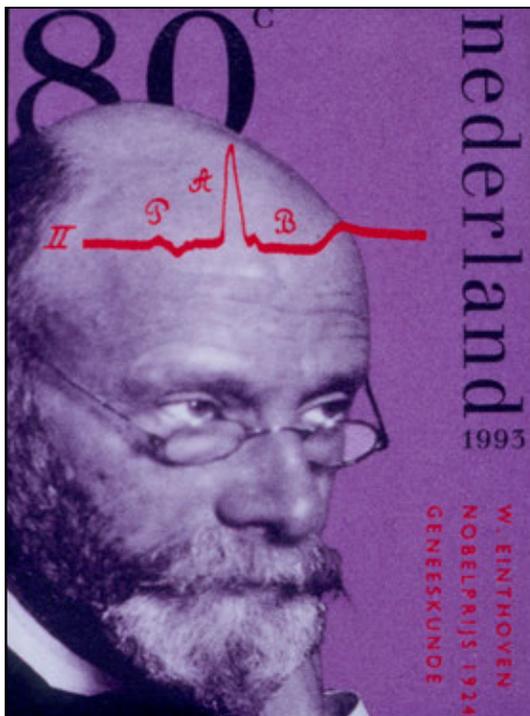


## **The Electrocardiogram.**

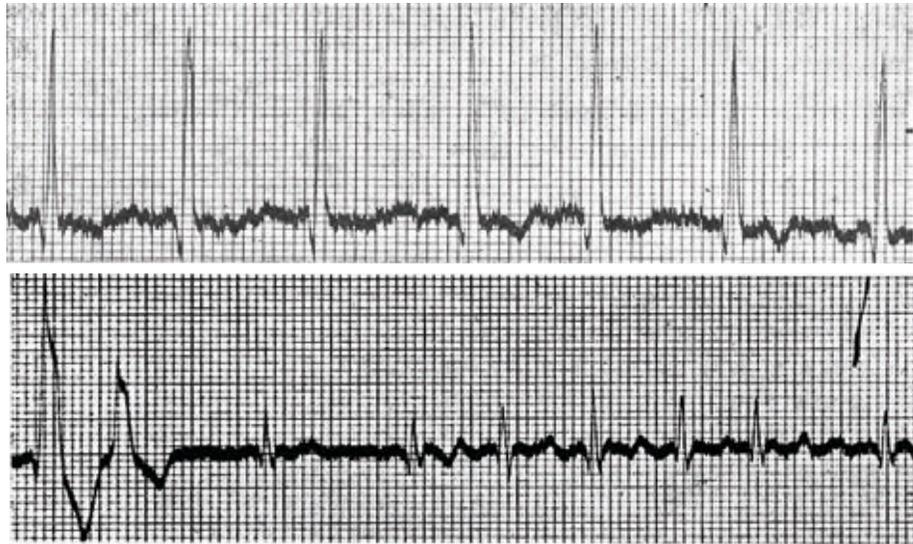
**In jeopardy more than a century  
after its introduction by Willem  
Einthoven?**

**Time for a revival.**

**by  
Hein J. Wellens MD**



**Willem Einthoven  
1860-1927**



Einthoven, 1905

## The ECG

- Everywhere available
- Easy and rapid to make
- Non-invasive
- Reproducible
- Inexpensive
- Patient-friendly

## Not so evident at the beginning...

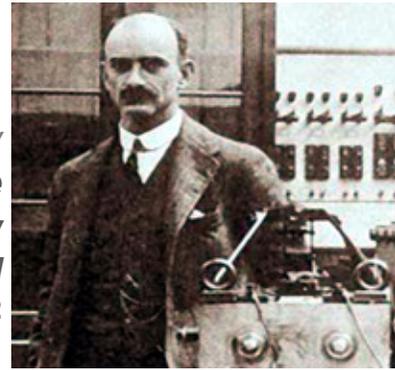


*I do not imagine that electrocardiography is likely to find any very extensive use in the hospital. It can at most **be of rare and occasional use to afford a record of some rare anomaly of cardiac action***

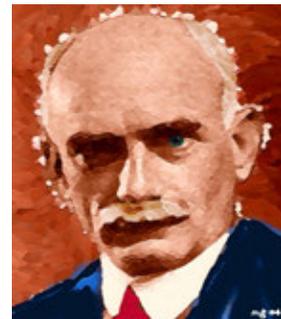
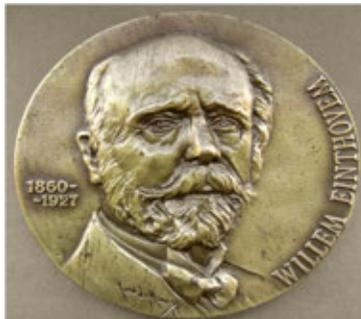
Augustus D. Waller 1911

*The time is at hand, if not already come, when **an examination of the heart is incomplete if this new method is neglected***

Thomas Lewis 1912



## Lewis developed clinical electrocardiology



**Einthoven**, Nobel Lecture, December 11, 1925

**Thomas Lewis**  
(1881-1945)

*the English investigator **Thomas Lewis**, ... has played a great part in the development of electrocardiography..., and I doubt whether without his valuable contributions I should have the privilege of standing before you today*

- Every day approximately 3.000.000 ECGs are made worldwide!
- The ECG is not only the tool most used but also the most valuable one giving instantaneous information about the heart.

By re-analyzing the ECG in the light of findings from invasive and non-invasive studies (coronary angiography, programmed electrical stimulation of the heart, intracardiac mapping, echo, MRI, nuclear, genetic information) and by using computer-assisted measurements, correlations and predictions the diagnostic value of the ECG continues to grow.

## The ECG

**Instantaneous information on diagnosis, management and effect of treatment:**

- Cardiac ischemia
- Rhythm- and conduction disturbances
- Structural changes in the cardiac chambers
- ECG changes caused by medication
- Evaluation and programming implantable devices
- Electrolyte and metabolic disorders
- Monogenic arrhythmology

**Risk estimation**

## The value of the ECG in cardiac ischemia

- Presence and staging of the ischemic process
- Localization of the site and size of the area at risk by determining the location of the culprit lesion in the coronary artery
- Diagnosing reperfusion
- Localizing extent of muscle loss
- Predictor of future cardiac events

## The ECG in Rhythm and Conduction disturbances

- Localizing the site of block
- Determining the type of tachycardia
- Localizing the site of origin or circuit of the tachycardia
- Determining the mechanism of a tachycardia
- Selecting optimal management

## Risk of sudden cardiac death

	% SCD	Predictability
No hx of cardiac disease	45	Poor
Hx cardiac event: LVEF > 40%	40	Limited
Hx cardiac event: LVEF ≤ 40%	13	Possible
Monogenic arrhythm disease	2	Limited

### The ECG for risk estimation sudden cardiac death

- Heart rate
  - rest
  - variability
  - turbulence
- Deceleration capacity after a VPB
- QRS
  - width
  - notching
  - signal averaging
- Left ventricular hypertrophy
- QT interval
  - length
  - dispersion
  - dynamic behavior
- T wave alternans
- Angle QRS complex – T wave
- Exercise ECG
- Monogenic electrocardiography

### ECG derived risk stratifiers reported to have prognostic value for overall cardiac and sudden death mortality in different clinical settings (1)

	GP	pLVEF	rLVEF	12 lead ECG	Holter ECG
<b>Sinus Rhythm</b>					
- Resting rate and profile during exercise	+	+	?	+	+
- Rate variability	+/-	+	+	-	+
- Heart rate turbulence	?	++	+	-	+
- Deceleration capacity	?	+	+	-	+
<b>Atrial dilatation</b>	?	?	?	+	-
<b>Atrial fibrillation</b>	+	+	+	+	+
<b>AV conduction</b>					
- Site of AV block	+	+	+	+	+
- Presence of accessory pathways	+	?	?	+	+

GP = general population; pLVEF=cardiac disease, preserved LVEF; rLVEF= cardiac disease, reduced LVEF

**ECG derived risk stratifiers reported to have prognostic value for overall cardiac and sudden death mortality in different clinical settings (2)**

	GP	pLVEF	rLVEF	12 lead ECG	Holter
<b>QRS</b>					
- Width > 100 ms	?	+	++	+	+
- Left bundle branch block	+	+	++	+	-
- Notching, fractionation	?	+	+	+	-
- Number & location of Q waves	?	+	+	+	-
- Reduced voltage (limb leads)	?	+	+	+	-
- SAEKG	?	+	+	+	+
- Left ventricular hypertrophy	+	+	+	+	-
- Mean QRS-T angle	+	+	+	+	-

GP = general population; pLVEF=cardiac disease, preserved LVEF; rLVEF= cardiac disease, reduced LVEF; SAEKG= signal averaged ECG

**ECG derived risk stratifiers reported to have prognostic value for overall cardiac and sudden death mortality in different clinical settings(3)**

	GP	pLVEF	rLVEF	12 lead ECG	Holter
<b>QT interval</b>					
- Duration	+	+	+	+	+
- Dispersion	?	+	+	+	-
- Dynamic	?	+	+	-	+
<b>ST-segment</b>					
- Elevation/depression	+/-	?	?	+	-
- J-point elevation (inferior leads)	+	?	?	+	-

GP = general population; pLVEF=cardiac disease, preserved LVEF; rLVEF= cardiac disease, reduced LVEF;

ECG derived risk stratifiers reported to have prognostic value for overall cardiac and sudden death mortality in different clinical settings(4)

	GP	pLVEF	rLVEF	12 lead ECG	Holter
<b>T wave</b>					
- Axis	+	+	+	+	-
- Negativity	+	+	+	+	+
- T-wave alternans	?	+	+	-	+
- Tpeak - Tend interval	?	+	+	+	+
- T amplitude V <sub>1</sub> and aVR	+	?	?	+	-
<b>Ventricular ectopy</b>					
- Width and site of origin	+	+	+	+	+/-
- VPB coupling interval	+/-	+	+	+	+
- Frequent VPBs	+/-	+	+	+	+
- Non-sustained VT	-	+	+	-	+
- Sustained VT	?	+	+	-	+

GP = general population; pLVEF=cardiac disease, preserved LVEF; rLVEF= cardiac disease, reduced LVEF;

**Presence of these different ECG risk-stratifiers gives information about:**

- A possible tachycardia substrate
- Tachycardia triggers
- The most likely tachycardia mechanism
- Autonomic status
- Genetic background
- Effect of management

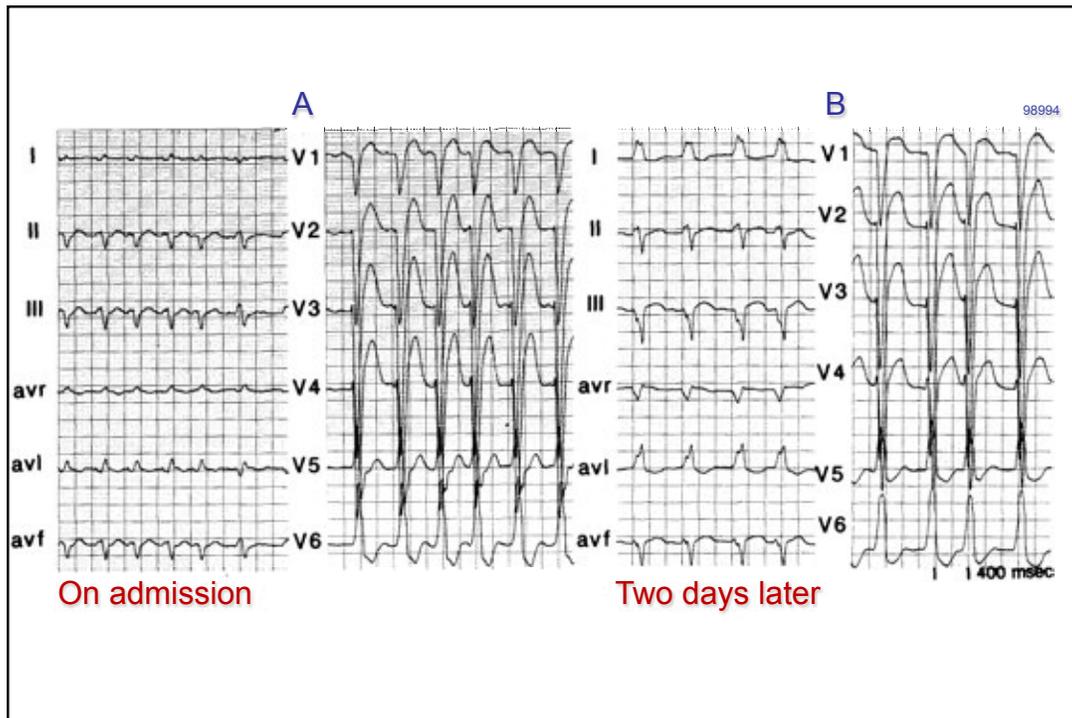
## Challenge

What will be the best combination of these ECG derived risk stratifiers in different clinical settings using the 12 lead ECG and Holter?

## Progress in recent years using the ECG

Examples:

- The ECG in LBBB
- The ECG to localize the coronary occlusion site in acute MI



## Important questions when analysing the LBBB ECG

1. QRS width
2. QRS axis in the frontal plane?
3. QRS voltage extremity leads vs precordial leads?
4. Notching in second half of QRS?
5. Duration of P-R interval?
6. RV enlargement/dysfunction?

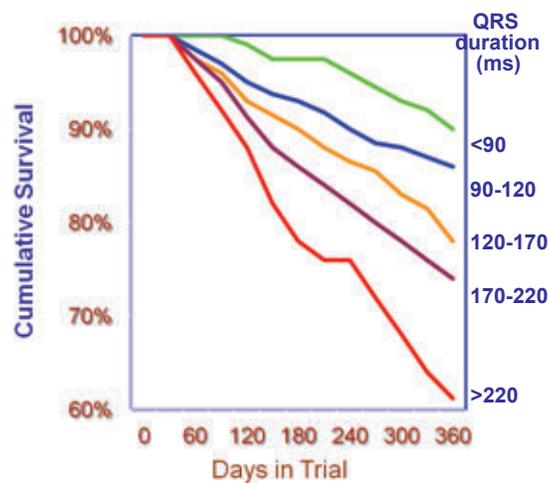
## ECG findings indicating poor prognosis in LBBB

1. QRS width: the wider the worse!
2. QRS axis in frontal plane: superior axis is bad news.
3. Low QRS voltage extremity leads: bad news.
4. Notching in second half QRS: scar
5. Prolonged PR interval

## Wide QRS-Proportional Mortality Increase

Vesnarinone Study<sup>1</sup>  
(VEST study analysis)

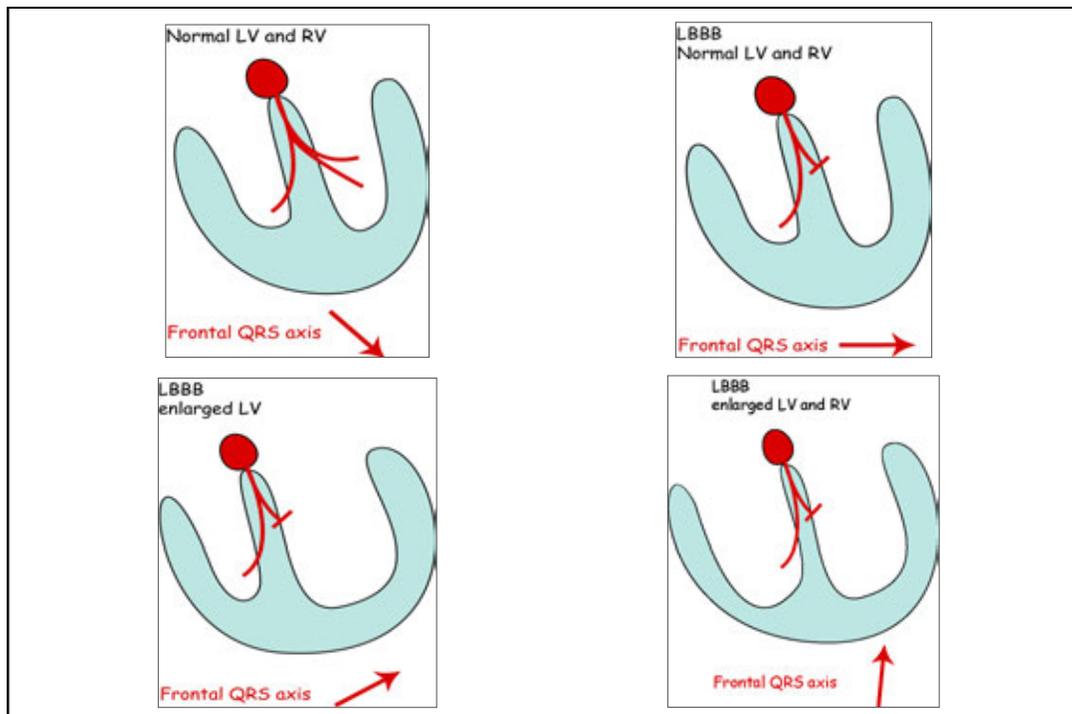
- NYHA Class II-IV patients
- 3,654 ECGs digitally scanned
- Age, creatinine, LVEF, heart rate, and QRS duration found to be independent predictors of mortality
- Relative risk of widest QRS group 5x greater than narrowest



<sup>1</sup> Gottipaty V, Krelis S, et al. ACC 1999 [Abstr];847-4

## ECG findings indicating poor prognosis in LBBB

1. QRS width: the wider the worse!
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## Frontal QRS axis in LBBB

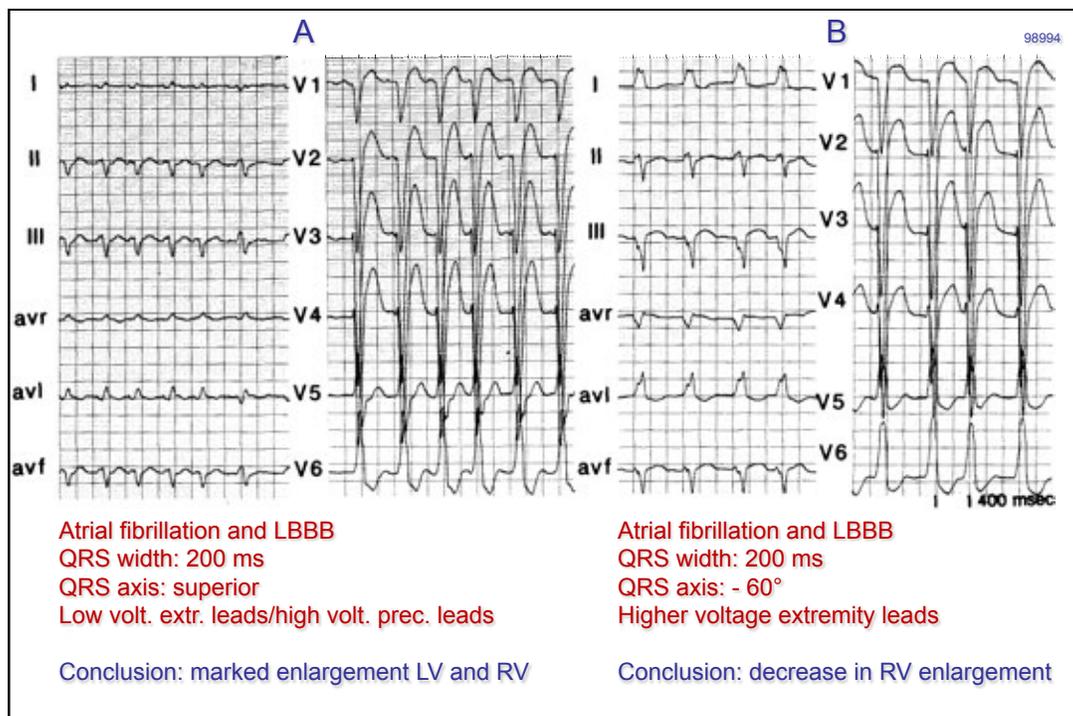
Superior axis indicates  
both left and right  
ventricular enlargement!

### ECG findings indicating poor prognosis in LBBB

1. QRS width: the wider the worse!
2. QRS axis in frontal plane: superior axis is bad news.
3. Low QRS voltage extremity leads: bad news.
4. Notching in second half QRS: scar
5. Prolonged PR interval

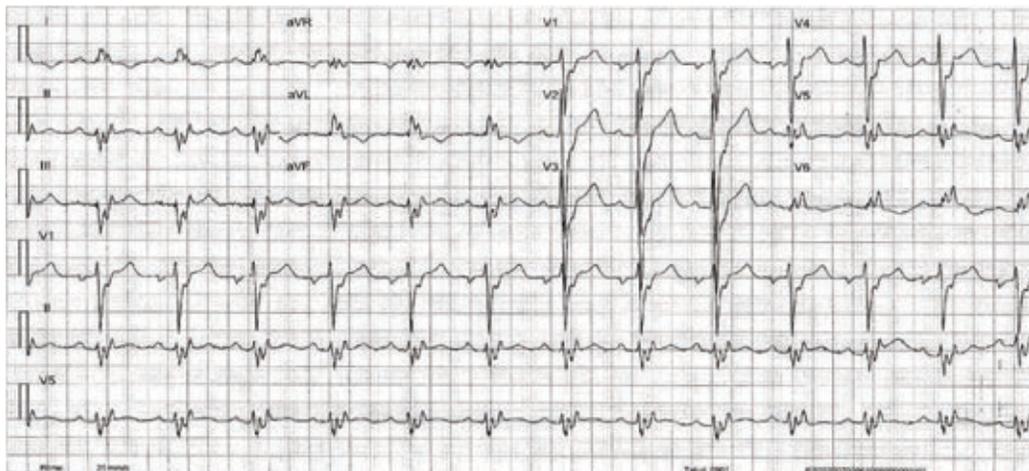
## ECG findings indicating poor prognosis in LBBB

1. QRS width: the **wider** the **worse!**
2. QRS axis in frontal plane: **superior axis** is **bad news**.
3. Low QRS voltage extremity leads: **bad news**.
4. **Notching** in second half QRS: **scar**
5. **Prolonged PR interval**



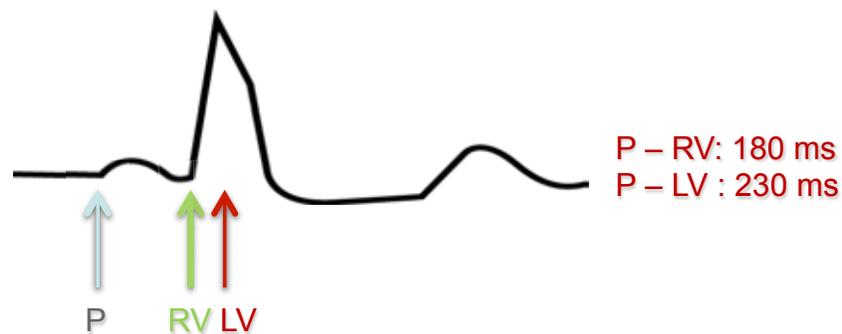
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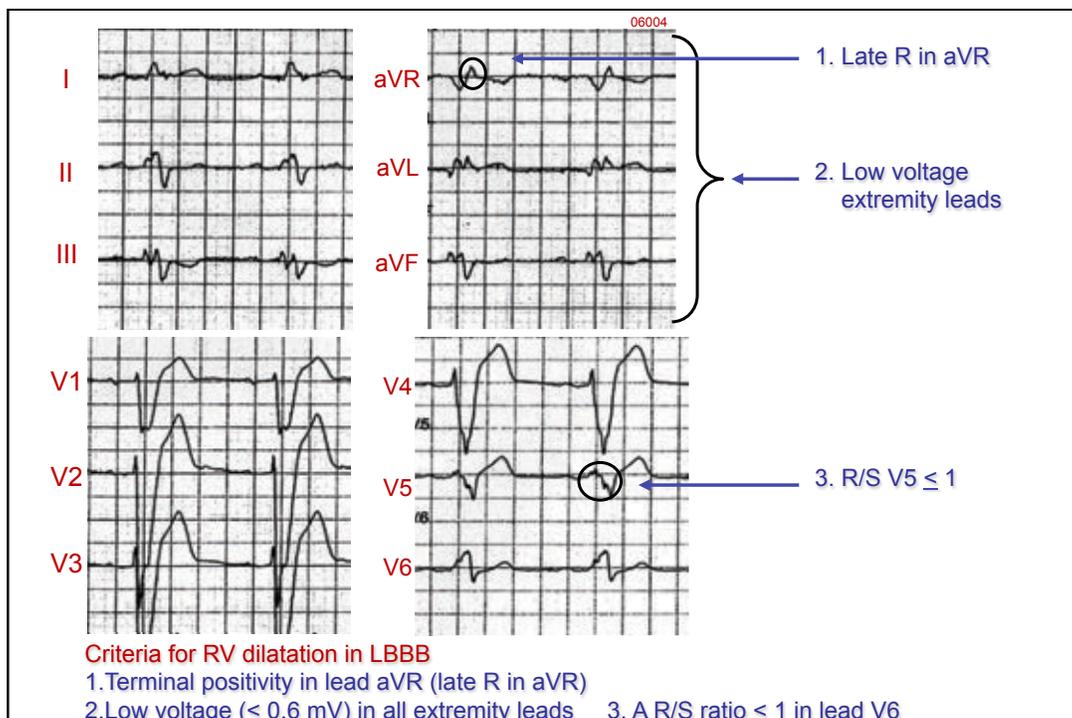
## ECG findings indicating poor prognosis in LBBB

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5. Prolonged PR interval



LV activation in LBBB 50-60 ms after start RV activation  
 → diastolic MI  
 reduced LV filling

# How to diagnose right ventricular enlargement in the patient with LBBB?



### Criteria for RV dilatation in LBBB

1. Terminal positivity in lead aVR (late R in aVR)
2. Low voltage ( $< 0.6$  mV) in all extremity leads
3. A R/S ratio  $< 1$  in lead  $V_5$

**$\geq 2$  Criteria positive:**

+ PV 89%

- PV 88%

Van Bommel RJ et al; Am J Cardiol 2011; 107:736-740

In acute cardiac ischemia the exact site of occlusion in the coronary artery and thereby the size of the area at risk is indicated by the direction of the deviation of the ST segment, because the ST segment deviation vector points to the dominant ischemic area.

**This knowledge is important to recognize a proximal coronary occlusion!**

Increasing super-specialization in cardiology threatens the implementation of new ECG knowledge in daily cardiology practice.

Both old and recent knowledge of the ECG should be in the core curriculum of every cardiologist, not only during the training phase but also during postgraduate education!

How to use the ECG to localize the exact site of the coronary artery occlusion in the patient with acute chest pain?

## The ST deviation vector

The ST deviation vector points to the dominant area of cardiac ischemia revealing the site and size of the area at risk and the location of the occlusion in the culprit coronary artery.

