Electrocardiographic recognition of right ventricular hypertrophy

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Abstract

The electrocardiogram (ECG) is a relatively insensitive tool for the detection of right ventricular hypertrophy (RVH), but some criteria have high specificity. The recommended ECG screening criteria for RVH are not sufficiently sensitive or specific for screening for mild RVH in adults without clinical cardiovascular disease. The greatest accuracy of the ECG is in congenital heart disease, with intermediate accuracy in acquired heart disease and primary pulmonary hypertension in adults.

Keywords: ECG; Right ventricular hypertrophy; ECG screening

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Introduction

The electrocardiogram (ECG) is a relatively insensitive tool for the detection of right ventricular hypertrophy (RVH), but some criteria have high specificity [1]. The right ventricle (RV) is located in front and to the right of the left ventricle (LV). Consequently, its forces are directed rightwards and anteriorly. In adolescents and adults, the RV forces are almost completely masked by the dominant forces of the LV (the ECG is a “levocardiogram”). Additionally, RVH causes ≥35 ms delay in the R-wave peak time in the right precordial leads [2]. RVH is more likely to be detected by the ECG in moderately/severely increased RV pressure. RVH is much rarer than left ventricular hypertrophy (LVH), and in its extensive form it is encountered in several congenital heart diseases. The recommended ECG screening criteria for RVH are not sufficiently sensitive or specific for screening for mild RVH in adults without clinical cardiovascular disease [3].

The greatest accuracy of the ECG is in congenital heart disease, with intermediate accuracy in acquired heart disease and primary pulmonary hypertension in adults [4]. The ECG pattern resulting from RVH is highly variable and dependent on intrinsic and extrinsic factors such as the hemodynamic pattern of overload (systolic or diastolic), severity, coexistent LVH, coexistence of bundle branch blocks, fascicular blocks, myocardial scars, rotation of the heart, and consequences from increased lung volume with a downshift of the diaphragm.

In the following different clinical entities are briefly presented with ECG and vectorcardiographic (VCG) correlations. It has to be pointed out that most
criteria were derived from autopsy studies in selected patient populations.

**Clinical causes of RVH**

A) Congenital heart disease
   I. Acyanotic congenital heart diseases
      Secundum atrial septal defect (ASD), common atrioventricular canal, pulmonary stenosis, ventricular septal defects with pulmonary stenosis, patent ductus arteriosus, aortic coarctation in the first 6 months of life, primary pulmonary hypertension.
   II. Cyanotic congenital heart diseases
      Fallot’s tetralogy, double-outlet right ventricle, transposition of the great arteries, single ventricle, aortic atresia, congenital mitral stenosis/atriea, truncus arteriosus, Eisenmenger syndrome.

B) Acquired causes

Athlete’s heart, mitral stenosis [5], tricuspid insufficiency, chronic obstructive pulmonary disease, acute pulmonary embolism, chronic thromboembolic pulmonary hypertension and miscellaneous.

**The three main hemodynamic modalities, severity of RVH and ECG correlates**

1) A.) Pressure (systolic) overload: type A RVH
   Two subtypes (Fig. 1):
   I) Adaptation overload
      Right intraventricular pressure never higher than left intraventricular pressure; tall R waves in V₁ [6] with Rs/R, and V₂, showing negative QRS predominance. A typical example is Fallot’s tetralogy.
   II) Severe systolic overload with strain pattern of repolarization in the right precordial leads
      Right intraventricular pressure may exceed the systemic one (supra-systemic): tall R waves or qR complexes in V₁, predominantly positive QRS complexes in V₂-V₃. A typical example is severe pulmonary stenosis.

VCG

I) RVH type A with adaptation overload. QRS loop with initial 10–20 ms preserved: directed to the front, and counter-clockwise rotation predominantly located in the anterior quadrants.
II) RVH type A with systolic overload and the strain pattern. QRS loop with initial 10–20 ms directed to the back and leftward, and CW rotation predominantly located in the anterior quadrants.

1) B.) Pressure (systolic) overload: type B RVH (Fig. 2)

2. Chronic obstructive pulmonary disease; type C RVH

This condition often causes a characteristic ECG pattern that reflects the low diaphragm resulting from the increased lung volume [7]. This pattern includes low QRS voltage in the limb leads, rightward shift of the QRS axis (superior or indeterminate), a rightward P-wave axis beyond +60°; a persistent rS pattern across all

![Fig. 1. ECG: Note prominent anterior QRS forces (PAF) aiding in the ECG diagnosis of RVH.](image-url)
precordial leads, also called anterior pseudo-infarction pattern (Fig. 3A).

3. Diastolic, volumetric or eccentric RVH [8]

The ECG shows typical incomplete right bundle branch block (RBBB), suggesting volume overload of the RV. The most representative example is atrial septal defect, which causes eccentric dilatation of the RV with selective predominance of hypertrophy in the right ventricular outflow tract (RVOT) (Fig. 3B).

Criteria for RVH

I) Precordial leads

- $RV_1 \geq 7$ mm
- $SV_1 < 2$ mm
- Ventricular activation time $\geq 35$ ms in $V_1$
- qR pattern in $V_1$
- Positive T wave in $V_1$ after 3 days of life and up to 6 years of age, if R/S ratio $> 1$
- Negative “primary” (symmetrical) T waves in $V_1$-$V_3$
- Ratio between precordial leads
- $RV_1 + SV_1 \geq 10.5$ mm (Sokolow-Lyon index).
- R/S ratio in $V_5 - V_6 \leq 1$
- $RV_1 > RV_6$
- Regression of R/S ratio across the precordium.

II) Limb leads

- Right axis deviation $>+110^\circ$ in adults.
- Sl-I-I-II-III pattern
- RaVR $> 4$ mm
- Q/R ratio of aVR $\leq 1$
- McGinn-White pattern: SI-QIII-TIII

III) Association of precordial with unipolar leads

- Deep S wave in V1 or V1-V2 associated with QRS complexes of positive predominance in aVR

Fig. 2. These changes are present in moderate RVH and at an earlier disease stage. The QRS loop in the horizontal plane shows counterclockwise rotation. The QRS loop is displaced anteriorly and to the left: $\geq 70\%$ of the area of the QRS loop of anterior location (in front of the X line = anterior quadrants), which generates prominent anterior forces. $V_{3b}$ and $V_1$ show R/s ratio $> 1$. R wave in $V_1 \geq 7$ mm; $V_5$ and $V_6$: qRS; ST/T vector deviated to the left and backward.
IV) Other

- RBBB associated with right atrial enlargement and right axis deviation
- RBBB of sudden onset, associated with sinus tachycardia, atrial fibrillation/flutter or left anterior fascicular block

Conclusion

The ECG has low sensitivity for the detection of RVH in the community. In a clinically ill population, the established ECG criteria may have clinical or prognostic utility. The highest sensitivity is observed in congenital heart diseases.

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References


