ICU arrhythmia: diagnosis and management.

WFSICCM
Seoul, South Korea
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Sydney, Australia
Rapid classification:

- slow
- narrow complex
- known cardiac pathology
- PRQRST configuration normal

- rapid
- broad complex
- no known cardiac pathology
- abnormal
Bradycardia  or  Tachycardia

Atrial tachycardia with 2:1 AV block

Type 1 sino-atrial block

Critical Care physician faced with hemodynamically unstable patient - should I speed the rate up or do I slow it down  ? ? ?
Bradycardia  or  Tachycardia

The decision may be easy

- cardioversion
- temporary pacemaker
• Distinguishing between narrow complex tachycardias
• Distinguishing between wide complex tachycardias
The average direction of the QRS vector through the ventricles as seen from the frontal plane leads
Calculating the axis

-30 to +90 is normal

- Determine which lead has the smallest algebraic sum of the QRS deflections
- The axis will be at right angles to this lead- look at both leads that lie at 90°
- Return to the first lead- is it slightly positive or negative?
  - If it is positive, the axis must point slightly toward this lead by 15° (eg from +90° to +75°)
  - If it is negative, the axis will point slightly away (eg from +90° to 105°)
- **RAD**
  - +90 to +180
  - Lead I negative
  - Lead III positive
  - Mainly pulmonary conditions
  - LPHB is rare due to dual blood supply

- **LAD**
  - -30 to -90
  - Not significant till predominantly negative deflection in II
  - Normally a conduction defect
  - LAHB
  - Can be indicative of myocardial ischaemia
Rapid classification:

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Diagnosis of Narrow QRS Complex Tachycardia (rate > 100/min, QRS < 0.10 sec)

- The commonest supraventricular tachycardia is sinus tachycardia
- Are flutter waves visible? *Atrial flutter*
- Is there absence of consistent P waves? *Atrial fibrillation*
Diagnosis of Narrow QRS Complex Tachycardia continued

Is there a regular atrial rate 180-240 bpm with twice as many P waves as QRS complexes (2:1 AV block)? *Atrial tachycardia with 2:1 AV block*

Is there a regular ventricular rate 180-240 bpm with no P waves visible? *Probably AV nodal re-entrant tachycardia*

Is there a regular ventricular rate 180-240/min with one P wave for every QRS? If so, is the R-P’ interval equal to the P’-R interval? *Possibly atrial tachycardia with 2:1 AV block (alternate P waves in the QRS)*
Broad Complex Tachyarrhythmia
<table>
<thead>
<tr>
<th>Evaluation</th>
<th>Assessment</th>
<th>Goal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vital signs</td>
<td>Hemodynamic stability</td>
<td>If hemodynamically unstable, treat with urgent DCCV or defibrillation</td>
</tr>
<tr>
<td>12-lead ECG</td>
<td>Tachycardia diagnosis</td>
<td>Differentiate VT from SVT with aberrancy; determine VT exit site</td>
</tr>
<tr>
<td>History</td>
<td>Symptoms (e.g., chest pain indicating ongoing ischemia)</td>
<td>Identify cause and triggers</td>
</tr>
<tr>
<td>Current medications</td>
<td>Antiarrhythmics, digoxin, QTC-prolonging medications</td>
<td>Identify pharmacologic contribution to a proarrhythmic state</td>
</tr>
<tr>
<td>Family history</td>
<td>Family history of SCD</td>
<td>Determine risk of inherited predisposition to SCD</td>
</tr>
<tr>
<td>Physical examination</td>
<td>Canon A waves, murmurs, sternotomy scar</td>
<td>Indicate AV dissociation, indicate existing structural heart disease</td>
</tr>
<tr>
<td>Laboratory tests</td>
<td>Electrolytes, creatinine, troponin, thyroid-stimulating hormone, toxicology assays</td>
<td>Identify metabolic, ischemic, or pharmacologic contributions to a proarrhythmic state</td>
</tr>
<tr>
<td>Imaging</td>
<td>Chest roentgenography, echocardiography</td>
<td>Indicated in all patients with VT to assess for structural heart disease</td>
</tr>
<tr>
<td></td>
<td>Coronary angiography</td>
<td>Indicated if VT occurs secondary to ischemia</td>
</tr>
<tr>
<td></td>
<td>Computed tomography, magnetic resonance imaging</td>
<td>Indicated in special cases when particular cardiomyopathies are suspected</td>
</tr>
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**Abbreviations:** A, atrial; AV, atrioventricular; DCCV, direct current cardioversion; QTC, corrected QT interval.
Wide QRS Tachycardia

Regular Monomorphic Wide QRS

Features Suggestive of VT
- AV dissociation or fusion beats
- QRS >140 ms (RBBB), >160 ms (LBBB)
- Extreme right axis deviation (180°–270°)
- LBBB with right axis deviation
- Absence of RS in precordial leads
- RS interval >100 ms
- Prior sternotomy or MI
- QRS morphology different from preexistent bundle branch block

If Diagnosis Is Still Unclear

RBBB Pattern
- Amplitude R > R' in V1
- QS or RS (S > R) in V6
- Initial deflection in V1 different from sinus rhythm

LBBB Pattern
- R wave in V1 >30 ms
- qR or QS in V6
- R to S wave nadir >60 ms
Diagnosis of wide QRS complex tachycardia

Capture beats found
Ventricular tachycardia

Fusion beats found
Ventricular tachycardia

QRS rate consistently greater than the P wave rate
Ventricular tachycardia
Diagnosis of wide QRS complex tachycardia

QRS duration > 0.16 sec

Ventricular tachycardia
Features that suggest VT

- P waves recognized with atrio-ventricular dissociation
- QRS > 0.16 sec
- Frontal plane QRS axis within the range -30° to -120° (ie superior axis)
- Comparison with previous records: R (tachycardia) > r (sinus rhythm)
Features that suggest VT

Shape of the QRS in $V_6$
Management of VT

- Correct electrolytes – $K^+ > 4.5$ mmo/l
  $Mg^{++} > 1.1$ mmol/l
- Electrical cardioversion
- Overdrive pacing
- Drugs

### Table 2

Acute and maintenance dosing of intravenous antiarrhythmic medications

<table>
<thead>
<tr>
<th>Antiarrhythmic</th>
<th>Dosing</th>
<th>Acute Adverse Reactions</th>
</tr>
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</table>
| Procainamide     | Load: 17 mg/kg                               | Hypotension
                 | Maximum rate: 50 mg/min                          | Hold if QRS prolongs $>50\%$ |
                 | Maintenance: 1–4 mg/min                       | Reduce dose in heart failure                               |
|                  |                                              | Monitor for neurotoxicity: delirium, seizures, or paresthesias|
| Lidocaine        | Load: 1–3 mg/kg                               |                                                            |
                 | Rate: 20–50 mg/min                            |                                                            |
                 | Maintenance: 1–4 mg/min                       |                                                            |
| Amiodarone       | Load: 150 mg over 10 min if blood pressure is normal; 300 mg over 19 min if hypotensive | Caution in cardiogenic shock TdP is rare Use with pacing if patient is severely bradycardic |
Not all VT is the same
Torsade de pointe

Vasospasm secondary to thyrotoxicosis
Torsade de Pointe

- Polymorphic VT in setting of prolonged QTc
- Secondary to electrolyte abnormalities, bradyarrhythmias, medications
- Correct potassium and magnesium levels
- Defibrillate, can then pace to give rate faster than intrinsic rate
- Discontinue drugs that prolong QT – be wary of regular antiarrhythmics
- Magnesium infusion
## Rapid classification:

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Initiation of arrhythmias
In acute coronary syndrome

Cardiac arrhythmias in ACS
Europace 2014;16:1655
<table>
<thead>
<tr>
<th>Occurrence of arrhythmias in STEMI patients during and immediately after primary PCI $^{71}$</th>
</tr>
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<tbody>
<tr>
<td>Accelerated idioventricular rhythm (50–120 b.p.m.)</td>
</tr>
<tr>
<td>Sinus bradycardia (&lt;50 b.p.m.)</td>
</tr>
<tr>
<td>Non-sustained VT</td>
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<tr>
<td>Sinus tachycardia (&gt;100 b.p.m.)</td>
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<tr>
<td>Atrial fibrillation</td>
</tr>
<tr>
<td>High-degree AV block</td>
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<tr>
<td>Sustained VT</td>
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<tr>
<td>VF</td>
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Antipsychotic drugs and QTC prolongation

Table 3  Atypical antipsychotics and FDA warnings regarding QT prolongation

<table>
<thead>
<tr>
<th>FDA warning</th>
<th>No FDA warning</th>
</tr>
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<tbody>
<tr>
<td>Asenapine</td>
<td>Aripiprazole</td>
</tr>
<tr>
<td>Clozapine</td>
<td>Lurasidone</td>
</tr>
<tr>
<td>Iloperidone</td>
<td>Olanzapine</td>
</tr>
<tr>
<td>Paliperidone</td>
<td>Risperidone*</td>
</tr>
<tr>
<td>Quetiapine</td>
<td>*Warning for risperidone was removed from label in 2003.</td>
</tr>
<tr>
<td>Ziprasidone</td>
<td></td>
</tr>
</tbody>
</table>

QTc Prolongation with Antipsychotics:  
Is Routine ECG Monitoring Recommended?
Rhythm restoration or control

In patients with advanced HF, the Antiarrhythmic Trial with Dronedarone in Moderate to Severe CHF Evaluating Morbidity Decrease (ANDROMEDA) found a 2-fold excess in mortality with dronedarone compared with placebo, primarily because of worsening HF, prompting the early termination of this study after just 2 months of clinical follow-up. 48

The cornerstone of antiarrhythmic therapy in patients with HF is the use of amiodarone, sotalol, and dofetilide. Amiodarone, a class III agent with some overlap activity of class I, is among the most effective antiarrhythmic agents for suppression of AF, 49 and seems to be safe and effective in patients with HF. Despite its effectiveness, the use of amiodarone in patients with HF is associated with an increased risk for symptomatic bradycardia requiring implantation of a permanent pacemaker. 50
Non ventricular arrhythmias
Sinus rhythm with atrial premature beats
Sinus rhythm with 2 uniform VEB

Sinus bradycardia

Sinus bradycardia with 1° heart block
Sinus rhythm with atrial ectopics, one of which initiates AF

Complete heart block, with 2 ventricular escape foci

2nd degree AV block
Complete heart block

Sinus bradycardia with transient sino-atrial block

Sinus rhythm with burst of VT
Atrial fibrillation (AF)

- Most common form of sustained heart arrhythmia\(^1\)
- Characterised by\(^2\)
  - Uncoordinated activation of the atria
  - Consequent decline of the mechanical function of the atria

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\(^1\) Camm et al *Eur Heart J* 2010;31:2369–429; \(^2\) Fuster et al *Circulation* 2011;123:e269–357.
Atrial fibrillation (AF)

- Many risk factors, eg,¹
  - Ischaemic heart disease
  - Hypertension
  - Obesity
  - Excessive alcohol ingestion
  - Sleep apnoea

- Up to 90% is nonvalvular AF¹
  - Occurs without associated valvular heart disease²
  - Defined as absence of significant mitral stenosis or prosthetic valves.

AF is common among older patients

Age 75–79 years

Estimated prevalence
~1 in 10 (9.6%)

Age 80–84 years

Estimated prevalence
~1 in 7 (13.8%)

Age ≥85 years

Estimated prevalence
~1 in 5.6 (17.6%)

61% of people with AF are aged ≥75 years

Atrial Fibrillation treatment

treatment objective defined – rhythm or rate control
- acute or chronic setting
- role of anticoagulation
  TEE or no TEE

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Drugs commonly used for rate and rhythm control in AF and HF</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Loading dose</td>
</tr>
<tr>
<td>Rate control</td>
<td></td>
</tr>
<tr>
<td>First-line treatment: β-blockers with demonstrated survival benefit</td>
<td></td>
</tr>
<tr>
<td>Carvedilol</td>
<td>–</td>
</tr>
<tr>
<td>Bisoprolol</td>
<td>–</td>
</tr>
<tr>
<td>Metoprolol Succinate</td>
<td>–</td>
</tr>
<tr>
<td>Second-line treatment: Digoxin</td>
<td></td>
</tr>
<tr>
<td>Digoxin</td>
<td>1 mg in divided doses</td>
</tr>
<tr>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Rhythm control</td>
<td></td>
</tr>
<tr>
<td>Amiodarone</td>
<td>10 g over several weeks</td>
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<td></td>
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<td></td>
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</tbody>
</table>
Acute therapy for atrial fibrillation

• Observe

• Drug therapy
  - digoxin
  - β blocker - metoprolol, sotalol
  - flecainide
  - verapamil
  - amiodarone

• Electrical cardioversion
Look beyond the arrhythmia!

Past medical history
Drugs
Electrolytes
Endocrine abnormalities
Temperature
........
The End