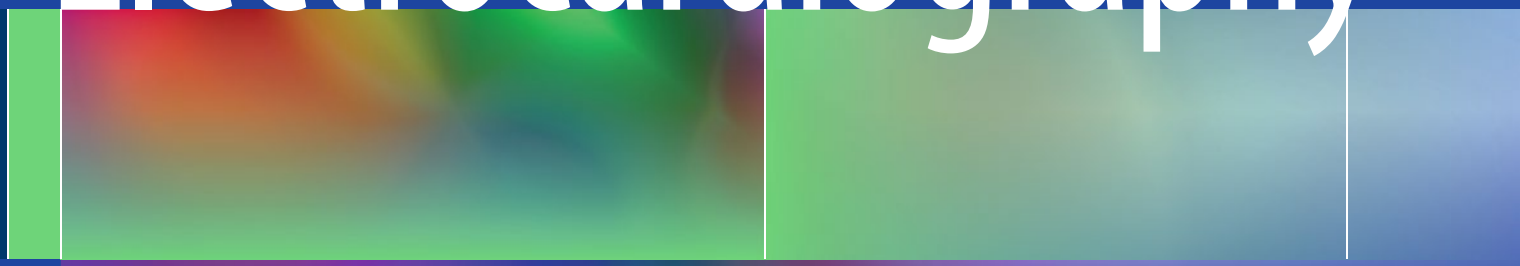
Brent G. Petty

Basic Electrocardiography



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To my wife, Joan, for her long-standing support and love.
To our four wonderful children—Elliott, Carter, Mason, and Hillary.
To the generation of medical students that I have had the pleasure to teach.

Preface

This book is intended to help students of all healthcare delivery fields and all levels of training to learn the basic concepts of interpreting electrocardiograms. While originally and primarily intended to be used by third-year medical students at The Johns Hopkins University School of Medicine, this book has also been used successfully by nurse practitioners and physician assistants who work at Hopkins as well as by nurse practitioner students in training at Hopkins.

The chapters are constructed to introduce basic themes, give examples from actual patient tracings, and then provide practice by providing self-test electrocardiograms that will reinforce the concepts taught in the chapter. Additionally, the practice tracings build on the information provided in earlier chapters as well as on the features of the current one. The citations provided in the chapters are not intended to be comprehensive. In fact, some of them are nearly 100 years old and are provided for historical interest.

The electrocardiograms shown in the book are from patients, collected over many years, and recorded in one of two ways: either as single-channel sequential tracings or, more contemporaneously, as multichannel tracings recorded and displayed in at least three leads simultaneously. I believe that seeing both types of tracings will help the student become comfortable with new tracings, as well as with those that may still be recorded one lead at a time, and with tracings from old medical records that were obtained before the simultaneous-lead methodology was developed.

One important principle for interpreting electrocardiograms is that nothing important occurs in only one beat in one lead. Important findings occur in multiple beats in multiple leads, and the leads involved are part of a group of leads that would be expected to show the same or similar changes.

An important goal of this book is to teach students the language of electrocardiograms. Like all facets of medicine, the interpretation of electrocardiograms is associated with terminology, even jargon, that has special meaning within that discipline. Becoming familiar with the terminology and the electrocardiographic appearance associated with the terms is a high priority. Clinical correlations are provided as much as applicable. On the other hand, the electrophysiological explanations for why the recordings have the appearance that they do are intentionally minimized.

While the vast majority of the tracings in this book are from my patients, I am grateful to Mr. Jim Clements, manager of The Johns Hopkins Hospital Heart Station, for several tracings that are included. Many thanks as well to my assistant, Latasha S. Graham, for her excellent work with the text, tables, and legends; to Diane Lamsback at Springer for her substantial assistance with figures and the text; and to Katherine Ghezzi at Springer for her editorial assistance.

Enjoy learning about EKGs!

Baltimore, MD — Brent G. Petty, M.D.

Contents

1 Components of the Electrocardiogram: The Normal Tracing

The heart is a remarkable organ, responsible for the coordinated pumping of blood through the pulmonary and systemic circulations. The muscular contraction of the chambers (systole), followed by a slightly longer period of relaxation (diastole), proceeds continuously, day in and day out, with precision and reliability in most circumstances. This book is an approach to the manifestations of the normal and abnormal electrical events associated with the function of the heart as a pump.

The electrocardiogram (EKG) is a graphic representation of the electrical activity of the heart. The heart's electrical waveform can be detected by electrodes placed on the surface of the body. From the initial 3 leads of Einthoven, who inaugurated electrocardiography, current EKGs are composed of 12 leads, each of which records the electrical pattern from a slightly different orientation or perspective. There are two types of leads (Table 1.1): (1) the limb leads, and (2) the precordial leads. The limb leads include Einthoven's original three, still called I, II, and III, plus the "augmented limb leads," aVR, aVL, and aVF. The precordial leads are denoted V_1, V_2, and so on through V_6. The leads are recorded by modern electrocardiography equipment by properly attaching the electrodes on the patient's surface (Fig. 1.1) and entering the patient's identifying information into the machine. The wiring in most current machines provides for recording at least 3 leads concurrently and then automatically switching to the next 3 leads until all 12 are recorded. The leads are recorded in a routine order, which is usually as follows: I, II, and III; then aVR, aVL, and aVF; next V_1, V_2, and V_3; and finally V_4, V_5, and V_6. Some machines record and display 6 leads concurrently, some all 12 leads.

When recorded in the usual format of three concurrent leads, the recorded tracing is usually displayed in a 3×4 pattern: three down and four across, and sometimes includes a rhythm strip across the bottom (Fig. 1.2).

Earlier EKG machines recorded only 1 lead at a time (single-channel), and a small section from the recorded strip from each of the 12 leads was cut from the strip and mounted on a single page. These 12 leads were typically mounted 3 across (I, II, III, then aVR, aVL, aVF, etc.) and 4 down (Fig. 1.3). The reason to include this information on the variability of EKG recording is twofold: (1) single-channel machines are still in use and (2) old EKGs in the patient's medical record could have been recorded in different ways, and one should be aware of the different fashions in which tracings may be presented.

The EKG records three electrical cardiac events: (1) atrial depolarization, (2) ventricular depolarization, and (3) ventricular repolarization. The electrocardiographic correlates of these events are called the P wave, the QRS complex, and the T wave, respectively [1] (Fig. 1.4). Atrial repolarization is not usually reflected on the EKG tracing because it occurs at about the same time as ventricular depolarization; the electrical manifestation of atrial repolarization is usually obscured by the QRS complex. Each contraction of the heart is dependent on electrical activation; the electrical event must take place before a mechanical event can occur. Depolarization must occur to cause the contraction of either the atrial or ventricular muscle fibers (i.e., systole), and repolarization must occur to allow the heart muscle to relax (i.e., diastole). The electrical events occur fractions of seconds prior to the mechanical events (Fig. 1.5). In certain situations, the electrical events occur but because of a pathological condition the mechanical events do not follow ("electrical–mechanical dissociation" or "pulseless electrical activity").

To summarize, the P wave is the electrical manifestation of atrial depolarization, not atrial systole; the QRS complex is the electrical manifestation of ventricular depolarization, not ventricular systole; and the T wave is the electrical manifestation of ventricular repolarization, not ventricular diastole. One systole/diastole, contraction/relaxation, P/QRS/T constitutes a cycle, the "cardiac cycle," which repeats an average of about 80 times per minute.

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Table 1.1 Electrocardiographic leads

Limb	Precordial
I, II, III	V_1, V_2, V_3
aVR, aVL, aVF	V_4, V_5, V_6

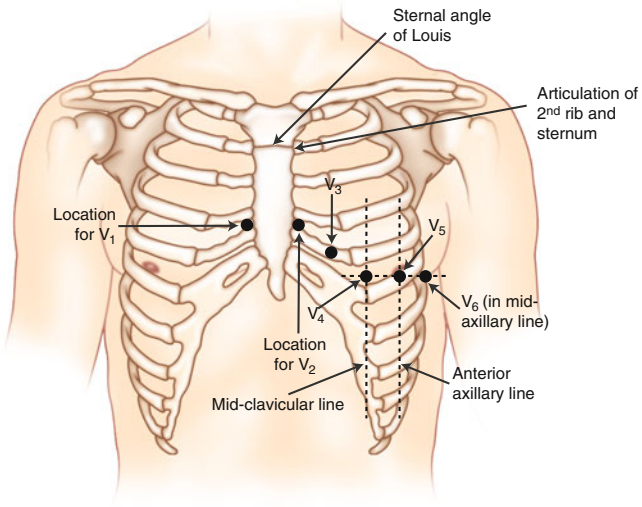


Fig. 1.1 Landmarks for placement of precordial leads

I	aVR	V_1	V_4
II	aVL	V_2	V_5
III	aVF	V_3	V_6

Fig. 1.2 Usual orientation of leads on the EKG taken with current machines, recording three leads simultaneously

I	II	III
aVR	aVL	aVF
V_1	V_2	V_3
V_4	V_5	V_6

Fig. 1.3 Typical orientation of leads on EKGs recorded one lead at a time

As one can see, the P and T waves are rounded or curved, while the QRS is sharp or spiked (see Fig. 1.4). The QRS complex can be a variety of shapes and sizes. These various configurations can be described by using the letters Q, R, and S with their associated definitions. The Q wave is the part of the QRS complex that is a negative (downward) deflection that may initiate the QRS complex. The R wave is the first positive (upward) deflection of the QRS complex, and the S wave is a negative deflection after the R wave. If another positive deflection follows the S wave, it is called R′ ("R-prime"). Not all QRS complexes have a Q, R, and S wave. In fact, a QRS complex may have only one of these waves. A positive deflection alone for the QRS is called an R wave, while only a negative deflection is called a QS wave, a notation that specifically indicates the absence of any positive deflection or R wave (Fig. 1.6). But even when only an R wave is present, for example, the whole complex is still called a QRS complex as a general term.

Occasionally one sees the letters of the QRS complex written with some letters in lower case and other letters capitalized. This is a convention used to reflect the relative size of each of the components of the QRS complex. Thus, a "Q" wave is deeper and wider than a "q" wave; an "R" wave is taller and wider than an "r" wave; and an "S" wave is deeper and wider than an "s" wave. Proper terminology can also describe the relative size of one component of the QRS compared to the other components. When one sees "qRs" written to describe a complex, one should expect to see a small initial downward deflection, followed by a tall, wider upward deflection, and a small terminal downward deflection. Examples of various QRS configurations are given in Fig. 1.7.

Sometimes people use incorrect nomenclature for the QRS complex. The R wave is *not* defined as the largest or most prominent part of the QRS complex. Rather, it is the first positive deflection of the QRS, regardless of its size. And there is no such thing as an "inverted R wave." Such a deflection would have to be a Q or S. There can be inverted P or T waves, but never inverted Q, R, or S waves.

When the EKG is recorded properly, the paper speed is 25 mm/s. Electrocardiographic paper is printed with intersecting lines 1 mm apart, and so by measuring the distance covered by each part of the cycle it is possible to determine the duration of each component (Fig. 1.8). The EKG paper has darker lines every 5 mm, and since the lines are printed both vertically and horizontally, square boxes are created. Each small box, since it is 1 mm in length and the paper speed is 25 mm/s, is equivalent to 1/25 of a second, or 0.04 s. Each large box, composed of five small boxes, is therefore equal to 5/25 of a second, or 0.2 s. Five large boxes comprise 1 s.

Small vertical marks at the top or at the bottom of the paper are separated by 15 large boxes, or 3 s. These vertical marks allow more rapid calculation of time on long electrocardiographic tracings, called "rhythm strips," either recorded concurrently in three or more leads (usually V_1, II,

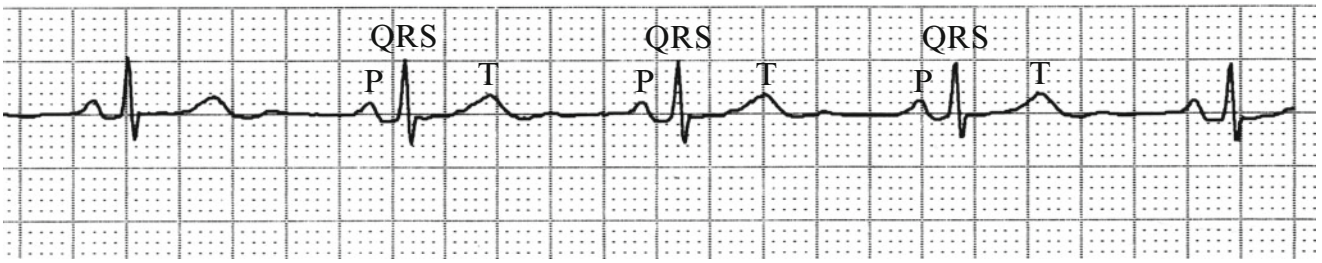


Fig. 1.4 The basic waves and complexes of the cardiac cycle. The P waves, QRS complexes, and T waves are labelled

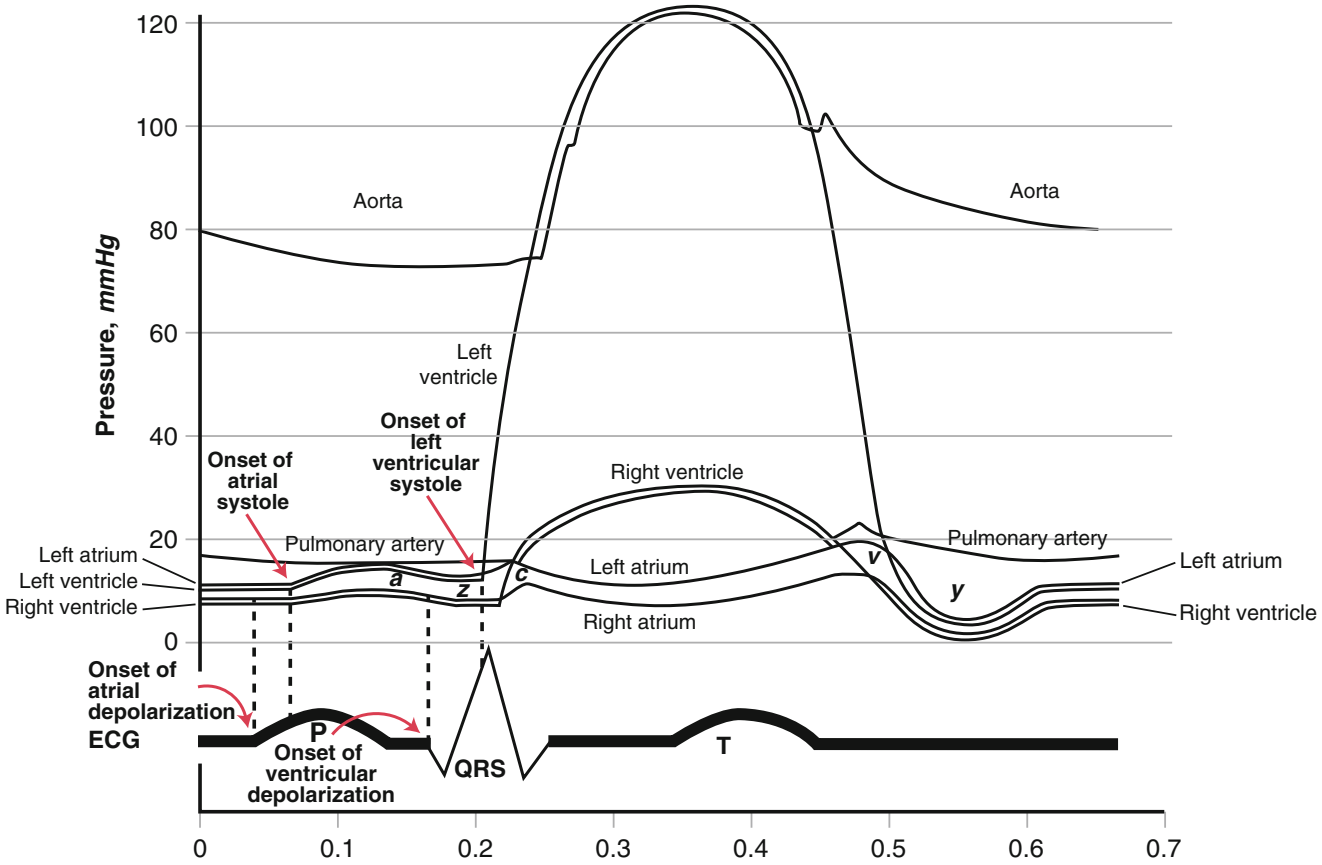


Fig. 1.5 Relationship of electrical and mechanical events in the cardiac cycle. Modified from [9]

and V_5) or in one single lead on single-channel machines (typically, lead II). When making EKG measurements, it is customary to estimate intervals to the nearest 1/4 of a small box, or 1/4 mm, and therefore to the nearest 0.01 s.

Now that you can recognize the electrical components of each cardiac cycle and know how to determine and how to time the electrical events, you are ready to set out on interpretation of the EKG. Each time you formally interpret an EKG, five principal items need to be included: (1) rate, (2) rhythm, (3) axis, (4) intervals, and (5) waveform (Table 1.2). Each of these five items will be covered separately. After you have reviewed and reported each of these five items, you provide an overall summary of the EKG. This is not a recapitulation of the information given in the five areas above, but rather a brief statement which provides a synthesis of the information gathered in the five categories.

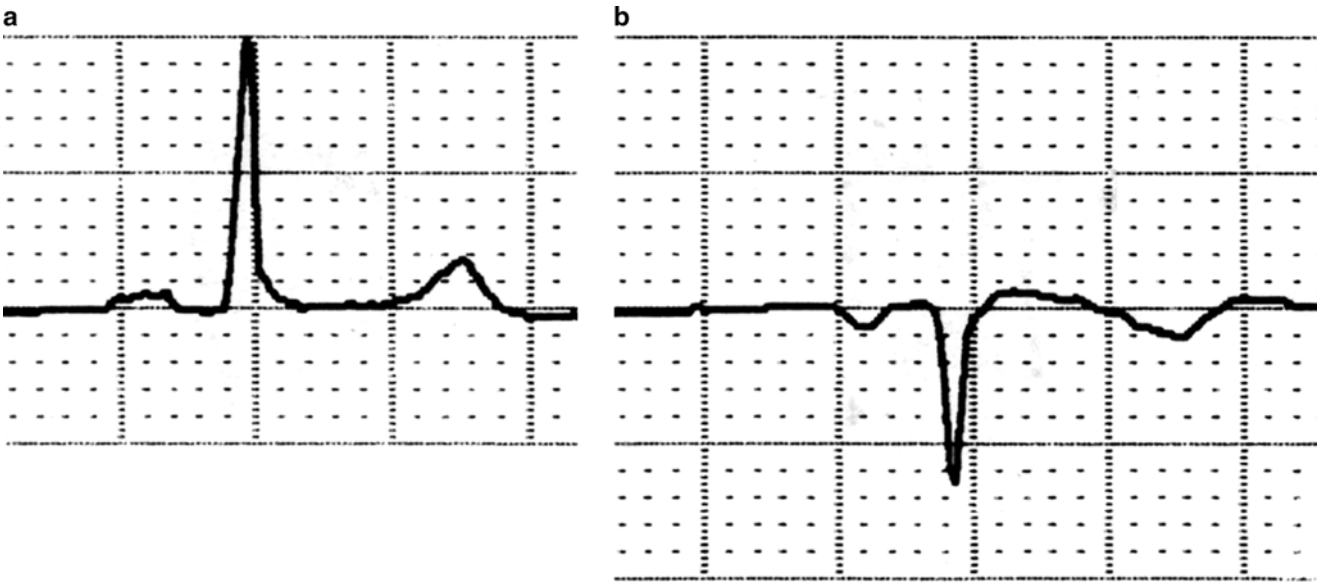


Fig. 1.6 (**a**) QRS complex comprised solely of an R wave. (**b**) QRS complex comprised of a QS wave

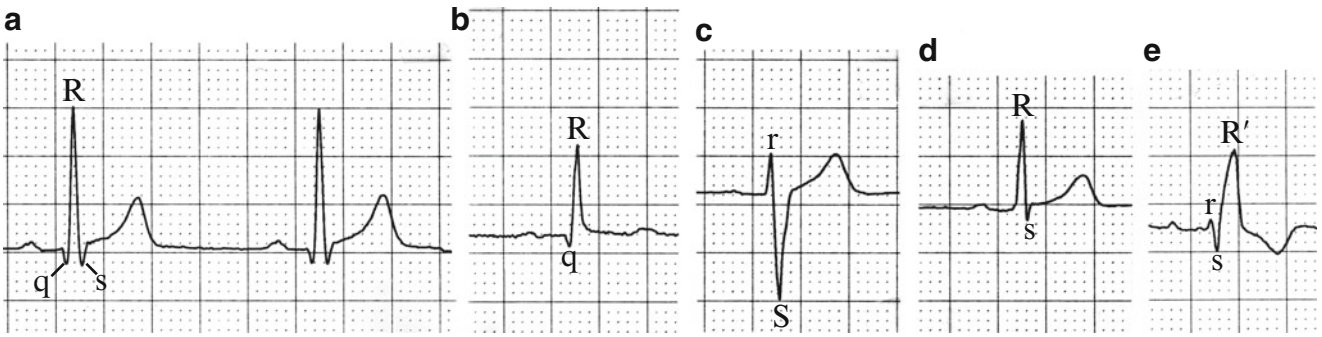


Fig. 1.7 QRS complexes: (**a**) qRs. (**b**) qR. (**c**) rS. (**d**) Rs. (**e**) rsR′

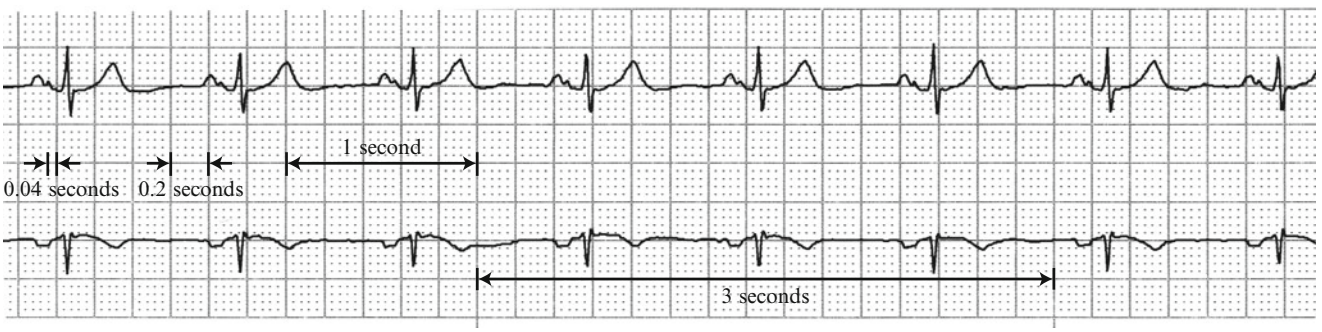


Fig. 1.8 Time/distance relationships at proper paper speed (25 mm/s)

Table 1.2 Components of the formal interpretation of an EKG

Rate
Rhythm
Axis
Intervals
Waveform
Summary

Rate

The heart rate is the number of cardiac cycles per minute. Two similar methods to determine heart rate are (1) recording a 60-s strip and simply counting the number of P waves or QRS complexes included, or (2) by recording a 6-, 10-, 15-, or 30-s rhythm strip, counting the number of complexes present, and multiplying by the correct number to convert to beats per minute. Both of these methods are tedious and hardly ever done, except in the presence of an irregular rhythm. Instead, the heart rate can be determined from the time elapsed between successive beats and converting this time to beats per minute. It is customary to determine both the atrial and ventricular rates. Usually these are the same, but in certain arrhythmias they are different. The atrial rate is determined by measuring the P–P interval (the distance between consecutive P waves), and the ventricular rate is determined by measuring the R–R interval (the distance between consecutive QRS complexes), and then converting the intervals into beats per minute by dividing 60 by the P–P or R–R interval. When the atrial and ventricular rates are the same, as in normal sinus rhythm, one can measure just the R–R interval and determine the ventricular rate, which will be equal to the atrial rate.

The most accurate method to determine heart rate is to measure the R–R interval in seconds and divide that interval into 60 (Fig. 1.9). This is easily accomplished with a pocket calculator or computer, less easily by long division, and perhaps most easily by using Table 1.3.

A good estimate of the heart rate, which doesn't require a calculator, long division, or dependence on a table, is achieved by simply measuring the R–R interval in units of "number of big boxes," and dividing that number into 300 (Fig. 1.10). If the R–R interval is four big boxes in duration, then the heart rate is 300/4, or 75 beats per minute. If the R–R interval is five big boxes, the heart rate is 300/5, or 60 beats per minute. If the R–R interval is not a whole number of boxes, the rate can be estimated by whether its duration is closer, for example, to four boxes (75) than to five boxes (60). Since this is only an estimate, an error of 5–10 beats per minute is to be expected. This method should not be used when formally interpreting an EKG. The more exact methods given above (using the measured R–R interval to the nearest 0.01 s and then

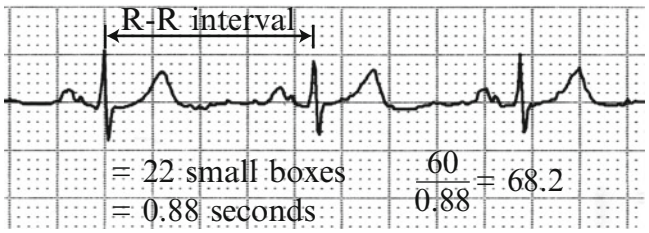


Fig. 1.9 Determining the heart rate. The R–R interval in this example is 18 small boxes, or 0.72 s, since each small box = 0.04 s. To determine the heart rate, divide 0.72 into 60, which is 83.3, and round off to the nearest whole number. With an R–R interval of 0.72 s, the heart rate is 83 beats per minute

Table 1.3 Converting R–R interval to heart rate

R–R interval	Heart rate	R–R interval	Heart rate
0.40	150	0.81	72
0.41	146	0.81	73
0.42	143	0.83	72
0.43	140	0.84	71
0.44	136	0.85	71
0.45	133	0.86	70
0.46	130	0.87	69
0.47	128	0.88	68
0.48	125	0.89	67
0.49	122	0.90	67
0.50	120	0.91	66
0.51	118	0.92	65
0.52	115	0.93	65
0.53	113	0.94	65
0.54	111	0.95	63
0.55	109	0.96	63
0.56	107	0.97	62
0.57	105	0.98	61
0.58	103	0.99	61
0.59	102	1.00	60
0.60	100	1.01	59
0.61	98	1.02	59
0.62	97	1.03	58
0.63	95	1.04	58
0.64	94	1.05	57
0.65	92	1.06	57
0.66	91	1.07	56
0.67	90	1.08	56
0.68	88	1.09	55
0.69	87	1.10	55
0.70	86	1.11	55
0.71	85	1.12	54
0.72	83	1.13	53
0.73	82	1.14	53
0.74	81	1.15	52
0.75	80	1.16	52
0.76	79	1.17	51
0.77	78	1.18	51
0.79	77	1.19	50
0.80	75	1.20	50

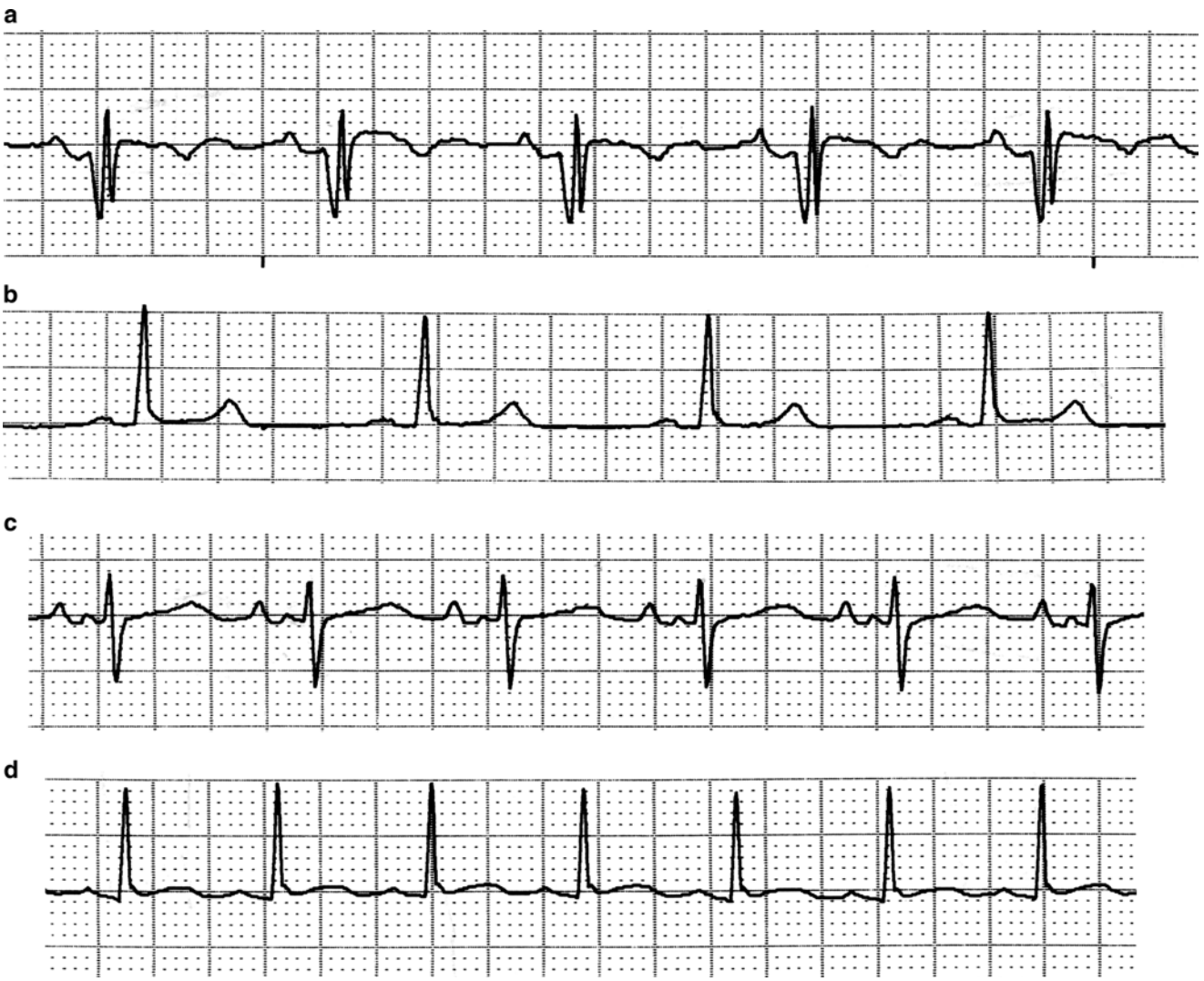


Fig. 1.10 Estimation of heart rate. (**a**) R–R interval is about 4 big boxes, so rate is about 300/4 = 75 (actual rate 71). (**b**) R–R interval is just over 5 boxes, so rate is slightly slower than 300/5 = 60 (actual rate = 59). (**c**) R–R interval is between 3 and 4 big boxes, so heart rate is between 300/3 = 100 and 300/4 = 75. The R–R interval is about halfway between 3 boxes and 4 boxes, so rate is about halfway between 100 and 75, or about 87 (actual rate = 85). (**d**) R-R interval is slightly less than 3 big boxes, so heart rate is slightly more than 300/3 = 100 (actual rate = 107)

dividing into 60, or using Table 1.3) is more appropriate for comprehensive interpretations.

The normal heart rate is 60–100 beats per minute. Rates slower than 60 are by definition bradycardias, and rates faster than 100 are tachycardias.

Rhythm

Most EKGs show normal sinus rhythm, which is a rhythm in the normal range for rate and with each P wave followed by a constant PR interval and a QRS complex. There are a large variety of rhythms other than normal sinus rhythm, which are covered in detail in Chap. 7.

Axis

The electrical axis is the average direction of electrical activity and is discussed in detail in Chap. 2.

Intervals

The intervals should be measured in the limb leads rather than in the precordial leads, since the normal ranges for intervals have been established from the limb leads. Generally, the limb leads with the most obvious and well-demarcated P waves, QRS complexes and T waves, plus the longest intervals on inspection, are used for the interval measurements.

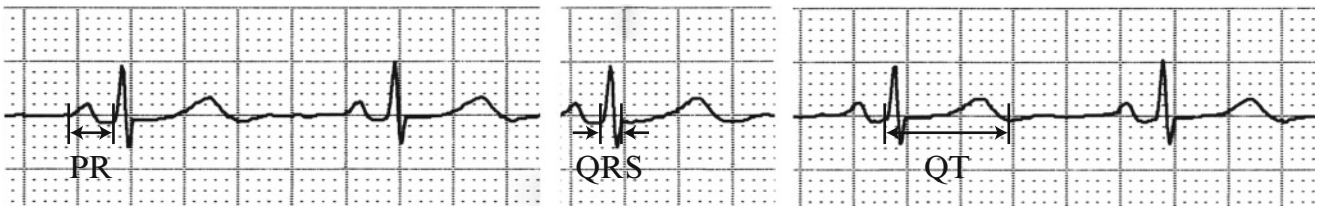


Fig. 1.11 Intervals. (**a**) PR interval. (**b**) QRS duration. (**c**) QT interval

Three intervals should be measured for every EKG: (1) PR interval, (2) QRS duration, and (3) QT interval (Fig. 1.11). The PR interval is from the beginning of the P wave to the beginning of the QRS complex. The normal PR interval ranges from 0.12 to 0.20 s. Either shorter or longer is abnormal. The QRS duration should be less than 0.12 s. There is no lower limit of normal to the QRS complex, but it is usually at least 0.04 s. Shorter deflections following the P wave may suggest artifact or pacemaker pulses. The QT interval varies with the heart rate; this observation is accurate primarily in situations of changed heart rates associated with exercise, hyperventilation, and presumably other situations of increased sympathetic nervous system stimulation [2]. The QT does not seem to change substantially with changes of heart rate that are mediated by vagal stimulation. The QT interval is indirectly proportional to the heart rate at rapid rates, so that as the heart rate increases the QT interval gets shorter, and as the heart rate decreases back into the normal range the QT interval gets longer. There are tables showing the normal range of QT intervals for various heart rates, and one must check the tables to get the specific range of QT interval that is normal for a certain heart rate. Another practice has been to use the Bazett correction of the QT interval [3] using the following formula:

$$\frac{QT}{\sqrt{R-R}}$$

where QT is the measured QT interval and R–R is the R–R interval in seconds. This formula gives a "corrected" QT interval (QT_c). The normal corrected QT interval is about 0.36–0.40 s, and this calculation allows one to mathematically adjust for the difference in QT interval induced by rate. As mentioned above, however, the association of decreased QT interval with increased heart rate is usually seen with increases in heart rate induced by exercise, hyperventilation, and presumably other conditions that stimulate the sympathetic nervous system, while not in other situations. A rule of thumb that can be used for the QT interval is that the QT interval should be less than half of the corresponding R–R interval. If the QT interval is less than half of the corresponding R–R interval, then it is highly likely that the QT interval is not too long. If the QT interval is longer than half of the R–R interval, then one should be suspicious that it may be too long. This rule of thumb is less reliable with rapid heart rates, where the QT interval may be longer than half of the R–R interval and still be within the normal range. The observed or measured QT interval without correction may be written "QT_o," but it is understood that "QT" written without any subscript refers to the measured, uncorrected QT interval. Another method of correcting the QT interval is using the Fridericia correction, which is the QT interval divided by the cube root of the R–R interval [4]. There are proponents of both corrections.

In addition to varying with heart rate, the QT interval is a little shorter in men than women, and it correlates inversely with serum calcium [5, 6] and serum magnesium concentrations. Many drugs (e.g., quinidine, procainamide, phenothiazines) can increase the QT interval.

Waveform

Waveform refers to the configuration of the QRS complex, ST segments, T waves, and precordial R wave progression. Q waves are not abnormal if they are small. The Q wave is abnormal if it is longer than or equal to 0.04 s in duration. It is primarily width, not depth, that is important for identifying a "pathological Q wave." It is important to keep in mind that Q waves (0.04 s or more) can be normal in some leads (Fig. 1.12). Lead aVR normally may have just a QS wave (QS because there is no R) or a Qr. A QS configuration may also be found in V_1. It is also normal to have what is called a "diaphragmatic" Q wave in lead III, which can be wide enough to suggest "pathological," but it is not. Instead, it is a normal variant (Fig. 1.13). If there is a wide Q wave in lead III but no q wave at all in leads II or aVF (the other "inferior" leads), don't suspect that the patient has an abnormality, but rather that the patient has a normal diaphragmatic Q wave. In leads other than III, aVR, and V_1, wide Q waves are usually abnormal.

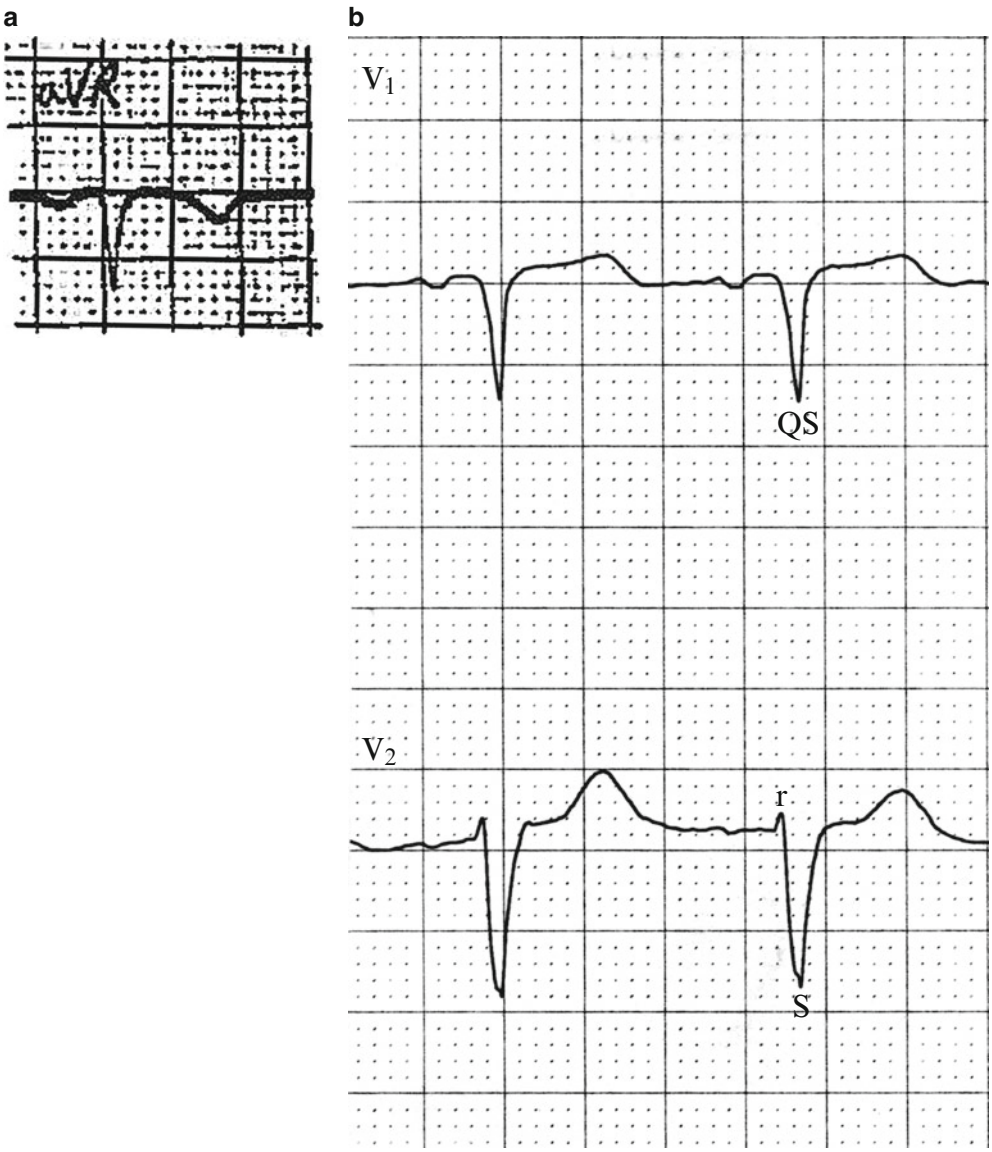


Fig. 1.12 "Normal" Q waves. (**a**) Lead aVR has a QS configuration as part of this patient's normal EKG. (**b**) Lead V_1 shows a QS configuration, but V_2 has an rS, which is compatible with normal QRS complexes in the early precordial leads

The QRS complex should contain an R or S wave in a limb lead of at least 5 mm, or an R or S wave in a precordial lead of at least 15 mm. When neither of these criteria are met, "low QRS voltage" is present. Low QRS voltage is seen in association with pericardial effusion, Addison's disease, severe lung disease with increased air between the heart and chest wall, severe obesity with increased soft tissue between the heart and chest wall, hypothyroidism, or infiltrative diseases of the heart (e.g., amyloidosis, sarcoidosis, hemochromatosis).

"R wave progression" refers to the appearance of the R waves in the precordial leads. Normal R wave progression is seen in Fig. 1.14. As mentioned above, there may or may not be an r wave in V_1. If there is not, it is not abnormal. Lead V_2, however, should have an r wave of some magnitude. If there is no r wave in V_2, that is usually abnormal. The R wave characteristically gets taller in absolute amplitude between V_1 and V_4 and then gets smaller from V_4 to V_6. The R:S ratio, comparing the relative size of the R and S waves, becomes greater across the precordial leads, from V_1 to V_6. There should be little or no s wave in V_6. The "transition point," where the R wave becomes larger than the S wave, is normally at or between V_3 and V_4.

There can be four varieties of deviation from normal R wave progression: (1) the R wave transition can be "early," where the R wave transition (R>S) occurs between V_2 and V_3 (Fig. 1.15). This suggests either that (a) the leads were placed wrongly (too far to the patient's left), or (b) the patient's heart is simply turned counterclockwise in the chest (counterclockwise from the perspective of looking up at the

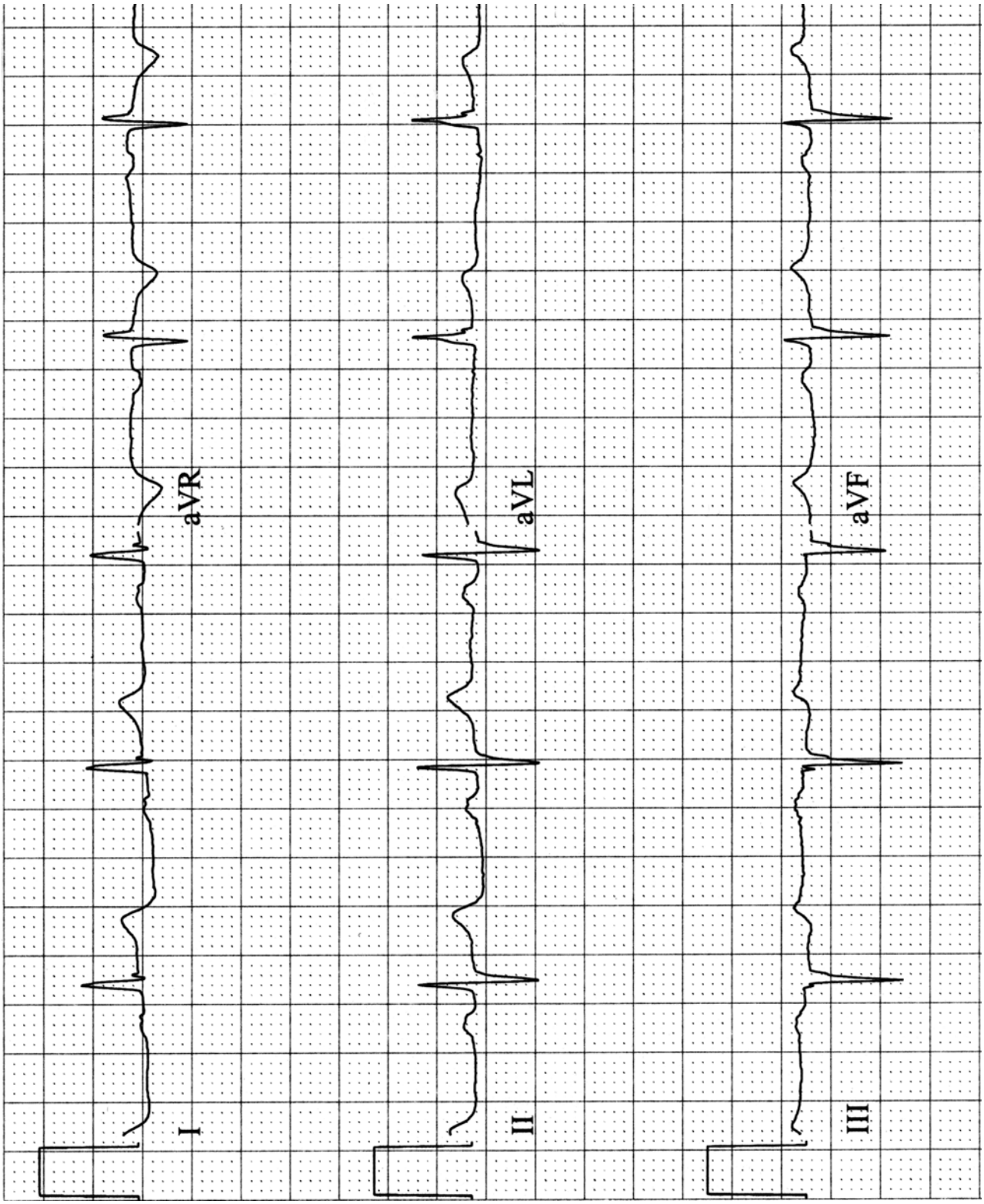


Fig. 1.13 Diaphragmatic Q wave. Note that there is a Q only in Lead III, while no Q wave is present in II or aVF

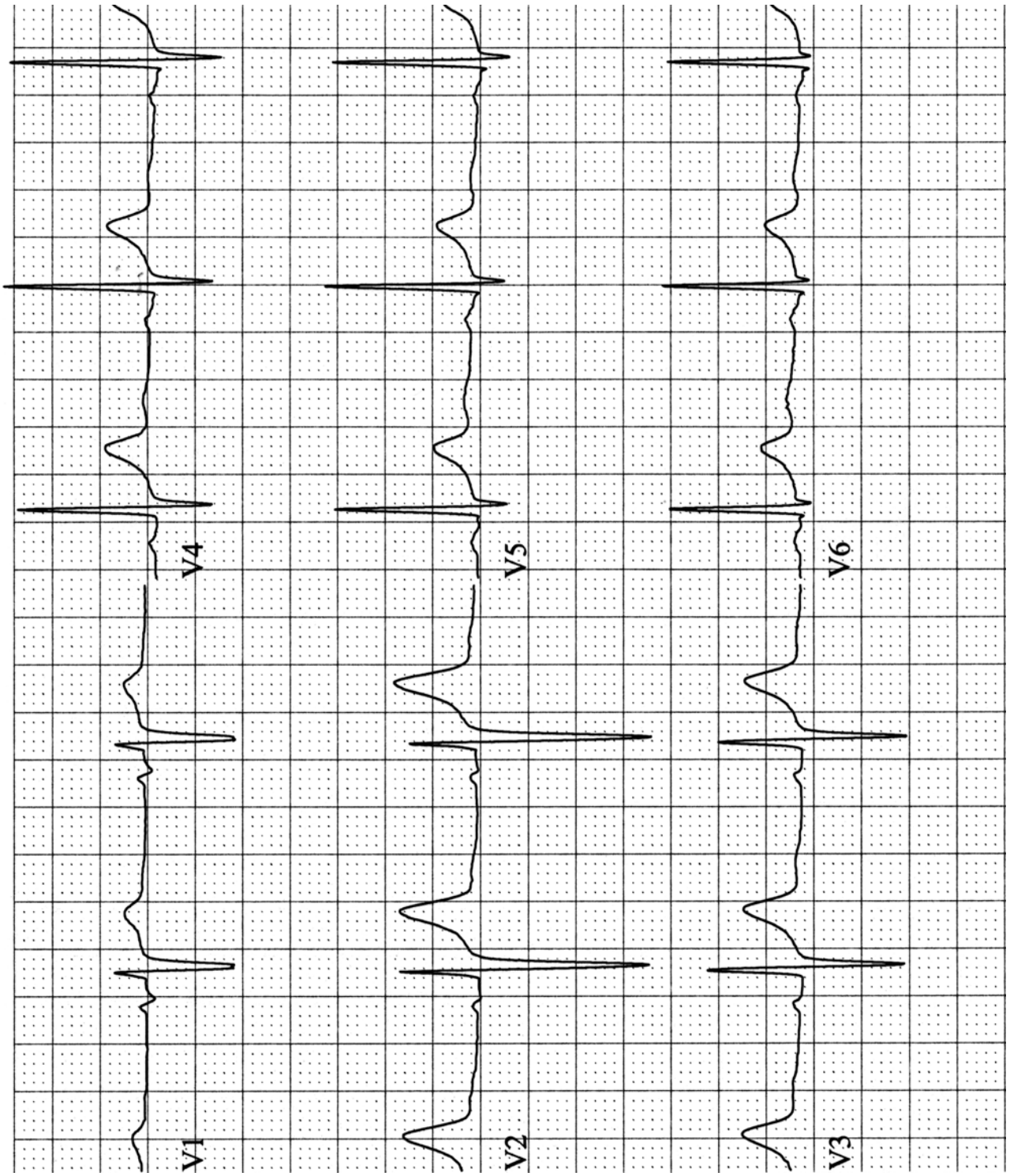


Fig. 1.14 Normal R wave progression

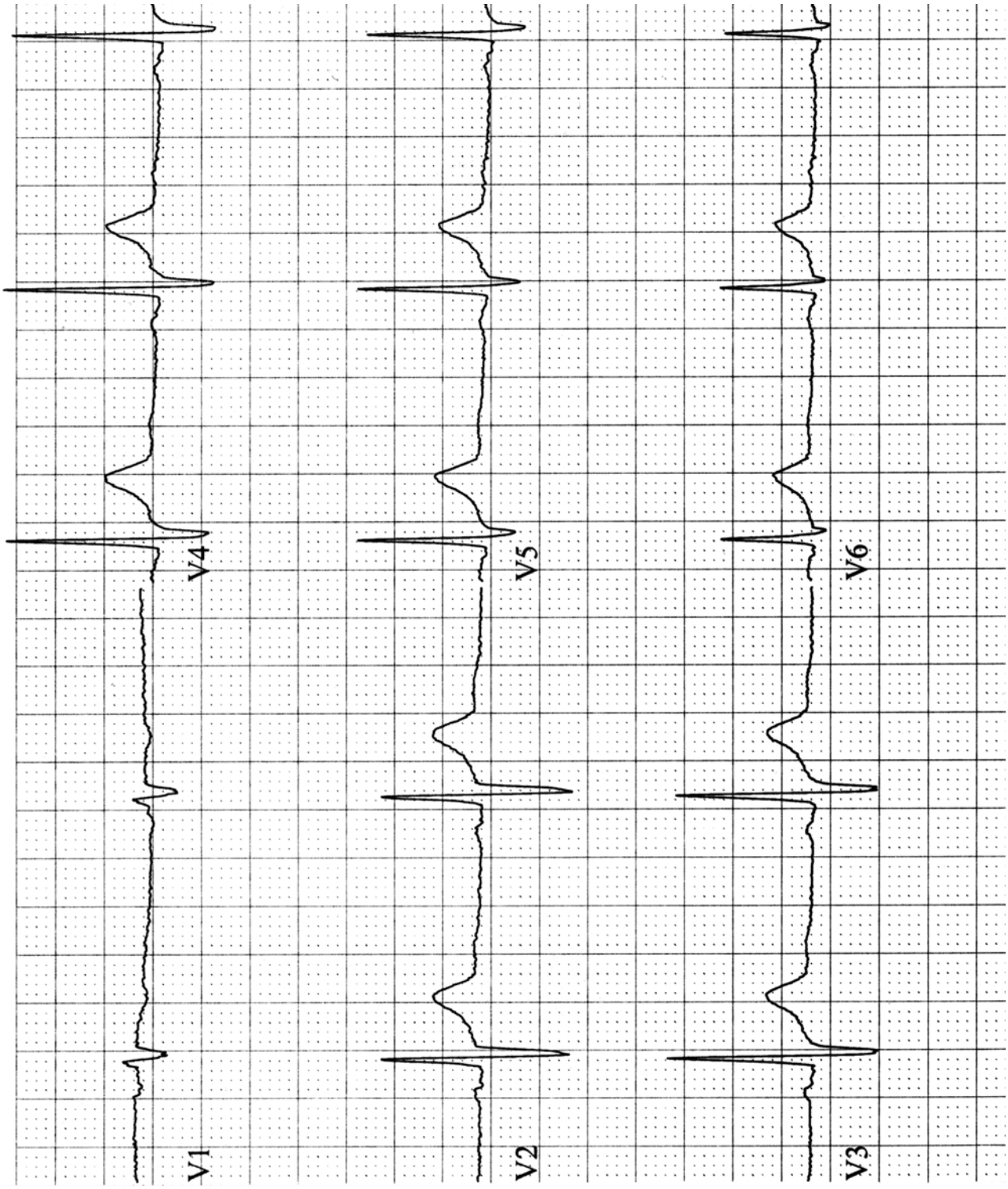


Fig. 1.15 Early R wave transition. The R wave is nearly equal to the S wave in V_2, and the R wave is greater than the S wave in V_3

thorax from the feet) and the left ventricle is oriented farther to the right than usual. (2) The R wave can be greater than the S wave in V_2 as well as V_3, which is "early R wave development" with the same two possible, rather innocent, explanations. (3) If the R wave is greater than the S wave in V_1, that is always abnormal, reflecting a pathological condition such as right ventricular hypertrophy, right bundle branch block, posterior infarction, or Wolff–Parkinson–White syndrome, Type A. (4) The last deviation from normal R wave progression is the opposite of early R wave development, namely poor R wave progression. This means that there are smaller than normal R waves from V_1 to V_3 and perhaps a small R wave ($R < S$) in V_4 and, rarely, even to V_5 and/or V_6 (Fig. 1.16). Poor R wave progression can be due to processes just the opposite of early R wave progression; that is, either (a) the precordial leads were placed too far to the patient's right, or (b) the patient's heart is turned clockwise in the chest, with the left ventricle closer to the left axilla, or (c) there is a pathological condition such as a loss of muscle mass in the anterior wall of the heart, perhaps by means of a heart attack. It must be emphasized that both early R wave progression and poor R wave progression are nonspecific findings, in that they can occur from a number of causes, as enumerated above.

The ST segment should be at the same level as the baseline (i.e., the flat part of the recording between the end of the T wave and the beginning of the next P wave). Variations of up to 1 mm above or below the baseline are considered normal. While ST segment elevation is often abnormal (see Chap. 3), there are some instances where it is not. Especially in the early precordial leads (V_1 to V_3) one can often see what is called junctional ST elevation (Fig. 1.17). The ST segment begins a little bit above the baseline. This junctional ST elevation can be as much as 2 or 3 mm above baseline, but as long as it has a smooth, curving configuration it is usually a normal variant.

Another normal variant is early repolarization, where the ST segment can be elevated above the baseline, most commonly in the lateral precordial leads (Fig. 1.18). The term "early repolarization" implies that ventricular repolarization begins earlier than normal, and as a consequence the ST segment becomes elevated, essentially reflecting the upslope of the T wave. The ST segments in early repolarization are smoothly curved at the junction of the QRS complex and ST segment, as contrasted with the sharp angle typically seen in patients with myocardial infarction (Chap. 3). "Junctional ST elevation" and "early repolarization" are essentially the same finding, simply observed in the early ($V_{1–3}$) or late ($V_{4–6}$) precordial leads, respectively. Some publications have suggested that early repolarization may not be the normal variant previously thought, but may be associated with increased cardiovascular mortality, particularly if the early repolarization is found in the inferior leads [7, 8]. Clinical correlation and review of old tracings, if available, are very important with ST segment elevation. More distinction of abnormal ST elevation from normal variants is covered in Chap. 3.

In the limb leads, normal T waves follow the same general direction as the QRS complexes. Thus, ventricular repolarization is usually in the same general direction as ventricular depolarization. Chapter 2 deals with determination of axis, and one can determine not only the QRS axis but also the P wave axis and the T wave axis. The P wave axis is normally between 0° and +90°. The T wave axis should be within 60° of the QRS axis. When the T wave axis is not within 60° of the QRS axis, then that is abnormal. There may be limb leads where there is a positive QRS complex and a negative T wave, but that would be normal if the T wave axis was still within 60° of the QRS axis. In the precordial leads, the T waves should be upright in leads $V_{2–6}$, regardless of the configuration of the QRS. In V_1, the T waves can be upright, flat, or inverted, and all are normal. The T wave should be less than 10 mm in height. When T waves are 10 mm tall or more in multiple leads, that is suggestive of hyperkalemia. On the other hand, flat T waves are a nonspecific abnormality, but they may occur in patients with hypokalemia.

The U wave is a usually positive, curved wave that follows the T wave (Fig. 1.19). While U waves have been associated with hypokalemia, they may also be seen as a normal variant.

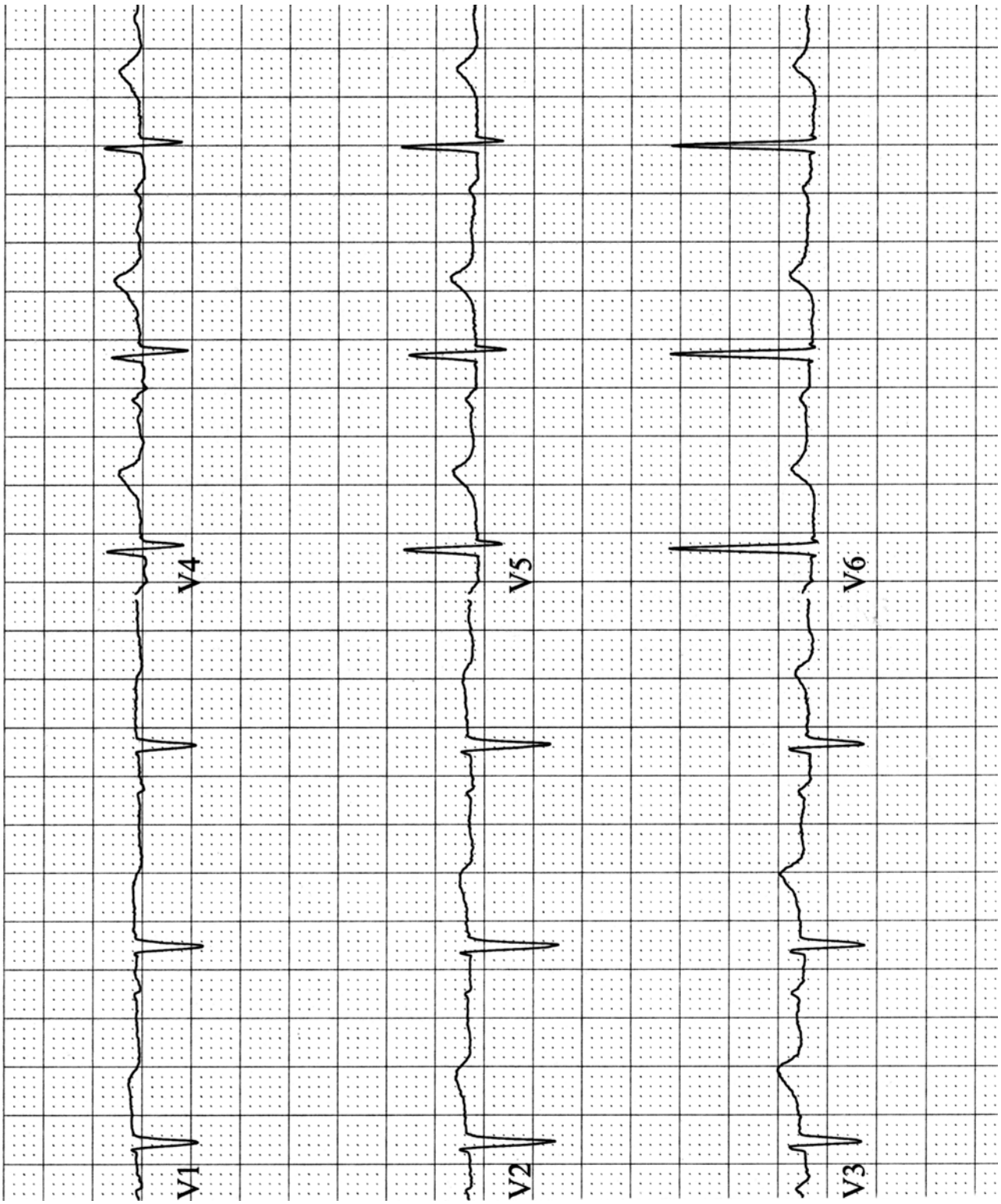


Fig. 1.16 Poor R wave progression. The R wave is smaller than the S wave through V_4, and R wave transition occurs between V_4 and V_5

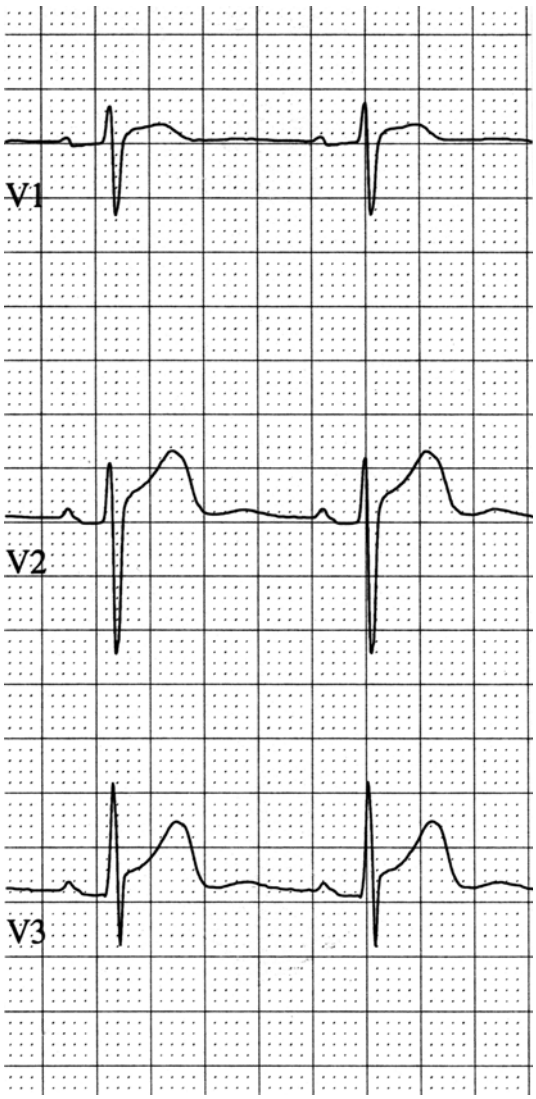


Fig. 1.17 Junctional ST elevation. Note that the "take-off" of the ST segment from the QRS complex is 1–2 mm above the baseline in $V_{1–3}$

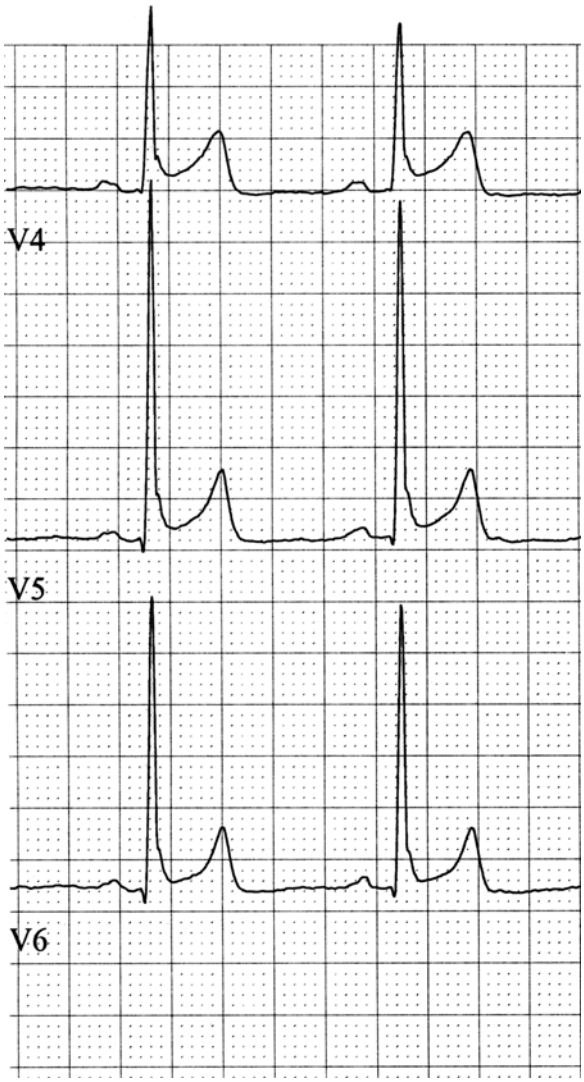


Fig. 1.18 Early repolarization. The ST segments are 1–2 mm above the baseline in $V_{4–6}$

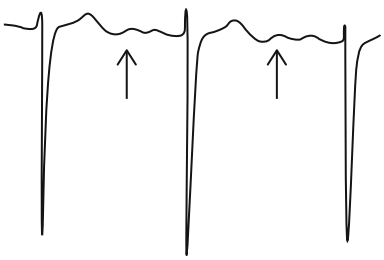
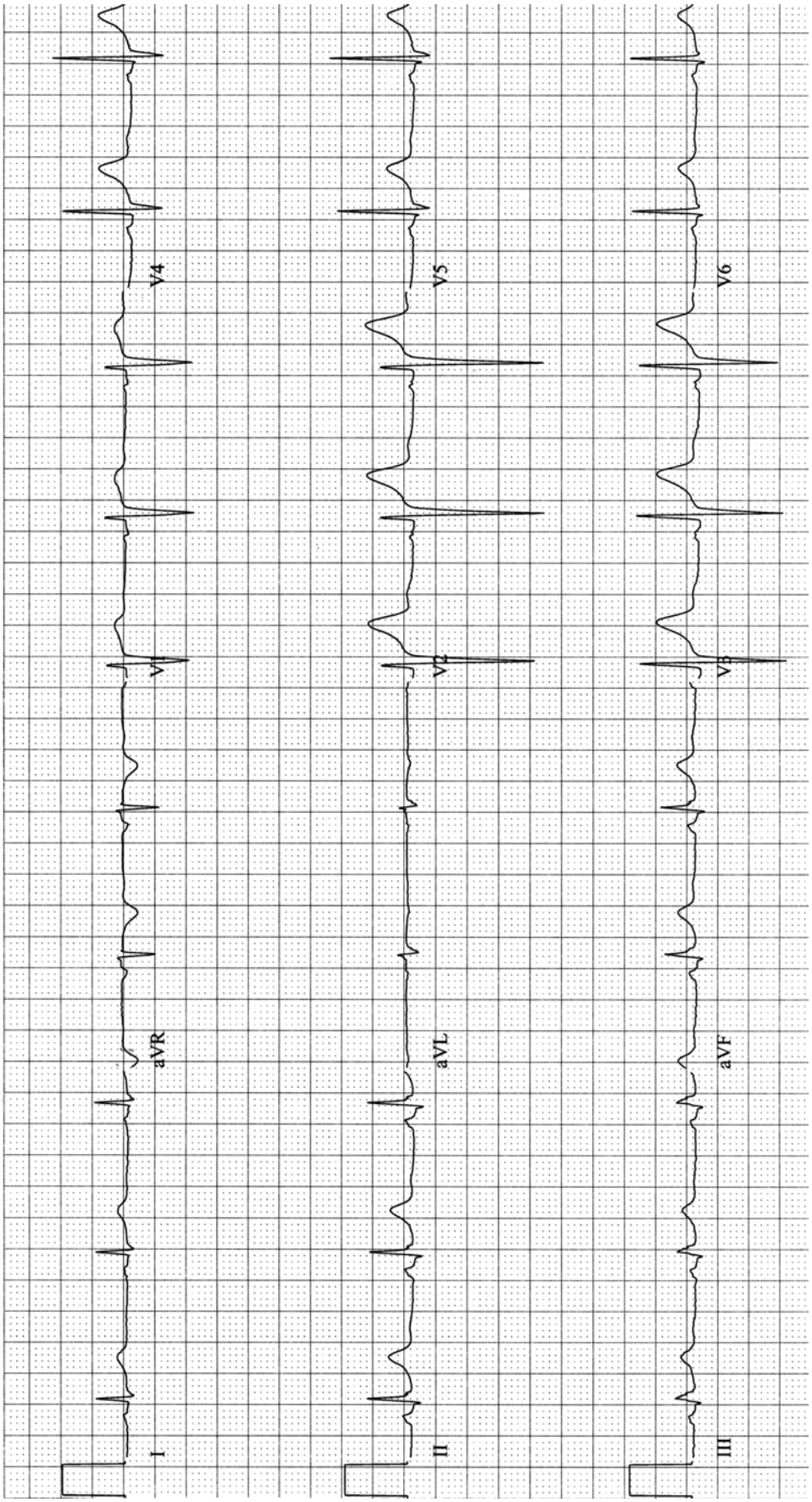


Fig. 1.19 U waves (*arrows*)

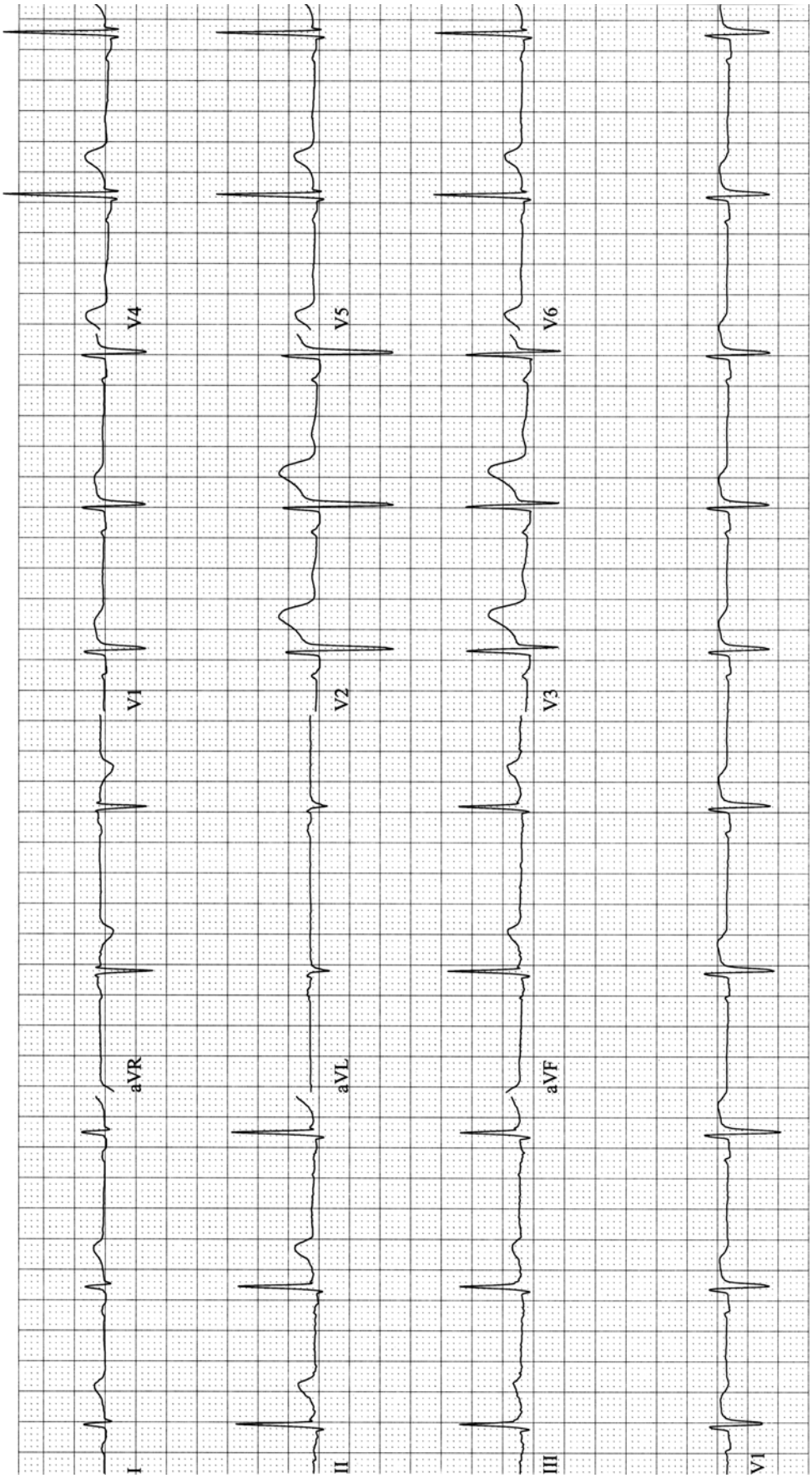
Exercise Tracings

At the end of each chapter there will be several electrocardiograms for you to interpret as a method of practice and review. There is space at the bottom of each tracing for you to write your interpretation, and the correct answer will be provided at the end of the tracings. The tracings will not only include material from the chapter just concluded but will also relate to material covered in previous chapters.

Exercise Tracing 1.1



Exercise Tracing 1.2



Interpretations of Exercise Tracings

Exercise Tracing 1.1	
RATE:	A 64 V 64
RHYTHM:	Normal sinus rhythm
AXIS:	+65°
INTERVALS:	PR 0.14 QRS 0.09 QT 0.38
WAVEFORM:	Unremarkable
SUMMARY:	Normal tracing

Exercise Tracing 1.2	
RATE:	A 58 V 58
RHYTHM:	Sinus bradycardia
AXIS:	+75°
INTERVALS:	PR 0.16 QRS 0.09 QT 0.38
WAVEFORM:	R>S in V_3, ST elevation V_{2-4}
SUMMARY:	Sinus bradycardia, early R wave transition V_{2-3}, early repolarization

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Axis

2

The electrical axis of any electrocardiogram (EKG) waveform is the average *direction* of electrical activity. It is not a vector, because by definition a vector has both direction and amplitude, while axis has only direction. While the axis of any of the waves (P, QRS, T) can be determined, the term "axis," unless otherwise specified, refers to the axis of the QRS complex.

One determines axis from the six limb leads only. These include the bipolar electrodes of Einthoven (I, II, and III) plus the augmented limb leads which can be thought of as bipolar electrodes with an intermediate orientation relative to leads I, II, and III. The six limb leads together constitute the "hexaxial reference system." A bipolar electrode provides for a positive, negative, or isoelectric deflection on the recording paper, depending on the orientation of the electrode and the direction of the electrical activity (Fig. 2.1).

When electrical activity is going towards the positive pole of a bipolar electrode, a positive or upright deflection is recorded on the graph paper (see Fig. 2.1a). When electrical activity is going towards the negative pole, a negative or downward deflection is recorded (see Fig. 2.1b). When electrical activity is perpendicular to the lead, no deflection is recorded (see Fig. 2.1c). Because the heart is of more than one dimension and the direction of electrical activity is not always exactly in the same direction, no totally flat bipolar record is possible. The equivalent of the flat bipolar record in electrocardiography is the "isoelectric" lead, in which an equal upward and downward deflection is recorded (see Fig. 2.1d). It must be noted that the *average* direction of electrical activity in Fig. 2.1d is the same as in Fig. 2.1c, i.e., the average direction of electrical activity in both Fig. 2.1c and Fig. 2.1d is perpendicular to the orientation of the electrode. It should also be noted that the magnitude of the deflection is judged by the *area* above or below the deflection, not the mere height or depth of the deflection. Applying this concept to each of the limb leads means that if the net deflection (positive area vs. negative area) of the wave is positive, the axis is on the positive side of the perpendicular to that lead.

The six limb leads are arranged such that their intersections equally divide the circle of the frontal plane into 30° sectors (Fig. 2.2). The positive side of each lead is labelled with the lead's identifier. As related to the expression of axis, horizontal towards the patient's left is arbitrarily designated as 0°, with positive extending downward (clockwise) and negative extending upward (counterclockwise) from left horizontal. Whenever axis is reported, the report must include either "+" or "−," except for 0° and 180°. A normal axis is between 0° and +90°, although some authors believe that the normal axis can actually extend as far to the left as −30°. Right axis deviation (RAD) is between +90° and 180°, left axis deviation (LAD) is between 0° and −90°, and either "extreme" right axis or "extreme" LAD is between 180° and +270°, or −90° and 180°, respectively.

With these fundamental concepts in mind, determination of axis can be easy and quick. There are three steps in determining axis (Table 2.1).

Step One: Examine leads I and aVF and see if the QRS deflections are net positive, net negative, or isoelectric. With this information, one can immediately determine the axis or, more usually, in which quadrant the axis is located—normal axis, RAD, LAD, or extreme axis deviation (Table 2.2). This is just a quick application of the positive vs. negative net deflection concept in the horizontal and vertical limb leads (I and aVF, respectively). If either I or aVF are isoelectric, then the axis is perpendicular to that lead and in the direction dictated by the other lead. Specifically, if I is isoelectric and aVF is positive, the axis is +90°. If aVF is isoelectric and I is positive, the axis is 0°. If I is isoelectric and aVF is negative, the axis is −90°. If aVF is isoelectric and I is negative, the axis is 180°.

Step Two: If I and aVF show that the axis is in a quadrant, look further for an isoelectric lead. Again, this is the bipolar (limb) lead with equal area deflected above and below the baseline. If there is an isoelectric lead, the axis is perpendicular to that lead in the quadrant determined in the first step. An isoelectric lead is not sought before quadrant determination because the axis could be in either direction perpendicular to the isoelectric lead, and observing the other leads becomes necessary to reveal which direction is correct.

B.G. Petty, *Basic Electrocardiography*, DOI 10.1007/978-1-4939-2413-4_2,

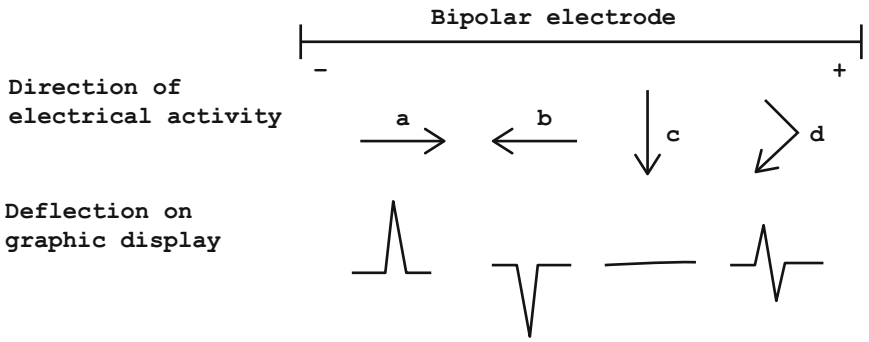


Fig. 2.1 Deflection of electrical activity with bipolar electrodes. (**a**) Electrical activity in direction parallel to orientation of electrode and towards positive pole, creating upward deflection. (**b**) Electrical activity in direction parallel to orientation of electrode and towards negative pole, creating downward deflection. (**c**) Electrical activity in direction perpendicular to orientation of electrode, creating no deflection. (**d**) Electrical activity first towards positive, then towards negative pole, with average direction perpendicular to electrode, creating equally positive and negative (isoelectric) deflection

Fig. 2.2 Intersection of bipolar electrodes

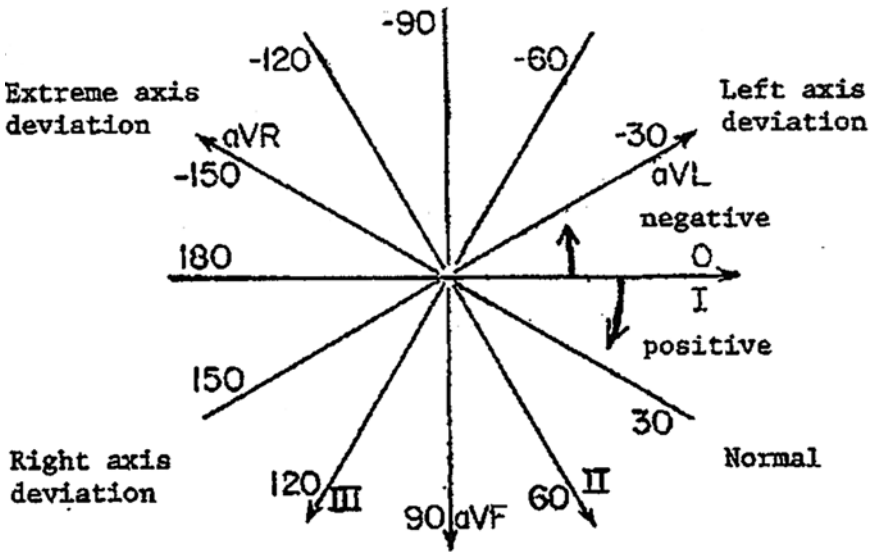


Table 2.1 Steps in determining electrical axis

1. Determine quadrant
2. Identify isoelectric lead, if present
3. If no isoelectric lead, interpolate

Table 2.2 Determining the quadrant of the axis

Lead I	Lead aVF	Quadrant
+	+	Normal
+	–	Left axis deviation
–	+	Right axis deviation
–	–	Extreme axis deviation

The leads to examine for an isoelectric lead depend on which quadrant the axis is in; they are the leads whose perpendiculars trisect the quadrant the axis is in, based on Step One. If the axis is in either the normal or extreme axis quadrants, the trisecting perpendiculars are those of leads III and aVL. With either right or LAD, the trisecting perpendiculars are those of leads II and aVR. If one of these trisecting leads is isoelectric, the axis is perpendicular to that lead in the correct quadrant.

Step Three: If there is not an isoelectric lead, then one interpolates. Interpolation is within a 30° sector between two perpendiculars to leads. The correct 30° sector is determined by the net deflection of the waves in the limb leads (Table 2.3). This means that the leads closest to isoelectric are determined, and the relative positivity or negativity of the leads is examined to assess how close to isoelectric those leads are. The closer the lead is to isoelectric, the closer the axis is to the perpendicular of that lead. There should be no more than 5–15° interobserver or intraobserver variability in reading axis, and axis is conventionally reported by humans to the nearest 5° (computers are programmed to report axis to the nearest 1°).

Table 2.3 Sectors of interpolation according to limb lead deflection

Lead							
I	II	III	aVR	aVL	aVF	Quadrant[a]	30° Sector
+	+	−	−	+	+	Nl	0° to +30°
+	+	+	−	+	+	Nl	+30° to +60°
+	+	+	−	−	+	Nl	+60° to +90°
+	+	−	−	+	−	LAD	0° to −30°
+	−	−	−	+	−	LAD	−30° to −60°
+	−	−	+	+	−	LAD	−60° to −90°
−	+	+	−	−	+	RAD	+90° to +120°
−	+	+	+	−	+	RAD	+120° to +150°
−	−	+	+	−	+	RAD	+150° to 180°
−	−	−	+	+	−	EAD	−90° to 120°
−	−	−	+	−	−	EAD	−120° to −150°
−	−	+	+	−	−	EAD	180° to +210°

[a]*Nl* normal quadrant, *LAD* left axis deviation, *RAD* right axis deviation, *EAD* extreme axis deviation

Some examples should be helpful in illustrating these concepts. Consider Fig. 2.3 in stepwise fashion regarding the determination of the QRS axis. First, look at leads I and aVF. Both have net positive QRS complexes, so you know immediately that the axis is in the normal quadrant, somewhere between 0 and +90°. Next, look for an isoelectric lead, and because the axis is in the normal quadrant we examine leads III and aVL. The QRS complexes in lead aVL have equal areas under the upward and above the downward deflections, i.e., lead aVL is isoelectric. Therefore, the QRS axis is perpendicular to the orientation of lead aVL and is in the normal quadrant, or +60°.

Now turn to the tracing in Fig. 2.4. First, look at leads I and aVF. Lead I is net positive, but lead aVF is net negative, so you know that the axis is in the LAD quadrant, somewhere between 0° and −90°. Next, look for an isoelectric lead. Because the axis is in the LAD quadrant, look at leads II and aVR. The QRS complexes in lead II are isoelectric, so the QRS axis is perpendicular to lead II in the LAD quadrant, or −30°.

Now try Fig. 2.5. Lead I is net negative and aVF is net positive, so the axis is in the RAD quadrant, somewhere between +90° and 180°. Because the axis is in the RAD quadrant, look at leads II and aVR for the isoelectric lead. The QRS complexes are isoelectric in aVR, so the axis is perpendicular to that lead in the RAD quadrant, or +120°.

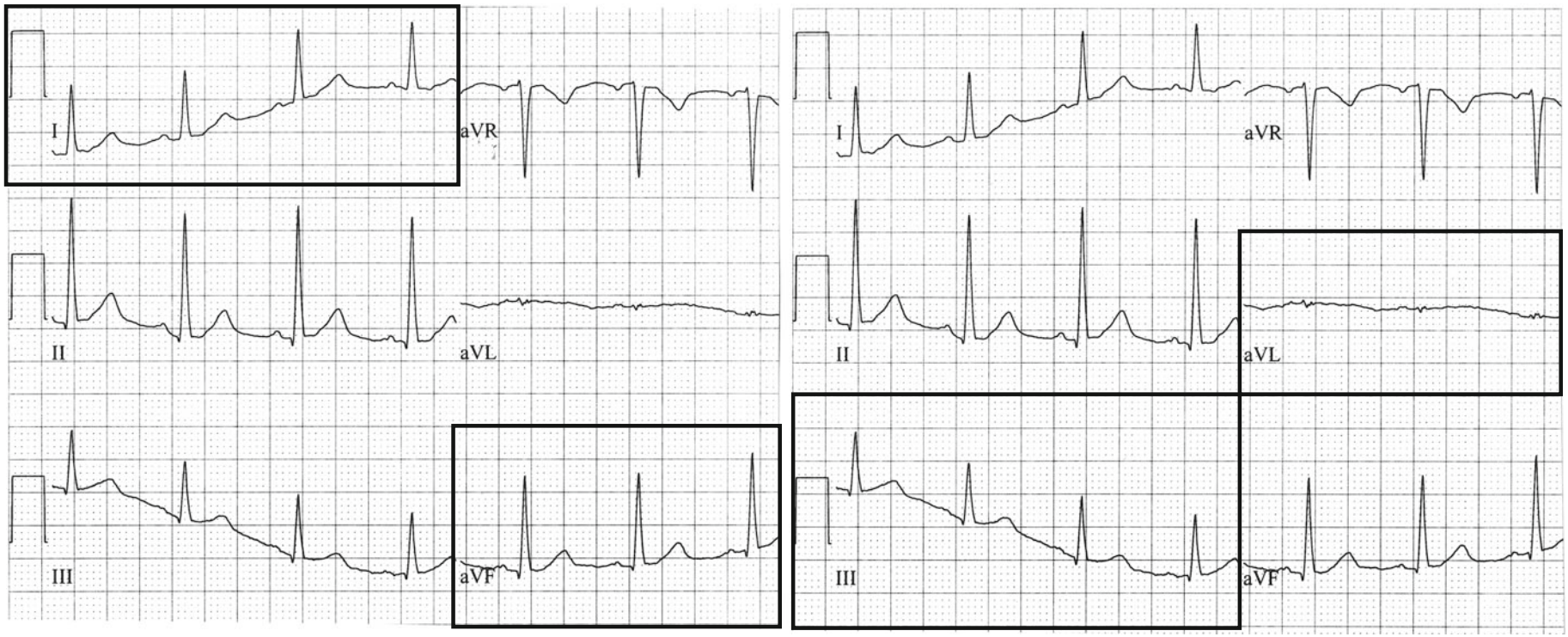
Now consider Fig. 2.6. First, look at leads I and aVF. Both leads I and aVF are positive. Therefore, the axis is in the normal quadrant. For that reason, we look at leads III and aVL for an isoelectric lead. It appears that III is isoelectric, so the axis is perpendicular to lead III in the normal quadrant, or +30°.

Next, consider the record in Fig. 2.7. In examining leads I and aVF, one finds that lead aVF is isoelectric, so the axis does not fall within a quadrant, but rather is on the horizontal. Because lead I is positive, the axis must be 0° (rather than 180°, which would require lead I to be negative). In Fig. 2.8, I is isoelectric and aVF is positive, so the axis is +90°.

All of the previous tracings have had an isoelectric lead, which makes determining the axis quite simple. Now let us turn to Fig. 2.9. Both leads I and aVF are net positive, so we know that the axis is in the normal quadrant. Next, we look for an isoelectric lead, and because the axis is in the normal quadrant we look at leads III and aVL. Neither of those is isoelectric, however, so we must proceed to Step Three, which is to interpolate. Considering leads I, III, aVL, and aVF, the leads closest to isoelectric are leads III and aVL. If III were isoelectric, the axis would be +30°, but since III is net positive, the axis must be on the positive side of III, or to the right of (more positive than) +30°. If aVL were isoelectric, the axis would be +60, but because aVL is net positive, the axis must be on the positive side of aVL, or to the left of (less positive than) +60°. Thus, the axis is between +30° and +60°. Carefully comparing the relative positive and negative deflections of leads III and aVL reveals that the QRS complexes in the two leads are very similar. Therefore, the axis is midway between the lines perpendicular to these two leads, or +45°.

Another example of interpolation is given in Fig. 2.10. Lead I is net positive but aVF is negative, so the axis is in the LAD quadrant. Examining leads II and aVR shows that neither is isoelectric, but lead II is closest to isoelectric. If lead II were isoelectric, the axis would be −30°, but since lead II is positive, the axis must be on the positive side of II, or to the right of (less negative than) −30°. Because lead aVF is negative, the axis must be to the left of (more negative than) 0°. Therefore, the axis is somewhere between 0° and −30°. Because lead II is closer to isoelectric than lead aVF, the axis is closer to −30° than 0°, or about −20°. Keep in mind that the closer a lead is to isoelectric, the closer the axis is to the perpendicular of that lead.

Rarely it appears that several or all of the bipolar leads have isoelectric QRS complexes (Fig. 2.11). In this case, the axis is "indeterminate" because no axis value is consistent



Step 1

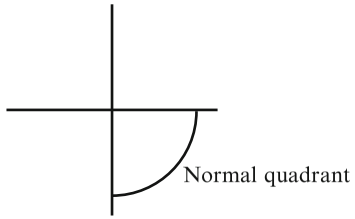
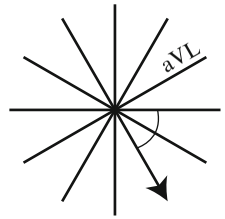
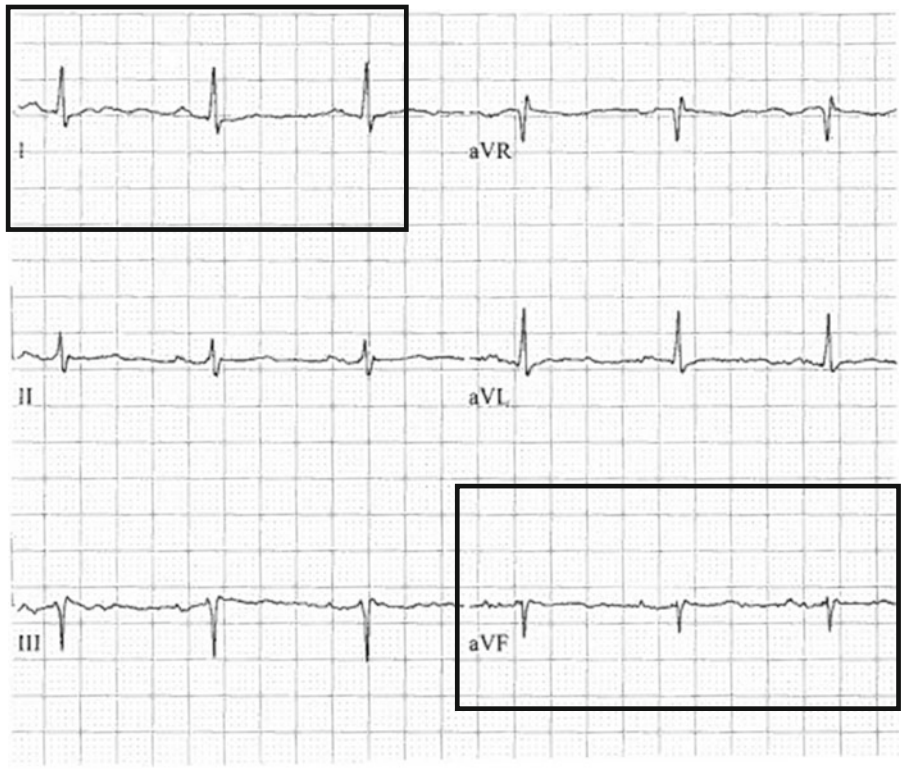


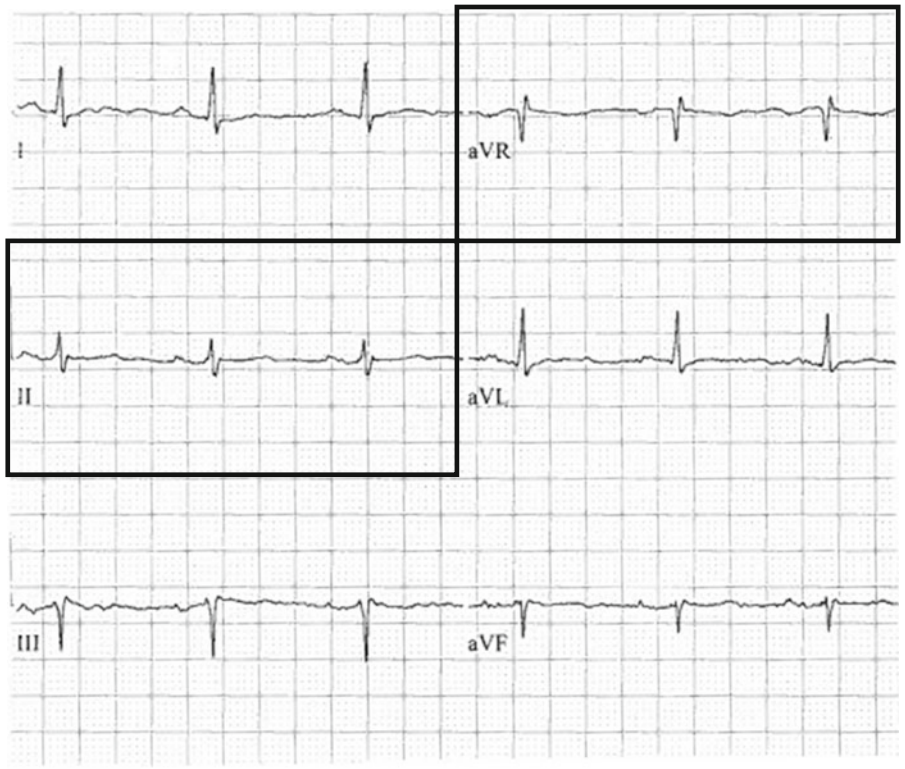
Fig. 2.3 **Step One:** Examine leads I and aVF. Result: Both I and aVF are positive. Interpretation: Axis in normal quadrant (0° to +90°).

Step 2

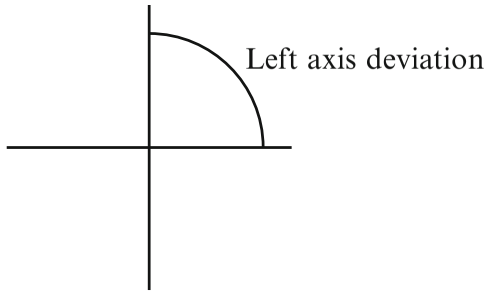


Step Two: Since the axis is in the normal quadrant, look for an isoelectric lead in leads III and aVL. Result: aVL is isoelectric. Interpretation: Axis is perpendicular to aVL in the normal quadrant, or +60°





Step 1



Step 2

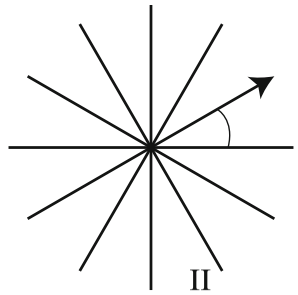
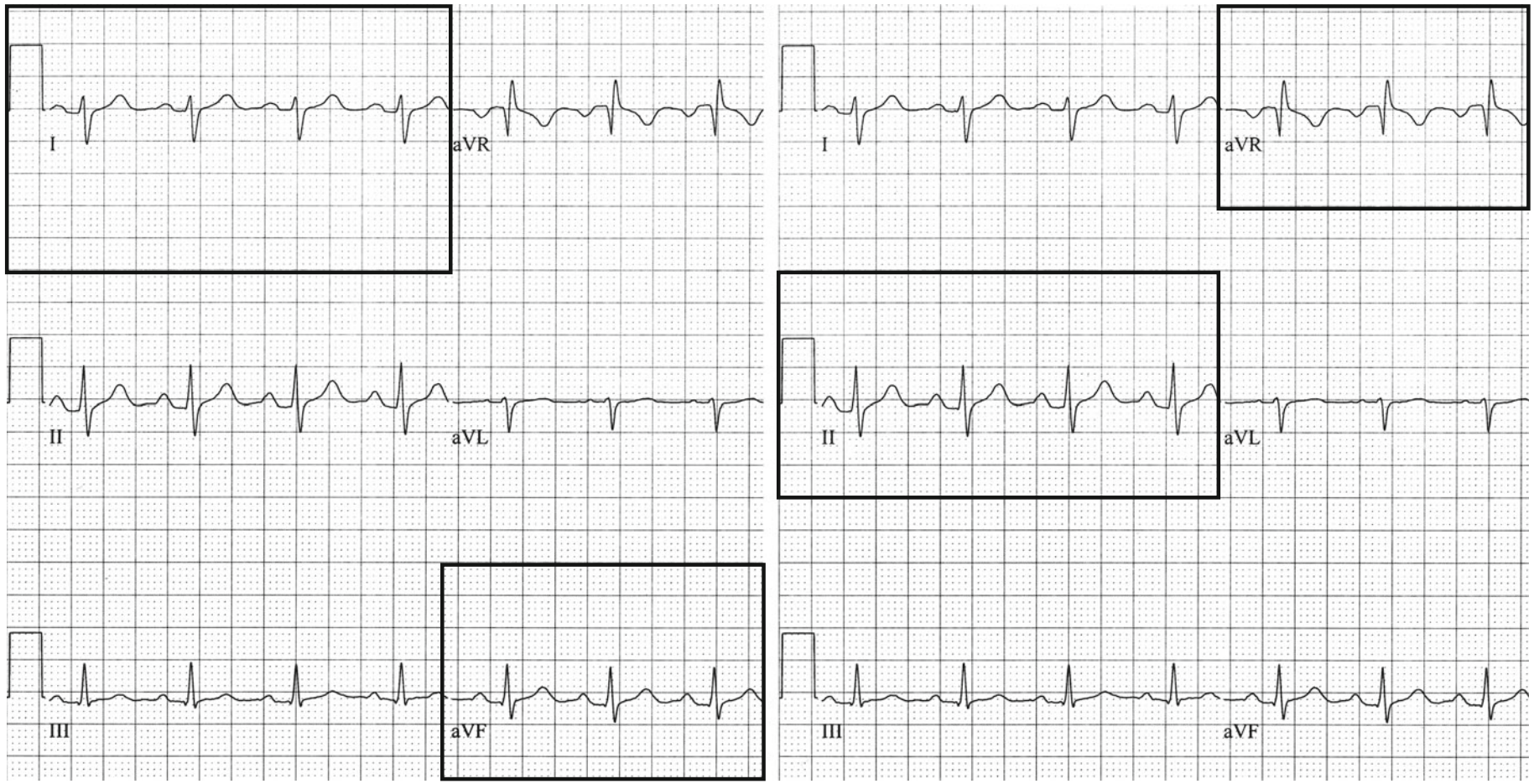
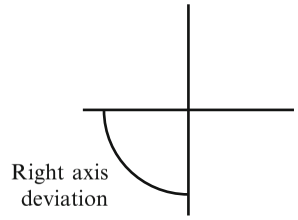


Fig. 2.4 Determining the QRS axis. **Step One:** Examine leads I and aVF. Result: Leads I is positive; aVF is negative. Interpretation: Axis in left axis deviation quadrant.

Step Two: Since the axis is in the left axis deviation quadrant, look for an isoelectric lead in leads II and aVR. Result: Lead II is isoelectric. Interpretation: The axis is perpendicular to II in the left axis deviation quadrant, or −30°



Step 1



Step 2

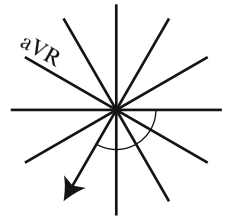
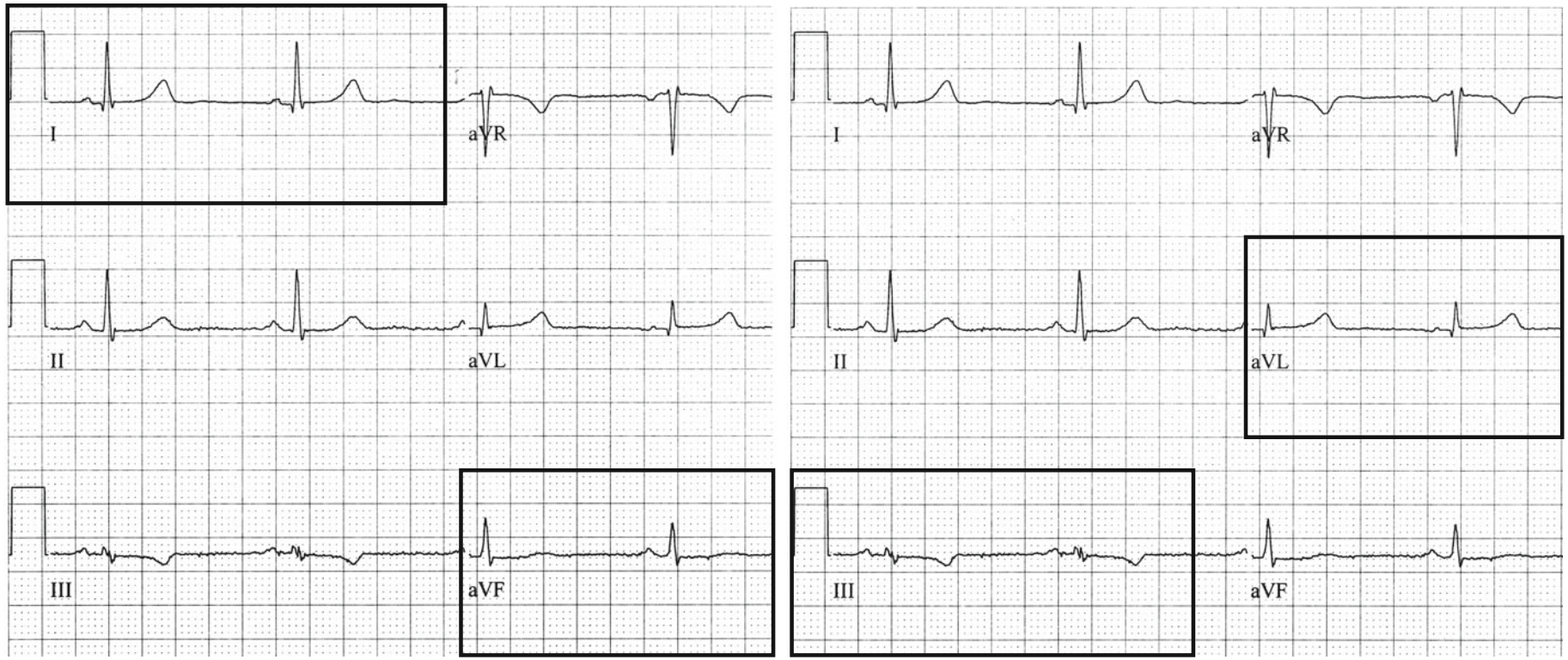
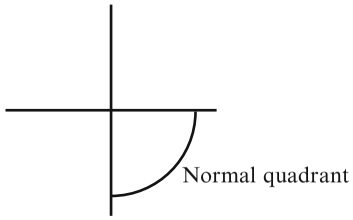


Fig. 2.5 Determining the QRS axis. **Step One:** Examine leads I and aVF. Result: Lead I is negative; aVF is positive. Interpretation: Axis in right axis deviation quadrant.

Step Two: Since the axis is in the right axis deviation quadrant, look for an isoelectric lead in leads II and aVR. Result: Lead aVR is isoelectric. Interpretation: The axis is perpendicular to aVR in the right axis deviation quadrant, or +120°



Step 1



Step 2

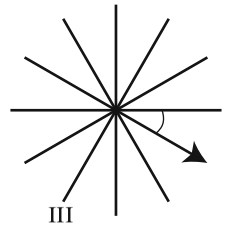


Fig. 2.6 Determining the QRS axis. **Step One:** Examine leads I and aVF. Result: Both I and aVF are positive. Interpretation: Axis in normal quadrant.

Step Two: Since the axis is in the normal quadrant, look for an isoelectric lead in leads III and III. Result: III is isoelectric. Interpretation: The axis is perpendicular to III in the normal quadrant, or +30°

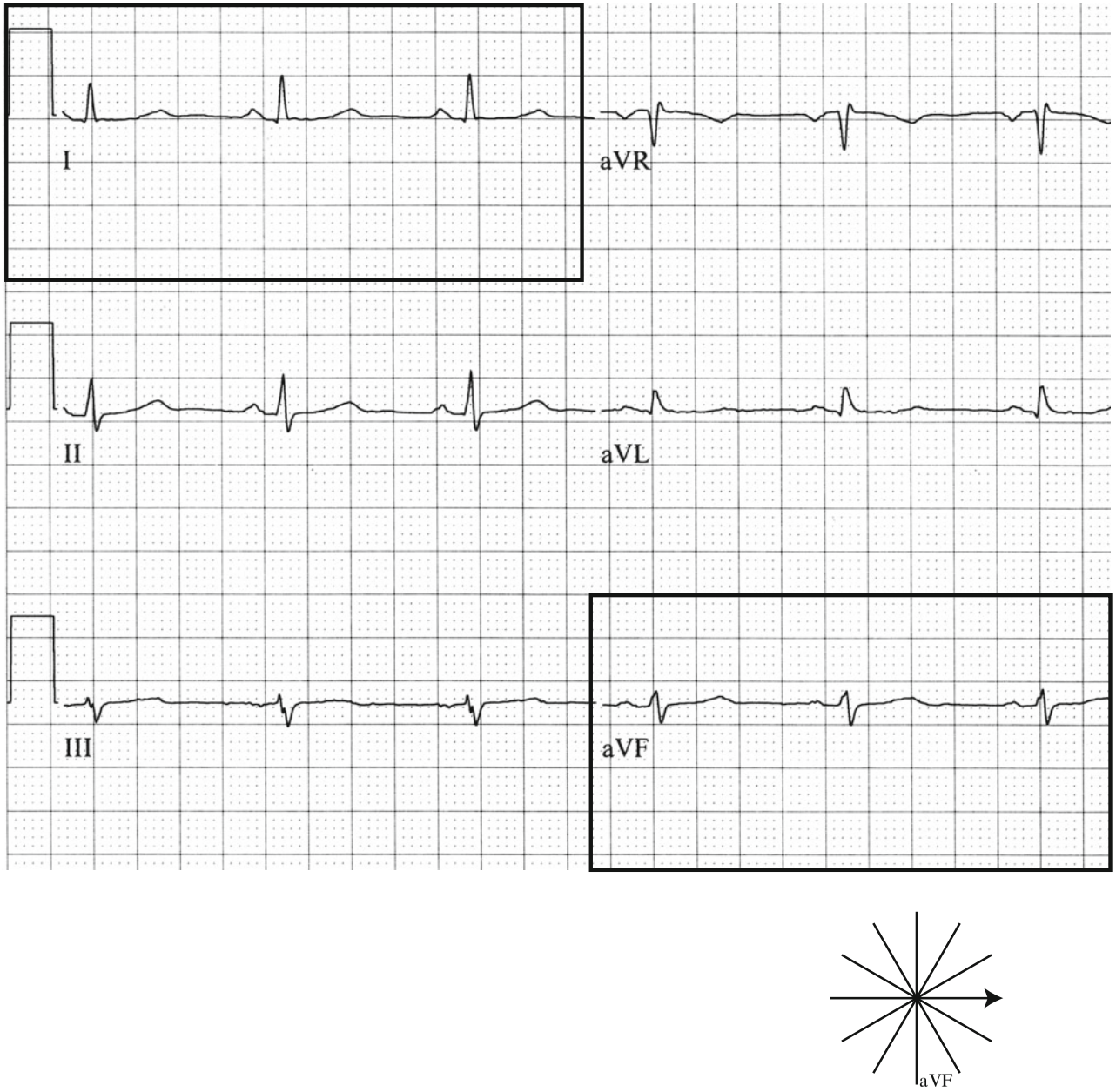


Fig. 2.7 Determining the QRS axis. **Step One:** Examine leads I and aVF. Result: Lead I is positive; lead aVF is isoelectric. Interpretation: The axis is perpendicular to aVF and to the left, or 0°

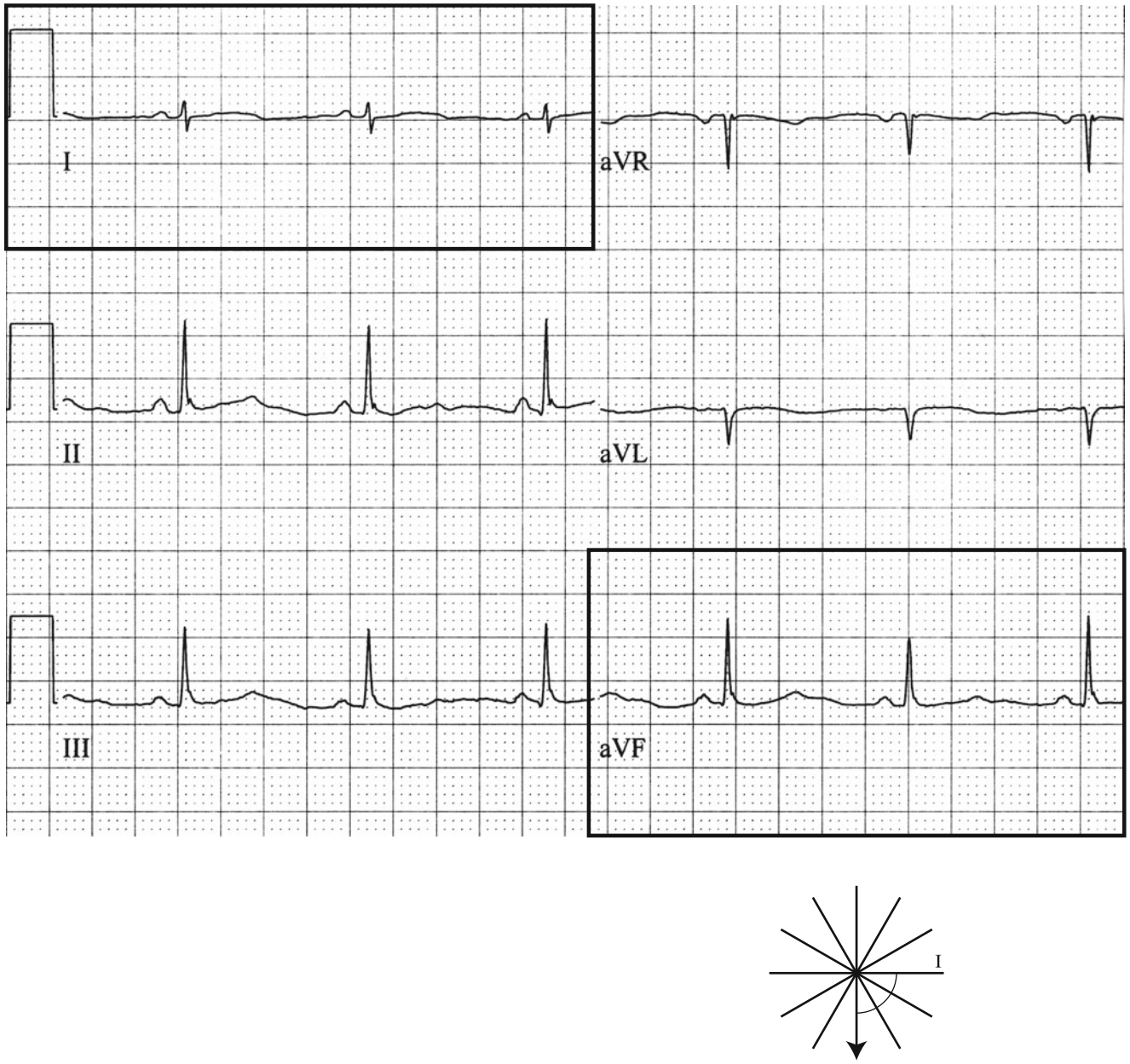
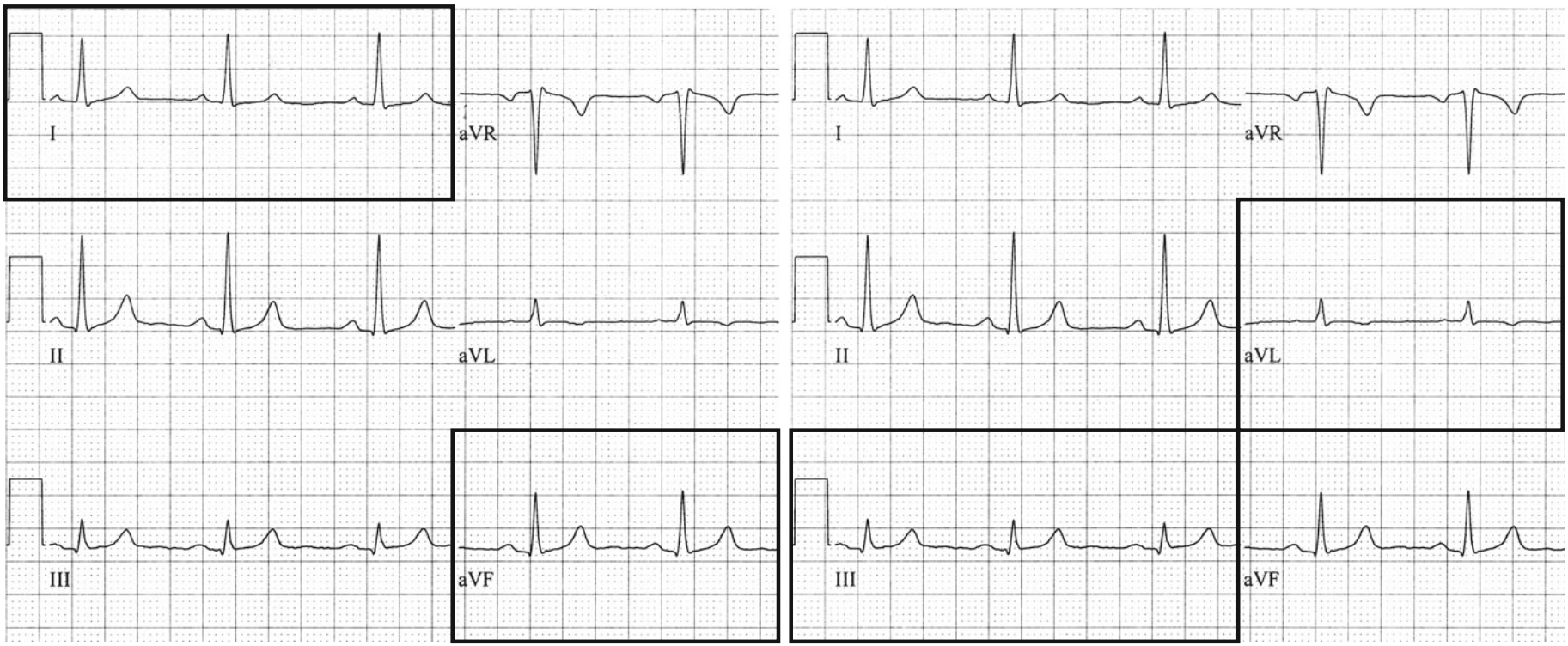
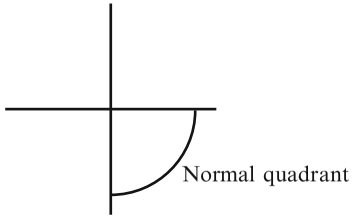


Fig. 2.8 Determining the QRS axis. **Step One:** Examine leads I and aVF. Result: Lead I is isoelectric; lead aVF is positive. Interpretation: The axis is perpendicular to I and positive, or +90°



Step 1



Step 2

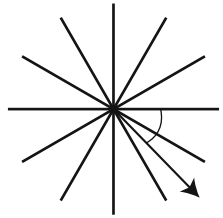
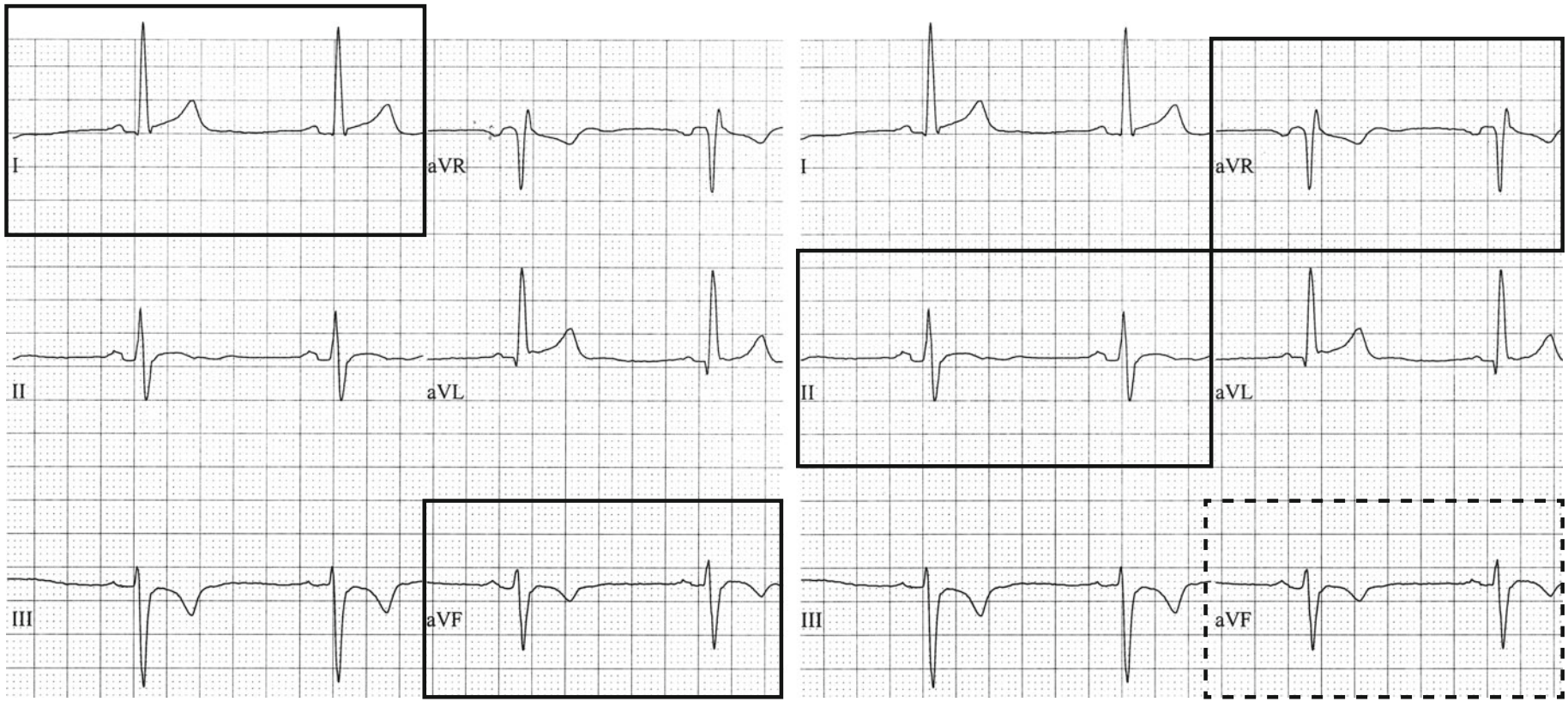


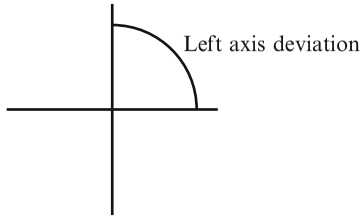
Fig. 2.9 Determining the QRS axis. **Step One:** Examine leads I and aVF. Result: Both I and aVF are positive. Interpretation: Axis in normal quadrant.

Step Two: Since the axis is in the normal quadrant, look for an isoelectric lead in III and aVL. Result: There is no isoelectric lead. Interpretation: Go to Step Three.

Step Three: Interpolate. Result: Leads III and aVL are closest to isoelectric and appear to be equally close to isoelectric. Interpretation: The axis is midway between the perpendiculars to leads III and aVL in the normal quadrant, or +45°



Step 1



Step 2

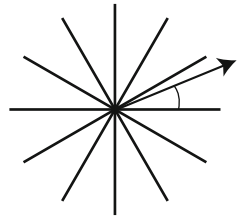


Fig. 2.10 Determining the QRS axis. **Step One:** Examine leads I and aVF. Result: Lead I is positive; lead aVF is negative. Interpretation: The axis is in the left axis deviation quadrant.

Step Two: Since the axis is in the left axis deviation quadrant, look for an isoelectric lead in leads II and aVR. Result: Neither II nor aVR is isoelectric. Interpretation: Go to Step Three.
Step Three: Interpolate. Result: The closest lead to isoelectric is lead II, but it is slightly positive, so the axis is less negative than −30°. Therefore, you must look at the lead whose perpendicular is "next door" to less negative than −30°, which is aVF (whose perpendicular is 0°). Lead II is closer to isoelectric than lead aVF. Interpretation: The axis is closer to the perpendicular of lead II than to the perpendicular of lead aVF, or −20°

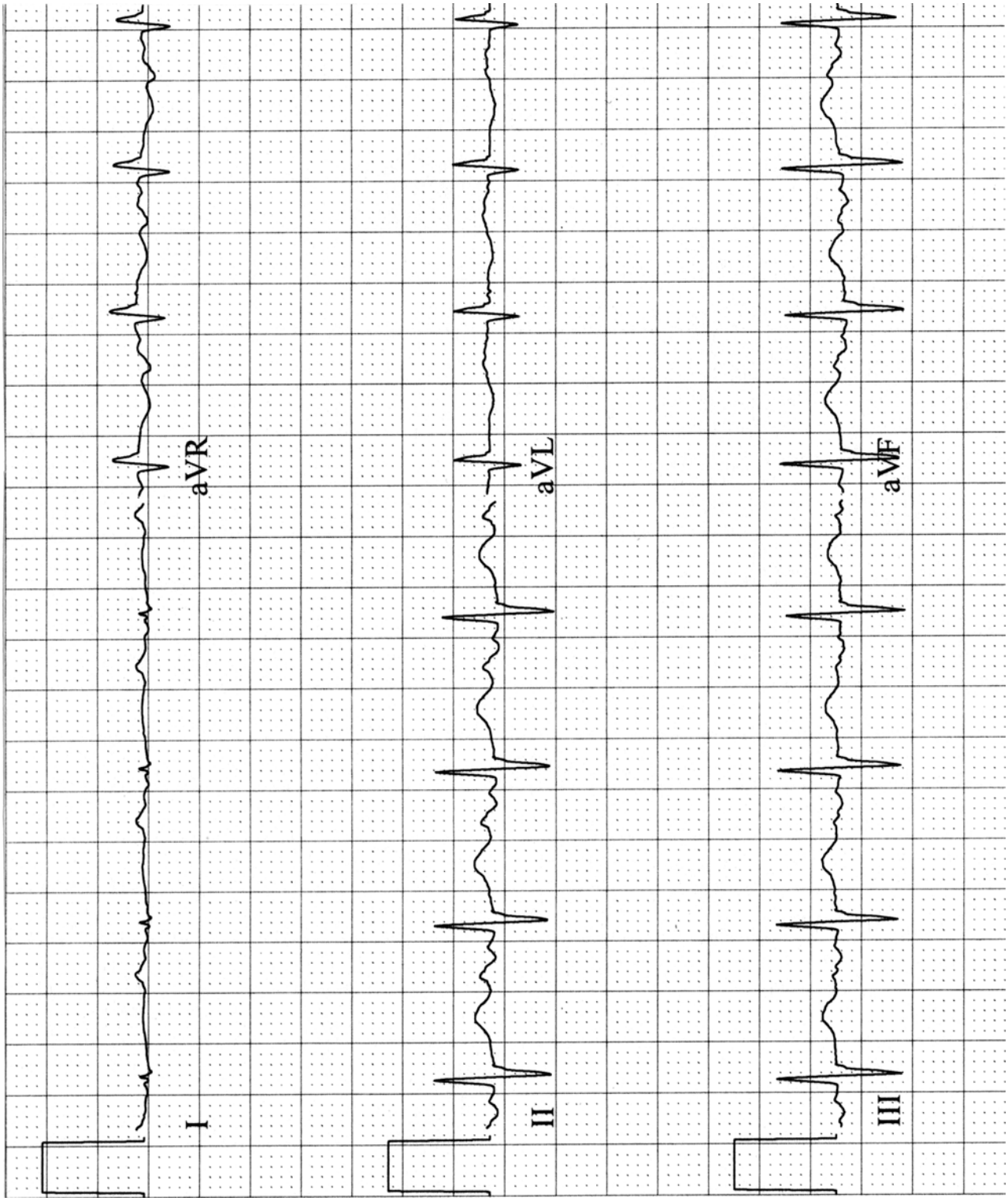


Fig. 2.11 Indeterminate axis. Note the QRS complex is very nearly isoelectric in almost all of the bipolar leads

Table 2.4 Conditions associated with axis deviation

Right axis deviation	Left axis deviation
Right ventricular hypertrophy	Left ventricular hypertrophy
As with COPD or	Inferior myocardial infarction
Congenital heart disease	Left anterior hemiblock
Right ventricular strain	Wolff–Parkinson–White
As with pulmonary embolism	Left bundle branch block
Or other acute lung disease	Normal variant
Left posterior hemiblock	
Wolff–Parkinson–White	
Normal variant	

with all of the observed QRS configurations. The pathological significance of an indeterminate axis is unclear, but it probably represents a normal variant.

The method presented above to determine axis is not the only method that can be used, but it is the method which I find easiest. With just a little practice and familiarity with the orientation of the leads, determining the axis by the method shown here can be done very rapidly.

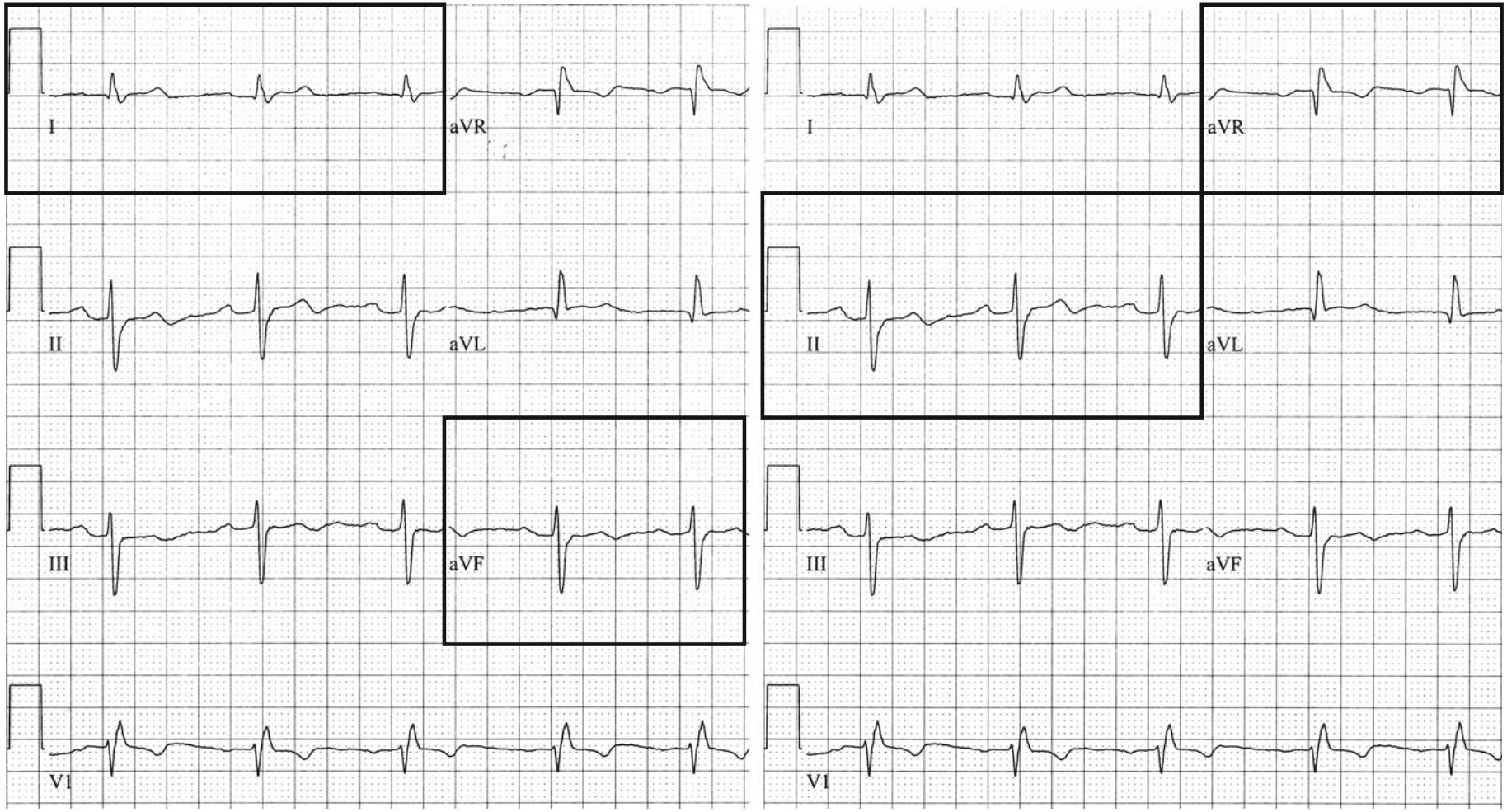
Electrical axis is important because deviations in the axis are associated with various conditions (Table 2.4). Furthermore, a change in a patient's axis may give a hint into the nature of the patient's problem. For example, consider a patient with chest pain and shortness of breath who has a previous EKG showing an axis of +10° and now has an EKG with an axis of +85° but no other changes in waveform and a normal chest X-ray. In both EKGs the axis is normal, but the significant rightward shift suggests right ventricular stress and should raise the suspicion of pulmonary embolism. Other acute pulmonary disease, such as pneumothorax, pneumonia, or hydrothorax, could conceivably shift the axis rightward as well.

As mentioned at the outset, the P wave axis and T wave axis can also be determined by applying the same principles explained above for the QRS axis. The P wave axis is usually in the normal quadrant, and the T wave axis should be no more than 60–70° different than the QRS axis. When the T wave axis deviates further from the QRS axis, an abnormality (e.g., ischemia, left ventricular strain, or metabolic disturbance) is present.

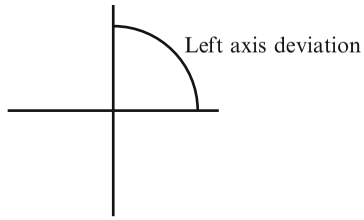
A special situation exists when determining the axis if the QRS duration is 0.12 s or longer. This is the situation with bundle branch blocks (see Chap. 5). Because of the distortion of conduction induced by the bundle branch block, it is proper to use only the first 0.08 s of the QRS complex to determine the electrical axis. The last portion of the QRS complex, the so-called terminal force, is a reflection of the conduction abnormality induced by the bundle branch block. Using the first 0.08 s is a method of at least partially correcting for the terminal forces, reducing the distortion induced by the bundle branch block. The terminal forces are especially misleading in the case of right bundle branch block and less so in left bundle branch block. An example of using the first 0.08 s for determining axis in the presence of a bundle branch block is given in Fig. 2.12.

Exercise Tracings

Determine the electrical axis for the QRS complexes in the following examples. In each case, only the limb leads are provided. Your answer should be within 10° of mine, but hopefully closer!



Step 1



Step 2

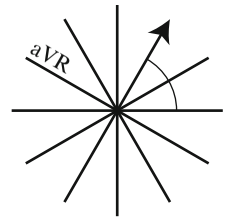
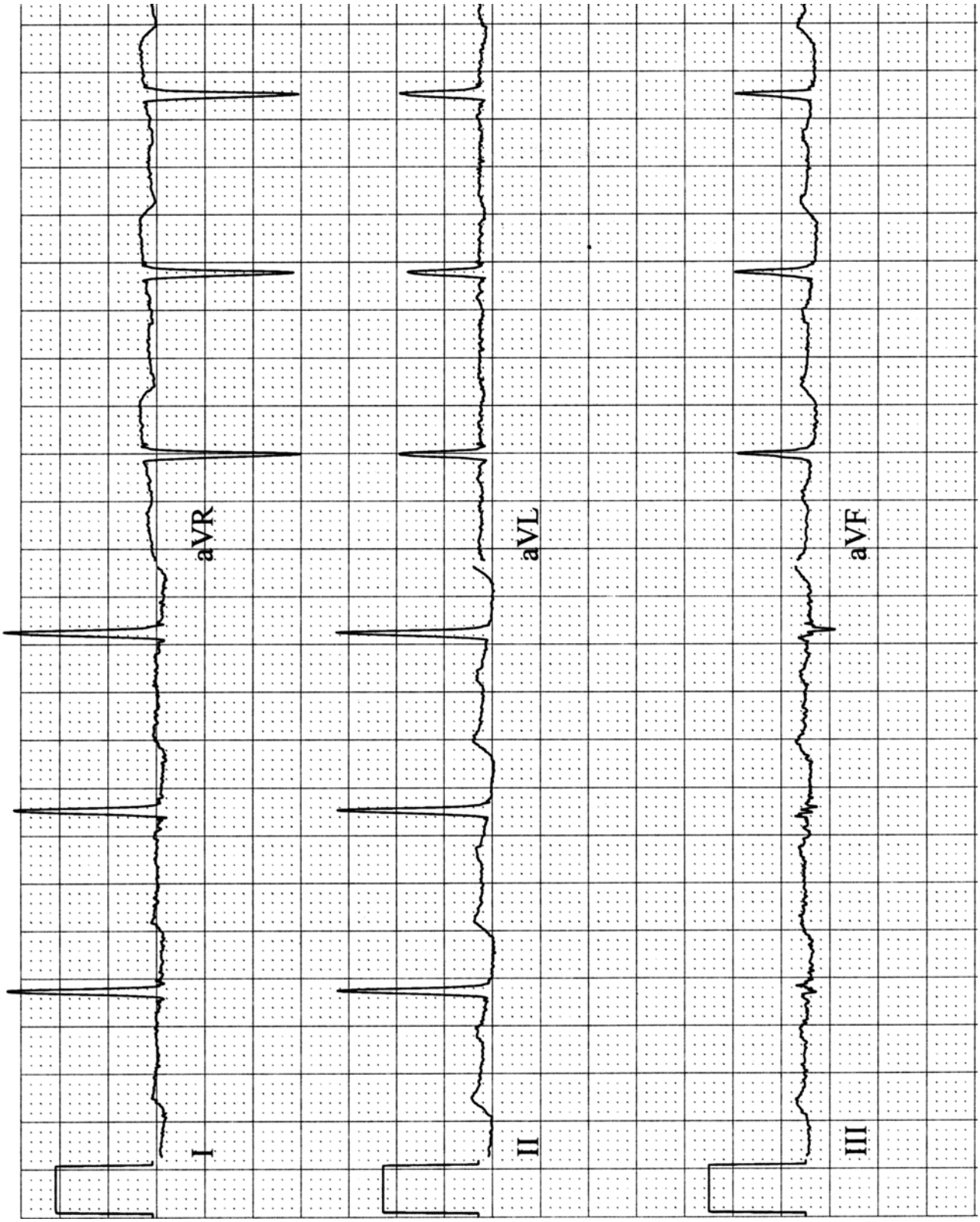
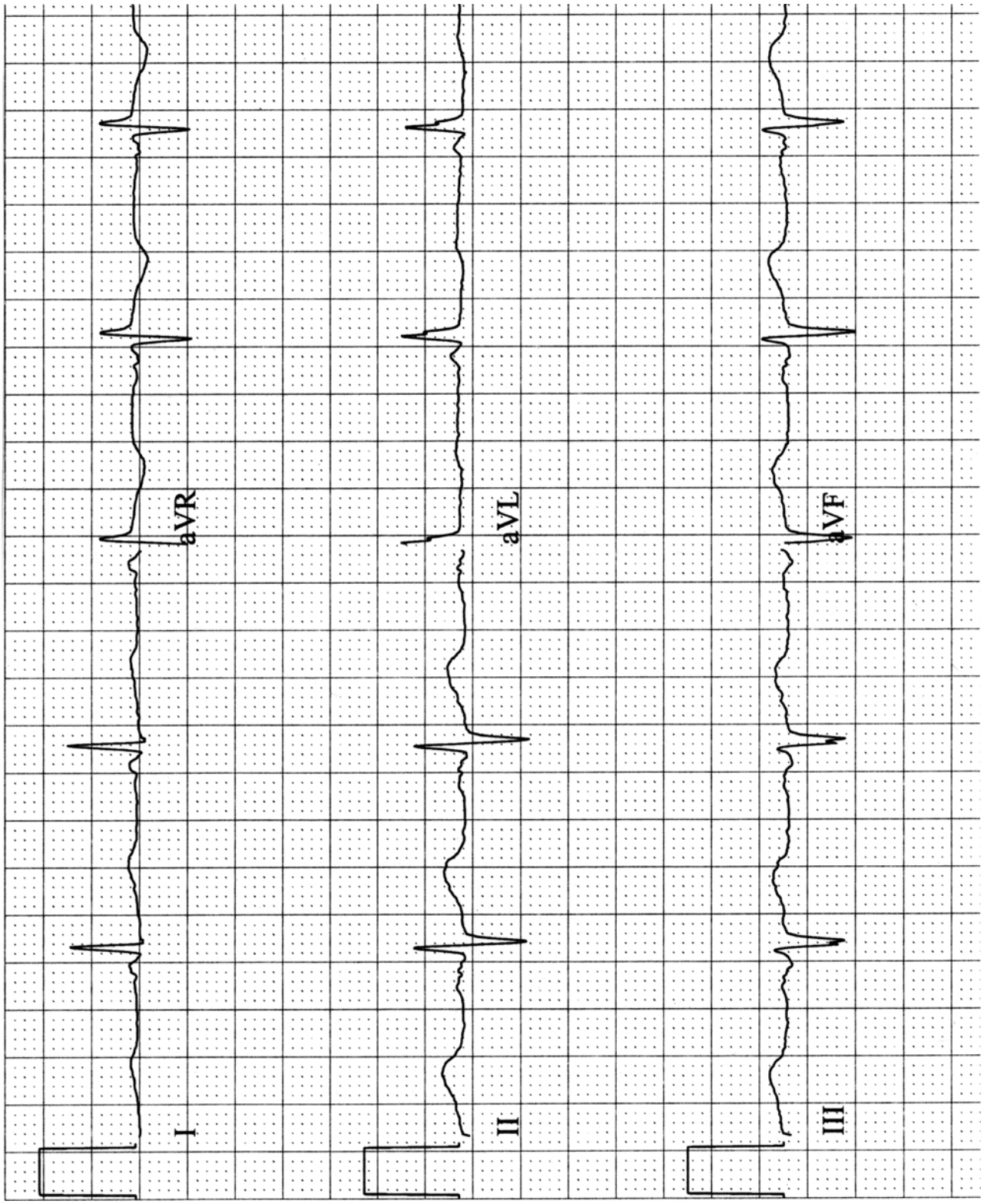


Fig. 2.12 Determining the QRS axis. **Step One:** Look at leads I and aVF. Result: Using the first 0.08 s of the QRS complex (since the total duration is 0.12 s), lead I is positive and lead aVF is negative. Interpretation: The axis is in the left axis deviation quadrant.

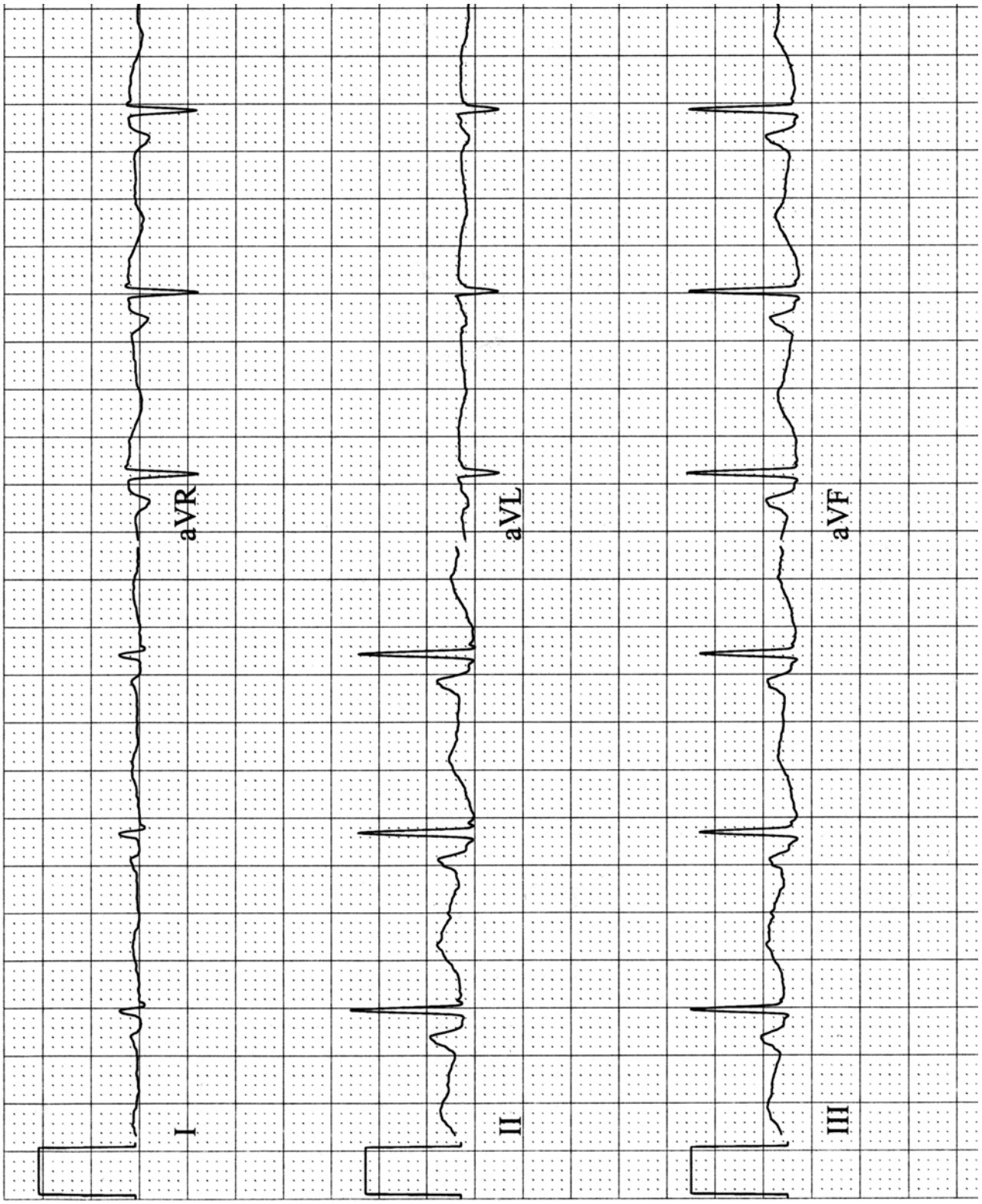
Step Two: Since the axis is in the left axis deviation quadrant, look at leads II and aVR (along with I and aVF) for an isoelectric lead. Result: Lead aVR is isoelectric. Interpretation: The axis is perpendicular to lead aVR in the left axis deviation quadrant, or −60°



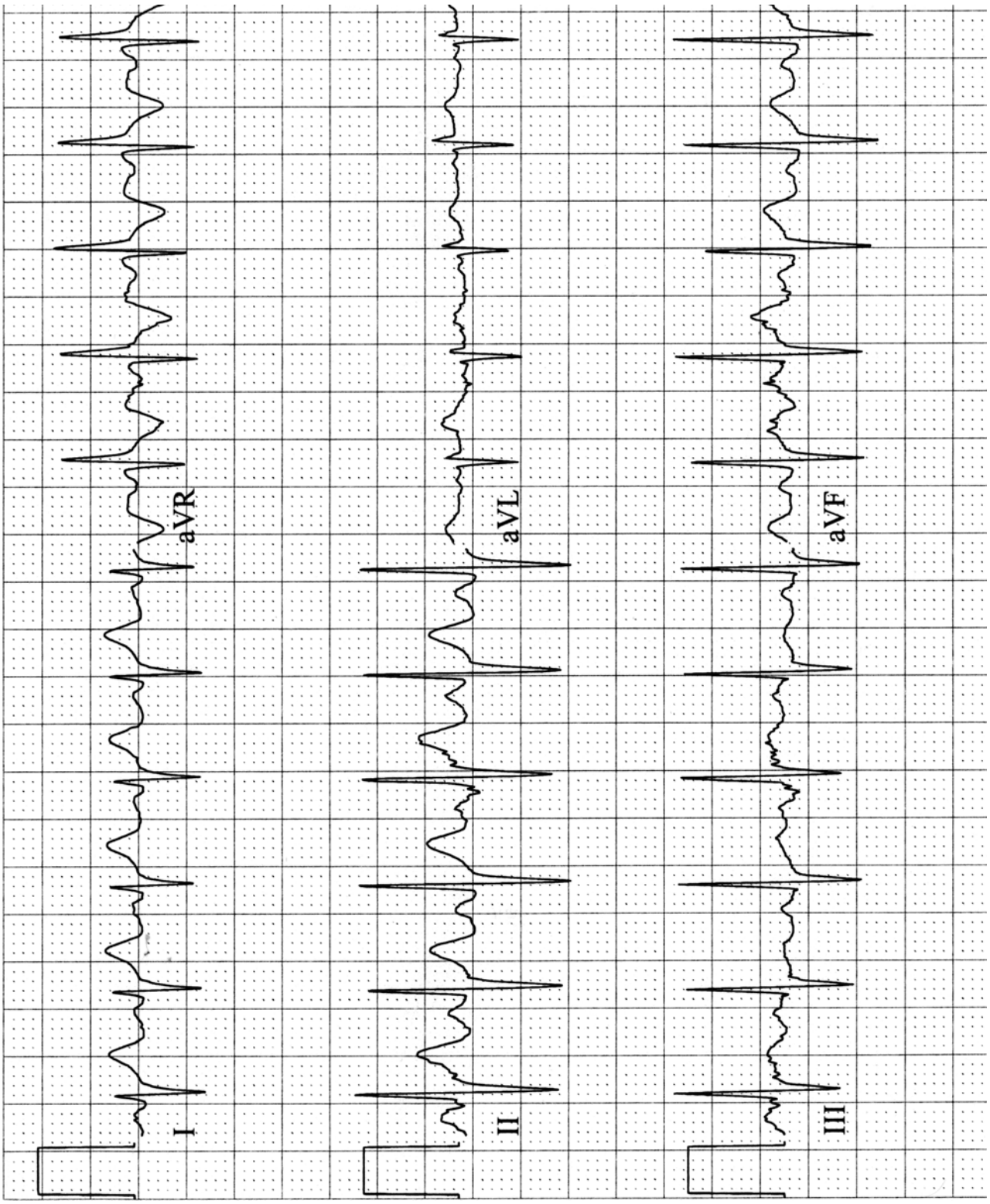
Exercise Tracing 2.1



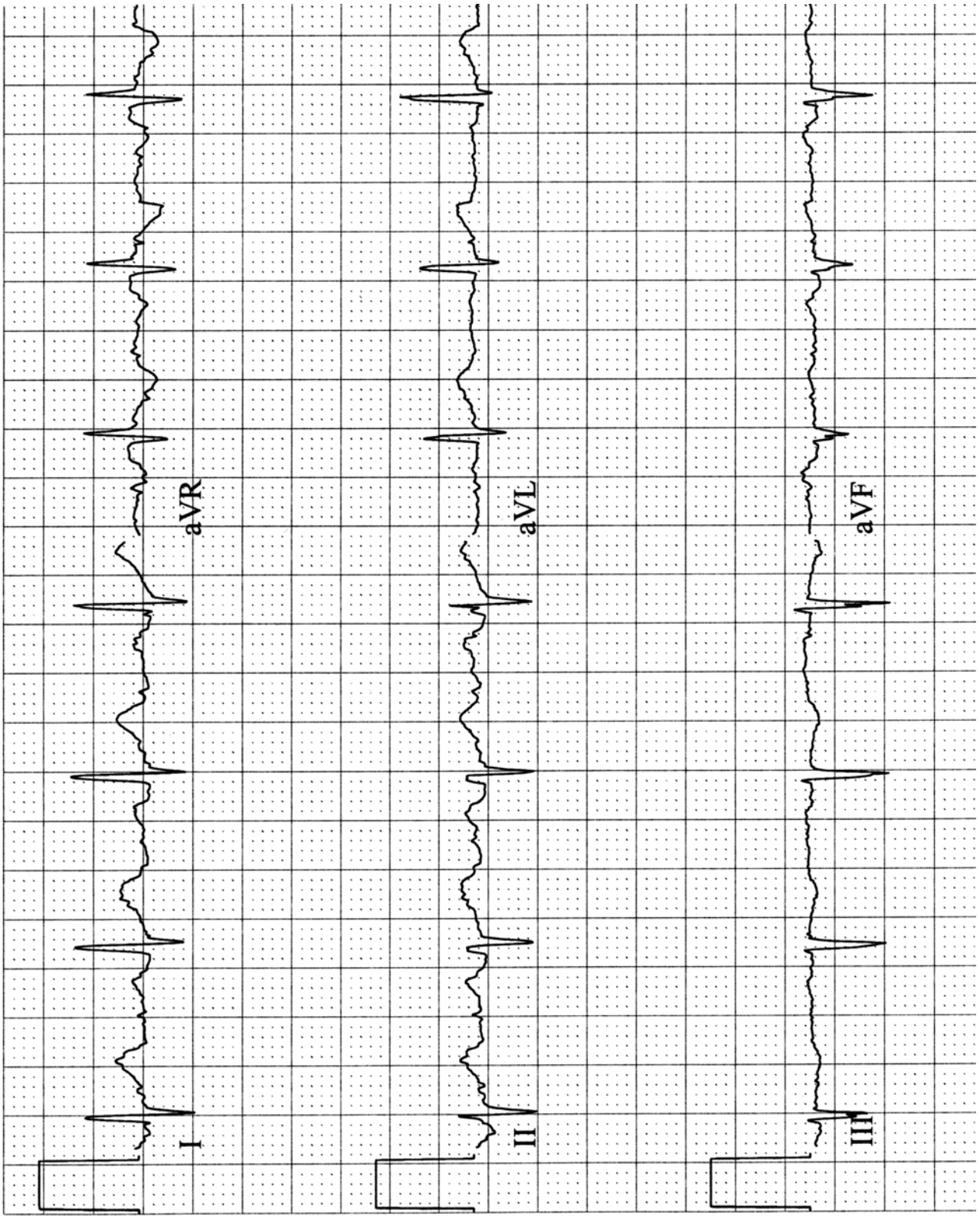
Exercise Tracing 2.2



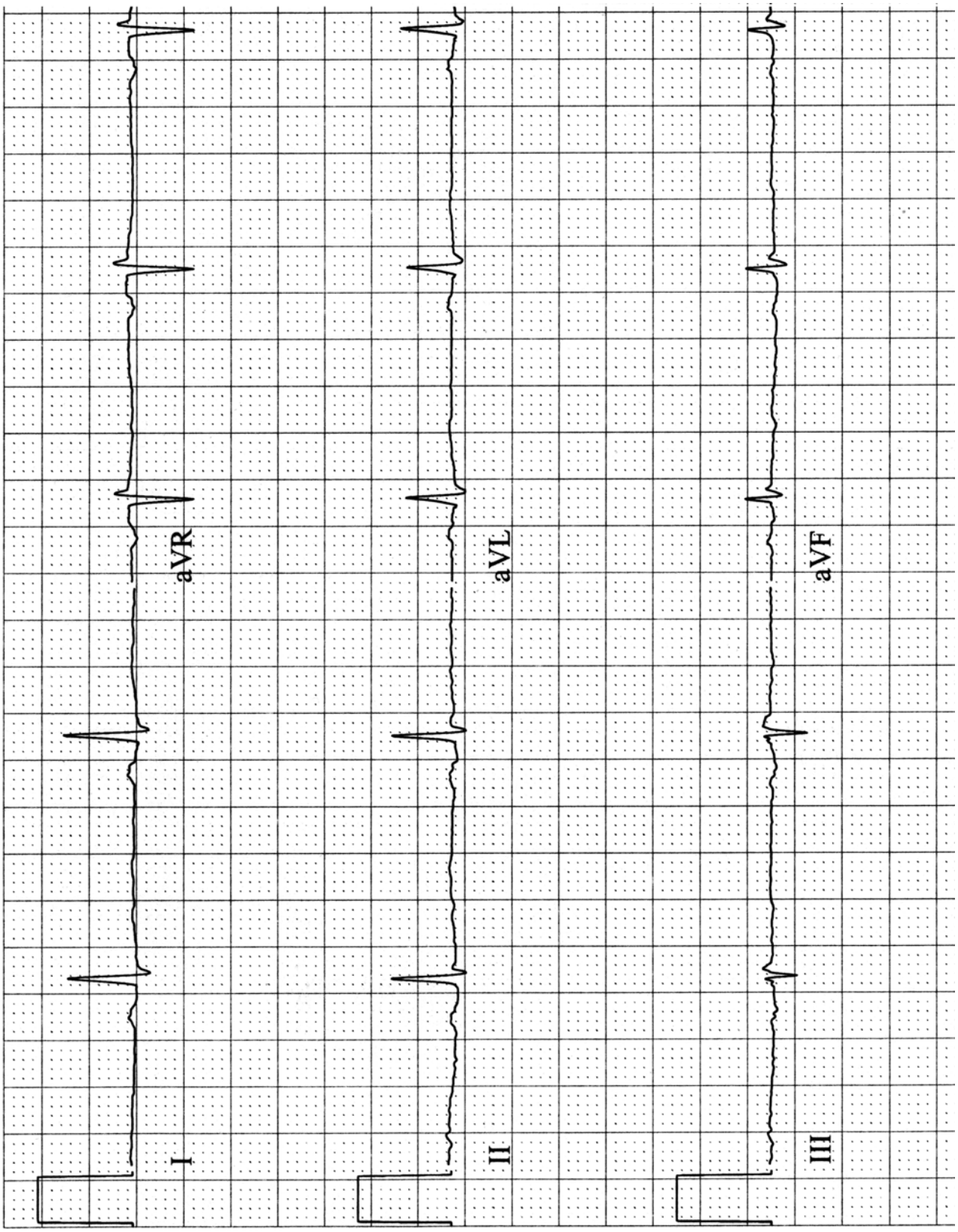
Exercise Tracing 2.3



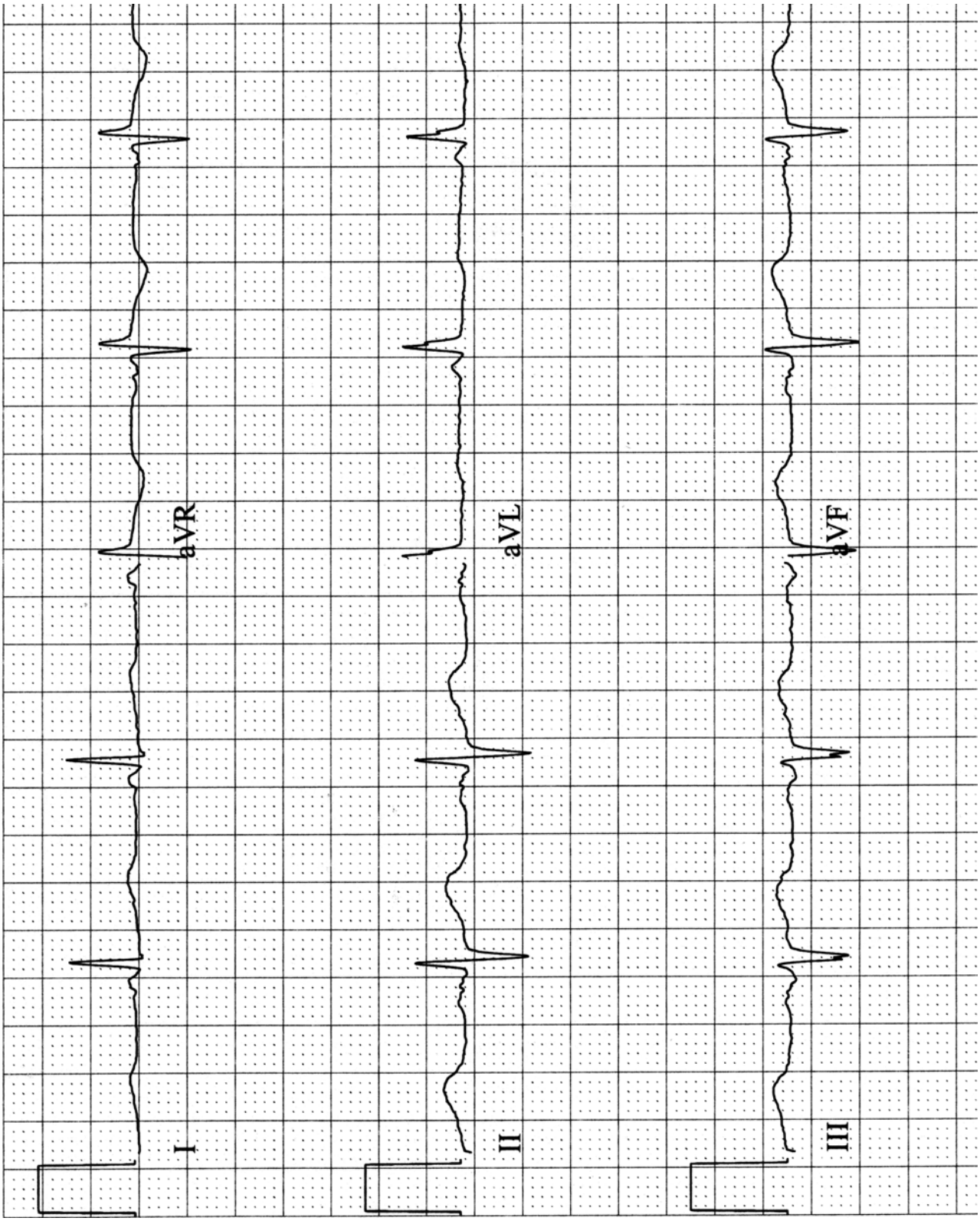
Exercise Tracing 2.4



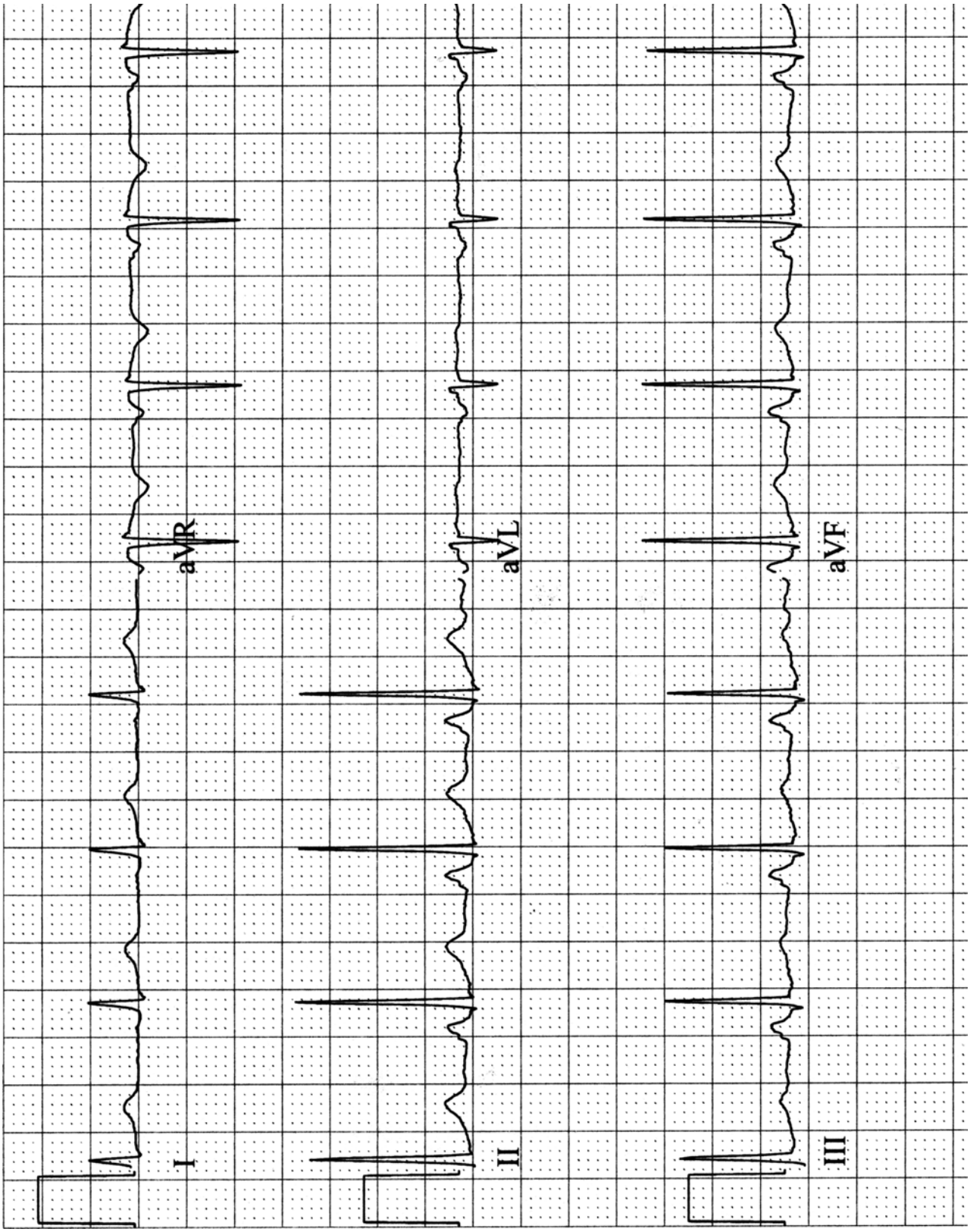
Exercise Tracing 2.5



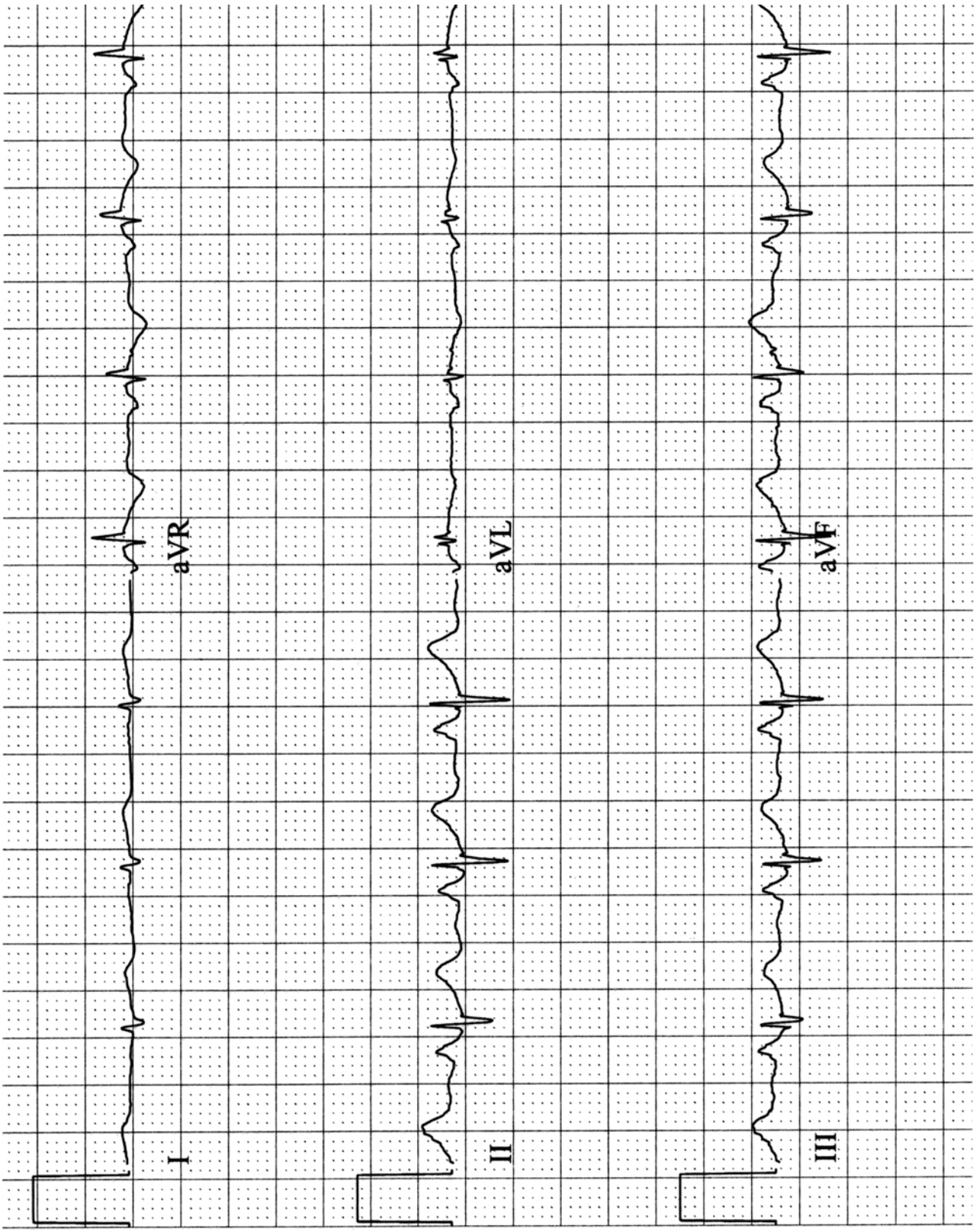
Exercise Tracing 2.6



Exercise Tracing 2.7

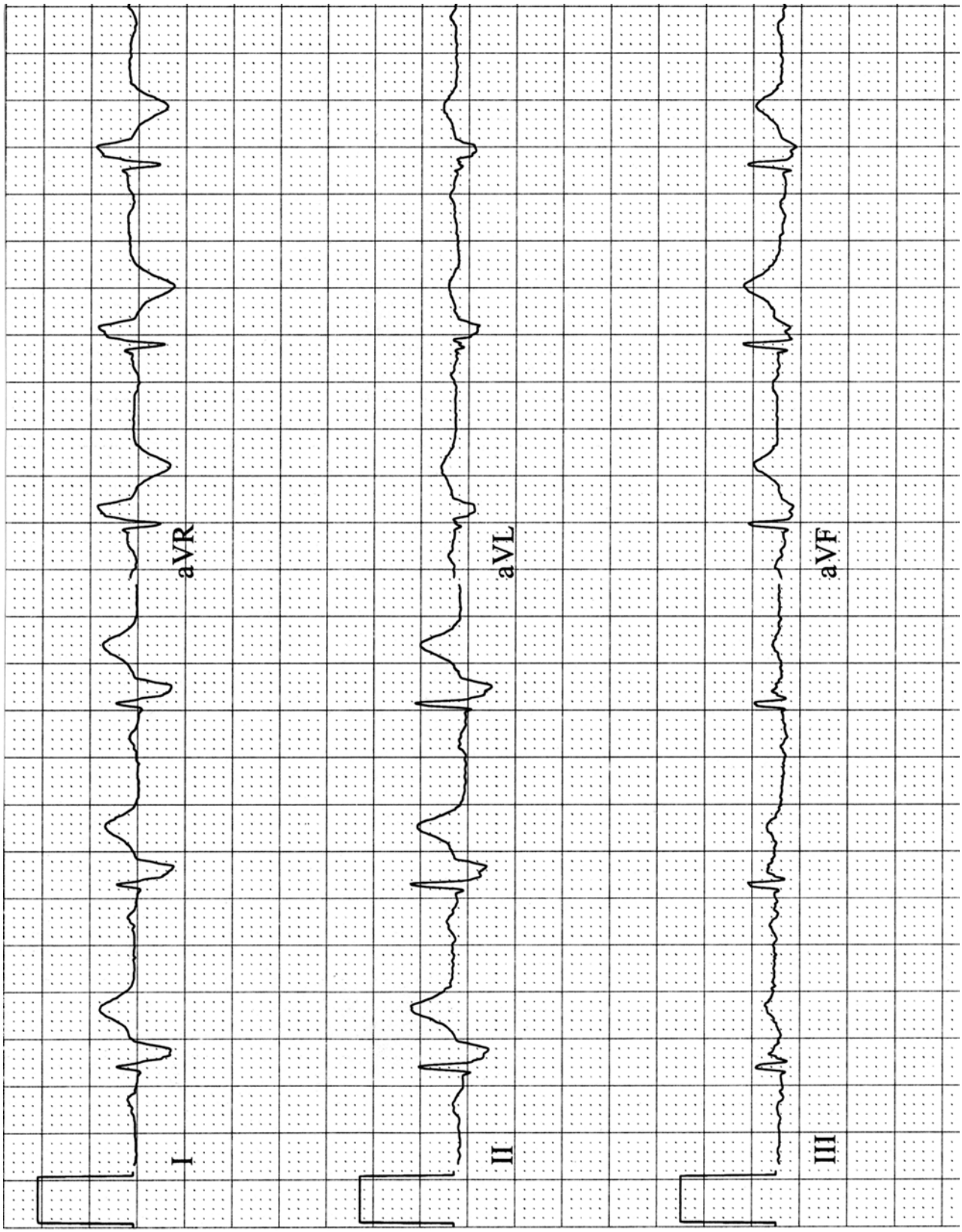


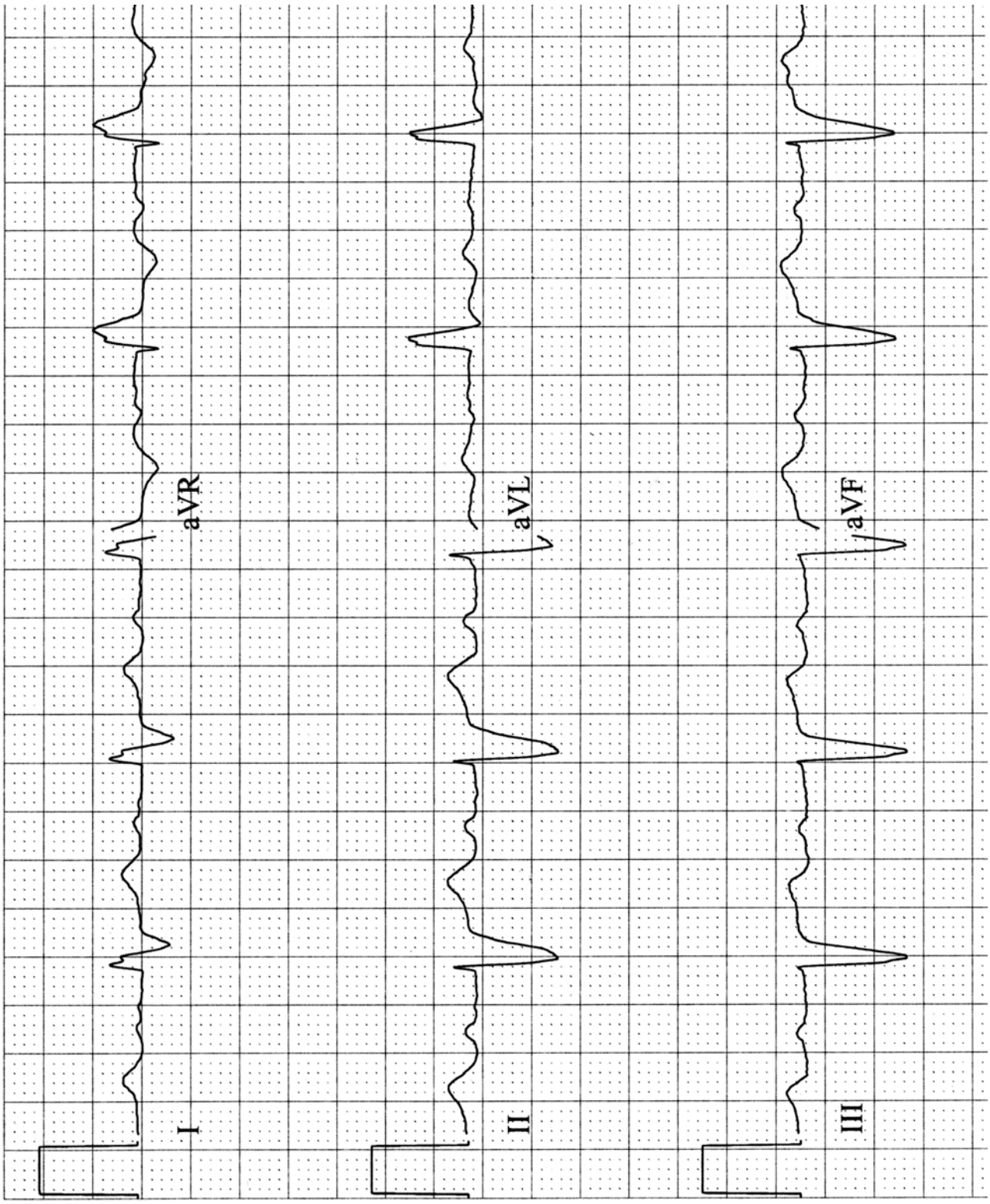
Exercise Tracing 2.8



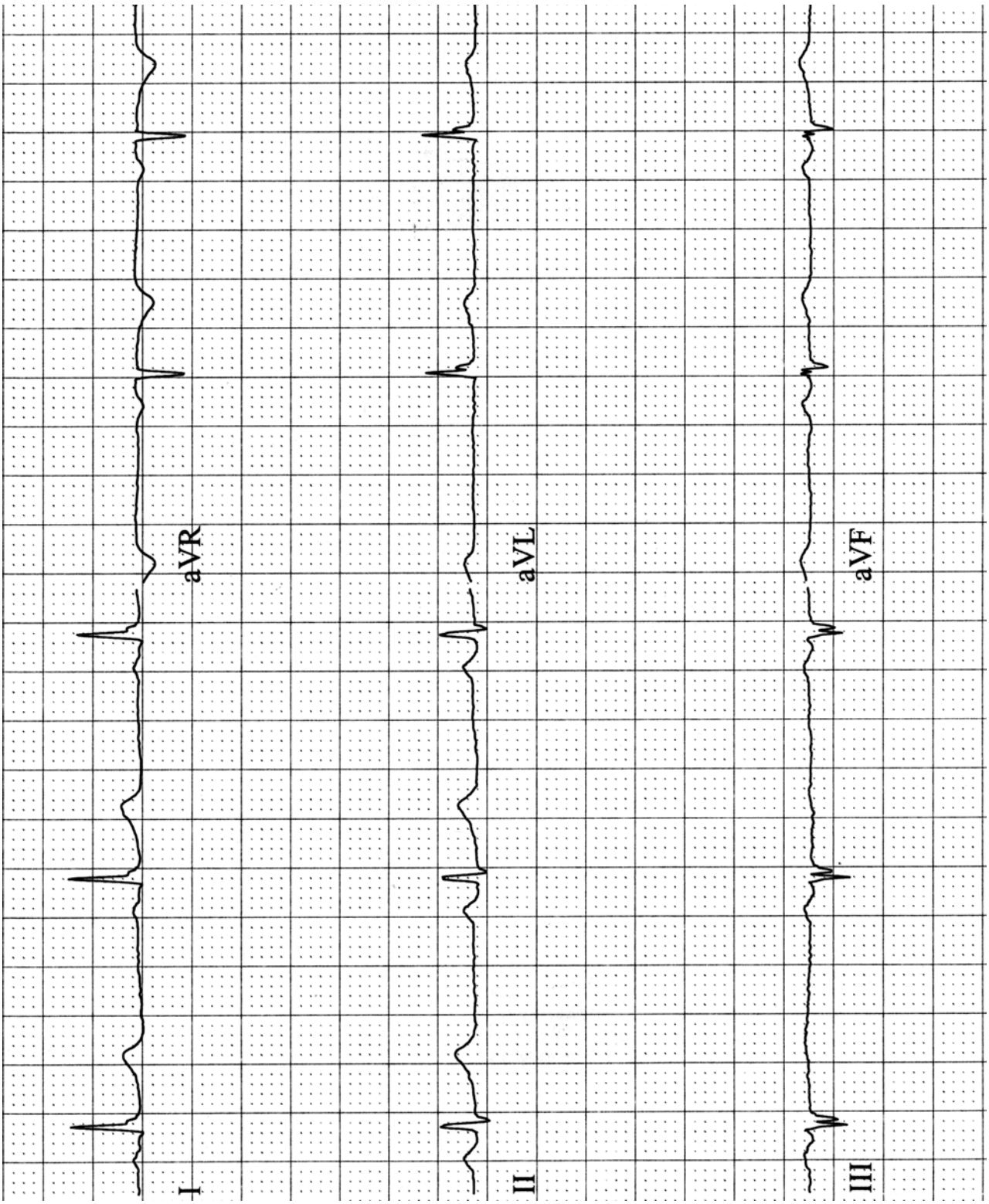
Exercise Tracing 2.9

Exercise Tracing 2.10

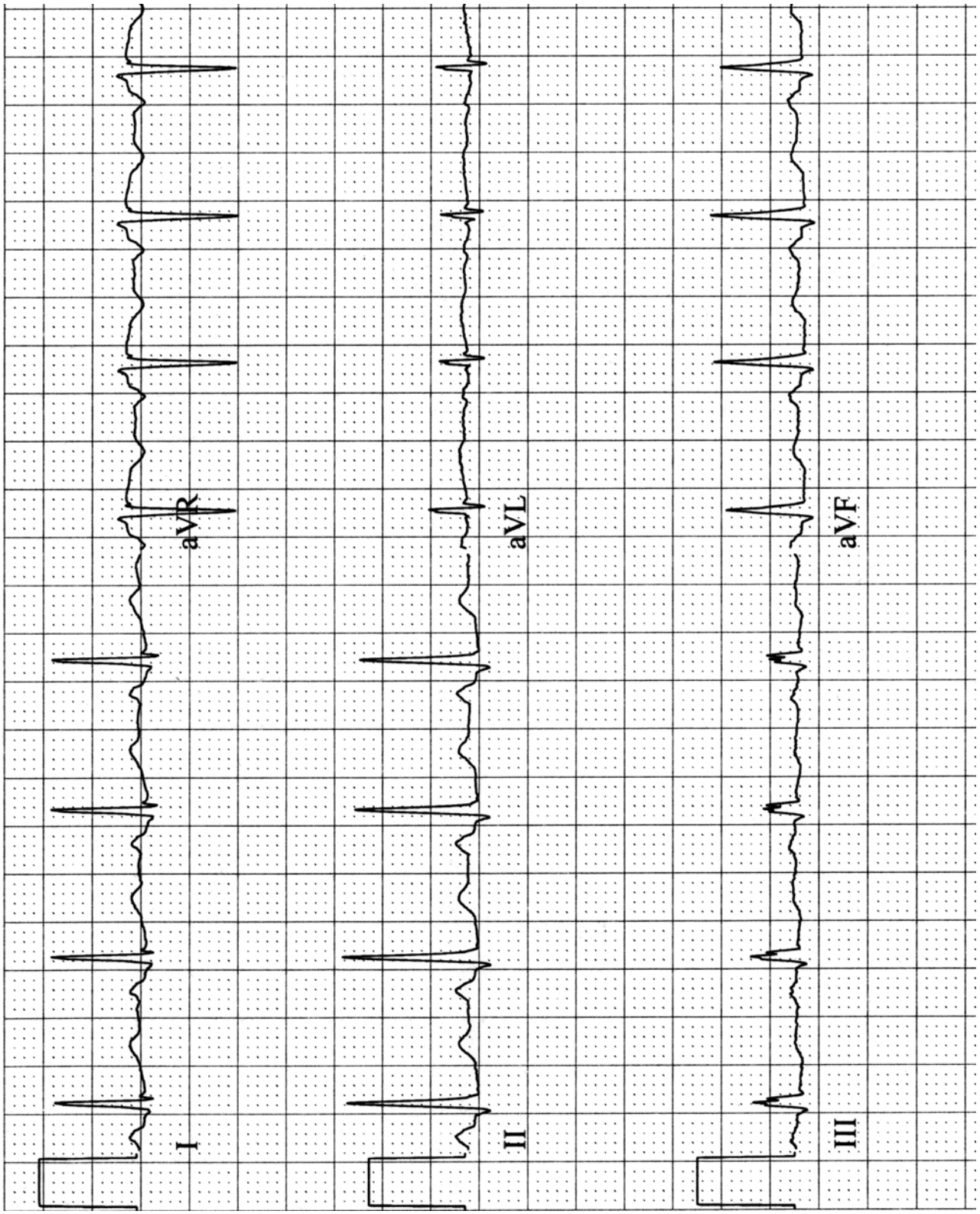




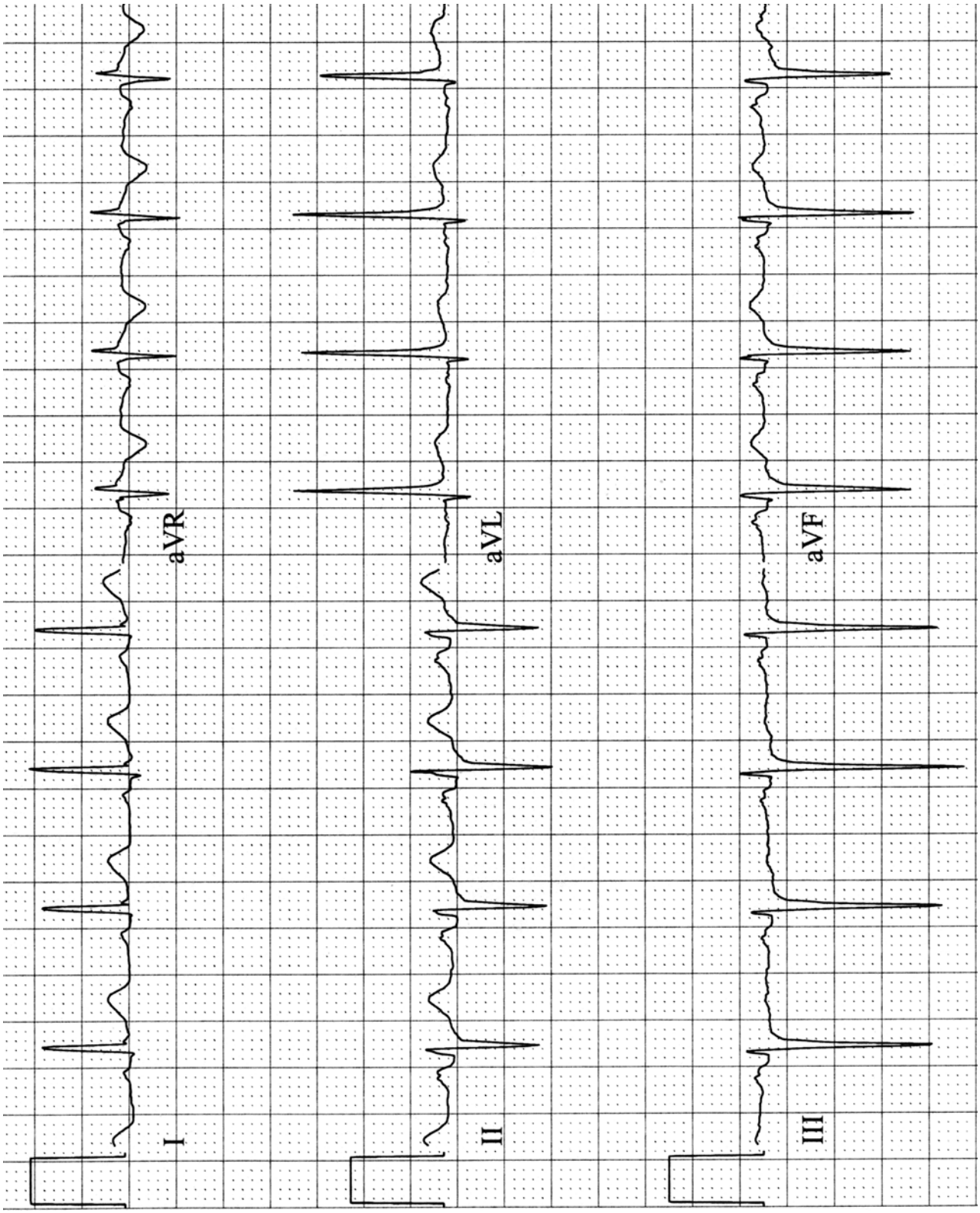
Exercise Tracing 2.11



Exercise Tracing 2.12



Exercise Tracing 2.13



Exercise Tracing 2.14

Exercise Tracings: Axis

Exercise Tracing 2.1. +30°
Exercise Tracing 2.2. −40
Exercise Tracing 2.3. +80°
Exercise Tracing 2.4. +120°
Exercise Tracing 2.5. −55°
Exercise Tracing 2.6. +10°
Exercise Tracing 2.7. −35°
Exercise Tracing 2.8. +70°
Exercise Tracing 2.9. Indeterminate
Exercise Tracing 2.10. +120°
Exercise Tracing 2.11. −70°
Exercise Tracing 2.12. −10°
Exercise Tracing 2.13. +50°
Exercise Tracing 2.14. −50°

Myocardial Infarction and Ischemia

3

Myocardial infarction occurs when oxygen delivery to the myocardium is inadequate for metabolic requirements and remains inadequate to the point of cellular death. This is in contrast to myocardial ischemia, wherein there is also inadequate oxygen delivery to the myocardium, but it is only temporary and is not prolonged or severe enough to lead to cell death. There is clearly a spectrum of response to inadequate oxygen delivery to the myocardium, ranging from mild ischemia through moderate and finally severe ischemia, and, if not reversible, ultimately to myocardial infarction (Fig. 3.1). The electrocardiographic appearance one observes depends on where on this spectrum the patient's oxygen compromise is located and which portion of the myocardium is affected.

Ischemia

Ischemia is usually manifested by *reversible ST segment depression*. The reversibility of the changes is critical to the diagnosis of ischemia. The changes are reversible within a period of minutes, not weeks or months as may be the case with the changes of infarction. The ST segment depression may be downsloping, horizontal, or upsloping in configuration (Fig. 3.2). The most specific configuration for ischemia is downsloping ST segment depression, but this may occur in other situations as well (e.g., left ventricular hypertrophy—see Chap. 6) so it is not completely specific for ischemia. What is most specific for ischemia is *reversible* downsloping ST segment depression associated with symptoms of angina (e.g., substernal pressing chest pain, dyspnea, diaphoresis). Horizontal ST segment depression is not as specific as downsloping ST depression for ischemia, and upsloping ST depression is the least specific. But horizontal and even upsloping ST segment depressions which are associated with symptoms and are reversible do indicate ischemia. Upsloping ST segment depression can sometimes be seen at rapid heart rates without symptoms and does not indicate ischemia, but rather is due to atrial repolarization. These changes are called T_A waves.

Two additional manifestations of ischemia should be mentioned. A less common form of ischemia than that described above is called Prinzmetal's angina, and is associated with coronary artery spasm with or without underlying fixed obstructive atherosclerotic disease. In Prinzmetal's angina, the ST segments are elevated, rather than depressed, and revert to normal over a period of minutes after the vasospasm-induced ischemia is resolved. The distinction between Prinzmetal's angina and transmural infarction, which is also associated with ST segment elevation (see below), is the rapid reversibility of the ST changes with Prinzmetal's angina as opposed to the persisting elevation in ST segments (hours to days) with transmural infarction. Finally, in rare circumstances, ischemia may be reflected on the electrocardiogram (EKG) by T wave inversions. This is very uncommon, and T wave inversions are more likely to be related to subendocardial infarction (see below). Nevertheless, when T wave inversions occur, are associated with symptoms of angina, and are reversible within minutes when symptoms resolve, that probably indicates ischemia.

Myocardial Infarction

There are two types of myocardial infarction, and the names have changed over the course of time, with three pairs of terms (Table 3.1).

The oldest designation of the three is (1) transmural and (2) subendocardial, or non-transmural. These two categories imply an anatomical correlation between the appearance of the EKG and the extent of the damage to the heart wall. This implication, however, is not always correct, and hence the attraction to alternative terminology is based on the EKG changes. When the EKG shows the changes of transmural infarction, anatomical studies sometimes but not always confirm an infarction through the *entire thickness* ("trans-") of the heart *wall* ("-mural"). Likewise, when the EKG shows a subendocardial infarction, the damage may not be limited to

B.G. Petty, *Basic Electrocardiography*, DOI 10.1007/978-1-4939-2413-4_3,

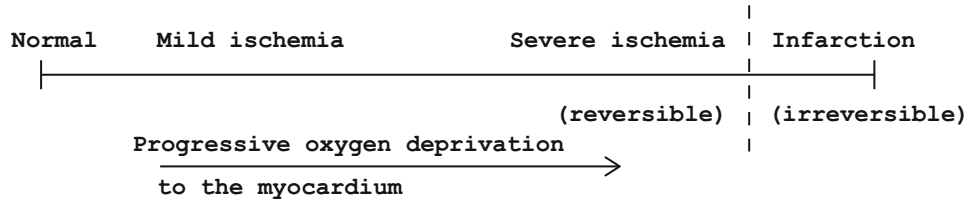


Fig. 3.1 Spectrum of injury from cardiac oxygen deprivation

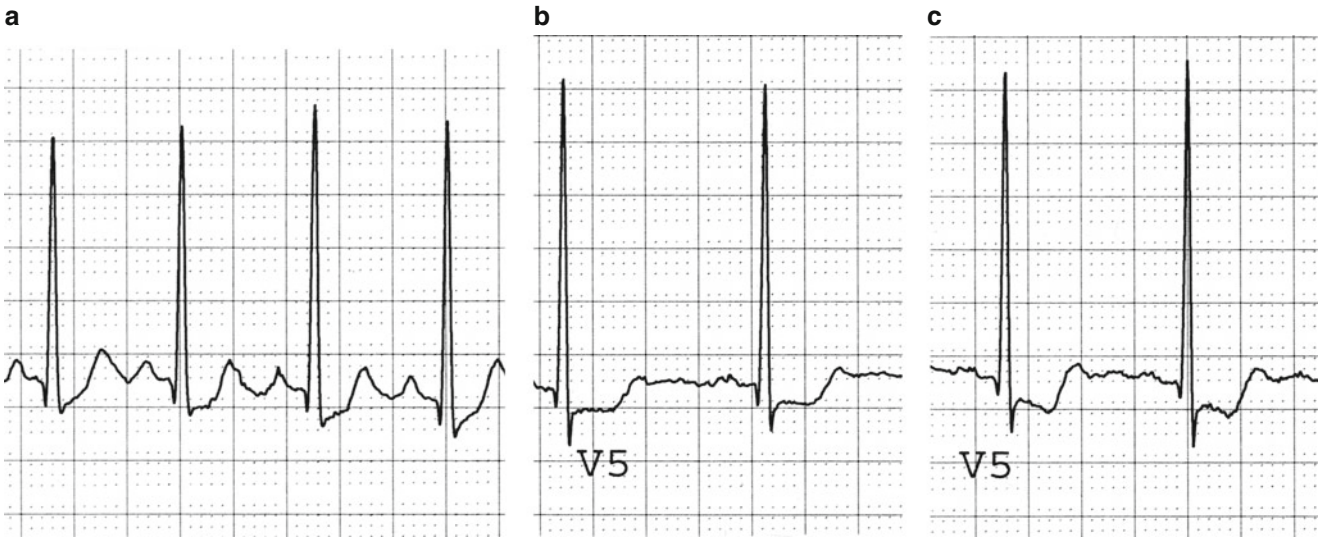


Fig. 3.2 Ischemia. (**a**) Upsloping ST segment depression. (**b**) Horizontal ST segment depression. (**c**) Downsloping ST segment depression

Table 3.1 Designations of myocardial infarctions

Transmural	Subendocardial
Q wave	Non-Q wave
ST elevation	Non-ST elevation

the subendocardium, but instead may be transmural, based on anatomical correlations [1]. In general, an anatomical transmural infarction associated with the electrocardiographic changes characteristic of "subendocardial" infarction is smaller in magnitude than a transmural infarction with typical transmural changes on the EKG.

Because of the discordance between anatomical findings and the electrocardiographic changes indicating "transmural" or "subendocardial" infarction, the terminology for infarctions changed to first "Q wave," then "ST elevation" myocardial infarction (STEMI) vs. "non-Q wave" or "non-ST elevation" myocardial infarction (non-STEMI), respectively [2]. Under this classification, the anatomical overtones are dropped, but the association of Q wave or ST elevation infarctions with higher in-hospital mortality, reduced ejection fraction, greater evidence of left ventricular failure, and less postinfarction exercise-induced ischemia compared to non-Q wave or non-ST elevation infarctions is similar to the findings in previous studies comparing "transmural" and "non-transmural" or "subendocardial" infarctions, respectively [3]. STEMIs have a higher in-hospital mortality rate than non-STEMI [4]. That non-STEMIs carry a serious prognosis, however, is clear. Reports suggest that the overall long-term mortality of myocardial infarction patients is the same if not greater after non-STEMI compared to STEMI [3, 5–8]. Non-STEMIs probably represent smaller infarctions with a larger area of myocardium still at risk for future infarction [3, 7]. This notion is supported by studies showing a higher frequency of reinfarction in patients with non-STEMIs compared to patients with STEMIs. The reinfarctions frequently occurred within weeks of the initial infarction and carried a poor prognosis [9, 10].

Subendocardial/Non-Q Wave/Non-ST Elevation Myocardial Infarction

The electrocardiographic change of a non-STEMI is T wave inversion (Fig. 3.3). There is usually little or no depression of the ST segments. Over a period of weeks to months, the acute changes of T wave inversion *may* revert back to normal, with no residual change to indicate a previous infarction. Alternatively, the T wave inversion may persist in some degree indefinitely.

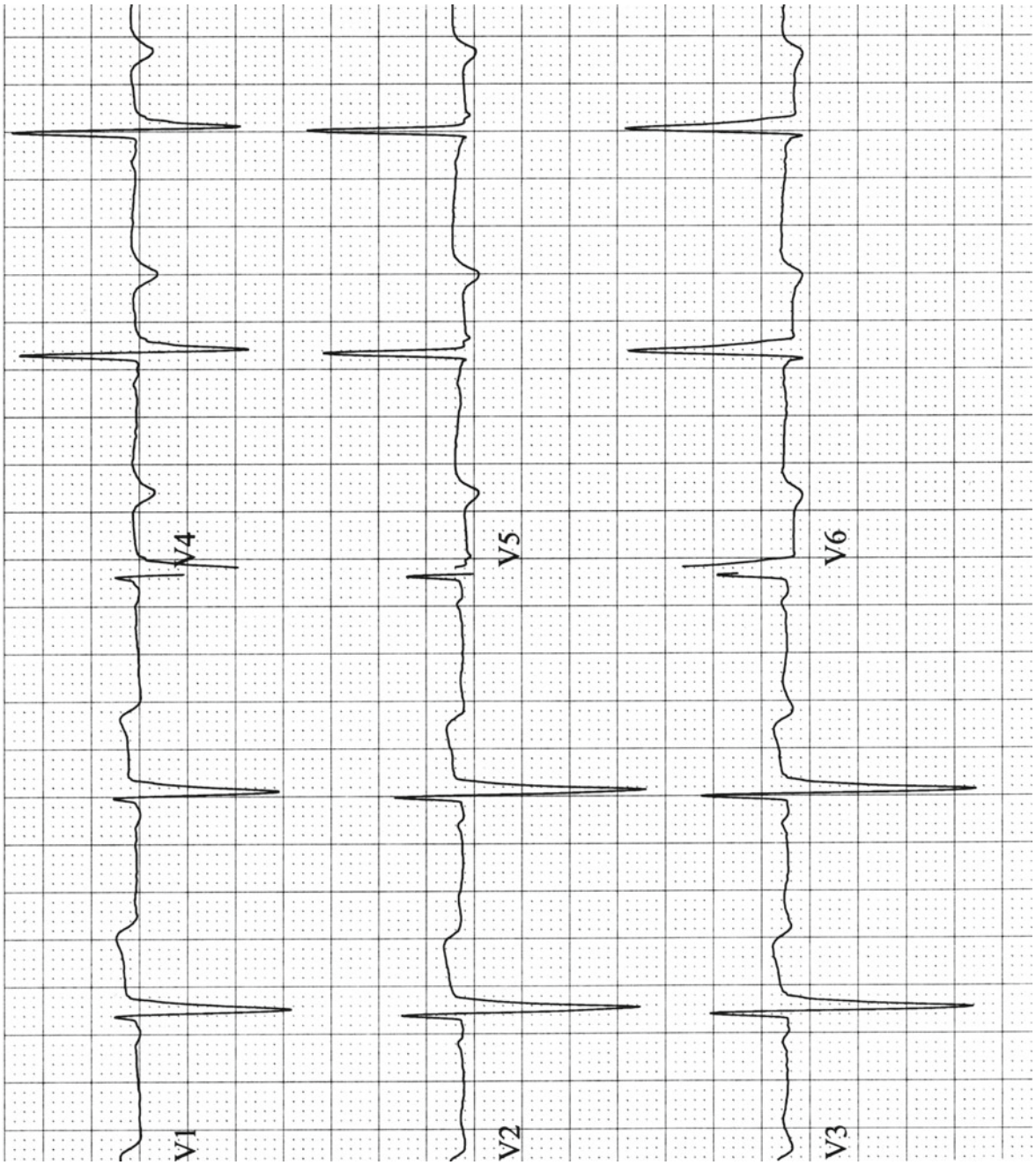


Fig. 3.3 Subendocardial infarction. These are the precordial leads in a patient with an acute anterior subendocardial infarction. Note that the ST segments are essentially normal, but the T waves are biphasic in V_3 and inverted in $V_{4–6}$

Transmural/Q Wave/ST Elevation Myocardial Infarction

The electrocardiographic changes associated with an acute STEMI follow a more complicated pattern (Fig. 3.4). The initial change is ST segment elevation, occurring usually within minutes of the interruption of oxygen delivery. The next change is the development of "pathological Q waves," Q waves that are new and are of at least 0.04 s duration. The Q waves appear within several hours of the infarction. Over the next few days, the T waves become inverted and the ST segments become less elevated, usually reverting to normal. Over a period of weeks to months, the T wave inversions may resolve, leaving only the Q waves as the residual manifestation of the infarction. This series of changes is known as the evolution of an acute STEMI. Occasionally, the Q waves that remain are not wide and deep and may be indistinguishable from the q waves that can be found as a normal variant. Thus, following either a STEMI or a non-STEMI, it is possible for the EKG to revert to a pattern that is within normal limits and does not reflect the previous infarction. This is more likely to occur with non-STEMI than STEMI, but it can occur with both.

Very rarely the first changes on the EKG seen with an acute STEMI are peaked T waves, the so-called hyperacute T waves. This is so rare as to only deserve mention for completeness. It is *not* a common finding, and when it does occur, it is soon supplanted by the far more typical ST segment elevations described earlier.

Sometimes patients are seen with myocardial infarction, suspected by the patient's history and confirmed by a typical and significant elevation of cardiac enzymes, but without any electrocardiographic abnormalities [2]. These "electrically silent" or "normal electrocardiographic" infarctions are uncommon.

Location of Infarction/Ischemia

There are three general EKG locations for infarctions or ischemia: inferior, anterior, and posterior. The portion of the heart that is affected by infarction or ischemia may be suspected by the electrocardiographic leads in which the changes are found. Even if the correlation is imperfect between the leads reflecting ischemia or infarction and the part of the heart affected, the designation of the location quickly identifies the leads showing changes. As discussed in Chap. 2, leads II, III, and aVF are positive in the legs and negative in the arms, and therefore are most reflective of changes in the inferior wall of the heart and are called the inferior leads. Leads I and aVL, along with the precordial leads, reflect changes in the anterior wall of the heart. Therefore, when there is ischemia or infarction in the inferior wall of the heart, the electrocardiographic changes described earlier in this chapter are typically seen in the inferior leads—II, III, and aVF. When there is anterior wall ischemia or infarction, the changes are typically seen in leads I, aVL, and the precordial leads. Occasionally a distinction is made between the anteroseptal and anterolateral portions of the anterior wall. "Anteroseptal" and "anterolateral" are simply subsegments of the anterior wall, with changes limited to $V_{1–3}$ or $V_{4–6}$, respectively. I believe this subdivision of the anterior wall in reference to myocardial injury is of little practical importance, but an anteroseptal infarction with precordial changes limited to $V_{1–3}$ probably does suggest a less extensive infarction than when the changes are seen in most or all of the precordial leads. The posterior wall of the heart is not reflected directly by any of the standard electrocardiographic leads because none of them are placed on the patient's back. Instead, posterior wall injury is indirectly reflected by changes in leads $V_{1–2}$. Since those two leads are directly anterior, they best show changes affecting the posterior wall of the heart, but the changes are reversed from the usual changes of damage described above. An acute posterior STEMI is reflected by ST segment depression and a tall R wave in $V_{1–2}$ as opposed to ST segment elevation and a Q wave. This is because $V_{1–2}$ are anterior leads, and in reflecting posterior wall injury these leads would be expected to have opposite changes compared to those seen when the area of injury is directly below the leads. Another way to look at this situation is that the complexes in $V_{1–2}$ can be rotated around the baseline as an axis when considering the posterior wall of the heart, so ST segment depression becomes ST segment elevation, and the R wave becomes a Q wave in posterior infarction (Fig. 3.5). The presence of ST segment depression in $V_{1–4}$ in association with changes of acute inferior STEMI is usually due to inferoposterior or posterolateral wall involvement of the infarction rather than a "reciprocal change" [11] (see below).

Even though the area of the heart damaged in an infarction can be suggested on the EKG, ascribing the event to the coronary artery involved is less predictable than identifying the area of the heart that is damaged. *Generally*, the right coronary artery serves the inferior wall of the heart, the left anterior descending serves the anterior wall, and the circumflex serves the posterior wall with minor contributions to the inferior or lateral wall. Thus, in the setting of an inferior infarction, the right coronary artery is probably involved, while in an anterior infarction the left anterior descending artery is probably involved [12]. The common conduction problems with those infarctions can be predicted because the right coronary artery generally supplies the inferior wall of the heart and frequently sends a branch to the atrioventricular (AV) node, while the left anterior descending artery, usually involved in an anterior infarction, sends "septal perforator" arteries into the interventricular septum where the bundle of His and the bundle branches are located (Fig. 3.6). Thus, inferior infarctions are more frequently associated with AV node dysfunction, while anterior infarctions are more typically associated with dysfunction of the bundle system.

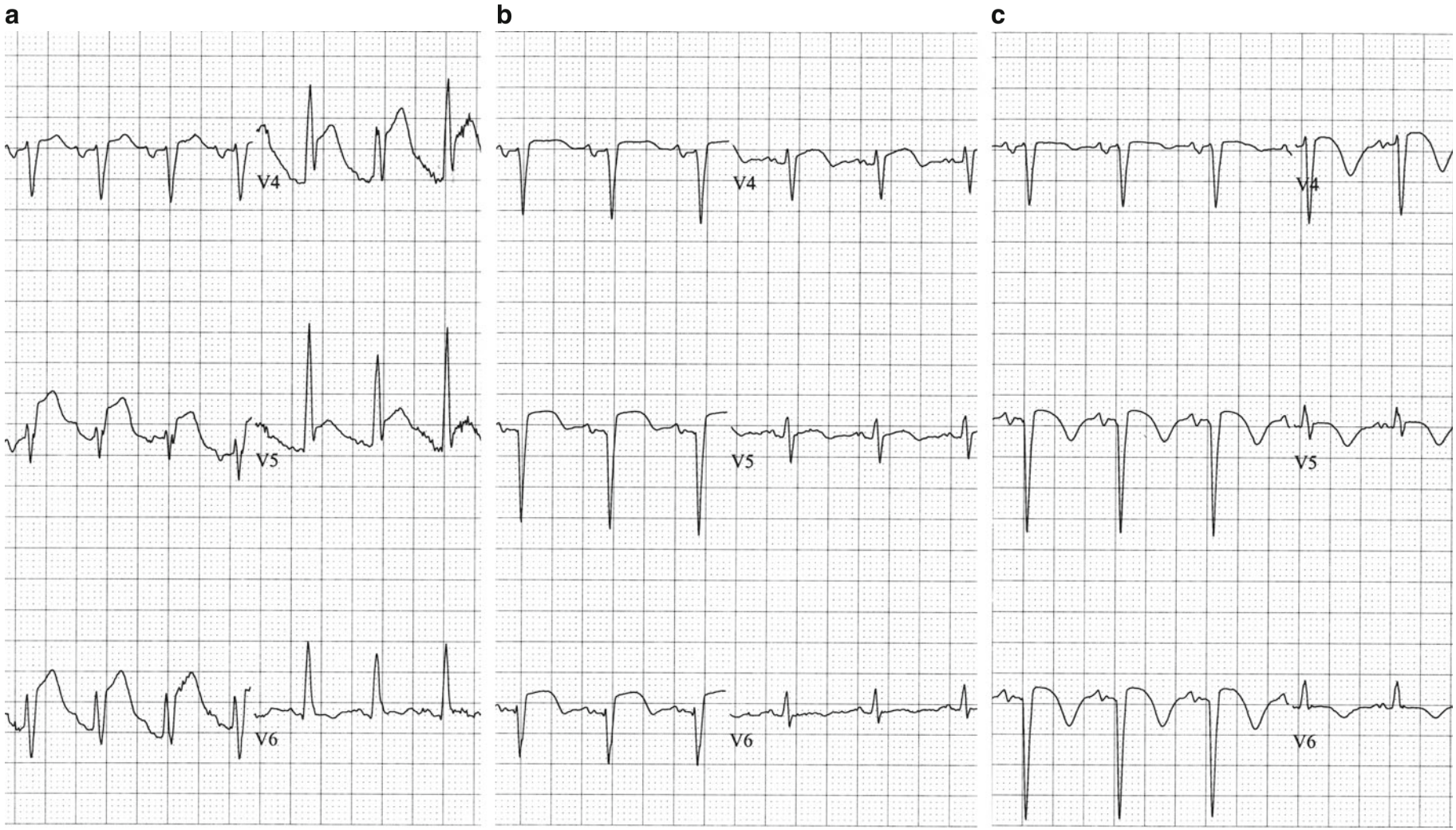


Fig. 3.4 Evolution of a transmural infarction. Precordial leads in a patient with an acute anterior transmural myocardial infarction. (**a**) At initial presentation the patient has acute ST segment elevation, only a small, nonpathological q wave in $V_{5–6}$, and no T wave inversions. (**b**) Four hours later there is a new Q wave in V_2 and less ST elevation remains. (**c**) One day after initial tracing the Q waves are in V_{2-3}, slight ST elevation persists, and the T waves are now inverted in $V_{2–6}$

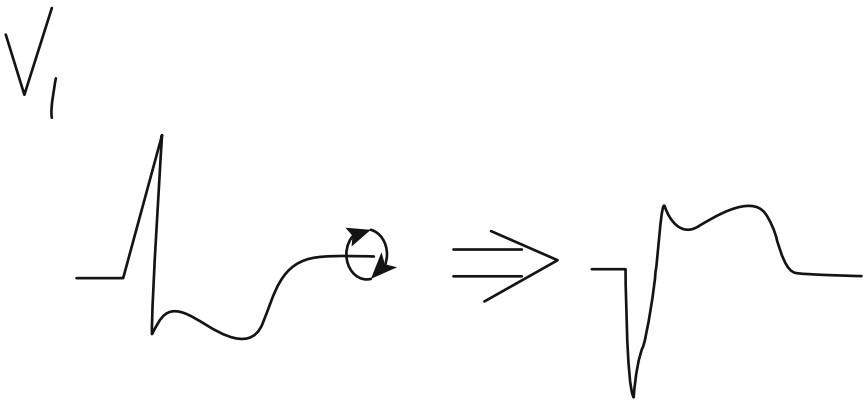


Fig. 3.5 Posterior infarction (diagrammatic). Lead V_1 has a tall R wave and ST depression. When the complex is rotated around the baseline as an axis, the R wave becomes a Q wave and the ST segment depression becomes ST segment elevation, now typical of an acute transmural myocardial infarction

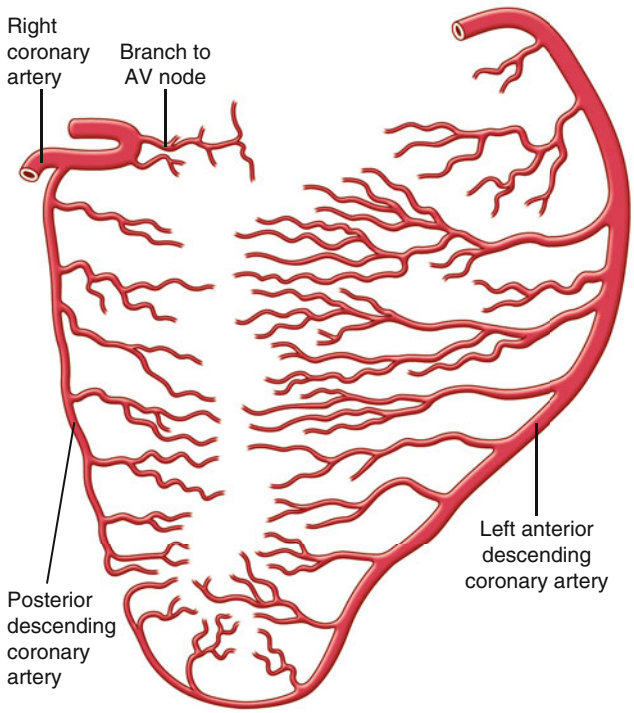


Fig. 3.6 Blood supply to the conduction system. The right coronary artery gives off a branch to the AV node, while the septal perforators off the left anterior descending artery are primarily responsible for blood supply to the interventricular system where the bundle system is located

Reciprocal Changes

Reciprocal changes are seen with STEMIs, never non-STEMIs, and are sometimes present with Prinzmetal's angina (see below). Reciprocal changes consist of ST segment depressions (reminiscent of ischemic changes) in leads "opposite" from those that show the typical ST segment elevations described above for STEMIs (sometime called "injury currents").

For example, in inferior STEMIs, with ST segment elevations in II, III, and aVF, there may be ST segment depressions as reciprocal changes in leads I and aVL, and perhaps in some precordial leads. On the other hand, in anterior STEMIs, with ST elevations in I, aVL and some precordial leads, there may be ST depressions in II, III, and aVF as reciprocal changes (Fig. 3.7).

Reciprocal changes are primarily observed in the limb leads and less often in the precordial leads and are seen more often in inferior than anterior infarctions [13, 14]. The degree of ST depression as a reciprocal change is proportional to the magnitude of ST elevation seen as an acute change in STEMIs [15]. Reciprocal changes are seen in approximately 50% of STEMIs, and their presence is associated with a more extensive infarction than when they are absent [16]. Accordingly, the prognosis for patients who have transmural infarction associated with reciprocal change is worse than that for patients whose infarctions are not associated with reciprocal change. The term "reciprocal change" implies secondary electrical change in the leads "opposite" those showing the primary acute injury, rather than ischemia in another part of the heart in addition to the acute infarction [14–16].

ST Elevation: Differential Diagnosis

Several conditions can cause ST segment elevation. To this point we have covered three such conditions: (1) acute STEMI, (2) Prinzmetal's angina, and (3) junctional ST segment elevation/early repolarization (Fig. 3.8 and Chap. 1). While most cases of Prinzmetal's angina are "idiopathic," hypomagnesemia has been reported to cause spasm of coronary arteries [17, 18]. There are two other conditions that can lead to ST segment elevation: (1) acute pericarditis, and (2) ventricular aneurysm.

Acute pericarditis is typically associated with ST segment elevation that resembles early repolarization. But the ST elevations are diffusely present in almost all the leads, not just in an inferior or anterior distribution. Additionally, there may be PR segment depression but there are never reciprocal changes with acute pericarditis.

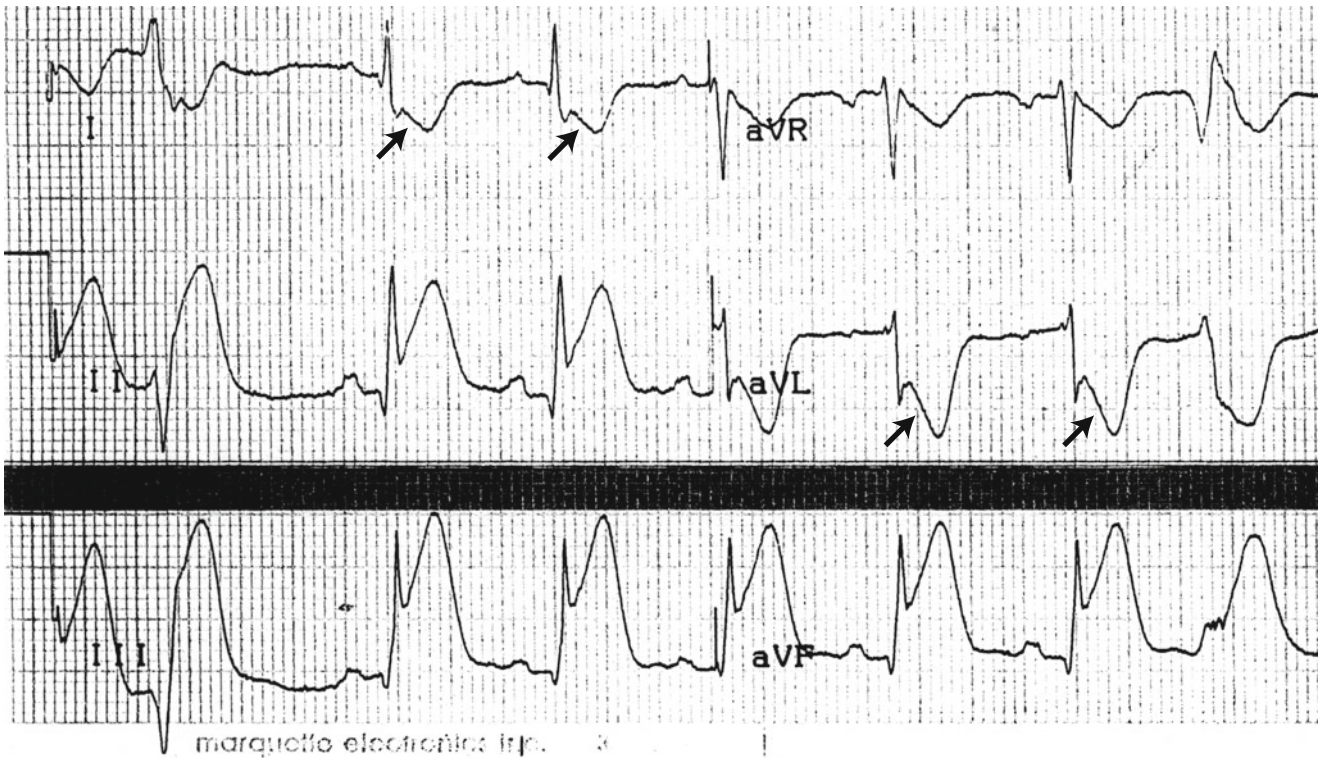


Fig. 3.7 Reciprocal changes. These are the limb leads of a patient with an acute inferior transmural myocardial infarction. There is marked ST segment elevation in II, III, and aVF, but reciprocal ST segment depression in leads I and aVL (*arrows*)

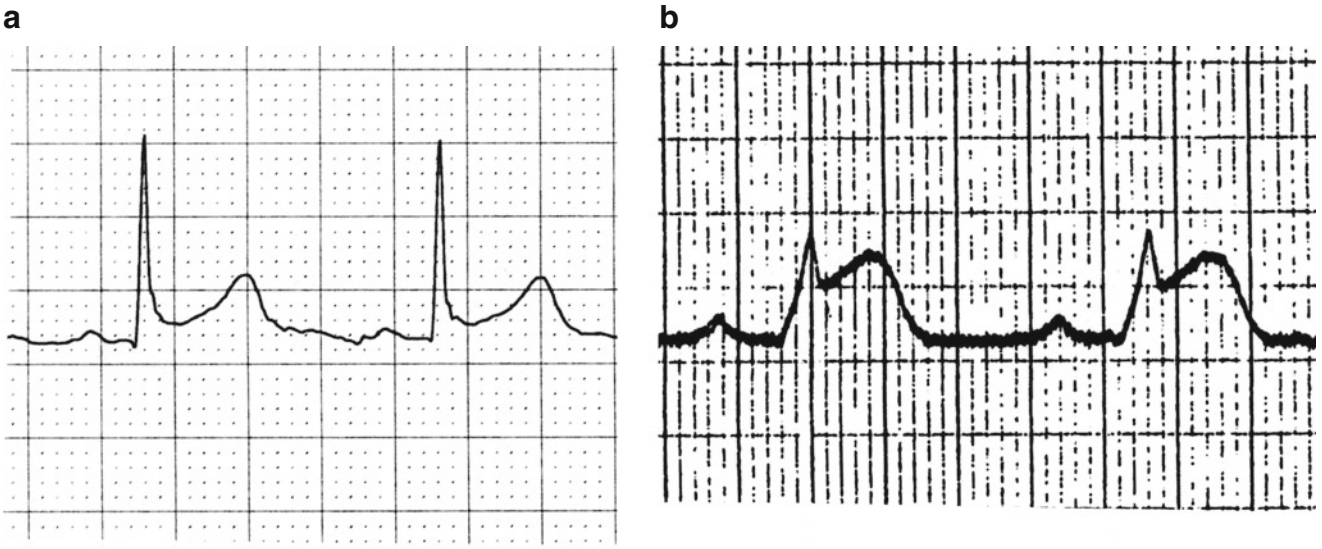
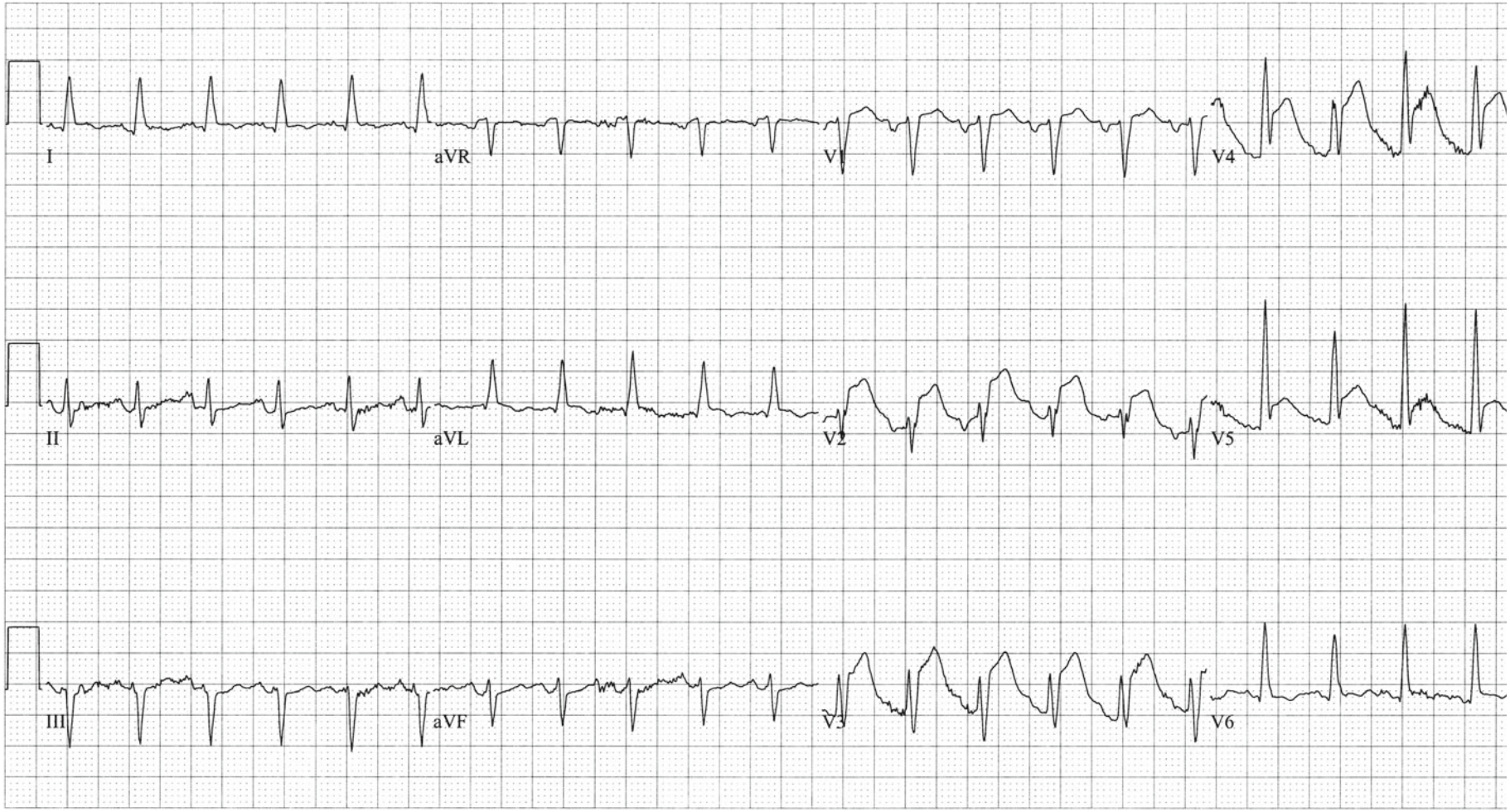


Fig. 3.8 Early repolarization (**a**) vs. acute transmural myocardial infarction (**b**). Note the sharp angle where the ST segment comes off the QRS complex with the infarction, while early repolarization has a smoothly curved ST segment

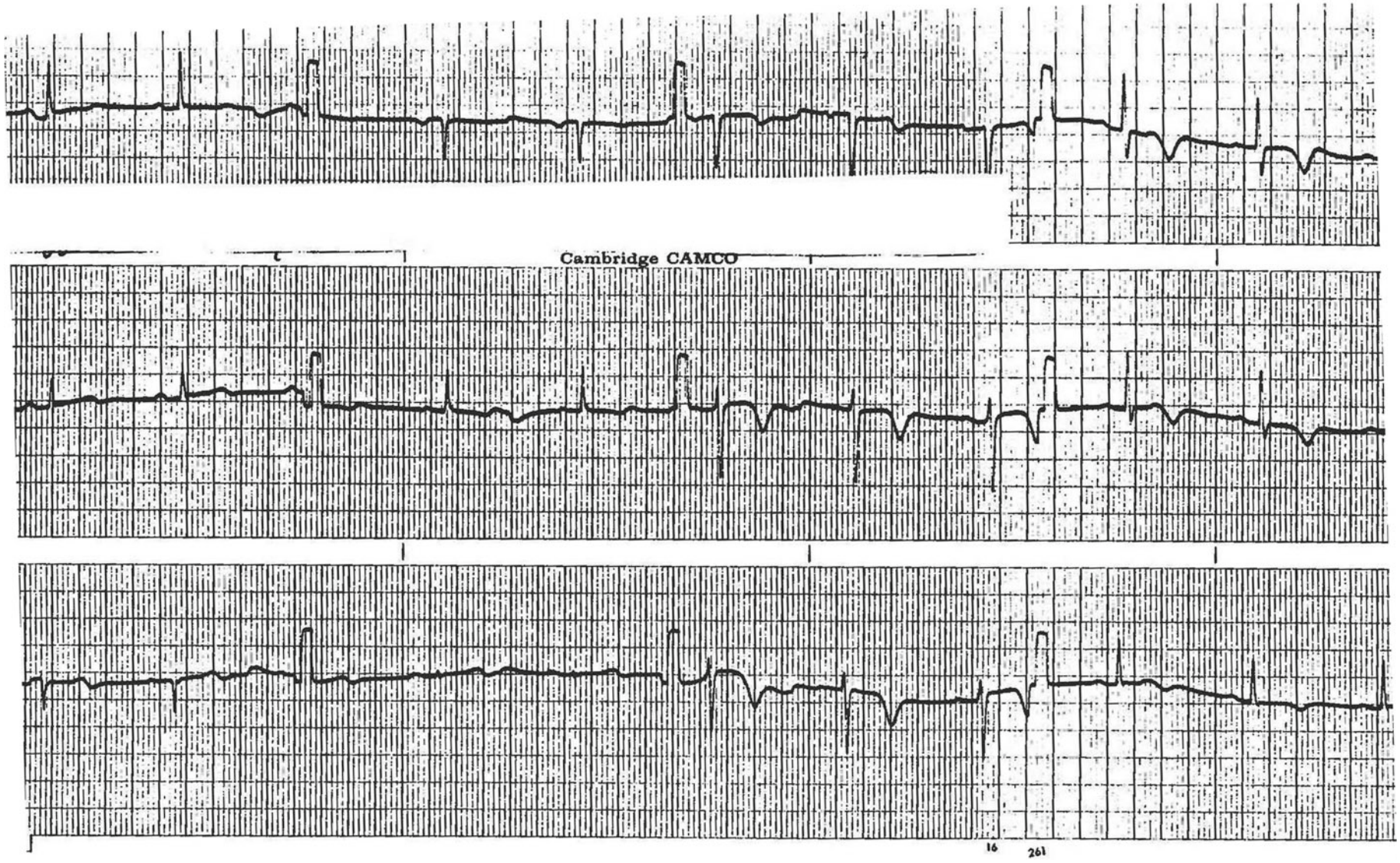
Ventricular aneurysms are usually due to previous STEMI, most commonly involving the anterior wall of the heart. The ST segments are typically elevated in the same leads associated with the previous STEMI and where the current ventricular aneurysm is located. It appears as if the acute ST elevations of a STEMI never resolved, in contrast to the usual evolution of myocardial infarction. So when ST segment elevations do not resolve following an acute STEMI, one should suspect the development of a ventricular aneurysm.

Exercise Tracings

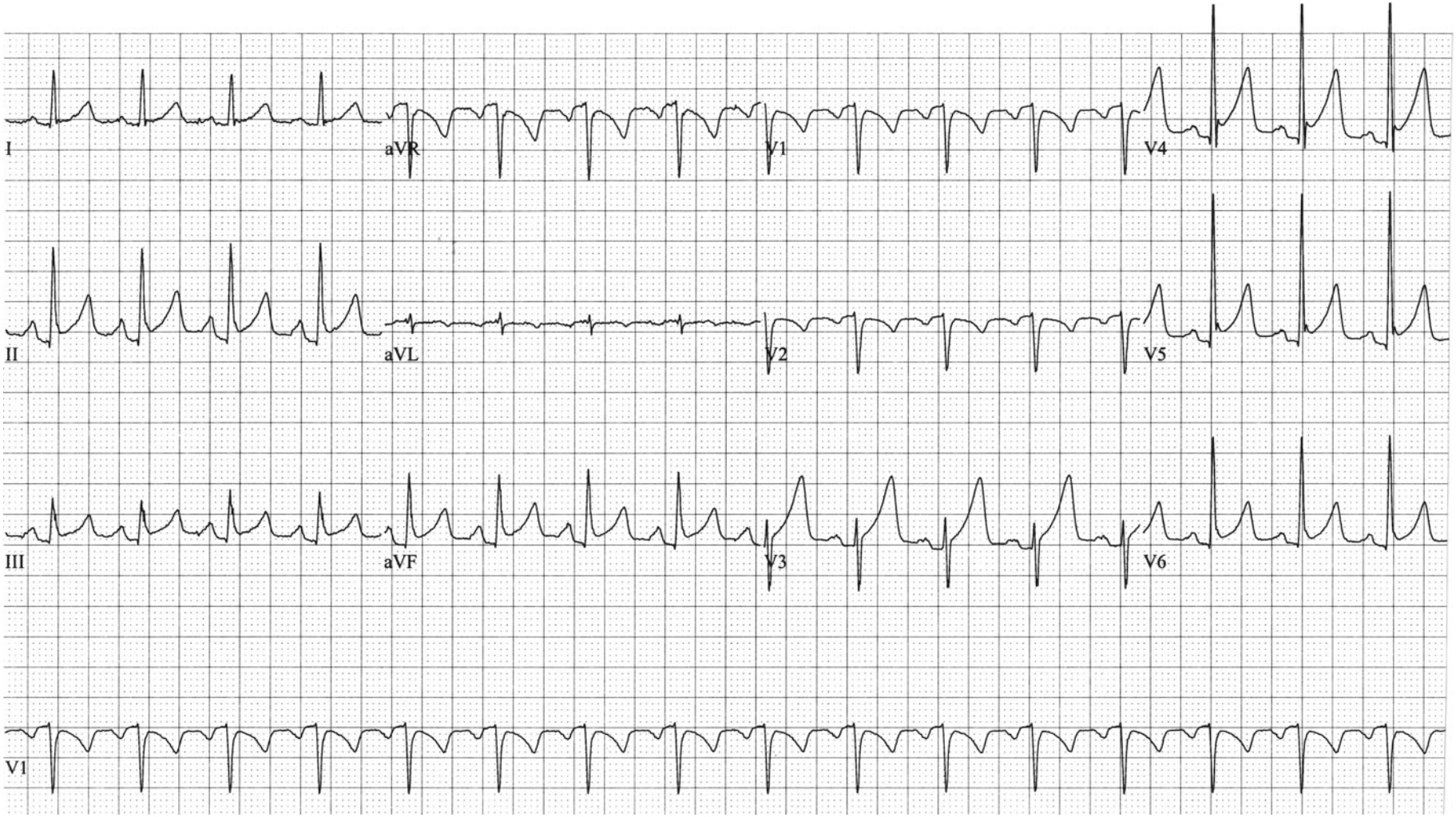
Exercise Tracing 3.1



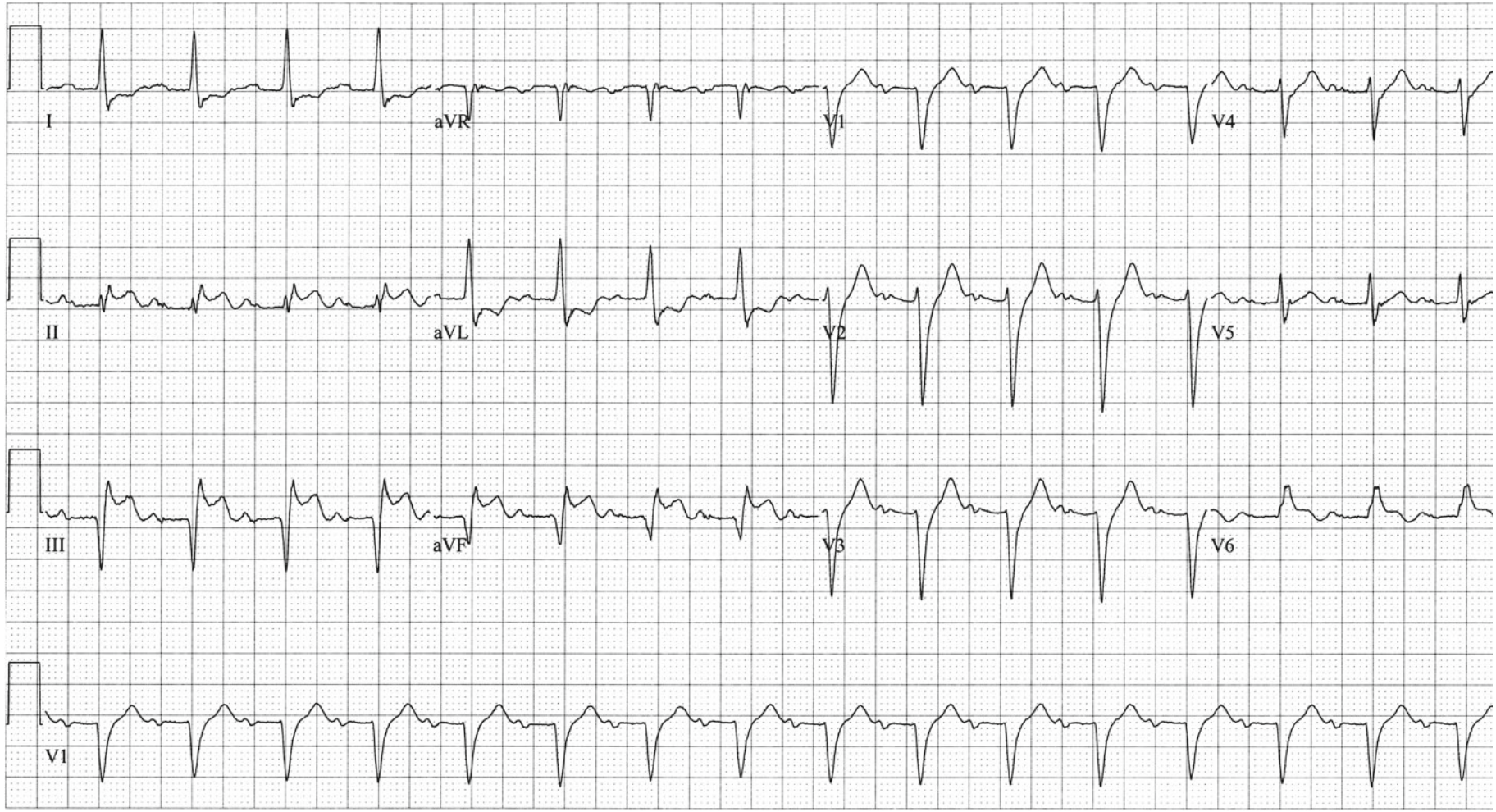
Exercise Tracing 3.2



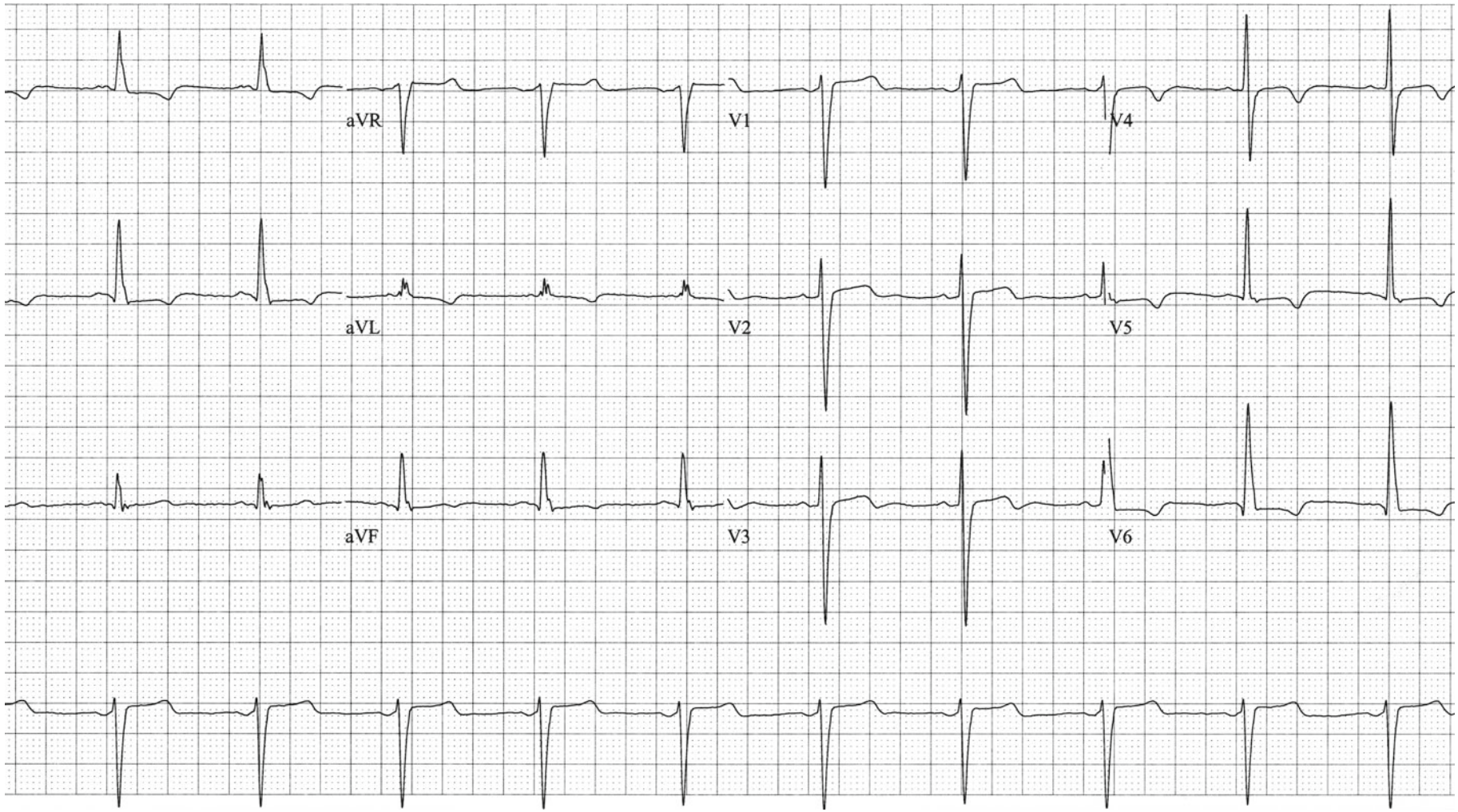
Exercise Tracing 3.3



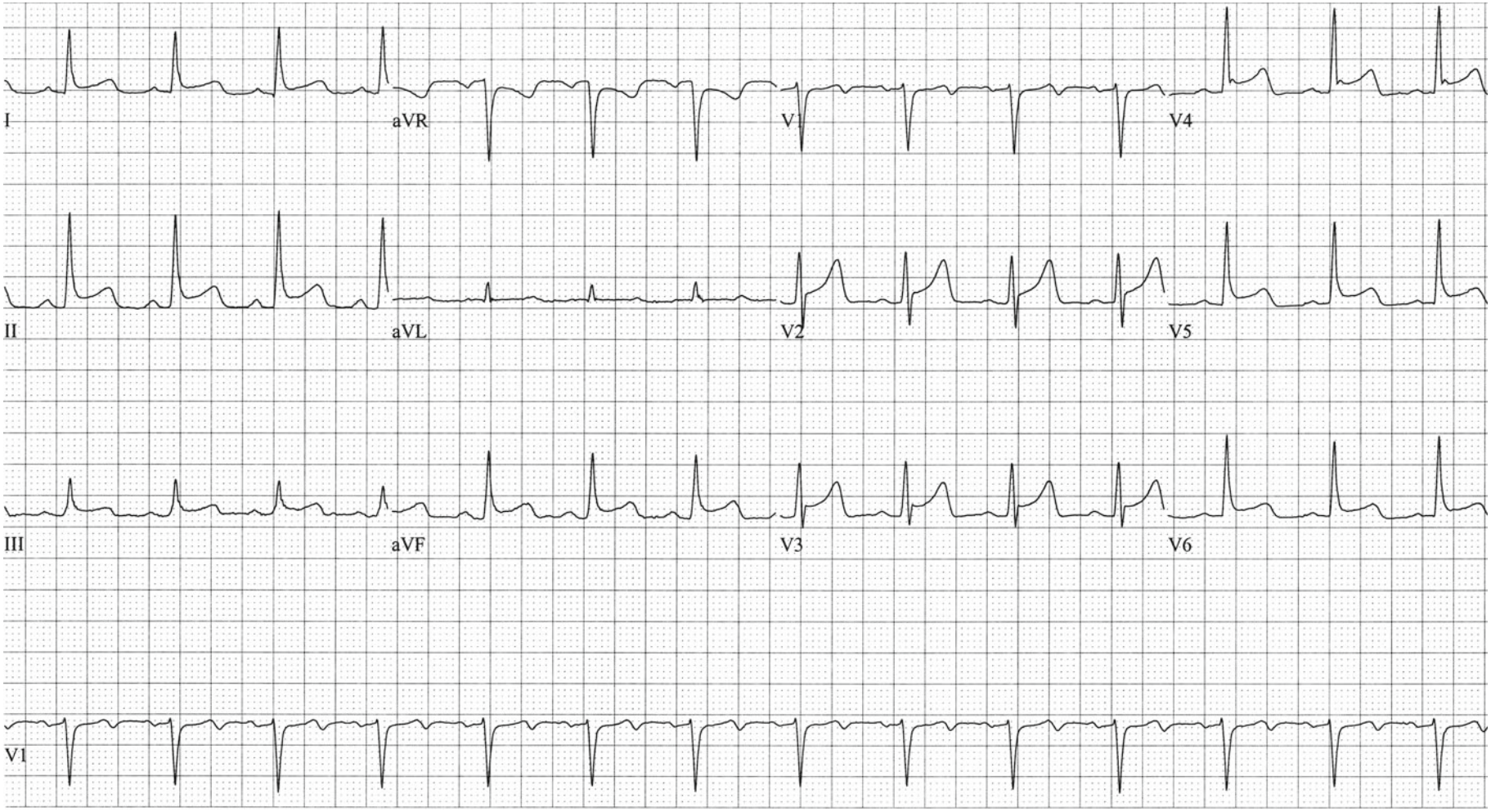
Exercise Tracing 3.4



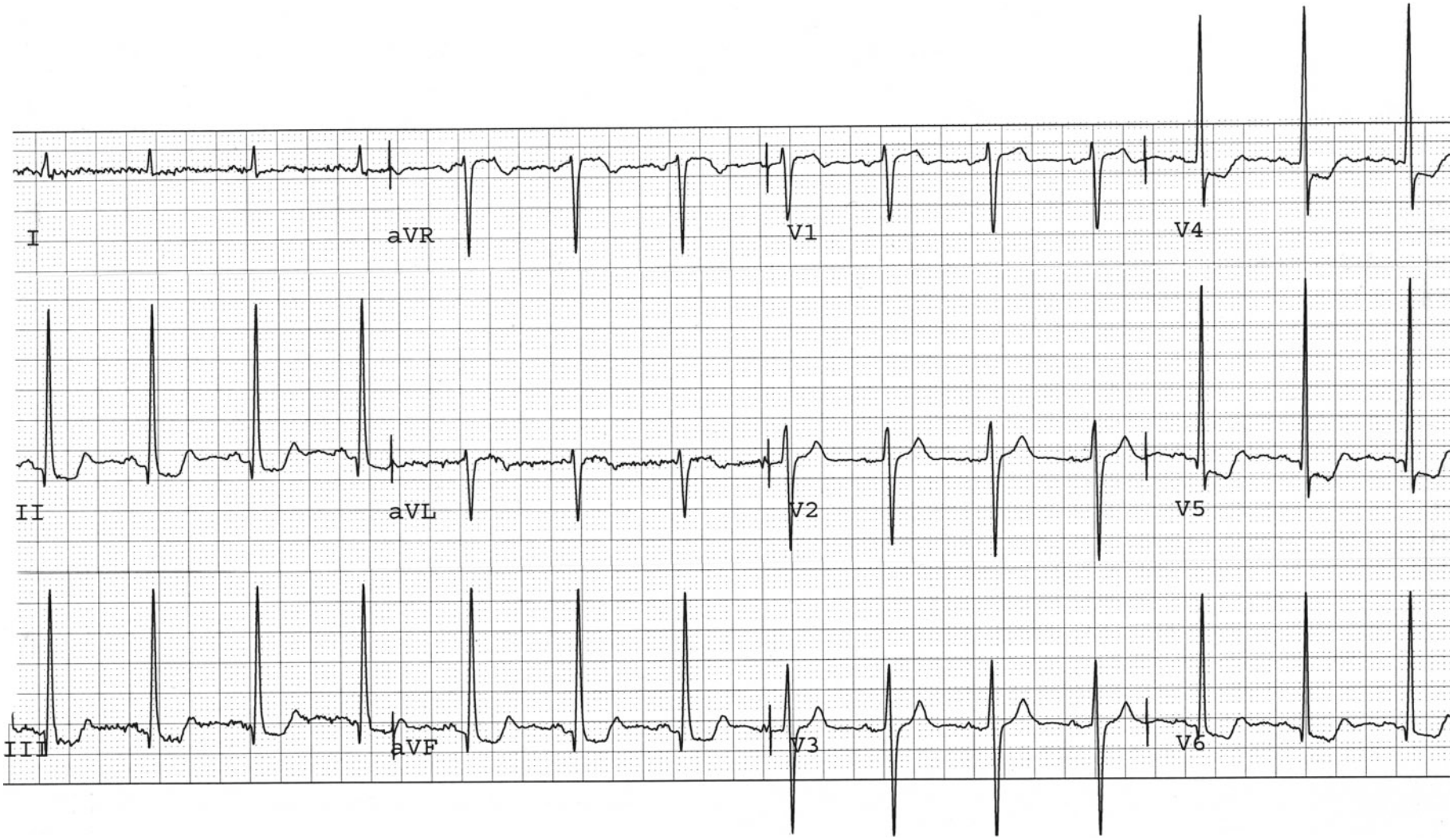
Exercise Tracing 3.5



Exercise Tracing 3.6



Exercise Tracing 3.7



Interpretations of Exercise Tracings

Exercise Tracing 3.1	
RATE:	A 130 V 130
RHYTHM:	Sinus tachycardia
AXIS:	–20°
INTERVALS:	PR 0.15 QRS 0.10 QT 0.30
WAVEFORM:	ST elevation in V1-V5; inverted T waves in I, aVL
SUMMARY:	Abnormal due to acute anterior wall ST elevation myocardial infarction, leftward axis deviation

Exercise Tracing 3.2	
RATE:	A 64 V 64
RHYTHM:	Normal sinus rhythm
AXIS:	0°
INTERVALS:	PR 0.16 QRS 0.08 QT 0.38
WAVEFORM:	T wave biphasic in I, aVL, inverted in $V_{2–6}$
SUMMARY:	Abnormal due to acute subendocardial anterior myocardial infarction

Exercise Tracing 3.3	
RATE:	A 102 V 102
RHYTHM:	Sinus tachycardia
AXIS:	+60°
INTERVALS:	PR 0.16 QRS 0.07 QT 0.34
WAVEFORM:	Diffuse ST elevation; PR segment depression in the inferior leads, especially lead II; inverted P waves in V_{1-2}
SUMMARY:	Abnormal due to ST changes and PR depression compatible with pericarditis; left atrial abnormality

Exercise Tracing 3.4	
RATE:	A 105 V 105
RHYTHM:	Sinus tachycardia
AXIS:	0°
INTERVALS:	PR 0.25 QRS 0.10 QT 0.30
WAVEFORM:	ST elevation in II, III, aVF; ST depression in I and aVL; Q III, aVF, Q in III, aVF
SUMMARY:	Abnormal due to acute inferior ST elevation myocardial infarction with reciprocal changes, first degree AV block (see Chap. 4)"

Exercise Tracing 3.5	
RATE:	A 65 V 65
RHYTHM:	Normal sinus rhythm
AXIS:	50°
INTERVALS:	PR 0.15 QRS 0.11 QT 0.44
WAVEFORM:	T wave inversions in I, II, aVL, $V_{4–6}$; non-pathological q waves in II, III, aVF, $V_{5–6}$
SUMMARY:	Abnormal due to acute subendocardial anterolateral myocardial infarction, inferior infarction, left axis deviation, prolonged QT interval and U waves subendocardial myocardial infarction

Exercise Tracing 3.6	
RATE:	A 86 V 86
RHYTHM:	Normal sinus rhythm
AXIS:	+50°
INTERVALS:	PR 0.16 QRS 0.09 QT 0.36
WAVEFORM:	Smooth ST elevation I, II, II, aVF, $V_{2–6}$; early R wave development $V_{2–3}$
SUMMARY:	Abnormal due to acute pericarditis

Exercise Tracing 3.7	
RATE:	A 88 V 88
RHYTHM:	Normal sinus rhythm
AXIS:	+80°
INTERVALS:	PR 0.16 QRS 0.09 QT 0.35
WAVEFORM:	ST depression II, III, aVF, $V_{4–6}$; tall R waves lead II and V_5
SUMMARY:	Abnormal due to inferolateral ischemia [NOTE: Even though this EKG meets the criteria for left ventricular hypertrophy (see Chap. 6), these ST segment depressions were quickly reversible, which confirms the diagnosis of ischemia]

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Atrioventricular (AV) Block

4

There are three types of atrioventricular (AV) node block: first-degree, second-degree, and third-degree. These are sometimes abbreviated 1°, 2°, and 3°, respectively (Table 4.1). These abbreviations should not be confused with "primary, secondary, and tertiary," which can carry the same annotation. While AV block may be a transient phenomenon (e.g., associated with ischemia, infarction, or drug intoxication), the block may be permanent.

First-degree AV block is simply a prolongation of the PR interval above the normal range, i.e., >0.20 s (Fig. 4.1). At slow heart rates the normal PR interval may extend up to 0.21 s, but for simplicity it is reasonable to read any prolongation of the PR interval over 0.20 s as first-degree AV block, and simply keep in mind that slight prolongations at slow heart rates are of little clinical consequence. In fact, first-degree AV block is essentially a benign condition and is very unlikely to be associated with progression to a higher degree of AV block [1].

Second-degree AV block is of two subtypes: Mobitz I and Mobitz II. Mobitz I is also commonly known as Wenckebach block. In Mobitz I (Wenckebach), there is gradual prolongation of the PR interval duration until finally one P wave is not conducted through the AV node to the ventricles (Fig. 4.2). As a consequence, a P wave is not followed by a QRS complex. Following the "dropped beat" (the missing QRS after a P wave), the PR interval is once again relatively short and then gradually prolongs again until another beat is dropped, and the cycle recurs. The length of the cycle (i.e., the number of conducted beats before the nonconducted beat) may vary. The extent of block is given in a ratio with the number of atrial beats observed in the cycle followed by a colon and then the number of ventricular beats in the cycle. For Mobitz I, the number of atrial beats in the cycle is always just one greater than the number of ventricular beats (e.g., 3:2, 4:3). This repetitive prolongation of PR intervals until a P wave is not conducted continues as long as the factor responsible for the block persists without improvement to a lower degree of block (first degree) or deterioration into a higher degree of block.

While the PR interval gradually increases in Mobitz I (Wenckebach), in classic cases the R–R interval actually *shortens*. This seems paradoxical, if not contradictory, at first glance. Yet the apparent paradox is possible because the *increment* of change in successive PR intervals gradually *decreases*, causing the R–R interval to shorten. This phenomenon is diagrammatically demonstrated in Fig. 4.3.

Even though in classic cases of Mobitz I the consecutive R–R intervals decrease, it is clear that this is not always true (Fig. 4.4).

Mobitz II is a higher degree of AV block than Mobitz I wherein a relatively constant fraction of P waves are conducted through the AV node to cause ventricular depolarization (Fig. 4.5). The PR intervals of the conducted beats are quite constant, rather than varying as in Mobitz I. The common ratios of conduction in Mobitz II are 3:1, 2:1, 4:1. While the ratio of P waves to QRS complexes is often constant, the ratio also may vary somewhat.

It should be noted that Mobitz II with a 2:1 block cannot be distinguished from Mobitz I block with a cycle length of only 2. In cases of 2:1 block, it is appropriate to simply call the phenomenon "second-degree AV block with 2:1 block" and not specify Mobitz I or II, unless other cycles clearly demonstrate the presence of one or the other (Fig. 4.6).

Mobitz I is generally considered to be a benign problem, primarily because it is usually transient in the setting of inferior myocardial infarction, and because in most cases only one beat out of every three or four fails to conduct through the AV node, so heart rate and cardiac output is not seriously affected. In the case of chronic Mobitz I, however, the prognosis appears to be just as poor as with chronic Mobitz II, with a 5-year survival of only about 60% [2].

Third-degree AV block is also known as complete heart block. It means that none of the P waves are being conducted through the AV node to the ventricles. As a consequence, the heart would stop depolarizing if "subsidiary pacemakers" below the point of block in the AV node did not take over the initiation of electrical activity (Fig. 4.7). These subsidiary pacemakers are in the node below the point of block, or in the

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Table 4.1 Atrioventricular block

First degree ("1°"): PR > 0.20 s
Second degree ("2°")
Mobitz I (Wenckebach): Gradual prolongation of PR interval until beat dropped
Mobitz II: Proportional conduction usually at a constant ratio and equal (usually normal) PR intervals for conducted beats
Third degree ("3°"): Also known as "complete heart block" No relationship between P waves and QRS complexes, each with independent rate

conduction bundles, or in the ventricles themselves. When complete heart block is present and a subsidiary pacemaker takes over the initiation of electrical activity, the rhythm is called an "escape" rhythm, and the location of the subsidiary pacemaker identified, i.e., "nodal escape" or "ventricular escape." Obviously, escape rhythms occur as a consequence of the nature of all cardiac cells to automatically depolarize. The rate at which this automatic depolarization occurs is fastest in the sino-atrial (SA) node, which therefore acts as the normal pacemaker for the heart. The automatic depolarization rate is slower in the node (about 45–55 beats per minute) and even slower in the ventricles (about 35–45 beats per minute). The rate of the escape rhythm, in addition to the QRS complex duration, can therefore give a clue as to where the escape rhythm originates. If the QRS complex is normal in duration (<0.12 s), the escape focus (point where electrical activity originates) must be in the node or high in the His bundle (before the bifurcation into the right and left bundle branches) with a normal or near normal conduction pattern. If the QRS complex is prolonged (≥0.12 s), the activation focus is either in the ventricles or in the bundle system below the bifurcation of the bundle of His, and the QRS complexes have the configuration of a bundle branch block. In these instances where the QRS is prolonged, a slower rate (35–45) favors a ventricular focus, while a relatively faster rate (45–55) favors a nodal or bundle focus. Occasionally there is what might be called an "iatrogenic escape rhythm," otherwise known as a transvenous pacemaker (Fig. 4.8).

With third-degree AV block, the P waves have no recognizable relationship to the QRS complexes. The PR intervals appear to be widely variable and without a pattern, as distinguished from the gradual prolongation of the varying PR intervals in Mobitz I (Wenckebach). It is usually possible in third-degree AV block to determine both the atrial rate (the frequency of P waves) and the ventricular rate (the frequency of QRS complexes) (Fig. 4.9). Since the electrical activity of the atria and ventricles in complete heart block is unrelated, or "dissociated," it is clear that complete heart block is an example of "AV dissociation," where the atria and ventricles beat at different rates and are totally independent. Yet AV dissociation is a general term and not synonymous with com-

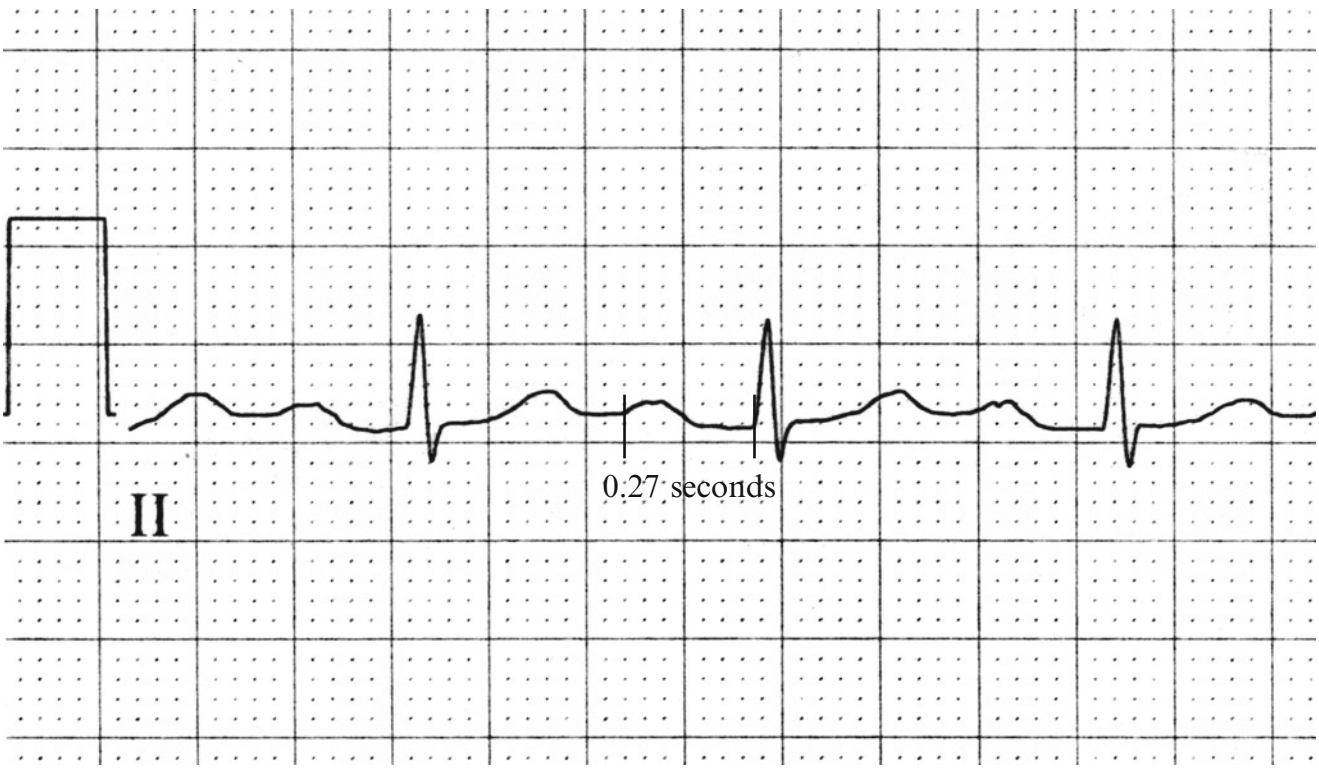


Fig. 4.1 First-degree AV block. The PR interval is 0.27 s in duration

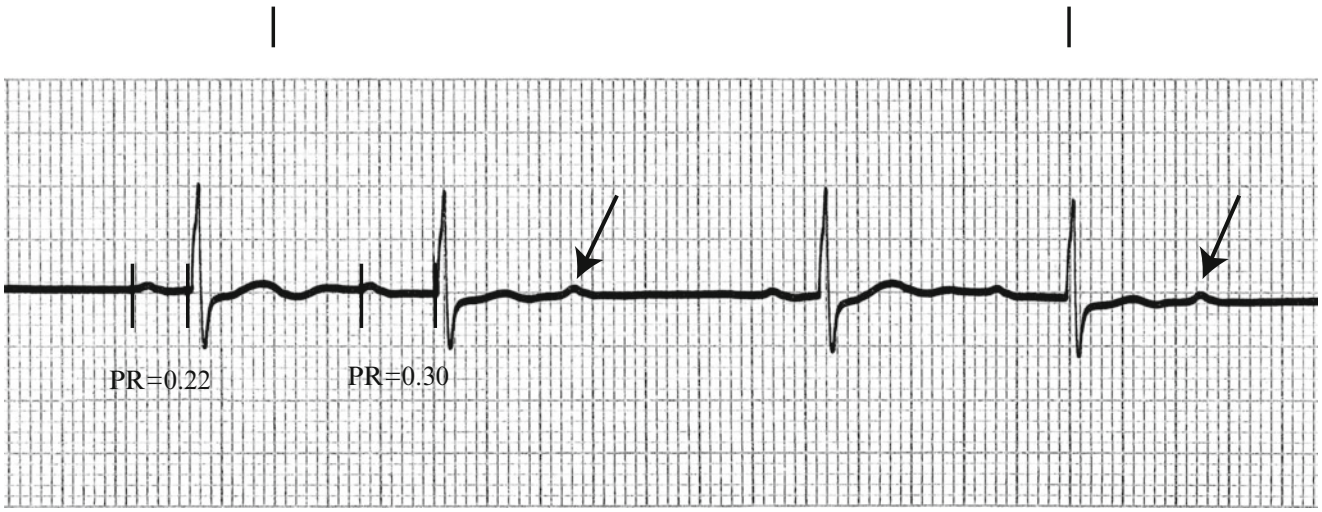


Fig. 4.2 Mobitz I (Wenckebach). The first and fourth P waves are conducted with a PR interval of 0.22 s. The second and fifth P waves are conducted with a PR interval of 0.30 s, and the third and sixth P waves are not conducted. This is a 3:2 block. *Arrows* indicate nonconducted P waves

Fig. 4.3 Mobitz I (Wenckebach). The diagram shows how the R–R interval may decrease while the PR interval gradually increases. The P–P interval is constant at 0.8 s (heart rate of 75 beats per minute). The PR gradually increases until the sixth P wave is not conducted into the ventricles (*arrow*). The RP interval is simply the P–P interval minus the PR interval. The R–R interval is determined by adding the RP and PR intervals of consecutive beats. The first PR interval is 0.16 s, and the second is 0.24 s, or an increment of 0.08 s. The third PR interval is 0.30 s, so the increment of increase in PR between the second and third P wave is 0.06 s. In this example, the increment of increase between consecutive PR intervals is 0.08, 0.06, 0.04, and 0.02 s. The R–R interval decreases, then, because the increment of change in consecutive PR intervals decreases

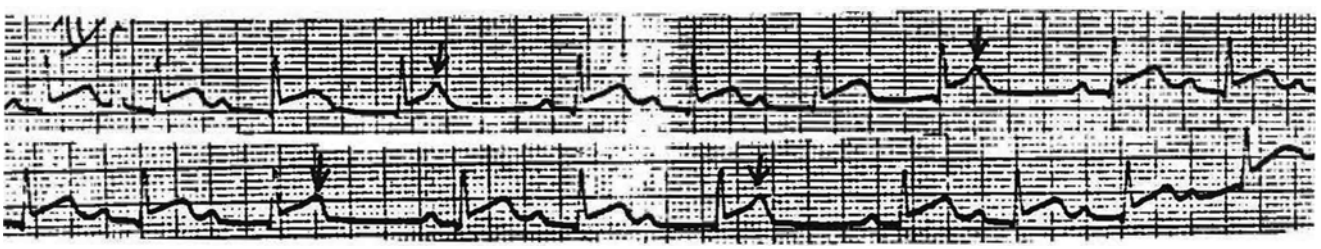


Fig. 4.4 Mobitz I (Wenckebach). The PR interval gradually increases until a beat is dropped. The dropped beats actually fall in the middle of ventricular repolarization, and so they distort the normal configuration of the T waves (*arrows*). In this example, the R–R intervals do not shorten

Fig. 4.5 Mobitz II. In this example the block is 3:1, with three P waves present for each QRS complex. Note that the PR intervals of the conducted beats are constant at 0.18 s

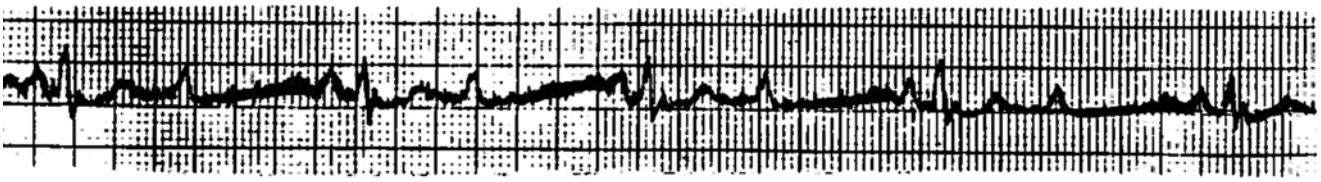


Fig. 4.6 Second-degree AV block with 2:1 conduction ratio. Without other cycles with typical features, this cannot be labelled either Mobitz I or Mobitz II with certainty

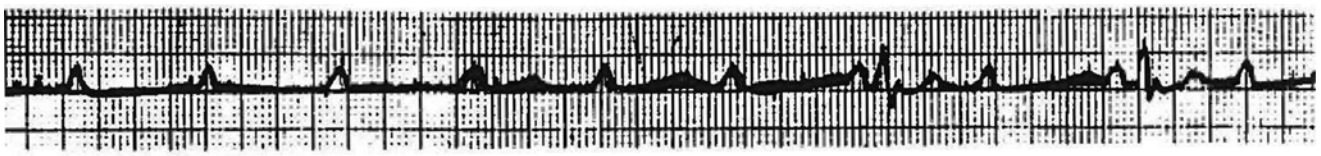


Fig. 4.7 Third-degree AV block (complete heart block). The first seven P waves are not conducted into the ventricles and no escape rhythm is present. Thus, there is a period over 5 seconds without ventricular depolarization. This is followed by the appearance of second-degree AV block (probably Mobitz II) with 2:1 conduction block

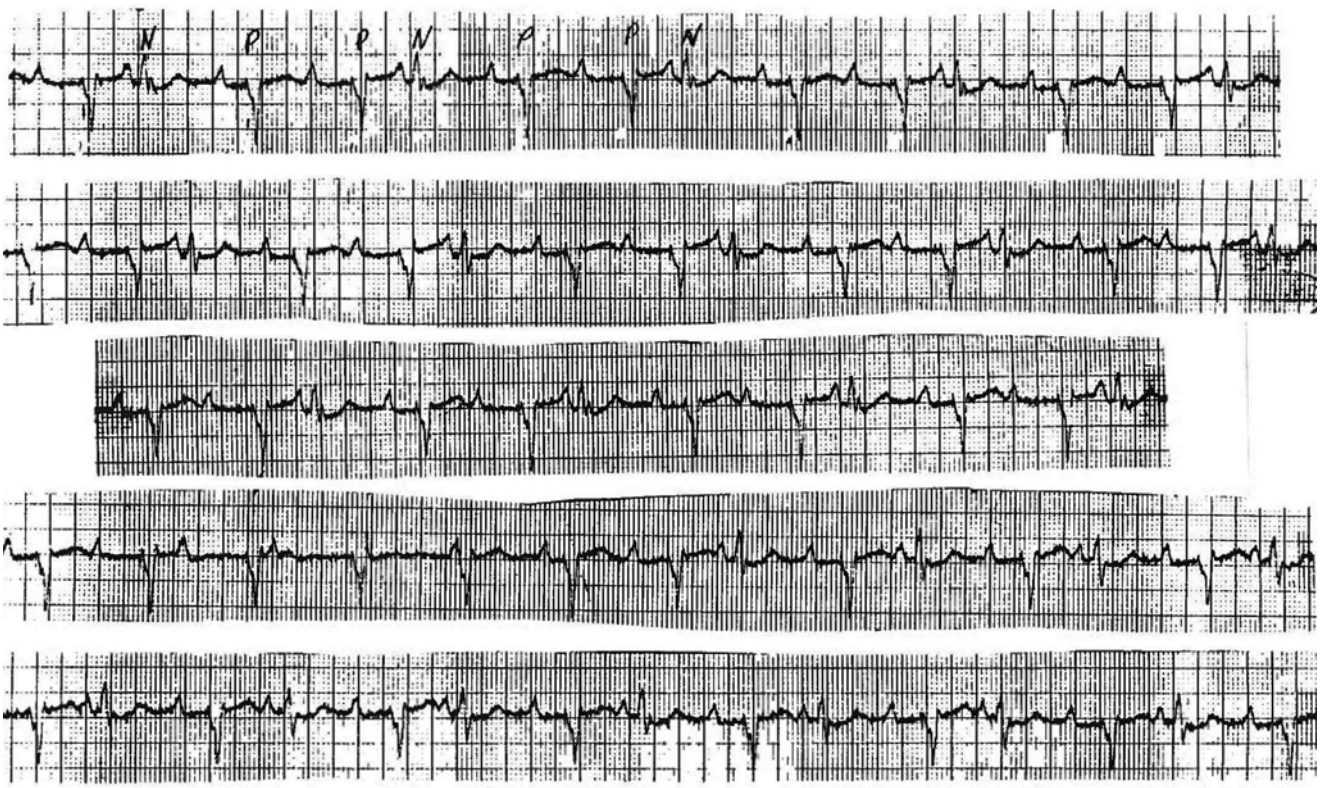


Fig. 4.8 Rhythm strip showing Mobitz II and third-degree AV block in a patient with a transvenous demand pacemaker. In the top panel, the patient's own or "native" QRS complexes are marked with "N," while the pacemaker complexes are marked with "P." If one ignores the pacemaker complexes, it is apparent that the patient is in 3:1 block in the top three panels. In the fourth panel, the patient has no native complexes until after the ninth P wave, so complete heart block is present initially, then is followed by Mobitz II with 2:1 block, which continues through the fifth panel. The pacemaker function is normal, with firing only 0.84 after the native complexes fail to appear, and with no firing when the native beats do appear. Panels 1, 5, and 4 were used with the pacemaker complexes removed for Figs. 4.5, 4.6, and 4.7, respectively, in this chapter

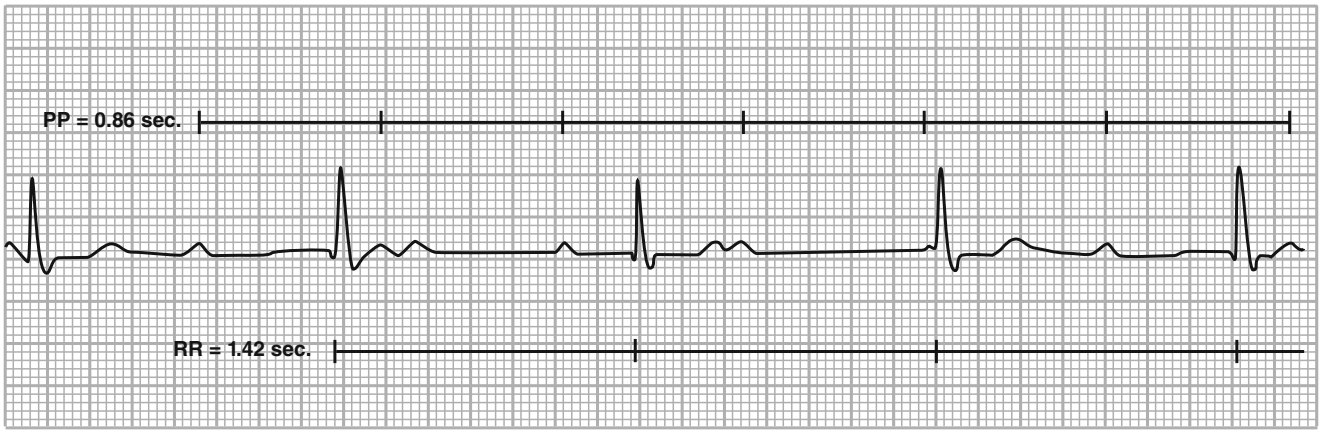


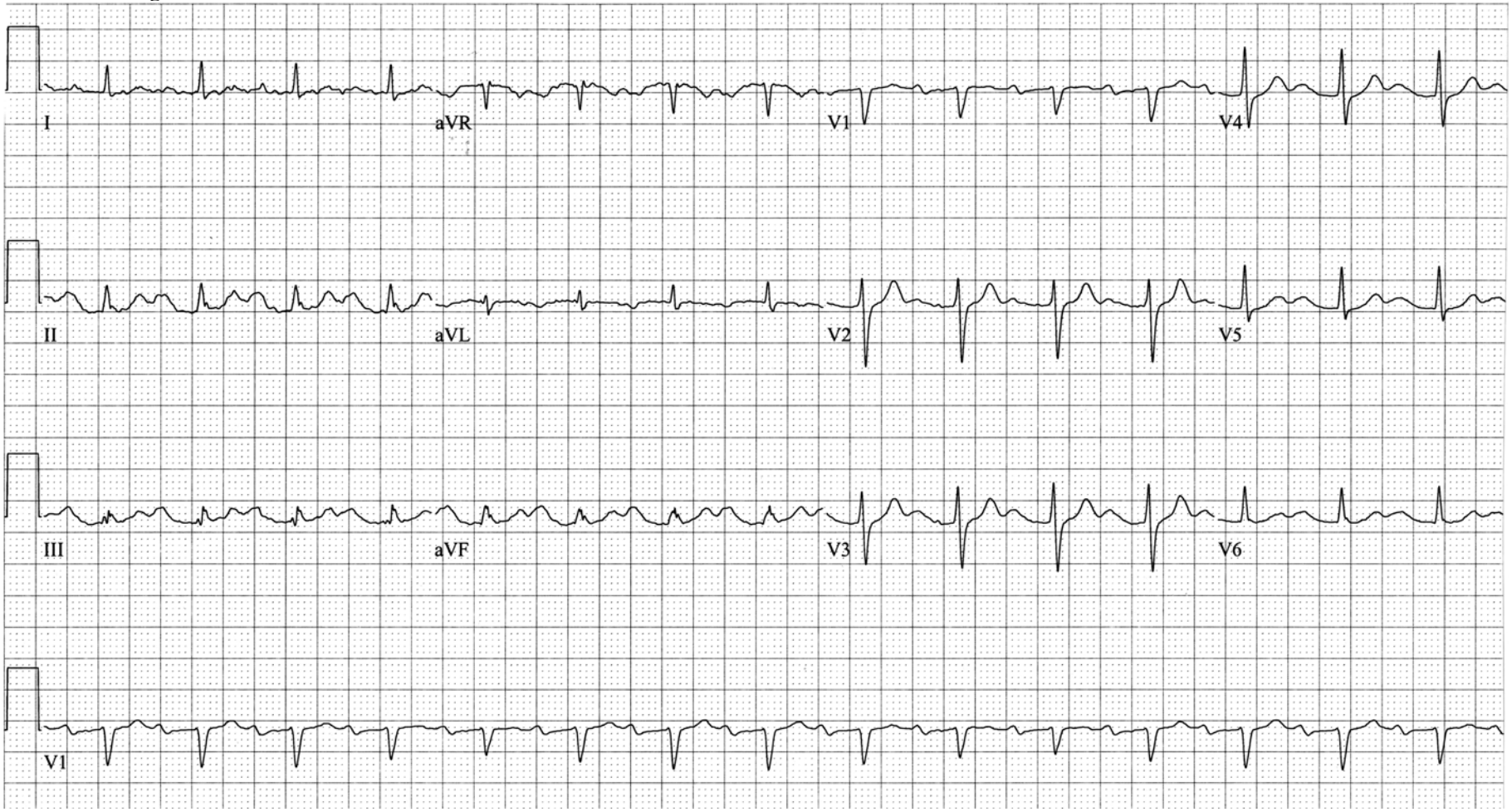
Fig. 4.9 Third-degree AV block. There is complete dissociation of the atria and ventricles. The PR interval appears to vary from as much as 0.88 s to as little as 0.06 s. The P–P intervals and R–R intervals are constant at a rate of 71 and 43 beats per minute, respectively. The QRS complex is about 0.12 s, and along with the rate suggests a ventricular escape rhythm

plete heart block. Ventricular tachycardia is also an example of AV dissociation, where the ventricular and atrial electrical activities are independent.

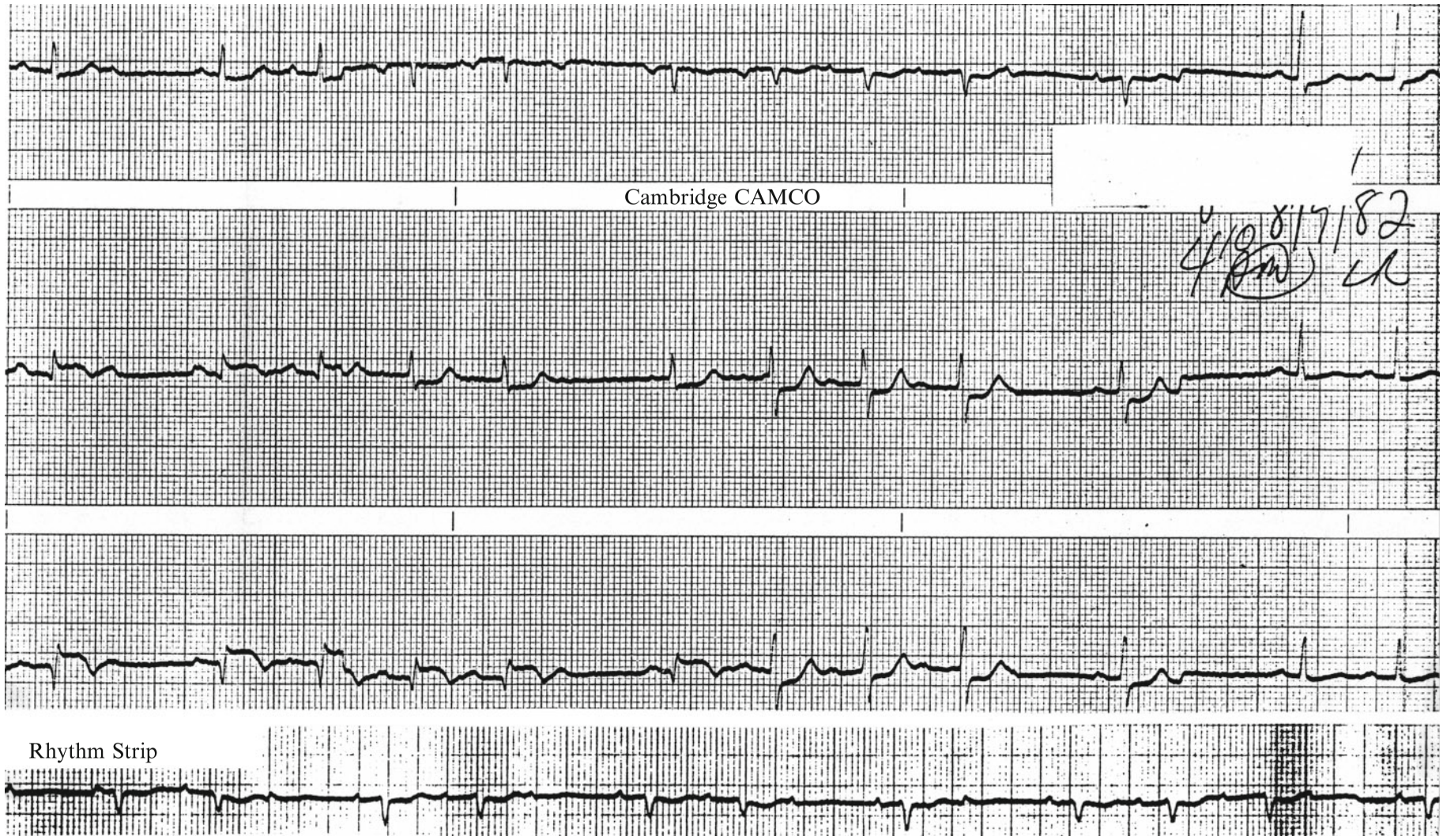
Though called "AV block," the actual site of block for the conduction abnormalities described above is not always the AV node. From the perspective of the 12-lead electrocardiogram, it is impossible to distinguish a conduction delay in the AV node from that in the bundle of His prior to bifurcation into the right and left bundles. Invasive electrophysiological studies have shown that most cases of first-degree AV block and Mobitz I are due to dysfunction in the AV node, while Mobitz II and complete heart block are usually (but not always) due to delay of conduction below the bundle of His rather than through the AV node [3]. Lyme carditis may cause reversible AV block of any degree, but only rarely requires cardiac pacemaker placement [4].

Exercise Tracings

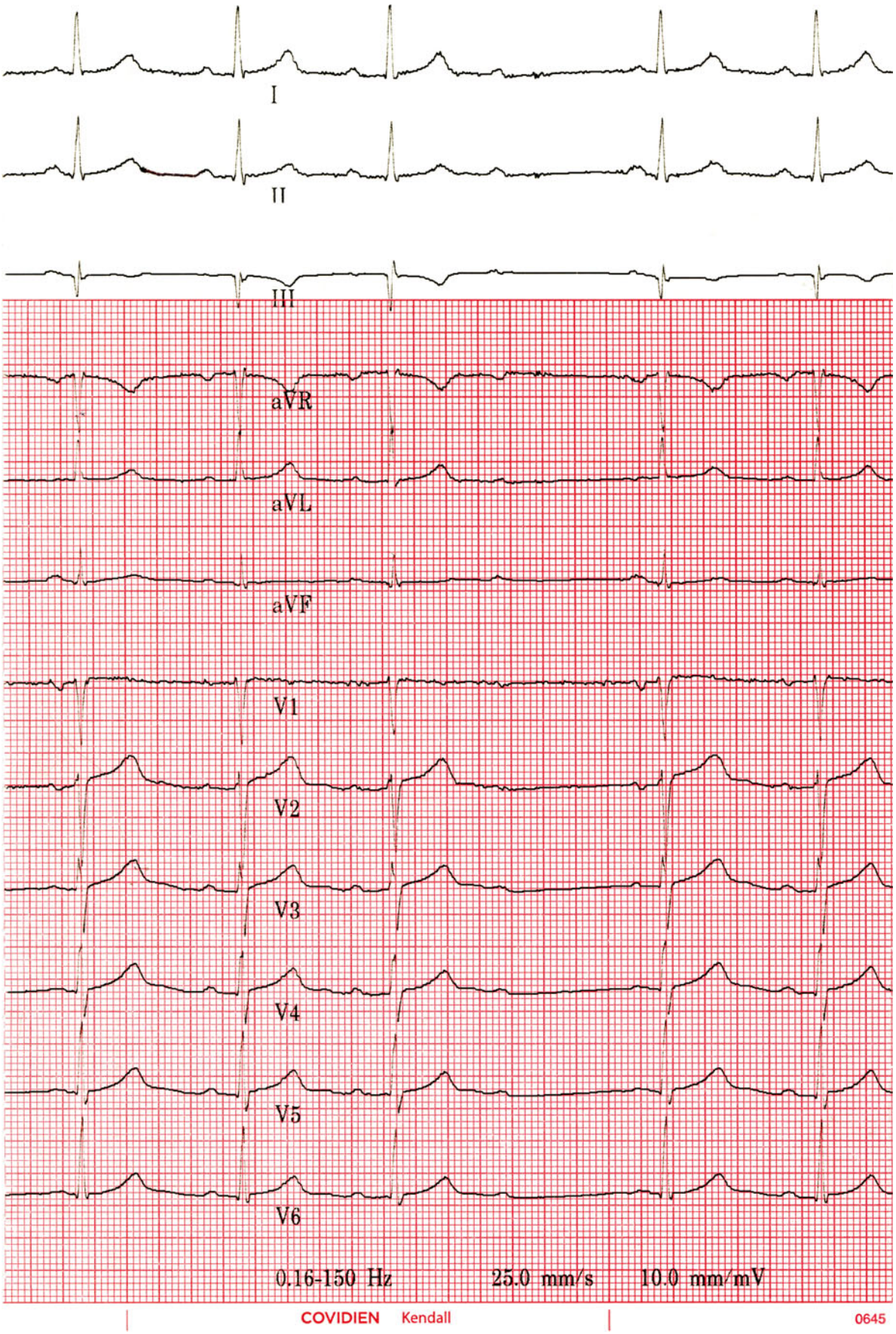
Exercise Tracing 4.1



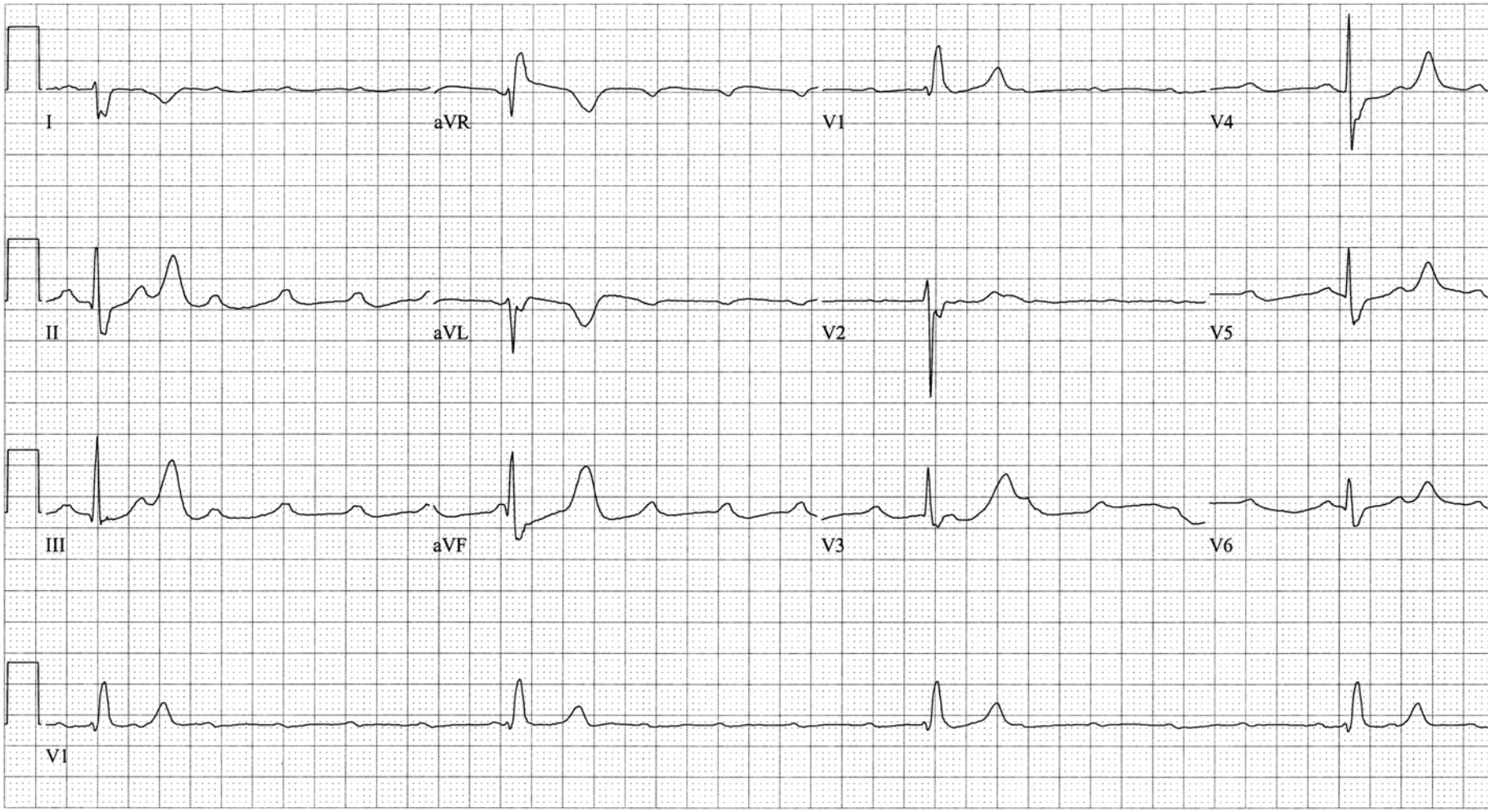
Exercise Tracing 4.2



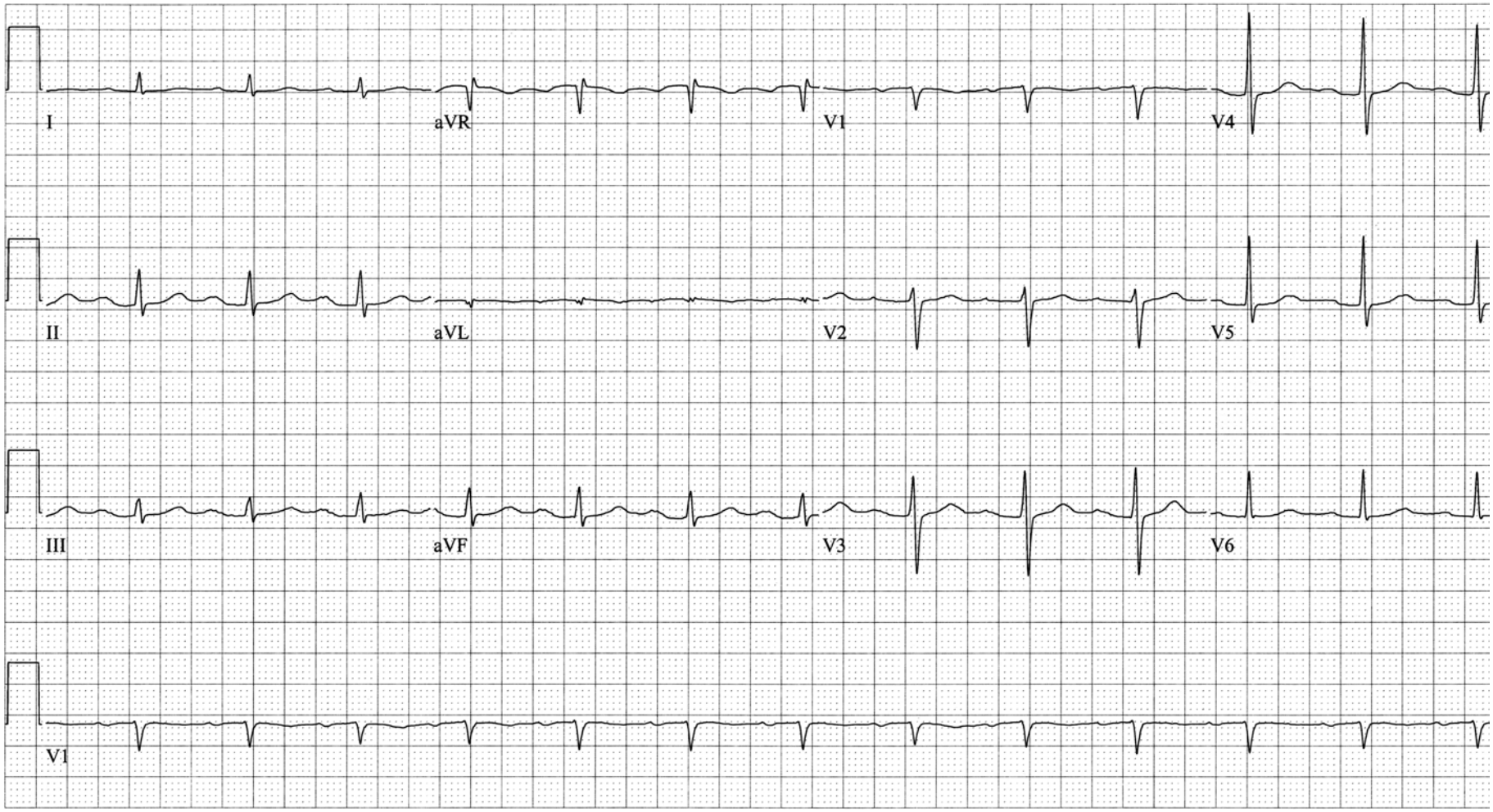
Exercise Tracing 4.3



Exercise Tracing 4.4



Exercise Tracing 4.5



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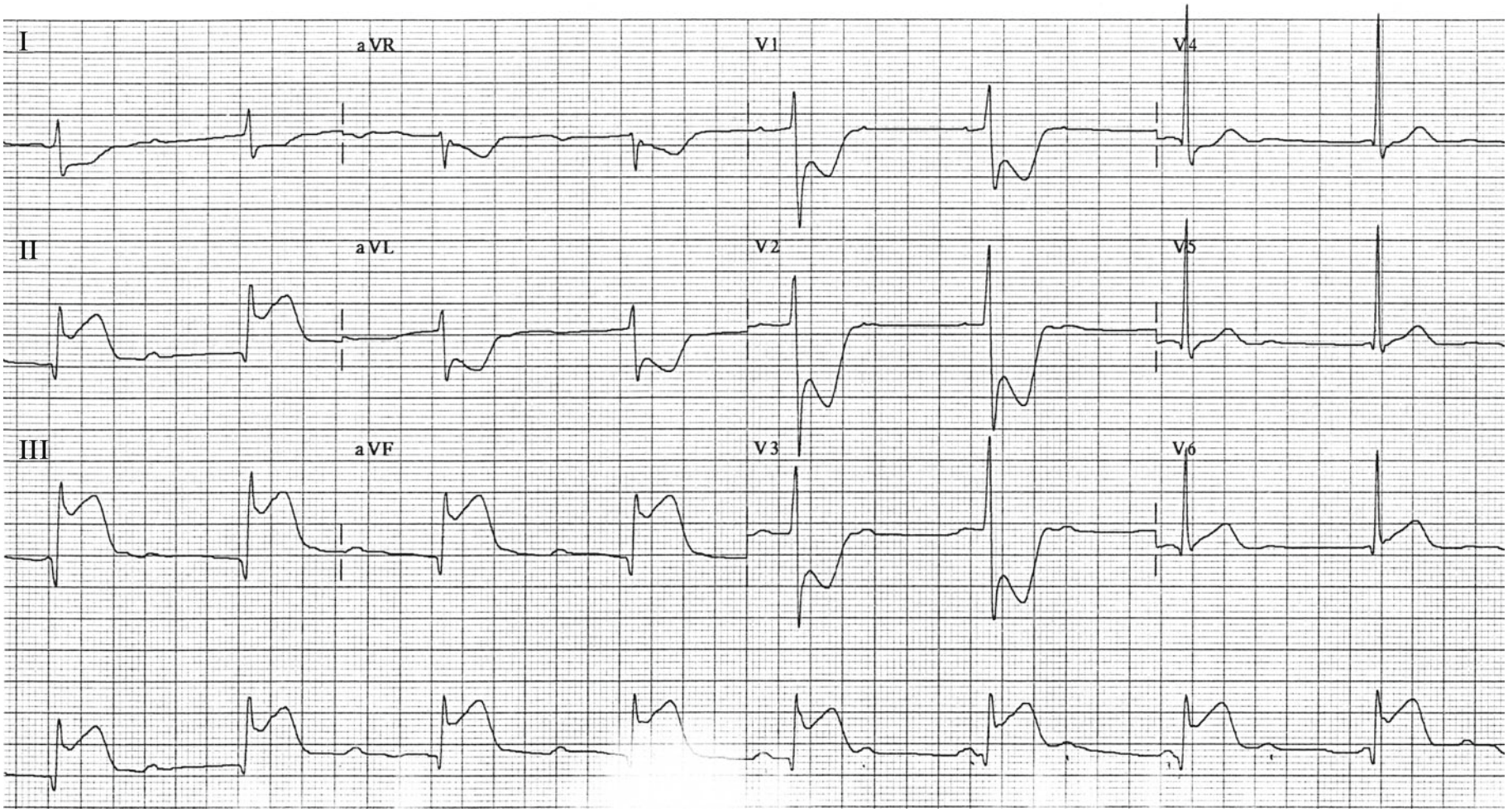
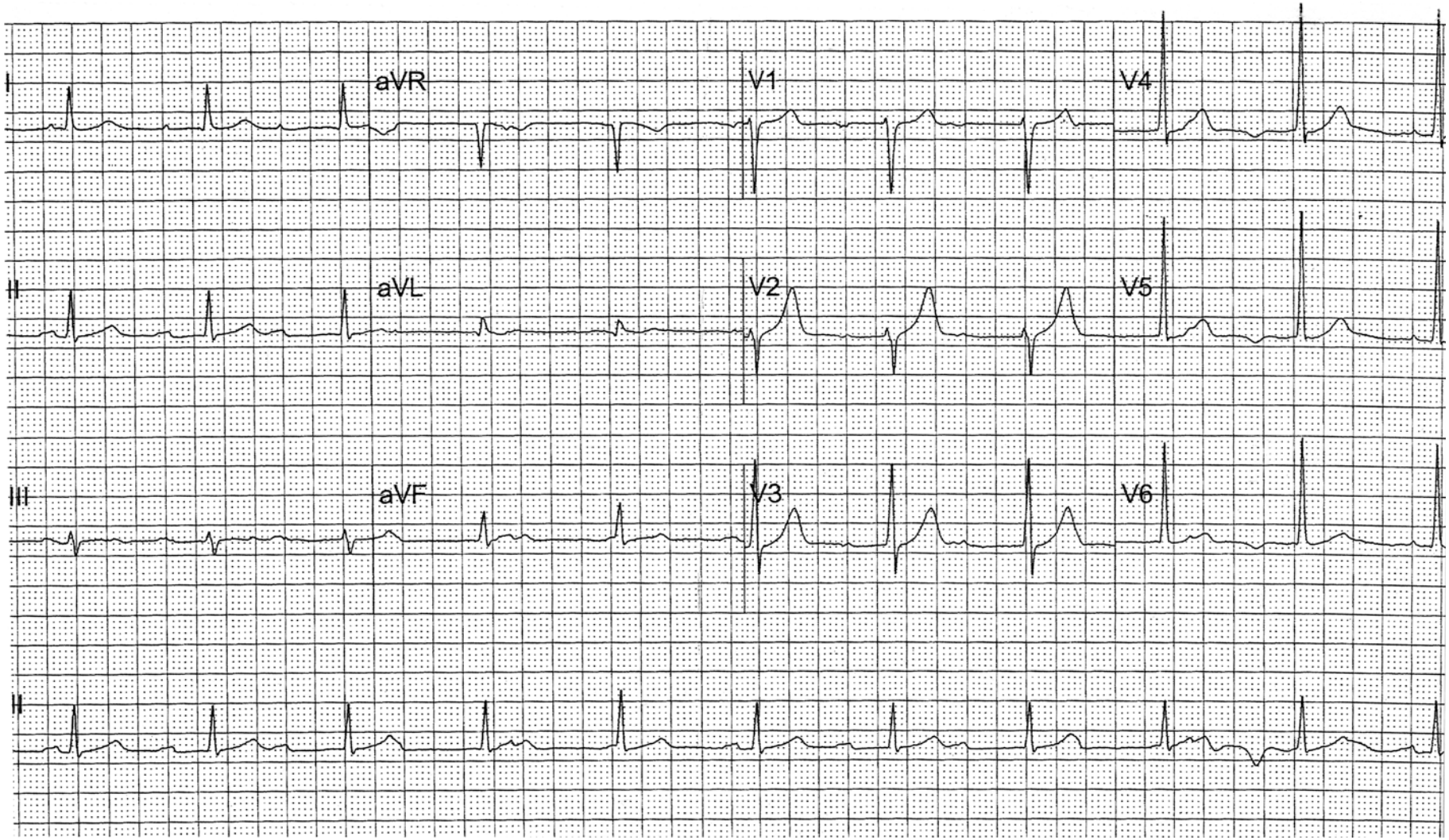


Exercise Tracing 4.7



Interpretations of Exercise Tracings

Exercise Tracing 4.1	
RATE:	A 99 V 99
RHYTHM:	Sinus rhythm with frequent PACs
AXIS:	+65°
INTERVALS:	PR 0.28 QRS 0.10 QT 0.32
WAVEFORM:	Unremarkable
SUMMARY:	Abnormal due to first-degree AV block

Exercise Tracing 4.2	
RATE:	A 102 V 70
RHYTHM:	Sinus rhythm with predominately 3:2 AV block of Wenckebach (Mobitz I) type
AXIS:	+30°
INTERVALS:	PR var. QRS 0.07 QT 0.29
WAVEFORM:	Q waves, ST elevation, and biphasic T waves in inferior leads with minor reciprocal ST depression in anterior leads
SUMMARY:	Abnormal due to recent acute inferior infarction with Mobitz I (Wenckebach) block

Exercise Tracing 4.3	
RATE:	A 66 V 53
RHYTHM:	Normal sinus rhythm
AXIS:	+20°
INTERVALS:	PR var. QRS 0.08 QT 0.44
WAVEFORM:	Q in III, q in II and aVF
SUMMARY:	Abnormal due to Mobitz I, possible old inferior infarction

Exercise Tracing 4.4	
RATE:	A 128 V 22
RHYTHM:	Sinus tachycardia with complete heart block and very slow ventricular escape rhythm
AXIS:	+130°
INTERVALS:	PR --- QRS 0.14 QT 0.64
WAVEFORM:	Broad S waves in I and V_6, rsR' in V_1
SUMMARY:	Abnormal due to sinus tachycardia and complete heart block with ventricular escape

Exercise Tracing 4.5	
RATE:	A 85 V 85
RHYTHM:	Normal sinus rhythm
AXIS:	+65°
INTERVALS:	PR 0.27 QRS 0.07 QT 0.39
WAVEFORM:	Normal
SUMMARY:	Abnormal due to first-degree AV block

Exercise Tracing 4.6	
RATE:	A 94 V 66
RHYTHM:	Sinus rhythm with 2:1 AV block. On the left side of the tracing, some P waves are hidden in the QRS/ST/T
AXIS:	+95°
INTERVALS:	PR --- QRS 0.09 QT 0.40
WAVEFORM:	ST elevation II, III, aVF; ST depression I, aVL, $V_{1–3}$
SUMMARY:	Abnormal due to acute inferoposterior ST elevation MI with reciprocal changes in I and aVL, 2:1 AV block, slight right axis deviation

Exercise Tracing 4.7	
RATE:	A 81 V 66
RHYTHM:	Sinus rhythm with complete heart block and nodal escape rhythm
AXIS:	+25°
INTERVALS:	PR --- QRS 0.08 QT 0.39
WAVEFORM:	Early R wave transition $V_{2–3}$
SUMMARY:	Abnormal due to complete heart block and nodal escape rhythm

References

1. Mymin D, Mathewson FAL, Tate RB, Manfreda J. The natural history of primary first-degree atrioventricular heart block. N Engl J Med. 1986;315:1183–7.
2. Shaw DB, Kekwich CA, Veale D, Gowers J, Whistance T. Survival in second degree atrioventricular block. Br Heart J. 1985;53:587–93.
3. Steiner C, Lau SH, Stein E, Wit AL, Weiss MB, Damato AN, et al. Electrophysiologic documentation of trifascicular block as the common cause of complete heart block. Am J Cardiol. 1971;28:436–41.
4. McAlister HF, Klementowicz PT, Andrews C, Fisher JD, Feld M, Furman S. Lyme carditis: an important cause of reversible heart block. Ann Intern Med. 1989;110:339–45.

Bundle Branch Blocks and Hemiblocks

5

The conduction system of the heart is shown in Fig. 5.1. Under normal circumstances, the electrical activity of the heart arises from the sino-atrial (SA) node, whose intrinsic rate of electrical depolarization is normally faster than any other portion of the heart. The electrical impulse leaves the SA node, spreads across the atria, goes through the atrioventricular (AV) node, and enters the His bundle system at the inferior aspect of the AV node. The bundle of His bifurcates into the right and left bundle branches, and the left bundle branch divides again into two fascicles, the left anterior fascicle and the left posterior fascicle. Thus, by the time the electrical impulse reaches the end of the bundle branch conduction system it is running in three fascicles: (1) the left anterior fascicle, (2) the left posterior fascicle, and (3) the right bundle branch. Either of the bundle branches can have some process which interferes with conduction, thus leading to a bundle branch block, and either fascicle of the left bundle branch can have an impairment to conduction leading to a "hemiblock." It is important to emphasize that hemiblocks and bundle branch blocks may represent simply a *delay* in electrical conduction, rather than a total absence of conduction down the fascicle in question.

Bundle Branch Block

Two criteria must be met to diagnose a bundle branch block: (1) the QRS duration must be abnormally prolonged (0.12 s or greater) and (2) there must be a supraventricular origin of electrical activity. The other common situation in which one observes prolonged QRS complexes is ventricular rhythms; less common causes of prolonged QRS complexes are hyperkalemia and Wolff–Parkinson–White syndrome, so these must be ruled out in order to be sure that the QRS prolongation is from a bundle branch block. A supraventricular rhythm is documented if there are P waves with a consistent PR interval before each QRS complex (sinus rhythm) or if there are other evidences of supraventricular rhythms (see Chap. 7). Only on rare occasions is it difficult to distinguish a ventricular rhythm from a supraventricular rhythm with a bundle branch block.

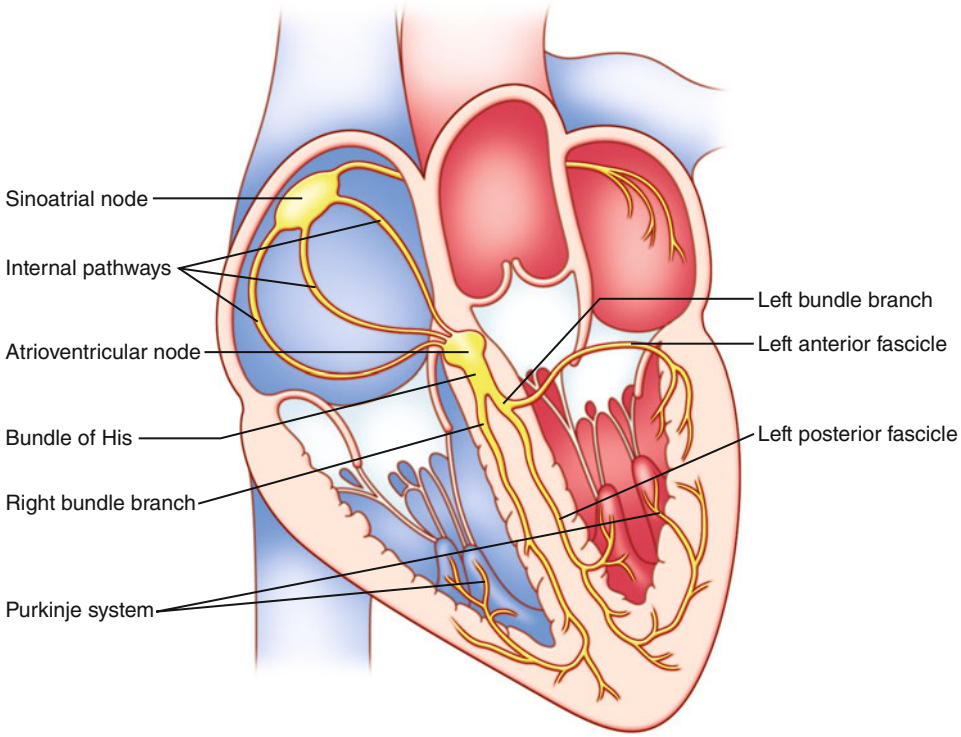
If the QRS complex is wide and there is a supraventricular focus of activation, then the most likely diagnosis is a bundle branch block. The issue then is whether it is a right bundle branch block or a left bundle branch block. The distinction is made by examining the QRS configuration in three leads: I, V_1, and V_6 (Fig. 5.2). With a left bundle branch block, there is a tall, broad R wave in I and V_6 and a QS or rS in lead V_1. With a right bundle branch block, the QRS configuration is markedly different, with a broad terminal S wave in leads I and V_6 and an rsR′ or a tall broad R wave in V_1.

The electrophysiology that creates these patterns may help you remember them. The key element of this electrophysiology is what is called the "terminal forces," or the last part of ventricular depolarization. With a right bundle branch block, the impulses go through the entire conduction system normally until the bifurcation of the bundle of His. At that point, the impulse continues normally (and quickly) down the left bundle, but it is delayed as it tries to traverse the right bundle. When the depolarization is complete on the left side, the wave of depolarization then sweeps towards the undepolarized tissue on the right, and the depolarization down the right bundle that had been delayed also may be able to finally get through. For either or both reasons, the terminal forces, those at the end of ventricular depolarization, are to the right (Fig. 5.3). Because leads I and V_6 have their positive directions to the left, the terminal forces in these leads are negative, leading to the typical "broad, terminal S waves" characteristic of right bundle branch block in these leads.

The changes in lead V_1 are likewise interesting in a patient with right bundle branch block. The normal QRS configuration in V_1 is a small r wave followed by a deep, narrow S wave as shown in (Fig. 5.4). Keeping in mind that the "right" side of the heart is not only to the right of the patient's body but also anterior, the terminal forces with a right bundle block are coming almost directly at V_1. Therefore, the normal rS pattern is altered by the substantial positive forces at the end of ventricular depolarization, causing the classic rsR′ in V_1 (and often in V_2).

B.G. Petty, *Basic Electrocardiography*, DOI 10.1007/978-1-4939-2413-4_5,

Fig. 5.1 Conduction system of the heart



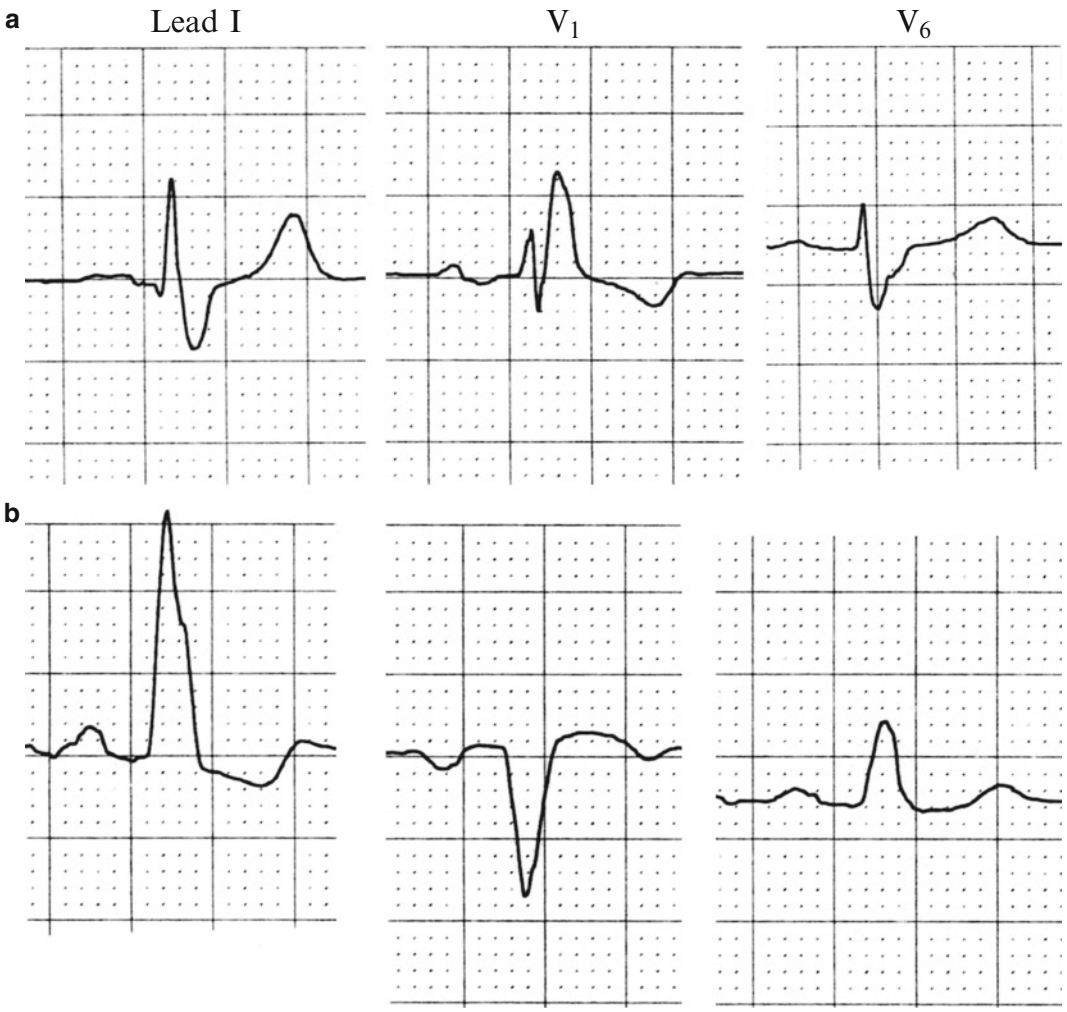


Fig. 5.2 Bundle branch blocks. (**a**) Right. (**b**) Left. For description, see text

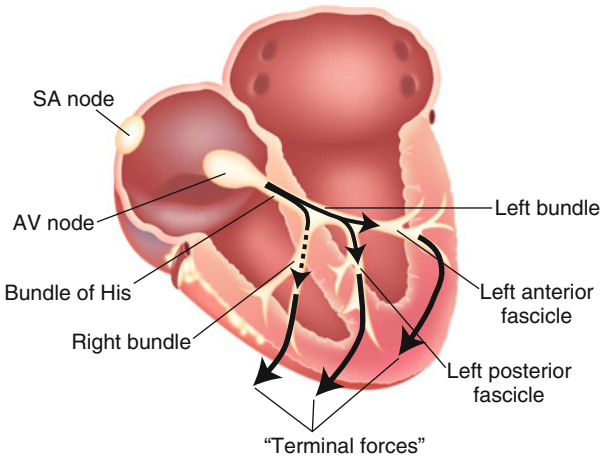


Fig. 5.3 Electrophysiology underlying the QRS configuration in right bundle branch block. The normal wave of depolarization originates in the SA node, goes through the atria, and then through the AV node into the bundle of His. It continues unimpeded down the left bundle branch (*arrow*), but is delayed going down the right bundle branch (*dotted arrow*). When the depolarization finishes through the left side of the heart, it then sweeps to the right side as that tissue is yet undepolarized because of the delay in the right bundle branch. When the delay in the right bundle branch is finally penetrated, the depolarization continues to the right. In both cases, the "terminal forces" (*large arrows*), which represent the last part of ventricular depolarization, sweep to the right, leading to the large "terminal S waves" in leads 1 and V_6 and the R′ in V_1

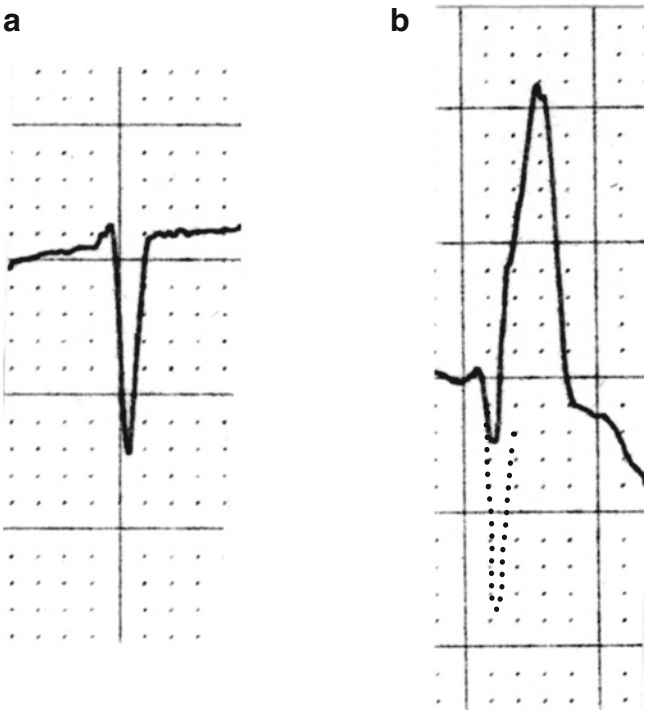


Fig. 5.4 QRS configuration in V_1. (**a**) Normal, with rS. (**b**) With right bundle branch block, where terminal forces anteriorly interrupt S wave and cause tall R′. Dotted lines indicate S wave appearance without interruption by terminal forces

Sometimes the terminal forces totally obscure the normal S wave and one may see just a tall, broad R wave in V_1, which is just as good as an rsR′ in suggesting right bundle branch block. 'Incomplete right bundle branch block' is when the QRS duration is normal and there is a small r' in V_1; this finding is of little clinical consequence except that patients with incomplete right bundle branch block have a greater chance of developing complete right bundle branch block than other patients.

The electrophysiology of a left bundle branch block is, as one would expect, somewhat "opposite" of what is found with a right bundle branch block. The initial ventricular depolarization goes quickly down the unimpaired right bundle, with the terminal forces sweeping to the left. Because the left ventricle is so much greater in thickness and muscle mass than the right ventricle, almost all of the QRS complexes reflect the terminal forces. Therefore, one sees a tall, broad R wave in I and V_6 (as the terminal forces sweep towards the positive sides of these leads), and a QS or rS in V_1 (as the terminal forces sweep directly away from the lead). Especially in the QRS configurations in V_1, one can easily see the opposite appearance of the right vs. left bundle branch block.

The ST segment and T waves are affected by bundle branch blocks. The ST segment is downsloping and the T wave is inverted with left bundle branch block (see Fig. 5.2). The T wave is also opposite in direction in right bundle branch block relative to the predominant, terminal deflection of the QRS, but this generally makes for a fairly normal T wave configuration, i.e., upright T in I, inverted in V_1 and upright in V_6. These ST-T wave changes are secondary to the abnormal conduction pattern of the bundle branch block itself, not ischemia (Chap. 3) or strain (Chap. 6). Secondary ST-T changes do not indicate an additional process beyond the bundle branch.

Hemiblock

When either of the two halves ("hemi-") of the left bundle is not conducting properly, a hemiblock is the result. In contrast to bundle branch blocks, the QRS duration with hemiblocks is normal, i.e., less than 0.12 s. The primary indication of a hemiblock is an abnormal axis deviation of the QRS complex. For left anterior hemiblock, there must be left axis deviation beyond −30° to the left, and for left posterior hemiblock, there must be right axis deviation of +120° or more to the right. After the axis deviation criterion has been met, the limb leads are examined for characteristic QRS configurations. For left anterior hemiblock, a small q wave is present in I and aVL, and a small r wave is found in III. For left posterior hemiblock, the opposite is found, namely a small r in I and aVL and a small q in III (Fig. 5.5).

Bifascicular Block

Bifascicular block means that two of the three fascicles are conducting abnormally. There are three possible combinations for bifascicular block: (1) right bundle branch block and left ante-

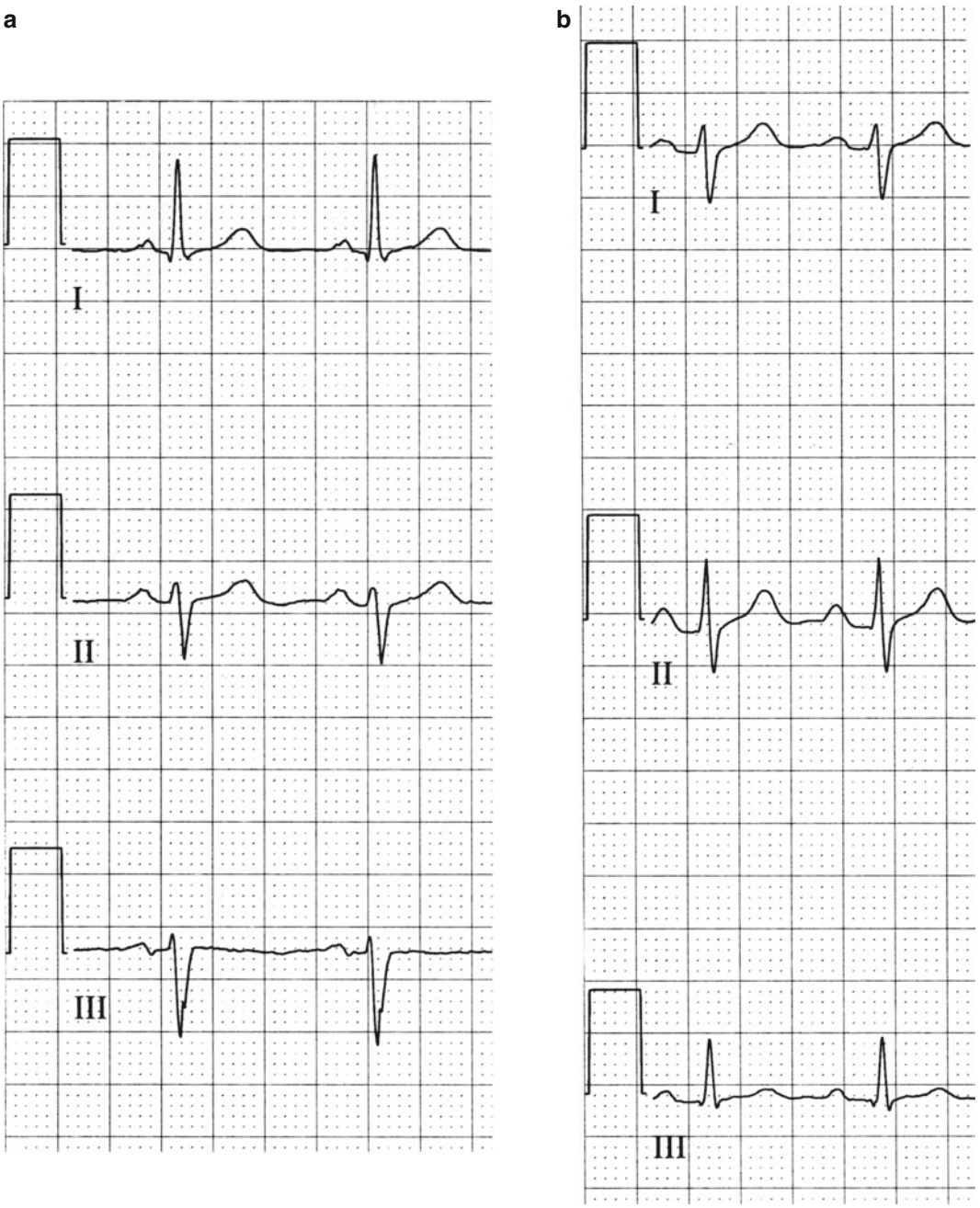


Fig. 5.5 Hemiblocks. (**a**) Left anterior hemiblock. (**b**) Left posterior hemiblock. For description, see text

rior hemiblock, (2) right bundle branch block and left posterior hemiblock, and (3) left bundle branch block. Even though the point of conduction abnormality may be proximal to where the left bundle branch divides into the two fascicles, left bundle branch block constitutes bifascicular block because if conduction down either fascicle were normal there would be at most a hemiblock. In the setting of right bundle branch block, an appropriate axis deviation, with or without associated q's and r's as described above, is adequate to denote block of a second fascicle. In the case of right bundle branch block and sufficient axis deviation, the bundle branch block may obscure the typical q's and r's that would otherwise be seen in the hemiblock.

Trifascicular Block

Sometimes there is a fairly diffuse process that impairs conduction in all three fascicles. If the process is severe, the electrocardiogram (EKG) may show complete heart block, indistinguishable from that which is due to severe, AV node conduction block. In fact, invasive electrophysiological studies ("His bundle studies") show that complete heart block is more often related to trifascicular block than to AV nodal dysfunction. Other manifestations of trifascicular block include alternating left and right bundle branch block (very

rare) and bifascicular block with a prolonged PR interval. As shown in Fig. 5.6, the conduction system below the AV node is responsible for the last portion of the PR interval, so a delay in conduction through those parts of the conduction system could prolong the PR interval. This last manifestation of trifascicular block cannot be distinguished on the regular 12-lead EKG from bifascicular block with a concurrent first-degree AV block. Invasive electrophysiological conduction studies are required to definitively establish which process is involved. It is not unreasonable to assume, however, that if two of the fascicles are conducting abnormally, then the third may be affected as well. If the third fascicle is affected to a lesser degree than the others, the EKG would not show complete heart block but would instead show bifascicular block with a prolonged PR interval, which would reflect the relatively less complete blockage of conduction through the third fascicle.

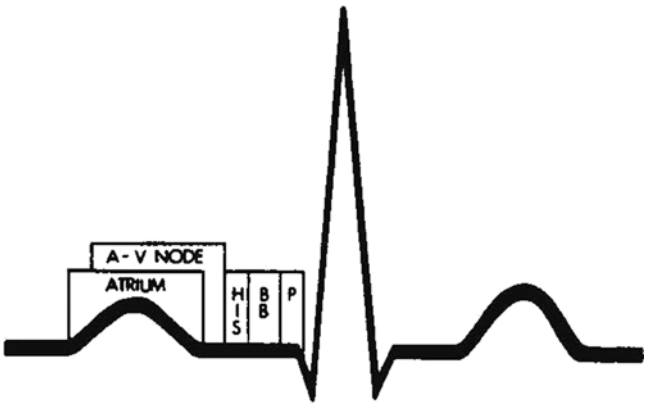


Fig. 5.6 Components of the PR interval. This diagram indicates the multiple portions of the heart which contribute to the PR interval, including the bundle of His, the bundle branches ("BB"), and the distal Purkinje fibers ("P")

It may be useful to approach bundle branch blocks and hemiblocks in the fashion outlined in Fig. 5.7. When one has an EKG with prolonged QRS complexes and a supraventricular focus of activation, then a bundle branch block is likely present. If it is a right bundle branch block, one should next examine the QRS axis because if deviated to more than −30° to the left or +120° or more to the right, a hemiblock is also present, and this is a bifascicular block. If the bundle branch block is a left bundle branch block, then by definition a bifascicular block is present. If one has a bifascicular block, the next question is whether a trifascicular block may be present. If the PR interval is prolonged (>0.20 s), then a trifascicular block *may* be present (vs. bifascicular block and first-degree AV block), and this should be mentioned in the interpretation.

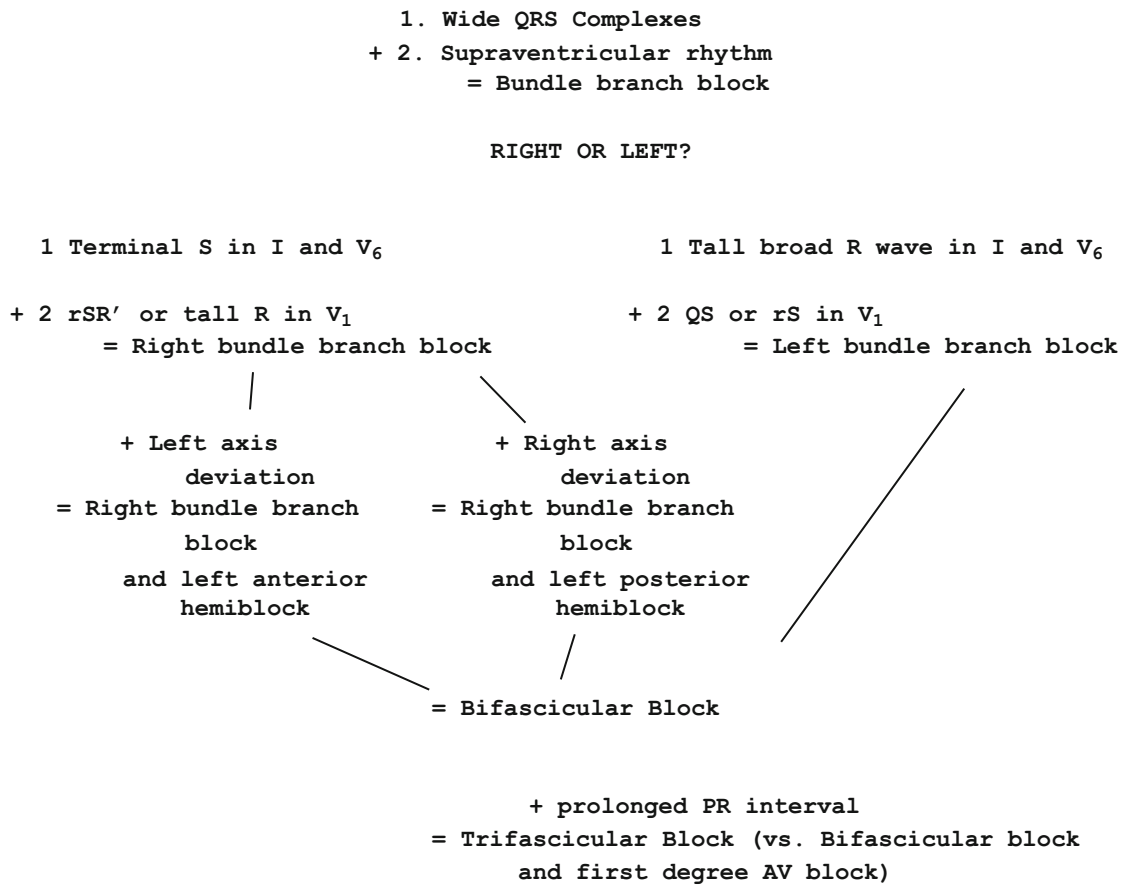
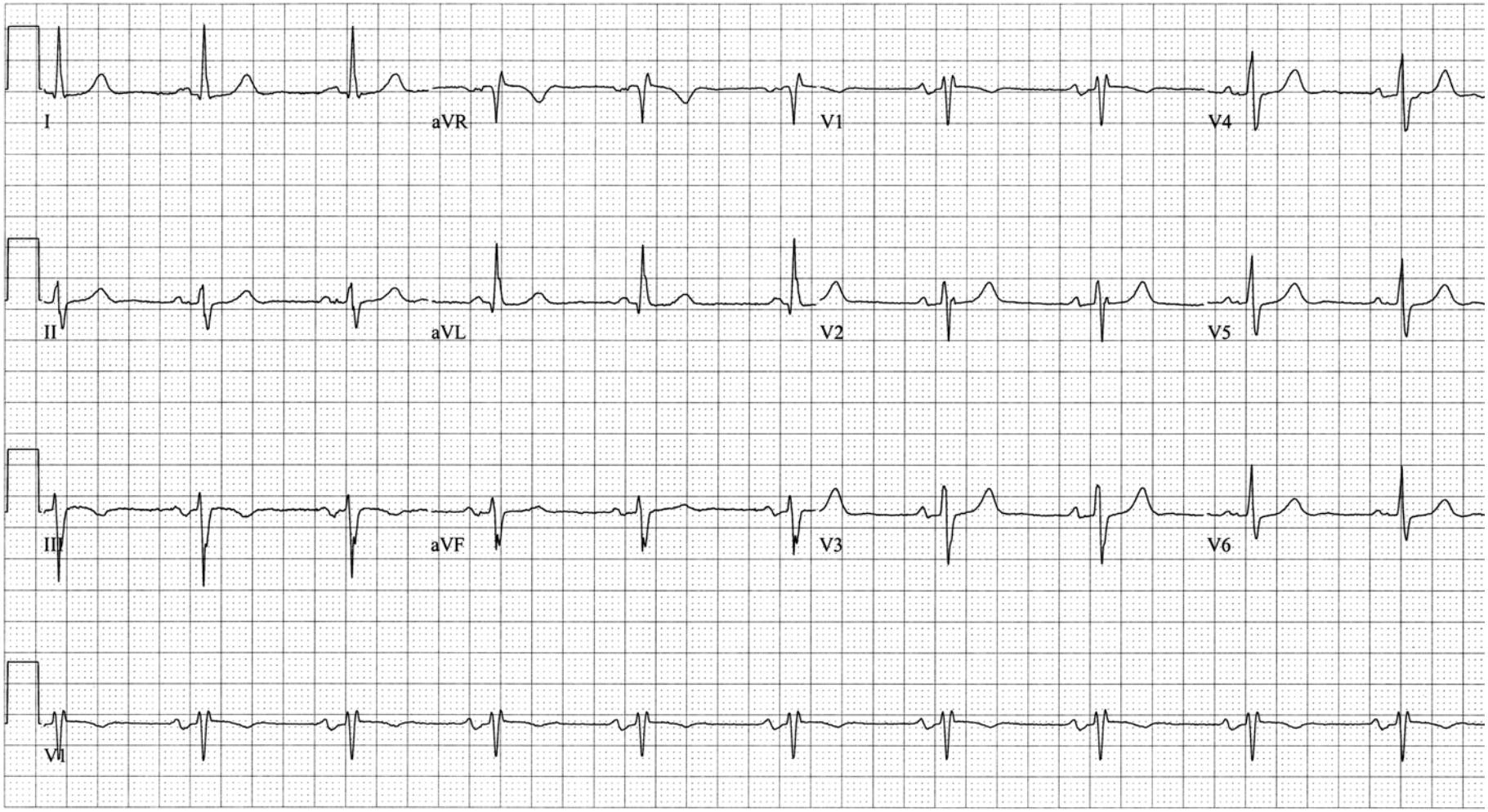


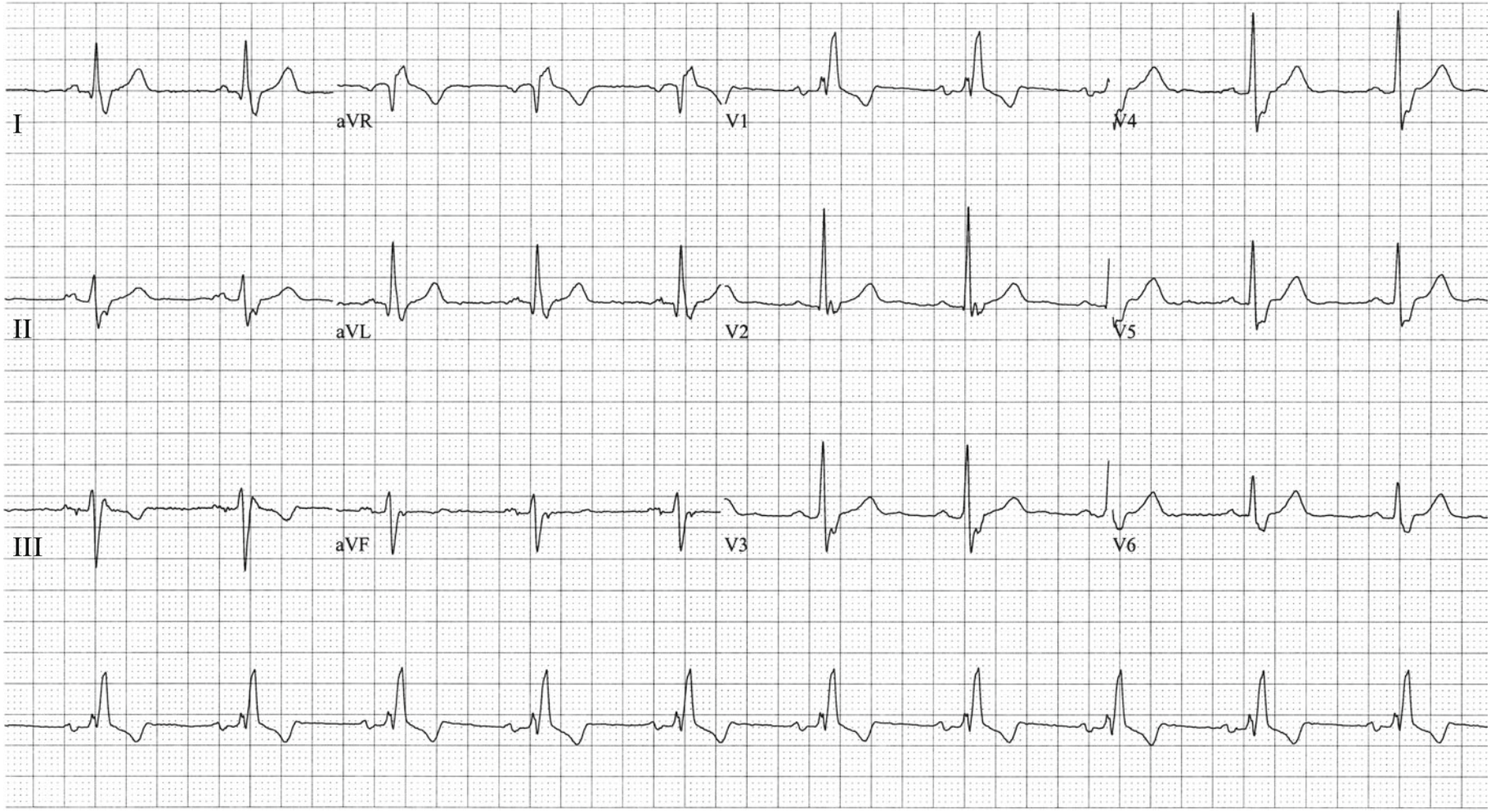
Fig. 5.7 An approach to bundle branch blocks

Exercise Tracings

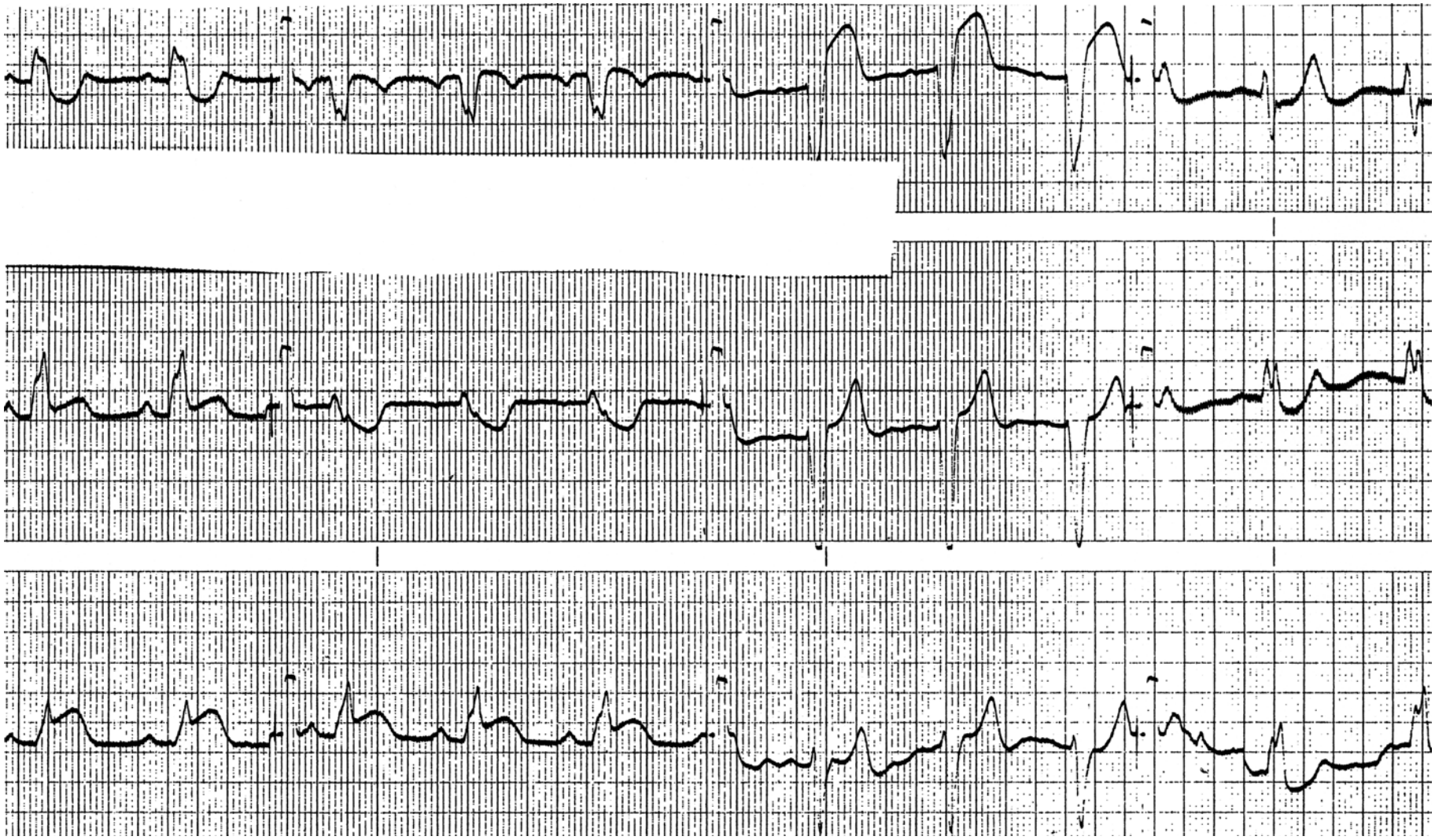
Exercise Tracing 5.1



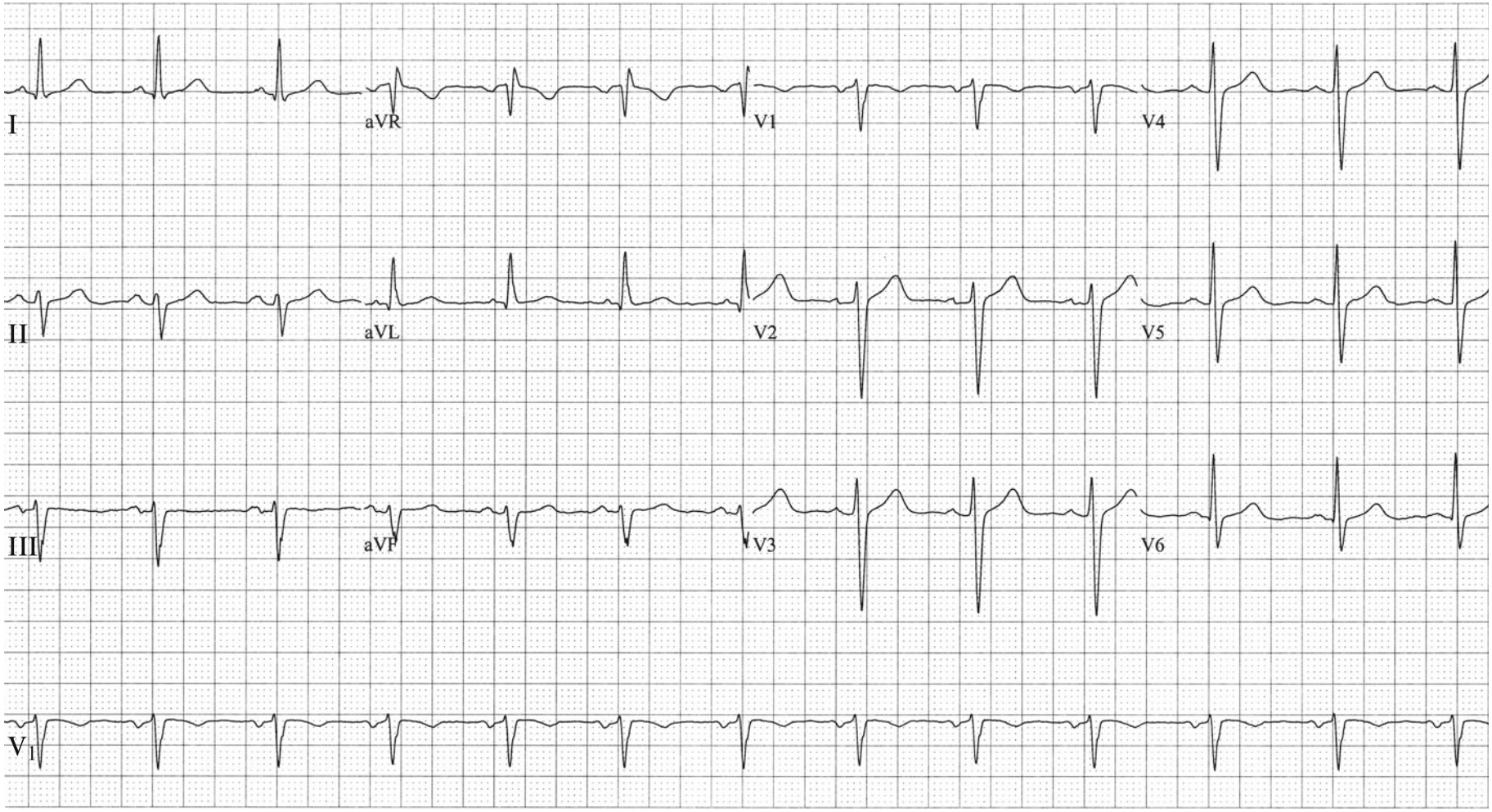
Exercise Tracing 5.2



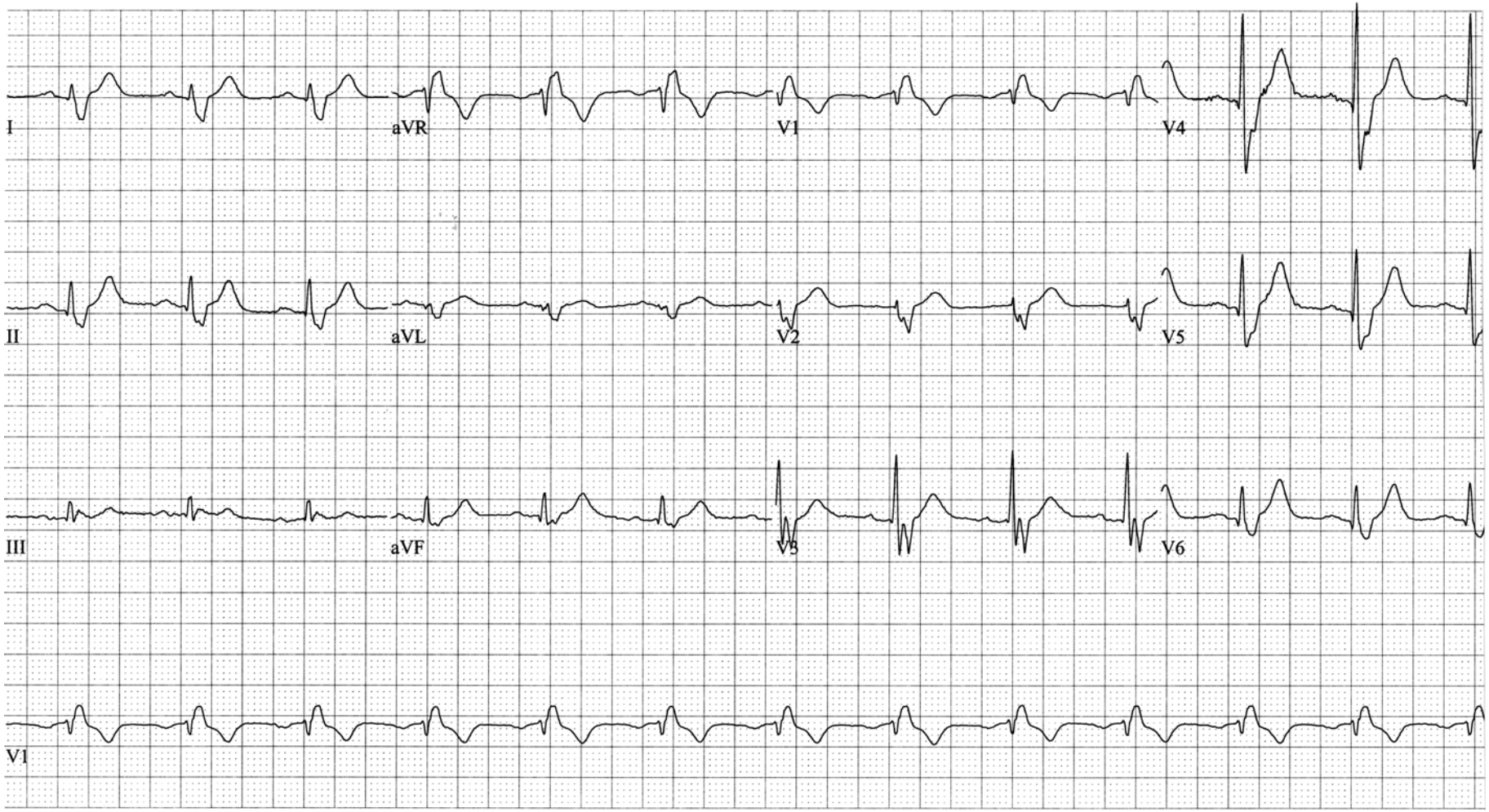
Exercise Tracing 5.3



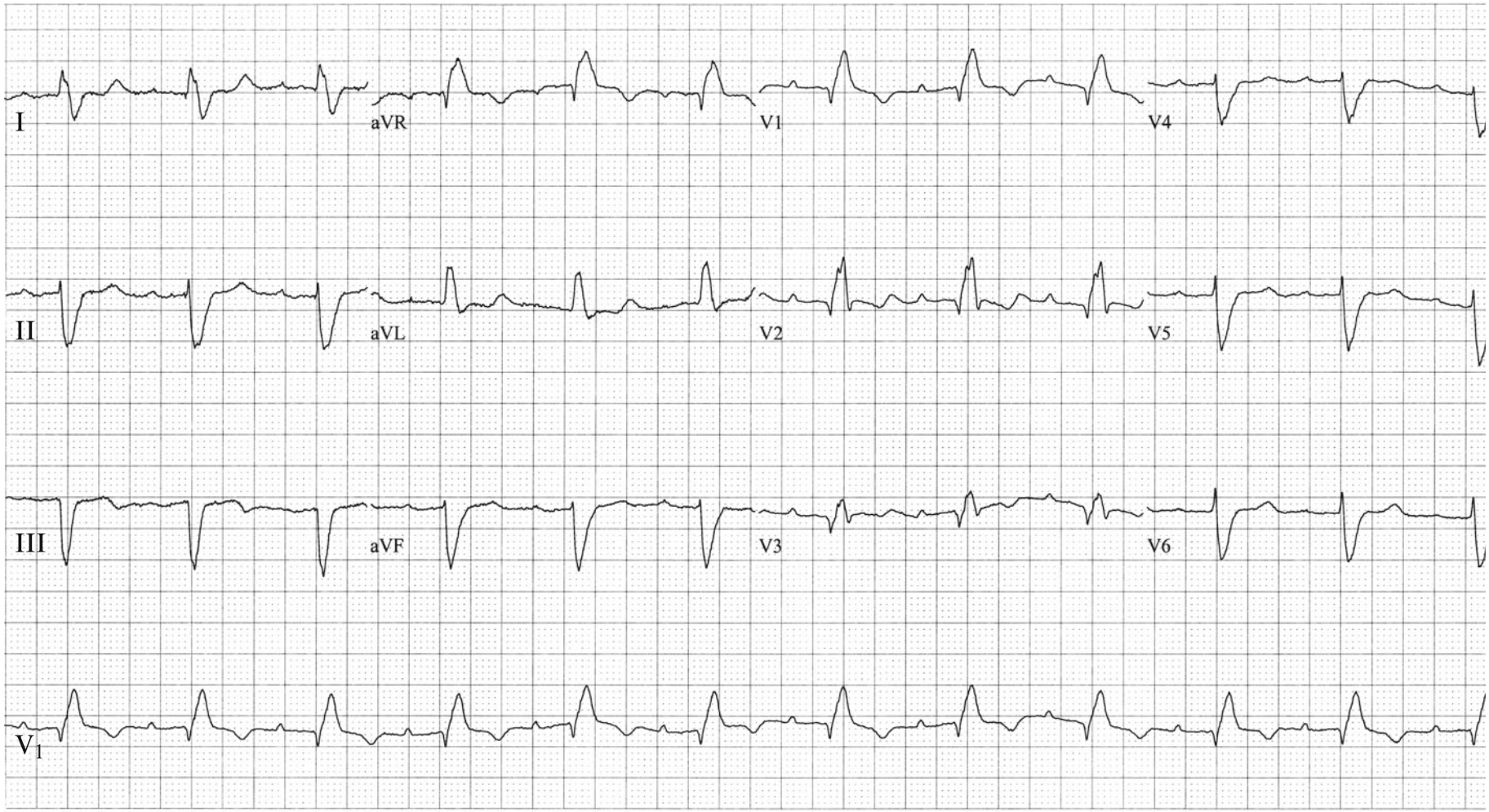
Exercise Tracing 5.4



Exercise Tracing 5.5



Exercise Tracing 5.6



Interpretations of Exercise Tracings

Exercise Tracing 5.1	
RATE:	A 62 V 62
RHYTHM:	Normal sinus rhythm
AXIS:	−40°
INTERVALS:	PR 0.17 QRS 0.10 QT 0.39
WAVEFORM:	q in I and aVL, r in III; rSr′ in V_1
SUMMARY:	Abnormal due to left anterior hemiblock, incomplete right bundle branch block

Exercise Tracing 5.2	
RATE:	A 64 V 64
RHYTHM:	Normal sinus rhythm
AXIS:	−40°
INTERVALS:	PR 0.17 QRS 0.15 QT 0.39
WAVEFORM:	Broad terminal S wave in I and V_6, rsR′ in V_1
SUMMARY:	Abnormal due to right bundle branch block and left anterior hemiblock (bifascicular block)

Exercise Tracing 5.3	
RATE:	A 70 V 70
RHYTHM:	Normal sinus rhythm
AXIS:	+60°
INTERVALS:	PR 0.22 QRS 0.14 QT 0.43
WAVEFORM:	Tall, broad R in I and V_6, rS in V_1; ST elevation in II, III, aVF; ST depression in I, aVL, $V_{4–6}$
SUMMARY:	Abnormal due to acute inferior ST elevation myocardial infarction with reciprocal changes in anterolateral leads, left bundle branch block with prolonged PR interval suggesting either trifascicular block or concurrent first-degree AV block

Exercise Tracing 5.4	
RATE:	A 78 V 78
RHYTHM:	Normal sinus rhythm
AXIS:	−50°
INTERVALS:	PR 0.16 QRS 0.10 QT 0.36
WAVEFORM:	q in I and aVL, r in III; delayed R wave progression $V_{1–5}$
SUMMARY:	Abnormal due to left anterior hemiblock, delayed R wave progression $V_{1–5}$

Exercise Tracing 5.5	
RATE:	A 80 V 80
RHYTHM:	Normal sinus rhythm
AXIS:	+120°
INTERVALS:	PR 0.18 QRS 0.14 QT 0.40
WAVEFORM:	rsR′ in V_1
SUMMARY:	Abnormal due to right bundle branch block and left posterior hemiblock (bifascicular block)

Exercise Tracing 5.6	
RATE:	A *73* V *73*
RHYTHM:	Normal sinus rhythm
AXIS:	−80°
INTERVALS:	PR 0.26 QRS 0.17 QT 0.44
WAVEFORM:	Broad S in I and V_6, tall R in V_1; Q waves in $V_{1–3}$
SUMMARY:	Abnormal due to right bundle branch block, left anterior hemiblock, and prolonged PR interval compatible with trifascicular block; old anteroseptal myocardial infarction

Chamber Enlargement

6

Any of the four chambers of the heart may become enlarged. Unfortunately, the electrocardiogram (EKG) is only slightly useful in detecting chamber enlargement, and that may be a generous characterization. The electrocardiographic criteria often cited to suggest the presence of chamber enlargement are of only limited value and have improved little in the past half century. This chapter is intended primarily to familiarize the reader with the terminology related to EKG associations with chamber enlargement, not to suggest that EKG criteria are reliable in establishing chamber enlargement or hypertrophy.

Left Ventricular Hypertrophy

There are several methods of determining left ventricular hypertrophy (LVH). One that has been used for years is called the Estes system [1]. The Estes system is a point system that assigns certain weight to various features on the EKG (Table 6.1). The point assignments for the Estes system are as follows: (1) increased QRS amplitude, specifically an R or S wave in any limb lead of 20 mm, an S wave in V_1 or V_2 of 30 mm, or an R wave in V_5 or V_6 of 30 mm = 3 points; (2) ST–T changes, specifically ST depression and inverted or biphasic T waves = 1 point if the patient is taking digitalis, 3 points if the patient is not taking digitalis; (3) "left atrial involvement," in which the terminal portion of the P wave in V_1 is greater than 0.04 s in duration and greater than 1 mm deep = 3 points; (4) left axis deviation of at least −30° = 2 points; (5) QRS duration of 0.09 s or more = 1 point; and (6) increased intrinsicoid deflection in V_{5-6}, which means the time between the beginning of the QRS and the peak of the R wave is 0.05 s or more = 1 point. This system gives a potential total of 13 points. Five points or more is considered definite LVH, while four points is "probable" LVH.

Some reflections are reasonable at this juncture. The first and most important criterion for LVH in any system is voltage. In my opinion, if there is not adequate voltage for LVH, one cannot make the EKG diagnosis, regardless of what other criteria may be present, since the nonvoltage findings may be present for many reasons other than LVH. Undoubtedly, requiring that the voltage criterion is always met will lead to some false negative readings of no LVH when it really is present. Nevertheless, insisting on the voltage criterion will provide increased specificity in exchange for less sensitivity. There are a variety of different findings that may satisfy the voltage criterion in addition to those mentioned above. The one that I use most is the amplitude of the R wave in lead V_5 plus the amplitude of the S wave in lead V_1. If the sum of these amplitudes is greater than or equal to 35 mm, that is adequate voltage for LVH. There are other variations in the criteria for voltage. Some authors accept an R wave amplitude of 11 mm or greater in aVL, but I believe that criterion is too dependent upon the QRS axis. The ST–T wave change associated with LVH is called "left ventricular strain" (Fig. 6.1). It looks like ischemia, but it is not acute ischemia because it is a persistent finding (not reversible within minutes) and is unassociated with symptoms of myocardial ischemia. One usually sees strain in leads I, aVL, V_5, and V_6. There may be variable degrees of ST and T wave abnormality, but just about any nonspecific ST–T change associated with voltage qualifies for strain. The reduced score for ST–T changes in the presence of digitalis is appropriate since the drug itself can cause ST segment abnormalities. Some authors object to the term "strain" because it implies a physiological statement related to hemodynamic work [2]. I find it a short, tidy EKG term that substitutes for "nonspecific ST–T changes associated with voltage criteria for LVH."

A prolonged intrinsicoid deflection is intuitively compatible with LVH since the increased left ventricular mass would take longer to completely depolarize. As the volume of the left ventricular wall gets larger, then the intrinsicoid area would get bigger. The QRS duration and the intrinsicoid deflection criteria may not be especially useful because conduction disturbances independent of LVH (but not severe enough to cause bundle branch block) may produce similar changes.

B.G. Petty, *Basic Electrocardiography*, DOI 10.1007/978-1-4939-2413-4_6,

Table 6.1 Estes scoring system for left ventricular hypertrophy

Criterion	Points
Voltage	3 points
"Strain"	3 points (1 point if patient takes digitalis)
Left atrial involvement	3 points
Left axis deviation	2 points
Prolonged QRS	1 point
Increased intrinsicoid	1 point

Right Ventricular Hypertrophy

Right ventricular hypertrophy (RVH) is suggested by right axis deviation (RAD) beyond 90°, early R wave development in $V_{1–3}$, and persisting S waves in $V_{5–6}$. One finds an unusually tall R wave in the early precordial leads and a persisting S wave in the lateral precordial leads, so the QRS complexes can look nearly the same across the precordium (Fig. 6.2). The R wave in the right precordial leads is large as a reflection of the increased size or thickness of the right ventricle. The persisting S wave in the lateral precordial leads correlates with right ventricular depolarization after the left ventricle is depolarized. So both of these changes reflect the abnormally increased mass of the right ventricle. Some criteria require a certain amplitude for the R wave in V_1, while others emphasize the importance of the relative sizes of R to S waves compared to the normal precordial R wave progression. Regardless of the criteria, none are felt to be especially reliable [2, 3]. Nevertheless, in the presence of RAD and early precordial R wave prominence, one may consider RVH.

Left Atrial Enlargement

Left atrial enlargement has been thought to be manifest by (1) a notched P wave in the inferior leads with 0.04 s or more between the peaks and (2) the P wave in lead I larger than the P wave in lead III. These are the criteria for what is called "P mitrale." This is a fairly rare finding now with the substantial fall in the prevalence of rheumatic valvular disease, particularly mitral stenosis, yet it may be a more specific sign than others associated with left atrial enlargement. A more common electrocardiographic finding, perhaps so common that it is of question whether it is very specific for left atrial enlargement, is what is called "left atrial abnormality." Instead of having the normal biphasic P wave in V_1 and an upright P in V_2, left atrial abnormality is characterized by a predominately negative P wave in V_1 and a biphasic P wave in V_2 (Fig. 6.3). There is evidence that the area of the negative deflection of the P wave in V_1 is proportional to the left ventricular end diastolic pressure (LVEDP). In a patient with congestive heart failure, left atrial abnormality may resolve as the LVEDP falls with proper treatment. On the other hand, a patient with mitral valvular disease may have left atrial abnormality, and it may not change at all despite drug treatment because the LVEDP may not fall significantly without surgical intervention. Left atrial abnormality is seen so commonly, perhaps because of improper lead placement, that one needs to be a bit circumspect about what it really means. In patients with heart failure and sometimes in patients with hypertension, it may be a manifestation of left atrial strain. Finally, the duration of the P wave in leads I, II, or III >0.11 is a third criterion applied to left atrial enlargement. None of the three criteria for left atrial enlargement have excellent positive or negative predictive value [4, 5].

Right Atrial Enlargement

Right atrial enlargement has typically been associated with P wave changes called "P pulmonale," in which tall (3 mm or more), peaked P waves are present in the inferior leads. The term "P pulmonale" implies pulmonary hypertension and right atrial enlargement in response to increased right ventricular pressures. The reliability of this feature, however, in predicting right atrial enlargement as documented by echocardiography has been seriously questioned. One group reported that only 2 of 11 patients with the EKG findings of P pulmonale really had an enlarged right atrium. This group found that a qR pattern in V_1 in the absence of clinical evidence of coronary artery disease was the most reliable of several QRS complex configurations in predicting the presence of right atrial enlargement [6]. But the QRS complex (including rightward deviation of the QRS axis) indicates only the ventricular abnormalities that may be indirectly associated with right atrial enlargement instead of a direct reflection of right atrial electrical forces. It may be that no P wave changes are accurate enough to justify their use in the diagnosis of right atrial enlargement [5].

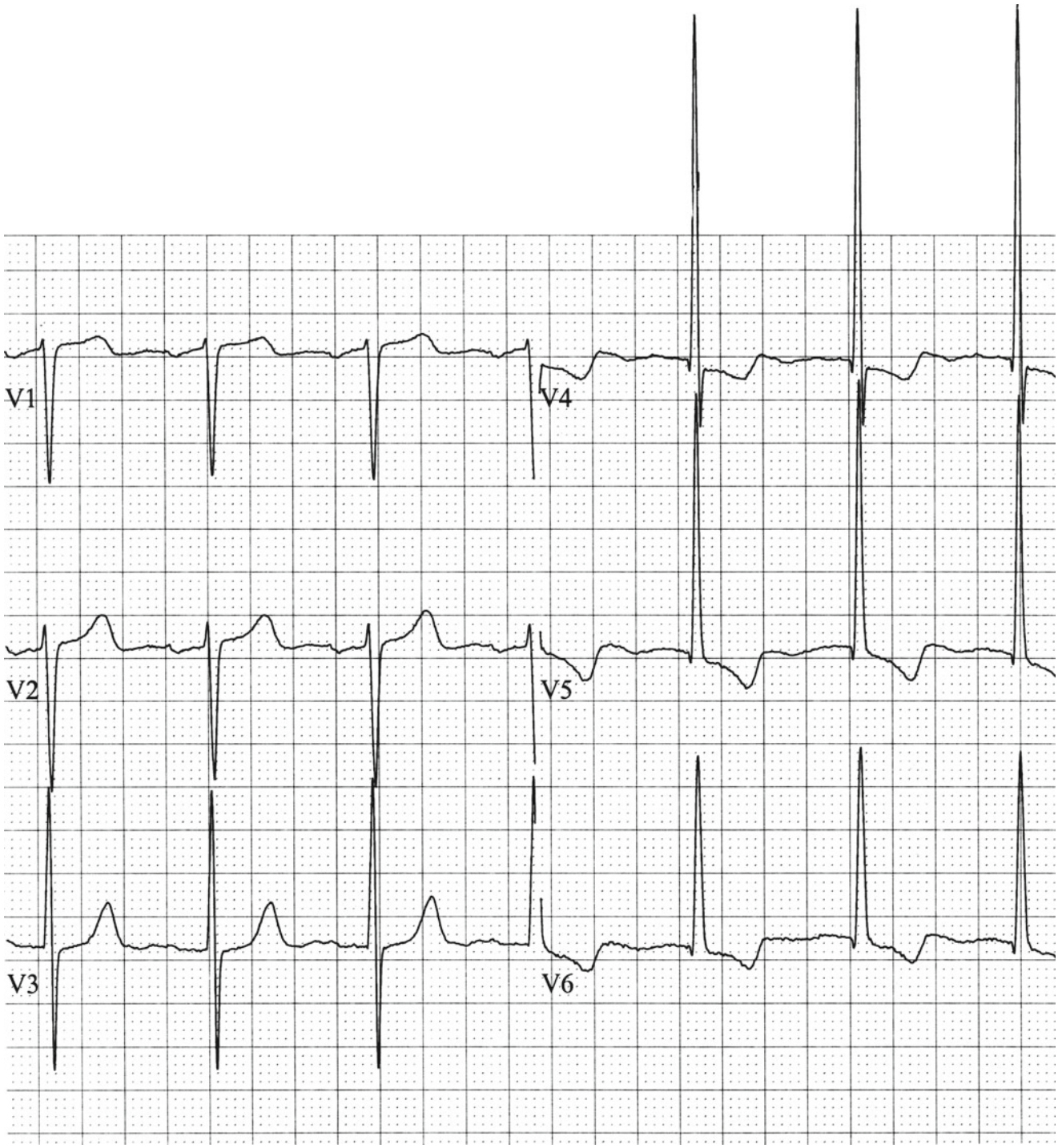


Fig. 6.1 Left ventricular hypertrophy with strain. The R wave in V_5 is 30–31 mm, the S wave in V_1 is 15 mm, and ST depression and T wave inversion are present in V_4–V_6

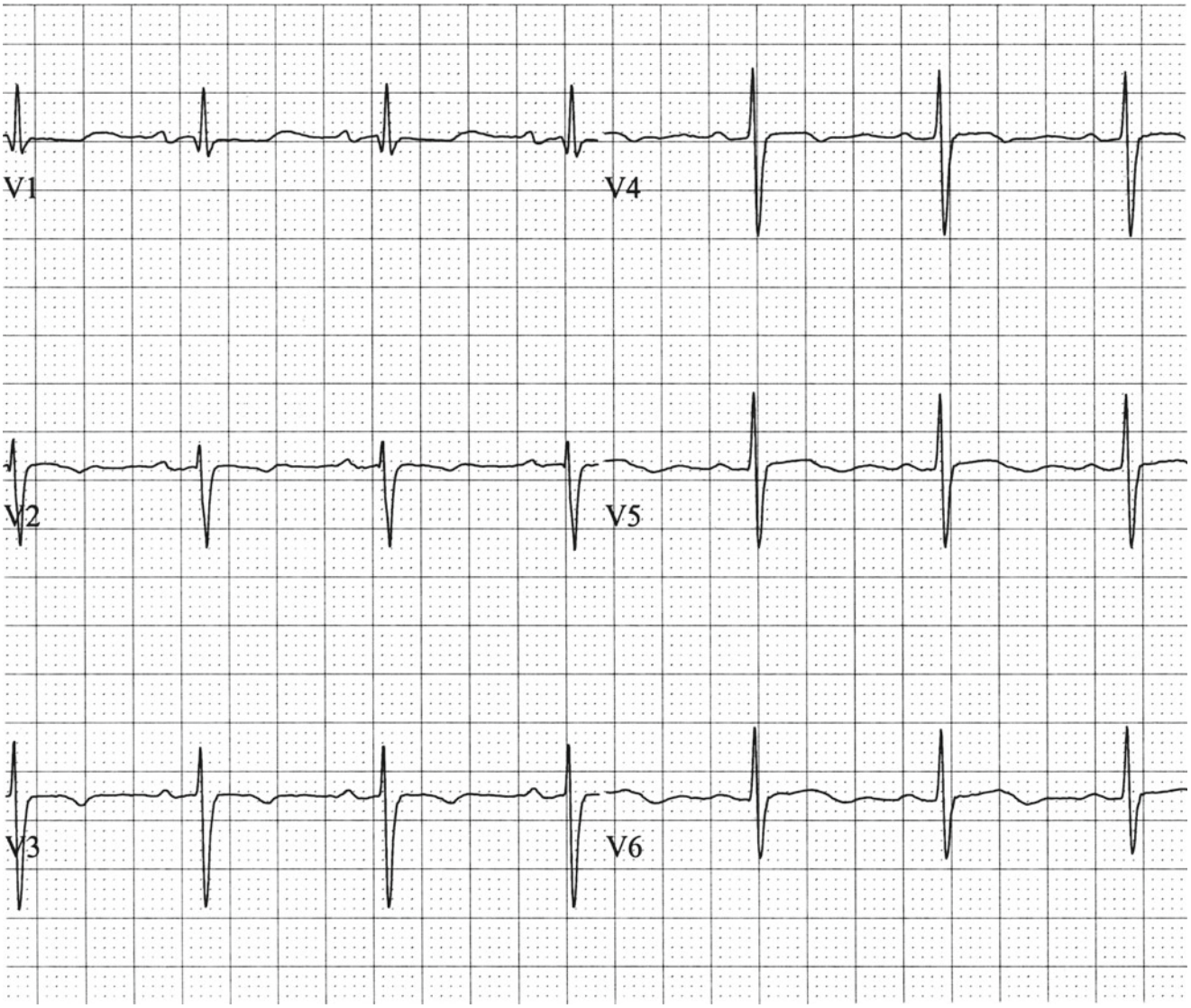
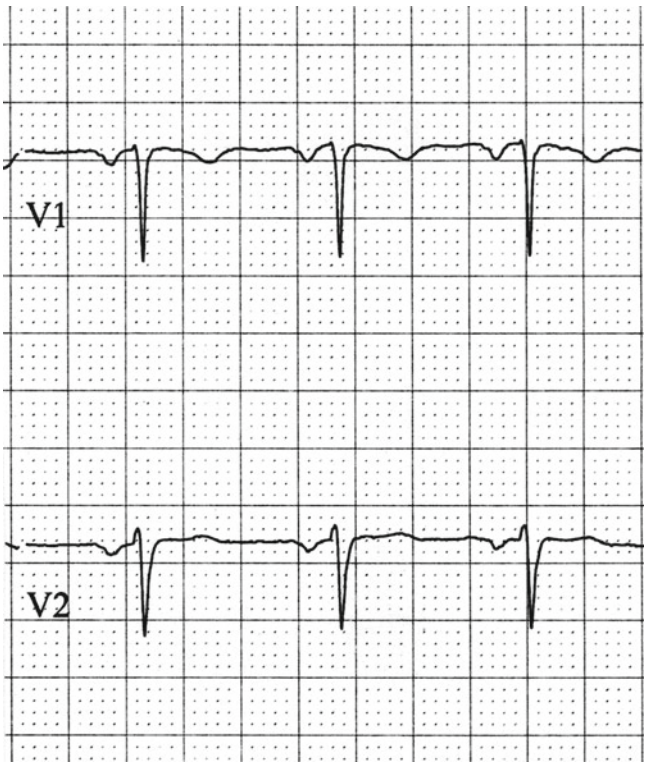


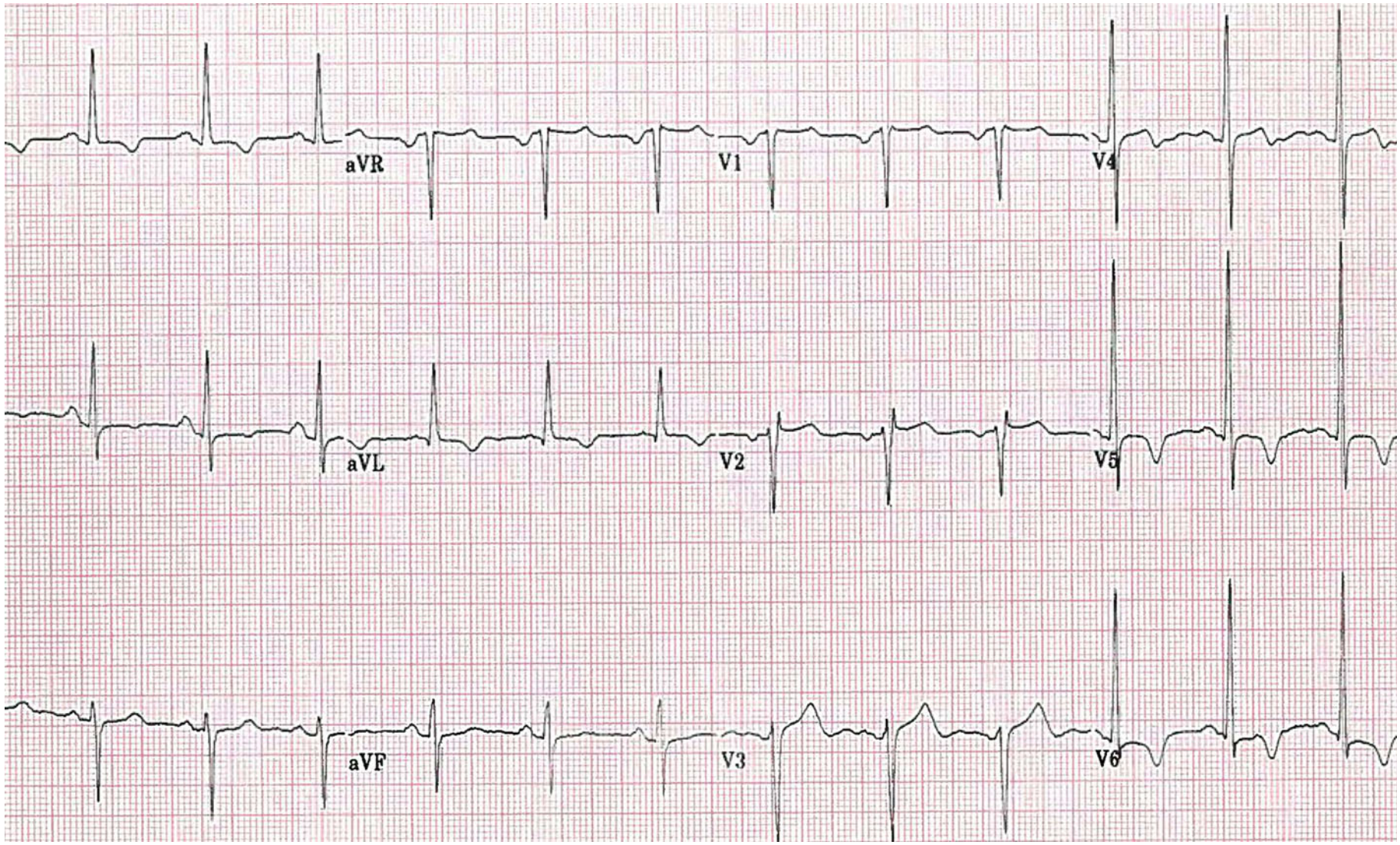
Fig. 6.2 Right ventricular hypertrophy. Note the tall R wave in V_1 and the persisting S waves through the lateral precordium

Fig. 6.3 Left atrial abnormality

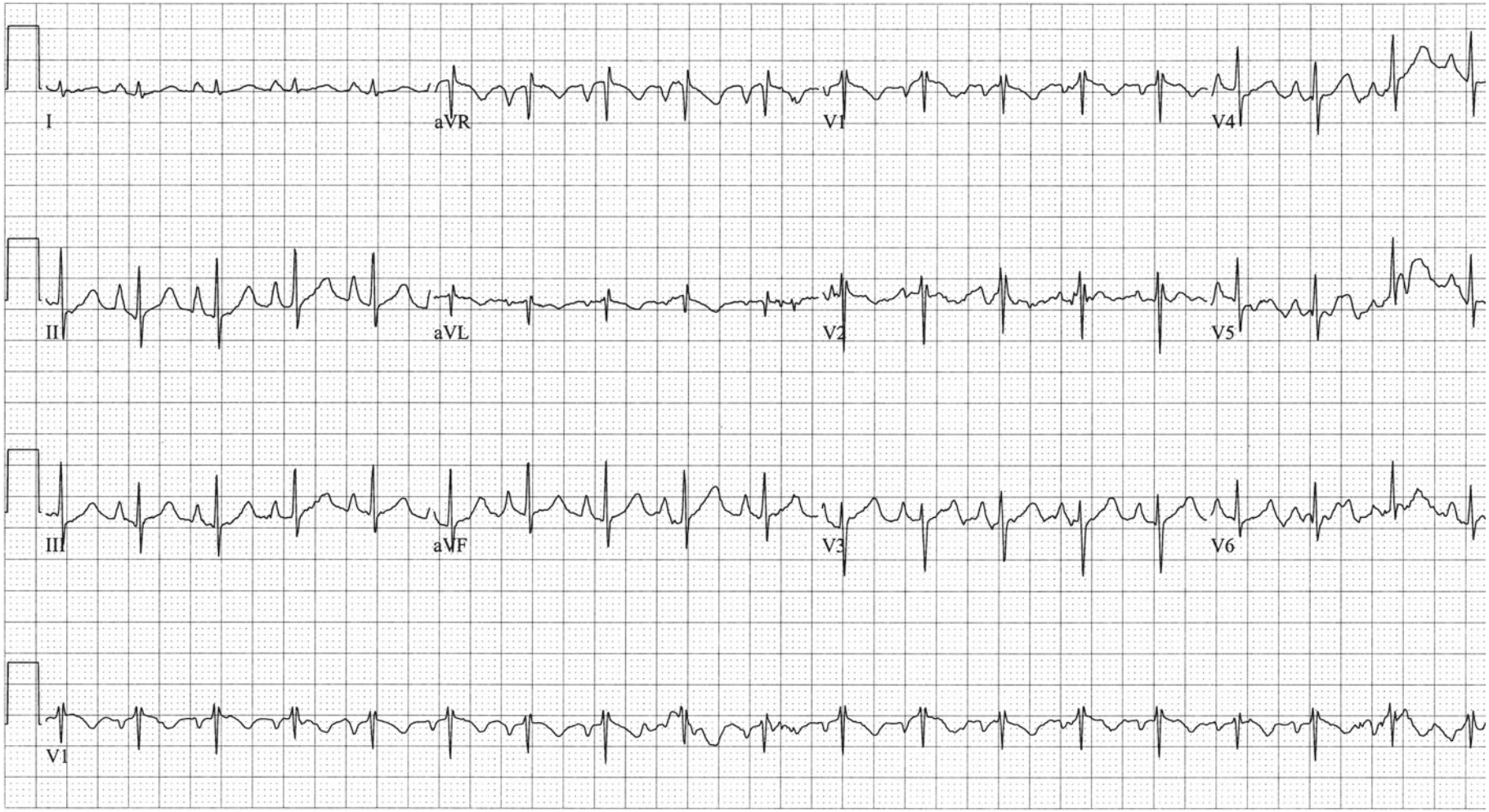


Exercise Tracings

Exercise Tracing 6.1



Exercise Tracing 6.2



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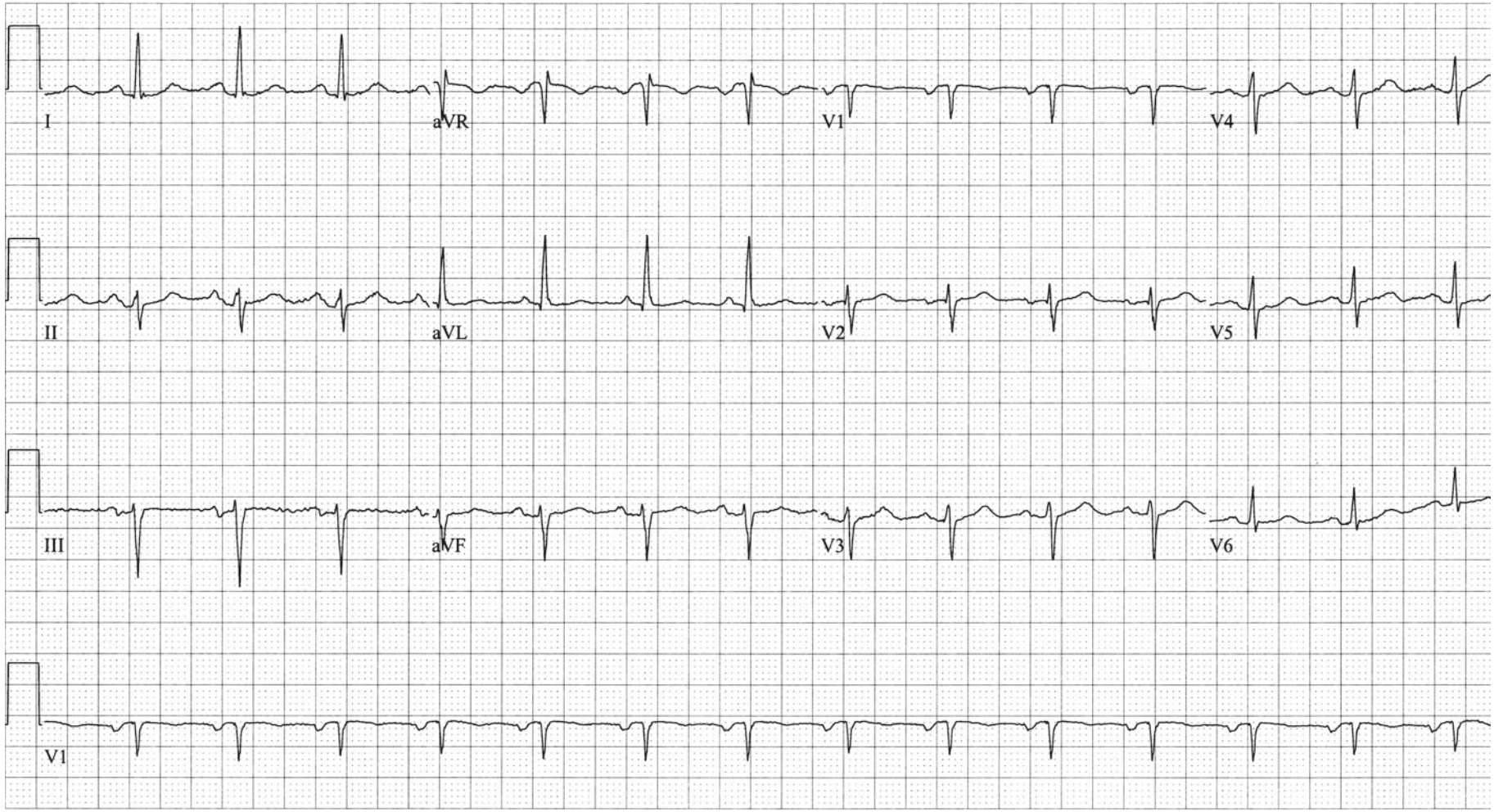
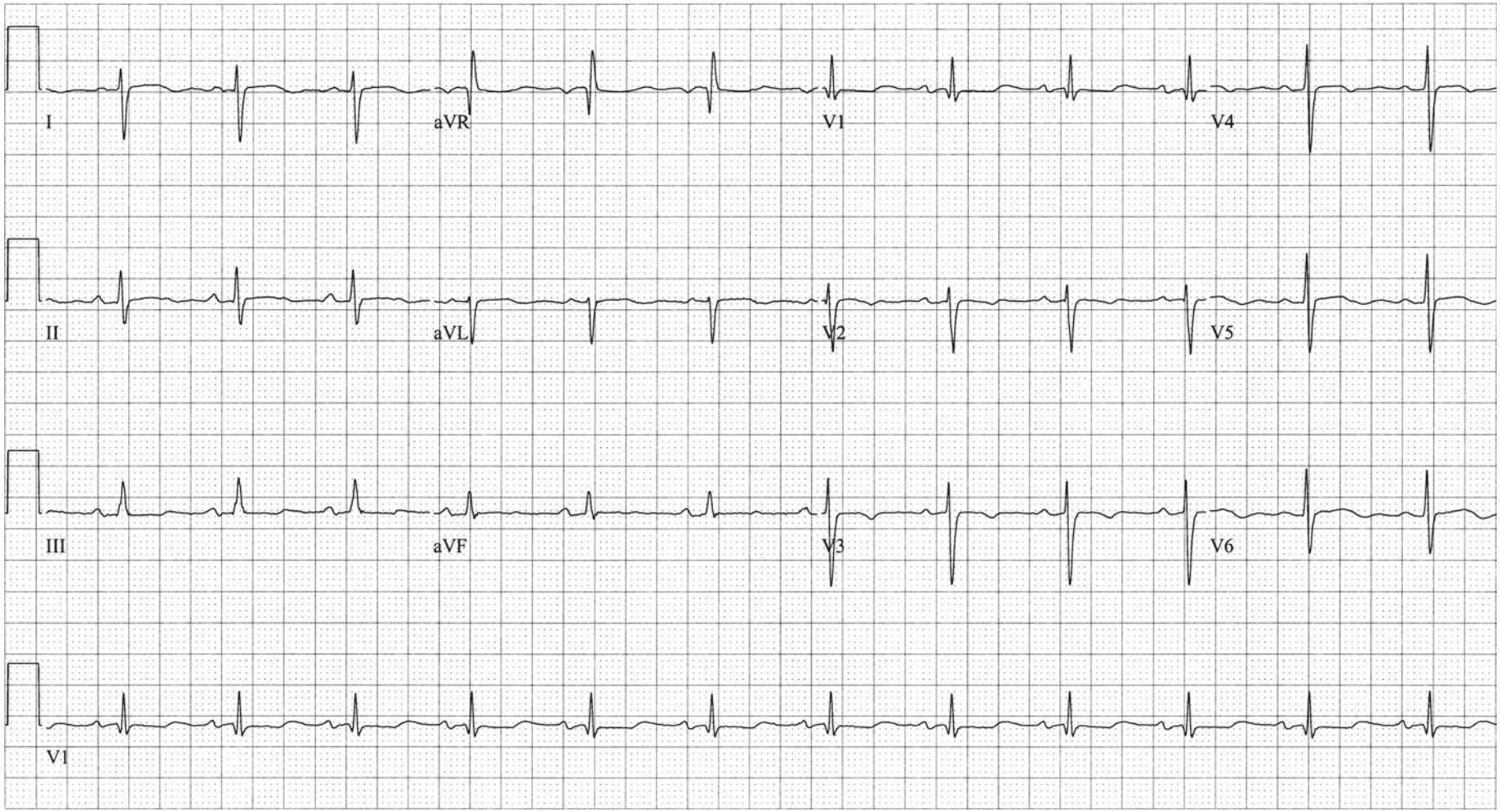


Exercise Tracing 6.4



Interpretations of Exercise Tracings

Exercise Tracing 6.1	
RATE:	A 79 V 79
RHYTHM:	Normal sinus rhythm
AXIS:	−10°
INTERVALS:	PR 0.16 QRS 0.10 QT 0.38
WAVEFORM:	Inverted T waves in I, II, aVL, $V_{5–6}$; biphasic T waves in V_4; ST depression in V_6; R wave about 30 mm in V_5, $RV_5 + SV_1 > 35$ mm; rSr′ in $V_{1–2}$; inverted P waves $V_{1–2}$
SUMMARY:	Abnormal due to LVH with strain, left atrial abnormality, incomplete right bundle branch block

Exercise Tracing 6.2	
RATE:	A 120 V 120
RHYTHM:	Sinus tachycardia
AXIS:	+80°
INTERVALS:	PR 0.13 QRS 0.08 QT 0.30
WAVEFORM:	rSr′ in V_1; tall P waves in inferior leads; inverted P in V_1, biphasic P in V_2
SUMMARY:	Abnormal due to left atrial abnormality, P pulmonale, sinus tachycardia, incomplete right bundle branch block

Exercise Tracing 6.3	
RATE:	A 92 V 92
RHYTHM:	Normal sinus rhythm
AXIS:	−40°
INTERVALS:	PR 0.17 QRS 0.08 QT 0.36
WAVEFORM:	Inverted P in V_1, biphasic P in V_2; small q in I, small r in III
SUMMARY:	Abnormal due to left atrial abnormality, left anterior hemiblock

Exercise Tracing 6.4	
RATE:	A 78 V 78
RHYTHM:	Normal sinus rhythm
AXIS:	+140°
INTERVALS:	PR 0.18 QRS 0.09 QT 0.37
WAVEFORM:	Tall R in V_1, persisting S in $V_{5–6}$
SUMMARY:	Abnormal due to right ventricular hypertrophy, non-specific T wave changes

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Arrhythmias

7

In the initial approach to an arrhythmia, three key questions must be answered. If the following questions are answered correctly, one can diagnose just about any arrhythmia:

1. Is it too fast or too slow?
2. Is it ventricular or supraventricular in origin?
3. Is it regular or irregular?

The first question can be answered easily. "Too fast" is a rate over 100 beats per minute; "too slow" is a rate less than 60 beats per minute. The answer to the second question is provided primarily with inspection of the duration of the QRS complex, and only secondarily with the presence or absence of P waves. If the QRS duration is normal (i.e., <0.12 s) then the rhythm *must* be supraventricular. If the QRS is prolonged (i.e., ≥0.12 s) one cannot immediately be sure if the rhythm is supraventricular in origin with aberrant conduction (e.g., bundle branch block) or is ventricular in origin. In cases of prolonged QRS complexes, the presence of P waves (or other evidences of supraventricular activity, such as flutter waves) and their constant relationship to QRS complexes can be helpful in distinguishing supraventricular from ventricular arrhythmias. In some cases, however, the distinction cannot be made with complete certainty from the surface 12-lead electrocardiogram (EKG). In such cases, esophageal or intracardiac leads may be required to absolutely determine the origin of the rhythm. The third question refers to the regularity of ventricular depolarization, or, in other words, the constancy of the RR interval. One should keep in mind that a minimal variation in RR interval is normal.

Once the determination is made of whether a rhythm is too fast or slow, regular or irregular, and ventricular or supraventricular, then one has taken the most important steps in distinguishing arrhythmias (Figs. 7.1 and 7.2). Each category of arrhythmias will be discussed separately.

Supraventricular Tachycardias

There are five primary supraventricular tachycardias (Fig. 7.2 and Table 7.1):

1. Sinus tachycardia
2. Atrial fibrillation
3. Atrial flutter
4. Paroxysmal (or reentrant) atrial tachycardia (PAT)
5. Multifocal atrial tachycardia (MAT)

The general category specified for these arrhythmias answers two of the three rhythm questions, namely that they are too fast and they are supraventricular. Now we need to answer the other question, namely whether these rhythms are regular or not (see Table 7.1). The (usually) regular supraventricular tachycardias are sinus tachycardia, atrial flutter, and PAT, while atrial fibrillation and MAT are irregular. After the question of regularity is determined, the atrial and ventricular rates may suggest one of the supraventricular tachycardias as opposed to another.

Sinus Tachycardia

In sinus tachycardia, the rhythm is regular. As already mentioned, there can be some minor variations in the RR interval with any sinus rhythm, but in general the RR interval is quite constant in sinus tachycardia. Each QRS is preceded by a P wave, which is usually but not always obvious in each lead (Fig. 7.3). The atrial rate with sinus tachycardia is 101 to about 190. It is impossible to make a normal heart go as fast as 250 beats per minute with sinus tachycardia. The ventricular rate in sinus tachycardia is the same as the atrial rate. The atrial rate limitation can be exceeded by atrial pacing, but at rates greater than about 200 beats per minute the normal atrioventricular (AV) node fails to conduct all of the supraventricular beats, and a pattern of AV block appears, usually Mobitz I (see Chap. 4). Thus, the AV node imposes a limit to the ventricular rate even with supraphysiological atrial rates.

Atrial Fibrillation

Atrial fibrillation is characterized by an irregularly irregular ventricular rhythm and a "wavy" baseline as a result of the

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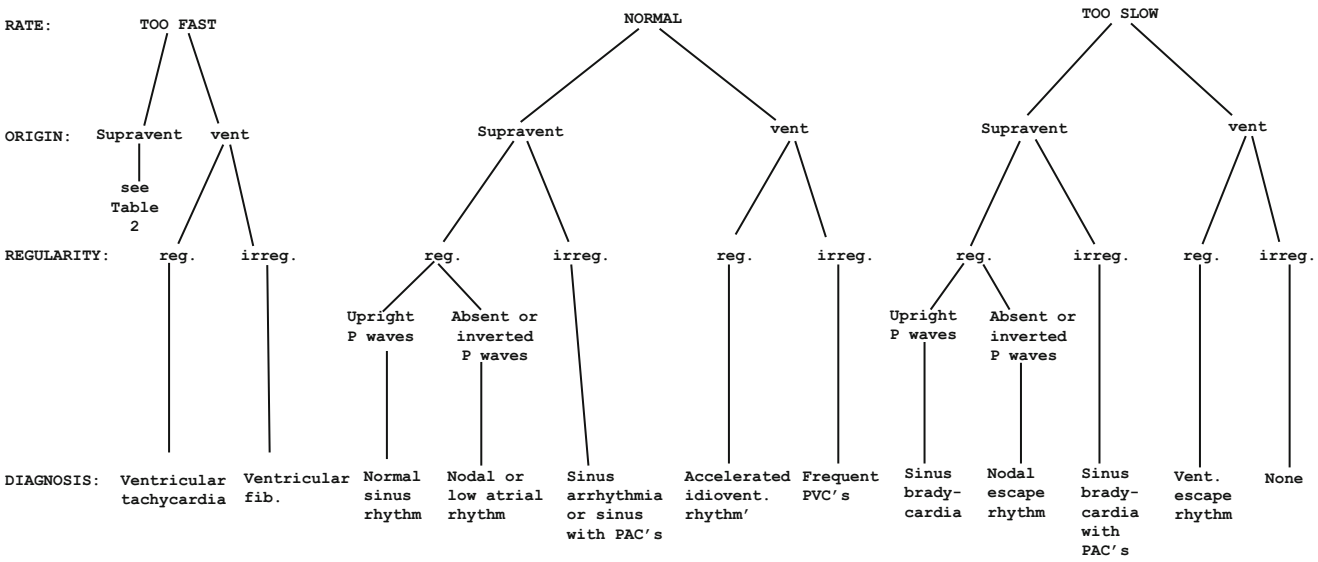


Fig. 7.1 Systematic approach to rhythm

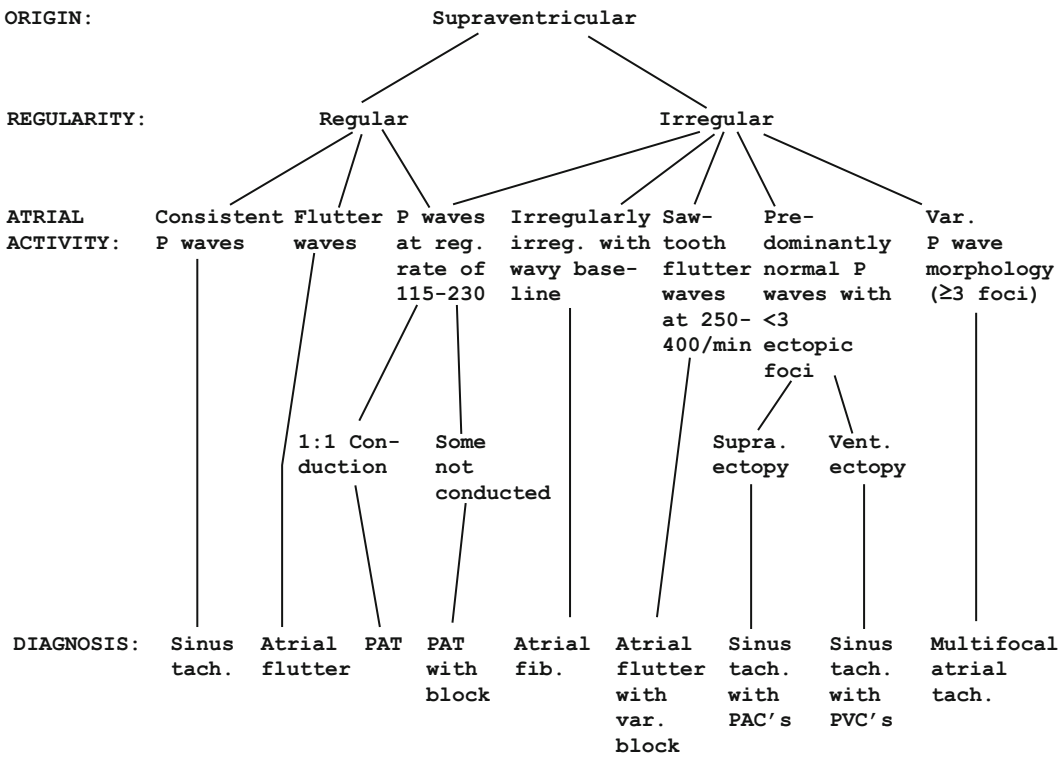


Fig. 7.2 Flow sheet for supraventricular tachycardia

chaotic, disorganized atrial activity. There is generally no pattern whatever to the sequence of ventricular (QRS) complexes in atrial fibrillation (Fig. 7.4). Sometimes there can be brief periods of regular or nearly regular ventricular depolarizations, but such regularity is short lived and may not be quite as regular as it may initially appear on casual inspection after when one takes a moment to carefully measure the RR intervals (Fig. 7.5).

Rarely, in patients with a history of atrial fibrillation who have received excessive digitalis ("digitalis intoxication"),

Table 7.1 Characteristics of the supraventricular tachyarrhythmias

SVT	Too fast?	Supraventricular?	Regular?	Atrial rate[a]	Ventricular rate[a]	Response to CSM[b]
Sinus tachycardia	+	+	+[c]	101–190	101–190	Transient atrial and ventricular slowing
Atrial fibrillation	+	+	–[d]	400–800	101–190	Transient ventricular slowing
Atrial flutter	+	+	+[e]	250–400 [1] (mean = 299)	101–190 (usually 2:1 block)	Transient ventricular slowing
Paroxysmal atrial tachycardia (PAT)	+	+	+	115–230 [1]	115–230 [1]	No response, or conversion to sinus rhythm
Multifocal atrial tachycardia (MAT)	+	+	–	101–190	101–190	Transient atrial and ventricular slowing

[a]Rates are general ranges, not absolute limits
[b]*CSM* carotid sinus massage
[c]Minor degrees of irregularity may occur
[d]Rarely the rate becomes regular (see text)
[e]Occasionally the AV block is not consistent, and the rhythm is irregular

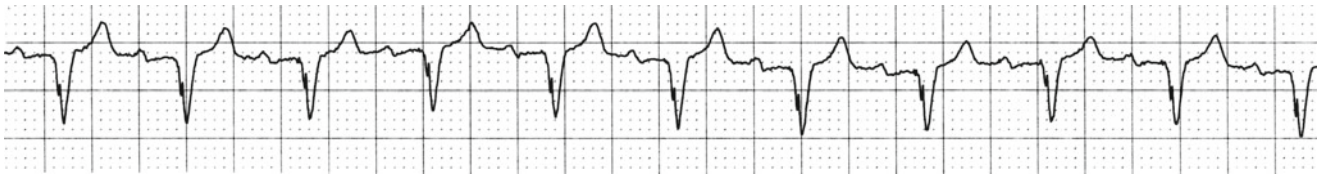


Fig. 7.3 Sinus tachycardia

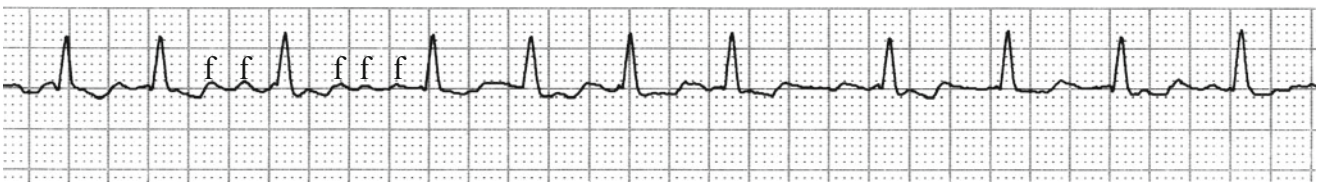


Fig. 7.4 Atrial fibrillation. Note the irregularly irregular fluctuation of the ventricular (QRS) complexes. The letter "f' is marked above typical fibrillatory waves

one can observe a regular rhythm. That regularization of rhythm as a manifestation of digitalis toxicity will be further described later in this chapter since it actually represents a different arrhythmia. However, these conditions of regularity associated with atrial fibrillation are rare, and atrial fibrillation is almost always irregularly irregular. In fact, any irregularly irregular rhythm should raise the likely possibility of atrial fibrillation until further evaluation proves otherwise. The presence of the small fibrillatory waves ("f" waves) serves to confirm the diagnosis. When the fibrillatory waves are large, the term "coarse" atrial fibrillation is sometimes used, as opposed to "fine" atrial fibrillation where the fibrillatory waves are small and nearly inapparent.

The atrial rate with atrial fibrillation is very fast, reaching 400–800 beats per minute. The ventricular rate is limited to about 200, just as with atrial pacing, since the normal AV node cannot conduct more rapidly than that. In the uncommon circumstance of an accessory pathway that bypasses the AV node, as with Wolff–Parkinson–White (WPW) or other preexcitation syndromes, the conduction can be much faster and may greatly exceed 200 beats per minute (Fig. 7.6). Therefore, in patients with preexcitation syndromes, the development of atrial fibrillation can be a catastrophic event since cardiac output falls greatly with rapid heart rates because ventricular filling time is severely shortened. Occasionally atrial fibrillation is present with a normal or slower-than-normal ventricular heart rate (Fig. 7.7). This usually occurs in patients on a medication that reduces AV conduction (e.g., verapamil) or with intrinsic AV node disease.

Atrial Flutter

It is interesting that the atrial rate with atrial flutter is usually within the fairly narrow range of about 300 ± 30 beats per

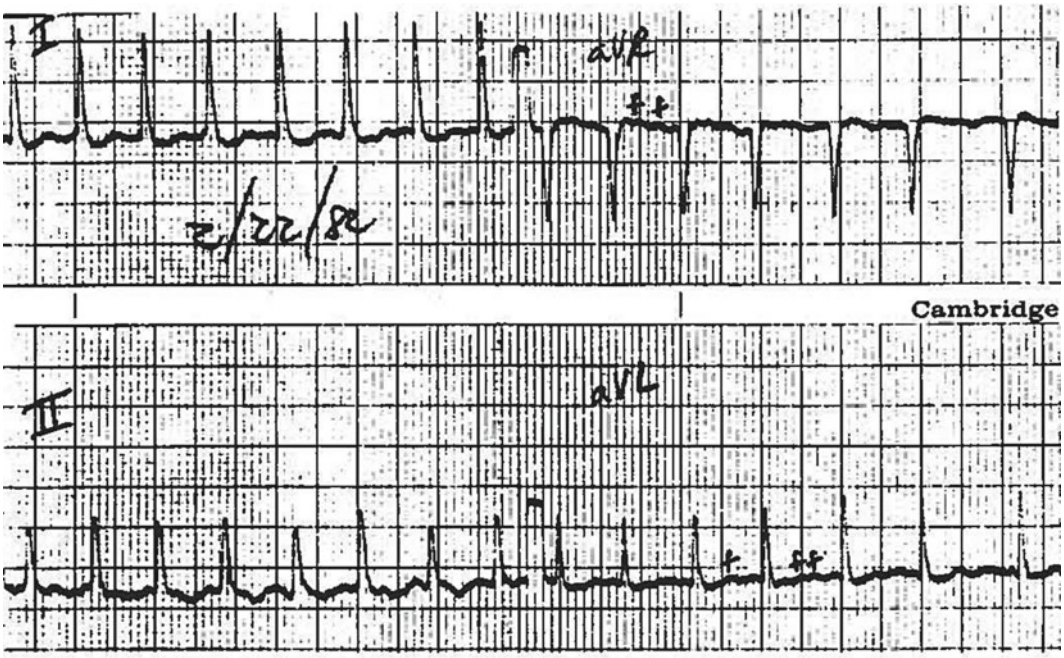


Fig. 7.5 Atrial fibrillation. The ventricular complexes are fairly regular at first glance, but with careful measurement of the RR intervals, especially near the end of the strip, the irregularity can be more easily appreciated. The letter "f" is marked above typical fibrillatory waves, which give the baseline a "wavy" appearance

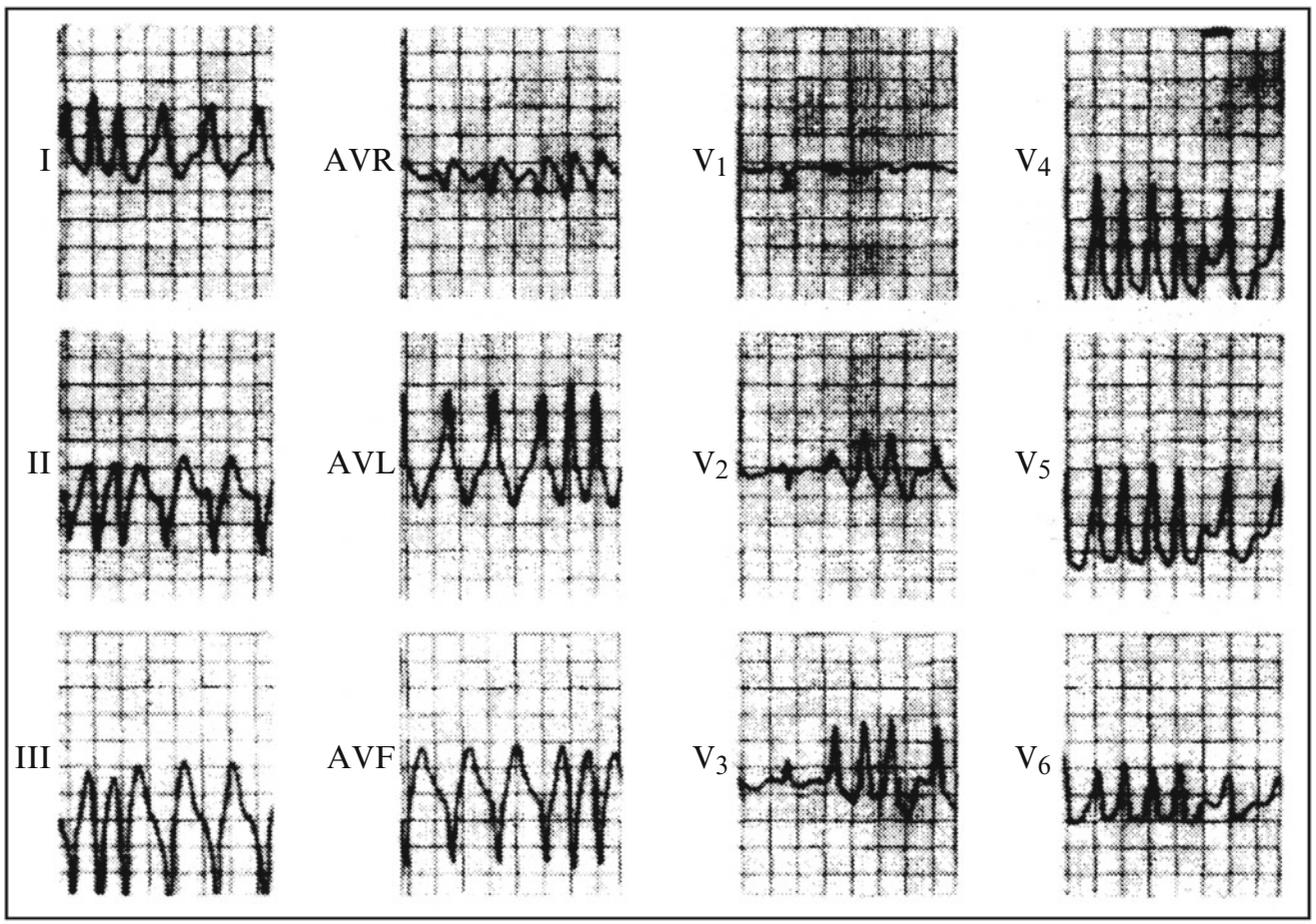


Fig. 7.6 Atrial fibrillation in a patient with Wolff–Parkinson–White syndrome. Note that the ventricular rate is frequently over 300 beats per minute. Reprinted from The American Journal of Medicine, 76(6), German LD, Gallagher JJ, Functional properties of accessory atrioventricular pathways in Wolff–Parkinson–White syndrome: Clinical implications, 1079–1086, Copyright 1984, with permission from Elsevier

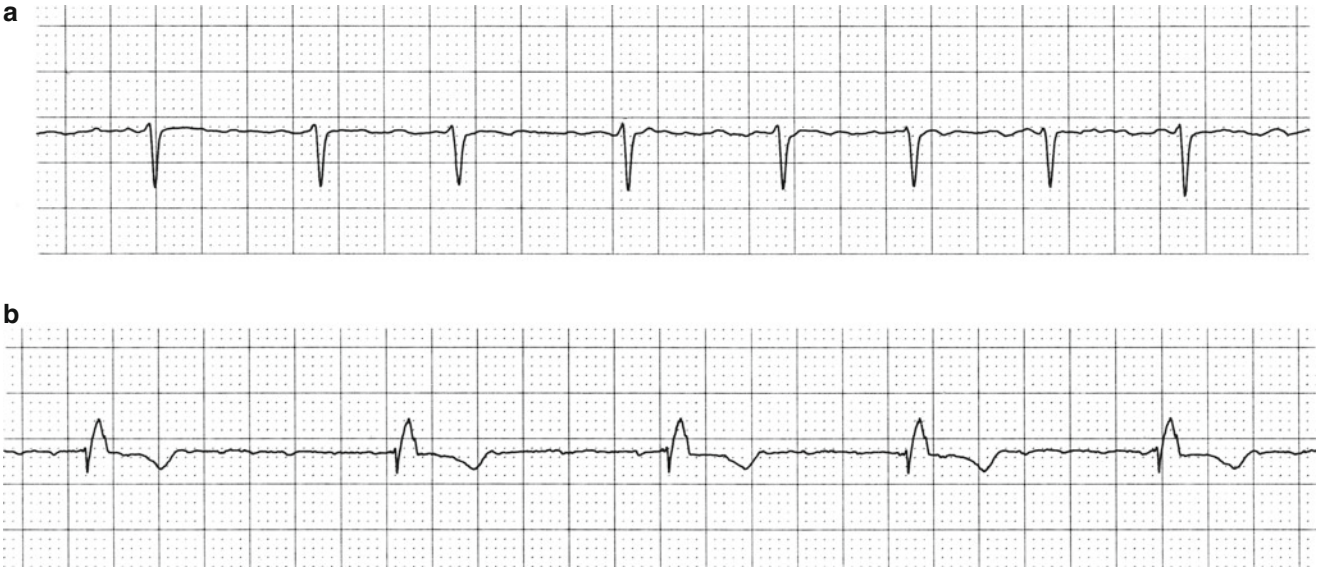


Fig. 7.7 Atrial fibrillation. (**a**) Normal ventricular rate (92 beats per minute). (**b**) Slow ventricular rate (51 beats per minute with pause of 1.35 s between the first and second beats)

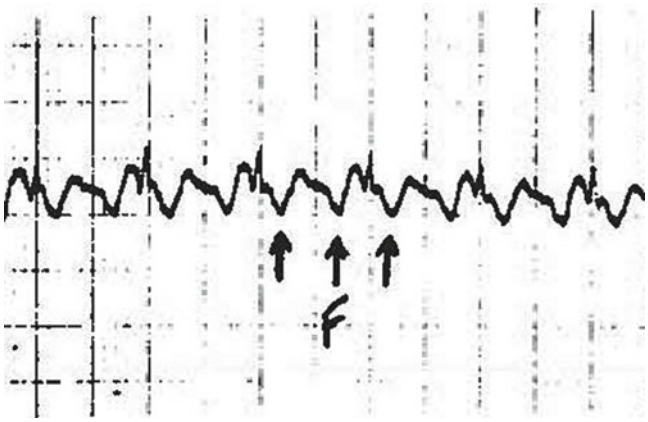


Fig. 7.8 Atrial flutter with 2:1 A:V block. *Arrows* indicate flutter ("F") waves. Note atrial rate is 300 beats per minute (FF interval = 0.2 s), while ventricular rate is 150 beats per minute (RR interval = 0.4 s)

minute, even though the rate can range between 250 and 400 beats per minute. The ventricular rate is usually about 150, since there is generally a fixed 2 to 1 block of conduction at the AV node when the atrial rate is so fast. The distinguishing flutter (F) waves in the classical "saw-tooth pattern" are often but not always recognizable on a rhythm strip, but generally are demonstrable on a 12-lead tracing (Fig. 7.8). Sometimes there is a greater degree of AV block, but this is not common and usually occurs, as with low rates in atrial fibrillation, if the patient is on a medication that increases AV block (e.g., verapamil) or the patient has intrinsic AV node disease (Fig. 7.9). Occasionally, the AV block with atrial flutter is variable, and this leads to an irregular ventricular rate. Any supraventricular tachycardia with a regular ventricular rate of 150 beats per minute should raise the possibility of atrial flutter and this should be the working diagnosis until further examination of the tracing confirms some other arrhythmia [1]. At least some cases of atrial flutter involve a large reentrant circuit involving the right atrium [2].

Paroxysmal Atrial Tachycardia

The atrial rate for PAT is usually 115–240 with an equal ventricular rate (1:1 AV conduction). If both the sino-atrial (SA) node and the AV node have rate limits of about 200 beats per minute, how is it possible that PAT achieves a rate over 200 beats per minute? First, PAT usually does not involve the SA node, and so the rate limitation imposed by the SA node is avoided. That also explains why the atrial depolarization with PAT usually causes abnormal P waves or no apparent P waves at all (hidden by QRS and/or T waves). Second, PAT is usually due to a reentry mechanism that involves the AV node. When electrophysiological testing is done and confirms that the reentry involves the AV node, it is properly called AV nodal reentry tachycardia, or AVNRT. When electrophysiologic studies show that the reentry does not include the AV node, it is called AV reciprocating tachycardia, or AVRT. Because at least part of the AV node (the usual bottleneck of conduction into the ventricle) is bypassed, the ventricular rate can reach 240 beats per minute. Whenever there is a supraventricular tachycardia with a ventricular rate greater than 200, the first thing that should come to mind is PAT. In fact, it is not likely to be anything else. Rarely, one might see a brief burst of atrial fibrillation that will go 200 beats per minute or so, but with rates around 240 the rhythm is usually PAT. The only exception to this dictum is the situation where

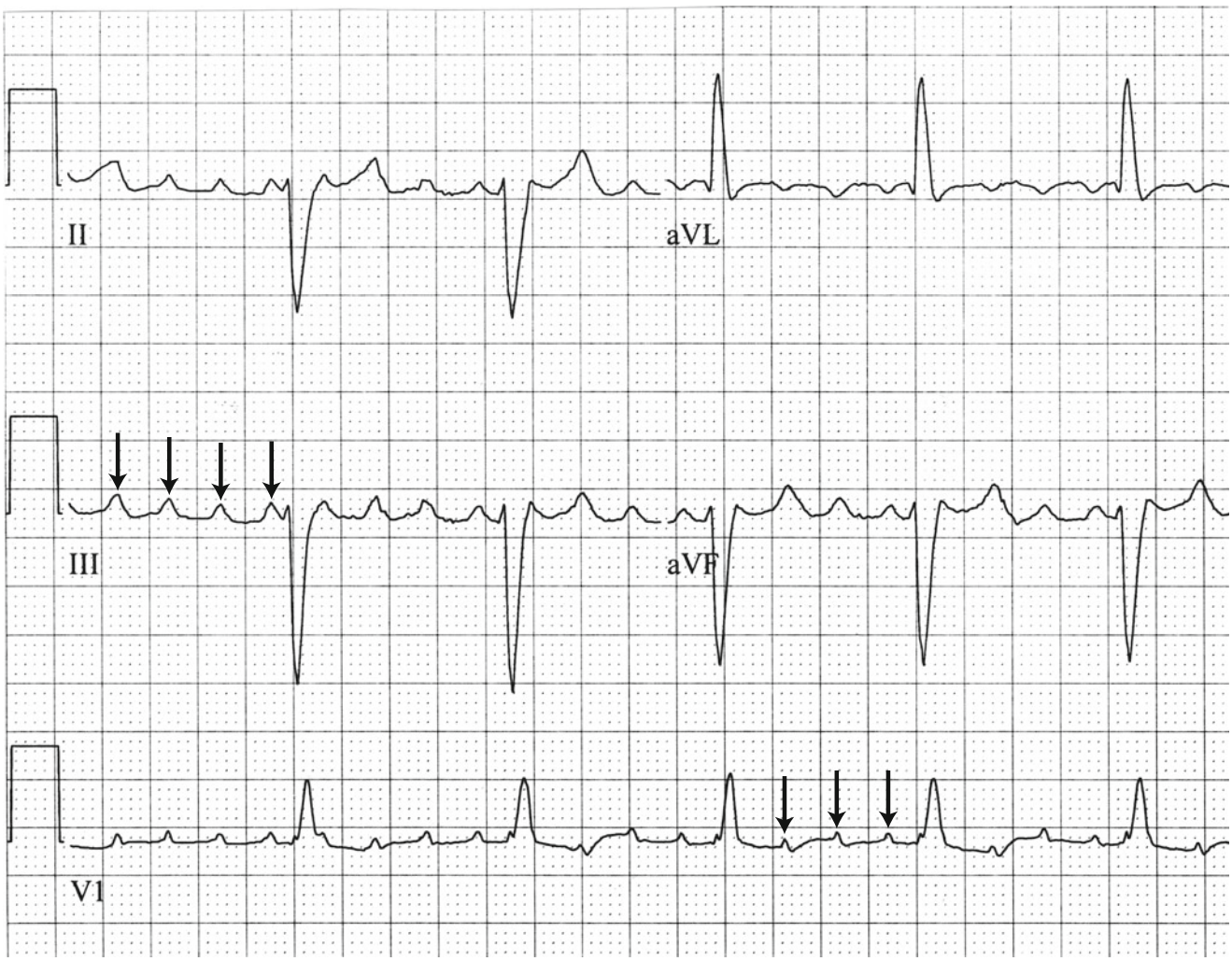


Fig. 7.9 Atrial flutter with 4:1 A:V block. Flutter waves, marked with *arrows*, appear at a rate of approximately 285 beats per minute

there is an accessory pathway bypassing the AV node, in which case atrial flutter or fibrillation may achieve rates as high or higher than PAT (see Fig. 7.6).

At this point it is appropriate to elaborate on WPW syndrome, the classic preexcitation syndrome. Accessory pathways between the atria and the ventricles are typical for preexcitation syndromes, and in WPW an accessory pathway is responsible for the short PR interval (because the impulse gets into the ventricle faster than going through the AV node) and the pathognomonic "delta wave" (from slightly aberrant initial depolarization of the ventricle) (Fig. 7.10). WPW syndrome is not an arrhythmia—it is a syndrome that predisposes to PAT because the accessory pathway can become part of a conduction circuit. WPW can be Type A (tall R wave in V_1) or Type B (negative QRS in V_1).

In the presence of digitalis toxicity, PAT may develop with a 2:1 or greater block, and the rhythm may still be regular (Fig. 7.11). When digitalis toxicity occurs in a patient previously in atrial fibrillation, the irregular rhythm of atrial fibrillation may be replaced by a regular PAT in fixed block. Without an EKG, this situation could be mistaken on physical examination for normal sinus rhythm. Therefore, when a patient previously in atrial fibrillation treated with digitalis presents with a regular rhythm, one must obtain a tracing to determine if the rhythm is really sinus rhythm or is PAT or atrial flutter with fixed block. On the other hand, PAT may rarely occur with variable block, and without an EKG it would be indistinguishable from atrial fibrillation. While PAT with block has classically been observed in digitalis intoxication, it may also occur in other settings (Fig. 7.12).

Multifocal Atrial Tachycardia

MAT is an uncommon supraventricular tachycardia that is usually irregular. The diagnosis of MAT requires that the patient has a supraventricular tachycardia with at least three

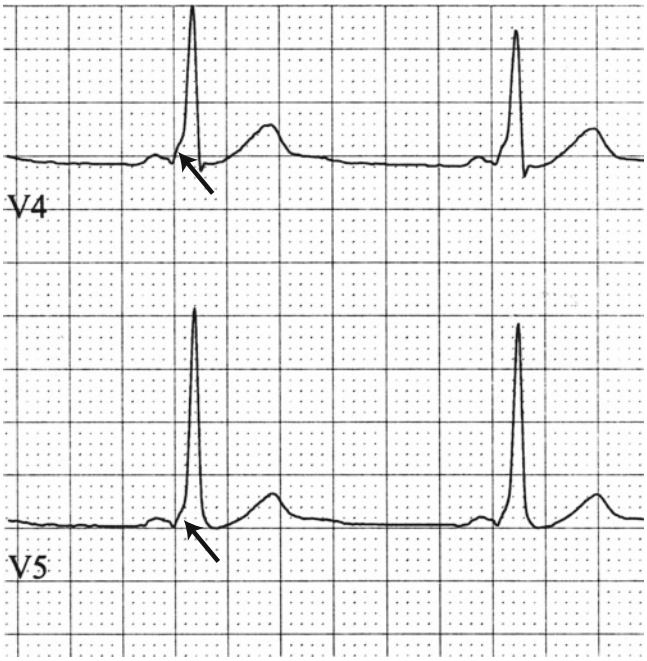


Fig. 7.10 Wolff–Parkinson–White syndrome. *Arrows* indicate diagnostic delta waves, the slurred upstroke of the QRS, which is distinctly different than the usual steep, linear upstroke

different configurations of P waves and varying PR intervals (Fig. 7.13). This means that there are at least three separate foci of supraventricular activation. It is called MAT when the rate is over 100 beats per minute. When the rate is normal and there are three or more supraventricular foci of activation, the rhythm is termed "wandering atrial pacemaker." Wandering atrial pacemaker, in a sense, is slow MAT, or MAT is wandering atrial pacemaker with a rapid rate.

Differentiating Supraventricular Tachycardias

It is clear that a patient with a supraventricular tachycardia and a ventricular heart rate of about 155 beats per minute could possibly have any of the five arrhythmias discussed above. If the rhythm is irregularly irregular, it is most likely atrial fibrillation, though atrial flutter or PAT with variable block, MAT, or sinus tachycardia with frequent premature contractions can give an irregularly irregular rhythm (Table 7.2).

When the rhythm is regular, there is sometimes difficulty in distinguishing sinus tachycardia, atrial flutter with 2:1 AV block, and PAT. When P waves or F waves are obvious and

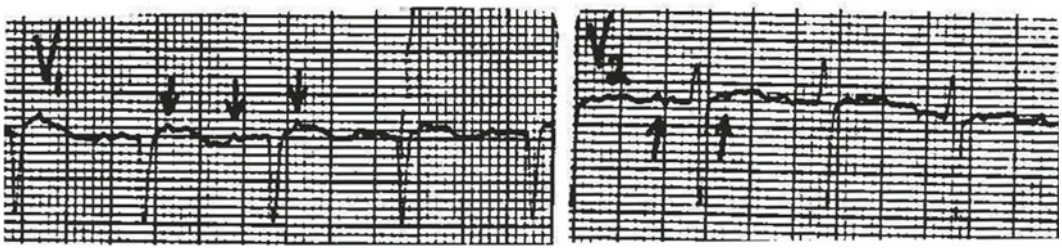


Fig. 7.11 PAT with 2:1 A:V block. *Arrows* show atrial depolarization

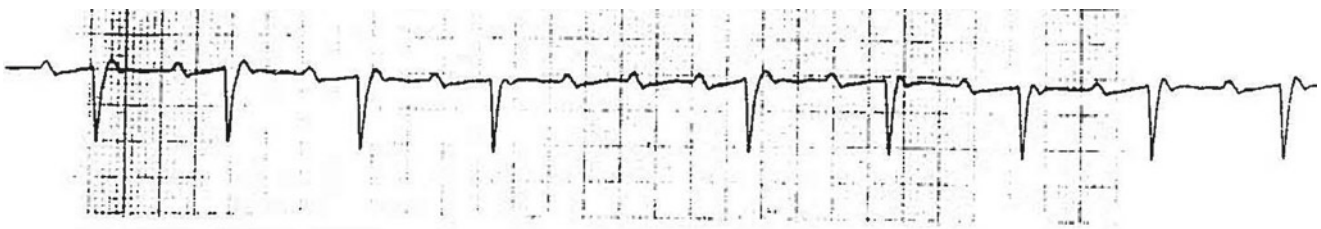


Fig. 7.12 PAT with block. Most of the strip is 2:1 block, but there is a cycle of 4:1 block in the middle of the strip

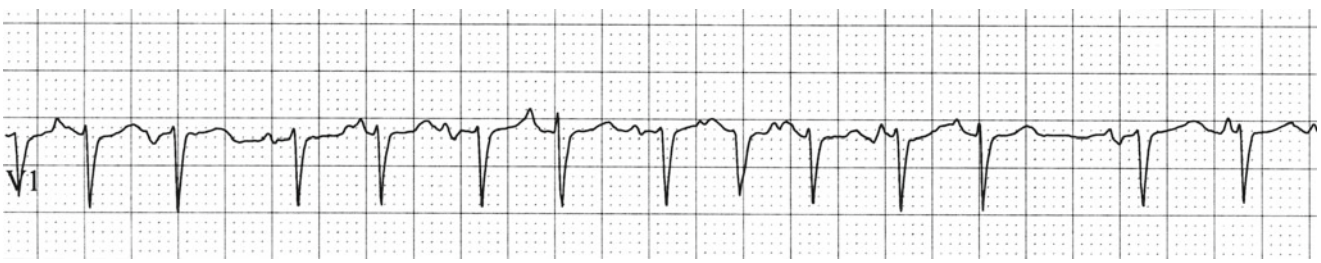


Fig. 7.13 Multifocal atrial tachycardia. Note the varying P wave configuration and PR intervals

Table 7.2 Differential diagnosis in a patient with a rapid, irregularly irregular pulse

Atrial fibrillation
Atrial flutter with variable block
PAT with variable block
Sinus tachycardia with frequent PACs
Sinus tachycardia with frequent PVCs
Multifocal atrial tachycardia

Table 7.3 Appropriate precautions with carotid sinus massage

Check for carotid pulsations and listen for bruits
Examine fundi for cholesterol emboli
Continuous EKG monitoring
Intravenous access established
Patient supine
Light–moderate pressure initially (especially in elderly patients)
Atropine immediately available
Never bilateral pressure simultaneously

recognizable, their presence can allow one to distinguish the rhythm. Sometimes, however, P waves and F waves are fairly flat in some leads and not well seen, or they may be "hidden" in T waves or QRS complexes. In such cases, where the distinction between these regular supraventricular tachycardias is not clearly obvious, there is a simple and cheap diagnostic maneuver that one can consider using. It is carotid sinus massage, which increases vagal tone and thereby increases the degree of block at both the SA and AV nodes. Other maneuvers may be used to increase vagal tone, but seem to be either more barbaric [e.g., eyeball pressure or putting the patient's face into cold water ("diver's reflex")] or may interfere with the quality of the EKG record (Valsalva maneuver, cough). The mechanics of carotid sinus massage are fairly simple (Table 7.3). It should be done with the patient supine, the EKG limb leads attached, and the machine running. It is desirable to have an intravenous line in place.

Before performing carotid sinus massage, one should listen for carotid bruits and palpate for reduced carotid pulsations, both suggesting the possibility of carotid stenosis. Though carotid stenosis is only a relative contraindication to carotid sinus massage, it suggests that you should be particularly cautious. You should also examine the fundi for cholesterol emboli, the presence of which suggests ulcerative plaque disease in the ipsilateral carotid system, another relative contraindication. In the face of such relative contraindications, other methods of increasing vagal tone may be considered, such as Valsalva or administration of intravenous adenosine or edrophonium. If carotid artery stenosis and evidence of cholesterol emboli on funduscopic exam are absent, one may proceed by warning the patient that there may be a bit of discomfort associated with the carotid sinus pressure but reassure the patient that all necessary precautions have been taken and the discomfort is not severe. The patient's head should be turned slightly away from the examiner, exposing the neck. The carotid sinus is at the bifurcation of the common carotid into the internal and external carotid arteries. This is located in the mid-neck at the point where the carotid pulsation is strongest to palpation. With the EKG machine running continuously, either you or an assistant mark the point on the tracing at which you start carotid sinus pressure. One pushes for 2–3 s, then stops, and observes on the EKG record what the response was to massage. If no changes occur on the tracing, one can try carotid massage again and push a little harder. If nothing happens a second time, one can try a third time or try massage of the carotid on the other side of the neck. One should *never* massage both carotids simultaneously. In elderly patients, one should apply only gentle carotid pressure initially because occasionally these patients are very sensitive to carotid sinus massage and may develop a marked increase in vagal tone and therefore a marked degree of block at the AV node in response to carotid sinus massage.

The response of each of the supraventricular tachycardias to carotid sinus massage or other vagal maneuvers is depicted in Table 7.1. In sinus tachycardia, the heart rate temporarily falls because both the SA and AV nodes are suppressed by vagal stimulation, but the rate soon recovers to baseline because nothing was done to reverse the underlying cause of sinus tachycardia, such as fever, anxiety, or hyperthyroidism. With atrial fibrillation, carotid sinus massage causes a temporary fall in the ventricular rate, but carotid sinus massage has little place in atrial fibrillation because the arrhythmia is generally so easily recognized. With carotid sinus massage in atrial flutter, one also sees a temporary fall in the ventricular rate, but the rate may recover in a more stepwise fashion as the AV block dissipates. Frequently, increasing the AV block in patients with atrial flutter allows one to expose and more easily identify the flutter waves, which may previously have been obscured by QRS complexes and/or T waves (Fig. 7.14). In other words, carotid sinus massage in atrial flutter, by increasing AV block, "spreads out" the QRS complexes, better revealing the flutter waves. In contradistinction to the previous arrhythmias, carotid sinus massage may have no effect at all with PAT. The only other possibility is that the electrical circuit perpetuating PAT can be broken and the rhythm converted to a sinus rhythm. Carotid sinus massage is an "all or nothing" phenomenon with PAT. One can convert the patient into sinus rhythm, or nothing happens. A temporary reduction in heart rate, followed by a gradual recovery to the pre-massage heart rate, does not occur if the arrhythmia is PAT. The reason PAT converts to sinus rhythm is that the vagus nerve inhibits the conduction through the AV node, and since the node is usually part of the circuit which perpetuates PAT, inhibiting AV nodal conduction may increase the block in the circuit and so interrupt the circuit and allow sinus rhythm to return. While carotid sinus massage is primarily a diagnostic maneuver, in the case of PAT it may be therapeutic as well.

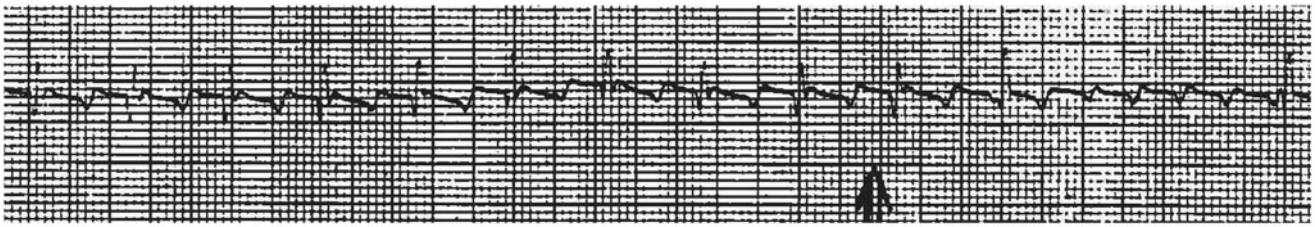


Fig. 7.14 Supraventricular tachycardia with a rate of 130 beats per minute. *Arrow* notes point where carotid sinus massage begins. Ventricular rate falls, and flutter waves are then clearly discernible, distinguishing this regular SVT (atrial flutter) from sinus tachycardia or PAT

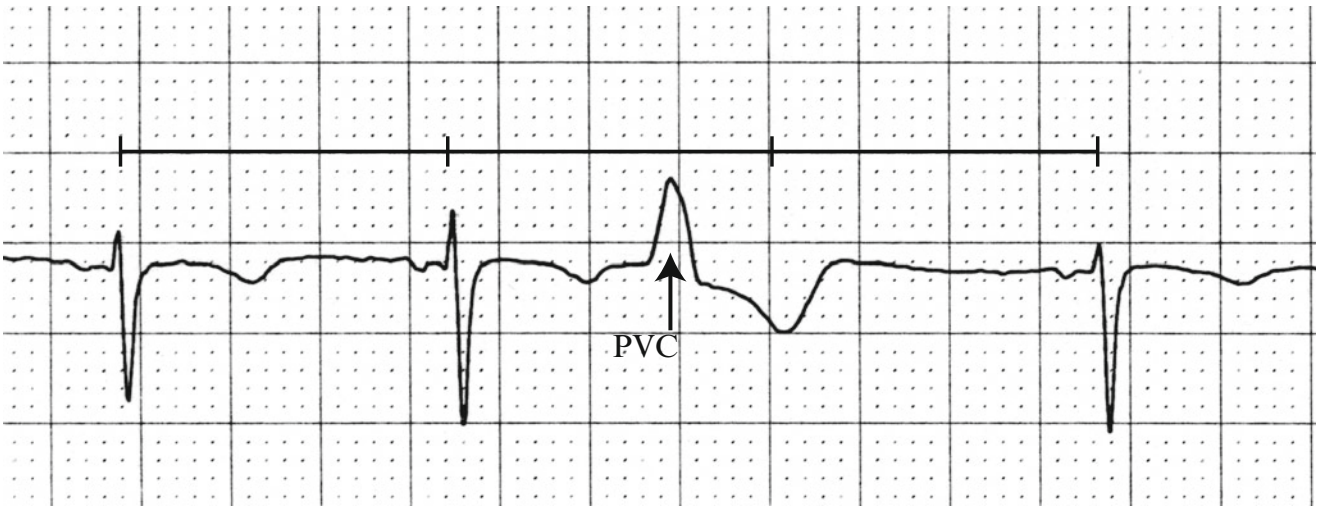


Fig. 7.15 Premature ventricular contraction (PVC). The R-R interval of the normal sinus rhythm is depicted above the complexes, and a compensatory pause follows the PVC, the next sinus beat occurring two R-R intervals after the previous sinus beat

To reiterate, never do carotid sinus massage without (1) having a continuous EKG record, (2) having an IV line in place, and (3) searching for and considering relative contraindications. Exuberant carotid sinus massage may cause excessive and dangerous AV block with a drastic fall in heart rate and consequently blood pressure.

Ventricular Arrhythmias

There are five ventricular arrhythmias:

1. Ventricular escape (covered in Chap. 4)
2. Premature ventricular contractions (PVCs)
3. Accelerated idioventricular rhythm (AIVR)
4. Ventricular tachycardia (VT)
5. Ventricular fibrillation

Premature Ventricular Contractions

PVCs are just what the name implies: premature beats arising from a ventricular focus. PVCs have a prolonged QRS duration (0.12 s or longer) and generally are somewhat different in axis than the normal supraventricular beats. They usually are followed by a "compensatory pause," in which the next supraventricular beat occurs at or very near the time of two normal R-R intervals after the last beat prior to the PVC (Fig. 7.15). This suggests that the SA node is not reset by the PVC and fires on time, one ventricular beat being dropped because of the refractoriness of the ventricle and/or AV node following the premature beat. The compensatory pause is a usual but not unique feature of ventricular ectopy. Supraventricular ectopy may also cause an apparent compensatory pause, but this is not common; more commonly, the SA node is reset by a premature supraventricular beat and a compensatory pause is not observed (Fig. 7.16).

Rarely, particularly with slow underlying heart rates, a PVC may occur and not be followed by a compensatory pause. The next sinus beat occurs on time and is conducted normally or is conducted with slight PR interval prolongation due to relative rather than total refractoriness of the AV node. The PVC falling between two sinus beats without inducing a compensatory pause is called an interpolated PVC, and if PR prolongation occurs in the sinus beat following the PVC, it is called concealed retrograde conduction—i.e., conduction into the node causing delay of antegrade

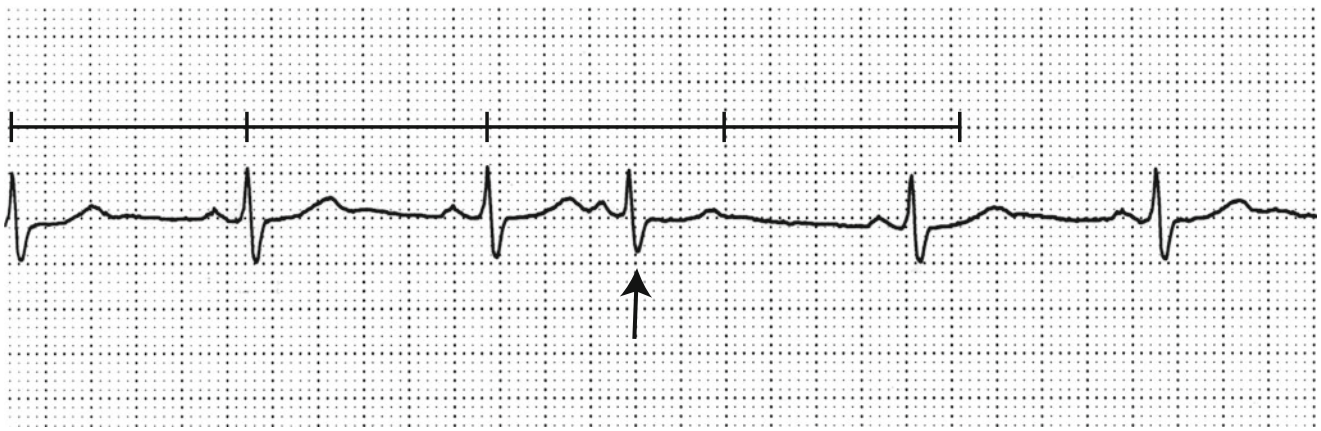


Fig. 7.16 Premature atrial contraction (PAC). This typical PAC (*arrow*) is normal in QRS duration and essentially identical to the normal QRS complexes. Furthermore, the next normal sinus beat following the premature beat is not close to two RR intervals from the previous sinus beat, and thus the pause following the PAC is not "compensatory"

conduction, but not causing depolarization in a retrograde direction up into the atria (Fig. 7.17).

Sometimes it is difficult with premature beats to make the distinction between ventricular origin and supraventricular origin with aberrancy of conduction. Invasive electrophysiological studies may be needed to confirm where the ectopic site is located. The presence of a "fusion beat," however, allows one to confirm the location of the ectopic focus to the ventricle. The shape of a fusion beat is a hybrid or intermediate shape between the normal, narrow supraventricular beat and the ectopic beat with a wide QRS (Fig. 7.18). Fusion beats indicate a combined pattern of ventricular depolarization from two directions: (1) normally through the AV node and the bundles, and (2) simultaneously from an ectopic ventricular focus.

Accelerated Idioventricular Rhythm

AIVR refers to a ventricular rhythm at a rate faster than the usual escape or automaticity rate of the ventricles (35–45 beats per minute), but at a slower rate than a tachycardia (i.e., lower than 100 beats per minute) (Fig. 7.19).

Ventricular Tachycardia

Ventricular tachycardia is a rhythm arising from a ventricle at a rate greater than 100 beats per minute. Ventricular tachycardia is regular, though at the initiation of ventricular tachycardia the rate may be slightly slower, then speed up promptly to a persistent, regular, and fast rhythm. The complexes are wide and bizarre (Fig. 7.20), but still have some degree of recognizable form and shape, as opposed to ventricular fibrillation, where there is only a wavy baseline and no recognizable orderly electrical activity (no recognizable QRS complexes) and there is likewise no ventricular (pump) function to it.

When one identifies a rhythm in a monitored patient as ventricular tachycardia, how should the patient initially be approached? The first thing you do, in my view, is to walk briskly, (but don't run) into the patient's room. This avoids a high-speed collision with another health care provider rushing to attend to the same patient! Upon your safe arrival in the patient's room, before initiating any therapy, you use the time-proven, thoughtful approach of medicine—you take a history and do a physical examination. That can be done very expeditiously (in seconds) and without negatively affecting the patient's outcome. The basic history in this situation consists of one question: "Hello, Mr. or Ms._____; how are you feeling?" You usually get one of two responses: (1) "Oh, I feel okay, a little tired, lightheaded, dizzy maybe, but not so bad," or (2) no response. In the second case, that is the end of the history. The physical exam is important after you get either history. You feel for the pulse and see if the patient is breathing. That physical examination will take only 15 s or less and is worth every second. If you get response #1, and feel a good pulse, measure the blood pressure. If the blood pressure is acceptable, then you can go on and expand the history and complete the cardiopulmonary physical examination. If you get response #2 and the patient is apneic and pulseless, then initiate cardiopulmonary resuscitation. The therapeutic modality you should employ to treat the arrhythmia in this setting is electrocardioversion. Why don't you go in and shock the patient first, when the arrhythmia is first noted? Because he may give you history #1, and he would not be pleased at all to undergo electrocardioversion if he is not unconscious! An alternative to electrocardioversion, especially before the defibrillator arrives in the room, is sternal thump. This is sometimes helpful to reverse asystole and may rarely convert episodes of ventricular tachycardia. As is

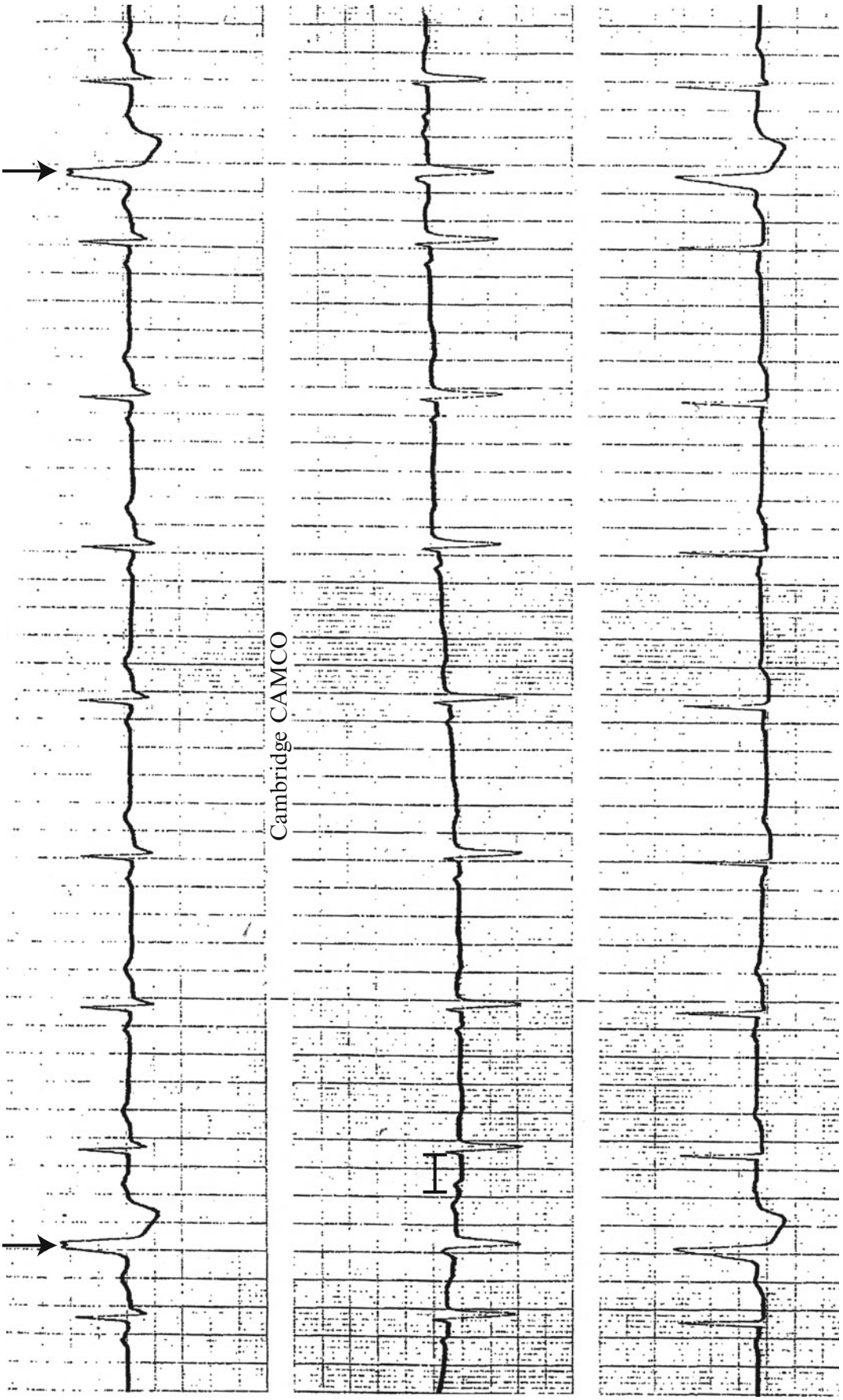


Fig. 7.17 Interpolated PVCs with concealed retrograde conduction. There is no compensatory pause following the PVCs since the underlying heart rate is so slow. The PR interval of the sinus beat after the PVC (*bracket*) is prolonged to about 0.25 s compared to the usual PR of about 0.17 s. This is because of incomplete or "concealed" conduction retrograde into the AV node after the premature ventricular contraction. This concealed conduction makes the AV node more refractory to antegrade conduction than during normal conduction, and so the PR interval of the sinus beat after the interpolated PVC is increased

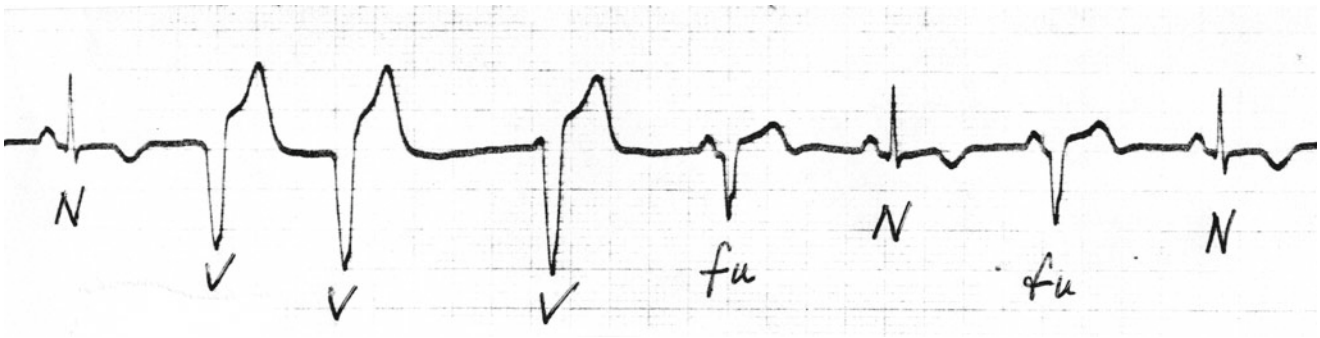


Fig. 7.18 Fusion beats. This tracing shows a sinus beat with normal antegrade conduction (N) and then three beats with wide QRS complexes. The sixth and eighth beats are also normal. The fifth and seventh beats, however, are not normal but are not as wide or deep as the previous beats. This intermediate configuration is typical for fusion beats (labelled "fu"), and denotes that the ectopic, wide complexes are definitely arising from the ventricle, and so are ventricular ectopic beats (labelled "V")

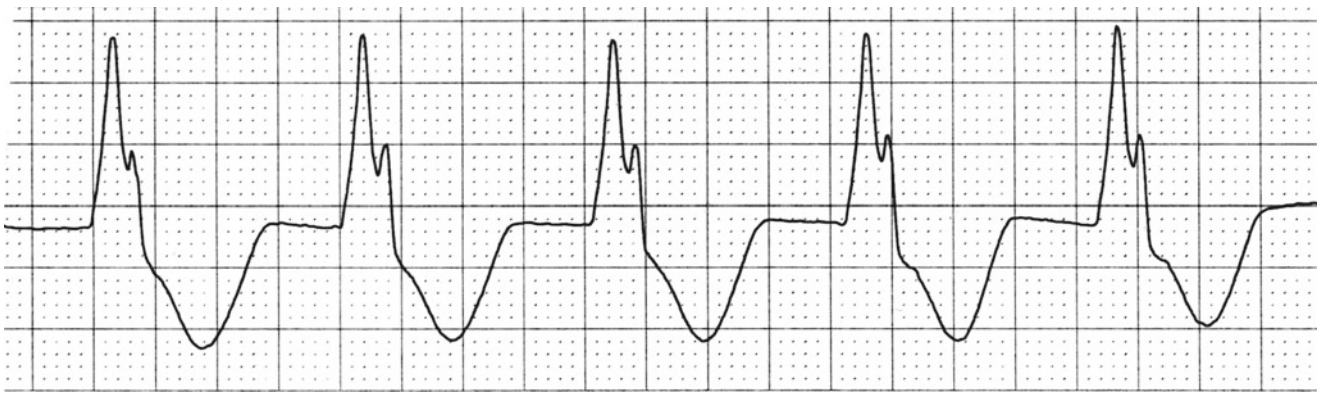


Fig. 7.19 Accelerated idioventricular rhythm. This is a regular ventricular rhythm at a relatively rapid rate (in this case 74 beats per minute)

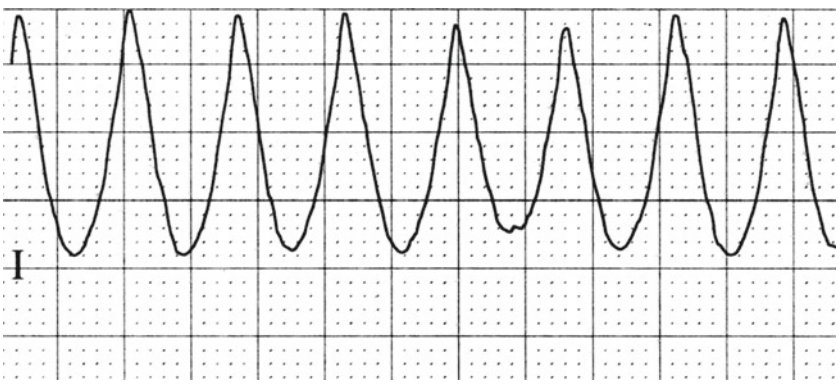


Fig. 7.20 Ventricular tachycardia

the case with electroshock, however, sternal thump is not appreciated in a patient who gives you history #1 or would have given that history if you had asked her/him. Additionally, it is important to note two things about ventricular tachycardia: (1) ventricular tachycardia is not always a lethal rhythm, and especially in younger patients with relatively healthy hearts (from the point of view of pump function) ventricular tachycardia may occur, yet still provide an adequate pump function and produce adequate blood pressure; (2) not everything that looks like ventricular tachycardia is ventricular tachycardia (Fig. 7.21). Therefore, when faced with clinical decisions for the management of any arrhythmia, always take a history and do a physical examination, and if the patient is responsive and the blood pressure is reasonably good, then you have the clinical luxury to do a 12-lead EKG and perhaps other maneuvers to determine what arrhythmia is present.

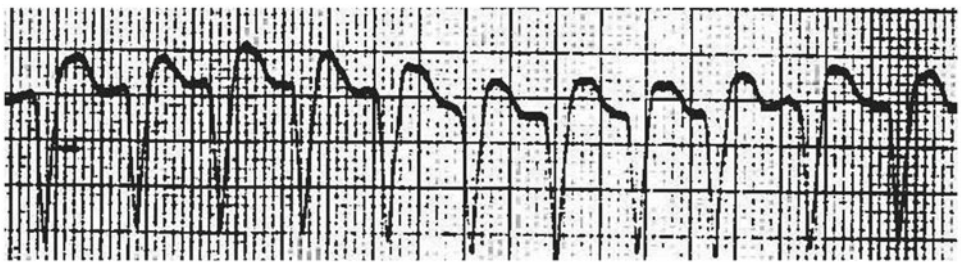


Fig. 7.21 Tachycardia that looks like ventricular tachycardia at first glance, but that on closer inspection is irregularly irregular. This strip is part of a tracing from a patient with left bundle branch block in atrial fibrillation

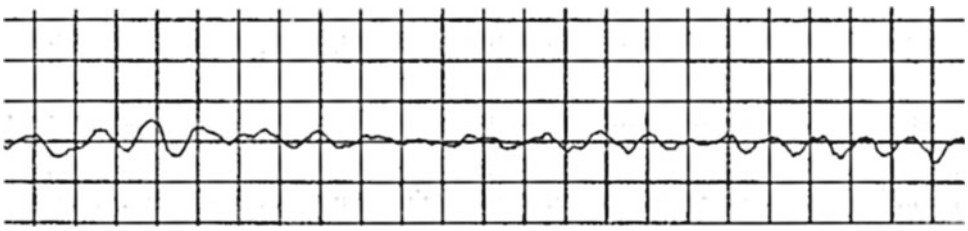


Fig. 7.22 Ventricular fibrillation. The tracing is composed of only a wavy baseline, with no recognizable QRS complexes

Another caution in dealing with arrhythmias: don't trust monitor (telemetry) strips to make a definitive diagnosis. In my opinion, monitor (telemetry) strips are good primarily to indicate that there is or is not an arrhythmia; they are not always reliable for diagnosis. They can tell you if an arrhythmia is regular or irregular and allow you to determine the heart rate, but that is about the limit of their usefulness. Whenever the clinical situation allows (i.e., the patient is not in cardiopulmonary arrest), an abnormal telemetry strip should initiate a 12-lead EKG, followed by diagnosis of the arrhythmia and proper treatment.

Ventricular Fibrillation

Ventricular fibrillation is characterized on the EKG by only a wavy baseline, without any organized ventricular depolarization (Fig. 7.22). In other words, there is only a wavy baseline with no recognizable, discrete QRS complexes. This rhythm is incapable of supporting coordinated ventricular contraction, so the patient has cardiac arrest. Without defibrillation, the patient will die in minutes.

Arrhythmias with Normal Rate

Occasionally, arrhythmias can have ventricular rates that are normal, i.e., between 60 and 100 beats per minute. We have already reviewed several of these, including atrial fibrillation, atrial flutter with 4:1 AV block, PAT with block, and AIVR. Two other rhythms with rates within the normal range deserve mention. The first is nodal or low-atrial rhythm (Fig. 7.23), in which the P waves are "upside down," reflecting retrograde rather than antegrade electrical conduction in the atria. Occasionally with nodal rhythms, there is no apparent retrograde conduction, and so no P wave is present at all. If the initiating focus in the node or in the lower aspect of the atrium fires at a rate exceeding 100, the rhythm would by definition be a nodal or low-atrial tachycardia, an infrequent supraventricular tachycardia.

The other rhythm to mention that occurs with a normal rate is sinus arrhythmia. Sinus arrhythmia is nothing more than an exaggeration of the normal physiological variability of heart rate with respiration. During inspiration, the heart speeds up somewhat, and during expiration the heart slows down. This minor variability in rate is probably mediated by the carotid sinus, other baroreceptors, and/or central nervous system factors that modulate heart rate via the autonomic nervous system, principally the vagus nerve. When the RR interval of consecutive QRS complexes differs by more than 0.2 s from the preceding R-R interval in the absence of premature beats, the diagnosis of sinus arrhythmia is appropriate (Fig. 7.24). Like sinus bradycardia, sinus arrhythmia is often found in healthy young individuals with normal hearts.

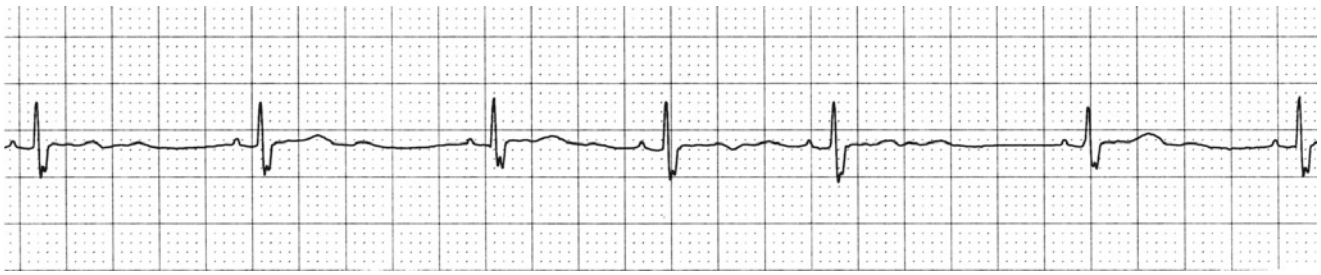


Fig. 7.24 Sinus arrhythmia

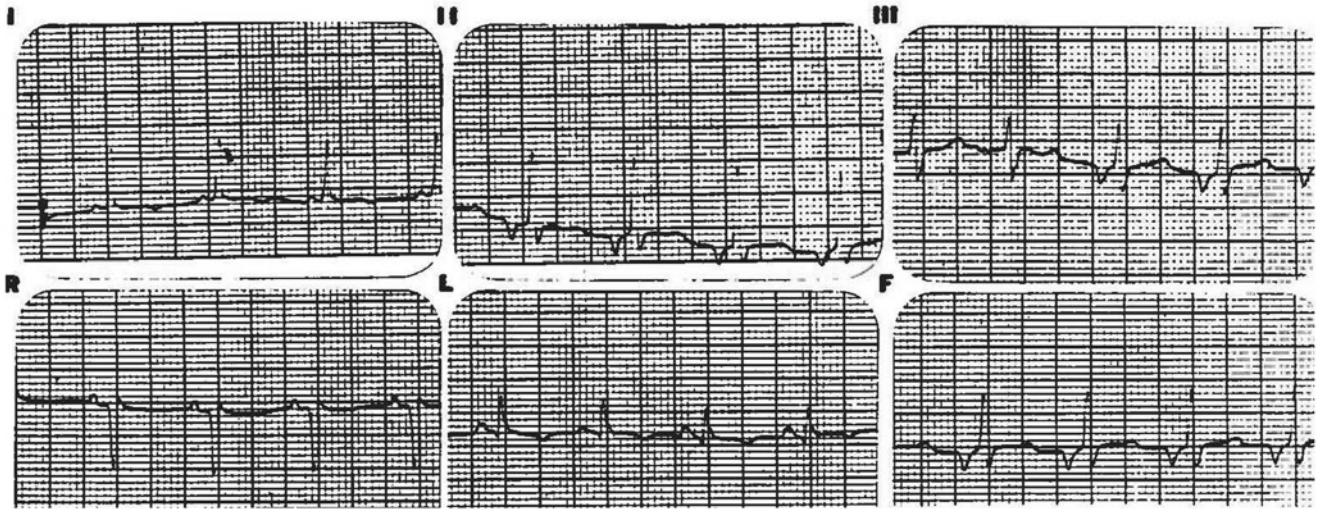
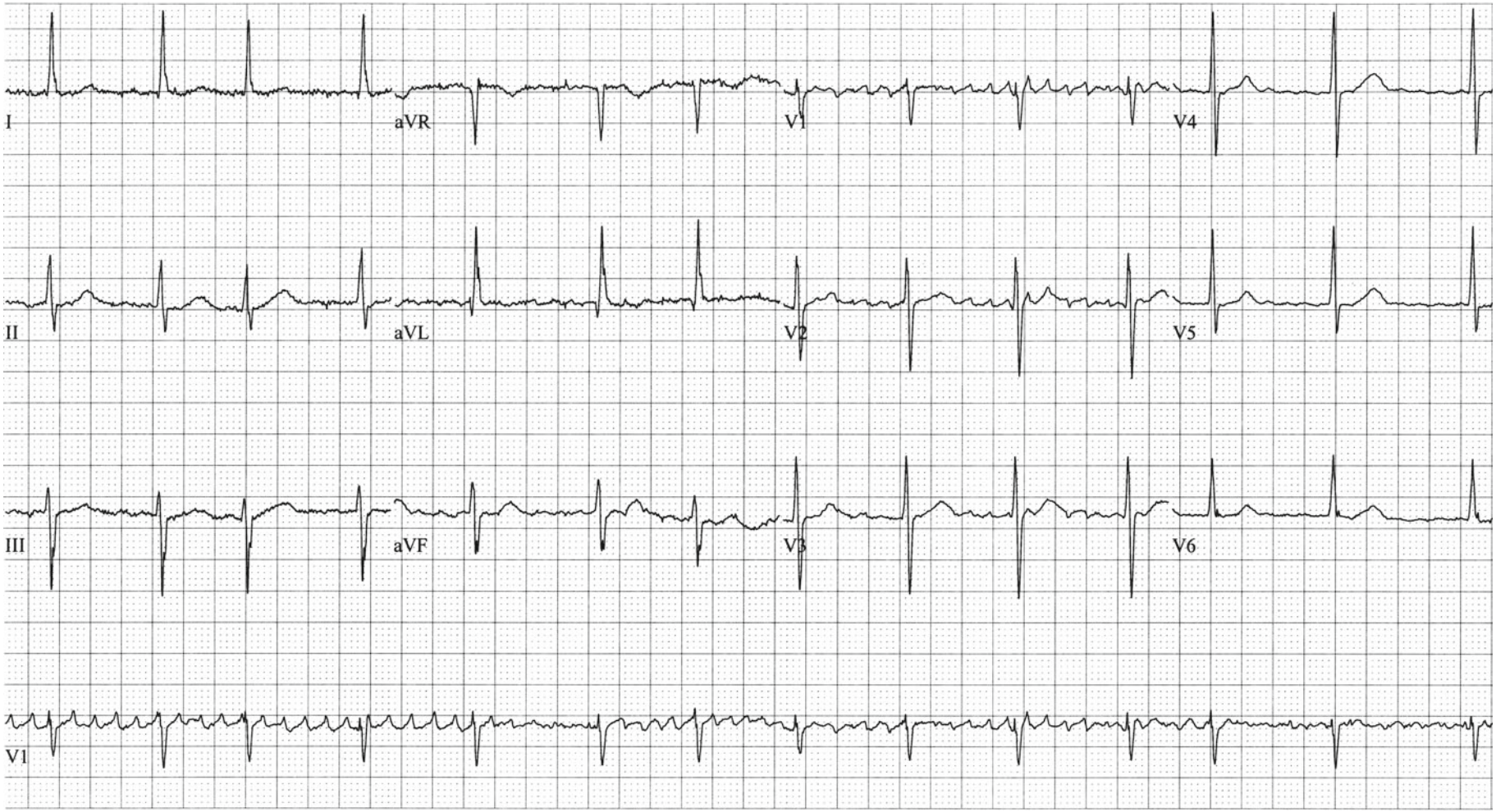


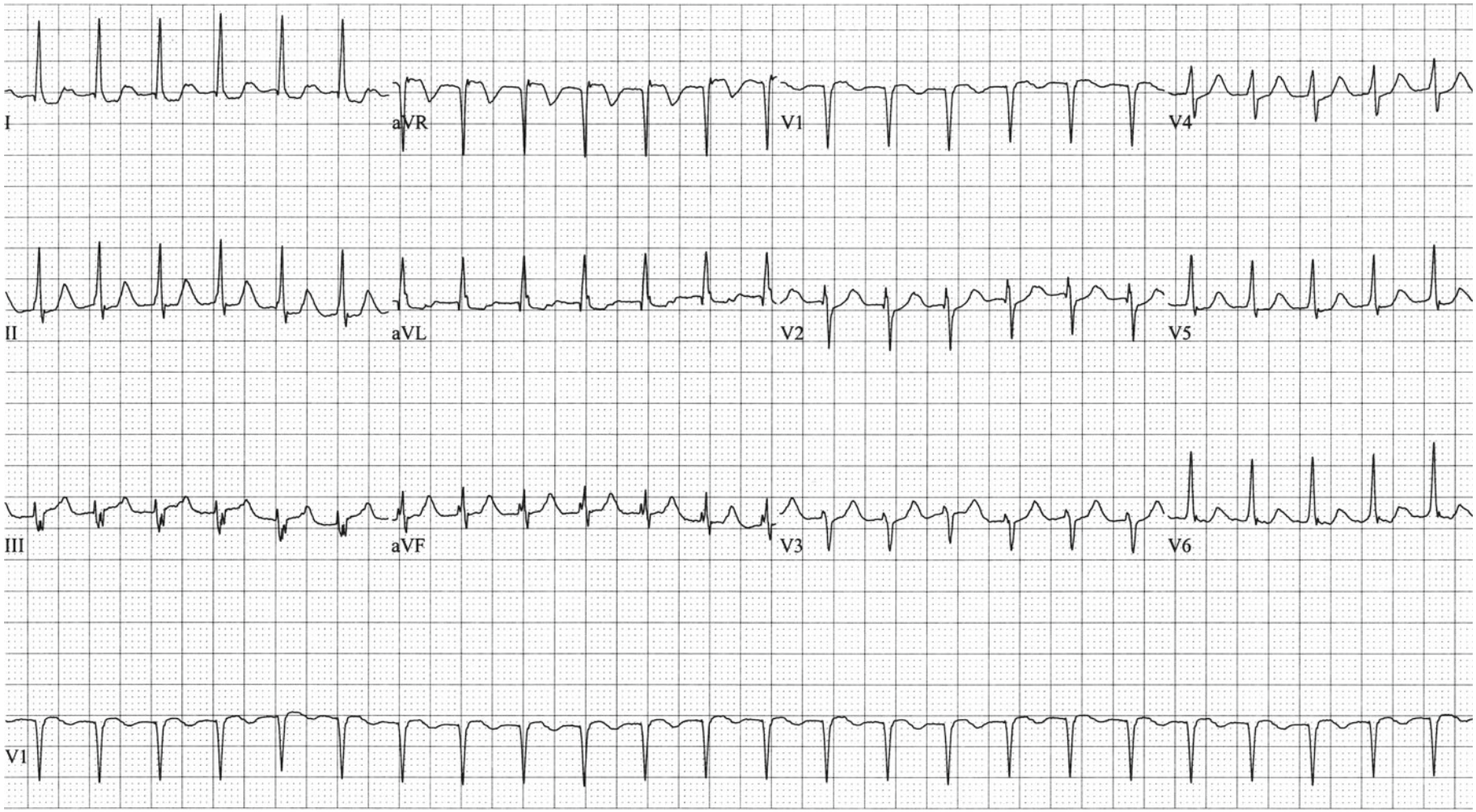
Fig. 7.23 Low-atrial or nodal rhythm. Note the inverted P waves in the inferior leads. The P wave axis is −75°

Exercise Tracings

Exercise Tracing 7.1

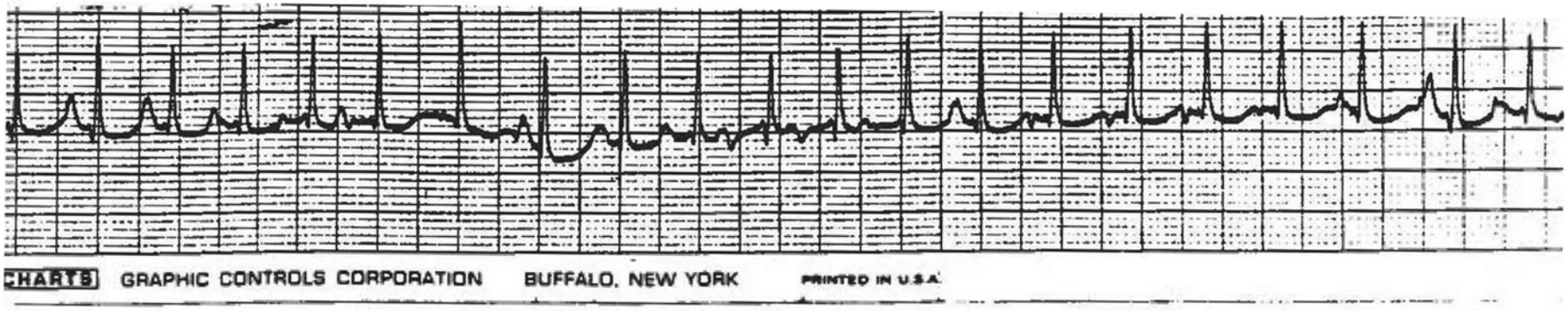


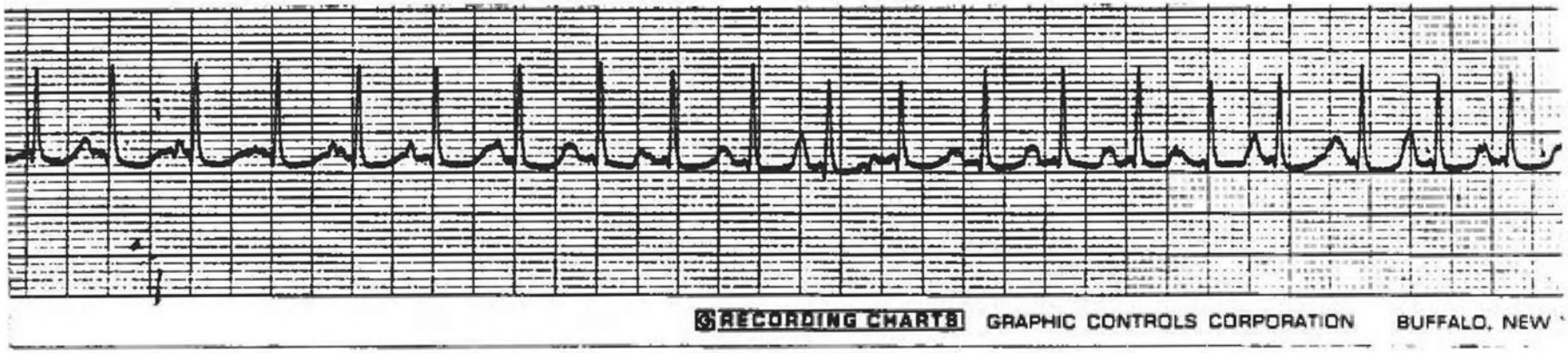
Exercise Tracing 7.2

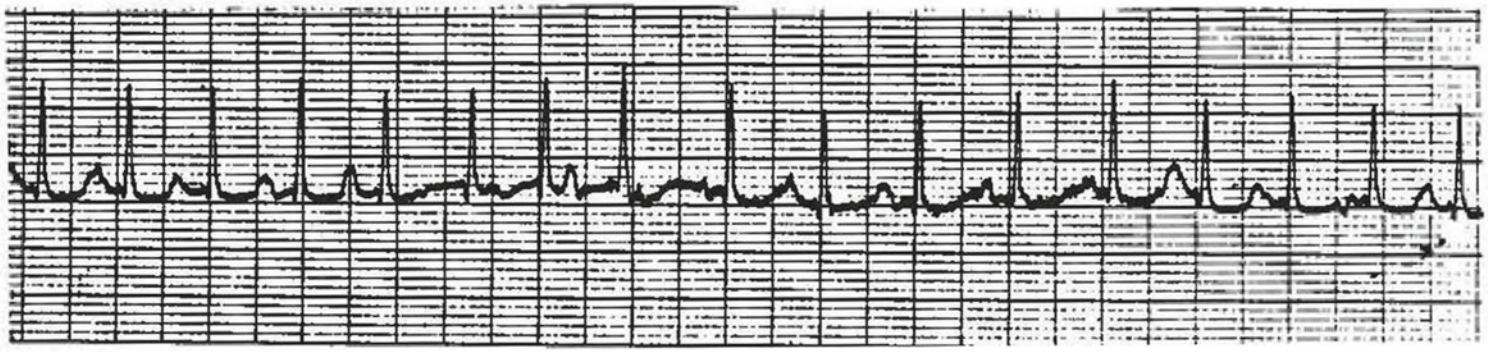


Rhythm Strip Only

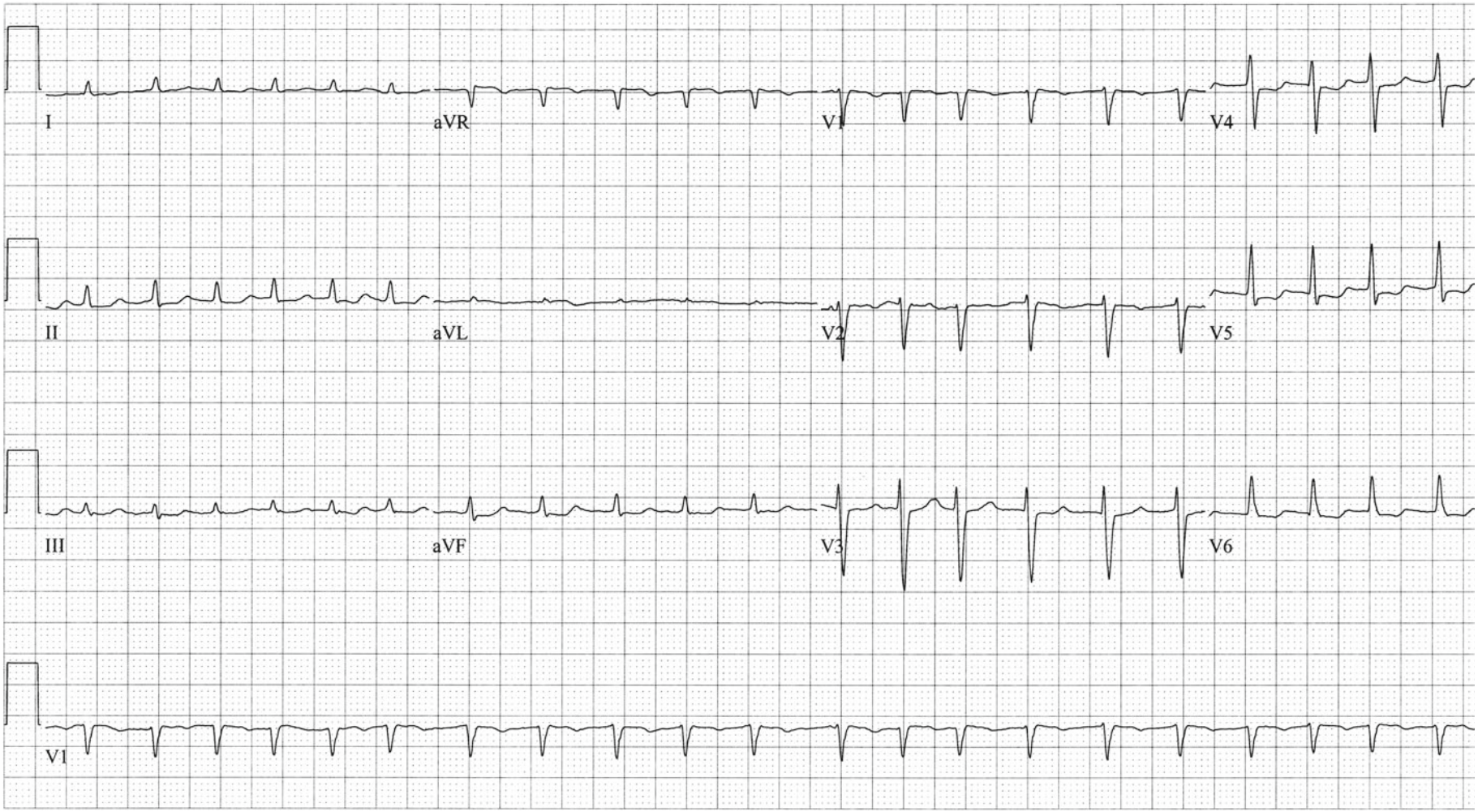
Exercise Tracing 7.3



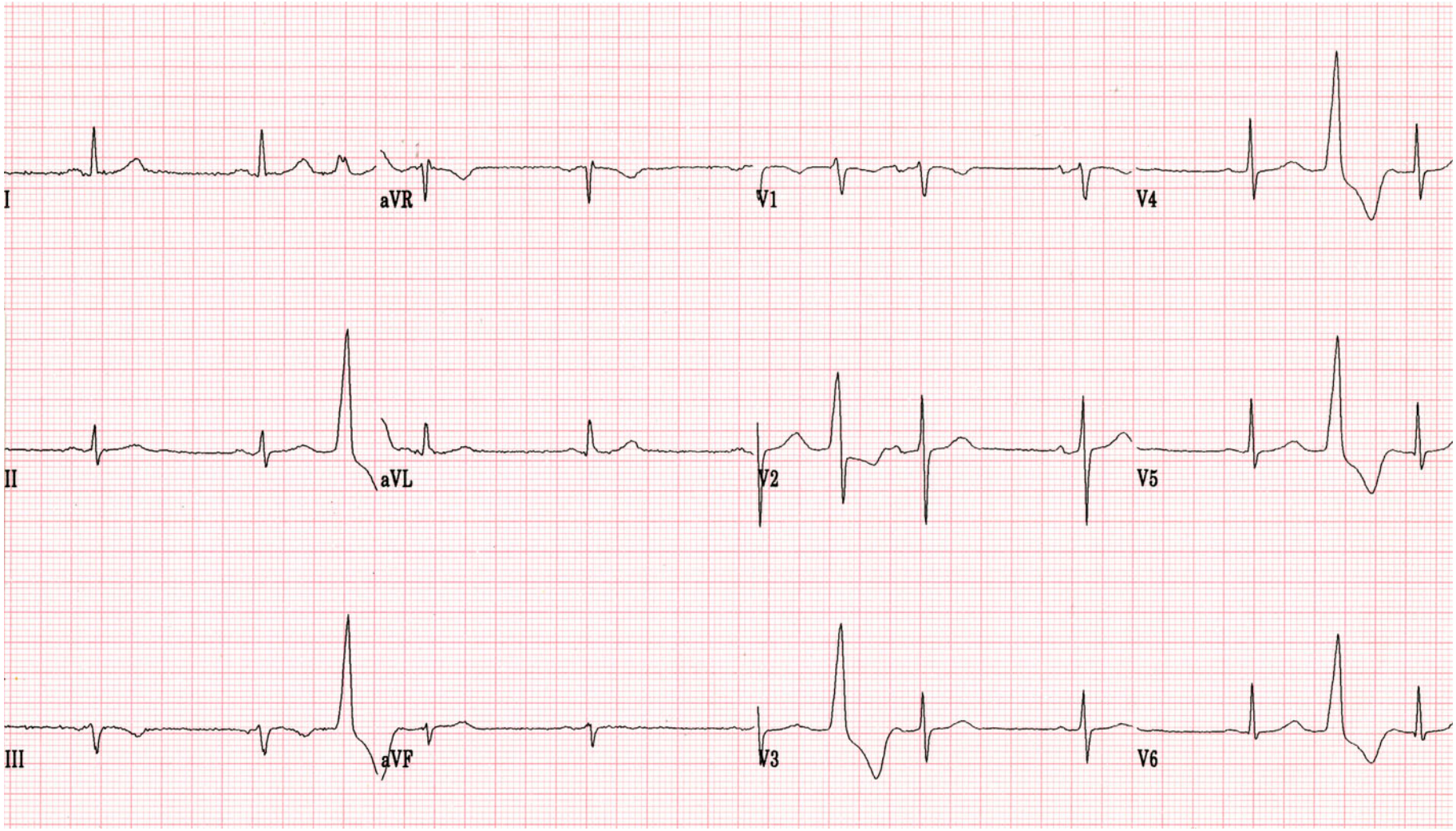




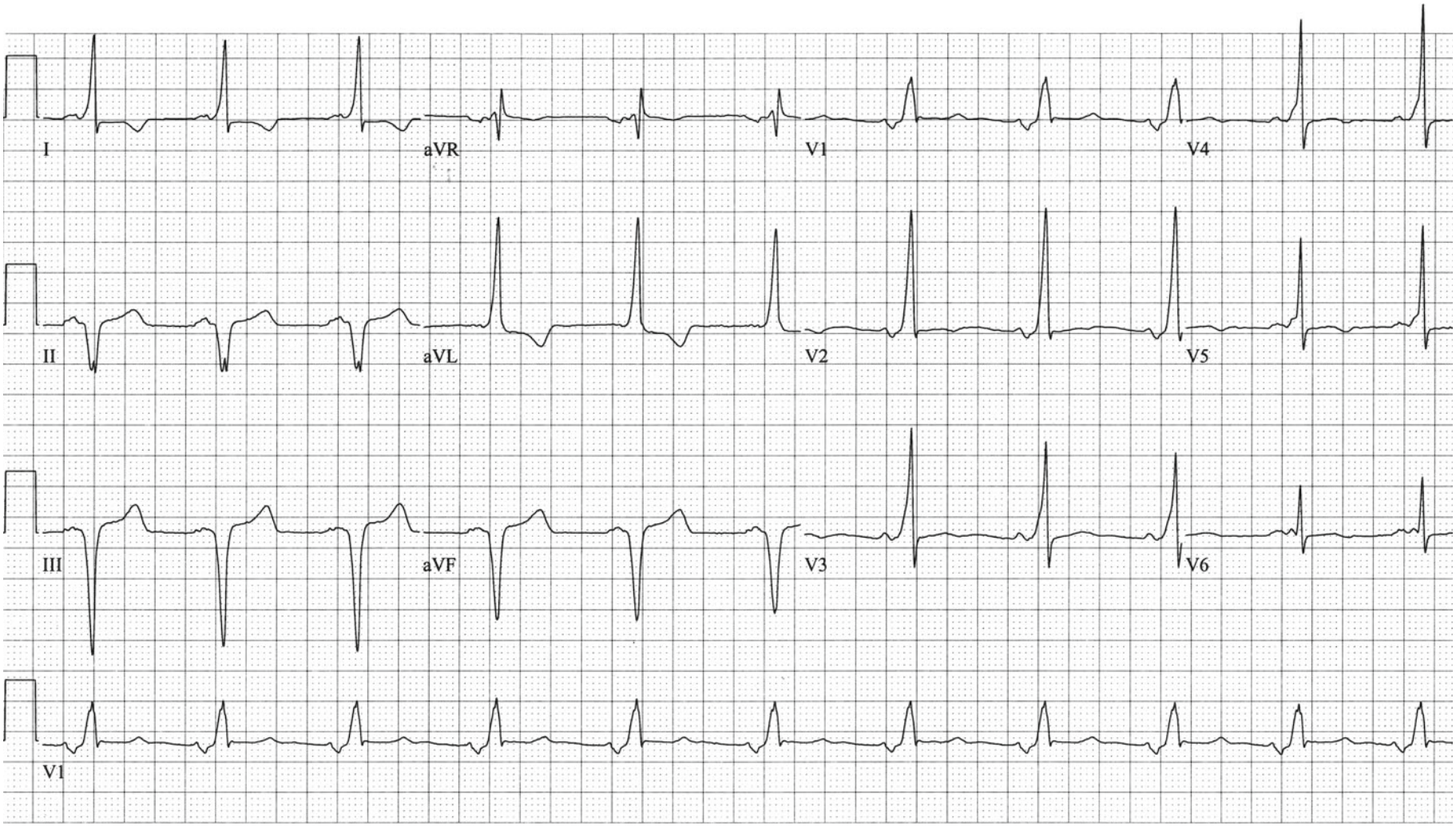
Exercise Tracing 7.4



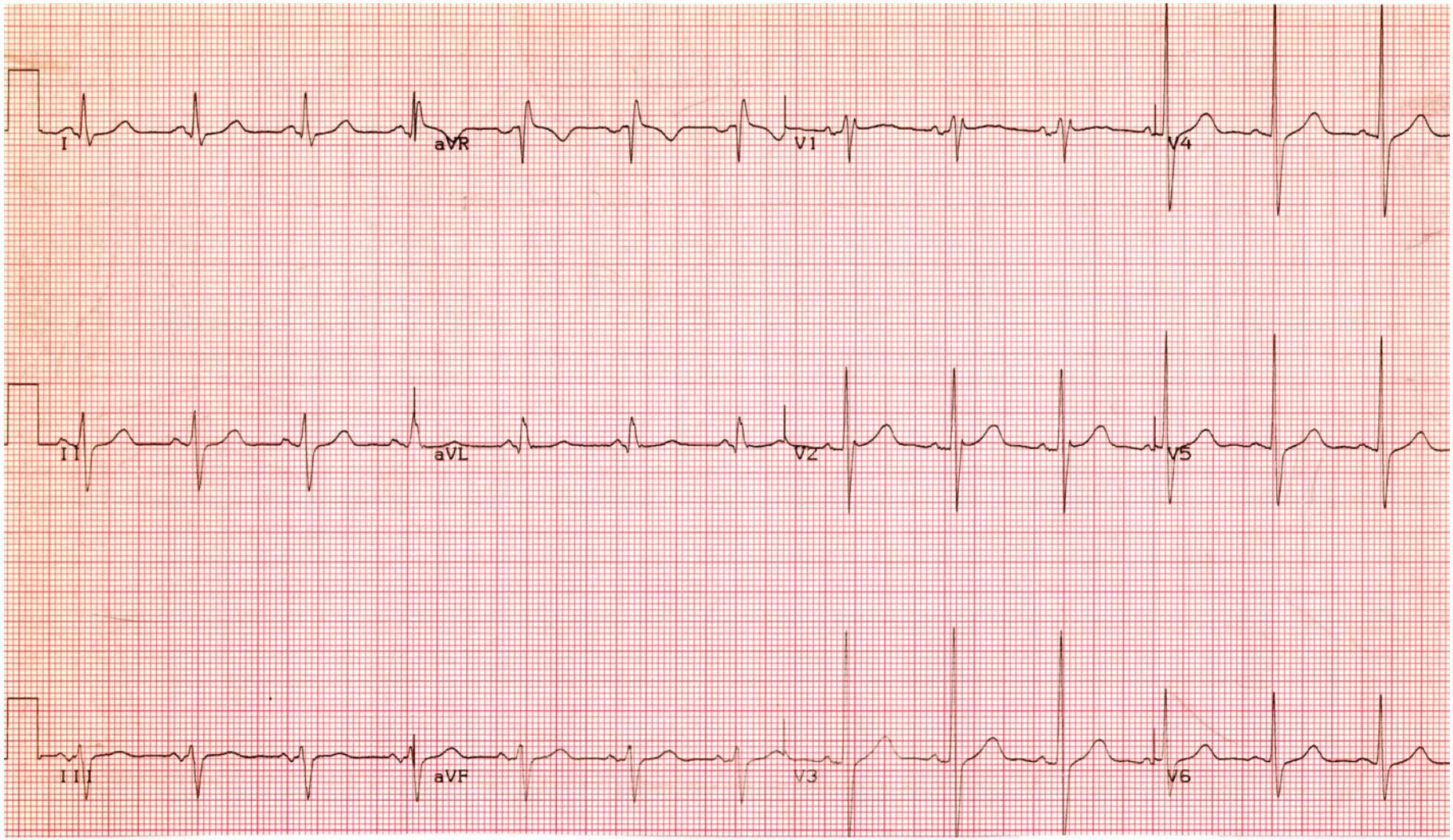
Exercise Tracing 7.5



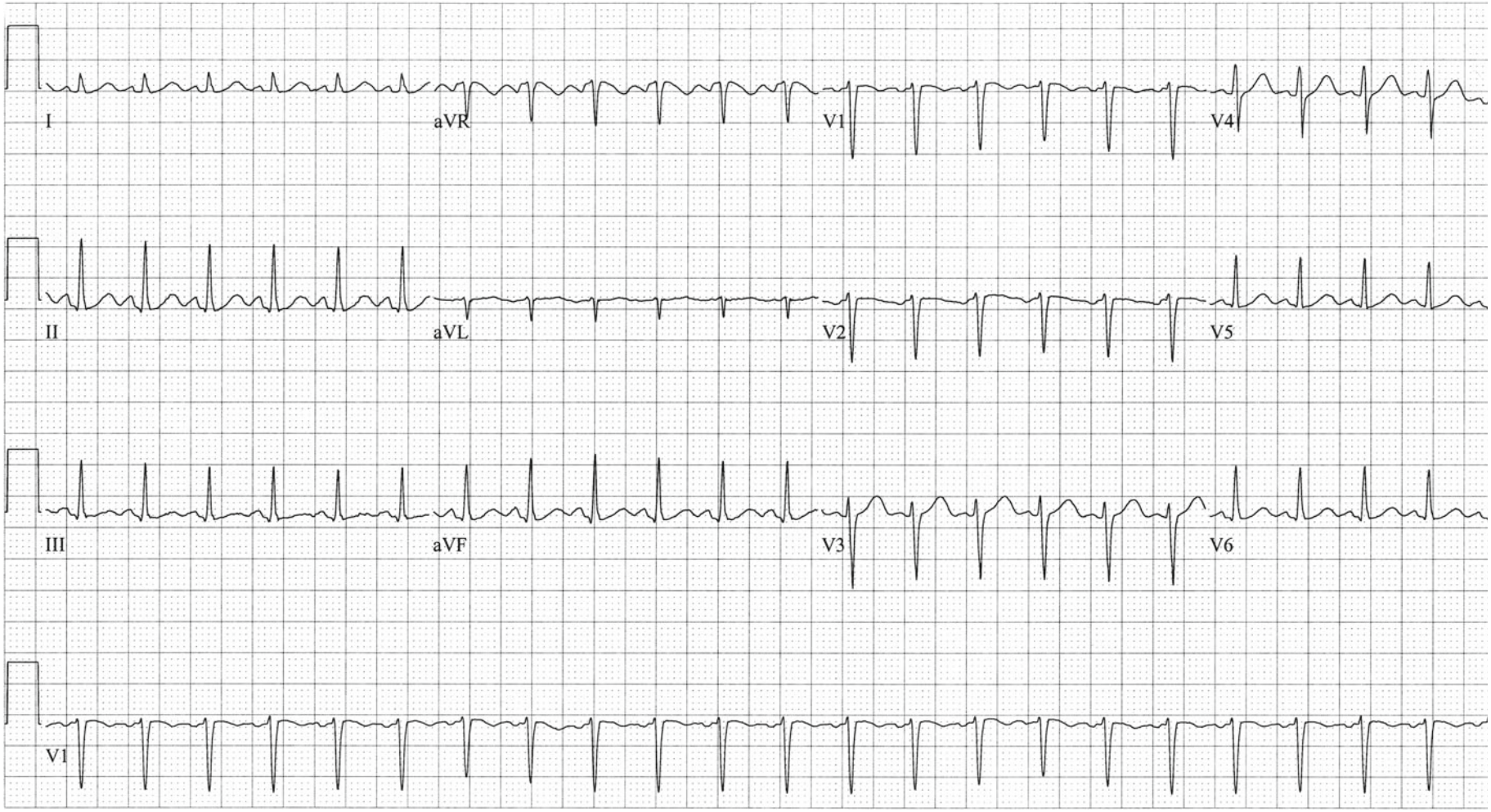
Exercise Tracing 7.6



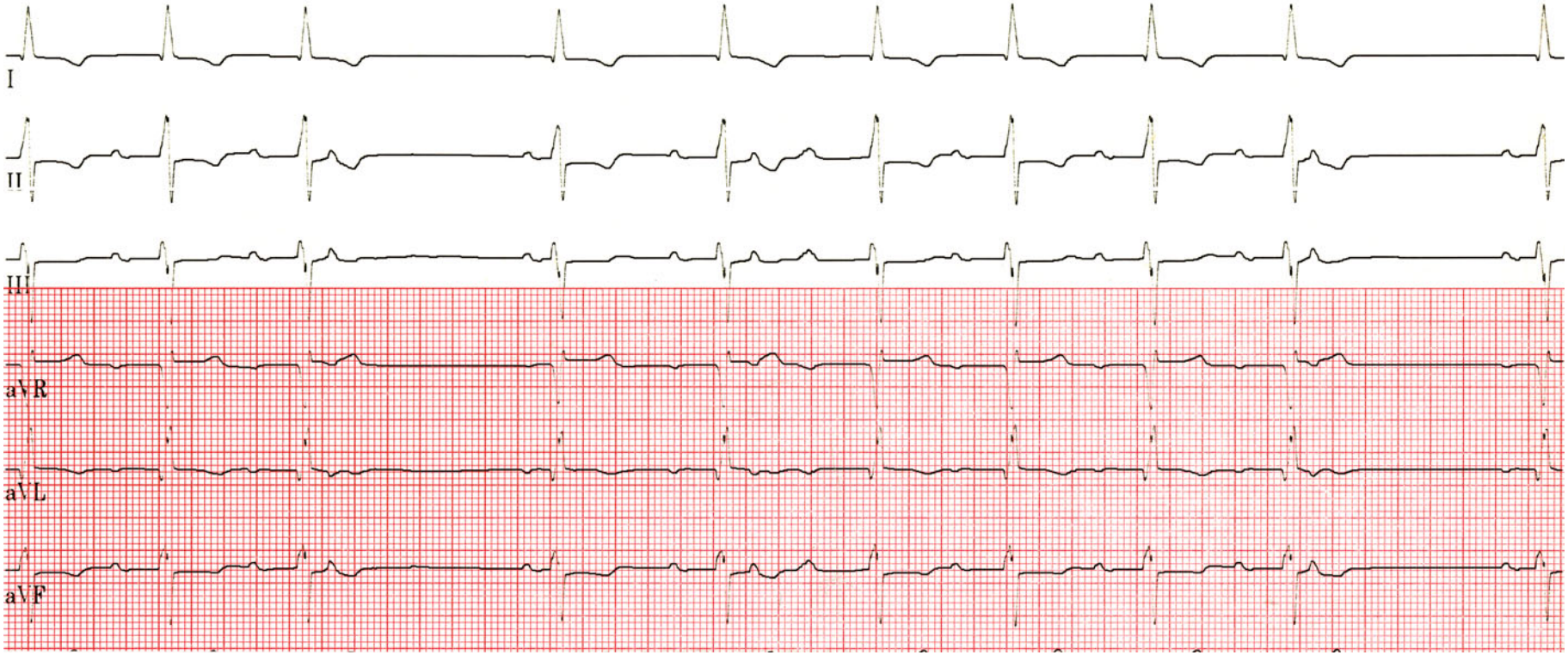
Exercise Tracing 7.7



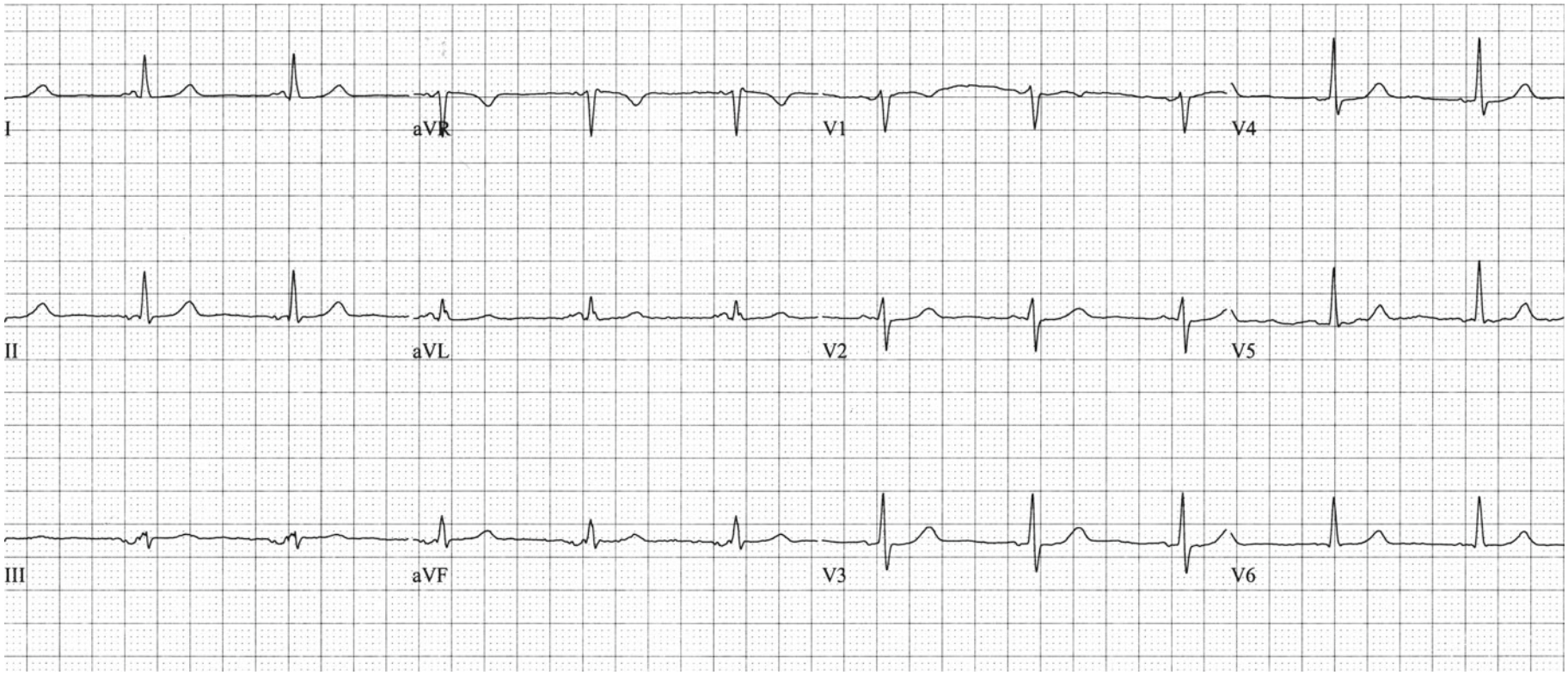
Exercise Tracing 7.8



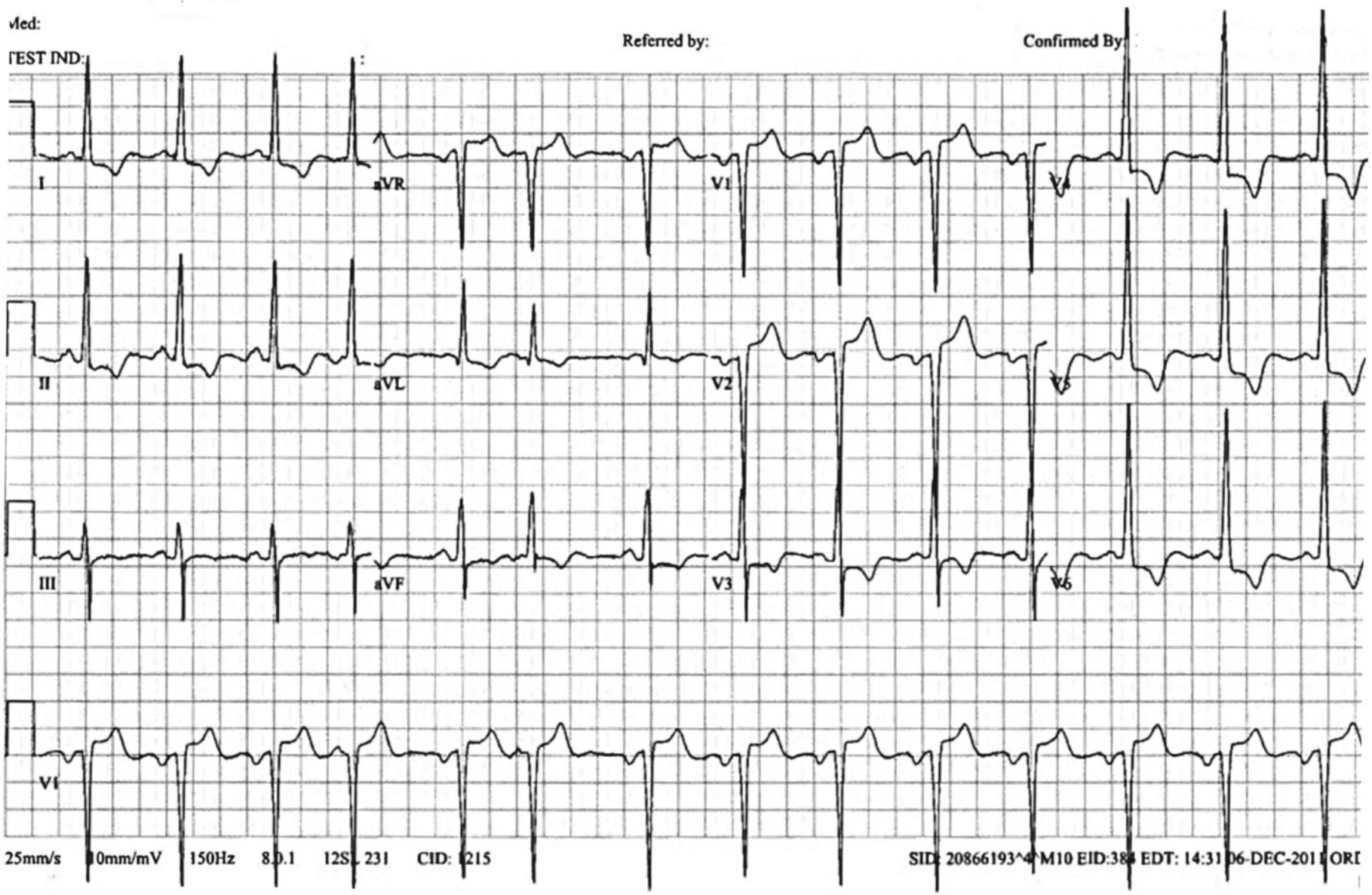
Exercise Tracing 7.9



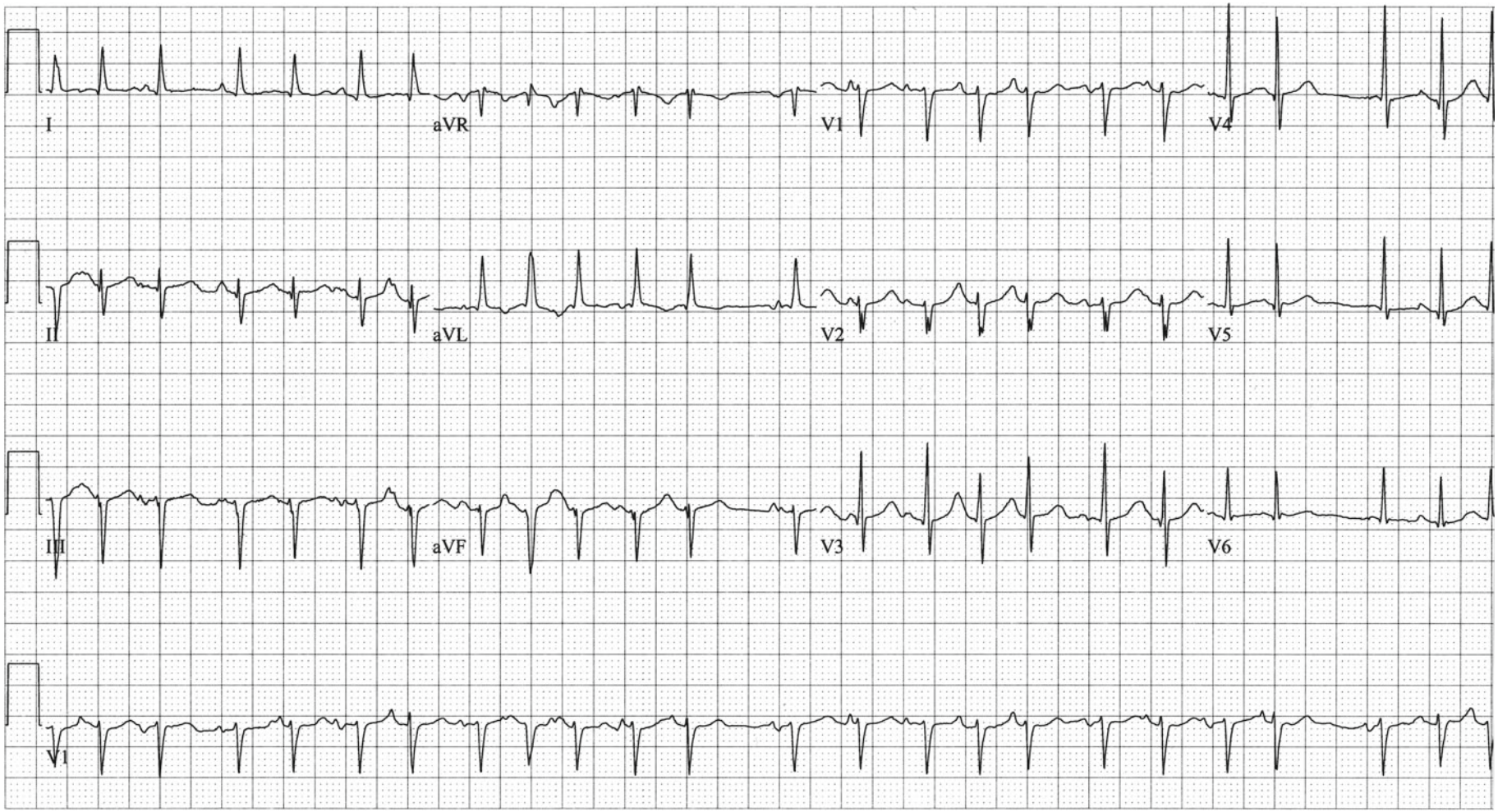
Exercise Tracing 7.10



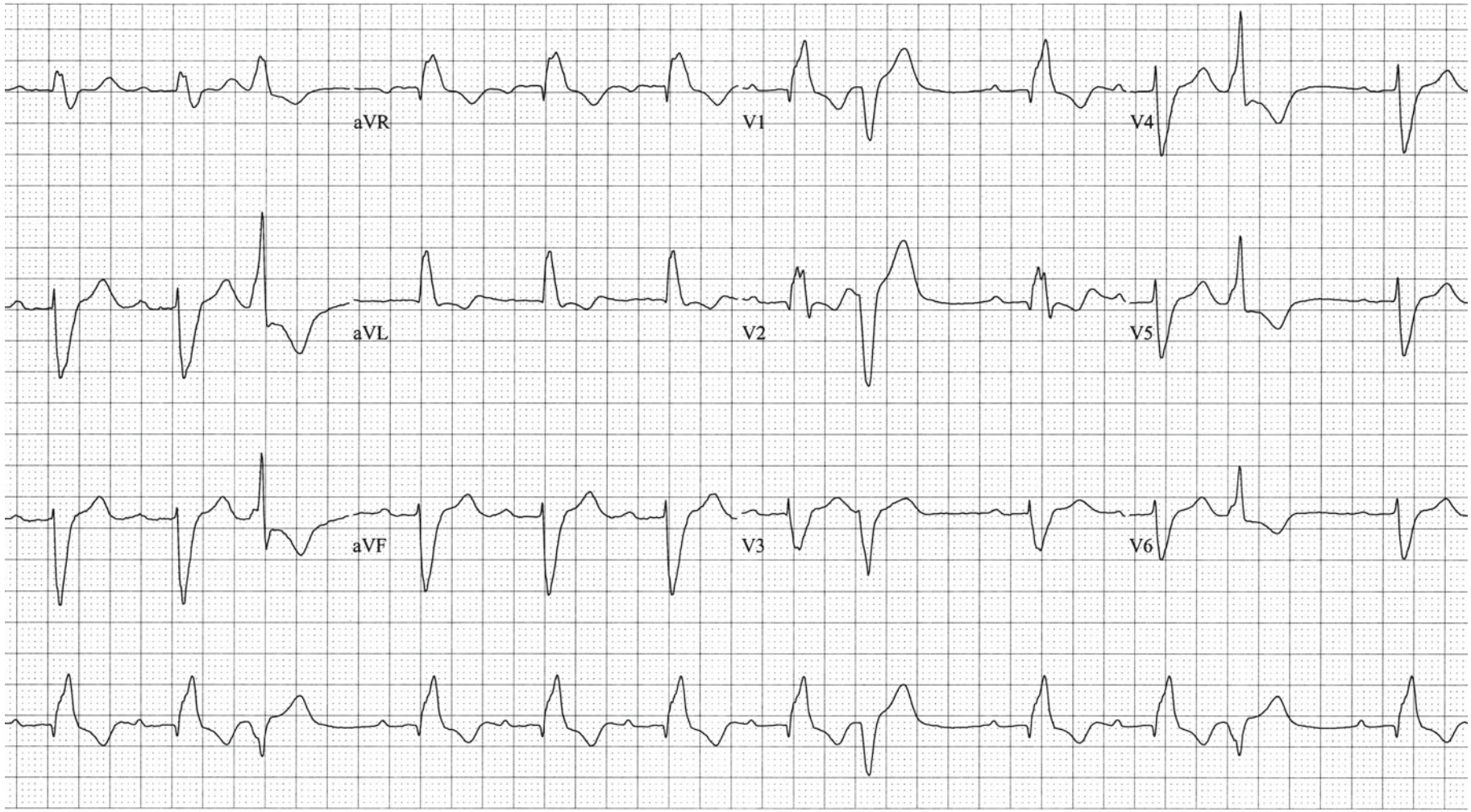
Exercise Tracing 7.11



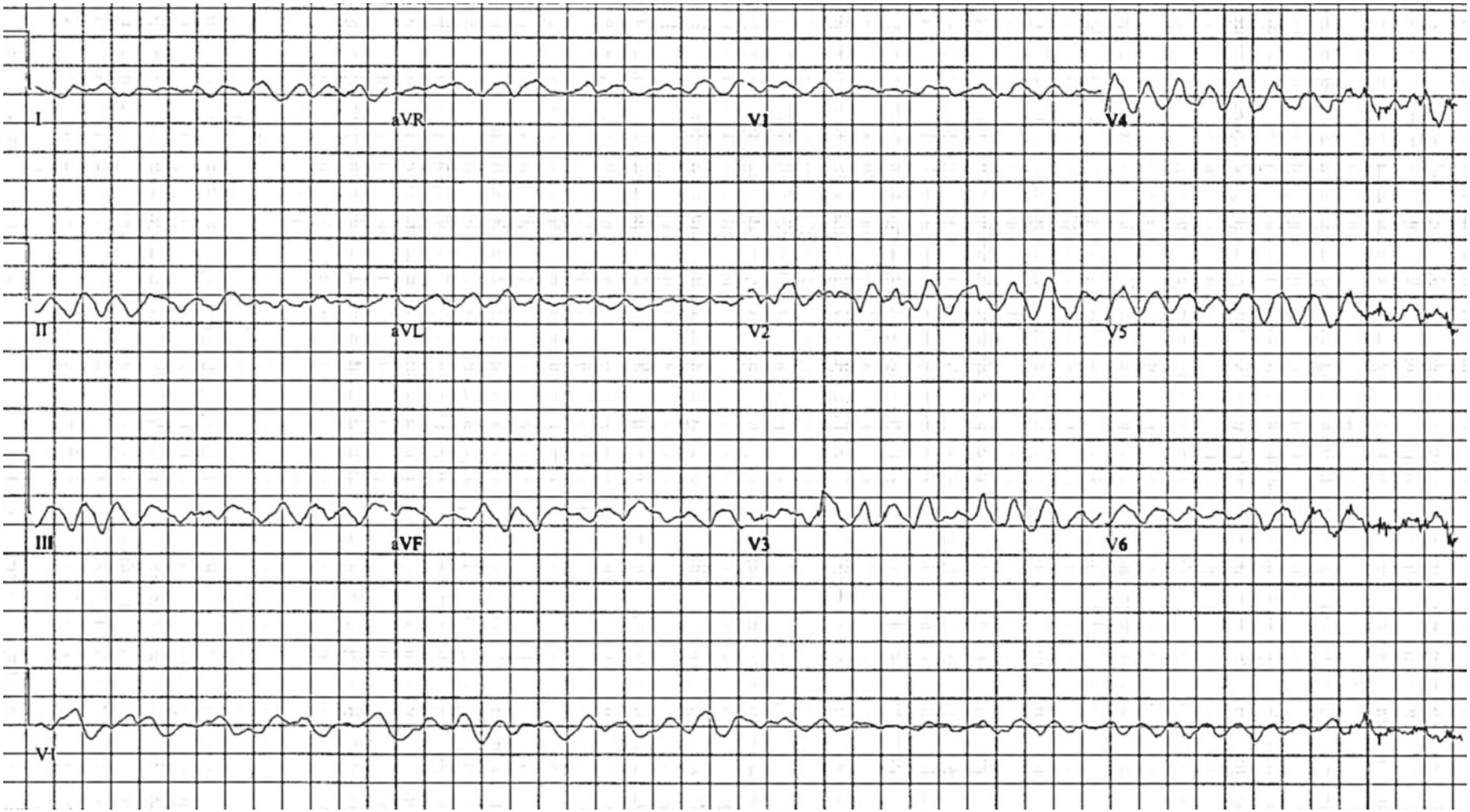
Exercise Tracing 7.12



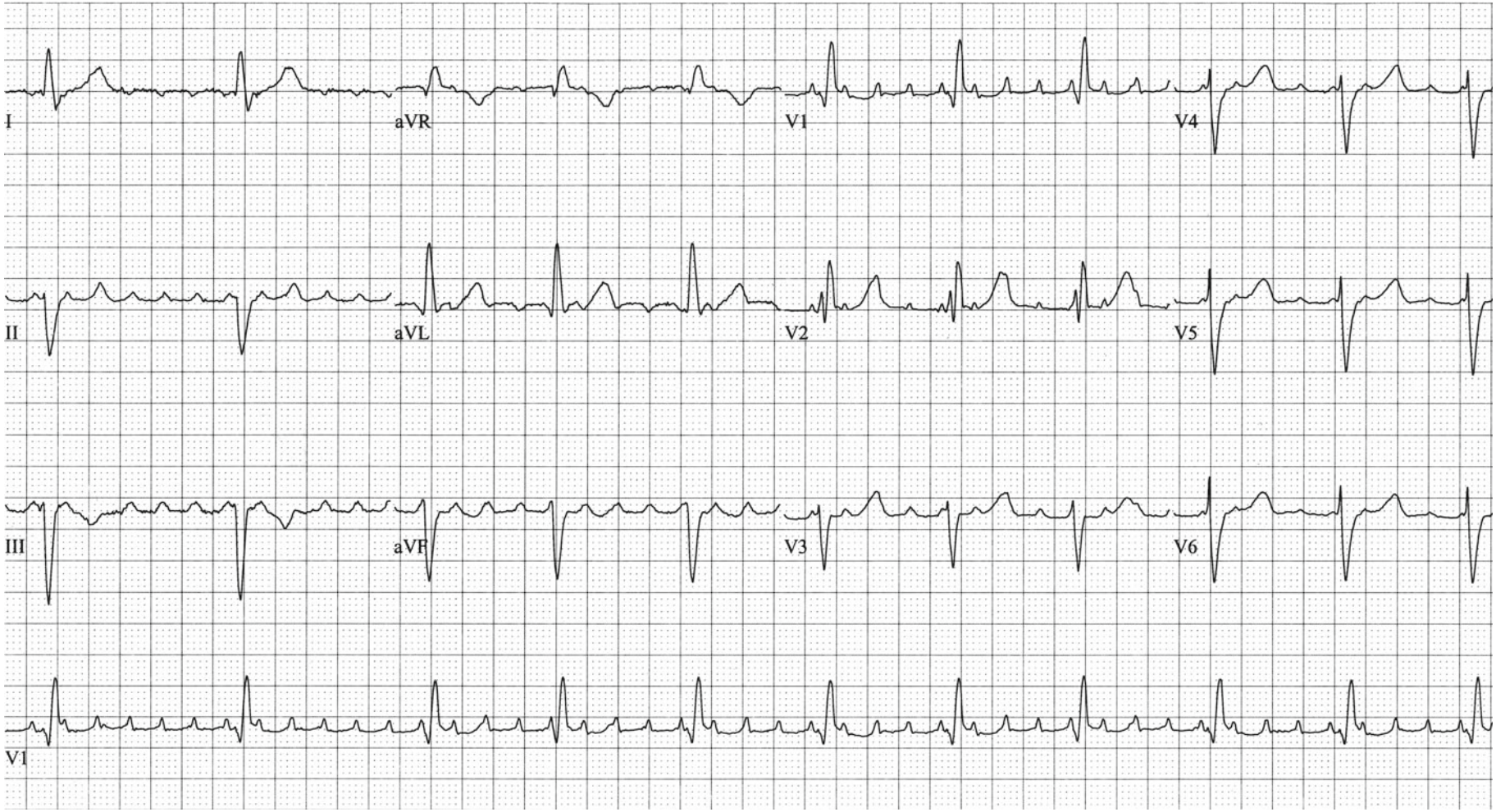
Exercise Tracing 7.13



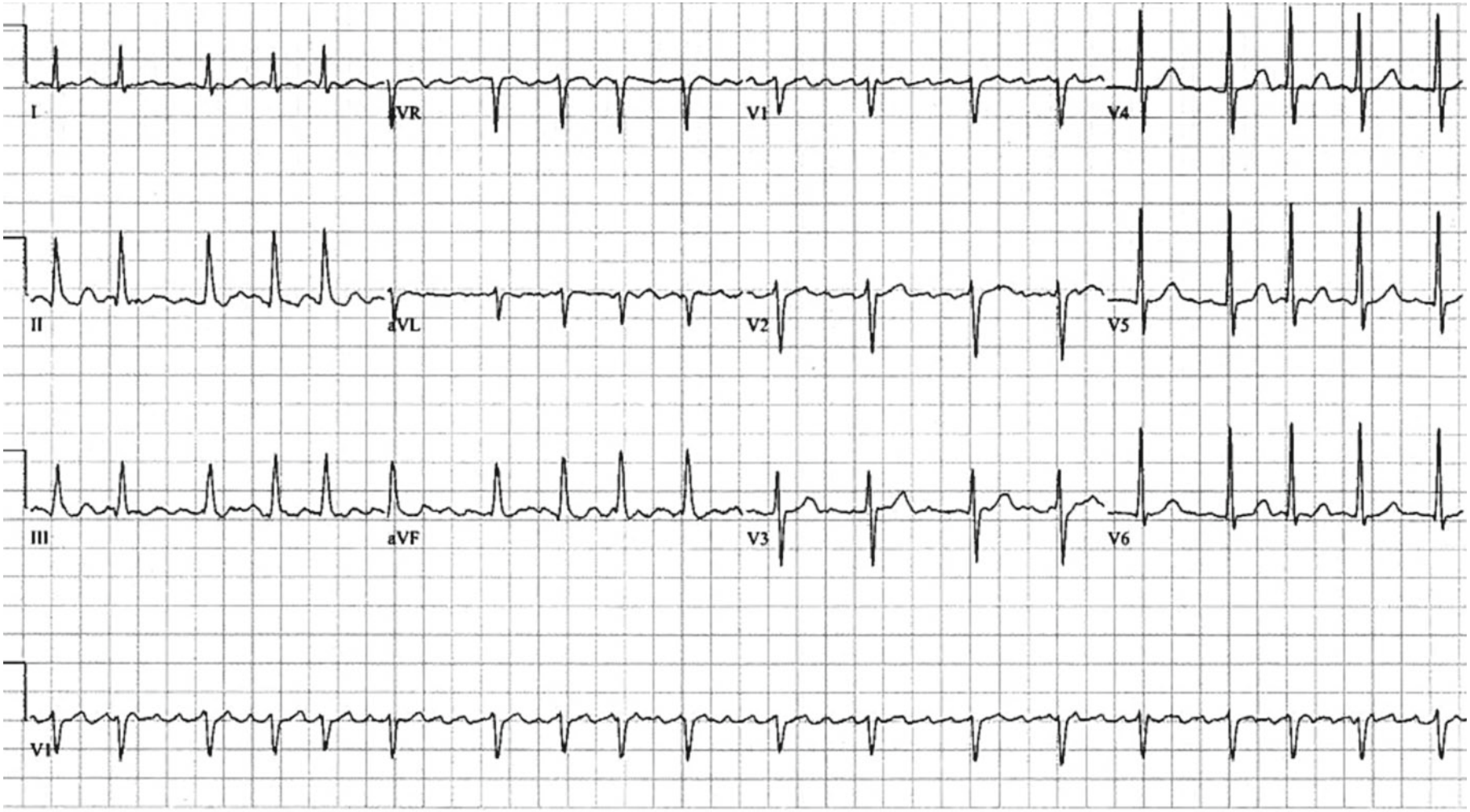
Exercise Tracing 7.14



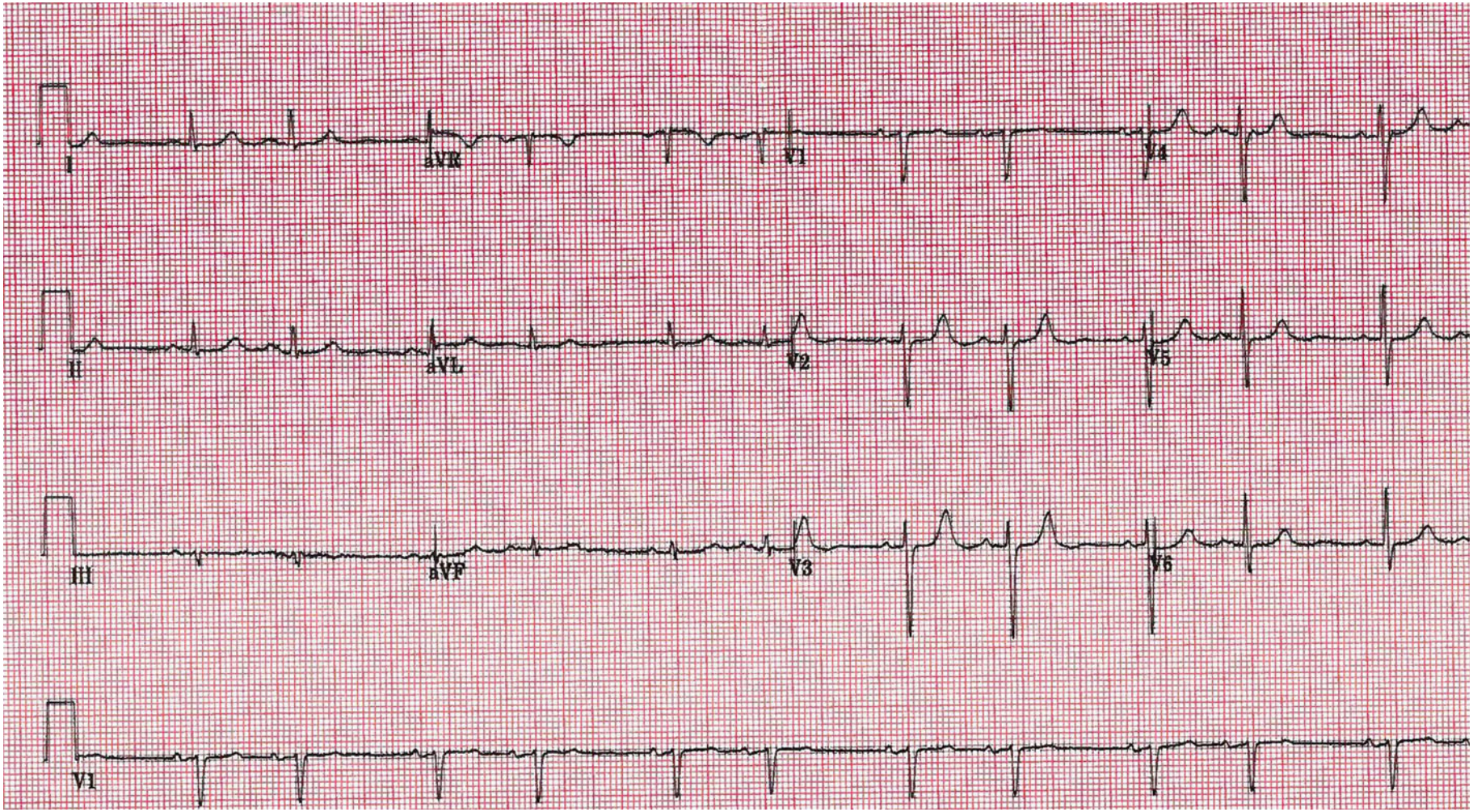
Exercise Tracing 7.15



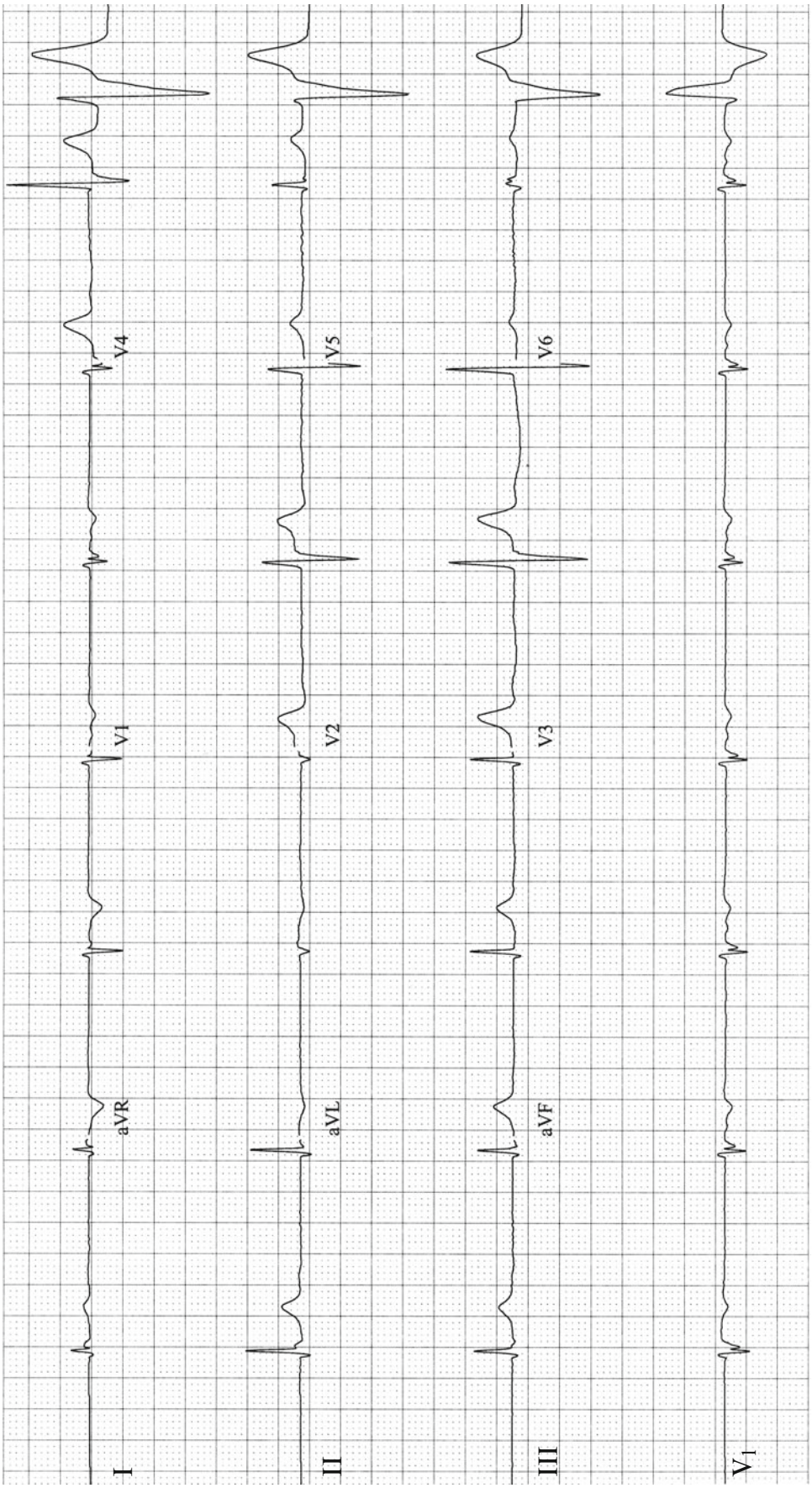
Exercise Tracing 7.16



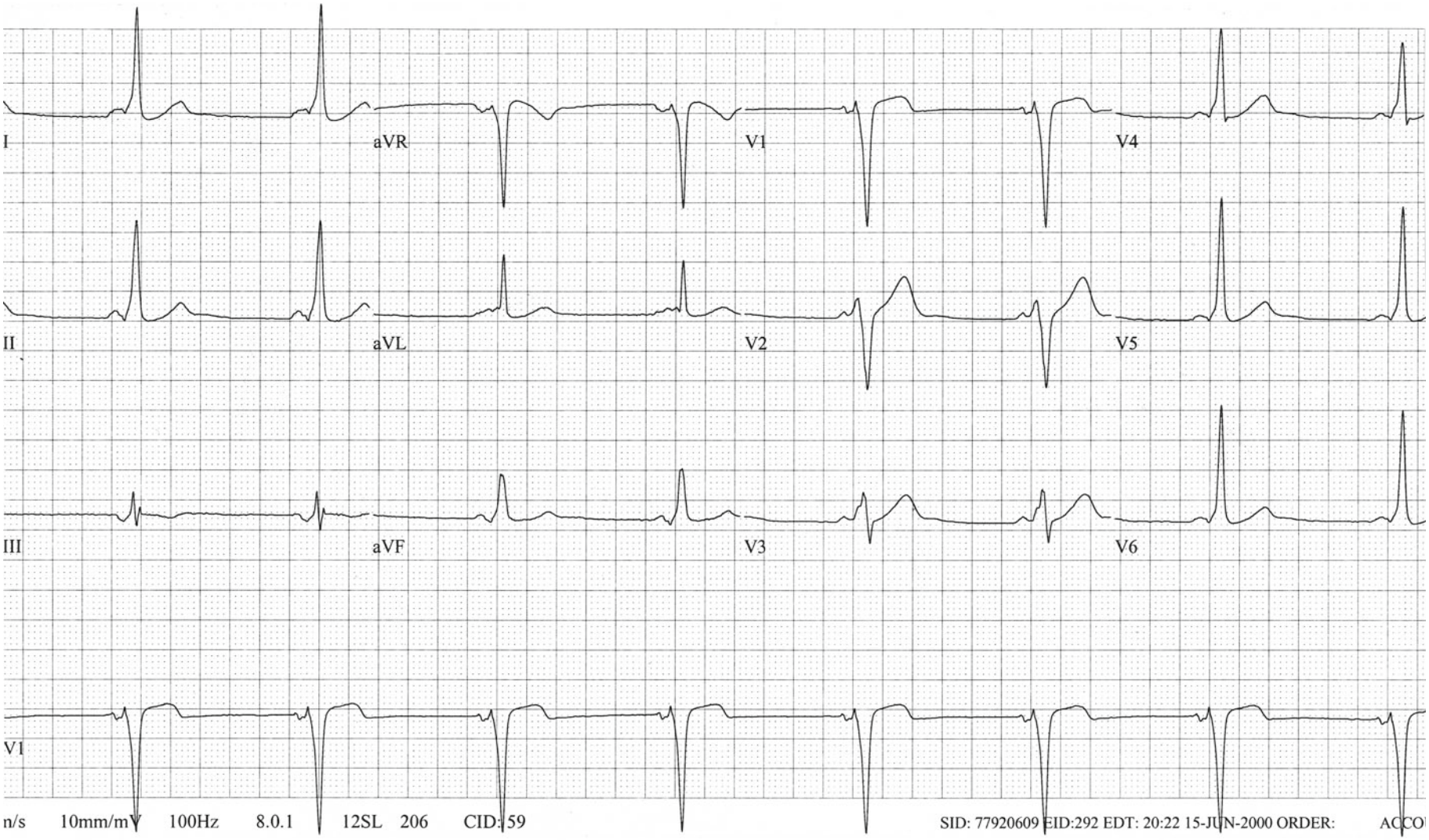
Exercise Tracing 7.17



Exercise Tracing 7.18



Exercise Tracing 7.19



Interpretations of Exercise Tracings

Exercise Tracing 7.1	
RATE:	A 460 V 85
RHYTHM:	Atrial fibrillation with controlled ventricular response
AXIS:	-10°
INTERVALS:	PR --- QRS 0.07 QT 0.35
WAVEFORM:	Unremarkable
SUMMARY:	Abnormal due to atrial fibrillation

Exercise Tracing 7.2	
RATE:	A ? V 154
RHYTHM:	Paroximal atrial tachycardia
AXIS:	+10°
INTERVALS:	PR --- QRS 0.08 QT 0.28
WAVEFORM:	Unremarkable
SUMMARY:	Abnormal due to paroxymal atrial tachycardia (confirmed when vagal maneuvers converted rhythm to normal sinus at rate of 87 beats per minute)

Exercise Tracing 7.3	
RATE:	A 140 V 140
RHYTHM:	Multifocal atrial tachycardia
SUMMARY:	Abnormal—multifocal atrial tachycardia

Exercise Tracing 7.4	
RATE:	A ?--no discrete atrial activity detected V 137
RHYTHM:	Atrial fibrillation with rapid ventricular response
AXIS:	+50°
INTERVALS:	PR --- QRS 0.07 QT 0.31
WAVEFORM:	Low voltage QRS, delayed R wave progression V_{1-4}
SUMMARY:	Abnormal due to atrial fibrillation, low voltage QRS, nonspecific ST-T changes

Exercise Tracing 7.5	
RATE:	A 54 V 75
RHYTHM:	Sinus bradycardia with frequent, interpolated PVCs
AXIS:	-20°
INTERVALS:	PR 0.16 QRS 0.09 QT 0.39 (PR longer after PVCs)
WAVEFORM:	Unremarkable
SUMMARY:	Abnormal due to frequent interpolated PVCs and concealed retrograde conduction, leftward axis deviation

Exercise Tracing 7.6	
RATE:	A 68 V 68
RHYTHM:	Normal sinus rhythm
AXIS:	-60°
INTERVALS:	PR 0.13 QRS 0.13 QT 0.44
WAVEFORM:	Mostly negative P in V_1, biphasic P in V_2. Q in II, III and aVF; delta waves, especially I and V_{3-4}; tall R waves V_{1-3} but not in II
SUMMARY:	Abnormal due to nonspecific ST-T changes, left atrial abnormality, Wolff-Parkinson-White syndrome, Type A (because V_1 is positive), ”pseudoinfarction” appearance of inferior leads (related to WPW)

Exercise Tracing 7.7	
RATE:	A 83 V 83
RHYTHM:	Normal sinus rhythm
AXIS:	-45°
INTERVALS:	PR 0.14 QRS 0.10 QT 0.38
WAVEFORM:	Early R wave development V_{2-3}; q in I, r in III; rSr' in V_1
SUMMARY:	Abnormal due to early R wave development V_{2-3}, left anterior hemiblock, incomplete right bundle branch block

Exercise Tracing 7.8	
RATE:	A 146 V 146
RHYTHM:	Sinus tachychardia
AXIS:	+75°
INTERVALS:	PR 0.12 QRS 0.08 QT 0.28
WAVEFORM:	Inverted P wave in V_{1-2}, delayed R wave progression V_{1-4}
SUMMARY:	Abnormal due to sinus tachycardia, left atrial abnormality

Exercise Tracing 7.9 (limb leads only)	
RATE:	A 80 V 60
RHYTHM:	Normal sinus rhythm with frequent non-conducted PACs
AXIS:	-20°
INTERVALS:	PR 0.30 QRS 0.10 QT 0.41
WAVEFORM:	Non-conducted P waves in the ST segments following the 3rd, 5th and 9th QRS
SUMMARY:	Abnormal due to rhythm as described, leftward axis deviation, first degree AV block

Exercise Tracing 7.10	
RATE:	A 67 V 67
RHYTHM:	Nodal or low-atrial rhythm
AXIS:	+30
INTERVALS:	PR 0.14 QRS 0.08 QT 0.38
WAVEFORM:	Inverted P in II, III, aVF; early R wave transition V_{2-3}
SUMMARY:	Abnormal due to rhythm as described, early R wave transition V_{2-3}

Exercise Tracing 7.11	
RATE:	A 85 V 85
RHYTHM:	Sinus rhythm with occasional PACs (last beat in I, second beat in aVR)
AXIS:	+15°
INTERVALS:	PR 0.16 QRS 0.09 QT 0.34
WAVEFORM:	$RV_5 + SV_1$ >35 mm, ST depression and T wave inversion in leads I, II, aVL, aVF, $V_{3–6}$; inverted P waves in $V_{1–2}$
SUMMARY:	Abnormal due to left ventricular hypertrophy with strain, left atrial abnormality, leftward axis deviation, occasional PACs

Exercise Tracing 7.12	
RATE:	A 150 V 150
RHYTHM:	Multifocal atrial tachycardia
AXIS:	-40°
INTERVALS:	PR var. QRS 0.07 QT 0.30
WAVEFORM:	Tall R waves V_3, q in I, r in III
SUMMARY:	Abnormal due to multifocal atrial tachycardia, left anterior hemiblock, early R wave transition $V_{2–3}$

Exercise Tracing 7.13	
RATE:	A 83 V 83
RHYTHM:	Sinus rhythm with frequent PVCs
AXIS:	–70°
INTERVALS:	PR 0.27 QRS 0.17 QT 0.44
WAVEFORM:	q in V_{1-2}, broad S in I and V_6, tall R in V_1
SUMMARY:	Abnormal due to right bundle branch block, left anterior hemiblock, and prolonged PR interval compatible with (but not diagnostic of) trifascicular block; frequent PVCs; possible old anteroseptal infarction

Exercise Tracing 7.14	
RATE:	A --- V 182
RHYTHM:	Ventricular fibrillation
AXIS:	–
INTERVALS:	PR --- QRS --- QT ---
WAVEFORM:	No recognizable QRS complexes
SUMMARY:	Abnormal due to ventricular fibrillation

Exercise Tracing 7.15	
RATE:	A 268 V 65
RHYTHM:	Atrial flutter with variable block (4:1, 6:1)
AXIS:	-70°
INTERVALS:	PR --- QRS 0.13 QT 0.41
WAVEFORM:	rsR' in V_{1-2}, terminal S in I and V_6
SUMMARY:	Abnormal due to rhythm as described, right bundle branch block and left anterior hemiblock

Exercise Tracing 7.16	
RATE:	A 375–500 V 114
RHYTHM:	Atrial fibrillation
AXIS:	+75°
INTERVALS:	PR --- QRS 0.10 QT 0.30
WAVEFORM:	Normal
SUMMARY:	Abnormal due to atrial fibrillation

Exercise Tracing 7.17	
RATE:	A 63–83 V 63–83
RHYTHM:	Atrial bigeminy
AXIS:	+10°
INTERVALS:	PR 0.17 QRS 0.08 QT 0.40
WAVEFORM:	Borderline low-voltage QRS complexes, delayed R wave progression $V_{1–4}$
SUMMARY:	Abnormal due to atrial bigeminy, borderline low-voltage QRS complexes, delayed R wave progression $V_{1–4}$

Exercise Tracing 7.18	
RATE:	A 0 V 44
RHYTHM:	Sinus arrest (no P waves or other evidence of atrial depolarization) with nodal escape rhythm and a PVC at the end of the tracing
AXIS:	+70°
INTERVALS:	PR --- QRS 0.08 QT 0.40
WAVEFORM:	Q wave only in V_6, ? significance
SUMMARY:	Abnormal due to sinus arrest, nodal escape, and one PVC

Exercise Tracing 7.19	
RATE:	A 49 V 49
RHYTHM:	Sinus bradycardia
AXIS:	+35°
INTERVALS:	PR 0.11 QRS 0.13 QT 0.47
WAVEFORM:	Delta waves I, II, aVR, aVF, $V_{3–6}$; QRS negative in V_1
SUMMARY:	Abnormal due to sinus bradycardia and Wolff–Parkinson–White syndrome, Type B

References

1. Bar FW, Brugada P, Dassen WRM, Wellens HJJ. Differential diagnosis of tachycardia with narrow QRS complex (shorter than 0.12 second). Am J Cardiol. 1984;54:555–60.
2. Link MS. Evaluation and initial treatment of supraventricular tachycardia. N Engl J Med. 2012;367:1438–48.

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