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Definition, Diagnosis and Treatment of Tachycardia

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1. Introduction

1.1 Anatomy of the human cardiac conduction system

1.1.1 Sinoatrial node

Sinoatrial node (SAN) serves as the natural pacemaker of the heart. It is a spindle shaped structure that is located at the junction of the superior vena cava and right atrium.

The electrical excitation during sinus heart rhythm originates from the SAN and spreads to the other regions of the heart. It receives arterial supply from either the right coronary artery (60 % of the time) or the left circumflex coronary artery (40-45% of the time). It is innervated with postganglionic adrenergic and cholinergic nerve terminals. The discharge rate of SAN is modulated by stimulation of beta adrenergic and muscarinic (cholinergic) receptors. Stimulation of beta-1 and beta-2 receptors results in positive chronotropic response where as stimulation of muscarinic M2 receptors results in negative chronotropic response.

1.1.2 Atrioventricular node

Atrioventricular node (AVN) is located right above the insertion of the septal leaflet of the tricuspid valve beneath the right atrial endocardium. It receives arterial supply from the AV nodal branch of the right coronary artery (85%-90% of the time) or left circumflex coronary artery (15 % of the time). Action potential from the SAN travels through the atrial myocardium to the AVN. The AVN serves to delay the atrial impulse transmission to the ventricles and thus coordinates atrial and ventricular contractions.

1.2 Bundle of His and bundle branches

Bundle of His is the continuation of the penetrating portion of the atrioventricular bundle on the ventricular side. It divides into the right and left bundle branches. It is supplied by the left anterior descending and posterior descending arteries. Thus it is relatively protected from ischemic damage.

The bundle branches begin at the superior margin of the muscular interventricular septum and divide into the left bundle branch, which may further divide into the anterosuperior

branch and central branch and the right bundle branch, which traverses along the right side of the interventricular septum.

1.3 Purkinje fibers

These fibers form an interweaving network on the endocardial surface of the myocardium and are connected to the ends of the bundle branches. They conduct the cardiac impulse simultaneously to both the ventricles.

1.4 Normal sinus rhythm

Normal sinus rhythm refers to generation of impulse from the SAN at a rate of 60-100 beats/min in an adult (Fig 1). In adults, rates below 60 beats/min are referred to as bradycardia and rates above 100 beats/min are referred to as tachycardia. The normal sequence of electrical activation originates from the SAN and spreads through the atria to the AVN and His Purkinje system and finally to the ventricular myocardium.

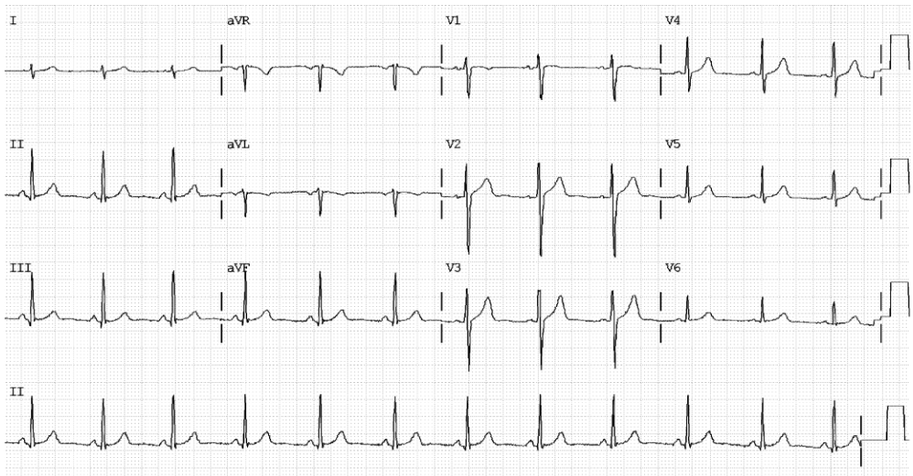


Fig. 1. Normal sinus rhythm

2.Tachycardia

Tachycardias are characterized on the basis of origin; those that originate above the ventricle are referred to as supraventricular tachycardias (SVTs) and those that originate from the ventricle or purkinje fibers are characterized as ventricular tachycardias. The distinction between the two types of tachycardias is critical at the beginning due to difference in their prognosis. Ventricular tachycardias overall have grave prognosis and usually result from significant heart disease. On the other hand, SVTs are usually nonlethal and have a more benign prognosis.

2.1 Narrow and wide complex tachycardia

Based on the duration of QRS complex on electrocardiogram (ECG), tachycardias can be divided into narrow and wide QRS complex tachycardias. Narrow QRS complex tachycardias have a QRS complex duration of less than 120 ms and wide QRS complex

tachycardias have a QRS complex duration of more than 120 ms. Wide complex tachycardias are due to ventricular origin of the tachycardia. Ventricular origin of the tachycardia results in slow conduction of electrical impulses across the ventricular myocardium due to longer conduction velocity of the ventricular myocardium unlike narrow complex tachycardia which are conducted over the normal conduction system which has faster conduction velocity resulting in narrow QRS complex on the surface electrocardiogram. Wide complex tachycardia however can be supraventricular in origin if there is pre-existing bundle branch block in either of bundles or if there is activation of the ventricles over the accessory bypass tract and subsequent spread of electrical activation through the ventricular myocardium resulting in a wider QRS complex.

2.2 Sinus tachycardia

Sinus tachycardia as the name suggests originates from the SAN and has a rate of more than 100 beats/min (Fig 2).

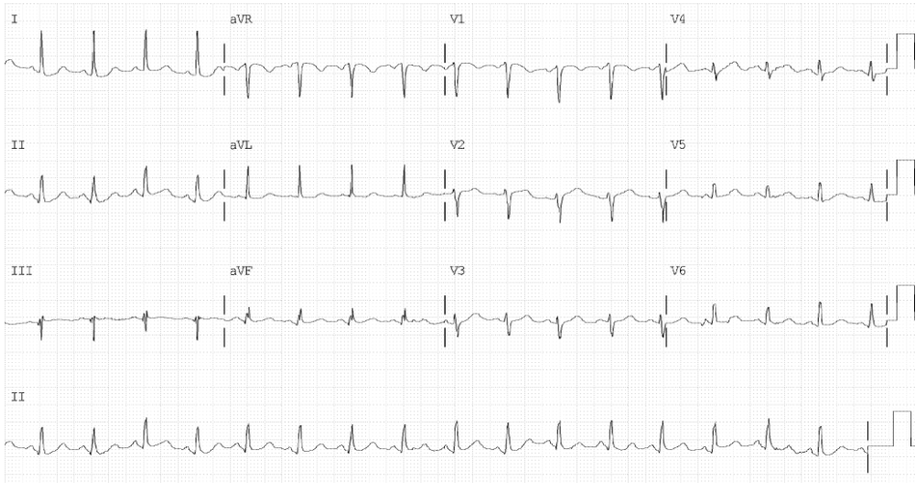


Fig. 2. Sinus tachycardia

It has a gradual onset and termination. It is characterized by presence of sinus P waves prior to each QRS complex on ECG. It is usually caused by increase in adrenergic discharge or decrease in parasympathetic discharge.

It is common during infancy and childhood. The maximum heart rate achieved is higher in young individuals and decreases with age. In adults, it occurs in response to various physiologic or pathological stresses such as exercise, fever, anxiety, thyrotoxicosis, anemia and shock. It may result from consumption of drugs such as atropine, amphetamines, caffeine and alcohol. Sinus tachycardia is often benign by itself and is usually a manifestation of underlying causes as mentioned above. Thus treatment of sinus tachycardia requires treatment of the underlying cause.

2.3 Inappropriate Sinus Tachycardia (IST)

Is a syndrome that can occur due to increased automaticity of the SAN or an automatic atrial focus present near the SAN. It may occur due to an imbalance in the vagal and sympathetic

control of the SAN. Treatment of IST involves treatment with beta-blockers or calcium channel blockers that decrease the SAN automaticity. Radiofrequency ablation of the SAN may be indicated in severe drug-refractory cases of inappropriate sinus tachycardia.

2.4 Postural Orthostatic Tachycardia Syndrome (POTS)

Refers to orthostatic decrease in blood pressure and sinus tachycardia in the absence of drugs or hypovolemia (1).

3. Atrial Fibrillation

Atrial fibrillation (AF) is a type of supraventricular arrhythmia characterized by presence of low amplitude fibrillatory waves on the ECG (Fig 3). The fibrillatory waves exhibit variable amplitude and shape and are placed irregularly. They are generated at a rate of 300-600 beats/min. The resulting ventricular rhythm is irregularly irregular. However, the ventricular rhythm may be regular in AF in the patients with third degree atrioventricular block with an escape pacemaker or artificial pacemaker.

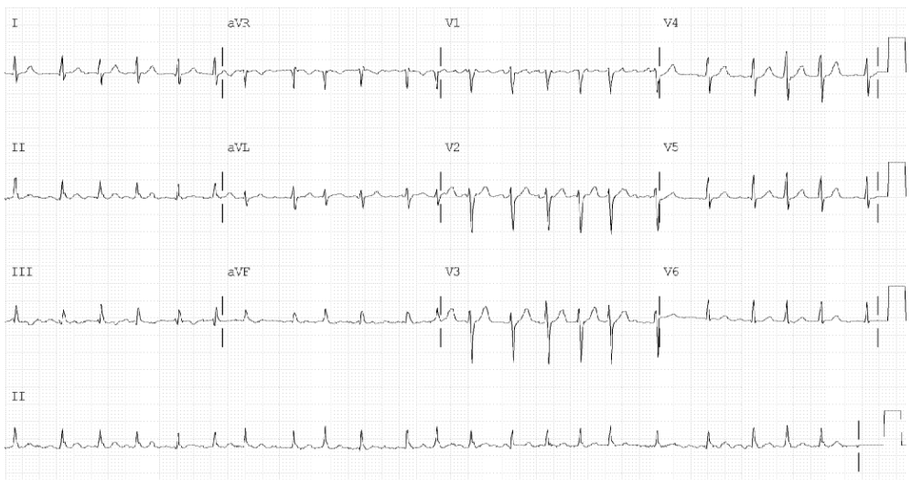


Fig. 3. Atrial fibrillation

3.1 Classification

AF is considered to be *paroxysmal* if it terminates spontaneously within 7 days. If it continues for more than 7 days, it is considered to be *persistent. Permanent* AF persists for more than a year and is resistant to cardioversion. *Lone* AF is a term used to describe the occurrence of AF in patients younger than 60 years of age that do not have hypertension or evidence of any structural heart disease.

3.2 Epidemiology

AF is the most common cardiac arrhythmia encountered in clinical practice. It is also the most common cause of hospitalization among all the arrhythmias. Advanced age, obesity, hypertension, congestive heart failure, mitral and aortic valve disease are independent risk factors for development of AF.

3.3 Causes of AF

Hypertension and hypertensive heart disease is by far the most common cause of AF. Other causes include ischemic heart disease, mitral valve disease, hypertrophic cardiomyopathy, dilated cardiomyopathy, pulmonary hypertension, and obesity with resultant obstructive sleep apnea. AF may be caused by reversible temporary causes such as excessive alcohol intake *(holiday heart syndrome),* myocardial infarction, pulmonary embolism, thoracic surgery, pericarditis and myocarditis. Hyperthyroidism is one of the correctable causes of AF.

3.4 Clinical features

AF may be symptomatic or asymptomatic. The most common symptoms of AF are palpitation, shortness of breath, tiredness and lightheadedness. It is often asymptomatic in elderly patients and those with persistent AF. Patients with AF may experience syncope at the conversion of AF to normal sinus rhythm from long sinus pause. Cerebrovascular accident (CVA) may be the initial manifestation of AF in patients with asymptomatic AF due to predisposition to thrombus formation in the atria, predominantly in the left atrial appendage due to chaotic and asynchronous atrial activity. It may also manifest as congestive heart failure due to tachycardia induced cardiomyopathy.

3.5 Physical examination

On physical examination, patients with AF often have irregularly irregular pulse. Patients often have pulse deficit, which is discordance between peripheral pulse and apical impulse. It results from short diastolic filling period for the ventricles during rapid AF resulting in low stroke volume and absence of peripheral pulse. There is variability in the intensity of the first heart sound on auscultation due to variable diastolic filling of the ventricles.

3.6 Diagnostic evaluation

History should be directed to assess the nature of AF (paroxysmal or persistent), nature of triggers, correctable causes such as excessive alcohol or caffeine intake, severity, frequency and duration of episodes of AF.

Twenty-four hour Holter monitoring may be helpful in patients with frequent episodes of AF whereas an event monitor would be helpful in patients with sporadic symptoms for detection of the episodes of AF.

Laboratory evaluation should include thyroid function tests to evaluate the thyroid function as a cause of AF. An echocardiogram is helpful to determine the left atrial size, left ventricular function and valvular heart disease. A stress test may be helpful for evaluation of ischemic heart disease as a cause of AF.

3.7 Thromboembolic complications

As described above, AF predisposes to thromboembolic complications such as CVA. However, the risk of these complications is not the same in all the individuals with AF. Thus it is imperative to stratify the risk of developing these complications. The risk for CVA is highest in patients with prior history of ischemic CVA and mitral stenosis whereas it is

lower in patients with lone AF. Gage et al developed a simple clinical scheme to stratify the patients on the basis of major risk factors. It is known by the acronym *CHADS-2* that includes *C*ongestive heart failure, *H*ypertension, *A*ge ≥ 75 years , *D*iabetes mellitus and previous *S*troke. Each of the first four risk factors is assigned 1 point and prior ischemic stroke or transient ischemic attack is assigned 2 points (2). There is a direct relationship between the annual risk of stroke and CHADS-2 score (Table 1).

Score	Annual risk (%)
0	1.9
1	2.8
2	4.0
3	5.9
4	8.5
5	12.5
6	18.2

CHADS-2 score is calculated by adding 1 point for each of recent congestive heart failure, hypertension, age 75 years and older, diabetes mellitus and 2 points for history of stroke or transient ischemic attack.

Table 1. Annual stroke risk according to CHADS-2 score (2).

The risk of thromboembolic complications is similar in patients with paroxysmal and persistent AF (3).

3.8 Prevention of thromboembolic complications

As discussed above, the risk of stroke increases with increase in the CHADS-2 score in patients with AF. The annual risk of stroke in patients with CHADS-2 score of 0 is approximately 1.9 %. In these patients, aspirin therapy alone is adequate due to the lower annual risk of stroke. Warfarin therapy is superior to aspirin in prevention of stroke (61 % vs. 18 %), however the risk of hemorrhagic complications is also higher and falls in the same range of 1-2 % annually (4, 5). Thus, considering the risk benefit ratio, aspirin therapy alone would be adequate in these patients.

In patients with CHADS -2 score of 1, the annual risk of stroke is 2.8 %. The decision to use aspirin alone or warfarin therapy should be individualized and patient preference may be taken into consideration. The dose of aspirin used for prevention of stroke ranges from 81-325 mg daily.

In patients with CHADS-2 score >1, aspirin therapy alone is inadequate to decrease the risk of stroke. These patients should receive warfarin for prevention of stroke. The target international normalized ratio (INR) should be 2.0-3.0 during warfarin therapy. The risk of major bleeding with warfarin is between 1-2 % annually as described above and it increases with INR >3.0 (6). Advanced age should not be a deterrent to starting warfarin therapy (7).

US-Federal Drug Administration (FDA) F recently approved direct thrombin inhibitors such as dabigatran for prevention of stroke in patients with non-valvular AF. It offers the advantage of fixed dosing and lack of the requirement for INR testing during therapy. Dabigatran given at a dose of 110 mg twice daily was associated with similar rates of stroke and systemic embolism as with warfarin and lower rates of major bleeding. When it was

administered at a dose of 150 mg twice daily, it was associated with lower rates of stroke and systemic embolism but similar rates of major hemorrhage (8).

4. Acute management of AF

In patients who present with an episode of AF and rapid ventricular response, control of ventricular rate should be the priority. It can be achieved with intravenous diltiazem or beta-blockers such as esmolol. However, if the patient is hemodynamically unstable, emergent transthoracic electric direct current (DC) cardioversion is appropriate.

The decision to restore sinus rhythm after control of ventricular rate should be individualized based on nature of AF (paroxysmal or persistent), duration of AF, presence of symptoms, age, prior episodes and antiarrhythmic and anticoagulation therapy.

The decision making for restoration of sinus rhythm in patients with AF has two components: one is early vs. late cardioversion and second is electrical cardioversion vs. pharmacologic cardioversion.

Early cardioversion is attempted in patients with AF lasting less than 48 hours and those who have been on therapeutic anticoagulation of 3-4 weeks prior to cardioversion. Late cardioversion is attempted in patients with left atrial appendage thrombus on transesophageal echocardiography (TEE), if the duration of AF is > 48 hours or if the duration is unclear and those with suspected correctable cause of AF. When the duration of AF is > 48 hours or unclear, the other option to avoid delayed cardioversion is TEE for surveillance of left atrial thrombus followed by cardioversion if there is no evidence of thrombus.

Cardioversion can be done using either electrical or pharmacologic means. Pharmacologic cardioversion can be performed by intravenous administration of ibutilide (success rate 60-70 %), amiodarone (success rate 40-50%) or procainamide (success rate 30 %). After infusion of ibutilide, patients should be monitored for polymorphic ventricular tachycardia that can occur as an adverse effect. In patients without structural heart disease, oral agents such as propafenone (300-600mg) or flecainide (100-200mg) can be administered for cardioversion. The success rate of transthoracic electrical cardioversion is very high (95 %). In some cases, electrical cardioversion may be required after administration of antiarrhythmics for maintenance of sinus rhythm.

4.1 Long-term management of AF

As far as long-term management of AF is considered, several studies have compared rate control (controlling ventricular rate) strategy vs. rhythm control (restoration of sinus rhythm) strategy. The largest of these trials (AFFIRM) failed to show any benefit of rhythm control strategy over rate control strategy. Rhythm control strategy in this study was associated with higher rates of hospitalization and adverse drug effects of antiarrhythmic therapy that were used for restoration of sinus rhythm (9). However, the decision to pursue rate control or rhythm control strategy is a complex one and should be individualized after taking several factors into account including, age, comorbidities, duration of AF, left atrial dimensions, presence of symptoms, response to prior cardioversions. In patients > 65 years of age with asymptomatic or minimally symptomatic AF, it would be reasonable to pursue

rate control strategy whereas symptomatic AF in younger and older age patients should be offered the option of rhythm control.

As far as antithrombotic therapy is considered, all patients with either paroxysmal or persistent AF should receive antithrombotic therapy with either aspirin or warfarin based on their risk stratification irrespective of their symptom status as described above.

4.2 Pharmacologic rate control

The goal for heart rate control in AF is to maintain heart rate less than 80 beats/min at rest and less than 110 beats /min during activity. Oral agents available for heart rate control in AF include digitalis, beta-blockers, calcium channel blockers and amiodarone. The first line agents for rate control are beta-blockers such as metoprolol and calcium channel blockers such as diltiazem and verapamil. Digitalis is useful to control heart rates due to its vagotonic effects but may not be adequate as a single agent for rate control during exertion. It is useful in patients with systolic heart failure due to its effects on prevention of heart failure hospitalization. Although amiodarone is not routinely used for rate control, it is useful in patients with contraindications for calcium channel blockers (such as systolic heart failure) and beta-blockers (such as reactive airway disease).

4.3 Pharmacologic rhythm control

A pharmacologic rhythm control strategy consists of either chronic prophylactic therapy with antiarrhythmic drugs or single pill in the pocket approach at each symptomatic recurrence of AF by typically using class IC (flecainide, propafenone) drugs depending on the frequency and severity of symptoms. Amiodarone is by far the most effective agent for rhythm control (maintenance of sinus rhythm) among all the antiarrhythmic agents. Its adverse effects including pulmonary and liver toxicity and effects on thyroids limit its use.

Ventricular arrhythmias are common from all the antiarrhythmic agents and they are poorly tolerated due to their adverse effects. Use of antiarrhythmic therapy for rhythm control should be individualized based on comorbidities such as hypertension, left ventricular hypertrophy, congestive heart failure and ischemic heart disease.

4.4 Catheter ablation of AF

Catheter ablation of AF should be offered to patients but requires appropriate selection of patients. Patients with lone paroxysmal AF and minimal structural heart disease are the ideal candidates for this procedure. It may be appropriate to consider this approach as first line therapy in patients younger than 35 years of age with symptomatic AF and SAN dysfunction that complicates antiarrhythmic drug therapy. Patients with persistent and symptomatic AF and minimal structural heart disease who have failed at least one antiarrhythmic drug therapy may be offered catheter ablation.

Isolation of pulmonary veins alone is often sufficient in patients with paroxysmal AF. However, in patients with persistent AF, a variety of ablation strategies have been used. The success rate for AF ablation is higher for paroxysmal AF (70-80%) than persistent AF (50 %). The risk of major complication after AF ablation is approximately 5-6 % and most common

complications include cardiac tamponade, pulmonary vein stenosis and stroke. Atrioesophageal fistula is a rare but lethal complication of this procedure.

4.5 Other nonpharmacologic approaches to AF management

AV nodal ablation and insertion of pacemaker may be considered in patients with persistent AF and inadequate ventricular rate control with medications or intolerance to medications, and those with failed attempt at catheter ablation of AF.

Maze procedure is a surgical procedure developed by Cox that involves multiple incisions involving the right and left atria but requires open thoracotomy and cardiopulmonary bypass (10). It may be considered concomitantly in patients who are undergoing coronary bypass graft surgery or valvular repair or replacement.

5. Atrial tachycardia

Atrial tachycardias are broadly classified into focal and macroreentrant types based on management. Focal atrial tachycardias originate from activation of one particular focus in the atria where as macroreentrant tachycardias use a relatively large reentrant circuit using conduction barriers for creation of the circuit.

5.1 Atrial flutter

Atrial flutter is the most common type of macroreentrant atrial tachycardia. The reentrant circuit of atrial flutter is constrained anteriorly by the tricuspid annulus and posteriorly by the crista terminalis and eustachian ridge. A typical atrial flutter circulates in a counterclockwise fashion around the tricuspid annulus and atypical flutter circulates in a clockwise fashion

Atrial rate during atrial flutter varies between 200-300 beats/min. Ventricular response to atrial flutter is determined by factors such as conduction through the AVN and concomitant antiarrhythmic drug therapy. On ECG, atrial flutter manifests as recurring, regular, saw tooth flutter waves (Fig 4).



Fig. 4. Typical atrial flutter

5.2 Clinical features

Atrial flutter occurs as a result of valvular heart disease such as mitral and tricuspid stenosis, pulmonary embolism, prior AF ablations, atrial septal defects that result in atrial dilation, thyrotoxicosis, alcoholism and following surgeries for congenital heart disease. Patients in atrial flutter often describe symptoms such as palpitations, diaphoresis, lightheadedness and dizziness.

On physical examination, patients have constant intensity of the first heart sound if the ventricular response is constant and rapid jugular venous pulsations. Carotid sinus massage often slows down the ventricular response but does not terminate it.

5.3 Management

Atrial flutter is much more difficult to rate control than AF. Thus, transthoracic synchronous DC cardioversion may be offered as an initial treatment to patients with atrial flutter. Pharmacologic agents such as procainamide and ibutilide are often successful in conversion of atrial flutter to sinus rhythm, however ibutilide is associated with *Torsades de pointes (Tdp)* arrhythmia. If atrial flutter persists despite cardioversion, class IA and IC agents or low dose amiodarone may be helpful for prevention of recurrences. However, patients should be on AV nodal blocking agent during this therapy to prevent faster ventricular response after slowing of atrial flutter rate that may allow more frequent AV nodal conduction.

The success rate for ablation of cavo-tricuspid isthmus dependent atrial flutter is 90-100%, thus it may be offered as an alternative to drug therapy as an initial therapy in patients with these types of atrial flutter.

As far as acute management of atrial flutter is considered, ventricular rate control can be attempted with intravenous diltiazem and verapamil or beta-blockers such as esmolol and metoprolol. Digoxin may be added if the combination of beta-blockers and calcium channel blockers is inadequate for ventricular rate control. Intravenous amiodarone has been found to be successful in rate control of atrial flutter.

The recommendations for antithrombotic therapy in patients with atrial flutter are the same as AF.

6. Focal atrial tachycardia

Focal atrial tachycardias originate from a single atrial focus and generally exhibit atrial rates between 150-200 beats/min. They typically occur in short bursts, however, may be incessant. On ECG, focal atrial tachycardia is differentiated from sinus tachycardia by P wave morphologies that are characteristically different in contour compared to sinus P wave morphology and from atrial flutter by presence of isoelectric interval in all the ECG leads. Right atrial tachycardias produce positive or biphasic P waves in lead V1 where as left atrial tachycardias produce negative P waves.

Focal atrial tachycardia occurs commonly in patients with structural heart disease, cor pulmonale, digitalis toxicity and electrolyte abnormalities. Stimulants such as caffeine can precipitate focal atrial tachycardia.

On examination, patients have variable intensity of the first heart sound due to variable conduction through the AVN with resultant variable R-R interval, and increased number of *a* waves on the jugular venous pulse. Carotid sinus massage or intravenous adenosine results in increased vagal activity and decrease in ventricular response but rarely terminates it.

Focal atrial tachycardia in patients on digitalis can be due to toxicity. Digitalis should be discontinued in these patients and electrolyte abnormalities such as hypokalemia should be corrected. Administration of digitalis antibodies should be considered in patients with rapid ventricular response after correction of electrolyte abnormalities. In patients who are not taking digitalis, beta-blockers, calcium channel blockers or digitalis may be considered for control of ventricular response. Persistent atrial tachycardia may require administration of class IA, IC or III agents. Patients intolerant of antiarrhythmic therapy or non compliant with antiarrhythmic therapy can be offered catheter ablation for elimination of the tachycardia based on the location of the focus and local expertise in ablation.

7. Multifocal (chaotic) atrial tachycardia

Multifocal atrial tachycardia is characterized by presence of variable P wave morphologies and PR intervals due to origin of this tachycardia from multiple foci (Fig 5). At least 3 different types of P wave contours are necessary for the diagnosis.

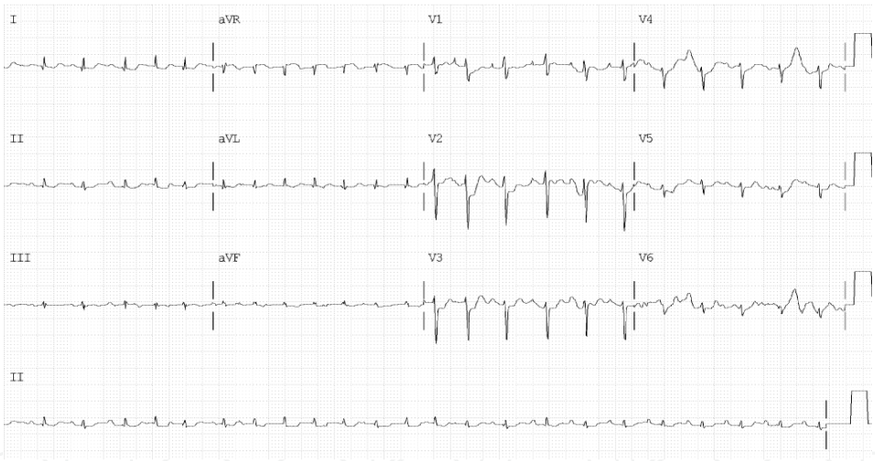


Fig. 5. Multifocal atrial tachycardia

It occurs in elderly patients with chronic obstructive pulmonary disease and congestive heart failure. It may be rarely caused by digitalis or theophylline administration.

Management of this tachycardia should be directed at treatment of underlying disease. Calcium channel blockers such as verapamil and diltiazem or amiodarone are helpful in management. Potassium and magnesium should be replaced. Beta-blockers are avoided in patients with reactive airway disease.

8. AV nodal reentrant tachycardia

AV nodal reentrant tachycardia (AVNRT) is a reentrant form of SVT involving AV junction. On surface ECG, it is characterized by presence of regular R-R intervals, narrow QRS

complexes (in absence of previous conduction defects), sudden onset and termination and ventricular rates between 150-250 beats/min (Fig 6).

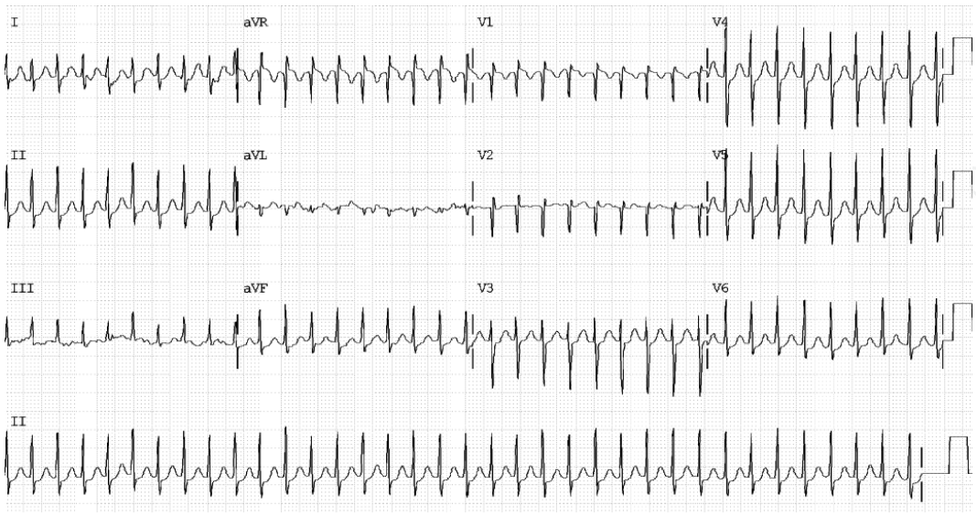


Fig. 6. AV nodal reentrant tachycardia

Conduction of the electrical impulses from the atria to the AVN occurs over slow and fast pathways. A premature atrial complex (PAC) or rarely a premature ventricular complex (PVC) often initiates this tachycardia. PAC conducts to the ventricle over the slow pathway during the refractory period of the fast pathway resulting in prolonged PR interval before the initiation of the tachycardia. After conduction over the slow pathway it returns and conducts over the fast pathway, which is no longer refractory and thus initiating the circus movement. This is the mechanism of a typical AVNRT. Atypical form of AVNRT is characterized by anterograde conduction to the ventricles over the fast pathway and retrograde conduction to the atrial over the slow pathway. Thus atrial activation is slightly before, during or slightly after the activation of QRS complex. If atrial activation occurs after the QRS complex, it manifests on the surface ECG as pseudo-r' in lead V1 and pseudo-S waves in leads II, III and aVF.

Typical form of AVNRT is characterized by short ventriculoatrial conduction (VA) interval due to near simultaneous activation of atria and ventricles where as atypical form of AVNRT is characterized by longer VA interval due to activation of ventricles over the fast pathway and retrograde activation of atria over the slow pathway. Typical AVNRT is a short R-P type of SVT due to faster retrograde conduction to the atria over the fast pathway whereas atypical AVNRT is a long R-P type SVT due to longer retrograde conduction to the atria over the slow pathway.

8.1 Clinical features

AVNRT usually occurs in patients with no structural heart disease. It manifests with symptoms such as palpitations, lightheadedness, anxiety, syncope, diaphoresis, angina and worsening of presence of new heart failure symptoms. It occurs in third or fourth decade of life. It is more prevalent in women than in men.

8.2 Management of acute attack

Patients with infrequent episodes of AVNRT that are well tolerated can be simply reassured. Vagal maneuvers such as carotid sinus massage, gagging, and Valsalva maneuver may sometimes be helpful in termination of the episode. Adenosine administered in doses of 6-12 mg is the initial drug of choice in patients with suspected AVNRT. However, it should be kept in mind that adenosine is contraindicated in patients with asthma. Calcium channel blockers such as diltiazem or verapamil administered intravenously may also be helpful in termination of the acute attack. Digitalis is rarely used in acute attack due to its slower onset of action. However, it may increase the success rate of vagal maneuvers. Beta-blockers are rarely used due to their inferior efficacy as compared to calcium channel blockers in termination of acute episode. DC synchronized cardioversion may be required in patients with hemodynamic instability due to AVNRT.

8.3 Prevention of recurrences

Long term medical therapy or radiofrequency ablation for prevention of recurrences is recommended in patients with frequent and severe episodes of AVNRT. Long acting calcium channel blockers, long acting beta-blockers or digitalis may be helpful in prevention of recurrence.

Radiofrequency ablation can be offered as an initial therapy in patients who are symptomatic from frequent and severe episodes. It should also be offered in patients who are intolerant or reluctant to pharmacologic therapy for prevention of recurrences. It has a very high success rate (95 %) and very low complication rate.

9. AV reciprocating (reentrant) tachycardia

AV reciprocating tachycardia (AVRT) occurs due to reentry over accessory pathways that connect the atrium or AVN to the ventricle outside the normal AVN and His- Purkinje system. These pathways can conduct impulses either anterogradely from the atria to the ventricles or retrogradely from the ventricles to the atria. When the conduction occurs anterogradely over the accessory pathway (manifest conduction), it results in pre-excited QRS complex where as if the accessory pathway conducts only retrogradely (concealed conduction), it does not produce ventricular preexcitation. Preexcited QRS complex with tachycardia is referred to as Wolf Parkinson White (WPW) syndrome.

9.1 Reentry over a concealed pathway (Orthodromic AVRT)

Orthodromic AVRT results from anterograde conduction from the atria to the ventricles over the normal AV nodal conduction system and retrograde conduction from the ventricles to the atria over the accessory pathway. Thus there is no ventricular preexcitation during the tachycardia and the accessory pathway is considered to be concealed.

On ECG, this results in retrograde P waves that occur in the ST segment or T wave portion of the ECG. The contour of P waves is different from sinus P waves due to eccentric activation of the atria in most cases.

Diagnosis is usually done during an electrophysiology study (EPS), where a PVC activates the atria prior to activation of the His bundle. Also if the PVC activates the atria when His bundle is supposed to be refractory, presence of accessory pathway is almost certain. The VA interval remains constant over a wide range of coupling intervals of the PVC as most of the accessory pathways exhibit all or none conduction unlike conduction over the AVN which shows decremental property at higher rates. The VA interval is < 50% of R-R interval.

9.2 Clinical features

Tachycardia rates in these patients tend to be faster than AVNRT and are around 200 beats/min. Patients can present with palpitations, anxiety, presyncope or syncope. On physical examination, they have regular pulse and constant intensity first heart sound due to constant R-R interval.

9.3 Management

As the tachycardia circuit involves the AVN and the fact that conduction over the accessory pathway occurs only retrogradely, the acute management of orthodromic AVRT is similar to AVNRT. Vagal maneuvers, adenosine, calcium channel blockers or digitalis that produce transient AV block may result in termination of this tachycardia. Chronic prophylactic therapy involves administration of antiarrhythmic drugs that prolong the conduction over the accessory pathway such as class I and class III drugs or radiofrequency ablation. Radiofrequency ablation of the accessory pathway has high success rates, low complication rate and should be considered for symptomatic patients early in their management or those with intolerance to antiarrhythmic therapy.

10. Preexcitation syndrome

Preexcitation syndrome results from activation of the ventricle in part or entirely from atrial impulses that are conducted over the accessory pathway that conducts anterogradely. Three features of WPW syndrome are –

1. PR interval less than 120 ms
2. Presence of slow up or down sloping QRS complex in some leads, often called as *delta* waves
3. Secondary ST-T wave changes that are generally directed in direction opposite to the major delta or QRS vector.

Accessory pathways can be *atriohisian* that connect the atria to the His bundle, *atriofascicular* that connect atria to one of the fascicles, *nodofascicular* that connect the AVN to the fascicles, or *fasciculoventricular* that connect the fascicles to the ventricle. *Mahaim fibers* are atriofascicular or nodofascicular fibers that exhibit progressive increase in the AV interval in response to atrial overdrive pacing unlike the usual atrioventricular accessory pathway.

Location of the accessory pathways can be predicted by careful analysis of the ECG. Accessory pathways that conduct to the right ventricle produce negative delta waves and QRS in lead V1 whereas those conducting to the left ventricle produce positive delta wave and QRS in lead V1. Posteroseptal pathways produce negative delta waves or QRS complex in leads II, III and aVF. Left free wall pathways produce negative or isoelectric delta wave in lateral leads.

Right free wall pathways produce left axis deviation where as if the accessory pathway is located in the right ventricle, presence of inferior axis indicates anteroseptal pathway.

Accessory pathways have a longer refractory period during long cycle lengths. Thus a PAC can result in conduction over the normal AVN and His bundle complex as the accessory pathway is refractory but when the impulse arrives to the ventricles, it has recovered excitability and can conduct retrogradely resulting in reciprocating orthodromic AVRT. If the conduction occurs anterogradely over the accessory pathways and retrogradely over the AVN-His bundle, it is referred to as antidromic AVRT.

10.1 Permanent form of AV junctional reciprocating tachycardia (PJRT)

Rfrom very slowly conducting posteroseptal accessory pathway. It is maintained by anterograde conduction over the AVN and retrograde conduction over the accessory pathway.

10.2 Clinical features

Incidence of preexcitation syndrome is approximately 1.5/1000 among healthy adults. Left free wall accessory pathway is the most common type of accessory pathway. Patients with Ebstein anomaly often have right-sided accessory pathways. The prevalence is higher in men and decreases with age. The frequency of tachycardia however increases with age with reciprocating tachycardia being the most common (80 %) of patients followed by AF (15-30 %). The incidence of sudden death is very rare (<0.1 %). Patients who have ventricular fibrillation (VF) have ventricular cycle lengths in the range of 240 ms or less. Presence of only intermittent preexcitation during sinus rhythm, abrupt loss of conduction during administration of procainamide are suggestive of longer refractory period of accessory pathway.

10.3 Management of preexcitation syndrome and tachycardia

Patients who have preexcitation on ECG with no history of palpitations or tachycardia may be managed conservatively without any further electrophysiologic evaluation. However, patients with preexcitation on the ECG and history of palpitations or documented tachycardia need further treatment. Therapeutic options include pharmacologic management and radiofrequency ablation.

While considering pharmacologic management of preexcited tachycardia and patients with accessory pathway (concealed or manifest), it is important to understand the effects of various drugs on these pathways. Class IA drugs increase the refractoriness of accessory pathways. Calcium channel blockers, adenosine, and digitalis act on AVN where as class IC agents (flecainide, propafenone) and class III agents such as amiodarone and sotalol increase the refractoriness of both AVN and accessory pathway.

10.4 Management of acute episode

Management of orthodromic AVRT (using the concealed accessory pathway) has been discussed above.

Patients with preexcited tachycardias suspected by presence of anomalous QRS complexes, or know prior history of preexcitation from previous ECGs, require drugs that prolong the

refractoriness of the accessory pathway (such as procainamide) often coupled with drugs that prolong the refractoriness of AVN to break the reentry circuit. However, patients that are hemodynamically unstable require electrical DC cardioversion.

10.5 Prevention of recurrence

Radiofrequency ablation of the accessory pathway has become the treatment of choice in patients with accessory pathway for prevention of recurrence due to its high success rate and low complication rate. However, if transvenous catheter ablation is unsuccessful epicardial ablation or surgical interruption of the accessory pathway may be necessary.

Drug therapy is an alternative approach to ablation however the effect of drugs on accessory pathways is unpredictable. Class IC agents, amiodarone and sotalol or combination of class IC agent and beta blocker may be effective as they prolong refractoriness in the accessory pathway as well as AVN. Further testing (such as exercise, isoproterenol infusion) is essential to be certain that ventricular response is controlled in patients with AF that are started on these agents.

11. Ventricular tachycardia

Ventricular tachycardia (VT) is defined as 3 or more consecutive premature ventricular complexes. It is considered to be nonsustained if it lasts less than 30 seconds and sustained if it last more than 30 seconds or requires termination due to hemodynamic collapse.

VT originates distal to His bundle in the ventricular muscle, specialized conduction system or combination of both tissues. It can occur in patients with no structural heart disease, as a part of inherited syndromes or in patients with structural heart disease.

On ECG, it is characterized by wide QRS complexes that are mostly regular, with rates varying between 70-250 beats/min and ST-T vector directed opposite to the QRS vector (Fig 7).



Fig. 7. Ventricular tachycardia

11.1 Wide QRS complex tachycardia and differentiation of VT from SVT with aberrant conduction

Differential diagnosis of wide QRS complex tachycardia includes VT, SVT with aberrant conduction due to rate dependent bundle branch block, SVT with preexisting bundle branch block, SVT /AF with anterograde conduction over the accessory pathway, or antidromic AVRT. VT is however the most common cause of tachycardia with wide QRS complex.

The features that favor the diagnosis of VT include –

1. Presence of underlying structural heart disease, or myocardial infarction
2. Duration of QRS complex greater than 140 ms
3. Presence of fusion beats (QRS complexes originating from co-activation of ventricles by ventricular and supraventricular rhythm on ECG)
4. Presence of capture beats (ventricular activation from supraventricular beats with contour different from rest of the complexes)
5. Presence of conduction disturbances on the baseline ECG and QRS complexes different in configuration compared to baseline ECG
6. AV dissociation i.e. no relation between ventricular and supraventricular rhythm; it is highly specific but not very sensitive
7. Positive or negative concordance in precordial leads (all QRS complexes in precordial leads are either negative or positive)
8. In the presence of right bundle branch block pattern, the initial pattern of activation is different from activation by sinus initiated QRS complexes, larger amplitude of R wave compared to R' and rS or QS pattern in lead V6
9. In the presence of left bundle branch block, R wave in V1 is longer than 30 ms and duration of start of R wave to nadir of S wave in V1 is longer than 60 ms and qR or qS pattern in lead V6
10. Extreme left axis deviation ("northwest axis") on the ECG

Electrophysiologic characteristics of VT include negative HV interval and dissociated His bundle deflections from ventricular activation.

11.2 Clinical features

VT can occur as nonsustained or sustained episode. Nonsustained VT is usually well tolerated, however patients may complain of palpitations and presyncopal symptoms. Sustained VT can be hemodynamically stable or it may present has unstable runs finally degenerating into ventricular fibrillation.

Ischemic heart disease is by far the most common cause of VT followed by cardiomyopathy. Patients with sustained monomorphic VT have myocardial substrate different from patients with ventricular fibrillation and often have reduced left ventricular ejection fraction, slowed intraventricular conduction and previous myocardial infarction.

Reduced left ventricular function, inducibility of sustained VT during electrophysiologic study, T wave alternans are some of the strong predictors of poor outcome in patients with VT (11).

Prognosis of patients with idiopathic VT, in absence of structural heart disease is good.

11.3 Acute management of sustained ventricular tachycardia

Sustained VT in hemodynamically stable patients can be terminated pharmacologically with administration of one of the antiarrhythmics such as amiodarone, lidocaine, procainamide or sotalol. Amiodarone administered by intravenous infusion is often effective. If the arrhythmia is non responsive to medical therapy or if patient is hemodynamically unstable, electrical DC cardioversion can be used. Ventricular pacing using transvenous catheter inserted into the right ventricle at rates faster than VT can terminate VT however runs the risks of degenerating VT into ventricular flutter or fibrillation.

After stabilization of the patient, one should look for reversible conditions such as ischemia, electrolyte abnormalities, and drugs as a cause of VT.

11.4 Therapy for prevention of recurrences

Symptomatic nonsustained VT in patients with normal left ventricular function can be treated with beta-blockers. Patients refractory to beta-blockers can be treated with class IC agents, sotalol or amiodarone.

Asymptomatic nonsustained VT in a patient with no evidence of structural heart disease does not require treatment.

11.5 Primary prevention

Patients with nonsustained VT and structural heart disease with a left ventricular ejection fraction of <0.35 to 0.40 should undergo EPS and implantable cardioverter defibrillator (ICD) implantation if they have inducible VT (12,13).

Patients with nonsustained VT, prior myocardial infarction and left ventricular ejection fraction of <0.30 do not require EPS and can directly undergo ICD placement (14)

Patients with ischemic and non-ischemic cardiomyopathy, left ventricular ejection fraction of <0.35 and NYHA class II or III heart failure should undergo ICD implantation due to 7 % absolute decrease in mortality (15). Amiodarone is the next best therapy in patients with heart failure, non-ischemic cardiomyopathy ejection fraction of <0.40, and frequent PVCs (16).

11.6 Secondary prevention

Survivors of sudden cardiac arrest or patients with sustained VT resulting in hemodynamic compromise and poor left ventricular function should be offered ICD implantation (17, 18). Patients who refuse ICD implantation, treatment with amiodarone is the next best therapy (19).

Optimal therapy for patients with sustained VT and normal left ventricular function is unknown however empiric amiodarone appears to be safe.

Radiofrequency ablation is effective in patients with idiopathic VT; however it is less effective in patients with structural heart disease and depressed LV function. It may be effective as an adjunctive therapy in patients with recurrent ICD shocks due to VT that is not responsive to combination of antiarrhythmic therapy.

12. Ventricular arrhythmia in arrhythmogenic right ventricular cardiomyopathy

Arrhythmogentic right ventricular cardiomyopathy (ARVC) is familial and progressive cardiomyopathy of the right ventricle. VT in ARVC is caused by reentry. It has left bundle branch contour due and often resembles right ventricular outflow tract tachycardia. Patients may be asymptomatic or may manifest signs and symptoms of right heart failure. ECG during sinus rhythm shows right bundle branch block pattern and T wave inversions in leads V1 through V3. Epsilon wave (which is a terminal notch in the QRS) can be present in these leads. ICDs are preferable for the treatment of this condition due to progressive nature of the disease.

13. Inherited arrhythmia syndromes

13.1 Catecholaminergic polymorphic ventricular tachycardia

Catecholaminergic polymorphic ventricular tachycardia (CPVT) is an inherited VT that occurs in children and adolescents without any structural heart disease or surface electrocardiographic abnormalities. It is stress induced and patients present with syncope or aborted sudden death. VT is bidirectional in nature and may eventually degenerate into polymorphic VT with exercise. The treatment of choice is beta-blockers and ICD. Patients should be instructed to avoid vigorous exercise.

13.2 Brugada syndrome

It is an inherited form of idiopathic ventricular fibrillation that occurs commonly in young healthy Southeast Asians. These patients have no structural heart disease. On ECG, they have ST segment elevation in the precordial leads at rest. Mutations in the sodium channel genes (SCN5A) and calcium channel have been identified in several families. Phase 2 reentry due to heterogenous loss of action potential dome is thought to be responsible for ventricular arrhythmias. There is no pharmacologic treatment available and ICD implantation is the only treatment that is effective for prevention of sudden death.

13.3 Torsades de pointes

Tdp refers to a VT characterized by presence of QRS complexes with varying amplitude that twist around the isoelectric baseline (Fig 8). The rate usually varies from 200-250 beats/min. It occurs in patients with prolonged ventricular repolarization manifesting as prolonged QT interval.

VT that is similar to *Tdp* but occurs in patients without QT prolongation is called polymorphic ventricular tachycardia.

Tdp can occur in two settings. Late PVCs may discharge during termination of long T wave that precipitates the episode of VT or short coupled variant occurring due to close-coupled PVC. *Tdp* is thought to occur from early afterdepolarizations.

The common predisposing factors for *Tdp* include severe bradycardia, hypokalemia, use of class IA or III antiarrhythmic drugs, QT interval prolonging drugs or congenital long QT syndrome.

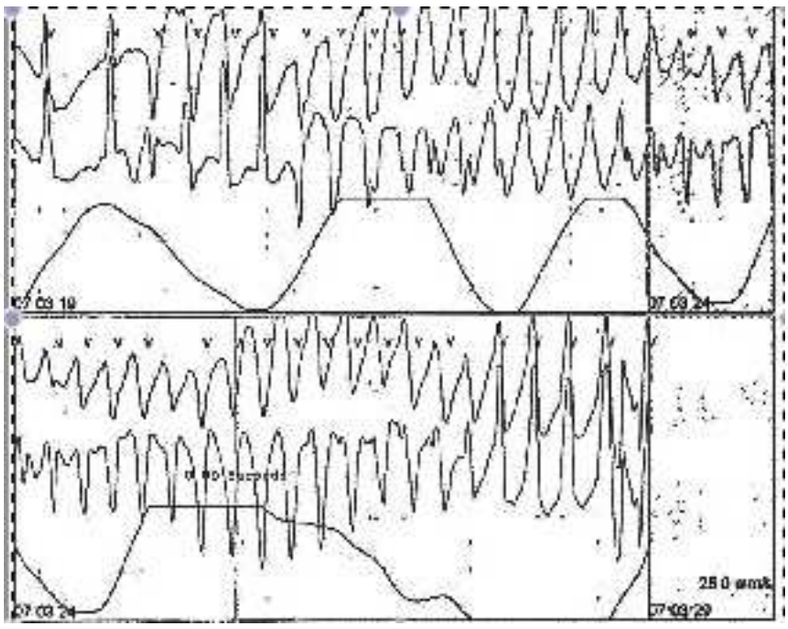


Fig. 8. Torsades de pointes

In patients with *Tdp*, the reversible cause should be corrected such as discontinuing Class IA or III antiarrhythmic, or QT prolonging drug. In patients with acquired long QT syndrome, intravenous magnesium should be given. This is followed by temporary atrial or ventricular pacing. Isoproterenol infusion can be tried prior to starting pacing.

Polymorphic VT however can be treated with standard antiarrhythmic drugs.

13.4 Long Q-T syndrome

Corrected long QT interval (QTc) is less than 460 ms for men and 470 ms for women. Patients with long QT syndrome have prolonged QT interval and the risk for life threatening arrhythmias increases with length of QTc.

Long QT syndrome can be congenital or acquired. Congenital long QT syndrome is usually due to inherited channelopathies. Acquired form of long QT syndrome as described above is caused by electrolyte abnormalities such as hypokalemia, drugs such as class IA and III antiarrhythmics, tricyclic antidepressants, phenothiazines, central nervous system lesions, and severe bradyarrhythmias.

Patients with long QT syndrome may present with syncope, however sudden death may the initial manifestation in pediatric patients. High risk factors for sudden death in patients with long QT syndrome include family history of sudden death and prior history of syncopal episodes.

In patients with idiopathic long QT syndrome, stress testing, electrocardiographic monitoring during various stimuli such as auditory stimuli, psychological stress, sudden exposure to cold valsalva maneuver can all be helpful to determine the risk of life threatening arrhythmias. ECG should be obtained for all family members in the presence of symptoms in the proband.

Tdp often develops during bradycardia in patients with acquired forms of long QT syndrome.

Idiopathic long QT syndrome patients, who are asymptomatic, have no family history of sudden cardiac death, and QTc shorter than 500ms need no therapy, however beta-blockers are generally recommended.

Patients with above risk factors but no syncopal episodes or aborted sudden death should be treated with beta-blockers.

Patients with syncope and aborted sudden death require ICD implantation. These patients should also be treated with beta-blockers.

Patients who continue to have symptoms despite maximal therapy can be treated with left sided cervicothoracic sympathetic ganglionectomy.

For treatment of *Tdp* from acquired long QT syndrome, refer to the discussion above.

13.5 Short QT syndrome

QT interval less than 350 ms at heart rates less then 100 beats/min is generally accepted as short QT interval. Short QT interval syndrome is an inherited disorder due to gain of function mutations in the genes that are responsible for long QT syndrome. It is associated with increased risk of ventricular fibrillation and sudden death. Patients are also predisposed to development of AF. In patients with short QT interval, high-risk features include history of syncope, family history of sudden death, palpitations, or AF. Reversible causes of short QT interval such as hyeprcalcemia, hyperthermia, and digitalis need to be excluded before making the diagnosis. ICDs are the treatment of choice in symptomatic patients with short QT syndrome. Antiarrhythmic agents such as quinidine have been found to be effective (20).

14. Idiopathic ventricular tachycardia and ventricular fibrillation

Idiopathic VT is a monomorphic VT occurring in patients with no structural heart disease. There are three distinct forms – outflow tract VT, annular VT, and fascicular VT. Prognosis for these patients is good and they are often amenable to ablation.

Idiopathic ventricular fibrillation occurs in less than 10 % cases of out of hospital cardiac arrest. Association with early repolarization has been suggested in some studies (21). Recurrences can occur and ICD implantation is the treatment of choice. It can occur due to short-coupled PVCs in which case ablation of PVC might be helpful to prevent recurrences.

14.1 Bidirectional ventricular tachycardia

It is characterized by QRS complexes with right bundle branch block pattern with alternating polarity and regular rhythm. It occurs in patients with digitalis toxicity and CPVT. In patients with digitalis excess, digoxin-binding antibodies should be given in addition to antiarrhythmic therapy such as lidocaine or phenytoin.

15. Bundle branch reentrant ventricular tachycardia

It is reentrant tachycardia due to a circuit established over the bundle branches or fascicles. It results in monomorphic sustained VT and is usually seen in patients with structural heart

disease. Therapy is similar to other types of VT. Pace termination is effective in acute setting and radiofrequency ablation is effective in elimination of VT.

15.1 Ventricular flutter and ventricular fibrillation

Ventricular flutter is characterized by regular large oscillations occurring at a rate of 150 -300 beats/min where as ventricular fibrillation is characterized by irregular undulations of varying amplitude and contour (Fig 9).

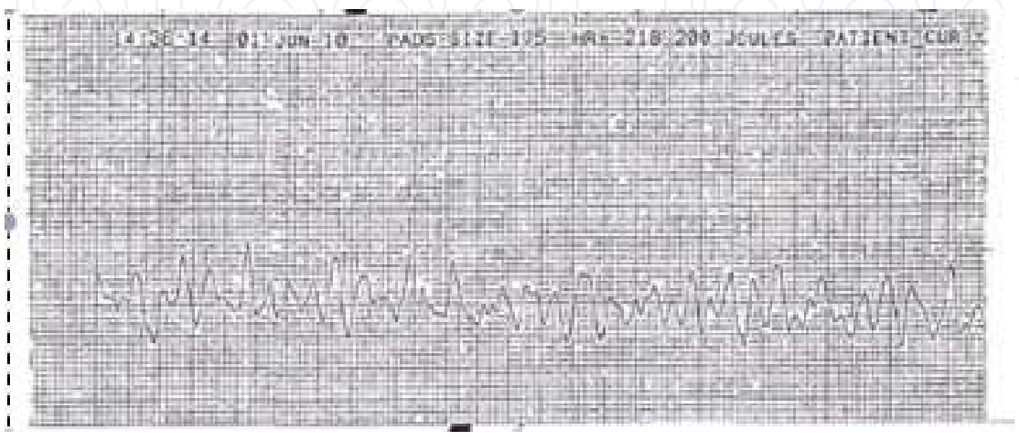


Fig. 9. Ventricular fibrillation

15.2 Clinical features

VT often precedes ventricular fibrillation; however it is not a rule. It is most commonly caused by coronary artery disease. Other causes include hypoxia, and antiarrhythmic drug therapy. It may occur during pace termination of VT that may degenerate into ventricular fibrillation.

Patients with ventricular fibrillation present with fainting episode, syncope, loss of consciousness, apnea and if the corrective measures are not taken, the event is fatal within 3-5 minutes.

15.3 Prognosis

Poor prognostic factors in resuscitated patients from ventricular fibrillation include decreased left ventricular ejection fraction, regional wall motion abnormalities, congestive heart failure, presence myocardial infarction in absence of an acute event and patients with anterior myocardial infarction complicated by ventricular fibrillation. Overall, prognosis of resuscitated patients from ventricular fibrillation patients is better than those presenting with asystole.

15.4 Management

There are three components to management of patients with sudden cardiac arrest from ventricular fibrillation – immediate resuscitation, search for etiology of the event and prevention of recurrence.

First of all, immediate resuscitative measures should be undertaken in patients with sudden cardiac arrest as per the advanced life support guidelines. The key to better outcome and survival in patients with ventricular fibrillation is early defibrillation. Nonsynchronized direct current shock (defibrillation) using 200 to 400 Joules is usually adequate. The shock energy required is low if defibrillation is done early.

After patients are stabilized, further evaluation should be directed towards the etiology of the event and correcting it if possible (such as correction of ischemia, or electrolyte abnormalities).

Pharmacologic approaches to prevent recurrences include intravenous administration of antiarrhythmic agents such as amiodarone, lidocaine or procainamide. Amiodarone tends to be the most effective of all. Pharmacologic approaches for prevention of recurrences should be used until the etiology of the event is identified. Patients with continued risk of VT or VF should be offered ICD placement if the etiology of the event is irreversible.

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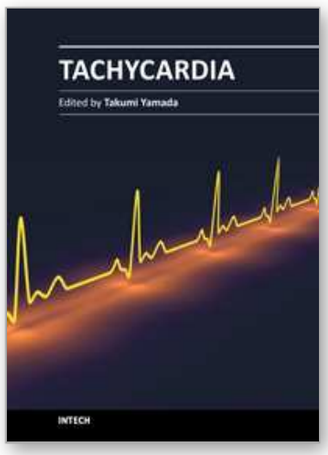
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Tachycardia
Edited by Prof. Takumi Yamada

ISBN 978-953-51-0413-1
Hard cover, 202 pages
Publisher InTech
Published online 30, March, 2012
Published in print edition March, 2012

Heart rates are normally controlled by a natural pacemaker, the sinus node, and normal heart rhythm is called sinus rhythm. Tachycardia is defined as a faster heart rhythm than normal sinus rhythm. Tachycardias can cause symptoms such as palpitations, chest pain, shortness of breath and fatigue, which reduce the quality of life. Fast tachycardias can cause hemodynamic collapse and sudden cardiac death. The causes, mechanisms, and origins of tachycardias are various. The diagnosis of tachycardias is made by electrocardiograms and electrophysiological testing. Tachycardias can be managed and treated by pharmacological and non-pharmacological approaches. This book covers these concerns from basic and clinical points of view and will lead to a further understanding and improvement in the clinical outcomes of tachycardias.

How to reference
In order to correctly reference this scholarly work, feel free to copy and paste the following:

Anand Deshmukh (2012). Definition, Diagnosis and Treatment of Tachycardia, Tachycardia, Prof. Takumi Yamada (Ed.), ISBN: 978-953-51-0413-1, InTech, Available from: http://www.intechopen.com/books/tachycardia/definition-diagnosis-and-management-of-tachycardia

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