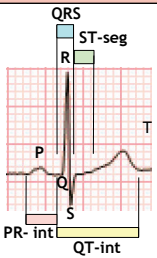
Canadian Family Medicine Clinical Card

ECG Rhythm Interpretations 1 & 2

The authors and reviewers have made every attempt to ensure the information in the Family Medicine Clinical Cards is correct – it is possible that errors may exist. Accordingly, the source references or other authorities should be consulted to aid in determining the assessment and management plan of patients. The Cards are not meant to replace customized patient assessment nor clinical judgment. They are meant to highlight key considerations in particular clinical scenarios, largely informed by relevant guidelines in effect at the time of publication. The authors cannot assume any liability for patient outcomes when these cards are used.

Yang M
Slawnych M
Khattab Y
Keegan DA

ECG Rhythm Interpretation 1

ECG Waveform	Seg/Int	Meaning	N- dur (s)*
	PR-int	AV node conduction	0.12-0.20
	P-Wave	Atrial depolarization	<0.12
	QRS	Ventricular depolarization	<0.12
	ST-Seg	Ventricular depolarization to repolarization delay	No elevation/ depression
	QT-Int	Ventricular depolarization to repolarization	Male: 0.45 Female: 0.46
	T-Wave	Ventricular repolarization	Upright except aVR and V1

* 1 small square= 1 x 1mm = 0.04 s 1 large square= 0.5 x 0.5cm= 0.2 s X-axis= sec, Y-axis= volts

SINUS ARRYTHMIAS

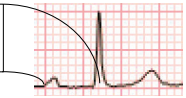
-Physiologically normal and commonly seen in children and young adults
-Originates from the *SA node* so all P-waves look the same but the R-R interval changes with respiration (usually ↑ with inspiration/↓ with expiration)

ATRIOVENTRICULAR (AV) CONDUCTION BLOCKS

First-Degree AV Block

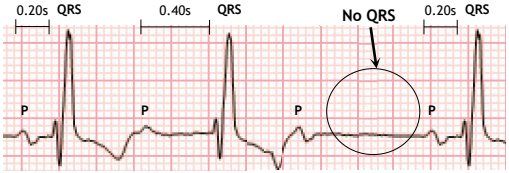
-Delay between atrial and ventricular depolarization
-1:1 P to QRS ratio.

Prolonged PR interval >5 little squares (>0.2s).



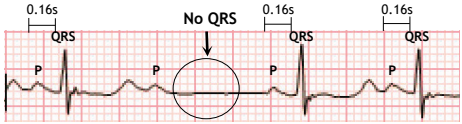
Second-Degree AV Block (Type I)

-Intermittent loss of AV conduction
-Progressive, prolongation of PR intervals resulting in a non-conducted P-wave, after which the cycle resets beginning with the original PR interval



Second-Degree AV Block (Type II)

-Intermittent loss of AV conduction without prior progressive prolongation of PR intervals. If the block persists for >1 beat this is called a high-grade AV block.

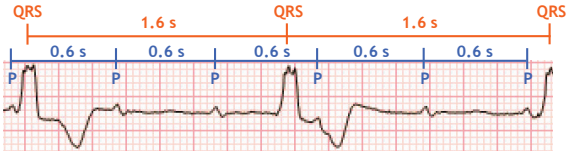


-First-degree and second-degree (Type I) AV blocks are typically benign
-Second degree (Type II) and third degree AV blocks are typically pathological and may require stabilization and EMS activation

Third-Degree AV Block (Complete heart block)

-Complete electrical disconnection between atria and ventricles
-Atrial depolarization is driven by the SA node but ventricular depolarization is driven by a distal escape rhythm (AV junction or ventricles)
-P-waves (normal) and QRS (variable shape/width) have independent rates

-No relationship between P waves and QRS complexes



Bundle Branch Blocks (BBB)

Right BBB
-RSR' (bunny ears) in right chest leads (V1 and V2)
-Widened QRS

Left BBB
-Broad, notched R in left chest leads (V5 and V6)
-Widened QRS (can merge with T wave)

PREMATURE BEATS AND ESCAPE RHYTHMS

-Premature beats arise from spontaneous discharge of ectopic foci resulting in a beat earlier than expected.
-Escape rhythms are discharges from ectopic foci resulting in a new rate and rhythm in response to a pause or block in the SA node pacemaker ability.
-Premature beats or escape rhythms from: 1) *Atrial foci* show a different looking P-wave with normal QRS, 2) *AV junction foci* usually show no P-wave with normal QRS; occasionally P-waves are observed and represent retrograde atrial activation, and 3) *Ventricular foci* show no P-wave with a large/wide QRS.

AV Junction Rhythm

No P-wave

Premature Atrial Contraction (PAC)

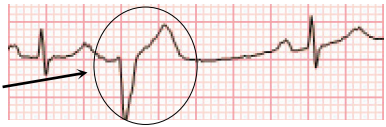
PAC:
Arises earlier than expected

-Majority of PACs are benign but may indicate underlying heart disease

Premature Ventricular Contraction (PVC)

PVC:
Arises earlier than expected

No P-wave and large/wide QRS



Key References: Surawicz, B., Childers, R., Deal B.J. and Gettes, L.S. (2009). AHA/ACCF/HRS recommendations for the standardization and interpretation of the ECG (Part III). *Circulation*. **119**: e235-e240.

Lau, S.H. and Damato, A.N. (1970). Mechanisms of A-V block. *Cardiovasc Clin.* **2**(2): 49-68; Lilly, L.S. (2011) Pathophysiology of heart disease: Fifrh ed.Baltimore, MD: Lippincott Williams & Wilkins.

Canadian Family Medicine Clinical Card

A4/9/12/13 2016
www.learnfm.ca

Yang M
Slawnych M
Keegan DA

ECG Rhythm Interpretation 2

RATE

Method 1: 300 ÷ (# of large squares between 2 beats)
Method 2: Count the # of big boxes between 2 beats using 300-150-100-75-60-50
Method 3:[# of QRS complexes on a standard strip (10 sec)] x 6
Tachycardia is a rate >100 bpm and bradycardia is a rate <60 bpm

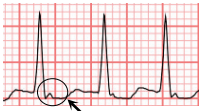
SINUS TACHYCARDIA

-Originates from the SA node
-Regular rhythm with normal P-waves and QRS

TACHYARRYTHMIA

Supraventricular Tachycardia (SVT)

-Paroxysmal (sudden start/end) or persistent tachycardia with a conducting origin from above the ventricles
-Show **narrow** QRS complexes (if no aberrant conduction) and P-wave inversion (if not hidden in QRS or T waves)
-Common mechanisms: 1) Focal (atria or AV junction) and 2) Re-entry circuits (AVNRT, AVRT, atrial flutter)

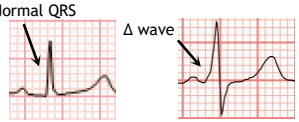


Inverted P-waves

-Re-entry: multiple excitations of the heart with a single impulse through an accessory pathway
-AV node reentrant tachycardia (AVNRT)- re-entry circuit within the AV node
-AV reentrant tachycardia (AVRT)-re-entry circuit between the atria and ventricles

Wolf-Parkinson-White (WPW)

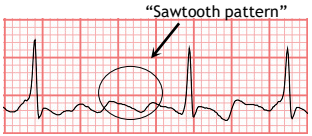
-Concurrent conduction through AV node and accessory pathway (anterograde)
-In sinus: shortened PR interval, shoulder prior to QRS (Δ wave) and wide QRS



Note: absence of Δ wave in normal QRS

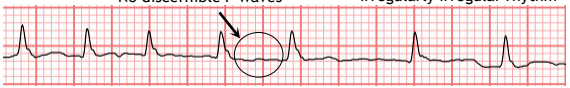
Atrial flutter

-Re-enty circuit confined within the right atrium
-Fixed AV block: fixed atrial: ventricular rate (e.g. 2:1 block)



Atrial Fibrillation (A-Fib)

-Chaotic atrial activity
-Irregular P wave conduction
"Irregularly irregular rhythm"



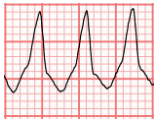
Consult current guidelines for urgent care management

-Tick method: Overlay paper and mark above all QRS's. Then shift the first tick over to the next QRS. If the ticks don't line up, the rhythm is irregular. Repeat.

Ventricular Tachycardia (VT)

-Rapid discharge of >3 PVC's in a row.

Monomorphic VT
-Wide QRS, regular rate, same QRS morphology



Polymorphic VT
-Irregular rate, changing QRS morphology

"waxing and waning" of QRS heights

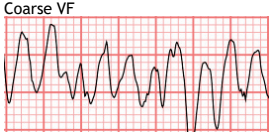
Torsades de pointes
-Polymorphic VT seen in context of a prolonged QT interval.

-Sustained episodes of VT (>30 s) often turn into VF

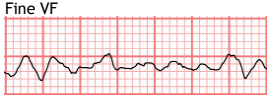
Ventricular fibrillation (VF)

-Chaotic, ventricular activity, no identifiable waveforms, bizarre deflections.

Coarse VF



Fine VF



-All forms of VF are immediately life threatening and require STAT care (follow ACLS guidelines)

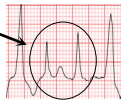
SVT with abberrancy vs VT

-SVT with aberrant conduction (e.g. due to BBB) can look very similar to VT

SVT with Aberrancy	VT
No AV dissociation	AV dissociation
No capture beats	Capture beats*
No fusion beats	Fusion beats

If seen: Pathognomonic

Fusion beats
-SV impulse coincides with V impulse



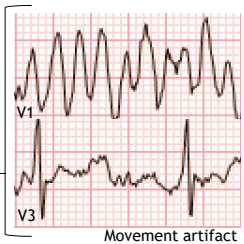
-Wide bizarre QRS complexes (>0.16s), Hx of MI and Hx of CHF are suggestive of VT

*Capture beat: SA node impulse is conducted during AV dissociation (normal P and QRS)

NOISE

-Common artifacts include: other physiological signals, baseline wander, high frequency random noise, power-line interference and movement artifact

In this example, from the same ECG, lead V1 appears to show VT but, as shown in other leads (e.g. V3), this is in fact a movement artifact.



Movement artifact

Key References: Delcretaz, E. (2006) Supraventricular tachycardia. *N Engl J Med.* **354:** 1039-1051; Martis, R.J., Acharya, U.R. and Adeli, H. (2014). Current methods in electrocardiograph characterization. *Comput Biol Med.* **48:** 133-149;

Fisch, C. and Knoebel, S.B. (1970). Junctional Rhythms. *Prog Cardiovasc Dis.* **13(2):** 141-158