Early repolarization syndrome: myth or reality?

Prof. Dr. Martin Borggrefe
Mannheim

Advances in Cardiovascular Arrhythmias and Great Innovations in Cardiology
Turin, October 20-22, 2011
Early Repolarization Syndrome

M 15y

M 17y

M 45y

M 38y

I

II

III

aVF

V1

V4

V5

V6
Screening of all idiopathic VF from ICD databases

Early repolarization on Baseline ECGs defined as:

- Slurring (late delta) or notch at the end of QRS, with J point > 0.1 mV in ≥ 2 leads

- Left precordial and/or inferior and/or lateral ECG leads (excluding V1-V3 for Brugada/ARVD)

Early Repolarization Syndrome

Prevalence ~ 5% of the population


First description: 1936
Shipley and Hallaran

- normal variant
- “normal RS-T segment elevation variant”
- “juvenile ST pattern”
Early Repolarization Syndrome

M 22yrs

April 2004

March 2006
Early Repolarization Syndrome

Ventricular Fibrillation with ‘Early Repolarization’

- **Incidence of early repolarization**
  31% ie 66 pts with IVF vs 4% in controls (p=0.002)

- **Amplitude of J point**
  2.15±1.2mm in IVF vs 1.05±0.2mm in controls

Early Repolarization Syndrome

Sudden Cardiac Arrest Associated with Early Repolarization

Michel Haïssaguerre, M.D., Nicolas Derval, M.D., Frederic Sacher, M.D., Laurence Jesel, M.D., Isabel Deisenhofer, M.D., Luc de Roy, M.D., Jean-Luc Pasquié, M.D., Ph.D., Akihiko Nogami, M.D., Dominique Babuty, M.D., Sinikka Yli-Mayry, M.D., Christian De Chillou, M.D., Patrice Scanu, M.D., Philippe Mabo, M.D., Seiichiro Matsuo, M.D., Vincent Probst, M.D., Ph.D., Solena Le Scouarnec, Ph.D., Pascal Defaye, M.D., Juerg Schlaepfer, M.D., Thomas Rostock, M.D., Dominique Lacroix, M.D., Dominique Lamaison, M.D., Thomas Lavergne, M.D., Yoshifusa Aizawa, M.D., Anders Engelund, M.D., Frederic Anselme, M.D., Mark O’Neill, M.D., Meleze Hocini, M.D., Kang Teng Lim, M.B., B.S., Sebastien Knecht, M.D., George D. Veenhuyzen, M.D., Pierre Bordachar, M.D., Michel Chauvin, M.D., Pierre Jais, M.D., Gaëlle Coureau, Ph.D., Genevieve Chene, Ph.D., George J. Klein, M.D., and Jacques Clémenty, M.D.

Early Repolarization Syndrome

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Long-Term Outcome Associated with Early Repolarization on Electrocardiography


Early Repolarization Syndrome

Death from Cardiac Causes and from Arrhythmia in Subjects with J-Point Elevation

![Graph showing survival free of death from cardiac causes](graph.png)

- No J-point elevation: Survival curve
- J-point elevation >0.2 mV in inferior leads: stairs-like curve

Survival Free of Death from Cardiac Causes

No. at Risk:
- No J-point elevation: 10,234, 9561, 8357, 6485, 1708
- J-point elevation: 36, 34, 23, 15, 1

P < 0.001

Association of Early Repolarization Pattern on ECG with Risk of Cardiac and All-Cause Mortality: A Population-Based Prospective Cohort Study (MONICA/KORA)


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Sinner et al, PLOS Medicine 2010; 7:e1000314
## Early Repolarization Syndrome

<table>
<thead>
<tr>
<th>ERP Prevalence n (%)</th>
<th>n Study Population (%)</th>
<th>n Death from Cardiac Causes (%)</th>
<th>n Death from Any Cause (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total n</td>
<td>6,213</td>
<td>511</td>
<td>1,496</td>
</tr>
<tr>
<td>Overall</td>
<td>812 (13.1)</td>
<td>89 (17.4)</td>
<td>244 (16.3)</td>
</tr>
<tr>
<td>Antero-lateral leads</td>
<td>275 (4.4)</td>
<td>25 (4.9)</td>
<td>78 (5.2)</td>
</tr>
<tr>
<td>Inferior leads</td>
<td>474 (7.6)</td>
<td>58 (11.4)</td>
<td>149 (10.0)</td>
</tr>
<tr>
<td>Combined antero-lateral and inferior leads</td>
<td>63 (1.0)</td>
<td>6 (1.2)</td>
<td>17 (1.1)</td>
</tr>
<tr>
<td>Slurring morphology</td>
<td>590 (9.5)</td>
<td>58 (11.4)</td>
<td>161 (10.8)</td>
</tr>
<tr>
<td>Notching morphology</td>
<td>219 (3.5)</td>
<td>31 (6.1)</td>
<td>83 (5.6)</td>
</tr>
<tr>
<td>Men</td>
<td>439 (7.1)</td>
<td>60 (11.7)</td>
<td>160 (10.7)</td>
</tr>
<tr>
<td>Women</td>
<td>372 (6.0)</td>
<td>29 (5.7)</td>
<td>84 (5.6)</td>
</tr>
<tr>
<td>35–54 y</td>
<td>422 (11.9)</td>
<td>20 (19.0)</td>
<td>57 (15.4)</td>
</tr>
<tr>
<td>55–64 y</td>
<td>277 (14.3)</td>
<td>51 (20.8)</td>
<td>120 (18.0)</td>
</tr>
<tr>
<td>65–74 y</td>
<td>114 (15.6)</td>
<td>18 (11.2)</td>
<td>67 (14.6)</td>
</tr>
</tbody>
</table>
Representative examples of ERP from our study population

Sinner et al, PLOS Medicine 2010; 7:e1000314
## Association of ERP with cardiac mortality

Sinner et al, PLOS Medicine 2010; 7:e1000314

<table>
<thead>
<tr>
<th>Study population</th>
<th>Substrata</th>
<th>ERP in any localization</th>
<th>ERP in inferior localization</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>HR (95% CI)</td>
<td>p-Value</td>
</tr>
<tr>
<td>All</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Main effect</td>
<td>ERP</td>
<td>3.44 (1.52–7.80)</td>
<td>0.003</td>
</tr>
<tr>
<td></td>
<td>ERP x age</td>
<td>0.95 (0.92–0.99)</td>
<td>0.005</td>
</tr>
<tr>
<td>Age-strata</td>
<td>35-54 y</td>
<td>1.96 (1.05-3.68)</td>
<td>0.035</td>
</tr>
<tr>
<td></td>
<td>55-64 y</td>
<td>1.12 (0.70-1.78)</td>
<td>0.63</td>
</tr>
<tr>
<td></td>
<td>65-74 y</td>
<td>0.59 (0.25-1.44)</td>
<td>0.25</td>
</tr>
<tr>
<td>Women</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Main effect</td>
<td>ERP</td>
<td>5.97 (0.85-42.04)</td>
<td>0.073</td>
</tr>
<tr>
<td></td>
<td>ERP x age</td>
<td>0.93 (0.86-1.00)</td>
<td>0.56</td>
</tr>
<tr>
<td>Age-strata</td>
<td>35-54 y</td>
<td>1.25 (0.34-4.58)</td>
<td>0.73</td>
</tr>
<tr>
<td></td>
<td>55-64 y</td>
<td>0.99 (0.39-2.50)</td>
<td>0.99</td>
</tr>
<tr>
<td></td>
<td>65-74 y</td>
<td>0.63 (0.15-2.72)</td>
<td>0.54</td>
</tr>
<tr>
<td>Men</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Main effect</td>
<td>ERP</td>
<td>2.69 (1.10-6.60)</td>
<td>0.030</td>
</tr>
<tr>
<td></td>
<td>ERP x age</td>
<td>0.96 (0.93-1.00)</td>
<td>0.058</td>
</tr>
<tr>
<td>Age-strata</td>
<td>35-54 y</td>
<td>2.65 (1.21-5.83)</td>
<td>0.015</td>
</tr>
<tr>
<td></td>
<td>55-64 y</td>
<td>1.16 (0.67-2.02)</td>
<td>0.60</td>
</tr>
<tr>
<td></td>
<td>65-74 y</td>
<td>0.67 (0.21-2.08)</td>
<td>0.49</td>
</tr>
</tbody>
</table>
Early Repolarization Syndrome

Kaplan-Meier curve: ERP, 35-54 yrs

Kaplan-Meier curve: inferior ERP, 35-54 yrs

Sinner et al, PLOS Medicine 2010; 7:e1000314
## Epidemiology

<table>
<thead>
<tr>
<th>Prevalence:</th>
<th>1-9% of the general population</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>15-70% of IVF cases</td>
</tr>
</tbody>
</table>

4-10-fold SD risk
Early Repolarization Syndrome

Epidemiology aspects

Gender: young (black) males

- large $I_{To}$ density

- Age <30
- Physical activity
- Vagal tone
- Slow heart rate
- (spinal cord injury)
Early Repolarization Syndrome

Electrocardiographic features (I)

- Heart rate: sinus bradycardia
- I° AV block: 5-39%
- Vertical electrical axis
- QRS duration \( \sim 90 \pm 10 \text{ ms} \)
- Tall R waves
Early Repolarization Syndrome

Electrocardiographic features (II)

- **St segment elevation**
  - elevation greater in precordial leads (prominent $V_4$)
  - distinct J wave in leads $V_4$–$V_6$

- **Localization of ST elevation**
  - inferior
  - lateral
  - mid precordial

  ➔  "**risky elevation**"

  ➔  "**benign elevation**"
J-Point Elevation in Survivors of Primary Ventricular Fibrillation and Matched Control Subjects

Incidence and Clinical Significance

Raphael Rosso, MD,* Evgeni Kogan, MD,* Bernard Belhassen, MD,* Uri Rozovski, MD,* Melvin M. Scheinman, MD,§ David Zeltser, MD,* Amir Halkin, MD,* Arie Steinvil, MD,* Karin Heller, MD,* Michael Glikson, MD,† Amos Katz, MD,‡ Sami Viskin, MD*

Tel Aviv and Beer-Sheva, Israel; and San Francisco, California

J Am Coll Cardiol 2008; 52: 1231-1238
# Early Repolarization Syndrome

## Incidence of J-Point Elevation Among 45 Patients With Idiopathic VF and 124 Healthy Control Subjects Matched for Age and Gender

<table>
<thead>
<tr>
<th></th>
<th>Idiopathic VF</th>
<th>Control Subjects</th>
<th>p Value*</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Any lead</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any J-point elevation</td>
<td>19 (42.2%)</td>
<td>16 (13.0%)</td>
<td>0.001</td>
<td>3.2</td>
<td>1.7-6.3</td>
</tr>
<tr>
<td>J-point &gt;1.0 mm</td>
<td>14 (31.1%)</td>
<td>11 (8.9%)</td>
<td>0.002</td>
<td>3.4</td>
<td>1.5-7.5</td>
</tr>
<tr>
<td><strong>Inferior leads</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any J-point elevation</td>
<td>12 (26.7%)</td>
<td>10 (8.1%)</td>
<td>0.006</td>
<td>3.2</td>
<td>1.4-7.5</td>
</tr>
<tr>
<td>J-point &gt;1.0 mm</td>
<td>8 (17.8%)</td>
<td>8 (6.5%)</td>
<td>0.052</td>
<td>2.6</td>
<td>1.0-7.1</td>
</tr>
<tr>
<td><strong>Leads I and aVL</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any J-point elevation</td>
<td>6 (13.3%)</td>
<td>1 (0.8%)</td>
<td>0.009</td>
<td>16.9</td>
<td>2.0-140.3</td>
</tr>
<tr>
<td>J-point &gt;1.0 mm</td>
<td>5 (11.1%)</td>
<td>0 (0)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Leads V₄ to V₆</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any J-point elevation</td>
<td>3 (6.7%)</td>
<td>9 (7.3%)</td>
<td>0.860</td>
<td>0.9</td>
<td>0.2-3.3</td>
</tr>
<tr>
<td>J-point &gt;1.0 mm</td>
<td>3 (6.7%)</td>
<td>6 (4.9%)</td>
<td>0.686</td>
<td>1.3</td>
<td>0.3-5.3</td>
</tr>
</tbody>
</table>

Rosso et al, J Am Coll Cardiol 2008; 52: 1231-1238
Incidence of Influence of Gender on the Incidence of J-Point Elevation in the Different Patient Groups

Rosso et al, J Am Coll Cardiol 2008; 52: 1231-1238
Distribution of J Waves Among Patients With Idiopathic VF, Matched Control Subjects, and Healthy Athletes

Rosso et al, J Am Coll Cardiol 2008; 52: 1231-1238
J-ST-T waves before and after electrical storm

Nam et al, Eur Heart J 2010; 31:330-339
Potential Mechanism of Early Repolarization

Early Repolarization Syndrome

Benito et al, J Am Coll Cardiol 2010;56:1177–86
Early Repolarization Syndrome

APs in Epicardium and Endocardium
With the Main Underlying Ionic Currents

Action potential

Ionic currents

Surface ECG

Benito et al, J Am Coll Cardiol 2010;56:1177–86
Early Repolarization Syndrome
Potential Mechanism for Early Repolarization Arrhythmogenesis

- Transmural voltage gradient
- Transmural and epicardial dispersion of repolarization

Endo
Epi₁
Epi₂
ECG

Benito et al, J Am Coll Cardiol 2010;56:1177–86
### Early Repolarization Syndrome

#### Inherited SCD syndromes involving ER

<table>
<thead>
<tr>
<th>Gene mutations/ion current</th>
<th>ER Syndrome</th>
<th>Brugada Syndrome</th>
<th>Short-QT Syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>KCNJ8/(I_{KATP}) (35)</td>
<td>SCN5A, SCN1B, SCN3B/(I_{Na}) (51,54,55)</td>
<td>KCNH2/(I_{Kr}) (59)</td>
<td></td>
</tr>
<tr>
<td>CACNA1C, CACNB2B/(I_{Ca,L}) (43)</td>
<td>GPD1-(I_{Na}) (52)</td>
<td>KCNQ1/(I_{Ks}) (60)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>CACNA1C, CACNB2B/(I_{Ca,L}) (53)</td>
<td>KCNJ2/(I_{K1}) (61)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>KCNE3/(I_{to}) (55)</td>
<td>CACNA1C, CACNB2B/(I_{Ca,L}) (53)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ECG</th>
<th>J-wave; ST-segment elevation</th>
<th>J-wave; ST-segment elevation</th>
<th>Short-QT; peaked T waves</th>
</tr>
</thead>
</table>

| Drug therapy | Quinidine (16) | Isoproterenol (15) | Quinidine (48) | Isoproterenol (49) | Quinidine (50) |

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Benito et al, J Am Coll Cardiol 2010;56:1177–86
Early Repolarization Syndrome

ER Effects on Action Potentials and ECG, Illustrating the Potential Role of J-Wave as a Marker of ER Risk

Benito et al, J Am Coll Cardiol 2010;56:1177–86
Giant Osborn Waves in Hypothermia

Temperature (°C)  | 24.1 | 29.4 | 36.6
Heart rate (beats/min) | 50  | 70   | 98
QRS interval (msec)   | 184 | 119  | 71
QTc interval (msec)   | 516 | 502  | 403
Early Repolarization Syndrome

Possible mechanisms

**Early** voltage gradients (phase 1)

- J wave
- phase 2 re-entry

**Late** voltage gradients

- ST-segment elevation
Early Repolarization Syndrome

**Genetic findings**

<table>
<thead>
<tr>
<th>KCNJ8 gene</th>
<th>inward rectifier ATP-dependent-K$^+$-channel</th>
</tr>
</thead>
<tbody>
<tr>
<td>CACNB2B gene</td>
<td>Ca$^+$-channel submit</td>
</tr>
</tbody>
</table>
Early Repolarization Syndrome

Unresolved issues

**J wave =** marker of risk ?

**Regional ER =** mechanisms ?

**Brugada syndrome =** right precordial ER ?

- Male gender
- Vagal tone
- Response to quinidine/isoproterenol
F 14 y

- Brady 48/min
- Calcium IV
- Isoproterenol
- Flecainide

Electrocardiogram tracings for different conditions and treatments, with leads I, II, III, aVR, aVL, aVF, V1 to V6.
Early Repolarization Syndrome

Therapeutic considerations

Secondary prevention

ER + VF → ICD

ER + electrical storm → isoproterenol, pacing, ablation?

Primary prevention

?
Early Repolarization Syndrome
The characteristic finding of this ECG is the \textit{lambda like}^1 \textit{or Gussak}^2 \textit{shape ST elevation} which goes with the previously reported cases about electrical storm.

Lambda-like ST segment elevation in acute myocardial infarction – a new risk marker for ventricular fibrillation? Three case reports

Uniesienie odcinka ST o kształcie litery lambda w ostrej fazie zawału serca – nowy wskaźnik ryzyka wystąpienia migotania komór? Opis trzech przypadków

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¹ The H. Klimontowicz Hospital, Gorlice
² 1st Department of Cardiology, Collegium Medicum, Jagiellonian University, Kraków
³ Department of Cardiology, Medical Center, Opole

Abstract

Sudden cardiac death (SCD) is responsible for almost 50% of all cardiac deaths in the U.S. The most common cause of SCD is coronary artery disease, especially during acute myocardial infarction (AMI). There are no publications concerning the shape of ST segment elevation in AMI and the risk of ventricular fibrillation (VF) or SCD. We present three cases with AMI and atypical ST segment elevation – lambda-wave-like complicated with episodes of VF.
Early Repolarization Syndrome
Electrical storm in acute myocardial infarction

Ihor Gussak, MD, PhD, FACC¹, Preben Bjerregaard, MD, DMSc, FACC²

¹ University of Medicine and Dentistry of New Jersey, Robert Wood Johnson Medical School, New Brunswick, NJ USA
² Saint Louis University Hospital, St Louis, MO USA

In the USA, the annual rate of acute myocardial infarction (AMI) is close to one million, and almost one quarter of patients with AMI will die suddenly due to the development of fatal ventricular tachyarrhythmias, such as ventricular tachycardia (VT) and ventricular fibrillation (VF) [1]. Although in more than half of such cases SCD occurs as the first symptom of coronary artery disease [2], conventional cardiovascular risk factors are not predictive of ‘coronarogenic’ SCD [3]. The search for identification of patients at risk for SCD, including those with AMI, has intensified in recent years. Promising results from experimental and clinical studies have emphasized the pivotal role of family history and subclinical mutation in cardiac channels in the development of repetitive life-threatening arrhythmias during acute ischemia, commonly described as ‘electrical storm’.

The term ‘electrical storm (ES)’ is commonly defined as a state of transient critically impaired electrical stability of the heart culminating in a sequence of life-threatening ventricular tachyarrhythmias (either self-terminating or requiring multiple electrical defibrillations) within a short time (typically during a 24-hour period), although there has been no consistency in the definition of this term. ES is highly resistant to prevention and treatment and is associated with pure clinical outcome, even in patients with implantable cardioverter-defibrillators [4]. Most frequently ES is observed in patients with: (a) primary electrical diseases (PED) of the heart, (b) acute ischemic (‘coronarogenic’) event, (c) hypothermia during aggressive rewarming, and (d) drug-associated cardiac toxicity. Neither the mechanism/s nor the precipitating factors for ES are well defined, although genetic mutation of cardiac ion channels or gap junctions are considered as a highly likely predisposing factor for ES in ACS. For instance, Dr. Dan Hu et al. [5] have identified ES in 1 out of 19 consecutive patients who developing VF during AMI. The patient with ES was the only one carrying the SCNSA mutation. Interestingly, this patient developed his first VF at age 70 years and only in the setting of AMI.

In the article by Dr. Piotr Kukla [6] published in this issue of the Journal, the authors present three patients with AMI associated with multiple episodes of polymorphic VT and VF that were observed during a short period of time.
Early Repolarization Syndrome

Summary

Thus, early repolarization syndrome is reality!
Early Repolarization Syndrome
Repolarization wave but not depolarization

Gradual/beat to beat fluctuations in contrast with QRS- attenuation during effort – possible accentuation by vagal maneuvers- No/little change with NaBlocker- HV normal

Rare late potentials - No endocardial activity synchronous of J-wave
Early Repolarization Syndrome

The less common causes of ST segment elevation (I)

1. Brugada syndrome
2. **Idiopathic ventricular fibrillation related to a prominent J wave in the inferior leads** (a variant of BrS with ST-segment elevation in the inferior leads but no coved or saddleback ST-segment elevation in the right precordial leads) [31] Gussak et al. [32] named this wave as lambda wave due to its morphology
3. **Arrhythmogenic right ventricular dysplasia:** Sometimes, the electrocardiographic phenotype is impossible to differentiate from the electrocardiographic pattern in BrS [33, 34]. In these cases, observed in the so-called minor or concealed forms, only magnetic nuclear resonance is useful in differentiating both entities
4. J wave syndrome
   4.1. J wave in hypothermal patients
   4.2. J wave in normothermal patients [35]
   4.2.A. Nervous system injuries: Acute brain injury i.e. subarachnoid hemorrhage, cardiac arrest, and dysfunction of cervical sympathetic system [36]
   4.2.B. Extreme hypercalcemia [37–42]
5. **Marked hyperpotassemia** [43]
6. **Myocardial bridging of the left anterior descending artery** [44]
7. Mitral valve prolapse syndrome [45]
8. **Prinzmetal’s angina secondary to coronary artery spasm:** Reversible ST segment elevation [45]
9. **Acute aortic dissection of the ascending aorta (type A)** [46]

Pérez Riera et al, Cardiol J 2008;15:4-16
10. **Transient left ventricular apical ballooning syndrome, transient apical ballooning syndrome without coronary stenosis, tako-tsubo cardiomyopathy or “broken heart”**: An acute and unique cardiac syndrome characterized by typical ischemic chest symptoms (chest pain or dyspnea), with ECG that shows ST-segment elevation and T-wave inversion, and minor elevated cardiac enzyme levels. Cardiac catheterization reveals absence of coronary stenosis. Left ventriculography and cardiac magnetic resonance imaging shows apical akinesia and compensatory hypercontractility of the basal ventricular segments (apical ballooning). Left ventricular systolic function recovers from ejection fraction. Wall abnormalities return to normal can occur after as long as 3 months. This new clinical entity may have a catecholamine-mediated neurogenic mechanism as the etiopathogenic substrate.

11. **Acute myocarditis or myopericarditis** [47]
12. Chagasic cardiomyopathy [48]
13. **Hypertrophic cardiomyopathy**: The electrocardiographic features of hypertrophic cardiomyopathy are numerous, including ST segment elevation that may simulate other ST segment elevation syndromes [49]
14. **After mitral valvuloplasty** [50]
15. **Septic noncardiogenic shock** [51]
16. **Cardiac tumor** [52]
17. **Acute pancreatitis**: It is considered a stress-related cardiomyopathy similar to transient apical ballooning syndrome without coronary stenosis [53]. More than 50% of the patients with acute pancreatitis have ECG abnormalities, and these changes could be related also to electrolyte alterations [54]
18. **Anaphylactic reaction/anaphylactic shock** [55]
19. **Gallbladder disease**: Acute cholecystitis or biliary colic may be associated with angina pectoris, arrhythmias, or nonspecific ST-T wave changes on the ECG. A vagally mediated cardio-biliary reflex is the presumed cause of these changes. The signs and symptoms of gallbladder and heart disease may overlap, making diagnosis difficult [56]
## Early Repolarization Syndrome
### Comparison of ECG

<table>
<thead>
<tr>
<th></th>
<th>ERV</th>
<th>Acute pericarditis</th>
<th>AMI</th>
<th>BrS type 1</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ST segment appearance</strong></td>
<td>Concave to the top</td>
<td>Concave to the top</td>
<td>Concave to the top</td>
<td>Convex to the top</td>
</tr>
<tr>
<td><strong>Pathological Q waves</strong></td>
<td>Absent</td>
<td>Absent</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td><strong>Mirror image changes</strong></td>
<td>Only in aVR</td>
<td>Absent</td>
<td>Present</td>
<td>Possible</td>
</tr>
<tr>
<td><strong>Leads involved</strong></td>
<td>Limb and precordial</td>
<td>Limbs and precordial</td>
<td>Segmentary pattern</td>
<td>Right precordial</td>
</tr>
<tr>
<td><strong>R voltage</strong></td>
<td>Normal or minimally augmented</td>
<td>Normal</td>
<td>Lost</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>PR interval</strong></td>
<td>Not affected</td>
<td>Possible depression</td>
<td>Variable</td>
<td>50% prolongation</td>
</tr>
<tr>
<td><strong>ST/T ratio in lead V6</strong></td>
<td>&lt; 0.25</td>
<td>&gt; 0.25</td>
<td>Not applicable</td>
<td>Not applicable</td>
</tr>
</tbody>
</table>

Pérez Riera et al, Cardiol J 2008;15:4-16