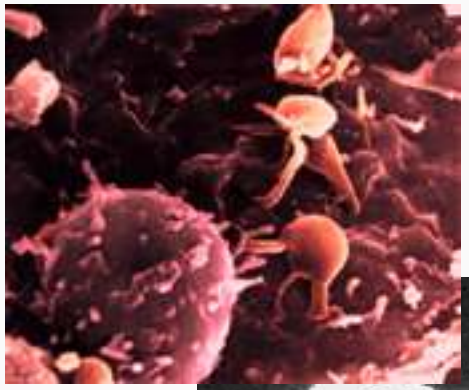
Hay Lugar Aun Para los Inhibidores GPIIbIIIa?

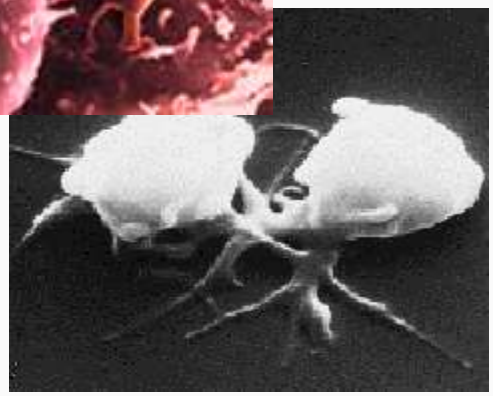
XXIII Jornada SOLACI/Sociedad Puertoriqueña Cardiologia Intervencional
San Juan Puerto Rico

Dr. Pedro Ureña Velásquez FACC FSCAI FACCP
Director Medicina Cardiovascular Asociada

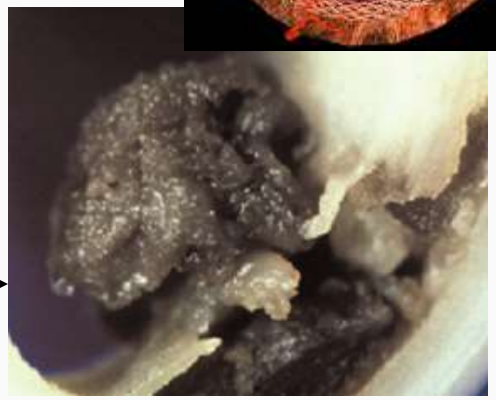
Rol Plaquetario en Trombosis y Estabilidad de la Placa

Moduladores Inflamatorios Producidos por la Plaqueta Activada



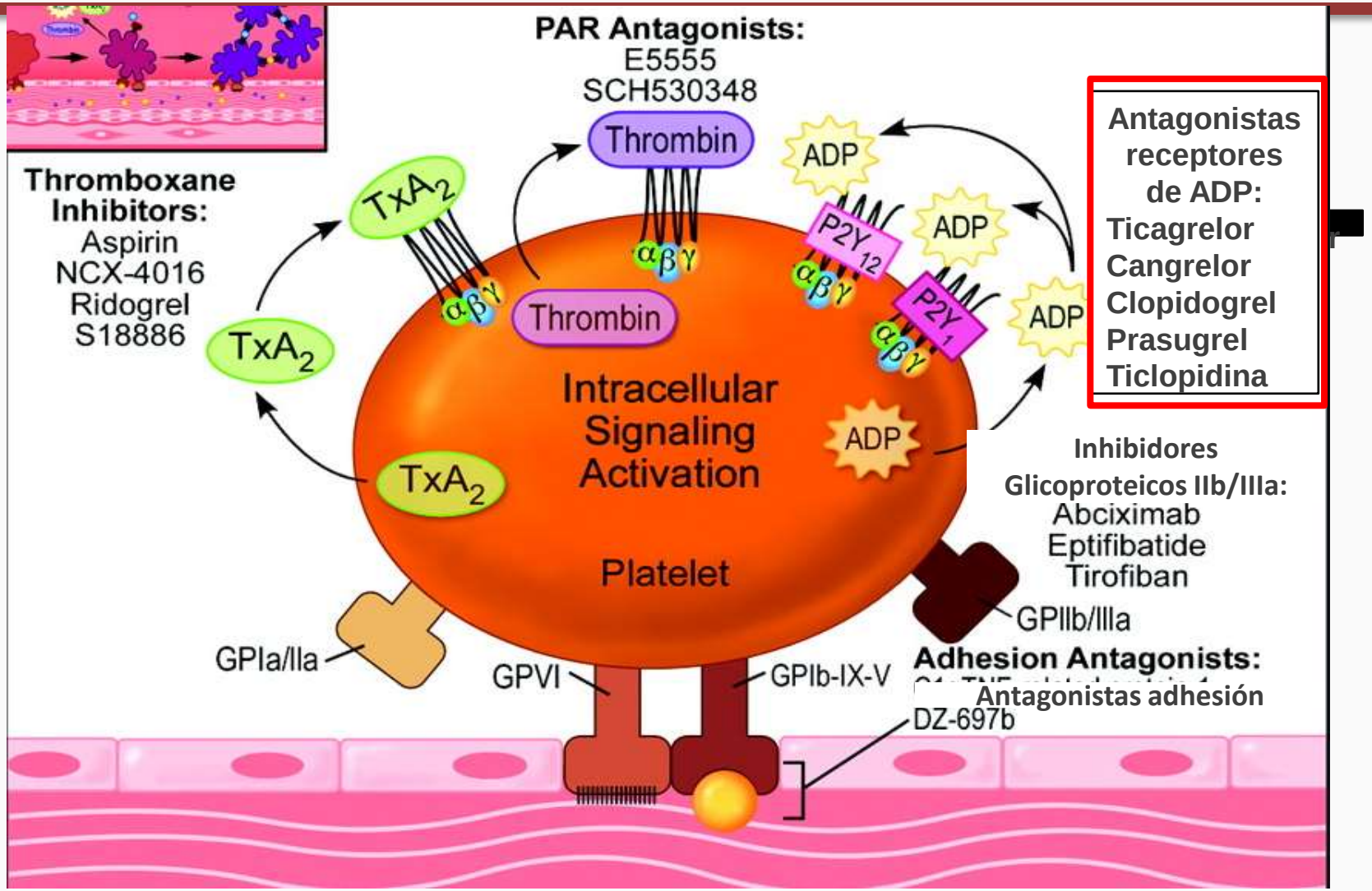


- Platelet-derived growth factor
- Platelet factor 4
- CD 154 (CD40L)
- RANTES*
- Thrombospondin
- Transforming growth factor-β
- Nitric oxide



*Regulated on activation, normal T-cell-expressed and -secreted.
Libby P et al. Circulation. 2001:103:1718-1720.

Sitio de acción de fármacos antiplaquetarios



Meadows TA, Bhatt DL. *Circ Res*. 2007;100:1261-1275.

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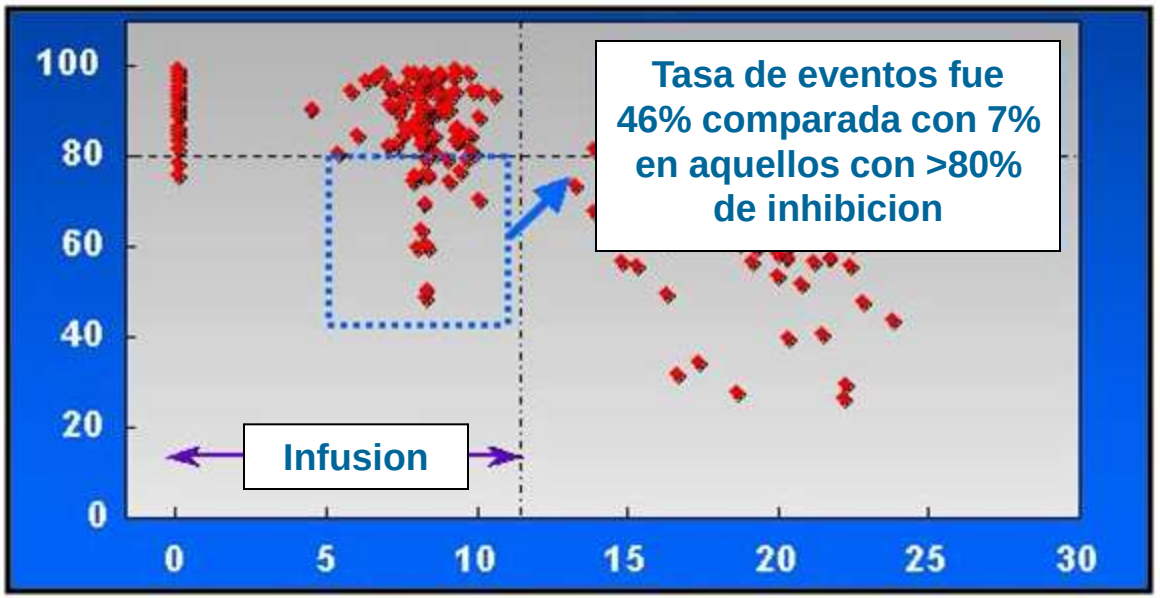
Table 1. Platelet-Membrane Glycoprotein Receptors Involved in the Adhesion and Aggregation of Platelets.

Receptor	Ligand	Receptor-Mediated Action	Amino Acid Sequence Recognized
Integrin			
$\alpha_2\beta_1$ (glycoprotein Ia/IIa)	Collagen	Adhesion	DGEA*
$\alpha_5\beta_1$ (glycoprotein Ic/IIa)	Fibronectin	Adhesion	RGD
$\alpha_6\beta_1$	Laminin	Adhesion	Not confined to a short sequence
$\alpha_{IIb}\beta_3$ (glycoprotein IIb/IIIa)	Fibrinogen	Aggregation	KQAGDV or RGD
	Fibronectin		RGD*
	von Willebrand factor		RGD
	Vitronectin		RGD
$\alpha_v\beta_3$	Vitronectin	Adhesion	RGD
	Fibrinogen		RGD
	Fibronectin		RGD
	von Willebrand factor		RGD
Nonintegrin			
Glycoprotein Ib	von Willebrand factor	Adhesion	Not confined to a short sequence
Glycoprotein IV	Thrombospondin	Adhesion	CSVTCG
	Collagen		?

Nivel de inhibición plaquetaria con Antagonistas GPIIb/IIIa

Data preliminar con RPFA

Estudio PARADISE



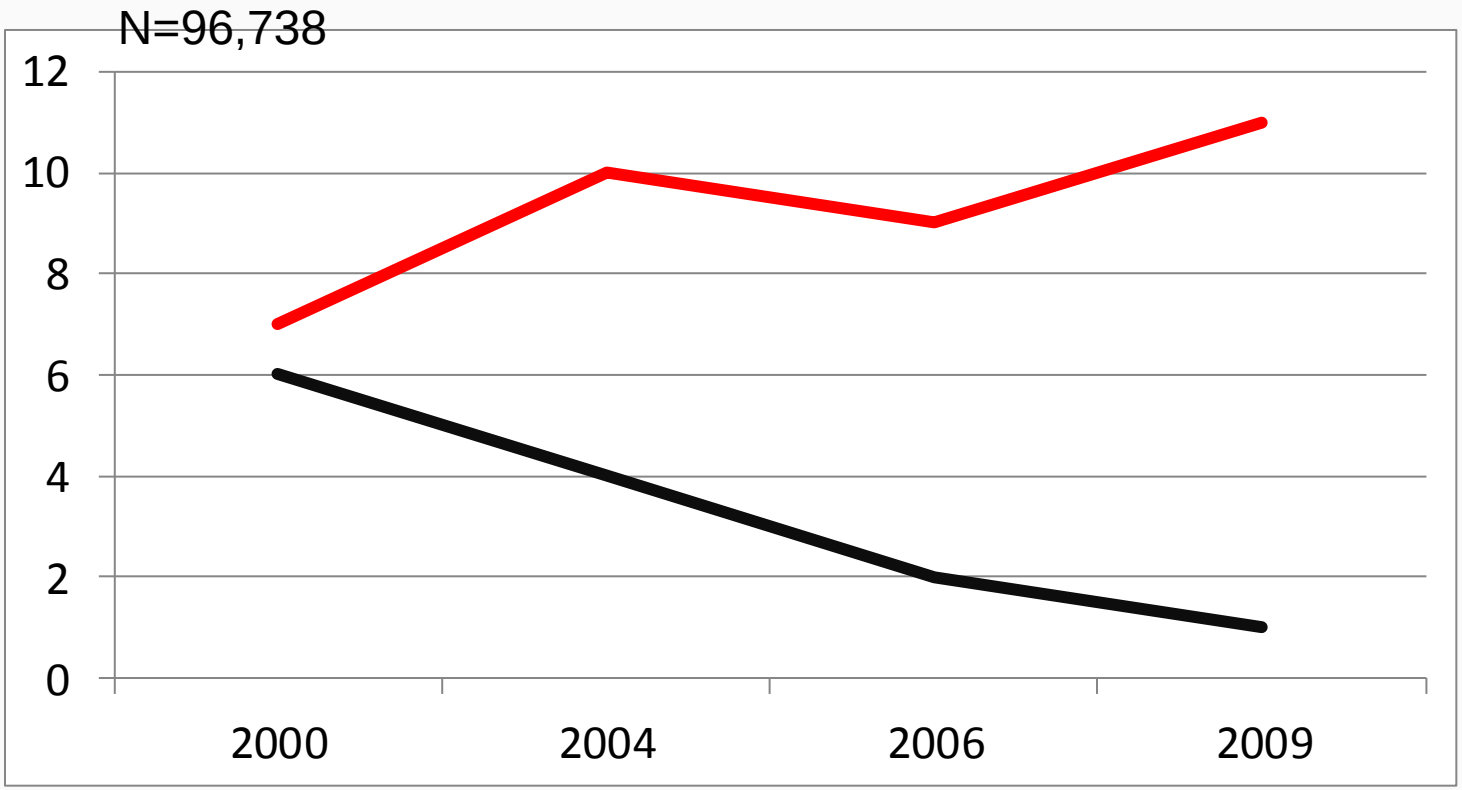
% de Inhibición plaquetaria

Horas luego del bolo de Abciximab

ACA
Medicina Cardiovascular Asociada

Steinhubi SR. Circulation 1999; 100:1977-1982

Use of GP2b3a inh vs Thrombin Inhibitors in Primary PCI 2000-2009



N Engl J Med 369:10 2013

Desarrollo de mejores antiagregantes?, Sangrado asociado?, Bivaluridina?

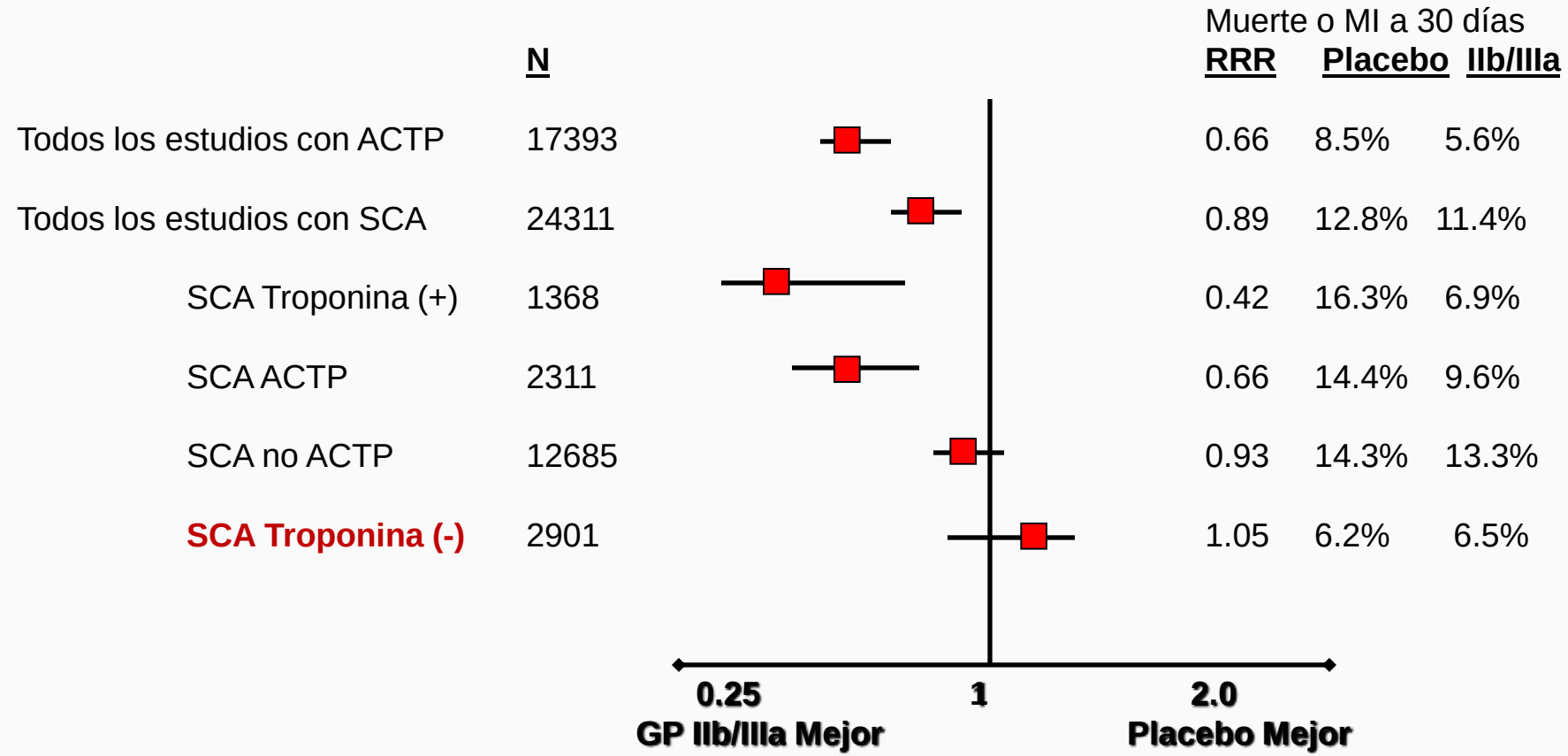
Elective PCI-

ACS NSTE-

ACS STE-

Revisión de Estudios con Inhibidores de la GP IIB/IIIA

El abordaje con antitrombóticos a corto plazo no reducirá los eventos en pacientes con Síndrome Coronario Agudo tratados médicamente



Chew et al JACC 2000 36;2028

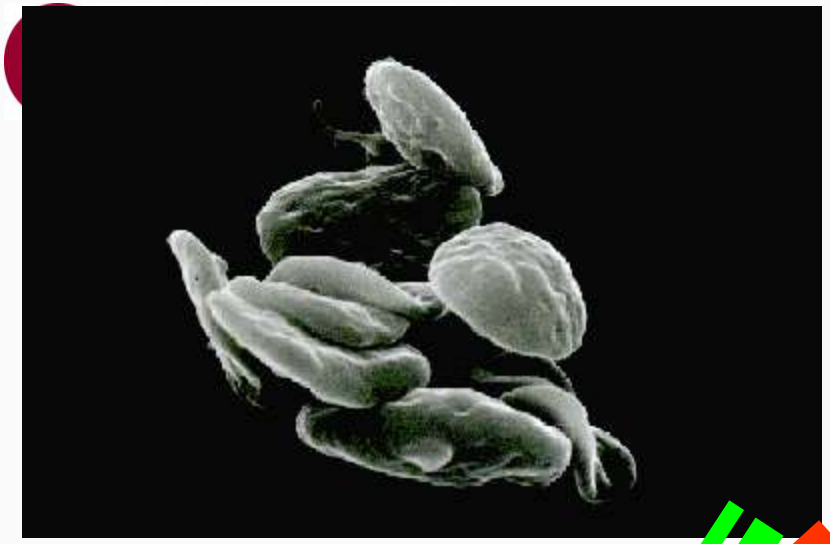
ISAR-REACT 2: Main efficacy Resultados a 30 días

End point	Abciximab (%)	Placebo (%)	RR (95% CI)
Death/MI/ urgent TVR*	8.9	11.9	0.75 (0.58–0.97)
Death	1.1	1.6	0.69 (0.32–1.47)
MI	8.1	10.5	0.77 (0.59–1.02)
Urgent TVR	1.0	1.2	0.83 (0.36–1.92)

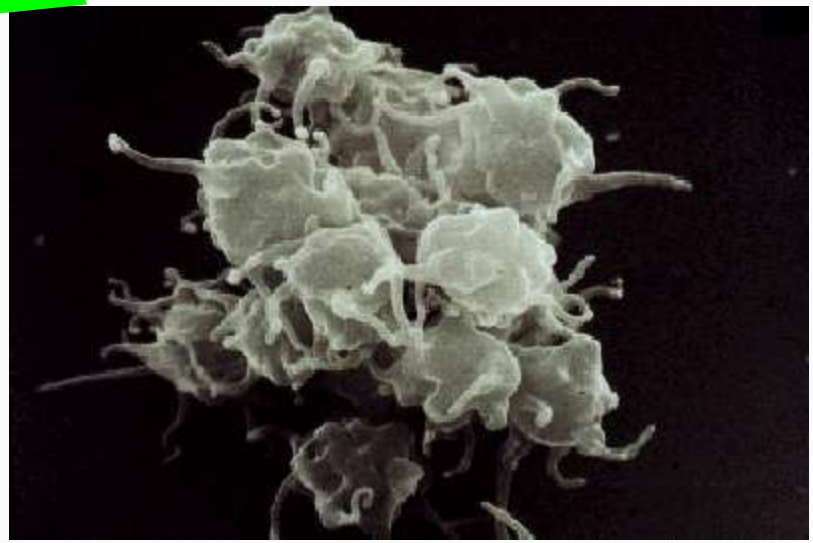
***Primary end point**

Placebo = Clopidogrel 600mg 2 hrs pre intervención

Kastrati A et al. JAMA 2006



Antagonistas Glycoprotein a IIb/IIIa NSTE



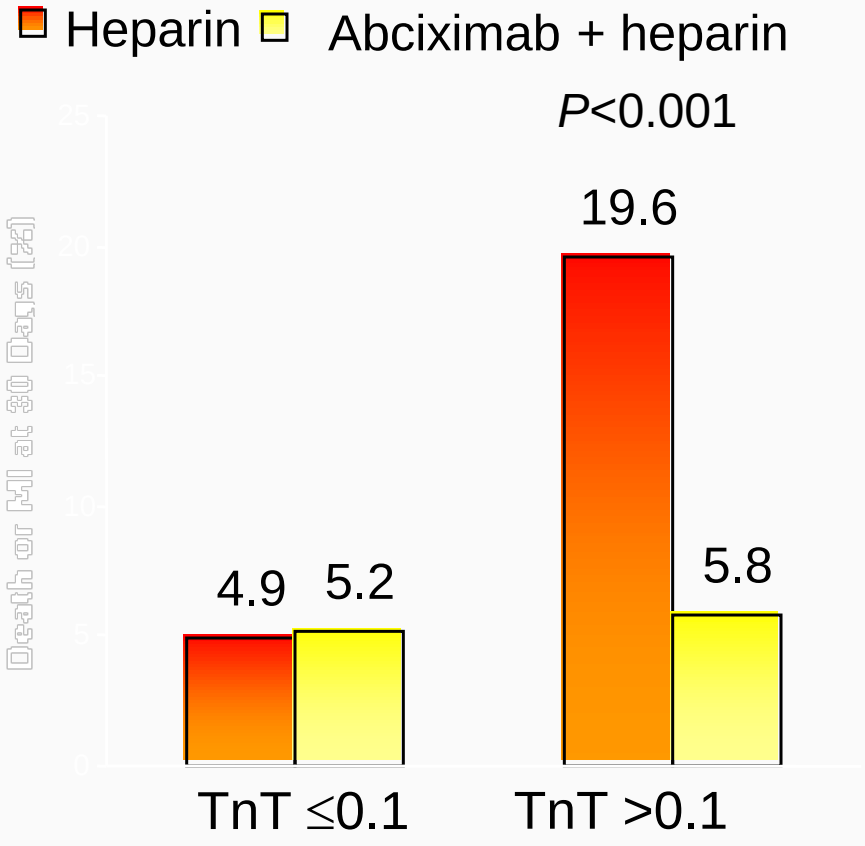
Abciximab

Eptifibatide

Tirofiban

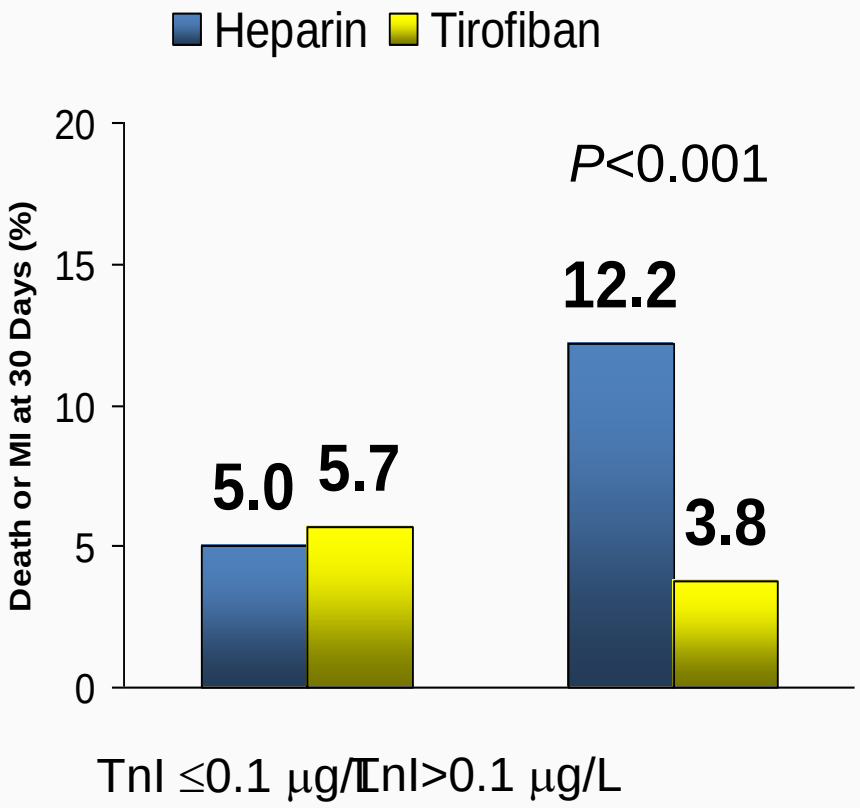
Benefit of IIb/IIIa inhibitors in UA/NSTEMI by Troponin

CAPTURE



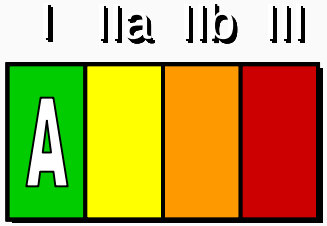
Hamm CW, *NEJM* 1999;340:1623-9.

PRISM

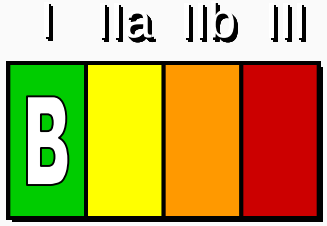


Heeschen, *Lancet*. 1999;354:1757-62

Initial Invasive Strategy: Antiplatelet Therapy



For UA/NSTEMI patients in whom an initial invasive strategy is selected, antiplatelet therapy in addition to ASA should be initiated before diagnostic angiography (upstream) with either clopidogrel (loading dose followed by daily maintenance dose)* or an IV GP IIb/IIIa inhibitor. (Box B2)



Abciximab as the choice for upstream GP IIb/IIIa therapy is indicated only if there is no appreciable delay to angiography and PCI is likely to be performed; otherwise, IV eptifibatide or tirofiban is the preferred choice of GP IIb/IIIa inhibitor.†

Some uncertainty exists about optimum dosing of clopidogrel. Randomized trials establishing its efficacy and providing data on bleeding risks used a loading dose of 300 mg orally followed by a daily oral maintenance dose of 75 mg. Higher oral loading doses such as 600 or 900 mg of clopidogrel more rapidly inhibit platelet aggregation and achieve a higher absolute level of inhibition of platelet aggregation, but the additive clinical efficacy and the safety of higher oral loading doses have not been rigorously established; †Factors favoring administration of both clopidogrel and a GP IIb/IIIa inhibitor include: delay to angiography, high-risk features, and early ischemic discomfort.

Recomendaciones **Guías ESC - SCA no ST**	Clase	Nivel
Se recomienda clopidogrel (dosis de carga de 300 mg, mantenimiento 75 mg/día) **para pacientes que no puedan recibir ticagrelor o prasugrel.**	I	A
Se recomienda una dosis de carga de 600 mg de clopidogrel (o una dosis suplementaria de 300 mg en la angioplastía, luego de una dosis de carga inicial de 300 mg) para pacientes en plan de estrategia invasiva, **cuando ticagrelor o prasugrel no sean una opción.**		
Debe ser considerada una dosis de mantenimiento mayor de clopidogrel 150 mg por los primeros 7 días en pacientes manejados con angioplastía y sin riesgo incrementado de sangrado.	IIa	B
No se recomienda como rutina incrementar la dosis de mantenimiento de clopidogrel basado en pruebas de función plaquetaria, pero puede ser considerado en casos seleccionados.	IIb	B
Pueden ser consideradas la genotipificación y/o las pruebas de función plaquetaria en casos seleccionados, cuando se utilize clopidogrel.	IIb	B
Se debe considerar, en pacientes pretratados con inhibidores $P2Y_{12}$ que necesiten someterse a una cirugía mayor no urgente (incluyendo CABG), posponer la cirugía al menos 5 días luego de suspender ticagrelor o clopidogrel, y 7 días prasugrel, si es clinicamente posible y excepto que el paciente esté en alto riesgo de eventos isquémicos.	IIa	C
Se debe considerar (re) iniciar ticagrelor o clopidogrel luego de la cirugía CABG tan pronto como se considere seguro.	IIa	B
No se recomienda la combinación de aspirina con un AINE (inhibidores selectivos COX-2 y AINEs no selectivos).	III	C

Guías ESC - SCA no ST - European Heart Journal 2011; 32: 2999-3054.

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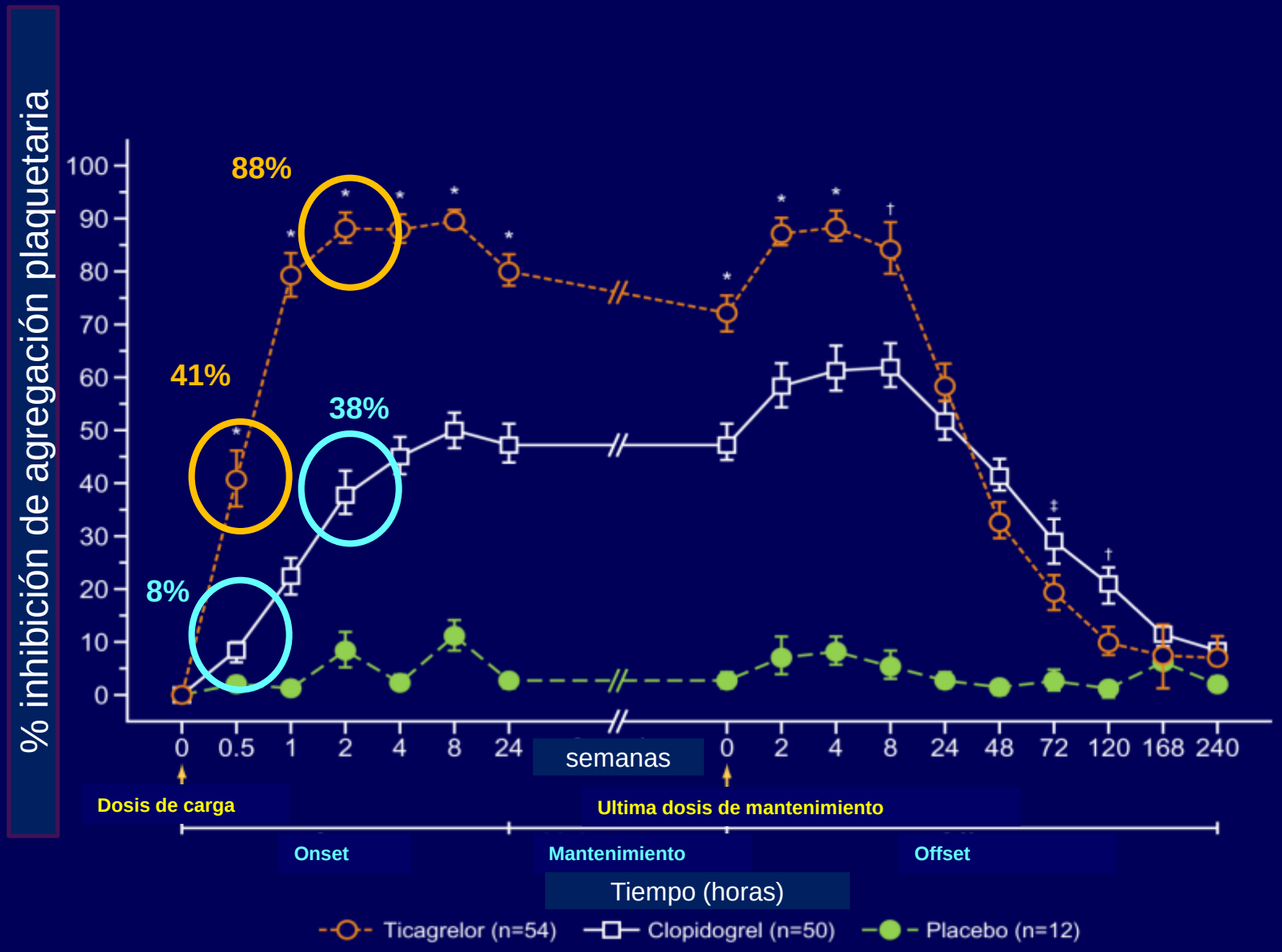
Inhibidores P2Y12

	Clopidogrel	Prasugrel	Ticagrelor	Cangrelor	Elinogrel
Mechanism of Action	**Irreversible**	**Irreversible**	**reversible**	**Competitive & reversible**	**Competitive & reversible**
Dosing route	oral	oral	oral	IV infusion	IV bolus and oral
Onset of action	3-8 h (prodrug)	1-4 h (prodrug)	min – hours (oral, direct)	seconds (direct)	seconds (direct)
Inhibition	irreversible	irreversible	reversible	reversible	reversible
Maximum Inhibition	~40%	Full	Full	Full	Full
Variability	+++	++?	+?	+?	+?
Selectivity	+++	+++	+*	+++?	+++

*off-target (adenosine receptor) adverse events: hypotension, dyspnea, heart block, etc

ONSET/OFFSET: Inhibición de la agregación plaquetaria



Gurbel PA, et al. *Circulation.* 2009;120:2577-2585.

Recommendations for GPIIb/IIIa receptor inhibitors

Recommendations	Class	Level
The choice of combination of oral antiplatelet agents, a GPIIb/IIIa receptor inhibitor, and anticoagulants should be made in relation to the risk of ischaemic and bleeding events.	I	C
Among patients who are already treated with DAPT, the addition of a GPIIb/IIIa receptor inhibitor for high-risk PCI (elevated troponin, visible thrombus) is recommended if the risk of bleeding is low.	I	B
Eptifibatide or tirofiban added to aspirin should be considered prior to angiography in high-risk patients not preloaded with $P2Y_{12}$ inhibitors.	IIa	C
In high-risk patients eptifibatide or tirofiban may be considered prior to early angiography in addition to DAPT, if there is ongoing ischaemia and the risk of bleeding is low.	IIb	C
GPIIb/IIIa receptor inhibitors are not recommended routinely before angiography in an invasive treatment strategy.	III	A
GPIIb/IIIa receptor inhibitors are not recommended for patients on DAPT who are treated conservatively.	III	A

European Heart Journal (2011) 32:2999–3054
doi:10.1093/eurheartj/ehr236

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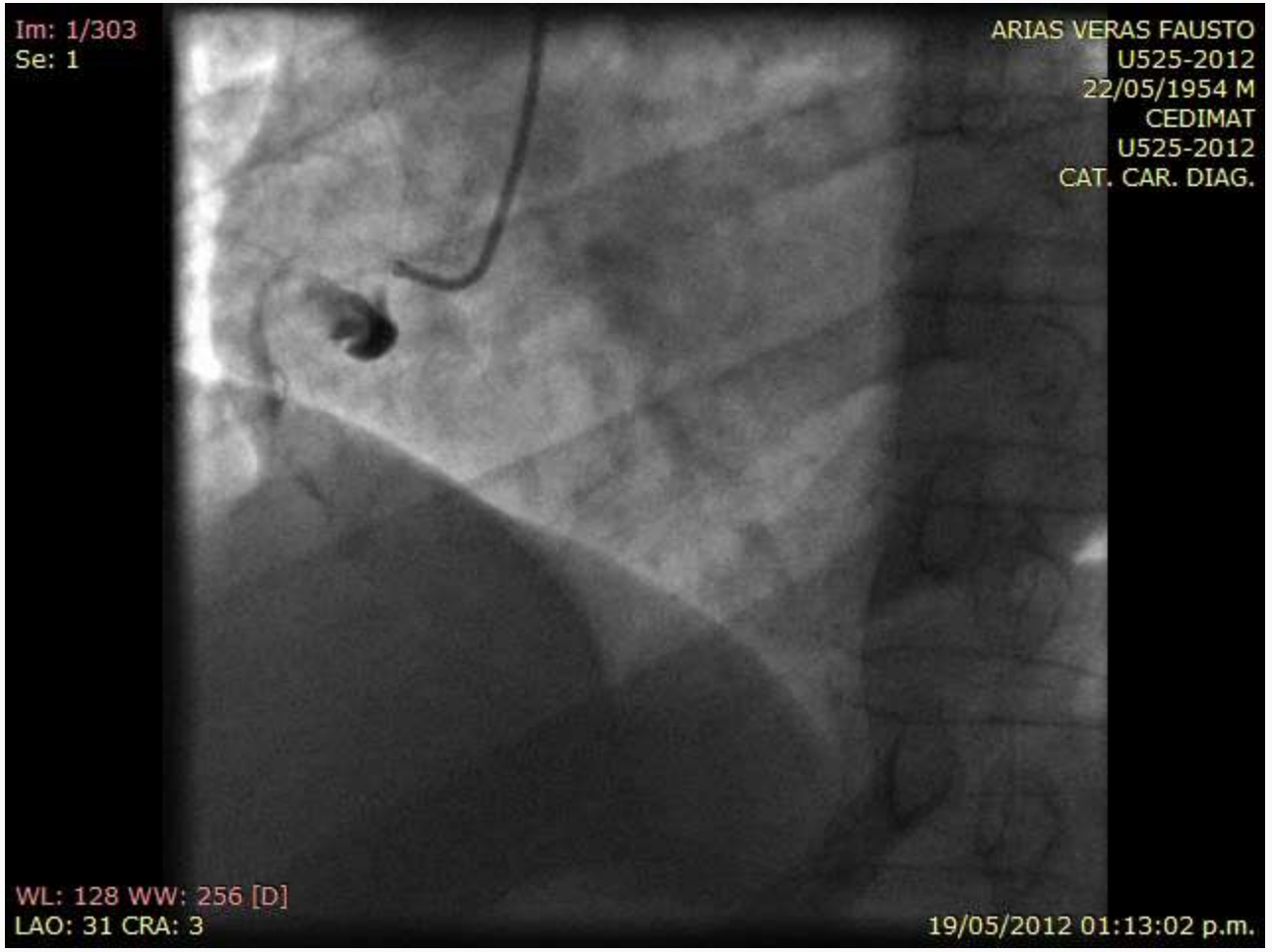
Tricagelor 180mg, Enoxaparin 100mg, ASA 325mg



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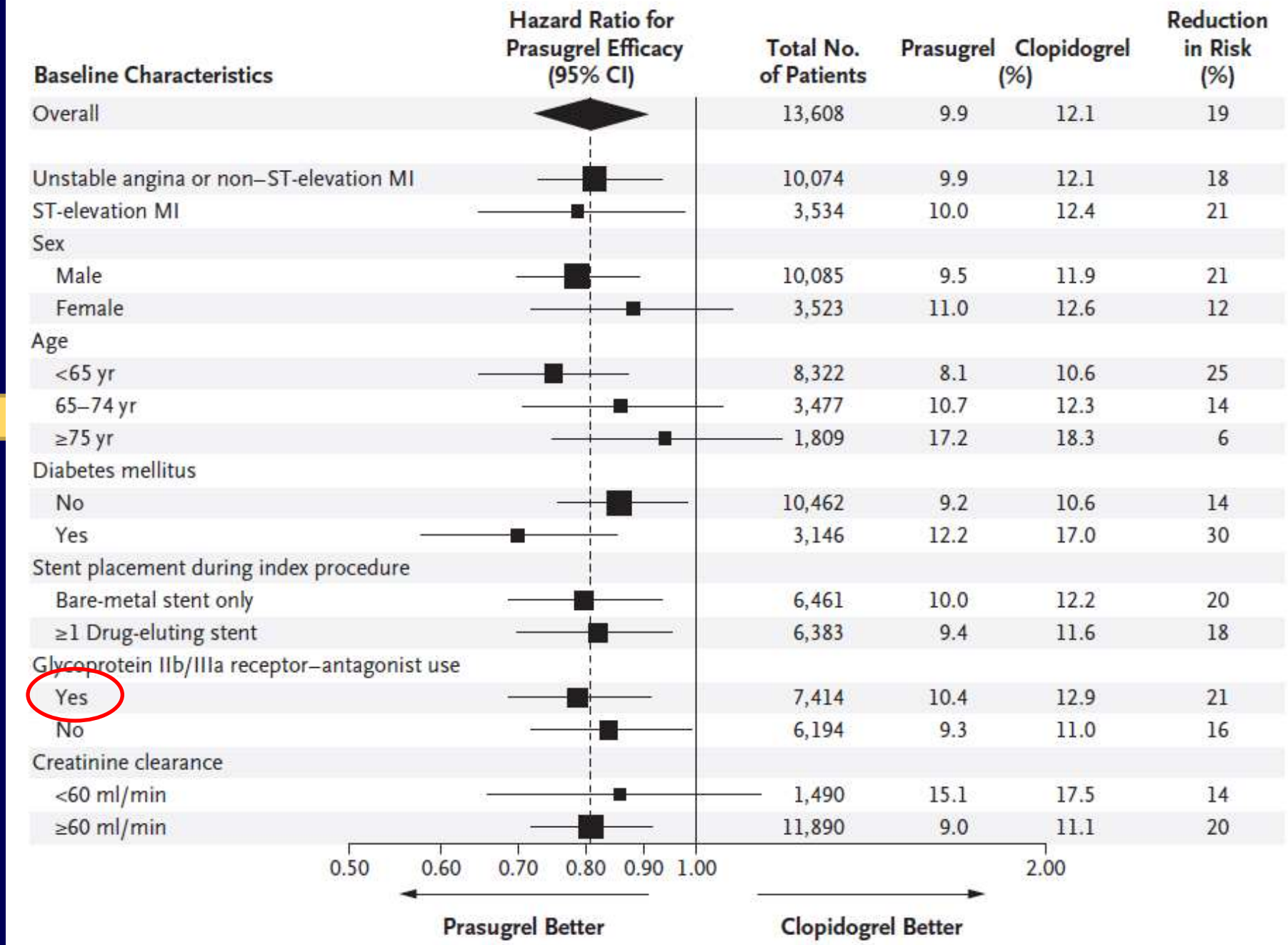

Wallentin L, et al. N Engl J Med. 2009;361:1045-1057 y tablas suplementarias

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Sangrado vs Trombo

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ORIGINAL ARTICLE

Early versus Delayed, Provisional Eptifibatide in Acute Coronary Syndromes

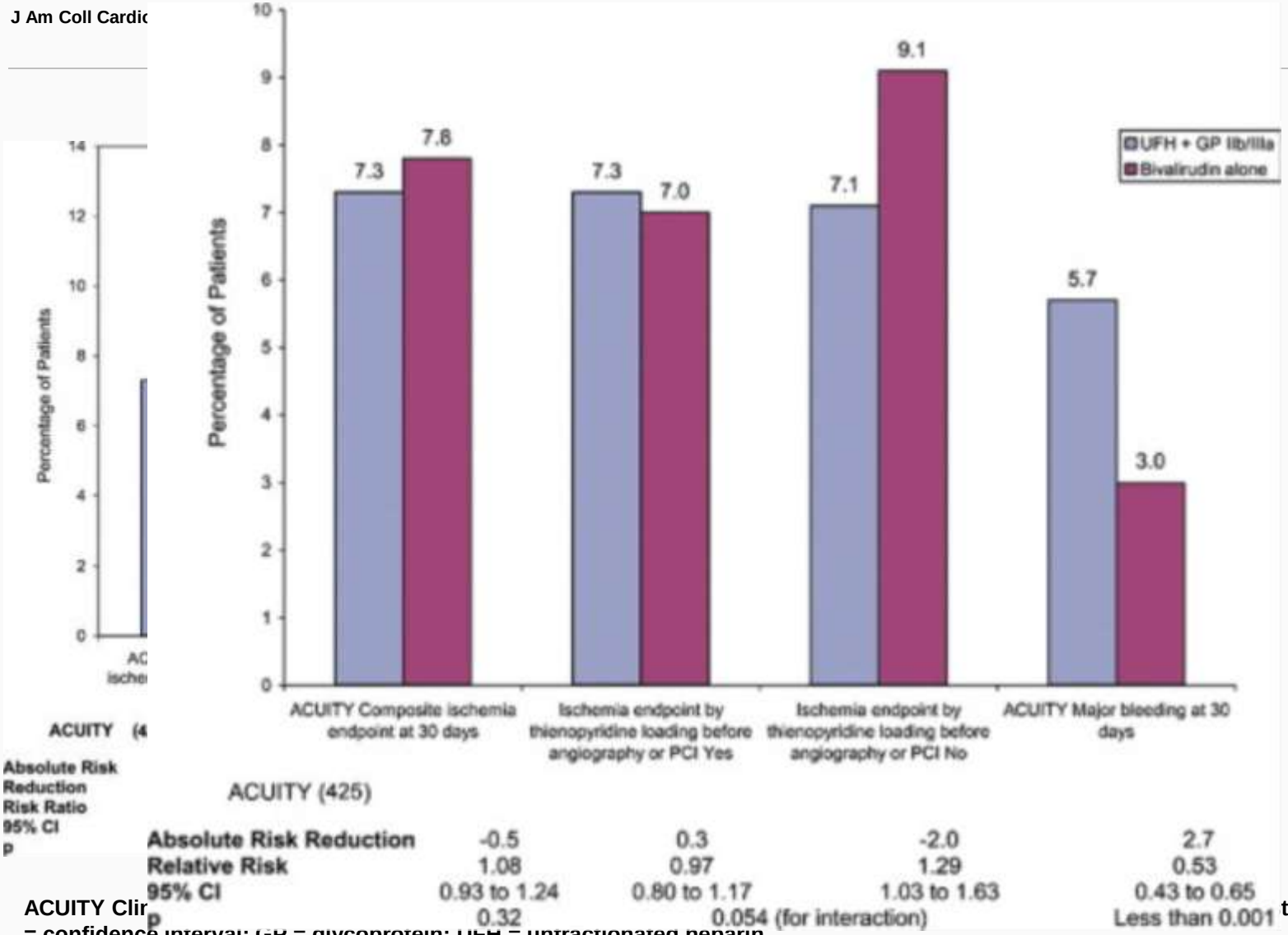
Robert P. Giugliano, M.D., S.M., Jennifer A. White, M.S., Christoph Bode, M.D., Paul W. Armstrong, M.D., Gilles Montalescot, M.D., Basil S. Lewis, M.D., Arnoud van 't Hof, M.D., Lisa G. Berdan, P.A., M.H.S., Kerry L. Lee, Ph.D., John T. Strony, M.D., Steven Hildemann, M.D., Enrico Veltri, M.D., Frans Van de Werf, M.D., Ph.D., Eugene Braunwald, M.D., Robert A. Harrington, M.D., Robert M. Califf, M.D., and L. Kristin Newby, M.D., M.H.S., for the EARLY ACS Investigators*

CONCLUSIONS

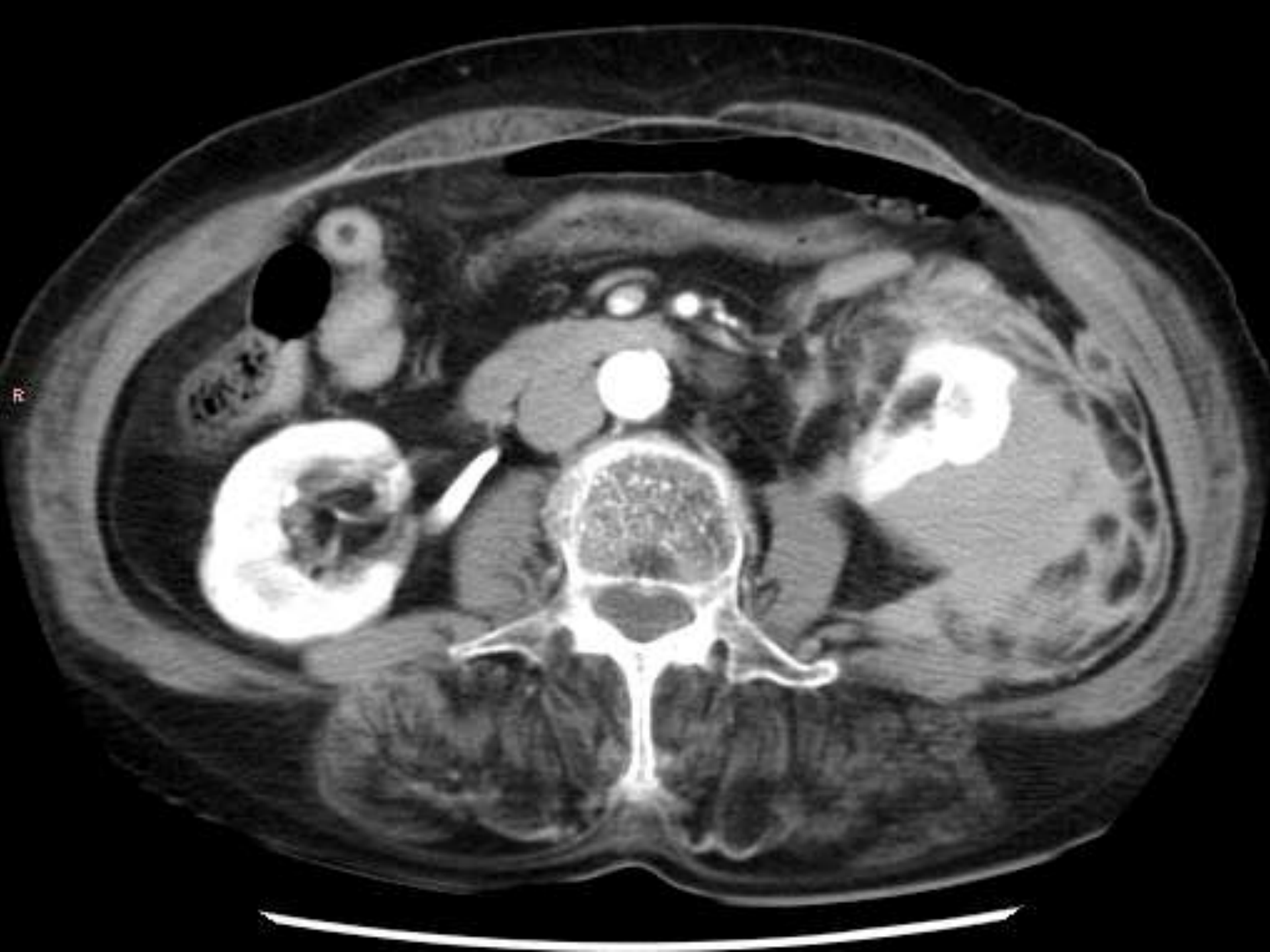
In patients who had acute coronary syndromes without ST-segment elevation, the use of eptifibatide 12 hours or more before angiography was not superior to the provisional use of eptifibatide after angiography. The early use of eptifibatide was associated with an increased risk of non–life-threatening bleeding and need for transfusion. (ClinicalTrials.gov number, NCT00089895.)

From: 2011 ACCF/AHA Focused Update Incorporated Into the ACC/AHA 2007 Guidelines for the Management of Patients With Unstable Angina/Non–ST-Elevation Myocardial Infarction: A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines

J Am Coll Cardio



ACUITY Clin trategY; CI = confidence interval; GP = glycoprotein; UFH = unfractionated heparin.



Hazard of Bleeding and Transfusions

(n=17,034)

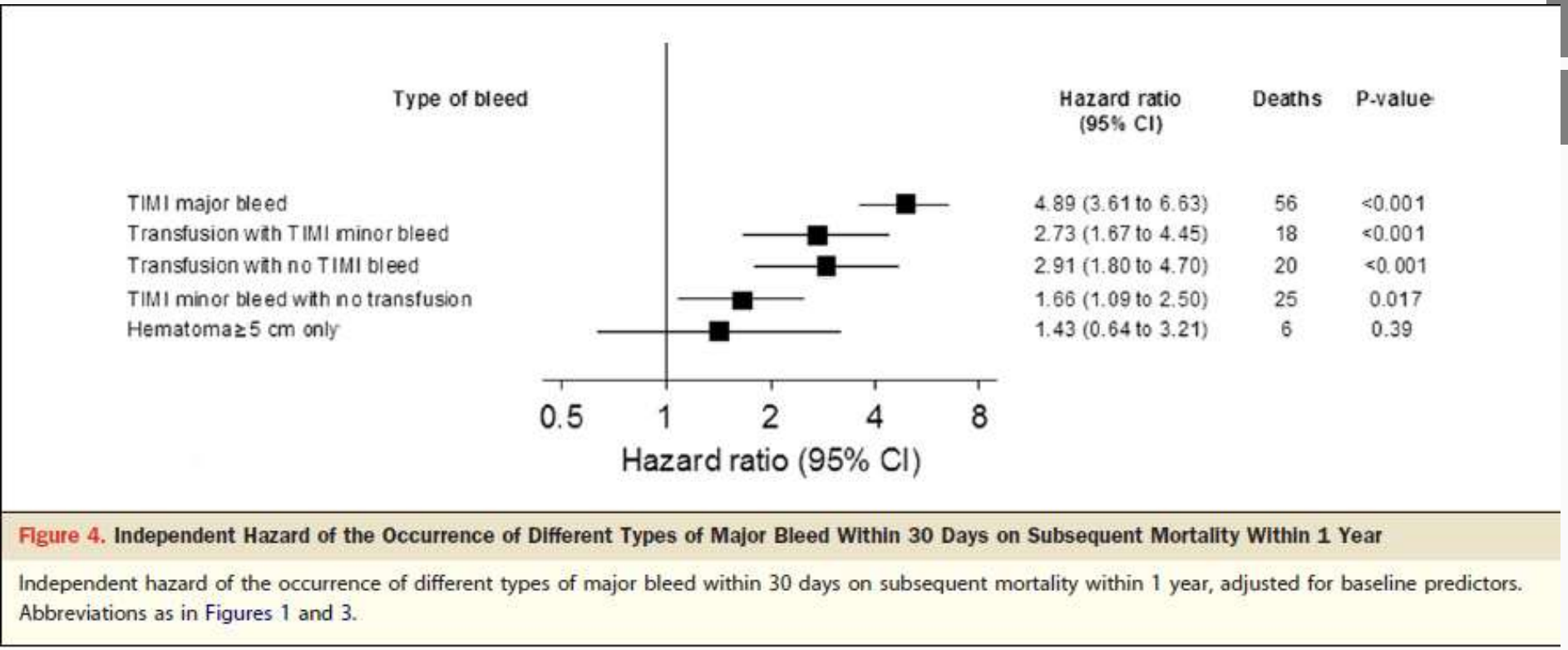
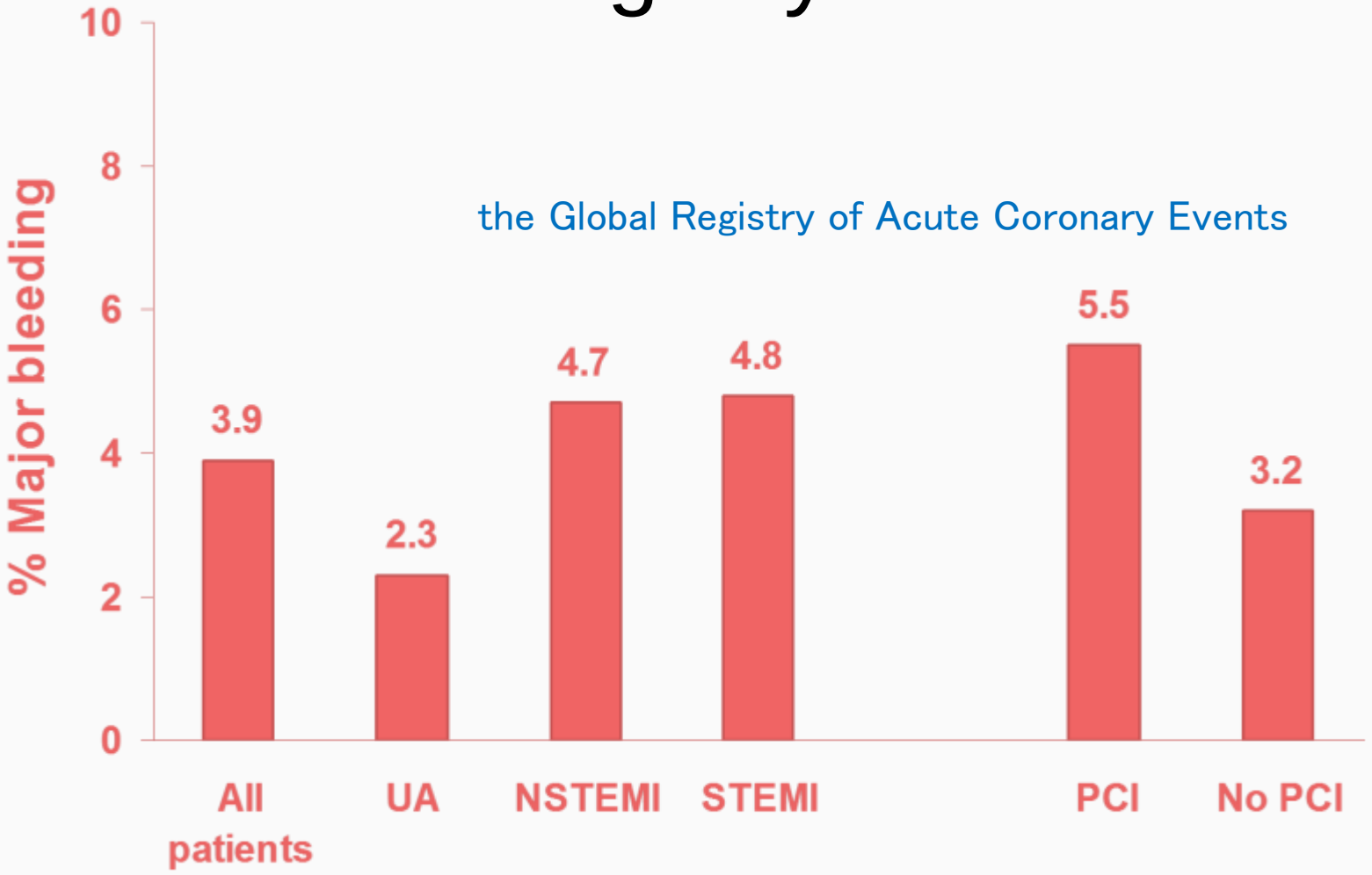


Figure 4. Independent Hazard of the Occurrence of Different Types of Major Bleed Within 30 Days on Subsequent Mortality Within 1 Year

Independent hazard of the occurrence of different types of major bleed within 30 days on subsequent mortality within 1 year, adjusted for baseline predictors. Abbreviations as in Figures 1 and 3.

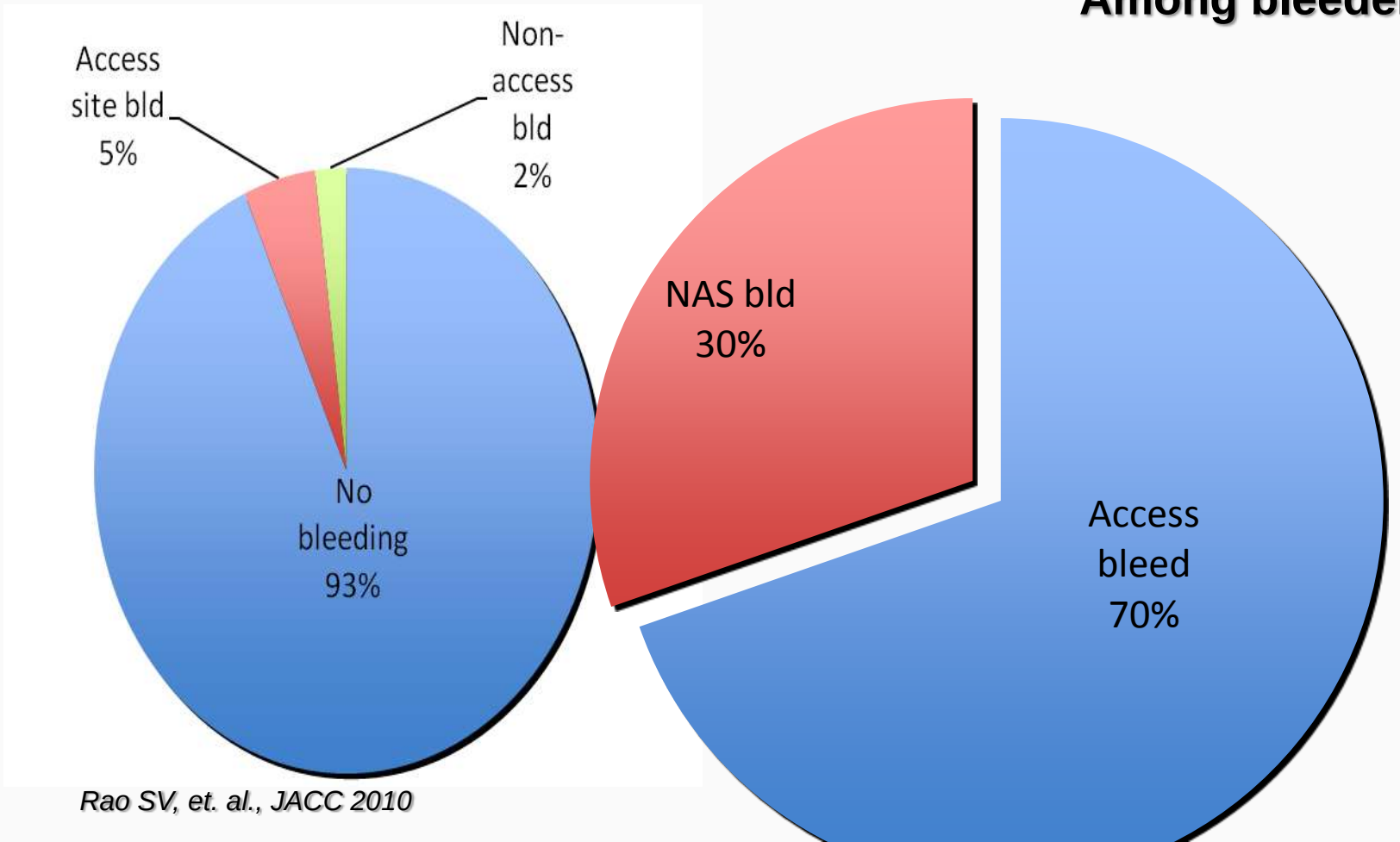
Mehran Et, Al, JACC *Interv*. 4:2011

Major bleeding in ACS GRACE registry



Moscucci, M. et al. Eur Heart J 2003 24:1815–1823

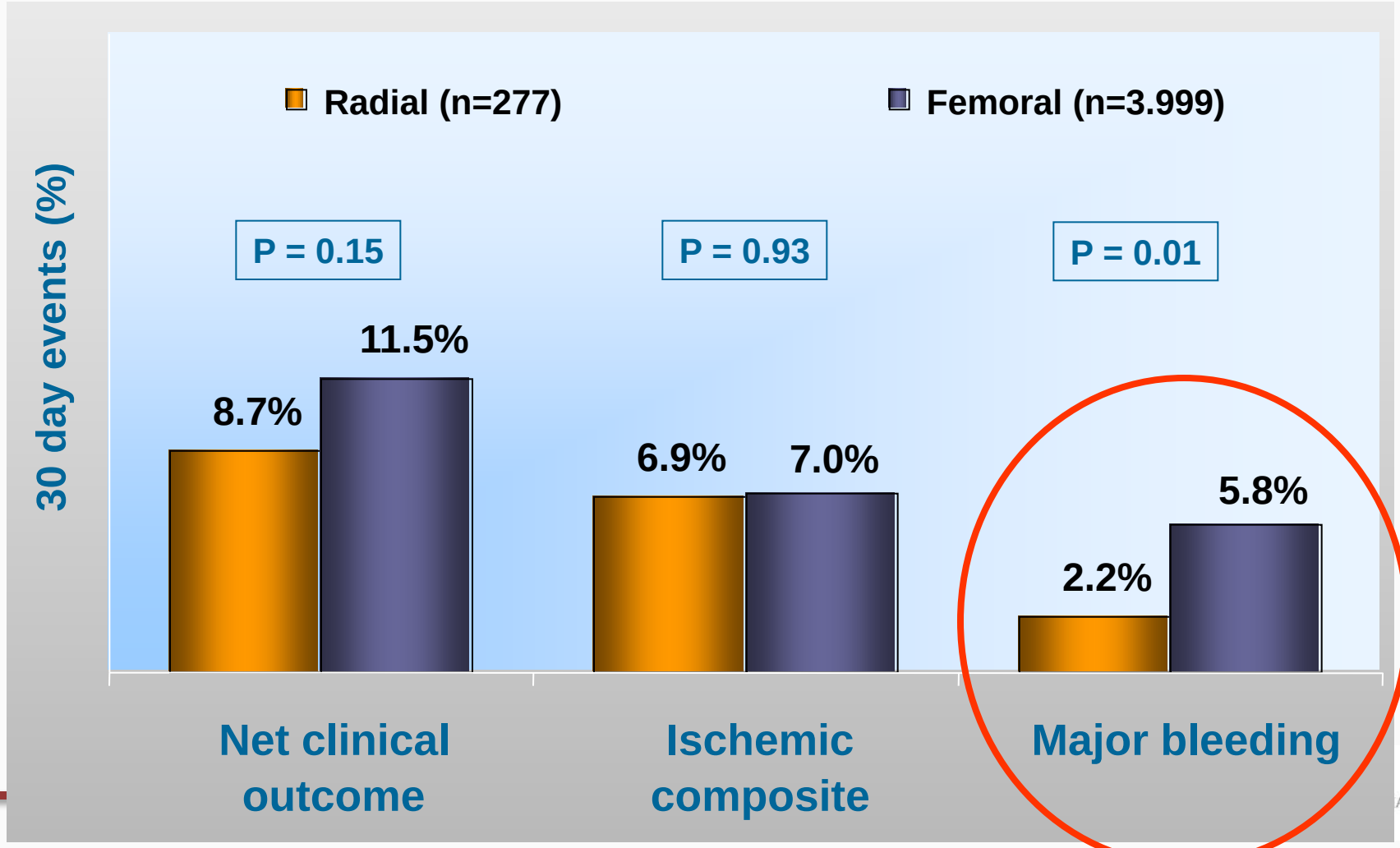
Bleeding in PCI Trials: Frequency and Site*



Rao SV, et. al., JACC 2010

Primary Endpoint Measures

Heparins + GPI



PCI with IIb/IIIa inhibitors for ACS

	TFI	TRI	p
N	130	531	
Pr Success	93.1%	91.0%	>0.2
Bleeding	29.2%	8.7%	<0.0001
Transfusion	7.7%	0.8%	<0.0001
Pr Death	1.5%	0.4%	>0.2
1-yr Death	10.0%	4.7%	0.02
1-yr MACE	20.8%	14.1%	0.06

Marco De Carlo et al. Cathet Cardiovasc Interv 2009; 74: 408-415.

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CRUSADE score of in-Hospital major bleeding

Predictor	Score
Baseline haematocrit, %	
< 31	9
31-33.9	7
34-36.9	3
37-39.9	2
≥ 40	0
Creatinine clearance, mL/min	
≤ 15	39
> 15-30	35
> 30-60	28
> 60-90	17
> 90-120	7
> 120	0

Predictor	Score
Heart rate (b.p.m.)	
≤ 70	0
71-80	1
81-90	3
91-100	6
101-110	8
111-120	10
≥ 121	11
Male	0
Female	8
Sex	
Male	0
Female	8
Signs of CHF at presentation	
No	0
Yes	7

Predictor	Score
Prior vascular disease	
No	0
Yes	6
Diabetes mellitus	
No	0
Yes	6
Systolic blood pressure, mmHg	
≤ 90	10
91-100	8
101-120	5
121-180	1
181-200	3
≥ 201	5

www.crusadebleedingscore.org

European Heart Journal (2011) 32:2999–3054
doi:10.1093/eurheartj/ehr236

www.escardio.org/guidelines

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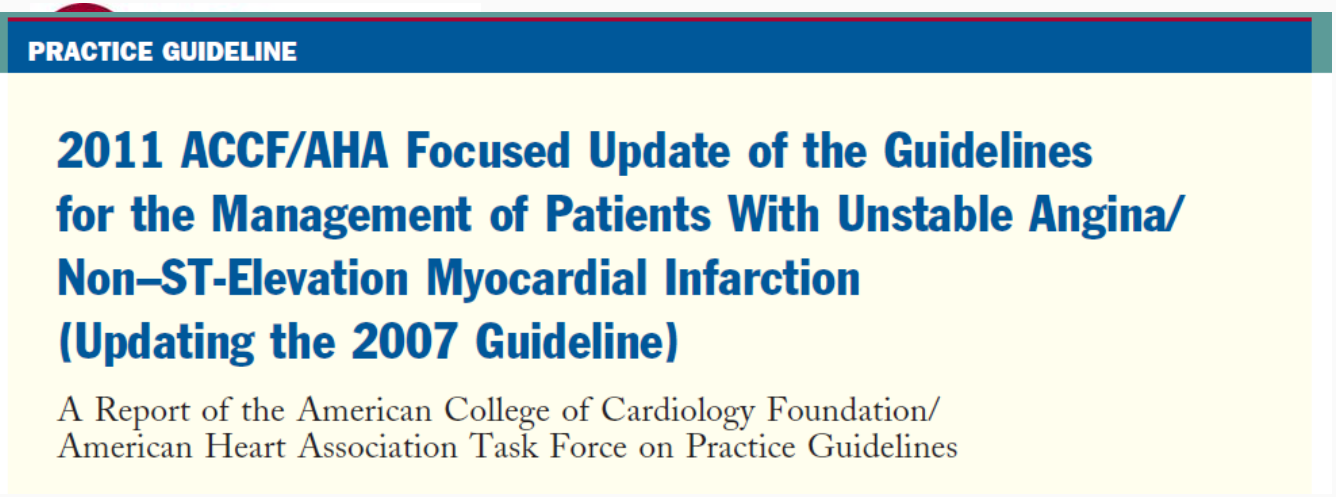

The use of a GP IIb/IIIa inhibitor should be undertaken when the risk-benefit ratio suggests a potential benefit for the patient. The use of these agents as part of triple-antiplatelet therapy may therefore not be supported when there is a concern for increased bleeding risk or in non–high-risk subsets such as those with a normal baseline troponin level, those without diabetes, and those ≥75 years of age, in whom the potential benefit may be significantly offset by the potential risk of bleeding.

La aparicion de nuevos y mas potentes antiplaquetarios orales , no contraindica el uso de inh Gp2b3a. Probablemente el uso “upstream” no este justificado, y su rol permanece ligado a carga trombotica alta en la angiografia.

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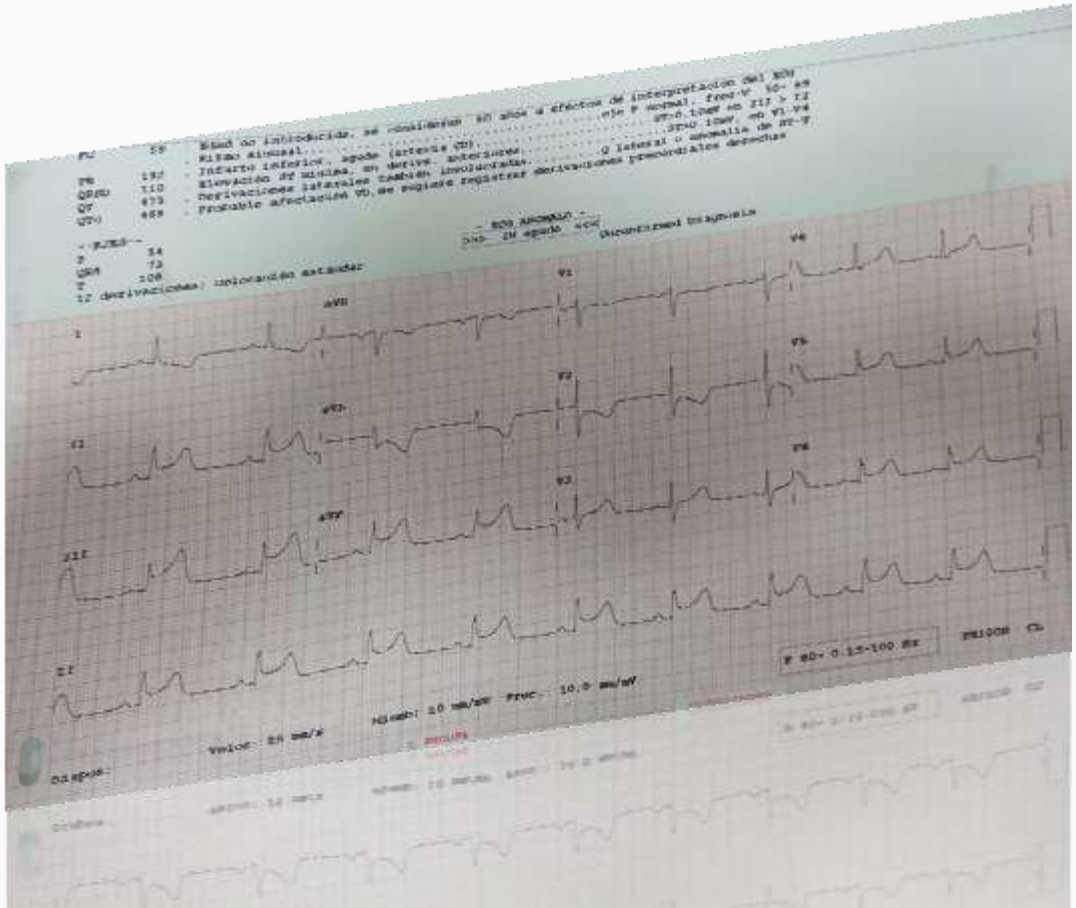
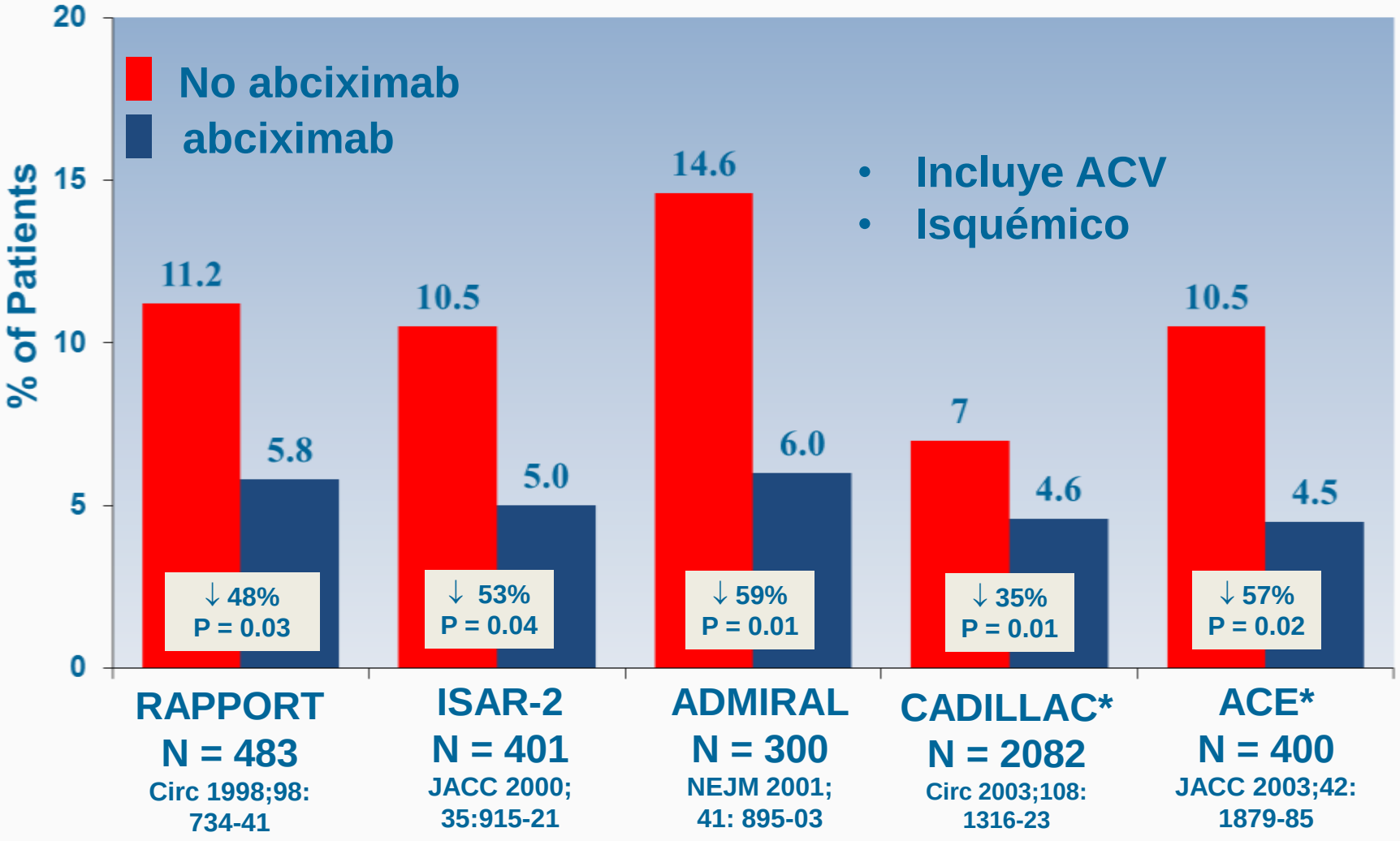
STE ACS



Punto Combinado a 30 Días

Muerte, IAM ó Revasc Urgente

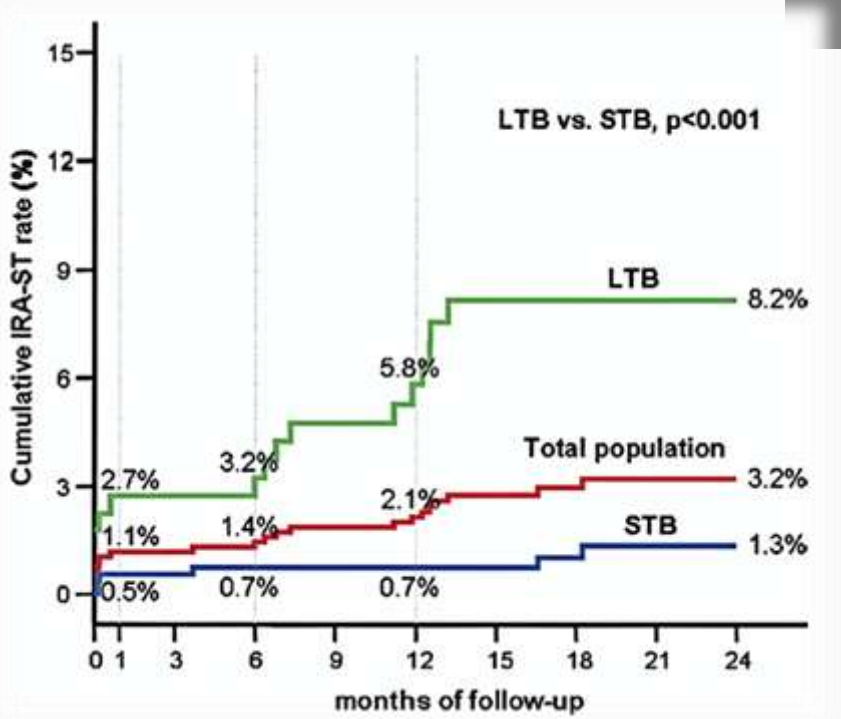


Impact of Thrombus Burden

798 STEMI Patients Treated with DES

	Small Thrombus	Large Thrombus
Final TIMI 3	94.9%	83.6%*
TMPG-3	53.2%	35.4%*
No-reflow	0.5%	4.0%*
Distal embolization	3.5%	17.3%*

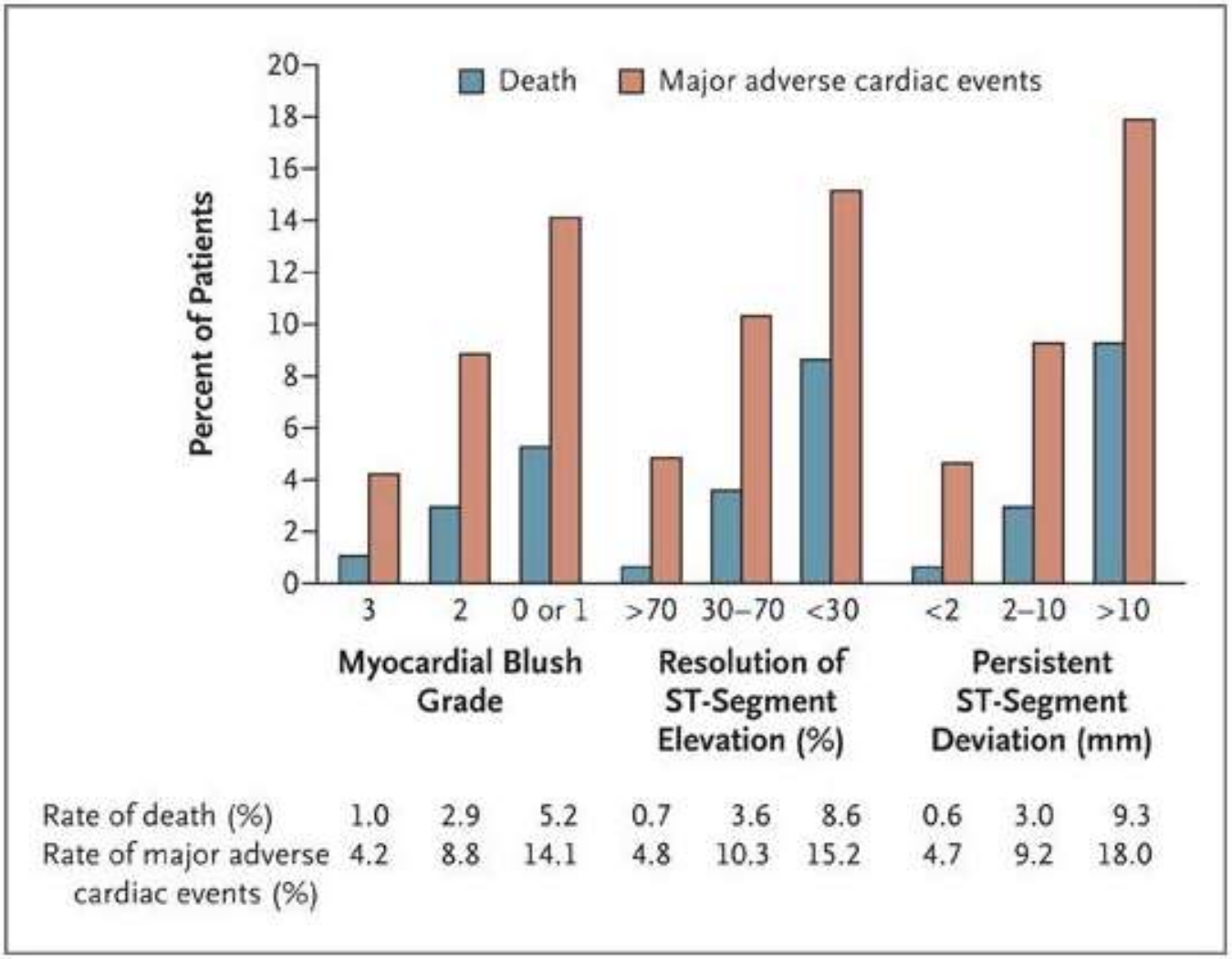
P<0.001



↑risk stent thrombosis

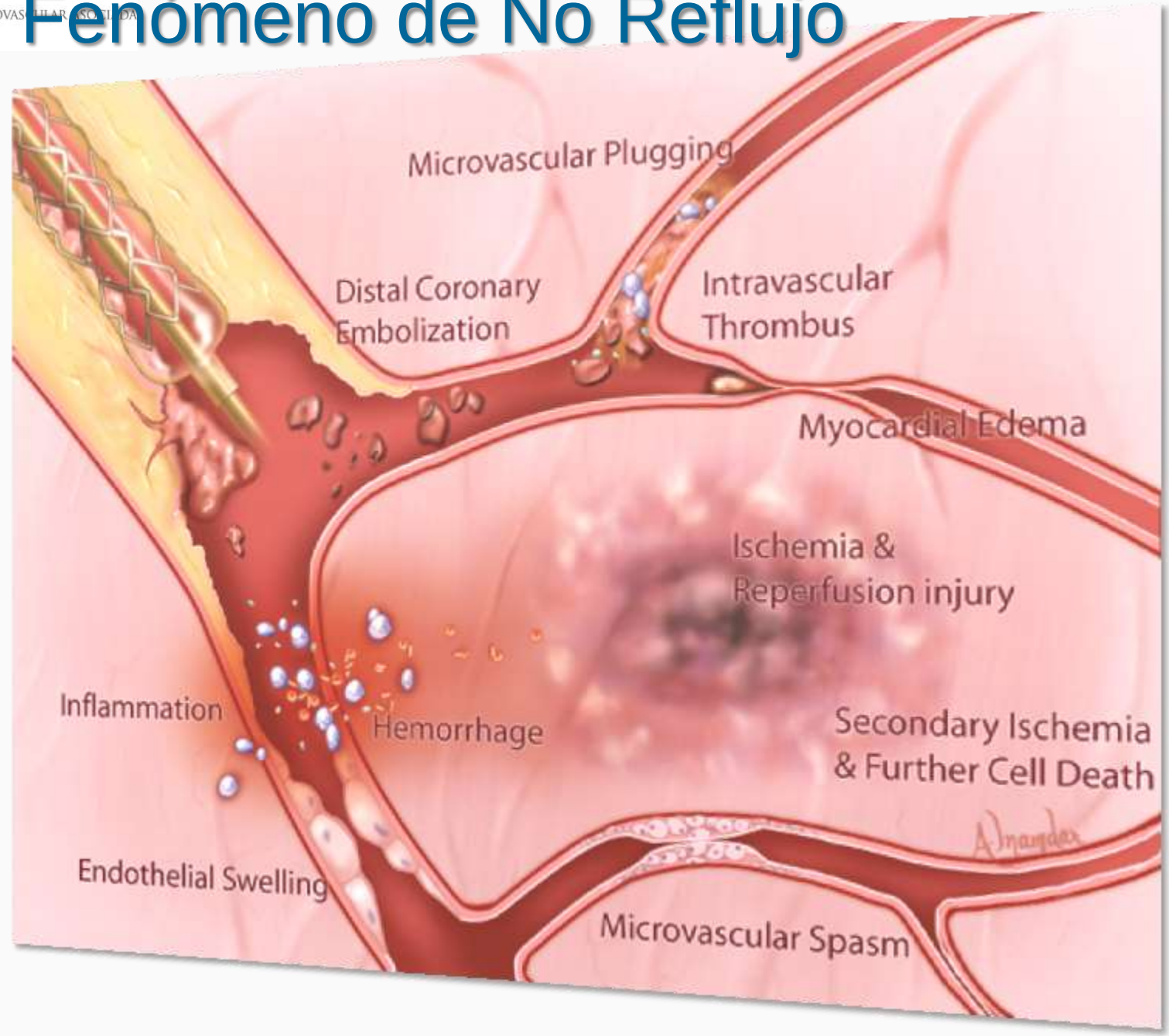
Sianos G. JACC 2007;50:573-83

Importancia de la Microcirculación post ACTP



Svilaas T et al. N Engl J Med 2008;358:557-567

Fenómeno de No Reflujo



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PRACTICE GUIDELINE

2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction

A Report of the American College of Cardiology Foundation/
American Heart Association Task Force on Practice Guidelines

CLASS IIb

1. It may be reasonable to administer intravenous GP IIb/IIIa receptor antagonist in the precatheterization laboratory setting (e.g., ambulance, ED) to patients with STEMI for whom primary PCI is intended (103,268,271–277). (*Level of Evidence: B*)
2. It may be reasonable to administer intracoronary abciximab to patients with STEMI undergoing primary PCI (223,278–284). (*Level of Evidence: B*)
3. Continuation of a $P2Y_{12}$ inhibitor beyond 1 year may be considered in patients undergoing DES placement. (*Level of Evidence: C*)

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Reasonable?

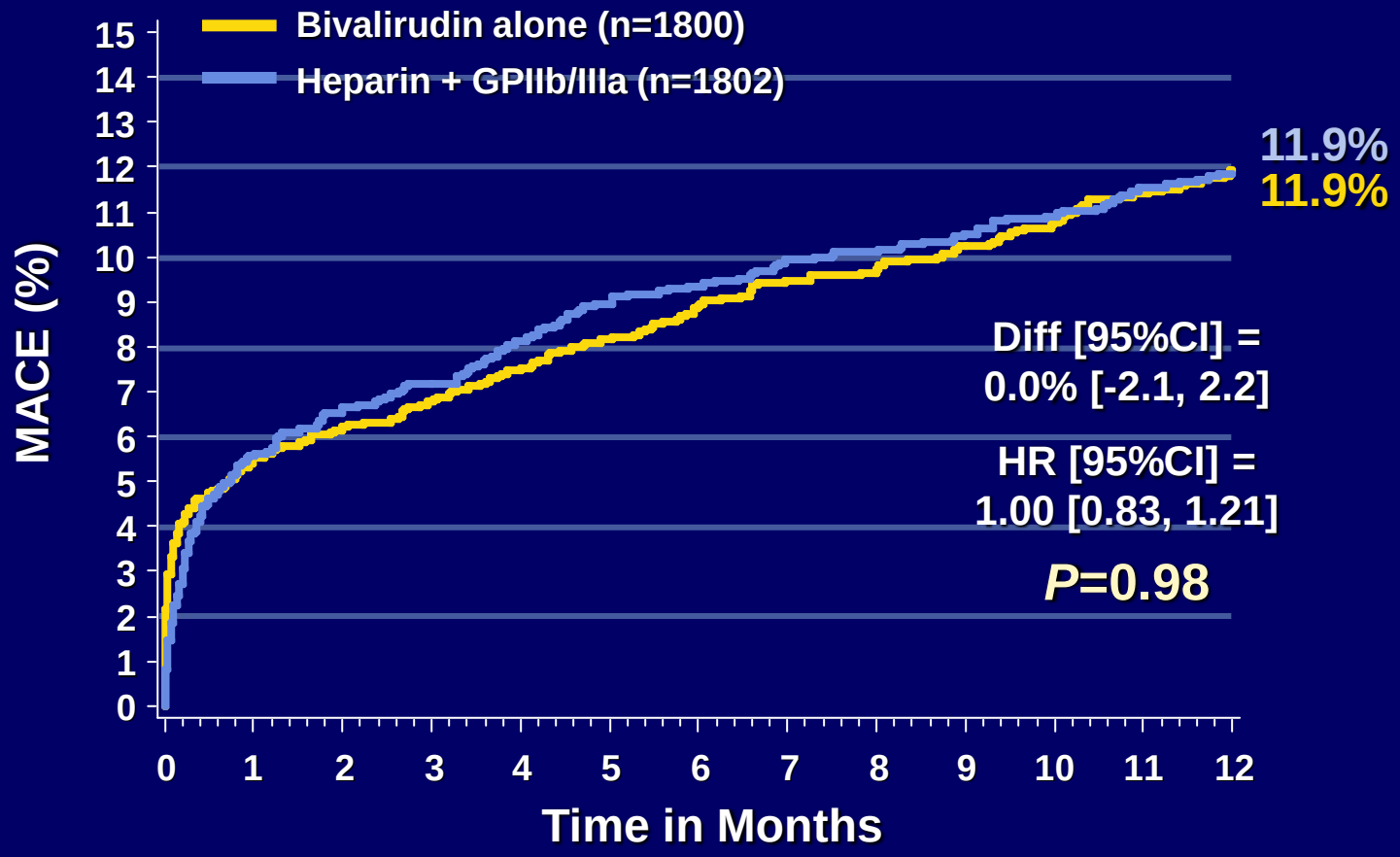
HORIZONS AMI Trial

A Prospective, Randomized Comparison of Bivalirudin vs. Heparin Plus Glycoprotein IIb/IIIa Inhibitors During Primary Angioplasty in Acute Myocardial Infarction

– One Year Results –

Roxana Mehran MD
for the HORIZONS-AMI Investigators, TCT 2008

1-Year Major Adverse CV Events*



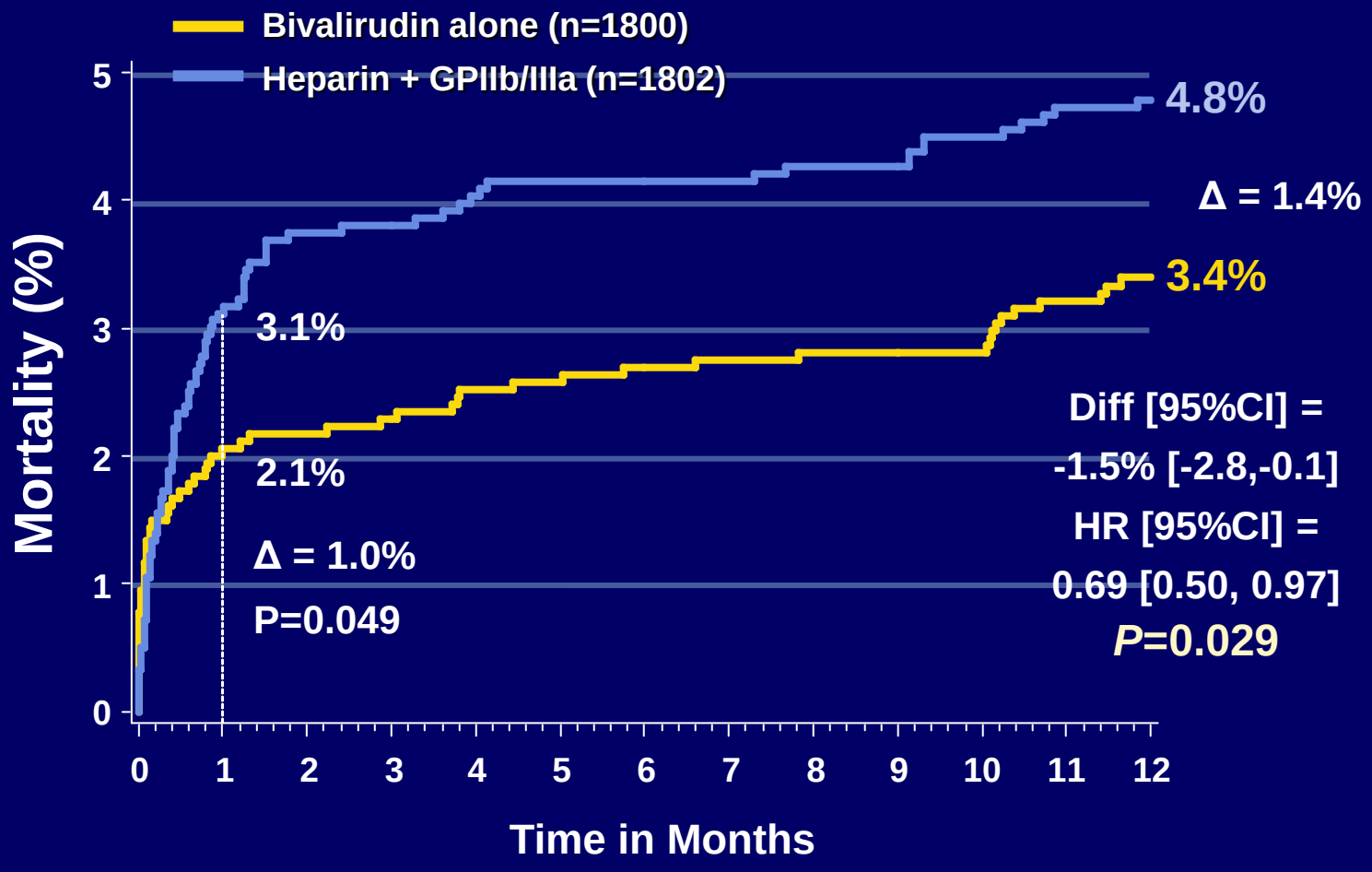
Number at risk

Bivalirudin alone	1800	1627	1579	1544	1394
Heparin+GPIIb/IIIa	1802	1619	1573	1540	1380

*MACE = All cause death, reinfarction, ischemic TVR or stroke

Mehran R, TCT 2008

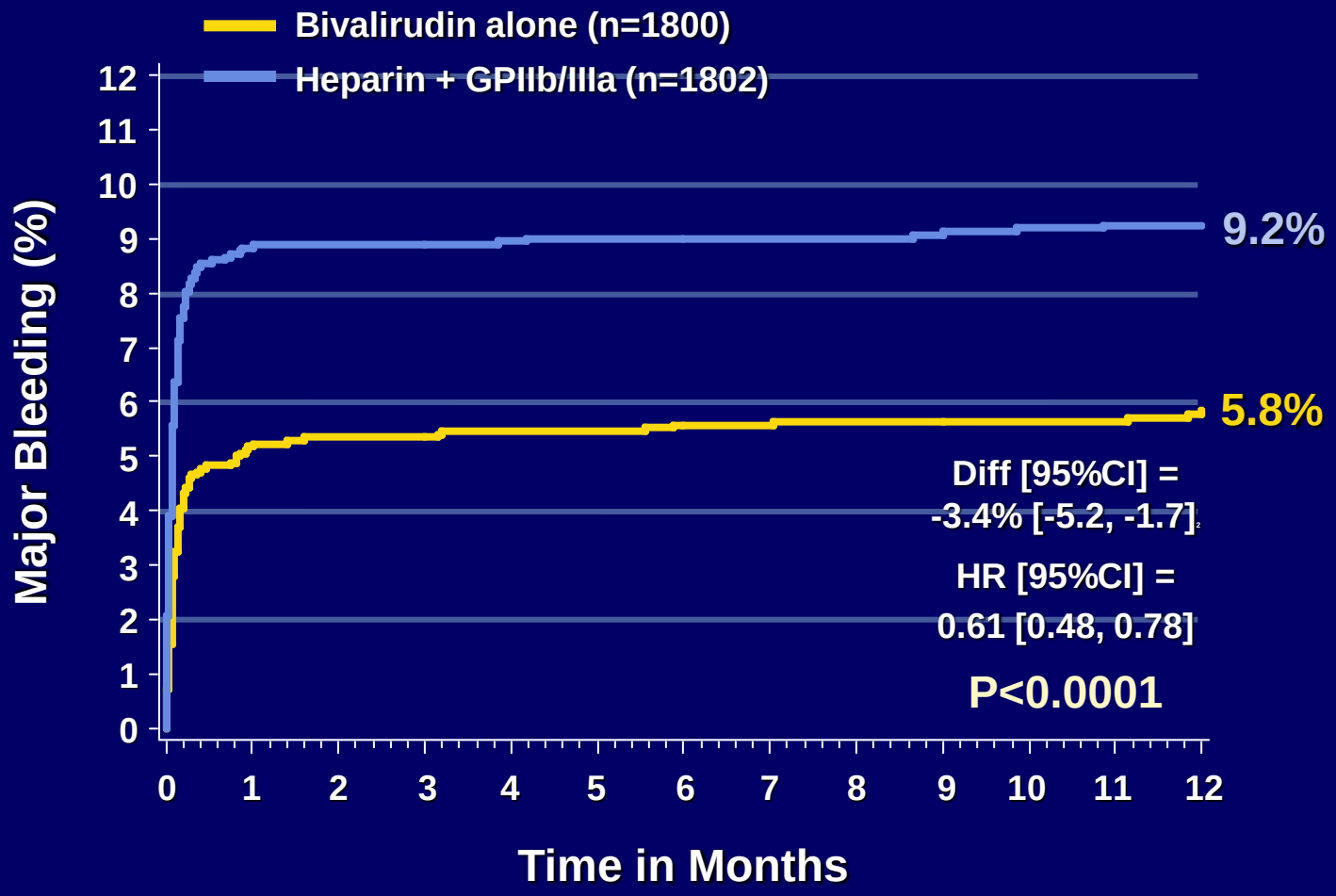
1-Year All-Cause Mortality



Number at risk					
Bivalirudin alone	1800	1705	1684	1669	1520
Heparin+GPIIb/IIIa	1802	1678	1663	1646	1486

Mehran R, TCT 2008

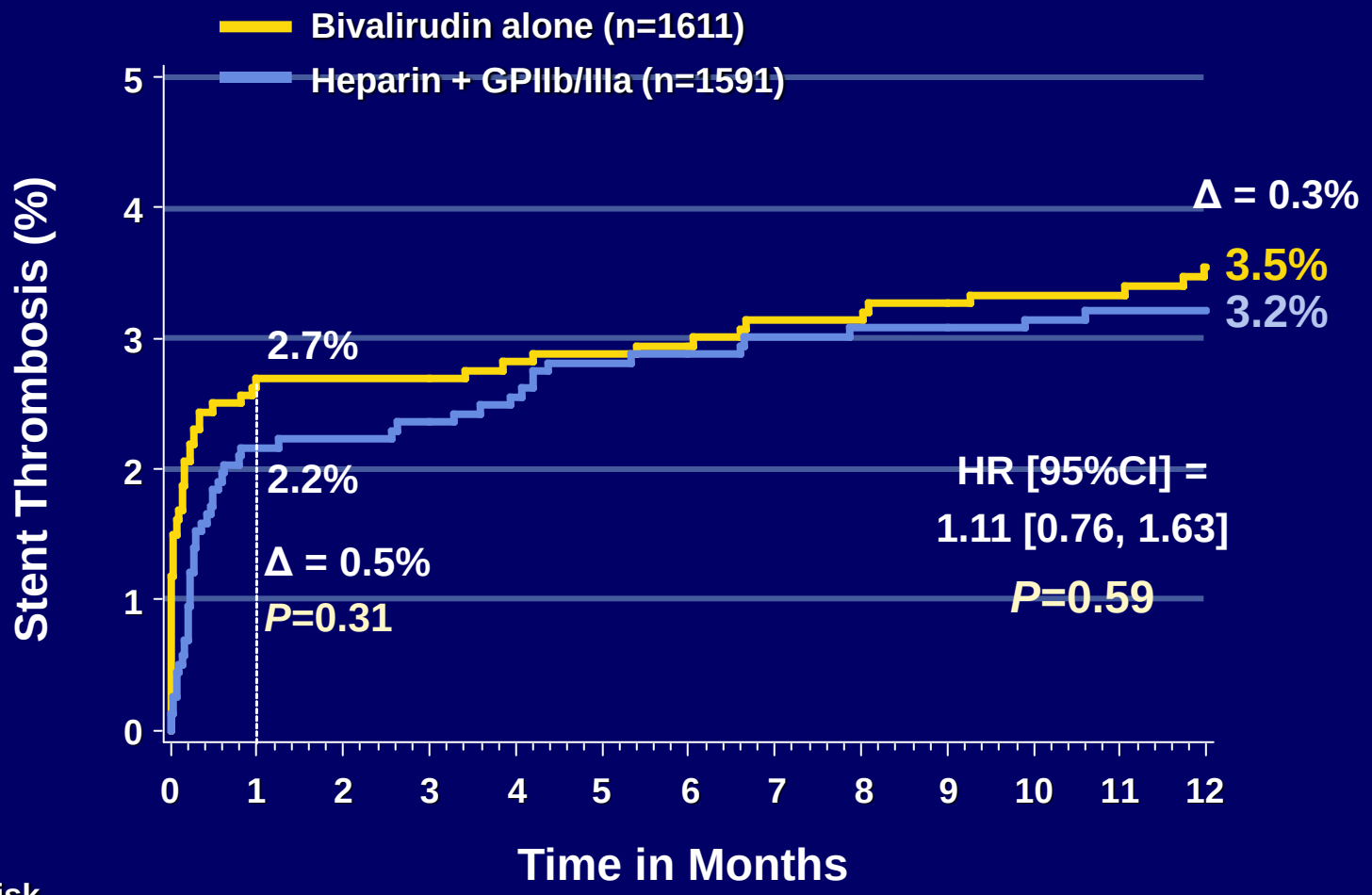
1-Year Major Bleeding (non-CABG)



Number at risk					
Bivalirudin alone	1800	1621	1601	1586	1448
Heparin+GPIIb/IIIa	1802	1544	1532	1515	1368

Mehran R, TCT 2008

1-Year Stent Thrombosis
(ARC Definite/Probable)



Number at risk					
Bivalirudin alone	1611	1525	1504	1486	1356
Heparin+GPIIb/IIIa	1591	1495	1475	1457	1315

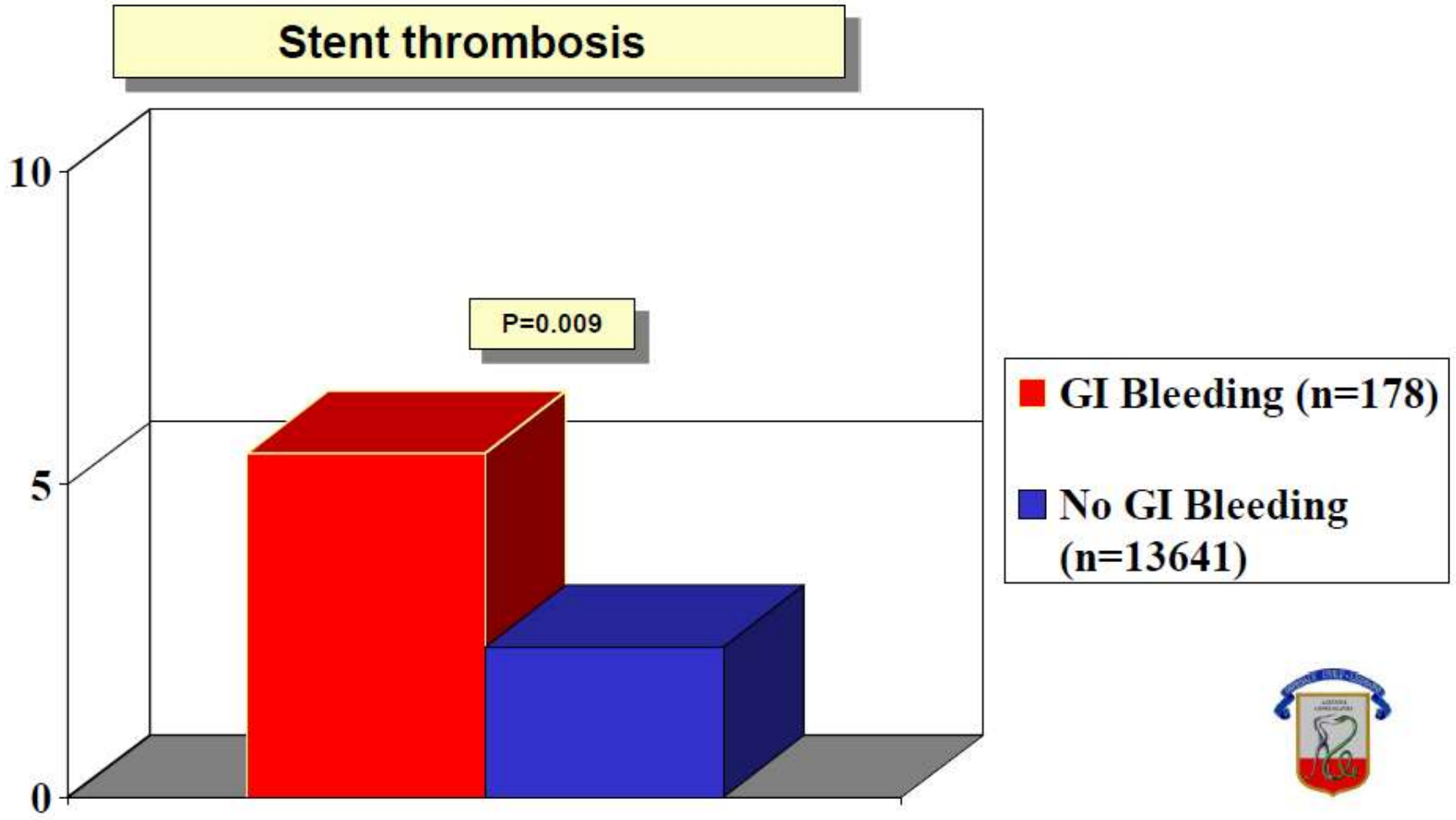
Mehran R, TCT 2008

Gastrointestinal Bleeding in Patients With Acute Coronary Syndromes: Incidence, Predictors, and Clinical Implications

Analysis From the ACUITY (Acute Catheterization and Urgent Intervention Triage Strategy) Trial

Eugenia Nikolsky, MD, PhD,* Gregg W. Stone, MD,* Ajay J. Kirtane, MD, SM,* George D. Dangas, MD, PhD,* Alexandra J. Lansky, MD,* Brent McLaurin, MD,‡ A. Michael Lincoff, MD,§ Frederick Feit, MD,† Jeffrey W. Moses, MD,* Martin Fahy, MSc,* Steven V. Manoukian, MD,‖ Harvey D. White, MD,** E. Magnus Ohman, MD,¶ Michel E. Bertrand, MD,†† David A. Cox, MD,# Roxana Mehran, MD*

New York, New York; Anderson, South Carolina; Cleveland, Ohio; Nashville, Tennessee; Durham, North Carolina; Allentown, Pennsylvania; Auckland, New Zealand; and Lille, France



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Biva

A

Philippe
Peter Cle
Jurrien T
Lu
Holger N
Efthym
Jayne Pr

Table 3. Clinical Outcomes at 30 Days.*

Outcome	Bivalirudin (N=1089)	Control (N=1109)	Relative Risk (95% CI)	P Value
	no. (%)			
Death or non-CABG major bleeding: primary outcome	55 (5.1)	94 (8.5)	0.60 (0.43–0.82)	0.001
Death, reinfarction, or non-CABG major bleeding: principal secondary outcome	72 (6.6)	102 (9.2)	0.72 (0.54–0.96)	0.02
Death	32 (2.9)	34 (3.1)	0.96 (0.60–1.54)	0.86
Cardiac cause	27 (2.5)	33 (3.0)	0.83 (0.50–1.38)	0.48
Noncardiac cause	5 (0.5)	1 (0.1)	5.09 (0.60–43.51)	0.12
Non-CABG bleeding				
Major	28 (2.6)	67 (6.0)	0.43 (0.28–0.66)	<0.001
Major or minor	85 (7.8)	149 (13.4)	0.58 (0.45–0.75)	<0.001
TIMI definition				
Major	14 (1.3)	23 (2.1)	0.62 (0.32–1.20)	0.15
Major or minor	85 (7.8)	146 (13.2)	0.59 (0.46–0.76)	<0.001
GUSTO definition				
Any	85 (7.8)	148 (13.3)	0.58 (0.45–0.75)	<0.001
Severe or life-threatening	6 (0.6)	10 (0.9)	0.61 (0.22–1.68)	0.33
Severe or life-threatening or moderate	14 (1.3)	26 (2.3)	0.55 (0.29–1.04)	0.06
Blood transfusion	23 (2.1)	43 (3.9)	0.54 (0.33–0.90)	0.02
Reinfarction				
Any	19 (1.7)	10 (0.9)	1.93 (0.90–4.14)	0.08
Q-wave	3 (0.3)	2 (0.2)	1.53 (0.26–9.12)	0.68
Non–Q-wave	16 (1.5)	8 (0.7)	2.04 (0.88–4.74)	0.09
Stent thrombosis†				
Definite	17 (1.6)	6 (0.5)	2.89 (1.14–7.29)	0.02
≤24 hr	12 (1.1)	2 (0.2)	6.11 (1.37–27.24)	0.007
>24 hr to 30 days	5 (0.5)	4 (0.4)	1.27 (0.34–4.73)	0.75
Probable	0	0	NA	NA

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HEAT PPCI

***H**ow **E**ffective are*
A**ntithrombotic **T**herapies in **PPCI

Heparin versus Bivalirudin in PPCI

Dr Adeel Shahzad
Dr Rod Stables (PI)
Liverpool Heart and Chest Hospital
Liverpool, UK

Procedural Information

Characteristic	Bivalirudin (%)	Heparin (%)
P2Y12 use - Any	99.6	99.5
- Clopidogrel	11.8	10.0
- Prasugrel	27.3	27.6
- Ticagrelor	61.2	62.7
GPI use	13.5	15.5
Radial arterial access	80.3	82.0
PCI performed	83.0	81.6

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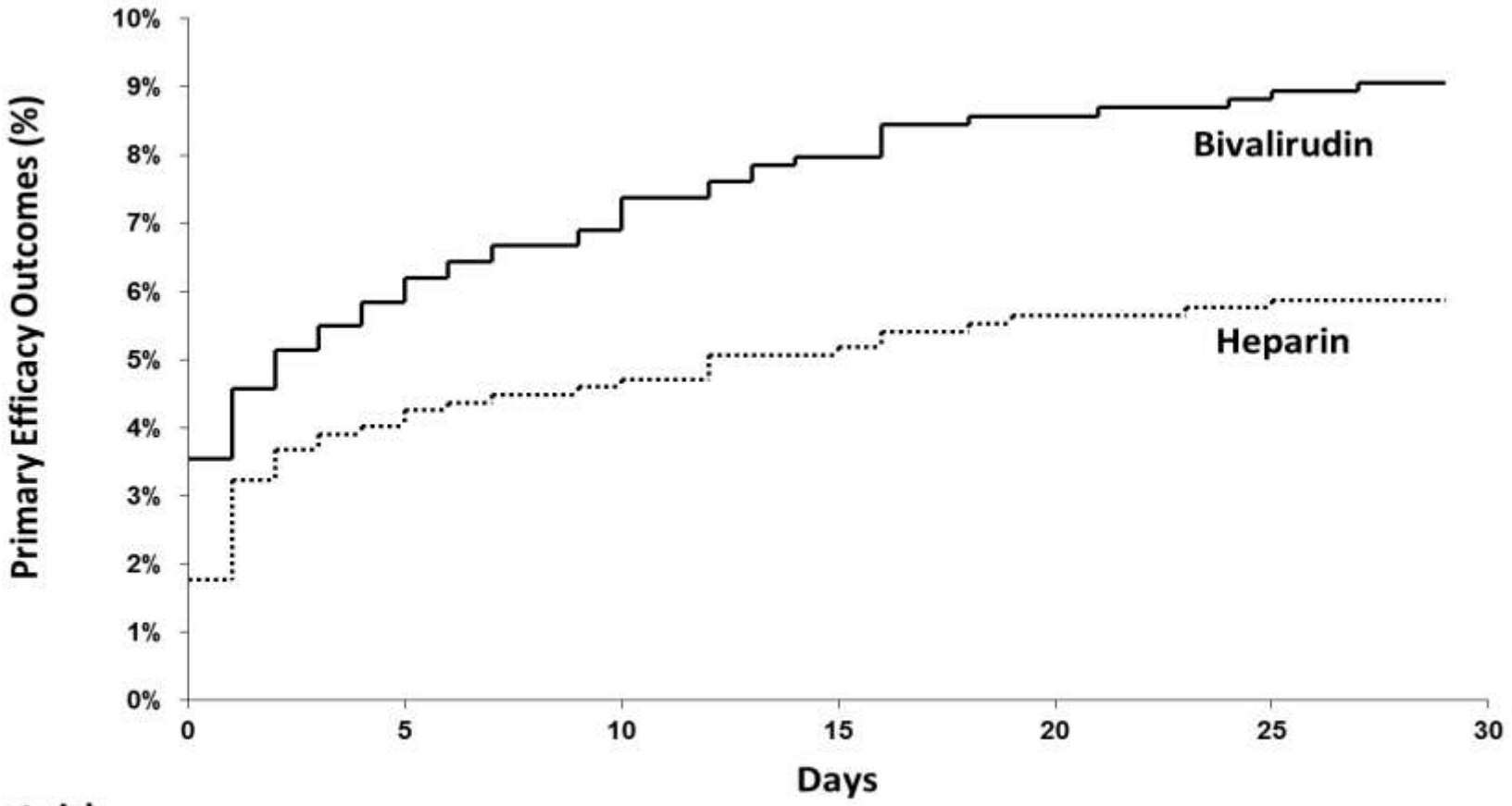
Timing of First MACE Event



No. at risk

Heparin	907	871	866	862	857	856
Bivalirudin	905	853	844	835	830	828

Event curve shows first event experienced

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Stent Thrombosis

ARC definite or probable stent thrombosis events

	Bivalirudin			Heparin	
	n	%		%	n
All Events	24	3.4 %	v	0.9 %	6
Relative risk = 3.91 (95% CI 1.6 - 9.5) P=0.001					

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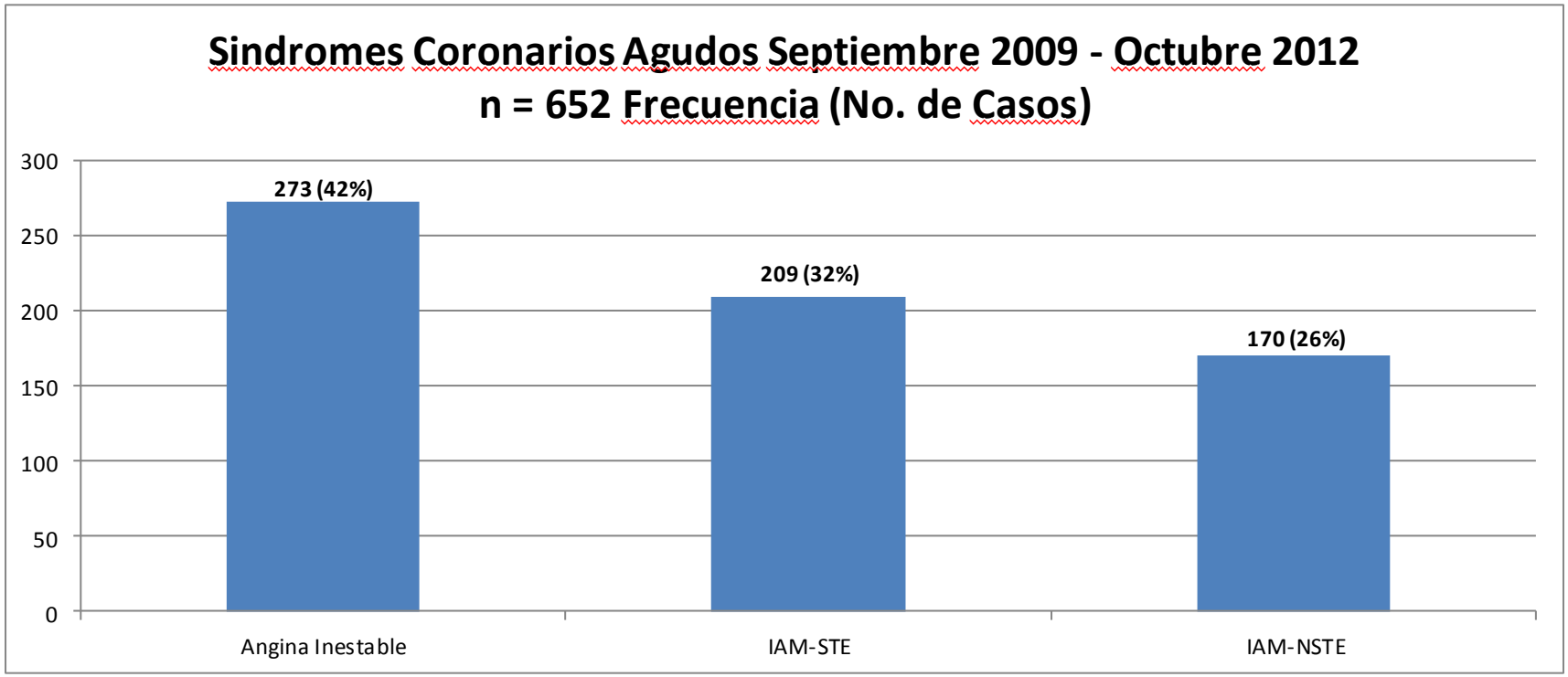
ACS TRI MCA 2009-2012



Villegas A. 2012

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AGIR2

STEMI undergoing **primary PCI**

Patient call

MICU

600 mg clopidogrel
250 mg aspirin
UFH 60 U/kg + inf

Randomize
Open Label

Medical Dispatcher

Pre-hospital

Tirofiban
25/0.15

MICU transportation

Cath lab

Tirofiban
25/0.15

Angiography

Angiography

Initial TIMI grade flow	Cath lab tirofiban N=156	Pre-hospital tirofiban N=163	p
TIMI 3	30.8%	32.5%	
TIMI 2	9.0%	11.7%	0.52
TIMI 0-1	60.3%	55.8%	

Final TIMI grade flow			
TIMI 3	91.7%	93.3%	
TIMI 2	6.4%	3.7%	0.98
TIMI 0-1	1.9%	3.0%	

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Conclusión

La aparicion de nuevos antiplaquetarios aunado al abordaje radial promueve cambios en los protocolos de atencion en el SCA. Los Inhibidores de glicoproteina todavia presentan utilidad en pacientes con carga trombotica y riesgo de sangrado bajo a moderado identificados en la sala de hemodinamia.

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Gracias

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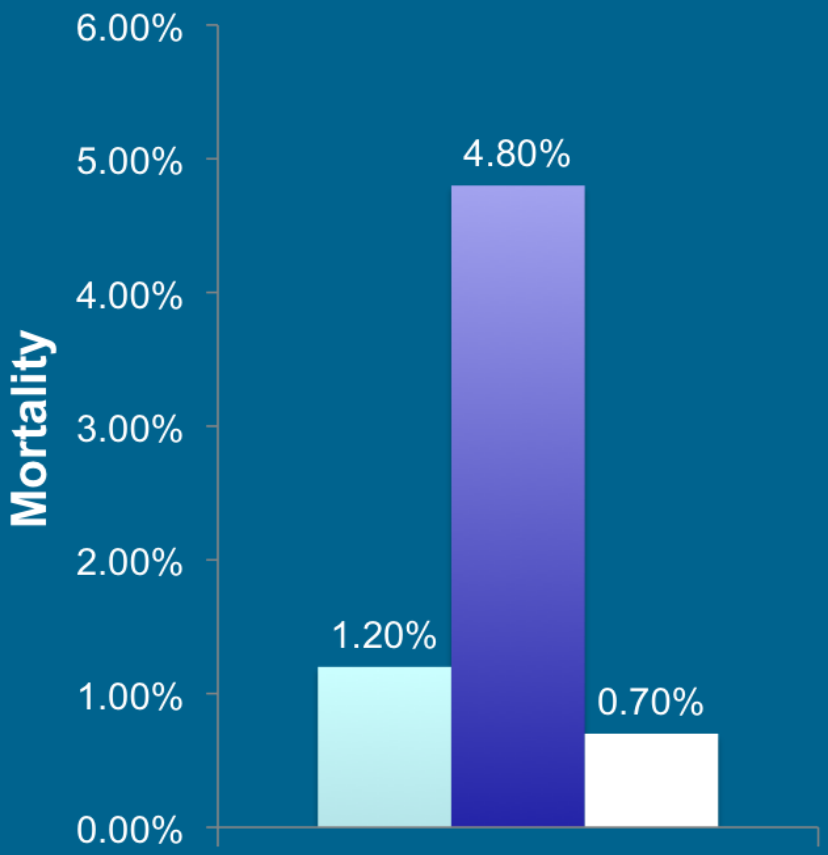
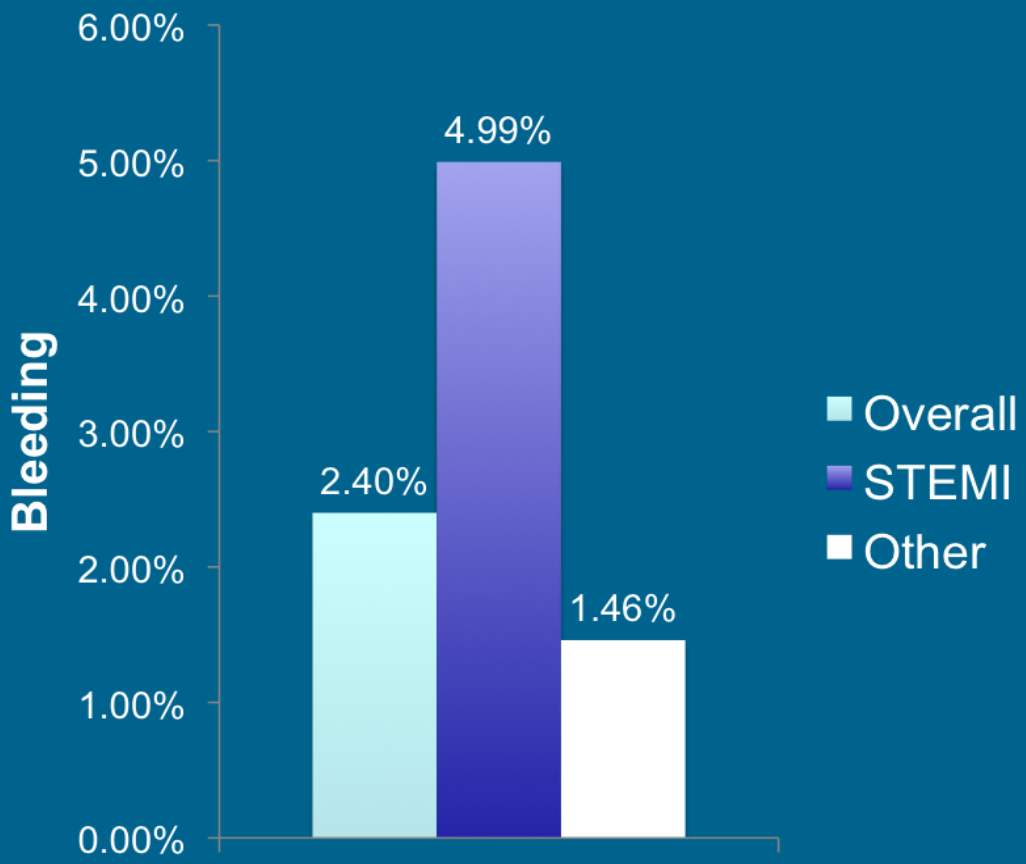
Gracias por su Atencion.

Why bleeding? - In Hospital PCI Mortality & Bleeding





Peterson ED ACC 2007

Mehta SR ACC 2007

Resolucion del Segmento ST

Razon para escoger este punto primario en IAMSTE

- **Resolucion del segmento ST se correlaciona con tamaño del infarto y transmuralidad por RNM o SPECT.**

  Circulation 2004;110(21):e506-10.
  Jama 2005;293(9):1063-72.
  Eur Heart J. 2007 Jun;28(12):1433-9.

- **Resolucion del segmento ST es un factor independiente de pronostico tanto en mortalidad como IAM.**

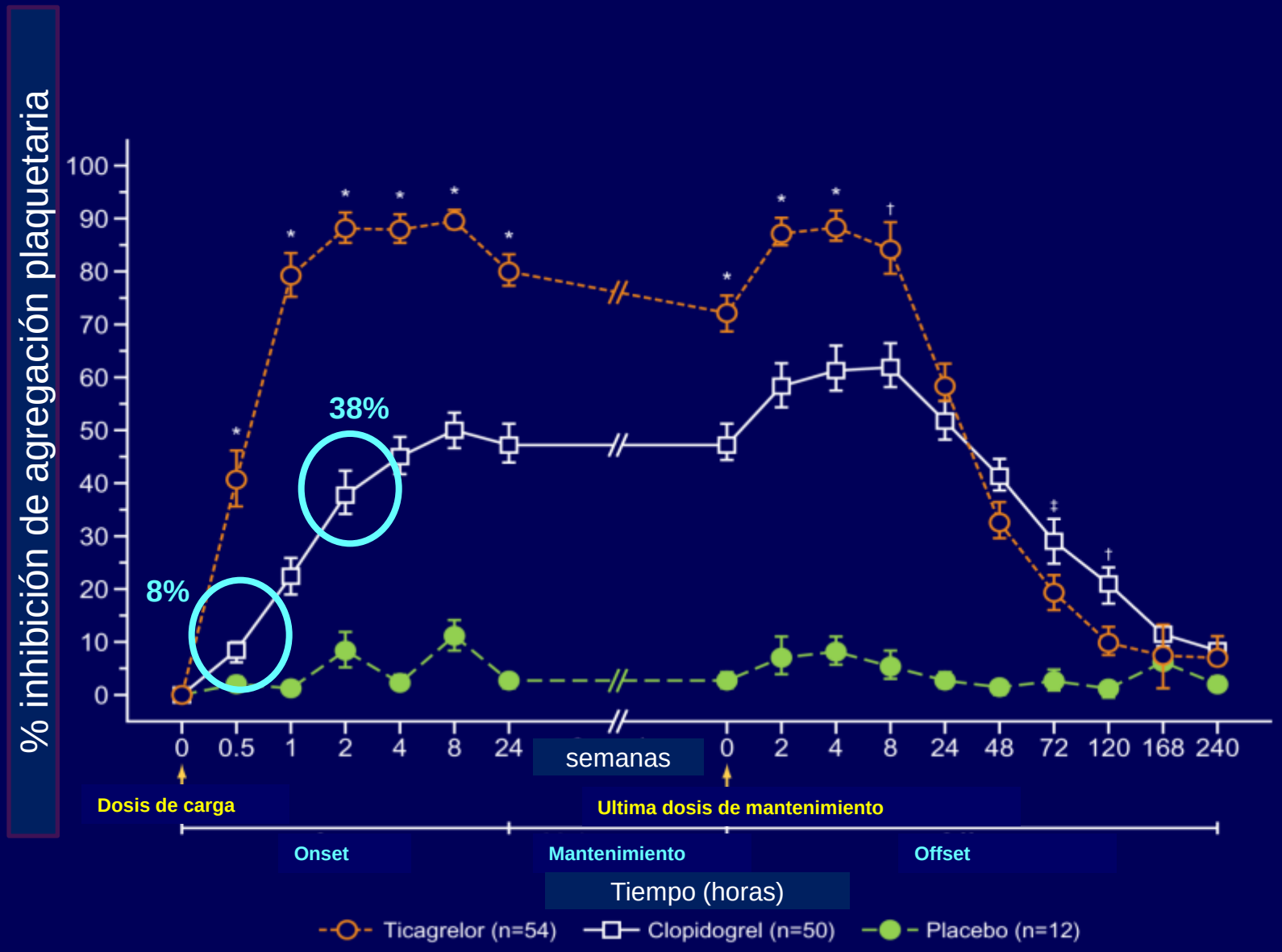
  Lancet 1997;350(9078):615-9

- **Intervenciones en IAMSTE que mejoran la resolucion del segmento ST tienen un efecto consistente en resultados**

  N Engl J Med. 2008 Feb 7;358(6):557-67
  J Am Coll Cardiol 2003;42(11):1879-85
  Jama 2005;293(9):1063-72.

MCA MULTISTRATEGY

ONSET/OFFSET: Inhibición de la agregación plaquetaria



Gurbel PA, et al. *Circulation.* 2009;120:2577-2585.

JACC Journals

From: 2011 ACCF/AHA Focused Update Incorporated Into the ACC/AHA 2007 Guidelines for the Management of Patients With Unstable Angina/Non–ST-Elevation Myocardial Infarction: A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines

J Am Coll Cardiol. 2011;57(19):e215-e367. doi:10.1016/j.jacc.2011.02.011