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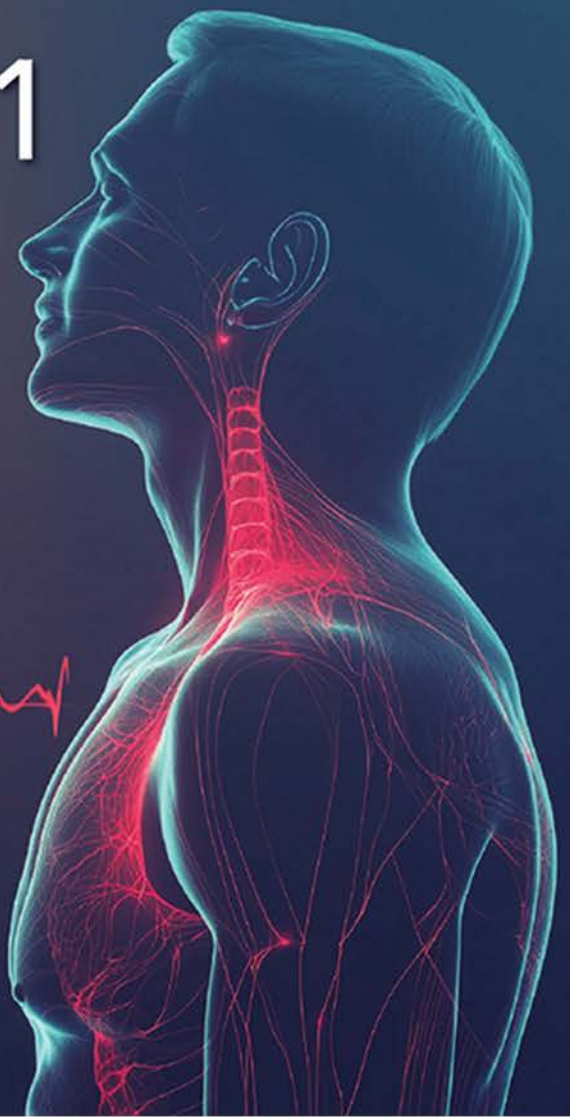
Arrhythmias 101

The Ultimate Easy-To-Read Introductory Book To Arrhythmias





Glenn N Levine

JAYPEE

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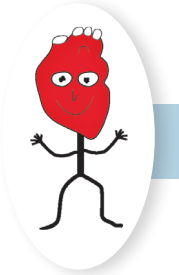

CHAPTER 4

Tachyarrhythmias Originating in the Atria

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In this chapter we will discuss the tachycardias that originate in the atria of the heart. Not surprisingly these arrhythmias are called **atrial arrhythmias**. The rhythms we will cover include:

- Sinus tachycardia
- Atrial tachycardia
- Multifocal atrial tachycardia (MAT)
- Atrial flutter
- Atrial fibrillation

SINUS TACHYCARDIA

Although **sinus tachycardia** is not technically an arrhythmia, it is important to briefly discuss sinus tachycardia, as it can be mistaken for a pathological arrhythmia, and because when it occurs, it is important to figure out what is *causing* the sinus tachycardia. Figure 4.1 shows

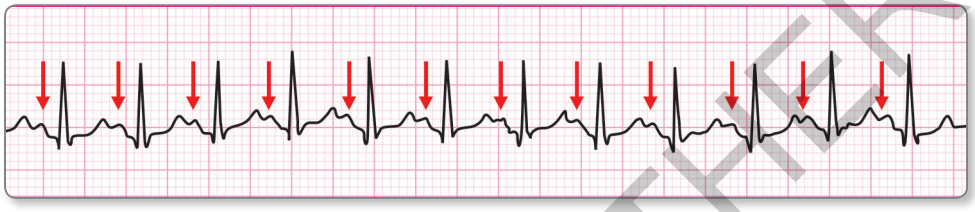

Figure 4.1 Sinus tachycardia. If one does not see there are P waves before each QRS complex, this rhythm could be mistaken for an arrhythmia

an example of sinus tachycardia. If one does not recognize that there are P waves before each QRS complex, this rhythm could be mistaken for a pathological arrhythmia.

Sinus tachycardia is, in general, caused by increased sympathetic stimulation of the SA node in response to some stressor on the body. Stressors that can cause a sinus tachycardia are listed in Table 4.1:

Table 4.1: Stressors and other causes in the differential diagnosis of persistent sinus tachycardia

• Anemia	• Hypotension	• Sepsis
• Acute severe bleeding	• Acute MI	• Dehydration
• Hypoxemia	• Heart failure	• Medications and medication overdose
• Pain	• Pericardial tamponade	
• Anxiety	• Tension pneumothorax	• Hyperthyroidism
• Fever	• Pulmonary embolism	

ATRIAL TACHYCARDIA

Atrial tachycardia is an arrhythmia that originates in the right or left atrium at a site other than where the SA node is located. Most commonly, atrial tachycardia is due to a focal area of

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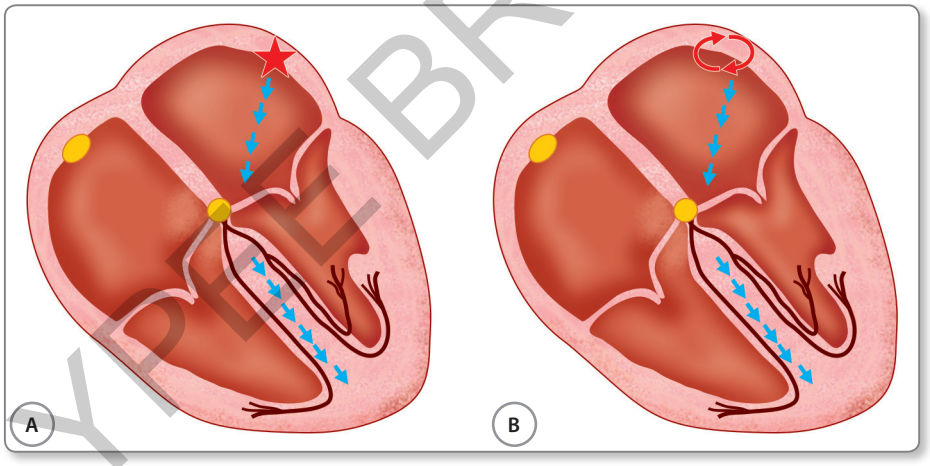
tissue that begins to spontaneously depolarize at an abnormal fast rate (Figure 4.2A). As you may remember, when a part of the heart begins to spontaneously depolarize at an abnormally fast rate we call this **enhanced automaticity**. Atrial tachycardia may also occasionally occur when a **reentrant circuit** develops in the atria (Figure 4.2B). As you may recall, the term "reentrant circuit" denotes any arrhythmia in which an electrical impulse repeatedly goes around and around part of the heart, causing an arrhythmia.

Whether the arrhythmia is caused by enhanced automaticity or a reentrant circuit, the depolarization impulse spreads across the atria, leading to depolarization of the atria. Depolarization of the atria leads to a P wave being

Heartman's Clinical Pearl

Remember, since sinus tachycardia is almost always a physiological response to some stress, one almost never tries to simply treat the tachycardia itself (such as with medications that slow the SA node). Rather, the key in patients with a persistent sinus tachycardia is to identify and treat the underlying cause of the sinus tachycardia.



Figures 4.2A and B Atrial tachycardia is usually caused by enhanced automaticity of an area of atrial tissue (A) but on occasion can be due to a reentrant circuit forming in the atria (B)

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seen on the ECG (Figure 4.3). However, because the atria are depolarized in a pattern different from that during sinus rhythm, the P waves will appear different in morphology from those seen during sinus rhythm. Depending upon where in the atria the arrhythmia develops, the P waves in some leads may appear inverted (as shown by the arrows in Figure 4.3). Once the depolarization has spread across the atria, the depolarization impulse continues down the AV node and into the ventricles. Depolarization of the ventricles leads to QRS complexes being seen on the ECG. Thus, in atrial tachycardia, we see abnormal or inverted P waves before each QRS complex, as is shown in the below rhythm strip (Figure 4.3).

Although the rate of atrial tachycardia can vary anywhere between 100–250 beats per minute, the heart rate most commonly seen in patients with atrial tachycardia is usually in the range of 160–180 beats per minute. Atrial tachycardia can occur in diseased hearts and in structurally normal hearts. Atrial tachycardia may be caused by digoxin toxicity. Very short runs (< 10–20 beats) of atrial tachycardia are not infrequently seen on Holter monitoring and are often not associated with symptoms, although more sustained episodes may cause symptoms such as palpitations.

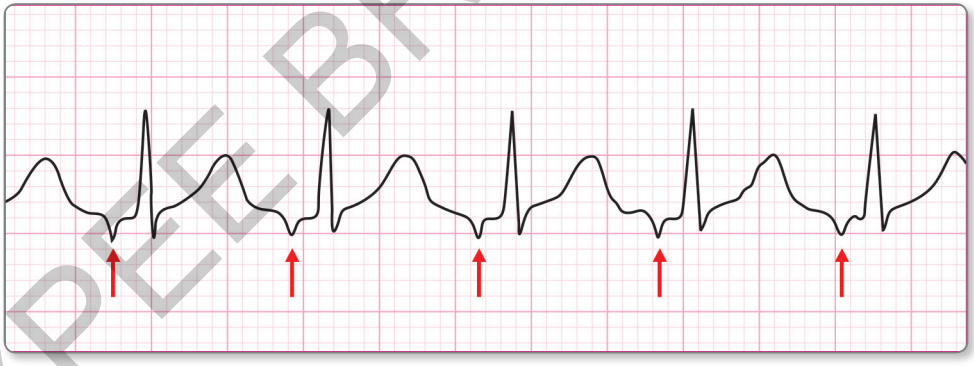

Figure 4.3 The ECG seen with atrial tachycardia. The P wave morphology with atrial tachycardia is different than the P waves that are seen with normal sinus rhythm, and in this case appear "inverted" in this telemetry strip

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Atrial tachycardia will usually resolve at some point on its own. Antiarrhythmic agents or other medications are usually not used to try to break the arrhythmia or slow the heart rate, since such drugs rarely acutely terminate the arrhythmia or slow conduction through the AV node. Atrial tachycardia will uncommonly be terminated by administration of adenosine. Since atrial tachycardia is more commonly the result of enhanced automaticity than of a reentrant circuit, cardioverting ("shocking") the patient usually does not terminate the arrhythmia.

MULTIFOCAL ATRIAL TACHYCARDIA

Multifocal atrial tachycardia (or "**MAT**" for short) is an arrhythmia that is caused by multiple sites of tissue in the atria depolarizing at different times (Figure 4.4). Each time one of these sites spontaneously depolarizes, the atria are depolarized. Because these different

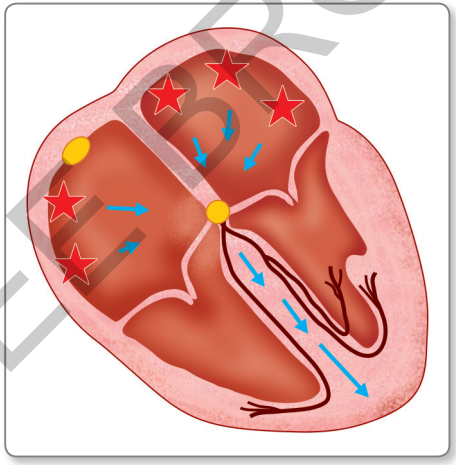

Figure 4.4 Multifocal atrial tachycardia (MAT). There are multiple areas in the atria in which the tissue has begun to spontaneously depolarize

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areas of depolarizing tissue are spontaneously depolarizing at different times, impulses reach the AV node and are conducted in to the ventricles at varying intervals, leading to an irregular heartbeat.

Because in MAT depolarization of the atria begins at different parts of the atria, the differing patterns of atrial depolarization leads to P waves of different morphologies (arrows in the Figure 4.5). Since spontaneous depolarization of different parts of the atria occurs at different times, the resulting rhythm is irregular (there are varying intervals between the QRS complexes). By definition, MAT is diagnosed when there are at least 3 distinct P wave morphologies and the heart rate is great than 100 beats per minute.

MAT most often occurs in patients with lung disease, particularly those with COPD. The usual "treatment" for MAT is to treat the *underlying cause* that has triggered it, such as a COPD exacerbation. Antiarrhythmic agents or other medications are usually not used to try to terminate the arrhythmia or slow the heart rate. Since the arrhythmia is caused by **enhanced automaticity** of tissues in the atria, "shocking" the patient will not terminate the arrhythmia.

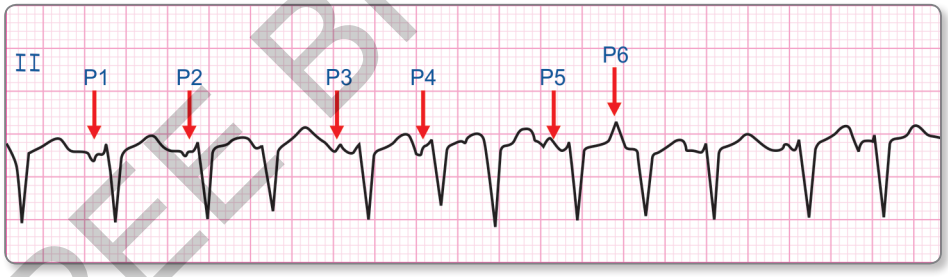

Figure 4.5 The ECG seen with multifocal atrial tachycardia (MAT). The rhythm is irregular and there are numerous P waves of differing morphology before each QRS complex. P1-P6 denote different P wave morphologies

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ATRIAL FLUTTER

Atrial flutter is another arrhythmia that occurs in the atria. An impulse travels around and around the atria in a repetitive loop, forming what is called a **reentrant loop** or more technically a **reentrant circuit**. This reentrant circuit is illustrated in Figure 4.6.

In most persons, the impulses travel around the reentrant circuit about 300 times per minute (with a range of 250–350 impulses per minute). Each time the impulse travels around the reentrant circuit, it depolarizes the atria, leading to the "saw tooth pattern" **flutter waves** seen in atrial flutter (Figures 4.7 and 4.8).

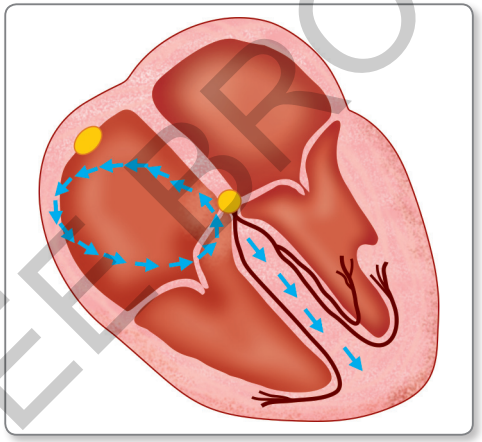

Figure 4.6. Atrial flutter. A reentrant circuit forms in the atria, leading to the arrhythmia

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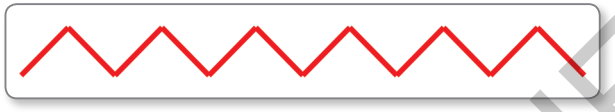

Figure 4.7 The "saw tooth" pattern on the ECG caused by atrial flutter



Figure 4.8 The "saw tooth" pattern of flutter waves that are seen in atrial flutter.

The AV node cannot conduct each of these 300 impulses per minute down into the ventricle. Instead, in many persons, it will conduct every other impulse down in the ventricle. When this occurs, we say that there is 2:1 AV conduction, meaning that for every 2 depolarizations in the atria, there is 1 depolarization of the ventricle. Thus, in many people who experience atrial flutter, the actual flutter rate is 300 beats per minute, but the ventricular rate is 150 beats per minute. In some people, if the AV node is "sick" or diseased, or if that person is on medications that slow conduction in the AV node, the AV node will conduct even fewer impulses from the atria down into

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the ventricle. Such persons may have 3:1 conduction or even 4:1 conduction, leading to ventricular rates of 100 or 75 beats per minute. In other persons, there may be a varying degree of conduction, leading to an irregular ventricular rate. We will discuss this more in a later chapter.

Although atrial flutter occurs in different parts of the atria, it most commonly occurs in the lower part of the right atrium. A wide spectrum of diseases is associated with the occurrence of atrial flutter. Depending on the ventricular response rate, patients may be asymptomatic or may experience symptoms such as palpitations, lightheadedness, shortness of breath, or chest discomfort.

When atrial flutter occurs, there is not the normal, forceful, contraction of the atria. Because of this, patients with atrial flutter are considered to be at risk for forming a clot (more technically called a **thrombus**) in a part of the left atrium called the **left atrial appendage.** Because there is a small chance that such a clot can **embolize** into the circulation and to the brain, patients with atrial flutter are considered to be at risk of having a stroke.

There are two basic approaches to the patient with atrial flutter. If the ventricular response rate is too fast, then the patient can be treated with medications that decrease conduction of impulses from the atria through the AV node into the ventricles. Medications that decrease conduction of impulses through the AV node include **beta blockers** (such as metoprolol, atenolol and carvedilol), certain **calcium channel blockers** (diltiazem and verapamil), and **digoxin**. The other approach is to shock (or more technically electrically **cardiovert**) the patient out of atrial flutter and back into normal sinus rhythm.

Long-term treatment to prevent the recurrence of atrial flutter includes either the use of antiarrhythmic agents or electrical ablation of part of the tissue involved in the reentrant circuit.

ATRIAL FIBRILLATION

Atrial fibrillation is a strange arrhythmia that also occurs in the atria. In atrial fibrillation, multiple "wavelets" of depolarization are constantly forming and reforming in the atria (Figure 4.9). As a result, there is no organized depolarization of the atria, and rather than contract in any organized manner, the atria just "fibrillate". Some of the many impulses generated by these wavelets of depolarization reach the AV node, and some of these are conducted down the atria into the ventricles. Because these depolarization impulses reach the AV node at irregular intervals, and because the AV node conducts some of these impulses down into the ventricle at irregular intervals,

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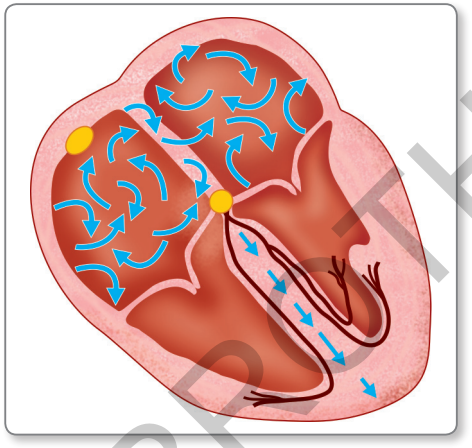

Figure 4.9 The multiple "wavelets" of depolarization impulses in atrial fibrillation

ventricular depolarization and contraction occurs at irregular intervals. Thus, with atrial fibrillation, there is what is called an **irregular ventricular response rate**, similar to what we see with MAT. In some patients with a healthy AV node, the ventricular response rate can be in the range of 150–180 beats per minute. In those with sicker AV nodes or those on medications that decrease conduction through the AV node, the ventricular response rate will be slower.

Because in atrial fibrillation there is no organized depolarization of the atria, there are no P waves seen on the ECG. Rather, there are only numerous, random, and varying small bumps (or deflections) of the ECG tracing. These varying deflections are called **fibrillatory waves** (Figure 4.10).

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Figure 4.10 The ECG in atrial fibrillation. No P waves or organized atrial activity is evident. The small random deflections of the ECG baseline are due to fibrillatory waves

There are numerous causes of atrial fibrillation. Atrial fibrillation not infrequently occurs in persons with dilated atria and in those with heart failure. Table 4.2 lists some of the important causes of atrial fibrillation to consider when you encounter a patient who has developed atrial fibrillation.

Table 4.2: Conditions associated with the development of atrial fibrillation

Dilated atria	Pericarditis
Heart failure and cardiomyopathy	Pulmonary embolism
Acute myocardial ischemia	Hyperthyroidism
Coronary artery disease	Toxins (including alcohol)
Hypertension	

In some patients, atrial fibrillation is believed to be triggered by impulse formation in one of the 4 **pulmonary veins** that drain blood from the lungs to the left atrium. The impulse begins in the pulmonary vein and is then propagated throughout the atria, leading to atrial fibrillation. This impulse initiation and spread is illustrated in Figure 4.11. In such patients, a procedure called a **pulmonary vein ablation** may be performed in order to prevent atrial fibrillation from recurring.

Since there is no organized contraction of the atria, patients with atrial fibrillation are at risk of forming a blood clot ("thrombus") in the part of the atria called the **left atrial appendage**. As with atrial flutter, such patients have a small but real risk of having such a blood clot embolize to the brain, causing a stroke. To decrease the risk of blood clots forming in the left atrial appendage of the heart, patients

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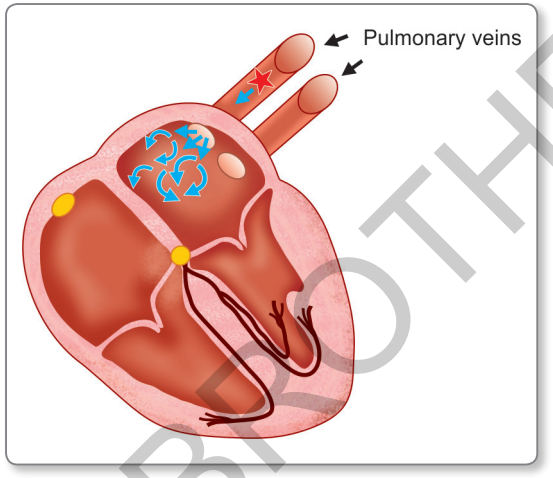

Figure 4.11 An impulse originating in the pulmonary vein, which then spreads through the atria, is the cause of many cases of paroxysmal atrial fibrillation.

with atrial fibrillation are often treated with blood thinners such as **warfarin (Coumadin)** or newer blood thinners such as **dabigatran (Pradaxa)**, **rivaroxaban (Xarelto),** and **apixaban (Eliquis).**

As with atrial flutter, there are two basic approaches to the patient with atrial fibrillation. If the ventricular response rate is too fast, the patient is treated with medications that decrease conduction through the AV node, such as **beta blockers**, certain **calcium channel blockers**, and **digoxin**. The other approach to patients with atrial fibrillation is to shock (or more technically electrically **cardiovert**) the patient out of atrial fibrillation and back into normal sinus rhythm. Unfortunately, many patients who develop atrial fibrillation will eventually go back into atrial fibrillation, even if successfully shocked back into normal sinus rhythm.

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