TRATAMIENTO INTERVENCIONISTA DEL INFARTO AGUDO DE MIOCARDIO CON ↑ DEL SEGMENTO ST

Hospital Provincial del Centenario

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www.hemodinamiahpc.com.ar

INFARTO AGUDO DE MIOCARDIO
ASPECTOS HISTORICOS

- El síndrome clínico de la angina de pecho fue descripto en 1770, pero recién en 1912 fue James Herrick quien describió el IAM como un cuadro producido por la oclusión trombótica de una arteria coronaria.

- De Wood demostró en su estudio angiográfico oclusión trombótica de la arteria responsable en el 87% de los p. en las primeras 4 hs. del IAM. Comenzó de esta manera un cambio revolucionario en el manejo del IAM.

INFARTO AGUDO DE MIOCARDIO
ASPECTOS HISTORICOS

Coronary Thrombosis

Causes

Myocardial Necrosis

Herrick, JB. Clinical features of sudden obstruction of the coronary arteries. *JAMA* 59:2015-20, 1912.

The Journal of the American Medical Association

Published Under the Auspices of the Board of Trustees

VOL. LIX, No. 23 CHICAGO, ILLINOIS DECEMBER 7,

CLINICAL FEATURES OF SUDDEN OBSTRUCTION OF THE CORONARY ARTERIES

JAMES B. HERRICK, M.D.
CHICAGO

Obstruction of a coronary artery or of any of its large branches has long been regarded as a serious accident. Several events contributed toward the prevalence of the view that this condition was almost always suddenly fatal. Parry's writings on angina pectoris and its relation to coronary disease, Jenner's observations on the same condition centering about John Hunter's case, Thorvaldsen's tragic death in the theater in Copenhagen with the finding of a plugged coronary, sharply attracted attention to the relation between the coronary and sudden death. In Germany Cohnheim supported the views of Hyrtl and Henle as to lack of considerable anastomosis, and as late as 1881 lent the influence of his name to the doctrine that the coronary arteries were end-arteries; his Leipsic necropsy experience, as well as experiments on dogs, forced him to conclude that the sudden occlusion of one of these vessels or of one of the larger branches, such as the ramus descendens of the left coronary, meant death within a few minutes. Others emphasized the same view.

No one at all familiar with the clinical, pathologic or experimental features of cardiac disease can question the importance of the coronaries. The influence of sclerosis of these vessels in the way of producing anemic necrosis and fibrosis of the myocardium, with such possible results as aneurysm, rupture or dilatation of the heart, is well known. So also is the relation of the coronaries to many cases of angina pectoris, and to cardiac disturbances rather indefinitely classed as chronic myocarditis, cardiac irregularities, etc. It must be admitted, also, that the reputation of the descending branch of the left coronary as the artery of sudden death is not undeserved.

The coronaries are not so strictly end-arteries, with merely capillary anastomoses, as Cohnheim others thought. By careful dissections, by injec of one artery from another, by skiagraphs of inj arteries and by direct inspection of hearts made t lucent by special methods, there is proof of an anat anastomosis that is by no means negligible.

Jamin and Merkel's† beautiful stereoscopic skia show the remarkably rich blood-supply of the with occasional anastomoses between vessels of con able size. The possibility of injection of one cor artery from the other is admitted even by thos deny that such injection proves more than non-functioning anastomosis. Amenomiya ing hearts of young persons, showed naked moses in the subepicardial tissue. He feels that H and Spaltcholz‡ have nearly cleared up the questio to the relation between the heart muscle and disea the coronary artery from the anatomic stand Hirsch says that in dogs the anastomosing ves functionally competent, and Spaltcholz says tha man the vessels are nearly the same as in dogs, in anastomoses even in those of considerable cal The latter investigator, by a method of injection treatment of the heart so as to make the muscle t parent, shows to the naked eye that there are an moses of considerable size.

Among others who are on record as believing there are non-negligible anastomoses may be menti Haller, Huchard, Orth, Michaelis, Langer, Legg, W All recognize, however, that there are individual di ences, and also that though the heart may show anastomoses, these are not necessarily functional, that an artery which anatomically is not a term artery may yet be such functionally.

But there is proof not only of anatomic connec between the two coronaries, but that in certain insta at least, such connection is of functional value. Ex ments on lower animals and the clinical experimen disease of the coronaries with autopsy findings

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- El síndrome clínico de la angina de pecho fue descripto en 1770, pero recién en 1912 fue James Herrick quien describió el IAM como un cuadro producido por la oclusión trombótica de una arteria

- Pasaron más de 60 años hasta que comenzó la era de la reperfusión.

- De Wood demostró en su estudio angiográfico oclusión trombótica de la arteria responsable en el 87% de los p. en las primeras 4 hs. del IAM.

ОРИГИНАЛЬНЫЕ СТАТЬИ

УДК 616.127-005.8-036.11-085.273.55-032:611.132.2

Е. И. Чазов, Л. С. Матвеева, А. В. Мазаев, К. Е. Саргин, Г. В. Садовская, М. Я. Руда

ВНУТРИКОРОНАРНОЕ ВВЕДЕНИЕ ФИБРИНОЛИЗИНА ПРИ ОСТРОМ ИНФАРКТЕ МИОКАРДА

Всесоюзный кардиологический научный центр (дир.— акад. АМН СССР Е. И. Чазов) АМН СССР, Москва

EI Chazov
Ter Arkh vol. 48, 1976

Intracoronary administration of fibrinolysis in acute myocardial infarction

Rusia. Ter Arkh 1976;48:8–19

Acute Myocardial Infarction: Intracoronary Application of Nitroglycerin and Streptokinase

K. P. RENTROP, M.D., H. BLANKE, M.D., K. R. KARSCH, M.D., V. WIEGAND, M.D., H. KÖSTERING, M.D., H. OSTER, M.D., K. LEITZ, M.D.

Department of Internal Medicine and Department of Thoracic Surgery, University of Goettingen, Goettingen, W. Germany

Summary: In five patients with acute myocardial infarction, the effects of both intracoronary nitroglycerin (NTG) and subsequent intracoronary streptokinase application were evaluated. In addition, transluminal recanalization was performed in one of these patients. Injection of NTG into the infarct-related coronary artery resulted in improved distal filling of the subtotally occluded left circumflex artery in one patient, and in transient patency of the completely occluded right coronary artery in a second patient. In a third patient patency of the totally occluded left anterior descending artery (LAD) was achieved by transluminal recanalization with a guide wire. In a fourth patient with occlusion of the LAD, there was no response to intracoronary NTG and mechanical recanalization was not attempted. Subsequent intracoronary infusion of streptokinase (1,000–2,000 U/min for 15–60 min) resulted in a further and long-term reduction of narrowing at the site of acute occlusion in patients I–III and in opening of the completely occluded LAD in patient IV. Improvement of lumen was paralleled by alleviation of symptoms. In a fifth patient, in whom the LAD was subtotally occluded, the degree of coronary obstruction could not be changed by intracoronary application of NTG or by lysis. In this patient, symptoms and ECG changes improved with reduction of pathologically elevated blood pressure values. The findings suggest that myocardial infarction had been caused by thrombotic occlusion in four patients, and that spasm of the infarct vessel could have been an additional factor in two of these patients. In the fifth patient, an increase of afterload in the presence of a subtotal lesion might have caused the critical imbalance between oxygen supply and demand, resulting in cell death.

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Accepted: August 29, 1979

Keywords: acute myocardial infarction, streptokinase, nitroglycerin, transluminal recanalization, aortocoronary bypass surgery

Clin Cardiol 1979;2:354–363

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Prevalence of Total Coronary Occlusion during the Early Hours of Transmural Myocardial Infarction

Abstract

To define the prevalence of total coronary occlusion in the hours after transmural myocardial infarction, we used coronary arteriography to study the degree of coronary obstruction in 322 patients admitted within 24 hours of infarction. Total coronary occlusion was observed in 110 of 126 patients (87 per cent) who were evaluated within four hours of the onset of symptoms; this proportion decreased significantly, to 37 of 57 (65 per cent), when patients were studied 12 to 24 hours after the onset of symptoms. Among 59 patients with angiographic features of coronary thrombosis, the thrombus was retrieved by Fogarty catheter in 52 (88 per cent) but was absent in seven (12 per cent false positive). Among an additional 20 patients without angiographic features of thrombosis, a thrombus was discovered in five (25 per cent false negative). Thus, total coronary occlusion is frequent during the early hours of transmural infarction and decreases in frequency during the initial 24 hours, suggesting that coronary spasm or thrombus formation with subsequent recanalization or both may be important in the evolution of infarction. (N Engl J Med. 1980; 303:897–902.)

N Engl J Med 1980;303:897–902

INFARTO AGUDO DE MIOCARDIO

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INFARTO AGUDO DE MIOCARDIO
ASPECTOS HISTORICOS

1977 FDA Approval of Thrombolytic Therapy

Approved Indications:

- Pulmonary Embolism
- Deep Vein Thrombosis
- Peripheral Artery Thrombosis
- Thrombosed Dialysis Fistulae
- Thrombosed Intravenous Catheters

No approval for acute myocardial infarction

INFARTO AGUDO DE MIOCARDIO
ASPECTOS HISTORICOS

Abandonment of Fibrinolysis in AMI in 1960s

1. Myocardial Necrosis Complete after 60 min
   (Jennings, 1960)
2. Reperfusion injury
   (Jennings, 1960)
3. Lysis of Thrombus requires 3 to 7 hrs
   (Ruegsegger, 1960)
4. Causative Role of Coronary Thrombus?
   (Branwood, 1956)

INFARTO AGUDO DE MIOCARDIO
ASPECTOS HISTORICOS

FDA Approvals of Thrombolytic Therapy

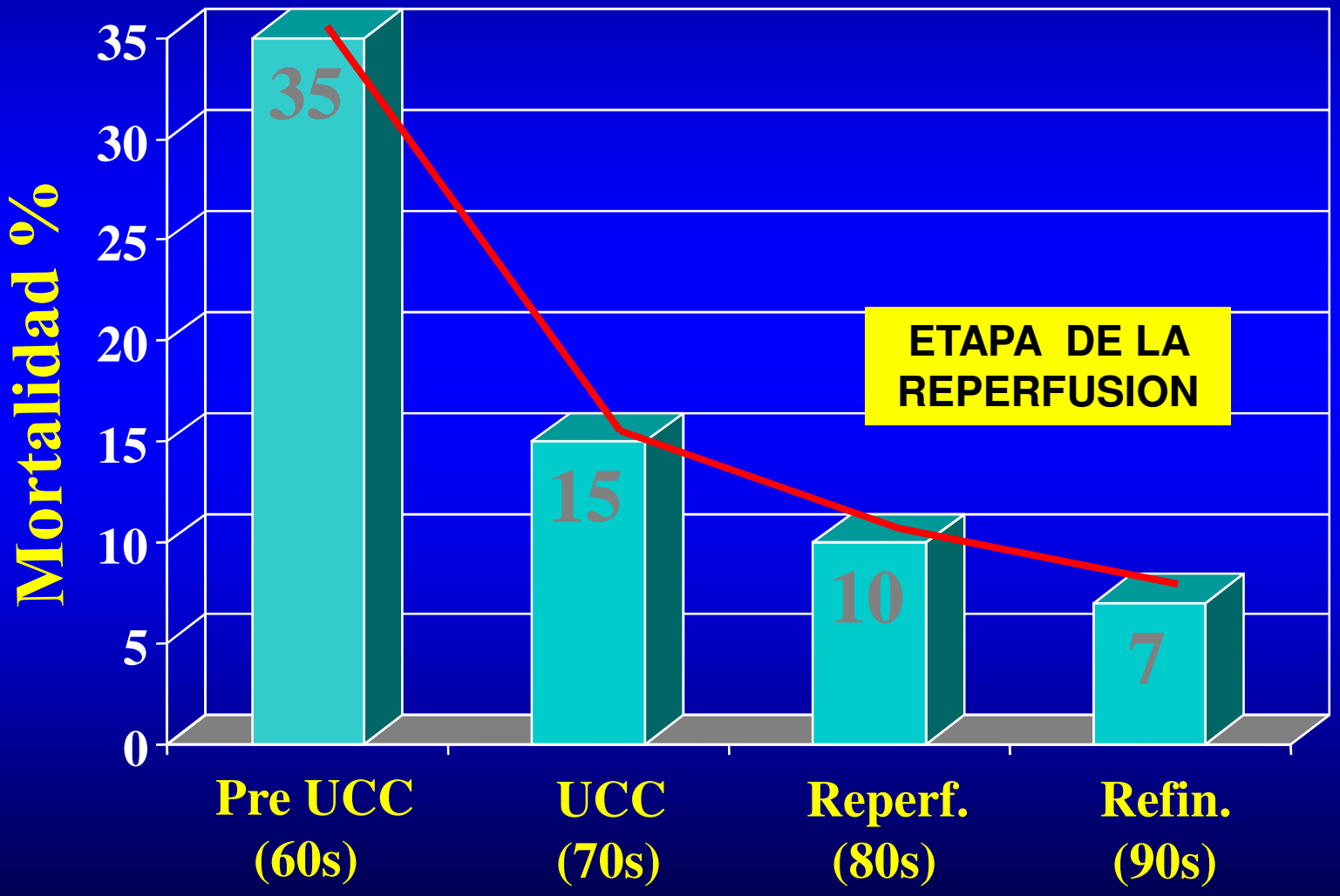
1977: Various thromboembolic conditions, not for AMI

1982: Intracoronary SK for AMI

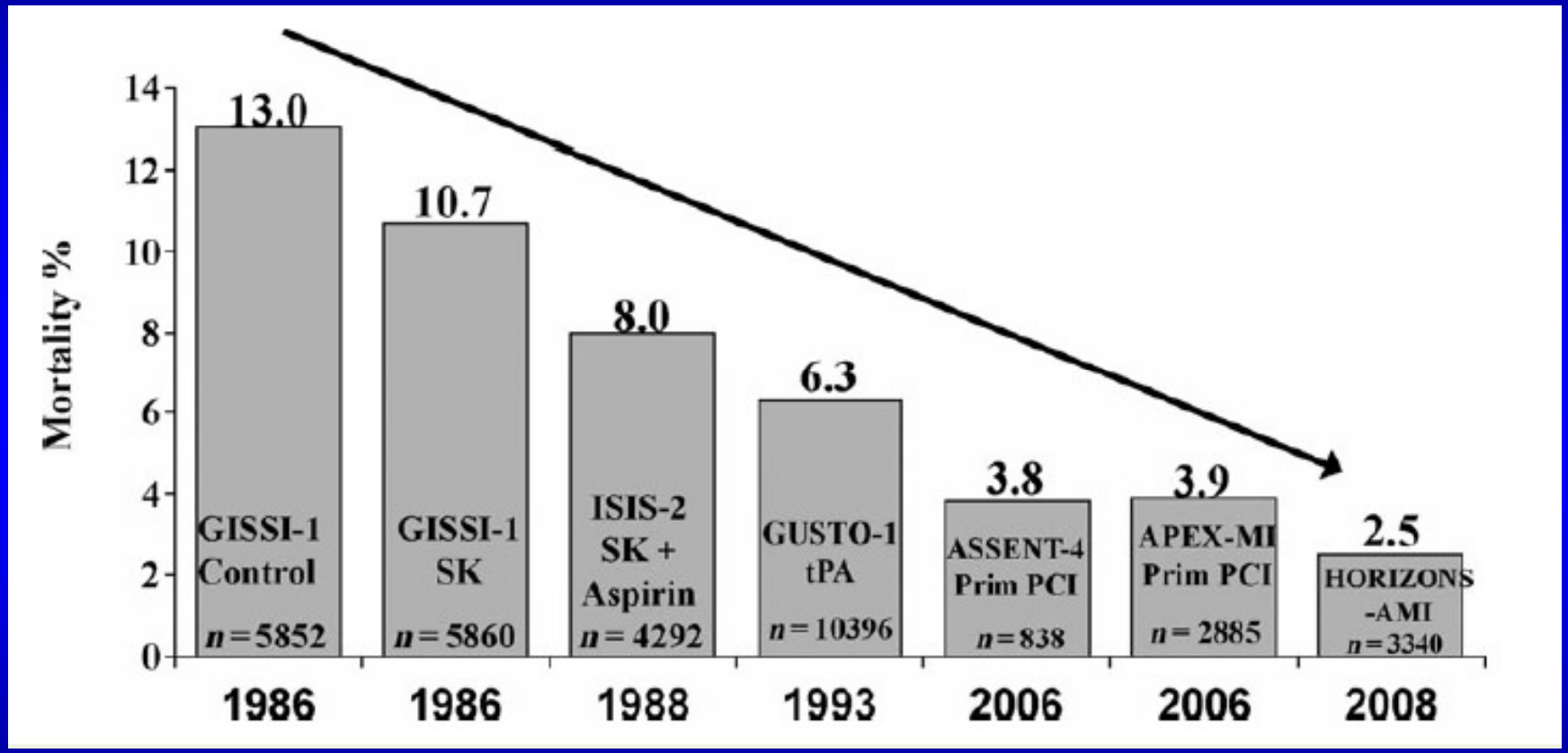
1987: Intravenous SK and rTPA for AMI

Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico (GISSI). Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. Lancet 1986;1:397–402

REDUCCIÓN DE LA MORTALIDAD

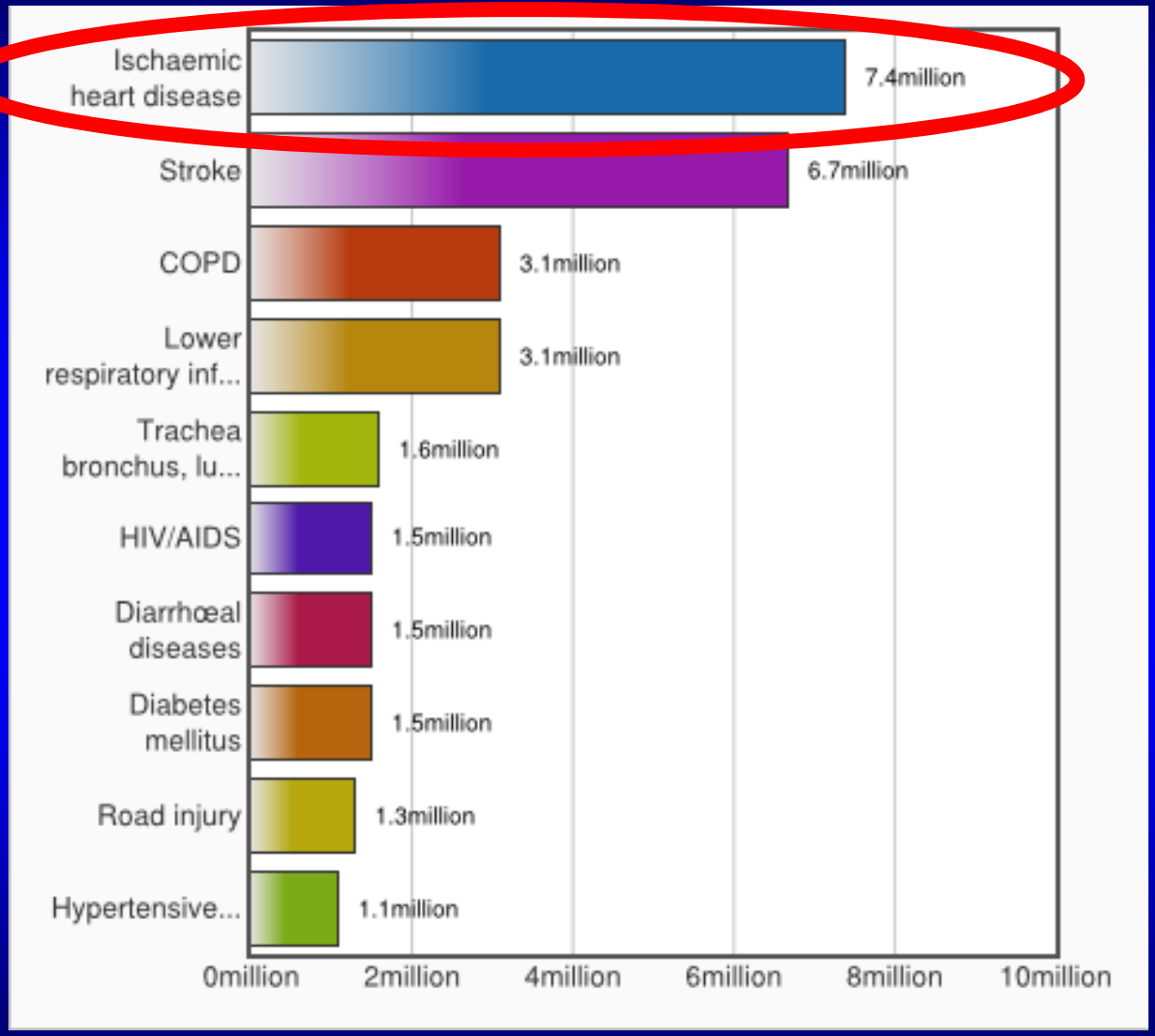


EVOLUCION DE LA TASA DE MORTALIDAD EN GRANDES ESTUDIOS RANDOMIZADOS



European Heart Journal (2014) 35, 2510–2515

10 PRINCIPALES CAUSAS DE MUERTE EN EL MUNDO AÑO 2102



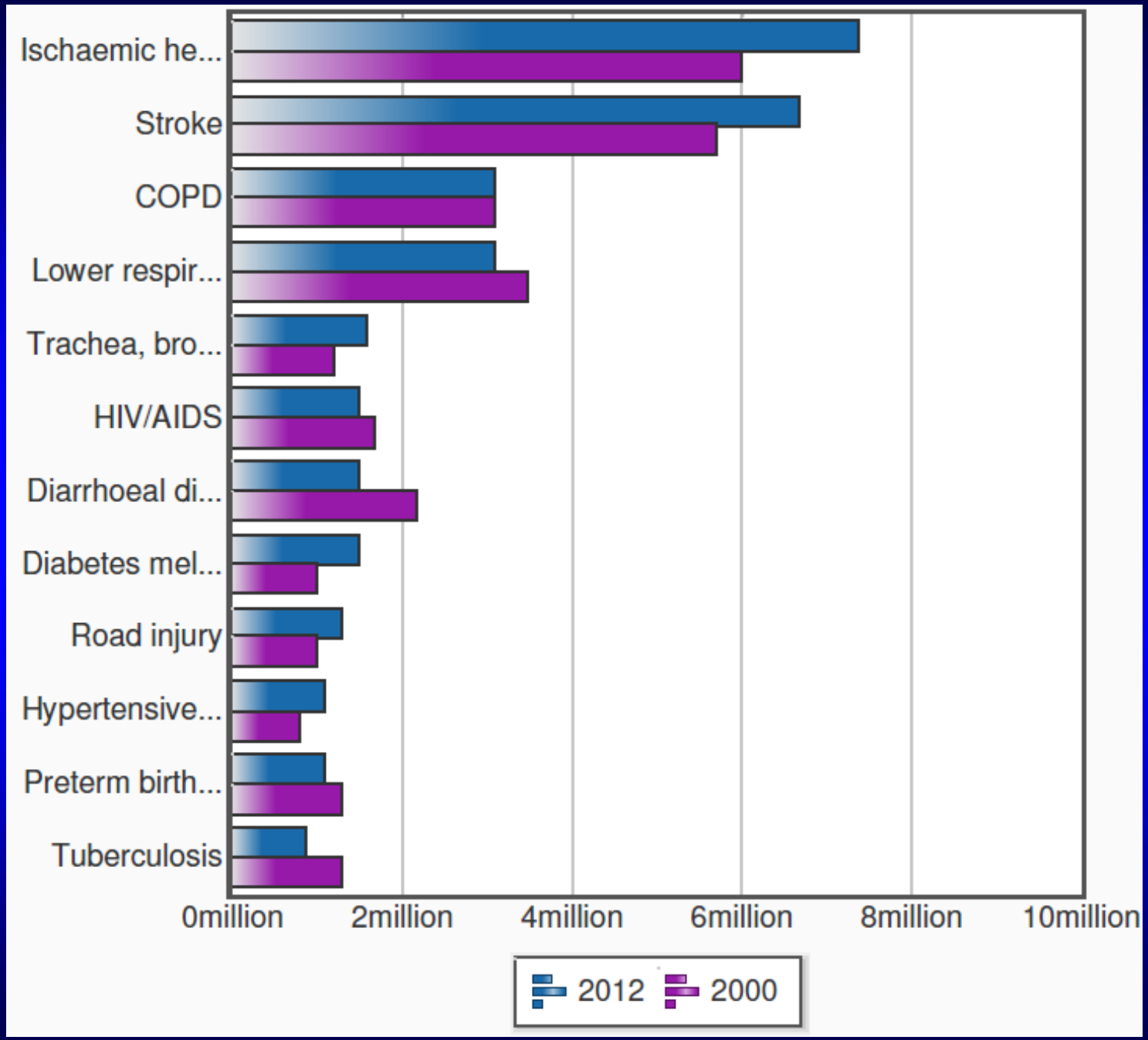
World Health Organization

World Health Organization Fact sheet Nº 310

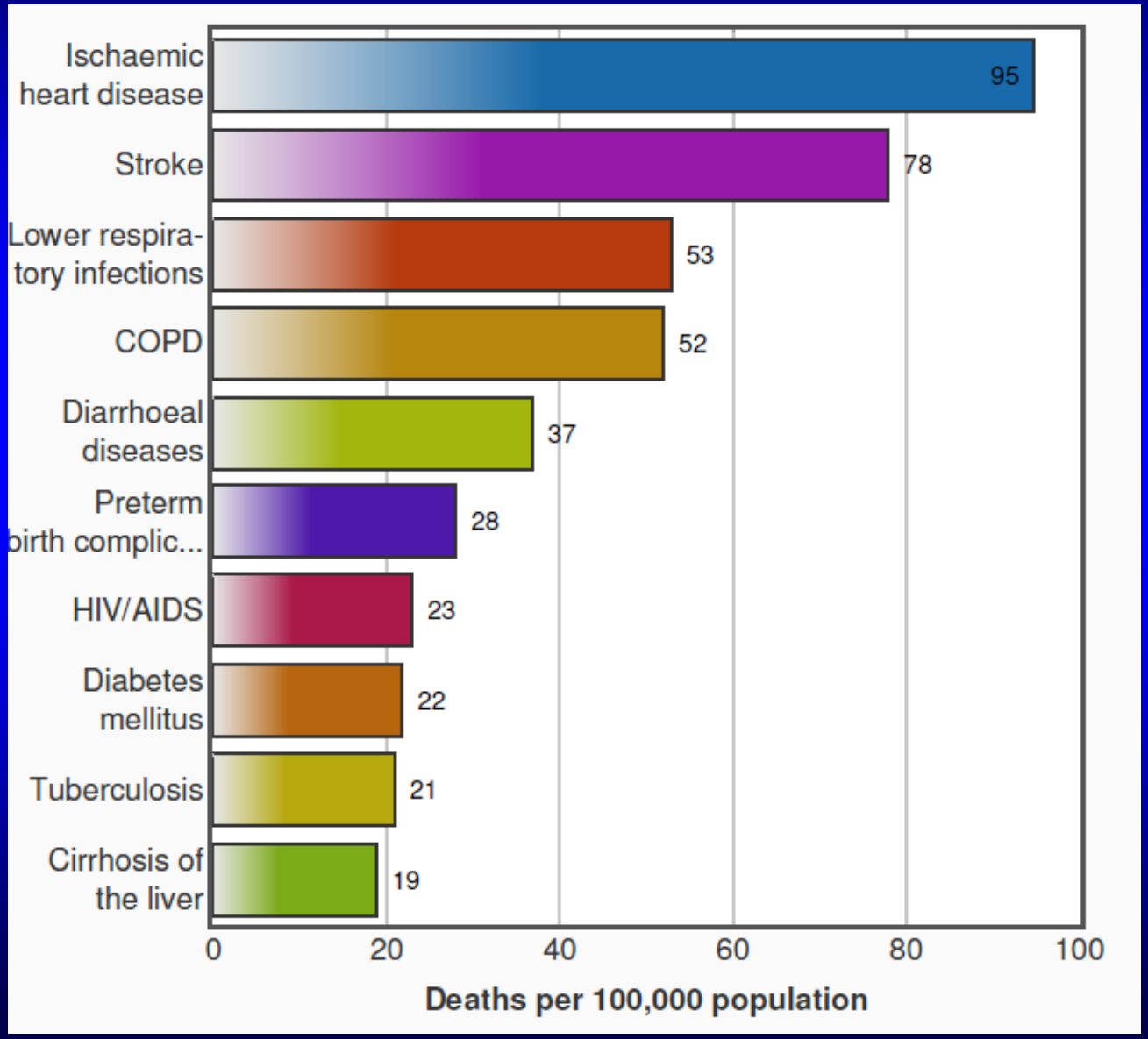
Comparación de las principales causas de muerte en el mundo entre 2000 y 2102

World Health Organization

World Health Organization Fact sheet Nº 310


Ischaemic he...
Stroke
COPD
Lower respir...
Trachea, bro...
HIV/AIDS
Diarrhoeal di...
Diabetes mel...
Road injury
Hypertensive...
Preterm birth...
Tuberculosis
0million
2million
4million
6million
8million
10million
2012
2000

Principales causas de muerte en 2012 en países de medianos ingresos



OBJETIVOS DEL TRATAMIENTO EN EL IAM

- REPERFUNDIR MAS PACIENTES
- REPERFUNDIR MAS RAPIDO
- REPERFUNDIR LO + COMPLETA/ POSIBLE
- REGENERAR EL TEJIDO NECROSADO

REDEFINICION DEL INFARTO AGUDO DE MIOCARDIO

Movimiento de los valores de biomarcadores cardíacos (preferiblemente troponina) con al menos un valor por encima del percentil 99 del límite superior de referencia y con al menos uno de los siguientes:

* **Síntomas de isquemia**
* **Nuevos cambios del segmento ST-T o nuevo BRI**
* **Desarrollo de ondas Q patológicas en el ECG**
* **Evidencia por imágenes de nueva pérdida de miocardio viable, o una nueva anormalidad de la motilidad regional**
* **Identificación de un trombo intracoronario por angiografía o autopsia**

CENTENARIO HOSPITAL PROVINCIAL

Thygesen . Universal definition of myocardial infarction. Eur Heart J 2007; 28:2525–2538.

METODOS PARA LOGRAR LA REPERFUSION

TRATAMIENTO TROMBOLITICO

INTERVENCIONISMO CORONARIO PERCUTANEO

ICP PRIMARIA (ICPP)

DEFINICION

Intervención coronaria realizada en el vaso culpable dentro de las 12 horas del comienzo de los síntomas SIN terapia trombolítica previa.

The NEW ENGLAND JOURNAL of MEDICINE

Volume 328:673-679 March 11, 1993 Number 10

A Comparison of Immediate Angioplasty with Thrombolytic Therapy for Acute Myocardial Infarction

Cindy L. Grines, Kevin F. Browne, Jean Marco, Donald Rothbaum, Gregg W. Stone, James O'Keefe, Paul Overlie, Bryan Donohue, Noah Chelliah, Gerald C. Timmis, Ronald E. Vlietstra, Michelle Strzelecki, Sylvia Puchrowicz-Ochocki, William W. O'Neill, for The Primary Angioplasty in Myocardial Infarction Study Group

Volume 328:680-684 March 11, 1993 Number 10

A Comparison of Immediate Coronary Angioplasty with Intravenous Streptokinase in Acute Myocardial Infarction

Felix Zijlstra, Menko Jan de Boer, Jan Hoornje, Stoffer Reiffers, Johan Reiber, and Harry Suryapranata

Volume 328:685-691 March 11, 1993 Number 10

Immediate Angioplasty Compared with the Administration of a Thrombolytic Agent Followed by Conservative Treatment for Myocardial Infarction

Raymond J. Gibbons, David R. Holmes, Guy S. Reeder, Kent R. Bailey, Mona R. Hopfenspirger, Bernard J. Gersh, for The Mayo Coronary Care Unit and Catheterization Laboratory Groups

¿ COMO REPERFUNDIR ?

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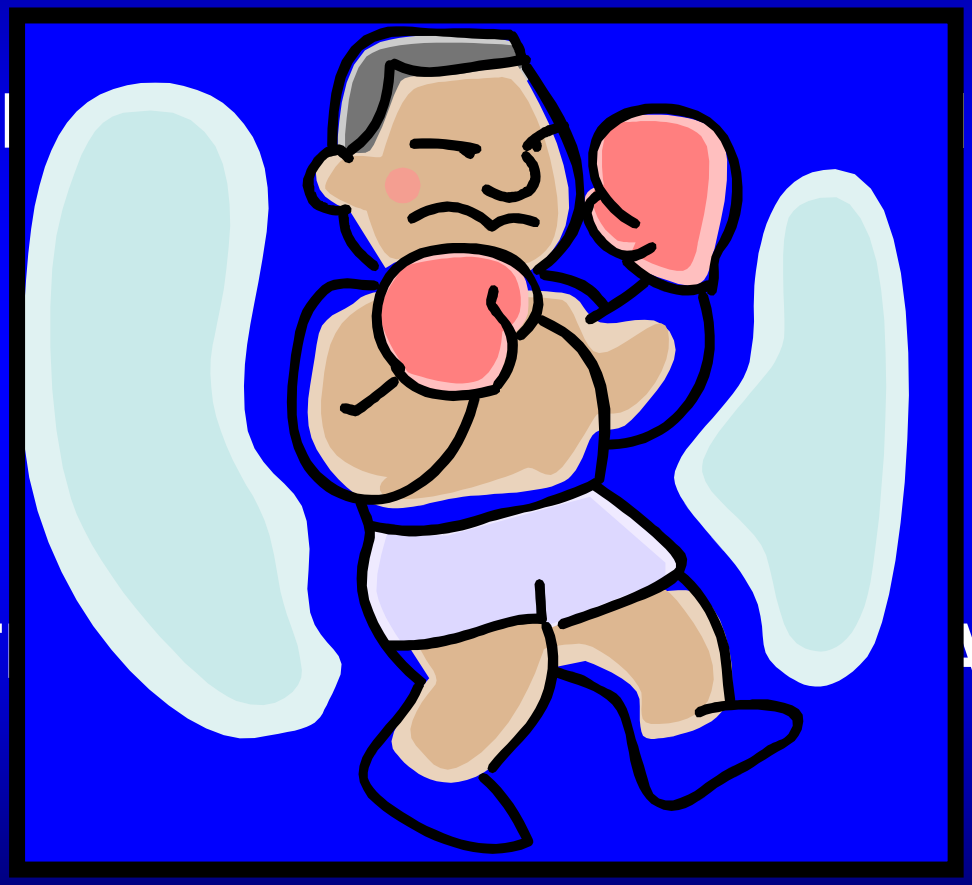


1990's Perception of Primary PCI Doctors

Articles

The Lancet Volume 361, No. 9351, p13–20, 4 January 2003

Primary angioplasty versus intravenous thrombolytic therapy for acute myocardial infarction: a quantitative review of 23 randomised trials

Ellen C Keeley, Judith A Boura, Cindy L Grines

Summary

Background Many trials have been done to compare primary

23 trials, which together randomly assigned 7739 thrombolytic-eligible patients with ST-segment elevation AMI to primary PTCA (n=3872) or thrombolytic therapy (n=3867). Streptokinase was used in eight trials (n=1837), and fibrin-specific agents in 15 (n=5902). Most patients who received thrombolytic therapy (76%, n=2939) received a fibrin-specific agent. Stents were used in 12 trials, and platelet glycoprotein IIb/IIIa inhibitors were used in eight. We identified short-term and long-term clinical outcomes of death, non-fatal reinfarction, and stroke, and did subgroup analyses to assess the effect of type of thrombolytic agent used and the strategy of emergent hospital transfer for primary PTCA. All analyses were done with and without inclusion of the SHOCK trial data.

Metaanálisis de 23 estudios randomizados

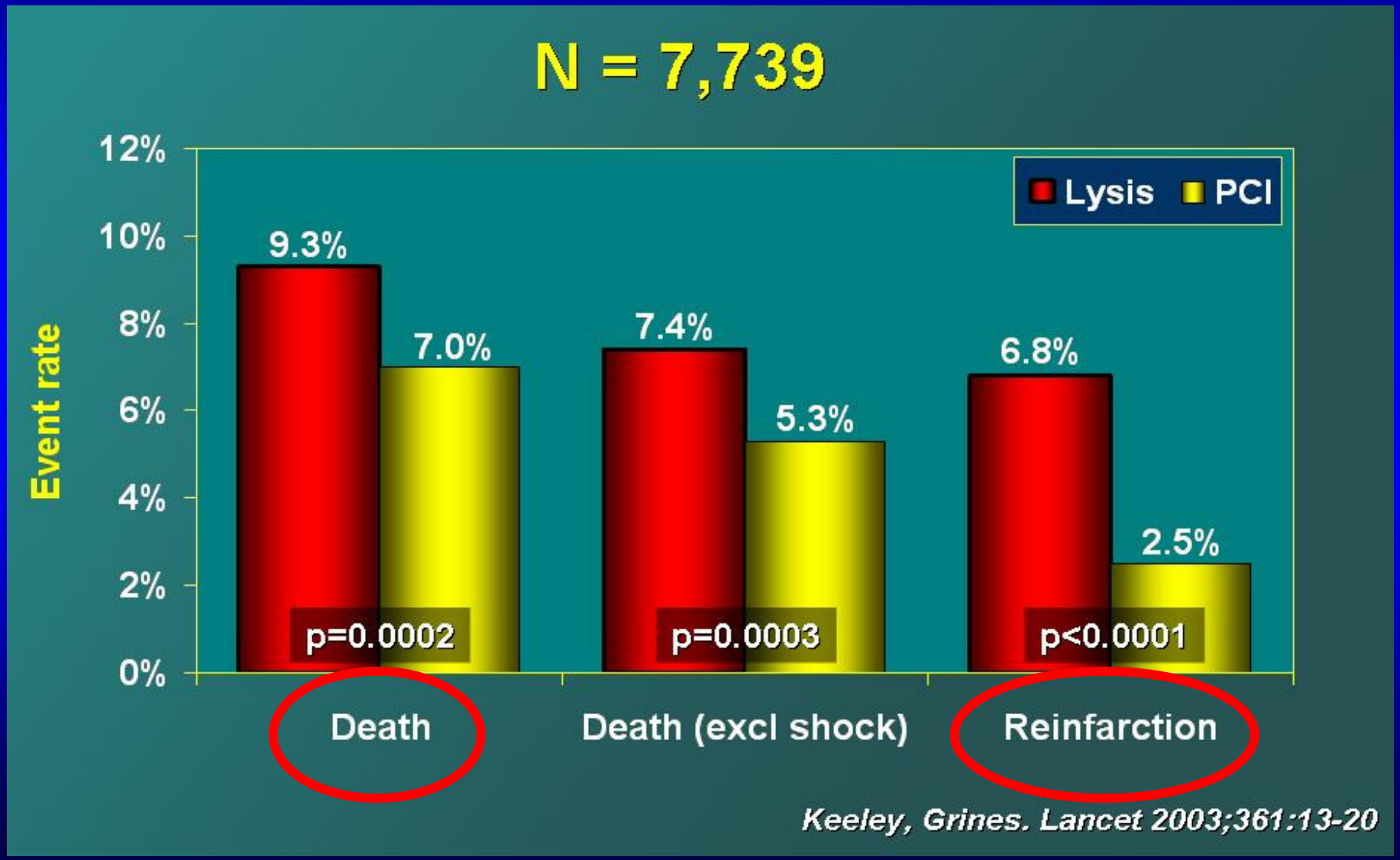
Introduction

In the mid-1970s, acute myocardial infarction (AMI) was identified, in nearly all cases, as being the result of a

that the prompt restoration of flow salvages myocardium, reduces infarct size, and prolongs life has been the driving force behind a large number of clinical trials, assessing thrombolytic therapy for AMI. The results of these trials, done in the early 1980s and involving tens of thousands of patients, unequivocally showed that thrombolytic therapy resulted in preserved left-ventricular function and decreased mortality in patients with AMI.[3]

Primary percutaneous transluminal coronary angioplasty (PTCA), defined as balloon angioplasty undertaken as the primary reperfusion strategy for AMI without previous or concomitant thrombolytic therapy, was initially compared with intracoronary thrombolytic therapy.[4] Over the next decade, ten trials, comparing primary PTCA with intravenous thrombolytic therapy for

23 ESTUDIOS RANDOMIZADOS DE PCI VS LÍTICOS



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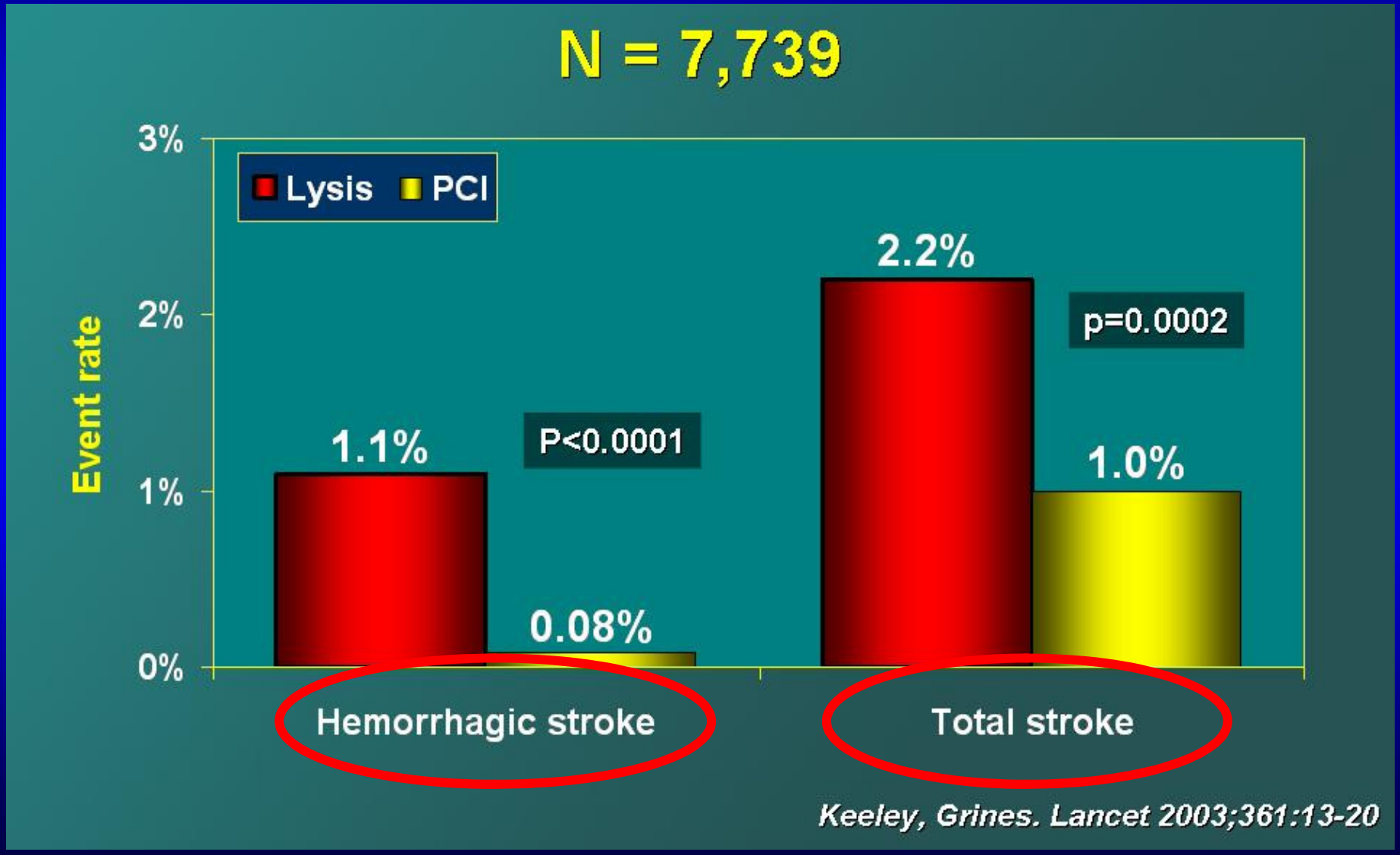


VENTAJAS DE LA ICPP SOBRE LOS LITICOS

- Menor mortalidad
- Menor reinfarto
- Menor incidencia de stroke
- Mejoría en la función ventricular

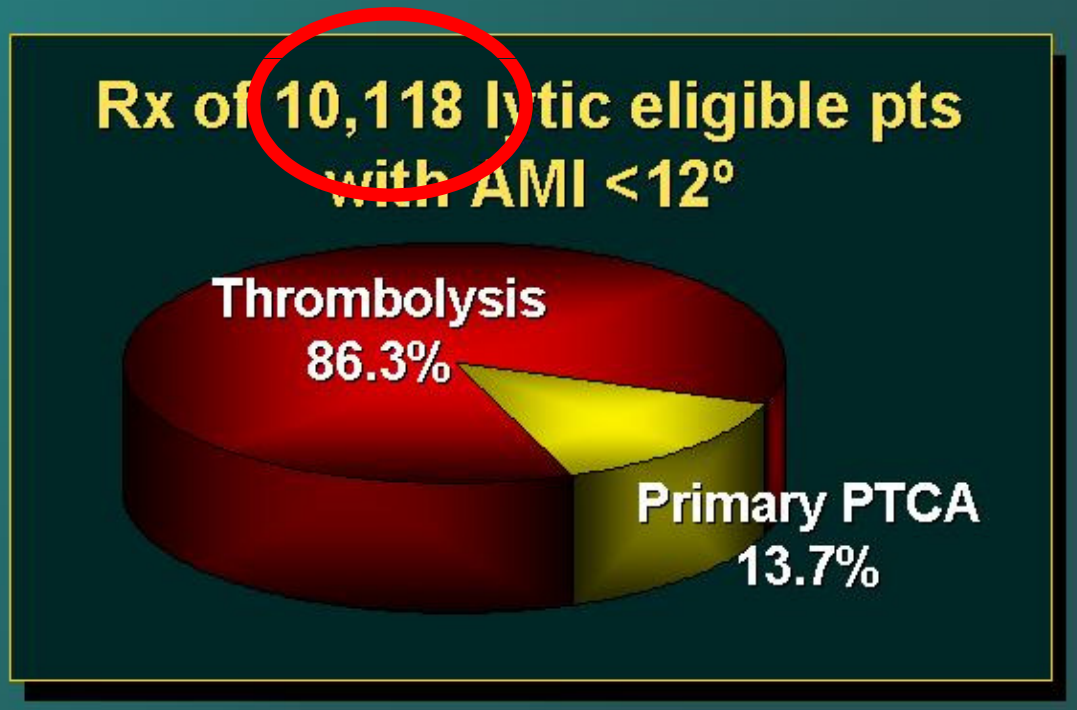
VENTAJAS ADICIONALES DE LA ICPP SOBRE LOS LITICOS

- Menor isquemia recurrente
- Menor necesidad de cateterismo no planeado y procedimientos de revascularización
- Alta hospitalaria más precoz
- Similares costos a largo plazo

MORTALIDAD ENTRE 1994 Y 1998 EN ALEMANIA

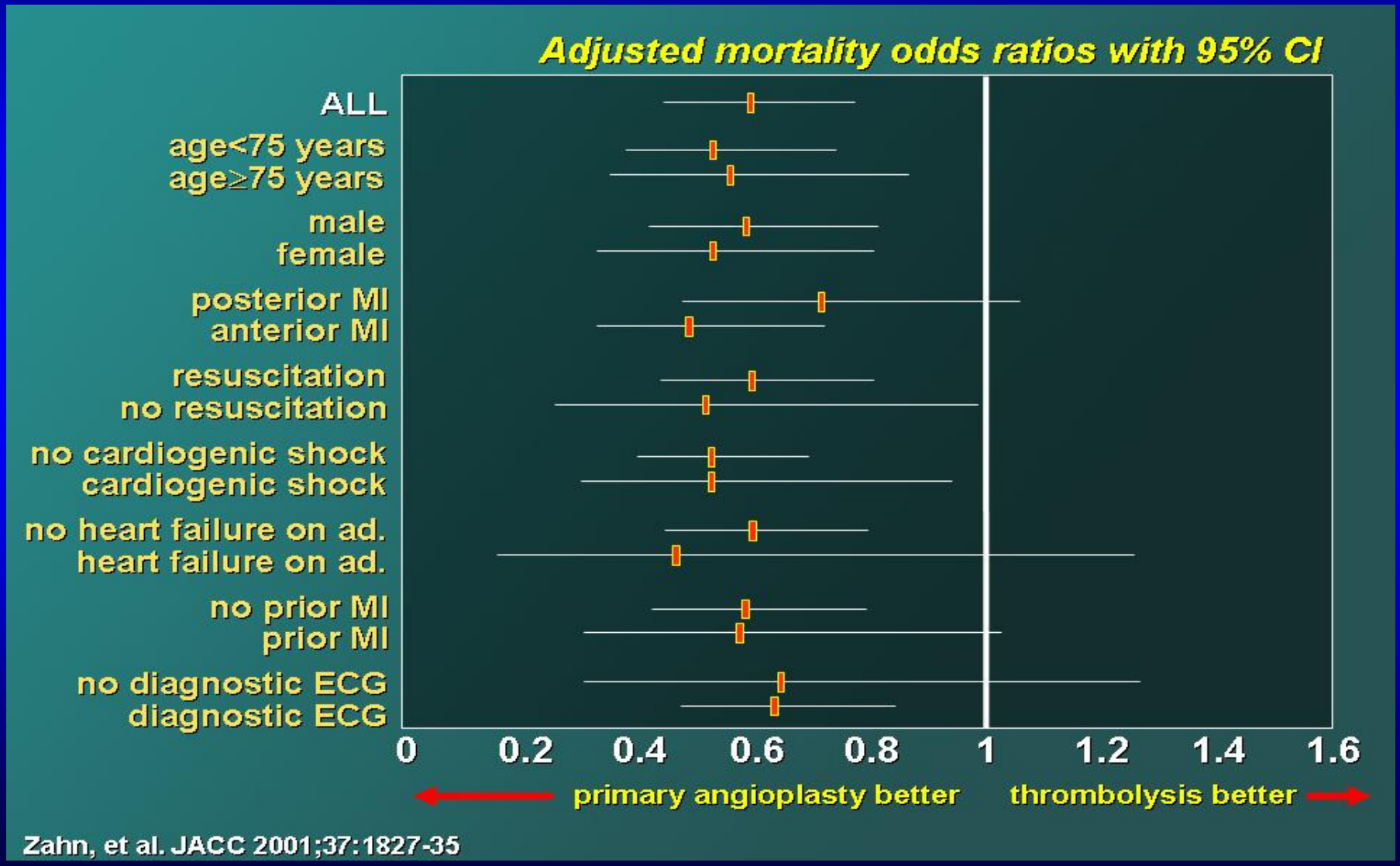
Maximal Individual Therapy in AMI (MITRA) and Myocardial Infarction Registry (MIR)

54 and 217 hospitals, respectively (w/o overlap)



Zahn R et al. JACC 2000;36:2064-71

MORTALIDAD ENTRE 1994 Y 1998 EN ALEMANIA



Zahn, et al. JACC 2001;37:1827-35

1º CONCLUSION

La ICP primaria es superior a los trombolíticos

pero ….

siempre y cuando se cumplan 2 condiciones

1) El tiempo puerta balón debe ser < de 120 m

1º CONCLUSION

La ICP primaria es superior a los trombolíticos

pero ….

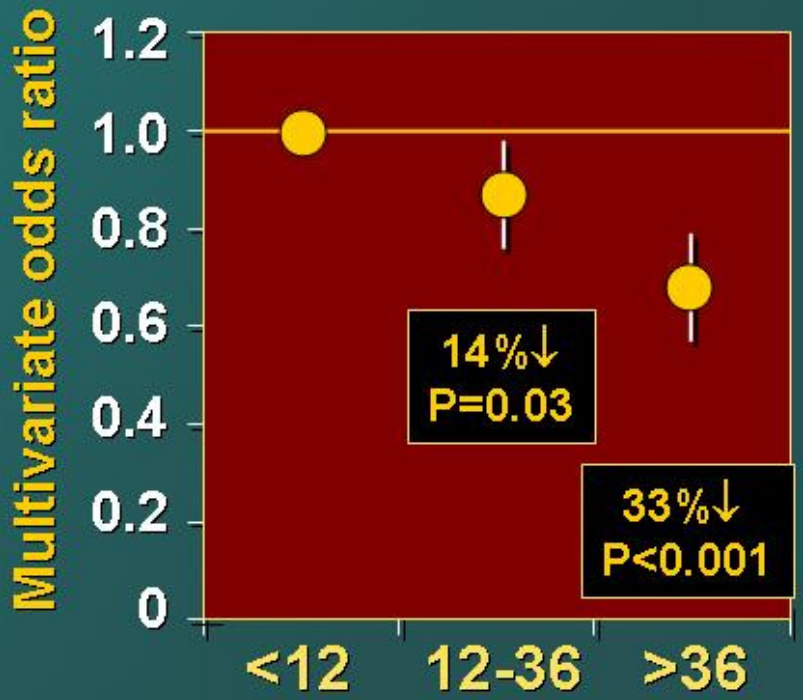
siempre y cuando se cumplan 2 condiciones

1) El tiempo puerta balón debe ser < de 120 m

2) El team que realiza la ICP debe estar bien entrenado como así también la institución

Relationship between institutional primary PCI volume and in-hospital mortality

The NRMI-2 Database (n=27,080)



Cannon CP et al. JAMA 2000;283:2941-7

www.usatoday.com

THE NATION'S NEWSPAPER

Monday, November 13, 2006

Newsline

News Money Sports Life

Hospitals too slow on heart attacks

Study finds only 35% react quickly enough

By Steve Sternberg
USA TODAY

CHICAGO — Only about one-third of hospitals provide emergency care to heart attack patients quickly enough to meet scientific guidelines for saving lives, researchers report today.

Even top performers meet American Heart Association and American College of Cardiology (ACC) guidelines for care in only half their cases, researchers say.

"Even among the better hospitals, only a few hospitals routinely meet the recommended guidelines," says Yale cardiologist Harlan Krumholz, a leader of the research team and an architect of a national campaign launched Sunday to help hospitals improve their performance. "By next year, we're going to change that."

About 200,000 people a year have heart attacks caused by blockages in crucial arteries supplying the heart with blood. About 16,000 patients die of these heart attacks in hospitals each year.

Studies show that reopening clogged arteries by inflating a tiny balloon at the site of the blockage is the best way to treat a severe heart attack. The procedure, balloon angioplasty, can cut a patient's risk of dying by 40%, but only if it is done within 90 minutes of the patient's arrival at the hospital, the so-called door-to-balloon time.

If every hospital met the guidelines, Krumholz says, doctors could save about 1,000 lives each year. A study out in March in the journal Circulation showed that 81% of people live within an hour's drive of a hospital that provides balloon angioplasty.

Yet only about one-third of heart attack patients get angioplasty within the 90-minute window. The new study surveyed 365 hospitals to determine what procedures they have in place to get patients angioplasty quickly. Only 35% report an average door-to-balloon time of 90 minutes or less, 48% had a door-to-balloon time of 91 to 120 minutes, 13% came in at 121 to 150 minutes, and 4% topped 150.

The study, sponsored by the National Heart, Lung and Blood Institute, will be released today at an AHA meeting and has been posted online by the *New England Journal of Medicine*. Hospitals agreed to participate if they weren't individually identified. Doctors said consumers can get an approximate measure of many hospitals' response times by checking a U.S. government website, www.hospitalcompare.hhs.gov.

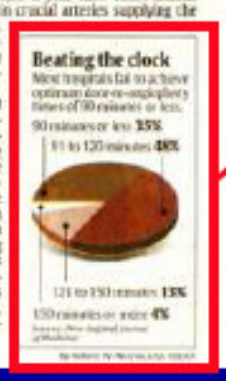
The study was designed to help launch the new campaign, called D2B, for door-to-balloon, by providing hospitals with ways to improve performance. "We're losing too many lives," says Steven Nissen of the Cleveland Clinic and president of the AC

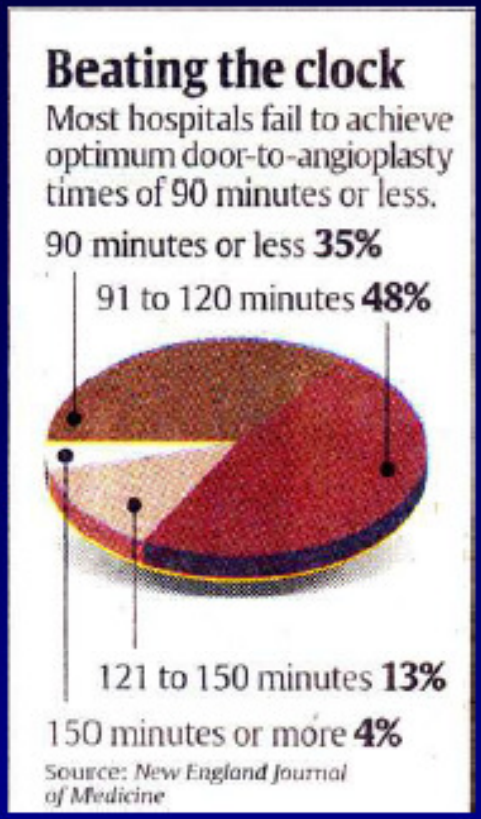
Momentum for change is building, Krumholz says, noting that hospitals, insurers and government agencies have signed on. Among the participants: United Healthcare, Premier Health Care Services, Aetna, WellPoint Inc. and Blue Cross Blue Shield.

The study found that the system works best when:

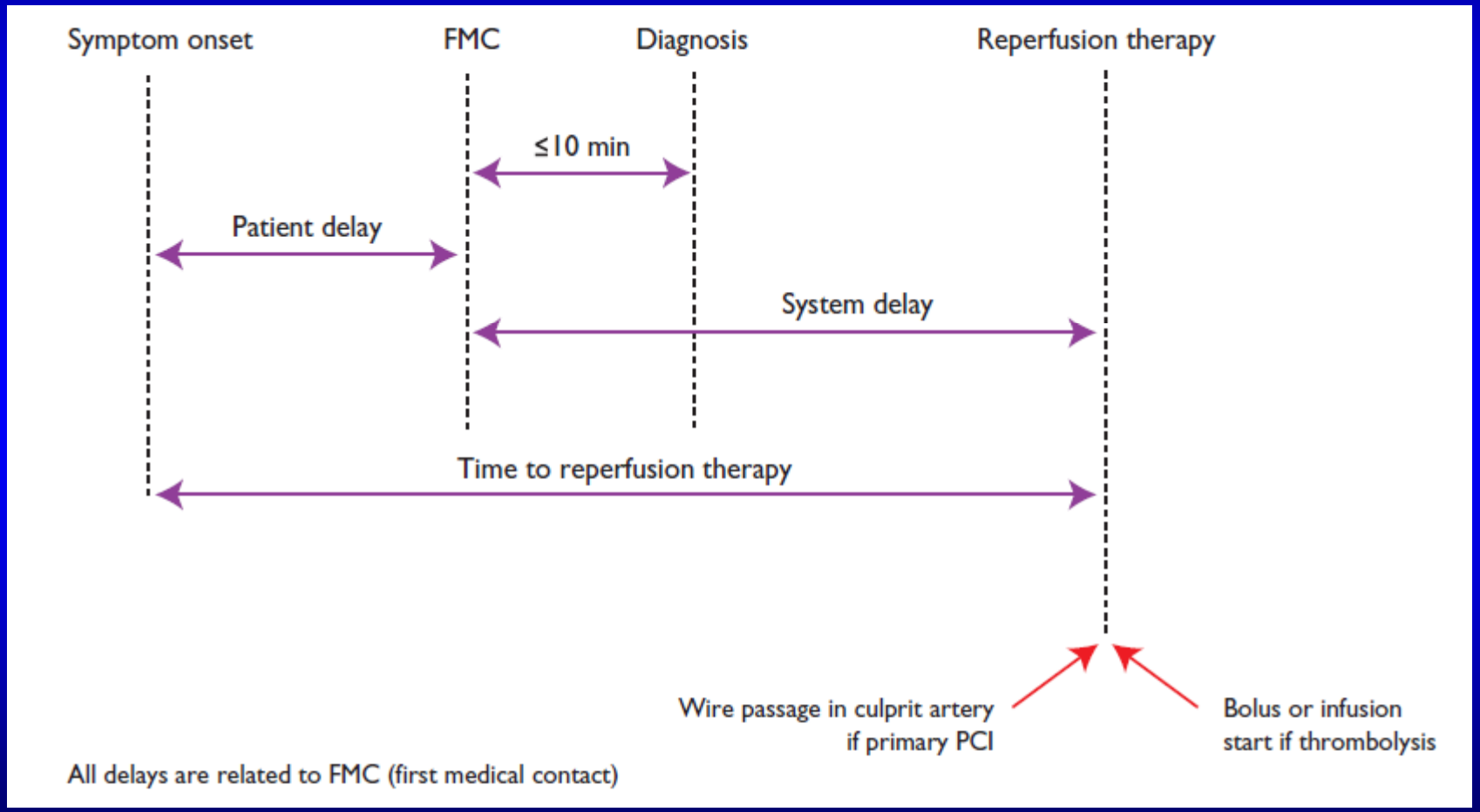
- Paramedics perform an electrocardiogram en route to the hospital.
- The ER doctor uses it to activate the angioplasty team without waiting to consult with anyone else.
- A page operator can reach each member of the team, and they can arrive within 20 minutes.
- The hospital posts feedback on each case for the angioplasty and ER teams.

Whatever option is offered, minutes count. "You've had a heart attack. You've rushed to the hospital, and now you're waiting for a procedure to open the blockage," Krumholz says. "Most Americans wait two hours or more. And every minute you wait, your heart is being deprived of oxygen."





COMPONENTES DE LA DEMORA EN STEMI E INTERVALOS DE TIEMPO IDEALES PARA LA INTERVENCION



RECOMENDACIONES AL DIAGNOSTICO INICIAL

Recommendations	Class [a]	Level [b]
A 12-lead ECG must be obtained as soon as possible at the point of FMC, with a target delay of ≤10 min.	I	B
ECG monitoring must be initiated as soon as possible in all patients with suspected STEMI.	I	B
Blood sampling for serum markers is recommended routinely in the acute phase but one should not wait for the results before initiating reperfusion treatment.	I	C

PRESENTACIONES ATIPICAS

- LBBB
- Ventricular paced rhythm
- Patients without diagnostic ST-segment elevation but with persistent ischaemic symptoms
- Isolated posterior myocardial infarction
- ST-segment elevation in lead aVR

ANGIOPLASTIA PRIMARIA

¿ Que debemos hacer si el Hospital donde recibimos al paciente con el IAM no cuenta con Sala de Hemodinamia de urgencia ?

Realizar Trombolíticos IV en el mismo sitio ?

Transferir al paciente a otro centro donde se le pueda realizar angioplastia primaria de urgencia

ANGIOPLASTIA PRIMARIA

¿ Que debemos hacer si el Hospital donde recibimos al paciente con el IAM no cuenta con Sala de Hemodinamia de urgencia ?

La decisión depende de la valoración del riesgo clínico individual.

La elección es frecuentemente decidida por cuestiones logísticas y problemas o demoras en el transporte del paciente.

ANGIOPLASTIA PRIMARIA

¿ Que debemos hacer si el Hospital donde recibimos al paciente con el IAM no cuenta con Sala de Hemodinamia de urgencia ?

LIMI

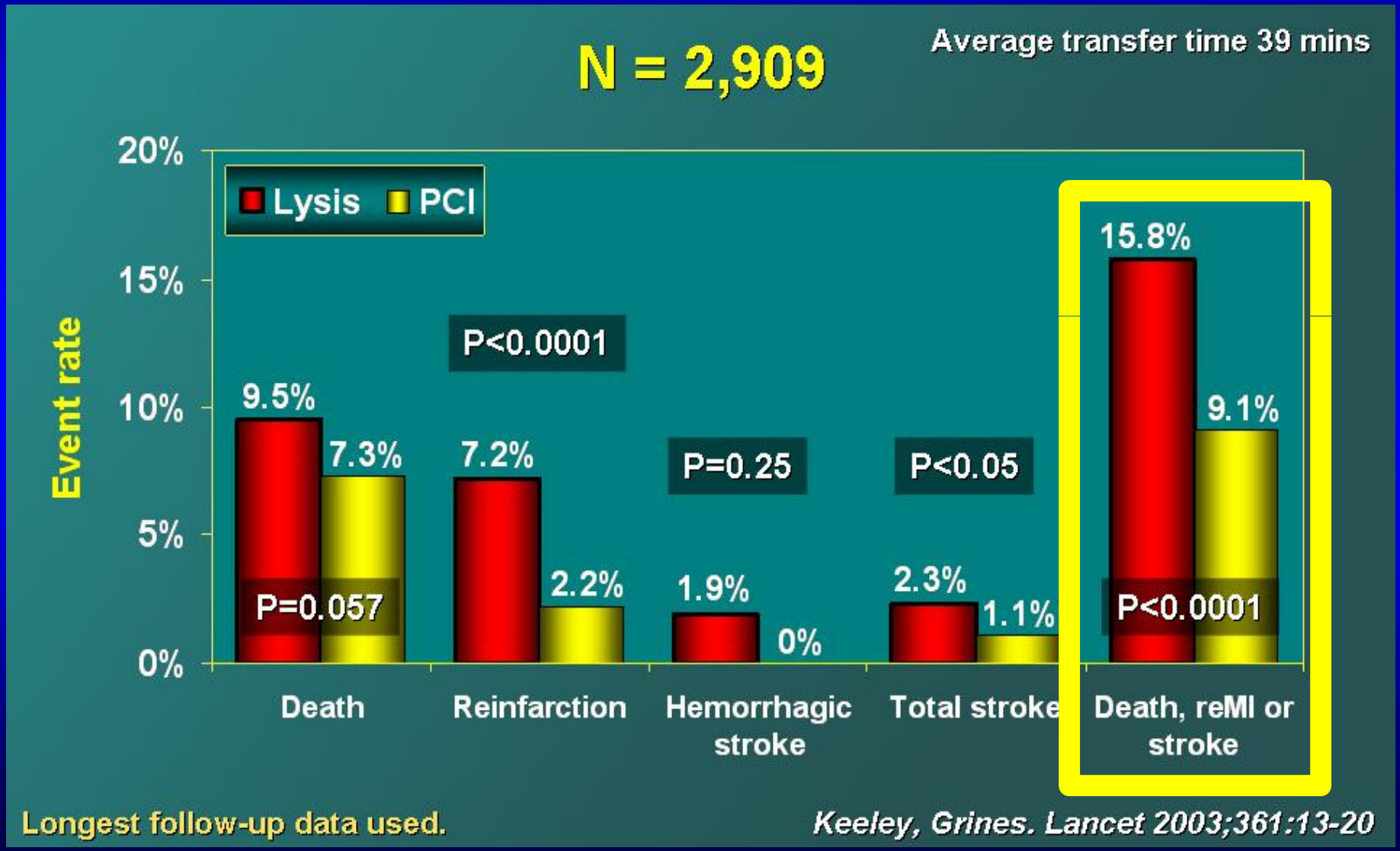
PRAGUE- 1

PRAGUE- 2

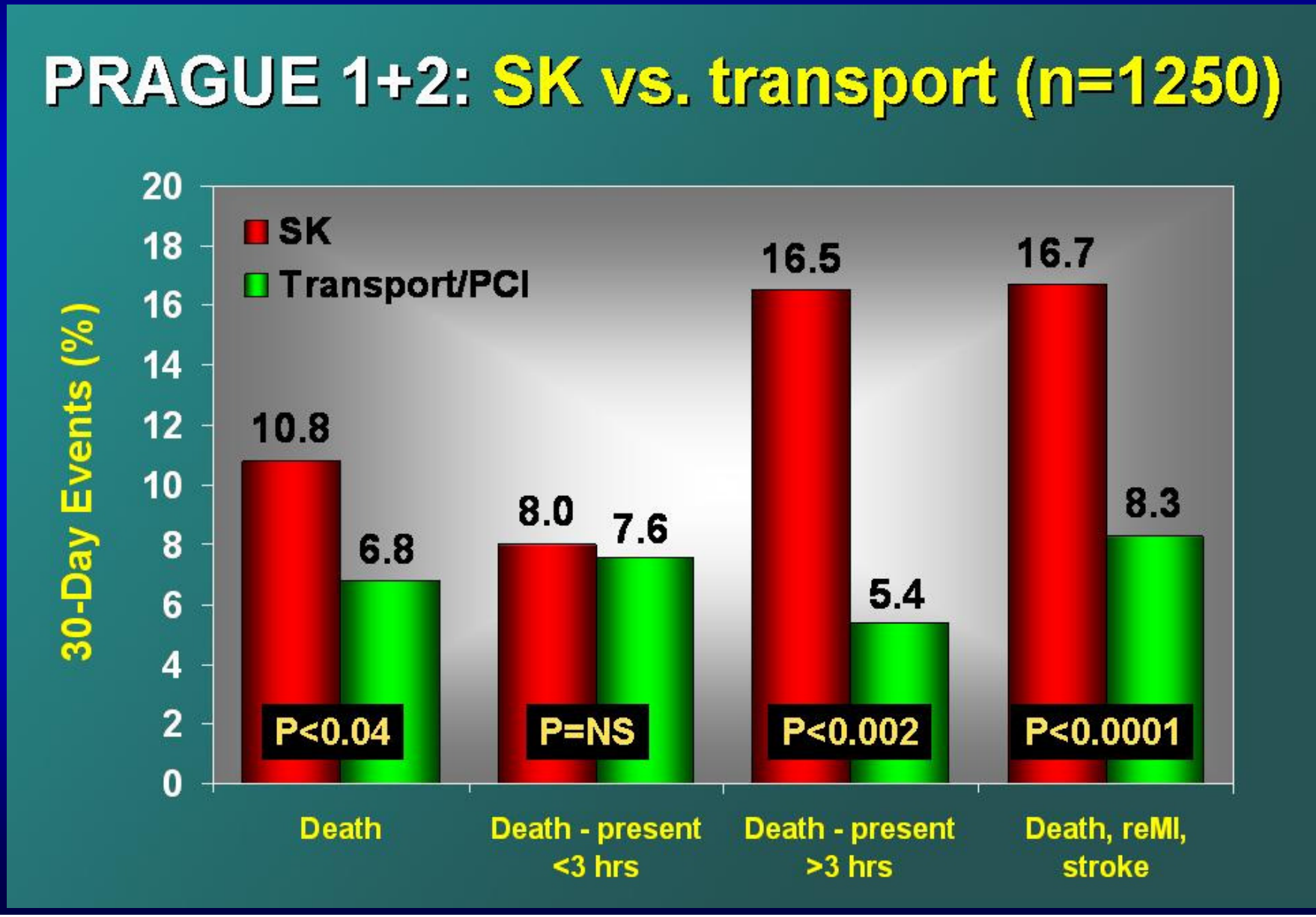
AIR PAMI

DANAMI 2

TROMBOLISIS vs TRANSPORTE PARA PCI
METAANALISIS DE LOS 5 TRIALS



LISIS vs TRANSPORTE PARA PCI



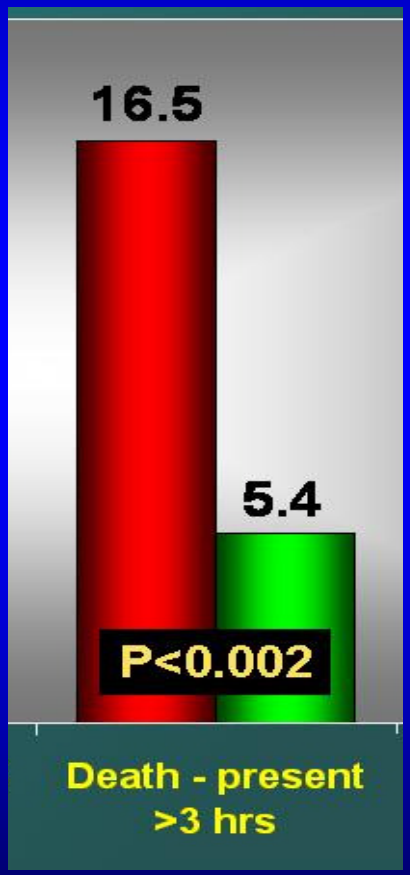
COMO ENFRENTAR EL TRASLADO?

LISIS vs TRANSPORTE PARA PCI
CONCLUSIONES 1

- **Dentro de las primeras 3 horas** la trombolisis es una alternativa viable.

- Ambas estrategias son igualmente efectivas en reducir la mortalidad y el tamaño del IAM

LISIS vs TRANSPORTE PARA PCI
CONCLUSIONES 2



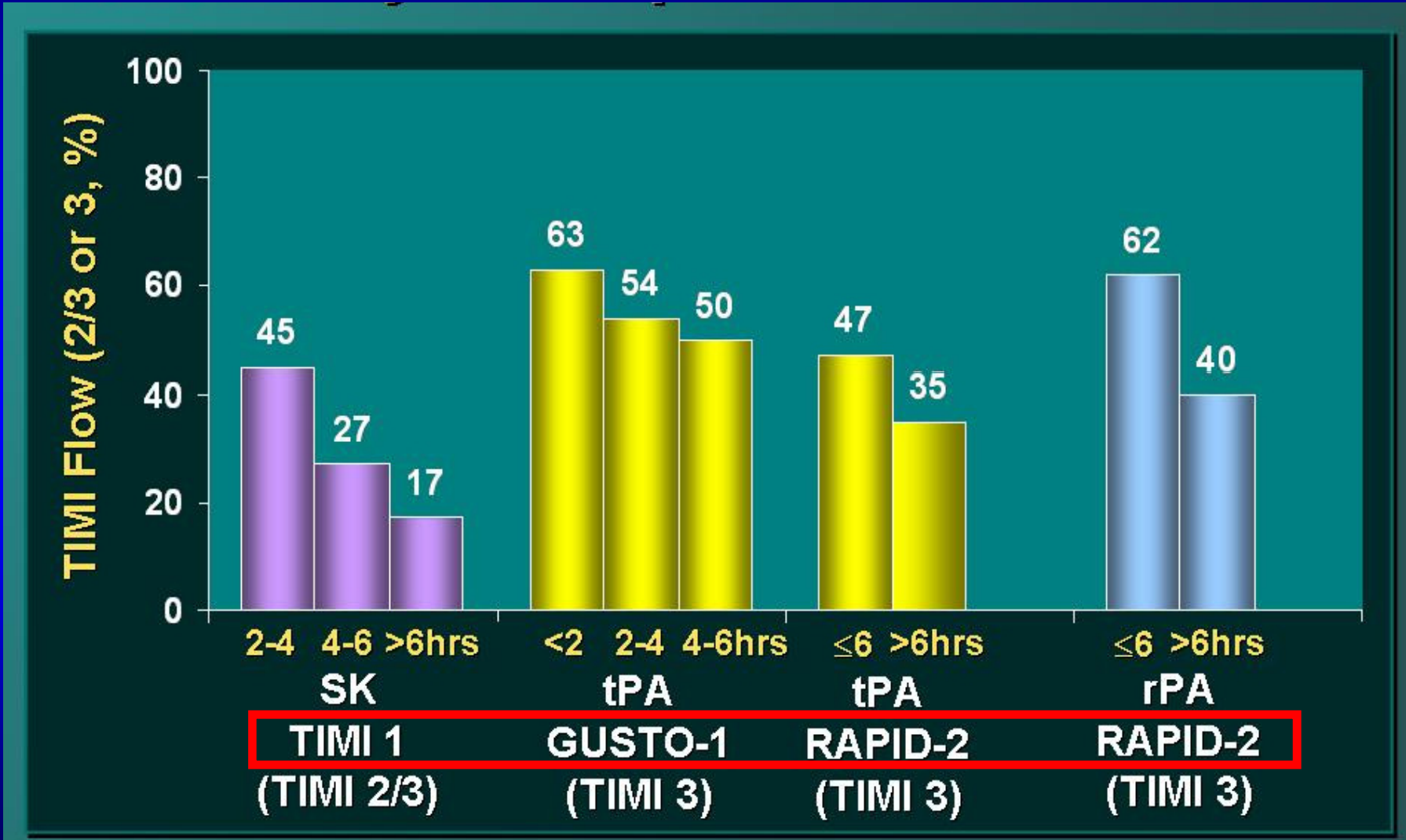
- **Más allá de las primeras 3 horas** la angioplastia disminuye la mortalidad y el tamaño del IAM.

- Además disminuye la incidencia de stroke

LA ANGIOPLASTIA PRIMARIA ES MENOS TIEMPO DEPENDIENTE QUE LA TROMBOLISIS

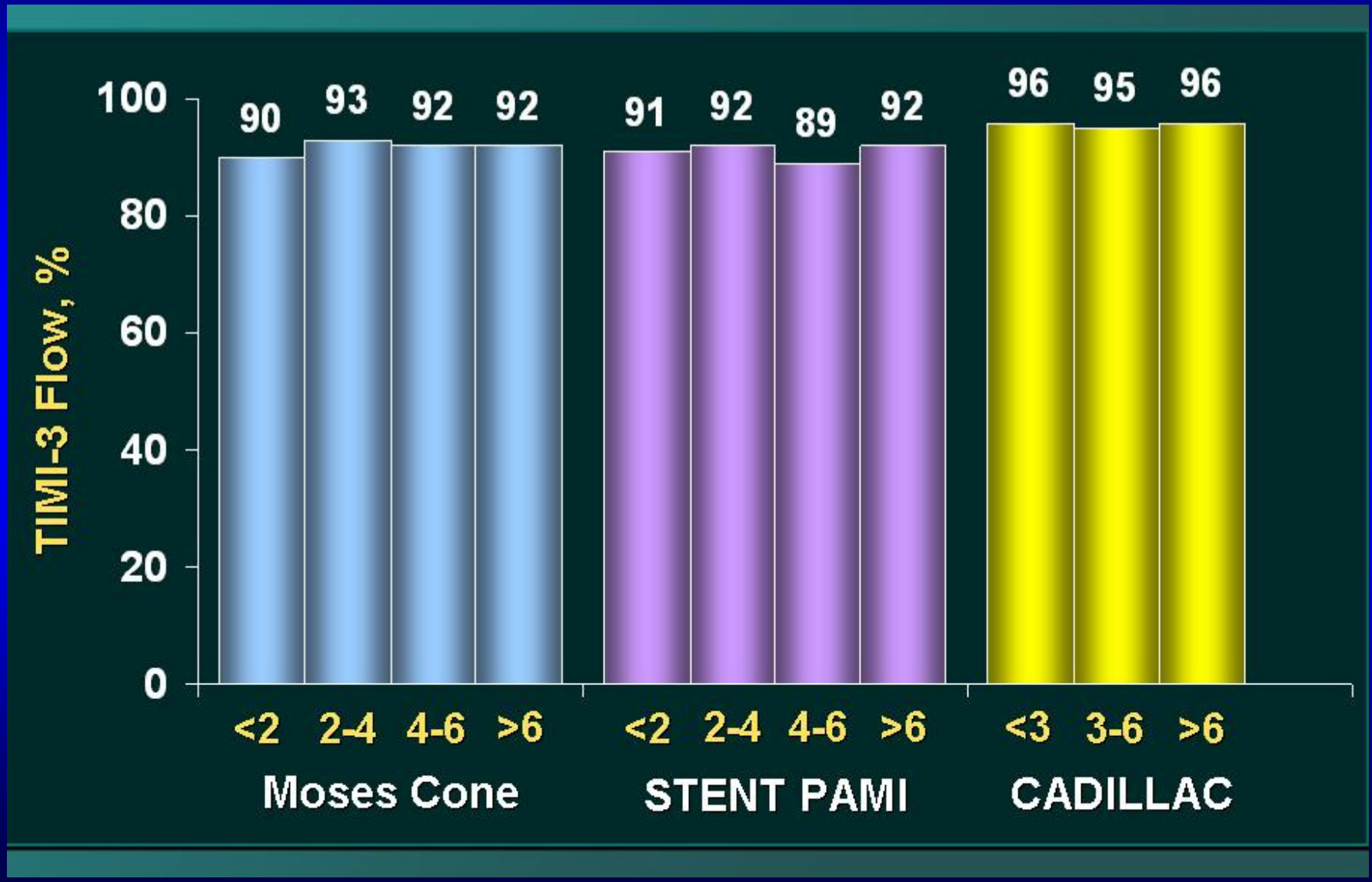
1) Thrombolysis is less effective at restoring infarct artery patency as the clot ages
   - Not so for primary PCI
2) Myocardial salvage and infarct size after thrombolytic therapy is very sensitive to time to reperfusion
   - No so for primary PCI
3) Cardiac rupture is more likley to occur as the time to thrombolysis increases
   - Cardiac rupture is rare after primary PCI

TROMBOLISIS: impacto del tiempo a la aguja



Chesebro JH Circulation 1987;76:142 Genetech GUSTO Database Bode C Circulation 1996;94:891

PCI PRIMARIA: impacto del tiempo al balón



ANGIOPLASTIA DE RESCATE

DEFINICION

Intervención coronaria realizada en el vaso culpable el cual permanece ocluído a pesar de la terapia trombolítica previa.

La trombolisis fallida se sospecha ante la persistencia del dolor de pecho y la NO resolución del ↑ ST 45 a 60 min. luego de comenzada la administración del TBL.

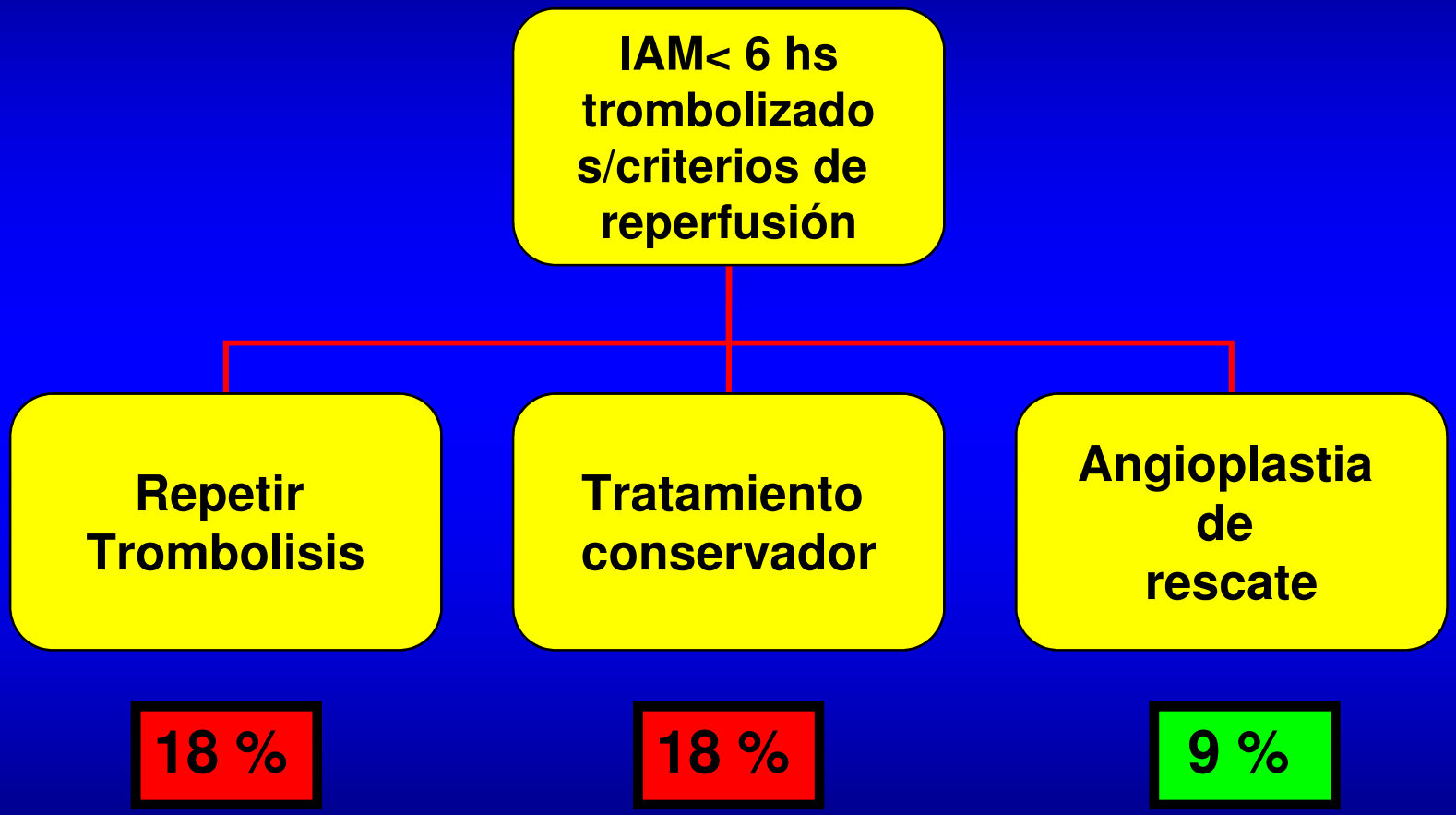
ANGIOPLASTIA DE RESCATE
Estudios randomizados

Randomized trials of intervention for patients with occluded coronary arteries

	Belenkie et al	PRAGUE	RESCUE I	Vermeer et al
No. of patients	28	40	151	149
Fibrinolytic agent(s)	Streptokinase or tPA	Streptokinase	Streptokinase or tPA	tPA
Time symptoms → PTCA (h)	4.3 ± 1.0	–	4.5 ± 1.9	4.0 ± 0.8
Anterior MI (%)	53.6	–	100.0	36.2
Cardiogenic shock (%)	0.0	–	0.0	–
In-hospital or 30-day death				
PTCA group (%)	6.3	20.0	5.1	9.4
Medical group (%)	33.3	18.0	9.6	6.7
In-hospital or 30-day repeat MI				
PTCA group (%)	–	0.0	–	5.4
Medical group (%)	–	9.0	–	12.0
30-day CHF class III-IV				
PTCA group (%)	–	15.0	1.3	–
Medical group (%)	–	27.0	7.0	–
6-Mo reintervention				
PTCA group (%)	–	–	16.6	27.0
Medical group (%)	–	–	23.3	33.3

ANGIOPLASTIA DE RESCATE

REACT Trial



Mortalidad a los 6 meses

NEJM 2005 353, 2758

TROMBOLITICOS (contraindicaciones)

ABSOLUTAS
Stroke hemorrágico previo en cualquier momento
Stroke isquémico en los últimos 6 meses
Neoplasia o daño en el SNC
Injuria en la cabeza/trauma mayor/cirugía dentro de las 3 semanas precedentes
Sangrado gastrointestinal en el último mes
Desorden que predisponga al sangrado conocido
Disección aórtica
Punciones no compresibles en las últimas 24 horas (biopsia hígado, punción lumbar)
RELATIVAS
AIT en los últimos 6 meses
Tratamiento con anticoagulantes orales
Embarazo o 1º semana post parto
HTA refractaria
Enfermedad hepática avanzada
Endocarditis infecciosa
Ulcera péptica activa
RCP traumática o prolongada

TIPO Y DOSIS DE AGENTES TROMBOLITICOS

	Initial treatment	Specific contraindications
Streptokinase (SK)	1.5 million units over 30–60 min i.v.	Prior SK or anistreplase
Alteplase (tPA)	15 mg i.v. bolus 0.75 mg/kg over 30 min (up to 50 mg) then 0.5 mg/kg over 60 min i.v. (up to 35 mg)	
Reteplase (r-PA)	10 units + 10 units i.v. bolus given 30 min apart	
Tenecteplase (TNK–tPA)	Single i.v. bolus: 30 mg if <60 kg 35 mg if 60 to <70 kg 40 mg if 70 to <80 kg 45 mg if 80 to <90 kg 50 mg if ≥90 kg	

The New England
Journal of Medicine

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Volume 329 SEPTEMBER 2, 1993 Number 10

AN INTERNATIONAL RANDOMIZED TRIAL COMPARING FOUR THROMBOLYTIC STRATEGIES FOR ACUTE MYOCARDIAL INFARCTION

THE GUSTO INVESTIGATORS*

Abstract *Background.* The relative efficacy of streptokinase and tissue plasminogen activator and the roles of intravenous as compared with subcutaneous heparin as adjunctive therapy in acute myocardial infarction are unresolved questions. The current trial was designed to compare new, aggressive thrombolytic strategies with standard thrombolytic regimens in the treatment of acute myocardial infarction. Our hypothesis was that newer thrombolytic strategies that produce earlier and sustained reperfusion would improve survival.

Methods. In 15 countries and 1081 hospitals, 41,021 patients with evolving myocardial infarction were randomly assigned to four different thrombolytic strategies, consisting of the use of streptokinase and subcutaneous heparin, streptokinase and intravenous heparin, accelerated tissue plasminogen activator (t-PA) and intravenous heparin, or a combination of streptokinase plus t-PA with intravenous heparin. ("Accelerated" refers to the administration of t-PA over a period of 1½ hours — with two thirds of the dose given in the first 30 minutes — rather than the conventional period of 3 hours.) The primary end point was 30-day mortality.

Results. The mortality rates in the four treatment groups were as follows: streptokinase and subcutaneous heparin, 7.2 percent; streptokinase and intravenous heparin, 7.4 percent; accelerated t-PA and intravenous heparin, 6.3 percent; and the combination of both thrombolytic agents with intravenous heparin, 7.0 percent. This represented a 14 percent reduction (95 percent confidence interval, 5.9 to 21.3 percent) in mortality for accelerated t-PA as compared with the two streptokinase-only strategies ($P = 0.001$). The rates of hemorrhagic stroke were 0.49 percent, 0.54 percent, 0.72 percent, and 0.94 percent in the four groups, respectively, which represented a significant excess of hemorrhagic strokes for accelerated t-PA ($P = 0.03$) and for the combination strategy ($P<0.001$), as compared with streptokinase only. A combined end point of death or disabling stroke was significantly lower in the accelerated–t-PA group than in the streptokinase-only groups (6.9 percent vs. 7.8 percent, $P = 0.006$).

Conclusions. The findings of this large-scale trial indicate that accelerated t-PA given with intravenous heparin provides a survival benefit over previous standard thrombolytic regimens. (N Engl J Med 1993;329: 673-82.)

TIPO Y DOSIS DE AGENTES TROMBOLITICOS

ESTREPTOQUINASA STREPTASE P.R.

*Tipo	Importado
*Droga	Estreptoquinasa
*Acción	Fibrinolítico
• 1500000UI Iny. Fco.	$ 3453.92
10.sep.14	

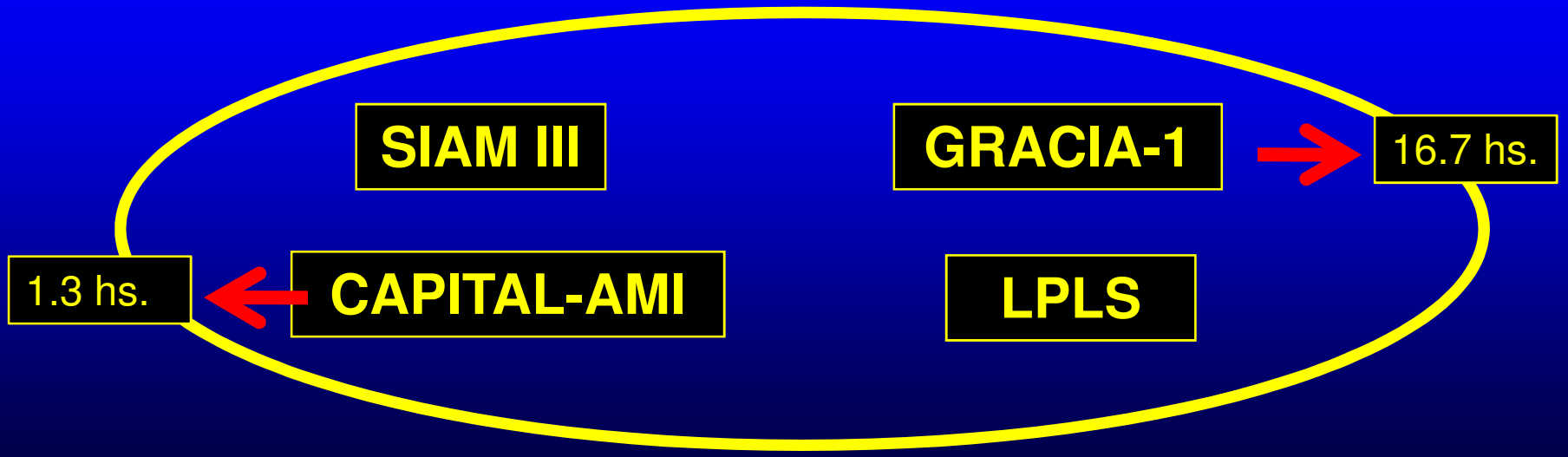
ACTILYSE P.R.

*Tipo	
*Droga	Alteplasa
*Acción	Trombolítico
• F.Amp. x 2	$ 9129.03
09.sep.14	

CONDUCTA ANTE LA TROMBOLISIS EXITOSA

El "talón de Aquiles" de la trombolisis es el Reinfarto

Cuatro estudios randomizados han contribuido a recomendar CCG de rutina post TBL, y si está indicada PCI temprana entre 3-24 hs post TBL.



CONDUCTA ANTE LA TROMBOLISIS EXITOSA

Cuatro estudios más recientes confirmaron la hipótesis

CARESS in AMI	Incluyó 600 p. Francia, Italia y Polonia *Lancet 2008;371(9612):559–568.*
TRANSFER AMI	Incluyó 1059 p en Canadá *N Engl J Med 2009;360(26):2705–2718*
NORDISTEMI	Incluyó 266 p en Noruega *J Am Coll Cardiol 2010;55(2):102–110*
STREAM	Incluyó 1892 p en Bélgica *N Engl J Med 2013;368(15): 1379–1387*

European Heart Journal (2010) **31**, 2156–2169
doi:10.1093/eurheartj/ehq204

CLINICAL RESEARCH
Interventional cardiology

Early routine percutaneous coronary intervention after fibrinolysis vs. standard therapy in ST-segment elevation myocardial infarction: a meta-analysis

Francesco Borgia[1,5], Shaun G. Goodman[2], Sigrun Halvorsen[3], Warren J. Cantor[4], Federico Piscione[5], Michel R. Le May[6], Francisco Fernández-Avilés[7], Pedro L. Sánchez[7], Konstantinos Dimopoulos[1], Bruno Scheller[8], Paul W. Armstrong[9], and Carlo Di Mario[1]*

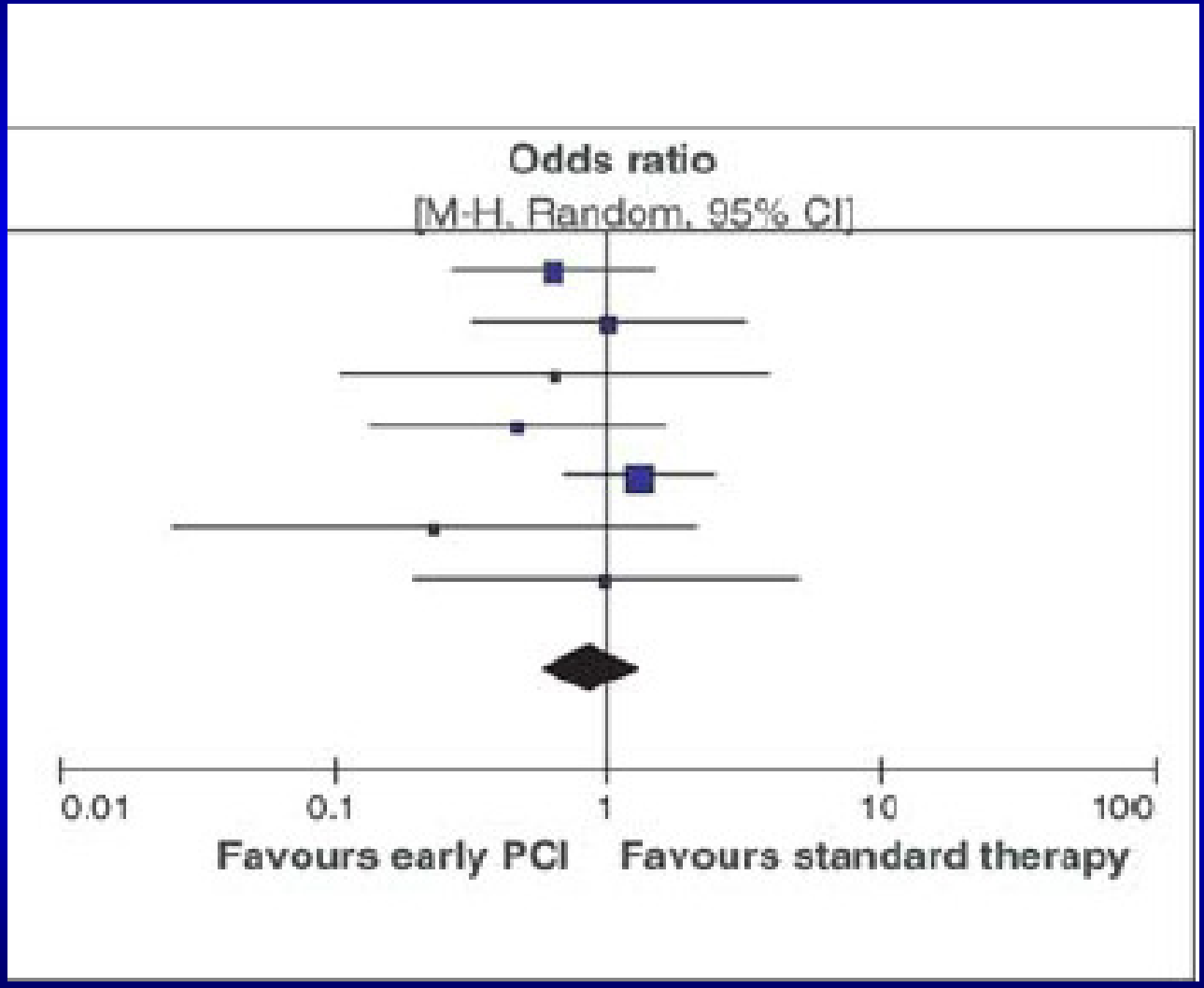
[1]Royal Brompton Hospital and Imperial College, London, UK; [2]Canadian Heart Research Centre and St. Michael's Hospital, University of Toronto, Toronto, Canada; [3]Oslo University Hospital, Ulleval, Oslo, Norway; [4]Southlake Regional Health Centre, Newmarket, ON, Canada; [5]Federico II University, Naples, Italy; [6]Heart Institute, Univeristy of Ottawa, Ottawa, ON, Canada; [7]Hospital General Universitario Gregorio Marañón, Madrid, Spain; [8]Innere Medizin III, Universitat des Saarlandes, Homburg, Germany; and [9]Canadian VIGOUR Centre, University of Alberta, Edmonton, AB, Canada

Received 1 February 2010; revised 10 April 2010; accepted 26 April 2010; online publish-ahead-of-print 2 July 2010

Study (year)	Inclusion criteria	Lytic agent (type)	Strategy	Symptoms to lytic therapy (min)	Lytic therapy to early PCI (min)	Primary endpoint
CARESS-IN-AMI (2008)	High-risk STEMI patients presenting <12 h from symptom onset.	r-PA (half dose)[a]	Standard therapy (301 patients) (rescue PCI in 31%)	165[b]	—	Combined death, reinfarction and recurrent ischaemia at 30 days.
			Immediate PCI after lysis (299 patients) (PCI in 86%)	161[b]	125[b]	
GRACIA-1 (2004)	STEMI patients presenting <12 h from symptoms onset.	Alteplase (accelerated dose)	Standard therapy (251 patients) (rescue PCI in 12%)	187	—	Combined death, reinfarction, ischaemic-induced revascularization at 12 months.
			Early PCI <6–24 h from lysis (248 patients) (PCI in 80%)	180	1002	
CAPITAL-AMI (2005)	High-risk STEMI patients presenting ≤6 h from symptom onset.	TNK	Standard therapy (84 patients) (rescue PCI in 9.5%)	120[b]	—	Combined death, reinfarction, ischaemic events or stroke at 6 months.
			Early PCI <3 h from lysis (86 patients) (PCI in 89%)	120[b]	84[b,c]	
SIAM-III (2003)	STEMI patients presenting <12 h from symptom onset.	r-PA	Standard therapy (81 patients) (rescue PCI in 12%)	216	—	Combined death, reinfarction, recurrent ischaemia, target lesion revascularization at 6 months.
			Early PCI <6 h from lysis (82 patients) (PCI in 100%)	192	210	
TRANSFER-AMI (2009)	High-risk STEMI patients presenting <12 h from symptom onset.	TNK	Standard therapy (522 patients) (rescue PCI in 25%)	115[b]	—	Combined death, reinfarction, recurrent ischaemia, new CHF, cardiogenic shock at 30 days.
			Early PCI <6 h from lysis (537 patients) (PCI in 85%)	113[b]	234[b]	
WEST (2006)[e]	STEMI patients presenting <6 h from symptom onset.	TNK	Standard therapy (100 patients) (rescue PCI in 14%)	113[b]	—	Combined death, reinfarction, recurrent ischaemia, new CHF, cardiogenic shock, and maior ventricular arrhythmia at 30 days.
			Early PCI <24 h from lysis (104 patients)[d] (PCI in 86%)	130[b]	295[b,c]	
NORDISTEMI (2010)	STEMI patients presenting <6 h from symptom onset.	TNK	Standard therapy (132 patients) (rescue PCI in 27%)	126[b]	—	Combined death, reinfarction, recurrent ischaemia, or stroke at 12 months.
			Immediate PCI after lysis (134 patients) (PCI in 86%)	117[b]	163[b]	

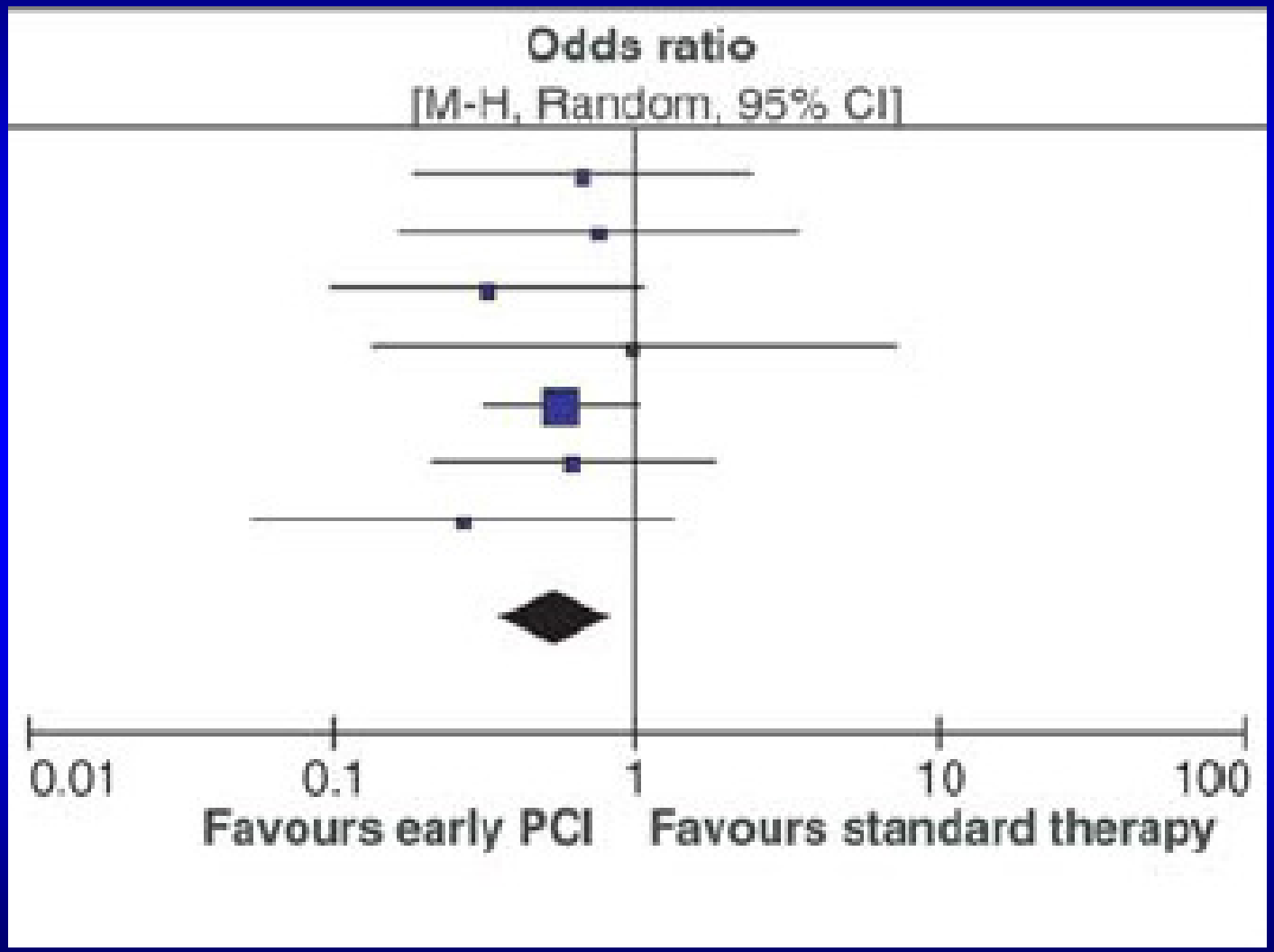
Eur Heart J 2010;31(17):2156–2169

MUERTE A 30 DIAS



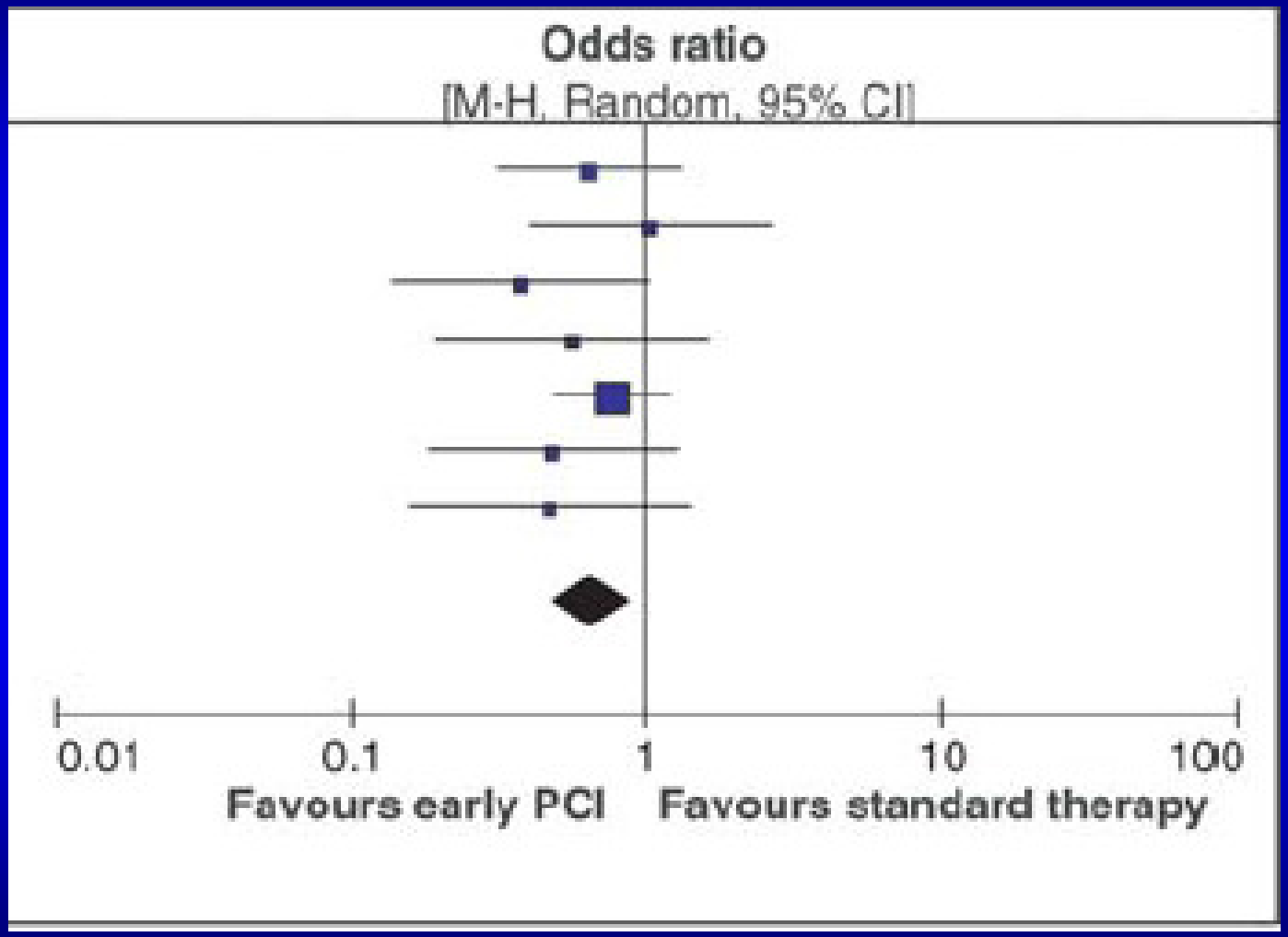
Eur Heart J 2010;31(17):2156–2169

RE IAM A 30 DIAS



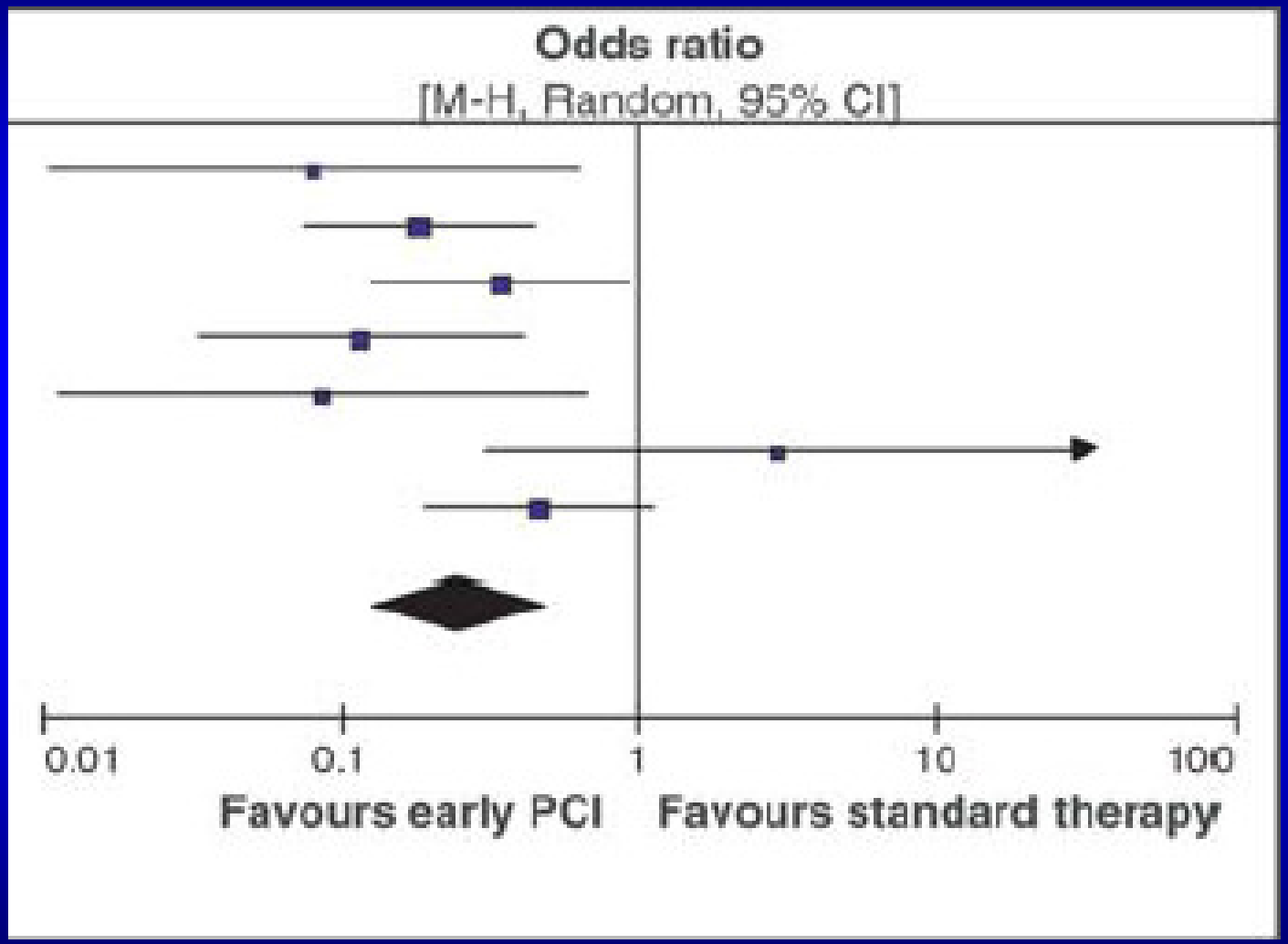
Eur Heart J 2010;31(17):2156–2169

MUERTE Y RE IAM A 30 DIAS



Eur Heart J 2010;31(17):2156–2169

RE ISQUEMIA A 30 DIAS



Eur Heart J 2010;31(17):2156–2169

CONCLUSIONES (meta-análisis)

- La ICPP post TBL en pacientes con STEMI reduce significativamente el reinfarto y la isquemia recurrente al mes, sin incremento significativo en sangrados comparado a la terapia standard.

- Los beneficios de la ICPP temprana post TBL persisten en el seguimiento a 6-12 meses.

Eur Heart J 2010;31(17):2156–2169

OBJETIVOS DEL TRATAMIENTO EN EL IAM

REPERFUNDIR MAS PACIENTES

REPERFUNDIR MAS RAPIDO

REPERFUNDIR LO + COMPLETA/ POSIBLE

REGENERAR EL TEJIDO NECROSADO

OBJETIVOS DEL TRATAMIENTO EN EL IAM

REPERFUNDIR MAS PACIENTES

REPERFUNDIR MAS RAPIDO

REPERFUNDIR LO + COMPLETA/ POSIBLE

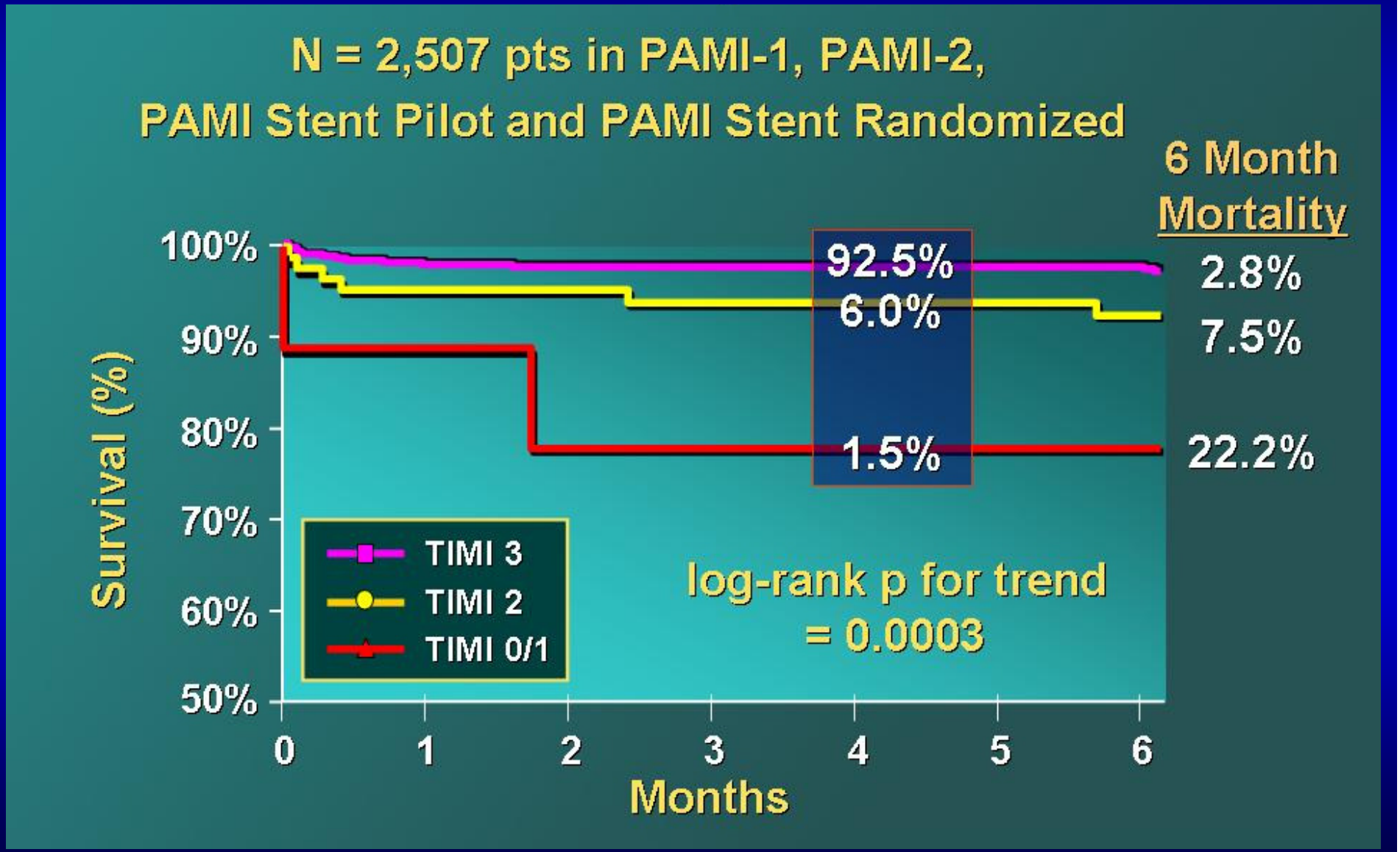
REGENERAR EL TEJIDO NECROSADO

REPERFUSION EN EL IAM

REPERFUSION EFECTIVA

Reperfusión precoz (60-90') con flujo TIMI 3 y sostenida en el tiempo.

EFECTO DEL FLUJO TIMI EPICARDICO POST PCI



EFECTO DEL FLUJO TIMI EPICARDICO POST PCI

Ahora bien:

- El flujo TIMI 3 es sinónimo de **adecuada recanalización** de la arteria infartada
- Pero, es sinónimo de **adecuada reperfusión miocárdica** de la arteria infartada ?

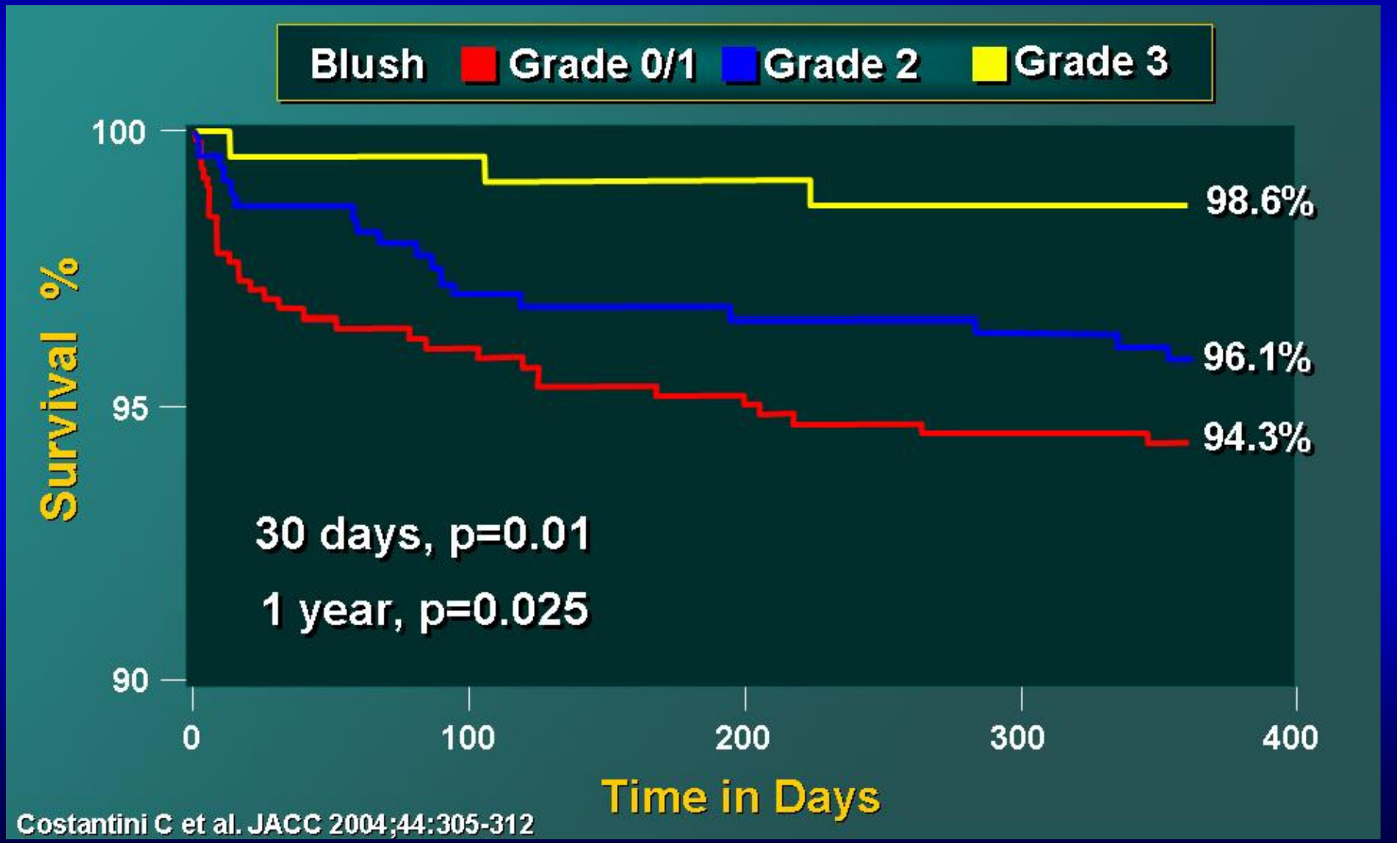
NO

GRADOS DE PERFUSION MIOCARDICA

- **TMP 0 : ausencia de impregnación del miocardio**
- **TMP 1 : impregnación del miocardio que no lava (presente en la próxima inyección).**
- **TMP 2 : impregnación del miocardio que lava lentamente (más de tres ciclos cardíacos)**
- **TMP 3 : impregnación del miocardio que lava totalmente en 3 ciclos cardíacos.**

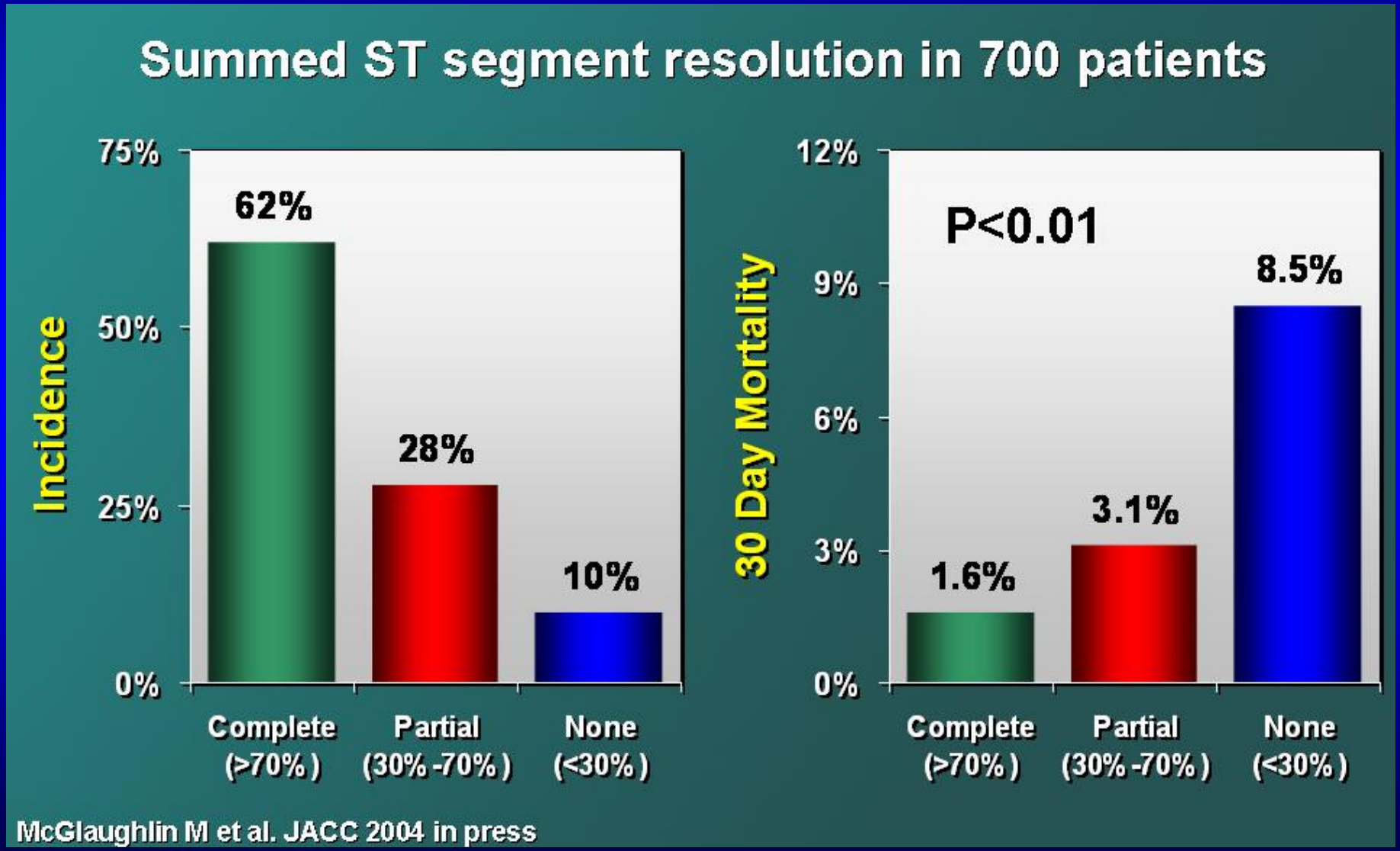
Gibson C M et al.Circulation 2000; 101:125

MORTALIDAD SEGÚN EL BLUSH EN PACIENTES CON FLUJO TIMI 3

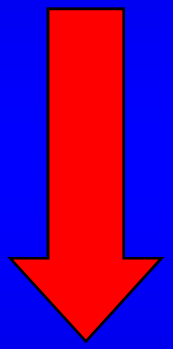


Costantini C et al. JACC 2004;44:305-312

CADILLAC: Impacto de la resolución del ST



ADECUADA RECANALIZACION (FLUJO TIMI 3) SIN REPERFUSION MIOCARDICA



FENOMENO DE NO REFLOW

AFECTA AL 10-20 % DE LOS IAM

CAUSAS DEL FENOMENO DE NO REFLOW POST PCI

- Tromboembolismo distal
- Vasoespasmo (sustancias neurohumorales)
- Inflamación
- Edema intersticial
- Mionecrosis con daño capilar

MANEJO DEL TROMBO ASOCIADO A LA LESION

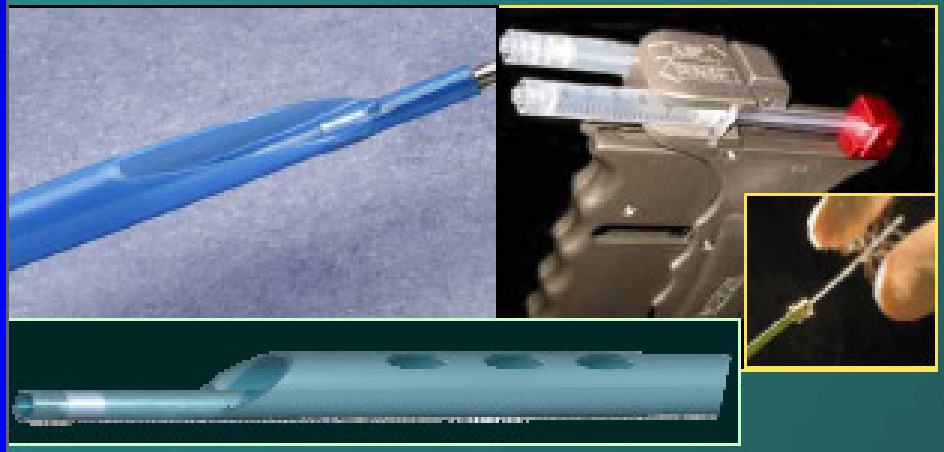
A pesar de los importantes avances en el desarrollo de agentes antiplaquetarios y antitrombóticos, la persistencia de remanentes de trombo en las lesiones está fuertemente asociada con un alto riesgo de embolia distal y el fenómeno de No reflow.

ABORDAJES MECANICOS

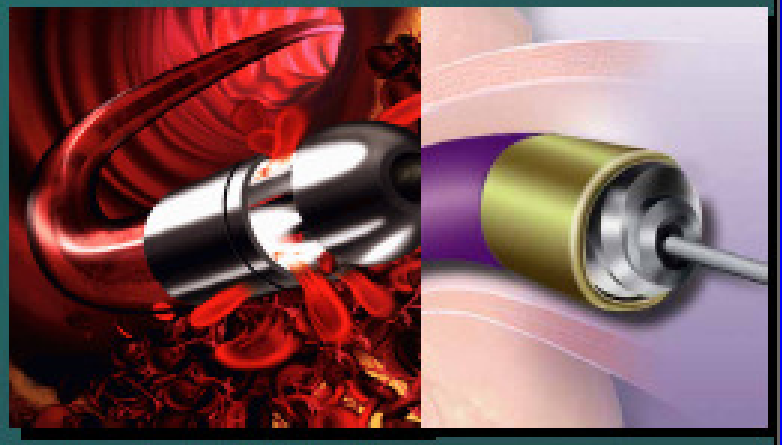
- CATETERES DE TROMBO ASPIRACION MANUAL
- REOLÍTICOS
- LISIS POR ULTRASONIDO
- PROTECCION DISTAL
- TROMBECTOMIA MECANICA

Mechanical Approaches to Thrombus

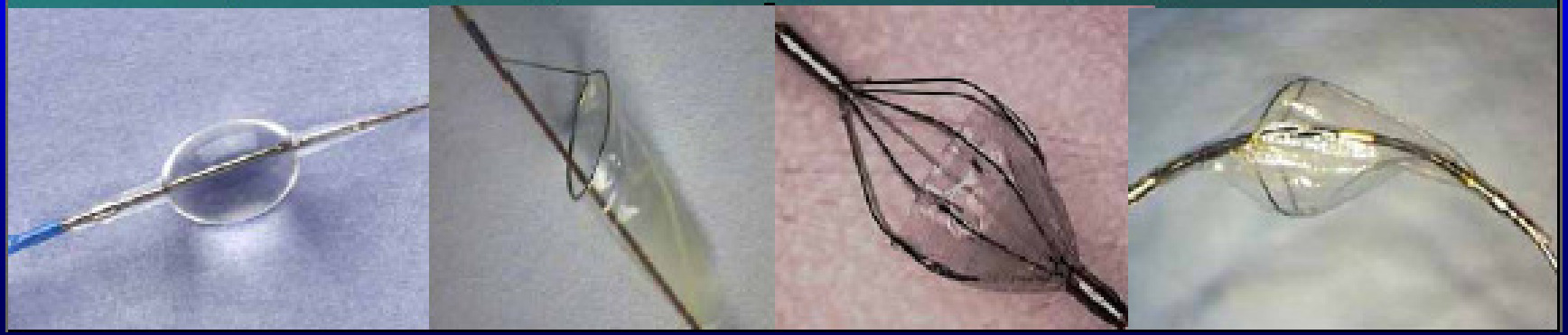
Thrombus aspiration
(Rinspirator, Pronto, Export, Rescue, Diver CE, etc.)



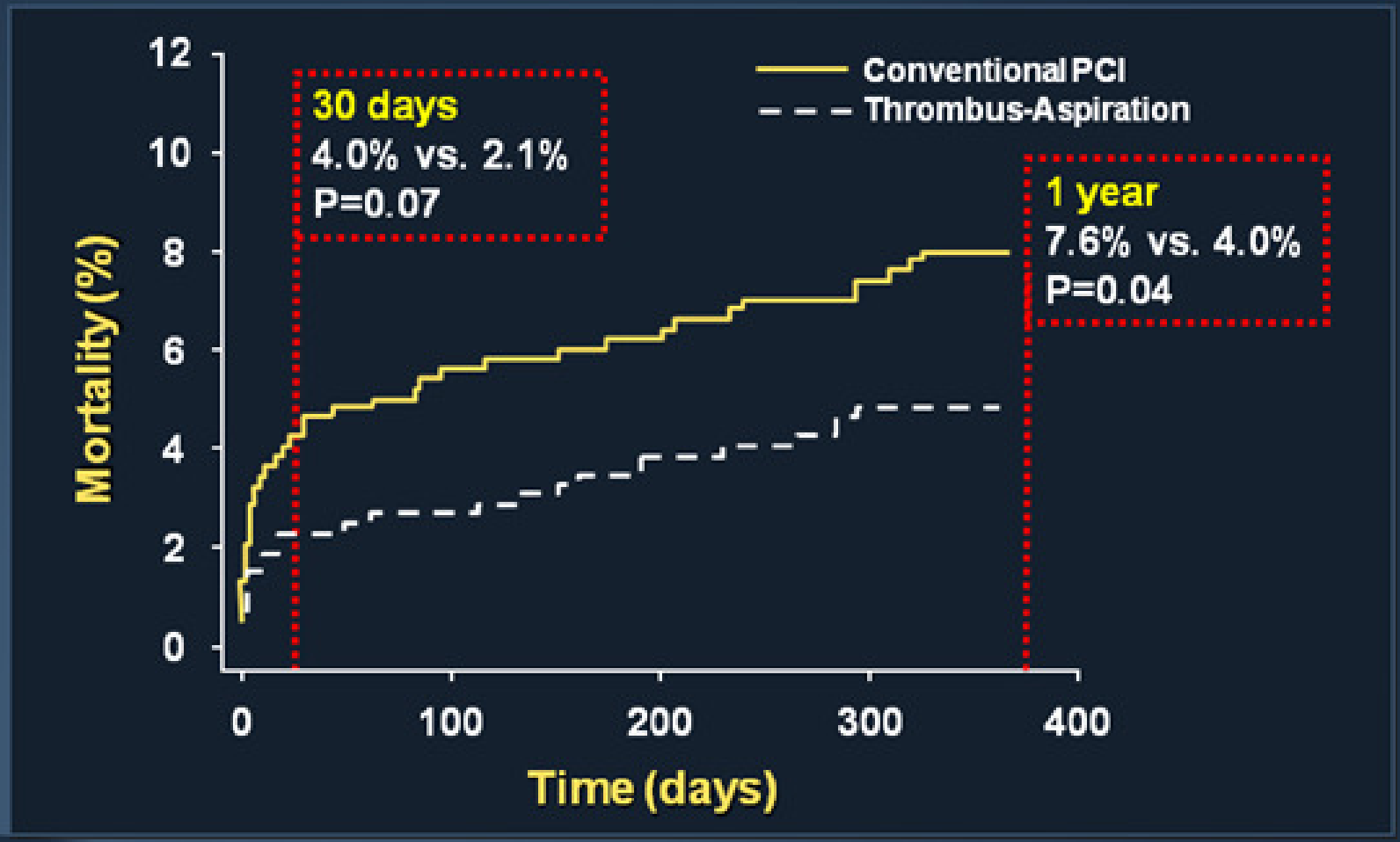
Thrombectomy
(AngioJet, X-Sizer)



Distal protection (GuardWire, FilterWire, AngioGuard, etc.)



TAPAS: 1,071 pts with STEMI undergoing primary PCI randomized in the ER to manual aspiration (Export) vs. control



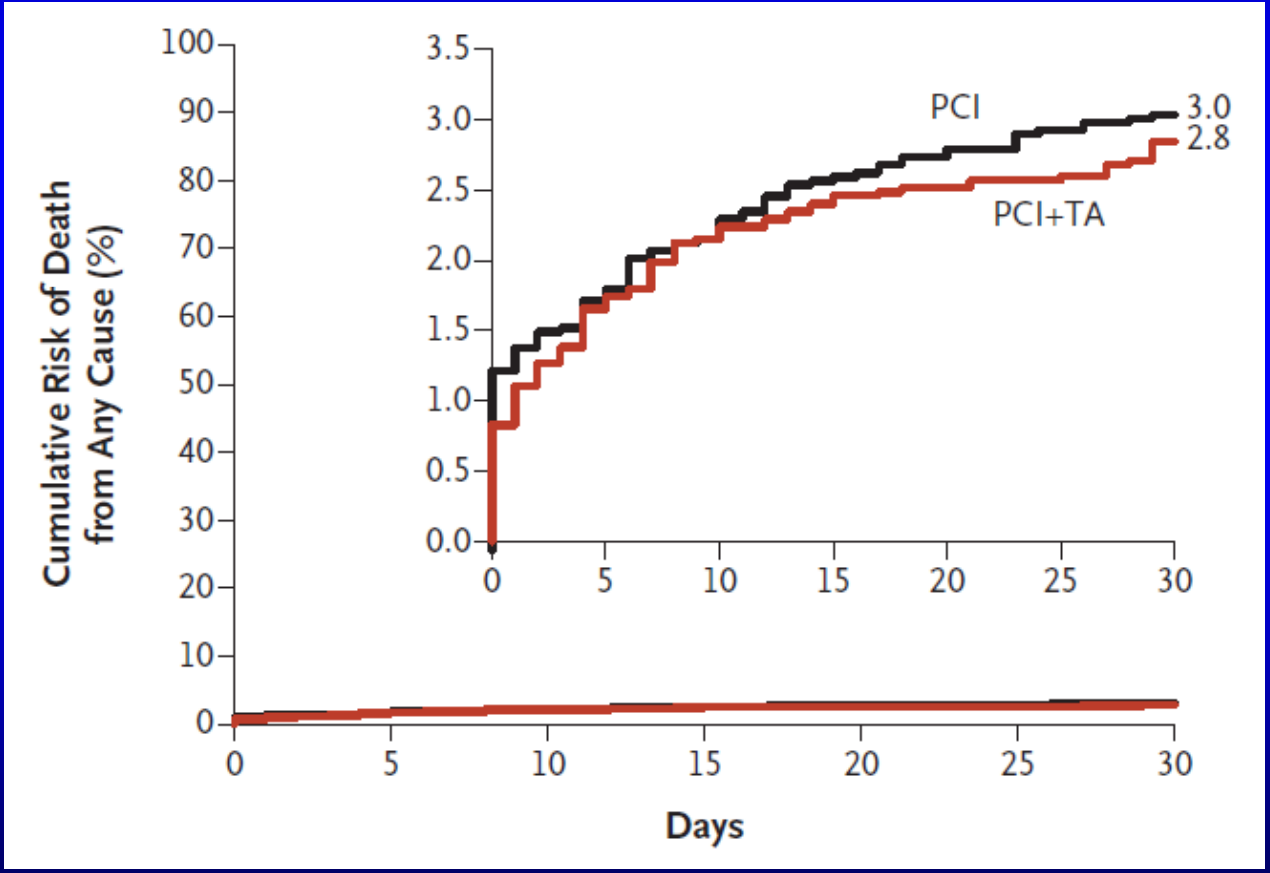
Vlaar et al. Lancet 2008;371:1915-20

CARDIOVASCULAR RESEARCH FOUNDATION

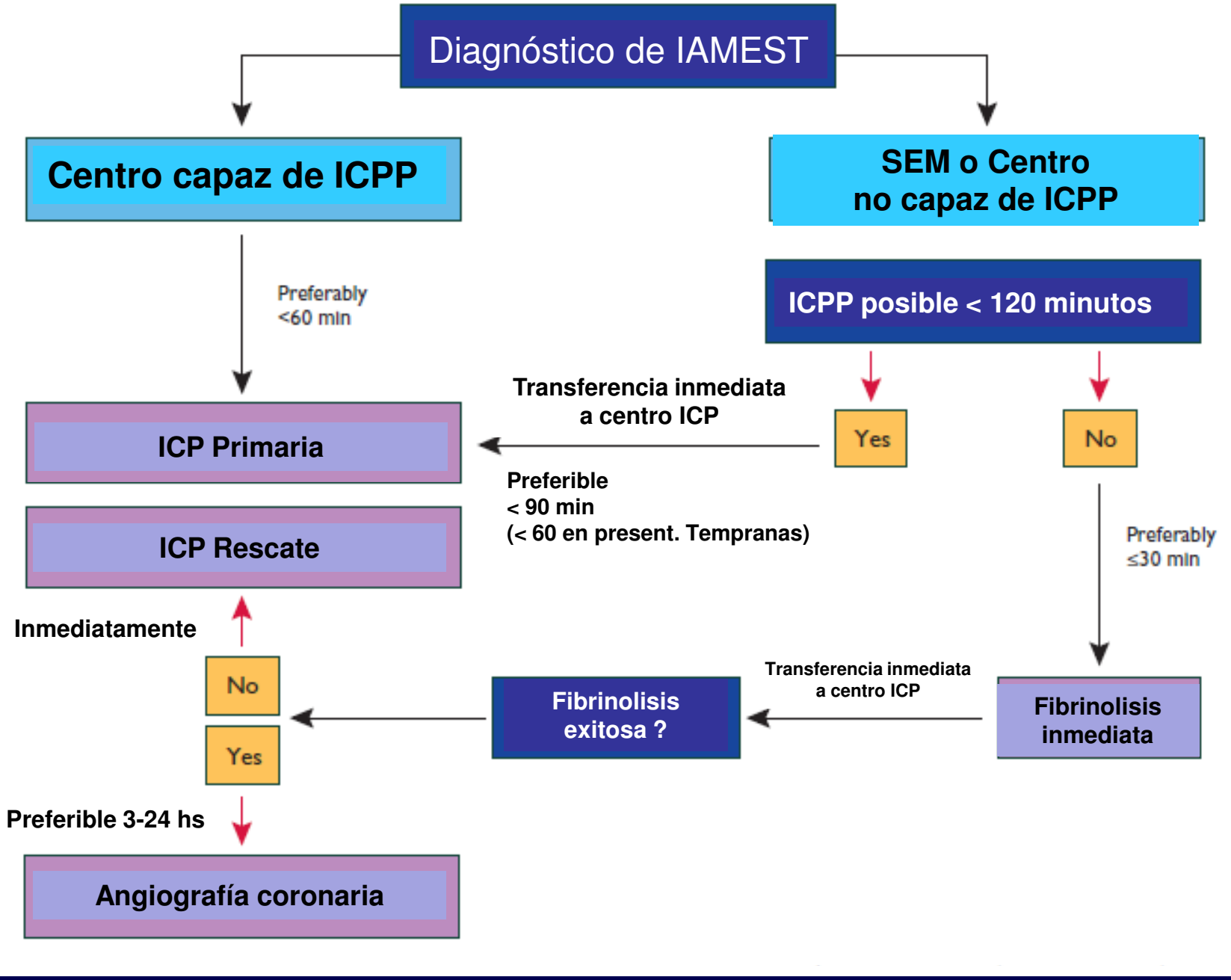
COLUMBIA UNIVERSITY MEDICAL CENTER

Thrombus Aspiration during ST-Segment Elevation Myocardial Infarction

TASTE TRIAL

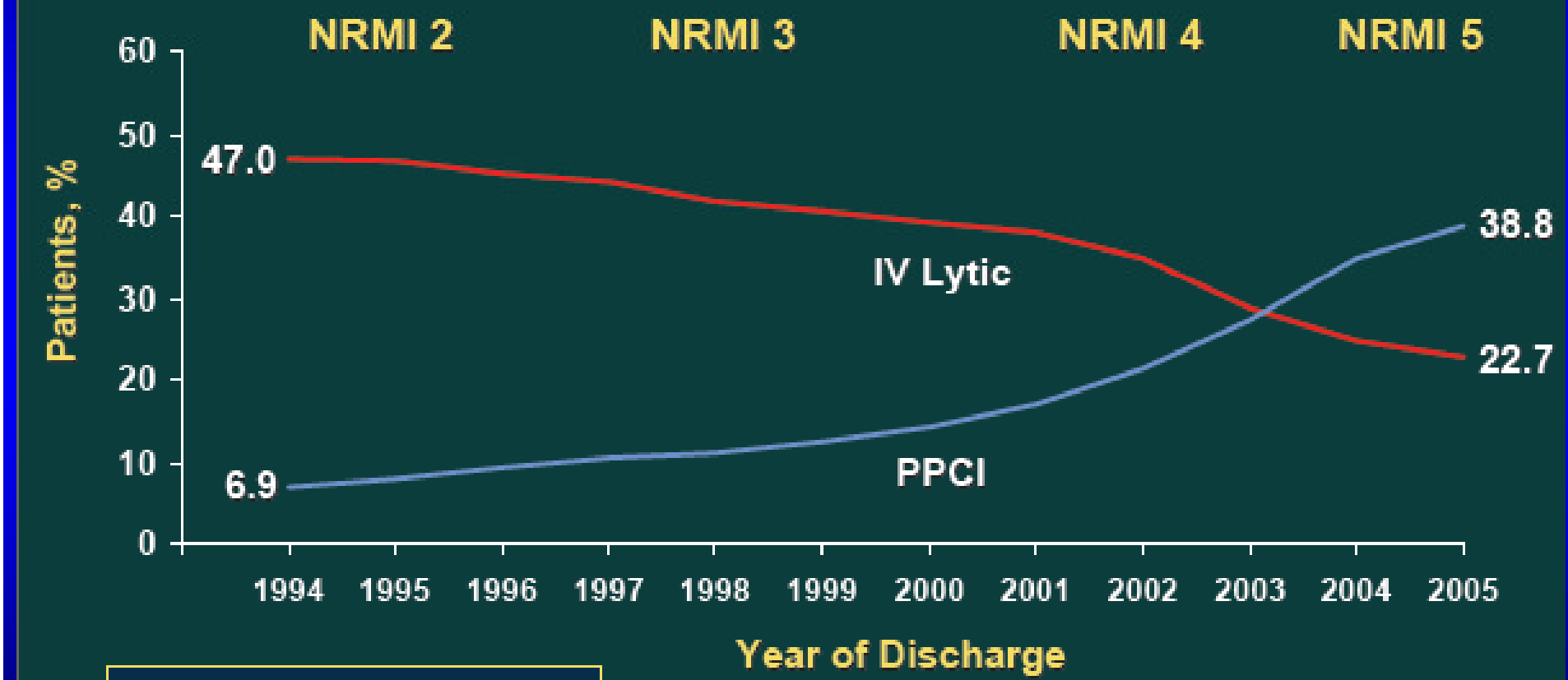


N Engl J Med 2013;369(17):1587–1597.


Diagnóstico de IAMEST
Centro capaz de ICPP
SEM o Centro no capaz de ICPP
Preferably <60 min
ICPP posible < 120 minutos
Transferencia inmediata a centro ICP
Yes
No
ICP Primaria
Preferible < 90 min (< 60 en present. Tempranas)
ICP Rescate
Preferably ≤30 min
Inmediatamente
Transferencia inmediata a centro ICP
No
Fibrinolisis exitosa ?
Fibrinolisis inmediata
Yes
Preferible 3-24 hs
Angiografía coronaria

Initial Reperfusion Therapy

(All STEMI patients)



Immediate CABG – Range
0.9 % – 1.7 %

ASPECTOS A CONSIDERAR
ANTE LA DERIVACION A HEMODINAMIA

ASPECTOS A CONSIDERAR ANTE LA DERIVACION A HEMODINAMIA

Pretratamiento antes de la derivación

- Derivar al paciente con una vía periférica confiable
- Soporte con O2 cuando se considere necesario
- Tratamiento del dolor con analgésicos opiáceos
- **Aspirina** (según la S.E.C. 150 a 300 mg de carga V.O.)
- **Clopidogrel** (carga de 600 mg)
- Si es factible se prefiere Prasugrel (carga de 60 mg) o Ticagrelor (carga de 180 mg)
- **Heparina 5000 U.I.**
- Resumen de H.C.
- Laboratorio en caso que esté disponible (U y Cr)

CARACTERISTICAS DE LA ICPP EN EL HOSPITAL CENTENARIO

- De rutina **acceso femoral**
- Si el paciente tiene TIMI 0-1 luego de cruzar la lesión responsable con la guía se realizar **TAM**
- Si se constata monto trombótico importante a pesar de la TAM se comienza tratamiento con inhibidores de la GP IIB-IIIA (**Eptifibatide** doble bolo e infusión contínua)
- De **rutina** implante de **stent metálico** (NO DES)
- Solo **ICPP de la arteria culpable** a excepción del shock cardiogénico o persistencia del dolor y ECG luego de resolver la arteria considerada responsable

ASPECTOS A CONSIDERAR AL REGRESO DE HEMODINAMIA

- Control estricto de **TA y FC**
- Control estricto del **introductor** y de los **pulsos distales**
- **ECG post ICPP** al regreso al Hospital
- **Soporte con O2** cuando se considere necesario
- **Extracción del introductor** según especificación de HIA (en general son 6 horas post refuerzo de heparina en Sala)
- Puede tener dieta líquida y/o liviana antes del retiro del introductor
- Comenzar tratamiento adyuvante correspondiente
- Continuar la doble antiagregación **al día siguiente**

TECNICAS ESPECIALES - FUTURO

- HIPOTERMIA CIRCULATORIA
- HIPEROXIGENACION
- PREVENCION DEL DAÑO POR REPERFUSION

  Fluosol, Mg, trimetazidina, adenosina, anti CD 18, eniporida, etc.

- REGENERACION DE TEJIDO NECRÓTICO

  Administración de stem cell

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Time Is Muscle

Translation Into Practice

Elliott M. Antman, MD, FACC

Boston, Massachusetts

In the future, advances in the care of patients with ST-segment elevation myocardial infarction (STEMI) will not come from the analysis of trials that do not reflect current practice in an effort to rationalize extending the percutaneous coronary intervention (PCI)-related delay time. We must move beyond such arguments and find ways to shorten total ischemic time. With the launching of the American College of Cardiology's D2B Alliance and the American Heart Association's Mission: Lifeline programs, the focus is now on systems improvement for reperfusion in patients with STEMI. The D2B Alliance was developed to focus on improvement in door-to-balloon times for patients with STEMI who are undergoing primary PCI. The American Heart Association Mission: Lifeline program is a broad, comprehensive national initiative to improve the quality of care and outcomes of patients with STEMI by improving health care system readiness and response to STEMI. Improvements in access to timely care for patients with STEMI will require a multifaceted approach involving patient education, improvements in the Emergency Medical Services and emergency department components of care, the establishment of networks of STEMI-referral hospitals (not PCI capable) and STEMI-receiving hospitals (PCI capable), as well as coordinated advocacy efforts to work with payers and policy makers to implement a much-needed health care system redesign. By focusing now on system efforts for improvements in timely care for STEMI, we will complete the cycle of research initiated by Reimer and Jennings 30 years ago. Time is muscle . . . we must translate that into practice. (J Am Coll Cardiol 2008;52:1216–21) © 2008 by the American College of Cardiology Foundation

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Time Is Muscle

En el futuro, los avances en la atención de pacientes con infarto con elevación del segmento ST (STEMI) vendrá a partir del análisis de los ensayos que reflejen la práctica actual en un esfuerzo por racionalizar la ampliación de la intervención coronaria percutánea relacionada con el tiempo de retardo.

Debemos ir más allá de todos los argumentos y encontrar formas de acortar el tiempo de isquemia total.

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Las mejoras en el acceso a la atención adecuada de los pacientes con STEMI requieren un enfoque multifacético que involucre la educación del paciente, las mejoras en los componentes de los SEM y el establecimiento de una red de hospitales de referencia (no ICP en STEMI) y (si ICP en STEMI)

the Emergency Medical Services and emergency department components of care, the establishment of net-

Haciendo foco en los sistemas de mejoras en la atención oportuna para el paciente con STEMI, vamos a completar el ciclo de la investigación iniciada por Reimer y Jennings hace 30 años

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