2. Premature Beats
- premature atrial contraction
  - ectopic supraventricular beat originating in the atria
  - P wave morphology of the PAC usually differs from that of a normal sinus beat
- junctional premature beat
  - ectopic supraventricular beat that originates in the vicinity of the AV node
  - P wave is usually not seen or an inverted P wave is seen and may be before or closely follow the QRS complex (referred to as a retrograde, or "traveling backward" P wave)
- treatment usually not required

3. Atrial Flutter
- rapid, regular atrial depolarization from a macro re-entry circuit within the atrium (most commonly right atrium)
- atrial rate 250-350 bpm, usually 300 bpm
- AV block usually occurs; it may be fixed (e.g. 2:1, 3:1, 4:1, etc.) or variable
- etiology: hypertension, cardiomyopathy , in association with atrial fibrillation; less often, CAD, thyrotoxicosis, mitral valve disease, cardiac surgery, COPD, PE, pericarditis
- ECG: sawtooth flutter waves (most common type of flutter, called "isthmus dependent, typical flutter) in inferior leads (II, III, aVF); narrow QRS (unless aberrancy); commonly seen as 2:1 block with HR of 150
- in atrial flutter with 2:1 block, carotid sinus massage (first check for bruits), Valsalva maneuver, or adenosine may decrease AV conduction and allow flutter waves to be more easily seen
- treatment of acute atrial flutter
  - if unstable (e.g. hypotension, CHF, angina): electrical cardioversion
  - if stable:
    1. rate control: β-blocker, diltiazem, verapamil, or digoxin
    2. chemical cardioversion: sotalol, amiodarone, type I antiarrhythmics, or electrical cardioversion
  - anticoagulation guidelines same as for patients with AFib
- treatment of long-term AFib includes antiarrhythmics and radiofrequency (RF) ablation (success rate dependent on site of origin of atrial flutter)

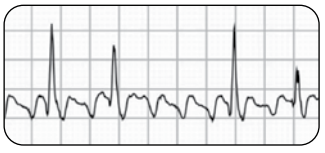

Figure 25. Atrial flutter with variable block

4. Multifocal Atrial Tachycardia (MAT)
- irregular rhythm caused by presence of 3 or more atrial foci (may mimic AFib)
- atrial rate 100-200 bpm
  - 3 or more distinct P wave morphologies and PR intervals vary, some P waves may not be conducted
- occurs more commonly in patients with COPD and hypoxemia; less commonly in patients with hypokalemia, hypomagnesemia, sepsis, theophylline, or digitalis toxicity
- treatment: treat the underlying cause; calcium channel blockers may be used (e.g. diltiazem, verapamil), β-blockers may be contraindicated because of severe pulmonary disease
- no role for electrical cardioversion, antiarrhythmics, or ablation

5. Atrial Fibrillation
- see *CCS Atrial Fibrillation Guidelines 2016* for details (free mobile app – iCCS available on iOS and Android)
- most commonly sustained arrhythmia
- incidence increases with age (10% of population >80 yr)
- symptoms: palpitations, fatigue, dyspnea, syncope, may precipitate or worsen heart failure
- **classification**
  - lone: generally occurs in persons <65 yr and in whom no clinical or echocardiographic causes are found
  - nonvalvular: not caused by valvular disease, prosthetic heart valves, or valve repair
  - paroxysmal: episodes that terminate spontaneously
  - persistent: AFib sustained for more than 7 d or AFib that terminates only with cardioversion
  - permanent/chronic: continuous AFib that is unresponsive to cardioversion or in which clinical judgement has led to a decision not to pursue cardioversion
  - recurrent: two or more episodes of AFib
  - secondary: caused by a separate underlying condition or event (e.g. MI, cardiac surgery, pulmonary disease, hyperthyroidism)
  - may be associated with thromboembolic events (stroke risk can be assessed by CHADS2 score in nonvalvular AFib; CHADS2-VASc if the former gives a score of 0 or 1)
- **initiation**
  - single circuit re-entry and/or ectopic foci, mostly arising from the pulmonary veins, act as aberrant generators producing atrial tachycardia (350-600 bpm), which leads to multiple re-entry circuity (microreentry)
  - impulses conduct irregularly across the atrial myocardium to give rise to fibrillation
  - in most cases, ectopic foci have also been mapped to the pulmonary vein ostia and can be ablated
- **maintenance**
  - the tachycardia causes atrial structural and electrophysiological remodelling changes that further promote AFib; the longer the patient is in AFib the more difficult it is to convert back to sinus rhythm
- **consequences**
  - the AV node irregularly filters incoming atrial impulses producing an irregular ventricular response usually <200 bpm, and the tachycardia leads to suboptimal cardiac output
  - fibrillatory conduction of the atria promotes blood stasis increasing the risk of thrombus formation
    - AFib is an important risk factor for stroke

Atrial Fibrillation – AFFIRM Trial
NEJM 2002;347:1825-1833
Study: Randomized, multicentre trial with mean follow-up of 3.5 yr.
Population: 4060 patients (mean age 70 yr, 61% male, 89% white) with AF and a high risk of stroke or death.
Intervention: Rate control (β-blockers, calcium channel blockers, or digoxin alone or in combination) vs. rhythm control (antiarrhythmic drug chosen by the treating physician).
Primary Outcome: All cause mortality.
Results: There was no difference in mortality or disabling stroke, anoxic encephalopathy, major bleeding, and cardiac arrest between the two groups. There were more incidents of hospitalizations (80.1% vs. 73%, p<0.001) and adverse events (Torsades de Pointes (12 vs. 2, p=0.007), pulseless or bradycardic arrest (9 vs. 1, p=0.01), pulmonary event (108 vs. 24, p<0.001), gastrointestinal event (127 vs. 35, p<0.001), prolonged QT interval (31 vs. 4, p≤0.001), bradycardia (105 vs. 64, p=0.001) in the rhythm-control group.
Conclusion: Rate-control was as effective as rhythm-control in AF and was better tolerated. There were more hospitalization incidents in the rhythm-control group.

Table 5. CHADS2 Risk Prediction for Non-Valvular AFib

Risk Factor	Points	CHADS2 Score	Stroke Risk (%/Yr)
Congestive Heart Failure	1	0	1.9 (low)
Hypertension	1	1	2.8 (low-mod)
Age >75	1	2-3	4.0-5.9 (mod)
Diabetes	1	4-6	8.5-18.2 (high)
Stroke/TIA (prior)	2		

JAMA 2001;285:2864-70 and Can J Cardiol 2014 Oct;30(10):1114-30.

Lenient versus Strict Rate Control in Patients with Atrial Fibrillation
NEJM 362:1363-1373
Study: Randomized, multi-centre Netherlands prospective study, follow-up for at least 2 yr
Population: 614 patients with permanent atrial fibrillation.
Intervention: Lenient control (resting HR <110 bpm) or strict control (resting HR <80 bpm)
Primary Outcomes: Death from cardiovascular causes, hospitalization for heart failure, and stroke, systemic embolism, bleeding, and life-threatening arrhythmic events.
Results: Goal of the study was to establish whether lenient control was equivalent to strict control for prevention of primary outcomes. Resulting hazard ratios were not significantly different between the treatment groups (P = 0.001). Frequencies of hospitalization and adverse effects were also similar. More patients were able to maintain lenient targets (97.7%) compared to strict targets (67%).
Conclusion: Lenient control was equivalent to strict control for prevention of primary outcomes in patients with atrial fibrillation. Furthermore, lenient control was more easily achieved.

- **ECG findings**
  - no organized P waves due to rapid atrial activity (350-600 bpm) causing a chaotic fibrillatory baseline
  - irregularly irregular ventricular response (typically 100-180 bpm), narrow QRS (unless aberrancy or previous BBB)
  - wide QRS complexes due to aberrancy may occur following a long-short cycle sequence ("Ashman phenomenon")
  - loss of atrial contraction, thus no "a" wave seen in JVP, no S4 on auscultation

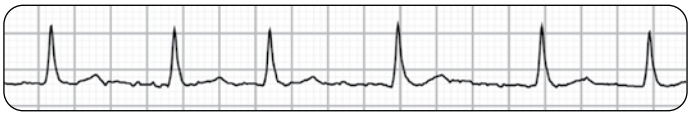

Figure 26. Atrial fibrillation (lead II)

- management (adapted from CCS Atrial Fibrillation Guidelines 2016 & 2018)
  - primary goal is symptom control
  - **major objectives (RACE)**: all patients with AFib (paroxysmal, persistent, or permanent), should be stratified using a predictive index for stroke risk and for the risk of bleeding, and most patients should receive either an oral anticoagulant or ASA
    1. **R**ate control: β-blockers, diltiazem, verapamil (in patients with heart failure: digoxin, amiodarone)
       - digoxin can be considered as a therapeutic option to achieve rate control in patients whose response to β-blockers and/or calcium channel blockers is inadequate, contraindicated or not tolerated
    2. **A**nticoagulation: use either warfarin or novel oral anticoagulant (NOACs) e.g. apixaban, dabigatran, rivaroxaban, edoxaban to prevent thromboembolism
       - for patients with non-valvular AF (NVAF): oral anticoagulant (OAC) is recommended for most patients aged >65 yr or $CHADS_2$ ≥1
         - ASA 81 mg is recommended only for patients with none of the risk outlined in the CCS algorithm (age <65 and no $CHADS_2$ risk factors) who also have arterial disease (coronary, aortic, or peripheral)
         - novel oral anticoagulant (NOAC) is to be used in preference to warfarin
    3. **C**ardioversion (electrical)
       - if AFib <48 h, can usually cardiovert without anticoagulation
       - if AFib >48 h, anticoagulate for 3 wk prior and 4 wk after cardioversion due to risk of unstable intra-atrial thrombus
       - if patient unstable (hypotensive, active angina due to tachycardia, uncontrolled heart failure) should cardiovert immediately
    4. **E**tiology
       - HTN, obesity, sleep apnea, CAD, valvular disease, pericarditis, cardiomyopathy, myocarditis, ASD, post-operative, PE, COPD, thyrotoxicosis, sick sinus syndrome, alcohol ("holiday heart")
       - may present in young patients without demonstrable disease ("lone AFib") and in the elderly without underlying heart disease
  - studies of patients with AFib suggest that there is no difference in long-term survival when treating patients with a rhythm-control versus rate-control strategy
  - many patients with a significant underlying structural heart lesion (e.g. valve disease, cardiomyopathy) will not tolerate AFib well (since may be dependent on atrial kick) and these patients should be cardioverted (chemical or electrical) as soon as possible
  - **newly discovered AFib**
    - anticoagulants may be beneficial if high risk for stroke
    - if the episode is self-limited and not associated with severe symptoms, no need for antiarrhythmic drugs
    - if AFib persists, 2 options:
      1. rate control and anticoagulation (as indicated above)
      2. cardioversion (as indicated above)
    - recurrent or permanent AFib
    - if episodes are brief or minimally symptomatic, antiarrhythmic drugs may be avoided; rate control and anticoagulation are appropriate
    - patients who have undergone at least one attempt to restore sinus rhythm may remain in AFib after recurrence; permanent AFib may be accepted (with rate control and antithrombotics as indicated by $CHADS_2$ score) in certain clinical situations
    - if symptoms are bothersome or episodes are prolonged, antiarrhythmic drugs should be used
      - no or minimal heart disease: flecainide, propafenone once proven to have no underlying CAD (may consider exercise stress testing)

- LV dysfunction: amiodarone
- CAD: β-blockers, amiodarone
- if antiarrhythmic drugs fail or are not tolerated, can consider RF ablation for rhythm/symptom control

6. AV NODAL RE-ENTRANT TACHYCARDIA (AVNRT)

- re-entrant circuit using dual pathways (fast conducting β-fibres and slow conducting α-fibres) within or near the AV node; often found in the absence of structural heart disease
  - cause is commonly idiopathic, although familial AVNRT has been reported
- sudden onset and offset, patients often describe "neck pounding" and "shirt flapping"
- fast regular rhythm: rate 150-250 bpm
- usually initiated by a supraventricular or ventricular premature beat
- AVNRT accounts for 60-70% of all paroxysmal SVTs
- retrograde P waves may be seen but are usually lost in the QRS complex
- treatment
  - acute: Valsalva maneuver or carotid sinus pressure technique, adenosine is first choice if unresponsive to vagal maneuvers; if no response, try metoprolol, digoxin, diltiazem, electrical cardioversion if patient hemodynamically unstable (hypotension, angina, or CHF)
  - long-term: 1st line – radiofrequency ablation (>98% cure rate and << 1% complication rate), β-blocker, diltiazem, digoxin; 2nd line – flecainide, propafenone

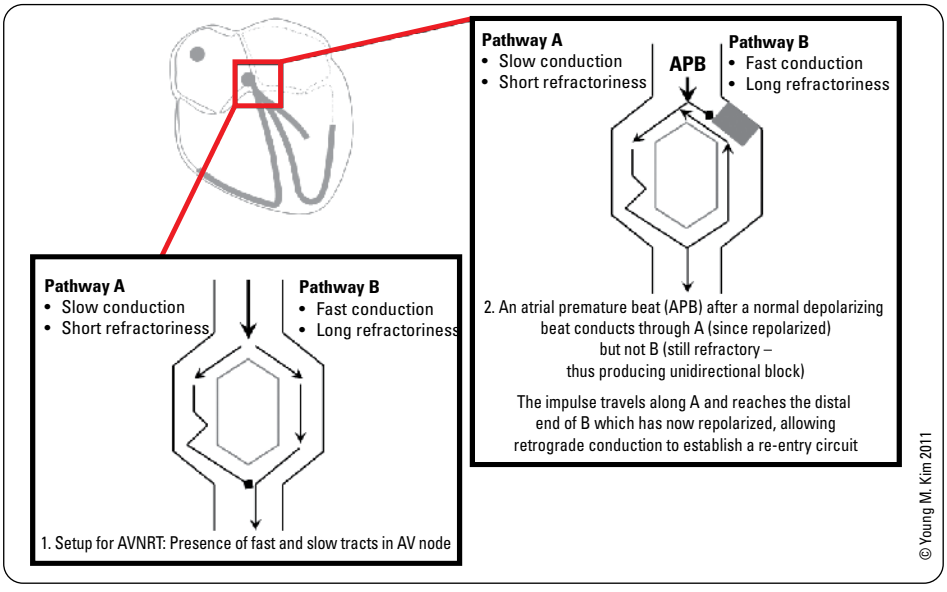

Figure 28. Mechanism for AVNRT

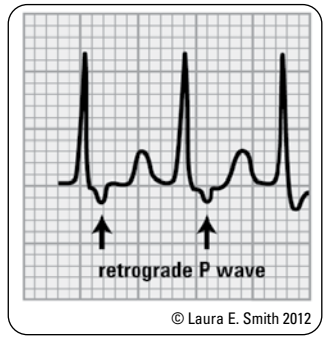

Figure 27. AVNRT

N.B. Refer to ECG Made Simple for further discussion and an animation of the mechanism (www.ecgmadesimple.com)

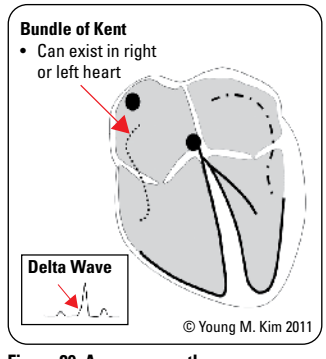

Figure 29. Accessory pathway conduction in WPW. Early ventricular activation leads to the appearance of a delta wave (slurred upstroke of the QRS) on the ECG before usual conduction across the AV node

Pre-Excitation Syndromes

- refers to a subset of SVTs mediated by an accessory pathway which can lead to ventricular pre-excitation

Wolff-Parkinson-White Syndrome (WPW)

- congenital defect present in 1.5-2/1000 of the general population
- an accessory conduction tract (Bundle of Kent; can be in right or left atrium) abnormally allows early electrical activation of part of one ventricle
- impulses travel at a greater conduction velocity across the Bundle of Kent thereby effectively 'bypassing' AV node
- since the ventricles are activated earlier, the ECG shows early ventricular depolarization in the form of initial slurring of the QRS complex – the so-called "delta wave"
- atrial impulses that conduct to the ventricles through both the Bundle of Kent and the normal AV node/His-Purkinje system generate a broad "fusion complex"
- ECG features of WPW
  - PR interval <120 msec
  - delta wave: slurred upstroke of the QRS (the leads with the delta wave vary with site of bypass)
  - widening of the QRS complex due to premature activation
  - secondary ST segment and T wave changes
  - tachyarrhythmias may occur – most often AVRT and AFib

AFib in WPW Patients

- AFib is the index arrhythmia in up to 20% of patients with WPW syndrome
  - usually intermittent rather than persistent or permanent
- rapid atrial depolarizations in AFib are conducted through the bypass tract which is not able to filter impulses like the AV node can and thus the ventricular rate becomes extremely rapid (>200 bpm) and the QRS complex widens