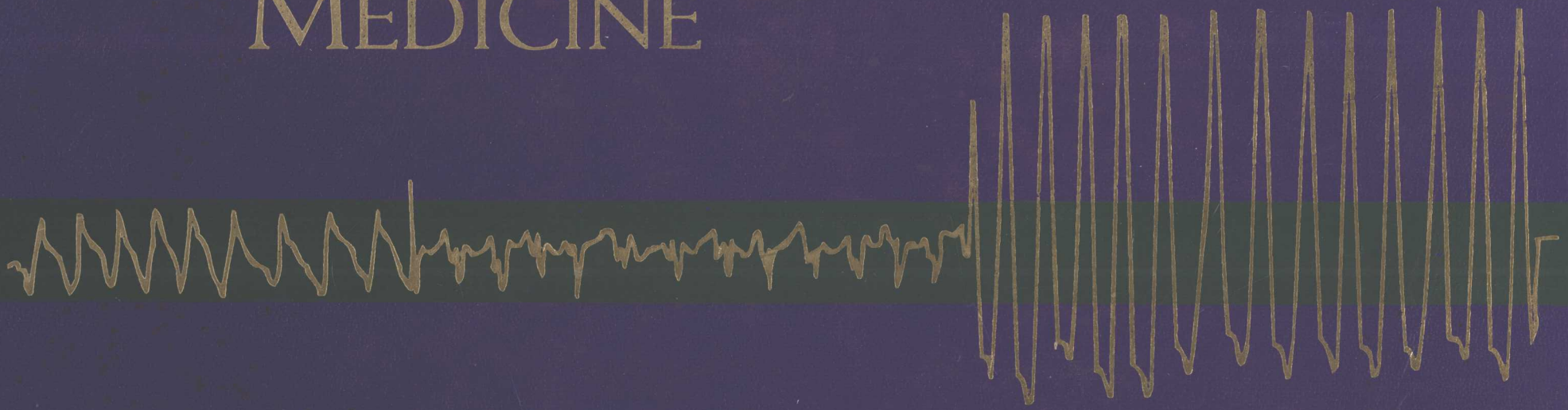
Electrocardiography in Acute Care Medicine



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Electrocardiography in Acute Care Medicine

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Preface

The ideal diagnostic test should be cheap and easy to do; be safe, noninvasive, and reproducible; and in addition, be highly sensitive and accurate. The 12 lead-surface electrocardiogram (ECG) frequently fails to meet the last two requirements. Yet because it fulfills all the others, it is commonly used in the evaluation of a variety of complaints. The ECG is, of course, irreplaceable for the diagnosis and management of cardiac arrhythmias. Finally, the changes caused by many drugs in the ECG become an important guide for the safe and effective use of these agents.

The intent of this book is to present the ECG as it is used in the evaluation of patients with an acute complaint. It is based on the principle that the ECG yields its greatest contribution when interpreted within the context of the clinical setting. The chapters reflect the normal sequence of events in medical care, that is, a particular symptom or sign is recognized and an ECG is secured to explore its significance. For each tracing a summary of the clinical presentation is provided, followed by a description of the ECG findings. The reader, after evaluating these elements, can verify the final interpretation, which then introduces a discussion of the particular entity involved.

The Case Studies have a different format, designed to emphasize the fact that valuable diagnostic information often does not become evident until serial ECG are compared. The follow-up clinical information and the corresponding ECG add the dimension of time to the diagnostic exercise.

It is our strong desire that this book be of special help to the clinicians who perform the difficult and lonely task of securing and interpreting the patient's initial data base. They are the unsung heroes of medicine.

Our gratitude to the staff of the Reingold Electrocardiography Center for their kind assistance and especially to Mr. Chris Jones for his unflagging patience. To their director, Dr. James Rosenthal, thanks for all the help with the "tough ones." Finally, my appreciation to Ms. Xenia Ruiz for the successful mentoring of a former computer illiterate.

Richard Davison

TO LISETTE AND SEBASTIAN

They make it all worthwhile

Electrocardiography in Acute Care Medicine

Contents

PART I

Introduction: The Normal Electrocardiogram

The standard electrocardiogram (ECG) comprises 12 leads: 6 limb leads and 6 precordial leads. The limb leads display the electrical activity of the heart as projected on the frontal plane; the precordial leads do so on a horizontal plane. Because the different leads predominantly reflect certain areas of the heart, they are commonly referred to by the corresponding anatomical region:

II, III, and aV_F	Inferior leads
I and aV_L	High lateral leads
I, aV_L, V_5, and V_6	Lateral leads
V_1 and V_2	Septal leads
V_1 through V_4	Anterior leads
V_1 through V_6	Anterolateral leads

Because no leads are placed directly over the posterior wall of the heart, this area is electrocardiographically "silent." In certain circumstances, the anterior precordial leads can indirectly reflect the posterior wall (Chapter 54). The morphology of the precordial leads depends on the placement of the exploring electrode over the chest sites. When comparing tracings obtained from the same patient, it is important to verify that the lead placement is the same before interpreting the significance of "changes" noted in the precordial leads.

Although no fixed rules about how to read an ECG exist, it is better, at least initially, to follow a predetermined "rountine." Eventually each individual will develop the technique with which he or she is most comfortable. Each ECG reading should address, in sequence, the following components:

Atrial depolarization (P waves)
Rhythm and atrioventricular conduction (PR interval)
Ventricular depolarization (QRS complexes)
Repolarization (ST segment, T waves, QT interval, and U waves)

ATRIAL DEPOLARIZATION

The P wave inscribed during normal atrial depolarization is upright in leads I and II and has a maximum duration of 0.11 seconds. It is often biphasic in V_1 and V_2, with the initial positive deflection originating in the anteriorly located right atrium and the terminal negative component reflecting the later activation of the more posterior left atrium.

RHYTHM AND ATRIOVENTRICULAR CONDUCTION

Three measurements—rate, regularity, and origin and conduction of the stimulus—are used to determine rhythm and AV conduction.

Rate

Electrocardiographic paper is marked at 3-second intervals. Heart rate can be easily calculated by counting the beats contained between two of these marks and multiplying the number by 20. If the rhythm is irregular, several 3-second intervals will have to be counted.

Normal sinus rhythm fluctuates between 60 and 100 beats per minute, although children can normally exceed the upper limit and athletes frequently show much slower rates. By convention, *sinus bradycardia* occurs when the rate falls below 60, *sinus tachycardia* when it exceeds 100.

Regularity

A rhythm is deemed regular when the separation between the beats is constant. Normal sinus rhythm often displays interbeat variability, usually related to respiration. *Sinus arrhythmia* is present when the interval between beats demonstrates cyclic shortening and lengthening exceeding 0.16 seconds (Chapter 24); this is a normal finding.

Origin and Conduction of the Stimulus

The PR interval starts when the sinus node depolarizes the atria. After the inscription of the P wave, the ECG returns to baseline as the impulse travels through the AV node (or junction), the main bundle of His, and the right and left bundle branches. When the stimulus is distributed to the ventricular myocardium by the Purkinje fiber system, the QRS starts and the PR interval ends. In normal sinus rhythm each P wave is followed by a QRS with a PR interval that is no longer than 0.20 seconds.

VENTRICULAR DEPOLARIZATION

The initial activation of the ventricles occurs on the left side of the interventricular septum and produces a vector directed anteriorly and to the right. In the limb leads (frontal plane), these initial forces are represented by the "septal" Q waves that are always less than 0.03 seconds in duration. In the precordial leads (horizontal plane), the septal forces are responsible for the initial R wave in V_1 and V_2 and for the narrow Q waves in V_5 and V_6. The remainder of the QRS is inscribed during the simultaneous depolarization of both ventricles, resulting in a total complex with a duration of up to 0.10 seconds. The intrinsicoid deflection is measured from the onset of the QRS to the moment when the downstroke of the R wave begins. This measurement is mostly used in the diagnosis of left ventricular hypertrophy (normal values in the left precordial leads do not exceed 0.045 seconds).

By convention the electrical axis of the QRS complex is measured in the frontal plane. The axis is perpendicular to the limb lead that shows an isoelectric QRS, and its polarity is indicated by the limb lead with the greatest positive defection. The value and polarity of the axis can be ascertained by referring to the hexaxial diagram (Fig. 1). An axis less than 0 degrees is called a *left axis* and is

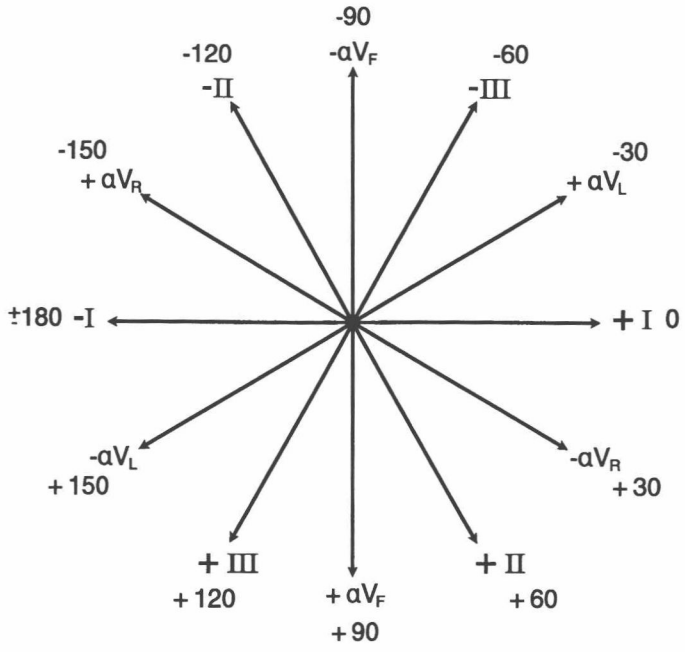


FIG. 1. Hexaxial Diagram.

considered abnormal when it exceeds -30 degrees. An axis greater than 90 degrees is a *right axis* and is considered abnormal if it exceeds 105. To provide the reader with an opportunity to practice axis calculations, the QRS axis for each ECG is provided.

REPOLARIZATION

ST Segment

The ST segment extends from the moment the ECG returns to the baseline after the QRS is completed (the so-called J point) to the beginning of the T wave. It is normally isoelectric in the limb leads. In the right precordial leads (V_1 through V_3), up to 3 mm of elevation may be normal, provided that the elevated ST segment displays a normal upward concavity.

T Wave

The repolarization of the ventricles, during which the T wave is inscribed, occurs in the same direction as the depolarization. In the limb leads, the orientation of the T wave resembles that of the QRS. Consequently, the axis of the T wave, which is calculated in the same manner as the QRS axis, is normally within 45 degrees of the QRS axis. This relationship, also called the *QRS-T angle,* is of special value when it is unclear if the T waves are normal or not. If the QRS-T angle is within the normal range, the T waves are normal.

The precordial leads normally have upright T waves, with the following exceptions: (1) a T wave that is only inverted in V_1 can be normal and (2) in young adults the presence of inverted T waves in V_1 through V_3 (a normal finding in children) is referred to as a *persistent juvenile pattern* and is not by itself an indication of cardiac disease.

QT Interval

The QT interval is measured from the beginning of the QRS to the end of the T wave. Since the QT interval varies with the heart rate, a "corrected" QT, or QT_c, can be calculated by dividing the QT interval by the square root of the interval between two R waves. The normal QT_c is up to 0.41 seconds for women and up to 0.39 seconds for men.

U Wave

The origin of the U wave, which is inscribed after the end of the T wave, remains controversial. It most likely represents the repolarization potential of the Purkinje fibers. U waves usually are oriented in the same direction as the T wave and are normally most prominent in the right precordial leads (V_1 through V_3). In the remaining leads, the amplitude of the U wave is always less than 50% of the amplitude of the T wave.

PART II

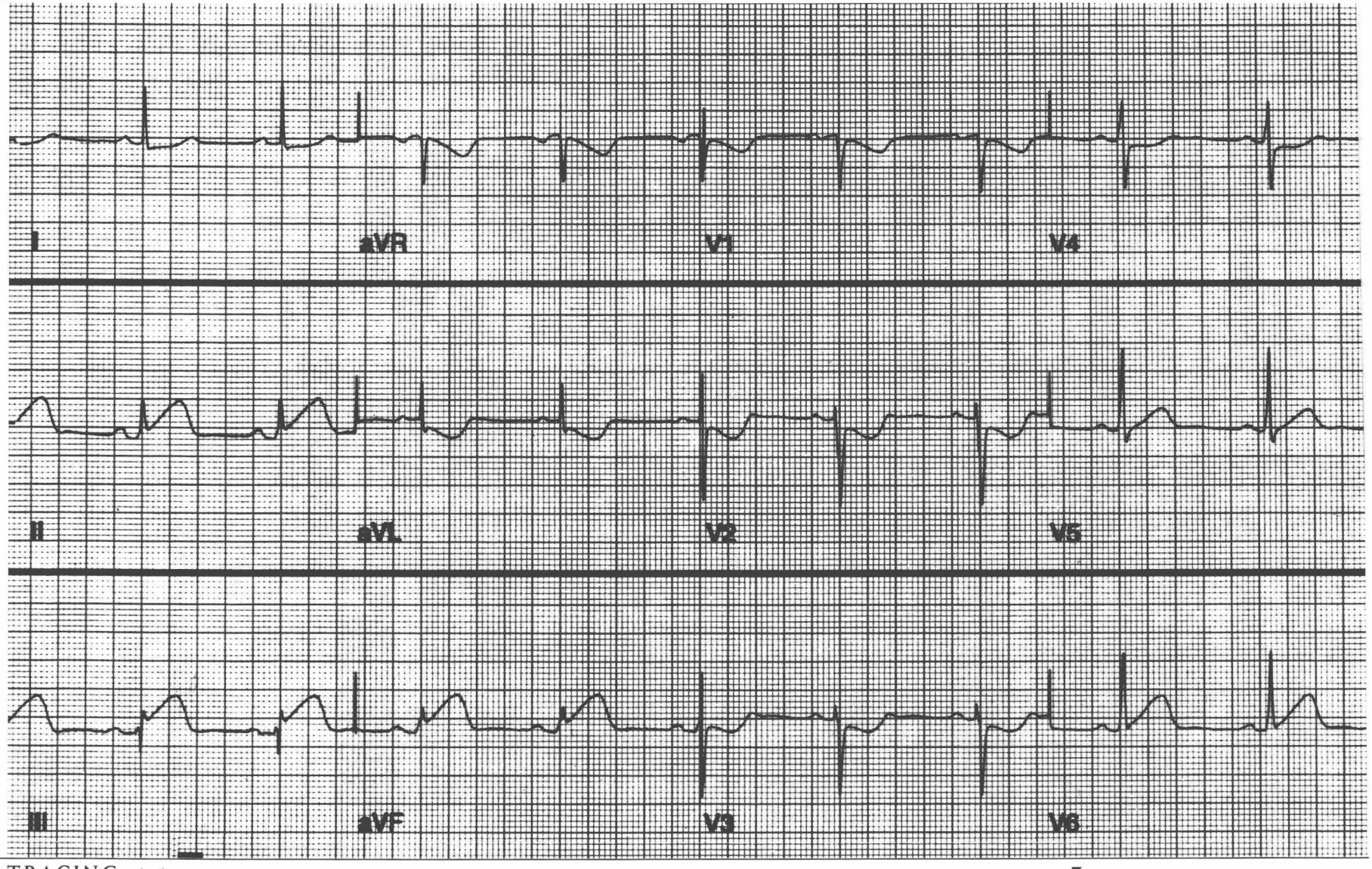
The Patient with Chest Pain

CLINICAL PRESENTATION:

A 50-year-old man is awakened from sleep by the sudden onset of severe retrosternal chest pain associated with diaphoresis and nausea.

ECG FINDINGS (TRACING 1-1):

1. Sinus rhythm at a rate of 60 beats per minute
2. ST segment elevation in the inferior leads and lateral precordial leads
3. Reciprocal ST segment depression in I and aV_L
4. ST segment depression in the right precordial leads
5. QRS axis: +30 degrees



TRACING 1-1

Acute Transmural Inferolateral Infarction

ECG INTERPRETATION:

- Acute, transmural inferolateral myocardial infarction.

DISCUSSION:

The prime ECG manifestation of acute myocardial ischemia is a shift of the ST segment. The ST segment becomes elevated with *transmural ischemia* and depressed with *nontransmural ischemia* (Chapter 3). In addition, during the "hyperacute" phase of transmural ischemia the T waves become tall and peaked (Fig. 1-1, *A*). The subsequent ECG changes depend on the outcome of the ischemic insult. If adequate coronary blood flow is restored, the ST segment returns to baseline and the T waves initially normalize. Later the T waves may become symmetrically inverted. Examples of clinical syndromes associated with this ECG sequence are an episode of transient coronary artery spasm (Prinzmetal's angina) or an attack of unstable angina. Subsequent myocardial necrosis can be established by serial serum enzyme determinations. Conversely, if coronary blood flow is not restored and a transmural infarction develops, the elevated ST segment changes from an upward concavity to an upward convexity (as in the index ECG). With time the T wave becomes inverted while the ST segment is still elevated, resulting in a contour called *coving*, which is reasonably specific for an evolving transmural infarction (Fig. 1-1, *B*). The hallmark of a transmural infarction is the "pathological" Q wave, characterized by a duration greater than 0.03 second (Fig. 1-1, *C*). The Q waves can appear within minutes of onset or become apparent only 1 or 2 days later.

As mentioned previously, during the early stages of the ischemic process, transmural ischemia and transmural infarction cannot be distinguished electrocardiographically. In the acute setting, and by convention, an ST segment that remains elevated beyond 30 minutes is interpreted as transmural infarction and considered sufficient evidence to initiate treatment for this condition.

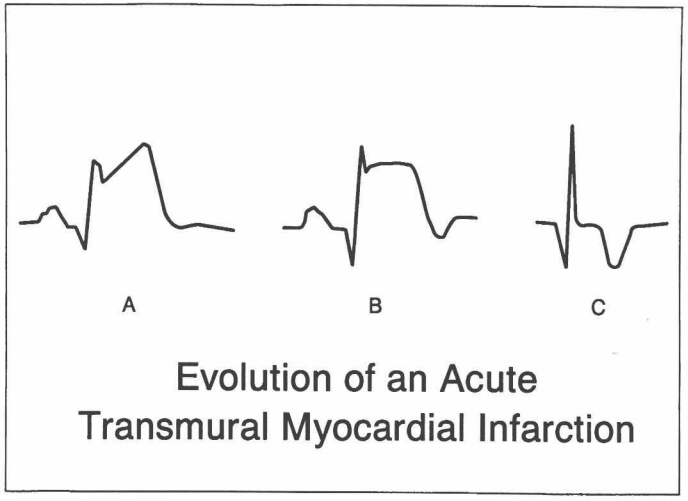


FIG. 1-1. **Evolution of an Acute Transmural Myocardial Infarction.**

An ST segment elevation associated with a transmural myocardial infarction that remains elevated for more than 2 months indicates a ventricular aneurysm involving the region reflected by the affected leads. Although this ECG finding is far from being consistently found in postinfarction aneurysms, when present it is fairly specific. Other conditions associated with ST segment elevation to be considered in the differential diagnosis are early repolarization variant (Chapter 7) and acute pericarditis (Chapter 10).

In the midst of an acute inferior wall myocardial infarction, the presence of ST segment depression in

leads V_1 through V_3 must not be construed as simple reciprocal changes. The true reciprocal changes to the ST elevation in the inferior leads is the ST segment depression in leads I and aV_L. When ST segment depressions in the right precordial leads occur during an inferior infarction, they are important markers of additional myocardium at risk, either in the septum, posterior wall, or ventricular apex. Thus they must be interpreted as indicating the potential for a large infarction and compel immediate attempts at revascularization.[1]

Whenever an inferior wall myocardial infarction is suspected, the possibility of right ventricular involvement must be entertained given the shared blood supply of these regions. An ECG should be obtained with the precordial leads placed over the right precordium, the so-called right-sided leads. ST segment elevation in any of the right-sided precordial leads confirms that the right ventricle is involved in the infarction.[2] This eventuality, which can be confirmed by bedside echocardiography, has the following potential clinical implications[3]: (1) arterial hypotension without associated pulmonary congestion that usually responds to volume loading; (2) exquisite sensitivity to the hypotensive effects of venodilators such as nitrates, morphine, and intravenous furosemide caused by the filling dependency of the damaged right ventricle; and (3) the coexistence of hypotension, distended neck veins, and clear lung fields, which pose the differential diagnosis with cardiac tamponade.

Tracing 1-2 shows ST segment elevation and Q waves in lead II, with associated ST elevation in a right-sided lead V_5, supporting the diagnosis of an inferior wall infarction with right ventricular involvement. Two related findings are (1) the presence of a complete AV block, which is a common complication of inferior wall infarction, and (2) ST segment elevation in V_1, which has also been described in right ventricular infarction.[4]

RHYTHM STRIP FINDINGS (SEE TRACING 1-2, p. 10):

1. The sinus tachycardia rate is 110 beats per minute.
2. The ventricles are driven by an independent, regular rhythm at a rate of 41 beats per minute, producing narrow QRS complexes (0.09 second).
3. An electronic ventricular pacemaker is set at a rate of 35 impulses per minute with intermittent capture.
4. All three leads (V_1, V_{II}, and right-sided V_5) show pathological Q waves, ST segment elevations, and inversion of the T waves.

ECG INTERPRETATION:

- Sinus tachycardia with complete (third-degree) AV block and an escape junctional pacemaker
- Electronic ventricular pacemaker with failure to sense
- Probable acute transmural inferior wall myocardial infarction with right ventricular involvement

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