

# Original Article: Paroxysmal Atrial Tachycardia (PAT)

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## ABSTRACT

It is a type of paroxysmal supraventricular tachycardia that appears suddenly following a sinus rhythm and drains suddenly. This rhythm may be caused by the rapid evacuation of an atrial fibrillation at a rate of 160-250 beats per minute, or conduction problems in the AV node, called AV node rotational tachycardia. In this way, an impulse is directed to an area of the AV node that causes the impulse to return repeatedly and rapidly from the same area (they have a shorter excitation phase) and each time the impulse is directed from this area to the ventricular side is also moved, causing a rapid ventricular response. Atrial tachycardia can be detected by three or more irregular atrial beats in a row with a rate of 250-220-250 beats per minute alternating with a normal sinus rhythm. Although PAT is also seen in clients with a healthy heart, it is often an underlying heart disease. Caffeine, nicotine, stress, hypoxemia, extreme fatigue, alcohol consumption, rheumatic heart disease, pulmonary embolism, cardiopulmonary disease, thyrotoxicosis, digitalis intoxication and heart surgery can trigger PAT. PAT reduces ventricular filling time and mean arterial pressure and increases myocardial oxygen demand. Clinical symptoms vary according to the rate and duration of tachycardia and the underlying cause. If the duration of dysrhythmia is short, the patient complains of palpitations and confusion. But if it reduces cardiac output, signs and symptoms such as restlessness, chest pain, shortness of breath, paleness, hypotension and decreased level of consciousness occur.

## Introduction

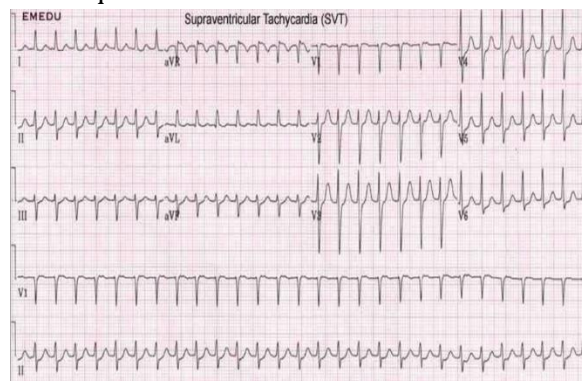
**T**he goal of treatment is to break the impulse cycle and is based on vagal stimulation [1-3]. Vagal stimulation is performed by stimulating the gag reflex, Valsalva maneuver, immersing the face in ice water [4-6], reducing the AV node conduction velocity and blocking impulse

rotation [7-9], and allowing the SA node to reactivate as the main pacemaker [10-12]. The above measures were not effective. Another method of vagal stimulation is used by carotid sinus massage [13-15]. Carotid sinus massage must have an open venous passage. This is contraindicated in the elderly and those with carotid hearing loss [16-18], as it may cause atrophic plaques in the carotid artery and

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cerebral embolism to rupture [19-21]. To perform a carotid massage, the patient should be placed on a flat surface and his neck should be bent backwards [22-25]. Massage of a carotid bulb in one step should be done by applying firm pressure just below the jaw angle for more than 5 seconds [26-28]. If the above methods are not effective, drug therapy is used, which includes the use of sedatives [29-31], adenosine attack dose, verapamil or diltiazem. When injecting verapamil to the patient, the drug needs to be diluted and the rhythm monitored [32-35]. This drug works by preventing the entry of calcium in the second phase of repolarization and by slowing down the input flow of calcium and sodium reduces the conduction velocity and prolongs the excitatory cycle of the AV node and supraventricular tachycardia in which the AV node plays the role of re-entry path. Opens, terminates [36-38]. If there is a large blockage of the calcium channels, there is a possibility of hypotension and cardiac arrest following the use of verapamil, and calcium injection should be given immediately [39-41]. The dose of verapamil is 5 mg diluted in 10 cc of normal saline and injected over 5 minutes. Adenosine is also one of the judges that slows down the conduction velocity of the AV node and inhibits the ways of re-entry into the AV [42-45], but due to the fact that the half-life of adenosine is less than 10 seconds and there is a possibility of recurrence, use Verapamil is preferred. The initial dose of adenosine 6 mg is an intravenous intravenous dose of 1-3 seconds after the injection of adenosine 20 ml of normal saline is injected into the patient's vein. If drug therapy fails [46-48], a temporary intravenous pacemaker is used to produce pulses faster than the PAT. This action breaks the re-entry cycle and by stopping the atrial tachycardia, we will have natural impulses through the SA node, which is called overdrive with a pacemaker [49-51]. Another method is to use cardioversion shock, before which the patient is sedated with

the drug and given 200-50 joules of shock. If the PAT is associated with Wolff-Parkinson-White syndrome, the arrhythmia-producing center can be destroyed using the catheter ablation technique.



**Figure 1.** PAT

### Atrial flutter

In atrial flutter, saw-like waves are seen in the lower leads (float line) and the vestibular velocity is 250 to 350 beats per minute. Due to the blockage in the AV node, not all atrial impulses are usually transmitted to the ventricles [52-55]. Atrial-ventricular conduction often has a fixed ratio. For example, 2 to 1, 3 to 1, 4 to 1 and so on. Sometimes the occurrence of a variable ratio leads to the production of an irregular rhythm [56-58]. Rarely, 1-on-1 guidance can cause tachycardia to occur very rapidly, in which case it may indicate a secondary conduction pathway. In the atrial float, due to the rapid and sudden activity of a highly excitable atrial foci (in the speed range of 250 to 350 times per minute), the depolarization waves are arranged in the same way as sawtooth teeth [59-61], and the baseline between the waves disappears. Has been [62-65]. The AV node has a long period of excitability, and atrial depolarizations are not able to reach the ventricles at this very high rate [66-68]. Therefore, although atrial depolarizations (for example, two or more commonly three atrial depolarizations) [69-71], only one is directed to the ventricles. The rapid sequence of similar and tandem atrial depolarization waves in the atrial

float reinforces the suggestion of a reversible origin [72-75].

### Reasons for flotation analysis

Ischemic heart disease, systemic hypertension, valvular heart disease, corpulmonary, cardiomyopathy, thyrotoxicosis, congenital heart disease [76-78].

Due to the high excitability time of AV node cells, atrial signals are not able to enter the ventricle, so 3 to 1 or 2 to 1 rhythms are seen in the atrial float [79-81].

### Atrial float rhythm characteristics

- ✓ Rate: Atrial rate between 150-300 times per minute, ventricular rate usually 75-75 times per minute [82-85].
- ✓ Rhythm: Regular atrial rhythm and ventricular rhythm may be irregular due to conduction changes in the AV node.
- ✓ P-waves: In the atrial float, it is known as F-waves and appears as a serrated tooth.
- ✓ P-R distance: The presence of multiple P-waves makes it difficult to measure [86].
- ✓ QRS Complex: Normal, but may be abnormal or unresponsive to some F-waves [87-89].
- ✓ Conductivity: Some blocked F-waves and passing waves have a natural conductivity, and the conductivity is relatively 3: 1 or 4: 1.

### Treatment

People with this dysrhythmia are often unaware. Unless they sometimes feel a heartbeat. Digoxin, verapamine, diltiazem [90-92], beta-blockers and amiodarone can be prescribed to return the floater rhythm to the sinus rhythm. In the case of digoxin, an additional dose should be used, which is 0.5-2-1.5 mg, which is injected every half an hour IV [93-95], and then continued with a daily dose of 0.25 mg until the rate of The ventricle reaches 60-100 beats per minute. Cardiogenic synchronization is essential when there are hemodynamic abnormalities [96-98].

### Nursing measures

Record dysrhythmia and control the signs and symptoms of decreased cardiac output. If needed, prepare the patient for cardioversion, establish an open venous passage for the patient, and place CPR equipment next to the bed [99].

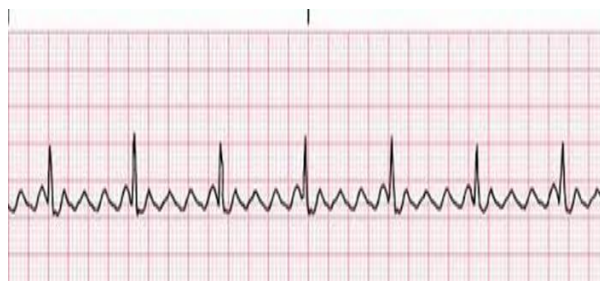


Figure 2. Af

### Atrial fibrillation

Atrial fibrillation is caused by the activation of several ectopic foci in the atria, which causes rapid, irregular, and uncoordinated contraction of the atrial muscle structure. In this dysrhythmia, there is no atrial contraction and 30-30% of cardiac output is reduced [100-102].

### Cause of atrial fibrillation

Heart failure, cardiomyopathy, hyperthyroidism, hypertension, pericarditis, valvular heart disease, lung disease, ecstasy use, following open heart surgery [103].

### Electrocardiographic symptoms of atrial fibrillation

- ✓ Rate: Between 300-600 beats per minute. The ventricular rate depends on the AV node block and is about 100-150 without treatment [104].
- ✓ Rhythm: Both atrial rhythm and ventricular rhythm are irregular. If the ventricular response is regular despite atrial fibrillation, ventricular atrial segregation should be suspected [105].

- ✓ P-waves do not exist, irregular waves are seen, which are called fibrillation waves or F-waves [106].
- ✓ P-R distance: Not measurable due to lack of P waves.
- ✓ Complex: QRS is usually normal, but may be abnormal.
- ✓ Conduction: The transmission of impulses inside the atrium is very irregular. Many atrial impulses are blocked at the junction junction, and impulses that are transmitted are usually normally transmitted across the ventricles [107-110].

### Treatment of atrial fibrillation

Treatment for atrial fibrillation involves three steps:

#### A) Ventricular response control:

#### B) Prevention of thromboembolism:

#### C) Establishing a sinus rhythm:

**A) Ventricular response control:** It is necessary to control the rapid ventricular response immediately, which is based on the reduction of the conduction of the ventricular atrial node (AV node). There are several drugs available for this purpose: Beta-blockers directly reduce the conduction velocity of the ventricular atrial node and are used by injection or orally and are the drug of choice in patients with coronary artery disease. Calcium channel blockers such as verapamil and diltiazem are effective in reducing the conduction of the ventricular node. Dihydropyridine calcium channel blockers, such as nifedipine, have no effect on the conduction of the ventricular node and are not used in this case. The effect of digoxin on the ventricular atrial node is to reduce the ventricular response by increasing the vagal effect. Therefore, after taking digoxin, the ventricular response is appropriate at rest, but during exercise, due to the effect of catecholamines, the ventricular response increases, so it is better to use digoxin with

verapamil. Adenosine is not used in this case due to its short-term effect. In patients with heart failure, the drug of choice is to control the ventricular response to digoxin.

Warfarin is essential in patients with AF rhythm who have a high risk of thromboembolism:

- ✓ Previous CVA or TIA.
- ✓ Systemic hypertension.
- ✓ Heart failure (LV dysfunction).

Patients over the age of 65 in whom the chances of bleeding due to warfarin use are high and should be closely monitored. 2 to 3 is appropriate when taking warfarin INR. If the patient cannot take warfarin, the use of aspirin is also somewhat effective in reducing vascular attacks [111-113].

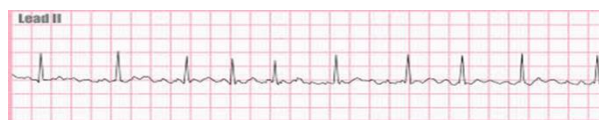


Figure 3. AF

### Junctional and ventricular dysrhythmias

The cells around the AV node and the AV junction are automatic and capable of generating impulses. Junctional arrhythmias include early junctional complexes, junctional rhythm, and junctional tachycardia. The junction wave and diatom are manifested in three ways in the ECG, which depends on the location of the impulse from the junction and the speed at which the impulse travels to the atrium and ventricle [114-116]. When the AV junction begins to generate impulses, the resulting impulse travels toward the atrium and ventricle, and the atrium depolarizes from bottom to top and ventricle from top to bottom, and the P wave can have the following states:

- ✓ If the depolarization wave depolarizes the atria first and then the ventricles, considering that the atria are depolarized from the bottom up and before the ventricles, we will have the reverse P wave before QRS and the PR

interval is less than 0.12 seconds. Is. This rhythm has a high junction origin [117].

- ✓ If the depolarization wave depolarizes the atria and ventricles simultaneously, the P wave will merge in the QRS and will not be visible. This rhythm has a mid-junction origin.
- ✓ If the depolarization wave moves from the junction and depolarizes first the ventricles and then the atria, we will have a QRS complex first and then a reverse P wave. This rhythm has a low junction origin [118-120].

### Premature functional complex (PJC) Premature functional complex

If a sensitive foci in the AV junction area act like a pacemaker and issue an early impulse, an ectopic pulse is generated, in all cases the atrium is retrograde from the bottom up and naturally depolarized [121-123]. In terms of electrocardiographic characteristics, we will have one of the three forms of junctional rhythm mentioned above early in the field of sinus rhythm. In PJC, it has all the PAC criteria, it has all the criteria, except that their PR wave and distance are different. That is, the P-wave may be before or after the QRS or not at all, and the P-R interval is less than 0.12 seconds [124-126].

### Causes of PJC

Excitement, smoking, nicotine, digital poisoning, congestive heart failure, and coronary artery disease (ischemia junction) are symptoms and treatment similar to PAC.

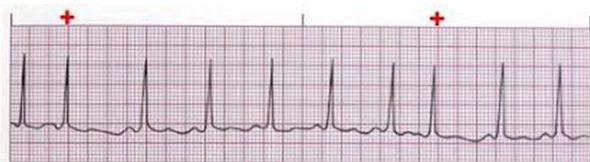


Figure 4. PJC

### Adjust the ventilator

This is usually done by setting specific indices and selecting the ventilator method. First, we set

it to 100% and after 20min we adjust it depending on the ABG answer. Next, we aim at 90% saturation pulse oximetry and reduce Fio<sub>2</sub>. Application of Peep to high levels is more than (10-20 cmH<sub>2</sub>) risk of barotrauma and hypotension. Therefore, we usually use values less than 0 10cmH<sub>2</sub>, and if values higher than 10cmH<sub>2</sub> are necessary, the pulmonary capillary pressure must be monitored with a Gens catheter. Peep discontinuation occurs when the patient has adequate oxygenation with or less than 40% oxygen with Fio<sub>2</sub>. It does not stop abruptly, but in a few steps, we reduce the Peep pressure and control it with pulse oximetry.

**A) ABG:** half an hour after any change in ventilator setting and every hour periodically up to 24 hours and then the decision is based on the patient's clinical condition [127-129].

**B) Daily chest x-ray:** evaluation of the location of the endotracheal tube and pneumothorax.

**C) Oxygen saturation:** Regular pulse oximeter is usually recommended [130-132].

**D) Tracheal tube cuff pressure:** up to 15. 5 mmHg, according to hemodynamic parameters, water and electrolyte. There are three main methods for hot extrusion of powder mixtures (which include some cermet's). The first method can be used for coarse and loose magnesium powder or pellets [133-136]. The materials are poured into the hot extrusion chamber and are directly extruded through the mold. In this method, the protective atmosphere is not used and the heat of the chamber increases the temperature of the powder to provide the extrusion conditions. The temperature should be increased between 15 and 30seconds in order to drive the hammer forward before extrusion. In the second method, cermet powder mixtures with aluminum base are transformed into ironed pellets by cold pressing and hot compaction. These pellets are then hardened in normal ways [137-139]. A third method that is widely used consists of steel cans filled with cold powder mixes and the mixes are compressed by

penetrating punches while the cans are held in a set of molds. (Pre-compacted mixes can be used instead of powdered mixes, in which case the compacting step is eliminated). A fastener is welded on the open end of the can and the can is evacuated. Then the can will be heated and the shape will be changed (extruded). On the other hand, a penetration punch can force the open can and powder mixture during the extrusion die. In both ways of this method, the extruded product is covered with a thick layer of the material used for packaging. This layer must be removed by etching or mechanical peeling.

### PAT electrocardiographic symptoms

- ✓ Rate: Atrial rate is usually 160-250 beats per minute and ventricular rate is equal to or slower atrial rate.
- ✓ Rhythm: Regular.
- ✓ P-waves: It is different in appearance from sinusoidal P-waves because its focus is out of place, but due to its integration with the previous T, it is very difficult to detect.
- ✓ The P-R distance is less than 0.12 seconds.
- ✓ QRS Complex: Normal, but if there is an ectopic connection, the QRS may appear wide.
- ✓ Conduction: Normal conduction from the AV node to the ventricles is normal, but in atrial tachycardia with ventricular block, impulses sent from the atrium into the ventricles are not routed. Absolute excitability phase of the previous impulse.

### Conclusion

Hot extrusion is an attractive molding and densification process for cermet's. Unfortunately, the applications of this method in this wide field have serious limitations due to the load of non-metallic materials, the selection of metallic components and the degree of

internal activity between the two materials. Definite limits on the volume of non-metallic materials do not pose a problem. Rather, when these materials increase from 18 to 25 percent of volume, the composite material shows thermal self-shrinkage to the extent that problems such as edge breakage, internal warping, and elongation of the extruded piece become unacceptable. When the non-metallic phase is present in the form of very fine-grained powders, the shape of thermal shortening worsens; And also it is intensified when the non-metallic substance is combined with the metallic component at the extrusion temperature to create new phases or eutectoids. How these complex problems are solved for each specific pair of cermet components is in the field and scope of this article. A careful study of the structure diagram of the metal binder and the hard phase is recommended before any serious operation. As with metal alloy pellets, cermet pellets are easier to extrude when aluminum or aluminum alloys are used for the metal components. Condensation and pellet extrusion occur at a lower temperature and pressure.

The process of canning and removing cans, which is expensive, can be easily eliminated. A protective atmosphere can also be used. In addition, smoothing and finishing of the extruded product can often be done at room temperature. Thermal shrinkage defects in extruded aluminum-based products only occur at high levels of hard material loading. Compared to cermet's containing aluminum or aluminum alloys, cermet's containing iron, nickel, cobalt or alloys of these metals are technically more dependent and more expensive to extrude. For example, pellet heating and hot pressing require a controlled atmosphere, pellet canning is unavoidable, and a verification process is required to lubricate and reduce die coating, smoothing, final rolling and the like also require expensive processes. An extruded cermet product can be expected to be harder,

looser, less malleable and more brittle than the bonded metal component. Because the high temperature of extrusion causes a metallurgical reaction between the alloy or base metal and the cermet hard phase component, especially if the product is a non-metallic compound from the carbide or boride group. Oxide-based hard phase components seem to react less with the metal component. The unwanted reaction between the hard material and the cermet metal component is more easily controlled by the solid state extrusion process than by any other densification process involving a liquid phase. Also, despite the aforementioned limitations and problems, hot extrusion continues to attract the attention of product development engineers as a possible production method for certain cermet's, especially those with an aluminum base. This method has the ability to be cheap in terms of labor cost, but it still requires a lot of capital for the product manufacturing process for high-tech pipe or rod materials.

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