NOTES



NOTES PREMATURE CONTRACTION

GENERALLY, WHAT IS IT?

PATHOLOGY & CAUSES

- Depolarizing potential from anywhere in heart other than sinoatrial (SA) node → contraction earlier than normal in cardiac cycle
- Triggered activity
 - Cells triggered by preceding action potential after repolarization
 - Cause: reperfusion therapy after myocardial infarction/digoxin toxicity
- Ectopic focus
 - Cells irritated by electrolyte imbalances, drugs, ischemic damage → increased sympathetic activity → enhanced automaticity → early depolarization
- Reentrant loop
 - Tissue unable to depolarize (e.g. scar tissue, amyloid) → no signal conduction
 → depolarizing wave obstructed → depolarizing wave circles tissue → abnormal electrical circuit

CAUSES

- Often idiopathic
- Electrolyte imbalances (hypokalemia, hypercalcemia, hypomagnesemia)
- Recreational/prescription drugs (methamphetamines, cocaine, digoxin intoxication)

- Alcohol use
- Heart dilation: cardiomyopathies, cor pulmonale
- Heart scarring: after myocardial infarction, myocarditis

COMPLICATIONS

Rarely atrial/ventricular fibrillation

SIGNS & SYMPTOMS

- Usually asymptomatic
- In case of frequent premature contractions: lightheadedness, palpitations

DIAGNOSIS

OTHER DIAGNOSTICS

- ECG
- Holter monitor
- ZIO patch

TREATMENT

See individual disorders

PREMATURE ATRIAL CONTRACTION (PAC)

osms.it/premature-atrial-contraction

PATHOLOGY & CAUSES

- Contraction of atria earlier than normal in cardiac cycle
- Atrial bigeminy: premature atrial contraction consistently occurs after each normal cardiac cycle
- Atrial trigeminy: premature atrial contraction consistently occurs after every two normal cardiac cycles

CAUSES

• Heart structural disorders, intoxication, electrolyte imbalances

COMPLICATIONS

Atrial fibrillation

SIGNS & SYMPTOMS

- Usually asymptomatic
- In case of frequent premature contractions: lightheadedness, palpitations

DIAGNOSIS

OTHER DIAGNOSTICS

ECG

- Early, abnormal P wave
 - \circ Ectopic focus in bottom of atria \rightarrow negative P wave
 - Ectopic focus closer to atrioventricular (AV) node \rightarrow PR interval shorter
 - P wave, T wave overlap

- Noncompensatory pause
 - Premature impulse enters sinoatrial (SA) node → shortens cycle
 - Distinct from compensatory pause: premature ventricular contraction → premature impulse does not reach SA node → if AV node still refractory, pauses → lengthens cycle
- Normal QRS
 - Premature impulse reaches AV node in refractory → blocked premature atrial contraction → QRS nonexistent
- Ashman phenomenon
 - R-R interval prolongs → increases refractory period of right bundle branch → abnormal conduction of subsequent impulse → right bundle branch block on ECG
- Holter monitor
 - 24h, detect premature contractions

TREATMENT

Typically requires no treatment

MEDICATIONS

- If symptomatic: beta blockers/calcium channel blockers
- Electrolyte replacement

SURGERY

• If triggering atrial fibrillation: radiofrequency catheter ablation



Figure 17.1 Illustration depecting abnormal P wave in atrial bigeminy and trigeminy.



Figure 17.2 Illustration comparing normal ECG tracing vs ECG tracing with premature atrial contraction.

PREMATURE VENTRICULAR CONTRACTION (PVC)

osms.it/premature-ventricular-contraction

PATHOLOGY & CAUSES

- Contraction of ventricles earlier than normal in cardiac cycle
- Ectopic focus
 - Latent pacemakers: AV node, bundle of His/Purkinje fibers take over SA node's function of pacemaker
 - \circ Irritated cardiac muscle cells \rightarrow early depolarization
- Triggered activity
 - Ventricular repolarization → ventricle cells triggered by preceding action potential
 - Cause: reperfusion therapy after myocardial infarction/digoxin toxicity
- Reentrant loop
 - Tissue unable to depolarize (e.g. scar tissue, amyloid) → no signal conduction
 → depolarizing wave obstructed → depolarizing wave circles tissue → abnormal electrical circuit
- Ventricular bigeminy: premature ventricular contraction consistently comes after each normal cardiac cycle
- Ventricular trigeminy: premature ventricular contraction consistently comes after every two normal cardiac cycles

CAUSES

• Heart structural disorders, intoxication, electrolyte imbalances

RISK FACTORS

 Hypertension, smoking, exercise, stress, people of African descent (+30% risk), biological male

COMPLICATIONS

 Ventricular tachycardia, ventricular fibrillation, increased risk for sudden cardiac death

SIGNS & SYMPTOMS

- Can be asymptomatic
- Lightheadedness, palpitations

DIAGNOSIS

OTHER DIAGNOSTICS

ECG

- Wide, bizarre QRS: signal goes through ventricular muscle, not normal conduction pathway → conduction is slower than normal
- Ectopic impulse in right ventricle
 - Left bundle branch block pattern of QRS complex
 - V1: large negative complex, dominating S wave
- Ectopic impulse in left ventricle
 - Right bundle branch block pattern of QRS complex
 - V1: large positive complex, dominating R wave

- Abnormal ST segments: deviation from isoelectric baseline in opposite direction from QRS complex
- Inverted T waves in leads, QRS complex predominantly positive
- Nonexistent P wave: covered by wide QRS complex
 - QRS followed by compensatory pause
- Ventricular fusion beat: premature QRS complex occurs during PR segment, combines with normal depolarization wave
- R-on-T phenomenon: premature QRS complex occurs at/near T wave apex
- Holter monitor

TREATMENT

Typically requires no treatment

MEDICATIONS

• If symptomatic: venodilators, calcium channel blockers, administer beta blockers with caution

SURGERY

• If triggering ventricular arrhythmias: radiofrequency catheter ablation to destroy ectopic focus/replacement if necessary

OTHER INTERVENTIONS

• If mild, no exercise restrictions; if severe, reduced physical activity



Figure 17.3 Illustration comparing premature ventricular contractions that occur during a P wave, during a PR segment, and during a T wave.



Figure 17.4 Illustration comparing ventricular bigeminy and trigeminy.