30-y.o. Woman with Sudden atypical ECG pattern during Exercise Stress test

Mulher de 30 anos com súbitas modificações atípicas do ECG durante prova de esforço

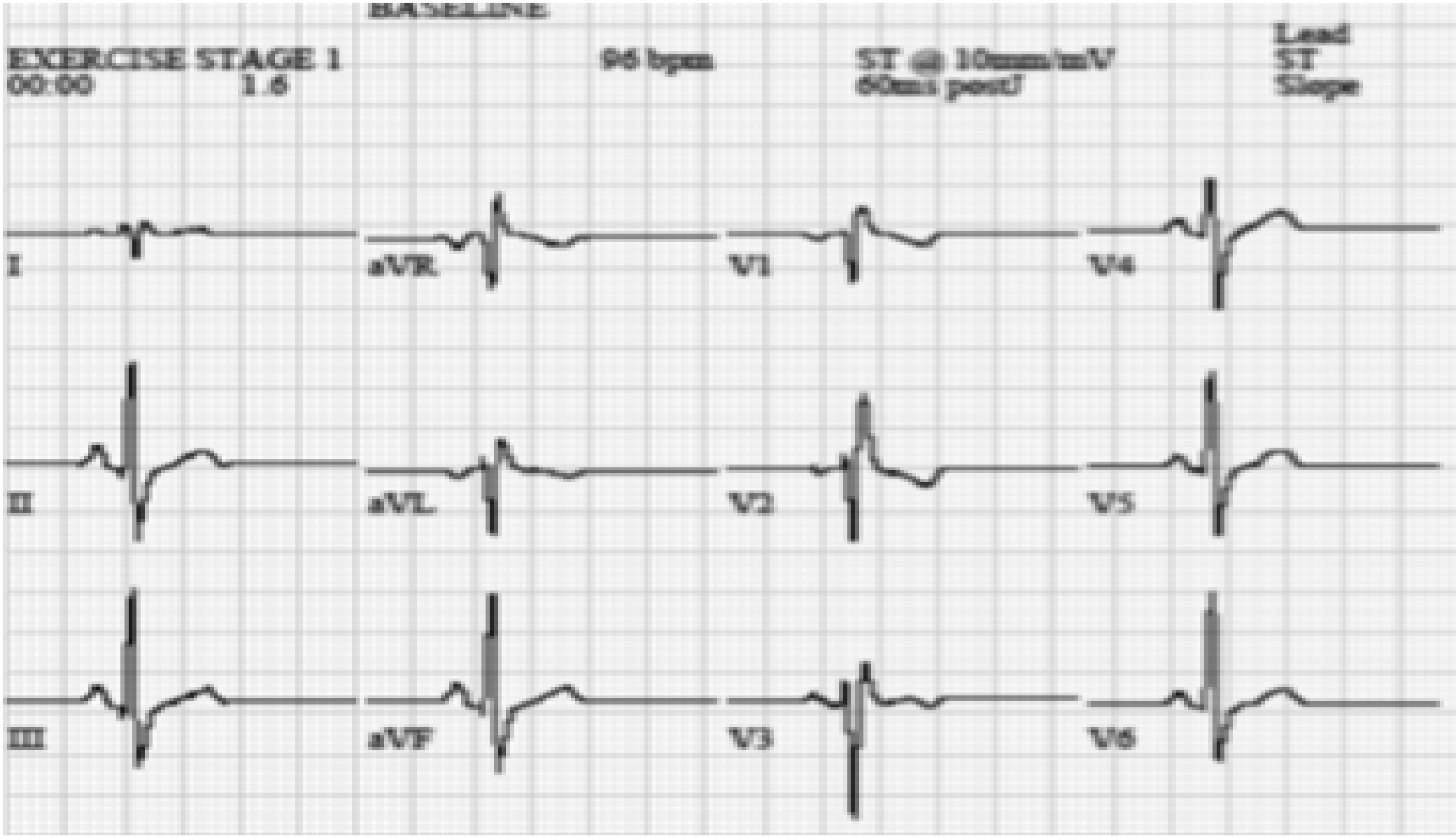
Case Report Dr Claudio Santibáñez Catalan M.D.

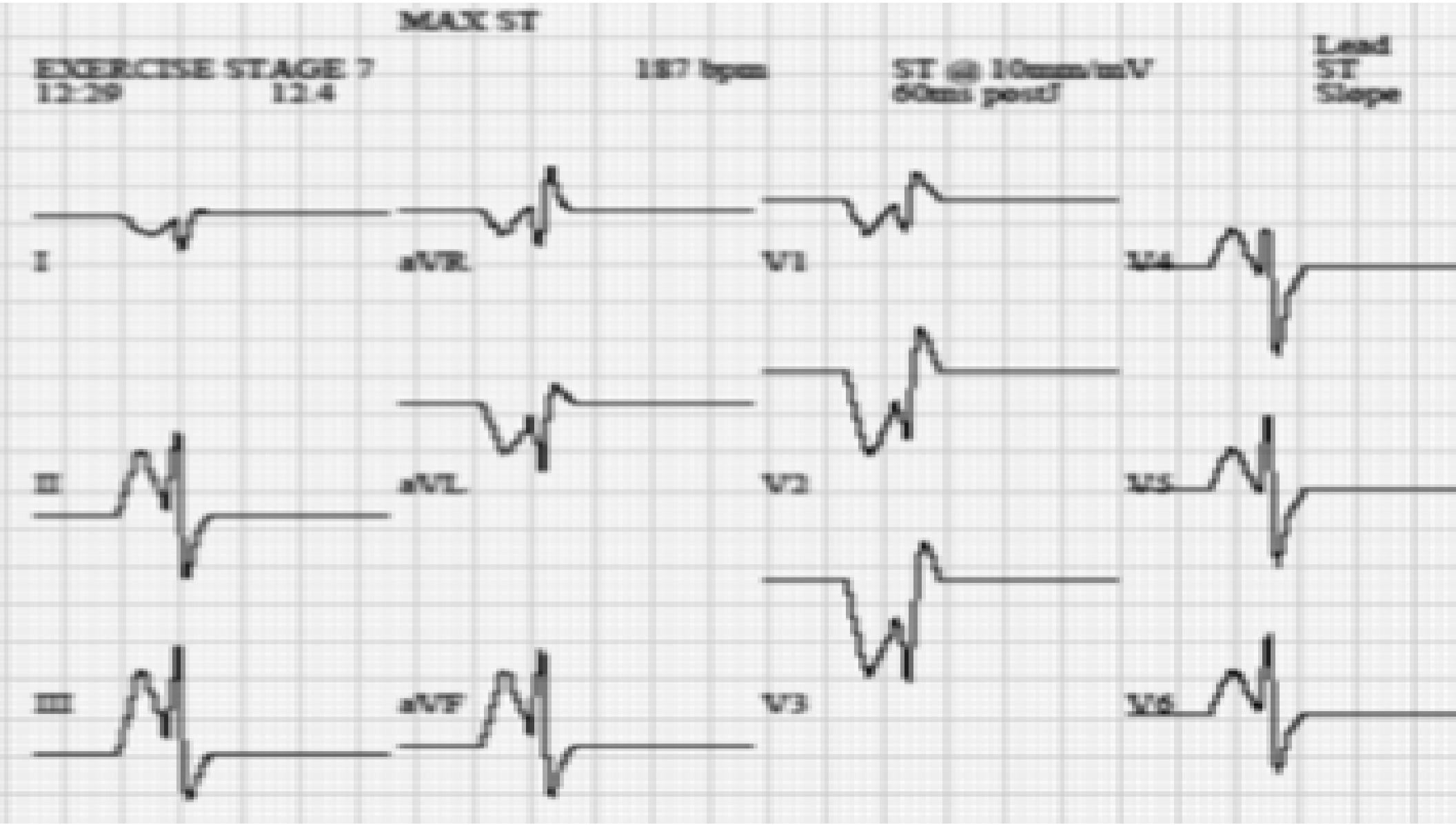
30-y.o. woman with abnormal ECG during stage 7 Exercise Stress test
First noted on evaluation for hypertension during pregnancy
Second at presentation to ED during febrile illness
No history of syncope or seizures
No family history of sudden death
Patient asymptomatic during exercise stress test
What is your diagnosis?

Portuguese

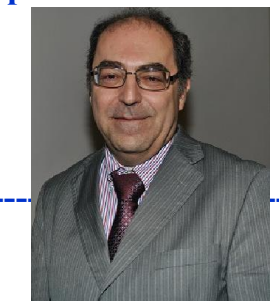
30-y.o. mulher com súbito aparecimento de ECG anormal atípico durante a fase7 da prova de esforço. Primeiro observou na avaliação da hipertensão durante a gravidez
Em segundo lugar na apresentação de ED durante a doença febril
Sem história de síncope ou convulsões
Sem história familiar de morte súbita
Paciente permaneceu assintomática durante o teste de esforço

Qual é o seu diagnóstico?


BASELINE
EXERCISE STAGE 1
00:00
1.6
96 bpm
ST @ 10mm/mV
60ms post J
Lead
ST
Slope
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6


MAX ST
EXERCISE STAGE 7
12:20
12.4
187 bpm
ST @ 10mm/mV
60ms post J
Lead
ST
Slope
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

Português: Andrés, gostaria de colocar minha opinião: Acho que existe alteração nos 2 eletrocardiogramas e não somente no segundo: Vejamos: No primeiro ECG a frequência cardíaca é de 96 bpm, mas vemos apenas um complexo QRS, sendo que antes e depois do complexo a linha esta iso-elétrica com se houvesse saturação do sinal. No segundo ECG a frequência cardíaca é de 187 e vemos novamente apenas um complexo QRS com novamente a linha isoelétrica antes e depois do QRS. A janela que tem fora da linha isoelétrica no primeiro ECG é de 13 mm e no segundo de 8 mm. Se observarmos ainda, a linha de base se localiza em locais diferentes nos ECG. O que parece aberrante para mim é o final da onda T do complexo precedente (visível somente esta parte do complexo sem o QRS) seguido do próximo batimento sem onda T que foi amputada. A frequência cardíaca foi registrada corretamente pelo monitor mas o registro teve problemas técnicos, diria que se trata de um caso de um problema técnico do aparelho de ECG. Vejo apenas um atraso final da condução localizada no ramo direito (BRD). Não dá para medir a amplitude de P (SAD?) e nem sua duração.



Aguardo os outros comentários dos colegas especialistas.

Claudio Pinho
Faculdade de Medicina PUC Campinas/ Brasil.

English

Andrés,

I would like for you to distribute my opinion: I think there is alteration in 2 electrocardiograms and not just in the second. Let's see, in the first ECG, heart rate is 96 bpm, but we only see a QRS complex, since before and after the complex the line is isoelectric as if there was signal saturation. In the second ECG, heart rate is 187 and we only see a QRS complex with isoelectric line again before and after QRS.

The window it has outside of the isoelectric line in the first ECG is 13 mm and in the second 8 mm. If we observe still, the baseline is located in different places in ECG. What seems aberrant for me is the end of T wave of the previous complex (this part of the complex is visible only without QRS) followed by the next beat without T wave that was amputated.

Heart rate was recorded properly by the monitor, but the recording had technical issues; I would say it is a case of a technical problem of the ECG device.

I only see end conduction delay located in the right branch (RBBB). It is not possible to measure P amplitude (RAE?) or duration either.

I wait for other comments by the specialists.

Claudio Pinho M.D.
Faculdade de Medicina PUC Campinas/ Brazil.

English

The repolarization of the previous beat is "summed" with the P wave of the next beat and then the QRS is recorded. After the QRS the recording is "clipped" - no further recording - in other words the recording window of the ECG machine has been set incorrectly. It has to do with the heart rate also.

Spanish

La repolarización del latido previo se “suma” a la onda P del latido siguiente y luego se registra el QRS. Después del QRS, el registro está “recortado” –no hay más registro-, en otras palabras, la ventana de registro del dispositivo ECG se configuró incorrectamente. También se relaciona con la frecuencia cardíaca.

Português

A repolarização do batimento precedente se “une" com a onda P do próximo batimento e, em seguida, o QRS é registrado. Após os QRS o registro é "cortado" - não mais é registrado - em outras palavras, a janela de gravação do aparelho do ECG foi definida incorretamente. Tem a ver com a frequência cardíaca também.

Prof. Dr. Pedro Brugada Terradellas
Chairman, Cardiovascular Division
UZ Brussel, Brussels,

Spanish Andrés, creo que ese ECG durante la ergometría tiene artefactos o problemas técnicos. Si no es así, muestrenmé varios latidos de todas las derivaciones.
Oscar Pellizon M.D. Argentina.

English: Andres, I think that these ECGs preformed during exercise testing have artifacts or technical problems. If not, show me several beats from each lead.

Hola
¿No se podrá tener mas de un latido por derivación? No se podrá tener mas de un latido por derivación? No se podrá tener mas de un latido por derivación? Se hace muy difícil con un solo latido especular o diagnóstico, pero la P negativa en I sugiere origen izquierdo y se hace masiva, salvo que sea un ritmo de la unión y que haya metido mucho QRS.
Seria importante poder ver mas de un latido en cada derivación...
Hello,
Can't we have more than one beat per lead? It is very hard to hypothesize a diagnosis with a single beat, but P becomes negative in I, suggesting left origin and it becomes massive, unless it is a junctional rhythm and that produced a lot of QRS.
It would be important to see more than one beat per lead…

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With exercise there is Left atrial rhythm probably from region of the LA appendage. The distortion of the QRS may be due to atrial repolarization which is inscribed opposite to that of the P wave.

Português:

Com o exercício há um ritmo atrial esquerdo provavelmente procedente da região do apêndice do AE. A distorção do QRS pode ser devida a repolarização atrial que é inscrita oposta a onda de despolarização atrial(onda P.)

Spanish

Con el ejercicio hay ritmo de la AI, probablemente desde la región de la orejuela de la AI. La distorsión de QRS puede deberse a la repolarización auricular, que se inscribe en forma opuesta a la de la onda P.

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Perdón el diagnóstico de que? Del ECG basal o del totalmente artefacteado donde las ondas P del plano frontal no coinciden con las del plano precordial, los complejos QRS parecen ser similares y la ondas T desaparecieron???
Bueno quizás la repolarizacion ventricular se produzca antes que la despolarizacion, en esta vida todo es posible.....👀👀
Cordialmente Julia

Coincido con Julia; me huele a un desperfecto en el aparato o algo artificial. No hace mucho me llegaron varios casos de Brugada fabricados por un mismo electrocardiógrafo (tener cuidado con los programas de computación) y una ergometría que partía de 180 x min y llegaba a 300 x min diagnosticado como aleteo auricular 1:1; el QRS medía aproximadamente 20 mseg.....Abrazo para todos
Pablo Ambrosio Chiale

Spanish

Adail no sirve un solo latido. 1. Esta bien tomado
2. Es una extrasistole auricular y un aumento en el grado de bloqueo de rama que presenta el paciente. No se observa la repolarizacion ventricular
3. Refiere 184 latidos por minuto. Así que sin la tira de ritmo (básico para poder evaluar una ergometria, así ni siquiera se puede evaluar la FC alcanzada ni la repolarización ventricular)
Tiene 2 opciones: una taquicardia auricular ectopica o el sistema de captura automatica captura una ESV.
Soy reiterativo pero sin la tira de electrocardiograma, me resulta insuficiente para realizar un diagnostico, solo realizar su pociones.
Un abrazo grande y ojala el Potro nos envié todo el trazado.
Martín

English
Dear Adail: With only one beat for lead is not possible the diagnosis
1. The strip is not inverted
2. It is an atrial extrasystole and an increase in the degree of bundle branch block. Ventricular repolarization is not observed
The second trip refers 184 beats per minute. So without the rhythm strip (to evaluate a basic ergometry, and even can not evaluate the maximal HR and the ventricular repolarization)
We have 2 options diagnosis: ectopic atrial tachycardia or the system automatic capture an atrial premature beat.
I'm repetitive but electrocardiogram strip is insufficient for the diagnosis. Consequently, any conclusion is mere supposition.
A big hug and hopefully Andrés sent us the entire trip.
Martin Ibarrola MD Argentina

Spanish

Estimado amigo Andrés el "Potro"
Mi opinión del segundo ECG durante el esfuerzo: es un trazado muy grotesco, para mi se trata de un artificio o se me ocurre de "ECG-copia" creado por la computadora del programa de la ergometría al promediar durante el esfuerzo con altas frecuencias de 187 min (según se observa en el trazado) la onda P sumado a la onda T y posiblemente a los ruidos generados por el movimiento de los cables y electrodos por el movimiento de los brazos y tórax . Todo eso generaría amplitudes que sumadas da esa pseudo P gigante y la ausencia de la onda T ,porque el programa se confunde y promedia todo junto. Por ello debería trabajarse con filtros de 05 Hertz y cables blindados para evitar esa "copia" grotesca.

Un abrazo grande

Juan José Sirena M.D. Santiago querido Argentina

El electro de 12 derivaciones que envió nuestro maestro Profesor Andrés Pérez Riera es de mandinga (el diablo en guarani entrerraniano (figura 1) Se observa un ST -T elevado en V4 ,V5 ,V6 Supongamos que una arteria marginal inferior se obstruyó , pero no asi, porque siempre en estos casos el ST-T en V6 es mas elevado que V4 , y aquí ocurre lo contrario.

El pasaje de ST elevado a ST deprimido es progresivo con zona borde, y aqui no hay zona borde electrocardiográfica Tampoco he visto algún caso de isquemia aguda con ST-T elevado en aVL y AVF, , pero con ST -T deprimido en DII ,DIII Esto esta contra toda lógica de la electrofisiologia de las isquemias agudas. Si es una obstrucción súbita de una arteria CX proximal y dominante que da origen a la arteria sinusal puede presentar una parada sinusal y ritmo nodal como este caso

Que le paso a esta mujer joven asintomática que presento una obstrucción súbita de la CX en medio de una ergometría ? Esto puede ocurrir en 2 situaciones clínicas 1) que la CX estaba obstruida 99% y al bajar el flujo coronario la arteria se obstruye 100% por colapso(pero no conozco personas con 99% de obstrucción asintomáticas); 2) que la paciente tenga una insuficiencia endotelial y desarrolló un Prinzmetal (pero esta entidad se desarrolla cuando existe predominancia colinérgica(es bien sabido que la acetilcolina es vasocontrictiva cuando hay deficiencia del ARDF, y esto no ocurre al esfuerzo que tiene predominancia adrenérgica

Que la paciente tenga una alteración genética en los receptores 1 y 2 de la adenosina ? Raro.

A pesar que no se entiende este caso hicimos un repaso de la electrofisiologia y fisiopatologia de las isquemias agudas.

Sabemos lo que no es ,pero no sabemos que es esto Es decir mandinga estuvo en el medio

Un fraternal abrazo

Samuel Sclarovsky

Responsable por este ECG según Samuel

Final Comments and diagnosis

By Andrés Ricardo **Pérez-Riera M.D.Ph.D**.
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Normally the location of atrial repolarization (Ta or TP wave) is coincident with ventricular depolarization (QRS complex). The absence of Ta is explained by to be concealed by the ventricular phenomenon. Ta wave usually not visible because it is temporally coincident with QRS. It is concealed by QRS. It represents atrial repolarization. See figure at side

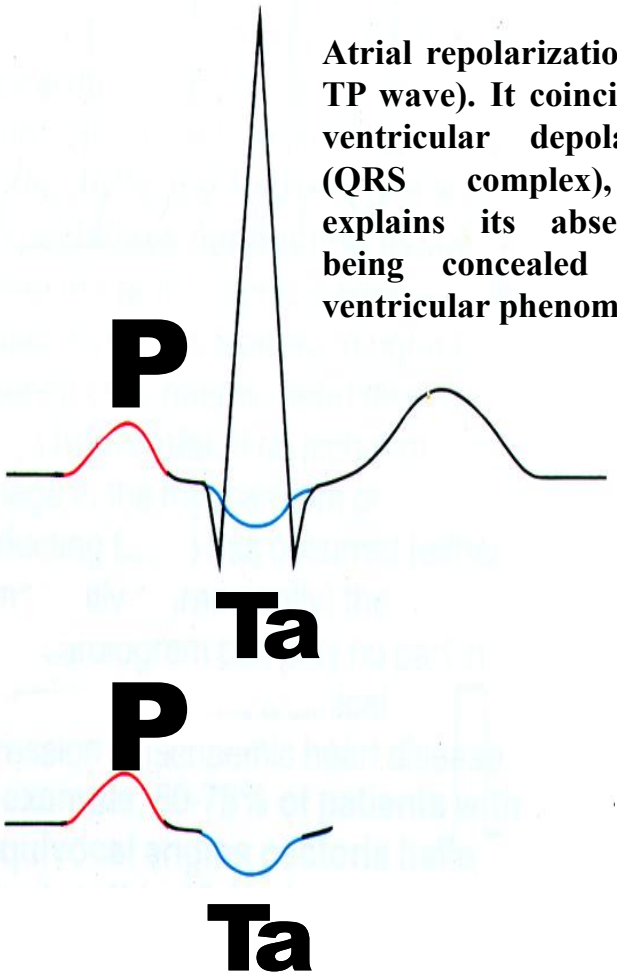
Ta polarity is opposite to the P wave and its magnitude is 100 to 200 mμV.

Sometimes it may appear in the PR segment, ST segment and the T wave.

During exercise, it may in theory, cause ST segment depression and resemble myocardial ischemia (1)

False positive must be suspected in the presence of (1)

1. ***Important PR segment depression in maximal strain;***
2. ***Longer time of exercise and maximal strain faster than those truly positive;***
3. ***Absence of effort-induced pain(such as the present case)***
4. ***P wave of voltage higher in maximal strain.***

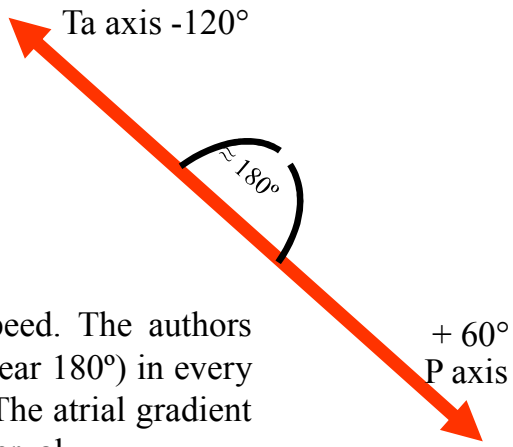


Atrial repolarization (Ta or TP wave). It coincides with ventricular depolarization (QRS complex), what explains its absence for being concealed by the ventricular phenomenon.

1. **Sapin PM, Koch G, Blauwet MB, McCarthy JJ, Hinds SW Gettes LS Identification of false positive exercise tests with use of electrocardiographic criteria: a possible role for atrial repolarization waves. J Am Coll Cardiol. 1991 Jul;18(1):127-35.**

Ta wave has a saucer-like shape

Normal Ta wave axis is near -120° ≈ 180º opposite to P axis



Hayashi et al (1) studied the P and the Ta waves of two patient groups with AV block:
Group A: patients minimal clinical evidence of heart disease
Group B: patients with more severe disease.
Waves were magnified with a direct-current amplifier and recorded at a high paper speed. The authors verified that in Group A the P and the Ta waves were recorded in the opposite direction (near 180º) in every lead and there was a linear relationship between the amplitude of the P and the Ta waves. The atrial gradient was nearly zero. There existed a positive correlation between the P + Ta time and the PP interval

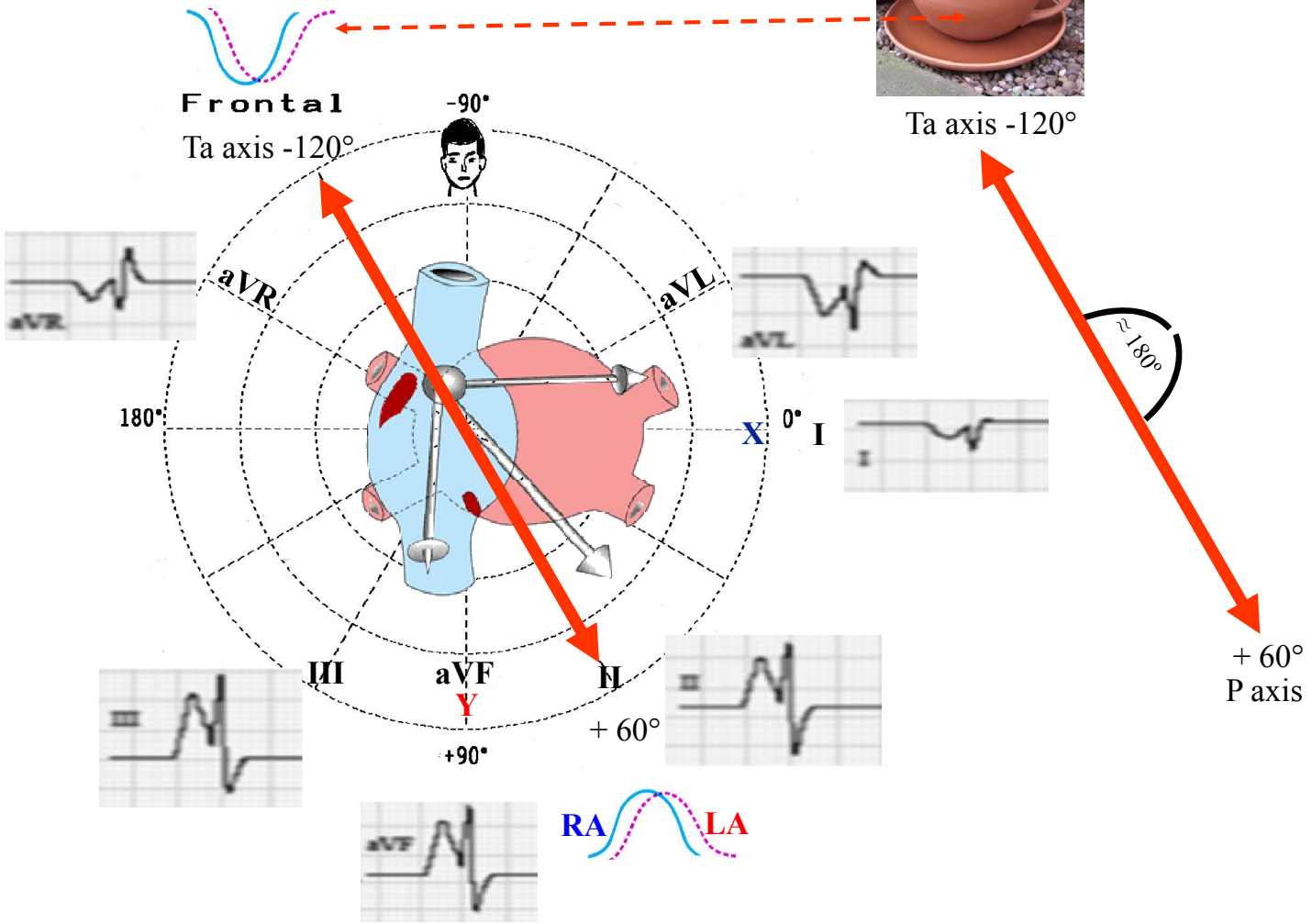
In Group B patients there were significant differences between P wave and Ta wave with respect to form, polarity, amplitude, duration, and the relationship between the Ta and the P waves. The atrial gradient was markedly large. Ta loop may be very useful in separating normal from diseased atria in individuals with AV block. There are some frequency differences between the Ta wave and the QRS complex. If the Ta wave could be extracted from the QRS complex by the use of some kind of filter when A-V block dose not exist, most of the Ta wave could be visualized. This, along with high fidelity recording techniques, may help detect atrial abnormalities in patients without AV block. Attention should be paid to the deviation of the PR segment caused by the Ta wave in daily ECG's to detect atrial abnormalities. The Ta wave extends into the ST segment and, while describing the deviation of the ST segment, the influence of the Ta wave should be kept in mind.

1. **Hayashi H, Okajima M, Yamada K.Atrial T(Ta) wave and atrial gradient in patients with A-V block. Am Heart J. 1976 Jun;91:689-98.Am Heart J. 1976 Apr;91:492-500**.

Normal Ta wave axis is near -120° ≈ 180º opposite to P axis

Ta wave has a saucer-like shape

Ta axis -120°



Holmqvist et al(1) studied, 40 consecutive patients with third-degree AV block to better analyze the Ta wave. In this population the Ta wave had the opposite polarity, a duration two to three times that, of the P wave and Ta peak may occasionally be located in the PR interval during normal AV conduction, it is unlikely that enough information can be obtained from analysis of this segment to differentiate normal from abnormal atrial repolarization. Hence, an algorithm for QRST cancellation during sinus rhythm is needed to further improve analysis.

1. **Holmqvist F, Carlson J, Platonov PG. Detailed ECG analysis of atrial repolarization in humans. Ann Noninvasive Electrocardiol. 2009 Jan;14:13-18.**

Br Heart J 1993;70:205–207

BRITISH
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Editorial

Concerning falsely negative and falsely positive electrocardiographic responses to exercise

The recognition that the progression of coronary artery disease can be slowed or even reversed by risk factor modification, that in some discrete subsets of patients revascularisation can prolong life as well as relieving symptoms, and that myocardial ischaemia may be symp-

the guide wire beyond the area of obstruction and connecting it to the V lead terminal will show marked ST segment changes while the simultaneously recorded body surface ECG often shows no change.[4] There are several explanations for the phenomenon of absent ECG

tomatically silent has led to an increased commitment to diagnosing ischaemic heart disease before the onset of ischaemic events. To accomplish this goal, exercise tests employing one of several protocols are being used with increasing frequency by family physicians, internists, and cardiologists not only to determine the causes of chest discomfort and the presence of residual ischaemia after a myocardial infarction but also to screen patients for the likelihood of ischaemia. Often the results of such exercise tests are used to determine whether or not other studies and procedures, particularly cardiac catheterisation and revascularisation, are indicated. For this reason an understanding of factors that can cause either falsely negative or falsely positive results has assumed increasing importance.

Effects of probability

It is now generally recognised that the probability of coronary artery disease in the population being screened affects the interpretation of the exercise electrocardiogram (ECG).[1] In a population with a low probability of ischaemic heart disease, a negative test is more likely to be a true negative whereas a test meeting the criteria for positivity would be suspect. Conversely, in those with a high probability of ischaemic heart disease a positive test is more likely to be an indication of ischaemia (and coronary artery disease) and a negative test would be suspect. For this reason, factors capable of giving both false negative changes in the setting of myocardial ischaemia. (*a*) The location of ischaemia may be electrically silent relative to the body surface ECG. This is particularly true if the ischaemia is within the distribution of the circumflex coronary artery.[5] (*b*) The magnitude of the ischaemic zone may be such that the solid angle created across the ischaemic margins from the body surface leads is inadequate to generate ST segment changes on the body surface.[6,7] This is probably the mechanism underlying the dichotomy between the changes in the local electrogram and the body surface electrocardiogram during angioplasty. (*c*) An area of ischaemia may be so diffuse that the electrical gradients oriented in one direction will be cancelled by electrical gradients oriented in the opposite direction.[8] This may occur in the setting of left main coronary artery disease. However, in this setting, one would expect that other findings such as a decrease in exercise tolerance, a fall in blood pressure during exercise, or the development of auscultatory findings suggestive of ventricular dysfunction—that is, a third sound or a murmur of mitral insufficiency after exercise—would suggest myocardial ischaemia. For this reason it is clearly important to incorporate these variables into the evaluation of the patient's response to exercise. The actual incidence of false negative ECG changes in patients either with or without a high pre-test likelihood of ischaemia is not known because in most studies the result is compared with the anatomical findings determined by coronary angiography rather than with the presence of

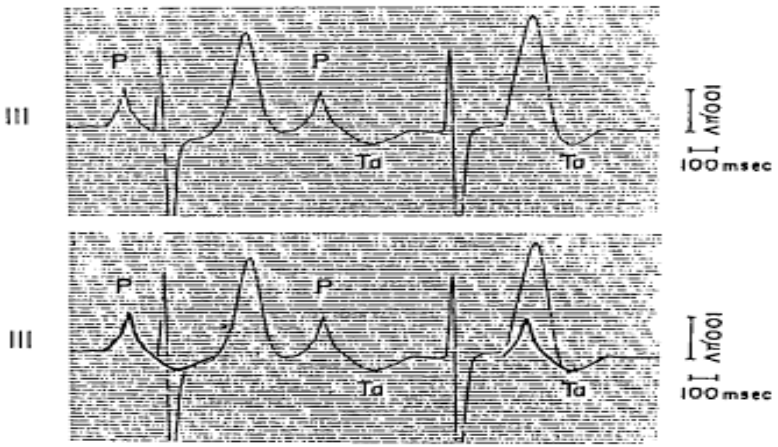
tive and false positive ECG responses must be appreciated and considered, particularly in patients in whom the result is contrary to that predicted by the likelihood of the disease. Indeed, even in patients in whom the result is as predicted by the probability of disease, it is important to appreciate that the specificity and sensitivity of the exercise test when referenced to coronary anatomy is between 75% and 85%.[2,3] Thus even in these patients myocardial ischaemia may occur in the absence of typical ECG changes and, in some situations, ECG changes characteristic of ischaemia may occur in the absence of myocardial ischaemia.

Falsely negative responses

Myocardial ischaemia may occur in the absence of changes in the body surface ECG during coronary angioplasty. In these patients the electrogram recorded over the ischaemic area during balloon inflation by extending ischaemia. In one recent study which compared the exercise ECG with coronary artery flow reserve[9] three of 17 patients with abnormal flow reserve had normal ECG exercise tests while three of 23 patients with normal flow reserve had abnormal exercise tests. In another study that compared cross sectional exercise echocardiograms with exercise electrocardiograms[10] nine of 13 patients with >50% obstruction of one or more coronary arteries showed normal exercise ECGs but abnormal exercise echocardiograms.

Falsely positive responses

Various factors can also cause ECG changes with exercise that meet the standard criteria for an ischaemia response—that is, horizontal or downsloping ST segment depression $\geqslant 100\ \mu V$ measured 80 ms (0·80 s) after the junction of the QRS complex and the onset of the ST segment (the J point)—even in the absence of exercise



Example of false ST segment depression caused by atrial repolarisation in a patient with high grade atrioventricular block. Reprinted with permission from the American Heart Journal.[14]

induced myocardial ischaemia or ECG changes at rest. These include agents that alter the plateau phase or the phase of rapid repolarisation of the action potential or both and by this mechanism lead to the development of electrical gradients which cause ST segment changes on the body surface ECG. Examples include, but are not limited to, hypokalaemia, drugs that directly affect the myocardial cell—that is, digitalis and the type I and III antiarrhythmic drugs—and left ventricular hypertrophy.[11]

Effects of atrial repolarisation

An additional factor that may affect the ST segment is the atrial repolarisation wave or the atrial ST segment and T wave. Most medical students and house officers realise that there should be an atrial ST segment and T wave but will state incorrectly that the atrial T wave is located within QRS complex. Most often, there is little appreciation of the magnitude, direction, or duration of the atrial repolarisation wave or that the atrial ST and T wave may cause electrocardiographic changes in the ST segment that are indistinguishable from those caused by wave with a negative atrial T wave (Ta). A P wave occurs just before the first QRS complex and the ST segment is depressed. The second P-Ta complex is completed before the second QRS complex and the ST segment is not depressed. In the lower panel the entire P-Ta complex is superimposed on the first QRS complex, and illustrates that the ST segment depression is due to the atrial repolarisation wave. The onset of the third P-Ta complex coincides with the onset of the second T wave and is responsible for the rather acute angle between the ST segment and the T wave.

The role of atrial repolarisation in causing false positive electrocardiographic responses to exercise was recently the topic of an article, editorial, and exchange of letters in the *Journal of the American College of Cardiology*.[12 13 16] In this study, patients with exercise-induced ST segment depression meeting criteria for ischaemia, but in whom ischaemia was viewed as unlikely on the basis of cardiac catheterisation or radionuclear studies, were compared with a matched group of patients in whom similar exercise-induced ST segment changes were attributed to ischaemia. The patients with a false positive exercise test were characterised by markedly downsloping PR segments at peak exercise which reflected the atrial ST segment. They were also identified by a longer exercise time and more rapid peak heart rates than patients in whom the ST segment depression was the result of ischaemia. This reflected the increase in the amplitude of the P wave and, in the opposite direction, of the amplitude of the atrial T wave induced by the increase in heart rate. The third factor identifying the patients with a false positive exercise test was the absence of exercise-induced chest pain. Patients with a false positive exercise test were also noted to have taller P waves during exercise and a greater augmentation of the P wave amplitude during exercise than patients with a truly positive test. In this retrospective study the combination of a downsloping PR segment in two of the three inferior leads (II, III, and/or aVF) and either an exercise duration of more than 4 minutes or heart rates at peak exercise of more than 125 bpm identified the false positive responders with a sensitivity of 84% and a specificity of 87%.

There is no reason to expect that patients with exercise-induced myocardial ischaemia will be any less liable to the influence of atrial repolarisation or to the other fac-

ischaemia. The potential impact of atrial repolarisation on the ST segment is appreciated by those with an interest in electrocardiography[12 13] and the characteristics of atrial repolarisation have been studied in laboratory animals and in humans.[12 13] It has been shown that the atrial ST segment and T wave are usually opposite in direction to the P wave and of significant magnitude, with the area under the atrial T wave roughly equivalent to the area under the P wave. In humans the duration of the interval from the onset of the P wave to the end of the atrial T wave averages 450 ms at a rate of 70 bpm[14] but may last as long as 600 ms.[15] Normally, the interval from the onset of the P wave to the end of the QRS complex is approximately 240 ms (PR = 160 ms, QRS = 80 ms). Thus approximately 200 ms of the atrial ST and T wave will extend beyond the end of the QRS complex. Because the interval from the end of the QRS complex to the end of the T wave is in the range of 300 ms, it is apparent that the atrial ST and T wave will extend well into the middle portion of the ventricular T wave. In those leads with a positive P wave, the downsloping nature of the atrial ST segments and the inverted atrial T wave may cause ST segment depression which is indistinguishable from that caused by ischaemia. The accompanying figure from an article by Hayashi *et al* shows an example of this effect. The upper panel shows lead III from a patient with high grade atrioventricular block and illustrates a positive P tors capable of causing a false positive test. The point is that ECG changes or their absence associated with exercise should not, by themselves, be considered an absolute marker for the presence or absence of ischaemia. As with any other test the results must be interpreted within the context of the clinical presentation and the historical and physical findings. As screening for coronary artery disease becomes more prevalent and as those with less and less experience in performing and interpreting the electrocardiographic response to exercise participate in this method of screening it will become increasingly important to recognise the potential mischief an incorrect interpretation can cause and the factors that may lead to both falsely negative and falsely positive results.

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3 Melin JA, Piret LJ, Vanbutsele RJM, *et al.* Diagnostic value of exercise electrocardiography and thallium myocardial scintigraphy in patients without previous myocardial infarction: A bayesian approach. *Circulation* 1981;**63**:1019–24.

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