Chapter

Recognition and Management of Supraventricular Arrhythmias and Atrial Fibrillation in the Acute Setting

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Abstract

Supraventricular tachycardia (SVT) is a type of tachyarrhythmia with a narrow QRS complex and regular rhythm. These patients are often symptomatic and present to the emergency department (ED) due to acute attacks called paroxysmal SVT. Attacks of SVT start suddenly with the reentry mechanism in most patients. Anginal chest pain and dyspnea occur in patients due to tachycardia. Vagal manoeuvers and adenosine is the treatments of choice for termination of SVT. In multifocal atrial tachycardia (MAT), at least three different P wave morphologies are observed in the ECG, along with variable PP, PR and RR intervals. Treatment is to correct the underlying disease. Patients with atrial flutter (AFl) tend to come to the ED with unstable findings. Atrial fibrillation (AF) is the term used to define the inactive 'worm bag-like' oscillations of the atria, with an absence of true atrium contraction. Ruling out atrial or ventricular thrombi with echocardiography is important to avoid embolization. Priority should be given to hemodynamic stability and the determination of factors triggering the underlying disease. IV beta-blocker and diltiazem can be used for rate control in AF with rapid ventricular response.

Keywords: arrhythmia, supraventricular tachycardia, atrial fibrillation, atrial flutter, narrow QRS complex, palpitations, vagal manoeuvers, adenosine

1. Introduction

Supraventricular tachycardias (SVT) are a type of arrhythmia defined with a narrow QRS complex (<120 msec) and regular tachycardia [>100 beats per minute (bpm)]. Erroneous impulse formation and abnormalities in the conduction pathways (known as reentry mechanism) are blamed for their pathophysiology. Patients may be admitted with a wide range of symptoms, from minor palpitations to severe symptoms such as hypotension and altered mental status. Complaints can last from a few minutes to hours. They often present to the emergency department (ED) due to acute episodes known as paroxysmal SVT (PSVT).

The incidence of SVT is approximately 1–3 cases per 1000 persons and its prevalence increases with age [1]. Atrioventricular (AV) Nodal Reentrant Tachycardia (AVNRT) is most common in middle age and older, while SVT with accessory conduction is more common in younger patients. PSVT can also be triggered by coronary artery disease, myocardial infarction (MI), mitral valve regurgitation (MVR), rheumatic heart disease, pericarditis, chronic obstructive pulmonary disease (COPD) and intoxications via alcohol/caffeine/energy drink.

2. Diagnostic characteristics

Ectopic SVT usually originates in the atrium and the atrial velocity is 100–250 bpm (mostly recorded to be between 140 and 200 bpm). Regular P waves may be misdiagnosed as atrial flutter or 2:1 AV block, sinus rhythm. Sixty-percent of the patients have reentry with AV node, and 20% have reentry via bypass pathways. In SVT, QRS complex is narrow (<0.12 seconds), or wide in conjunction with a previously known branch/fascicular block or rate-dependent aberrant conduction.

Reentrant SVT usually starts when the AV node encounters an ectopic atrial impulse while the AV node is in the partial refractory period. From here, the impulse proceeds with two different functional parallel arms (**Figure 1**). The node is below the ventricular ending and above the atrium. In the case of AV nodal reentry, QRS complexes usually hide the P waves and are not visible. These have a 1:1 message and QRS complexes are normal (**Table 1**).

Ectopic SVT can be seen in patients with acute MI, chronic lung disease, pneumonia, alcohol or digoxin intoxication (often associated with AV block). Atrial tachycardia with block is also frequently (75%) associated with digoxin toxicity. Reentrant SVT is mostly associated with normal heart or rheumatic heart disease, acute pericarditis, MI, and MVR.

2.1 Clinical course and findings

Tachycardia is usually the only finding in those with normal hemodynamic reserve. A previously 'normal' heart can tolerate a 'typical' SVT rate of 160–200 bpm in a given timeframe. However, cardiac output is generally decreased and may lead to signs of heart failure (HF) in the intact myocardium in those with high heart rates. Coronary artery disease, chest pain and dyspnea occur in patients due to tachycardia. Heart failure and pulmonary edema may occur with decreased left ventricular function. In the presence of right heart failure, tachypnea, hypotension, third heart sound (S3), jugular venous distension (JVD), peripheral edema and hepatomegaly may be observed.

Although serious signs such as loss of consciousness and syncope increase the risk, SVT is generally not a severe or life-threatening condition. The most common complaints encountered in symptomatic patients presenting with PSVT are as follows: palpitation (96%), dizziness/lightheadedness (75%), dyspnea (47%) and chest pain (35%). Patients with a heart rate above 170 bpm may experience dizziness and syncope.

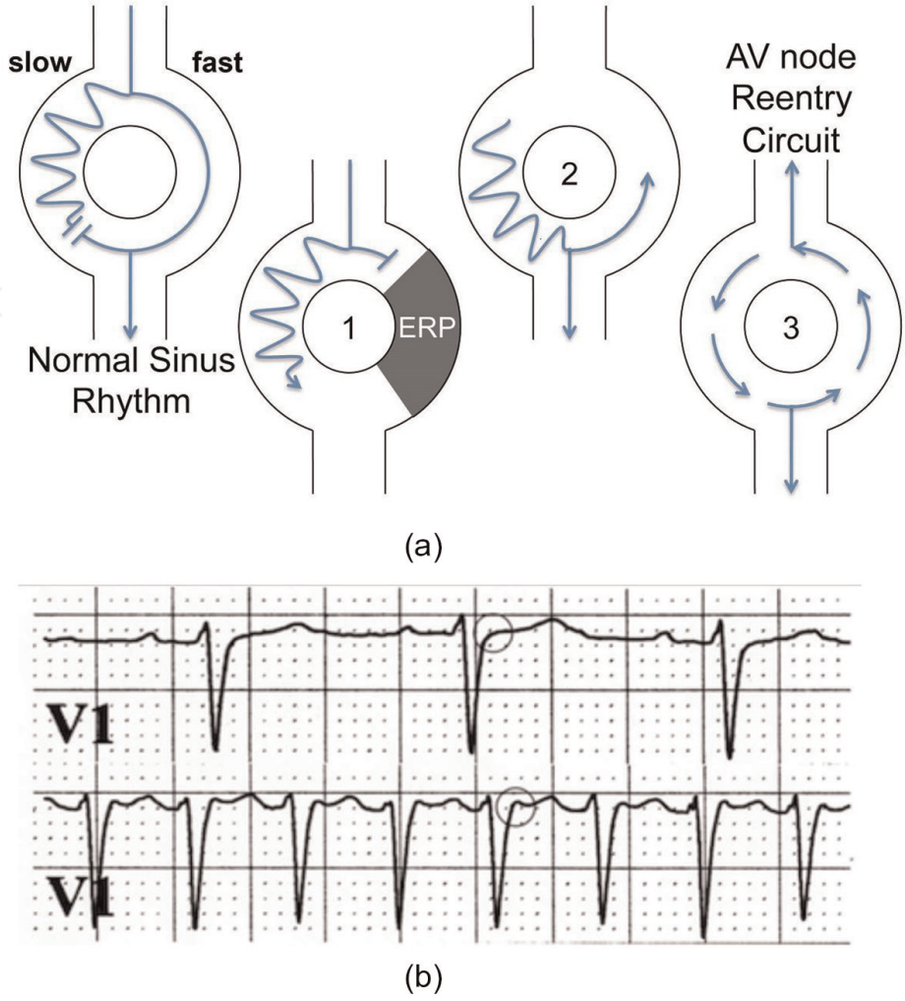

Figure 1.
(a) Re-entry mechanism in AVNRT. Among the subtypes, the most common type is slow-fast re-entrant type AVNRT. (b) ECG findings of this type of SVT:P waves are often hidden in QRS complexes, a false R′ wave (pseudo-R) may be seen in V1 and/or V2 and false S waves (pseudo-S) may be seen in II, III, aVF.

2.2 Evaluation and history

Since the hypotensive event and episode of hypoxia may have triggered the arrhythmia, the history should be extended in terms of diseases such as gastrointestinal bleeding, ruptured ectopic pregnancy, carbon monoxide poisoning and pneumonia, especially in cases presenting with the first attack. Drugs such as nitrate or diuretic, phosphodiesterase inhibitors (PDEI)-5 (sildenafil), alpha-blocker that may cause new onset hypotension should also be questioned in history.

2.3 Laboratory

Further examination of the patients who are stable at admission and have been converted to NSR immediately is unnecessary in the emergency setting. Cardiac enzymes should be evaluated in patients with risk factors for MI, those presenting with chest pain, unstable patients and patients with HF, hypotension, and pulmonary edema. A complete blood count (CBC) is useful in showing anaemia

Tachycardias	A/AV	ECG findings
Sinus tachycardia	A	Heart rate > 100/min Sinus rhythm (P waves) Regular PR intervals
Inappropriate sinus tachycardia (IST)	A	Similar to sinus tachycardia Sinus rhythm (P waves)
Sinus nodal reentrant tachycardia	A	Sudden start and end Sinus rhythm (P waves)
Atrial tachycardia	A	Heart rate 120–250/min P waves with different configurations Prolonged PR interval
Multifocal atrial tachycardia	A	Heart rate > 100–200/min Three different P wave morphologies
Atrial flutter	A	Atrial rate 200–300/min Sawtooth flutter waves AV conduction ratio 2:1 or 4:1
Atrial fibrillation	A	Irregular rhythm P waves are not visible
Atrioventricular nodal reentrant tachycardia	AV	Heart rate 150–200/min P wave inside or immediately after the QRS complex Short PR interval in typical AVNRT Long PR interval in atypical AVNRT
Atrioventricular reentrant tachycardia	AV	Heart rate 150–250/min Narrow QRS complexes in orthodromic conduction Wide QRS complexes in antidromic conduction P waves after the QRS complex

Table 1.
The differential diagnosis of narrow QRS tachycardia with ECG findings in patients presenting with PSVT. (A) Denotes atrial tachyarrhythmias and (AV) atrioventricular tachyarrhythmias.

that can cause ischemia or tachycardia. Thyroid function tests can be used to rule out hyperthyroidism. Serum digoxin levels should be checked in patients using the agent.

2.4 Subtypes of SVT

If the reentry circuit is within the atrial myocardium; atrial fibrillation, atrial flutter, and some types of atrial tachycardia may occur. In this situation, AV node suppressing agents slow down the tachyarrhythmia but do not convert it.

In some re-entry tachycardias, the reentry circuit is in the AV node. These arrhythmias, which are characterized by a sudden onset and end, are recognized with a resting heart rate of over 150 bpm.

- AV nodal reentry tachycardia (AVNRT) occurs when both arms of the reentry circuit are in the AV node; the typical finding is that P waves cannot be discerned in this form (**Figure 1B**).

- One arm of the reentry circuit is in the accessory pathway and the other is in the AV node, called 'AV reentry tachycardia' (AVRT).

- AVNRT and AVRT are both PSVT.

- If at least one branch of the circuit is in the AV node, AV node suppressing agents will have a chance to terminate the arrhythmia.

- Another group called automatic tachycardias are generated due to an excited automatic stimulus focus, including ectopic atrial tachycardia, MAT and junctional tachycardias. Termination of these will be more gradual and slower. These do not respond to electrical cardioversion (ECV), furthermore, these are arrhythmias for which ECV is contraindicated. These are treated with rate control with agents that slow down AV conduction.

2.5 Imaging

Triggering diseases and infections such as edema and pneumonia can be displayed on chest radiography. Point-of-care ultrasound (POCUS)/echocardiography may be useful in suspected structural heart disease.

3. Treatment

First choice should be vagal manoeuvers (VM) followed by adenosine for termination of stable PSVT. The most common practical applications for this purpose in the context of VM include carotid sinus massage (CSM) and Valsalva. Apart from these, there are also techniques such as cold-water immersion and massage on the eyeball, which used to be more common in the past (**Figure 2**).

The VM slows down the AV node and lengthens the refractory period within the node. It also has a negative inotropic effect on the ventricular myocardium. VM is also indicated for the examination of cardiac murmurs.

3.1 Valsalva manoeuver and simplified modified Valsalva (SMV)

Valsalva manoeuver is the most commonly used VM because it is easy and practical. In most guidelines, it is primarily recommended for regular tachycardia treatment with narrow QRS. It is more successful in AVRT than AVNRT. Valsalva should not be administered to patients whose hemodynamic stability is not attained, those with known aortic stenosis, recent MI, and those who have diseases in which an increase in venous pressures may be dangerous.

Different percentages (19–54%) are reported for clinical success rate. Although a Cochrane review recently announced that there is insufficient evidence to support or refute its effectiveness, it is widely used all over the world [2]. In a recent multicentric controlled study in China, SMV was found to be 43% successful and standard Valsalva 17% successful in terminating PSVT.

For standard Valsalva, the patient with stable narrow QRS and regular tachycardia sits in a comfortable position. After inspiration with normal tidal volume, it is asked to try to move the piston for 15 seconds by blowing into the injector with a 10- or 20-ml needle removed (trying to reach a pressure of 40 mmHg).

SMV is commenced in a semi-sitting position, after blowing for 15 seconds, the patient assumes a supine position and the legs are passively lifted concurrently. After

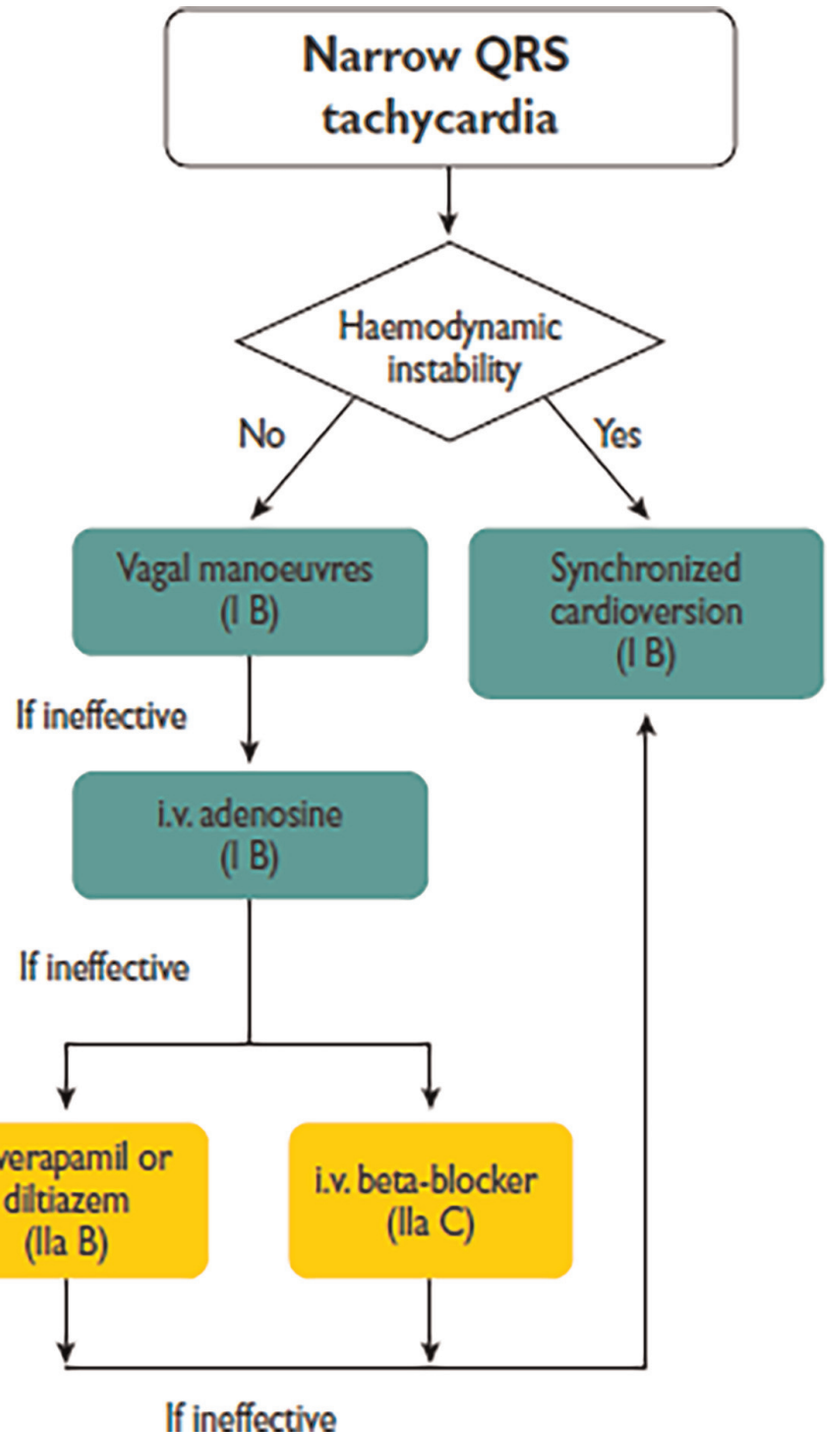

Figure 2.
Algorithm for emergency treatment of narrow-QRS tachycardia in the absence of previously established diagnosis.

waiting in this way for 15–30 seconds, the resting period starts, and the process is completed in around 1 minute.

CSM is applied under monitoring in the supine position, unilaterally with the neck facing the opposite side and in extension. The procedure can be performed with or without Valsalva manoeuver. At the carotid bulb where the carotid pulsation is taken, rubbing is performed with gentle pressure, without occluding the arterial flow. The process is completed in 5 seconds in a posteromedial direction, aiming toward the vertebral column. Double-sided procedure is absolutely contraindicated. It is not

attempted in patients with a history of transient ischemic attack (TIA), stroke and a murmur in the carotid artery area.

CSM is applied to clarify the diagnosis, especially in syncope work up, in people who are thought to have a sensitive carotid sinus. Hypersensitive carotid sinus findings comprise persistent asystole or a drop in systolic blood pressure > 50 mmHg, lasting for at least 3 seconds just after CSM.

3.2 Drug therapy

Six-milligrams of adenosine is given via IV route in patients with PSVT who do not respond to the VM (Class I, LOE B). Then 20 ml of saline is flushed via the same vein. If sinus rhythm is not restored within 1–2 minutes, repeat 12 mg of the agent [3]. Continuous ECG monitoring is required to keep track of the rhythm.

In case of continuation of SVT or triggering of AF/flutter, non-dihydropyridine group CCB (diltiazem or verapamil) (Class IIa, LOE B) or beta-blocker (metoprolol) need to be given (Class IIa, LOE C) to slow down the AV node for a long time. CCB should not be given in cases of decompensated HF or wide-complex tachycardia in adults and in young children. Likewise, concomitant use of different antiarrhythmic medications should be discouraged. Combined use of AV nodal blocking agents can cause severe bradyarrhythmias. Agents such as amiodarone, procainamide, sotalol can provide rate control in AF/flutter, and sometimes they can cause sinus rhythm. Thromboembolic complications should also be sought after conversion.

The aforementioned agents may not be beneficial in patients with PSVT triggered by preexcitation such as WPW or LGL, and they may trigger fatal arrhythmias by increasing the ventricular rate (Class III, LOE C). It will be correct to ask for a consultation.

4. Multifocal atrial tachycardia (MAT)

There are at least three different P wave morphologies originating from the atrium in the ECG tracks. There are also variable PP, PR and RR ranges. The heart rate is above 100 beats per minute and the rhythm is irregular. It can be mistakenly interpreted as AF. The typical patient is the elderly one with chronic lung disease such as COPD. Treatment is to correct the underlying disease. Standard antiarrhythmics are ineffective in suppressing multiple atrial ectopias and these agents may have toxic effects. Also, digoxin depresses the AV node and slows the ventricular rate. In the management, magnesium sulphate 2 g IV is given within 60 seconds; followed by administration of 1–2 g/hour with a constant infusion rate. Verapamil slows down the ventricular response with 5–10 mg IV administration, reduces ectopic stimuli in some patients, and provides conversion to sinus rhythm in many patients.

5. Atrial fibrillation: definition, recognition and treatment

Atrial fibrillation (AF) is the inactive 'worm bag-like' oscillations of the atria, which means there is no true atrium contraction. Different electrical vectors move in different directions simultaneously from the atrial myocardium, causing irregular and rapid impulses. Regular atrial activity is not recorded in AF. There are no P waves and

no sawtooth/flutter waves. There are only irregular, unequal oscillations called F waves. Although the atrial rate is around 400 bpm, it is limited by the refractory period and can be very variable. The RR intervals are unequal; thus it is an irregular rhythm. Acute AF, which prompts a visit to the ED is therefore common in the emergency, is often discloses a high ventricular rate.

AF is the most common rhythm disorder encountered and managed by emergency physicians following only sinus tachycardia [4, 5]. Majority of patients with AF and atrial flutter (AFL) can be treated and discharged in the ED without the need for hospitalization. As the age increases, AF is encountered more frequently, with a male predominance.

AF can be acute or chronic. Patients with acute AF (AAF) mostly present with a rapid ventricular response, that is, more than 100 beats per minute. In chronic AF, the ventricular response can be normal, high or low (**Figure 3A** and **B**).

AAF is the term used for AF that lasted no longer than 48 hours from the onset of an attack. It is an entity triggered by many reasons; poor outcomes of AAF arise mostly due to hemodynamic instability and thromboembolic events. The most common causes are mitral valve diseases, acute coronary syndromes (ACS)/ischemic heart disease and thyrotoxicosis (**Table 2**).

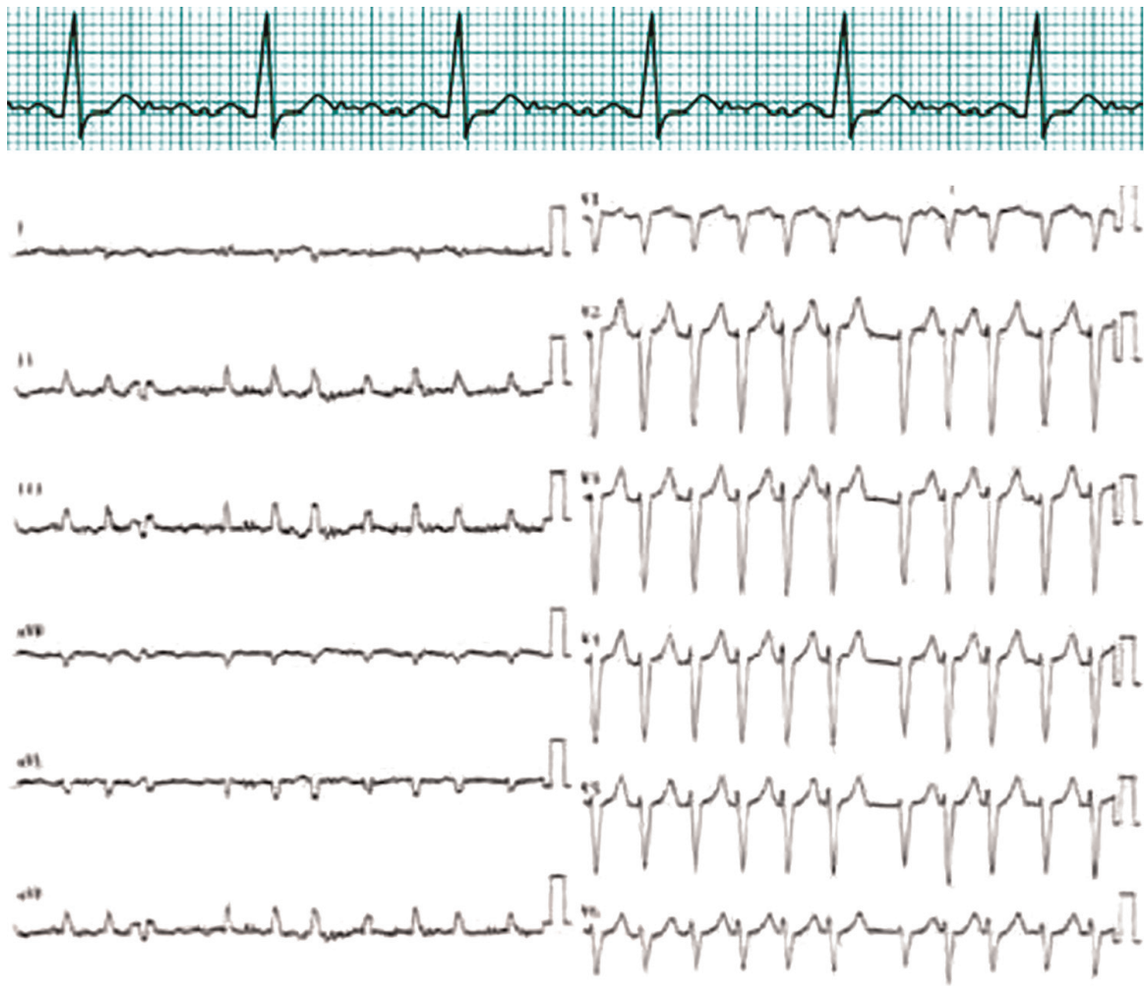

Figure 3.
Atrial fibrillation with normal (A) and rapid (B) ventricular response.

Causes that trigger AAF	Factors predisposing to acute or chronic AF
Structural cardiac abnormalities/cardiomyopathies/valvular diseases, mainly mitral	• atrial enlargement, • congestive heart failure • vagal stimulation, • atherosclerosis • hyperthyroidism • refractory period differs in various parts of the atrium.
Inflammation/infections/fever (may or may not affect the heart directly	
Fluid/electrolyte disorders, dehydration	
Strenous exercise/athletic training	
Hormonal and autonomic nervous system disorders	
ACS/atherosclerosis/coronary heart disease	
Intoxications: carbon monoxide/alcohol	
Hypoxemia	
Pulmonary embolism	
Overt release/intake of thyroid hormones	

Table 2.
Triggering causes of AAF and predisposing factors to acute or chronic AF.

In AF, HF may ensue because the heart cannot achieve pump activity as it did normally before, especially at high ventricular rates. Patients with AF are also predisposed to peripheral venous and atrial embolism with the risk of pulmonary and systemic arterial embolism. Like cancer, cardiovascular diseases including AF impose several risk factors and pathophysiologic mechanisms, including inflammation and alterations in platelet function, which ultimately result in thromboembolism [6]. Likewise, some risk factors increase tendency of AF and venous thrombosis together, such as obesity [7]. Five to 15% of patients with chronic AF can be expected to have thromboembolism once a year. Therefore, patients with chronic AF should regularly use anticoagulant medication while addressing other necessary measures to mitigate any other risk factors for thromboembolism.

6. Clinical status

Hemodynamic stability and recognition of factors which precipitate the arrhythmia are to be addressed at first. The onset of rhythm disorder should be especially questioned with special regard to acute/chronic distinction. It should be treated as chronic AF and the presence of thrombi should be ruled out if the clinician is not persuaded that it is an acute/*de novo* attack. Stroke risk should be evaluated with validated scores.

In patients with AAF a pulse rate of around 180 bpm which may cause instability, is recorded frequently, and in rare cases, it may pose a danger by exceeding 200 bpm.

In cases with Wolff-Parkinson-White (WPW) syndrome, which has an aberrant conduction pathway, extremely high heart rates can be life-threatening as a result of by-passed AV node [8].

Approximately half of the patients presenting with AAF return to sinus rhythm within 48 hours. This rule is especially true for those whose etiology is highlighted, and treatment is commenced.

AF frequently accompanies hypertension. In contrary developed countries, AF is also common in the setting of rheumatic heart diseases, especially with mitral stenosis in developing countries. Loss of atrial contractions, especially in cases of left ventricular failure, may lead to the acute exacerbation or worsening of HF.

Common complaints in patients with AF are fatigue, palpitations, chest pain, and shortness of breath. The reason for this is that the ejection fraction is reduced due to AF. Another important consequence of AF is that it predisposes to stroke and peripheral acute artery occlusions as a result of the formation of atrial thrombi. Troponin-positive coronary artery disease was detected in 5% of the patients who presented to the ED with AAF. Acute-onset AF, especially, should be considered ACS until proven otherwise. Thromboembolic disease is a major complication of AF and must be ruled out in every patient with AF. Five to 15% of these patients experience embolic/ischemic stroke every year.

Differential diagnosis of AAF includes entities such as AFL, PSVT, sinus rhythm, and AV nodal tachycardia [9]. These diseases can be easily differentiated from each other basically both with their clinical features and their ECGs.

6.1 Clinical distinction

Unlike the clinical features of AAF outlined above, PSVT cases describe the onset of the arrhythmia very clearly. Most of the cases have gained experience and are knowledgeable about CSM, Valsalva manoeuver, and rhythm correcting agents, and some even present to the ED only after failing to treat themselves with manoeuvers. Its findings are more stable, serious findings are rarely detected. Pharmacological cardioversion (PCV) with agents such as metoprolol and diltiazem is generally uneventful. Of these, verapamil is used less frequently than before, due to its side effect profile. Electrical cardioversion (ECV) is required in a small group, ECV can be applied with low doses such as 50 J.

Patients with AFL (flutter) can notice that the attack has started more clearly than those with AF, and they tend to come to the ED more acutely and a worse condition can be noted. It may accompany acute coronary syndromes including AMI and unstable angina pectoris. Therefore, these patients present with chest pain, dyspnea and hypotension more commonly in this group, compared to AF. The ventricular rate, or pulse rate, changes according to the degree of AV block, which directly affects the clinical status and stability. Since the atrial rate is generally around 300 bpm, the ventricular rate is often recorded as 75, 100 or 150 bpm, which exactly correspond to 1:2, 1:3, 1:4 blocks. For ECV, 30–50 J with monophasic defibrillators is usually sufficient.

Multifocal atrial tachycardia (MAT) is mostly seen in advanced stages of chronic lung diseases and critical HF. It has also been reported in cases of theophylline poisoning and those who consume caffeine excessively. Atrial rhythm is usually between 100 and 180 bpm. ECV is not indicated in this entity.

Differential diagnosis by ECG:

In a patient whose clinic is suitable for the diagnosis of MAT, at least three different P wave structures and variable P–P, P-R, and R-R intervals are sought in the ECG tracing. In AFL, at around 300 atrial velocities, regular waves called saw tooth (seesaw), characterized by the absence of isoelectric line, are seen. Findings are best observed in limb leads II, III, and aVF.

The presence of fibrillation (f) waves replacing the isoelectric line in the ECG in patients with AF, the absence of a consistent and continuously traceable P wave, are observed as 'irregular' rhythm characterized by the inequality of R-R intervals. As a rule, QRS waves are normal, that is, narrow. Delta waves and short PR should be investigated for concomitant WPW syndrome.

7. Imaging and laboratory

The purpose of imaging in AAF cases under emergency conditions is to collect information about differential diagnoses and triggering causes, and more importantly, to anticipate and prevent thromboembolic events.

There are no 'routine' laboratory examinations to be ordered in every case of AF, instead, a cost-effective list of workup can be culminated for each individual patient. For example, pulmonary embolism can be distinguished by POCUS-computed tomography-angiography. Other diseases associated with or triggering AF can be sought for via chest radiography; findings compatible with pneumonia, congestive heart failure, enlargement of the heart chambers, chronic pericarditis, aneurysm can be investigated. Carboxyhaemoglobin (COHb) level is critical in guiding the treatment in cases where carbon monoxide poisoning is thought to be the trigger for AAF attack. ECG, troponin and creatine kinase levels can yield vital findings in terms of acute ischemic heart disease. Haemoglobin and haematocrit should be requested in terms of acute haemorrhagic losses, blood urea nitrogen and creatinine levels should be requested for uremic pericarditis or RF. Oxygenation should be measured under emergency conditions with pulse oximetry, hypoxemia should be excluded and corrected, if any. In cases such as diabetic ketoacidosis or COPD, arterial blood gas analysis should be used.

In a recent study, it has been reported that patients with NT-proBNP levels below 450 pg./ml mostly reverted to sinus rhythm during their hospital stay, whereas patients with values above 1800 pg./ml are found to have persistent AF [10]. For this reason, BNP levels need to be requested in patients with dyspnea, in order to distinguish between heart failure and lung disease. If thyroid diseases such as thyrotoxicosis, Basedow-Graves' disease, multinodular goitere are considered as triggering causes, T3, T4 and thyroid stimulating hormone levels will be useful.

It is vital to exclude pericardial tamponade due to trauma and patients with suspected cardiac arrest (asystole or pulseless electrical activity) despite the electrical activity on the ECG under emergency conditions. In addition, POCUS/echocardiography evaluates the size and motion of the heart chambers, the presence of a heart-related mass, valve problems and the presence of intracardiac thrombus. In this way, important information can be obtained with a low-cost, simple application at the bedside.

The most important limitation of the technique is that its reliability and accuracy are dependent on the experience and ability of the operator. Since the results can also change with the position that can be given to the patient, there is a limitation in cases who cannot easily change position or those with obesity.

For ideal cardiac USG/POCUS, lowering the pulse is important. For this, depending on the condition of the patient with atrial tachyarrhythmia, beta-blocker or calcium channel blocker agents can be used.

Beta-blockers (metoprolol) may be preferred in young people with anxiety, coronary artery disease or hyperthyroidism, and calcium channel blockers in other groups and in patients who are contraindicated to beta-blockers such as asthma. PO/IV metoprolol should be preferred for beta blockade and IV esmolol in more urgent cases. Diltiazem, one of the calcium channel blockers, is the first choice due to its more positive side effect profile than verapamil. Concomitant administration of metoprolol and calcium channel blocker in selected cases is acceptable, but attention should be paid to dose adjustment and titration to effect.

Echocardiography can be performed by transesophageal (TEE) or transthoracic (TTE) methods in patients with AF. Since TEE is performed using the oesophageal probe, it both takes images from the window closer to the heart anatomically and gives a more accurate result as it eliminates the blocking effect of the chest and ribs. However, in some cases, it is difficult to tolerate and may take longer. TTE is a faster diagnostic tool chosen under emergency conditions. TEE is more sensitive in showing the left atrium, left atrial appendix (LAA), atrial septum, and aortic arch. For example, potential sources of cardiac embolism that may be overlooked by TTE in stroke cases can be determined by TEE.

The cause of cardiac embolism can be found in nearly 80% of the cases with TEE in patients with an uncertain cause of stroke. With TTE, this rate is lower, between 15 and 40%. TTE is often chosen in elderly patients with a known cardioembolic cause. TEE yields better results in patients under 50 years of age for whom the cause of stroke cannot be determined.

In a detailed echocardiographic examination to be performed in patients with AF, hemodynamic variables such as mean diastolic mitral gradient, pulmonary artery pressure, mitral valve area, tricuspid and mitral regurgitation, left atrial diameter should be measured and given quantitatively. In addition, the Wilkins mitral valve score is calculated. For example, in different studies, the mitral valve score and tricuspid valve involvement among those with mitral stenosis were found to be higher in patients with AF than those with sinus rhythm, indicating the prevalence of rheumatic activity [11].

Echocardiographic findings are closely related to the clinical course. For example, in studies examining the variables that affect the conversion of AF cases to sinus rhythm, the duration of AF attack less than 24 hours, young age, left atrial diameter and absence of primary heart disease appear as independent variables. Doğan et al. found only the duration of AF as an effective factor in conversion [12]. In this study, the mean left atrium diameter of patients presenting with AF was found to be 39.0 mm.

In recent years, after correction of AF with ECV, attention has been drawn to the 'stunning' phenomenon and related TTE findings have been underlined. Decreased LAA flow rates, decreased LAA discharge fraction, decreased transmitral inflow rates, and the appearance of spontaneous echo contrast has been reported as findings indicating atrial stunning.

8. Contemporary management of AAF in the acute setting

In cases presenting with AAF, the treatment of the underlying disease, eliminating pain and anxiety, providing oxygenation and correcting the hemodynamics are

primary and mandatory goals to achieve. After this treatment, a return to sinus rhythm can be accomplished without any other intervention in most cases.

Apart from this basic principle, there are three mainstays in the treatment of AF cases.

1. Provision of normal sinus rhythm and maintaining its stability,
2. Although AF is not terminated, reducing the heart rate to a normal/acceptable level
3. Prevention of formation of mural thrombi (besides ACS, some malignancies, aneurysm etc. can also lead to a tendency of thrombus formation)

8.1 Pearl

If rapid ventricular rate AAF is accompanied by rate-related symptoms (HF, pulmonary edema, dyspnea, impaired consciousness, ischemic chest pain), rate control should be provided immediately. As with flutter, agents such as diltiazem, verapamil or beta blocker (propranolol or metoprolol) can be used. ECV should be considered in those with acute hemodynamic compromise.

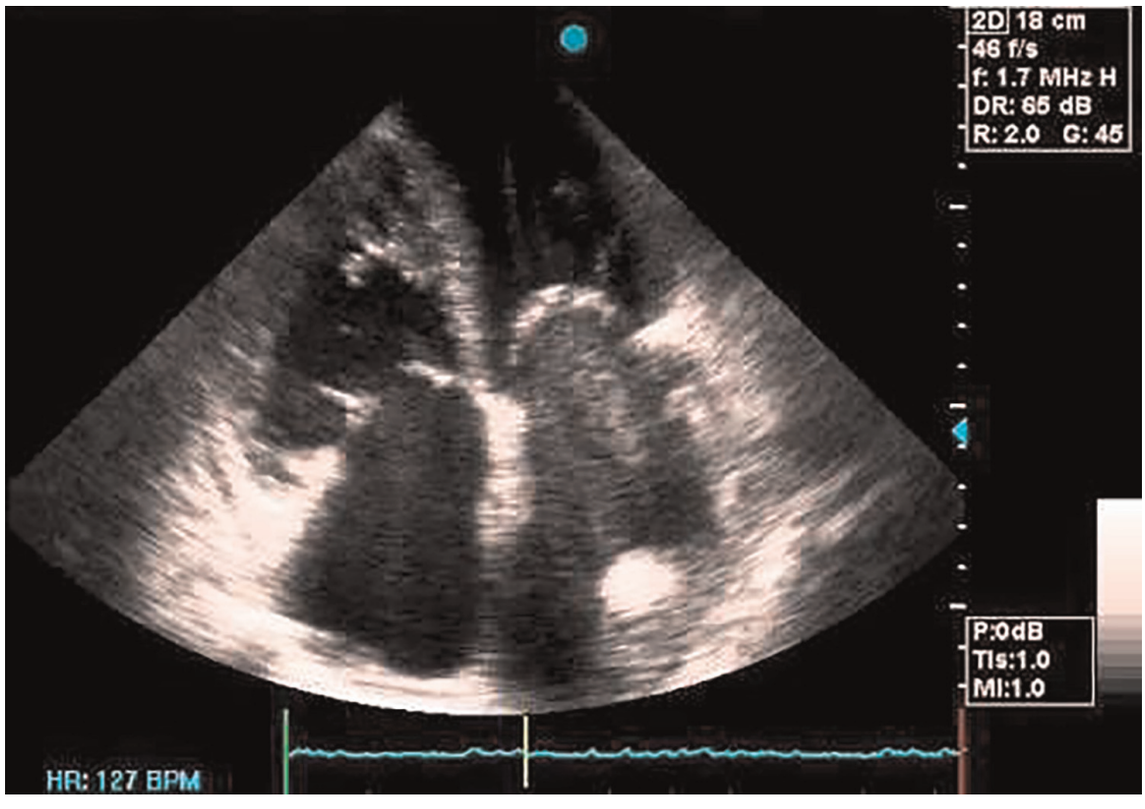

Figure 4.
Left atrial thrombus appearance in POCUS/ECHO in a female patient presenting with AF with rapid ventricular rate. The patient's condition was improved with medical therapy, diltiazem infusion, and electrical cardioversion was abandoned.

When AAF cases are considered to be hemodynamically stable, one of two ways is chosen in treatment. These two options, known as rhythm control or rate control, are selected and applied in accord with the patient's clinical characteristics. Stiell et al. in their population-based study in Canada examined whether the methods conducted in different centres differed from each other [4]. It was observed that the rhythm control strategy was applied to approximately 60% of 1018 AAF and AFL cases, and ECV was performed primarily for 40% of them. In total, 83% of the patients were discharged from the ED successfully. The variables that affect the choice of rhythm control strategy and the decision of ECV independently from other factors were determined as age, previous ECV history, presence of HF, and the centre admitted the patient (**Figure 4**).

In general, treatment strategies can be divided into conservative (preparation for elective ECV after speed control and anticoagulation) and aggressive (performing cardioversion as ECV or chemical/pharmacological cardioversion (PCV) in appropriate patients under safety precautions in the ED). It has been reported that there are important differences between physicians and disciplines in terms of approach to AAF.

9. Rhythm control and cardioversion (conversion to sinus rhythm)

ECV or PCV can be applied under emergency conditions to provide the conversion of AAF to sinus rhythm. The difference between cardioversion performed under emergency conditions and elective procedure is that the rhythm is returned to normal without anticoagulation in emergency practice, whereas in an elective procedure, it would be clarified that there is no thrombus and other contraindications are excluded, and the cardioversion procedure is performed.

Therefore, it should be ensured as much as possible that less than 48 hours have passed from the onset of AAF for ECV and that the patient does not pose a high risk for embolic stroke [5]. If you cannot be sure how long has passed, a rate control strategy should be adopted at first. Therapeutic anticoagulation should be commenced in these cases 3 weeks before and 4 weeks following ECV. However, in the case of unplanned cardioversion, AHA and ESC guidelines agree that cardioversion can be performed after an echocardiographic examination for thrombus exclusion [13]. Canadian Cardiology Society guidelines recommend that in patients with AF lasting for 12–48 hours, transesophageal echocardiography (TEE) is solely abdicable in patients with a low risk of stroke (CHADS2-score: 0–1). Since the duration of AF does not necessarily correlate with the occurrence of thrombi and AF-onset is often unsure, low-risk TOE prior to cardioversion should be employed [14].

In a very recent study, Bellone et al. randomized 247 patients into these two groups (ECV vs. PCV) [15]. The success rate of ECV was higher (89 vs. 73%). The mean time period spent in the ED is also much shorter in the ECV group (180 vs. 420 minutes). Side effects and adverse events occurred in a short time and in very small subgroups. Similarly, Cristoni et al. published a study in 2010, which showed that by establishing a short observation unit linked with the ED, these cases could be effectively treated without the need for hospitalization [16]. In this study, they were randomized to ECV (n = 171) and PCV (n = 151) groups, and discharge in sinus rhythm was achieved in 93 and 51%, respectively. During the 6-month follow-up, two patients in the ECV group had stroke. As a result, the treatment of AAF cases can be safely performed by applying ECV in the short observation unit.

More than 60% of patients return to normal sinus rhythm with 100 J and more than 80% with 200 J. ECV will benefit the patient only if the precipitating event is resolved. Therefore, ECV is only an emergency solution to save time (**Table 3**).

Drugs used in PCV are predominantly Class IC and III, according to Vaughan-Williams classification. Recently, Conti et al. reported that flecainide, propafenone or amiodarone were administered to a total of 378 patients in a non-randomized design for PCV in the Italian Eds, and 87% of the total group returned to sinus rhythm within 6 hours [17]. The success rates of the three drugs were 72, 55 and 30%, respectively ($p < 0.001$). Conversion times were also sorted by the same sequence, with an average of 178, 292 and 472 minutes ($p < 0.001$). They postulated that IC group antiarrhythmics (flecainide, propafenone) are apparently more efficacious than Class III drugs (amiodarone) and should be selected first for PCV. Propafenone 150 mg tablets, which are commercially available in most places from the IC group, can be administered orally as 300 or 600 mg. We can safely point out that PCV can be safely and successfully administered in the ED.

In a Croatian study, it has been reported that a success rate close to 100% has been achieved via PCV by administering three drugs from different pharmacological classes consecutively within a certain protocol [18].

Class I agents that produce antiarrhythmic effects by blocking sodium channels are divided into groups IA, IB and IC. Class IC drugs are one of the groups with the highest potential to terminate AAF via PCV. Propafenone, which is frequently used for this purpose in many centres, is also in this group. It should be selected in young patients with AF without structural heart disease. Bradycardia and heart failure, which can be life-threatening, can be seen rarely with this agent.

Although amiodarone (Class III; blocking sodium and potassium channels) is one of the most effective antiarrhythmic agents, it is less effective in the termination of

ESC	AHA/ACC/HRS	CCS
Favouring rhythm control:		
• First AF episode or short history	• Symptoms under rate control	• Highly symptomatic or significant Quality of life (QoL) impairment
• Younger age	• Younger age	• Recently diagnosed (<1 year)
• Arrhythmia induced cardiomyopathy	• First AF episode	• Multiple recurrences
• No or few comorbidities/heart disease	• Arrhythmia induced cardiomyopathy	• Difficulty to achieve rate control
• Normal to moderate increased Left atrial volume-index.(LAVI)/atrial conduction delay (limited atrial remodelling)	• Patient's choice	• Arrhythmia induced cardiomyopathy
• Rate control difficult to achieve	• Arrhythmia induced cardiomyopathy	
• AF precipitated by a temporary event	• Failed rate control	
• Patient's choice		

Table 3.
ESC, AHA/ACC/HRS and CCS guidelines on indications for rhythm control.

AAF by PCV. A feature that does not exist in other drugs is that it can be used safely in individuals with the poor general condition, the elderly and/or those with underlying structural heart disease.

9.1 Use of amiodarone for PCV

IV: Infused primarily 150 mg/10 minutes. Then 1 mg/min is infused for 6 hours, 0.5 mg/min for 18 hours. The maximum total dose is 10 g. It can be continued orally (100–200 mg/day).

Can the patients terminate AF themselves? Yes. One approach that can also be seen as a variant of the rhythm control strategy is that individuals with infrequent attacks are given a certain training, and these individuals immediately receive PCV by taking the appropriate medication right next to them. This approach, also known as the 'drug in the pocket' concept, can be used very beneficially in selected patient groups.

Flecainide (95%) and ibutilide (76%) were found to be the most effective drugs used for PCV in 376 patients with AAF in the ED [19]. Amiodarone, digoxin and diltiazem have very low success rates. A striking result is an observation that patients presenting with low blood pressure and who had a short period of time from the onset of AF to treatment revert to sinus rhythm with higher success.

9.2 Procedure recommendation

For PCV, propafenone from group IC can be used for conversion of AF to NSR. For this purpose, 600 mg propafenone (4 × 150 mg tb) is administered orally at a time and the patient is expected to convert within a few hours. This procedure is only recommended in the hospital.

ECG monitoring is recommended because a prolonged period of asystole, rarely syncope or prolonged bradyarrhythmias may occur during 'successful' conversion.

Medication	Starting dose (adult ~70 kg)	Dose/kg (for titration)	Duration of effect	Side effect
Midazolam (for sedation, anxiolysis)	1–2 mg IV	0.04–0.06 mg/kg (IV/IM)	60–90 minutes	Respiratory depression, hypotension (very rare at recommended doses)
Etomidate (for sedation, anxiolysis)	15–20 mg IV	0.2–0.3 mg/kg IV	20–30 minutes	Myoclonus, adrenocortical suppression in long-term administration
Fentanyl (for analgesia)	1–2 mcg/kg IV	0.5–1.0 mcg/kg IV	1–2 hours	Respiratory depression, chest rigidity (very rare at recommended doses)
Naloxone (narcotic antagonist)	0.2–0.4 mg IV (up to 2 mg)	0.4 mg IV	2–3 hours	Tachycardia

Table 4.
Sedatives and narcotics, doses, duration of action and expected side effects for sedoanalgesia used in ECV procedure in the ED. the drugs are used by an experienced emergency physician or anaesthesiologist, titrated with respect to the response of the patient.

10. Synchronized/simultaneous cardioversion

When midazolam and fentanyl are administered at recommended doses and under appropriate conditions, the side effect profile is at an acceptable level (**Table 4**). In the presence of signs of opioid overdose that may occur, albeit very rarely, naloxone should be used by titrating at the recommended doses in the table. Ketamine, which is widely used for sedoanalgesia in other indications, is not preferred in this group of cases as it may have tachycardic effects.

11. Rate control

Rate control strategy is a treatment approach preferred in patients with AF attacks thought to recur especially in those with structural heart disease, coronary artery disease, and advanced age. It is aimed to improve the symptoms by reducing the heart rate. Patients who are not selected/unsuitable for ECV are evaluated in the rate control group.

Antiarrhythmic agents such as beta-blockers (metoprolol), calcium channel blockers (diltiazem), and amiodarone are widely used for rate control. The serious toxic effects of amiodarone on many organs and systems limit its use. Digoxin is also a frequently used agent in the past, but it is rarely recommended in contemporary practice.

IV beta-blocker and nondihydropyridine CCB (diltiazem) is the drug of choice for acute rate control in AF with rapid ventricular response (Class IIa, LOE A).

Digoxin and amiodarone can be used for rate control in patients with CHF + AF. However, after the use of amiodarone, the risk of return to sinus rhythm and resultant embolization should be taken into account.

A large complex, irregularly irregular rhythm may reflect an AF with pre-excitation (WPW/LGL). Expert consultation should be obtained early.

- Adenosine, CCB, digoxin and β-blocking agents that cause AV nodal block should be avoided, as they pose a risk of increasing the ventricular rate.
- These cases are often converted to NSR with emergency ECV. If this is unavailable, other than above mentioned objectionable agents can be tried.

Procainamide is a widely used agent in most parts of the world for PCV. In a study in which the 'Ottawa Aggressive Protocol' put forward in Canada in the last decade was tested, 660 AAF (4.9% AFL) cases were taken as a cohort [20]. If it could not be understood whether the AAF attack lasted longer than 48 hours, the presence of mural thrombus was examined by TEE. Initially, IV procainamide was given to all cases, and a conversion rate of 58.3% was achieved. ECV procedure was performed in all remaining 243 cases, it was successful in 91.7% of them. Recurrent attacks were

observed in 8.6% of the cases within 7 days. The patients stayed in the ED for an average of 4.9 hours (3.9 hours in patients with PCV, 6.5 hours in patients with ECV) and 96.8% were discharged from the ED.

12. Antithrombotic-antiaggregant treatment in patients with AF

In AF cases in which effective contractions do not occur, blood may undergo stasis and turn into clots, especially in the left atrium. This phenomenon is clearly seen in AF patients lasting for 48 hours and older. The delivery of these clots to the arterial circulation via the aorta occurs most often during the conversion of AAF to normal sinus rhythm, so it is important to demonstrate that there is no clot for ECV/PCV of chronic AF. Arterial circulation may stop in any part of the body with this event called thromboembolism of mural thrombi. Apart from AF, cancer, haematological problems, coronary artery disease, heart failure and ventricular aneurysms can pave the way to thrombus formation. Acute arterial occlusion, renal infarction, acute mesenteric embolism may occur, but the most important morbidity is that it causes cerebral thromboembolism or stroke.

Warfarin (Coumadine) and heparin are agents that prevent fibrin formation, they are in the anticoagulant group. Acetyl salicylic acid (ASA, Aspirin) and clopidogrel (Plavix) or ticagrelor (Brilinta) are antiaggregant or antiplatelet agents. The combined use of P2Y12 inhibitor and ASA is called dual antiplatelet treatment (DAPT). Among these, warfarin prevents clot formation in the most efficient way, provides full effectiveness within a few days after its use, and requires monitoring with prothrombin time (PT) and international normalized ratio (INR) level every 4–8 weeks. In recent years, the 'triple antiplatelet therapy' (TAPT) method has been in question, with the addition of an agent from the DOAC group (such as dabigatran/rivaroxaban) to the DAPT regimen. Recent reports pointed out that TAPT strategy may be associated with higher bleeding episodes and mortality compared to a DAPT regimen—the combination of an anticoagulant and a P2Y12 inhibitor [21].

As of the update in January 2019, the following recommendations are in effect: [22].

- If the CHA2DS2-VASc score is > = 2 in AF + ACS cases, warfarin (vitamin K antagonist-VKA) 'triple therapy/triple therapy' (TAPT) is recommended in addition to the DAPT (aspirin + P2Y12 inhibitor) regimen.
- Double therapy in the form of warfarin (vitamin K antagonist) with P2Y12 inhibitor + dose adjustment after stenting is an acceptable treatment.
- P2Y12 inhibitor (clopidogrel or ticagrelor) + oral anticoagulant rivaroxaban, dabigatran or VKA with dose adjustment can be given as an alternative.

13. Does AF in my patient cause a cerebrovascular accident?

Yes, it can. In NVAF, the CHA2DS2-VASc score and risk criteria have been used in recent years instead of the CHADS2 criteria in 2001 in order to predict this more clearly (**Tables 5** and **6**).

Score	CHA2DS2-VASc risk criteria
1	CHF
1	Hypertension
2	Age ≥ 75
1	Diabetes mellitus
2	Stroke/TIA/thromboembolic event
1	Vascular disease (MI, PAH, aortic disease)
1	Age 65–74 years
1	Woman

Table 5.
CHA2DS2-VASc score and risk criteria.

CHA2DS2-VASc score	Annual stroke risk (%, per year)	Recommendation
0	0	No treatment
1	1.3	No treatment or aspirin or OAC
2	2.2	OAC
3	3.2	
4	4.0	
5	6.7	
6	9.8	
7	9.6	
8	6.7	
9	15.2	

Table 6.
CHA2DS2-VASc score and annual stroke risks with relevant treatment recommendations.

14. How is emergency anticoagulation done before ECV?

If PCV or ECV is planned in a patient with AF that is thought to last longer than 48 hours, one of the following three regimens should be started:

- emergency IV (unfractionated) heparin (target PTT, 60 seconds)
- or LMWH (enoxaparin 0.5 mg/kg or 40 mg once or twice daily)
- or VKA/warfarin for at least 5 days (target INR, 2.5).

When no thrombus is seen and ECV/PCV is successful, anticoagulation is continued for another 4 weeks (target INR, 2.5). If a thrombus is seen, ECV/PCV is delayed. TEE should be repeated before each ECV attempt.

At the earliest period in a hemodynamically unstable case with a suspected ECV

- emergency IV (unfractionated) heparin (target PTT, 60 seconds)
- or LMWH (at DVT therapeutic dose) should be initiated.

After successful cardioversion, VKA/warfarin (target INR, 2.5) is continued for at least 4 weeks. Long-term anticoagulation will depend on the patient's risk status, previous cardioversion procedures or another embolism status. For example, maintenance with rivaroxaban, one of the NOACs, is more suitable for those who have had a pulmonary embolism.

The above regimens are also valid in cases with flutter (Grade 2C).

The following agents were found to be effective in cases where treatment with OAC was considered:

- Warfarin (with the target INR in the range of 2–3)
- Dabigatran
- Rivaroxaban

Variable	**Score**
Hypertension	1
Renal failure (dialysis, transplant, creatinine >2.26 mg/dL or 200 μmol/L)	1
Liver disease (cirrhosis or bilirubin >2x normal + AST/ALT/ALP > 3x normal)	1
A history of stroke	1
A previous history of significant bleeding from any area	1
Labile INR (unstable/high INR, time within the therapeutic range < 60%)	1
Age > 65	1
Use of drugs such as aspirin, clopidogrel, NSAID that may cause bleeding tendency	1
Alcohol use (≥8 times a week)	1

HAS-BLED score	Risk group	Major bleeding risk (%)	Percentage of pts. with a bleeding annually	Recommendation
0	Low	0.9	1.13	
1		3.4	1.02	Anticoagulation can be considered
2	Moderate	4.1	1.88	Anticoagulation can be considered
3	High	5.8	3.72	Alternatives for anticoagulation can be considered
4		8.9	8.70	
5		9.1	12.50	
>5	Very high	—	—	Think about individualized options

Table 7.
HAS-BLED score.

- Apixaban
- Edoxaban

15. I am treating the patient with OAC. Do I cause a bleed in the patient?

Yes, there is a risk of bleeding. HAS-BLED score is the best tool developed for this (**Table 7**). This term acronym consists of the initials of the criteria 'Hypertension, Abnormal liver/renal function, Stroke history, Bleeding predisposition, Labile INR, Elderly, Drug/alcohol usage'.

16. Conclusion and summary

SVT AF represents a costly public health problem due to its high prevalence and high morbidity attributed to the disease itself and inherent complications. It causes serious consequences through hemodynamic complications and thromboembolic problems such as stroke and acute arterial occlusion. POCUS/echocardiography is vital in patients with AF because it shows potential thromboembolism at its source and also reveals cardiac functions, valve and chamber problems. Safe and effective pharmacological or electrical cardioversion (PCV/ECV) are current treatments of choice in cases with acute AF while selected cases will be candidates for rate control strategy. BB, CCB, adenosine, amiodarone and propafenone are among the most commonly used agents in this regard. There is not a standard regimen to apply to every case, but specific agents need to be selected in regard to the patient and the situation.

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