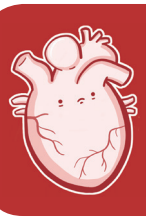

NOTES
SUPRAVENTRICULAR TACHYCARDIA

GENERALLY, WHAT IS IT?

PATHOLOGY & CAUSES

- Abnormally fast heart rhythms due to inappropriate electrical activity in upper portion of heart, atria/atrioventricular (AV) node
- Ventricles contract > 100 beats per minute, pathology originates above ventricles
  - Ventricles protected by gating at AV node

CAUSES
- Reentry conductive loops
- Increased automaticity
- Triggered activity

RISK FACTORS
- *Cardiac:* coronary artery disease, heart failure
- *Non-cardiac:* chronic obstructive pulmonary disease (COPD), pulmonary embolism, alcohol abuse, hyperthyroidism

SIGNS & SYMPTOMS

- Palpitations, chest pain, anxiety, dyspnea, syncope, lightheadedness

DIAGNOSIS

LAB RESULTS
- Electrolytes, thyroid stimulating hormone levels

OTHER DIAGNOSTICS
- ECG

TREATMENT

- See individual disorders

MEDICATIONS
- Calcium channel blockers, beta blockers, anticoagulants

SURGERY
- Catheter ablation

ATRIAL FIBRILLATION

osms.it/atrial-fibrillation

PATHOLOGY & CAUSES

- Rapid, irregular (no discernible rhythm) heart rate
- Progression
  - *Paroxysmal:* intermittent rhythm, may revert back to sinus rhythm spontaneously
  - *Persistent:* > seven days, requires intervention to convert back to sinus rhythm
  - *Permanent:* long-standing atrial fibrillation, cardioversion unsuccessful

CAUSES

- Disorganized waves of atrial depolarization, exact mechanisms not well understood
  - Regular impulses of sinus node overwhelmed by rapid electrical discharges from various sources (automatic foci, multiple reentry phenomena)
  - Arise from left more than right atrium

RISK FACTORS

- *Old age:* affects 4% 60–70, 14% > 80
- Obesity, diabetes mellitus, excessive alcohol consumption, genetic predisposition
- *Cardiovascular disease:* heart failure, hypertension, coronary artery disease, non-rheumatic mitral regurgitation, mitral valve prolapse, rheumatic heart disease, damaged atrial myocytes
- Increased catecholamine levels
- Lung disease
- Hyperthyroidism

COMPLICATIONS

- Thromboembolic events, heart failure, hypotensive shock

SIGNS & SYMPTOMS

- May be asymptomatic
- Dyspnea, fatigue, palpitations, lightheadedness, weakness, chest pain, hemodynamic shock

DIAGNOSIS

LAB RESULTS

- *Thyroid stimulating hormone (TSH) levels:* exclude hyperthyroidism

DIAGNOSTIC IMAGING

Transthoracic echocardiogram

- Evaluate atrial, ventricular size; valvular disease; left ventricular function; pericardial disease

Transesophageal echocardiogram

- Evaluate for atrial thrombi

OTHER DIAGNOSTICS

ECG

- Absent P waves
- Irregularly timed QRS complexes (irregular R-R intervals)
- No sawtooth wave in atrial fibrillation

TREATMENT

MEDICATIONS

Anticoagulation

- E.g. warfarin, dabigatran, apixaban, rivaroxaban
- CHA2DS2-VASc/CHADS2 score
  - Estimate risk of stroke in non-rheumatic atrial fibrillation; higher score = greater risk of stroke
  - *Score 0 (biological male)/1 (biological female):* low risk, no anticoagulation recommended
  - *Score 1 (biological male):* moderate risk, consider anticoagulation
  - *Score ≥ 2:* high risk, anticoagulation recommended
  - See table of scores

Rate control

- < 100 beats per minute
- Beta blockers (preferably β1 selective)
- Non-dihydropyridine calcium channel blockers (e.g. diltiazem, verapamil)
- Digoxin

Chemical cardioversion

- Administer antiarrhythmic medication
- Class Ic antiarrhythmics
- Class III antiarrhythmics
- Maintenance of sinus rhythm after cardioversion
  - Class Ic antiarrhythmics
  - Class III antiarrhythmics

OTHER INTERVENTIONS

Rhythm control

- Restore sinus rhythm via cardioversion

Electrical cardioversion

- Defibrillator for synchronization

Catheter ablation

- Destruction of heart regions responsible for abnormal impulses

CHA_2DS_2-VASc SCORING SYSTEM

	CONDITION	POINTS
C	Congestive heart failure: left ventricular systolic dysfunction	+1
H	Hypertension: consistently > 140/90 mmHg	+1
A_2	Age: ≥ 75 years	+2
D	Diabetes mellitus	+1
S_2	Prior stroke, TIA, thromboembolism	+2
V	Vascular disease: peripheral artery disease, myocardial infarction, aortic plaque	+1
A	Age: 65–74 years	+1
Sc	Sex: biological female	+1

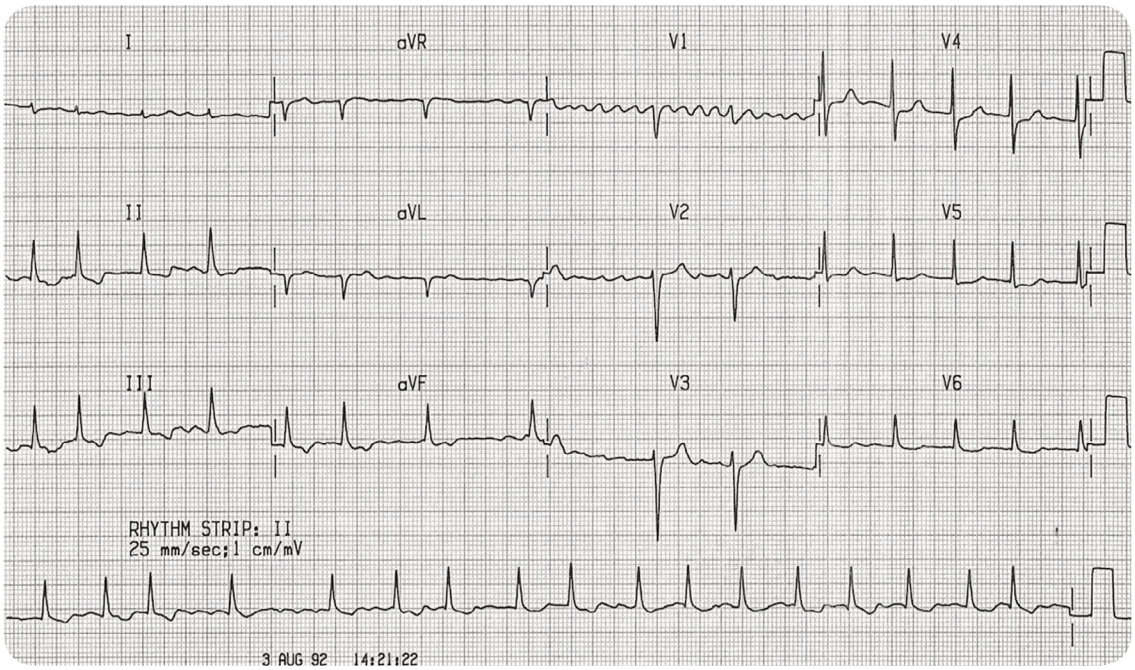

Figure 20.1 An ECG demonstrating atrial fibrillation.

ATRIAL FLUTTER

osms.it/atrial-flutter

PATHOLOGY & CAUSES

- Atria depolarize regularly at very high rates (200–350bpm), appear to flutter

TYPES

Typical atrial flutter (AKA Type 1 flutter)

- More common
- Single reentrant circuit, right atrium
- *Isthmus-dependent:* reentry circuit crosses cavotricuspid isthmus
- Circles tricuspid annulus (ring), usually counterclockwise (viewed from below)
- Cavotricuspid isthmus tissue propagates signal slower than surrounding tissue → circuit loops → slows propagation → surrounding tissue exits refractory period

Atypical atrial flutter (AKA Type 2 flutter)

- Less common
- Isthmus-independent
- Reentrant circuit develops in either atrium
- Associated with variety of reentry loops (common after incomplete atrial ablation procedures, right atrial surgical scars)

CAUSES

- Reentrant electrical signal from either atrium
- Reentrant signal loops back on itself → overrides normal sinus rhythm → establishes endless loop of stimulation
- Underlying disease (e.g. heart failure, valvular disease, hypertension, pulmonary disease) → heart cells less electrically stable → alters refractory periods → increased risk of reentrant circuits
- Reentrant circuits initiated by premature atrial contraction (PAC) → partial premature contraction, normal tissue relaxes → wave of stimulation propagates → normal tissue

contracts, premature tissue recovers → chance of reentrant circuit, stimulation wave doubles back on itself

RISK FACTORS

- Diseases that change atrial heart cell properties → differing electrophysiological properties in adjacent areas → reentry circuit
- Ischemia, fibrosis, previous myocardial infarction, heart failure, high blood pressure, diabetes, valvular heart disease, obstructive sleep apnea

COMPLICATIONS

- Heart failure, thromboembolic events, atrial fibrillation

SIGNS & SYMPTOMS

- Palpitations, tachycardia, fatigue
- Pain/tightness/discomfort in chest
- Heart failure
  - Exercise intolerance
  - Difficulty breathing at night/while lying flat
  - Edema of legs, abdomen

DIAGNOSIS

DIAGNOSTIC IMAGING

Echocardiogram

- Evaluate size of right, left atria, ventricles
- Detect pericardial/valvular heart disease
- Decreased ejection fraction (% of blood pumped by heart per contraction)

LAB RESULTS

- Serum electrolytes
- Renal function
- *Thyroid stimulating hormone (TSH) levels:* exclude hyperthyroidism

OTHER INTERVENTIONS

ECG

- Typical P waves absent
- *Typical atrial flutter:* P waves, saw tooth shape (F waves) localised to leads II, III, aVF
- *Atypical atrial flutter:* atrial activity (sawtooth waves/otherwise) may occur anywhere, dependent on reentrant circuit location
- Ventricular rate usually 1/2 atrial flutter rate (even ratios 2:1, 4:1 more common than odd, 3:1, 5:1)
- *1:1 atrial:* catecholamine excess, presence of accessory bypass tract/class 1A, 1C antiarrhythmic drug therapy

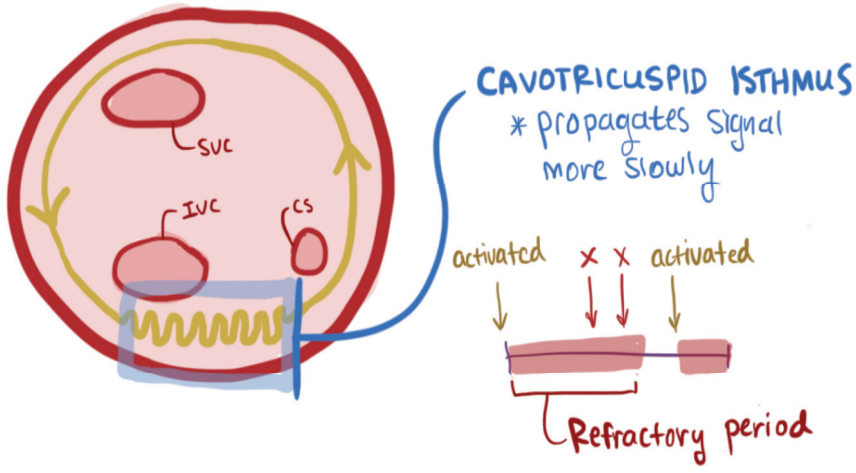

Figure 20.2 An electrocardiogram demonstrating atrial flutter with a 3:1 AV nodal block. The atrial trace demonstrates a characteristic sawtooth pattern.

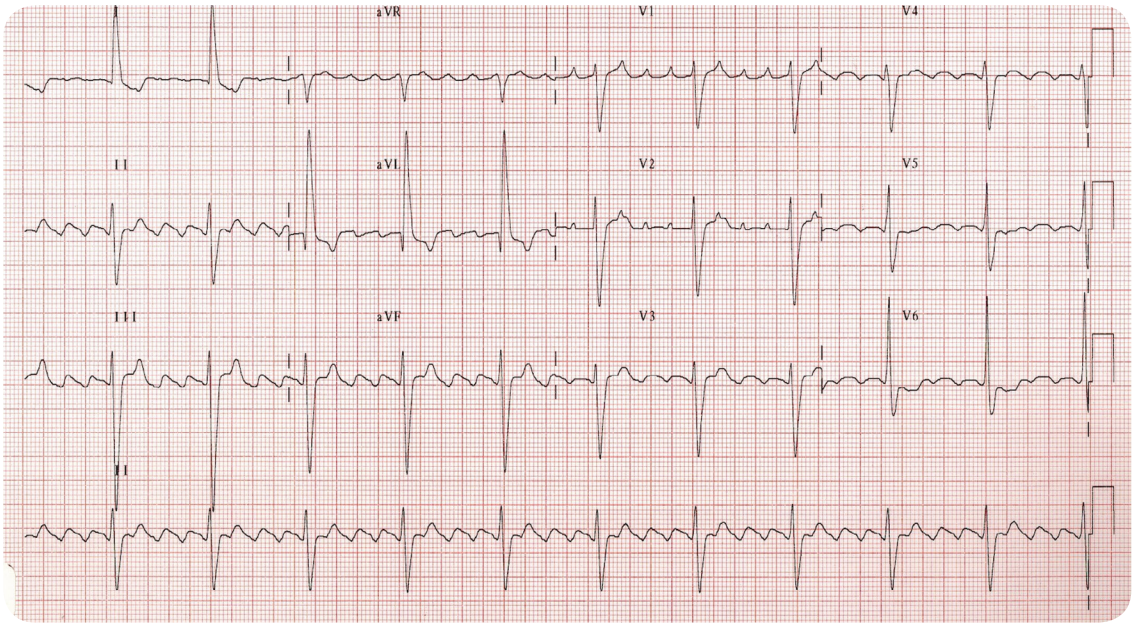

Figure 20.3 An electrocardiogram demonstrating atrial flutter with a 3:1 AV nodal block. The atrial trace demonstrates a characteristic sawtooth pattern.

TREATMENT

MEDICATIONS

- Anticoagulants (reduce chance of clot formation), beta blockers/calcium channel blockers (control rates of ventricles)

SURGERY

Radiofrequency catheter ablation

- Cavotricuspid isthmus no longer able to carry electrical signal, prevents reentry

OTHER INTERVENTIONS

Electrical cardioversion

- Depolarize atrial tissue, resynchronize contraction

ATRIOVENTRICULAR NODAL REENTRANT TACHYCARDIA (AVNRT)

osms.it/av-nodal-reentrant-tachycardia

PATHOLOGY & CAUSES

- Heart rate disturbance due to accessory pathway in/near AV node
- Electric conduction splits into two pathways → forms loop
  - *Alpha pathway:* slow conduction, short refractory period
  - *Beta pathway:* fast conduction, long refractory period

TYPES

Slow-fast/"typical" AVNRT

- Anterograde conduction to ventricles via slow pathway (alpha)
- Retrograde to atria conduction via fast pathway (beta)
- Depolarization down both pathways → reaches end of beta pathway first → signal splits
  - Travels to ventricles → contraction
  - Travels up alpha pathway → meets slow signal → signals cancel each other out
- Depolarization wave from premature beat reaches AV node → refractory fast (beta) pathway → signal initially down alpha pathway only → splits
  - Travels to ventricles → contraction
  - Travels up beta pathway → signal travels down alpha pathway, beta pathway comes out of refractory period → signal reaches end of alpha pathway →splits back up beta pathway

Fast-slow/"atypical" AVNRT

- Anterograde conduction via fast pathway, retrograde conduction via slow pathway

RISK FACTORS

- Biologically-female individuals → 75% of cases, emotional stress → alcohol use disorder, hyperthyroidism, electrolyte disturbances

SIGNS & SYMPTOMS

- Palpitations, transient chest pain, bouts of transient tachycardia, transient hypotension, (pre)syncope

DIAGNOSIS

LAB RESULTS

- Thyroid function
- Serum electrolytes

OTHER DIAGNOSTICS

ECG

- Tachycardia 140–280bpm
- Absent P waves
- P waves immediately before/after QRS complex
- P waves inverted/retrograde
- R' waves (small secondary R waves)

TREATMENT

MEDICATIONS

- Adenosine, beta blockers, calcium channel blockers to slow AV node conduction

OTHER INTERVENTIONS

Radiofrequency catheter ablation

- Definitive treatment
- Ablation of slow alpha pathway

Slow AV node conduction

- Vagal maneuver (carotid sinus massage/ Valsalva maneuver) → activates vagus nerve

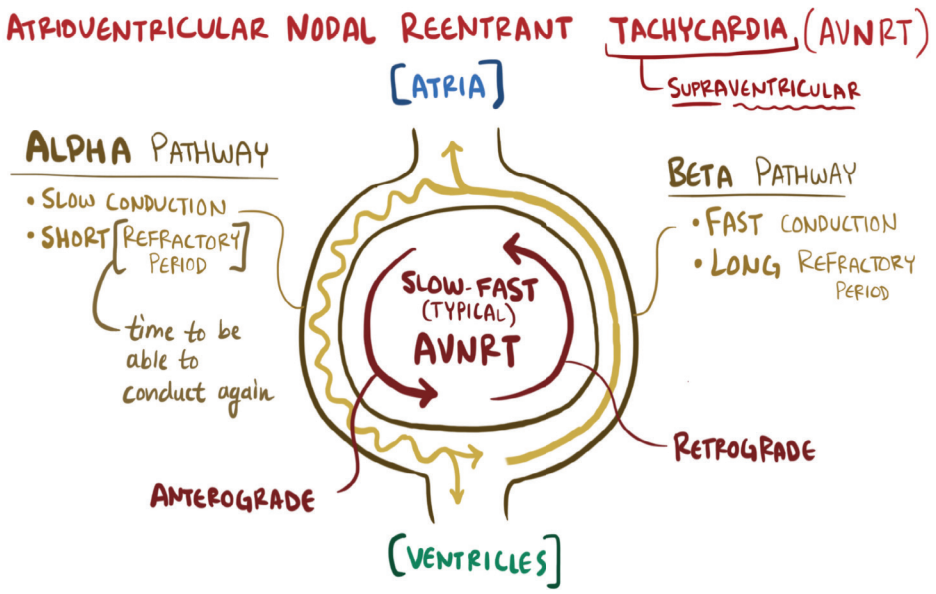

Figure 20.4 Illustration depicting path of electrical conduction in AV node during slow-fast AVNRT.

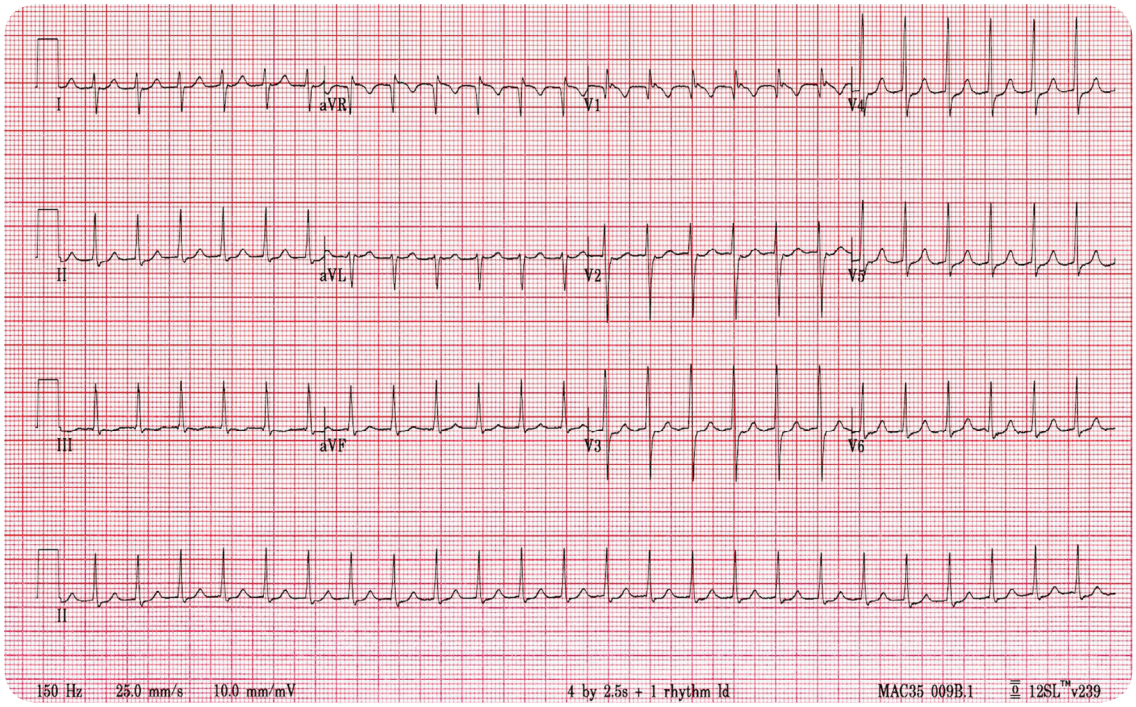

Figure 20.5 An ECG demonstrating typical (slow-fast) AVNRT. R waves are best seen in lead V1.