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CHAPTER 1

Complexes and intervals

An *electrocardiogram* (ECG) is a recording of cardiac electrical activity made from the body surface and displayed on graph paper scored horizontally and vertically in 1 millimeter (mm) increments. Each millimeter on the horizontal axis represents 40 milliseconds (0.04 second) of elapsed time and each millimeter on the vertical axis represents 0.1 millivolt (mV) of electrical force. Each 5 millimeter mark on the paper is scored with a heavier line representing 200 milliseconds (msec) or 0.20 seconds on the horizontal axis or time line and 0.5 millivolt on the vertical axis or amplitude line. Recordings of electrical activity made from within the cardiac chambers are called *intracardiac electrograms*.

Paper used for routine cardiac monitoring is marked across the top by small vertical lines placed at 3-second intervals. Heart rate per minute can be rapidly estimated by counting the number of beats in a 6-second recording and multiplying that number by 10, or can be precisely calculated by counting the number of small squares between complexes and dividing that number into 1500. All monitoring systems currently marketed display the heart rate both on screen and on paper recordings.

The complexes

An electrocardiogram consists of only two elements: *complexes* and *intervals*. The normal complexes are (1) the P wave, (2) QRS complex, (3) T wave, and (4) U wave (Figure 1.1).

The *P wave* represents depolarization of the atrial myocardium. Normal P waves are rounded, do not exceed 0.25 mV (2.5 mm) in amplitude in any lead or exceed 110 milliseconds (0.11 second) in duration. Normal P wave axis is +15 to +75 degrees in the frontal plane leads. The amplitude of the P wave is measured from the baseline or *isoelectric line* to the top of the waveform. Because the right atrium is depolarized slightly before the left atrium, the first

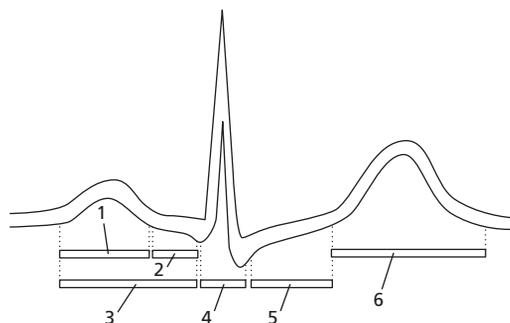


Figure 1.1 1: P wave, 2: PR segment, 3: PR interval, 4: QRS complex, 5: ST segment, 6: T wave.

half of the P wave represents right atrial depolarization and the last half left atrial depolarization, but normally these events overlap, producing a single deflection.

Figure 1.2 correlates the features of the surface ECG with cardiac electrical events. It is essential to note that sinus node discharge (1) is *electrocardiographically silent* on surface tracings, as is conduction through the atrioventricular node (4), the bundle of His and bundle branches (5).

The recovery sequence can be divided into three phases: (1) the *absolute refractory period* (7), during which the conduction structures are unresponsive to any stimulus; the *supernormal period* (8), and the *relative refractory period* (9), during which the conduction tissues will transmit an impulse, but typically at a slower rate than is normally observed. Refractory periods shorten and lengthen incrementally as the heart rate accelerates or slows, i.e. as the cycle length changes. Therefore the exact length of the refractory periods will vary according to the heart rate and the health of the conduction system.

The so-called *supernormal period* (8) is one of medicine's great misnomers. In fact, the phenomenon of *supernormal conduction* is nearly always observed

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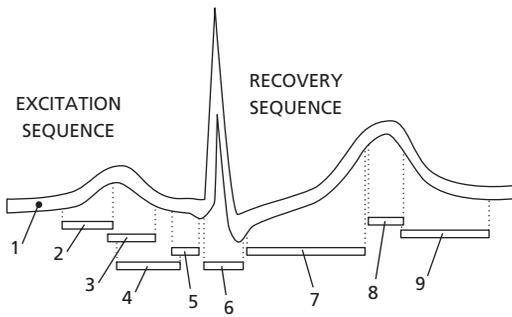


Figure 1.2

EXCITATION SEQUENCE	ECG
1. Sinus node depolarization	Silent
2. Right atrial activation	1 st half of P wave
3. Left atrial activation	2 nd half of P wave
4. Atrioventricular node	Silent
5. His bundle/bundle branches	Silent
6. Ventricular activation	QRS complex
RECOVERY SEQUENCE	ECG
7. Absolute refractory period	ST segment
8. Supernormal period	Peak of T wave
9. Relative refractory period	T wave.

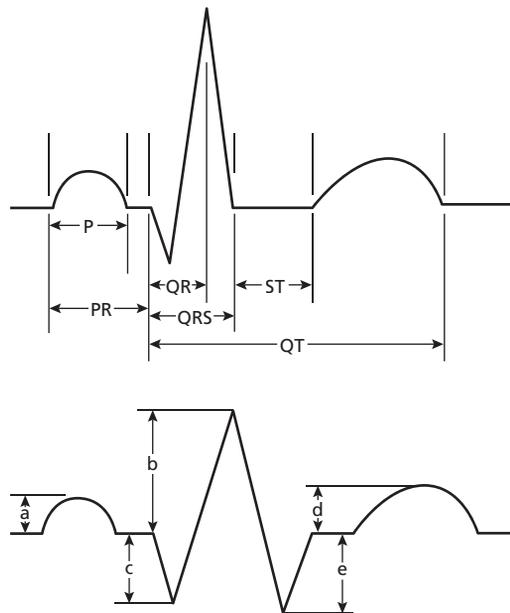


Figure 1.3 Complexes and intervals. a: P wave amplitude, b: R wave amplitude, c: Q wave amplitude, d: T wave amplitude, e: S wave amplitude.

in the setting of severe conduction impairment when conduction is *subnormal*, not 'supernormal.' Supernormal conduction is a function of timing: impulses that fall on the peak of the T wave are conducted whereas impulses arriving earlier or later are not. Supernormality is therefore characterized by (1) conduction that is better than expected and (2) better earlier than later.

The *QRS complex* represents ventricular myocardial depolarization. The QRS amplitude exhibits a wide range of normal values, but an amplitude greater than 1.1 mV (11 mm) in lead aVL, greater than 2.0 mV (20 mm) in lead aVF in the frontal plane leads, or greater than 3 mV (30 mm) in the horizontal plane (precordial) leads is considered abnormally high. The duration of the normal QRS complex ranges from 50 to 100 msec (0.05 to 0.10 sec).

The positive and negative deflections of the QRS complex are named according to universal conventions. The first deflection of the QRS complex, if *negative*, is called a *Q wave*. The Q wave amplitude is measured from the baseline to the deepest point of the written waveform (Figure 1.3). Small, narrow

Q waves are expected findings in leads I, III, aVL, aVF, V5 and V6. Normal Q waves do not exceed 30 msec (0.03 sec) duration in any lead. The Q wave may be represented by a lower case (*q*) or upper case (*Q*) letter according to its size in relation to the other QRS deflections. Completely negative QRS complexes or QRS complexes in which no positive deflection reaches more than 1 mm above the baseline are called *QS complexes* (Figure 1.4).

The first *positive* deflection of the QRS complex, whether preceded by a negative deflection (Q wave) or not, is called the *R wave*. The R wave amplitude is measured from the baseline to the peak of the written waveform (Figure 1.3). In the case of polyphasic QRS complexes, subsequent positive deflections are labeled R'. The R wave may be represented by an upper or lower case letter according to its relative size (Figure 1.4).

A *negative* deflection following an R wave is called an *S wave*. The S wave amplitude is measured from the baseline to the deepest point of the written waveform. In the case of polyphasic QRS complexes, a subsequent negative deflection following the first S wave is called an S' wave. Like Q waves

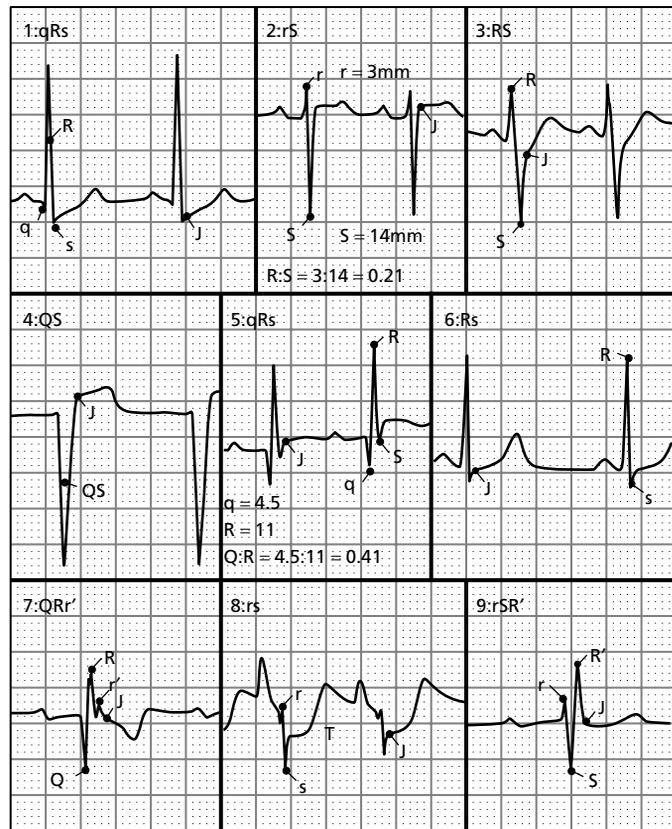


Figure 1.4 Waveform nomenclature.

and R waves, an S wave may be represented by a lower or upper case letter according to its size.

The *T wave* represents ventricular myocardial repolarization. Its amplitude, which is measured from the baseline to the highest point of the written waveform, does not normally exceed 0.5 mV (5 mm) in any frontal plane lead or 1.0 mV (10 mm) in any horizontal plane (precordial) lead. The proximal limb of a normal T wave exhibits a gentle upward slope, while the distal limb, the descending component, has a steeper slope as it returns to the baseline (compare 1a to 3a in Figure 1.6). In other words, normal T waves are not sharply pointed ('tented'), nor are they symmetrical. T wave polarity varies according to the lead, being normally positive (upright) in leads I, II, and V3–V6 in adults, negative (inverted) in lead aVR, and variable in leads III, aVL, aVF, and V1–V2.

The *U wave*, a low-voltage deflection that probably represents repolarization of the Purkinje fibers,

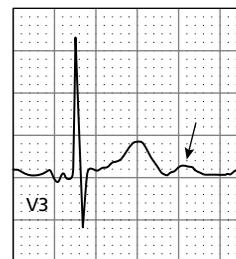


Figure 1.5 The U wave.

is sometimes seen following the T wave (Figure 1.5). Its polarity is usually the same as the preceding T wave. The U wave begins *after* the T wave has reached the isoelectric base line. The second component of a bifid T wave should not be mistaken for a U wave. The presence of a U wave may be attributed to electrolyte imbalance (particularly hypokalemia), drug effects, and myocardial ischemia. Bradycardia tends to accentuate the U wave.

The intervals

The clinically relevant ECG intervals are shown in Figure 1.3.

The *PR interval* consists of two components: (1) the P wave and (2) the PR segment. The duration of the PR interval, measured from the beginning of the P wave to the first deflection of the QRS complex, is typically 120 to 200 msec (0.12 to 0.20 sec) in adults. A PR interval greater than 180 msec (0.18 sec) in children or 200 msec (0.20 sec) in adults is considered *first-degree atrioventricular block*.

The *QR interval*, measured from the beginning of the QRS complex to the highest point of the R wave, is an indirect reflection of ventricular activation time. Its clinical importance and applications are discussed in subsequent chapters.

The *QRS interval*, measured from beginning to end of the total QRS complex, normally ranges from 50 to 100 msec (0.05 to 0.10 sec) in duration. If the QRS interval is 120 msec (0.12 sec) or more, *intraventricular conduction delay* is present.

The *ST segment* is measured from the end of the QRS complex to the beginning of the T wave. The junction of the QRS complex and the ST segment is called the *J point* (Figure 1.4). The ST segment is normally *isoelectric* at the J point (in the same plane as the baseline) but may be normally elevated up to 1 mm in the frontal plane leads and up to 2 mm in the horizontal plane leads. Any ST segment depression greater than 0.5 mm is regarded as abnormal.

The *QT interval*, measured from the beginning of the QRS complex to the end of the T wave, normally varies with heart rate and to a lesser extent with the sex and age of the subject. The QT interval adjusted for rate is called the *corrected QT interval* (QTc). The upper limits of normal QT intervals, adjusted for rate, are shown in Table 1.1. Prolongation of the QT interval is seen in congenital long QT syndromes (Romano–Ward, Jervell and Lange–Nielsen), myocarditis, myocardial ischemia, acute cerebrovascular disease, electrolyte imbalance, and as an effect of a rather long list of drugs. Polymorphic ventricular tachycardia, known as *torsade de pointes* (TDP), is often associated with QT prolongation. Since women normally have longer QT intervals, they are more susceptible to torsade than males.

Table 1.1 Upper limits of the QTc interval.

Rate	QTc interval (sec)
40	0.49–0.50
50	0.45–0.46
60	0.42–0.43
70	0.39–0.40
80	0.37–0.38
90	0.35–0.36
100	0.33–0.34
110	0.32–0.33
120	0.31–0.32

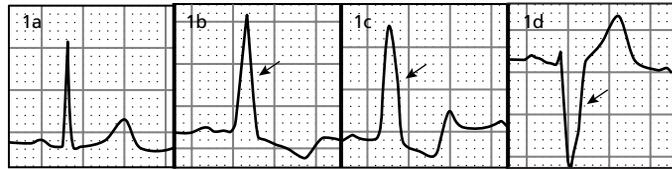
A word of caution is in order about the measurement of intervals. It is often the case that the inscription of a wave is not crisply demarcated, leaving some doubt about exactly when a complex begins or ends. Exact measurement may be particularly problematic if the complex is of low voltage or if the ascent from or return to the baseline is slurred. It is often difficult to determine when T waves end, for example. Exact measurement of the PR interval may be difficult if the beginning of the P wave or the QRS complex is not clearly inscribed. In such cases, clear delineation of the complexes must be sought by examining different leads. A tracing in which baseline wander or artifact obscures the complexes is of little or no diagnostic value.

Two other commonly used intervals are the *P to P interval* (P–P), the time in seconds from one P wave to the following P wave, used to indicate atrial rate and/or regularity, and the *R to R interval* (R–R), the time in seconds from one QRS complex to the next QRS complex, used to indicate ventricular rate and/or regularity.

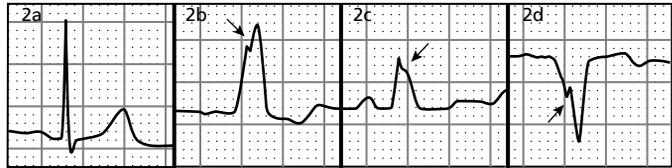
Slurring, notching and splintering

As shown in Figure 1.6, the normal QRS complex is narrow and displays deflections that are crisply inscribed. In the presence of intraventricular conduction delay, the QRS widens and the initial deflection tends to drift, a finding known as *slurring*. In addition, *notching* may be noted on the initial deflection, whether it is positive or negative. Notches are localized deformities that do not extend downward or upward to the baseline, i.e. they are

SLURRING



NOTCHING



DELTA WAVES

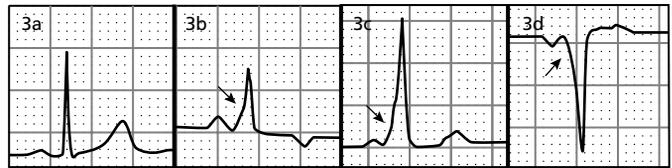


Figure 1.6 Slurring, notching and delta waves.



Figure 1.7 Splintering of the QRS complex.

not discrete waves. Very occasionally a QRS deformity known as *splintering* is encountered (Figure 1.7). Splintering of the QRS complex is associated with advanced, severe myocardial disease.

Several QRS deformities are associated with specific conditions: *delta waves* are the result of ventricular fusion due to pre-excitation and are one

of the hallmarks of the Wolff–Parkinson–White syndrome. They are described in the chapter devoted to that syndrome. *Osborne waves* or *J waves*, hump-shaped depressions noted at the J point, are most often noted in extremely hypothermic subjects. They are described in the chapter on myocardial ischemia.

