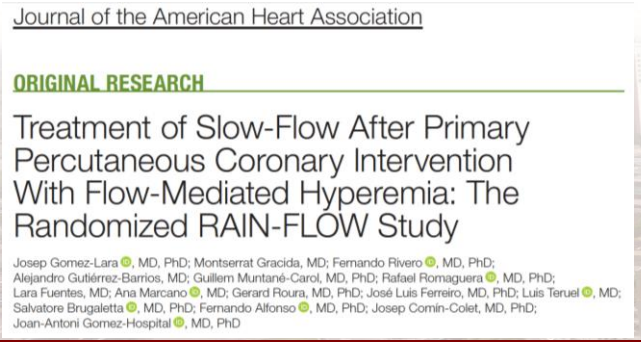
Treatment of slow Flow after Primary Percutaneous coronary Intervention with Flow-mediated hyperemia:

The RAIN-FLOW randomized trial


Journal of the American Heart Association

ORIGINAL RESEARCH

Treatment of Slow-Flow After Primary Percutaneous Coronary Intervention With Flow-Mediated Hyperemia: The Randomized RAIN-FLOW Study

Josep Gomez-Lara, MD, PhD; Montserrat Gracida, MD; Fernando Rivero, MD, PhD; Alejandro Gutiérrez-Barrios, MD; Guillem Muntané-Carol, MD, PhD; Rafael Romaguera, MD, PhD; Lara Fuentes, MD; Ana Marcano, MD; Gerard Roura, MD, PhD; José Luis Ferreiro, MD, PhD; Luis Teruel, MD; Salvatore Brugaletta, MD, PhD; Fernando Alfonso, MD, PhD; Josep Comin-Colet, MD, PhD; Joan-Antoni Gomez-Hospital, MD, PhD

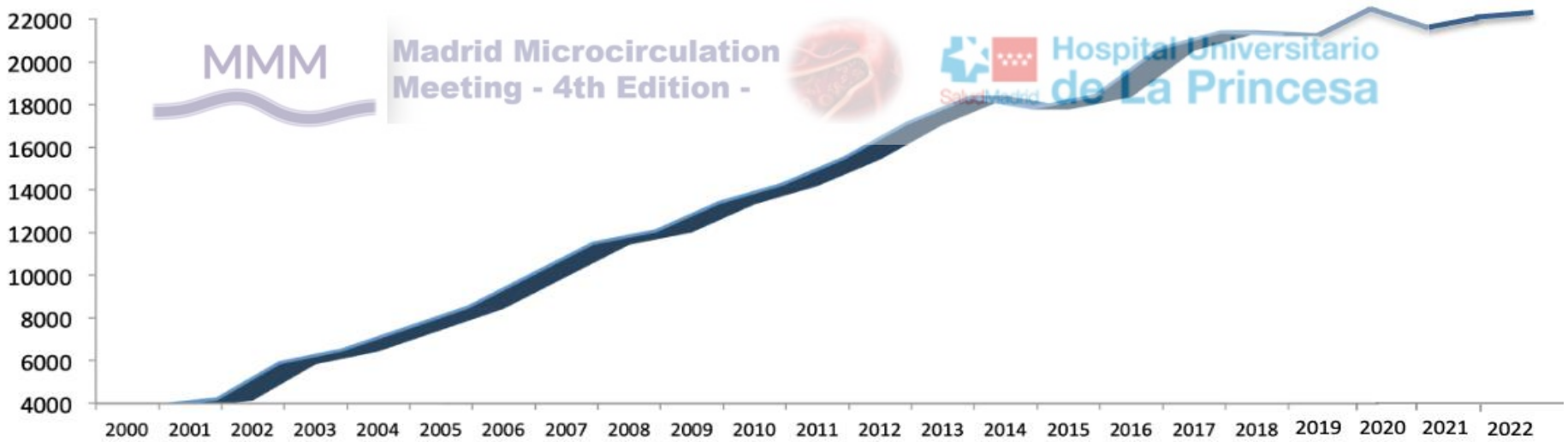
Josep Gómez Lara
Hospital Universitari de Bellvitge, Barcelona
30/11/2023

STEMI in Spain

- Around **30%** of Percutaneous Coronary Interventions (PCIs) are performed as emergent **Primary-PCI** in STEMI patients.

Number of Primary-PCIs

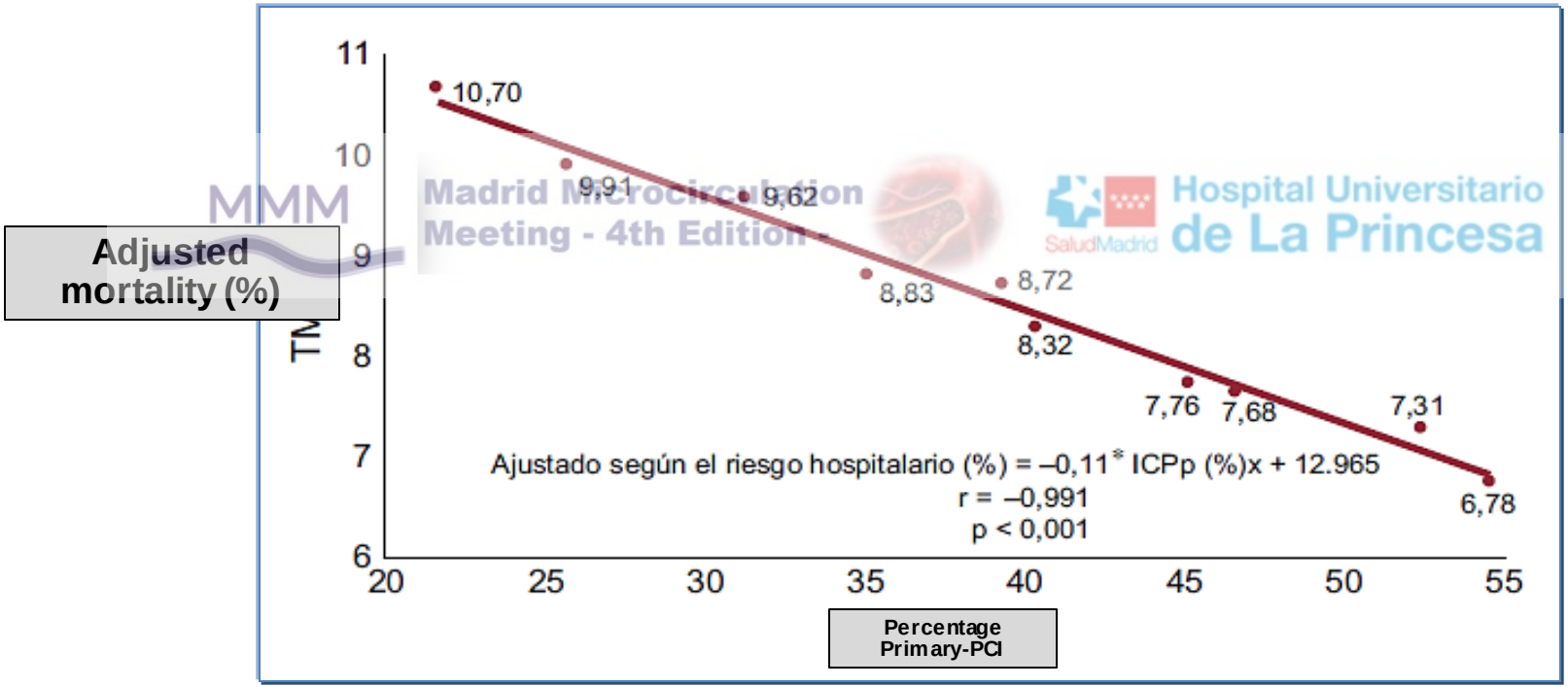
2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022
13.395	14.248	15.491	17.125	18.337	17.825	18.418	20.588	21.395	21.261	22.529	21.039	21.993	22.163



Edited from the Registry of the Interventional Cardiology Association; Spanish Journal of Cardiology

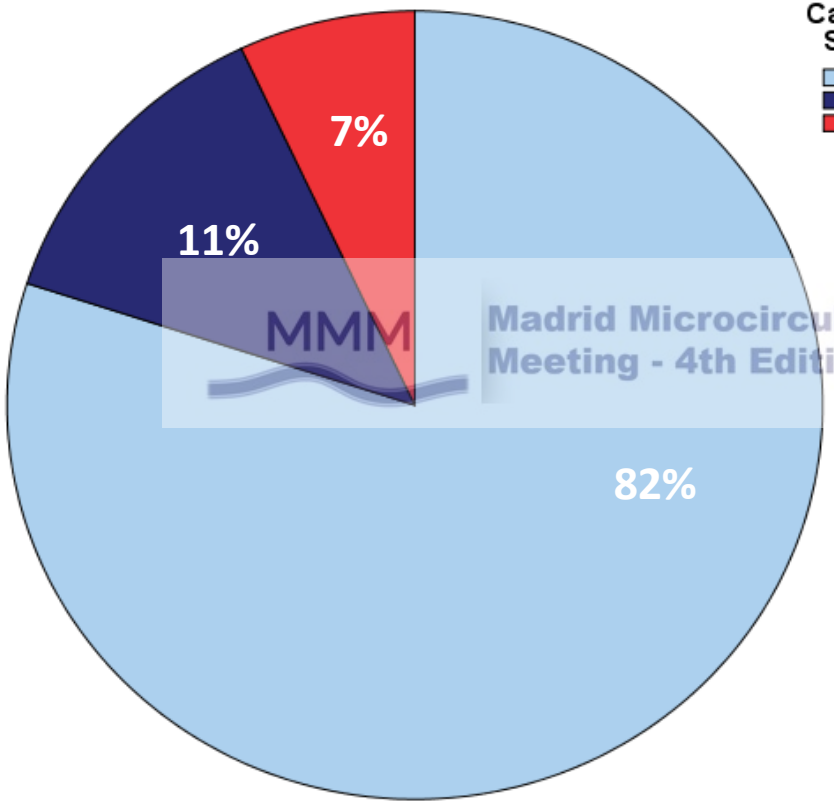
STEMI in Spain

- In the recent years, Primary-PCI programs have been shown effective to reduce the mortality rate in STEMI patients. In 2022 around **5% of in-hospital death in STEMI patients**.



Cequier A, et al.; REC 2017; 70 (3): 155-61

Causes of In-hospital death in STEMI patients:



Pedersen F, et al.; JACC 2014; 64:2101–8

Causes of In-hospital death in STEMI patients:

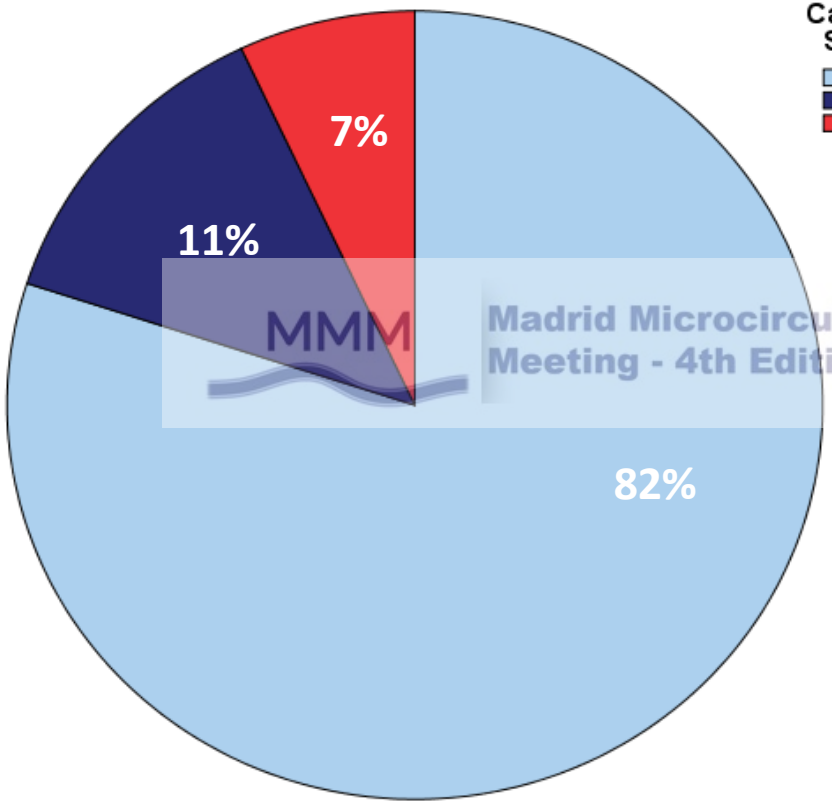


Table 4. Predictors of Mortality During the First Year Obtained From Univariable and Multivariable Cox Proportional Hazards Models

Characteristic	Unadjusted HR (95% CI)	Adjusted HR (95% CI)
No reflow	3.35 (1.97 to 5.69)	1.91 (1.11 to 3.30)
Age (for 10-year increase)	2.06 (1.67 to 2.55)	1.85 (1.49 to 2.28)
Diabetes	2.53 (1.58 to 4.04)	1.81 (1.11 to 2.94)
Killip class (for 1-class increase)	2.76 (2.30 to 3.30)	2.38 (1.97 to 2.88)
Creatinine (for 1-mg/dL increase)	2.19 (1.77 to 2.71)	1.80 (1.38 to 2.35)

LV indicates left ventricle; HR, hazard ratio.

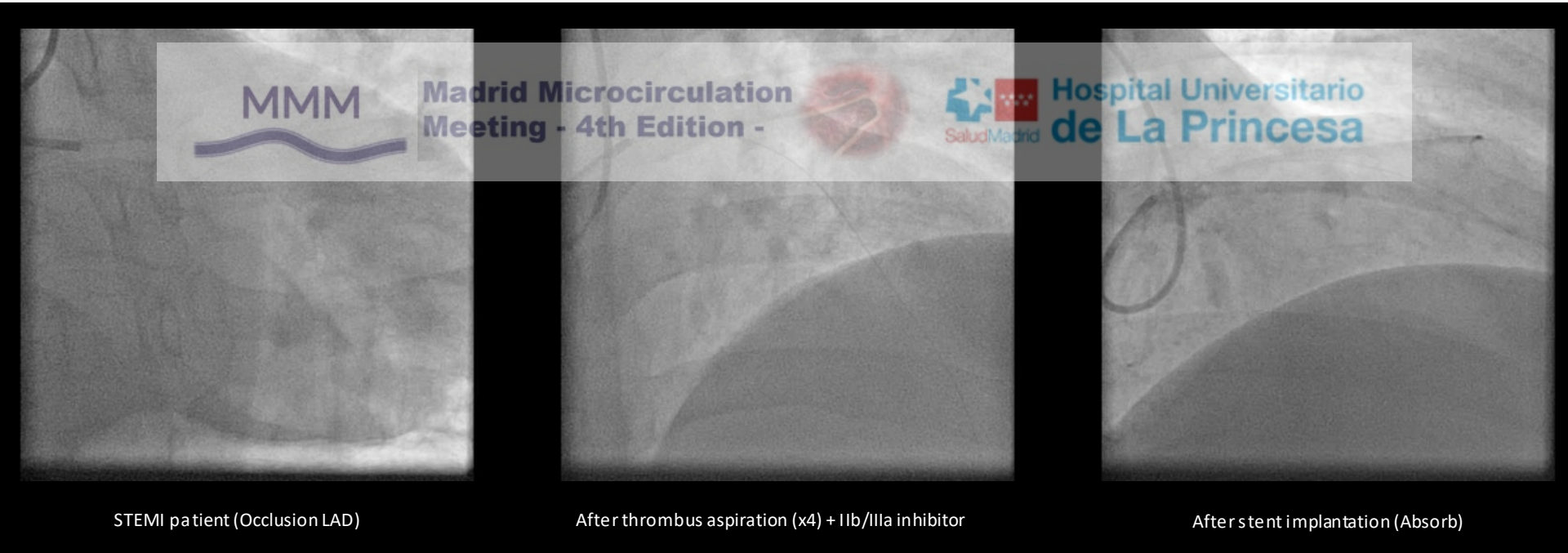
Pedersen F, et al.; JACC 2014; 64:2101–8

Ndrepepa G, et al.; *Circulation CV Int*. 2010; 3:27-33

Slow flow / No reflow

Definition:

Coronary **TIMI flow ≤ 2 after appropriate revascularization** (with stent implantation) without significant residual stenosis (>50% DS), coronary dissection, spasm or thrombus.



STEMI patient (Occlusion LAD)

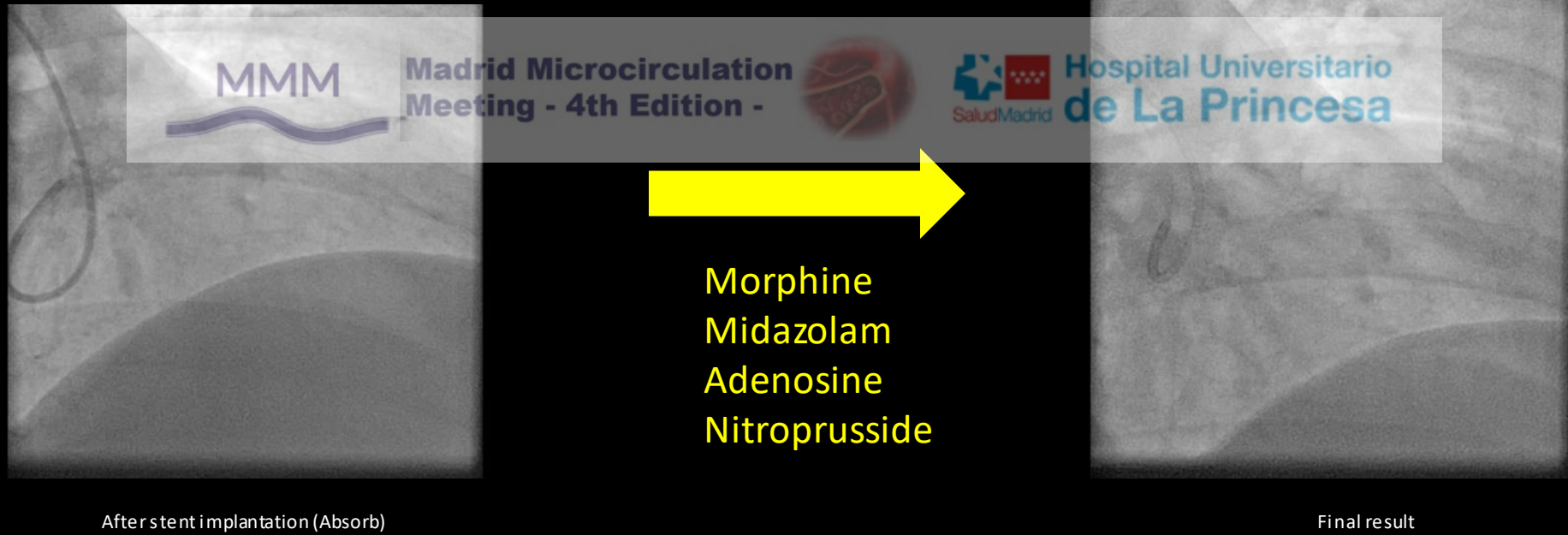
After thrombus aspiration (x4) + IIb/IIIa inhibitor

After stent implantation (Absorb)

Slow flow / No reflow

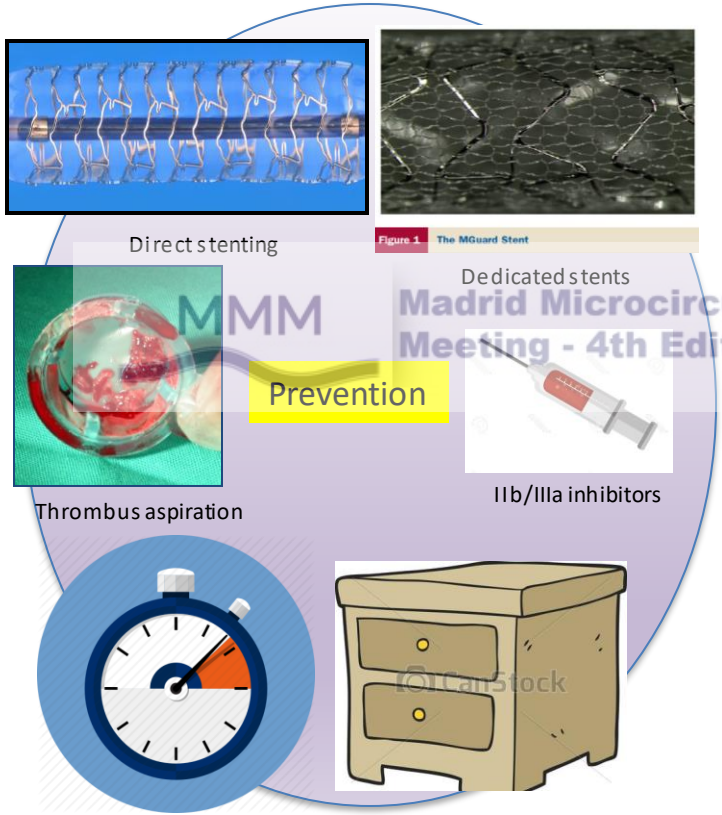
Definition:

Coronary **TIMI flow ≤ 2 after appropriate revascularization** (with stent implantation) without significant residual stenosis (>50% DS), coronary dissection, spasm or thrombus.



Slow flow / No reflow

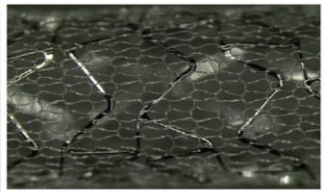
Treatment strategies:



Slow flow / No reflow

Treatment strategies:

Direct stenting



Dedicated stents

Thrombus aspiration

IIb/IIIa inhibitors

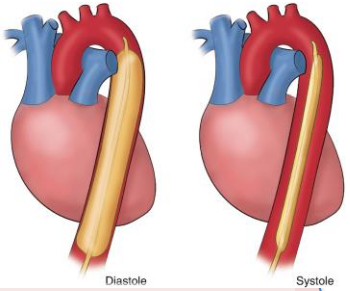
Reperfusion time

Others: B-blokers, statines,,...

Prevention

Treatment

Post-conditioning



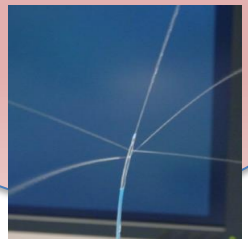
Diastole Systole

IABP

Nicorandil

Adenosine

Nitroprusside



Flow-mediated hyperemia

Slow flow / No reflow

Guidelines:

9.1.5.2. Interventions to protect the microcirculation

The damage inflicted on the myocardium during AMI is the result of ischaemia and subsequent reperfusion (ischaemia/reperfusion injury). In patient-level pooled analyses, infarct size and MVO are independent predictors of long-term mortality and HF in survivors of STEMI.[436,478] Strategies to reduce ischaemia/reperfusion injury in general (and MVO in particular) remain an unmet clinical need. Further information regarding interventions to protect the microcirculation that are under clinical or experimental investigation is presented in the Supplementary data online.

Recommendation Table 5 — Recommendations for antiplatelet and anticoagulant therapy in acute coronary syndrome

Recommendations	Class[a]	Level[b]
Antiplatelet therapy		
GP IIb/IIIa receptor antagonists should be considered if there is evidence of no-reflow or a thrombotic complication during PCI.	IIa	C

Byrne R, et al.; EHJ 2023

The randomized RAIN-FLOW trial

Journal of the American Heart Association

ORIGINAL RESEARCH

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Hypothesis:

- Flow-mediated hyperemia with the Ray-Flow microcatheter at 20 ml/min induces a more powerful, well-tolerated and steady hyperemia than hyperemic drugs.

The randomized RAIN-FLOW trial

Journal of the American Heart Association

ORIGINAL RESEARCH

Treatment of Slow-Flow After Primary Percutaneous Coronary Intervention With Flow-Mediated Hyperemia: The Randomized RAIN-FLOW Study

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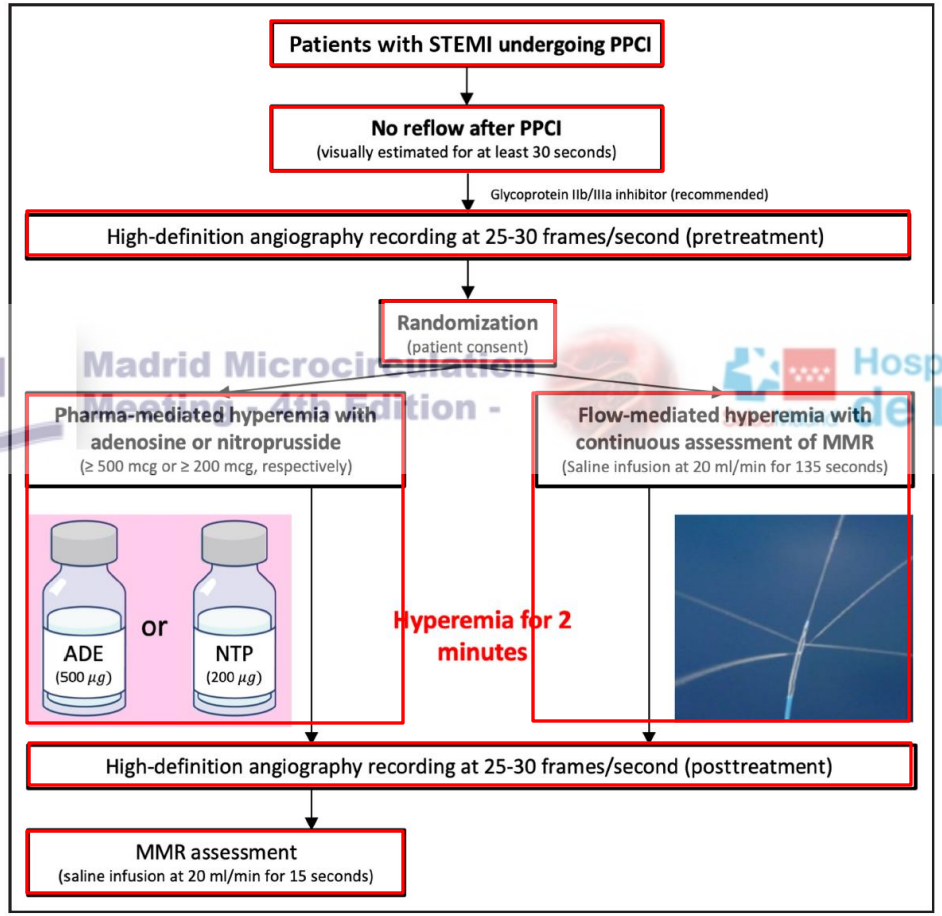
Flow-chart:



Estimated sample size = 100 patients

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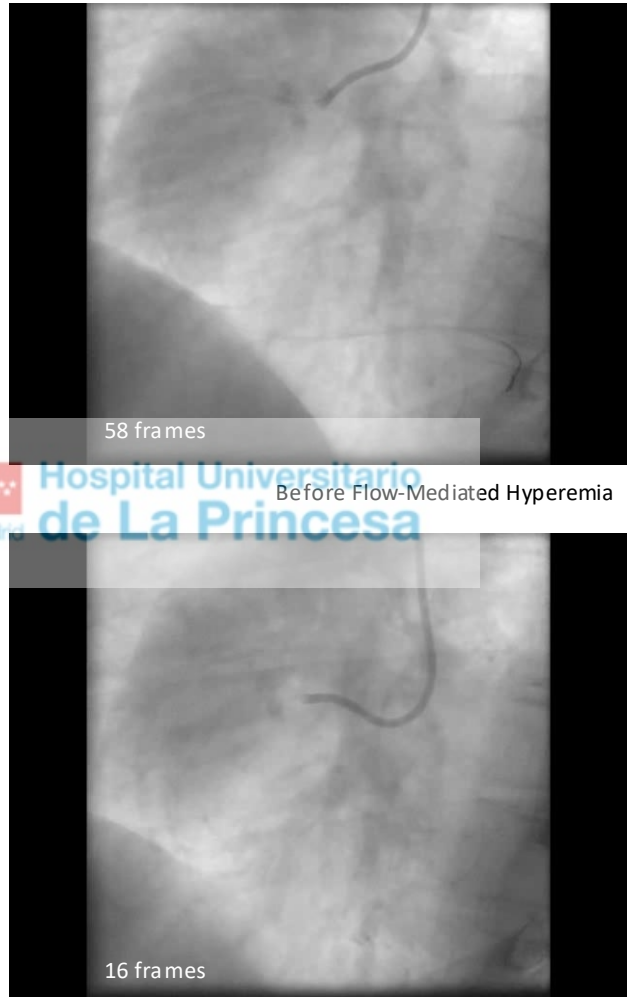
Endpoints:

1) To compare the **TIMI frame count** after 2-min of hyperemia (flow-mediated vs. pharma-mediated). Core-lab assessment.

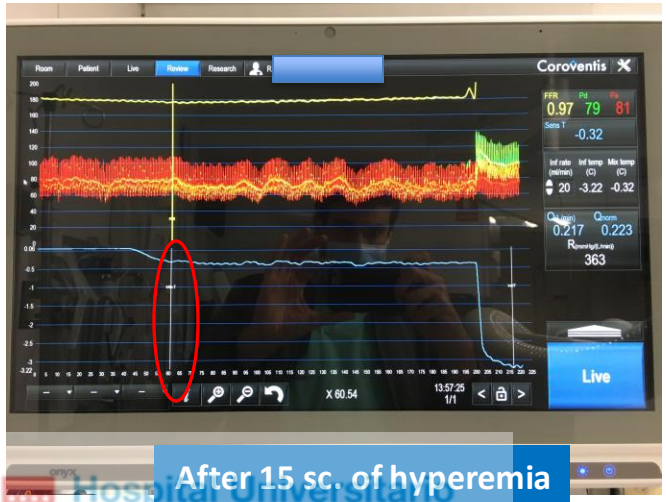


The randomized RAIN-FLOW trial

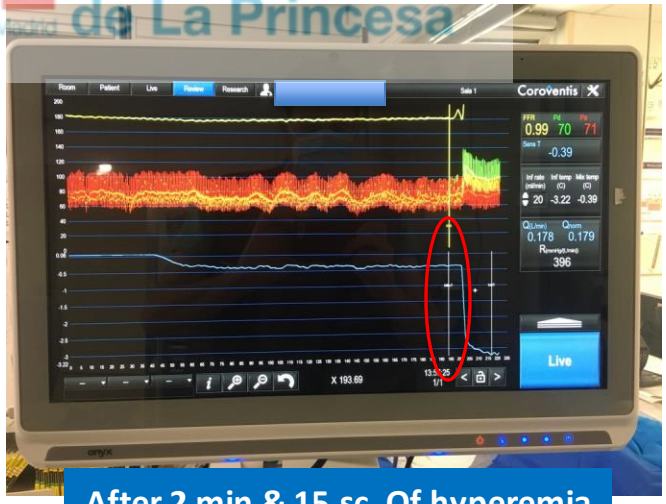
Endpoints:

1) To compare the **TIMI frame count** after 2-min of hyperemia (flow-mediated vs. pharma-mediated). Core-lab assessment.

2) To compare the **Minimal Microcirculatory Resistance (MMR)** after 2'15'' of Flow-Mediated hyperemia (in the flow-mediated group) vs. the MMR at 15' in the Pharma-Mediated group (with prior administrarion of hyperemic drugs). Core-lab assessment.



After 15 sc. of hyperemia



After 2 min & 15 sc. Of hyperemia

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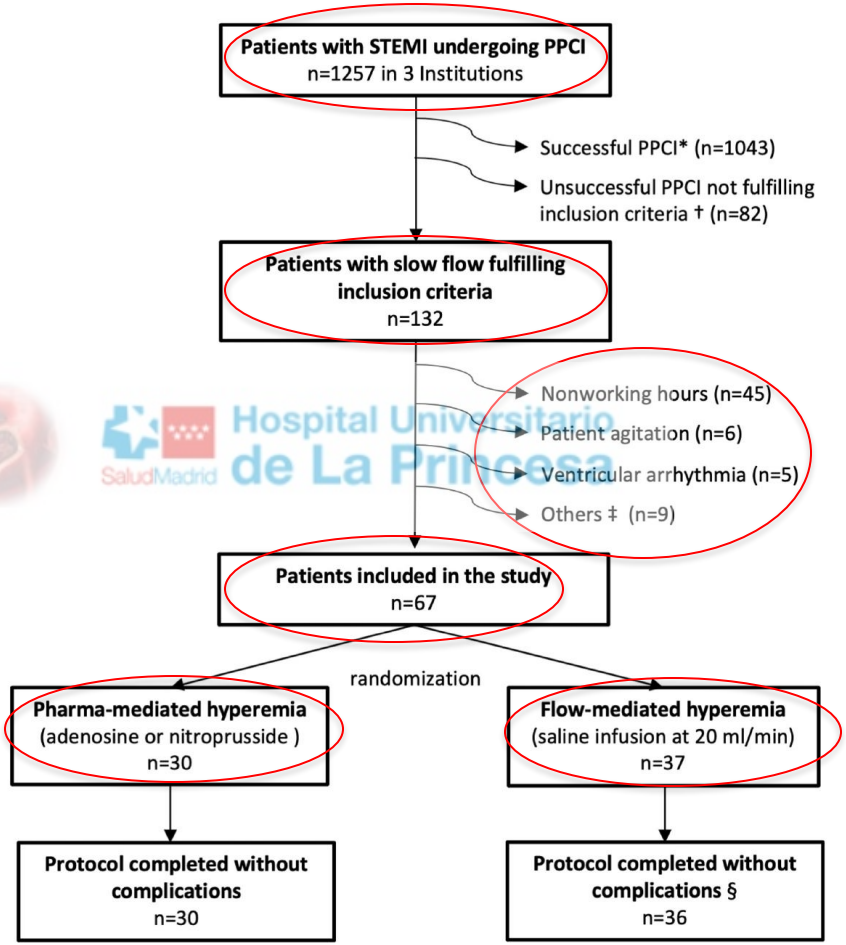
The randomized RAIN-FLOW trial

Results:

The study was prematurely terminated due to:

- Slow recruitment (67 patients after 2 years of inclusion period).
- An interim analysis of efficacy showed no differences in both endpoints.



The randomized RAIN-FLOW trial

Results:

There was no difference regarding both co-primary endpoints:

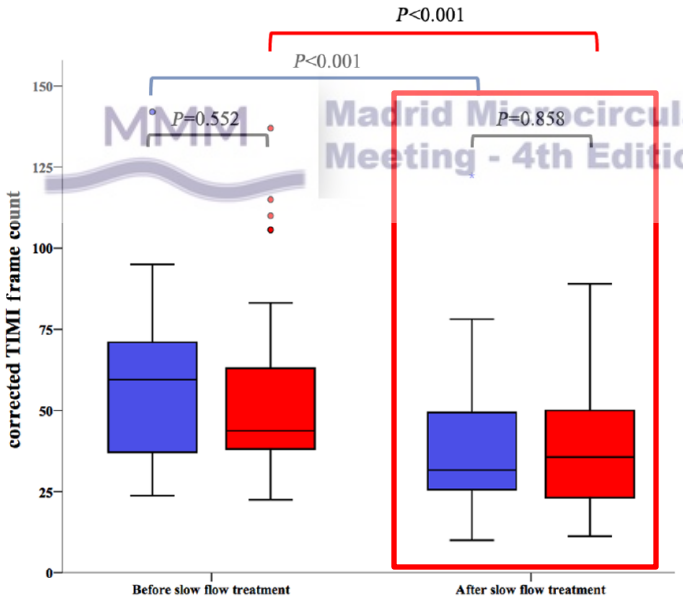


Table 3. Angiographic and ECG Results Before and After Slow Flow Treatment

	Pharma-mediated hyperemia (n=30)	Flow-mediated hyperemia (n=37)	*P* value
TIMI flow before slow-flow treatment			0.917
0	1 (3.3%)	2 (5.4%)	
1	13 (43.3%)	14 (37.8%)	
2	16 (53.3%)	21 (56.8%)	
3	0	0	
TIMI flow after slow-flow treatment			0.938
0	0	0	
1	3 (10.0%)	5 (13.5%)	
2	19 (63.3%)	22 (59.5%)	
3	8 (26.7%)	10 (27.0%)	

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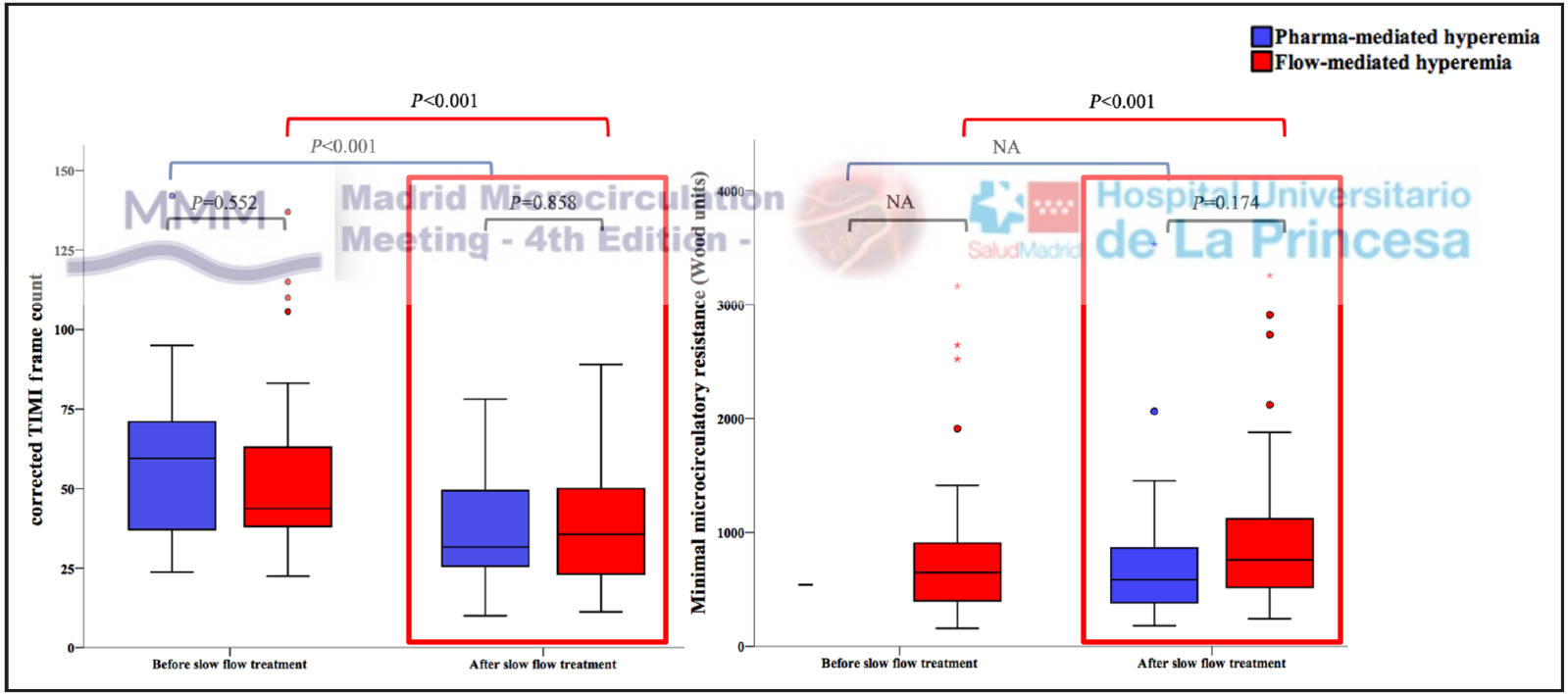
Results:

There was no difference regarding both co-primary endpoints:



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The randomized RAIN-FLOW trial

Results:

In-hospital outcomes:

Table 5. In-Hospital Outcomes

	All patients (n=67)	Pharma-mediated hyperemia group (n=30)	Saline-mediated hyperemia group (n=37)	*P* value
All-cause death	7 (10.4%)	2 (6.7%)	5 (13.5%)	0.447
Cardiac rupture	2 (3.0%)	0	2 (5.4%)	
Acute ventricular septal defect	1 (1.5%)	0	1 (2.7%)	
Cardiogenic shock	3 (4.5%)	1 (3.3%)	2 (5.4%)	
Stent thrombosis	1 (1.5%)	1 (3.3%)*	0	
Nonfatal heart failure	18 (26.9%)	5 (16.7%)	13 (35.1%)	0.105
Hemodynamic support				
Inotropic drugs	9 (13.4%)	2 (6.9%)	7 (18.9%)	0.279
Inotropic drugs+left ventricle assist device	4 (6.0%)	0	4 (11.1%)	0.120
Stent thrombosis	2 (3.0%)	1 (3.3%)*	1 (2.7%)	1.000
Revascularization of nonculprit vessels	20 (30.0%)	7 (23.3%)	13 (35.1%)	0.140
Other nonfatal complications				
Atrial fibrillation (unknown)	2 (3.0%)	2 (6.7%)	5 (13.5%)	0.498
Need permanent pacemaker	1 (1.5%)	0	1 (2.7%)	1.000
Major bleeding	3 (4.5%)	2 (6.7%)	1 (2.7%)	1.000
Intraventricular thrombus	3 (4.5%)	1 (3.3%)	2 (5.4%)	1.000

*One patient with stent thrombosis presented with in-hospital cardiogenic shock and death.

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The randomized RAIN-FLOW trial

Discussion:

Not reliable results of MMR after 2 minutes of saline infusion at 20 ml/min?:

- Angiographic TIMI flow improved after saline but this was not reflected in Q/R values.

Table 4. Physiologic Results After Slow Flow Treatment

	Pharma-mediated hyperemia at 15 s (n=30)	Flow-mediated hyperemia group (n=37)				
		At 15 s	At 135 s	*P**	*P* value†	*P* value‡
Pressure at hyperemia, mm Hg						
Aortic	84.0±18.9	82.5±19.6 =	82.4±18.1	0.901	0.747	0.715
Distal	81.4±19.5	77.0±20.1 =	77.0±18.7	1.000	0.372	0.353
Fractional flow reserve, value	0.97±0.05	0.93±0.07 =	0.93±0.08	0.254	0.021	0.048
Absolute coronary blood flow, mL/min	161.8±101.1	149.6±122.5 >	117.4±84.0	<0.001	0.665	0.056
Normalized value	166.2±105.8	159.8±130.4	121.6±82.0	0.001	0.828	0.058
Minimal microcirculatory resistance, Wood units	753.6±661.5	849.9±702.0 <	993.3±740.8	<0.001	0.571	0.174

*P value indicates the paired differences of physiologic parameters at 15 and at 135 seconds in the flow-mediated hyperemia group.
†P value indicates the difference between the study groups at 15 seconds of the saline infusion.
‡P value indicates the difference between the physiologic results obtained at 15 seconds in the pharmacologic and at 135 seconds in the flow-mediated hyperemia group.

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The randomized RAIN-FLOW trial

Discussion:

Not reliable results of MMR after 2 minutes of saline infusion at 20 ml/min?:

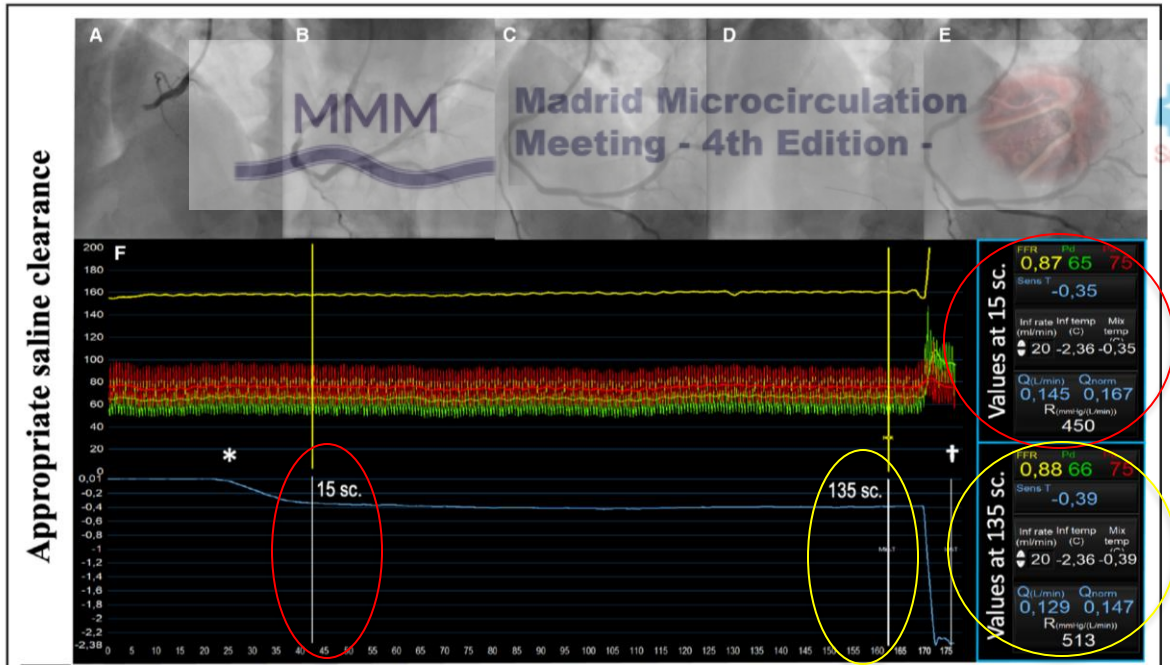
- Angiographic TIMI flow improved after saline but this was not reflected in Q/R values.

The randomized RAIN-FLOW trial

Discussion:

Not reliable results of MMR after 2 minutes of saline infusion at 20 ml/min?:

- Angiographic TIMI flow improved after saline but this was not reflected in Q/R values.
- Two patterns of Q/R during saline infusion in the Flow-Mediated hyperemia group:



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The randomized RAIN-FLOW trial

Discussion:

Not reliable results of MMR after 2 minutes of saline infusion at 20 ml/min?:

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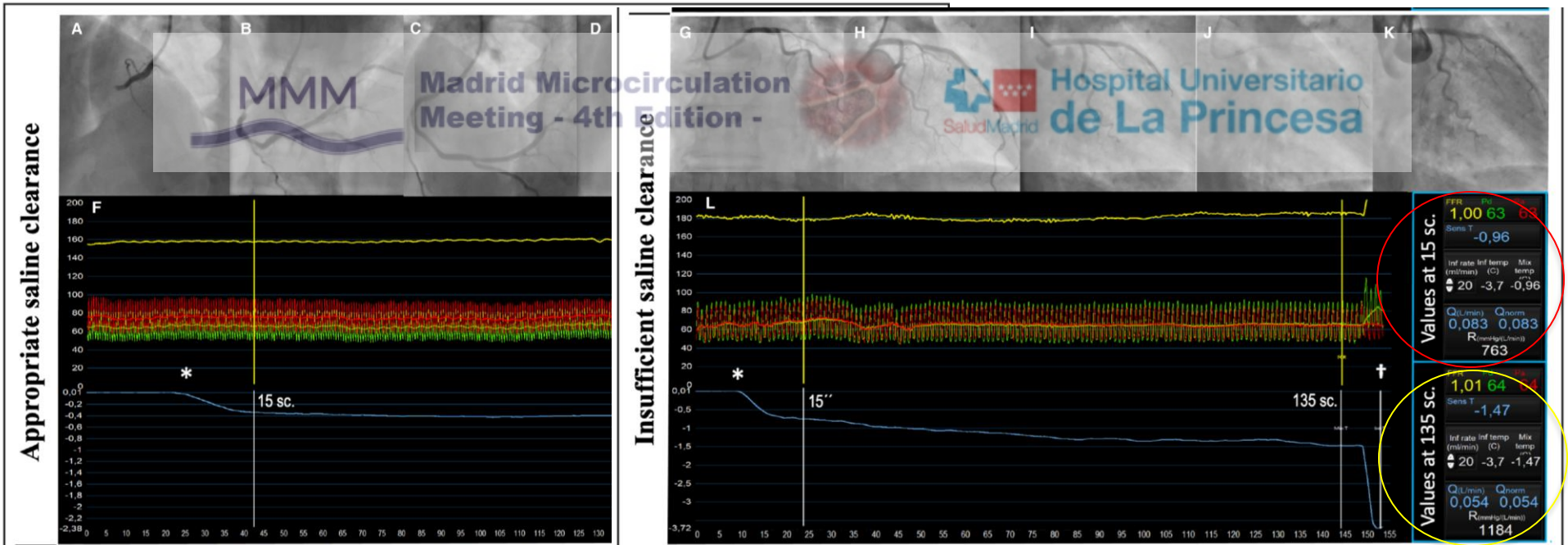

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Discussion:

Insufficient saline clearance was associated with:

- Sub-acute MI
- Worse pre-treatment TIMI flow
- Worse final TIMI flow
- Outcomes

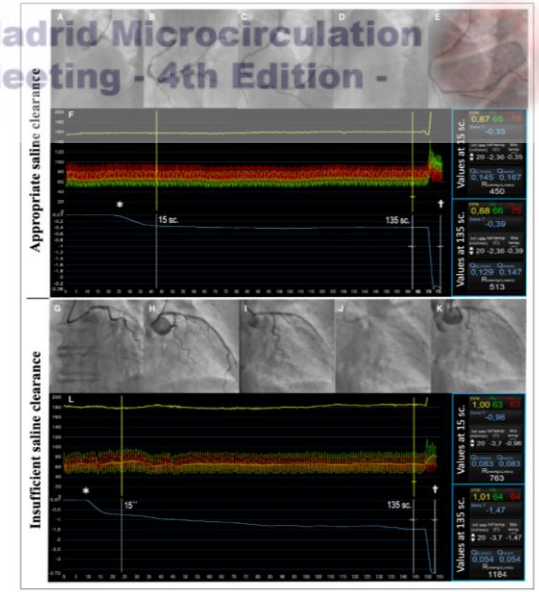

Table S1. Main characteristics of patients with different thermodilution patterns undergoing 2-minute flow-mediated hyperemia.

	Insufficient saline clearance pattern (n=7)	Appropriate saline clearance pattern (n=30)	p
Baseline clinical characteristics:			
Age	72.7 ± 9.6	69.4 ± 13.8	0.552
Males	3 (42.9%)	22 (75.9%)	0.167
Hypertension	6 (85.7%)	15 (51.7%)	0.200
Hypercholesterolemia	3 (42.9%)	18 (62.1%)	0.418
Diabetes mellitus	3 (42.9%)	11 (39.3%)	1.000
STEMI characteristics:			
Chest pain onset to PPCI, min	555.0 ± 246.6	295.0 ± 189.8	0.004
Killip class > 1	3 (42.9%)	11 (36.7%)	1.000
Number vessel disease > 1	3 (42.9%)	16 (53.3%)	0.684
Initial TIMI flow 0	7 (100.0%)	21 (72.4%)	0.309
LAD as culprit vessel	4 (57.1%)	19 (65.5%)	0.686
TIMI flow before slow flow treatment			0.161
0	0	2 (6.9%)	
1	5 (71.4%)	9 (31.0%)	
2	2 (28.6%)	18 (62.1%)	
3	0	0	
TIMI flow after slow flow treatment			0.023
0	0	0	
1	3 (42.9%)	2 (6.9%)	
2	4 (57.1%)	18 (62.1%)	
3	0	9 (31.0%)	
Angiographic cTFC, n:			
Before slow-flow treatment*	76.3 ± 41.9	50.6 ± 23.2	0.045
After slow-flow treatment	60.3 ± 23.2	34.9 ± 17.0	0.002
Delta	15.7 ± 18.2	14.6 ± 18.7	0.891
Physiologic values at 15 seconds			
Absolute coronary blood flow, ml/min	68.9 ± 28.0	169.1 ± 128.7	0.050
Minimal microcirculatory resistance	1384.0 ± 872.3	721.0 ± 603.6	0.023
Fractional flow reserve	0.95 ± 0.09	0.93 ± 0.07	0.408
Physiologic values at 135 seconds			
Absolute coronary blood flow, ml/min	51.9 ± 22.9	133.2 ± 85.9	0.019
Minimal microcirculatory resistance	1711.0 ± 967.7	820.1 ± 570.4	0.003
Fractional flow reserve	0.94 ± 0.11	0.93 ± 0.07	0.723
In-hospital outcomes:			
Death	2 (28.6%)	3 (10.3%)	0.244
Non-fatal Heart Failure	3 (42.9%)	10 (34.5%)	0.686

The randomized RAIN-FLOW trial

Conclusions:

1) Flow-Mediated Hyperemia with 2-min of saline infusion via microcatheter is as effective as hyperemic drugs to improve the final TIMI flow

MMM Madrid Microcirculation Meeting - 4th Edition - Hospital Universitario de La Princesa

The randomized RAIN-FLOW trial

Conclusions:

1) Flow-mediated hyperemia with 2-min of saline infusion via microcatheter is as effective as hyperemic drugs to improve the final TIMI flow

2) However, neither pharmacologic or flow-mediated hyperemia seem effective to restore a normal final TIMI flow and patients still present with remarkable number of in-hospital events.

The randomized RAIN-FLOW trial

Conclusions:

1) Flow-mediated hyperemia with 2-min of saline infusion via microcatheter is as effective as hyperemic drugs to improve the final TIMI flow

2) However, neither pharmacologic or flow-mediated hyperemia seem effective to restore a normal final TIMI flow and patients still present with remarkable number of in-hospital events.

3) Assessment of the hyperemic response to 2-min of flow-mediated hyperemia may distinguish patients with different response to hyperemic stimuli.

MMM

Madrid Microcirculation Meeting - 4th Edition

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Thank You!

Gracias!

Dank je!

Gràcies!

The randomized RAIN-FLOW trial

Results:

Serial assessment of the coronary Q/R in the culprit vessel few days latter:

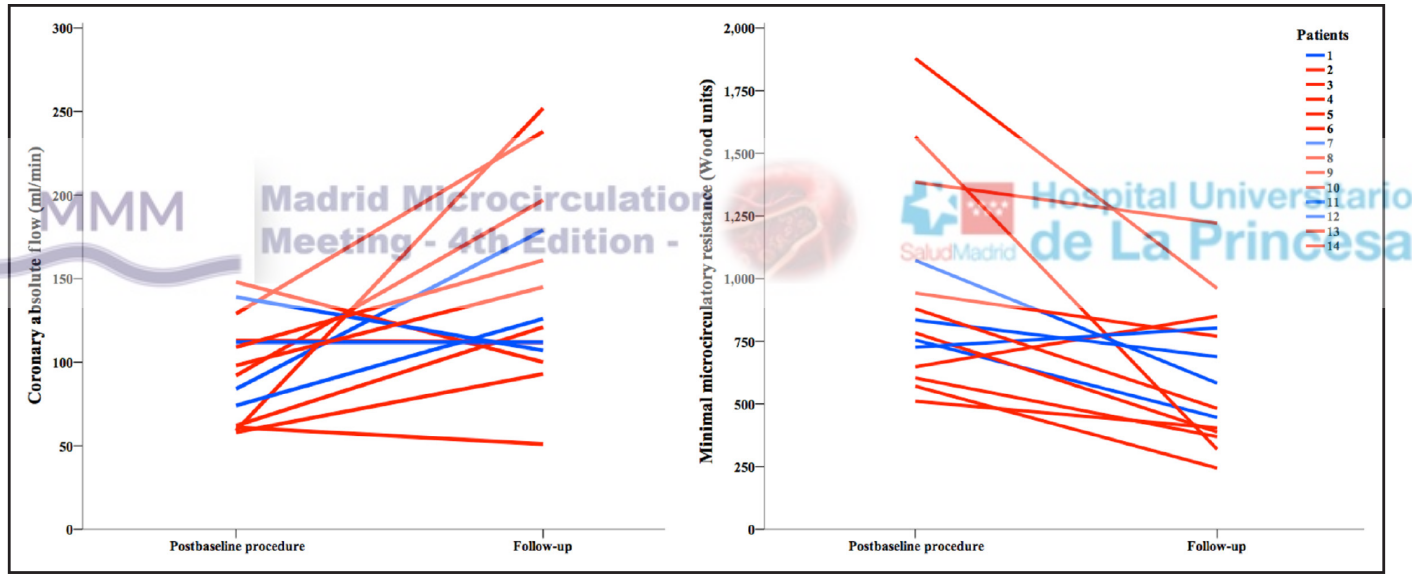

Figure 5. Absolute coronary blood flow and minimal microcirculatory resistance changes between baseline and follow-up procedures.
Fourteen patients underwent thermodilution-based physiologic assessment at baseline (post intervention) and at follow-up. Baseline values were estimated at 15 seconds in the pharmacologic (blue) and at 135 seconds in the flow-mediated hyperemia group (red).

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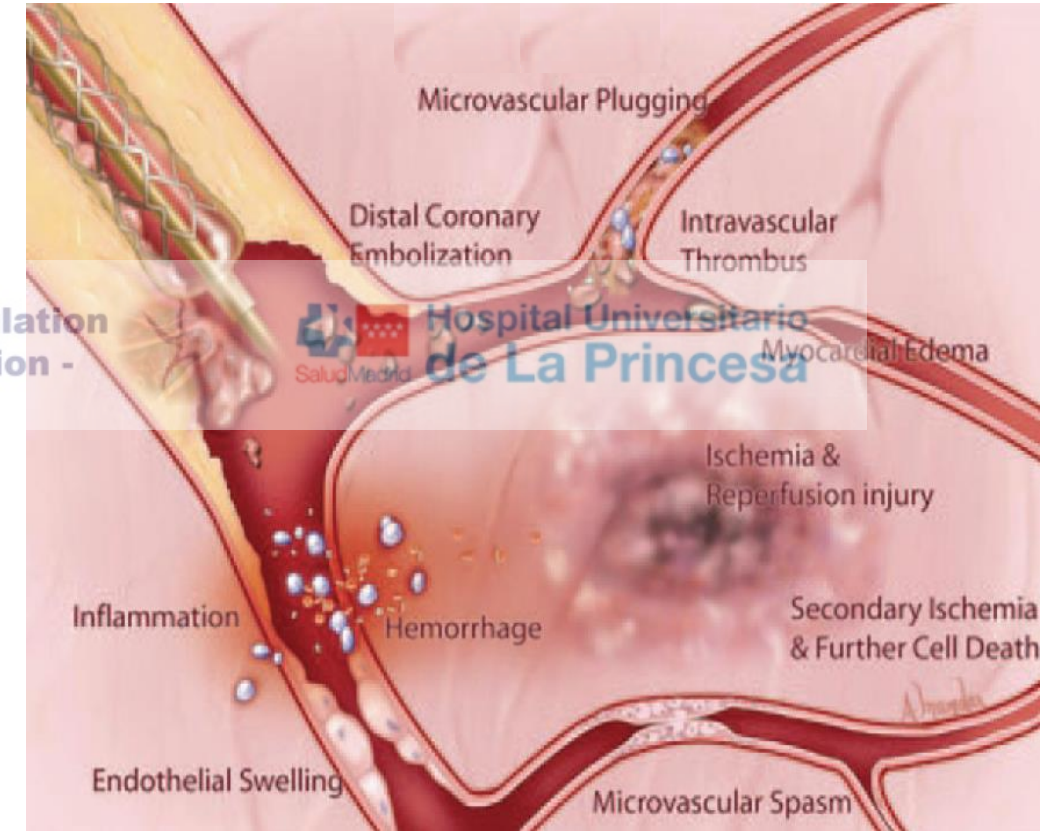
Slow flow / No reflow

Causes:

- Slow flow / No reflow is caused due to **temporary microvascular dysfunction**.



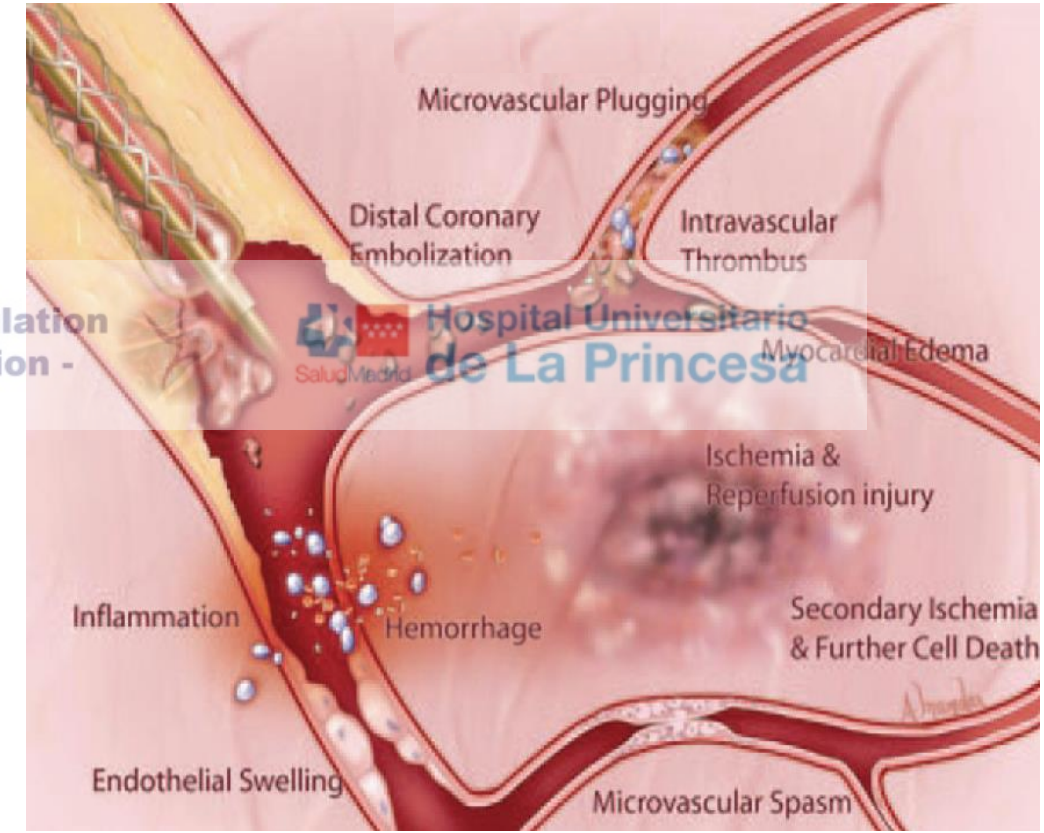
Slow flow / No reflow

Causes:

- Slow flow / No reflow is caused due to **temporary microvascular dysfunction**.

- In STEMI patients with slow flow, microvascular dysfunction is mainly mediated by **microvascular obstruction (MVO)** associated with:

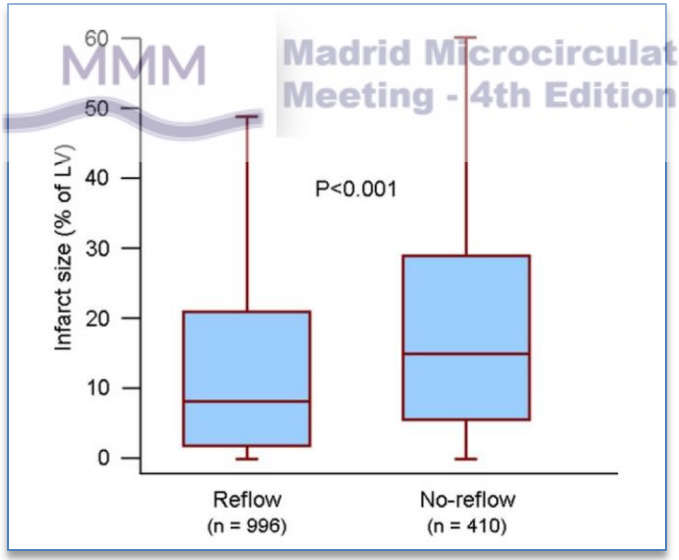
- Thrombus embolization.
- Intra-myocardial edema.
- Microvascular spasm.
- Endothelial dysfunction and swelling.
- Microcirculatory hemorrhages.
- Reperfusion injury.



Slow flow / No reflow

Prevalence and clinical significance:

- n = 1406
- Pts with STEMI undergoing PCI
- No reflow defined as TIMI <3 flow or TMPG 0-1 after successful PCI (10 min post-PCI)
- Occurred in 30% of pts.

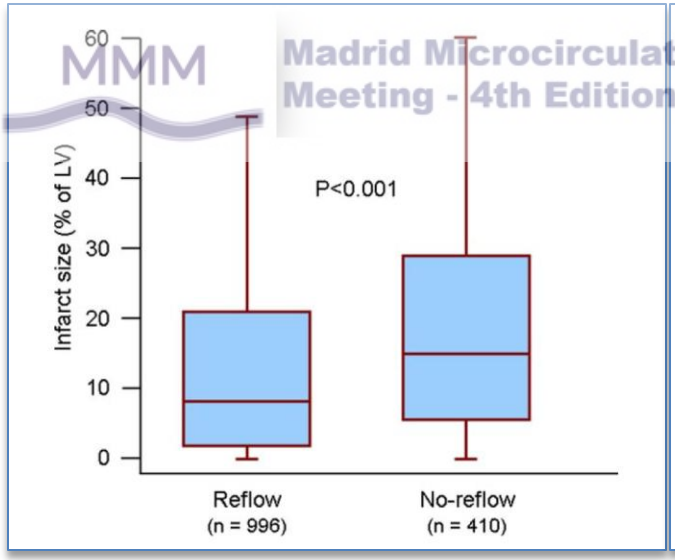


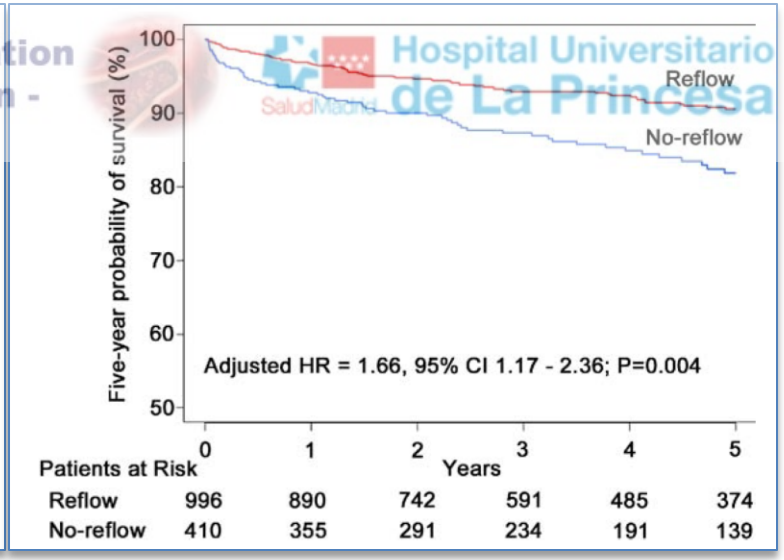
Ndrepepa G, et al. *J Am Coll Cardiol*. 2010;55:2383-2389.

Slow flow / No reflow

Prevalence and clinical significance:

- n = 1406
- Pts with STEMI undergoing PCI
- No reflow defined as TIMI <3 flow or TMPG 0-1 after successful PCI (10 min post-PCI)
- Occurred in 30% of pts.





Ndrepepa G, et al. *J Am Coll Cardiol*. 2010;55:2383-2389.

Slow flow / No reflow

AMISTAD II trial

Adenosine:

- A total of 2118 patients with STEMI treated with Primary-PCI or Thrombolysis.
- Randomized to 3: placebo *vs.* Adenosine 50 μg/Kg/min *vs.* 70 μg/Kg/min for 3 hours.
- Underwent to SPECT at 24 hours of the hospitalization.

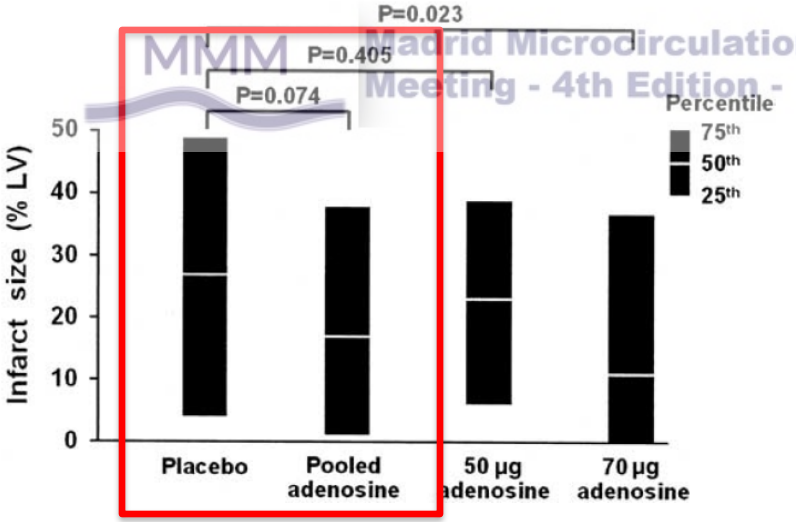


Table 3. Primary Clinical End Points

End Point	Treatment Groups: Placebo	Pooled Adenosine	Adenosine* 50 µg/kg/min	Adenosine 70 µg/kg/min	p Value†
Intention-to-treat analysis					
n	703	1,414	701	713	
Death	83 (11.8%)	146 (10.3%)	73 (10.4%)	73 (10.2%)	0.29
In-hospital CHF	28 (4.0%)	60 (4.2%)	28 (4.0%)	32 (4.5%)	0.75
Re-hospitalization for CHF	30 (4.3%)	56 (4.0%)	27 (3.9%)	29 (4.1%)	0.81
Composite	126 (17.9%)	231 (16.3%)	116 (16.5%)	115 (16.1%)	0.43

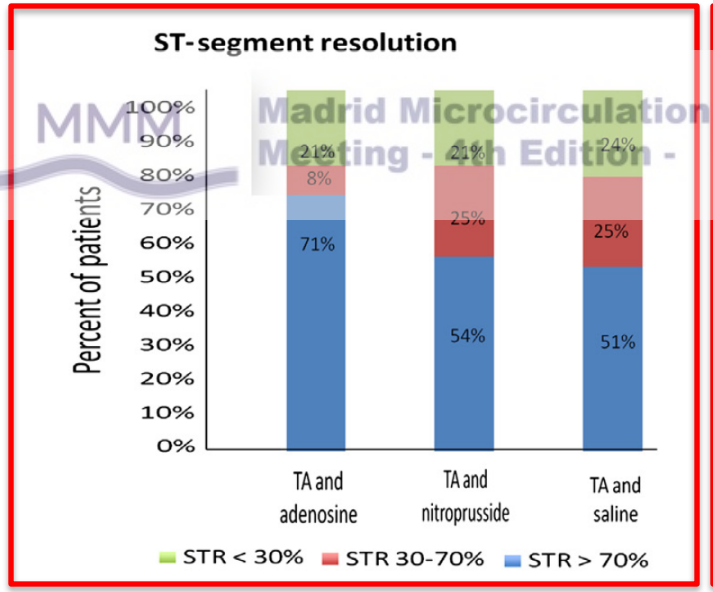
Ross AM, et al. *J Am Coll Cardiol*. 2005; 45:1775– 80.

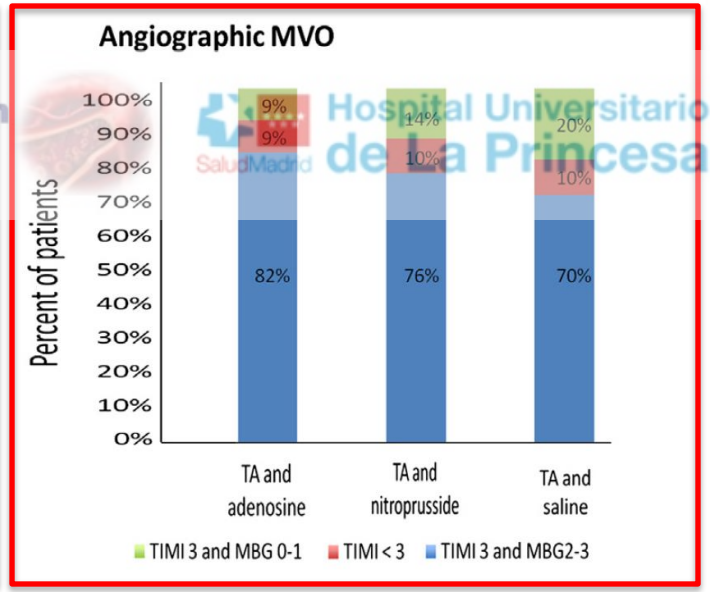
Slow flow / No reflow

REOPEN-AMI trial

Adenosine & Nitroprusside:

- A total of 240 patients with STEMI and occluded culprit lesion (TIMI 0/I pre-ICP).
- Randomized to: thrombus aspiration (TA) vs. TA+Adenosine vs. TA+Nitroprusside via microcatheter distal to the occlusion.





Niccoli G, et al.; J Am Coll Cardiol Intv 2013;6:580–9.

Slow flow / No reflow

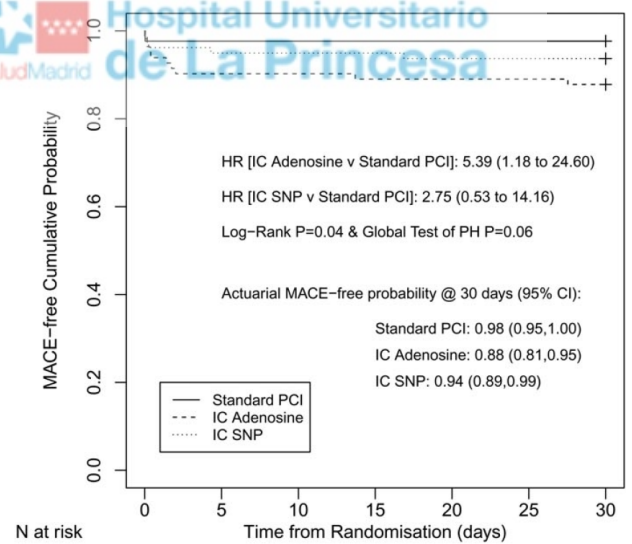
REFLO-STEMI trial

Adenosine & Nitroprusside:

- A total of 247 patients with STEMI.
- Randomized to: thrombus aspiration (TA) vs. TA+Adenosine vs. TA+Nitroprusside via microcatheter distal to the occlusion.
- Underwent to cardiac MRI 1-4 days after PCI.

Table 3 Cardiac magnetic resonance data according to treatment group

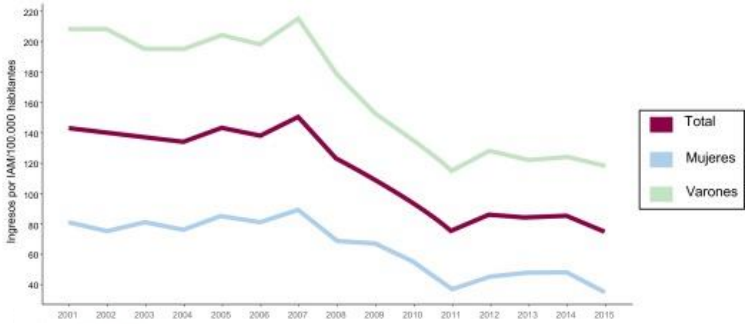
Characteristic	Adenosine, *n* = 63	SNP, *n* = 69	Control, *n* = 65	*P*-value[c]
Time from MI to CMR (h)	49.0 (28.4–75.0)	49.7 (26.2–76.1)	49.0 (38.0–74.8)	0.881
Primary endpoint[d]				
Infarct size (%LVM)	10.1 (4.7–16.2)	10.0 (4.2–15.8)	8.3 (1.9–14.0)	0.133



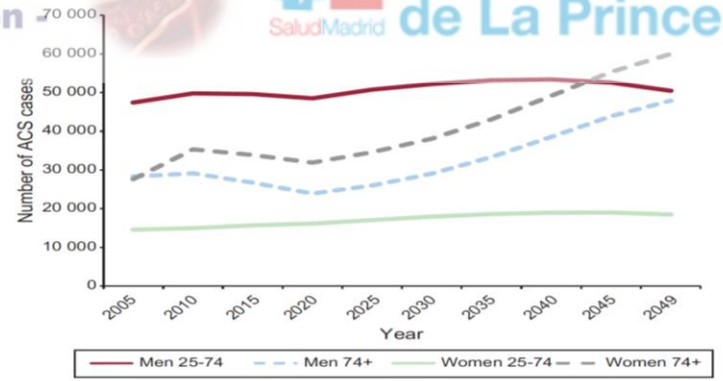
Nazir SA, et al.; EHJ 2016; 37:1910–9.

SCACEST en nuestro medio:

- En los últimos años se ha evidenciado una disminución de los ingresos por SCA.



- No obstante, los modelos muestran que estos se incrementaran en los próximos años debido al envejecimiento de la población.



Dégano IR, et al.; REC 2013

Slow flow / No reflow

Table 3. Predictors of No Reflow Phenomenon Obtained From Univariable and Multivariable Logistic Regression Models

Characteristic	Unadjusted OR (95% CI)	Adjusted OR (95% CI)
Previous MI	1.61 (1.02 to 2.73)	2.17 (1.18 to 3.99)
C-reactive protein (for 1-mg/L increase)	1.02 (1.01 to 1.03)	1.02 (1.01 to 1.04)
Baseline TIMI flow grade (for 1-grade decrease)	1.98 (1.52 to 2.57)	2.02 (1.47 to 2.76)
Initial perfusion defect (for 5% of the LV increase)	1.10 (1.04 to 1.16)	1.07 (1.01 to 1.13)

LV indicates left ventricle; OR, odds ratio.

Ndrepepa G, et al. *Circulation CV Int*. 2010; 3:27-33

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Registro Nacional de Actividad en Cardiología Intervencionista 2019

Intervencionismo en el IAMCEST

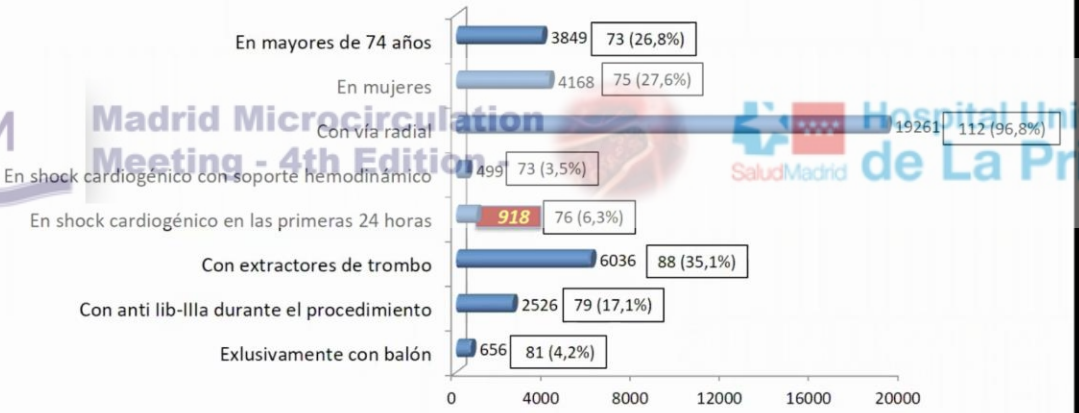
2019

22.529

Dato disponible en 97 centros

	Percentil 25	Mediana	Percentil 75
% de DES	95	99	100

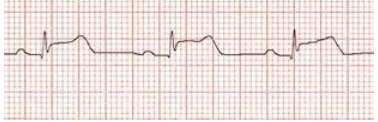
% calculados sobre el total de procedimientos/nº de centros con ese dato (%) sobre procedimientos en estos centros.



- **Éxito angiográfico sin complicaciones, se responde en 75 centros, en estos hay un total de 15180 intervenciones: 14329 (94,4%)**
- **Complicaciones graves: 379. En 75 centros con 15831 intervenciones. (2,4%)**

Generalitat de Catalunya Departament de Salut
Institut Català de la Salut Gerència Territorial Metropolitana Sud
Bellvitge Hospital Universitari
IDIBELL Institut d'Investigació Biomèdica de Bellvitge
UNIVERSITAT DE BARCELONA

Causas muerte intrahospitalaria en el SCACEST:



Hospital Germans Trias i Pujol (Can Ruti)

Table 2. In-Hospital Prognosis and Mortality

	Period 1 1989–1994 (N=1337)	Period 2 1995–1999 (N=960)	Period 3 2000–2004 (N=1059)	Period 4 2005–2009 (N=1535)	Period 5 2010–2017 (N=2698)	*P* for Trend
Angina, %	9.8	10.3	8.9	6.0	2.1	<0.001
Reinfarction, %	1.3	1.5	2.1	1.7	1.7	0.686
Primary VF, %	7.6	6.9	6.9	6.6	6.8	0.114
VT, %	8.7	7.7	4.4	7.8	3.7	<0.001
AV block, %	9.7	12.5	6.2	5.8	5.3	<0.001
AFib/flutter, %	8.4	11.9	8.5	7.6	6.1	<0.001
VS rupture, %	0.7	0.8	0.9	0.6	0.5	0.609
PM rupture, %	0.3	0.5	0.4	0.7	0.3	0.066
FW rupture, %	1.9	2.1	1.4	0.7	0.9	0.002
Pericarditis, %	7.6	3.6	2.8	2.1	2.8	<0.001
RV dysfunction, %	9.6	12.1	6.9	5.1	5.0	<0.001
ACCU LoS, d	5.0	5.5	4.7	3.3	2.4	<0.001
ACCU mortality, %	8.9	8.1	5.8	3.8	4.2	<0.001
Anterior wall AMI	11.2	11.0	6.1	4.3	4.7	<0.001
Inferior wall AMI	8.9	8.1	5.8	3.4	3.8	<0.001
28-d mortality, %	10.4	9.9	7.3	5.1	6.0	<0.001
1-y mortality, %	11.7	13.4	10.5	8.7	9.0	<0.001

ACCU indicates acute cardiovascular care unit; AFib, atrial fibrillation; AV, atrioventricular; FW, free-wall; LoS, length of stay; PM, papillary muscle; RV, right ventricle; VF, ventricular fibrillation; VS, ventricular septum; and VT, sustained ventricular tachycardia.

FV y reinfarto

Arritmias (no FV)

Complicaciones mecánicas

Insuficiencia cardíaca/Shock

García-García C, et al.; JAHA 2020; 9:e017159

Causas muerte intrahospitalaria en el SCACEST:

Hospital Germans Trias i Pujol (Can Ruti)

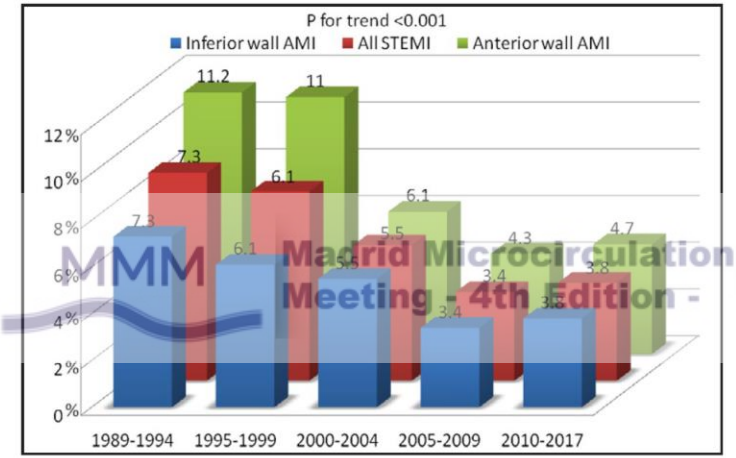


Figure 3. Trends in changes in 28-day case fatality related to infarct location between periods.
Inferior wall AMI (blue), anterior wall AMI (green), all STEMI (red). AMI indicates acute myocardial infarction; and STEMI, ST-segment–elevation myocardial infarction.

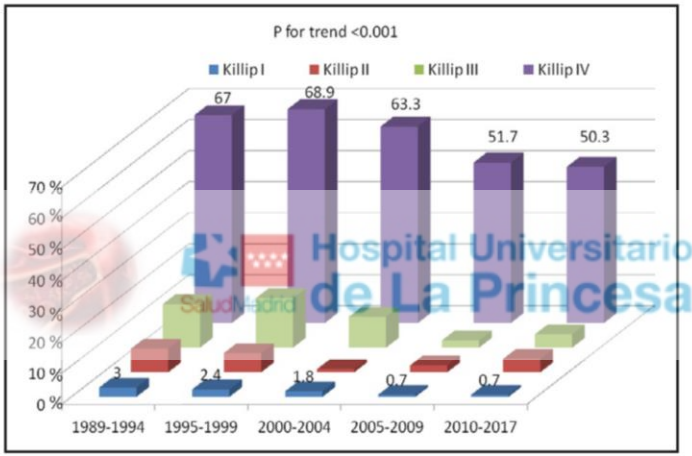


Figure 6. Early acute-phase mortality relative to maximum Killip–Kimball class during intensive cardiac care unit admission.
Period 1 (dark blue), period 2 (red), period 3 (green), period 4 (violet) and period 5 (light blue).

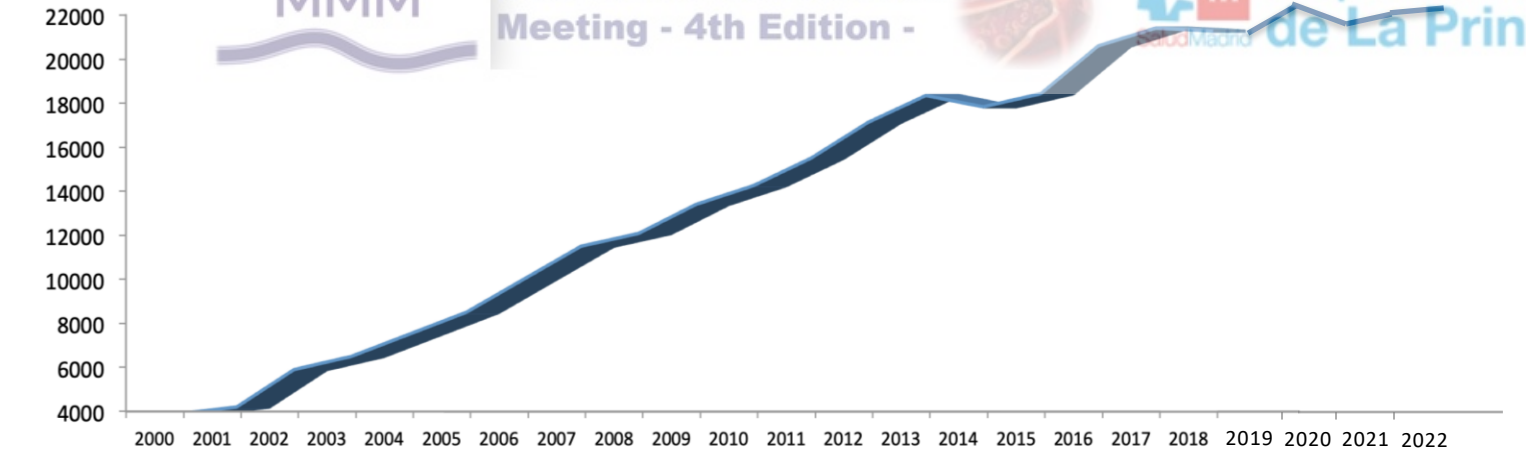
García-García C, et al.; JAHA 2020; 9:e017159

STEMI in Spain

- Around **30%** of Percutaneous Coronary Interventions (PCIs) are performed as emergent **Primary-PCI** in STEMI patients.

Number of Primary-PCIs

2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022
13.395	14.248	15.491	17.125	18.337	17.825	18.418	20.588	21.395	21.261	22.529	21.039	21.993	22.163



Edited from the Registry of the Interventional Cardiology Association; Spanish Journal of Cardiology