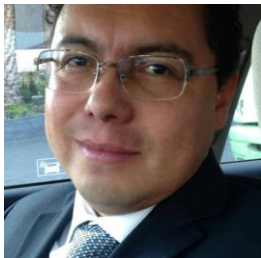




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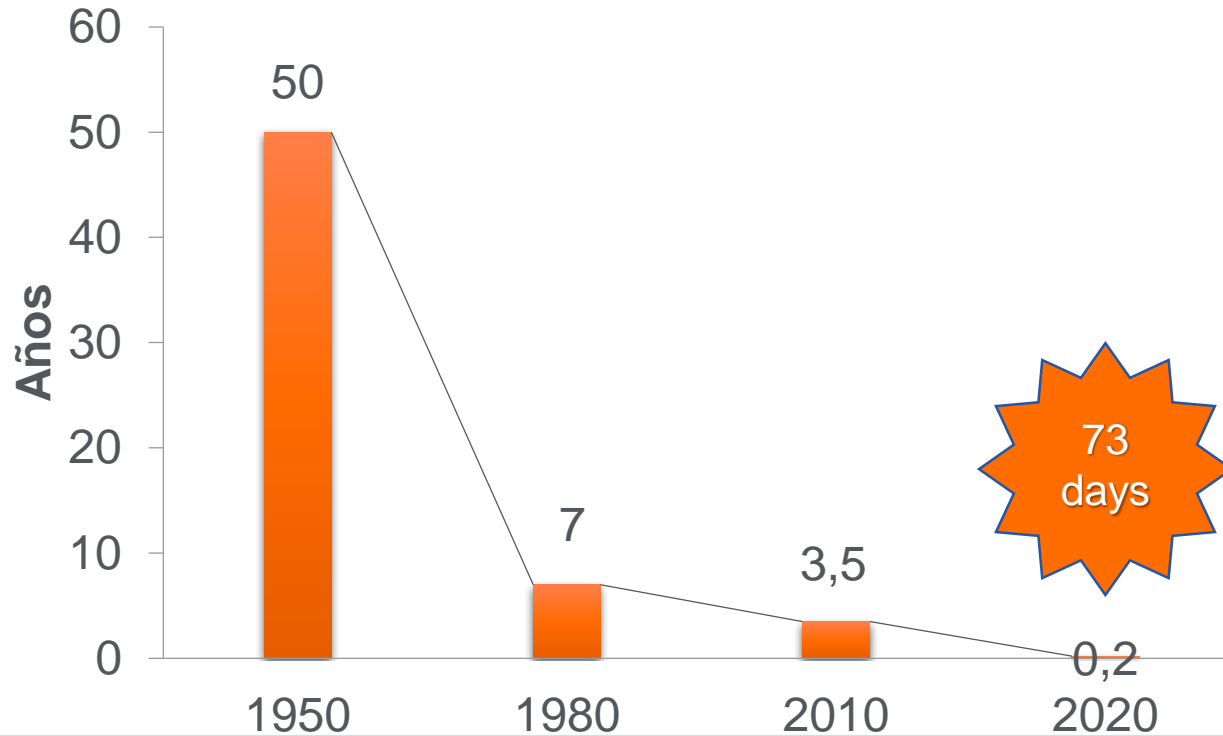
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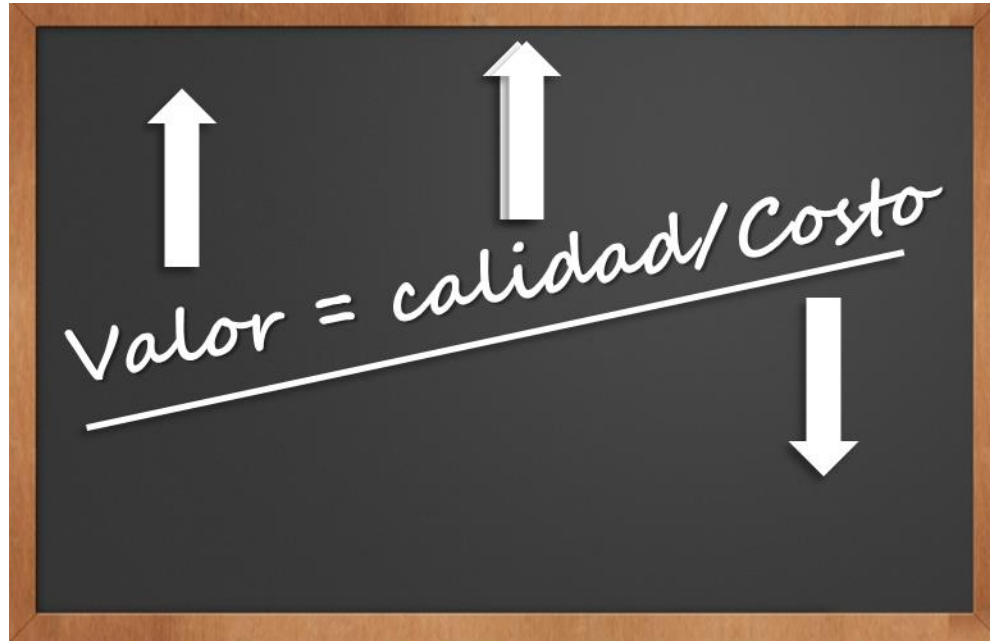
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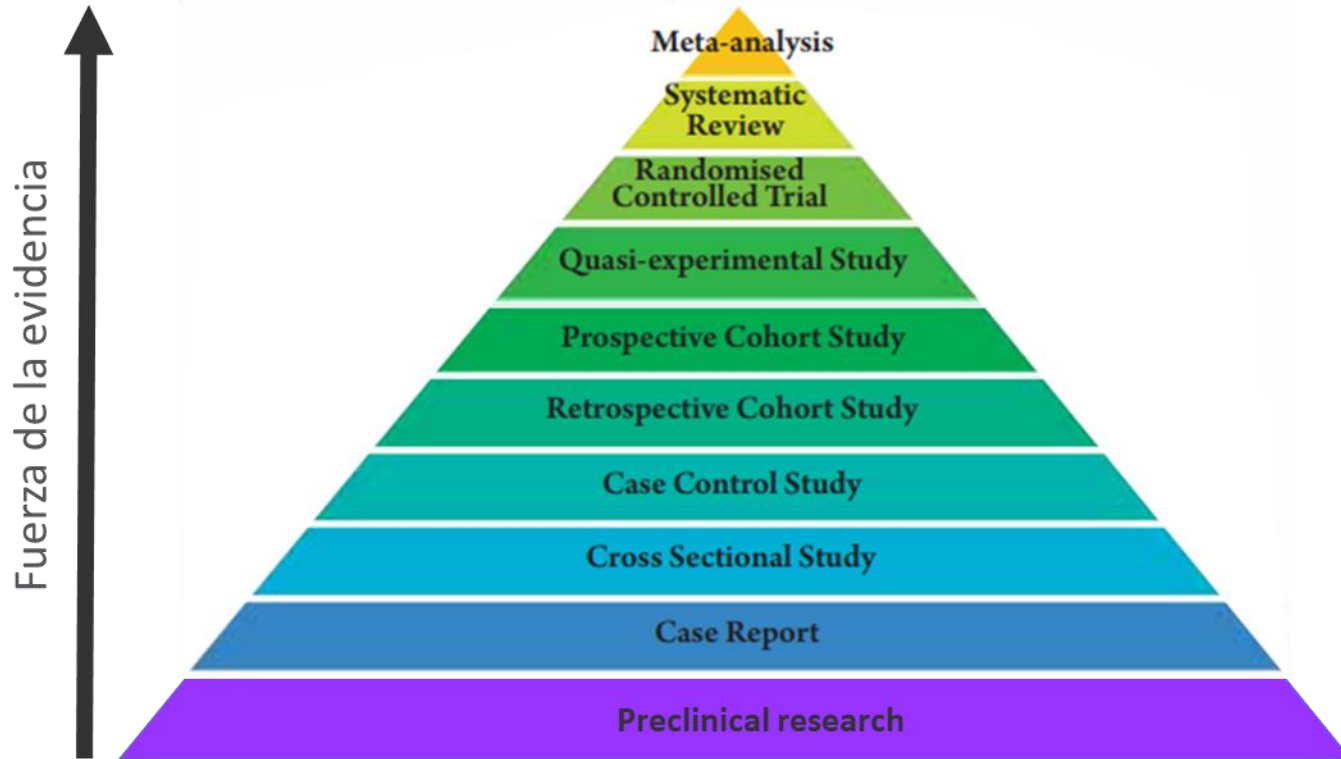
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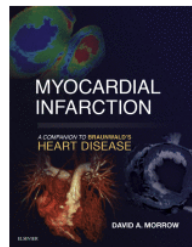
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Acute myocardial infarction (MI) is a leading cause of morbidity and mortality worldwide (see [Chapter 2](#)). ¹ Although an estimated 157,000 patients will die each year in the United States as a result of an MI, this statistic belies the wide variability in the rate of mortality and recurrent ischemic events across the spectrum of patients with MI. Although patients presenting with ST-elevation MI (STEMI) are at higher risk for short-term mortality, patients with non-ST-elevation MI (NSTEMI) are at higher long-term risk, which is believed to be related to older age and comorbid medical conditions.

Risk stratification is an integral component of the management of patients presenting with acute MI. Prognostic information is important for appropriate triage and resource allocation to provide the appropriate intensity and location of care for MI patients. Patients and their families, when provided with information about its severity and anticipated consequences.



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Risk Stratification in Acute Myocardial Infarction



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Individualized risk assessment for acute MI involves the integration of multiple data points that are available at first medical contact and initially is composed of baseline demographic and clinical characteristics. Upon presentation, additional information gleaned during the initial evaluation, including physical examination findings, the electrocardiogram (ECG), and biomarkers of cardiomyocyte necrosis, is integrated. These data may then be combined into a validated risk model, such as the GRACE (Global Registry of Acute Coronary Events) Risk Score or the TIMI (Thrombolysis In Myocardial Infarction) Risk Score, to provide clear prognostic guidance on short- and long-term risks of death or major adverse cardiovascular events. Such risk scores may be com-



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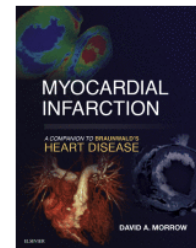
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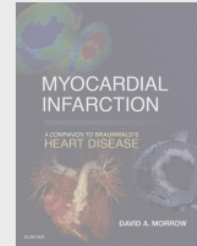
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ARTÍCULO

2017 AHA/ACC/HRS Guideline for Management of Patients With Ventricular Arrhythmias and the...

Artículo en prensa: Manuscrito aceptado

Heart Rhythm.
Al-Khatib, Sana M., MD, MHS, FACC, FAHA, FHRS; Stevenson, William G., MD, FACC, FAHA, FHRS...
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The diagnosis of AMI is established on the basis of a high clinical suspicion based on the history and physical examination findings in addition to changes in cardiac biomarkers (creatinine kinase–MB [CK-MB],... [Más](#)

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Myocardial infarction (agute): Early rule out using high-sensitivity troponin tests (Elecsys Troponin...

National Institute for Health and Care Excellence (NICE). Publicado October 1, 2014.

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Síndrome coronario agudo sin elevación del segmento ST

Alfredo Barrio Rodríguez; Xacobe Flores Ríos; José Manuel Vázquez Rodríguez. Fisterra. Publicado September 26, 2017. © 2014.

Búsquedas relacionadas con iam

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Acute myocardial infarction

Roxana Mehran, MD, Professor of Medicine (Cardiology) and Director of Interventional Cardiovascular Research and Clinical Trials, Zena and Michael A. Wiener Cardiovascular Institute, Mount Sinai School of Medicine, New York, New York; Kleanthis N. Theodoropoulos, MD, PhD, Interventional Cardiology Research Fellow, Mount Sinai Medical Center, New York, New York. Publicado March 13, 2014. Última actualización March 11, 2014.

 GUÍA CLÍNICA

Myocardial infarction (acute): Early rule out using high-sensitivity troponin tests (Elesys Troponin...

National Institute for Health and Care Excellence (NICE). Publicado October 1, 2014.

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Resumen de la enfermedad

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Key points

The estimated annual incidence of acute myocardial infarction (AMI), including both ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI), in the U.S. is 600,000 new and 320,000 recurrent attacks. In 2004, AMI resulted in 695,000 hospital stays and \$31 billion in hospital charges

Patients with AMI usually present with sudden or gradual onset of discomfort in the anterior chest, which also may be described as heaviness, pressure, or gas

Some patients, particularly women, patients with diabetes, and elderly patients, may have atypical symptoms, such as nausea or worsening glucose control, whereas others may have no symptoms ('silent myocardial infarction'). A high level of suspicion is required in these patient groups

The diagnosis of AMI is established on the basis of a

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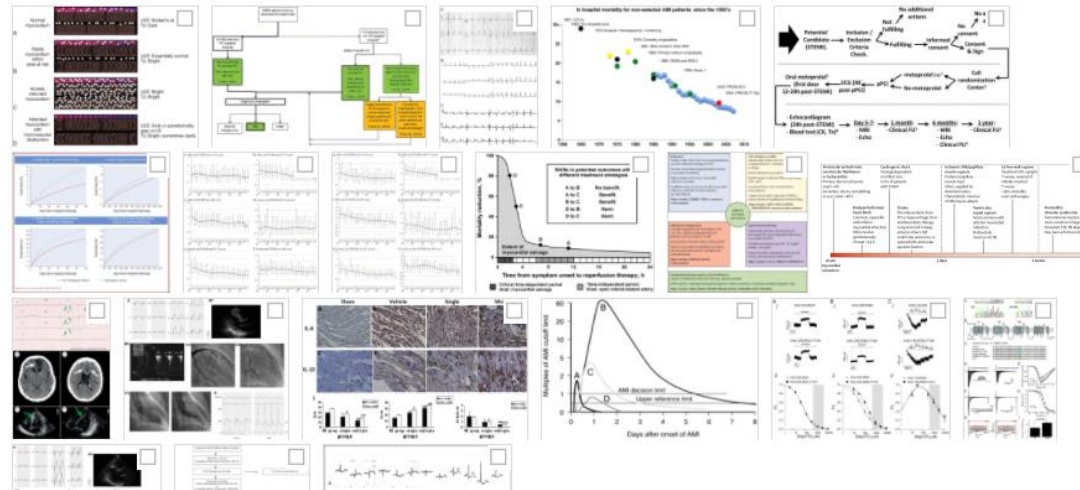
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Añadir a una presentación

A		Normal myocardium LGE: Nulled to define normal T2: Dark
B		Viable myocardium within area at risk LGE: Essentially normal T2: Bright
C		Acutely infarcted myocardium LGE: Bright T2: Bright
D		Infarcted myocardium with microvascular obstruction LGE: Dark or paradoxically gray on IR T2: Bright (sometimes dark)

make normal myocardium appear uniformly dark (nulled). B, Viable myocardium within the area at risk has intact cell membranes and therefore excludes gadolinium contrast agents from the intracellular space. However, some aspect of the ischemic period reversibly damaged this part of the heart enough to raise the water content and thus lengthen the T2 of the tissue. Therefore, the area at risk is brighter than normal myocardium on T2-weighted images. Because LGE images are essentially normal, there must be relatively balanced increases in the intracellular and extracellular volumes. Thus, the myocardium appears essentially normal on LGE CMR. C, Acutely infarcted myocardium is characterized by loss of cell membrane integrity. Hence, gadolinium contrast agents rapidly enter not only the extracellular space but also what used to be the intracellular space as long as there has been adequate reperfusion. As indicated by the large number of white crosses, acutely infarcted

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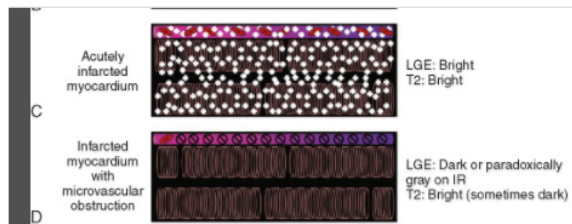
Acute Myocardial Infarction : Cardiovascular Magnetic Resonance Detection and Characterization

Cardiovascular Magnetic Resonance.
 Arai, Andrew E., Publicado January 1, 2010. Páginas 241-252. © 2010.
 Figure 18-2 In the setting of acute myocardial infarction, different types of myocardium have distinctive appearances on late gadolinium enhancement (LGE) images and T2-weighted images. A, Normal myocardium outside the ischemic area at risk is characterized by healthy cardiomyocytes (striped cylinders) with intact cell membranes and a characteristic intracellular to extracellular ratio and blood volume. The extracellular gadolinium contrast agents (white crosses) arrive via the bloodstream and rapidly enter the interstitial space but are excluded from the intracellular space. Thus, normal myocardium has a shorter T1 after contrast administration, but the amount of enhancement is modest, and the inversion recovery time can be adjusted to

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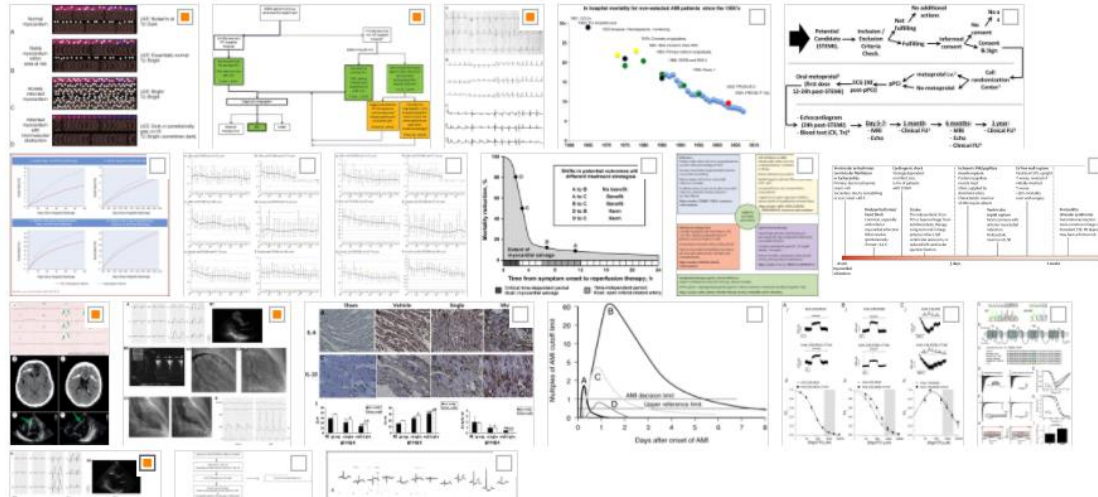
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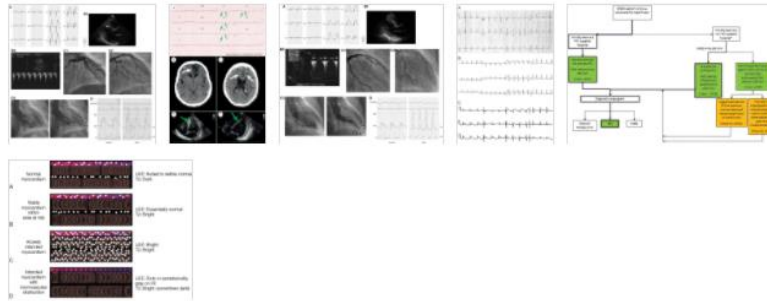
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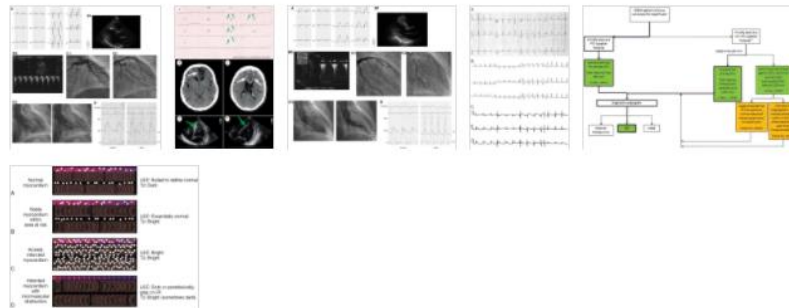
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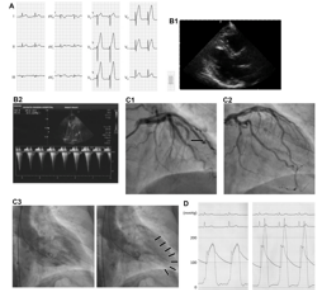
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(A) Electrocardiogram upon admission in Case 1. ST elevation was shown in the precordial leads. (B1) Transesophageal echocardiography findings upon admission in Case 1. Parasternal long-axis view of two-dimensional echocardiography showed a sigmoid-shaped septum with diminished aorto-septal angle of 89°. There were no indications of left ventricular hypertrophy. (B2) Spectral imaging of continuous wave Doppler of the left ventricular outflow tract from the apical position revealed the peak velocity value of 3.92 m/s indicating a peak gradient of 61 mmHg. (C) The findings of coronary angiography (CAG) in the right anterior oblique (RAO) cranial view (1), RAO view (2), and left ventriculography (LVG) in the RAO view in Case 1. Emergent CAG revealed one-vessel coronary heart disease with total occlusion of the mid left anterior descending artery (LAD) (arrow) and blood flow maintained in the major septal branch (1). A metal stent was implanted into the occluded lesion and recanalization of the LAD was achieved (2). LVG findings in diastole (3, left) and systole (3, right), revealed antero-septal-apical akinesis (arrows in 3, right) and hypercontractility of other regions. (D) The findings of the left ventricular apex (LVA) and the ascending aorta (AAo) pressure before (left) and after (right) dobutamine (DOB) infusion in Case 1. DOB infusion provoked a 110 mmHg pressure gradient between the LVA and the AAo.

Manifestation of latent left ventricular outflow tract obstruction caused by acute myocardial infarction: An important complication of acute myocardial infarction
 Ozaki, Kazuyuki, MD, *Journal of Cardiology*, Volume 65, Issue 6, 514-518


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Braunwald. Tratado de cardiología.

Sabatine, Marc S.; Cannon, Christopher P. Publicado January 1, 2016. Páginas 1057-1067. © 2016.

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Parada cardíaca y muerte súbita cardíaca

Braunwald. Tratado de cardiología.

Myerburg, Robert J.; Castellanos, Augustin. Publicado January 1, 2016. Páginas 821-860. © 2016.

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Infarto de miocardio con elevación del ST: tratamiento

Braunwald. Tratado de cardiología.

Mega, Jessica L.; Morrow, David A.. Publicado January 1, 2016. Páginas 1095-1154. © 2016.

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Key points 

The estimated annual incidence of acute myocardial infarction (AMI), including both ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI), in the U.S. is 600,000 new and 320,000 recurrent attacks. In 2004, AMI resulted in 695,000 hospital stays and \$31 billion in hospital charges


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

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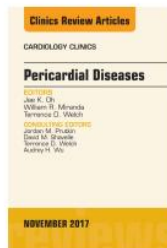
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Cardiology Clinics

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ARTÍCULO

Evaluation of Chest Pain and Acute Coronary Syndromes



Artículo en prensa: Prueba corregida

Cardiology Clinics.

Chang, Anna Marie, MD, MSCE; Fischman, David L., MD; Hollander, Judd E., MD. © 2017.

ARTÍCULO

Aldosterone Receptor Blockade in Patients with Left Ventricular Systolic Dysfunction Following Acute...



Cardiology Clinics.

Brandimarte, Filippo, MD; Blair, John E.A., MD... [Mostrar todo](#). Publicado February 1, 2008. Volume 26, Issue 1. Páginas 91-105. © 2008.

ARTÍCULO

Cardioembolic Stroke and Postmyocardial Infarction



Infarto Agudo De Miocardio

Resumen de la enfermedad [Ver tema completo](#)

First Consult

Key points



The estimated annual incidence of acute myocardial infarction (AMI), including both ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI), in the U.S. is 600,000 new and 320,000 recurrent attacks. In 2004, AMI resulted in 695,000 hospital stays and \$31 billion in hospital charges

Patients with AMI usually present with sudden or gradual onset of discomfort in the anterior chest, which also may be described as heaviness, pressure, or gas

Some patients, particularly women, patients with diabetes, and elderly patients, may have atypical symptoms, such as nausea or worsening glucose control, whereas others may have no symptoms ('silent myocardial infarction'). A high level of suspicion is required in these patient groups


The diagnosis of AMI is established on the basis of a


Algunas herramientas que debemos enfatizar por su utilidad, aportación y beneficio

All Types  ami 


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DRUG MONOGRAPH

Metoprolol

First-Metoprolol | KAPSPARGO | Lopressor | Toprol XL


Drug Information Provided By Gold Standard


Description

Description: Metoprolol is a competitive, beta-1 selective (cardioselective) adrenergic antagonist, which is most similar to atenolol. It has moderate lipid solubility, lacks intrinsic sympathomimetic activity (ISA), and has weak membrane stabilizing activity (MSA). Metoprolol is more lipid soluble than atenolol, but less than propranolol and betaxolol. The degree of lipid solubility affects metoprolol's route of elimination (extensively metabolized) and, theoretically, it's potential for CNS side effects. Metoprolol has a relatively short elimination half-life compared to other cardioselective beta-blockers. It is used for the treatment of hypertension, myocardial infarction, angina, atrial fibrillation or flutter, tremor, migraine, and heart failure. Cardioselective beta blockers are also utilized in the management of heart failure. The COMET trial evaluated carvedilol vs. metoprolol in patients with chronic heart failure (NYHA Class II–IV), and demonstrated a significantly lower all-cause mortality for carvedilol (34% vs. 40% for metoprolol).²⁷⁵⁶¹ The COMET study has been criticized for utilizing the immediate-release form of metoprolol (tartrate) vs. the extended-release formulation (succinate), which has been FDA-approved for heart failure based on the favorable findings of the MERIT-HF trial.²⁶¹⁹⁴ In the MERIT-HF trial, extended-release metoprolol significantly reduced the incidence of sudden death and mortality due to progressive heart failure in patients receiving conventional therapy (e.g., ACE inhibitors, diuretics, and, in most

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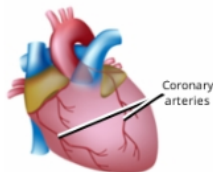
PATIENT EDUCATION

Heart Attack

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A heart attack (*myocardial infarction*, MI) is a condition that occurs when an artery in the heart (*coronary artery*) becomes narrowed or blocked. The narrowing or blockage cuts off the blood and oxygen supply to the heart, which permanently damages the heart. When one or more of your coronary arteries becomes blocked, that area of your heart begins to die. This causes symptoms felt during a heart attack.



A heart attack is a medical emergency. If you think you are having a heart attack, **do not** wait to see if the symptoms will go away. Get medical help right away.


What are the causes?

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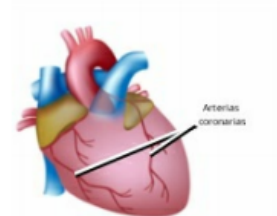
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Infarto de miocardio

Heart Attack

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Un infarto de miocardio (IM) es una afección que ocurre cuando una arteria del corazón (*arteria coronaria*) se estrecha o se obstruye. Este estrechamiento u obstrucción corta el suministro de sangre y oxígeno al corazón, lo que lo daña de manera permanente. Cuando una o más arterias coronarias se obstruyen, esa zona del corazón comienza a morir. Esto causa los síntomas que se experimentan durante el infarto de miocardio.



Un infarto de miocardio es una emergencia médica. Si cree que está sufriendo un infarto de miocardio, **no** espere a ver si los síntomas desaparecen. Solicite atención médica de inmediato.

¿Cuáles son las causas?

Muchas afecciones pueden causar un infarto de miocardio, lo que incluye:

- Aterosclerosis. Esto ocurre cuando una sustancia grasa (*placa*) se acumula gradualmente en las arterias. Esta acumulación puede obstruir o reducir el flujo sanguíneo a una o más de las arterias coronarias. Esta es la causa más frecuente del infarto de miocardio.
- Un coágulo de sangre. Un coágulo de sangre puede formarse de manera repentina cuando la placa se desintegra (*se rompe*) dentro de una arteria coronaria. Un coágulo

[Synopsis](#)[Key Points](#)[Pitfalls](#)[Terminology](#)[Clinical Clarification](#)[Classification](#)[Diagnosis](#)[Clinical Presentation](#)[History](#)[Physical Examination](#)[Causes and Risk Factors](#)[Causes](#)[Risk Factors And/or Associations](#)

CLINICAL OVERVIEW

Heart failure

Elsevier Point of Care ([see details](#))

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Synopsis

Key Points

- Heart failure is a clinical syndrome characterized by structural or functional impairment of ventricular filling or ejection of blood resulting in insufficient perfusion to meet metabolic demands. Cardinal manifestations include edema, dyspnea, and fatigue
- Diagnosis is suspected based on a thorough history and physical examination. Measurement of natriuretic peptide levels, 2-dimensional echocardiography with Doppler, and chest radiography support the diagnosis of heart failure
- Can be categorized as either heart failure with reduced ejection fraction (left ventricular ejection fraction of 40% or less) or heart failure with preserved ejection fraction (left ventricular ejection fraction of 50% or more). Patients with values of 41% to 49% are classified as having borderline reduced ejection fraction
- Coronary artery disease is the predominant cause of heart failure with reduced ejection fraction, which most commonly results in left

Urgent Action

- Life-threatening conditions (eg, acute coronary syndromes, cardiac arrhythmias) may precipitate acute decompensated heart failure and must be treated emergently

PROCEDURES CONSULT

Right Heart Catheterization and Endomyocardial Biopsy



Last Reviewed Date: 5/1/2013

Editor(s): Catherine M. Otto, MD, J. Ward Kennedy-Hamilton

Endowed Chair in Cardiology, Professor of Medicine

University of Washington School of Medicine, Director, Heart Valve Disease Clinic, Associate Director,

Procedures Consult

8 Especialidades, +200 procedimientos

FULL DETAILS

PRE-PROCEDURE

Introduction

See Figure 1 .



Figure 1 Insertion of tra

Transvenous or tempor
compromised by tachya
acutely improve cardiac

PROCEDURE

Clinical Pearls:

The following text describes blind in
placed under ECG or fluoroscopic gu
or when the equipment required for

- **Explain the procedure to pa**
 - If the clinical situation allows,
of the procedure.
 - Complications of ventricular p

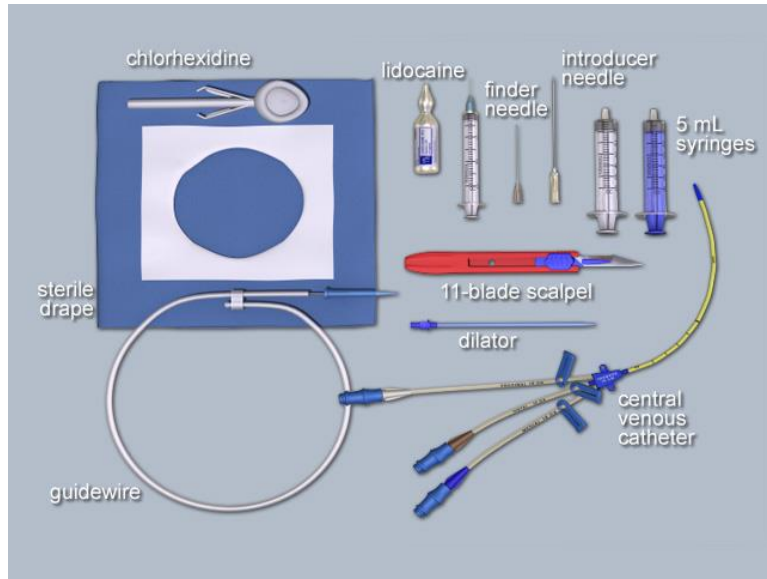
Checklist

- Explain the procedure and obtain consent.
- Observe universal precautions and use sterile technique.
- Prepare resuscitation equipment.
- Establish a right internal jugular (preferred) or left subclavian central line.
- Attach the sterile sleeve of the pacer catheter to the sheath introducer.
- Check the balloon on the catheter then insert the pacing catheter into the sheath introducer.

POST-PROCEDURE

POST-PROCEDURE CARE

- **Obtain a chest radiograph**
 - Assess for proper placement of the pacing wire in the right ventricular apex.
 - Assess for any complications related to central line insertion (e.g., pneumothorax).
- **Obtain a 12-lead electrocardiogram**
 - The electrocardiogram should show captured pacer spikes before every QRS.
 - The QRS should be in a left BBB pattern.
 - Right BBB morphologies suggest either coronary sinus pacing or septal perforation. ¹¹



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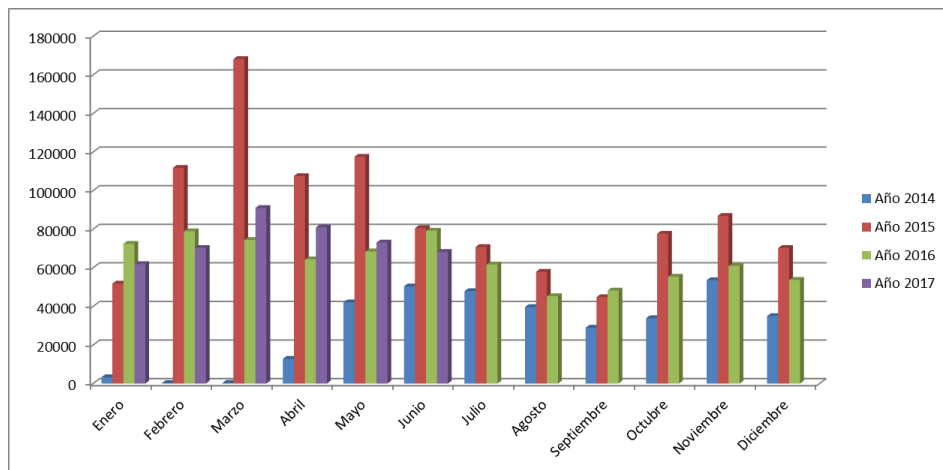
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Diciembre	34967	70246	53756	

	Eventos	Consumo
Jan	85,624	65,912
Feb	92,898	71,646
Mar	67,840	49,187
Apr	76,521	56,403
	322,883	243,148

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CpE			
Jan	85,624	\$ 13,667	\$ 0.16
Feb	92,898	\$ 13,667	\$ 0.15
Mar	67,840	\$ 13,667	\$ 0.20
Apr	76,521	\$ 13,667	\$ 0.18

CpC			
Jan	65,912	\$ 12,500	\$ 0.19
Feb	71,646	\$ 12,500	\$ 0.17
Mar	49,187	\$ 12,500	\$ 0.25
Apr	56,403	\$ 12,500	\$ 0.22



Gracias

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