

Before you start

Check name, date, time, paperspeed (25 mm/sec), scale (10 mm/mV). Continue with the 7+2 step-plan.

Step 1: Rhythm

Sinus rhythm(SR) (60-100/min): every P wave is followed by a QRS

Narrow QRS tachycardias (QRS<120ms; >100/min) are always supraventricular tachycardias (SVT):

Sinustachycardia: sinusrhythm

>100/min. Eg. Fever / Psych. stress / Cardiomyopathy

Atrial fibrillation (AFIB): irregular

- Permanent = chronic.

- Persisting = recurring after

- chemical / electrical cardioversion

- Paroxysmal = comes and goes spontaneously: SR → AFIB → SR

Atrial flutter: flutter waves on baseline. Often regular 300 / min with a 2:1, 3:1 or 4:1 block.

AVNRT: AV nodal tachycardia.

Regular, 180-250 / min. P in QRS complex (resulting in Rs'R' in V1), often young patients and paroxysmal. *Valsalva / carotid massage / adenosine can terminate episode.*

Wide complex tachycardias (QRS>120ms): possible risk of sudden death, always consult with cardiologist.

Ventricular tachycardia: Arguments for VT (Brugada criteria): fusion (sudden narrow beat), absence of RS precordially, RS > 100ms, AV dissociation, atypical LBBB. *Typically in older patient with previous MI. Unconscious? → proceed to immediate defibrillation.*

SVT with aberrancy. Typical in younger patient. How was the QRS duration / shape on a previous non-tachycardic ECG?

Ventricular fibrillation = no QRS-complexes, but chaotic ECG-pattern, like 'noise' → mechanical cardiac arrest → resuscitate. If patient is conscious it probably is noise.

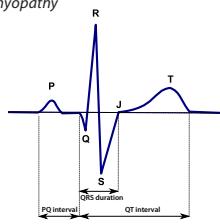
Bradycardia (<60/min). Consider stop / reduce beta-blocker / digoxin / Ca-antagonist. Asymptomatic sinusbradycardia with a normal blood pressure in general doesn't require treatment.

- 1st degree AV-block: prolonged PQ-interval (> 200ms)
- 2nd degree AV-block type I (Wenckebach): PQ interval increases until 1 QRS complex is blocked. *Good prognosis.*
- 2nd degree AV-block type II (Mobitz): PQ interval is normal, but not every P wave is followed by QRS. Requires pacemaker.
- 3rd degree AV-block = complete block. AV dissociation: no relationship between P waves and QRS. Requires pacemaker.
- Ventricular escape rhythm:** wide complex rhythm < 40/min; dangerous. Consult cardiologist. Ischemia? Severe electrolyte shift?

Step 2: Heart rate

Count the number of large grids between two QRS complexes: 1 box in between = 300/min, 2=150/min - 100 - 75 - 60 - 50 - 40. Or use methods at the bottom of this page.

Heart rate = 10 times number of QRS complexes within these 15 cm (= 6 seconds x 25 mm/sec)

**Step 3: Conduction intervals (PQ, QRS, QT)**

Normal: PQ <200ms (5 small squares), QRS <120ms (3 squares), QTc < 450 ms, $\frac{QT}{RR}$ < 460 ms, preferably measured in lead II or lead V5.

PQ > 200ms = AV block (above)

PQ < 120ms + delta-wave = Wolff-Parkinson-White syndrome (WPW), risk of a circus movement tachycardias (= AVRT: AV re-entrant tachycardia)

QRS > 120ms = wide QRS complex, check V1:

- Left Bundle Branch Block (LBBB)**

Latest activity towards the left, away from V1, so QRS ends negatively in V1. New LBBB? Consider ischemia.

- Right Bundle Branch Block (RBBB)**

Rsr' (rabbit ear) latest activity rightwards, (on average) positive in V1

- Intraventricular conduction delay**= if it's not LBBB nor RBBB

QTc > 450ms: consider hypokalemia, post myocardial infarction, long QT syndrome, medication (full list on torsades.org). Risk of torsade de pointes deteriorating into ventricular fibrillation (risk increases especially >500ms).

$$QTc = \frac{QT}{\sqrt{RR(\text{in sec})}}$$

Maximal QTc per given heart rate: what QT value at what heart rate results in a QTc of 450ms?	
50/min:	QT 493ms
60/min:	QT 450ms
70/min:	QT 417ms
80/min:	QT 390ms
90/min:	QT 367ms
100/min:	QT 349ms

Step 4: Heart axis

Heart axis: vector of the average electrical activity. Normal between -30° and +90°. Especially axis deviation compared to previous ECG is relevant.

Normal heart axis: QRS positive in II and AVF

Left axis: AVF and II negative. Eg. left anterior fascicular block (LAFB), LVH.

Right axis. I negative, AVF positive. Eg. pulmonary embolism, COPD.

Step 5: P wave morphology

Normal P wave: positive in I and II, bifasic in V1, similar shape in every beat. Otherwise consider ectopic atrial rhythm.

Left atrial enlargement: terminal negative part in V1 > 1mm². e.g. mitral-regurgitation.

Right atrial enlargement P>2.5mm high in II, III, AVF and / or P>1.5mm in V1. e.g. COPD

Step 6: QRS morphology

Pathologic Q waves? Old myocardial infarction (see ischemia)

Left ventricular hypertrophy (LVH): R in V5/V6 + S in V1 > 35 mm. Seen in e.g. hypertension, aortic valve stenosis.

Wave progression: R increases V1-V5. R-S beyond V3

Microvoltages (<5mm in extremity leads): E.g. cardiomyopathy, tamponade, obesity, pericarditis

Wide QRS complex (QRS > 120ms): see Step 3

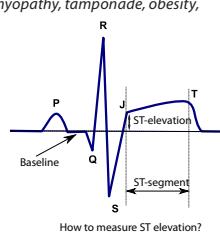
Step 7: ST morphology

ST elevation: consider ischemia, pericarditis, LVH, benign ST elevation, 'early repolarisation'

ST depression: can be reciprocal in ischemie, strain pattern in LVH, digoxin intoxication

Negative T wave: (not in the same direction as the QRS complex) consider (subendocardial) ischemia, LVH

Flat T wave (<0.5 mm): aspecific

**Step +1: Compare with previous ECG**

New LBBB? Change in axis? New pathologic Q waves? Reduced R wave height?

Step +2: Conclusion (1 sentence)

Example: Sinustachycardia with ST elevation in the chest leads with a trifascicular block consistent with an acute anterior myocardial infarction

Ischemia

Acute myocardial infarction (AMI): symptoms (chest pain, vagal response), ECG consistent with transmural ischemia (ST elevations (+reciprocal depressions), new LBBB, sometimes already pathologic Q waves), sometimes already elevated cardiac markers for AMI (Troponin / CKMB). 'Time is muscle'. If you suspect AMI → consult cardiologist immediately (< 5 min.)

ST-elevation points at the infarcted area:

- Anterior:** V1-V4. Coronary territory: LAD. sometimes tachycardia
- Inferior:** II, III, AVF. Coronary: 80% RCA (bradycardia, elevation III>II; depression in I and / or AVL), otherwise RCX (in 20%).
- Right ventricular MI:** ST ↑ in V1 and V4R. IV fluids if hypotensive
- Posterior:** high R wave and ST depression in V1-V3
- Lateral:** elevation in I, AVL, V6. Coronary: LAD (Diagonal branch)
- Left main:** diffuse ST depression with ST elevation in AVR. Very high risk of cardiogenic shock

Reciprocal depression: depression in reciprocal territory (e.g. ST depression in II, III, AVF during anterior MI).

IP-L-infarction: inferior-posterior-lateral. They frequently come together

Pathologic Q-wave (any Q in V1-V3 or Q width > 30ms in I, II, AVL, V4-V6; minimal in 2 contiguous leads, minimal depth 1 mm): previous MI. Leads III and AVR may have a Q wave, which is non-pathological.

Miscellaneous

VPB (ventricular premature beat, VES: ventricular extrasystole, PVC, Premature ventr. contr.). QRS > 120ms. Seen in 50% of healthy men. Increased risk of arrhythmias if: complex form, very frequent occurrence (> 30 / hour) or R on T. Consider: Ischemia? Previous MI? Cardiomyopathy?

PAC (premature atrial contraction, AES): abnormal P wave, mostly narrow (normal) QRS complex

Pericarditis: ST elevation in all leads. PTA depression in II (between the end of the P wave and the beginning of Q wave)

Hyperkalemia: tall T waves. QRS wide, flat P

Hypokalemia: QT prolongs, U wave, torsade

Hypocalcemia: ST prolongs, 'normal' T

Hypercalcemia: QT short, high T

Digoxin-intoxication: sagging ST depressions

Pulmonary embolism: sinustachycardia, deep S in I, Q wave and negative T in III, negative T V1-V3, right axis, sometimes RBBB

Chest lead positioning: V1= 4th intercostal space right (IC4R), V2=IC4L, V3=between V2 en V4, V4=IC5 in midclavicular line, V5=between V4 and V6, V6= same height as V4 in axillary line. To register V4R, use V3 in the right mid-clavicular line.

Hearteate: measure 2 cardiac cycles



