October 6th, 2022
3:00 PM ET

AIM TAP WEBINAR:
Cardiac Conditions in Obstetrical Care:
Considerations for Cardiomyopathy in Pregnancy

with Dr. Karen Florio, DO

ALLIANCE FOR INNOVATION
ON MATERNAL HEALTH

The Alliance for Innovation on Maternal Health is a national, cross-sector commitment designed to support best practices that make birth safer, improve maternal health outcomes, and save lives.

This program is supported by a cooperative agreement with the Health Resources and Services Administration (HRSA) of the U.S. Department of Health and Human Services (HHS) under grant number UC4MC28042, Alliance for Innovation on Maternal Health. This information or content and conclusions are those of the author and should not be construed as the official position or policy of, nor should any endorsements be inferred by HRSA, HHS or the U.S. Government.

Before we get started

AIM TAP Webinars are recorded

Resources discussed are not vetted or endorsed by AIM or ACOG and are provided for your convenience

Resources should be used at your discretion

If you need help during the call, please chat an AIM staff member

Cardiac Conditions in Obstetrical Care:

Considerations for Cardiomyopathy in Pregnancy

Dr. Karen Florio,
DO

PERIPARTUM CARDIOMYOPATHY

Karen Florio DO MPH
Associate Professor, University of Missouri
Vice Chair of Patient Safety and Quality
Chair, Missouri MMRC
Maternal Section Chair, Missouri PQC

I have no conflicts of interest and nothing to disclose

Outline

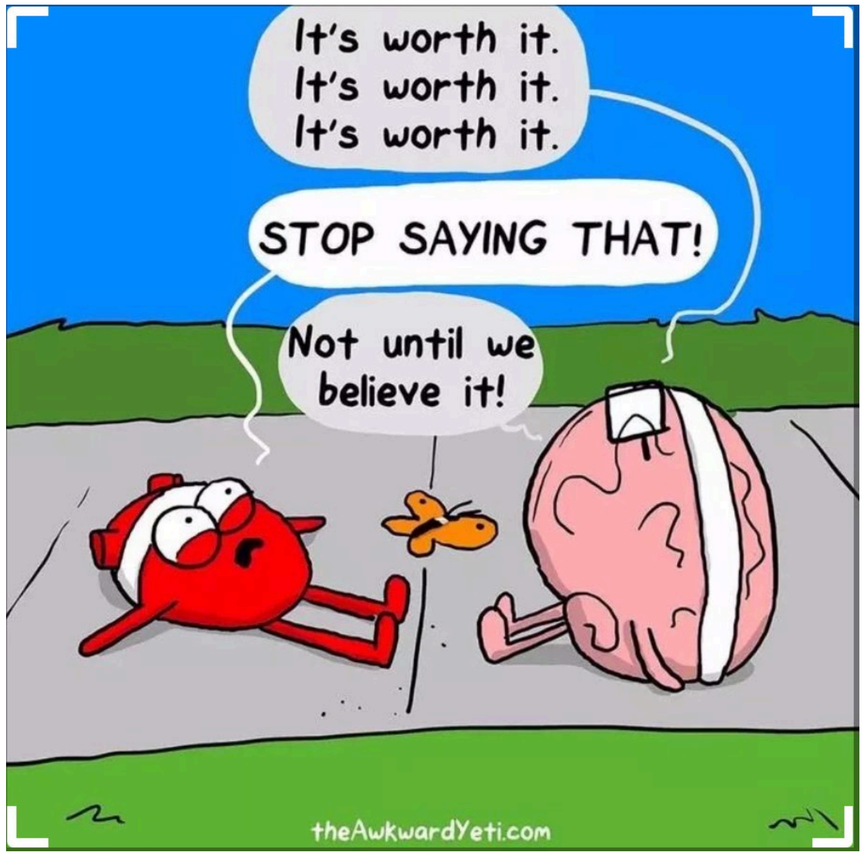
- Pregnancy physiology
  - TBF (throw back ….all the way back, to med school!)

- PPCM
  - Incidence
  - Risk factors and genetics
  - Molecular biology
  - Symptoms and presentation
  - Treatment and long-term outcomes
  - Why this all matters!

Cardiac Output

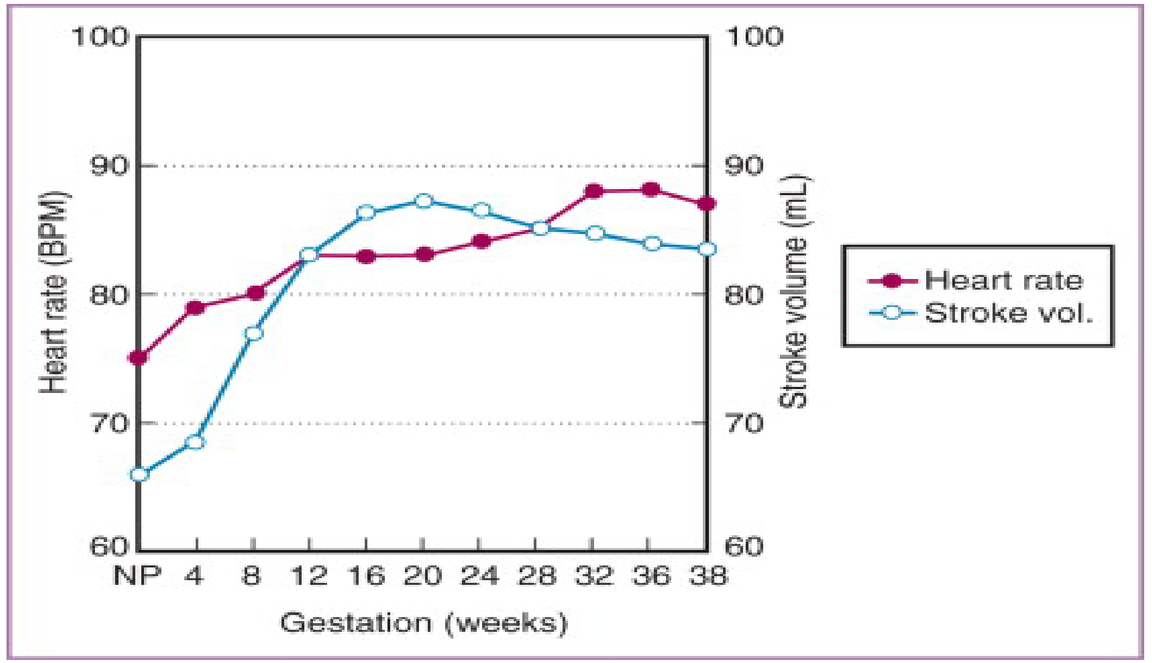
- Different from non-pregnant
  - ***5.5-7.5 L/min***

- What are the determinants of cardiac output?
  - Preload
  - Afterload
  - Contractility
  - Heart rate



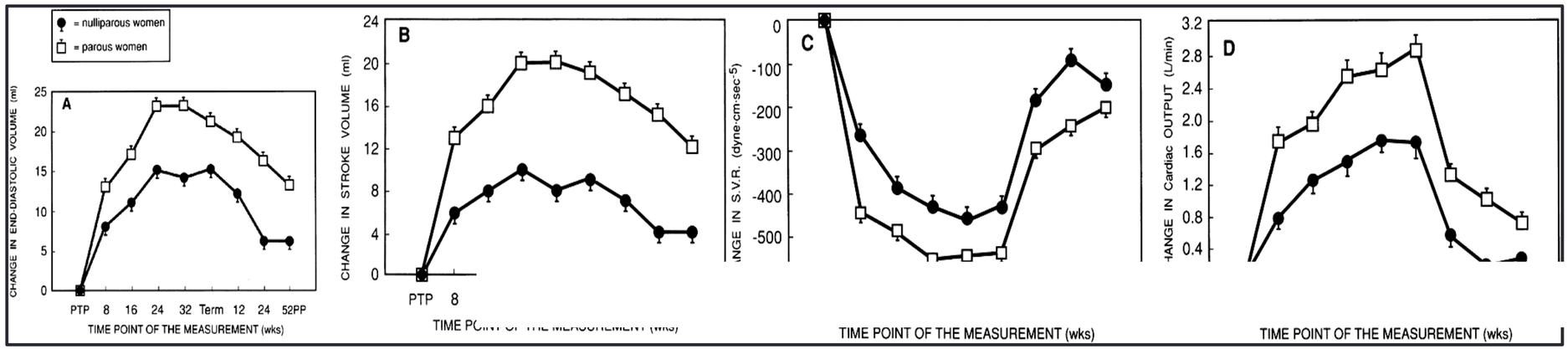
Cardiac Output

- **Cardiac output (SV x HR)**
  - Increased by 30-50%
    - Occurs by 8 weeks
    - Parous women have higher rise in SV (as well as lower BP)
  - Significantly affected by maternal posture
    - Supine = drop in CO by as much as 30%
  - *Labor = increase by 30-40%*
- **Uterine blood flow**
  - Increases by 10-fold (500-800 mL/min)
  - Shift from 2% to 17% at term of CO from heart



Creasy, Robert K., eds. *Creasy And Resnik's Maternal-fetal Medicine: Principles And Practice*. Philadelphia, PA : Saunders/Elsevier, 2009. Print

Pregnancy hemodynamics

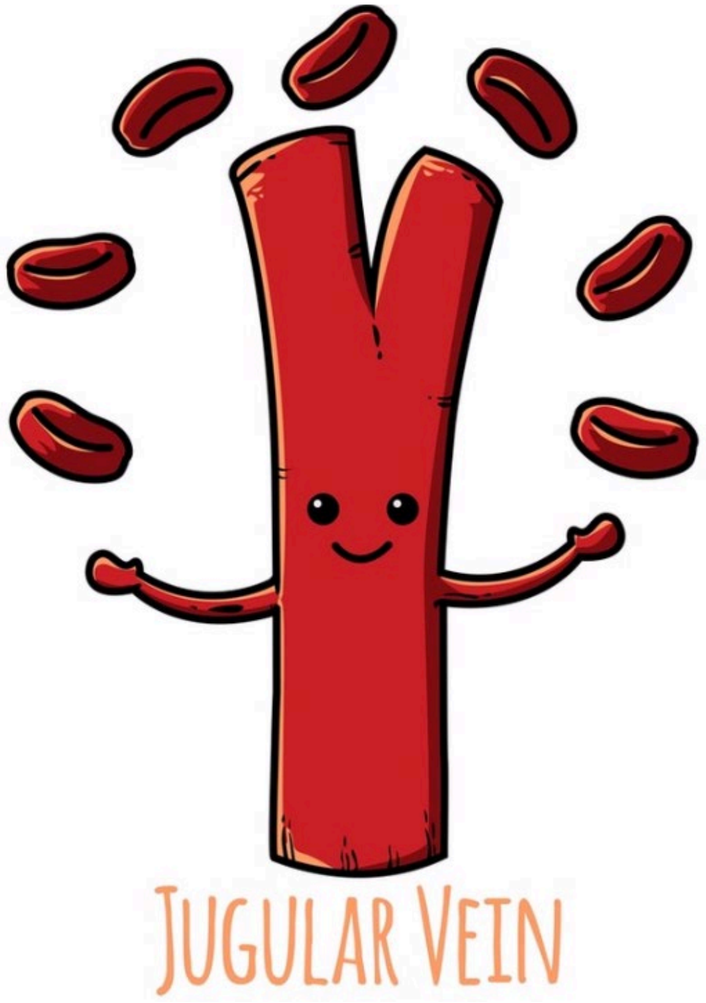


Clapp AF III, Capeleas E: Am J Cardiol 80:1469–1473, 1997

Determinants of CO - **Preload**

- MCFP (mean circulating filling pressure)
  - System at equilibrium
  - Determined by *venous smooth muscle* activation (tone/compliance) and changes in *blood volume*

1. ***Venous distensibility*** (compliance) increases during pregnancy
   - Increases progressively during pregnancy
   - Results in decrease in flow velocity and leads to stasis
   - *Mechanism:* **progesterone**

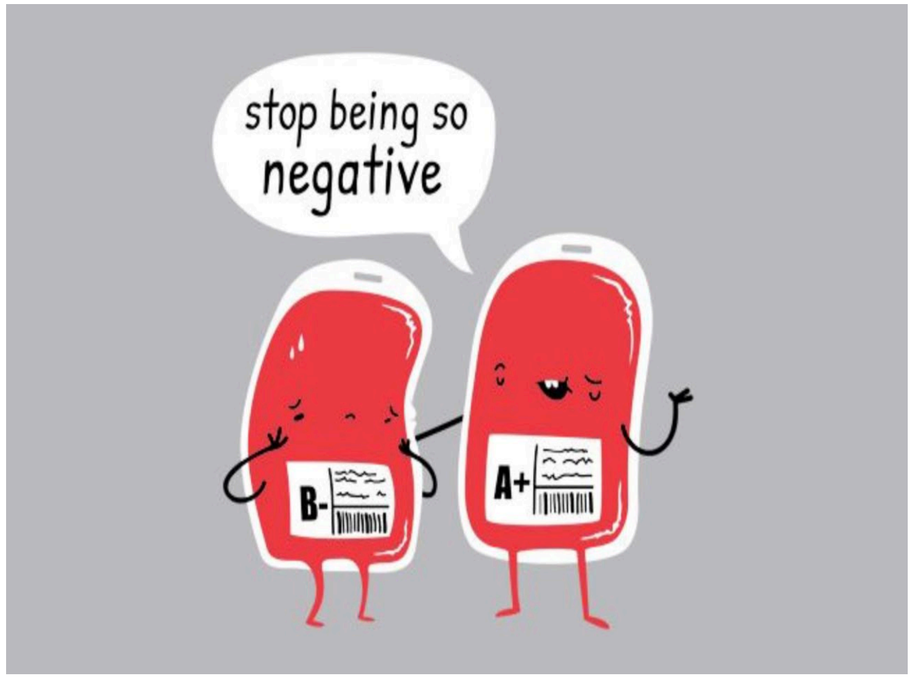


Determinants of CO - **Preload**

2. **<u>Blood volume</u>**
   - Increase begins at 8 weeks
     - Maximal volume at 28-32 weeks (4700-5200mL)
     - *Mechanism*: NO-related vasodilation which induces RAAS and stimulates Na+ and H_20 retention
   - RBC mass increases by 20-30%
     - Increase in 2,3-DPG which lowers the affinity of maternal hemoglobin for O2
     - *Mechanism*: placental chorionic somatomammotropin, **progesterone** and ***prolactin***

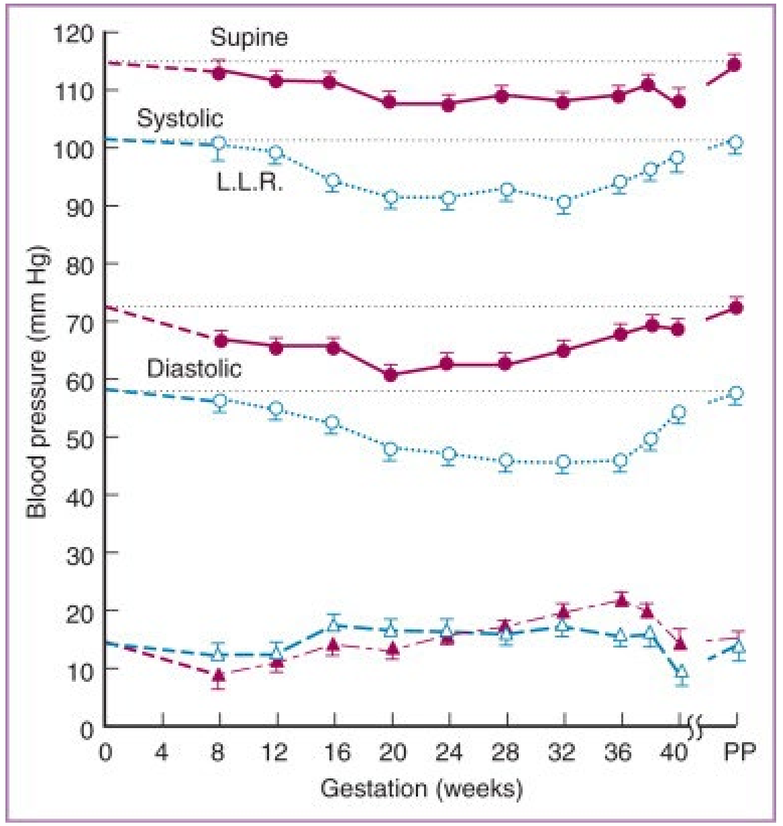
Determinants of CO - **Preload**

- Right preload = CVP 4-10 mmHg
  - How do you increase preload in the pregnant patient??
    - IV fluids
    - Colloid or blood administration
    - *Left lateral decubitus position*
  - How do you decrease preload in the pregnant patient??
    - Diuretics*
    - Hemorrhage
    - Sitting position/lying flat



Determinants of CO – **Afterload**

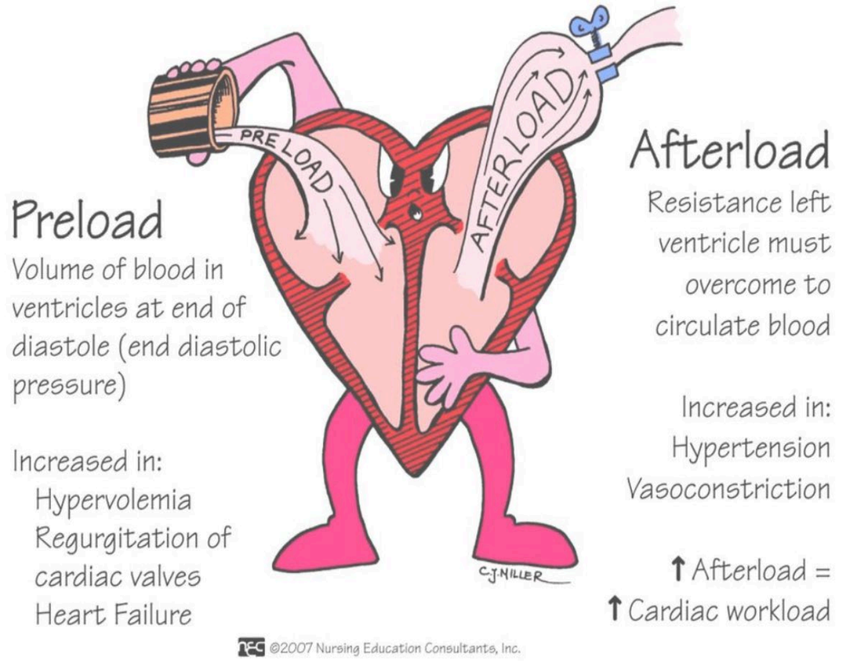
- Blood pressure
  - Decrease can be seen as early as 7 weeks
  - Systolic remains relatively stable while diastolic can decrease by a max of 10mmHg at 28 weeks
  - Increase pulse pressure
  - Marked circadian variation
    - Nadir of both in the early morning and a peak in the late afternoon and evening



Creasy, Robert K., eds. *Creasy And Resnik's Maternal-fetal Medicine: Principles And Practice*. Philadelphia, PA : Saunders/Elsevier, 2009. Print

Determinants of CO – **Afterload**

- Right-sided afterload
  - Pulmonary vascular resistance during pregnancy
- Left-sided afterload
  - Systemic vascular resistance
- How do you increase afterload in pregnancy?
  - Increase preload, administer vasopressors
- How do you decrease afterload in pregnancy?
  - Anti-hypertensives



Determinants of CO – Contractility

- **Anatomic Changes**
  - Ventricular wall muscle mass (1st trimester) and end-diastolic volume (2nd and 3rd trimester) increases
    - This increases cardiac compliance from softening of collagen without a reduction in EF
  - Myocardial contractility increases
  - Remodeling of the intimal lining
  - Internal dimensions of all cardiac chambers are increased
  - Slight regurgitation through the four valves is frequently observed
  - Increase in cross-sectional area of the left ventricular outflow tract measured at aortic annulus

Review of Maternal Physiology

	Non-pregnant	Pregnant	Change
CO (L/min)	4.3 ± 0.9	**6.2 ± 1.0**	+ 43%
HR (bpm)	71 ± 10	**83 ±10**	+ 17%
SVR (dyne-sec cm^{-5})	1530 ± 520	**1210 ± 266**	- 21%
PVR (dyne-sec cm^{-5})	119 ± 47	**78 ± 22**	- 34%
CVP (mmHg)	3.7 ±2.6	**3.6 ± 2.5**	NS
COP	20.8 ± 1.0	**18 ± 1.5**	- 14%

Review of Maternal Physiology

- **Intrapartum dynamics**
  - ***1st stage*** = 30% rise in cardiac output
  - ***2nd stage*** = 50% rise in cardiac output
  - Laboring with epidural decreases this increase
  - Contractions result in a 300-500 mL increase in blood to circulation
  - Blood pressure increases

Review of Maternal Physiology

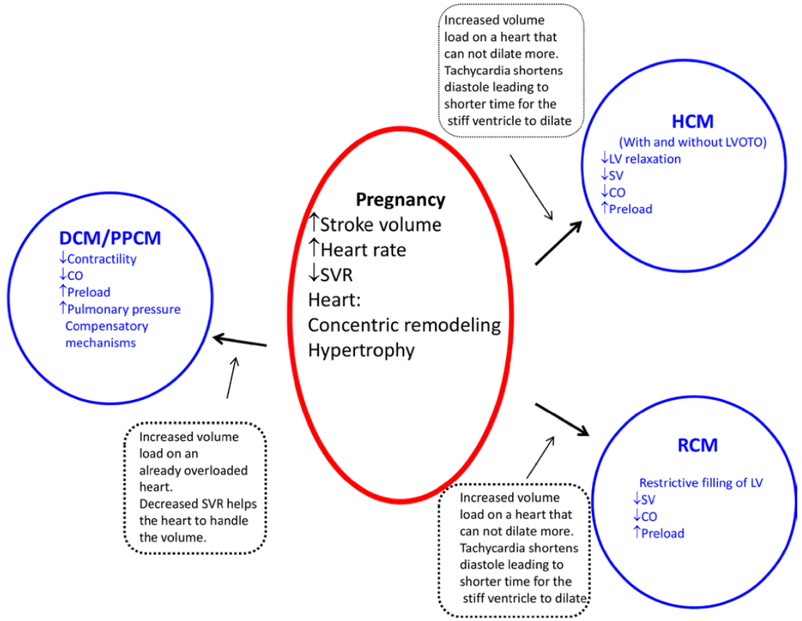
- **<u>Postpartum dynamics</u>**
  - Immediate puerperium is associated with ***80%*** increase in cardiac output
  - Release of venacaval obstruction → autotransfusion
  - Increased venous return to the heart
  - CO returns to pre-labor values 1 hr post delivery

PERIPARTUM CARDIOMYOPATHY

Peripartum Cardiomyopathy: Definition

- **Historically**
  - HF within 1 mon delivery or 5 mon PP
  - Absence of determinable etiology
  - Absence of HF before last month of pregnancy

- **ESC**
  - “an idiopathic cardiomyopathy presenting with HF secondary to LV systolic dysfunction toward the end of pregnancy where no other cause of HF is found”
  - LV dysfunction
    - LVEF < 45%
    - Fractional shortening of < 30%
    - Both



Peripartum Cardiomyopathy: Timing of Diagnosis

Elkayam U. Clinical Characteristics of Peripartum Cardiomyopathy in the United States: Diagnosis, Prognosis, and Management. JACC. 2011; 58 (7): 659-670

Peripartum Cardiomyopathy: Incidence

- **Incidence**
  - Different by geographic location
  - United States = 1 in 1000 to 1 in 4000
  - Haiti = as high as 1% of a pregnancies

Table 1 | Worldwide variation in incidence of peripartum cardiomyopathy

Country/ Region	Incidence (per live births)	Reference	Data source
Nigeria	1/102	Isezuo et al[18]	Usmanu Danfodiyo University Teaching Hospital, Sokoto, Nigeria
Haiti	≈1/300	Fett et al[19]	Hospital Albert Schweitzer PPCM Registry
China	1/346	Huang et al[20]	Liaocheng People's Hospital, Shandong Province, China
United States	1/968	Kolte et al[12]	US Nationwide Inpatient Sample
South Africa	1/1000	Desai et al[21]	King Edward VIII Hospital, Durban, South Africa
California, US	1/2066	Gunderson et al[11]	Kaiser Permanente Northern California hospitals
Malaysia	1/2941	Chee et al[22]	University Malaya Medical Centre
Sweden	1/5719*	Barasa et al[23]	National Inpatient, Cause of Death, and Medical Birth Registries
Denmark	1/10 149	Ersbøll et al[24]	Danish National Birth and Patient Registers
Japan	≈1 in 20 000	Kamiya et al[25]	Japanese Nationwide Survey of Peripartum Cardiomyopathy

*Heart failure in late pregnancy and the postpartum period.

Honigberg MC, Givertz MM. Peripartum Cardiomyopathy. BMJ. 2019; 364: k5287

Peripartum Cardiomyopathy: Risk Factors

Age

- Known independent risk factor with OR 1.7-1.8
- IPAC study with mean age 30
- US sample
  - 20-29: 1 in 1200
  - 30-39: 1 in 790
  - 40-54: 1 in 270

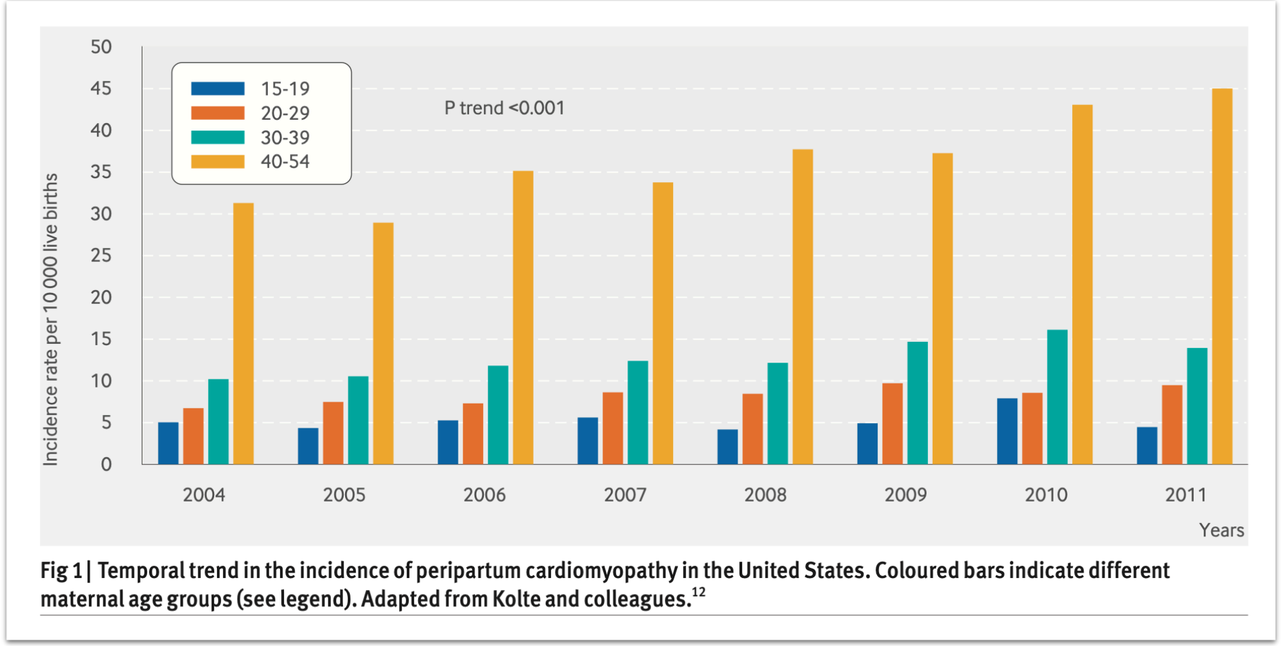
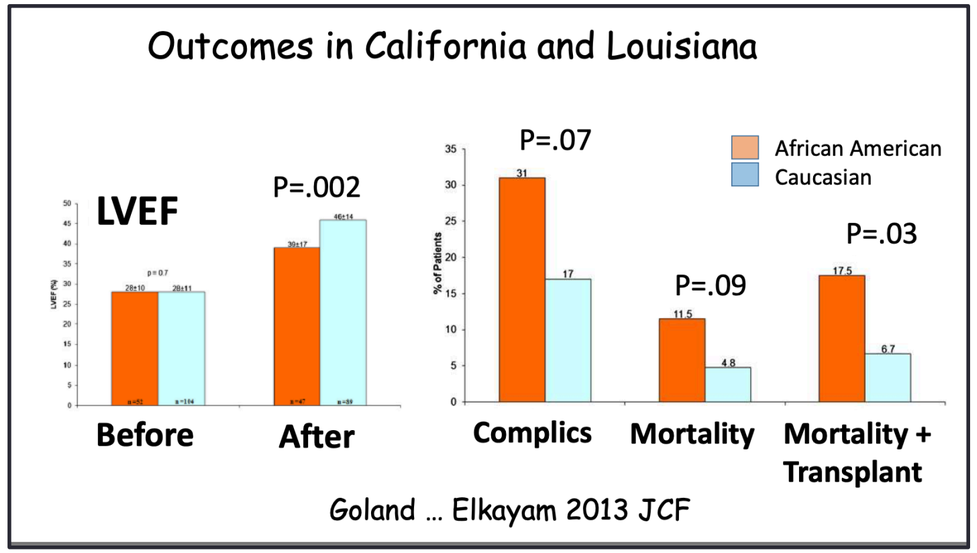


Fig 1 | Temporal trend in the incidence of peripartum cardiomyopathy in the United States. Coloured bars indicate different maternal age groups (see legend). Adapted from Kolte and colleagues.[12]

Honigberg MC, Givertz MM. Peripartum Cardiomyopathy. BMJ. 2019; 364: k5287

Peripartum Cardiomyopathy: Risk Factors

- **Race**
  - 16-fold higher in Black women
    - Accounted for half of all PPCM but only 15% of all births (US studies – *Krishnamoorthi et al, 2016*)
  - Five-fold higher mortality rates *(Harper et al, 2012)*
  - Typically younger, have higher rates of PEC, lower rates of LVEF recovery and worse outcomes *(Goland et al, 2013)*



Peripartum Cardiomyopathy: Risk Factors

- **Hypertension/Pre-eclampsia**
  - Effects up to 23% of all PPCM in US (Elkayam et al, 2005; Bello et al, 2013)
  - 37% with any hypertensive disorder (Bello et al, 2013)
  - Having PEC 12-fold increased risk of PPCM
  - ECHO findings PEC
    - LV diastolic dysfunction
    - Cardiac hypertrophy
    - PPCM with PEC only diagnosed with LV systolic dysfunction

Peripartum Cardiomyopathy: Risk Factors

- **Multifetal gestation**
  - Up to 10% of cases world-wide
  - Overall incidence of 9% of PPCM cases (compared to twin incidence of 3%)
- **Parity**
  - Silwa et al, world-wide registry reported mean avg parity of 3.6
- **Diabetes**
  - Recently added to list

Genetics of PPCM

- Current thinking favors two hit model with genetic underpinning
  - Vascular insult + underlying predisposition
- First noted familial clusters
- Similar gene variants to women with DCM
  - 172 women with PPCM and compared variants in gene sequencing to women with DCM
  - Of the 43 genes known to DCM sequenced in women with PPCM, discovered 26 variants, of which 65% were in titin gene
  - Identified in 15% of the PPCM cohort, similar to the 17% in reports of DCM
  - Presence also dictated lower EF at 12 months
  - Also found more commonly in Black women (Ware JS et al, NEJM 2016)

Pathophysiology of PPCM

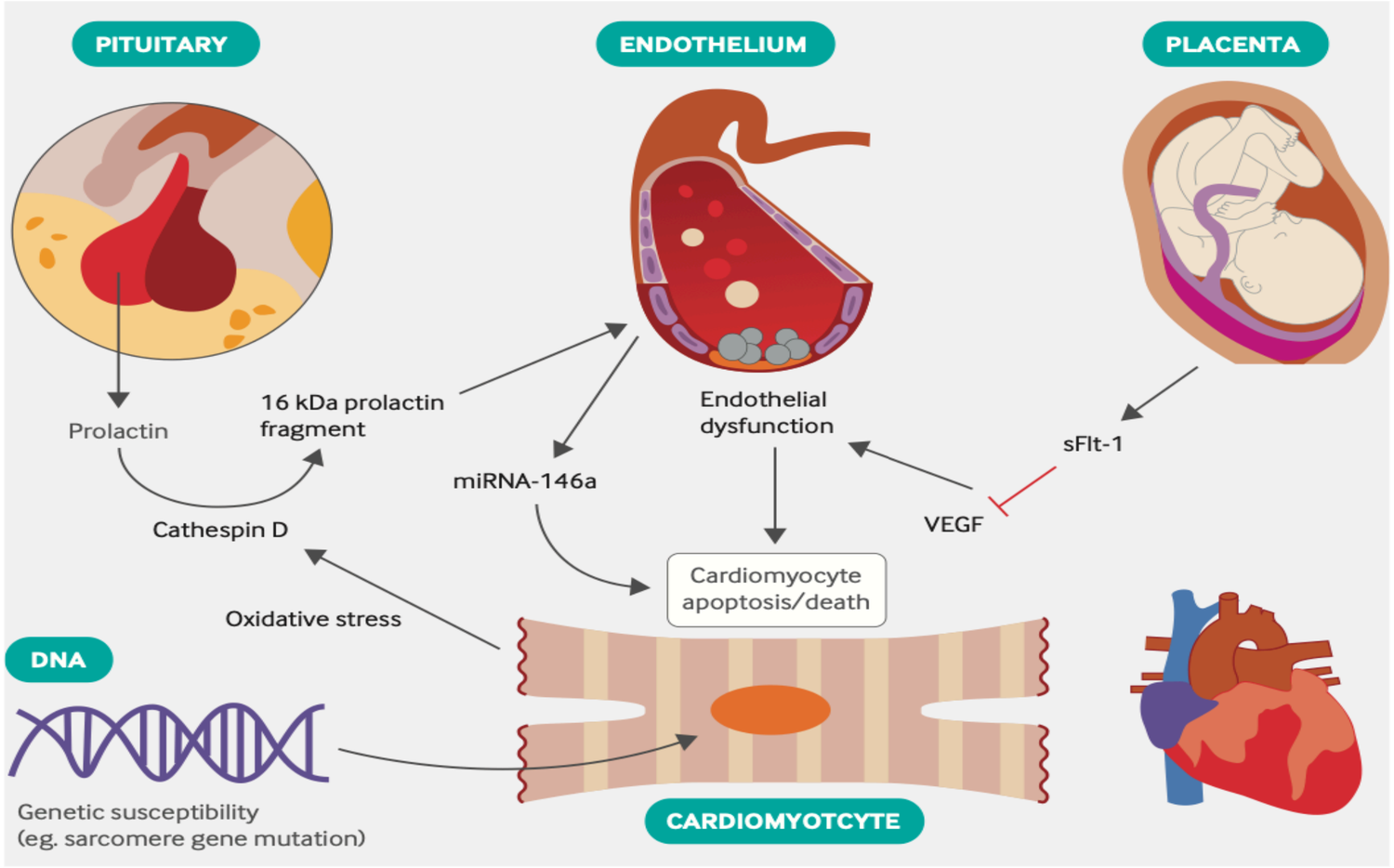


Fig 2| Pathobiology of peripartum cardiomyopathy. Secretion of prolactin by the anterior pituitary gland, upregulation of endothelial microRNA-146a (miRNA-146a), and placental secretion of soluble fms-like tyrosine kinase receptor 1 (sFlt-1) lead to endothelial dysfunction and cardiomyocyte death; genetic susceptibility is also present in some patients. VEGF=vascular endothelial growth factor. See text for details.

Honigberg MC, Givertz MM. Peripartum Cardiomyopathy. BMJ. 2019; 364: k5287

Clinical Presentation

Table 2. Cardiovascular Signs and Symptoms of Pregnancy vs. Worsening Heart Failure

Sign or symptom	Normal pregnancy	Heart failure
Fatigue	Common, relieved with rest	Progressive
Dyspnea	Common, mild not progressive	Progressive
Decreased exercise capacity	Mild	Severe or progressive
Peripheral edema	Mild, common	Severe or progressive
Jugular venous distention	Usually elevated	More pronounced
Heart murmurs	Physiologic systolic flow murmur	Fourth heart sound with CHF exacerbation
Heart rate	Sinus tachycardia common in later stage of pregnancy	SVT, ventricular arrhythmias, A-fib
Orthopnea	Common in third trimester	Not related to the stage of pregnancy
Paroxysmal nocturnal dyspnea	Not present	Increased, indicating volume overload
Persistent cough	Not present	Likely indicative of CHF
Pulmonary crackles	Rarely observed	Presence suggests CHF
Hepatomegaly	Usually absent	Usually present with CHF exacerbation
Sinus tachycardia	10%–15% above normal heart rate	>15% above normal heart rate

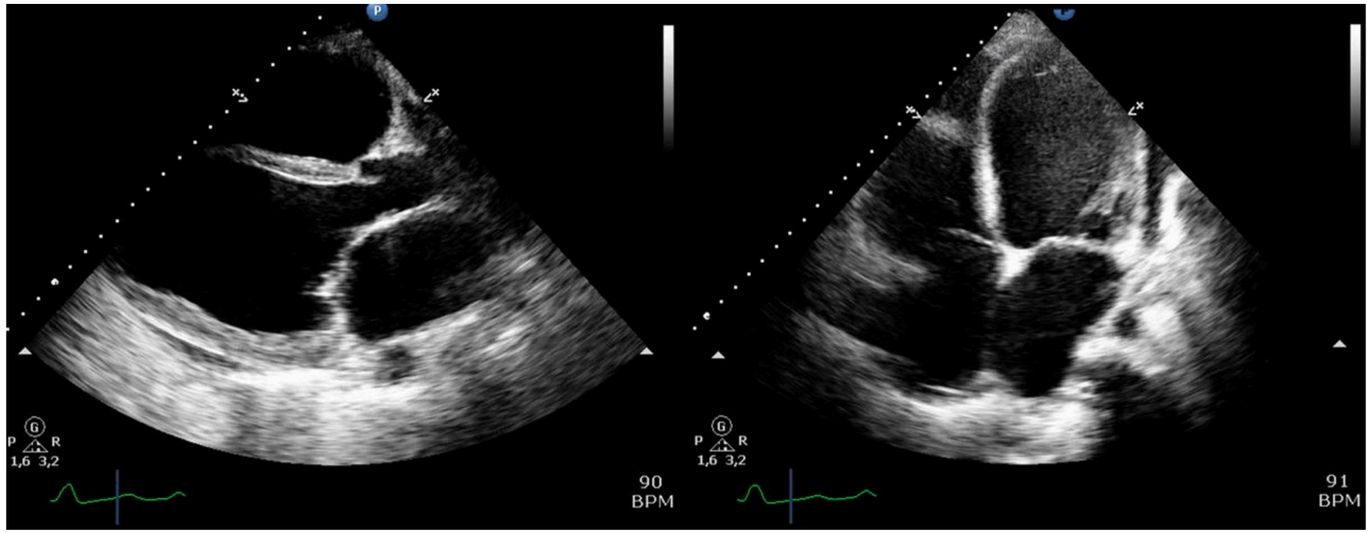
Note. A-fib = atrial fibrillation; CHF = congestive heart failure; SVT = supraventricular tachycardia. Adapted from Thorne (2004).

Cardiovascular Signs and Symptoms of Pregnancy vs. Worsening Heart Failure

Clinical Presentation

- Most women present postpartum
- Black women present later in postpartum (delay in care or true difference?)
- Physical exam/imaging findings:
  - Tachycardia
  - Third heart sound
  - Pulmonary edema
  - Non-specific T wave abnormalities on EKG
  - Elevated BNP (usually >100)
  - +/- troponin rise

Clinical Presentation: ECHO



- Evidence of LVEF < 45% and often (but not always) left ventricular dilation
- May also show:
  - Right ventricular dilation and dysfunction
  - Pulmonary hypertension
  - Left atrial or biatrial enlargement
  - Functional tricuspid or mitral regurgitation
  - Intracardiac thrombus

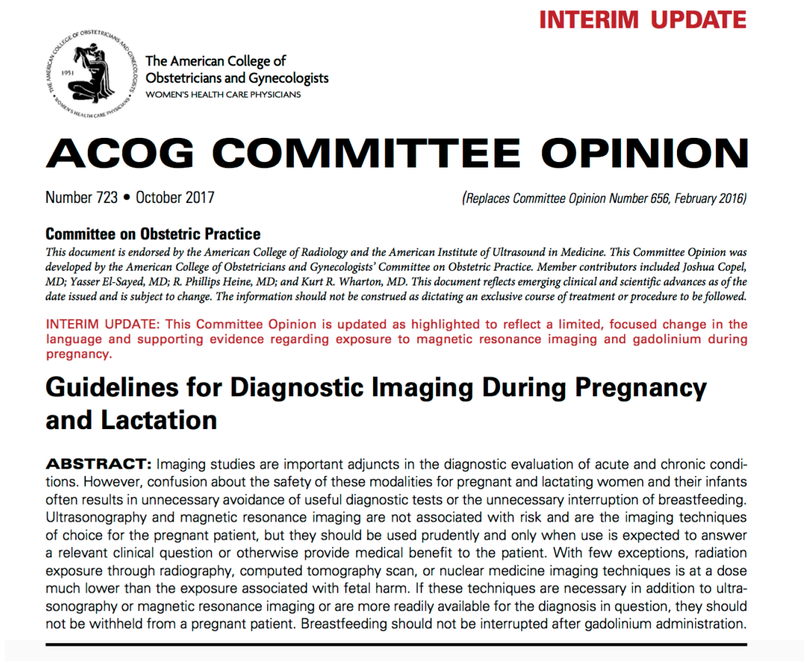
TABLE 1 Differential Diagnosis for Heart Failure During Pregnancy

Differential Diagnosis	Considerations
Takotsubo cardiomyopathy	Echocardiogram may show classic apical ballooning
Familial cardiomyopathy	Family history, genetic testing
Pre-existing cardiomyopathy	History of HF prior to pregnancy; prior echo studies with low LVEF before pregnancy
Recurrent peripartum cardiomyopathy	Ask about symptoms of HF that occurred after a prior pregnancy
Pre-eclampsia	Preserved systolic function on echocardiogram
Hypertrophic cardiomyopathy	Left ventricular hypertrophy, LVOT obstruction, preserved systolic function, genetic testing
Myocarditis	Consider if viral prodrome, histological diagnosis, fulminant presentation
Arrhythmogenic right ventricular cardiomyopathy	Consider with family history, genetic testing, echocardiographic findings
Left ventricular noncompaction	Echocardiographic and CMR findings
Chemotherapy-related cardiomyopathy	History of chemotherapy, particularly doxorubicin
Valvular heart disease	Echocardiographic findings; congenital aortic stenosis; mitral stenosis from rheumatic heart disease in endemic country. Patients with PPCM may also have valve disease, i.e., mitral regurgitation
Congenital heart disease	May be diagnosed for the first time during pregnancy by echocardiography
Tachycardia-arrhythmia mediated cardiomyopathy	Consider if specific underlying rhythm abnormality. Note that sinus tachycardia may be secondary to heart failure during pregnancy
Hypertensive heart disease	Left ventricular hypertrophy; less common in young people unless very longstanding history of hypertension
Ischemic heart disease	Cardiovascular risk factors; angina; prior CAD; consider SCAD and MINOCA diagnoses
Cardiomyopathy related to other systemic medical diseases	Consider in the appropriate context, i.e., systemic lupus erythematosus, antiphospholipid syndrome, hemochromatosis
Cardiomyopathy related to other acute conditions	May consider if patient has other conditions such as sepsis, treatment in intensive care unit, post-respiratory arrest
Pulmonary embolism	Dyspnea, tachycardia with preserved LVEF

CAD = coronary artery disease; CMR = cardiac magnetic resonance imaging; HF = heart failure; LVEF = left ventricular ejection fraction; LVOT = left ventricular outflow tract; MINOCA = myocardial infarction in non-obstructive coronary arteries; PPCM = peripartum cardiomyopathy; SCAD = spontaneous coronary artery dissection.

Diagnostic testing in pregnancy

- **CXR**
  - Remember the heart is going to be displaced upwards and left (mimicking a large heart)
  - Shielding of the abdomen
  - < 1 mRad exposure
- **MRI**
  - Particularly useful in women with aortopathies or aortic valve disease
  - Should only be used when benefits clearly outweigh risks
    - Crosses placenta, some studies have found higher uptake in fetal brain tissue, stillbirth and rheumatologic diseases
- **CT**
  - Should only be used when benefits outweigh risks (not studied in PPCM)
- **Cardiac Catheterization**
  - Exposes fetus to radiation but this can be minimized if procedure is necessary
  - Can shorten fluoro time



INTERIM UPDATE

The American College of Obstetricians and Gynecologists
WOMEN'S HEALTH CARE PHYSICIANS

ACOG COMMITTEE OPINION

Number 723 • October 2017 *(Replaces Committee Opinion Number 656, February 2016)*

Committee on Obstetric Practice

This document is endorsed by the American College of Radiology and the American Institute of Ultrasound in Medicine. This Committee Opinion was developed by the American College of Obstetricians and Gynecologists' Committee on Obstetric Practice. Member contributors included Joshua Copel, MD; Yasser El-Sayed, MD; R. Phillips Heine, MD; and Kurt R. Wharton, MD. This document reflects emerging clinical and scientific advances as of the date issued and is subject to change. The information should not be construed as dictating an exclusive course of treatment or procedure to be followed.

INTERIM UPDATE: This Committee Opinion is updated as highlighted to reflect a limited, focused change in the language and supporting evidence regarding exposure to magnetic resonance imaging and gadolinium during pregnancy.

Guidelines for Diagnostic Imaging During Pregnancy and Lactation

ABSTRACT: Imaging studies are important adjuncts in the diagnostic evaluation of acute and chronic conditions. However, confusion about the safety of these modalities for pregnant and lactating women and their infants often results in unnecessary avoidance of useful diagnostic tests or the unnecessary interruption of breastfeeding. Ultrasonography and magnetic resonance imaging are not associated with risk and are the imaging techniques of choice for the pregnant patient, but they should be used prudently and only when use is expected to answer a relevant clinical question or otherwise provide medical benefit to the patient. With few exceptions, radiation exposure through radiography, computed tomography scan, or nuclear medicine imaging techniques is at a dose much lower than the exposure associated with fetal harm. If these techniques are necessary in addition to ultrasonography or magnetic resonance imaging or are more readily available for the diagnosis in question, they should not be withheld from a pregnant patient. Breastfeeding should not be interrupted after gadolinium administration.

Diagnostic testing in pregnancy

Table 3. Fetal Radiation Doses Associated With Common Radiologic Examinations

Type of Examination	Fetal Dose* (mGy)
Very low-dose examinations (<0.1 mGy)	
Cervical spine radiography (anteroposterior and lateral views)	<0.001
Head or neck CT	0.001–0.01
Radiography of any extremity	<0.001
Mammography (two views)	0.001–0.01
Chest radiography (two views)	0.0005–0.01
Low- to moderate-dose examinations (0.1–10 mGy)	
Radiography	
Abdominal radiography	0.1–3.0
Lumbar spine radiography	1.0–10
Intravenous pyelography	5–10
Double-contrast barium enema	1.0–20
CT	
Chest CT or CT pulmonary angiography	0.01–0.66
Limited CT pelvimetry (single axial section through the femoral heads)	<1
Nuclear medicine	
Low-dose perfusion scintigraphy	0.1–0.5
Technetium-99m bone scintigraphy	4–5
Pulmonary digital subtraction angiography	0.5
Higher-dose examinations (10–50 mGy)	
Abdominal CT	1.3–35
Pelvic CT	10–50
^{18}F PET/CT whole-body scintigraphy	10–50

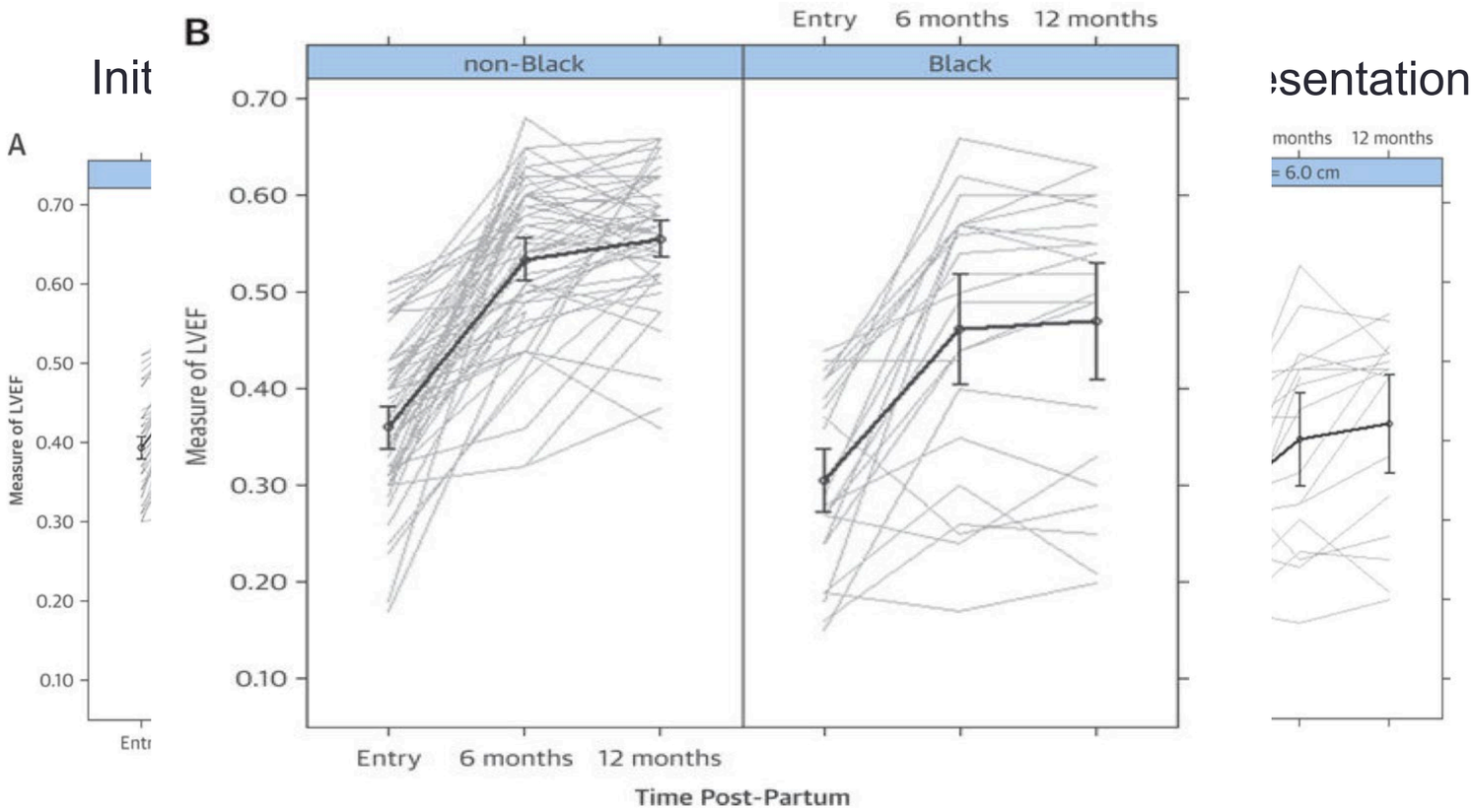
Abbreviations: CT, computed tomography; PET, positron emission tomography.

*Fetal exposure varies with gestational age, maternal body habitus, and exact acquisition parameters.

Note: Annual average background radiation = 1.1–2.5 mGy, ^{18}F = 2-[fluorine-18]fluoro-2-deoxy-D-glucose.

Modified from Tremblay E, Therasse E, Thomassin-Naggara I, Trop I. Quality initiatives: guidelines for use of medical imaging during pregnancy and lactation. Radiographics 2012;32:897–911.

Prognosis: Investigation of Pregnancy-Associated Cardiomyopathy: IPAC Trial



McNamara D et al. Clinical outcomes for peripartum cardiomyopathy in North America. JACC. 2015; 66(8): 905-914.

Complications

- **Thromboembolism**
  - Affects up to 6.6%
  - Can occur in both left and right side
  - Mechanism: cardiac dilation, hypocontractility, endothelial injury, hypercoagulable state of pregnancy

- **Arrhythmias**
  - Can affect up to 18.7% *(Mallikethi-Reddy S et al, Clin Res Cardiol 2017)*
  - Risk of sudden death from ventricular tachyarrhythmias

- **Cardiogenic shock and need for assist device**

Medical Management

- Standard HF treatments
  - Sodium restriction
  - Loop diuretics
  - β-blockade if hemodynamics permit (preferably β1 selectives)
  - ACE inhibitors (not while pregnant)
  - Hydralazine and nitrites
  - Digoxin
  - Spironolactone (not while pregnant)
  - Anticoagulation
  - Antiarrhythmics
    - Cardioversion is compatible with pregnancy

FIGURE 1 Heart Failure and Anticoagulant Medications: Indications and Safety in Pregnancy and During Lactation

MEDICATION	DURING PREGNANCY	POTENTIAL ADVERSE EFFECTS	INDICATIONS	DURING LACTATION
HEART FAILURE MEDICATIONS				
Loop diuretics	Yes	Caution for hypovolemia or hypotension that may lead to decreased placental perfusion	For signs and symptoms of congestion and fluid overload.	Yes, but over-diuresis can lead to decreased milk production.
Beta blockers (metoprolol tartrate used most commonly)	Yes	IUGR; fetal bradycardia and hypoglycemia	For standard treatment of HF; consider treatment of women with subsequent pregnancy.	Yes
Hydralazine/nitrates	Yes	Caution with hypotension	Use for afterload reduction during pregnancy (instead of ACE-I/ARB) when needed.	Yes, but ACE-I/ARB typically chosen post-partum
Digoxin	Yes	No associated congenital defects	Can be used with symptomatic heart failure and/or systolic dysfunction during pregnancy, or afterwards per guidelines.	Yes
ACE-I/ARB	No	Anuria, oligohydramnios, fetal limb contractures, craniofacial deformation, pulmonary atresia, fetal hypocalvaria, intra uterine growth restriction, prematurity, patent ductus arteriosus, stillbirth, neonatal hypotension and death	Cannot use during pregnancy. After delivery, should be used as part of guideline-directed medical therapy for afterload reduction and LV remodeling.	Enalapril and captopril can be used
Aldosterone receptor antagonists	No	Spironolactone has been associated with antiadrenergic activity, feminization of male rat fetuses and permanent changes in reproductive tract in both sexes	As per guideline-directed medical therapy for heart failure.	Spironolactone can be used
Sacubitril-valsartan	No	Same as ACE-I/ARB	As per guideline-directed medical therapy for heart failure.	No information in human, present in rat milk
Ivabradine	Scant data in humans; would avoid due to concerns in animal studies	Scant data in humans, animal data suggest risk	As per guideline-directed medical therapy for heart failure.	No information in human, present in rat milk
ANTICOAGULANTS				
Low molecular weight heparin	Yes	Caution at time of delivery and with neuraxial anesthesia; does not cross placenta; consider the need for monitoring anti-Xa levels	For prevention and treatment of thromboembolic complications during pregnancy and as bridge to warfarin postpartum.	Yes
Warfarin	Avoid	Warfarin embryopathy and fetopathy	For prevention and treatment of thromboembolic complications postpartum.	Yes

Legend:

Data or experience to support use
Caution with using this medication
Data is limited or inconclusive

Safety of medications need to be considered during pregnancy and lactation. ACE-I = angiotensin-converting enzyme inhibitors; ARB = angiotensin receptor blocker; HF = heart failure; IUGR = intra-uterine growth restriction; LV = left ventricular; SSP = subsequent pregnancy.

Experimental Treatments

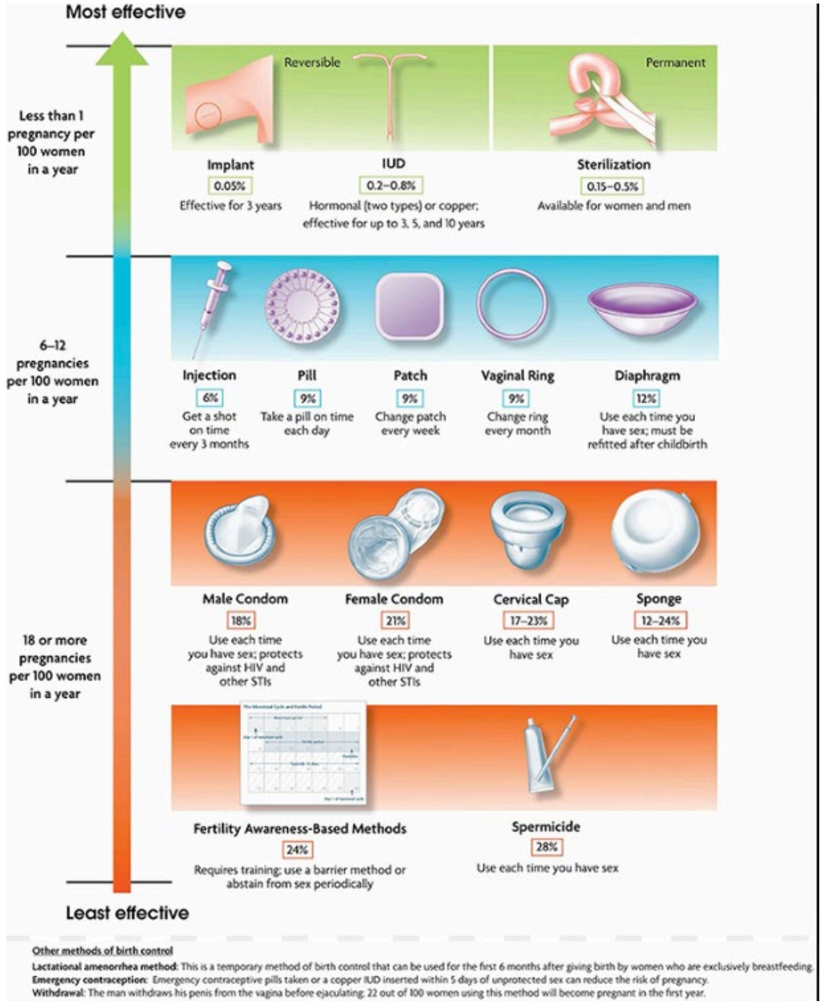
- Prolactin inhibition
  - Bromocriptine is a dopamine agonist and inhibits release of prolactin
  - PPCM is driven by 16kD form of prolactin → will this work??
  - Multiple European and African trials showing success
    - German trial: observational registry showed 72% improvement with bromocriptine use compared to 35% without treatment
    - African trial: 20 women received and experienced larger improvement in LVEF from 27% to 58% vs. non-medical intervention group (27% to 36%)

Subsequent Pregnancies with PPCM

- LVEF is predictor of subsequent outcomes
  - Review of 191 subsequent pregnancies
    - Women with LVEF < 50% = 50% risk of acute heart failure with worsening cardiomyopathy *(Elkayam U et al, ACC 2014)*
  - Series from South Africa
    - 20-25% mortality rates *(Elkayam U et al, ACC 2014)*
  - Normalized LVEF
    - Still have a 20% risk of deterioration
- Fetal outcomes
  - Stillbirth, miscarriage and preterm delivery are more common in women with persistent LVEF < 45%
- Recommendations for future pregnancy
  - Strong counseling re: permanent loss of function
  - Use of stress echo to evaluation contractile reserve??

Contraception

- Do not shy away from this conversation!!
- Consideration of systemic hormones, local hormones, desire to breastfeed, uterine structure, and future fertility plans
- In general, LARC preferred for this population



Curr Treat Options Cardio Med (2020) 22: 68
DOI 10.1007/s11936-020-00853-7

Pregnancy and Cardiovascular Disease (N Scott, Section Editor)

Contraception for the Cardiac Patient: a Cardiologist's Primer

Karen L. Florio, DO[1,2,*]
Monica Kao, MD MPH[2]
Traci Johnson, MD[2]
Heidi A. Tuttle, PharmD CDCES[3]
Darcy White, RN[1]
Lynne Nelson, WHNP[1]
Neil Patel, MD[2]
Devon Ramaeker, MD[1]
Sue Kendig, JD, BC-WHNP[5]
Laura Schmidt, MD[2,4]
Anna Grodzinsky, MD, MSc[2,4]
Katherine Economy, MD[6]

Address
[*,1]Heart Disease in Pregnancy Program, Saint Luke's Hospital of Kansas City, Kansas City, MO, 64111, USA
Email: Kflorio@saintlukeskc.org
[2]University of Missouri-Kansas City School of Medicine, Kansas City, MO, USA
[3]Kansas City, MO, USA
[4]Saint Luke's Mid-America Heart Institute, Kansas City, MO, USA
[5]SSM Health Saint Louis University Hospital, St. Louis, MO, USA
[6]Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA

FIGURE 2 Counseling and Management of Subsequent Pregnancies in PPCM

Subsequent Pregnancy	Recovered (LVEF ≥50%)	Nonrecovered (LVEF <50%)
Preconception or First Visit	Preconception risk counseling and follow-up planning. Clinical and LVEF reassessment off renin-angiotensin blocking agents for 3 months. Baseline echocardiogram and BNP/NT-proBNP level.	Preconception risk counseling including discussion of alternative ways to build a family. If pregnant and not considering termination: Close follow-up planning, stop renin-angiotensin blocking agents and switch to hydralazine/isosorbide dinitrate. Baseline echocardiogram and BNP/NT-proBNP level.
Maternal Risks	~20% have a relapse Severe deterioration is rare Mortality unlikely Rate of subsequent recovery is high	Higher risk of relapse ~50% show further deterioration in LV dysfunction Increased morbidity and mortality Premature delivery and abortion more common
Medications	Continue beta blocker therapy (metoprolol tartrate preferred). Yield of starting prophylactic beta blocker therapy unclear. Diuretics and hydralazine/isosorbide dinitrate in case of clinical or LV functional deterioration.	Continue beta blocker therapy (metoprolol tartrate preferred). Hydralazine/Isosorbide dinitrate for hemodynamic and symptomatic improvement. Consider digoxin. Consider anticoagulation if severe LV dysfunction (LVEF <35%).
Follow-up	Close monitoring of symptoms during pregnancy and the postpartum period with repeat echocardiographic assessment of LV function and BNP/NT-proBNP level at the end of the 1st and 2nd trimesters, 1 month prior to delivery, after delivery prior to hospital discharge, 1 month postpartum, and at any time if symptoms develop.	
Labor and Delivery	Multidisciplinary team for planning; patient involved. Spontaneous vaginal delivery preferred unless fetal or maternal instability. Monitor for volume overload in the first 48 hours after delivery in cases of recurrent LV dysfunction.	Multidisciplinary team for planning; patient involved. Spontaneous vaginal delivery preferred unless fetal or maternal instability. Early delivery if further decrease in LV function and hemodynamic deterioration. Consider hemodynamic monitoring for optimization prior to delivery and monitoring during and after delivery. Monitor for volume overload in the first 48 hours after delivery.

Risks of a subsequent pregnancy differ based on the pre-conception recovery status. There is higher risk with nonrecovered myocardial function and pregnancy should be discouraged. Peripartum management options depend on the clinical status and myocardial function. ACE = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; LV = left ventricular; LVEF = left ventricular ejection fraction; PPCM = peripartum cardiomyopathy.

CENTRAL ILLUSTRATION **Diagnosis, Management, and Outcomes for Peripartum Cardiomyopathy**

Peripartum Cardiomyopathy (PPCM)

Definition:

- Non-ischemic cardiomyopathy with reduced LVEF (<45%)
- Commonly presents in the first months postpartum or towards the end of pregnancy

Risk Factors:

- African-American race, preeclampsia, hypertension, multigestational pregnancies, age >30 years

Symptoms:

- Heart failure symptoms can be confused with common symptoms of normal pregnancy

Management Options for PPCM

During Pregnancy:

- Beta-blockers, loop diuretics, hydralazine/isosorbide dinitrate, digoxin, low-molecular-weight heparin
- (No ACE/ARB/aldosterone receptor antagonists)
- MCS for severe heart failure/cardiogenic shock
- Consider early delivery if unstable

Delivery:

- Plan ahead with a Cardio-Obstetrics Team
- If unstable, consider hemodynamic monitoring and optimization
- Caution for fluid overload, especially after delivery

After Pregnancy:

- Heart failure management. Beta-blockers, enalapril, and spironolactone are compatible with breastfeeding.
- Anticoagulation for LV thrombus; consider if severe LV dysfunction (LVEF <35%)
- Consider a wearable cardioverter/defibrillator if severe LV dysfunction
- Discuss Contraception

Outcomes

Worse prognosis with lower LVEF, dilated LV, African-American race, and delayed diagnosis.

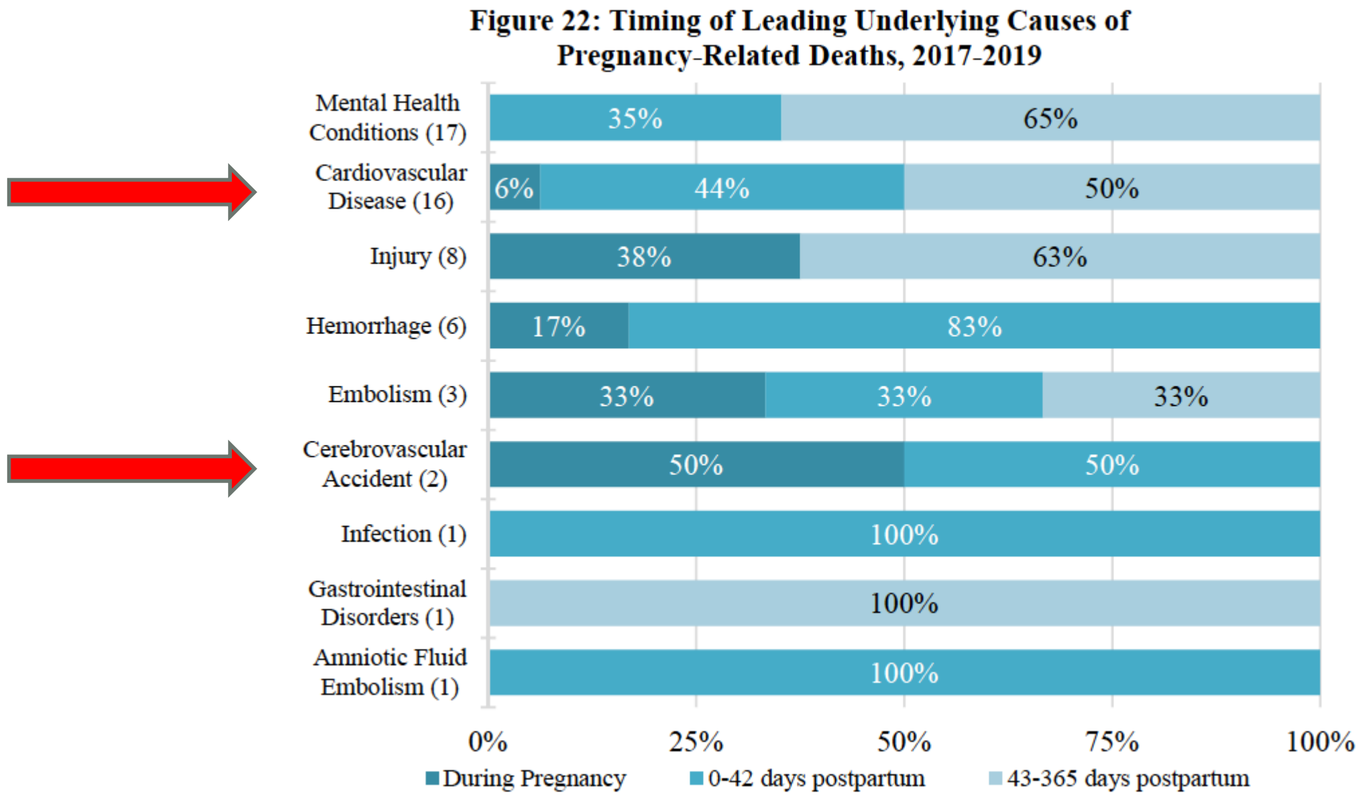
Long-term Outcomes

- After recovery, optimal duration of medication treatment is unknown
- In the case of stopping medications, wean gradually and observe closely
- Continue surveillance after recovery

Davis, M.B. et al. J Am Coll Cardiol. 2020;75(2):207-21.

Diagnostic criteria, symptoms, and risk factors can aid in the diagnosis. Medications need to be tailored to pregnancy and breastfeeding status. Short- and long-term outcomes are variable and serial follow-up is important. ACE = angiotensin converting enzyme inhibitor; ARB = angiotensin receptor blocker; LV = left ventricle; LVEF = left ventricular ejection fraction; MCS = mechanical circulatory support; PPCM = peripartum cardiomyopathy.

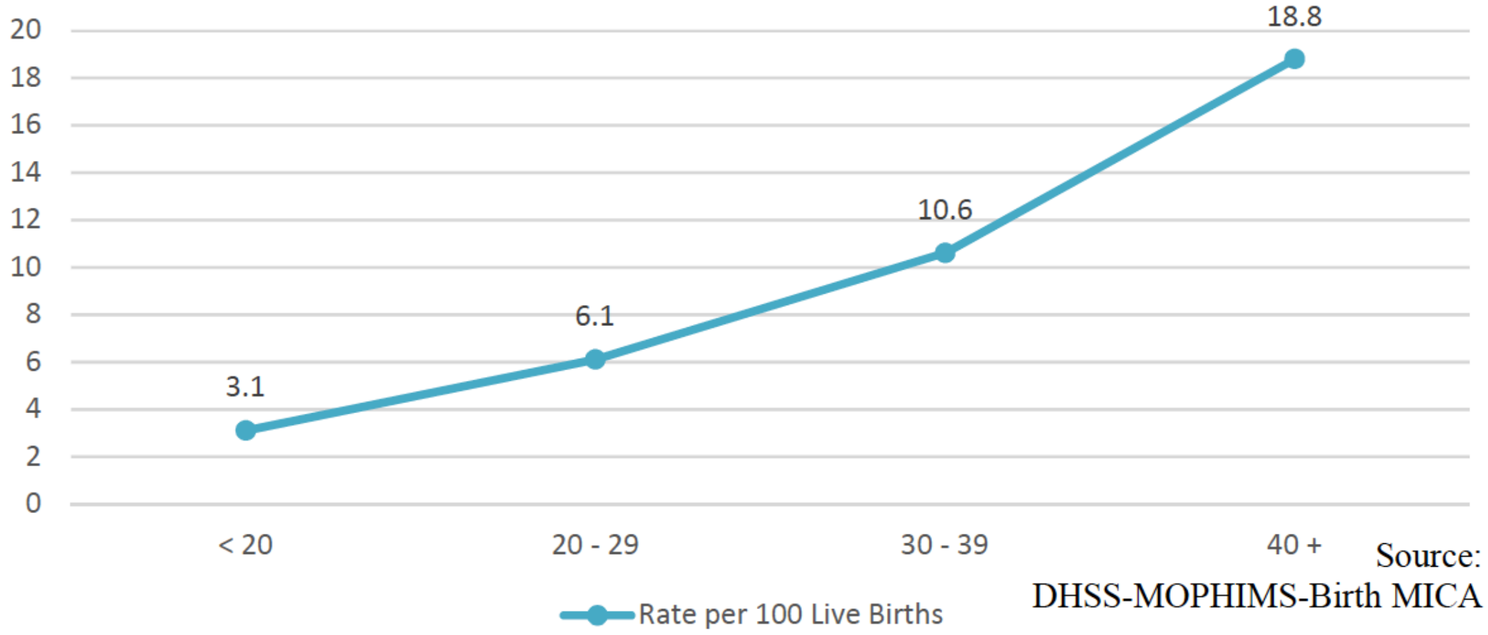
LEADING CAUSE OF PREGNANCY-RELATED DEATH IN MO 2017-2019



Of the 18 cases where the board disagreed with cause of death, 72% of the pregnancy-related were deemed to be from CVD and 50% died postpartum

Why is this happening??

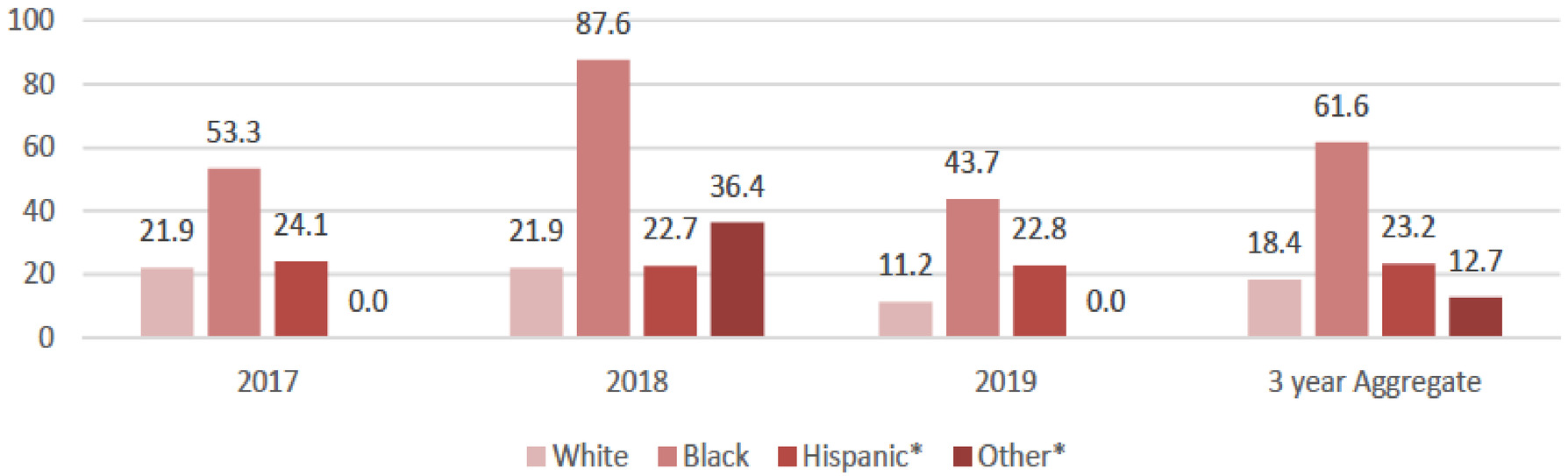
Figure 6: Rate of Diabetes by Age, 2017-2019



Source: DHSS-MOPHIMS-Birth MICA

Who is affected??

Figure 16: Pregnancy-Related Mortality Ratio per 100,000 Live Births by Race



Timing of Deaths

Figure 10: Timing of Death by Relationship to Pregnancy, 2017-2019

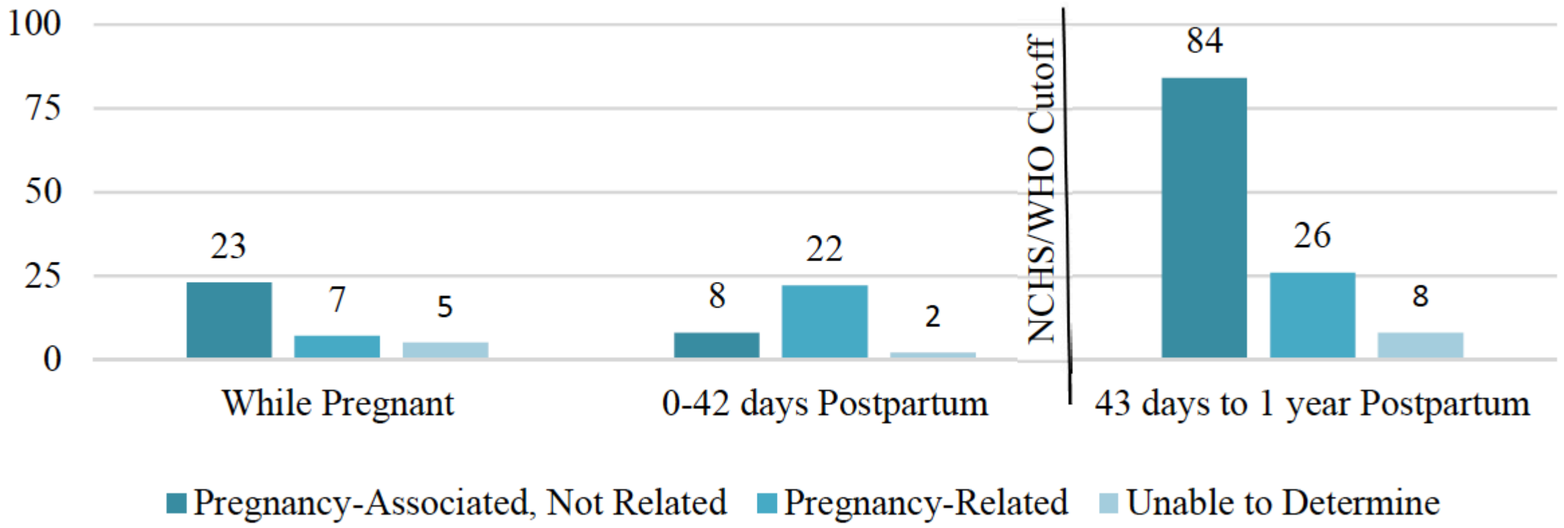
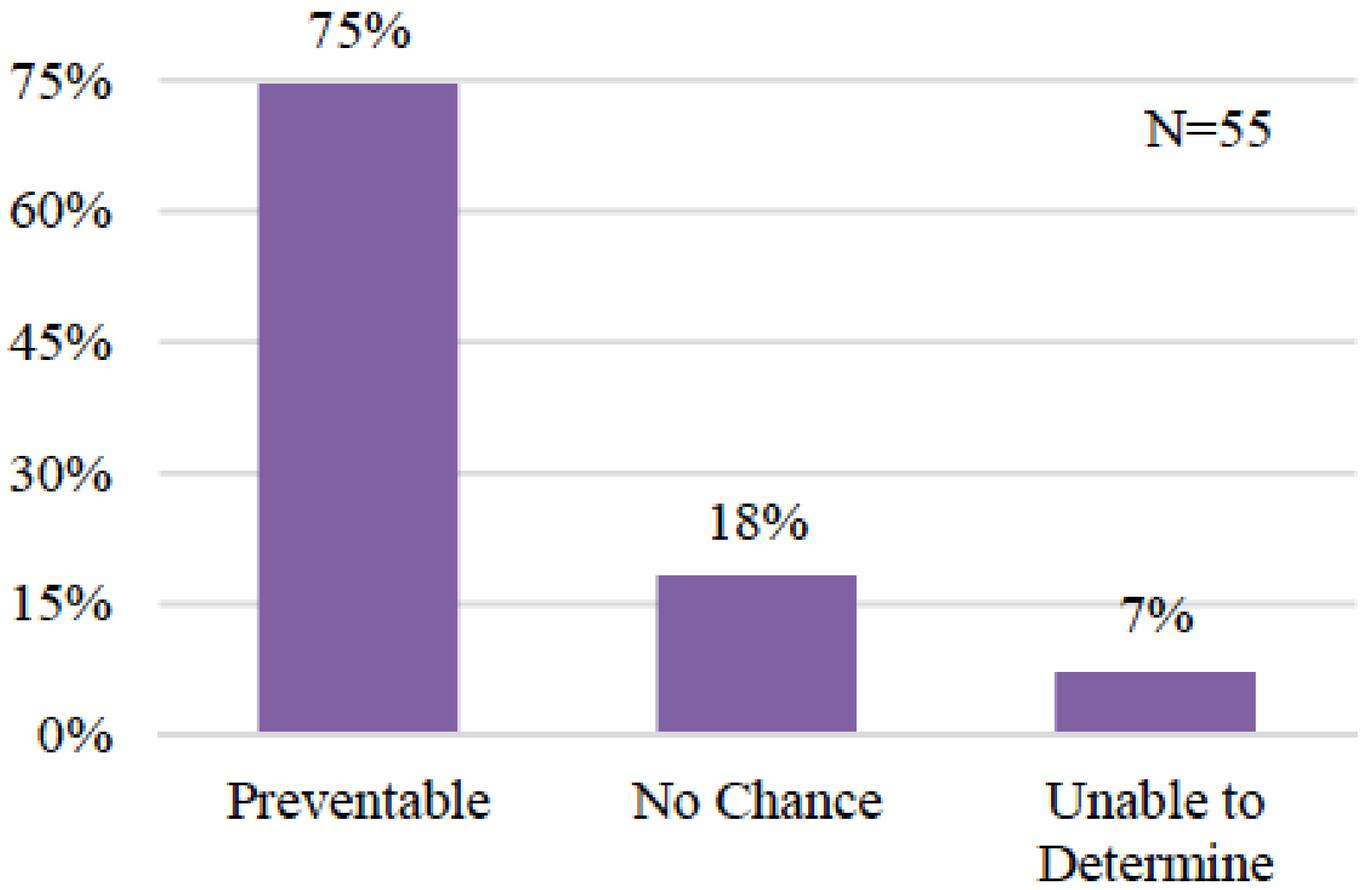


Figure 31: Chance to Alter Outcome of Pregnancy-Related Deaths, 2017-2019



56% of deaths due to CVD were determined to be preventable and the leading cause was ***access/financial barriers, assessment and continuity of care***

What can we do about it??

AIM

ALLIANCE FOR INNOVATION
ON MATERNAL HEALTH

**Cardiac Conditions
in Obstetrical Care**

Summary

- There are a number of cardiovascular changes in pregnancy that contribute to disease states
- PPCM is likely a two-hit disease, with vasogenic insult coupled on top of genetic predisposition
- The signs and symptoms of PPCM can be subtle so don’t blow them off for “normal pregnancy symptoms”
- Treatment is similar to HF, with some exceptions during gestation
- The risk for poor outcomes and future pregnancy complications are predicted on the LVEF and LVEDD
- Don’t be afraid of the contraceptive talk

Questions??

Resources

1. Arany Z. Understanding Peripartum Cardiomyopathy. *Annu Rev Med*. 2018;69(1):165-176. doi:10.1146/annurev-med-041316-090545
2. Baris L, Cornette J, Johnson MR, Sliwa K, Roos-Hesselink JW. Peripartum cardiomyopathy: disease or syndrome? *Heart Br Card Soc*. 2019;105(5):357-362. doi:10.1136/heartjnl-2018-314252
3. Bello N, Rendon ISH, Arany Z. The Relationship Between Pre-Eclampsia and Peripartum Cardiomyopathy. *J Am Coll Cardiol*. 2013;62(18):1715-1723. doi:10.1016/j.jacc.2013.08.717
4. Davis MB, Arany Z, McNamara DM, Goland S, Elkayam U. Peripartum Cardiomyopathy: JACC State-of-the-Art Review. *J Am Coll Cardiol*. 2020;75(2):207-221. doi:10.1016/j.jacc.2019.11.014
5. Elkayam U. Clinical Characteristics of Peripartum Cardiomyopathy in the United States. *J Am Coll Cardiol*. 2011;58(7):659-670. doi:10.1016/j.jacc.2011.03.047
6. Elkayam U. *Cardiac Problems in Pregnancy*. 4th ed. Wiley Blackwell; 2020.
7. Florio KL, DeZorzi C, Williams E, Swearingen K, Magalski A. Cardiovascular Medications in Pregnancy: A Primer. *Cardiol Clin*. 2021;39(1):33-54. doi:10.1016/j.ccl.2020.09.011
8. Florio KL, Kao M, Johnson T, et al. Contraception for the Cardiac Patient: a Cardiologist's Primer. *Curr Treat Options Cardiovasc Med*. 2020;22(12):68. doi:10.1007/s11936-020-00853-7
9. Goland S, Modi K, Hatamizadeh P, Elkayam U. Differences in Clinical Profile of African-American Women With Peripartum Cardiomyopathy in the United States. *J Card Fail*. 2013;19(4):214-218. doi:10.1016/j.cardfail.2013.03.004
10. Haghikia A, Podewski E, Libhaber E, et al. Phenotyping and outcome on contemporary management in a German cohort of patients with peripartum cardiomyopathy. *Basic Res Cardiol*. 2013;108(4):366. doi:10.1007/s00395-013-0366-9
11. Hilfiker-Kleiner D, Haghikia A, Berliner D, et al. Bromocriptine for the treatment of peripartum cardiomyopathy: a multicentre randomized study. *Eur Heart J*. 2017;38(35):2671-2679. doi:10.1093/eurheartj/ehx355
12. Honigberg MC, Givertz MM. Peripartum cardiomyopathy. *BMJ*. Published online January 30, 2019:k5287. doi:10.1136/bmj.k5287
13. Jain C. ACOG Committee Opinion No. 723: Guidelines for Diagnostic Imaging During Pregnancy and Lactation. *Obstet Gynecol*. 2019;133(1):186. doi:10.1097/AOG.0000000000003049
14. Krishnamoorthy P, Garg J, Palaniswamy C, et al. Epidemiology and outcomes of peripartum cardiomyopathy in the United States: findings from the Nationwide Inpatient Sample. *J Cardiovasc Med*. 2016;17(10):756-761. doi:10.2459/JCM.0000000000000222
15. McNamara DM, Elkayam U, Alharethi R, et al. Clinical Outcomes for Peripartum Cardiomyopathy in North America. *J Am Coll Cardiol*. 2015;66(8):905-914. doi:10.1016/j.jacc.2015.06.1309
16. Sliwa K, Petrie MC, Hilfiker-Kleiner D, et al. Long-term prognosis, subsequent pregnancy, contraception and overall management of peripartum cardiomyopathy: practical guidance paper from the Heart Failure Association of the European Society of Cardiology Study Group on Peripartum Cardiomyopathy. *Eur J Heart Fail*. 2018;20(6):951-962. doi:10.1002/ejhf.1178
17. Moulig V, Pfeffer TJ, Ricke-Hoch M, et al. Long-term follow-up in peripartum cardiomyopathy patients with contemporary treatment: low mortality, high cardiac recovery, but significant cardiovascular co-morbidities. *Eur J Heart Fail*. 2019;21(12):1534-1542. doi:10.1002/ejhf.1624
18. Schaufelberger M. Cardiomyopathy and pregnancy. *Heart*. 2019;105(20):1543-1551. doi:10.1136/heartjnl-2018-313476
19. Ware JS, Seidman JG, Arany Z. Shared Genetic Predisposition in Peripartum and Dilated Cardiomyopathies. *N Engl J Med*. 2016;374(26):2601-2602. doi:10.1056/NEJMc1602671
20. Williams R. *Missouri Pregnancy Associated Morality Review: 2017 Annual Report*. Missouri Department of Health of Human Services; 2020:46.
21. Resnik R, Lockwood CJ, Moore T, Greene M, Copel J, Silver R. *Creasy and Resnik's Maternal-Fetal Medicine: Principles and Practice*. 8th ed. Saunders/Elsevier; 2018.

How did you find us?








Upcoming TAP Webinars

Register at saferbirth.org under Resources > Events

Improving Post-birth Education about Potential Complications

October 20th at 3 PM ET

LaShea Haynes
M.Ed., MSN, AGCNS-BC, APRN, RNC-OB/EFM

Implementing A Stage-Based Obstetric Hemorrhage Emergency Management Plan

November 3rd at 3 PM ET

Dr. Dena Goffman, MD
& Dr. Lisa Nathan, MD, MPH

Thank you!

The recording will be emailed to all attendees once ready

AIM will also post the recording on our website, saferbirth.org

Any questions about this webinar or the series can be sent to AIM@acog.org

Remember to register for upcoming webinars!