The Arrhythmic Patient in the Emergency Department

A Practical Guide for Cardiologists and Emergency Physicians

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Editors
Massimo Zecchin
Cardiovascular Department
Cattinara University Hospital
Trieste
Italy

Gianfranco Sinagra
Cardiovascular Department
Cattinara University Hospital
Trieste
Italy

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Foreword

Treatment of patients with cardiovascular diseases has dramatically changed over the past 20 years. Accompanied by an incredible increase in pathophysiological understanding and availability of treatment options, specialized fields of expertise have rapidly evolved.

Electrophysiology is one of these newcomers. Based on the analysis of basic principles of electrical activation in the human heart, the field has developed into sophisticated treatment strategies of device therapy and catheter ablation. The majorities of today's EP patients originate from the mainstream of everyday clinical cardiology and present with endemic, bradycardic, and tachycardic arrhythmias.

With this background, electrophysiology has also arrived in the ER department.

Now it is our obligation to transport our knowledge and expertise for treatment of arrhythmia patients to our cardiology colleagues and specialized ER physicians who encounter emergency situations due to or accompanied by cardiac arrhythmias in a significant number of patients, next to a variety of other medical emergencies.

Dedicated literature on that topic is really scarce. I therefore want to thank the editors and authors of this book to take the challenge, efforts, and work and to bring together a vast amount of EP knowledge and to focus it to the special situation in the ER.

This book should become an integral part of training for young cardiology fellows, and it will be a practical guide and help for all medical staff involved into the management of ER patients.

Christopher Piorkowski
Head of EP Department – University of Dresden

Preface

The book is a practical guide designed for physicians (both emergency physicians and cardiologists) who first evaluate and treat patients with arrhythmias or potentially arrhythmic problems in the emergency setting. It can also be a useful learning tool for students and residents in Cardiology and Emergency Medicine.

In all chapters, every effort was made to provide a brief but comprehensive summary of the topic with both theoretical and practical suggestions, considering the different needs of the specialists involved in the primary care of arrhythmic patients.

The diagnostic pathways and treatment options of patients presenting in the Emergency Department with syncope or arrhythmias, including bradyarrhythmias, atrial fibrillation, and narrow and wide QRS tachycardias, are discussed. In addition, clear advice for the management of patients with cardiac devices and possible dysfunction, electrical storm, or a requirement for urgent surgery are provided.

Practical suggestions are offered for short-term management, e.g., regarding the decision on when to hospitalize the patient and some hints for long-term pharmacological and non-pharmacological treatment.

In the first chapter, an overview of the management of arrhythmic patients, from the emergency physician's point of view, is provided. In the second chapter, some considerations, beyond published guidelines, for the management of syncope are given by a leading expert. An extensive theoretical overview of brady- and tachyarrhythmias are then followed by practical flowcharts in Chaps. 3, 4 and 5, while in the following chapter the differential diagnosis of wide-QRS tachycardias with clear examples are discussed by one of the greatest experts in this field. Chapters 7 and 8 deal with quite rare cardiac conditions, sometimes not so known by emergency physicians and even by cardiologists, who nonetheless in such cases sometimes face difficult decisions. Differently, situations frequently observed in the Emergency Department, but with an arrhythmogenic potential which is not always well defined, are presented in Chaps. 9 and 10. Finally, in the last three chapters, some indications for the management of patients with implanted cardiac devices presenting in Emergency Department or who need urgent surgery are provided, again considering the different skills of the various medical figures involved in the primary care of such patients.

Considering the heterogeneity of the topics, some differences in the chapters' frameworks were necessary. However, the book was conceived to offer quick information and solutions to the single issues, as required in the emergency setting, rather than providing a systematic review.

Trieste, Italy Massimo Zecchin
Gianfranco Sinagra

Contents

Management of Arrhythmic Patients in the Emergency Department: General Principles

1

Alessandro Surian and Luca Visintin

Arrhythmic patients are common in the duty of the emergency physician and the cardiology consultant. Both specialists, with their different approach and method, well know the clinical and statistical relevance of arrhythmias.

A wide range of symptoms leading the patient to the emergency department may be related to a cardiac rhythm disorder. They may vary from simple palpitations to a cardiac arrest. Otherwise, the diagnosis of arrhythmia can be made in patients who came to the ED for other diseases.

Emergency physician is the first doctor approaching the patient and must initially define the hemodynamic state induced by the arrhythmia. The need to assure hemodynamic stability must be assumed as the first important target and should not be delayed by any other consideration.

Once a stabilization is obtained, the consultant cardiologist can improve diagnostic and therapeutic definition. A tight cooperation between the two specialists warrants the best patient outcome.

1.1 Triage

Triage of arrhythmic patient must focus on hemodynamic state and time of onset of symptoms and signs. Unstable arrhythmias should be admitted as soon as possible to physician evaluation, while asymptomatic patients may wait longer.

A. Surian (✉) • L. Visintin
Emergency Medicine Unit, Cardiovascular Department,
Cattinara University Hospital, Trieste, Italy
e-mail: dottalesurian@yahoo.it

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Hemodynamic criteria for quick admission are:

- SBP (systolic blood pressure): <90 mmHg
- DBP (diastolic blood pressure): <60 mmHg
- Heart rate per minute: >120 and <50 bpm
- Respiratory rate per minute: >30 and <10
- Body temperature: >39.0 °C and <36.0 °C
- Sat O_2: <90 %

Moreover, chest pain, dyspnea, acute heart failure, acute altered mental status, and signs of shock are evaluated during triage.

Unstable patients should be addressed immediately to the shock room of the ED, while stable patient can wait or be addressed to an examination room provided with ECG for a first evaluation and subsequently be treated when the adequate setting is available [1, 2].

1.2 Emergency Department Physician Approach to the Arrhythmic Patient

Emergency physician's main task is to identify "*hemodynamically unstable*" patients, quickly evaluating parameters like level of consciousness, ventilation, oxygenation, heart rate, and blood pressure (Table 1.1). Clinical evaluation is focused on the investigation of signs of shock (altered mental status, cool and clammy skin, weak and rapid pulse, rapid and shallow breathing, anxiety, lightheadedness, chest pain, decrease of urine, thirst and dry mouth, hypoglycemia, confusion, nausea, lackluster eyes) dyspnea and tachypnea, or oxygen desaturation.

Patient's ECG, blood pressure, and O_2 saturation (sat O_2) should be immediately put under continuous monitoring and intravenous line with blood samples provided. Airways have to be kept patent, breathing assisted, and oxygen given if sat O_2 is below 94 %. A 12-lead ECG should be obtained as soon as possible for a correct diagnostic evaluation of the arrhythmia. Medical history must be gathered.

Table 1.1 First steps

Look for:
shock signs
Chest pain
Respiratory distress
Do:
Monitor the patient, IV line, blood samples
Ensure patent airway and ventilation and give oxygen (if needed)
Support perfusion pressure (use mean arterial pressure as a good index)
12-leads ECG; gather medical history
Treat reversible causes

Should a cardiac arrest occure, advanced life support protocols have to be applied.

Hemodynamic instability, defined as an acute organ failure or a near-cardiac arrest situation, may be due to tachy- or bradyarrhythmia.

In the event of a tachyarrhythmia, an immediate defibrillation or synchronized cardioversion should be done regardless of the arrhythmia mechanism.

In addition, bradyarrhythmias may lead to a severe decrease in cardiac output, causing hemodynamic instability with hypotension, mental dizziness, decreased consciousness level, cyanosis, dyspnea, etc.

A treatment based on atropine, catecholamine, or an electrical stimulation may be helpful or even lifesaver [1, 2].

1.2.1 Tachyarrhythmia

By definition, tachycardia is a heart rate exceeding 100 beats per minute.

By far the most common tachycardia diagnosed in the emergency department is sinus tachycardia.

In the healthy patient, it is a physiological response to physical stress or anxiety. Sinus tachycardia is also a normal condition during the pregnancy. In most other cases, it is due to an underlying pathological condition (e.g., fever, dehydration, anemia and hypoxia, ACS, P.E., hyperthyroidism, high blood pressure, smoking, alcohol, beverages containing caffeine, medication side effects, abuse of recreational drugs, such as cocaine, or imbalance of electrolytes) [3].

"Appropriate" sinus tachycardia offsets an underlying condition, while "inappropriate" sinus tachycardia can be a consequence of deficit of vagal tonus or a hyperactivity of/excessive sensibility to the sympathetic nervous system. During sinus tachycardia heart rate is usually lower than 140–150 bpm, even if, in young people under extreme stimulation, it can exceed 220 bpm. Typically, in sinus tachycardia, the P wave is positive in inferior and lateral leads (as in sinus rhythm). As sympathetic activation increases AV conduction, PR interval is shorter than in sinus rhythm; therefore, with few exceptions, the coexistence of long PR and sinus tachycardia is unlikely, even in patients with I degree AV block during normal sinus rhythm, and usually suggests other mechanisms of tachycardia, as atrial tachycardia or atrial flutter, possibly with 2:1 conduction and a P wave hidden within the QRS complex.

In order to identify if the tachycardia is the main cause of the patient's symptoms, a complete physical examination, blood draw to test metabolic and renal function, emogasanalysis (EGA), 12-lead ECG results and medical history should be performed and any potential reversible causes should be corrected.

Usually tachycardia may be considered as hemodynamically significant when they exceed 150 bpm.

However, it is important to remark that even frequencies lower than 150 bpm may cause hemodynamic compromise, mainly if it is sustained for a prolonged time and/or coexists with an underlying heart disease, leading to chest pain, altered mental status, pulmonary edema, or cardiogenic shock, requiring an emergency electrical cardioversion.

It is advised to perform an effective pre-procedural sedation if the patient is conscious, although hemodynamically unstable.

1.2.1.1 Procedural Sedation/Anesthesia During Cardioversion

Sedative or dissociative drugs, coupled with or analgesics, are used to relieve the patient from unpleasant procedures. Many of these drugs can lead to central nervous system and cardiac and/or respiratory depression. Given the potential risks, regulatory agencies are debating about the medical privileges needed to perform this procedure, particularly about the presence of an anesthesiologist during the procedure.

Recommendations for a safe employ consist of a proper setting (ECG, respiratory rate, sat O2, NIBP monitoring, advance life support trained personnel, devices for life support) and frequent reevaluations (prior to, during, and after procedure); trained staff should choose appropriate drugs and dosing depending on the distinctive features of each patient.

A growing literature highlights the safety of administration of ketamine, midazolam, fentanyl, propofol, and etomidate in the ED [4–6].

- *Equipment and supplies*: oxygen, suction, reversal agents, advanced life support medications and equipment, defibrillator, and CO_2 capnography. An IV line should be set; reversal agents should be available whenever opioids and benzodiazepines are administered.
- *Personnel*: during the procedure, personnel dedicated to patient monitoring should focus only to the sedation and not to other tasks.
- *Training*: the physician should know drug's pharmacology of the agents used and their antagonists. Personnel with experience in Advanced Cardiac Life Support should be present.
- *Drugs*. Electrical cardioversion is a brief but painful procedure. Light sedation is inadequate for a pain-free relaxed patient. Therefore, a moderate to deep sedation and analgesia or general anesthesia is required. In most US and Europe hospitals, emergency physicians are not allowed to provide general anesthesia, so sedation can be the only option if an anesthesiologist is not present. Drugs: midazolam and fentanyl are commonly used, but their long-lasting effects make them not handy or straight dangerous. Instead, for a brief and titratable deep sedation, it makes more sense to employ propofol, etomidate, or methohexital combined with fentanyl. There are contradictory statements from the American Society of Anesthesiologists (ASA) guidelines about authorization for propofol use by emergency physicians. Evidence is accumulating that non-anesthesiologist-administered propofol sedation has a safety and efficacy profile comparable or superior to that provided by benzodiazepines with or without opioids. Medications should be administered gradually, allowing sufficient time between dose and effect assessment. Concurrent administration of sedative and analgesic drugs requires evaluation on dose reduction.
- *Recovery*: observation should be prolonged until there are no more risks for cardiorespiratory depression. Medical institution should set up appropriate discharge criteria [4–6].

1.2.1.2 Cardioversion/Defibrillation

If cardioversion is chosen, set the defibrillator into the synchronized mode. This to avoid shock delivery during ventricular "electric vulnerability" period (apex and descending branch of T wave), a potential trigger of ventricular fibrillation. Defibrillation, used for interruption of pulseless VT, VF, and torsade de pointes synchronization, should be avoided, as QRS complexes may not be identified.

Emergency physician should be trained to recognize the presence of the P wave and distinguish between narrow-complex (supraventricular) tachycardia and wide complex tachycardia, which in condition of urgency should be considered and treated as ventricular tachycardia.

The different types of tachycardia can be treated with different energies:

- As recommended by international guidelines for regular narrow-complex tachycardia, the initial energy cardioversion should be 50–100 J with biphasic defibrillators and 200 J if monophasic (Class IIa, LOE B).
- For irregular narrow-complex tachycardia, the recommended initial biphasic energy is 120–200 J (Class IIa; LOE A).
- Regular wide complex tachycardia may resolve after discharge at 100 J by both biphasic and monophasic defibrillators (Class IIb, LOE C).

Anyway, if the first shock is inadequate to resolve the arrhythmia, increase energy "in a stepwise fashion."

When using monophasic defibrillators, initial energy should be set to 200 J, proceeding in a stepwise fashion in the event of failure.

The irregular wide complex tachycardia should be treated with high-energy unsynchronized shock (i.e., defibrillation), because of the difficulty of the machine to distinguish between the QRS complex and T wave.

Even if there were doubts whether the tachycardia is monomorphic or polymorphic, the shock should not be delayed and a high-energy unsynchronized shock must be delivered.

In the unstable patient (if not hypotensive) presenting with a regular narrow QRS complex tachycardia, *adenosine* is safe to be used while cardiac electrical cardioversion is being set up both for therapeutic (in case of tachycardia involving the AV node as a part of the reentry circuit) and diagnostic (in case of atrial arrhythmias, unmasking atrial activity slowing AV conduction (Class IIb (LOE C)).

If the patient with tachycardia is stable, the emergency physician will have more time for a correct diagnosis and to choose the most appropriate therapy, with the help of a cardiologist if necessary.

After obtaining a complete medical history and a careful physical examination, QRS complex evaluation is needed. QRS duration should be measured in at least two orthogonal derivations: narrow-complex tachycardia (QRS duration <120 ms) should be always considered, by definition, as supraventricular: examples are sinus tachycardia, atrial fibrillation (AF), atrial flutter, AV nodal reentrant tachyarrhythmia (AVNRT), tachyarrhythmia mediated by accessory pathways, atrial tachycardia, multifocal atrial tachycardia (MAT), and junctional tachycardia (rare in adults) .

Based on ECG findings, the regularity of RR intervals and the relationship between P waves and QRS complexes along with the timing of onset of tachycardia may help to differentiate among the various kinds of supraventricular tachyarrhythmia.

If the anamnesis highlights sudden onset of palpitations and its rapid resolution, it is likely to be atrial fibrillation, atrial flutter, AVNRT, atrioventricular reciprocating tachycardia, and atrial tachycardia. Instead, sinus tachycardia, permanent atrial fibrillation, and permanent flutter, together with MAT and premature atrial contractions, show symptoms that arise and resolved more gradually [7].

P waves immediately preceding the QRS complexes address the ED physician's diagnosis to sinus tachycardia, atrial tachycardia, multifocal atrial tachycardia or multiple atrial premature contractions.

P waves following QRS complexes suggest atrioventricular nodal reentrant tachycardia, atrioventricular reciprocating tachycardia, or atrial tachycardia. However, heart rate can be high enough to have T waves overlapping the P waves.

If tachycardia has a narrow QRS complex, vagal maneuvers and, if ineffective, the administration of adenosine at doses of 6–12 mg, (always under cardiac monitoring) may have a dual purpose:

- *Diagnostic*, since the increase of degree of AV block can unmask the nature of the underlying rhythm; a transient slowing of ventricular rate may highlight atrial fibrillation, atrial flutter, and sinus tachycardia, while there might not be any effect on multifocal atrial tachycardia or frequent atrial premature contractions.
- *Therapeutic*, because the increase in parasympathetic tone may slow electrical conduction through the AV node interrupting reentrant arrhythmias involving tissues sensitive to vagal stimulation (AV nodal reentrant tachycardia, AV reciprocating tachycardia, and sometimes atrial tachycardia).

If vagal maneuvers and adenosine are unsuccessful in converting to sinus rhythm or atrial fibrillation and atrial flutter are diagnosed, it is recommended to administer:

- *Diltiazem* (15–20 mg or 0.25 mg/kg IV over 2 min); if needed, after 15 min an additional IV dose of 20–25 mg (0.35 mg/kg) can be administered; the infusion dose is 5–15 mg/h, titrated according to heart rate.
- *Verapamil* (2.5–5 mg IV bolus over 2 min); if no response and non-drug-induced adverse events occur, it is possible to repeat doses of 5–10 mg every 15–30 min up to a total dose of 20 mg.
- *Beta-blockers* (metoprolol, atenolol, propranolol, esmolol, and labetalol).

These drugs are able to convert the reentrant tachycardia by acting on the nodal tissue or slowing the ventricular response in case of other supraventricular arrhythmias [1].

In patients with atrial fibrillation/flutter/tachycardia lasting more than 48 h (or if the onset of the arrhythmia is unknown), electrical or pharmacological cardioversion should not be attempted in absence of adequate anticoagulation in the

preceding 3 weeks. Otherwise, when prompt restoring of sinus rhythm is needed or preferred, cardioversion can be done after excluding the presence of thrombi in the left atrium by transesophageal echocardiography (Class IIa, LOE B) [8].

In most cases the patient with narrow QRS tachycardia is treated in ED, restoring the RS or starting a therapy aimed to control the heart rate, and resigned to be entrusted to the outpatient cardiologist, who will complete the diagnostic process and improve, if necessary, the treatment started in the emergency department.

Handling of wide-QRS complex tachycardia (>120 ms) is different. These tachycardias cannot be treated in PS only but require both an initial cardiac evaluation in the emergency department, including a careful analysis of the ECG and echocardiography, and hospitalization in the specialist department.

1.2.1.3 When should the Cardiology Consultant be called?

Cardiologist called in ED for a patient with a wide-QRS complex tachycardia has a daunting task. In fact, a mistaken diagnostic may lead to disastrous effects in terms of prognosis.

A wide-complex tachycardia can be:

- Ventricular tachycardia.
- Supraventricular tachycardia in a patient with preexisting bundle branch block.
- Tachycardia-dependent bundle branch block (aberrancy).
- Tachycardia caused by drug that have a widening effect on QRS.
- Atrial arrhythmias in the presence of ventricular pre-excitation.

In the diagnostic path of a wide-QRS complex tachycardia, attention must be paid to the clinical examination (variability of the first tone and amplitude variable radial pulse lay for the presence of AV dissociation) and the careful analysis of the ECG [9].

Here is a summary of some general criteria that can help the cardiologist identify the origin of tachycardia (for detailed discussion of the ECG, see Chap. 6):

A. Search if the electrical activity of the atria is present; P waves, independent of QRS, are separated by constant intervals, paying more attention in derivation II and V1, where these waves can be easier to find.

   If some ventricular impulses are not conducted to the atria and the QRS/P ratio is greater than 1, a diagnosis of ventricular tachycardia can be made.

   Small deflections, fitting in a rhythmic manner inside the QRS complexes, suggest the presence of underlying sinus P waves when their rate is lower than the ventricular rate. Hence, a diagnosis of ventriculoatrial dissociation and therefore of ventricular tachycardia can be made. If there is a mathematical relationship between ventricular and atrial electrical activity, a retrograde ventricular-atrial conduction is likely, as it can be found in about 50 % of cases..

   In presence of a clear QRS/P ratio = 1, the diagnosis may be more difficult; it may be expression of atrial tachycardia, sinus rhythm with aberrant conduction, nodal reentrant tachycardia, automatic junctional tachycardia, reciprocating orthodromic tachycardia with aberrant conduction, or ventricular tachycardia with 1:1 retrograde conduction.

B. Search for "concordance" aspect of QRS in the precordial leads. The presence of concordance suggests that the tachycardia has a ventricular origin. Common definitions are "*positive concordance*" if the QRS complex is "R wavelike" from V1 to V6 and "*negative concordance*" in the presence of a "QS-like" morphology from V1 to V6.

   Cardiologists must remember that although a negative concordance is absolutely specific for VT, the positive could, in rare cases, be expression of a pre-excited tachycardia due to a left posterior Kent bundle (pre-excited tachycardia with conduction through ancillary pathway).

C. As stated by Brugada et al., in the diagnostic algorithm of regular wide-QRS complex tachycardia, the presence of RS complexes (R waves followed by S wave) in precordial leads suggests a diagnosis of VT when the interval between the beginning of the R wave and nadir of the S wave is >100 ms [10].

D. The analysis of QRS complexes, in particular in leads V1 and V6, is certainly useful.

In a wide-QRS complex tachycardia with right bundle branch block (positive QRS in V1), morphologies R, Rs, RrĐ, qR in V1 and QS, qR, rS in V6 are suggestive of ventricular tachycardia.

A three-phasic morphology of V1 (rsR' and rSR'), biphasic morphology in V1 (rRĐ), or three-phasic morphologies in V6 (qRs) suggest a supraventricular genesis with aberrant conduction.

In wide-QRS complex tachycardia with left bundle branch block (negative QRS in V1), an initial R wave >30 ms in V1, an interval between the beginning of the QRs complex and nadir of the S wave >60 ms, the presence of a notch in the descending limb of the S wave, and Q wave in V6 (qR aspects, QRS or QS) suggest a ventricular origin of arrhythmia.

Initial R wave <30 ms and an interval between the QRS onset and the S wave nadir <60 ms are suggestive of supraventricular tachycardia with aberrant conduction.

The maneuvers of vagal stimulation can be useful in the diagnosis of wide complex tachycardia, and depending on the response, we can obtain important information:

- If the tachycardia ceases, a supraventricular reentrant tachycardia is likely (but in some cases even the idiopathic ventricular tachycardia is resolved with the vagal stimulation).
- Modifications of the atrioventricular conduction in atrial tachycardia and atrial flutter can be observed.
- In ventricular tachycardia with ventriculoatrial (VA) 1:1 conduction, a variation of VA interval or transient second-degree retrograde VA block may be recorded.

1.2.1.4 Treatment of a Wide-QRS Complex Tachycardia

As mentioned earlier, if the patient gets worse and becomes unstable, staff must be ready to perform an *immediate electrical cardioversion* or to deliver high-energy unsynchronized shock, if ventricular fibrillation emerges or instability is caused by a polymorphic VT.

When diagnostic doubts about the origin of tachycardia are present, it should be treated as if it were of ventricular origin.

In presence of regular and monomorphic complexes, it is reasonable to administer *adenosine*, considered safe, and useful for both diagnosis and treatment purposes (Class IIb, LOE B). Adenosine should not be administered if the patient is unstable or has irregular or polymorphic complexes: in this condition, it could lead to degeneration in VF (Class III, LOE C).

Once diagnosed a ventricular tachycardia, treatment consists of antiarrhythmic drugs such as *procainamide* (Class IIa, LOE B), *amiodarone* (Class IIb, LOE B), or *sotalol* (Class IIb, LOE B) or *electrical cardioversion*.

In patients with known long QT during sinus rhythm, procainamide and sotalol should be avoided.

Procainamide is administered in the initial dose of 10 mg/kg, at rate of 20–50 mg/min. Maximum dose is 17 mg/kg. Maintenance infusion is 1–4 mg/min.

Amiodarone is given 150 mg IV over 10 min; dosing should be repeated to a maximum dose of 2.2 g IV for 24 h.

If an antiarrhythmic drug was already administered without success, it is advisable not to use a second drug without a cardiologist consult (Class III, LOE B) or proceed with electrical cardioversion (Class IIa, LOE C).

Lidocaine is now considered a drug of second choice for the treatment of ventricular tachycardia (dose: 1–1.5 mg/kg IV bolus). Maintenance infusion is 1–4 mg/kg (30–50 mcg/kg per min).

If the wide-QRS complex tachycardia is irregular, the underlying rhythm is likely to be an atrial fibrillation with aberrant conduction. In this case some considerations about the best treatment (rate control or rhythm control) are necessary, in particular:

- Avoid cardioversion if the arrhythmia has been present for more than 48 h (and the patient is stable enough). Consider treatment options with the consultant cardiologist, in particular transesophageal echocardiography to exclude the presence of a thrombus in the left atrium.
- Administer IV *heparin* before cardioversion if not contraindicated.

Irregular polymorphic tachycardia needs an immediate defibrillation. Drugs that may prolong the QT interval should be withdrawn and serum electrolytes corrected.

Myocardial ischemia is the most common cause of polymorphic VT in absence of a prolonged QT interval. In this circumstance amiodarone and sotalol are able to reduce the recurrence of the arrhythmia (Class IIb, LOE C).

1.2.2 Bradycardia

A heart rate below 60 bpm is usually defined as bradycardia. While in young healthy subjects and particularly in athletes it can be a common and non-suspect remark, it can conceal various kinds of diseases.

Usual symptoms of bradycardia are asthenia, fatigue, dyspnea, chest discomfort or pain, pre- or complete loss of consciousness, light-headedness, and decreased level of

consciousness. Signs often noticeable are hypotension and/or orthostatic hypotension, diaphoresis, bradycardia-related (escape) frequent premature ventricular complexes, or other ventricular tachyarrhythmias. All the signs and symptoms are due to the discrepancy between the low heart rate and the metabolic requests of the organism.

Usually symptoms are relevant when lower than 40 bpm or higher in presence of a pre- or coexistent cardiac disease [1, 2, 11–13].

First approach: whatever is the underlying cause, ED physician must define the hemodynamic compensation. If low heart rate is the cause of the symptoms, the patient should be immediately treated with drugs and percutaneous or transvenous pacing. The consultant cardiologist should be called to provide support to the diagnosis and treatment.

If the bradycardia is asymptomatic or the hemodynamic condition is acceptable, the thorough diagnosis can be ruled out with more smoothness.

As soon as possible, a 12-lead ECG should be obtained, with a long stripe in II or V1, to unmask atrial activity, for example, the presence of not-detected 2:1 AV block. A complete physical examination and blood tests for troponin, drugs, electrolytes, and serum creatinine must be performed in the meanwhile. If available, an echocardiogram should be used. Chest X-rays or thoracic echography can help to clear up pulmonary edema or congestive heart failure [1, 2, 11, 12].

Based on the ECG findings, we can discern the following rhythms:

1.2.3 Sinus Bradycardia

It can be a sign of underlying pathologies (e.g., vagal hypertonia, drug effect, hypoxia, ischemia of sinoatrial node due to occlusion of right coronary arteria, etc.).

ECG shows a regular sinus rhythm with heart rate lower than 60 bpm and a constant 1:1 AV conduction with PR interval of 120–200 ms (in the absence of coexistent AV block); P waves are regular, with identical waveform, axis between 0 and 90°.

Symptoms can be absent at rest and may appear only during effort.

Common causes are listed in Table 1.2.

Table 1.2 Common causes of sinus bradycardia

Vagal stimulus	Vomit, abdominal pain (i.e., acute retention of urine, acute abdomen, aortic aneurysm), Valsalva maneuver, carotid sinus hypersensitivity
Drugs	β-blockers, Ca^{++} channel blockers, ivabradine, digoxin, amiodarone, quinidine, and virtually all the antiarrhythmic drugs
Hyperkalemia	Acute or chronic heart failure, ACE-I or K+ savers drugs
Hypothermia	
Hypothyroidism	Autoimmune diseases, inappropriate levothyroxine dosage in known hypothyroidism
Endocranial hypertension	Acute endocranial hemorrhage
Sinoatrial node hypoperfusion	Right coronary ischemia
Sinoatrial sick syndrome	

It is clear from the analysis of the abovementioned causes how crucial it is to find the underlying etiology of the sinus bradycardia [14].

1.2.4 Pitfalls

Not maintaining a high and broad index of suspicion for underlying causes

1.2.5 Low-Rate Atrial Fibrillation

It is characterized by the absence of recognizable P wave, irregular RR intervals, and narrow or wide QRS complexes depending on the previous history of the patient.

Most common causes are drugs (as most antiarrhythmic drugs, digoxin, β-blockers, Ca^{++} antagonists), especially in older patients with reduced renal and/or liver function, vagal hypertonia (mainly in young subjects), or atrioventricular (AV) block. The presence of complete AV block ventricular rate is regular, because of junctional (usually at about 35 bpm) or infrahisian ventricular escape (<30 bpm).

Treatment of symptomatic extreme bradycardia consists in drugs (amines) or transcutaneous or intracardiac transvenous pacing to reach hemodynamic stability.

In elderly patients the most bradyarrhythmias are drug related and will wear off as the involved drugs (e.g., digoxin, β -blocker) wash out; in some cases consider starting with atropine followed by amines [7].

1.2.6 Sinus Node Dysfunction: Sick Sinus Syndrome

Sick sinus syndrome is a condition characterized by a wide spectrum of rhythm disturbances: bradycardia, sinusal arrest, paroxysmal atrial tachycardia, and bradycardia/asystole.

Clinical appearance ranges from asthenia, mental dizziness to syncope, vertigo, and cardiac failure.

Common causes are idiopathic degeneration of sinoatrial node and/or the atrial conduction tissue, right coronary ischemia, and flogistic and infiltrative diseases. More often drugs can be blamed, as beta-blockers, digoxin, Class I and III antiarrhythmic agents, and Ca++ channel blockers.

Tricyclic antidepressant, 4-phosphodyesterase inhibitors and Beta stimulant may induce atrial tachyarrhythmia.

Diagnosis can be reached by anamnesis, ECG, dynamic ECG (Holter, loop recorder), and electrophysiological study (endocavitary or transesophageal).

Depending on the prevalence of tachy- or bradycardia and the underlying cardiac disease, the therapy can vary from drugs to definitive pacing.

Indications for pacemaker implant are symptoms related to bradycardia.

1.2.7 Atrioventricular (AV) Blocks

AV blocks are commonly caused by:

- Lesions of the electrical conduction system of the heart (necrosis, fibrosis, sclerosis)
- Vagal hypertonia (inferior acute myocardial infarction, hypersensitivity of carotidal sinus, vagal maneuvers, abdominal pain, etc.)
- Increase of the refractory period (drugs)

Based on clinical and ECG findings, AV blocks are divided into:

- First-degree AV block (*prolonged* AV conduction *without any AV interruption*)
- Second-degree AV block (*intermittent* AV conduction)
- Third-degree (or complete) AV block (*complete* interruption of AV conduction)

1.2.8 First-Degree AV Block

PR interval is >200 ms with all P waves conducted to the ventricle; it is most commonly iatrogenic in patients treated with β- and Ca + channel blockers and digoxin or can be secondary to vagal hypertonia; less often the cause is an acute coronary syndrome of the right coronary artery involving AV node [15].

1.2.9 Second-Degree AV Block Type I (Wenckebach: Mobitz I)

There is a progressive increase of the PR interval, until a P wave is not followed by a QRS complex. The block is usually located in the AV node ("suprahisian"). Most common causes are drugs (β- and Ca^{++} channel blockers, digoxin) and vagal hypertonia. It can also be secondary to ischemia of the AV branch of the right or the circumflex coronary artery. It can rarely evolve to a higher degree AV block. Therefore therapeutic options are based on the identification of the causes and usually require no more than observation. When symptomatic and if vagal tone is involved, atropine 0.5 mg IV bolus (up to 3 mg in total) can transiently improve clinical status.

1.2.10 Higher Degree AV Blocks (Second-Degree AV Block Type II and Third-Degree AV Block)

Advanced AV blocks are a severe condition, which can quickly evolve into hemodynamic instability and/or cardiac arrest.

1.2.11 Second-Degree AV Block Type II (Mobitz II)

One or more P waves are not followed by a QRS complex without a progressive increase of the PR interval. Causes can be drugs (β- and Ca^{++} channel blockers,

digoxin, and other drugs as lithium) or a damage of the conduction pathways. It can be related to an acute coronary syndrome sometimes involving the left anterior descending coronary or one of its septal branches. It may easily evolve to a third-degree block or asystole, so it should be closely monitored.

1.2.12 Third-Degree AV Block

The ECG in the complete AV block shows a complete dissociation between atrial and ventricular activity with the complete absence of any AV conduction. Depending on the level of the block (suprahisian or infrahisian), the QRS morphology and ventricular rate can be different: in suprahisian block, escape rhythm arises from the AV junction (usually at 35–40 bpm, with narrow QRS complex); in infrahisian block, the rhythm arises from the ventricle; rate is less than 30 bpm with wide QRS complex.

In the presence of acute coronary syndrome, suprahisian blocks are usually secondary to an ischemia of the right coronary artery, while infrahisian blocks are frequently due to a huge ischemia within the interventricular septum due to a stenosis/occlusion of the left anterior descending coronary. Suprahisian blocks, like second-degree AV block type I, can be secondary to ischemia of the AV branch of the right or circumflex coronary artery and are usually more benign and recover spontaneously.

Complete AV block may also be related to drugs reducing AV conduction (β- and Ca^{++} channel blockers or digoxin) or reducing intraventricular conduction (as most antiarrhythmic drugs, leading to infrahisian blocks).

1.2.13 Accelerated Idioventricular Rhythm

In the presence of increased automaticity, ventricular rate may be higher than sinus rate (especially in the presence of sinus bradycardia), despite not exceeding 100/min. It is usually benign and asymptomatic; it can be a sign of reperfusion during acute coronary syndromes and should be treated only in presence of significant symptoms or hemodynamic impairment with drugs increasing sinus rate amines and atrial or ventricular pacing.

1.2.14 Treatment:

Treating an advanced-degree AV block requires fast choices [11–13, 16, 17]:

If hemodynamically unstable (Table 1.3):

- Activate the cardiologist for an IV pacing; discuss with him about the value of a coronarography when acute coronary syndrome is likely.
- As soon as possible, start transcutaneous pacing (with sedation).
- If not available, start drugs: dopamine (2–10 mcg/kg/min) or adrenaline (2–10 mcg/min).

Table 1.3 Unstable bradyarrhythmias

Treatment basic points consist in:
ECG, blood pressure, heart rate, respiratory rate, sat O_2 monitoring, hemodynamic assessment
Drugs: atropine, adrenaline or dopamine, isoproterenol
Guarantee the normal hemodynamic state
Emergency pacing should be considered if the need to maintain hemodynamic stability is imminent. As obvious this can be used only as a temporary means to bring the patient to a more stable solution (e.g., IV pacemaker)

If stable:

Evaluate the patient, and collect medical and drugs history; discuss with the cardiologist the management.

1.2.15 Pitfalls

- Confounding third-degree AV block with other bradyarrhythmias, in particular when similar atrial and ventricular rate are present ("iso-rhythmic dissociation"). It is necessary to evaluate very carefully the presence of the P wave (especially in leads V1 and DII) and the correlation with ventricular activity.
- Know your drugs: Atropine is quite ineffective on infrahisian blocks; amines increase oxygen consumption; isoprenaline–isoproterenol may provoke ventricular tachyarrhythmias, so it should be avoided, if possible, in the presence of ischemia.
- Always check if the transcutaneous pacing is achieving consistent capture by checking the femoral pulse.
- A low heart rate may not always be the cause of symptoms; it can be just a sign of other diseases. For example, sinus bradycardia and hypotension may be due to the vagal response in the presence of aortic dissection or Cushing's reflex during intracranial hypertension; in acute renal failure, elevated serum potassium can lead to significant bradyarrhythmias, while dehydration in prerenal acute renal failure can be the cause of the hypotension.

1.2.16 When Should the Consultant Cardiologist Be Called?

Any hemodynamic instability requires immediate intervention and support by the cardiologist to define the underlying causes and to help in the treatment.

High-degree AV blocks should be admitted to a monitoring-capable structure. Drugs withdrawn and indication to permanent pacemaker implantation have to be defined with the cardiologist [13].

A drug-related bradyarrhythmia may resolve after withdrawing the proarrhythmic treatment and requires amines or only temporary pacing.

Not all bradyarrhythmias need to be admitted into a cardiology ward. In the absence of high-degree AV block, stable patients may be safely admitted into medicine ward or even considered for discharge with an outpatient clinic follow-up program [1, 13, 17].

1.2.17 Definitive Pacemaker Indications

Briefly, indications to permanent pacing, in absence of transient or correctable causes, can be summarized as follows [19]:

Alternating or progressive bundle block (right bundle alternating with left bundle, right bundle + left anterior alternating with left posterior fascicular hemiblock)

Second-degree type II AV block: requires pacing when hemodynamically unstable and when the block is located in His bundle or below, even in asymptomatic patients

Third-degree AV block: indicates pacing in all acquired types, associated with syncope, hemodynamic instability, HR <40 bpm, or RR >3000 ms pauses

In older patients it's often difficult to determine the real cost/benefit ratio of a permanent pacemaker, and the balance between conservative and aggressive therapy should be discussed.

Absence of symptoms, a basal heart rate greater than 40 bpm, and the capacity of substitutive rhythm to increase the rate during physical activity may allow an observational strategy instead of a pacing immediate intervention [17].

1.2.18 A Suggested Algorithm/Pathway for Diagnosis and Treatment

	What to do	**How to do**
All patients	Assess hemodynamic status	Clinical evaluation NIBP, HR, RR, sat O2, body temperature monitoring, IV access
	Identify arrhythmia	ECG
	Identify underlying causes	Medical history, clinical evaluation, ECG, labs, EGA, echocardiography, chest X-ray, expert consulting
	Treat hemodynamic instability	Treat reversible causes; administer appropriate drugs; consider cardioversion or pacing if indicated

Tachyarrhythmia		**What to do**	**How to do**
		Treat instability	Perform immediate cardioversion (with procedural sedation if possible)
		Identify arrhythmia	ECG
	If narrow QRS complex		Manual maneuvers, adenosine if regular, beta-blockers, or Ca++ channel blockers. Consider rhythm control (CVES or drugs) if onset <48 h or rate control ± anticoagulant therapy if >48 h. Consider cardiologist consultant. Admit to ward if poorly tolerated hemodynamic status, severe or evolving underlying pathology, and uncontrolled heart rate regardless initial therapy
	If wide QRS complex		Consider adenosine only if regular monomorphic QRS is present; antiarrhythmic infusion, cardiologist consulting. Admit to ward if evolutive organic cardiopathy, persistence of tachyarrhythmia regardless antiarrhythmic therapy, polymorphic tachycardia, poorly tolerated hemodynamic status

Bradyarrhythmia	**What to do**	**How to do**
	Treat instability	Use atropine, dopamine, adrenaline, or isoprenaline or isoproterenol or perform immediate transthoracic pacing (with procedural sedation if possible). Consult the cardiologist
	Sinus bradycardia	Hemodynamic support, atropine. Identify and treat the underlying causes
	Identify arrhythmia	ECG
	Sick sinus syndrome	Dynamic ECG loop recorder, cardiologist evaluation; consider pacemaker implant
	If First-degree A V block or Second-degree AV block type I	Identify and treat underlying causes. Admit to ward if poorly tolerated hemodynamic status, severe or evolving underlying pathology, uncontrolled heart rate regardless initial therapy
	If high-degree AV blocks (II type II or III)	Be prepared to pace; consult the cardiologist. Admit to a monitoring capable ward

References

1. Neumar RW, Otto CW, Link MS, Kronick SL, Shuster M, Callaway CW, Kudenchuk PJ, Ornato JP, McNally B, Silvers SM, Passman RS, White RD, Hess EP, Tang W, Davis D, Sinz E, Morrison LJ. Adult advanced cardiovascular life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2010;122(18):S729–67.
2. Hood RE, Shorofsky SR. Management of arrhythmias in the emergency department. Cardiol Clin. 2006;24:125–33.

3. Delacrétaz E. Supraventricular tachycardia. N Engl J Med. 2006;354:1039–51.
4. Gross JB, Farmington CT, Bailey PL, Rochester NY, Connis RT, Woodinville WA, Cote´ CJ, Chicago IL, Davis FG, Burlington MA, Epstein BS, Washington DC, Gilbertson L, Boston MA, Nickinovich DG, Bellevue WA, Zerwas JM, Houston TX, Zuccaro G, Cleveland OH. Practice guidelines for sedation and analgesia by non-Anesthesiologists: an updated report by The American Society of Anesthesiologists Task Force on Sedation And Analgesia By Non-Anesthesiologists. Anesthesiology. 2002;96:1004–17
5. Tan G, Irwin MG. Recent advances in using propofol by non-anesthesiologists. F1000 Med Rep. 2010 Nov 11,2:79. doi:10.3410/M2-79.
6. Godwin SA, Burton JH, Gerardo CJ, Hatten BW, Mace SE, Silvers SM, Fesmire FM. Procedural sedation and analgesia in the emergency department. Ann Emerg Med. 2014;63:247–58.
7. Link MS. Evaluation and initial treatment of supraventricular tachycardia. N Engl J Med. 2012;367:1438–48.
8. January CT, Wann LS, Alpert JS, Calkins H, Cigarroa JE, Cleveland Jr JC, Conti JB, Ellinor PT, Ezekowitz MD, Field ME, Murray KT, Sacco RL, Stevenson WG, Tchou PJ, Tracy CM, Yancy CW. 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society. J Am Coll Cardiol. 2014;64:e1–76.
9. Oreto G, Luzza F, Satullo G, Donato A, Carbone V, Calabrò MP. Tachicardia a QRS larghi: un problema antico e nuovo. G Ital Cardiol. 2009;10:580–95.
10. Brugada P, Brugada J, Mont L, Smeets J, Andries EW. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. Circulation. 1991;83:1649–59.
11. Grantham HJ. Emergency management of acute cardiac arrhythmias. Aust Fam Physician. 2007;36:492–7.
12. Brady WJ, Harrigan RA. Evaluation and management of bradyarrhythmias in the emergency department. Emerg Med Clin North Am. 1998;16:361–88.
13. Lewalter T, Lickfett L, Schwab JO, Yang A, Lüderitz B. The emergency management of cardiac arrhythmia. Dtsch Arztebl. 2007;104:1172–80.
14. Semelka M, Gera J, Usman S. Sick sinus syndrome: a review. Am Fam Physician. 2013;87:691–6.
15. Crisel RK, Farzaneh-Far R, Na B, Whooley MA. First-degree atrioventricular block is associated with heart failure and death in persons with stable coronary artery disease: data from the Heart and Soul Study. Eur Heart J. 2011;32:1875–80.
16. Brignole M, Auricchio A, Baron-Esquivias G, Bordachar P, Boriani G, Breithardt OA, et al. 2013 ESC guidelines on cardiac pacing and cardiac resynchronization therapy: the Task Force on cardiac pacing and resynchronization therapy of the European Society of Cardiology (ESC). Developed in collaboration with the European Heart Rhythm Association (EHRA). Eur Heart J. 2013;34:2281–329.
17. Edhag O, Swahn A. Prognosis of patients with complete heart block or arrhythmic syncope who were not treated with artificial pacemakers. A long-term follow-up study of 101 patients. Acta Med Scand. 1976;200:457–63.

Syncope: First Evaluation and Management in the Emergency Department

2

Franco Giada and Andrea Nordio

2.1 Epidemiology of Syncope

Epidemiological studies conducted in the USA have estimated that 30 % of the general population experience at least one episode of syncope in their lifetime [1], which is responsible for about 1–3 % of admissions to the emergency department (ED) and 1–3 % of hospitalizations [2]. In Italy, syncope accounts for 1–2 % of both admissions to the ED and all hospitalizations [3–7]. About half of the patients attending emergency facilities for syncope are subsequently hospitalized; the mean duration of hospitalization is about 8 days [3–7]. Moreover, in industrialized countries the progressive aging of the population, together with the higher prevalence of syncope among elderly subjects, are likely to increase the impact of syncope on healthcare systems in the near future [6].

2.2 Costs of Syncope

The fact that syncope may be caused by pathological conditions that have a severe prognosis, together with the lack of a diagnostic gold standard, results in frequent hospitalization and the prescription of numerous costly instrumental investigations, which increase healthcare costs. One North American study [8] estimated in 1993 that the mean annual cost per patient hospitalized for syncope was US$4132. In the case of recurrent syncope, this figure rose to US$5281. In the USA the total annual

F. Giada (✉)
Cardiovascular Department, P.F. Calvi Hospital,
Via Largo San Giorgio 3, 30033 Noale-Venice, Italy
e-mail: francogiada@hotmail.com

A. Nordio
Cardiovascular Department, Ospedali Riuniti and University of Trieste, Trieste, Italy

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cost of syncope amounts to $2.4 billion, similar to that for chronic respiratory diseases and human immunodeficiency virus. In studies conducted in Italy, the mean cost per patient hospitalized for syncope is reported to vary from €1000 to €3000 [9]. These costs are chiefly conditioned by the duration of hospitalization and by the number and type of diagnostic investigations carried out.

Thus, syncope places a considerable burden on healthcare resources. The implementation of an appropriate diagnostic and therapeutic strategy for tackling the problem of syncope in the ED therefore constitutes a challenge from clinical, organizational, and economic standpoints.

2.2.1 Difficulties in the Management of Patients with Syncope in the ED

Syncope is defined as a self-limiting transient loss of consciousness (TLOC), which usually causes falls. The onset of syncope is relatively sudden and the recovery is spontaneous, complete, and rapid. The underlying pathophysiological mechanism is transitory global brain hypoperfusion [10].

The first difficulty encountered in ED in the management of patients who present with TLOC is to distinguish syncope from losses of consciousness (Fig. 2.1) not caused by cerebral hypoperfusion (e.g., epilepsy, metabolic disorders, vertebrobasilar transient ischemic attack, hypoxia), or from conditions with only apparent loss

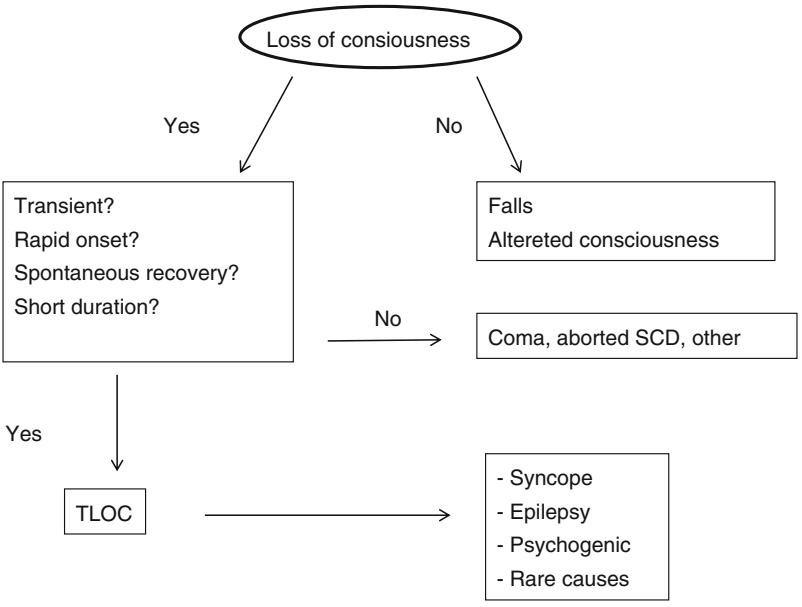

Fig. 2.1 Context of transient loss of consciousness. *SCD* sudden cardiac death, *TLOC* transient loss of consciousness

Table 2.1 Conditions incorrectly diagnosed as syncope

Disorders with partial or complete LOC (without global cerebral hypoperfusion)
Epilepsy
Metabolic disorders including hypoglycemia, hypoxia, hyperventilation with hypocapnia
Intoxication
Vertebrobasilar TIA
Disorders without impairment of consciousness
Cataplexy
Drop attacks
Falls
Functional (psychogenic pseudosyncope)
TIA of carotid origin

LOC loss of consciousness, *TIA* transient ischemic attack

of consciousness (drop attack, cataplexy, psychogenic pseudosyncope, accidental falls in the elderly) [10] (Table 2.1). Moreover from an etiological standpoint, syncope is classified within three main groups: neuromediated or reflex (representing the epidemiologically prevalent cause of syncope), orthostatic, and cardiac (arrhythmic or structural, the second most common cause of syncope). Each of these groups comprises numerous disorders that may manifest in the form of syncope and are not always easy to evaluate (Table 2.2). Finally, the prognostic significance of syncope depends on the underlying pathology, ranging from the generally benign prognosis seen in neuromediated syncope to the more severe prognosis associated with cardiac syncope.

This syncope often raises diagnostic and therapeutic problems which is proved by the diversity of the pathways followed by patients attending the ED. Indeed, data from the literature [4–7] reveal that these patients may be hospitalized in cardiology, neurology, pediatric, geriatric, internal medicine, and orthopedic wards. These patients then undergo, often inappropriately, several instrumental examinations that are costly and have a low diagnostic yield. The result is that hospitalization is prolonged and healthcare costs rise, while a correct diagnosis of the cause of syncope fails to be reached in a high percentage of cases. As the risk of malpractice suits is high in this field, ED physicians often adopt a strategy of "self-defense," which results in an increase of the number of hospitalizations and inappropriate instrumental investigations. Thus, the current management of syncope displays little diagnostic efficacy and considerable economic inefficiency.

The factors underlying these disappointing results can be summed up as follows: insufficient competence and attention on the part of physicians with regard to the differential diagnosis of TLOC, owing to the difficulty of tackling this problem in a multidisciplinary manner; a "defensive" stance prompted by the possible legal implications of a wrong diagnosis; and, in various healthcare institutions, the lack of diagnostic and therapeutic facilities specialized in the management of syncope patients [11, 12].

Table 2.2 Classification of syncope

Neurally mediated syncope
Vasovagal cause
Mediated by emotional distress (fear, pain, instrumentation blood phobia)
Orthostatic stress
Situational
Sneeze, cough
Gastrointestinal stimulation (defecation, abdominal pain, swallowing)
After micturition
After exercise
Postprandial
Others
Carotid sinus syncope
Forms without apparent triggers or atypical presentation
Orthostatic hypotension syncope
Primary autonomic failure
Pure autonomic failure, multiple atrophy, Parkinson disease with autonomic failure
Lewy body dementia
Secondary autonomic failure
Diabetes, amyloidosis, uremia, spinal injuries
Drug-induced orthostatic hypotension
Alcohol, vasodilators, diuretics, antidepressants
Volume depletion
Hemorrhage, diarrhea, vomiting, etc
Cardiovascular syncope
Arrhythmia as primary cause
Bradycardia: sinus node dysfunction, AV conduction system disease, implanted device dysfunction
Tachycardia: supraventricular, ventricular
Drug-induced bradycardia and arrhythmias
Structural disease
Cardiac: valvular diseases, AMI, hypertrophic cardiomyopathy, etc
Others: acute aortic dissection, pulmonary hypertension, pulmonary embolus

2.2.2 The Recommended Management of Syncope in the ED

Most of the direct cost of syncope is attributable to hospitalization. A proper diagnostic evaluation and prognostic stratification of syncope in the ED should limit hospitalization to patients suffering from heart disease, serious neurological diseases, or severe secondary traumas.

In 2009 the European Society of Cardiology developed guidelines on the management of syncope [10]. This document defined what has become the current standard for the management of patients with TLOC and the most valuable diagnostic pathway, and gave recommendations on indications and interpretation of diagnostic tests with indications for hospitalization and treatment.

According to the guidelines, the cornerstone in syncope management in the ED is the initial clinical evaluation, i.e., history, physical examination, recumbent and orthostatic blood pressure measurement, and electrocardiogram (ECG).

Patients should be interrogated about:

- The circumstances (position and activity of the patient, presence of predisposing factors) and symptoms (nausea, dizziness, palpitations) occurring just before the attack
- Manner of fall (slumping or kneeling over), duration, and whether movements were present during LOC (if witnessed)
- Number, frequency of spells, and age at first episode
- Family history of cardiac disease or sudden death, neurological history, metabolic disorders

Consultation by a cardiologist is indicated when a cardiac cause is suspected or ascertained, particularly in cases of:

- Presence of definite heart disease, family history of sudden death, or channelopathy
- Syncope during exertion or in supine position
- Sudden onset of palpitation prior to the syncope

ECG abnormalities such as intraventricular delay with QRS duration >120 ms, Mobitz II second-degree atrioventricular block, sinoatrial pauses >3 s, nonsustained ventricular tachycardia, preexcitation, long or short QT intervals, Brugada pattern, negative precordial T waves suggestive of right ventricular dysplasia, Q waves suggesting previous myocardial infarction, pacemaker/implantable cardioverter-defibrillator malfunction.

Neurological consultation (Fig. 2.2) is indicated for a nonsyncopal TLOC cause (epilepsy, transient ischemic attack, subclavian steal syndrome, psychogenic pseudosyncope), especially in cases of:

- Presence of aura before the event
- Tonic-clonic movements coinciding with the onset of LOC (and not some seconds later) or hemilateral
- Clear automatisms or tongue biting
- Prolonged confusion after the episode
- Family history of seizures, sleepiness or headache after the event

Specific tests such as neurological investigations or blood tests are only indicated for a suspicion of nonsyncopal TLOC.

This strategy requires adequate clinical competence on the part of all of the physicians involved in the management of these patients. Table 2.3 presents the diagnostic criteria that permit a diagnosis of the causes of syncope in the ED by means of initial evaluation, and Table 2.4 lists the clinical features that suggest a diagnosis.

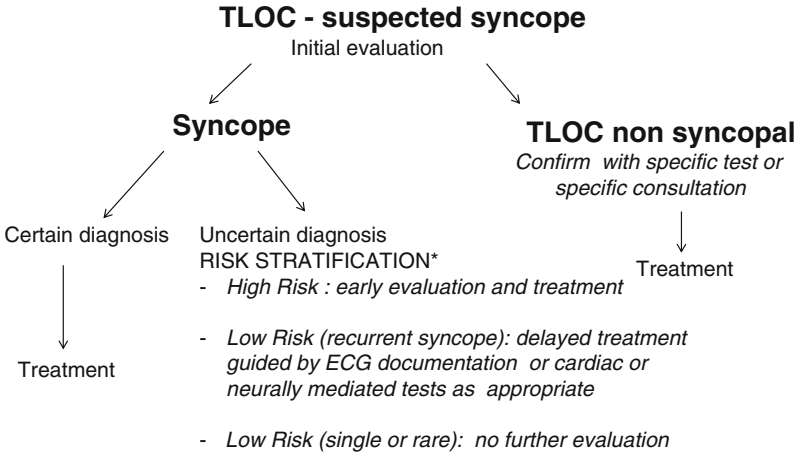

Fig. 2.2 Diagnostic flowchart of patients with suspected T-LOC.*May require laboratory investigations. *ECG* electrocardiographic, *TLOC* transient loss of consciousness

Table 2.3 Diagnostic criteria of causes of syncope with initial evaluation

Vasovagal syncope is diagnosed if syncope is precipitated by emotional distress or orthostatic stress, and is associated with typical prodrome
Situational syncope is diagnosed if syncope occurs during or immediately after specific triggers listed in Table 2.2
Orthostatic syncope is diagnosed when it occurs after standing up and there is documentation of OH
Arrhythmia-related syncope is diagnosed by ECG when there is:
Persistent bradycardia (<40 bpm in awake or repetitive sinoatrial block or sinus pauses >3 s)
Mobitz II or III degree AV block (second or third degree)
Alternating BBB (left and right)
VT or rapid paroxysmal SVT
Polymorphic VT nonsustained and long or short QT interval
PM or ICD malfunction with cardiac pauses
Cardiac ischemia related syncope is diagnosed when syncope presents with ECG evidence of acute ischemia with or without myocardial infarction
Cardiovascular syncope is diagnosed when syncope presents in patients with prolapsing atrial myxoma, severe aortic stenosis, pulmonary hypertension, pulmonary embolus, or acute aortic dissection

AV atrioventricular, *BBB* bundle-branch block, *ECG* electrocardiogram, *PM* pacemaker, *ICD* implantable cardioverter-defibrillator, *OH* orthostatic hypotension, *SVT* supraventricular tachycardia, *VT* ventricular tachycardia, *VVS* vasovagal syncope

The ideal TLOC management should lead, through initial clinical evaluation, to an identification of the nature of the TLOC (syncope or syncope-like conditions) and, in the case of syncope, to an etiological diagnosis and a rapid stratification of patients into three categories (Fig. 2.3):

Table 2.4 Clinical features suggesting a diagnosis at initial evaluation

Neurally mediated syncope
Absence of heart disease
Recurrent syncope history
After sudden unexpected unpleasant sight, sound, smell, pain
Vomiting, nausea during syncope
Prandial or postprandial
After exertion
With head rotation or pressure on carotid sinus
Syncope due to orthostatic hypotension
After standing up
Start or changes of dosage of vasodepressive drugs
Prolonged standing (crowded hot places)
Autonomic neuropathy or Parkinsonism
Standing after exertion
Cardiovascular syncope
Structural heart disease
Family history of channelopathy or USD
Supine or during exertion
ECG findings suggesting arrhythmic syncope:
LBBB or RBBB combined with LA or LP fascicular block
Intraventricular conduction abnormalities (QRS >120 s)
Mobitz I second-degree AV block
Asymptomatic inappropriate sinus bradycardia (<50 bpm)
Nonsustained VT
Preexcited QRS complexes
Long or short QT intervals
Early repolarization
RBBB pattern with ST elevation in leads V1–V3
Q waves suggesting myocardial infarction

USD unexpected sudden death, *LBBB* left bundle-branch block, *RBBB* right bundle-branch block, *LA* left anterior, *LP* left posterior, *VT* ventricular tachycardia

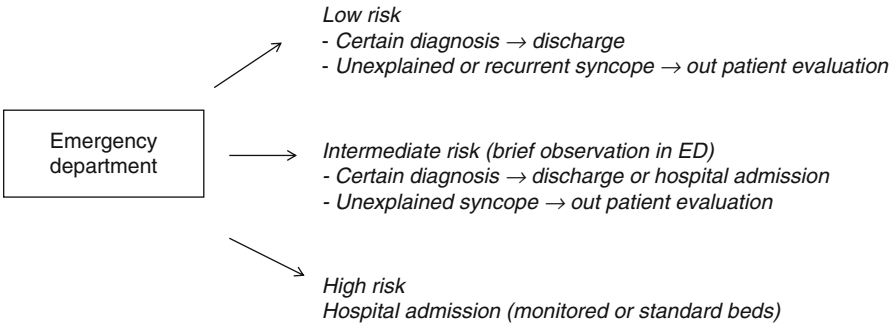

Fig. 2.3 Diagnostic pathway for the management of patients admitted to the ED for syncope

- *Low-risk patients*, who can be handled in the outpatient clinic by the general practitioner or specialist, on an ordinary time schedule, and requiring few, if any, selected examinations [15]; this group includes patients with very likely neurogenic syncope and without injuries
- *Intermediate-risk patients*, who need to be managed within a short time in a specialist outpatient clinic or by means of brief observation in the emergency department, when high risk is not evident but cannot be immediately excluded
- *High-risk patients*, who need to be hospitalized urgently and to immediately undergo appropriate diagnostic and therapeutic procedures. This group includes patients with severe structural or coronary heart disease (heart failure, severe left ventricular dysfunction, previous myocardial infarction), clinical or ECG features suggesting arrhythmic syncope (see above), or important comorbidities (Table 2.5)

Finally, the European guidelines recommend that rapid risk stratification in the ED can be achieved by using the following two scores:

- The OESIL Risk Score [12], which is easy to apply and it includes age >65 years, underlying disease, syncope without prodromes, and an abnormal ECG. A total score of 0 represents 0 % mortality and a score of 1 is associated with <1 % mortality at 1 year. Scores of 3 (mortality of 35 % derivation/29 % validation sets) and 4 (mortality of 57 % and 53 % derivation and validation sets, respectively) carry significantly greater risk.
- The EGSYS Risk Score [13], which shows that the presence of abnormal ECG and/or the presence of structural cardiac disease are the main factors predictive

Table 2.5 Clinical criteria to identify short-term high-risk patients

Severe structural CAD (HF, low LVEF, MI)
Clinical or ECG aspects suggesting arrhythmic syncope
During exertion or supine
Palpitations (during the syncope)
Bifascicular block
Intraventricular conduction abnormalities (QRS >120 ms)
Inadequate sinus bradycardia or SA block in absence of negative chronotropic medications or physical training
Preexcited QRS complex
Prolonged or short QT interval
RBBB pattern with ST elevation (V1–V3 leads)
Negative T waves in right precordial leads, epsilon waves, and ventricular late potentials (ARVC)
Important comorbidities
Anemia (severe)
Electrolyte disturbance

CAD coronary artery disease, *HF* heart failure, *LVEF* left ventricular ejection fraction, *MI* myocardial infarction, *RBBB* right bundle-branch block, *ARVC* arrhythmogenic right ventricular cardiomyopathy

Table 2.6 Risk stratification at initial evaluation in OESIL and EGSYS studies

Study	Risk factors	Score	Endpoints	Results (validation cohort)
OESIL [12]	Abnormal ECG	0–4 (1 point each item)	1 year total mortality	0 % score 0
	Cardiovascular disease (history)			0.6 % score 1
	Lack of prodrome			14 % score 2
	Age >65 years			29 % score 3
EGSYS [13]	Palpitations before syncope (+4)	Sum of + and − points	2 year total mortality	2 % score < 3
	Abnormal ECG and or heart disease (+3)			21 % score ≥3
	Syncope during effort (+3)			
	Syncope while supine (+2)		Cardiac syncope	2 % score < 3
	Predisposing/precipitating factors (−1)			13 % score 3
	Autonomic prodrome (nausea, vomiting) (−1)			33 % score 4
				77 % score > 4

of mortality in 2 years; this score has a very good diagnostic yield in identifying syncope of cardiac origin (Table 2.6).

This strategic course underlines that prognostic stratification and the choice of instrumental investigations selected on the basis of the characteristics of each patient are crucial to the cost-effective management of patients with syncope in the ED [14–17].

References

1. Savage DD, Corwin L, McGee LD, et al. Epidemiology features of isolated syncope: the Framingham study. Stroke. 1985;16:626–9.
2. Day SC, Cook EF, Funkenstain H, Goldman L. Evaluation and outcome of emergency room patients with transient loss of consciousness. Am J Med. 1982;73:15–23.
3. Ammirati F, Colivicchi F, Minardi G, et al. Gestione della sincope in ospedale: lo studio OESIL (Osservatorio Epidemiologico della Sincope nel Lazio). G Ital Cardiol. 1999;29:533–9.
4. Del Greco M, Cozzio S, Scillieri M, Cappari F, Scivales A, Disertori M. The ECSIT study (Epidemiology and Costs of Syncope in Trento). Diagnostic pathway of syncope and analysis of the impact of guidelines in a district general hospital. Ital Heart J. 2003;4:99–106.
5. Haan MN, Selby JV, Quensenberry CP, et al. The impact of aging and chronic disease on use of hospital and outpatient service in a large HMO. J Am Geriatr Soc. 1997;45:667–74.

6. Nyman JA, Krahn AD, Bland PC, et al. The costs of recurrent syncope of unknown origin in elderly patients. PACE. 1999;22:1386–94.
7. Olde Nordkamp LAR, van Dijk N, Ganzeboom KS, Reitsma JB, Luitse JSK, Dekker LRC, et al. Syncope prevalence in ED compared to that in the general practice and population: a strong selection process. Am J Emerg Med. 2009;27:271–9.
8. Sun BC, et al. Direct medical costs of syncope-related hospitalizations in the United States. Am J Cardiol. 2005;95(5):668–71.
9. Del Rosso A, Bernadeschi M, Ieri A. Costi sociali della sincope. Ital Heart J Suppl. 2000;1:772–6.
10. The Task Force for the Diagnosis and Management of Syncope of the European Society of Cardiology (ESC). Developed in collaboration with, European Heart Rhythm Association (EHRA), Heart Failure Association (HFA), and Heart Rhythm Society (HRS). Guidelines for the diagnosis and management of syncope. Eur Heart J. 2009;30(21):2631–71.
11. Giada F, Ammirati F, Bartoletti A, Del Rosso A, Dinelli M, Foglia-Manzillo G, et al. La Syncope Unit: un nuovo modello organizzativo per la gestione del paziente con sincope. G Ital Cardiol. 2010;11:323–8.
12. Colivicchi F, Ammirati F, Melina D, Guido V, Imperoli G, Santini M. Development and prospective validation of a risk stratification system for patients with syncope in the emergency department: the OESIL risk score. Eur Heart J. 2003;24:811–9.
13. Del Rosso A, Ungar A, Maggi R, Giada F, Petix NR, De Santo T, et al. Clinical predictors of cardiac syncope at initial evaluation in patients referred urgently to a general hospital: the EGSYS score. Heart. 2009;94:1620–6.
14. Shen WK, Decker WW, Smars PA, Goyal DG, Walker AE, Hodge DO, et al. Syncope evaluation in the emergency department study (SEEDS). A multidisciplinary approach to syncope management. Circulation. 2004;110:3636–45.
15. Brignole M, Disertori M, Menozzi C, et al. The management of syncope referred for emergency to general hospitals with and without Syncope Unit facility. Europace. 2003;5:293–8.
16. Disertori M, Brignole M, Menozzi C, et al. Management of syncope referred for emergency to general hospitals. Europace. 2003;5:283–91.
17. Brignole M, Menozzi C, Bartoletti A, Giada F, Lagi A, Ungar A, et al. A new management of syncope: prospective systematic guideline-based evaluation of patients referred urgently to general hospitals. Eur Heart J. 2006;27:76–82.

Management of Bradyarrhythmias in Emergency

3

Luca Salvatore, Silvia Magnani, Gerardina Lardieri, and Elena Zambon

The sudden appearance in patients of changes in heart rhythm is a situation that requires rapid diagnosis and treatment in emergency departments and intensive care units. The slowdown and block of cardiac impulse conduction form the basis of symptomatic bradycardia.

The prevalence of cardiac bradyarrhythmias and conduction disturbances in outpatients evaluated for the first time is 30–40 %, with an incidence of 3 % for atrioventricular (AV) block and sinus node dysfunction, and 8 % for disorders of intraventricular conduction [1].

Dysfunction of the sinus node has significant clinical implications. Development of concomitant AV block is not uncommon (incidence 8.4 %), as is the presence or appearance of atrial fibrillation (AF), especially in patients already treated with only ventricular pacing (incidence 22 %), with a consequent high risk of systemic thromboembolism [2, 3]. In elderly patients a low heart rate can promote the development or worsening of heart failure, even in the presence of preserved systolic function [4].

The onset of arrhythmias after cardiac and noncardiac surgery, a relatively common occurrence, is a major predictor of morbidity of surgical procedures and is associated with a prolonged hospital stay, although severe bradyarrhythmias requiring treatment occur in less than 1 % of cases. Triggers are generally transient events such as hypoxia, myocardial ischemia, excess catecholamine, or electrolyte disorders [5, 6].

L. Salvatore (✉) • S. Magnani • G. Lardieri • E. Zambon
Cardiovascular Department, Ospedali Riuniti and University of Trieste, Trieste, Italy
e-mail: lucasalvatore@alice.it

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3.1 Anatomy and Pathophysiology

The cardiac impulse begins in the *sinus atrial node*, located in the upper lateral wall of the right atrium near the superior vena cava, vascularized by the right coronary artery (60 %) and the circumflex artery (40 %). The impulse reaches the AV node, which, through the nodal-Hisian system, connects the atria with the ventricles. The nodal-Hisian system is formed by the *AV node* (located in the front part of the Koch triangle, vascularized by the right coronary artery) and the *bundle of His* (vascularized by both the left and right coronary artery). The bundle of His is divided into the *right branch* (vascularized by the anterior descending artery and right coronary artery) and the *left branch*, which in turn is divided into two bundles, the anterior (vascularized by the anterior descending artery) and the posterior (vascularized by the circumflex artery). From here the Purkinje fibers originate, guaranteeing simultaneous activation of the ventricles.

3.2 Bradycardia

A heart rate of less than 60 bpm is usually considered as bradycardia. The usual symptoms of bradycardia are asthenia, fatigue, dyspnea, chest discomfort or pain, prior or complete loss of consciousness, light-headedness, and decreased level of consciousness. Frequent noticeable signs include hypotension and/or orthostatic hypotension, diaphoresis, bradycardia-related (escape) frequent premature ventricular complexes, or other ventricular tachyarrhythmias. Usually the symptoms are relevant when the heart rate is lower than 40 bpm, or higher in the presence of a pre- or coexistent cardiac disease.

Hemodynamically unstable bradycardia may represent a serious life-threatening emergency, with 30-day mortality rates of up to 16 % being reported [7–9]. Bradycardia can be the expression of the dysfunction of *automaticity* or the *conduction* of the electrical impulse, and can be detected at all levels of the conduction system (sinoatrial node, AV node, intraventricular branches).

3.3 Sinus Node Dysfunction

The main manifestations of sinus node dysfunction are *sinus bradycardia* and *sinus arrest*.

Sinus node dysfunction recognizes *intrinsic* causes: degenerative disease, chronic or acute ischemia, infiltrative disease (amyloidosis, hemochromatosis, tumors), inflammatory causes (pericarditis, myocarditis), musculoskeletal disease (Duchenne dystrophy, Friedreich ataxia), vasculitis (systemic lupus erythematosus, scleroderma), and surgical (atrial septal defect correction); and *extrinsic* causes: drugs (amiodarone, flecainide, propafenone, sotalol, chinidina, disopyramide, procainamide), electrolyte disorders (hyperkalemia), endocrinopathies (hypothyroidism), vagal reflex during ischemia, neuromediated syndrome (vasovagal, cough, carotid sinus hypersensitivity), intracranial hypertension, and obstructive jaundice.

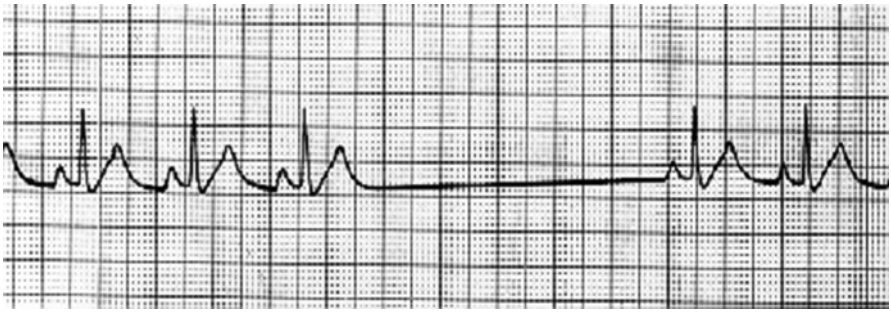

Fig. 3.1 Sinus arrest

3.3.1 Sinus Bradycardia

Sinus bradycardia can be secondary to intrinsic factors such as fibrosis (aging or loss of "pacing cells" [10, 11] resulting from inferior myocardial infarction, or to extrinsic factors such as an increased vagal tone, an abnormal neurocardiogenic reflex, drugs such as β-blockers or Ca^{2+} antagonists, or drugs reducing sympathetic tone such as sympatholytic antihypertensives [12].

The electrocardiogram (ECG) shows a regular sinus rhythm with heart rate lower than 60 bpm and a constant 1:1 AV conduction, with a normal PR interval (120–200 ms); P waves are regular, with the same shape and axis between 0° and 90°.

Symptoms can be absent at rest and may appear only during effort. In general, sinus bradycardia does not require treatment; intravenous atropine can be effective, but is useful only for short-term treatment because of its short half-life; sometimes temporary or permanent pacing may be necessary, favoring atrial over ventricular stimulation [13, 14].

3.3.2 Sinus Arrest

Sinus arrest is the absence of onset of spontaneous atrial impulse. It is generally considered significant when this pause lasts longer than 3 s. The most common causes are tissue fibrosis, ischemia, digoxin intoxication, cerebral ischemia, and excessive vagal tone (Fig. 3.1).

3.4 Atrioventricular Block

Atrioventricular block occurs when the conduction of the atrial impulse to the ventricles slows down or is blocked (paroxysmal or permanently), even though the AV junction is not physiologically refractory.

In the presence of a high atrial rate (e.g., atrial tachycardia or AF), the term AV block is correct only if the ventricular rate is lower than 60 bpm.

There are three anatomical sites of block: (1) above Hisian (inside the AV node), (2) intra-Hisian (inside the His bundle), and (3) infra-Hisian (distal His bundle or bundle branches).

The identification of anatomical site is relevant, as the distal blocks are those with the worst prognosis.

3.4.1 First-Degree AV Block

Every single atrial impulse is followed by a QRS complex, but the PR interval is greater than 200 ms. The block is usually above the AV node, even if there may be a wide QRS complex caused by a coexisting bundle-branch block. In general it is not associated with heart disease, and is usually secondary to increased vagal tone or is drug related (β-blocker or digoxin), although sometimes it can be associated with myocardial ischemia, infiltrative myocardiopathies, myocarditis, Addison disease, or congenital heart disease (septal defect, Ebstein). Prognosis, however, is benign, and no treatment is required. An exception can be the occurrence of a first-degree AV block in aortic valve endocarditis resulting from an abscess of the interventricular septum.

3.4.2 Second-Degree AV Block

Conduction from the atria to the ventricles is intermittent. There are two types of second-degree AV block.

Type I, also known as Mobitz I or Luciani-Wenckebach, involves progressive increase of the PR interval and consequent shortening of the RR interval until a nonconducted P wave develops. The morphology of the QRS complex is normal. The site of the block is usually at or above the AV node [15]. Etiology mainly includes digoxin intoxication, myocardial ischemia (inferior myocardial infarction), excess calcium deposits such as occur in aortic stenosis, or renal insufficiency. It can be also observed in both trained athletes and normal persons during sleep, attributable to increased vagal tone [16–18]. Prognosis is excellent if asymptomatic, while the onset of symptoms such as syncope or heart failure may require the placement of a definitive pacemaker.

Type II (Mobitz II) shows a sudden block of a P wave with a ratio of 3:2, 4:3, and so forth. PP and RR intervals are regular (Fig. 3.2). The block is intra- or infra-Hisian, and tends to progress toward complete AV block with prolonged ventricular pause and syncope [19]. Guidelines favor permanent pacing even in asymptomatic patients with type II block [20].

3.4.3 Atrioventricular Block with 2:1 Conduction

This condition is characterized by an intermittent block with a P/QRS ratio of 2:1, thus having features of both Mobitz I and II. Block can be at any nodal site and

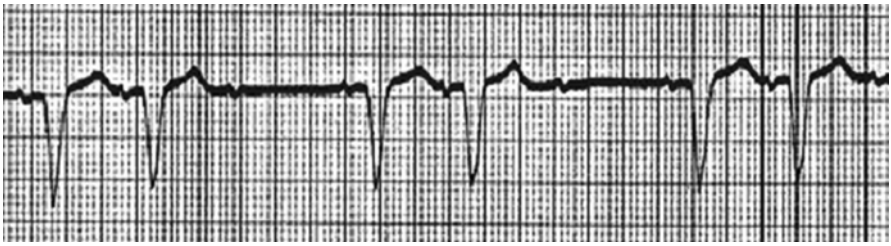

Fig. 3.2 II degree–Mobitz 2 AV block (with intraventricular delay)

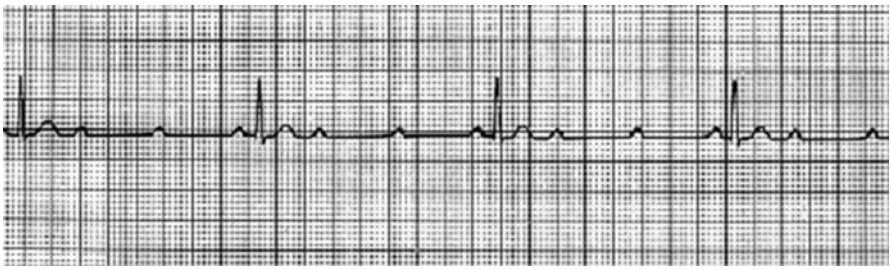

Fig. 3.3 Advanced (3:1) tipo II AV block

intra- or infra-Hisian, the severity depending on the resultant ventricular rate. In an asymptomatic patient a 2:1 block with a coexisting bundle-branch block is an indication for pacing, assuming that the block is infranodal [21].

3.4.4 Advanced AV Block

In advanced AV block, two or more consecutive P waves are blocked with a P/QRS ratio of 3:1, 4:1, and so forth (Fig. 3.3). In everyday practice this term is also used for low-rate permanent AF.

3.4.5 Low-Rate Atrial Fibrillation

The absence of recognizable P wave, irregular RR intervals, and narrow or wide QRS complexes characterizes low-rate AF. Causes are usually drugs (antiarrhythmic, digoxin, β-blockers, Ca^{2+} antagonists), especially in elderly patients with impaired renal and/or liver function, vagal hypertone (young subjects), or impaired AV conduction.

Treatment consists in ensuring hemodynamic normality using drugs (atropine followed by amines) or transcutaneous or transvenous pacing in symptomatic bradycardia.

3.4.6 Third-Degree AV Block

The inability of P waves to conduct to the ventricles (complete block of AV conduction) characterizes third-degree AV block. There is no relationship between P waves and QRS complexes, which are escape beats. Narrow QRS escape rhythms, with regular RR and a frequency between 40 and 60 bpm, are secondary to blocks within the AV node. Wide QRS escape rhythms with frequency between 40 and 20 bpm are secondary to infra-Hisian blocks.

In patients with AF and complete AV block a regular ventricular rate is observed, because of junctional (usually at about 35 bpm) or infra-Hisian escape rhythm.

Third-degree AV block can be secondary to pharmacologic treatments (digoxin, β-blockers, Ca^{2+} antagonists, quinidine, procainamide) or sclerodegenerative phenomena of conduction fibers, or may occur during the acute phase of myocardial infarction. Prognosis is more favorable if associated with inferior myocardial infarction (supra-Hisian block, narrow QRS escape rhythm, and generally spontaneous resolution), while the prognosis is worse in anterior myocardial infarction (infra-Hisian block and wide QRS escape rhythm, generally associated with a wider area and less reversibility of ischemic injury).

Less common causes include infectious diseases (myocarditis, Lyme disease, Chagas disease), rheumatic etiology (Reiter syndrome, scleroderma, rheumatoid arthritis), infiltrative disease (amyloidosis, sarcoidosis, multiple myeloma), electrolyte disorders, and iatrogenic factors (cardiac surgery, radiofrequency ablation) (Fig. 3.4).

3.4.7 Interventricular Block

Left bundle-branch block (LBBB) is a rare finding in the young population, and when present should raise the suspicion of an underlying heart disease. Incidence increases with age [22] and is associated with various causes of heart disease (hypertensive, ischemic, valvular, idiopathic dilated cardiomyopathy). Prognosis depends on the severity of the underlying disease.

Left anterior hemi-block (LAHB), more frequent in the adult male population, is associated with hypertension and ischemic heart disease. Progression to LBBB or complete AV block is uncommon [23].

Left posterior hemi-block (LPHB) is a rare isolated finding because of both the short course and double vascularization of the left posterior fascicle.

Right bundle-branch block (RBBB) as an incidental ECG finding is not often linked to heart disease, but may be associated with systemic hypertension, pulmonary heart disease, or ischemic heart disease.

Bifascicular block commonly indicates the association of an interruption or delay in impulse conduction along both sets of left bundle branch, or along the right branch and one of the two sets of left bundle branch.

RBBB + LAHB, a more common condition, shows a progression to complete AV block in less than 6 % of patients, while RBBB + LPHB, less frequent, has a greater progression toward complete AV block (close to 75 %).

Patients with bifascicular block associated with first-degree AV block (especially infranodal, when the HV interval, documented during invasive electrophysiological evaluation, is >100 ms) have a higher risk for developing complete AV block, and some authors recommend permanent cardiac stimulation [24]. The diagnosis of trifascicular block can be made on surface ECG only in the presence of alternating bundle-branch block (LBBB alternating with RBBB, or RBBB + LAHB alternating with RBBB + LPHB).

3.4.8 Atrioventricular Dissociation

AV dissociation is present when atrial activity is independent from ventricular activity, both occurring at regular rates. AV dissociation is not synonymous with AV block; in fact while patients with AV block also have an AV dissociation, patients with AV dissociation may not have an AV block, for example when an escape junctional (or ventricular) rate is higher than the sinus rate in the absence of retrograde ventricular-atrial conduction.

Elderly patients with degenerative cardiac disease are more likely to have AV dissociation [25].

Treatment depends on the causes, and ranges from antiarrhythmic drug treatment to permanent pacemaker placement.

3.4.9 Conduction Disturbances during Acute Myocardial Infarction

Sinus node dysfunction in the course of myocardial infarction may be secondary to the occlusion of the circumflex artery or the right coronary artery. The bradycardia that follows may favor escape rhythms with narrow QRS, which originate at the junction or in the His bundle and have a heart rate of about 50–60 bpm becoming stable over time, whereas escape rhythms with wide QRS originate from the Purkinje fibers with a heart rate below 40 bpm, are less stable, and may evolve to asystole. The AV block that occurs in the early stages of myocardial infarction is generally secondary to hypervagotonia and is reversible with atropine, while blocks occurring later are frequently related to ischemia of the AV node.

Second-degree type I AV block during inferior myocardial infarction tends to resolve spontaneously within 48 h and does not require invasive treatment unless there is hemodynamic compromise.

Second-degree type II AV block is more likely to progress to complete AV block, and occurs more frequently in anterior myocardial infarction, although it can also occur in inferior myocardial infarction because of occlusion of a large posterior descending artery.

Complete AV block, which is present in 8–13 % of patients with myocardial infarction, occurs in both inferior and anterior myocardial infarction, and can be preceded by fascicular blocks or bundle-branch block with second-degree AV block.

It is associated with a higher incidence of ventricular fibrillation/tachycardia, pulmonary edema, and in-hospital mortality [26].

3.4.10 Cardiac Arrest and Sudden Cardiac Death

The incidence of sudden cardiac death (occuring within 1 h from the onset of cardiac symptoms) is variable, depending on the varying prevalence of coronary disease in different countries [27]. In the USA there are 300,000 cardiac deaths annually, with a total annual incidence of 1 in 1,000 individuals [28].

Ventricular fibrillation is the first rhythm detected in 75–80 % of cases, while in the remaining 20–25 % extreme bradycardia or pulseless electrical activity are causes of cardiac arrest.

Bradyarrhythmic cardiac arrest is secondary to the inability of cardiac automatism to emerge and replace the sinus node or normal function of the AV junction.

Cardiac arrest is more common in advanced cardiac disease, generally as a consequence of extracellular potassium accumulation (secondary to hypoxemia, acidosis, shock, or renal insufficiency), which reduces myocellular automatism.

Pulseless electrical activity is an electrical stable cardiac rhythm in the absence of any effective myocardial contraction. The *primary form* is generally the terminal event of a severe cardiopathy or expression of acute myocardial ischemia, and the *secondary form* is a severe expression of a sudden failure in venous return, such as in pulmonary embolism, cardiac tamponade, or acute cardiac prosthetic valve dysfunction.

Reversible causes such as hypovolemia, hypoxia, cardiac tamponade, pneumothorax, acidosis, and hyperkalemia must be detected and corrected. Pharmacological support involves the use of intravenous epinephrine or atropine (the latter, however, is not effective in infra-Hisian AV block).

External cardiac pacing can be useful, although "asystolic" patients still have a poorer prognosis, regardless of the effectiveness of the stimulation [29].

3.5 Treatment

Sinus node dysfunction generally does not require treatment if asymptomatic. When needed, parasympatholytic drugs (atropine 0.5 mg intravenously, repeatable) can acutely increase the automaticity of the sinus node and improve sinus atrial conduction. Sympathomimetic drugs (theophylline per os) are less frequently used for long-term treatment because of their low efficacy and frequent side effects.

If there is any hemodynamic compromise, such as during an inferior myocardial infarction with right ventricle involvement, temporary endocardial pacing is preferred.

The presence of low cardiac output or heart failure, secondary to bradycardia, should be treated with permanent cardiac stimulation, preferably of the right atrium, to allow the spontaneous maintenance of AV conduction and avoid ventricular stimulation.

Supra-Hisian II AV block generally acutely responds to vagolytic drugs. As mentioned earlier, these drugs should be used with caution in suspected infra-Hisian blocks, which can be exacerbated by the increased cardiac rate and supra-Hisian conduction.

Sympathomimetic drugs such as isoproterenol, which are able to increase the automatism of ventricular "pacemaker subsidiary," can be used in the short term regardless of the level of AV block, in particular when it is likely that the block can be transient or if a temporary pacemaker cannot be immediately applied.

In third-degree AV block vagolytic drugs are not particularly effective, especially in the presence of infra-Hisian block, while sympathomimetic drugs provide valuable support in the acute phase while awaiting a permanent pacemaker.

3.5.1 Temporary Pacemaker

Temporary cardiac pacing is used acutely for critically ill patients who require immediate treatment, and sometimes in noncritical situations when patients are at high risk of developing advanced blocks or cardiac arrest.

An intracardiac approach is usually preferred, generally through the internal jugular vein, the subclavian vein, or the femoral vein.

Transcutaneous pacing, despite not having shown a significant benefit in patients with cardiac arrest [29], may be beneficial for the treatment of severe bradycardia as a "bridge" to a permanent pacemaker.

The main indication remains hemodynamically unstable acute myocardial infarction (Table 3.1). Drug treatment with digitalis, quinidine, class 1C antiarrhythmics, β-blockers, and some calcium antagonists can inhibit sinus node automatism up to the development of severe bradycardia, requiring, if associated with signs and symptoms of hemodynamic compromise, temporary pacing to allow pharmacological washout in safety [30].

Hypervagotonia can occur in carotid sinus hypersensitivity, in pharyngeal or gastroesophageal handling during intubation and/or endoscopy, and with parasympathomimetic or sympatholytic drugs. It is associated with three types of clinical response: the first, or cardioinhibitory, with hemodynamically significant bradyarrhythmia; the second, vasodepressor, with hypotension by peripheral vasodilation; and the third, which is a combination of the first two forms. Sometimes temporary pacing can be useful in intensive care, although these clinical situations are easily reversible with atropine [31].

Myocarditis can require temporary pacing because of transient involvement of the ventricular conduction system.

Complications of temporary pacing include capture failure caused by electrode dislocation (25 %), ventricular tachyarrhythmia related to local irritation of the electrode (10 %), pericarditis (5 %), and cardiac tamponade secondary to right atrium perforation (1 %).

Complications related to central venous access are air embolism (1 %), arterial trauma (1.2 %), pneumothorax (1 %), phlebitis (5.1 %), venous thrombosis (18-34 %), and bacteremia (50 %), although infection of the electrode is low [31].

Table 3.1 Therapeutic approach in conduction disturbances

Sinus atrial dysfunction	Asymptomatic patient: observation Symptomatic patient: atropine 0.5 mg iv repeatable up to 2 mg temporary/ permanent pacemaker
AV block	First-degree AV block: observation Second-degree AV block Type I asymptomatic: observation symptomatic – atropine 0.5 mg iv repeatable up to 2 mg Type II asymptomatic (advanced or Mobitz 2) or symptomatic → pacemaker implantation narrow QRS → atropine, if ineffective → pacemaker wide QRS → PM implantation Third-degree AV block a/symptomatic: narrow QRS → atropine, if ineffective → pacemaker wide QRS → PM implantation
AV block during myocardial infarction	Inferior myocardial infarction First-degree AV block → observation Second-degree AV block: Type I → atropine, if ineffective → temporary pacemaker Type II → temporary pacemaker Third-degree AV block → atropine, if ineffective → temporary pacemaker Anterior myocardial infarction: First-degree AV block → observation Second-degree AV block Type I atropine, if ineffective → temporary pacemaker Type II → temporary pacemaker Third-degree AV block → temporary pacemaker

3.5.2 Permanent Pacemaker

Sinus node dysfunction is a major reason for pacemaker implantation (about 46 % of implanted devices) [32] and has a long natural history with a good prognosis in the early stages, although with the progress of the disease and the appearance of symptoms the prognosis becomes less favorable [33].

Atrial pacing is preferable to ventricular pacing alone, although the advantage is reduced in the presence of thromboembolic events or the occurrence of AF [14, 34].

AV block is the second leading cause of pacemaker implantation. Permanent cardiac pacing has improved survival in patients with complete or advanced AV block, particularly if associated with symptoms (syncope or hemodynamic compromise) or in the presence of heart disease [35].

Asymptomatic first- and second-degree (type I) AV blocks do not require permanent cardiac pacing.

Symptomatic or second-degree AV block with slow escape rhythm (type I) and asymptomatic type II block are class II indications for permanent pacemaker implantation.

Complete or advanced AV block (both asymptomatic and symptomatic) and second-degree (type II) AV block, regardless of the level of block, are class I indications for a permanent pacemaker.

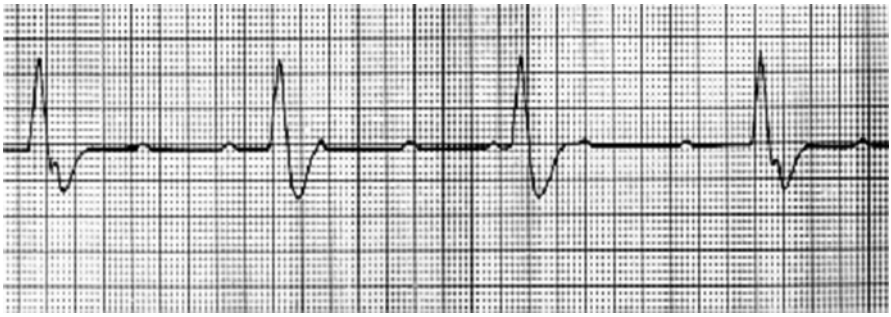

Fig. 3.4 III degree AV block with wide QRS (infra-hisian) escape

Care should be taken in cases of reversible AV block.

Survival in patients with complete AV block not treated with pacemaker implantation is 60 % at 1 year and 30 % at 5 years [35]. Patients treated with permanent pacemakers have been shown to have a 1-year survival comparable with that of the general population [36].

European and American guidelines for Pacemaker implantation [37, 38]
Class I indications
Sinus node dysfunction
Symptomatic sinus bradycardia or chronotropic incompetence
AV block
Second-degree AV block type II
Advanced AV block during sinus rhythm or AF
Complete AV block
Intraventricular block
Trifascicular block (RBBB alternating with LBBB, RBBB + LAHB alternating with RBBB + LPHB)
Bifascicular block (LBBB, RBBB + LAHB, RBBB + LPHB) and second-degree type II or third-degree AV block

Bibliography

1. Vàzquez Ruiz de Castroviejo E, Munoz Bellino J, Lozano Cabezas C, et al. Analysis of the frequency of cardiac arrhythmias and conduction disturbances from a health-care perspective. Rev Esp Cardiol. 2005;58(6):657–65.
2. Sutton R, Kenny RA. The natural history of sinus sick syndrome. Pacing Clin Electrophysiol. 1986;9(6):1110–4.
3. Mandel WJ, Jordan JL, Karagueuzian HS. Disorders of sinus function. Curr Treat Options Cardiovasc Med. 1999;1:179–86.
4. Alboni P, Brignole M, Menozzi C, Scarfò S. Is sinus bradycardia a factor facilitating overt heart failure? Eur Heart J. 1999;20:252–5.

5. Amar D. Strategies for perioperative arrhythmias. Best Pract Res Clin Anaesthesiol. 2004;18(4):565–77.
6. Heintz KM, Hollenberg SM. Perioperative cardiac issues: postoperative arrhythmias. Surg Clin North Am. 2005;85(6):1103–14.
7. Swart G, Brady Jr WJ, DeBehnke DJ, Ma OJ, Aufderheide TP. Acute myocardial infarction complicated by hemodynamically unstable bradyarrhythmia: prehospital and ED treatment with atropine. Am J Emerg Med. 1999;17:647–52.
8. Brady WJ, Swart G, DeBehnke DJ, Ma OJ, Aufderheide TP. The efficacy of atropine in the treatment of hemodynamically unstable bradycardia and atrioventricular block: prehospital and emergency department considerations. Resuscitation. 1999;41:47–55.
9. Schwartz B, Vermeulen MJ, Idestrup C, Datta P. Clinical variables associated with mortality in out-of-hospital patients with hemodynamically significant bradycardia. Acad Emerg Med. 2004;11:656–61; Delise P. ARITMIE. C.E.S.I. srl; 2004.
10. Demoulin JC, Kulbertus HE. Histopathological correlates of sick sino-atrial disease. Br Heart J. 1978;40:1384.
11. Shaw DB, Linker NJ, Heaver PA, Evans R. Chronic sinoatrial disorder (sick sinus syndrome): a possible result of cardiac ischemia. Br Heart J. 1987;58:598.
12. Oreto G. riconoscimento del blocco seno atriale di I grado all' elettrocardiogramma standard. G Ital Cardiol. 1982;12:762–6.
13. Rosenquist M, Brandt J, Schuller H. Atrial versus ventricular pacing in sinus node disease: a treatment comparison study. Am Heart J. 1986;111:292.
14. Andersen HR, Nielsen JC, Thomen PE, et al. Long term follow-up of patients from a randomised trial of atrial versus ventricular pacing for sick-sinus syndrome. Lancet. 1997;350:1210–6.
15. Zipes DP. Second degree atrioventricular block. Circulation. 1979;60:465–72.
16. Braunwald E, Zipes DP, Libby P, editors. Heart disease: a textbook of cardiovascular medicine. 6th ed. Philadelphia: WB Saunders; 2001.
17. Strasberg B, Amat-Y-Leon F, Dhingra RC, et al. Natural history of chronic second-degree atrioventricular nodal block. Circulation. 1981;63:1043–9.
18. Meytes I, Kaplinsky E, Yahini JH, Hanne-Paparo N, Neufeld HN. Wenckebach A-V block: a frequent feature following heavy physical training. Am Heart J. 1975;90:426–30.
19. Schweitzer P, Mark H. The effect of atropine on cardiac arrhythmias and conduction. Am Heart J. 1980;100:225–61.
20. Lee TH. Use of cardiac pacemakers and antiarrhythmic devices: ACC/AHA guidelines summary. In: Braunwald E, Zipes DP, Libby P, editors. Heart disease: a textbook of cardiovascular medicine. 6th ed. Philadelphia: WB Saunders; 2001. p. 810–4.
21. Kusomoto FM, Goldschlager N. Cardiac pacing. N Engl J Med. 1996;334:89–98.
22. Siegman-Igra Y, Yahini JH, Goldbourt U. Intraventricular conduction disturbances: a review of prevalence, etiology, and progression for 10 years within a stable population of Israeli adult males. Am Heart J. 1978;96:669.
23. Rowland E. SA node dysfunction, AV block and intraventricular conduction disturbances. Eur Heart J. 1992;13(Suppl H):130–5.
24. Scheinman MM, Peters RW, Modin G, et al. Prognostic value of infranodal conduction time in patients with chronic bundle branch block. Circulation. 1977;56:240–4.
25. Harrigan RA, Perron AD, Brady WJ. Atrioventricular dissociation. Am J Emerg Med. 2001;19:218–22.
26. Clemmensen P, Bates ER, Califf RM. Complete atrioventricular block complicating inferior wall acute myocardial infarction treated with reperfusion therapy. Am J Cardiol. 1991;67:225–30.
27. Priori SG, Aliot E, Blomstrom-Lundqvist C. Task Force on Sudden Cardiac Death of the European Society of Cardiology. Europace. 2002;4:3–18.
28. Myerburg RJ, Kessler KM, Castellanos A. Sudden cardiac death: epidemiology, transient risk, and intervention assessment. Ann Intern Med. 1993;119(12):1187–97.

29. Cummins RO, Graves JR, Larsen MP. Out-of-hospital transcutaneous pacing by emergency medical technicians in patients with asystolic cardiac arrest. N Engl J Med. 1993;328(19):1377–82.
30. Alpert MA, Flaker GC. Arrhythmias associated with sinus node disease. Circulation. 1971;43:836–44.
31. Silver M, Goldschlager N. Temporary transvenous cardiac pacing in the critical care setting. Chest. 1988;93(3):607–13.
32. Elmqvist R. Review of early pacemaker development. Pacing Clin Electrophysiol. 1978;1:535–6.
33. Mond HG, Irwin M, Morillo C, et al. The world survey of cardiac pacing and cardioconvertor defibrillators: calendar 2001. Pacing Clin Electrophysiol. 2004;27:955–64.
34. Lamas GA, Lee KL, Sweeney MO, et al. Ventricular pacing or dual chamber pacing for sinus node dysfunction. N Engl J Med. 2002;346:1854–62.
35. Edhag O, Swahn A. Prognosis of patients with complete heart block or arrhythmic syncope who were not treated with artificial pacemakers. A long term follow up study of 101 patients. Acta Med Scand. 1976;200:457–63.
36. Edhag O. Long term cardiac pacing: experience of fixed rate pacing with an endocardial electrode in 260 patients. Acta Med Scand. 1969;502:64.
37. Brignole M, et al. ESC guidelines on cardiac pacing and cardiac resynchronization therapy. Eur Heart J. 2013; doi:10.1093/eurheartj/eht150.
38. Epstein EE, et al. ACC/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities. J Am Coll Cardiol. 2008; doi:10.1016/j.jacc.2008.02.032.

Supraventricular Arrhythmias in Emergency

4

Elisabetta Bianco, Marco Bobbo, and Davide Stolfo

The term "supraventricular tachycardia" refers to arrhythmias requiring atrial or atrioventricular nodal tissue, or both, for their initiation and maintenance. Supraventricular tachycardias are often recurrent, occasionally persistent, and a frequent cause of visits to emergency departments (ED) [1].

The following chapter is organized in two main sections dedicated to physician working in ER and cardiologist in order to discuss arguments related to each specific expertise so that patients can receive assessment and treatment personalized on global clinical condition, not only on arrhythmia, in the ED.

4.1 What Physicians Working in ED Should Know

- Timing and modality to treat arrhythmias depends on patient's hemodynamic condition.
- Hemodynamic instability imposes emergent treatment with electrical cardioversion before diagnosis.
- In case of hemodynamic stability it is possible to think about diagnosis and choose best treatment.
- Hemodynamic instability depends on heart rate and on patients' features as age, underlying cardiomyopathy or chronic therapies, electrolyte disturbance, hyperthyroidism, hyperthermia, and drugs intoxication rather than on the type of arrhythmia [2, 3].

E. Bianco
Cardiovascular Department, Cattinara University Hospital, Trieste, Italy
e-mail: elisabetta.bianco@aots.sanita.fvg.it

M. Bobbo • D. Stolfo
Cardiovascular Department, Cattinara University Hospital, Trieste, Italy

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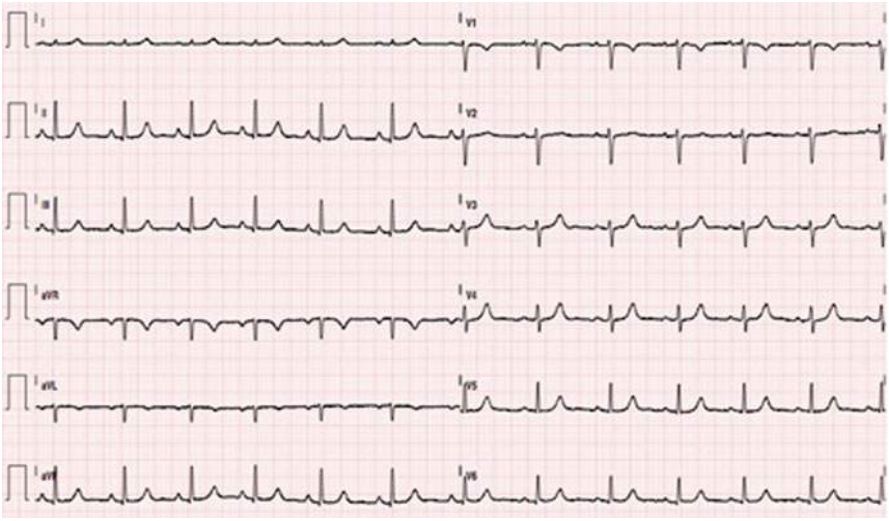

Fig. 4.1 ECG in normal sinus rhythm; with usual setting (paper speed 25 mm/s) 1 mm equals with 40 ms interval

Supraventricular arrhythmias are [4]:

1. Sinus tachycardia
2. Atrial flutter (typical or atypical)
3. Atrial fibrillation (AF)
4. Atrioventricular nodal reentrant tachycardia (AVNRT)
5. Atrioventricular reciprocating tachycardia (AVRT)
6. Atrial/junctional tachycardia (AT)

ECG during arrhythmias differs from normal ECG observed in sinus rhythm. So, first of all, normal ECG must be known [5, 6] (Fig. 4.1):

- Each QRS complex is preceded by a *P wave* with a rate between 60 and 100 bpm with the exception of newborns and infants in whom normal HR is higher (150–230 bpm in newborn) [7].
- P wave is positive in leads I–II and aVL–aVF and in precordial leads from V2–3 to V6, is negative in aVR and can be negative in III, and is biphasic in V1–V2; P wave lasts 120 ms (3 mm).
- Between P wave and QRS complex, there is an isoelectric region (flat line on ECG) which lasts 120–200 ms: the *PR interval.*
- *QRS is narrow* (<110 ms).

While approaching a patient with symptomatic supraventricular tachycardia, physicians must evaluate the patient's clinical status to identify potential reversible causes of tachycardia and to assess the degree of instability and understand if it is related to the tachycardia itself.

Arterial blood pressure, oxygen saturation, electrolyte, and hemoglobin must be evaluated; ECG monitoring and intravenous access must be established.

If possible a 12-lead ECG should be performed during the arrhythmia (and after its interruption) [2].

4.1.1 Patients in Unstable Condition

If the patient demonstrates rate-related cardiovascular compromise with signs and symptoms such as acute altered mental status, ischemic chest discomfort, acute heart failure, hypotension, or other signs of shock suspected to be due to a tachyarrhythmia, proceed to immediate synchronized electrical cardioversion (after sedation of the patient if conscious); provide oxygen if oxygen saturation is below 94 % [2]:

- Narrow regular tachycardia → biphasic synchronized direct current shock 50–100 J
- Narrow irregular tachycardia → biphasic synchronized direct current shock 120–200 J
- Wide regular tachycardia → biphasic synchronized direct current shock 100 J
- Wide irregular tachycardia → biphasic NOT synchronized direct current shock 200 J

If there's no response to the first shock, it may be reasonable to increase the dose in a stepwise fashion.

Cardioversion with monophasic waveforms should begin at 200 J and increase in stepwise fashion if not successful [2].

In newborn, use 0.5 → 2 J/kg [3].

Heparin (low molecular weight or unfractionated heparin) should be administered before cardioversion in case of atrial flutter or atrial tachycardia and after it, depending on patients' features (CHA_2DS_2VASc – HAS BLED) [8].

In patients with implanted pacemaker or defibrillator pads, it should be positioned 8 cm from the device battery; anteroposterior paddle positioning and biphasic energy are recommended [2, 4, 8].

4.1.2 Patients in Stable Condition

The initial assessment should:

1. Distinguish between narrow and wide complex tachycardia.
2. Determine whether the rhythm is irregular or regular (<10 % of variation in cycle length).
3. Consider the rapidity of onset.
4. Identify sinus tachycardia (the theoretical upper rate is 220bpm minus the patient's age in years).

Supraventricular tachycardias have narrow QRS in 90 % of cases. Wide QRS complex could be due to pre-excitation through an accessory pathway or to bundle-branch block or to pacemaker stimulation. Previous ECG and ECG after termination of tachycardia can help to make the diagnosis.

While considering the patient's history, the clinician should assess the duration and frequency of episodes, the mode of onset, and possible triggers (including the intake of alcohol, caffeine or other drugs) as well as previous cardiac or other diseases or previous ECGs.

Supraventricular tachycardias have a sudden onset and termination, in contrast to sinus tachycardias or atrial tachycardias, which accelerate and decelerate gradually; however, some patients do not perceive the sudden onset of supraventricular tachycardia; furthermore, sometimes, they describe sudden onset and gradual offset due to reactive sinus tachycardia after termination of paroxysmal SVT. It may be misdiagnosed as panic disorder [5, 9].

Physical examination during episodes may reveal the "frog sign" which is a prominent jugular venous A waves due to atrial contraction against the closed tricuspid valve. After sinus rhythm is restored, physical examination is usually normal, but a careful examination is warranted to rule out evidence of structural heart disease [11, 12].

4.1.3 Management

4.1.3.1 Narrow QRS

If narrow QRS is present, atrioventricular conduction is certainly entirely through atrioventricular node. That's why maneuvers that increase vagal tone and block atrioventricular conduction are safe: they can interrupt tachycardias depending on AV node conduction (TRNAV or orthodromic TRAV) and can slow down those which don't depend on AV node conduction (atrial flutter or atrial fibrillation). They also interrupt focal tachycardia due to enhanced automaticity.

Physicians have these possibilities:

- *Vagal maneuvers* (Valsalva, carotid sinus massage, facial immersion in cold water, and bearing down). The efficacy is about 25 % [13]. Remember that carotid sinus massage is contraindicated in case of carotid stenosis.
- *Adenosine:* very short-acting endogenous nucleotide (<10 s). It blocks atrioventricular nodal conduction and terminates most of atrioventricular nodal reentrant tachycardias and atrioventricular reciprocating tachycardias as well as up to 80 % of atrial tachycardias [14–16]. Since it may also have proarrhythmic effect (atrial fibrillation in 12 % of patients), it must be administered under ECG monitoring with an accessible defibrillator.

  Common side effects include chest tightness, flushing, and a sense of dread. Avoid in asthmatic patients since it can produce bronchospasm; patients with autonomic failure and with cardiac transplant or patient chronically treated with

antiarrhythmic drugs are more sensitive to adenosine [17]; that's why doses must be halved.

Way of administration and doses: 0.1–0.2 mg/kg (usually 6–12 mg) through peripheral vein; start with 6 mg in a rapid flush immediately followed by 20 ml of saline flush. If there is no effect, try again with 12 mg. Same dose for newborn.

- *Verapamil-diltiazem*: Intravenous verapamil and diltiazem are effective in interrupting tachycardias or can slow down AV conduction, but can cause hypotension thus are not recommended as first choice in the emergency setting [18]. They are contraindicated in heart failure and in newborn because of risk of cardiac arrest or atrioventricular dissociation [3, 19].
- *Electrical cardioversion*: best choice for atrial fibrillation and atrial flutter (typical o atypical) with onset within 48 h or in anticoagulated patients [8]. Biphasic synchronized shock (50→200 J; 0.5→2 J/kg in infants) is recommended in sedated patient. Before cardioversion, a heparin bolus is recommended in all patients not yet anticoagulated. If there is no efficacy or there is arrhythmia recurrence, pharmacological therapy can be added.

Remember that:

- Sinus tachycardia is usually reactive to other causes so identification and treatment of them are required. No specific drug treatment is usually necessary.
- Atrial fibrillation and atrial flutter can give rise to endocardial thrombus which can cause stroke or thromboembolism: the risk is higher after 48 h of persistent tachycardia and in patients with some specific features ($CHA_2DS_2VASc > 1$). For this reason, anticoagulation (for at leat 3 weeks) is mandatory before cardioversion (or, in alternative, perform trans-esophageous echo to rule out intracardiac thrombi); in case of arrhythmias lasting more than 48 h or non-datable; nevertheless, heparin administration before cardioversion is preferable in all patients. For more details, see specific guidelines [16, 19–21].
- Patients with uncomplicated tachycardias and without cardiomyopathy can be discharged after sinus rhythm restoration; cardiological evaluation can be postponed.
- Sodium channel blockers increase both pacing threshold and the energy required to defibrillation [22, 23].
- Being genetic setting unknown, the effect of an antiarrhythmic drug given for the first time is *never* predictable. That's why administration of antiarrhythmic medications for the first time must be done under ECG monitoring lasting for at least the drug's half-time; after discharge, if chronic therapy is set up, ECG must be checked at the steady state (five half-times) (Fig. 4.2).

4.1.3.2 Wide QRS

Wide QRS can be due to preexisting or frequency-dependent bundle-branch block or to conduction over an accessory pathway. Nevertheless, the majority of wide QRS complex tachycardia has ventricular origin. If the patient is in stable hemodynamic condition, it is better to refer him/her to a specialist for diagnosis and treatment.

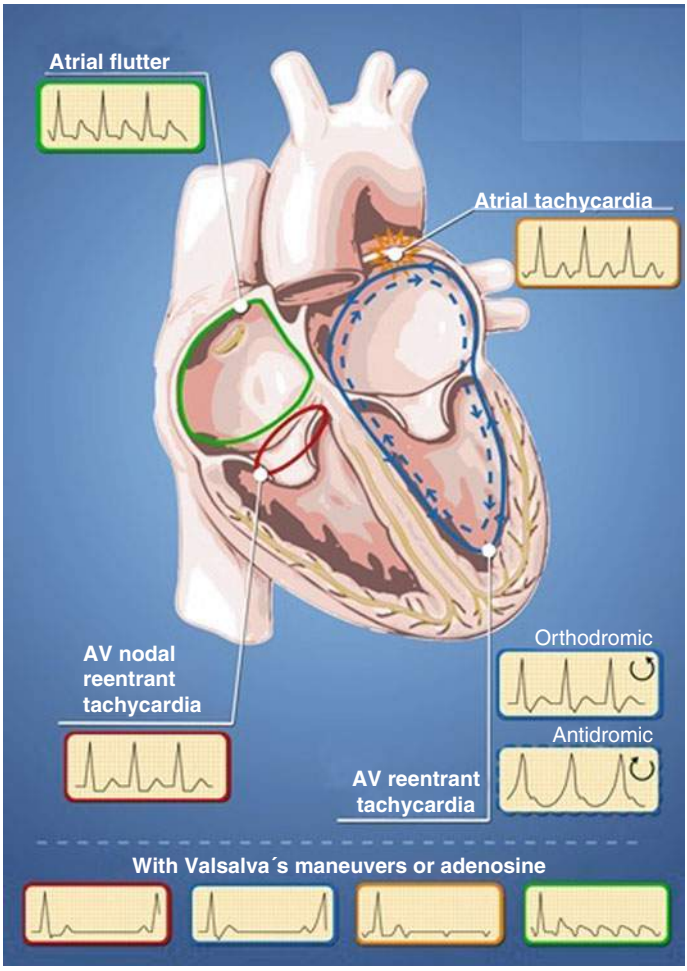

Fig. 4.2 Supraventricular tachycardias (Data from Delacrétaz [12])

4.2 What Cardiologists Should Know

Cardiologist should evaluate all patients with hemodynamic instability related to tachycardias after their stabilization with cardioversion in order to exclude or confirm the presence of cardiomyopathy, to decide the best medical treatment, and to decide for hospitalization or discharge and follow-up.

Non-urgent cardiological examination within few weeks can be reserved to uncomplicated tachycardias, in absence of cardiomyopathy, especially in case of preexisting diagnosis.

Cardiologist should know mechanisms underlying arrhythmias and modality of action of antiarrhythmic drugs in order to choose and combine them, to maximize

efficacy and minimize proarrhythmic and side effects [22, 23], and to remove precipitating features as ischemia and electrolyte imbalance.

For this reason, the next part of the chapter is dedicated to pharmacology of antiarrhythmic drugs and basic electrophysiology.

At the end of the chapter, a scheme of therapies for each arrhythmia is provided.

4.2.1 Basic Electrophysiologic Principles

Every heart contraction is driven by inversion of cell potential from negative to positive. At the cell membrane site, this potential variation from negative to positive (depolarization) and back to negative (repolarization) is called action potential (AP). This is possible thanks to the activation in sequence of different channels driving ion currents (I) inward and outward the cell; every channel is designed to allow passage of its own specific ion and to do it only during a specific phase of AP (some channels open only at negative potential, other at positive). Potassium (K^+) is mainly inside the cell, while sodium (Na^+) is outside and calcium (Ca^{++}) is both outside and stored inside the cell; sodium and calcium are linked to one another. To generate cell depolarization, a specific potential threshold must be reached.

Action potential has been divided in five phases:

- 0 → Depolarization
- 1 → Fast repolarization
- 2 → Plateau
- 3 → Terminal repolarization
- 4 → Resting

During the first phase of action potential, cell membrane is depolarized, and no external stimulus can modify it (absolute refractoriness); during repolarization, external factors can excite the cell with "extra-AP" (relative refractoriness).

Propagation of action potential from a cell to another is called conduction and is characterized by a speed and a safety factor.

Action potential has different morphologies in different tissues (sinus node, AV node, atrioventricular cells). This is due to different kinds of channels and explains the heterogeneous effects of antiarrhythmic drugs on each cardiac tissue.

For example, AV node cell depolarization is mainly calcium mediated, while atrial and ventricular depolarization is sodium mediated; automaticity of sinus node is due to specific channels (HCN) which are permeable to both sodium and potassium and open only at very negative potentials (hyperpolarization).

Function alteration of ion channels (pathological, heart rate or drug mediated) or modifications of ion concentrations modify action potential in terms of duration, velocity of depolarization, or repolarization and determine abnormal rhythm propagation [5, 21].

4.2.2 Mechanisms of Arrhythmias

Arrhythmias are divided into focal and reentrant.

4.2.2.1 Focal

Focal supraventricular tachycardias are defined as an activation starting rhythmically from a small area in atria or AV junction and spreading out centrifugally. Focus can be single or multiple. Mechanism underlying focal tachycardias is automaticity, triggered activity, and micro-reentrant.

- *Triggered activity*: abnormal fluctuations of action potential occurring after or during the repolarization (phase 2–3 or 4 of action potential), which are able to depolarize cell membrane to the depolarization threshold; differently from automatic foci, they are linked to the previous action potential. Three kinds of triggered activities are known (Fig. 4.3):

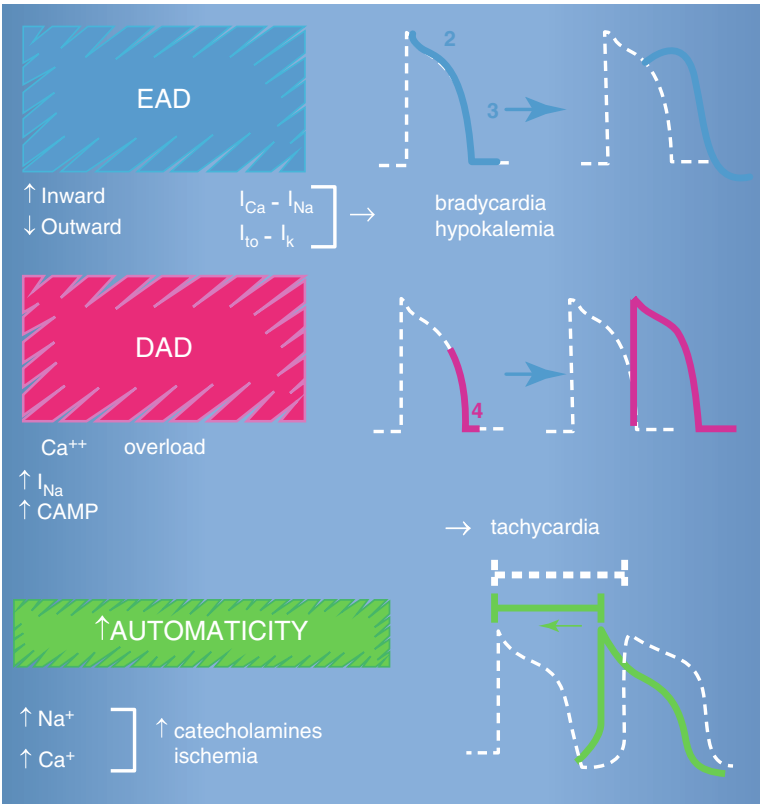

Fig. 4.3 Effects of early after depolarization (EAD), delayed after depolarization (DAD), and automaticity on action potential

(a) *Early after depolarization (EAD)* → occurring during repolarization (phases 2–3 of action potential), due to decreased potassium conductance (typically bradycardia, hypokalemia, or class III antiarrhythmic drugs). They are suppressed by factors that increase potassium outward current (*overdrive – tachycardias*, *K+ channels activators*) or suppress sodium/calcium inward currents (e.g., *magnesium*).
(b) *Delayed after depolarization (DAD)* → occurring during diastolic phase of action potential (phase 4) as consequence of Ca^{++} intracellular overload and Ca^{++} sparks due to increase of I_{Na} (ischemia or digoxin intoxication) or cAMP after beta-stimulation (tachycardia). They are suppressed by *beta-blockers*, *calcium channel blockers*, *vagal stimulation*, or *adenosine*.
(c) *Enhanced automaticity:* positive ionic influx (mostly inward calcium current) during phase 4 (diastolic) depolarization (normal automaticity is driven by sodium current while pathological by calcium). Beta-adrenergic stimulation enhances calcium current and facilitates these arrhythmias. Automatic tachycardias are the most common kind of focal arrhythmias and are characterized by sudden onset with a "warm-up" period. *Vagal stimulation*, *adenosine*, *beta-blockers*, or *calcium channel blockers* may suppress them.

Focal tachycardias are:

- *Atrial tachycardias* → P wave is generally visible with a morphology different from sinus one; 1:1 AV ratio is expected; heart rate can be very variable.
- *Inappropriate sinus tachycardia* → normal P wave morphology; 1:1 AV ratio; heart rate > 100 bpm not related to physiological needs.
- *Junctional tachycardia* → QRS not preceded by P; P wave can be visible after QRS, with morphology typical of AV junction origin (negative in DII–DIII and aVF, positive in V1 or not visible); similar to nodal or atrioventricular reentrant tachycardias; typical of infants.
- *Polymorphic atrial tachycardia/atrial fibrillation* → recurrent paroxysms of polymorphic premature beats starting with warm-up and degenerating in disorganized atrial activity; P wave is polymorphic and not always visible (during AF, sometime P is visible only in V1–V2 due to organization of atrial activity around tricuspid annulus).

4.2.2.2 Reentry

Reentry is a self-perpetuating circuit in which, during a single cardiac cycle, areas still depolarized and areas repolarized coexist. Reentry can be functional or anatomical. If the reentry circuit is >2–3 cm, it's classified as a macro-reentrant; if the reentry circuit is <2–3 cm, instead it's classified as micro (focal tachycardia) [25].

To have a reentry, the following features are required: unidirectional conduction (one way block usually due to extra beat), areas of different conduction rate (short and fast), and shortening of refractory period. Common triggers of reentry

are sympathetic or parasympathetic stimulation, premature beats, and myocardial ischemia.

Wave length (WL) is the product of conduction velocity (CV) and refractory period (RP); WL is theoretical and is always shorter than the real one, so there is an excitable gap (EG) inside the circuit. Reentry has a wave front, a tail, and an excitable gap between them. Excitable gap is necessary to perpetuate reentry.

Factors eliminating the excitable gap block the reentrant circuit making the wave front crash on the tail. There are two ways to obtain it: to *increase the refractory period* (with potassium current blockers or sodium current blockers with slow kinetics) or to *increase the conduction velocity*. In addiction to drugs, the excitable gap can be suppressed stimulating the circuit at a rate just faster than the arrhythmia (overdrive pacing), possible in patients with dual chamber (or atrial) pacemakers.

Ablation can eliminate reentrant circuits by targeting areas of slow conduction that sustain circuit (called isthmus of the tachycardia) and blocking them (creating a permanent lesion with bidirectional block). When ablation lesion is incomplete, it can create an area of slower conduction that is potentially proarrhythmic.

Sometimes, the degree of increase of refractory period brought by drugs is not enough to interrupt tachycardia even at therapeutic concentration: this is more frequent in large anatomic circuit and with drugs that slow down propagation velocity as INa^+ blockers. In this situation arrhythmia became more stable and resistant to therapies. Examples are AVNRT slow/slow or permanent junctional reciprocating tachycardia (PJRT) after calcium or beta-blockers or transformation of atrial fibrillation (small circuit) in atrial flutter (large circuit) after flecainide or propafenone administration.

Reentrant tachycardias are:

- *Atrial flutter (typical and atypical)* → organized atrial rhythm; typical flutter is characterized by negative/positive sawtooth waves (F wave) in leads II, III, and aVF and positive in V_1; in atypical flutter, F waves have different axis depending on circuit; atrial heart rate is between 250 and 350 bpm (but can vary in patient previously treated with ablation or in patients taking antiarrhythmic drugs). In typical atrial flutter, circuit is counterclockwise around tricuspid annulus; slow conduction is between tricuspid annulus and inferior vena cava and conduction block along the crista terminalis and Eustachian ridge.
- *Post-incisional atrial tachycardia* → organized atrial activity due to reentrant around a scar; typical of patients with a history of cardiac surgery or catheter ablation.
- *AVNRT* → P wave arising from junction (negative in DII–DIII and aVF, positive in V1) with RP <PR; two limbs are slow and fast nodal pathways. Reentrant circuit is most of the time down through slow pathway and back through fast pathway (slow/fast), and P wave is visible as a pseudo-r′ in V1 or a pseudo-s′ in inferior leads (P can be not visible); in atypical AVNRT, circuit is down through slow or fast pathway and back through slow (slow/slow, fast/slow), and P wave is separated from QRS; RP >PR). The circuit is inside the AV node so that the

onset of atrioventricular block during tachycardia doesn't interrupt it (AVNRT can have 2:1 AV ratio).

- *AVRT* → reentrant circuit across AV node and accessory truck. It's called orthodromic when it passes from the atrium to the ventricle through AV node and back from the ventricle to the atrium through accessory pathway (RP >PR); it's called antidromic when anterograde wave front is through accessory pathway. The atria and ventricles are part of the circuit, and AV dissociation is not possible without interrupting tachycardia. Presence of pre-excitation is not diagnostic of AVRT (accessory pathway can be a "bystander" during AVNRT or atrial tachycardia, meaning that ventricles are partially or totally activated through the accessory pathway, which nevertheless is not part of the reentry circuit causing the arrhythmia). Evidence of tachycardia cycle length (=RR) augmentation of more than 35 ms (more and less 1 mm with 25 mm/s paper speed) in case of ipsilateral bundle brunch block is diagnostic of AVRT [24], because the ipsilateral block causes a lengthening of the circuit.
- *Permanent junctional reciprocating tachycardia (PJRT) – Mahaim tachycardia* → are rare AVRT through accessory pathway with particular features. PJRT is due to a septal accessory path with only retrograde conduction and decremental properties; it determines incessant narrow QRS tachycardia. Mahaim pathway is an accessory path with decremental properties generally right sided (from anterolateral atrial portion of tricuspid annulus to right ventricle or right bundle); in sinus rhythm, there is typically little or no pre-excitation; during antidromic tachycardia, the QRS is wide (150 ms), with left bundle block morphology, transition after V4.

4.2.3 Antiarrhythmic Drugs

Antiarrhythmic drugs are channel or receptor blockers. They bind specific sites on protein and modify its function with an affinity that varies on the ion channel state (open-inactivated-resting). Furthermore each drug has a specific rate of recovering from block which is a constant time ($T_{recovery}$).

This is why antiarrhythmic drugs that bind open channel, untie during resting state, and have long $T_{recovery}$ (as flecainide – 11 s) reach the steady state earlier having greater effect when a higher percentage of cells are depolarized as during ischemia or high heart rates (direct use dependence). Those drugs that, instead, own affinity for inactivated/resting channels (amiodarone or lidocaine) and own a very short $T_{recovery}$ (lidocaine – 0.1 s) have a greater effect (anti- and proarrhythmic!) under the condition that prolong action potential duration (as bradycardia-hypokalemia) and on rapidly driven tissue (ventricular tachycardia).

Antiarrhythmic drugs have been divided in four classes according to their main effect on action potential (Vaughan Williams classification) [25, 26]. However mostly all of them share properties among multiple classes (quinidine below to I°

class but have also III° class properties). Moreover, ivabradine, digoxin, and adenosine don't belong to any of the four classes (Table 4.1) (Fig. 4.4):

- INa+blockers (I class): shorten action potential, reduce propagation velocity, and act on refractoriness depending on kinetics (flecainide increase relative refractory period; lidocaine no); suppress EAD; reduce inotropism.
- Ica++ blockers (class IV): reduce propagation velocity; suppress abnormal automaticity, EAD, and DAD; reduce inotropism. All have direct use dependence.
- IK+ (class III): lengthening of action potential and of absolute refractory period. Increase inotropism. Have inverse use dependence. Proarrhythmic trough induction of EAD (during hypokalemia/bradycardia)

Key Messages

Remember that:

- *Electrolytes MUST be known, and their alterations MUST be correct before administering any antiarrhythmic drug!*
- Effect of antiarrhythmic drugs can be very different (and very dangerous) among patients with altered function of ion channels (channelopathies, myocardial ischemia) or variants of metabolic capacity or concomitant therapies [27–29]. Therefore, in patients with supraventricular arrhythmias and altered basal ECG (conduction disturbance, repolarization abnormalities), it is important to exclude those abnormalities and choose the correct therapy. When a channelopathy is suspected, further evaluation must be organized [30]; the decision about hospitalization or delayed ambulatory evaluation depends on the risk of arrhythmic death (arrhythmic instabilities, predisposing factors).
- Being genetic setting usually unknown, the *effect of an antiarrhythmic drug given for the first time is never predictable* [31]. That's why the first administration of any antiarrhythmic medications should be done under ECG monitoring lasting for at least the drug's half-time; after discharge, if chronic therapy is set up, ECG must be checked at steady state (five half-times).
- Ic are *effective on accessory pathway*.
- Ic are *poorly effective on atrial macro-reentrant tachycardias (flutter o post-incisional)*; they slow atrial cycle length without interrupting tachycardia; if AV nodal conduction is enhanced (e.g., during exercise), they can promote 1:1 AV conduction. For this reason it's better to associate an AV blocker (Ca^{++} or Na^{+} blocker) to a Ic drug. Vernakalant is not effective on atrial flutter.
- Ic *have a marked negative inotropic effect*; if administered in rapid bolus, it causes broad hypotension even in people without cardiomyopathy so they must be administered under arterial pressure monitoring.
- *Calcium-blockers are dangerous in newborn* because of risk of cardiac arrest and are contraindicated in this setting [3, 19].
- *In newborn and children, beta-blockers are highly effective* especially if combined with amiodarone [32, 33]. Procainamide has also been demonstrated to be more effective than amiodarone in sinus rhythm conversion of pediatric SVT [34].

Table 4.1 The effects, doses, side effects, and contraindications of antiarrhythmic drugs

CLASS	DRUG		INTRAVENOUS	MAINTENANCE (day)	$T_{1/2}$	ELIMINATION	SIDE EFFECTS	CONTRAINDICATIONS
I_a ↓ phase 0 ↓ conduction ↑ repolarization	DISOPYRAMIDE	↓ Na^+,K^+ ↓ M_2	//	400-800 mg →300/400 mg	4-8h	renal/hepatic	↑QT, TdP,gastrointestinal ailment,vasculitis, glaucoma, hypoglycemia	SA dysfuncion, bifascicular BBB, BAV II-III, severe heart failure, hypokalemia, CrCl <20 ml/min
	PROCAINAMIDE	↓ Na^+,K^+	30 mg/min (max 1 g) + 1-4 mg/min	250-1000 mg	3-4h	hepatic, renal	Hypotension, QRS widening, TdP	Severe heart failure, QRS widening > 50% basal
	QUINIDINA	↓ Na^+,K^+ ↓ M_2 α,β	//	275mg(x3) 250-500mg (bid)		hepatic, renal	↑QT, diarrhea	
I_b ↓ Na^+ in phase 2 (effective on VT in abnormal tissue)	Lidocaine	↓ Na^+	//	//	//	//	//	//
	Mexiletin	↓ Na^+	//	//	//	//	//	//
	Tocainide	↓ Na^+	//	//	//	//	//	//
	Phenytoin	↓ Na^+	//	//	//	//	//	//
I_c ↓ phase 0 ↓ conduction	FLECAINIDE	↓ Na^+,K^+	2 mg/Kg in 10′	3-5 mg/Kg	12-20h	renal	Hypotension, negative inotropic effect, QRS widening, SA,AV block, dizziness, visual impairment, gastrointestinal ailment	Heart failure, Brugada Syndrome, LQT3, ischemic heart disease
	PRPPAFENONE	↓ Na^+, β	2 mg/Kg in 10′	10 mg/Kg	variable	hepatic		
	MORICIZINA	↓ Na^+						
II **β-blockers**	ESMOLOL	↓ β	500 γ/Kg 1′ →20-200 γ /Kg/min	//	8	esterases - renal	Bradicardia Heart block Hypotension, Negative inotropic effect Bronchospasm	Asthma II-III degree AV block Heart failure
	METOPROLOL	↓ $β_1$	5 mg in 2 ′→ max 15 mg in 15′	50-200 mg	variable	hepatic		
	PROPANOLOL	↓ $β_1$- $β_2$	0,15-0,2 mg/Kg in 2′	80-240 mg	4 h	hepatic		
	ATENOLOL	↓ $β_1$	2,5 mg in 1′ →every 5′, max 10 mg	25-100 mg	6 h	renal		
	NADOLOL	↓ $β_1$- $β_2$	//	20-80 mg	20 h	renal		
	BISOPROLOL	↓ $β_1$	//	2,5-10		hepatic/renal		
III ↑repolarization	AMIODARON	↓K^+↑Na^+, Ca^{++},β	5 mg/Kg in 20′-2h →15 mg/Kg/24h	20 mg/Kg/week	240h	hepatic/renal	Skin discoloration, dyshyroidism, gastrointestinal up- set, hepatotoxicity, corneal deposits, tremor, optic neuropathy, pulmonary toxicity	LQT,hypokalemia Severe heart failure
	SOTALOL	↓ K^+, β	//	3-5 mg/Kg (bid)	8 h	renal	↑QT, TdP, bradicardia, heart block, hypotension, negative inotropic effect, bronchospasm	LQT, hypokalemia, severe heart failure asthma, renal failure
	DOFETILIDE	↓ K^+	//	1 mg	10h	renal/hepatic	↑QT, TdP (start in hospital)	LQT, ↓ K^+, SA dysfuncion, AV II-III
	IBUTILIDE	↓K^+,↑Na	1 mg in 10′x2 (if <60Kg 0,01 mg/Kg)	//	2-12h	renal	↑QT, TdP,bradicardia	LQT, ↓ K^+,Mg^{++}, SA dysfuncion, BAV II-III, recent MI
	DRONEDARON		//	800 mg		renal		LQT, -SA dysfuncion, BAV II-III, heart failure
	VERNAKALANT	↓ K^+,Na^+	//	//	//	//	//	//
IV **Ca^{++}Antagonist**	VERAPAMIL	↓ Ca^{++}	5mg/KginS′→max10mg	120-480 mg	3-7 h	hepatic	Bradicardia, SA,AV block, hypotension, negative inotropic effect	II-III degree AV block, heart failure, newborn
	DILTIAZEM	↓ Ca^{++}	0,25-0,35 mg/Kg in 2′ → 5-15 mg/h	180-360 mg	4h	hepatic		
	ADENOOSINE	↓A_1	0,1-0,2 mg/Kg	//	<10″	esterases	bronchospasm, flushing	asthma
	DIGOXIN	↑M_2	1 mg/12-24 h	0,125-0,375 mg	36h	renal	tachyarrhythmias, SA,AV block	hypokalemia, severe renal disease
	IVABRADINE	↓	//	2,5-7,5 mg (bid)	12 h	renal	bradicardia, bradicardia-mediated VT and AF, phosphenes, headhache	bradyarrhythmias, LQT, severe heart failure

Different colors identify the four classes of Vaughan Williams classification; in *black* drugs not belonging to any class. Drugs without indications for supraventricular tachycardias are only mentioned

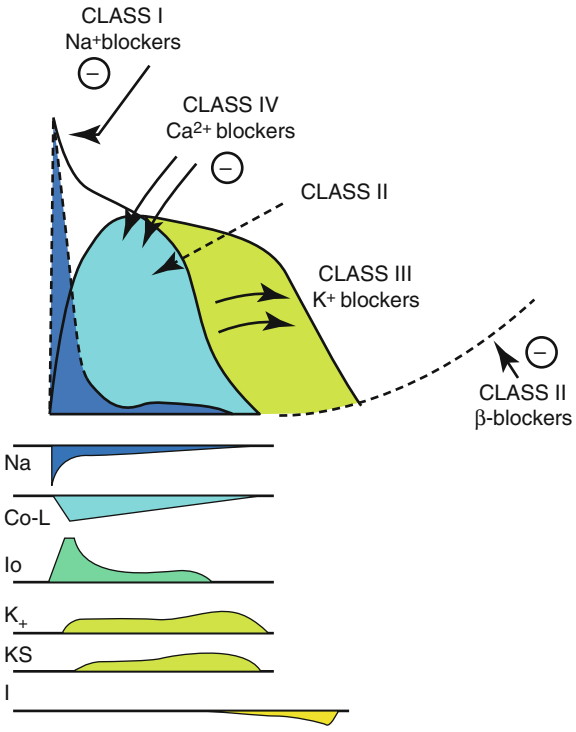

Fig. 4.4 Major effects of antiarrhythmic drugs on action potential

- *Newborn and children aren't* "small adults": they have different metabolism that MUST be known (usually they are better metabolizer than old people); drug's doses must be setup on body weight.
- Amiodarone is effective on different substrates due to its multiple antiarrhythmic affect (class from I to IV).
- *Risk of EAD due to a drug that blocks IK is reduced by addition of a INa/ICa blocker* (as quinidine + verapamil).
- *Supraventricular tachycardias can be associated with troponin level and/or ST abnormalities that rise in absence of coronary artery disease* [35, 36]. Suspicion of coronary syndrome must be based on risk factors, symptoms, ECG, or echocardiographic alteration.
- *Catheter ablation is safe and highly effective in curing supraventricular arrhythmias permanently*. It is a reasonable option of antiarrhythmic therapies in most of tachycardias and must be considered and proposed to patients [4, 37, 38] (Fig. 4.5).

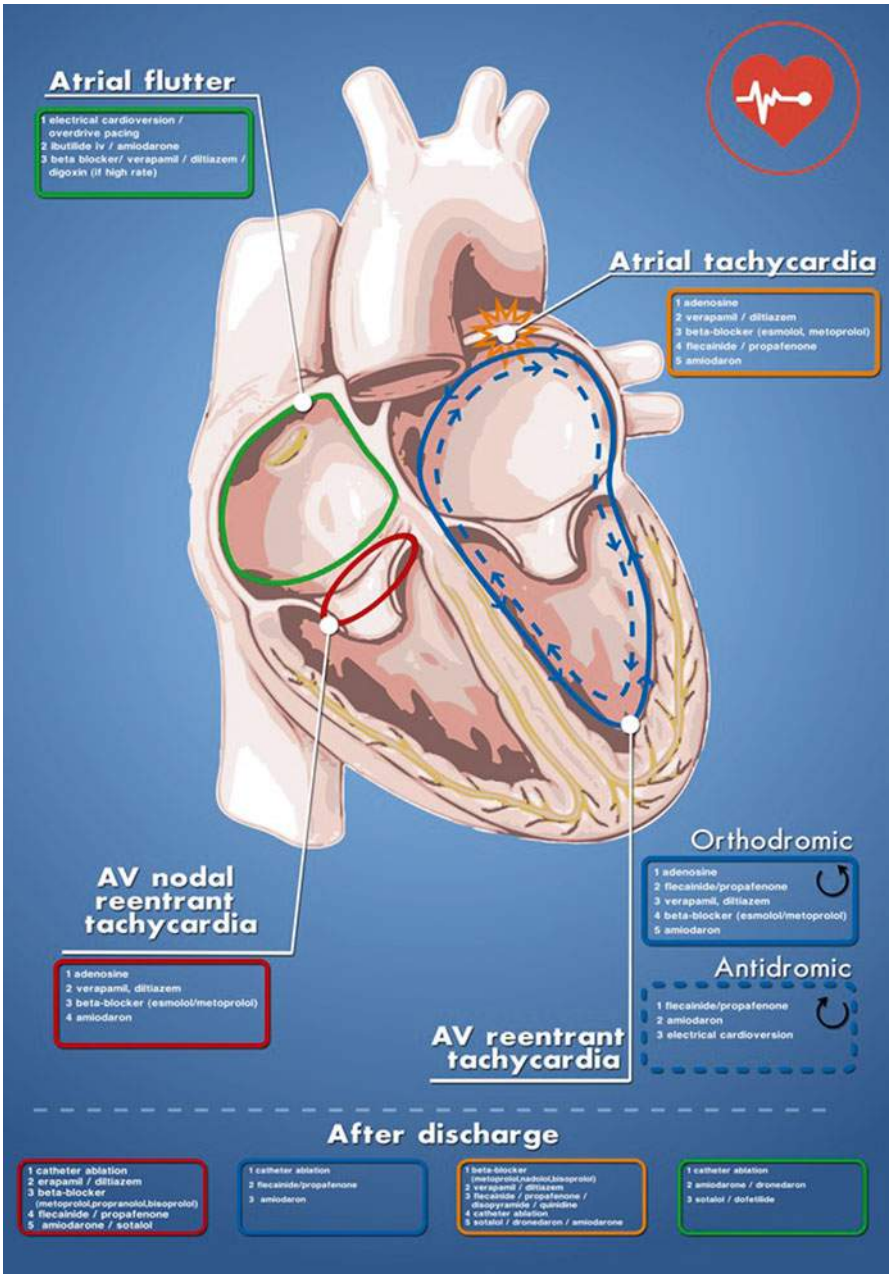

Fig. 4.5 Mechanisms, acute and long-term treatment of major supraventricular arrhythmias due to reentry circuits

References

1. Orejarena LA, Vidaillet Jr H, DeStefano F, et al. Paroxysmal supraventricular tachycardia in the general population. J Am Coll Cardiol. 1998;31:150–7.
2. Neumar RW, Otto CW, Link MS, et al. Part 8: adult advanced cardiovascular life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2010;122 Suppl 3:S729–67.
3. Kleinman ME, Chameides L, Schexnayder SM, Samson RA, Hazinski MF, Atkins DL, Berg MD, de Caen AR, Fink EL, Freid EB, Hickey RW, Marino BS, Nadkarni VM, Proctor LT, Qureshi FA, Sartorelli K, Topjian A, van der Jagt EW, Zaritsky AL. Part 14: pediatric advanced life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2010;122(18 Suppl 3):S876–908.
4. Blomström-Lundqvist C, Scheinman MM, Aliot EM, Alpert JS, Calkins H, Camm AJ, Campbell WB, Haines DE, Kuck KH, Lerman BB, Miller DD, Shaeffer CW, Stevenson WG, Tomaselli GF, Antman EM, Smith Jr SC, Alpert JS, Faxon DP, Fuster V, Gibbons RJ, Gregoratos G, Hiratzka LF, Hunt SA, Jacobs AK, Russell Jr RO, Priori SG, Blanc JJ, Budaj A, Burgos EF, Cowie M, Deckers JW, Garcia MA, Klein WW, Lekakis J, Lindahl B, Mazzotta G, Morais JC, Oto A, Smiseth O, Trappe HJ, European Society of Cardiology Committee, NASPE-Heart Rhythm Society. ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias – executive summary. a report of the American college of cardiology/American heart association task force on practice guidelines and the European society of cardiology committee for practice guidelines (writing committee to develop guidelines for the management of patients with supraventricular arrhythmias) developed in collaboration with NASPE-Heart Rhythm Society. J Am Coll Cardiol. 2003;42(8):1493–531.
5. Bonow R, Mann D, Zipes D, Libby P. Braunwald's heart disease. 9th ed. Philadelphia: Elsevier Inc; 2012.
6. Rijnbeek PR, Witsenburg M, Schrama E, Hess J, Kors JA. New normal limits for the paediatric electrocardiogram. Eur Heart J. 2001;22(8):702–11.
7. Schwartz PJ, Garson Jr A, Paul T, Stramba-Badiale M, Vetter VL, Villain E, Wren C. Linee guida Linee guida per l'interpretazione dell'elettrocardiogramma neonatale Task Force della Società Europea di Cardiologia. Ital Heart J Suppl. 2003;4(2):138–53.
8. Dankner R, Shahar A, Novikov I, Agmon U, Ziv A, Hod H. Treatment of stable atrial fibrillation in the emergency department: a population-based comparison of electrical direct-current versus pharmacological cardioversion or conservative management. Cardiology. 2009;112(4):270–8.
9. Lessmeier TJ, Gamperling D, Johnson Liddon V, et al. Unrecognized paroxysmal supraventricular tachycardia: potential for misdiagnosis as panic disorder. Arch Intern Med. 1997;157:537–43.
10. Brunton L, Blumenthal D, Buxton I, Parker K. Goodman and Gilman's. Manual of pharmacology and therapetutics. New York: McGraw-Hill Companies; 2008.
11. Link MS. Clinical practice. Evaluation and initial treatment of supraventricular tachycardia. N Engl J Med. 2012;367(15):1438–48.
12. Delacrétaz E. Supraventricular tachycardia. N Engl J Med. 2006;354(10):1039–51.
13. Lim SH, Anantharaman V, Teo WS, Goh PP, Tan AT. Comparison of treatment of supraventricular tachycardia by Valsalva maneuver and carotid sinus massage. Ann Emerg Med. 1998;31:30–5.
14. diMarco JP, Sellers TD, Lerman BB, Greenberg ML, Berne RM, Belardinelli L. Diagnostic and therapeutic use of adenosine in patients with supraventricular tachyarrhythmias. J Am Coll Cardiol. 1985;6:417–25.
15. Paul T, Pfammatter JP. Adenosine: an effective and safe antiarrhythmic drug in pediatrics. Pediatr Cardiol. 1997;18(2):118–26.
16. January CT, Wann LS, Alpert JS, Calkins H, Cigarroa JE, Cleveland Jr JC, Conti JB, Ellinor PT, Ezekowitz MD, Field ME, Murray KT, Sacco RL, Stevenson WG, Tchou PJ, Tracy CM, Yancy CW. 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society. Circulation. 2014;130(23):e199–267.

17. Biaggioni I, Olafsson B, Robertson RM, Hollister AS, Robertson D. Cardiovascular and respiratory effects of adenosine in conscious man: evidence for chemoreceptor activation. Circ Res. 1987;61:779–86.
18. Delaney B, Loy J, Kelly AM. The relative efficacy of adenosine versus verapamil for the treatment of stable paroxysmal supraventricular tachycardia in adults: a meta-analysis. Eur J Emerg Med. 2011;18:148–52.
19. Roguin N, Shapir Y, Blazer S, Zeltzer M, Berant M. The use of calcium gluconate prior to verapamil in infants with paroxysmal supraventricular tachycardia. Clin Cardiol. 1984; 7(11):613–6.
20. Camm AJ, Kirchhof P, Lip GY, Schotten U, Savelieva I, Ernst S, Van Gelder IC, Al-Attar N, Hindricks G, Prendergast B, Heidbuchel H, Alfieri O, Angelini A, Atar D, Colonna P, De Caterina R, De Sutter J, Goette A, Gorenek B, Heldal M, Hohloser SH, Kolh P, Le Heuzey JY, Ponikowski P, Rutten FH. Guidelines for the management of atrial fibrillation: the Task Force for the Management of Atrial Fibrillation of the European Society of Cardiology (ESC). European Heart Rhythm Association; European Association for Cardio-Thoracic Surgery. Eur Heart J. 2010;31(19):2369–429.
21. Camm AJ, Lip GY, De Caterina R, Savelieva I, Atar D, Hohnloser SH, Hindricks G, Kirchhof P, ESC Committee for Practice Guidelines (CPG). 2012 focused update of the ESC Guidelines for the management of atrial fibrillation: an update of the 2010 ESC Guidelines for the management of atrial fibrillation. Developed with the special contribution of the European Heart Rhythm Association. Eur Heart J. 2012;33:2719–47.
22. Brunton L, Blumenthal D, Buxton I, Parker K. Goodman and Gilman's. Manual of pharmacology and therapeutics. New York: McGraw-Hill Companies; 2008.
23. Opie LH, Gersh BJ. Drug for the heart. 7th edn. Philadelphia: Saunders Elsevier; 2009.
24. Yang Y, Cheng J, Glatter K, Dorostkar P, Modin GW, Scheinman MM. Quantitative effects of functional bundle branch block in patients with atrioventricular reentranttachycardia. Am J Cardiol. 2000;85(7):826–31.
25. Vaughan Williams EM. Classification of antidysrhythmic drugs. Pharmacol Ther B. 1975;1:115–38.
26. Members of the Sicilian Gambit. The search for novel antiarrhythmic strategies. Sicilian Gambit. Eur Heart J. 1998;19:1178–96.
27. Antzelevitch C, Brugada P, Borggrefe M, Brugada J, Ramon B, Domenico C, Ihor G, Herve LM, Koonlawee N, Andres Ricardo Perez R, Wataru S, Eric S-B, Hanno T, Arthur W. Brugada syndrome: report of the second consensus conference: endorsed by the Heart Rhythm Society and the European Heart Rhythm Association. Circulation. 2005;111:659–70.
28. Antzelevitch C. J wave syndromes: molecular and cellular mechanisms. J Electrocardiol. 2013;46(6):510–8.
29. Minoura Y, Di Diego JM, Barajas-Martinez H, Zygmunt AC, Hu D, Sicouri S, Antzelevitch C. Ionic and cellular mechanisms underlying the development of acquired Brugada syndrome in patients treated with antidepressants. J Cardiovasc Electrophysiol. 2012;23(4):423–32.
30. Ackerman MJ, Priori SG, Willems S, Berul C, Brugada R, Calkins H, Camm AJ, Ellinor PT, Gollob M, Hamilton R, Hershberger RE, Judge DP, Le Marec H, McKenna WJ, Schulze-Bahr E, Semsarian C, Towbin JA, Watkins H, Wilde A, Wolpert C, Zipes DP, Heart Rhythm Society (HRS); European Heart Rhythm Association (EHRA). HRS/EHRA expert consensus statement on the state of genetic testing for the channelopathies and cardiomyopathies: this document was developed as a partnership between the Heart Rhythm Society (HRS) and the European Heart Rhythm Association (EHRA). Europace. 2011;13(8):1077–109.
31. Van Opstal JM, Volders PG, Crijns HJ. Provocation of silence. Europace. 2009;11(3):385–7.
32. Drago F, Mazza A, Guccione P, Mafrici A, Di Liso G, Ragonese P. Amiodarone used alone or in combination with propranolol: a very effective therapy for tachyarrhythmias in infants and children. Pediatr Cardiol. 1998;19(6):445–9.
33. Akin A, Karagöz T, Aykan HH, Özer S, Alehan D, Özkutlu S. The efficacy of amiodarone-propranolol combination for the management of childhood arrhythmias. Pacing Clin Electrophysiol. 2013;36(6):727–31.

34. Chang PM, Silka MJ, Moromisato DY, Bar-Cohen Y. Amiodarone versus procainamide for the acute treatment of recurrent supraventricular tachycardia in pediatric patients. Circ Arrhythm Electrophysiol. 2010;3(2):134–40.
35. Zellweger MJ, Schaer BA, Cron TA, Pfisterer ME, Osswald S. Elevated troponin levels in absence of coronary artery disease after supraventricular tachycardia. Swiss Med Wkly. 2003;133:439–41.
36. Redfearna DP, Ratibb K, Marshalla HJ, Griffitha MJ. Supraventricular tachycardia promotes release of troponin I in patients with normal coronary arteries. Int J Cardiol. 2005;102:521–2.
37. Macías Gallego A, Díaz-Infante E, García-Bolao I. Spanish catheter ablation registry. 10th official report of the Spanish Society of Cardiology Working Group on Electrophysiology and Arrhythmias (2010). Rev Esp Cardiol. 2011;64(12):1147–53.
38. Scheinman M, Calkins H, Gillette P, Klein R, Lerman BB, Morady F, Saksena S, Waldo A, North American Society of Pacing and Electrophysiology. NASPE policy statement on catheter ablation: personnel, policy, procedures, and therapeutic recommendations. Pacing Clin Electrophysiol. 2003;26(3):789–99.

Atrial Flutter and Fibrillation in the Emergency Setting

5

Ermanno Dametto, Martino Cinquetti, Federica Del Bianco, and Matteo Cassin

5.1 Focus on the Topic

Atrial fibrillation (AF) is a supraventricular arrhythmia characterised by uncoordinated atrial activation which determines on the electrocardiogram (ECG) the presence of fibrillatory waves of different timing, shape and amplitude, associated with an irregular and frequently rapid ventricular response (Fig. 5.1). It is the most frequent arrhythmia with a prevalence of 1.5–2 % in the general population [1], increasing with age and is the most frequent cause of arrhythmic access to the emergency department (ED), 1.5 % in "FIRE" and 3.1 % in "HERMES-AF" registries [2, 3] and hospitalisation (3.3 % in "FIRE" registry [2]).

European (ESC 2010) [4] and US (AHA/ACC/HRS 2014) [5] guidelines (GL) classify AF as *first diagnosed* or *recurrent*. Recurrent AF episodes are defined as *paroxysmal* if they terminate spontaneously (AHA/ACC/HRS 2014 GL [5] include also AF treated with cardioversion within 7 days), *persistent* (lasting longer than 7 days), *long-lasting persistent* (continuous for more than 12 months with intention of rhythm control strategy) or *permanent* (further attempts to restore and/or maintain sinus rhythm excluded).

AF may be asymptomatic, so the patient may be referred to the ED by a general physician for an occasional finding of irregular heart rhythm, or symptomatic. Symptoms described on arrival at the ED are most frequently palpitations (69.2 %), followed by dyspnoea (27.5 %), congestive heart failure (10.9 %), fatigue (105), angina (9 %), pulmonary oedema (4.5 %), syncope (3.3 %), transitory ischaemic attack (TIA) or stroke (2.2 %) [2]. Apart from acute symptoms, atrial fibrillation

E. Dametto (✉) • F. Del Bianco, • M. Cassin
Cardiology Unit, Presidio Ospedaliero di Pordenone, AAS5-Azienda per l'Assistenza Sanitaria n.5 "Friuli Occidentale", Pordenone, Italy
e-mail: ermanno.dametto@gmail.com

M. Cinquetti
Cardiovascular Department, Ospedali Riuniti and University of Trieste, Trieste, Italy

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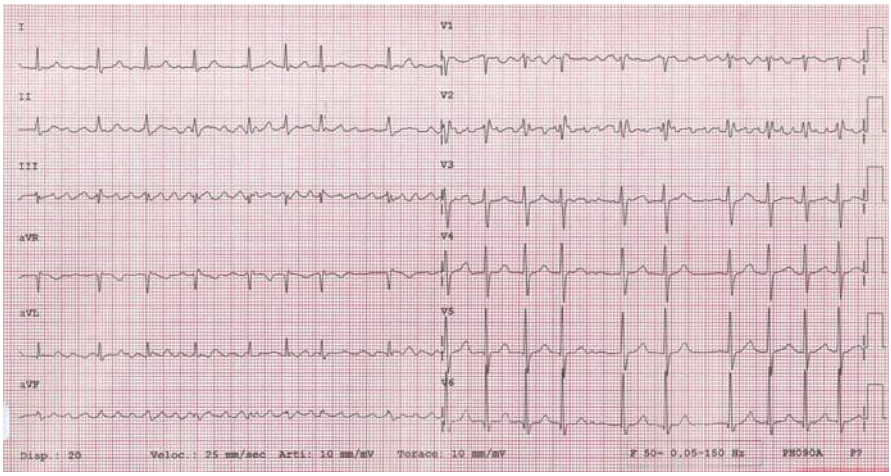

Fig. 5.1 *Atrial fibrillation* with moderately fast ventricular response (about 100 bpm). RR intervals are irregular and coarse F waves are visible

causes a fivefold risk of stroke, a threefold risk of heart failure and doubles dementia and mortality [5]. Furthermore, it reduces the quality of life [6]. For stroke, risk stratification and treatment of *nonvalvular* and *valvular AF* are distinguished. Valvular AF carries a very high thromboembolic risk if not anticoagulated: in patients with mitral stenosis and AF it is about 20 times over patients not affected by AF. Definition of valvular AF is not univocal; according to the AHA/ACC/HRS 2014 Guidelines, it is defined as AF associated with rheumatic mitral stenosis, a mechanical or bioprosthetic heart valve or mitral valve repair [5].

Atrial flutter (*AFL*) is less common (about 10 % of ED admissions for AF or AFL were due to AFL in the "FIRE" registry [2]) but shares similar clinical features and treatment with AF. It is a macroreentrant atrial arrhythmia with regular atrial activation and shows electrocardiographically continuous atrial activity in at least one lead which may have various morphologies depending on the location of the circuit. The ventricular rate is usually regular due to a fixed atrioventricular (AV) conduction rate, mostly 2:1 with a frequency of 120–150 bpm (Fig. 5.2), but may be slower or irregular due to a variable AV conduction rate.

Both arrhythmias are usually associated with cardiovascular pathologies, mostly hypertension (64 %) but also coronary artery disease (32 %), heart failure (32 %), valvular hear disease (26 %) and cardiomyopathy (10 %). Chronic obstructive pulmonary disease (COPD) may be associated in 13 % of the patients and thyroid disease in 9 %. In about 10 %, none of these comorbidities are found, and the arrhythmia is called "lone atrial fibrillation" (or flutter), if the patient is younger than 65 years old [7].

Acute diseases may be associated with AF/AFL like myocardial infarction, pulmonary embolism, pneumonia, severe infections, alcohol abuse, drug toxicity, hypothermia and electrolyte abnormalities [8].

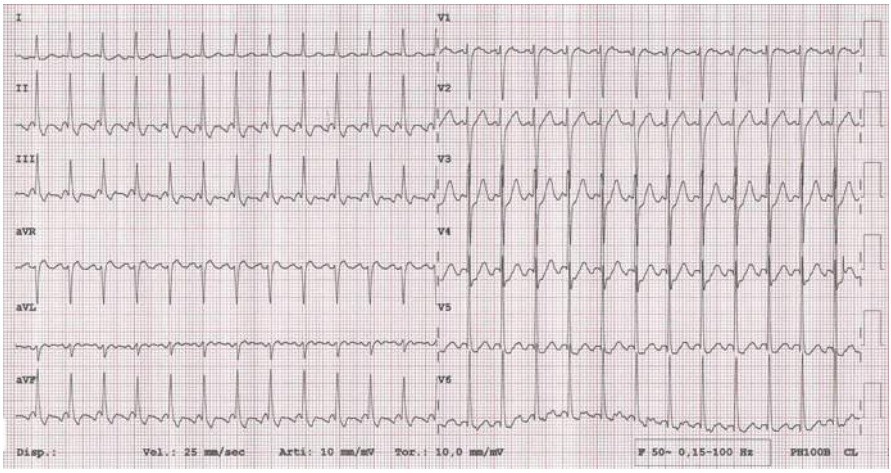

Fig. 5.2 *Atrial flutter with 2:1 AV conduction and quite rapid ventricular rate* of 150 bpm. Regular RR intervals

5.2 What Physicians Working in ED Should Know

The ED physician has several tasks:

1. *Rule out severe clinical instability*

Altered mental status, hypotension, pulmonary oedema and ongoing ischemia necessitate acute electrical cardioversion (ECV) once diagnosis of AF/AFL is confirmed and is thought to be the primary cause [9], at least if there is no prompt response to rate control therapy [4, 5, 10].

2. *Diagnosis of the arrhythmia*

The pulse presents irregular in case of AF but is usually regular in case of flutter. Electrocardiographic diagnosis of AF is usually easy for the irregular RR intervals and f waves. Atrial flutter appears usually as a regular arrhythmia due to fixed conduction rate, mostly 2:1 with ventricular rates around 120–150 bpm, but may be less or conduction may be variable. When rapid conduction is present, the flutter waves may not be so evident. In these cases and if the patient is stable, we recommend to perform a continuous 12-lead ECG registration during carotid sinus massage (after exclusion of any carotid bruit) or adenosine infusion (6, 12 or 18 mg bolus followed by 20 ml saline solution flush) to clear up differential diagnosis with other supraventricular arrhythmias and to record the flutter wave morphology, which helps the cardiologist to define the long-term therapeutic approach (e.g. radiofrequency ablation). *Typical atrial flutter* (also called *cavotricuspid isthmus-dependent atrial flutter*) is a macroreentrant circuit posterior to the tricuspid annulus, crossing the

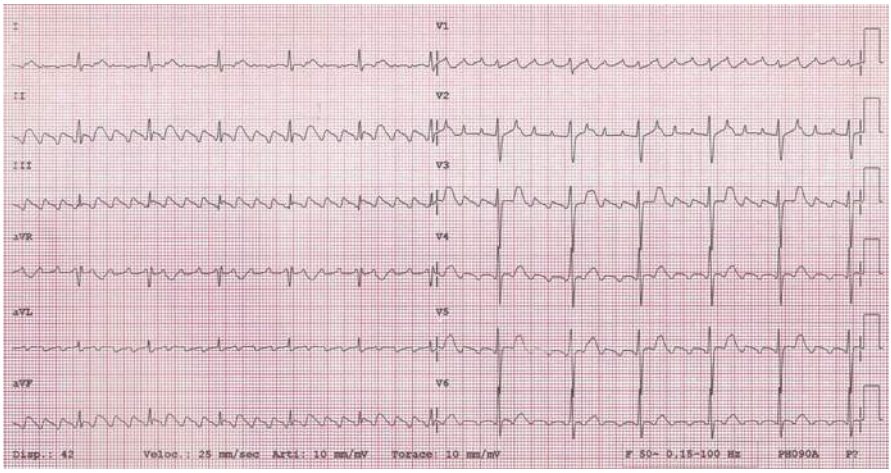

Fig. 5.3 *Typical atrial flutter with counterclockwise atrial activation (common atrial flutter)* with 4:1 AV conduction, 70 bpm. Atrial flutter waves are clearly seen: negative in inferior leads and positive in V1

isthmus between the inferior vena cava orifice and tricuspid annulus. Activation sequence is more frequently counterclockwise (downwards the right atrial free wall and upwards the interatrial septum), and in this case, the arrhythmia is called *common atrial flutter* or *counterclockwise AFL* (Fig. 5.3). Less common is *clockwise typical atrial flutter*, also called *reverse AFL* (Fig. 5.4). On ECG, the former shows classical "sawtooth" flutter waves with negative polarity in inferior leads and positive in V1, and the latter shows flutter waves with positive polarity in the inferior leads and negative in V1. Atypical flutters (noncavotricuspid isthmus dependent) show flutter waves with other morphologies (Fig. 5.5) [5].

In patients with AFL treated with flecainide or propafenone sometimes rapid 1:1 AV conduction is favoured due to flutter wave slowing. This phenomenon is frequently accompained by conduction aberrancy and QRS widening (Fig. 5.6).

In patients with Wolff–Parkinson–White Syndrome, a *pre-excited AF* (Fig. 5.7) is characterised by irregular RR intervals with various degrees of QRS widening (various degrees of fusion between accessory pathway and nodal conduction). In case of fast conduction properties of the accessory pathway, ventricular rate can be extremely high, possibly leading to severe haemodynamic consequences or even to ventricular fibrillation. *Pre-excited AFL* appears as a wide QRS tachycardia.

3. *Clinical history and physical examination for evaluation of concomitant heart disease and comorbidities*

A complete medical history including previous cardiac and noncardiac diseases, history of former AF, medication use and drug or alcohol abuse should be obtained. Onset of the current AF episode should be carefully defined as it is a crucial point for decision making, in particular, it has to be cleared if onset is within or more than

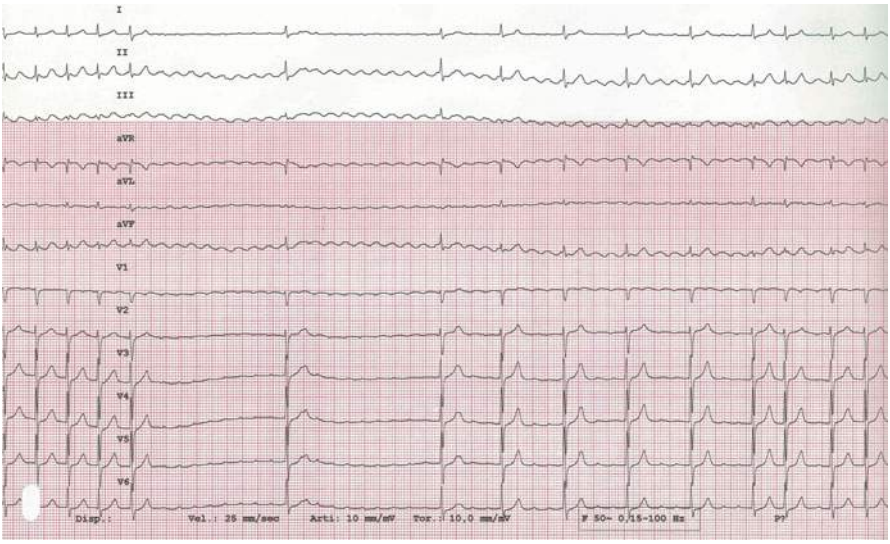

Fig. 5.4 *Typical atrial flutter with clockwise atrial activation (reverse atrial flutter) during adenosine injection*. Initially 2:1 AV conduction, 150 bpm and masked flutter waves. A marked conduction slowing follows, unmasking flutter waves: positive in inferior leads and negative in V1. Diagnosis was confirmed by electrophysiological study

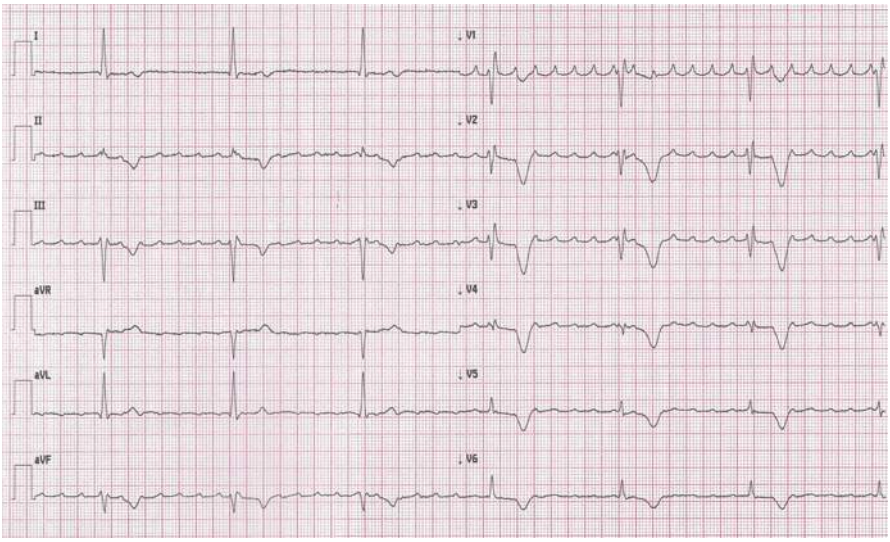

Fig. 5.5 *Atypical atrial flutter:* flutter waves are positive in inferior leads and in V1

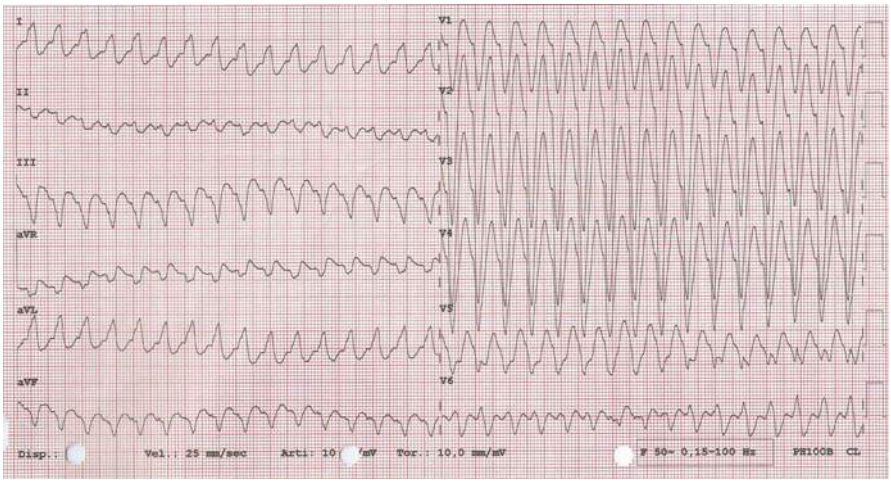

Fig. 5.6 *Atrial flutter with 1:1 AV conduction and aberrancy* 190 bpm (during i.v. flecainide infusion). Flutter waves are not visible. Resembles a wide QRS tachycardia

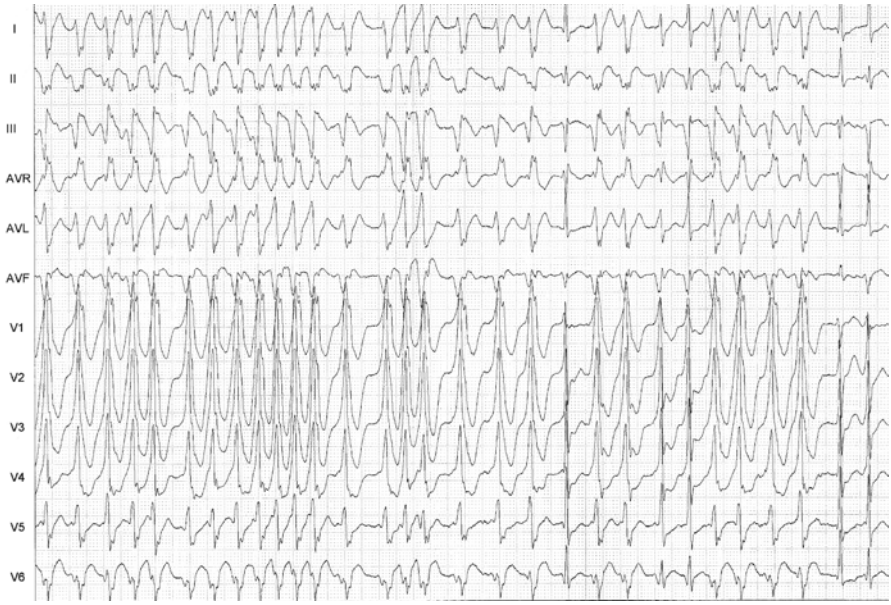

Fig. 5.7 *Pre-excited atrial fibrillation with fast AV conduction*, at times near 300 bpm. Bizarre wide QRS morphology with irregular RR intervals. In between, there are a few beats with normal QRS, conducted through the AV node

48 h (time within acute cardioversion without previous anticoagulation can be considered). Frequently, AF presents as continuous palpitations with acute onset. In this case, onset definition is easy. Other times, palpitations are perceived intermittently

or other symptoms dominate as dyspnoea or fatigue which can be vague. In these cases, it can be of help if patients are used to evaluate daily their pulse (often evaluated together with arterial pressure in the hypertensive). If onset of AF cannot be clearly dated, the arrhythmia should be regarded as *"AF of undefined onset"*.

Physical examination should initially be aimed to vital signs like blood pressure, pulse frequency, signs of pulmonary or peripheral congestion and oxygen saturation. Cardiac assessment should include evaluation of heart sounds and murmurs and a careful evaluation of both arterial and jugular pulse (to assess the rhythm and possible AV dissociation).

Laboratory tests should be tailored to patient's presentation. A complete blood count, serum electrolytes, renal and hepatic function tests, coagulation status and thyroid-stimulating hormone should be routinely obtained. Brain natriuretic peptide, troponin I or T, C-reactive protein and D-dimer test help ruling out heart failure, myocardial ischemia, concomitant inflammatory or infectious disease and pulmonary embolism if these conditions are suspected.

Thyroid disease is not uncommon in AF patients and overt hyperthyroidism is associated in about 4 % of AF patients [11]. Thyroid-stimulating hormone is a reliable screening tool which can be applied in some EDs and may help in decision making (choosing rate control strategy or defining contraindication for amiodarone use [12].

An urgent echocardiogram should be performed in haemodynamically compromised patients [4]. Alternatively, a focused ultrasonography performed by the ED physician can determine underlying cardiac condition like structural heart disease, pericardial effusion and pulmonary embolism, or clarify causes of shock or hypotension and guide resuscitation by volume repletion measuring the inferior vena cava diameter. Ultrasound competence is not uniformly available among ED physicians, but a focused examination of the heart may help guiding therapy in AF management [13]. However, a complete echocardiographic examination should be performed as part of initial evaluation [4, 5, 14] as soon as possible, even on outpatient basis in stable patients.

4. *Symptom relief by cardioversion (electrical or pharmacological) or rate control*

Unstable patients In patients with haemodynamic compromise like hypotension, pulmonary oedema or ongoing myocardial ischemia thought to result mainly from acute AF or AFL, electrical cardioversion is indicated [9], especially if it does not respond promptly to pharmacological rate control measures [4, 5, 10].

Anticoagulation with low molecular weight heparin (LMWH) or unfractionated heparin (UFH) should be initiated as soon as possible. Do not delay ECV if the patient is extremely unstable [9]. In the author's opinion if the patient is hypotensive, ECV is preferable, whereas if pulmonary oedema or myocardial ischaemia is present, rate control should be tried first.

Atrial fibrillation can be an epiphenomenon of an "alternative" primary diagnosis, and in these cases, mortality rate is high (11 % in the study of Atzema et al. [15]). Patients may present with vague symptoms. Scheuermeyer et al. [16] reported

on a series of patients with underlying medical illness like sepsis, acute coronary syndrome (ACS), decompensated heart failure, pulmonary embolism, obstructive pulmonary disease exacerbation, acute renal failure and gastrointestinal bleedings. Those who underwent immediate rate or rhythm control attempts suffered a much higher complication rate (40.7 %) than those who did not (7.1 %) and were unlikely to achieve success. So it is more advisable to delay rhythm management strategies and manage these patients first correcting the primary acute disease, administrating intravenous fluids guided by bedside echocardiography (to assess volume status) and performing frequent reassessments while waiting for confirmatory diagnostic tests [16].

Stable patients with recent-onset AF (<48 h) There is still controversy on optimal management of these patients, being rate and rhythm control the two alternative strategies without clear-cut evidence favouring one or the other. *Rate control strategy* in the ED consists of pharmacological control of ventricular rate and anticoagulation treatment if indicated. With *rhythm control strategy*, the patient is cardioverted to sinus rhythm (SR) either pharmacologically or electrically, usually discharged from the ED and followed as outpatient afterwards. A delayed cardioversion (CV) after at least 3 weeks of adequate anticoagulation can be planned in some patients if the initial rate control strategy is not well tolerated.

Although several trials on AF patients like the AFFIRM [17] did not find significant differences on the main clinical outcomes between rate and rhythm control, the latter seems the preferred strategy worldwide in the long treatment of AF [18, 19]. General indications for preference of either strategy are listed in Italian guidelines on AF management [20]: rhythm control strategy should be the option for (1) patients with first AF episode (taking into account also age and comorbidities) and (2) for patients with recurrent AF with high probability of long-term sinus rhythm maintenance, or in whom rate control proved uneffective or if AF determines negative haemodynamic effects. Rate control strategy is preferred for (1) patients refractory to antiarrhythmic therapy, with frequent AF relapses non-candidable to AF ablation or not candidable to rhythm control strategy because of age or underlying heart disease and (2) for older asymptomatic patients with persistent AF or older patients with recurrent AF, heart failure and poor ventricular function.

As far as ED practice concerns, there is a considerable variability among different countries: rate control strategy is largely favoured in the USA (74 %), while in Canada, most patients undergo cardioversion (66 %). The UK and Australia adopt rate or rhythm control in equal proportions [21].

In our clinic, rhythm control is generally the preferred management strategy.

The theoretical advantages of an aggressive rhythm control approach are rapid discharge without hospital admission, immediate resumption of normal activities, potential avoidance of medications and minimization of healthcare resources and costs. In addition, with rate control strategy, many patients can derive physical and psychological inconvenience maintaining the arrhythmia and drugs after discharge [22].

A recent review of five studies involving 1593 cases undergoing ED cardioversion [23] demonstrated that success rate was high (around 90 %), with a discharge rate of about 90 % and a complication rate generally lower than 5 %, exceptionally leading

to hospital admission. Thromboembolic complication rate was very low (0.06 %). So in selected patients with recent-onset AF, ED physicians should feel comfortable with this approach which should ideally be standardised by a local ED protocol and include timely and appropriate follow-up. The first description of an ED protocol dealing with recent-onset AF or flutter was the "Ottawa Aggressive Protocol" [24]. Excluding patients presenting with AF onset >48 h or of unknown duration and those with other primary diagnosis necessitating admission, it involved sequential pharmacologic and, when indicated, electrical cardioversion by the emergency physician. It allowed discharge of 96.8 % of the 660 enrolled patients, 93.3 % of those in sinus rhythm, after a median length of stay of 4.9 h overall. Adverse outcomes, mainly transient hypotension, occurred in 7.6 % and only 3.2 % of patients required hospitalisation. No patient died or suffered a stroke. Despite these encouraging results, limited data help guide emergency physicians on which AF patients can be safely managed as outpatients (see below). An interesting implementation of an ED cardioversion protocol for acute AF patients after exclusion of high-risk features was described by Bellone et al. [13]; their workup included morphological echocardiographic investigation before treatment by certified emergency physicians, which has the potential of improving the risk stratification and the therapeutical choice.

Another management strategy consists in conservative "wait-and-watch approach" which relies on the known discrete likelihood of spontaneous cardioversion of recent-onset AF (from 20 % up to 68 % in the study of Danias et al. [25]); early (<24 h) presentation is the best predictor of spontaneous cardioversion. Patients may be discharged on rate control medication and anticoagulation if appropriate and scheduled for return visit to ED to permit CV within the 48 h period [26] from symptom onset if AF persists, or may be admitted to an ED observation unit, reassessed after some hours and eventually cardioverted if AF persists [27]. This strategy can be applied following the patient's preference but may be an option if ED is overcrowded.

Stable patients with AF onset >48 h or with AF of uncertain onset If the patient has properly been anticoagulated for at least the preceeding 3 weeks, acute cardioversion can be considered as an alternative to rate control. On the contrary, in non-anticoagulated patients, rate control is generally the most appropriate strategy. In the acute setting, the target ventricular rate should usually be 80–100 bpm [4]. Once rhythm control is achieved and anticoagulation started, the patient can be considered for discharge and referred to a cardiologist or a general practitioner. If adequate rate control is not achieved, the patient is still symptomatic or if new ventricular dysfunction is to be detected, cardioversion should be considered: the patient must be fully anticoagulated independently of the thromboembolic risk (start with UFH or LMWH for immediate action and bridge then with vitamin K antagonists; novel anticoagulants may be used as alternative), then undergo a transoesophageal echocardiogram (TEE) to exclude intracardiac thrombi and finally electrically cardioverted. Anticoagulation should be continued at least 4 weeks or indefinitely, according on the thromboembolic risk profile. If thrombosis is detected, the patient should undergo another TEE after at least 3 weeks of effective anticoagulation and if it has resolved undergo ECV thereafter [4, 5, 10].

According to the Canadian Guidelines 2014 [10], the TEE-based CV approach should also be applied for a AF patients presenting <48 h after AF onset if high-risk features like valvular AF or stroke/TIA in the last 6 months are present.

5. *Antithrombotic therapy*

It is generally believed that antithrombotic therapy is the mainstay of AF management in any care setting, as from this therapy, most of prognostic benefit in terms of morbidity (stroke prevention) and mortality has to be gained. Nevertheless, it is still much underprescribed, usually in less than 55 % of AF patients, as results from studies are conducted in different settings. EDs constitute excellent opportunities to start thromboprophylaxis in the absence of contraindications and help filling this gap [3]. Treatment options include LMWH or UFH, vitamin K antagonists (VKAs) and novel oral anticoagulants (NOACs).

Immediate anticoagulation can be obtained with unfractionated heparin (i.v. bolus of 80 UI/kg followed by continuous i.v. infusion adjusted to an activated partial thromboplastin time of twice the control value (initially 18 UI/kg/h)) [28] or LMWH (most popular being enoxaparin 100 UI/kg BID) based on ACUTE II [29] ad ACE trials [30]. Heparin therapy is generally used as bridging with VKAs which have a delayed onset of action which takes some days.

At present, three NOACs have been tested for AF cardioversion (prospectively or in post hoc analysis) [31–33]:

Dabigatran 150 mg twice daily (TD) if creatinine clearance > 50 ml/min, 110 mg TD if CrCl between 30 and 50. In the USA, the FDA approved 150 mg TD if CrCl >30 and 75 mg TD if ClCr 30–15. Do not use in association with dronedarone; reduce dose if concomitant verapamil is used.
Rivaroxaban 20 mg once daily or 15 mg if CrCl 49–30 ml/min.
Apixaban 5 mg TD or 2.5 mg TD if two or more risk factors for bleeding: age <80 years, weight <60 kg, creatinine >1.5 mg/dl.
Time to peak action is around 3 h for all three.

Valvular AF carries a very high risk per se and must always be treated with VKAs. NOACs were not tested or resulted inferior to VKAs in this setting [5, 34].

For patients with nonvalvular AF, a number of risk stratification schemes have been proposed, the mostly recommended [1, 5] being the CHA2DS2-VASc scoring system [35] (Table 5.1). As chronic therapy, anticoagulation is not indicated for score = 0, indicated for score ≥2, recommended for score = 1 by European guidelines [4], while US guidelines [5] indicate alternatively oral anticoagulants, aspirin or no antithrombotic treatment (Class IIb). We favour oral anticoagulation for patients with score = 1 unless at risk of bleeding.

Any type of AF (paroxysmal, persistent, permanent) carries a similar risk of stroke, so anticoagulation should be chosen regardless of AF type. Atrial flutter has shown to have similar thromboembolic risk as AF and should be managed the same way.

All patients with AF >48 h duration (including those with CHA2DS2-VASc score = 0) candidate to cardioversion must be adequately anticoagulated for at least

Table 5.1 CHA2DS2-VASc scoring system

Acronym	Risk parameter	CHA2DS2VASc score	Stroke rate (%/year)
C	Cardiac failure or dysfunction	0	0
H	Hypertension	1	1.3
A (2)	Age >75	2	2.2
D	Diabetes	3	3.2
S (2)	Stroke/TIA or thromboembolism	4	4
V	Vascular disease	5	6.7
A	Age 65–74	6	9.8
Sc	Sex category (female)	7	9.6
		8	6.7
		9	15.2

Based on Lip et al. [35]
One point is assigned to each parameter except for Age >75 and Stroke whom 2 points are assigned

the three preceding weeks before the procedure, unless intraatrial thrombus has been ruled out by TEE or in case of rare emergency situations and continued on anticoagulation for at least 4 weeks afterwards. Lifelong anticoagulation is indicated for patients at high risk of stroke or AF recurrence.

For patients with AF onset <48 h, current guidelines [4, 5, 10] indicate cardioversion without previous anticoagulation, as former studies demonstrated low embolic complications (0.8 % stroke at 30 days in the study of Weigner et al. [36]). Some concern has raised as it was demonstrated that 4–14 % of these patients had an intraatrial thrombus on transoesophageal echocardiography [37, 38], and ESC 2010 guidelines [4] recommend to start anticoagulation with UFH or LMWH before cardioversion even for AF lasting <48 h. In high-risk patients, heparin therapy has to be followed by long-term oral anticoagulation (class I recommendation), while in patients with no risk factors, heparin therapy may be considered only pericardioversion (IIb, level C recommendation), but this latter recommendation is not evidence based. The 2014 AHA/ACC/HRS guidelines [5] make similar recommendations on use of peri- and postcardioversion antithrombotic therapy, adding the option to use NOACs for the purpose (Class I recommendation). In this context, also a recent European Expert Consensus [28] agrees with the use of NOACs with a class IIa recommendation. A recent large cohort study [39] on 7660 cardioversions of AF lasting <48 h supports the recommendation of prolonged anticoagulation in high-risk patients after CV: without anticoagulation, thromboembolic risk resulted 0.7 % at 30 days. Risk factors were age, heart failure, diabetes, and female gender (elements of CHA2DS2-VASc score system). Patients without risk factors experienced a very low complication rate of 0.2 %, whereas those with multiple risk factors had a thromboembolic risk approaching 10 %.

For patients with AF lasting >48 h or of unknown origin with no risk factors (CHA2DS2-VASc = 0), anticoagulation is not advised unless a delayed cardioversion is planned.

6. *Decision for cardiologist consultation or hospitalisation*

Consultation with a cardiologist should be requested for:

- Unstable patients (hypotension, angina, heart failure)
- Patients with known significant cardiac disease
- If significant underlying heat disease is suggested by objective findings, laboratory data or diagnostic testing
- If rate or rhythm control has not been achieved
- If cardiovascular complications have occurred as consequence of antiarrhythmic or rate control therapy
- If cardioversion preceded by TEE is the choice
- If Brugada pattern emerges on ECG after pharmacological cardioversion

Most of these patients will also require hospitalisation, as well as patients with concomitant acute illness (sepsis, pneumonia, pulmonary embolism, etc.).

5.3 What Cardiologists Should Know

"Pill-in-the-pocket approach": useful treatment strategy for patients with no or minimal heart disease, no conduction disturbances and infrequent but prolonged and well tolerated AF recurrences. Patients have to take orally 200–300 mg flecainide or 450–600 mg propafenone (the higher dosage for weight >70 kg). This approach should be prescribed by a cardiologist after full diagnostic workup (and therefore in the author's opinion avoided in first diagnosed AF) and must first be tested in the ED for safety and efficacy before it can be used at home. If this strategy is the choice, the cardiologist should specify this indication on his written evaluation of the patient so that instructions can be promptly be followed in the ED for the first test treatment. In the original article by Alboni et al., [40] rapid 1:1 flutter complicated only 1 case (0.6 %), so ESC 2010 guidelines [4] recommend this strategy as it was described, but AHA/AC/HRS 2014 [5] and CCS 2010 [41] guidelines recommend addition of beta blocker or nondihydropyridine calcium channel blocker to avoid this side effect. This strategy should not be used for patients with AFL.

Patients already on chronic antiarrhythmic therapy may present to the ED with relapsing acute onset AF. If cardioversion is judged to be the choice, we suggest electrical CV as first choice. Alternatively, pharmacological cardioversion (PCV) with the same antiarrhythmic agent administered intravenously at reduced dose can be performed although there is little evidence in literature. In our centre, it has been common practice for more than a decade to use flecainide or propafenone half dose bolus (1 mg/kg in 10 min) on top of chronic therapy with the same agents or intravenous amiodarone with conventional dosages and infusion rates on top of chronic amiodarone therapy, after checking for contraindications. We have not yet experienced significant side effects. Patients are discharged on the same antiarrhythmic agent, even at higher dose, as single or few AF relapses should not be interpreted as treatment failures if the antiarrhythmic burden is lowered on medication. We advise

in general against the use of different antiarrhythmic drugs on top of chronic antiarrhythmic drug therapy although limited experience on intravenous ibutilide in patients taking amiodarone, propafenone or flecainide seems safe [42, 43].

Coronary artery disease and atrial fibrillation are often associated diseases [7] and share common risk factors. AF may complicate myocardial infarction in about 18 % [44] and is frequently associated with severe LV damage and heart failure. Fast ventricular rate contributes to increased myocardial damage and heart failure, requiring prompt treatment. Adequate rate control can be obtained preferably by administration of beta blockers (uniformly class I recommendation by ESC 2010, AHA/ACC/HRS 2014, ESC 2012 STEMI guidelines [4, 5, 45]) or in alternative nondihydropyridine calcium antagonists (class IIa in ESC 2010; class IIb in AHA/HRS/ACC 2014; class I in ESC STEMI 2012), in patients who do not display HF or haemodynamic instability. In patients with associated severe left ventricular dysfunction, or signs and symptoms of heart failure, rate control is more safely achieved with i.v. amiodarone or i.v. digoxin (class I in ESC STEMI 2012 [45]; Class IIb in AHA/ACC/HRS 2014 [5]), also in association [45]. Urgent electrical cardioversion should be considered in patients presenting with atrial fibrillation and intractable ischaemia or haemodynamic instability [45].

Pharmacologic cardioversion should be considered if ischemic trigger has resolved with amiodarone being the agent of choice [45].

A still yet unsolved issue is the significance of two common findings, *mildly elevated troponin I (TnI) levels or ST depression* on ECG in the absence of overt acute coronary syndromes (ACS). It is common practice in the EDs to check troponin levels in patients presenting with AF even after excluding primary diagnosis of acute coronary syndrome (76 % of 662 patients in the study of Gupta et al. [46]). The prevalence of mildly positive TnI tests in acute AF patients without evident ACS varies from 9 [47] to 44 % [46]. Troponin I elevation predicts increased the risk of death [48, 49] or MI [46, 48] in the long term. This seems true in the acute setting for minor TnI elevations (detectable but subthreshold) as well as for positive TnI tests (with the higher the value the higher the risk [48]), but also in more chronic AF settings [49]). Troponin elevation is associated with older age, CAD risk factors, known CAD disease, creatinine elevation and ventricular rate but not with angina or ischemic ST depression [46, 28].

It is known that TnI elevations can occur also in the absence of significant coronary stenoses probably because of supply–demand mismatch or atrial–ventricular stretch [46]. Moreover, it has to be taken into account that troponin levels may be elevated for other reasons in acute AF patients (79 % in the study of Meshkat et al. [50] including demand-related rise in 36 %, congestive heart failure in 29 %, renal failure in 17 %, sepsis in 14 % and pneumonia in 2 %).

TnI elevation leads to increased prescription of diagnostic tests for CAD which demonstrated mild diagnostic yield (absent in the study of Gupta et al. [46], useful leading to threefold coronary revascularisation rate and better outcome in the study of Conti et al. [47]). Although these studies confirm that biomarkers seem useful for improving risk prediction in AF, the link between AF and coronary artery disease needs additional investigation.

ECG changes suggesting ischemia (ST segment depression) even in the absence of angina or troponin elevations are also frequent. The positive predictive value for

significant CAD varies from 31 [51] to 50 % [52]. Pradhan et al. [51] observed a strong correlation between grade of ST depression and ventricular rate. In their series, the percentages of positive results for diagnostic tests for CAD did not differ significantly between patients with (31 %) or without (21 %) ST depression during AF, so in conclusion, they stated that ST depression during AF is a prevalently rate-related phenomenon.

Absence of ECG changes resulted highly reassuring in the study of Tsigkas et al. [52] (only 4 % prevalence of CAD). In the series of Androulakis et al. [53], the prevalence of ST depression during fast FA was 32.5 %, and non-invasive diagnostic tests performed quite well in identifying patients with CAD.

So what to do in the presence of TnI elevation or new "ischemic" ECG changes during rapid AF?

- *Negative TnI* and *no ECG changes* do not require further investigation.
- *Mildly positive TnI values or ECG changes* should be evaluated in a polyparametric context which accounts for risk factors and cardiac comorbidities.
  - *Lower-risk* patients may be discharged after 12-24 h in an observation unit if stable and troponin descending, possibly after an echocardiogram and non-invasive provocative test for CAD (otherwise these tests should be arranged shortly after as outpatients).
  - *Higher-risk* patients should be admitted.

Heart failure (HF) and AF are strongly associated diseases and can interact to promote their perpetuation as onset of AF can worsen symptoms in patients with HF. These patients should be managed according to clinical severity. If markedly compromised (pulmonary oedema), they should be cardioverted if there is no prompt response to rate control (amiodarone or digoxin are agents of choice) [4]. If clinical presentation is less severe and AF duration is less than 48 h, pharmacological cardioversion with amiodarone or ECV can be considered as alternative to rate control strategy. For rate control purposes, cautious use of beta blockers in patients with systolic heart failure (or diltiazem if HF with preserved EF) can be considered (AHA/ACC/HRS 2014 class I recommendation) [5].

Conversely, worsened HF can promote a rapid ventricular response in permanent AF. These patients usually do not respond to cardioversion and rate control is as well more difficult to achieve. The mainstay of therapy in this setting should be aimed to optimise HF treatment.

Patients may present with newly detected HF in the presence of AF with a rapid ventricular response. These should be presumed to have a rate-related cardiomyopathy, with the arrhythmia being a potentially reversible cause of HF [54]. In this situation, two options can be considered: (1) rate control and re-evaluation for EF improvement and (2) rhythm control, which common practice is to initiate amiodarone and cardiovert in the patient electrically at least 3 weeks later [54]. In our clinic, we try to pursue a more rapid rhythm control strategy for patients with suspected tachycardiomyopathy which consists in performing TEE after clinical stabilisation with HF medications, rate control and anticoagulation. If no thrombus is

detected, the patient receives a 24 h amiodarone infusion after which ECV is performed and followed by oral loading dose of amiodarone.

AF in Pre-excitation If very rapid and unstable, urgent ECV should be performed [4, 5, 9]. If heart rate is not too high and the arrhythmia is well tolerated, it can be cardioverted with intravenous flecainide, propafenone, ibutilide and procainamide. The same agents are effective of rate control [4, 5, 20]. Drugs acting on the AV node, like calcium channel blockers, digitalis and adenosine, are contraindicated, because conduction over the accessory pathway is favoured and the rate transmission to the ventricle may become very rapid potentially inducing ventricular fibrillation. Amiodarone has recently been defined as harmful in the AHA/ACC/HRS 2014 guidelines [5], whereas ESC guidelines 2010 [4] recommend it class I for rate control. Beta blockers are contraindicated according to ESC guidelines 2010 [4] but might be used with caution according to the AHA/ACC/HRS 2014 guidelines [5].

5.4 A Possible Algorithm/Pathway for Diagnosis and Treatment

The management of patients with AF depends on their clinical presentation which may be unstable (Fig 5.8), stable with AF onset < 48 hours (Fig. 5.9) or stable with AF onset > 48 hours/undefined onset (Fig 5.10).

If cardioversion is the choice, check potassium levels (unless emergent ECV is necessary) and correct it if necessary proceeding. If patient is on chronic digoxin therapy, plasma level should be checked (ECV is contraindicated in case of intoxication).

In general, ECV is more effective than pharmacological cardioversion (PCV) and requires less time, but greater organisational efforts.

SR is achieved in about 90 % with ECV and 70 % with PCV [55]. If PCV fails, ECV restores SR in the majority of cases. On the contrary, if AF relapses immediately (IRAF) after ECV, the use of an antiarrhythmic agent before repeat CVE enhances the probability of maintaining SR. If AF does not convert to sinus rhythm with ECV (no or a maximum single sinus beat achieved, as defined by ESC 2010 guidelines [4]), the patient can be pretreated with ibutilide (1 mg over 10 min) which lowers defibrillation threshold and facilitates greatly achievement of SR on repeat ECV [56].

Pharmacologic cardioversion is little effective after 7 days of AF onset; therefore, in this case, ECV is preferred.

Atrial flutter poorly responds to antiarrhythmic drugs except for ibutilide; therefore, ECV should be first choice. If propafenone or flecainide are used for AFL conversion, slowing of atrial rate occurs, which sometimes can conduct faster to the ventricles (from usual 2:1 to 1:1 conduction), often with conduction aberrancy (Fig. 5.6). The result is a very rapid, frequently untolerated arrhythmia which should be electrically cardioverted. So if these agents are used for treatment of AFL, co-administration of rate control drugs is advised. The same phenomenon may occur when flecainide or propafenone is used for conversion of AF, as in some cases AF

organizes into AFL ("IC flutter"). In rare cases of patients treated with these agents, an electrocardiographic "Brugada pattern" may become evident (coved-type ST elevation in at least one lead from V1 to V3). These patients should undergo cardiologic consultation before eventual discharge.

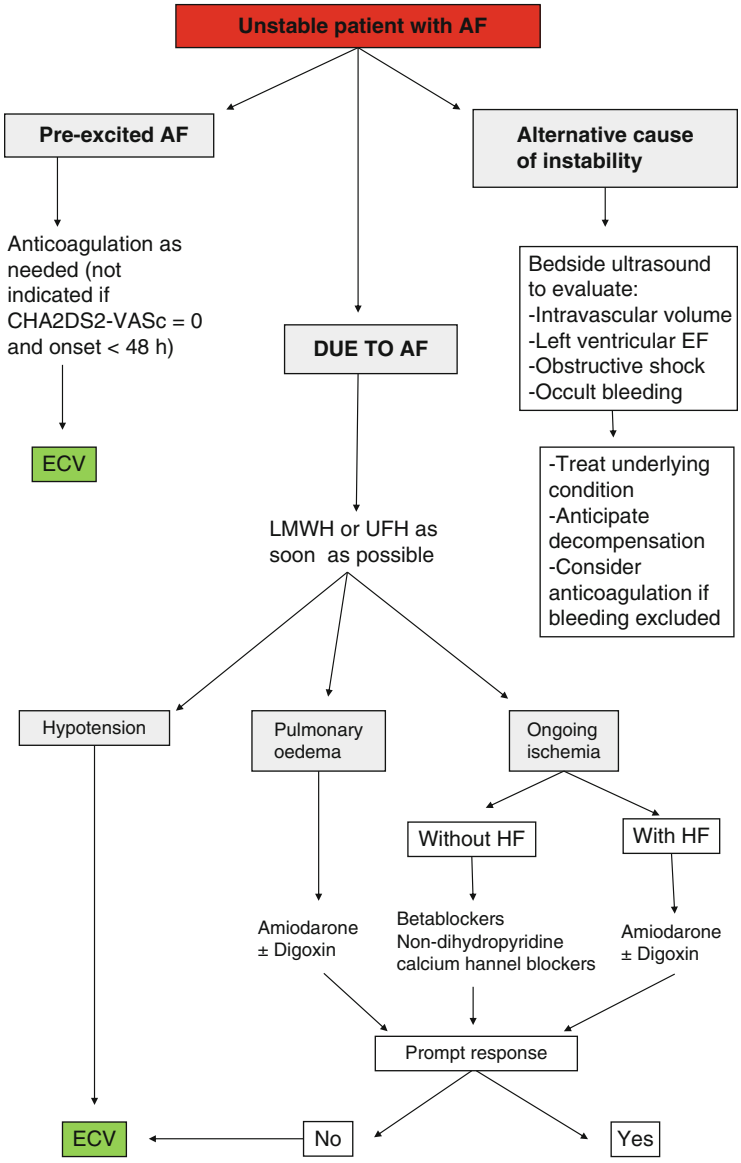

Fig. 5.8 Algorithm for management of patients with unstable AF

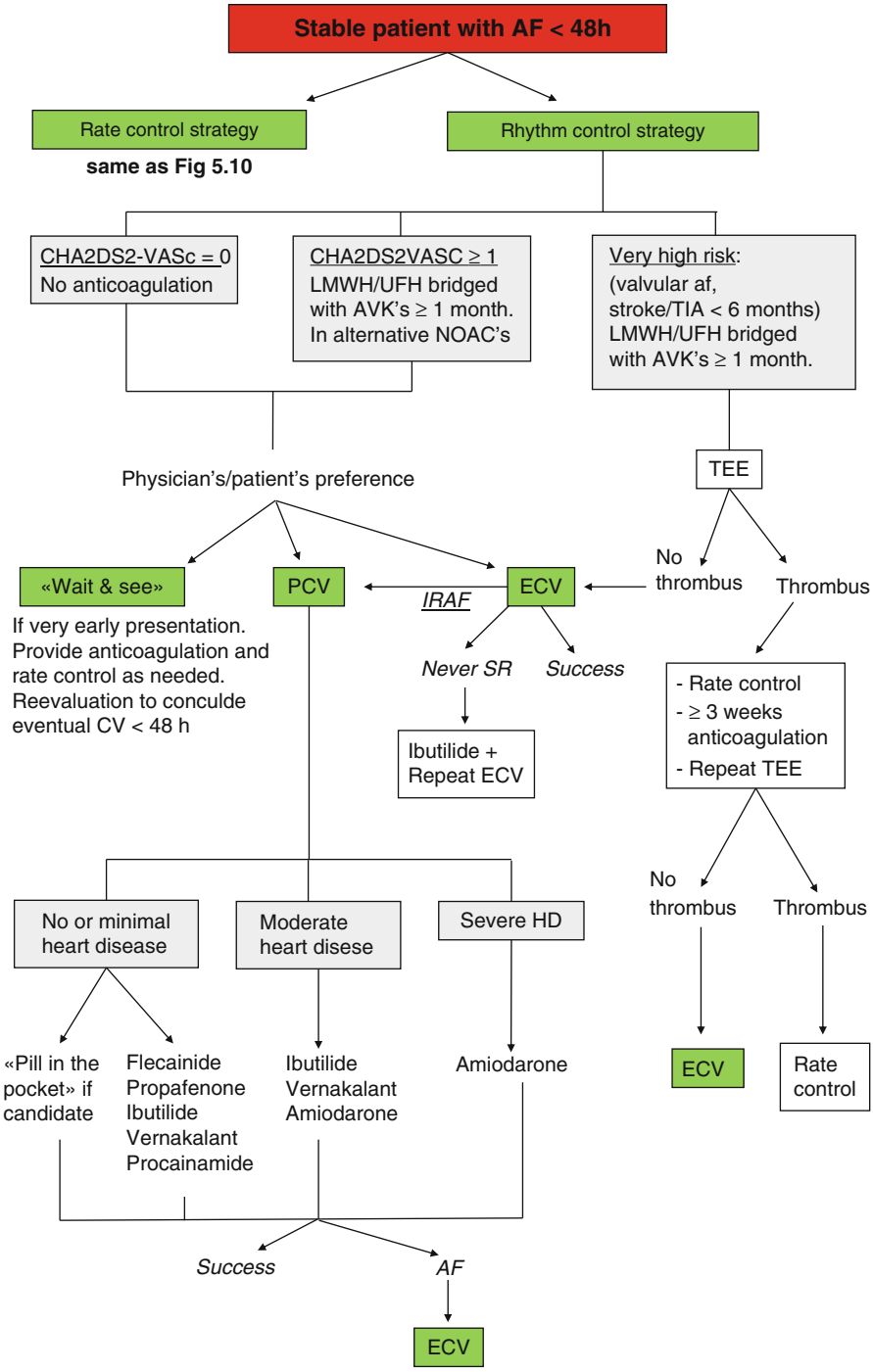

Fig. 5.9 Algorithm for management of patients with stable AF presenting <48 h from onset

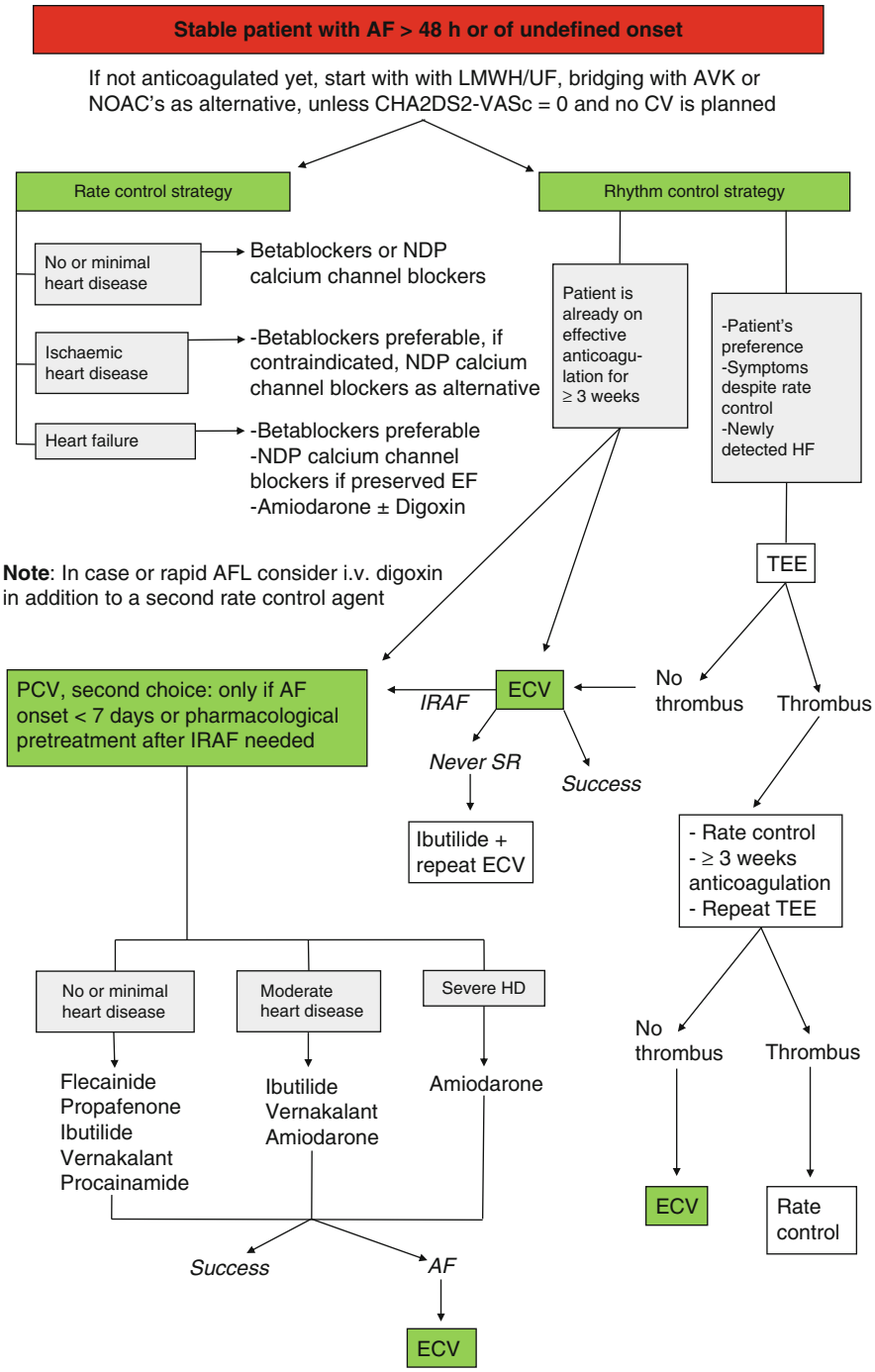

Fig. 5.10 Algorithm for management of patients with stable AF presenting >48 h from onset

A patient who has undergone pharmacological cardioversion should be monitored for a period afterwards (usually half of the drugs elimination's half life) to rule out bradycardia, AV block or ventricular proarrhythmia or hypotension, before discharge. If a patient has been electrically cardioverted, at least 3 h of monitoring is needed [4].

5.4.1 Electrical Cardioversion

Sedation can be provided by different professionals, based on competence, organisation and therapy used. Emergency physicians are involved in the great majority throughout the world for AF cardioversion in the ED. Less often, cardiologists or anaesthesiologists supervise the procedure [21].

A number of medications can be used, like benzodiazepines, narcotics and propofol, alone or in combination. *Midazolam*, a benzodiazepine with a short half-life is gaining increased diffusion because of its pharmacodynamics, wide therapeutic range and availability of a reversal agent (flumazenil). In the two major series [57, 58], adequate sedation was achieved in nearly all patients and no serious complications were observed. Usually, initial bolus doses of 0.01–01 mg/kg are used (the higher ones when the agent is used alone), which may be followed by repeated boli. Mean total dosage used throughout major studies on the use of midazolam as single agent was around 10 mg [59]. *Propofol* is another popular agent for sedation as it has an even more rapid onset of action and washout. On the other hand, it has a greater potential for hypotension and respiratory depression. It is used at dosages of 1–2.5 mg/kg [60].

The procedure can be performed with older monophasic or newer biphasic defibrillators. The latter are more effective with lower energy requirements [61]. The device is connected to the body by a couple of adhesive pads or a couple of handheld paddles.

Four pad positions are currently used: anterolateral, anteroposterior, anterior–left infrascapular and anterior–right infrascapular. They are equally effective to treat atrial or ventricular arrhythmias [62]. If paddles are used (anterolateral position), the operator should press firmly, the optimal force being 8 kg. If the patient has an implantable cardioverter defibrillator (or a pacemaker), anteroposterior or anterolateral locations are acceptable and the nearest pad or paddle should be positioned at least 8 cm away from the device [63]. After the procedure, the device should be interrogated and evaluated to ensure normal function [4]. The DC shock must be synchronised to the R wave to avoid the risk of inducing ventricular fibrillation.

The recommended initial biphasic energy dose for ECV of adult atrial fibrillation is 120–200 Joules (J), whereas for atrial flutter, 50–100 J is usually sufficient. If CV fails, energy should be stepwise increased or the position of the pads changed. For monophasic defibrillators, cardioversion should start at 200 J (100 J for AFL) [10, 62, 63].

Complications They are more often related to arrhythmias, usually transient asystole or bradycardia (0.9 % in the acute CV setting of the Fin CV study [39]).

Pre-cardioversion antiarrhythmic therapy, rate control therapy or slow ventricular rate did not predict bradyarrhythmias, whereas older age, female sex or unsuccessful CV did.

Thromboembolic complications are rare but not negligible if patients are adequately stratified and treated.

Ventricular arrhythmias are exceptional as are episodes of pulmonary oedema.

Complications can rarely be related to sedation like hypotension and prolonged respiratory arrest but are managed with conventional support (fluids and manual ventilation) in the great majority.

5.4.2 Drugs for Rhythm and Rate Control

- Note1: not all drugs or formulations for i.v. or oral use are available in all countries.
- Note 2: for rate and rhythm control drugs, dosages and infusion protocols usually vary between the different guidelines [1, 4, 5, 10, 20, 41]. If there is agreement between sources (or with little variations), one protocol is reported, which also corresponds to the author's practice. If there are marked differences, alternative protocols are mentioned.

5.4.2.1 Rhythm Control Drugs

Flecainide Sodium channel blocker with quite rapid action restores SR in 67–92 % in 6 h. Dosage: 2 mg/kg i.v. over 10 min. Ideal agent for patients with no or minimal heart disease. Avoid in patients with ischaemic heart disease, left ventricular dysfunction, intraventricular conduction delay or Brugada Syndrome. Risks are ventricular proarrhythmia, AV block and electromechanical dissociation. When used for conversion of AFL, 1:1 conduction may ensue with rapid ventricular rate if no rate control agent has been associated. In some countries (USA), approved only for oral use. In this case, 200–300 mg is administered orally (the same dose as the "pill-in-the-pocket" approach), AHA/ACC/HRS 2014 [5] and CCS 2010 [64] guidelines recommending administration of a beta blocker or calcium channel blocker before.

Propafenone Similar properties, indications and efficacy as for flecainide. Dosage: 2 mg/kg i.v. over 10 min. Avoid also in severe COPD because of its weak noncompetitive beta blocking property. In the USA available orally only: administrate 450–600 mg preceded by AV node-blocking agent as above.

Procainamide Sodium channel blocker. Dosage: 15–17 mg/kg i.v. over 1 h with conversion rate around 60 %. Very popular in Canada. Recommended for AF conversion in stable patients in CCS guidelines 2010 [64]. Causes transient hypotension in 5 %.

Amiodarone Multichannel blocker, also with beta- and calcium channel blocking activity if used intravenously. Prolongs prevalently refractoriness in chronic use. Dosage: 5 mg/kg in 1 h, then 50 mg/h for 24 h (ESC 2010 [4]) or 150 mg in 10 min, then 1 mg/min for 6 h, then 0.5 mg/min for 18 h (AHA/ACC/HRS 2014 [5]).

Amiodarone should be diluted in 5 % glucose solution (otherwise there is a risk of crystallisation of the solution). It restores SR with delayed action but similar efficacy as flecainide and propafenone at 12–24 h. It has also significant rate-slowing properties. It is the agent of choice for patients with severe heart disease for both purposes of cardioversion and rate control. Contraindicated in hyperthyroidism, avoid if possible in patients with severe COPD and hypothyroidism. Most common acute side effects are hypotension (especially if infusion is too fast) and phlebitis (the longer the infusion lasts, the more probable).

Ibutilide Rapid-acting agent, prolongs repolarisation. Dosage: 1 mg i.v. over 10 min, if unsuccessful, repeat after 10 min same dose. AF conversion rate is around 50 % with time to conversion about 30 min. Best available conversion agent for AFL. It carries a considerable risk of QTc prolongation and torsades de pointes (4 %). Patients should be monitored for a minimum of 4 h after. Some authors suggest pretreatment with 1–2 g $MgSO_4$ to lower the proarrhythmic risk. Contraindicated if patients are hypotensive, have manifest congestive heart failure, have very low EF (<30 %) and hypokalemic or if QTc >440 ms. Pretreatment with 1 mg over 10 min facilitates great ECV in patients who failed it before.

Dofetilide Prolongs repolarization. Has been mostly studied in the setting of persistent AF. A response may take days or weeks, and therefore it is not an ideal agent in the ED setting. Mentioned and recommended class I only in 2014 AHA/ACC/HRS guidelines [5]. Dosages: 500 mcg BID if creatinine clearance (CrCl) >60, 250 mcg BID if 40–60, 125 mg BID if 20–40. Do not use if CrCl under 20 ml/min. Do not initiate treatment out of hospital (risk of torsades de pointes).

Vernakalant Fast-acting atrial selective multichannel blocker with conversion rate around 50 % and median time to conversion about 10 min. Dosage: 3 mg/kg i.v. over 10 min, followed, if unsuccessful, by second infusion of 2 mg/kg i.v. over 10 min after 15. Contraindicated in hypotensive patients (<100 mmHg), NYHA 3–4 heart failure, FE <35 %, ACS in the last 30 days, severe aortic stenosis and QTc >440 ms.

5.4.2.2 Rate Control Drugs

Beta blockers Very effective, first choice in acute coronary syndrome or heart failure patients (avoid or use with extreme caution in acute decompensation). Do not use in severe COPD and pre-excitation syndrome. Metoprolol, atenolol and propranolol can be used intravenously and orally and are therefore the most useful for acute and chronic treatment. Esmolol is available intravenously only and is frequently used in the intensive care setting. Other beta blockers like bisoprolol or carvedilol are available for oral administration only and can be used if rate control is not too urgent and for chronic therapy.

- Metoprolol: 2.5–5 mg i.v. in 2 min every 5 min up to three doses (max 15 mg), oral maintenance dose 50–200 mg/day
- Atenolol: 2.5 mg i.v. at a rate of 1 mg/min to be repeated at intervals of 5 min up to a maximum of 10 mg
- Propranolol 1 mg i.v. over 1 min up to three doses at 2 min intervals, oral maintenance dose 30–160 mg/day
- Esmolol 0.5 mg/kg i.v. bolus over 1 min, then 50–300 mcg/kg/min i.v.

Nondihydropyridine (NDP) and Calcium Channel Blockers (Diltiazem, Verapamil) Very effective, diltiazem is the less inotropic negative and hypotensive of the two. Avoid in heart failure patients (with the exception of HF with preserved systolic function if not acutelly decompensated) and pre-excitation syndrome.

- Verapamil: 0.075–0.15 mg/kg i.v. bolus over 2 min, may repeat 10 mg bolus after 30 min if uneffective, then 0.005 mg/kg/min infusion [5]. Oral maintenance dose 160–360 mg/day
- Diltiazem 0.25 mg/kg i.v. bolus over 2 min, may give a second dose of 0.35 mg/kg, then 5–15 mg/h infusion. Oral maintenance dose 180–360 mg/day

Amiodarone Usually second-line agent. It is well tolerated in critically ill patients where it can be the first choice.

Digoxin Usually a second-line agent with moderate and delayed efficacy on rate control. When used intravenously, onset of action takes 1 h and peak rate-slowing activity is reached at 6 h. It is first-line agent in patients with acute heart failure. Due to its vagomimetic activity, it tends to transform atrial flutter in fibrillation (about 25 %, personal observation) so it turns out very helpful in rate control for flutter, which is harder to achieve than for AF. In the author's practice, digoxin is used as first-line agent, associated with another rate control drug when fixed. 2:1 AV conduction with rapid ventricular rate is present. Avoid in moderate or severe renal failure due to risk of intoxication.

Dosage 0.25–0.5 mg i.v. with repeat dosing, maximum 1.5 mg/24 h. Oral maintenance dose 0.0625–0.25 mg/day

5.5 Indications for Hospitalisation, Follow-Up and Referral

Hospitalisation should be in general considered for patients with AF and severe symptoms (heart failure, severe angina, syncope), when a significant heart disease is suspected, important comorbidities are present, symptoms are not properly controlled (failed cardioversion or rate control) or if complications occur during first approach treatment.

Decision algorithms like RED-AF [65] and AFFORD [66] have recently developed for predicting 30 days adverse events in AF patients presenting to ED with AF. Nomograms contain many clinical predictors and points are assigned to each predictor. Adverse events vary from ED return visit for recurrent AF to death, through different grades of severity. The AFFORD [66] model fared well in risk prediction and in the identification of patients at very low risk which the authors stated could be safely discharged after cardioversion or adequate rate control, but these were only 17 % of the total. This still poses the problem of the acceptable risk threshold for indicating hospitalisation (e.g. is it worth to hospitalise a patient to avoid return visit for AF relapse? Is stroke preventable by hospitalisation if patient is properly anticoagulated also at home?). Furthermore, the same authors

admit that the effect of hospitalising ED patients with acute AF on short-term adverse events remains unanswered [65] (RED-AF). Uncertainties on hospitalisation criteria are still reflected by the wide discrepancy of the discharge rate after successful CV: 85 % in Canada, 76 % in Australasia, 48 % in the USA and 27 % in the UK [21].

In the absence of obvious severe presentation or comorbidities, careful individual judgement on high-risk features should be performed to guide this decision.

It should be stressed that new-onset AF carries a worse prognosis [7, 18]. In the study of Miyasaka et al. [67], hazard ratio for mortality risk was 9.6 within the first 4 months and 1.7 thereafter, compared with age- and gender-matched general population. The main reason is thought to be that AF complicates underlying conditions with poor prognosis like cardiovascular diseases or malignancies. As a consequence, patients with first episode should undergo a rapid medical workup which can be done of course during hospitalisation, but alternatively, if there is no clinical instability, on outpatient basis. In order to favour the latter strategy, diagnostic pathways which include evaluation by cardiologist and other professionals as well as diagnostic testings should be defined locally and be easily accessible, which is unfortunately not always the case and gives reason for the high percentage of hospitalisation of AF patients in some countries [23].

Follow-up of AF patients should be individualised and take into account symptomatic status, comorbidities (which should be followed up as specifically needed), antiarrhythmic treatment, thromboprophylaxis and complications. Visits should be usually performed by the primary care physician or a cardiologist.

Asymptomatic patients in sinus rhythm without medications and comorbidities do not need a structured follow-up, and a periodic self-check of pulse regularity should be recommended.

Patients on antiarrhythmic or on rate control medications should get an ECG 1 week after beginning or up-titration of therapy to check for efficacy and proarrhythmic complications and a visit every 6–12 months.

At every follow-up visit, the following issues should be reassessed:

- Effectiveness of symptom control either while pursuing rate or rhythm control strategy
- Need for antithrombotic therapy, re-evaluation of risks and benefits
- Optimization of therapy for comorbidities
- Lifestyle counselling and check for adequate compliance

Furthermore, in relation to individual therapy, there may be a need for periodical lab tests (e.g. thyroid function for patients on amiodarone, liver function for patients on dronedarone, blood count and renal function for patients using novel anticoagulants, etc.).

Echocardiography should be performed as recommended for underlying structural heart disease or in case of worsening symptoms despite apparent achievement of rate or rhythm control. Holter recording may be needed for assessment of

adequate rate control or if proarrhythmia is suspected. An exercise stress test can help to adjust rate control medications in active patients with permanent AF who complain uncontrolled palpitations during exercise.

Referral to a cardiologist *is generally indicated for patients with a first AF episode.* A possible exception is older patients with asymptomatic AF with adequate rate control. Basic screening examinations like laboratory tests, thyroid function and echocardiography should be arranged before referral. Other reasons for referral to a cardiologist are inadequate rhythm control strategy (ineffective or not tolerated antiarrhythmic medications), ineffective rate control strategy (inadequate rate control or poor quality of life despite adequate rate control) and evaluation for radiofrequency ablation.

References

1. Camm AJ, Lip GYH, De Caterina R, et al. 2012 focused update of the ESC guidelines for the management of atrial fibrillation. An update of the 2010 ESC guidelines for the management of atrial fibrillation. Eur Heart J. 2012;33:2719–47.
2. Santini M, De Ferrari GM, Pandozi C, et al. Atrial fibrillation requiring urgent medical care. Approach and outcome in the various departments of admission. Data from the atrial Fibrillation/flutter Italian Registry (FIRE). Ital Heart J. 2004;5:205–13.
3. Coll-Vinent B, Martìn A, Malagòn F, et al. Stroke prophylaxis in atrial fibrillation: searching for management improvement opportunities in the emergency department: the HERMES-AF Study. Ann Emerg Med. 2015;65:1–12.
4. Camm JA, Kirchhof P, Lip GYH, et al. Guidelines for the management of atrial fibrillation. The task force for the management of atrial fibrillation of the European Society of Cardiology (ESC). Eur Heart J. 2010;31:2369–429.
5. January CT, Wann LS, Alpert JS, et al. 2014 AHA/ACC/HRS Guideline for the management of patients with atrial fibrillation. A report of the American College of Cardiology/American Heart Association and the Heart Rhythm Society task force of practical guidelines. J Am Coll Cardiol. 2014;64:2246–80.
6. Thrall G, Lane D, Carrol D, et al. Quality of life in patients with atrial fibrillation: a systematic review. Am J Med. 2006;119:448e1–448e19.
7. Nieuwlaat R, Cappucci A, Camm JA, et al. Atrial fibrillation management: a prospective survey in ESC member countries. Eur Heart J. 2005;26:2422–34.
8. Oishi ML, Xing S. Atrial fibrillation: management strategies in the emergency department. Emerg Med Pract. 2013;15:1–26.
9. Neumar R, Otto CW, Link MS, et al. Adult advanced cardiovascular life support: part 8. 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation. 2010;122 Suppl 3:S 729–767.
10. Verma A, Cairns JA, Mitchell LB, et al. 2014 focused update of the Canadian Cardiovascular Society guidelines for the management of atrial fibrillation. Can J Cardiol. 2014;30:1114–30.
11. Nabauer M, Gerth A, Limbourg T, et al. The registry of german competence network on atrial fibrillation: patient characteristics and initial management. Europace. 2009;11:423–34.
12. Buccelletti F, Carroccia A, Marsiliani D, et al. Utility of routine thyroid-stimulation hormone determination in new-onset atrial fibrillation in the ED. Am J Emerg Med. 2011;29:1158–62.
13. Bellone A, Etteri M, Vettorello M, et al. Cardioversion of acute atrial fibrillation in the emergency department: a prospective randomised trial. Emerg Med J. 2012;29:188–91.
14. Healey JS, Parkash R, Pollak T, et al. Canadian Cardiovascular Society guidelines 2010: etiology and initial investigations. Can J Cardiol. 2011;27:31–7.

15. Atzema C, Lam KM, Young C, et al. Patients with atrial fibrillation and an alternative primary diagnosis in the emergency department: a description of their characteristics and outcomes. Acad Emerg Med. 2013;10:193–9.
16. Scheuermeier FX, Pourvali R, Rowe BH, et al. Emergency department patients with atrial fibrillation or flutter and acute underlying medical illness may not benefit from attempts to control rate or rhythm. Ann Emerg Med. 2015;65:511–22.
17. Wyse DG, Waldo AL, Di Marco JP, The atrial fibrillation follow-up investigation of rhythm management (AFFIRM) investigators, et al. A comprison of rate control and rhythm control in patients with atrial fibrillation. N Engl J Med. 2002;347:1825–33.
18. Camm JA, Breithardt G, Crijns H, et al. Real-life observations of clinical outcomes with rhythm- and rate control therapies for atrial fibrillation. RECORDAF (registry on cardiac rhythm disorders assessing the control of atrial fibrillation). J Am Coll Cardiol. 2011;58:493–501.
19. Alam M, Jamaluddin Bandeali S, Shahzad SA, et al. Real–life global survey evaluating patients with atrial fibrillation (REALISE-AF): results of an international observational registry. Expert Rev Cardiovasc Ther. 2012;10:283–91.
20. Raviele A, Disertori M, Alboni P, et al. Linee guida AIAC per la gestione ed il trattamento della fibrillazione atriale. Aggiornamento 2013. G Ital Cardiol. 2013;14:215–40.
21. Rogenstein C, Kelly A-M, Mason S, et al. An international view of how recent onset atrial fibrillation is treated in the emergency department. Acad Emerg Med. 2012;19:1255–60.
22. Stiell IG, Birnie D. Clinical controversies: in a vacuum of good evidence, rhythm control is the sensible option for patients with symptomatic recent onset atrial fibrillation or flutter. Ann Emerg Med. 2011;57:31–2.
23. Cohn BG, Keim SM, Yealy DM. Is emergency department cardioversion of recent-onset atrial fibrillation safe and effective? J Emerg Med. 2013;45:117–27.
24. Stiell IG, Clement CM, Perry JJ, et al. Association of the "Ottawa aggressive protocol" with rapid discharge of emergency department patients with recent-onset atrial fibrillation or flutter. CJEM. 2010;12:181–91.
25. Danias PG, Caulfield TA, Weigner MJ, et al. Likelihood of spontaneous conversion of atrial fibrillation to sinus rhythm. J Am Coll Cardiol. 1998;31:588–92.
26. Vinson DR, Hoehn T, Graber DJ, et al. Managing emergency department patients with recent-onset atrial fibrillation. J Emerg Med. 2012;42:139–48.
27. Decker WW, Smars PA, Vaidjanathan L, et al. A prospective randomized trial of an emergency department observation unit for acute onset atrial fibrillation. Emerg Med. 2008;52:322–8.
28. De Caterina R, Husted S, Wallentin L, et al. Parenteral anticoagulants in heart disease: current status and perspectives (section II). Position paper of the ESC working group on thrombosis – task force on anticoagulants in heart disease. Thromb Haemost. 2013;109:769–86.
29. Klein AL, Jasper SE, Katz WE, et al. The use of enoxaparin compared with unfractionated heparin for short-term antithrombotic therapy in atrial fibrillation patients undergoing transoesophageal echocardiography–guided cardioversion: assessment of cardioversion using transoesophageal echocardiography (ACUTE) II randomized multicenter study. Eur Heart J. 2006;27:2858–65.
30. Stellbrink C, Nixdorff U, Hofmann T, et al. Safety and efficacy of enoxaparin compared with unfractionated heparin and oral anticoagulants for prevention of thromboembolic complications in cardioversion with nonvalvular atrial fibrillation: the anticoagulation in cardioversion using enoxaparin (ACE) trial. Circulation. 2004;109:997–1003.
31. Nagarakanti R, Ezekowitz MD, Oldgren J, et al. Dabigatran versus warfarin in patients with atrial fibrillation. An analysis on patients undergoing cardioversion. Circulation. 2011;123:113–36.
32. Cappato R, Ezekowitz MD, Klein AL, et al. Rivaroxaban vs vitamin K antagonists for cardioversion in atrial fibrillation. Eur Heart J. 2014;35:3346–55.
33. Flaker G, Lopes RD, Al-Kahib SM, et al. Efficacy and safety of apixaban after cardioversion for atrial fibrillation. J Am Coll Cardiol. 2014;62:1082–7.

34. Van der Werf F, Brueckmann M, Connolly SJ, et al. A comparison of dabigatran etexilate with warfarin in patients with mechanical heart valves: the randomized, phase II study to evaluate the safety and pharmacokinetics of oral dabigatran etexilate in patients after heart valve replacement (RE-ALIGN). Am Heart J. 2012;163:931–7.
35. Lip GY, Frison L, Halperin J, et al. Identifying patients at risk of stroke despite anticoagulation. Stroke. 2010;41:2731–5.
36. Weigner MJ, Caufield TA, Danias PJ, et al. Risk for clinical thromboembolism associated with conversion to sinus rhythm in patients with atrial fibrillation lasting less than 48 hours. Ann Intern Med. 1997;126:615–20.
37. Kleemann T, Becker T, Strauss M, et al. Prevalence of left atrial thrombus and dense spontaneous echo-contrast in patients with short term atrial fibrillation <48 hours undergoing cardioversion: value of transesophageal echocardiography to guide cardioversion. J Am Soc Echocardiogr. 2009;22:1403–8.
38. Stoddart MF, Dawkins PR, Prince CR, et al. Transesophageal echocardiographic guidance of cardioversion in patients with atrial fibrillation. Am Heart J. 1995;129:1204–15.
39. Airaksinen KEJ, Groeneberg T, Nuotio I, et al. Thromboembolic complications after cardioversion of acute atrial fibrillation. The FinCV (Finnish CardioVersion) Study. J Am Coll Cardiol. 2013;62:1187–92.
40. Alboni P, Botto GL, Baldi N, et al. Outpatient treatment of recent onset atrial fibrillation with the "pill in te pocket" approach. N Engl J Med. 2004;351:2384–91.
41. Gillis AM, Verma A, Talajic M, et al. Canadian Cardiovascular Society guidelines 2010: rate and rhythm control management. Can J Cardiol. 2011;27:47–59.
42. Glatter K, Yang Y, Chatterjee K, et al. Chemical cardioversion of atrial fibrillation or flutter with ibutilide in patients receiving amiodarone therapy. Circulation. 2001;103:253–7.
43. Hongo RH, Themistoclakis S, Raviele A, et al. Use of ibutilide in cardioversion of patients with atrial fibrillation or atrial flutter treated with class IC agents. J Am Coll Cardiol. 2004;44:864–8.
44. Sugiura T, Iwasaka T, Ogawa A, et al. Atrial fibrillation i myocardial infarction. Am J Cardiol. 1985;56:27–9.
45. Steg G, James SK, Atar D, et al. ESC guidelines for the management of acute myocardial infarction presenting with ST-segment elevation. Eur Heart J. 2012;33:2569–619.
46. Gupta K, Pillarisetti J, Biria M, et al. Clinical utility and prognostic significance of measuring troponin I levels in patients presenting to the emergency room with atrial fibrillation. Clin Cardiol. 2014;37:343–9.
47. Conti A, Mariannini Y, Canuti E, et al. Role of masked coronary heart disease in patients with recent onset atrial fibrillation and troponin elevations. Eur J Emerg Med. 2015;22:162–9.
48. Van den Bos E, Constantinescu AA, van Domburg RT, et al. Minor elevations in troponin I are associated with mortality and adverse cardiac events in patients with atrial fibrillation. Eur Heart J. 2011;32:611–7.
49. Hijazi Z, Oldgren J, Andersson U, et al. Cardiac biomarkers are associated with an increased risk of stroke ad death in patients with atrial fibrillation. A randomized evaluation of long-term anticoagulation therapy (RE-LY) substudy. Circulation. 2012;125:1605–16.
50. Meshkat N, Austin E, Moineddin R, et al. Troponin utilization in patients presenting with atrial fibrillation/flutter to the emergency department: retrospective chart review. Int J Emerg Med. 2011;4:25.
51. Pradhan R, Chaudhary A, Donato AA. Predictive accuracy of ST depression during rapid atrial fibrillation in the presence on obstructive coronary artery disease. Am J Emerg Med. 2012;30:1042–7.
52. Tsigkas G, Kopsida G, Xanthoupoulou I, et al. Diagnostic accuracy of electrocardiographic ST segment depression in patients with rapid atrial fibrillation for the prediction of coronary artery disease. Can J Cardiol. 2014;30:920–4.
53. Androulakis A, Aznaouridis KA, Aggeli CJ, et al. Transient ST-segment depression during paroxysms of atrial fibrillation in otherwise normal individuals. J Am Coll Cardiol. 2007;19:1909–13.

54. Yancy CW, Jessup M, Bozkurt B, et al. 2013 ACCF/AHA guideline for the management of heart failure: a report of the American College of Cardiology Foundation/American Heart Association task force on practice guidelines. Circulation. 2013;128:e240–327.
55. Crijns HJGM, Weijs B, Fairley A-M, et al. Contemporary real life cardioversion of atrial fibrillation: results from the multinational RHYTHM-AF study. Int J Cardiol. 2014;172:588–94.
56. Oral H, Souza JJ, Michaud GF, et al. Facilitating electrical cardioversion of atrial fibrillation with ibutilide pretreatment. N Engl J Med. 1999;340:1849–59.
57. Huebner PJ, Gupta S, McClellan I, et al. Simplified cardioversion service with intravenous midazolam. Heart. 2004;90:1447–9.
58. Notarstefano P, Pratola C, Toselli T, et al. Sedation with midazolam for electrical cardioversion. PACE. 2007;30:608–11.
59. Thomas SP, Thakkar J, Kovoor P, et al. Sedation for electrophysiological procedures. PACE. 2014;37:781–90.
60. Wood J. Procedural sedation for cardioversion. Emerg Med J. 2006;23:932–4.
61. Mittal S, Ayati S, Stein KM, et al. Transthoracic cardioversion of atrial fibrillation: comparison of rectilinear biphasic versus damped sine wave monophasic shocks. Circulation. 2000;101:1282–7.
62. Link MS, Atkins DL, Passmann RS, et al. American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 6: electrical therapies. Automated external defibrillators, defibrillation, cardioversion and pacing. Circulation 2010; 2010;122 Suppl 3:S706–19.
63. Deakin DD, Nolan JP, Sunde K, et al. European Resuscitation Council guidelines for resuscitation 2010 Section 3. Electrical therapies: automated external defibrillators, defibrillation, cardioversion and pacing. Resuscitation. 2010;81:1293–304.
64. Stiell IG. Macle L and the CCS Atrial Fibrillation Guidelines Committee: Canadian Cardiovascular Society guidelines 2010: management of recent-onset atrial fibrillation and flutter in the emergency department. Can J Cardiol. 2010;27:38–46.
65. Barret TW, Jenkins CA, Self WH. Validation of the risk estimator decision aid for atrial fibrillation (RED-AF) for predicting 30-day adverse events in emergency department patients with atrial fibrillation. Ann Emerg Med. 2015;65:13–21.
66. Barrett TW, Storrow AB, Jenkins CA, et al. The AFFORD clinical decision aid to identify emergency department patients with atrial fibrillation at low risk for 30-day adverse events. Am J Cardiol. 2015;115:763–70.
67. Miyasaka Y, Barnes ME, Bailey KR, et al. Mortality trends in patients diagnosed with first atrial fibrillation. J Am Coll Cardiol. 2007;49:986–92.

Wide QRS Complex Tachycardia in the Emergency Setting

6

Giuseppe Oreto, Francesco Luzza, Gaetano Satullo, Antonino Donato, Vincenzo Carbone, and Maria Pia Calabrò

6.1 Wide QRS Complex Tachycardia

A wide QRS complex tachycardia can be (1) ventricular tachycardia (VT); (2) supraventricular tachycardia (SVT) with bundle branch block that may be either preexisting or due to aberrant conduction, namely, tachycardia-dependent abnormal intraventricular conduction; a further possibility is the effect of some antiarrhythmic drugs that slow down intraventricular conduction, resulting in marked QRS complex widening; and (3) supraventricular tachycardia with conduction of impulses to the ventricles over an accessory pathway (preexcited tachycardia).

In the presence of wide QRS tachycardia, the correct diagnosis is of paramount importance, since the treatment commonly used in SVT is different from that of VT, and some drugs useful in the former (e.g., verapamil) are harmful in the latter [1–3].

The origin of a wide QRS complex tachycardia can be reliably identified using a "holistic" approach, namely, taking into account all of the available items: no single criterion is able to provide a simple and quick solution of the problem in all cases. The available ECG signs are, without any exception, suggestive of ectopy, namely, ventricular origin of the impulses; SVT with aberrant conduction may only be diagnosed by excluding all of the items favoring VT. The recognition of ventricular or supraventricular origin of wide QRS complex tachycardias is not difficult if a

G. Oreto (✉) • F. Luzza • V. Carbone
Department of Clinical and Experimental Medicine, University of Messina, Messina, Italy
e-mail: goreto@unime.it

G. Satullo • A. Donato
Department of Cardiology, "Papardo" Hospital, Messina, Italy

M.P. Calabrò
Department of Pediatrics, University of Messina, Messina, Italy

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detailed analysis is used, taking into account several diagnostic signs: [4–12] the idea that a single quick item can offer an immediate and reliable solution is something of an illusion.

If, despite a complete diagnostic approach, the dilemma cannot be resolved, it is necessary to assume a ventricular origin of the arrhythmia since (1) a wide QRS tachycardia is more likely VT than SVT and (2) it is less dangerous to treat an SVT like it were ventricular in origin than applying to a patient with VT the treatment commonly used for SVTs. In particular, intravenous verapamil should be avoided whenever SVT diagnosis is not certain, since this drug is harmful in some VT patients [1–3].

6.2 General Criteria

6.2.1 Atrioventricular Dissociation

Whenever the electrical activity of the atria is recognizable, two different situations may occur:

1. Atrioventricular (A-V) dissociation
2. Relationship between P waves and QRS complexes.

A-V dissociation demonstrates the ventricular origin of the wide QRS complexes and occurs in a percentage variable from 19 to 70 % of VT cases [5, 6, 10, 11]. Dissociation, however, is often difficult to be diagnosed since in several cases, sinus P waves are not easily recognizable, being simultaneous to QRS complexes or T waves. Moreover, in the presence of atrial fibrillation, A-V dissociation cannot be appreciated. Before excluding, in a wide QRS complexes tachycardia, the presence of P waves independent of QRS complexes, however, one should observe with great attention the configuration of several consecutive complexes in all 12 leads, paying the greatest attention to leads II and V1 (the ones where sinus P waves are usually evident). Aim of this analysis is comparing consecutive complexes searching for slight differences in QRS or T morphology: with this approach it is not rare to discover, in the presence of VT, that in some leads, slight variations in QRS complex or T wave configuration occur. To be sure that such differences express the presence of P waves dissociated from QRS complexes, and superimposed on these, it is necessary to measure the intervals separating the "disturbing" events: in case of A-V dissociation, they are separated from relatively constant intervals, being "long" intervals in multiples of the "short" ones (Fig. 6.1a). When, in contrast, the intervals separating the changes in morphology of T waves and/or QRS complexes are irregular, it is more likely that artifacts, rather than dissociated sinus P waves, are involved (Fig. 6.1b).

The best ECG leads to be analyzed, searching for "dissociated" P waves, are leads II and V1, the ones where sinus P wave voltage is usually relatively high; it is also advisable to observe the leads where the QRS complex and/or the T wave is of

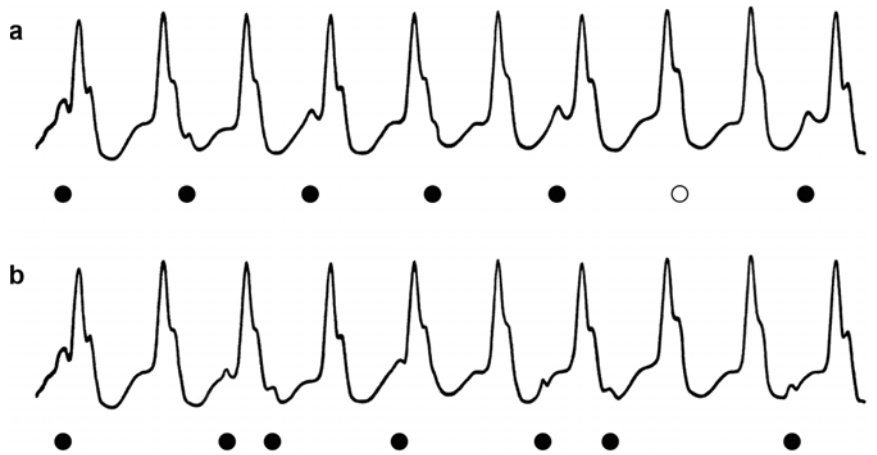

Fig. 6.1 Diagrams (**a**, **b**) show two wide QRS complex tachycardias. In both diagrams, small positive deflections, independent of QRS complexes, are present. In diagram (**a**) these deflections are rhythmic and separated by constant intervals; whenever a deflection is invisible, being coincident with a QRS complex (*circle*), the interval between two manifest waves is twice the basic interval. These small waves are, therefore, sinus P waves: accordingly, A-V dissociation can be diagnosed, revealing a ventricular origin of tachycardia. In diagram (**b**), in contrast, the small positive deflections are arrhythmic: they are not P waves but artifacts

low voltage, since it is relatively easy to detect the small atrial waves whenever these are not "buried" within large QRS or T deflections. This is expressed by the "haystack principle": if you are searching for a needle in a haystack, select a small haystack" (Fig. 6.2).

The bedside diagnosis of A-V dissociation can be improved by heart sound auscultation and arterial pulse palpation: whenever the atrial contraction is dissociated from ventricular activity, it is possible to appreciate a variable loudness of the 1st heart sound and variability in peripheral pulse amplitude. This is because (1) whenever atrial contraction occurs immediately before ventricular systole, the blood flow "opens" the atrioventricular valves, resulting in a relatively loud 1st heart sound, a phenomenon that does not occur if mitral and tricuspid valves are closed at the time of atrial systole, and (2) if atrial contraction occurs when the A-V valves are open, the diastolic ventricular filling is improved, resulting in a relatively increased stroke volume: accordingly, the pulse amplitude will be higher with respect to that of heart beats in which atrial systole occurs while the A-V valves are closed.

6.2.2 Second-Degree V-A Block

In ventricular tachycardia, atrial electrical activity may be not dissociated from ventricular one if retrograde ventricular-atrial (V-A) conduction occurs, as it happens in about one half of cases. The V-A ratio may be 1 (every QRS complex is followed by a

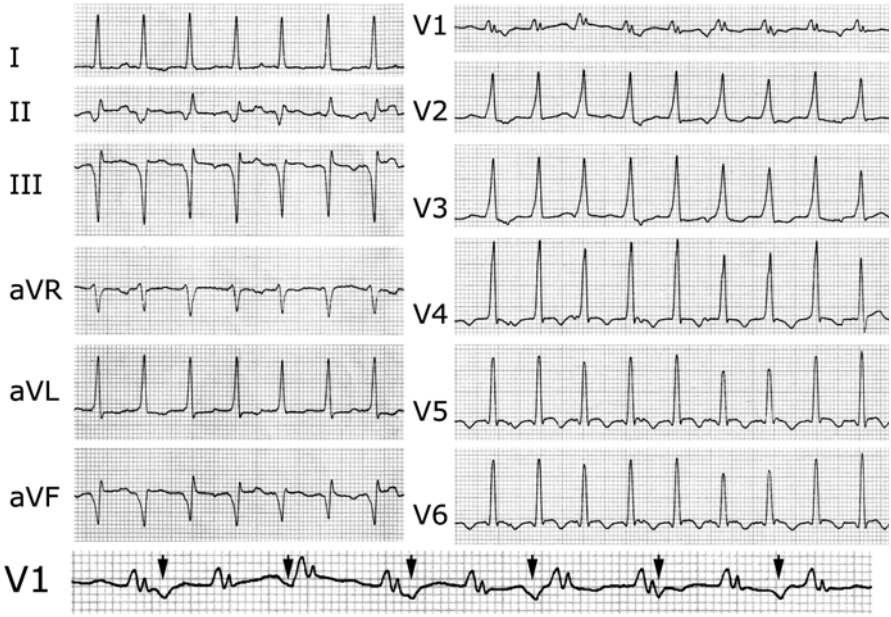

Fig. 6.2 Wide QRS complex tachycardia. QRS duration is 0.12 s, but since in some leads ventricular complexes are relatively narrow, a supraventricular tachycardia could be diagnosed at first glance. The ventricular origin of tachycardia is demonstrated by A-V dissociation; the P waves independent of ventricular complexes (*arrows*), and separated from constant intervals, are easily recognized in lead V1, since in this lead both QRS complexes ant T wave voltages are very low (the haystack principle)

retrograde P wave) or less than 1 when some ventricular impulses are not conducted to the atria. In a wide QRS complex tachycardia, a QRS/P ratio >1 (more QRS complexes than P waves) demonstrates the ventricular origin of the arrhythmia [6–8, 12], (Fig. 6.3), whereas a 1:1 ratio does not permit any definite conclusion since P waves may (a) express a supraventricular tachycardia with 1:1 A-V conduction or (b) represent the retrograde atrial activation during ventricular tachycardia. If analysis of P wave configuration is possible, a main P vector directed inferiorly demonstrates supraventricular origin of the arrhythmia, whereas a P vector directed superiorly (negative P waves in the inferior leads) does not permit any conclusion since not only VT but also several supraventricular tachycardias share a retrograde activation of the atria. In some cases of VT, however, retrograde P waves appear as positive in the inferior leads, a phenomenon that has been called "the illusion of retrograde positive P waves" (Fig. 6.4) [8, 13, 14].

6.2.3 Capture and Fusion Beats

The presence of narrow, or relatively narrow, beats during wide QRS tachycardia suggests a diagnosis of VT, *provided that narrow complexes are preceded by a P wave with an interval consistent with anterograde A-V conduction of the impulse*. The narrow, or less wide, complexes are *capture* or *fusion* beats that occur

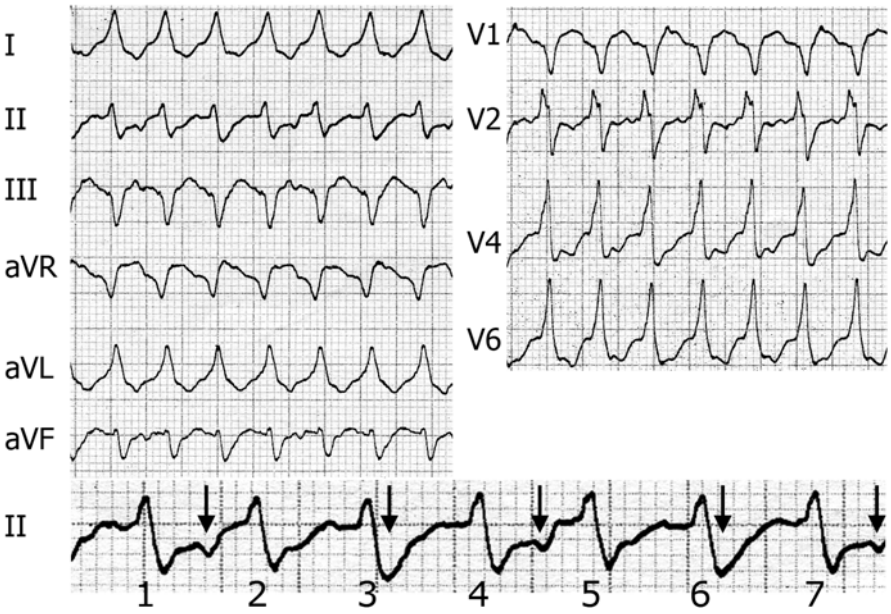

Fig. 6.3 Ventricular tachycardia with 3:2 retrograde block of the Wenckebach type. Lead II (enlarged in the *bottom row*) analysis reveals that ventricular complexes *1*, *4,* and *7* are followed by negative P waves occurring midway between two consecutive QRS complexes. Beats *3* and *6*, in turn, show very wide "S waves" that never occur in the other beats, whereas complexes *2* and *5* do not show any of the 2 above characteristics (negative P wave, wide "s wave"). It is, therefore, evident that in a group of 3 beats, the 1st one (complexes *3* and *6*) is followed by a retrograde P wave with a short R-P interval, whereas the P wave following the 2nd beat of the group (complexes *1*, *4*, *7*) occurs with a relatively long interval, and after the 3rd complex of the series (beats *2* and *5*), no P wave occurs. In other words, there is a retrograde 2nd-degree 3:2 Wenckebach type block, and this establishes the diagnosis of ventricular tachycardia. In this tracing, the r wave peak time in lead II is 30 ms

whenever, during VT, a sinus or supraventricular impulse succeeds in reaching the ventricles, whose depolarization is due totally (capture) or partially (fusion) to that impulse (Fig. 6.5). Capture and fusion beats are reliable signs of VT, but they are rare (4 % in a study based on 96 cases of proved VT [10]) and can be observed only in the presence of A-V dissociation. Since the latter phenomenon is, in itself, a clear sign of VT, the further help provided by capture and fusion beats is trivial, provided that these never occur whenever the heart rate is very high, being the A-V node made always refractory by ectopic ventricular impulses [6, 8].

6.2.4 Precordial QRS Concordance

A "concordant" QRS morphology in all the precordial leads, namely, ventricular complexes totally negative (QS pattern) or positive (R or qR pattern), demonstrates a ventricular origin of tachycardia, since no intraventricular conduction