The emergency management of cardiac arrhythmias is a major challenge. Faced with potentially life-threatening situations, the treating physician has only a short time to make strategic decisions, initiate concrete measures, and see them through to completion. To do this correctly requires thorough knowledge of the various causes of cardiac arrhythmias and of the measures to be taken in an emergency.

Arrhythmias are acutely treated either with medications or with electrotherapy. Electrotherapy may be of any of the following types:

- external defibrillation or R-wave-synchronized cardioversion
- antitachycardic stimulation, e.g., to terminate ventricular tachycardias or atrial flutter
- antibradycardic stimulation, which, in the acute setting, usually consists of temporary transvenous or transcutaneous pacemaker treatment.

For hemodynamically unstable tachyarrhythmic patients, the precise diagnosis of the ECG pattern for specific treatment of the type of arrhythmia that is present is less important than rapid institution of effective treatment according to criteria that are as simple as possible and easy to recognize even under time pressure. The treatment of tachyarrhythmias in hemodynamically stable patients is primarily with medications (table) (1).

In this article, we will present practically applicable treatment algorithms for the differential treatment of bradyarrhythmic patients, as well as of tachyarrhythmic patients who are hemodynamically stable or unstable.

These treatment recommendations are based on the authors' longstanding clinical experience as well as on the recommendations of the European Resuscitation Council (2005) and on...
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### Antiarrhythmic agents for emergency treatment

<table>
<thead>
<tr>
<th>Agent</th>
<th>Acute treatment</th>
<th>Recurrence prevention</th>
<th>Extracardiac side effects</th>
<th>Contraindications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>IA</strong></td>
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<tr>
<td>Ajmaline</td>
<td>25–50 mg I.V.</td>
<td>Up to 300 mg I.V./12hr</td>
<td>Nausea, headache, feeling of warmth, cholestasis</td>
<td>Heart failure, bradycardia, Stokes-Adams attacks</td>
</tr>
<tr>
<td>Disopyramide</td>
<td>2 mg/kg BW I.V. (&lt; 150 mg)</td>
<td>400–600 mg po qd</td>
<td>Anticholinergic effects: dry mouth, visual and urinary disturbances</td>
<td>Heart failure, Bradycardia, Stokes-Adams attacks</td>
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<tr>
<td><strong>IB</strong></td>
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<tr>
<td>Lidocaine</td>
<td>1.5–2 mg/kg BW I.V.</td>
<td>60–120 mg/h I.V. (maximum 4 g/24hr)</td>
<td>If infused rapidly or at a high dose, central nervous manifestations including dizziness, seizures</td>
<td>Heart failure</td>
</tr>
<tr>
<td><strong>IC</strong></td>
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<tr>
<td>Flecainide</td>
<td>1–2 mg/kg BW I.V.</td>
<td>50–100 mg po bid</td>
<td>Dizziness, headache, diplopia</td>
<td>Heart failure, LVEF &lt; 35%, status post myocardial infarction (less than one year previously)</td>
</tr>
<tr>
<td>Propafenone</td>
<td>1–2 mg/kg BW I.V.</td>
<td>450–900 mg po qd</td>
<td>Visual disturbances, dizziness, gastrointestinal symptoms, insomnia</td>
<td>In addition: severe hypotension, severe COPD, myasthenia gravis</td>
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<tr>
<td><strong>II Beta-blockers (selected)</strong></td>
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<tr>
<td>Metoprolol</td>
<td>5–10 mg I.V. (up to 20 mg)</td>
<td>50–200 mg po qd</td>
<td>Bradycardia, fatigue, loss of drive, insomnia, dizziness, headache, intermittent claudication, psoriatiform rash, bronchospasm</td>
<td>High-grade AV block, cardiogenic shock, bradycardia, hypotension, COPD, bronchial asthma, PAOD, metabolic acidosis, prior I.V. administration of verapamil or diltiazem, simultaneous administration of MAO inhibitors</td>
</tr>
<tr>
<td>Esmolol</td>
<td>0.5 mg/kg BW I.V. over 2–3 minutes I.V.</td>
<td>0.1–0.2 mg/kg BW per minute I.V.</td>
<td>See metoprolol</td>
<td>In addition: hepatic or renal insufficiency, electrolyte disturbance, allergy, psoriasis</td>
</tr>
<tr>
<td><strong>III</strong></td>
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<tr>
<td>Sotalol</td>
<td>20 mg I.V. over 5 minutes (up to 1.5 mg/kg BW)</td>
<td>80–160 mg x 2 po tid</td>
<td>Hypotension, nausea, fatigue</td>
<td>See beta-blockers; in addition, preexisting QT prolongation</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>5 mg/kg BW I.V. (up to 450 mg)</td>
<td>I.V. or po loadings dose 0.6–1.0 g qd for 7–10 days; maintenance dose: 100–400 mg po qd</td>
<td>Thyroid dysfunction, keratodermia, light sensitization; rarely, interstitial pulmonary fibrosis, toxic neuropathy, hepatopathy</td>
<td>High-grade AV block, sinus node syndrome, thyroid dysfunction</td>
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<tr>
<td><strong>IV Calcium antagonists (selected)</strong></td>
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<tr>
<td>Verapamil</td>
<td>5–10 mg I.V.</td>
<td>80–120 mg po tid</td>
<td>Hypotension, gastrointestinal symptoms, constipation, ankle edema, headache</td>
<td>Heart failure, high-grade AV block, sinus node syndrome, atrial fibrillation in Wolff-Parkinson-White syndrome</td>
</tr>
<tr>
<td>Diltiazem</td>
<td>0.3 mg/kg BW I.V.</td>
<td>0.2–1 mg/min I.V. 60 mg po bid 90–120 mg po bid</td>
<td>Hypotension, headache, dizziness, ankle edema</td>
<td></td>
</tr>
<tr>
<td><strong>Miscellaneous</strong></td>
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<tr>
<td>Adenosine</td>
<td>3–12 mg I.V.</td>
<td>—</td>
<td>Flush, agitation, nausea, dysgeusia, dyspnea, bronchospasm</td>
<td>Second- and third-degree AV block, sick sinus syndrome, atrial fibrillation, atrial flutter, COPD, prolonged AT interval</td>
</tr>
</tbody>
</table>

Classification of antiarrhythmic agents into Classes IA, IB, IC, II, III, and IV according to Vaughan-Williams. AV block, atrioventricular block; COPD, chronic obstructive pulmonary disease; PAOD, peripheral arterial obstructive disease; MAO inhibitors, monoamine oxidase.
the Guidelines of the American College of Cardiology, American Heart Association, and European Society of Cardiology for the treatment of supraventricular tachycardias (2003). In contrast to many other clinical situations, it is often not possible to assign recognized recommendation and evidence levels for the emergency treatment of cardiac arrhythmias: most of the treatment recommendations are based on expert consensus, or on at most a few small and usually retrospective studies. For this reason, in the treatment recommendations of the European Resuscitation Council (2005), for example, there is no indication of the level of evidence in support of each treatment.

Because this article is intended as a summary of existing recommendations and does not itself constitute an official guideline, the authors have decided not to provide any independent indication of recommendation or evidence levels. When recommendations are supported by adequate empirical data, this will be stated directly.

**The emergency treatment of bradyarrhythmias**

Bradyarrhythmia is said to be present when the effective ventricular rate is less than 60 beats per minute. Typically, bradyarrhythmia with a ventricular rate less than 40 beats per minute requires emergency treatment. The extent and temporal urgency of the necessary treatment are a function of the hemodynamic effects of bradycardia and its associated clinical manifestations. Any identifiable causes should be determined, to the extent that this is possible in the acute situation, e.g., electrolyte disturbances, excessive vagal stimulation, pericardial tamponade, or myocardial ischemia. In cases of severe bradycardia or asystole with loss of consciousness, resuscitation should be initiated immediately (2, 3). Atropine or catecholamines should be given when resuscitation is begun, or as soon afterward as possible, until stimulation through a temporary pacemaker is available.

**Treatment algorithm for bradycardia**

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**Emergency treatment of marked bradycardia or asystole**

- Immediate resuscitation
- Concomitant administration of atropine or catecholamines until temporary pacemaker stimulation is available

**Hemodynamically unstable drug-resistant bradycardia**

- Temporary pacemaker stimulation (with a transvenous, transcutaneous pacemaker) is the preferred treatment, along with mechanical resuscitation.
For severe, symptomatic, but still hemodynamically compensated bradycardia, the primary treatment is with atropine. A precondition for atropine treatment, however, is the exclusion of an AV block below the bundle of His. This entity is characterized by a substituted rhythm with wide complexes or absent PQ prolongation before the loss of nodal conduction in second-degree AV block. Catecholamines should be given for the secondary treatment of severe, symptomatic, but still hemodynamically compensated bradycardia. If bradycardia persists, temporary pacemaker treatment must be instituted (4, 5).

Atropine – The indications for the administration of the parasympatholytic agent atropine are vagally mediated sinus bradycardia and asystole and AV block above the bundle of His. Atropine can be given in a dose of 0.5 to 1.0 mg every 2 to 5 minutes, up to a maximum dose of 0.04 mg/kg, corresponding to approximately 3 mg in a 70-kg patient. If venous access is unavailable, atropine can also be given through an endotracheal tube (2 to 3 times the I.V. dose in 10 to 20 ml of normal saline). If the ECG shows evidence of AV block below the bundle of His (second-degree AV block of Mobitz type or third-degree AV block with a substituted rhythm and a widened QRS complex), atropine may cause paradoxical bradycardia. In this setting, therefore, catecholamines should be used for primary treatment, or, even better, antibradycardic stimulation should be instituted as soon as possible.

Catecholamines: orciprenaline and adrenaline – The initial administration of catecholamines such as orciprenaline or adrenaline is recommended if the heart rate fails to rise adequately in response to atropine, or if asystole requiring resuscitation is already present at the beginning (2).

Temporary pacemaker treatment is the treatment of first choice for bradyarrhythmias requiring catecholamines and should be instituted soon afterward. Catecholamines are usually given as a bolus for the acute treatment of bradyarrhythmias that require them: orciprenaline, 0.25 to 0.5 mg I.V., or adrenaline, 0.02 to 0.1 mg I.V. (if given endotracheally, the dose is 2 or 3 times higher, in 10 to 20 ml of normal saline solution).

Selected indications for temporary pacemaker stimulation, according to the guidelines of the German Cardiological Society, are listed in the box (5).

The emergency treatment of tachyarrhythmias
Tachycardia is defined as a heart rate above 100 beats per minute, though a symptomatic or hemodynamically relevant emergency usually arises only when the heart rate is 150 beats per minute or higher. The mode of treatment in the acute situation critically depends on whether the patient is hemodynamically stable or unstable (diagram 2).

**Selected indications for temporary pacemaker stimulation**
- Acute myocardial infarction with 2nd-degree AV block, Mobitz II, 2:1 or higher-grade, or 3rd-degree AV block, alternating fascicular block, or progressive bifascicular block
- as a temporary treatment of symptomatic bradyarrhythmia until a permanent pacemaker can be implanted
- in bradyarrhythmia caused by acute intoxication
- in acute emergencies with asystole or atropine-resistant, symptomatic bradycardia of unknown cause

For severe, symptomatic, but still hemodynamically compensated bradycardia, the primary treatment is with atropine. A precondition for atropine treatment, however, is the exclusion of an AV block below the bundle of His. This entity is characterized by a substituted rhythm with wide complexes or absent PQ prolongation before the loss of nodal conduction in second-degree AV block. Catecholamines should be given for the secondary treatment of severe, symptomatic, but still hemodynamically compensated bradycardia. If bradycardia persists, temporary pacemaker treatment must be instituted (diagram 1) (4, 5).

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Tachycardia is defined as a heart rate above 100 beats per minute, though a symptomatic or hemodynamically relevant emergency usually arises only when the heart rate is 150 beats per minute or higher. The mode of treatment in the acute situation critically depends on whether the patient is hemodynamically stable or unstable (diagram 2).
Hemodynamically unstable tachycardia with shock, alteration of consciousness, or pulmonary edema should be treated as soon as possible with cardioversion and/or defibrillation. Hemodynamically unstable tachycardia with a narrow QRS complex, e.g., due to atrial fibrillation or flutter, usually responds to low defibrillation energies of 50 or 100 joules. On the other hand, polymorphic ventricular tachycardia or ventricular fibrillation should be treated primarily with defibrillation energies of at least 200 to 300 joules. If this fails, further defibrillations should be at the maximum energy (360 joules). Ventricular fibrillation or flutter that cannot be terminated is treated by the intravenous administration of amiodarone (150 to 300 mg I.V.) in addition to the usual resuscitation measures (6) (this recommendation is based on data from a prospective, randomized study). If the patient is still conscious but hemodynamically unstable and requires defibrillation or cardioversion because of the clinical manifestations of the arrhythmia, then premedication should be given, with either a sedative (e.g., midazolam) or an analgesic (e.g., an opioid). Analgesia and sedation can only be given, however, if they are readily available. In conscious patients, a brief attempt to alter the cardiac rhythm with the intravenous administration of a specific antiarrhythmic drug, such as amiodarone, can be made before external cardioversion (7).

Hemodynamically stable tachycardia is treated with medication. In general, a 12-channel ECG should be obtained in hemodynamically stable patients to enable classification of the tachycardia (diagram 3) as the basis for specific treatment, usually pharmacotherapy. The width of the QRS complex is the deciding criterion for a presumed supraventricular or ventricular origin of the tachycardia: if the QRS complex is narrow (less than 120 msec), this is considered to reflect physiological antegrade conduction in the His-Purkinje system, and the tachycardia is therefore presumed to have a supraventricular origin above the bundle of His.

As an exception to this rule, a wide QRS complex is seen in tachycardia of supraventricular origin or with antegrade conduction in the His-Purkinje system if there is a fascicular block or antegrade conduction in an accessory pathway in the Wolff-Parkinson-White (WPW) syndrome.

Tachycardia with a wide QRS complex is assumed to be of ventricular origin except in the following situations:

**Atrial fibrillation**
- Acute therapy usually consists of rate control
- In some cases, cardioversion is performed after clot formation has been ruled out

**The treatment of narrow-complex, regular tachycardia**
- Valsalva maneuver or carotid massage
- Adenosine if tachycardia fails to respond
- If there is still no improvement, calcium antagonists or beta-blockers are given
The acute treatment of atrial fibrillation or flutter (QRS width < 120 msec; irregular RR distance) may be either with medications that slow conduction in the AV node, such as verapamil, digitalis glycosides, or beta-blockers, or with cardioversion (diagrams 3 and 4).

Acute cardioversion should only be performed if atrial fibrillation is known with certainty to have been present for less than 48 hours, or if intra-atrial thrombus formation has been excluded with transesophageal echocardiography. In all other cases, rate normalization is the initial goal; it should be followed later by elective cardioversion.

"Narrow-complex" regular tachycardia (QRS < 120 msec) is treated initially with a vagal maneuver such as carotid massage or a trial Valsalva maneuver (8). If the tachycardia

**Sources of error in the treatment of tachyarrhythmias**
- Misdiagnosis of wide-complex tachycardia as supraventricular tachycardia
- Administration of verapamil I.V. in ventricular tachycardia (danger of hypotension and cardiac decompensation)

**A further source of error in the treatment of tachyarrhythmias**
- Intravenous administration of multiple antiarrhythmic agents, particularly ones with similar mechanisms of action
fails to respond, a rapid bolus of adenosine is given (6 mg at first, with possible further increments up to a total of 12 or 18 mg [9]). The tachycardia can be terminated with a dose of 12 or 18 mg of adenosine in 90% to 95% of cases, as long as it is not due to atrial fibrillation or atrial flutter. Alternatively, or if adenosine is ineffective, calcium antagonists or beta-blockers can be used (verapamil I.V.: 2.5 mg to 5 mg are given intravenously while the blood pressure is monitored; a second dose of 5 mg to 10 mg may be given, but the total dose should not exceed 20 mg). In case the tachycardia fails to respond to all of these measures while the patient’s hemodynamic and respiratory function remains stable, the administration of a specific antiarrhythmic agent is indicated, e.g., flecainide I.V., ajmaline I.V., or disopyramide (table).

It should be noted that the use of ajmaline in this situation is based on the authors’ clinical experience, the findings of small-scale prospective studies, and the usual practice in Germany, but is not found in the recommendations of the European Resuscitation Council. The reason for this may be that most intravenous formulations of class IC antiarrhythmic agents have not been approved for use in the USA. If pharmacotherapy fails, one must resort to electrical cardioversion under analgesia and sedation.

Wide-complex tachycardia (QRS ≥ 120 msec) is either of ventricular origin (in about 80% of wide-complex tachycardias of acute onset) or else of supraventricular origin with aberrant conduction. It is rarely due to atrial fibrillation, atrial flutter, or atrial tachycardia with accessory conduction in Wolff-Parkinson-White syndrome.

In the authors’ experience, most errors are made in the treatment of initially hemodynamically tolerated tachycardia with a wide QRS complex. The error typically consists of over-interpretation of the ECG, such as the misdiagnosis of a functional bundle branch block when ventricular tachycardia is the correct finding, or the misconception that

**Polymorphic ventricular tachycardia**

- Polymorphic ventricular tachycardia with prolongation of the QT interval requires specific treatment with high-dose magnesium as well as beta-blockers or catecholamines.
hemodynamic stability, mild symptoms, and tachycardia with a wide QRS complex necessarily imply the diagnosis of supraventricular tachycardia with bundle branch block. A correct statement would be that every tachycardia with wide complexes should be regarded as ventricular tachycardia until proven otherwise. Nonetheless, in rare cases, a hemodynamically stable patient whose tachycardia is thought to be of supraventricular origin can be initially treated with an intravenous adenosine bolus for diagnostic purposes, even if the QRS complex is wide (8).

The authors’ recommendations for the treatment of hemodynamically tolerated, regular tachycardia with a wide QRS complex are shown in diagram 3. In monomorphic, regular ventricular tachycardia, amiodarone or, alternatively, ajmaline can be given intravenously (7, 10). Here, too, the use of ajmaline is based on the authors’ clinical experience, the findings of small-scale prospective studies, and the usual practice in Germany, but is not found in the recommendations of the European Resuscitation Council. The reason for this may again be that most intravenous formulations of class IC antiarrhythmic agents have not been approved for use in the USA.

One of the advantages of using ajmaline to treat wide-complex tachycardia whose precise mechanism is unknown is that ajmaline can terminate tachycardia, or at least significantly lower the heart rate, even in cases of supraventricular tachycardia with bundle branch block. Ajmaline is also useful as an alternative treatment for atrial fibrillation with rapid accessory conduction in Wolff-Parkinson-White syndrome (11); this recommendation is supported by the findings of small, but nonetheless prospective and randomized studies; it is not found in current guidelines. It should be remembered that the I.V. administration of adenosine, verapamil, and digitalis is contraindicated. One of the reasons for this is that accessory atrioventricular conduction may be accelerated by a shortening of the antegrade refractory period of the accessory fibers while, at the same time, the conducting ability of the AV node is inhibited.

If acute infarction is in progress or an ischemic cause of tachycardia is suspected, then the treatment may be either with amiodarone or – as supported by the authors’ clinical experience – with lidocaine in a dose of 100 mg to 150 mg I.V. Other specific antiarrhythmic agents such as sotalol, flecainide, or propafenone play no more than a secondary role in the acute treatment of ventricular tachycardia, e.g., as second-line drugs in intensive care.

In cases of polymorphic ventricular tachycardia, it is very important to recognize "torsades de pointes" tachycardia, the setting of a congenital or acquired prolongation of the QT interval (3). This type of polymorphic ventricular tachycardia is due not to stable reentrant excitation, but rather to the presence of multiple focal discharges, producing a markedly fluctuating heart rate and the typical ECG appearance of tachycardia with reversing spikes. Such cases constitute a contraindication to the use of repolarization-prolonging antiarrhythmic agents such as sotalol or ajmaline. Their treatment is based, rather, on high doses of magnesium (1 g to 2 g I.V.) combined with beta-blockers or catecholamines, lidocaine, and electrotherapeutic measures (overstimulation).

Polymorphic ventricular tachycardia that is not due to delayed repolarization but rather to ischemic heart disease or dilated cardiomyopathy can be treated effectively with amiodarone (150–300 mg I.V.). Concomitantly, any electrolyte disturbance or cardiac ischemia that may be present must be treated, and a toxic effect of medications must be ruled out.

Like monomorphic ventricular tachycardia, polymorphic tachycardia that is not due to delayed repolarization can be treated with intravenous lidocaine and beta-blockers.

**Summary**

- The most important decision-making criteria in the emergency treatment of cardiac arrhythmia are hemodynamic stability or instability in tachycardia and the width of the QRS complex, which serves as a marker for a presumed ventricular or supraventricular origin of the disturbance.
Summary
The algorithms presented here enable rapid initiation of the correct therapeutic strategy in the emergency treatment of cardiac arrhythmia. The most important criteria for decision-making are hemodynamic stability or instability in tachycardia and the width of the QRS complex, which serves as a marker for a presumed ventricular or supraventricular origin of the disturbance.

Conflict of Interest Statement
The authors state that they have no conflict of interest as defined by the Guidelines of the International Committee of Medical Journal Editors.

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REFERENCES

Further Information
This article has been certified by the North Rhine Academy for Postgraduate and Continuing Medical Education. The Deutsches Ärzteblatt provides certified continuing medical education (CME) in accordance with the requirements of the Chambers of Physicians of the German federal states (Länder). CME points of the Chambers of Physicians can be acquired only through the Internet by the use of the German version of the CME questionaire within 6 weeks of publication of the article. See the following website: www.aerzteblatt.de/cme.

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The solutions to the following questions will be published in Volume 20/2007. The CME unit “Asperger’s Syndrome – an Autism Spectrum Disorder” (Volume 13/2007) can be accessed until the 11th May. For Volume 21/2007 we plan to offer the topic “Disorders of Blood Clotting”.

Solutions to the CME questionaire In Volume 9/2007:
Parzeller M, Wenk M, Zedler B, Rothschild M: Patient Information and Informed Consent before and after Medical Intervention: 1/a, 2/b, 3/a 4/d, 5/a, 6/a, 7/b, 8/b, 9/a, 10/a
Question 1
The following medications are used to treat life-threatening bradycardia:
(a) Sodium-channel blockers, such as lidocaine, to increase the frequency of spontaneous depolarization of the pacemaker cells
(b) Only adrenaline, because the treatment of life-threatening bradycardia requires not only an increase in the heart rate, but also an increase in blood pressure
(c) Intravenous administration of atropine and intravenous or endotracheal administration of catecholamines such as orciprenaline and adrenaline
(d) Beta-blockers, statins, and ACE inhibitors, in order to exert a long-term beneficial effect on cardiovascular risk status
(e) There are no effective medications for the treatment of life-threatening bradycardia.

Question 2
Which of the following measures is preferred for the treatment of hemodynamically unstable bradycardia that is resistant to treatment with medications?
(a) Forced mouth-to-nose respiration
(b) External chest compression
(c) Transcutaneous or transvenous-endocardial electrostimulation; if necessary, external chest compression until this is available
(d) Continuation of alternating I.V. administration of atropine and adrenaline
(e) If necessary, repeated defibrillation to activate the substitute pacemaker center

Question 3
The choice of cardioversion versus medical treatment to terminate a tachyarrhythmia depends primarily on the following parameters:
(a) The heart rate, as documented by ECG
(b) The width of the QRS complex
(c) The presence or absence of underlying heart disease
(d) Hemodynamic stability or instability during tachycardia
(e) The patient’s ability to perceive pain

Question 4
In hemodynamically unstable tachycardia with progressive impairment of consciousness, the following measures should be undertaken:
(a) Repeated administration of adenosine in increasing doses
(b) The immediate administration of amiodarone I.V. is preferred above all other measures, including other antiarrhythmic agents
(c) Rapid cardioversion or defibrillation
(d) Intubation and I.V. administration of catecholamines to stabilize circulatory function
(e) The most important measure is endocardial overstimulation to terminate the tachycardia with certainty.

Question 5
In the acute treatment of hemodynamically stable atrial fibrillation of uncertain duration, the measure of choice is:
(a) Anticoagulation with vitamin K antagonists to reduce the risk of stroke
(b) R-wave-synchronized cardioversion to terminate atrial fibrillation and prevent electrical remodeling
(c) Rate control with medications and cardioversion after thrombus formation has been ruled out
(d) The administration of an oral bolus of class IC antiarrhythmic agents for painless cardioversion without the need to rule out thrombus formation
(e) The administration of potassium and magnesium and of a loading dose of amiodarone
Question 6
Symptomatic atrial flutter should be treated with:
(a) Rapid cardioversion because of the risk of 1:1 conduction
(b) I.V. adenosine even if the diagnosis is clear
(c) Analogous treatment to atrial fibrillation
(d) Catheter ablation of the cavitricuspid isthmus, even as an emergency measure
(e) Anticoagulation for several weeks in all cases, because the risk of intracardiac thrombus formation is high, just as in atrial fibrillation

Question 7
The recommended treatment for the termination of hemodynamically stable, narrow-complex tachycardia is as follows:
(a) After vagal maneuvers have been performed, administer adenosine I.V.; alternatively, or if this is ineffective, give verapamil or other antiarrhythmic agents (class IC)
(b) Cardioversion with low defibrillation energy (50–100 joules)
(c) Amiodarone I.V. (300 mg / 30 min), then continuous infusion at 50 mg/hr for 7 to 10 days
(d) Electrical overstimulation through a transvenous stimulating electrode
(e) Digitalis glycosides I.V. and administration of potassium and magnesium

Question 8
Regular tachycardia with a wide complex is:
(a) A supraventricular tachycardia with bundle branch block (functional aberrancy)
(b) Always a supraventricular tachycardia with bundle branch block if it does not lead to hemodynamic instability
(c) Tachycardia of ventricular origin, and must be treated as ventricular tachycardia until proven otherwise
(d) Possibly due to atrial flutter with accessory AV nodal conduction
(e) The typical ECG appearance of ventricular tachycardia when the QT interval is prolonged ("torsades de pointes")

Question 9
Ventricular fibrillation that resists initial attempts to terminate it with medication should be treated with the following type of additional antiarrhythmic therapy:
(a) No further antiarrhythmic agents, because of the danger of proarrhythmia
(b) Beta-blockers I.V., e.g., metoprolol 5–10 mg.
(c) Class I antiarrhythmic agents, such as lidocaine; alternatively, or if these are ineffective, class IC agents (flecainide) or
(d) Magnesium I.V.
(e) Amiodarone I.V. followed by a continuous infusion

Question 10
Polymorphic ventricular tachycardia combined with QT prolongation in sinus rhythm requires specific treatment with the administration of:
(a) Ajmaline I.V. to terminate the tachycardia and shorten the QT interval
(b) I.V. administration of magnesium and lidocaine, and possibly raising the heart rate with antibradycardic stimulation or catecholamine administration
(c) Amiodarone I.V. especially to produce homogeneous rather than inhomogeneous QT prolongation
(d) Sedation, e.g., with diazepam I.V.
(e) Class IC medications (proprafenone, flecainide); digitalis glycosides in addition, if hemodynamically necessary (if there is evidence of heart failure)
Case illustration

Emergency treatment of cardiac arrhythmia

Surface ECG recording in leads I, II, III, aVR, aVL, aVF, V1, V6; paper speed 50 mm/sec

ECG and clinical findings
Tachycardia with a narrow QRS complex (duration 96 msec), heart rate 140/min, regular RR interval, no P-wave identifiable in the surface ECG.

The patient is a 54-year-old woman who has suffered from paroxysmal tachycardia for about 20 years. The episodes last up to 3 hours; they can usually be terminated with vagal maneuvers, but two recent episodes required termination with adenosine (12 mg I.V.). There has been no syncope to date and the left ventricular ejection fraction is normal. The patient states that the attacks have become more frequent in the last 6 months and now occur approximately once per week.

Differential diagnosis

Presumptive diagnosis
Following the differential diagnostic algorithm, one arrives at the following presumptive diagnosis: tachycardia with narrow complexes; regular; no visible P-wave; therefore, presumably AV-nodal reentrant tachycardia (AVNRT).

Basic therapeutic considerations
In general, medical treatment can be used for chronic recurrence prophylaxis (beta-blockers, calcium antagonists, class IC antiarrhythmic agents), but catheter ablation is recommended as the treatment of first choice for recurrent tachycardia due to AV-nodal reentry. When the rate is relatively slow, as in this case (140/min), and most episodes can be terminated with vagal maneuvers, the indication for catheter ablation is only relative and symptom-oriented. Thus, the patient should decide whether to undergo ablation after being fully informed about the risks and benefits of catheter ablation for AVNRT (greater than 95% success rate with a 1% risk of AV block).

Treatment provided and further course
The patient decided to undergo catheter ablation because the episodes had become more frequent and more difficult to terminate with vagal maneuvers. A typical “slow-fast” AVNRT was documented during the procedure. The slowly conducting pathway was modulated with two applications of radiofrequency energy. The patient has now been asymptomatic for three years since the procedure.

REFERENCES

Algorithm for the differential diagnosis of tachycardia with a narrow QRS complex (< 120 msec). AVNRT, AV-nodal re-entrant tachycardia; AVRT, atrioventricular re-entrant tachycardia; PJRT, permanent junctional reciprocating tachycardia; MAT, multifocal atrial tachycardia (according to the ACC/AHA/ESC Guidelines for the Management of Patients with Supraventricular Arrhythmias, Circulation 2003)