

**MI & cardiogenic shock -  
how would I treat my sister?**


  
University Heart  
Center Graz

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LUCCA 2024

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Friday evening, I am the master of disaster in cardiology  
 Busy day, longing for dinner with the intensive care crew ...  
 ... but the surgeons need an echo in the OR, aortic dissection

  
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**My younger sister is in the emergency ward!**

Age 58

Risk Smokes a lot

History A month ago she had told me on the phone she had experienced a strange sensation in her chest and was not feeling well, two months ago, just once, she said she had felt cold sweat


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Symptoms of heart attack in women

- Chest pain, but not always
- Pain or pressure in the lower chest or upper abdomen
- Jaw, neck or upper back pain
- Nausea or vomiting
- Shortness of breath
- Fainting
- Reeling a cold sweat
- Indigestion
- Extreme fatigue

**Feeling a cold sweat**

American Heart Association.  
  
 for women.

  
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<https://www.goredforwomen.org/en/about-heart-disease-in-women>


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Sinusrhythm 102/min, SIQIII, PQ, QRS QT normal  
 high Twave V2V3

K<sup>+</sup> 4.4, hsTroponin T: 305

Diffuse hypokinesia  
 EF 30%

**Is coronary angio indicated?**

  
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What do we expect to find?

SCAD?

Spasm?

Takotsubo?

None of that,  
it is just up in her head?



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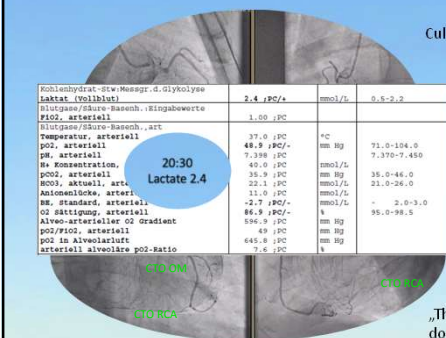
Culprit lesion?

Is it important?

Is culprit what is easy to fix?

Why open CTO  
as nice collateral flow?

„This weak myocardium  
does not need better perfusion!“



Kohlenhydrat-glucose Messgr. d.glykolyse	2.4	µmol/l	0.6-2.2
Laktat (Volllakt)	1.89	µmol/l	
Blutgas/Säure-Basenb.-Ringabewerte			
PO2, arteriell	37.0	µmol/l	
Temperatur, arteriell	48.9	µmol/l	
PO2, arteriell	7.398	µmol/l	
PO2, arteriell	40.0	µmol/l	
PO2, arteriell	35.9	µmol/l	
PO2, arteriell	22.1	µmol/l	
PO2, arteriell	21.0	µmol/l	
PO2, arteriell	2.7	µmol/l	
PO2, arteriell	86.9	µmol/l	
PO2, arteriell	596.9	µmol/l	
PO2, arteriell	49	µmol/l	
PO2, arteriell	645.8	µmol/l	
arteriell alveoläre PO2-Ratio	0.6	µmol/l	

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After angio she senses cold sweat again, BP drops to 60/50, lactate 2.4

Is she going into shock? We start epinephrine ...

**Stage E: Extremis CS.** Patients experiencing cardiac arrest with ongoing cardiopulmonary resuscitation (CPR) and/or ECMO


**Stage D: CS signals deteriorating or "Doom".** Similar to stage C but getting worse and failing to respond to initial interventions.

**Stage C: Classic CS.** Manifest CS with hypoperfusion requiring intervention (inotropes, vasopressors or MCS, excluding ECMO) beyond volume resuscitation to restore perfusion

**Stage B: Clinical evidence of relative hypotension or tachycardia without hypoperfusion** being at "Beginning" of CS (pre-shock).

**Stage A: Currently no signs/symptoms of CS, but being "At risk" for its development.**

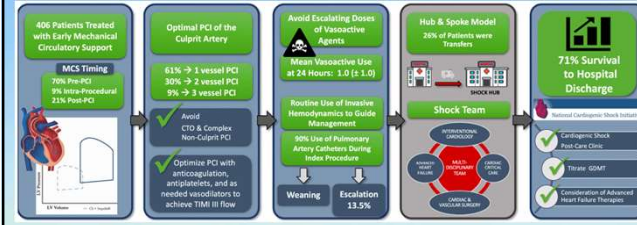
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Table 6. Comparison of Characteristics in Acute Myocardial Infarction and Cardiogenic Shock Treat

Group	Sample size	Age	Inotropes (%)	Cardiac arrest (%)	HR, bpm	BP, mmHg	Lactate, mmol/L	Lactate Δt, mmol/L	30-day survival %
SHOCK	202	68	99	28	102	89/54	N/A	N/A	53
WMH SHOCK	680	70	90	45	90	100/50	4.1	75	60
Culprit SHOCK	688	70	90	54	91	100/80	5.1	66	49
DeGae	100	68	84	0	N/A	76/50	8.5	100	N/A
MCS	408	64	98	48	98	75/50	4.8	77	68



406 Patients Treated with Early Mechanical Circulatory Support


Optimal PCI of the Culprit Artery

Assist Escalating Doses of Vasoactive Agents

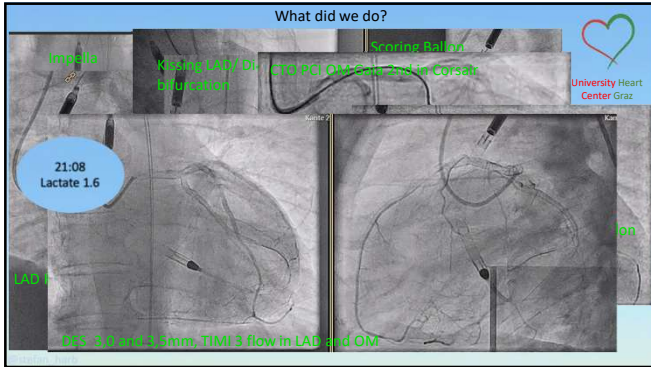
Hub & Spoke Model

71% Survival to Hospital Discharge

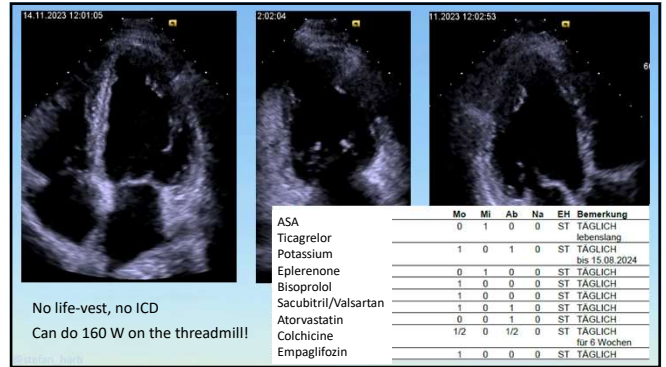
J Am Heart Assoc. 2023;12:e031401. DOI: 10.1161/JAHA.123.031401



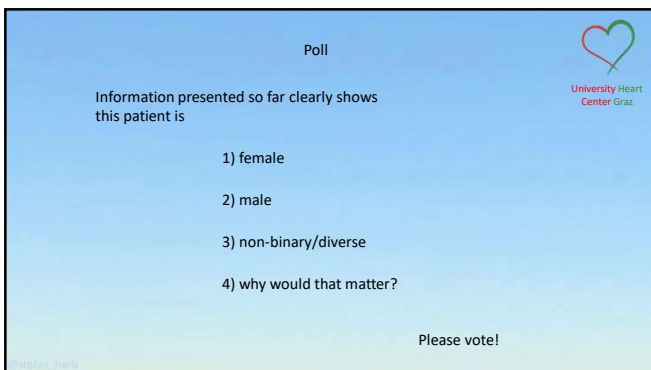
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**Reasons to open this RCA**

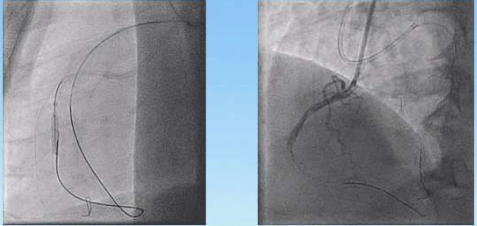
Survivor of double CTO 3VD card shock  
Had been nearly asymptomatic then

Complete revascularization like surgeon would have offered  
Only subendocardial scar lateral in stress MR  
Viable myocardium on echo inferior and RV


No randomized data for this case, neither pro nor con

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We did work on RCA CTO in second session, retrograd was difficult, only partially successful, dissected after DCB, will bring patient back



He is still doing 160 W every day



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**The Future in Cardiogenic Shock**

The Future is to prevent cardiogenic shock!

# shock develops, then:

**Potential therapeutic control options**

**Shock Centers!**

SHOCK  
CULPRIT SHOCK  
Radial Access

↓

Revascularization

MCS:  
IABP-SHOCK II  
ECLS-SHOCK  
DanGer  
Inotropes:  
DOREMI II  
Lancet/Heart

↓


Cardiac support / Hemodynamics

Inflammation  
modulation/inhibition  
DPP3-Inhibition  
Others?

↓

Systemic treatment

Holger Thiele Innsbruck 2024




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**Definition of cardiogenic shock in clinical trials and guidelines**

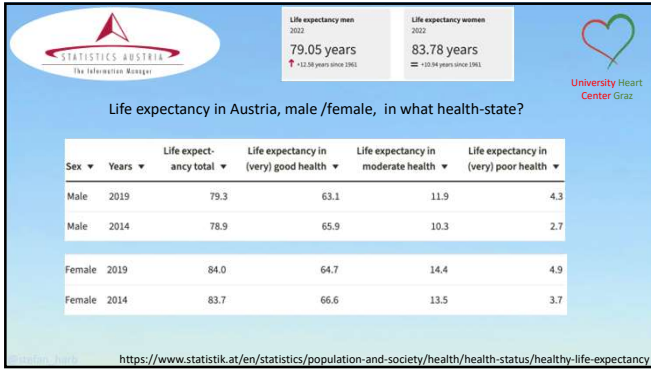
Shock	Ismp Shock II	Calpeli-Shock	ESC Heart Failure Guidelines	Ohh Risk Score
<ul style="list-style-type: none"> <li>a. SBP &lt; 90 mmHg for &gt; 30 min or</li> <li>b. End-organ hypoperfusion (urine output &lt; 30 mL/h or cool extremities and heart rate &gt; 60 bpm)</li> <li>c. CI of &lt; 2.2 L/min/m<sup>2</sup> and</li> <li>d. PCWP ≥ 15 mmHg</li> </ul>	<ul style="list-style-type: none"> <li>a. SBP &lt; 90 mmHg for &gt; 30 min or catecholamines to maintain SBP &gt; 90 mmHg and</li> <li>b. Impaired end-organ perfusion with at least one of the following criteria:                             <ul style="list-style-type: none"> <li>a. Altered mental status</li> <li>b. Cold/damp skin and extremities</li> <li>c. Urine output &lt; 30 mL/h</li> <li>d. Lactate &gt; 2.0 mmol/L</li> </ul> </li> </ul>	<ul style="list-style-type: none"> <li>Failed early revascularization by PCI</li> <li>Multifocal coronary artery disease defined as &gt; 75% stenosis in at least two major vessels (&gt; 2 mm diameter) with identifiable culprit lesion</li> <li>a. SBP &lt; 90 mmHg for &gt; 30 min or</li> <li>b. Catecholamines required to maintain SBP &gt; 90 mmHg</li> <li>c. Pulmonary congestion</li> <li>d. Impaired organ perfusion with at least one of the following criteria:                             <ul style="list-style-type: none"> <li>a. Altered mental status</li> <li>b. Cold/damp skin and extremities</li> <li>c. Urine output &lt; 30 mL/h</li> <li>d. Lactate &gt; 2.0 mmol/L</li> </ul> </li> </ul>	<ul style="list-style-type: none"> <li>SBP &lt; 90 mmHg with adequate volume and distal or laboratory signs of hypoperfusion</li> <li>Clinical hypoperfusion: Cold extremities, oliguria, mental confusion, dizziness, and narrow pulse pressure</li> <li>Laboratory hypoperfusion: Metabolic acidosis, Elevated lactate, Elevated creatinine</li> </ul>	<ul style="list-style-type: none"> <li>SBP &lt; 90 mmHg for &gt; 30 min following exclusion of hypovolemia, with clinical evidence of hypoperfusion, inotrope dependence or mechanical left ventricular support to correct this situation</li> </ul>

\* Not required in anterior infarction or if pulmonary congestion in chest X-ray; CI, cardiac index; ESC, European Society of Cardiology; PCI, percutaneous coronary intervention; PCWP, pulmonary capillary wedge pressure; SBP, systolic blood pressure.

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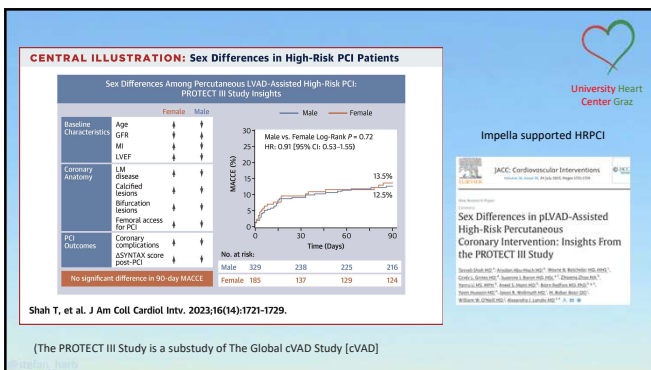
This patient is male, 48 years of age

Considerations, open questions, ...

Given that resources are restricted, should there be an age limit for protected PCI?

Given later onset of atheromatosis in women and higher female life-expectancy, should such an age limit be higher for women?

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