows right end conduction delay (dashes closer to each other) consequence of the RBBB in association.

Table 2. Vereckeai Algorithm versus RWPT Algorithm³⁰

	New Vereckeai Algorithm	RWPT Criterion
Accuracy	84.3%	79.6%
Sensitivity	92.4%	79.1%
Negative predictive value (NPV)	77.8%	95%
Positive predictive value (PPV)	86.4%	95%
Specificity	64.7%	80.9%

Figure 9. R-peak time in wide complex tachycardias. The figure depicts the R wave peak time (RWPT) (as called in Pava's manuscript) in lead II ≥ 50 ms in both positive and negative QRS complexes. VT is diagnosed when the RWPT is ≥ 50 ms.

Pava et al.²⁸ proposed an ultra-simple and highly sensitive criterion called *lead II R wave peak time*, that discriminates VT from SVT-A. The original

study of 218 patients showed good performance of this criterion, which waits prospective testing in a larger study. With this in mind, Szelényi et al.²⁹ compared the "Vereckeai algorithm"³⁰ with the "lead II R wave peak time algorithm" (Table 2). The authors studied 483 wide complex tachycardias (142 VTs, 62 SVT-A, and eight preexcited SVTs) from 145 patients with proven electrophysiological diagnoses analyzed by seven examiners blinded to the electrophysiologic diagnoses. Incorrect diagnoses made by the Vereckeai algorithm were mainly due to misdiagnosis of SVT-A as VT (65.7% of cases), while those based on the R wave peak time criterion were due to (more dangerous) misdiagnosis of VT as SVT (72.5% of cases). The

Vereckei algorithm was superior in overall test accuracy, sensitivity, and NPV for VT diagnosis and inferior in specificity and PPV to the R wave peak time criterion. For each of the particular ECG features, the results of the statistical analysis in "real life" were inferior to those reported in the original publications. The authors concluded that the aVR lead four steps algorithm (Vereckei) is associated with excellent accuracy rate and sensitivity for the differential diagnosis of wide complex tachycardias. This algorithm is simple and could be easily learned and applied by physicians.³¹

Cardiac resynchronization therapy (CRT)

Up to one-third of the patients who undergo cardiac resynchronization therapy (CRT) may be nonresponders. Del-Carpio et al.³² measured the R-peak time in leads I, aVL, V₁, V₂, V₅, and V₆ in 135 patients who underwent CRT implantation. A CRT response was defined as a decrease in LV end-systolic volume (LVESV) >15% at 6 months. In patients with LBBB or nonspecific intraventricular conduction delay, response was predicted by longer R-peak time in leads I, aVL, and in lead I minus lead V₁ adjusting for baseline QRS duration and LVESV. Results were similar after adjusting for postimplant or change in QRS duration. The R-peak time parameters were better predictors of response than QRS duration parameters. R-peak time in lead I/QRS duration ratio (OR 3.1; 95% CI 1.6–5.9) also increased the odds of response after adjusting for baseline LVESV. Cutoff values for R-peak time for the different leads were: I 110 ms; aVL 130 ms; I minus V₁ 90 ms. R-peak time in lead I/QRS duration ratio of 0.69 yielded a sensitivity and a specificity as high as 83% and 81%. The authors concluded that the measurement of R-peak time from the ECG permits a preimplant and noninvasive way of determining the LV activation delay. It was found a good predictor of CRT response, and represents a promising alternative to QRS duration parameters.

CONCLUSIONS

R-peak time is an easy to use ECG parameter that helps in a large variety of conditions. It should be used as a supplement of other ECG criteria to facilitate diagnosis and in some circumstances, to determine prognosis.

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