***Section 8*. Recommendations for aortopathies, cardiac surgery, and pregnancy**					
Pregnancy is not recommended in patients with vascular Ehlers–Danlos syndrome.	III	C	It is recommended that women with vascular Ehlers–Danlos syndrome wishing to become pregnant are counselled regarding the very high risk of pregnancy-related adverse events by a multidisciplinary team, considering family history, genetic variant, and previous vascular events.	I	C
Beta-blocker therapy throughout pregnancy should be considered in women with Marfan syndrome and other heritable thoracic aortic diseases.	IIa	C	Beta-blocker therapy throughout pregnancy and in the post-partum period is recommended in women with MFS and other HTADs.	I	C
***Section 9*. Recommendations for congenital heart disease and pregnancy**					
Patients with a systemic right ventricle (Mustard/Senning or congenitally corrected TGA), in NYHA class III/IV, systemic ventricular dysfunction (EF <40%), or severe TR should be advised against pregnancy.	IIa	C	It is recommended that women with a systemic RV (Mustard/Senning or congenitally corrected TGA), in NYHA class III/IV, systemic ventricular dysfunction (EF <40%), or severe TR wishing to become pregnant are counselled by the Pregnancy Heart Team regarding the high risk of pregnancy-related adverse events.	I	C
***Section 12*. Recommendations for acquired heart disease and pregnancy**					
An invasive management strategy should be considered for NSTE ACS with high-risk criteria.	IIa	C	It is recommended to manage pregnant women with ACS in the same way as non-pregnant women, including diagnostic investigations and interventions.	I	C
Catheter ablation with electro-anatomical systems should be considered in experienced centres in cases of drug-refractory and poorly tolerated SVT.	IIa	C	Catheter ablation may be considered in pregnant women with recurrent, long symptomatic SVT or with contraindications to pharmacological therapies.	IIb	C
Balloon aortic valvuloplasty should be considered during pregnancy in patients with severe aortic stenosis and severe symptoms.	IIa	C	In very selected symptomatic pregnant women with severe aortic stenosis not responding to medical therapy, non-surgical options such as balloon valvuloplasty or TAVI may be considered.	IIb	C
A bioprosthesis should be considered in young women contemplating pregnancy.	IIa	C	A bioprosthetic valve is recommended (over a mechanical valve) in young women contemplating pregnancy requiring a valve prosthesis.	I	B
During the second and third trimesters, LMWH with anti-factor Xa level monitoring and dose adjustment (see separate recommendations) may be considered in women who need a high dose of VKA after patient information and consent.	IIb	C	During the second and third trimesters until the 36th week, continuing VKAs should be considered in women with prosthetic heart valves at higher risk of thrombosis.	IIa	C

ACS, acute coronary syndrome; CMP, cardiomyopathy; EF, ejection fraction; HCM, hypertrophic cardiomyopathy; HTAD, heritable thoracic aortic disease; LMWH, low-molecular-weight heparin; MFS, Marfan syndrome; NSTE ACS, non-ST-elevation myocardial infarction; NYHA, New York Heart Association; RV, right ventricle; SVT, supraventricular tachycardia; TAVI, transcatheter aortic valve implantation; TGA, transposition of the great arteries; TR, tricuspid regurgitation; VKA, vitamin K antagonist.
[a]Class of recommendation.
[b]Level of evidence.

3. General considerations

3.1. Epidemiology

Within high-income countries the number of pregnancies and deliveries in women with acquired, congenital, or inherited CVD is growing significantly.[19–21] This trend originates from several factors: higher maternal age at first pregnancy, a growing number of women with congenital heart disease reaching childbearing age, and a rising prevalence of cardiovascular comorbidities.[19–21] Globally, up to 4% of pregnancies are complicated by CVD, rising to 10% when including hypertensive disorders.[22–25] Maternal CVD is now the leading cause of non-obstetric mortality in pregnant women[24,26], accounting for 33% of pregnancy-related deaths worldwide.[25,27,28] In women with pre-existing CVD, up to 16% of pregnancies are complicated by CVD.[29–32] Notably, 68% of pregnancy-related deaths caused by CVD are preventable.[33] Women with CVD during pregnancy face higher risk of cardiac events later in life, making secondary prevention crucial.[25] Adverse neonatal outcomes occur in ~25% of these pregnancies in women with CVD[34,35] with high rates of obstetric complications (17%) and maternal mortality/morbidity (11%).[34,35] Of note, recent research indicates that pre-existing CVD in the mother is associated with an increased risk of CVD in her children, and this association is unlikely to be explained by unmeasured familial or genetic factors.[36]

3.2. Physiology of pregnancy

Pregnancy induces physiological changes in the cardiovascular system to meet the increased metabolic needs of the mother and foetus (*Figure 1*). These changes occur from the early stages of pregnancy onward. Starting at 6 weeks of gestation, stroke volume and cardiac output (CO) increase