Ventricular Arrhythmias – Approach to Management in Pregnancy

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Arrhythmia Symptoms in Pregnancy

• Only 10% of “palpitations” associated with arrhythmia.
• Premature atrial or ventricular beats….can be seen in more than 50% of pregnant women.
  • Poor symptom-rhythm correlation

<table>
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<th>Table 2</th>
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<tbody>
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<td>Pregnant women with no heart disease$^a$</td>
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<tr>
<td>PACs 56%</td>
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<td>&gt;100/hour 7%</td>
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<tr>
<td>PVCs 59%</td>
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<td>&gt;50/hour 22%</td>
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Wide Complex Rhythms

Aberrancy

Pre-excitation

Ventricular arrhythmia
PVCs in Structurally Normal Hearts

Non-pregnant population:

- In “normal hearts”, 4% individuals will have more than 100 PVCs/day.

In populations with normal 2DE there is no increase in mortality or cardiovascular outcomes.

- HOWEVER...
- A high burden of ectopy (>10-20%, or >10 000/d) may result in LV dysfunction.
  - Occurs in as many as ~38%
  - Greater risk if non-outflow tract origin, or baseline broad QRS complex.

Ventricular Arrhythmias in Pregnancy

Prevalence ~3.7%

- Isolated PVCs most common.
- 21.7% have previously documented PVCs
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Rule out structural heart disease

- ECG (hypertrophy, pathologic T wave inversion, conduction delay)
- Echocardiogram

Ventricular Arrhythmias in Pregnancy

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Rule out structural heart disease
- Echocardiogram
- ECG (hypertrophy, pathologic T wave inversion, conduction delay)

Screen for high risk features
- Heavy burden of ectopy (>10-20%, or >10 000/d)
- Family history
- Syncope/hemodynamic compromise related to arrhythmia
- Sustained VT, Polymorphic PVCs/VT
Idiopathic VT

• Resting ECG is normal between VT episodes
  – There may be temporary repolarization abnormalities immediately post-termination.

• Imaging is normal between VT episodes.
  – Echocardiogram, MRI
  – There may be temporary wall motion abnormalities immediately post-termination.

• Exercise stress test is normal.

• Coronary angiogram (if indicated) is normal.
The Idiopathic VTs

- RV/LV Outflow tract VT
- Paroxysmal sustained VT
- Fascicular VT
- Papillary Muscle VT
Repetitive Monomorphric VT

- Ventricular outflow tract origin
  - LBBB with inferior axis.

- Presentation
  - PVCs, NSVT, VT
    - Adrenergically mediated
      - During or immediately post-exertion
      - Emotional stress

- Mechanism
  - Intracellular calcium overload resulting in delayed afterdepolarizations that lead to triggered activity.

J Am Coll Cardiol 2015;66:1714–28
Heart Rhythm 2009;6:1507–1511
The prognosis is favourable, with two uncommon exceptions:

- Tachycardia induced cardiomyopathy
- Polymorphic ventricular tachycardia / VF
Tachycardia Induced cardiomyopathy

Threshold burden:
- ~10 000 PVCs/d

Reported incidence:
- With significant PVC burden 4-39%

Also be seen in atrial tachycardia, atrial fibrillation
- Never in sinus tachycardia, POTS

Markers of risk:
- Asymptomatic
- Interpolated PVCs (no compensatory pause)
- Retrograde atrial activation
- Non-outflow tract PVCs
- PVC QRS duration >153 ms
- Male sex, higher BMI

References:
- Curr Probl Cardiol 2015;40:379–422
“Malignant” Variant of Repetitive Monomorphic VT

Rare

- True frequency not known

High risk subsets:

- History of syncope/presyncope
- Family history of sudden unexplained death
- Nonsustained or polymorphic VT on Holter monitoring
- Multiple PVC morphologies
- Extremely frequent PVCs
  - >20,000/d
  - Relatively short coupling interval of PVCs
    - 340 ms ± 30 ms

Mandates more aggressive therapy

- Catheter ablation
- Beta blockade/Calcium channel blockade, IC
- Close monitoring of LV function

J Cardiovasc Electrophysiol. 2005 Aug;16(8):912-6
J Am Coll Cardiol 2005;46:1288–94
J Am Coll Cardiol 2009;54:522–8
Heart Rhythm 2009;6:1507–11
“Malignant” Variant of Repetitive Monomorphic VT
Acute Management:

- Procainamide IV – First choice for acute termination in hemodynamically stable VT
- Cardioversion if hemodynamically unstable

Chronic Management

- When to treat?
  - Symptomatic patient
  - If PVC burden <10% or <10,000/d beta blockade indicated for control of symptoms ONLY if there are no high risk features.

- High risk features
  - Heavy burden of ectopy (>10-20%, or >10,000/d)
  - Possible family history of cardiomyopathy or arrhythmia syndrome
  - Syncope/hemodynamic compromise related to arrhythmia
  - Sustained VT
  - Polymorphic VT
  - Repetitive Monomorphic VT
Emergency defibrillation in Pregnancy

- No different from standard ACLS.
- Same pad placement
- Same energy delivery (maximum)
- Never delay therapy to remove fetal scalp monitor
Repetitive Monomorphic VT

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Repetitive Monomorphic VT

Chronic Management – How to treat?

- Oral beta blockers or calcium channel blocker (verapamil) can be effective in up to ~67%
  - Success rate may be higher in pregnancy
- Class Ic antiarrhythmic - Flecainide
- RF ablation of the PVC/VT focus
  - Consider post-partum if PVCs continue
  - Long term success rate >80%
  - May be preferred over antiarrhythmic agents due to adverse effect profile.
- Indicated if medical therapy fails or is poorly tolerated, or if there is reduced LVEF
Tachycardia Induced cardiomyopathy

Mandates aggressive therapy:

- Goal is 80% reduction in PVCs, and <5000 PVCs per day.
- Success 70-90%
- Improvement in LVEF, LV dimensions, mitral regurgitation, functional class.
  - Can take 3-4 months for improvement.
- Definitive management is catheter ablation of PVCs
- Medical therapy is second-line therapy, with lower chance of success.

Long term outcome:

- Risk of sudden death remains above baseline – reflecting vulnerable myocardial substrate?
  - Risk greater in those who present with very low EF (<20%)
- Overall prognosis likely excellent but arrhythmia recurrence can result in very rapid deterioration of LV function.
  - Incomplete recovery or presence of LGE on MRI may suggest higher risk subset.
- Importance of long term HF therapy

J Am Coll Cardiol 2015;66:1714–28
- Asymptomatic patient referred for extrasystoles noted on physical exam.
- No history of syncope, family history negative
- Holter monitor 6% PVCs, monomorphic, no runs
- Echocardiogram normal
- ECG shows classic RVOT PVCs with no abnormality of QRS complex

Holter monitor for ~48 hours each trimester
No beta blocker indicated unless PVC burden exceeds 10-20% or 10 000/day
- 29F referred for PVCs noted on routine physical exam
- Unaware of extrasystoles (asymptomatic)
- Holter monitor: only 2% PVCs, no VT
1. On further questioning, a maternal cousin died suddenly at age 20, of unknown causes
2. Anterior T wave inversion Leads V1-
3. Frequent, non-uniform PVCs
4. Echocardiogram revealed RV dilatation with focal free wall dyskinesis
5. Exercise stress testing revealed non-sustained VT from RV body.

Meets criteria for ARVC.
Beta blocker indicated for arrhythmia prophylaxis.
Genetic testing
Family screening
Fascicular VT

• Idiopathic VT originating in the left fascicles:
  – Left posterior fascicle most common site (>90%)
  – Left anterior fascicular VT
  – Left upper septal fascicular VT

• Classic features:
  – Induction with atrial pacing
  – ECG signature:
    • RBBB with LAD configuration most common
  – No structural heart disease
  – Verapamil sensitive
  – Benign
Papillary Muscle VT

• Idiopathic focal ventricular arrhythmia.
  – Similar arrhythmias may arise from the mitral annulus, aortomitral continuity, elsewhere.
• No underlying myocardial abnormality.
• May present as PVCs/NSVT or sustained VT.
• Arrhythmia typically catecholamine mediated.
• Likely low risk, with no reports of syncope/SCD.
• Tachycardia mediated cardiomyopathy
  – if PVC burden >16-20%
• Management:
  – Beta blockers
  – Calcium channel blockers
  – Ablation
Management of Labour and Delivery

• Do not interrupt beta blockade if initiated.
• Unassisted vaginal delivery not contraindicated.
• Pain management as needed
• Telemetry?
  – No clear guidelines.
  – Telemetry indicated if there is history of NSVT or VT.
  – Routine hospital monitoring may be acceptable if stable, low (<10%) burden of isolated PVCs.