Table 9 Predictors of neonatal events in pregnancies of women with cardiovascular disease

Predictors of neonatal events
NYHA class III/IV or cyanosis during baseline pre-natal visit
Maternal left heart obstruction
Low maternal oxygen saturation (<90%)
Multiple gestations
Use of anticoagulants
Cardiac medication before pregnancy
Mechanical valve prosthesis
Maternal cardiac event during pregnancy
Maternal decline in CO during pregnancy

Derived from the 2018 ESC Guidelines for the management of cardiovascular diseases during pregnancy.[43]
CO, cardiac output; NYHA, New York Heart Association.

In women with beta-blocker exposure, higher small for gestational age (SGA) rates and, more rarely, bradycardia have been reported, indicating the need for appropriate foetal monitoring.[12,146] Foetal ductus venosus Doppler velocity is a useful adjunct to evaluate foetal well-being and determine the time to delivery in cases of increased risk of IUGR.[147]

4.4.4. Foetal assessment of heritable primary arrhythmias

In families with primary arrhythmia, the foetus may present with arrhythmias. Therefore, the foetal heart rate should be assessed at baseline and during each pre-natal visit and compared against gestation-specific norms. In pregnancies complicated by suspected primary arrhythmia-related foetal arrhythmias, complete foetal echocardiography, typically performed at 20–22 weeks, is recommended to evaluate heart anatomy, ventricular function, and the arrhythmia mechanism.[148] Foetal magnetocardiography, if available, offers valuable insights into arrhythmia type and severity and monitors anti-arrhythmic drug therapy, as it captures all cardiac time intervals (P, QRS, T-wave) between 17 and 24 weeks of gestation. It is currently the only method to detect repolarization abnormalities, such as QT interval prolongation.[149]

4.5. Timing and mode of delivery

An individualized delivery plan should be made that covers the needs for induction of labour, labour management, delivery, and post-partum surveillance, in shared decision-making with the pregnant women. This delivery plan should be widely accessible to the patient, her partner, and relevant health professionals, and should be placed in the patient's (electronic) health record.

4.5.1. Timing of delivery

Pregnant women with CVD are more likely to have comorbidities and experience adverse events during delivery than those without CVD, and require additional monitoring and care.[150] Any maternal benefit of early term delivery (from 37 weeks 0 days to 38 weeks 6 days of gestation) should be weighed against the increased likelihood of adverse foetal outcomes.[151] Induction of labour between 39 and 40 weeks reduces the risk of emergency caesarean section by 12% and the risk of stillbirth by 50% in women without CVD. The benefit is likely to be greater for women with CVD who have higher rates of obstetric complications.[152,153] In the absence of maternal or foetal indications for early birth, induction of labour before 39 weeks should be reserved for obstetrical indications.[154]

4.5.2. Induction of labour

Mechanical methods, prostaglandin E1 analogue (misoprostol), slow-release formulation of 10 mg prostaglandin E2 (dinoprostone), oxytocin, and artificial rupture of membranes are all considered safe to induce labour.[4,155,156] High-dose (600 mg) misoprostol does not affect cardiac parameters in women without heart disease, although there remains a theoretical risk of coronary vasospasm and arrhythmias.[155] Dinoprostone may cause profound hypotension, but only when injected blindly into the myometrium, and this route of administration should be avoided.[157] The use of an additional 2 IU of oxytocin for the management of the third stage in women with CVD has no cardiac consequences and is associated with significantly lower blood loss.[158] In women at high risk (mWHO 2.0 classes III–IV), oxytocin is generally considered as a first-line uterotonic, misoprostol and carboprost are second line (see Supplementary data online, *Table S3*).[159]

Mechanical methods such as a cervical ripening balloon might be preferable in women where a drop in systemic vascular resistance would be detrimental.[160] If membranes are ruptured, augmentation of labour should be immediate to reduce the risk of infection and should be undertaken with oxytocin to minimize the number of vaginal examinations.[4]

4.5.3. Vaginal or caesarean delivery

Vaginal delivery is associated with less blood loss and lower risk of infections and venous thromboembolism and should be advised for most women.[161] Planned caesarean section does not confer any advantage over planned vaginal delivery in terms of maternal outcomes and may be associated with adverse foetal outcomes.[162,163]

Caesarean section is the preferred mode of delivery for obstetric indications and for women presenting in labour who use or have used vitamin K antagonist (VKA) within the past 2 weeks, with high-risk aortopathy (mWHO 2.0 class III), with hypertrophic cardiomyopathy (HCM) and severe left ventricle outflow tract obstruction, or in acute intractable HF.[43]

4.5.4. Haemodynamic monitoring during delivery

Pulse oximetry, blood pressure monitoring, and continuous ECG monitoring may help detect early signs of decompensation, arrhythmias, and ischaemia in women with significant CVD and identify those in whom delivery should be expedited.[164] Arterial lines should be reserved for those women who have haemodynamic instability or are at risk of it. A right-heart catheter is of uncertain benefit, is associated with complications, and should be avoided in most cases. Minimally invasive CO monitoring is preferable, where possible.[110]

4.5.5. Anaesthesia/analgesia

Analgesia is crucial for labour in pregnant women with CVD to reduce physical stress. Neuraxial methods are very effective analgesic blocks. The onset of conventional epidural analgesia is relatively slow (±15 min) and allows for careful titration of a local anaesthetic–opioid mix.[165] Spinal analgesia is suitable for women with high-risk CVD, where a faster onset of sympathetic block is desirable.[166] Combined spinal–epidural analgesia typically has a faster onset time (±5 min). However, adverse effects such as hypotension and foetal heart rate abnormalities occur more quickly and are more pronounced. Different techniques for administering low-concentration, high-volume local anaesthetic–opioid regimens allow maintenance of epidural analgesia through the epidural