Arrhythmia or Dysrhythmia

• An arrhythmia (ah-RITH-me-ah) is a problem with the rate or rhythm of the heartbeat. During an arrhythmia, the heart can beat too fast, too slow, or with an irregular rhythm.

• Mostly classified according to the origin of the arrhythmia
  – Sinus arrhythmia
  – Atrial arrhythmia
  – Ventricular arrhythmia
  – Junctional arrhythmia
Sinus arrhythmia

- Rate: varies
- Most of time all the ECG components are normal
- Common in infants and respiratory variation, also called respiratory sinus arrhythmia, HR ↓ with expiration and ↑ inspiration
- No treatment is required
Sinus Arrest

- Rate: varies often less than 60 bpm
- Occasionally noted in elderly patients
- Causes: cardiomyopathy, MI
- No treatment is required, pace maker may be considered in the deteriorated cases
Sinus Bradycardia

- Hypothermia, hypothyroidism
- Increased vagal tone (due to vagal stimulation or e.g. drugs);
- Beta blockers, digoxin, Ca channel blockers;
- Marked intracranial hypertension;
- Treatment of underlying cause
- Atropine, Isuprel can be used
- Pacemaker may be needed
Sinus Tachycardia

- Thyrotoxicosis; fever
- Any cause of adrenergic stimulations (e.g. pain, anxiety)
- Hypovolaemia; anaemia, pregnancy;
- Vagolytic drugs (e.g. atropine)
- Vasodilator drugs, including many hypotensive agents;
- Treatment of underlying cause
- Beta and Ca channel blockers may be used
Atrial Flutter

- **Atrial flutter** is a type of supraventricular tachycardia caused by a re-entry circuit within the right atrium.
- **Rate**: atrial rate 250 – 350 bpm, ventricular rate varies according to atrioventricular (AV) block ratio; 2:1, 3:1, 4:1
- **Rhythm**: usually regular but can be irregular
- **Classification**:
  - Typical atrial flutter
Atrial Flutter

Classification:

• **Typical atrial flutter**: The reentrant loop circles the right atrium, passing through the cavo-tricuspid isthmus - a body of fibrous tissue in the lower atrium between the inferior vena cava, and the tricuspid valve
  – **Clockwise atrial flutter**: The flutter waves in this rhythm are upright in ECG leads II, III, & aVF
  – **Counterclockwise atrial flutter**: The flutter waves in this rhythm are inverted in ECG leads II, III, & aVF.

• **Atypical Atrial flutter**:
  – Less common
  – Does not fulfil criteria for typical atrial flutter.
  – Often associated with higher atrial rates and rhythm instability.
  – May be associated with left atrial flutter
Atrial Flutter: ECG examples

Atrial Flutter with 2:1 Block
- There are inverted flutter waves in II, III + aVF at a rate of 300 bpm (one per big square)
- There are upright flutter waves in V1 simulating P waves
- There is a 2:1 AV block resulting in a ventricular rate of 150 bpm
**Atrial Flutter with Variable Block**

- Inverted flutter waves in II, III + aVF with atrial rate ~ 300 bpm
- Positive flutter waves in V1 resembling P waves
- The degree of AV block varies from 2:1 to 4:1
Counterclockwise atrial flutter. The flutter waves in this rhythm are inverted in ECG leads II, III, and aVF.
Clockwise atrial flutter: flutter waves are upright in II, III, and aVF.
Vagal Maneuvers

- **Valsalva maneuver** is performed by forcibly exhaling against a closed glottis (a closed airway).
- **Carotid sinus massage**
Atrial Flutter

- **Causes:** CAD, cardiomyopathy, COPD, PE
- **Treatment:** Adenosine, Diltiazem, Verapamil, Digoxin, Warfarin
- **Cardioversion** is considered in emergency
- **Ablation**
Atrial Fibrillation

- Atrial Fibrillation (AF) is the most common sustained arrhythmia.
- The incidence and prevalence of AF is increasing.
- Lifetime risk over the age of 40 years is ~25%.
- Complications of AF include hemodynamic instability, cardiomyopathy, cardiac failure, and embolic events such as stroke.
- Characterized by disorganized atrial electrical activity and contraction.

Mechanism of Atrial Fibrillation

- The mechanisms underlying AF are not fully understood but it requires an initiating event (focal atrial activity / PACs) and substrate for maintenance (i.e. dilated left atrium).
Atrial Fibrillation

Causes of Atrial Fibrillation

- Ischemic heart disease
- Hypertension
- Valvular heart disease (esp. mitral stenosis / regurgitation)
- Acute infections
- Electrolyte disturbance (hypokalemia, hypomagnesaemia)
- Thyrotoxicosis
- Drugs (e.g. sympathomimetics)
- Pulmonary embolus
- Pericardial disease
- Acid-base disturbance
- Pre-excitation syndromes
- Cardiomyopathies: dilated, hypertrophic.
- Phaeochromocytoma
Atrial Fibrillation

ECG Features of Atrial Fibrillation

- Irregularly irregular rhythm.
- No P waves.
- Absence of an isoelectric baseline.
- Variable ventricular rate.
- QRS complexes usually $< 120$ ms unless pre-existing bundle branch block, accessory pathway, or rate related aberrant conduction.
- Fibrillatory waves may be present and can be either fine (amplitude $< 0.5$mm) or coarse (amplitude $> 0.5$mm).
- Fibrillatory waves may mimic P waves leading to misdiagnosis.
Atrial Fibrillation

Classification of Atrial Fibrillation

- First episode – initial detection of AF regardless of symptoms or duration
- Recurrent AF – More than 2 episodes of AF
- Paroxysmal AF – Self terminating episode < 7 days
- Persistent AF – Not self terminating, duration > 7 days
- Long-standing persistent AF – > 1 year
- Permanent (Accepted) AF – Duration > 1 yr in which rhythm control interventions are not pursued or are unsuccessful
Atrial Fibrillation

Normal Conduction

Atrial Fibrillation
AF with rapid ventricular response

- Irregular narrow-complex tachycardia at ~135 bpm.
- Coarse fibrillatory waves in V1.
AF with slow ventricular response

- Irregular heart rate with no evidence of organized atrial activity.
- Fine fibrillatory waves in V1.
- The slow ventricular rate suggests that the patient is being treated with an AV-nodal blocking drug (e.g. beta-blocker, verapamil/diltiazem, digoxin). Another cause of slow AF is hypothermia.
Paroxysmal Atrial Tachycardia

- **Supraventricular tachycardia (SVT)** arises from improper electrical activity of the heart which presents as a rapid heart rhythm originating at or above the atrioventricular node.

- **Two common types:**
  1. Atrioventricular nodal reentrant tachycardia
     - Occurs when a reentry circuit forms within or just next to the atrioventricular node
     - It is more common in women than men (approximately 75% of cases occur in females)
     - In yellow, is evidenced the P wave that falls after the QRS complex.
Paroxysmal Atrial Tachycardia

- **Two common types:**
  - II. Atrioventricular reciprocating tachycardia
    - an accessory pathway allows electrical signal from the ventricles to enter the atria and cause premature contraction and repeat stimulation of the atrioventricular node

  - Regular, narrow complex tachycardia at 225 bpm.
  - No discernible P-waves.
  - The QRS complexes are narrow because impulses are being transmitted in an orthodromic direction (A -> V) via the AV node.
Paroxysmal Atrial Tachycardia

- Rate: atrial rate between 150 – 250 BPM
- Rhythm: regular to irregular
- Ectopic P wave with very short PR interval
- Causes: cardiac disease, caffeine, nicotine, & alcohol
- Treatment of the underlying causes
- Vagal stimulation, adenosine can be used
Reentry Phenomenon

- **AV nodal reentrant - Atrioventricular reentrant**
  - Re-entry dysrhythmias occur when an electrical impulse recurrently travels in a tight circle within the heart, rather than moving from one end of the heart to the other and then stopping.
Adenosine Treatment
First-degree AV block

- Prolongation of AV conduction > 200ms (five small squares)
- Marked’ first degree block if PR interval > 300ms
- Rate: 60 -100 bpm
- Rhythm: regular with constant prolonged PR interval

Causes:
- Increased vagal tone
- Athletic training
- Inferior MI
- Mitral valve surgery
- Myocarditis (e.g. Lyme disease)
- Hypokalemia
- AV nodal blocking drugs (beta-blockers, calcium channel blockers, digoxin, amiodarone)
- May be a normal variant

Treatment: treat the underlying cause
**First-degree AV block**

- Prolongation of AV conduction
- Rate: 60 - 100 bpm
- Rhythm: regular with constant prolonged PR interval
- Causes: CAD, MI, myocarditis

<table>
<thead>
<tr>
<th>P Wave</th>
<th>PR Interval (in seconds)</th>
<th>QRS (in seconds)</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before each QRS, identical</td>
<td>.20</td>
<td>.12</td>
<td>Regular rhythm</td>
</tr>
</tbody>
</table>
Second-degree AV block, Mobitz type I
(Wenckebach Phenomenon)

- Progressive prolongation of the PR interval culminating in a non-conducted P wave
- The PR interval is longest immediately before the dropped beat
- The PR interval is shortest immediately after the dropped beat
- The Wenckebach pattern tends to repeat in P:QRS groups with ratios of 3:2, 4:3 or 5:4.
- Rate: 60 -100 bpm
- Rhythm: regular atrial, irregular ventricular

Causes:
- Drugs: beta-blockers, calcium channel blockers, digoxin, amiodarone
- Increased vagal tone (e.g. athletes)
- Inferior MI
- Myocarditis
- Following cardiac surgery (mitral valve repair, Tetralogy of Fallot repair)

- **Treatment:** treat the underlying cause
- Consider atropine, temporary pace-maker
Second-degree AV block, Mobitz type I
Second-degree AV block, Mobitz type II

- Intermittent non-conducted P waves *without* progressive prolongation of the PR interval.
- The PR interval in the conducted beats remains constant.
- The P waves ‘march through’ at a constant rate.
- The RR interval surrounding the dropped beat(s) is an exact multiple of the preceding RR interval (e.g. double the preceding RR interval for a single dropped beat, treble for two dropped beats, etc).
- Mobitz II is usually due to failure of conduction at the level of the His-Purkinje system (i.e. below the AV node).
- In around 75% of cases, the conduction block is located *distal to the Bundle of His*, producing *broad QRS complexes.*
Second-degree AV block, Mobitz type II

• **Causes:**
  - Anterior MI (due to septal infarction with necrosis of the bundle branches).
  - Idiopathic fibrosis of the conducting system (Lenegre’s or Lev’s disease).
  - Cardiac surgery (especially surgery occurring close to the septum, e.g. mitral valve repair).
  - Inflammatory conditions (rheumatic fever, myocarditis, Lyme disease).
  - Autoimmune (SLE, systemic sclerosis).
  - Infiltrative myocardial disease (amyloidosis, hemochromatosis, sarcoidosis).
  - Hyperkalemia.
  - Drugs: beta-blockers, calcium channel blockers, digoxin, amiodarone.

• **Treatment:**
  - Treat the underlying cause
  - Consider atropine, isoproterenol, temporary pace-maker
Third-degree AV block or Complete Heart Block

- Rate: 60 - 100 bpm
- Rhythm: regular atrial & ventricular but independent of each other
- QRS: normal if originated from the AV junction or wide if originated from the ventricles
- Causes: MI, CAD, CHF and as progressive deterioration of Mobitz type II, B-Blocker, Ca channel blocker, & digoxin
- Treatment: treat the underlying cause
- Consider atropine, isoproterenol, temporary or permanent pace-maker
Third-degree AV block or Complete Heart Block

- In complete heart block, there is complete absence of AV conduction - *none* of the supraventricular impulses are conducted to the ventricles.
- Perfusing rhythm is maintained by a junctional or ventricular escape rhythm.
- Alternatively, the patient may suffer ventricular standstill leading to syncope (if self-terminating) or sudden cardiac death (if prolonged).
- Typically the patient will have severe bradycardia with independent atrial and ventricular rates, i.e. AV dissociation.
- Complete heart block is essentially the end point of either Mobitz I or Mobitz II AV block.
• Atrial rate is ~ 60 bpm.
• Ventricular rate is ~ 27 bpm.
• None of the atrial impulses appear to be conducted to the ventricles.
• There is a slow ventricular escape rhythm.
Junctional Rhythm

### Junctional Rhythm

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Rhythm</th>
<th>P Wave</th>
<th>PR interval (in seconds)</th>
<th>QRS (in seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>40-60 bpm</td>
<td>Regular</td>
<td>Inverted, absent or after QRS</td>
<td>&lt;.12</td>
<td>&lt;.12</td>
</tr>
</tbody>
</table>

Lead II
Accelerated Junctional Rhythm

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Rhythm</th>
<th>P Wave</th>
<th>PR interval (in seconds)</th>
<th>QRS (in seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>60-100 bpm</td>
<td>Regular</td>
<td>Inverted, absent or after QRS</td>
<td>&lt;.12</td>
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Rt Bundle Branch Block

RBBB

- In RBBB, activation of the right ventricle is delayed as depolarization has to spread across the septum from the left ventricle.
- The left ventricle is activated normally, meaning that the early part of the QRS complex is unchanged.
- The delayed right ventricular activation produces a secondary R wave (R’) in the right precordial leads (V1-3) and a wide, slurred S wave in the lateral leads.
- Delayed activation of the right ventricle also gives rise to secondary repolarization abnormalities, with ST depression and T wave inversion in the right precordial leads.

Tall R’ wave in V1 ("M" pattern) with wide, slurred S wave in V6 ("W" pattern)
Rt Bundle Branch Block

Diagnostic Criteria of RBBB

- Broad QRS > 120 ms
- RSR' pattern in V1-3 (‘M-shaped’ QRS complex)
- Wide, slurred S wave in the lateral leads (I, aVL, V5-6)

Typical RSR' pattern ('M'-shaped QRS) in V1

Wide slurred S wave in lead I
What do you see???
Lt Bundle Branch Block

LBBB

- Normally the septum is activated from left to right, producing small Q waves in the lateral leads.
- In LBBB, the normal direction of septal depolarization is reversed (becomes right to left), as the impulse spreads first to the RV via the right bundle branch and then to the LV via the septum.
- This sequence of activation extends the QRS duration to > 120 ms and eliminates the normal septal Q waves in the lateral leads.
- The overall direction of depolarization (from right to left) produces tall R waves in the lateral leads (I, V5-6) and deep S waves in the right precordial leads (V1-3), and usually leads to left axis deviation.
- As the ventricles are activated sequentially (right, then left) rather than simultaneously, this produces a broad or notched (‘M’-shaped) R wave in the lateral leads.

Dominant S wave in V1 with broad, notched (‘M’-shaped) R wave in V6
Lt Bundle Branch Block

Diagnostic Criteria of LBBB

- QRS duration of 120 ms
- Dominant S wave in V1
- Broad monophasic R wave in lateral leads (I, aVL, V5-V6)
- Absence of Q waves in lateral leads (I, V5-V6; small Q waves are still allowed in aVL)
- Prolonged R wave peak time > 60ms in left precordial leads (V5-6)

`M'-shaped QRS complex

RS complex

(tiny R wave, deep S wave)
Bundle Branch Block

Left bundle branch block

I
P  R  T
0.15 s
1 mV

V₁
P  T
Deep S

V₆
P  R  T
1 s

Right bundle branch block

I
S
Double R
0.16 s
1 mV

V₁

V₆
Broad S
1 s

Fig. 11-13
The Q wave represents septal depolarization

The r wave represents right ventricular depolarization

S wave represents left ventricular depolarization

R wave represents septal and right ventricular activation

R' wave represents left ventricular depolarization

Left Bundle Branch Block - Lead V₁

Surface ECG demonstrating the different electrophysiologic events that occur with left bundle branch block

Left Bundle Branch Block - Lead V₆
Pacemaker Rhythm

Electronic Pacemaker Spikes

Artificially induces electronic stimulus that paces the patient's rhythm causing a blip or spike on the ECG waveform
Failure to Capture