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An overview of key changes in the cardiovascular system during uncomplicated pregnancy, birth and the postnatal period

Introduction

The physiological changes associated with the pregnancy and peripartum period poses a physiological stress test to women, and can be particularly challenging to those with previously undiagnosed or known cardiovascular conditions (Sliwa and Böhm 2014). Between 1 and 4% of pregnancies are complicated by maternal heart disease, accounting for up to 15% of maternal deaths (Alkema, Chou et al. 2016, Knight, Kenyon et al. 2017, Regitz-Zagrosek, Roos-Hesselink et al. 2018). Whilst leading causes of mortality such as haemorrhage and infection are declining, mortality due to maternal heart disease is increasing (Khan, Wojdyla et al. 2006, Knight, Kenyon et al. 2017). Knowledge about the morphological and functional changes in normal pregnancy is therefore essential for cardiac nurses in order to recognise and provide care for this growing cohort of patients.

During pregnancy the cardiovascular system undergoes significant changes. As early as conception, the cardiovascular system begins to undergo radical remodelling to meet the demands of the growing foetus. Key increases in cardiac output, vascular resistance and circulating blood volume are accompanied by adjustments to the position and size of the heart. Changes occur due to hormonal and physical influences. This editorial gives an overview of key changes in the cardio-vascular system during uncomplicated pregnancy, birth and the postnatal period, whilst providing an overview of some of the resulting transient effects.

Increase in plasma volume

Circulating plasma volume increases by 30-50%, beginning at 6 to 10 weeks (1st trimester[1]) and reaching its maximum at 28 to 32 weeks (Blackburn, 2018). Augmented plasma volume occurs via activation of the renin-angiotensin-aldosterone system leading to sodium and water retention. Circulating blood flow is required to meet the demands of the uterus, placenta and growing foetus, and is also increased to the breasts, kidneys, lungs and skin, accommodated by increased vasculature. The increase in blood is required to support proliferation of breast tissue to accommodate lactation, to increase levels of oxygen to the lungs and to ensure optimal demands of pregnancy on kidney function.

Pregnancy related 'hypervolemia' leads to (temporary) haemodilution due to the increase in plasma volume not being initially matched by an increase in red blood cells. Maximisation of red cells usually occurs in the second trimester peaking in the third, whilst increases in plasma volume begin earlier

[1] First trimester = 0-12 weeks, Second trimester = 13-26 weeks, Third trimester = 27 weeks – end of pregnancy

during the first trimester. An ensuing physiological anaemia may ensue, although this is usually corrected once red cells reach an equilibrium peaking at 30-34 weeks gestation (Blackburn, 2018).

Increase in heart rate, stroke volume and cardiac output

Alongside the increase in blood volume there is a corresponding increase in maternal heart rate up to 15-20% above baseline (Morton 2021), which is further increased during labour and birth. This can lead to palpitations, cardiac arrythmias and a physiological tachycardia although it is important to recognise that the latter may be pathological (Coard and Frise 2021). Stroke volume increases progressively peaking to approximately 30% of non-pregnant values (Tan and Tan, 2013). This is due to an increase in end diastolic flow and with the increase in heart rate contributes to an overall increase in cardiac output. Cardiac output is increased by as much as 50% during pregnancy in order to sustain the blood pressure, commencing early in pregnancy and maintaining this increase throughout. Like heart rate, cardiac output is increased during labour and birth (Sanghavi and Rutherford 2014).

Decreased vascular resistance and vasodilation

Decreased vascular resistance and vasodilation take place to accommodate the increase in circulating blood volume, a result of the effects of the hormones progesterone and relaxin on vessel walls. This 'high flow low resistance' state impacts maternal blood pressure, which usually lowers during the first two trimesters. In some cases, this can lead to fainting, dizziness and postural hypotension. Blood pressure returns to normal values in the third trimester. When blood pressure does not lower or is increased this can be indicative of existing hypertensive disorders or pre-eclampsia (Poon et al 2023). Relaxation of vessel walls can also lead to varicosities or exacerbation of existing ones. Increase in venous pressure as a result of relaxed vessels walls results in incomplete vessel valve closure and resultant backflow and 'pooling'.

Changes in size and position of heart

The heart is enlarged in size due to left ventricular wall thickness, increase filling and physiological cardiac hypertrophy. This meets increased maternal and foetal metabolic demands and increase in circulating blood volume. In addition, the heart moves upwards and rotated forwards (dextro-rotation). The apex beat is now directed forwards to the anterior chest well and the ECG is altered. Common changes include non-specific T-wave and ST-segment changes with shortened PR and QT intervals (Anathakrisnan et al, 2020) however practitioners should be cautious in assuming altered physiology when interpreting the ECG's.

Compression of the inferior vena cava and reduction in venous return

When in the recumbent position supine hypotension can occur, due to compression of the gravid uterus on the inferior vena cava (Humphries et al 2020). This is most marked from the second trimester onwards when the uterus begins to become more pronounced. Left tilting can mitigate this. The gravid uterus also impedes venous return, which is aggravated by progesterone related vasodilation. This leads to oedema, particularly in the lower extremities.

Hypercoagulability

Fibrinogen and clotting factors increase during and particularly towards the end of pregnancy, in readiness for birth and the third stage of labour, where blood loss is expected and can in some cases be prolific. This leads to resultant hypercoagulability, which can increase the risk of thromboembolic disorders (Devis and Knuttien 2017). This risk is exacerbated by reduced mobility particularly

towards the end of pregnancy, vessel wall damage during labour and birth and impaired venous return due to vasodilation and the gravid uterus. Hypercoagulability remains up until around 6 weeks postpartum, with women at risk of deep vein thrombosis and pulmonary embolism until then.

Reversion to the pre-pregnancy state

The cardio-vascular system reverts to its pre-pregnancy state by 6 weeks postpartum, with some changes being immediate and some by 6 weeks. It should be noted however that some studies have found persistent changes up to one year postpartum in arterial stiffness and subsequent mean arterial pressure (Morris et al 2015).

Conclusion

Pregnancy presents complex demands on the cardio-vascular system which, in uncomplicated pregnancies, are met with a series of intricately linked modifications to accommodate them. These modifications, although necessary to ensure a healthy pregnancy, can also create an environment for various pathological outcomes to occur. Changes to the cardio-vascular system tend to be short-lived however, with the majority reverting to a pre-pregnancy state by 6 weeks postpartum.

Table 1 Changes in cardiovascular system in uncomplicated pregnancy, birth and postanal period

Change	When	Cause	Impact
Increased heart rate (15-25 bpm)	First trimester, peaking in 3rd trimester, Further increase during labour	Increase in plasma volume	Physiological tachycardia Palpitations Cardiac arrythmias Increased cardiac output
Increased stroke volume (10%)	From first to second trimester, decreasing in third trimester	Increase in end diastolic volume	Increased cardiac output
Increased cardiac output (30-50%)	From first trimester peaking during second/third trimester Further increase during labour (20%)	Increased heart rate and stroke volume	Maintains blood pressure
Increased plasma volume (30-50%)	Early on in first trimester and throughout pregnancy	Increased activation of the renin-angiotensin-aldosterone system	Increased blood flow to: uterus, placenta, breasts, skin, kidneys, lungs Haemodilution 'Physiological anaemia' until red blood cell production is maximised (2nd to 3rd trimester)
Decreased vascular resistance	From 5 weeks gestation	Progesterone	Fall in blood pressure until approx. 26-28 weeks gestation Fainting, dizziness, postural hypotension Varicosities
Peripheral vasodilation	From first trimester throughout pregnancy	Progesterone	Increase in temperature Feeling hot
Increased red blood cell production (40%)	From first trimester through to second/third trimester	Elevated erythropoietin levels	To match increase in plasma volume and reduce anaemia

Hypercoagulability	Gradual during pregnancy, peaks during third trimester	Increase in fibrinogen and clotting factors	Risk of DVT, Pulmonary embolism Helps reduces bleeding during birth and at placental site during third stage and postnatal period
Compression of inferior vena cava	Second to third trimester	Pressure on inferior vena cava from gravid uterus	Supine hypotension
Reduction in venous return	From first trimester more marked in third trimester	Vasodilation due to progesterone Gravid uterus	Oedema
Increase in size of heart (up to 12%)	From first trimester	Increased filling Oestrogen causes cardiac cell hypertrophy	Accommodation of increased plasma volume and venous return
Heart displaced upwards and forwards	From second trimester onwards	Displacement of diaphragm due to gravid uterus	Apex beat at anterior chest wall Changes to ECG

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