Is it just only an innocent early repolarization electrocardiographic pattern?

¿ Será apenas un inocente patrón de repolarização precóz?

Será apenas um inocente padrão eletrocardiográfico de repolarização precoce?

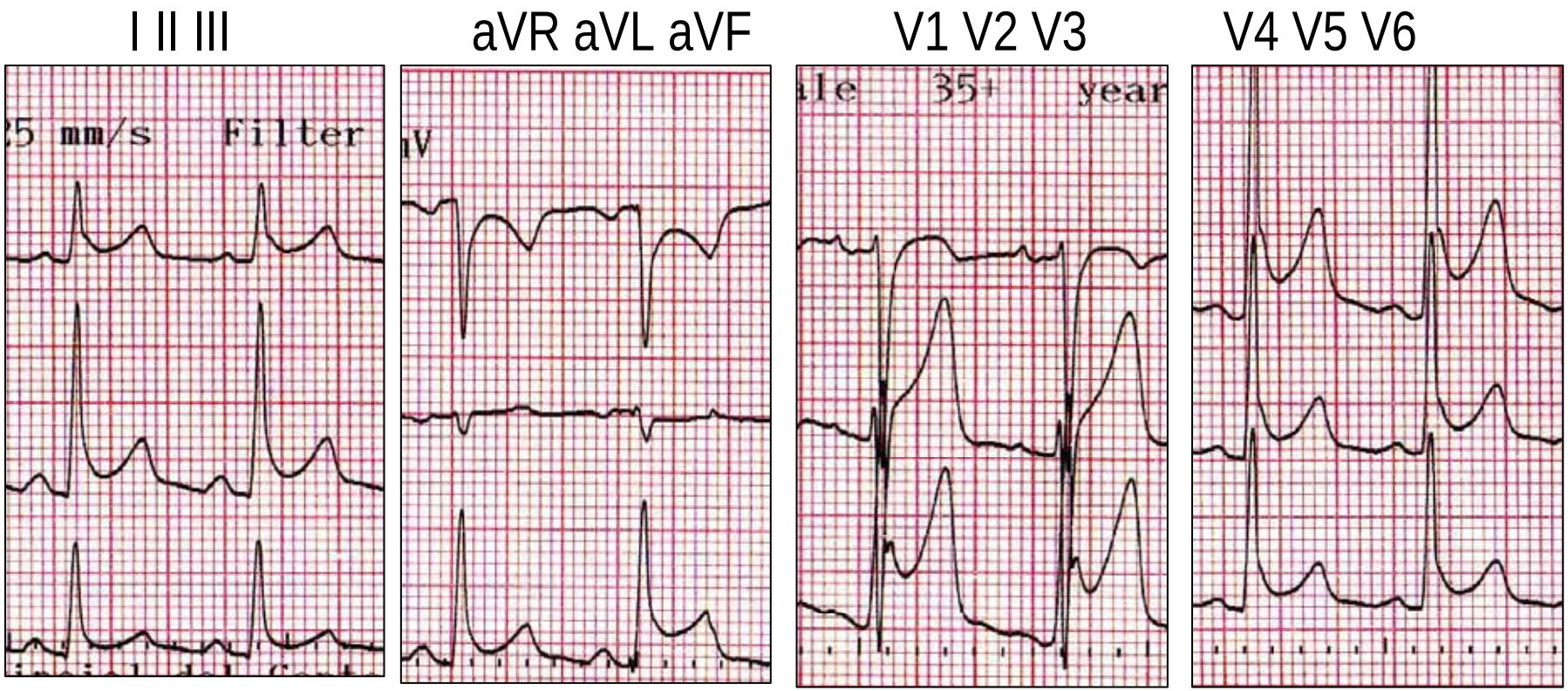
Case report from Oscar Pellizzon M.D. Argentine Republic
opeli@fibertel.com.ar

Masculino, 42 años con queja de angor de reposo de varias horas de evolución. Consumidor de cocaína. ECG sin cambios evolutivos.Troponina negativa. ECO 2D normal. Coronariografia sin lesiones.

1) ¿ Cual es el diagnóstico Electrocardiográfico? 2) **¿** Cual es la conducta apropiada a ser seguida?

Male, 42 years of age, with complaints of resting angina several hours earlier. Cocaine user. Negative ECG evolutive pattern. Normal Troponin. 2D ECO normal. Coronariography without coronary lesions.

1) What electrocardiographic diagnosis? 2) What is the appropriate course of action to be followed?



The ST elevation in Benign ER is usually < 2mm (but can rarely be > 5mm such this case) in the precordial leads and the greatest ST elevation is usually seen in the mid-to-left precordial leads; the ST segment elevation is usually < 0.5 mm in the limb leads

What is meant by negative evolution? Did the J waves persist?

Melvin Sheinman
scheinman@medicine.ucsf.edu

Estimado Profesor Melvin: Gracias por su pregunta. Quise decir que la patente electrocardiográfica sigue igual a la presentada a pesar del pasar de los dias. La onda J continua sin cambios despues de 48 hs del ingreso. Lo dejé unos dias internado, y el patrón electrocardigráfico sigue idéntico.

Dear Professor Melvin: Thank very much for your question. I meant that the ECG patent is equal to electrocardiographic presented despite the passing of the days. The J wave remains unchanged after 48 hours of admission. I left a few days hospitalized, and the electrocardiographic pattern remain identical.

OscarPellizon M.D..

The height of the J wave is worrisome but the concave upward pattern or fishhook sign points to a more benign assignment.

La altura de la onda J es preocupante pero el patrón del ST de concavidad superior o en anzuelo es una característica mas benigna.

Melvin

Maestro Andrés Ricardo Dare mi opinion del caso enviado por ud y el Dr Oscarcon lo que espero no sea muy descabellada mi opinion.

Es un electrocardiogram en ritmo sinusal con FC aproximada de 90latidos por minuto, donde se observa patron compatible con repolarización precoz, pero me impresionan ademas cambios observados en la pericarditis. Los segmentos PR están con tendencia a estar deprimidos. Existe elevación del segmento ST, difusa y cóncava en muchas derivaciones.

La ligera elevación del segmento PR en aVR, es sugestiva, pero no diagnóstica, de pericarditis aguda. Los cambios del segmento PR y ST son más pronunciados en DII, que en DIII; otro dato típico de pericarditis, comparado con lo que sucede en la cardiopatía isquémica.

Lo que desconozco es la relación que podria existir en un paciente cocainómano y si estos cambios electrocardiográficos

1. Spodick DH. Acute pericarditis: current concepts and practice. JAMA. 2003; 289: 1150-1153.

Abrazos

Jesus Antonio Chacón

Master Andrés Ricardo: I will give my opinion of the case submitted by Dr Oscar.

It is an electrocardiogram in sinus rhythm with HR ≈ 90lpm, showing early repolarization pattern but also impress me with changes observed in acute pericarditis: PR segments are prone to be depressed. There is diffuse ST segment elevation, and concave upward in many derivations. The slight PR segment elevation in aVR, is suggestive but not diagnostic of acute pericarditis. Changes PR and ST segment are more pronounced in II, III that in other leads. This data are typical of acute pericarditis, compared to what happens in ischemic heart disease. I dont know the relationship that may exist in a patient cocaine-user and its electrocardiographic changes

Querido Oscar: espero se encuentre Ud bien. Digale que el proximo pase de coca que se pegue, va ser el ultimo.
Hablando enserio desde cuando realmente no consume?
El patron de repolarizacion es de alto riesgo presenta empastamiento del final del QRS con mayor repolarizacion precoz en cara inferolateral mayor a 2 mm y acortamiento del QT. El problema es que pienso que es inducido por toxicos drogas y/o alcohol y comunmente en esta poblacion es comun la asociacion con otros psicofarmacos y estimulantes como las bebidas energizantes (speed). Mientras no abandone los mismos y sin arrtimias documentadas, el angor en el consumo de cocaina suele ocurrir por espasmo coronario.
No refirio antecedentes familares positivos y no encuentro criterios para sospechar un Brugada oculto y desconozco como respondera a pruebas medicamentosas o al EEF si ha consumido en las ultimas semanas, el patron de repolarizacion impresiona de alto riesgo, pero inducido por toxicos no espontaneo en mi criterio.
1. Desintoxicacion.
2. Prohibirle la actividad fisica temporariamente
3. No consumo de toxicos.
4. Se podria intentar si mejora el patron con amiodarona o bisoprolol en esto espero me corrijan si no piensan que puede mejorar con la utilizacion de estas.
5. Reevaluar el ECG y patron de repolarizacion luego de detoxificacion y si persisten pruebas farmacologicas y decidir conducta.
Un saludo querido Oscar
Martin barrola
Síndrome de repolarización precoz: un fenómeno electrocardiografico benigno o “no tan benigno”. Su relación con la muerte súbita cardiaca. Early repolarization and its relationship with sudden cardiac death. http://www.fac.org.ar/1/revista/12v41n1/art_revis/revis01/pellizon.phpOscar A. Pellizzón, Mario D. González.

Querido Martin

Si te interesa el tema ECG y Cocaina, recientemente publicamos una linda revisión sistemática este año(1). Buscalo, que creo te puede interesar

AB

Dear Martin If this topic is of your interests (Cocaine ECG finding), recently we publishied a nice systematic review (1).
AB

1. **Ramirez FD, Femenía F, Simpson CS, Redfearn DP, Michael KA, Baranchuk A. Electrocardiographic findings associated with cocaine use in humans: a systematic review. Expert Rev Cardiovasc Ther. 2012 Jan;10:105-127.**

Si el paciente niega sincope, historia familiar de muerte subita, palpitaciones y llego fue por angor con coronarias normales, yo le haria una prueba de esfuerzo, potenciales tardios ventriculares y holter antes de ser egresado, no le daria b bloqueador ni amiodarona, el agonismo b adrenergico mas bien hace desaparecer los cambios del ecg en mi experencia en este tipo de paciente, puede ser visto en la prueba de esfuerzo, en caso de que todos estos estudios sean normales alta, con las recomendaciones de martin y que acuda a un centro de rehabilitacion, si los potenciales tardios resultan positivos se enredaria el asunto porque tampoco hay nada de evidencias totalmente confirmadas, es un tema donde estamos aprendiendo a mi juicio.

Kako

Hola Kako

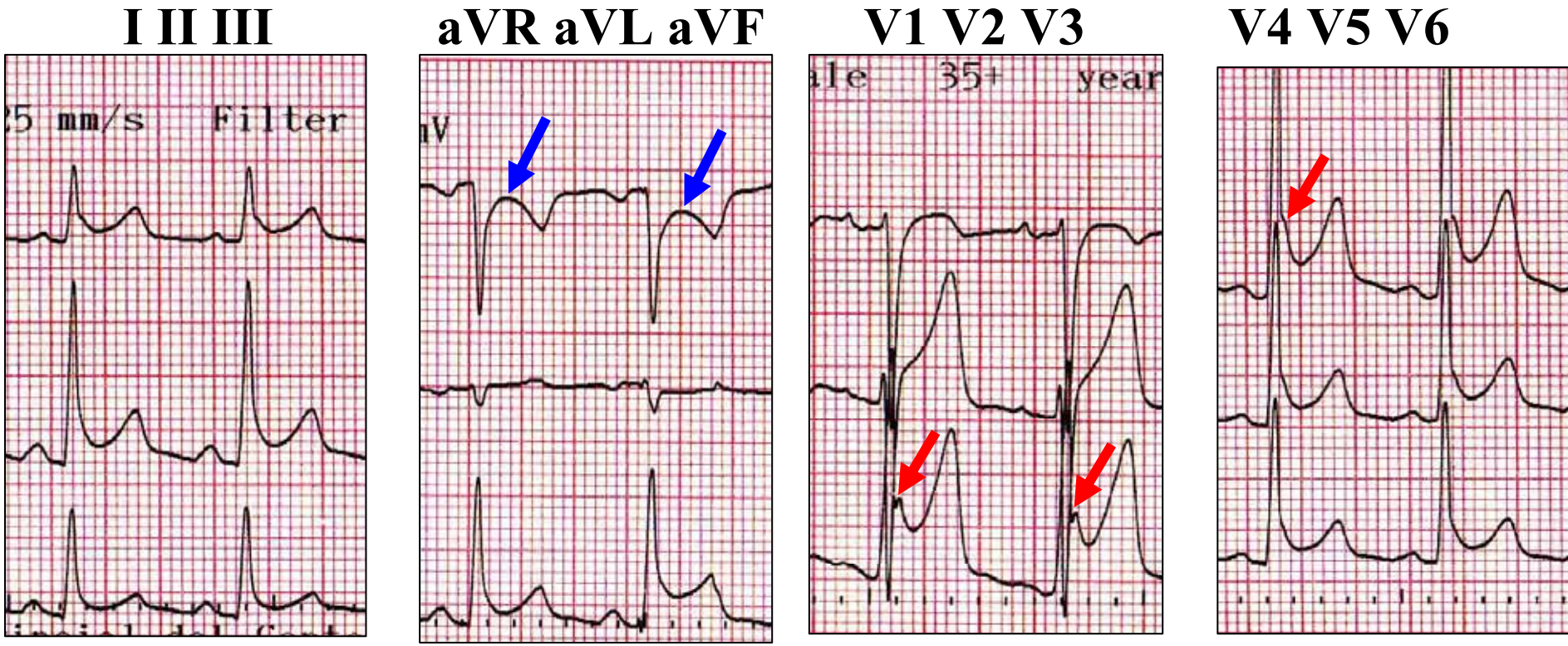
No segui el caso pero me llamo la atencion tu frase final: si los potenciales dan positivos, se enredaria la cosa, dices tu...Y yo pienso: si dan negativos, no me dice nada, porque el valor predictivo negativo es muy bajo; y si me da positivo, como dices tu, se enredaria la cosa...y yo me pregunto (sobre este caso y tantos otros!!!) para que le pedimos estudios a los pacientes si en cualquiera de las 2 posibilidades (positivo / negativo) no va a cambiar el manejo del paciente?

Digo, para pensar cuantas cosas hacemos (estudios, manejos, etc) que objetivamente NO nos llevan a ningun sitio.
Recibe un abrazo y espero verte pronto en Baires

AB

Final comments

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



Sinus rhythm, HR 94lpm, P axis +63º and to front in HP, P duration 100ms, PR duration 160ms, frontal QRS axis + 63º; ST segment axis +60º and frontal T wave axis +55º: The frontal plane QRS axis and ST segment axis and T wave axis are all in the same direction in benign early repolarization such as this case. A rapid QRS transition is noted from V2 to V3. The ST elevation in Benign ER is usually < 2mm (but can rarely be > 5mm In this case is 4,7mm) in the precordial leads and the greatest ST elevation is seen in the mid-to-left precordial leads; upward concavity of the initial portion of the upsloping ST segment is observed, the ST segment elevation is < 0.5 mm in the limb leads.

Mirror image is confined to aVR lead (blue arrows).

Notching or slurring is noted at terminal portion of the QRS complex (red arrow) this is the J point (from V3 to V4). Symmetric, concordant T waves are observed.

In the present case a relative temporal stability of the ST segment and T wave pattern was observed and Q waves dint not appear

Electrocardiographic criteria that suggest Benign Early Repolarization (BER)

1. *Sinus bradycardia is often present*
2. *The frontal plane QRS axis and ST segment axis and T wave axis are all in the same direction*
3. *Minimally elevated J point*
4. *Upward concavity of the initial portion of the upsloping ST segment*
5. *Notching or slurring of the terminal QRS complex (J point)*
6. *Symmetric, concordant T waves of large amplitude - the T waves may appear "peaked" or pointed*
7. *Elevated ST segments are most commonly seen in the mid-to-left precordial leads, and they are also sometimes seen in both the limb leads (I, II, II, aVF and aVL) + chest leads (V2 - 6) with the degree of precordial lead ST segment elevation > limb lead ST segment elevation*
8. *The ST elevation in BER is usually < 2mm (but can rarely be > 5mm) in the precordial leads and the greatest ST elevation is usually seen in the mid-to-left precordial leads; the ST segment elevation is usually < 0.5 mm in the limb leads*
9. *Strongly consider an inferior AMI if the elevated segments are only seen in the inferior leads - II, III and aVF, and a lateral AMI if the ST segment elevations are only seen in leads I and aVL)*
10. *Associated R waves are usually tall*
11. *Prominent, relatively deep but narrow, q waves may appear in the left precordial leads*
12. *A rapid transition may occur from right oriented complexes to left oriented complexes in the precordial leads*
13. *Reduction in ST segment elevation may occur secondary to sympathomimetic influences The ST segment elevation is reversed with isoproterenol.*
14. *Mirror image is possible only in a VR*
15. *Relative temporal stability of the ST segment and T wave pattern*
16. *There are no evolutionary short-term changes in the ST segment and T waves; and Q waves do not appear*
17. *BER is seen more commonly in young/middle-aged adult males (mainly in athletes)and the magnitude of the BER may diminish as the patient ages (20 - 30% of cases)*

Main features in Benign Early Repolarization (BER) (1;3)

1. *Widespread ST segment elevation (STSE) (precordial greater than limb leads)*
2. *J-point elevation*
3. Upward concavity of the initial portion of the ST segment,
4. *Notching of the terminal QRS complex*
5. *Concordant T waves of large amplitude*
6. *Stable ECG pattern*
7. *Negative cardiac injury markers*
8. *Normal cardiac stress test or angiography.*

Main AMI criteria were

1) Regional STE
2) Positive cardiac injury markers: abnormal serum troponin I values (>0.1 mg/dL) followed by a rise and fall of the serum marker;(3)
3) Identification of culprit coronary artery by angiography in less than eight hours of presentation.
4) Typical evolution of ST-T wave(3)

1. Turnipseed SD, Bair AE, Kirk JD, et al. Electrocardiogram differentiation of benign early repolarization versus acute myocardial infarction by emergency physicians and cardiologists. Acad Emerg Med. 2006 Sep;13:961-966.
2. Engelmann MD, Hasbak P. Early repolarization. Differential diagnosis of electrocardiographic ST segment elevation. Ugeskr Laeger. 2000 Oct 30;162:5914-5917.
3. Brady WJ, Perron AD, Ullman EA, et al. Electrocardiographic ST segment elevation: a comparison of AMI and non-AMI ECG syndromes. Am J Emerg Med. 2002 Nov;20:609-612.
4. Snider RL, Pai RK, Kusumoto FM. The importance of the evolution of ST-T wave changes for differentiating acute pericarditis from myocardial ischemia. Cardiol Rev. 2004 May-Jun;12:138-140.

The potential misdiagnosis of pericarditis for acute MI has led to unfortunate complications when thrombolytic therapy has been given.

Some ECG findings that may be helpful include the following:

1. Repolarization does not progress through stages and is uncommonly associated with PR depression.
2. Serial monitoring of ECGs in young patients with chest pain helps differentiate ERP from acute pericarditis.
3. An ST-segment–to–T-wave ratio ≥0.25 in V6 can distinguish acute pericarditis from ERP.
4. The ST segment in acute MI is usually convex, bowing upward with reciprocal changes, as opposed to concave ST segments without reciprocal changes observed in acute pericarditis and only in aVR in ERP.

ECG Differential Diagnosis Among Acute Pericarditis, Acute Myocardial Infarction And ERP (1)

	Acute pericarditis	**Acute Myocardial infarction**	**ERP**
PR-segment depression	Present	Only when associated with atrial infarction	Absent
Lost of R-wave voltage	Absent	Present	Absent. *Associated R waves are usually tall*
Q waves duration	<40ms	≥40ms	<40ms *Q waves do not appear*
ST-segment shape	Concave upward	Convex upward	*Concavity of initial up-sloping portion of ST segment*
Reciprocal ST-segment changes	Absent	Present	Possible only in aVR
Location of ST segment elevation number of leads with ST elevation	Diffuse	Segmental/Regional	*Most commonly seen in the mid-to-left precordial leads. Usually < 0.5 mm in the limb leads*
ST/T ratio in lead V6	>0.25	not applicable	<0.25

1. **Modified from Marinella MA. Electrocardiographic Manifestations and Differential Diagnosis of Acute Pericarditis. Am Fam Physician. 1998 Feb 15;57:699-704.**

The ratio of the amplitude of ST segment to the amplitude of the T wave in leads I, V4, V5 and V6 proved to be a significant discriminator at a value of ≥0.25 Leads I, V4, V5 and V6 can all be used to differentiate acute pericarditis from early repolarization pattern and early repolarization pattern associated with LVH. When ST elevation is present in lead I, the ST/T ratio has the best predictive value (0.82) to more accurately discriminate between acute pericarditis from early repolarization pattern and early repolarization pattern associated with LVH.(1)

R-wave amplitude is lower, ST-segment elevation greater, and QTc longer for subtle anterior STEMI versus ERP. In combination with other clinical data, this derived and validated ECG equation could be an important adjunct in the diagnosis of anterior STEMI.(2)

1. **Bhardwaj R, Berzingi C, Miller C, et al. Differential Diagnosis of Acute Pericarditis from Normal Variant Early Repolarization and Left Ventricular Hypertrophy with Early Repolarization: An Electrocardiographic Study. Am J Med Sci. 2012 Jul 17. [Epub ahead of print]**
2. **Smith SW, Khalil A, Henry TD, et al. Electrocardiographic differentiation of** early repolarization **from subtle anterior ST-segment elevation myocardial infarction. Ann Emerg Med.2012 Jul;60:45-56.e2.**

The latest American College of Cardiology/American Heart Association guidelines for the treatment of STEMI emphasize that the physician at the ED should make reperfusion decisions within 10 minutes of performing the initial ECG. STE in ≥2 adjacent ECG leads should receive immediate reperfusion therapy. However, not all ECGs with STE necessarily reflect transmural infarction from acute thrombotic occlusion of an epicardial coronary artery, as a large number of patients presenting with compatible symptoms have baseline STE. Novel strategies aimed to reduce door-to-balloon time, such as prehospital wireless ECG transmission, may be dependent on the interpretation accuracy of the ECG readers.(1) In some cases a pattern of benign nonischemic STE (NISTE) can be recognized fairly easily. Other times, differentiating between true STEMI and NISTE may be difficult. Patients presenting with chest pain and showing benign pattern of NISTE (eg, ERP or STE secondary to LVH) may have true ischemic pain and non-STEMI or even STEMI on top of the baseline benign pattern. The ability of physicians to differentiate NISTE from STEMI based on the presenting ECG pattern widely varies and depends on the prevalence of baseline NISTE in the patient population. Further studies are needed to assess the ability of various ECG criteria to accurately differentiate between STEMI and NISTE.(2)

The information obtained from the ECG at presentation should be complemented by serial ECGs especially during symptoms indicative of ischemia and, if applicable, by comparing the findings with reference ECGs. Continuous ECG recording in a coronary care setting, including the comparison of ECGs with and without pain, adds to the information gained at patient presentation.(3)

1. **Jayroe JB, Spodick DH, Nikus K, et al. Differentiating ST elevation myocardial infarction and nonischemic causes of ST elevation by analyzing the presenting electrocardiogram. Am J Cardiol. 2009 Feb 1;103(3):301-306.**
2. **Huang HD, Birnbaum Y. ST elevation: differentiation between ST elevation myocardial infarction and nonischemic ST elevation.J Electrocardiol.2011 Sep-Oct;44:494.e1-494.e12.**
3. **Nikus K, Pahlm O, Wagner G, et al. Electrocardiographic classification of acute coronary syndromes: a review by a committee of the International Society for Holter and Non-Invasive Electrocardiology. J Electrocardiol. 2010 Mar-Apr;43:91-103**

Left precordial terminal QRS notching is more prevalent in malignant variants of ER than in benign cases. These findings could have important implications for risk stratification of patients with ER.(1) In the present case notching is more conspicuous in V3-V4.

In the great majority of cases the ECG pattern of ERP is a benign phenomenon observed predominantly in teenagers, young adults, male athletes and the black race.(2)

The ERP is common and associated with risk of SCD ERP is heritable, and mutations have been described in syndromatic cases. In a genome-wide association study, Sinner et al (3) were not able to reliably identify genetic variants predisposing to ERP, presumably due to insufficient statistical power and phenotype heterogeneity. The reported heritability of ERP warrants continued investigation in larger well-phenotyped populations.

1. **Merchant FM, Noseworthy PA, Weiner RB, Singh SM, Ruskin JN, Reddy VY. Ability of terminal QRS notching to distinguish benign from malignant electrocardiographic forms of early repolarization. Am J Cardiol. 2009 Nov 15; 104:1402-1406.**
2. **Pérez-Riera AR, Abreu LC, Yanowitz F, Barros RB, Femenía F, McIntyre WF, Baranchuk A. "Benign" early repolarization versus malignant early abnormalities: clinical-electrocardiographic distinction and genetic basis. Cardiol J. 2012;19:337-346.**
3. **Sinner MF, Porthan K, Noseworthy PA, et al. A meta-analysis of genome-wide association studies of the electrocardiographic early repolarization pattern. Heart Rhythm. 2012 Oct;9:1627-1634.**

Cocaine

Cocaine remains the most common cause of illicit drug-related visits to emergency departments, 40% of which result from chest pain. It is estimated that over half of the 64,000 patients evaluated annually for cocaine-associated chest pain will be admitted to hospitals for the evaluation of myocardial ischemia or infarction, at a health care cost of over eighty million dollars. Although the link between cocaine use and myocardial ischemia is well established, only about 6% of patients with cocaine-associated chest pain will demonstrate biochemical evidence of myocardial infarction.

Patients who have chest pain following the use of cocaine have become more common in EDs throughout the United States, with approximately 6% of these patients sustaining an AMI. Myocardial infarction in patients who have cocaine-associated chest pain is not uncommon.

No clinical parameter available to the physician can adequately identify patients at very low risk for MI. Therefore, all patients with cocaine-associated chest pain should be evaluated for MI.(1)

Patients with cocaine-associated chest pain who do not have evidence of ischemia or cardiovascular complications over a 9-to-12-hour period in a chest-pain observation unit have a very low risk of death or AMI during the 30 days after discharge.

1. **Hollander JE, Hoffman RS, Gennis P, et al. Prospective multicenter evaluation of cocaine-associated chest pain. Cocaine Associated Chest Pain (COCHPA) Study Group.Acad Emerg Med. 1994 Jul-Aug;1:330-339.**
2. **Weber JE, Shofer FS, Larkin GL, et al. Validation of a brief observation period for patients with cocaine-associated chest pain. N Engl J Med. 2003 Feb 6;348:510-517.**

Patients who have used cocaine may present with chest pain even in the absence of clear cardiac risk factors. An abnormal ECG has been reported in 56% to 84% of patients with cocaine-associated chest pain; however, many of these patients are young and commonly have the normal variant of early repolarization pattern (ERP), which may be interpreted by physicians as an abnormal ECG finding.(1)

Gitter and colleagues(2) reported an ERP in 32% of patients with cocaine-associated chest pain, a LVH pattern in 16%, and a normal ECG in only 32% of patients. Overall, 42% of patients in their cohort of 101 patients manifested ECG ST-segment elevation, although all of them eventually had MI excluded by cardiac marker testing.

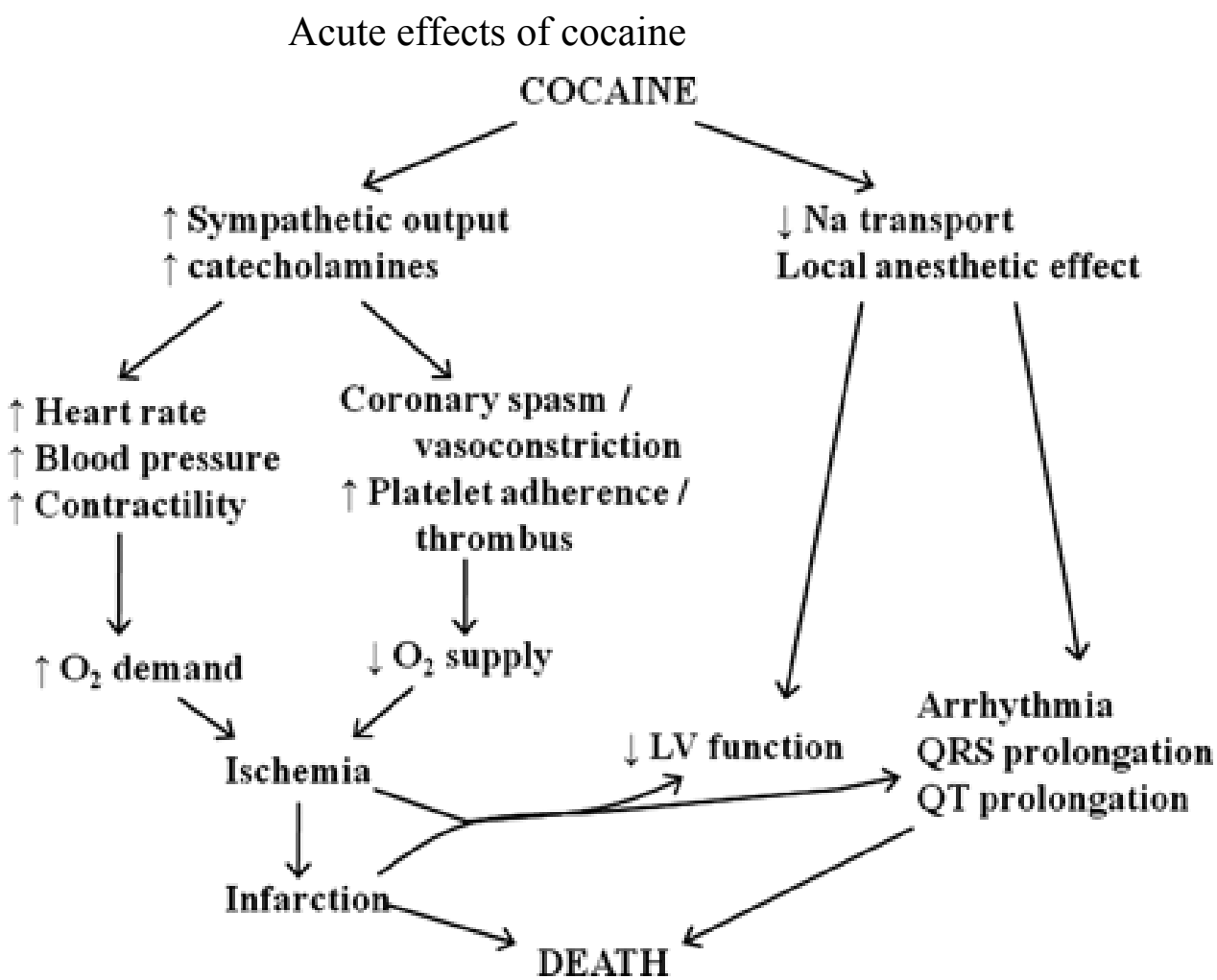
In the COCHPA study, the sensitivity of an ECG revealing ischemia or MI to predict a true MI was only 36%.(3) The specificity, positive predictive value, and negative predictive value of the ECG were 89.9%, 17.9%, and 95.8%, respectively.(3)

In a series of 238 patients with chest pain after cocaine use, 33% had normal ECGs, 23% had nonspecific changes, 13% had a LVH pattern, 6% had LVH and ERPs, and 13% had ERP only. ECG findings specific for ischemia or MI were present in only a minority of patients; 2% had changes typical for STEMI and 6% had changes specific for acute ischemia.(4)

1. ***Forrester JM, Steele AW, Waldron JA, et al. Crack lung: an acute pulmonary syndrome with a spectrum of clinical and histopathologic findings. Am Rev Respir Dis. 1990 Aug;142:462-467.***
2. ***Gitter MJ, Goldsmith SR, Dunbar DN, et al. Cocaine and chest pain: clinical features and outcome of patients hospitalized to rule out myocardial infarction. Ann Intern Med. 1991 Aug 15;115:277-282.***
3. ***Hollander JE, Hoffman RS, Gennis P, et al. Prospective multicenter evaluation of cocaine-associated chest pain. Cocaine Associated Chest Pain (COCHPA) Study Group.Acad Emerg Med. 1994 Jul-Aug;1:330-9.***
4. ***Kontos MC, Schmidt KL, Nicholson CS, et al. Myocardial perfusion imaging with technetium-99m sestamibi in patients with cocaine-associated chest pain. Ann Emerg Med. 1999 Jun;33:639-645.***

Cocaine is an alkaloid prepared from the leaves of the plant *Erythroxylon coca*. The crystalline or powder form of cocaine is prepared by dissolving the alkaloid in hydrochloric acid to form the water-soluble salt cocaine hydrochloride. "Freebase" or "crack" cocaine is the cocaine alkaloid in its basic, non-salt form and is prepared by organic extraction from a basic solution with ether. Crack cocaine melts at high temperature and vaporises at even higher temperatures without losing any of its potency, thus allowing it to be smoked.(1) When inhaled, cocaine crosses the alveolar endothelium and is rapidly absorbed into the bloodstream. In fact, cocaine is rapidly absorbed from all mucous membranes, allowing it to be administered sublingually, intravaginally, rectally and through the respiratory system, as well as by intramuscular or intravenous injection. Onset of action generally varies between three seconds and five minutes, depending on the route of administration. Peak effect is usually reached within 20 minutes and the duration of action is also variable, ranging from five to 90 minutes. Cocaine has a plasma half-life of 30–60 minutes in humans.(2) The drug is metabolised by plasma and hepatic cholinesterases to the water-soluble compounds benzoylecgonine and ecgonine methyl ester, which are excreted in the urine, thus enabling detection by urinary drug screening for these compounds.(2) The primary mechanism of action underlying cocaine's central and peripheral effects is blockade of norepinephrine, serotonin and dopamine reuptake into the presynaptic terminals from which these transmitters are released.(2) This blockade potentiates and prolongs the central and peripheral actions of these catecholamines. In particular, prolongation of dopaminergic effects in the brain's "pleasure centre" (the limbic system) produces the intense euphoria that cocaine initially causes.(2) Chronic intake of cocaine depletes dopamine, leading to the intense depression experienced and described by cocaine addicts.(2) This depletion triggers a cycle of craving for cocaine and temporary relief of depression by further cocaine ingestion.

1. Lange RA, Hillis LD. Cardiovascular complications of cocaine use. *N Engl J Med* 2001; 345: 351-357.
2. Zimmerman FH, Gustafson GM, Kemp HG Jr. Recurrent myocardial infarction associated with cocaine abuse in a young male with normal coronary arteries: evidence for coronary artery spasm culminating in thrombosis. *J Am Coll Cardiol* 1987; 9: 964-968.



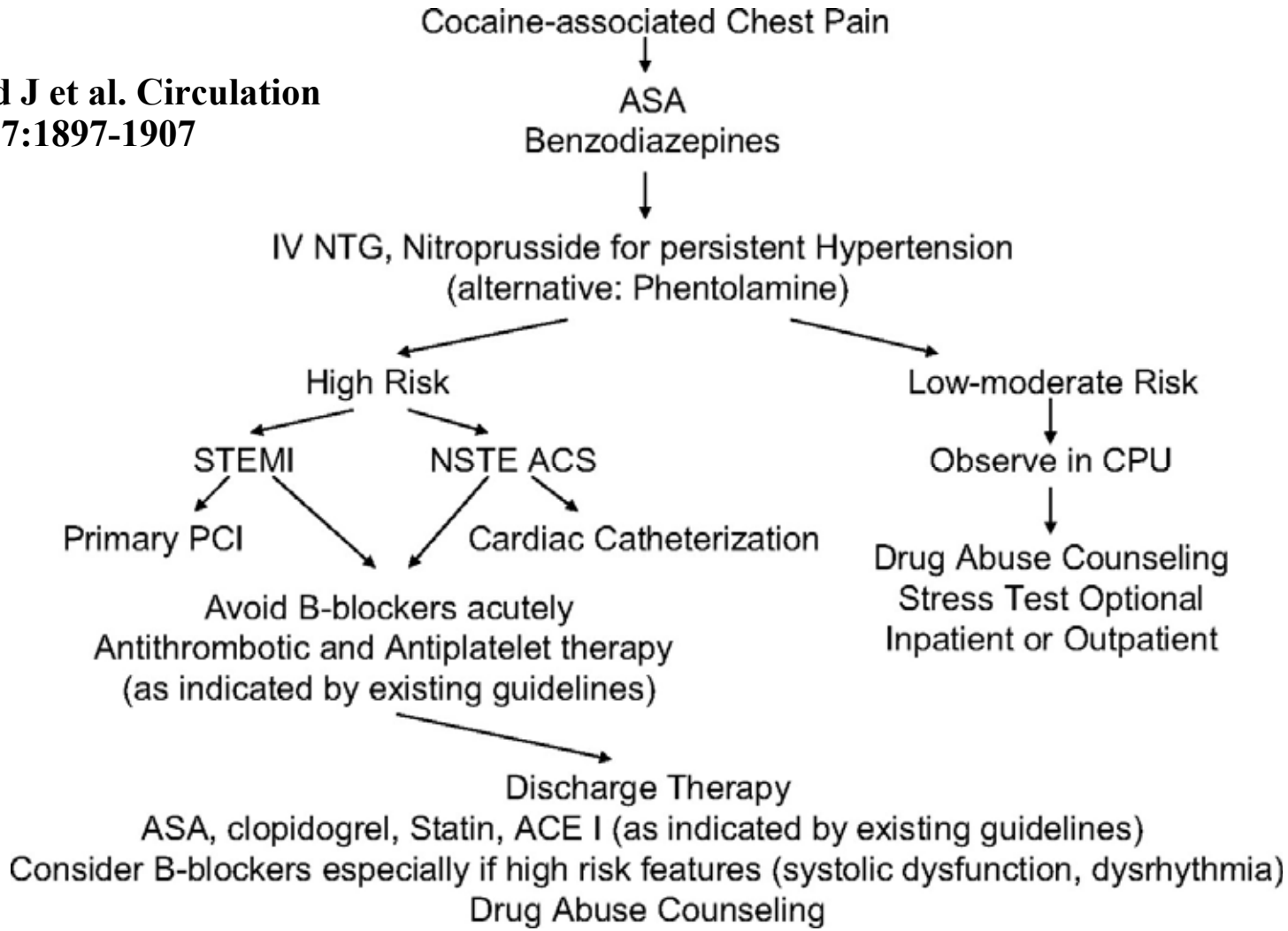
Cocaine affects the cardiovascular system through 2 major pathways: increased sympathetic output and a local anesthetic effect. Through increased sympathetic tone and catecholamine levels, cocaine increases heart rate, blood pressure, and myocardial contractility, all of which increase myocardial oxygen demand. Myocardial oxygen supply is decreased through coronary vasoconstriction and enhanced thrombosis. Myocardial oxygen demand may exceed myocardial oxygen supply, leading to ischemia or infarction. Cocaine affects cardiac myocytes directly by blocking sodium channels, which decreases LV contractility and is arrhythmogenic.

Cardiovascular Effects of Cocaine

Causes Myocardial Oxygen Supply-Demand Mismatch	Worsens Myocardial Performance	Causes Cardiovascular Disease	Causes Clinical Cardiovascular End Points
Increases heart rate	Decreases ejection fraction	Arrhythmias	Myocardial infarction
Increases blood pressure	Increases end-systolic volume	QT prolongation	Arrhythmias
Decreases coronary artery diameter	Increases end-diastolic pressure	Thrombosis	Congestive heart failure
Decreases coronary blood flow	Lengthens deceleration time	Atherosclerosis	Cardiomyopathy
	Increases left ventricular hypertrophy	Endothelial dysfunction	Aortic dissection
		Microvascular disease	Endocarditis
			Sudden death

Therapeutic and diagnostic recommendations in cocaine-associated chest pain

McCord J et al. Circulation 2008;117:1897-1907



Therapeutic and diagnostic recommendations in cocaine-associated chest pain. ASA indicates aspirin; NTG, nitroglycerin; STEMI, ST-segment–elevation MI; NSTE ACS, non–ST-segment–elevation ACS; CPU, chest pain unit; PCI, percutaneous coronary intervention; B-blockers, β-blockers; and ACE, angiotensin-converting enzyme inhibitor.