

## ARRHYTHMIAS

1. Bradycardia is caused by impaired impulse generation (automaticity) or conduction (heart block). Idiopathic causes include calcification & fibrosis of sinus node & conduction system; reversible causes include Lyme disease, hypothyroidism, hyperkalemia, meds, & ischemia.
2. Bradycardia causes fatigue, exercise intolerance, dizziness, syncope.
3. Holter or event monitor are used to correlate symptoms with HR.
4. Symptomatic bradycardia is treated by alleviation of underlying cause or insertion of a pacemaker. Causative meds should be stopped unless their benefit outweighs risk of pacemaker placement.
5. *Sick sinus syndrome (sinus node dysfunction)* includes reduced SA node automaticity, exit block, & sinus arrest. Affects elderly, 50% have paroxysmal afib/flutter, & 11% distal conduction disease.
6. *Tachy-brady syndrome* is SB alternating with afib/RVR. Bradycardia is worsened by rate control meds; pacemaker is usually required.
7. AV block (AVB) is due to fibrosis & degeneration of the conduction system, ischemia & meds. Symptoms are of cerebral hypoperfusion.
8. 1<sup>st</sup> degree & Mobitz I AVB- rarely symptomatic; no therapy needed.
9. Mobitz II AVB (*high-grade* or *advanced 2<sup>nd</sup>-degree AVB* if  $\geq 2$  consecutive nonconducted P waves) & complete (3<sup>rd</sup> degree) AVB require pacemaker in the absence of modifiable causes.
10. MRI is contraindicated in most pacemaker/implanted cardiac device.
11. Wide-complex tachycardia should be treated as VT, unless aberrant SVT or accessory pathway tachycardia is certain. CCB & adenosine should be avoided with possible VT, esp. with LV dysfunction.
12. Class IC antiarrhythmic agents (AA) "organize" fib to flutter & 1:1 conduction; AV blockers ( $\beta$ -blockers, CCB, digoxin) should be used in conjunction. They are contraindicated in CAD patients (increase SCD)
13. Class III AA prolong the QT causing torsade. Initiation of sotalol & dofetilide (*not amiodarone*) should be on an inpatient basis.
14. Dig blocks AVN by  $\uparrow$  vagal tone; effective in elderly/inactive patients.
15. Adenosine transiently blocks AVN; terminates AVN-dependent SVTs.
16. Afib causes palpitations, fatigue, dyspnea, chest pain, dizziness, syncope; CHF & stroke. Symptoms lessen as it becomes permanent.
17. With long-standing uncontrolled high ventricular rates ( $>130$  beats/min), tachycardia-related cardiomyopathy can develop.
18. Associated conditions include HTN, CAD; valvular disease; dilated, hypertrophic, & restrictive CM; CHF; preexcitation syndromes.
19. Afib is common, self-limited, post cardiac surgery. Also caused by alcohol, thyroid disorders, myocarditis or pericarditis, & PTE.
20. 50% of afib spontaneously convert. Unstable patients need prompt cardioversion. Otherwise AVN blocking agents are given.
21. In preexcited afib with RVR, AVN blockers must be avoided as they can increase conduction through accessory pathway & provoke vfib.
22. ASA or warfarin can be initiated outpatient. If heparin is initiated inpatient, it is usually continued till warfarin is therapeutic.

23. In asymptomatic afib, rate control strategy is acceptable.
24. Patients in afib for < 48 hrs are cardioverted without anticoagulation. Otherwise, they need 3 wks warfarin (INR 2 – 3), or TEE (r/o LAA clot) while on IV heparin. 4 wks warfarin are needed post-cardioversion.
25. Recurrence rate of symptomatic afib is 20 – 50% (>1 yr) even on AA.
26. For symptomatic refractory afib- AVN ablation + pacemaker for rate control; LA/pulm vein ablation or surgical maze for rhythm control.
27. Aflutter is often associated with congenital heart disease, pulmonary disease, hypertension, DM, obesity, thyroid disorders, sick sinus syndrome, and major cardiothoracic surgery.
28. In pharm conversion of aflutter, class I are less effective than class III AA.  $\beta$ -blockers & CCB control rate; digoxin can be effective adjunct.
29. A flutter recurrence rate is 60% (>6 months). RF ablation is first-line for recurrence (> 90% success); with 1 – 2% major complications risk (vascular damage, heart block, stroke, tamponade, death).
30. Warfarin is recommended for afib/aflutter with  $\geq 1$  CHADS2 score.
31. SVT comprises AVNRT (60%), AVRT (30%); & atrial tachycardia (10%).
32. Other rare forms include sinus node reentrant tachycardia, inappropriate ST, & junctional tachycardia.
33. In *typical* AVNRT, conduction is antegrade via *slow pathway*, & retrograde via *fast pathway*. In *atypical* AVNRT, circuit is reversed.
34. Accessory pathways (AVRT) conduct antegrade (atrium to ventricle) & *manifest* delta wave on ECG; or only retrograde (*concealed*).
35. Atrial tachycardia results from enhanced automaticity. Triggered activity (after-depolarizations that reach threshold potential) or reentry (in congenital HD or cardiac surgery) are less common.
36. SVT causes palpitations with chest or neck pounding, dyspnea, dizziness, anxiety, and, rarely, syncope. It has abrupt onset and termination, & responds to vagal maneuvers, such as Valsalva, squatting, carotid sinus massage, or cold water facial immersion.
37. Adenosine is effective at terminating SVT that uses the AV node; it is contraindicated in severe bronchospastic disease. IV CCB are also recommended. IV  $\beta$ -blockers or amiodarone are second-line.
38. Patients with rare SVT episodes that can be easily terminated with Valsalva may opt for watchful approach, avoiding triggers. Low-dose  $\beta$ -blockers or CCB can abbreviate or eliminate episodes.
39. Ablation- 95 – 99% success in AVNRT/AVRT; less in atrial tachycardia.
40. AVN blockade is contraindicated in patients with preexcited afib.; Procainamide should be used to treat the arrhythmia. RF ablation is first-line for WPW because of very small, yet persistent, risk of SCD.
41. PVCs at rest with structurally normal heart do not increase mortality. Exercise/recovery-induced PVCs, repetitive or complex ectopy in the setting of heart disease increase mortality; risk is from pathologic substrate, suppression of ambient PVCs does not reduce mortality.
42. Many patients with symptomatic PVCs respond to reassurance. First-line therapy is  $\beta$ -blocker or CCB. Class IC & III AA also can be used. RF ablation is appropriate with severe symptoms refractory to AA.

43. NSVT ( $\geq 3$  consecutive PVCs,  $< 30$  seconds) prevalence increases with age, LV dysfunction, & CM. In chronic CHF prevalence is 30 – 80%.
44. NSVT within 48 hrs of acute MI does not confer additional risk. In the year post MI it increases mortality. With normal EF, no therapy is needed; EF  $\leq 35\%$ , ICD is considered; EF 35 – 55%, EPS is considered.
45. NSVT is treated similar to PVCs. CAD contraindicates Class IC AA.
46. Sustained VT ( $> 100$ /min episode lasting  $> 30$  seconds, or requiring cardioversion) most commonly is due to reentry around prior MI.
47. Monomorphic VT prognosis depends on underlying heart disease.
48. Polymorphic VT is related to genetic defect; causes fatal arrhythmias.
49. Sustained, symptomatic VT in ischemic or nonischemic CM is an adverse prognostic indicator with a high risk of recurrence.
50. Medical Therapy does not improve survival in VT with heart disease.
51. Idiopathic VT (mostly RVOT tachycardia) is usually benign; because of overlap with ARVC; MRI helps assess for RV fibrofatty infiltrate. No therapy for mild, infrequent symptoms. CCB or  $\beta$ -blockers help suppress symptoms. RF ablation is  $> 90\%$  successful.
52. SCD is collapse within 1 hr of symptoms. Majority of events occur out of hospital; 50% are first expression of undiagnosed CAD.
53. SCD risk factors include CAD risk factors; CHF, LVH, heavy alcohol use. Triggers include electrolyte disturbances, drugs (cocaine, QT-prolonging drugs, AA drugs), acute exercise, and emotional stress.
54. Most SCD victims do not have identifiable risk factors. Vfib is most common mechanism of SCD.
55. Disposition to SCD- familial DCM, ARVC, & HCM; channelopathies; & gene polymorphisms with normal cardiac structure.
56. Long QT syndrome arrhythmia risk is predicted by QTc  $> 500$  msec, female sex, & genotype.  $\beta$ -blockers are main therapy. ICD in patients at high SCD risk (events on  $\beta$ -blockers, strong family history of SCD).
57. Spontaneous Brugada with syncope is high SCD risk; ICD is indicated.
58. Other inherited disorders with SCD risk include short QT syndrome (QT  $< 300$  msec); and catecholaminergic polymorphic VT (ryanodine receptor mutation; VT during physical activity or emotional stress).
59. Cardiac arrest survivors and patients with malignant ventricular arrhythmias have 20 – 50% recurrence; no trials of AA drugs have shown benefit in outcome.
60. ICDs reduce SCD risk & overall mortality in patients with impaired EF & CM. ICDs also reduce the risk of recurrent SCD in survivors.

### Anticoagulation in Atrial Fibrillation.

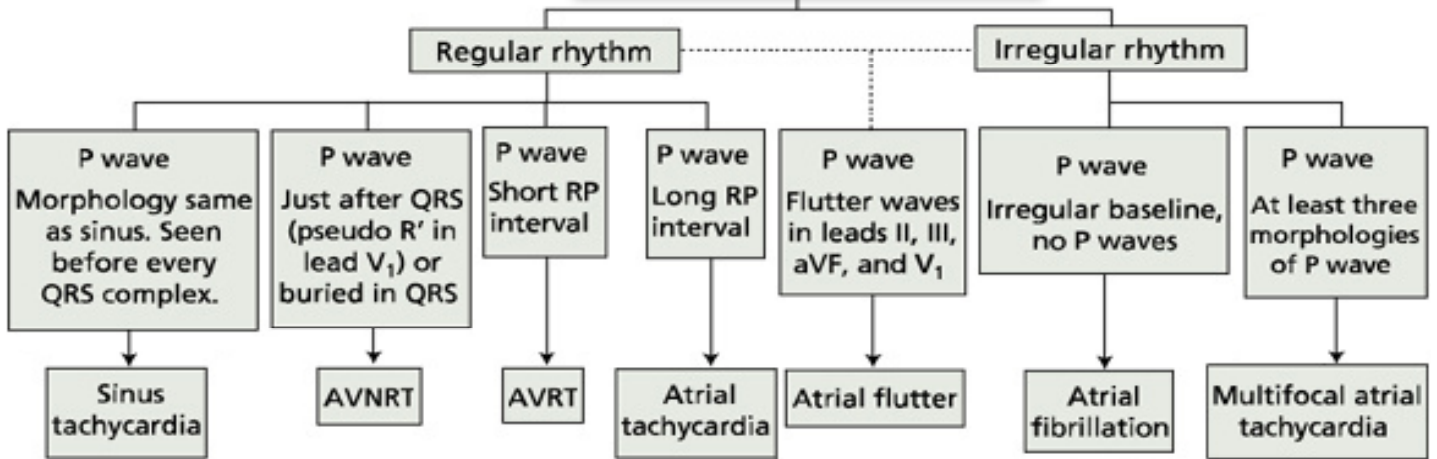
CHADS2 score is calculated by assigning 1 point each for presence of chronic heart failure, hypertension, age 75 yrs or older, & DM; and 2 points for history of stroke or transient ischemic attack.

History of stroke or TIA confers high risk regardless of score.

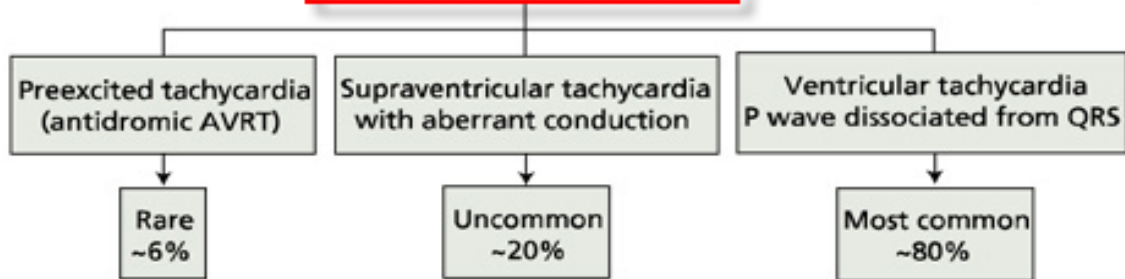
| CHADS <sub>2</sub> | Stroke Risk          | Warfarin       |
|--------------------|----------------------|----------------|
| 0                  | Low                  | No             |
| 1                  | Intermediate         | Individualized |
| 2                  | Intermediate or high | Individualized |
| 3                  | High                 | Yes            |
| 4                  | High                 | Yes            |
| 5                  | High                 | Yes            |
| 6                  | High                 | Yes            |

## Classification of Narrow- and Wide-Complex Tachycardias

### Narrow-complex tachycardia



### Wide-complex tachycardia



## Antiarrhythmic Drugs

| Vaughan Williams Classification | General Mechanism                                  | Commonly Used Drugs               | Clinical Uses   |
|---------------------------------|--|-----------------------------------|---|
| IA                              | Fast sodium channel blockade; prolongs AP duration | Procainamide                      | Preexcited AF, SVTs, ventricular arrhythmias  |
| IB                              | Fast sodium channel blockade; shortens AP duration | Lidocaine                         | Ventricular arrhythmias, particularly in setting of ischemia  |
| IC                              | Fast sodium channel blockade; slows conduction     | Flecainide                        | Life-threatening ventricular arrhythmias, SVT, PAF, AFL   |
|                                 |  | Propafenone                       | Life-threatening ventricular arrhythmias, PAF, PAFL, PSVT   |
| II                              | $\beta$ -Blockade                                  | Atenolol, metoprolol, propranolol | Adrenergically mediated tachycardias, rate control of AF, AFL, SVTs using AV node, ventricular arrhythmias, reduction in SCD after MI |
| III                             | Potassium channel blockade                         | Amiodarone                        | Useful in a wide spectrum of ventricular and supraventricular arrhythmias   |
|                                 |  | Sotalol                           | VT, AF, AFL, SVT  |
|                                 |  | Dofetilide                        | SVT, particularly AF, AFL   |
| IV                              | Calcium channel blockade                           | Verapamil, diltiazem              | Termination of SVTs, rate control of AF, AFL, therapy of SVTs, tachycardias mediated by triggered activity                            |