

Arrhythmias in pregnancy



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Gender differences in arrhythmias



Women have:

- **Higher** intrinsic heart rates and **shorter** SNRT than men
- **Longer** QTc intervals and **more frequent** long QT syndrome
- A **higher** incidence of drug induced TdP, **but fewer** SCD episodes
- A 2:1 female **predominance** of AV nodal reentrant tachycardia

The mechanism of these gender differences is not entirely clear, but *sex hormones* certainly play a role

Pregnancy may be pro-arrhythmic



- **Pregnancy alters the hormonal state:**
 - ↑ estrogen
 - ↑ β -human chorionic gonadotropinmay have an effect on the expression of cardiac ion channels
- **Haemodynamic changes:**
 - ↑ in the circulating volume that doubles the cardiac output results in myocardial stretch and an increase in cardiac end diastolic volumes
- **Pregnancy increases sympathetic tone:**
 - high plasma catecholamine concentrations and adrenergic receptor sensitivity

All of these changes in expectant women can create an environment conducive to arrhythmogenesis

Arrhythmia management during pregnancy



- **Fortunately**, most arrhythmias in this population are **benign** and therapy mainly takes the form of reassurance
- However, there are cases in which **drug administration** is required and fortunately few cases in which **invasive electrophysiologic** procedures are necessitated
- The effects of **drug administration** and **radiation exposure** can lead to severe problems for the developing foetus:
 - the first half of pregnancy: **congenital malformations and mental retardation**
 - the second half of pregnancy: increases the **risk of childhood malignancies**

ECG changes in pregnancy



- An increase in resting heart rate about 10bpm
 - this results in decreased PR, QRS and QT intervals, **but** no change in the amplitude of the P wave, QRS complex and T wave
- Shift in the electrical axis, more commonly leftward
 - due to rotation of the heart secondary to the enlargement of the gravid uterus
- Premature atrial and ventricular depolarizations are common

Palpitations



- A **frequent** reason for referral to an electrophysiologist
- A source of **significant anxiety** for the pregnant woman
- A study examined Holter data of **104** pregnant women with palpitations:
 - 76% had no associated arrhythmias
 - 24% arrhythmias were found, but most were benign
- Most cases, were attributed to **sinus tachycardia**

Palpitations



Non-invasive work-up

- **Caution** is advised with *exercise stress testing*, because of foetal bradycardia at maximal exercise
 - a low-level protocol with foetal monitoring is advised
- When benign arrhythmias are found, patients need:
 - reassurance
 - should avoid stimulants, such as caffeine, smoking and alcohol

Supraventricular tachycardia



- Conflicting data regarding the **incidence** of SVT during pregnancy:
 - *Tawan et al, Am J Card 1993* reported:
 - a 34% increase in the risk of new onset SVT
 - a 29% exacerbation of SVT episodes during pregnancy
 - **On the other hand**, *Lee et al, Am J Card 1995* found:
 - a much lower prevalence of SVT
- Women with **accessory pathways** may experience a **more pro-arrhythmic** response from pregnancy than those with AVNRT

Management of SVT



- **Vagal maneuvers** should be tried first
- If the tachycardia persists, **intravenous adenosine** is the **drug of choice**
- **Safety** of adenosine use :
 - During the first trimester insufficient data
 - In the second and third trimesters is safe and effective
- Foetal heart **monitor** during administration of the drug to detect possible foetal bradycardia

Management of SVT



- If adenosine fails, **intravenous propranolol or metoprolol**
- **Verapamil** should be **avoided** owing to possible prolonged hypotension
- If SVT cannot be chemically converted, or if hemodynamically unstable, **cardioversion** should be performed
- **Chronic medical therapy** should be **withheld** unless episodes of tachycardia become frequent, severe and hemodynamically significant

Management of SVT



- Women with **known SVT** should be advised to undergo a **curative catheter ablation** prior to planned pregnancy due to the fact that:
 - SVT episodes can become more frequent
 - all drugs should be avoided during this time
- If ablation is absolutely necessitated during pregnancy it should be performed in the **second trimester**
- A **radiation shield** over the abdomen and use of **pulsed fluoroscopy** could help reduce radiation risk for the foetus



Atrial Fibrillation and Atrial Flutter

- **Rarely** seen during pregnancy
- **Commonly** occurs in pts with congenital heart disease, rheumatic valvular disease and hyperthyroidism

Management of AF/Flutter

- Ventricular rate control with **β -blocking agents or digoxin**
- **Electrical cardioversion** should be considered within 48 h of onset of AF to **avoid the need for anticoagulation**
 - **Coumadin** is teratogenic and contraindicated during the first trimester of pregnancy
 - **Heparin** is not absorbed by the placenta and is considered the drug of choice

Long QT Syndrome



A difficult management problem in the past

- Retrospective data revealed that there is a **significant increase** in the risk for cardiac events in the **postpartum period** (the 40-week period post delivery), **but not during pregnancy**

Cardiac events	Pre-pregnancy, %	Pregnancy, %	Post-partum, %
Multiple	0.9	4.5	9.1*
New-onset	0	1.8	9.0†

- This **delay** might be related to the drop in heart rate after delivery and the increased stress of caring for an infant
- During pregnancy, the heart rate normally increases and might have a **protective effect**
- **β -blocker therapy** should be continued throughout pregnancy and postpartum

Rashba et al, Circulation 1998

Ventricular tachycardia



- The most **common** VT during pregnancy might be **catecholamine-sensitive or benign idiopathic right ventricular outflow tract tachycardia**
- **Rare** cases of life-threatening ventricular tachycardia associated with **structural heart disease**:
 - Peripartum cardiomyopathy
 - Hypertrophic cardiomyopathy
 - Coronary artery disease
 - Rheumatic heart disease
 - Congenital heart disease
 - Right ventricular dysplasia
 - Mitral valve prolapse
 - Long QT syndrome
- The general approach is to determine if the woman has **structural heart disease or long QT syndrome**, by doing an **ECG & echo**

Management of VT



- A morphology that is **monomorphic, LBBB, and inferior axis** would be consistent with the diagnosis of **RVOT tachycardia**
 - β -blocker is the drug of choice
- The finding of **structural heart disease** would put the mother at increased risk of **SCD**
 - Antiarrhythmic drug therapy and possibly an ICD might be required
- **Electrical** cardioversion for **unstable** and **lidocaine** for **more stable** tachycardia

Implantable devices



- The presence of an ICD **should not deter** a woman from considering pregnancy, unless her underlying structural cardiac disease is a contraindication
- If a woman with an ICD delivers **vaginally**, the device's shock therapy **should be left activated**
- The shock therapy **should be turned off during a Cesarean section** to avoid an inappropriate shock secondary to electrocautery
- If it becomes necessary to implant an ICD or PM, the **smallest sized** generator should be placed **subpectorally**
 - leads with echoguidance or with a radiation shield

Antiarrhythmic drug risk



The FDA has developed a drug classification system to rate the level of foetal risk

<i>Class A</i>	<i>controlled studies show no risk</i>
<i>Class B</i>	<i>no evidence of risk in pregnant women, but either animal studies do show risk, or no adequate human studies have been conducted</i>
<i>Class C</i>	<i>studies in pregnant women are lacking, and animal studies are positive for fetal risk, or lacking</i>
<i>Class D</i>	<i>positive evidence of risk – can be used if potential benefit outweighs risk</i>
<i>Class X</i>	<i>contraindicated – do not use, regardless of potential benefit</i>

Antiarrhythmic drug risk



No anti-arrhythmic drugs are rated class A because **no** controlled trials involving pregnant women have been conducted

Drug	FDA risk category	Reported side effects
Quinidine	C	Fetal thrombocytopenia, premature labor at toxic doses
Procainamide	C	None
Disopyramide	C	Uterine contraction
Lidocaine	B	Fetal central nervous system depression and bradycardia at birth
Mexiletine	C	Fetal bradycardia, low birth weight, low Apgar score, and hypoglycemia
Flecainide	C	None (data limited)
Propafenone	C	None (data limited)
Propranolol	C	Fetal growth retardation, hypoglycemia, bradycardia, birth apnea
Sotalol	B	None (data limited); expect effects similar to beta blockers
Amiodarone	D	Congenital hypothyroidism, growth retardation, premature birth, bradycardia, large fontanelles
Verapamil	C	Bradycardia, heart block, hypotension
Digoxin	C	Low birth weight
Adenosine	C	None

Prophylactic drug therapy for SVT



The ACC/AHA/ESC Guidelines recommend:

- First-line drugs **digoxin** or **β-blocker**
 - Digoxin is relatively safe with careful monitoring
 - Propranolol or metoprolol can be used, but should be avoided during the first trimester
 - Atenolol should not be used (class D)
- If these drugs fail, **sotalol** (class B), or **flecainide** if the patient has no structural heart disease

Prophylactic drug therapy for VT



The ACC/AHA/ESC Guidelines recommend:

- **Sotalol** should be considered if **β-blocker** therapy is not adequate
- No data to the use of **dofetilide** in the pregnant patient
- **Amiodarone** is a risk **class D** drug, and the only anti-arrhythmic drug with known teratogenic risk
 - **only** when there are no other therapy options

If drug therapy cannot be avoided...



- **Frequent monitoring** of the patient's **ECG and drug levels** to reduce the risk of toxicity
 - plasma concentration can be altered due to:
 - increased renal blood flow
 - increases in hepatic metabolism
 - changes in gastrointestinal absorption
- **Teratogenic risk** is greatest in the **first 8 weeks** of gestation
 - begin AADs as late in pregnancy as possible
 - use the lowest effective drug dose
- A **low dose drug combination** may be **preferable** to a higher dose of a single drug, because side effects can be dose-related

The risk/benefit ratio



Must be considered in the decision regarding AAD therapy:

- **Benign** arrhythmias, even when symptomatic, **should not be treated**
- Treatment **should not be denied** in **malignant** arrhythmias
- When possible, **invasive procedures** involving fluoroscopy **should be avoided** until after delivery
- **Known** arrhythmias **should be urged** to undergo curative catheter ablation prior to pregnancy

Conclusions



- During pregnancy, significant changes occur in the **hormonal and hemodynamic** state of women that make arrhythmias **more likely** to occur
- **Palpitations** are frequently reported, but are usually found to be associated with sinus tachycardia
- The incidence of paroxysmal **supraventricular** tachycardia is increased during pregnancy, whereas **atrial fibrillation** and **ventricular** tachycardia are rare

Conclusions



- Women with **long QT** syndrome experience significantly more cardiac events in the **postpartum** period, making β -blocker therapy most important during this time
- **Acute** treatment of arrhythmias for pregnant women is much the same as that for non-pregnant patients
- **Chronic** drug therapy during pregnancy should be reserved **only** for the frequent, hemodynamically significant arrhythmias