Rhythm

Rhythm is the pattern of the complexes, waves, and intervals, the regularity or irregularity of their occurrence, and the relationships among these constituents. Disturbances or irregularities of rhythm are usually called arrhythmias. A perhaps more accurate, but less common, term for the same phenomenon is dysrhythmia. In keeping with usual clinical terminology, the term arrhythmia will be used in this book.

### SUPRAVENTRICULAR RHYTHMS I

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### CLINICAL VIGNETTE

A new patient in phase II cardiac rehabilitation reports feeling like his “heart was pounding” while he was cooling down from his first exercise session.

![ECG waveform]

**Rhythm**

Rhythm is the pattern of the complexes, waves, and intervals, the regularity or irregularity of their occurrence, and the relationships among these constituents. Disturbances or irregularities of rhythm are usually called arrhythmias. A perhaps more accurate, but less common, term for the same phenomenon is dysrhythmia. In keeping with usual clinical terminology, the term arrhythmia will be used in this book.
Inspection of more than one ECG lead is necessary for many diagnoses (and can sometimes be of great help in determining the rhythm), but often only one lead is needed to determine the rhythm. In the interest of simplicity and clarity, here it is assumed that the ECGs described in this section are recorded using lead II. Lead II is one of several bipolar leads, so named because these leads possess positive and negative sensing electrodes. For the current discussion the important point is that electrical current heading toward the positive pole of a lead will result in positive (rising above the baseline) deflections on the ECG, and electrical events heading away from the positive pole (and therefore toward the negative pole) will result in negative (below baseline) deflections. A current traveling perpendicular to the axis of a lead will result in a waveform with roughly equal positive and negative components. Depending on various factors, either the positive or the negative component may come first, or it may manifest as a flat line. Figure 2.1 illustrates these concepts in relation to P waves. The same concepts apply to other waves as well.

As the positive pole of lead II is placed on the left leg, and the negative pole is located on the right arm, lead II is in line with the normal plane of key electrical events because depolarization normally tends to propagate through the heart from upper right to lower left and therefore toward the positive pole of lead II.

The discussion here begins with rhythms that have their origin above the ventricles, thus termed supraventricular.

**Sinus Rhythms**

As shown in Figure 2.2, the sinoatrial (SA) node is normally the first area of the heart to depolarize. (This and similar figures throughout the book are not anatomically correct representations of the heart, but rather diagrammatic illustrations of the conduction system.) As the SA node is located in the right upper portion of the right atrium, the net current spreads down and toward the left. Actually current is spreading in many directions, but the standard ECG measures the average (net) vector of the currents.

![Diagram of ECG leads and P waves](image-url)

**Figure 2.1** P waves resulting from current flow with differing orientation to electrodes.
The positive pole of lead II is located on the left leg, so a P wave beginning in the SA node and spreading through the atria in the normal fashion will (in terms of the net vector of the current) be heading toward the positive pole of lead II and therefore result in a positive deflection on the cardiogram.

**Normal Impulse Origin and Pattern of Conduction**

The normal pacemaker of the heart is the SA node. In this context, the term pacemaker refers to intrinsic areas of the heart that have the property of automaticity, that is, a regular pattern of depolarization in the absence of external influence (although various factors including the autonomic nervous system can alter the rate of these depolarizations). Battery-operated electronic pacemakers, which are often used to pace the hearts of patients with very slow rates and other conditions, are a separate issue, which is discussed in Chapter 5. In the present context the term pacemaker refers to the naturally occurring pacemakers.

As is the case with several areas of the heart, the SA node depolarizes regularly in the absence of external stimuli. Normally the SA node depolarizes at a faster rate than the other pacemakers so the other pacemakers remain quiescent as they are
"reset" by the depolarizing current that arises from the SA node before they get a chance to fire. In the absence of depolarizations arising from the SA node, subsidiary pacemakers located in areas including the AV junction (defined below) and the ventricles can take control of pacing the heart. Usually the lower pacemakers (i.e., those located in the ventricles) have slower inherent rates; this means that the higher pacemakers (e.g., AV junction) will typically take over pacing if the SA node fails to depolarize or does so at too slow a rate. Even if the SA node is firing at a normal rate, numerous factors can excite subsidiary pacemakers, resulting in beats that do not arise from the SA node.

The normal spread of depolarization in the heart is shown in Figure 2.3. As previously discussed, the depolarization normally begins spontaneously in the SA node (Fig. 2.3A). The intracellular fluid of the atrial cells is in contact with that of neighboring cells via gap junctions, allowing the depolarization to spread from cell to cell throughout the atria (Fig. 2.3B). This results in the P wave of the ECG. The atrioventricular (AV) node is surrounded by atrial tissue; therefore, the wave of depolarization spreading throughout the atria reaches the AV node. Normally, the tissue separating the atria from the ventricles does not conduct current, the only normal pathway for the depolarizing current to reach the ventricles is via the AV node and the next portion of the conduction system, which is known as the bundle of His (pronounced "hiss"). The tissue of the AV node conducts current at a much slower rate than other portions of the conduction system; this results in what appears to be a slight "delay" before the current reaches the ventricles (Fig. 2.3C). Physiologically, this delay permits time for the atria to contract and further fill the ventricles with blood prior to ventricular depolarization (and subsequent contraction). The area where the AV node and the bundle of His meet is called the AV junction.

Following the "delay" at the AV node, current spreads down the bundle of His (Fig. 2.3D), and then down the left and right bundle branches (Fig. 2.3E). The bundle of His and the bundle branches are areas of tissue specialized for the conduction of current (i.e., they function as "wires" rapidly bringing the current down into the ventricles). The left bundle branch actually has two pieces, known as anterior and posterior fascicles, but for the present it is sufficient to simply think of a right bundle branch bringing the current down into the right ventricle and a left bundle branch bringing the current down into the left ventricle. Sprouting off of the bundle branches are the smallest "wires" of the conduction system, the Purkinje fibers (Fig. 2.3F). Purkinje fibers are well dispersed throughout the ventricular myocardium but do not reach every cell. Cells that are not directly in contact with Purkinje fibers are depolarized by neighboring cells, because, as in the atria, the intracellular fluid of ventricular cells is in contact with that of adjoining cells via cellular connections known as gap junctions.

Because ventricular cells are linked together electrically by gap junctions, depolarizing currents reaching the ventricles (or arising from the ventricles) can spread without using the conduction system, but would spread more slowly and in a less-organized fashion. The purpose of the conduction system is to cause a more rapid and organized ventricular depolarization. Depolarizing current that spreads via the normal conduction system usually results in a "narrow" QRS complex (i.e.,
<100 ms) because it travels quickly; depolarizations that do not go through the conduction system normally, but rather largely spread via the slower cell to cell pathways, result in a “wide” (i.e., $\geq 100$ ms) QRS complex.

**Normal Sinus Rhythm**

The normal sinus rhythm (NSR) is the “normal” rhythm of the heart. Several requirements must be met for the designation of NSR. A “normal” heart rate (HR) has been somewhat arbitrarily designated as between 60 and 100 beats per minute (bpm). As the “sinus” part of the name indicates, in this rhythm it is assumed that the SA node is pacing the heart. Therefore, a positive P wave (representing atrial depolarization) should be present in lead II. This is because, as indicated in the previous section, the net current is heading down and to the left from the SA node and, therefore, toward the positive pole of lead II. Actually many authorities believe that the P wave must be positive in leads I, II, $V_5$, and $V_6$ to truly confirm sinus rhythm.

**Figure 2.3** Steps of the conduction system. **A:** SA node depolarizes. **B:** Depolarization spreads throughout the atria. **C:** Delay at the AV node. **D:** Bundle of His depolarizes. **E:** Left and right branches depolarize. **F:** Purkinje fibers depolarize.
As a practical matter, if the P wave is positive in lead II, it usually (but not always) is positive in leads I, V5, and V6.

In addition to the above requirements, the rhythm must be regular and one P wave must be present for each QRS complex. **Figure 2.4** shows examples where all of these conditions are met. The two short strips of Figure 2.4A show simultaneous recordings in lead II, and a lead called aVR. Notice that the P waves are positive in lead II and negative in lead aVR. The positive pole of lead aVR is located on the right arm; therefore, current moving down and to the left should be seen as a negative deflection in lead aVR as it is traveling away from the positive pole of this lead. If normal sinus rhythm is present the P waves should be positive in lead II and negative in lead aVR. In the absence of technical error, if the P waves are positive in lead II it is virtually ensured that they will be negative in aVR. The longer lead II strip in Figure 2.4B illustrates the regularity of the rhythm and the consistent presence of one P wave for each QRS complex. Longer strips such as this one are commonly referred to as rhythm strips.

**Sinus Arrhythmia**

If the other conditions for NSR are present (HR between 60 and 100 bpm, positive P waves in lead II, and one P wave for each QRS complex), but the rhythm is irregular (i.e., the R-R interval is varying), then sinus arrhythmia is present. Compare Figures 2.4 and **2.5**. Notice with NSR (Fig. 2.4), the R-R interval shows little variation. In other words, the rhythm is regular. Contrast this with Figure 2.5, where the R-R interval varies significantly. One commonly used definition of significant

![Figure 2.4](image)

**Figure 2.4** Normal sinus rhythm. **A:** P waves are positive in lead II and negative in lead aVR. **B:** Rhythm is regular with a rate between 60 and 100 bpm. PR intervals are consistent with one P wave for each QRS.
variation is a difference of at least 80 ms (two small boxes on the ECG paper) between the shortest and longest R-R intervals.

Some variation in the R-R interval is normal and often is associated with respiration. With inspiration the diaphragm descends, decreasing intrathoracic pressure, leading to less vagal stimulation. This in turn increases HR. The opposite effect occurs with exhalation; vagal stimulation is increased and HR is decreased. It is quite normal for HR to increase (decreased R-R interval) with inspiration and decrease (lengthened R-R interval) with expiration. Sinus arrhythmia is simply an exaggeration of this normal variation. Sinus arrhythmia that is associated with ventilation is sometimes termed *phasic sinus arrhythmia*. Sometimes sinus arrhythmia is not related to ventilation; this can be referred to as a *nonphasic sinus arrhythmia*.

### Sinus Tachycardia and SA Nodal Re-Entrant Tachycardia

If all of the conditions for NSR are present, with the exception that the HR is >100 bpm, then sinus tachycardia is present (Fig. 2.6). This term simply indicates a fast heart rate. As the heart rate increases it may become more difficult to discern P waves. In Figure 2.6A, P waves are clearly visible. In Figure 2.6B, the rate is faster and the P waves have begun to merge with the T waves of the preceding beats, making them more difficult to distinguish. If Figure 2.6.B were recorded during exercise it would be reasonable to assume that it represented sinus tachycardia. However, if this tracing...
were obtained under resting conditions, it likely would represent another sinus rhythm, SA nodal re-entrant tachycardia, an abnormal depolarizing current that spins rapidly around in the area of the SA node resulting in fast heart rates. As the current circles around repeatedly re-entering the SA node it is called a re-entrant, or circuit, current. Since the initial depolarizations come from the SA node, the P waves appear normal. The rate is typically >100 bpm, and the rhythm is regular.

Sinus tachycardia usually comes on gradually, for example, the rate may increase from 70 to 80 to 90 and so on until it exceeds 100 bpm. Sinus tachycardia also shows a gradual decline in rate when returning to NSR. In contrast, SA nodal re-entrant tachycardia has a sudden onset and a sudden cessation. For example, the rate may jump in one beat from 70 to 150 bpm. The termination is equally abrupt. If the onset or termination of the tachycardia is witnessed the diagnosis is simplified. If the rhythm shown in Figure 2.6 had a sudden onset and sudden termination this would virtually ensure that it is an SA nodal re-entrant tachycardia. Gradual onset and termination of the same rhythm implies sinus tachycardia. If the onset is not witnessed, the rate and regularity offer important clues. Higher rates and constant R-R intervals more likely represent SA nodal re-entrant tachycardia, lower rates and R-R variability favor a diagnosis of sinus tachycardia. In cases wherein the rhythm cannot be discerned with certainty, the term versus is often inserted between the possibilities, so Figure 2.6B might be described as “sinus tachycardia versus SA nodal re-entrant tachycardia.”

**Sinus Bradycardia**

Sinus bradycardia (Fig. 2.7) is simply a slow HR, usually defined as <60 bpm. Other than the low rate, the criteria for NSR are met. The term marked sinus bradycardia is sometimes used when the rate is very low, as in Figure 2.7B. Slow rates can indicate pathology, but are also seen in highly trained endurance athletes (due to high vagal tone) and patients taking beta adrenergic antagonist medications (commonly known as beta blockers).

**Sinus Pause**

A delay occurring before the appearance of a P wave (Fig. 2.8) may be caused by several mechanisms. These include failure of the SA node to depolarize or an SA
“block,” which does not permit the depolarization to escape from the SA node. It is often difficult, if not impossible, to determine the mechanism of the delay from a standard ECG. Regardless of mechanism, the general term sinus pause can be used to describe this phenomenon.

Note in Figure 2.8A that after the pause the rhythm resumes with a P wave that looks like the normal P waves. In Figure 2.8B the rhythm resumes with a normal appearing QRS that is not preceded by a P wave. As discussed in the following sections, this implies that the beat does not originate in the SA node.

Pauses can be quantified. For example, Figure 2.8A could be described as “sinus rhythm with a sinus pause of 1.16 sec.” The length of the pause is measured from the beginning of the P wave of the beat preceding the pause to the beginning of the P wave of the beat following the pause (in this case, 29 little boxes, which is equivalent to 1,160 ms or 1.16 sec).

**Premature Atrial Complex**

A premature atrial complex (PAC) is an early (premature) beat arising from somewhere above the ventricles other than the sinus node (i.e., in the atria or the AV junction). In general, depolarizations arising in areas of the heart other than the
normal pacemaker (SA node) are referred to as *ectopic*. The site of origin of these ectopic depolarizations is termed an ectopic focus.

Early beats can arise from numerous areas of the heart. A premature depolarization arising from above the ventricles (supraventricular) can be called by many names, perhaps the most common is PAC. Other terms used to describe these beats include atrial premature contraction (APC), atrial premature depolarization (APD), and atrial premature beat (APB). The term junctional premature complex (JPC) is also sometimes used to describe certain supraventricular beats.

A PAC starts in an ectopic focus somewhere in the atria or the AV junction. Since the P wave represents atrial depolarization, it follows that depolarizations arising in areas of the atria other than the SA node will typically have P waves that differ in appearance from the sinus P waves. They will usually also take a different amount of time to travel through the atria and down into the ventricles, thus the PR intervals of PACs may differ from that of sinus beats. A PAC originating in the upper or middle parts of the atria may still result in an upright P wave in lead II. This is because the mean electrical vector of atrial depolarization is generally heading toward the positive pole of lead II (*Fig. 2.9*). In order to be a PAC the beat must

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**FIGURE 2.9** Atrial ectopic focus. The corresponding ECG is shown at the top right of this figure (* indicates the ectopic focus).
be early; notice how the P-P interval of the PAC shown in Figure 2.10 is shorter than the other P-P intervals. This is just a way of indicating that this P wave came early.

Typically a PAC is conducted normally through the AV node and ventricles, resulting in a normal appearing QRS complex. This is because conduction throughout the ventricles follows the normal path (AV node, bundle of His, bundle branches, Purkinje fibers), the only abnormality is in atrial conduction. Cases where PACs lead to abnormal (aberrant) ventricular conduction will be discussed later. Sometimes a PAC is not conducted through the ventricles at all, usually because the AV node is still in a refractory period; this is called a blocked or non-conducted PAC (Fig. 2.11). Notice how the P wave of the nonconducted PAC in Figure 2.11 merges with the T wave of the preceding beat. This P wave came so early that it failed to conduct down into the ventricles, probably because the rest of the conduction system was still in a refractory period. The P waves in this example
are biphasic, as this strip was recorded using lead V1, a lead that normally has biphasic P waves.

If an early beat arises lower in the atria or at the AV junction, the resulting wave of depolarization typically spreads through the atria in the opposite direction as beats originating in the SA node (Fig. 2.12). In other words, these PACs may spread through the atria from bottom to top, as opposed to the normal top to bottom conduction of sinus beats. In this case atrial depolarization is spreading away from the positive pole of lead II, resulting in a negative P wave in that lead. The depolarization also typically spreads down into the ventricles; however, here the impulse is usually conducted normally, resulting in a normal appearing QRS complex. Depending on exactly where the PAC arises and the prevailing conditions in the atria, junction, and ventricles, it may happen that the atria depolarize ahead of the ventricles (but backward, also called retrograde, in that the impulse spreads from bottom to top), at roughly the same time as the ventricles, or after the ventricles have finished depolarizing.

If the depolarization spreads through the atria first, then a negative P wave will be seen before the QRS complex (Fig. 2.13A); if the depolarization spreads through the ventricles first and then through the atria, a negative P wave will be seen after
the QRS complex (Fig. 2.13B). If the depolarization of the atria and ventricles occurs at roughly the same time, no P wave may be visible (Fig. 2.13C), as the much larger electrical events of ventricular depolarization (QRS complex) obscure the relatively small events of atrial depolarization (P wave). Thus, beats originating low in the atria or at the AV junction may have negative P waves before or after the QRS complex or no P wave at all.

**Junctional Premature Complexes**

Figure 2.14 shows a PAC with a negative P wave prior to the QRS complex. Premature beats with negative or missing P waves are sometimes referred to as JPCs because many of them arise at or near the AV junction. From a practical
standpoint, it is often difficult, if not impossible, to determine from a standard ECG whether these beats arose from low in the atria or from the AV junction.

**Atrial Bigeminy, Trigeminy, and Quadrigeminy**

Additional terminology can be used to describe PACs that occur in patterns. Atrial bigeminy (Fig. 2.15A) refers to a situation wherein every second beat is a PAC. The terms atrial trigeminy (Fig. 2.15B) and atrial quadrigeminy (Fig. 2.15C) are used to describe conditions where every third or fourth beat, respectively, is a PAC.

**Ectopic Atrial Tachycardia**

By definition, three or more PACs occurring consecutively is called atrial tachycardia. Since the term *ectopic* is used to describe depolarizations originating in areas other than the sinus node, ectopic atrial tachycardia can be used to describe tachycardias that originate from the atria but not from the sinus node. Two examples of ectopic atrial tachycardia are shown in Figure 2.16. It is sometimes difficult to distinguish sinus tachycardia from ectopic atrial tachycardia. As previously discussed, ectopic beats usually have P waves and/or PR intervals that differ from the norm. Unfortunately, even if the normal PR interval and P wave morphology are known, sinus tachycardia may affect the shape of the P waves and the time needed for AV conduction.

A few general rules can help, although exceptions to the rules occur. If the initiation or termination of the tachycardia is witnessed, diagnosis is much easier as ectopic atrial tachycardias typically have a sudden onset and termination. Notice in Figure 2.16B how the rate abruptly changes. The initial rhythm is sinus tachycardia. A PAC then initiates a short run of ectopic atrial tachycardia. Notice how the P wave of the first ectopic beat merges with the T wave of the proceeding beat.

**Figure 2.15** PAC patterns. A: Atrial bigeminy. B: Atrial trigeminy. C: Atrial quadrigeminy.
P waves are then not readily apparent (but are present on top of the T waves) until the sinus P waves re-emerge toward the end of the strip. Sinus tachycardia typically has a more gradual onset and termination. An exception to this is the previously described SA nodal re-entrant tachycardia, which has a sudden onset. The P waves of an SA nodal re-entrant tachycardia usually resemble sinus P waves (as they originate in the SA node), while the P waves of an ectopic atrial tachycardia should differ in appearance from sinus P waves. Electrical alternans, a phenomenon where the height (amplitude) of the R waves regularly alternates, commonly occurs with ectopic atrial tachycardia, but is not typical in SA nodal re-entrant tachycardia. Figure 2.16A shows this phenomenon.

Inspection of R-R variability (the variation between R-R intervals) can also be helpful. In ectopic atrial tachycardia and SA nodal re-entrant tachycardia the R-R interval is often very constant, whereas in sinus tachycardia variation is typically seen. Recall from the discussion of sinus arrhythmia that ventilation normally alters HR (i.e., the R-R interval). If ventilation causes changes in the R-R interval it is more likely to be a sinus tachycardia as ectopic atrial tachycardia and SA nodal re-entrant tachycardia are usually not significantly affected by ventilation. Ectopic atrial tachycardias and SA nodal re-entrant tachycardias often exhibit rates >160 bpm; the higher the rate, the less likely that it is sinus tachycardia. The setting is also relevant. The previous discussion assumes that the patient is at rest. During strenuous exercise sinus rates as
high as 180 to 200 bpm are commonly observed in younger people, but it is unusual (although possible) for sinus tachycardia to have a rate of >140 to 160 bpm at rest.

**Atrioventricular Nodal Re-Entrant Tachycardia**

Current can rapidly circulate around the AV node, similar to what happens near the SA node in SA nodal re-entrant tachycardia. As might be expected, if the re-entry is occurring around the AV node, the resulting tachycardia is called AV nodal re-entrant tachycardia (AVNRT). The P waves are usually not seen or are negative and follow the QRS complex.

*Figure 2.17* A shows an ectopic atrial tachycardia and two variations of AVNRT (Fig. 2.17B,C). Notice that the ectopic atrial tachycardia has negative P waves visible before the QRS complexes (recall that ectopic atrial tachycardias can have positive or negative P waves). Contrast this with the two AVNRTs where P waves either are not visible (Fig. 2.17C) or are negative and follow the QRS complexes (Fig. 2.17B).

It is common practice to refer to any of these three rhythms as junctional tachycardia. Because the mechanisms underlying the rhythms are different, it is preferable to distinguish between ectopic atrial and atrioventricular re-entrant tachycardia.

**Junctional Escape**

As previously discussed, areas near the AV junction possess the ability to spontaneously depolarize. If left alone the “junction” will rhythmically depolarize at a rate of around 40 to 60 times per minute. Usually the SA node depolarizes at a faster rate. As each sinus depolarization resets the other pacemakers of the

![Figure 2.17](image-url)
heart, secondary pacemakers such as the AV junction will not typically become apparent unless the SA node fails to depolarize or does so at an abnormally slow rate.

The morphology of junctional escape beats is identical to what was described earlier for early beats arising from at or near the AV junction. The difference is that these beats are not early. In fact they could be thought of as late, as they usually only occur if the SA node or other higher pacemaker fails to fire. This is a protective mechanism; in the event that the SA node fails to depolarize the heart in a timely fashion these and other subsidiary pacemakers can take over.

As previously discussed, depolarizations arising at or near the AV junction will typically have a normal QRS complex and either (a) no apparent P wave, (b) a negative P wave before the QRS complex, or (c) a negative P wave after the QRS complex. The QRS complexes are typically normal, as ventricular depolarization occurs via the normal ventricular conduction system.

Figure 2.18A shows a single junctional escape beat (the underlying rhythm is sinus bradycardia). In this case an isolated delay in sinus depolarization has occurred. The delay is long enough to allow the AV junctional pacemaker to fire.

In Figure 2.18B the sinus pacemaker has either failed or slowed sufficiently that a junctional pacemaker has assumed control of the rhythm. This is usually referred to as a junctional escape rhythm. The HR in a junctional escape rhythm is generally <60 bpm.

**Accelerated Junctional Rhythm**

Rhythms that appear to be originating at or near the AV junction (narrow QRS complexes and negative or absent P waves) and have rates between 60 and 100 to 120 bpm can be called accelerated junctional rhythms. Although tachycardias are generally defined as rates >100 bpm, some authorities define junctional rhythms with rates from 60 to 120 bpm as accelerated junctional, while others use 60 to 100 bpm. The term simply indicates that the rate is faster than the junctional
FIGURE 2.19 Accelerated junctional rhythms. **A:** Shows an accelerated junctional rhythm with a rate of about 70 bpm and no apparent P waves. **B:** Shows an accelerated junctional rhythm with a rate of about 64 bpm and negative P waves after QRS.

escape rate, yet slower than a junctional (ectopic atrial) tachycardia. Two examples are shown in **Figure 2.19.** As with other junctional rhythms, P waves may be absent or negative.

**CLINICAL VIGNETTE REVISITED**

A new patient in phase II cardiac rehabilitation reports feeling like his heart was pounding while he was cooling down from his first exercise session.

A brief run of supraventricular tachycardia, which most likely was atrioventricular re-entrant tachycardia, was noted on the telemetry monitor by the physiologist. The patient was stable and in no distress after the episode. Blood pressure was taken and the patient queried about any associated symptoms, changes in medications, habits, activities, or other factors, then the patient’s physician was contacted for instructions.
Quiz 2

1. Describe the normal sequence of myocardial depolarization.

2. Describe the rhythm.

3. Describe the rhythm.

4. Describe the rhythm.