



# Quand le flux TIMI reste à 0 ...

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# Admission SAU

- homme 67 ans
- Précordialgies transitoires évolutives depuis 24h
- FdRRCV :
  - TABAC actif à 100 PA
  - HTA
  - Dyslipidémie
  - Hérité coronaire (IDM père 30 ans)
- test ischémique (dépistage) il y a 3 mois maximal négatif

# ECG per critique



# ETT



- VG non dilaté non hypertrophié, hypokinésie inférieure, FEVG 50%
- Pas de valvulopathie mitroaortique
- PRVg basses
- Bon VD non dilaté, absence d'argument pour HTP
- Absence d'épanchement péricardique

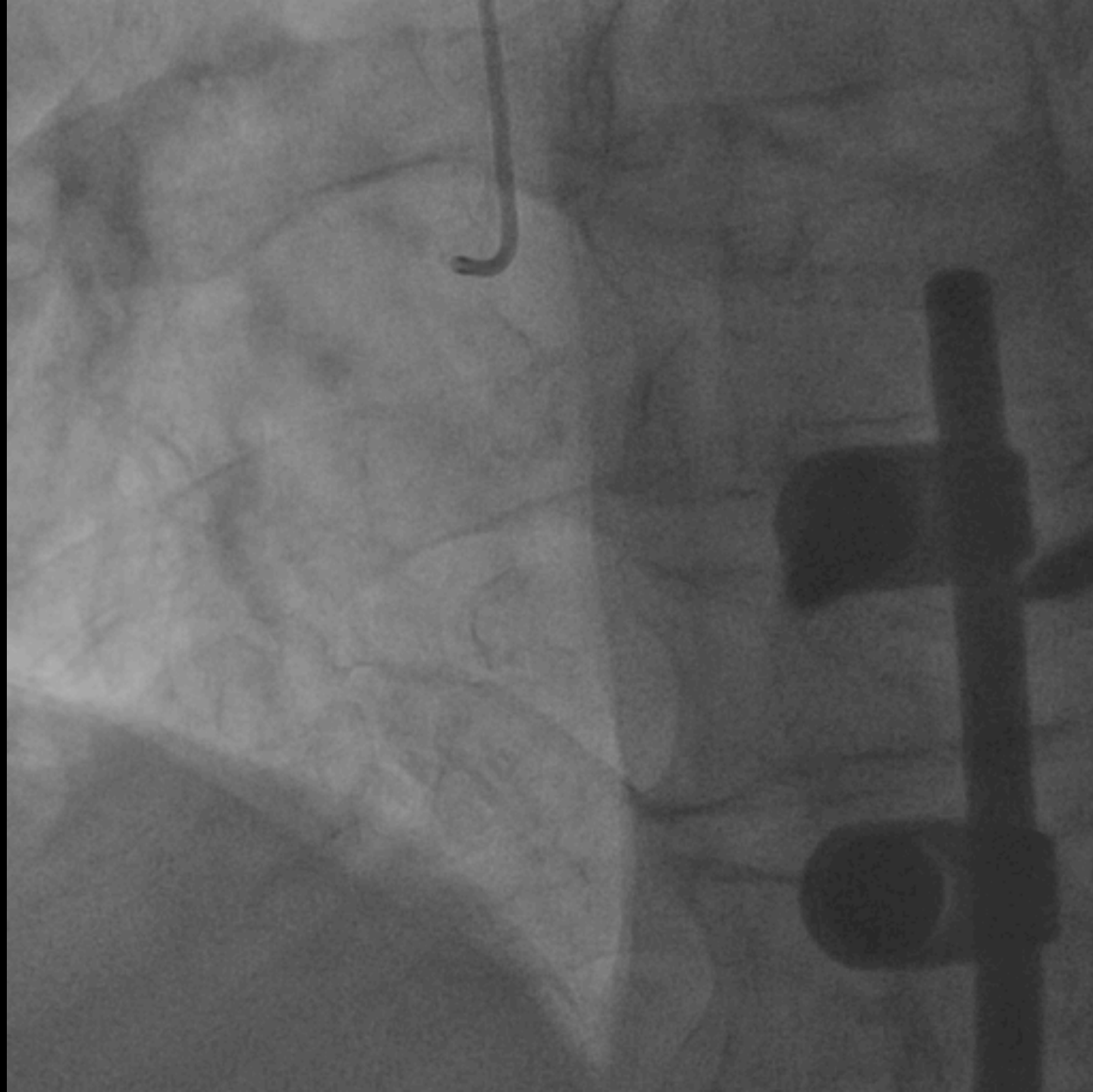
# Bio

- Troponine 27,3  $\mu\text{g/L}$  (N< 0,05)

# Hospitalisation UDT



- Récidive douloureuse dans la nuit résolutive sous Risordan IVSE
  - Récidive douloureuse sous traitement médical le lendemain matin
- => Coronarographie en urgence**









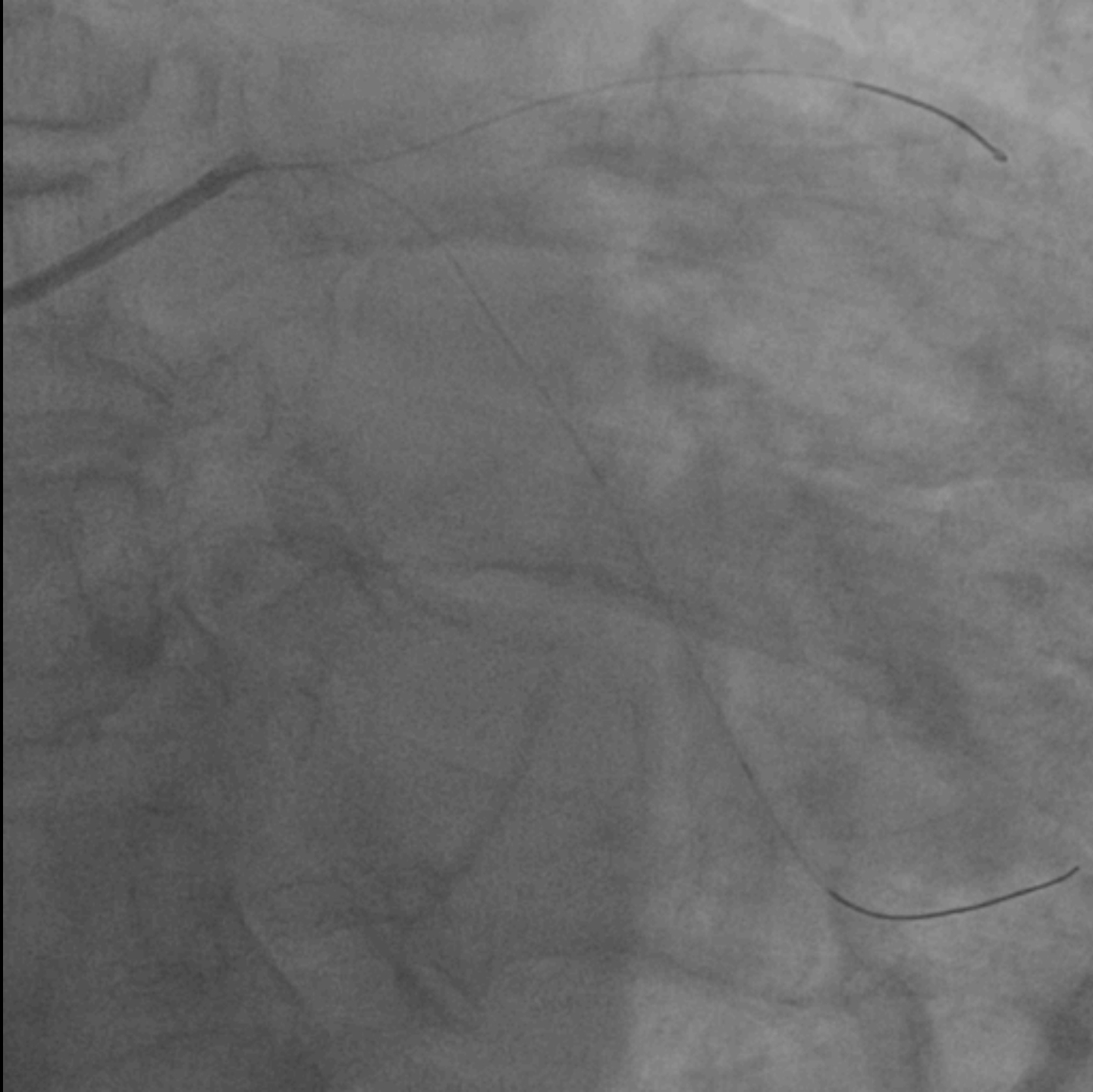
# Angioplastie Cx

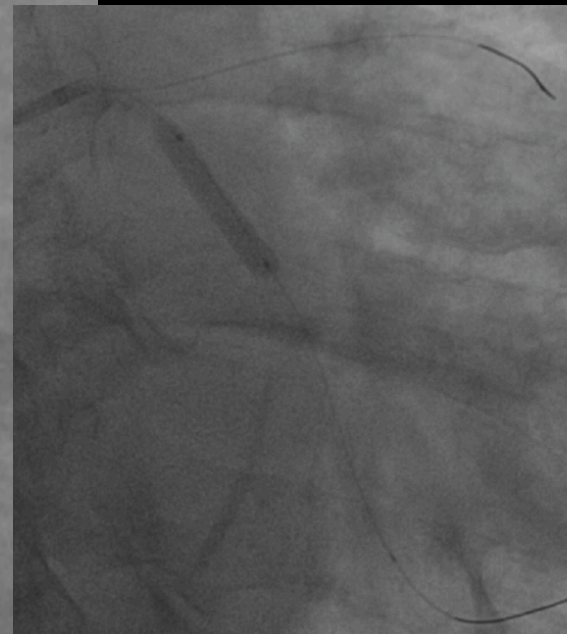
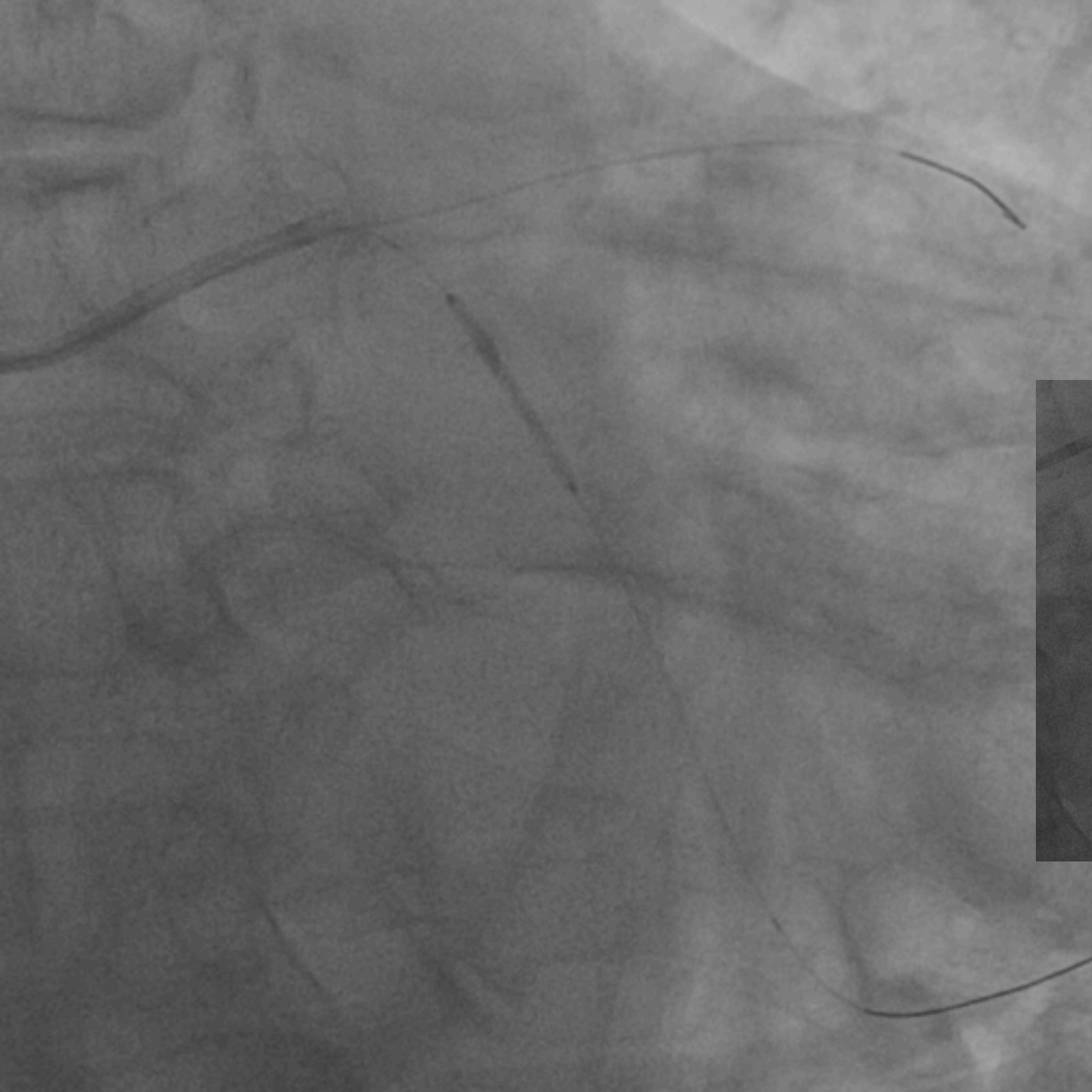
- Récurrence douloureuse
- Douleur évolutives depuis 48h + occlusion d'allure thrombotique  
=> GpIIb/IIIa (Agrasta IV)



# EBU 3.5 6F – Pilot 50 Thrombo-aspiration

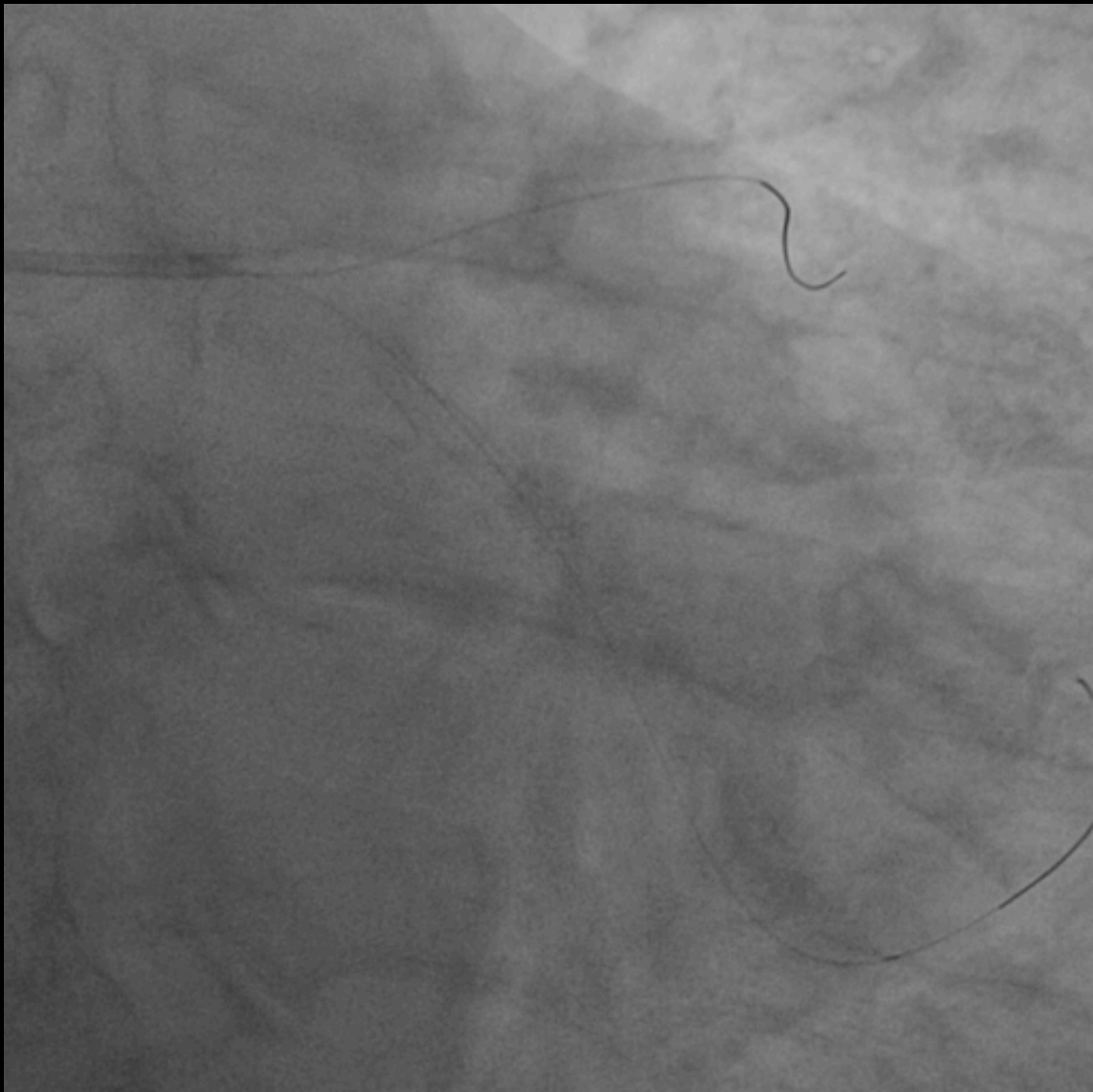












# Collapsus



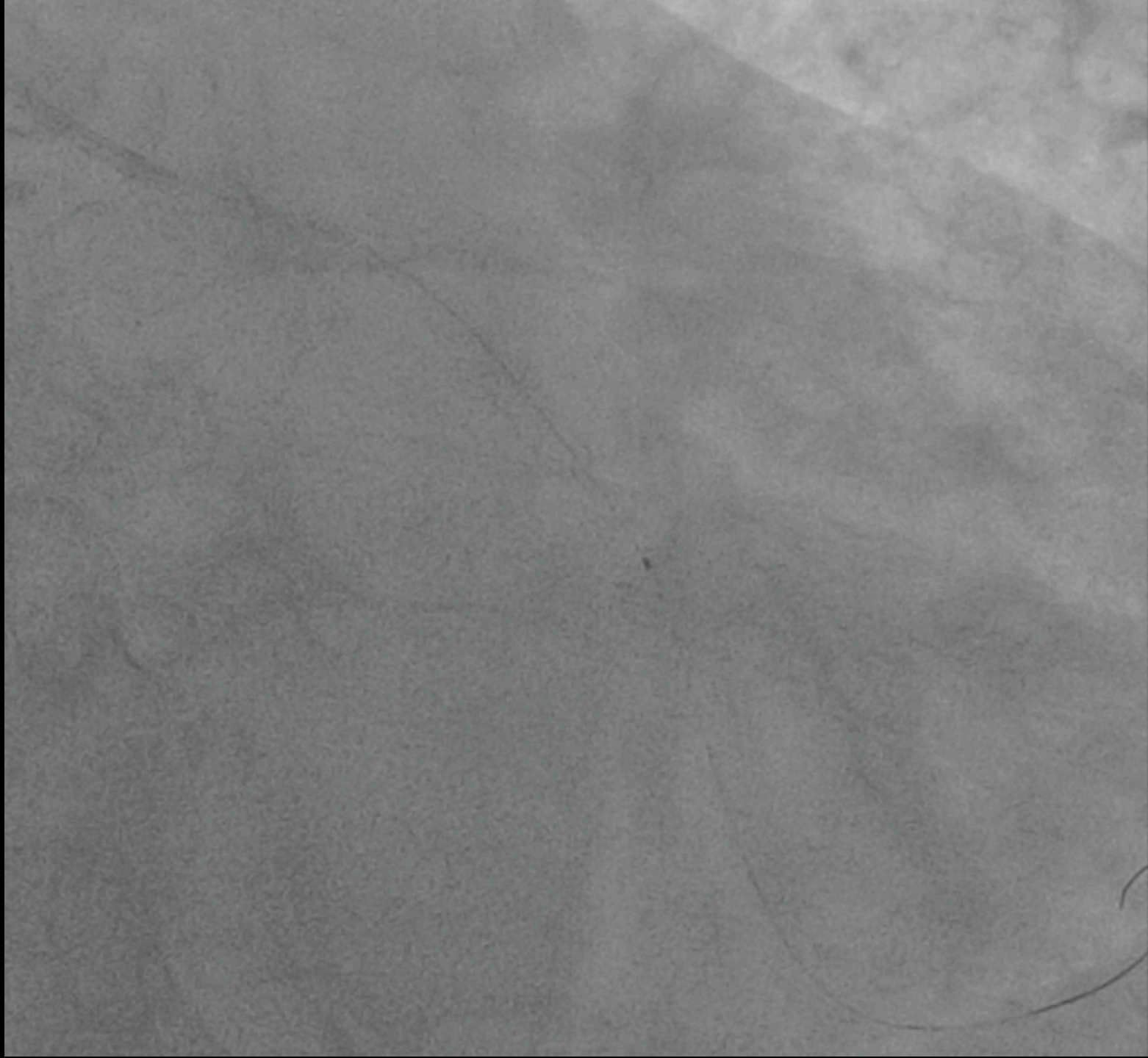
- 1cc d'éphédrine x 3
- Remplissage 1,5L NaCl

=> restauration PAM > 65 mmHg et PAS > 100mmHg

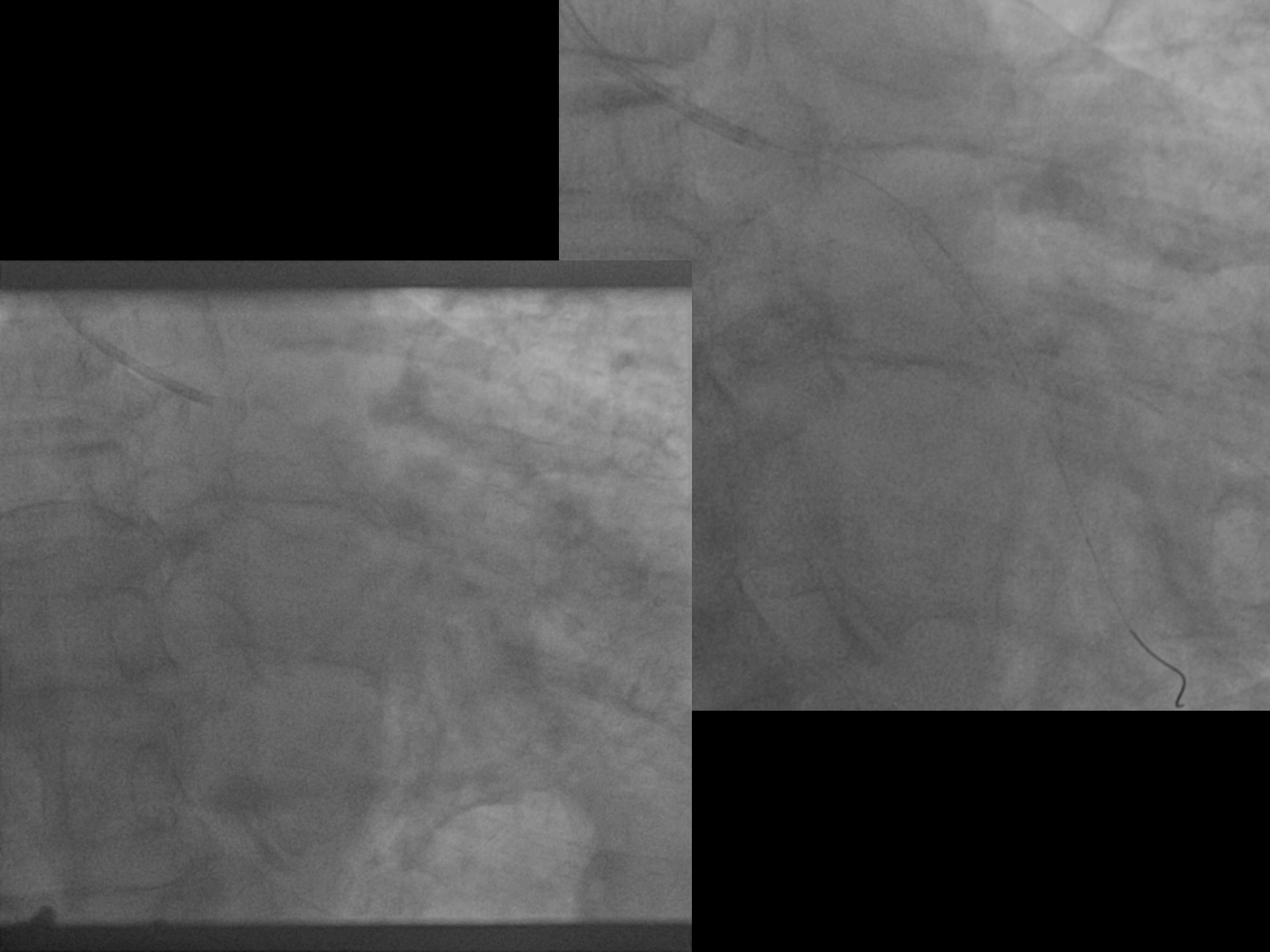












# 'No Reflow'

## Definition:

Inadequate myocardial perfusion through a given segment of the coronary circulation without angiographic evidence mechanical vessel obstruction

## Incidence according to classification

**Table 1. Classification of the no-reflow phenomenon.**

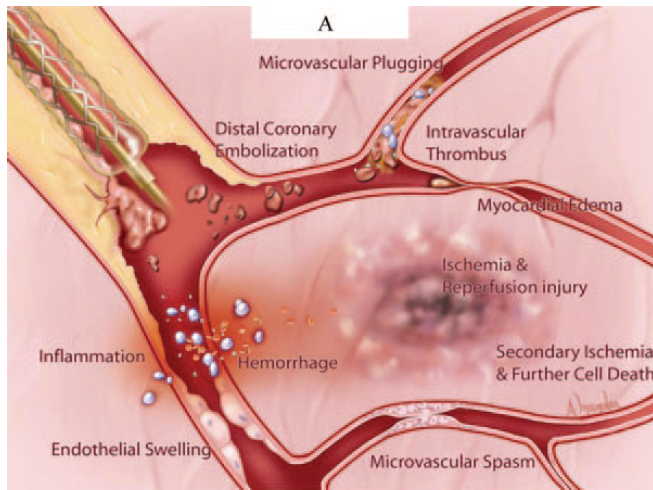
1. Myocardial infarction no-reflow	<b>25%</b>
2. Interventional no-reflow	
2a. Native coronary PCI no-reflow	<b>&lt;2%</b>
2b. SVG PCI no-reflow	<b>20%</b>
3. Experimental no-reflow	

Rates of cardiac death and nonfatal cardiac events are increased in patients with compared to those without no-reflow

# 'No Reflow': Mechanisms



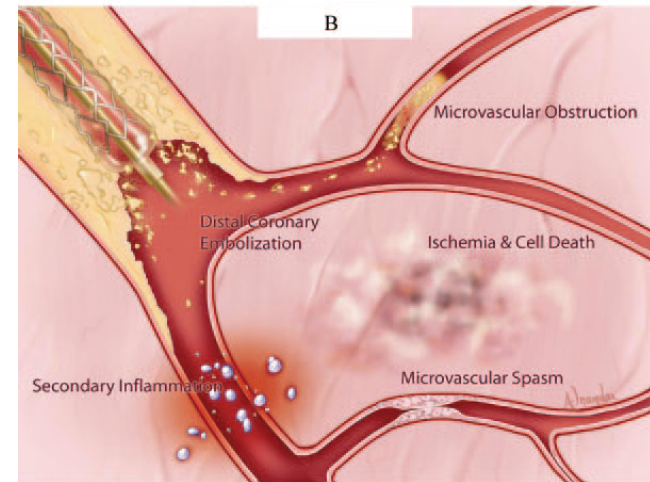
## STEMI: Reperfusion No Reflow



**Distal embolisation**  
**Microvascular spasm**  
**Inflammation**  
**Ischaemic-reperfusion cell injury**

**Endothelial Oedema** Muller, Windecker, Cuisset et al, Eurointervention 2008

## PCI: Interventional No Reflow



**Distal embolisation**  
**Microvascular spasm**  
**Inflammation**

# 'No Reflow': Predictors

## Management of two major complications in the cardiac catheterisation laboratory: the no-reflow phenomenon and coronary perforations

Olivier Muller<sup>1</sup>, MD; Stephan Windecker<sup>2</sup>, MD; Thomas Cuisset<sup>3</sup>, MD; Jean Fajadet<sup>4</sup>, MD; Michael Mason<sup>5</sup>, MD; Andrea Zuffi<sup>6</sup>, MD; Alexander Doganov<sup>7</sup>, MD; Eric Eeckhout<sup>6</sup>, MDPH FESC

### Predictors of no-reflow.

- Duration of preceding myocardial ischaemia
- Infarct size
- Procedural variables
  - ◇ Rotational atherectomy
- Vessels
  - ◇ Saphenous vein grafts
- Lesion characteristics
  - ◇ Plaque size
  - ◇ Eccentric or fissured plaque
- Patient characteristics
  - ◇ Diabetes mellitus
  - ◇ Absence of pre-infarction angina
  - ◇ Advanced age

**Late Presenters 'STEMI'**

**Rotablator**

**SVG**



# **‘No Reflow’: Prevention and Treatment**

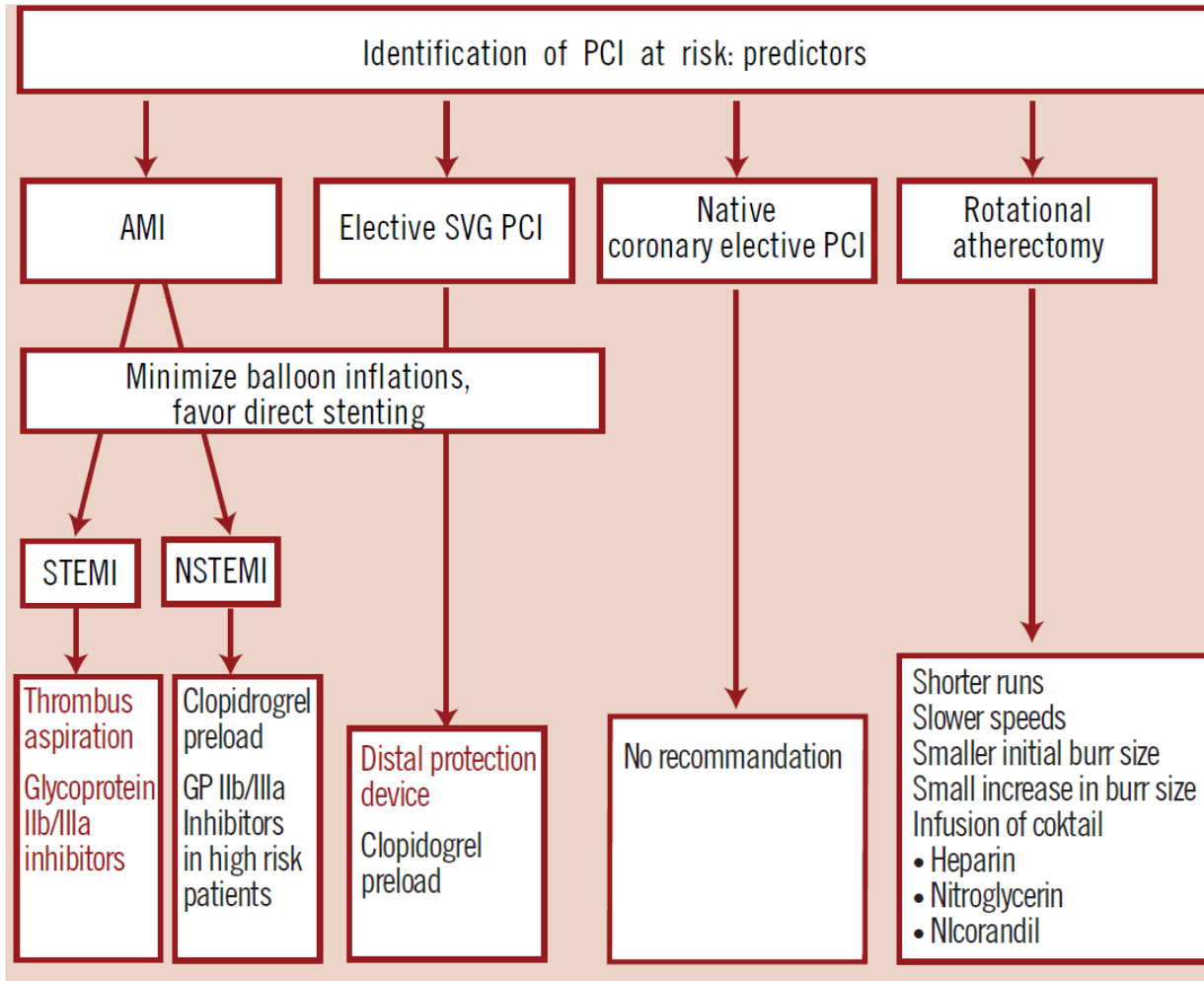


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**Prevention of ‘no reflow’ is much easier than  
Treatment**

# 'No Reflow': Prevention



Techniques  
Anti-thrombotics

# Evidence is lacking for optimal treatment



Guidelines Myocardial revascularisation  
Wijns et al, EHJ 2010

Guidelines STEMI  
Steg et al, EHJ 2012

## 2017 ESC Guidelines

STEMI  
Ibanez et al, EHJ 2017

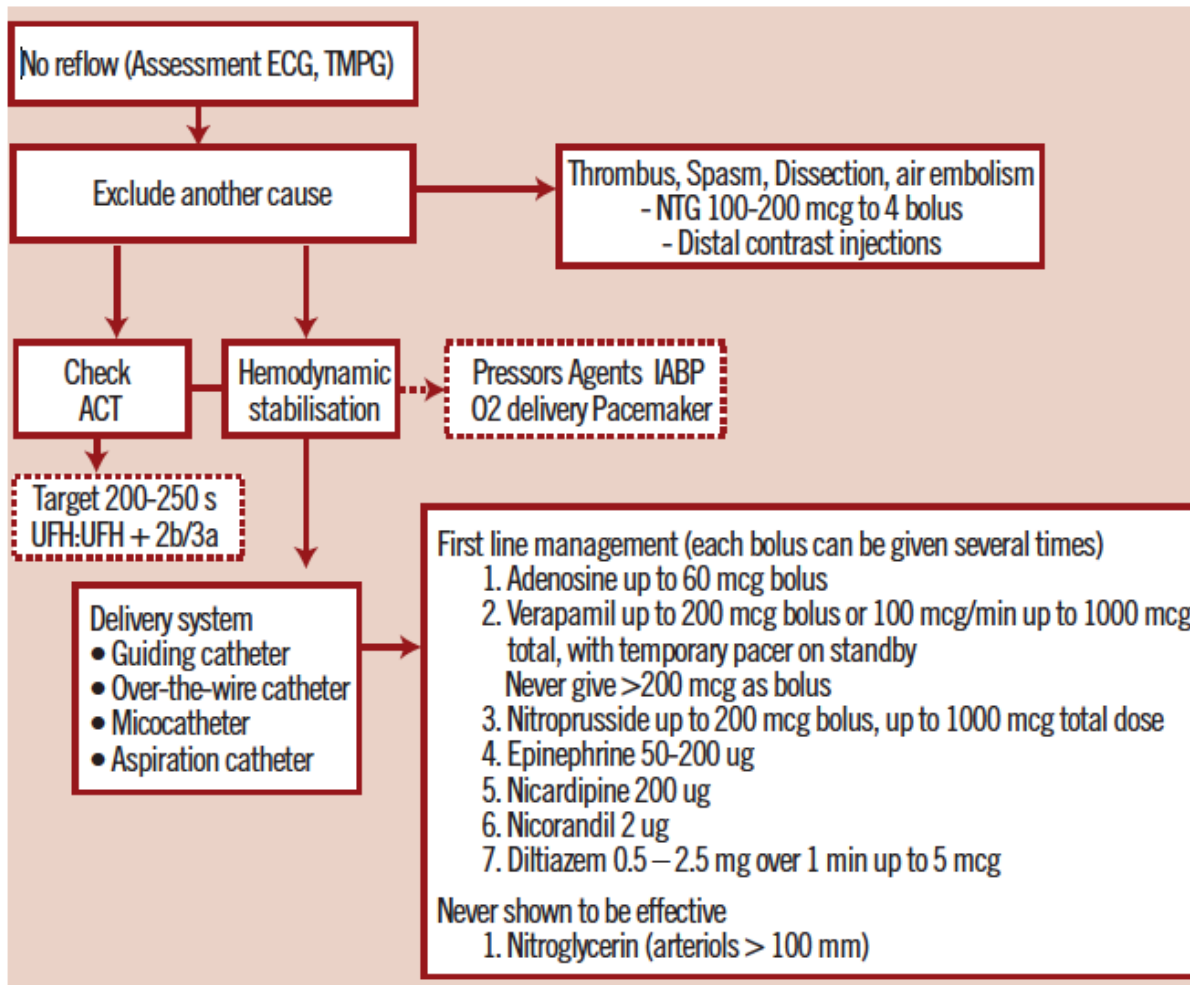
tile function. Intracoronary administration of vasodilators such as adenosine, verapamil, nicorandil, papaverine, and nitroprusside during and after primary PCI improves flow in the infarct-related coronary artery and myocardial perfusion and/or reduces infarct size, but large RCTs are lacking.<sup>55</sup> High-dose i.v. adenosine infusion

There have been many attempts to treat no-reflow using intracoronary vasodilators, i.v. infusion of adenosine or abciximab, but there is no definitive proof that these therapies affect clinical outcomes

lirudin.<sup>197</sup> Using GP IIb/IIIa inhibitors as bailout therapy in the event of angiographic evidence of a large thrombus, slow- or no-reflow, and other thrombotic complications is reasonable, although this strategy has not been tested in a randomized trial. Overall, there is no evi-

GP IIb/IIIa inhibitors should be considered for bailout if there is evidence of no-reflow or a thrombotic complication.	<b>IIa</b>	<b>C</b>
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# 'No Reflow': Treatment



Muller, Windecker, Cuisset et al, Eurointervention 2008

Figure 2. Algorithm for the treatment of no-reflow. The use of a microcatheter should be strongly considered for diagnosis and treatment.

# Take Home message

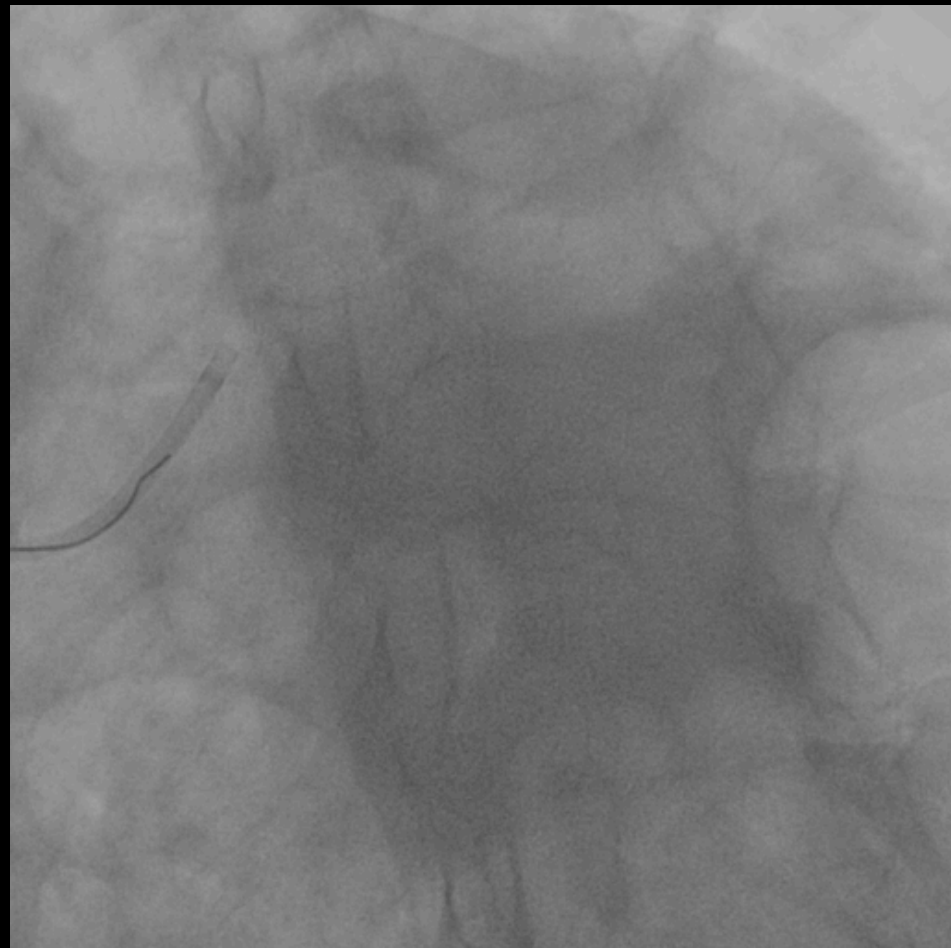
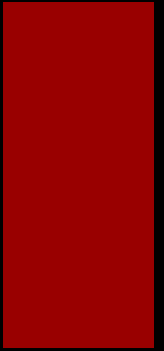


- No reflow
  - pas si rare
  - peu prévisible MAIS situations à risque
    - STEMI tardif-étendu / ACT ponts veineux / rotablator
  - impact pronostic clinique
- Mécanisme multifactoriel
- **Prévention +++**
- Traitement (algorithme) :
  - Restauration hémodynamique
  - Vasodilatateur intra coronaire (Adénosine)
    - Utilisation micro-cathéters

A 3 semaines



# Revascularisation du TCG



Merci de votre attention

