Arrhythmias in the intensive care patient
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Purpose of review
Atrial fibrillation, atrial flutter, AV–nodal reentry tachycardia with rapid ventricular response, atrial ectopic tachycardia, and preexcitation syndromes combined with atrial fibrillation or ventricular tachyarrhythmias are typical arrhythmias in intensive care patients. Most frequently, the diagnosis of the underlying arrhythmia is possible from the physical examination, the response to maneuvers or drugs, and the 12-lead surface electrocardiogram. In all patients with unstable hemodynamics, immediate DC-cardioversion is indicated. Conversion of atrial fibrillation to sinus rhythm is possible using antiarrhythmic drugs. Amiodarone has a conversion rate in atrial fibrillation of up to 80%. However, caution in the use of short-term administration of intravenous amiodarone in critically ill patients with recent-onset atrial fibrillation is absolutely necessary, and the duration of therapy should not exceed 24 to 48 hours. Ibutilide represents a relatively new class III antiarrhythmic agent that has been reported to have conversion rates of 50% to 70%; it seems that ibutilide is even successful when intravenous amiodarone failed to convert atrial fibrillation.

Recent findings
Newer studies compared the outcome of patients with atrial fibrillation and rhythm- or rate-control. Data from these studies (AFFIRM, RACE) clearly showed that rhythm control is not superior to rate control for the prevention of death and morbidity from cardiovascular causes. Therefore, rate-control may be an appropriate therapy in patients with recurrent atrial fibrillation after DC-cardioversion. Acute therapy of atrial flutter in intensive care patients depends on the clinical presentation. Atrial flutter can most often be successfully cardioverted to sinus rhythm with energies less than 50 joules. Ibutilide trials showed efficacy rates of 38–76% for conversion of atrial flutter to sinus rhythm compared with conversion rates of 5–13% when intravenous flecainide, propafenone, or verapamil was administered. In addition, a high dose (2 mg) of ibutilide was more effective than sotalol (1.5 mg/kg) in conversion of atrial flutter to sinus rhythm (70% versus 19%).

Summary
There is general agreement that bystander first aid, defibrillation, and advanced life support is essential for neurologic outcome in patients after cardiac arrest due to ventricular tachyarrhythmias. The best survival rate from cardiac arrest can be achieved only when (1) recognition of early warning signs, (2) activation of the emergency medical services system, (3) basic cardiopulmonary resuscitation, (4) defibrillation, (5) management of the airway and ventilation, and (6) intravenous administration of medications occurs as rapidly as possible. Public access defibrillation, which places automatic external defibrillators in the hands of trained laypersons, seems to be an ideal approach in the treatment of ventricular fibrillation. The use of automatic external defibrillators by basic life support ambulance providers or first responder in early defibrillation programs has been associated with a significant increase in survival rates. Drugs such as lidocaine, procainamide, sotalol, amiodarone, or magnesium were recommended for treatment of ventricular tachyarrhythmias in intensive care patients. Amiodarone is a highly efficacious antiarrhythmic agent for many cardiac arrhythmias, ranging from atrial fibrillation to malignant ventricular tachyarrhythmias, and seems to be superior to other antiarrhythmic agents.

Keywords
atrial fibrillation, arrhythmias, amiodarone, ibutilide, automatic external defibrillators

Introduction
Emergency medicine and critical care are fields that often require rapid diagnosis and intervention for specific situations [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 [2]. 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 [3,4]. Treatment of cardiac arrhythmias in intensive care and emergency medicine is sometimes difficult. Correct therapy based on an understanding of the mechanism that caused the arrhythmia may not only be lifesaving in the immediate situation, but may also improve the quality of life. The purpose of the present chapter is to summarize new strategies for patients with supraventricular or ventricular tachyarrhythmias in intensive care or cardiac emergencies.
Types of tachyarrhythmias
Supraventricular tachyarrhythmias include atrial fibrillation, atrial flutter, AV-nodal reentrant tachycardia with rapid ventricular response, atrial ectopic tachycardia, and preexcitation syndromes combined with atrial fibrillation. Ventricular tachyarrhythmias still remain the main cause of death; these arrhythmias include monomorphic and polymorphic ventricular tachycardia, torsade de pointes tachycardia, ventricular flutter, and ventricular fibrillation. In the assessment of patients with life-threatening supraventricular or ventricular arrhythmias, attention should be given to identify whether the tachycardia is associated with worsening angina or low cardiac output. There is general agreement that many cardiac or extracardiac causes can lead to supraventricular or ventricular tachyarrhythmias and to cardiac arrest; left ventricular function (or dysfunction) plays an important role in prognosis and outcome (Fig. 1).

Incidence and type of cardiac arrhythmias in critically ill or elderly patients
It has been well known for many years that cardiac arrhythmias can occur in healthy people or in patients with cardiac or extracardiac diseases. Arrhythmias are well defined in patients after myocardial infarction, in patients with underlying cardiac or pulmonary disease, and in patients after cardiac surgery or heart transplantation [5]. However, only few data are available regarding incidence and type of arrhythmias in critically ill patients. Reinel et al. studied all consecutive arrhythmia episodes in critically ill patients in a medical–cardiologic intensive care unit between 1996 and 1999 [5]. All episodes of patients with new-onset, sustained arrhythmias (duration ≥30 seconds) were included that were either self-terminated or that required intervention. A total of 310 arrhythmia episodes were assessed during the study period in 135 patients, with a mean of 2.91 episodes per patient (range 1–14). Admission diagnoses were cardiac (n = 48), cardiac surgery (n = 45), resuscitation (n = 12), pulmonary (n = 15), sepsis (n = 5), neurologic (n = 2), and others (n = 6). Among the 310 arrhythmia episodes, there were 278 tachycardia (179 episodes regular, 97 episodes irregular) and 32 bradycardia events (heart rate <40 beats/min). There were 108 narrow-QRS complex tachycardia and 168 wide-QRS complex tachycardia with two episodes of primary ventricular fibrillation. Among the 278 tachycardias, 135 episodes (48.6%) were ventricular, 13 episodes (4.7%) were torsade de pointes tachycardia, and 83 episodes (29.8%) were atrial fibrillation, 10 episodes were atrial flutter (3.6%), 21 were supraventricular tachycardia (7.6%), and 2 were ectopic atrial tachycardia (0.7%). It seems that in critically ill patients, two types of arrhythmias are dominantly visible: sustained ventricular tachycardia and atrial fibrillation. Lampert and Ezekowitz reported recently that the aging heart is susceptible to diverse cardiac arrhythmias, and in many of these arrhythmias intensive care medicine is mandatory [6]. Baine et al. studied the incidence of arrhythmia types, hospital and intensive care stay in 144,512 elderly patients (age 65 years or older) with cardiac arrhythmias (time interval 1991–1998) [7]. In 1998, atrial fibrillation was the most frequent arrhythmia and accounted for 44.8% of the relevant discharges. Atrial flutter was observed in 5.2%, sinoatrial node dysfunction in 13.2%, and complete AV block in 5.8%. Supraventricular tachycardia was observed in only 3.8%. Cardiac arrest was present in 1.3%, ventricular tachycardia in 6.9%, and ventricular fibrillation in 1.3%. It is interesting to note that lengths of stay in hospital and intensive care are determined by the underlying arrhythmia: mean hospital stay was 7.8 days.

Figure 1. Diagnostic methods in intensive care patients after cardiac arrest caused by ventricular fibrillation
Mechanisms and diagnostic methods in intensive care patients after cardiac arrest caused by ventricular fibrillation. Angio, angiography; BRS, baroreflex sensitivity; ECG, electrocardiogram; EPS, programmed electrical stimulation; HRV, heart rate variability; LVEF, left ventricular ejection fraction.
in ventricular fibrillation, 7.0 days in ventricular tachycardia in contrast to 4.7 days in atrial fibrillation, 4.5 days in atrial flutter, or 5.6 days in complete AV block. Mean intensive care stay was 2.7 days in ventricular fibrillation, 1.7 days in ventricular tachycardia, 0.9 days in atrial fibrillation, 1.0 days in atrial flutter, or 1.5 days in complete AV block. In addition to the study by Reinelt et al., management of atrial fibrillation is also a major problem in elderly patients.

**Atrial fibrillation**

Since the time of Hippocrates, there has been interest in how the heartbeat is initiated. It was concluded that the nervous system initiates the heartbeat. In 1907, Keith and Flack described the sinus node and suggested it as the site of initiation within the atrium. It became clear that electrical disturbances in the atrium (disruptions in the normal spread of activation) will lead to atrial arrhythmias. Clinical, there is an increased interest in atrial arrhythmias, particularly atrial fibrillation, because the incidence of this arrhythmia is high and the associated morbidity and mortality higher than previously recognized.

**Acute management**

Atrial fibrillation or flutter is the most frequent arrhythmia in emergency rooms and intensive care units, both in surgical and cardiologic intensive care units [8]. Knotzer et al. found that 14.8% of surgically ill patients developed atrial tachyarrhythmias compared with 47.4% of patients treated in a cardiologic intensive care unit [5,9]. The goal of acute treatment of atrial fibrillation with rapid ventricular response is to restore sinus rhythm or to control the ventricular rate (Fig. 2). 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. Patients who are hemodynamically unstable (significant hypotension, severe angina, pulmonary edema) should be promptly cardioverted after administration of an anesthetic agent. Cardioversion should always be performed in a synchronized mode.

**Hemodynamically stable situation: restoration of sinus rhythm**

In patients in whom atrial fibrillation is of known acute onset (present for less than 48 hours) DC-cardioversion should be considered early. Pharmacologic conversion to sinus rhythm with antiarrhythmic drugs is a widely used...
therapeutic alternative with different efficacy rates [10]. Amiodarone has a conversion rate in atrial fibrillation of up to 80% [11]. However, only a single retrospective study has been published that includes intensive care unit patients with left ventricular dysfunction, although this is frequent in critically ill or even in elderly patients with recent onset of atrial fibrillation [12]. In critically ill patients, amiodarone is indicated for a wide range of arrhythmias including recent-onset atrial fibrillation. Despite many published reports on safety and efficacy of intravenous amiodarone in the treatment of patients with recent-onset atrial fibrillation, there are an increasing number of reports in critically ill patients highlighting occasional serious acute pulmonary toxicity [13]. Therefore, caution in the use of short-term administration of intravenous amiodarone in the critically ill patient with recent-onset atrial fibrillation is absolutely necessary, and the duration of therapy should not exceed 24 to 48 hours, except when absolutely necessary. In contrast to amiodarone, ibutilide represents a relatively new class III antiarrhythmic agent that has been reported to have high conversion rates [14]. Proarrhythmic effects occur in 5 to 8% of patients, and careful monitoring is required. The conversion rates of recent-onset atrial fibrillation to sinus rhythm with ibutilide range from 50 to 70%, and it seems that ibutilide is even successful when intravenous amiodarone failed to convert atrial fibrillation [15]. Hennersdorf et al. studied 26 patients in whom atrial fibrillation or flutter persisted for a maximum of 6 hours. All patients initially received amiodarone (150 mg IV) and after 2 hours of persistent arrhythmia ibutilide (1 mg IV; in the case of persisting arrhythmia and body weight > 70 kg, a second infusion of 1 mg ibutilide was administered after 30 minutes). Before the administration of ibutilide, magnesium (1 g) was administered, and high normal levels of potassium serum levels were achieved (4.5 to 5.0 mmol/L). After amiodarone administration, atrial flutter persisted in 73% and atrial fibrillation in 27% of patients. After administration of ibutilide, conversion to sinus rhythm was achieved in 82% patients after a median time of 7 minutes (range 3 to 12 minutes). The rate of cardioversion with ibutilide was higher in atrial flutter (84% patients) than in atrial fibrillation (71% patients). Therefore, in implanted automatic cardioverter defibrillator patients with atrial fibrillation or flutter, ibutilide is an effective drug even when amiodarone failed. Ibutilide seems to be well suited for cardioversion of recent-onset atrial fibrillation or flutter [15].

**Hemodynamically stable situation: rate control or rhythm control?**

In every intensive care unit, there are many patients with recent-onset atrial fibrillation. Within the last few years, cardioversion to sinus rhythm was the goal to treat these patients. However, there are new important studies reporting the role of rate control in these patients: the AFFIRM and the RACE studies [16••,17••]. Although these studies are not performed in critically ill patients, they have many implications for the management of patients with atrial fibrillation, arriving in an intensive care unit. A total of 4060 patients (mean age 69.7 ± 9.0 years) with atrial fibrillation and a higher risk of stroke or death were enrolled in the AFFIRM study [16••]. Patients were assigned to rhythm-control (2033 patients) or to rate-control therapy (2027 patients). The primary endpoint of this study was overall mortality. In the rhythm-control group, more than two thirds of patients started therapy with amiodarone or sotalol. By the end of the study almost two thirds of the patients in this group had undergone at least one trial of amiodarone. In the rate-control group, beta-blocking agents were used initially in nearly one half of the patients, and of the calcium-channel blockers, diltiazem was used more frequently than verapamil. In this study, there were 356 deaths among the patients assigned to rhythm-control therapy and 310 deaths among patients assigned to rate-control therapy ($P = 0.08$). The prevalence of sinus rhythm in the rhythm-control group at follow-up was 82.4%, 73.3%, and 62.6% at 1, 3, and 5 years, respectively. In the rate-control group, at 5 years, 34.6% of the patients were in sinus rhythm, and over 80% of those in the atrial fibrillation group had adequate heart rate control. More patients in the rhythm-control group than in the rate-control group were hospitalized, and there were more adverse drug effects in the rhythm-control group as well. In both groups, most strokes occurred after warfarin had been stopped or when the international normalized ratio was subtherapeutic. From this study it seems clear that management of atrial fibrillation with the rhythm-control strategy offers no survival advantage over the rate-control regimen. In the RACE study, 522 patients with persistent atrial fibrillation after a previous electrical cardioversion were randomized to a rhythm-control (266 patients) or a rate-control group (256 patients) [17••]. The endpoint was a composite of death from cardiovascular causes, heart failure, thromboembolic complications, bleeding, implantation of a pacemaker, and severe adverse effects of drugs. Patients in the rhythm-control group were treated with sotalol (160 to 320 mg daily), flecainide (200 to 300 mg daily), or propafenone (450 to 900 mg daily). If there was a recurrence within 6 months after start of the drug treatment, a loading dose of amiodarone was given (600 mg daily for 4 weeks) and thereafter continued with 200 mg daily. Rate control was achieved with the administration of digitalis, calcium channel blockers, and beta-blocking agents, alone or in combination. After a mean of 2.3 ± 0.6 years, 39% of patients in the rhythm-control group had sinus rhythm, as compared with 10% of patients in the rate-control group. The primary endpoint occurred in 22.6% of patients in the rhythm-control group and in 17.2% of patients in the rate-control group. The distribution of the various components of the primary endpoint was similar between both groups. Data from this study clearly showed that rhythm control is not
superior to rate control for the prevention of death and morbidity from cardiovascular causes. Therefore, rate control may be an appropriate therapy in patients with a recurrence of atrial fibrillation after electrical cardioversion.

**Alternatives for 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 [3]. Attempts to restore sinus rhythm are frequently unsuccessful or (after AFFIRM and RACE) even debatable [16•,17••]. Heart rate control becomes the main therapeutic goal in this situation. The optimal regimen for pharmacologic rate control during atrial fibrillation in critically ill patients is unclear. It has been well known for many years that treatment with intravenous digoxin, verapamil, beta-blocking agents, or diltiazem is effective in patients with atrial fibrillation and rapid ventricular response [1]. 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 to 10 mg IV), diltiazem (20 mg IV), or beta-blocking agents as such as propranolol (1 to 5 mg IV, additional infusion of 10 to 120 mg per day) and esmolol (500 µg/kg over 1 minute, followed by a 4-minute maintenance infusion of 50 µg/kg/min with further dose adjustment as necessary) [11]. However, beta-blocking agents or calcium channel blockers may cause additional hypotension. Just recently, Delle Karth et al. compared another pharmacologic approach for rate control in critically ill patients: these authors studied the role of amiodarone or diltiazem in a prospective, randomized trial [18]. 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/h for 24 hours) (group I), amiodarone (300 mg bolus) (group II) or amiodarone (300 mg bolus followed by 45 mg/h for 24 hours) (group III). The primary endpoint was a >30% heart rate reduction within 4 hours. The secondary endpoint was a heart rate <120 beats/min. Tachyarrhythmias were considered uncontrolled if the heart rate was >120 beats/min four hours after study drug administration. The primary endpoint 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). Uncontrolled tachyarrhythmias were observed more frequently in group II (45%) than in group I (0%) or group III (5%). This study shows that sufficient rate control can be achieved in critically ill patients with atrial tachyarrhythmias using either diltiazem or amiodarone. Although diltiazem allowed significantly better 24-hour heart rate control, this effect was offset by a significantly higher incidence of hypotension requiring discontinuation of the drug. It seems clear that amiodarone may be an alternative in patients with severe hemodynamic compromise.

**Atrial flutter**

Acute therapy for patients with atrial flutter in intensive care depends on the clinical presentation. If the patient presents with acute hemodynamic collapse or congestive heart failure, emergent direct-current synchronized shock is indicated. Atrial flutter may 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 to 76% for conversion of atrial flutter to sinus rhythm [19,20]. For those who responded to ibutilide, the mean time interval to conversion was 30 minutes. In the largest available study, the efficacy of intravenous ibutilide (76%) was significantly higher than that of intravenous procainamide (14%) [21]. 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 to 13%); in addition, the conversion rate of intravenous sotalol varied from 20 to 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%) [22].

**Narrow-QRS complex tachycardia**

Narrow QRS tachycardia is a cardiac rhythm with a rate faster than 100 beats per minute and a QRS duration of less than 0.12 seconds. The patient with narrow QRS tachycardia usually seeks medical attention because of palpitations, light-headedness, shortness of breath, or anxiety. In many patients with narrow-QRS complex tachycardia, the tachycardia rate is very high (180-240 beats per minute) and therefore, after onset of the tachycardia the patient will arrive very soon in an intensive care unit for diagnosis and treatment. In intensive care and emergency medicine, narrow QRS complex tachycardia is a common problem [1].

**Acute management**

The definitive diagnosis can be made in most of the patients based on 12-lead electrocardiogram and clinical criteria. Acute (and chronic) treatment should be initiated based on the underlying mechanism. In regular narrow QRS complex tachycardia, vagal maneuvers should be initiated to terminate the arrhythmia or to modify AV conduction. If this fails, intravenous antiarrhythmic drugs should be administered for arrhythmia termination.
in hemodynamically stable patients. Adenosine, calcium channel blockers, or beta-blocking agents are the drugs of first choice. The advantage of adenosine relative to intravenous calcium antagonists or beta-blockers relates to its rapidity of onset and short half-life [3,4]. 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 clearly necessary to avoid concomitant use of intravenous calcium channel blockers and beta-blocking agents because of possible increase of hypotensive and/or bradycardic effects [23].

The “pill-in-the-pocket” concept
In patients who arrive in an intensive care unit or even in critically ill patients who develop narrow QRS complex tachycardia, the “pill-in-the-pocket” concept may be of interest. This concept means single-dose administration of the drug only during an episode of tachycardia for the purpose of termination of the arrhythmia when vagal maneuvers alone are not effective. This approach may be considered for patients with infrequent episodes of AV nodal reentrant tachycardia that are prolonged but yet well tolerated, and obviates exposure of patients to chronic and unnecessary therapy between their rare arrhythmic events [24]. However, patients should be free of significant left ventricular dysfunction, sinus bradycardia, or preexcitation. It has been reported by Musto et al. [23] and Alboni et al. [24] that a single dose of flecainide (3 mg/kg) terminated acute episodes of AV nodal reentrant tachycardia. Single-dose therapy with diltiazem (120 mg) combined with propranolol (80 mg) was superior to both placebo and flecainide [20]. In addition, long-term follow-up of single-dose therapy with diltiazem plus propranolol was associated with a significant reduction in emergency room visits.

Ventricular tachyarrhythmias
One of the most important problems in intensive care, emergency medicine, and cardiology is to manage patients with recurrent ventricular tachycardia, ventricular flutter, or ventricular fibrillation [25]. Management of cardiac arrest due to life-threatening ventricular tachyarrhythmias is one of the most important goals in these patients to avoid serious problems and to avoid sudden cardiac death [26]. However, treatment of the underlying arrhythmia requires correct diagnosis; in most patients this is possible using the 12-lead surface electrocardiogram [27]. Nevertheless, it is necessary to understand in every patient why the arrhythmia was initiated and what the trigger of initiation was in any patient (Fig. 3).

Cardiac arrest
Patients who are successfully resuscitated from out-of-hospital cardiac arrest have a high rate of mortality after hospital discharge. Unfortunately, we are not able to recognize high-risk patients with subsequent cardiac arrest. Therefore, it is necessary to treat cardiac arrest patients in the emergency situation as well as possible and to develop algorithms for long-term therapy.

General considerations
Approximately 1000 people in the United States experience cardiac arrest each day, most often as a complication of acute myocardial infarction with accompanying ventricular fibrillation or unstable ventricular tachycardia (Fig. 4). In the year 2000, the American Heart Association 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 [28,29]. This publication presents the conclusions of the International Guidelines 2000 Conference on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. 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: (1) recognition of early warning signs, (2) activation of the emergency medical services system, (3) basic cardiopulmonary resuscitation, (4) defibrillation, (5) management of the airway and ventilation, and (6) intravenous administration of medications. It is impossible to review all guideline changes related to basic life support; this has been described in detail elsewhere [29]. However, it seems important to stress some important issues: change ventilation volumes and inspiratory times for mouth-to-mask or bag mask ventilation without oxygen ventilation should have a tidal volume of approximately 10 mL/kg (700 to 1000 mL) over 2 seconds; oxygen supplementation of a smaller tidal volume of 6 to 7 mL/kg (approximately 400 to 600 mL) may be delivered over 1 to 2 seconds. The compression rate for adult cardiopulmonary resuscitation should be approximately 100 per minute and the compression–ventilation ratio for one and two rescuers should be 15 compressions to 2 ventilations when the victim’s airway is unprotected (not intubated).

Neurologic outcome
There is general agreement that bystander first aid, defibrillation, and advanced life support are essential for neurologic outcome in patients after cardiac arrest. Bur et al. evaluated the effects of basic life support, time to first defibrillation, and emergency medical service arrival on neurologic outcome in 276 patients after cardiac arrest [30]. In contrast to intubation (odds ratio 1.08; 95% CI, 0.51–2.31; P = 0.84), basic life support (odds ratio 0.44; 95% CI 0.24–0.77; P = 0.004) and time to first defibrillation (odds ratio 1.08; 95% CI 1.03–1.13; P = 0.001) were significantly correlated with good neurologic outcome. In addition to the better neurologic outcome, among the patients who did not receive basic life support, the av-
Average cost per patient with good neurologic outcome significantly increased with the delay of the first defibrillation \( (P < 0.001) \) [30]. The importance of cerebral perfusion and pressure and cerebral tissue oxygen tension during cardiopulmonary resuscitation has been described recently [31].

**Early defibrillation**

Public access defibrillation, which places automatic external defibrillators (AED) in the hands of trained laypersons, has the potential to be the single greatest advance in the treatment of ventricular fibrillation since the development of cardiopulmonary resuscitation. Time to defibrillation is the most important determinant of survival from cardiac arrest [32]. The earlier the defibrillation is performed the better the success rates for resuscitation, irrespective of who is doing the first defibrillation [33]. In the last few years, there has been a significant increase in the use of AEDs in early defibrillation programs in a variety of settings, including hospitals, emergency medical service, police departments, casinos, airport terminals, and commercial aircraft, among others. In most of these settings, use of AEDs by basic life support ambulance providers or first responder in early defibrillation programs has been associated with a significant increase in survival rates [34–36].
World-wide experience

Automatic external defibrillators were used in 105 patients with ventricular fibrillation occurring in casinos [34]. Fifty-six of the patients (53%) survived to discharge from the hospital. Among the 90 patients whose collapse was witnessed (86%), the clinically relevant time intervals were a mean of 3.5 ± 2.9 minutes from collapse to the delivery of the first defibrillation shock, and 9.8 ± 4.3 minutes from collapse to the arrival of the paramedics. The survival rate was 74% for those who received their first defibrillation no later than 3 minutes after a witnessed collapse and 49% for those who received their first defibrillation after more than 3 minutes. AEDs were used in a US airline study in 200 patients, including 99 patients with documented loss of consciousness. The administration of shock was advised in all 14 patients with documented ventricular fibrillation, and no shock was advised in the remaining patients. The first shock successfully defibrillated the heart in 13 patients, and defibrillation was withheld in one patient at the family’s request [35]. Recently, Caffrey et al. reported the public use of AEDs in three Chicago airports [36•].

Defibrillation in hospitals and intensive care units

Hospitals need to pay attention to the timeliness of defibrillation including monitored and unmonitored ward settings, intensive care units, and ambulatory care facilities. There is a prevalent misconception that hospitals inherently provide rapid defibrillation followed by prompt advanced life support. In addition, many hospitals both in the United States and in Europe have been
slow to accept and implement the readily available technology that allows defibrillation to be performed before arrival of a physician. Until recently, hospitals and intensive care units have been staunchly reluctant to evaluate critically their process for providing early defibrillation [37]. It is well known from previous studies that delays of 5 to 10 minutes can be expected before the conventional in-hospital code teams deliver the first shock to patients with ventricular fibrillation. It has been shown that nurses in the hospital setting can be easily trained to use an AED and to safely and effectively operate the device.

**Role of antiarrhythmic drugs in intensive care and emergencies**

Prompt cardiopulmonary resuscitation and early defibrillation either by DC-countershock or an AED significantly improve the likelihood of successful resuscitation from ventricular fibrillation [38]. Despite these circumstances, there is still a place for antiarrhythmic drugs to treat patients with life-threatening ventricular tachyarhythmias such as monomorphic or polymorphic ventricular tachycardia in intensive care or emergencies [4•]. The American Heart Association has clearly defined when and how to use these agents [29]. Drugs such as lidocaine, procainamide, sotalol, amiodarone, or magnesium were recommended for treatment of ventricular tachyarrhythmias in intensive care or emergencies.

**Class I drugs**

In summary, there is still a place for lidocaine in the treatment algorithm for stable, monomorphic, or polymorphic ventricular tachycardia, and lidocaine is acceptable for these arrhythmias with normal or impaired cardiac function. In contrast, there is little evidence supporting a benefit from treatment with lidocaine for cardiac arrest. Lidocaine seems to be more effective during acute myocardial infarction. However, the US guidelines postulated that lidocaine is a second-tier choice and other drugs are preferred over lidocaine in each ventricular tachycardia scenario. In addition, the prophylactic use of lidocaine is not recommended. Use of procainamide is indicated in patients with ventricular tachycardia and is an alternative in pulseless ventricular tachycardia/ventricular fibrillation. Its utility in pulseless cardiac arrest is less well studied and limited by the need for a relatively slow infusion (maximum rate 50 mg/min) and the achievement of potentially toxic concentrations if administered more rapidly. Procainamide should be avoided in patients with preexisting QT prolongation and torsade de pointes tachycardia. The electrocardiogram and blood pressure must be monitored continuously during procainamide administration. Precipitous hypotension may occur if the drug is injected too rapidly [29]. Propafenone and flecainide are class I antiarrhythmic drugs with significant conduction-slowing and negative inotropic effects. In addition, propafenone has nonselective beta-blocking properties. Intravenous propafenone is used for the same indication as flecainide and is acceptable for treatment of both supraventricular and ventricular tachycardia. Because of significant negative inotropic effects, propafenone and flecainide should be not given to patients with impaired left ventricular function. In addition, both propafenone and flecainide should be avoided when coronary artery disease is suspected.

**Class III drugs**

Sotalol prolongs action potential duration and increases cardiac tissue refractoriness. In addition, it has nonselective beta-blocking properties. Sotalol is effective in both supraventricular and ventricular tachycardia. Side effects include bradycardia, hypotension, and torsade de pointes tachycardia. Intravenous sotalol in intensive care medicine is limited by its need to be infused relatively slowly. This may be impractical and has uncertain efficacy in emergent circumstances, particularly under compromised circulatory conditions [29]. 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 tachyarhythmias [39]. 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 with placebo (34%) (P = 0.03) [40]. The role of amiodarone as an emergency drug has been reported recently by Taylor [39].

**Magnesium**

Severe magnesium deficiency is associated with cardiac arrhythmias, symptoms of heart failure, and sudden cardiac death. Hypomagnesemia can precipitate refractory ventricular fibrillation and can hinder the replenishment of intracellular potassium. Magnesium deficiency should be corrected in intensive care and emergencies if present. Magnesium is not usually categorized as an antiarrhythmic agent. However, it has been known for a long time that in emergent circumstances, magnesium sulfate 1 to 2 g is helpful to suppress life-threatening ventricular tachyarrhythmias and should be administered over 1 to 2 minutes [4•]. The role of magnesium in intensive care and emergency medicine was described by Kaye and O’Sullivan in 2002 [41]. These authors concluded that magnesium should be the first-line therapy in eclampsia and torsade de pointes tachycardia. Data from other studies show that magnesium has a clearly defined role as a second-line therapy in acute severe bronchial asthma.
Clinical implications

Atrial fibrillation, ventricular tachycardia, and patients after cardiac arrest due to ventricular fibrillation are “classical” patients in intensive care units and emergencies. It has been shown that evaluation of history, physical examination, and the 12-lead-surface electrocardiogram are essentials for correct diagnosis and treatment. In all patients with cardiac arrhythmias in intensive care and emergencies who have unstable hemodynamics, external DC cardioversion is the method of choice. In patients with atrial fibrillation, there are numerous studies that analyzed outcome and recurrences after antiarrhythmic drug treatment. However, recently published studies showed that rhythm control is not superior to rate control and, therefore, a new strategy seems to be advisable to treat these patients. Nevertheless, there is general agreement that rate control is necessary in patients with atrial fibrillation and rapid ventricular response. For this indication, several well-known agents have long been available. Treatment of recurrent ventricular tachycardia, ventricular flutter, or cardiac arrest due to ventricular fibrillation is one of the most important problems in intensive care and emergencies. A fascinating idea to treat cardiac arrest caused by ventricular fibrillation is the automatic external defibrillation concept. First results of different studies are very promising.

Conclusion

Emergency medicine and critical care are fields that often require rapid diagnosis and intervention for specific situations. It is well known that in all patients with tachyarrhythmias, evaluation of the underlying etiology and the degree of left ventricular function (dysfunction) is essential. Correct treatment of arrhythmias in the intensive care patient is based on an understanding of the mechanism that caused the situation. The therapeutic role of antiarrhythmic drugs in the management of atrial fibrillation or cardiac arrest is debatable. After the AFFIRM and RACE studies, the restoration of sinus rhythm is not the goal in any patient, and rate control is an alternative way for these patients. The prophylactic administration of antiarrhythmic drugs has never been studied formally in victims of cardiac arrest. The optimal dose of antiarrhythmic drugs, whether best given before or only after multiple defibrillation shocks have failed to restore circulation, is not yet known.

References and recommended reading

Papers of particular interest, published within the period of review, have been highlighted as:

- Of special interest
- Of outstanding interest


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