Arrhythmia Induction in the EP Lab
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The practice of electrophysiology requires a good understanding of intracardiac electrical signals, but beyond that it needs practical application of the theoretical principles. Induction of arrhythmia is the cornerstone of electrophysiology. Understanding the signals implies having the arrhythmia in front of the eyes, struggling to deduce the initiation, morphology, and timeline of the signals.

There have been remarkable advances in the mapping techniques, imaging, and catheters used for catheter ablation, but arrhythmia induction can be obtained by using the same old drugs described in this book.

*How to Induce Arrhythmias* was written to assist EP practitioners, fellows, and nurses in finding the doses and specific medications in the EP lab when the clinical arrhythmia cannot be induced by classical programmed stimulation. In the first chapters, classical drugs are described: isoprenaline, orciprenaline, and atropine, as well as old drugs, used when the first two are unavailable—adrenaline, noradrenaline, dopamine, and dobutamine. Other various medications rarely used in the EP lab are also described because case reports showed their usefulness in clinical settings: aminophylline, adenosine, salbutamol, and ephedrine. Specific doses for children are shown in a chapter dedicated to pediatric electrophysiology. Two distinct chapters on energy drinks and caffeine were written because anecdotic cases reported sustained arrhythmias as side effects after their use.

The text is written in the same style for all chapters, in a simple way, following the same structure, with drawings, figures, and tables that assist easy finding of the information for a specific drug: all chapters start with an introduction, the chemical structure of the product, pharmacology, and mechanism of action; continue with doses, protocol of administration, side effects, and contraindications; and finish with the electrophysiological effects of the drug in animal and humans for different types of arrhythmias.

Electrophysiologists at different levels will find this book useful. Beginners will find out what medication can be used to facilitate arrhythmia induction, what is the sensibility and specificity of different drugs, how to administrate the medication, and what side effects to expect. EP practitioners will gain knowledge by reviewing the doses they might have long forgotten.

We would like to receive your critique and positive feedback on ResearchGate so that a new edition of *Arrhythmia Induction in the EP Lab* can improve to meet EP’s needs.

Cluj-Napoca, Romania

Gabriel Cismaru
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About the Author

Gabriel Cismaru received his doctorate in medicine from the “Iuliu Hatieganu” University of Medicine and Pharmacy, Cluj-Napoca, in 2016, and earned his MD degree from the same university in 2005. After completing his residency in cardiology, Dr. Cismaru began his electrophysiology fellowship at the Institut Lorrain du Coeur et des Vaisseaux Louis Mathieu, Nancy, France. In 2011, he started to work at the Electrophysiology Laboratory of the Rehabilitation Hospital Cluj-Napoca. He has authored or coauthored peer-reviewed articles and book chapters in the field of electrophysiology and cardiac pacing.

The new advances in cardiac arrhythmia techniques increased the success rate of complete treatment for patients suffering from arrhythmias. However, the reproduction of the clinical arrhythmia might be difficult in hospital settings.

Arrhythmia Induction in the EP Lab is an extension of a PhD thesis in the field of cardiac electrophysiology. Various chapters describe methods to induce clinic arrhythmias, with a special chapter dedicated to arrhythmia induction in children. These topics are of great interest to electrophysiologists that deal with patients suffering from different types of arrhythmias.
Introduction: Why Do We Need Arrhythmia Induction?

Sorin Lazar

Abbreviations

AP Accessory pathway
AT Atrial tachycardia
AVNRT Atioventricular reentry (or reciprocating) tachycardia
AVRT Atrioventricular reentrant (or reciprocating) tachycardia
EKG Electrocardiogram
EP Electrophysiology
ORT Orthodromic reciprocating tachycardia
PVC Premature ventricular contractions
Short RP tachycardia Short R wave to P wave tachycardia
SVT Supraventricular tachycardia
VT Ventricular tachycardia
1.1 Better Outcome of Catheter Ablation When Clinical Arrhythmia Is Inducible

One of the main challenges in evaluating a patient with palpitations is determining the type of arrhythmia that is responsible for the patient’s symptoms. Many of the available outpatient monitors have limitations in the number of channels they can record or the amount of data they can store for analysis. In many instances, the initiation and termination of the arrhythmia are not captured on these monitors, and essential information for a correct diagnosis is not available. Ideally, the arrhythmia should be recorded on a 12-lead electrocardiogram for a correct diagnosis, but most of the time, this is possible only when the arrhythmia is sustained long enough to be still present when the patient arrives at the hospital. In the best case scenario, the arrhythmia is recorded on a heart monitor or 12-lead EKG, and then a pre-ablation strategy can be developed. Depending on the protocol used to induce arrhythmia, the clinical arrhythmia might be inducible along with other arrhythmias which might not be clinical. Knowing the type of arrhythmia the patient has as outpatient helps guide the ablation of the inducible arrhythmia that has similar characteristics, rather than map and ablate all inducible arrhythmias in the EP lab.

One example of this is a patient with palpitations and WPW at baseline, which suggest possible AVRT, but during the EP study, either the accessory pathway (AP) has an ERP that would make AVRT unlikely, with the clinical arrhythmia being AT or AVNRT and AP being a bystander [1].

Another example is PVCs with morphology suggesting outflow tract origin, but during the EP study with isoproterenol infusion, multiple PVC morphologies originating from other areas of the heart are induced. The target of the ablation is usually the morphology suggested by the outpatient recording.

For arrhythmias identified by event recorders only, where many times we don’t have the initiation or termination of the tachycardia, the cycle length of the inducible tachycardia should be close to the outpatient one, although the specificity of this finding might be low due to different physiologic states between outpatient and during the electrophysiological study, where the patient might be sedated and undergoing drug infusions [2–4]. In the situations when we don’t have a recorded clinical arrhythmia, then inducing an arrhythmia that reproduces the patient symptoms might be useful, although it has a lower specificity. Most of the time, in this situation, ablation of the arrhythmia induced during the EP study might be useful [5]. Special mentioning needs to be made of the patients who have an implantable device, where intracardiac electrograms are very useful in determining the mechanism of arrhythmia.

1.2 Catheter Ablation of Accessory Pathways During ORT and AVNRT

There are different strategies to map and ablate an AP [6]. If there is evidence that the AP is involved in the tachycardia, then ablation in sinus rhythm can be performed either by antegrade mapping or retrograde mapping during ventricular
pacing. The end goal of the ablation is elimination of preexcitation. Alternatively, AP mapping can be performed during sustained SVT. If the arrhythmia is proven to be AVRT (atrioventricular reentrant tachycardia) and if the mechanism of the arrhythmia is ORT (orthodromic reentrant tachycardia), then the earliest retrograde atrial activation during sustained SVT is targeted. If the AVRT is antidromic, the earliest ventricular activation is mapped to determine the ventricular insertion of the AP. Ablation of the AP in sinus rhythm without being able to induce SVT carries the risk that the AP might be a bystander, and the only way to prove or disprove this is during sustained tachycardia. For this reason, it is mandatory to induce the arrhythmia for a correct diagnosis, especially if there is no documentation of the clinical arrhythmia on 12-lead EKG or cardiac monitor. Every effort should be made to induce arrhythmia to prove that AP is involved in the arrhythmia mechanism.

If the clinical suspicion of AVNRT (atrioventricular node reentrant tachycardia) is high despite non-inducibility of arrhythmia, slow pathway modification could be performed, but the outcomes are less favorable than when SVT is inducible in the EP lab. In the absence of inducible arrhythmia, some operators might hesitate to perform empirical ablation of the slow pathway, as there is no clear procedural end point and due to the risk of AV block [7, 8]. For patients with inducible AVNRT, the overall procedural success approaches 96% both for cryotherapy and for radiofrequency catheter ablation [9]. On the contrary, Shurrab et al. [10] showed that the success rate for patients with non-inducible AVNRT was 83.7% at 17 months, and this might be due to inability to determine if the mechanism of the clinical short RP tachycardia, suggesting AVNRT, is not actually AT, with an AV delay close to the tachycardia cycle length. Therefore every attempt should be made to induce AVNRT in the EP lab using isoprenaline, adrenaline, atropine, adenosine, etc. to create a perfect balance between the slow and fast pathway conductions to facilitate arrhythmia induction to be able to differentiate between the two arrhythmias.

1.3 Activation Mapping in Patients with Premature Ventricular Contractions

The success of a PVC ablation is dependent on the frequency of the PVC during the procedure. There are different ways to correctly identify the source of the PVC. One of the most commonly used method in the EP laboratory is activation mapping, when using a three-dimensional mapping system, the site of earliest activation during PVC is mapped and targeted. Unfortunately, many of the PVCs are adrenergic dependent, and with minimal sedation required for the procedure, the frequency of PVCs decreases to the point where activation mapping is almost impossible. In this situation, a few monomorphic PVCs are enough to create a template, and then pacemaps are correlated with the template to determine the origin of the PVC. The drawback of this method is that similar pacemaps are correlating reasonably well over wide areas of the ventricle, making it a less preferred method of mapping. For a PVC ablation, inducibility and activation mapping (either spontaneous or during the drug infusion) are essential for a good long-term outcome [11, 12].
1.4 Activation Mapping in Patients with Ventricular Tachycardia

Similar with PVC mapping, VT mapping can be performed using pacemaps, with similar drawbacks. This method is preferred only when VT is hemodynamically unstable. Stevenson et al. published the algorithm of determining the critical isthmus regions in hemodynamically stable ischemic VT during sustained arrhythmia [13]. During reentrant VT, using entrainment mapping, the authors showed that the VT exit sites can be localized precisely, and RF application in the area successfully terminates the VT. For a successful ablation of a focal VT, inducibility of the arrhythmia is essential for a good long-term outcome. Most of the time, we have to use multiple-drug infusions, in escalating doses, to induce clinical arrhythmia.

1.5 Reproducibility of Symptoms During Electrophysiological Testing

When the outpatient arrhythmia documentation is not available, the only way to determine if the arrhythmia we induced in the EP lab is the clinical one is to determine if the symptoms the patient is experiencing during induced arrhythmia are correlating with the patient’s symptoms as outpatient. This method though has a very low sensitivity and specificity.

References

1 Introduction: Why Do We Need Arrhythmia Induction?


How to Induce Arrhythmias by Atrial and Ventricular Programmed Stimulation?

Celestino Sardu, Valerio Giordano, Antongiulio Donatiello, Raffaele Marfella, Giuseppe Paolisso, Maria Rosaria Rizzo, and Michelangelo Barbieri
2.1 Introduction

In recent decades, invasive electrophysiological study (ES) has become an important instrument to evaluate patients with conduction disturbance and cardiac arrhythmias [1]. Using catheters placed in heart chambers via central vein and/or central arterial access, ES may evaluate sinus node function, atioventricular conduction, and tachyarrhythmias. Cardiac arrhythmias may not always be present in the baseline condition. Therefore, it is necessary to induce these arrhythmias by programmed pacing protocols [1]. Commonly arrhythmias are seen as chaotic alterations of the normal heart conduction and of the normal heart rhythm, and then they are defined as cardiac rhythm disorders [2]. Cardiac arrhythmias present with a common phenotype, characterized by irregularity of the cardiac rhythm and related clinical symptoms [2]. In this setting, ES may be performed by using different diagnostic and pacing catheters and pacing protocols. Programmed pacing protocols involve incremental pacing, coupled with the introduction of single or multiple premature stimuli during one or more drive cycles [3]. The pacing protocols are performed with a current output of twice the diastolic threshold or more, and at one or more sites [3]. Therefore, a great discrepancy may exist between arrhythmia induction techniques in different laboratories. However, in the majority of cases, arrhythmias are due to specific arrhythmic electrical, anatomical, and/or electroanatomical circuits [2]. These circuits respond to specific conduction properties of the systolic and diastolic electrical phases, which are reproducible and evocable by external triggers and by specific pacing techniques [2]. Moreover, in the light of these observations, we have to stress the concept that induction and stimulation programs have to be selected and then paced to test the arrhythmic circuits for refractoriness and to trigger the conduction properties of the arrhythmic pathways. Therefore, we have to make arrhythmia induction protocols more uniform and as standardized as possible to avoid all possible bias. Indeed, how to induce arrhythmias by atrial and ventricular stimulation remains a relevant question that needs a specific and unique response. To respond to this question, we would like to introduce the concept that a pacing protocol to induce cardiac arrhythmias may be standard and programmed [2]. As first, by programmed pacing, physicians may study the properties of the cardiac conduction system. This may be secondarily achieved by introduction of early stimuli to determine the conduction response [2], as a specific arrhythmia induction protocol. As discussed earlier, the type of induction and the chosen programmed stimulation protocol may be selected with regard to the type of arrhythmia the patient is suspected to have. In fact, re-entry tachycardias may usually be triggered using extrastimuli to stimulate the conduction pathways in slow conduction and fast conduction ways [2]. Differently, automatic tachycardias not due to re-entry mechanisms may be more easily induced by burst pacing [2]. In this chapter we would like to introduce pacing protocols to induce cardiac arrhythmias. Apart from the similarity of the diagnostic and pacing catheters, and in the setting of programmed stimulation protocols, the differences in the paced heart chambers and in the induced tachycardias teach us to separate the discussion of atrial stimulation from ventricular stimulation...
protocols. Therefore, we schematically divide the arrhythmia induction protocols into two separate chapter sections, discussing atrial stimulation protocols and ventricular stimulation protocols.

### 2.2 Atrial Stimulation

To perform atrial stimulation, the authors place diagnostic quadripolar and/or decapolar catheters in the atrial chambers. These catheters are introduced by central vein access, to map and to pace the right atrium appendage, the coronary sinus, and along the tricuspid valve annulus as indicated by a radioscopic biplane view of the heart chambers. Sometimes, pacing maneuvers may be performed by direct access to the left atrium, at the authors’ discretion. To induce atrial arrhythmias the authors perform programmed pacing protocols divided into coupled pacing protocols and a burst pacing protocol [2]. In the case of a programmed coupled pacing protocol, the authors set a standard pacing protocol choosing a drive of 8 beats as an S1 interval of 600, 500, and then 400 ms, coupled with a first extrastimulus, called S2, that is conventionally at least 60 ms higher than the documented Wenckebach interval [2] (Fig. 2.1). Normally, the authors start with an S1 of 600 ms, then decreasing to 500 and 400 ms. The choice of the S1 cycle length may also depend on the patient’s heart rate. Therefore, at the authors’ discretion the coupled S2 interval is decreased by 10–20 ms for each new pacing train, in a manual and/or automatic way [2]. During each pacing train it is relevant to observe a resting period of 4 s, and to register and to note every arrhythmic event that occurs. In the case of atrial stimulation of re-entrant arrhythmias we may assist with an increase in the supra-Hisian (AH) interval by at least 50 ms from one train to the next, and this is called an “AH jump” [4]. This phenomenon is due to the pacing of the slow pathway of a re-entrant

![Fig. 2.1](image-url)  
**Fig. 2.1**  Representation of programmed coupled atrial pacing for atrial arrhythmia induction. The drive of 8 beats is indicated as S1, at a cycle length of 600 ms. The coupled extrastimulus (S2) is at an interval of 450 ms. The pulse amplitude is 10 mA; the duration is 2 ms. The upper part of the figure shows the DI, DII, aVF, and V1 surface ECG derivations. The yellow color denotes the right atrium (RA: 1–2 is distal, 3–4 is proximal); the green color denotes the His bundle (HIS: d is distal, p is proximal). In this case the authors prefer to use quadripolar diagnostic catheters. (Image created by C. Sardu, WorkMate Claris polygraph, Abbot)
arrhythmic circuit, during the pass on a slow conduction pathway, indicating dual AV node physiology [4]. During programmed and coupled atrial pacing, we may reach a stimulation interval where the atrial pacing is not conducted through the atrioventricular node to the ventricles. This stimulation interval is called the atrioventricular node effective refractory period (AVNERP) [2] (Fig. 2.2). To induce atrial arrhythmias during programmed pacing, we start to shorten the S2 interval until the pacing signal no longer causes the atrium to contract, reaching the atrial effective refractory period (AERP) [2]. Therefore, reaching the refractory S2 interval, we increase the S2 interval to at least 20 ms above the AVNERP, and we repeat this coupled programmed stimulation introducing the secondary (S3) and then the third (S4) coupled extrastimulus as discussed earlier for S2 [2] and/or the AVNERP (in the case of re-entrant atrial arrhythmia induction). Reaching triple refractoriness of the coupled extrastimuli (S2–S3–S4), we stop the S1 programmed stimulation, switching the S1 interval from 600 to 500 and then 400 ms [2]. Moreover, we repeat the same induction protocol until S4 refractoriness occurs and/or in the case of tachycardia induction [2] (Fig. 2.3). Completing all this programmed pacing protocol from 600 to 400 ms in the S1 interval, and until refractoriness of the triple coupled extrastimulus (S2–S3–S4) occurs, we may start burst pacing to achieve a clinical arrhythmia and especially in the case of clinical atrial tachycardia induction [5] (Fig. 2.4). This second type of programmed pacing modality is different from coupled pacing for atrial arrhythmias due to re-entry circuits, and is related to continuous atrial pacing from 300 ms and down by 10–20 ms until 200 ms [5]. In the case of atrial burst pacing there are a few rules to follow. First, the authors consider

![Fig. 2.2](image.png) Representation of programmed coupled atrial pacing to evaluate the atrioventricular node effective refractory period (AVNERP). The drive of 8 beats is indicated as S1, at a cycle length of 600 ms. The coupled extrastimulus (S2) is at an interval of 400 ms. The pulse amplitude is 10 mA; the duration is 2 ms. As can be seen, we may reach the stimulation interval where the pacing interval does not conduct through the AV node to the ventricles, then called the AVNERP. The upper part of the figure shows the DI, DII, aVF, and V1 surface ECG derivations. The yellow color denotes the right atrium (RA: 1–2 is distal, 3–4 is proximal); the green color denotes the His bundle (HIS: d is distal, p is proximal). In this case the authors prefer to use quadripolar diagnostic catheters. (Image created by C. Sardu, WorkMate Claris polygraph, Abbot)
atrial burst pacing below 200 ms to be contraindicated [5]. Second, it seems intuitive and is commonly agreed to turn off the stimulator immediately in the case of atrial arrhythmia induction [6] (Fig. 2.5). During these pacing protocols we have to choose a standard value for the pacing pulse amplitude in milliamperes, and a duration in milliseconds. Normally these values have to be calculated and then chosen as the lowest values for atrial local capture [2]. All these pacing protocols are started in baseline conditions and may be repeated without coexisting contraindications during infusion of drugs interfering with vagal and sympathetic tone, and during patient maneuvers such as a hand grip [2].
2.3 Ventricular Stimulation

To perform ventricular stimulation the authors set a programmed pacing protocol with a catheter placed in the ventricular chambers. The choice of the catheter and the catheter position may differ between authors. Commonly, the authors prefer to perform quadripolar mapping and place a pacing catheter in at least two different right ventricular positions, such as the right ventricular apex and right ventricular outflow tract, as indicated by biplane fluoroscopic imaging of the heart chambers. Uncommonly, ventricular stimulation may be performed by left ventricular access. During ventricular pacing we study retrograde conduction of the atrioventricular node, and/or we may stimulate a concealed accessory pathway, and this may be the first relevant observation during an ES [2]. In fact, ventricular pacing may be used first to study retrograde atioventricular node conduction, and secondarily to induce suspected arrhythmias [2]. Therefore, during ventricular pacing, and in the case of a suspected supraventricular arrhythmia, it is important to map the left atrium and left ventricle conduction, introducing a secondary diagnostic catheter into the coronary sinus (a few authors also use a third catheter placed in the right atrial appendage) to observe the retrograde atioventricular node activation [2] (Fig. 2.6). In fact, during ventricular pacing we may observe concentric and decremental retrograde atioventricular node conduction in the case of retrograde normal atioventricular node activation [2]. Sometimes, during S1 ventricular pacing we may observe retrograde atioventricular dissociation, which confirms retrograde normal atioventricular node activation [2]. On the other hand, we may not see decremental retrograde atioventricular node conduction, which may also be eccentric (not retroactivated as proximal to distal by local coronary sinus electrogram analysis), by concealed retrograde atioventricular accessory pathway pacing [2] (Fig. 2.7). Therefore, during
Fig. 2.6  Representation of programmed and coupled ventricular pacing to study retrograde atrioventricular node conduction. As indicated in the text, the authors perform 8-beat drive pacing as S1 (in this case 500 ms) coupled with gradually increasing pacing as S2 (in this case S2 is 210 ms). It can be seen that S2 is not conducted to the right ventricle, reaching the effective refractory period of the right ventricle. More than this, retrograde normal nodal conduction can be seen as atrioventricular retrograde dissociation during the drive pacing. The upper part of the figure shows the DI, DII, aVF, and V1 surface ECG derivations. The pulse amplitude is 10 mA; the duration is 2 ms. The yellow color denotes the right atrium (RA: 1–2 is distal, 3–4 is proximal); the green color denotes the His bundle (HIS: d is distal, p is proximal), which in this image is placed in the right ventricular apex. In this case the authors prefer to use quadripolar diagnostic catheters. (Image created by C. Sardu, WorkMate Claris polygraph, Abbot)

Fig. 2.7  Representation of programmed and coupled ventricular pacing to study retrograde atrioventricular node conduction. As indicated in the text, the authors perform 8-beat drive pacing as S1 (in this case 600 ms) coupled with gradually increasing pacing until retrograde atrioventricular refractoriness occurs, as S2 (in this case S2 is 350 ms). The figure is stopped at the S1 drive because during S1 pacing, retrograde eccentric nodal conduction occurs via a concealed retrograde bypass tract. The upper part of the figure shows the DI, DII, aVF, and V1 surface ECG derivations. The pulse amplitude is 10 mA; the duration is 2 ms. The yellow color denotes the His bundle (HIS: 1–2 is distal, 3–4 is proximal) placed in the right ventricular apex; the red color denotes the coronary sinus (CS: d is distal, p is proximal), in this case placed in the CS ostium. In this case the authors prefer to use a quadripolar diagnostic catheter for HIS and a decapolar catheter for CS (discussed in the text). (Image created by C. Sardu, WorkMate Claris polygraph, Abbot)
ventricular pacing, we may perform a pacing protocol to study retrograde atrioventricular node activation, and ventricular pacing to induce supraventricular and/or ventricular arrhythmias [2]. As mentioned earlier, we may perform induction pacing for ventricular arrhythmias by a programmed pacing protocol similar to that used for atrial arrhythmia induction. Moreover, we may perform coupled stimulation with eight pacing trains in the drive as S1 with progressive extrastimuli until the refractory periods are found, and/or burst pacing [7] (Figs. 2.8, 2.9, 2.10, and 2.11). During coupled pacing, one may reach the retrograde atrioventricular node effective refractory period (RAVNERP) as an interval in which ventricular pacing is not followed by a retroconducted atrial signal, and then the ventricle effective refractory period (VERP) as an interval in which the ventricular paced extrastimulus is not followed by a local ventricular electrogram [7]. The principal scope of ventricular stimulation is to induce ventricular arrhythmias by programmed pacing protocols. These pacing protocols may be coupled pacing protocols and/or a burst pacing protocol, and the choice of the correct pacing protocol to perform may represent the most relevant question in the setting of life-threatening ventricular arrhythmia induction [8]. As part of this, sometimes ventricular pacing may induce several types of supraventricular tachycardia such as atrioventricular node re-entry, atrioventricular re-entry, and even atrial fibrillation. Generally, to induce ventricular tachycardias the authors perform ventricular pacing by programmed coupled ventricular pacing, by a drive of 8 beats at an S1 interval of 600, 500, and then 400 ms (sometimes the authors prefer only two different drives), decreasing the extrastimuli by 10–20 ms for each new pacing train, in a manual and/or automatic way [7]. The extrastimuli are coupled with the pacing interval until ventricular conduction

Fig. 2.8 Representation of programmed and coupled ventricular pacing to induce ventricular arrhythmias. As indicated in the text, the authors perform 8-beat drive pacing as S1 (in this case 500 ms) coupled with gradually increasing pacing by triple coupled beats as S2, S3, and S4 until refractoriness occurs (in this case S2, S3, and S4 are 200 ms). The upper part of the figure shows the DI, DII, aVF, and V1 surface ECG derivations. The pulse amplitude is 5 mA; the duration is 2 ms (the refractory ventricular conduction is at 2 mA with a duration of 2 ms). The yellow color denotes the right atrium (RA: 1–2 is distal, 3–4 is proximal); the green color denotes the His bundle (HIS: d is distal, p is proximal), which in this image is in the right ventricular apex. In this case the authors prefer to use quadripolar diagnostic catheters. (Image created by C. Sardu, WorkMate Claris polygraph, Abbot)
refractoriness is reached, as a conduction interval in which there is no ventricular capture with pacing [7]. Therefore, the coupled S2 interval is increased by 10–20 ms and repeated as coupled programmed stimulation introducing the secondary (S3) and then the third (S4) coupled extrastimulus in the same modality as discussed earlier in the text (Figs. 2.8 and 2.9). Similarly to atrial arrhythmia induction, during each pacing train we observe a resting period of 4 s, and we register and note every
arrhythmic event that occurs. Therefore, we stop the S1 programmed stimulation, switching the S1 interval from 600 to 500 and/or 400 ms (F9), and repeat the same process until S4 (the third coupled extrastimulus) no longer conducts or tachycardia is triggered [2]. Completing all this programmed pacing protocol from a 600- to 400-ms S1 interval, and until refractoriness of the triple coupled extrastimulus (S2–S3–S4) occurs, we may start burst pacing to achieve a clinical ventricular arrhythmia [9] (Figs. 2.10 and 2.11). This second type of programmed pacing modality is different from coupled pacing for ventricular arrhythmias. The burst pacing is due to continuous ventricular pacing from 300 ms and down to 10–20 ms until 200 ms [9] (Figs. 2.10 and 2.11). When pacing protocols are completed through S4 by at least two S1 cycle lengths (600 and 500 and/or 400 ms), with trough burst pacing until 200 ms, we may choose another pacing site such as the right ventricular outflow tract [10]. Indeed, the programmed pacing protocol to induce ventricular arrhythmias is started over again at the second site of pacing [10]. Few authors advocate introduction of the fourth (S5) extrastimulus during ventricular tachycardia induction [7, 11]. This is a relevant discussion point, and authors express different opinions about the fourth extrastimulus to induce ventricular tachycardias [7, 11]. In fact, we have to report a great discrepancy of results regarding the specificity and sensitivity of the fourth coupled pacing protocol, to induce a ventricular arrhythmia [7, 11]. Moreover, we may speculate that it may be enough to perform a coupled pacing maneuver from the right ventricle with two different drives (500, 500, and/or 400 ms), until refractoriness of the third coupled extrastimulus (S2, S3, and S4) occurs, and at two different sites to induce ventricular tachycardias [11]. As referred to by the authors, once the process of pacing at two different ventricular sites has been completed, Isuprel (isoproterenol/isoprenaline) may be used to enhance cardiac conduction, repeating all the programmed pacing induction.