

# Demystifying Cardiogenic Shock and Coronary Disease Management

VERMONT CARDIAC NETWORK

ERIC ROTHSTEIN, MD

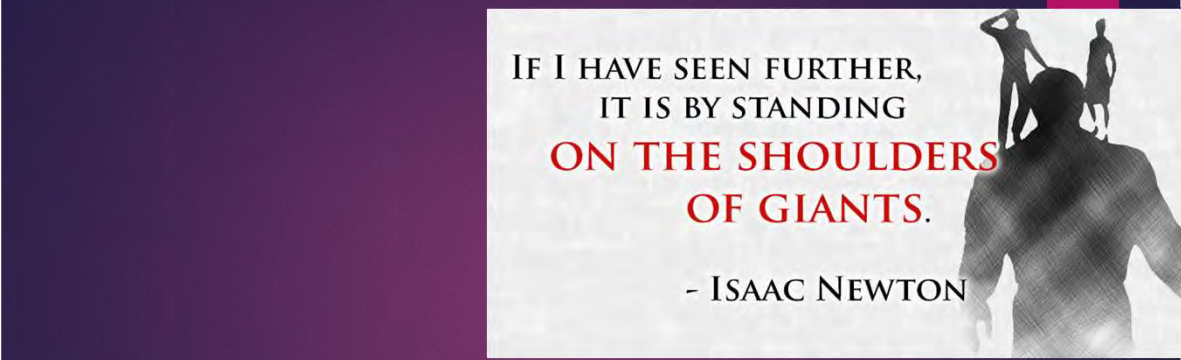
INTERVENTIONAL CARDIOLOGY

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## Outline and Objectives

- ▶ Discuss the evolution of the modern cath lab with respect to
  - ▶ Coronary Interventions
  - ▶ Shock
- ▶ Understand the challenges of complex PCI
- ▶ Review management of cardiogenic shock and mechanical circulatory support

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IF I HAVE SEEN FURTHER,  
IT IS BY STANDING  
**ON THE SHOULDERS  
OF GIANTS.**  
- ISAAC NEWTON

# The History Of Our Field

THE BRILLIANT MINDS WHO DEFIED THE ODDS TO CREATE THE FIELD OF  
CARDIOLOGY

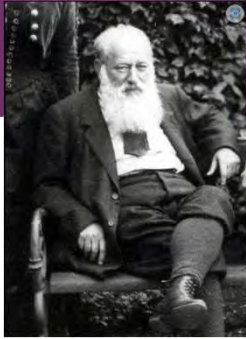
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“

A surgeon who tries to suture a heart wound deserves to lose the esteem of his colleagues.

”

THEODOR BILLROTH: 1852



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## The First Heart Surgery

- ▶ September 7, 1896
  - ▶ Wilhelm Justus, a gardener's assistant, got into a bar fight and was stabbed in the chest
  - ▶ He found semi conscious outside a tavern by a policeman and brought to Frankfurt City Hospital
  - ▶ Dr. Ludwig Rehn percussed his chest and examined his neck veins and diagnosed tamponade
  - ▶ Dr. Rehn opened his chest, performed a pericardiectomy and saw a small hole in the RV
  - ▶ He occluded the hole with his finger and then placed three sutures in the RV
  - ▶ Patient awoke two hours later and survived

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## The Next 50 years

- ▶ Multiple attempts at heart surgery all ended in disaster
  - ▶ Failed battlefield shrapnel removal in The Great War
  - ▶ Failed mitral valve procedures
  - ▶ Multiple failures to treat battlefield cardiac wounds early on in WWII in France
- ▶ The United States enters World War 2
  - ▶ US Army Corps forbids its surgeons from operating on the heart
  - ▶ Dr. Dwight Harken argued for the chance to try
    - ▶ British Royal College of Surgeons agrees



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## June 6, 1944: D-Day

- ▶ A soldier with a gaping chest wound and intracardiac shrapnel was brought to his OR
- ▶ Dr. Harken clamped the fragment of shrapnel and pulled it out
  - ▶ Immediately plugged the hole with his finger
  - ▶ Four sutures were placed and the soldier survived
- ▶ He then repeated the procedure on 16 more soldiers with intracardiac shrapnel over the next few months
  - ▶ 100% survival
  - ▶ He publishes his experiences and the field of cardiac surgery is born



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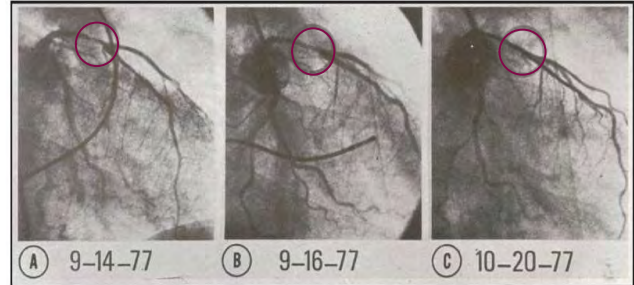
## After the War

- ▶ Dr. Charles Bailey attempts the first mitral stenosis repair
  - ▶ Patient hemorrhages to death after sutures tear the left atrium
- ▶ He tries again on another patient with mitral stenosis
  - ▶ The valve is torn to pieces and the patient dies of severe MR
  - ▶ Dr. Bailey earns the name "Butcher Bailey"
  - ▶ Operating privileges revoked
- ▶ He gets privileges at another hospital and tries on a third patient
  - ▶ Uses a finger blade
  - ▶ Patient dies of severe MR
  - ▶ Privileges revoked
- ▶ He gets privileges at 2 more hospitals
  - ▶ Schedules two more patients with MS on the same day at different hospitals
    - ▶ Kills his fourth patient
  - ▶ He starts the 5<sup>th</sup> surgery before news traveled
    - ▶ The fifth patient survived her mitral valve repair

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## A Brief History of Coronary Revascularization

- ▶ **1929:** Dr. Werner Forssmann performs the first right heart catheterization on himself
- ▶ **1953:** Dr. Jack Gibbon creates the first heart/lung, cardiopulmonary bypass machine and uses it to perform an ASD closure
- ▶ **1958:** Dr. Mason Sones takes the first selective coronary angiogram
- ▶ **1967:** Dr. Rene Favaloro performs the first CABG procedure
- ▶ **1977:** Dr. Andrees Gruntzig performs the first coronary angioplasty in a stable patient
- ▶ **1980:** Dr. Geoff Harzler performs angioplasty in an unstable STEMI
- ▶ **1987:** The first Palmaz-Schatz bare metal stent is deployed in a human coronary artery



Smilowitz, N.R., Feit, F. The History of Primary Angioplasty and Stenting for Acute Myocardial Infarction. *Curr Cardiol Rep* 18, 5 (2016). <https://doi.org/10.1007/s11866-015-0681-x>

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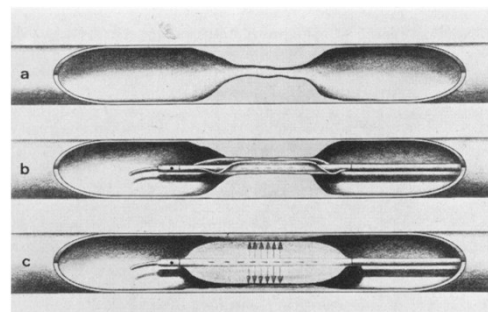
## Angioplasty in the 80's

ORIGINAL ARTICLE [ARCHIVE](#)

### Nonoperative Dilatation of Coronary-Artery Stenosis — Percutaneous Transluminal Coronary Angioplasty

Andreas R. Gruntzig, M.D., Åke Senning, M.D., and Walter E. Siegenthaler, M.D.

- ▶ 10F sheath placed in femoral artery
- ▶ 9F (3mm) catheter tracked to the artery
- ▶ Balloon dilatation catheter with built in wire steered through the artery and inflated for 3 seconds to low pressure
- ▶ Equipment removed



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## PCI in the 80's

### ▶ Dangerous

- ▶ Complication rate of 10-20%
  - ▶ Myocardial infarction rate: 5%
  - ▶ 6-8% Emergency Bypass
    - ▶ Much higher mortality rates than standard bypass surgery
  - ▶ 1% VF
  - ▶ 4% Access site hemorrhage
  - ▶ 1-3% mortality
    - ▶ 2.5% mortality w single vessel PCI
    - ▶ 15% mortality with multivessel PCI

### ▶ Questionable Efficacy

- ▶ Success defined as a 20% increase in luminal area
- ▶ Despite "cherry picking" lesions
  - ▶ Failure rate of 10-60%
    - ▶ Unable to deliver the balloon
    - ▶ Unable to dilate the lesion with a balloon
    - ▶ Angioplasty creates a dissection and the vessel abruptly closes

Vandormael, M., Ischinger, T., Roth, R. (1986). Angioplasty Equipment and Supplies: Technical Considerations. In: Practice of Coronary Angioplasty. Springer, Berlin, Heidelberg. [https://doi.org/10.1007/978-3-642-70815-2\\_7](https://doi.org/10.1007/978-3-642-70815-2_7)

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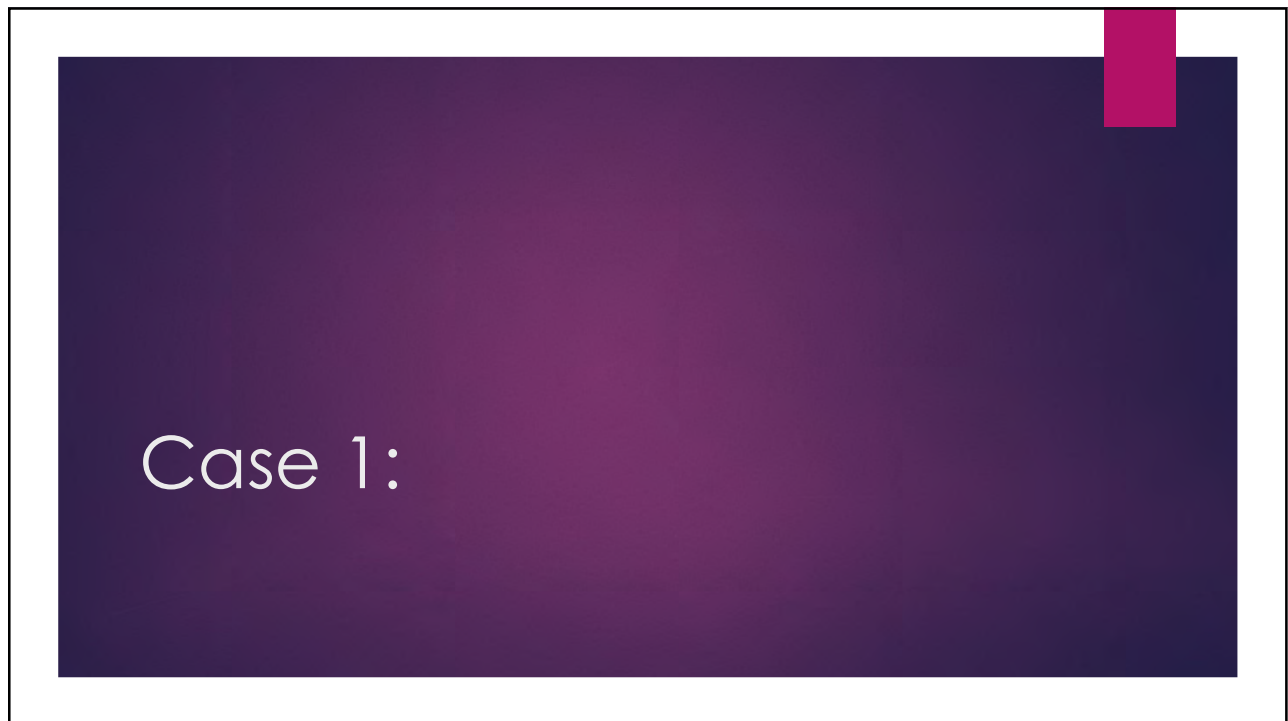
## A Brief History of PCI at DHMC

- ▶ **1985:** Dr. Bruce Hettleman performed the first coronary angioplasty in the state of New Hampshire and is soon joined by Dr. John Robb
- ▶ **1986-2000:** Core group of interventional cardiology faculty developed proficiency with angioplasty and stenting
- ▶ **2001:** Dr. Nathaniel Niles develops the first acute MI program
- ▶ **2002-2003:** Dr. Donald Baim is brought to DHMC to assist with complex and high risk cases
- ▶ **2006:** Dr. Craig Thompson starts developing initial techniques for PCI of chronic total occlusions
- ▶ **2009:** Multiple doctors start working through radial access
- ▶ **2010-Present:** Increasing case complexity with focus on minimization of complications and maximizing long lasting, durable results

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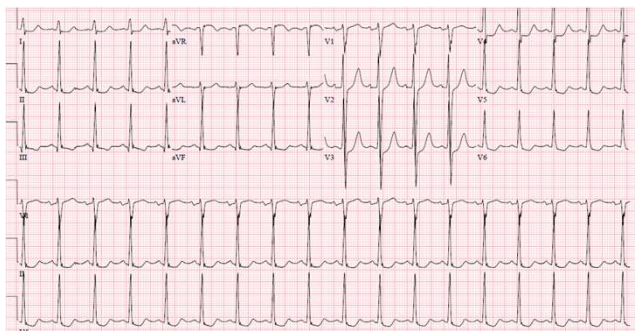
## Case 1: Introduction

- ▶ 21 year old college student with no prior medical or psychiatric history was brought to the DHMC ED via EMS with concern for intentional sodium-fluoride poisoning
  - ▶ Campus security called to his apartment by roommate after he had locked himself in his room and had begun vomiting and slurring his speech
  - ▶ Security found him unresponsive but breathing with a pulse
  - ▶ A bottle of concentrated sodium-fluoride powder from the chemistry laboratory was found next to him

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## ED Presentation (Midnight, Day 0)

- ▶ Pertinent Exam Details:
  - ▶ Vitals: T- 98.9 | BP- 90/30 | P-117 | R- 29 | SaO<sub>2</sub>- 79%
  - ▶ Gen: Unresponsive, diaphoretic
  - ▶ HEENT: Nasopharyngeal airway in place, bloody secretions in oropharynx
  - ▶ Pulm: Tachypneic, shallow respirations
  - ▶ Card: Tachycardic, no murmurs
  - ▶ Neuro: Minimally responsive to painful stimuli
- ▶ Initial ED Management
  - ▶ Intubated for airway protection
  - ▶ ECG Obtained: QT/QTc- 384/504 ms

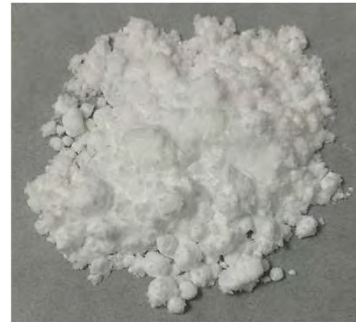


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## An Interventionalist's Guide to Fluoride

- ▶ Sodium Fluoride (NaF) is a colorless, odorless, tasteless powder
  - ▶ Used in trace amounts in toothpaste and drinking water
  - ▶ Easily available for purchase online
- ▶ Hydrofluoric Acid (HF) is a weak acid
  - ▶ Found in wheel cleaner, insecticides, brick cleaners



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## An Interventionalist's Guide to Fluoride (2)

- ▶ Aqueous solutions of fluoride are rapidly absorbed in the stomach
  - ▶ Slowly deposited in the bones and teeth
  - ▶ Rapidly complexes with calcium and magnesium
- ▶ 5-10g is considered a lethal dose
  - ▶ Our patient is estimated to have taken well over 200g
- ▶ **Non-cardiac Complications**
  - ▶ Profound hypocalcemia
  - ▶ Hypomagnesemia
  - ▶ Delayed hyperkalemia
  - ▶ Coagulopathy / DIC
  - ▶ GI Hemorrhage / Necrosis
- ▶ **Cardiac Complications**
  - ▶ Ventricular arrhythmias

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## ED Course (00:00 – 00:30, Day 0)

### Presenting Labs

- ▶ CBC
  - ▶ WBC: 7 | Hgb: 17 | Plts: 228
- ▶ CMP
  - ▶ Na: 143 | K: 3.9 | Cl: 101 | **CO2: 16** | BUN: 18 | **Cr: 1.4**
  - ▶ LFT's: Unremarkable
- ▶ Coags
  - ▶ **INR 1.1** | Fibrinogen: Normal | Thrombin Time: 15 | **D-Dimer: 14,000**
- ▶ ABG
  - ▶ **pH: 7.3** | **pCO2: 35** | **PO2: 60** | **Bicarb 16**
- ▶ Drugs / Toxins:
  - ▶ Negative
- ▶ Other Labs
  - ▶ **Ca: 6.2 (ref range 8.5-10.5)**
  - ▶ **Lactate: 4.3**

### Management

- ▶ 3 PIV's, central and arterial access
- ▶ Toxicology consult
  - ▶ Calcium chloride boluses
  - ▶ Calcium gluconate & Mag sulfate drips
- ▶ 2L IV crystalloid

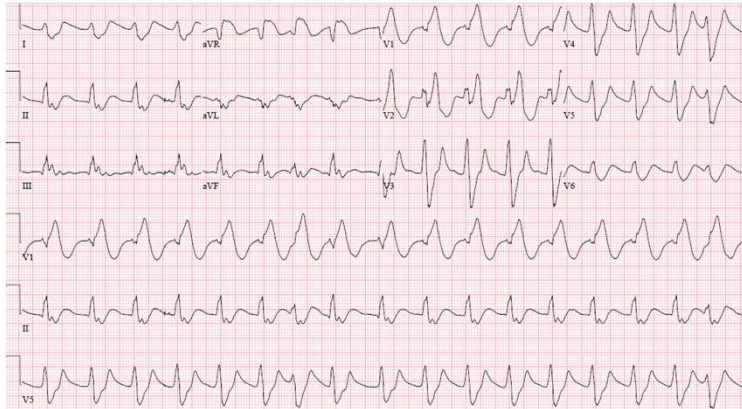
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## ED Course (00:30 – 02:00, Day 0)

- ▶ **Persistently hypotensive**
  - ▶ Another 2L crystalloid and norepinephrine at 3 mcg/min
- ▶ **Bleeding (Oropharyngeal and oozing from all access sites)**
  - ▶ 2 units RBC's administered
  - ▶ CT Imaging shows edematous stomach but no perforation.
  - ▶ GI recommends against scoping
- ▶ **Nephrology Consultation**
  - ▶ Initiate emergent CVVH
  - ▶ Lasix & Insulin/D10 for hyperkalemia
- ▶ **Critical Care**
  - ▶ Stable enough for transfer to the ICU

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## ECG (02:00, Day 0)



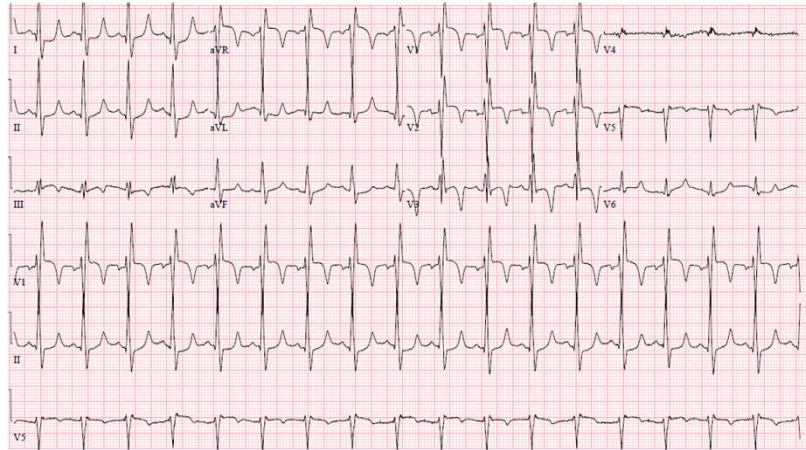
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## ICU Course (02:00 – 05:00, Day 0)

- ▶ Patient remains hemodynamically stable
- ▶ ICU Team Management:
  - ▶ Trying to correct electrolytes
    - ▶ Hypocalcemia and hypomagnesemia worsen (5.1 & 0.2 respectively)
  - ▶ DIC worsens despite treatment
    - ▶ INR: 4 | Fibrinogen: undetectable | D-Dimer: Too high to measure
    - ▶ Persistent bleeding from oropharynx and all IV sites
    - ▶ Transfusing FFP, Cryo, RBC's and Plts
  - ▶ Dialyzing
  - ▶ Checking EKG's
    - ▶ QRS Narrowing, QTc remains under 500
    - ▶ Deferring empiric antiarrhythmic therapy

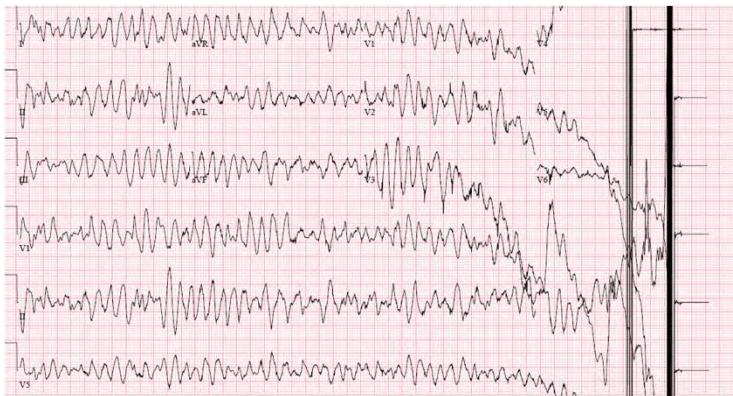
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## ECG (04:55, Day 0)



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## ECG (05:09, Day 0)



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## Polymorphic VT Arrest

- ▶ Unresponsive to 200J defibrillation
- ▶ CPR initiated
- ▶ Amiodarone + Epinephrine administered
- ▶ Defibrillation attempted every two minutes
- ▶ Lidocaine administered
- ▶ More amiodarone administered (450mg total)
- ▶ More lidocaine administered (200mg total)
- ▶ CPR Continued
- ▶ Refractory to 12 attempts at defibrillation
- ▶ Cardiac surgery / Interventional cardiology consulted for extracorporeal cardiopulmonary resuscitation (e-CPR)



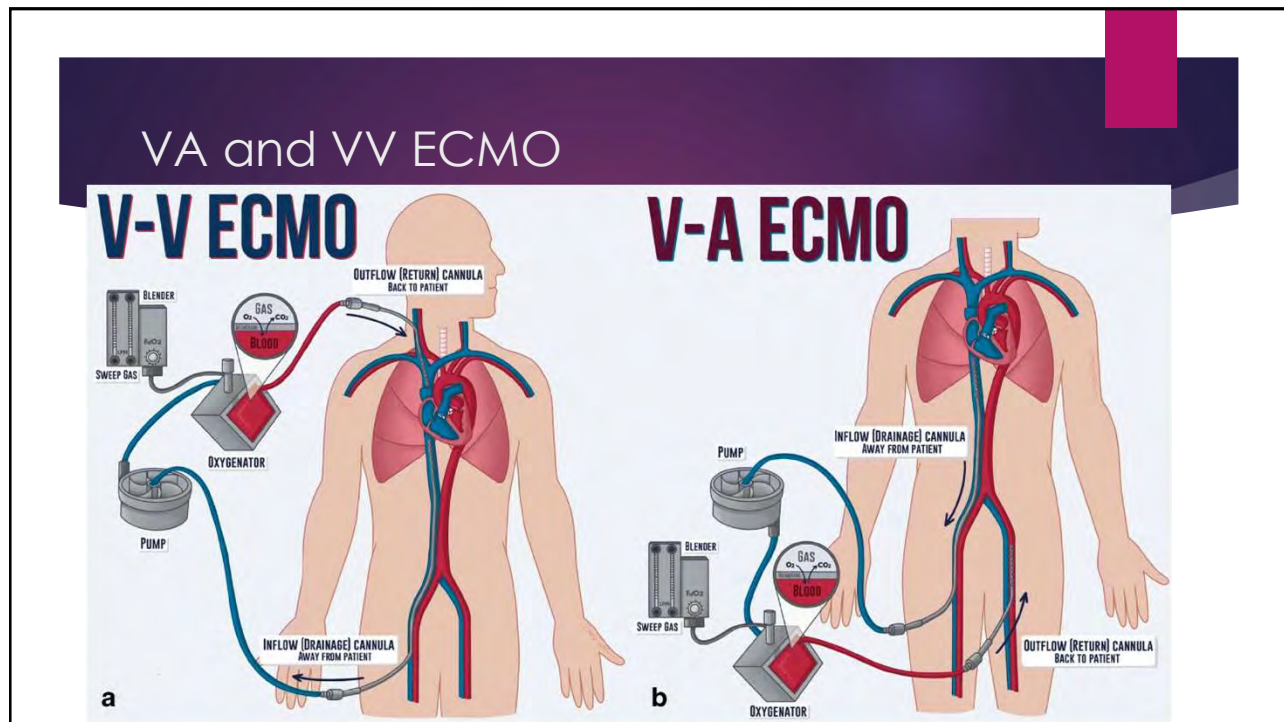
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## Why Add the E to CPR?

- ▶ Survival rates after cardiac arrest in hospital are dismal despite ACLS and CPR
  - ▶ All comers: 17% survival
  - ▶ Patients with VF: 34% survival
- ▶ Survival rates are even worse for out of hospital arrests
- ▶ Adding extracorporeal life support has been trialed since 1976
  - ▶ Current extracorporeal membranous oxygenation (ECMO) platforms allow for this to be rapidly deployed and utilized in the setting of cardiac arrests
- ▶ Contemporary E-CPR survival rates range from 40-60%

Peberdy MA, Kaye W, Omato JP, Larkin GL, Nadkarni V, Mancini ME, Berg RA, Nichol G, Lane-Trull T. Cardiopulmonary resuscitation of adults in the hospital: a report of 14720 cardiac arrests from the National Registry of Cardiopulmonary Resuscitation. *Resuscitation*. 2003 Sep;58(3):297-308. doi: 10.1016/s0300-9572(03)00215-6. PMID: 12969608.

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## Indications for ECMO

### Veno-arterial (VA)

Common	Other
1. <i>Cardiogenic shock</i> : AMI and complications (including wall rupture, papillary muscle rupture, refractory VT/VF) refractory to conventional therapy including IABP	1. Pulmonary embolism
2. <i>Postcardiac surgery</i> : Unable to wean safely from cardiopulmonary bypass using conventional supports	2. Cardiac or major vessel trauma
3. Drug overdose with profound cardiac depression	3. Massive hemoptysis / pulmonary hemorrhage
4. Myocarditis	4. Pulmonary trauma
5. <i>Early graft failure</i> : Postheart / heart-lung transplant	5. Acute anaphylaxis
	6. Peripartum cardiomyopathy
	7. Sepsis with profound cardiac depression
	8. Bridge to transplant

### Venvenous (VV)

#### Pathological Processes Suitable for Venovenous Extracorporeal Membrane Oxygenation

##### Common

- Severe pneumonia
- ARDS
- Acute lung (graft) failure following transplant
- Pulmonary contusion

Optimal ventilation  
(Consider recruitment,  
prone, inhaled prostacyclin)  
and  $paO_2/FiO_2 < 60$   
or  
 $paO_2/FiO_2 < 100$  and  $paCO_2 > 100$  mm Hg for  $> 1$  hr

##### Other

- Alveolar proteinosis
- Smoke inhalation
- Status asthmaticus
- Airway obstruction
- Aspiration syndromes.

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## Indications for ECMO

### Veno-arterial (VA)

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### Venvenous (VV) Not MCS

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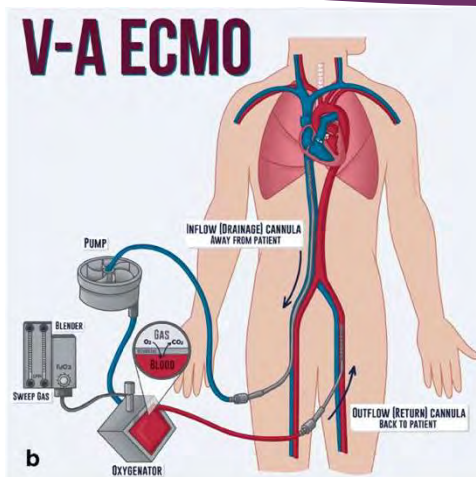
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(Consider recruitment, prone, inhaled prostacyclin) and  $paO_2/FiO_2 < 60$  or  $paO_2/FiO_2 < 100$  and  $paCO_2 > 100$  mm Hg for  $> 1$  hr

##### Other

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## The VA-ECMO Circuit

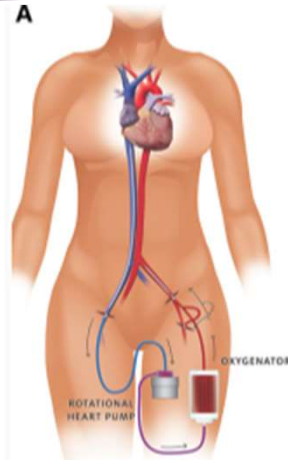


- ▶ Blood is pulled through the venous cannula into the pump
- ▶ The pump pressurizes the blood, which then flows to the oxygenator
- ▶ The oxygenator allows for
  - ▶ Diffusion of oxygen into the blood ( $FiO_2$ )
  - ▶ Removal of  $CO_2$  (Sweep gas flow)
  - ▶ Temperature control (heat exchanger)
- ▶ Blood is returned to the body through the arterial cannula (pressurized and oxygenated)

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## What is E-CPR

- ▶ Transitioning the patient from CPR to VA-ECMO for systemic perfusion
- ▶ Steps:
  - ▶ Continue CPR (occasional pauses as needed for operators)
  - ▶ Large bore access and cannulation of the femoral artery and femoral vein
  - ▶ Anticoagulation and priming of the system
  - ▶ Perfusionist starts, calibrates and optimizes the ECMO circuit
  - ▶ CPR stopped



Honeya A, Philipp A, Diez C, Schopka S, Bein T, Zimmermann M, Lubnow M, Luchner A, Agha A, Hiker M, Hirt S, Schmid C, Müller T. A 5-year experience with cardiopulmonary resuscitation using extracorporeal life support in non-postcardiotomy patients with cardiac arrest. *Resuscitation*. 2012 Nov;83(11):1331-7. doi: 10.1016/j.resuscitation.2012.07.009. Epub 2012 Jul 20. PMID: 22819880.

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## Back to Our Case: Arrest Course

- ▶ Patient prepared for transport to the cath lab for Extracorporeal cardiopulmonary resuscitation (e-CPR)
- ▶ Procainamide 100mg administered
- ▶ Patient is defibrillated to normal sinus rhythm on (lucky) shock number 13
  - ▶ MAP >65mmHg
  - ▶ e-CPR deferred..... For now...

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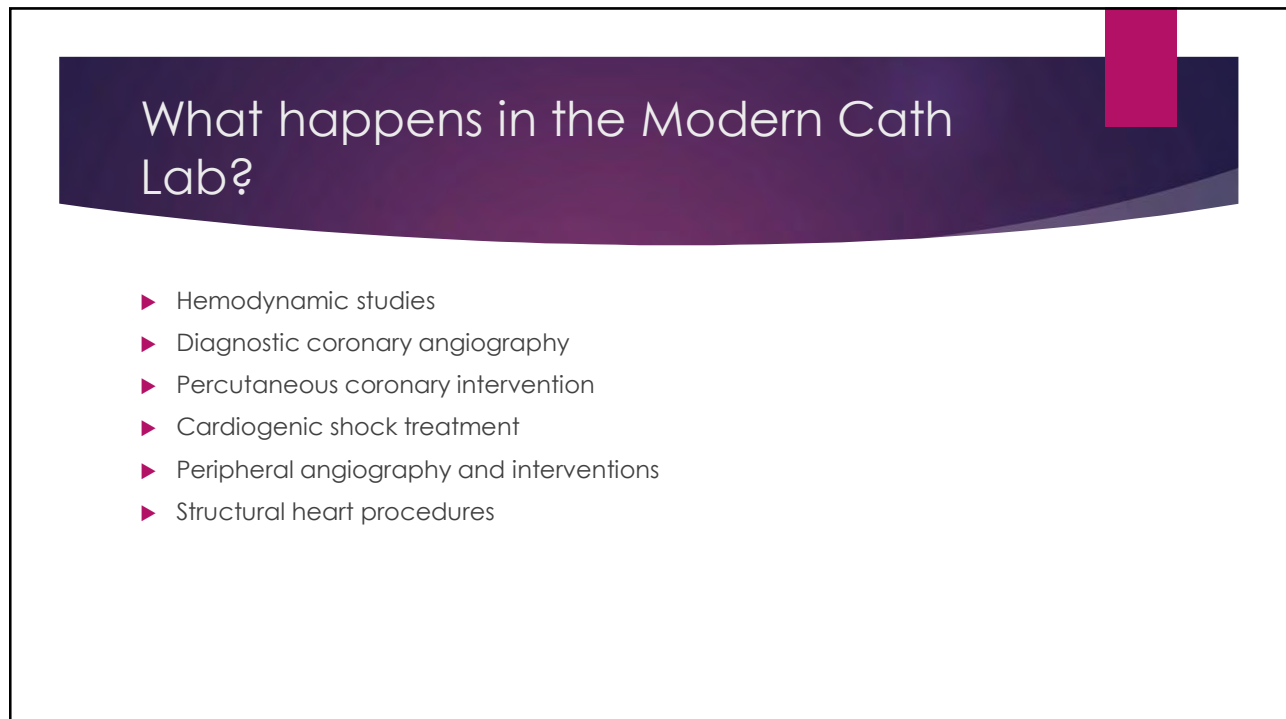




The Modern Cath Lab

THE ROLE CONTINUES TO EVOLVE AND GROW

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What happens in the Modern Cath Lab?

- ▶ Hemodynamic studies
- ▶ Diagnostic coronary angiography
- ▶ Percutaneous coronary intervention
- ▶ Cardiogenic shock treatment
- ▶ Peripheral angiography and interventions
- ▶ Structural heart procedures

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## What happens in the Modern Cath Lab?

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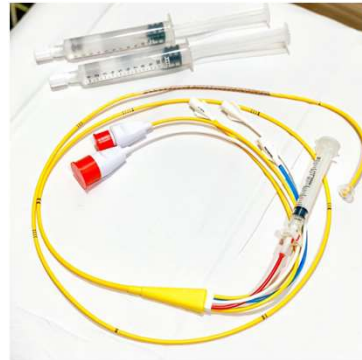
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## Invasive Hemodynamics

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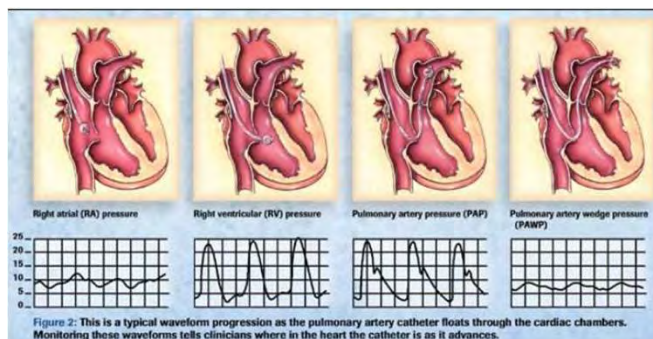
## Invasive Hemodynamics

- ▶ Allows for understanding and differentiation of shock etiologies
  - ▶ Hypovolemic / Hemorrhagic
  - ▶ Distributive
    - ▶ Neurogenic
    - ▶ Anaphylaxis
    - ▶ Sepsis / SIRS
  - ▶ Cardiogenic
  - ▶ Obstructive
    - ▶ PE
    - ▶ RV failure
    - ▶ Tension PTX
    - ▶ Tamponade



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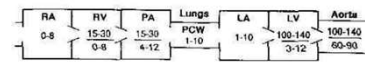
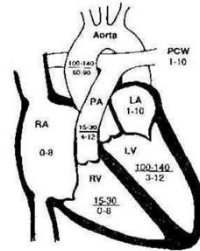
## Typical Waveforms



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## Measured Waveforms & Values

- ▶ Measured by waveforms:
  - ▶ RA: 5 mmHg
  - ▶ RV: 25 mmHg
  - ▶ PA: 25/10 (15) mmHg
  - ▶ PCW: 10 mmHg
  - ▶ LVEDP: 10 mmHg
- ▶ Measured using thermistor
  - ▶ Cardiac output (thermodilution): 5 L/min



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## Hemodynamic Studies



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## Calculated Values

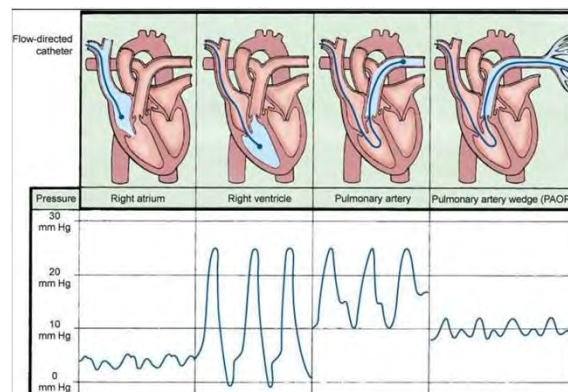
- ▶ Cardiac Output (Estimated Fick)
  - ▶  $CO = VO_2 / [(SaO_2 - MVO_2) * k]$
- ▶ Cardiac Index
  - ▶  $CI = CO/BSA$
- ▶ Pulmonary vascular resistance
  - ▶  $PVR = (Pam - PCW) / CO$
- ▶ Systemic Vascular Resistance
  - ▶  $SVR = 80 * (MAP - CVP) / CO$
- ▶ Pulmonary Artery Pulsatility Index
  - ▶  $PAPI = (PAs - Pad) / CVP$
- ▶ Cardiac Power Output
  - ▶  $CPO = (MAP * CO) / 451$



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## Invasive Hemodynamics

- ▶ Allows for optimal management of cardiogenic shock
  - ▶ Divides the heart into separate manageable components
    - ▶ Right sided preload (RA pressure)
    - ▶ Right sided contractility (PAPI)
    - ▶ Right sided afterload (PVR)
    - ▶ Left sided preload (PCW / LVEDP)
    - ▶ Left sided contractility (CO\*)
    - ▶ Left sided afterload (SVR)



\*Cardiac output is a rough surrogate for LV performance

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## What happens in the Modern Cath Lab?

- ▶ Hemodynamic studies
- ▶ Diagnostic coronary angiography
- ▶ Percutaneous coronary intervention
- ▶ **Cardiogenic shock treatment**
- ▶ Peripheral angiography and interventions
- ▶ Structural heart procedures

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## Cardiogenic Shock Management (According to the Interventionalist)

- |   |  |
|---|--|
| <ul style="list-style-type: none"> <li>▶ Right sided preload (RA pressure)           <ul style="list-style-type: none"> <li>▶ Too high: Diurese</li> <li>▶ Too low: IV Fluids</li> </ul> </li> <li>▶ Right sided contractility (PAPI)           <ul style="list-style-type: none"> <li>▶ Too low: Inotropes or <b>RV MCS</b></li> </ul> </li> <li>▶ Right sided afterload (PVR)           <ul style="list-style-type: none"> <li>▶ Too high: iNO or epoprostinol</li> </ul> </li> </ul> | <ul style="list-style-type: none"> <li>▶ Left sided preload (PCW / LVEDP)           <ul style="list-style-type: none"> <li>▶ Too high: Diurese</li> <li>▶ Too low: Fluids</li> </ul> </li> <li>▶ Left sided contractility (CPO or CO)           <ul style="list-style-type: none"> <li>▶ Too low: Inotropes or <b>LV MCS</b></li> </ul> </li> <li>▶ Left sided afterload (SVR)           <ul style="list-style-type: none"> <li>▶ Too high: vasodilators</li> <li>▶ Too low: vasopressors</li> </ul> </li> </ul> |
|---|--|

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## What happens in the Modern Cath Lab?

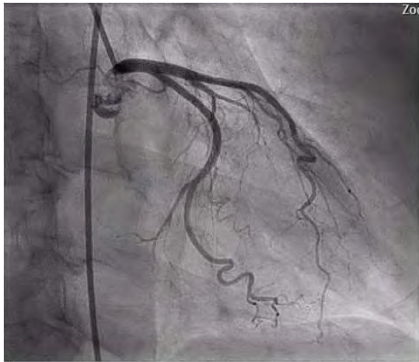
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## Brief Coronary Angiogram Tutorial

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## Diagnostic Angiography: Normal Arteries

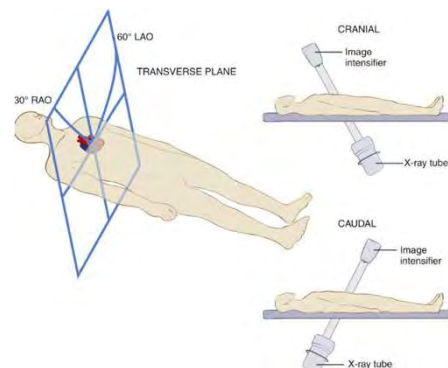


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## Warning: Lots of Pictures in This Talk

- ▶ Coronary angiography is a 2D representation of a 3D structure
  - ▶ "Shadow Puppets"

	Cranial	AP	Caudal
RAO	RAO/CRA A	RAO	RAO/CAU
AP	AP/CRA	Flat AP	AP/CAU
LAO	LAO/CRA A	LAO	LAO/CAU

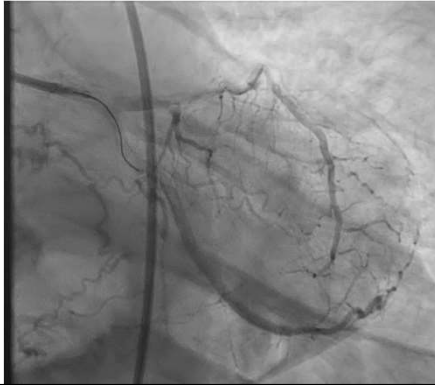


48



## Quick Primer on Left System

Caudal



Cranial



49

## Quick Primer on the Right System



50

## What happens in the Modern Cath Lab?

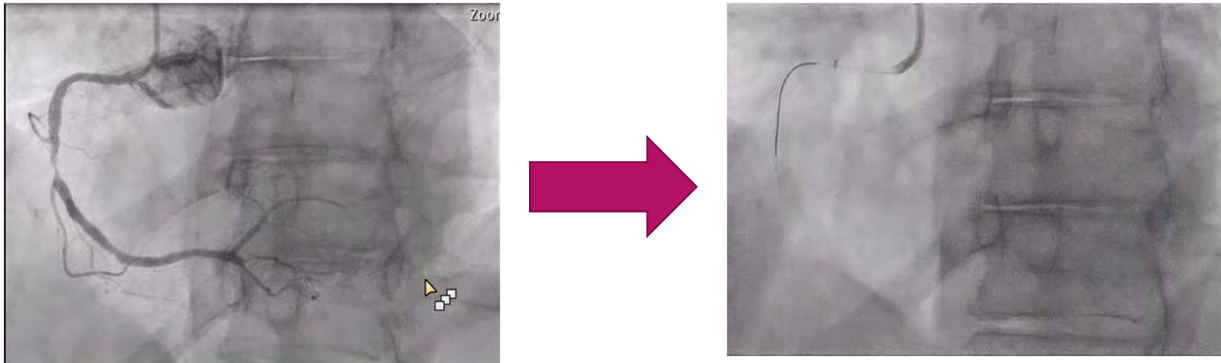
- ▶ Hemodynamic studies
- ▶ Diagnostic coronary angiography
- ▶ Percutaneous coronary intervention
- ▶ Cardiogenic shock treatment
- ▶ Peripheral angiography and interventions
- ▶ Structural heart procedures

51

## Brief Tutorial on Percutaneous Coronary Intervention (PCI)

52

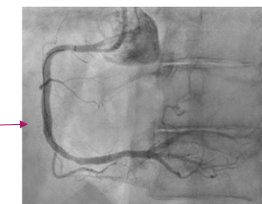
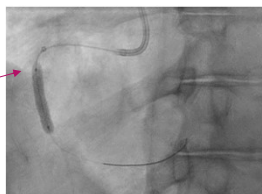
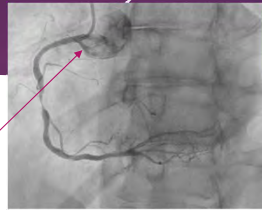
## Quick Primer on PCI



53

## How to Fix a Coronary Artery

1. Insert sheath into radial artery
2. Engage the coronary artery with a catheter and inject contrast
3. Advance a wire across the blockage
4. Advance a balloon over the wire, squash the plaque to the side, then repeat with a stent mounted on a balloon
5. Take a final picture showing the beautiful fixed artery



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## What happens in the Modern Cath Lab?

- ▶ Hemodynamic studies
- ▶ Diagnostic coronary angiography
- ▶ Percutaneous coronary intervention
- ▶ Cardiogenic shock treatment
- ▶ Peripheral angiography and interventions
- ▶ Structural heart procedures

55

## What happens in the Modern Cath Lab?

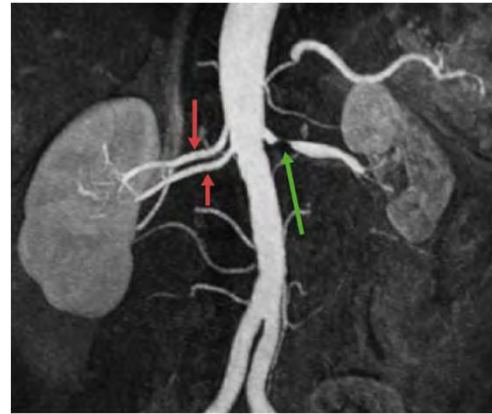
- ▶ Hemodynamic studies
- ▶ Diagnostic coronary angiography
- ▶ Percutaneous coronary intervention
- ▶ Cardiogenic shock treatment
- ▶ Peripheral angiography and interventions
- ▶ Structural heart procedures



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## A Coronary Operator's Take on Peripheral Intervention

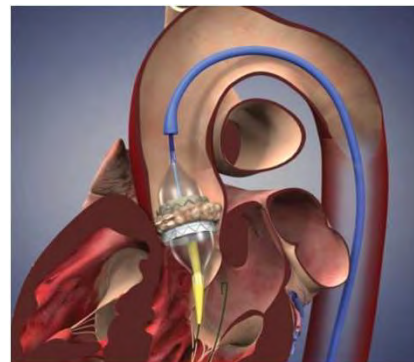
- ▶ There's a blockage in an artery that's not a coronary (leg & arm arteries, visceral arteries, renal arteries, carotid arteries)
  - ▶ Balloon (and maybe stent) it with much bigger devices than I use in the coronaries
- ▶ There's a pulmonary embolus
  - ▶ Put in a giant vacuum and suck it out
- ▶ There's an embolic stroke
  - ▶ Pull the clot out of the brain



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## A Coronary Operator's Take On Structural Intervention

- ▶ Make a large hole in the femoral artery or vein
- ▶ Put in a large device to fix the problem
  - ▶ Afib = Watchman
  - ▶ AS = TAVR
  - ▶ MR = Mitraclip
  - ▶ PFO / ASD / VSD = Occluder
- ▶ Close the hole
- ▶ Get coffee and repeat



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## What happens in the Modern cath lab?

- ▶ Hemodynamic studies
- ▶ Diagnostic coronary angiography
- ▶ **Percutaneous coronary intervention**
- ▶ **Cardiogenic shock treatment**
- ▶ Peripheral angiography and interventions
- ▶ Structural heart procedures

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## Cardiogenic Shock And Mechanical Circulatory Support

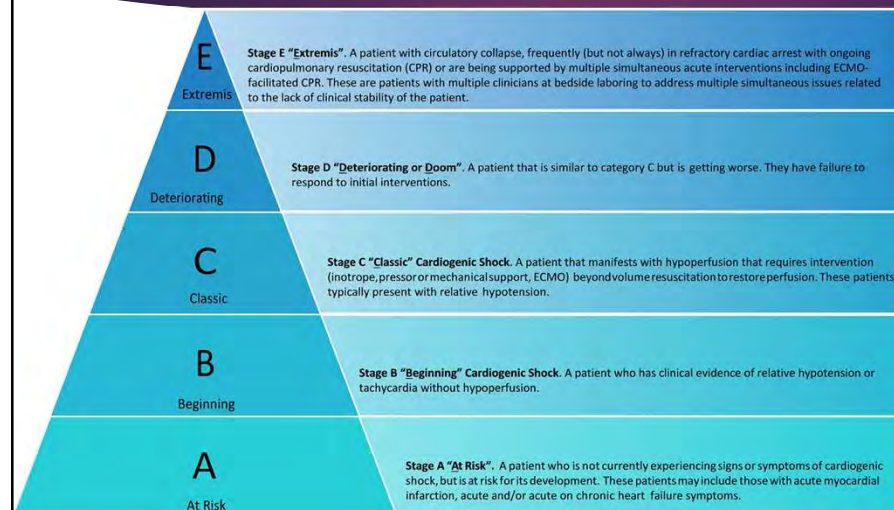
60

## Classic Definition

- ▶ Cardiogenic Shock: Inability of the heart to provide systemic perfusion
  - ▶ **Pump dysfunction**
    - ▶ Left sided (MI, myocarditis, decompensated cardiomyopathies)
    - ▶ Right Sided (PE, RV infarct)
  - ▶ Electrical
    - ▶ Bradycarrhythmias (Heart block with inadequate escape rhythm)
    - ▶ Tachycarrhythmias (VT, VF etc.)

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## Transition to Stages (SCAI 2021 Update)



Naidu SS, Baran DA, Jentzer JC, Hollenberg SM, van Diepen S, Basir MB, Grines CL, Diercks DB, Hall S, Kapur NK, Kent W, Rao SV, Samsky MD, Thiele H, Truesdell AG, Henry TD. SCAI SHOCK Stage Classification Expert Consensus Update: A Review and Incorporation of Validation Studies: This statement was endorsed by the American College of Cardiology (ACC), American College of Emergency Physicians (ACEP), American Heart Association (AHA), European Society of Cardiology (ESC), Association for Acute Cardiovascular Care (ACVC), International Society for Heart and Lung Transplantation (ISHLT), Society of Critical Care Medicine (SCCM), and Society of Thoracic Surgeons (STS) in December 2021. J Am Coll Cardiol. 2022 Mar 8;79(9):933-946. doi: 10.1016/j.jacc.2022.01.018. Epub 2022 Jan 31. PMID: 35115207.

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# Shock Stages Simplified

- ▶ A- Stable STEMI
- ▶ B- STEMI with pulmonary edema
- ▶ C- Sick STEMI on norepinephrine
- ▶ D- STEMI on 3 pressors
- ▶ E- STEMI getting CPR

The SCAI pyramid of cardiogenic shock classification <sup>1</sup>		Physical exam	Biochemical markers	Hemodynamics
<b>E</b>	<b>Extremis</b> A patient experiencing cardiac arrest with ongoing CPR and/or ECMO, being supported by multiple interventions.	Near pulselessness Cardiac collapse Metabolic acidosis Diffuse cyanosis	CPR (A-metric) pH < 7.2 Lactate > 8 mmol/L	No aPP without intubation PEA or refractory VT/VF Hypotension despite maximal support
<b>D</b>	<b>Deteriorating</b> A patient who fails to respond to initial interventions. Similar to category C but getting worse.	May include any of: Look unwell, panicked ashen, mottled, dusky cold, clammy Volume overload Extensive rales Killip class 3 or 4 NIV or MV Altered mental status Urine output <30 mL/h	Stage C and deteriorating  May include any of: Lactate ≥ 2 mmol/L Creatinine doubling > 50% drop in GFR Elevated LFTs Elevated BNP	Stage C and need for multiple pressors or TCS devices  SBP < 90 or MAP < 60 mmHg and need for drugs/device to maintain BP Cardiac Index < 2.2 L/min/mg PCWP > 18 mmHg RAP/PCWP ≥ 0.8 mmHg PAPI < 1.85 Cardiac power output ≤ 0.8 W
<b>C</b>	<b>Classic</b> A patient manifests with hypoperfusion that requires intervention (inotrope, pressor or TCS) beyond volum resuscitation to restore perfusion.	Elevated JVP Rales in lung fields No sign of peripheral hypoperfusion	Normal lactate acid Minimal renal function impairment Elevated BNP	SBP < 90 or MAP < 60 mmHg Pulse > 100 bpm Cardiac Index < 2.2 L/min/mg PA sat ≥ 65%
<b>B</b>	<b>Beginning</b> A patient who has clinical evidence of relative hypotension or tachycardia without hypoperfusion.	Normal JVP Normal physical exam	Normal lactate acid Normal renal function	Normal BP Cardiac Index ≥ 2.5 L/min/mg CVP < 10 mmHg PA sat ≥ 65%
<b>A</b>	<b>At risk</b> A patient who is not currently experiencing signs or symptoms of CS, but is at risk of developing CS.			

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## Case 2:

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## HPI & PMH

- ▶ 63 yo man p/w sudden onset chest pain to Springfield Hospital, Concern for STEMI
- ▶ HPI
  - ▶ Woke up 1 hour ago, crushing 7/10 substernal CP
  - ▶ Associated diaphoresis and dyspnea
- ▶ History:
  - ▶ 30+ pack year smoker (quit 1m ago)
  - ▶ Bipolar disorder
  - ▶ DM2

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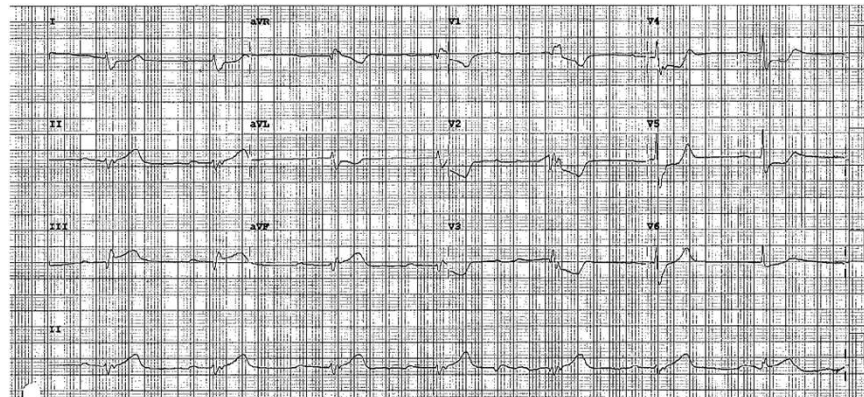
## Exam and EKG

### Focused Exam:

Vitals: AF, BP-60/30, HR: 35,  
O2: pulse ox not reading

Gen: Ashen appearing  
obese man in distress

CV: Slow rate, regular, S1S2  
very soft, no murmurs, Distal  
pulses not palpable



66

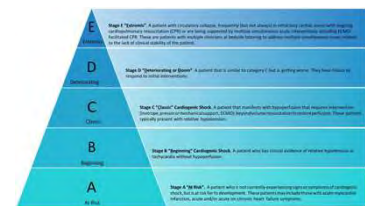
# Patient Comes to the Cath lab



- ▶ Exam from across the room
  - ▶ Vitals: BP 85/30, HR: 35
  - ▶ Monitor: 3<sup>rd</sup> deg. AVB
  - ▶ Drips: levophed @ 50, epi @ 20
  - ▶ Gen: Patient is awake and talking, and "pretty with it"
  - ▶ CV: He's grey, probably not perfusing too well
  - ▶ Pulm: He's on a lot of O2
  - ▶ Ext: Mottled

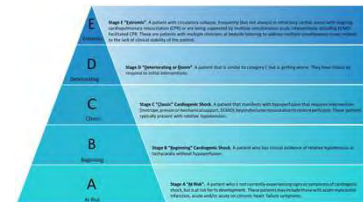
67

Stage	Description	Physical exam/bedside findings	Biochemical markers	Hemodynamics
A At risk	A patient who is not currently experiencing signs or symptoms of CS, but is at risk for its development. These patients may include those with large acute myocardial infarction or prior infarction acute and/or acute on chronic heart failure symptoms.	Normal JVP Lung sounds clear Warm and well perfused • Strong distal pulses • Normal mentation	Normal labs • Normal renal function • Normal lactic acid	Normotensive (SBP ≥ 100 or normal for pt.) If hemodynamics done • cardiac index ≥ 2.5 • CVP < 10 • PA sat ≥ 65%
B Beginning CS	A patient who has clinical evidence of relative hypotension or tachycardia without hypoperfusion.	Elevated JVP Rales in lung fields Warm and well perfused • Strong distal pulses • Normal mentation	Normal lactate Minimal renal function impairment Elevated BNP	SBP < 90 OR MAP < 60 OR > 30 mmHg drop from baseline Pulse ≥ 100 If hemodynamics done • cardiac index > 2.2 • PA sat ≥ 65%
C Classic CS	A patient that manifests with hypoperfusion that requires intervention (inotrope, pressor or mechanical support, including ECMO) beyond volume resuscitation to restore perfusion. These patients typically present with relative hypotension.	<b>May Include Any of:</b> Looks unwell Panicked Ashen, mottled, dusky Volume overload Extensive rales Killip class 3 or 4 BiPap or mechanical ventilation Cold, clammy Acute alteration in mental status Urine output < 30 mL/h	<b>May Include Any of:</b> Lactate ≥ 2 Creatinine doubling OR > 50% drop in GFR Increased LFTs Elevated BNP	<b>May Include Any of:</b> SBP < 90 OR MAP < 60 OR > 30 mmHg drop from baseline AND drugs/device used to maintain BP above these targets Hemodynamics • cardiac index < 2.2 • PCWP > 15 • RAP/PCWP ≥ 0.8 • PAPI < 1.85 • cardiac power output ≤ 0.6
D Deteriorating/ doom	A patient that is similar to category C but are getting worse. They have failure to respond to initial interventions.	<b>Any of stage C</b>	<b>Any of Stage C AND:</b> Deteriorating	<b>Any of Stage C AND:</b> Requiring multiple pressors OR addition of mechanical circulatory support devices to maintain perfusion
E Extremis	A patient that is experiencing cardiac arrest with ongoing CPR and/or ECMO, being supported by multiple interventions.	Near Pulselessness Cardiac collapse Mechanical ventilation Defibrillator used	<b>"Trying to die"</b> CPR (A-modifier) pH < 7.2 Lactate ≥ 5	No SBP without resuscitation PEA or refractory VT/VF Hypotension despite maximal support



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Stage	Description	Physical exam/bedside findings	Biochemical markers	Hemodynamics
A At risk	A patient who is not currently experiencing signs or symptoms of CS, but is at risk for its development. These patients may include those with large acute myocardial infarction or prior infarction acute and/or acute on chronic heart failure symptoms.	Normal JVP Lung sounds clear Warm and well perfused • Strong distal pulses • Normal mentation	Normal labs • Normal renal function • Normal lactic acid	Normotensive (SBP > 100 or normal for pt.) If hemodynamics done • cardiac index $\geq 2.5$ • CVP < 10 • PA sat $\geq 65\%$
B Beginning CS	A patient who has clinical evidence of relative hypotension or tachycardia without hypoperfusion.	Elevated JVP Rales in lung fields Warm and well perfused • Strong distal pulses • Normal mentation	Normal lactate Minimal renal function impairment Elevated BNP	SBP < 90 OR MAP < 60 OR > 30 mmHg drop from baseline Pulse $\geq 100$ If hemodynamics done • cardiac index $\geq 2.2$ • PA sat $\geq 65\%$
C Classic CS	A patient that manifests with hypoperfusion that requires intervention (inotrope, pressor or mechanical support, including ECMO) beyond volume resuscitation to restore perfusion. These patients typically present with relative hypotension.	<b>May Include Any of:</b> Looks unwell Panicked Ashen, mottled, dusky Volume overload Extensive rales Killip class 3 or 4 BiPap or mechanical ventilation Cold, clammy Acute alteration in mental status Urine output < 30 mL/h	<b>May Include Any of:</b> Lactate $\geq 2$ Creatinine doubling OR > 50% drop in GFR Increased LFTs Elevated BNP	<b>May Include Any of:</b> SBP < 90 OR MAP < 60 OR > 30 mmHg drop from baseline AND drugs/device used to maintain BP above these targets Hemodynamics • cardiac index < 2.2 • PCWP > 15 • RAP/PCWP $\geq 0.8$ • PAPI < 1.85 • cardiac power output < 0.6
D Deteriorating/ doom	A patient that is similar to category C but are getting worse. They have failure to respond to initial interventions.	<b>Any of stage C</b>	<b>Any of Stage C AND:</b> Deteriorating	<b>Any of Stage C AND:</b> Requiring multiple pressors OR addition of mechanical circulatory support devices to maintain perfusion
E Extremis	A patient that is experiencing cardiac arrest with ongoing CPR and/or ECMO, being supported by multiple interventions.	Near Pulselessness Cardiac collapse Mechanical ventilation Defibrillator used	<b>"Trying to die"</b> CPR (A-modifier) pH < 7.2 Lactate $\geq 5$	No SBP without resuscitation PEA or refractory VT/VF Hypotension despite maximal support



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## What Should I Do First?

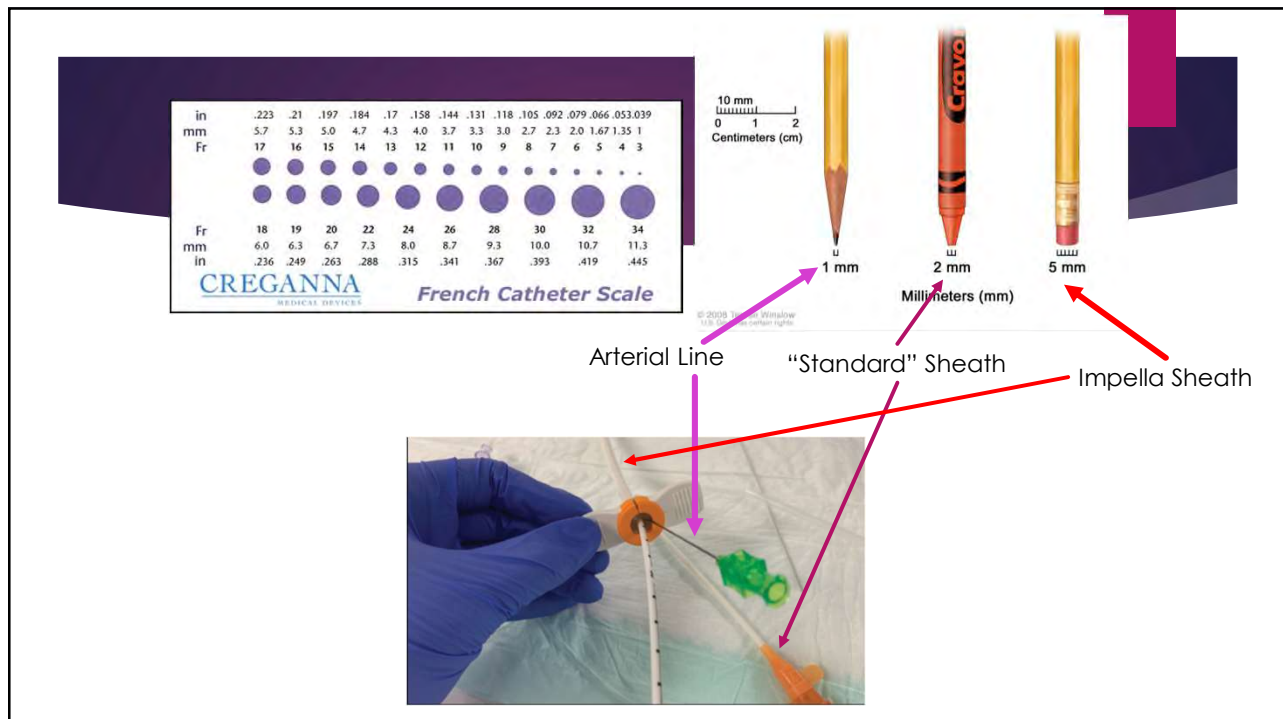
1. Access the radial artery, perform a coronary angiogram and proceed to PCI?
2. Access the femoral artery and insert an IABP?
3. Access the femoral vein and perform a right heart catheterization?
4. Access the femoral artery and insert an Impella?
5. Access the femoral vein and insert a temporary pacemaker?

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## What I Did

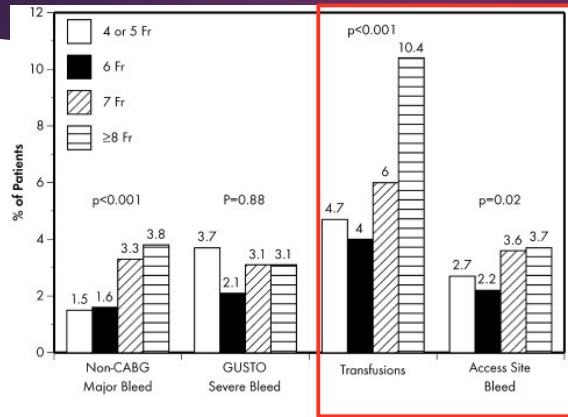
1. Access the radial artery, perform a coronary angiogram and proceed to PCI?
2. **Access the femoral artery and insert an IABP!**
3. Access the femoral vein and perform a right heart catheterization?
4. Access the femoral artery and insert an Impella?
5. **Access the femoral vein and insert a temporary pacemaker!**

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## Bleeding Risk By Femoral Sheath Size



Risk of major bleeding from  
A 14F Impella sheath: 17%

Cantor et al. 2007

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## Intra-aortic Balloon Pump

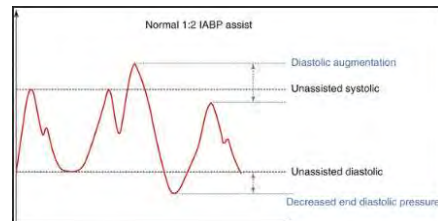
- ▶ Most commonly used form of MCS
- ▶ Catheter is a double lumen (7.5F / 8F) catheter
  - ▶ Polyethylene balloon at the distal end
  - ▶ Lumen 1: Fills balloon with helium
  - ▶ Lumen 2: Guidewire tracking and pressure transduction



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## Hemodynamic Effects of an IABP

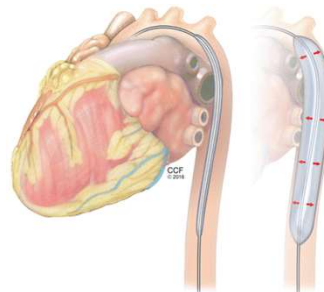
- ▶ Increases diastolic blood pressure
- ▶ Decreases afterload
- ▶ Decreases myocardial oxygen consumption
- ▶ Increases coronary artery perfusion
- ▶ Modestly Enhances cardiac output
- ▶ Ventricular unloading (modest) is partially offset by increase in MAP
- ▶ Patients need to have some level of LV fxn and electrical stability



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## Contraindications to IABP

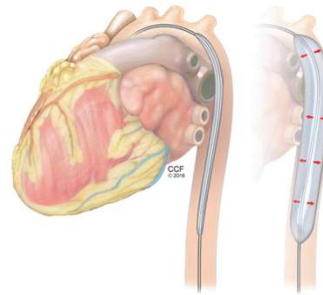
- ▶ Aortic regurgitation (more than mild)
  - ▶ IABP inflation worsens degree of AR
- ▶ Severe PAD or aortic disease
  - ▶ Risk of thromboembolism



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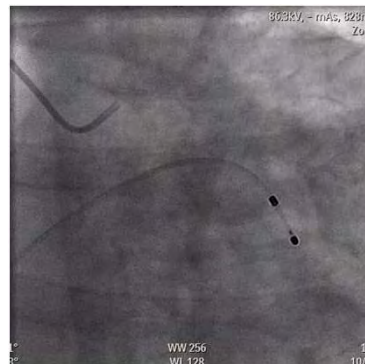
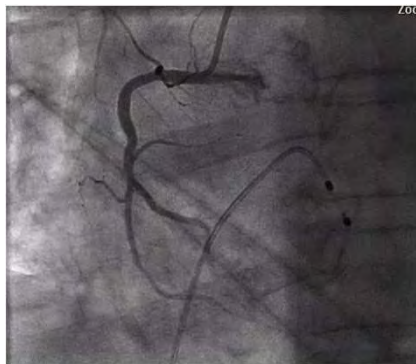
## Complications from IABP

- ▶ Vascular Injury
- ▶ Stroke
- ▶ Thrombocytopenia
  - ▶ Platelet deposition on IABP membrane
  - ▶ HIT
- ▶ Aortic / visceral artery trauma
  - ▶ Bowel ischemia
  - ▶ Atheroembolism
  - ▶ AKI



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## Initial Angiography



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## Uh Oh

- ▶ Patient develops PMVT
  - ▶ IABP unable to adequately assist
- ▶ Defibrillated successfully to NSR
  - ▶ BP now 40/20(Essentially PEA)
- ▶ CPR
- ▶ IABP removed & Impella CP Inserted
- ▶ Impella initiated, now with 3.5L flow



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## LV to Aorta Assist Devices

- ▶ Impella CP and 5.5 available at DHMC
- ▶ Nonpulsatile axial flow Archimedes screw pump
- ▶ Connects to a (14F and 23F) cannula
  - ▶ Contains:
    - ▶ Pump inlet
    - ▶ Pump outlet
    - ▶ Motor housing
    - ▶ Pump pressure monitor



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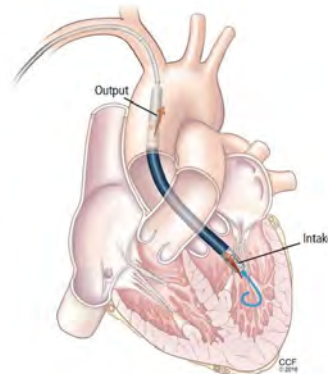
## Impella Insertion

- ▶ CP designed to be placed in the femoral artery
  - ▶ Impella CP can be inserted percutaneously (14F sheath)
  - ▶ 5.5 Requires a cardiac or vascular surgeon to sew a graft to the axillary artery
- ▶ Alternative access sites have been described (Subclavian, Transcaval and Axillary arteries)
- ▶ Axillary impella support has the possible advantage of longer term support

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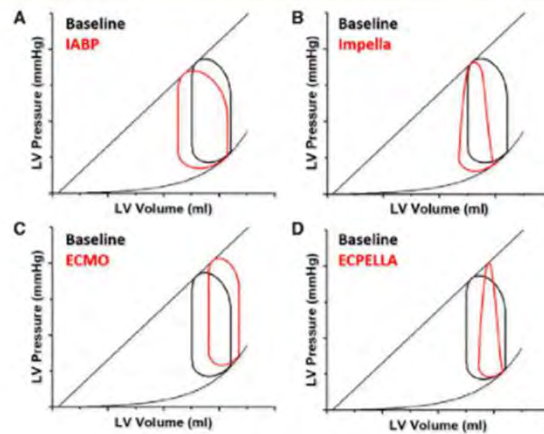
## Impella Hemodynamic Effects

- ▶ Pumps blood from the LV into the ascending aorta
  - ▶ 3-4L/min for CP
  - ▶ 6L/min for 5.5
- ▶ Reduces LV myocardial oxygen consumption and PCW pressure, increases MAP by increasing systemic perfusion



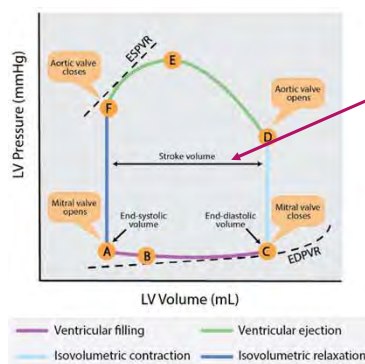
82

## Hemodynamics and PV Loops



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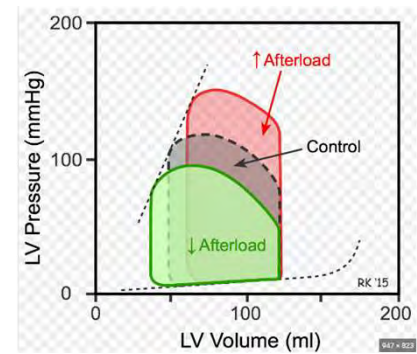
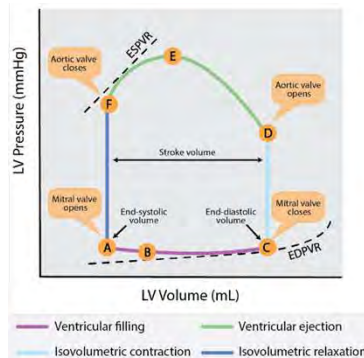
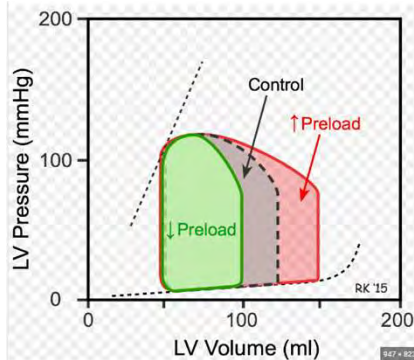
## Pressure Volume Loop Basics



This is the important line for us

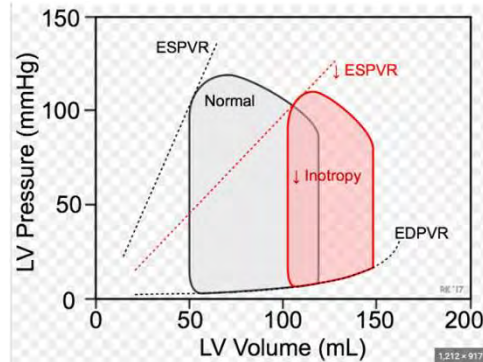
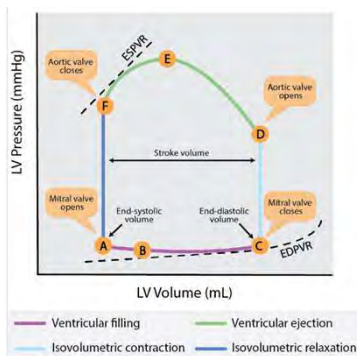
84

## Pressure Volume Loop Basics



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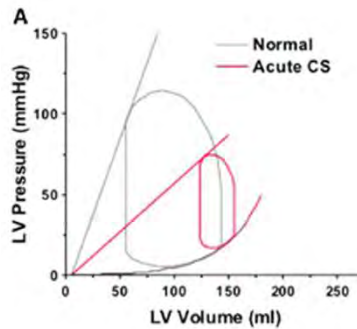
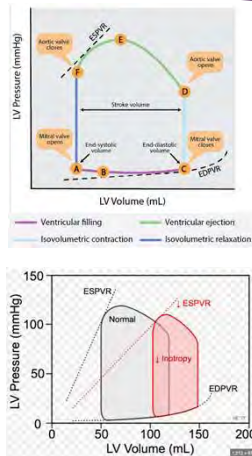
## Pressure Volume Loop Changes: Heart Failure



- Heart Failure =
  - ↓ Squeeze (↓ inotropy)
  - LV Compensates by ↑ preload (↑ LVEDP)
  - ↑ LVEDP will ↑ stroke volume

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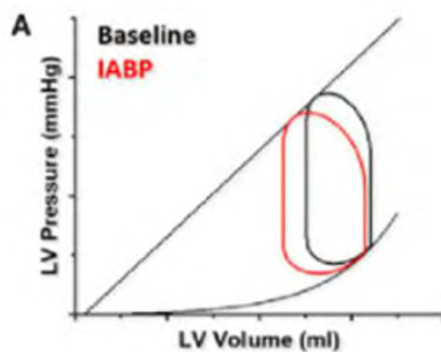
## Pressure Volume Loop Changes: Cardiogenic Shock



- Cardiogenic shock=
  - $\downarrow\downarrow\downarrow$  Squeeze ( $\downarrow\downarrow\downarrow$  inotropy)
  - LV  $\uparrow\uparrow\uparrow$  preload, but unable to  $\uparrow$  stroke volume significantly

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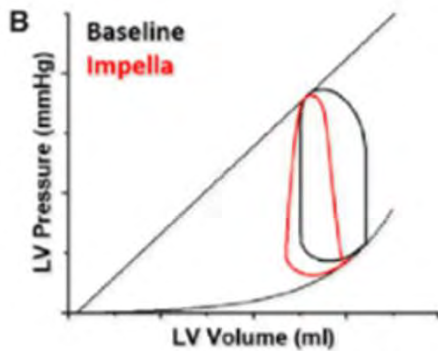
## Hemodynamics and PV Loops



- ▶ IABP
  - $\downarrow$  afterload
  - $\uparrow$  SV
  - $\uparrow$  Coronary perfusion

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## Hemodynamics and PV Loops



### ▶ Impella

- ↓ LVEDP
  - ↓ Wall tension
  - ↓ Ischemia
- ↑ Aortic pressure
  - ↑ Coronary perfusion

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## Contraindications to Impella

- ▶ Requires adequate RV function to supply LV preload
  - ▶ Ventricular arrhythmias are poorly tolerated
- ▶ Mechanical aortic valve
- ▶ LV thrombus
- ▶ Bleeding diatheses
- ▶ Severe PAD precluding pump placement



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## Impella Complications

- ▶ Vascular injury
  - ▶ Hematoma
  - ▶ AVF
  - ▶ RP bleed
- ▶ Limb ischemia
  - ▶ Can also be alleviated by placing an antegrade bypass sheath
- ▶ Hemolysis (up to 10% of patients)
  - ▶ AKI



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## For Practical Purposes

### IABP

- ▶ \$800
- ▶ Up and running <5 minutes
- ▶ Modest support at best
- ▶ "Small-ish" bore access

### Impella CP

- ▶ \$20,000
- ▶ Takes 10-15 minutes
- ▶ Significant LV unloading and support
- ▶ Large bore access

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# Back to Our First Case

21 Y/O MAN WITH SODIUM FLUORIDE POISONING AND REFRACTORY VF

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## ICU Course (05:28-09:10, Day 0)

- ▶ Patient neurologically intact and able to follow commands
  - ▶ Further sedated
- ▶ Three more episodes of PMVT
  - ▶ Each responded to a single 200J defibrillation
- ▶ Finally, a fourth episode of PMVT occurred that was refractory to more antiarrhythmics (procainamide, lidocaine and amiodarone) and six more defibrillations
- ▶ Now patient is brought to the cath lab for e-CPR
  - ▶ Accompanied by ~30 healthcare providers actively coding him
    - ▶ *Anesthesia & ICU attendings, RT's, many nurses, code team, ICU & cardiology fellows, IM residents*

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# Coordinating a Complex Critical Situation



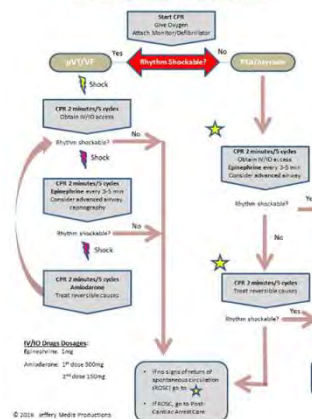
95

## Coordination Challenges in the Cath Lab

- ▶ Codes in the cath lab
  - ▶ Highly complicated situations
  - ▶ Varying team member expertise
  - ▶ Multiple "team leaders"
  - ▶ Complex procedures
  - ▶ Team experience level highly variable
- ▶ ACLS algorithms and training
  - ▶ Insufficient for the task at hand

### AHA ACLS Adult Cardiac Arrest Algorithm

Shout for Help/Activate Emergency Response



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## Recommended Practice

### U.S. Army Aircrew Coordination Training

*Helicopter-Safety Enhancement 22A  
Detection and Management of Risk Level Changes During Flight*

September 23, 2020

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## Guiding Principles

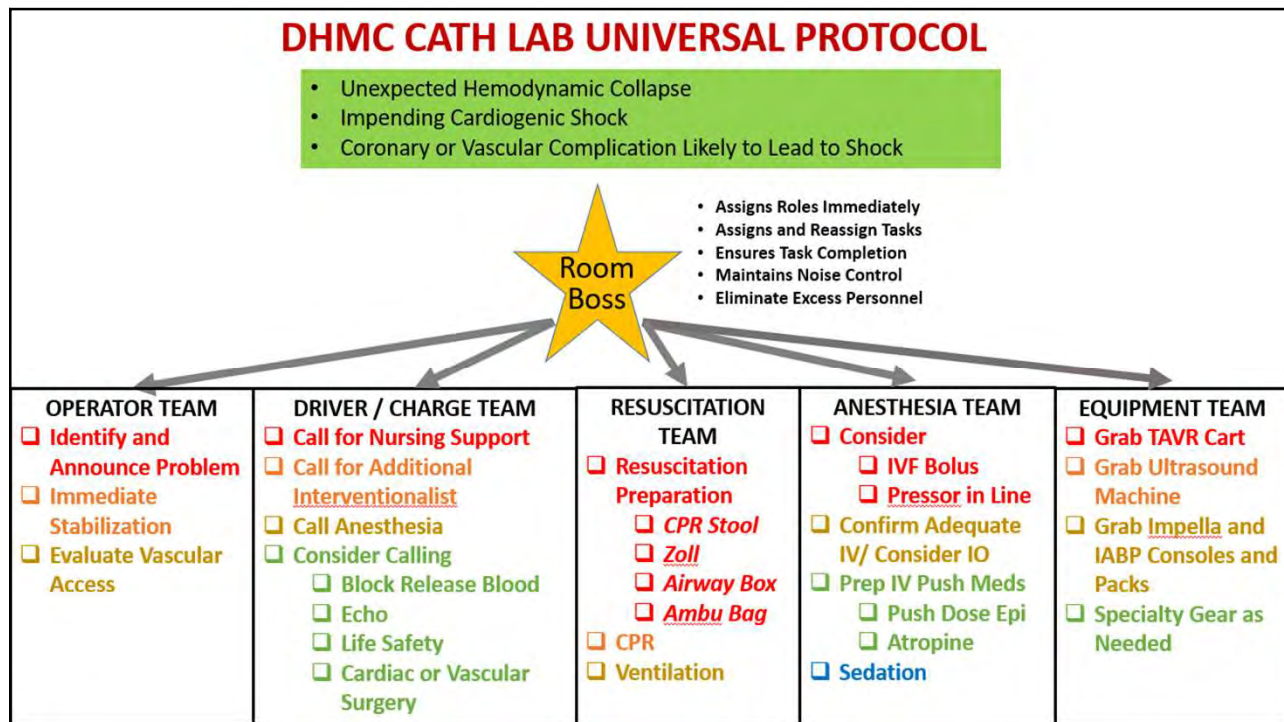
Crew Coordination Principles Combine to Produce Coordinated Objectives



### Translation to Cath Lab Codes

- ▶ Assignment of a room boss
  - ▶ Nurse or paramedic
  - ▶ **Not** the primary physician!
- ▶ Arriving personnel divided into teams
  - ▶ A room boss coordinates the teams
- ▶ Workload divided among teams
  - ▶ Room boss ensures task completion

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## Back To Our Case: Arrival in the Cath Lab

### Teams

- ▶ Room Boss: Cath lab nurse
- ▶ Operator team: Int. card, CT surg, perfusionist
- ▶ Driver/ Charge: cath lab nurses
- ▶ Resuscitation: critical care nurses and internal medicine residents
- ▶ Anesthesia: Cardiac anesthesiologist, MICU Physician, RT, Code team nurses
- ▶ Equipment Team: OR and Cath lab nurses

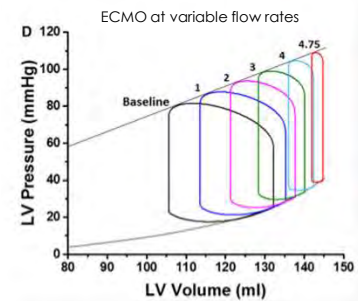
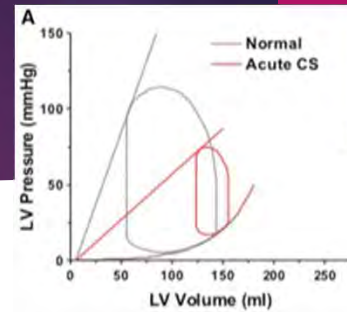
### Actions

- ▶ Room boss assigns everyone to a team, then kicks everyone else out into the hallway
- ▶ Resuscitation team continues CPR
- ▶ MICU and anesthesiology physicians lead ACLS
- ▶ Operator and equipment teams
  - ▶ Prep patient for E-CPR
  - ▶ Insert cannulae
  - ▶ VA ECMO started

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## ECMO Hemodynamics

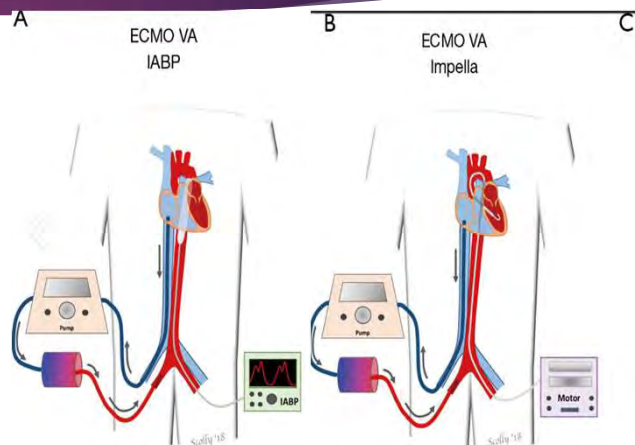
- ▶ Pressurized blood flows retrograde up the aorta from the arterial cannula
  - ▶ Provides systemic perfusion ☺
  - ▶ Increases Left Ventricular:
    - ▶ Afterload ☹
    - ▶ Wall tension ☹
    - ▶ Ischemia ☹
    - ▶ Distension ☹



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## Left Ventricular Venting

- ▶ Goal is to reduce the LV (end diastolic) pressure
- ▶ Optimal strategy unknown at this time
  - ▶ Medical therapy (inotropes / vasodilators)
  - ▶ Mechanical solutions
    - ▶ Atrial septostomy
    - ▶ Surgically inserted transapical catheter
    - ▶ Balloon Pump
    - ▶ Impella
    - ▶ Tandem Heart and LA-VA ECMO



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## Cath Lab Course (09:20-12:30, Day 0)

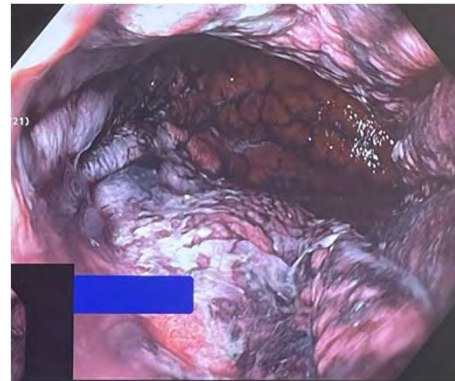


- ▶ Impella CP placed from the contralateral femoral artery to vent the LV
- ▶ Another attempt at 200J defibrillation was again unsuccessful
- ▶ Two defibrillators were then used simultaneously to deliver a 400J shock, which restored sinus rhythm
- ▶ Device / cannulae positions secured
- ▶ Patient transferred to the CCU

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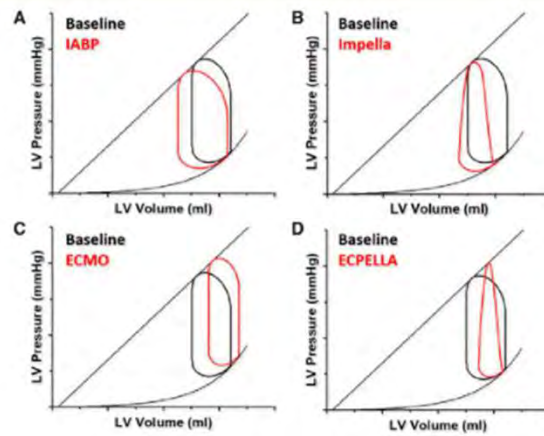
## Patient Course

- ▶ Stabilized on "Ecpella" support
  - ▶ Severe biventricular failure
- ▶ Transferred to Tufts for further advanced cardiac care
- ▶ Further multisystem organ failure
  - ▶ Developed AKI with anuria requiring HD
  - ▶ Developed necrosis of his stomach due to direct NaF toxicity
- ▶ 6 weeks supportive care
  - ▶ Recovered cardiac function
  - ▶ Kidney function normalized
  - ▶ Tolerating a normal diet
- ▶ Discharged to psychiatry and then outpatient counseling



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## Hemodynamics and PV Loops



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## Case 3

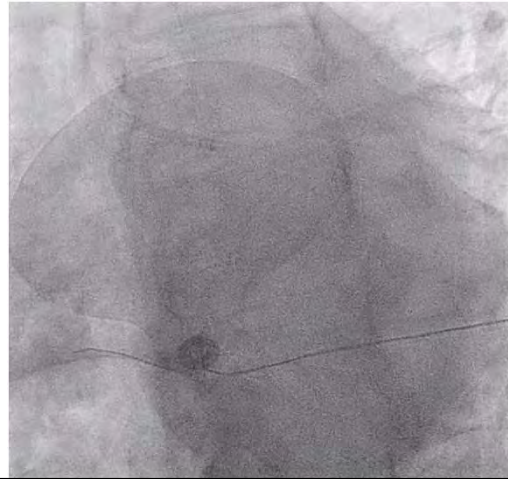
58 Y/O MAN P/W ANTERIOR STEMI AND PULMONARY EDEMA WITH A NORMAL BLOOD PRESSURE

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# Rapid Progression Through Shock Stages

The SCAI pyramid of cardiogenic shock classification<sup>1</sup>

	Physical exam	Biochemical markers	Hemodynamics
<b>E</b> <b>Extremis</b> A patient experiencing cardiac arrest with ongoing CPR and/or ECMO, being supported by multiple interventions.	Wax (pupils/retinae) Cardiac collapse Distended or red/purple Cyanosis (low)	SBP (<40mmHg) pH < 7.2 Lactate > 4.0 mmol/L	Do not attempt resuscitation EUA to determine if PVP Hypothermia? Aspart Bilirubin? Aspart?
<b>D</b> <b>Deteriorating</b> A patient who fails to respond to initial interventions, similar to category C but getting worse.	May include any of: Look unwell, panicked achy, mottled, dusky cold, clammy Volume overload Extensive rales Weg sign 3/4 or 4 NIV or MV Altered mental status Urine output <0.5 mL/h	Stage C and deteriorating  May include any of: Lactate ≥ 2 mmol/L Creatinine doubling > 50% drop in SBP Elevated LFTs Elevated BNP	Stage C and need for multiple pressors or TCS devices  SBP < 60 or MAP < 60 mmHg and need for drugs/egence to maintain BP Cardiac index < 2.2 L/min/m <sup>2</sup> PCWP > 18 mmHg PAP < 16d Cardiac power output < 0.8 W
<b>C</b> <b>Classic</b> A patient manifests with hypoperfusion that requires intervention (inotropic, pressor or TCS) beyond volume resuscitation to restore perfusion.	Flushed skin Rales in lung fields No sign of peripheral hypoperfusion	Normal lactate and Normal renal function impairment Elevated BNP	SBP < 90 or MAP < 60 mmHg Pulse < 100 bpm Cardiac index < 2.2 L/min/m <sup>2</sup> PA sat < 80%
<b>B</b> <b>Beginning</b> A patient who has clinical evidence of relative hypoperfusion or tachycardia without hypoperfusion.			Normal BP Cardiac index < 2.5 L/min/m <sup>2</sup> CVP < 10 mmHg PA sat > 80%
<b>A</b> <b>At risk</b> A patient who is not currently experiencing signs or symptoms of CS, but is at risk of developing CS.	Normal JVP Normal physical exam	Normal lactate and Normal renal function	



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# Cardiogenic Shock

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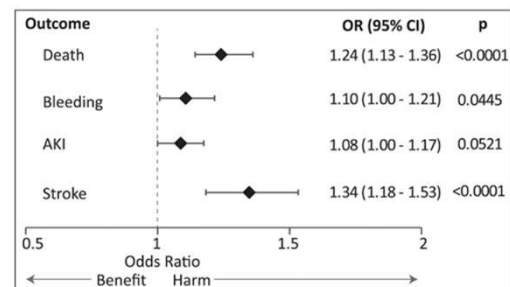
## Prognosis

- ▶ Mortality is still 50% for acute myocardial infarction complicated by cardiogenic shock
- ▶ Unchanged over the past 20 years despite
  - ▶ Advances in mechanical circulatory support
  - ▶ Improvements in STEMI systems
  - ▶ Improvements in PCI techniques

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## Could MCS Be Causing More Harm?

- ▶ Amin et al. demonstrated that Impella use is increasing for treatment of cardiogenic shock
  - ▶ Impella associated with increased costs, bleeding, AKI, stroke and death
  - ▶ Retrospective, propensity matched data from 2004-2016
    - ▶ Impossible to account for all confounders



Amin AP, Spertus JA, Curtis JP, Desai N, Masoudi FA, Bach RG, McNeely C, Al-Badarin F, House JA, Kulkarni H, Rao SV. The Evolving Landscape of Impella Use in the United States Among Patients Undergoing Percutaneous Coronary Intervention With Mechanical Circulatory Support. *Circulation*. 2020 Jan 28;141(4):273-284. doi: 10.1161/CIRCULATIONAHA.119.044007. Epub 2019 Nov 17. PMID: 31735078.

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## Thoughts on Impella Mortality

- ▶ Dr. Amin (the pessimist):
  - ▶ Cardiogenic shock patients are incredibly sick
  - ▶ Impella is associated with high rates of bleeding complications
  - ▶ These patients lack the reserve to tolerate these complications
  - ▶ Any benefit with regards to cardiac improvements are negated by these complications
- ▶ Me (the optimist):
  - ▶ Since the publication of this data, there have been extensive efforts to improve complication rates
    - ▶ Better sheaths, optimized devices, preclosing the access site, driving sutures, etc.
  - ▶ Best practice guidelines have now been published and complication rates are decreasing (anecdotally)
  - ▶ This will allow for reduced complications and for us to realize the cardiac benefits of MCS in another

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## Case 4

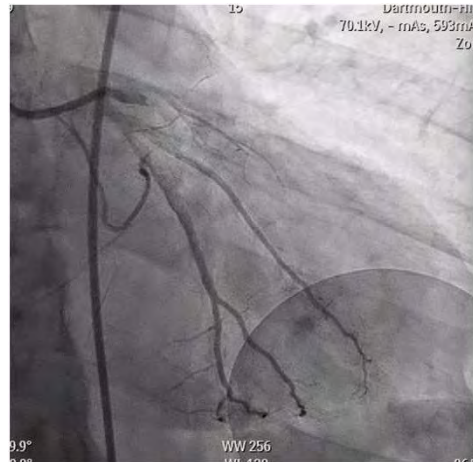
WHEN THE IMPELLA CP ISN'T ENOUGH

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## Late Presenting Anterior STEMI

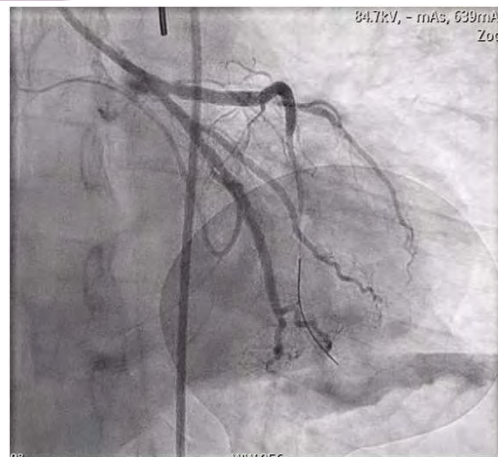
- ▶ 50 y/o man presented 3 days after chest pain started in cardiogenic shock
- ▶ Found to have anterior STEMI
- ▶ Still having chest pain



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## Microvascular Obstruction / "No-Reflow"

- ▶ IABP inserted
- ▶ Optimal treatment of the LAD
  - ▶ Ballooned
  - ▶ Thrombus aspirated
  - ▶ Stents deployed
  - ▶ Intracoronary vasodilators administered
- ▶ Poor final result (TIMI 1 flow)



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## Over the Next Two Days

- ▶ Poor perfusion
  - ▶ Worsening AKI requiring CVVH
  - ▶ Relative hypotension requiring inotropes / vasopressors
- ▶ Complete heart block
  - ▶ Tx'ed with temporary pacemaker
- ▶ Sent back to the cath lab for upgrade of MCS

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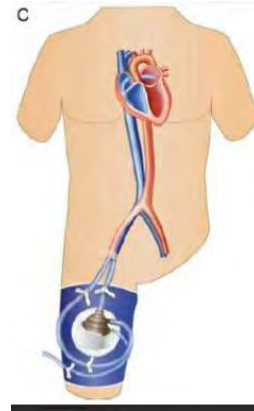
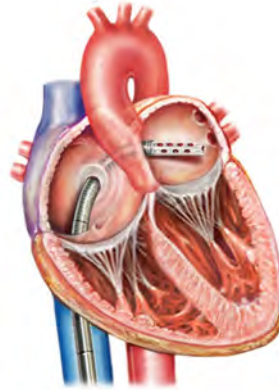
## Upgraded Left Sided MCS Options...

- ▶ Impella CP
- ▶ Tandem Heart
- ▶ VA ECMO
- ▶ Impella 5.5

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## Left Atrial to Aorta Assist Devices

- ▶ One device commercially available: Tandem Heart
- ▶ Pumps blood from the left atrium to the iliofemoral system
- ▶ Provides ~5L of Flow
- ▶ Not currently utilized at DHMC



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## Peripheral Angiography

Our Patient



A Normal Patient



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## Upgraded Left Sided MCS Options...

- ▶ Impella CP\*\*
  - ▶ Tandem Heart
  - ▶ VA ECMO\*
  - ▶ **Impella 5.5**
- Require an iliofemoral system capable of supporting large bore access

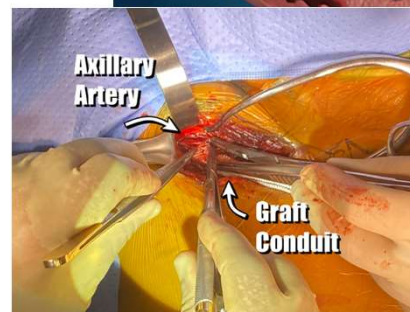
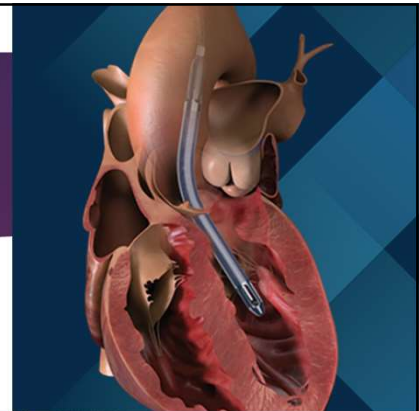
\*Open surgical central cannulation allows for ECMO in diseased iliofemoral systems

\*\*Percutaneous axillary and transcaval access allows for impella CP in diseased iliofemoral systems

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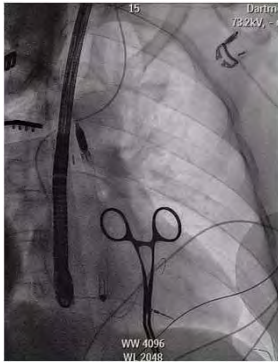
## Impella 5.5

- ▶ Open incision with the sheath inserted through a graft sewn into the axillary artery
- ▶ Same concept as Impella CP (Archimedes screw)
- ▶ Provides up to 6L / min flow



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## Back To Our Case



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## After the Cath Lab

- ▶ Stabilized, weaned off of vasopressors
  - ▶ Lactic acidosis improved
- ▶ Transferred to Tufts Medical Center for ongoing advanced care
  - ▶ Extubated and started PT / ambulating with the impella in place
  - ▶ Kidneys recovered and CVVH discontinued
- ▶ Discharged after 1 month
- ▶ Started on medical therapy for heart failure
  - ▶ EF recovered to 45%
- ▶ Complete and full recovery of functional status

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# What About The Right Heart?

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## Cardiogenic Shock: Right Heart Failure

- ▶ Challenging to diagnose
  - ▶ Imaging (echo) of the right ventricle has limitations
  - ▶ Hemodynamic studies can sometimes be misleading
- ▶ Etiologies of acute RV failure
  - ▶ RV Pump dysfunction
    - ▶ RV Infarction
    - ▶ Myocarditis
  - ▶ Excessive RV Afterload
    - ▶ Pulmonary embolus
    - ▶ Pulmonary hypertension
    - ▶ LV failure

**Table 1. Hemodynamic Formulas to Assess Right Ventricular Function**

Hemodynamic Formulas to Assess RV Function		
Cardiac filling pressures	RAP-PCWP	>0.03 (RVF after LVAD) <sup>13</sup> -0.88 (RVF in acute MI) <sup>23</sup>
PA pulsatility index (PAPI)	(PASP-PADP)/RAP	<1.85 (RVF after LVAD) <sup>13</sup> <1.0 (RVF in acute MI) <sup>23</sup>
Pulmonary vascular resistance	mRAP-PCWP/CO	>3.8 (RVF after LVAD) <sup>13</sup>
Transpulmonary gradient	mRAP-PCWP	Undetermined <sup>23</sup>
Diastolic pulmonary gradient	PADP-PCWP	Undetermined <sup>23,24</sup>
RV stroke work	(mRAP-RAP)×SV/0.0136	<15 (RVF after LVAD) <sup>13</sup> <10 (RVF after acute MI) <sup>23</sup>
RV stroke work index	(mRAP-RAP)/SV index	<0.3-0.5 (RVF after LVAD) <sup>13,21</sup>
PA compliance	SV/(PASP-PADP)	<2.5 (RVF in chronic heart failure) <sup>24</sup>
PA elastance	PASP/SV	Undetermined <sup>23</sup>

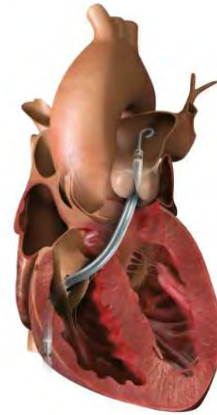
CO indicates cardiac output; LVAD, left ventricular assist device; MI, myocardial infarction; mRAP, mean pulmonary artery pressure; PA, pulmonary artery; PADP, pulmonary artery diastolic pressure; PASP, pulmonary artery systolic pressure; PCWP, pulmonary capillary wedge pressure; RAP, right atrial pressure; RV, right ventricular; RVF, right ventricular failure; and SV, stroke volume.

Kapur NK, Esposito ML, Bader Y, Morine KJ, Kiernan MS, Pham DT, Burkhardt D. Mechanical Circulatory Support Devices for Acute Right Ventricular Failure. *Circulation*. 2017 Jul 18;136(3):314-326. doi: 10.1161/CIRCULATIONAHA.116.025290. PMID: 28716832.

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## IVC to PA Assist Device (Impella RP)

- ▶ Inflow: Right atrium
- ▶ Outflow: Pulmonary artery
  - ▶ Direct augmentation of PA flow
  - ▶ Reduced RV preload
- ▶ Increases cardiac output
  - ▶ 2-4 L/min of support

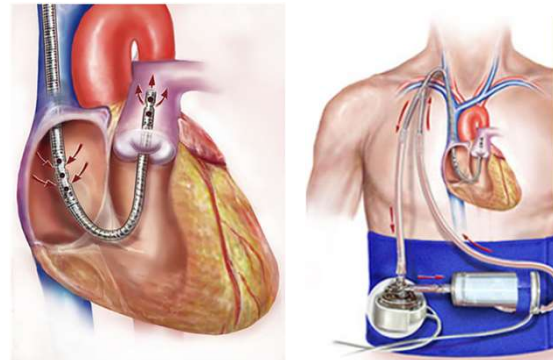


M.B. Anderson, J. Goldstein, C. Milano, *et al.*  
**Benefits of a novel percutaneous ventricular assist device for right heart failure: The prospective RECOVER RIGHT study of the Impella RP device**  
*J Heart Lung Transplant*, 34 (2015), pp. 1549-1560

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## Protek Duo

- ▶ Inflow: Right atrium
- ▶ Outflow: Pulmonary artery
  - ▶ Direct augmentation of PA flow
  - ▶ Reduced RV preload
- ▶ Increases cardiac output
  - ▶ 2-4 L/min of support



Kapur NK, Esposito ML, Bader Y, Morine KJ, Kiernan MS, Pham DT, Burkhardt D. Mechanical Circulatory Support Devices for Acute Right Ventricular Failure. *Circulation*. 2017 Jul 18;136(3):314-326. doi: 10.1161/CIRCULATIONAHA.116.025290. PMID: 28716832.

126

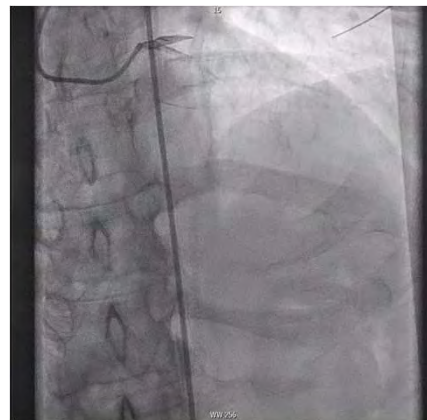
# Case 5- 9/2022

LET'S PRACTICE WHAT WE LEARNED

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## Undifferentiated Shock

- ▶ 65 y/o woman w hx of DM2 and HTN p/w COVID-19 and undifferentiated shock
  - ▶ BP 75/40 despite 3L IVF
  - ▶ CTA chest shows no dissection or PE
  - ▶ EKG with Sinus tachycardia and diffuse T wave inversions
  - ▶ TTE shows LVEF of 15% (global hypokinesia) and diminished RV function
  - ▶ Started on 30 mcg/min norepinephrine
  - ▶ Referred to cath lab due to concerns for MI



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## Invasive Hemodynamics

Left Heart Pressures						
Resting	Sys.	Dias.	EDP	a	v	m
Ao	68	41				51
LV	68		32			
LA						

Right Heart Pressures						
	Sys.	Dias.	EDP	a	v	m
RA				18	18	14
RV	25		17			
PA	25	20				22
PC				23	21	21

Hemodynamics Profile	
Resting	Profile 1
VO <sub>2</sub>	
Cardiac Output:	2.33
Cardiac Index:	1.19
TSR	1,751
SVR	1,270
TPR	755
PVR	34
Technique(s):	Estimated Fick

Pressure Comments (limit 40 lines)

Shock Hemodynamics

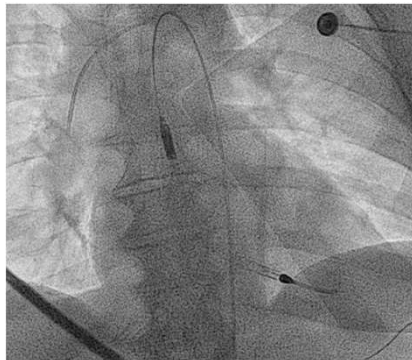
Cardiac Power Output:	0.19
Cardiac Power Index:	0.10
PAPI:	0.36
RAP/PCWP Index:	0.67

**Is this left, right or biventricular failure?**

- ▶ Right sided preload (RA pressure)
  - ▶ High
- ▶ Right sided contractility (PAPI)
  - ▶ Very Low
- ▶ Right sided afterload (PVR)
  - ▶ Normal
- ▶ Left sided preload (PCW / LVEDP)
  - ▶ High
- ▶ Left sided contractility (CPO & CO\*)
  - ▶ Very Low
- ▶ Left sided afterload (SVR)
  - ▶ Normal

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## Step 1: Impella CP Insertion



### Hemodynamics after Impella

- ▶ Right sided preload (RA pressure: 20)
  - ▶ High
- ▶ Right sided contractility (PAPI: 0.2)
  - ▶ Very Low
- ▶ Right sided afterload (PVR: 1)
  - ▶ Normal
- ▶ Left sided preload (PCW / LVEDP: 11)
  - ▶ Low
- ▶ Left sided contractility (CPO 0.3 & CO 3.1)
  - ▶ Very Low
- ▶ Left sided afterload (SVR: 1200)
  - ▶ Normal

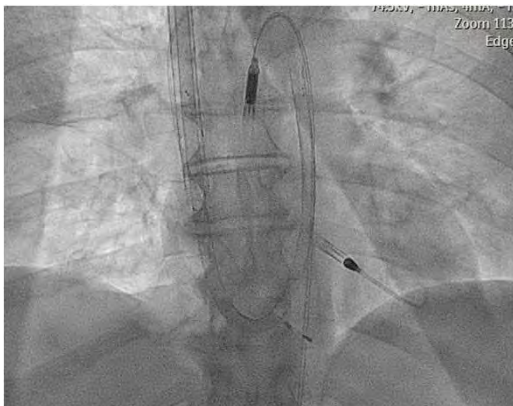
130

## Transferred to CVCC

- ▶ Persistent lactic acidosis
- ▶ Worsening AKI
- ▶ Multiple Impella suction alarms
  - ▶ More IVF administered to optimize biventricular preload
- ▶ TTE demonstrating minimal RV function and hyperdynamic LV
- ▶ Brought back to the cath lab for RV support

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## Step 2: Protek Duo Insertion



### Hemodynamics after Protek Duo

- Right sided preload (RA pressure: 5)  
Normal
- Right sided contractility  
Unable to measure with continuous flow
- Right sided afterload (PVR: 1)  
Normal
- Left sided preload (PCW / LVEDP: 15)  
Normal
- Left sided contractility (CPO 0.8 & CO 6)  
Normal
- Left sided afterload (SVR: 1200)  
Normal

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## Hospital Course

- ▶ Clinically diagnosed with viral myocarditis from COVID-19
  - ▶ Viral prodrome, +COVID-19 PCR, heart block & fulminant biventricular failure
- ▶ Underwent pacemaker implantation for complete heart block
- ▶ Biventricular MCS for 5 days with inotropes
  - ▶ Slowly weaned down
- ▶ Impella and protek removed at day 5
- ▶ Biventricular function normalized (LVEF 75% and RV function normal)
- ▶ Medications optimized
- ▶ Discharged home in 3 weeks
- ▶ Full recovery

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## That's Cardiogenic Shock & Mechanical Circulatory Support

QUESTIONS?

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## What happens in the Modern Cath Lab?

- ▶ Hemodynamic studies
- ▶ Diagnostic coronary angiography
- ▶ Percutaneous coronary intervention
- ▶ Cardiogenic shock treatment
- ▶ Peripheral angiography and interventions
- ▶ Structural heart procedures

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## Percutaneous coronary Intervention (PCI)

CORONARY ANGIOPLASTY, ATHERECTOMY AND STENTING

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## Treatment Options For Coronary Disease

- ▶ Optimal Medical Therapy (OMT) alone:
  - ▶ Antiplatelet agent: aspirin
  - ▶ Aggressive lipid management: statins, ezetimibe, PCSK9 inhibitors, etc.
  - ▶ Antianginal therapy
    - ▶ Beta blockers
    - ▶ Calcium channel blockers
    - ▶ Long acting nitroglycerine
    - ▶ Ranolazine
- ▶ Optimal Medical Therapy and Revascularization
  - ▶ Coronary Artery Bypass Grafting (CABG)
  - ▶ Percutaneous Coronary Intervention (PCI)

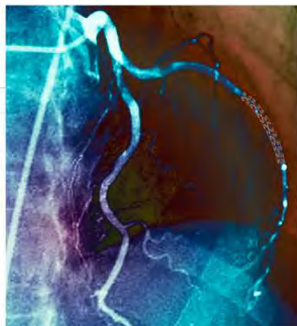
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## Why Bother to Revascularize Patients at All?

The New York Times

### *Surgery for Blocked Arteries Is Often Unwarranted, Researchers Find*

Drug therapy alone may save lives as effectively as bypass or stenting procedures, a large federal study showed.



The Washington Post  
*Democracy Dies in Darkness*

Stents and bypass surgery are no more effective than drugs for stable heart disease, highly anticipated trial results show

### TCT: Stent No Better Than Sham for Stable Angina in ORBITA

— No symptom relief beyond placebo effect in first sham-controlled trial

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## Revascularization According to the Guidelines

### ACC/AHA/SCAI CLINICAL PRACTICE GUIDELINE

#### **2021 ACC/AHA/SCAI Guideline for Coronary Artery Revascularization: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines**

Jennifer S. Lawton, MD, FAHA, Chair, Jacqueline E. Tamis-Holland, MD, FAHA, FACC, FSCAI, Vice Chair, Sripal Bangalore, MD, MHA, FACC, FAHA, FSCAI, Eric R. Bates, MD, FACC, FAHA, Theresa M. Beckie, PhD, FAHA, James M. Bischoff, MEd, John A. Bittl, MD, FACC, Mauricio G. Cohen, MD, FACC, FSCAI, J. Michael DiMaio, MD, Creighton W. Don, MD, PhD, FACC, Stephen E. Fries, MD, FACC, Mario F. Gaudino, MD, PhD, MSCE, FACC, FAHA, Zachary D. Goldberger, MD, FACC, FAHA, Michael C. Grant, MD, MSE, Jang B. Jaswal, MS, Paul A. Kurlansky, MD, FACC, Roxana Mehran, MD, FACC, Thomas S. Metkus Jr, MD, FACC, Lorraine C. Nnacheta, DrPH, MPH, Sunil V. Rao, MD, FACC, Frank W. Sellke, MD, FACC, FAHA, Garima Sharma, MD, FACC, Celina M. Yong, MD, MBA, MSc, FSCAI, FACC, FAHA, and Brittany A. Zwischenberger, MD

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## Revascularization

- ▶ The guideline committee reviewed all available evidence
  - ▶ 1165 studies reviewed by a panel of experts
- ▶ Stratified patients into two populations
  - ▶ Stable ischemic heart disease
  - ▶ Acute coronary syndromes

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## Revascularization Guidelines

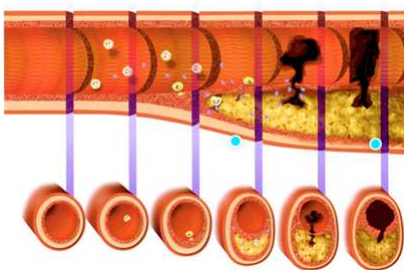
- ▶ According to the guidelines, the goals for revascularization differ for the two groups
  - ▶ ACS Patients: Improve mortality, LV function and symptoms
  - ▶ Stable angina: Predominantly improve symptoms



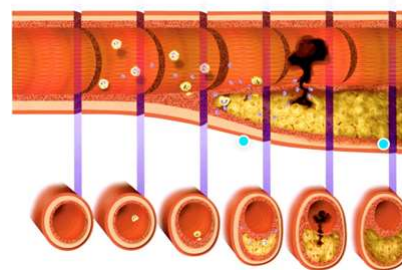
141

## Two Distinct Phenotypes of Coronary Atherosclerosis

ACS (STEMI & NSTEMI)

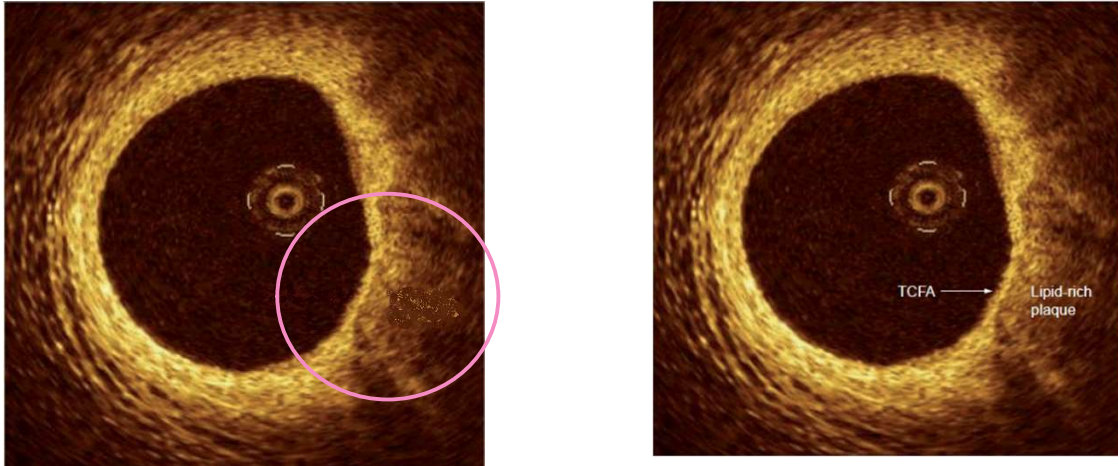


SIHD



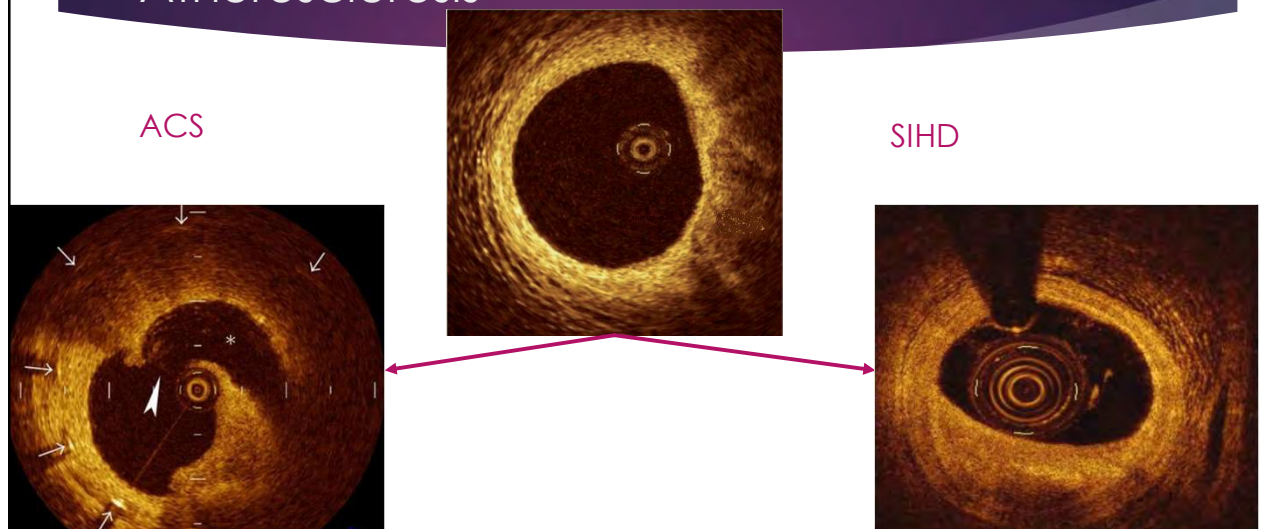
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## Atherosclerotic Plaque Optical Coherence Tomography



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## Two Distinct Phenotypes of Coronary Atherosclerosis



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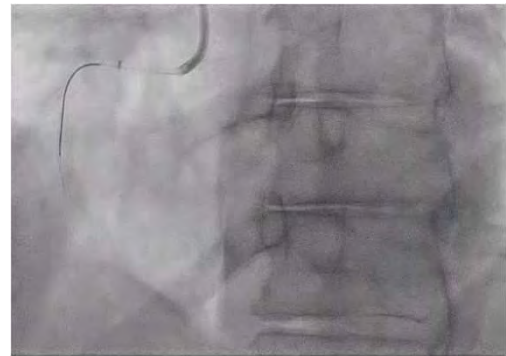
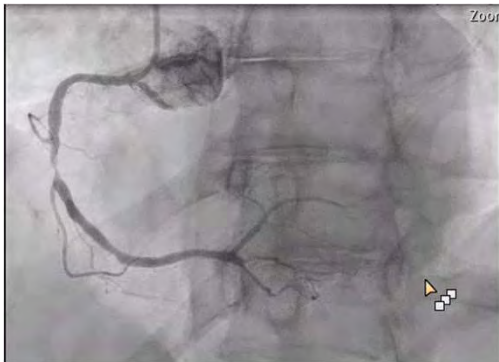


## My Personal Practice Pattern

- ▶ Patients should be offered coronary revascularization if they:
  - ▶ Present with ACS
  - ▶ Have a large area of ischemia
  - ▶ Have symptoms that are not controlled with antianginal medications
    - ▶ Reduce their activity level
    - ▶ Push through angina to complete activities
  - ▶ Don't want to take or have significant side effects from antianginal medications
- ▶ Revascularization modality (CABG vs PCI) is tailored to the patient

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## Diagnostic Angiography and PCI of a Simple Lesion Today



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## How to Stent a Coronary Artery

- ▶ Simple procedure
  - ▶ 10 minutes to take pictures
  - ▶ 25 minutes to stent the RCA
- ▶ Low risk
  - ▶ 0.5% to 1% chance of major complication
- ▶ Patient discharged home 4 hours later

149

## CABG: The Gold Standard for Revascularization

150

## CABG Is Wonderful

- ▶ CABG allows the operator to ignore all of the proximal obstacles and the complexities of the lesion itself
- ▶ Also allows operator to address valvular disease at the same time
- ▶ CABG achieves complete revascularization of multivessel coronary artery disease is more reliably achieved than PCI (ART Study)
- ▶ CABG improves mortality when there is multivessel CAD and severe LV dysfunction compared to medical (STICH, CASS and VA CABG COOP Studies)
- ▶ Mortality rates for CABG are 1% for the lowest risk patients
- ▶ Bypass graft patency at 5 years varies by graft choice (95% for LIMA & 80% for SVG's)

van den Brand M.J, Rensing B.J, Morel M.A, Foley D.P, de Valk V, Breeman A, Suryapranata H, Haalebos M.M, Wijns W, Wellens F, Balcon R, Magee P, Ribeiro E, Buffolo E, Unger F, Serruys P.W. The effect of completeness of revascularization on event-free survival of one year in the ARTS trial. J Am Coll Cardiol. 2002 Feb 20;39(4):559-64. doi: 10.1016/s0735-1097(01)01785-5. PMID: 11849851.

151

## CABG Continues to Evolve and Improve

- ▶ Minimally invasive techniques including robotic LIMA
- ▶ Total arterial revascularization (LIMA, RIMA and radial artery grafts)
  - ▶ Improved patency rates compared to SVG's
- ▶ Off pump "beating heart" CABG

152

## CABG Does Have Some Issues

- ▶ Mortality: 1-5%
- ▶ Noncardiac complications
  - ▶ Hemorrhage requiring reoperation (5%)
  - ▶ Stroke (6%)
  - ▶ Cognitive dysfunction (up to 80%)
  - ▶ Sternal infection & mediastinitis (1-3%)
  - ▶ Graft harvest site complications (18%)
  - ▶ Acute kidney injury (3%)
- ▶ Cardiac Complications
  - ▶ Perioperative MI (1-4%)
  - ▶ Early SVG occlusion (5-10%)
  - ▶ Cardiogenic (5%) & vasodilatory shock
  - ▶ Ventricular arrhythmias (1-3%)
  - ▶ Atrial fibrillation (15-40%)
  - ▶ Pericarditis (10-22%)
- ▶ Typical Recovery:
  - ▶ 5 days in hospital
  - ▶ 6-12 weeks of cardiac rehab
  - ▶ Full recovery: 3 months

153

How Do You Treat Patients with Indications for Revascularization that Should Not or Do not Want to Undergo CABG?

154

# “Complex High Risk Interventional Procedures in Indicated Patients”

CHIP PCI

155

“

Patients are referred to CABG  
because we don't think we can  
do a good enough job with PCI

”

AARON GRANTHAM, MD

156

## What's Changed Over the Past 20 Years...

- ▶ **Equipment has improved**
  - ▶ Better fluoroscopy
  - ▶ Intravascular imaging
  - ▶ Better catheters
  - ▶ Purpose built wires
  - ▶ More deliverable balloons
  - ▶ Calcium modification now available
  - ▶ Thinner stents
- ▶ **Techniques have improved**
  - ▶ Equipment delivery techniques
  - ▶ Modern bifurcation techniques
  - ▶ Hybrid algorithm for CTO
  - ▶ Image guided PCI optimization techniques
- ▶ **Medications have improved**
  - ▶ Antiproliferative drug polymers on stents
  - ▶ Antiplatelet Therapy

157

## What Lesions are Challenging For the Interventionalist?

- ▶ **Traditionally**, less likely to get an optimal result
  - ▶ Bifurcations
  - ▶ Left Main Disease
  - ▶ Calcified Lesions
  - ▶ Long Lesions with no stent landing zone
  - ▶ Chronic Total Occlusions
  - ▶ In Stent Restenosis
- ▶ **Modern Techniques** allow us to treat these lesions in the cath lab

158

## Why is CHIP Necessary?

- ▶ Anatomic and clinical complexity of patients with CAD is increasing in the US and around the world
- ▶ As patient complexity increases, procedural challenges of revascularization and the risk of complications increase
- ▶ Give patients the choice to undergo options for revascularization that are less invasive than CABG

Borhick AE, Epps KC, Seiber F, et al. Five-year follow-up of patients treated for coronary artery disease in the face of an increasing burden of co-morbidity and disease complexity (from the NHLBI dynamic registry). *Am J Cardiol.* 2014; 113(4): 573- 579.

159

## What is CHIP



- ▶ Lack of formal definitions:
  - ▶ "Complex CAD"
  - ▶ "High Risk CAD"
- ▶ Encompasses patients with:
  - ▶ Decompensated hemodynamics
  - ▶ Severe comorbidities
  - ▶ Complex lesions that will require extensive procedural work

Riley, RF, Henry, TD, Mahmud, E, et al. SCAI position statement on optimal percutaneous coronary interventional therapy for complex coronary artery disease. *Catheter Cardiovasc Interv.* 2020; 96: 346– 362. <https://doi.org/10.1002/ccd.28994>

160



“

Every interventional cardiologist  
thinks they do complex and high  
risk PCI

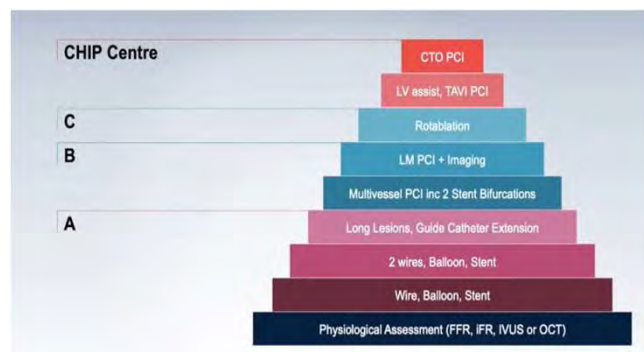
”

BILL LOMBARDI, MD

161

## Training for CHIP PCI

- ▶ Following interventional cardiology fellowship there are two pathways
  - ▶ One year specific CHIP-CTO fellowship
  - ▶ On the job training
    - ▶ On site proctoring
    - ▶ Intensive courses
    - ▶ Continued mentorship

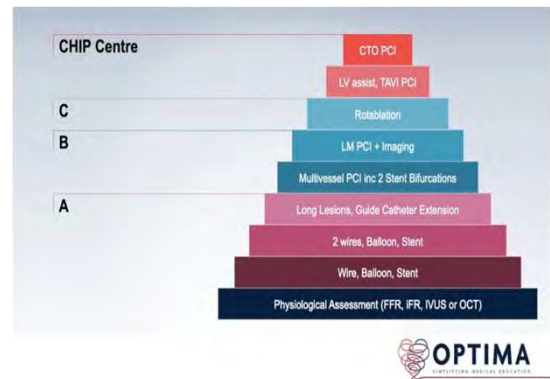


**OPTIMA**  
EMPOWERING MEDICAL EDUCATION

162

## Skills Required for CHIP PCI

- ▶ Management of complex lesions
- ▶ Expertise in intravascular imaging
- ▶ Ability to manage complications effectively
- ▶ Comfort with Mechanical Circulatory Support

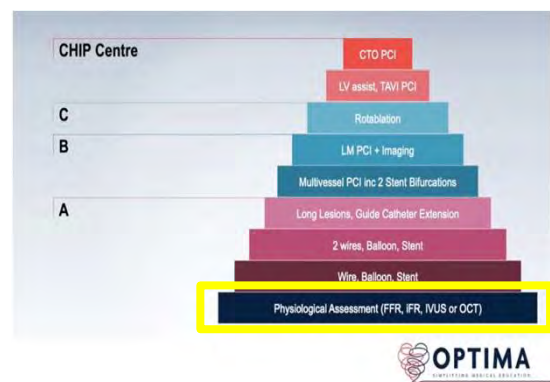


Riley, RF, Henry, TD, Mahmud, E, et al. SCAI position statement on optimal percutaneous coronary interventional therapy for complex coronary artery disease. *Catheter Cardiovasc Interv.* 2020; 96: 346–362. <https://doi.org/10.1002/ccd.28994>

163

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Riley, RF, Henry, TD, Mahmud, E, et al. SCAI position statement on optimal percutaneous coronary interventional therapy for complex coronary artery disease. *Catheter Cardiovasc Interv.* 2020; 96: 346–362. <https://doi.org/10.1002/ccd.28994>

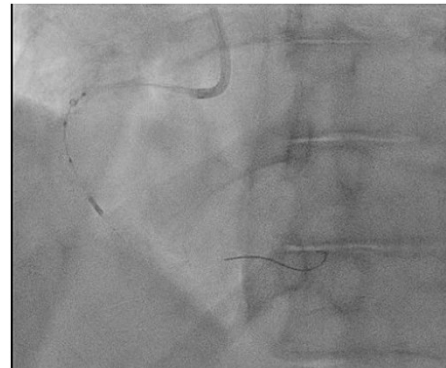
164

# Intravascular Imaging

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## What is Intravascular Imaging

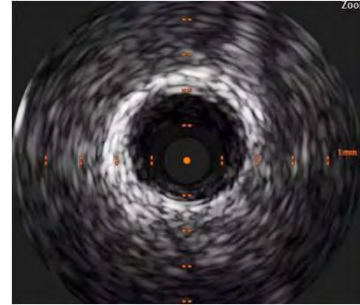
- ▶ Tomographic imaging of the coronary arteries using:
  - ▶ Intravascular Ultrasound (IVUS)
  - ▶ Optical Coherence Tomography (OCT)
- ▶ An imaging catheter is placed in the coronary artery
- ▶ Imaging occurs while catheter is pulled back through the artery
- ▶ Allows for visualization of the artery from the inside out



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## IVUS

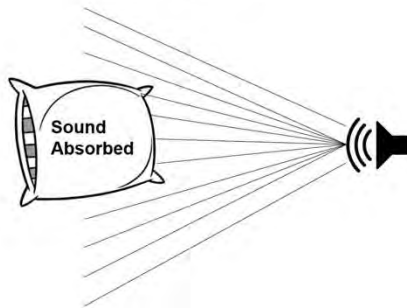
- ▶ Utilizes ultrasound for imaging
  - ▶ Transducer contains piezoelectric crystals
  - ▶ Electricity in the crystal creates sound waves
  - ▶ Reflected waves return to the transducer
    - ▶ Timing dependent upon:
      - ▶ Density & position of surrounding tissue
  - ▶ Computer calculates an image based on these densities
- ▶ Creates a high definition image of the artery



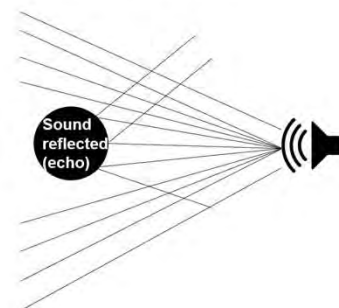
167

## IVUS Signal

Hypoechoic



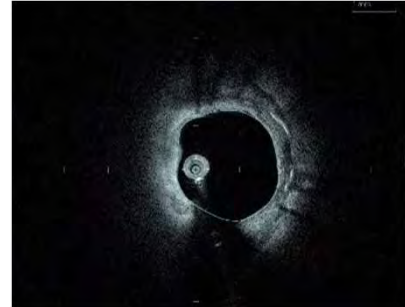
Hyperechoic



168

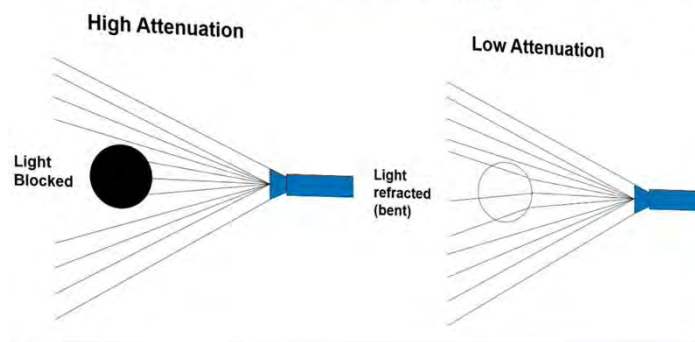
## Optical Coherence Tomography (OCT)

- ▶ Utilizes near infrared light for tomographic imaging
  - ▶ Red light is emitted from the catheter
  - ▶ Light is then reflected back to the catheter
  - ▶ Computer generates a picture of the surrounding tissues based on the reflective / backscattering properties of the surrounding tissue
- ▶ Creates an ultra-high definition image of the inside of a coronary artery



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### OCT Signal Attenuation



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## Normal Vessels

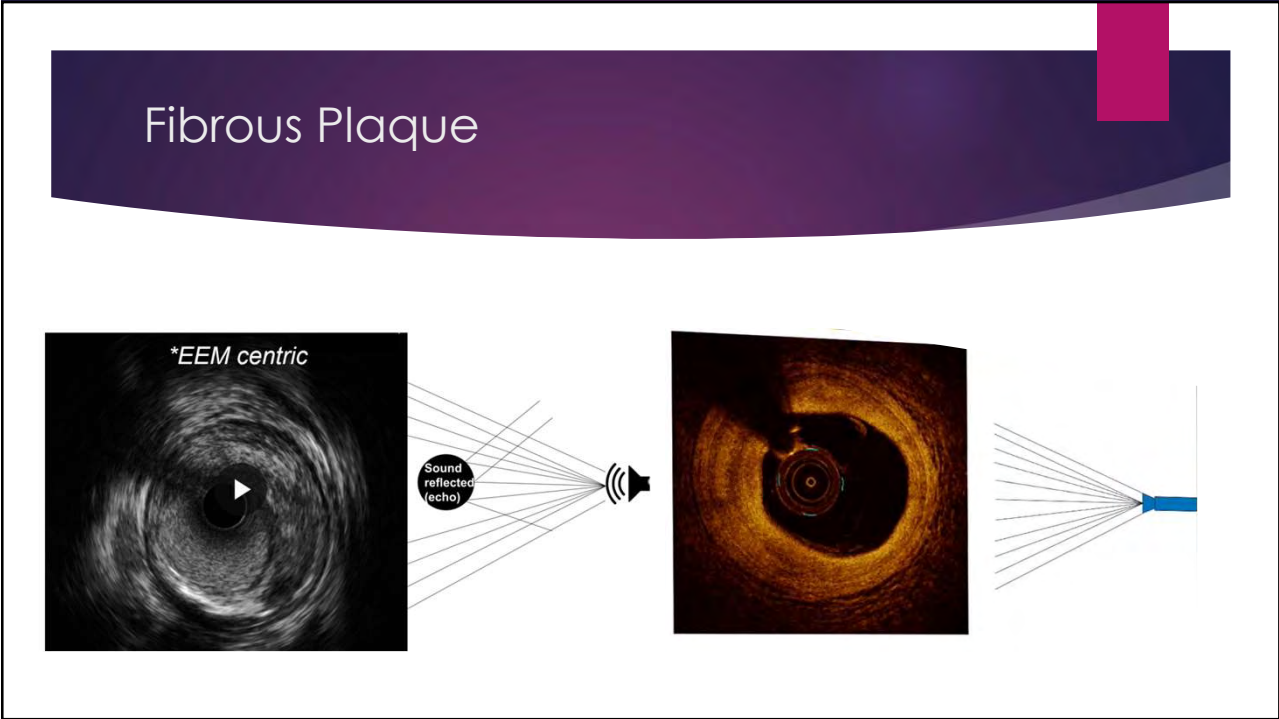
The slide displays four images related to normal vessel morphology. From left to right: 1) A B-mode ultrasound image showing concentric layers of the vessel wall labeled as Adventitia, Media, and Intima. 2) A histological section of a vessel wall with labels for Adventitia, Media, and Intima. 3) An IVUS image showing a catheter and a wire artifact, with labels for Adventitia, Media, and Intima. 4) A histological section showing the Internal Elastic Lamina (IEL) and External Elastic Lamina (EEL) with labels for Media and Intima.

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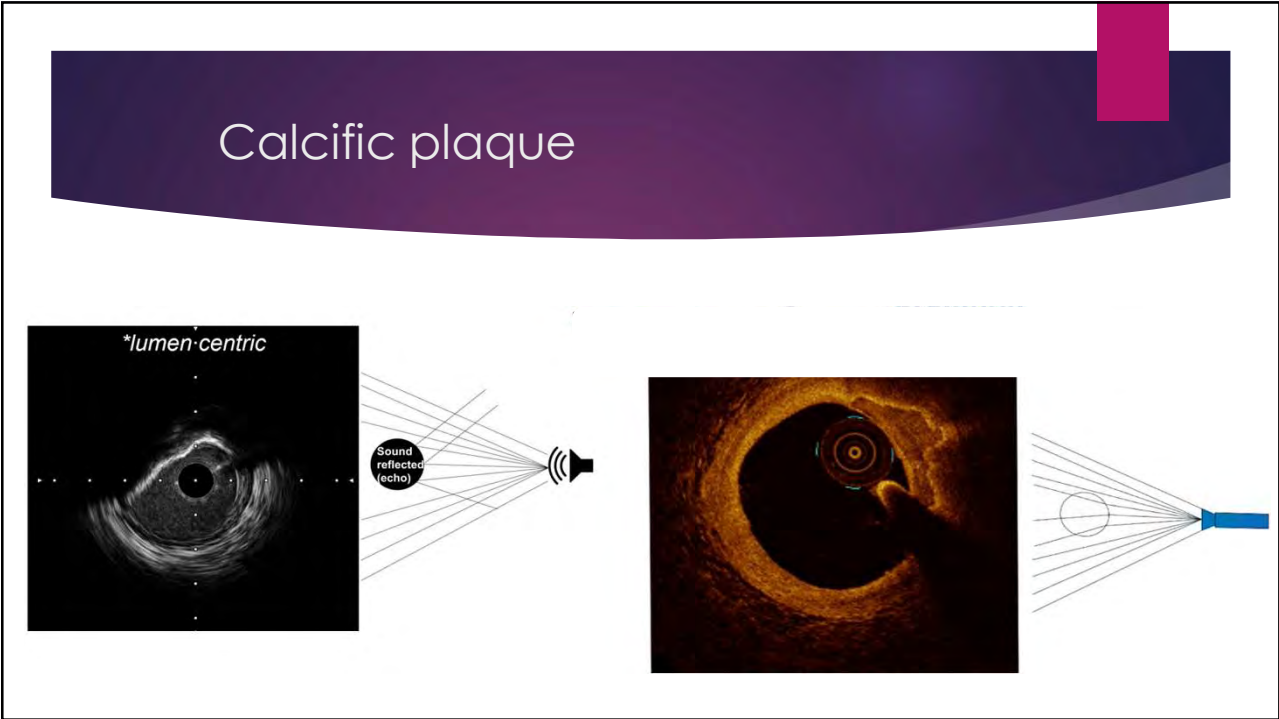
## Lipid Rich Plaque

The slide compares sound absorption in normal vessels versus lipid-rich plaques. On the left, a B-mode ultrasound image of a normal vessel is shown next to a diagram of a sound beam hitting the vessel wall, with a label 'Sound Absorbed' indicating that the vessel wall absorbs the sound. On the right, an IVUS image of a vessel with a plaque is shown next to a diagram of a sound beam hitting the plaque, where the beam is blocked and does not reach the vessel wall, illustrating how plaque affects sound absorption.

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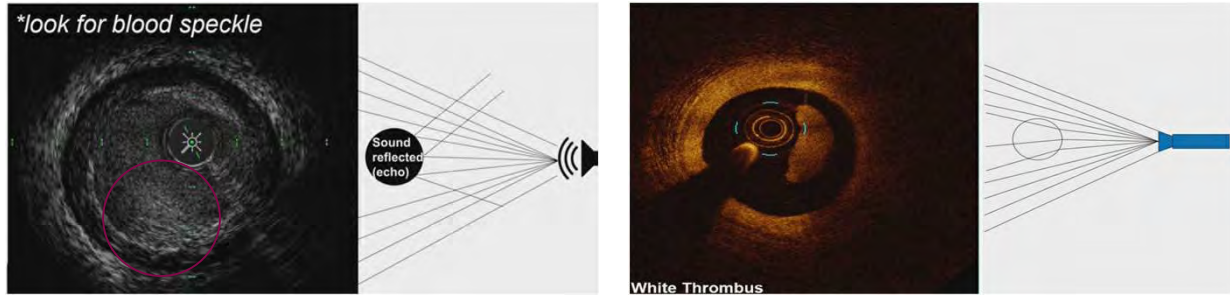


173



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# Thrombus



175

“

“Great, You Took a Pretty Picture,  
How Does This Help?”

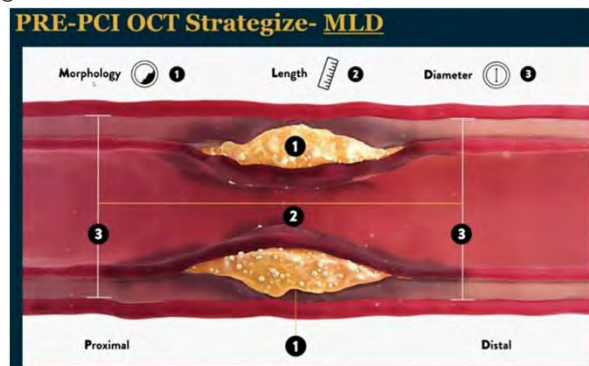
”

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## Imaging Guided PCI

- ▶ Using imaging to focus your intervention can lead to safer interventions with better long term results



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## Quick Primer on Coronary Calcium

- ▶ Atherosclerotic plaque composition:
  - ▶ Lipids
  - ▶ Fibrous tissue (collagen and proteoglycans)
  - ▶ Thrombus
  - ▶ Cells (smooth muscle and macrophages)
  - ▶ Calcium



- ▶ Everything but calcium can be dilated with balloons
- ▶ Heavily calcified lesions resist dilation with balloons
- ▶ Stents deployed in heavily calcified lesions without adequate preparation will be underexpanded
- ▶ Underexpanded stents fail
  - ▶ Thrombosis
  - ▶ Restenosis

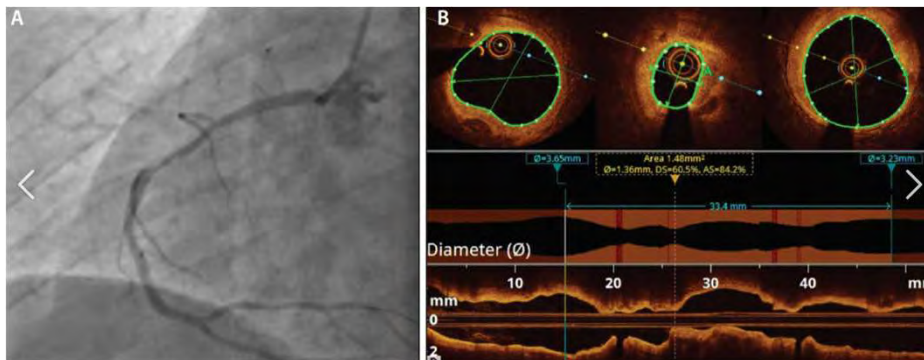
178

## How to Perform Imaging Guided PCI: Morphology



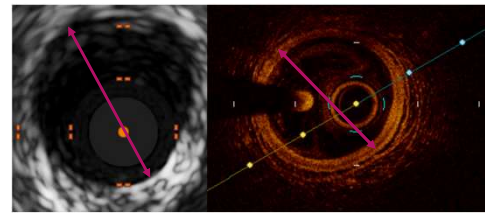
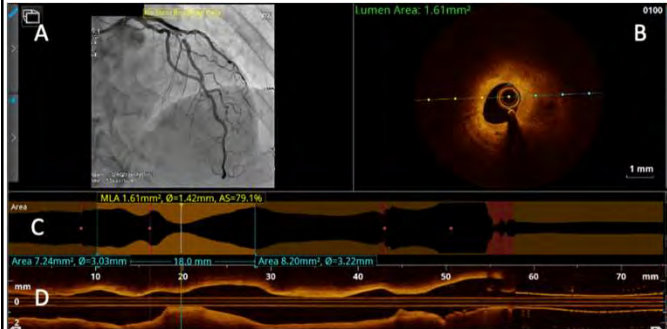
179

## How to Perform Imaging Guided PCI: Length



180

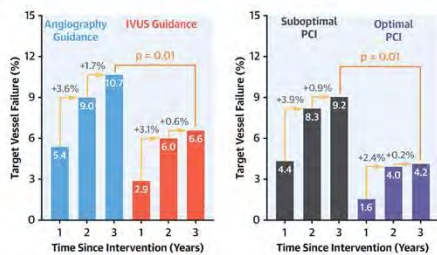
# How to Perform Imaging Guided PCI: Diameter



181

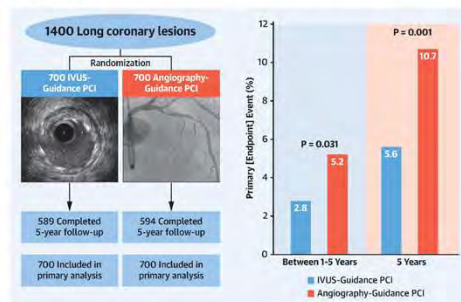
# Why Bother to Image?

**CENTRAL ILLUSTRATION: 3-Year Follow-Up of the Intravascular Ultrasound Guided Drug-Eluting Stents Implantation in "All-Comers" Coronary Lesions Trial**



Gao, X.-F. et al. J Am Coll Cardiol Intv. 2021;14(3):247-57.

**CENTRAL ILLUSTRATION: 5-Year Follow-Up of the IVUS-XPL Randomized Trial**



182

## How To Take on the Tough Lesions.....

- ▶ **Calcified Lesions**
- ▶ Bifurcations
- ▶ Left Main Disease
- ▶ Long Lesions with no stent landing zone
- ▶ Chronic Total Occlusions
- ▶ In Stent Restenosis

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## Complex Lesions: Calcium

"WHEN YOU'RE STUCK BETWEEN A ROCK AND A HARD PLACE"

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## Atherosclerosis Progression

- ▶ Atherosclerosis begins as a disease of the arterial intima
  - ▶ Lipid deposition in the intima triggers macrophage infiltration
  - ▶ Macrophages become foam cells
  - ▶ Lipid accumulates extracellularly
  - ▶ Smooth muscle infiltration
  - ▶ Increasing fibrosis and calcification
  - ▶ Plaque rupture or stabilization

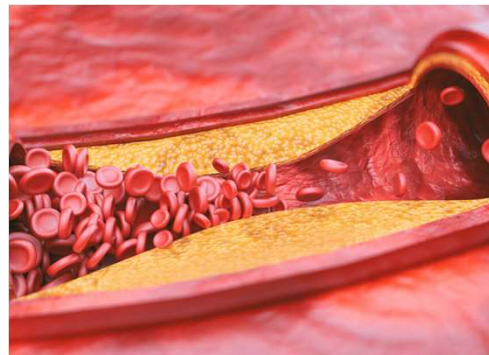


<b>Initial lesion</b>	"normal" histology some foam cells
<b>Fatty streak</b>	intracellular lipid accumulation
<b>Intermediate lesion</b>	extracellular lipid accumulation
<b>Atheroma</b>	lipid core development
<b>Fibroatheroma</b>	fibrotic/calcific layers
<b>Complicated lesion</b>	surface defect thrombosis

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## What's Actually in the plaque?

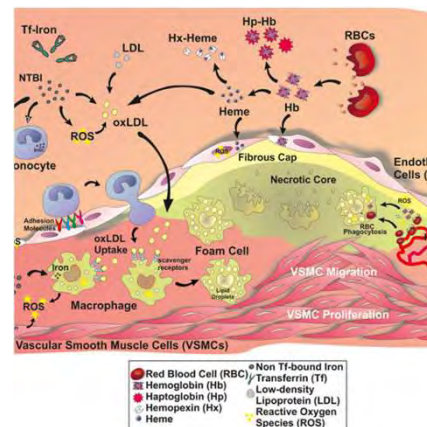
- ▶ Fat
  - ▶ Cholesterol
  - ▶ Triglycerides
- ▶ Fibrous Tissue
  - ▶ Proteoglycans
  - ▶ Collagen
- ▶ Thrombus
- ▶ Inflammatory Cells
  - ▶ Macrophages / foam cells
  - ▶ Smooth muscle cells
- ▶ Calcium



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## Why is there calcium in the Artery

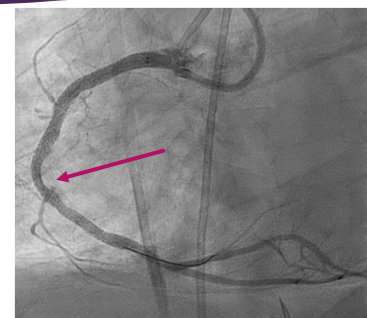
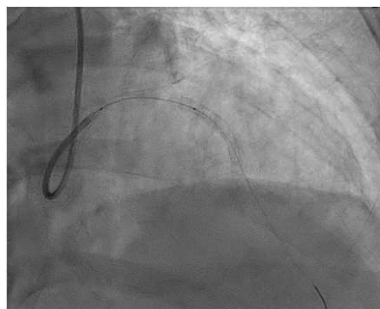
- ▶ Over 99% of the body's calcium is in our bones stored as hydroxyapatite crystal:  $\text{Ca}_{10}(\text{PO}_4)_6(\text{OH})_2$ 
  - ▶ Secreted by osteoblasts
- ▶ Atheroma progression leads to vascular smooth muscle cell recruitment
  - ▶ These smooth muscle cells behave like osteoblasts & secrete hydroxyapatite



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## Why Do I Care About Calcium

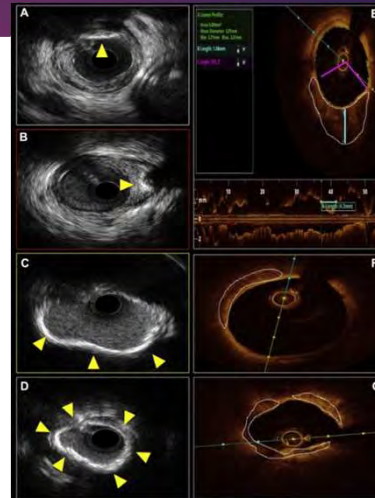
- ▶ Calcium increases the risk of complications
  - ▶ Stent underexpansion
    - ▶ In stent thrombosis
    - ▶ In stent restenosis
  - ▶ Perforation



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## Calcified Plaque Preparation

- ▶ Identification of significant calcium on intravascular imaging is necessary to select proper treatment modality
- ▶ Low to moderate burden of calcium can be ballooned and stented
- ▶ Heavy burden of calcium (circumferential) needs to be ablated or fractured



De Maria GL, Scarsini R, Banning AP. Management of Calcific Coronary Artery Lesions: Is it Time to Change Our Interventional Therapeutic Approach? JACC Cardiovasc Interv. 2019 Aug 12;12(15):1465-1478. doi: 10.1016/j.jcin.2019.03.038. PMID: 31395217.

189

## Lesion Preparation

- ▶ Two options
  - ▶ Calcium ablation
    - ▶ Rotational atherectomy
    - ▶ Orbital atherectomy
  - ▶ Calcium fracture
    - ▶ High pressure balloons
    - ▶ Cutting / scoring balloons
    - ▶ Intravascular lithotripsy (shockwave)

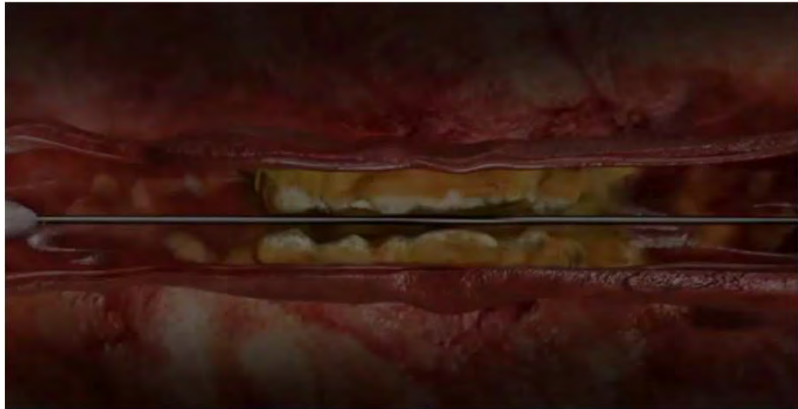


De Maria GL, Scarsini R, Banning AP. Management of Calcific Coronary Artery Lesions: Is it Time to Change Our Interventional Therapeutic Approach? JACC Cardiovasc Interv. 2019 Aug 12;12(15):1465-1478. doi: 10.1016/j.jcin.2019.03.038. PMID: 31395217.

190

## Calcium Ablation: Rotational Atherectomy

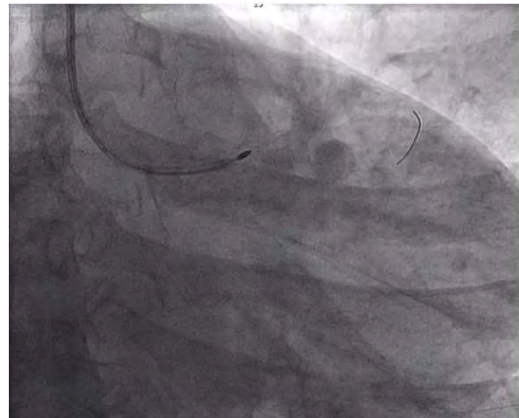
- ▶ Rotating burr
  - ▶ High speed (135-200k RPM)
  - ▶ Diamond coated
  - ▶ 1.25mm-2mm
- ▶ Ablates fibrocalcific plaque
  - ▶ Spares elastic tissue
- ▶ Pulverizes tissue to 5-10um particles



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## Calcium Ablation: Rotational Atherectomy

- ▶ Complications
  - ▶ Complete heart block
  - ▶ "No-Reflow"
  - ▶ Burr Entrapment
  - ▶ Perforation



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## Calcium Ablation: Orbital Atherectomy



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## Calcium Fracture: Balloons

- ▶ Specialized balloons can be inflated to higher pressures
  - ▶ Car tires: 2 atm
  - ▶ Road bike tires: 6 atm
  - ▶ Standard angioplasty: 12-18 atm
  - ▶ High pressure angioplasty: 20-25 atm
  - ▶ \*Super high pressure angioplasty: 35 atm
    - ▶ \*Only available in Europe & Asia
- ▶ Cutting/Scoring balloons focus the pressure



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## Calcium Fracture: IV Lithotripsy

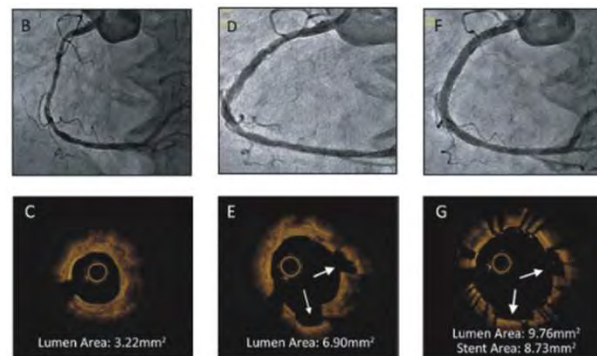
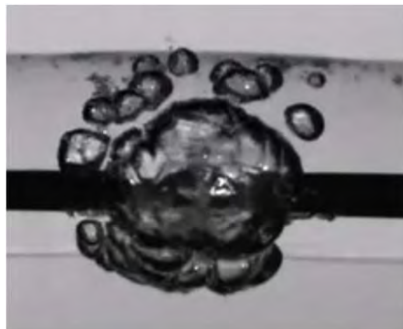
- ▶ Newest technology on the market
- ▶ Low pressure inflations (4 atm)
- ▶ Electric pulses create acoustic energy
  - ▶ Waves transfer their energy into calcific tissue
  - ▶ Fracture superficial and deep calcium
- ▶ User friendly
- ▶ Balloons are bulky and challenging to deliver



Hill JM, Kereiakes DJ, Shlofmitz RA, Klein AJ, Riley RF, Price MJ, Hermann HC, Bachinsky W, Waksman R, Stone GW; Disrupt CAD III Investigators. Intravascular Lithotripsy for Treatment of Severely Calcified Coronary Artery Disease. *J Am Coll Cardiol*. 2020 Dec 1;76(22):2635-2646. doi: 10.1016/j.jacc.2020.09.603. Epub 2020 Oct 15. PMID: 33069849.

195

## Calcium Fracture: IV Lithotripsy



Hill JM, Kereiakes DJ, Shlofmitz RA, Klein AJ, Riley RF, Price MJ, Hermann HC, Bachinsky W, Waksman R, Stone GW; Disrupt CAD III Investigators. Intravascular Lithotripsy for Treatment of Severely Calcified Coronary Artery Disease. *J Am Coll Cardiol*. 2020 Dec 1;76(22):2635-2646. doi: 10.1016/j.jacc.2020.09.603. Epub 2020 Oct 15. PMID: 33069849.

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## Calcium: Summary

- ▶ Calcified lesions can prevent stent delivery, stent expansion and lead to many complications
- ▶ Refining older techniques and adopting new technologies can allow us to treat patients with calcified lesions

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## Complex Lesions

- ▶ ~~Calcified Lesions~~
- ▶ **Bifurcations**
- ▶ Left Main Disease
- ▶ Chronic Total Occlusions

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# Bifurcation Management

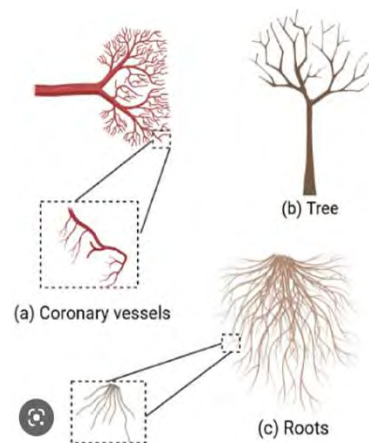
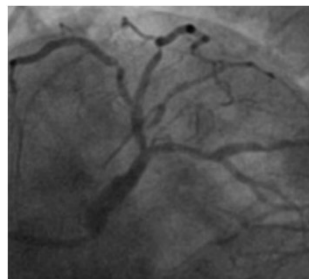
"WHEN YOU COME TO A FORK IN THE ROAD, TAKE IT"

-YOGI BERRA (MAYBE)

199

## What is a Coronary Bifurcation Lesion

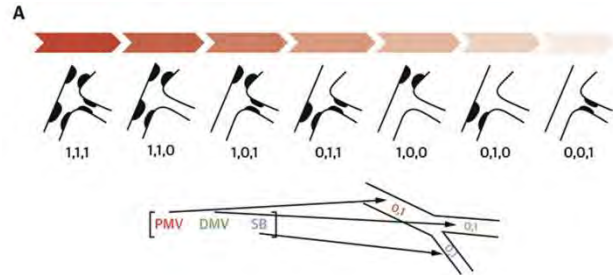
- ▶ Coronary tree is an object of pseudofractal geometry
- ▶ Branches off into asymmetrical, increasingly smaller bifurcations
- ▶ EBC Definition: Coronary narrowing occurring adjacent to or involving a **significant** side branch
  - ▶ Significant: loss is of consequence to the patient



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## Classification of Bifurcations

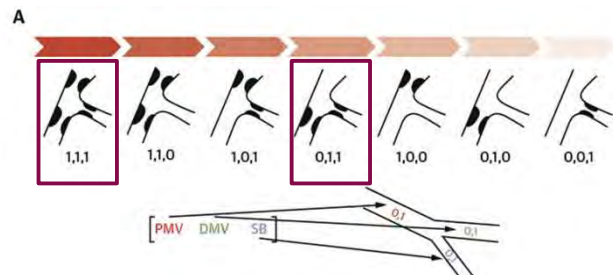
- ▶ Medina Classification:
  - ▶ Simplest
  - ▶ Binary classification of main vessel, main branch and side branch
    - ▶ >50% stenosis = 1
    - ▶ <50% stenosis = 0
    - ▶ ie: (1,0,1) or (0,1,0)



201

## Classification of Bifurcations

- ▶ Medina Classification:
  - ▶ Simplest
  - ▶ Binary classification of main vessel, main branch and side branch
    - ▶ >50% stenosis = 1
    - ▶ <50% stenosis = 0
    - ▶ ie: (1,0,1) or (0,1,0)
  - ▶ True bifurcations; (1,1,1) or (0,1,1)



202

## Why Do Bifurcations Represent A Challenge?

- ▶ Associated with lower procedural success rates
- ▶ Higher rates of long term cardiovascular events



Sawaya FJ, Lefèvre T, Chevalier B, Garof P, Hovasse T, Morice MC, Rab T, Louvard Y. Contemporary Approach to Coronary Bifurcation Lesion Treatment. JACC Cardiovasc Interv. 2016 Sep 26;9(18):1861-78. doi: 10.1016/j.jcin.2016.06.056. PMID: 27659563.

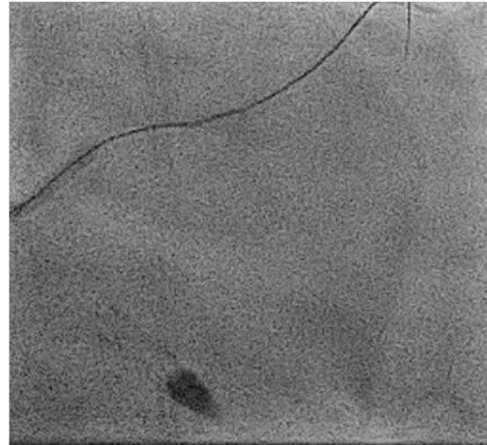
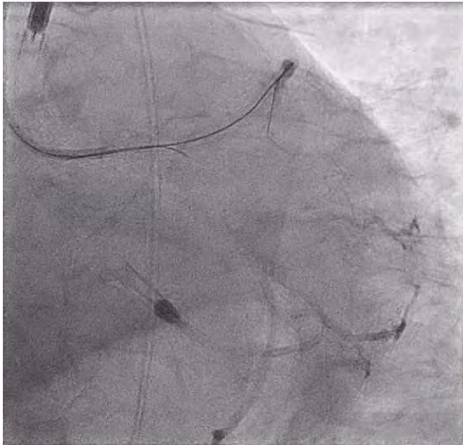
203

## Side Branch Jailing



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## How To Treat a Coronary Bifurcation: Step 1- Wire Both Branches



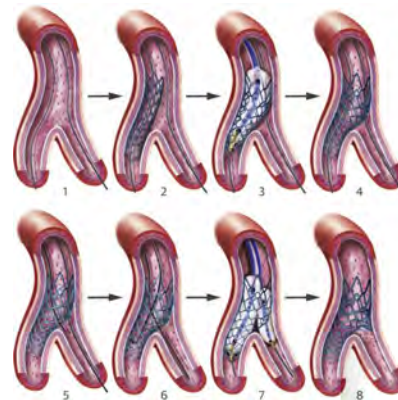
205

## Step 2: Pick a Strategy, 1 or 2 Stents

### One Stent

- ▶ "Provisional approach"
  - ▶ Standard approach for bifurcations
    - ▶ No data that two stents is superior
  - ▶ Simple & faster
    - ▶ Lower rates of complications
    - ▶ Typically well tolerated

### Provisional Approach



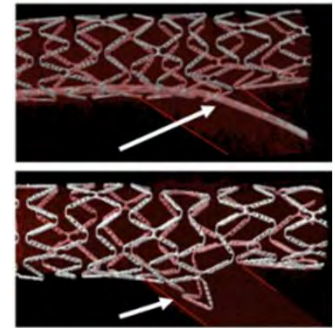
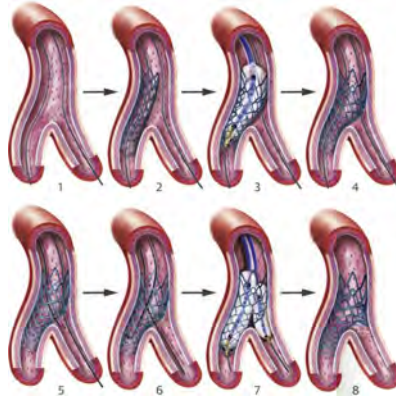
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## Step 2: Pick a Strategy, 1 or 2 Stents

### One Stent

- ▶ "Provisional approach"
  - ▶ Standard approach for bifurcations
    - ▶ No data that two stents is superior
- ▶ Simple & faster
  - ▶ Lower rates of complications
  - ▶ Typically well tolerated

### Provisional Approach



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## Step 2: Pick A Strategy, One or Two Stents

### Two Stents

- ▶ Two stents needed when:
  - ▶ Flow compromised in side branch
  - ▶ Side branch got dissected
  - ▶ Large side branch severely diseased

### Options

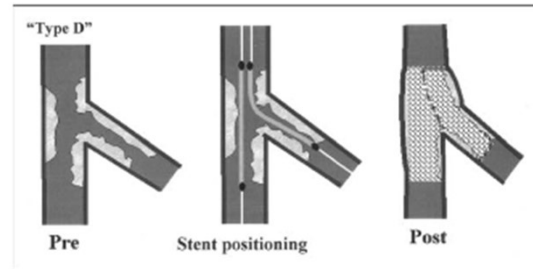
- ▶ Easiest > Hardest
  - ▶ Simultaneous kissing stents (double barrel)
  - ▶ T and Protrusion (TAP)
  - ▶ Culotte
  - ▶ Crush and DK Crush

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## Two Stent Strategies: SKS or Double Barrel

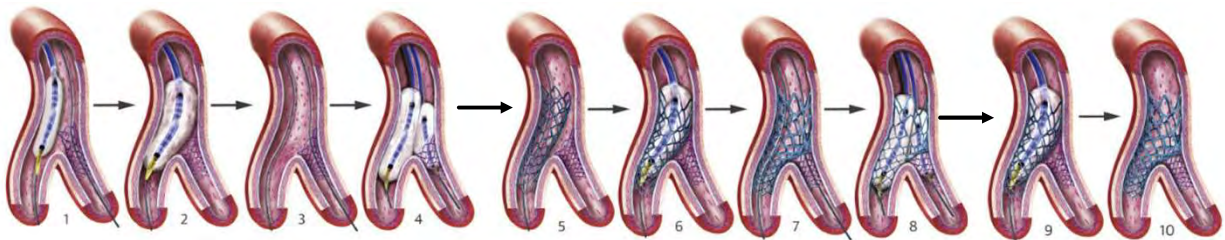
- ▶ Pros:
  - ▶ Faster and easier than any strategy (including provisional)
  - ▶ Covers all the disease
- ▶ Cons:
  - ▶ Will never endothelialize
  - ▶ Can never stop P2Y12... Ever!



S.K. Sharma, A. Choudhury, J. Lee, et al. *Simultaneous kissing stents (SKS) technique for treating bifurcation lesions in medium-to-large size coronary arteries* Am J Cardiol. 94 (2004), pp. 913-917

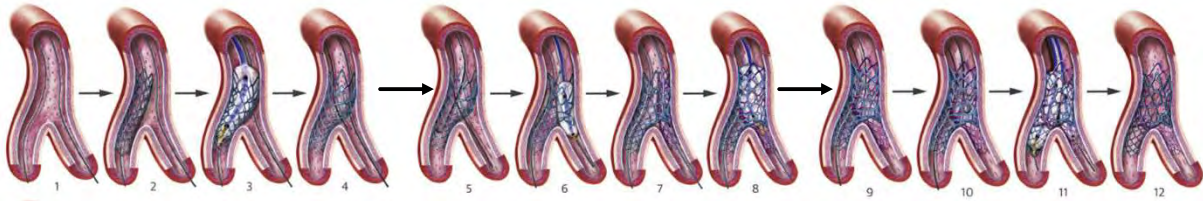
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## How To Do a DK Crush: The Right Way



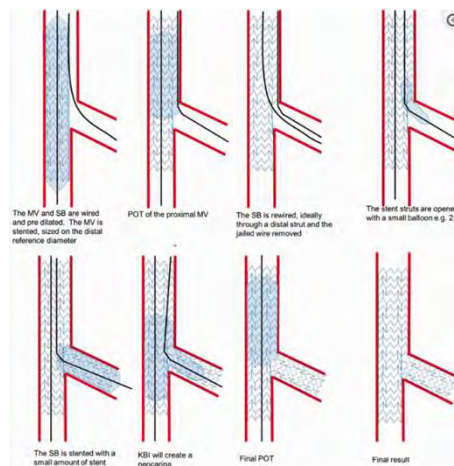
210

## Other Two Stent Techniques: Culotte



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## Other Two Stent Techniques: TAP



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## Complex Lesions

- ▶ Calcified Lesions
- ▶ Bifurcations
- ▶ **Left Main Disease**
- ▶ Chronic Total Occlusions

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## Left Main Disease

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## Multiple Studies Evaluating PCI vs CABG for LM Disease

- ▶ SYNTAX, PRECOMBAT, NOBLE AND EXCEL
  - ▶ All cause mortality: Equivalent (possible small benefit favoring CABG (<0.2% per year))
  - ▶ Repeat Revascularization: CABG is superior
  - ▶ Spontaneous MI: CABG is superior
  - ▶ Stroke: PCI is superior

Sabatine MS, Bergmark BA, Murphy SA, O'Gara PT, Smith PK, Serruys PW, Kappetein AP, Park SJ, Park DW, Christiansen EH, Holm NR, Nielsen PH, Stone GW, Sabik JF, Braunwald E. Percutaneous coronary intervention with drug-eluting stents versus coronary artery bypass grafting in left main coronary artery disease: an individual patient data meta-analysis. *Lancet*. 2021 Dec 18;398(10318):2247-2257. doi: 10.1016/S0140-6736(21)02334-5. Epub 2021 Nov 15. Erratum in: *Lancet*. 2022 Apr 23;399(10335):1606. Erratum in: *Lancet*. 2022 Oct 15;400(10360):1304. PMID: 34793745.

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## What Makes Left Main Disease Hard to Stent?

- ▶ Large area of myocardium at risk
  - ▶ Ischemia with devices can lead to hemodynamic collapse
  - ▶ Complications can be disastrous
    - ▶ Acute closure can lead to hemodynamic collapse
- ▶ LAD / Circumflex bifurcation
  - ▶ Often requires 2-stent approaches
  - ▶ Operator needs to be comfortable moving quickly through those steps

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## How to Stent A Left Main

- ▶ Be comfortable with multiple bifurcation techniques
- ▶ Ensure the patient is well compensated beforehand
  - ▶ Perfusing well with normal filling pressures
- ▶ Use mechanical support if necessary

Davies RE, Rier JD, McCabe JM. Patient and Device Selection for Hemodynamic Support in High-Risk Percutaneous Coronary Intervention. *Interv Cardiol Clin.* 2021 Jan;10(1):121-130. doi: 10.1016/j.iccl.2020.09.001. Epub 2020 Oct 29. PMID: 33223101.

### PROTECTED PCI ALGORITHM

LVEF < 50%: EVALUATE ALGORITHM
LVEF < 40%: RECOMMEND RHC PRIOR TO PCI
+2 Cardiac index < 2.0 L/min/m <sup>2</sup> or PA sat < 55%
+1 Syntax score ≥ 22
+1 Ejection fraction < 25%
+1 Systolic BP < 100 mm Hg at baseline
+1 ACS presentation
+1 Planned revascularization > 2 territories
+1 Likely prolonged ischemia <ul style="list-style-type: none"> <li>- Retrograde chronic total occlusion</li> <li>- Atherectomy</li> </ul>
+1 Severe mitral regurgitation
+1 Decompensated state <ul style="list-style-type: none"> <li>- LVEDP &gt; 20 mm Hg</li> <li>- Significant new orthopnea</li> </ul>
-1 High-risk vascular injury/significant bleeding
-1 Hemoglobin < 8 g/dL

Score ranges and corresponding support recommendations:

- 0-2 → UNLIKELY TO NEED SUPPORT
- 3 → CONSIDER SUPPORT
- ≥4 → STRONGLY CONSIDER SUPPORT

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## Complex Lesions

- ▶ Calcified Lesions
- ▶ Bifurcations
- ▶ Left Main Disease
- ▶ **Chronic Total Occlusions**

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# Chronic Total Occlusions (CTO's)

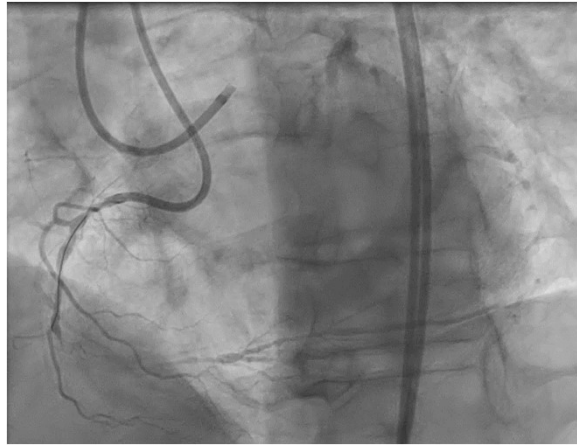
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## A Note On Chronic Total Occlusions

- ▶ Defined as a 100% occlusion that has been present for > 3 months
  - ▶ Viewed as the last frontier for interventional cardiology
  - ▶ Traditionally associated with high rates of procedural failure
  - ▶ Most common reason for referral to CABG
- ▶ Downstream territories kept alive via collaterals formed by angiogenesis
- ▶ Modern hybrid approaches have raised success rates

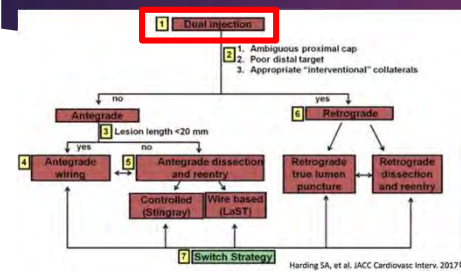
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# An RCA CTO

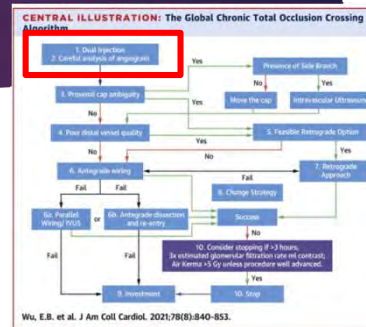
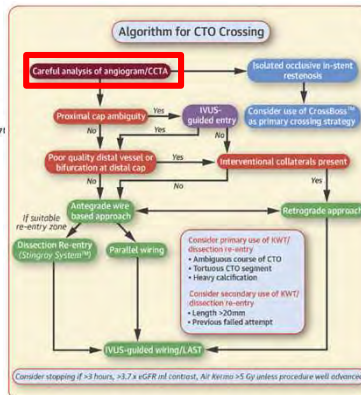


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# All CTO Algorithms Start With Dual Injection



Harding SA, et al. JACC Cardiovasc Interv. 2017<sup>1</sup>

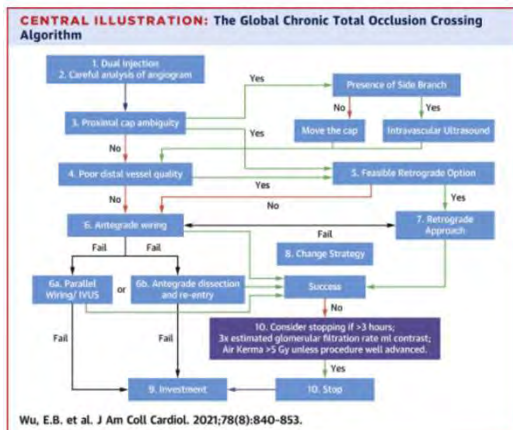


Wu, E.B. et al. J Am Coll Cardiol. 2021;78(8):840-853.

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## Complex and Higher Risk

### CTO: Wiring

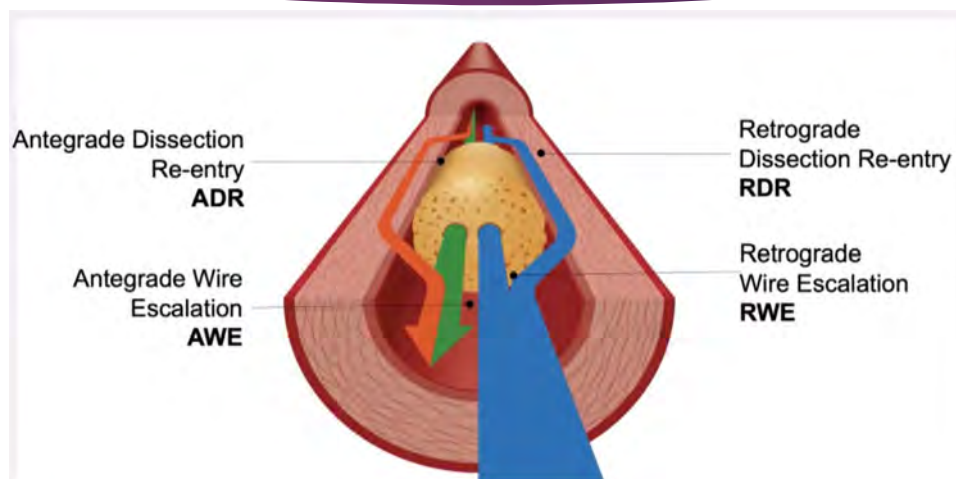


### Standard PCI

1. Engage Artery
2. Wire Lesion
3. Balloon
4. Stent

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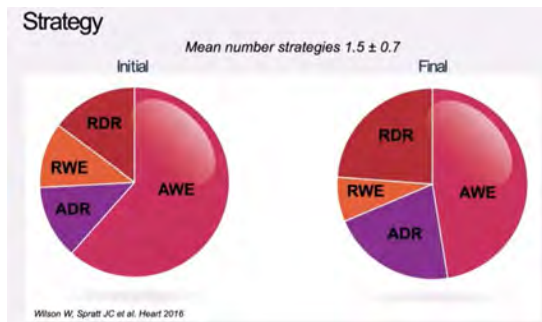
## Four Basic Strategies to Cross



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## A CTO Operator Needs to Know All Techniques



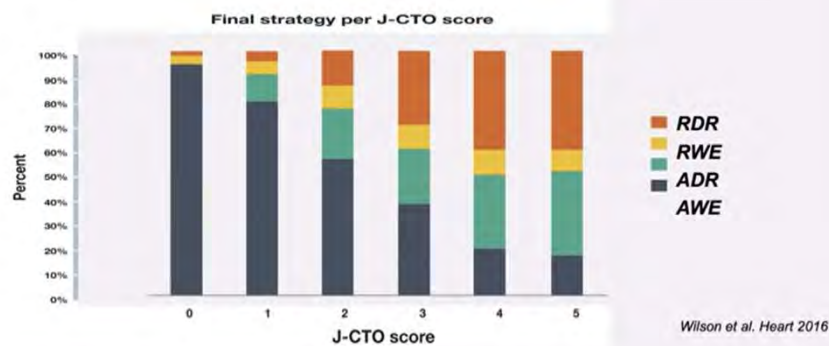
Greater chance of success = Hybrid Skills



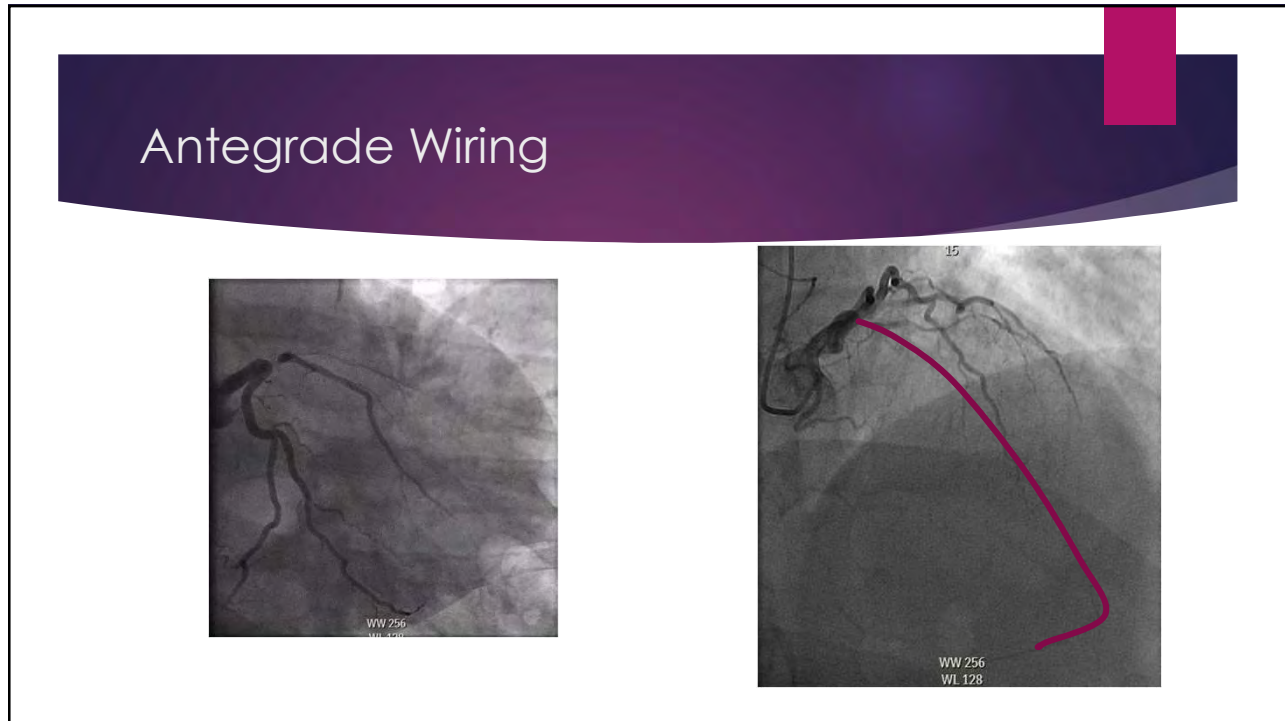
225

## Harder Lesions Require Different Skills

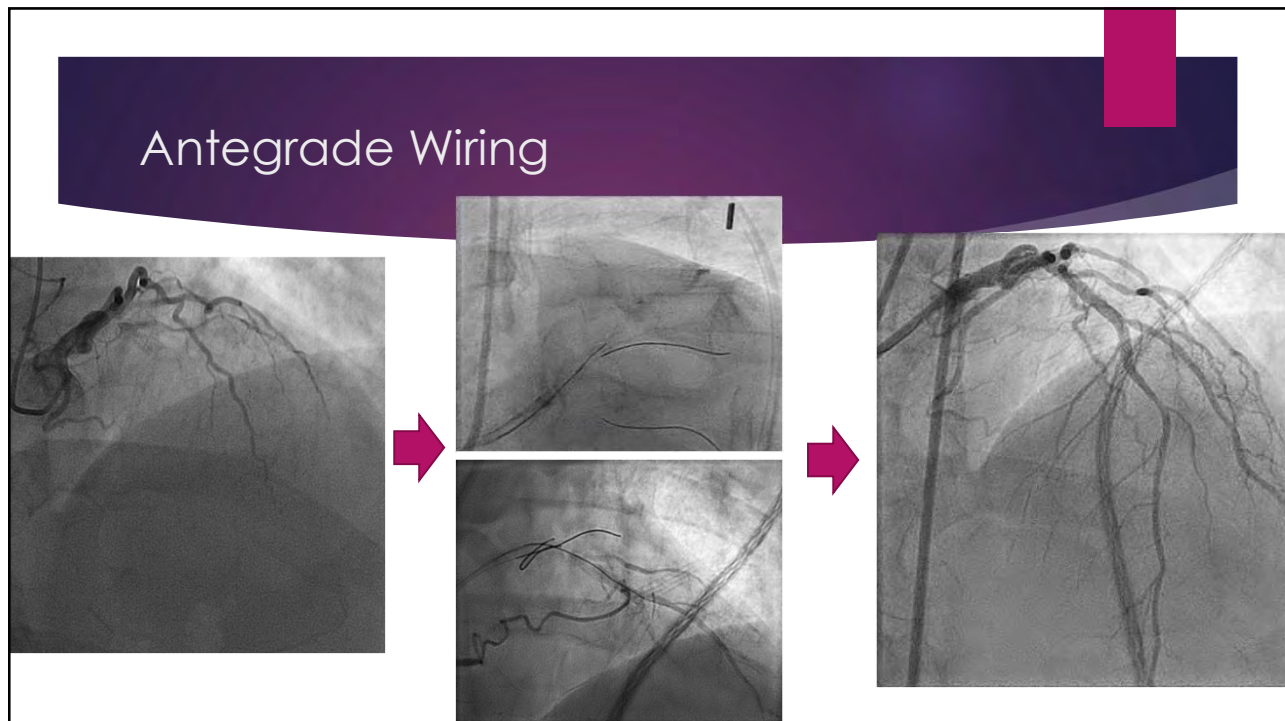
- ▶ The plan should be designed around the patient not the operator



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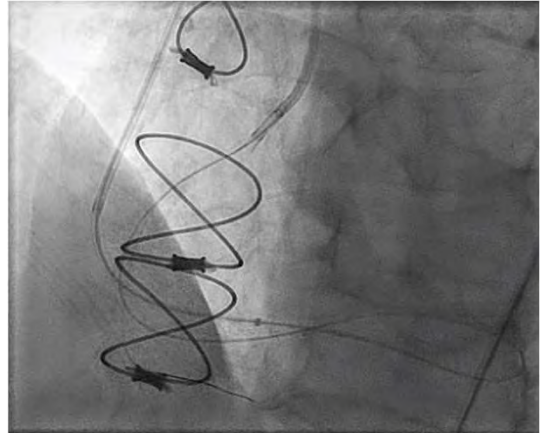
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## Retrograde Techniques

- ▶ The distal cap of a CTO is not exposed to systemic pressure so it is softer
  - ▶ Less fibrous tissue and calcium
  - ▶ Easier to cross
- ▶ The distal cap is accessed through natural collaterals or through surgical bypass grafts
- ▶ The lesion can be tracked through or around the plaque with a wire
- ▶ After crossing the proximal cap, the wire is directed into the antegrade guide catheter and exchanged for an externalization wire
- ▶ The CTO can then be fixed



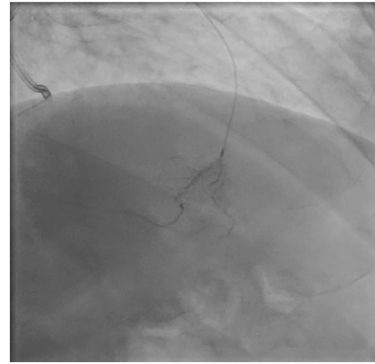
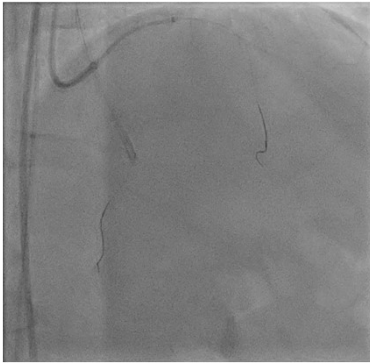
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## Retrograde Wiring of an RCA CTO



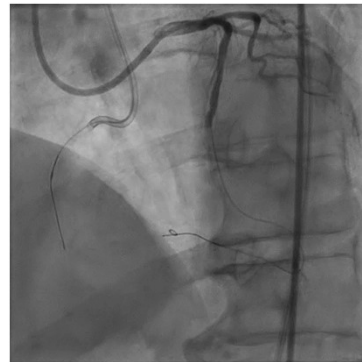
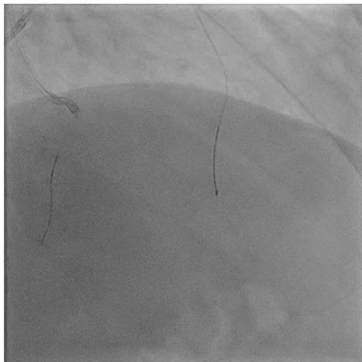
230

## Retrograde Wiring of an RCA CTO



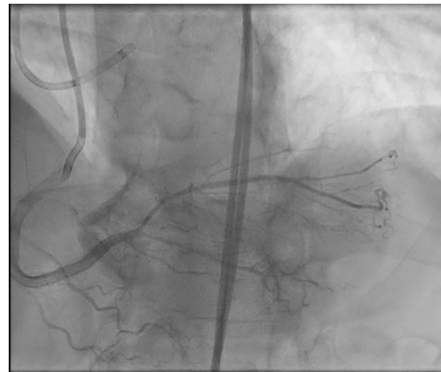
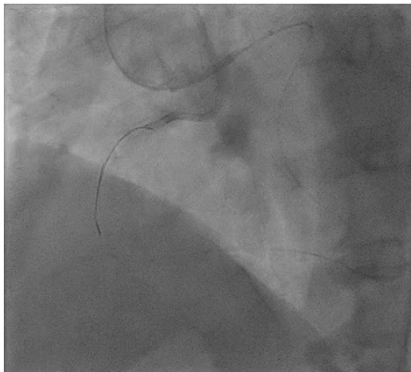
231

## Retrograde Wiring of an RCA CTO



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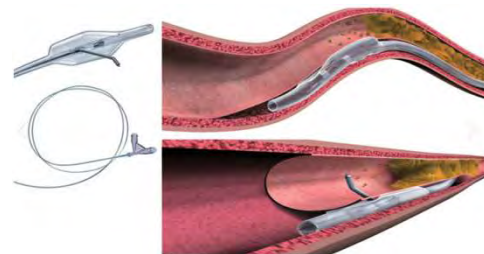
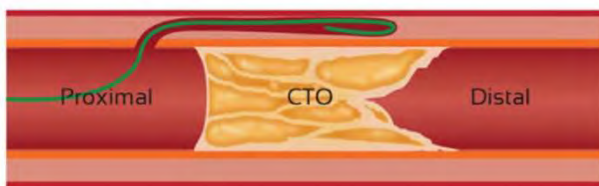
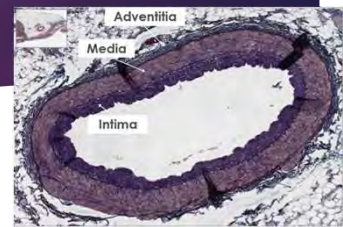
## Retrograde Wiring of an RCA CTO



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## Antegrade Dissection Reentry Techniques

- ▶ Sometimes it's smarter to go around the plaque than through it
  - ▶ Avoid sharp wires that put holes in vessels
  - ▶ The subintimal space can be traversed with a knuckled wire
- ▶ Once past the plaque, the vessel can be reentered
- ▶ Stent deployed around the blockage



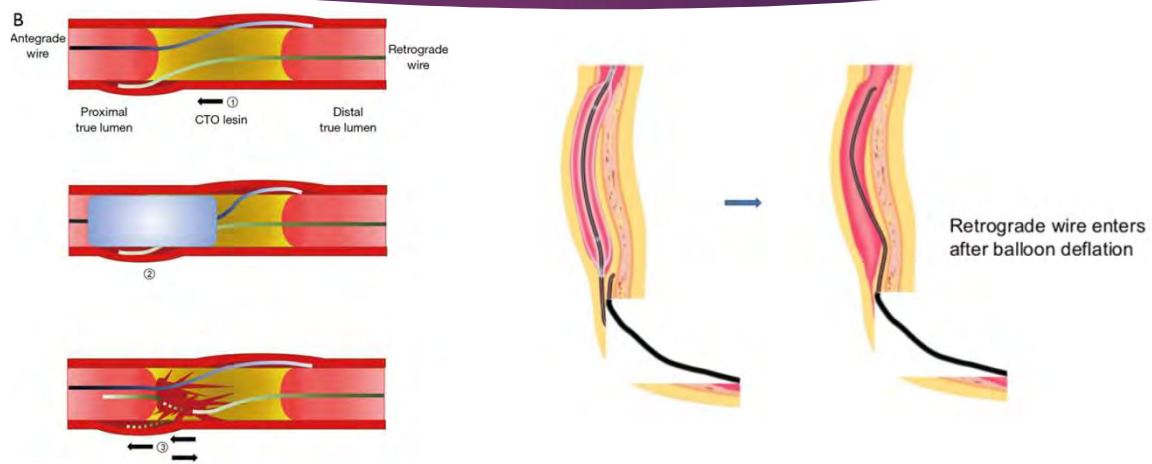
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## Antegrade Reentry Using a Stingray



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## Retrograde Dissection Reentry (Reverse CART)



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## CTO PCI

- ▶ Risk of significant complications 2-4%
- ▶ Typically two operators working for 2-4 hours
- ▶ Requires significant expertise at specialized centers
  - ▶ Experience growing throughout northern New England
  - ▶ Successful hybrid operators now practicing at:
    - ▶ University of Vermont
    - ▶ Eastern Maine Medical Center
    - ▶ Central Maine Medical Center
    - ▶ Maine Medical Center
    - ▶ Catholic Medical Center
    - ▶ Dartmouth Hitchcock Medical Center

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## In Conclusion

- ▶ The progress made in the invasive management of heart disease is astounding
- ▶ For patients that require coronary revascularization, we can now tailor therapy to them rather than to us (the operators)
- ▶ Our combined surgical & cath lab team is now successfully stabilizing & recovering many patients in severe cardiogenic shock that would have died before

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## Future Directions in Shock and Complex PCI

- ▶ Continue to improve our skillsets to ensure that patients are offered the procedure that is right for them
- ▶ Optimizing a team based response to patients requiring complex interventions for cardiogenic shock
- ▶ Look towards expanding our ability to treat patients outside of DHMC with refractory cardiac arrest