CHAPTER

17

Practical Approach to a Patient with ECG Changes

Shirish MS Hiremath

The ECG is the oldest cardiologic test, but even 100 years after its inception, it continues as the most commonly used cardiologic test.

Historical milestones in ECG[1]

1887: Augustus Desire Waller, recorded electric current preceding cardiac contraction.

1903: Einthoven, developed string galvanometer.

1911: Sir Thomas Lewis published his pioneering work on ECG

1929: Dock, use of cathode ray oscilloscope for ECG

1932: Wolferth CC and Wood CC, introduced chest leads

1942: Goldberger E, Introduced unipolar limb leads.

ECG in emergency room: Despite the advent of expensive and sophisticated alternatives, ECG dictates the timely diagnosis and management in acute coronary syndrome (ACS) and arrhythmias.

In a suspected case of ACS, ECG should be acquired and interpreted promptly (i.e. target within 10 min) after clinical presentation, and initial ECG is non-diagnostic then serial ECG should be acquired (15-20 minute interval) to catch the dynamic changes.[3]

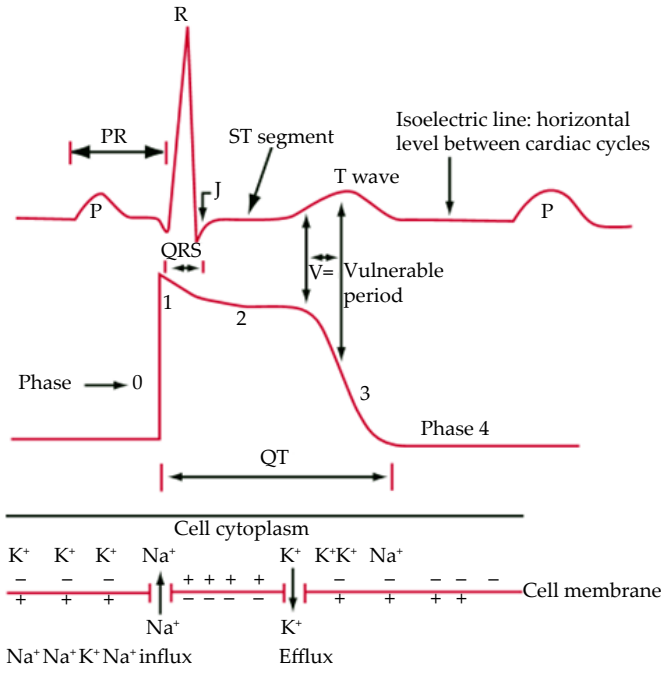

Fig. 1: The action potential, and the electrocardiogram[2]

Acute or evolving changes in the ST– T waveforms and Q waves, when present, potentially allow the clinician to time the event, to identify the infarct-related artery, to estimate the amount of myocardium at risk as well as prognosis, and to determine therapeutic strategy. Various algorithms predict the IRA in case of STEMI, though this correlation is not valid in NSTEMI.

Coronary artery size and distribution of arterial segments, collateral vessels, location, extent and severity of coronary stenosis, and prior myocardial necrosis can all impact ECG manifestations of myocardial ischaemia. Electrocardiographic evidence of myocardial ischaemia in the distribution of a left circumflex artery is often overlooked and is best captured using posterior leads at the fifth intercostal space (V7 at the left posterior axillary line, V8 at the left mid-scapular line, and V9 at the left paraspinal border). A cut-point of 0.05 mV ST elevation is recommended in leads V7–V9; specificity is increased at a cut-point ≥0.1 mV ST elevation and this cutpoint should be used in men, 40 years old. ST depression in leads V1–V3 may be suggestive of infero-basal myocardial ischaemia (posterior infarction), especially when the terminal T wave is positive (ST elevation equivalent), however this is non-specific. In patients with inferior and suspected right ventricular infarction, right pre-cordial leads V3R and V4R should be recorded, since ST elevation ≥0.05 mV (≥0.1 mV in men , 30 years old) provides supportive criteria for the diagnosis (ref Fig---). Other ECG signs associated with acute myocardial ischaemia include cardiac arrhythmias, intraventricular and atrioventricular conduction delays, and loss of precordial R wave amplitude. Coronary artery size and distribution of arterial segments, collateral vessels, location, extent and severity of coronary stenosis,

Table 1: ECG manifestations of acute myocardial ischaemia (in absence of LVH and LBBB)[4]

ST elevation	ST depression and T wave changes
New ST elevation at the J point in two contiguous leads with the cut-points: ≥0.1 mV in all leads other than leads V2–V3 where the following cutpoints apply: ≥0.2mVinmen≥40years;≥0.25mVinmen <40 years,or ≥0.15 mV in women.	New horizontal or down-sloping ST depression ≥0.05 mV in two contiguous leads and/orT inversion ≥0.1 mV in two contiguous leads with prominent R wave or R/S ratio >1.

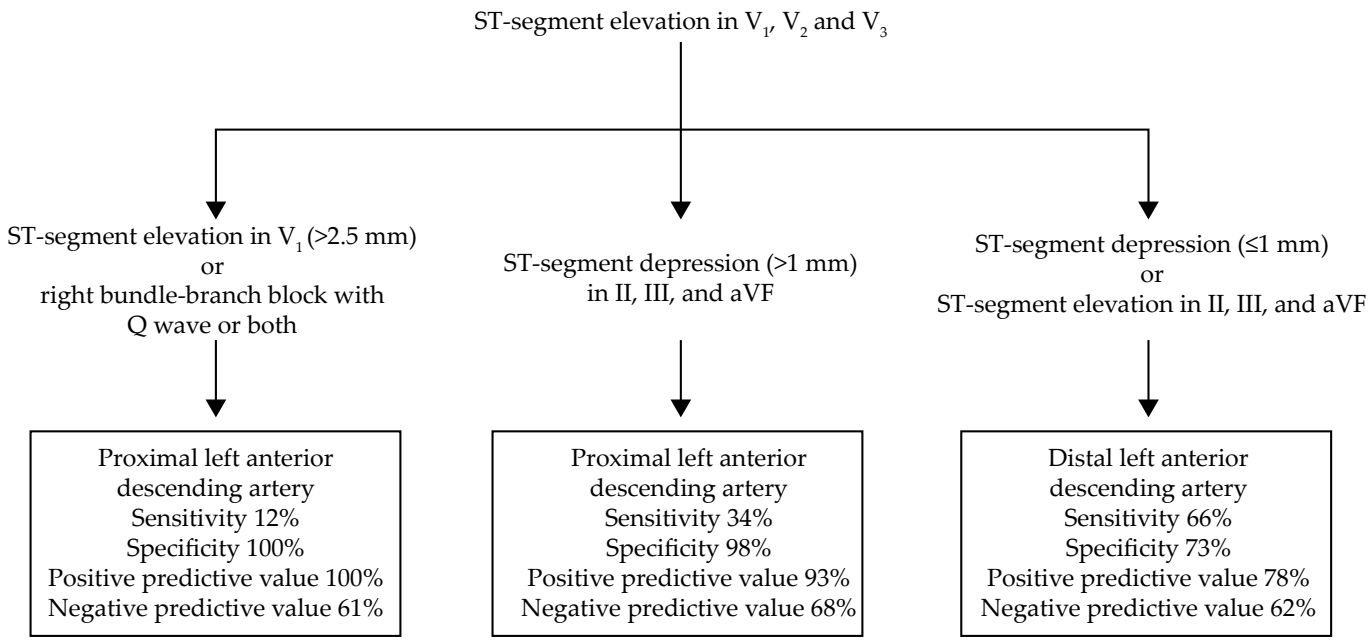

Fig. 2: Algorithm for identifying IRA in anterior wall MI[5]

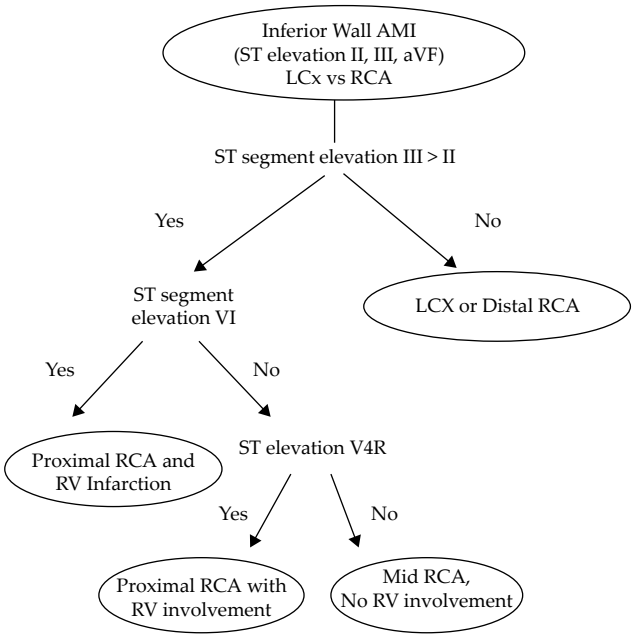

Fig. 3: Algorithm for identifying IRA in inferior wall MI[6]

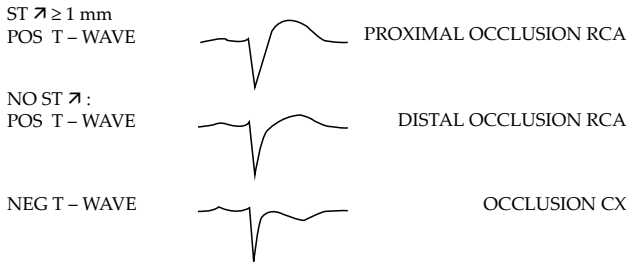

Fig. 4: Value of ST-T changes in acute infero-posterior MI

and prior myocardial necrosis can all impact ECG manifestations of myocardial ischaemia.

When the patient has LBBB on admission and recent previous ECGs do not show LBBB, the patient is presumed to have new-onset LBBB, which many investigators and current guidelines accept as the equivalent of electrocardiographic findings supportive of AMI.[8,9] When the patient has LBBB on arrival and no preexisting ECG is available for comparison, then there are specific ECG criteria which could distinguish between patients with new injury or infarction and those without. Sgarbossa et al. proposed specific electrocardiographic criteria for the diagnosis of AMI in the presence of LBBB applied to patients in GUTO-I trial.[10] But this criteria performed poorly because of a low sensitivity of 10%, although the specificity was high at 82%.

Using a criterion of twice the upper limit of normal value for serum troponin as the 'gold standard' for confirmation of myocardial injury, the investigators in the Wong et al.[11] study demonstrate a high specificity for the criteria.

Acute pericarditis very closely resembles acute coronary syndrome in terms of symptoms and the ECG changes. The two classical ECG findings of acute pericarditis are ST elevation (most sensitive and consistent) and PR depression (most specific). Characteristic ST-elevation of acute pericarditis is concave upwards and it is present in the majority of the standard ECG leads, except in leads AVR and V1 where the ST segment is always depressed. This ST-elevation is often transient and are sometimes followed (after a variable time) by diffuse T-wave inversions.

Event ECG at the time of palpitation is of foremost importance in diagnosis and management of arrhythmia. For evaluation of arrhythmia, traditional step-wise assessment of event-ECG should be followed i.e. rate, rhythm, axis, tachycardia cycle-length, QRS morphology, p-wave morphology, p-QRS relationship. ECG during asymptomatic period has equal importance in diagnosis i.e. p/QRS-morphology, QRS duration (QRSd), QT-

Table 2: Differential diagnosis of ST-elevation[7]

1.	**Ischemia/myocardial infarction** Noninfarction, transmural ischemia (Prinzmetal's angina, and probably Tako-Tsubo syndrome, which may also exactly simulate classical acute infarction) Acute myocardial infarction Postmyocardial infarction (ventricular aneurysm pattern)
2.	**Acute pericarditis**
3.	**Normal variants** (including "early repolarization" patterns)
4.	**Left ventricular hypertrophy/left bundle branch block**
5.	**Other (rarer)** Acute pulmonary embolism Brugada patterns (right bundle branch block–like pattern with ST elevations in right precordial leads) Class 1C antiarrhythmic drugs DC cardioversion Hypercalcemia Hyperkalemia Hypothermia [J (Osborn) waves
6.	**Nonischemic myocardial injury** Myocarditis Tumor invading left ventricle Trauma to ventricles

Table 3: Sgarbossa criteria for diagnosis of AMI in LBBB (score ≥3 have high specificity)[10]

	ECG criteria	Score
1.	ST-segment elevation ≥1 mm and concordant with QRS complex	5
2.	ST-segment depression ≥1 mm in lead V 1 V 2 V 3	3
3.	ST-segment elevation ≥5 mm and discordant with QRS complex	2

interval, presence of pre-excitation, presence of pathological q-wave.

First step in assessment of tachycardia is QRSd, less than 120msec is defined as narrow-complex tachycardia (NCT) and ≥120msec is defined as broad complex tachycardia (BCT). With few exceptions NCT is almost always supraventricular in origin therefore less likely to be life-threatening. With few exceptions BCT is almost always ventricular in origin therefore more likely to be life-threatening.

Initial step in assessment of NCT is regularity of R-R interval, as AV-junctional tachycardias are always regular. In management of NCT, rate control in emergency room

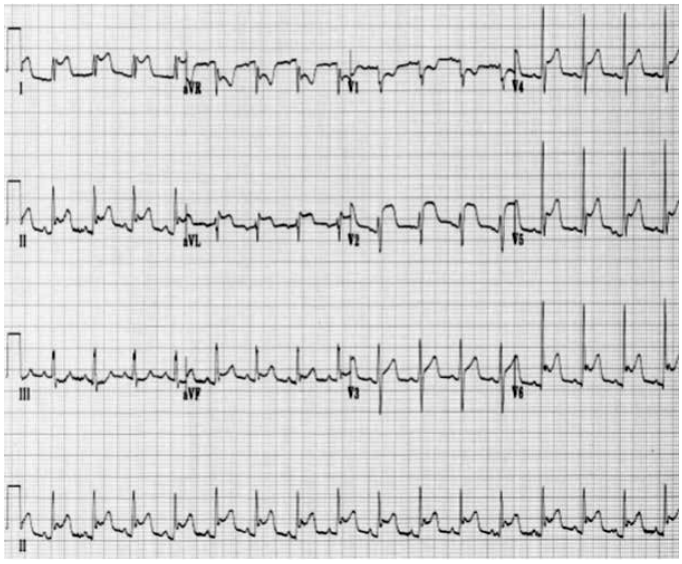

Fig. 5: An electrocardiogram in acute pericarditis.

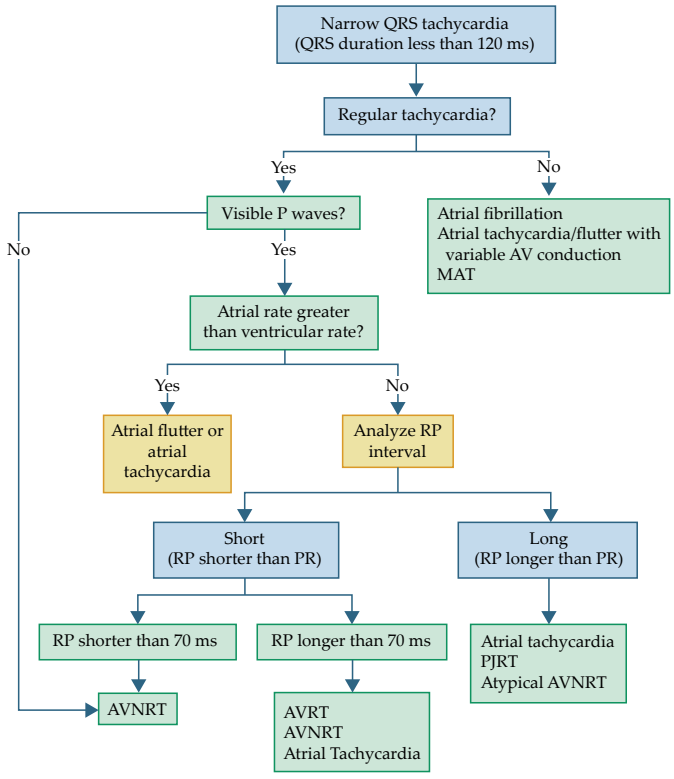

Fig. 6: Algorithm for diagnosis of a narrow QRS tachycardia[12]

can be done with intravenous beta-blocker, calcium channel blocker or adenosine. Long-term management largely depends on number of recurrences and underlying structural heart disease. For tachycardia involving AV-junction (AVRT/AVNRT) electrophysiological study with radiofrequency ablation is highly effective, with more than 95% curing rate in long-term and very low risk of recurrence.

For atrial tachycardias (Afib, Aflutter and ATach), rate control versus rhythm control strategy depends on age of the patient and underlying structural heart disease.

Ventricular tachycardia (VT) arises distal to the bifurcation of the His bundle in the specialized conduction system,

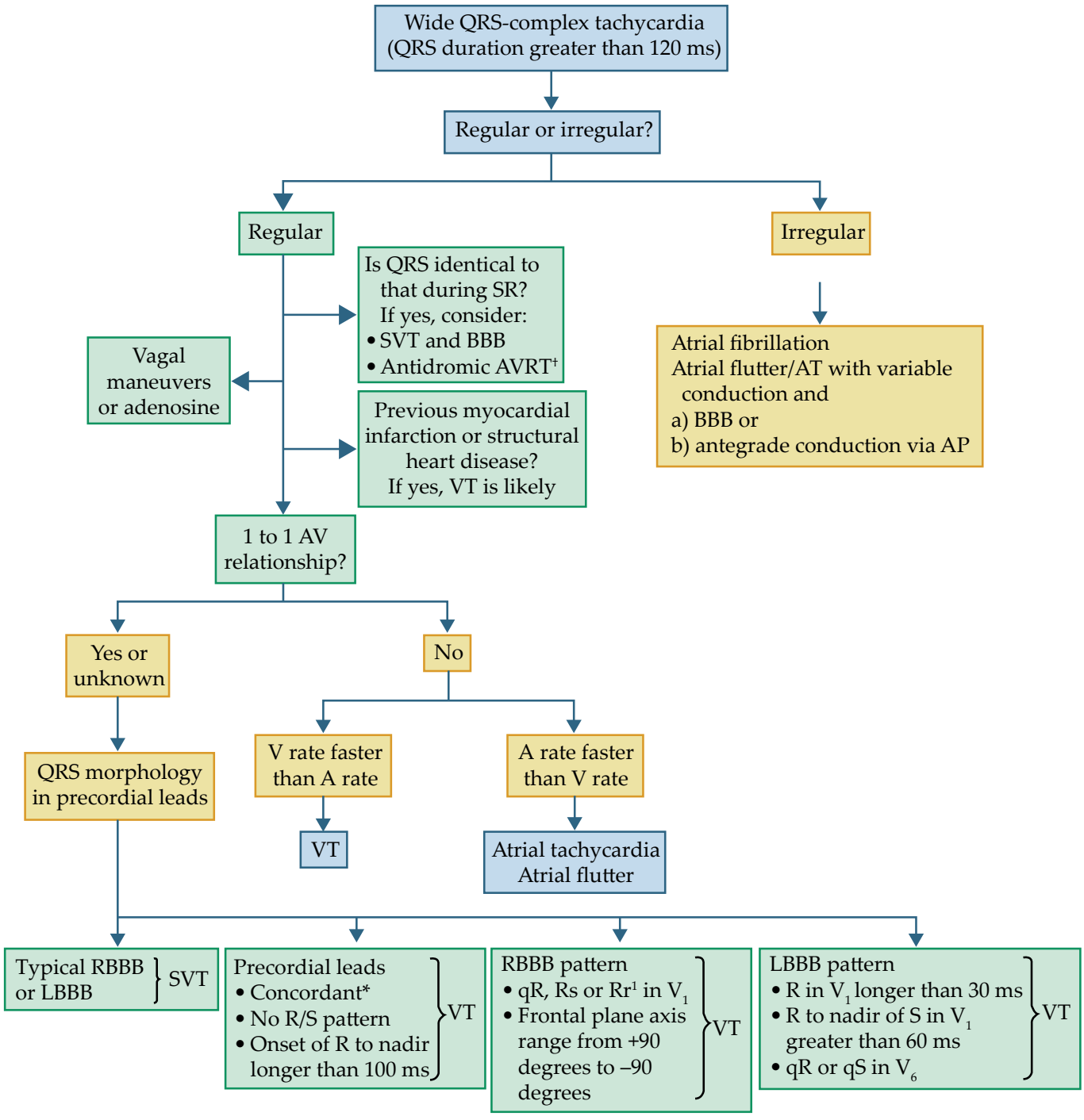

Fig. 7: Algorithm for diagnosis of a wide QRS tachycardia[12]

ventricular muscle, or combinations of both tissue types. Mechanisms include disorders of impulse formation (enhanced automaticity or triggered activity) and conduction (reentry). In patient without underlying structural heart disease, prognosis of VT is good, but with underlying heart disease there is high risk of sudden cardiac death. Differentiating VT from SVT with aberrant conduction is very important, though may be difficult at times.

Various algorithms are available for early diagnosis of VT in emergency room. Of these, Brugada's algorithm is the most commonly used.[13]

After acute management of sustained VT, the goal of long-term therapy is to prevent sudden cardiac death and recurrence of symptomatic VT. Patients with structural heart disease especially LV systolic dysfunction (ejection fraction ≤35%) are at highest risk of recurrence of VT and subsequent SCD.

Idiopathic VT is defined as monomorphic VT in patients without any structural heart disease or coronary disease. There are three distinct entities with characteristic ECG pattern, based on the location of the VT—outflow tract tachycardia, annular tachycardia, and fascicular tachycardia. These type of VTs have very favourable

Table 4: Brugada Algorithm: Steps[13]

	ECG criteria	Cumulative Sensitivity for VT	Cumulative Specificity for VT
1.	Absence of an RS complex in all precordial leads	21%	100%
2.	Precordial RS interval >100 ms	66%	98%
3.	VA dissociation	82%	98%
4.	Morphological criteria for VT	99%	97%

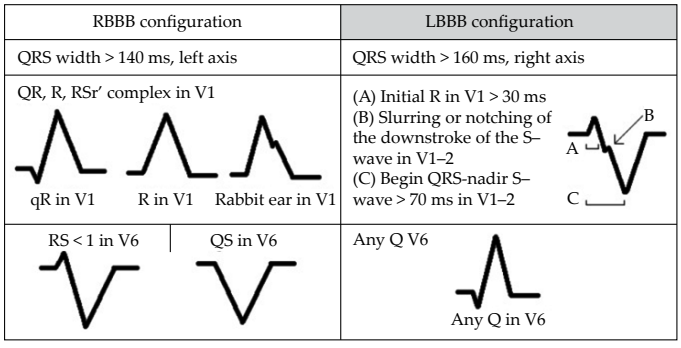

Fig. 8: Brugada's morphological criteria[13]

prognosis and RF catheter ablation effectively eliminates tachycardia in symptomatic patients.

Right ventricular outflow tract (RVOT) VTs have a characteristic electrocardiographic appearance of a left bundle branch block contour in V1 and an inferior axis in the frontal plane. It responds best to vagal maneuvers, beta-blocker. Most probable mechanism is cyclic adenosine monophosphate–triggered activity resulting from early or delayed afterdepolarizations. ECG of LV outflow tract VT mimics RVOT-VT with presence of an S wave in lead I and an early precordial R wave transition (V1-V2).

For mitral annular VT, the ECG pattern is typically right bundle branch block pattern (transition in V1 or V2), S wave in V6, and monophasic R or Rs in leads V2 through V6. For tricuspid annular VT, the foci generally originate in the septal region, and thus the typical ECG pattern is left bundle branches block pattern (Qs in lead V1), an early transition in precordial leads (V3), and narrower QRS complexes.

Left fasicular VT is characterized by a right bundle branch block contour, those originating from posterior fascicle have right-ward axis and those originating from anterior fascicle have left-ward axis. Re-entry is the probable mechanism, so they very well respond to calcium channel blockers (verapamil/diltiazem) and highly amenable to radiofrequency ablation.

Bradyarrhythmias are arbitrarily defined as a heart rate below 60 beats/min. Bradyarrhythmias can be categorized

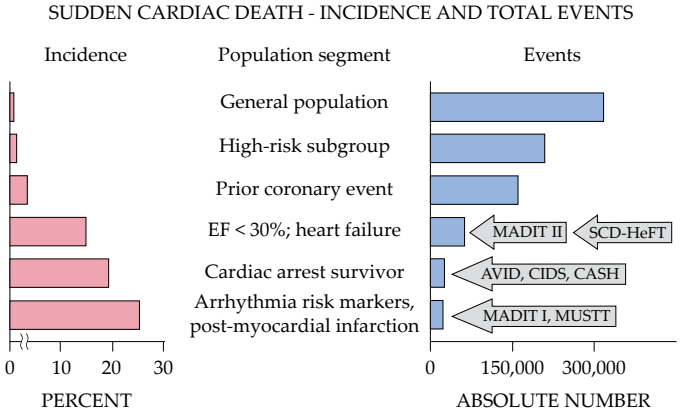

Fig. 9: Impact of population subgroups and time from events on the clinical epidemiology of Sudden cardiac death (SCD)[14]

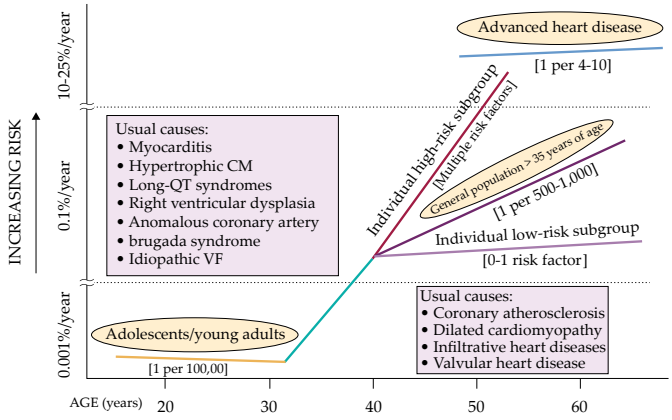

Fig. 10: Age-related and disease-specific risk for SCD

on the basis of the level of disturbance in the hierarchy of the normal impulse generation and conduction system (from sinus node to AV node to His-Purkinje system).

Sinus bradycardia is defined as sinus rhythm at a rate slower than 60 beats/min. P waves have a normal contour and occur before each QRS complex, usually with a constant PR interval longer than 120 milliseconds. Sinus arrhythmia often coexists.

Sick sinus syndrome is a term applied to a syndrome encompassing a number of sinus nodal abnormalities, like persistent spontaneous sinus bradycardia inappropriate for the physiologic circumstance; sinus arrest or exit block; combinations of SA and AV conduction disturbances; alternation of paroxysms of rapid regular or irregular atrial tachyarrhythmias and periods of slow atrial and ventricular rates (bradycardia-tachycardia syndrome). Sick sinus syndrome can occur in the absence of other cardiac abnormalities. The course of the disease is frequently intermittent and unpredictable because it is influenced by the severity of the underlying heart disease.

An AV block exists when the atrial impulse is conducted with delay or is not conducted at all to the ventricle when the AV junction is not physiologically refractory. The conduction disturbance is classified by severity into three categories. Most common cause is age-related degenerative AVB followed by congenital, post-operative.

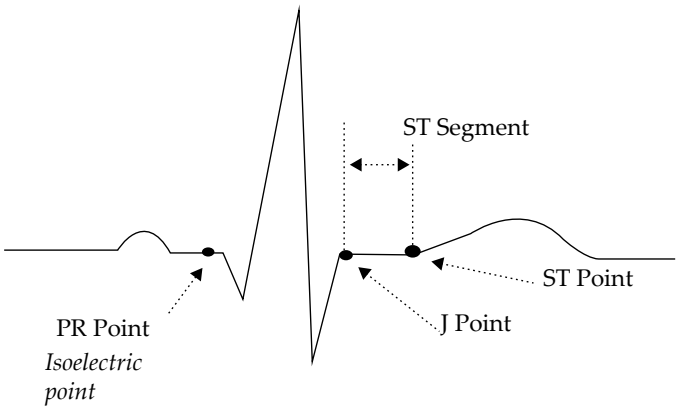

Fig. 11: Automated ST-segment monitoring; reference points

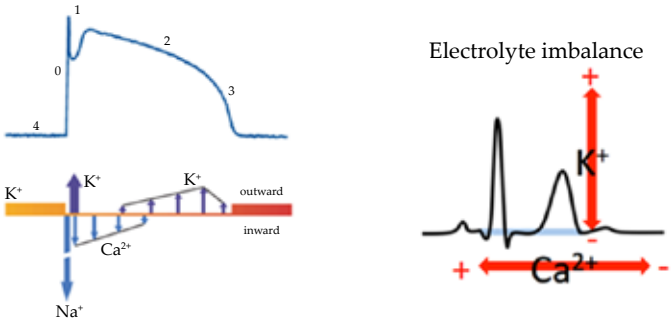

Fig. 12: A ventricular action potential with a schematic of the ionic currents flowing during the phases of the action potential

Depending on severity and level of block, there 3 types of AVB.

1. First degree: During first-degree AV block, every atrial impulse conducts to the ventricles and a regular ventricular rate is produced, but the PR interval exceeds 0.20 second in adults, mostly with no QRS aberration. Clinically important PR interval prolongation can result from a conduction delay in the AV node (A-H interval), in the His-Purkinje system (H-V interval), or at both sites.

2. Second-degree: Blocking of some atrial impulses conducted to the ventricle at a time when physiologic interference is not involved constitutes second-degree AV block. Electrocardiographically, typical type I second-degree AV block is characterized by progressive PR prolongation culminating in a nonconducted P wave , whereas in type II second-degree AV block, the PR interval remains constant before the blocked P wave. In both cases, the AV block is intermittent and generally repetitive. Type II AV block often antedates the development of Adams-Stokes syncope and complete AV block, whereas type I AV block with a normal QRS complex is generally more benign and does not progress to more advanced forms of AV conduction disturbance. Type I AV block with a normal QRS complex almost always takes place at the level of the AV node, proximal to the His bundle. Type I AV block in a patient with a bundle branch block can be caused by a block in the AV node or in the His-Purkinje system. Type II AV block, particularly in association with a bundle branch block, is localized to the His-Purkinje system.

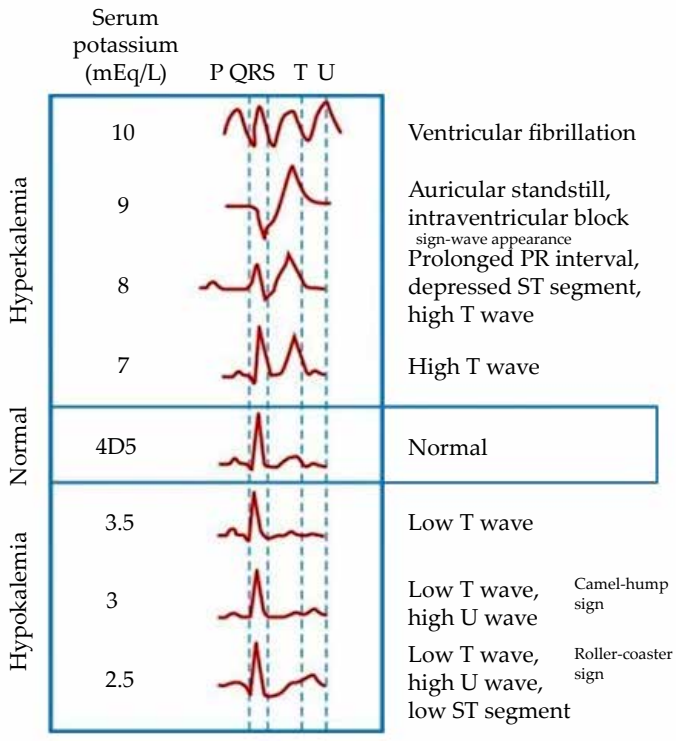

Fig. 13: ECG changes in hyperkalemia

3. Third degree (complete AVB): Third-degree or complete AV block occurs when no atrial activity is conducted to the ventricles and therefore the atria and ventricles are controlled by independent pacemakers, thus there is complete AV dissociation. The ventricular rate in acquired complete heart block is < 40 beats/min but can be faster in congenital complete AV block. Complete AV block can result from block at the level of the AV node (usually congenital), within the bundle of His, or distal to it in the Purkinje system (usually acquired).

ECG in intensive care unit: For patients admitted in medical or surgical ICU continuous ECG monitoring is very essential for early recognition of myocardial ischaemia, electrolyte disturbances, and dysarrhythmias.

Myocardial ischaemia: For ischaemia monitoring, commonly applied standard criteria suggested by Ellestad and colleague includes horizontal or down sloping depression of more than 1 mm at 60 msec from the 'J' point ('J' point is where QRS complex changes its slope) lasting for at least 60 seconds.[6] This has been accepted by American college of cardiology.

On ECG monitor, single best lead, for detection of myocardial ischaemia is V5. V4, II, V3 and V6 are in decreasing order of sensitivity. Chances of ischaemia detection among combined leads is; V5 & II in 80%, V4 & V5 in 90%, V4 , V5 & II in 96% and II & V2 -V5 in 100% of cases.[8]

Electrolyte disturbances in ICU: Potassium and calcium

are essential for maintenance of transmembrane potential and propagation of action potential, so changes in these electrolytes are reflected in resting ECG. But ECG changes don't always correlate with serum level.

ECG changes in hypokalemia are due to delayed repolarisation so the changes are seen in ST-segment, T-wave and U-wave. These are most marked when serum K^+ is < 2.7 mmol/L.

Hyperkalemia is a medical emergency because it leads to cardiac arrhythmias, increase in plasma K^+ concentration depresses intracardiac conduction, with progressive prolongation of the PR and QRS intervals. Severe hyperkalemia results in loss of the P wave and a progressive widening of the QRS complex.

Extracellular calcium is important for transmembrane action potential so changes in serum ionised calcium level affects conduction time leading to changes in QT interval (no changes in QRS-T duration). Hypercalcemia causes shortening and in severe cases absence of ST-segment, conversely in hypocalcemia there is prolong ST-segment.

Marked QT prolongation, sometimes with deep, wide T-wave inversions, may occur with intracranial bleeds, particularly subarachnoid haemorrhage ("CVA T-wave" pattern). Systemic *hypothermia* also prolongs repolarization, usually with a distinctive convex elevation of the J point (Osborn wave). Similar J-point elevation is also seen in severe Hypercalcemia, called as nonhypothermic Osborn wave.

REFERENCES

1. R S Atkinson, G B Rushman, J Alfred Lee. Notes on Physiology. A Synopsis of Anaesthesia. Bombay: K M Varghese Company, 1987; 51–52.
2. Khan, M. Gabriel: On Call Cardiology, 3rd ed., Philadelphia, 2006, WB Saunders, Elsevier Science
3. Thygesen K, Alpert JS, White HD, Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction. Universal definition of myocardial infarction. *Eur Heart J* 2007; 28:2525–2538.
4. ThygesenK, AlpertJS, JaffeAS. Third universal definition of myocardial infarction. *European Heart Journal* 2012; 33:2551–2567.
5. Engelen DJ, Gorgels AP, Cheriex EC, De Muinck ED, Ophuis AJ, Dassen WR, et al. Value of the electrocardiogram in localizing the occlusion site in the left anterior descending coronary artery in acute anterior myocardial infarction. *J Am Coll Cardiol* 1999; 34:389-95.
6. Gorgels AP, Engelen DJ, Wellens HJ. The electro cardiogram in acute myocardial infarction. In: Hurst's the Heart. 11th edition, Fuster V, Alexander RW, O'Rourke RA (Eds.), McGraw-Hill: New York 2004:p.1351-60.
7. Braunwald E, Bonowro, Mann DL, Zipes DP Et Al, Editors. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 9th Ed. Saunders, An Imprint Of Elsevier 2012
8. Edhouse JA, Sakr M, Angus J, et al. Suspected myocardial infarction and left bundle branch block: electrocardiographic indication of acute ischemia. *J Accid Emerg Med* 1999; 16:331–5.
9. Antman EM, Anbe DT, Armstrong PW, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction—executive summary: a report of the ACC/AHA Task Force on Practice Guidelines (Writing Committee to Revise the 1999 Guidelines for the Management of Patients with Acute Myocardial Infarction). *Circulation* 2004; 110:588–636.
10. Sgarbossa EB, Pinski SL, Barbagelata A, et al., for the GUSTO-1 Investigators. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. *N Engl J Med* 1996; 334:481–7.
11. Wong C-K, French JK, Aylward PEG, et al. Patients with prolonged ischemic chest pain and presumed-new left bundle branch block have heterogeneous outcomes depending on the presence of ST-segment changes. *J Am Coll Cardiol* 2005; 46:29–38.
12. Blomstrom-Lundqvist C, Scheinman MM, Aliot EM, et al: ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias—executive summary: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines [Writing Committee to Develop Guidelines for the Management of Patients With Supraventricular Arrhythmias]. *Circulation* 2003; 108:1871.
13. P Brugada, J Brugada, L Mont, J Smeets and E W Andries, A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. *Circulation* 1991; 83:1649-1659.
14. Myerburg RJ, Kessler KM, Castellanos A: Sudden cardiac death: Structure, function, and time-dependence of risk. *Circulation* 1992; 85[Suppl I]:I2.
15. Ellestad M H, Crooke DM Jr, Greenberg P S. Stress testing: principles and practice. Philadelphia: F A Davis, 1980; 85.
16. London MJ, Hollenberg M, Wong MG et al. Intraoperative myocardial ischaemia: localization by continuous 12 lead electrocardiography. *Anesthesiology* 1998; 9:232-241.