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CD Contents

- The Technique of Recording an ECG.
ECG Made Easy®

Fifth Edition

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Dedicated to

My Parents
Ms Prem Luthra
and
Mr Prem Luthra
who guide and bless me
from heaven
The imaging techniques of contemporary ‘high-tech’ cardiology have failed to eclipse the primacy of the 12-lead ECG in the initial evaluation of heart disease. This simple, cost-effective and readily available diagnostic modality continues to intrigue and baffle the clinician as much as it confuses the student. A colossal volume of literature on understanding ECG bears testimony to this fact.

This book is yet another humble attempt to bring the subject of ECG closer to the hearts of students and clinicians in a simple and concise form. As the chapters unfold, the subject gradually evolves from basics to therapeutics. Although emphasis is on ECG diagnosis, causation of abnormalities and their clinical relevance are briefly mentioned too. This should help students preparing for their examinations without having to search through voluminous textbooks.

While some arrhythmias are harmless, others are ominous and life-threatening. The clinical challenge lies in knowing the cause of an arrhythmia, its significance, differential diagnosis and practical aspects of management. Therefore, seemingly similar cardiac rhythms are discussed together under individual chapter headings. Medical students, resident doctors, nurses and technicians will find this format particularly useful.

I have thoroughly enjoyed the experience of writing this book and found teaching as pleasurable as learning. Since the scope for further refinement always remains, it is a privilege to bring out the vastly improved 5th edition of *ECG Made Easy*. Your appreciation, comments and criticisms are bound to spur me on even further.

Atul Luthra
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NORMAL WIDE QRS RHYTHM

A regular cardiac rhythm at a rate of 60–100 beats per minute is considered to be a normal rhythm. If the QRS complexes during such a rhythm are wide, it indicates abnormal intraventricular conduction of the impulses from the SA node. The P waves and the QRS complexes during sinus rhythm maintain a 1:1 relationship with each other.

The well-known causes of wide QRS complexes during sinus rhythm are bundle branch block, intraventricular conduction defect and Wolff-Parkinson-White (WPW) syndrome. There is one more condition where wide QRS complexes arise from a ventricular pacemaker at a rate of 60–100 beats/minute and is known as accelerated idioventricular rhythm (AIVR).

Let us see how this rhythm differs from sinus rhythm with wide QRS complexes.

ACCELERATED IDIOVENTRICULAR RHYTHM

Accelerated idioventricular rhythm (AIVR) is an ectopic rhythm originating from a latent subsidiary pacemaker located in the ventricular myocardium. Normally, such a
pacemaker is subdued when the cardiac rhythm is governed by the SA node.

However, when a ventricular pacemaker undergoes enhancement of its inherent automaticity, it produces an idioventricular rhythm. Since the heart rate during such rhythm exceeds the inherent ventricular rate, it is known as accelerated idioventricular rhythm (AIVR).

AIVR produces a regular rhythm at a rate of 60–100 beats/minute that is greater than the inherent rate of the ventricular pacemaker which is 20–40 beats/minute. The QRS complexes are bizarre and wide because of ventricular origin of the rhythm (Fig. 20.1).

The distinctive feature of AIVR is atrioventricular dissociation or lack of relationship between the P waves and the QRS complexes. This is because, the ventricles are activated by the ventricular pacemaker, and the atria continue to be activated by the SA node.

AIVR can be differentiated from ventricular tachycardia only by the ventricular rate. The rate is 60–100 beats/minute.
in AIVR and 150–200 beats/minute in VT, although both rhythms originate from the ventricles.

**Clinical Relevance of Regular Wide QRS Rhythm**

*Sinus Rhythm with Wide QRS Complexes*

A normal sinus rhythm when associated with a conduction abnormality in the ventricles, produces wide QRS complexes. The morphology of the QRS complex depends upon the cause of the conduction abnormality. Importantly, the 1:1 P-QRS relationship is maintained during sinus rhythm.

The significance of wide QRS complexes during sinus rhythm depends upon the cause of QRS widening. Causes of wide QRS complexes are:
- Complete bundle branch block
- Intraventricular conduction defect
- Ventricular pre-excitation syndrome.

*Accelerated Idioventricular Rhythm*

AIVR is most often observed in coronary care units in a setting of acute myocardial infarction. It either occurs spontaneously or as a reperfusion arrhythmia after thrombolytic therapy. Other infrequent causes of AIVR are:
- Digitalis toxicity
- Rheumatic carditis
- Cardiac surgery.

The above causes of AIVR are quite akin to those of a junctional tachycardia or accelerated idiojunctional rhythm. Both are examples of an idiofocal tachycardia.

AIVR is most often picked up from the monitor screen of an intensive coronary care unit (ICCU). It needs to be differentiated from its more serious counterpart, ventricular tachycardia that often produces hemodynamic
embarrassment, carries a poor prognosis and requires aggressive management. AIVR differs from VT, only in terms of the ventricular rate.

AIVR also needs to be differentiated from bundle branch block of recent onset, which is not uncommon in an ICCU setting. While AIVR produces bizarre and wide QRS complexes unrelated to P waves, bundle branch block is associated with a triphasic QRS contour and a maintained P-QRS relationship.

AIVR is usually asymptomatic as it occurs at the same rate range as sinus rhythm. It rarely causes serious hemodynamic embarrassment. Only the loss of atrial contribution to ventricular filling (AV dissociation) causes slight fall in cardiac output.

AIVR is usually transient and does not herald the onset of serious ventricular arrhythmias. Therefore, it is considered to be a benign arrhythmia with an excellent prognosis.

Active treatment of AIVR is generally not required as it is transient, asymptomatic and has few hemodynamic consequences. The hallmark of management of AIVR is constant observation. If treatment is required, it is only in patients with poor left ventricular function.

Atropine can be administered to accelerate the sinus rate, overdrive the ventricular rhythm and eliminate atrioventricular dissociation. Antiarrhythmic drugs, DC cardioversion and artificial pacing are unnecessary in the management of accelerated idioventricular rhythm.