Tidsskriftet Tidsskrift for Den norske legeforening

A man in his 30s with cardiac arrest

EDUCATIONAL CASE REPORT

MAGNUS NOSSEN

magnus.nossen@hotmail.com
Department of Cardiology
Oslo University Hospital, Ullevål
Magnus Nossen, speciality registrar in cardiology
The author has completed the ICMJE form and declares no conflicts of interest.

GEIR ØYSTEIN ANDERSEN

Department of Cardiology
Oslo University Hospital, Ullevål
Geir Øystein Andersen, specialist in internal medicine and cardiology, medical director at the Cardiac Intensive Care Unit
The author has completed the ICMJE form and declares no conflicts of interest.

Background

Early repolarisation syndrome (ERS) is a rare but important cause of sudden cardiac death in otherwise healthy individuals. It is characterised by early repolarisation changes on the electrocardiogram (ECG) and the occurrence of polymorphic ventricular tachycardia or ventricular fibrillation in the absence of other identifiable causes.

Case presentation

A man in his thirties was admitted to hospital following an out-of-hospital cardiac arrest. Coronary angiography and echocardiography revealed no causative abnormalities. Continuous 12-lead ECG monitoring during hospitalisation showed widespread and dynamic J-waves, short-coupled premature ventricular complexes (PVCs) and progressive J-wave augmentation leading up to an in-hospital cardiac arrest. Notably, J-wave augmentation was observed after post-extrasystolic pauses in the period preceding the in-hospital cardiac arrest. Ventricular fibrillation occurred during the phase of maximal J-wave augmentation and was triggered by an 'R-on-T'

PVC. Extensive evaluation failed to identify any alternative explanation for the cardiac arrest, and ERS was the final diagnosis. The patient in this case sustained severe anoxic brain injury, and ultimately the decision was made to withdraw life support.

Conclusion

ERS should be considered as a potential cause of cardiac arrest in patients who display early repolarisation changes on the ECG when no other aetiology is found. The diagnosis is further supported by the presence of dynamic J-waves, along with progressive and post-extrasystolic J-wave augmentation observed prior to cardiac arrest.

A man in his thirties undergoing treatment for hypertension was hospitalised following a cardiac arrest at home. We describe here an unusual cause of cardiac arrest and highlight the importance of thorough knowledge of ECG changes and careful ECG review in establishing the correct diagnosis.

The patient's spouse awoke to abnormal breathing sounds from the patient. Unable to rouse him, she initiated basic cardiopulmonary resuscitation, which continued for 15 minutes until the ambulance crew arrived. They found a shockable rhythm consistent with ventricular fibrillation. Return of spontaneous circulation was achieved after three defibrillation attempts, but the patient remained unconscious and was therefore intubated. The first ECG, obtained in the ambulance during ongoing adrenaline infusion, showed atypical ST elevation in leads I, aVL and V2–V6 (Figure 1). A repeat ECG 15 minutes later showed no ST elevation.

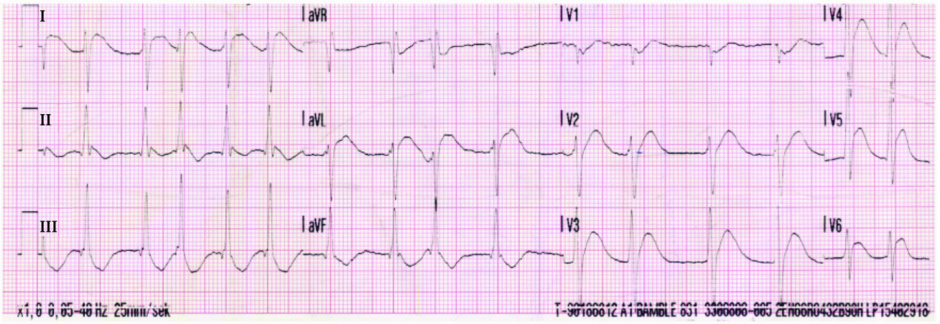

Figure 1 ECG recorded in the ambulance immediately after return of spontaneous circulation showed atrial fibrillation with atypical ST elevation in leads V2–V6, I and aVL (paper speed 25 mm/s).

The most common cause of cardiac arrest in Norway is cardiac arrhythmia associated with acute coronary syndrome (1). In younger patients, cardiomyopathies and channelopathies represent relevant differential diagnoses.

At the time of the initial hospital assessment, the patient was intubated and had a Glasgow Coma Scale score of 3. Blood pressure was 119/51 mmHg, supported by a low-dose adrenaline infusion (0.04 µg/kg/min). Heart rate was 106 beats per minute, and oxygen saturation was 96 %. The pupils were equal with intact light reflexes, and the corneal reflex was present bilaterally. A repeat ECG on admission showed sinus

tachycardia with no apparent cause for the cardiac arrest. No ischaemic ST-segment or T-wave changes were observed. Echocardiography showed mild concentric left ventricular wall hypertrophy but was otherwise unremarkable. Arterial blood gas analysis on admission revealed a mild combined respiratory and metabolic acidosis, without other significant abnormalities: sodium 144 mmol/L (reference range 137–144), chloride 112 mmol/L (98–107), pH 7.23 (7.35–7.45), pCO_2 6.9 kPa (4.7–6.0), pO_2 12.5 kPa (12.0–14.0), lactate 2.8 mmol/L (0.5–2.5), HCO_3^- 20.6 mmol/L (22.0–26.0), base excess (BE) −6 (−3 to 3), calculated anion gap 11 mmol/L (8–12), and an osmolal gap of 6 mosmol/kg H_2O (< 10).

Urgent coronary angiography is indicated in cardiac arrest patients who present with ST-elevation myocardial infarction, an ECG showing a STEMI equivalent, or in unstable patients where ischaemia is suspected as the underlying cause. As our patient demonstrated transient ST elevations on the initial ECG following return of spontaneous circulation, the criteria for coronary angiography were considered to be met. Pulmonary embolism, aortic pathology and cerebrovascular accident are also conditions with therapeutic implications that should be rapidly excluded. These conditions are less commonly associated with a shockable rhythm. Intoxication and adverse drug effects must be considered in all cardiac arrest patients without an obvious aetiology.

Coronary angiography revealed no coronary occlusion, coronary artery spasm or spontaneous coronary artery dissection. Computed tomography (CT) of the head, thorax and aorta ruled out pulmonary embolism, aortic disease and cerebrovascular catastrophe. Blood tests, including C-reactive protein (CRP), haemoglobin, leukocyte count and platelet count, were all within the reference ranges. Serum electrolyte levels showed no significant abnormalities and did not suggest a causal relationship. Clinically and biochemically, there was no suspicion of intoxication. Serum creatinine was 123 µmol/L (reference range 60–105), and the cardiac biomarker troponin T was elevated at 359 ng/L (< 14).

Interpreting ST-segment changes on an ECG immediately after return of spontaneous circulation is challenging. The QRS complex, ST segment and T waves are often affected by global ischaemia due to coronary hypoperfusion secondary to cardiac arrest. Adrenaline and metabolic disturbances can further influence repolarisation, producing pronounced ST–T abnormalities. Consequently, ST elevation on ECG immediately after return of spontaneous circulation has lower specificity for acute coronary syndrome than under normal circumstances (2). The ECG should always be repeated after 15–20 minutes if the clinical situation allows. Persistent ST elevation increases the likelihood of an acute coronary occlusion. Elevated troponin T levels are common after cardiac arrest and, in isolation, do not indicate a cardiac aetiology. Following the initial assessment, no clear cause of the cardiac arrest had been established.

The patient was admitted to the cardiac intensive care unit for ongoing treatment and monitoring with continuous 12-lead ECG. Sedation was maintained with an intravenous combination of fentanyl (2 µg/kg/hour) and propofol (3 µg/kg/hour), while noradrenaline (0.2 µg/kg/min) was administered to maintain adequate perfusion pressure. Targeted temperature management was continued for 72 hours.
During the first 24 hours of admission, the patient suffered a further cardiac arrest. A 12-lead ECG captured the cardiac rhythm immediately prior to the event (Figure 2). The patient was promptly defibrillated, restoring sinus rhythm.

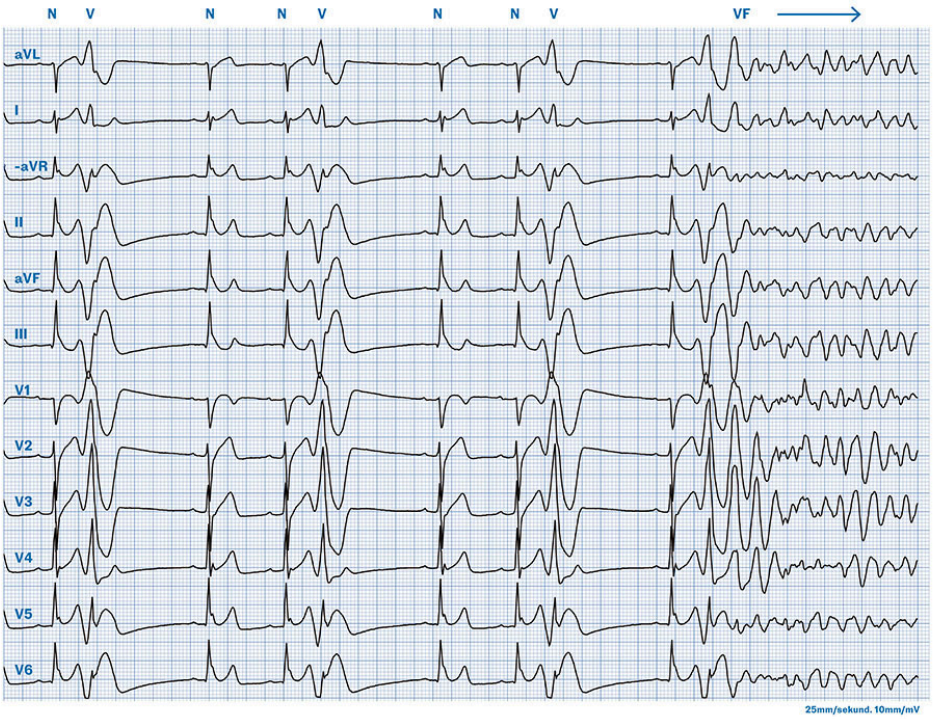

Figure 2 ECG immediately before cardiac arrest (Cabrera format, paper speed 25 mm/s). Sinus rhythm, rate 65 beats per minute. The sinus beats (labelled N) show a vertical axis and narrow QRS complexes. The PR interval is normal (174 ms), as is the corrected QT interval (414 ms), and there are no ischaemic ST-segment or T-wave changes. Frequent unifocal premature ventricular complexes (PVCs, labelled V) are present, occurring with a relatively short coupling interval (400 ms) and coinciding with the T wave (R-on-T phenomenon). Distinct J waves are visible in the inferior and lateral leads. Towards the end of the tracing, an R-on-T PVC triggers ventricular fibrillation (labelled VF).

Because the patient had experienced ventricular fibrillation and demonstrated a normal corrected QT interval during sinus rhythm, intravenous amiodarone therapy was initiated (1200 mg/24 hours). As frequent premature ventricular complexes had been noted before the cardiac arrest, a temporary right atrial pacemaker was inserted with the aim of increasing the heart rate above the patient's intrinsic heart rate (overdrive pacing). Overdrive pacing is particularly effective in preventing polymorphic ventricular tachycardia in the presence of a prolonged corrected QT interval, as cardiac arrest in such cases is often linked to rate- and pause-dependent QT variability. It may also be beneficial in ventricular arrhythmias triggered by ventricular extrasystoles occurring during relative bradycardia. Increasing the pacing rate can suppress premature ventricular complexes and prevent bradycardia, thereby serving as an effective strategy in selected patients. Following an increase in heart rate to 90 beats per minute and the initiation of amiodarone infusion, the patient remained free of arrhythmias.

An ECG gives the clinician a snapshot of the heart's electrical activity. Conditions predisposing to cardiac arrest may be permanent in nature (e.g. fibrosis, hypertrophy) or dynamic (e.g. ischaemia, electrolyte disturbances, neurohormonal changes). Comparing serial ECGs from the same patient is therefore often valuable. In Figure 3, the inferior and lateral leads from the admission ECG are compared with the ECG immediately before the in-hospital cardiac arrest. Figure 4 shows lead V4 prior to the arrest, demonstrating a finding that predicts cardiac arrest in patients with the diagnosis ultimately established in our patient.

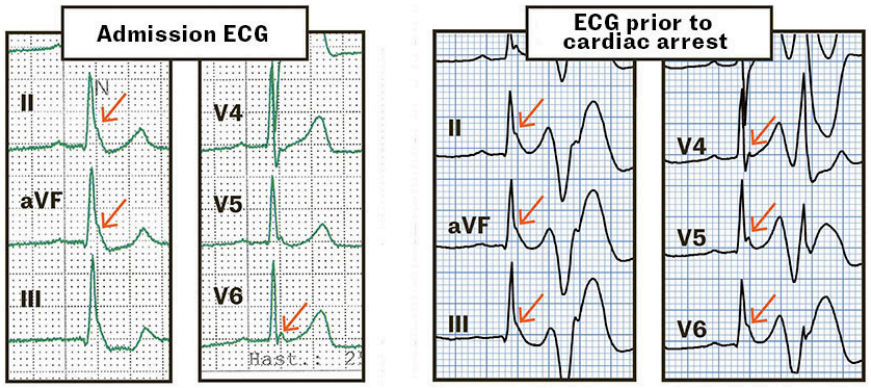

Figure 3 J waves indicated by arrows. The left panel shows the inferior and lateral leads from the admission ECG, while the right panel shows the same leads immediately prior to cardiac arrest. J waves are more prominent immediately before ventricular fibrillation, and the ECGs demonstrate clear dynamic changes, with J waves increasing in both amplitude and spatial distribution in the period preceding cardiac arrest (paper speed 25 mm/s).

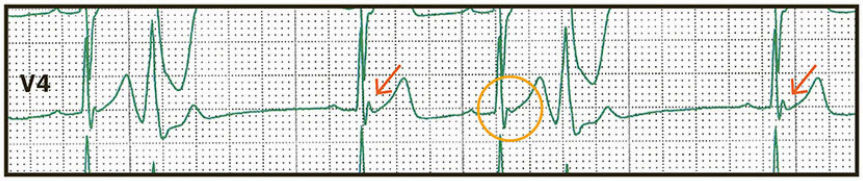

Figure 4 Rhythm strip from lead V4. QRS complexes preceded by a post-extrasystolic pause, and thus a prolonged RR interval, show more prominent J waves (arrows) compared with QRS complexes not preceded by RR interval prolongation (circle). This phenomenon is called post-extrasystolic J-wave augmentation (paper speed 25 mm/s).

Based on the ECG obtained during the second cardiac arrest and the exclusion of other potential causes, it was concluded that the patient's cardiac arrest was attributable to early repolarisation syndrome (ERS), a rare cause of arrhythmic death.
The patient sustained severe anoxic brain injury as a consequence of the pre-hospital cardiac arrest. He did not regain consciousness following sedation withdrawal and subsequently developed myoclonus. Following multidisciplinary evaluation and clinical and radiological prognostication, a decision was made on medical ethical grounds to withdraw treatment.

Discussion

Our patient experienced cardiac arrest secondary to ERS. Diagnosis requires the presence of an early repolarisation pattern on the ECG, together with arrhythmias in the form of either polymorphic ventricular tachycardia or ventricular fibrillation (3). Other potential causes of polymorphic ventricular tachycardia and ventricular fibrillation must be ruled out, and no structural heart disease should be present.

An early repolarisation pattern is characterised by J waves, which may be well-defined or manifest as fragmentation or distortion of the terminal portion of the QRS complex (Figure 5). The presence of an early repolarisation pattern does not necessarily require ST-segment elevation on the ECG, although this is often observed (4).

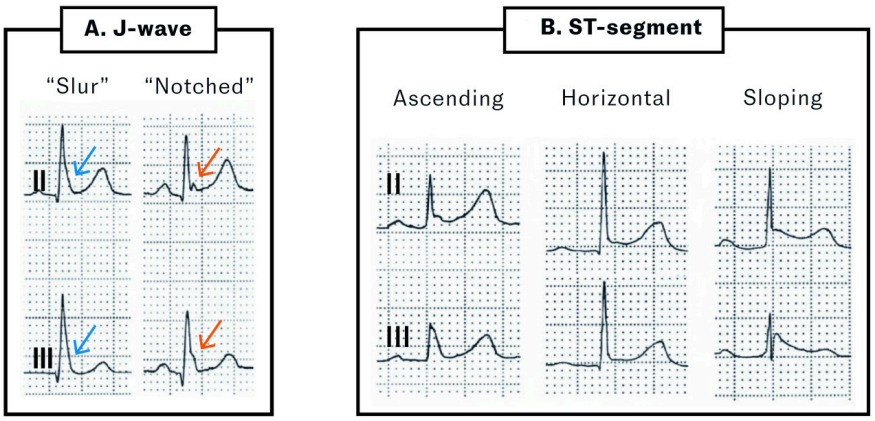

Figure 5 Different manifestations of early repolarisation. Modified from Antzelevitch et al. (3). a) The J wave may be well-defined (red arrow) or appear as a distortion of the terminal portion of the QRS complex, with the J wave 'buried' at the end of the complex (blue arrow). b) ST-segment elevation is common in early repolarisation patterns but is not an absolute criterion. The ST segment may be ascending, horizontal or descending.

Many patients exhibit some degree of ST elevation in the precordial leads (typically V2–V3), which is often incorrectly classified as early repolarisation. Isolated ST elevation without distinct J waves should not be interpreted as an early repolarisation pattern and has no established prognostic significance.

Early repolarisation pattern on ECG is common and frequently observed in healthy patients. Historically, early repolarisation was considered a benign finding. However, in two studies of patients with idiopathic ventricular fibrillation, a correlation was observed between this condition and early repolarisation on ECG in 31 % (5) and 60 % of patients (6), respectively. By comparison, early repolarisation pattern was observed in only 5 % and 3.3 % of the control groups.

The prevalence of early repolarisation varies with age, sex and ethnicity, and is particularly common in younger men and athletes. Because early repolarisation is common in the general population while idiopathic ventricular fibrillation is rare, the presence of an early repolarisation pattern on an ECG is generally considered an incidental finding with no clinical significance (4). The risk of idiopathic ventricular fibrillation in individuals under 45 years of age is approximately 3 per 100,000 person-years, increasing to 11 per 100,000 person-years in patients with J waves (7).

Our patient exhibited a typical clinical presentation, ECG findings and dynamic ECG changes consistent with ERS. ERS shares several features with Brugada syndrome. Both conditions are associated with J waves on the ECG and an increased risk of sudden cardiac death. In Brugada syndrome, J waves are confined to the right precordial leads (V1–V3) and can mimic ST-segment elevation. In ERS, J waves are primarily observed in the inferior leads. Together, Brugada syndrome and ERS are classified as J-wave syndromes (3).

J waves are caused by a voltage gradient between the epicardial and endocardial myocardium (4). These voltage differences are primarily influenced by regional variations in the density of potassium channels responsible for the current underlying early repolarisation. Mutations in the genes encoding these ion channels can alter the channels' function. Both Brugada syndrome and ERS are ion channelopathies, with causal mutations identified to varying extents. In addition to genetic mutations, other factors, such as medications, body temperature, electrolyte disturbances and neurohormonal fluctuations, can modulate ion channel function. The interaction of these factors explains the dynamic behaviour of J waves often observed in patients with Brugada syndrome and ERS.

J waves can be observed in a variety of conditions and were first described in 1920 in association with hypercalcaemia. In 1953, Osborn conducted animal studies demonstrating that J waves are a reproducible finding in severe hypothermia (<30 °C), with the amplitude of the J wave increasing progressively with the severity of hypothermia until the onset of ventricular fibrillation (8). J waves are sometimes eponymously referred to as Osborn waves, a term commonly used for hypothermia-induced J waves. Arrhythmia in the form of ventricular fibrillation is a common cause of death in patients with hypothermia (9).

ERS, Brugada syndrome and severe hypothermia are all conditions in which prominent or augmented J waves are associated with ventricular fibrillation. In patients with J-wave syndromes, cardiac arrest most commonly occurs at rest or during sleep, as in our patient. J waves typically become more pronounced immediately prior to cardiac arrest (6), and premature ventricular complexes with short coupling intervals are typically present in the period preceding ventricular fibrillation. Post-pause J-wave augmentation is strongly associated with ventricular fibrillation in patients with ERS (4). These findings were evident in our patient (Figures 3 and 4). In ERS, the amplitude and distribution of J waves, as well as accompanying horizontal or descending ST segments, are predictive of ventricular arrhythmia.

Sudden death in young individuals is dramatic. When such events occur, follow-up is often required, which is particularly challenging when a common finding could reflect serious disease and guidance for follow-up is limited. Our patient had several siblings, and the potential need to screen first-degree relatives of patients with ERS naturally became a topic of discussion. The literature on this subject is sparse, and no clear guidelines currently exist.

Several gene mutations are associated with ERS, but the relationship between specific genetic variants and the clinical risk of ventricular fibrillation remains uncertain. Consequently, routine testing for all such mutations is not performed in cases of arrhythmic death, and systematic genetic screening of first-degree relatives of patients with ERS is not recommended (10); it was therefore not undertaken in our case. Given that early repolarisation is common in the general population, its presence in first-degree relatives has limited diagnostic value in the absence of a clinical correlate.

In our patient, genome sequencing was performed using the NGS-Cardiac Arrhythmia gene panel, which did not reveal any pathogenic variants. Most genes associated with ERS are not included in this panel. The diagnosis must be based on ECG findings and the patient's arrhythmic history, after exclusion of other potential causes. Patients with a confirmed diagnosis have a Class IA indication for an implantable cardioverter-

defibrillator (ICD). As early repolarisation pattern is relatively common in the population and ventricular fibrillation is rare, an ICD is primarily indicated for secondary prevention.

Following discussions with the National Centre for Cardiac Arrhythmias at Oslo University Hospital, Rikshospitalet and the Cardiac Genetics Unit at Oslo University Hospital, we concluded that first-degree relatives of a patient with ERS who present with symptoms consistent with arrhythmia and concurrent early repolarisation on ECG should be referred for evaluation by a specialist in cardiac arrhythmias. Specialist evaluation typically involves a comprehensive assessment of arrhythmic risk, and an implantable loop recorder (ILR) may be considered as part of the follow-up (11).

ERS is a rare cause of cardiac arrest but should be considered in patients with structurally normal hearts in whom no other cause for the arrest has been identified and an early repolarisation pattern is observed on ECG. Diagnosis must be based on ECG findings and the patient's arrhythmic history, after exclusion of other potential causes. It should be emphasised that an early repolarisation pattern on ECG is common and, as an isolated finding, does not warrant further investigation. Isolated ST elevation in V2–V3 without J waves, known as 'high take-off', does not constitute an early repolarisation pattern and should not be regarded as a marker of arrhythmic risk.

It is important to remember that an ECG only gives a snapshot of the heart's electrical activity. This case illustrates the value of repeat ECGs. Even in an era of increasingly advanced diagnostic techniques, the benefit of simple, inexpensive, non-invasive investigations that can yield significant information should not be overlooked.

The patient's next of kin consented to publication of the article.

The article has been peer-reviewed.

REFERENCES

1. Tjelmeland IBM, Kramer-Johansen J, Andersson LJ et al. Norsk hjertestansregister. Årsrapport for 2023. Nasjonale medisinske kvalitetsregistre. https://www.kvalitetsregistre.no/49ece0/siteassets/dokumenter/arsrapporter/hjertestansregisteret/arsrapport-2023-norsk-hjertestansregister.pdf Accessed 22.10.2025.

2. Stær-Jensen H, Nakstad ER, Fossum E et al. Post-Resuscitation ECG for Selection of Patients for Immediate Coronary Angiography in Out-of-Hospital Cardiac Arrest. Circ Cardiovasc Interv 2015; 8. doi: 10.1161/CIRCINTERVENTIONS.115.002784. [PubMed][CrossRef]

3. Antzelevitch C, Yan GX, Ackerman MJ et al. J-Wave syndromes expert consensus conference report: Emerging concepts and gaps in knowledge. J Arrhythm 2016; 32: 315–39. [PubMed][CrossRef]

4. Watanabe A, Morita H. Risk Stratification of the J Wave Syndrome. In: Shimizu W, editor. Early Repolarization Syndrome: Etiology and Therapeutics. Singapore: Springer Singapore, 2018: 55–69.

5. Haïssaguerre M, Derval N, Sacher F et al. Sudden cardiac arrest associated with early repolarization. N Engl J Med 2008; 358: 2016–23. [PubMed][CrossRef]

6. Nam GB, Kim YH, Antzelevitch C. Augmentation of J waves and electrical storms in patients with early repolarization. N Engl J Med 2008; 358: 2078–9. [PubMed] [CrossRef]

7. Aagaard P, Sydow J, Börjesson M et al. Early repolarization in ECG. Definition, prevalence and prognostic significance. Lakartidningen 2015; 112: DLUT. [PubMed]

8. Osborn JJ. Experimental hypothermia; respiratory and blood pH changes in relation to cardiac function. Am J Physiol 1953; 175: 389–98. [PubMed][CrossRef]

9. Dietrichs ES, Tveita T, Smith G. Hypothermia and cardiac electrophysiology: a systematic review of clinical and experimental data. Cardiovasc Res 2019; 115: 501–9. [PubMed][CrossRef]

10. Wilde AAM, Semsarian C, Márquez MF et al. European Heart Rhythm Association (EHRA)/Heart Rhythm Society (HRS)/Asia Pacific Heart Rhythm Society (APHRS)/Latin American Heart Rhythm Society (LAHRS) Expert Consensus Statement on the state of genetic testing for cardiac diseases. J Arrhythm 2022; 38: 491–553. [PubMed][CrossRef]

11. Kamakura T, Gourraud JB, Clementy N et al. Outcome of patients with early repolarization pattern and syncope. Heart Rhythm 2022; 19: 1306–14. [PubMed] [CrossRef]

Publisert: 17. December 2025. Tidsskr Nor Legeforen. DOI: 10.4045/tidsskr.25.0261
Received 9.4.2025, first revision submitted 9.4.2025, accepted 22.10.2025.