Contemporary Management Strategy in STEMI Patients Complicating Cardiogenic Shock

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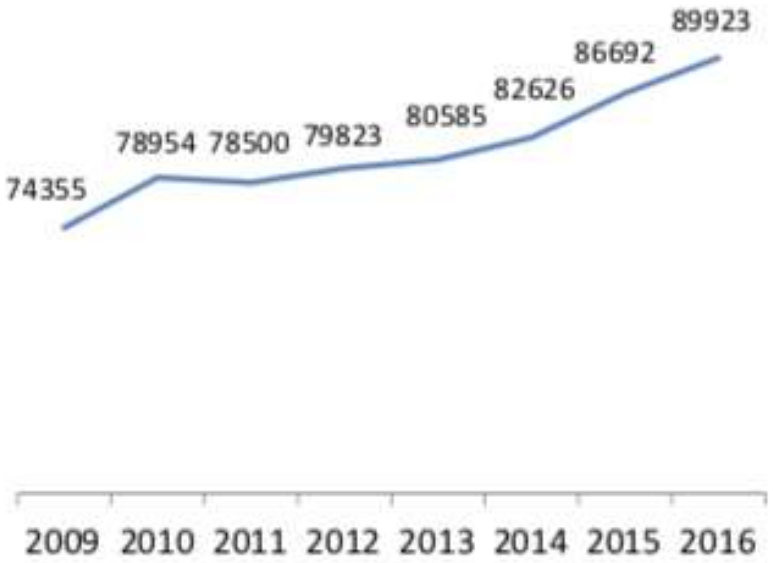
SHOCK trial: Definition of Cardiogenic Shock

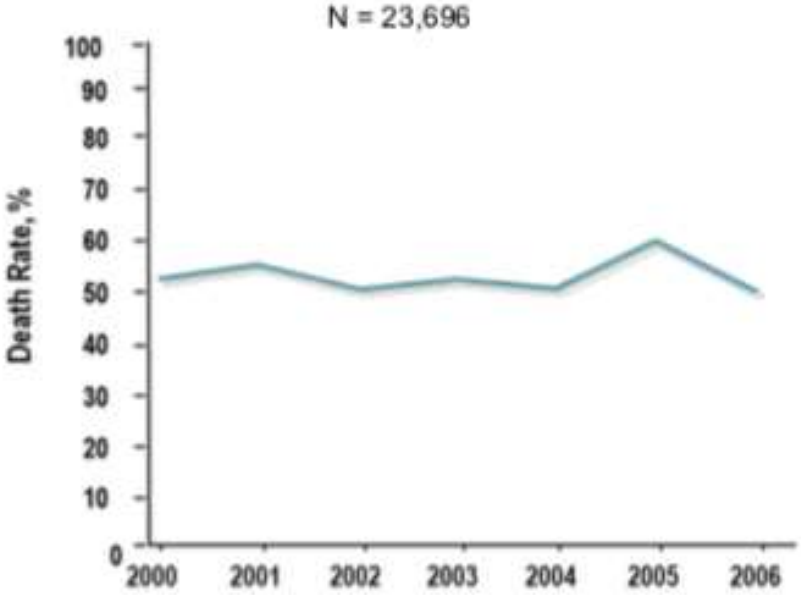
Clinical
SBP <90 mm Hg for 30 min
Supportive measures needed to maintain SBP >90 mm Hg
End-organ hypoperfusion
Cool extremities
UOP <30 ml/h
HR >60 beats/min
Hemodynamic
Cardiac index <2.2 ml/min/m^2
PCWP >15 mm Hg

AMI Shock Mortality Unchanged in > 20 years

US AMI/CGS cases per year[1,2]



High In-Hospital Mortality During AMI Cardiogenic Shock[3]



1. *Sandhu A, McCoy I, Negi S, et al. Use of Mechanical Circulatory Support in Patients Undergoing Percutaneous Coronary Intervention; Insights from the National Cardiovascular Data Registry. Circulation, 2015;132:1243-1251*
2. *Acute Cardiac Assist Report, Health Research International – August 2015*
3. *Jeger, et al. Ann Intern Med. 2008*

AMI Shock Frequency and Mortality

(2010 -2016, Fuwai Hospital)

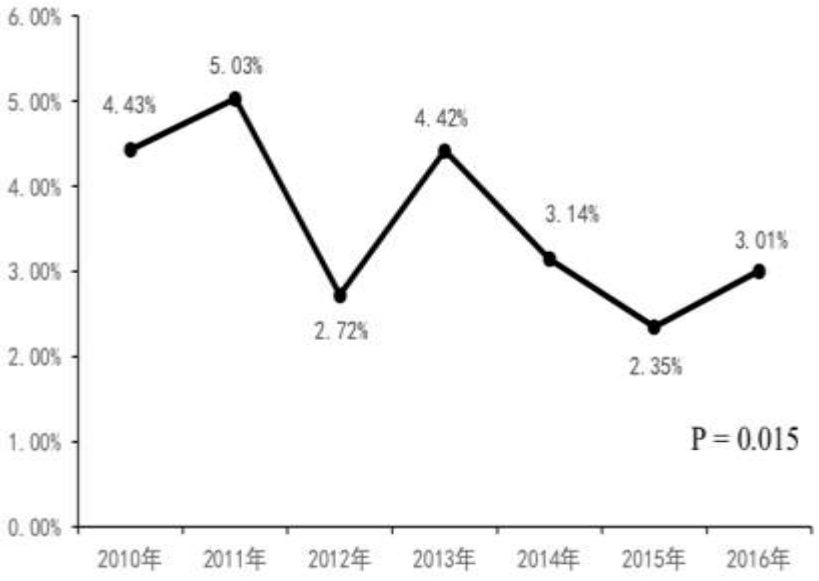


图 1. 急性心肌梗死合并心源性休克发生率的变化趋势

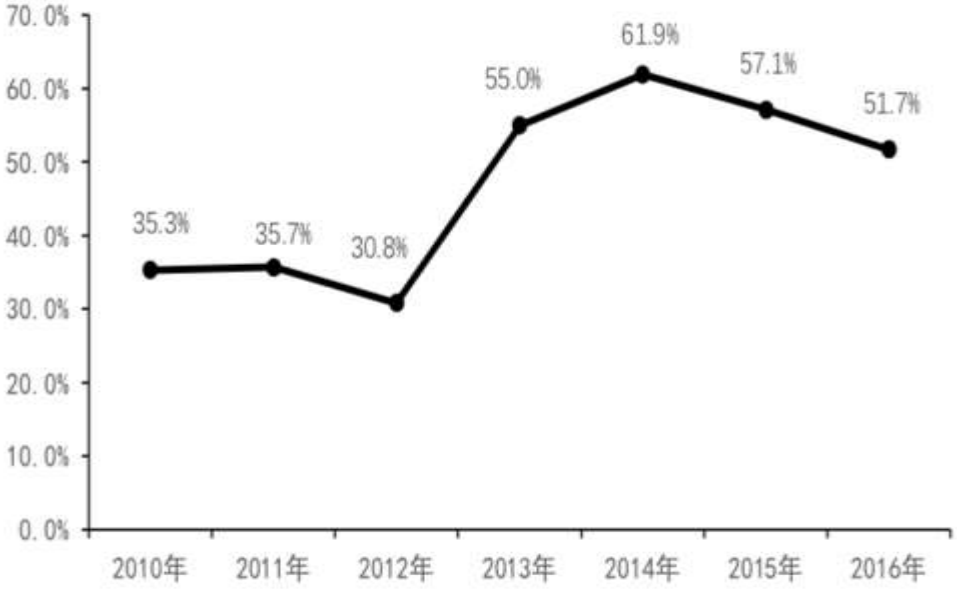


图 2. 急性心肌梗死合并心源性休克住院死亡率的变化趋势

Data from 4,400 AMI Pts

2017 ESC STEMI Guidelines

Procedural aspects of the primary percutaneous coronary intervention strategy

Recommendations	Class	Level
Non-IRA strategy		
Routine revascularization of non-IRA lesions should be considered in STEMI patients with multivessel disease before hospital discharge.	IIa	A
Non-IRA PCI during the index procedure should be considered in patients with cardiogenic shock.	IIa	C

Multivessel PCI in STEMI Patients With Cardiogenic Shock

KAMIR-NIH registry: 659 pts who underwent multivessel PCI (39.5%) or infarct-related artery (IRA)-only PCI (60.5%), Nov 2011-Dec 2015.

1-Year Outcomes	Multivessel PCI	IRA-Only PCI	Adjusted HR (95% CI)
All-Cause Death	21.3%	31.7%	0.52 (0.38-0.73)
Non-IRA Repeat Revascularization	6.7%	8.2%	0.33 (0.14-0.78)

No differences in new requirement for renal replacement therapy by 30 days between the two groups, with an overall rate of 3.3%.

Conclusion: Patients with STEMI and cardiogenic shock who undergo multivessel PCI stand to derive improved 1-year outcomes.

Lee JM, et al. J Am Coll Cardiol. 2018;71:844-856.

CULPRIT-SHOCK: A Randomized Trial of Multivessel PCI in Cardiogenic Shock

Holger Thiele, MD

on behalf of the CULPRIT-SHOCK Investigators

UNIVERSITÄT LEIPZIG HERZZENTRUM

Conclusions

- In patients with multivessel coronary artery disease and cardiogenic shock complicating acute myocardial infarction culprit lesion only PCI with possible staged revascularization reduced the composite of mortality or requirement for renal replacement therapy at 30 days.
- This effect in the primary outcome was mainly driven by a 30-day mortality reduction.
- This largest randomized European multicenter trial in cardiogenic shock complicating myocardial infarction challenges current guideline recommendations.

tct2017 Cardiovascular Research Foundation

Dr Grines: Culprit Shock Questions

- Severity of illness?
  - Pressors >90%, Mechanical Ventilation in 82%, Resuscitation in 53% suggest patients are very sick
  - Lactate normal in 30%, median systolic BP of 100 and HR of 90 suggest that not all were in shock
- No data on invasive hemodynamics, type and dose of vasopressors or inotropic drugs
- Limited use of hemodynamic support
  - When used was it placed pre- PCI?
  - Would multivessel PCI results have been better if support used?
- Should multivessel PCI have been staged?

Culprit Shock: No Difference in Cardiac Causes of Death

Cause	Culprit only	Multivessel
Sudden death	11 (7.4%)	12 (6.8%)
Recurrent MI	2 (1.3%)	2 (1.1%)
Refractory Shock	104 (69.8%)	108 (61.4%)

Multivessel PCI did not worsen cardiac outcomes

Culprit Shock
Non-Cardiac Causes of Death

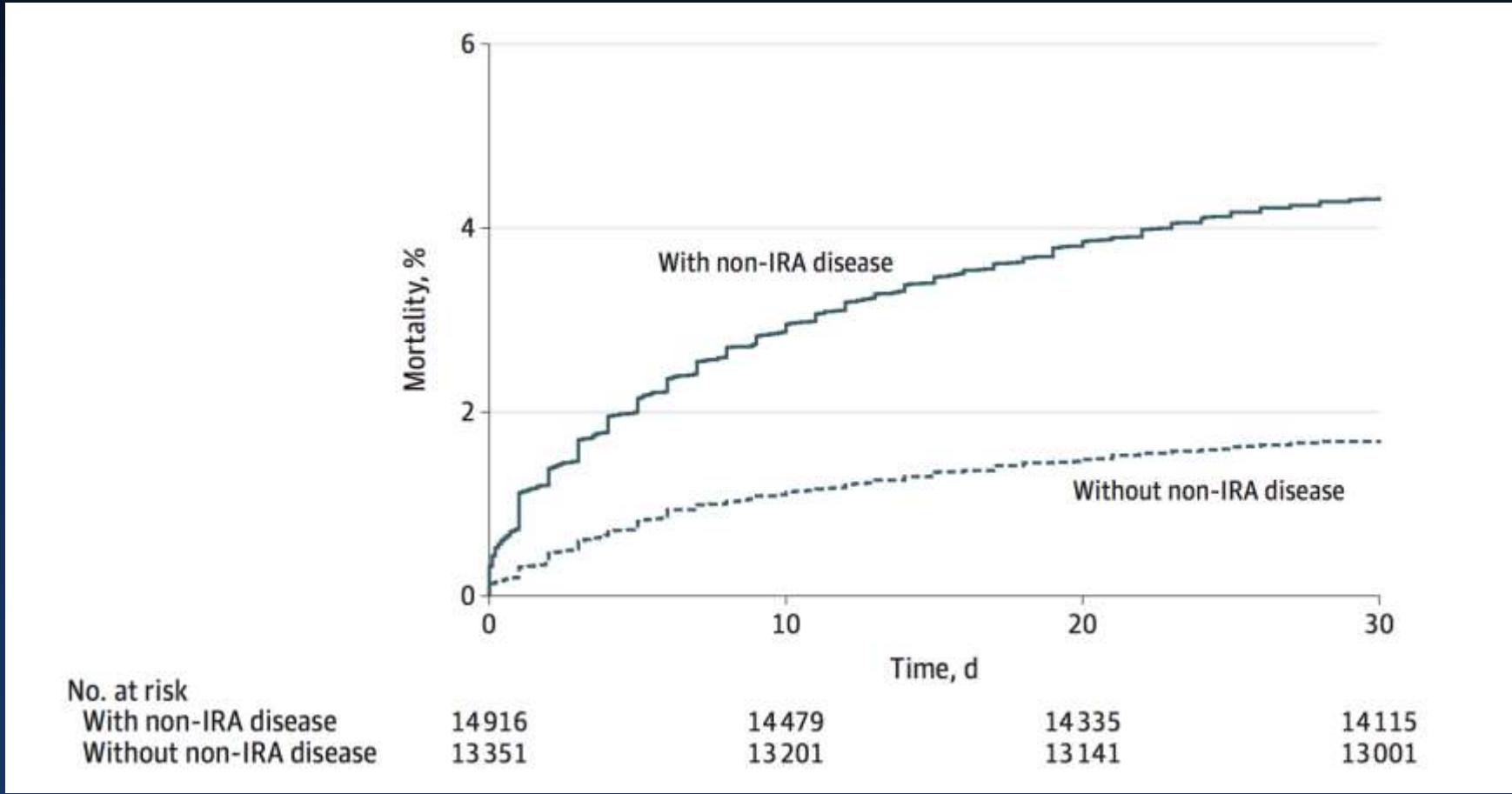
Cause	Culprit only	Multivessel
Brain Injury	11 (7.4%)	25 (14.2%)
Unknown	2 (1.3%)	4 (5.1%)
Other	9 (6%)	12 (6.8%)

Should Cardiac Arrest Patients been Excluded?

Post-mortem study of Shock hearts

- At least 40% of the myocardium infarcted in the aggregate (old and new injury)
- 80% have significant LAD disease
- 2/3 have severe 3 vessels lesions

52.8%合并多支病变，30天死亡率增加58%



Park D-W, et al. JAMA. 2014;312:2019-2027.

4907例 AMI-PCI患者

(2010-2016， FWH)

	STEMI（%）	NSTE-ACS（%）	卡方检验	
			χ2 值	P 值
单支	936（25.1%）	234（19.7%）	13.44	＜0.01
双支	1097（29.5%）	298（25.1%）	10.01	＜0.01
三支	1595（42.8%）	568（47.8%）	6.67	＜0.05
左主干	291（7.8%）	170（14.3%）	40.30	＜0.01
合计	3725（100%）	1189（100%）	-	-

- More NSTEMI patients:
  - Older yrs
  - Women (30.1%),
  - Hypertension (69.1%)
  - Previous PCI (25.8%) or CABG (3%)
  - Left main (14%) or MVD (47.8%)

Pathophysiology of Shock

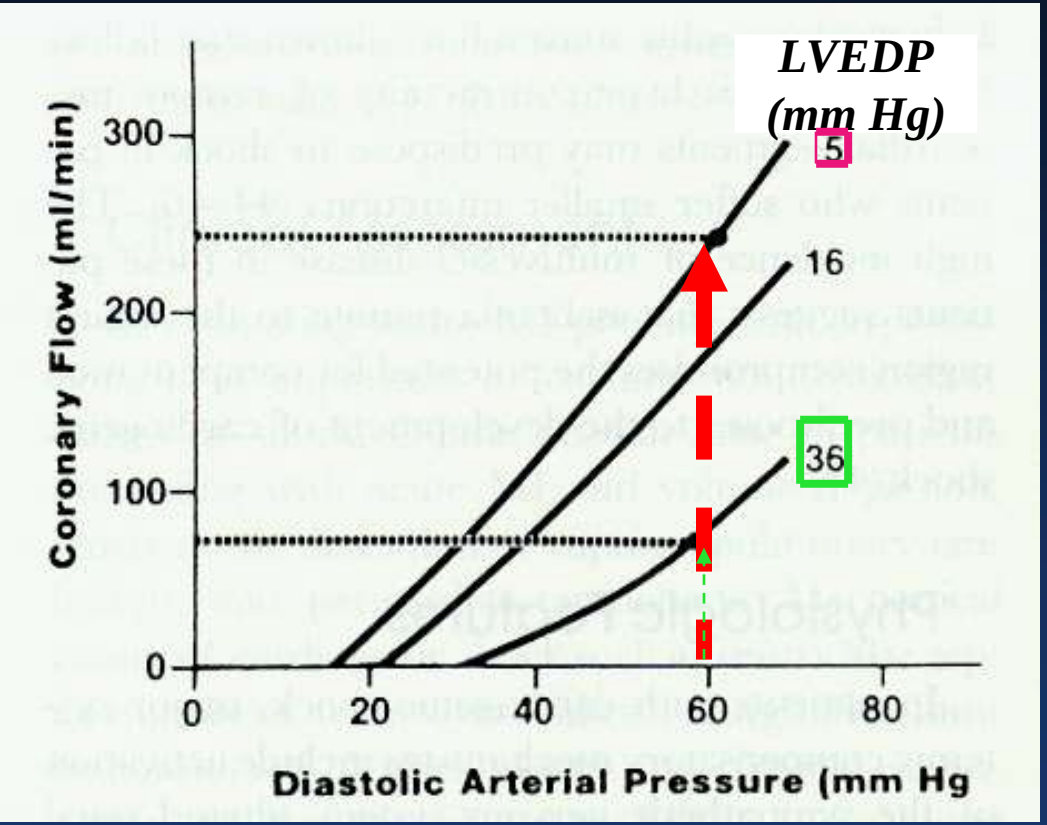
- Effect of Hypotension
  - Flow in normal coronary:
    - Regulated by microvascular resistance
    - Coronary flow may be preserved at AO pressures as low as 50 mm Hg
  - In coronary vessel with critical stenosis:
    - Vasodilator reserve of microvascular bed is exhausted
    - Decrease in AO pressure => Coronary hypoperfusion

Pathophysiology of Shock

Effect of:

Elevated LVEDP

on coronary flow



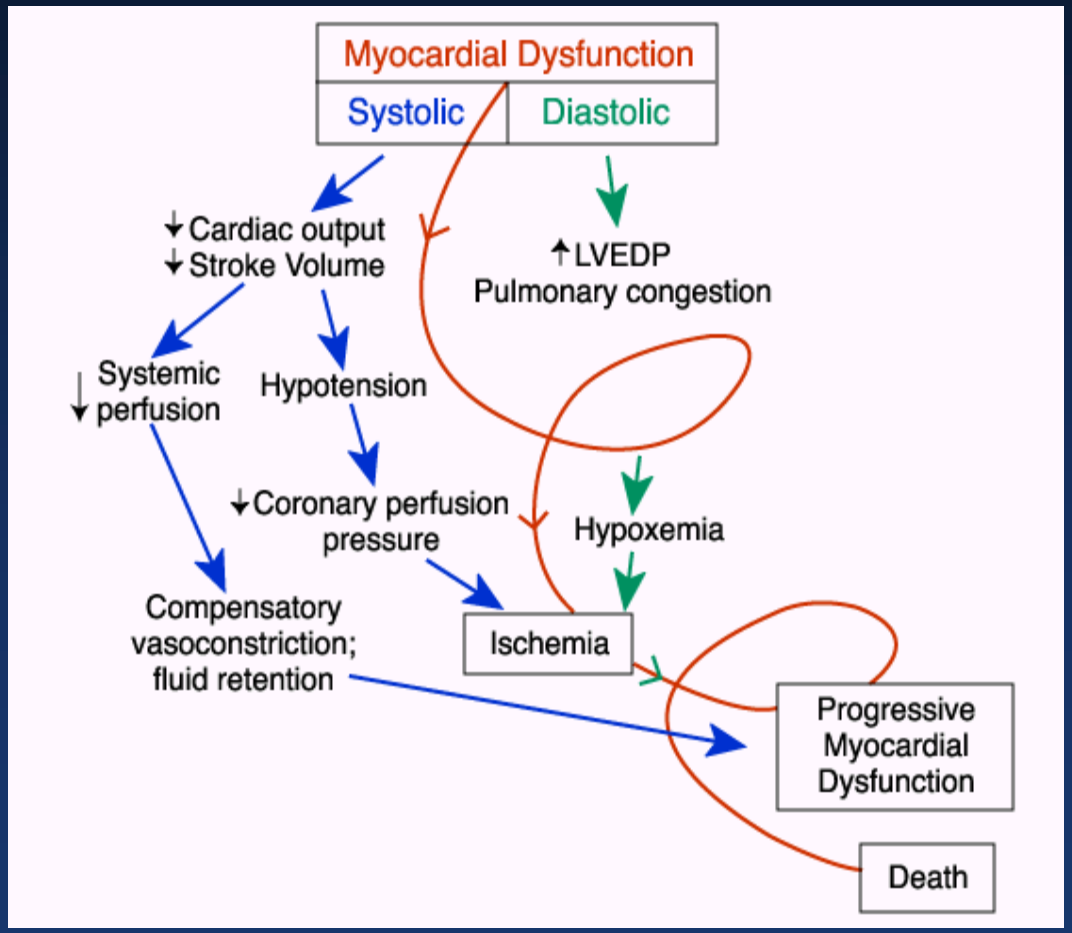
Pathophysiology of Shock

Hypotension + LVEDP ↑ and critical stenosis

→ Myocardial Hypoperfusion →LV dysfunction → Systemic lactic acidosis → Impairment of non-ischemic myocardium → worsening hypotension.

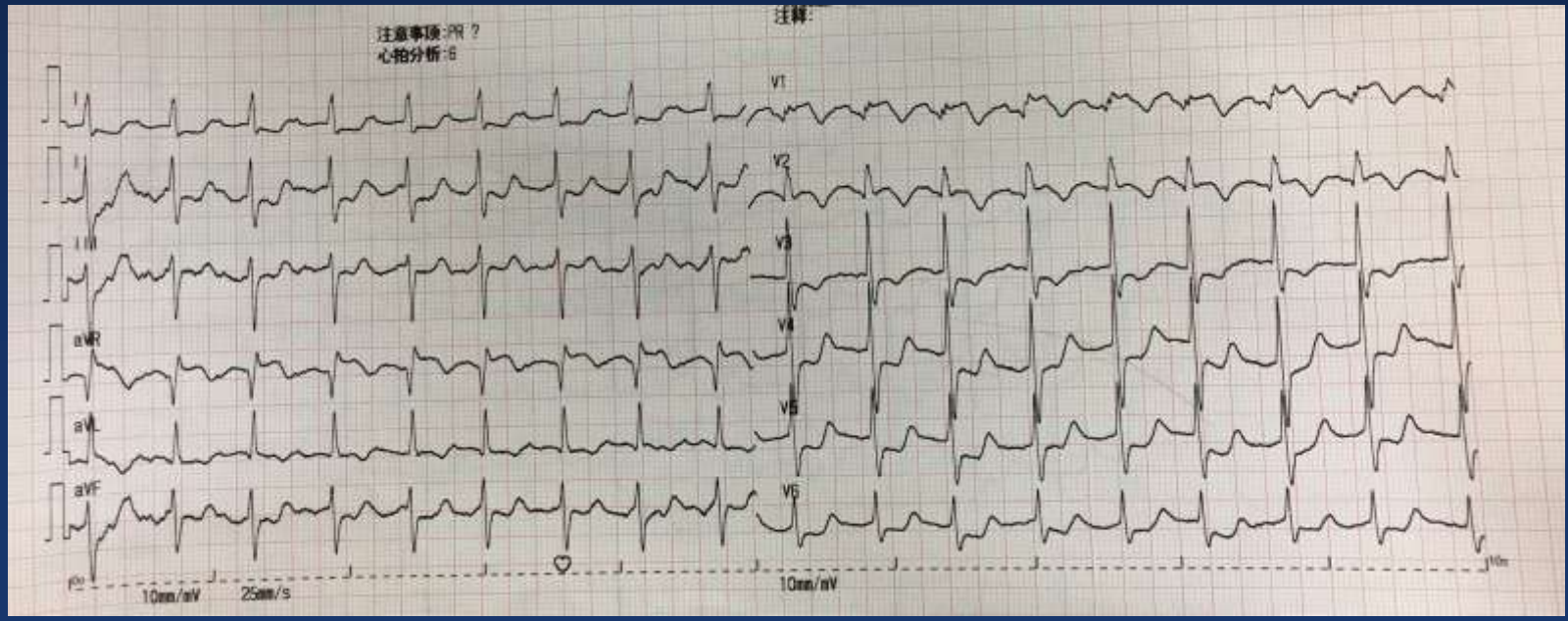
Relieving severe coronary artery stenosis is the basis for improving patients with cardiogenic shock !

- ✓ *LVEDP elevation*
- ✓ *Hypotension*
- ✓ *Decreased coronary*
- ✓ *perfusion*
- ✓ *Ischemia*
- ✓ *Further myocardial*
- ✓ *dysfunction*
- ✓ *Neurohormonal*
- ✓ *activation →*
- ✓ *Vasoconstriction*
- ✓ *Endorgan hypoperfusion*

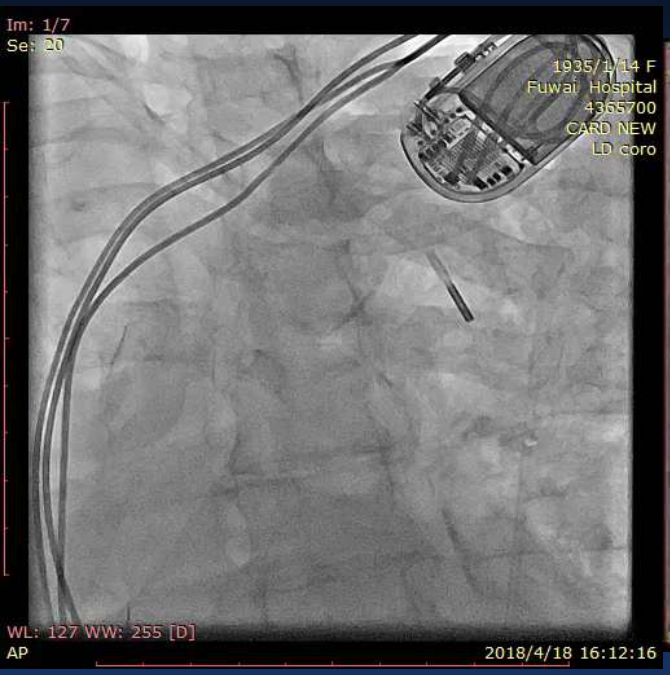


Case 1

- 女，84岁
- AMI1d
- cTnI：2 ng/ml
- LV 50mm，EF25%
- 高血压、高脂血症
- 既往多次心梗病史，多次PCI



IABP辅助后造影

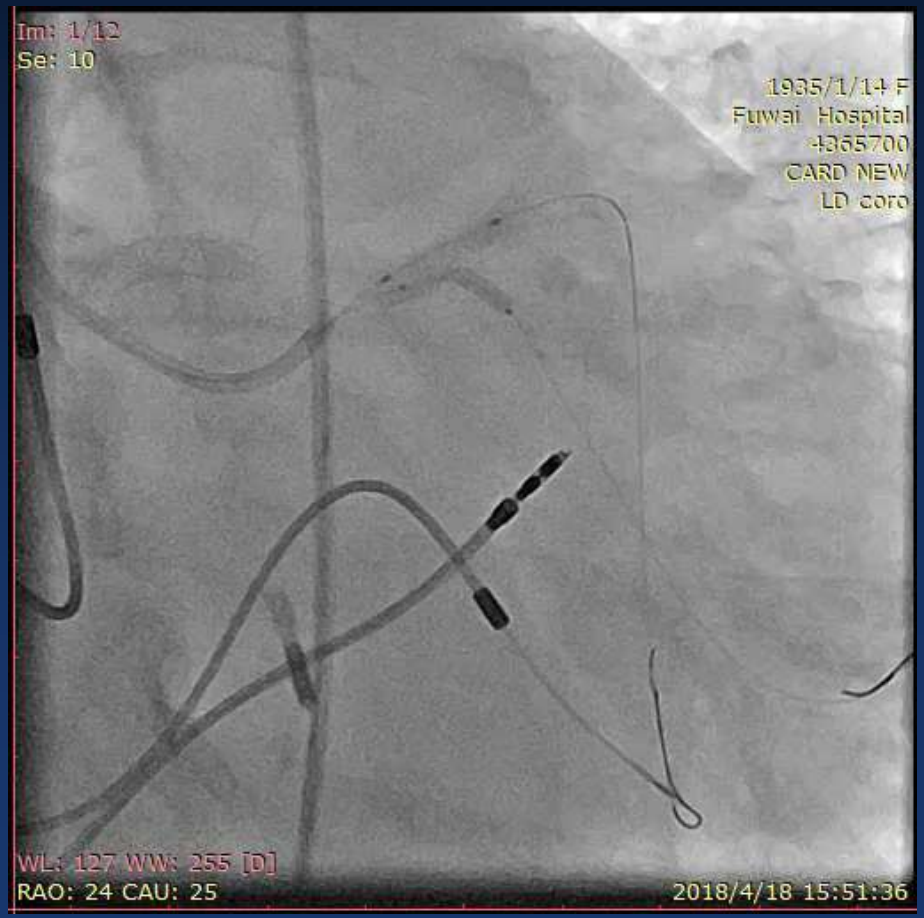

Im: 1/7
Se: 20
1935/1/14 F
Fuwai Hospital
4365700
CARD NEW
LD coro
WL: 127 WW: 255 [D]
AP
2018/4/18 16:12:16

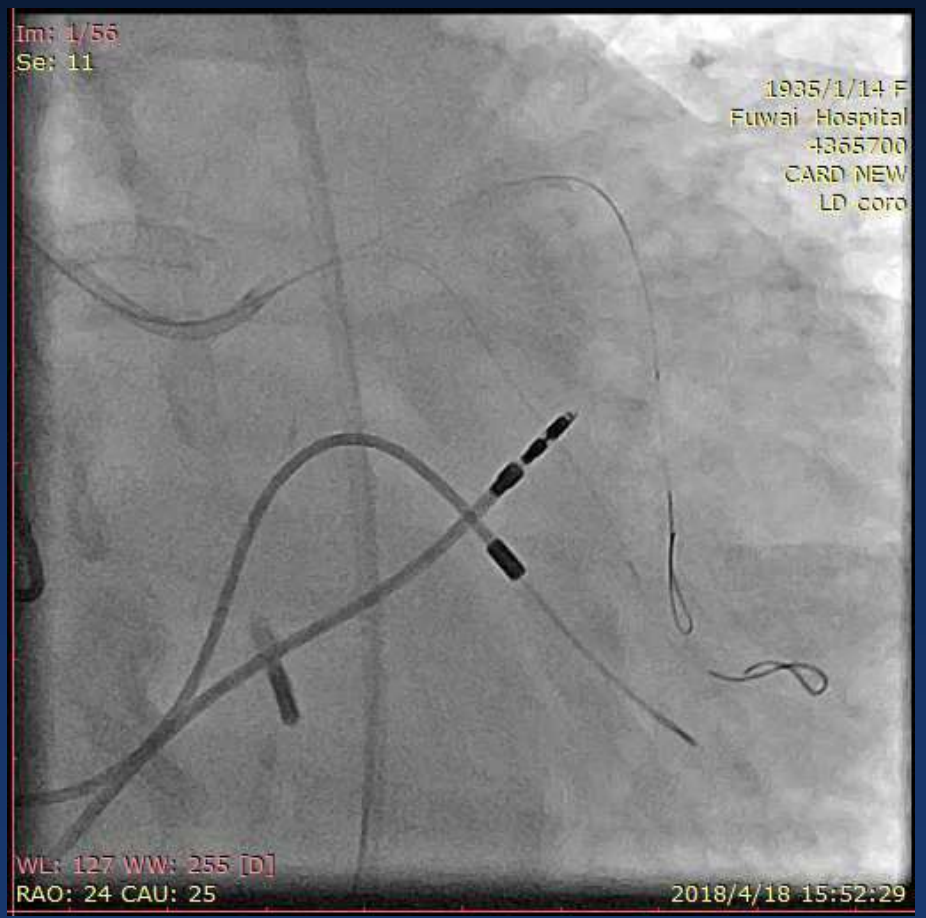

RAO: 2 CRA: 20
2018/4/18 15:38:41


Se: 8
RAO: 26 CAU: 25

对吻扩张

- 6F JL 3.5
- BMW*2
- Trek 2.5*15mm Quantum 3.5*15



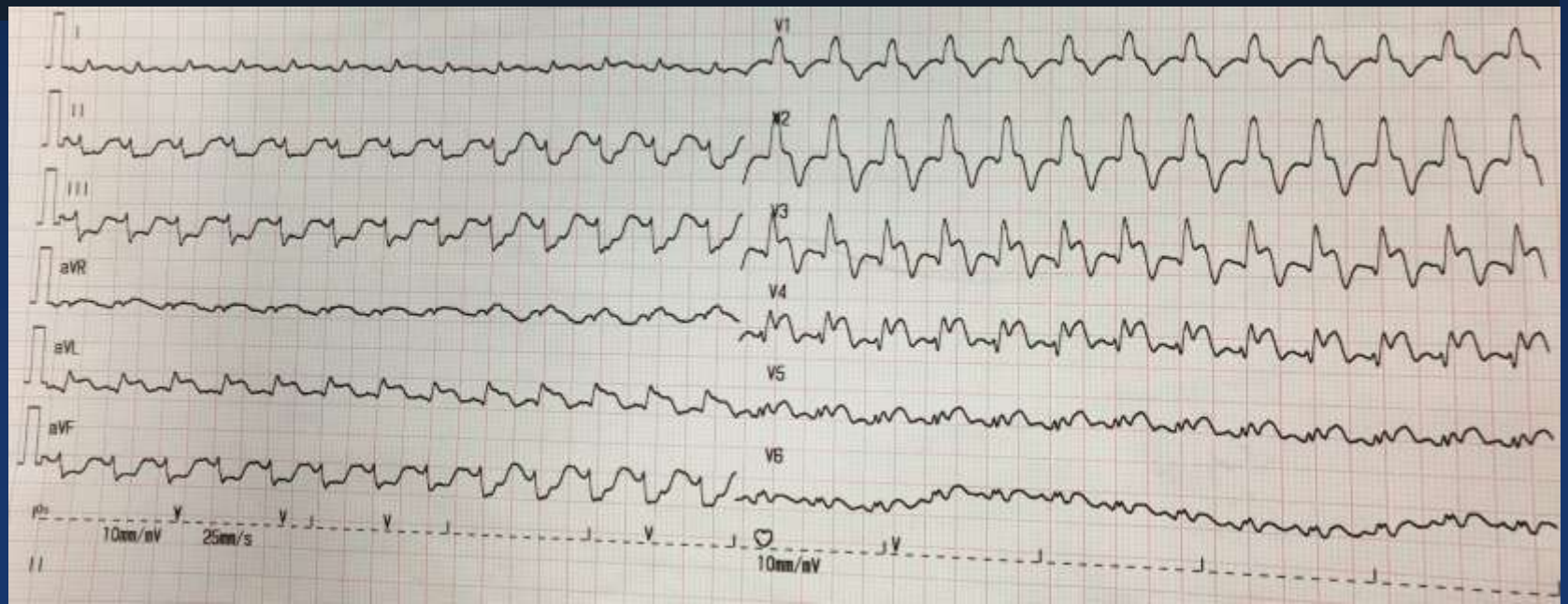


RCA-PTCA

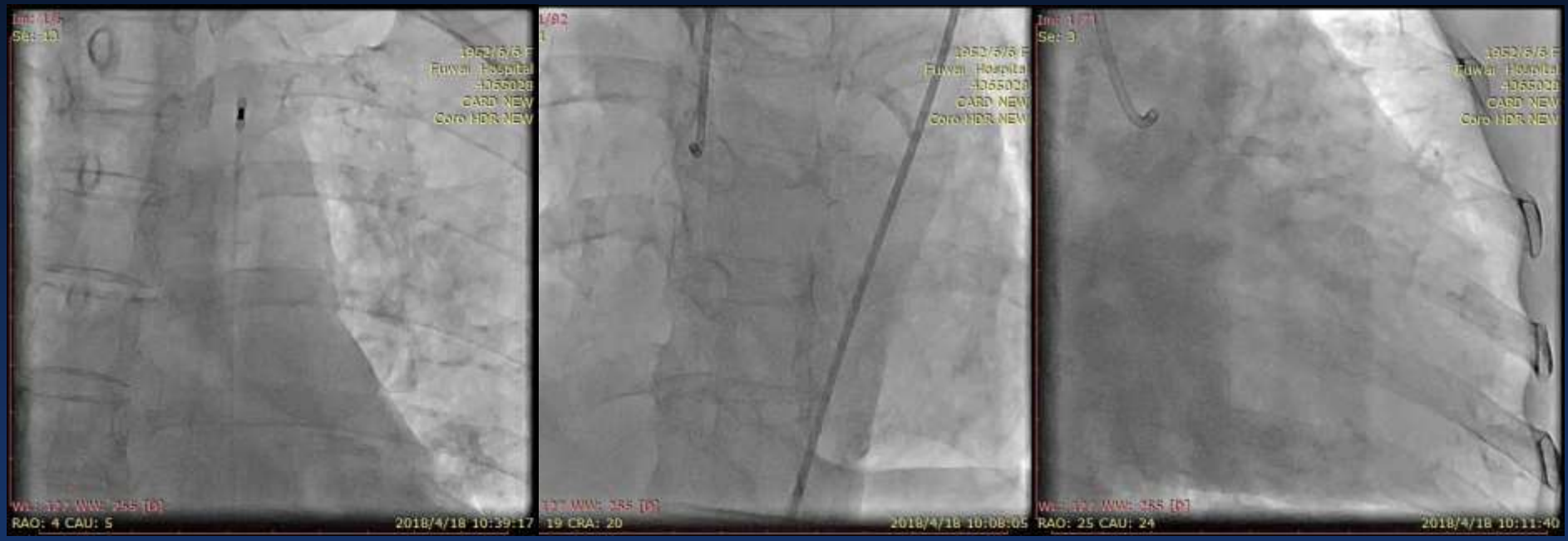

Im: 1/65
Se: 17
1935/1/14 F
Fuwai Hospital
4365700
CARD NEW
LD coro
WL: 127 WW: 255 [D]
LAO: 19 CRA: 19
2018/4/18 16:03:18

Case 2

- 女，65岁
- AMI3d，症状再发
- PE：BP87/60mmHg（DA8ug/kg/min）HR110bmp
- cTnI：1.7 ng/ml
- LV 48mm，EF30%
- 高血压、高脂血症
- 2年前曾于RCA置入支架1个



IABP辅助&造影所见

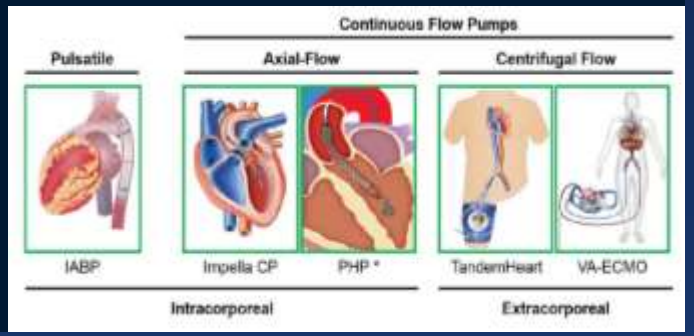

RAO: 4 CAU: 5
2018/4/18 10:39:17
19 CRA: 20
2018/4/18 10:08:05
RAO: 25 CAU: 24
2018/4/18 10:11:40

球囊扩张后


Im: 1/32
Se: 11
WL: 127 WW: 255 [D]
RAO: 30 CRA: 30
2018/4/18 10:34:5
Im: 1/39
Se: 12
WL: 127 WW: 255 [D]
RAO: 26 CAU: 25
2018/4/18 10:35:11

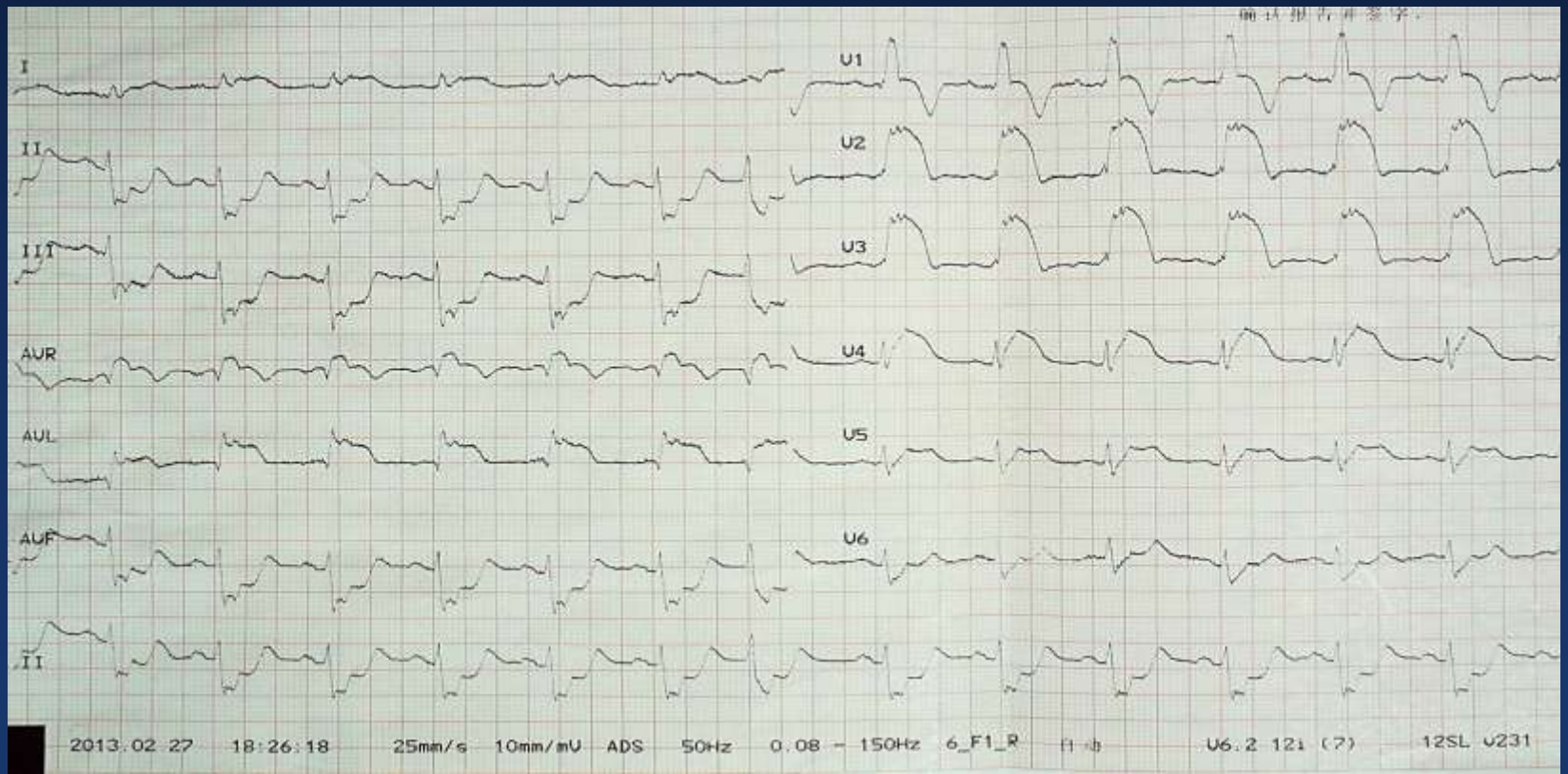
IABP生理学

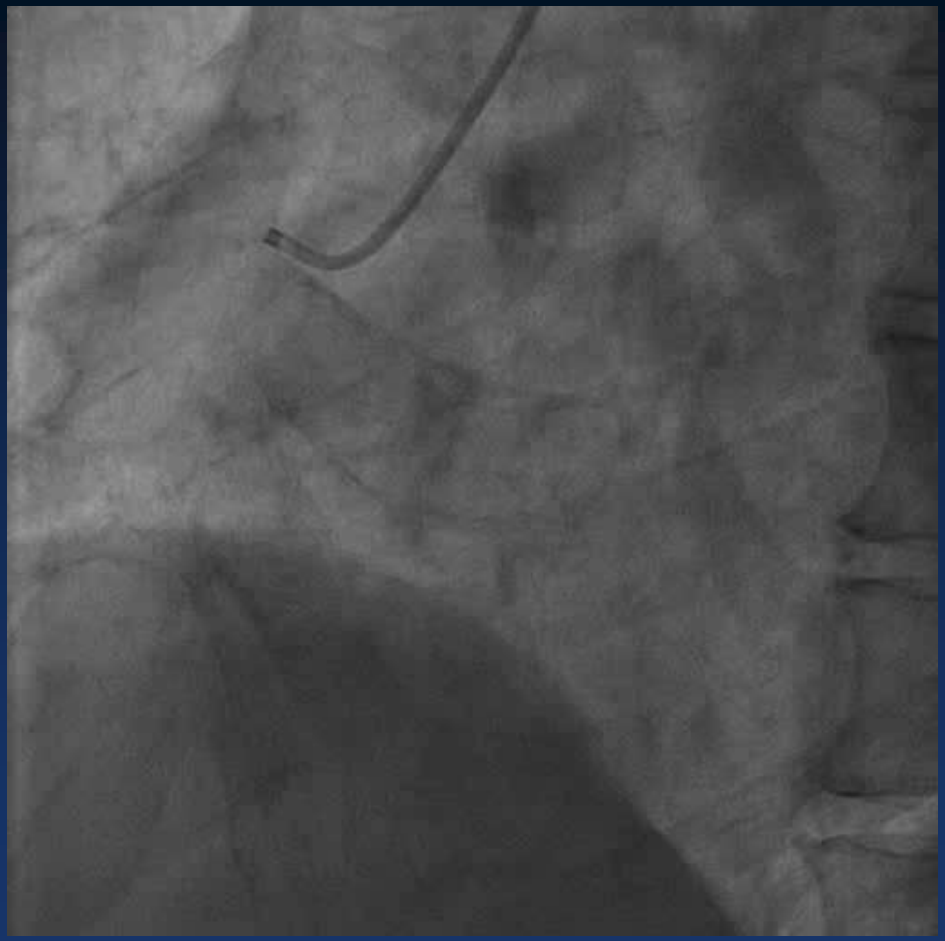
- 基于反搏
  - 舒张期球囊充盈，收缩期去充盈，增加舒张压和降低收缩压
- 降低心肌耗氧量，增加冠脉灌注，降低后负荷，并适度提高新输出量（0.5-1l /min）
- 周围组织灌注无明显增加
- 取决于自体左室收缩力



Case 3

- Male, 60 yrs.
- Chest pain for 2 hrs.
- PE: BP 70/50 mmHg, HR109 bpm, rales in both lungs
- Risk Factor: hypertension (+) , heavy smoker



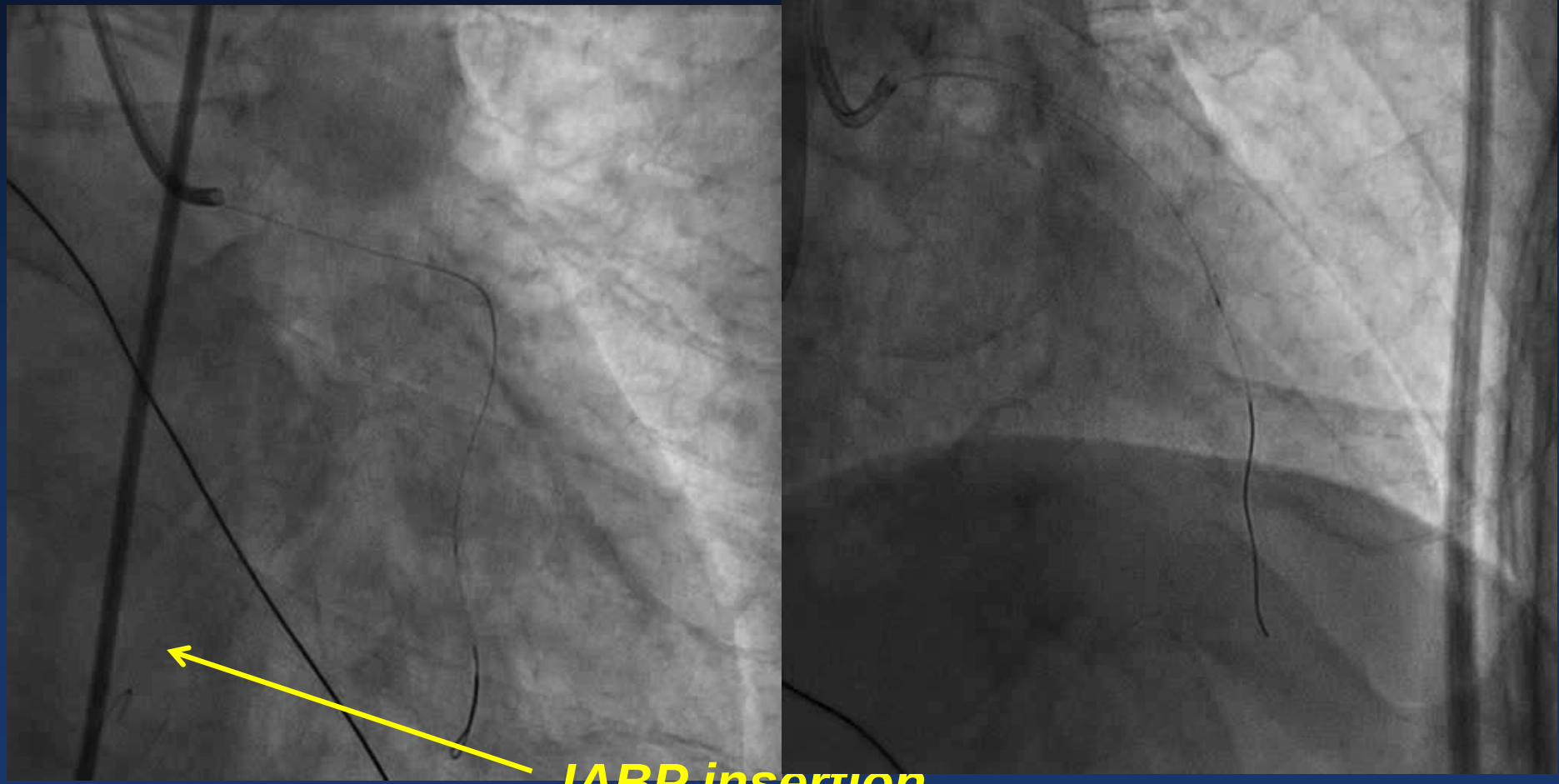


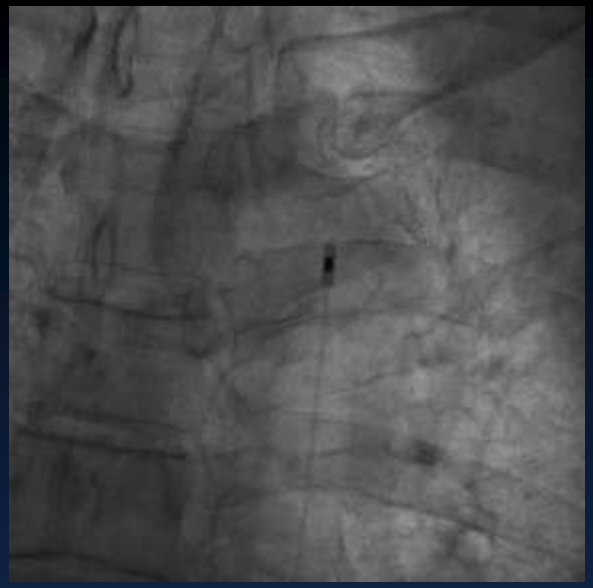
7F JL4.0

BMW

Diver

Tirofiban 1000 µg ic.





i-STAT CG8+

Pt:9
Pt Name:_____

37.0°C

pH	7.352	
PCO2	42.3	mmHg
PO2	46	mmHg
BEecf	-2	mmol/L
HCO3	23.5	mmol/L
TCO2	25	mmol/L
sO2	79	%

Na	143	mmol/L
K	5.0	mmol/L
iCa	1.06	mmol/L
Glu	235	mg/dL
Hct	43	%PCV
Hb*	14.6	g/dL

*via Hct

CPB: No

23:13 27FEB13

After PCI :

- ***Timely reperfusion: D to B = 37min***
- ***IABP assistance***
- ***DA 1500-2000μg/min iv.***
- ***NE 5 μg/min iv.***

No improvement of cardiogenic shock :

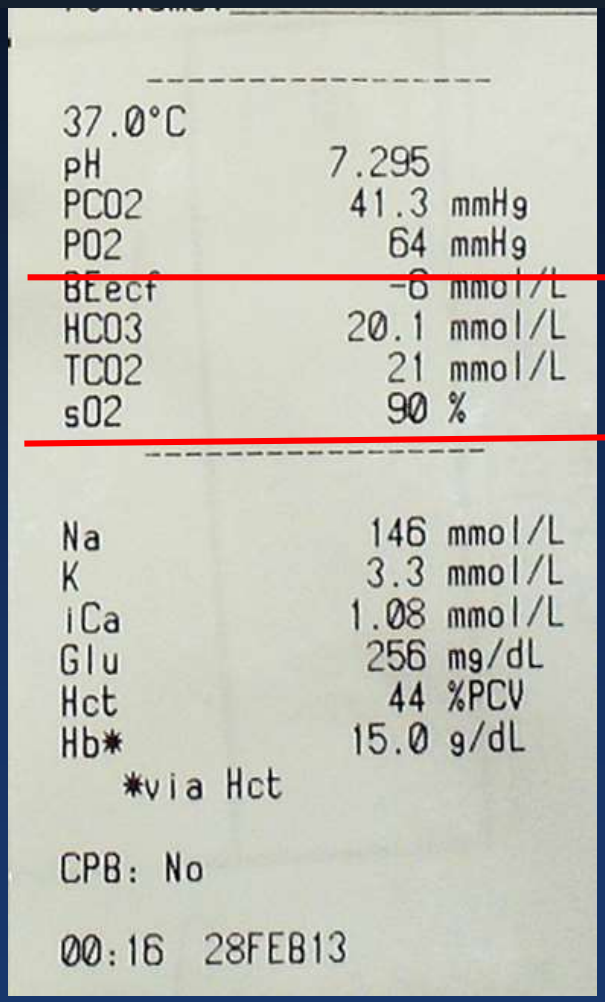
- ***PBP 80-85 mmHg, HR 115-120 bpm***

ECMO生理学

- 微型旁路提供充分的心肺支持
- 减少右室和左室容量，同时增加平均动脉压
- 降低左室前负荷，但增加后负荷，增加心肌耗氧
- 迅速改善组织氧合



	2 hrs later	6 hrs later
O2 : 10L/min	O2 : 10L/min	O2 : 10L/min



37.0°C

pH	7.295
PCO2	41.3 mmHg
PO2	64 mmHg
BEecf	-6 mmol/L
HCO3	20.1 mmol/L
TCO2	21 mmol/L
sO2	90 %
Na	146 mmol/L
K	3.3 mmol/L
iCa	1.08 mmol/L
Glu	256 mg/dL
Hct	44 %PCV
Hb*	15.0 g/dL

*via Hct

CPB: No

00:16 28FEB13

37.0°C

pH	7.279
PCO2	38.2 mmHg
PO2	92 mmHg
BEecf	-9 mmol/L
HCO3	17.9 mmol/L
TCO2	19 mmol/L
sO2	96 %
Lac	8.08 mmol/L

01:34 28FEB13

- *PBP 106-141mmHg*
- *HR 116-139 bpm*

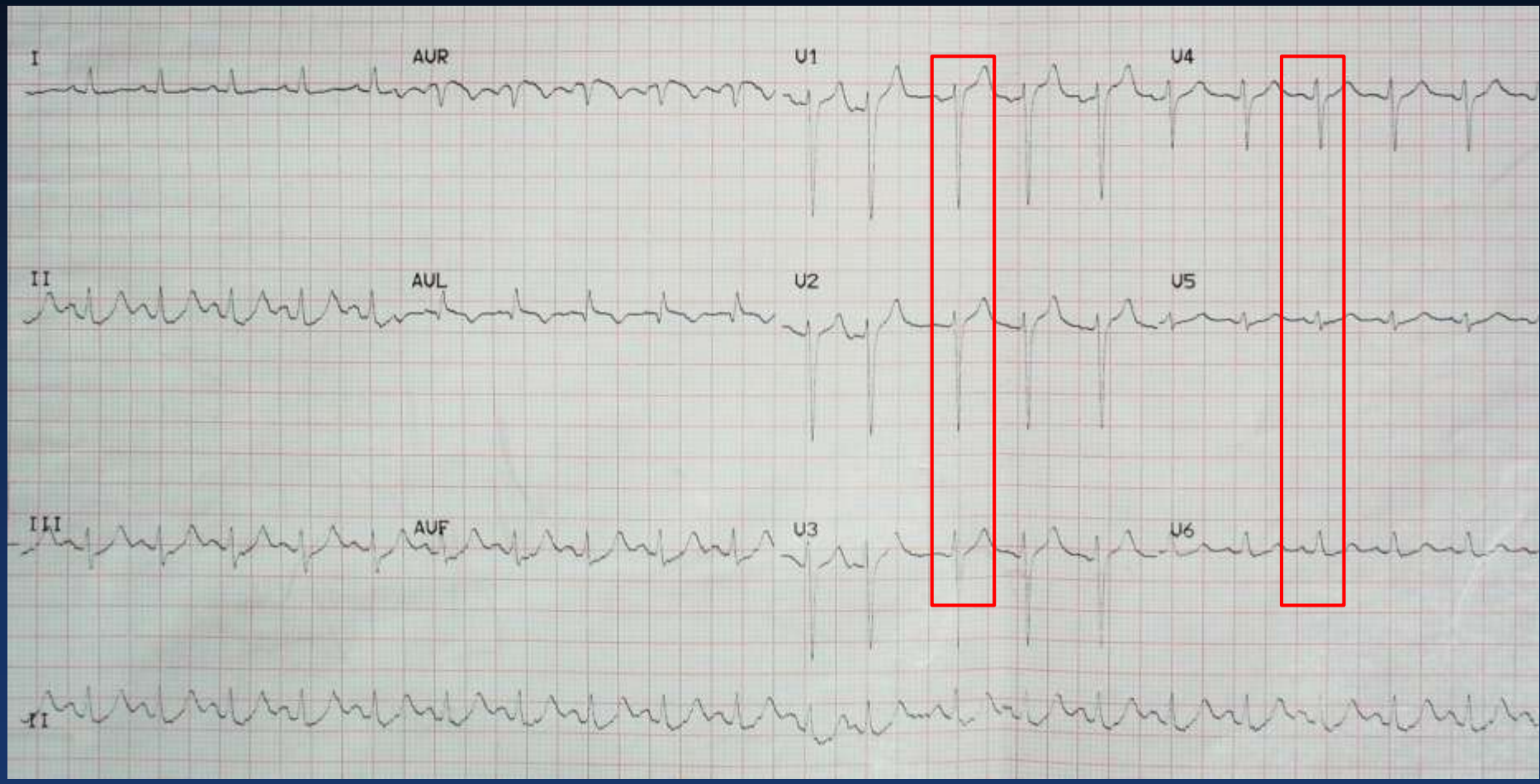
37.0°C

pH	7.409
PCO2	39.8 mmHg
PO2	175 mmHg
BEecf	0 mmol/L
HCO3	25.1 mmol/L
TCO2	26 mmol/L
sO2	100 %
Na	148 mmol/L
K	3.9 mmol/L
iCa	1.11 mmol/L
Glu	212 mg/dL
Hct	43 %PCV
Hb*	14.6 g/dL

*via Hct

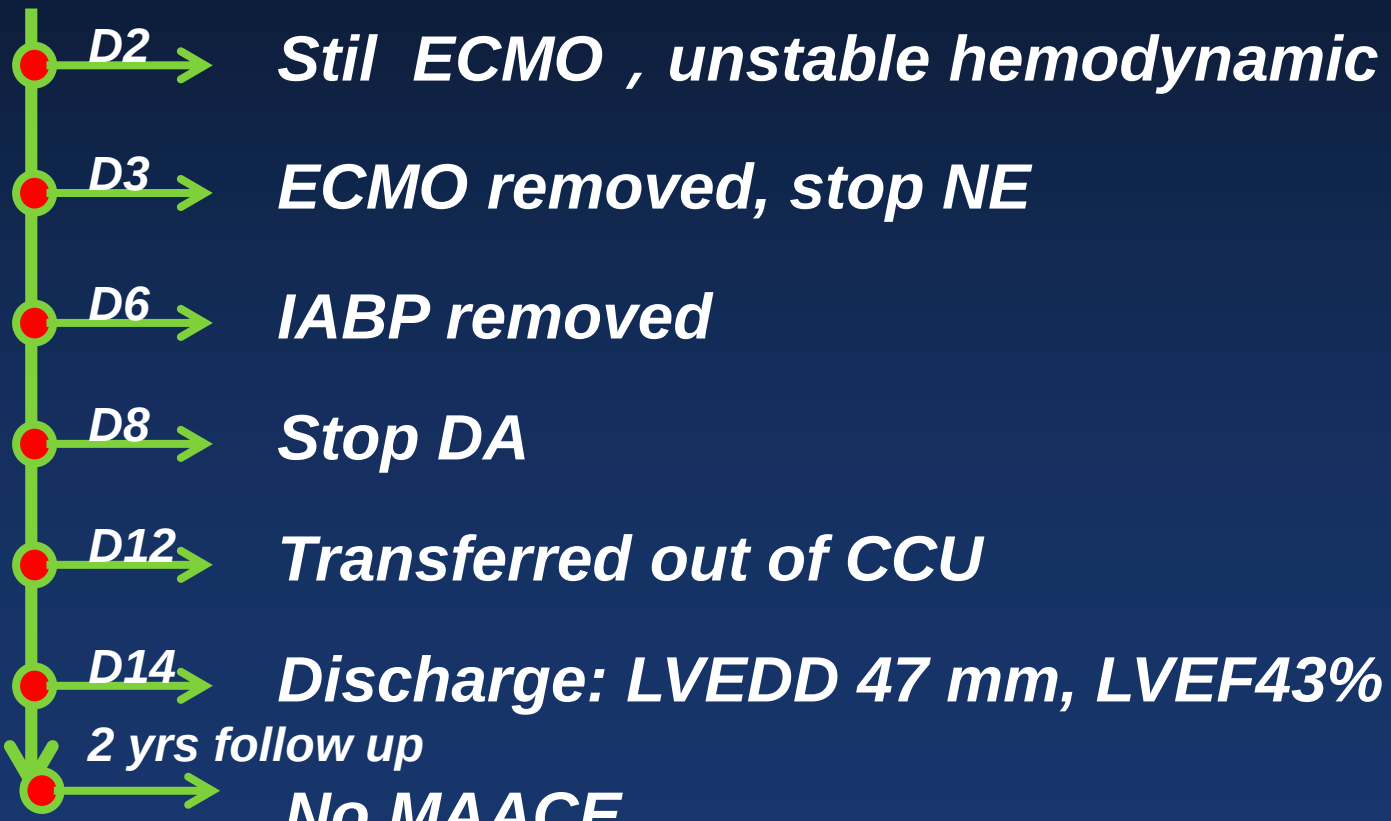
CPB: No

05:38 28FEB13

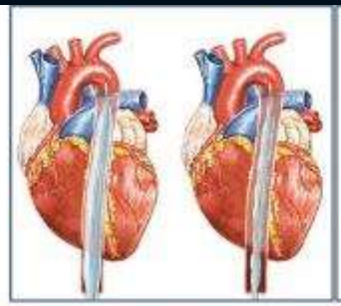
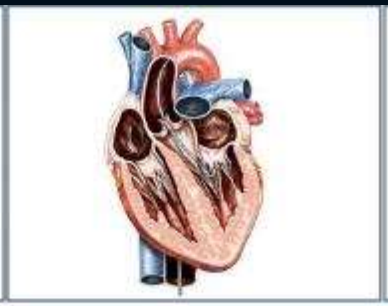

I
AUR
U1
U4
II
AUL
U2
U5
III
AUF
U3
U6
VI

Clinical Course

- Anticoagulation: UFH iv. APTT ≈ 50-70 s
- DAPT: aspirin & clopidegrol



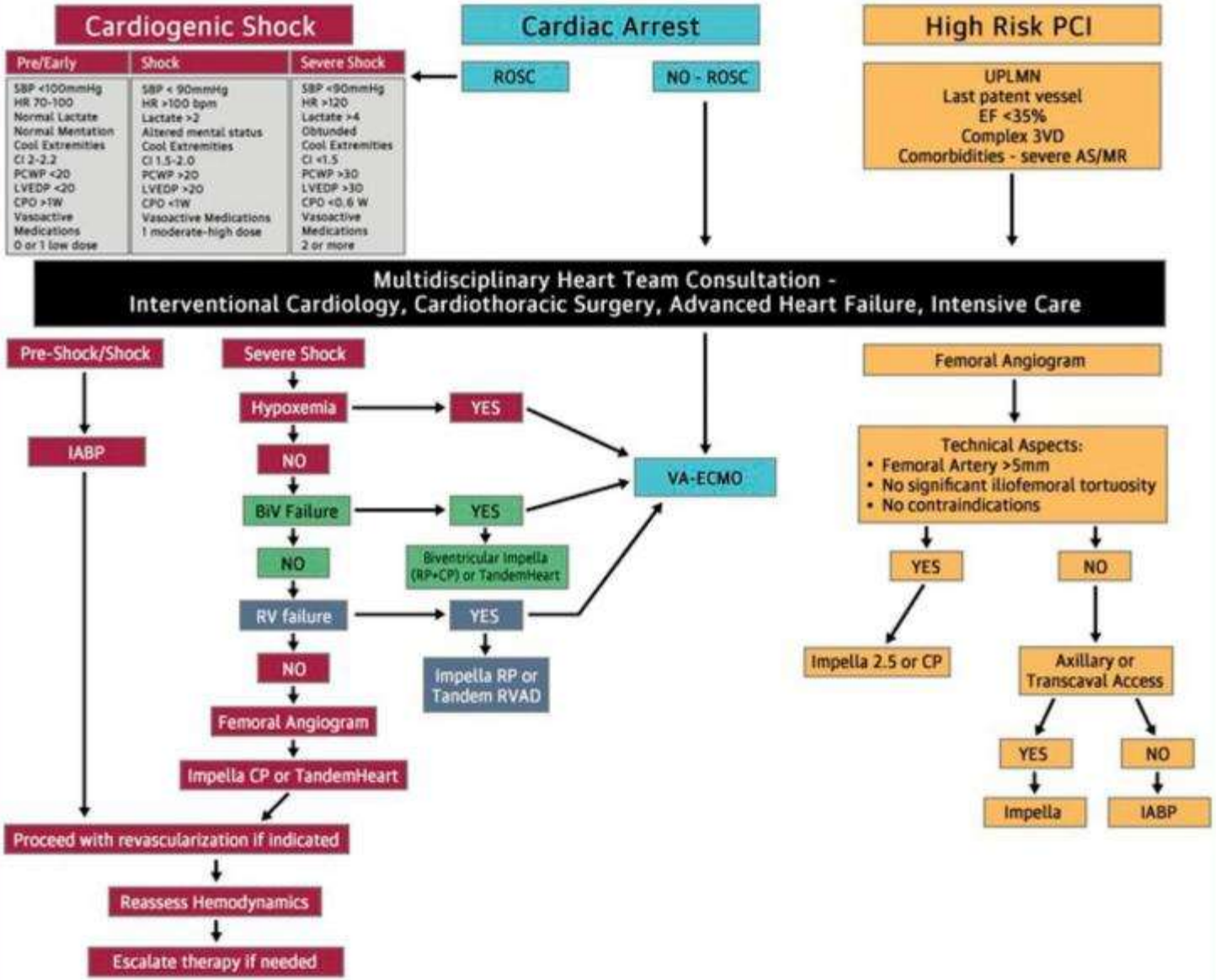
Comparison of Percutaneous Mechanical Support Devices Available

	IABP	IMPELLA	TANDEMHEART	VA-ECMO
Cardiac Flow	0.3-0.5 L/ min	1-5L/ min (Impella 2.5, Impella CP, Impella 5)	2.5-5 L/ min	3-7 L-min
Mechanism	Aorta	LV → AO	LA → AO	RA → AO
Maximum implant days	Weeks	7 days	14 days	Weeks
Sheath size	7-8 Fr	13-14 Fr Impella 5.0 - 21 Fr	15-17 Fr Arterial 21 Fr Venous	14-16 Fr Arterial 18-21 Fr Venous
Femoral Artery Size	>4 mm	Impella 2.5 & CP - 5-5.5 mm Impella 5 - 8 mm	8 mm	8 mm
Cardiac synchrony or stable rhythm	Yes	No	No	No
Afterload	↓	↓	↑	↑↑↑
MAP	↑	↑↑	↑↑	↑↑
Cardiac Flow	↑	↑↑	↑↑	↑↑
Cardiac Power	↑	↑↑	↑↑	↑↑
LVEDP	↓	↓↓	↓↓	↔
PCWP	↓	↓↓	↓↓	↔
LV Preload	---	↓↓	↓↓	↓
Coronary Perfusion	↑	↑	---	---
Myocardial oxygen demand	↓	↓↓	↔↓	↔

Tamara M. Atkinson et al. JCIN 2016;9:871-883

CENTRAL ILLUSTRATION: Algorithm for Percutaneous MCS Device Selection in Patients with Cardiogenic Shock, Cardiac Arrest, and HR-PCI



Atkinson, T.M. et al. J Am Coll Cardiol. 2016;9(9):871–83.

Outcomes for 15,259 US Patients With Acute MI Cardiogenic Shock (AMICS) Supported With Impella

Data from Abiomed's IQ registry on 1,010 hospitals, 2009-2016.

- Survival lowest for patients treated at hospitals in the lowest quintile of volume (< 1 case/yr) vs top quintile (> 7 cases/yr) at 30% vs 76% (*P* < 0.0001)
- Independent predictors of better survival were first-line vs salvage Impella use (OR 1.34; 95% CI 1.20-1.50) and use of hemodynamic monitoring (OR 1.66; 95% CI 1.48-1.87)
- Impella CP was linked to better survival vs the Impella 2.5 (OR 1.28; 1.12-1.47)

Implications: Impella use in AMICS has varied widely among US hospitals in recent years, with higher hospital volume tied to better survival.

O'Neill WW, et al. Am Heart J. 2018;Epub ahead of print.

Conclusions

- The key of contemporary management strategy in STEMI patients complicating CS is an organized approach with rapid diagnosis and prompt initiation of therapy to maintain BP and CO
  - A few available options at least
  - Understanding underlying mechanism and Individualization
  - Familiarity with assist devices critical
  - Tailor therapy based on clinical scenario and anatomy
  - Reassess rapidly and escalate to advanced therapies early before a downward spiral starts