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Editorial

Cardio-Obstetrics: A Review for the Cardiac Anesthesiologist

MATERNAL cardiac disease complicates up to 4% of pregnancies and is the leading cause of pregnancy-related mortality in the Unites States.[1,2] Care of pregnant patients with cardiovascular disease is performed best with a multidisciplinary team, referred to as the Pregnancy Heart Team. The Pregnancy Heart Team typically consists of maternal-fetal medicine specialists, cardiologists, obstetric anesthesiologists, and neonatologists. For patients with severe cardiac disease, additional specialists, such as intensivists, cardiothoracic (CT) surgeons, CT anesthesiologists, obstetric and critical care nurses, and others, may need to be involved. The Pregnancy Heart Team evaluates cardio-obstetric patients and devises a comprehensive plan to manage the cardiovascular disease during pregnancy, delivery, and the postpartum period. The design of the Pregnancy Heart Team facilitates multidisciplinary coordination and communication. The CT anesthesiologist may be called to participate in the care of patients with severe cardiac lesions for cardiac procedures during pregnancy or to assist in hemodynamic management during delivery. A strong partnership between CT and obstetric anesthesiologists will allow for comprehensive, optimal specialty specific care.

Cardiovascular Physiology of Pregnancy and Delivery

The maternal cardiovascular system undergoes major changes during pregnancy to provide adequate oxygenation for the growing fetus. Cardiac output (CO) increases by up to 50% during pregnancy due to an increase in both heart rate (HR) and stroke volume. Plasma volume increases throughout pregnancy, reaching a plateau of a 50% increase in the third trimester. Systemic vascular resistance and pulmonary vascular resistance decrease, and central filling pressures are unchanged.[3] Red blood cell mass increases proportionally less then plasma volume, resulting in dilutional anemia. Plasma oncotic pressure decreases by 15%. Cardiac imaging, including transthoracic echocardiography and magnetic resonance imaging, demonstrates evidence of cardiac remodeling during pregnancy. Atrial and ventricular chamber dilation are present and usually more prominent in the atria. Left ventricular mass increases and mild physiologic regurgitation can be seen in the mitral, tricuspid, and pulmonic valves.[4,5]

The process of labor and delivery includes additional cardiovascular changes to accommodate the progressive increase in CO with uterine contractions (300- to 500-mL increase in preload may occur with every uterine contraction). Cardiac output peaks immediately after delivery at 150% over the prelabor baseline. Heart failure can occur during this period.[6] Delivery of the infant usually requires maternal expulsive efforts and the use of the Valsalva maneuver, which can cause significant physiologic changes with each effort. During initial stages of the Valsalva maneuver, intrathoracic pressure increases and venous return decreases, resulting in decreased preload and mean arterial pressure (MAP) and a compensatory increase in HR. As the Valsalva is released, venous return is restored and MAP increases, potentially exceeding baseline values.[7] This drastic change in cardiac filling and output results in increased shear stress to the aorta, which can be deleterious to women with preload-sensitive lesions or aortic pathology. During delivery, this cycle can continue for minutes or hours before the infant is delivered. Immediate and complete contraction of the uterus is necessary to prevent hemorrhage after delivery of the fetus and placenta. Uterine involution and removal of vena caval compression results in a sudden increase in preload to the heart, requiring continued myocardial contraction augmentation for the hours following delivery.

Postpartum hemorrhage (PPH), defined as the loss of greater than or equal to 1,000 mL, or blood loss accompanied by signs or symptoms of hypovolemia, complicate 2.9% of pregnancies.[8] The most common cause is uterine atony. Anticoagulated patients, particularly those with a mechanical valve, and patients with congenital heart disease, particularly those with a Fontan circulation, have a higher risk of PPH.[9-13] [14] Several medications used for the management of PPH can have significant hemodynamic effects. Rapid administration of oxytocin can cause vasodilation, hypotension, tachycardia, and myocardial ischemia.[15] When given as a slow three-unit bolus or an infusion of 18-to-36 units/h, these side effects are avoided and any vasodilation is treated easily with vasopressor medication.[16,17] Carboprost can cause bronchoconstriction and elevated

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pulmonary artery pressure. Methylergonovine increases smooth muscle contraction and can cause hypertension, coronary vasospasm, and myocardial ischemia.[18]

Cardiac output remains elevated for the first few days after delivery to compensate for the intravascular fluid mobilization. Blood volume decreases by about 10% in the first week after delivery. Systemic vascular resistance and HR return to normal within two weeks postpartum, and stroke volume and CO gradually decrease over several months.[19] These persistent postpartum physiologic changes make heart failure and arrythmia continued risks in the days and weeks after delivery.

Risk Stratification

Determination of cardiac risk to each patient during pregnancy is critical to provide appropriate pregnancy counseling and care for safe pregnancy and delivery. Ideally, patients with cardiac disease are evaluated prior to pregnancy by the Pregnancy Heart Team. The team communicates to patients the anticipated risks during pregnancy and may recommend medical optimization or further cardiac procedures or surgery before pregnancy to reduce risk. Risk stratification helps determine the need for referral to a higher level of care, frequency of follow-up, likely complications and management during pregnancy, additional necessary consultation, and ensuring adequate delivery planning. At times, women with cardiovascular disease are not identified and optimized prenatally and present with symptoms of decompensation during pregnancy. These patients should be treated at a tertiary care center by the Pregnancy Heart Team.

The most common adverse cardiac events that occur during pregnancy, delivery, and postpartum are heart failure and arrythmias. Common obstetric complications include premature delivery and preeclampsia. To aid the Pregnancy Heart Team in counseling women about risk of adverse events during pregnancy, multiple risk stratification models exist, including CARPREG II (Cardiac Disease in Pregnancy Study), ZAHARA, and the modified World Health Organization (mWHO) classification.[1,20-22] In prospective validation, the mWHO classification has been shown to best predict a patient's risk of a cardiac event during pregnancy.[23] The mWHO classification assigns patients into one of five risk groups based on the cardiac lesion:

- I: No detectable increased risk of maternal mortality and no or mild increased risk in morbidity (2.5%-5% cardiac event rate)
- II: Small increased risk of maternal mortality or moderate increase in morbidity (5.7-10.5 cardiac event rate)
- II-III: Intermediate increased risk of maternal mortality or moderate to severe increase in morbidity (10%-19% cardiac event rate)
- III: Significantly increased risk of maternal mortality or severe morbidity (19%-27% cardiac event rate)
- IV: Extremely high risk of maternal mortality or severe morbidity (40%-100% cardiac event rate)

Patients with mWHO class III or IV lesions should be treated at a Maternal Level 4 Care center.[24] They should be followed frequently during pregnancy (at least monthly visits, with imaging or other testing as clinically indicated).[25,26] Pregnancy in patients with mWHO class IV lesions is contraindicated, and if pregnancy occurs, termination should be discussed. The mWHO class III and IV lesions, anticipated associated complications, and major hemodynamic management goals can be found in Table 1. Patients with these lesions are most likely to require the involvement of CT anesthesiologists in their care.

Cardiac Procedures and Cardiopulmonary Bypass During Pregnancy

Cardiac surgery in pregnant women usually is deferred until postpartum unless urgent. As with other nonobstetric surgery during pregnancy, the preferred time for surgery or intervention is during the second trimester to minimize the risk of fetal loss (first trimester) or preterm labor (third trimester).[27] If surgery is required, it should be performed in a tertiary care center with a Pregnancy Heart Team and a neonatal intensive care unit.[28] Although maternal mortality with cardiac surgery during pregnancy is similar to nonpregnant patients undergoing similar surgeries, fetal mortality from cardiac surgery with cardiopulmonary bypass (CPB) is estimated to be 15%-to-50%.[29-31] If the fetus is viable, combined cesarean delivery and cardiac surgery may be preferred. Indications for cardiac surgery include severe, symptomatic valvular heart disease, Stanford Type A aortic dissection, and mechanical valve thrombosis or prosthetic valve dysfunction.[28,30,31] Maternal mortality for aortic surgery is higher during pregnancy than other types of cardiac surgery.[30] Electrophysiology ablations are reserved for refractory or hemodynamically significant arrhythmias.[1] Experience with percutaneous cardiac interventions, including transcatheter aortic valve replacement, is limited to case reports or case series.[32]

Cardiopulmonary bypass can be harmful to the fetus. Placental perfusion solely is dependent on maternal blood pressure. Maternal hypotension can lead to fetal bradycardia and acidosis. Initiation of nonpulsatile CPB flow can trigger placental vasoconstriction, decreasing fetal blood flow. Fetal bradycardia can be observed upon initiation of CPB even in the absence of hypotension.[33] The mechanism is unknown, but usually it can be corrected with high flows from the CPB circuit. Uterine contractions are the most important marker of fetal mortality in CPB cases and become more common with increasing gestational age.[32,34] They are noted frequently during CPB and most commonly occur during rewarming from hypothermia. The cooling and rewarming phases of CPB most frequently are associated with fetal bradycardia, acidosis, and death. Recommendations for CPB parameters that optimize fetal protection can be found in Table 2.

Table 3.

If cardiac surgery will be performed during pregnancy, fetal monitoring is a critical consideration. The decision to use fetal

monitoring will be made by the Pregnancy Heart Team. Elements to discuss include when to monitor, appropriate type of monitoring, and expected interventions in the event of fetal decompensation. Fetal monitoring can include Doppler measurement of fetal heart tones before and after the procedure (most appropriate for a previable fetus in a mother undergoing a minimally invasive procedure) or continuous fetal Doppler HR and uterine tocometry throughout the procedure (used for viable fetuses in mothers undergoing percutaneous or invasive surgery). If intraoperative continuous fetal monitoring is planned, a qualified provider should be designated to monitor the fetal tracing. If applicable, the patient should provide informed consent for emergency cesarean delivery and an obstetrician capable of performing an emergency cesarean delivery should be available immediately in case of fetal decompensation. Continuous intraoperative fetal monitoring also can be used to optimize maternal hemodynamics to improve fetal oxygenation in cases of fetal bradycardia.[27] Continuous fetal monitoring and uterine tocometry are recommended for 12-24 hours after surgery to monitor for uterine contractions and fetal well-being.[27]

Delivery Planning for High-Risk Cardiac Patients

In preparation for delivery, the Pregnancy Heart Team considers the individual patient's risk profile, including the most likely complications, expected interventions if a complication occurs, mode and location of delivery, maternal monitoring and access, anesthetic care, location of recovery, and safety of commonly used obstetric medications (tocolytics and uterotonics). It now is well-accepted that vaginal delivery with good neuraxial anesthesia is the preferred mode of delivery for women with cardiac disease.[1,18] Cesarean delivery is reserved for women with obstetric or fetal indications for cesarean, or women with certain high-risk lesions such as severe aortopathy, severe left side valvular stenosis, or any maternal decompensation otherwise requiring expedited delivery or intubation for cardiopulmonary indication.[35] General anesthesia for cesarean delivery is reserved for women who require emergency cesarean delivery who have not had prior neuraxial anesthesia, women who require intubation for cardiopulmonary indication, or women who are on anticoagulation with contraindications to neuraxial anesthesia. If the operating rooms in the labor and delivery suite do not have adequate resources for a complex cesarean delivery (for example, space for a TEE machine, extracorporeal membranous oxygenation [ECMO] circuit), delivery should take place in the main, CT, or hybrid operating room if peripartum ECMO is being considered. Space and resources vary per institution, and although it is best to perform vaginal deliveries in the labor and delivery suite if possible, a cesarean delivery can be performed easily in the location most suitable for the CT surgeons and perfusionists to assist.

Regardless of mode of delivery, patients with severe cardiac disease will require invasive monitoring and appropriate venous access for delivery. Invasive arterial monitoring is recommended. Central venous access is recommended if the use of vasopressors or inotropes is expected. A peripherally inserted central catheter allows for central access while minimizing impact on patient movement during delivery and infant bonding after birth and should be considered strongly unless a pulmonary artery catheter is required for delivery. Patients with a high risk of arrhythmia should have continuous electrocardiogram monitoring during labor and delivery and for 12-to-24 hours postpartum. The duration of monitoring after delivery will vary by patient condition but should remain for at least 24 hours postpartum. If the labor and delivery suite cannot support the degree of monitoring required, alternate arrangements should be made for a delivery and recovery location or nursing staff that can accommodate intensive monitoring.

Patients are at particularly high risk for right or left heart failure (depending on underlying condition) in the immediate postdelivery period, when the myocardium may not be able to augment contractility to accommodate the expected large increase in plasma volume. Peridelivery maternal cardiac output reaches 10 L per minute in healthy women to accommodate this plasma volume shift after delivery. Prophylactic inotropes started at the time of delivery should be considered strongly for these patients. Arrythmias due to inotrope use peridelivery are rare and should not preclude use of inotrope medications, although the theoretical concern should be noted. If there is concern that inotrope medications may not be sufficient (for example, in pulmonary hypertension patients with severely reduced right ventricular (RV) function, MAP pressures >50 mmHg, already on multiple pulmonary vasodilators, or patients with left ventricular ejection fraction (LVEF) $<20\%$), placement of femoral arterial and venous micropuncture catheters should be considered to facilitate emergent ECMO if needed after delivery. Micropuncture catheters do not preclude labor and vaginal delivery.

Epidural analgesia initiated via traditional epidural, dural puncture epidural, or combined spinal epidural technique is preferred for vaginal delivery, as it can attenuate the hypertension, tachycardia, and potential for arrythmias due to sympathetic activation associated with painful contractions. A T10 sensory level is adequate to blunt the discomfort of uterine contractions and cervical dilation.[36] The sympathectomy associated with epidural local anesthetic administration may not be tolerated well by patients with severe cardiovascular disease. Local anesthetic should be administered slowly, and if crystalloid preload or coload is used, it should be judiciously administered. If an assisted second stage is planned to minimize maternal expulsive efforts, adequate sacral nerve root coverage is important to ensure maternal comfort.[37] Patients who require a low HR or who are at high risk of arrhythmia should not receive epinephrine in the test dose, as inadvertent intravascular injection of epinephrine could be deleterious.[18] Fentanyl, 50 μg, is an adequate intravascular test dose.[38]

As a T4-6 sensory level is required for cesarean delivery anesthesia, the associated sympathectomy is more hemodynamically significant than that for epidural labor analgesia. Spinal anesthesia produces a rapid, reliable block for surgery but is accompanied by rapid decreases in preload and

Table 1
Risks and Hemodynamic Goals for mWHO Class III and IV Lesions[1,18]

Diagnosis	mWHO Class	Major Risks in Pregnancy/Delivery	Hemodynamic and Anesthetic Goals
Mechanical valve	III	• Valve thrombosis • Bleeding secondary to anticoagulation	• Prepare for PPH if still anticoagulated.
Fontan circulation	Without complications: III With complications: IV	• Thrombosis • Bleeding secondary to anticoagulation • Heart failure • Atrial arrhythmia • Cyanosis • Preterm delivery	• Avoid increases in PA pressure. • Avoid hypovolemia. • Maintain myocardial contractility. • Monitor for arrhythmia during and after delivery.
Systemic right ventricle	Normal/mildly decreased function: III Moderate or severely decreased function: IV	• Heart failure • Arrhythmia	• Maintain myocardial contractility. • Maintain euvolemia. • Avoid hypertension.
Unrepaired cyanotic heart disease	III	• Increased cyanosis • Heart failure • Specific risks depend on the underlying lesion	• Maintain afterload to decrease right-to-left shunt. • Maintain baseline oxygenation. • Maintain euvolemia.
Left ventricular impairment	Moderate: III Severe: IV Previous peripartum cardiomyopathy with any impairment: IV	• Heart failure	• Normal HR (avoid tachycardia) • Afterload reduction (avoid hypertension and hypotension) • Maintain normovolemia. • Maintain myocardial contractility. • May require inotropic support at the time of delivery to augment LV function
Mitral stenosis	Moderate: III Severe: IV	• Pulmonary edema (increased blood volume and HR, decreased plasma oncotic pressure) • Atrial arrhythmia (left atrial distension from increased plasma volume)	• Maintain normovolemia. • Normal to low HR to optimize LV filling • Maintain NSR.
Aortic stenosis	Severe asymptomatic: III Severe symptomatic: IV	• Pulmonary edema • Decreased coronary perfusion	• Maintain afterload to optimize coronary perfusion. • Maintain normal to low HR to optimize LV filling. • Maintain NSR.
Aortic dilation	Moderate: III Severe: IV	• Increased risk of aortic dissection or rupture • Specific risk depends on underlying condition and degree of aortic dilation	• Decrease shear stress on aorta by minimizing hemodynamic swings. • Avoid hypertension.
Pulmonary arterial hypertension	IV	• Right heart failure (increased plasma volume and fixed PVR) • Pulmonary embolism • Preterm delivery	• Optimize RV function. • Minimize PVR (consider pulmonary vasodilators). • Maintain strict euvolemia (avoid hypervolemia). • Maintain SVR to optimize coronary perfusion. • Patients may require vasopressor or inotropic agents.
Eisenmenger syndrome	IV	• Increased right-to-left shunt owing to decreased SVR • Cyanosis • Heart failure • Paradoxical embolism • Preterm delivery	• Optimize RV function. • Maintain afterload to decrease right-to-left shunt. • Minimize PVR. • Maintain strict euvolemia (avoid hypervolemia). • Avoid paradoxical embolism (use filters in IV lines). • Patients may require pulmonary vasodilators or inotropes.
Vascular Ehlers-Danlos syndrome	IV		

(*continued*)

Table 1 (*continued*)

Diagnosis	mWHO Class	Major Risks in Pregnancy/Delivery	Hemodynamic and Anesthetic Goals
Severe aortic (re)coarctation	IV	• Dissection of major arteries or veins • Uterine rupture • Aortic dissection • Aortic aneurysm rupture (if present) • Preeclampsia	• Avoid hemodynamic swings to decrease shear stress on vessels. • Avoid hypertension. • Avoid hypertension. • Blood pressure measured in right arm

Abbreviations: HR, heart rate; LV, left ventricle; NSR, normal sinus rhythm; PA, pulmonary artery; PPH, postpartum hemorrhage; PVR, pulmonary vascular resistance; R→L, right to left RV, right ventricle; SVR, systemic vascular resistance.

Table 2
Optimizing Fetal Perfusion for Cardiac Intervention During Pregnancy

General Considerations	CPB Considerations
Strongly consider fetal HR and uterine tone monitoring in discussion with obstetric team.	Maintain normothermia (hypothermia harmful to fetus).
Maintain left uterine displacement to prevent aortocaval compression.	High flow rate (>2.5 L/(min × m^2)
Maintain maternal hematocrit >28%.	Maintain perfusion pressure >70 mmHg
Maintain maternal PaO_2 >100 mmHg and O_2 saturation >92%-95%.	Consider pulsatile perfusion
Maintain maternal $PaCO_2$ >28 mmHg.	α-stat pH management
• Normal $PaCO_2$ during pregnancy is 32 mmHg	
Maintain maternal blood pressure within 20% of baseline	
Consider prophylactic tocolytic therapy (in consultation with an obstetrician)	

Abbreviations: CPB, cardiopulmonary bypass; HR, heart rate.

Table 3
Suggested Dosing for Neuraxial Anesthesia and Analgesia in Patients With Severe CV Disease

	Labor Epidural	Cesarean Delivery CSE
Local anesthetic	Intrathecal: 2-2.5 mg of bupivacaine Epidural: Dose slowly with 10 mL of 0.125% bupivacaine or 20 mL of 0.1% ropivacaine	Intrathecal: 2-7.5 mg of hyperbaric bupivacaine Epidural: 2% lidocaine with sodium bicarbonate dosed to T4-6 sensory level
Neuraxial opioid	Intrathecal: 10 μg of fentanyl Epidural: 50- to 100-μg bolus of fentanyl; can use fentanyl in epidural infusion	Intrathecal: 10-15 μg of fentanyl, 150 μg of morphine Epidural: 50-100 μg of fentanyl, 1.5-3 mg of morphine

Abbreviations: CSE, combined spinal epidural; CV, cardiovascular.

afterload, leading to significant hypotension and compensatory tachycardia.[39] Bradycardia can be observed if the local anesthetic affects the cardioaccelerator fibers. Epidural anesthesia (including dural-puncture epidural, or combined spinal epidural with intrathecal opioid with or without low-dose local anesthetic) provides a more hemodynamically stable alternative while maintaining good surgical anesthesia. Continuous phenylephrine or norepinephrine infusion and cautious fluid administration also should be considered to prevent neuraxial anesthesia-related hypotension during cesarean delivery.[40] Multimodal pain control is the gold standard after cesarean delivery and includes intrathecal morphine and scheduled acetaminophen and ketorolac followed by ibuprofen.[41-44] Transversus abdominal plane blocks also provide good postoperative pain relief for patients who have a contraindication to neuraxial anesthesia.[45-48]

Summary

Management of cardiovascular disease during pregnancy has become a significant concern. Cardiothoracic anesthesiologists may be required to participate in the care of such patients, either for cardiac intervention during pregnancy or during delivery. Collaboration with an obstetric anesthesiologist and the Pregnancy Heart Team will allow for optimal outcomes for patients.

Conflict of Interest

None.

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References

1 Regitz-Zagrosek V, Roos-Hesselink JW, Bauersachs J, et al. 2018 ESC Guidelines for the management of cardiovascular diseases during pregnancy. Eur Heart J 2018;39:3165–241.
2 Pregnancy Mortality Surveillance System. 2021.
3 Soma-Pillay P, Nelson-Piercy C, Tolppanen H, et al. Physiological changes in pregnancy. Cardiovasc J Afr 2016;27:89–94.
4 Sanghavi M, Rutherford JD. Cardiovascular physiology of pregnancy. Circulation 2014;130:1003–8.
5 Savu O, Jurcut R, Giusca S, et al. Morphological and functional adaptation of the maternal heart during pregnancy. Circ Cardiovasc Imaging 2012;5:289–97.
6 Ruys TPE, Roos-Hesselink JW, Hall R, et al. Heart failure in pregnant women with cardiac disease: Data from the ROPAC. Heart 2014;100:231–8.
7 Nishimura RA, Tajik AJ. The Valsalva maneuver and response revisited. Mayo Clin Proc 1986;61:211–7.
8 Callaghan WM, Kuklina EV, Berg CJ. Trends in postpartum hemorrhage: United States, 1994-2006. Am J Obstet Gynecol 2010;202;353.e1-6.
9 van Hagen IM, Roos-Hesselink JW, Ruys TPE, et al. Pregnancy in women with a mechanical heart valve: Clinical perspective. Circulation 2015;132:132–42.
10 Vause S, Clarke B, Tower CL, et al. Pregnancy outcomes in women with mechanical prosthetic heart valves: A prospective descriptive population based study using the United Kingdom Obstetric Surveillance System (UKOSS) data collection system. BJOG 2017;124:1411–9.
11 Wang EH, Marnoch CA, Khurana R, et al. Haemorrhagic complications of peripartum anticoagulation: A retrospective chart review. Obstet Med 2014;7:77–83.
12 Cauldwell M, Von Klemperer K, Uebing A, et al. Why is post-partum haemorrhage more common in women with congenital heart disease? Int J Cardiol 2016;218:285–90.
13 Girnius A, Zentner D, Valente AM, et al. Bleeding and thrombotic risk in pregnant women with Fontan physiology [e-pub ahead of print]. Heart 2021. https://doi.org/10.1136/heartjnl-2020-317397; Accessed May 17, 2021.
14 Committee on Practice Bulletins-Obstetrics. Practice Bulletin No. 183: Postpartum Hemorrhage. Obstet Gynecol 2017;130:e168–86.
15 Langesaeter E, Rosseland LA, Stubhaug A. Hemodynamic effects of oxytocin during cesarean delivery. Int J Gynaecol Obstet 2006;95:46–7.
16 Butwick AJ, Coleman L, Cohen SE, et al. Minimum effective bolus dose of oxytocin during elective Caesarean delivery. Br J Anaesth 2010;104:338–43.
17 Heesen M, Carvalho B, Carvalho JCA, et al. International consensus statement on the use of uterotonic agents during caesarean section. Anaesthesia 2019;74:1305–19.
18 Arendt KW, Lindley KJ. Obstetric anesthesia management of the patient with cardiac disease. Int J Obstet Anesth 2019;37:73–85.
19 Robson SC, Dunlop W, Hunter S. Haemodynamic changes during the early puerperium. Br Med J (Clin Res Ed) 1987;294:1065.
20 Siu SC, Sermer M, Colman JM, et al. Prospective multicenter study of pregnancy outcomes in women with heart disease. Circulation 2001;104:515–21.
21 Silversides CK, Grewal J, Mason J, et al. Pregnancy outcomes in women with heart disease: The CARPREG II study. J Am Coll Cardiol 2018;71:2419–30.
22 Drenthen W, Boersma E, Balci A, et al. Predictors of pregnancy complications in women with congenital heart disease. Eur Heart J 2010;31:2124–32.
23 Balci A, Sollie-Szarynska KM, van der Bijl AGL, et al. Prospective validation and assessment of cardiovascular and offspring risk models for pregnant women with congenital heart disease. Heart 2014;100:1373–81.
24 Obstetric Care Consensus No. 2: Levels of maternal care. Obstet Gynecol 2015;125:502–15.
25 Elkayam U, Goland S, Pieper PG, et al. High-risk cardiac disease in pregnancy: Part I. J Am Coll Cardiol 2016;68:396–410.
26 Elkayam U, Goland S, Pieper PG, et al. High-risk cardiac disease in pregnancy: Part II. J Am Coll Cardiol 2016;68:502–16.
27 ACOG Committee Opinion No. 775: Nonobstetric surgery during pregnancy. Obstet Gynecol 2019;133:e285–6.
28 Otto CM, Nishimura RA, Bonow RO, et al. 2020 ACC/AHA Guideline for the Management of Patients With Valvular Heart Disease: A report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation 2021;143:e72–227.
29 Chandrasekhar S, Cook CR, Collard CD. Cardiac surgery in the parturient. Anesth Analg 2009;108:777–85.
30 Shook LL, Barth WH Jr.. Cardiac surgery during pregnancy. Clin Obstet Gynecol 2020;63:429–46.
31 Liu Y, Han F, Zhuang J, et al. Cardiac operation under cardiopulmonary bypass during pregnancy. J Cardiothorac Surg 2020;15:92.
32 Cauldwell M, Johnson M, Jahangiri M, et al. Cardiac interventions and cardiac surgery and pregnancy. Int J Cardiol 2019;276:43–7.
33 Higgins MF, Pollard L, McGuinness SK, et al. Fetal heart rate monitoring in nonobstetric surgery: A systematic review of the evidence. Am J Obstet Gynecol MFM 2019;1:100048.
34 Parry AJ, Westaby S. Cardiopulmonary bypass during pregnancy. Ann Thorac Surg 1996;61:1865–9.
35 Ruys TP, Roos-Hesselink JW, Pijuan-Domenech A, et al. Is a planned caesarean section in women with cardiac disease beneficial? Heart 2015;101:530–6.
36 Hawkins JL. Epidural analgesia for labor and delivery. N Engl J Med 2010;362:1503–10.
37 Sultan P, Sultan E, Carvalho B. Regional anaesthesia for labour, operative vaginal delivery and caesarean delivery: A narrative review. Anaesthesia 2021;76(Suppl 1):136–47.
38 Guay J. The epidural test dose: A review. Anesth Analg 2006;102:921–9.
39 Langesaeter E, Dyer RA. Maternal haemodynamic changes during spinal anaesthesia for caesarean section. Curr Opin Anaesthesiol 2011;24:242–8.
40 Ngan Kee WD, Khaw KS, Ng FF. Prevention of hypotension during spinal anesthesia for cesarean delivery: An effective technique using combination phenylephrine infusion and crystalloid cohydration. Anesthesiology 2005;103:744–50.
41 Jakobi P, Solt I, Tamir A, et al. Over-the-counter oral analgesia for postcesarean pain. Am J Obstet Gynecol 2002;187:1066–9.
42 Altenau B, Crisp CC, Devaiah CG, et al. Randomized controlled trial of intravenous acetaminophen for postcesarean delivery pain control. Am J Obstet Gynecol 2017;217;362.e1-362.e6.
43 Bollag L, Lim G, Sultan P, Habib AS, et al. Society for Obstetric Anesthesia and Perinatology: Consensus Statement and Recommendations for Enhanced Recovery After Cesarean. Anesth Analg 2021;132:1362–77.
44 Practice Guidelines for Obstetric Anesthesia: An updated report by the American Society of Anesthesiologists Task Force on Obstetric Anesthesia and the Society for Obstetric Anesthesia and Perinatology. Anesthesiology 2016;124:270–300.
45 Eslamian L, Jalili Z, Jamal A, et al. Transversus abdominis plane block reduces postoperative pain intensity and analgesic consumption in elective cesarean delivery under general anesthesia. J Anesth 2012;26:334–8.
46 McDonnell JG, Curley G, Carney J, et al. The analgesic efficacy of transversus abdominis plane block after cesarean delivery: A randomized controlled trial. Anesth Analg 2008;106:186–91;table of contents.
47 Kanazi GE, Aouad MT, Abdallah FW, et al. The analgesic efficacy of subarachnoid morphine in comparison with ultrasound-guided transversus abdominis plane block after cesarean delivery: A randomized controlled trial. Anesth Analg 2010;111:475–81.
48 Costello JF, Moore AR, Wieczorek PM, et al. The transversus abdominis plane block, when used as part of a multimodal regimen inclusive of intrathecal morphine, does not improve analgesia after cesarean delivery. Reg Anesth Pain Med 2009;34:586–9.