Patient with SVT just given adenosine –
1) what is rhythm?, 2) what is Mx?
The GP refresher course
16 June 2012

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Holter shows AF – who to refer?

### SUMMARY

<table>
<thead>
<tr>
<th>Hook-up date</th>
<th>09/02/2012 17:59:00</th>
<th>Min Sinus HR (( \star / \square ))</th>
<th>48 (48/49) at 18:54:20</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration</td>
<td>22:53</td>
<td>Mean Sinus HR (( \star / \square ))</td>
<td>71 (72/69)</td>
</tr>
<tr>
<td>Recorder n°</td>
<td>VIA09001099</td>
<td>Max Sinus HR (( \star / \square ))</td>
<td>185 (185/144) at 10:53:18</td>
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<tr>
<td>Duration analysed</td>
<td>22:52:29</td>
<td>SDNN</td>
<td>202 ms</td>
</tr>
<tr>
<td>Number of QRS</td>
<td>115421</td>
<td>PNN50</td>
<td>38.34 %</td>
</tr>
</tbody>
</table>

- Bradycardias: 0
- Pauses: 0; RR max 1.51 seconds (7:00:52)
- Relative pauses: 192; longest 1.51 seconds (7:00:52)
- AF: 3; longest 37832 QRS, 6:29:03 at 97 min\(^{-1}\) (4:22:03)
- AF burden: 41\% (09:28); Mean HR 102 min\(^{-1}\) (48/231 min\(^{-1}\))
- ST: 0

#### Ventricular events

<table>
<thead>
<tr>
<th>VPB</th>
<th>285 (0%)</th>
<th>Templates</th>
<th>1</th>
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<tbody>
<tr>
<td>Couplets</td>
<td>6</td>
<td>Triplets</td>
<td>0</td>
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<tr>
<td>Bigeminy cycles</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VT</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IVR/AIVR</td>
<td>0</td>
<td></td>
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</table>

#### Supraventricular events

<table>
<thead>
<tr>
<th>SVPB</th>
<th>883 (0%)</th>
<th>Couplets</th>
<th>173</th>
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</thead>
<tbody>
<tr>
<td>Triplets</td>
<td>45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SVPT</td>
<td>31</td>
<td>longest 6 QRS at 112 min(^{-1}) (1:34:01)</td>
<td></td>
</tr>
</tbody>
</table>

### Technical Report:

Sinus Rhythm with AF 41\% of the duration of the recording (AF start 04:20:48 continues until 14:10:07)
Please see strips attached

- VE couples x 6
- VE singles x 260
- VE bigeminy cycles x 4
- VE runs x 31
- VE triplets x 45
- VE couples x 173
- VE singles x 225
- VE trigeminy cycles x 6
- VE bigemeyini cycles x 1

Max HR: (AF) 231bpm at 11:31:52
Min HR: (SR) 48bpm at 18:54:20

No diary events entered
Sinus Rhythm with AF 41% of the duration of the recording (AF start 04:20:48 continues until 14:10:07)
Please see strips attached
4:22:03 Atrial fibrillation; Number of QRS = 37892; Duration = 06:29:03s; Mean HR = 97 min⁻¹ (48/209)
Definitions

Paroxysmal: spontaneous termination <7 days
Persistent: AF > 7 days
Longstanding Persistent: AF > 1 year
Permanent: “State of mind” of physician/patient - acceptance of long term AF
Caveat, Post “early” DCCV: within 48h (Paroxysmal)
: > 48 hours (Persistent)
Case scenario 1

76 year old lady with HTN, DM, with persistent AF for 5 years and NYHA 2. Echo shows mildly impaired LV and LA size of 4.9cm. What is optimal management?
Case scenario 2

- 45 year old man with palpitations weekly, lasting up to 3 hours – unable to work during this time as highly symptomatic. Normal LV and LA 3.6cm. No underlying heart disease. ? Management
Case scenario 3

- 66 yo man with persistent AF for 12 months, LA size 4.6, Normal LV. Hypertensive. Fatigued but thinks it’s “old age” catching up with him. ? Management
Management

Questions.
1. Rate vs Rhythm?
2. Anticoagulate or not?
Mechanisms of AF

- **Focal Triggers**
- **Re-entrant circuit**
- **Multiple re-entrant circuits**
Rational for AF ablation – elimination of triggers

- **Paroxysmal**
  - Focal Triggers

- **Persistent**
  - Recurrent circuits

- **Permanent**
  - Multiple re-entrant circuits
RUPV tachycardia in presence of SR after WACA
RUPV tachycardia in presence of SR after WACA
Rate vs rhythm

• Decision based on
  1. Symptoms, despite best drug therapy
  2. Likelihood of achieving sinus rhythm
     » LA size < 5cm
     » Duration of AF < 4 years
     » Younger age
     » Less atrial fibrosis (i.e. CMR imaging)

• Options for treatment are
  » Anti arrhythmics: amiodarone, flecainide, propafenone, Sotalol
  » AV nodal blocking: Digoxin, beta blockers, calcium antagonists
  » DCCV – even if only to assess symptoms
  » Ablation (earlier – and evolving indications)

Calkins et al. HRS Expert Consensus Statement, Heart Rhythm 2012
AFFIRM Study
Rate control versus anticoagulation in 4,000 patients

Cumulative Mortality (%) vs. Years

- Rhythm control
- Rate control

P = 0.08

<table>
<thead>
<tr>
<th>Years</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
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</thead>
<tbody>
<tr>
<td>Rhythm control</td>
<td>0</td>
<td>80 (4)</td>
<td>175 (9)</td>
<td>257 (13)</td>
<td>314 (18)</td>
<td>352 (24)</td>
</tr>
<tr>
<td>Rate control</td>
<td>0</td>
<td>78 (4)</td>
<td>148 (7)</td>
<td>210 (11)</td>
<td>275 (16)</td>
<td>306 (21)</td>
</tr>
</tbody>
</table>

Affirm NEJM 2002
• Maintenance of SR regardless of means associated with much better outcome

  – DIAMOND
    » 506 patients with heart failure & AF
    » Randomised to dofetilide vs. placebo
    » Restoration of SR regardless of means was associated with a >50% reduction in mortality (after adjusting for covariates) (RR, 0.44; 95% CI, 0.30 to 0.64; p<0.0001)

  – AFFIRM sub-study
    » Currently available AADs are not associated with improved survival, suggesting that any beneficial antiarrhythmic effects are offset by their adverse (?proarrhythmic) effects
    » Sinus rhythm and warfarin use associated with reduced mortality

Circulation 2001; 104:292-296
Circulation 2004;109:1509-1513
Anti-coagulation

- Assessment of stroke risk, independent of arrhythmia
- CHADS2 and CHADSVASc score validated.
- Options for treatment
  1. Aspirin
  2. Warfarin
  3. Newer agents (dabigatran, rivaroxaban, apixaban)
  4. Left atrial appendage closure devices.
What is the risk of CVA with AF?

• CHADS2
  - Congestive Heart Failure
  - Hypertension
  - Age >75yo
  - Diabetes
  - Previous CVA/TIA (scores 2)

• Score range 0-6

• CHADSVASc
  - Congestive Heart Failure
  - Hypertension
  - Age >75yo (scores 2)
  - Diabetes
  - Previous CVA/TIA (scores 2)
  - ADD IN 3 points
  - Sex (F scores 1)
  - Age >65 (scores 1)
  - Vascular disease (1)

Score range 0-9, being female =1

JAMA 2001
# CHADS2 score

<table>
<thead>
<tr>
<th>Score</th>
<th>Risk of Stroke Per Year</th>
</tr>
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<tbody>
<tr>
<td>0</td>
<td>1.9%</td>
</tr>
<tr>
<td>1</td>
<td>2.8%</td>
</tr>
<tr>
<td>2</td>
<td>4.0%</td>
</tr>
<tr>
<td>3</td>
<td>5.9%</td>
</tr>
<tr>
<td>4</td>
<td>8.5%</td>
</tr>
<tr>
<td>5</td>
<td>12.5%</td>
</tr>
<tr>
<td>6</td>
<td>18.2%</td>
</tr>
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</table>

Multiply CHADS2 score x2 and this is your rough estimate of annual CVA risk (unless full score = 18%). – starts at 2!

OR

2, 3, 4, 6, 8, 12, 18

JAMA 2001
Anticoagulation

- Key points
  1. Decision to anti-coagulate made on risk factors for CVA alone, not on rate/rhythm strategy
  2. Consideration of bleeding risk – often elderly who benefit from anticoagulation, also have highest risk of CVA
  3. Evolving field, with new data/strategies for minimising CVA i.e. new drugs, left atrial appendage closure
Case scenario 1

- 76 year old lady with HTN, DM, with persistent AF for 5 years and NYHA 2. Echo shows mildly impaired LV and LA size of 4.9cm. What is optimal management?

- Rate/Rhythm: unlikely to maintain SR (duration > 5 y, LA size almost 5.0cm, HTN) whatever the strategy. Likely therefore to assign “permanent AF” state. For rate control (including AV nodal ablation + pace)

- CVA risk: CHADS2 = 3 (i.e approx 6% annual CVA risk), therefore for full anticoagulation.

- Heart failure management: ACE, Beta blockers, frusemide, etc.
Case scenario 2

45 year old man with palpitations weekly, lasting up to 3 hours – unable to work during this time as highly symptomatic. Normal LV and LA 3.6cm. No underlying heart disease. ? Management?

- Rate/Rhythm : likely to maintain SR (PAF, duration unspecified, LA size normal). For rhythm control.
  - Drugs vs ablation?
    - 8 prospective randomised trials now comparing success rates
      - AAD (9-40%) vs Ablation (66-89%)
    - Improved QOL in ablation arms in all trials

CVA risk: CHADS2 = 0 (low annual CVA risk), not for anticoagulation

Calkins et al. HRS Expert Consensus Statement, Heart Rhythm 2012
Case scenario 3

- 66 yo man with persistent AF for 12 months, LA size 4.6, Normal LV. Hypertensive. Fatigued but thinks it’s “old age” catching up with him. ? Management

- Rate/Rhythm : ? likely to maintain SR (duration 12 months, LA size 4.6, large but not too large for ablation, ? symptomatic).
  - Start on AAD
  - Consider DCCV with full anticoagulation to assess symptoms
    » If symptomatic with AF, and failed AAD therapy, can have AF ablation.

- CVA risk: CHADS2 = 1 (HTN), medium risk. “Grey area”, but would opt for full anticoagulation if ablation is considered.

- CHADS2 score evolve with time: Old patients get older! / HTN / DM / HF
Holter shows VT – who to refer?

Case of 56 yo woman with HTN and obesity only

REFER?
5:56:46 VT; Number of QRS = 13; Duration = 5.34s; Mean HR = 135 min⁻¹
Management of non-sustained BCT

- **Refer** if uncertain
- If VT + syncope → will need consideration for ICD
- Fhx sudden death is relevant.
- IHD = high likelihood that this is ischaemic VT (commonest)
- If no IHD, Will need work up for Arrhythmogenic right ventricular cardiomyopathy, Brugada, Hypertrophic cardiomyopathy, Long QT syndrome, Early repolarisation syndromes
- At tertiary centre: thorough Hx, CMRI, ajmaline and adrenaline challenge, VT stimulation study, Reveal device,
Holter shows VT – who to refer?

Case of 30 yo woman with palp, but no syncope. Normal echo

REFER?
17:52:37 V bige; Number of QRS = 12; Duration = 8.82s; Mean HR = 75 min⁻¹
Other clinical cases – the HOLTER report, what does it mean?

- Echo – Normal
- VE > 10,000, but no sustained VT

**Challenging / Unanswered questions**

- What is natural progression of disease?
- What burden of ectopy is considered “high” enough to warrant therapy?
- At what stage do you start therapy?
- Drugs or ablation?
The GP refresher course
16 June 2012

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The tilt report – what does it mean?

- 32 yo lady with infrequent palpitations, followed by collapses with loss of consciousness. Sometimes feels lightheaded on standing
- Normal echo, normal 24 hour tape, normotensive
Report and management

• Vasovagal syncope with a VASIS 1 (mixed cardioinhibitory and vasodepressor pattern)

• Management is conservative first
  – Isometric counter-pressure manoeuvres
  – Increase fluids ± salt
  – Evasive action
  – Regular meals
  – Avoid caffeine
  – Pharmacotherapy 2\textsuperscript{nd} line (midodine, ivabradine, fludrocortisone, salt tablets)
  – PPM? ILR? Ablation??
The rapid access arrhythmia clinic

• Aim: To provide access to specialist arrhythmia service with full investigations to support early Dx and Mx of arrhythmias
GP referral

Assessment by arrhythmia team

Diagnosis and definitive Mx

Follow up after Rx before discharge

Initial ECG, echo and 24 hour tape

Further Ix: Reveal/tilt test/ EP study

Definitive Mx: Drugs / Ablation / Pacing / ICD
Imperial College Healthcare EP and Pacing Dept

• 6 EP and pacing consultants in academic unit

• 2 cath labs running high volume, >300 complex (AF/VT ablation), >700 total EP cases pa, and >300 pacing cases pa

• Syncope service, and clinic + tilt dept~ 600 head-up tilts pa

• Weekly outreach arrhythmia clinics in Lister + Stevenage, Teddington, Roehampton, Maida Vale, West Middlesex
  • Local clinics in St Mary’s, Charing Cross, and Hammersmith.

• Innovation, with first-in-man studies of several applications: Robotic catheter technology, cryoablation, 256 ECG jacket (ECGvue), novel multipoint electroanatomical mapping technology (Rhythmia, and Ripple mapping), syncope pacemaker algorithm development and trials (ISSUE3)
Anatomy of the conducting system - Bradycardia

- Sinus bradycardia
- Sinus arrest
- Sick sinus syndrome
- Carotid sinus hypersensitivity

- 1st degree heart block
- 2nd degree heart block
  - Wenkebach
  - Mobitz II
- 3rd degree heart block
  - Trifascicular block
Questions (for you)
**Anatomy of the conducting system - Tachycardia**

**AV Node independent:**
- Atrial fibrillation (AF)
- Atrial flutter
- Atrial tachycardia (AT)
- Sinus tachycardia
- Inappropriate sinus tachycardia
- Atrial premature beats

**AV Node dependent:**
- Atrio-ventricular nodal re-entrant tachycardia (AVNRT)
- Atrioventricular re-entrant tachycardia (AVRT)

**Ventricular**
- Ventricular tachycardia (VT)
- Ventricular fibrillation (VF)
AVNRT
AVRT
Atrial flutter
Atrial tachycardia
Question 1

- 45 yo lady with CP and palpitations
- Infrequent palpitations
AVNRT
Question 2

• 25 yo woman with palpitations since childhood
• Worse during pregnancy
• Palpitations can last from seconds to minutes
AVRT
Spectrum of pre-excitation

A. Maximal preexcitation: the ventricles are activated totally by delta force.

B. Less than maximal preexcitation, resulting in a fusion beat.

C. Minimal preexcitation; as the degree of preexcitation becomes less, the delta wave becomes smaller.

D. No preexcitation, in spite of the presence of an accessory pathway capable of conducting.

Fig. 23-6. Degrees of preexcitation. A. Maximal. B. Less than maximal. C. Minimal. D. None.
Mechanisms of AVRT

Orthodromic

Antidromic

Figure 7.9 Antidromic tachycardia with very large delta waves, preceded by period of orthodromic tachycardia and followed by sinus rhythm
Post adenosine – what is rhythm?
Q3: 35 yo with palps, terminated with adenosine
For every arrhythmia, think of...

Age of onset
Duration of symptoms
Frequency of symptoms
Mode of presentation: triggers, relieving factors
Previous medical history
Family history
Unique features
Key points from clinical history

• Age
• Symptoms
  – Asymptomatic/ Syncope/ Palpitations/ Chest pain/ Dyspnoea
• 1st time or recurrent?
• Situation
  – Anger / Fright/ Exercise/ Sleep/ Micturition
• Mode of onset
  – Gradual or rapid
• Mode of termination
  – With a valsalva/ vagal manouevres
• Drug history
  – Anti-arrhythmics/ Stimulants/ Antibiotics- consult the BNF
  – Toxicity- accidental overdose
• Family history
• History of structural heart disease
• History of previous cardiac surgery/ablation
Management of narrow complex tachy

• File rhythm strip in notes (write patient name/DOB/date on strip)

• Drugs are ineffective! But try beta blockers, CCB, flecainide.

• Most arrhythmias should be referred on to an EP centre
  - High success rates for ablation for Aflutter, A tach, AVNRT, AVRT (95+ %)
  - Lower rates for AF (50-80%)

Take home messages

1. Symptomatic bradycardia = pacing indication
2. Tachycardias can be diagnosed in most cases from history + ECG
3. Most SVTs should be considered for ablation if refractory to drug therapy
4. AF Management = 1) Rate vs Rhythm and 2) CHADS-2
Managing palpitations
The GP refresher course
11 May 2012

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Diagnosis and management of Broad Complex Tachycardia
Differential diagnosis of BCT

• VT

• SVT + aberrant conduction

• Antidromic AV reentrant tachycardia

• VF

• Any pre-excited tachycardia (i.e. an accessory pathway in presence of atrial tachycardia/flutter/AF).
Differentiating VT vs SVT with aberration

• ECG criteria is difficult to remember at 2am with unwell patient – and mostly non-absolute in making diagnosis.

• If uncertain, treat as VT

• Key features
  – Ischaemic heart disease (assume VT always)
  – Structural heart disease
  – Sympathomimetics
  – Beware electrolytes.
  – Known history of VT
Differentiating VT vs SVT with aberration (for medical finals)

- Fusion/capture beats
- Dissociated P waves
- Broad QRS (>140ms)
- Concordance
- Extreme axis deviation
Differentiating VT vs SVT with aberration (for 2am in ER!)

• Does it look like a typical RBBB or LBBB?
Normal QRS complex – observe onset QRS
Typical LBBB – observe the onset of QRS
VT or SVT?
VT or SVT?
VT or SVT?
VT or SVT?
VT or SVT?
VT or SVT?
VT or SVT?
Acute management of BCT

• Haemodynamic instability – DCCV
• If not unstable, time to think
  – Accurate/relevant history/ drugs
  – Check electrolytes (K/Mg)
  – Attempt to make accurate diagnosis – 12 lead ECG
  – Acute ischaemia (primary PCI call ? Thrombolysis)
• Pharmacology (VT)
  – Magnesium/ K+ correction
  – Amiodarone
  – Beta blockers
  – Lignocaine
  – Procainamide
• Non pharmacology
  – Overdrive pacing
  – DCCV
Chronic management of VT
Why drugs have a bad name in VT?

Encainide and flecainide increase cardiac mortality  Results of the Cardiac Arrhythmia Suppression Trial (CAST) in patients with ventricular premature beats after myocardial infarction. Patients receiving encainide or flecainide had, when compared to those receiving placebo, a significantly lower rate of avoiding a cardiac event (death or resuscitated cardiac arrest) (left panel, $p = 0.001$) and a lower overall survival (right panel, $p = 0.0006$). The cause of death was arrhythmia or cardiac arrest. (Data from Echt, DS, Liebson, PR, Mitchell, B, et al, N Engl J Med 1991; 324:781.)
Drugs don’t prevent SCD

Bardy, NEJM 2005

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Hazard Ratio (97.5% CI)</th>
<th>P Value</th>
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</thead>
<tbody>
<tr>
<td>Amiodarone vs. placebo</td>
<td>1.06 (0.86–1.30)</td>
<td>0.53</td>
</tr>
<tr>
<td>ICD therapy vs. placebo</td>
<td>0.77 (0.62–0.96)</td>
<td>0.007</td>
</tr>
</tbody>
</table>

Mortality Rate

- Placebo: (244 deaths; 5-yr event rate, 0.361)
- Amiodarone: (240 deaths; 5-yr event rate, 0.340)
- ICD therapy: (182 deaths; 5-yr event rate, 0.289)

Bardy, NEJM 2005
Only ICD saves lives!

<table>
<thead>
<tr>
<th>Trial Name, Pub Year</th>
<th>Hazard ratio</th>
<th>LVEF, other features</th>
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<tbody>
<tr>
<td>MADIT-I (1996)</td>
<td>0.46</td>
<td>0.35 or less, NSVT, EP positive</td>
</tr>
<tr>
<td>AVID (1997)</td>
<td>0.62</td>
<td>Aborted cardiac arrest</td>
</tr>
<tr>
<td>CABG-Patch (1997)</td>
<td>0.82</td>
<td>Aborted cardiac arrest</td>
</tr>
<tr>
<td>CASH* (2000)</td>
<td>0.83</td>
<td>Aborted cardiac arrest or syncope</td>
</tr>
<tr>
<td>CIDS (2000)</td>
<td>0.69</td>
<td>0.30 or less, prior MI</td>
</tr>
<tr>
<td>MADIT-II (2002)</td>
<td>0.65</td>
<td>0.35 or less, NICM and PVCs or NSVT</td>
</tr>
<tr>
<td>DEFINITE (2004)</td>
<td>0.77</td>
<td>0.35 or less, MI within 6 to 40 days and impaired cardiac autonomic function</td>
</tr>
<tr>
<td>DINAMIT (2004)</td>
<td></td>
<td>0.35 or less, LVD due to prior MI and NICM</td>
</tr>
<tr>
<td>SCD-HeFT (2005)</td>
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Future challenges

Figure 1. The Incidence of Sudden Death in Specific Populations and the Annual Numbers of Sudden Deaths in Those Populations. Most of the deaths occur in the larger, lower-risk subgroups. Modified from Myorburg et al.10 with the permission of the publisher.
Atrial Flutter

• A macro re-entrant arrhythmia
  – Anatomical barrier
  – Zone of slow conduction

• Typical atrial flutter
  – Contained within the right atrium
  – Constrained anteriorly by the tricuspid valve
  – Constrained posteriorly by the crista terminalis and eustachian ridge
  – Travels in a counterclockwise direction around the atrium

• Atypical atrial flutters
  – Counterclockwise flutter
  – ASD/ scar related flutter
  – Perimitral flutter
Atrial flutter
Atrial Flutter

• Tends to occur in middle age
  – Probably due to atrial dilatation

• Pulmonary embolism
  – Commonly presents with a sinus tachycardia

• Associated valve disease
  – Mitral or Tricuspid disease
  – Atrial septal defects
  – Chronic ventricular failure

• Toxic and Metabolic conditions
  – Alcohol/ thyrotoxicosis/ pericarditis

• Previous ablation
Atrial Flutter

Examination

• Rarely helpful in establishing the diagnosis
• Regular pulse (150bpm- 2:1, 75bpm 4:1- can be slower)
• May see rapid, regular flutter waves in the JVP
• Heart sounds
  – Constant intensity of S1 if relationship of flutter waves to QRS is constant
• Carotid massage or adenosine
  – Allows flutter waves to be seen more easily
  – Ventricular rate will increase when CSM is stopped
Atrial Flutter
AVNRT

• Commonest supraventricular arrhythmia
  – ie dependent upon the AV node
  – AV Node with Dual (or more) physiology: Fast and slow pathway
AVNRT

• Typically 3rd and 4th Decade
• Recurrent palpitations
• RAPID onset and RAPID offset
• Patient may feel an ectopic beat to initiate/ terminate the arrhythmia
• Vagal manoeuvres to terminate the arrhythmia
• Anxiety/ breathless/ palpitations
  – Syncope (due to high rate or due to transient asystole at termination)
AVRT

• Due to accessory pathway – abnormal electrical AV conduit
  – Patients can have multiple pathways

• Accessory pathways may conduct
  – Antegradely
  – Retrogradely
  – Combination of the two

• Wolf- Parkinson -White Syndrome
  – Short PR interval (<120ms)
  – Delta wave
  – Palpitations and narrow complex tachycardia
AVRT
Definitions

• Orthodromic
  – Conduction travels in the normal direction (ie A to V)

• Antidromic
  – Conduction travels in an abnormal direction (ie V to A)
AVRT

Presentation
• Young patient typically 3rd to 4th decade
• May be asymptomatic- part of a medical
• RAPID onset and RAPID offset
• Patient may feel an ectopic beat to initiate/ terminate the arrhythmia
• Vagal manoeuvres to terminate the arrhythmia
• Anxiety/ breathless/ palpitations
  – Syncope (due to high rate or due to transient asystole at termination)
AVRT

• History of structural heart disease
  – Ebstein’s anomaly
    » Multiple right sided accessory pathways

• Family history
  – Higher prevalence in the children; especially if multiple accessory pathways

• Examination
  – Frequently normal
AVRT
Focal Atrial Tachycardia

- Typically older patients >6th decade
- Frequently have structural heart disease, pulmonary disease
- Symptoms are related to:
  - Rate (120-250bpm)
  - Underlying heart disease
- Rapid initiation
  - Rate can increase over a few beats as the AV node “warms up”
- No consistent effect with vagal manoeuvres
- Digoxin / Alcohol/ Lung disease/ Metabolic derangements
Atrial tachycardia
Focal Atrial Tachycardia

- Regular pulse
  - Exceptions
    - If atrial tachycardia is fast the AV node may Wenckebach (Mobitz Type I)
    - If more than one focus (Multifocal atrial tachycardia)
- Check for signs of pulmonary disease
- Cannon A waves
- Variable S1
Atrial Tachyarrhythmias - review

AV Node dependent:
» Atrio-ventricular nodal re-entrant tachycardia (AVNRT)
» Atrioventricular re-entrant tachycardia (AVRT)

AV Node independent:
» Atrial fibrillation (AF)
» Atrial flutter
» Atrial tachycardia (AT)
» Sinus tachycardia
» Inappropriate sinus tachycardia
» Atrial premature beats
Ventricular Tachycardia

• May be asymptomatic
• Heart rate is NOT a useful guide to the arrhythmia
• More likely if
  – Previous MI / History of IHD
  – Cardiac risk factors
• Sudden onset/ offset
• Is it recurrent?
• Do they have a pacemaker or an ICD
• Family History
  – Sudden cardiac death
  – Unexplained death
  – HOCM/ Long QT syndrome / Brugada
Physical Examination

• Is the patient compromised?
  – DC cardioversion if any doubt

• Assess the JVP
  – Cannon A waves?

• Assess the praecordium
  – Pacemaker/ICD/ Median sternotomy scar / LV Heave/ Double apical impulse?

• Ausculate
  – Variable S1; Ejection systolic murmer
ECG Findings- VT

• Regular broad complex tachycardia (QRS > 120ms)
  – Normally RBBB >140ms
  – LBBB>160ms
• Evidence of A-V Dyssynchrony
• Fusion beats
• Capture beats
• Concordance
• If a 12 lead in sinus rhythm is available
  – ?Q waves; Delta waves; RBBB and ST Elevation
Ventricular Tachycardia
Ventricular Tachycardia
Right Ventricular Outflow Tract Tachycardia (RVOT VT)

• Young patients
• Athletic
• Occur during exercise
• Can be terminated by vagal manoeuvres

• ECG Findings
  – LBBB morphology in V1
  – Inferior axis
Ventricular Fibrillation

• No cardiac output
  – DC Cardioversion

• Normally cause is evident
  – Myocardial ischaemia
  – Cardiomyopathy - DCM/ HCM/ HOCM
  – Torsade de pointes and causes of long QT syndrome
  – Brugada syndrome
  – Commotio Cordis
Brugada Syndrome

- Due to a mutation in a sodium channel (SCN5A)
- 1st presentation may be failed sudden cardiac death
- Family history
- ECG
  - Right bundle branch block
  - ST elevation in the anterior precordial chest leads (V1-3)
- No evidence of structural heart disease
Brugada Syndrome
Summary

• The arrhythmia must be seen in the context of the patient
  – Not just the ECG

• The state of the patient will depend on the heart rate and underlying heart disease *not* the arrhythmia per se

• The age of the patient, and associated disease can guide the provisional differential diagnosis before seeing the ECG

• Examine for signs of AV dissociation