81-year-old African-American, man with history of Alzheimer's disease, malaise, bradycardia, hypotension and death in two weeks

Anciano de 81 anõs, afro-americano, con historia de Alzheimer, mal estado general, bradicardia, hipotensión y muerte en dos semanas

Case of Dr Jason E. Roediger

Final comments by **Andrés Ricardo Pérez-Riera M.D.Ph.D.**
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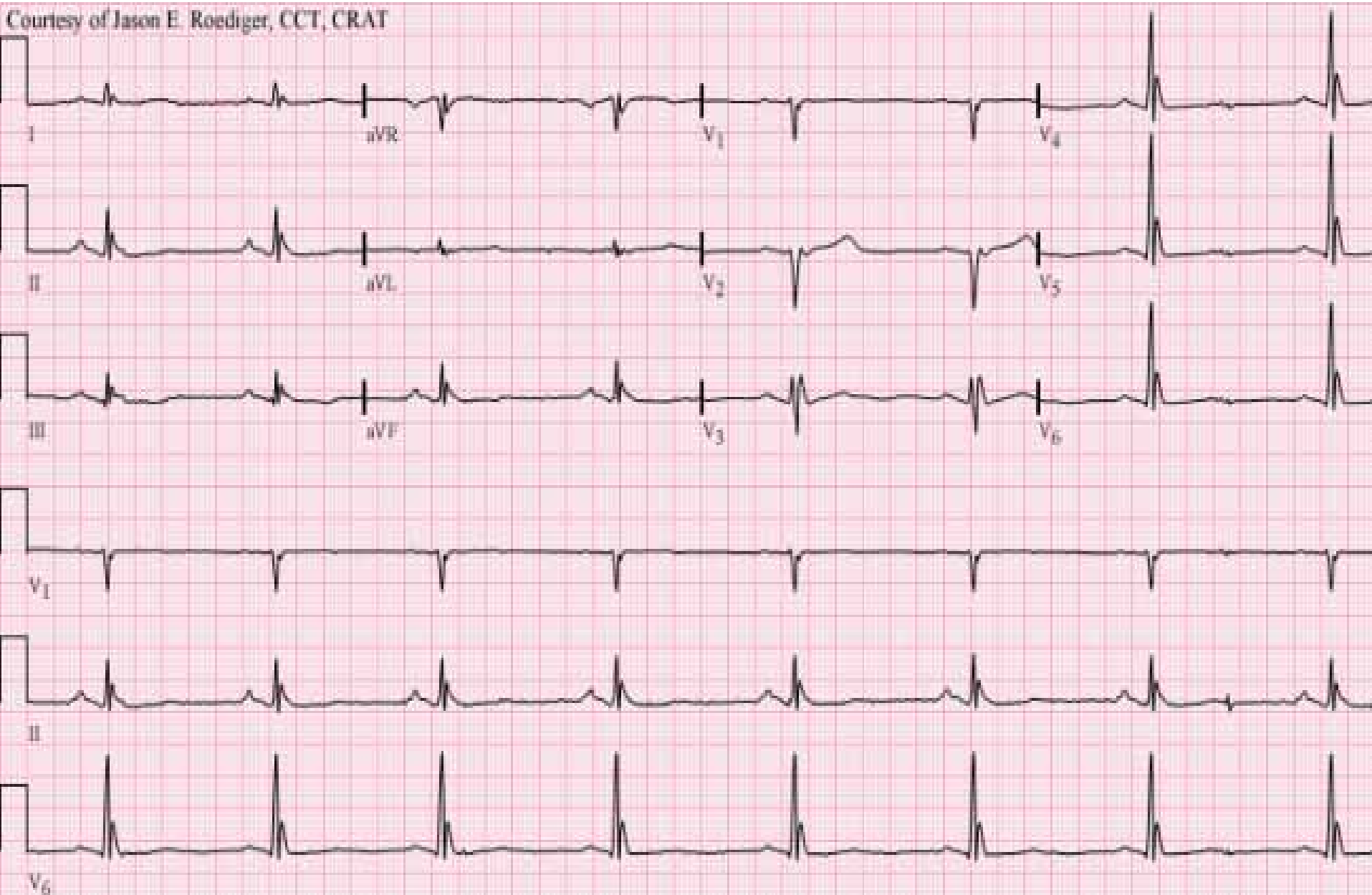
Patient's clinical data: 81-year-old Afro-American man with history of Alzheimer's disease (dementia). The patient's son transported his father via POV to the emergency department's triage room.
Patient had essentially been "failing to thrive" and his presenting blood pressure was hypotensive at 80/62. Subsequently, he was admitted to the ICU, intubated, and placed on a ventilator.
He ended up dying about two weeks later.
What is the probable clinical disorder and ECG diagnose?
Datos clínicos del paciente: 81-años de edad, masculino, negro con antecedentes de enfermedad de Alzheimer (demencia).
El hijo lo havia transportado en seu propio veículo para sala de triaje de urgencias.
El paciente esencialmente no estava llendo bien y su presión arterial muy baja era de 80/62 mmHg.
Posteriormente fue ingresado en la UCI, intubado, y conectado a un respirador.
Acabó muriendo alrededor de 2 semanas después
Cual es el diagnóstico clínico y electrocardiográfico probable?


Courtesy of Jason E. Roediger, CCT, CRAT
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V6

Colleagues opinions

Queridos amigos del forum con respecto al paciente anciano con Alzheimer de 81 años, con hipotensión y bradicardia sinusal de 30lpm

Es muy probable que sea un shock bradicárdico por enfermedad del nódulo sinusal "sick sinus syndrome" asociado a drogas bradicardizantes

La única forma de estabilizar este pacientes seria con drogas estimulantes de los beta receptores en alta dosis, hasta aumentar la frecuencia cardiaca

El ECG no está relacionado a la muerte del paciente salvando la bradicardia sinusal severa

El ECG muestra una onda P con muesca y ancha sugeriendo fibrosis severa de la auricula izquierda

En el QRS se observan dos vectores bien diferenciados:

1. En los primeros 40ms la coducción es rápida y alta
2. El segundo vector (de los 50 a 70 ms) es muy lento. Esta velocidad lenta sugiere fibrosis ubicada entre la punta y la base cardiaca prodiciendo esta imagen tan atipica del QRS

Un fraternal abrazo

Samuel Sclarovsky, Israel

English

Dear friends of the forum regarding elderly Alzheimer patient 81yo of age, with hypotension and sinus bradycardia (30lpm) .It is very likely to be a bradycardic shockconsequence of sick sinus syndrome associated with bradycardic drugs effect. The only way to stabilize the serious patients status is with β adrenergic stimulant drugs in high doses, with the objetive of to increase heart rate.

The ECG is not related to the patient's death saving severe sinus bradycardia

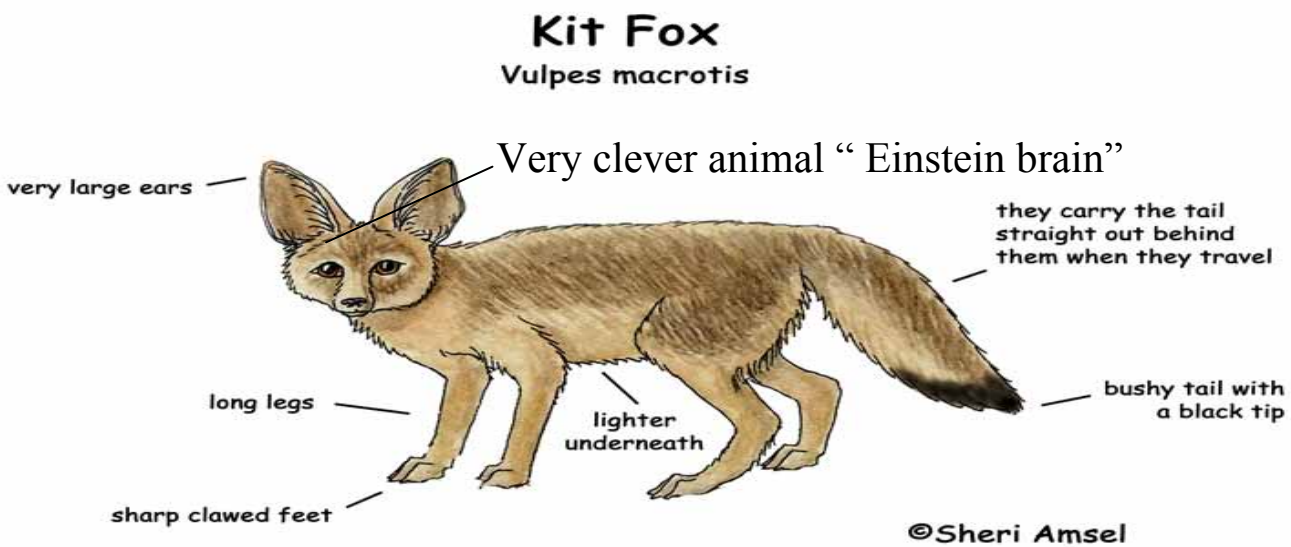
The ECG shows a P wave, wide and notched suggesting severe fibrosis of the left atrium

In the QRS shows we observe two distinct vectors: During the first 40ms the conduction is fast and high

The second vector (from 50 to 70 ms) is very slow. This suggests slow fibrosis located between the apex and the cardiac base producing this atypical QRS

Lo que llama la atención es: Bradicardia sinusal, bloqueo interauricular parcial(onda P > 120ms)onda J (Osborn) y QT prolongado
Etiología probable: accidente cerebrovascular hemorrágico con hipotermia .
El diagnóstico diferencial se plantea con intoxicación medicamentosa.
Raimundo Barbosa Barros M.D. Fortaleza – Brasil

What flame the attention is: Sinus bradycardia, partial interatrial block, J wave (Osborn) and prolonged QT interval Likely etiology: ischemic stroke with hypothermia.
The differential diagnosis it ponders with drug intoxication.
Raimundo **Barbosa Barros M.D.** Fortaleza – Brazil Nickname: " The Fox"
Chief of the Coronary Center of the Hospital de Messejana Dr. Carlos Alberto Studart Gomes. Fortaleza - Brazil.



Very clever animal " Einstein brain"

Estimados colegas
El analisis del ECG me sugiere:
Bradicardia sinusal a 50 lpm (no veo el trazado a 30, pero igualmente, está bradicárdico) No mata a nadie y no se que quiere decir Samuel con eso de shock bradicárdico. Este término no esta en mis libros, pero entiendo que debe referirse a bajo flujo por bradicardia, lo cual en alguien con buen ritmo sinusal es muy difícil que suceda).
Creo que ya se señaló que hay marcada Onda de Osborn lo que puede deberse a: Hemorragia intracraneana (y esto ser la causa de muerte) o b. Hipotermia (clinica o inducida para el tratamiento del ACV)
Se observa un bloqueo intraauricular avanzado con onda P> 140 ms y componente positivo-negativo en cara inferior (ver Consensus recientemente publicado en J Electrocardiol, con el Prof. Bayes como primer autor).
Bueno eso. Creo haber leido una opinión semejante de otro de los foristas, pido disculpas si el caso ya fue resuelto. Pero me encantaria conocer la anatomia patologica del caso.

Un abrazo
AB

Final comments

By Andrés Ricardo **Pérez-Riera M.D. Ph.D**.

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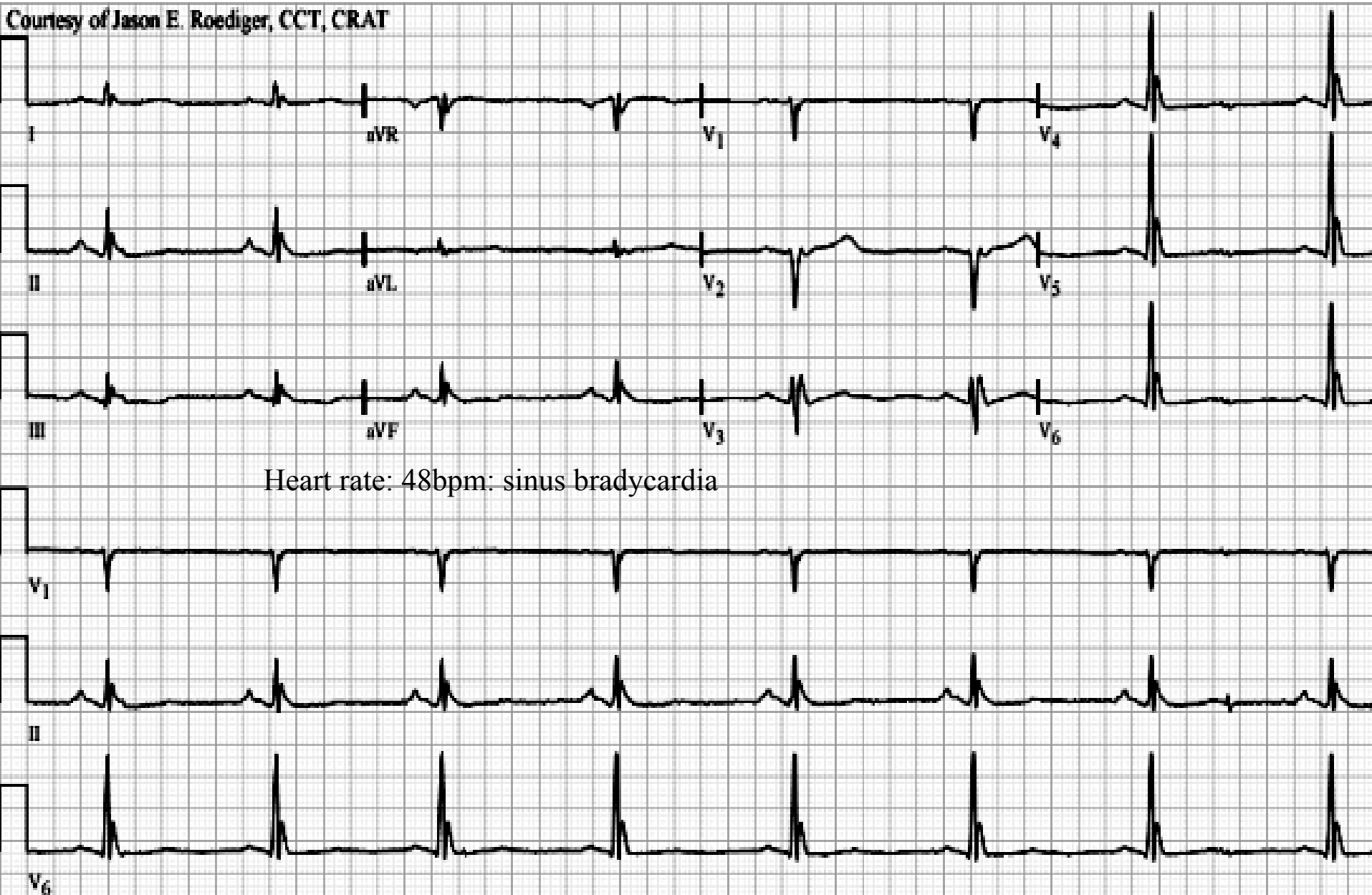
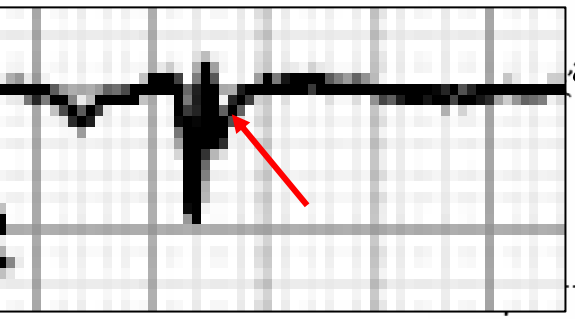
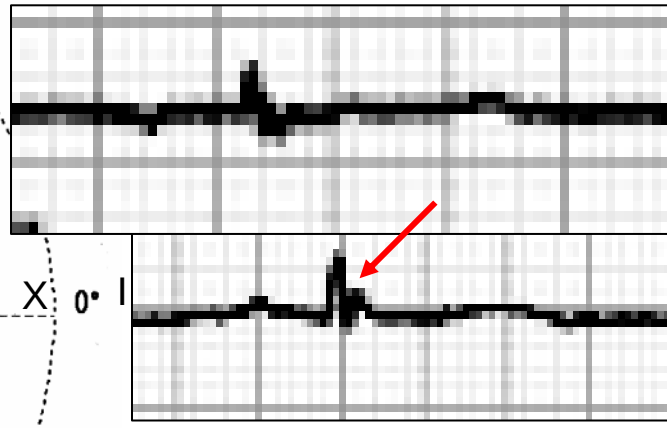


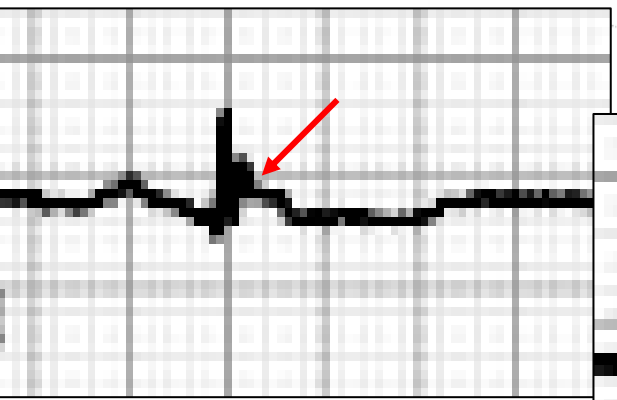
Heart rate: 48bpm: sinus bradycardia

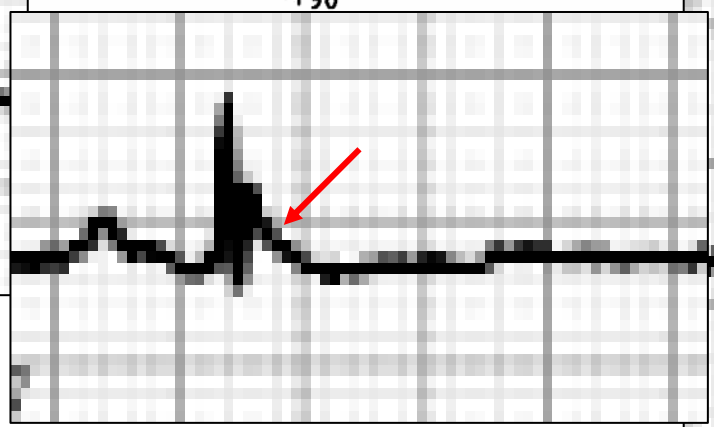
The Osborn wave (J wave) is a positive deflection at the J point (negative in aVR and V_1-V_2)

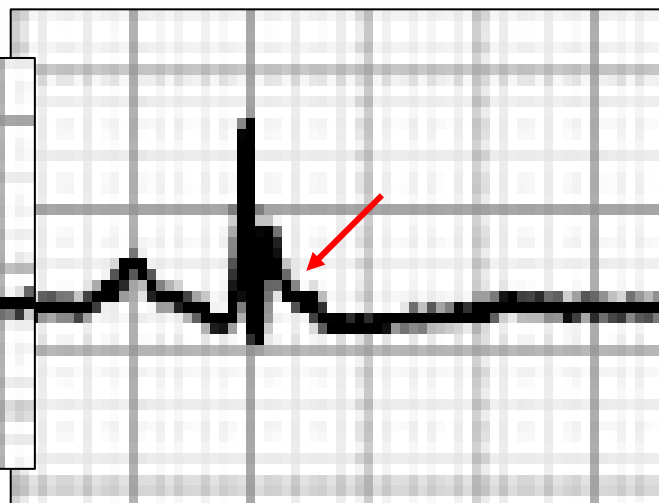
Frontal Plane leads





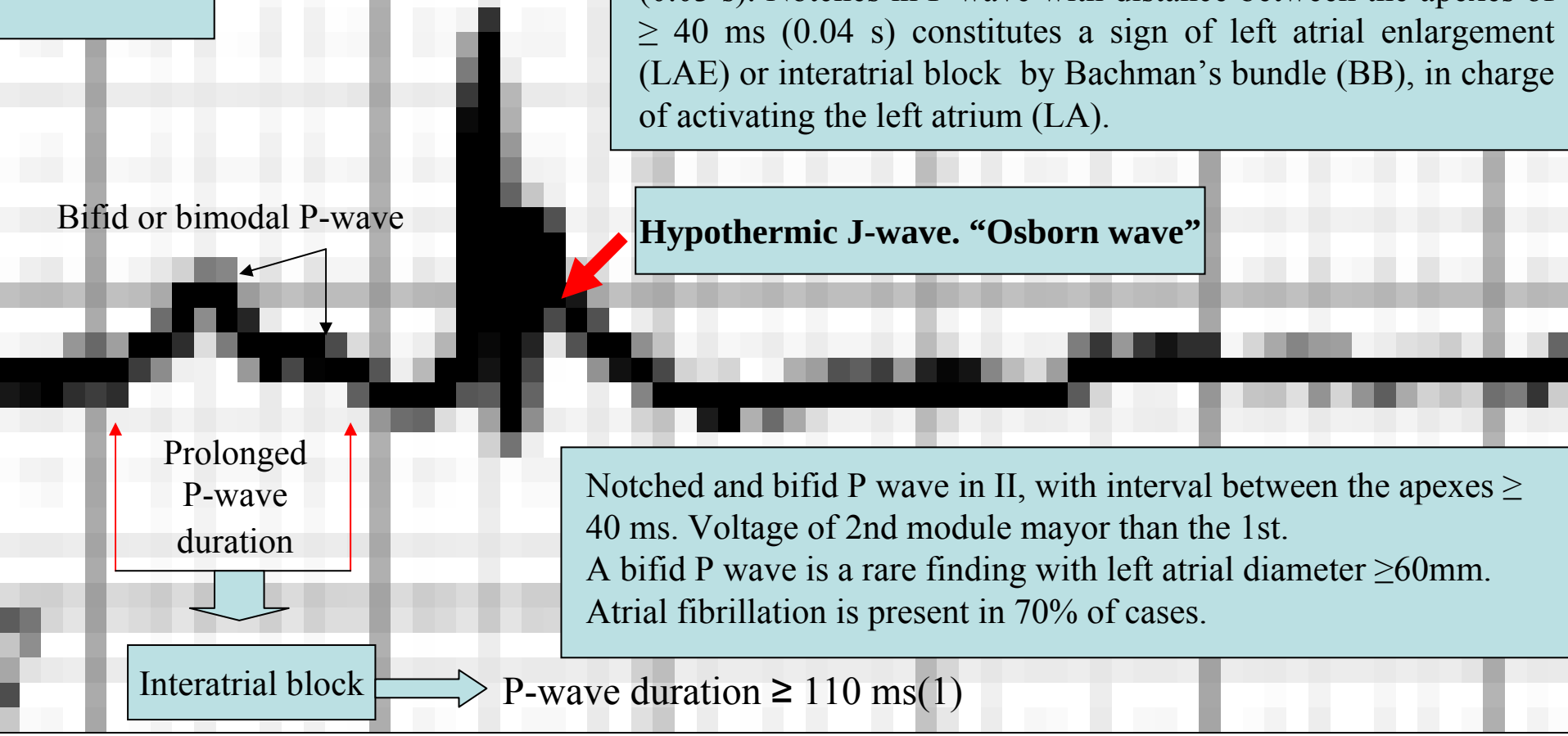






II

The normal shape of P-wave is rounded and monophasic, and there may be small notches (more frequent in V_3 and V_4) and the distance between these notches should not exceed 30 ms (0.03 s). Notches in P wave with distance between the apexes of ≥ 40 ms (0.04 s) constitutes a sign of left atrial enlargement (LAE) or interatrial block by Bachman's bundle (BB), in charge of activating the left atrium (LA).



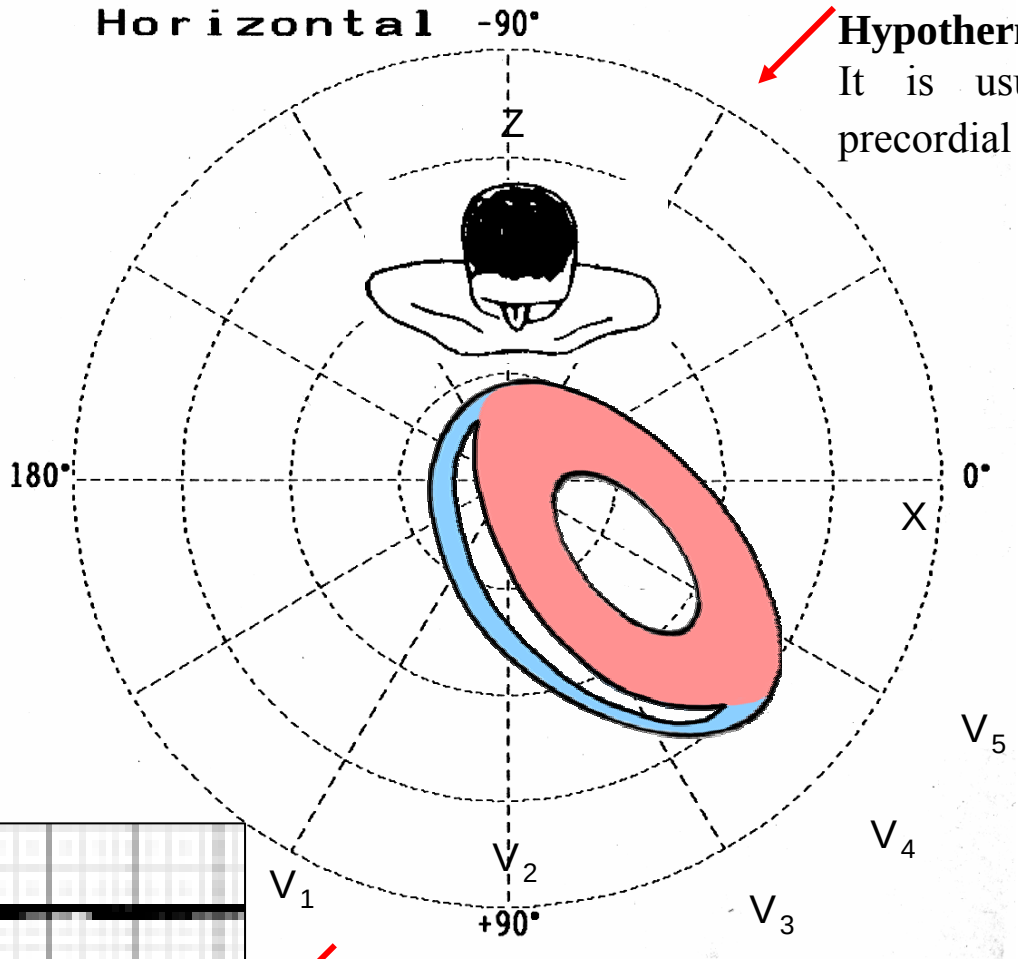
Notched and bifid P wave in II, with interval between the apexes ≥ 40 ms. Voltage of 2nd module mayor than the 1st.
A bifid P wave is a rare finding with left atrial diameter ≥60mm.
Atrial fibrillation is present in 70% of cases.

Interatrial block → P-wave duration ≥ 110 ms(1)

The presence of interatrial blocks may be seen in the absence of atrial enlargement but often are present in case of LAE. The first degree interatrial blocks are very common, and their relation with AF and an increased risk for global and cardiovascular mortality has been demonstrated

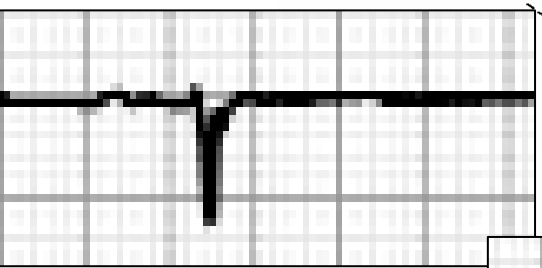
The third degree interatrial blocks is less frequent but it is a strong markers of LAE and paroxysmal supraventricular tachyarrhythmias. Their presence is considered a true arrhythmologenic syndrome.(2)

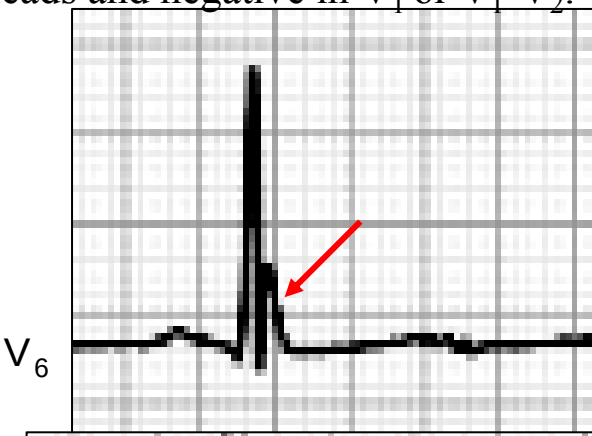
Horizontal Plane leads

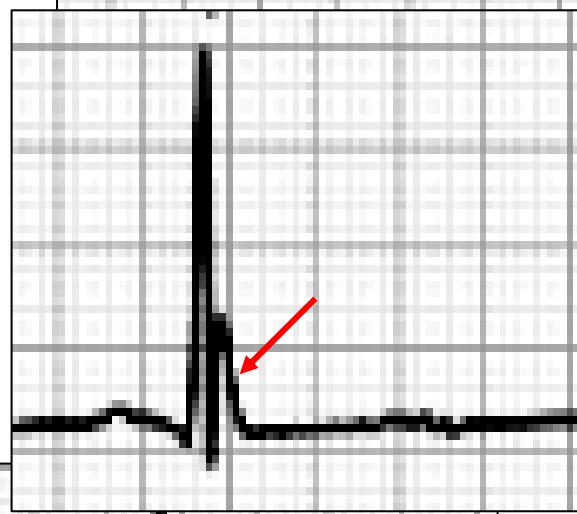


Hypothermic J-wave. "Osborn wave"

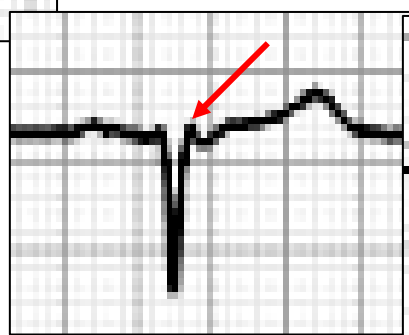
It is usually most prominent in the precordial leads and negative in V_1 or V_1-V_2.

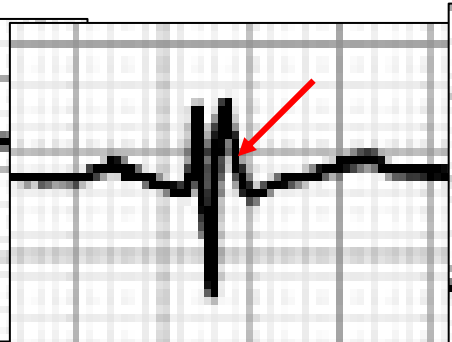


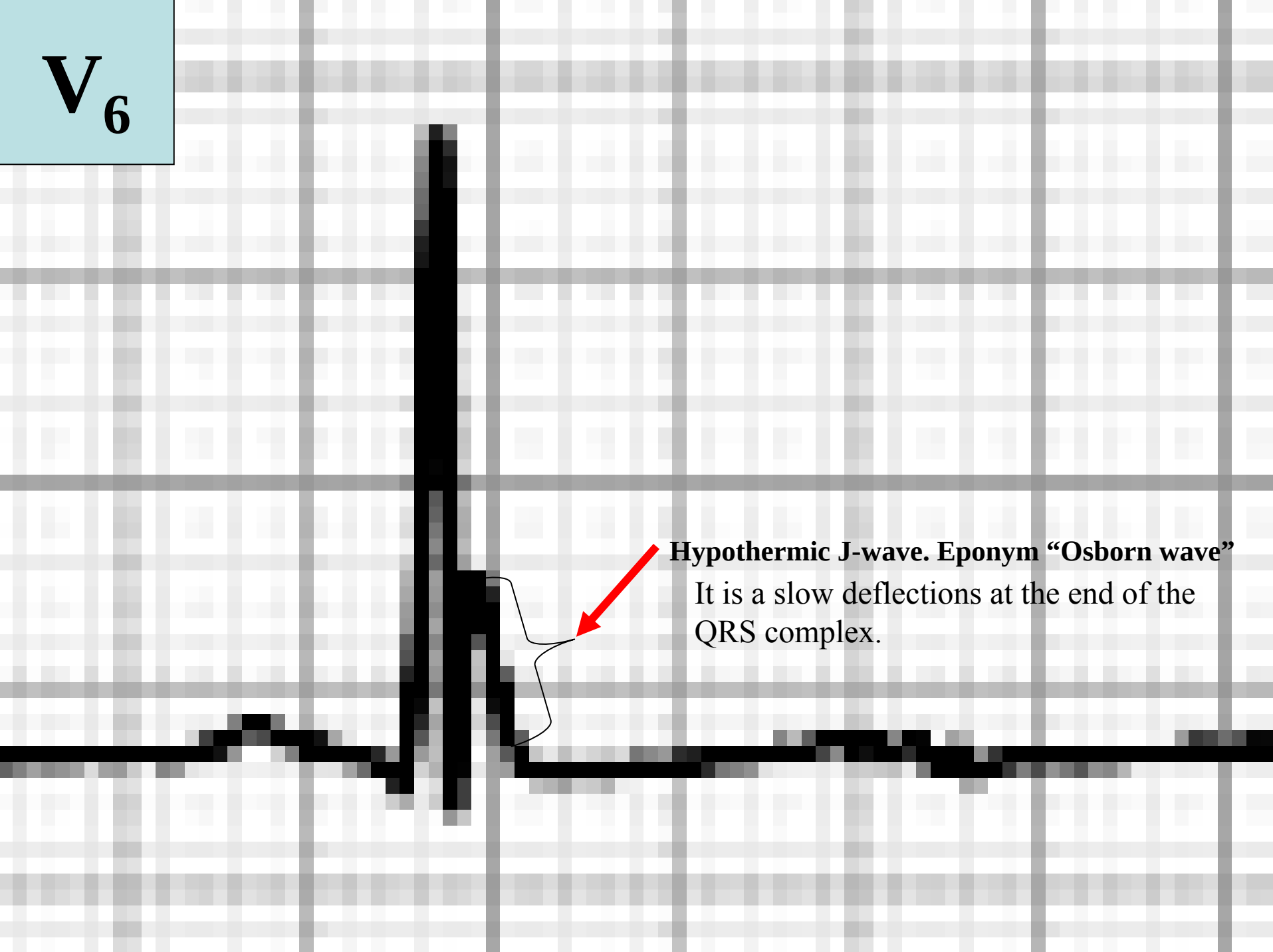




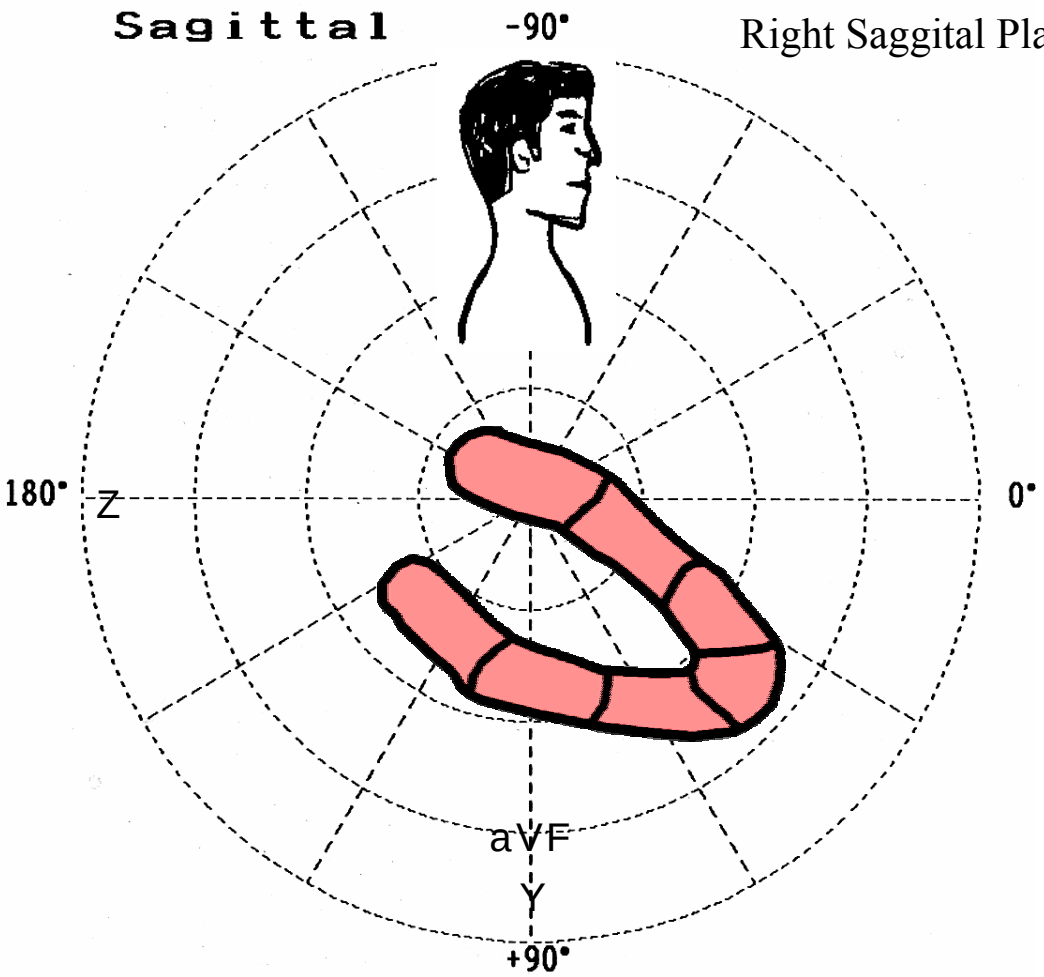
Hypothermic J-wave. Osborn wave

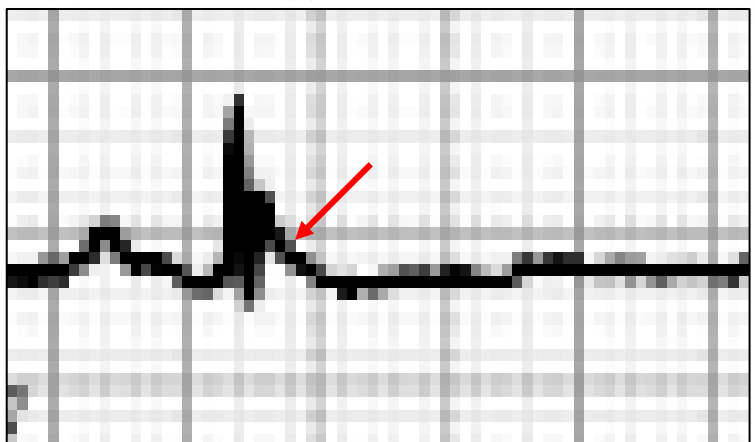


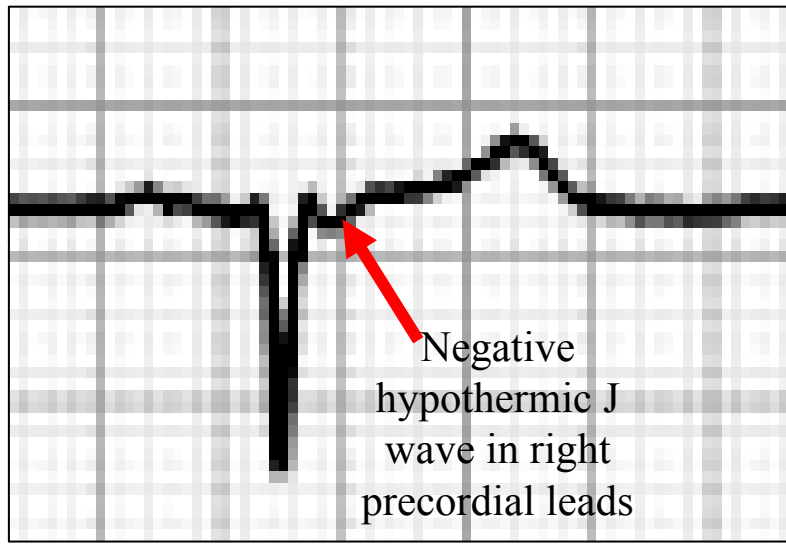



V6
Hypothermic J-wave. Eponym “Osborn wave”
It is a slow deflections at the end of the QRS complex.

Right Saggital Plane V2 and aVF leads



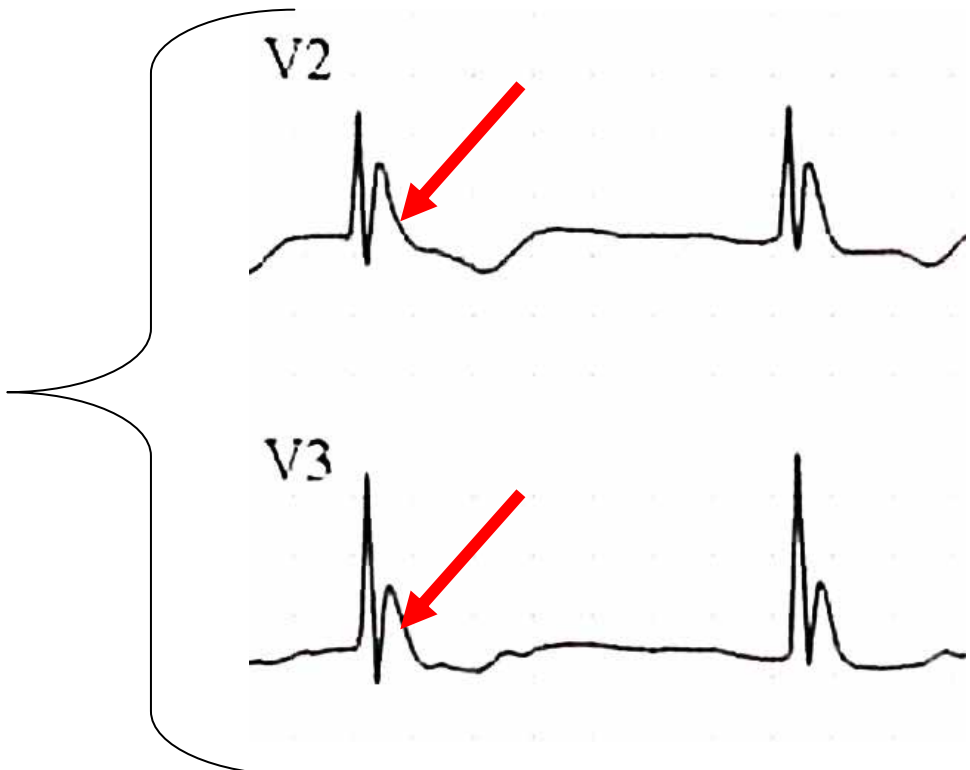


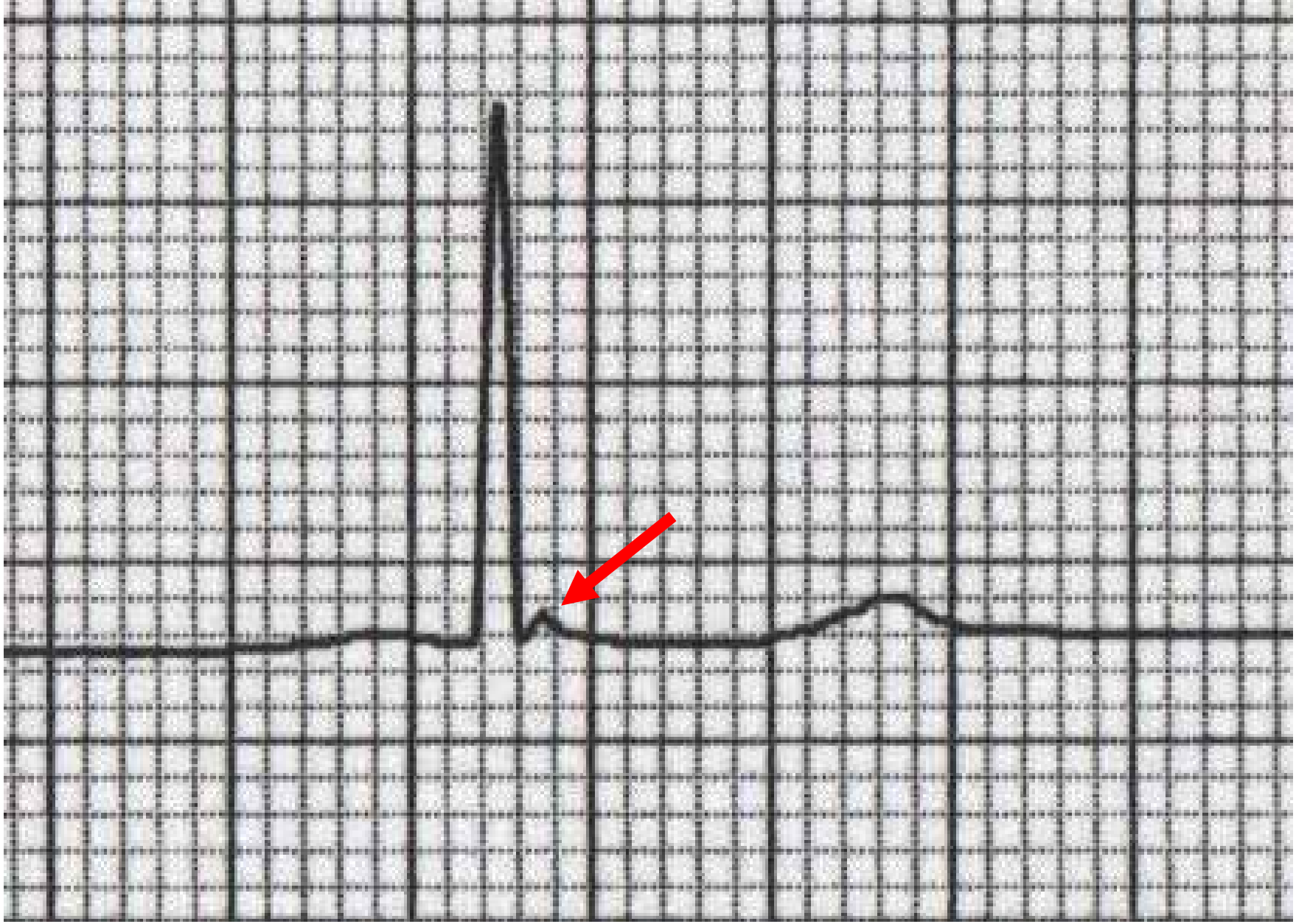


The Osborn wave (J wave) is a positive deflection at the J point (negative in aVR and V_1 or V_1-V_2 such as the present case)

The Osborn wave (J wave) is a positive deflection at the J point (negative in aVR and V_1 or V_1-V_2)
It is usually most prominent in the precordial leads
Characteristically seen in hypothermia (typically T<30C), but they are not pathognomonic.
In hypothermia is observed sinus bradycardia, prolonged QTc-interval, ST segment elevation inferior an left precordial leads, Osborn waves (slow deflections at the end of the QRS complex.
J waves defined as an upward deflection, and slur as a conduction delay on the QRS downstroke.(3)

Typical **Hypothermic** J Waves
The height of the J wave is roughly proportional to the degree of hypothermia:

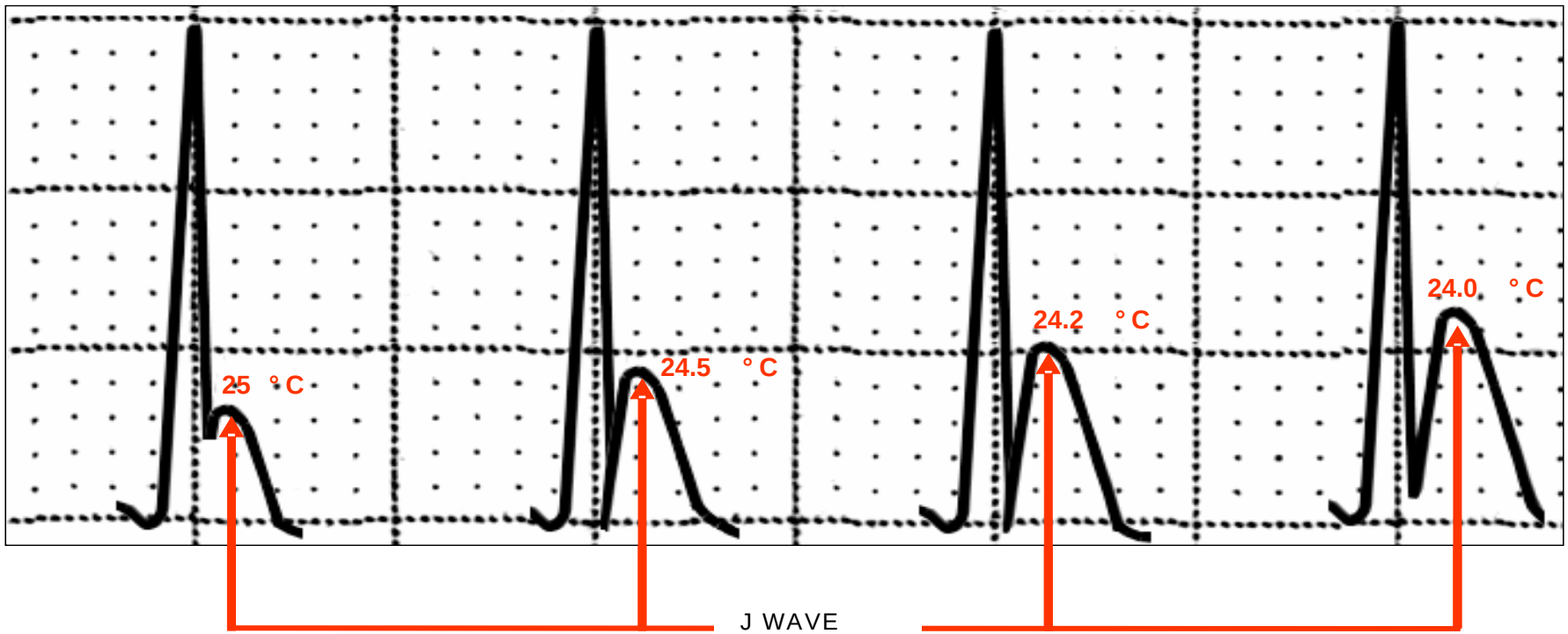




Subtle J waves in mild hypothermia (body temperature 32.5°C)

Sutis J ondas em hipotermia moderada (temperatura corporal 32.5°C)

Inverse and significant correlation between J wave voltage (mm) and central temperature in hypothermia.



J wave profile of hypothermia.

Non-hypothermic J wave or unrelated to hypothermia

J waves may be seen in a number of other conditions without hypothermia:

I) Normal variant(4) With high-prevalence subpopulations such as athletes, children, and adolescents.

II) Hypercalcaemia(5)

III) Medications

IV) Neurological insults such as:

Intracranial hypertension
Severe head injury
Subarachnoid haemorrhage secondary to aneurysm rupture

V) Brugada syndrome

VI) Le syndrome d'Haïssaguerre (idiopathic ventricular fibrillation)(6) Pause-dependent augmentation of J waves was confirmed in about one-half of the patients with idiopathic VF after sudden R-R prolongation. Such dynamicity of J waves was specific to idiopathic VF and may be used for risk stratification.

VII) Vasospastic angina(7)

VIII) Cardiopulmonary arrest from over sedation(7)

IX) Early recovery phase after an acute MI(8):The presence of J-waves was associated with ventricular arrhythmias, including ventricular fibrillation. The J-wave amplitude increased in the conducted atrial premature beats, mechanistically suggesting a phase 3 block.

X) As a hyperacute sign of Takotsubo syndrome.(9)

XI) When present a KCNJ8 mutation: member 8 (KCNJ8)-S422L missense mutation in patients with J-wave syndromes (10)

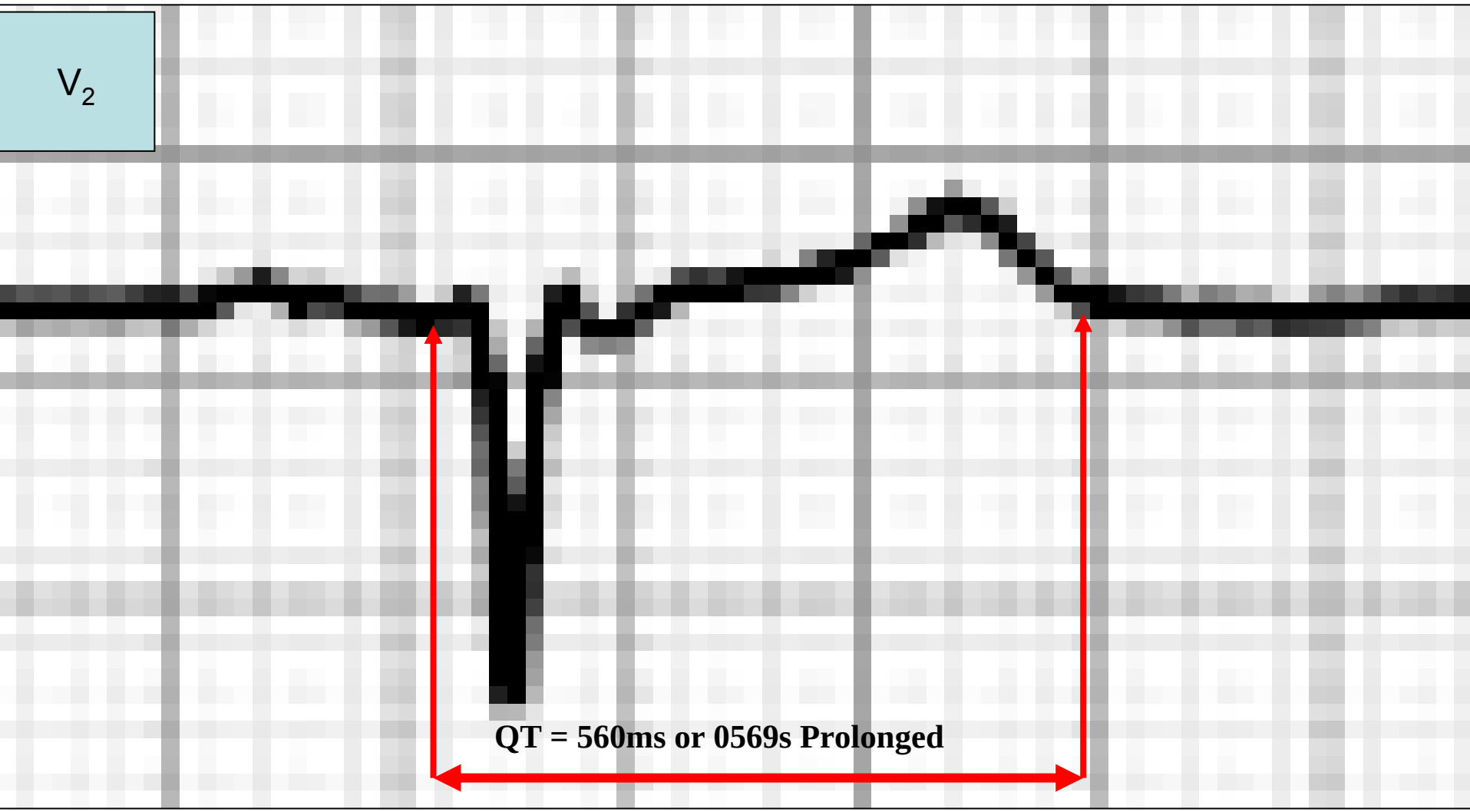
XII) In Brugada phenocopyes(12)

XIII) In Short QT syndrome variant 6 (SQTS6).(13)

XIV) In concealed forms of arrhythmogenic right ventricular cardiomyopathy/ dysplasia (14)

Heart rate: 48bpm: sinus bradycardia

QT for men (seconds) HR/bests/min 48 RR (Seconds) = 1.23; QT Mean Value: 0.414s Lower Limit: 0.37s Upper Limit: 0.458s or 450ms.(11)



Definition and Causes of hypothermia

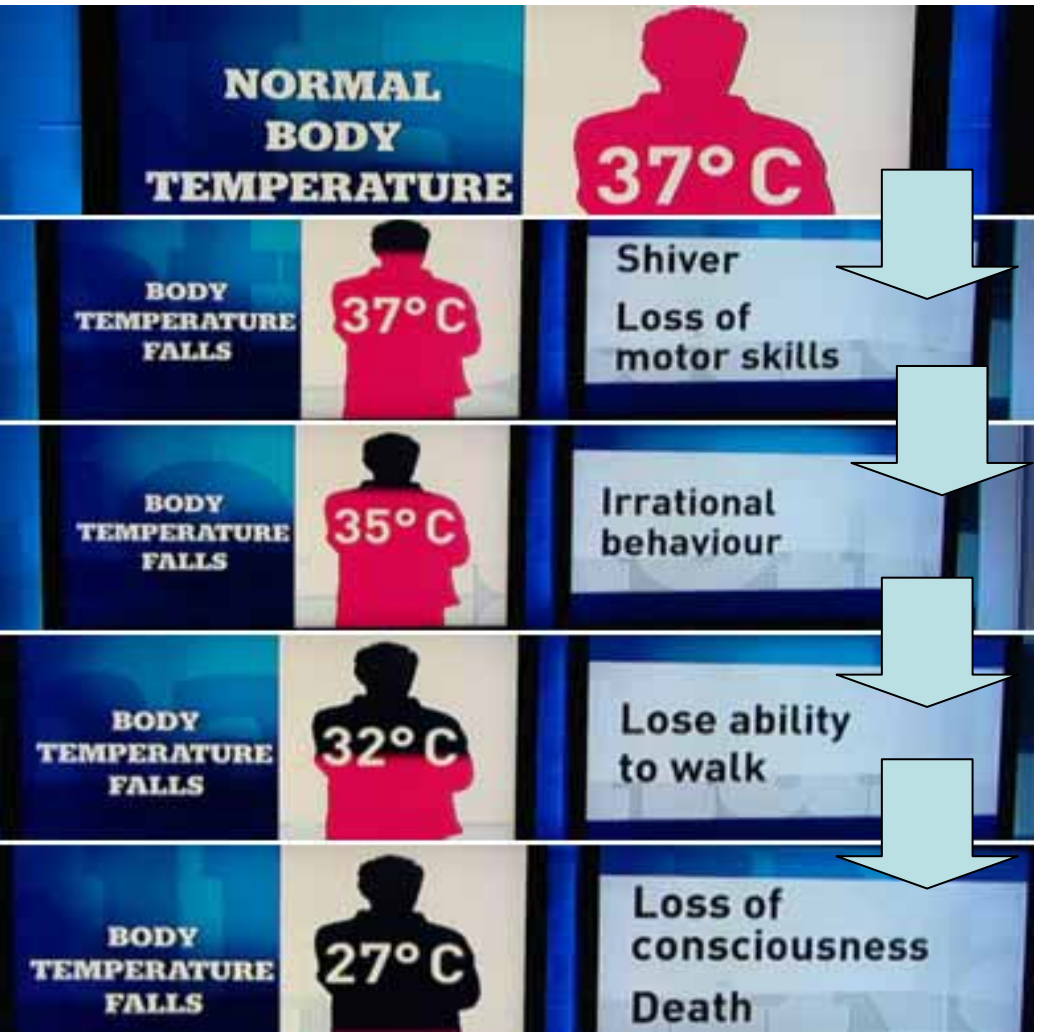
CONCEPT: hypothermia is defined as the condition where central temperature (rectal, esophageal or tympanic) is below 35ºC. Hypothermia may be accidental, metabolic, or therapeutic.

Accidental hypothermia is more frequent in countries with cold weather, during winter season. Hypothermal state is characterized by drop in basal metabolism, decrease in O^2 consumption and greater production of CO^2**,(15)**

During hypothermia, gradual decrease of heart rate is observed and systolic volume, with progressive drop of blood pressure later, which becomes significant when central temperature values close to 23ºC are reached.(16)

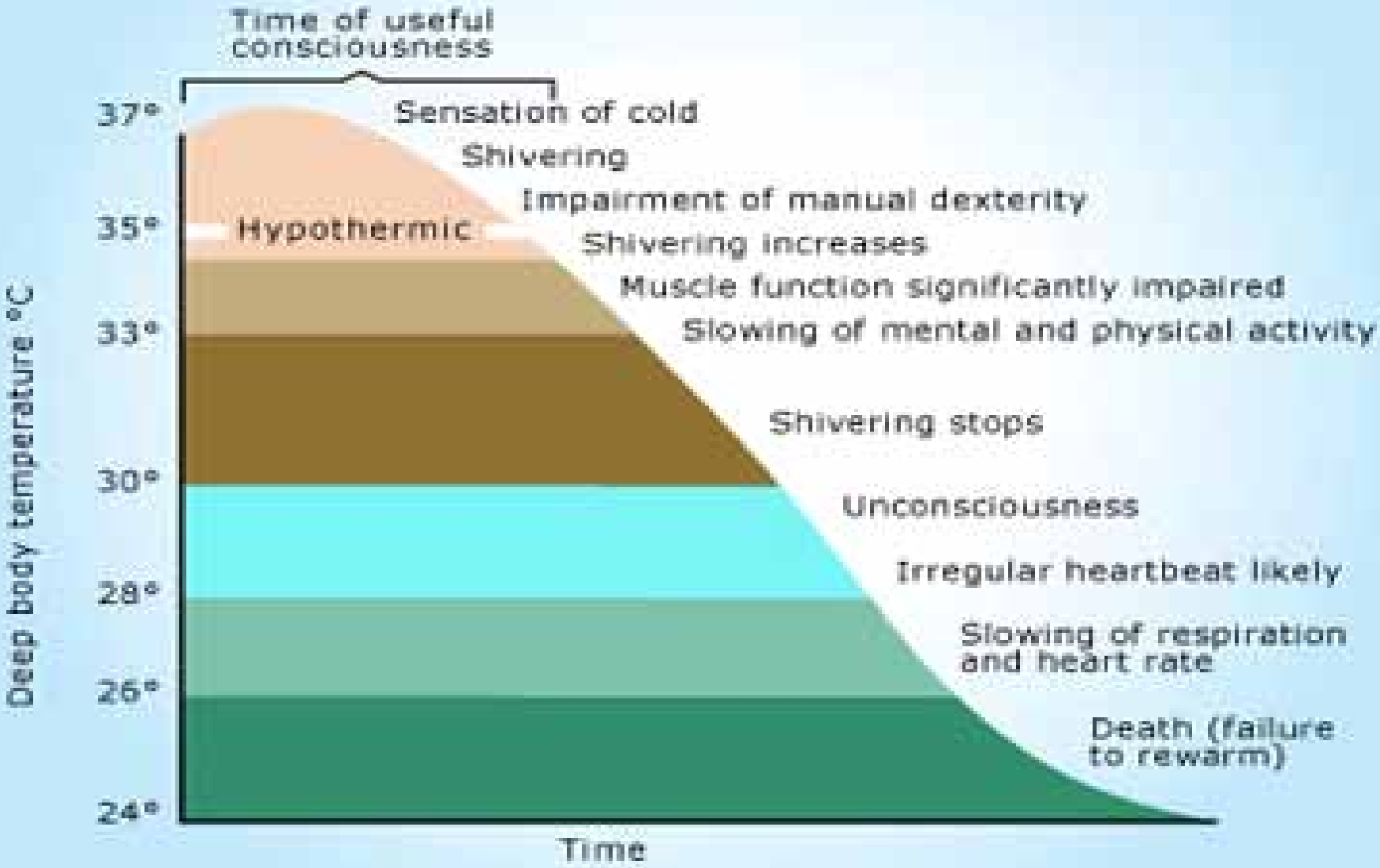
1. Trauma victims: mainly majors traumas (17)
2. Therapeutic hypothermia(18)
3. Accidental(19)
4. Severe cases of anorexia nervosa(20) clinical findings suggesting hypothyroidism, e.g., cold intolerance, constipation, bradycardia, hypothermia and hypercholesterolemia in association with decreased serum total T3 and T4 Chromic medical conditions such as hypothyrodism
5. Sepsis(21)
6. Homelessness(22)
7. Hydrogen sulfide exposure: It is a toxic gas produced as a by-product of organic waste and many industrial processes.(23)
8. Substance abuse
9. Exposure to cold environments

7. Alcohol consumption: It increases the risk of hypothermia via its action as a vasodilator. It increases blood flow to the body's skin and extremities, making a person *feel* warm, while increasing heat loss. Between 33 and 73% of cases of hypothermia are complicated by alcohol.
8. Diving in cold water.
9. Use of gas mixtures containing helium at extreme depths, the use of argon inflation for dry suits, or hot water suits become a necessity for diving deep in colder waters.



From F. Golden and M. Tipton, 2002, Essentials of sea survival (Champaign, IL: Human Kinetics), 102. Originally adapted from F.S. Golden, 1973, ?Recognition and treatment of immersion hypothermia,? Proceedings of the Royal Society of Medicine 66: 1058-1061.

Effects of fall in body temperature



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Factors that increase the risk of hypothermia

Age: The very young and very old may be less able to generate heat. The elderly with underlying medical conditions such as hypothyroidism or Parkinson's disease that limit the ability of the body to regulate temperature are less able to generate heat. Infants don't generate heat as efficiently, and with their relatively large head size compared to the body, they are at risk for increased heat loss by radiation.

Mental status: Impaired judgment and mental function can lead to cold exposure. Patients with Alzheimer's disease are prone to wander and become exposed to the elements.

Substance abuse: Alcohol and drug abuse increase the risk of hypothermia in two ways. First, impaired judgment can lead to cold exposure. Additionally, alcohol and similar drugs can dilate blood vessels near the skin and decrease the efficiency of the shivering mechanism, both of which decrease the body's ability to compensate for cold exposure.

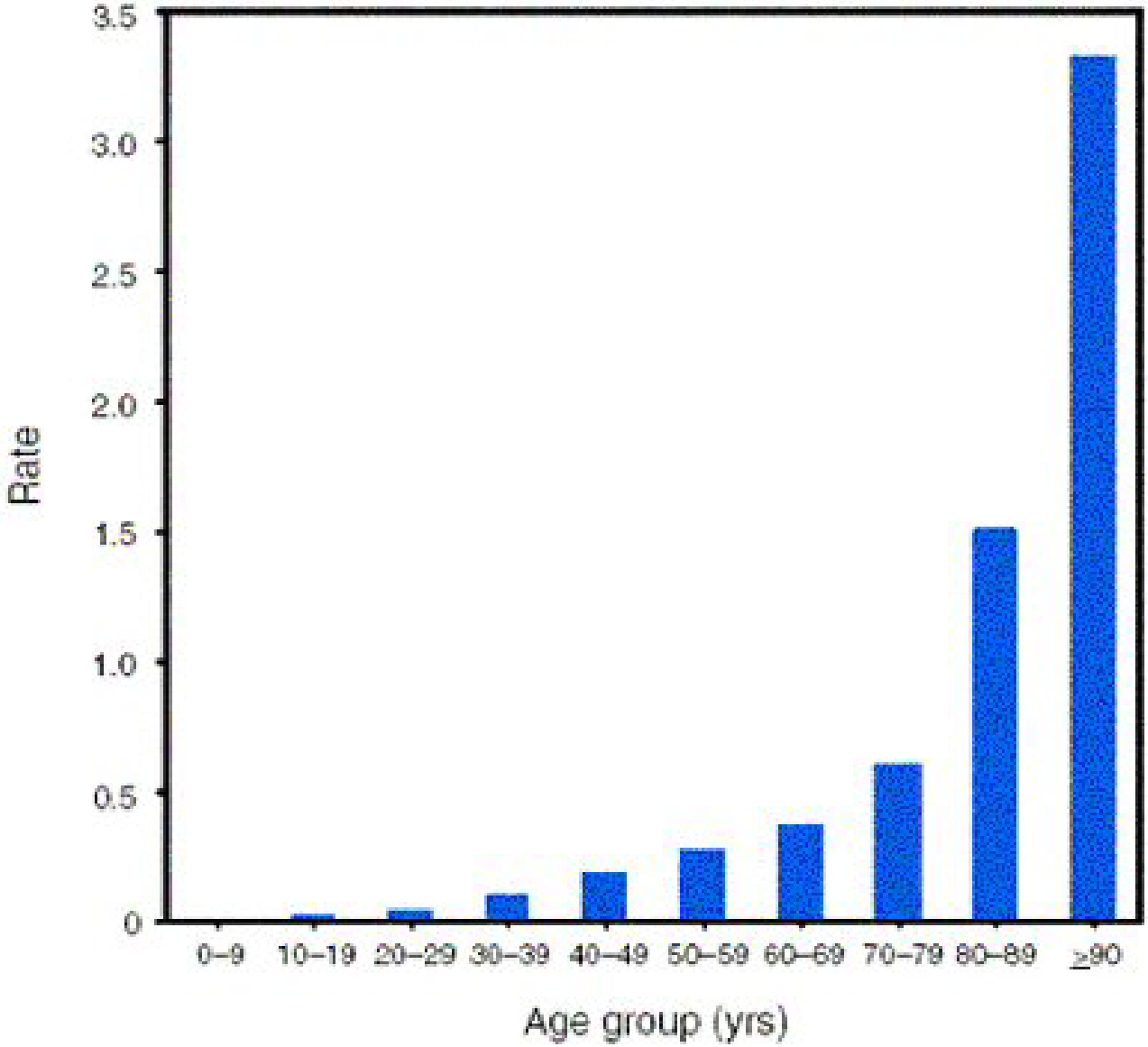
Medical conditions: Underlying medical conditions can also lead to accidental hypothermia. Patients with hormonal abnormalities (thyroid, adrenal, pituitary), and those with peripheral neuropathy (due to diabetes or other conditions) or may be less able to feel the cold and generate a shivering response. Patients with spinal cord injuries, similarly, may not be able to adequately shiver.

• Patients who have suffered strokes or brain tumors may have impaired thermal regulation centers in the brain.

•Overwhelming infection and sepsis may both present with a lowered temperature instead of fever.

•People with diabetes who have very low blood sugar can appear unconscious and very cold.
•**Medications:** Some medications can increase the risk of hypothermia by limiting the shivering mechanism including some psychiatric medications.

FIGURE. Age-adjusted rate* of hypothermia-associated death, by age group — United States, 2001



* Per 100,000 population.

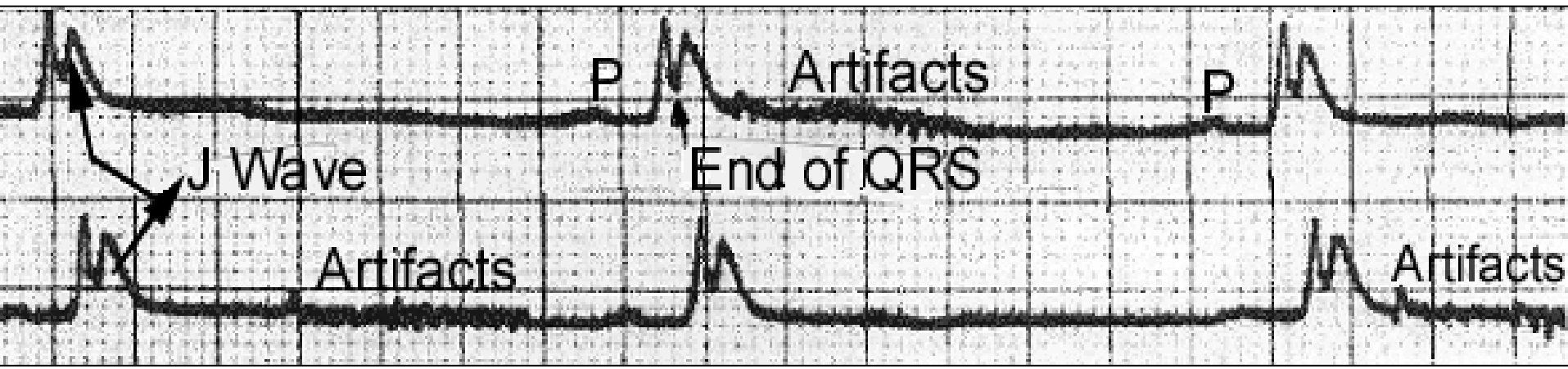
Hypothermia risk factors include: any condition that affects judgment (hypoglycemia), the extremes of age, such the present case (the present case) poor clothing, chronic medical conditions (such as hypothyroidism and sepsis), substance abuse, homelessness, and living in a cold environment. Hypothermia also occurs frequently in major trauma.

Hypothermia also is observed in severe cases of anorexia nervosa.

Main ECG features of hypothermia

1. **Rhythm:** Sinus bradycardia or AF present in 50-60% of the cases, when temperature is lower than 32ºC. (24) Less frequently, atrial flutter may be found, junctional rhythm and even degeneration into VF. These events appear in the second phase of hypothermia (out of exhaustion) when temperature is between 27ºC and 34ºC. Idioventricular rhythm and total AV block are described. Bradycardia is due to decrease in the ascending ramp in phase 4 in the P cells of the sinus node (decrease of diastolic depolarization, rhythmicity or automatism) by increased vagal reflex, even coexisting with increase of circulating catecholamines.Hypothermia constitutes one of the three causes for chronic metabolic bradycardia; the other two being obstructive jaundice (by bradycardizing effect of biliary salts on the sinus node) and myxedema.(25) On the other hand, hypothyroidism is one of the causes for metabolic hypothermia.

2. **Artifacts:** frequent fluctuation in the baseline as a consequence of artifact caused by the muscular trembling of the patient. This fact is found only in the initial phase (of struggle), when body temperature is between 36 and 32ºC



3. P wave: voltage decrease is described.(26) In the present case clear interatrial block(2)
4. PR interval prolongation. it tends to be prolonged as body temperature decreases;(27)
5. QT and QTc intervals prolongation.
6. Different types of arrhythmias (both supraventricular and ventricular).
7. Appearance of very characteristic extra wave, called J wave, sign of "camel hump", hump-like deflection, injury potential, and the eponym Osborn wave, located between the end of QRS complex and ST segment onset. J wave is characteristic of hypothermia; however, not pathognomonic, since it may be observed in other conditions in normothermia.
8. **QRS complex:** decrease in voltage and increase in duration. The latter may be mistakenly considered to be increased, by the presence of the so-called J wave (see item as belonging to QRS), resembling branch block or intraventricular disorder of the stimulus.The explanation for QRS broadening is in the decrease of rest potential and consequently, phase 0 rise velocity and negative dromotropism. The J wave "per se" is not significant for ventricular fibrillation (VF) appearance, since QRS complex duration prolongation constitutes a reliable sign for VF appearance.When rapidly induced hypothermia for heart surgery causes QRS prolongation, it causes VF in almost all patients.(28)
9. **J wave:** it constitutes the most typical electrocardiographic element; however, not pathognomonic, since it may be found in other clinical circumstances. It is located at the point where QRS ends (late δ wave) and the initial part of ST segment (J point). It corresponds to phases 1 and 2 of action potential. The J wave is due to different densities in the Ito channels concentration (phase 1) in ventricular myocardium thickness. These channels of the initial potassium outflow are very numerous in the epicardium and scant or absent in the endocardium. This fact justifies phase 1 of AP in the epicardium showing a notch. On the contrary, phase 1 of endocardial cells lacks a notch.

Characteristics location and polarity of hypothermic J wave

The J wave, also referred to as the J deflection, “the camel's hump”, camel-hump sign (29), “late delta wave”, elevated J-point(30), hat hook junction, hypothermic wave, K wave, H wave, current of injury or Osborn wave.(31)

Hipothermal or cool wave(32)

Normotermal

Hypothermic J wave presents the following features:

LOCATION: the J wave is located at the point where the QRS complex ends (late δ wave) occupying the initial part of the ST segment, corresponding to phase 1 and 2 of monophasic action potential.(33)

POLARITY: always positive and prominent in the leads that face the left ventricle: V_5 and V_6 and possibly and mainly in hearts in a vertical position in inferior wall leads,(34) unlike J wave found in Brugada syndrome, located in right precordial leads V_1 to V_2 or V_3.

VOLTAGE: slow and lasting inscription, and voltage greater in left leads V5 and V6 and inversely proportional to the severity of hypothermia, i.e. the lower the central temperature, the greater the voltage of J wave. In certain cases, J wave acquires a great voltage associated to superior convexity, mimicking the acute phase of myocardial infarction: "evolving myocardial infarction", which reverses with central temperature normalization.(35)

There is inverse and significant correlation between J wave voltage (mm) and central temperature in hypothermia.

ASPECT: it may appear resembling small secondary R wave (R'), falsely mimicking RBBB. The J wave is characteristic of hypothermia; however, not pathognomonic, since it may observed in normothermal circumstances.(36)

T WAVE MODIFICATIONS AND QT/QTc INTERVAL IN HYPOTHERMIA

T WAVE OF HYPOTHERMIA

T wave is altered, directly related with the degree of hypothermia.

When the J wave begins to show a concomitant great voltage, the T wave of the corresponding lead gets inverted.

T waves with changes in polarity or in the axis are called T wave alternans, and are observed during rapid blood transfusion in surgical hypothermia[1].

Flattening and asymmetrical inversion are described, mostly in the anterior wall.

QT/QTc INTERVAL IN HYPOTHERMIA

In hypothermia it is prolonged in most cases, as a consequence of appearance of extra wave in the J point, and ventricular repolarization slowing.

In all cases, the electric systole prolongation is reversible with hypothermia reversion.

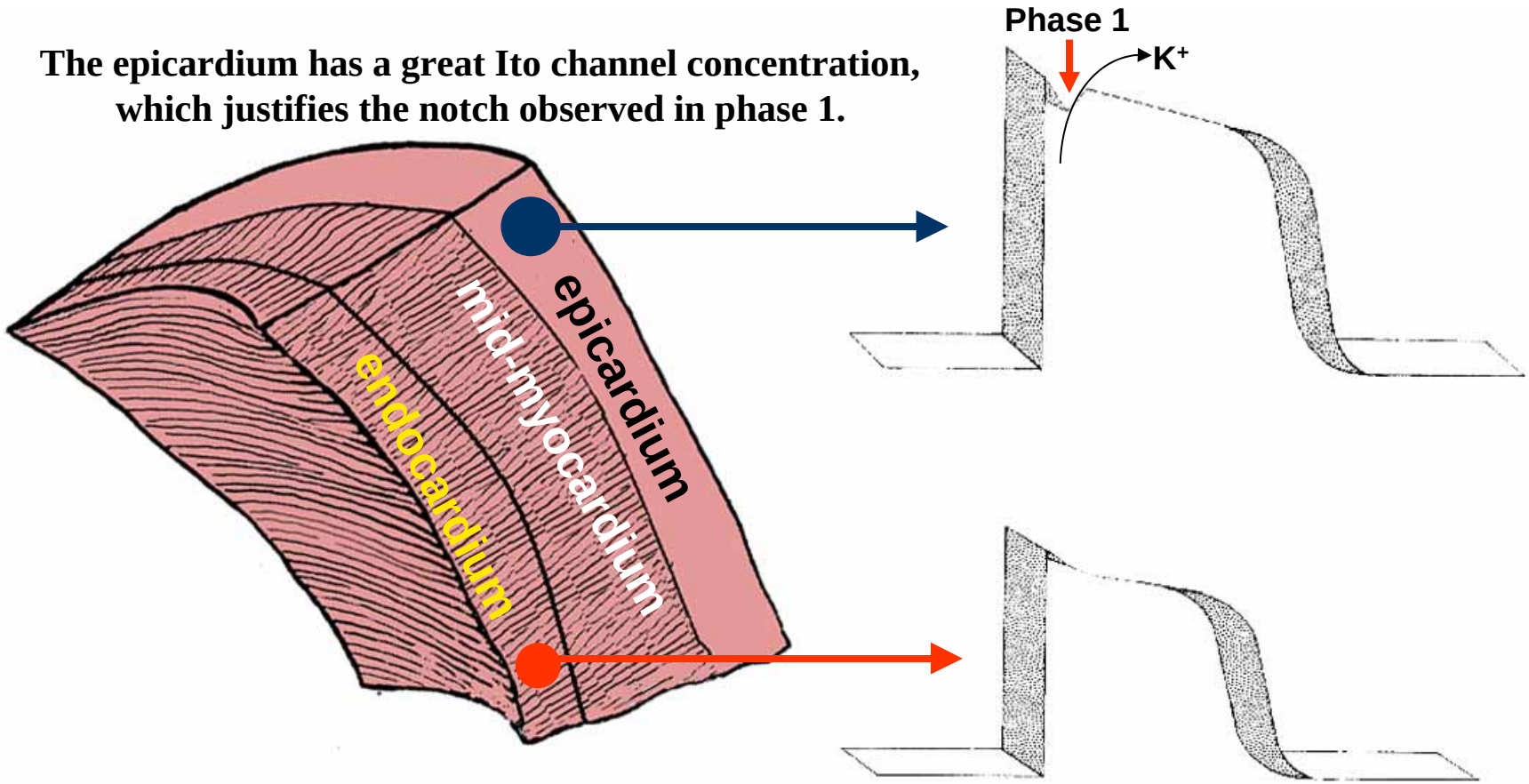
RHYTHM DISORDERS IN HYPOTHERMIA

Hypothermia is associated with the presence of greater incidence of several arrhythmias, both supraventricular and ventricular. Significant arrhythmias do not appear with body temperatures above 32ºC. This is the reason why this is the limit of moderate hypothermia, induced with therapeutic purposes.

AF with low rate of ventricular response, is described as an arrhythmia with significant incidence in moderate hypothermia, in some series being observed in up to 50% to 60% of cases. The percentage differences found in AF appearance in the different series in literature, relate with the heterogeneous nature of the populations studied. Elderly patients display a greater prevalence of atrial fibrillation. Thus, when the average age of patients in hypothermia is lower, AF percentage will be so too. VF has great possibilities of appearing with temperatures below 28ºC. When it occurs with values above 28ºC, associated myocardial disease must be suspected.

ELECTRIC HETEROGENEITY WITHIN VENTRICULAR MYOCARDIUM

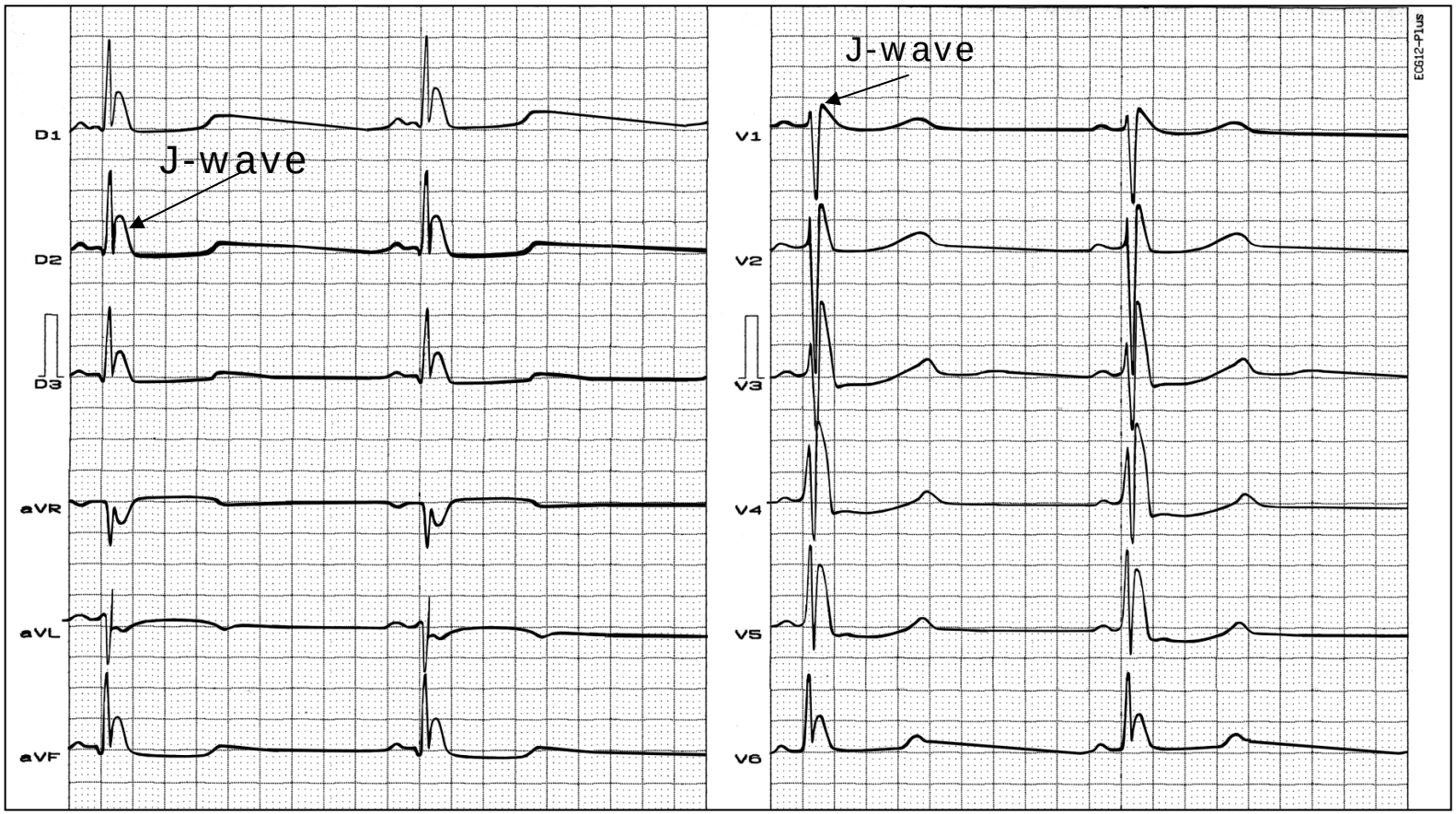
The epicardium has a great Ito channel concentration, which justifies the notch observed in phase 1.

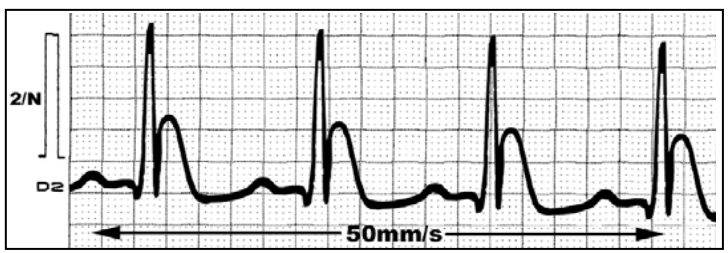


The endocardium does not have Ito channels, which conditions absence of notch in phase 1 of AP in these cells.

Outline of heterogeneity in the profile of action potential in ventricular wall thickness.

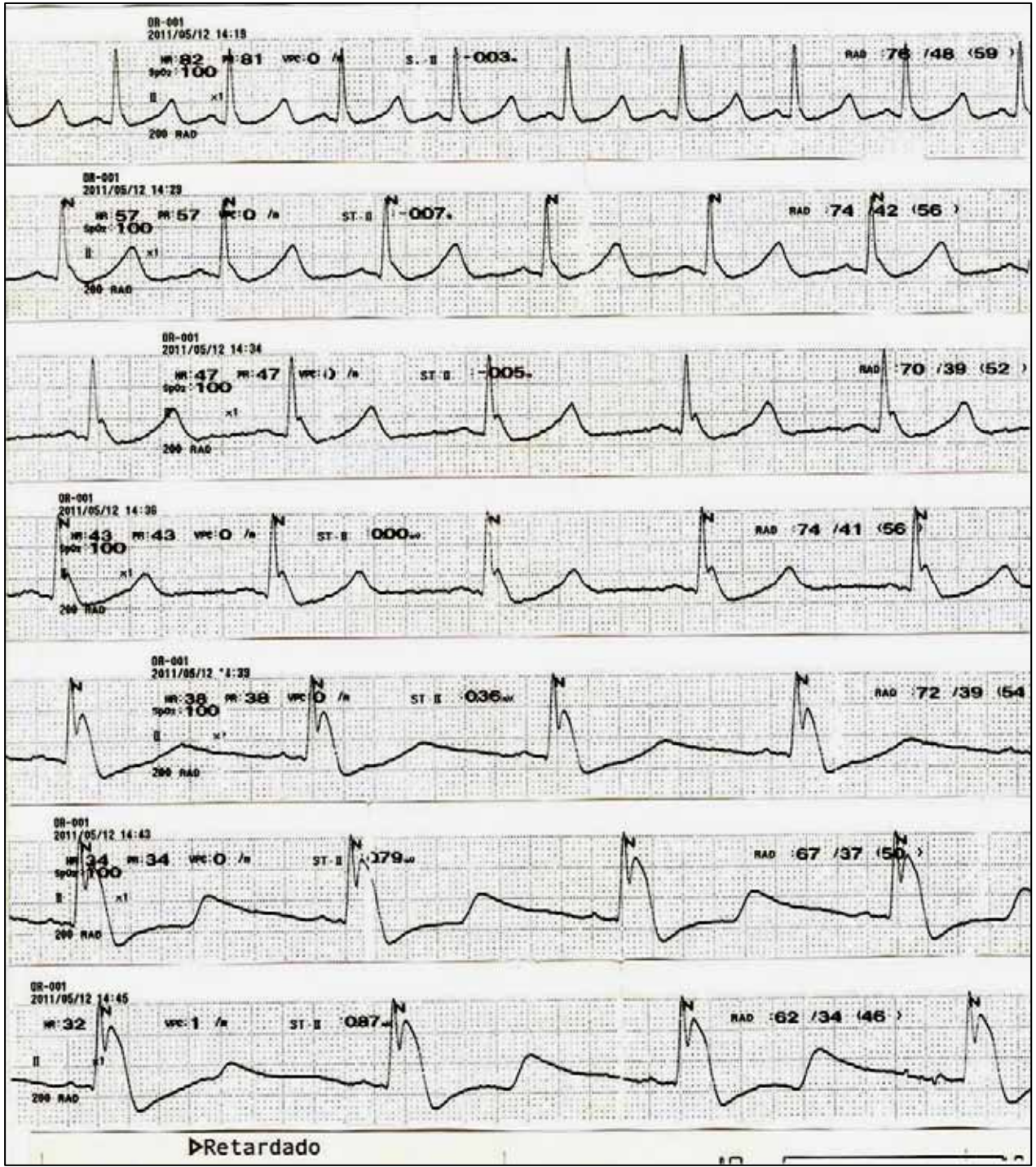
Name: ADS; **Gender:** Male; **Age:** 32 y.o.; **Ethnic group**: mulatto; **Weight:** 68 Kg; **Height:** 1.64 m; **Date:** 03/04/2002;
Steps to increase body temperature.





ECG diagnosis: sinus bradycardia of 30 bpm, prominent J wave, very evident in inferior leads and DI, as well as in all precordial leads.
Pseudo CRBBB determined by J wave, which is not part of the QRS complex.

Characteristic ECG of patient in severe hypothermia (40)



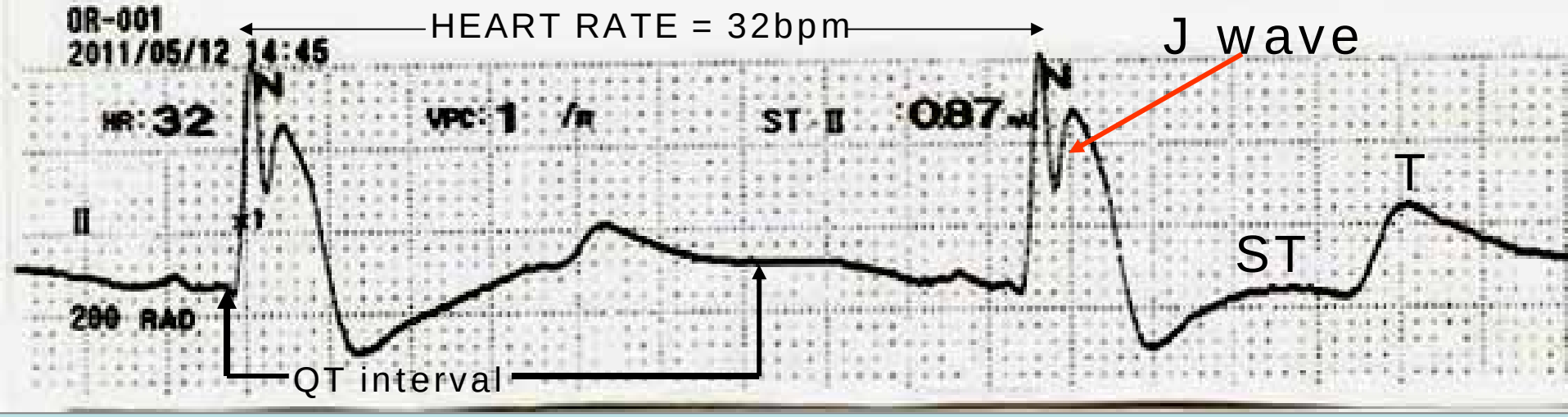
The tracing was obtained during cooling of the blood before a surgical procedure of the heart.

Although the ECG obtained was somewhat expected, what was striking is that the progressive development and augmentation of the J wave was recorded.

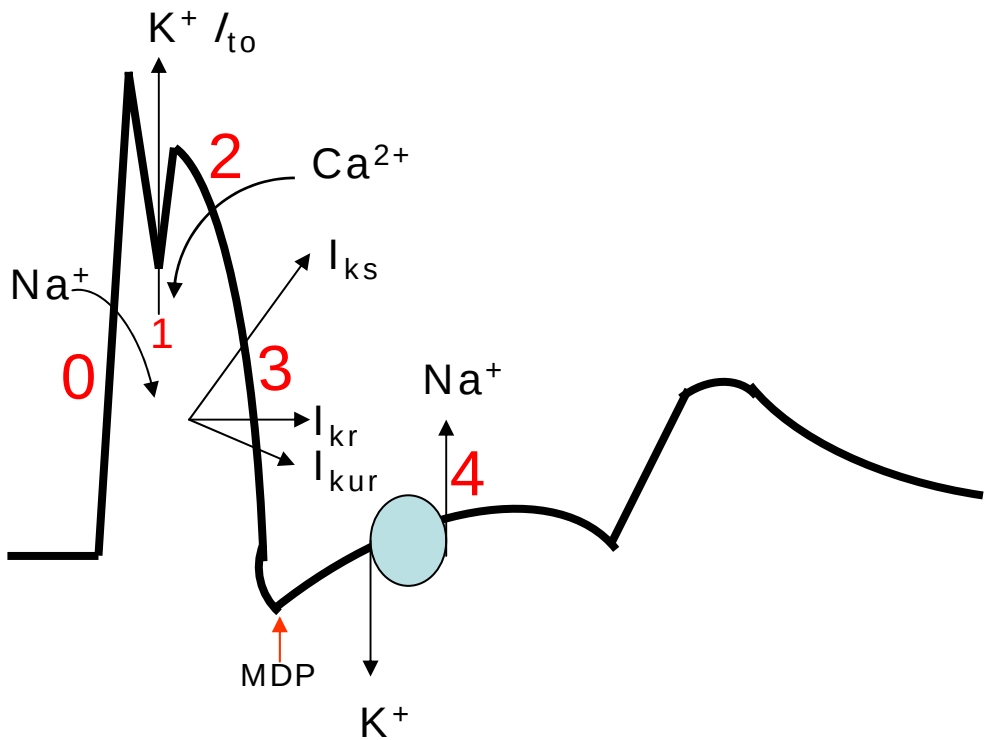
Most of the hypothermia cases are published in the moment when the patient is rescued and after recovery. On the other hand, in this case we can see the time course of changes up to the simulation of a monophasic action potential.

Additionally, significant bradychardia is observed and the QT interval was too prolongued, something that usually is not given much attention in the published cases.

Courtesy Prof. Dr Raimundo Puerta from Cuba



MONOPHASIC ACTION POTENTIAL



MDP: the most negative transmembrane potential achieved by a cardiac cell during repolarization. Also called maximum diastolic potential

ELECTROPHYSIOLOGICAL SUBSTRATE OF J WAVE

Experimental studies point out that J wave appearance is the consequence of the presence of transmural gradient in ventricular wall thickness, secondary to existence in the epicardium but not the endocardium, of significant notch in phase 1, mediated by a greater activity or density of initial transient outward potassium current. This greater activity and/or density of the Ito channel in epicardial cardiomyocytes, but not endocardial ones, accounts for the characteristic aspect of AP known as *"spike-and-dome configuration of the monophasic action potential"*. Moreover, the greater initial potassium outflow in the epicardium than the endocardium, causes phase 2 shortening in the epicardium, which conditions transmural dispersion of repolarization and J wave appearance, which carried to a certain level, causes a greater tendency to appearance of ventricular arrhythmia by the mechanism called functional reentry in phase 2.(37)

Experimental located cooling of the right ventricular outflow tract (RVOT) in vivo in dogs, resembles the electrophysiological alterations that occur in Brugada syndrome, causing J wave appearance secondary to Ito channel activation, and causing the classical aspect of "spike-and-dome configuration" in monophasic action potential of epicardial cells in the RVOT.(38)

Experimental evidence support the hypothesis that one heterogeneous distribution of the Ito channel in the ventricular wall thickness accounts for the spike-and-dome configuration in monophasic AP in the epicardium, and prominent notch in phase 1 and phase 1 shortening, which results in voltage gradient that manifests by J wave.(39)

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