# The J wave of the ECG. Concepts

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The J wave is a positive deflection in the ECG normal (present in 2%–14% of healthy individuals and is more prevalent in young males, particularly if athletic and African descent. Additionally, ERP is a common finding in young teen athletes (the prevalence in the athletic population rises to 20-90%.).1 In this population ERP in both inferior and lateral leads is more common (18.2%) than isolated inferior (9.1%) or lateral (8.2%) ERP. Young age might be a contributing factor in causing a more diffuse repolarization abnormality. 2 or pathological that occurs approximately (There is an overlap of ≈10 msec. 3) after the junction between the end of the QRS complex and the beginning of the ST segment, also known as the J point (junction point), QRS end, J-junction, ST0 [zero millisecond] or ST beginning to occur after the notch/slur or J wave 4. It is described as J deflection as slurring/lambda 5 or notching of the terminal portion of QRS complex. Currently, J waves, is defined as an elevation of the QRS-ST junction ≥1 mm either as QRS slurring or notching in at least 2 contiguous leads. Additionally, when it becomes more accentuated, it may appear as a small, R wave (R′) or ST segment elevation.

The term J deflection or J-wave has been used to designate the formation of the wave produced when there is a large, prominent deviation of the J point from the baseline with two shapes: notching/spike-and-slurring/lambda 5 or dome 6 variety. All J-wave deflections do not look alike. Some are elevations of J-point and ST segments ≥2 mm followed by negative symmetrical T-wave in leads V1 and/or V2 in at least one lead: BrS whereas others are of the spike-and-dome variety. This suggests that different mechanisms may be responsible for the size and shape of J-wave deflections. The J point in the ECG is the point where the QRS complex joins the ST segment. It represents the approximate end of depolarization and the beginning of repolarization as determined by the surface ECG. There is an overlap of ≈10 msec3

***The numerous denominations for J wave***

J wave has been named by different nomenclatures 7: The nonspecific camel "hump sign", 8 Osborn wave, 6; 8 late delta (d) wave, 9;10 a hook junction, hypothermic wave or hypothermic hump, 11, 12 camel hump

sign, **13** J point wave, **14** K wave, **15** H wave, injury current **3, 6;16;17** or more specifically positive injury current. It represents the approximate end of depolarization and the beginning of repolarization as determined by the surface ECG. There is an overlap of ≈10 msec.

## I) Early Repolarization Syndrome/J-Wave Syndromes (ERS/ JWSs)

ERS or JWSs is diagnosed in the presence of J-point elevation ≥1 mm in ≥2 contiguous in the inferior and/or lateral leads of a standard 12-lead ECG in a patient resuscitated from otherwise unexplained VF/PVT. ERS or JSW can be diagnosed in an SCD victim with a negative autopsy and medical chart review with a previous ECG demonstrating J-point elevation ≥1 mm in ≥2 contiguous in the inferior and/or lateral leads of a standard 12-lead ECG.[30] Sudden death in JWS occurs predominantly in men at night when parasympathetic tone is strong. Acetylcholine (ACh) activates ventricular the parasympathetic transmitter, activates $I_{KAS}$. ACh and ajmaline induce JWS and facilitate the induction of ventricular arrhythmias more in male than in female ventricles. **18** JWSs are defined as a distinct ECG phenotype (slur/notch) affecting the junction between the QRS complex and the ST segment in the inferolateral leads. The background mechanisms have heterogeneous substrates related to either delayed depolarization due to microstructural alterations or early repolarization abnormalities. Both IVF and JWSs are the result of a wide spectrum of pathophysiologic processes. The individual phenotypic characterization is essential given its implications in therapy, genetic testing, and risk stratification. **19**

It is very important to determinate the ECG characteristics of the J wave because there are several aspects that are indicative of “innocent” or malignant J waves.

Table 1 shows the differential diagnosis between both patterns.

| Characters | Physiological J-wave, "innocent" J-waves | Pathologic J-waves, malignant J waves[29, 56]**20;21** |
|---|---|---|
| J-point elevation | <0.1 mV | High amplitude ≥0.2 mV |
| High of J wave | 1-2 mm rarely more | >0.2 mV |
| Transient/ fluctuating global J-wave augmentation | No | The occurrence of VF episodes is always accompanied by an accentuation of the J wave amplitude. Isoproterenol (1–4 μM/min) or pacing at rates of 90–100 bpm abolished these ECG changes and prevented the recurrence of VF.**21** Characteristic dynamic amplitude J-wave level portends a high risk for VF in patients with ER and should be closely monitored, since this could signify an imminent risk for the development of Electrical Storm. |

| | | |
|---|---|---|
| ST segment shape | Rapidly ascending/ upsloping ST-segment morphology after the J-point[57] and tall/symmetric T wave, is generally considered to be benign[25, 58] when there is 0.1 mV elevation of the ST-segment within 100 msec after the J-point and the ST-segment gradually merge with the T-wave. An ST-segment with upward concavity followed by a T-wave is seen in Caucasians, and an elevated ST-segment with upward convexity and negative T-wave in African-Caribbean athletes.[57, 59] | J-wave without typically rapidly ascending ST segment Horizontal or Down sloping when the ST-segment elevation is 0.1 mV within 100 ms after the J-point and continues as a flat ST-segment until the onset of the T-wave. In other words, e combination of J waves with horizontal/descending ST segment "horizontal/ descending" pattern. **22, 23** Note a flat is named "horizontal" |

| | | |
|---|---|---|
| ECG leads | Anterolateral ST-segment elevation. It is observed in 1-2 % of the general population. Or only lateral or inferior leads | Global J wave or widespread J-wave in inferior, lateral and anterior walls leads was associated with a higher incidence of VF recurrence in patients with JWS.**24, 25** |
| Coexisting channelopathies | No | Possible BrS, SQTS |
| Short coupled PVC | No | Suggestive and characteristic R-on-T phenomenon. The PVCs preceding the VF episodes in the Electrical Strom group exhibited significantly shorter coupling intervals than in patients with BrS; 328 (320, 340) msec vs. 395 (350, 404) msec Additionally, PVCs with a short–long–short (SLS) sequence was observed in more than 70% of cases in ERS. **21** |
| Unknown syncope or malignant of family history of SCD | Always absent | Frequently present |

Table 1. Electrocardiographic differences between benign versus malignant/evil of J wave patterns.

**Electrocardiographic J-Wave causes: Classification proposal**

**I)** Hypothermic J-wave (Osborn wave) **24** (core body temperature ≤95°F = 35°C). The amplitude and duration of Osborn waves is inversely related to core temperature.

**II)** Non-hypothermic conditions: The "normothermic" Osborn wave.

- *Physiological:* prevalent among healthy young male athletes, which displays an ERP predominantly in the lateral precordial leads. *Physiological* ERP is named type 1 ERP. Stable ST-segment elevation (STE) in the lateral leads (I, aVL, V5, V6). are frequently encountered in association with stable and transient

complete RBBB. These STE in the lateral ECG leads in patients with CBBB, represent parallel changes to the ST-segment depression seen in the V1-V3 leads, and both represent secondary ECG changes expected in patients with intraventricular conduction delays, and they are opposite in polarity to the latter part of the QRS complexes.**25** The following features suggest physiologic ERP: sinus bradycardia, phasic-respiratory sinus arrhythmia , axes of QRS, ST segment and T wave, oriented in the same direction in the frontal plane, deep and narrow Q waves followed by R wave of great voltage in left precordial leads, notch or slurring of R wave descending branch (J-wave ) possible but not obligatory, transition area in precordial leads of sudden occurrence, J point and ST segment elevation, usually < 2 mm (exceptionally it may be > 5 mm) of superior concavity in middle and/or left precordial leads and possibly in inferior leads, possible reduction in J point and ST segment elevation by sympathetic action and sympathomimetic drugs, absence of reciprocal or mirror image (exception in VR lead), symmetrical or pseudo symmetrical T waves, with great width and polarity matching QRS, the ERP consists of J-point and convex ST segment elevation, often with a notch at the intersection of the two. **26** It is frequently seen in athletes, up to 35%, **27** and is more common in those who are younger, male or of African-Caribbean descent.**28** While the ERP has been associated with SCA in the general population, especially if present in the inferior leads, it is enhanced by exercise training and there are no data suggesting an increased risk of SCA in young athletes. No secondary evaluation is needed for these athletes.

➢ *Pathological*

**A) Inherited arrhythmia syndromes**

· BrS

· ERS: **29** Both syndromes share similarities in their pathophysiology, genetic background, and clinical presentation.

· JWS: The term JWS incorporates 2 arrhythmogenic conditions, BrS and ERS, characterized by terminal QRS and ST segment abnormalities (J-wave) and by increased risk of cardiac events. JWS I called + ERS or type 3 ERS: Overlapping between BrS and ERS (highest

risk).30 Interesting VF can be suppressed by catheter ablation of the triggering PVCs originating from the Purkinje system, RVOT, and papillary muscles. 31; 32

- Congenital SQTS: 33; 34 The prevalence of ERP following MacFarlane et al. 2015 consensus criteria in a cohort of 73 SQTS patients, ER was present in 29% of their cases comparable with that observed in 146 age- and sex-matched controls, who demonstrated a prevalence of 27%, consequently ERP in congenital SQTS patient remains uncertain.

Note: IVF was not included because a recent publication suggests that IVF should not have a phenotype (negative phenotype). Polemic! 35

**B) Acquired**

- **Ischemic:**
  - STEMI. 36
  - Vasospastic angina, variant angina, or Prinzmetal angina. 37
  - Takotsubo Cardiomyopathy (TTC). 38; 39
  - Cardiopulmonary arrest from over sedation. 40
  - Accessory third papillary muscle with a prominent J-wave. 41
- **Severe hypercalcemia** 42;43;44;45;46;47
- **Neurological insults**
  - Subarachnoid hemorrhage (SAH) 48;49
  - Brain/head injuries
  - Transient postictal hemiplegia (Todd's paralysis) 50
  - Sympathetic nerves in the neck injury51-53
- **Acute myocarditis.** ST elevation with an ERP is associated with a better prognosis. On the other hand, are associated with poorer prognosis: pathological Q wave, broad QRS complex, QRS/T angle ≥ 100°, prolonged QT interval, high-degree atrioventricular block and malignant ventricular tachyarrhythmia. 54
- **LVH due to hypertension**24

**Inherited arrhythmia syndromes**

- **ER**
  - **Type 1:** which displays an ERP predominantly in the lateral precordial leads, and is prevalent among healthy young male athletes.

- **Type 2:** An ERP in inferior and lateral leads associated with moderate level of risk.

· **BrS**

· **JWSs** (also denominated **Type 3**): highest risk characterized by an ERP in inferior, lateral and right precordial leads (anteroseptal wall). **30**

· **SQTS**

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