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Golwalla's Electrocardiography *for* Medical Students and General Practitioners



FIFTEENTH EDITION

Revised and Edited by
Sharukh A Golwalla

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CHAPTER

3 Method of Analysis of the Electrocardiogram

Correct recording—Before starting to analyze an ECG, it is necessary to ensure that the record was made correctly by observing the following:

1. **The ECG grid and Standardization:** Horizontal and vertical lines are marked on all ECG papers. The vertical lines represent time and are divided into large and small squares. Every fifth line is marked deeper than the others. Each large square represents 0.2 seconds and each small is one-fifth of this, i.e., 0.04 seconds. The horizontal lines represent voltage, 1 mm being equal to 0.1 millivolt. For correct standardization 1 millivolt of potential difference should give a 10 mm vertical deflection **(Fig. 11)**.
2. **Paper speed:** It should be 25 mm/second. A speed of 50 mm/second will space out the entire PQRST complex.

Interpretation: In the interpretation of an electrocardiogram, it is advisable to follow a method, such as one described below so that the individual features of the tracings are recorded in a definite sequence thus facilitating the diagnosis.

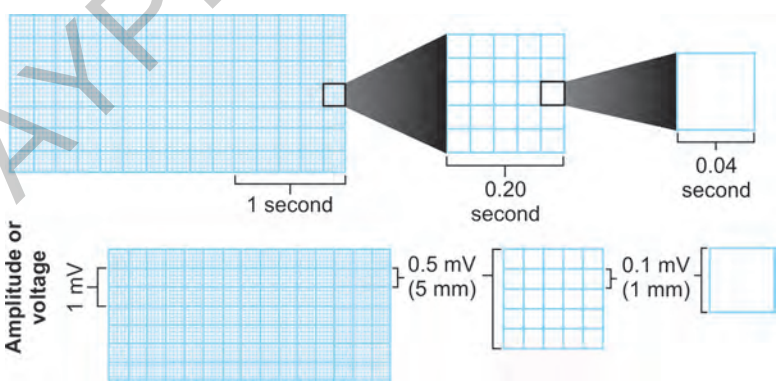

Fig. 11: Time lines, voltage lines and correct standardization in an electrocardiogram.

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I. Rate
II. Rhythm
III. Voltage
IV. Electrical axis
V. P wave
VI. PR interval
VII. Q wave
VIII. QRS complex
IX. ST segment
X. T wave
XI. QT interval
XII. U wave
XIII. J wave

RATE

The rate of the heart per minute can be calculated from the electrocardiogram in the following ways:

Regular rhythm

a. Count the number of 0.2 second time intervals (one big square) between the peaks of any two successive R waves for ventricular rate (or P waves for atrial rate or pacemaker spikes for pacemaker rate). Divide $\frac{60}{0.2} = 300$ by this figure. In **Figure 12,** there are four 0.2 second time intervals between two successive R waves. Hence, the ventricular rate is about 75 per minute.

b. Count the number of R waves falling in 15 successive big squares (3 seconds) and multiply this by 20, or in 10 big squares and multiply by 30. In **Figure 13,** there are 7 R waves in 15 large squares, therefore, the heart rate per minute is $7 \times 20 = 140$.



Fig. 12: Measurement of the heart rate, regular rhythm. Determining the time interval between two successive R waves.

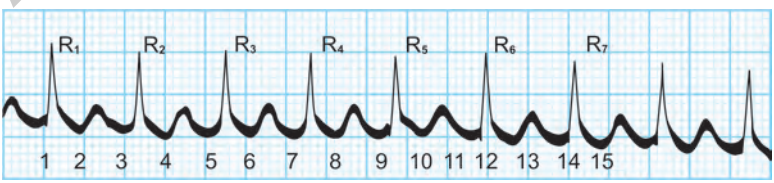

Fig. 13: Measurement of the cardiac rate, regular rhythm. Counting the number of R waves falling in 15 successive 0.2 second intervals.

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Number of small squares	Heart rate
5 (1 large square)	300
6	250
7	214
8	188
9	167
10 (2 large squares)	150
11	136
12	125
13	115
14	107
15 (3 large squares)	100
16	94
17	88
18	83
19	79
20 (4 large squares)	75
21	71
22	68
23	65
24	63
25 (5 large squares)	60
26	58
27	56
28	54
29	52
30 (6 large squares)	50
3 1	48
32	47
33	45
34	44
35 (7 large squares)	43
36	42
37	41
38	39
39	38
40 (8 large squares)	37

c. Count the number of small squares between 2 R waves and divide 1500 by this number, for heart rate per minute (See above table)

Irregular rhythm (Fig. 14): Count the number of squares over a given period of time, say 3.0 seconds interval. Since, each large square = 0.2

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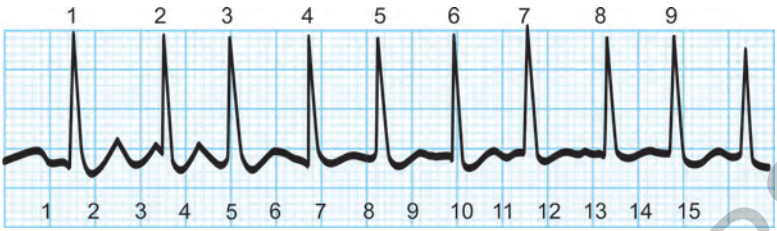

Fig. 14: Determination of heart rate, irregular rhythm. Count the number of complexes (R waves) in span of 15 large squares (3 Secs), in the illustration, the number of R waves is 9, therefore, heart rate/minute = 20 x 9 = 180.

second, 15 large squares = 15 × 0.2 = 3 seconds. Multiply this Figure by 20 to give rate/60 seconds.

RHYTHM

For diagnosis of an *arrhythmia,* the following observations must be made:

1. *P waves*—for atrial rhythm.
2. *QRS complexes*—for ventricular rhythm.
3. *Relationship between P waves and QRS complexes*—for atrio-ventricular conduction.
4. *Any unusual complexes*—early, late or of unusual contour.

A. Sinus Rhythm

Sinus rhythm P waves are upright and regular in leads II, III, aVF

1. **Normal sinus rhythm (Fig. 15):** Impulses originate at SA node at normal rate of 60–100/minute.
2. **Sinus bradycardia (Fig. 16)**: Heart rate less than 60/minute.
3. **Sinus tachycardia (Fig. 17)**: Heart rate more than 100/minute.
   Bradycardia-Tachycardia syndrome (Sick sinus syndrome). The bradycardia-tachy-cardia syndrome, consisting of alternating bradycardia, due to sinus arrest, sinus bradycardia, or sinoatrial exit block combined with tachycardia from paroxysmal atrial or junctional arrhythmias **(Figs. 18A and B)** may produce symptoms referable to the slow or fast heart rates.
4. **Sinus arrhythmia (Fig. 19):** P waves identical and upright in leads II, III, aVF, but rhythmically irregular. Longest PP interval exceeds

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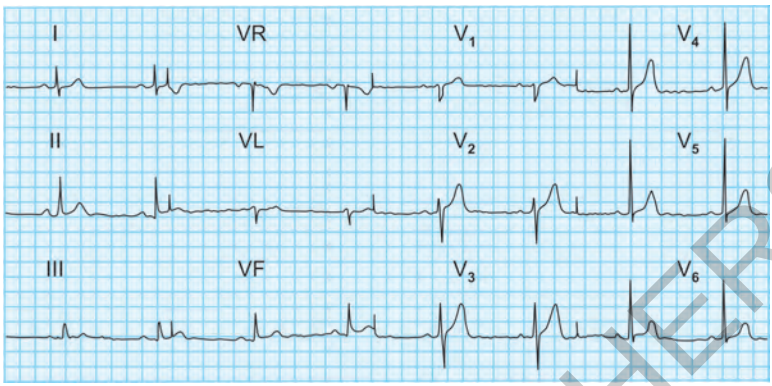

Fig. 15: Normal sinus rhythm.

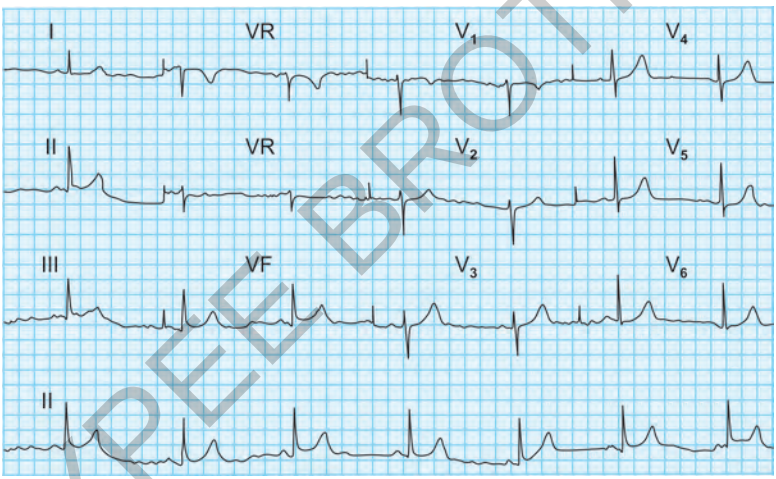

Fig. 16: Sinus bradycardia.

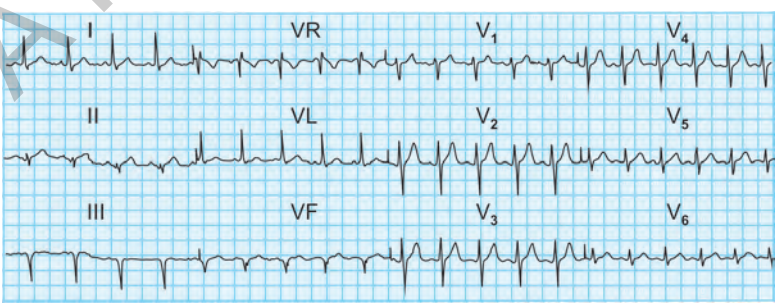

Fig. 17: Sinus tachycardia.

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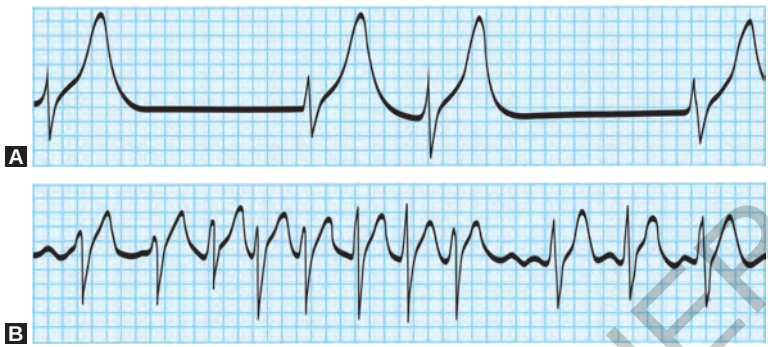

Figs. 18A and B: The 'sick sinus syndrome': (A) Irregular nodal bradycardia; (B) Paroxysmal atrial tachycardia.

the shortest such interval by 0.1 second or more. The commonest form is *respiratory,* the rate increasing during inspiration and slowing during expiration.

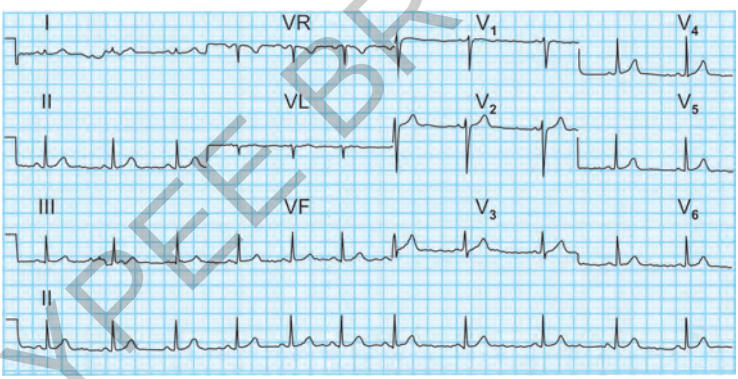

Fig. 19: Sinus arrhythmia. The first part of the tracing is during expiration and inspiration increases the rate.

B. Atrial Rhythms

1. **Non-sinus node (coronary sinus) atrial rhythm (Fig. 20)**: Impulses originate low in the atrium, travel retrograde and also distally. P waves identical and regular but inverted in leads, II, III, aVF implying an origin other than SA node located in upper right corner of the atrium.

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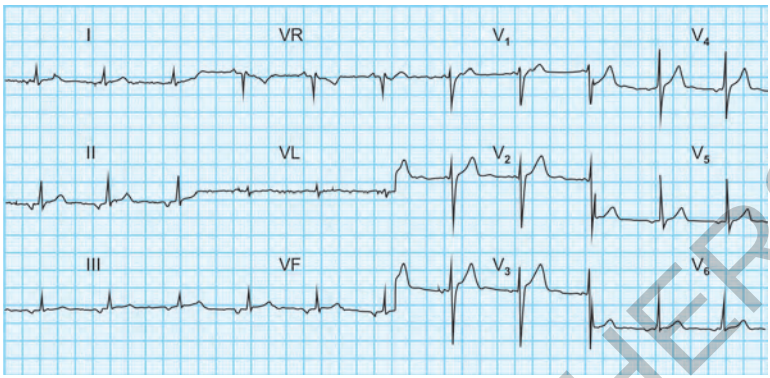

Fig. 20: Non-sinus node atrial rhythm.

2. **Wandering atrial pacemaker (Fig. 21)**: Impulses originate from various foci in atria. The shape or contour of P wave varies from beat to beat in a single lead, often associated with variation of PR interval and PP (and RR) intervals.



Fig. 21: Wandering atrial pacemaker, variation in P wave contour, PR interval, PP and hence RR intervals.

3. **Multifocal atrial tachycardia (Fig. 22)**: Impulses originate rapidly and irregularly at different points in atria. MAT is similar to wandering pacemaker, but the difference is that the heart rate is usually more than 100/minute. It is usually associated with severe pulmonary disease often with respiratory decompensation or congestive heart failure (CHF).
4. **Paroxysmal atrial tachycardia (Figs. 23A and B):** Impulses recycle repeatedly in and in vicinity of AV node due to slowing in area of unidirectional block (re-entry tachycardia). P waves regular, identical in lead II, sometimes merged with T waves and very rapid rate (usually 160–220/minute).
   If the rate is sufficiently rapid in PAT, normal refractoriness of the A-V junction may prevent transmission of many stimuli—atrial

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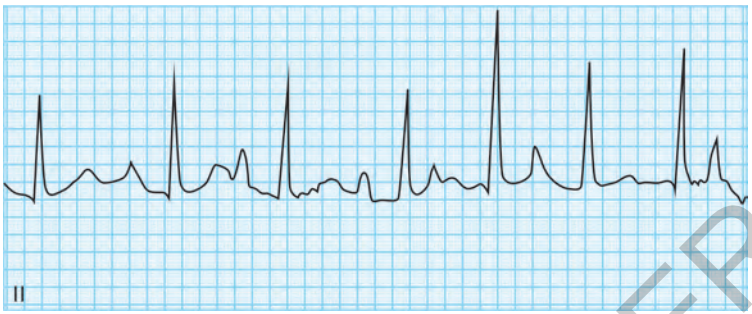

Fig. 22: Multifocal atrial tachycardia. P wave contours, PR, PP and thus RR intervals may all vary.

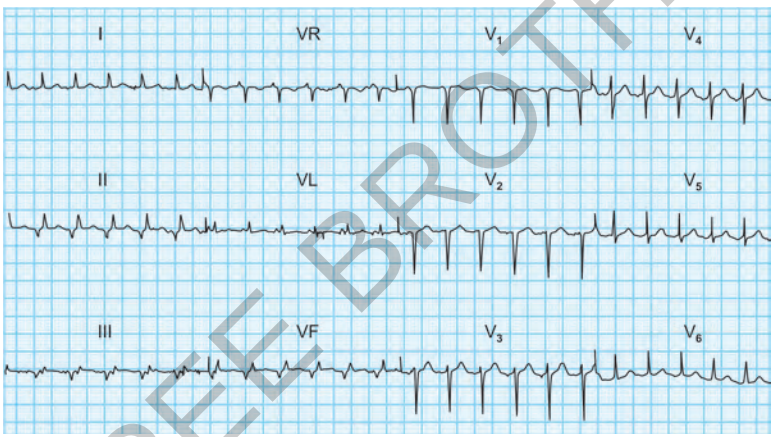

Fig. 23A: Paroxysmal atrial tachycardia (PAT). P waves regular and inverted.



Fig. 23B: Paroxysmal atrial tachycardia with varying AV block. Note the irregular ventricular rate.

tachycardia with varying block **(Fig. 23B).** This arrhythmia is often due to digitalis toxicity.

5. **Atrial fibrillation (Figs. 24A and B)**: Impulses follow chaotic, random pathway in atria. P waves not seen because of lack of

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effective pumping motion of the atria. QRS complexes irregularly irregular. The base line may be coarsely or finely irregular. The ventricular rate may be rapid or slow.

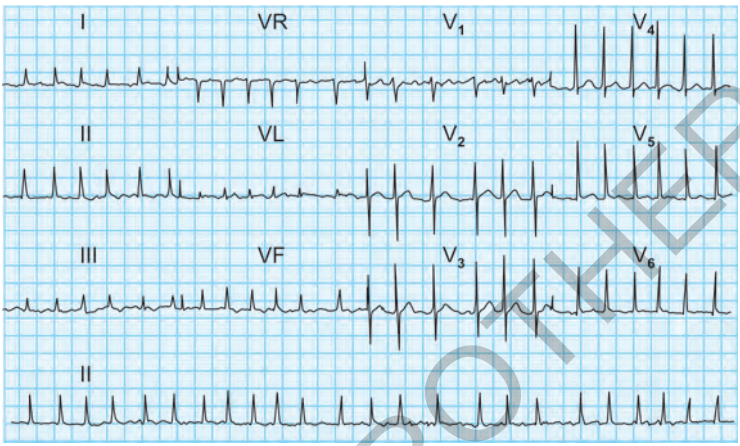

Fig. 24A: Atrial fibrillation with rapid rate.

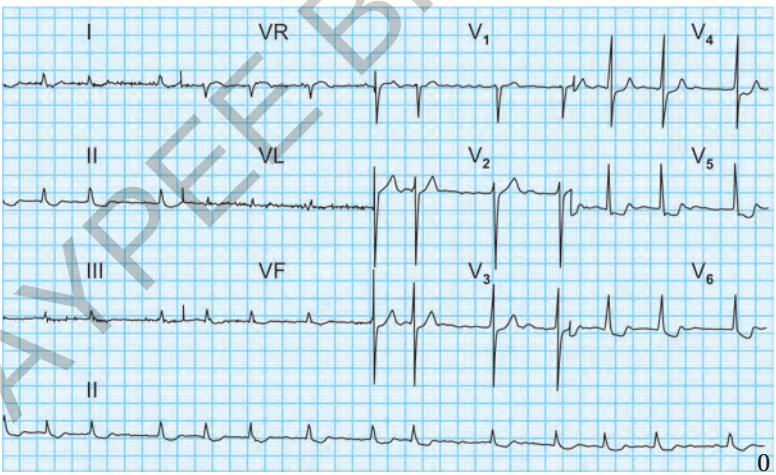

Fig. 24B: Slow atrial fibrillation. Digitalis effect.

6. **Atrial flutter (Fig. 25):** Impulses travel in a circular course in the atria, setting up regular, rapid (220–300/minute) flutter waves. Rapid, identical undulating P waves of longer duration and at times

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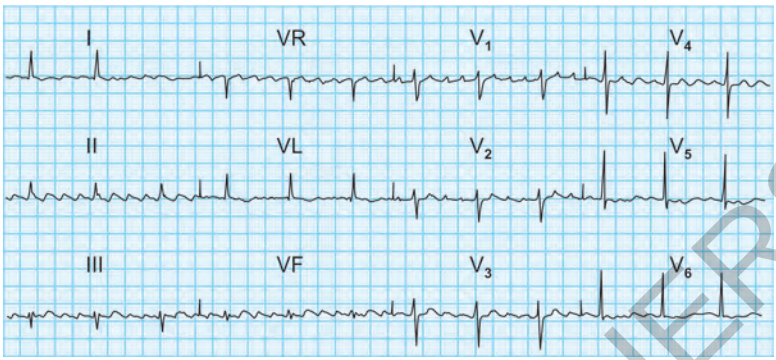

Fig. 25: Atrial flutter with 4:1 block. The rhythm is regular rate is slow, QRS duration normal.

of longer amplitudes than normal P waves (flutter or F waves). The F waves have a rate of about 300/minute, but depending on the AV block which is usually present, the rate may be 150 (2:1 block), 100 (3:1 block), or 75 (4:1 block) per minute. The baseline of atrial flutter is never isoelectric. Coarse atrial fibrillation may at times resemble atrial flutter and a rhythm with characteristics of both may be observed (Flutter-fibrillation) **(Fig. 26)**.

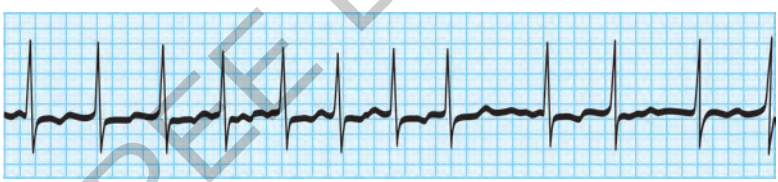

Fig. 26: Atrial flutter-fibrillation. Combination of flutter and fibrillation waves.

C. Junctional (Nodal) Rhythm

Impulses originate in AV node with retrograde and antegrade transmission. P waves may not be identified and QRS complexes are regular and of normal duration. The site of origin of the impulse is the AV node, hence, the heart rate is usually 40–60/minute. (Rate of junctional cells is lower than the rate of SA node). P waves in leads II, III, aVF tend to be inverted (because impulses that originate in AV node are transmitted in a retrograde direction to the atrium). Depending on whether the junctional rhythm reaches and depolarizes the atrium before, at the same time or after the ventricles, the P wave will precede, be buried in or follow QRS complex **(Fig. 27)**.

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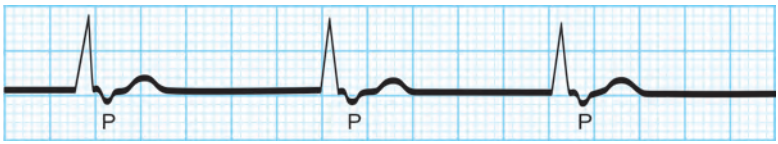

Fig. 27: Junctional rhythm. P waves inverted and follow QRS. Rate slow. QRS duration normal.

D. Ventricular Rhythms

a. **Idioventricular rhythm (Fig. 28)**: The rhythm originates from the ventricle itself. QRS complexes are wide, often somewhat bizarre with T waves in opposite direction to QRS complexes and with rate of 20–40/minute.

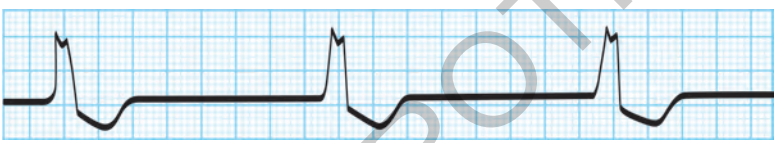

Fig. 28: Idioventricular rhythm. Total absence of P wave. RR interval regular.

Accelerated idioventricular rhythm (Slow ventricular tachycardia)—Wide, often bizarre **QRS** complexes with rate usually between 80-120/minute **(Fig. 29).**



Fig. 29: Accelerated idioventricular rhythm (AIVR).

b. **Ventricular tachycardia (Fig. 30):** Rapid, broad and often bizarre QRS complexes with rates above 140/minute.

c. **Torsade de pointes ventricular tachycardia (Fig. 31):** It is an uncommon arrhythmia characterised by a ventricular tachycardia, often of moderate rate but associated with an undulating QRS height due to a slow but continual variation in the QRS axis (polymorphic **VT).** Its importance is due to its precipitation by antiarrhythmic drugs, particularly if electrolyte disturbances coexist, along with prolonged QT interval.

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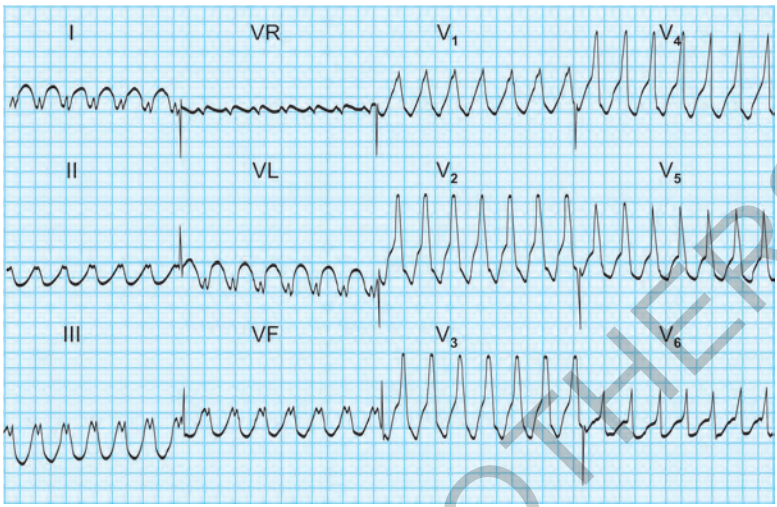

Fig. 30: Ventricular Tachycardia (VT).

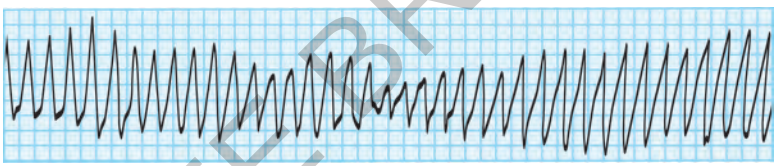

Fig. 31: Drug-induced ventricular tachycardia of the torsade de pointes type. Note the variation in the axis of the QRS complex causing an undulated (or twisted) appearance.

d. **Ventricular fibrillation (Fig. 32):** There is chaotic ventricular depolarization. There are no true QRS complexes and the ECG baseline shows coarse or fine chaotic undulations.

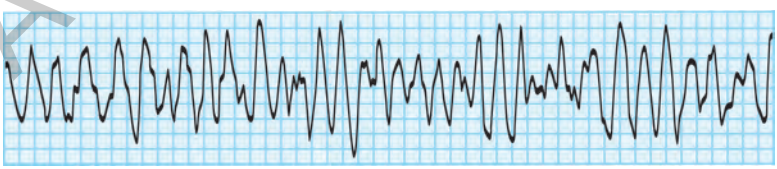

Fig. 32: Ventricular fibrillation. Complete absence of properly formed ventricular complexes.

e. **Pacemaker rhythm (Fig. 33):** Transvenous pacemaker produces a beat in the right ventricle; and therefore a wide QRS. Besides

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