Antiarrhythmic drug therapy in patients with supraventricular or ventricular tachyarrhythmias in emergencies

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Abstract

Atrial fibrillation (AF), atrial flutter, AV-nodal reentry tachycardia (AVNRT) with rapid ventricular response, atrial ectopic tachycardia and preexcitation syndromes (AVRT) sometimes combined with AF or ventricular tachyarrhythmias (VTA) are typical arrhythmias in emergency patients. Most frequently, the diagnosis of the underlying arrhythmia is possible from the 12-lead surface electrocardiogram, the physical examination and the response to manoeuvres or drugs. In unstable hemodynamics, immediate DC-cardioversion is indicated. Conversion of AF to sinus rhythm (SR) is possible using antiarrhythmic drugs. Amiodarone has a conversion rate in AF of up to 80%. A new drug for AF conversion is vernakalant. Acute therapy of atrial flutter (Aflut) in intensive care pts depends on the clinical presentation. It can most often be successfully cardioverted to SR with DC-energies less than 50 joules. In narrow complex tachycardia, if the patient is hemodynamically stable, treatment should start with vagal manoeuvre. If tachycardia persists and atrial flutter is excluded, use of adenosine (6 mg as rapid i.v. bolus) is suggested. Successful termination by vagal manoeuvre or adenosine indicates that it was AVNRT or AVRT. If there is no response to adenosine (even after a second bolus) a longer-acting drug (e.g. verapamil, diltiazem) is recommended. Drugs like procainamide, sotalol, amiodarone or magnesium are recommended for treatment of VTA pts. However, today only amiodarone is the drug of choice in VTA pts and also effective even in pts with defibrillation-resistant out-of-hospital cardiac arrest.

Key words: tachyarrhythmias, intensive care, emergency medicine

Introduction

Emergency medicine and critical care are fields that require rapid diagnosis and intervention to avoid sudden cardiac death (1). These critical interventions can be life-saving or severely debilitating depending on their appropriateness and timeliness. In cardiac emergencies, accurate differentiation of ventricular and supraventricular tachyarrhythmias is essential for appropriate management. Most frequently, the diagnosis of the underlying arrhythmia is readily apparent, but occasionally it is necessary to use clues from the physical examination, the response to maneuvers or drugs, in addition to the 12-lead surface electrocardiogram. Treatment of supraventricular or ventricular arrhythmias in
emergencies is sometimes difficult (2,3). The purpose of the present manuscript is to summarize new strategies for patients with supraventricular and ventricular tachyarrhythmias under emergency situations.

**Atrial fibrillation**

Atrial fibrillation is the most frequent arrhythmia, both in surgical and cardiological intensive care units (4). Knotzer et al. (5) found that 14.8% of „surgically ill patients“ developed atrial tachyarrhythmias compared to 47.4% of patients treated in a cardiological ICU. The goal of acute treatment of atrial fibrillation with rapid ventricular response is to restore sinus rhythm (rhythm control). If cardioversion to sinus rhythm is not possible, the secondary goal is to slow the ventricular response, usually to a rate of less than 100 beats per minute (rate control). Patients who are hemodynamically unstable (significant hypotension, severe angina, pulmonary edema) should be promptly cardioverted after administration of an anesthetic agent (Fig. 1). Cardioversion should always be performed in a synchronized mode.

**Restoration of sinus rhythm**

In patients in whom the duration of atrial fibrillation is less than 48 hours DC-cardioversion should be considered early. Pharmacological conversion to sinus rhythm with antiarrhythmic drugs is a widely used therapeutic alternative with different efficacy rates (6). Amiodarone has a conversion rate in atrial fibrillation of up to 80% (7,8). However, the time interval between amiodarone administration and cardioversion is long, and therefore amiodarone is not the drug of choice for pharmacologic cardioversion in patients with atrial fibrillation. Iibutilide represents another class III antiarrhythmic agent that has been

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**Figure 1: Treatment algorithm for patients with tachyarrhythmias.** Abbreviations: ACS = acute coronary syndrome, DC = direct current, ECG = electrocardiogram, PE = physical examination
reported to have high conversion rates (9). Proarrhythmic effects occur in 5-8% of patients and careful monitoring is required. The conversion rates of recent-onset atrial fibrillation to sinus rhythm with ibutilide range from 50-70%. However, ibutilide is not available in Germany. A new drug for atrial conversion is vernakalant (10). It has been shown that the conversion rates are approximately 50%; however, vernakalant is very expensive and therefore of limited use in clinical practice (11).

In the setting of ineffective chemical cardioversion, electrical cardioversion may be an option that will restore more patients than chemical cardioversion. In Germany electrical cardioversion is preferred.

**Rate-control in atrial fibrillation with rapid ventricular response**

Both a loss of atrial synchrony and the rapid ventricular response may be poorly tolerated in hemodynamically compromised patients. Attempts to restore sinus rhythm are frequently unsuccessful or even debatable (12). Thus, heart rate control becomes the main therapeutic goal in this setting. It is well known for many years that treatment with intravenous digoxin, verapamil, beta-blocking agents, or diltiazem alone or in combination is effective in patients with atrial fibrillation and rapid ventricular response via blocking the AV-Node. Digoxin may be helpful for rate control with an initial dose of 0.5 mg. After 30 minutes, 0.25 mg digoxin should be administered again. In intensive care and emergency medicine other therapeutic strategies are verapamil (5-10 mg iv), diltiazem (20 mg iv) or beta-blocking agents as propranolol (1-5 mg iv,) and esmolol. However, beta-blocking agents or calcium channel blockers may cause additional hypotension. Delle Karth et al. (13) compared another pharmacological approach for rate control: they studied the role of amiodarone or diltiazem in a prospective, randomized trial. Sixty patients with atrial tachyarrhythmias (atrial fibrillation 57 patients, atrial flutter 2 patients, atrial tachycardia 1 patient) were randomized to diltiazem (25 mg bolus followed by a continuous infusion of 20 mg/hour for 24 hours)(group I), amiodarone (300 mg bolus)(group II) or amiodarone (300 mg bolus followed by 45 mg/hour for 24 hours)(group III). The primary end point was a >30% heart rate reduction within 4 hours. The secondary end point was a heart rate <120 beats/min. The primary end point was achieved in 70% of group I patients, 55% of patients in group II and in 75% of patients in group III (p=0.38). In patients achieving heart rate control, diltiazem showed a significantly better rate reduction when compared with group II and III (p<0.01). However, premature drug discontinuation due to hypotension was required significantly more often in group I (30%) than in group II (0%) or group III (5%)(p<0.01).

**Atrial flutter**

Acute therapy for patients with atrial flutter in emergencies depends on the clinical presentation. If the patient presents with acute hemodynamic collapse or congestive heart failure, emergent direct-current synchronized shock is indicated (Fig. 1). Atrial flutter can most often be successfully cardioverted to sinus rhythm with energies less than 50 joules. A number of drugs have been shown to be effective in conversion of atrial flutter to sinus rhythm. Placebo controlled intravenous ibutilide trials showed efficacy rates of 38-76% for conversion of atrial flutter to sinus rhythm (14,15). For those who responded to ibutilide, the mean time interval to conversion was 30 minutes; the efficacy of intravenous ibutilide (76%) was significantly higher than that of intravenous procainamide (14%)(16). Several single blinded, randomized control trials comparing intravenous flecainide with either intravenous propafenone or intravenous verapamil have shown relatively poor efficacy for acute conversion (5-13%); in addition, the conversion rate of intravenous sotalol varied from 20-40% depending on the
sotalol dose, but was not different from placebo. High dose (2 mg) of ibutilide was more effective than sotalol (1.5 mg/kg) in conversion of patients with atrial flutter to sinus rhythm (70% versus 19%) (17).

Acute management in narrow-QRS complex tachycardia

If the patient presents with narrow QRS-complex tachycardia (QRS width < 0.12 s) with acute hemodynamic collapse or congestive heart failure, emergent direct-current synchronized shock is indicated. However, while preparations are made for synchronized cardioversion, it is reasonable to give adenosine. In hemodynamically stable situations, vagal maneuvers should be initiated to terminate the arrhythmia or to modify AV conduction (Fig. 1). Carotid sinus massage is by applying pressure over the carotid artery on one side. If it is not working, use of the opposite side is recommended. Valsalva manoeuvre – expiration against the occluded glottis – is the most effective technique. If this fails, intravenous antiarrhythmic drugs should be administered for arrhythmia termination in hemodynamically stable patients. Adenosine, calcium channel blockers (verapamil) or beta-blocking agents are the drugs of first choice. The advantage of adenosine (9-12 mg iv) relative to intravenous calcium antagonists or beta-blockers relates to its rapidity of onset and short half-life. Longer acting agents (intravenous calcium channel blockers or beta-blocking agents) are of value, particularly for patients with recurrences of narrow QRS tachycardia. It is clear to avoid concomitant use of intravenous calcium channel blockers and beta-blocking agents because of possible increase of hypotensive and/or bradycardiac effects.

Acute management in wide QRS complex tachycardia

The patient with wide QRS complex tachycardia (QRS width ≥ 0.12 s) and hemodynamically unstable situation should be promptly cardioverted with a direct current synchronized shock (Fig. 1). Once the hemodynamically unstable patient has been cardioverted and stabilized, it is important to evaluate the preconversion 12-lead ECG for QRS configuration and signs of AV dissociation. For hemodynamically stable patients, a 12-lead ECG should allow an accurate diagnosis in the majority of patients. If after analysing the ECG the diagnosis is uncertain, the patient should be treated for VT. This is by far the most common diagnosis in patients with wide complex tachycardia and VT is potentially life-threatening (18,19).

Monomorphic ventricular tachycardia

In patients with sustained (duration > 30 sec) hemodynamically stable monomorphic ventricular tachycardia (Fig. 2) amiodarone plays an important role to terminate ventricular tachycardia (150-300 mg in 5 min i.v., followed by an infusion of 1000 mg/24 hrs.). Alternatives are the administration of procainamide (10 mg/kg i.v.) or ajmaline (25-50 mg i.v. over 5 min). In patients with VT and in the setting of acute myocardial ischemia, lidocaine (100-150 mg i.v.) was the treatment of choice for long term (20). However, it is well known that the efficacy of ajmaline is higher than the effect of lidocaine, whereas lidocaine is associated with high risk for degeneration of monomorphic ventricular tachycardia into ventricular fibrillation. Therefore, lidocaine is no more indicated in these patients and should be avoided. Other antiarrhythmic drugs like sotalol (20 mg in 5 min i.v.), propafenone (1-2 mg/kg i.v.) or flecainide (1-2 mg/kg i.v.) do not play a role as “first line” drugs in stable monomorphic VT (21-23). In 2012, these drugs are rarely used in monomorphic VT.
Polymorphic ventricular tachycardia

Less frequently, patients may present with polymorphic ventricular tachycardia (Fig. 3). Factors associated with this arrhythmia are electrolyte abnormalities, acute myocardial ischemia, reperfusion arrhythmia, proarrhythmic antiarrhythmic drugs with QT prolongation. The treatment of choice of polymorphic ventricular tachycardia is, after coronary care unit monitoring, discontinuation of the offending drug, and isoproterenol infusion with shortening repolarization and increasing the heart rate (1-4 µg/min i.v.) as initial steps. Atrial or ventricular pacing may be an option to suppress the polymorphic VT. In these situations with polymorphic ventricular tachycardia there is also a clear indication for emergency treatment with amiodarone (150-300 mg i.v., followed by infusion of 1000 mg/day)(20,22). Despite all considerations about the “ideal” therapeutic strategy, in polymorphic VT patients evaluation of the underlying disease and the mechanism of the arrhythmia is the most important step. In some cases, acute myocardial ischemia is present (“acute coronary syndrome”) and reperfusion therapy (PCI, bypass grafting) will be helpful in terminating the arrhythmia (24).

Torsade de pointes tachycardia

Torsade de pointes is a type of polymorphic ventricular tachycardia associated with marked QT prolongation. It may occur after administration of class Ia and class III antiarrhythmic drugs. The tachycardia is paroxysmal and may result in ventricular fibrillation and sudden death (25,26). Its onset is promoted by a slow basic rhythm and frequently follows a pause induced by a premature ventricular contraction. The tachycardia is characterized by polymorphic QRS complexes. Progressive lengthening of the QT interval and the development of a prominent U wave are important warning signs. The degree of QT prolongation that predicts torsade de pointes tachycardia is not known. However, prolonged QT syndromes may be congenital (Romano-Ward syndrome, Jervell-Lange-Nielsen syndrom) or acquired (class I a- and class III-antiarrhythmic drugs). The treatment in intensive care and emergencies is stopping...
the offending drug and correction of electrolyte abnormalities with potassium and magnesium. Intravenous magnesium sulfate (initial bolus of 2g i.v. over 10 min., another bolus of 2g after 15 min if the initial bolus failed, followed by a continuous infusion of 500 mg/h i.v.) may be effective, even if the serum magnesium level is within the normal range (27,28). Although magnesium has been used to treat arrhythmias for several decades, its mechanism of action and efficacy remain controversial (29,30).

Ventricular fibrillation and cardiac arrest

Approximately 1.000 people in the United States suffer from cardiac arrest each day, most often as a complication of an acute myocardial infarction with accompanying ventricular fibrillation. In 2010, the new resuscitation guidelines reported again the chain of survival concept, with four links – early access, cardiopulmonary resuscitation, defibrillation, and advanced care – as the way to approach cardiac arrest (24). It has been pointed out that the highest potential survival rate from cardiac arrest can be achieved only when the following sequence of events occurs as rapidly as possible: (a) recognition of early warning signs, (b) activation of the emergency medical services system, (c) basic cardiopulmonary resuscitation, (d) defibrillation, (e) management of the airway and ventilation, and (f) intravenous administration of medications. Prompt cardiopulmonary resuscitation with early defibrillation either by DC-countershock or an automated external defibrillator (AED) significantly improves the likelihood of successful resuscitation from ventricular fibrillation. According to the new

Figure 3: 12-lead ECG of a patient with fast polymorphic ventricular tachycardia after acute myocardial infarction.
guidelines, the antiarrhythmic drug of choice in patients with recurrent ventricular fibrillation or fast ventricular tachycardia refractory to DC defibrillation is amiodarone. It also appears to improve the response to DC-countershock. Amiodarone is a complex drug with effects on sodium, potassium, and calcium channels as well as alpha- and beta-adrenergic blocking properties. Amiodarone is a highly efficacious antiarrhythmic agent for many cardiac arrhythmias, ranging from atrial fibrillation to malignant ventricular tachyarrhythmias (31). In most published studies, intravenous amiodarone has been administered in patients with ventricular tachyarrhythmias only after failure of other antiarrhythmic drugs. In 1999, Kudenchuk described in 504 randomized patients with out-of-hospital cardiac arrest due to refractory ventricular arrhythmias (ARREST study) that treatment with amiodarone (single 300 mg dose of intravenous amiodarone) resulted in a higher rate of survival to hospital admission (44%) compared to placebo (34%) (p=0.03) (32).

Conclusions

In all patients with tachyarrhythmias, evaluation of the underlying etiology and the degree of left ventricular function (dysfunction) is essential. Correct treatment is based on an understanding of the mechanism that caused the situation. In hemodynamic unstable situation DC cardioversion/defibrillation is indicated. In hemodynamic stable patients there is still a place for antiarrhythmic drug treatment. However, in 2012 only few specific antiarrhythmic drugs are recommended. Amiodarone is the drug of choice in life-threatening ventricular tachyarrhythmias.

References

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