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Track 2A

Women's Cardiology



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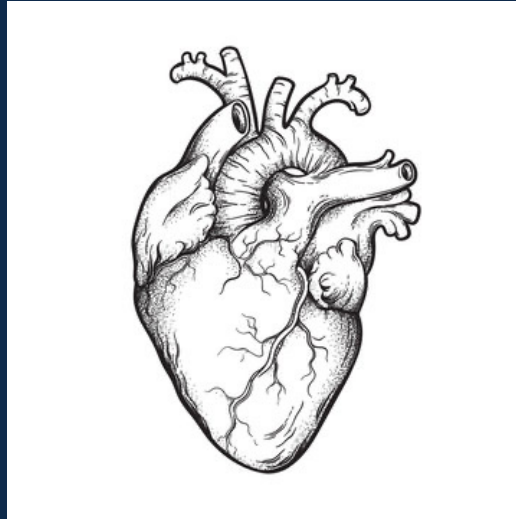
Trainee Presentation: Brianna Skaff



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Spontaneous Coronary Artery Dissection



*Brianna Skaff
University of Kentucky
Internal Medicine PGY-3*



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Learning Objectives

- Understand **Spontaneous Coronary Artery Dissection (SCAD)**: Recognize the definition, risk factors, and clinical presentation of SCAD.
- Diagnostic Approaches: Identify the diagnostic methods and challenges in detecting SCAD.
- Management Strategies: Learn about the treatment options and management approaches for SCAD patients.



Case presentation

- 53-y.o. F
- PMH: anxiety
- CC: chest pain
- **HPI:**
 - Chest tightness that started one day ago that radiated down her arms and up her neck
 - Worse with exertion
 - Relieved with certain positions, including leaning forward
 - Denies personal or family cardiac history
 - Medications: Bupropion, Zoloft, cetirizine



Physical Exam

- GENERAL: no acute distress, cooperative
- SKIN: warm and dry, no rashes, wounds, ulcers
- RESPIRATORY/THORAX: **breath sounds equal, respirations non-labored, no wheezes, rales, rhonchi**
- CARDIOVASCULAR: **regular rate and rhythm, normal S1 and S2, no murmurs, pulses 2+, no edema**
- MUSCULOSKELETAL: no joint swelling, full range of motion in all extremities



Differential Diagnosis

MORE LIKELY

- **Acute coronary syndrome**
- **Pulmonary embolism**
- **Aortic dissection**
- **Pericarditis**
- Myocarditis
- Pneumothorax

LESS LIKELY

- Musculoskeletal pain
- GERD
- Costochondritis

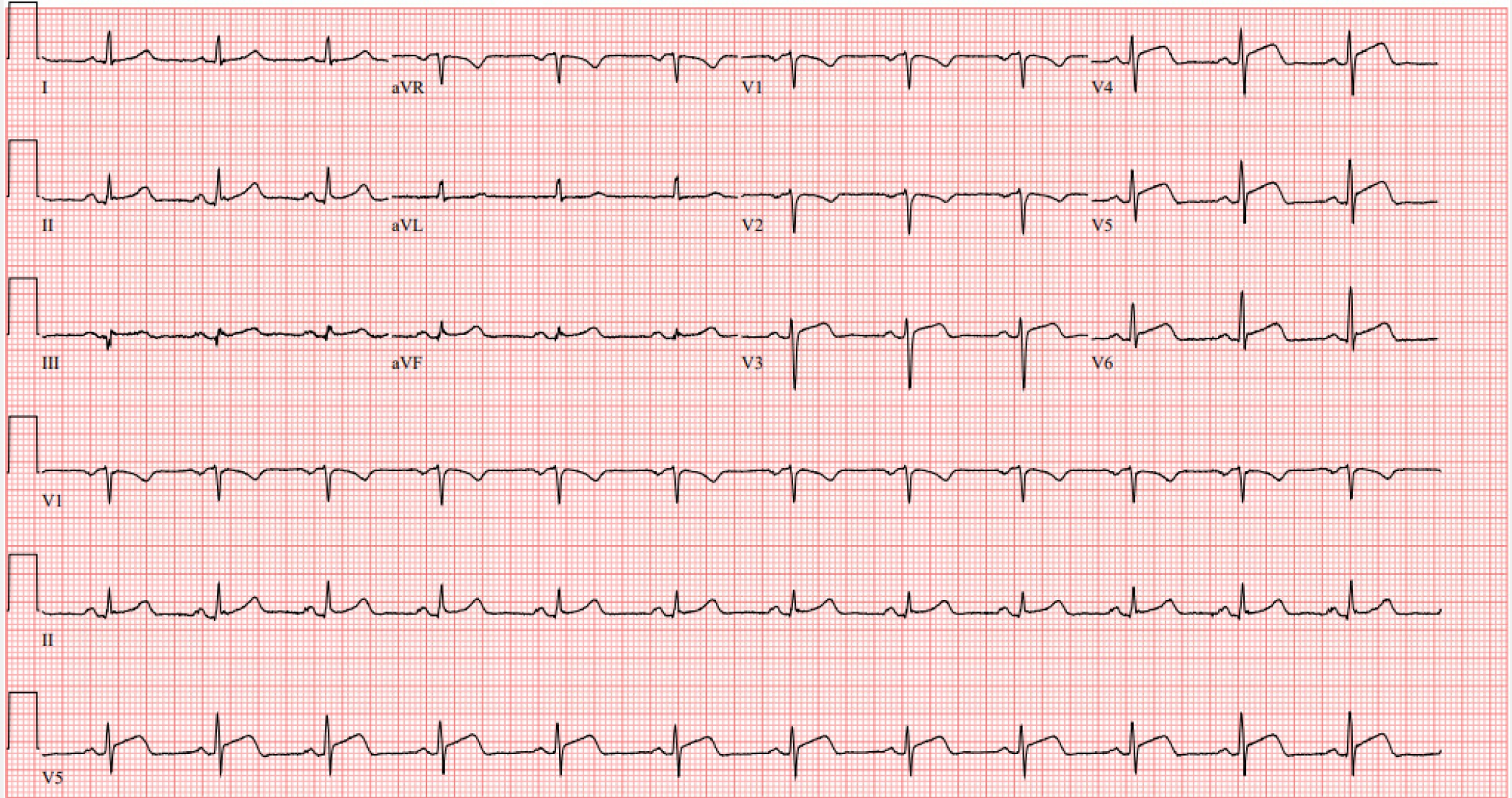


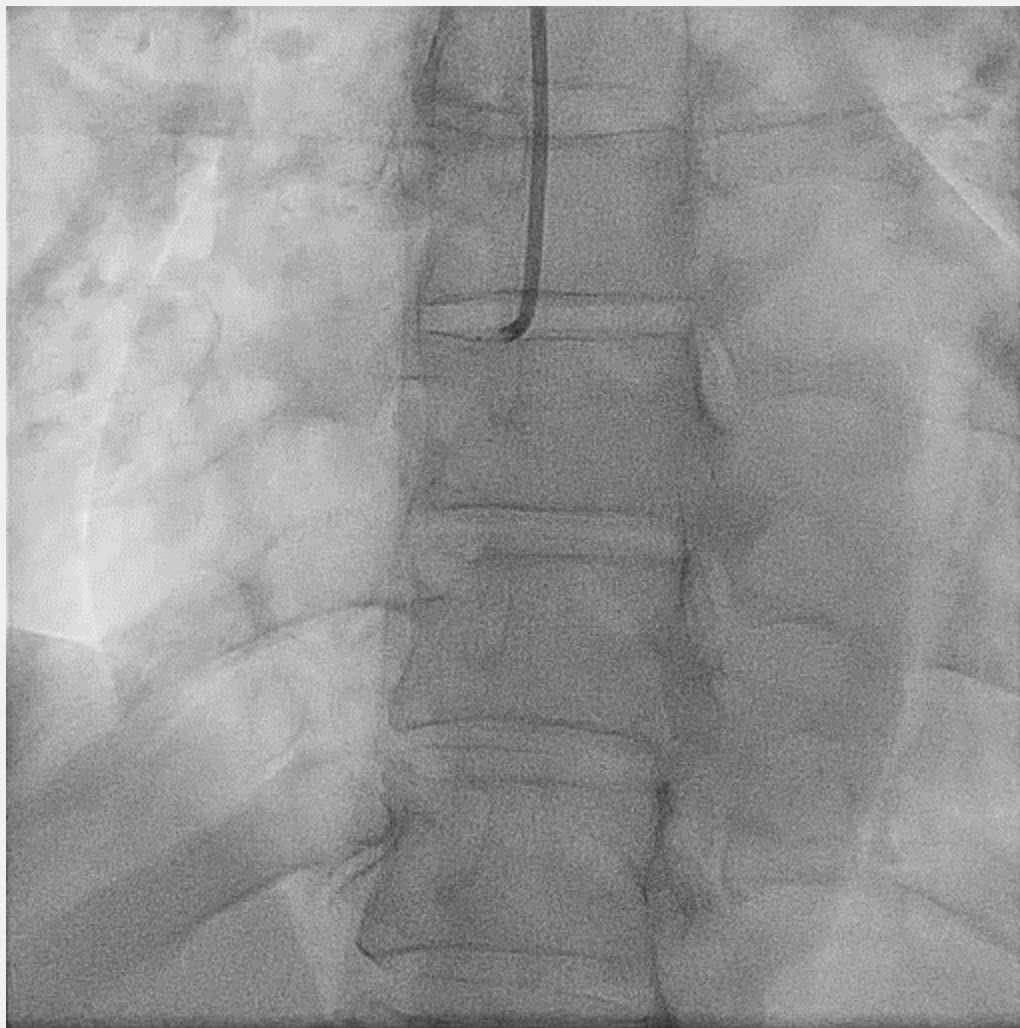
Labs

Lab	Value
WBC	9.36
Hgb	12.3
Plt	283
Na	136
K	3.8
Cr	0.69
Glucose	106
Troponins	276 --> 254 (delta 22)



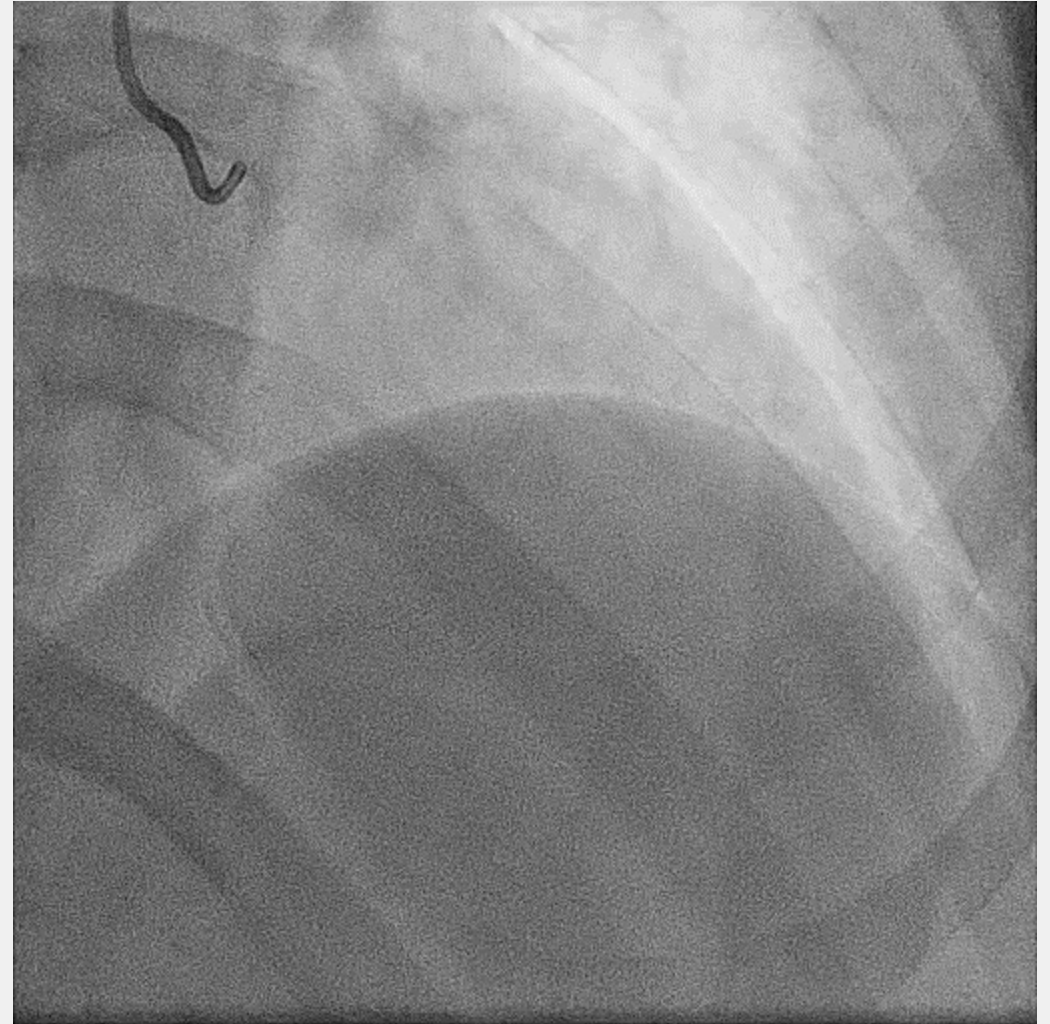
EKG



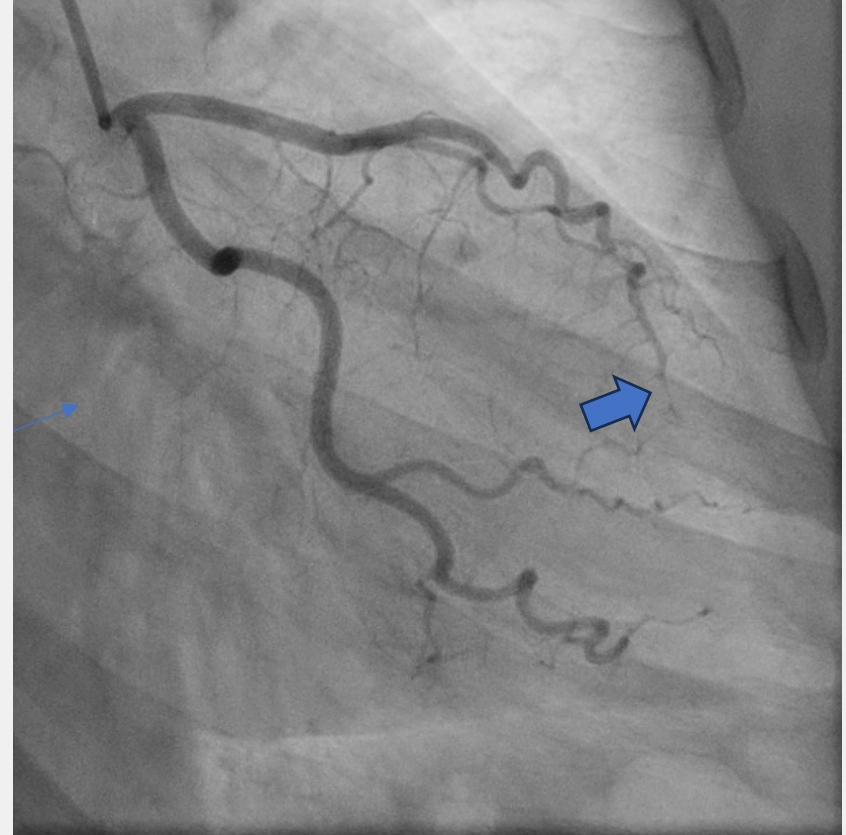
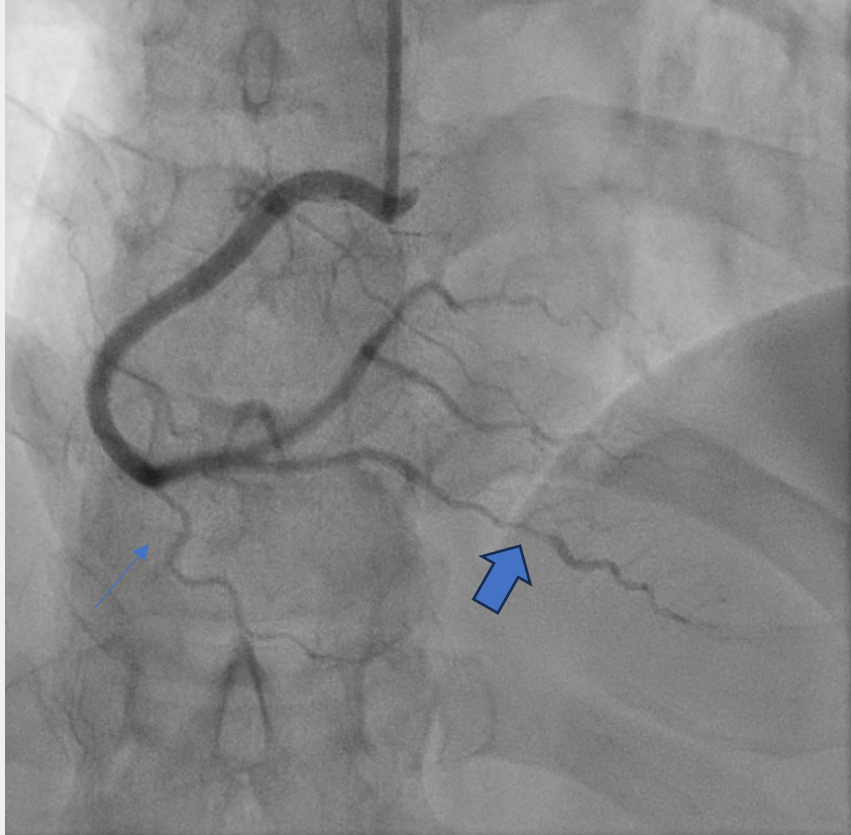


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Cath Report



Right dominant coronary circulation with tortuous arteries and evidence of spontaneous coronary artery dissection involving the RPDA (Type 2A) and distal LAD (Type 4).



Spontaneous Coronary Artery Dissection



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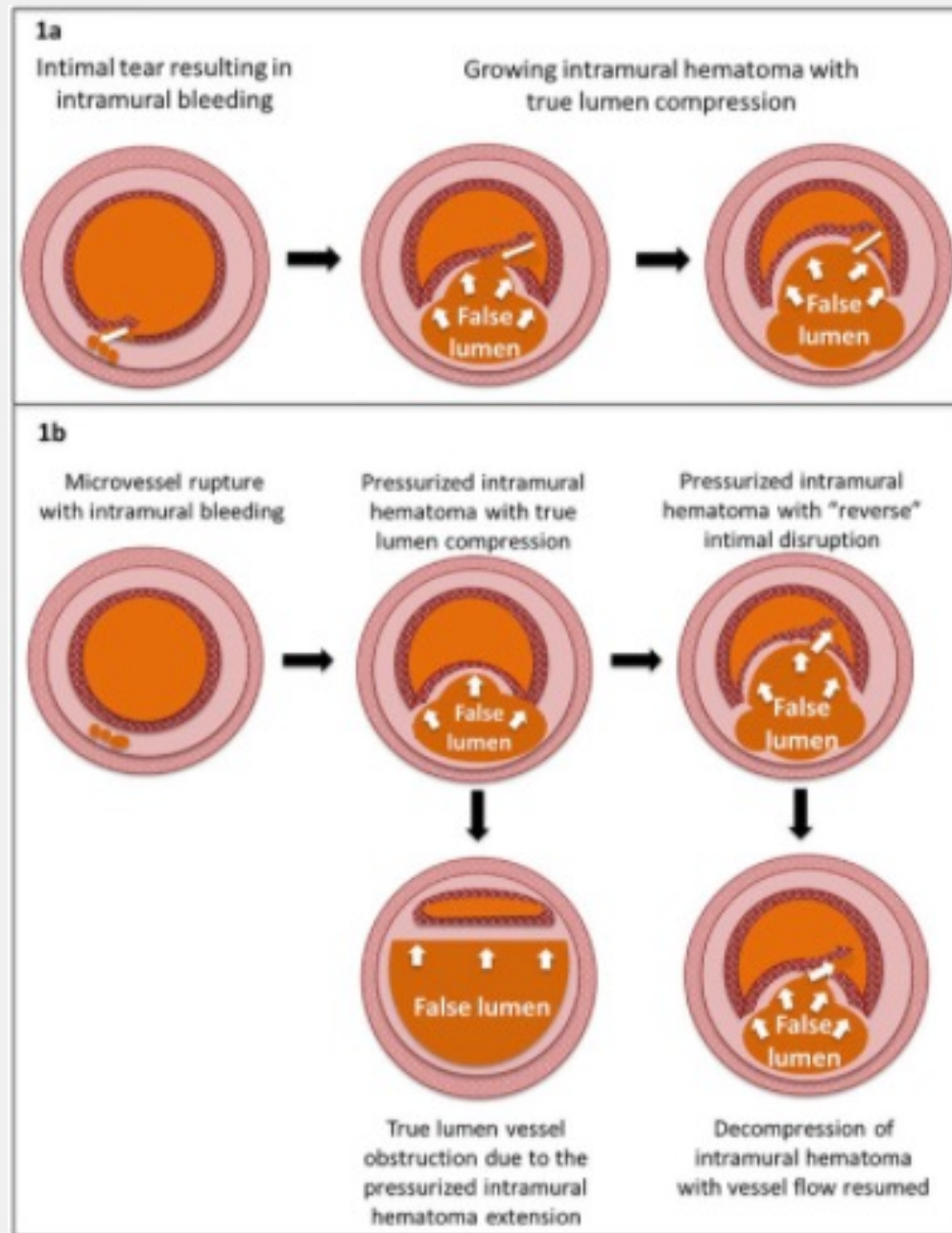
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What is SCAD?

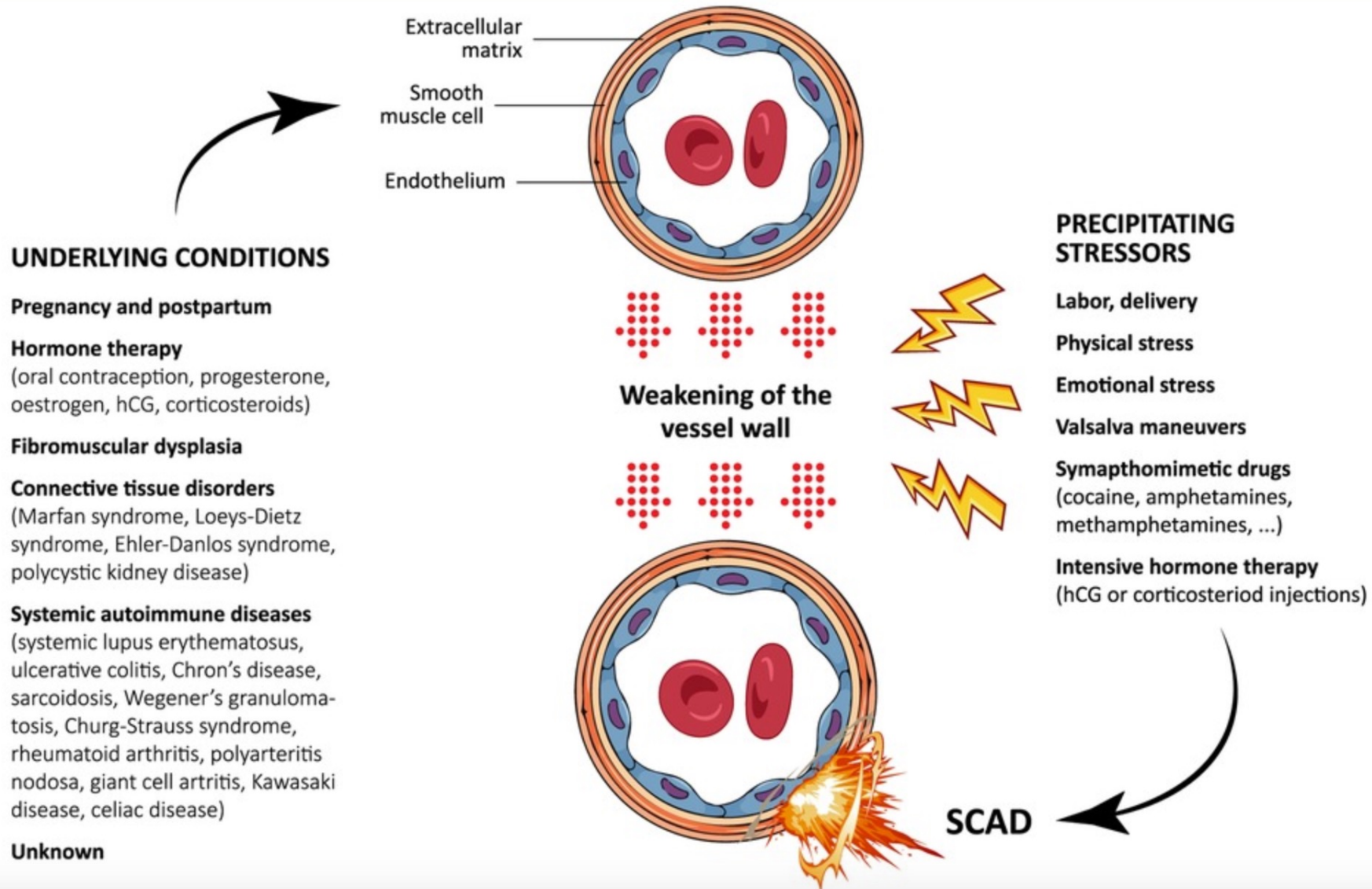
- A cause of non-atherosclerotic acute coronary syndromes leading to myocardial infarction
- Presence of blood entering and separating layers of coronary artery wall to form a false lumen
- Affects a young, **predominantly female** population
- Clinical presentation can include chest pain, STEMI, ventricular fibrillation, sudden death
- Usually precipitated by emotional or physical distress
- Hallmark finding – *dissection of coronary intima or media, hematoma formation within the vessel wall*



Pathophysiology of SCAD

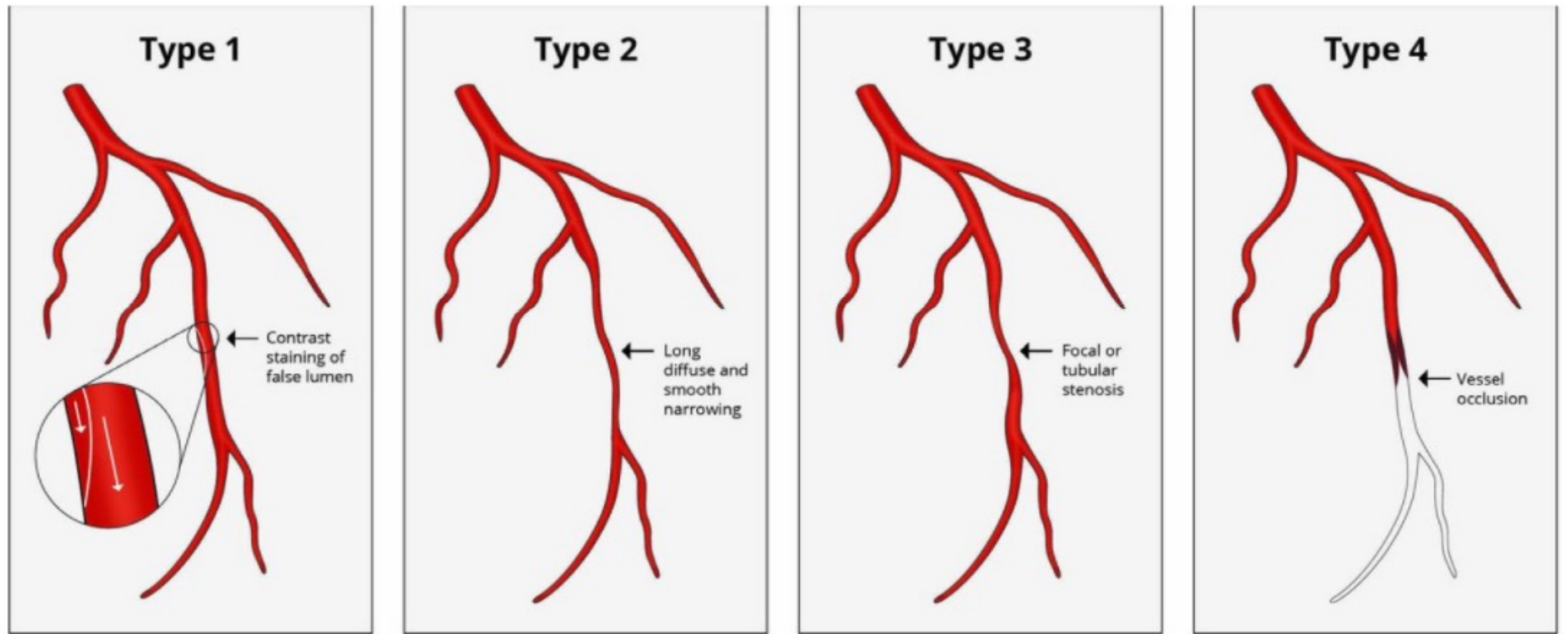


PATHOPHYSIOLOGY OF SCAD



Types of SCAD

Figure 1. Angiographic spontaneous coronary artery dissection classification proposed by Saw et al. [17].



SCAD's effect on women

- SCAD accounts for **1%-4%** of all patients undergoing cardiac catheterization
- **~90%** of the cases affect women
- **24%-40% of acute myocardial infarction in women <50 y.o.**
- Young, physically active females who are otherwise healthy are disproportionately affected by SCAD
- Main cause of ACS during pregnancy (~**40%** of SCAD cases in postpartum women)



Post-SCAD care

Management

- With medications alone rather than PCI

Medications

- Antiplatelets
- Antihypertensives
- Anti-anginals
- Statins if any evidence of atherosclerosis

Imaging

- Evaluate for underlying arteriopathy (head to pelvis angiographic imaging)

Consider genetic testing in

- Recurrent SCAD
- Pregnancy-related SCAD
- Extensive dissection especially involving the left main or proximal coronary arteries

Cardiac Rehab



Why is this important for our female patients?

- Women are more likely to present with atypical symptoms of ACS (shortness of breath, fatigue, backpain, headache, dizziness) →
- SCAD can present with these atypical symptoms →
- SCAD remains underdiagnosed →
- SCAD is seen in young and otherwise healthy females, which may cause SCAD to be pushed farther down the differential if we aren't thinking about it →

Why it's important to have a high index of suspicion for SCAD for young women with concerns for ACS



Our patient outcome

- Discharged on aspirin 81 mg, atorvastatin 80 mg, and metoprolol succinate 25 mg daily
- Assessing for causative conditions such as fibromuscular dysplasia and vascular connective tissue disorders
- Workup outpatient with genetic testing and CTA to assess for fibromuscular dysplasia

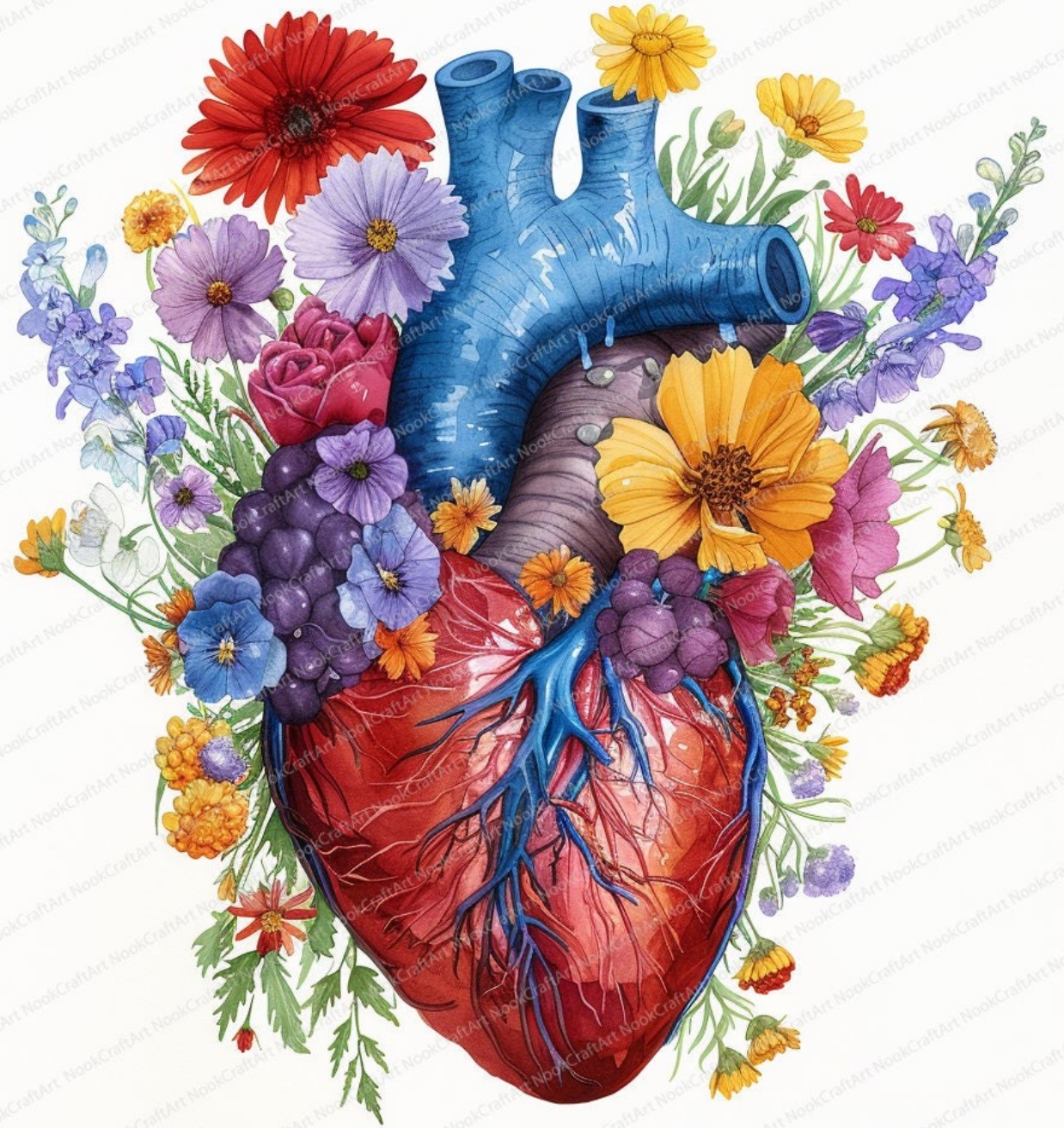


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- <https://www.scielo.br/j/abc/a/6LBrxmZwBqzgcwcCzgQQwzL/?lang=en>
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- <https://www.sciencedirect.com/science/article/pii/S1050173821000037>
- <https://www.mdpi.com/2077-0383/10/24/5925>
- <https://link.springer.com/article/10.1007/s11886-023-01989-1>
- <https://journals.sagepub.com/doi/full/10.1177/1358863X19878265>



Thank you!



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Talk 1: Amr Idris



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Cardiovascular Imaging Through a Sex-Specific Lens

Amr Idris, MD, FACC, FASE, FASNC

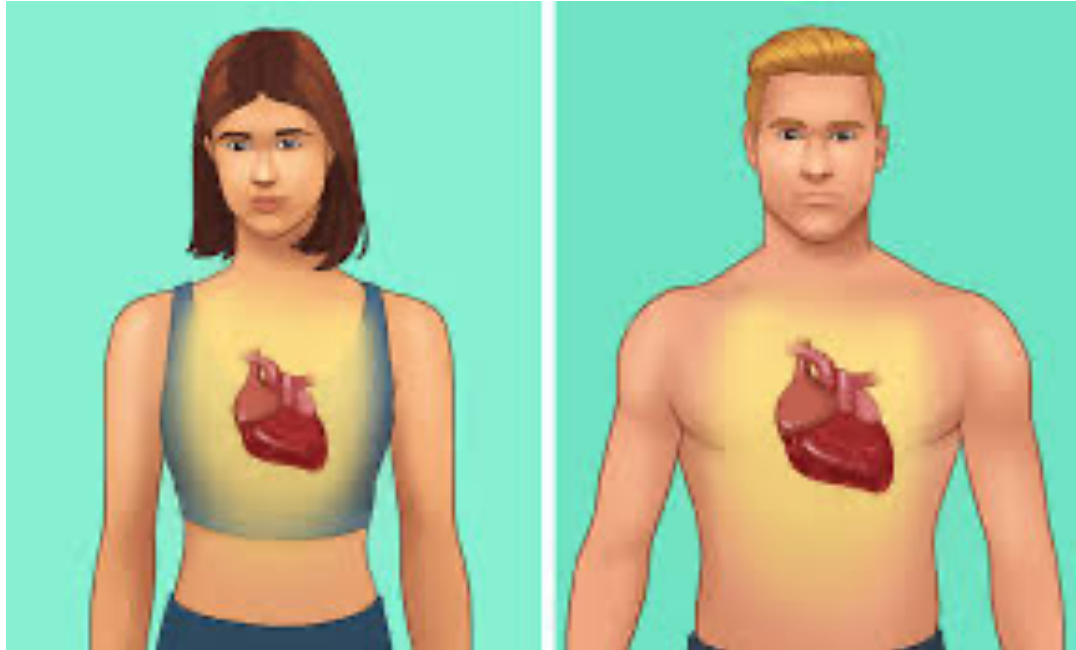
Assistant Professor of Medicine
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Disclosure: No relevant financial disclosures.

Differences exist



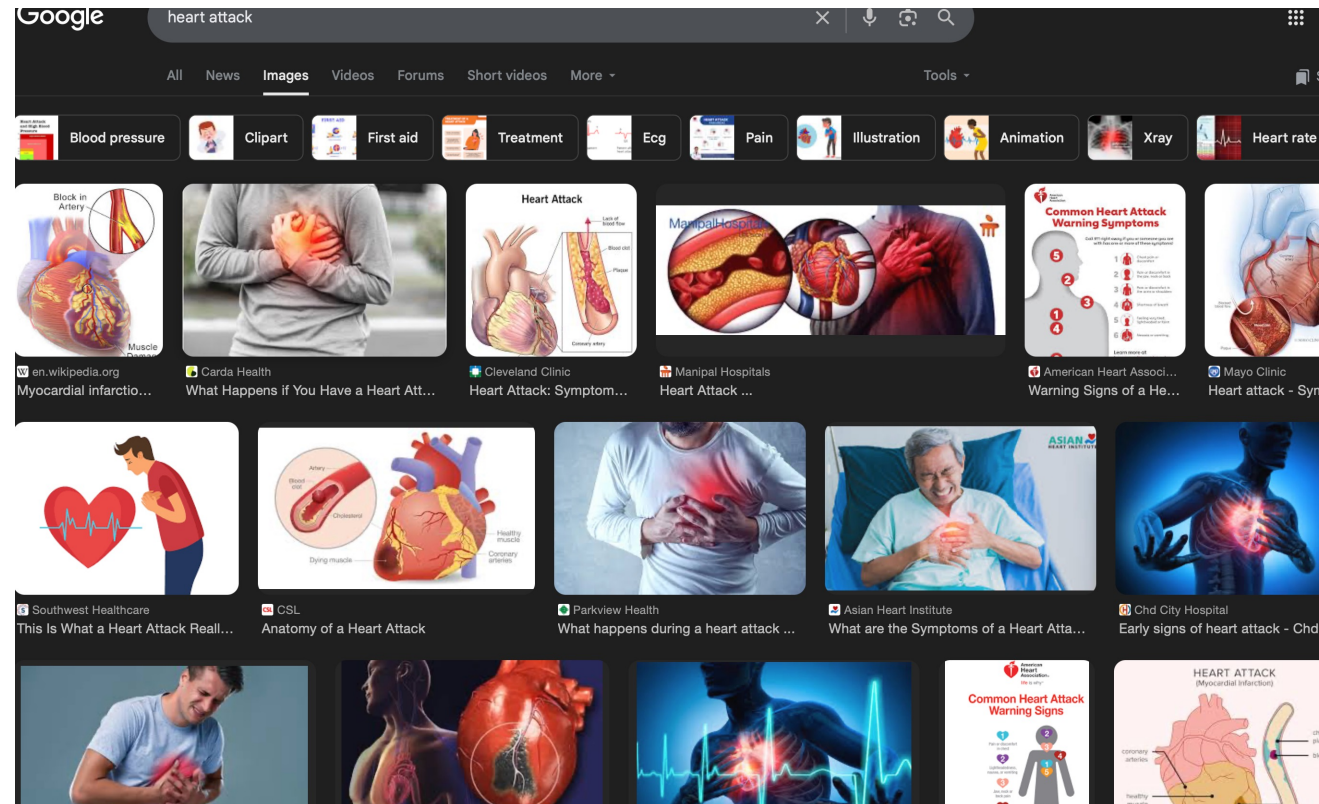
Sex differences impact presentation, diagnosis, and outcomes in cardiovascular disease



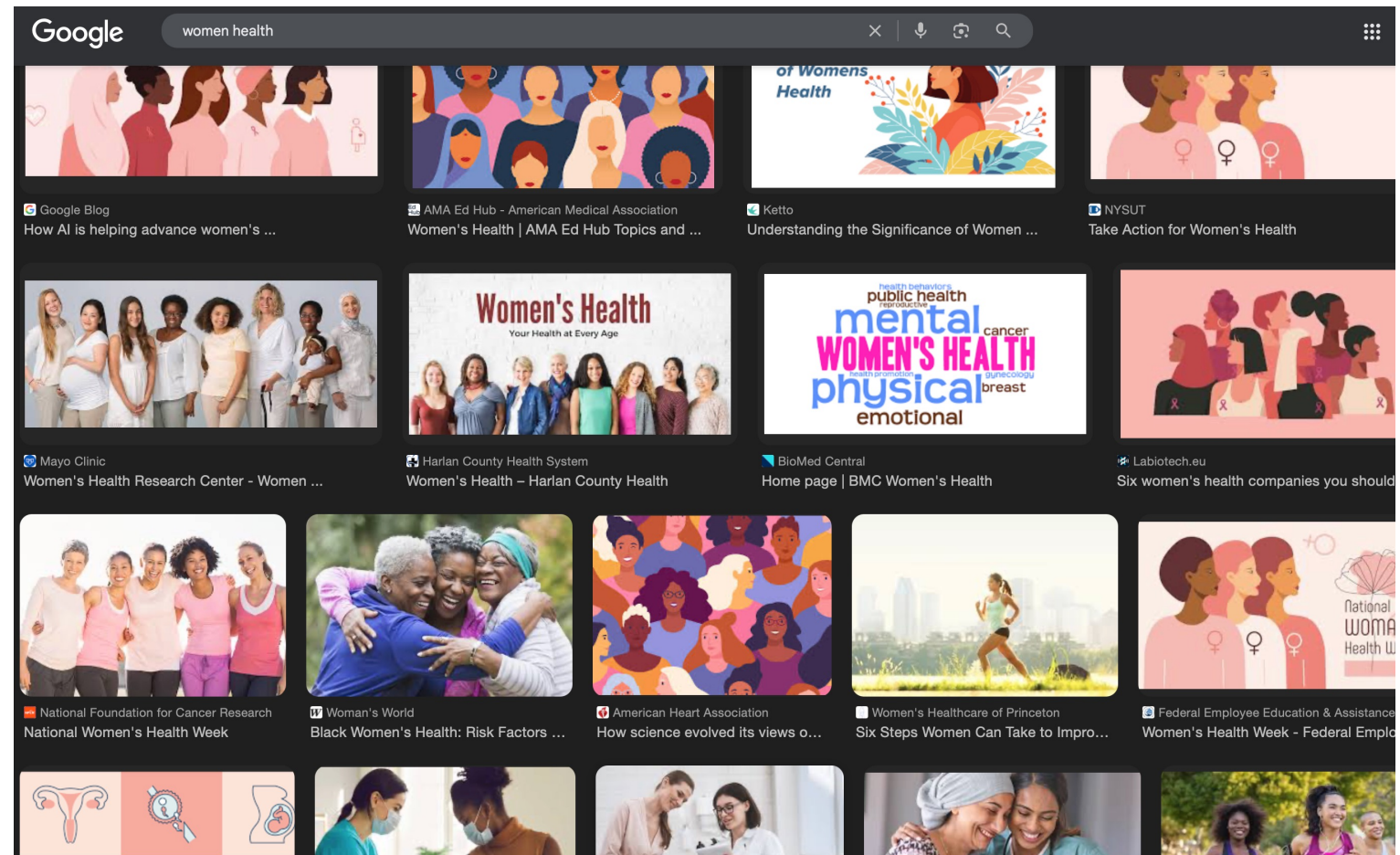
	Author, year	N (women + men), age	Women	Men	Women compared to men
Left ventricle					
LVEDV (ml)	Petersen et al., 2017 [4]	433 + 371, 45–74 years	124 (88–161)	166 (109–218)	↓
	Maicera et al., 2006 [5]	60 + 60, 20–80 years	128 (88–168)	156 (115–198)	↓
	Alfakih et al., 2003 [6]	30 + 30, 20–65 years	135 (96–174)	169 (102–235)	↓
LVEDVi (ml/m ²)	Petersen et al., 2017 [4]	433 + 371, 45–74 years	74 (54–94)	85 (60–110)	↓
	Maicera et al., 2006 [5]	60 + 60, 20–80 years	75 (57–92)	80 (63–98)	↓
	Alfakih et al., 2003 [6]	30 + 30, 20–65 years	78 (56–99)	82 (53–112)	↓
LVESV (ml)	Petersen et al., 2017 [4]	433 + 371, 45–74 years	49 (31–68)	69 (39–97)	↓
	Maceira et al., 2006 [5]	60 + 60, 20–80 years	42 (23–60)	53 (30–75)	↓
	Alfakih et al., 2003 [6]	30 + 30, 20–65 years	49	61	↓
LVESVi (ml/m ²)	Petersen et al., 2017 [4]	433 + 371, 45–74 years	29 (19–40)	36 (21–49)	↓
	Maceira et al., 2006 [5]	60 + 60, 20–80 years	24 (15–34)	27 (16–38)	↓
	Petersen et al., 2017 [4]	433 + 371, 45–74 years	75 (49–100)	96 (59–132)	↓
LVSV (ml)	Maceira et al., 2006 [5]	60 + 60, 20–80 years	86 (58–114)	104 (76–132)	↓
	Alfakih et al., 2003 [6]	30 + 30, 20–65 years	86	108	↓
LVSVi (ml/m ²)	Petersen et al., 2017 [4]	433 + 371, 45–74 years	45 (30–59)	49 (32–67)	↓
	Maceira et al., 2006 [5]	60 + 60, 20–80 years	50 (38–63)	53 (41–65)	↓
	Alfakih et al., 2003 [6]	30 + 30, 20–65 years	50	53	↓
LVEF (%)	Petersen et al., 2017 [4]	433 + 371, 45–74 years	61 (51–70)	58 (48–69)	↑
	Maceira et al., 2006 [5]	60 + 60, 20–80 years	67 (58–76)	67 (58–75)	→
	Alfakih et al., 2003 [6]	30 + 30, 20–65 years	64 (54–74)	64 (55–73)	→
LVM (g)	Petersen et al., 2017 [4]	433 + 371, 45–74 years	70 (46–93)	103 (64–141)	↓
	Maceira et al., 2006 [5]	60 + 60, 20–80 years	108 (72–144)	146 (108–184)	↓
	Alfakih et al., 2003 [6]	30 + 30, 20–65 years	90 (66–114)	133 (85–181)	↓
LVMI (g/m ²)	Petersen et al., 2017 [4]	433 + 371, 45–74 years	42 (29–55)	53 (35–70)	↓
	Maceira et al., 2006 [5]	60 + 60, 20–80 years	63 (48–77)	74 (58–91)	↓
	Alfakih et al., 2003 [6]	30 + 30, 20–65 years	52 (37–67)	65 (46–83)	↓
Right ventricle					
RVEDV (ml)	Petersen et al., 2017 [4]	433 + 371, 45–74 years	130 (85–168)	182 (124–258)	↓
	Maceira et al., 2006 [7]	60 + 60, 20–80 years	126 (84–168)	163 (113–213)	↓
	Alfakih et al., 2003 [6]	30 + 30, 20–65 years	131 (83–178)	177 (111–243)	↓
RVEDVi (ml/m ²)	Petersen et al., 2017 [4]	433 + 371, 45–74 years	77 (53–99)	93 (68–125)	↓
	Maceira et al., 2006 [7]	60 + 60, 20–80 years	73 (55–92)	83 (60–106)	↓
	Alfakih et al., 2003 [6]	30 + 30, 20–65 years	75 (48–103)	86 (58–114)	↓
RVESV (ml)	Petersen et al., 2017 [4]	433 + 371, 45–74 years	55 (27–77)	85 (47–123)	↓
	Maceira et al., 2006 [7]	60 + 60, 20–80 years	43 (17–69)	57 (27–86)	↓
	Alfakih et al., 2003 [6]	30 + 30, 20–65 years	52	79	↓
RVESVi (ml/m ²)	Petersen et al., 2017 [4]	433 + 371, 45–74 years	33 (17–46)	43 (25–63)	↓
	Maceira et al., 2006 [7]	60 + 60, 20–80 years	25 (12–38)	29 (14–43)	↓
RVSV (ml)	Petersen et al., 2017 [4]	433 + 371, 45–74 years	75 (48–99)	97 (68–125)	↓
	Maceira et al., 2006 [7]	60 + 60, 20–80 years	83 (57–108)	106 (72–140)	↓
	Alfakih et al., 2003 [6]	30 + 30, 20–65 years	78	98	↓
RVSVi (ml/m ²)	Petersen et al., 2017 [4]	433 + 371, 45–74 years	45 (30–59)	50 (34–67)	↓

Doctor google on heart attack.

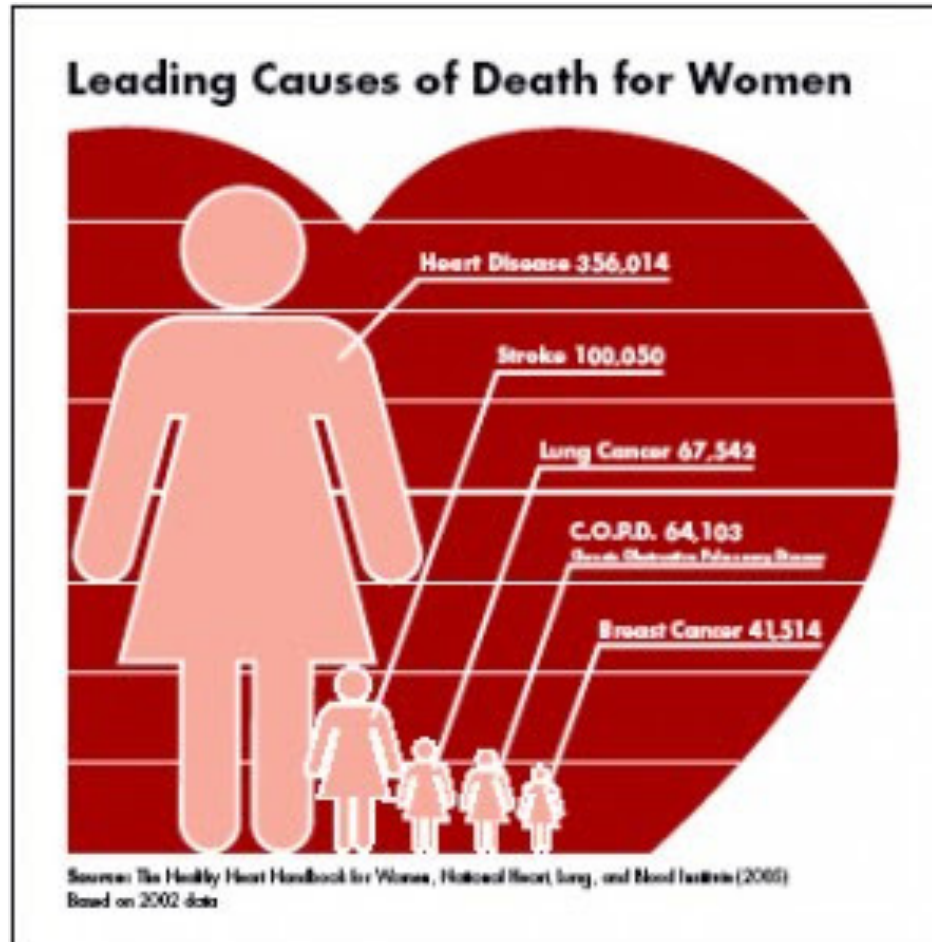
Online searches reflect awareness gaps, women often don't associate chest pain with heart disease



Doctor google on women health.



Epidemiology



- CVD affect ~ 60 million US women
- Breast cancer affects ~ 3.5 million US women

Clinical Presentation and Diagnosis

Women are less likely to have obstructive CAD

Impact of Ethnicity and Gender Differences on Angiographic Coronary Artery Disease Prevalence and In-Hospital Mortality in the American College of Cardiology–National Cardiovascular Data Registry

Leslee J. Shaw, PhD, Richard E. Shaw, PhD, C. Noel Bairey Merz, MD, Ralph G. Brindis, MD, Lloyd W. Klein, MD, Brahmajee Nallamothu, MD, Pamela S. Douglas, MD, ... [SHOW ALL](#) ... , and on behalf of the American College of Cardiology–National Cardiovascular Data Registry

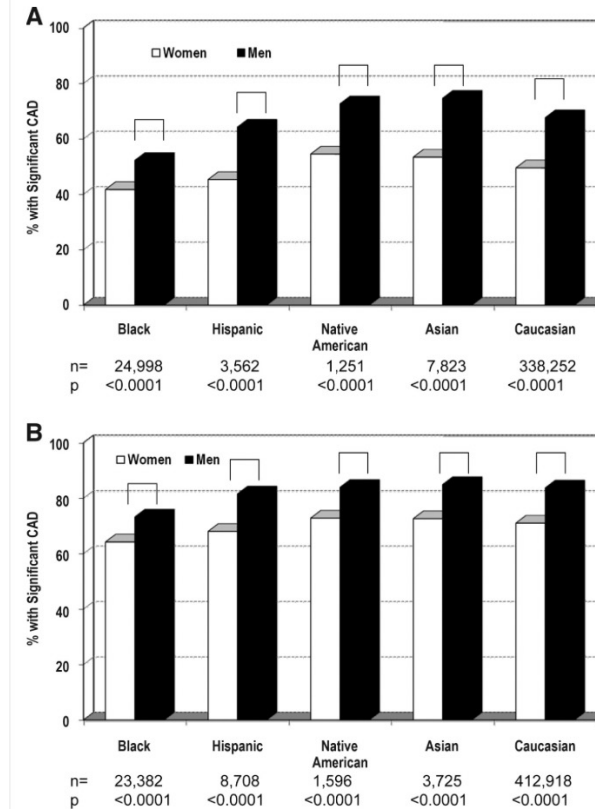


Figure 2. A, Observed frequency of significant CAD (defined as $\geq 70\%$ stenosis in 1 or more epicardial coronary arteries) by gender and ethnicity in patients presenting with suspected ischemic heart disease with stable chest pain symptoms (n=375 886). B, Observed frequency of significant CAD by gender and ethnicity in patients presenting with ACS (n=450 329).

Women presenting with CP were less likely to have obstructive CAD compared with men

Stable chest pain

ACS

CONFIRM trial

Is any plaque ok?

JACC Journals › JACC › Archives › Vol. 58 No. 8

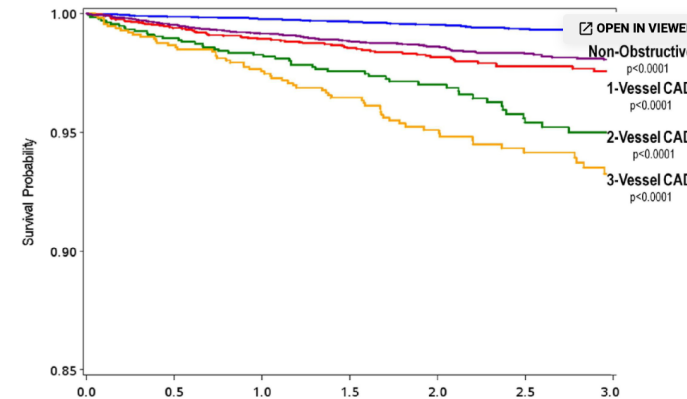
FREE ACCESS | Cardiac Imaging | 9 August 2011

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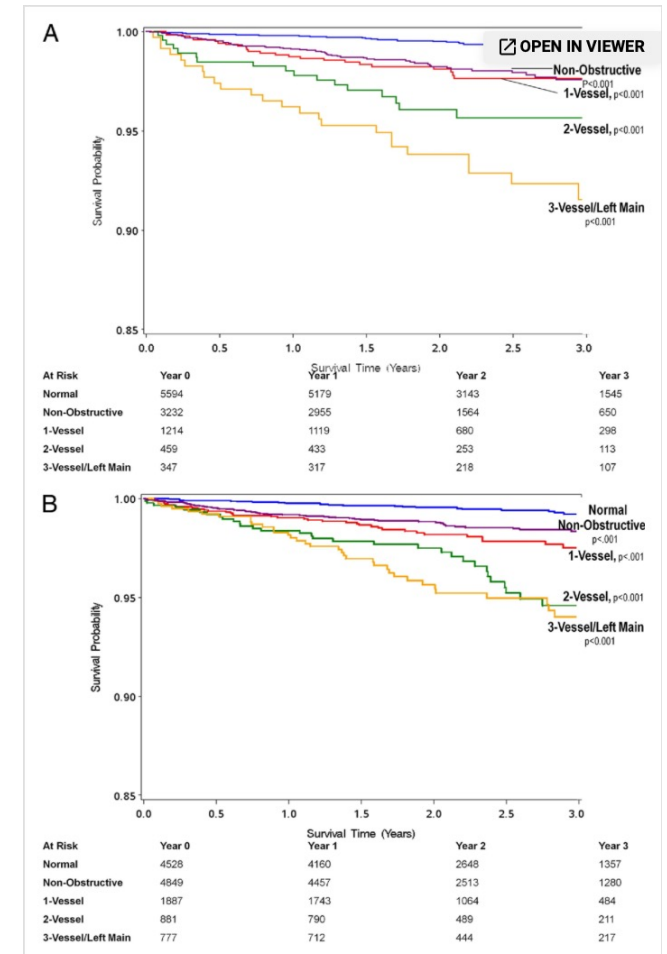
Age- and Sex-Related Differences in All-Cause Mortality Risk Based on Coronary Computed Tomography Angiography Findings: Results From the International Multicenter CONFIRM (Coronary CT Angiography Evaluation for Clinical Outcomes: An International Multicenter Registry) of 23,854 Patients Without Known Coronary Artery Disease

Cardiac Imaging: Editorial Comment: Coronary Computed Tomography Angiography
Letter to the Editor: Coronary Computed Tomography Angiography Versus Coronary Calcium Computed Tomography for Prognosis With Regard to Mortality

Authors: James K. Min, MD, Allison Dunning, MS, Fay Y. Lin, MD, Stephan Achenbach, MD, Mouaz Al-Mallah, MD, Matthew J. Budoff, MD, Filippo Cademartiri, MD, ... [SHOW ALL](#) ... CONFIRM Investigators | [AUTHORS INFO & AFFILIATIONS](#)



At Risk	Year 0	Year 1	Year 2	Year 3
Normal	10146	9357	5800	2907
Non-Obstructive	8114	7437	4081	1930
1-Vessel	3118	2873	1747	782
2-Vessel	1346	1228	742	324
3-Vessel/Left Main	1130	1034	664	324



Calcified plaque on CAC score

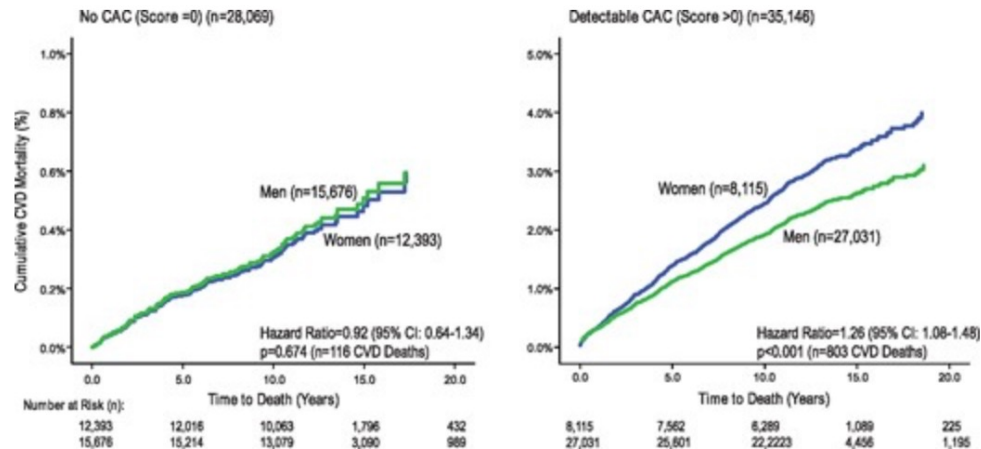
JOURNAL ARTICLE

Sex differences in calcified plaque and long-term cardiovascular mortality: observations from the CAC Consortium ^{FREE}

Leslee J Shaw ✉, James K Min, Khurram Nasir, Joe X Xie, Daniel S Berman, Michael D Miedema, Seamus P Whelton, Zeina A Dardari, Alan Rozanski, John Rumberger ... [Show more](#)

European Heart Journal, Volume 39, Issue 41, 01 November 2018, Pages 3727–3735, <https://doi.org/10.1093/eurheartj/ehy534>

Published: 12 September 2018 [Article history](#) ▼

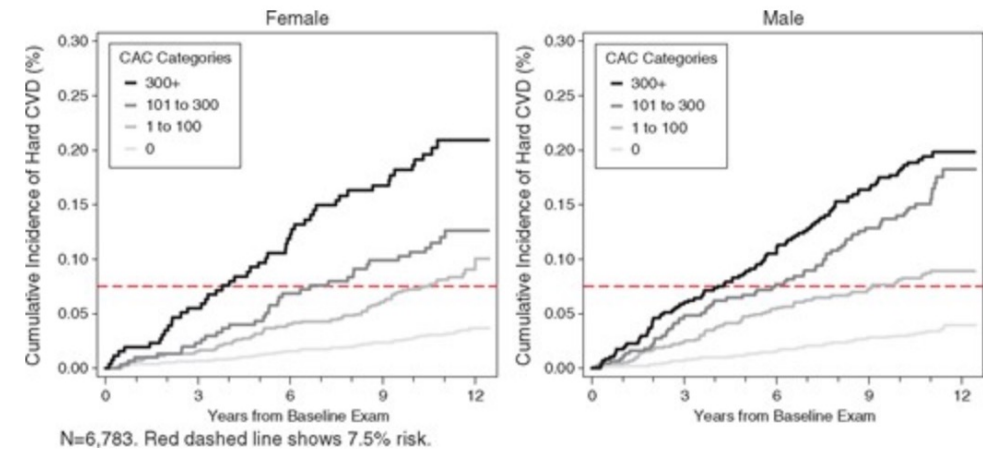


JOURNAL ARTICLE

Ten-year association of coronary artery calcium with atherosclerotic cardiovascular disease (ASCVD) events: the multi-ethnic study of atherosclerosis (MESA) ^{FREE}

Matthew J Budoff ✉, Rebekah Young, Gregory Burke, J Jeffrey Carr, Robert C Detrano, Aaron R Folsom, Richard Kronmal, Joao A C Lima, Kiang J Liu, Robyn L McClelland ... [Show more](#)

European Heart Journal, Volume 39, Issue 25, 01 July 2018, Pages 2401–2408, <https://doi.org/10.1093/eurheartj/ehy217>

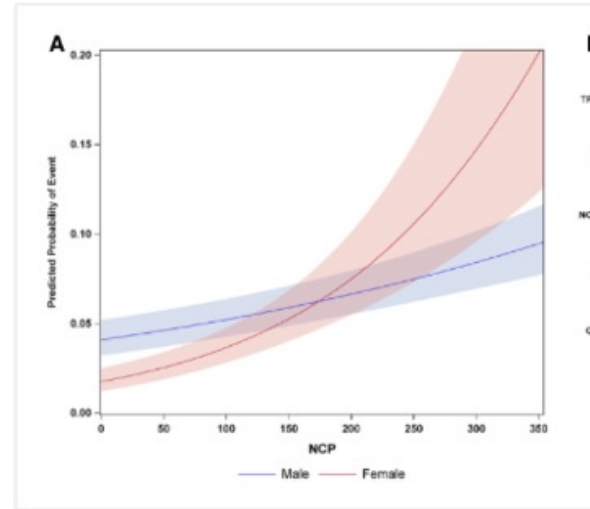


Women have higher mortality than men for any detectable calcified plaque.

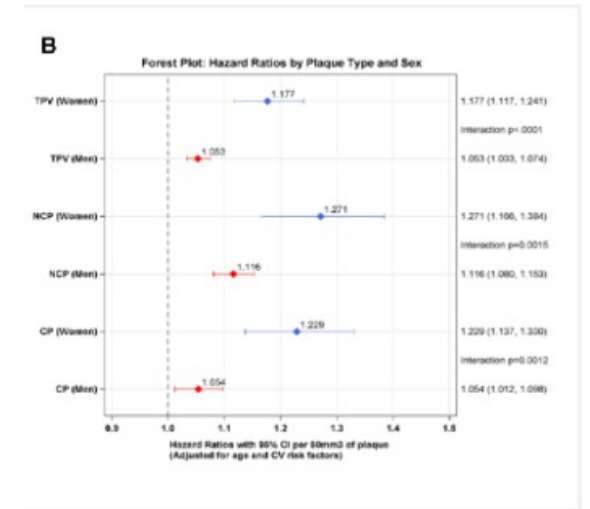
AI-Quantitative CT Coronary Plaque Features Associate With a Higher Relative Risk in Women: CONFIRM2 Registry

Gudrun M. Feuchtnr, MD , Pietro G. Lacaita, MD, Jeroen J. Bax, MD, PhD , Fatima Rodriguez, MD, MPH , Rine Nakanishi, MD , Gianluca Pontone, MD, PhD , Saima Mushtaq, MD , ... [SHOW ALL](#) ... , and Alexander van Rosendaal, MD, PhD | [AUTHOR INFO & AFFILIATIONS](#)

AI-based quantitative coronary CT (AI-QCT) reveals greater risk with increasing plaque volume, especially in women.



- The probability of MACE increased more steeply for women than men with increasing non-calcified plaque volume above 180 mm³.



- Hazard ratios per 50-mm³ increment. plaque volume increase for risk of MACE in women vs men (adjusted for age and cardiovascular [CV] risk factors).

AI-based plaque quantification may bridge the diagnostic gap in women

CAD in women

JACC Journals > JACC > Archives > Vol. 66 No. 17

 **FREE ACCESS** | State-of-the-Art Review | 19 October 2015



Emergence of Nonobstructive Coronary Artery Disease: A Woman's Problem and Need for Change in Definition on Angiography

3rd party ad content

Letters: Association Between Migraine Headache and Cardiac Syndrome X

Authors: Carl J. Pepine, MD , Keith C. Ferdinand, MD, Leslee J. Shaw, PhD, Kelly Ann Light-McGroary, MD, Rashmee U. Shah, MD, MS, Martha Gulati, MD, MS, Claire Duvernoy, MD, Mary Norine Walsh, MD, and C. Noel Bairey Merz, MD ACC CVD in Women Committee | [AUTHORS INFO & AFFILIATIONS](#)



NONOBSTRUCTIVE CAD

- Prevalence Women > Men
- Predominantly younger, middle-aged women
- Preserved LV systolic function
- Possible plaque erosion with subsequent thrombus formation
- Often multiple mechanisms for ischemia
- Associated with heightened risk for adverse outcomes

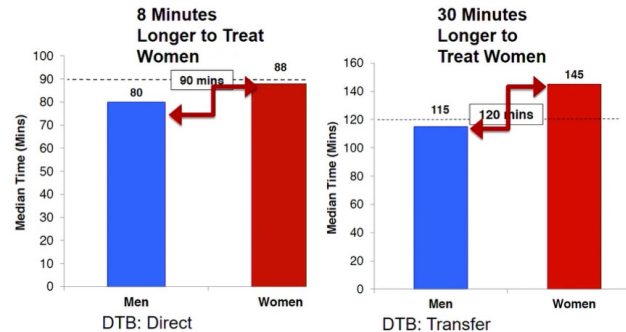
Sex Differences in Medical Care and Early Death After Acute Myocardial Infarction

Table 3. Adjusted ORs* for Clinical Performance Measures, Invasive Procedures, and In-Hospital Death

Measure/Treatment/Outcome	n	Adjusted OR (95% CI) (Women vs Men)	P
Early medical therapy			
Aspirin within 24 h	70/360	0.86 (0.81–0.90)	<0.0001
β-Blocker within 24 h	64/681	0.90 (0.86–0.93)	<0.0001
Invasive procedures			
Cardiac catheterization	74/769	0.91 (0.88–0.94)	<0.0001
PCI	67/477	0.78 (0.74–0.81)	<0.0001
CABG	67/477	0.60 (0.55–0.65)	<0.0001
Revascularization	67/477	0.68 (0.65–0.71)	<0.0001
Acute reperfusion and timeliness of reperfusion†			
DTN ≤30 min	2807	0.78 (0.65–0.92)	0.004
DTB ≤90 min	7673	0.87 (0.79–0.95)	0.004
Reperfusion therapy	24/742	0.75 (0.70–0.80)	<0.0001
Primary PCI	24/742	0.83 (0.78–0.87)	<0.0001
Fibrinolytic therapy	24/742	0.87 (0.81–0.93)	<0.0001
In-hospital death			
Overall AMI cohort	70/105	1.04 (0.99–1.10)	0.1
STEMI subpopulation	23/015	1.12 (1.02–1.23)	0.015

*ORs, which are for women vs men, were adjusted for age, race, BMI, insurance type, systolic blood pressure, cardiac diagnosis, initial ECG with diagnostic ST-segment elevation or left bundle-branch block, diabetes, hypertension, hyperlipidemia, heart failure, previous MI, peripheral vascular disease, renal insufficiency, stroke, chronic obstructive pulmonary disease, and adult history of smoking. The generalized estimation equations approach was also employed to adjust for clustering within hospitals.

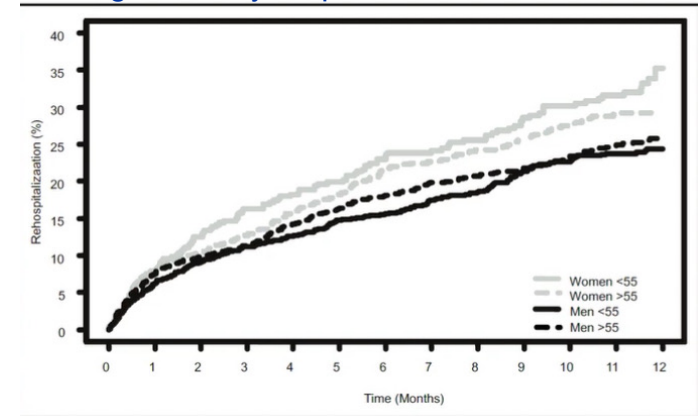
†STEMI subpopulation.



Virgo study. Delays in care.

	Women (n=2009)	Men (n=976)	P*
Decision to seek medical care, %			
Patient did not perceive cause of symptoms to be heart-related, %	54.7	52.3	0.379
Perceived reason, %			
Indigestion or acid reflux	42.8	49.4	0.076
Muscle pain	15.4	21.2	0.029
Stress/anxiety	20.9	11.8	<0.001
Stomach illness or flu	11.6	9.8	0.592
Asthma	10.7	8.0	0.281
Fatigue	5.9	5.7	0.856
Diabetes mellitus	4.5	2.0	0.076
Other cause	8.9	6.3	0.281
Time to hospital presentation, %			
≤2 h	32.9	38.1	0.002
>2–6 h	15.2	18.5	
>6 h	38.7	31.8	
Median time (IQR), h	3.2 (0.8–21.2)	2.4 (0.7–13.0)	0.004

Women of all ages have higher risk of rehospitalization during the first year post-AMI.



	Women (n=2009)	Men (n=976)	P*
Sought medical care for similar symptoms, %			
Provider did not think symptoms were heart-related, %	53.4	36.7	<0.001
Perceived cause of symptom, %			
Indigestion or acid reflux	29.1	40.5	0.401
Stress/anxiety	25.0	15.2	0.401
Muscle pain	13.3	15.2	1
Asthma	14.9	10.1	1
Stomach illness or flu	5.1	3.8	1
Diabetes mellitus	5.7	2.5	1
Fatigue	5.1	0.0	0.401
Other	7.3	3.8	1
Among those with suspected symptoms of heart disease, %			
Tested for a heart condition	89.1	89.0	0.589
Test showed evidence of a heart condition	57.4	56.6	0.268

- Women report >3 additional non-chest pain symptoms compared with men.
- Women who sought care before hospitalization were less likely to be told their symptoms were cardiac-related

- Physician response to symptoms

Do Current Guidelines Adequately Address Women?

2012 ACCF/AHA/ACP/AATS/PCNA/SCAI/STS Guideline for the Diagnosis and Management of Patients With Stable Ischemic Heart Disease

A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, and the American College of Physicians, American Association for Thoracic Surgery, Preventive Cardiovascular Nurses Association, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons

- The goal of diagnostic testing is to detect obstructive CAD

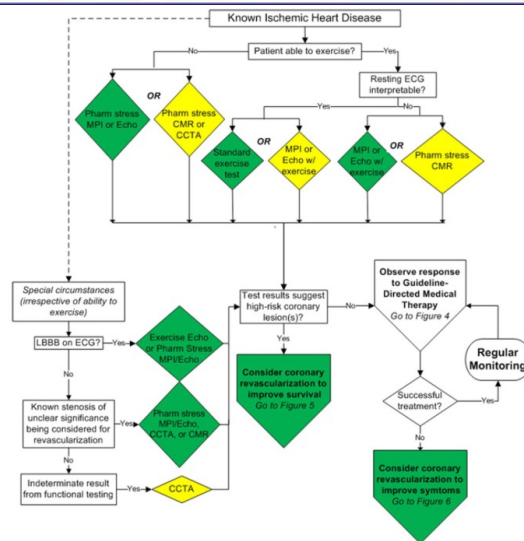


Figure 3. Algorithm for risk assessment of patients with SIHD.* *Colors correspond to the class of recommendations in the ACCF/AHA Table 1. The algorithms do not represent a comprehensive list of recommendations (see text for all recommendations). CCTA indicates coronary computed tomography angiography; CMR, cardiac magnetic resonance; ECG, electrocardiogram; Echo, echocardiography; LBBB, left bundle-branch block; MPI, myocardial perfusion imaging; and Pharm, pharmacological.

2021

AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR Guideline for the Evaluation and Diagnosis of Chest Pain: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines

Martha Gulati, MD, MS, FACC, FAHA, Chair, Phillip D. Levy, MD, MPH, FACC, FAHA, Vice Chair, Debabrata Mukherjee, MD, MS, FACC, FAHA, Vice Chair, Ezra Amsterdam, MD, FACC, Deepak L. Bhatt, MD, MPH, FACC, FAHA, Kim K. Birtcher, MS, PharmD, AACC, Ron Blankstein, MD, FACC, MSCCT, ... [SHOW ALL](#) ... and Leslee J. Shaw, PhD, FACC, FAHA, MSCCT | [AUTHOR](#)

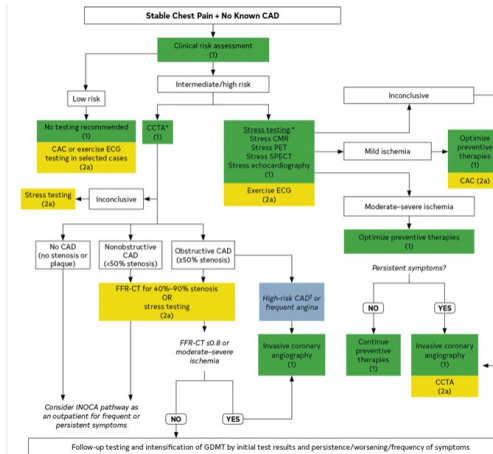
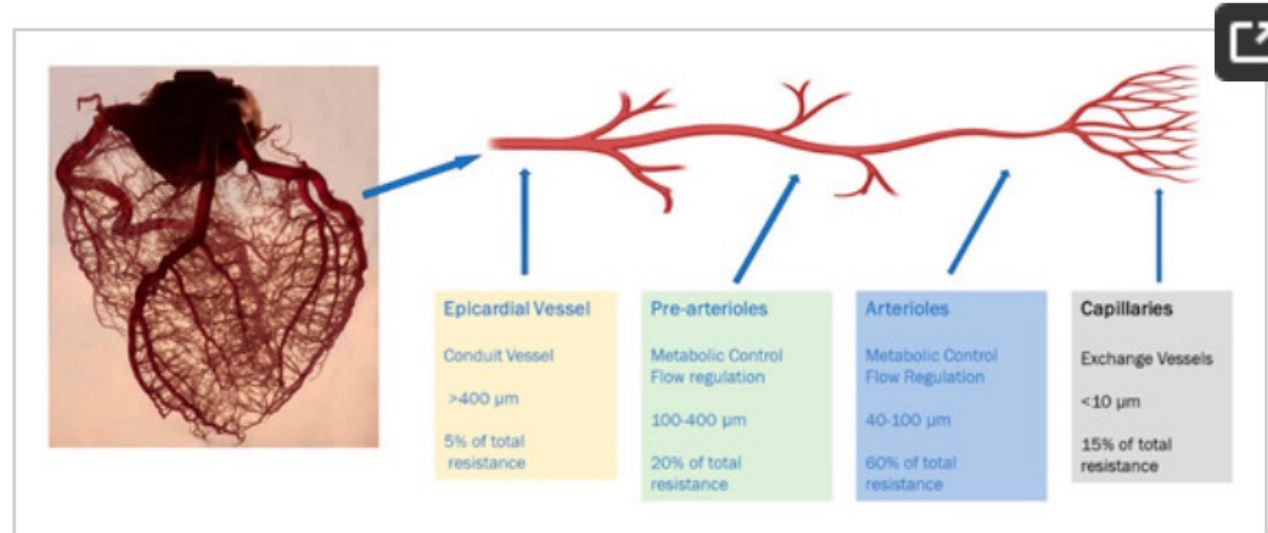


Figure 12. Clinical Decision Pathway for Patients With Stable Chest Pain and No Known CAD Test choice should be guided by local availability and expertise. *Test choice guided by patient's exercise capacity, resting electrocardiographic abnormalities; CCTA preferable in those <65 years of age and not on optimal preventive therapies; stress testing favored in those ≥65 years of age (with a higher likelihood of ischemia). †High-risk CAD means left main stenosis ≥50%; anatomically significant 3-vessel disease (≥70% stenosis). CAD indicates coronary artery disease; CCTA, coronary CT angiography; CMR, cardiovascular magnetic resonance imaging; CT, computed tomography; FFR-CT, fractional flow reserve with CT; GDMT, guideline-directed medical therapy; INOCA, ischemia and no obstructive CAD; PET, positron emission tomography; and SPECT, single-photon emission CT.

Beyond Obstructive CAD: The Role of Microvascular Disease

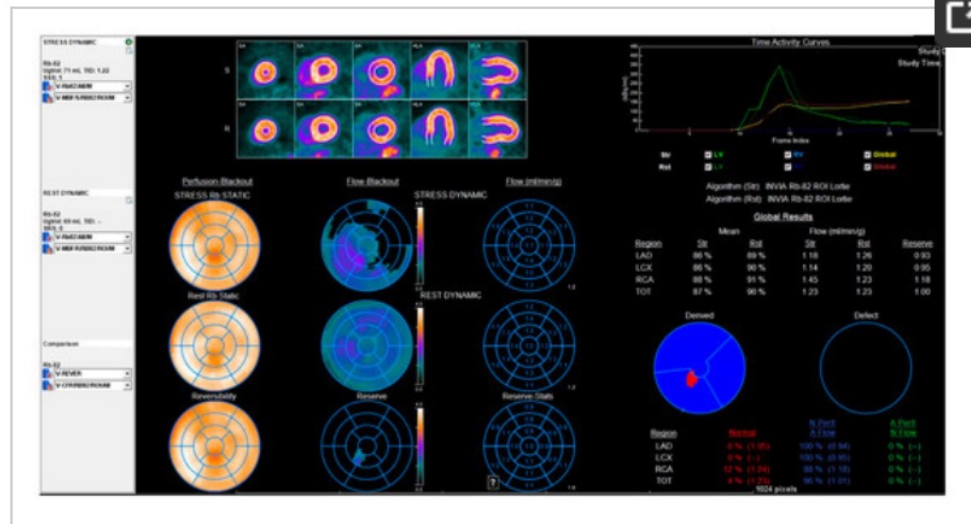
How about patients with non obstructive disease with symptoms?



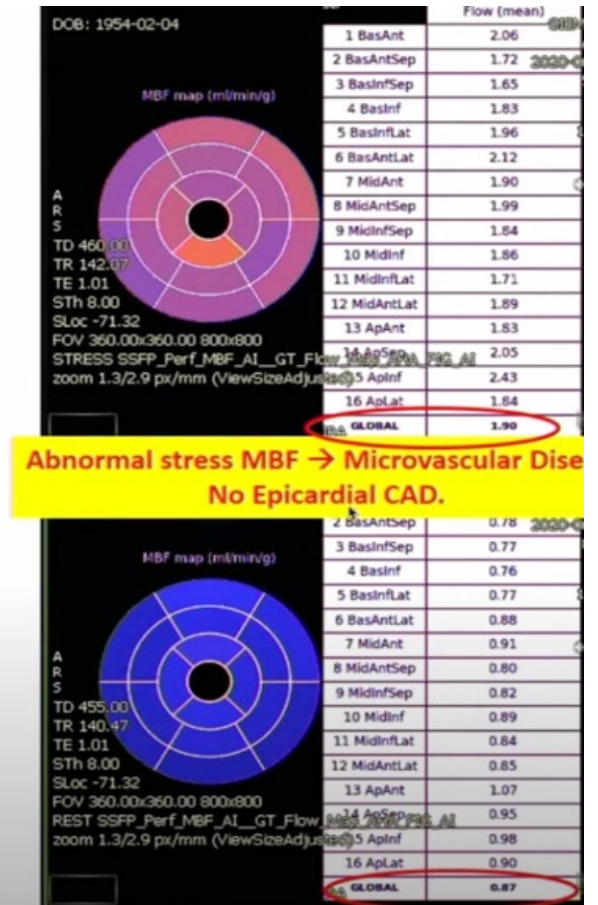
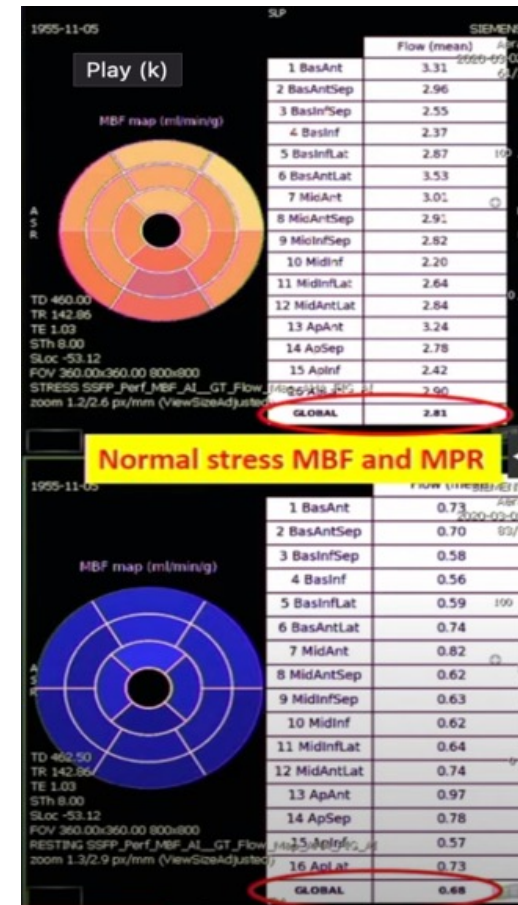
- Refractory angina with non obstructive disease despite GDMT could be related to microvascular disease.

Microvascular disease case: PET

Mild epicardial CAD on invasive angiography with abnormal global myocardial perfusion reserve (MPR <2), consistent with coronary microvascular dysfunction



Cardiac MRI confirms perfusion abnormalities consistent with microvascular dysfunction

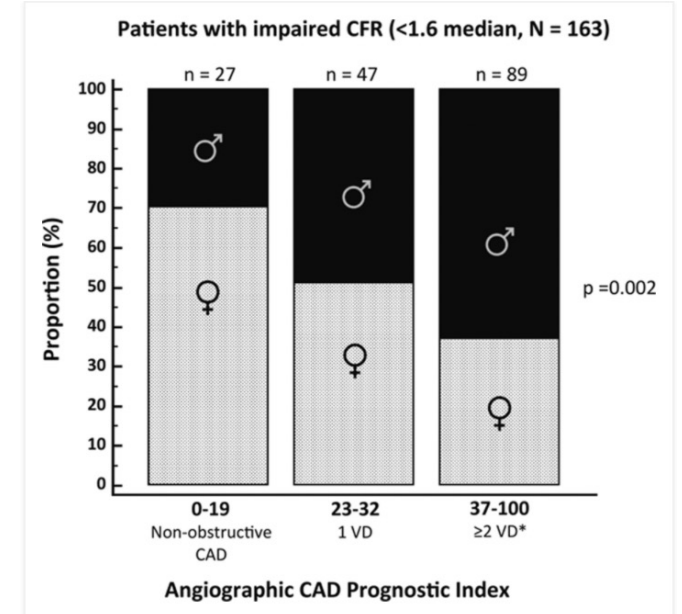
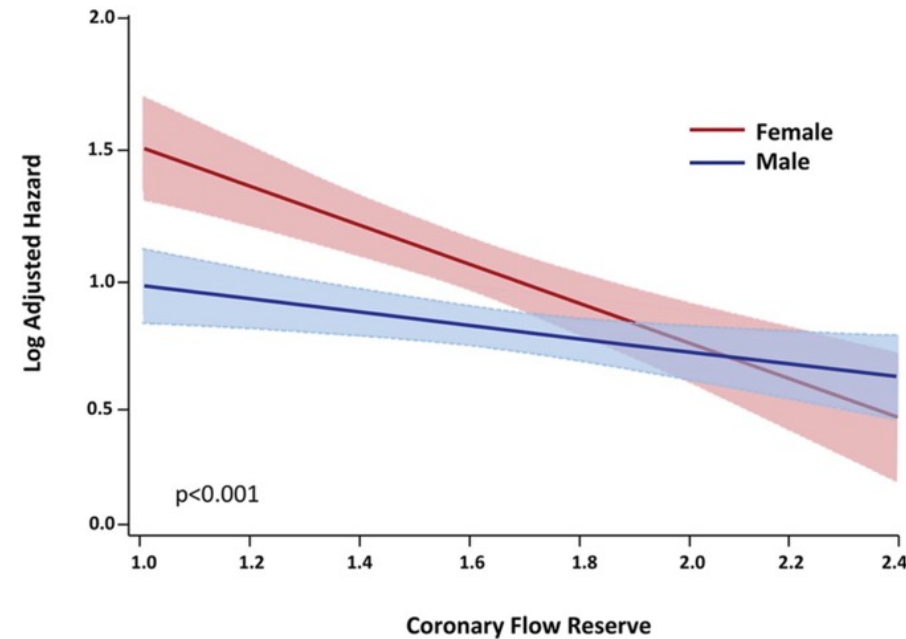


Prognosis CFR

RESEARCH ARTICLE | Originally Published 14 November 2016 | [Check for updates](#)

Excess Cardiovascular Risk in Women Relative to Men Referred for Coronary Angiography Is Associated With Severely Impaired Coronary Flow Reserve, Not Obstructive Disease

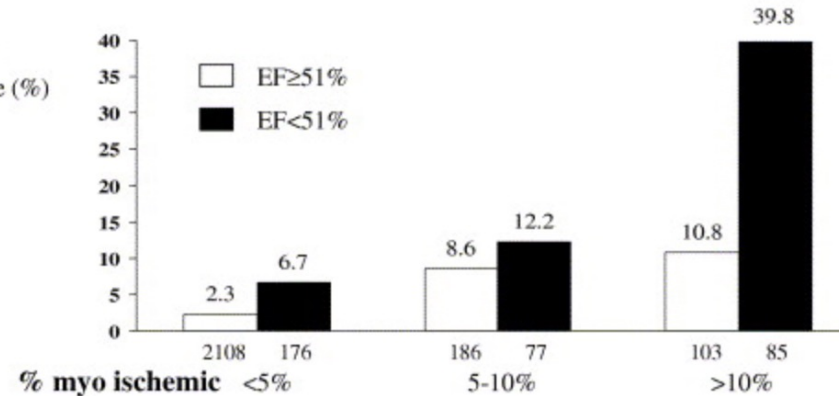
Viviany R. Taqueti, MD, MPH, Leslee J. Shaw, PhD, Nancy R. Cook, ScD, Venkatesh L. Murthy, MD, PhD, Nishant R. Shah, MD, MPH, Courtney R. Foster, MS, Jon Hainer, BS, Ron Blankstein, MD, Sharmila Dorbala, MD, MPH, and Marcelo F. Di Carli, MD | [AUTHOR INFO &](#)



Prognostic Role of Perfusion Imaging in Women

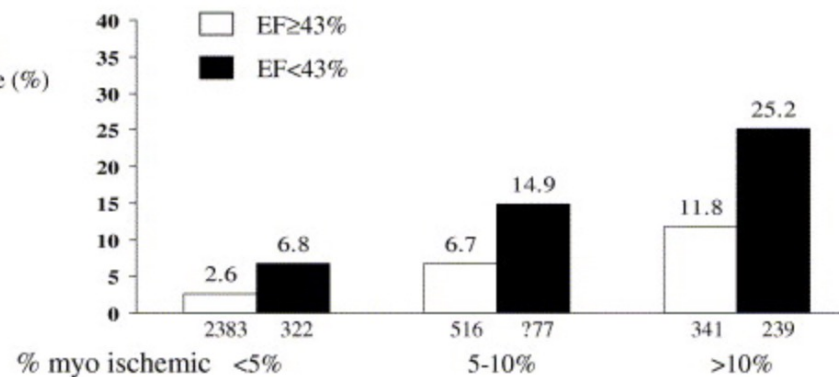
A women

3-year
Adjusted
Event Rate (%)



B men

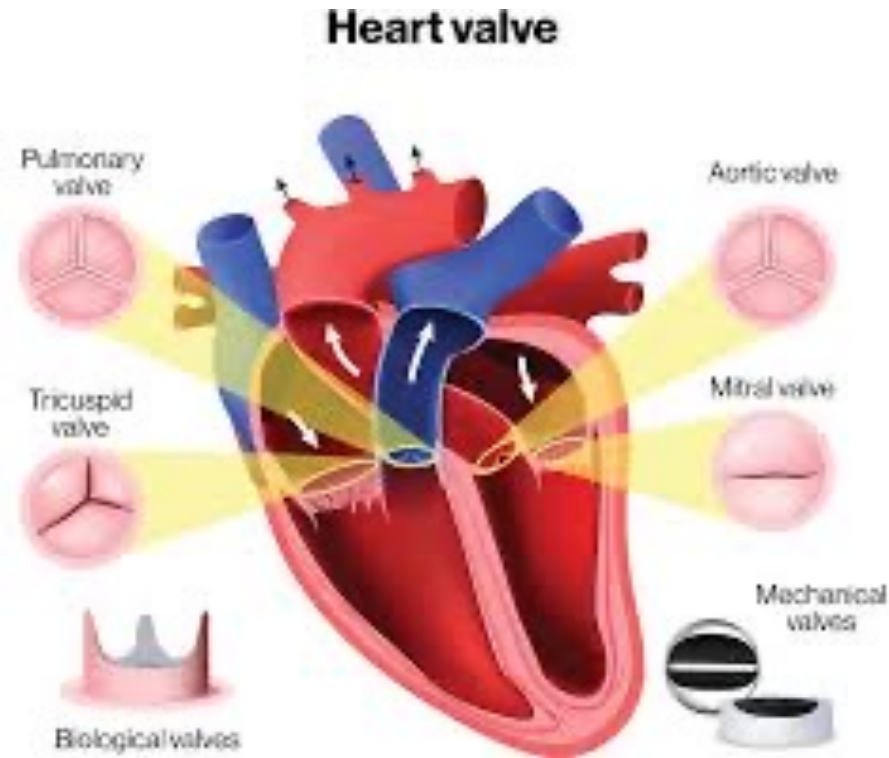
3-year
Adjusted
Event Rate (%)



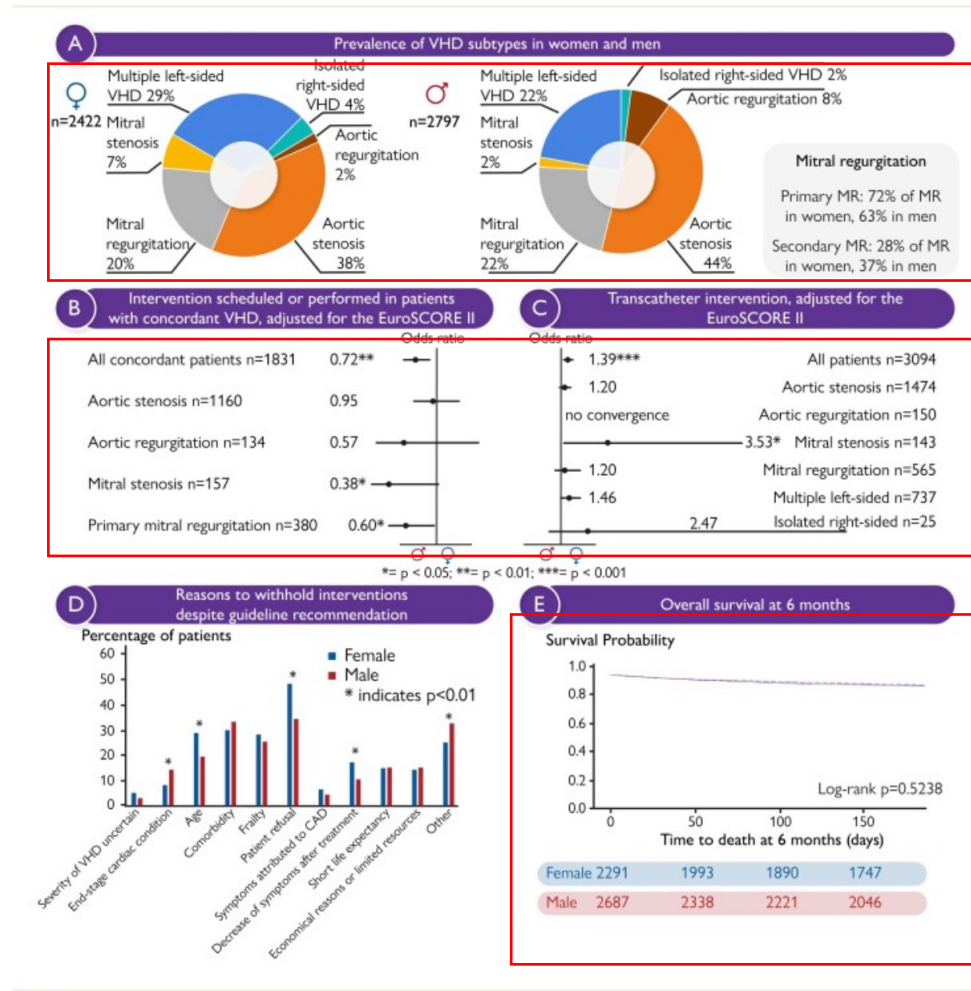
Perfusion added more power to predict adverse events in women

Sex Differences in Valvular Heart Disease

How about valvular heart disease in women? Any difference?



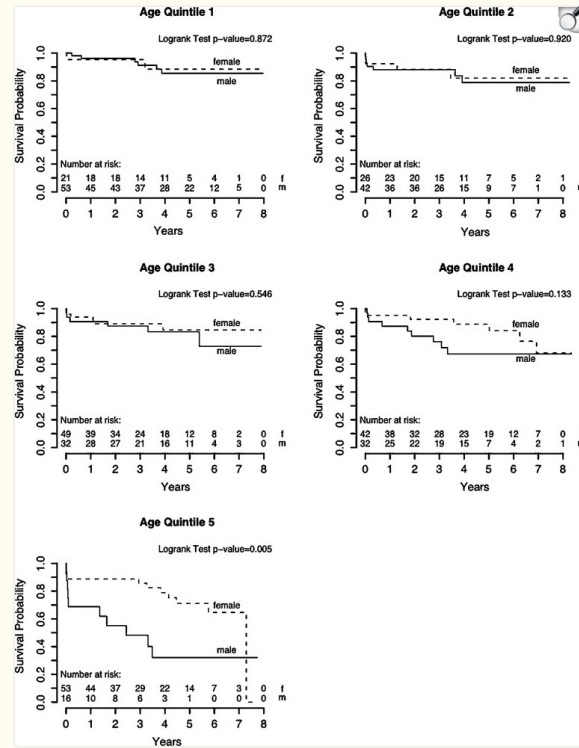
Difference between Male and female in valvular heart disease



Sex differences exist in valve morphology, presentation, and outcomes

Mortality after SAVR or TAVR

Fig. 1.

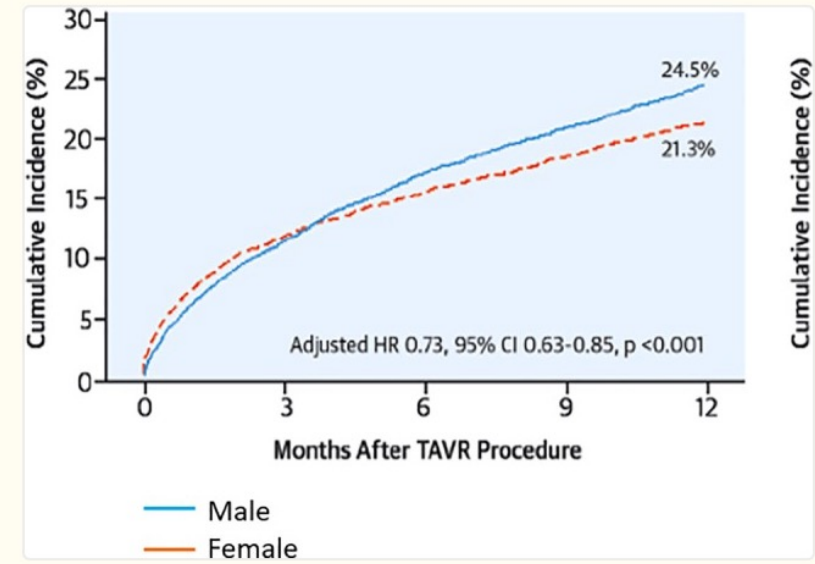


[Open in a new tab](#)

Outcomes following surgical aortic valve replacement. Survival rates after surgical aortic valve replacement are comparable between sexes across age groups apart from the fifth age quintile (78 years or older), where women showed superior survival as compared to men. (Figure printed with permission from Fuchs et al. [14])

- No gender difference in mortality in either.

Fig. 2.



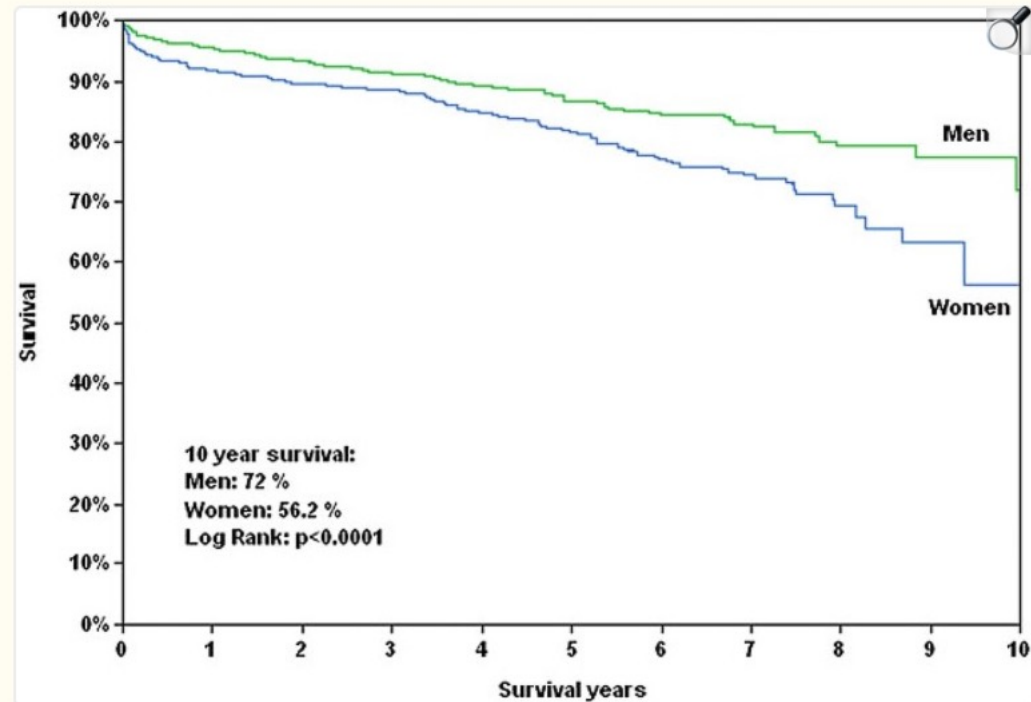
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Differences in outcomes following transcatheter aortic valve replacement. Women with severe aortic stenosis (AS) experience lower mortality rates compared to men following trans-catheter aortic valve replacement (TAVR). HR hazard ratio, CI confidence interval. (Figure printed with permission from Chandrasekhar et al. [14])

Mortality after Mitral valve procedure

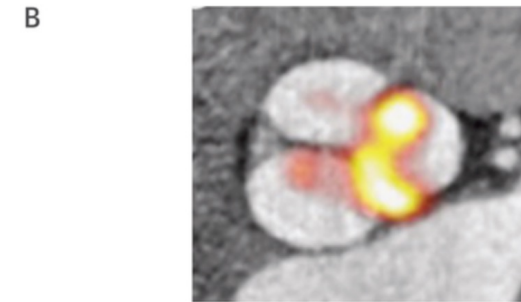
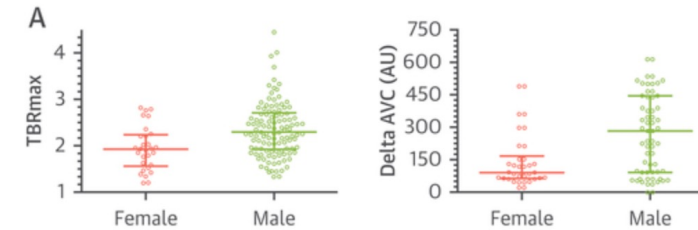
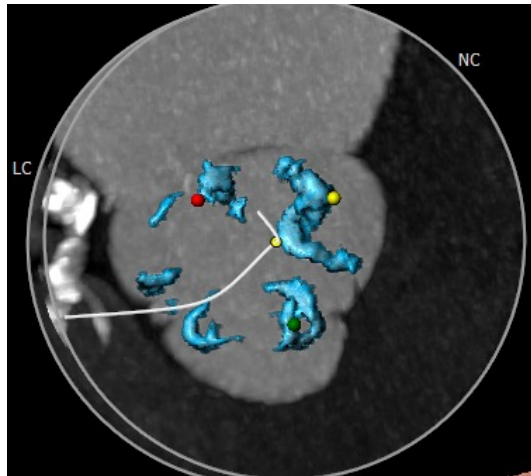
Outcomes following minimally invasive mitral valve surgery. Women experience worse long-term survival after minimally invasive mitral valve surgery as compared to men.

Fig. 4.



Aortic valve

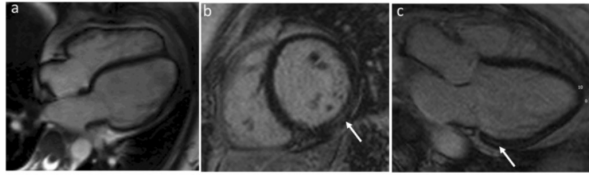
- AV calcification:
 - Important for diagnosis in patients low AV gradients on ECHO
- Measured similar to non contrast CT coronary calcium score
- Gender-specific aortic valve calcium cutoffs:
 - Male: 2065 AU
 - Female: 1274 AU



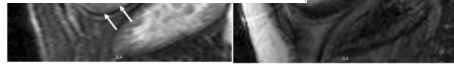
Calcification activity on ^{18}F -NaF PET and progression in aortic valve CT calcium score is increased in men compared to women

Other diseases detected by imaging

Fig. 3



Peripartum cardiomyopathy. Cine long-axis four chamber view, end-diastolic frame (a), late gadolinium enhancement short-axis (b), and three chamber view (c) in a woman with postpartum cardiomyopathy. The images show only a mildly dilated LV cavity (a) and mid-wall late gadolinium enhancement of the basal inferolateral wall (white arrows)



Takotsubo cardiomyopathy. Three-chamber view of a patient with Takotsubo cardiomyopathy. a shows the T2 weighted image with increased signal intensity of the mid-cavity and apical segments (white arrows) without late gadolinium enhancement (b)

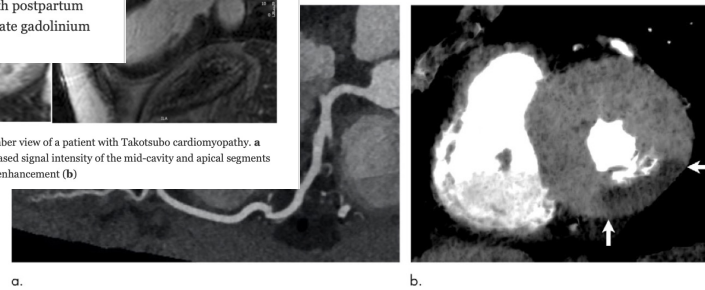
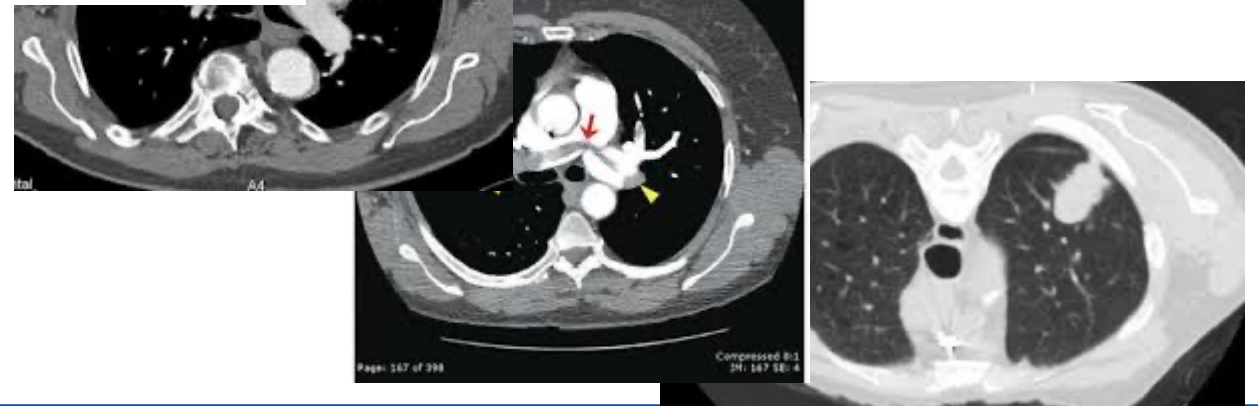


Figure 5: (a) Curved planar reformatted and (b) minimum intensity projection short-axis coronary CT angiography images in a 68-year-old woman, who presented with chest pain, show luminal occlusion of posterior left ventricular branch (arrow in a) with myocardial hypoperfusion in the corresponding territory (arrows in b). Involvement of single coronary artery territory and absence of an embolic source favored diagnosis of spontaneous coronary artery dissection over coronary artery embolism.



Key Takeaways

- **Sex differences in cardiovascular disease** extend beyond traditional risk factors. Recognizing biological and imaging differences leads to better outcomes
- **Women often present differently:** Non-obstructive CAD and microvascular dysfunction are common.
- **Imaging matters:** CCTA, PET, and MRI reveal disease missed by traditional tests.
- **AI adds precision:** Quantitative plaque analysis improves risk prediction, especially in women.
- **Valvular and other cardiac conditions** (e.g., aortic stenosis, mitral disease, SCAD) also show distinct sex-related patterns that imaging helps uncover, emphasizing the need for a *sex-specific approach to diagnosis and management*.

A normal stress test does not mean a normal heart, especially in women

Thank you

Questions?

Thank you

Talk 2: Ashley Brunmeier



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Unique Risk Factors... Pregnancy

Ashley Brunmeier, MD



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No disclosures.



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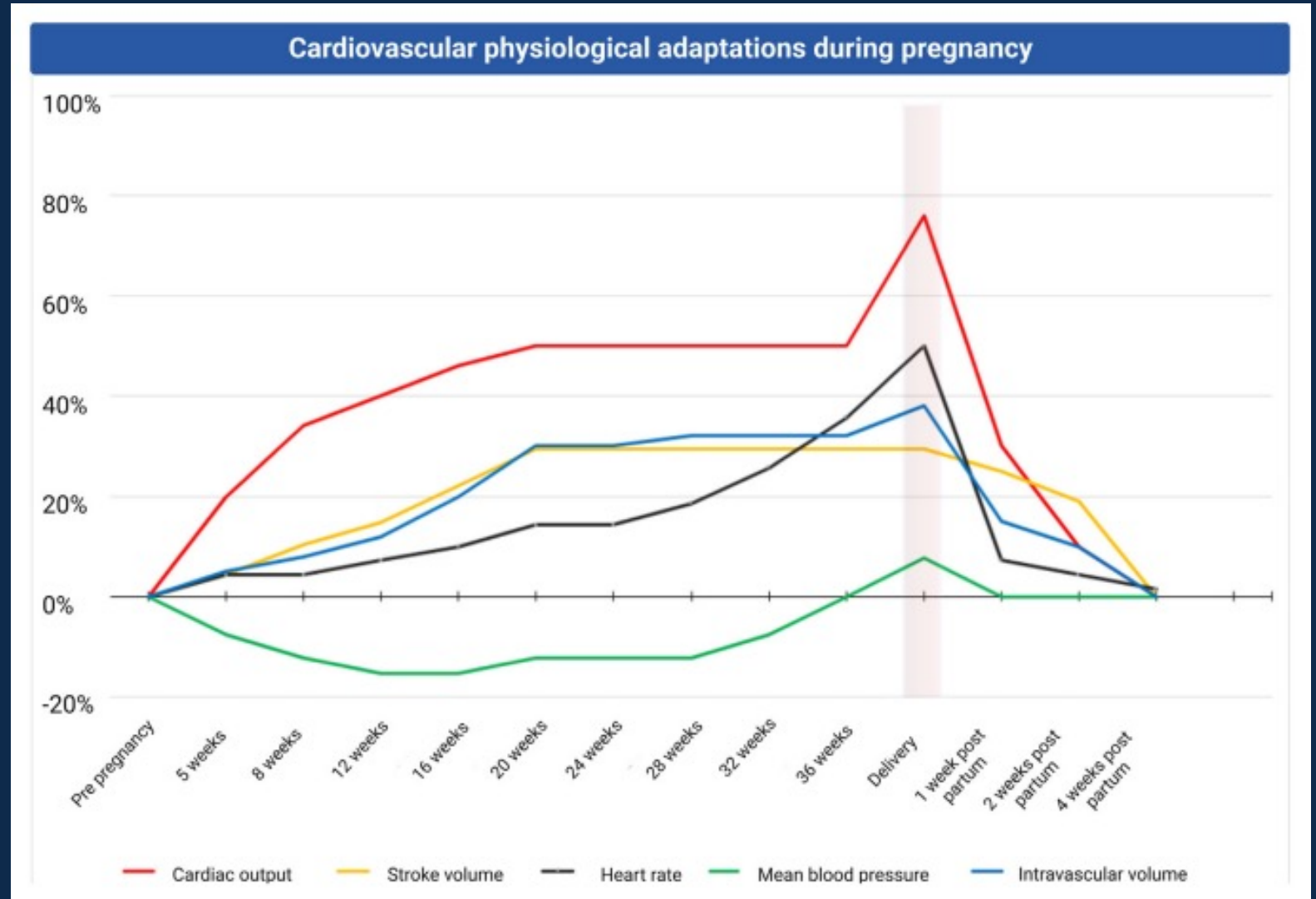
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Outline

- Discuss the physiological changes of pregnancy
- Risk score calculators
- Preconception, Antepartum, and Postpartum Care
- Interesting Cases



Cardiovascular Changes



Highest Risk Patients

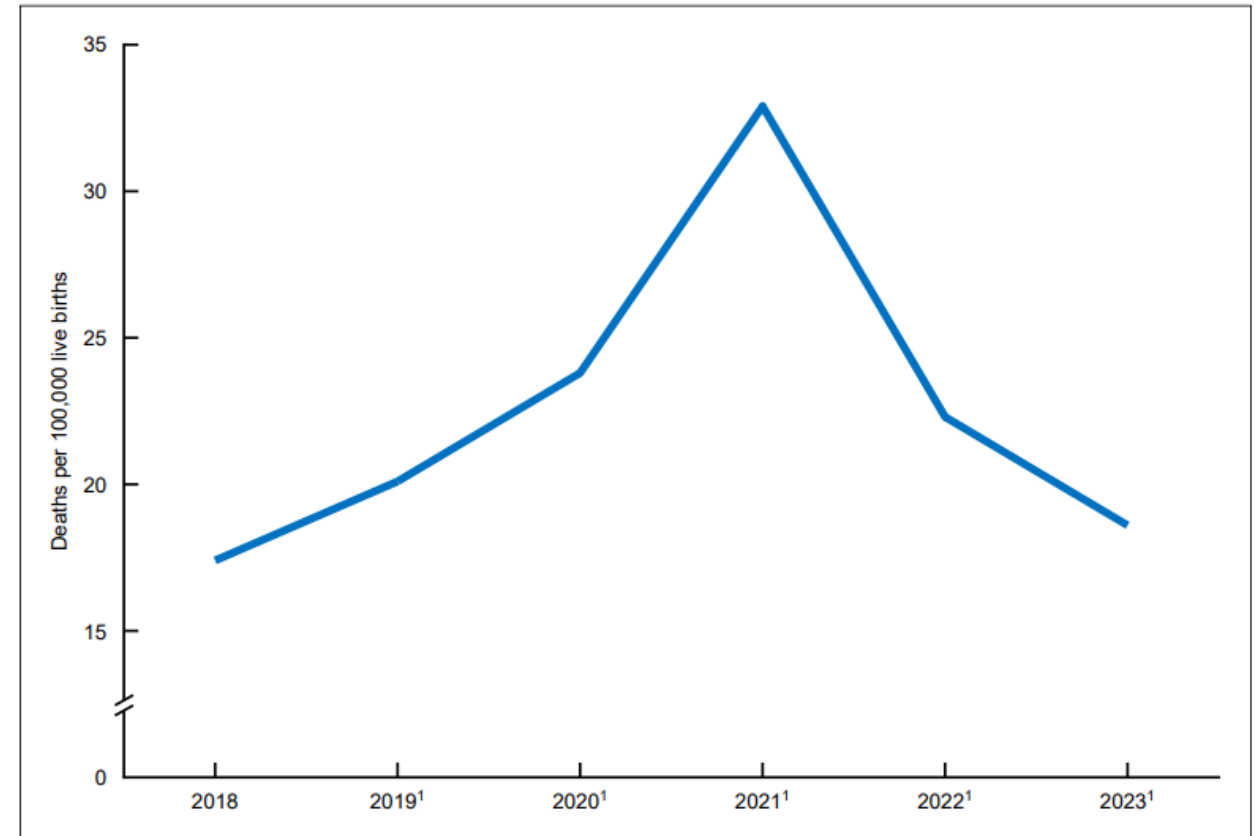
- Unable to accommodate the increase in cardiac output
 - Ventricular dysfunction
 - Significant coronary artery disease
 - Obstructive lesions
 - Pulmonary arterial hypertension
- Other increased risk
 - Mechanical Valves
 - Aortopathies



Overview

Overall, the maternal morbidity and mortality has been rising in the United States over the past several decades, 2023 there was a downtrend in this Cardiovascular disease is a leading cause of pregnancy-related deaths

Figure 1. Maternal mortality rate: United States, 2018–2023



¹Statistically significant change in rate from previous year ($p < 0.05$).

SOURCE: National Center for Health Statistics, National Vital Statistics System, mortality data file.



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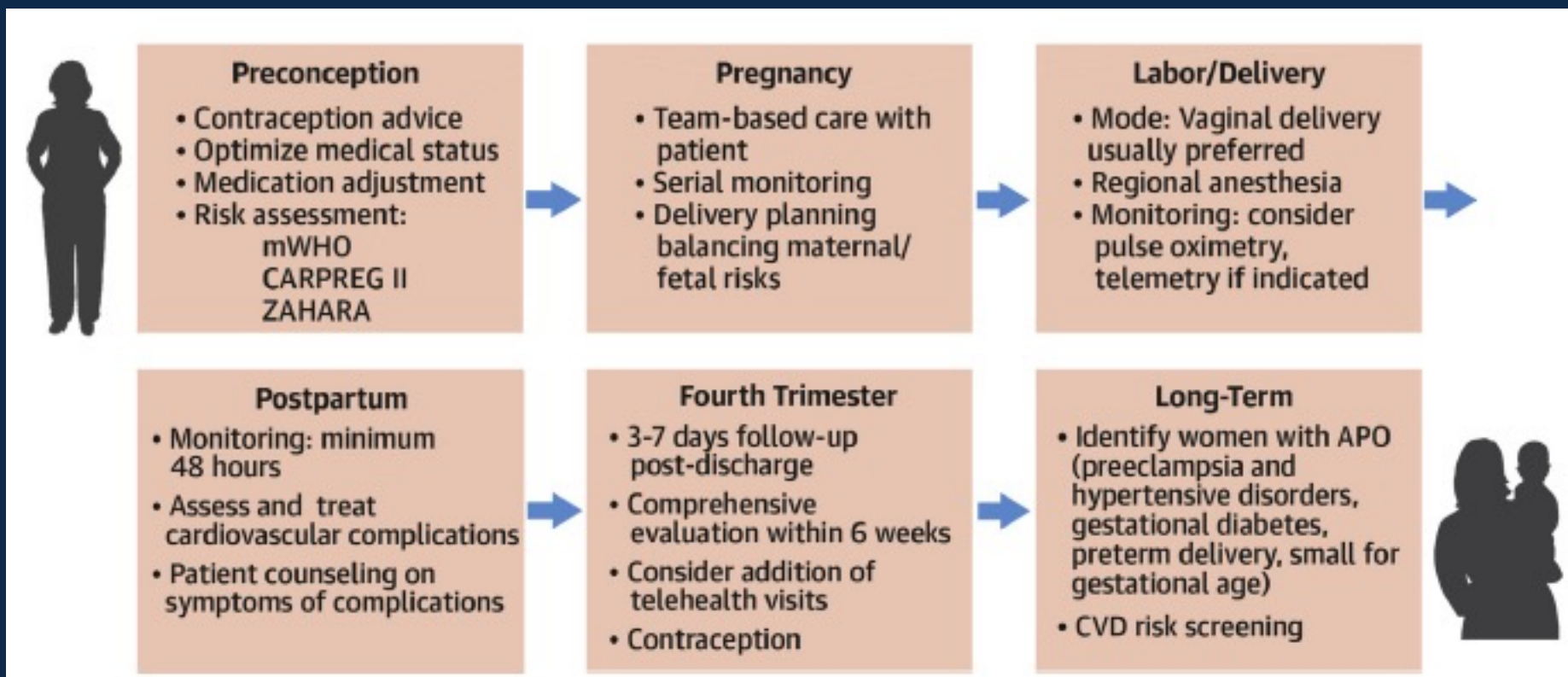
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Team Based Care



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Risk

- CARPREG
- ZAHARA
- mWHO
- CARPREG2

2018 ESC Guidelines for the management of cardiovascular diseases during pregnancy

The Task Force for the Management of Cardiovascular Diseases during Pregnancy of the European Society of Cardiology (ESC)

Pr Endorsed by: the International Society of Gender Medicine (IGM),
Pr the German Institute of Gender in Medicine (DGesGM), the
Di European Society of Anaesthesiology (ESA), and the European Society of Gynecology (ESG)

Di Authors/Task Force Members: Vera Regitz-Zagrosek* (Chairperson) (Germany),
Samuel C. Siu, MD, SM, MBA^{a,b,e} Jolien W. Roos-Hesselink* (Co-Chairperson) (The Netherlands), Johann Bauersachs^a (Germany), Carina Blomström-Lundqvist (Sweden), Renata Cífková (Czech

Pregnancy Outcomes in Women With Heart Disease

The CARPREG II Study

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Marla Kiess, MD,^c Valerie Rychel, MD,^d Rachel M. Wald, MD,^{a,b} Jack M. Colman, MD,^{a,b}
Samuel C. Siu, MD, SM, MBA^{a,b,e}

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ZAHARA Investigators



CARPREG II

TABLE 1 CARPREG II Risk Prediction Model

CARPREG II Predictors	Points
Prior cardiac event or arrhythmia	3
Baseline NYHA functional class III to IV or cyanosis	3
Mechanical valve	3
Ventricular dysfunction	2
High-risk left-sided valve disease/LVOT obstruction	2
Pulmonary hypertension	2
Coronary artery disease	2
High-risk aortopathy	2
No prior cardiac intervention	1
Late pregnancy assessment	1
CARPREG II Score	Predicted Risk, %
0 to 1	5
2	10
3	15
4	22
>4	41

CARPREG = Cardiac Disease in Pregnancy Study; LVOT = left ventricular outflow tract; NYHA = New York Heart Association.

- Prior cardiac event includes heart failure, stroke, TIA, or arrhythmia
- Late pregnancy assessment: first visit: > 20 weeks gestation



WHO

Table 3 Modified World Health Organization classification of maternal cardiovascular risk

	mWHO I	mWHO II	mWHO II–III	mWHO III	mWHO IV
Diagnosis (if otherwise well and uncomplicated)	Small or mild – pulmonary stenosis – patent ductus arteriosus – mitral valve prolapse Successfully repaired simple lesions (atrial or ventricular septal defect, patent ductus arteriosus, anomalous pulmonary venous drainage) Atrial or ventricular ectopic beats, isolated	Unoperated atrial or ventricular septal defect Repaired tetralogy of Fallot Most arrhythmias (supraventricular arrhythmias) Turner syndrome without aortic dilatation	Mild left ventricular impairment (EF >45%) Hypertrophic cardiomyopathy Native or tissue valve disease not considered WHO I or IV (mild mitral stenosis, moderate aortic stenosis) Marfan or other HTAD syndrome without aortic dilatation Aorta <45 mm in bicuspid aortic valve pathology Repaired coarctation Atrioventricular septal defect	Moderate left ventricular impairment (EF 30–45%) Previous peripartum cardiomyopathy without any residual left ventricular impairment Mechanical valve Systemic right ventricle with good or mildly decreased ventricular function Fontan circulation. If otherwise the patient is well and the cardiac condition uncomplicated Unrepaired cyanotic heart disease Other complex heart disease Moderate mitral stenosis Severe asymptomatic aortic stenosis Moderate aortic dilatation (40–45 mm in Marfan syndrome or other HTAD; 45–50 mm in bicuspid aortic valve, Turner syndrome ASI 20–25 mm/m ² , tetralogy of Fallot <50 mm) Ventricular tachycardia	Pulmonary arterial hypertension Severe systemic ventricular dysfunction (EF <30% or NYHA class III–IV) Previous peripartum cardiomyopathy with any residual left ventricular impairment Severe mitral stenosis Severe symptomatic aortic stenosis Systemic right ventricle with moderate or severely decreased ventricular function Severe aortic dilatation (>45 mm in Marfan syndrome or other HTAD, >50 mm in bicuspid aortic valve, Turner syndrome ASI >25 mm/m ² , tetralogy of Fallot >50 mm) Vascular Ehlers–Danlos Severe (re)coarctation Fontan with any complication
Risk	No detectable increased risk of maternal mortality and no/mild increased risk in morbidity	Small increased risk of maternal mortality or moderate increase in morbidity	Intermediate increased risk of maternal mortality or moderate to severe increase in morbidity	Significantly increased risk of maternal mortality or severe morbidity	Extremely high risk of maternal mortality or severe morbidity
Maternal cardiac event rate	2.5–5%	5.7–10.5%	10–19%	19–27%	40–100%
Counselling	Yes	Yes	Yes	Yes: expert counselling required	Yes: pregnancy contraindicated: if pregnancy occurs, termination should be discussed
Care during pregnancy	Local hospital	Local hospital	Referral hospital	Expert centre for pregnancy and cardiac disease	Expert centre for pregnancy and cardiac disease
Minimal follow-up visits during pregnancy	Once or twice	Once per trimester	Bimonthly	Monthly or bimonthly	Monthly
Location of delivery	Local hospital	Local hospital	Referral hospital	Expert centre for pregnancy and cardiac disease	Expert centre for pregnancy and cardiac disease

ASI = aortic size index; EF = ejection fraction; HTAD = heritable thoracic aortic disease; mWHO = modified World Health Organization classification; NYHA = New York Heart Association; WHO = World Health Organization.

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Plans

Primary Cardiologist:

Primary MFM:

Due Date:

CARPREG Score:

Modified WHO Group:

Cardiac Diagnosis:

Anesthesia Consult Recommended:

Telemetry:

Assisted Second Stage:

Echo 48 hours post-partum:

Special recommendations:

Pre-Admit:

Cardiac Medications:

Anticoagulation:

Cardiology Consult on Admission:

Location of delivery:



Postpartum

Follow up Postpartum

- Contraception
- Long term monitoring and management
- Medications



Case

40 year old female with history of CAD s/p PCI in 2011 and 2014 at OSH (no records available).

- Echo from 1 year prior LVEF 39% with wall motion abnormalities in the inferoseptal and inferior walls are akinetic.
- Last seen 10 months ago
- Having palpitations
- Currently 18 weeks pregnant



What to do next?

Medications?

- Last Appt: Lisinopril 20mg, Spironolactone 25mg, Jardiance, Carvedilol 12.5mg BID, Zetia, Rosuvastatin, ASA 81mg
 - Stopped all of these once finding out pregnant. Currently on nothing.

LV Function

- ECHO: LVEF 40-45% and WMA in the inferior wall.

Exam

- Euvolemic on exam. Blood pressure normal.



What to do next?

Plan

- Start Metoprolol succinate 25mg and restart ASA 81mg

Labs

- NT-proBNP today: <50

Follow up

- Return 2 weeks reassess volume and BP



What to do next?

Doing great on follow up. Improved palpitations with Metoprolol
Heart monitor for palpitations

- PACs, PVCs, and NSVT 8 beats

Plan

- Increase Metoprolol Succinate to 50mg daily and start Hydralazine 20mg TID and Isosorbide Dinitrate 20mg TID, Lasix 20mg PRN

Plan

- Follow up 4 weeks. Trimester BNPs or sooner with symptoms. Echo in third trimester or with symptoms



What to do next?

Follow ups continued to do well with normal BNP and tolerating medications.

Third trimester echo LVEF 48% with inferior wall hypokinesis.

Repeat monitor similar on increased dose of Metoprolol Succinate 50mg.

Delivered at 38w2d vaginal delivery

Medications: Hydralazine and Isosorbide stopped and transitioned to Enalapril.

Follow up Echo: LVEF 45% with similar RWMA.



Case

19 yo with history of bicuspid aortic valve s/p balloon aortic valvuloplasty at 12 years of age then lost to follow up.

- Comes to clinic 25 weeks pregnant.
- Increased shortness of breath, lower extremity swelling,

ECHO: Normal biventricular function; PV 4.8 m/s; PG/MG 93/53 mmHg – Severe Aortic Stenosis



Plan

Labs: Normal BNP and troponin in clinic.

Monitor performed

Medication: Lasix 20mg daily

Follow up – starting to notice dizziness and chest pain. Lasix decreased to every other day.

Due to high risk for delivery

- Seen monthly with Cardiology and MFM, Anesthesia consult prior to admission, had slow epidural titration, delivered via C-Section in Main OR with arterial line, CCU admission afterwards.

Discharged after 48 hours with Lasix as needed and follow up in 6 weeks.



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<https://doi.org/10.1016/j.jacc.2021.02.033>

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Questions?



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The Future of Critical Care Cardiology

Jeff Spindel, DO



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Disclosures

None



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Outline

Patient Population

- Diagnoses
- Comorbidities

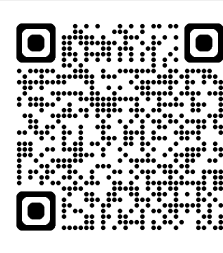
People

- Training
- Care Models

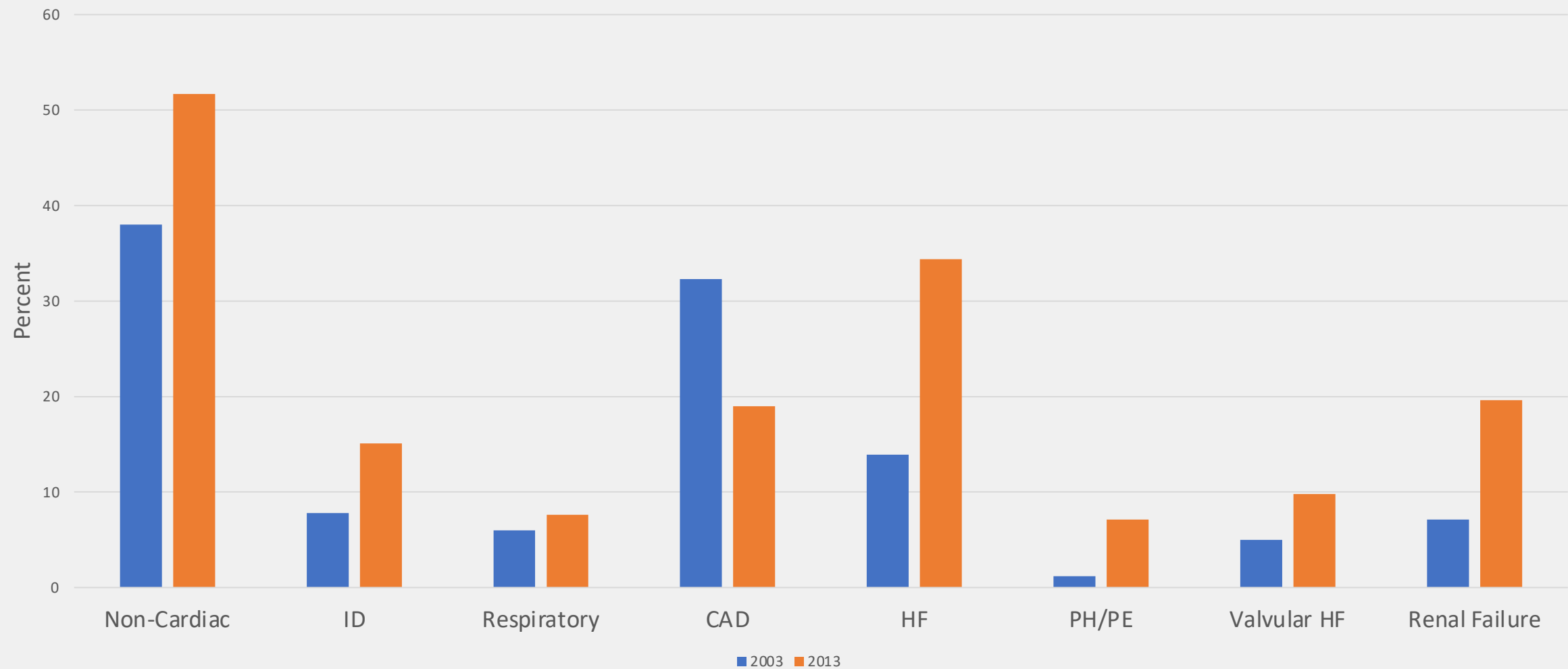
Systems

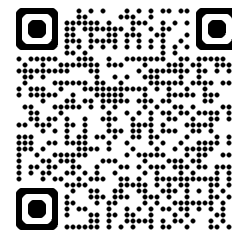
- Shock Teams
- Centers



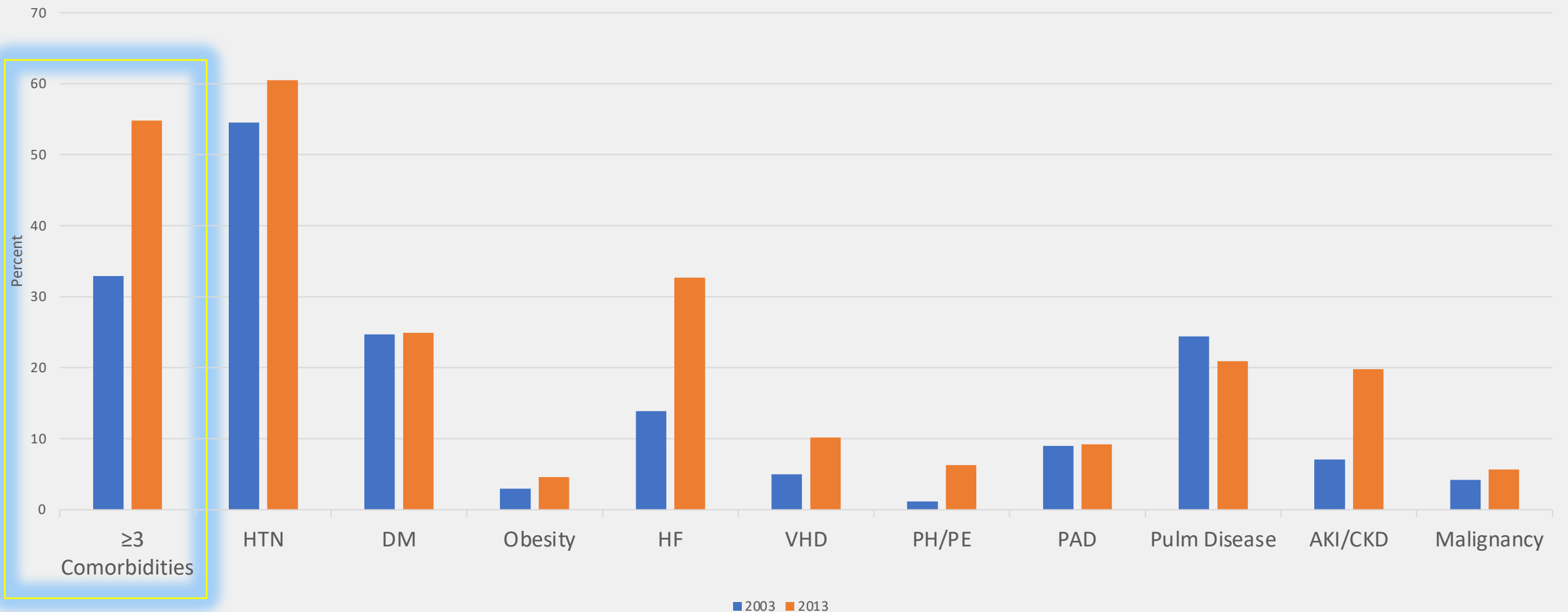


CICU Discharge Diagnosis





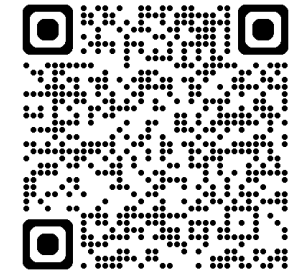
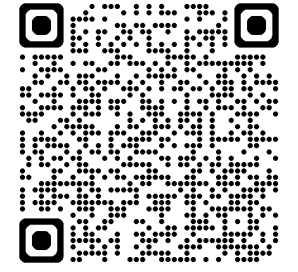
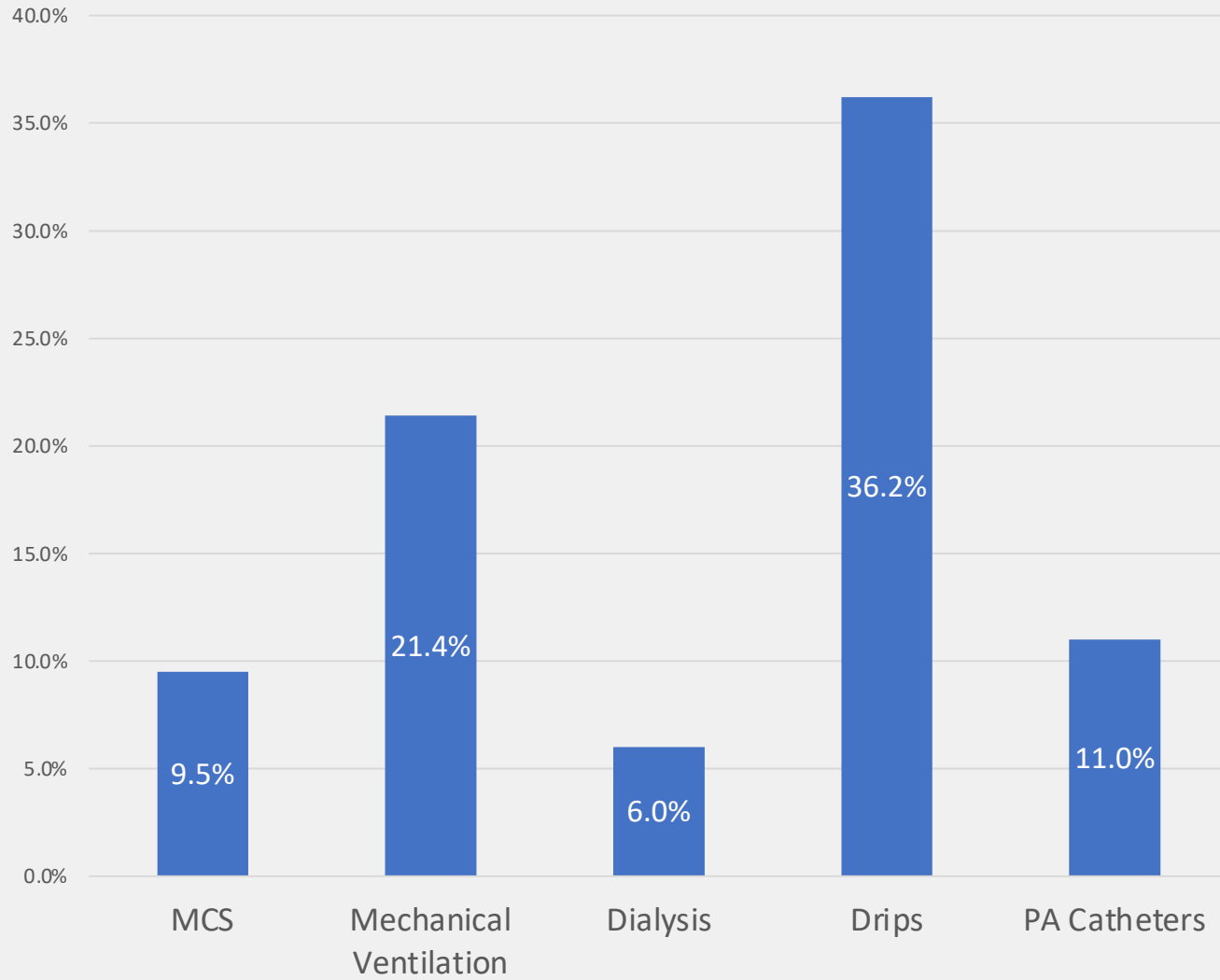
Comorbidities



■ 2003 ■ 2013



CICU Therapies and Treatment



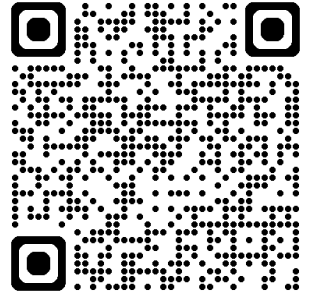
Changes in patient populations...



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...Requires changes in expertise



Training in Critical Care Cardiology Within Critical Care Medicine Fellowship



A Novel Pathway

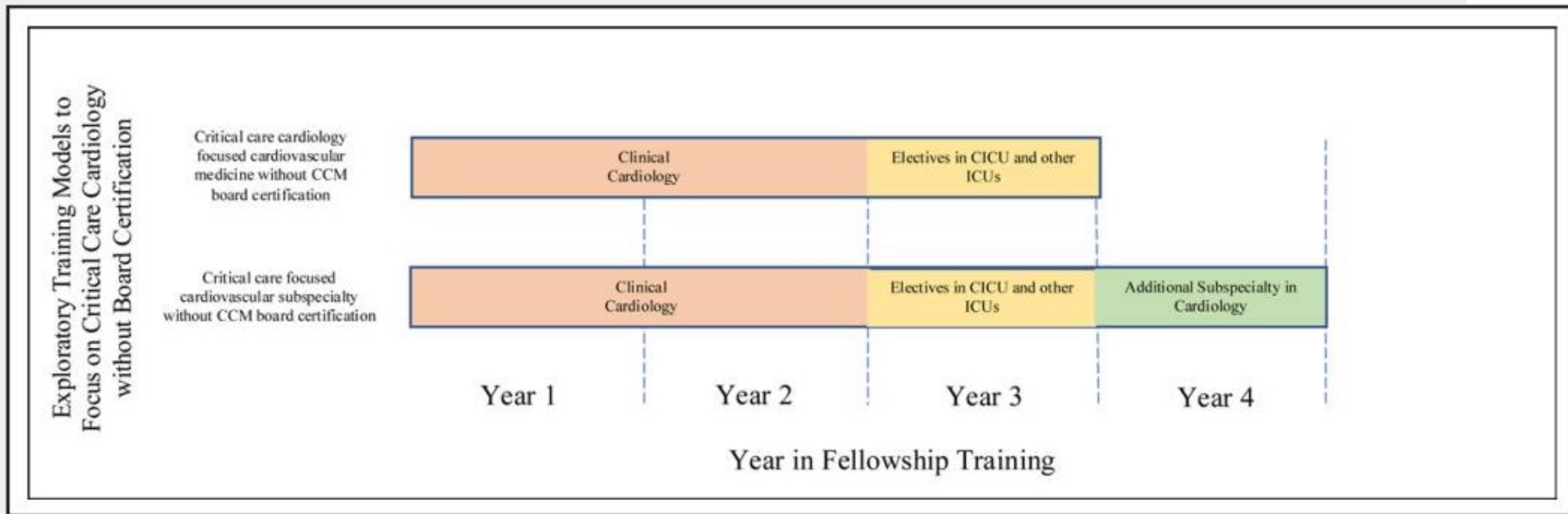
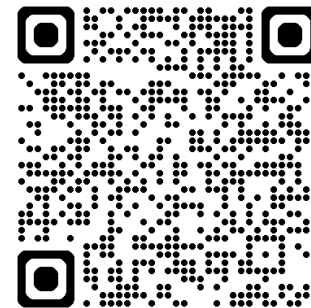
Connor G. O'Brien, MD,^a Christopher F. Barnett, MD, MPH,^a David M. Dudzinski, MD, JD,^b Pablo A. Sanchez, MD,^c
Jason N. Katz, MD, MHS,^d John G. Harold, MD,^e Erin K. Hennessey, MD,^f Paul K. Mohabir, MD^g

1. Airway/ventilator management
2. Management of mixed shock
3. Management of MCS
4. Familiarity with renal replacement therapy
5. Management of neurologic emergencies
6. Postoperative cardiac surgery management



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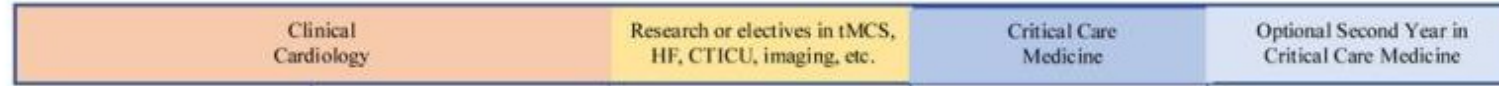
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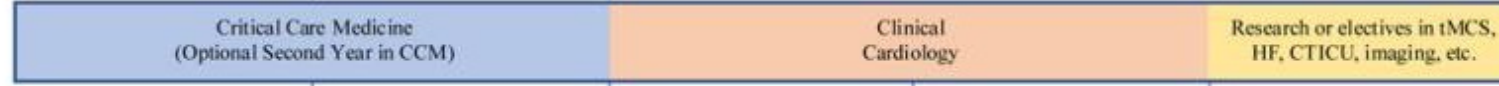
Training Models to Achieve Board Certification in Cardiovascular and Critical Care Medicine

Current Pathways

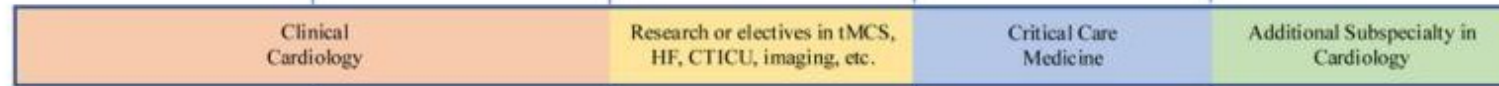
Cardiovascular medicine followed by CCM fellowship/board certification



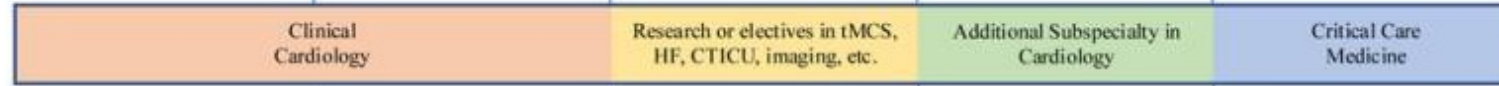
CCM fellowship/board certification followed by cardiovascular medicine



Multiple subspecialties with CCM fellowship/board certification

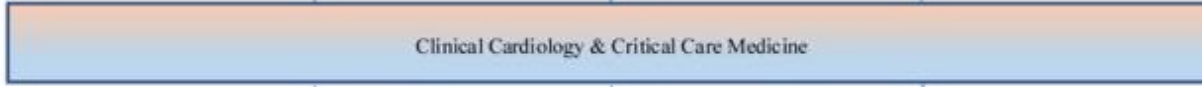


Multiple subspecialties with CCM fellowship/board certification



Possible Future Pathways

Integrated cardiovascular & CCM fellowship with board certification



Integrated cardiovascular & CCM fellowship with board certification and an additional subspecialty



Year 1

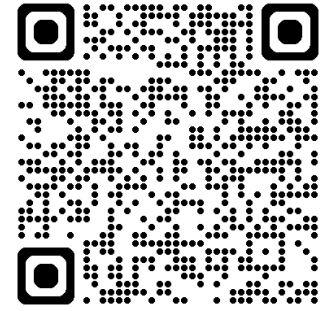
Year 2

Year 3

Year 4

Year 5

Year in Fellowship Training



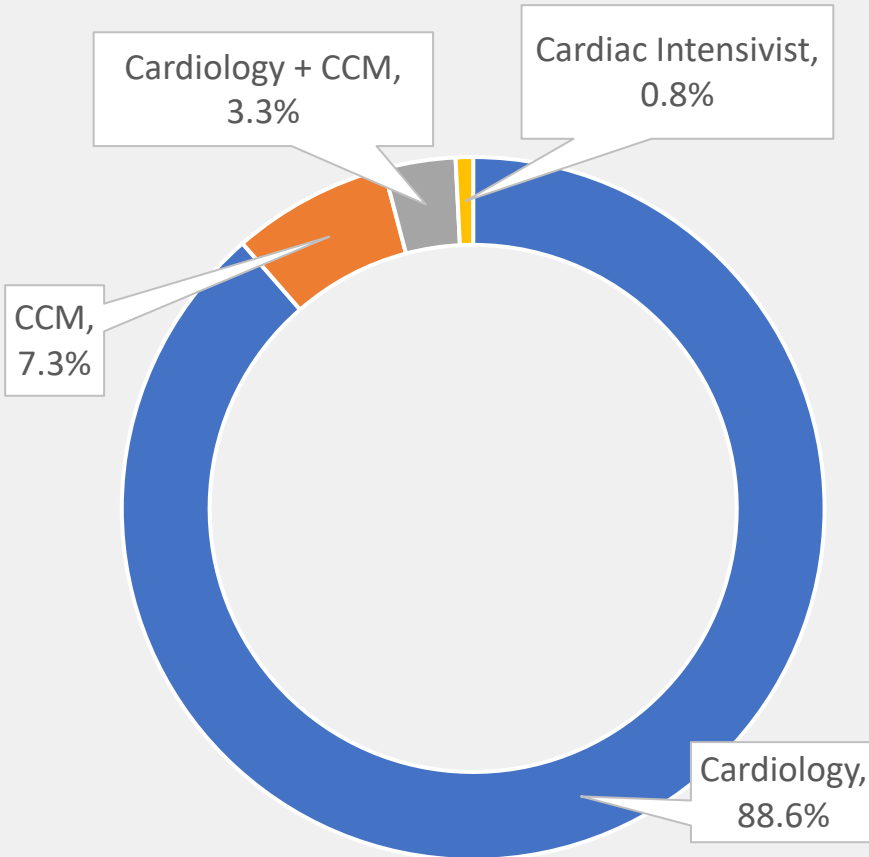
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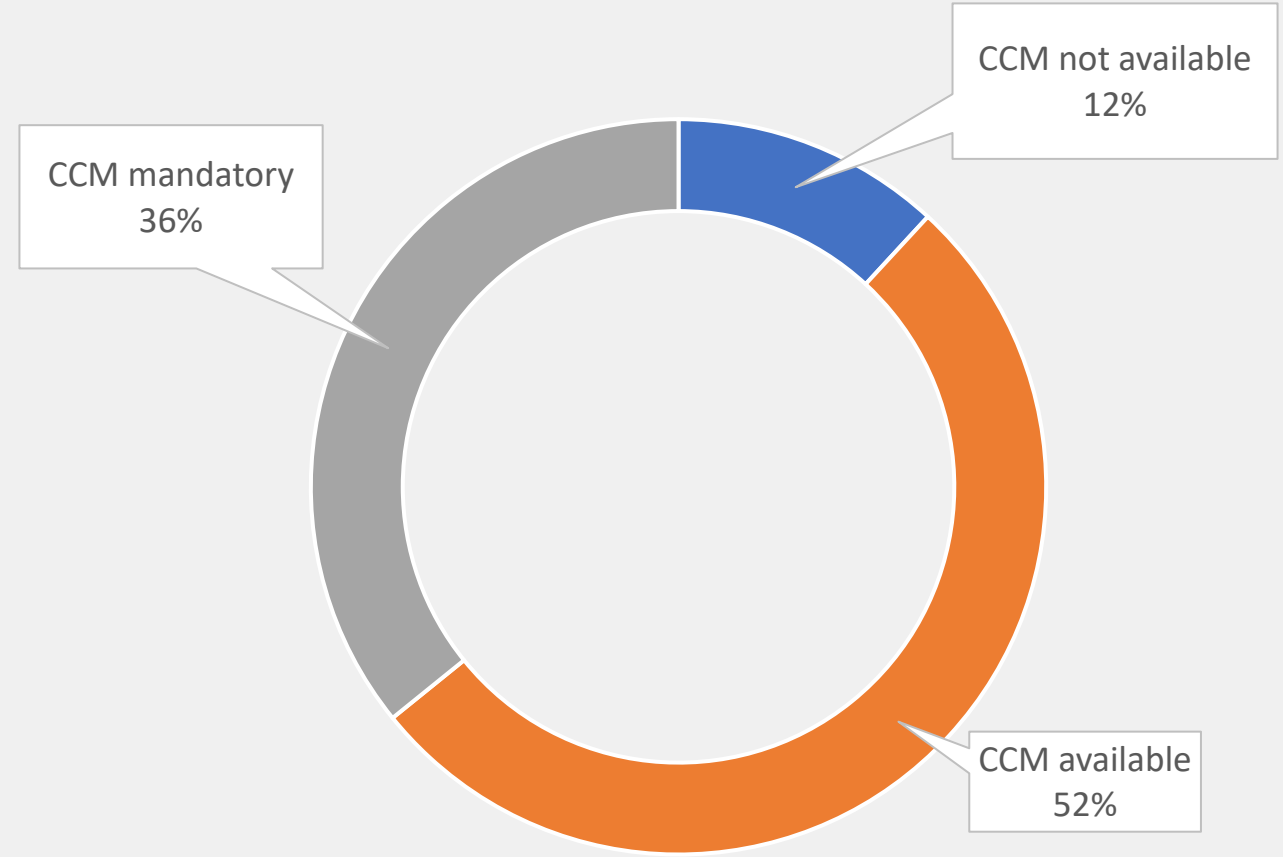
Staffing Models



Primary Physician

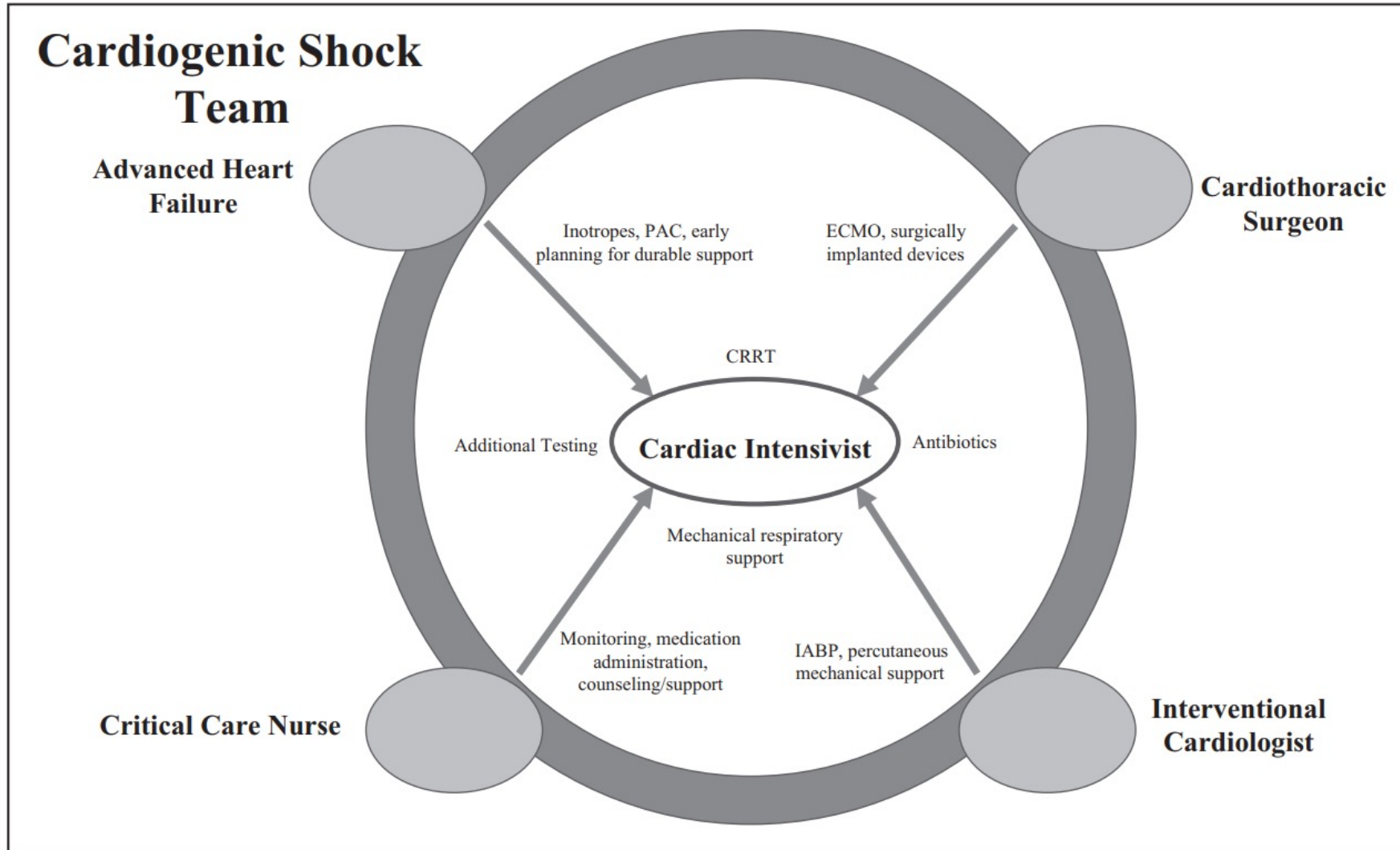
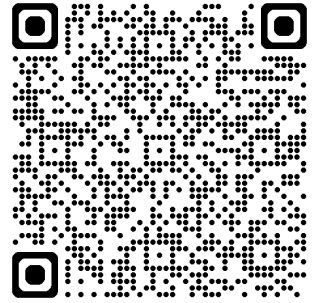


Consultations

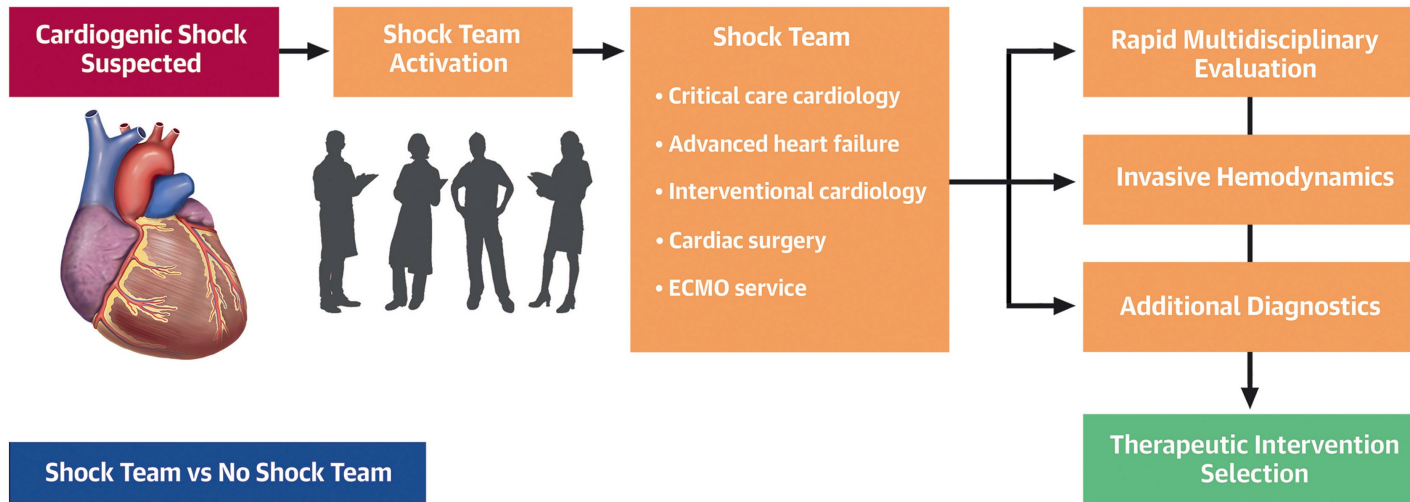


Intervention	Mortality	Cost	LOS	Reference
Closed Unit	✓			2
HF/CC Staffing	✓			3,4
24/7 Staffing	✓			5
Mandatory CC Consultation (IMV)		✓	✓	6
Shock Team	✓	✓	✓	7,8,9,10

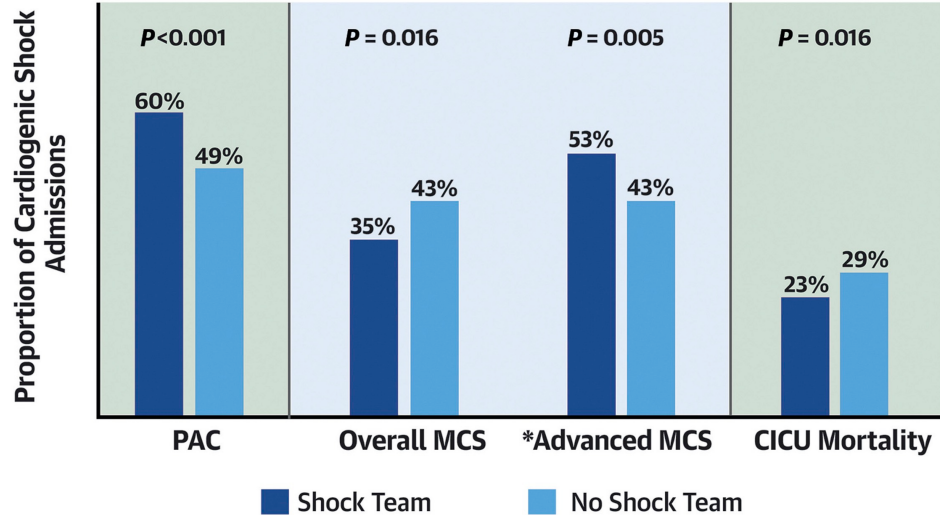
Shock Teams



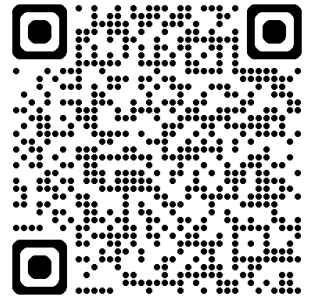
CENTRAL ILLUSTRATION: Prototypical Shock Team Workflow and Associated Outcomes



Shock Team vs No Shock Team Center Population Characteristics	
Cardiogenic shock admissions (n)	546 vs 696
AMI-CS (%)	27 vs 28
Admission lactate (mmol/L)	2.3 vs 2.3
PCWP (mm Hg)	25 vs 22
CI (L/min/m ²)	1.9 vs 2.0
CPO (W)	0.62 vs 0.64



Papolos, A.I. et al. J Am Coll Cardiol. 2021;78(13):1309-1317.



Lower mortality

- (23 vs 29%), aOR 0.72

Cost saving¹¹



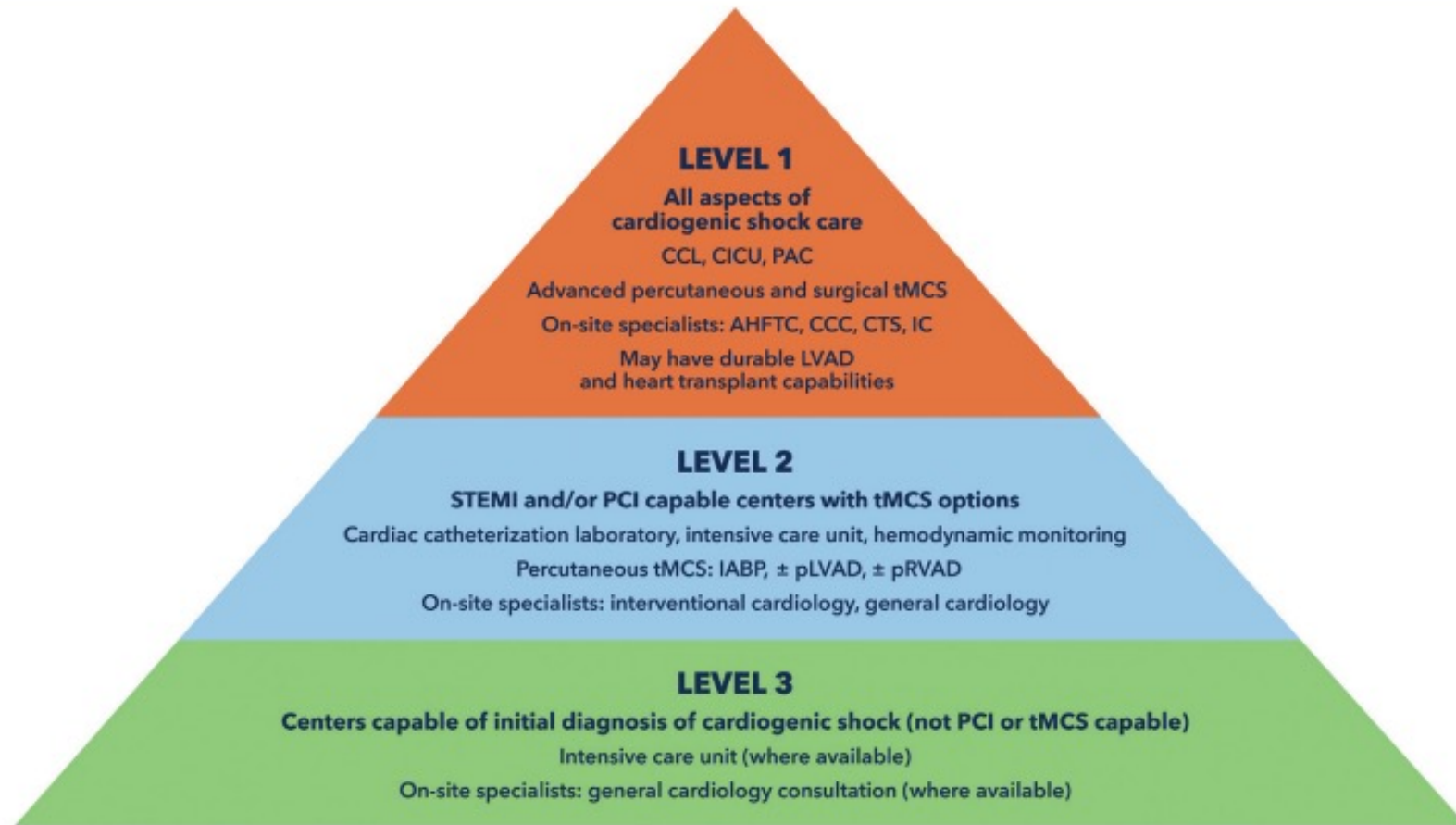
So, what is the future of CCC?



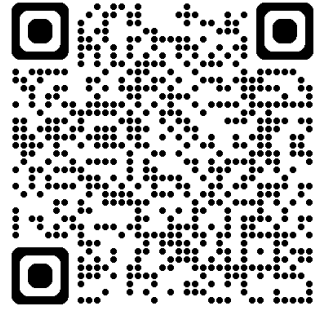
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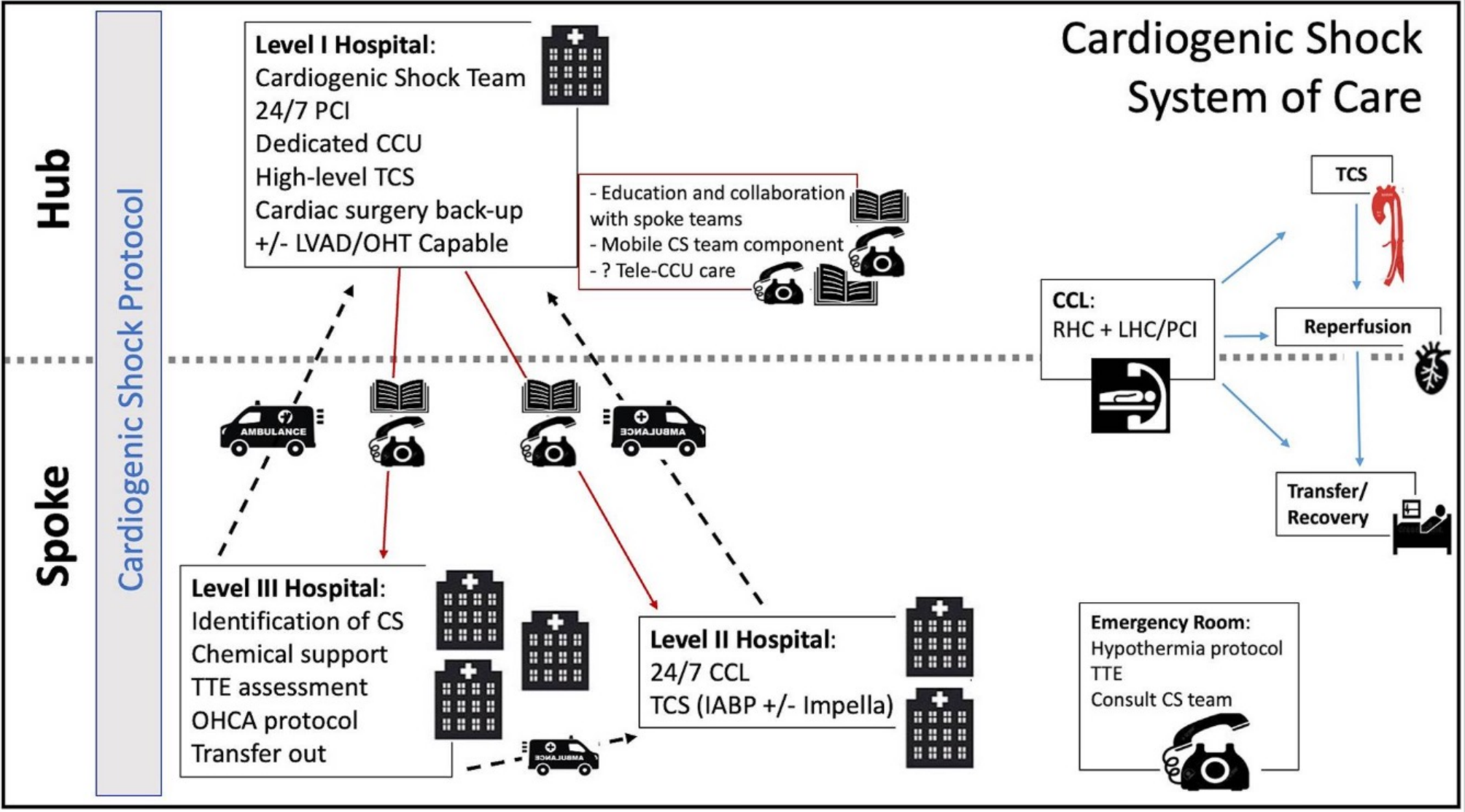
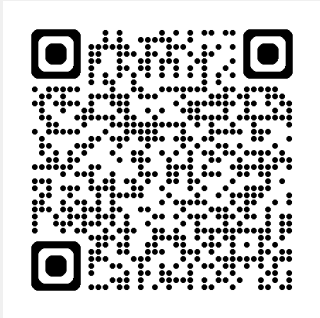
FIGURE 3 Proposed Classification of CS Centers of Care Based on Locally Available Resources and Expertise



AHFTC = advanced heart failure and transplant cardiology; CCC = critical care cardiology; CCL = cardiac catheterization laboratory; CICU = cardiac intensive care unit; CTS = cardiothoracic surgery; IABP = intra-aortic balloon pump; IC = interventional cardiology; LVAD = left ventricular assist device; PAC = pulmonary artery catheter; PCI = percutaneous coronary intervention; pLVAD = percutaneous left ventricular assist device; pRVAD = percutaneous right ventricular assist device; STEMI = ST-segment elevation myocardial infarction; tMCS = temporary mechanical circulatory support.



Systems of Care



Intraaortic Balloon Support for Myocardial Infarction with Cardiogenic Shock

Holger Thiele, M.D., Uwe Zeymer, M.D., Franz-Josef Neumann, M.D., Miroslaw Fejzi, M.D., Hans-Georg Olbrich, M.D., Jörg Hausleiter, M.D., Gert Richardt, M.D., Marcus Hennersdorf, M.D., Georg Fuernau, M.D., Steffen Desch, M.D., Ingo Eitel, M.D., Rainer Hambrecht, M.D., Jörg Michael Böhm, M.D., Henning Ebelt, M.D., Steffen Schneider, Ph.D., Gerhard Schuler, M.D., and for the IABP-SHOCK II Trial Investigators*

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Extracorporeal Life Support in Infarct-Related Cardiogenic Shock

H. Thiele, U. Zeymer, J. Alkin, M. Behnes, T. Basse, A. A. Mahabadi, P. Lehmann, P. Clemens, S. Ewer, T. Goslar

ORIGINAL CONTRIBUTION

JAMA-EXPRESS

Extracorporeal Membrane Oxygenation for Cardiogenic Shock

The NEW ENGLAND JOURNAL of MEDICINE

Effect of Extracorporeal Life Support With Atrial Fibrillation and Cardiogenic Shock

The TRIUMPH Randomized Controlled Trial

Percutaneous Mechanical Circulatory Support Versus Intra-Aortic Balloon Pump for Cardiogenic Shock After Acute Myocardial Infarction

tially.¹ However, cardiogenic shock (CS) may occur prior to or following reperfusion. Even those who survive acute intervention may later develop CS and the overall 30-day mortality for patients with CS in association with MI is approximately 40–50%. Unfortunately, this incidence has not changed in the past 20 years since the publication of the landmark SHOCK (SHould we emergently revascularize Occluded Coronaries for cardiogenic shock) trial.^{2–5}

ORIGINAL RESEARCH ARTICLE

and patients aged >75 years.⁹ The incidence of CS has increased in recent years, while the reason for increasing incidence is unclear, improved diagnosis and better access to care are both likely contributory.⁹ While the in-hospital mortality has improved,¹ the 6- to 12-month mortality in cardiogenic shock has remained unchanged at ≈50% over the past 2 decades.^{3, 4, 11}

Volodymyr M. Olovov, MD, Miroslava Seyfydova, MD, Alexander H. J. J. van der Wal, MD, PhD, and Jan Belohlavek, MD, PhD

Richard G. Jung, Ph.D., Jennifer A. Marbach, M.D., B.S., Jordan Hultson, M.D., Trevor Simard, M.D., F. Daniel Ramirez, M.D., David T. Harnett, M.D., Anas Merdad, M.B., B.S., Aws Almufleh, M.B., B.S., Willy Weng, M.D., Omar Abdel-Razek, M.D., Shannon M. Fernando, M.D., Kwadwo Kyeremanteng, M.D., M.H.A., Jordan Bernick, M.Sc., George A. Wells, Ph.D., Vincent Chan, M.D., Michael Froeschl, M.D., C.M., Marino Labinaz, M.D., Michel R. Le May, M.D., Juan J. Russo, M.D., and Benjamin Hibbert, M.D., Ph.D.



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Future of Critical Care Cardiology

Patients

- More comorbid
- More non-cardiac illnesses
- More IMV
- More RRT

People

- Expanded CCC interest and training opportunities
- Designated staffing

Systems

- Dedicated Units
- Multidisciplinary Shock Teams
- Interhospital communication and escalation



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Talk 3: Nachiket Apte



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NO DISCLOSURES



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POTS

Clinical Implications and management



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POTS

- Most common form of orthostatic intolerance in young people – 0.2-1.0 % of population below 40 years
- Increasing awareness – higher prevalence
- Debilitating symptoms during peak productive ages
- Predominantly noted in young women – premenopausal . 75% to 80% are female, and most patients are between the ages of 15 and 25 years at diagnosis.
- Commonly misdiagnosed – anxiety disorder, panic attacks , chronic fatigue syndrome

POTS - DEFINITION

- **orthostatic intolerance** characterized by a heart rate (HR) increment of 30 beats/min or more, often with standing HRs >120 beats/min, **within 10 min of standing** or head-up tilt (HUT), **and in the absence of orthostatic hypotension** (a decrease in systolic blood pressure (BP) of 20 or more mm Hg and/or decrease in diastolic BP of 10 or more mm Hg)
- Dysautonomia may have POTs/orthostatic intolerance as a feature but often autonomic testing may be normal
- Some patients may express symptoms at rest – however standing tends to almost always exacerbate symptoms.
- Symptoms are relieved with lying down or resting.

POTS - characteristics

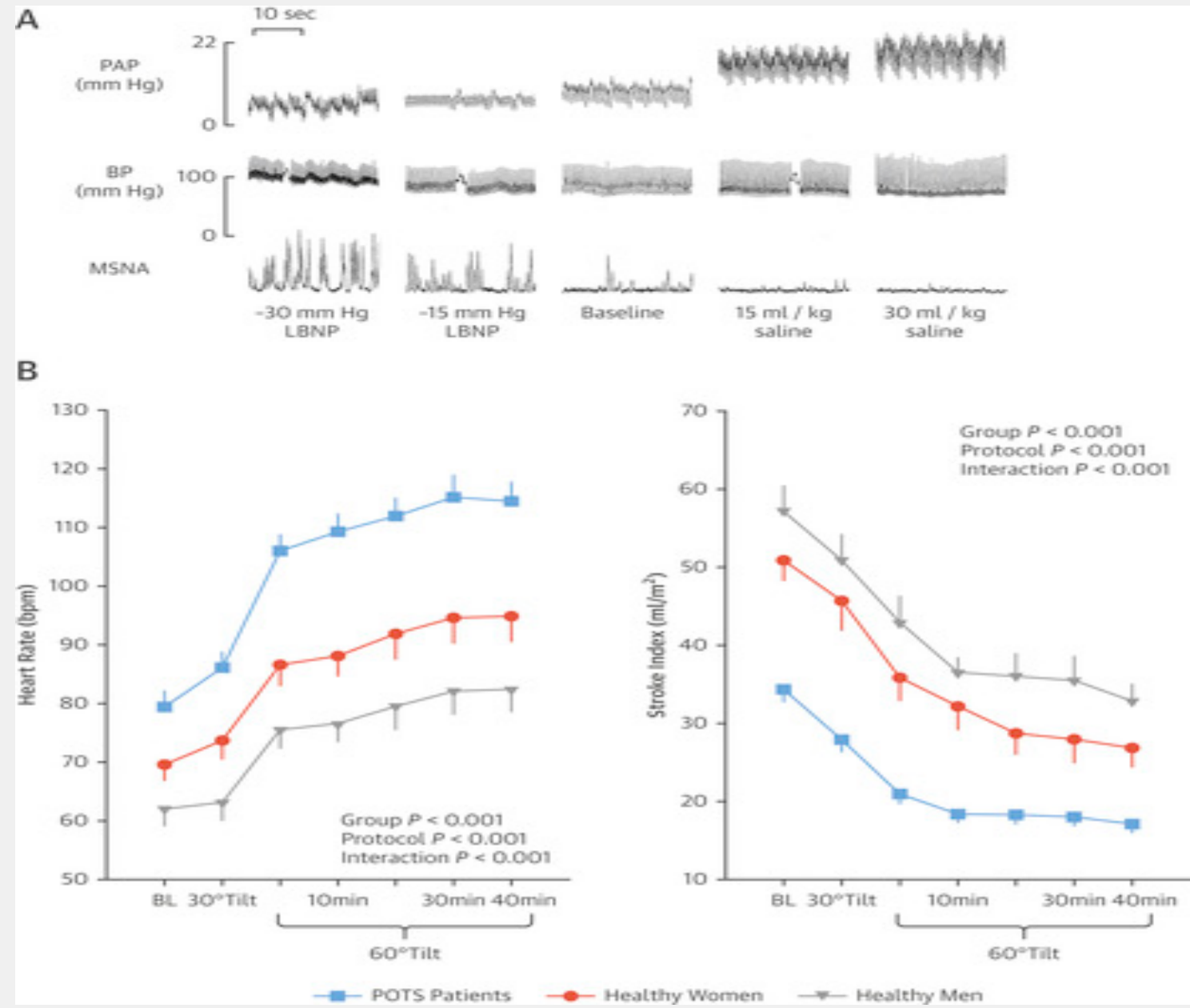
- lightheadedness and palpitations when upright, particularly when standing. **Pre-syncope is much more common than syncope**
- syndrome, not a disease – overlapping symptoms.
- bloating, nausea, diarrhea, abdominal pain, fatigue, sleep disturbance, headache,

Comorbid conditions	Prevalence
Migraine headaches	40%
Hypermobile Ehlers–Danlos syndrome and hypermobile spectrum disorder	25%
Myalgic encephalomyelitis/chronic fatigue syndrome	21%
Fibromyalgia	20%
Autoimmune disorders	16%



Cardiovascular Response to Orthostatic Stress

- a small (5 to 20 beats/min) increase in HR and a corresponding small decrease in systolic and diastolic BP is noted with standing from a lying down position
- women with POTS have both an exaggerated fall in stroke volume and an apparent compensatory rise in HR.



POTS - pathophysiology

- **1) Decreased venous return**
 - Low volume
 - Relative orthostatic hypovolemia
 - Cardiac deconditioning
 - Small fiber neuropathy
 - Impaired relaxation
- **2) Excessive sinus node activation**
 - Inflammatory mediators
 - Excessive sympathetic activation
 - ? Auto antibodies

CMAJ. 2022 Mar 14;194(10):E378-E385.

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Clinical sub types

Hyper-adrenergic POTS

- excessive increase in plasma norepinephrine levels(≥ 600 pg/mL) and a rise in systolic blood pressure on standing.
- palpitations, anxiety, tachycardia, and tremor
- Hypersensitivity to isoproterenol

Neuropathic POTS – 50 % MC

- Autonomic dysfunction of small and distal postganglionic sudomotor fibers,
- obstruction of compensatory venoconstriction during upright posture
- Venous pooling -

Hypovolemic POTS

- low levels of plasma renin activity and aldosterone despite hypovolemia. angiotensin II levels are 2 to 3 times higher than normal



POTS – other mechanisms

- **Mast Cell Activation syndrome**

- episodes of flushing, shortness of breath, headache, lightheadedness, excessive diuresis, and GI symptoms (including abdominal pain, diarrhea, nausea and vomiting)
- abnormally increased levels of histamine metabolites
- May respond to anti histaminics rather than beta blockers

- **Effect of Sex and Menstrual Cycle**

- worsening during either the pre-menstrual or early follicular phase when both estrogen and progesterone levels are dropping or low
- smaller stroke volume and smaller, less distensible hearts

- **Autoimmunity**

- antibodies against ganglionic acetylcholine receptors (AChR) and antibodies against alpha-1 adrenergic receptors
- Mostly neuropathic
- Stress or viral illness

JACC Focus Seminar. JACC. 2019 Mar, 73 (10) 1207–1228..

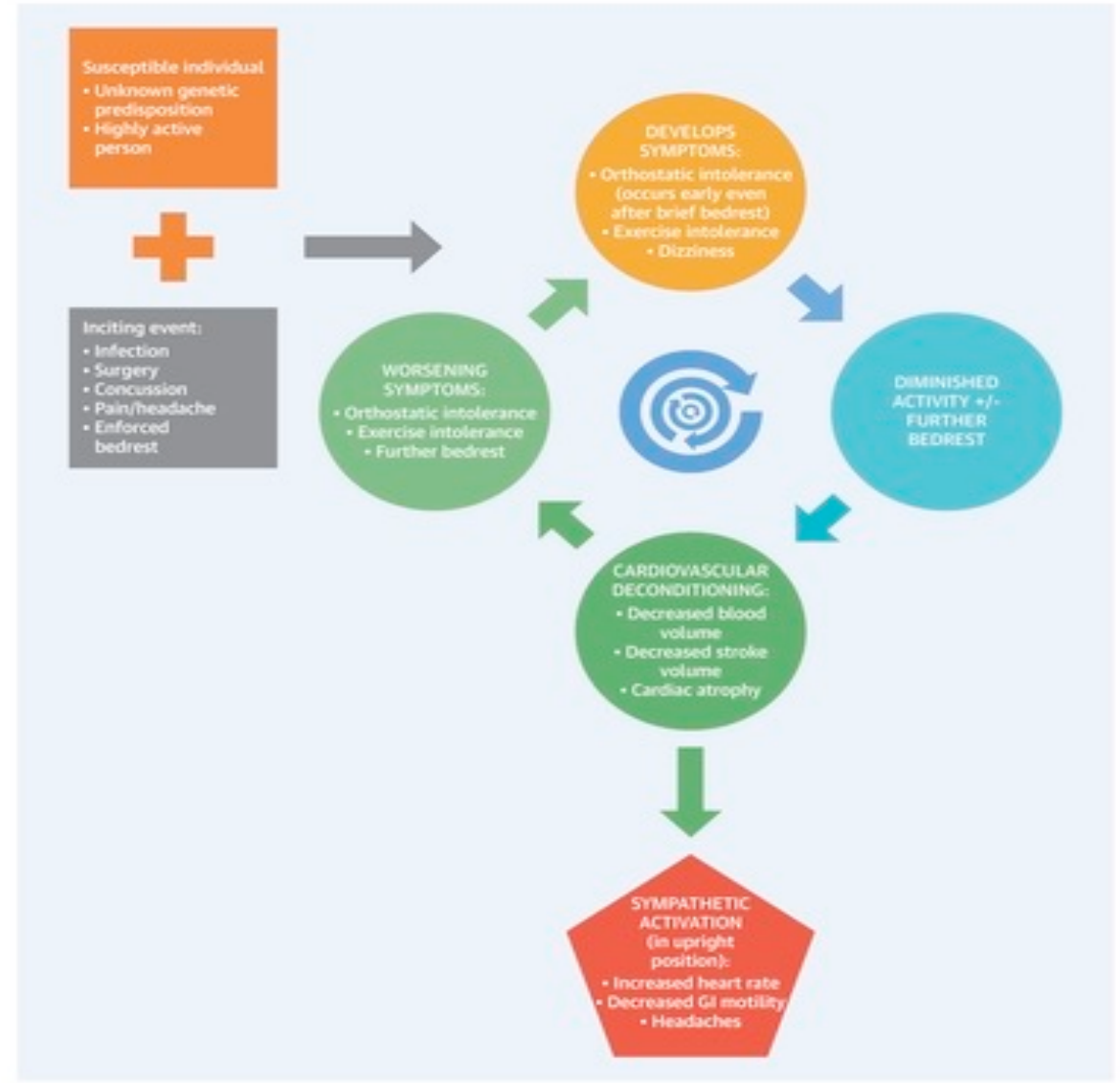
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POTS

- Diurnal variation – early morning tachycardia
- Physical enforced deconditioning leading to cardiovascular deconditioning.
- positive response to exercise training

CENTRAL ILLUSTRATION: Postural Orthostatic Tachycardia Syndrome: Downward Spiral



Bryarly, M. et al. J Am Coll Cardiol. 2019;73(10):1207-28.

POTS in the post COVID era

- Higher number of individuals have reported POTS like symptoms post COVID-19 exposure or illness
- “Long COVID syndrome ” – prolonged fatigue , exertional intolerance accompanied with orthostatic tachycardia
- No unifying mechanism to explain symptoms could include a combination of Autoimmunity, small fiber neuropathy, cardiac deconditioning as well as physical deconditioning
- Most commonly – neuropathic vs hypovolemic sub types
- Other findings – intolerance to temperature variations, GI disturbances , “brain fog ” etc.
- Management is currently on similar lines
- Exercise regimen along with lifestyle changes seem to significantly improve symptoms.



Differentials

- Physiological orthostatic tachycardia
- Anxiety
- Pheochromocytoma endocrine disorders
- Medication use
- Neurally mediated syncope (NMS)
- Neural disorders - multiple system atrophy, Parkinson disease, Lewy body dementia, pure autonomic failure, autoimmune autonomic ganglionopathy,
- Inappropriate sinus tachycardia (IST)



Diagnosis

- Detailed history
- Review of medications
- Orthostatic vitals – can be done in the office – standing at 1 min and 3-5 min intervals.
- Orthostatic tachycardia-more pronounced in the morning – testing should be done
- Physical examination – other causes, hypemobility, skin changes, thyroid swelling
- Formal autonomic testing – beat to beat monitoring- Valsalva response, sweat chloride, peripheral nerve testing – can be helpful with subtypes – usually requires more comprehensive testing.
- Response to isuprel or nitrates is not considered diagnostic

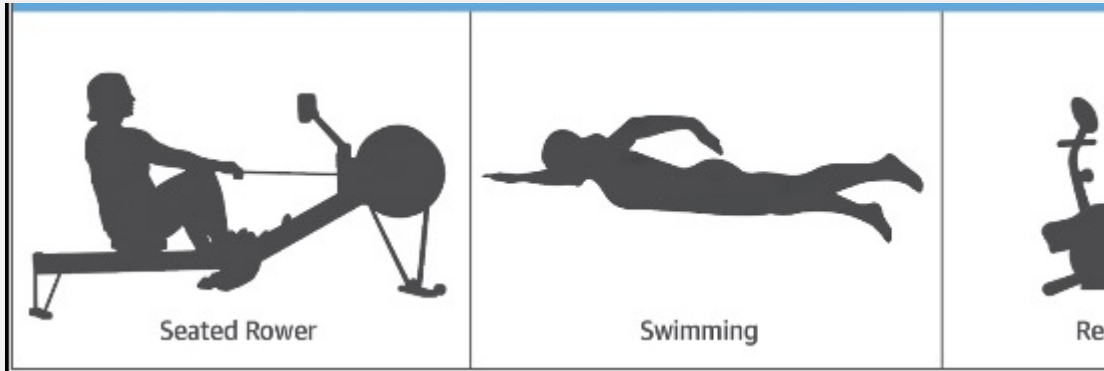


Management

- Non pharmacological measures as first step – at UofL – minimum of 3 months
 - Include – **graded exercise program(Class IIa)**, adequate electrolyte rich hydration, avoidance of stimulants if possible, avoidance of triggers, compression stockings if applicable
- Medications – depending on symptoms
- Palpitations – beta blockers, ivabradine
- Midodrine Class IIb
- Fludrocortisone- mineralocorticoid Class IIb



Management



Donkey Kicks



Hamstring curls



Click for the full workout

www.betterbythebeat.com

More to explore ▾



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Medications

- Medications – depending on symptoms
- Palpitations –
- Beta blockers Class II b –
- prefer non selective options – propranolol ,. Alternatives include metoprolol and atenolol for ease of use.
- Midodrine Class Iib – Alpha1 agonist- orthostatic symptoms with background of low BP
- Fludrocortisone- mineralocorticoid Class Iib -



Medications

- Intravenous saline - acute clinical decompensation attributable to hypovolemia - not recommended for routine management
- - Limited subset – severe gastroparesis with inability to swallow significant amounts of fluids.
- Pyridostigmine – Class II b - cholinergic agonist
 - Potential side effects include abdominal cramping, diarrhea – may benefit those with constipation

Others

- Clonidine, phenobarbital, SSRI s , anti histaminics , modafinil
- Ivabradine – I_f inhibitor



Summary

- POTS is a syndrome – one size may not fit all – however orthostatic prolonged tachycardia is a classic finding
- Patients may have overlapping symptoms with other neurally mediated disorders – management may be similar
- Extensive history is extremely helpful.
- Non-pharmacological management is the mainstay of therapy. Medications help in certain cases.
- Reassurance and persistence to graded exercises is helpful

THANK YOU

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