The ECG in the evaluation of pacemaker function and diagnosis of malfunction

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The ECG in the Evaluation of Pacemaker Function

and

Diagnosis of Malfunction

Atrial Noncapture

Atrial Capture

Retrograde P wave

Berry van Gelder

The ECG in the Evaluation of Pacemaker Function and Diagnosis of Malfunction

The ECG in the Evaluation of Pacemaker Function and Diagnosis of Malfunction

PROEFSCHRIFT

ter verkrijging van de graad van doctor aan de
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op gezag van de Rector Magnificus, prof.dr. J.H. van Lint,
voor een commissie aangewezen door het
College van Dekanen in het openbaar te verdedigen op
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door

LAMBERTUS MATHIJS VAN GELDER

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prof.dr.ir. J.E.W. Beneken
en
prof.dr. H.J.J. Wellens

copromotor: dr.ir. J.A. Blom

Voor Anneke, Monique, Anouk
en mijn Vader

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The ECG in the Evaluation of Pacemaker Function and Diagnosis of Malfunction

Introduction

History of implantable cardiac pacemakers

The first implantation of an asynchronous cardiac pacemaker (Fig.0.1) on October 8, 1958 by Senning and Elmqvist [1] was the start of the development of pulse generators and leads for permanent cardiac pacing. The first step forward was the replacement of the rechargeable nickel-cadmium batteries by the mercury-zinc cells designed by Greatbach and Chardack [2]. These batteries were not rechargeable but extended the pacemaker life to over one year. It was also Chardack who introduced a warning system of battery exhaustion incorporated in the pulse generator, which gradually decreased the stimulation rate when the battery power decreased. Pacemakers at that time were unable to sense intrinsic cardiac activity and stimulated at a fixed rate (asynchronous pacing).
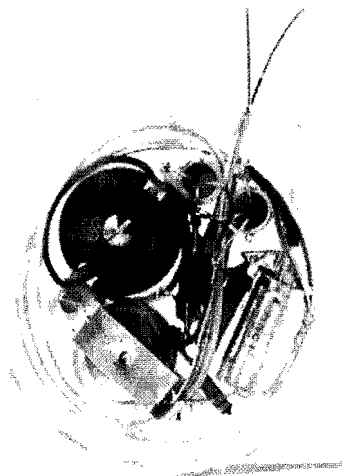
Fig. 0.1. Photograph of the first implantable cardiac pacemaker with rechargeable nickel cadmium batteries (Courtesy of Siemens-Elema AB, Sweden).

In 1962, four years after the implantation of the first asynchronous pacemaker a completely implantable atrial-triggered ventricular pacemaker was described by Keller et al.[3], and used clinically in July of that year [4]. In this pacemaker the atrial activity is sensed by an atrial electrode and after a suitable delay a stimulation pulse is emitted from the ventricular electrode. Although this stimulation form had the advantage of maintaining atrio-ventricular synchronism and adequate rate response in the presence of normal sinus node function, its widespread use was limited by the disadvantages of the system, which necessitated a thoracotomy for implantation of the atrial electrode and had a short life time due to the relatively high stimulation rate, and the detection and amplification of the atrial signal.

Application of asynchronous cardiac pacing revealed the problem of interference between intrinsic rhythm and pacemaker stimuli falling in the ventricular vulnerable phase, with the potential risk of evoking ventricular arrhythmias. To overcome this problem Lemberg, Castellanos and Berkovitz [5] described the first implantable on-demand pacemaker in 1965. The

principle of this pacing mode had already been described and applied in an external unit by Leatham, Cook and Davies in 1956 [6].

In the on-demand pacemaker the ventricle is stimulated at a preset interval in the absence of intrinsic ventricular activity. When intrinsic ventricular activity is sensed the ventricular stimulus is inhibited and the timer determining the pacemaker rate is reset.

In 1966 an alternative form of non-competitive ventricular pacing was introduced by Donato and Denoth [7]; they reported on the R-wave triggered pacemaker. In the R-wave triggered pacemaker the ventricle is stimulated at a preset interval in the absence of intrinsic ventricular activity. When intrinsic activity is sensed, a ventricular stimulus is emitted synchronously with the sensed ventricular activity. The ventricular stimulus delivered in the R-wave does not contribute to either the ventricular depolarization or the ventricular contraction. The main disadvantage of this pacing mode is the higher energy consumption in the presence of intrinsic ventricular activity.

Fig. 0.2. Photograph of one of the first implantable AV sequential demand pacemakers (DVI mode).

The lack of atrio-ventricular synchronism was a drawback of ventricular pacing, sometimes resulting in disabling symptoms in patients treated with ventricular pacing devices. Symptoms would even be aggravated in the presence of retrograde ventriculo-atrial conduction during ventricular stimulation [8]. To overcome this drawback, a pacing system stimulating atrium and ventricle sequentially was introduced by Berkovits in 1969 [9].

The bifocal or AV sequential pacemaker (Fig. 0.2) paces in the atrium and the ventricle sequentially. Sensing occurs only in the ventricle and the mode of response to a ventricular sensed event is inhibited. This form of stimulation permitted competitive pacing in the atrium, with the risk of inducing atrial arrhythmias.

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The lack of reliable atrial electrodes for transvenous implantation, combined with the dimensions and energy consumption of the pulse generator, limited the widespread use of this pacing mode at that time.

Along with the development of pulse generators, lead design had changed significantly. In the late 60s and early 70s efforts were made to improve stability and long term use for transvenous application. The early leads had high electrode surface areas varying between 50 and 80 mm2. Reducing the electrode surface area resulted in a higher current density and thus a lower stimulation threshold [10]. Reduction of the stimulation threshold was important in order to prolong the longevity of the pulse generator. However, effective prolongation of the longevity was only possible with output adjustable pulse generators.
The first non-invasive output programmable pacemaker was introduced by Medtronic. In this pulse generator adjustment of the pulse width was made non-invasively with an external controller that was magnetically coupled with a potentiometer inside the implanted pulse generator [11]. By manual rotation of the controller, a magnet inside the device was rotated, which rotations were followed by the potentiometer inside the pulse generator, thus adjusting the pulse width. In 1972, the Cordis Corporation introduced their Omnicor System. With this system non-invasively electromagnetic programming of rate and output currentwas possible. In the late 70s, most of the pacemaker manufacturers introduced pulse generators that were multiprogrammable for rate, pulse width, sensitivity and refractory period.

About the same time of the introduction of programmable pulse generators, lithium was used in the power cells as successor to the mercury-zinc cells. These power cells offered a high energy density and could be manufactured in every required shape. This allowed further reduction in size without shortening of the pulse generator longevity.

Technical refinement of the pulse generator in the mid 70s was, however, limited to single chamber systems due to the lack of reliable transvenous atrial leads. Development of reliable atrial J leads [12] and atrial leads employing active fixation [13] opened the way for permanent atrial pacing in sino-atrial disease with normal atrioventricular conduction and application of dual chamber pacing systems. The use of reliable transvenous atrial electrodes resulted in a revival of atrial synchronous ventricular and AV sequential pacemakers, which then, also by the application of lithium power cells could be manufactured in acceptable sizes.

The drawback of the atrial synchronous ventricular pacing was the inability to sense intrinsic ventricular activity, whereas the AV sequential pacemaker had a similar disadvantage for intrinsic atrial activity. To overcome both problems and using the advantages of both systems, Funke [14] combined the concept of atrial synchronous ventricular pacing and AV sequential pacing in one device. This pulse generator stimulated atrium and ventricle sequentially in the absence of intrinsic cardiac activity. Sensing of intrinsic atrial activity resulted in inhibition of the atrial stimulus and stimulation of the ventricle after a preset interval, if no intrinsic ventricular activity occurred during this interval. Sensing of intrinsic ventricular activity resulted in inhibition of the ventricular stimulus and resetting of the lower rate interval. The pulse generator was not programmable, all settings were fixed and even a reed switch to convert the system to asynchronous pacing was not incorporated. The introduction of this concept in the past 18 years

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evolved into a completely programmable pulse generator with bidirectional telemetry, Holter functions and algorithms to handle pacemaker mediated tachycardia and undesirable tracking of paroxysmal supraventricular arrhythmias.

Atrial synchronous ventricular pacing, resulting in adequate rate adaption in the presence of normal sinus node function, has limitations in patients with chronotropic incompetence, in whom the acceleration of the sinus node activity is below normal limits. It is not applicable in patients with chronic atrial fibrillation. To provide rate adaptation in patients in whom the atrial rate cannot be used for ventricular synchronization, sensors capable of sensing physiological parameters are incorporated in the lead or pulse generator.
Physiological parameters which are used for rate adaptation are PH, temperature, stimulus-T interval, venous oxygen saturation, respiration, stroke volume, ejection fraction, right ventricular pressure and body activity. Sensor driven rate adaption initially was intended for single chamber systems but later it was also integrated in dual chamber pacing systems.

In the evolving field of cardiac pacing, it became obvious that there was a need for an unequivocal identification of the pacing system, to avoid using different names for the same pacing system, e.g., fixed rate or asynchronous pacemaker, stand-by or on demand pacemaker. Therefore in 1974, the Pacemaker Study Group of the Inter-Society Commission for Heart Disease Resources (ICHD) recommended a pacemaker coding system that was inspired by the need for a simple way of identifying pacemakers. They introduced a three-letter code describing the properties of the pacing system. The first letter indicated the chamber(s) being paced, which could be A for atrium, V for ventricle or D for dual (atrium and ventricle). The second letter indicated the chamber(s) being sensed, which could be A for atrium, V for ventricle, D for dual or O indicating that this pacing device was unable to sense. The third letter indicated the mode of response(s) to the sensed event, which could be T for triggered, I for inhibited, D for dual or O for none. "D" indicated that the device responded to sensed events by triggering a ventricular stimulus (atrial sensed events) and/or inhibition of the atrial output (atrial sense events) and the ventricular output (ventricular sensed events).

Table I. The NASPE/BPEG Generic (NBG) Pacemaker Code

Position	I	II	III	IV	V
Category	Chamber(s) paced	Chamber(s) sensed	Response to sensing	Programmability, rate modulation	Antitachy functions
	O = None	O = None	O = None	O = None	O = None
	A = Atrium	A = Atrium	T = Triggered	P = Simple	
	V = Ventricle	V = Ventricle	I = Inhibited	M = Multi	
	D = Dual (A+V)	D = Dual (A+V)	D = Dual (I+T)	C = Communicating	D = Dual (P+S)
	S = Single (A or V)	S = Single (A or V)		R = Rate modulation	

Note: Positions I through III are used exclusively for antibradycardia function.

This code was rapidly accepted as being generally very helpful. Due to the growing pacemaker capabilities, the code was expanded to five positions to represent degree of programmability and antitachyarrhythmia functions [16]. Finally in 1987 the joint efforts of the North American Society of Pacing and Electrophysiology (NASPE) and the British Pacing and Electrophysiology

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Group (BPEG) resulted in NASPE/BPEG Generic, the NBG code [17], which is summarized in Table I.

For the first three positions there are no significant differences between the original three position code and the NBG code. For the benefit of the manufacturers, an "S" has been added to first and second position. This indicates that the pulse generator has been designed for single chamber pacing and sensing. Thus, an SSI could be used in the atrium as AAI or in the ventricle as VVI.

Evaluation of pacemaker function and diagnosis of malfunction.
Normal pacemaker function is characterized by effective stimulation and proper sensing of intrinsic depolarization of the chamber in which the lead is implanted. Malfunction of the pacing system can only be detected by recording the ECG. Malfunction in single chamber pacing systems is either related to stimulation or sensing of intrinsic depolarization or both. In dual chamber systems, malfunction in one channel may be related to stimulation or sensing, but also the interaction between the two channels can be a source of malfunction.

Most papers describing pacemaker malfunction are limited to case reports. Titles of case reports usually describe the electrocardiographic presentation and the cause of malfunction (e.g., P wave oversensing in a VVI pacemaker). In clinical practice, however, the interpreter is confronted with an ECG showing pacemaker malfunction without knowing the underlying cause. Being faced with an ECG showing malfunction the interpreter has to analyze the problem in a systematic way, to find out the origin of failure.
In chapters describing pacemaker malfunction [18,19] the lay-out is often similar to the case reports. The authors summarize the causes of malfunction, which are illustrated by ECGs, without describing a systematical differentiation approach. The purpose of this thesis is, to describe a systematical guide for the analysis of the ECG showing pacemaker malfunction. If more information is needed to discover the origin of malfunction, additional techniques that are available in the pacemaker clinic (e.g., pacemaker telemetry), will be used primarily. Some problems may need further radiological investigation either to confirm the diagnosis or to localize the defect in case of lead fracture. Occasionally malfunction can be corrected by reprogramming the pulse generator, although surgical intervention may sometimes be required.

Many problems related to cardiac pacing are intermittent. This implies that the standard 12 lead ECG is not the most suitable for recording pacemaker function, and malfunction can easily be overlooked in these recordings. Therefore, we use in a three channel analog ECG recorder capabale of continuous registration of the ECG during pacemaker follow-up in our clinic. The ECG leads I, II and III are used routinely for this purpose. Because of the limited number of leads that can be recorded simultaneously in this setting, the description of the ECGs in this thesis is often restricted to the appearance of pacemaker (mal)function in lead I, II and III. The 12 lead ECG, however, is useful for the determination of the stimulation site. Selected precordial leads (sometimes combined with double amplification) can be used to define intrinsic or stimulated atrial depolarization, when its presence or absence cannot be concluded from the standard leads.

The analysis of the pacemaker ECG for pacing and sensing, in this thesis is limited to ECGs from single and dual chamber systems without sensor driven rate adaptation. The reason for this is twofold:

1. With regard to effectiveness of stimulation and sensing, there is no essential difference between sensor driven pacemakers and non-sensor driven pacemakers. The only difference is the varying escape interval in rate adaptive systems, which can be easily disabled during follow-up, to evaluate pacing and sensing function. The interpreter, however, should be aware of the fact that activation of the rate adapative mode shortens the alert period of the pacemaker. The alert period is the period that the pacemaker is able to sense and is determined by the escape interval minus the refractory period. Because the latter is fixed and the escape interval decreases during rate adaptation the alert period shortens under these conditions.

2. The inappropriate rate of a sensor driven pacemaker is related to the adjustment of the sensor response or to false triggering of the sensor. The first problem should not be considered as pacemaker malfunction and can easily be corrected. The second problem is not related to stimulation or sensing of the system, both of which can be normal, but to false triggering of the sensor, leading to an inappropriate high pacing rate. The mechanisms for these tachycardias are described in chapter VII, 7.2.

Additional techniques, described in chapter I, are used to confirm the electrocardiographical diagnosis or to differentiate between the possible causes of malfunction. Some of these techniques are also used to evaluate the quality of the pacing system with respect to safety margins for pacing and sensing.

The information obtained from the pacemaker ECG is not limited to normal and abnormal pacing and sensing. During stimulation there is a direct relationship between the site of stimulation and the morphology of the resulting depolarization. This implies that a changing morphology of stimulated depolarization may indicate a change in the position of the stimulation electrode, which could be a predictor of electrode displacement. Factors affecting the morphology of stimulated depolarization are discussed in chapter II.

The pacemaker stimulus in the ECG is determined by several factors directly related to the pacing system, such as the distance between the anodal and cathodal electrode and duration and amplitude of the stimulus. Variation in amplitude of the pacemaker stimulus may contain information in the case of pacemaker malfunction. However, the pacemaker stimulus can be distorted by the recording equipment. The effect of recording equipment and the factors determining the pulse amplitude in analog recordings of the ECG are described in chapter III.

To conclude effectiveness of stimulation, the interpreter should be able to recognize the pacemaker stimulus and the resulting depolarization from the ECG. The pacemaker stimulus has to be recognized to discriminate between no output and noncapture. Depolarization has to be recognized to determine capture or noncapture. Recognition of atrial capture, especially in unipolar dual chamber systems, sometimes requires non standard precordial ECG leads at double standard amplification. In single chamber systems atrial capture can be worked out from a 1:1 ventricular response. Stimulation of one cardiac chamber through a lead placed in another cardiac chamber (cross stimulation), can be found in dual chamber systems. ECGs showing some

pitfalls in recognizing effectual stimulation and the causes of ineffectual stimulation are described in chapter IV.

Failure in pacemaker sensing is characterized by a prolongation of the escape interval (oversensing) or a shortening of the interval between an intrinsic cardiac event and the following paced event (undersensing) in single chamber inhibiting systems. The mechanisms and causes of oversensing and undersensing are explained in chapter V.

In dual chamber systems, pacemaker behavior with respect to timing and its electrocardiographic presentation may be complicated. Even in normally functioning systems, the ECG can be misleading and may represent apparent malfunction. Apparent malfunction can be caused by the interaction between the programmable parameters (e.g., upper rate limit, atrial refractory period) and the interaction between the atrial and ventricular channel (cross talk, ventricular safety pacing, initiation of refractory periods in one channel caused by sensed events in the other channel). Therefore a major part of chapter VI is devoted to timing intervals in the normal functioning dual chamber pacing system. The last section of this chapter describes the electrocardiographic presentation of malfunction.

The various types of pacemaker tachycardia are summarized in chapter VII. This chapter describes the underlying mechanisms of tachycardia and the management of prevention or termination of pacemaker tachycardia.

In chapter VIII a practical approach to pacemaker malfunction or apparent malfunction is described by a troubleshooting flow chart. In this approach schematic ECGs are used to illustrate pacemaker malfunction. This pragmatic approach links up the best to the clinical situation. The electrocardiographic problem is relatively easily identified and the strategy to find the underlying cause is described.

The rules used by the author in the analysis of the pacemaker ECG are implemented in a first prototype of an expert system developed at the Eindhoven University of Technology. A description of the expert system and the first results of the application will be discussed chapter IX.

References

1. Elmqvist R, Senning A. Implantable pacemakers for the heart. In Smyth CN, ed: Medical electronics Proc Sec Int Conf on Med Electronics. Paris 1959, London Iliffe and Sons, Lts, 1960.
2. Chardack W, Gage AD, Greatbatch W. A transistorized self-contained, implantable pacemaker for the long-term correction of complete heart block. Surgery 1960; 48: 643-649.
3. Keller JW, Nathan DA, and Center S. An implantable synchronous cardiac pacemaker, Digest 1962, 15th Annual Conf. Engrg in Med.& Biol., November 5-7, 1962.
4. Nathan DA, Center S, Wu CY, and Keller JW, Jr. An implantable, synchronous pacemaker for the long-term correction of complete heart block. Circulation 1963; 27: 682-689.
5. Lemberg L, Castellanos A, Berkovits BV. Pacemaking on-demand in A-V block. JAMA 1965; 191-1: 106-111.
6. Leatham A, Cook P, Davies JG. External electric stimulator for treatment of ventricular standstill. Lancet 1956; 1185-1187.

7. Donato L, Denoth F. *Self-synchronizing cardiac pacemaker for the treatment of intermittent AV-block. Lancet 1966; 2: 233-239.*

8. El Gamal MIH, van Gelder LM. *Chronic ventricular pacing with ventriculo-atrial conduction versus atrial pacing in three patients with symptomatic sinus bradycardia. PACE 1981; 4: 100-105.*

9. Berkovits BV, Castellanos A, and Lemberg L. *Bifocal demand pacemaker. Circulation 1969; 39,40, III-44.*

10. Lindemans FW. *Electrical stimulation of heart muscle. Drukkerij Elinkwijk BV, Utrecht. Thesis, Utrecht 1977.*

11. Rodrigo FA. *How useful is threshold measurement? In C. Meere (ed): Proceedings of the VIth World Symposium on Cardiac Pacing, Montreal, October 2-5, 1979. Montreal, Canada, PACESYMP, 1979, chap. 19-5.*

12. Kleinert M, Bock M, Wilhelmi, F. *Clinical use of a new transvenous atrial lead. Amer. J. Cardiol. 1977; 44: 237-243.*

13. El Gamal M, van Gelder B. *Preliminary experience with the Helifix electrode for transvenous atrial implantation. PACE 1979; 2: 444-454.*

14. Funke H, Herpers L. *Electrocardiographic findings in patients treated with optimized sequential stimulation. First European Symposium on Cardiac Pacing. London, May 7-9, 1978.*

15. Parsonnet V, Furman S, Smyth NPD. *Implantable cardiac pacemakers: Status report and resource guidelines. Pacemaker Study Group, Inter-Society Commission for Heart Disease Resources (ICHD). Circulation 1974; 50: A21.*

16. Parsonnet V, Furman S, Smyth NPD. *A revised code for pacemaker identification. PACE 1981; 4: 400-403.*

17. Bernstein AD, Camm AJ, Fletcher RD, et al. *The NASPE/BPEG generic pacemaker code for antibradyarrhythmia and adaptive-rate pacing and antitachyarrhythmia devices. PACE 1987; 10: 794-799.*

18. Barold SS, Falkoff MD, Ong LS, et al. *Differential diagnosis of pacemaker pauses. In SS Barold (Ed.) Modern Cardiac Pacing. Mount Kisco, New York, Futura Publishing Company, 1985, pp.587-613.*

19. Sutton R, Bourgeois I. *The Foundations of Cardiac Pacing. Chapter 12, Troubleshooting. Mount Kisco, New York, Futura Publishing Company, 1991, pp. 259-301.*

Chapter I. **Additional Techniques in the Evaluation of Pacemaker Function and Malfunction**

Introduction

The ECG is the most important tool for determining pacemaker function and malfunction. However, the information obtained from the ECG has a yes/no function: it indicates effective stimulation and sensing but provides only incidental information about safety margins with regard to stimulation and sensing threshold. Additional techniques are used during follow up in order to check the quality of the pacing system e.g. stimulation and sensing threshold, battery status and lead impedance.

Lead impedance is also an important parameter in case of pacemaker malfunction or impending malfunction. Lead impedance can be measured by pulse waveform analysis or pacemaker telemetry. In clinical practice, a rise in lead impedance may indicate a partial lead fracture and a fall may indicate lead insulation rupture. It might be necessary to use chest X-ray or fluoroscopy in order to determine electrode position, lead integrity or the lead/pulse generator connection. Provocative maneuvers like muscle contraction can be used to evaluate the influence of muscle potentials on the pacing system. Chest wall stimulation can be useful in the evaluation of the patient's underlying rhythm, pulse generator function and detection of insulation defects.

In the pacemaker clinic repetition rate and pulse width of the pulse generator are determined by electronic devices. For dual chamber systems these devices measure both atrial and ventricular pulse and the atrioventricular interval. Changes in pulse generator's repetition rate and/or pulse width indicate impending battery depletion. In general, this change is slowing of the repetition rate, usually detectable in the magnet mode, sometimes combined with prolongation of the pulse duration.

1.1 Pulse waveform analysis

Pulse waveform analysis was first reported by Nickel in 1964 [1] and has been further elucidated by the University Hospital of Leiden and Groningen [2]. Pulse waveform analysis makes use of the pacemaker stimulus as recorded from the Einthoven leads (Leads I,II,III). The pacemaker stimulus is displayed and photographed from an oscilloscope or recorded on an electronic printer. From the voltage decay of the impulse, representing the discharge of the output capacitor over the impedance in the stimulation system, the lead impedance can be calculated. Because of the polarization effect around the stimulation electrode, which stabilizes after about 0.5 to 1.0 ms, the pacemaker impulse needs to be longer than 1.0 ms in order to perform an accurate calculation by this method [3]. Initially lead impedance was calculated manually from the photograph of the pacemaker pulse and this was time consuming. Later on automatic and computerized systems were developed which permitted fast and easily measurement of lead impedance [4,5].

Alomst simultaneously with this development, pacemaker manufacturers incorporated in their devices the measurement of lead impedance by telemetry. There is a discrepancy in the absolute value of lead impedance obtained by pacemaker telemetry and pulse waveform analysis, usually due to the fact that lead impedance measured by telemetry is calculated in the initial portion of the pacemaker pulse, where changes in polarization still affect the measurement. Despite this discrepancy, the trend of impedance changes, indicative for lead performance, can be followed reliably by pacemaker telemetry without the use of additional equipment.

The advantage of pulse waveform analysis over lead impedance measured by pacemaker telemetry is twofold:

1. During pulse waveform analysis the area of the pacemaker pulse used for calculation of lead impedance is well defined and the effect of changing polarization can be excluded for the measurement, thus giving a more accurate result.
2. The waveform of the pacemaker pulse as presented during pulse waveform analysis not only reflect the ohmic component of lead impedance, but also inductive and capacitive changes associated with lead related problems, will be shown.

The interpreter should be aware that lead impedance is measured during emission of the stimulation impulse, which is only a short period of the pacing cycle. Variation in lead impedance in between pacemaker stimuli cannot be measured neither by the lead impedance nor by pulse waveform analysis.

1.2 Pacemaker programmability

The majority of currently implanted pacemakers are programmable. Some programmable parameters are used to adapt the pacing system to the patient's clinical need (e.g. repetition rate, hysteresis), whereas others are used to evaluate the quality of the pacing system and if possible, to correct pacemaker malfunction or impending malfunction.

1.2.1 Output parameters

Stimulation threshold can be measured during follow-up (at a fixed pulse amplitude) by decreasing pulse width until capture is lost. If pulse generator pulse amplitude is also programmable this test can be repeated at different settings of the amplitude. These measurements can be used to monitor stimulation threshold and adjust pulse generator energy output appropriately. Appropriate output setting means an adequate safety margin for stimulation at the lowest output setting in order to extend the longevity of the power source. In the majority of pulse generators stimulation threshold can be measured automatically by the pacemaker programmer, by connecting the patient to an ECG and placing the programmer head over the pacemaker.
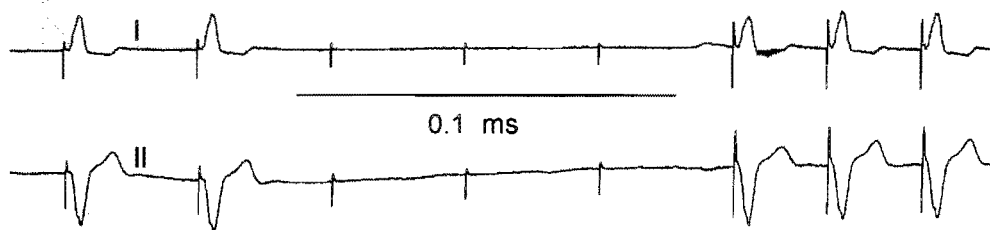
Fig. 1.1. Two channel ECG (Lead I,II) illustrating automatic noninvasive measurement of stimulation threshold during follow-up. The pulse width is automatically decremented by 0.1 ms at a fixed pulse amplitude. Capture is lost when the pulse width is decreased from 0.2 to 0.1 ms. Release of the programmer key restores pacing at the preset pulse width of 0.5 ms

By pressing the appropriate key the pulse duration is decremented every few seconds and the value is displayed on the programmer. When capture is lost, the key is released and the pulse

duration returns to its set value (Fig.1.1). If available in the pulse generator, stimulation threshold can also be measured by decrementing pacemaker pulse amplitude at a fixed pulse width.

Gradual increase in stimulation threshold during follow-up without evidence of major changes in lead impedance is usually due to local effects such as formation of fibrous tissue around the stimulation electrode. As long as effective stimulation can be maintained with a sufficient (100%) safety margin no further action should be undertaken, but careful monitoring will be necessary to prevent development of exit block, especially in the pacemaker dependent patient.

1.2.2 Sensing parameters

Sensing threshold testing can be performed by gradually reducing the sensitivity until sensing is lost. The programmed value for sensitivity should also have a safety margin compared to the sensing threshold, because of variations in the intracardiac signals caused by physiological changes. The safety margin should also be high enough to recognize intrinsic signals from a different origin, with different sensing characteristics. It is evident that the presence of intrinsic cardiac activity is essential to perform the sensing threshold test. Accuracy of the sensing threshold depends on the number of programmable steps in the sensitivity setting.

Increasing or decreasing sensitivity can be attempted to correct the problem of undersensing and oversensing respectively. If undersensing or oversensing is observed, combined with a significant change in lead impedance, the system should be evaluated for lead integrity and lead connection (See chapter V, 5.14.1, 5.14.2). Even if normal pacemaker function is restored by reprogramming sensitivity under these circumstances, one should consider surgical revision of the pacing system. The change in lead impedance indicates an impending problem in the galvanic continuity or insulation, which may degenerate to complete lead fracture.

Undersensing with maintenance of normal lead impedance is usually due to local effects around the stimulation and sensing electrode such as formation of fibrous tissue. This problem can be corrected by programming the pulse generator to a more sensitive sensing level

1.2.3 Refractory period

The refractory period is the time after a paced or sensed event during which the escape interval timer cannot be reset by any sensed event. The refractory period consists of two parts, the absolute refractory and the relative refractory period. In most implantable pacemakers only the total refractory period is progammable, whereas the absolute refractory period is a fixed interval. Prolongation of the refractory period can be used if phenomena directly related to stimulation occur outside the refractory period and are sensed by the pacemaker, thus prolonging the escape interval. These phenomena have a more or less fixed interval to the paced event.

T-wave sensing by a ventricular pacemaker is an example of oversensing that can be corrected by reprogramming the refractory period to a longer interval. R-wave oversensing in an atrial pacemaker can be corrected identically. Avoiding oversensing of these phenomena, however, should first be attempted by reducing sensitivity of the pacemaker. If this fails, prolongation of the refractory period is the next best alternative.

The atrial refractory period in dual chamber systems determines the atrial tracking rate, the fastest atrial rate that can be sensed by the atrial channel. This refractory period together with the upper rate interval determines the upper rate behavior of the pacing system, which will be explained in detail in chapter VI 6.3.

1.3 Pacemaker telemetry

Pacemaker telemetry allows information to be transmitted from the pulse generator via a radio frequency signal to a receiver in the pacemaker programmer head. There is a variety of information that is avaible by pacemaker telemetry. The standard information includes the programmed settings of the pacing system and the pacemaker identification and serial number [6]. In modern cardiac pacemakers event counters are incorporated allowing presentation of rate histograms by telemetry. Undesired high rates, either spontaneous or pacemaker mediated and inappropriate rate response, can be detected by this method [7]. Because the major importance of this option is the adaptation of the pacing systems to the patient's need and it only rarely gives a contribution to the diagnosis of pacemaker malfunction it will not further be discussed.

The most import parameters in the diagnosis of pacemaker function and malfunction obtained by telemetry are:

1. Lead impedance
2. Marker channel ™
3. Intracardiac ECG
4. Battery status

1.3.1 Lead impedance

Although the term lead impedance is generally accepted, the impedance measured by the pacing system is not only the impedance of the lead. The larger component is derived from the impedance formed by the electrode-myocardium interface and the polarization effect around the stimulation electrode [8]. Because the polarization is affected by the output of the pacemaker pulse (amplitude and pulse duration), lead impedance should be measured at the same output setting of the pulse generator. Otherwise variation in lead impedance during follow-up can be induced by the method of measurement instead of variations in the electrical properties of the lead. Lead impedance variations, after implantation, in the order of 10%, with a tendency to a gradual decline after one year are normally observed, and are no indicator of lead malfunction [9].

Polarization is not constant but varies during the stimulation pulse. This implies that measurement of lead impedance depends on the measuring time point after onset of the stimulation pulse. Because the measuring time point or method of measurement can be different for pulse generators from different manufacturers, comparison of lead impedance in individual patients is difficult. During follow-up in an individual patient, in whom pacemaker output is kept constant during measurement of lead impedance, the value of the lead impedance is a reliable indicator for the electrical performance of the lead.

1.3.1.1 Increase in lead impedance

Increase in lead impedance by more than 20% generally indicates a problem or impending problem in the conductor or the connection between lead and pulse generator. It will be associated with an increase in stimulation threshold (provided that the surface area of the stimulation electrode is unchanged), because the stimulation current is directly proportional to the applied output voltage.

An increase in lead impedance in a unipolar pacing system indicates a problem or impending problem in the cathodal conductor, which is the only conductor in this system, or in the connection between pulse generator and lead. Radiological investigation preferably by

fluoroscopy employing digital cardiac imaging (DCI) systems should be performed in order to determine suspected areas in the conductor or connector. Extensive kinking and fixation points of the lead are often locations for partial or total conductor breakage. If extensive kinking or visible damage to the conductor is observed together with an increase in lead impedance and a rise in stimulation threshold, prophilactic replacement or repair of the lead should be carried out in pacemaker dependent patients.

If in bipolar systems an increase in lead impedance is observed, the pacing system should be programmed in the unipolar configuration and measurement of lead impedance repeated. A normal lead impedance in the unipolar configuration indicates a (impending) problem in the anodal conductor. Under these conditions radiological investigation is also advisable. Extensive kinking of the lead apparently leading to damage of the anodal coil of the conductor might progress and also affect the cathodal conductor. Programming the system to the unipolar configuration and careful monitoring of the lead impedance in this configuration probably is the best approach.

If lead impedance is still high after programming the system to the unipolar configuration it can be concluded that the (impending) problem is located in the cathodal conductor or in both conductors. The approach to this problem is similar to that of unipolar leads.

1.3.1.2 Decrease in lead impedance

Decrease in lead impedance generally indicates a defect in the insulation of the lead or an insulation leakage in the connector of the pulse generator. The effect of an insulation defect on lead impedance in unipolar leads depends upon the size of the defect and, in bipolar leads in the location and the size of the defect. In bipolar leads the effect on lead impedance depends on the location, as there is a difference between defects in the outer insulation of the lead and an insulation defect between anodal and cathodal conductor. This will be illustrated later.

insulation defect unipolar lead
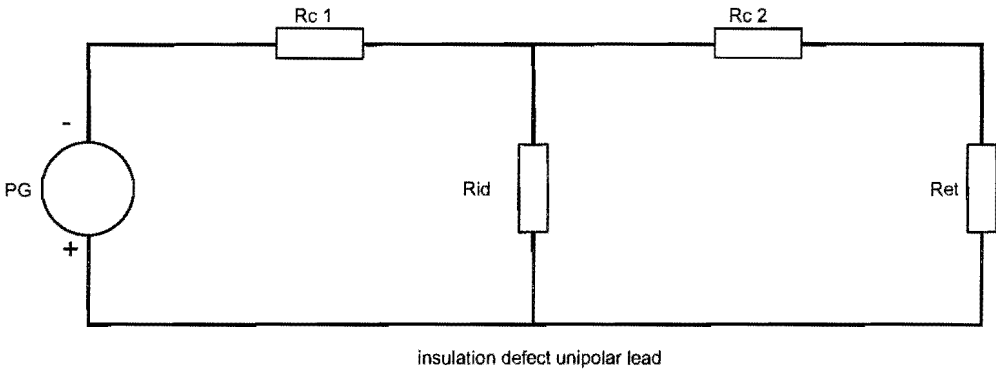
Fig. 1.2. Simplified diagram of an insulation defect in a unipolar lead. The resistance of the cathodal conductor is represented by Rc1 + Rc2, Ret is representing the electrode-tissue interface and Rid is the additional impedance formed by the insulation defect. The relationship between Rc1 and Rc2 is determined by the location of the insulation defect. PG = pulse generator. Normal value for Rc1 + Rc2 = 60-120 Ohms; Ret = 400-600 Ohms.

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The effect of an insulation defect in a unipolar lead is demonstrated in figure 1.2. Due to the insulation defect there is an impedance parallel to the impedance formed by the electrode-tissue interface. The resistance of the conductor is relatively low, between 60 - 120 Ohms. An insulation defect, creating an impedance of the same order as the electrode-tissue impedance, will reduce the lead impedance to approximately 50% of its original value.

From this diagram it is clear that the stimulation current is not affected as long as the total current delivered by the pulse generator does not exceed the maximum capacity. In practice, the stimulation threshold will only be affected if the total current drain from the pulse generator exceeds its maximum capacity. The maximum current delivered by a pulse generator depends on the capacity of the output capacitor, which is manufacturer and type related.

The effect of an insulation defect in the outer insulation of a coaxial bipolar lead is demonstrated in figure 1.3. A defect in the outer insulation of a bipolar lead creates a resistance parallel to the resistance formed by a part of the anodal conductor. Because the resistance of the conductor is low (between 50 and 100 Ohms) there will be only a small effect on the total lead impedance. This situation is also comparable with an increase of the surface area of the anodal electrode, thus the stimulation threshold will not be affected by this type of insulation defect. Electrocardiographically it will lead to "unipolarization" of the pacemaker stimulus as illustrated in chapter III, figure 3.11 and chapter IV, figure 4.64.

outer insulation defect bipolar lead
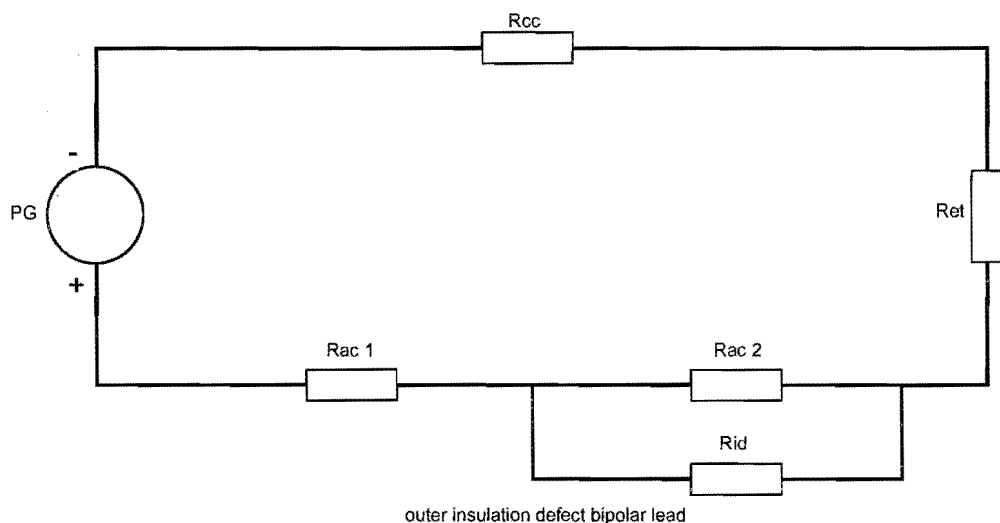
Fig. 1.3. Simplified diagram of a defect in the outer insulation of a coaxial bipolar lead. Rcc is the resistance in the cathodal conductor, Rac1 + Rac2 represent the resistance of the anodal conductor, Ret is the electrode-tissue impedance and Rid is the additional impedance formed by the insulation defect. The relationship between Rac1 and Rac2 is determined by the location of the insulation defect. PG = pulse generator. Normal values for Rac1 + Rac2 = Rcc = 60-120 Ohms; Ret = 400-600 Ohms.

The effect of an insulation defect in the inner insulation of a coaxial bipolar lead, between anodal and cathodal conductor is illustrated in figure 1.4. Electrically it is more or less comparable with

an insulation defect in a unipolar lead, because of the low resistance of the conductors. In practice however, the implications of an insulation defect might be different. First of all an insulation defect in a unipolar lead will easily be penetrated by body fluids, thus causing leakage current through the defect. Secondly the possibility of a complete short circuit between anodal and cathodal conductor is higher in coaxial bipolar leads because of the short distance between both conductors. In unipolar systems it is necessary that the insulation defect enables contact between the cathodal conductor and the pacemaker can in order to form a short circuit.

inner insulation defect bipolar lead
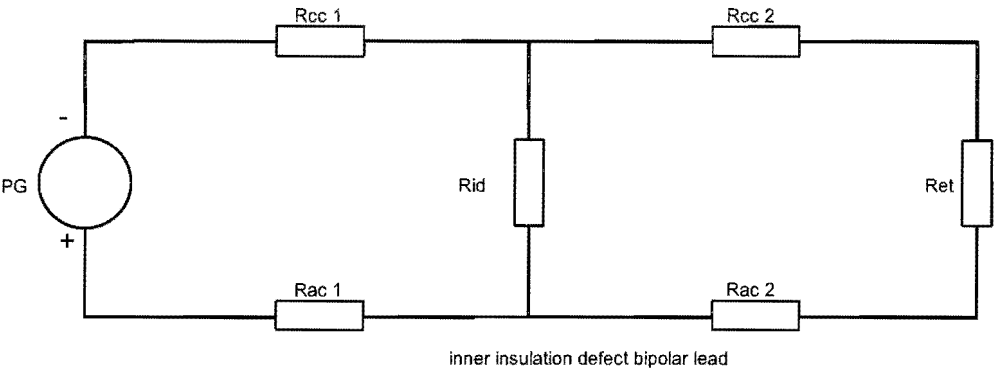
Fig. 1.4. Simplified diagram of a defect in the inner insulation of a coaxial bipolar lead. Rcc1 + Rcc2 and Rac1 + Rac2 represent the resistance of the cathodal and anodal conductor respectively, Ret is the electrode-tissue impedance and Rid is the additional impedance formed by the insulation defect. The relationship between Rcc1 and Rcc2, and Rac1 and Rac2 is determined by the location of the insulation defect. PG = pulse generator. Normal values for Rcc1 + Rcc2 = Rac1 + Rac2 = 60-120 Ohms; Ret = 400-600 Ohms.

In a bipolar lead with an insulation defect between the two conductors, without penetration of body fluid, it is also possible to find a normal lead impedance. During emission of the stimulation pulse both conductors may be separated, resulting in a normal lead impedance. The mechanical action of the heart however, may provoke intermittent contact between both conductors with resulting potentials, which can be sensed by the pacemaker. Therefore oversensing in a bipolar pacing system may indicate an insulation defect, even in the presence of a seemingly normal lead impedance.

1.3.2 The Marker Channel [TM]

Although the term Marker Channel [TM] is a registered trade mark of Medtronic Inc. and should correctly be replaced by the phrase ECG interpretation channel [10], to achieve better understanding and general acceptance, the phrase marker channel will be used in this text.

The marker channel is obtained by placing a programmer head over the pulse generator, which receives telemetered data, while surface ECG leads connected to the programmer supply the ECG. The programmer printer then formats and displays the data. The marker channel provides precise indications of pacemaker sense and pace events. Annotated series of pulses are recorded simultaneously with the ECG. The pulses indicate pace or sense events and in case of sense

events, it also shows whether these sense events occur in or outside the relative refractory period [11].

Indication of pace events in the marker channel is only useful in case of no output (due to a transmission defect) or in bipolar systems programmed at low stimulation output, which sometimes result in greater difficulty in visualizing the pacemaker artifacts on the ECG. Generally pace events are easily recognized from the ECG.

The marker channel is extremely useful in the analysis of sensing problems and the interpretation of complex ECGs from dual chamber systems [12]. In dual chamber systems sense events in one channel affect the behavior of the other channel. If these sense events are caused by oversensing of signals not visible on the ECG, the ECG can be misleading in the absence of the marker channel. This is illustrated in figure 1.5. The first impression from the ECG registration is the presence of atrial undersensing; a P wave is not followed by a ventricular stimulus. Undersensing of the P wave, however, is caused by oversensing in the ventricular channel, shown by the marker channel as well as the telemetered intracardiac electrogram. The erroneous ventricular sense event initiates a postventricular atrial refractory period (PVARP), resulting in the occurrence of the intrinsic P wave during this PVARP. The latter is also indicated in the marker channel that shows detection of the P wave during the relative portion of PVARP. Because the intrinsic P wave occurs during PVARP no ventricular stimulus is evoked and the ECG is suggestive of atrial undersensing. Ventricular oversensing in this patient was caused by an intermittent contact between the cathodal and anodal conductor in the bipolar ventricular lead. Lead impedance measured in the unipolar as well as the bipolar configuration showed normal values; 524 and 550 Ohms respectively.
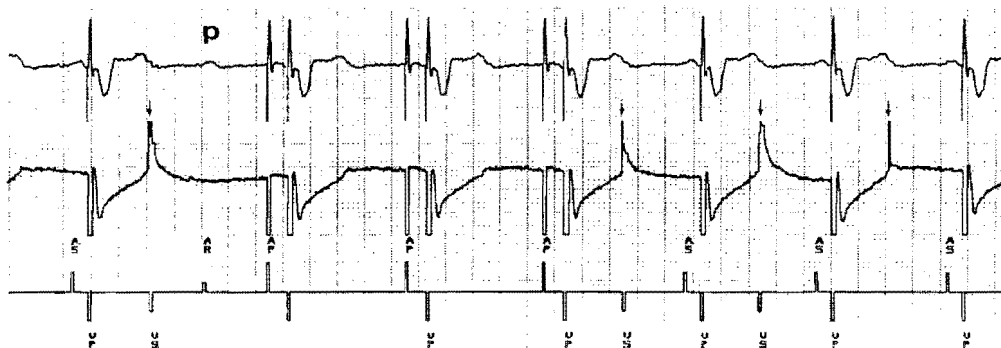
Fig. 1.5. Three channel recording of the ECG lead II (upper tracing), intracardiac ventricular electrogram (middle tracing) and marker channel (lower tracing) from a patient with an implanted bipolar DDD pacemaker. The ECG shows an intrinsic P wave (P) not followed by ventricular stimulation, suggestive of atrial undersensing. The marker channel, however, indicates that this P wave occurs during the atrial refractory period (AR). This atrial refractory period is initiated by oversensing in the ventricular channel (VS). The intracardiac electrogram shows signals (arrows) caused by an intermittent contact in the ventricular lead, responsible for oversensing. AS = atrial sensing, VP = ventricular pacing, VS = ventricular sensing, AR = atrial sense event during the atrial refractory period, AP = atrial pacing.

In single chamber pacemakers the marker channel is useful in the ECG interpretation, especially in those case where sense events occur in the relative portion of the refractory period. Because sense events that only occur in the relative refractory period do not affect the escape interval, apparently normal pacemaker function is be observed. However sense events in the refractory period, indicated by the marker channel, may be an indication for lead malfuction even in the presence of a normal lead impedance and an ECG showing normal pacemaker function as illustrated in figure 1.6.
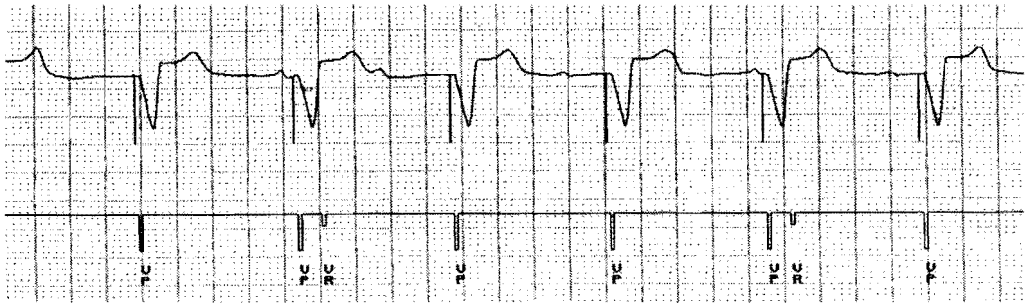
Fig. 1.6. Two channel recording of the ECG lead II (upper tracing) and the marker channel (lower tracing) from a patient with a bipolar VVI pacemaker. The ECG shows normal pacing at the programmed rate, suggesting normal pacemaker function. The marker channel shows, however, ventricular sense events during the ventricular relative refractory period (VR). Because these sense events occur during the relative refractory period, the escape interval is not affected. These sense events are indicative of lead malfunction even in the presence an ECG showing normal pacing. An intermittent contact between anodal and cathodal conductor of the bipolar lead was confirmed after further evaluation.

1.3.3 The intracardiac electrogram
The number of pulse generators that can record and transmit the intracardiac electrogram from the chamber in which the lead is implanted is increasing. In the majority of pulse generators equipped with this feature, the intracardiac electrogram is combined with a marker channel or annotation, indicating pace- sense events and the programmed refractory period(s).
The information obtained from the telemetered intracardiac electrogram is twofold:
1. The amplitude and morphology of the intrinsic intracardiac electrogram.
2. The nature of the signals responsible for pacemaker inhibition and/or pacemaker triggering.

1.3.3.1 Evaluation of the intrinsic intracardiac electrogram
From the telemetered intracardiac electrogram the morphology and amplitude of the intracardiac electrogram can be evaluated. The morphology of the intracardiac electrogram is determined by the position of the electrode and the performance of the lead. This implies that changes in electrode position are reflected by changes in the morphology of the electrogram. This feature can be useful in the diagnosis of lead perforation occurring shortly after pacemaker implantation as demonstrated in figure 1.7. However, dislocation or perforation of a lead is recognized earlier by an increasing pacing and/or sensing threshold. The intracardiac electrogram can be an additional indication for the final diagnosis.

The evaluation of morphology and amplitude of the intracardiac electrogram during pacemaker follow-up can be useful for the detection of lead failure. In particular insulation failure, also indicated by a decrease in lead impedance, affect the morphology of the intracardiac electrogram measured by the pulse generator.

Van Beek et al. [13] demonstrated that diminishing electrogram amplitude at follow-up predicted insulation failure several months before becoming clinically manifest. The figures illustrating van Beek's report, however, showed not only a diminishing amplitude but also a change in morphology of the electrogram. See chapter V, 5.14.2 for the explanation of this phenomenon.

Although not routinely performed during follow-up, this finding suggests that measurement of an intracardiac electrogram may be a useful tool in predicting impending lead malfunction.
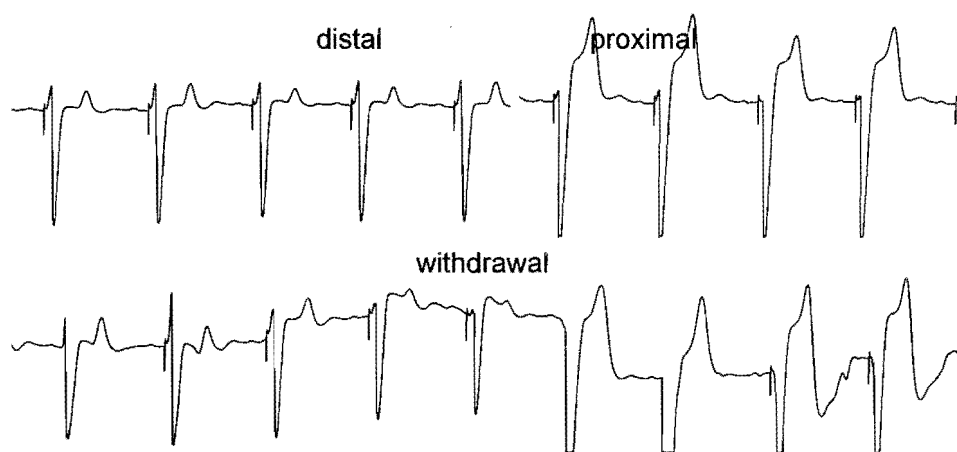
Fig. 1.7. Intracardiac electrogram recorded from the distal and proximal electrode of a bipolar lead for temporary pacing. The electrograms show a higher amplitude of the QRS complex from the proximal electrode than from the distal electrode. An injury pattern is observed at the proximal electrode, indicating that this electrode in in contact with the myocardium. These observations are suggestive of lead perforation, which is confirmed by the recording of the electrogram during withdrawal of the lead.

1.3.3.2 Evaluation of signals responsible for pacemaker inhibition and/or triggering

A signal with a well defined amplitude and frequency content can be sensed by a pacemaker resulting in inhibition and/or triggering of the pacing system. A marker channel indicates the presence of such signals without giving information about the nature of the signals. The telemetered electrogram is a real-time graphic display of the voltage differential between the electrode of the selected implanted pacing lead and the pulse generator in unipolar systems or between the two intracardiac electrodes in bipolar systems. The telemetered electrogram is very similar to that obtained directly from the pacing lead at the time of implantation. The interpreter should be aware of the fact that the bandpass filter of the amplifier of the electrogram differs from the bandpass filter of the pacemaker sensing amplifier [14]. The practical consequence of this difference is that portions of the signal recorded from the electrogram may have an amplitude higher than the sensitivity setting of the pacemaker, whereas they are ignored by the sensing amplifier. For example, T waves during ventricular stimulation usually have an

amplitude higher than the pacemaker sensitivity but they are rarely sensed by the pacemaker in spite of the relative high amplitude (Fig. 1.8).

If oversensing is observed, resulting in inhibition of a pacing system without indication of the nature of the signals responsible for inhibition on the surface ECG, the intracardiac electrogram can provide valuable information. It immediately discriminates between physiological signals (T-wave,,R-wave, P-wave) and signals generated in the pacing system due to lead malfunction.

For example, the signals generated by intermittent contacts in a pacemaker lead are easily recognized in the telemetered electrogram [15]. Figure 1.9 illustrates the presence of such signals generated in a bipolar lead, in which an intermittent contact between anodal and cathodal conductor is present. The morphology of these signals is specific and easily discriminated from physiological signals. They are characterized by a sharp and sudden deflection of the base line. From this recording it is most likely that the intermittent contact is provoked by ventricular contraction, as these signals only occur during ventricular systole. This indicates that the lead problem is located in the intracardiac section of the lead.

Fig. 1.8. ECG lead II (upper tracing), marker channel (middle tracing) and intracardiac electrogram (lower tracing) of a patient with an implanted VVI pacemaker, programmed at a sensitivity of 2.5 mV. The intracardiac electrogram shows a T wave, whose last portion has an amplitude of approximately 5.5 mV. In spite of this large amplitude the final portion of the T wave is not sensed by the pacemaker due to the specifications of the bandpass filter of the sensing amplifier. Non-sensing of the T waves is confirmed by the marker channel, which only indicates ventricular pacing (P).

The telemetered electrogram is also a valuable tool for patients with dual chamber pacemakers and intact retrograde conduction. Because these patients are prone to pacemaker mediated tachycardia (Chapter VII, 7.3), the interval from the ventricular stimulus to the retrograde P wave can be measured by recording the intra atrial electrogram during VVI pacing. This will facilitate the choice of the postventricular refractory period, which will prevent recognition of the retrograde P wave by the atrial sensing amplifier, thus precluding initiation of pacemaker mediated tachycardia.

The telemetered electrogram has limitations for paced rhythms, because telemetry of the near-field electrogram from the chamber being paced results in a massive deflection of the telemetered recording and an inability to identify any other waveform for a period varying from 50 to 200 ms, due to polarization at the electrode-myocardial interface (see also figure 1.5, AP and VP). In addition, due to the automatic gain setting of the telemetered electrogram, cardiac potentials between the stimulation electrodes are recorded with a relatively low amplitude.

1.3.4 Battery status

Pulse generator service life depends on many factors, such as initial power cell capacity, pacing rate, lead impedance, percentage pacing versus inhibition, pulse amplitude and pulse duration [16]. The distribution of these parameters in the individual patient makes pacemaker longevity hard to predict and battery status should be monitored carefully during pacemaker follow-up. Pacemaker telemetry enables the clinician and/or technician to measure battery voltage and/or battery impedance.
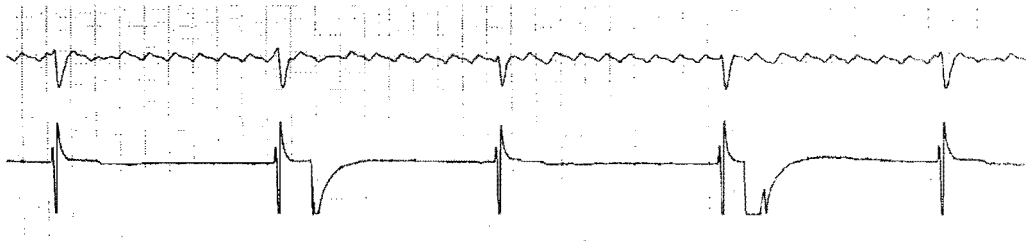
Fig. 1.9. Two channnel recording of the surface ECG (upper tracing) and the bipolar intracardiac electrogram (lower tracing) of a patient with an implanted VVI pacemaker. The pulse generator is programmed to a rate below the intrinsic rate. The electrogram shows sharp defelections, characteristic of an intermittent contact in the bipolar lead. The intermittent contact occurs during ventricular systole, which indicates that these contacts are provoked by ventricular systole. This observation suggests that the insulation defect (responsible for the intermittent contact) is located in the intraventricular portion of the lead.

The battery voltage gradually drops, whereas battery impedance increases during the pulse generator service life. When a predetermined level in the output voltage is reached the pulse generator should be replaced. Before this level is reached no changes in pacing parameters occur, so battery voltage and/or battery impedance is the only indicator for battery status. When this predetermined level in output voltage is reached, elective replacement is indicated by the pacemaker telemetry. Elective replacement time is confirmed by a change in (magnet) rate and/or pulse width, determined by the specifications of the pulse generator. In dual chamber pacing elective replacement is often associated with a change in pacing mode e.g. from DDD to VVI. Some dual chamber pacemakers programmed in the AAI mode can even revert to the VVI mode due to battery depletion [17]. If reversion in pacing mode is observed during pacemaker follow-up, battery depletion should first be excluded as the possible cause.

1.4 Radiological investigation

Posteroanterior and lateral chest X-rays are routinely used after pacemaker implantation to confirm and document adequate placement of the pacemaker lead(s). During follow-up in the

pacemaker clinic X-rays are not used routinely but it remain of value in confirmation of lead displacement and inspection for lead integrity.

Lead displacement may occur and is a common cause of no capture. Displacement of the lead usually occurs shortly after implantation (within 2 months) and rarely later during follow-up. Electrocardiographically it may be associated with an increase in stimulation threshold and intermittent noncapture while sensing may be maintained. This indicate lead displacement within the chamber where the lead was originally implanted (see chapter II, fig.2.16). Macrodisplacement, where the lead can be anywhere other than its original site of implantation, i.e., the superior or inferior vena cava, pulmonary artery, coronary sinus, right ventricle (for atrial leads), will electrocardiographically be associated with noncapture and failure to sense. Macrodisplacement of an atrial lead, in which the atrial lead is migrated across the tricuspid valve may result in ventricular stimulation, if the electrode is in contact with the ventricular endocardium (see Chapter IV, 4.5.2). In a very rare example of dislocation of the atrial and the ventricular lead of a dual chamber pacemaker, atrial pacing from the ventricular lead was observed on the ECG (Fig.1.10), while at the same time pacing and sensing from the atrial electrode was ineffective. This electrocardiographic observation confirmed macrodisplacement of both leads.
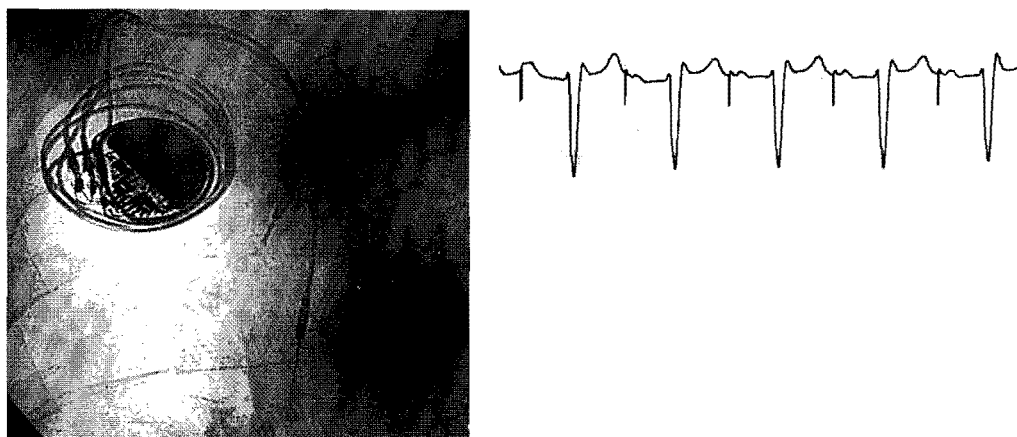
Fig. 1.10. DCI image and ECG (lead III) of a patient with an implanted bipolar DDD pacemaker. The pulse generator is programmed in the VVI mode, resulting in effective atrial stimulation, indicating macrodisplacement of the ventricular lead. This is confirmed by the DCI image, which shows total displacement of the atrial and ventricular lead into the superior vena cava and high right atrium respectively. Displacement was most probably caused by the patient himself, by manipulation and rolling around the pulse generator. Displacement was observed 2 weeks after implantation.

If during follow-up increase in lead impedance associated with an increase in stimulation threshold is perceived, the pacing system should be inspected radiographically. Instead of chest films, fluoroscopy is preferred under these conditions. In our clinic, the X-ray equipment in the catheterization laboratory is used for this purpose. This equipment allows complete rotation around the patient, using the most suitable right or left anterior oblique projection as well as cranial and caudal angulation. The freedom to use all these projections enables the clinician to

make a complete inspection of all the parts of the lead and pulse generator, even of those parts that are foreshortened or would be completely hidden in the standard posteroanterior and lateral X-rays. The Digital Cardiac Imaging (DCI) system, which is part of this equipment offers the possibility of pulsed fluoroscopy. The image obtained by pulsed fluoroscopy is digitized; therefore magnification and manipulation of brightness and contrast to identify essential parts of the pacing system can be easily performed. Even an incompletely tightened set screw can be visualized by this technique (see chapter IV, figs. 4.50, 4.51).

In case of suspected or evident galvanic (partial) interruption in the pacing system, indicated by an increase in lead impedance and noncapture or no output, the connector block should be inspected to determine that connector and pin are firmly in contact. Incomplete advancement of the pin into the connector receptacle (fig. 1.11) or incomplete tightening of the set screw should be excluded.

The conductor and possibly used adapters should be checked for interruptions. Lead fractures often occur at positions where strain is applied to the lead. In practice this will be between the connector block and the subcutaneous tissue at the point of fixation of the lead to the vein of entry or within the vein near the point of entry [18]. Infrequently lead fracture occurs within the superior vena cava or within the heart [19]. If lead fracture occurs outside the venous system repair may be possible and relatively easy in unipolar systems. Lead repair may not be possible if fracture occurs inside the venous system.
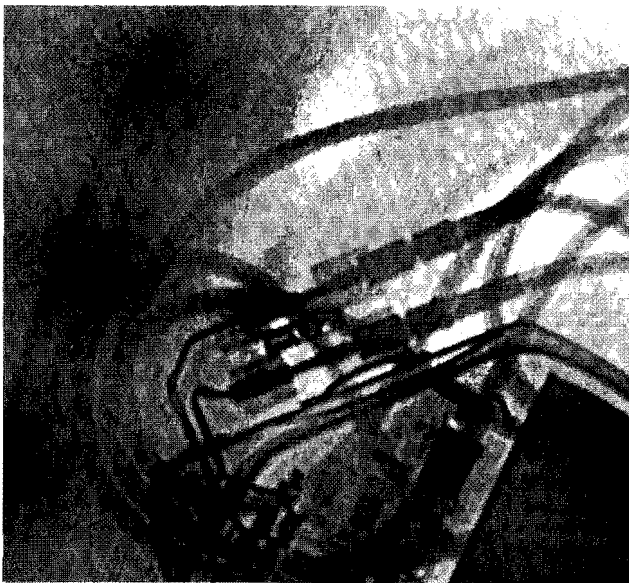
Fig. 1.11. DCI image of a bipolar DDD pacemaker illustrating incomplete advancement of the atrial lead pin (upper lead) into the connector receptacle of the pulse generator.

Incidental radiographic findings like "pseudofracture" may be misleading [20]. This applies to the indentation caused by a holding suture usually at the site of insertion of the lead into the vein. The conductor coil may be deformed by the suture giving a radiological finding similar to a lead fracture, but with a normal or unchanged lead impedance (Fig.1.12). This finding requires no immediate action but may later be a site of insulation breakdown or fracture of the whole lead. If this is observed in a lead with polyurethane insulation, which can easily lead to polyurethane

deterioration, it is better to replace the lead prophylactically. Use of a protection sleeve may avoid both the angiographic finding and its associated consequences.

1.5 Provocative testing procedures

In patients presenting in the pacemaker clinic with symptoms that are suggestive of intermittent pacemaker dysfunction, but who have a normal functioning pacing system, unchanged lead impedance and stimulation threshold at the time of arrival, provocative testing should be performed. Provocative testing consists of the evaluation of the influence of myopotentials on pacemaker sensing and manipulation of the pulse generator and lead in the subcutaneous part of the system to assess the continuity of the lead and lead connector.

1.5.1 Testing for myopotential sensing

Sensing of myopotentials in unipolar pacing systems is a well-known phenomenon. The potentials generated by the muscle adjacent to the pulse generator may be sensed by the pacemaker can, which is the anodal electrode of the unipolar system. In the majority of cases sensing of myopotentials results in cessation of stimulation in inhibiting systems. Incidentally undersensing can be observed due to sensing of myopotentials by the noise sampling circuitry, which results in reversion to asynchronous interference mode pacing. In triggered systems sensing of myopotentials results in rapid stimulation. The latter is rarely symptomatic except for the initiation of pacemaker mediated tachycardia in dual chamber systems (see chapter VII, 7.3).
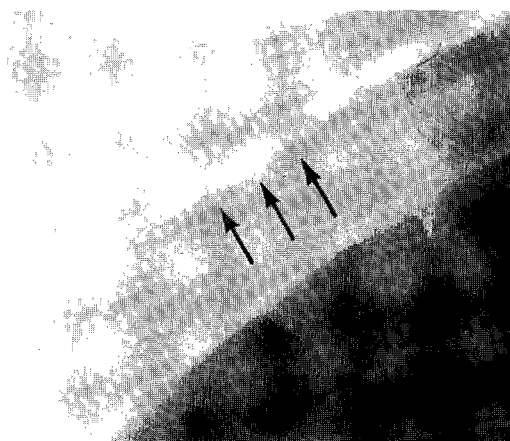
Fig. 1.12. Detail of an anteroposterior X-ray of a bipolar ventricular lead. There is identation of the lead (arrows), caused by holding sutures at the site of insertion of the lead into the vein. "Pseudofracture" of the lead, because effective stimulation and normal lead impedance was maintained.

To evaluate the pacemaker's susceptibility to myopotential sensing in unipolar systems, contraction of the muscle adjacent to the pulse generator should be provoked. The provocation method for the pectoral muscle, mentioned in the literature, is not standardised. Exercise of the pectoral muscle can be performed by sustained isometric pushing and pulling against a non-standardized load [21], adducting the upper arm at the site of pacemaker implantation against resistance [22], raising a weight or vigorously pressing the hands together [23], hyperadduction of the arm ipsilateral to the pulse generator, reaching as far as possible around the chest [24].

The latter method described by Fetter et al. compared this method of provocation with isometric tension by pushing with the biceps and pectoral muscles at maximum sustained force. Their

results showed that the reach exercise produced substantially more (28%) myopotential sensing than the push test.

For patients with pulse generators implanted in the abdominal region, the influence of myopotentials can be evaluated by instructing the patients to perform straight leg raising and sit-ups to a 45 degrees upright position [25].

In the previously mentioned provocative tests, myopotentials were detected at the anodal electrode of unipolar systems. Rarely myopotentials are sensed at the ventricular cathodal electrode [26]. These myopotentials are generated by the diaphragm during respiration. If sensing of the diaphragm myopotentials is suspected, these myopotentials may be provoked by active contraction of the diaphragm during deep inspiration, straining, Valsalva maneuver and coughing. Perforation of the ventricular stimulation electrode may sometimes be responsible for pacemaker inhibition by diaphragmatic myopotentials [27]

1.5.2 Testing for lead integrity

The most vulnerable part of the lead system is located between the connection of the lead and pulse generator and the point of entry in the venous system. This implies that the majority of lead problems are located in this area. If a patient visits the pacemaker clinic with symptoms that suggestive of pacemaker dysfunction, the subcutaneous part of the pacing system should be checked for lead integrity. This can be done by gentle manipulation of the pulse generator and the subcutaneous tissue between pulse generator and venous entry, while recording the ECG. Attenuation of the pacemaker stimulus associated with loss of capture is indicative of a lead, connector or adapter failure (see chapter IV, figs. 4.46, 4.49). The attenuation of the pacemaker stimulus can be caused by a (partially) fractured lead or a short circuit between the anodal and cathodal electrode. If it is possible to measure lead impedance during attenuation of the pacemaker stimulus, discrimation between a lead fracture and a short circuit is easy. In case of a fractured lead, lead impedance will be increased, whereas a short circuit will show a decrease in lead impedance [28]. If pacemaker dysfunction is observed during manipulation, with the inability to measure lead impedance in this phase, the maneuver should be repeated during fluoroscopy, to exclude or confirm lead fracture (see chapter IV, figs. 4.47, 4.48).

If testing for myopotentials is performed, the operator should be aware that contraction of the muscle adjacent to the pulse generator may provoke lead system failure. If cessation of stimulation is observed during muscle contraction, dicrimination should be made between suppression of the pacing system by sensing the myopotentials and a short circuit or broken conductor provoked by muscle contraction. This can be done by programming the pacing system in the asynchronous (non sensing) mode, while repeating the maneuver. If cessation of pacing is observed in the asynchronous mode, it excludes sensing of myopotentials as the possible source for dysfunction and indicates a short circuit or broken conductor in the system. On the contrary if normal pacemaker function is observed in the asynchronous mode, sensing of myopotentials can be concluded.

1.6 Chest wall stimulation

Chest wall stimulation is performed by connecting an external pacemaker to two ECG electrodes that are applied to the chest wall. The ECG electrodes should be positioned as close as possible to the anodal and cathodal electrode of the implanted pacing system. After adjusting the output of the external pacemaker, the pulse can be sensed by the implanted pacing system and, depending

on the pacing mode, the implanted pacemaker will be suppressed (inhibited mode), or a pacemaker stimulus will be evoked (triggered mode). Sensing of chest wall stimuli is easily performed in unipolar systems but might be difficult or impossible in bipolar systems, in which the distance between anodal and cathodal electrode is very short.

If the chest wall electrodes are connected to a stimulator used for electrophysiological studies, the pacemaker pulse of the implanted pacemaker can be sensed by the stimulator amplifier. This enables the clinician to apply one or more programmed stimuli (stimuli with adjustable coupling intervals) to the implanted pacing system.

1.6.1 Chest wall stimulation in single chamber inhibited systems

Chest wall stimulation applied in single chamber inhibiting systems (AAI, VVI) at a rate higher than the rate of the implanted pacing system will result in complete inhibition of the pacing system. It enables the clinician to evaluate the underlying cardiac rhythm [29,30]. If the patient has no underlying rhythm, the external pacemaker can be switched off or one of the electrodes detached from the chest wall to restore normal pacing. Inhibition of the implanted pacemaker by chest wall stimuli suggests that the sensing circuit is intact, but gives no information about the value of the sensitivity level of the pacemaker.
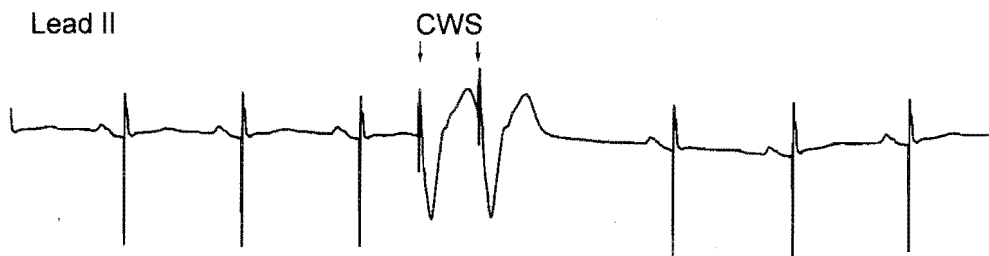
Fig. 1.13. Single channel ECG (lead II) of a patient with an implanted unipolar pacemaker, programmed in the triggered mode. The intrinsic rhythm shows a pseudofusion pattern, because the pacemaker stimuli are emitted in the QRS complex. Two programmed chest wall stimuli (CWS) are applied to the patient. These stimuli are sensed by the implanted system resulting in ventricular stimulation at intervals corresponding with the intervals of the programmed chest wall stimuli. Demonstration of the use of chest wall stimuli for programmed stimulation.

Programmed chest wall stimuli can be used to determine the refractory period of the implanted pacemaker. If the coupling interval between a paced beat and the programmed chest wall stimulus is shorter than the refractory period the escape interval will remain unchanged. If the coupling interval is longer than the refractory period, the escape interval will be prolonged. In pulse generators with a marker channel that indicates sensing in the refractory period, the absolute refractory period can be determined in a similar way.

The electrocardiographic appearance of oversensing and undersensing due to noise mode reversion, can be imitated by application of chest wall stimuli.

1.6.2 Chest wall stimulation in single chamber triggered systems

Chest wall stimulation applied in single chamber triggered systems (AAT, VVT) or single chamber systems programmable in the triggered mode will result in an increase in pacing rate of

25

the implanted system if the rate of chest wall stimulation is higher than the rate of the implanted pacemaker. Chest wall stimulation performed by a stimulator for electrophysiological studies enables the clinician to perform programmed stimulation (fig. 1.13) or high rate stimulation. The mechanism and time interval of the sensing inhibiting period in triggered single chamber systems (see chapter V, 5.9) can also be demonstrated by this technique.

Chest wall stimulation by means of a rate adaptive pacemaker with an activity sensor can be useful in patients in whom the implanted pacemaker has to be replaced e.g. because of battery depletion (Fig.1.14). By instructing the patient to perform exercise, the pacing rate of the external rate adaptive pacemaker will increase. This means that the implanted system (which is in the triggered mode) follows the pacing rate indicated by the rate adaptive pacemaker. This technique enables the clinician to compare exercise tolerance with rate adaptive versus fixed rate pacing, and the patient's benefit from the pacing mode can be evaluated before replacement of the pulse generator.

1.6.3 Chest wall stimulation in dual chamber systems

In dual chamber systems with sensing in the atrial and ventricular channel, pacemaker reponse is determined by the channel that senses the chest wall stimulus. A chest wall stimulus sensed in the atrial channel will inhibit atrial stimulation and evoke a ventricular stimulus. If this is the intention of the investigator, the chest electrodes should be carefully positioned as close as possible to the anodal and cathodal electrode of the atrial channel. Also adjustment of the amplitude of the external pulse is critical because sensing of the chest wall stimulus in the ventricular channel will result in complete inhibition of the system. To obtain a functional separation of the sensing channels for this condition, the atrial sensitivity has to be programmed to its most sensitive level, whereas the ventricular sensitivity should be low. In pacing systems with programmable electrode configuration (uni- or bipolar), independently for both channels, the atrial channel should be programmed in the unipolar and the ventricular channel in the bipolar configuration.
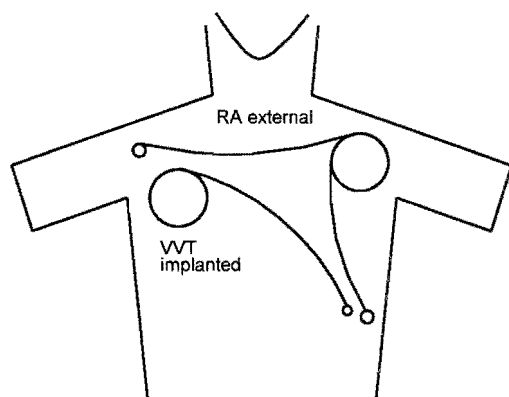
Fig. 1.14. Schematic presentation of the use of chest wall stimulation in the evaluation of the clinical usefulness of rate adaptive pacing in an individual patient. The implanted VVT pacemaker is triggered by the stimulaton pulses of an external rate adaptive pacemaker (RA external) with an activity sensor. The pulses are applied to the chest wall electrodes.

This form of chest wall stimulation is a simple technique for verification of some of the programmable functions of pulse generators, such as maximum tracking rate, duration of the atrial refractory period and characterization of the predicted response to the programmed upper rate [31].

Because chest wall stimuli sensed in the atrial channel and not sensed in the ventricular channel result in ventricular stimulation without preceding atrial depolarization it may induce retrograde atrial activation in patients with intact ventriculo-atrial conduction. This may lead to pacemaker circus movement tachycardia (see chapter VII, 7.3). Chest wall stimulation can be used for noninvasive evaluation of ventriculo-atrial conduction and prevention of pacemaker mediated tachycardia in predisposed patients [32].

Chest wall stimuli that are sensed by the ventricular channel in dual chamber pacemakers will inhibit the pacing system completely. It does not matter whether these stimuli are also sensed by the atrial channel or not, as the electrocardiographic response of the pacing system is identical. Chest wall stimuli sensed by the ventricular channel can be used to inhibit the pacing system and to evaluate the underlying intrinsic rhythm. Programmed stimuli can be used to demonstrated the effect of ventricular oversensing in dual chamber pacing systems (see Chapter VI, figs. 6.44,6.45,6.46).

1.6.4 Chest wall stimulation for detection of insulation break
Chest wall stimuli can be used in unipolar systems to indicate the presence of an insulation break. For this test three ECG skin electrodes are applied to the patient's chest. Two electrodes are positioned as close as possible to the anodal and cathodal electrode of the pacing system. The third electrode is positioned above the site of the suspected insulation break, which is usually located between the pulse generator and the venous entry of the lead. The lowest amplitude of the chest wall stimulus applied to the electrodes above the anodal and cathodal electrode, at which sensing of the implanted system occurs, is determined. This value is compared to the value obtained by repeating the test, while using the electrode above the anodal electrode` and the electrode above the suspected insulation break location. When both values are approximately the same, it indicates that the chest wall stimulus in the second test was detected by the pacing system similar to the first test. This can only be explained by the presence of an insulation defect, causing penetration of the chest wall stimulus to the sensing amplifier of the pacing system.

If the value of the second test is more than five times higher than the result of the firstt, proper insulation is proved. This number was found empirically in a group of 541 patients during a two years follow-up [33].

1.7 Summary and conclusions
During pacemaker follow-up a number of techniques are used to ascertain the quality of the pacing system or to detect (impending) malfunction even in the presence of a normal ECG. The threshold for pacing and sensing are determined, by using the pacemaker programmer. Increase in stimulation threshold, after having reached the chronic value, is often an indicator of (impending) lead malfunction. The same is true for the sensing threshold. A significant change in lead impedance associated with one of these observations is an extra argument for (impending) lead malfunction.

Battery status is easily accessed by telemetry and excludes the power source as the cause of noncapture or no output. A battery level below the point for elective replacement might show a change in pacing mode in dual chamber systems.

Lead impedance measured by pacemaker telemetry has limitations. The measured value depends on the method of measurement (depending on make and type), and the pacemaker ouput setting. This implies that comparison of lead impedance in different patients is difficult and, that lead

impedance followed in the same patient has to be performed at the same output setting of the pulse generator. Pulse waveform analysis is a more accurate method but requires additional equipment.

A normal lead impedance does not always imply a normal lead. Because lead impedance is measured during the pacemaker pulse, intermittent interruption in the conductor or intermittent contact between cathodal conductor and anodal conductor or electrode, can take place in the time interval between two stimuli, without affecting the lead impedance. Because these intermittent contacts usually result in signals that can be sensed by the pulse generator, these problems can be elucidated in the marker channel or intracardiac electrogram. The marker channel is extremely useful in those case where the signals generated by the intermittent contacts occur in the refractory period of the pulse generator. Due to the occurrence of the signals in the refractory period, the escape interval is not affected, so electrocradiographically normal pacemaker function with a normal lead impedance is observed, in the presence of a lead integrity problem as illustrated in figure 1.6.

Provocative maneuvers like muscle contraction and manipulation should be performed in patients presenting with symptoms suggestive of pacemaker dysfunction. Provocative maneuvers are helpful to discriminate between oversensing from myopotentials with resulting inhibition and lead malfunction resulting in noncapture or no output. If these test are negative and there are no radiological abnormalities, a 24 hour Holter recording should be performed. If symptoms are associated with pacemaker dysfunction in the Holter recording, the pacing system should be re-evaluated to discover the cause of malfunction.

References.

1. Nickel J. Oscilloskopisches Verfahren zur unblutigen Analyse von Überleitungsstörungen implantierter elektrischer Schrittmacher. Z Kreislauff 1964; 53: 1149-1155.

2. Van den Berg Jw, Rodrigo FA, Thalen HJTh, et al. Photo-analysis of the condition of implanted pacemakers and electrode circuits I. Proceedings Series C, 70, No. 4, Koninkl Nederl Akademie van Wetenschappen 1967; 419-447.

3. Sedney MI. The implantable cardiac pacemaker. Implications of measurements during implantation and follow-up. Drukkerij JH Pasmans BV, 's Gravenhage. Thesis, Leiden 1986.

4. Geheniau J, Thijssen A. Report on an automatic equipment for control of pacemaker patients (In Dutch). MSc. thesis, Dept. of Electr. Engng., Eindhoven University of Technology, 1976.

5. Sedney MI, De Melker R, Waegemakers A, et al. Computerized pacemaker pulse waveform analysis. (abstract) PACE 1991; 14: 672.

6. Castellenet MJ, Garza J, Shaner SR, et al. Telemetry of programmed and measured data in pacing system evaluation and follow-up. J. Electrophysiology 1987; 1; 360-375.

7. Levine PA. Utility and clinical benefits of extensive event counter telemetry in the follow-up and management of the rate-modulated pacemaker patient. Siemens Pacesetter, Inc, February 1992, pp. 1-26.

8. Barold SS, Ong LS, Heinle RA. Stimulation and sensing thresholds for cardiac pacing: Electrophysiological and technical aspects. Progr. Cardiovasc. Dis. 1981; 14: 1-9.

9. Platia EV, Brinker JA. Time course of transvenous pacemaker stimulation impedance, capture threshold, and electrogram amplitude. PACE 1986; 9: 620-625.

10. Furman S. Personal communication, February 8, 1994.

11. Duffin EG. The marker channel: A telemetric diagnostic aid. PACE 1984; 7: 1165-1169.

12. Kruse I, Markowitz T, Rydén L. Timing markers showing pacemaker behavior to aid in the follow-up of a physiological pacemaker. PACE 1983; 6:801-805.

13. Van Beek GJ, den Dulk K, Lindemans FW, et al. Detection of insulation failure by gradual reduction in noninvasively measured electrogram amplitudes. PACE 1986; 9: 772-775.

14. Levine PA, Sholder J, Duncan JL. Clinical benefits of telemetered electrograms in assessment of DDD function. PACE 1984; 7: 1170-1177.

15. Sarmiento JJ. Clinical utility of telemetered intracardiac electrograms in diagnosing design dependent lead malfunction. PACE 1990; 13: 188-195.

16. Gerrard DJ. Pacemaker longevity modelling. PACE 1988; 11: 302-307.

17. Medtronic dual chamber pulse generators; Physician's information manual, 1992.

18. Alt E, Volker R, Blomer H. Lead fracture in pacemaker patients. J Thorac Cardiovasc Surg 1987; 35: 101-104.

19. Clarke B, Jones S, Gray HH, et al. The tricuspid valve: An unusual site of endocardial pacemaker lead fracture. PACE 1989; 12: 1077-1079.

20. Witte AA. Pseudo-fracture of pacemaker lead due to securing suture: A case report. PACE 1981; 4: 716-718.

21. Secemsky SI, Hauser RG, Denes P, et al. Unipolar sensing abnormalities: Incidence and clinical significance of skeletal muscle interference and undersensing in 228 patients. PACE 1982; 5: 10-19.

22. Breivik K, Ohm O-J, Engedal H. Long-term comparison of unipolar and bipolar pacing and sensing, using a new multiprogrammable pacemaker system. PACE 1983; 6: 592-600.

23. Furman S. Pacemaker follow-up. In S Furman, DL Hayes, DR Holmes, Jr (Eds). : A practice of cardiac pacing. Mount Kisco, NY, Futura Publishing Company, Inc, 1989, p.530.

24. Fetter J, Bobeldyk GL, Engman FJ. The clinical incidence and significance of myopotential sensing with unipolar pacemakers. PACE 1984; 7: 871-881.

25. Michalik RE, Williams WH, Hatcher CR. Myopotential inhibition of unipolar pacing in children. PACE 1985; 8: 25-31.

26. Van Gelder B, El Gamal M, Bracke F, et al. Are bipolar systems prone to oversensing at high sensitivity setting. (abstract) PACE 1991; 14: 731.

27. El Gamal M, van Gelder B. Suppression of an external demand pacemaker by diaphragmatic myopotentials: A sign of electrode perforation ? PACE 1979; 2: 191-195.

28. Van Gelder BM, Bracke FALE, El Gamal MIH. Adapter failure as a cause of pacemaker malfunction. PACE 1993; 16: 1961-1965.

29. Staessen J, Ector H, De Geest H. The underlying heart rhythm in patients with an artificial cardiac pacemaker. PACE 1982; 5: 801-807.

30. Marinato PG, Bressan M, Buja GF, et al. Programmed chest-wall stimulation to evaluate the progress of A-V block after pacemaker insertion in patients with trifascicular disease. PACE 1982; 5: 658-666.

31. Barold SS, Falkoff MD, Ong LS, et al. Clinical usefulness of chest wall stimulation in patients with implanted DDD pacemakers. (abstract) PACE 1983; 6: A44

32. Den Dulk K, Lindemans F, Wellens HJJ. Noninvasive evaluation of pacemaker circus movement tachycardias. Am J Cardiol 1984; 53: 537-543.

33. Ferek B, Pasini M, Pustisek S, et al. Noninvasive detection of insulation break. PACE 1984; 7: 1063-1068.

Chapter II. **Morphology of Stimulated Depolarization**

Introduction
The evaluation of the implanted pacemaker function through the ECG interpretation is generally limited to the effectiveness of stimulation and adequate sensing of the pacemaker system. Little or no attention is paid to the morphology of the stimulated depolarization. The position of the stimulation electrode can be deduced from the morphology of the stimulated depolarization, because of its direct relationship with the site of stimulation. This is important during implantation of a ventricular lead, to prevent placement of the electrode in the coronary sinus [1], and during follow up shortly after implantation. During this period dislocation of the lead, even in the presence of effective stimulation and sensing, can be detected by careful appraisal of the morphology of the stimulated QRS complex [2].
Changes in the morphology of the stimulated QRS complex related to ischemia and myocardial infarction will not be discussed in this chapter as they are generally associated with ST segment changes and not specifically in changes in depolarization.

2.1 P wave morphology during atrial stimulation
Determination of the stimulation site in atrial pacing in unipolar systems may be difficult because of the large stimulus artifact.
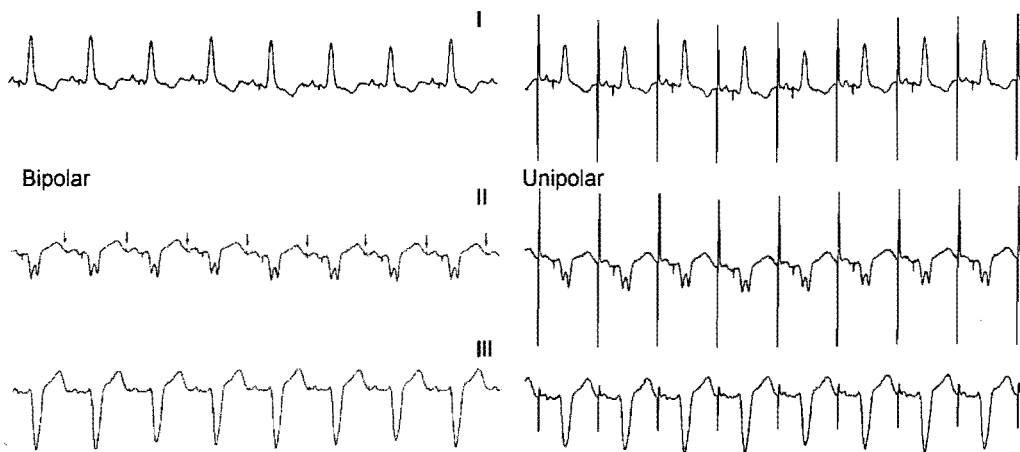
Fig. 2.1. Three channel ECG, lead I,II,III, of a patient with an implanted DDD pacemaker. In the left panel the atrial output is programmed in the bipolar configuration, showing a small pacemaker stimulus (arrows) with low amplitude without significant deformation of the P wave. In the right panel, where the atrial output is programmed in the unipolar configuration, the initial portion of the P wave is deformed by the pacemaker stimulus and its subsequent exponential decay.

This large pacemaker stimulus associated with an overshoot (with subsequent exponential decay) may obscure the atrial depolarization and even the presence or absence of capture is somestimes hard to identify under these conditions (see also chapter IV 4.3.2).
Due to the overshoot of the pacemaker stimulus determination of the morphology of the atrial depolarization is impossible in every ECG lead. This implies that the morphology of the

stimulated atrial depolarization in unipolar systems can only be determined from ECG leads with a low amplitude stimulus artifact. In practice this means only in those leads that are more or less perpendicular to the anodal-cathodal dipole. Because there are only a few ECG leads fulfilling this criterium, only a rough estimation of the direction of atrial depolarization and thus the location of the stimulating electrode can be made.

In bipolar pacing systems the pacemaker stimulus is small with a small overshoot, so determination of the morphology and the location of the stimulation electrode is relatively easy and can be performed more accurately (Fig. 2.1).

2.1.1 Stimulation from the right atrial appendage

The right atrial appendage most commonly used for placement of endocardial atrial J-leads. In patients with previous cardiac surgery in whom the atrial appendage has been cannulated or amputated, finding a stable position in the atrial appendage may be more difficult but it is usually still possible [3,4].

Atrial depolarization originating from the right atrial appendage is directed inferiorly and posteriorly and from right to left. This is illustrated in figure 2.2 (panel RAA), showing a positive P wave in lead I (leftward axis), positive P wave in leads II and III indicating superior-inferior direction, and a negative P wave in the precordial leads V1, V2, V3 indicating antero-posterior direction of the atrial depolarization [5]. When comparing the morphology of the atrial depolarization in right atrial appendage stimulation to the morphology of high right atrial stimulation (morphology similar to sinus P waves), there are only some slight differences mostly related to the initial portion of the stimulated atrial depolarization. In lead I, the initial part of the P wave during right atrial appendage stimulation is more or less isoelectric whereas there is a positive deflection in the same part during high right atrium stimulation. This is explained by the fact that during stimulation in the right atrial appendage there is initially a depolarization vector with a left and a right axis of more or less equal size. The rightward directed vector is short in duration because the amount of atrial myocardium located on the right side of the electrode is small, so total depolarization of this part is achieved before depolarization of the atrial myocardium in the left sided location has been accomplished. The latter results in a positive deflection in the last portion of the P wave in lead I. Because of the anatomical anterior orientation of the right atrial appendage, the antero-posterior oriented depolarization is more pronounced during right atrial appendage stimulation, compared with high right atrial stimulation, as illustrated in the precordial leads V1, V2, V3 (Fig. 2.2, panel RAA).

2.1.2 Stimulation from the free right atrial wall

Stimulation from the free right atrial wall is generally not the first choice for placement of endocardial atrial leads. However, when no adequate pacing and/or sensing threshold can be obtained from the right atrial appendage, implantation of active fixation leads in the free atrial wall can be used as an alternative technique [6,7]. Care should be taken during implantation of such a lead in the free atrial wall. Because the stimulating electrode may be located in the proximity of the phrenic nerve, atrial pacing from this site can be associated with phrenic nerve stimulation. High output pacing should be performed during implantation to exclude phrenic nerve stimulation. Stimulation from the atrial free wall is characterized by a leftward axis illustrated by a positive P wave in lead I. In the leads II and III the P wave is biphasic with a negative last portion of the P wave indicating depolarization in a superior direction. The latter is

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explained by the anatomical position of the left atrium, which has a superior orientation for an electrode positioned in the mid free atrial wall. This is also confirmed by the highest P wave
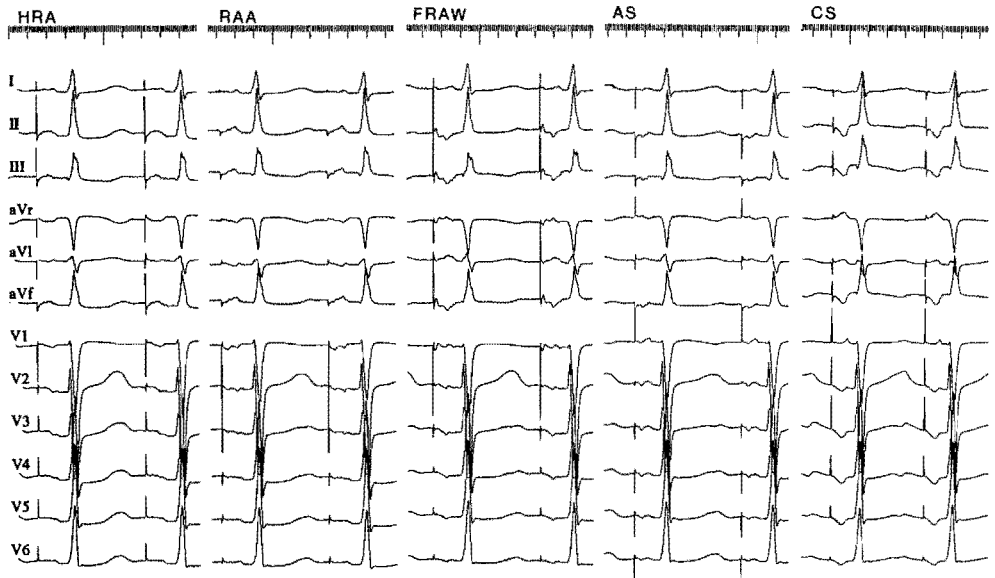
Fig. 2.2. Twelve lead ECG showing the difference in morphology of the stimulated atrial depolarization with varying stimulation sites. HRA = high right atrium; RAA = right atrial appendage; FRAW = free right atrial wall; AS = atrial septum; CS = coronary sinus. For further explanation see text.

amplitude in lead aVl in comparison with right atrial appendage and high right atrium stimulation and the predominantly negative P wave in lead aVf (Fig 2.2, panel FRAW).

2.1.3 Stimulation from the right atrial septum
Reports on permanent atrial stimulation from the right atrial septum are limited. Only leads with active fixation can be employed for permanent pacing from this site. Although advocated by Stirbys in 1986 [8] with the argument that this technique presented a lesser risk of perforation of the atrial wall and avoided phrenic nerve stimulation, no widespread use of this method followed his publication. The same author published in 1991 a paper describing an efficient technique for implantation of atrial leads in the right atrial appendage from the left subclavian vein [9]. In this communication interatrial septum pacing was not mentioned and it seemed to have been abandoned.

Stimulation from the right atrial septum is characterized by a biphasic (negative-positive) P wave in the leads II and III (Fig. 2.2, panel AS). The first part of the P wave is negative in these leads because the atrial septum is depolarized from inferior to superior. After depolarization of the atrial septum the depolarization is propagated along the left and right atrial free wall in an superior-inferior direction causing a last positive portion of the P wave. In the presence of normal AV conduction, the time interval between atrial stimulation and ventricular depolarization is

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shorter during atrial septum stimulation than during stimulation from the right atrial appendage, since during septal stimulation the electrode is in the proximity of the AV node (Fig.2.3).
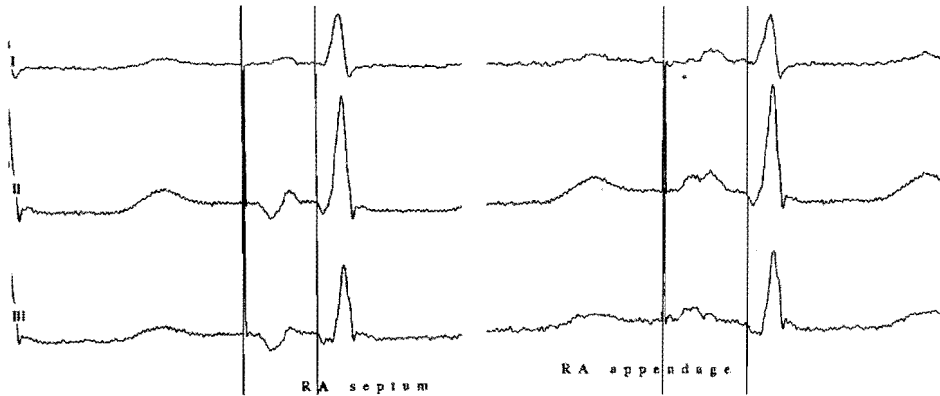
Fig. 2.3. Three channel ECG, lead I,II,III showing the difference in morphology of the stimulated P wave from the right atrial appendage and right atrial septum. In the presence of normal AV conduction, the time interval from atrial stimulation to ventricular depolarization is shorter during atrial septum stimulation (110 ms) than during stimulation from the right atrial appendage (127 ms).

2.1.4 Stimulation from the coronary sinus

Stimulation of the left atrium and recording of left atrial depolarization is routinely used during electrophysiologic investigations by placement of multipolar catheters into the coronary sinus [10].

In the past, long term atrial pacing has been reported employing transvenous leads implanted in the proximal portion of the coronary sinus [11,12,13]. After development of preshaped atrial J-leads and leads employing active fixation mechanisms implantation of leads in the coronary sinus for permanent atrial pacing and/or sensing was abandoned. Specific problems with coronary sinus pacing include inaccurate positioning within the coronary sinus, and inadvertent ventricular pacing or inappropriate sensing of ventricular potentials due to the position of the electrode in the direct proximity of the ventricle. Coronary sinus leads are not fixed but move within the coronary sinus. This movement, which may result in alternating atrial and ventricular stimulation, is illustrated in figure 2.4.

The morphology of the P wave during pacing from the coronary sinus is characterized by a negative P wave in the ECG leads I,II, III and aVf. A negative P wave in lead II, III and aVf indicates atrial depolarization from inferior to superior, while the rightward axis is illustrated by the negative P wave in lead I (Fig. 2.2, panel CS).

2.2 Morphology of the QRS complex during ventricular stimulation

Left ventricular endocardial stimulation, during electrophysiological studies, has been used to determine the site of origin of ventricular arrhythmias by comparing morphology of the stimulated QRS complex to the configuration of the QRS complex during these ventricular

arrhythmias. In stimulation for bradycardia performed endocardially from the right ventricle, less attention is paid to the morphology of the stimulated QRS complex.
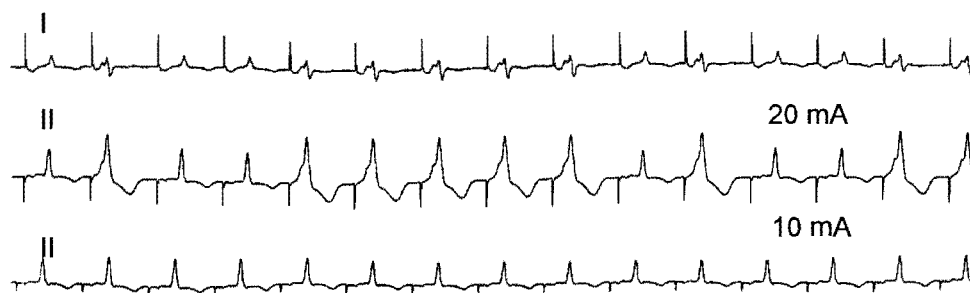
Fig. 2.4. Two channel ECG lead I and II (upper tracings) recorded from a patient with a bipolar lead positioned in the coronary sinus. Alternating atrial and ventricular stimulation is recorded at a pacemaker output of 20 mA, probably due to movement of the electrode. After decreasing the stimulation current to 10 mA (lower tracing) only atrial stimulation is observed.

The higher amplitude of the stimulated QRS complex, however, offers the possibilty of determining the location of the ventricular electrode even during unipolar stimulation, and this in contrast to unipolar atrial stimulation. During implantation of endocardial ventricular leads, attention should be paid to the morphology of the stimulated QRS complex with respect to its bundle branch block appearance [15]. A right bundle branch block pattern, indicating that the left ventricle is stimulated first, is very unusual under normal conditions (see also chapter II, 2.3) and inadvertent malplacement of the endocardial lead into the coronary sinus and/or one of its tributaries should be excluded.

During follow up, a changing morphology of the stimulated QRS complex may indicate a change in pacing site, due to displacement of the stimulation electrode. The interpreter, however, should be aware of the fact that other clinical factors such as increasing size of the heart, myocardial infarction and even a change in output setting in bipolar pacemakers (see also chapter II, 2.4) may change the morphology of the stimulated QRS complex without a change in the position of the stimulation electrode.

2.2.1 Morphology of the QRS complex during right ventricular apex stimulation

During right ventricular apical stimulation the morphology of the QRS complex exhibits a typical left bundle branch block pattern in leads I, aVl and V1. This pattern is often less characteristic in the left precordial leads V5 and V6, which may show deep S waves. The latter is explained by the fact that the main vector of ventricular depolarization is moving away superiorly from the horizontal level where V5 and V6 are recorded [16].

The main electrical axis in the frontal plane has a superior orientation because the sequence of activation travels from apex to base. The inferior to superior direction of activation is reflected in the ECG by the negative deflection of the QRS complex in leads II, III and aVf [17]. The QRS complex in lead I during RV stimulation generally shows a positive deflection, indicating a right to left depolarization vector in the frontal plane.

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The right to left vector is the result of both a left to right vector (mainly due to right ventricular activation) and a right to left vector (mainly due to septal and left ventricular activation). Because
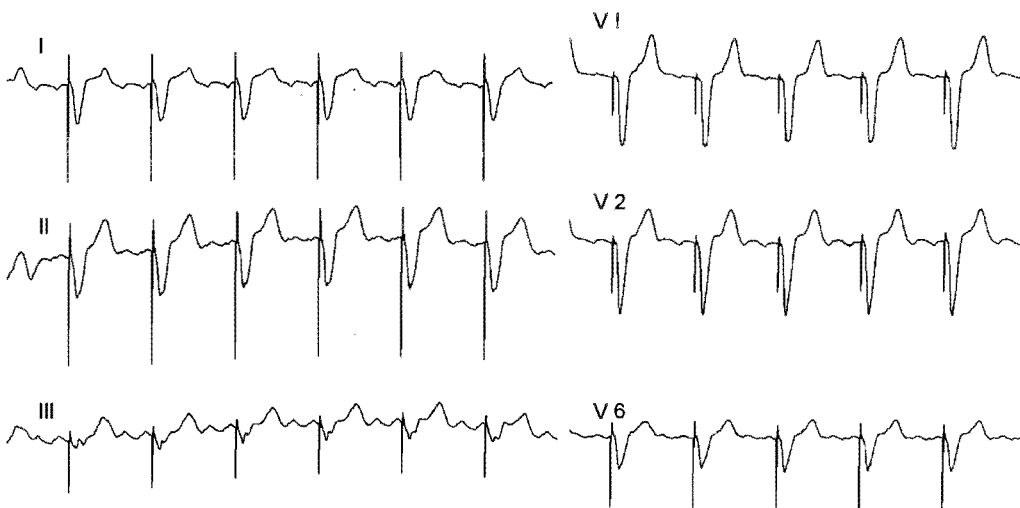
Fig. 2.5. Six channel ECG (I,II,III,V1,V2,V6) of a patient with dextrocardia after implantation of a ventricular lead in the right ventricular apex. Note the QS pattern in lead I and V6 indicating a left to right ventricular activation, this in contrast with a normal heart position. Lead V1 still shows a left bundle branch block pattern (see also fig. 2.6A-B).
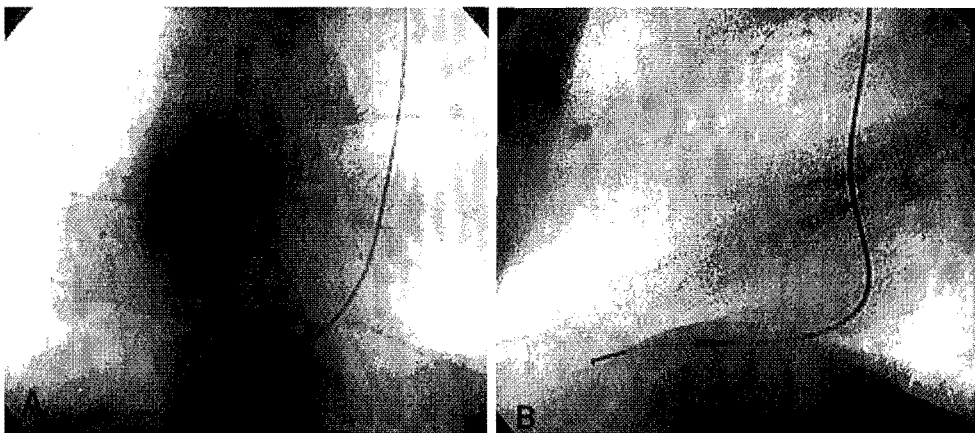
Fig. 2.6. Anterior-posterior (A) and lateral (B) X-ray of a patient with dextrocardia after implantation of a ventricular lead in the right ventricular apex position (see also fig. 2.5).

the latter dominates, due to the larger amount of septal and left ventricular depolarized myocardium, the result is a right to left vector and a positive deflection in lead I (Figs. 2.7 panel RVA, 2.8).

A rare exception to the electrocardiographic pattern of right ventricular apex stimulation can be encountered in dextrocardia, which has a very low incidence (1/5000) in the general population
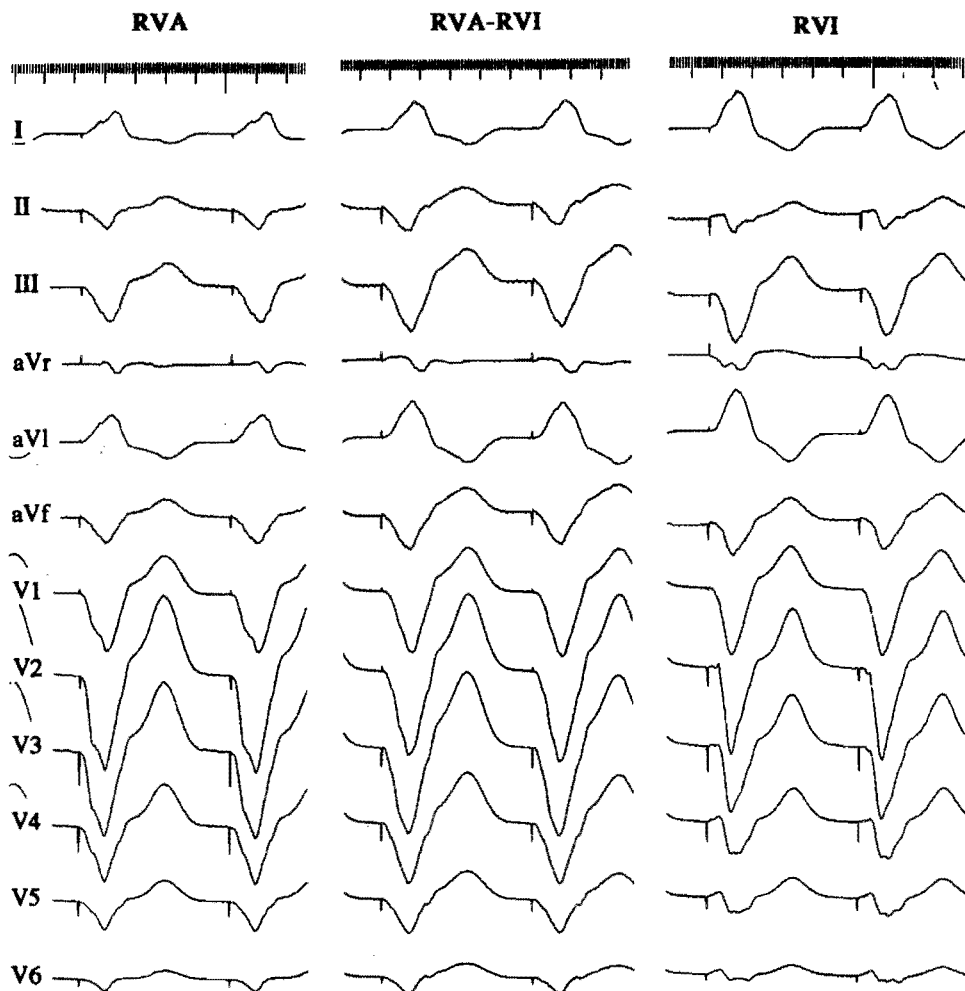
Fig. 2.7. Twelve lead ECG comparing the morphology of the stimulated QRS complex during right ventricular apex (RVA), an intermediate position between RVA and RVI (RVA-RVI) and right ventricular inflow (RVI) stimulation. Note the increase in amplitude of the QRS complex in lead I during right ventricular inflow stimulation and a shift in electrical axis showing a decrease in left axis deviation during inflow stimulation. The latter is illustrated by an increase in the amplitude of the QRS complex in lead III, a decrease in lead II, and an increase in amplitude in aVL during inflow stimulation. See also figures 2.8. and 2.20.

[18]. Most specific is the QS pattern in lead I and V6 indicating a left to right activation in the frontal plane. A left bundle branch block pattern is still present in lead V1 (Figs. 2.5,2.6 A-B).
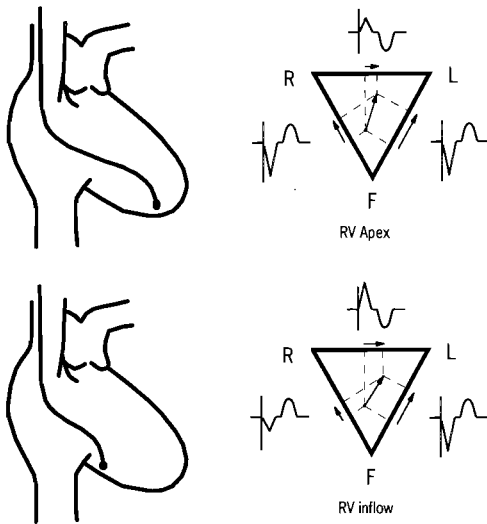
Fig. 2.8. Schematic presentation of right ventricular apical (RV apex) and right ventricular inflow (RV inflow) stimulation and its effect on the amplitude of the stimulated QRS complex in the leads I, II and III. See also figure 2.7.

2.2.2 Morphology of the QRS complex during right ventricular inflow stimulation

In right ventricular inflow stimulation, the morphology of the QRS complex will also show a left bundle branch block pattern. The major difference between inflow and apical stimulation is the direction of the frontal axis. Due to the anatomical location of the right ventricular inflow and therefore the dimished ventricular myocardium that can be depolarized in a left to right direction, the right to left vector will increase and the inferior superior vector (which is basically still present) will turn towards the left shoulder, showing diminished left axis deviation. This is reflected in the ECG by an increase in the amplitude of the positive QRS complex in lead I. The change of the inferior-superior vector towards the left shoulder is illustrated electrocardiographically by an increase of the amplitude of the QRS complex in lead III, which will be more negative than in lead II, and an increase of the amplitude of the positive QRS complex in lead aVl during inflow stimulation to positive during apical stimulation (Figs. 2.7 panel RVI, 2.8).

The changes in morphology of the QRS complex when comparing right ventricular apex with right ventricular inflow stimulation are similar to the changes that can be observed during endocardial bipolar pacing at different output settings of the pulse generator (see chapter II, 2.4).

2.2.3 Morphology of the QRS complex during right ventricular outflow stimulation

The morphology of the QRS complex during right ventricular outflow pacing shows a left bundle branch block pattern indicated by a completely negative deflection in lead V1 and positive deflection in the last portion of the QRS complex in lead I. It is not surprising that a shift in electrical axis, in comparison with inflow and apical pacing, occurs independent of whether the right ventricular outflow is stimulated from the septum or the lateral wall (Fig. 2.9).

Under both conditions the electrical activation travels from superior to inferior. This is reflected in the ECG by the positive deflection of the QRS complex in leads II and III and a positive deflection in aVf. The left bundle branch block pattern already indicates depolarization travelling from right to left, which is confirmed by the positive deflection in lead I. The only lead that discriminates septal pacing from pacing from the lateral wall is lead aVL.

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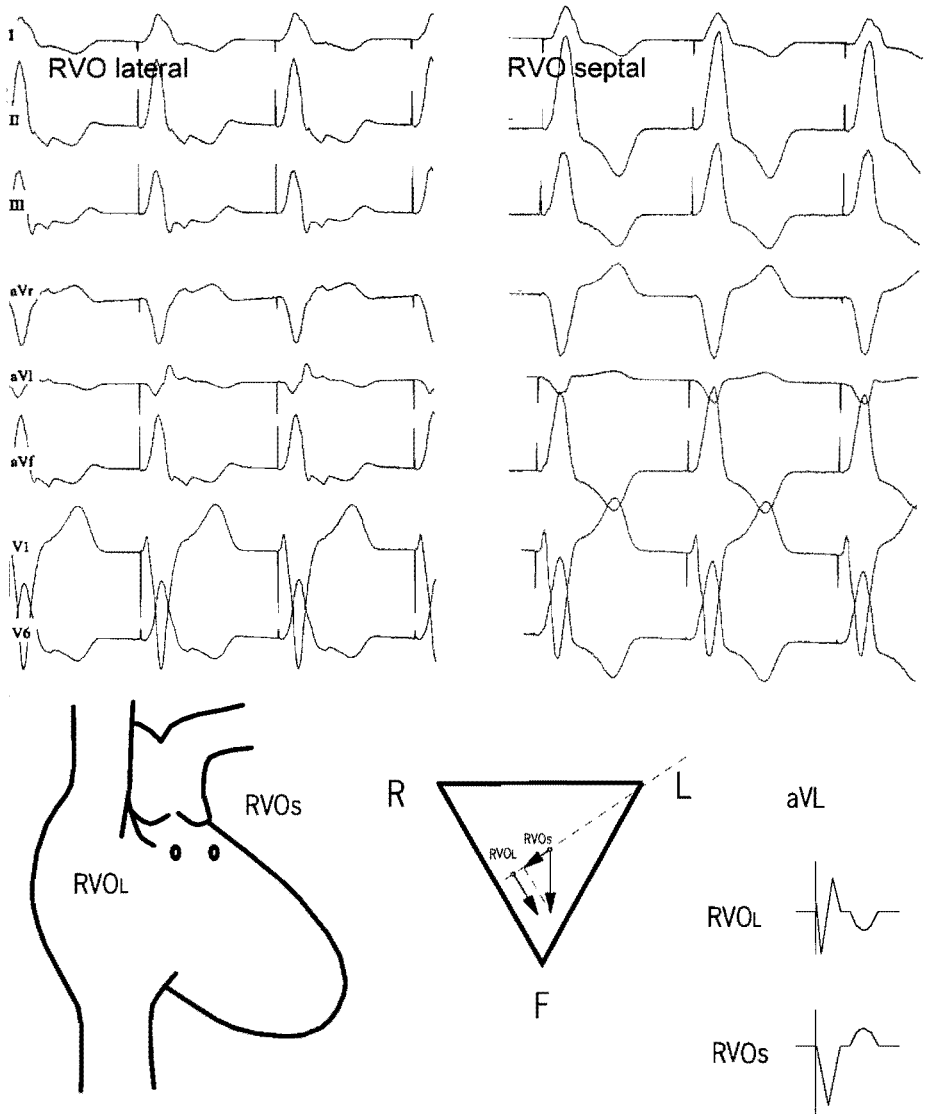
Fig. 2.9. Eight channel ECG comparing the morphology of the stimulated QRS complex during stimulation from the septal (RVOS) and lateral side (RVOL) of the right ventricular outflow tract. For both positions electrical activation is from superior to inferior, indicated by a positive QRS complex in leads II, III and aVf. Initial right ventricular activation is confirmed by the left bundle branch block pattern in lead V1 and the positive deflection of the QRS complex in lead I indicates right to left activation. Pacing from the lateral wall of the right ventricular outflow tract can be best discrminated from pacing from the septal site by lead aVL. The latter is illustrated in the schematic presentation.

Pacing from the lateral wall of the right ventricular outflow tract results in a biphasic ventricular depolarization in which the positive and negative portions are equal in amplitude, whereas stimulation from the septal site results in a negative deflection.

A pattern of activation similar to stimulation from the lateral wall of the right ventricular outflow tract can be observed during epicardial pacing from a stimulation electrode sutured on or screwed into the right ventricular outflow tract during cardiac surgery (Fig. 2.10).
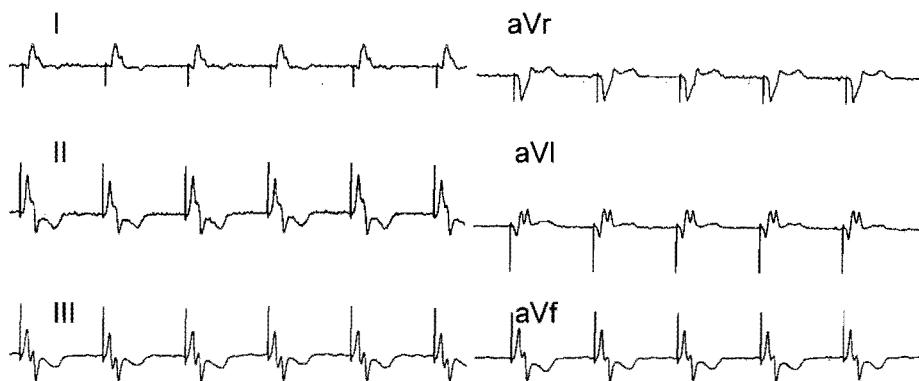
Fig. 2.10. Six lead ECG of a patient with an epicardial electrode attached to the right ventricular outflow tract. The morphology of the QRS complex is similar to endocardial stimulation from the lateral side of the right ventricular outflow tract (see also fig 2.9). The polarity and amplitude of the unipolar pacemaker stimulus indicate a right abdominal implantation of the pulse generator, which is common position in combination with epicardial electrodes.

2.3 QRS complex with right bundle branch block configuration during transvenous ventricular stimulation

The expected morphology of the QRS complex during transvenous ventricular stimulation with an electrode positioned in the apical position is a left bundle branch block pattern. However, a right bundle branch block pattern can be observed after implantation of pacemaker electrodes. This right bundle branch block pattern, which indicates that the left ventricle is stimulated first, can be observed after incorrect placement of the stimulation electrode. However, it is unusual to observe this phenomenon after correct placement of the stimulation electrode in the right ventricular apex.

2.3.1 Inadvertent left ventricular stimulation

The most common cause of inadvertent left ventricular stimulation during transvenous pacing is malplacement of the stimulation electrode in the coronary sinus or middle cardiac vein [19,20,21]. If ventricular stimulation results in a right bundle block pattern after lead implantation, a malposition of the electrode should be excluded by lateral fluoroscopy (Fig. 2.11).

Perforation of intraventricular septum and migration of the electrode into the left ventricle has been reported as a cause of inadvertent left ventricular stimulation [22]. Left ventricular stimulation can also be caused by perforation of the right ventricular apex by the stimulation electrode, which migrates through the pericardium towards the left ventricular epicardium [23].

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This phenomenon always includes an elevated stimulation threshold often associated with no capture. Inadvertent pacing of the left ventricle has been described in a patient with an atrial septal

implanted pacemaker   external pacemaker
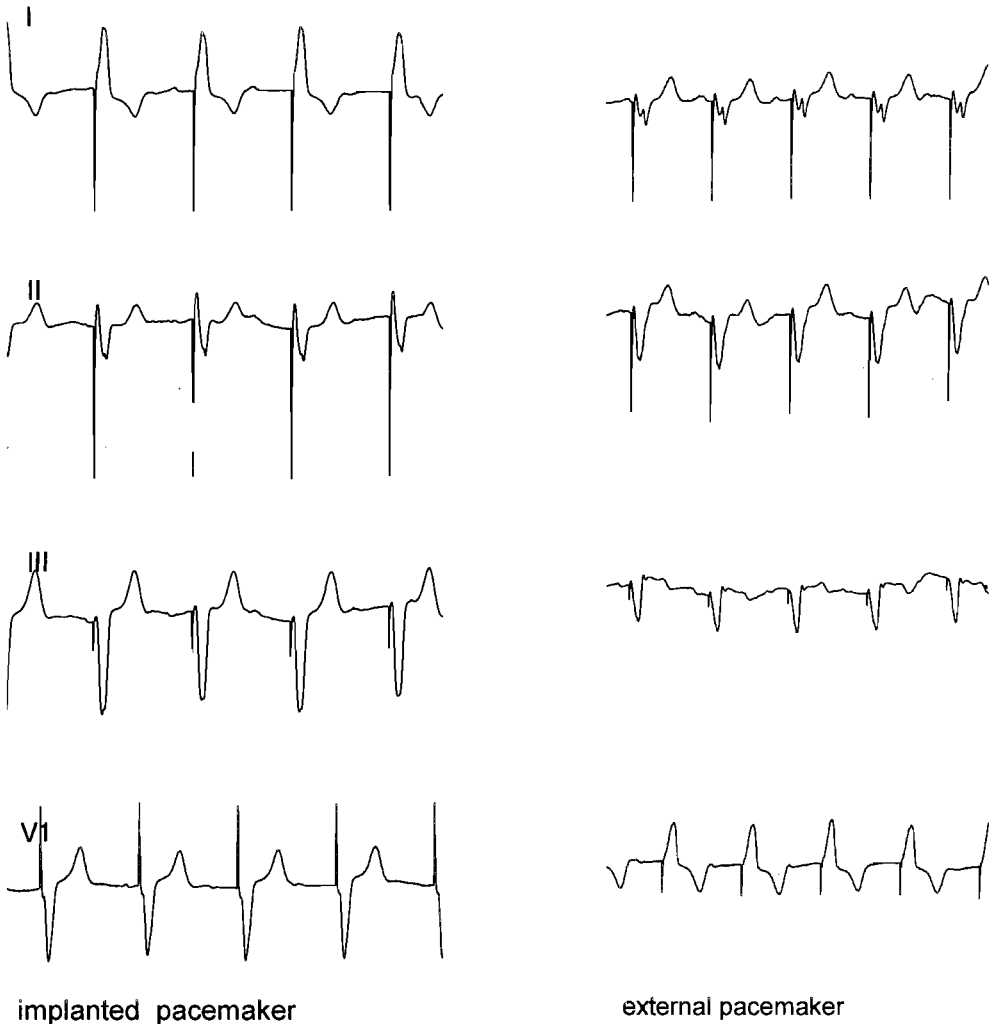
Fig. 2.11. Four channel recording (lead I,II,II,V1, not simultaneously) from a patient with an implanted and an external pacemaker. The stimulation electrode connected with the external pacemaker was inadvertently placed in the middle cardiac vein resulting in left ventricular stimulation, indicated by a right bundle branch block pattern. The electrode for the implanted pacemaker was correctly positioned in the right ventricular apex resulting in a left bundle branch block pattern of the stimulated QRS complex.

defect [24] or a patent foramen ovale, in whom the stimulation electrode passed accidentally through the atrial septum into the left ventricle.

If right bundle block pattern during ventricular pacing after implantation of a transvenous electrode is observed, the causes mentioned above should be excluded by radiological or echocardiographical techniques [25].
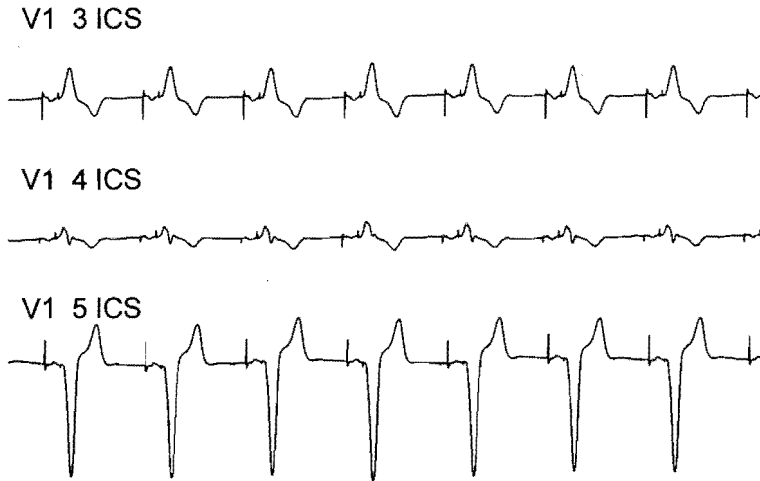
Fig. 2.12. Three channel ECG of a patient with an implanted DDDR pacemaker. The middle tracing shows standard lead V1 in the fourth intercostal space (4ICS), the upper tracing lead V1 in the third intercostal space (3 ICS), and the lower tracing lead V1 in the fifth intercostal space (5 ICS). There is a small R wave in standard lead V1 indicating right bundle branch block pattern but the QS pattern in V1 (5 ICS) indicates right ventricular stimulation. Note that the morphology of lead V1 in the third ICS shows a right bundle branch block pattern.

2.3.2 Right bundle branch block configuration with a correctly placed stimulation electrode in the right ventricular apex

In spite of the theory that stimulation of the right ventricle will result in a QRS complex with a left bundle branch block configuration, several reports describe the phenomenon of a right bundle branch block pattern with a correctly positioned electrode within the right ventricle [26,27,28,29,30].

Several explanations have been offered for this phenomenon. Mower et al. [31] suggested that the pacemaker stimulus enters the right bundle branch and is conducted retrogradely to the atrioventricular node. From there, the left bundle branch is activated followed by initial depolarization of the left ventricle with resulting right bundle branch configuration of the QRS complex.

An other explanation has been proposed by Sodi-Pallares and Calder [32] who suggested the presence of areas in the right ventricular septum that were electrically and functionally left ventricle. If these areas are stimulated by the pacing electrode, the left ventricle is first depolarized. A similar hypothesis is the presence of rapidly conducting Purkinje bridges between the right and left bundle branches.

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Barold et al. [33] had a different explanation. They stated that a right bundle branch block configuration of the QRS complex occurring during right ventricular stimulation appears to represent a right ventricular activation delay due to disease of the conduction system of the ventricle. Also the combination of the last explanation with one of the previously mentioned causes might be considered as the genesis of this phenomenon, but the true genesis is still speculative even in recently published articles on this subject [30].
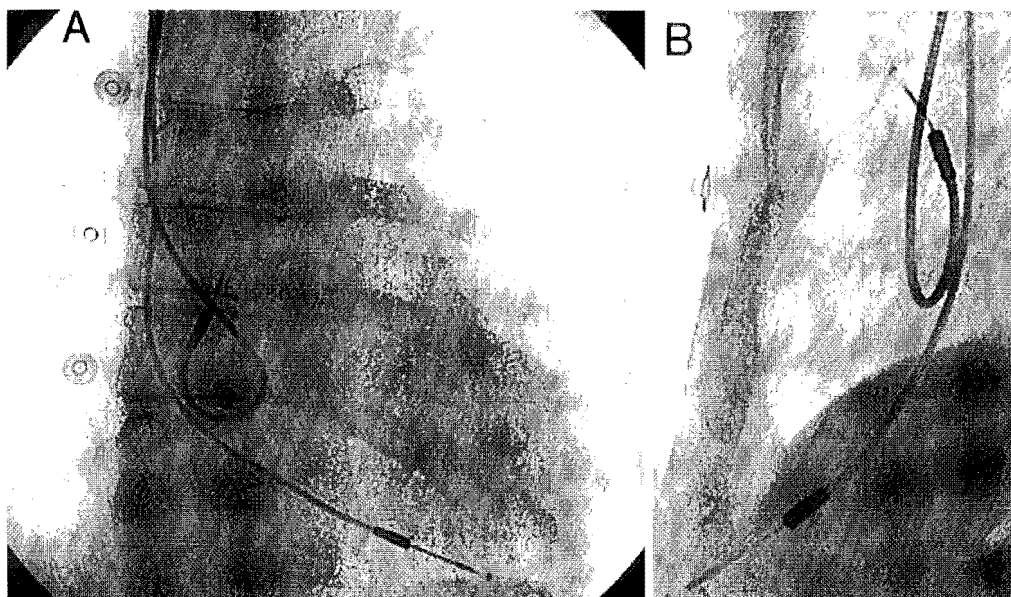
Fig. 2.13. Chest X-ray of the same patient as figure 2.12, showing the position of the atrial and ventricular lead and the position of the ECG electrodes in the third, fourth and fifth intercostal space corresponding with the tracing of figure 2.12. The lateral view confirms the right ventricular apical position of the ventricular electrode. There is an unusual curve in the atrial lead, but the atrial electrode is properly positioned in the right atrial appendage.

If a positive R wave (indicating a right bundle branch block pattern) of a relatively small amplitude is recorded in V1, the stimulation electrode is in all likelihood in the right ventricle, if V1 is negative when recorded at the level of the fifth intercostal space.

On the other hand, a dominant positive R wave may be observed in lead V1 when recorded at the level of the third intercostal space, so careful checking of the ECG electrode position should be done before conclusions about the type of bundle branch configuration can be drawn (Figs. 2.12, 2.13).

It is important to recognize that a right bundle branch block pattern can be observed with uncomplicated right ventricular apical stimulation, without the necessity of replacing or repositioning a normally functioning lead, but other pathological causes as mentioned in paragraph 2.3.1 should be excluded first.

2.4 Changes in morphology of the paced QRS complex related to pacemaker output.

L.M. van Gelder, M.I.H. El Gamal, C.H.J. Tielen.

Summary
The influence of pacemaker output on the morphology of the paced QRS complex was studied from standard lead electrocardiograms in 69 patients with bipolar pacemakers. In 40 of the 69 patients (58%), there was a significant ($P<0.001$) change in electrical axis, from -75 degrees at the low output setting (2.7 V, 0.15 msec) to - 67 degrees at the high output setting (8.1 V, 2.29 msec). In 30 patients, these changes were also associated with changes in the QRS morphology and in the T-wave. This phenomenon may be explained by additional stimulation from the proximal electrode at high output, thus altering the pattern of depolarization. (PACE, Vol. 12, October 1989)

Introduction
Changes in morphology of paced QRS complexes are rarely reported in the literature, suggesting a low incidence of this phenomenon [1,2].
After the implantation of a bipolar lead, we observed a morphological change in the QRS complex during threshold measurement. Subsequently, we systematically recorded standard ECG leads at pacemaker follow-up in all patients with bipolar pacing systems, with the pacemaker output programmed to its lowest and its highest values.
This article describes our observations in 69 patients with bipolar pacing systems. A high incidence of changes appear unrelated to changes in the position of the electrode, but seem dependent on the output of the pulse generator.

Patients and methods
Standard lead electrocardiograms of 69 consecutive patients (39 men and 30 women) ranging in age from 53 to 91 years (mean 76.8 years) were studied during outpatient pacemaker follow-up.
All patients had transvenous bipolar pacing systems implanted for more than 3 months. The pulse generator was a Quantum model 254-10 bipolar unit (Intermedics, Inc., Freeport, TX, USA) and the lead was an Intermedics model 484-02 in-line bipolar lead. The distance between the proximal and distal electrodes is 25 mm. Surface of the proximal and distal electrodes are 50 mm2 and 10 mm2 respectively.
All leads were implanted in the right ventricular apex. During pacemaker follow-up the pacemaker was programmed to a rate above patients' intrinsic rate, in order to maintain continuous ventricular pacing. Subsequently, the pacemaker was programmed to its lowest output (2.7V pulse amplitude, 0.15 msec pulse width). Ventricular capture was maintained in all patients at this output setting.
Standard lead electrocardiograms (I, II, III, aVR, aVL, aVF) were recorded for at least 20 seconds using a three-channel electrocardiographic recorder (Gould 2400, Gould, Inc., Cleveland, OH, USA).
After this, the pulse generator was programmed to its highest output (8.1V pulse amplitude, 2.29 msec pulse width) and the electrocardiogram was recorded again.

We studied the differences in ventricular depolarization, repolarization, and frontal electrical axis recorded at the two output settings. Clinical and electrocardiographic profiles of the patients showing no changes in the morphology of the paced QRS complex are summarized in Table I and those with changes in Table II. Profile of the paced electrocardiogram is defined by the amplitude of the QRS complex in lead I and the frontal electrical axis. Changes in electrical axis and QRS amplitude were statistically analyzed using the Student's t-test for differences with paired samples. Associated T-wave changes and their significance were statistically analyzed using Student's t-test for two samples.

Table I.

Clinical, Electrocardiographical and Radiological Profiles. (Group A)

No	Sex	Age	El. Ax.	Ampl. I	Dist.	El/Hb	Rhythm dist.
01	M	67	−56	1.10	5.0	58%	SSS-BT, I AVB
02	M	82	−82	0.25	—	—	SB
03	M	62	−74	0.40	1.5	69%	RBBB-LAH
04	F	68	−40	1.00	1.5	62%	AF-SVR
05	M	74	−75	0.40	2.0	78%	AF-SVR, RBBB
06	M	78	−55	1.20	5.0	42%	III AVB
07	M	53	−48	0.70	1.0	46%	SB
08	M	83	−75	0.45	—	—	III AVB
09	M	75	−48	0.70	—	—	AF, I AVB
10	M	71	−60	0.80	1.5	54%	AF-SVR
11	M	78	−58	0.50	1.5	65%	AF-SVR
12	M	72	−70	0.40	2.5	65%	III AVB
13	M	74	−51	1.10	6.0	35%	III AVB
14	F	85	−60	1.15	2.0	63%	SB, I AVB
15	F	74	−58	0.90	—	—	AF-SVR
16	F	74	−40	1.30	—	—	III AVB
17	F	69	−81	0.20	2.5	75%	SB
18	F	82	−52	1.40	3.0	53%	III AVB
19	F	85	−77	0.30	—	—	AF-SVR
20	F	79	−33	1.40	—	—	SSS-BT, I AVB
21	F	82	−52	1.40	4.0	—	III AVB
22	F	72	−54	1.30	3.0	50%	SSS-BT
23	F	83	−49	1.40	—	—	III AVB
24	F	72	−65	1.30	4.5	47%	SSS-BT
25	F	80	−56	1.10	4.0	59%	RBBB-LAH, III AVB
26	F	75	−51	0.80	—	—	AF-SVR
27	F	70	−35	1.30	1.0	60%	AF-SVR
28	F	85	−75	0.60	—	—	RBBB-LAH
29	F	79	−45	0.90	1.5	63%	III AVB

Abbreviations: El. Ax. = Electrical axis. Ampl. I = Amplitude lead I (mV). Dist. = distance (in cm between electrode tip and right ventricular apex, in lateral view). El/Hb = ratio electrode position, heartbase in frontal view (for explanation see text). Rhythm dist. = rhythm disturbance. SSS-BT = sick sinus syndrome, brady tachy. I AVB = first degree AV block. SB = sinus bradycardia. RBBB = right bundle branch block. LAH = left anterior hemiblock. AF-SVR = atrial fibrillation, slow ventricular response. III AVB = complete heat block.

Electrode position was retrospectively reviewed in all patients who had frontal and lateral X rays after pacemaker implantation.In frontal projection the position was defined as the distance between the electrode tip and the right border of the heart expressed as a percentage of the transverse diameter of the heart.This implies that the higher the percentage, the more lateral the position of the electrode tip in the frontal view. Results are found in Tables I and II.

Results

In 29 of the 69 patients, (42%; 14 males, 15 females), the morphology of ventricular depolarization and repolarization was not affected by the output setting of the pacing system (group A; Table I). In group A, patients with no change in morphology, the electrocardiogram showed a left bundle branch block with electrical axis varying from -33 degrees to - 82 degrees, a mean of -57.75 degrees, standard deviation of 13.935 and a standard error of 2.634. In the same group, the amplitude of the R wave in lead I varied from 0.2 to 1.4mV with a mean of 0.89 mV, a standard deviation of 0.408 and a standard error of 0.077.
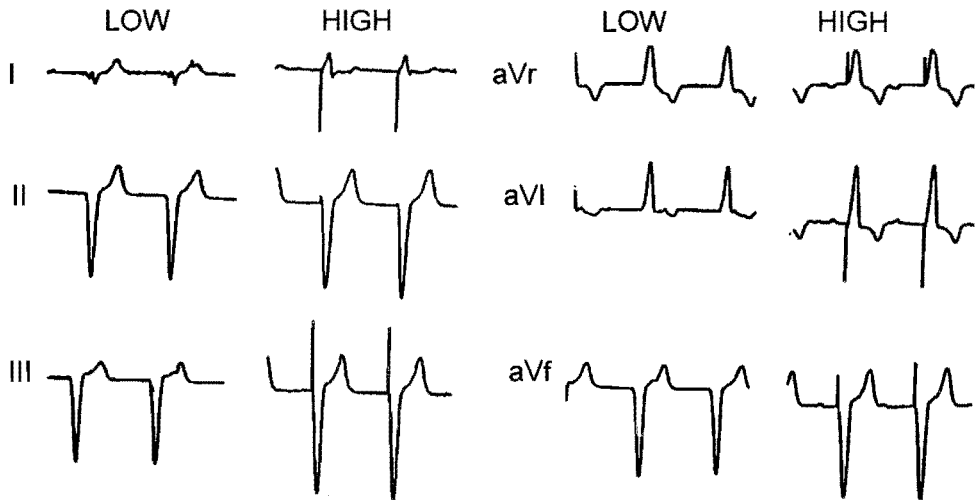
Fig. 2.14. Standard lead ECG at low (L) and high (H) pacemaker output setting. Note the significant change in frontal axis from right superior to left superior quadrant.

In 36 patients (52%; 24 males, 12 females), there was a consistent and significant difference in the morphology of the paced QRS complex at the different output settings (group B; Table II).
Only 4 patients (6%; 1 male, 3 females) showed intermittent change in morphology of the QRS complex at the high output setting (group C; Table II).
When the pulse generator was programmed to low output, all group B patients except one showed left bundle branch block (LBBB) and electrical axis in the left superior quadrant. Only one patient showed LBBB with electrical axis in the right superior quadrant. The electrical axis varied from -54 degrees to -109 degrees with a mean of -74.72 degrees, standard deviation of 9.41 and mean standard error 1.57. The amplitude of QRS complex in lead I varied from 0.2 to 1.2 mV, with a mean of 0.48 mV, standard deviation of 0.31 and mean standard error 0.05. After programming the pulse generator to high output, the LBBB pattern was maintained, but there

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was a significant shift in frontal axis. The frontal axis at this setting varied from -51 degrees to -80 degrees with a mean of -66.78 degrees, standard deviation of 8.35, mean standard error 1.392. The patient with electrical axis in the right superior quadrant at low output showed a shift in axis to the left superior quadrant at high output (Fig. 2.14).

The amplitude of the QRS complex in lead I varied from 0.2 to 1.6 mV with a mean of 0.726 mV, standard deviation of 0.311, mean standard error 0.05. An increase in amplitude in lead I was recorded in all patients after programming the pulse generator to high output (Fig. 2.15). Both the shift in electrical axis and increase in R wave amplitude in lead I were highly significant ($P<0.001$).
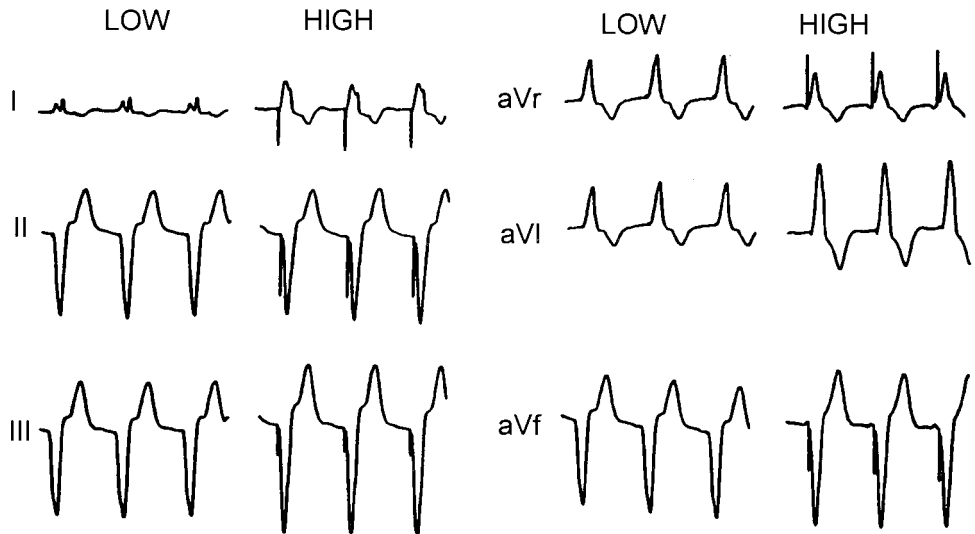
Fig. 2.15. Standard lead ECG at low (L) and high (H) output setting showing characteristic change in morphology of the paced QRS complex.

In 32 of the 40 group B and C patients (80%), changes in morphology of the QRS complex were also associated with T-wave changes. The T-wave changes were most clearly reflected in lead I of the electrocardiogram and could be divided into four types (Fig. 2.16):

1. Negative T-wave at low output, increase in T-wave amplitude at high output (15 patients; 47%)
2. Biphasic T-wave at low output, negative T-wave at high output(12 patients; 38%)
3. Positive T-wave at low output, negative T-wave at high output (4 patients; 12%)
4. Positive T-wave (electrical axis right superior quadrant) at low output, decrease in T-wave amplitude with electrical axis changing to the left superior quadrant at high output (1 patient; 3%).

Although there were four types of T-wave changes, a negative component added to the T-wave was common to all when the pulse generator was programmed to high output.

Table II.

Clinical, Electrocardiographical and Radiological Profiles (Group B, C)

No	Sex	Age	E.A. low	E.A. high	Twave	Ampl. I low	Ampl. I high	Dist.	El/Hb	Rhythm dist.
01	F	79	−67	−56	I	0.75	1.05	2.5	73%	III AVB
02	M	75	−78	−73	II	0.45	0.70	6.0	67%	AF-SVR
03	M	79	−84	−76	0	0.35	0.75	2.0	73%	I AVB, RBBB
04	M	82	−71	−59	I	0.70	1.00	2.0	63%	III AVB
05	M	74	−84	−80	I	0.15	0.30	5.0	74%	SSS-BT
06	M	75	−81	−78	I	0.35	0.50	2.0	73%	SSS-BT
07	M	82	−73	−61	II	0.55	0.90	1.5	69%	II AVB, RBBB-LAH
08	M	68	−82	−70	III	0.20	0.45	2.5	70%	AF-SVR
09	F	77	−82	−79	I	0.30	0.55	2.5	75%	RBBB
10	M	81	−54	−51	I	0.60	0.80	2.5	71%	SSS-BT, RBBB
11	F	75	−78	−69	II	0.15	0.80	3.0	69%	I AVB, LBBB
12	M	73	−75	−64	II	0.40	0.60	1.0	79%	AF-SVR
13	M	91	−72	−70	III	0.60	0.80	1.0	73%	II AVB, RBBB-LAH
14	M	55	−78	−75	I	0.35	0.45	2.0	78%	AF-SVR
15	M	86	−55	−52	I	0.40	0.50	—	—	III AVB
16	M	64	−66	−58	II	0.25	0.35	1.0	54%	AF-SVR
17	F	82	−69	−62	I	0.50	0.80	1.5	81%	AF-SVR
18	M	80	−74	−64	I	0.50	0.60	2.0	71%	II-III AVB
19	M	60	−82	−78	I	0.35	0.55	—	—	III AVB
20	M	82	−81	−71	II	0.20	0.50	1.5	47%	RBBB-LAH
21	M	72	−73	−55	III	0.55	0.90	3.0	73%	AF-SVR
22	M	56	−80	−78	I	0.60	0.80	2.5	72%	SSS
23	F	77	−75	−72	II	0.45	0.55	2.0	69%	SSS
24	F	81	−78	−62	II	0.40	0.90	4.0	70%	SSS-BT, RBBB
25	F	85	−69	−62	II	1.00	1.30	7.5	63%	III AVB
26	F	88	−69	−62	I	1.20	1.60	—	—	III AVB
27	M	83	−81	−57	II	0.10	0.40	—	—	II AVB, RBBB
28	F	80	−71	−63	I	0.40	0.55	2.0	64%	II AVB
29	F	85	−74	−71	0	0.50	0.70	—	78%	SSS-BT
30	M	74	−71	−68	0	1.05	1.20	—	—	III AVB
31	M	82	−73	−70	0	0.10	0.40	2.0	86%	III AVB
32	F	79	−63	−60	0	1.20	1.20	1.0	57%	AF-SVR
33	F	85	−64	−61	0	1.00	1.10	—	—	AF-SVR
34	M	78	−109	−79	IV	−0.20	0.20	—	—	AF-SVR
35	M	79	−81	−76	II	0.30	0.45	—	—	SSS
36	F	75	−73	−62	0	0.65	0.95	—	—	II AVB
37	F	88	−73	−67	II	0.35	0.60	3.0	75%	SSS, RBBB
38	F	81	−69	−65	I	0.70	0.90	1.5	68%	III AVB
39	F	63	−78	−73	0	0.20	0.30	—	—	AF-SVR
40	M	73	−83	−73	III	0.30	0.65	1.5	66%	III AVB

Abbreviations: E.A. low = electrical axis at low output. E.A. high = electrical axis at high output. T-wave 0 = T-wave not changed. T-wave I = T-wave change type I. T-wave II = T-wave change type II. T-wave III = T-wave change type III. T-wave IV = T-wave change type IV. Amp. I, low = amplitude lead I at low output. Ampl. I, high = amplitude lead I at high output. (mV) Dist. = distance (in cm between electrode tip and right ventricular apex in lateral view). El/Hb = ratio electrode position, heart base in frontal view (for explanation see text). Rhythm dist. = rhythm disturbance. III AVB = complete heart block. AF-SVR = atrial fibrillation, slow ventricular response. I AVB = first degree AV block. RBBB = right bundle branch block. SSS-BT = sick sinus syndrome, brady tachy. LAH = left anterior hemiblock. II AVB = second degree AV block. LBBB = left bundle branch block.

In the lateral X ray, the mean distance between the electrode tip and the right ventricular apex was 2.79 cm, varying between 1.0 and 6.0 cm in group A. Standard deviation 1.53, standard error 0.35.

In groups B and C, the distance was 2.47 cm, varying between 1.0 and 7.5 cm, standard deviation 1.49, standard error 0.28. Although there is a difference in distance between both groups, this is not statistically significant. The position of the electrode in the frontal projection was 58% in group

A, standard deviation 11.2, standard error 2.64. In groups B and C the position was 70%, standard deviation 7.93, standard error 1.45. The difference in position in the frontal view is highly significant between both groups (P<0.001).
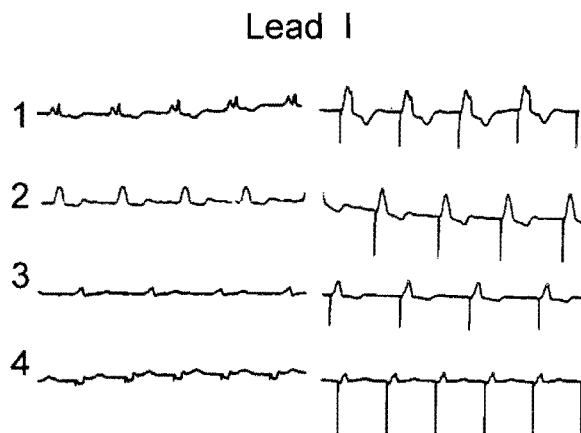
Lead I

Fig. 2.16. Four types of T wave changes elicited after changing the pacemaker output from low to high. All recordings represent lead I.

Discussion

The configuration of the QRS complex during ventricular pacing depends on the site of stimulation. Right ventricualr pacing from the apex produces left bundle branch block in the majority of cases, while the mean electrical axis is deviated superiorly and generally to the left, because the sequence of activation travels from apex to base. Occasionally, the mean electrical axis may be in the right superior quadrant and cause a predominantly negative deflection in lead I, as well in leads II, III and aVF. Lead aVR will register a dominant R wave [3]. Changes in the morphology of the paced QRS complex, excluding fusion and pseudofusion beats, may be due to respiration. However, this could clearly be excluded in our patients. Another situation in which morphological change can occur is secondary to mechanical changes, particularly in the presence of a mobile electrode. In our patients, mobility of the electrode was very unlikely because all leads had been implanted more than 3 months. Further reports on this phenomenon are rare, and the mechanism is still speculative [1].

Although 42% of our study patients showed no difference in the paced QRS complex, significant changes were observed in the remaining 58%. Analysis of these changes indicated that the direction of ventricular depolarization changed to a more horizontal orientation, indicated by the increase amplitude of the QRS complex in lead I. This phenomenon was observed in 40 patients and is highly significant. In our view the explanation of this phenomenon is as follows: at the low

output setting, the ventricular depolarization originates from the distal electrode (cathodal stimulation) and the electrocardiogram recorded corresponds to the typical pattern for right ventricular apical stimulation, i.e., left bundle branch block and electrical axis -75 degrees. At high output setting, however, ventricular depolarization is not only emanating from the distal electrode but from the proximal electrode as well, due to the anodal effect of the pacemaker stimulus [4]. The effectiveness of the anodal impulse is mainly dependent on the distance and/or contact area of the proximal electrode to the right ventricular endocardium. This may explain why the phenomenon was not present in all patients. If the right ventricle is stimulated simultaneously from a distal electrode at the apex as well as from a proximal electrode in the right ventricular outflow, a second component of depolarization (from RV inflow towards RV apex, in frontal view from the right to left) has to be added to the depolarization arising from the apical electrode. Thus, stimulation from the distal as well as from the proximal electrode increases the horizontal component of ventricular depolarization, with a resulting increase in the amplitude of the QRS complex in lead I and a shift in electrical axis to a more horizontal position (Fig. 2.17).

LOW OUTPUT   HIGH OUTPUT
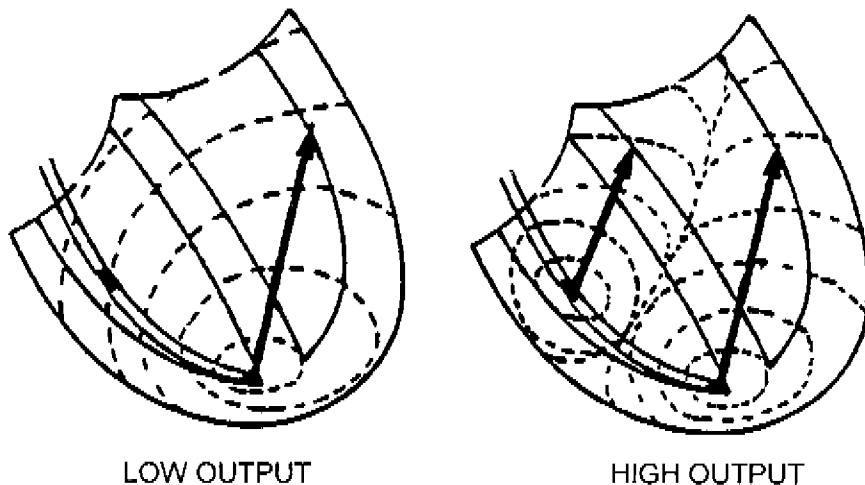
Fig. 2.17. Schematic presentation of the effects of bipolar stimulation at low and high output settings. At low output, depolarization starts from the tip electrode, while at high output depolarization starts from both the proximal and distal electrodes.

A pacing electrode in a deep apical position will facilitate contact of the proximal electrode with the right ventricular endocardium, while when a pacing lead is positioned just distal to the tricuspid valve, the proximal electrode might be in the tricuspid area or even outside the right ventricle. The latter position diminishes the possibility of anodal stimulation from the proximal electrode. This finding is supported by the radiological appearance of the pacing leads that showed more inflow oriented position in group A. Inflow position of the electrode tip in this group was also confirmed by the electrocardiographic findings of higher amplitude of the R

wave in lead I and a more horizontal orientation of the frontal axis in group A as compared to group B.

Oreto et al [2] described a change of morphology of the paced QRS complex related to atrial contraction. They explained this change by the displacement of the lead tip caused by atrial contraction. In our study however, its seems more likely that atrial contraction affected the position of the proximal electrode, causing intermittent anodal stimulation from this site. Study of the T-wave changes showed that at high output negative component in lead I was added to the repolarization wave as compared to pattern at low output. In LBBB, the direction of depolarization (in lead I) is opposite to the direction of repolarization. A change in depolarization to a more horizontal direction will produce a change in repolarization in the same direction. In other words, an increase in amplitude of the QRS complex will result in an increase in amplitude of the T-wave. It can also be assumed that there is a proportional relationship between change in electrical axis and changes in T-waves. This hypothesis is supported by the fact that the eight patients who did not show T-wave changes, the changes in electrical axis were smaller than the remaining 32 patients (4.9 degrees vs 8.5 degrees; P<0.06).

Lead I

2.7 V

5.4 V

8.1 V
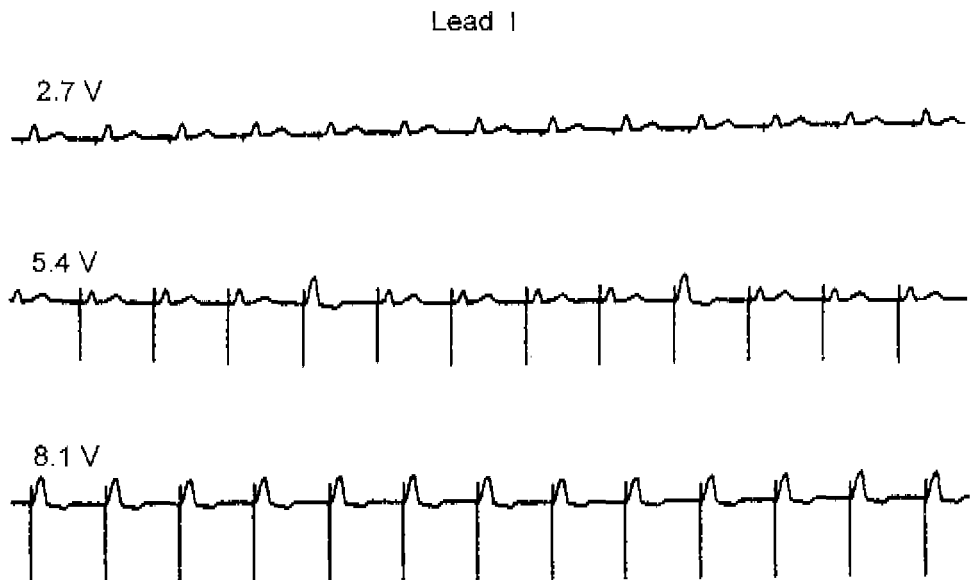
Fig. 2.18. Lead I ECG at 2.7, 5.4 and 8.1 V respectively showing a morphological change intermittently at 5.4 V (probably respiration dependent) and consistently at 8.1 V.

Although increasing the voltage of the pulse generator does not significantly deform the QRS complex in bipolar systems, there might be some influence of the amplitude of the pacemaker artifact on the QRS complex. If this is true, there should be a linear relationship between the amplitude of the pacemaker impulse and the amplitude of the QRS complex. Figure 2.18, however, demonstrates that changes in morphology are abrupt; it is an all-or-nothing phenomenon without a linear relationship. At an amplitude of 5.4V (constant amplitude of the pacemaker impulse), two morphologies of QRS complexes were recorded, thus excluding the

influence of the pacemaker artifact. The intermittent change in morphology in this recording is most probably related to respiration. Respiration may affect the distance or contact area of the proximal electrode to the right ventricular endocardium, thus exceeding the anodal stimulation threshold at the proximal electrode under these circumstances. To exclude that high energy cathodal stimulation was responsible for the electrocardiographic changes, we did the following experiment during implantation of a bipolar pacing lead. After implantation of the lead in the right ventricular apex we stimulated bipolar at high and low output settings recorded the electrocardiogram. After conversion of the system to unipolar, using the same distal electrode as cathode and a subcutaneous electrode as anode, the recording was repeated at the same low and high output (Fig. 2.19). Analysis of the electrocardiograms showed that in the unipolar configuration, there was no difference in the morphology of the QRS complex at high and low output, while there was a clear
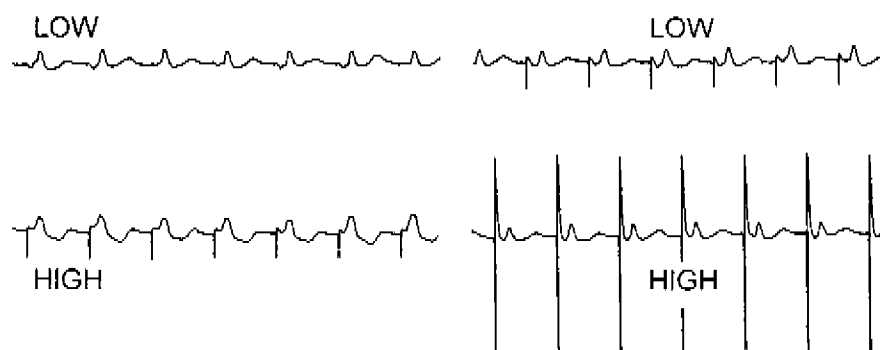
Fig. 2.19. Lead I ECG at high and low output as well as unipolar and bipolar configuration. There is no difference in morphology at low and high output in the unipolar configuration.

difference in the bipolar configuration. We conclude that the changes in the morphology are related to the bipolar configuration and associated anodal stimulation and not to high energy cathodal stimulation.

Conclusion
Our study indicates that there is a high incidence (58%) of change in morphology of the paced QRS complex, frequently associated with T-wave changes (80%) in patients with bipolar pacemakers, depending on the output of the pulse generator. At high output settings, ventricular depolarization can be initiated at the distal electrode (cathodal stimulation). High output settings should be avoided if the stimulation threshold permits, because of the higher risk of arrhythmia induction by anodal stimulation [5]. At low output settings there was no fundamental difference between unipolar and bipolar stimulation.

References.
1. *Kleinfeld M, Barold SS, Rozanski JJ. Pacemaker alternans: A review. PACE 1987; 10:924-933.*
2. *Oreto G, Satullo G, Luzza F, et al. Changes in morphology of the paced QRS Complex related to atrial contraction. PACE 1987;10:100-102.*

3. Barold SS, Falkoff MD, Ong LS, et al. Electrocardiographic analysis of normal and abnormal pacemaker function. In Dreifus LS (ed): Pacemaker Therapy.Philadelphia, F.A. Davis Co., 1983: 97-134.
4. Van Dam RTh, Durrer D, Strackee J, et al. The excitability cycle of dog's left ventricle determined by anodal, cathodal and bipolar stimulation. Circ Res 1956; 4:196-204.
5. Preston TA. Anodal stimulation as a cause of pacemaker induced ventricular fibrillation. Am Heart J 1973; 86:366-372.

2.5 Summary and conclusions

The evaluation of the ECG of the pacemaker patient during implantation and follow up is generally limited to the effectiveness of stimulation and adequate sensing of the pacemaker system. However, the morphology of the stimulated depolarization is related to the location of the stimulation electrode and in bipolar systems it can be related to the pacemaker output as well. Application of this rule to the pacemaker ECG, means that the site of stimulation can be deduced from the ECG. The information obtained from the site of stimulation during atrial pacing is
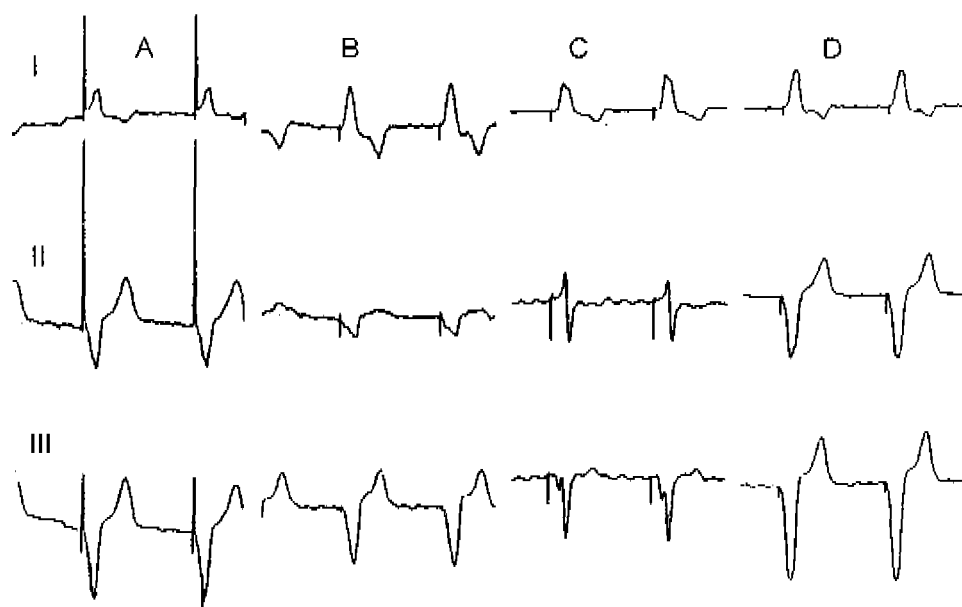
Fig. 2.20. ECG lead I,II,III recorded immediately after implantation of a VVI pacemaker (A). Two days later (B) after programming the pulse generator to the bipolar configuration, which was not responsible for the change in QRS morphology, a change in electrical axis indicating movement of the stimulation electrode towards the right ventricular inflow was recorded. Two months later a further change in QRS morphology was observed indicating displacement of the stimulation electrode. A longer recording of the ECG also showed noncapture (Fig. 2.21) and chest X-ray confirmed displacement of the electrode towards the right ventricular inflow. Panel D showed the ECG after repositioning of the lead in the right ventricular apex. The mophology is now identical to the morphology in panel A after the initial implantation.

limited in unipolar systems, due to the fact that atrial depolarization is obscured by the large stimulus artifact and only a rough estimation of the site of stimulation can be made from those ECG leads, in which the stimulus artifact is relatively small. For ventricular. pacing, determination of the electrical axis and thus the position of the stimulation electrode is relatively easy, even in unipolar systems. During implantation of ventricular leads monitoring of ECG lead V1 is advisable, because a right bundle branch block pattern is best recognized in this lead. If a right bundle branch block pattern during ventricular stimulation is observed after implantation of a ventricular lead, the lead position should be verified by lateral screening to exclude positioning of the lead in the coronary sinus and/or one of its tributaries (Fig. 2.11). A right bundle branch block pattern during ventricular stimulation with normal pacing and sensing thresholds and exclusion of coronary sinus or left ventricular malplacement, is no indication for lead repositioning. The correct position of the ECG lead V1 electrode should be verified.

Lead II

Fig. 2.21. Single channel ECG (lead II) same patient as figure 2.20, panel C. Effective ventricular stimulation followed by ineffective pacemaker stimuli are recorded, whereas ventricular sensing is maintained.
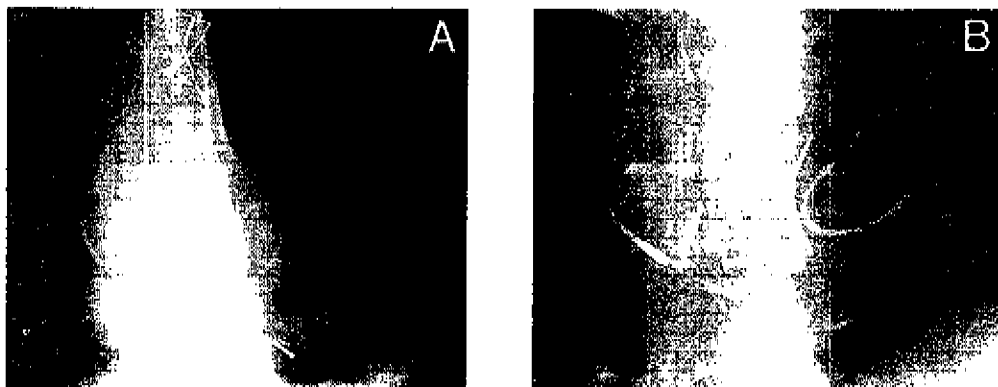
Fig. 2.22. Anterior posterior chest X-ray showing a bipolar ventricular lead correctly positioned at the right ventricular apex (A). X-ray of the same patient two months after implantation showing displacement of the lead to the right ventricular inflow tract (B). X-ray corresponds with figure 2.20 panel A and figure 2.20 panel C.

During pacemaker follow up, careful appraisal of the ventricular depolarization during ventricular stimulation should reveal a constant pattern from one examination to the next (Figs. 2.20, 2.21), provided no myocardial infarction has occurred in between. A change in depolarization pattern suggests dislocation of the stimulation electrode, even in the presence of normal pacing and sensing thresholds. The position of the stimulation electrode should be verified by chest X-ray (Fig. 2.22).

The interpreter, however, should be cautious with this conclusion in bipolar pacing systems. In bipolar pacing systems abrupt changes in the morphology of the stimulated QRS complex can be observed, that are not an indication for electrode dislocation. As explained in paragraph 2.4, the anodal electrode might play an (intermittent) active role in the onset of depolarization, thus giving the electrocardiographic pattern of changing morphology indicating a shift of the stimulation electrode towards the right ventricular inflow, without lead dislocation.

References

1. Meyer AJ, Millar K. Malplacement of pacemaker catheters in the coronary sinus. J Thorac Cardiovasc Surg 1969; 57: 511-517.

2. Sutton R, Bourgeois I. The Foundations of Cardiac Pacing. Part I. Mount Kisco, NY, Futura Publishing Company, 1991, pp. 275-276.

3. Kerr CR, Mason MA, Tyers GFO, et al. Transvenous atrial pacing following amputation of the right atrial appendage at open heart surgery. PACE 1985; 8: 497-501.

4. El Gamal MIH, van Gelder LM. Implantation of tined atrial J-leads after open heart surgery. In FP Gómez (Ed).Cardiac Pacing. Electrophysiology. Tachyarrhythmias. Madrid, Editorial Grouz, 1985 pp.649-653.

5. Hardebeck CJ. Electrocardiographic characteristics of pacing from the right atrial appendage during atrioventricular sequential pacing. PACE 1988; 11: 193-202.

6. Holmes DR, Hayes DL, Furman S. A Practice of Cardiac Pacing. Mount Kisco, NY, Futura Publishing Company, Inc, 1989, p.247.

7. Gillette PC, Wampler DG, Shannon C, et al. Use of atrial pacing in a young population. PACE 1985; 8: 94-100.

8. Stirbys P. Permanent interatrial septal pacing: Feasibility and advantages. PACE 1986; 9: 209-211.

9. Stirbys PP. An efficient technique for implantation of atrial leads from the left side. PACE 1991; 14: 980-981.

10. Josephson ME, Seides SF. Clinical Cardiac Electrophysiology. Techniques and Interpretations. Philadephia, Lea & Febiger, 1979, p. 12.

11. Moss AJ, Rivers RJ, Cupper M. Long term pervenous atrial pacing from the proximal portion of the coronary vein. JAMA 1969; 209: 543-545.

12. Greenberg P, Castellanet M, Messenger J. Coronary sinus pacing - clinical follow up. Circulation 1978; 57: 98-103.

13. Moss AJ, Rivers RJ. Atrial pacing from the coronary vein: 10 years experience in 50 patients with implanted pervenous pacemakers. Circulation 1978; 57: 103-106.

14. Waxman HL, Josephson ME. Ventricular activation during ventricular endocardial pacing: Electrocardiographic patterns related to the site of pacing. Am J Cardiol 1982; 50: 1-10.

15. Gulotta SJ. Transvenous cardiac pacing. Techniques for optimal electrode positioning and prevention of coronary sinus placement. Circulation 1970; XLII; 701-718.

16. Barold SS, Falkoff MD, Ong LS, et al. Normal and abnormal patterns of ventricular depolarization during cardiac pacing. In SS Barold (Ed). Modern Cardiac Pacing. Mount Kisko NY, Futura Publishing Company, 1985, pp 545-547.

17. El-Sherif N, Javier RP, Gann D, et al. Pacemaker electrocardiography. In P Samet and N El-Sherif (Eds); Cardiac Pacing. New York, Grune and Straton, 1980, 149-206.

18. Moss AJ, Adams FH. Heart Disease in Infants, Children and Adolescents. The Williams & Wilkins Company, Baltimore 1968.

19. Meyer JA, Millar K. Malplacement of pacemakers in the coronary sinus. J Thorac Cardiovasc Surg 1969; 57: 511-518.

20. Kemp A, Kjersgaard J, Kjoegaard E. Malplacement of endocardial pacemaker electrodes in the middle cardiac vein. Acta Med Scand 1976; 199: 7-12.

21. Barold SS, Banner R. *Unusual electrocardiographic pattern during transvenous pacing from the middle cardiac vein. PACE 1978; 1: 31-34.*

22. Stillman MT, Richards AM. *Perforation of the interventricular septum by transvenous pacemaker catheter. Am J Cardiol 1969; 24: 269-273.*

23. Meyer JA, Millar K. *Perforation of the right ventricle by electrode catheters. Ann Surg 1968; 168: 1048-1060.*

24. Huang WM, Xin ZP, Tung CL. *An unusual electrocardiographic pattern of left ventricular endocardial pacing. PACE 1980; 3: 597-599.*

25. Gondi B, Nanda NC. *Real time two dimensional echocardiographic features of pacemaker perforation. Circulation 1981; 64: 97-103.*

26. Bauman DJ, Lamb KC, Tsagaris TJ. *Unusual QRS wave forms associated with permanent pacemakers. Chest 1973; 64: 480-485.*

27. Abernathy WS, Crevey BJ. *Right bundle branch block during transvenous ventricular pacing. Am Heart J 1975; 90: 774-776.*

28. Ishikawa K, Yanagisawa A. *Evaluation of unusual QRS complexes produced by pacemaker stimuli, with special reference to the vectorcardiographic and echocardiographic findings. J Electrocardiol 1980; 13: 409-416.*

29. Saksena S. *Mechanism of an unusual QRS pattern associated with right ventricular pacing. Am Heart J 1983; 105: 337-339.*

30. Buys EM, Cramer MJ, van Hemel NM. *Ventricular pacing with unusual QRS pattern. Neth J Cardiol 1993; 6: 369-373.*

31. Mower MM, Aranaga CE, Tabatznik B. *Unusual patterns of conduction produced by pacemaker stimuli. Am Heart J 1967; 74: 24-28.*

32. Sodi-Pallares D, Calder RM. *New Basis of Electrocardiography. The C.V. Mosby Company, St.Louis 1956.*

33. Barold SS, Narula OS, Javier RP, et al. *Significance of right bundle branch block patterns during pervenous ventricular pacing. Br Heart J 1969; 31: 285-290.*

34. Barold SS, Falkoff MD, Ong LS, et al. *Normal and abnormal patterns of ventricular depolarization during cardiac pacing. In Barold SS (Ed). Modern Cardiac Pacing. Mount Kisco, New York, Futura Publishing Company, 1985, pp. 552-553.*

Chapter III. **Presentation of the Pacemaker Stimulus on the ECG**

Introduction
A large number of factors play a role in the presentation of the pacemaker stimulus in the ECG. The electrode configuration of the pacing system (unipolar or bipolar), the charge generated by the pacemaker impulse (amplitude and pulse width), the location of the anodal and cathodal electrode, the recorded ECG lead, lead impedance and the recording equipment determine the electrocardiographic presentation of the pacemaker stimulus. The conventional analogue electrocardiograph is designed to accept small signals (mVolt level) and because of the filtering characteristics, high amplitude pacemaker stimuli are often attenuated. Pacemaker stimuli from unipolar pacemaker systems may have amplitudes (measured between two ECG electrodes) varying between 2 to 100 mV. This implies that a quantitative analysis cannot be made from the conventional ECG but should be made by oscilloscopic analysis if necessary [1].
Although a quantitative analysis cannot be performed from ECG recordings, the pacemaker stimulus, even on ECGs from unipolar systems, contains qualitative information about the pacing system and can be useful in the determination of pacemaker abnormalities. In this respect analog recordings are superior to digital recordings, because quantitative and qualitative analysis of digital recordings is impossible because of distortion by the equipment.

3.1 Distortion by recording equipment
A wide variety of equipment is used to record the ECG of the pacemaker patient. Although proper pacemaker function can be determined in the majority of cases using these different types of equipment, some of them have limitations especially with regard to the validation of the pacemaker stimulus. In some of them pacemaker timing can be misleading and signals mimicking pacemaker stimuli can be generated by the recording equipment, even in the absence of actual pacemaker stimuli on the analog recorded ECG (see paragraph 3.1.4).

3.1.1 Distortion by analog recording equipment
Distortion other than attenuation caused by analog recording equipment is shown in figure 3.1.
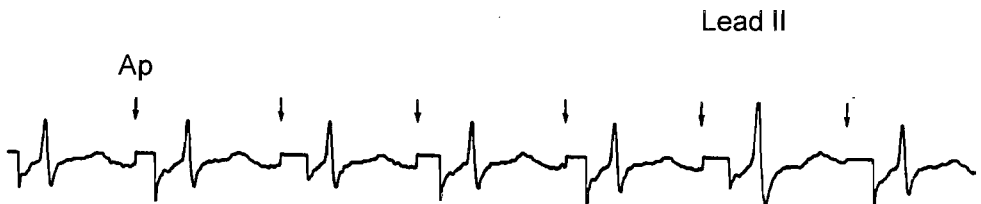
Fig. 3.1. One channel ECG (lead II) recorded from a patient with a unipolar DDD pacemaker programmed in the AAI mode. Gross distortion of the pacemaker stimulus is observed, caused by the automatic baseline reset of the recording equipment. Atrial depolarization is not visible, but effective atrial stimulation can be concluded from the 1:1 ventricular response.

This ECG recorded from a patient with a DDD pacemaker programmed in the AAI mode, illustrates not only distortion of the pacemaker stimulus but also of the ECG following the stimulus.

The pacemaker impulse is completely clamped by the automatic baseline reset of the equipment and electrocardiographic depolarization following the stimulus cannot be concluded. The automatic baseline reset allows the stylus of the recorder to return to the baseline when changing leads. The stimulus artifact in this lead will also operate the automatic reset so that the stylus will return to the baseline and remain inoperable for about 160 ms [2]. Thus no P wave is visible after the stimulus artifact. Because there is a ventricular response following atrial stimuli in a 1:1 ratio, effective atrial pacing can be concluded even in the absence of visible atrial depolarization (see also chapter IV 2.1).

In some electrocardiographs a similar phenomenon can be observed, caused by the inability of the equipment to process the pacemaker stimulus [3,4].

3.1.2 Distortion by digital recording equipment

In equipment employing digital processing, pacemaker stimuli during normal pacemaker function may show a varying amplitude and morphology from biphasic to monophasic (Fig. 3.2). In recording equipment that uses digital processing the ECG is converted into numerical data.
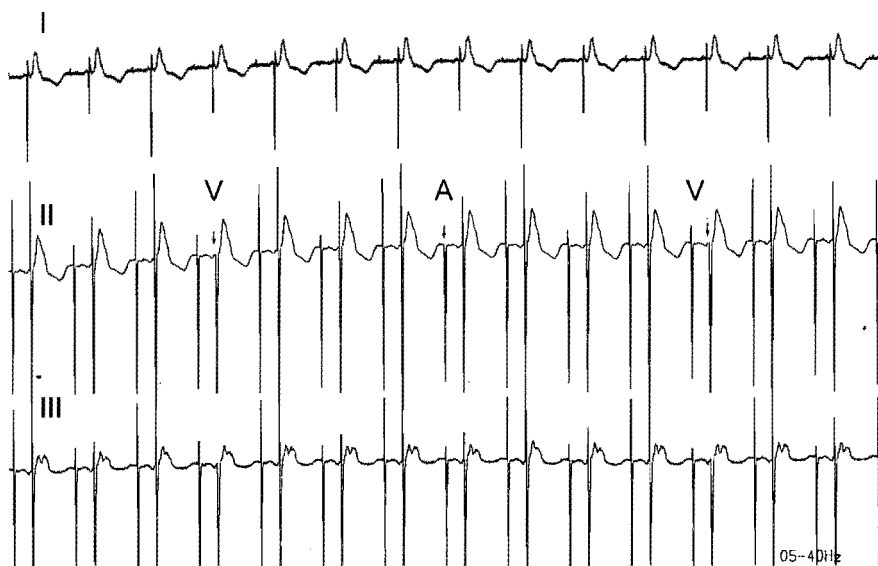
Fig. 3.2. Digital recording of a three channel ECG (lead I,II,III) from a patient with an implanted unipolar DDD pacemaker. There is an alternans in the amplitude of the pacemaker stimuli recorded in lead I, while in lead II changes in the morphology of the pacemaker stimulus from biphasic to monophasic (arrows A,V) is observed. There is no relationship between the amplitude variations in lead I and the variations in morphology in lead II.

These data are stored in a memory for a few milliseconds, and then the ECG is reconstructed from the data for display. Since the sample rate of the equipment is lower compared to the duration of the pacemaker stimulus, the sample may randomly contain all, part, or none at all of the stimulus resulting in variations of the reconstructed pacing artifact. No conclusions can be

drawn from variations in amplitude and morphology of the pacemaker stimulus. Variations should not be interpreted as pacemaker or impending pacemaker malfunction [5].

Clamping of the paced ECG is described in digital equipment, with an electrocardiographic appearance similar to the ECG shown in figure 3.1 [6].

3.1.3 Distortion by monitoring equipment

In monitoring equipment the heart rate is counted by detection of the QRS complex and alarm limits are set to indicate brady- or tachycardia. In order to prevent indication of a normal heart rate by counting noncaptured pacemaker stimuli, the pacemaker stimulus is separated from the ECG in order to count heart rate only from true ventricular depolarization. For the recording part and monitoring display of the equipment an artificial spike is generated to mark the detected pacemaker signal [7]. Because these marker spikes inscribed in the displayed or recorded ECG can be triggered by false signals, and interpreted by the monitoring equipment as pacemaker stimuli, the ECG can be confusing.

19:59:49 29-JUN-1993   25mm/sec
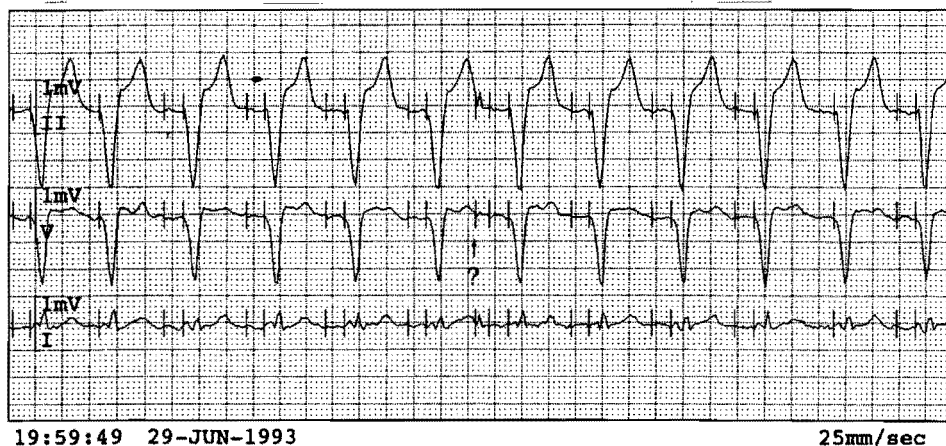
Fig. 3.3. Three channel ECG recorded from a patient with an implanted bipolar DDD pacemaker. The ECG is recorded from monitoring equipment in which an artificial spike is displayed indicating a pacemaker stimulus. In the middle of the recording (arrow?) a "pacemaker spike" is generated erroneously, most probably by detection of an artefact, interpreted by the monitoring equipment as a pacemaker stimulus.

This is illustrated in figure 3.3 and 3.4. Figure 3.3 represents a three lead ECG from a patient with a bipolar DDD pacemaker. In the middle of the recording a "pacemaker stimulus" (arrow) is generated, which is followed by a deflection resembling atrial depolarization. Within 100 ms, this "atrial stimulus" is followed by a stimulus that should be the true atrial stimulus, based on the subsequent ventricular stimulus at the preset AV interval. The first "atrial stimulus" is probably generated by an artefact, which was interpreted by the monitoring equipment as a pacemaker stimulus.

Figure 3.4 shows two consecutive "pacemaker spikes" recorded from a patient with a bipolar DDD pacemaker. Because the coupling interval between these two spikes and the preceding ventricular stimulus is shorter than the VA interval, it can only be interpreted as ventricular

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stimuli. However, the coupling interval between these two "ventricular stimuli" is 180 ms, which is in conflict with the programmed upper rate limit of 120 ppm (= 500 ms). The interval between the second "ventricular stimulus" and the following atrial stimulus does not correspond with the programmed VA interval.

17:00:24 26-APR-1993   25mm/sec
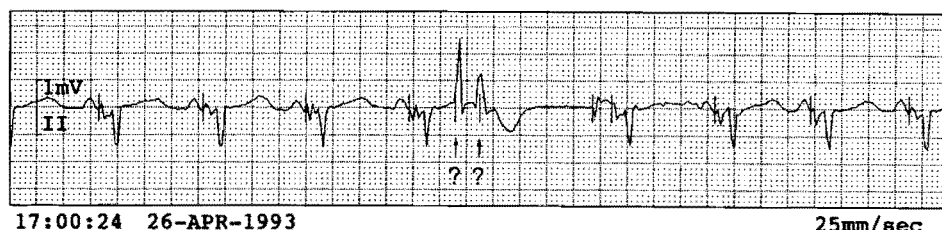
Fig. 3.4. One channel ECG recorded from a patient with an implanted bipolar DDD pacemaker. In the middle of the recording two consecutive "ventricular stimuli" (arrows) are emitted. According to the programmed lower rate (72 ppm) and upper rate (120 ppm=500 ms) the first stimulus can not be an atrial nor a ventricular stimulus and should be generated by an artefact. The second "stimulus" is also generated by an artefact because the interval between this "ventricular stimulus" and the following atrial stimulus does not correspond with the VA interval.

Since pacemaker spikes are artificially generated in this type of equipment, no variations in amplitude and morphology can be observed. Interpretation of pacemaker timing from these recordings should be done with caution, because pseudo-pacemaker stimuli can be generated by events unrelated to the pacing system.

3.1.4 Distortion by pacemaker programming equipment
The majority of the currently available pulse generators are equipped with telemetry. Telemetry offers the possibility of checking the programmed settings, lead impedance, battery status and other parameters essential for the evaluation of the pacing system. One of the options available in pacemaker telemetry is a marker channel or ECG interpretation channel, which indicates atrial and/or ventricular pace and sense events. Because the events indicated by the marker channel have to be related to the ECG, the programmer affords the possibility of a simultaneous recording of the surface ECG and the marker channel. In the ECG recorded with the programmer a pacemaker stimulus of a fixed amplitude (not related to the output setting of the pulse generator) is artificially generated simultaneously with pace events indicated by the marker channel. Figure 3.5 is an example of an ECG and a marker channel recorded from a pacemaker programmer. The marker channel indicates ventricular pace events synchronously with ineffective pacemaker stimuli on the programmer ECG. A simultaneously recorded ECG (same lead as the programmer ECG) from an analog recorder revealed the absence of pacemaker stimuli. No pacemaker stimuli were visible in a 12 lead ECG. The programmer ECG will be interpreted as no capture, while the analog ECG concludes no output, which is an essentially different diagnosis with different underlying potential causes.

3.1.5 Distortion by Holter equipment
In some Holter equipment the pacemaker impulse is filtered and clipped when it is recorded. The recorded pacemaker stimulus is not only difficult to identify, but also distorts the underlying

ECG. As a result, analysis systems can neither identify the existence of paced pulses nor have reliable recognition of the QRS complexes. To overcome these problems in Holter equipment, the pacemaker artifact is detected unequivocally by the recorder electronics and its presence with correct timing is recorded on a separate track. A specially designed circuit eliminates the pacemaker impulse from the ECG. The analyzer then reconstitutes the pacemaker artifact and adds it to both ECG channels from screen display and printout [8].
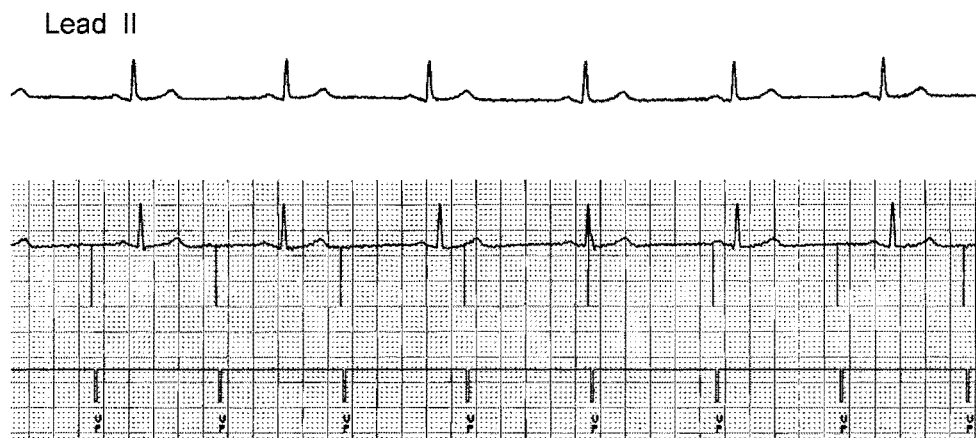
Lead II

Fig. 3.5. Three tracings simultaneously recorded from a patient with an implanted unipolar AAI pacemaker. The upper tracing shows ECG lead II on an analog recorder, the middle tracing shows ECG lead II on a pacemaker programmer together with the marker channel (lower tracing). Pacemaker stimuli with a fixed amplitude are present in the programmer ECG and completely absent in the analog recorded ECG. The analog ECG indicates no output, whereas the programmer ECG suggests no capture and no sensing. No output was confirmed by a interruption in the lead connector. Inspite of AAI pacing, pace events are indicated by Vp in the marker channel.

Addition of the previously separated ECG and pacemaker impulse by incorrectly adjusted recording equipment can lead to a time difference between pacemaker impulse and depolarization, thus creating an ECG with an erroneous delay between depolarization and stimulation impulse. In dual chamber systems it can be interpreted as pacemaker malfunction. Because of this delay the ECG can show ventricular depolarization from the atrial stimulus [9]. See chapter IV. 4.5.4 for detailed explanation.

3.2 Factors affecting amplitude and polarity of the pacemaker stimulus on the ECG

When ECGs of pacemaker patients are recorded on analog equipment the amplitude and polarity of the pacemaker stimulus is able to give qualitative information about the pacing system. The following factors determine the amplitude and polarity of the pacemaker stimulus in the ECG:
1. The amount of electrical charge flowing through the body during stimulation
2. Distance between the anodal and cathodal electrode
3. Position of the anodal and cathodal electrode
4. Recorded ECG lead

5.   Current leakage through insulation defects causing flow of electrical charge through the body other than the charge generated by the stimulation current passing the myocardium.

3.2.1 Effect of the electrical charge

The electrical charge (Q) flowing through the body during stimulation is equal to the stimulation current (I) multiplied by the duration of the stimulation current (t) or pulse width ($Q = I \times t$). The displacement of charge generates a voltage across the ECG electrodes. The amplitude of the pacemaker pulse recorded by the electrocardiograph depends on the stimulation current and the pulse width of the pacemaker stimulus. Figure 3.6 (upper panel) illustrates the simplified schematic presentation of the pacemaker output and the amplifier of the electrocardiograph. If the pacemaker pulse, recorded by the ECG electrodes (Vin), is considered as a rectangular pulse, the signal at the output of the ECG amplifier (Vout) is:

$$\text{Vout} = \text{Vin} \left(1 - e^{-t/RaC} \right) \qquad \{I\}$$

In this formula the values of Ra and C of the amplifier determine the properties of the low band pass filter. The duration of the pacemaker pulse (t) is in practice much shorter than the RC time of the amplifier. This implies that the maximum value of the output voltage (Vout) is significantly smaller than Vin, and that the resulting Vout is determined by the pulse duration t as illustrated in the lower panel of figure 3.6.

The input signal for the electrocardiograph, however, is not a rectangular pulse, but the discharge pulse of the output capacitor (Cp) over the patient's impedance (Rp). The latter is for simplification considered as an ohmic resistance, so the input signal for the electrocardiograph is given in the formula:

$$\text{Vin} = G. \, Vb. \, e^{-t/RpCp}$$

In this formula G is a factor < 1, indicating the attentuation of the measured pacemaker stimulus by the ECG electrodes. The attenuation is determined by the selected ECG lead and the location of the stimulation electrodes in the body. The value of Vin can be substituted in formula I. Although this substitution alters the absolute result of formula I, the previous conclusion showing the relationship between pulse duration and stimulus amplitude on the ECG is not essentially changed. The effect of the pacemaker pulse width on the recorded ECG is illustrated in figure 3.7. Because pulse width is a programmable parameter, the interpreter should be informed about the programmed pulse width at which an ECG is recorded before conclusions can be drawn from amplitude variations in two ECGs recorded at different times. The stimulation current, the second factor determining the amplitude of the pacemaker impulse in the ECG, depends on the programmed output voltage and lead impedance.

This implies that variations in lead impedance are reflected in the ECG by variations in the amplitude of the pacemaker stimulus, provided that the ouput voltage is at a constant level and no secondary electrical pathways caused by insulation defects are present. Figure 3.8 demonstrates variations in pulse amplitude, where during attenuation of the pacemaker stimulus capture and non capture is observed [10]. The inverse statement that variation in pulse amplitude indicates variation in lead impedance is not always true, because the amplitude can also be

affected by changing distance and location of the anodal and cathodal electrode, as for example during respiration. Changes of the pacemaker pulse on the ECG caused by respiration usually show gradual variations, this in contrast to changes in lead impedance that may show abrupt variations of the pacemaker stimulus.
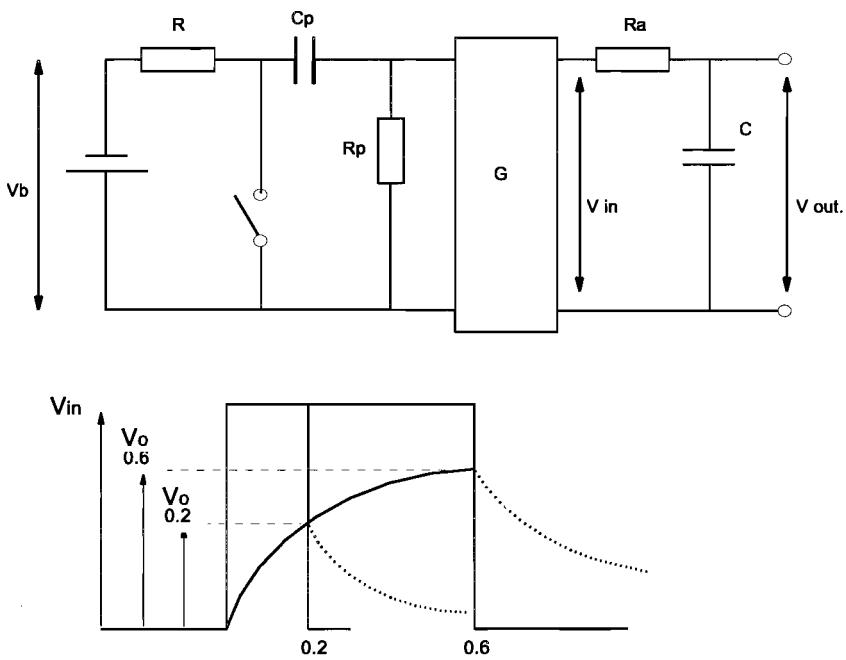
Fig. 3.6. Simplified diagram of the pulse generator and the electrocardiograph (upper panel) illustrating the effect of the pulse width on the amplitude of the recorded pacemaker stimulus (lower panel). See text for detailed explanation.
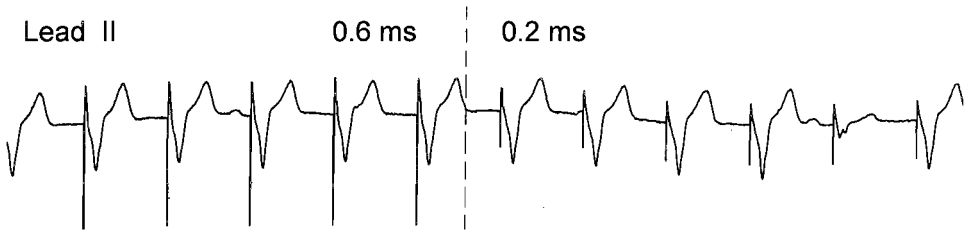
Fig. 3.7. Single channel ECG (lead II) of a patient with an implanted unipolar VVI pacemaker. The ECG shows attenuation of the pacemaker stimulus after programming the pulse width from 0.6 to 0.2 ms, at the same output voltage.

3.2.2 Effect of distance between the anodal and cathodal electrode
The effect of the distance between the anodal and cathodal electrode on the amplitude of the pacemaker pulse in the ECG is best illustrated by comparing unipolar and bipolar pacing (Fig. 3.9).

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Although the terms unipolar and bipolar suggest different pacing systems, by strict definition, all electrical circuits including pacemaker systems are bipolar [11]. The difference between unipolar and bipolar systems is simply the size and location of the anodal electrode [12]. In bipolar systems, in which the cathodal and anodal electrode are incorporated in one lead, the distance is short (usually 25 mm in the currently used electrodes). In unipolar systems the distance between the cathodal and anodal electrode is large and can vary between 10 and 40 cm. The distance depends on the size of the patient and the site of implantation of the pulse generator. The larger the distance between the anodal and cathodal electrode, the larger the electrical vector that is projected on the recorded ECG lead.
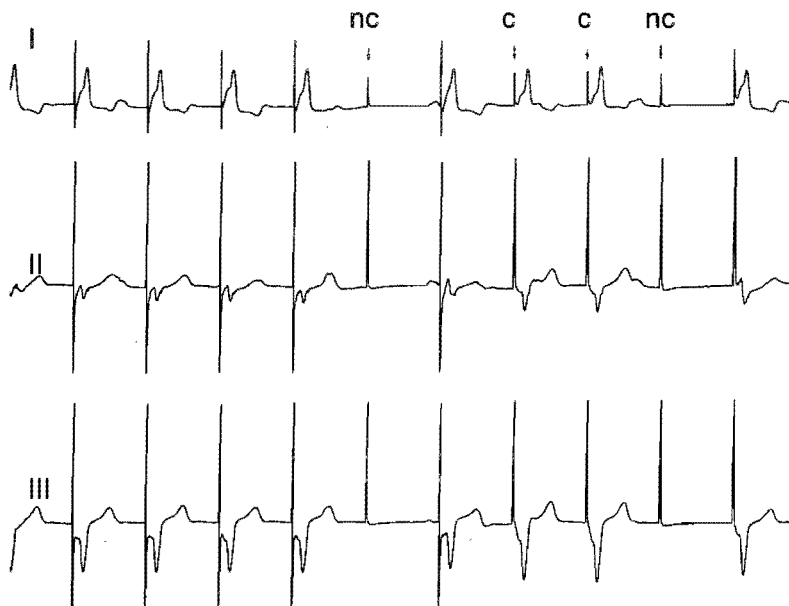
Fig. 3.8. Three channel ECG (lead I,II,III) of a patient with an implanted unipolar VVI pacemaker. The ECG shows abrupt attenuation (lead I) and change in morphology (lead II,III) of the pacemaker stimulus indicating a decrease in stimulation current. Attenuation and change in morphology is associated with capture (C) as well as no capture (NC).

3.2.3 Effect of position of the anodal and cathodal electrode and recorded ECG lead

The positions of the anodal and cathodal electrode determine the direction of the stimulation current through the body. In bipolar ECG leads (lead I,II,III) the amplitude of the pacemaker stimulus is determined by the direction of the stimulation current in relationship to the dipole formed by the two ECG electrodes used to record a specific lead. E.g. Lead II is recorded between the electrode on the right arm and left leg.

When the direction of the stimulation current is parallel to the recorded ECG lead, the pacemaker stimulus will show the largest amplitude. When the direction of the stimulation current is perpendicular to recorded ECG lead, the pacemaker stimulus will show the smallest amplitude. The latter is demonstrated in figure 3.10, which shows lead I, II and III of a patient with an

implanted unipolar VVI pacemaker. The pulse generator itself is the anodal electrode, implanted in the right pectoral region and the cathodal electrode is positioned in the right ventricular apex. Lead I,II and III form a equal sided triangle (Einthoven triangle) in which the vector of the stimulation current is more or less projected equally on lead I and II (same amplitude of the stimulation impulse). This implies that the direction of the stimulation current is more or less perpendicular to lead III, resulting in a low amplitude of the pacemaker stimulus in this lead. Variations in polarity of the stimulation pulse in this lead are due to respiration. Rules for polarity of the pacemaker stimulus are explained in paragraph 3.4 of this chapter.
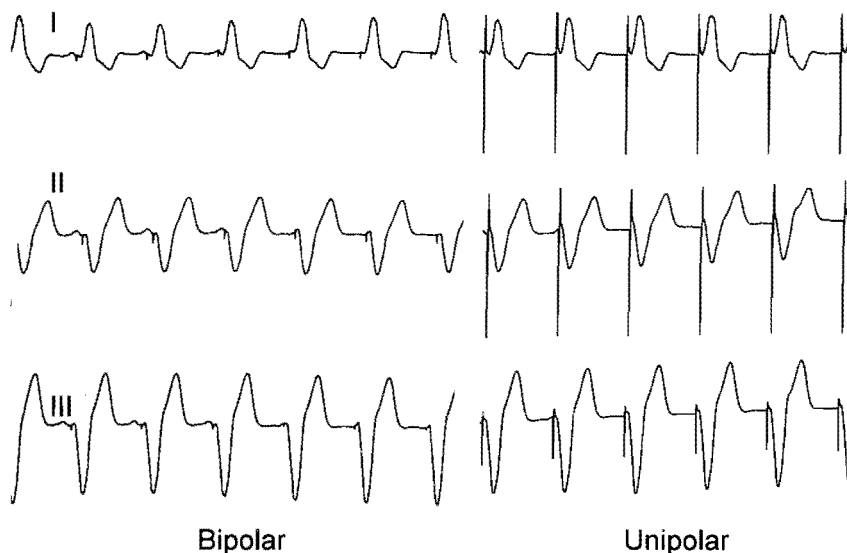
Bipolar   Unipolar

Fig. 3.9. Three channel ECG (lead I,II,III) of a patient with an implanted VVI with a programmable electrode configuration. In the left panel the electrode configuration is programmed in the bipolar and in the right panel in the unipolar configuration, demonstrating the relationship between the pacemaker stimulus and the distance between anodal and cathodal electrode.

3.2.4 Effect of current leakage through an insulation defect

In the previous paragraphs it was assumed that there was only one electrical pathway for the stimulation current, which in practice means that the insulation of the lead is intact and the current flows from cathodal to anodal electrode. In the presence of an insulation defect an alternate pathway for current flow and therefore an additional dipole between the insulation break and the electrode of opposite polarity is created. The pacemaker stimulus in ECG is the vectorial result of the stimulation current (between cathodal and anodal electrode) and the leakage current (between the conductor which is in contact with the body through the insulation defect and the electrode of opposite polarity).

3.2.4.1 Insulation defects in unipolar systems

The site of the leak is usually located near the pulse generator or the site of venous entry and is rarely within the venous system. For unipolar pulse generators implanted in the right pectoral

region the vector of the current leakage (provided that the leak is in the vicinity of the pulse generator) is relatively small and has approximately the same direction as the vector of the stimulation current. This implies that the effect on the original vector of the stimulation current is minimal and will not be noticed in the ECG. In unipolar pacemakers implanted in the left pectoral region the vector of the current leakage is also relatively small, but the direction will be more or less perpendicular to the vector of the stimulation current. Due to this difference in direction the resulting stimulus in the ECG may show a significant change in direction [13].
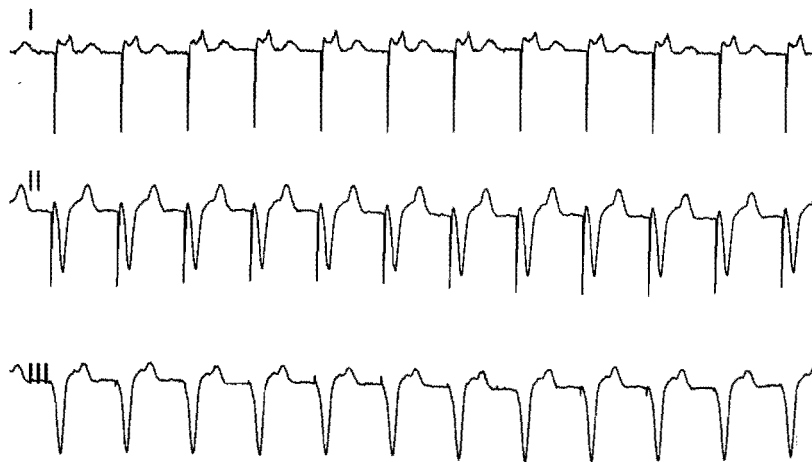
Fig. 3.10. Three channel ECG (lead I,II,III) recorded from a patient with an unipolar VVI pacemaker implanted in the right pectoral region. The small amplitude of the pacemaker stimulus in lead III indicates that the direction of the stimulation current is more or less perpendicular to lead III, confirming the right pectoral position of the pulse generator.

The exception to this theory is in those cases where the insulation defect creates a direct contact between the cathodal conductor and the pulse generator can (anodal electrode). The (almost) short circuit causes a high current pathway with an extremely small vector, and a reduction of the stimulation current by the extremely high load on pacemaker output, resulting in attenuation of the pacemaker stimulus [14]. See also chapter IV 4.7.6.

3.2.4.2 Insulation defects in bipolar systems
The currently used bipolar leads have a coaxial construction. In the coaxial construction the cathodal conductor is insulated and then covered with the insulated coil of the anodal conductor. Therefore, one should differentiate between insulation breaks between anodal and cathodal conductor and breaks of the outer insulation of the lead. An insulation break between the anodal and cathodal conductor will have little or no effect on the amplitude of the pacemaker impulse in the ECG. The insulation break will affect the current drain from the pulse generator, but the stimulation current between anodal and cathodal electrode will not vary unless a complete or almost complete short circuit is created through the insulation defect (see chapter I, fig. 1.4). Because occurrence of insulation breaks between anodal and cathodal conductor is a gradually

developing process, sensing problems caused by variations in lead impedance are observed before decrease in the pacemaker impulse amplitude becomes manifest (see chapter V 5.8.2).
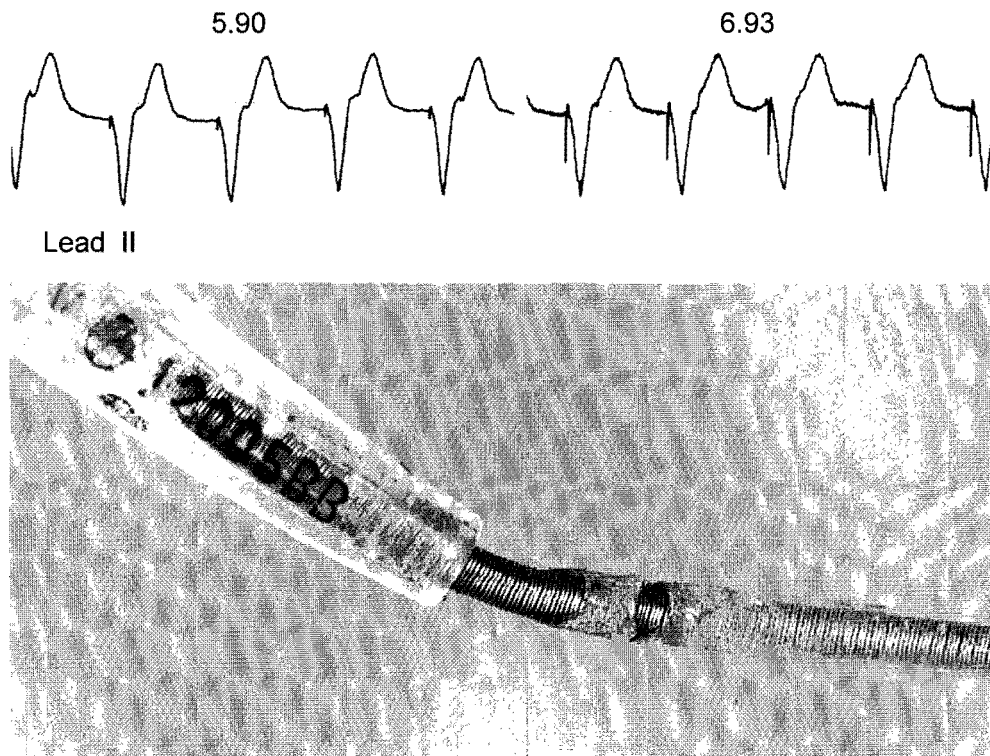
Fig. 3.11. One channel ECG (lead II) of a patient with an implanted bipolar VVI pacemaker. The ECG recorded in may 1990 (left upper panel) shows a low amplitude of the bipolar pacemaker impulse. The ECG recorded three years later, june 1993 (right upper panel) shows an increase of the pacemaker impulse, associated with a decrease in lead impedance which is suggestive for a break in the outer insulation of the lead; "unipolarization" of the pacemaker stimulus. No changes were made in output settings; the same ECG equipment was used for both recordings. The lower panel shows a photograph of the proximal portion of the lead, which was removed after surgical intervention in april 1994. Deterioration of the polyurethane insulation material is observed.

A break in the outer insulation of a bipolar lead occurring in the vicinity of the pulse generator has a clear effect on the ECG. Through this defect an alternate pathway for current is formed between the cathodal electrode and the site of the defect [15]. The new electrocardiographic impulse is the vectorial result of the original impulse and the leakage vector. Because the cathodal-anodal distance is much larger for the leakage vector, it will dominate the electrocardiographic appearance and will show "unipolarization" of the ECG as illustrated in figure 3.11. In this patient the increase in amplitude was accompanied by a small decrease in lead impedance suggestive of an insulation break causing an alternate pathway for current flow. Later surgical intervention confirmed the presence of an insulation defect near the lead connector (Fig. 3.11, lower panel).

3.3 Pseudo variation in pacemaker stimuli

In the majority of pulse generators the output pulse is generated by discharging a capacitor over the circuit formed by the positive pacemaker electrode, the heart and, the negative pacemaker electrode; this can be represented by closing switch S in the diagram of figure 3.12. After discharge, the output capacitor is recharged (S open) over the internal resistance (R) and the patient's resistance Rp (Rp << R). Recharge time depends on the values of R, Rp, C, the amount of charge delivered during stimulation and is limited by the interval between pacing pulses. To obtain a short recharge time from the output capacitor the internal resistance should be low. A short recharge time, thus a low value of R, allows complete recharging even at high pacing rate and high pacing output. A low internal resistance also helps to quickly reduce the poststimulus polarization potential (sometimes called afterpotential). However a low internal resistance increases internal current consumption during the stimulus, when S is closed, and reduces the input impedance of the pulse generator, thereby impairing measurement of intracardiac signals. The solution for this dilemma was the introduction of fast recharge circuitry. This circuit reduces the resistance of R for a period of less than 10 ms. In the schematic presentation fast recharge circuitry is represented by a switch (FRS, fast recharge switch) that is closed for less than 10 ms after delivery of the pacemaker stimulus.
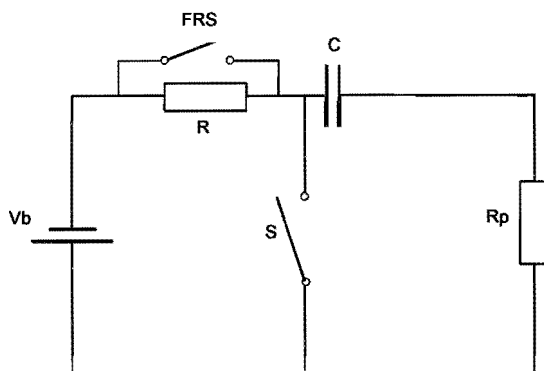
Fig. 3.12. Simplified diagram of the fast recharge circuitry. Stimulation is represented by closing switch S, which results in discharge of the output capacitor C over Rp, representing patient's resistance. Stimulation stops after reopening of S, followed by recharge of the output capacitor. Recharge time is determined by the value of C, Rp and R (internal resistance of the pulse generator). By closing the fast recharge switch (FRS) for a short period of time (< 10 ms), capacitor recharge time will be diminished.

In some older pulse generators (Medtronic 5985) rapid recharge was also initiated after sensing. This was designed in the belief that rapid recharging of the output capacitor might also be desirable during sensing [16]. As a result of this circuit design rapid recharge pulses can be recorded in the ECG during sensing of intrinsic activity (Fig. 3.13). Because the rapid recharge pulses have a smaller amplitude than the pacemaker stimuli they may be confused with attenuation of the pacemaker stimulus. Rapid recharge pulse may be invisible in VVI pacemakers, because this low amplitude pulse is masked by the amplitude of the QRS complex, but is evident in the intrinsic P wave in AAI pacemakers, as illustrated in figure 3.13. Although

rapid recharge during sensing is eliminated in the currently available pulse generators, these units are still implanted and can be encountered during pacemaker follow up.
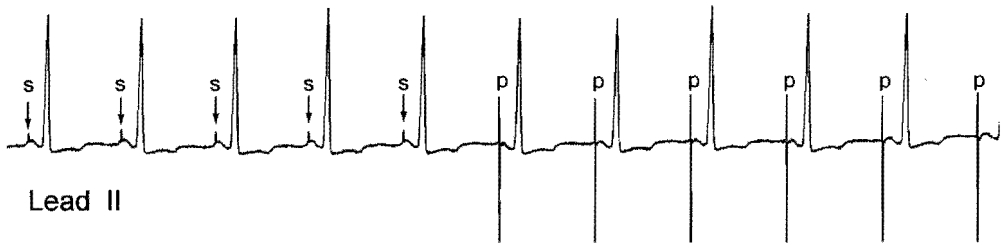
Fig. 3.13. Single channel ECG (lead II) of a patient with an implanted unipolar AAI pacemaker. The last portion of the ECG shows a large unipolar pacemaker stimulus (P) followed by atrial depolarization. In the first portion of the recording small "stimuli" (S) are recorded in the P wave. The latter are rapid recharge pulses during sensing, and should not be interpreted as attenuated pacemaker stimuli.

3.4 Location of the anodal electrode in normal functioning unipolar pacing systems

The amplitude of the pacemaker pulse on the ECG from a unipolar pacemaker is directly proportional to the current in the stimulation system. In a constant voltage pacemaker (most of the presently used implantable pulse generators) the amplitude of the pacemaker stimulus on the ECG depends on the output voltage of the pulse generator and the system impedance.
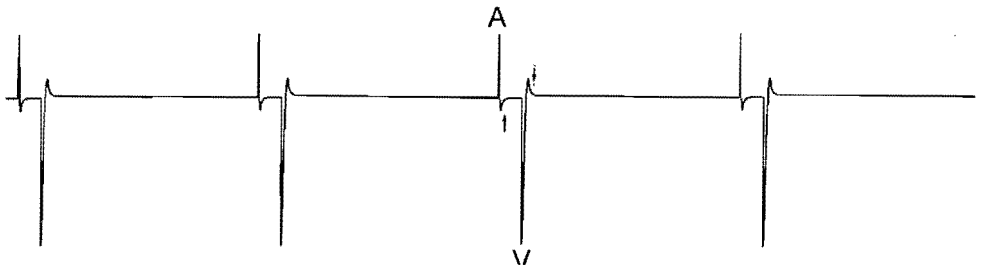
Fig. 3.14. Atrial (A) and ventricular (V) stimuli recorded from a DDD pacemaker in a saline bath, to illustrate the fast recharge (arrows), which has an opposite direction and a longer duration as compared with the pacemaker stimulus

The amplitude and polarity of the pacemaker spike, as recorded on the ECG, are not only affected by this output setting but also by the location of anodal and cathodal electrode and the recorded ECG lead. Polarity of the pulse, assuming that current is flowing from cathode to anode, follows the electrocardiographic rules for depolarization. E.g., stimulation current in a superior-inferior direction gives a positive deflection in lead II and III and stimulation current going from right to left a positive deflection in lead I. For unipolar leads, a stimulation current flowing towards the recording electrode gives a positive deflection and a negative deflection is recorded when a stimulation current moves away from the recording ECG electrode. One should take into consideration that the real pacemaker pulse is only reflected in the initial portion of the

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pacemaker spike on the ECG. The junction between pacemaker impulse and QRS complex reflects effects due to polarization and rapid recharge of the output capacitor (Fig. 3.14). The latter effect, which is in polarity opposite to the pacemaker pulse, should be omitted in analysis of the location of the anodal electrode. For instance, if a pulse generator is implanted in the right pectoral region and the cathode is located in the right ventricular apex, the stimulation current in the frontal plane flows from inferior to superior and from left to right. The frontal plane ECG leads I, II, III, will show a pacemaker spike with negative deflection (Fig. 3.15).
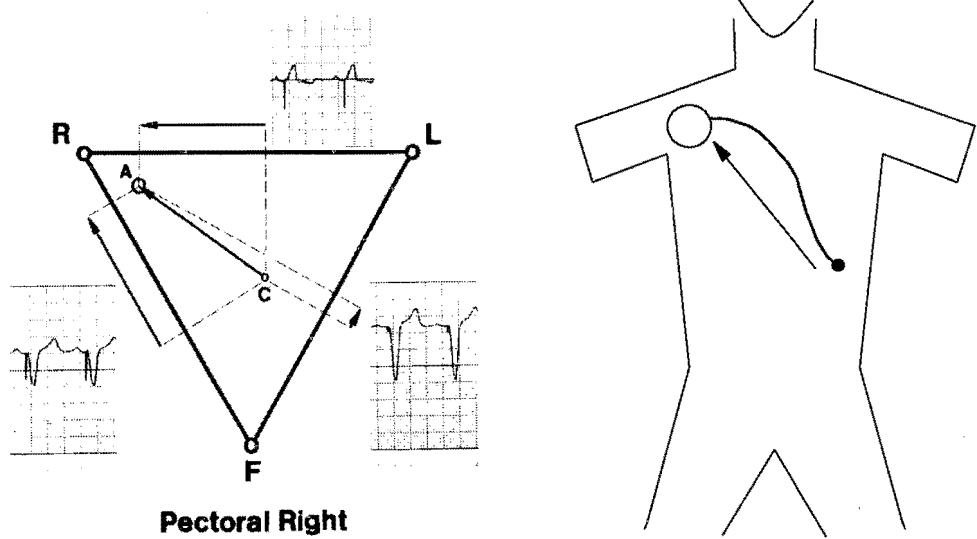
Pectoral Right

Fig. 3.15. Diagram illustrating determination of the location of the anodal electrode of a patient with a unipolar ventricular pacemaker. The anodal electrode is located in the right pectoral region.

Pulse generators implanted in the left pectoral region with a current flow in an inferior-superior and right-left direction in the frontal plane will demonstrate a positive deflection in lead I, and a negative deflection in leads II and III (Fig. 3.16). In addition, the direction of the current flow of a pulse generator implanted in the right abdominal region with the cathodal electrode in the right ventricular apex is from superior to inferior and from left to right. The ECG in these patients displays a positive stimulus artifact in leads II and III and negative deflections in lead I (Fig. 3.17). The rules to determine the location of the anodal electrode in unipolar systems (location of the pulse generator) can be summarized as follows:

Negative deflection II and III	→ *Pectoral location*
Negative deflection I	→ *Right pectoral location*
Positive deflection I	→ *Left pectoral location*
Positive deflection II and III	→ *Abdominal location*
Negative deflection I	→ *Right abdominal location*
Positive deflection I	→ *Left abdominal location*

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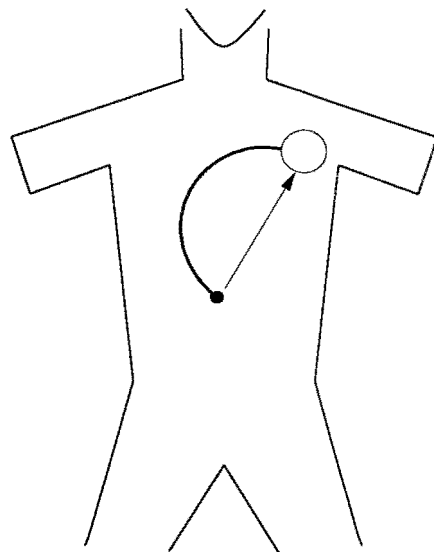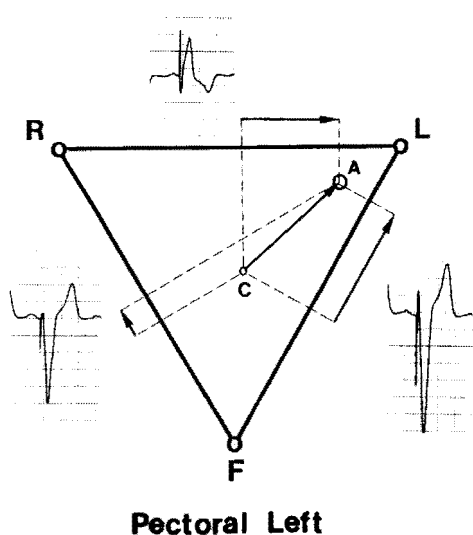
Pectoral Left

Fig. 3.16. Diagram illustrating determination of the location of the anodal electrode from the ECG of a patient with a unipolar ventricular pacemaker. The anodal electrode is located in the left pectoral region.
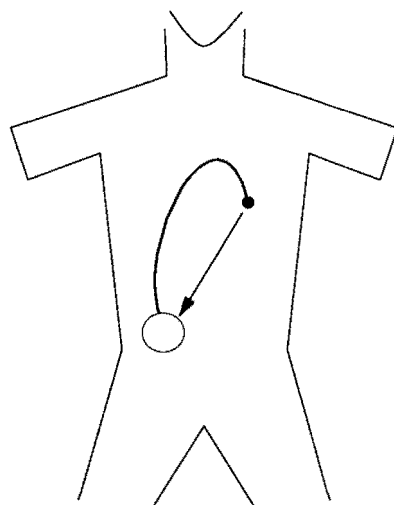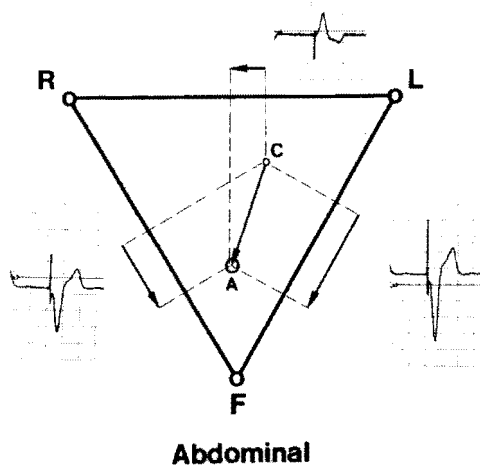
Abdominal

Fig. 3.17. Diagram illustrating determination of the location of the anodal electrode from the ECG of a patient with a unipolar ventricular pacemaker. The anodal electrode is located in the right abdominal region.

3.5 Location of the cathodal electrode in malfunctioning unipolar pacing systems

As explained in the previous paragraph the location of the anodal electrode (site of the pulse generator in unipolar systems) can be determined from the stimulus axis in the ECG. Because the location of the pulse generator hardly varies after implantation, the same rule can be applied to

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the cathodal electrode in unipolar systems. This means that variations in the position of the cathodal electrode will be reflected in the ECG by an alteration of the pacemaker stimulus axis. Because only gross displacement will be detected by this method, displacement for instance of a ventricular lead that still captures the ventricle will be difficult to detect by employing the stimulus axis as a diagnostic tool. The diagnosis can usually be made by other means, including changes in ventricular depolarization, which definitely is a more sensitive parameter [17].

Gross displacement of a ventricular lead towards the right atrium or pulmonary artery will be seen in the ECG by a change in stimulus axis. For a right pectoral implantation the electrical stimulus axis will change to a more vertical position in case of displacement of the lead into the right atrium, while displacement into the pulmonary artery will give the axis a more horizontal position. A recent publication by Storm and van Mechelen clearly demonstrated the use of this method in a patient with a completely broken lead. The position of the end of the broken lead, functioning as the cathodal electrode, could be established from the vector analysis of the pacemaker stimulus [18].

3.6 Summary and conclusions

The pacemaker stimulus properly recorded by ECG electrodes, represents the stimulation current and the direction of the current. In digital equipment distortion of the pacemaker stimulus may occur by the signal processing technique. This results in variation of the amplitude and/or polarity, not related to the direction and strength of the stimulation current. In some digital equipment, the pacemaker stimulus is filtered and an artificially generated pulse is displayed on the ECG. This pulse has a fixed amplitude and polarity, independent of the variations of the pacemaker pulse as recorded on the ECG electrodes. Artifacts resembling pacemaker stimuli, will also result in erroneously generated pacemaker stimuli. In some Holter equipment even the time relation between pacemaker stimuli and resulting depolarization can be distorted. This implies that no conclusions can be drawn from ECGs obtained from digital equipment with respect to the strength and direction of the stimulation current. The interpreter should also be aware of the possibility of "pseudo pacemaker spikes", indicative of pacemaker malfunction, which can be generated in some monitoring equipment.

In analog recording equipment the amplitude of the pacemaker pulse is directly related to the electrical charge displacement inside the body, whereas the polarity reflects the direction of the stimulation current. Because the amplitude of the pacemaker stimulus is small in bipolar systems, variations in pulse amplitude will be difficult to observe in these pacing systems. In unipolar systems, where the amplitude of the stimulus is much larger, variations in amplitude are caused by a change in the position of the electrical dipole and/or a change in stimulation current. Changes in amplitude, due to variation of the electrical dipole, are usually caused by respiration, affecting the position of the stimulation electrode. These variations are characterized by a gradual change, synchronously with respiration movements. This implies that variations in amplitude of the pacemaker spike on the ECG, other than respiration related changes, are caused by variation in the stimulation current, which is inversely proportional to the lead impedance. In practice this generally means that attenuation of the pacemaker spike indicates an increase in lead impedance. The exception to this rule is a short circuit between the pacemaker case (anodal electrode) and the conductor of the lead (cathodal electrode). Under these conditions the stimulation current is diminished, due to the additional dipole, and it will be evident that the measured lead impedance will be extremely low.

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The amplitude of the pacemaker stimulus on the ECG depends on the output voltage, the pulse duration of the pulse generator, lead impedance and the electrical properties of the recording equipment. This means that comparison of ECGs, recorded at different times, with respect to the amplitude of the pacemaker stimulus can only be made if none of these conditions (with exception of lead impedance) are changed. Variation in amplitude can be monitored more reliably by direct measurement of the pacemaker stimulus by an electronic device or pacemaker pulse waveform analysis.

The amplitude and polarity of the pacemaker stimulus in unipolar systems can be used to determine the position of the anodal electrode (pacemaker case). It is a rough technique that allows the interpreter to discriminate between pectoral left and right and abdominal left and right implantations of the pulse generator. Microdislodgement of the stimulation electrode cannot be established with this technique. Macrodislodgement or a completely broken lead, which is electrically equivalent to macrodislodgement, and the position of the cathodal electrode (or the site of the lead breakage) can be determined with these rules.

As previously mentioned, the amplitude of the pacemaker stimulus recorded in bipolar systems contains only limited information. An increase in amplitude, provided that output voltage, pulse duration and equipment are the same, is indicative of a defect in the outer insulation of the lead in the proximity of the pulse generator. The increase in amplitude is largely caused by the change in the electrical dipole ("unipolarization") and less by an increase in stimulation current, although it is often associated with a slight decrease in lead impedance.

The absolute value of the information of the pacemaker stimulus on the ECG is limited. However in the case of malfunction or impending malfunction it may contain clues to the origin of the (impending) failure mechanism.

References

1. Escher DJW, Furman S. Modern methods of follow-up of the patient with an implanted cardiac pacemaker. Am J Cardiol 1971; 28: 359-365.

2. Mond HG, Sloman JG. The malfunctioning pacemaker system. Part II. PACE 1981; 4: 168-181.

3. Barold SS, Falkoff MD, Ong LS, et al. The abnormal pacemaker stimulus. In SS Barold (Ed): Modern Cardiac Pacing. Mount Kisco, New York, Futura Publishing Company, 1985, p.577.

4. Murdock DK, Moran JF, Hwang MH, et al. Pacemaker malfunction simulated by amplifier saturation. PACE 1985; 8: 757-758.

5. Engler RL, Goldberger AL, Bhargava V, et al. Pacemaker spike alternans: An artifact of digital signal processing. PACE 1982; 5: 748-750.

6. Higgins JR, Swartz JF, Fessenden KK. Pseudopacemaker malfunction secondary to digital ECG artifact. Am Heart J 1986; 111: 608-610.

7. Instruction manual Siemens Sirecust 1280/1260/960. Chapter 10. EKG and Heart Rate Monitoring, p. 10-7. October 1992.

8. Operator's Manual for the CardioData Holter Analysis System, Prodigy, Version 7.01. Milwaukee, WI, Mortara Instrument, Inc.

9. Van Gelder LM, Bracke FALE, El Gamal MIH. Fusion or confusion on Holter recording. PACE 1991; 14: 760-763.

10. Barold SS, Scovel J, Ong LS, et al. Periodic pacemaker spike attenuation with preservation of capture: An unusual electrocardiographic manifestation of partial pacing electrode fracture. PACE 1978; 1: 375-380.

11. Mond HG. Unipolar versus bipolar pacing- Poles apart. PACE 1991; 14: 1411-1424.

12. Van Gelder LM, Nagelkerke D, Mulder H, et al. Unipolar versus bipolar electrode configuration in cardiac pacing. Neth J Cardiol 1992; 5: 280-285.

13. Sheridan DJ, Reid DS, Williams DO, et al. Mechanical failure causing current leakage with unipolar pacemakers. Significance and detection. Eur J Cardiol 1978; 8: 1-8.

14. Van Gelder LM, Bracke FALE, El Gamal MIH. Adapter failure as a cause of pacemaker malfunction. PACE 1993; 16: 1961-1965.

15. Fontaine G, Sautel JM, Bonnet M, et al. Le syndrome de fuite électrique chez les porteurs de pacemaker. Arch Mal Coeur 1972; 65: 190-198.

16. Barold SS, Kulkarni H, Thawani AJ, et al. Electrocardiographic manifestations of the rapid recharge function of pulse generators after sensing. PACE 1988; 11: 1215-1219.

17. Preston TA. Electrocardiographic diagnosis of pacemaker catheter displacement. Am Heart J 1973; 85: 445-451.

18. Storm CJ, van Mechelen R. A severed pacemaker lead entrapped in a hepatic vein. PACE 1993; 16: 1349-1353.

Chapter IV. **Effectiveness of Stimulation**

Introduction
Effectiveness of stimulation is the most essential part of cardiac pacing. Effective ventricular stimulation in single chamber systems is easily recognized from the ECG. In dual chamber systems it sometimes can be misleading in the presence of atrial pacing and/or sensing and normal atrioventricular conduction associated with left bundle branch block. Effectiveness of atrial stimulation is also more difficult to conclude in dual chamber pacemakers than in single chamber systems, where the 1:1 ventricular response can be used to confirm effective stimulation.

Different pittfalls may be encountered in the ECG interpretation before the conclusion "effective or ineffective stimulation" can be drawn. After confirmation of the diagnosis "ineffective stimulation", it should be differentiated between noncapture and no output. Causes of noncapture and no output, together with the electrocardiographic recognition, are discussed in this chapter.

4.1 Definitions

Capture
Depolarization of the myocardium in response to an electrical stimulus emitted by the pacemaker.

Nonpacing
Nonpacing is a definition used for two phenomena. Nonpacing is either the lack of capture (noncapture) or the lack of an output from the pulse generator (no output). To avoid confusion in specific circumstances the term nonpacing should not be used under these conditions.

Noncapture
Noncapture is defined as no depolarization of the myocardium in response to an electrical stimulus by the pacemaker.
Ineffectiveness of the stimulus may not be caused by refractoriness of the adjacent myocardium. If the latter is present on the ECG it should be classified as undersensing rather than noncapture.

No output
No output means that there is no pacemaker stimulus visible on the ECG. However, there are 2 prerequisites:
1.   The programmed pacing rate must be higher than the patient's intrinsic rate, provided that the pacing system has no hysteresis.
2.   After fulfilling the first condition magnet application must **not** result in the appearance of pacemaker stimuli. If magnet application results in asynchronous stimulation the phenomenon must be classified as oversensing instead of no output. This should be verified in a multichannel ECG; a single lead ECG can be misleading in this respect.

Fusion beat
Fusion beat is defined by Bellet [1] as the electrocardiographic complex that results from the collision or summation of wavefronts within the ventricle or atrium, the resulting beat

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representing the algebraic summation of the two electrical forces. Later on the American College of Cardiology Task Force I on Standardization of Terminology and Interpretation stated that "a fusion complex is produced by simultaneous or nearly simultaneous activation of either the atria or ventricles by impulses coming from different directions. This results in an electrocardiographic complex that is intermediate in form between the pure complexes resulting from the differently originating excitation waves." [2]

When the latter definition is applied to patients with artificial cardiac pacemakers a fusion complex is produced by simultaneous activation of ventricles (or atria) by electrical stimulation and intrinsic ventricular (or atrial) activity [3]. Figure 4.1 demonstrates fusion beats in a patient with a VVI pacemaker.
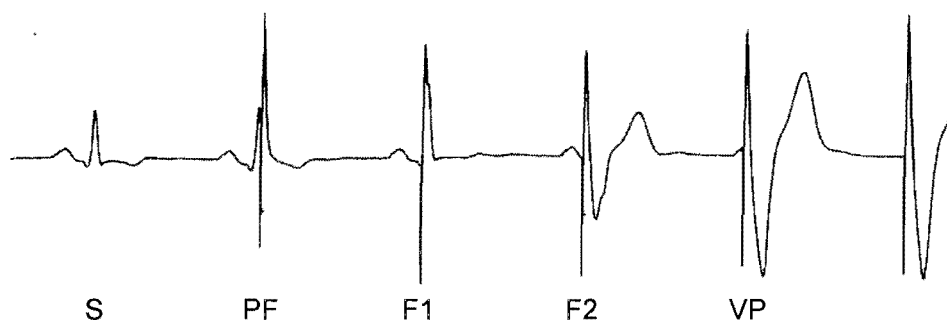
Fig. 4.1. ECG lead II of a patient with an implanted VVI pacemaker showing subsequently normal conducted sinus beat(S), pseudofusion beat (PF), fusion beat with dominant intrinsic depolarization (F1), fusion beat with dominant pacemaker depolarization (F2), ventricular paced beat (VP).

Pseudofusion beat

In cardiac pacing a pacemaker pulse can be emitted simultaneous or nearly simultaneously with intrinsic cardiac activity without resulting fusion beat. The pacemaker pulse is emitted at the time that depolarization of the ventricular (or atrial) myocardium adjacent to the electrode has already started. Because the refractoriness of the adjacent myocardium, the stimulus is ineffective and does not contribute to ventricular (or atrial) depolarization. This phenomenon is called pseudofusion. Pseudofusion represents a normal manifestation of demand pacing and occurs simply because a large portion of the surface QRS (or P wave) may be inscribed before its intracardiac voltage generates the required signal to inhibit a pulse generator [4].

Electrocardiographically pseudofusion is similar to the single chamber triggered pacing mode in the presence of intrinsic activity. See also figure 4.1.

Pseudopseudofusion beat

In dual chamber pacing systems an atrial spike can be delivered within the QRS complex producing beats similar to ventricular pseudofusion beats. According to Barold et al. [5,6] these may be considered as "pseudopseudofusion beats", because sensing and pacing occurs in different chambers. The mechanism is identical to that of conventional ventricular pseudofusion beats, because the pulse generator delivers its atrial output just before the QRS complex has

generated sufficient intracardiac voltage to be sensed from the ventricular electrode. Figure 4.2 shows an example of pseudopseudofusion beat in a patient with an implanted DVI committed pacemaker.

Lead II

ppf

Fig. 4.2. ECG lead II of a patient with an implanted "committed" DVI pacemaker. First 3 beats show normal AV sequential pacing, fourth beat shows ventricular pseudofusion, fifth beat illustrates pseudopseudofusion (ppf). Due to the committed mode, there is no ventricular sensing during the AV interval and an ineffectual ventricular stimulus is delivered in the first part of the T wave. Normal pacemaker function.

4.2 Atrial capture in single chamber pacing

Due to the low amplitude of atrial depolarization and the relatively high amplitude of the pacemaker pulse in the ECG, atrial depolarization caused by the pacemaker stimulus is sometimes difficult to recognize. Particularly in unipolar atrial pacing the stimulus can completely mask the atrial depolarization (fig. 4.3).
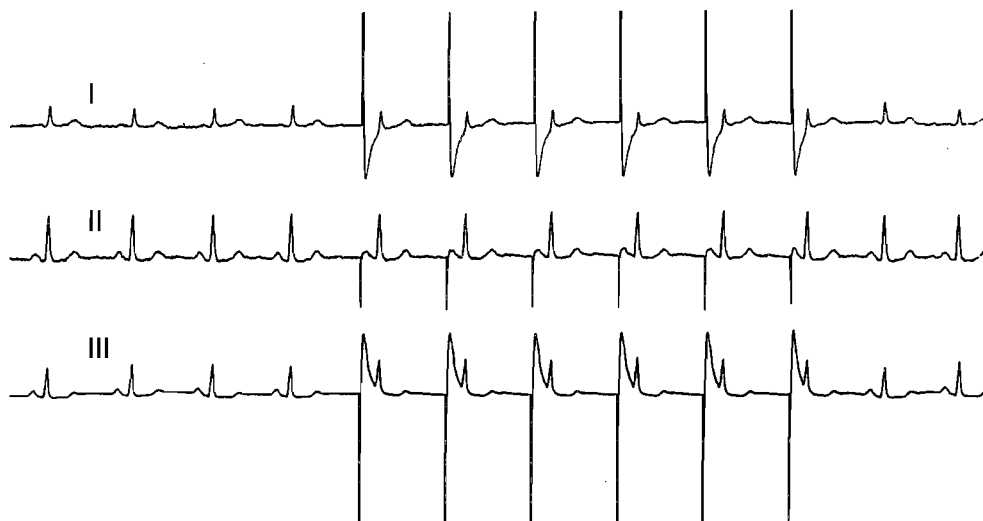
Fig. 4.3. ECG lead I, II and III of a patient with an implanted AAI pacemaker. In leads I and III atrial depolarization is invisible, but lead II clearly shows effective atrial stimulation.

Interpretation of atrial capture in bipolar atrial pacing is rarely a problem, although recognition of the pacemaker stimulus can be difficult in single lead ECGs.

4.2.1 Ventricular response in atrial pacing

Although interpretation of the ECG with respect to atrial depolarization in unipolar pacing systems can be difficult, effectiveness can be shown by consistent normal ventricular depolarization following the atrial stimulus by a relatively fixed atrioventricular interval, even when the P wave is obscured by the large atrial stimulus artifact [7].

To exclude the coincidence of an atrial pace pulse just preceeding an intrinsic P wave both at the same rate, alteration of the pacing rate should be attempted (e.g. rate programming, magnet application). If ventricular response varies synchronously with the atrial pacing rate, atrial capture is confirmed (Fig. 4.4).
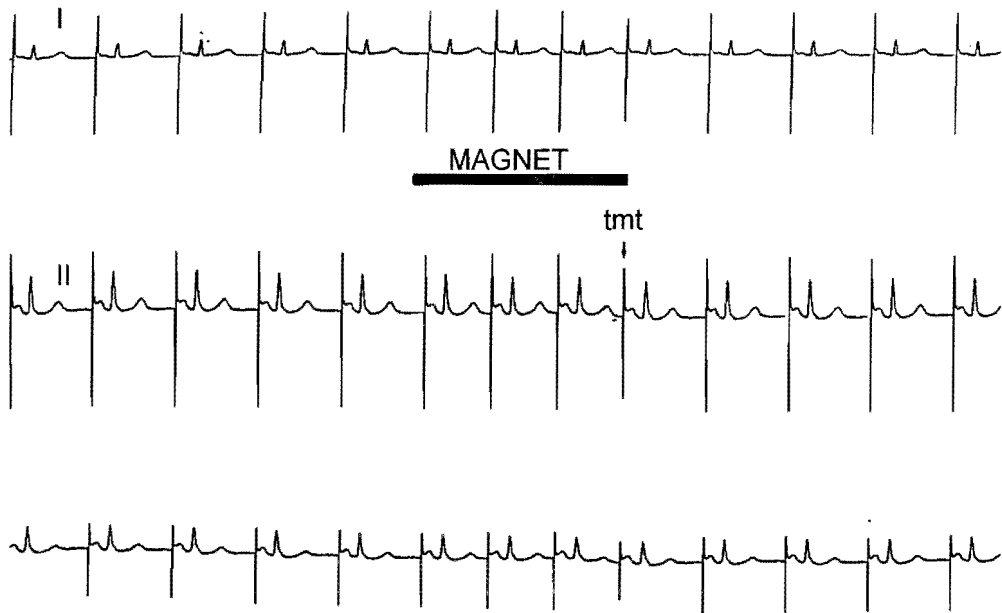
Fig. 4.4. ECG lead I, II, III of a patient with an implanted AAI pacemaker. Pacing rate 80 ppm.; during application of a magnet a set of 3 asynchronous pulses is emitted at a rate of 100 ppm. Ventricular response varies synchronously with the atrial pacing rate, thus confirming atrial capture. During magnet application the first 2 pulses are at the programmed pulse width, the third has a pulse width that is 75% of the programmed pulse width. Threshold margin test (tmt).[8]

Also in atrial fibrillation, when the ventricular rate is equal to the atrial pacing rate and the atrial stimulus is preceding the QRS complex with an interval equal to the AV conduction time, pseudo atrial capture is observed (Fig. 4.5). Recording the ECG for a longer period often demonstrates that the relationship between atrial stimuli and QRS complex is a coincidence (Fig. 4.6).

The 1:1 atrial pacing ventricular response used as confirmation of atrial capture can sometimes be misleading. It is a well known fact that AV conduction depends on the pacing cycle length. Shortening of the paced cycle lengths can induce AV nodal block.

AV conduction also depends on medication and sympathetic and vagal tone [9]. The latter implies that prolongation of the AV interval even with second degree type I AV block can be observed with relative low stimulation rates. Under both conditions 1:1 atrial pacing ventricular response can be lost while effective atrial stimulation is maintained.

Figure 4.7 shows the ECG of a patient with an atrial demand pacemaker (AAI) with a pacing rate of 80 ppm. During application of the magnet, the pacing rate is increased to 100 ppm. In the upper tracing (lead I) it is clear that after the first interval of 600 ms (100 ppm) the atrial stimulus is not followed by a ventricular depolarization. From this lead only it is hard to say whether atrial stimulation was effective or not. However, lead II (lower tracing) clearly shows an atrial stimulus followed by atrial depolarization. This ECG illustrates that atrial capture can sometimes be maintained without a 1:1 ventricular response.
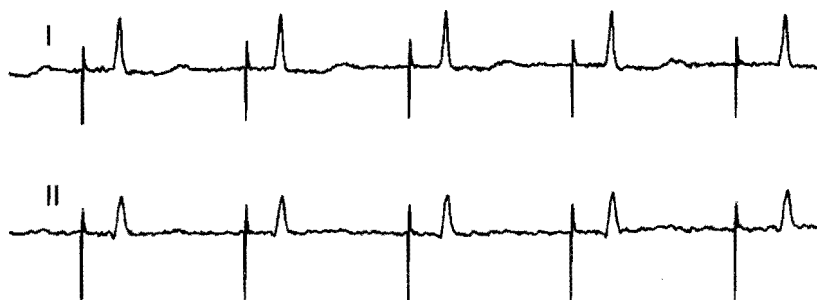
Fig. 4.5. ECG lead I and II of a patient with an implanted AAI pacemaker. Although no atrial depolarization is visible on the ECG, there seems to be a 1:1 relation between the atrial stimulus and the following QRS complex. This relationship does not exists but is purely coincidental (see also fig. 4.6).
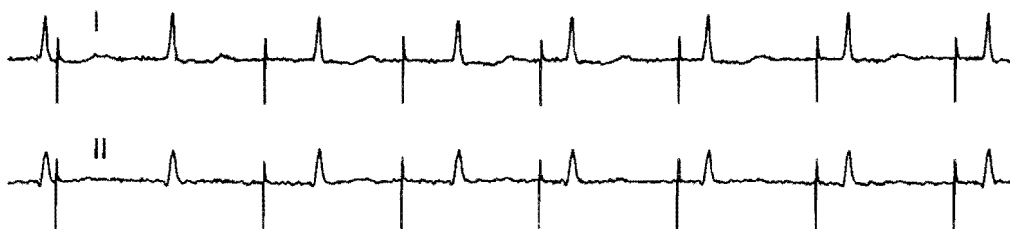
Fig. 4.6. ECG lead I and II, same patient as figure 4.5, recorded for a longer period, showing ineffectual atrial stimuli due to atrial fibrillation.

4.2.2 Atrial noncapture

To conclude atrial noncapture from the ECG, the following prerequisites should both be fulfilled:
1.   Absence of paced atrial depolarization in the conventional 12-lead electrocardiogram.

2. No 1:1 relation between atrial stimulus and QRS complex at the lowest pacing rate setting.

If there is any doubt in the interpretation of the 12-lead ECG, additional recording of right precordial leads at different intercostal spaces with a higher gain setting of the recording equipment should be performed (see also paragraph 4.3.2)

If atrial noncapture is confirmed by the rules described above, electrical inexcitablity of the atrium due to atrial fibrillation or atrial standstill should be excluded. However, atrial standstill with atrial inexcitability is a rare event and only reported in patients prior to implantation of a ventricular (rate adaptive) pacemaker [10,11]. It has not been reported as a cause of atrial noncapture in patients with implanted atrial pacemakers. Atrial standstill cannot be diagnosed from the 12-lead electrocardiogram but should be confirmed by electrophysiological study.
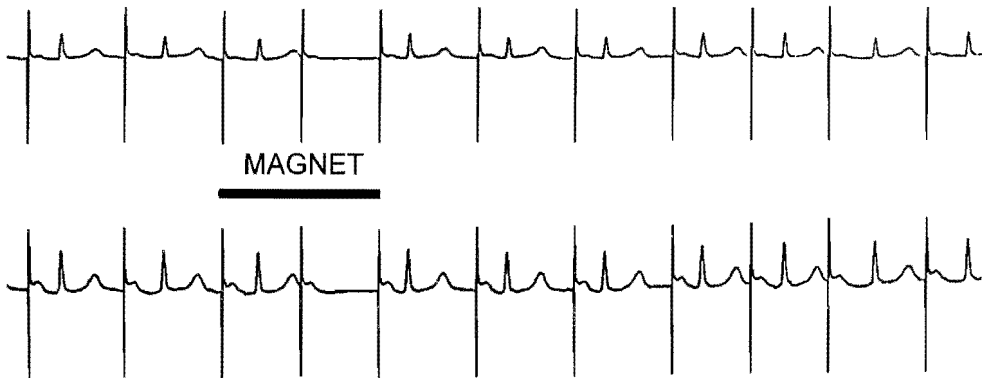
Fig. 4.7. ECG lead I (upper tracing) and lead II (lower tracing) of a patient with an implanted AAI pacemaker, pacing rate 80 ppm. Application of the magnet, resulting in an increase in pacing rate to 100 ppm, shows AV nodal block due to the sudden shortening of the pacing interval. Atrial capture is confirmed in lead II, but hardly visible in lead I.

Figure 4.8 shows an ECG of a patient with an implanted AAI pacemaker, pacing rate 70 ppm. It illustrates that continuous atrial capture is present without a fixed AV interval. In spite of the low stimulation rate there is gradual prolongation of the AV interval from 280 to 330 ms. After prolongation of the AV interval to 330 ms, the QRS complex is sensed by the atrial electrode and resets the escape interval (farfield QRS sensing; Chap V 5.6.2) Decrease of the stimulation rate to 60 ppm resulted in a 1:1 response with an AV interval of 240 ms.

Fig. 4.8. Single channel ECG showing gradual prolongation of the stimulus-R interval while atrial capture is maintained. When the stimulus-R interval exceeds the refractory period, QRS is sensed (arrow) and resets the escape interval. Numbers indicate stimulus-R interval.

4.2.3 Ventricular pacing by the atrial electrode

Dislodgement of atrial J leads can be observed after implantation in the atrial appendage. Displacement usually results in noncapture and/or atrial undersensing. Very rarely the atrial electrode can displace across the tricuspid valve and even move between right atrium and the outflow tract of the right ventricle (chapter IV 4.5.2). Under these circumstances the atrial electrode may be able to induce atrial as well as ventricular stimulation in an alternating way [12,13]. This phenomenon is also reported in a patient with an atrial screw-in lead that dislodged 6 months after implantation. This atrial lead migrated to the right ventricle resulting in right ventricular apical stimulation [14].
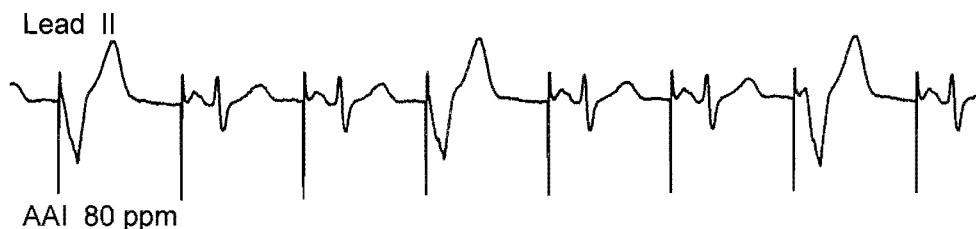
Fig. 4.9. ECG lead II of a patient with an implanted AAI pacemaker, pacing rate 80 ppm. The first and fourth stimulus suggests ventricular pacing by the atrial stimulus. The delay between the atrial stimulus and the third wide QRS complex eliminates this option. Normal pacemaker function. See text for additional information.

The ECG (lead II) shown in figure 4.9 can be misleading in this respect. It shows the ECG of a patient with an implanted AAI pacemaker, pacing rate 80 ppm. The second and third pacemaker stimulus captures the atrium. The first atrial stimulus, however, is followed by ventricular depolarization with a wide QRS complex suggesting ventricular stimulation by the atrial stimulus. The question is asked, are we dealing with a displaced electrode that moves across the tricuspid valve stimulating either the atrium or the ventricle? The answer is found in the last part of the tracing. After the fourth stimulus a ventricular premature beat is shown, just preceded by an atrial stimulus but with a latency between stimulus and QRS complex. This indicates that the ventricular premature beat has no relation to the atrial stimulus. Another argument against ventricular stimulation by the atrial impulse is the morphology of the ventricular premature beat. If this ventricular depolarization was caused by the atrial stimulus, ventricular depolarization would have begun in the right ventricular outflow tract, resulting in a positive QRS complex (direction of depolarization is from superior to inferior), whereas the ventricular depolarization has a negative deflection indicating depolarization from inferior to superior. The latter is in conflict with pacing from the right ventricular outflow tract, thus excluding lead dislodgement.

4.3 Atrial capture in dual chamber pacing

The recognition of appropriate atrial capture in unipolar dual chamber pacemakers may be quite difficult on the conventional electrocardiogram. This problem has already been elucidated in the previous paragraph. In single chamber atrial pacing, however, atrial capture can be confirmed by consistent normal ventricular depolarization following the atrial stimulus by a relatively fixed atrioventricular interval, even when the P wave is obscured by the large unipolar pacing artifact.

In dual chamber systems this assessment is precluded in the absence of AV conduction enabling a 1:1 ventricular response. Van Mechelen et al. [15] reported inability to assess atrial capture on the ECG in unipolar dual chamber pacemakers during follow up in 13 out of 87 patients (15%).

Increase atrial output
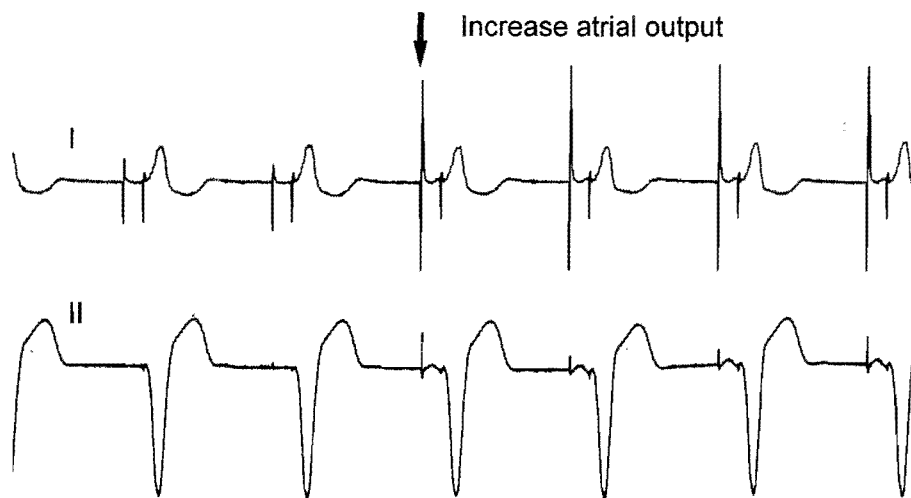
Fig. 4.10. ECG lead I and II of a patient with an implanted DDD pacemaker, showing an isoelectric PR interval in lead I even after increasing atrial output (arrow). Lead II confirms atrial noncapture at low output but effectual stimulation after increasing atrial output. At low output even lead II is suggestive of atrial no output.

In patients with unipolar dual chamber pacemakers and high grade AV block, in whom atrial stimulation is followed by ventricular stimulation with a preset interval, and recognition of atrial capture can be extremely difficult, especially in single lead ECGs. [16].

c   nc rp
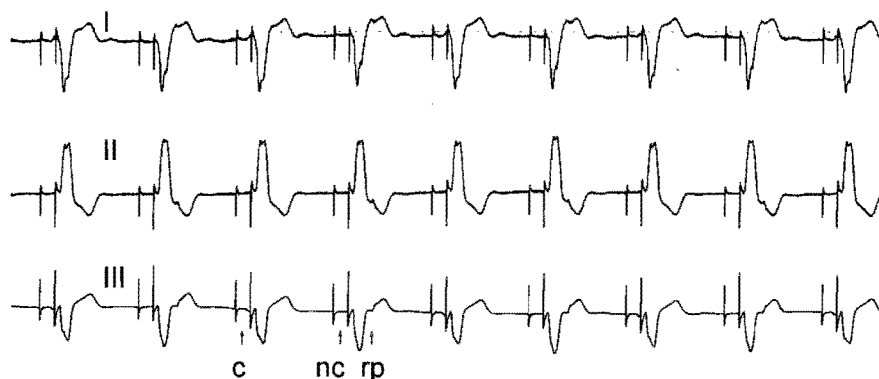
Fig. 4.11. Three lead ECG of a patient with an implanted DDD pacemaker. Atrial capture is hard to identify in all three leads but the presence of retrograde conducted P waves (RP) indicate atrial non capture (NC), while during effective atrial stimulation (C) no retrograde P waves are recorded.

In some cases it is not only the large stimulus artifact that obscures atrial depolarization, but even with relatively low amplitude of the atrial stimulus, the ECG mayshow an isoelectric PR interval suggestive of atrial noncapture

Figure 4.10 demonstrates in the upper tracing (lead I) that there is no evidence of atrial capture even after reprogramming the atrial output pulse to a higher value (arrow). The PR interval remains more or less isoelectric. Lead II however (lower tracing) clearly illustrates that there is no atrial capture at low atrial output, but after increasing atrial output atrial capture is restored. Note that lead II at a low atrial output setting is suggestive of atrial no output.

AV 145 ms

Fig. 4.12. AV sequential pacing with an AV interval of 145 ms. In spite of the short AV interval and effective atrial stimulation, retrograde P waves are present in this patient with an AV nodal bypass tract.

In patients with intact ventriculoatrial (V-A) conduction, presence or absence of retrograde P waves can be an indicator for atrial capture. Appearance of retrograde P waves during AV sequential pacing indicates atrial noncapture, while disappearance of retrograde P waves indicate atrial capture even if effective atrial pacing is not recognized from the ECG. This is demonstrated in figure 4.11. The three lead ECG illustrates intermittent retrograde conduction (RP). Atrial capture is hard to identify in all three leads, in spite of the low amplitude of the atrial stimulus. When atrial capture (C) is present no retrograde P wave is recorded, whereas during atrial noncapture (NC) retrograde P waves are recorded confirming no atrial capture.

AV 140 ms

Fig. 4.13. Same patient as figure 4.12 after shortening the AV interval to 140 ms. Retrograde P waves have disappeared. Only in the third complex where atrial fusion took place (resulting in a prolongation of the PV interval) a retrograde P wave is recorded.

Retrograde conduction is exceptional during effective AV sequential pacing with normal AV intervals. It can be encountered in patients with ventriculo-atrial bypass tracts and even rarely in patients without bypass tracts (see also chapter VII 7.3). Figure 4.12 shows AV sequential pacing with an AV interval of 145 ms in a patient with an AV nodal bypass tract. In spite of the short

AV interval, retrograde P waves are recognized in the T wave. Shortening the AV interval to 140 ms stopped retrograde conduction (Fig. 4.13).

4.3.1 Role of the AV interval in the interpretation of atrial capture

When atrial stimulation is followed by ventricular stimulation with a relatively short AV interval, interpretation of the ECG with respect to atrial capture can be difficult. Latency between the atrial stimulus and atrial depolarization is only an explanation in a minority of patients. Other explanations are low amplitude of the atrial depolarization and the initial direction of atrial depolarization. When the initial direction of depolarization is more or less perpendicular to the recorded ECG lead, initial deflection of atrial depolarization has a very low voltage.

Prolongation of the AV interval may reveal the visible part of the atrial depolarization that is otherwise obscured by ventricular stimulation. This is illustrated in figure 4.14. In the upper tracing (lead I) as well as the lower tracing (lead II), AV interval 120 ms, the PR interval is isoelectric and atrial capture cannot be concluded from this recording. After programming the AV interval to 200 ms, lead I clearly shows atrial depolarization, while in lead II there is still some doubt about the effectiveness of atrial stimulation because the PR interval looks isoelectric. Although in lead II atrial depolarization is still doubtful, it can be concluded from the change of ventricular depolarization that the atrial stimulus is effective. The change of ventricular depolarization is due to effective atrial stimulation and AV conduction leading to ventricular fusion.
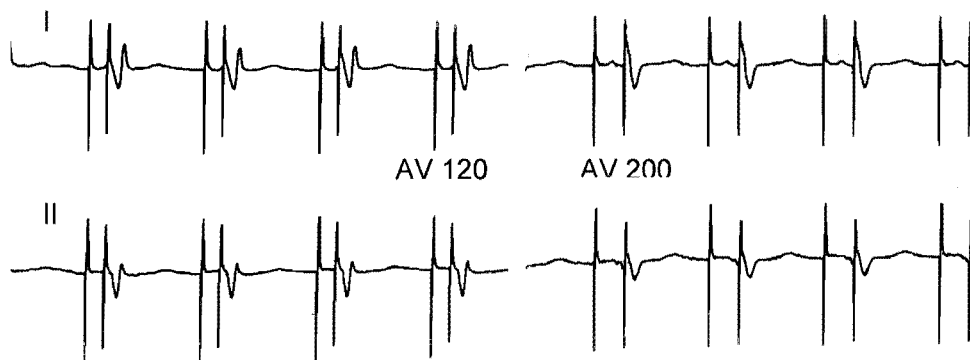
Fig. 4.14. Upper tracing lead I, lower tracing lead II. Atrial capture cannot be concluded when the AV interval is programmed to 120 ms. After programming the AV interval to 200 ms lead I clearly demonstrates atrial capture. Although atrial capture is still difficult to see in lead II after prolongation of the AV interval, the changes in QRS complex suggest effective stimulation leading to ventricular fusion.

4.3.2 Invisible atrial capture on the 12-lead ECG

Occasionally it is impossible to determine whether the large atrial stimulus actually depolarizes the atrium. In this situation the P wave of a paced atrial depolarization should be carefully searched for in the full 12-lead ECG. Evidence of artificially induced atrial depolarization may be quite subtle, with only slight deformity of the decaying overshoot of the atrial stimulus. When the conventional 12-lead ECG completely obliterates any evidence of paced atrial depolarization, the demonstration of effectual atrial capture may require the recording of an ECG at a double

standard (1mV = 2 cm) and/or recording of a right precordial lead at a higher or lower intercostal space. Also the use of non-standard bipolar leads can be helpful in detecting atrial capture. Suggested configurations are: V3R-V3, V4R and right axilla, V3R-epigastrium, and upper left back-epigastrium [17].
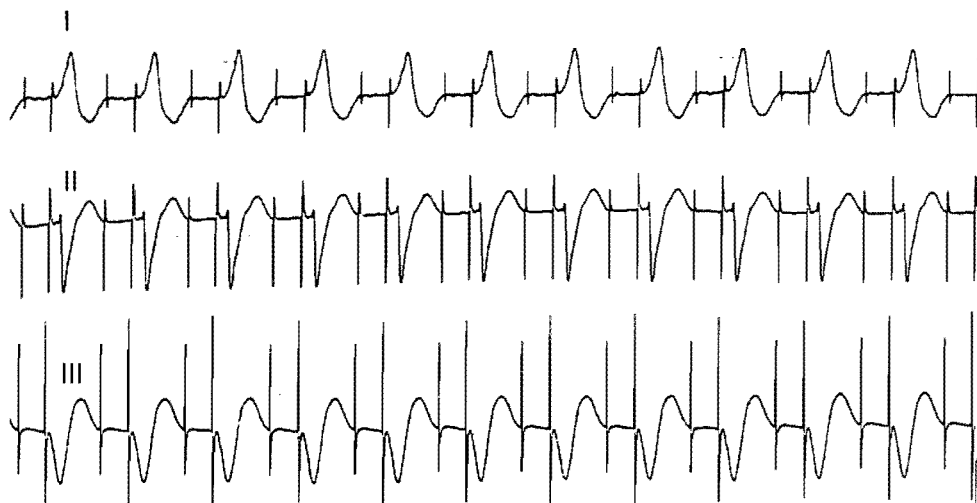
Fig. 4.15. Three lead ECG showing AV sequential pacing at a rate of 80 ppm, AV interval 250 ms. Atrial capture is not clearly visible.
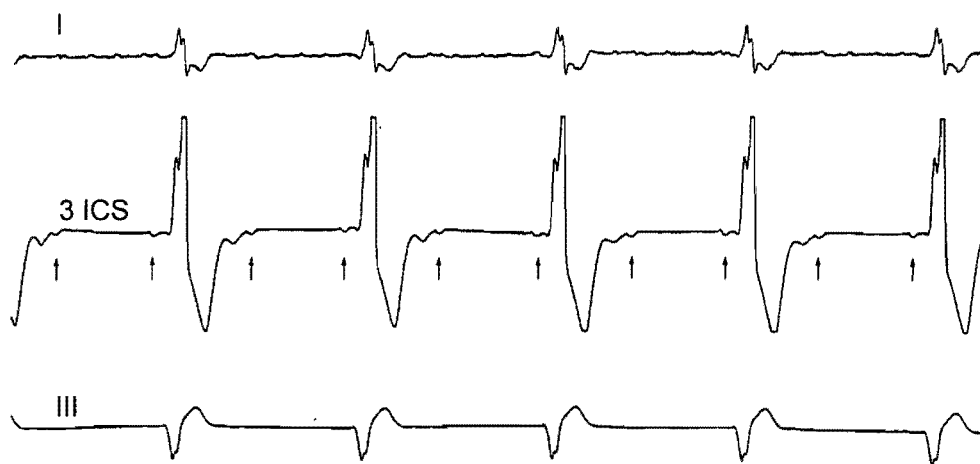
Fig. 4.16. Same patient as figure 4.15 during suppression of the pacing system. Lead I is suggestive of atrial fibrillation. The ECG recorded in the third right intracostal space (3ICS) at double standard, P waves of small amplitude become apparent (arrows).
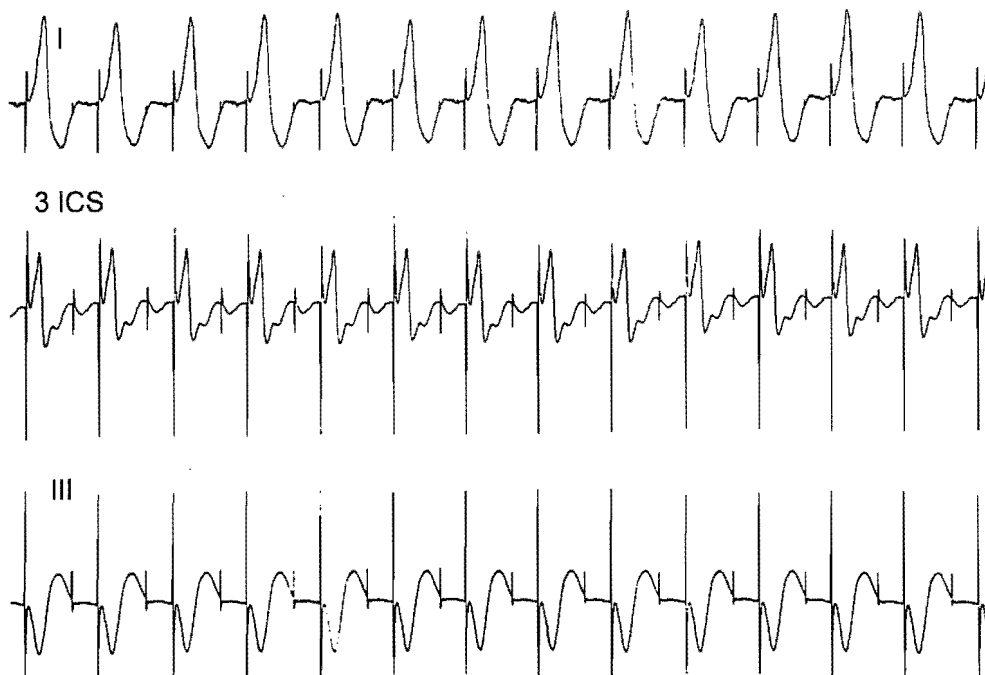
Fig. 4.17. Same patient as figure 4.15 after programming the pacing rate to 80 ppm. The ECG in 3 ICS now reveals atrial capture.
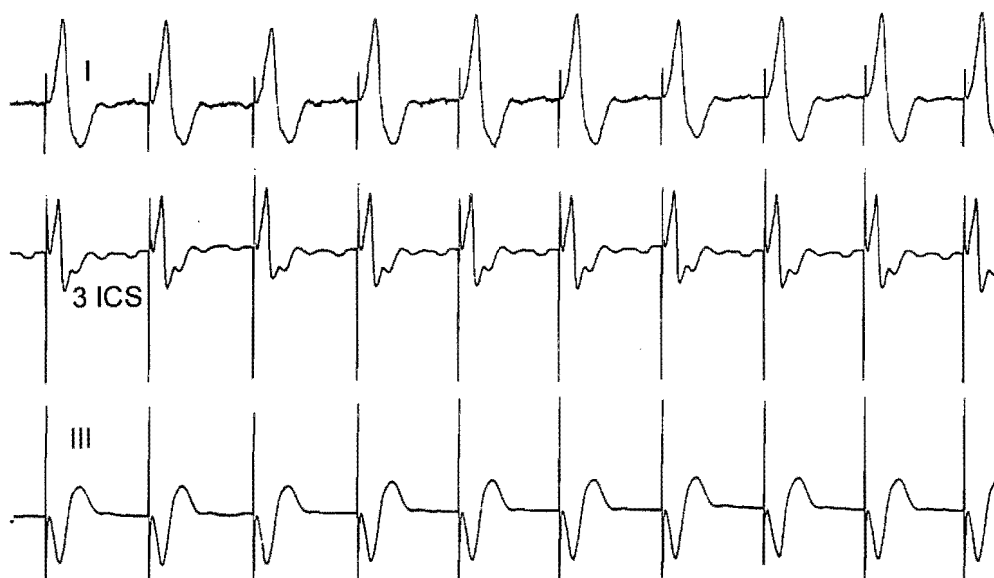
Fig. 4.18. Same patient as in figure 4.15 after decreasing the lower rate below the atrial rate, normal atrial sensing is obvious.

This is best demonstrated in the ECG of figure 4.15. This ECG shows AV sequential pacing at a rate of 80 ppm, the programmed AV interval is 250 ms. It is unclear from this recording whether atrial capture is present or not. The AV interval is isoelectric in lead II and III, while the baseline deflections during the AV interval in lead I are more suggestive of the presence of atrial fibrillation than effective atrial stimulation. In order to exclude the presence of atrial fibrillation, the pacing system was completely suppressed. The ECG showed a regular intrinsic rhythm with a wide QRS complex, without clearly visible atrial depolarization, while some leads were even suggestive of atrial fibrillation (lead I, Fig. 4.16). Simultaneous recording of lead I, lead III and an ECG recorded at the third right intracostal space (3ICR), at a double standard revealed the presence of regular P waves, with a 2:1 AV block (Fig. 4.16). This indicated that the possiblity of atrial noncapture was not due to atrial fibrillation. When the pulse generator was subsequently programmed to its initial rate the ECG recorded in the third right intercostal space at double standard revealed effective atrial stimulation (Fig. 4.17). Note that atrial capture in lead I is still difficult to recognize. Programming the lower rate below the atrial rate showed effective atrial sensing. This is also best demonstrated in lead 3ICR (Fig. 4.18).

Occasionally it might be necessary to use non-standard ECG leads combined with double standard (1mV = 2cm), to confirm the presence of atrial fibrillation. This is illustrated in figures 4.19 - 4.21. During AV sequential pacing successful atrial capture can not be concluded from the recorded ECG (Fig. 4.19). After complete inhibition of the pulse generator, the ECG showed a regular rhythm at a rate of 36 bpm. Atrial depolarization is not visible in this ECG (Fig. 4.20). Recording of the ECG in the third right intracostal space with double standard revealed atrial fibrillation (Fig. 4.21) with low amplitude of the atrial fibrillation waves (1mV = 2cm). This confirms that atrial non-capture was present in figure 4.19.
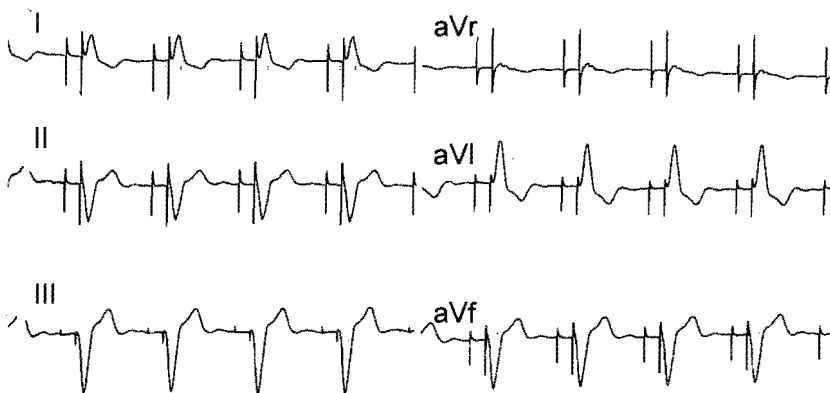
Fig. 4.19. Six lead ECG showing AV sequential pacing at a rate of 70 ppm, AV interval 150 ms. Atrial capture cannot be concluded from this ECG.

Van Mechelen et al.[15] described a method of obtaining rapid and definitive proof of appropriate atrial pacing in dual chamber pacing, by means of reprogramming the pulse generator and recording the ECG. The technique is based on the electrophysiological principles of overdrive suppression of the sinus node and the response of the sinus node to premature atrial stimulation. Resetting of the sinus node pacemaker by an atrial stimulus (even if atrial capture

cannot be seen) demonstrates successful atrial capture. The authors indicated the limitation of this technique in patients with atrio-sinus entrance block, in whom no resetting of the sinus cycle would be present during adequate AV sequential pacing at a slower rate than the spontaneous sinus rate. However, there is another limitation to this technique, in that it is based on resetting of the sinus node pacemaker. Use of the technique is only applicable if intrinsic P waves are visible in the recorded ECG. It has been demonstrated in figure 4.16 that non-standard leads recorded at double standard were necessary to demonstrate the presence of P waves.
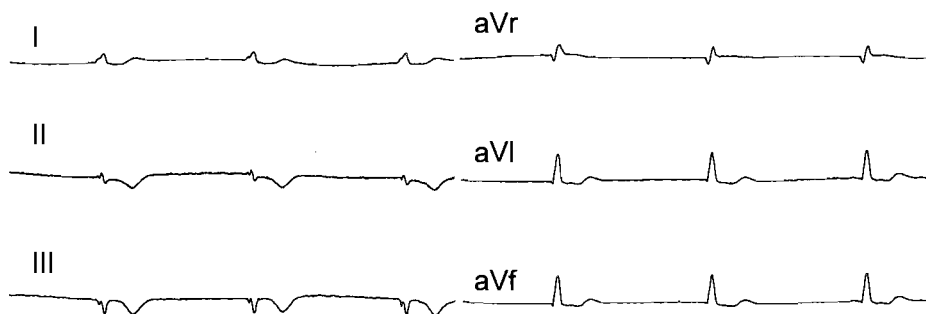
Fig. 4.20. Same patient as figure 4.19 after total inhibition of the pulse generator. The six lead ECG shows a regular rhythm with a wide QRS complex, rate 36 bpm. No P waves are visible.

Fig. 4.21. Same patient as figures 4.19 and 4.20. The ECG recorded in the third right intracostal space at double standard revealed atrial fibrillation, thus confirming the absence of atrial capture in figure 4.19.

In patients with V-A conduction during VVI pacing in whom retrograde P-waves disappear after programming the pulse generator to the DVI or DDD mode, effective atrial pacing can be presumed even though no atrial depolarization is visible.

In patients in whom atrial capture cannot be confirmed using the described techniques, confirmation can be achieved by echocardiography [18,19], recording of esophageal leads or invasively by recording the intracardiac atrial electrogram. Although the latter is an invasive technique, it is the only technique that gives a quick and unequivocal proof of atrial capture.

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4.4 Atrial noncapture caused by ventriculoatrial conduction

Retrograde atrial activation is frequently observed during ventricular stimulation [20]. If ventricular depolarization is preceded by atrial depolarization, as in normally functioning DDD pacemakers, ventriculoatrial conduction is not likely to occur. This is explained by refractoriness of atrial myocardium or the retrograde pathway caused by the preceding atrial depolarization. If, however, the atrial events and the paced ventricular events are sufficiently separated, so that the retrograde pathway is no longer refractory, ventriculoatrial conduction may result. There are several causes of separation of atrial events and paced ventricular events to induce ventriculoatrial conduction in DDD pacemakers (see chapter VII 7.3).

If ventriculoatrial conduction occurs in a dual chamber pacemaker this can result in atrial noncapture [21]. However there are a number of prerequisites to induce atrial noncapture under these circumstances:

1. Induction of ventriculoatrial conduction by separation of atrial and paced ventricular events (mechanism of initiation).

2. A programmed VA interval of the pulse generator, shorter than the sum of the ventriculoatrial conduction time and the effective refractory period of the atrial myocardium. This implies that the atrial stimulus is emitted during atrial refractoriness.

3. Retrograde atrial activation should be ignored by the atrial sensing amplifier (DDD mode). If it is not, the atrial output is inhibited and the phenomenon will not be observed.

Figure 4.22 demonstrates atrial noncapture due to ventriculoatrial conduction, in the first part of the tracing. The interval between retrograde P wave and the following atrial stimulus is approximately 160 ms. After a ventricular premature contraction, which also gave rise to retrograde conduction, the interval between this retrograde P wave and the subsequent atrial stimulus was approximately 240 ms. Because of this longer interval, the atrial myocardium was no longer refractory and atrial capture was restored.
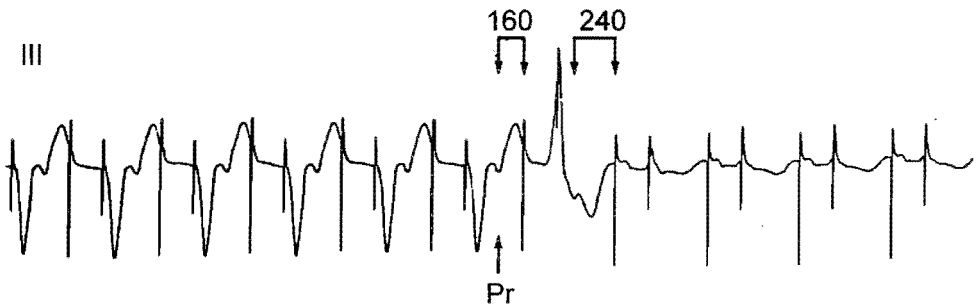
Fig. 4.22. ECG lead III of a patient with an implanted DDD pacemaker showing atrial noncapture in the first part of the tracing. Atrial noncapture is due to retrograde conduction. After a ventricular premature contraction, also associated with retrograde conduction, the coupling interval between this retrograde P wave and the subsequent atrial stimulus is prolonged to 240 ms. After this interval the atrium is no longer refractory and atrial capture is restored.

As explained under (2) the relative short VA interval is a major factor in this phenomenon. Relative short VA intervals are created by programming a high lower rate and/or a long AV

interval. Although this is a rarely used combination in DDD pacing, increase of the lower rate is one of the aims of rate responsive DDD pacemakers. This implies that this phemonon can be induced more easily in these devices [22].

4.5 Cross stimulation

Cross stimulation is defined as stimulation of one cardiac chamber through a lead placed in another chamber. In practice cross stimulation will mainly be observed as ventricular pacing from the atrial lead. Reports on the electrocardiographic phenomenon illustrating ventricular pacing by the atrial stimulus can be divided into four groups.

1.   The morphology of the QRS complex caused by the atrial stimulus is identical to the morphology of the QRS complex caused by the ventricular stimulus.
2.   The morphology of the QRS complex caused by the atrial stimulus differs from the QRS complex caused by the ventricular stimulus.
3.   The atrial stimulus causes ventricular pacing while the ventricular stimulus captures the atrium.
4.   Pseudoventricular capture by the atrial stimulus due to the recording equipment. See section 4.5.4

Under bizarre circumstances a form of cross stimulation, where the ventricular as well as the atrial impulse stimulates the atria, is observed.

4.5.1. Morphology of the QRS complex caused by the atrial stimulus is identical to the QRS complex during ventricular stimulation

When the morphology of the QRS complex caused by the atrial stimulus is identical to the QRS complex initiated by ventricular pacing, it indicates that there is cross stimulation through the pacing system without dislocation of the atrial lead. This has been reported by Doi et al. [23] and Puglisi et al. [24] in a series of Siemens 544 unipolar dual chamber pacemakers. Both authors observed the phenomenon only temporarily immediately after implantation and it ended after two weeks. It was suggested that the atrial stimulus produced a "small" current from the atrial to the ventricular electrode, that became clinically significant in the presence of a very low ventricular threshold and/or high atrial output, resulting in cross stimulation. The increase in ventricular stimulation threshold and the increase in impedance at electrode-tissue interface after implantation explained why the cross stimulation disappeared after a short period of time.

Irwin et al [25], however, reported cross stimulation in a series of Pacesetter 283 DDD pacemaker (which model is electronically identical to Siemens 544) up to 52 months after implantation. They also reported that cross stimulation was not only related to high atrial output but also to low ventricular output. An explanation for the latter was not given.

Goldschlager and Francoz [26] reported on cross stimulation using a pacing system analyzer. In their cases bipolar leads were used for atrial and ventricular pacing. In the stimulator atrial and ventricular anodes were not separated but connected to a common ground within the analyzer, each through a resistance of approximately 15 Ohms. As a result, atrial and ventricular anodal electrodes are separated from each other by only 30 Ohms. This implemented that the atrial stimulus delivered at the atrial cathodal electrode not only resulted in a current towards the atrial anodal (ring) electrode but also to the ventricular anodal electrode. Under these conditions employing a high atrial output cross stimulation could easily be provoked. Although cross stimulation in this setting is usually of no clinical significance, recognition is of considerable

importance in order to avoid a mistaken diagnosis of lead migration or loss of integrity, resulting in inappropriate pacing system explanation and/or unnecessary prolongation of the implantation procedure. The correct diagnosis can be made by verifying absence of atrial lead migration fluoroscopically and by documenting the similarity of paced QRS complexes to those resulting from pacing through an existing, properly positioned ventricular lead.

Levine et al.[27] reported cross stimulation in Intermedics Cosmos (model 283-01) unipolar dual chamber pacemakers during magnet application. The explanation for this observation is that a ventricular discharge switch designed to minimize afterpotential sensing remains closed during the first five asynchronous cycles at the magnet rate of 90 ppm. This allows an internal cross-over of energy from the atrial output capacitor to the ventricular electrode. If the ventricular stimulation threshold is very low, ventricular capture may result. Following these five cycles, the ventricular discharge switch is opened, thus terminating this internal communicating circuit. This form of cross stimulation is a self-limiting clinically benign phenomenon that resolves with electrode maturation and increase in ventricular stimulation threshold.

A type of cross stimulation where cathodal contact between the atrial and ventricular lead played a role was reported by Anderson and Nathan [28]. They described a patient with a pacemaker-twiddler's syndrome [29] after implantation of a dual chamber pacemaker. Both leads were extensively entwined, which resulted in degradation of the insulation of the leads as evidenced by reduced lead impedance. The contact between the damaged leads enabled sufficient current leakage for ventricular capture to occur from a pacing spike emitted in the atrial channel.
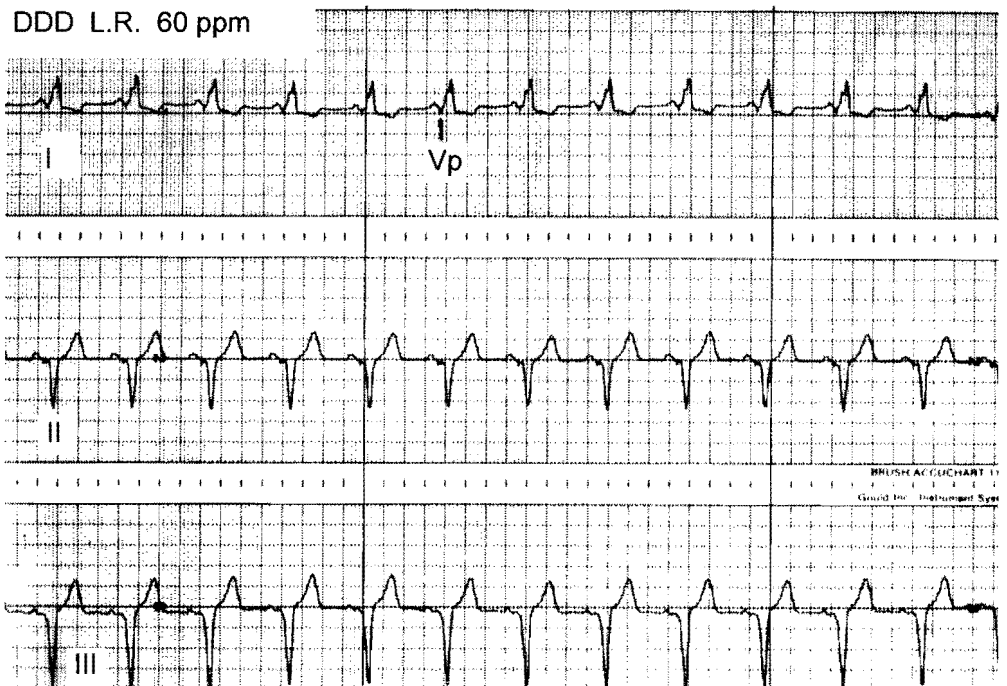
Fig. 4.23. Three channel ECG (lead I, II and III) of a patient with a bipolar DDD pacemaker; lower rate 60 ppm, AV interval 150 ms. ECG shows atrial sensing ventricular pacing (Vp).

4.5.2. Morphology of the QRS complex caused by the atrial stimulus differs from the QRS complex by ventricular stimulation

When the morphology of the QRS complex caused by an atrial stimulus of a dual chamber pacemaker is **not** identical to the QRS complex during ventricular pacing, it is suggestive of dislodgement of the atrial lead [30,31]. J-leads implanted in the right atrial appendage can easily migrate across the tricuspid valve into the right ventricular outflow tract after dislocation.

Being in contact with the ventricular endocardium emission of an atrial impuls may result in ventricular capture. The morphology of this QRS complex will differ from the QRS complex during ventricular pacing, provided that regular ventricular pacing is performed from the right ventricular apex. Although atrial J-leads employing passive fixation are more vulnerable to dislodgement than active fixation electrodes, Nathan and Camm [32] reported displacement of such an electrode. This electrode displaced after 6 months into the right ventricle with resulting electrocardiographic cross stimulation.
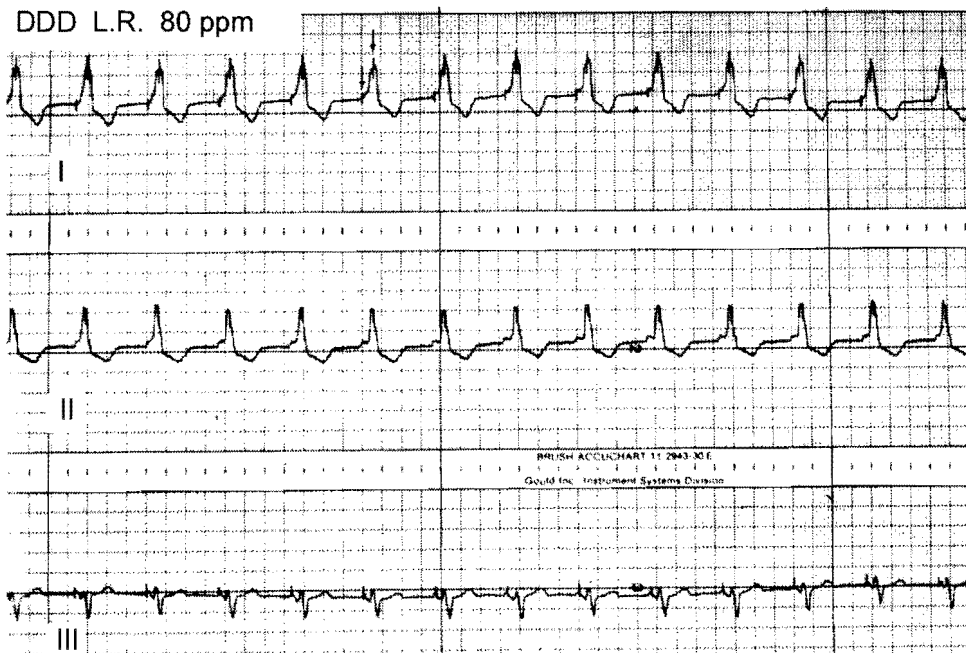
Fig. 4.24. Three channel ECG (lead I, II, III) of the same patient as figure 4.23, after programming the lower rate to 80 ppm, showing two consecutive stimuli with a coupling interval of approximately 100 ms. The first (atrial) stimulus depolarizes the ventricles, while the second (ventricular) stimulus occurs during the QRS complex. Note the change in ventricular depolarization compared to figure 4.23, indicating that direction of ventricular depolarization altered from superior to inferior (positive QRS complex in lead II).

We encountered cross stimulation in a patient with a DDD pacemaker after programming the upper rate from 60 to 80 ppm. In the initial setting with a lower rate of 60 ppm atrial synchronous ventricular pacing was recorded (Fig. 4.23). When the lower rate was programmed to 80 ppm the ECG represented in figure 4.24 was recorded. It shows AV pacing at a rate of 80

ppm, where every atrial stimulus depolarized the ventricle followed by a ventricular stimulus with an interval of approximately 100 ms (programmed AV delay 150 ms), falling in the absolute refractory period of the ventricular myocardium. Fluoroscopy revealed dislocation of the bipolar atrial lead, that migrated across the tricuspid valve into the right ventricular outflow tract (Fig. 4.25).
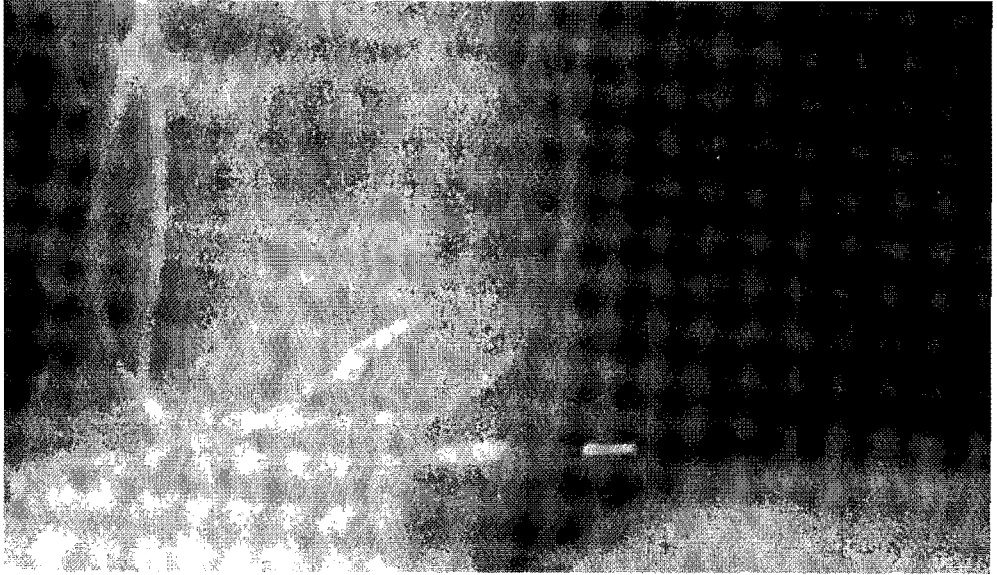
Fig. 4.25. Antero-posterior X-ray confirming dislocation of the atrial lead. The distal end has migrated across the tricuspid valve.
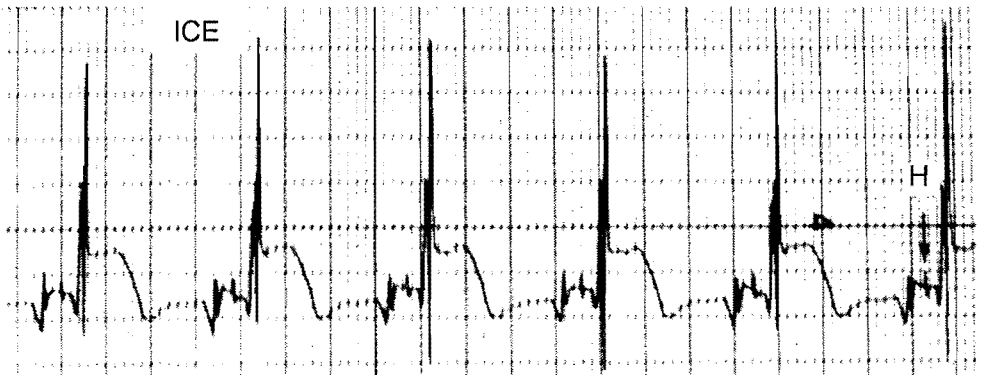
Fig. 4.26. Bipolar intracardiac electrogram recorded from the dislodged atrial lead showing atrial and ventricular depolarization. Due to its position across the tricuspid valve even the His bundle potential (H) could be recorded.

The intracardiac electrogram recorded from the dislodged lead showed not only ventricular activity but also atrial activity and even the His bundle potential was visible in this recording (Fig. 4.26). The presence of atrial depolarization of sufficient amplitude in the intracardiac electrogram explained the fact that normal atrial sensing ventricular pacing was observed when the lower rate was programmed below the patients intrinsic rate.

The morphology of the QRS complex during cross stimulation had already indicated that the ventricles were depolarized by the atrial electrode since depolarization took place from superior to inferior; this is in contrast with right ventricular apical pacing that induces depolarization from inferior to superior. The absence of spontaneous P waves during AV pacing at a rate of 80 ppm, supports the hypothesis that the atrial stimulus is not only pacing the ventricle but the atrium as well.

The shortening of the AV interval from 150 to 100 ms (Fig. 4.24) and the phenomenon of double ventricular pacing is due to the ventricular safety pacing modality (chapter VI 6.5.2) of this particular unit. In a situation like this, if the stimulus from the atrial channel did not capture the ventricle while still being sensed by the ventricular amplifier, safety pacing would ensure ventricular pacing. In pulse generators without safety pacing, only one pacemaker stimulus from the atrial channel can be observed which also can be sensed by the ventricular amplifier, thus causing ventricular inhibition.

If the timing circuit of these pacemakers is ventricular based, the pacing rate increases. The effective pacing interval is equal to the programmed lower rate minus the programmed AV interval (= VA interval).

4.5.3. Atrial stimulus causes ventricular stimulation while the ventricular stimulus captures the atrium

A phenomenon that is rarely reported [33] and is related to the reversal of leads due to incorrect connections. Although this will be easily recognized by bizarre looking ECGs, it can be overlooked when pacemakers are implanted in patients that at the time of implantation have an intrinsic rhythm which completely inhibits the pacing system. Before connecting the leads to the pulse generator the identity (atrial or ventricular position) of the lead can be verified by recording the intracardiac electrogram or by stimulation from the implanted lead.

4.5.4. Pseudo ventricular capture by the atrial stimulus due to recording equipment.

Fusion or confusion on Holter recording
L.M. van Gelder, F.A.L.E. Bracke, and M.I.H. El Gamal
PACE 14; 760-763, 1991.

Summary
Holter recording of a patient with an implanted dual chamber rate responsive pacemaker revealed an electrocardiogram, where ventricular depolarization seemed to be initiated by the atrial stimulus. In a second patient with a VVI pacemaker, Holter recording showed delay of the pacemaker impulse that was registered after the onset of ventricular depolarization. Misalignment in one of the recorder heads of the display system was responsible for this phenomenon, which in case of dual chamber pacing could have been easily misinterpreted as pacemaker malfunction.

Introduction

Holter monitoring is an important tool in the evaluation of pacemaker function [1-3]. Several systems are in use to improve the quality of Holter recordings obtained from pacemaker patients. They all have one goal in common, increase visibility of the pacemaker impulse in order to analyze pacemaker function [4]. New Holter equipment was recently installed in our department (CardioData, Prodigy, CardioData Corp., Nothboro, MA, USA). This report describes confusing electrocardiographic findings, obtained by this new equipment, in two Holter recordings of patients with implanted pacemakers.

Case report

Patient 1.

Patient 1 was a 22-year-old male, suffering from sinus bradycardia and AV conduction disturbances. A rate responsive DDD pacemaker (Medtronic Synergist 7070, Medtronic, Inc., Minneapolis, MN, USA) was implanted in March, 1989. The pacing system was functioning properly, according to clinical and outpatient checkup. A 24-hour Holter recording showed the electrocardiogram represented in Figure 4.27. This electrocardiogram revealed an atrial and ventricular impulse with intervals corresponding to the programming of the pulse generator (lower rate 80 ppm, AV interval 150 msec.) The timing between impulses and ventricular depolarization suggested that ventricular depolarization was initiated by the atrial impulse, followed by a ventricular impulse in the absolute refractory period. Speculation about the cause brought up the possibility of atrial lead displacement. Patient was called for further examination and control of the stimulation system. This checkup did not reveal any pacemaker malfunction. Stimulation and sensing threshold values were all satisfactory. Patient was monitored by clinical telemetry while he was walking and performing physical exercise.

There were no abnormalities in the pacing system, pacing rate responded very well to the amount of exercise. During exercise there was exclusively AV sequential pacing rhythm with rate increase up to 115 ppm depending on the degree of exercise. No arrhythmias were observed (Fig. 4.28). Chest X ray showed normal atrial and ventricular lead position.

HR = 79   21.45,0-·
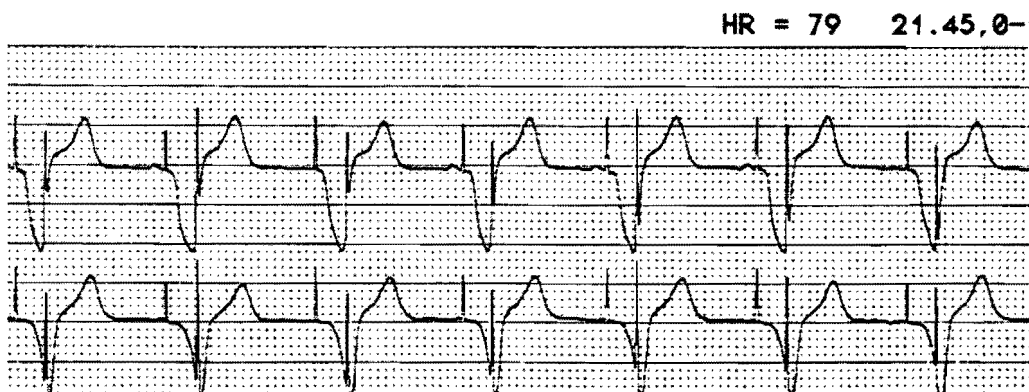
Fig. 4.27. Holter recording of patient 1, with a dual chamber pacemaker showing atrial and ventricular impulses, suggesting ventricular depolarization by the atrial impulse.

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Patient 2.

The solution of the problem was found in the Holter recording of the second patient. This patient was a 75-year-old female. She had a VVI pacemaker since 1972 and had exclusively pacemaker rhythm. The Holter recording, however, showed at rest a rhythm with a wide QRS complex and left bundle branch block configuration. There was also a pacemaker impulse visible approximately 60 ms after initiation of the QRS complex (Fig. 4.29). Simultaneous recording of Holter and standard electrocardiogram showed normal pacing rhythm on routine electrocardiography (Fig. 4.30).
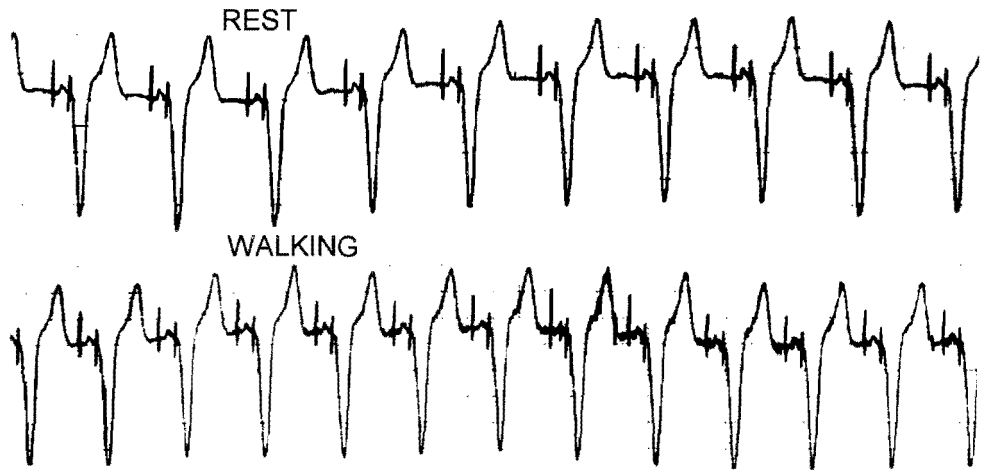
Fig. 4.28. ECG of patient 1 showing normal AV pacing during rest and exercise.

This implicated that the Holter equipment delayed the pacemaker impulse producing a tracing suggestive of pseudofusion in this patient. The delay between myocardial depolarization and the
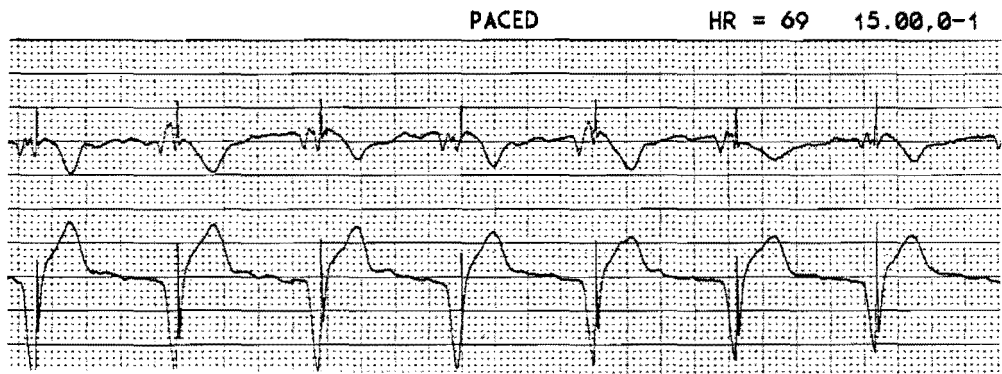
Fig. 4.29. Holter recording of patient 2, with a ventricular pacemaker suggesting ventricular pseudofusion.

pacemaker impulse also explained the phenomenon observed in the Holter recording of the first patient. Later Holter recordings of different pacemaker patients also showed the same phenomenon, confirming that the problem was structural and related to the display and recording technique of the equipment.
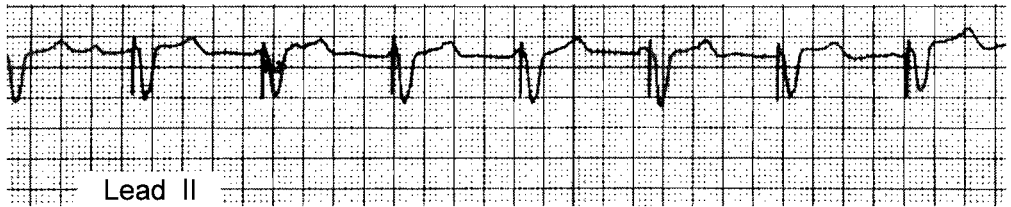
Fig. 4.30. ECG lead II of patient 2 showing normal ventricular pacing.

Discussion

Holter equipment has limitations in regard to analysis of pacemaker patients. The pacemaker impulse is filtered and clipped when it is recorded. The recorded spike is not only difficult to identify, but also distorts the underlying ECG. As a result, analysis systems cannot accurately identify the existence of paced pulses or have sensible recognition of the QRS complexes [5].

To overcome these problems in Holter equipment (CardioData Corp.), the pacemaker artifact is detected unequivocally by the recorder electronics and its presence with correct timing is recorded on a separate track. A specially designed circuit further eliminates the pacemaker impulse from the electrocardiogram. The analyzer then reconstitutes the pacemaker artifact and adds it to both ECG channels from screen display and printout.

Addition of the previously separated ECG and pacemaker impulse was responsible for the electrocardiographic phenomenon described in our two patients. Misalignment in one of the recorder heads caused a time difference between pacemaker impulse and depolarization, thus creating an ECG with a delay between depolarization and the stimulation impulse. The Holter recording of patient 1 in whom a dual chamber pacemaker was implanted, was suggestive of dislocation of the atrial lead into the ventricle because ventricular depolarization followed the atrial spike, with the ventricular impulse falling in the absolute refractory period. The recording of patient 2 with a pacemaker spike falling 60 msec after initiation of a wide QRS complex clearly indicated malfunction of the Holter recording system. When the rhythm strips are closely examined, a variation is found in the intervals between A and V stimuli in patient 1, and the V-V intervals in patient 2, with respect to the actual QRS complexes. The finding of this nonconstancy between pacing stimulus and QRS interval should alert the clinician to the problem. Repair of the alignment in the recorder head solved the problem. Unexplained timing intervals between pacemaker depolarization and pacemaker impulse in Holter recordings, employing the system described above, should be considered as a failure of the recording system. In case of dual chamber pacing, ECGs can be confusing and interpreted as pacemaker malfunction.

References
1. Jacobs LJ, Kerzner JS, Diamond MA, et al. Pacemaker inhibition by myopotentials detected by Holter monitoring. PACE 1982;5:30-33.

2. *Secemsky SI, Hauser RG, Denes P, et al. Unipolar sensing abnormalities: Incidence and clinical significance of skeletal muscle interference and undersensing in 228 patients. PACE 1982; 5:10-19.*
3. *Van Gelder LM, El Gamal MIH. Undersensing in VVI pacemakers detected by Holter monitoring. PACE 1988; 11:1507-1511.*
4. *Kelen GJ, Bloomfield DA, Hardage M, et al. A clinical evaluation of an improved Holter monitoring technique for artificial pacemaker function. PACE 1980; 3:192-197.*
5. *Feldman CL, Hubelbank M. Analysis of ambulatory ECG's for patients with dual chamber pacemakers. IEEE 1984. Proceedings of Computers in Cardiology. Salt Lake City, Utah, September 18-21, 1984.*

4.5.5. Atrial stimulation from the ventricular and the atrial channel

One of the problems connected with pacemaker implantation has been the correct positioning of the electrode tip in the apex of the right ventricle.

DVI 103 ppm
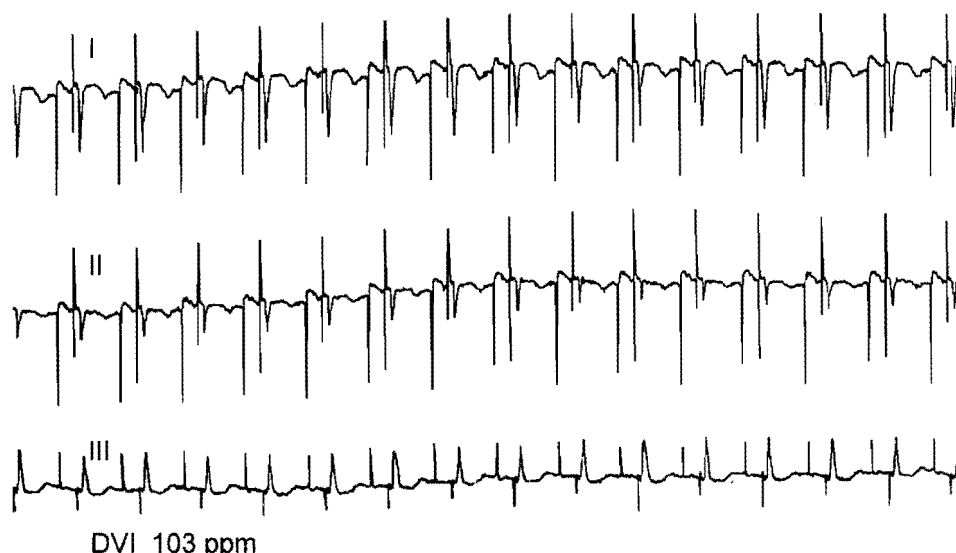
Fig. 4.31. Three channel ECG showing DVI pacing at a rate of 103 ppm. There is atrial capture with a positive P wave in lead II and ventricular pseudofusion.

If the electrode is passed directly towards the tricuspid valve, it may enter the coronary sinus, a position that on the posteroanterior projection may mimic the right ventricular apex [34]. In the posteroanterior projection the anatomic position of the electrode may appear to be satisfactory when in truth, the electrode lies in the middle cardiac vein. Howerver, a satisfactory ventricular pacing threshold can be obtained with the electrode in this position. Malplacement into the coronary sinus can be prevented by passage of a loop through the tricuspid valve, and by passing the electrode tip completely into the pulmonary artery. Then, upon withdrawal of the electrode, the tip will lie in the midventricle, where it can be advanced to the area of the apex.

Placement of the electrode into the coronary sinus can be recognized from the ECG during ventricular pacing with a resulting right bundle branch block pattern indicating left ventricular stimulation [35]. Lateral fluoroscopy will show the electrode to be posterior instead of anterior.

The ECG shown in figure 4.31 was recorded from a patient with a DVI pacemaker. It shows AV sequential pacing at a rate of 103 ppm with atrial capture and ventricular pseudofusion. After

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programming the pulse generator to the VVI mode, the ECG showed atrial pacing with a 1:1 ventricular response. Careful analysis of the ECG, however, showed negative P waves in lead II and III, indicating inferior-superior depolarization of the atrium (Fig. 4.32). Anteroposterior (Fig. 4.33) and lateral (Fig. 4.34) X-rays of the patient revealed a normal position of the atrial electrode in the right atrial appendage. The ventricular lead however, was positioned in the coronary sinus, clearly illustrated in the lateral view. This explained atrial pacing after programming in the VVI mode.

VVI 103 ppm
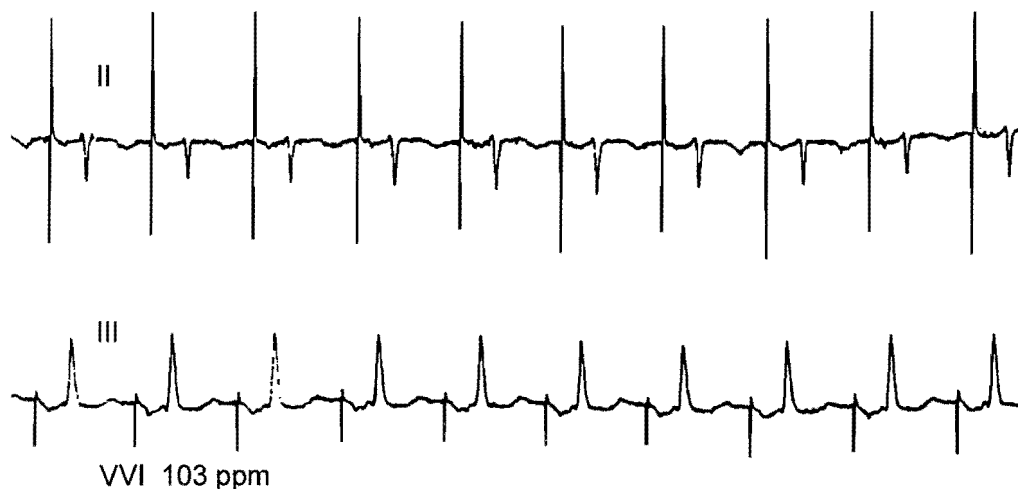
Fig. 4.32. Two channel ECG (lead II and III) of the same patient as figure 4.31 after programming to VVI mode. The ECG shows atrial pacing at a rate of 103 ppm with a 1:1 ventricular response. Note the negative P waves in lead II and III.

Fig. 4.33. Anteroposterior X ray of the patient described in figures 4.31-4.32. Note the slightly upward position of the ventricular lead.

Fig. 4.34. Lateral X ray of the same patient showing the posterior position of the ventricular lead in the coronary sinus.

Permanent atrial pacing from the coronary vein has been published in the late seventies [36,37], but after introduction of reliable atraumatic atrial [38] and active fixation leads [39,40] this method was abandoned. The ECG shown in figure 4.35 is presented as a curiosity. It shows AV sequential pacing with two stimuli at different strengths, 5 and 12 mA respectively. Both stimuli are applied to one bipolar

S1: 5 mA   S2: 12 mA   S1 S2
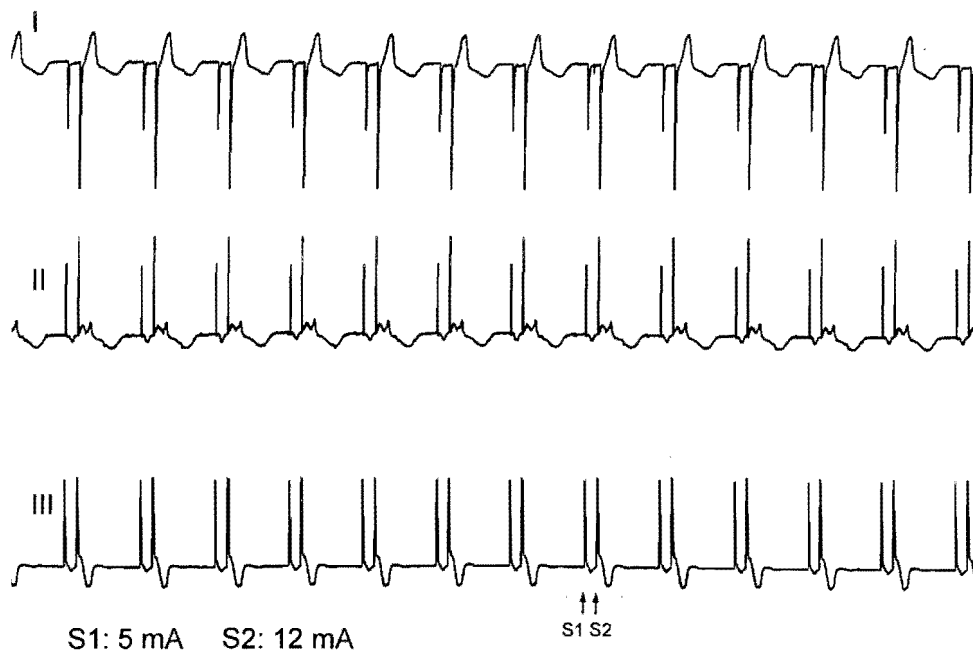
Fig. 4.35. Three channel ECG (lead I,II,III) of a patient with a bipolar lead positioned in the coronary sinus. Two stimuli, different in strength are applied to the same lead. The 5 mA stimulus depolarizes the atria while the ventricles are captured by a 12 mA stimulus.

lead that was positioned in the coronary sinus. Because the pacing threshold for ventricular stimulation was higher than for atrial stimulation, the 5 mA stimulus depolarized the atrium while the ventricles were depolarized by a 12 mA stimulus applied to the same lead.

4.6 Ventricular capture
Recognition of ventricular capture depends on visibility of the pacemaker stimulus as well as the following ventricular depolarization and repolarization. Under normal circumstances pacemaker stimuli are easily recognized from the ECG. It might be more difficult in bipolar systems combined with a low output setting of the pulse generator and in unipolar systems, where the direction of stimulation current is more or less perpendicular to the recorded ECG lead. Using a high-quality analog ECG machine produces the best information about presence, absence, size and direction of the pacemaker stimulus.

4.6.1 Pseudo ventricular noncapture

Ventricular depolarization and repolarization resulting from a ventricular stimulus are easily recognized. However, pseudo-malfunction has been reported from ECG machines which have an automatic instow. The automatic instow allows the stylus to return to the baseline when changing leads. The stimulus artifact will also operate the automatic instow so that the stylus will return to the baseline and remain inoperable for about 400 ms. Thus no QRS or T wave is recorded after the stimulus artifact, giving the impression of no capture [41].
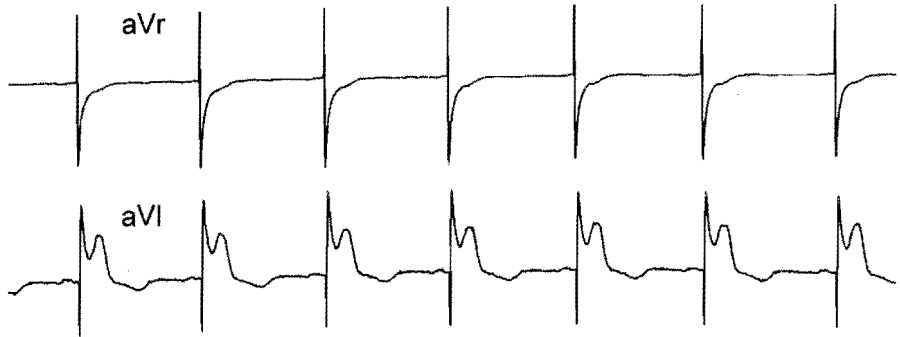
Fig. 4.36. Two channel ECG representing pseudo noncapture in the upper tracing (lead aVR), while the lower tracing (lead aVL) demonstrates effectual stimulation.

The ECG pattern is similar to the ECG of figure 3.1, chapter III. A large unipolar pacemaker stimulus may occasionally mask the paced QRS complex because the voltage-decay waveform may distort or even conceal the paced QRS complex following pacemaker discharge. This might be related to the inability of some ECG machines to process a large signal through the amplifier. The presence of a subsequent T wave confirms that true ventricular depolarization occurred.
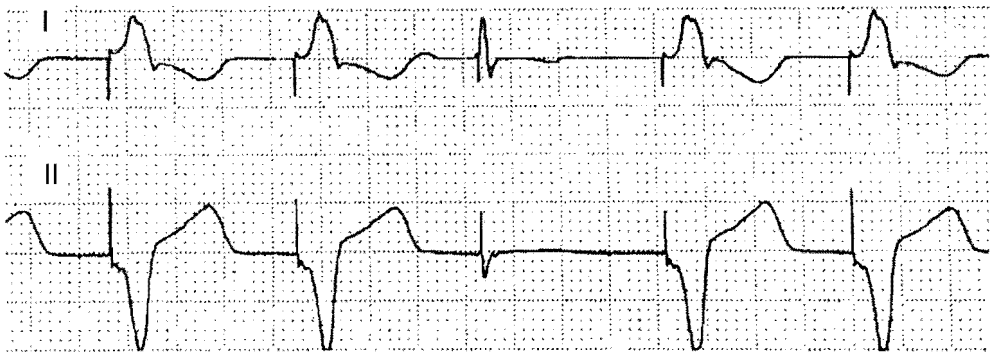
Fig. 4.37. Monitor lead I (upper tracing) and II (lower tracing) of a patient with an external VVI pacemaker. The third pacemaker stimulus shows a pattern of noncapture in lead II, whereas lead I illustrates pseudofusion.

Figure 4.36 illustrates this phenomenon. The upper tracing (lead aVR) suggests the presence of ineffectual pacemaker stimuli because the QRS complex and T wave are masked by the voltage-

decay waveform following the pacemaker stimulus. Presence of de- and repolarization is clearly demonstrated in the simultaneously recorded lead aVL (lower tracing).Ventricular pseudofusion can easily be misinterpreted as noncapture. Pseudofusion might result in an isoelectric complex in one ECG lead, even without the visible presence of a T wave. Figure 4.37 shows a monitor lead corresponding with lead II. The first and second stimulus are followed by ventricular depolarization. The third stimulus, however, suggests noncapture. A simultaneously recorded monitor lead corresponding with lead I reveals the presence of a spontaneous QRS complex and a pseudofusion pattern thus excluding non capture (Fig. 4.37).

4.6.2 Ventricular pseudocapture

When ineffectual pacemaker stimuli precede a spontaneous QRS complex, especially when left bundle branch block (LBBB) is present, and the rates are interlocked, the constant relationship between ineffectual stimulus and spontaneous QRS complex may be misinterpreted as successful ventricular capture. Long rhythm strips and variations of the spontaneous rate should prevent this serious misinterpretation and make the diagnosis of pacemaker failure obvious [42].

DDD L.R. 60 ppm
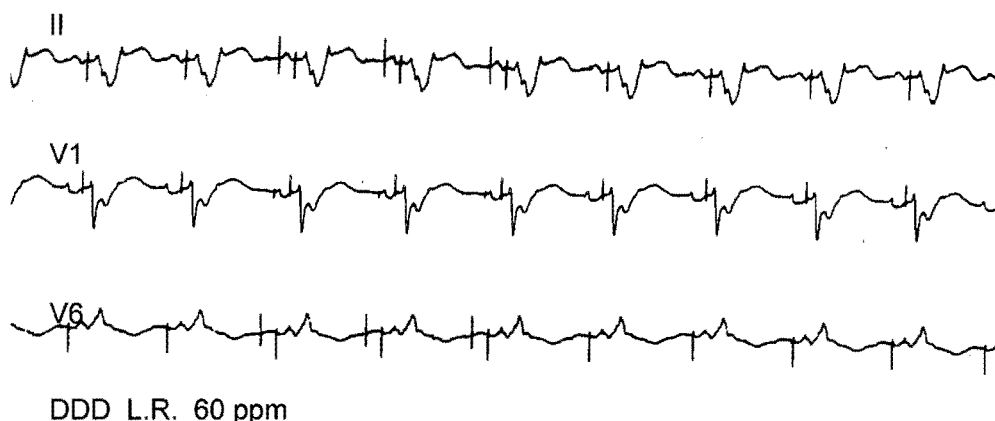
Fig. 4.38. Three channel ECG of a patient with an implanted DDD pacemaker. There is normal atrial pacing and sensing and every ventricular stimulus is followed by depolarization with LBBB configuration suggesting effective ventricular stimulation.

Recording long rhythm strips can clarify this problem in single chamber systems, where the pacing rate has a fixed interval, but it might be more complicated in dual chamber systems. In DDD pacemakers, the ventricular pacing interval is determined by the preceding atrial event. This implies that the ventricular pacing interval is not fixed but varies with the atrial rate. Figure 4.38 shows a three channel ECG (lead II,V1,V6) of a patient with a DDD pacemaker. The rhythm shows atrial sensing-ventricular pacing and atrial pacing-ventricular pacing. All ventricular stimuli are followed by ventricular depolarization. Ventricular depolarization has an inferior-superior direction in the frontal plane (negative in lead II) and a LBBB configuration (Lead V1,V6) compatible with right ventricular apical pacing. One could conclude from this recording that there is normal function of the pacing system with effective ventricular stimulation. After programming the pulse generator to the VVI mode (Fig. 4.39) with the same ventricular output it is demonstrated that there is no ventricular capture and no ventricular

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sensing. Note that the morphology of the spontaneous QRS complex is the same in the DDD mode. So it must be concluded that ventricular pacing was not effective in the ECG presented in figure 4.38. Ventricular noncapture and undersensing was caused by an ineffective connection of the ventricular lead to the pulse generator (Fig.4.39).

II

V1

V6

VVI 40 ppm

Fig. 4.39. Same patient as figure 4.38 after programming the pulse generator to the VVI mode at the same ventricular output. The ECG reveals ventricular noncapture and ventricular undersensing. Note the morphology of the QRS complex is identical to figure 4.40, thus confirming ventricular noncapture.
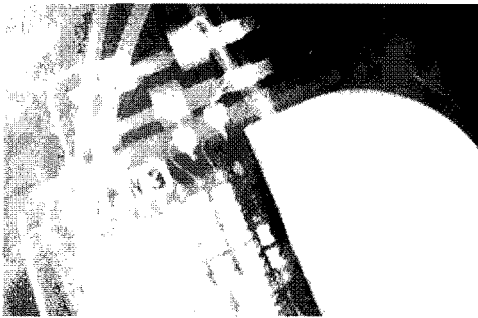
Fig. 4.40. X-ray of the pulse generator (ECGs figs. 4.38, 4.39) showing inappropriate connection of the ventricular lead (lower lead), which was not advanced far enough into the connector. Compare the position of the atrial lead (upper lead) that was properly connected.

One can argue that the ECG of figure 4.38 was already suspicious for ventricular noncapture because of the delay between the ventricular stimulus and the following QRS complex. However, a delay from the pacing stimulus to the onset of ventricular depolarization is a known phenomenon in cardiac pacing and is called latency. Figure 4.41 illustrates the delay between pacemaker stimulus and ventricular depolarization, also in a patient with a DDD pacemaker. Delay is present in all recorded standard leads and the QRS complex gives the impression of being narrow, suggestive of ventricular noncapture. Recording of a precordial lead (V2, Fig. 4.42) simultaneously with lead I and III elucidate no delay between the ventricular stimulus and QRS complex. The QRS complex is wide (0.16 s) demonstrating effective ventricular pacing

4.6.3 Intermittent ventricular noncapture
An electrical stimulus emitted by the pacemaker not followed by depolarization is named noncapture. Noncapture can be observed for each stimulus, total loss of capture, or for some

stimuli, intermittent loss of capture. Intermittent loss of capture can occur under two circumstances. One with an unstable electrode endocardial interface, usually observed shortly after implantation and related to a varying contact between electrode and endocardium. The other in a stable system if the output of the pulse generator is close to the stimulation threshold. Causes of noncapture will be discussed in paragraph 4.8 of this chapter. Capture only in the supernormal phase of excitability suggests a stimulation threshold close to the output of the pulse generator, but gives no clue as to the etiology of the increased threshold [44,45].
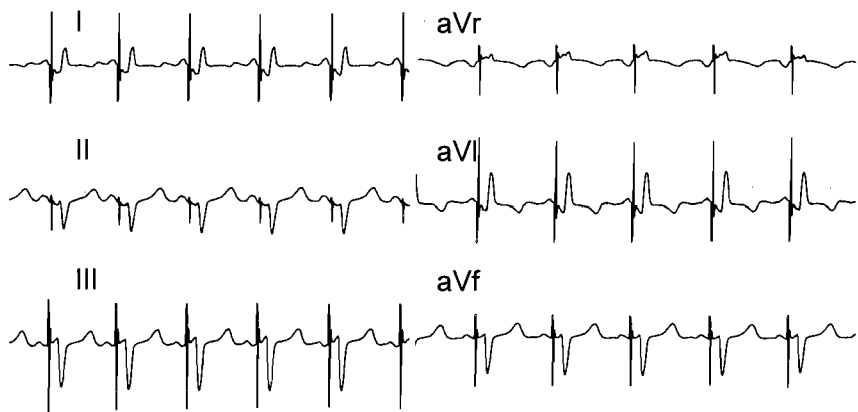
Fig. 4.41. Six channel standard leads ECG in a patient with a DDD pacemaker showing a delay between ventricular stimulus and QRS complex. Pseudo ventricular capture?
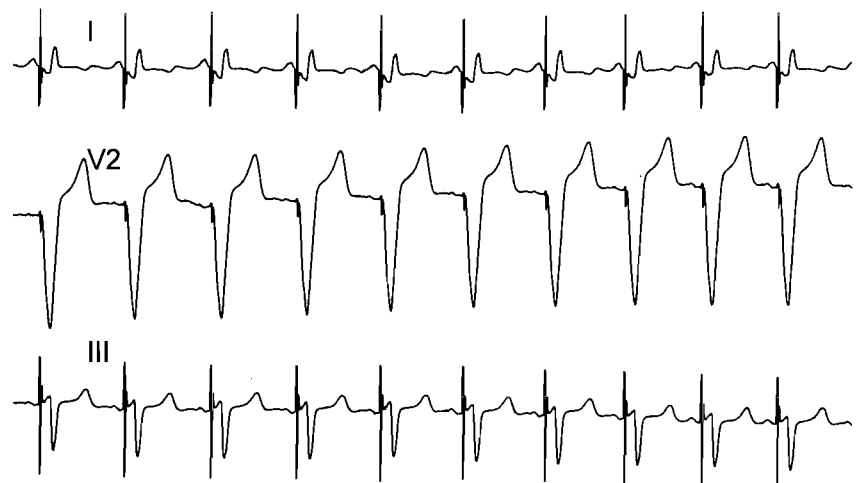
Fig. 4.42. Three channel ECG (leads I,V2,III) of the same patient as figure 4.41. Lead V2 shows no delay between stimulus and QRS complex. Effectual ventricular stimulation.

Figure 4.43 illustrates ventricular capture only in the supernormal period of excitability that extends from the apex of the T wave to its terminal portion. Stimuli occurring during the diastolic period are ineffectual.

Electrocardiographic presentation of latency, the delay from the pacing stimulus to the onset of ventricular depolarization, has been discussed in the previous paragraph. However, figure 4.41 demonstrated that this pattern can be misleading. The latter was concluded from the absence of latency in the precordial lead (V2) and the prolonged duration of the QRS complex. True latency demonstrated in all leads can be caused by myocardial infarction, severe myocardial disease, hyperkalemia and antiarrhythmic drug toxicity [46].
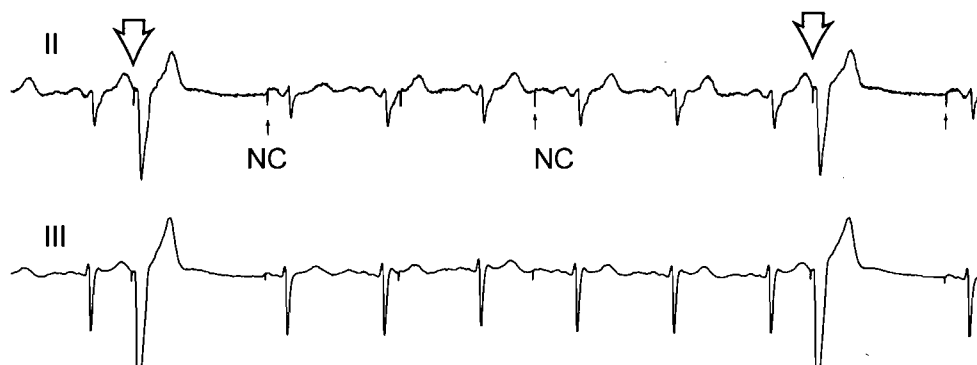
Fig. 4.43. Two channel ECG (lead II and III) of a patient with an inappropriately functioning VVI pacemaker. Capture is only observed during the supernormal phase of excitability (open arrow). Small arrows, marked NC, indicate noncapture in the diastolic period.

Latency can be considered as first-degree block between the stimulation electrode and the myocardium. Under extreme conditions a Type I second-degree Wenckebach block between ventricular electrode and myocardium can occur. It is represented in the ECG as a gradual prolongation of the stimulus to QRS interval, finally resulting in an ineffectual pacemaker spike (Fig. 4.44).

Fig. 4.44. Terminally sick patient with a VVI pacemaker pacing at a rate of 96 ppm, showing Wenckebach type of pacemaker-exit conduction.

This phenomenon is reported during hyperkalemia and antiarrhythmic drug toxicity of particularly Type I agents [47], but occurs mostly in terminal situations. It may thus represent an important adverse prognostic sign [48].

The mechanism of Wenckebach periodicity between electrode and myocardium is uncertain but could possibly be explained on the basis of one or more of the following factors:

1. It may be a result of a gradually increasing block around the electrode site similar to that seen in the classic form of AV nodal Wenckebach periodicity.
2. It may be the result of a gradual decrease in the effectiveness of each succeeding stimulus to break out of the immediate electrode area into surrounding myocardium. As a result an

increasing amount of myocardial mass is thus dependent on secondary propagated excitation and a decreasing amount on direct stimulation. In a depressed myocardium in which propagated excitation may proceed in a very non-homogeneous manner, this could result in variable delays with pronounced differences in the interval between stimulus and ventricular depolarization.

3. It could possibly result from time dependent refractoriness, extending well beyond the end of the T wave so that succeeding impulses occur progressively earlier in the refractory period.

Figure 4.45 demonstrates latency and Wenckebach periodicity between ventricular pacing electrode and ventricular myocardium in a terminally sick patient. It illustrates that the degree of block between electrode and myocardium is rate dependent. When Wenckebach periodicity occurs secondary to potentially reversible abnormalities such as severe hyperkalemia and antiarrhytmic drug toxicity, it can also be prevented by reducing the pacing rate or increasing pacemaker output.
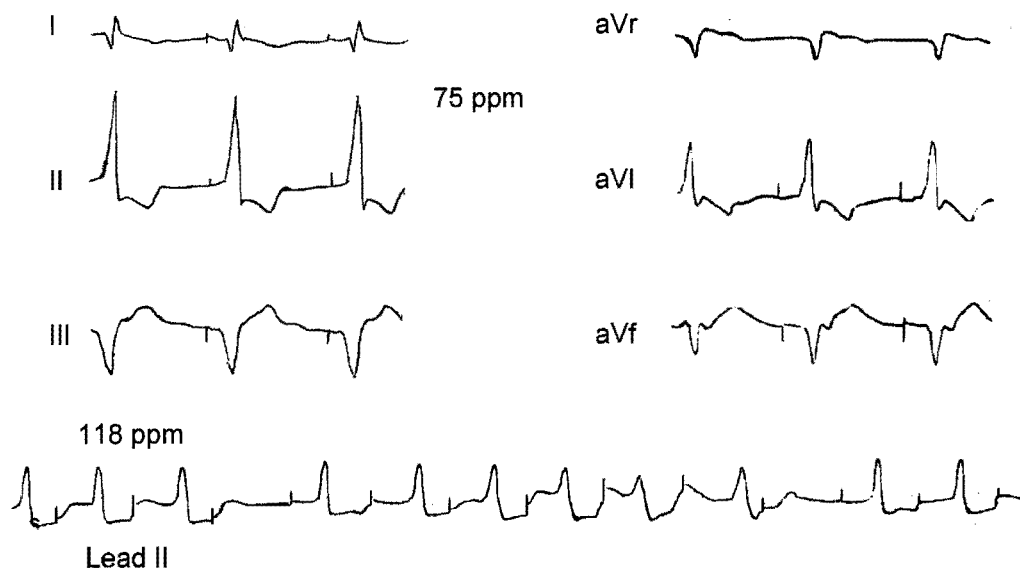
Fig. 4.45. Standard lead ECG in a terminally sick patient showing conduction delay between ventricular stimulus and QRS complex when pacing at rate of 75 ppm. After increasing stimulation rate to 118 ppm Wenckebach exit conduction is observed.

4.6.4 Intermittent ventricular capture associated with attenuation of the pacemaker stimulus

In the previous paragraph different types of intermittent capture are described in which the pacemaker pulse in the ECG showed a constant amplitude. Because no variations in amplitude were recorded during loss of capture, impedance in the lead system was excluded as a source of failure. Variation in amplitude of the pacemaker stimulus due to recording equipment or respiration are excluded.

In patients in whom intermittent loss of capture is suspected, because of intermittent recurrence of symptoms, but not recorded during routine ECG, manipulation of the pulse generator and lead

should be done while recording the ECG, in order to assess the continuity of the lead and connector in the subcutaneous part of the pacing system.
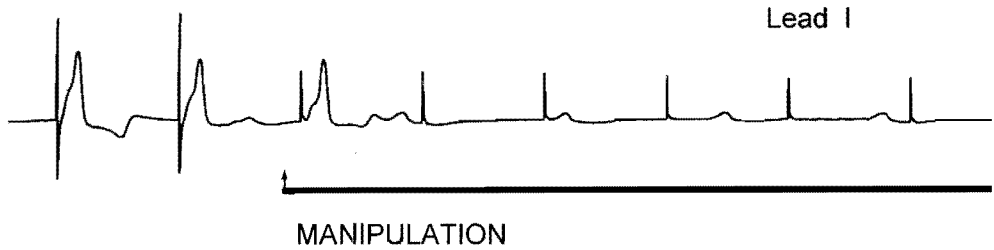
MANIPULATION

Fig. 4.46. ECG lead I during manipulation of the pulse generator showing attenuation of the pacemaker stimulus.

This is illustrated in figure 4.46 in a patient with a VVI pacemaker implanted in the upper abdomen. During manipulation of the pulse generator the ECG shows attentuation of the pacemaker stimulus with loss of capture. Fluoroscopy, employing digital cardiac imaging (DCI) equipment, initially showed no abnormalities of the pulse generator and lead (Fig. 4.47).
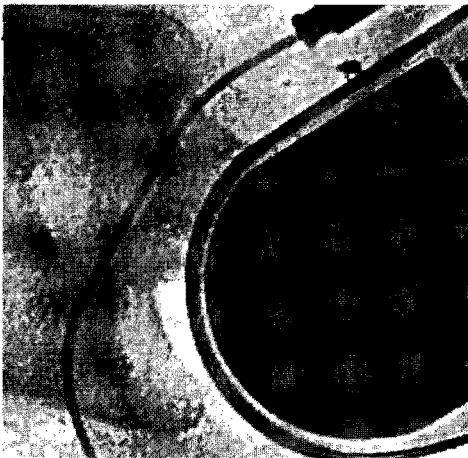
Fig. 4.47. Photograph of the pulse generator (DCI technique) showing no abnormalities in the pacemaker lead.
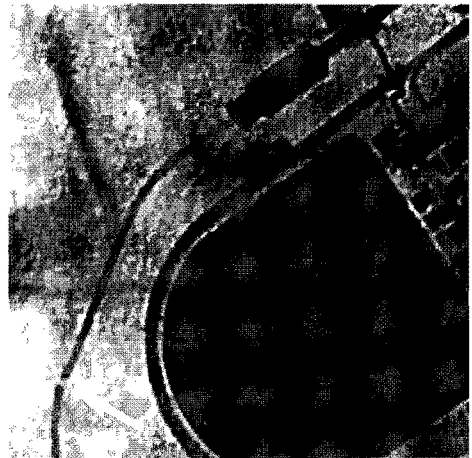
Fig. 4.48. Photograph of the pulse generator (DCI technique) during manipulation of the pacemaker. Arrow indicates discontinuity of the conductor.

During manipulation a break in the conductor coil became obvious (Fig. 4.48). It is obvious that breakage of the conductor coil was responsible for noncapture. Presence of some body fluid between both parts of the lead maintained a reduced current (below stimulation threshold) through the system, electrocardiographically confirmed by a decrease in pulse amplitude. Lead impedance during noncapture was elevated. However, inversion of the statement that attenuation of the pacemaker stimulus indicates an increase in impedance, is not always true. This is

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demonstrated in figure 4.49, that shows great similarity to the ECG in figure 4.46. It was recorded in a patient with an implanted DDD pacemaker, who showed intermittent ventricular capture with attenuation of the pacemaker stimulus during manipulation of the pulse generator. Although a poor contact between the lead and an adapter used to connect the lead with the pulse generator was expected, during noncapture the lead impedance telemetry was < 100 Ohm and the stimulation current over 20 mA. This indicated a low resistance pathway, i.e. an insulation defect between the lead (cathode) and the pacemaker system housing (anode). At exploration protrusion of the setscrew through the silicone rubber adapter sleeve was found, which formed a short-circuit between conductor and pulse generator can [50].
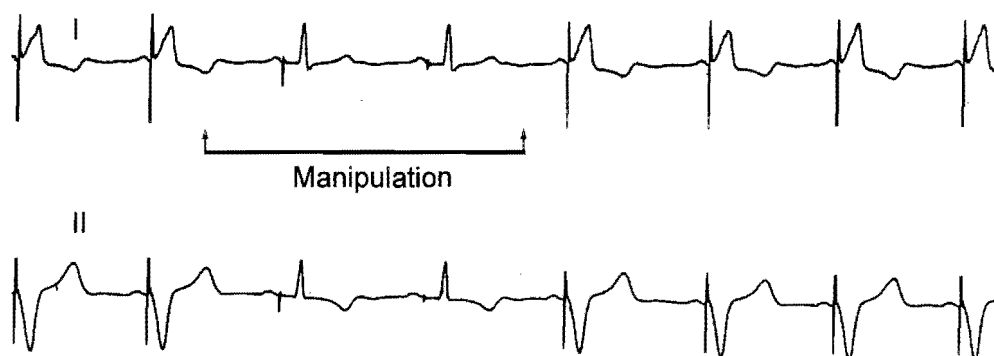
Fig. 4.49. ECG lead I and II of a patient with a DDD pacemaker showing ventricular noncapture during manipulation. Compare the similarity of this recording to figure 4.46. In this patient however, noncapture was caused by a short circuit between pacemaker can and an inappropriately functioning adapter.

In general the amplitude of the pacemaker stimulus on the ECG is proportional to the current and thus inversely proportional to the impedance in the pacing system. This statement is true only if there is a current flow in one direction. If, however, an additional current pathway, forming a complete or partial short circuit, is present, the amplitude of the pacemaker stimulus in the ECG depends on the site of the short circuit and the relationship between the strength of the short circuit current and the stimulation current.

4.7 Causes of no output
In order to confirm the diagnosis no output, two criteria should be fullfilled:
1. Pacing rate should be higher than patient's intrinsic rate. If hysteresis is activated in a pacing system, hysteresis rate should be higher than the patient's intrinsic rate.
2. Magnet application should **not** result in pacemaker stimuli in the recorded ECG. If magnet application results in asynchronous pacing the phenomenon should be classified as oversensing.

No output may be due to failure of the pulse generator circuitry, including battery failure, or by failure of the pulse generator connector block or the lead system. This means that no output is caused by:
1. Failure in the electronic circuitry due to component failure or battery failure; no pulse generation,

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or

2. Discontinuity in the lead system or in the connector block of the pulse generator; no pulse transmission.

4.7.1 Component and battery failure.

Battery failure is defined as a battery voltage at a level below the operating level of the pulse generator circuitry. Battery depletion whether normal or premature rarely results in no output, but is indicated by a change in pacing mode, pulse width and/or (magnet) rate.

This implies that the effect of battery failure and component failure are similar if it results in no output; no pulse is generated. Component failure resulting in no output has been reported after external defibrillation [51], radio frequency ablation and radiation therapy [52].
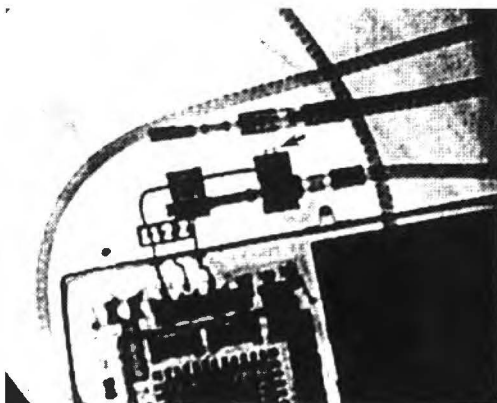
Fig. 4.50. DCI picture of a pulse generator with a bipolar lead. Note the position of the setscrew in the pulse generator connector block that is still visible at the proximal electrode, indicating incomplete tightening. Patient presented with no output in bipolar mode. A second identical lead was held above the pulse generator as a reference to confirm correct positioning of the lead connector.
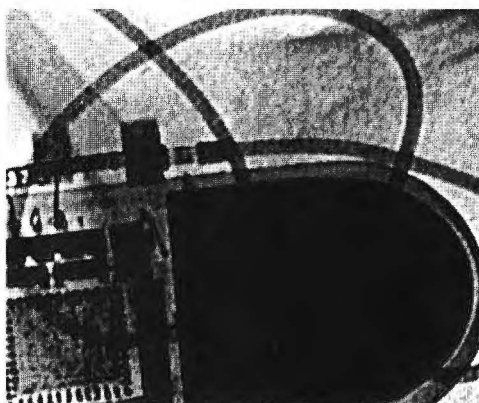
Fig. 4.51. DCI picture of the same pulse generator (fig. 4.50). After tightening the setscrew which is no longer visible. Normal pacemaker function in bipolar mode is restored.

Pacemaker telemetry and programming is the only tool to confirm no output due to battery or component failure. If no output is observed and programming and telemetry fails, component/battery failure can be concluded, provided that there is no defect in the pacemaker programmer. In case of component/battery failure, magnet application will not result in pacemaker stimuli in the ECG, which is also true for lead discontinuity. Absence of pacemaker stimuli after magnet application only confirms no output but gives no information about the cause. Before replacement of a pulse generator showing no output due to component/battery failure, fluoroscopy should confirm lead integrity. During pulse generator replacement lead impedance should be checked to exclude an insulation defect, which probably increased battery drain resulting in premature battery failure.

4.7.2 Lead discontinuity

In case of lead discontinuity as a cause of no output, normal communication for programming and telemetry between pulse generator and programmer should be possible. Telemetry of lead impedance will reveal a high impedance often beyond the range of the measuring capability of the system. A high impedance, however, gives no indication of the location of the defect. The lead system integrity can be interrupted at one or more of the following sites:

1. Connection lead-pulse generator
2. Lead connector
3. Lead adapter, if applicable
4. Lead conductor
5. Anodal insulation in unipolar systems
6. Insulation failure
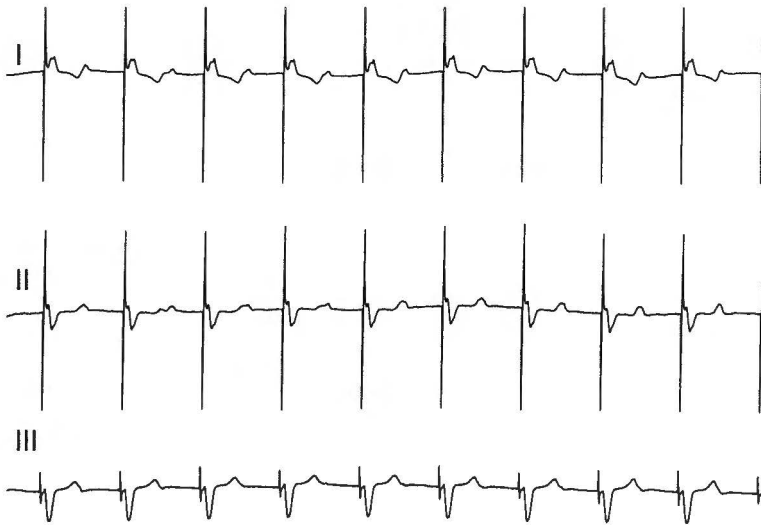
Fig. 4.52. Three channel ECG of a patient with a unipolar DDD pacemaker. There is ventricular pacing at the programmed lower rate without an atrial stimulus preceding the ventricular stimulus indicating no output from the atrial channel. VVI pacing ?

Fig. 4.53. Same patient as figure 4.52 showing an ECG recorded in the right precordium (RP) at the fifth intracostal space at double standard (1mV=2cm) with ineffectual atrial stimuli. This demonstrates atrial noncapture instead of no output or VVI pacing.

4.7.3 Connection lead pulse generator
When a poor connection between the lead and the pulse generator is the cause of no output, it is related to the penetration of the lead connector into the pulse generator receptacle or to the fixation of the set screw in the connector block. X-ray, fluoroscopy or DCI are techniques used to confirm the probable diagnosis. The DCI technique is preferable because of the "editing" possibilities; brightness and contrast can be adapted and the picture can be magnified. Figure 4.50 shows a DCI picture of a bipolar lead connected to a pulse generator. The setscrew for the distal electrode is not visible, but the setscrew for the proximal electrode is still visible, indicating that it was not completely tightened. DCI after complete tightening of the setscrew during re-operation revealed that the setscrew was no longer visible (Fig. 4.51). The patient presented with no output, when programmed in the bipolar electrode configuration, while pacing and sensing were normal in the unipolar configuration. After tightening the set screw, normal pacing and sensing in the bipolar mode was restored.
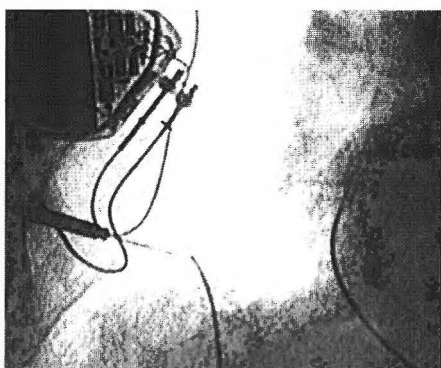
Fig. 4.54. DCI picture of the same patient showing a broken atrial epicardial lead. The interruption is at the site of a connector used to repair a previous lead fracture.

4.7.4 Interruption conductor, connector, adapter
No output caused by an interruption in conductor, connector or adaptor can only occur with an intact insulation, without penetration of body fluids between the two broken parts. If there is a "fluid" bridge between both broken parts, lead impedance will be increased and a pacing artifact will be seen on the ECG. The absence of a pacemaker stimulus indicating no output should be confirmed by a 12 channel ECG and if necessary by a non-standard lead ECG at double standard. When using a non-standard bipolar lead, the best electrode configuration is between the anodal and cathodal position of the pacing system. Figure 4.52 demonstrates no output from the atrial channel in a patient with a DDD pacemaker recorded in leads I,II,III. Recording of an ECG at the right precordium at the level of the fifth intracostal space (corresponding with the position of the epicardial atrial electrode) at double standard revealed ineffectual atrial stimuli with a low amplitude (Fig. 4.53). The latter recording showed that this was a case of no capture instead of no output, which could not be deduced from the standard leads ECG in figure 4.52. The DCI picture demonstrated a broken conductor at the site where earlier a connector was mounted to repair a previous lead fracture (Fig. 4.54).

4.7.5 Anodal insulation in unipolar systems
A very rare cause of no output may be encountered in unipolar pacing systems where the anodal electrode is insulated by subcutaneous air. Unipolar pulse generators are partially coated with

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insulation material in order to prevent muscle stimulation. The insulated area is implanted towards the pectoral muscle while only a small window permits contact between subcutaneous tissue and the metallic case (anode). When air is trapped between this window and the subcutaneous tissue the current pathway is interrupted, resulting in no output [53,54]. This phenomenon is diagnosed by application of some pressure on the overlying skin which results in normal pacing and capture (Fig. 4.55). Air entrapment is only encountered shortly after implantation or thoracic surgery and can be caused by subcutaneous emphysema.

4.7.6 Insulation failure
All the previously mentioned causes of no output associated with galvanic discontinuity of the pacing system are characterized by a high lead impedance. However, if a complete short circuit between anode and cathode exits, no pacemaker stimulus may be visible in the ECG. Figure 4.56 demonstrates complete cessation of pacing during manipulation of the pulse generator. During
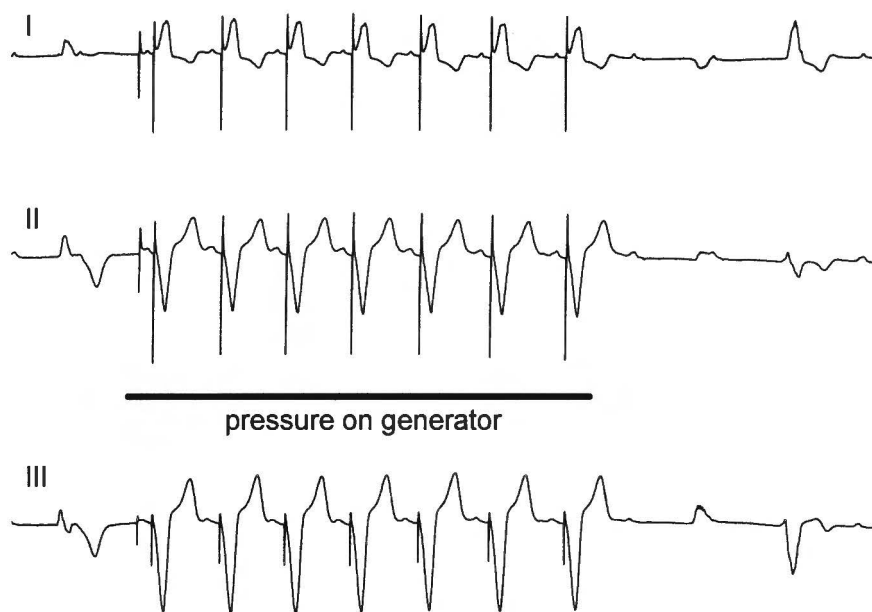
Fig. 4.55. Three channel ECG (lead I,II,III) of a patient with an implanted unipolar DDD pacemaker. Pacing is only resumed during minor pressure on the pulse generator. After release of pressure no output is observed. Subcutaneous air insulating the anodal electrode was the cause of no output.

this maneuver the case of the pulse generator was in direct contact with the cathodal conductor. This contact was caused by a setscrew protruding from an adapter that was implanted underneath the pulse generator [50]. Lead impedance during no output was below the measuring capability of the pacemaker telemetry, which was limited to 100 Ohm as the lowest value.

Thus in cases like this there is a high current between the anode and the point of short circuit with the conductor (cathode), but because of the infinitively small dipole no pacemaker stimulus is recorded on the ECG.

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In all the patients presenting with a history of failure to pace (no output or noncapture) with normal pacing at the time of control, continuity of the lead and connector in the subcutaneous part of the system should be assessed by manipulation of the pulse generator and lead while recording the ECG.

MANIPULATION
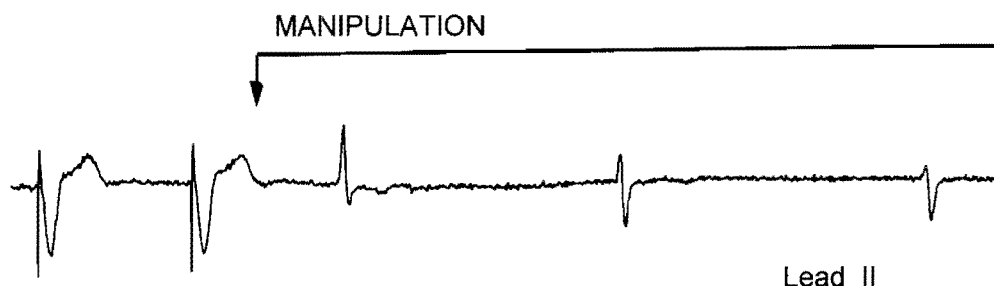
Lead II

Fig. 4.56. Single channel ECG (lead II) of a patient with an implanted VVI pacemaker. During manipulation no output is observed. A short circuit between the setscrew of an adapter and the case of the pulse generator was the cause of no output.

4.8 Causes of noncapture
The cause of noncapture can be located in one of the following parts of the pacing system:
1.   The position of the stimulation electrode (cathodal electrode).
2.   The cathodal-myocardial interface.
3.   The integrity of the lead system.
4.   The pulse generator.

4.8.1 Displacement of the stimulation electrode
When noncapture occurs relatively soon after pacemaker implantation, the most likely explanation is displacement or malposition of the pacemaker lead. Minor displacement of the stimulation electrode is hard to prove and is often not evident on X ray. Fluoroscopy sometimes reveals instability of the pacing lead during respiration, indicating displacement as the cause of noncapture. Very rarely intermittent ventricular noncapture is related to the atrial contraction. In case of instability of the electrode the atrial contraction may displace the tip of the lead distally toward the ventricular myocardium and render the stimuli to be more effective (Fig. 4.57). Less commonly, however, the atrial contraction increases the distance between the catheter tip and right ventricular endocardium and may make pacemaker stimuli less effective. A possible mechanism for this increased threshold is the filling and distension of the right ventricle, consequent upon atrial emptying, resulting in separation between the stimulation electrode and the ventricular endocardium [55]. Macrodisplacement of the pacing lead into the inferior caval vein, right atrium or pulmonary artery can be observed in ventricular leads. In case of an atrial lead it can be displaced into the right atrium cavity, superior caval vein or right ventricle. The latter may result in effective ventricular pacing. See also chapter IV, 4.5.2.
If diaphragmatic stimulation via the phrenic nerve develops shortly after implantation of an atrial lead in the right atrial appendage, it is suggestive of displacement of the lead towards the atrial free wall, even in the presence of effectual atrial stimulation. In unipolar systems, macrodisplacement of the pacing lead can be recognized by a change in direction and amplitude

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of the stimulus. Displacement of the stimulation electrode, where the electrode is not in contact with the myocardium results in a decrease of lead impedance, because the specific resistance of blood is approximately 30% lower than the specific resistance of the myocardium.
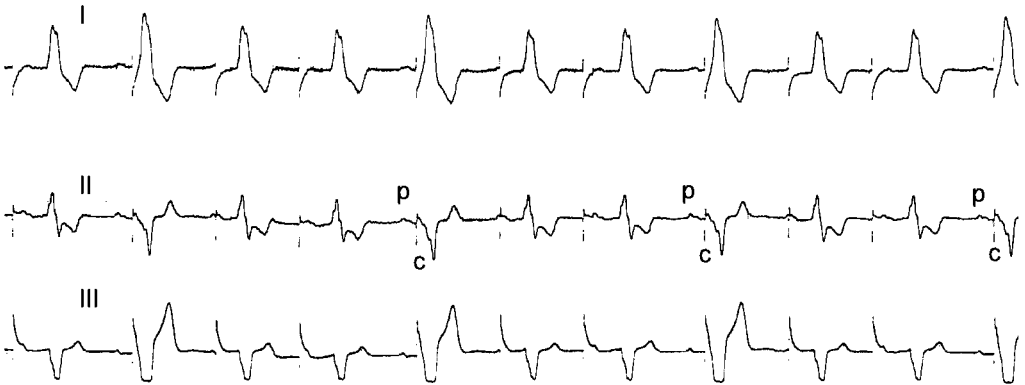
Fig. 4.57. Three channel ECG of a patient with an implanted VVI pacemaker showing intermittent capture. Capture is only observed if the ventricular stimulus is preceded by a P wave with a interval corresponding to the normal PQ time. Capture is most probably caused by the atrial contraction, bringing the electrode in contact with the ventricular myocardium.

4.8.2 Perforation of the stimulation electrode

Perforation or partial perforation which is mainly encountered in ventricular leads may result in noncapture. Perforation is sometimes hard to identify from X-rays or fluoroscopy, but a "deep"
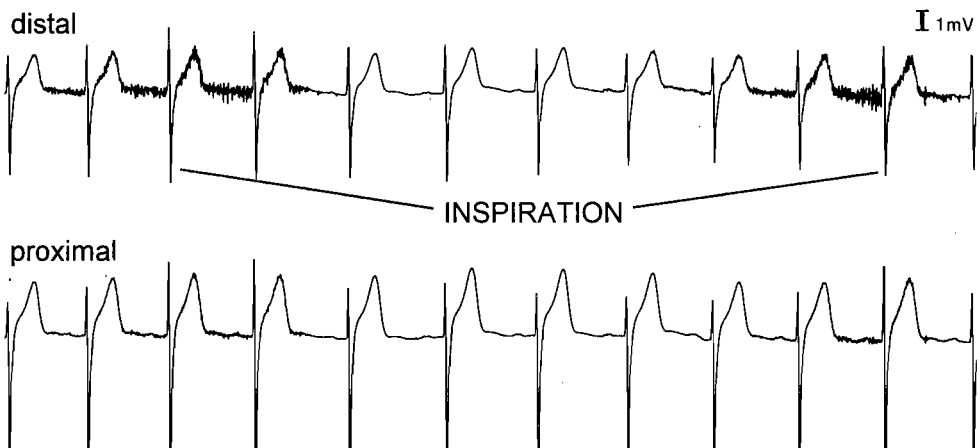
Fig. 4.58. Intracardiac recordings from the distal and proximal electrode of a temporary pacemaker lead. Note the taller R wave and more pronounced injury pattern in the proximal electrogram, while during deep inspiration diaphragmatic myopotentials are recorded from the distal electrode. The latter implies that the distal electrode is closer to the diaphragm than the proximal, while the injury pattern indicates that the proximal electrode is in better contact with the myocardium. Electrograms are compatible with lead perforation

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position into the right ventricular apex is suggestive of perforation. Presence of noncapture associated with direct diaphragmatic stimulation will support the diagnosis. The diagnosis of perforation may be confirmed by recording characteristic sequential electrograms from the electrode tip during withdrawal from the extracardiac position; chapter I, Fig. 1.7. When perforation occurs early after implantation the electrogram often fails to exhibit an injury pattern. Withdrawal of the lead with sequential recordings of the electrogram from the tip of the electrode may show progressive increase in amplitude of the electrogram together with the reappearance of the injury pattern as the stimulation electrode makes contact with the myocardium [56,57]. In a bipolar ventricular lead, perforation can more easily be confirmed by the intracardiac electrogram and stimulation threshold. When the intracardiac electrogram from the proximal electrode has a more pronounced injury pattern and a taller R wave than the distal electrode, this implies that the proximal electrode is in better contact with the myocardium than the distal electrode, indicating perforation (Fig. 4.58) [58].

Fig. 4.59. Anteroposterior X ray of a patient with a VVI pacemaker 2 days after implantation of the pacing system. Note the "deep" position of the ventricular lead.

Fig. 4.60. Anteroposterior X ray of the same patient (Fig. 4.59) three months later, showing dramatic enlargement of the heart contour due to pericardial tamponade. Patient presented with noncapture.

For using the stimulation threshold to confirm perforation, stimulation should be measured between a subcutaneous electrode (anodal electrode) and subsequently the proximal and distal electrode as the cathodal electrode. If the configuration subcutaneous electrode-proximal electrode gives a lower stimulation threshold than the configuration subcutaneous electrode-distal electrode it implies that the proximal electrode is in better contact with the myocardium than the distal electrode, and this supports the diagnosis of perforation. Perforation of ventricular leads can be associated with inhibition of the pacing system by diaphragmatic myopotentials (See chapter V 5.7.2).

In the absence of hemopericardium withdrawal of a perforated lead withdrawal can be performed without clinical sequelae. However care should be taken in withdrawal of a perforated chronic lead associated with pericardial tamponade (Fig. 4.59, 4.60, 4.61). In our experience withdrawal of such a lead during thoracotomy revealed excessive bleeding requiring surgical repair of the site of perforation [59].

4.8.3 Malposition of the stimulation electrode

Malposition of a ventricular lead into the coronary sinus or one of the other branches of the coronary venous system may result in noncapture. It is usually observed shortly after pacemaker implantation since the stimulation threshold is higher in the coronary sinus compared to right ventricular pacing and a stable position is hard to achieve. Normally lateral X ray or fluoroscopy during implantation indicates implantation of the ventricular lead into the coronary sinus by its posterior position. Electrocardiographically it is characterized by a right bundle branch block pattern during stimulation instead of a left bundle branch block pattern as observed during right ventricular pacing.

Fig. 4.61. DCI picture, right anterior oblique view showing complete perforation of the ventricular electrode; same patient as figure 4.60.

4.8.4 The electrode-myocardial interface

The electrode-myocardial interface is influenced by local processes that occur around it but also by the general state of excitability of the myocardium.

4.8.4.1 Local effects

During the first month after implantation, the myocardial stimulation threshold increases, but usually remains constant thereafter. In animal studies the increase in stimulation threshold correlates with local tissue reaction around the stimulation electrode, and includes necrosis, cellular infiltration, and fibrosis. Decreasing the intensity of the tissue reaction with corticosteroids minimizes the normally observed increase in stimulation threshold [60].
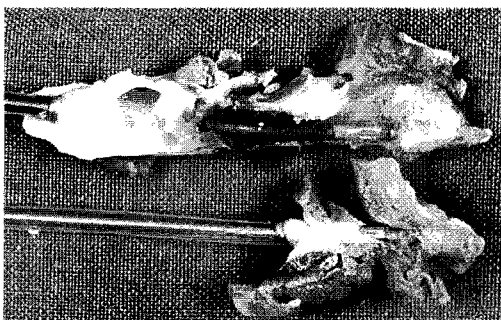
Fig. 4.62. Autopsy specimen showing two ventricular electrodes implanted in the same patient. The upper electrode showed exit block during follow up due to extensive fibrosis formation (white area), while the lower electrode functioned satisfactory during follow up with limited fibrosis formation.

This effect of corticosteroids is used to develop electrodes that are capable of eluting small amounts of steroids in a time-release fashion that prevents both acute and chronic rises in stimulation threshold [61].

The rise in stimulation threshold shortly after implantation can be explained by the healing process that forms a layer of fibrous tissue around the electrode. This layer consists of conductive but non excitable cells, which extends the distance between the stimulation electrode and excitable myocardium. Practically it is comparable with an increase in the electrode surface area. Because increase in electrode surface area results in a decrease of current density, the stimulation threshold increases (Fig. 4.62).
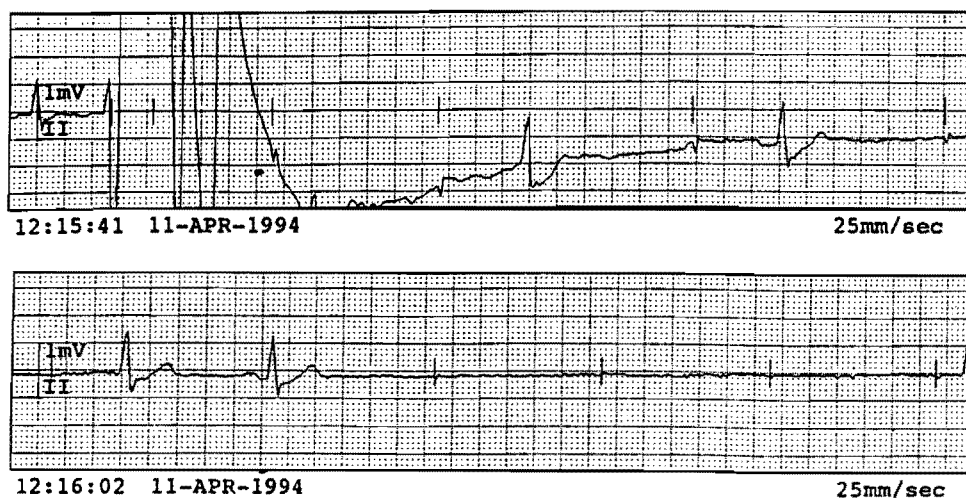
Fig. 4.63. One channel ECG of a patient with an implanted VVI pacemaker. After application of a DC countershock for supraventricular arrhythmia noncapture is recorded while ventricular sensing is maintained. Note the ST segment depression immediately after the DC countershock (upper tracing), which has almost disappeared after 21 seconds (lower tracing). In the upper tracing the pacemaker stimulus is separated from the ECG, due to the recording equipment (see also chapter III 3.1.3).

Excessive rise in stimulation threshold above the maximum output level of the pulse generator without stimulation electrode displacement is named exit block. Exit block rarely occurs with atraumatic endocardial leads but can be observed in endo- and epicardial leads employing screw-in electrodes as a result of a progressive reaction around the electrode [62]. It is clear that inferior wall infarction with resulting necrosis in the area of the stimulation electrode can result in an increase of stimulation threshold or even exit block. Also local ischemia can give rise to a (temporary) increase in stimulation threshold [63].

An abrupt excessive rise in stimulation threshold can be observed immediately after a DC countershock for conversion of supraventricular or ventricular arrhythmias. Several explanations are given for this phenomenon. Dysfunction has been demonstrated to be due to damage to the pulse generator circuitry as a consequence of inadequate generator shielding or to the extreme proximity of the lead to the high energy delivered by the DC countershock [64,65]. A second

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potential mechanism for noncapture may be due to transient changes in the excitability at the electrode tissue interface. The pacemaker lead system may act as a lower resistance pathway thereby leading to a high current density at the stimulation electrode [66]. This high current density may cause a local trauma resulting in transient loss of capture. A third possible mechanism is the effect of DC countershocks on myocardial oxygen extraction. Following transthoracic DC countershock myocardial oxygen extraction is reduced [67]. This reduction in myocardial oxygen extraction may lead to ischemia, which is a known factor for increase in stimulation threshold. The ECG in figure 4.63 illustrates noncapture after a DC countershock for supraventricular arrhythmia, while ventricular sensing is not affected. The intrinsic QRS complex immediately after DC countershock shows transient ST segment depression, indicative of ischemia, whereas maintenance of ventricular sensing excludes pulse generator failure.

4.8.4.2 General effects
The myocardial stimulation threshold can vary with normal changes in physiologic states during the day. The energy threshold increases up to 40% while sleeping, and while eating up to 42%, and decreases from 11 to 40% with postural changes and exercise. These observations have been explained on the basis of changes in sympathetic tone. An increase in sympathetic tone is associated with a decrease in stimulation threshold, and a decrease in sympathetic tone, or increase in parasympathetic tone is associated with an increase in stimulation threshold [68].

Increases in stimulation threshold that occur during sleep as a result of sympathetic withdrawal and vagotonia may be important clinically, especially when combined with other factors like an abnormally high basal stimulation threshold.

Electrolyte disturbance can also be a cause of increase in stimulation threshold. Most frequently encountered is hyperkalemia, which causes an increase in myocardial stimulation threshold resulting in failure to capture. Elevations of serum potassium to levels exceeding 7.0 mEq/L consistently increase the stimulation threshold. Failure to capture due to hyperkalemia can be intermittent and show Wenkebach periodicity. See also chapter IV, 4.6.3.

Loss of capture can also be caused by the application of antiarrhythmic agents. Type I antiarrhythmic agents such as quinidine and procainamide cause increase in stimulation threshold, especially when plasma concentrations of these agents are elevated. As in hyperkalemia, failure to capture may be intermittent or complete, and may take the form of fixed block or Wenckebach periodicity. The mechanism by which Type I antiarrhytmic agents exert their effects in patients with pacemakers is probably an extension of their therapeutic effects of increasing myocardial refractoriness, decreasing conduction velocity, and increasing excitability threshold by depressing membrane responsiveness and reducing membrane potential [69].

4.8.5 Integrity of the lead system
Every increase in impedance in the lead system, whether it is located in the conductor, connector or adapter decreases the stimulation current through the myocardium, according to Ohm's law. Only in the case of a complete interruption with an infinitely high impedance, the stimulation current is equal to zero and no output will be observed. Noncapture will occur if the decrease in stimulation current, due to an increase in impedance, falls below the stimulation threshold. In the majority of conductor fractures observed in pacing systems, there is still a contact between both ends of the fracture by body fluids penetrating the lead system. This implies a high impedance, where pacemaker stimuli will still be visible on the ECG. Capture is dependent on the

impedance, the voltage applied and the current stimulation threshold. Increase in impedance, thus reducing stimulation current, will result in a decrease of the pacemaker stimulus on the ECG.

Very rarely complete lead fracture (fracture of conductor and insulation) is observed. It will be clear that the ECG will represent noncapture, but in this case noncapture is associated with a change in vector of the pacemaker stimulus. The change in stimulus vector depends on the site of the complete fracture, as the site of fracture corresponds with the location of the end of the fractured lead acting as the cathodal electrode [70].

Fig. 4.64. Single channel ECG (lead II) recorded after implantation of a bipolar ventricular lead. In the first part of the recording fracture of the outer insulation is simulated by placing an external resistance of 500 Ohms between cathode and a subcutaneous electrode demonstrating "unipolarization" of the ECG by an insulation defect. Voltage stimulation threshold was not affected by this simulated insulation defect

Although it has been demonstrated in paragraph 4.7.6 that insulation defects can cause noncapture, the insulation defect will create an extremely low resistance pathway before failure to capture occurs. Because the pacemaker pulse is a voltage pulse, failure to capture will only occur if the current through the insulation defect is large enough to decrease the current densitity below the threshold of the myocardium. The parallel impedance through the insulation break will be extremely low and before this low level is reached sensing abnormalities will be observed in the majority of cases. The parallel impedance of an insulation defect has more effect on the sensing capabilities than on the stimulation properties of the pacing system. This mechanism will be discussed in chapter V 5.14.2.

In bipolar leads, defects in the outer insulation may become apparent by an increase of the stimulus artifact indicating "unipolarization" of the pacing system, which is rarely associated with noncapture (Fig. 4.64). See also chapter III, figure 3.11.

4.8.6 Pulse generator.

Noncapture due to pulse generator malfunction can be encountered in case of battery exhaustion. Battery exhaustion is easily recognized by measuring battery voltage by telemetry, or a change in pacing parameters (e.g. mode, rate, pulse width) indicating battery status. Decrease in output resulting in failure to capture is rarely due to spontaneous component failure but has been described by iatrogenic causes like external defibrillation [71] or radiation therapy [72] and radio frequency ablation.

Loss of capture can be encountered in a normally functioning pulse generator in which one of the output parameters (pulse width and/or voltage or current) has been inappropriately programmed.

4.7 Summary and conclusion

Effectiveness of stimulation can be deferred from the ECG if the pacemaker stimuli and the subsequent depolarization of the stimulated chamber is visible. Manifestations of ineffective stimulation are no output and noncapture. Before the conclusion no output can be drawn from the ECG, the pacing system should be reverted to the asynchronous mode by magnet application or programming to this mode. Absence of pacemaker stimuli should be confirmed in a 12 lead ECG and supplementary modified chest leads at the site of the stimulation electrode, recorded at double standard. If no pacemaker stimuli are present inspite of the asynchronous mode, no output can be deferred; if reversion to the asynchronous mode results in pacemaker stimuli on the ECG oversensing is established.

No output can be caused by failure of the pulse generator (no pulse formation) or failure of the lead or lead connection to the pulse generator (no pulse transmission). In pulse generator failure communication between pacemaker programmer and pulse generator is impossible. In lead or lead connection failure communication between programmer and pulse generator is maintained and the lead impedance measured by telemetry is abnormal. Intermittent no output is always associated with a lead or lead connection failure. In intermittent no output the distance between pacemaker stimuli is a multiple of the escape interval.

A high lead impedance - higher than 2000 Ohms, which is the maximum measurable value in most pulse generators - a conductor break or poor lead connection can be concluded. If manipulation of the pulse generator results in recurrence of pacemaker stimuli the defect is located between pulse generator and the entrance of the lead in the venous system. Fluoroscopy usually reveals the site of fracture or a poor connection. A rare cause of no ouput is entrapment of subcutaneous air above the pulse generator in unipolar systems. This phenomenon is easily diagnosed by application of slight pressure on the overlying skin and occurs only shortly after surgical intervention.

In a low lead impedance - lower than 100 Ohms, which is the mimimum measurable value in most pulse generators - a short circuit can be concluded. In unipolar psystems the short circuit is located between the pulse generator (anodal electrode) and the conductor of the lead (cathodal electrode), due to an insulation defect of the lead. In bipolar systems a short circuit is caused by an insulation defect between anodal and cathodal conductor. Location of the insulation defect in bipolar leads is almost impossible and is pointless, because it can only be solved by implantation of a new lead.

Loss of capture is caused by displacement of the stimulation electrode, decrease of stimulation current passing the cathodal electrode or decreased excitability of the myocardium adjacent to the cathodal electrode.

Displacement of the stimulation electrode rarely occurs later than 2-3 months after implantation. If intermittent capture is observed in this period, one should compare the morphology of the stimulated complexes with previous recordings. A change in morphology indicates displacement of the stimulation electrode. If stimulation or sensing in a chamber other than the original implantation site is observed, macrodisplacement of the electrode can be deferred. E.g., if atrial stimulation or sensing is observed from a lead that was originally implanted in the right ventricle, the lead is displaced into the right atrium.

Sudden onset of diaphragmatic stimulation is an indication of lead displacement. In ventricular leads, by direct stimulation of the diaphragm (perforation) and in atrial leads by phrenic nerve stimulation.

Intermittent capture associated with abrupt changes in the amplitude of the pacemaker stimulus on the ECG indicates a change in stimulation current. Increase in lead impedance indicates a conductor or lead connection problem, while decrease in lead impedance is an indicator of insulation failure. Manipulation of the pulse generator can be useful to locate the site of failure. A conductor failure is unlikely to occur shortly after implantation.

Observation of noncapture in patients in whom lead displacement is excluded and lead impedance is not significantly changed, administration of threshold elevating drugs, electrolyte imbalance and myocardial infarction should be excluded as the possible cause. After exclusion of these causes, exit block can be deferred. Exit block is caused by an excessive healing reaction at the electrode site with fibrosis formation and is mainly encountered in electrodes employing active (screw-in) fixation.

References

1. Bellet, S. Clinical disorders of the heart beat, 2nd edition. Philadelphia, Lea and Febiger, 1971, pp. 263,267,1066.

2. American College of Cardiology Task Force I. Standardization of terminology and interpretation. Am J. Cardiol.1978;41: 130.

3. Watanabe Y. Terminology and electrophysiologic concepts in cardiac arrhythmias.IV. Fusion beats and summation of impulses. PACE 1979;2: 76-87.

4. Barold SS, Ong LS, and Heinle RA. Demand pacemakers: Normal and abnormal mechanisms of sensing. In P. Samet and N. El-Sherif (Eds): Cardiac Pacing, Grune & Stratton, New York, 1980, p. 551.

5. Barold SS, Falkoff MD, Ong LS, and Heinle RA. Characterization of pacemaker arrhythmias due to normally functioning AV demand (DVI) pulse generators. PACE 1980;3: 712-723.

6. Barold SS, Falkoff MD, Ong LS, and Heinle RA. Interpretation of electrocardiograms produced by a new unipolar multiprogrammable "committed" AV sequential demand (DVI) pulse generator. PACE 1981;4: 692-708.

7. Levine PA, Brodsky SJ, and Seltzer JP. Assessment of atrial capture in committed atrioventricular sequential (DVI) pacing systems. PACE 1983;6: 616-623.

8. Medtronic technical manual Spectrax SXT, p. 9, 1983.

9. Josephson ME and Seides SF. Clinical cardiac electrophysiology; Techniques and interpretations.Philadelphia, Lea & Febiger, 1979, pp.33-35.

10. Amram SS, Vagueiro MC, Pimenta A, and Machado HB. Persitent atrial standstill with atrial inexcitability. PACE 1978;1: 80-89.

11. Solti F, Rényi-Vámos F, and Gyöngy T. Atrial standstill: An indication for rate adaptive pacing. PACE 1990;13: 830-832.

12. Greenspon AJ, Cox J, and Greenberg RM. Atrial dislodgement with a DDD pacemaker. PACE 1986;9: 436-440.

13. Stark S, Farshidi A, Hager WD, and Donaldson MC. Unusual presentation of DDD pacemaker system malfunction. PACE 1985;8: 255-260.

14. Nathan AW and Camm AJ. Double ventricular pacing in a patient with a "DDD" universal pacemaker. PACE 1984;7:432-435.

15. Van Mechelen R and Vanderkerckhove Y. Atrial capture and dual chamber pacing. PACE 1986;9: 21-25.

16. Dreifus LS. In search of atrial sensing and capture. PACE 1988;11: 381-383.

17. Barold SS, Nanda NC, Ong LS, et al. Determination of successful atrial capture during unipolar dual-chambered pacing. In SS Barold (Ed): Modern Cardiac Pacing. Mount Kisco, New York, Futura Publishing Company, 1985, pp.677-692.

18. Nanda NC, Bhandari A, Barold SS, et al. Doppler echocardiographic studies in sequential atrioventricular pacing. PACE 1983; 6: 811-814.

19. Zugibe FT, Nanda NC, Barold SS, et al. Usefulness of Doppler echocardiography in cardiac pacing: Assessment of mitral regurgitation, peak aortic flow velocity and atrial capture. PACE 1983; 6: 1350-1357.

20. Schuilenburg RM. Patterns of V-A conduction in the human heart in the presence of normal and abnormal A-V conduction. In HJJ Wellens, KI Lie, MJ Janse (eds.). The conduction system of the heart. Leiden, Stenfert Kroese BV, 1976, p. 485.

21. Van Gelder LM, El Gamal MIH. Ventriculoatrial conduction: A cause of atrial malpacing in AV universal pacemakers. A report of two cases. PACE 1985; 8: 140-143.

22. Slegers LC, van Gelder LM. Dual chamber rate responsive pacing. Neth J Cardiol 1992; 5: 256-260.

23. Doi Y, Takada K, Nakagaki O, Yoshimura S, Hiroki T, Arakawa K. A case of cross stimulation. PACE 1989; 12: 569-573.

24. Puglisi A, Ricci R, Azzolini P, Peraldo Neja C, Fioranelli M, Special G, Angrisani G. Ventricular cross stimulation in a dual chamber pacing system: Phenomenon analysis. PACE 1990; 13: 993-1001.

25. Irwin M, Cameron D, Louis C, Harris L, Goldman B. Crosstalk stimulation of the ventricle in the implanted dual unipolar system.(abstract) PACE 1988; 11: 508.

26. Goldschlager N, Francoz R. Ventricular cross stimulation using a pacing system analyzer. PACE 1990; 13:986-992.

27. Levine PA, Rihanek BD, Sanders R, Sholder J. Cross-stimulation: The unexpected stimulation of the unpaced chamber. PACE 1985; 8: 600-606.

28. Anderson MH, Nathan AW. Ventricular pacing from the atrial channel of a DDD pacemaker: A consequence of pacemaker twiddling ?. PACE 1990; 13: 1567-1570.

29. Bayliss CE, Beansland DS, Baird RJ. The pacemaker-twiddler's syndrome: A new complication of implantable transvenous pacemakers. Can Med Assoc J 1968; 99: 371-373.

30. Stark S, Farshidi A, Hager WD, Donaldson MC. Unusual presentation of DDD pacemaker system malfunction. PACE 1985; 8: 255-260.

31. Greenspon AJ, Cox J, Greenberg RM. Atrial lead dislodgement with a DDD pacemaker. PACE 1986; 9: 436-440.

32. Nathan AW, Camm AJ. Double ventricular pacing in a patient with a "DDD" universal pacemaker. PACE 1984; 7: 432-435.

33. Bognolo DA. Clinical application of A-V sequential (DVI) pacing. In DA Bognolo (ed.) Practical approach to physiologic cardiac pacing. Tarpon Springs, Florida, Tampa Tracings, 1983, p. 88.

34. Parsonnet V. Permanent transvenous pacing. In S. Furman and DJW Escher (Eds). Modern Cardiac Pacing. A clinical overview. Bowie, Maryland, USA, Charles Press, 1975, pp. 77-82.

35. Gulotta SJ. Transvenous cardiac pacing. Techniques for optimal electrode positioning and prevention of coronary sinus placement. Circulation 1970; 42: 701-718 .

36. Moss AJ, Rivers RJ. Atrial pacing from the coronary vein. Circulation 1978; 57: 103-106.

37. Greenberg P, Castellanet M, Messenger J, et al. Coronary sinus pacing. Circulation 1978; 57: 98-103.

38. Smyth NPD, Citron P, Keshishian JH, et al. Permanent atrial sensing and pacing with a new J-shaped electrode. J. Thorac. Cardiovasc. Surg. 1976; 72: 565-570.

39. El Gamal M, van Gelder B. Preliminary experience with the Helifix electrode for transvenous atrial implantation. PACE 1979; 2: 444-454.

40. Van Hemel N, El Gamal MIH, Bakema H, et al. Long term results of endocardial atrial electrodes employing positive fixation. Proceedings Second European Symposium on Cardiac Pacing, Florence 1981, pp.627-630.

41. Mond HG, Sloman JG. The malfunctioning pacemaker system. Part II. PACE 1981; 4: 168-181.

42. Barold SS, Falkoff MD, Ong LS, et al. Electrocardiographic diagnosis of pacemaker malfunction. In HJJ Wellens and HE Kulbertus (Eds). What's new in electrocardiography. The Hague, The Netherlands, Martinus Nijhoff Publishers, 1981, p. 259.

43. Barold SS, Falkoff MD, Ong LS, et al. Normal and abnormal patterns of ventricular depolarization during cardiac pacing. In SS Barold (Ed), Modern Cardiac Pacing. Mount Kisco, New York, USA, Futura Publishing Company, 1985, pp. 554-555.

44. Castellanos A, Luceri RM, Zaman L, et al. The value of analysis of pacemaker-induced arrhythmias in the understanding of naturally occurring rhythm disorders of the heart. In SS Barold (Ed), Modern Cardiac Pacing. Mount Kisco, New York, USA, Futura Publishing Company, 1985, pp.530-531.

45. Parker DP, Kaplan MA. Demonstration of the supranormal period in the intact human heart as a result of pacemaker failure. Chest 1971;59: 461-466.

46. Barold SS, Falkoff MD, Ong LS, et al. Normal and abnormal patterns of ventricular depolarization during cardiac pacing. In SS Barold (Ed). Modern Cardiac Pacing. Mount Kisco, New York, USA. Futura Publishing Company, 1985, pp. 554-555.

47. Dohrman, Goldschlager N. Metabolic and pharmacologic effects on myocardial stimulation threshold in patients with cardiac pacemakers. In SS Barold (Ed) Modern Cardiac Pacing, Mount Kisco, New York, Futura Publishing Company, 1985, p.166.

48. Klein HO, Di Signi E, Kaplinsky E, et al. The Wenckebach phenomenon between electric pacemaker and ventricle. Br Heart J, 1976;38: 961-965.

49. Sutton R, Bourgeois I. The Foundations of Cardiac Pacing. Mount Kisco, NY, Futura Publishing Company, 1991, p.275.

50. Van Gelder BM, Bracke FALE, El Gamal MIH. Adapter failure as a cause of pacemaker malfunction. PACE 1993;16: 1961-1965.

51. Gould L, Patel S, Gomes GI, et al. Pacemaker failure following external defibrillation. PACE 1981; 4: 575-577.

52. Adamec R, Haefliger JM, Killisch JP, et al. Damaging effect of therapeutic radiation on programmable pacemakers. PACE 1982; 5: 146-150.

53. Lasala AF, Fieldman A, Diana DJ, et al. Gas pocket causing pacemaker malfunction. PACE 1979; 2: 183-185.

54. Kreis DJ, LiCalzi L, Shaw RK. Air entrapment as a cause of transient cardiac pacemaker malfunction. PACE 1979; 2: 641-644.

55. Massumi RA, Beeson CR, Udhoji VN. Mechanically mediated pacemaker pseudomalfunction. PACE 1986; 9: 316-321.

56. Vera Z, Mason DT, Awan N, et al. Perforation of the right ventricle by endocardial pacemaker electrodes: clinical observations with special reference to the role of intracardiac electrography. In: Meer C (Ed) Cardiac pacing, Proceedings of the 6th World Symposium on Cardiac Pacing. Ch 14-15, Pacesymp, 1979.

57. Ven Kataraman K, Bilitch M. Intracardiac electrocardiography during permanent pacemaker implantation: predictors of perforation. Am J Cardiol 1979; 44: 225-230.

58. El Gamal MIH, van Gelder LM. Suppression of an external demand pacemaker by diaphragmatic myopotentials; A sign of electrode perforation ? PACE 1979; 2: 191-195.

59. Bracke F, van Gelder B. Treatment of perforation and cardiac tamponade in the chronic phase after implantation of a tined ventricular lead.(abstract) Neth J Cardiol. 1993;6: 128. 60. Szabo Z, Solti F. The significance of the tissue reaction around the electrode on the late myocardial threshold. In M. Schaldach and S. Furman (Eds) Advances in Pacemaker Technology. New York, Springer-Verlag, 1975, 273.

61. Stokes K, Graf J, Bornzin G, et al. A steroid eluting low threshold, low polarizing electrode. PACE 1983; 6: A-65.

62. Van Gelder LM, El Gamal MIH, Tielen CHJ. Long term follow up of myocardial screw-in electrodes.(abstract) PACE 1989;12: 1201.

63. Snow N. Acute myocardial ischemia during pacemaker implantation: Implication for threshold determination and potential complications. PACE 1983; 6: 35-37.

64. Aylward P, Blood R, Tonkin A. Complications of defibrillation with permanent pacemaker in situ. PACE 1979; 2: 462-464.

65. Palac RT, Hwang MH, Klondnycky ML, et al. Delayed pulse generator malfunction after D.C. countershock. PACE 1981; 4: 163-167.

66. Slepian M, Levine JH, Watkins L, et al. *Automatic implantable cardioverter defibrillator/permanent pacemaker interaction: Loss of pacemaker capture following AICD discharge.* PACE 1987; 10: 1194-1197.

67. Trouton TG, Allen JD, Young IS, et al. *Altered cardiac oxygen extraction, lactate production and coronary blood flow after large dose transthoracic DC countershocks.* PACE 1993; 16: 1304-1309.

68. Westerholm CJ. *Threshold studies in transvenous cardiac pacemaker treatment.* Scand J Thorac Cardiovasc Surg (Suppl). 1971; 8: 1-12.

69. Dohrmann ML, Goldschlager N. *Metabolic and pharmacological effects on myocardial stimulation threshold in patients with cardiac pacemakers. In SS Barold (Ed). Modern Cardiac Pacing. Mount Kisco, New York, USA. Futura Publishing Company, 1985, p 162-166.*

70. Storm CJ, van Mechelen R. *A severed pacemaker lead entrapped in a hepatic vein.* PACE 1993; 16: 1349-1353.

71. Das G, Eaton J. *Pacemaker malfunction following transthoracic countershock.* PACE 1981; 4: 487-490.

72. Venselaar JLM, van Kerkoerle HLMJ, Vet AJTM. *Radiation damage to pacemakers from radiotherapy.* PACE 1987; 10: 538-542.

Chapter V. **Timing in Single Chamber Systems**

Introduction
Timing intervals plays an essential role in the evaluation of pacemaker sensing. For inhibited systems (AAI,VVI) a general rule is that prolongation of the escape interval is called oversensing. A coupling interval between an intrinsic cardiac event and a following pacemaker stimulus shorter than the escape interval is defined as undersensing. For triggered systems (AAT,VVT), in general, the interval between pacemaker stimuli is equal to or shorter than the escape interval even for a normally functioning pacing system. Under specific circumstances a pacing interval longer than the escape interval can be encountered, which is also associated with oversensing. If the pacing interval is shorter than the escape interval, the morphology of the QRS complex determines normal or abnormal function. Pseudofusion appearance of the QRS complex indicates normal function, while effective stimulation indicates oversensing. Undersensing in triggered systems is indicated by the disappearance of the pacemaker stimulus in intrinsic activity.

The purpose of this chapter is to illustrate the electrocardiographic appearance of sensing abnormalities and to summarize the sources and mechanisms. Analysis of oversensing is important, to discriminate between benign and malign causes, which can be present on the ECG in a similar way. Oversensing caused by physiological signals (e.g. T-wave, myopotentials) can generally be considered benign. In most of the cases the problem can be corrected by reprogramming the pulse generator. Oversensing caused by changes in lead impedance should be considered as malign. These changes are related to either insulation defects or intermittent contacts in conductor or connector. Both causes might finally deteriorate into a complete wire fracture and often require prophylactic replacement of the lead(s), especially in pacemaker dependent patients.

In the analysis of electrocardiographic undersensing, oversensing producing interference mode pacing (asynchronous stimulation), should be excluded first as the possible cause. If oversensing is the cause of electrocardiographical undersensing, correction of the problem should be attempted by decreasing sensitivity, instead of increasing sensitivity of the pulse generator. The latter can only aggravate the phenomenon. For the same reasons mentioned in the analysis of oversensing, lead related undersensing should be distinguished from other, more benign causes. Undersensing, even when a benign cause is diagnosed, should be corrected as soon as possible, because of the potential risk of inducing arrhythmias.

5.1 Definitions

Escape interval
The escape interval is defined as the time between a paced or sensed cardiac event and the subsequent pacing stimulus in the same chamber, provided there is no hysteresis. The escape interval is measured in milliseconds.

Hysteresis
In cardiac pacing hysteresis is the extension of the escape interval after a sensed event. Hysteresis increases the escape interval following an intrinsic event to allow more time for intrinsic activity.

Refractory period
The time after a paced or sensed event during which the escape interval timer cannot be reset by any sensed event. The refractory period consists of two parts, the absolute refractory period and the relative refractory period.

Absolute refractory period
During this period, initiated by a sensed or paced event, the pulse generator is unable to sense and the escape interval cannot be reset. The absolute refractory period is also called blanking period.

Relative refractory period
The last part of the refractory period, is sometimes called the noise sampling period, during which sensing is possible. A sensed event during the relative refractory period resets the refractory period, but does not reset the escape interval.

Rate limit
The maximum rate at which permanent stimulation can be performed. Protection circuit against high rate failures (pacemaker runaway, see chapter VII 7.1) and high rate pacing in the triggered mode (AAT,VVT). In some pulse generators the rate limit can be temporarily disabled in order to perform overdrive pacing.

Interference rate
Reversion of the pacemaker to asynchronous pacing due to sensing of extraneous electrical ("interference") signals. Signals should occur at a certain rate and be sensed in subsequent relative refractory periods. The number of sensed events necessary to reverse to asynchronous pacing is determined by the specifications of the pulse generator.

5.2 Prolongation of the escape interval in the inhibited mode (AAI,VVI)
If the escape interval of an inhibited pacing system is prolonged, there are 4 possible explanations:
1.   Intermittent no output.
2.   Irregular pulse formation.
3.   Oversensing.
4.   Hysteresis.

5.2.1 Intermittent no output.
In case of intermittent no output, the length of the prolonged interval will be double, triple or an exact multiple of the escape interval (Fig. 5.1). This can be explained by the fact that there is undisturbed timing of a normally functioning pulse generator delivering its impulse into a transiently disrupted circuit. Reversion to an asynchronous pacing mode by, for instance, magnet application will have no effect on the ECG and prolongation of the escape interval will be maintained. Intermittent no output, caused by intermittent discontinuity in the lead system, is rarely observed as an isolated phenomenon. In most cases it will be associated with oversensing, which will be observed more frequently and usually is the primary manifestation of intermittent discontinuity (see paragraph 5.8.3).

Lead II

840   1680
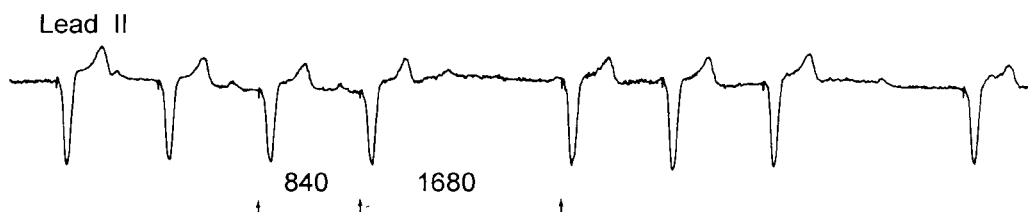
Fig. 5.1. Single channel ECG (lead II) of a patient with an implanted VVI pacemaker, showing prolonged escape intervals. The prolonged interval is exactly double the escape interval, indicating intermittent no output and not oversensing. Pacemaker function was not affected by magnet application.

5.2.2 Irregular pulse formation

Irregular pulse formation resulting in prolongation of the escape interval is rarely encountered in modern cardiac pacemakers. It has been observed in older types of pacemakers due to component failure in the generator. Pulse formation is irregular and there is no relation between the escape interval and the prolonged interval. Magnet application has no effect on the irregularity (Fig 5.2).
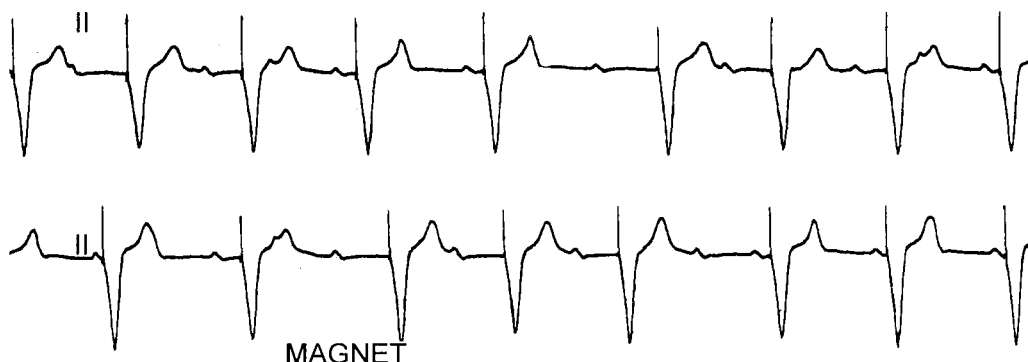
MAGNET

Fig. 5.2. Two single channel ECGs (lead II) of the same patient with an implanted VVI pacemaker. The upper channel shows irregular stimulation with prolongation of the escape interval. After magnet application (lower tracing) irregular pacing is maintained, indicating irregular impulse formation (pulse generator malfunction).

5.2.3 Oversensing

If prolongation of the escape interval is caused by oversensing, regular pacing with a fixed stimulus interval will be observed after application of a magnet or by programming the pacemaker to the asynchronous mode (Fig. 5.3). The pacing interval during magnet application can be different from the programmed escape interval,. depending on the specifications of the pulse generator.

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5.2.4 Hysteresis

In cardiac pacing hysteresis is defined as the extension of the escape interval after a sensed intrinsic event. Hysteresis increases the escape interval following an intrinsic event to allow more time for intrinsic activity te be sensed by the pacing system. If no intrinsic event occurs before the end of the hysteresis period (which is longer than the escape interval) the pacemaker will deliver an output pulse and pacing at the programmed escape interval will be resumed
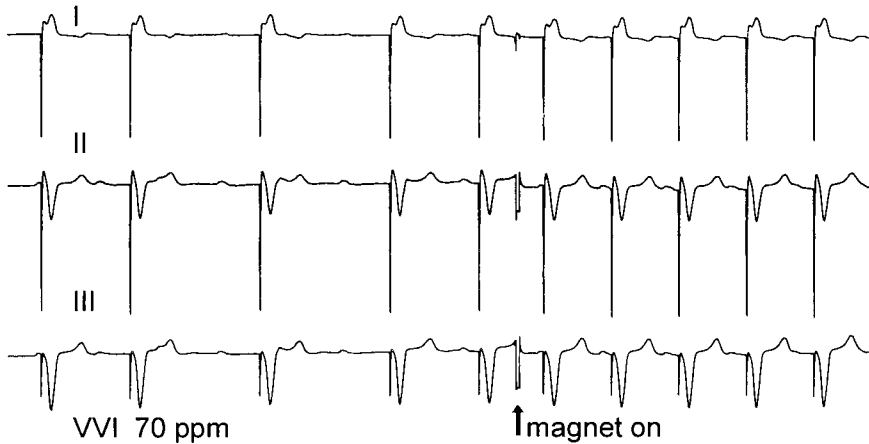
Fig. 5.3. Three channel ECG (leads I,II,III) of a patient with an implanted VVI pacemaker showing prolongation of the escape interval. Regular pacing is restored after magnet application, indicating oversensing.

5.3 Causes of prolongation of the escape interval by oversensing

Basically all causes of oversensing have in common the fact that a voltage across the anodal and cathodal electrode of a pacemaker is built up and reaches or exceeds a certain level. This level is determined by the sensitivity of the pulse generator. Exceeding this level resets the escape interval. However, it is not only the amplitude of the signal that determines sensing by the pulse generator but also the frequency content of this signal. This is explained by the input filter characteristics of the pacemaker that has its maximum sensitivity for signals between 10 and 100 Hz.

Sources of interference causing oversensing can be divided into two groups:

1.   Interference from exogenous signals
2.   Interference from endogenous signals

5.4 Interference from exogenous signals

Exogenous signals are defined as signals whose origin is located outside the human body. Exogenous signals can be coupled to the pacing system directly because of a direct contact between the interference source and the patient and indirectly by a magnetic or electromagnetic field [1]. The effect of directly coupled sources of interference is more likely than for indirectly coupled sources. Examples of directly coupled interference sources, frequently encountered in the hospital environment, are electrocauterization or coagulation equipment, nerve or muscle stimulators etc. (Fig. 5.4).

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Application of current in medical practice should always be monitored reliably and throughout the whole procedure [2]. Leakage current from equipment or other electrical installations is able to affect pacemaker function by temporary inhibition or reversion to interference mode (see paragraph 5.12). Figure 5.5 shows reversion to interference mode caused by an inappropriately grounded lighting installation of a swimming pool.

The effect of magnetic or electromagnetic interference sources on pacing systems is difficult to predict. The influence is determined by the intensity of the interference source itself, the distance to the pacemaker patient, implant configuration, electrode configuration, shielding and sensitivity of the pulse generator [3]. Because of the variety of electromagnetic interference sources and situations in which they are encountered, it is not possible to generalize about the electromagnetic interference immunity of even a particular pacemaker. Pacemaker patients with a high electromagnetic interference work environment present a unique situation and should ideally be evaluated individually [4].
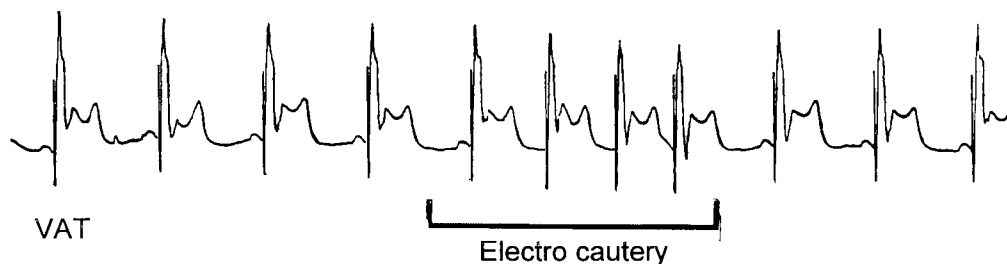
Fig. 5.4. Single channel ECG of a patient with a VAT pacemaker. During electrocautery oversensing in the atrial channel is observed, resulting in acceleration of the pacing rate.

Sources of electromagnetic interference encountered in daily life, like household equipment, anti-theft systems used in shops and libraries, weapons and metal detectors used on airports generally do not affect pacing systems [5,6]. Most communications on this subject confirming the influence of these sources are outdated or are based on anecdotal observations.
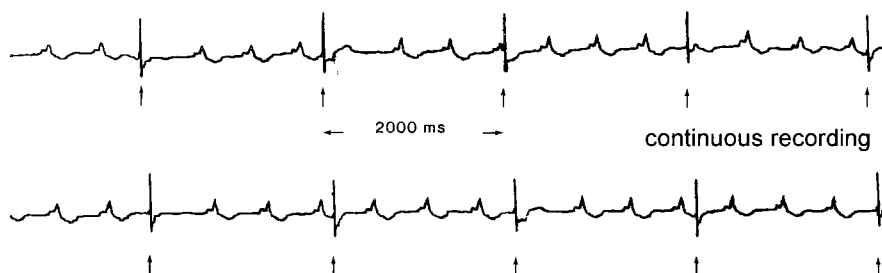
Fig. 5.5. One channel ECG, continuous recording, of a patient with an implanted VVI pacemaker. The pacemaker was programmed at a rate of 30 ppm (2000 ms); the interference rate was equal to the programmed pacing rate. Asynchronous pacing (interference rate) was observed when the patient was in the neighbourhood of an inappropriately installed lighting installation in a swimming pool. Normal function was restored after leaving the area.

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However, it will be hard to predict whether in the rapidly developing field of electronics and electronic communication new sources of electromagnetic interference will come up that will affect pacemaker function.

5.4.1 Magnetic Resonance Imaging (MRI)

An extremely strong source of magnetic and electromagnetic interference is the equipment used for magnetic resonance imaging (MRI), which is rapidly developing as an important clinical diagnostic tool. The (electro)magnetic fields generated by MR imagers are:
1. The static magnetic field
2. The pulsed magnetic field (magnetic gradients)
3. The radio frequency (RF) electromagnetic (EM) field

The static magnetic field results in reed switch closure with asynchronous pacing, which is usually not a problem. However, in selected patients with increased myocardial irritability it could induce an arrhythmia if stimulating within the atrial or ventricular vulnerable period.

The pulsed magnetic field, also called time-varying gradient fields are impressed upon the static field to obtain positional information. While the field gradient contribution to the total magnetic field is negligible, an effect can occur as these fields are rapidly applied and removed. Changing magnetic fields will produce an electrical potential in a conductor, but the induced voltage (less than 80 mV) is too low to evoke stimulation [7,8]. Although these induced potentials are theorically of sufficient amplitude to inhibit the pacing system, this effect is overruled by the reed switch closure (by the static magnetic field) with asynchronous pacing.

The most serious problem appears to be the potential for rapid stimulation. Rapid stimulation is caused by the RF pulse period, because the rapid rate in the reported cases is equal to the pulse period of the RF field [9,10] The mechanism for rapid stimulation is not absolutely clear. There are two possible explanations. The first is the direct effect of RF energy on the pacemaker circuitry resulting in abnormal circuit operation. The second explanation is that the lead(s) may act as an antenna and couple the RF energy back into the pacemaker output circuit.

The ferromagnetic components of the pulse generator will exhibit a force of attraction and torque in a magnetic field. The force of attraction has not been reported as a major problem, contraindicating the use of MRI. Heat induction at the pulse generator or electrode side, which is a theoretical possibility, has no negative effect on pacemaker function [7,11].

Although guidelines for MRI safety reported in 1991, advise exclusion of pacemaker patients from MRI examinations, more recent studies report that MRI investigations were safely performed on pacemaker patients [13,14,15,16]. To exclude the possibility of inducing rapid stimulation, in vitro tests of the particular pulse generator should be performed, prior to exposure of the patient to MRI examination. Dual chamber systems should be programmed to the single chamber mode in order to minimize the risk of interference.

5.5 Interference from endogenous signals

The sources for interference from endogenous signals can be divided into 3 groups:
1.   Signals from intra-cardiac origin.
2.   Physiological signals from extra-cardiac origin.
3.   Signals generated by the pacing system.

5.6 Oversensing by intracardiac potentials

Oversensing by intracardiac potentials can be divided into 3 groups:
1.   T wave oversensing in ventricular sensing pacemakers.
2.   R wave oversensing in atrial sensing pacemakers.
3.   P wave oversensing in ventricular sensing pacemakers.

5.6.1 T wave oversensing in ventricular sensing pacemakers

T wave oversensing [17], sensing of the T wave by the ventricular electrode, is relatively rare and is usually seen after paced beats rather than spontaneous beats probably because the polarization voltage (see paragraph 5.8.1) contributes a larger signal and therefore tends to generate a larger T wave. Besides the effect of the polarization voltage, the QT interval in a spontaneous beat is shorter than in a paced beat, so spontaneous T waves usually occur during the pacemaker refractory period. T wave oversensing is characterized by prolongation of the escape interval with a more or less constant interval (Fig. 5.6). Shortening of the refractory period will not affect the prolongation of the escape interval if this is caused by T wave sensing, while it will shorten the prolonged interval with an interval equal to shortening of the refractory period in case of afterpotential sensing.
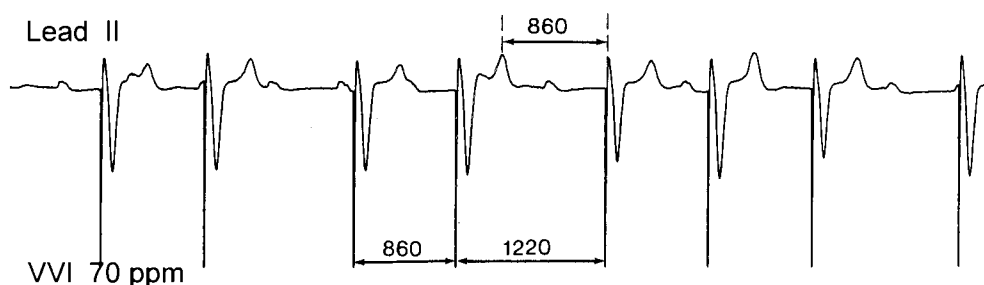
Fig. 5.6. Single channel ECG (lead II) of a patient with an implanted VVI pacemaker, showing prolongation of the pacing interval (1220 ms). All prolonged intervals are more or less the same in length and the point of oversensing corresponds with the preceding T wave; T wave oversensing.

Apparent T wave sensing, which is relatively benign, should be differentiated from prolongation of the escape interval by intermittent contacts in the conductor. The latter is often preceding complete wire fracture, which will be followed by loss of stimulation. Differentation between intermittent wire fracture and T wave oversensing can be made by prolongation of the refractory period. If the refractory period is programmed to a value longer than the QT interval the escape interval should become normal, whereas prolongation of the escape interval will persist (probably with a lower incidence) in case of intermittent wire fracture.

One should take into consideration that if a T wave is of sufficient amplitude to be sensed by the pacemaker, but occurs during the refractory period, the escape interval is not affected. However, when the T wave occurs during the relative refractory period, the refractory period will be extended. If during extension of the refractory period an intrinsic event takes place, this event will be ignored by the pacemaker and undersensing (see paragraph 5.12) can be manifested.

5.6.2 R wave oversensing in atrial sensing pacemakers

R wave oversensing can be encountered in unipolar and less frequently in bipolar atrial pacemakers [18]. The atrial electrode may sense spontaneous intracardiac signals from the ventricles (far-field sensing). Although these R waves have a relative low amplitude in the atrium, these signals can be sensed by the atrial electrode, due to a high sensitivity setting. The electrocardiographic appearance of R wave oversensing in atrial pacemakers is similar to T wave oversensing in ventricular pacemakers and results in prolongation of the escape interval by a more or less fixed value (Fig. 5.7). The same consideration for a differential diagnosis as in T wave oversensing should be made in cases of apparent R wave oversensing in atrial pacemakers [19]. Bipolar atrial sensing eliminates far-field R wave sensing in the majority of atrial sensing pacemakers.

R wave oversensing in atrial pacemakers programmed in the triggered mode (AAT) may lead to pacemaker mediated tachycardia (see chapter VII 7.4.1).
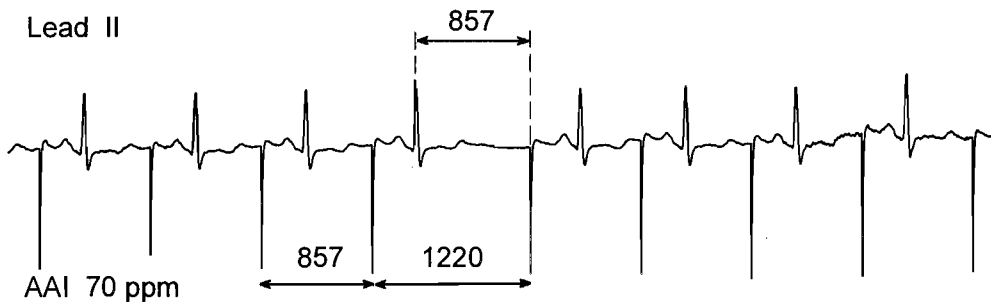
Fig. 5.7. Single channel ECG (lead II) of a patient with an implanted unipolar AAI pacemaker. The escape interval is programmed at 857 ms. Prolongation of the escape interval (1220 ms) is observed; the point of oversensing corresponds with the preceding R wave (far-field R wave sensing).

5.6.3 P wave oversensing in ventricular sensing pacemakers

P wave oversensing in ventricular pacemakers is a very rare phenomenon [20,21]. A unipolar endocardial lead positioned in the right ventricular apex never registers sufficient P wave voltage to suppress a demand circuit. Sensing of P waves may be seen in the following circumstances:

1. Electrode positioned in the right ventricular inflow tract in the proximity of the tricuspid valve;
2. Displacement or malpositioning of the lead into the coronary sinus;
3. Epicardial electrodes implanted near the atrio-ventricular groove [22].

When using bipolar endocardial leads implanted in the right ventricle with the tip of the lead not completely in the right ventricular apex, the ring electrode can be located in the tricuspid area, thus enabling P wave oversensing (Fig. 5.8).

Although the incidence of P wave oversensing is extremely low, clinical implication is high because pacemaker output can be inhibited completely, depending on the sinus rate and programmed escape interval (Fig. 5.9).
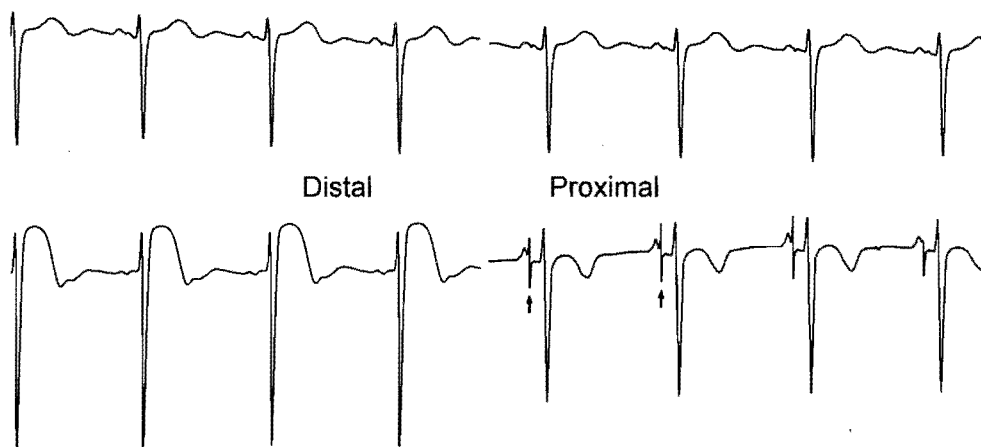
Fig. 5.8. Unipolar electrograms from a bipolar lead for permanent ventricular pacing; electrode separation 25 mm. The electrode was implanted in the right ventricular inflow tract. The distal electrogram shows a normal pattern; in the proximal electrogram a sharp deflection of the atrial depolarization (arrows) was recorded in the last part of the P wave.

Lead II

VVI 60 ppm
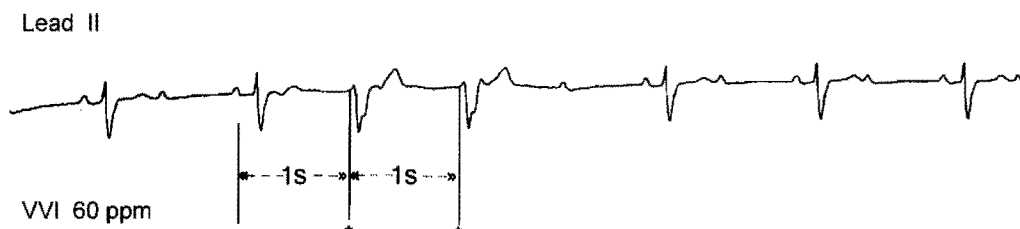
Fig. 5.9. Single channel ECG (lead II) of a patient with a bipolar VVI pacemaker. Pacemaker was programmed to a rate of 60 ppm. Intrinsic ventricular rate 46 bpm. Only two effective pacemaker stimuli are recorded. The point of oversensing corresponds with the preceding P wave.

VVT 60 ppm   Lead I
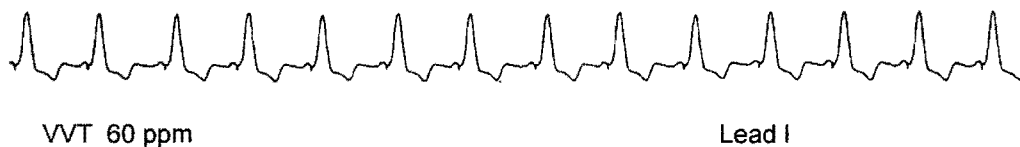
Fig. 5.10. Single channel ECG of the same patient as figure 5.9. The pacemaker is programmed to the VVT mode. Regular pacing is observed synchronously with the intrinsic P waves. This recording is the definitive proof of P wave oversensing.

P wave oversensing can be confirmed by programming the pacing system to the triggered mode (VVT). Pacemaker stimuli will be emitted synchronously with the registered P waves. In the case of continuous P wave sensing one may try not to avoid P wave oversensing but to program the pacing system to the triggered mode (Fig. 5.10). In this way the system is converted to a

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ventricular pacing system, with AV synchrony and a physiological rate response, if sinus node function is normal [23].
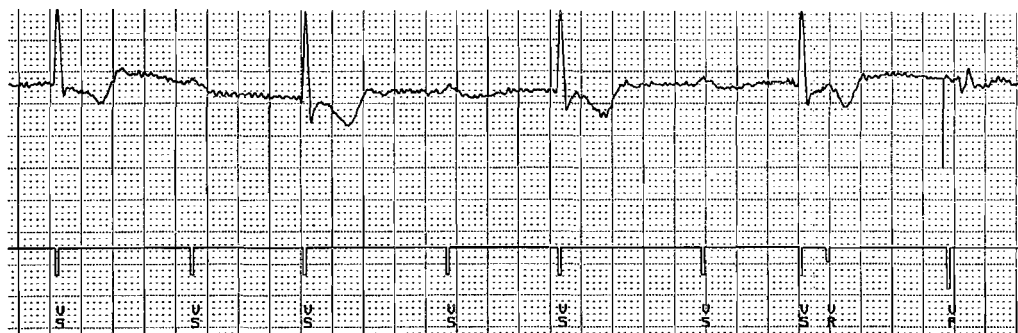
Fig. 5.11. ECG and marker channel recording of a patient with an implanted unipolar VVI pacemaker. The marker channel indicates ventricular sense events (Vs) during intrinsic P waves. Vr indicates a ventricular sense event during the relative refractory period.

CHART SPEED 25.0 mm/s
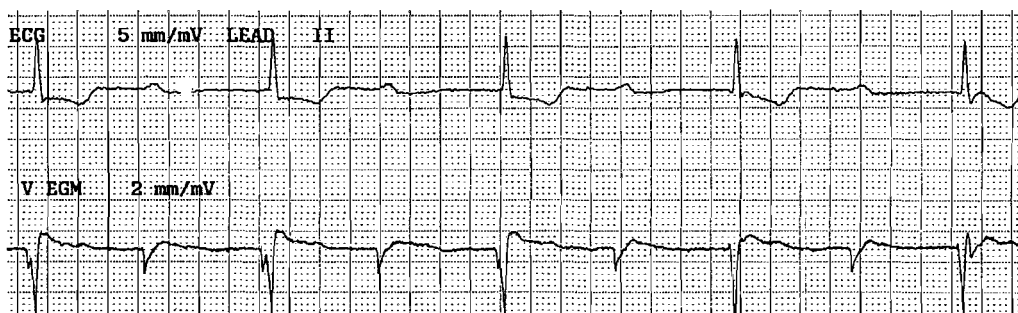
Fig. 5.12. ECG and intracardiac electrogram (same patient as figure 5.11) from the ventricular lead by pacemaker telemetry. Sharp negative P waves are recorded from the ventricular lead. The intrinsic deflection of the P wave in the intracardiac electrogram occurs simultaneously with the onset of the P wave in the surface ECG.

A very unusual case of P wave oversensing has been encountered in a patient with a unipolar VVI pacemaker [24]. The patient had two unipolar (one abandoned) leads for more than 20 year when he presented with P wave oversensing. P wave oversensing was confirmed by the marker channel of the pacemaker, indicating ventricular sense events synchronously with P waves (Fig. 5.11). Telemetry of the intracardiac ECG showed P waves in the ventricular electrogram with a relatively sharp negative deflection (Fig. 5.12). This sharp deflection occurred synchronously with the onset of the P wave on the surface ECG. From this recording it was concluded that the P wave was detected in the high right atrium. This can be explained by an insulation defect of the functioning ventricular lead, bringing the conductor coil in contact with the high right atrium.

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this insulation defect was probably caused by long lasting friction between both leads, which was observed during fluoroscopy (Fig. 5.13).

Fig. 5.13. Posteroanterior X-ray of the same patient (figures 5.11, 5.12) showing crossing of the abandoned and functional lead (arrow) at the level of high right atrium. See text for further explanation.

5.7 Oversensing from extra-cardiac physiological signals (myopotentials)

Every skeletal muscle generates electrical potentials during contraction. When the muscle is in direct contact with or in the direct vicinity of one of the electrodes of the pacing system, these potentials can be detected by the sensing amplifier and result in inhibition. The period of inhibition depends on the time of contraction and the amplitude of the potentials generated during contraction. In a minority of patients, detection of myopotentials will result in interference rate reversion (see paragraph 5.12). Because myopotentials can be detected from both electrodes, oversensing from myopotentials can be divided into:
1.   Anodal oversensing.
2.   Cathodal oversensing.

5.7.1 Anodal oversensing of myopotentials

The best known form of myopotentential oversensing is oversensing in unipolar systems which has already been reported in 1972 [25]. In unipolar systems the myopotentials are detected by the case of the pulse generator, the (anodal) electrode, that is in contact with one of the skeletal muscles e.g. the pectoral muscle (Fig. 5.14). The reported incidence of musculoskeletal inhibition of unipolar demand pacemakers is up to 69% during normal daily activity, objectivated by Holter recording [26]. However, in only 12% of these patients was inhibition associated with symptoms.

Muscle interference can be demonstrated in the large proportion of patients with unipolar pulse generators by instructing the patient to contract his pectoral or abdominal muscles, while recording the electrocardiogram. Inhibition by myopotentials has been decreased by partially isolating the pulse generator case and the improvement in amplifier design, as well as programmability of pacemaker sensitivity, but has never been eliminated completely in unipolar systems. In the case of symptomatic pacemaker inhibition by muscle potentials programming a

lower sensitivity (less sensitive) setting can be attempted [27]. If this fails because undersensing of the intrinsic rhythm occurs, especially in atrial pacemakers employing high sensitivity settings, symptoms can be prevented by programming the pacemaker to the triggered mode (AAT,VVT). In the triggered mode acceleration of the pacing rate will be induced by myopotentials but this rarely results in symptoms.

One should take into consideration that reversion from inhibited to triggered mode increases current consumption if an intrinsic rhythm is partially present, with a negative effect on pacemaker longevity [28]. Skeletal muscle oversensing from muscles adjacent to the pulse generator cannot be demonstrated in bipolar systems under ordinary circumstances, because the pacemaker case is no part of the electrical circuit. Reports documenting inhibition in bipolar systems from myopotentials originating from the muscle adjacent to the pulse generator were associated with insulation defects [29,30].

AAI 72 ppm

contraction pectoral muscle
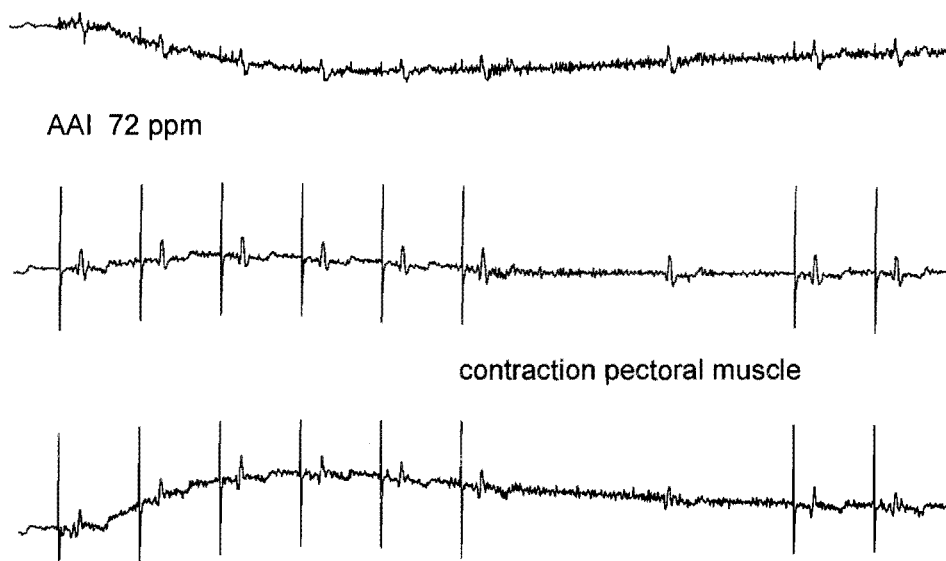
Fig. 5.14. Three channel ECG of a patient with an implanted unipolar atrial pacemaker. During contraction of the pectoral muscle adjacent to the pulse generator, inhibition of the pacemaker is observed.

5.7.2 Cathodal oversensing of myopotentials

On rare occasions oversensing from myopotentials can occur at the cathodal electrode of ventricular pacemakers. The ventricular electrode, traditionally implanted in the right ventricular apex, is in the direct vicinity of the diaphragm, which also generates myopotentials during inspiration. Because normally there is no direct contact, and there is sufficient distance between the pacing electrode and the diaphragm this phenomenon is only rarely encountered [31]. Circumstances enabling sensing of myopotentials generated by the diaphragm are:

1. Perforation of the pacing electrode [32].
2. Thin wall of the right ventricular myocardium.
3. High sensitivity of the pulse generator [33].

The diagnosis of thin wall of the right ventricle can only be made by exclusion. If X-ray and intracardiac electrogram exclude perforation of the lead as a cause of oversensing from diaphragmatic myopotentials, the most likely explanation is the thickness of the right ventricular wall. It is obvious that oversensing from diaphragmatic myopotentials can be encountered in unipolar as well as bipolar pacing systems (Fig. 5.15).
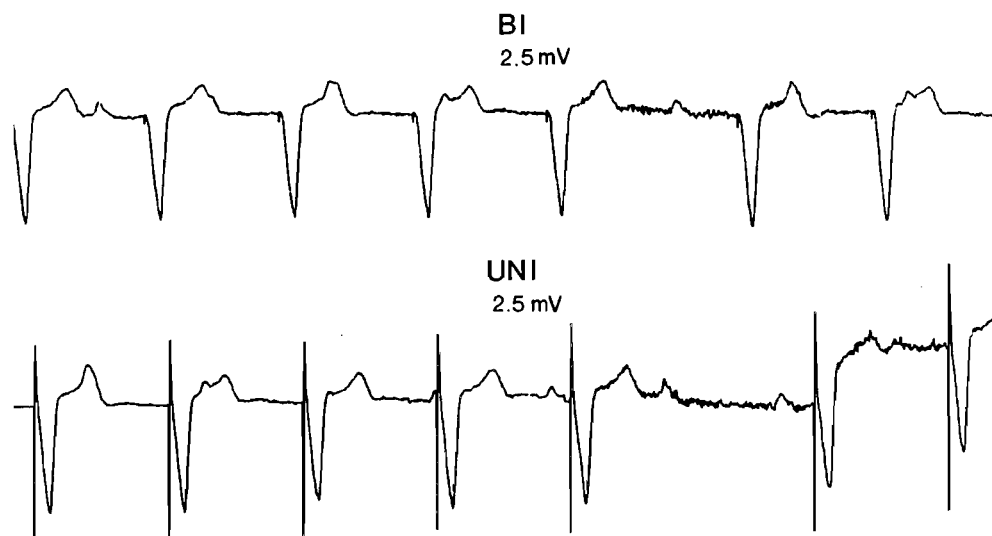
Fig. 5.15. Two ECG's of the same patient with a bipolar VVI pacemaker. In the upper tracing the electrode configuration is programmed bipolar (bi) and the lower tracing in the unipolar (uni) configuration. Prolongation of the escape interval is recorded in the bipolar and in the unipolar configuration. This indicates oversensing from the cathodal electrode. In this patient it was related to respiration and caused by myopotentials from the diaphragm.

5.8 Oversensing from signals generated within the pacing system

There are two types of signals generated by the pacing system that are able to reset the escape interval timer and prolong the pacing interval. The signals are caused by the stimulation pulse itself called afterpotential or by abrupt changes in the impedance within a pacing system.

5.8.1 Oversensing of afterpotentials

After stimulation of the myocardium there are 3 mechanisms that determine the electrical potential at the cathodal electrode.

1. Stimulation of the myocardium charges the electrode-tissue interface to a certain potential [34]. During delivery of the cathodal stimulation pulse, positively charged ions accumulate at the electrode-myocardium interface. The negatively charged electrode and the positively charged ions are both surrounded by water molecules. The effect of this double layer is comparable with a capacitor (Helmholtz capacitance). Parallel with this capacitance a resistance (Faraday resistance) is created by oxydo-reduction reactions [35].

When a stimulation pulse is delivered and a current is passing the electrode a charge and voltage (polarization voltage) is built up over the capacitance (Fig. 5.16). The level of charge and voltage

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depend on the amount of charge delivered by the stimulation pulse, the geometry and material of the stimulation electrode, the stimulation rate and the elapsed time after stimulation. This voltage (afterpotential) dissipates exponentially after the stimulation pulse.

2. The recharge current of the ouput capacitor also passes the electrode-tissue interface. The recharge current is relatively high during the fast recharge time (chapter III 3.3) but only for a period of 10 ms or less. It is determined by the amount of electrical charge delivered during stimulation and the impedances of the various components in the pacemaker-electrode-tissue circuit [36].

3. Depolarization and repolarization of the adjacent myocardium. Depolarization evoked by stimulation occurs during the absolute refractory period of the pacemaker (blanking period) but repolarization (T wave) takes place in the alert or relative refractory period of the pacing system. If the level of the sum of potentials at the end of the refractory period is above the level of the sensitivity of the pulse generator, it will be sensed and reset the escape interval timer.
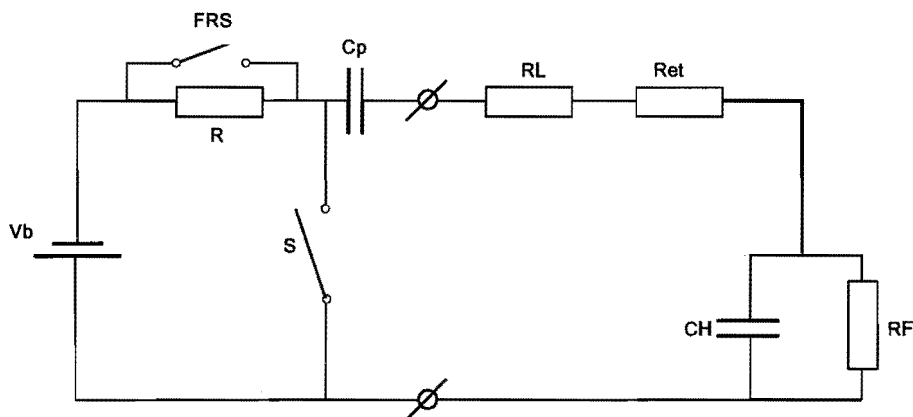
Fig. 5.16. Diagram of the simplified equivalent circuit model, in which Ret is the electrode-tissue resistance, RI is the conductor resistance, Ch the Helmholtz capacitance and Rf the Faraday resistance. Ch and Rf are related to the electrical double layer created by the stimulation current passing the electrode-tissue interface. R = internal resistance of the pulse generator, FRS = fast recharge switch, Cp = output capacitor, S = switch, closed during stimulation.

Pure afterpotential sensing is most likely when the afterpotentials changes most rapidly. Therefore, afterpotentials sensing is most likely immediately after the return of the sensing function of the pacemaker, i.e. immediately after the blanking or absolute refractory period. If the pacemaker had a noise sampling interval starting at the end of the blanking period and ending at the end of the refractory period, sensing of after potentials will result in prolongation of the refractory period and not in extension of the escape interval.

Afterpotential sensing can be eliminated by :
1)   Decreasing sensitivity.
2)   Decreasing pacemaker output; amplitude, pulse width or both.
3)   Decreasing stimulation rate.

On some occasions the afterpotentials may aggravate T wave sensing. After expiration of the refractory period the potential at the electrode is determined by the afterpotential and the

intracardiac repolarization (T wave). If the afterpotential and T wave are phasically equal, the potential at the electrode is the sum of both. Reducing the afterpotential may reduce the total potential in such a way that it is below the sensing capability of the pulse generator.

5.8.2 Oversensing by abrupt changes in lead impedance

Sudden changes in pacing system impedance are able to generate voltage changes between the pacing electrodes large enough to be sensed by the pacemaker [37]. There are several explanations given for the origin of this mechanism:

1. Interruption of the polarization voltage after the pacemaker impulse [38].
2. Interruption of the recharge current, after discharge of the output capacitor of the pacing system [39].
3. Disturbance of the much smaller permanent DC voltage across the electrodes in the absence of pacemaker stimuli [38].

The DC voltage in the pacing system is explained by the physical law, that dissimilar metals in an electrolytic solution are able to generate potentials. Whatever the exact mechanism in a specific situation, they have in common the presence of a voltage between anode and cathode of the pacing system, other that the stimulation voltage. A sudden change in impedance will result in an abrupt variation of this voltage. This variation in voltage can be detected by the sensing amplifier resulting in prolongation of the pacing interval.

Although changes in system impedance can be related to mechanical heart action and aggravated by ventricular (or atrial) systole or diastole, they are in general randomly distributed throughout the cardiac cycle. This implies that oversensing by these changes cannot be prevented by prolongation of the refractory period, this in contrast to for example T wave (in ventricular pacemakers) or R wave (in atrial pacemakers) oversensing.

5.8.3 Causes of abrupt changes in lead impedance

Variation in system impedance can be caused by short lasting, complete or partial, interruption in the conductor or connector of the lead. Partial fracture can be encountered in multifilar leads, where one or more of the conductor strands are broken and the intermittent contact between the broken strands is responsible for the impedance variation.
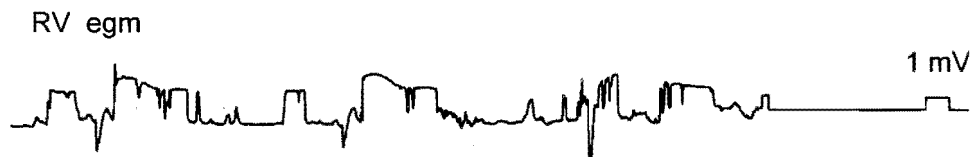
Fig. 5.17. Intracardiac ventriculair electrogram (RV egm) from a broken lead with a fluid bridge between both fractured ends. The intermittent contact between both fractured ends are responsible for the spurious signals.

Also in broken monofilar leads with an intact insulation, where a fluid bridge, forms a contact between both ends is able to generate potentials of sufficient amplitude to be sensed by the pacemaker (Fig. 5.17). Intermittent loose connection of the lead connector with the pacemaker receptacle can be a cause of varying impedance resulting in oversensing.

Changes in system impedance can also occur in an electrically intact electrode that touches a second (inactive) electrode. Intermittent contact between the two electrodes may create spurious signals which can be sensed by the pacing system resulting in oversensing [40]. Oversensing is also described by movements of a fixation mechanism in a stimulation electrode.

In this particular electrode the tip contains two fine wires attached to a movable pin placed within a metallic cylinder (Fig. 5.18). Once positioned, the lead stylet is used to eject the wires in order to achieve active fixation. Movement of the metal pin within the cylinder causes impedance changes responsible for pacemaker inhibition (Fig. 5.19) [41,42].
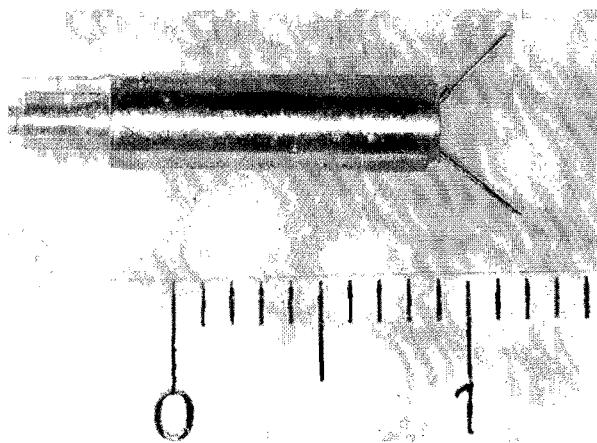
Fig. 5.18. Photograph of the Biotronik E-60-I electrode with the fixation wires in the ejected position. Fixation wires are attached to a movable (metal) pin within the cylinder, which forms the stimulation electrode.

Signals caused by abrupt impedance changes are rarely recorded on the surface ECG. In case of an intermittent contact in a lead connector, however, the ECG represented in figure 5.20 was recorded during manipulation of the pulse generator.

It shows spurious signals during manipulation, that are sensed by the pacemaker. Because these signals are in general not visible in the ECG, telemetry of the intracardiac electrogram is a valuable diagnostic tool to detect signals responsible for oversensing
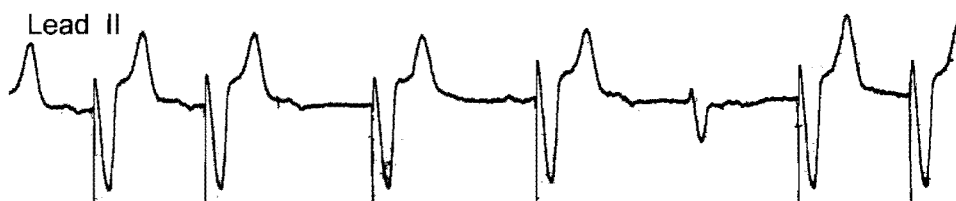
Lead II

Fig. 5.19. One channel ECG (lead II) of a patient with a Biotronik E-60-I lead (see also figure 5.18) implanted in the right ventricle. There is prolongation of the pacing interval with a more or less fixed interval. The electrocardiographic pattern is similar to T wave sensing, probably because of violent movement of the electrode during the end of ventricular systole, thus generating spurious signals.

Changes in lead impedance are most likely to occur through intermittent discontinuity of the conductor in the lead. Decrease in lead impedance has also been reported due to insulation defects [43]. Even a very small defect (Fig. 5.21) in a polyurethane lead caused by stress

corrosion cracking was able to generate impedance changes, most probably caused by movement of the lead. Recording of the intracardiac ECG from this lead showed different spurious signals with varying morphology (Fig. 5.22).

The amplitude of these spurious signals was small, less than 2 mV but pacemaker oversensing was observed even at a sensitivity level of 5 mV. Although exact measurement of the spurious signals by an electrocardiograph is debatable, in this case it is very likely that not the spurious signal itself but the effect on the recharge current or the pacemaker afterpotential combined with impedance changes was the final cause of oversensing.
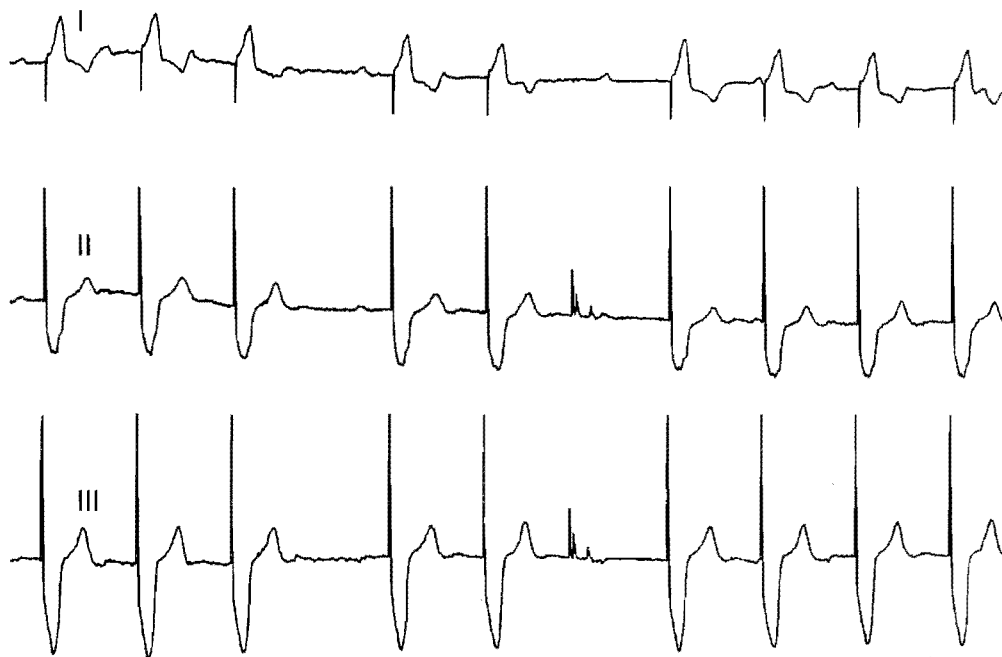
Fig. 5.20. Three channel ECG (lead I, II, III) of a patient with an implanted unipolar VVI pacemaker that showed inhibition of the pacing system during manipulation. Spurious signals visible on the surface ECG are generated in the pulse generator-lead connector.

In bipolar systems changes in lead impedance can be observed in leads showing an insulation defect between the anodal and cathodal conductor. In particular leads using polyurethane as an insulation material between both conductors, are prone to these defects [44]. If oversensing is observed in bipolar leads and if this is associated with a decrease in lead impedance, it is strongly suggestive of an insulation defect between the two conductors.

Figure 5.23 demonstrates spurious signals intracardially recorded from the distal electrode of a bipolar lead. The same type of signals, however, with a lower amplitude are recorded from the proximal electrode (Fig. 5.24). By making an external electrical connection between the two conductors, the impedance changes between the conductors are eliminated, the recorded electrogram is normalized and no spurious signals are visible (Fig. 5.25). This proves that the spurious signals are caused by changes in the lead impedance.

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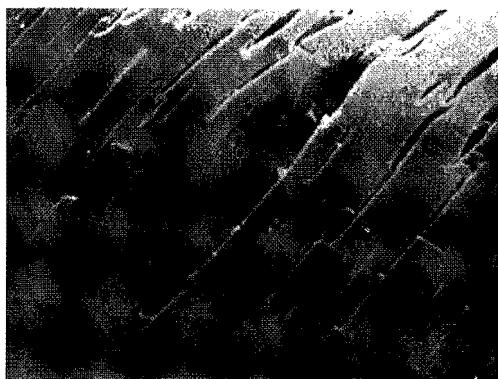
Fig. 5.21. Scanning electronic microscope (SEM) photograph of a tear in a polyurethane lead. Dimension of the tear 0.1 x 0.7 mm. This lead was removed from a patient showing oversensing in a AAI pacemaker. See also figure 5.22.

RA egm

Fig. 5.22. Right atrial electrogram (RAE) recorded from an atrial lead showing oversensing. Spurious signals with an amplitude < 2 mV were recorded. Signals were caused by an insulation defect (see figure 5.21). Of note is the fact that the majority of the spurious signals are recorded during ventricular systole, which suggest that movement of the lead affected the size of the tear and thus the change in lead impedance.
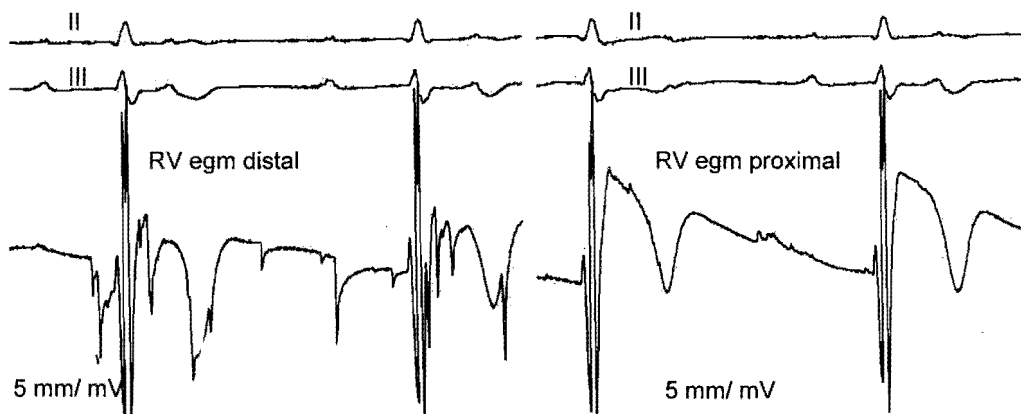
Fig. 5.23. Intracardiac electrogram recorded from the distal electrode of a bipolar ventricular lead. Spurious signals are recorded during the complete cardiac cycle. The pacing system showed oversensing caused by an insulation defect between both conductors.

Fig. 5.24. Intracardiac electrogram from the proximal electrode (same lead as figure 5.23) also showing spurious signals with a much lower amplitude.

5.9 Prolongation of the escape interval in the triggered mode (AAT/VVT)

In single chamber triggered pacing systems the interval between pacemaker stimuli is equal to or shorter than the programmed escape interval. However, an interval between pacemaker stimuli shorter than the programmed escape interval can indicate normal function or oversensing. In the case of normal function, the evoked pacemaker pulse will give rise to a pattern of pseudofusion; the pacemaker pulse is emitted after sensing the intrinsic cardiac activity. In the case of oversensing the pacemaker impulse is triggered by a "false" signal and will result in stimulation of the adjacent myocardium, provided that the myocardium is not refractory at the time of pulse delivery.
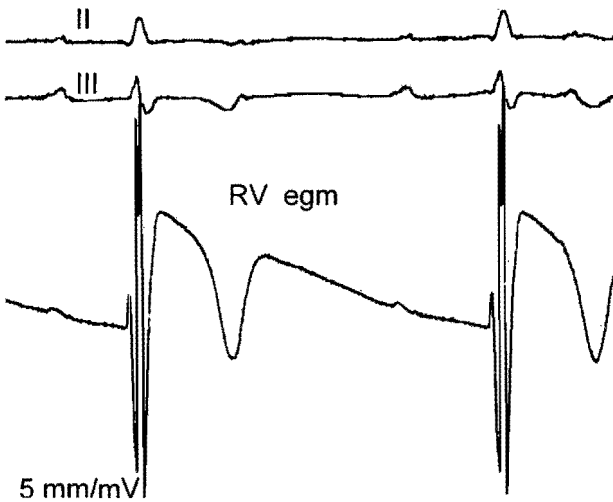
Fig. 5.25. Intracardiac electrogram of the same lead as figure 5.23 and 5.24 after an external short circuit between both conductors,which eliminated the impedance changes. The electrogram is normalized and free of spurious signals.

Under certain circumstances, however, prolongation of the pacing interval can be observed in single chamber triggered pacing systems. Figure 5.26 illustrates prolongation of the escape interval in a patient with a VVT pacemaker in the relaxation phase after pectoral muscle contraction. During contraction of the pectoral muscle, adjacent to the pulse generator case, shortening of the pacing interval is observed, which is normal pacemaker function if the myopotentials are sensed by the pulse generator. In the relaxation phase a pacing interval of 1250 ms is recorded. The explanation for this phenomenon is as follows:

After every paced event a refractory period is initiated. In this example, the refractory period was 325 ms and the escape interval 857 ms. During the refractory period all signals are ignored. Coinciding with the start of the refractory period, the upper rate limit counter (which is the protection mechanism against undesired high pacing rates) is started. In this example the upper rate limit was 129 ppm, which is equal to 465 ms. If a sensed event is detected outside the refractory period but still within the upper rate limit interval, the escape interval will be reset but no pacemaker pulse will be emitted. In practice this means, that if the upper rate interval is longer than the refractory period, a sensing inhibiting window is created. In our example there is a sensing inhibiting window of 140 ms (465 - 325 ms). Depending upon the timing of the sensed event, immediately after the refractory period (325 ms) or at the end of the upper rate interval (465 ms), the pacing interval will be prolonged from 1182 (857+325 ms) to 1322 ms (857+465 ms).

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Although contradictory to the definition of the triggered pacing mode this phenomenon has to be considered as normal pacemaker function [45].
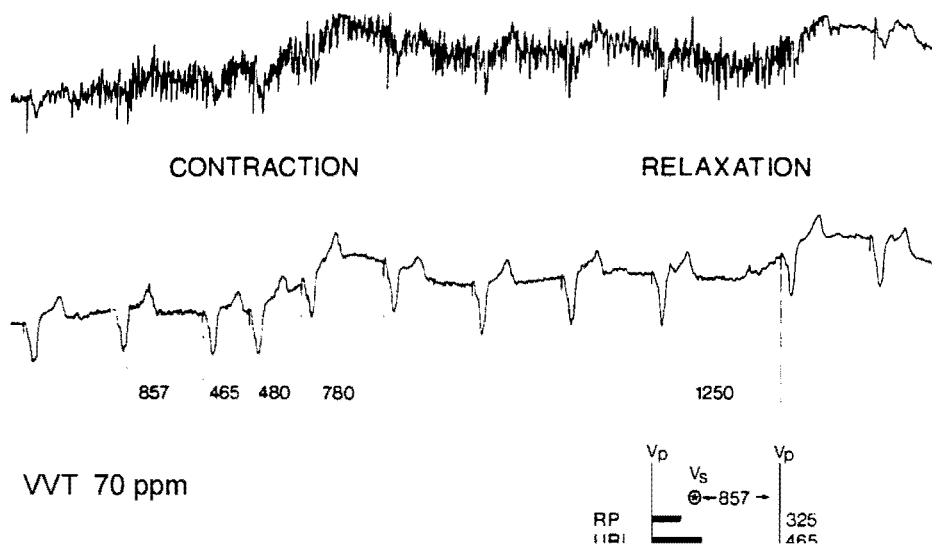
Fig. 5.26. Two lead ECG of a patient with an implanted unipolar VVT pacemaker; escape interval 857 ms. During contraction of the pectoral muscle acceleration of the pacing rate was recorded. However, during relaxation a prolonged pacing interval (1250 ms) was observed. See text for further explanation.

5.10 Coupling interval between an intrinsic event and a subsequent paced event, shorter than the escape interval (undersensing)

Shortening of the coupling interval between intrinsic activity and subsequent pacemaker stimuli, electrocardiographically known as undersensing can be divided into three categories, related to the cause of undersensing:

1. The intrinsic intracardiac electrogram is of sufficient amplitude to be sensed by the pacemaker but occurs in the refractory period of the pacing system.
2. The intrinsic intracardiac electrogram is not of sufficient amplitude and/or slew rate to be sensed by the pacemaker at the programmed sensitivity level.
3. The intrinsic intracardiac electrogram is of sufficient amplitude and/or slew rate to be sensed by the pacemaker but the signal is attenuated by electrical dysfunction of the lead or the pulse generator.

Magnet application resulting in asynchronous pacing (VOO/AOO), thus presenting electrocardiographically undersensing in the presence of intrinsic cardiac activity, should not be considered undersensing.

5.11 Effect of the total refractory period

A coupling interval between an intrinsic event (R wave for ventricular inhibited pacemakers and P waves for atrial inhibited pacemakers) shorter than the escape interval is electrocardiographically defined as undersensing. An additional condition should be added to

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this definition; the intrinsic event has to occur outside the refractory period of the pulse generator. This implies that the refractory period must be known for the correct interpretation of the pacemaker ECG.

Figure 5.27 illustrates shortening of the escape interval in a VVI pacemaker. The QRS complex preceding the pacemaker stimulus is not sensed by the pacemaker, while the interval from the previous QRS complex to the pacemaker stimulus corresponds with the escape interval. Due to the high intrinsic rate, the coupling interval between two consecutive QRS complexes is shorter than the refractory period of the pacemaker. This explains why the second QRS complex is not sensed. From the previous definition of undersensing this example should not be considered to be undersensing, but normal pacemaker function, in spite of the shortening of the coupling interval.
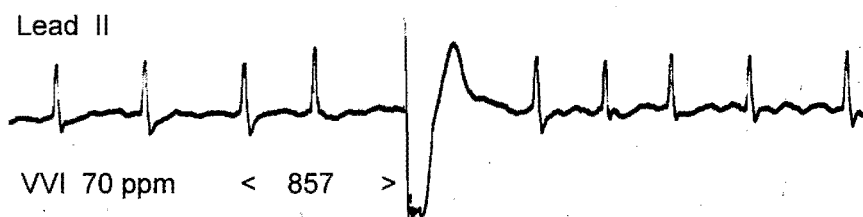
Fig. 5.27. One channel ECG of a patient with an implanted VVI pacemaker. Escape interval is 857 ms (70 ppm); refractory period 400 ms. The intrinsic QRS complex preceding the stimulated QRS complex is shorter than the escape interval. The interval between the apparently sensed QRS complex and the non-sensed complex is shorter than the refractory period and cannot be sensed by the pacemaker. Normal pacemaker function.

5.12 Effect of the relative refractory period

If a sense event is detected in the relative refractory period, the escape interval is not reset. The sense event, however, initiates a new refractory period. This implies that the effective refractory period of a pacemaker is prolonged. The prolongation of the refractory period is determined by the specifications of the pulse generator. Prolongation of the refractory period by sensing in the relative refractory period is demonstrated in figure 5.28. During ventricular pacing two chest wall stimuli with a coupling interval of 300 and 220 ms respectively were applied to the patient. The total refractory period of the pulse generator was 325 ms. The first chest wall stimulus (in the relative refractory period) initiated an additional refractory period. In the relative part of this refractory period, the second chest wall stimulus was sensed. The latter again initiated a refractory period, in which the ventricular premature contraction took place. The ventricular premature contraction was also sensed in the relative refractory period and the escape interval was not affected by any of the three sensed events. The pacemaker marker channel demonstrated refractory sense marker VR (sensing during the relative refractory period) of the chest wall stimuli. This example demonstrates that undesired sense events, which often are invisible on the surface ECG, can cause an electrocardiographic pattern of undersensing [46,47].

In the presence of continuous interference of sufficient intensity, the refractory period will continuously be reset, ultimately resulting in asynchronous pacing; interference or noise mode. The lowest interference frequency at which the pulse generator switches to asynchronous pacing depends on the duration of the relative refractory period e.g. for a relative refractory period of

125 ms the lowest interference frequency is 8 Hz. The rate of the interference mode determines the minimum duration of the interference before reversion takes place, e.g. for an interference rate of 60 ppm, the duration of interference should be at least 1000 ms minus the absolute refractory period. This means that in those cases were the interference rate is equal to the programmed rate, the duration of interference needed to cause asynchronous pacing depends on the programmed rate and is not a fixed criterium. A fixed interference rate independent of the programmed rate is preferred, because electrocardiographic recognition is easier.
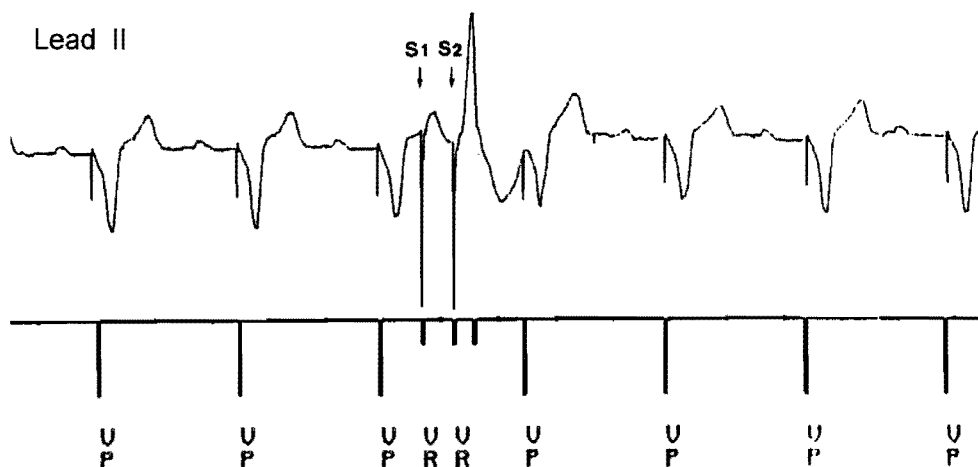
Fig. 5.28. One channel ECG (lead II) of a patient with an implanted VVI pacemaker demonstrating the effect of sensing during the relative refractory period. Two programmed chest wall stimuli (S1, S2) with a coupling interval of 300 and 220 ms respectively are applied to the patient. The total refractory period is 325 ms. The first stimulus is sensed during the relative refractory period, thus initiating a second refractory period, in which relative portion the second stimulus is sensed. A third refractory period is initated in which relative portion a spontaneous ventricular premature beat is sensed. The escape interval is not affected by the sensed events in the relative refractory periods. The marker channel illustrates sensing in the relative refractory period (VR); VP = ventricular pacing.

Reversion to the interference mode is illustrated in figure 5.29, in a patient with an implanted unipolar VVI pacemaker. During contraction of the pectoral muscle asynchronous pacing with an interval of 660 ms (corresponding to the interference rate) was recorded. The pacing rate was programmed at 50 ppm. After decreasing the sensitivity of the pulse generator to 4.2 mV interference reversion could not be induced by the same maneuver [48,49]. Interference mode can be activated by continuous interference from different sources as already illustrated in figure 5.5. A very uncommon cause of interference mode reversion is shown in figure 5.30. In this patient with an AAI pacemaker programmed at a rate of 50 ppm regular pacemaker stimuli with a 660 ms interval (indicating interference mode) were recorded. Reversion was caused by sensing atrial fibrillation, whose rate was high enough to activate the interference mode criteria. Also the low amplitude pulses, used to measure intrathoracic impedance in minute ventilation rate adaptive pacemakers are described as a source of interference rate reversion [50].

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False signals caused by fractured leads as shown in figure 5.17 sometimes have a bizarre morphology with respect to frequency as well as duration of the signals. Sometimes the signals have a sufficient frequency and duration to activate the interference mode. This can result in an electrocardiographic pattern with alternating inhibition (oversensing) and asynchronous pacing (Fig. 5.31). This ECG demonstrates the sense of an interference rate which is not equal to the programmed rate. If in this patient the interference rate had been the same as the programmed rate, asynchronous pacing due to interference rate would not be recognized and in the presence of intrinsic activity even interpreted as undersensing.
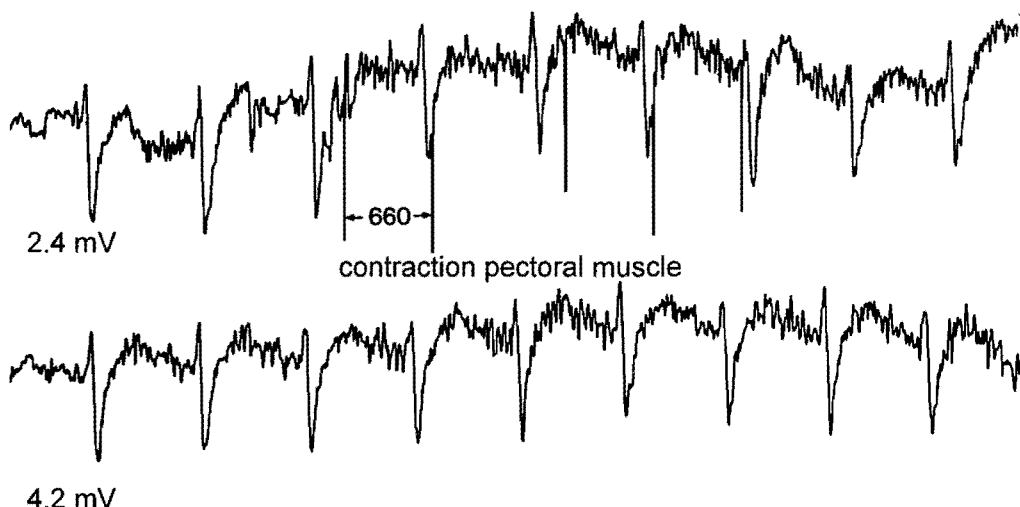
Fig. 5.29. Two single channel ECGs of a patient with an implanted unipolar VVI pacemaker programmed at a rate of 50 ppm. During contraction of the pectoral muscle (upper tracing) asynchronous pacing with an interval of 660 ms (corresponding with the interference rate) was recorded. After programming the sensitivity to 4.2 mV instead of 2.4 mV, the maneuver was repeated without any influence on the pacing system.

Fig. 5.30. Single channel ECG (lead II) of a patient with an implanted AAI pacemaker (rate 50 ppm). The presence of atrial fibrillation sensed by the pacemaker induced interference rate pacing (90 ppm = 660 ms).

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5.13 Inadequate intracardiac electrogram

If a signal cannot be sensed by a pulse generator at its maximum sensitivity, without oversensing of other cardiac related signals, it should be considered to be an inadequate signal. Although the amplitude of the intracardiac electrogram expressed in mV and the sensitivity of the pulse generator are used as a standard for proper sensing, using only these two criteria has limitations. It is not only the amplitude but also the spectral content of the signal, as well as the characteristics of the input amplifier, that determine sensing function [51]. Information about spectral content can be obtained by Fourier analysis. This requires extensive equipment, which is not available on a routine basis in implanting centres. An indication of the frequency spectrum of the intracardiac signal can be obtained from the slew rate or the voltage amplitude versus time (dv/dt). This parameter can be measured by the majority of pacing system analyzers, that are used during implantation or replacement of pulse generators.

Lead II   1180   840   660   840   1360

VVI 70 ppm
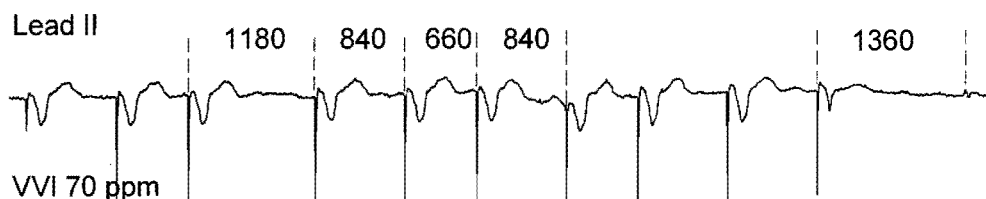
Fig 5.31. Single channel ECG (lead II) of a patient with an implanted VVI pacemaker. The pacing interval is longer or shorter than the programmed escape interval (840 ms = 72/min). There is oversensing in the pacing system due to an electrode fracture with intermittent contact between both ends. The signals generated by this intermittent contact give rise to inhibition (prolonged intervals) as well as interference mode reversion (shortened intervals of 660 ms).

Discrepancy between amplitude and slew rate is illustrated in figure 5.32. This recording shows a much lower slew rate of a ventricular ectopic beat as compared to a normally conducted sinus beat, this in spite of a much higher amplitude of the ventricular ectopic beat. The lower slew rate is explained by the fact that ventricular depolarization caused by a ventricular premature contraction orginates from a ventricular focus and the wave form is propagated through the ventricular myocardium, whereas ventricular activation is more or less simultaneous during normal activation. It explains, however, that undersensing of ventricular premature beats can be observed in spite of an adequate amplitude of the intracardiac electrogram.

Pacemaker sensing is not only determined by amplitude and slew rate of the intracardiac electrogram but also by the sensitivity and characteristics of the band-pass filter of the input amplifier [52]. Because the latter is not uniform in the different models of pulse generators, predictability of proper sensing can be difficult in the case of border line signals [53]. In practice, chronic values for slew rates should be > 0.75 V/s for ventricular and > 0.50 V/s for atrial leads to avoid undersensing.

5.13.1 Causes of an inadequate intracardiac electrogram

An indequate electrogram encountered during lead implantation should not be accepted. A different location, with proper pacing and sensing characteristics, for the electrode should be

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explored. This sometimes necessitates the use of leads with active fixation. An inadequate electrogram after implantation may be due to the following causes:

1. Displacement of the electrode.

When an electrode is displaced or perforated and is no longer in contact with the myocardium, there is a marked reduction in amplitude as well as in slew rate of the intrinsic signal. It will be clear that displacement is not only presented as undersensing but will be combined with an elevated stimulation threshold or no capture.
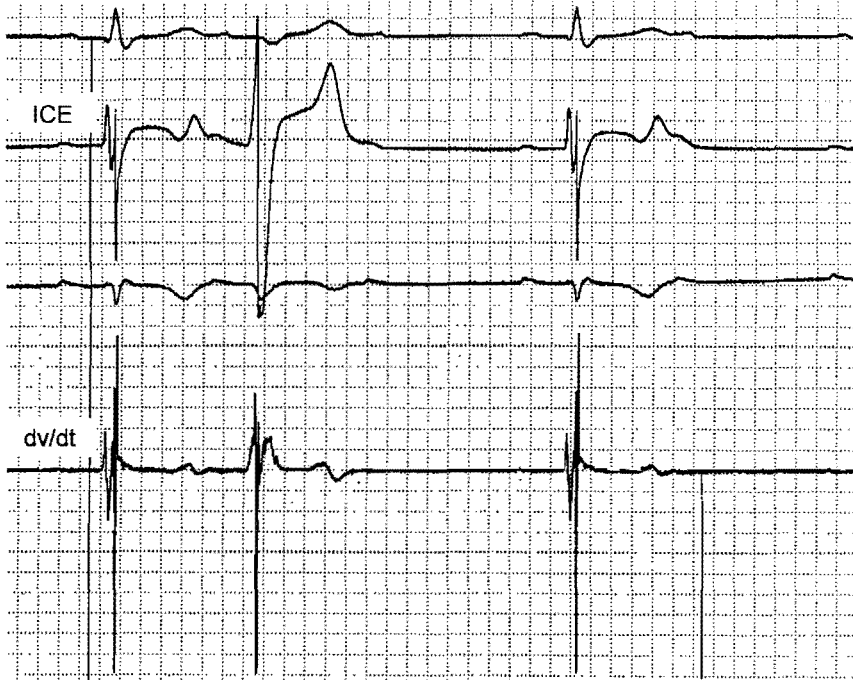
Fig. 5.32. Four channel recording showing the intracardiac ventricular electrogram (ICE) and the slew rate of this electrogram (dv/dt). It illustrates that the slew rate of the ventricular premature beat is much lower than that of the sinus beats, in spite of a higher amplitude of the ventricular premature beat.

2. Myocardial fibrosis formation around the electrode.

Due to the increased distance between electrode and myocardium, there is a reduction in amplitude and slew rate. Failure to sense and pace depends on the degree of fibrosis formation, which develops after implantation of a lead. (see also figure 4.62, chapter IV). Fibrosis formation can also be due to diseases like sarcoidosis and amyloidosis [54]. Although of the latter only an elevated stimulation threshold is reported [55], undersensing can be expected under these circumstances.

3. Myocardial infarction, or scarring.

If myocardial infarction occurs in the area of the electrode the distance between the viable myocardium and the pacing/sensing electrode is increased, resulting in a decrease in amplitude and slew rate [56].

4. Changes in intraventricular conduction.

As illustrated in figure 5.32, slew rate is directly related to the intraventricular conduction. Development of bundle branch block, which may cause a significant change in the ventricular electrogram, can be a cause of undersensing.

In those cases where an inadequate signal is considered to be the cause of undersensing, this should be confirmed by recording the intracardiac electrogram [57]. If this option is not available in the implanted pacing system, the diagnosis should be made by elimination of the other causes (refractoriness of the pacing system, dysfunction of the lead or pulse generator).

5.14 Attenuation of the intracardiac electrogram by the pacing system

The intrinsic electrogram can be of sufficient amplitude and slew rate but can be attenuated by dysfunction of the lead system or pulse generator. Under normal conditions there will be some attenuation of the intracardiac electrogram but this will generally be negligible.

The myocardial depolarization may be regarded as a voltage source (Vh) that is connected to the input amplifier of the pulse generator. The connection from this voltage source to the pulse generator input is formed by the lead and the electrode-tissue interface. The conductor of the lead is an ohmic resistance (Rlead), whereas the electrode tissue interface has an ohmic component, R electrode-tissue (Ret) and a capacitive component (Helmholz capacitance Ch) parallel with the Faraday resistance (RF) due to concentration polarization around the electrode [58]. These components together form the lead impedance (Zl), shown in a simplified diagram in figure 5.33. In this diagram Vh is the voltage source (amplitude of the intracardiac electrogram) and Zi is the input impedance of the pulse generator. Vs is the amplitude of the intracardiac electrogram sensed by the pulse generator.

From this diagram it will be easy to understand that the relationship between the sensed voltage (Vs) and the true amplitude of the electrogram (Vh) is:

$$Vs = \frac{Zi}{Zi + Zl} \quad Vh \quad \{I\}$$

For the sake of simplicity it is assumed that Zl and Zi are not frequency dependent. If Zi is much greater than Zl, Zi/Zi+Zl is approximately 1.0 and there will be only a minor difference between the sensed voltage (Vs) and the true electrogram (Vh), which is the ideal situation.

5.14.1 Attenuation of the sensed signal by increase of lead impedance

Formula I illustrates the effect of increased lead impedance on pacemaker sensing. If for instance Zl is equal to Zi (due to an increase in Zl), Zi/Zi+Zl = 0.5 and the sensed signal will be half the amplitude of the intracardiac electrogram. For ventricular pacemakers, where the amplitude of the electrogram is usually > 5 mV, the sensed signal will be > 2.5 mV, which is the nominal sensitivity setting for most pulse generators and sensing will be maintained. On the other hand, if the lead impedance is increased to the value of the input impedance of the pulse generator e.g. 20 KOhms, the resulting stimulation current is reduced to 0.25 mA, if the pacemaker output is 5.0 V. This implies noncapture in the majority of patients.

It can be concluded that an increase in lead impedance (fractured leads, poor connections) first affects stimulation before sensing, provided that the high lead impedance is stable and not

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varying, since variation in lead impedance may cause oversensing as described in paragraph 5.8.3.

5.14.2 Attentenuation of the sensed signal by decrease of lead impedance

If a decrease in lead impedance is caused by a decrease in the electrode-tissue resistance, there will be no adverse effect on pacemaker sensing. However, if a decrease in lead impedance is caused by an insulation defect in a lead and a low resistance path is formed between the conductors of a bipolar lead or between the lead conductor and the pacemaker case in a unipolar system, sensing can be affected.
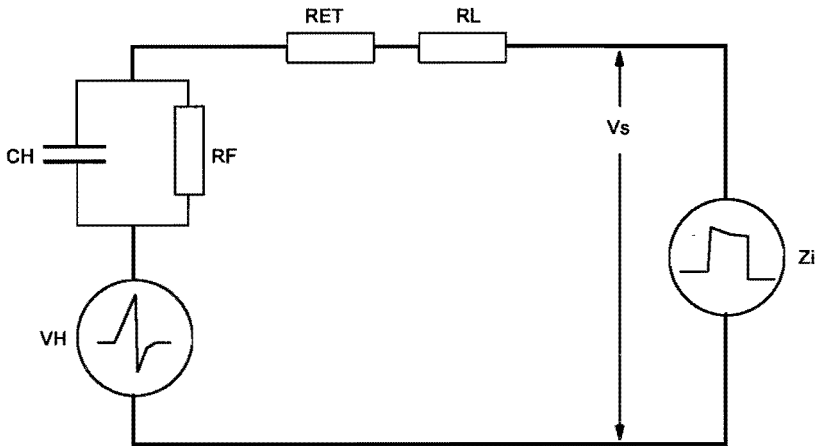
Fig. 5.33. Diagram of the simplified equivalent circuit model, in which the heart is the signal source (Vh), the Helmhotz capacitance (CH), the Faraday resistance (RF) and the electrode tissue resistance (Re-t) are the components of the electrode-tissue interface, Rl is the conductor resistance, Zi is the input impedance of the pulse generator, Vs is the signal sensed by the pulse generator.

This can best be explained by using a diagram, similar to the diagram of figure 5.33, but with an additional resistance simulating an insulation defect (Rid), whose resistance is parallel with the pulse generator input impedance (Fig. 5.34). In a simplified approach, the total impedance (Zt) formed by Rid and Zi, is equal to

$$Zt = \frac{Zi.Rid}{Zi + Rid} \quad \{II\}$$

If Zi is 20 KOhms and the resistance of the low resistance pathway formed by the insulation defect 1 KOhm, the total impedance will be 20/21 = 0.952 Kohm. Using this value in equation I for Zi and substitute 0.5 KOhm for Zl, the relation between the amplitude of the intracardiac electrogram and the sensed signal is 952/952+500 = 0.65, so the sensed signal is only 65% of the true amplitude [59].

The effect of an insulation defect between anodal (conductor) electrode and cathodal conductor can be simulated by placing an external resistor between anodal and cathodal conductors as is

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demonstrated in figure 5.35. The signal is measured with an electrocardiograph with a high input impedance, so the signal loaded with 20 KOhms (which is of the order of pulse generators input
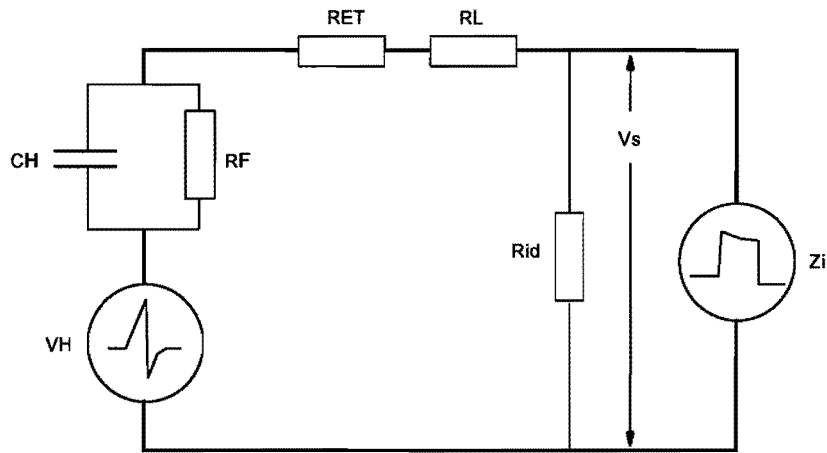
Fig. 5.34. Same diagram as figure 5.33 with an additional resistor (Rid) across the input impedance (Zi) of the pulse generator. This resistor simulates an insulation defect of the lead. The effect of the insulation defect on the sensed signal (Vs) is explained in the text.

impedance), is comparable with the signal measured by the pulse generator. Loading the signal source with 5 and 1 KOhms respectively shows attenuation and differentiation of the signal.
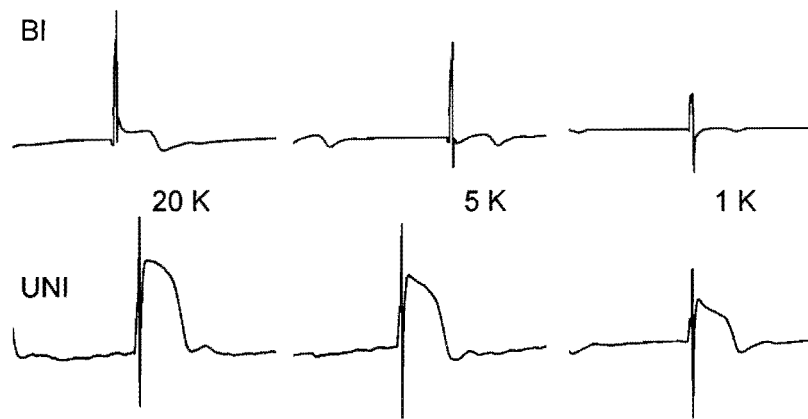
Fig. 5.35. Recording of a bipolar and unipolar electrogram from the same bipolar lead. The signal is loaded with 20, 5, 1 KOhm respectively, simulating an insulation defect. Note the attenuation and differentiation of the sensed signal. Differentiation is more pronounced in the bipolar configuration.

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The slowly changing portions are greatly attenuated and differentiated while the fast changing portions are mainly attenuated. Differentiation is the result of the R-C equivalents of the electrode being frequency dependent [60]. The differentiating as well as attenuating effect is more pronounced in bipolar systems than in unipolar systems because of the additional capacity of the ring electrode [61].

It also will be clear from the diagram (Fig. 5.34) that the effect of an insulation defect on stimulation threshold will be limited, as most pulse generators are constant voltage sources. Often pacing may continue even though sensing was lost. Of course pacemaker longevity will decrease by the increased current drain trough the insulation defect

Measurement of the intracardiac electrogram at pacemaker follow-up may be a useful tool in predicting insulation defects, because the diminishing amplitude and changing morphology of the intracardiac electrogram is observed before sensing problems become clinically manifest [62].

5.14.3 Undersensing due to pulse generator malfunction

An adequate intracardiac signal, in terms of amplitude and spectral characteristics, and a normally functioning lead can be ignored by a defective pulse generator resulting in undersensing. Undersensing by a malfunctioning pulse generator is rarely an intermittent phenomenon. It can be caused by a component failure in the electrical circuitry or a defective reed switch, which remains in the closed position [63]. In both cases this will result in asynchronous pacing. The best tool to diagnose undersensing caused by pulse generator malfunction is chest wall stimulation. If electrical stimuli applied, by an external stimulator, at its

Fig. 5.36. Two lead ECG (II upper tracing, III lower tracing) of a patient with an acute inferior wall infarction and an external pacemaker. There is undersensing of the pacing system, causing competition between ventricular stimuli and the intrinsic rhythm, resulting in ventricular fibrillation.

maximum output, to skin electrodes close to the anodal and cathodal electrode, are unable to inhibit a pacing system, pulse generator malfunction can be concluded. Before this test electrical integrity of the lead system should be confirmed.

5.15 Clinical impact of undersensing

Electrical pulses delivered to the ventricular myocardium during the vulnerable period can result in repetitive ventricular arrhythmias including ventricular fibrillation [64]. It is rarely encountered in a healthy myocardium but well known during enhanced myocardial vulnerabilty (Fig. 5.36).

Bipolar pacing has been reported as carrying a potentially higher risk for the initiation of ventricular tachycardia or fibrillation [65]. A more recent study, however, revealed that only at high pacemaker output (> 5V, 1.0 ms) does the anodal electrode play a role in ventricular depolarization [66]. Thus it is unlikely that at nominal or low pacemaker output there is a difference between unipolar and bipolar stimulation with respect to the induction of ventricular arrhythmias.

Similar to the mechanism of inducing ventricular arrhythmias by competitive pacing in the ventricle, atrial arrhythmias can be induced by competitive pacing in the atrium [67]. Because atrial arrhythmias are rarely lifethreatening in the absence of fast conducting accessory pathways, scant attention is paid to this phenomenon in pacemaker patients. However, when atrium flutter/fibrillation is induced by competitive atrial pacing in a patient with an atrial tracking dual chamber pacemaker (Fig. 5.37), it can result in an undesired high stimulation rate with discomfort for the patient [68].
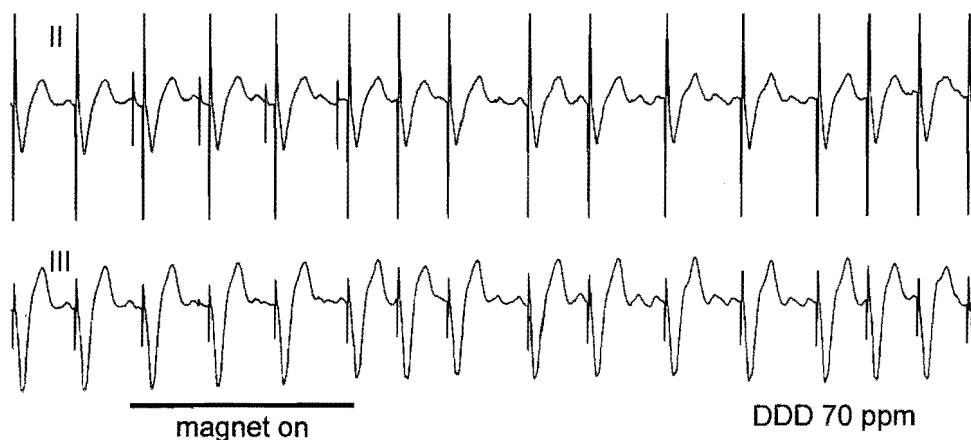
Fig. 5.37. Two channel ECG (lead II, III) of a patient with an implanted DDD pacemaker. During magnet application, resulting in DOO pacing and competition between P waves and atrial stimuli, atrial flutter is induced.

5.16 Summary and conclusions

The timing intervals between consecutive cardiac events either stimulated or intrinsic and stimulated, are essential in the evaluation of pacemaker sensing.

Before the diagnosis of oversensing can be made definitively from an ECG showing prolongation of the escape interval intermittent no output or irregular pulse formation should be excluded. The latter can be carried out by magnet application, which results in asynchronous pacing. If a regular escape interval after magnet application is observed, oversensing is present.

Oversensing from exogenous signals will be rarely encountered during regular pacemaker follow-up. If a pacemaker patient enters an environment where strong electromagnetic fields, (which probably may affect pacemaker function), are present, the ECG should be monitored and precautionary measures should be taken to restore normal pacemaker function if necessary. If no influence on pacemaker function is observed in an individual patient, no general conclusion can be drawn from this single observation since many factors determine the influences of electromagnetic fields on pacing systems.

Prolongation of the escape interval encountered during pacemaker follow-up can be divided into prolongation with a more or less fixed interval and prolongation with varying intervals. Generally prolongation with a more or less fixed interval is directly related to stimulation or the cardiac response to stimulation. E.g., T-wave sensing in VVI pacemakers. These forms of oversensing are benign and they are characterized by the fact that oversensing can be corrected by decreasing sensitivity of the pulse generator. Recording of a marker channel and intracardiac electrogram usually elucidate the origin of the signals responsible for oversensing. Lead impedance will be within normal limits.

Oversensing with varying prolongation of the escape interval is caused by signals randomly distributed throughout the cardiac cycle. The random distribution of the signals excludes the possibility that they are directly related to stimulation or the cardiac response to stimulation. Abrupt change in lead impedance is the most likely explanation for oversensing. These abrupt changes are caused either by conductor or insulation defects in the pacing lead or an inproper lead connection. Although in a number of cases lead impedance may be changed normal values for lead impedance can be found even in a clearly malfunctioning lead. The interpreter should be aware of the fact that lead impedance is measured during the stimulation pulse and that variation in lead impedance between two pacemaker stimuli is not reflected in this measurement. Confirmation of lead malfunction is best obtained from the intracardiac electrogram, it is characterized by the presence of spurious signals varying in amplitude and morphology and randomly distributed throughout the cardiac cycle. Very rarely these signals have a more or less fixed relation to stimulation, probably because the intermittent contact in the lead is provoked by ventricular or atrial contraction. The surface ECG may be misleading by the more or less fixed prolongation of the escape interval but the morphology of the signals on the intracardiac electrogram are very specific. Recording the electrogram for a longer period of time usually reveals the presence of these signals at different intervals.

Patients presenting with intermittent recurrence of pre-pacing symptoms, which are provoked by contraction of the muscle adjacent to the pulse generator, should be evaluated for myopotential inhibition of the pacing system. Pacemaker manipulation should be performed to exclude intermittent contacts within the pacing system as the cause of inhibition. Inhibition of the pacing system by myopotentials can be corrected by decreasing sensitivity. If this fails or sensitivity has to be programmed to a level incapable of sensing intrinsic cardiac activity, programming to the triggered mode (AAT, VVT) is a practical solution for the symptomatic patient.

If electrocardiographically undersensing is present, oversensing producing interference mode pacing or prolongation of the refractory period should first be excluded as the possible cause. In patients with predominantly intrinsic rhythm, oversensing as the possible cause can be easily overlooked and correction of the problem by increasing sensitivity results in aggravation of undersensing. Marker channel recording immediately illustrates oversensing as the possible cause of undersensing. In patients with predominantly pacing rhythm, undersensing may be

present on the ECG. However, this will often be associated with prolongation of the escape interval, indicating oversensing as the explanation for undersensing.

True undersensing is caused by either an inadequate electrogram or attenuation of the electrogram by the pacing system, and rarely by malfunction of the pulse generator. Undersensing associated with an increase or decrease in lead impedance is indicative for attenuation of the electrogram by the pacing system. Lead malfunction either by conductor or insulation failure is the cause of undersensing. A new lead system should be implanted before noncapture occurs.

If an inadequate electrogram is the cause of undersensing, lead impedance will be within normal limits. Displacement can be diffentientiated from fibrosis formation by the morphology of the stimulated QRS complex, which is unchanged in the latter case, compared to previous recordings. It will be obvious that under both conditions a rise in stimulation threshold will be observed and in case of displacement, noncapture may be present. Undersensing due to changes in intraventricular conduction will be recognized from the surface ECG if a normally conducted QRS complex is properly sensed and a wide QRS complex is ignored by the pacing system.

Pulse generator failure as the cause of undersensing can be excluded by chest wall stimulation. If chest wall stimulation fails to inhibit the implanted pacing system, pulse generator failure is demonstrated.

References

1. Irnich W. Interference in pacemakers. PACE 1984; 7: 1021-1047.

2. Levine PA, Balady GJ, Lazar HL, et al. Electrocautery and pacemakers: Management of the paced subject to electrocautery. Ann Thoracic Surg 1986; 41: 313-317.

3. Toivonen L, Valjus J, Hongisto M, et al. The influence of elevated 50 Hz electric and magnetic fields on implanted cardiac pacemakers: The role of the lead configuration and programming of the sensitivity. PACE 1991; 14: 2114-2122.

4. Marco D, Eisinger G, Hayes DL. Testing of work environments for electromagnetic interference. PACE 1992; 15: 2016-2022.

5. Pannizzo F, Furman S. Pacemaker and patient response to the "Point of sale" terminal as an actual and simulated electromagnetic interference source. PACE 1980; 3: 461-469.

6. Copperman Y, Zarfati D, Laniado S. The effect of metal detector gates on implanted permanent pacemakers. PACE 1988; 11: 1386-1387.

7. Gleason CA, Kaula NF, Hricak H, et al. The effect of magnetic resonance imagers on implanted neurostimulators. PACE 1992; 15: 81-94.

8. Pavlicek W, Geisinger M, Castle L, et al. The effects of nuclear magnetic resonance on patients with cardiac pacemakers. Radiology 1983; 147: 149-153.

9. Fetter J, Aram G, Holmes DR, et al. The effects of nuclear magnetic resonsce imagers on external and implantable pulse generators. PACE 1984; 7: 720-727.

10. Holmes DR, Hayes DL, Gray JE, et al. The effects of magnetic resonace imgaing on implantable pulse generators. PACE 1986; 9: 360-370.

11. Iberer F, Justich E, Tscheliessnigg K-H, et al. Nuclear magnetic resonance imgaing in pacemaker patients. In JL Atlee, H Gombotz, K- H Tscheliessnigg (Eds): Perioperative management of pacemaker patients. New York, Springer Verlag, 1992:86-90.

12. Shellock FG, Kanal E, SMRI Safety Committee. Policies, guidelines, and recommendations for MR imaging safety and patient management. JMRI 1991; 1: 97-101.

13. Lauck G, van Smekal A, Jung W, et al. Influence of nuclear magnetic resonance imgaing on software-controlled cardiac pacemakers.(abstract) PACE 1993; 16: 1140.

14. Johnson D. *Magnetic resonance imaging effects and considerations with permanent cardiac pacemakers.(abstract) PACE 1994; 17: 772.*

15. Von Smekal A, Wolke S, Seelos KC, et al. *The influenec of static magnetic fields of 1.5 Tesla on implantable cardiac pacemakers and components.(abstract) JACC 1994; 324A.*

16. Lauck G, Sommer T, Wolke S, et al. *Magnetic resonance imaging in patienst with implanted cardiac pacemakers.(abstract) PACE 1995; 18: 1168.*

17. Bermann ND. *T wave sensing with a programmable pacemaker. PACE 1980; 3: 656-659.*

18. De Boer H, Kofflard M, Scholtes T, et al. *Differential bipolar sensing of a dual chamber pacemaker. PACE 1988; 11: 158-165.*

19. Brandt J, Fahraeus T, Schüller H. *Far-field QRS complex sensing via the atrial pacemaker lead. 1. Mechanism, consequences, differential diagnosis and countermeasures in AAI and VDD/DDD pacing. PACE 1988; 11: 1432-1438.*

20. Mond HG, Sloman JG. *The malfunctioning pacemaker system. Part II. PACE 1981; 4: 168-181.*

21. Strasberg, Erdman S, Agman J. *P wave oversensing and pseudo QRS undersensing by a programmable ventricular demand pacemaker. Clin Prog 1985; 1: 25-28.*

22. Mond HG. *The cardiac pacemaker: Function and malfunction. New York : Grune & Stratton, 1983: 349-378.*

23. Van Gelder LM, El Gamal MIH, Tielen CHJ. *P wave sensing in VVI pacemakers: Useful or a problem ? PACE 1988; 11: 1413-1418.*

24. Van Gelder BM, Bracke FALE, El Gamal MIH. *P wave oversensing in a unipolar VVI pacemaker. PACE 1995; 18: 370-373.*

25. Wirtzfeld A, Lampadius M, Ruprecht EO. *Unterdruckung von Demand-Schrittmachern durch Muskelpotentiale. Dtsch Med Wochenschr 1972; 97: 61-66.*

26. Breivik K, Ohm OJ. *Myopotential inhibition of unipolar QRS-inhibited (VVI) pacemakers, assessed by ambulatory Holter monitoring of the electrocardiogram. PACE 1980; 3: 470-478.*

27. Berger R, Jacobs W. *Myopotential inhibition of demand pacemakers: Etiology, diagnostic, and therapeutic considerations. PACE 1979; 2: 596-602.*

28. Watson WS. *Myopotential sensing in cardiac pacemakers. In SS Barold (ed.): Modern Cardiac Pacing. Mount Kisco NY: Futura Publishing Co, 1985, pp. 813-837.*

29. Widlansky S, Zipes DP. *Suppression of a ventricular-inhibited bipolar pacemaker by skeletal muscle activity. J Electrocardiol 1974; 7: 371-374.*

30. Amikam S, Peleg H, Lemer J, et al. *Myopotential inhibition of a bipolar pacemaker caused by electrode insulation defect. Br Heart J 1977; 39: 1279-1281.*

31. Barold SS, Ong LS, Falkoff MD, et al. *Inhibition of bipolar demand pacemaker by diaphragmatic myopotentials. Circulation 1977; 56: 679-683.*

32. El Gamal M, van Gelder B. *Suppression of an external demand pacemaker by diaphragmatic myopotentials: A sign of electrode perforation ? PACE 1979; 2: 191-195.*

33. Van Gelder LM, El Gamal MIH, Bracke FALE. *Are bipolar systems prone to oversensing at high sensitivity setting ? (abstract) PACE 1991; 14: 1991.*

34. Barold SS, Ong LS, Falkoff M, et al. *Complex demand pacemaker arrhythmias: the differential diagnosis of pacemaker pauses. In HJTh Thalen en JW Harthorne (Eds.): To pace or not to pace. The Hague, Martinus Nijhoff Medical Division, 1978, p. 312.*

35. Sinnaeve A, Willems R, Backers J, et al. *Pacing and sensing: How can one electrode fulfill both requirements. PACE 1987; 10; 546-554.*

36. Lindemans FW. *Personal communication. November 1994.*

37. Lassetter KC, Buchanan JW, Yoshonis KF. *A mechanism for "false" inhibition of demand pacemakers. Circulation 1970; 42: 1093-1097.*

38. Barold SS, Falkoff MD, Ong LS, et al. *In HJJ Wellens and HE Kulbertus (Eds): What's new in electrocardiography. The Hague, Martinus Nijhoff Publishers, 181, p. 278.*

39. Sutton R, Bourgeois I. The foundations of cardiac pacing, Part I. Mount Kisco, NY, Futura Publishing Company, Inc. 1991, p. 281.

40. Waxman HL, Lazarra R, El-Sherif N. Apparent malfunction of demand pacemakers due to spurious potentials generated by contact between two endocardial electrodes. PACE 1978; 1: 531-534.

41. Furman S, De Caprio V. Electrode causation of pacemaker inhibition. Chest 1972; 72: 117.

42. Furman F, Pannizzo F, Campo I. Comparison of active and passive adhering leads for endocardial pacing. PACE 1979; 2: 417-427.

43. Van Gelder LM, El Gamal MIH. False inhibition of an atrial demand pacemaker caused by an insulation defect in a polyurethane lead. PACE 1983; 6: 834-839.

44. Raymond RD, Nanian KB. Insulation failure with bipolar polyurethane pacing leads. PACE 1984; 7: 378-380.

45. Van Gelder LM, El Gamal MIH. Prolongation of pacing intervals in triggered pacing systems (AAT, VVT). (abstract) PACE 1987; 10: 757

46. Warnowicz MA, Goldschlager N. Apparent failure to sense (Undersensing) caused by oversensing: Diagnostic use of a noninvasively obtained intracardiac electrogram. PACE 1983; 6: 1341-1343.

47. Falkoff M, Ong LS, Heinle RA, et al. The noise sampling period: A new cause of apparent sensing malfunction of demand pacemakers. PACE 1978; 1: 250-253.

48. Van Gelder LM, El Gamal MIH. Undersensing in VVI pacemakers detected by Holter monitoring. PACE 1988; 11: 1507-1511.

49. Erkkila KI, Singh JB. Reversion mode activation by myopotential sensing in a ventricular inhibited demand pacemaker. PACE 1985; 8: 50-51.

50. Wilson JH, Lattner S. Apparent undersensing due to oversensing of low amplitude pulses in a thoracic impedance sensing, rate responsive pacemaker. PACE 1988; 11: 1479-1481.

51. Barold SS, Ong LS, Heinle RA. Demand pacemakers: Normal and abnormal mechanisms of sensing. In P Samet and N El-Sherif (Eds): Cardiac Pacing. New York, Grune & Stratton, 1980, p.559.

52. Ohm OJ. Inhibition/filter characteristics and input impedances of QRS inhibited demand pacemakers determined by in vitro studies. PACE 1980; 3: 318-331.

53. Arai Y, Yamazoe M, Toeda T, et al. Optimal pacemaker sensing with respect to amplitude and slew rate of intracardiac electrograms: Theoretical analysis by computer simulation. PACE 1984; 7: 778-783.

54. OJ Ohm. Demand failures occurring during permanent pacing in patients with serious heart disease. PACE 1980; 3: 44-55.

55. Jansson JH, Eriksson P, Boman K, et al. Cardiac stimulation threshold in familial amyloidosis with polyneuropathy. PACE 1987; 10: 817-821.

56. Chatterjee K, Rouse W. Ventricular endocardial potentials after experimental coronary artery occlusion in dogs. Amer. Heart J. 1971; 82: 352-357.

57. Sarmiento. Clinical utility of telemetered intracardiac electrograms in diagnosing a design dependent lead malfunction. PACE 1990; 13: 188-195.

58. Bornzin G, Stokes K. The electrode-biointerface: sensing. In SS Barold (Ed): Modern Cardiac Pacing. Mount Kisco, New York, Futura Publishing Company, 1985, p.79.

59. Ohm OJ. The interdependence between electrogram, total electrode impedance necessary to obtain adequate functioning of demand pacemakers. PACE 1979; 2: 465-485.

60. Raber MB, Cuddy TE, Israel DA. Pacemaker electrodes act as high-pass filters on the electrogram. In Y Watanabe (Ed): Cardiac pacing. Proceedings of the Vth international symposium, Tokyo, March 14-18, 1976. Amsterdam-Oxford, Excerpta Medica, 1977, p.507.

61. Sutton R, Bourgeois I. The Foundations of Cardiac Pacing. Part I. Mount Kisco, New York, Futura Publishing Company 1991, p. 59.

62. Van Beek GJ, den Dulk K, Lindemans FW, et al. Detection of insulation failure by gradual reduction in noninvasively measured electrogram amplitudes. PACE 1986; 9: 772-775.

63. Driller J, Barold SS, Parsonnet V. Normal and abnormal function of the pacemaker magnetic reed switch. J. Electrocardiol 1976; 9: 283-288.

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64. Kastor JA, Leinbach RC. *Pacemakers and their arrhythmias. In P. Samet (Ed). Cardiac Pacing, New York, Grune & Stratton, 1973, p.241.*

65. Preston TA. *Anodal stimulation as a cause of pacemaker induced ventricular fibrillation. Am Heart J. 1973; 28: 366-370.*

66. Van Gelder LM, El Gamal MIH, Tielen CHJ. *Changes in morphology of the paced QRS complex related to pacemaker output. PACE 1989; 12: 1640-1649.*

67. Furman S, Cooper JA. *Atrial fibrillation during AV sequential pacing. PACE 1982; 5: 133-135.*

68. Van Gelder LM, El Gamal MIH. *Magnet application, a cause of persistent arrhythmias in physiological pacemakers. Report of 2 cases. PACE 1982; 5: 710-714.*

Chapter VI. **Timing in Dual Chamber Systems**

Introduction
Failure to capture and sense in the atrium and/or the ventricle can occur with dual chamber systems as with single chamber systems. Interpretation of capture has already been discussed in chapter IV, while problems and causes of sense failures in single chamber systems have been described in chapter V. In dual chamber systems atrial and ventricular capture and sensing should be separately assessed, similar to a single chamber system, in order to be certain that normal function of both channels exist. However, it is possible that stimulation or sense failures in one channel will affect the function of the other channel. Because of this interaction between both channels timing intervals can be complex and determination of normal pacing and sensing from the surface ECG can be difficult. Sometimes the explanation of the ECG can be difficult or even impossible without the recording of a marker channel, indicating sense and pace events in both chambers. It is not only pace and sense events that affect pacemaker function but also combinations of parameter settings determine pacemaker behavior. The upper rate behavior, for example, is determined by the values of upper rate, atrial refractory period and AV interval. Mechanisms to prevent one channel from being affected by events in another lead to complex ECGs, that can be interpreted as malfunction. Understanding time intervals and the relation between the different parameters of both channels is therefore essential in the interpretation of dual chamber ECGs.

6.1 The timing diagram and definitions
Figure 6.1 illustrates the timing intervals in dual chamber pacemakers (DDD).

Timing DDD.
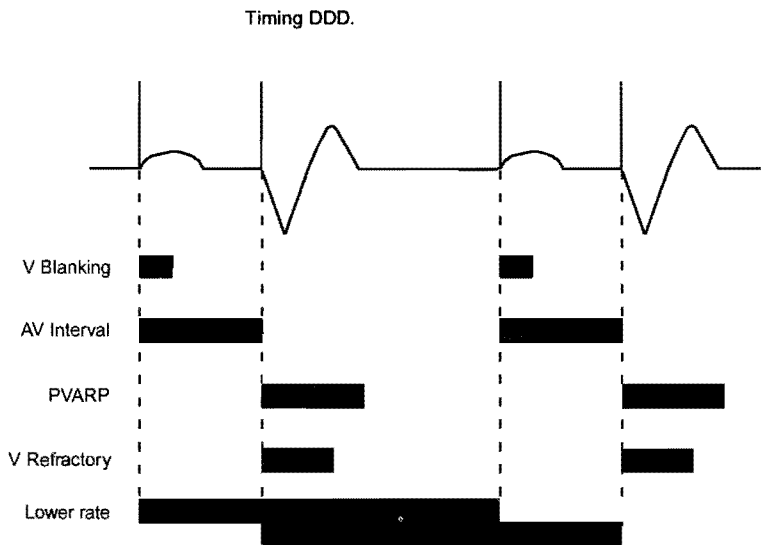
Fig. 6.1. Schematic presentation of the timing interval in dual chamber pacemakers. V blanking = ventricular blanking period; PVARP = Post ventricular atrial refractory period; V refractory = ventricular refractory period. The total atrial refractory period is not illustrated, but is the summation of AV interval and PVARP.

Lower rate
The preset or programmed rate at which the pacemaker will emit an atrial and ventricular output pulse in the absence of intrinsic cardiac activity.

AV interval
The interval between the initiation of the paced or sensed atrial event and the delivery of a consecutive ventricular output pulse. Depending upon the timing system of the pulse generator (atrial or ventricular based, see paragraph 6.2) the AV interval timer is affected by an intrinsic ventricular event.
In order to maintain a constant interval between atrial and ventricular contraction, not affected by atrial pacing or sensing, some pulse generators have a shorter AV interval (40-50 ms) after atrial sensing compared to atrial pacing. This shorter interval compensates for the delay between the onset of the P wave and the moment that the P wave is sensed.
Some pulse genererators are equipped with an adaptive AV delay. This feature adapts the AV interval to changes in the preceding P-P interval in a fixed ratio. This means that at increasing atrial rates the AV interval becomes shorter, and longer at decreasing atrial rates, thus mimicking physiological AV conduction.

Total atrial refractory period (TARP)
The time after an atrial pace or sense event during which the lower rate and upper rate timer cannot be reset by any atrial sense event. The atrial refractory period is the summation of the AV interval and the post ventricular atrial refractory period (PVARP).

Post ventricular atrial refractory period (PVARP)
The time after a ventricular paced or sensed event during which the lower rate and upper rate timer cannot be reset by any atrial sense event. PVARP consists of two periods, an absolute refractory period and a relative refractory period. Definitions for absolute and relative refractory period are identical to the definitions used in chapter V 5.1. PVARP is the major programmable parameter used for prevention of pacemaker circus movement tachycardia. See also chapter VII 7.3.1.

Ventricular refractory period
The time after a ventricular paced or sensed event during which the lower rate and upper rate timer cannot be reset by any ventricular sense event. The ventricular refractory period consists of two parts, the absolute refractory period and the relative refractory period. Definitions for absolute and relative refractory period are identical to the definitions used in chapter V 5.1.
In dual chamber pacemakers the ventricular refractory period should be equal to, or shorther than PVARP. If PVARP is shorter than the ventricular refractory period, an atrial sense event could evoke ventricular pacing, even in the presence of intrinsic ventricular activity.

Upper rate interval
The shortest interval between two ventricular paced events or a sensed ventricular event followed by a paced ventricular event.

Maximum atrial tracking rate

The fastest atrial rate that can be sensed by the atrial channel and which can result in ventricular stimulation, provided that the latter is allowed by the programmed upper rate interval. The maximum atrial tracking rate is limited by the total atrial refractory period. For example, if the TARP = 400 ms, the maximum atrial tracking rate is limited to 150 bpm (60.000 divided by 400 ms). If the atrial rate exceeds the maximum tracking rate, a 2:1 or even higher degree of AV block will occur, depending upon the atrial rate.

Blanking period

A time period during which one of the pacemaker sensing amplifiers is temporarily disabled following delivery of an output pulse by the other channel. The blanking period prevents inappropriate sensing of afterpotentials from the pacemaker output pulse and, in dual chamber pacemakers, prevents sensing of pacemaker output pulses or intrinsic events in the chamber other than that in which the event occurred. In dual chamber pacemakers, sensing of the atrial output pulse in the ventricular channel is prevented by ventricular blanking. The term blanking period is also used for the absolute refractory period.

6.2 Atrial and ventricular based timing

At lower rate pacing, in the absence of intrinsic atrial and ventricular activity, the timing intervals in dual chamber pacemakers are well defined. The interval between two consecutive atrial stimuli and consecutive ventricular stimuli is equal to the programmed lower rate interval. The interval between the atrial and ventricular stimulus is equal to the programmed AV interval. However if during lower rate pacing normal AV conduction takes place within the programmed AV interval, pacemaker behavior can be different. This difference depends on the timing system of the pulse generator, which can be atrial or ventricular based timing.

6.2.1 Atrial based timing system

DDD lower rate 70 ppm; AV 250 ms

Fig. 6.2. Two lead ECG (lead I,II) of a patient with an atrial based timing dual chamber pacemaker. Lower rate 70 ppm, AV interval 250 ms, upper rate 125 ppm. There is normal AV conduction within the programmed AV interval, which inhibits ventricular pacing. The A-A interval (interval between two consecutive atrial stimuli) is 857 ms (measured with an electronical counter), which is equal to the programmed lower rate. The A-A interval is fixed and not affected by ventricular sensing.

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In an atrial based timing system, the A-A interval (interval between 2 consecutive atrial stimuli) is fixed. In a system with atrial based timing, a sensed R wave that occurs during the AV interval will inhibit the ventricular output but will not alter the basic AA timing.

Atrial Based Timing

Lower rate 1000 ms
AV interval 250 ms
AR interval 150 ms

Effective Ventricular Rate
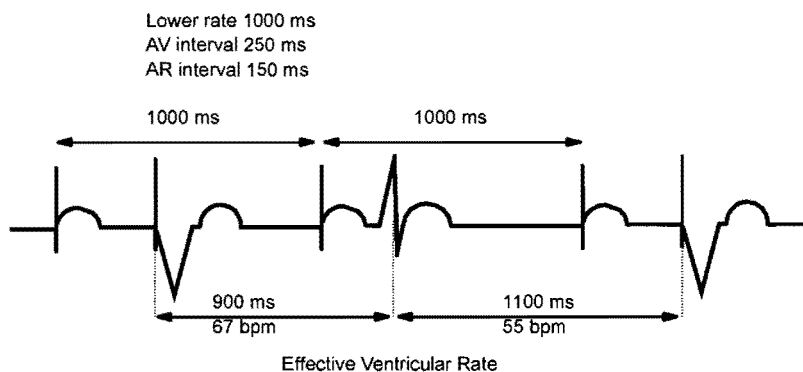
Fig. 6.3. Schematic presentation of an atrial based timing DDD pacemaker. Due to the fact that ventricular sensing has no effect on the A-A interval and the variation in AV conduction, variation in the effective ventricular rate is observed with a constant A-A interval. The effective ventricular rate could be higher and lower than the atrial rate, as illustrated.

Hence, the interval between two consecutive atrial stimuli will be equal to the programmed lower rate interval (Fig. 6.2). If lower rate pacing is performed with an atrial based timing system and alternating AV conduction is present, variations in the R-R intervals can be observed. These variations can be higher and lower than the programmed lower rate interval (Fig. 6.3).

6.2.2 Ventricular based timing system

In a ventricular based timing system, timing starts with a ventricular paced or sensed event after which the VA interval timer is started. The VA interval is determined by the difference between the programmed lower rate interval and the programmed AV interval. A sensed ventricular event ocurring during the AV interval terminates the AV interval and initiates the VA interval. If there is intact AV conduction following atrial stimulation in such a way that the interval between the atrial stimulus and the sensed R wave is shorter than the programmed AV interval, the resulting paced rate will accelerate by a small amount. For example, assume a pacemaker is programmed to a lower rate of 60 ppm (= 1000 ms). With a programmed AV interval of 250 ms, the VA interval is 750 ms (VA interval = lower rate interval - AV interval). If AV conduction time is 150 ms, the conducted or sensed R-wave inhibits the ventricular output and initiates the VA interval, which will remain stable at 750 ms. The resulting interval between consecutive atrial pacing stimuli will be 900 ms, or a rate of 67 ppm, which is slightly higher than the programmed lower rate (Fig. 6.4). So in ventricular based systems the interval between two consecutive atrial stimuli, in the presence of normal AV conduction within the programmed AV interval, will be shorter than the programmed lower rate interval, while under the same conditions in atrial based

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timing systems the interval between two consecutive atrial stimuli will be equal to the programmed lower rate.

DDD lower rate 60 ppm; AV 250 ms
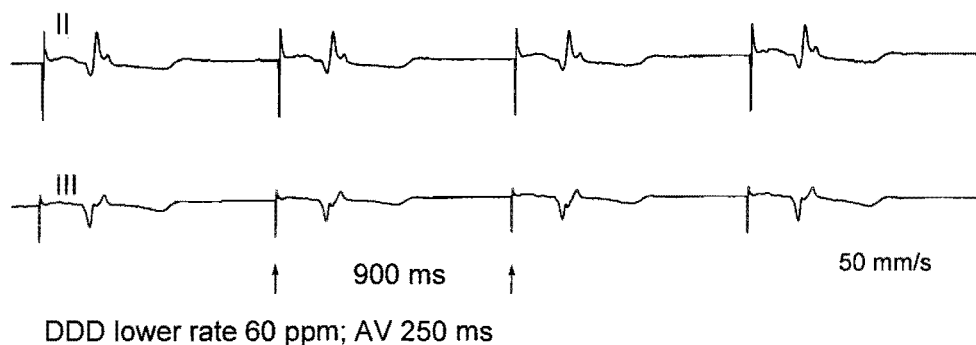
Fig. 6.4. Two lead ECG of a patient with a ventricular based timing dual chamber pacemaker. Lower rate 60 ppm, AV interval 250 ms, upper rate 120 ppm. There is normal conduction within the programmed AV interval, which inhibits ventricular pacing. Because the sensed ventricular event resets the VA counter (VA interval = 1000-250 ms = 750 ms) and the AV conduction time is 150 ms, the interval between two consecutive atrial stimuli equals 900 ms, corresponding with a pacing rate of 67 ppm. In a ventricular based timing system the interval between two consecutive atrial stimuli, in the presence of normal AV conduction within the programmed AV interval, is shorter than the programmed lower rate interval.

In atrial based timing systems the R-R interval can vary, depending upon alternation in AV conduction, whereas the intervals between atrial stimuli remain stable. The interpreter should be aware of the type of timing in the pacing system in order to prevent misinterpretation of the ECG.

6.3 Upper rate behavior in DDD pacemakers

The upper rate behavior in DDD pacemakers is determined by the upper rate interval and the maximum atrial tracking rate. Because the use of the latter definition is sometimes confusing in the literature, in the context of this chapter maximum atrial tracking rate is defined as the maximum atrial rate that can be sensed by the atrial channel. In terms of intervals, it is the shortest interval between consecutive P waves that allows sensing of the second P wave, regardless of the duration of the AV interval initiated by this second P wave [1].

The effective atrial refractory period corresponds to the minimum effective atrial sensing interval and is therefore identical to the total atrial refractory period (TARP). Because the upper rate behavior depends on both upper rate interval (URI) and total atrial refractory period the following theoretical conditions can be encountered:

1. URI < TARP
2. URI = TARP
3. URI > TARP

6.3.1 URI < TARP

If the URI is shorter than TARP the maximum rate at which the ventricles can be paced is higher than the maximum atrial rate that can be sensed. For example, with an URI of 400 ms (allowing maximum ventricular pacing at a rate of 150 ppm) and a TARP of 500 ms (allowing atrial sensing up to a rate of 120 bpm), ventricular stimuli can only be emitted at a maximum rate of 120 ppm. If the ventricular stimulation rate is limited by the maximum atrial tracking rate, it is inappropriate to program a ventricular upper rate higher than the maximum atrial tracking rate.
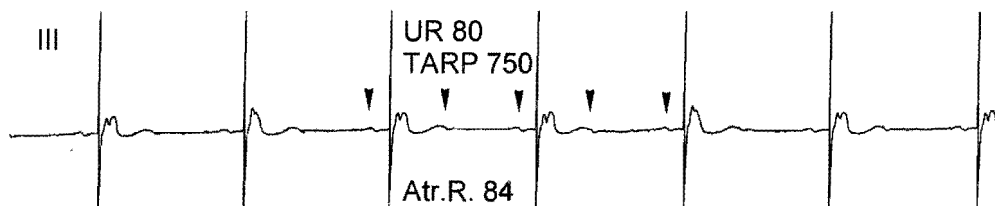
Fig. 6.5. Single channel ECG (lead III) of a patient with an implanted DDD pacemaker, URI = TARP = 750 ms. The atrial rate is 84 bpm. The rhythm shows 2:1 AV block with a rate of 42 ppm, because every other P wave occurs in TARP. P waves are indicated by arrows. See also figure 6.7.

6.3.2 URI = TARP

If the URI is equal to TARP, the maximum rate at which the ventricles can be paced and the maximum rate at which the atrium can be sensed are the same. If the atrial rate exceeds the maximum atrial tracking rate or in other words, the coupling interval between two consecutive P waves is shorther than TARP, every second P wave falls within the atrial refractory period leading to 2:1 AV block (Fig. 6.5). TARP defines the 2:1 AV block point. Should this occur during exercise, the rapid halving of the ventricular pacing rate may often lead to significant
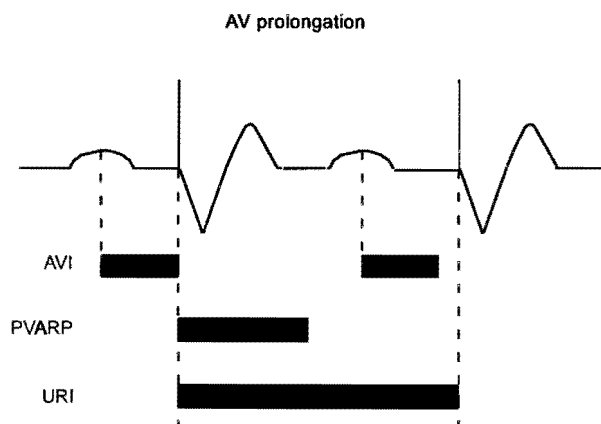
Fig. 6.6. Schematic presentation of AV interval prolongation if the atrial rate exceeds the ventricular upper rate in a DDD pacemaker where URI > TARP.

hemodynamic deterioration and symptoms [2]. In this case no prolongation of the AV interval occurs.The AV interval (interval between atrial sensing and ventricular pacing) will always be equal to the programmed AV interval. Given the length of the TARP in ms, the atrial rate at

which 2:1 AV block occurs is equal to 60.000/TARP. For example, if TARP is programmed to 500 ms, 2:1 AV block occurs when the atrial rate exceeds 120 bpm.

6.3.3 URI > TARP

If the URI is longer than the TARP, the maximum rate at which the ventricles can be paced is lower than the maximum atrial tracking rate, or in terms of intervals, the coupling interval between two consecutive ventricular paced events is longer than the coupling interval between two consecutive atrial sensed events. Initially when the atrial rate exceeds the upper ventricular pacing limit, a sensed atrial event will cause a ventricular paced event.
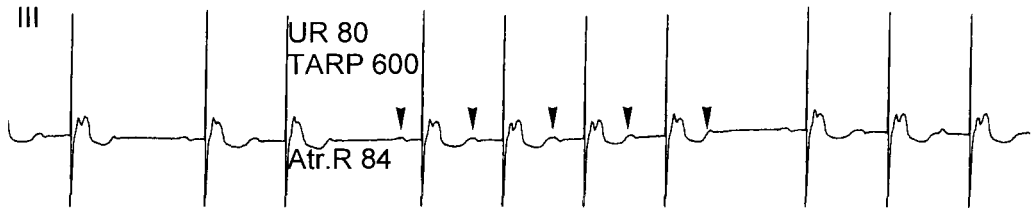
Fig. 6.7. Single channel ECG (lead III) of the same patient as figure 6.5, URI > TARP. The atrial rate is still 84 bpm. The rhythm now shows Wenckebach type AV block. The last arrow (indicating P wave) occurs in PVARP.

Under these circumstances, the timing of the ventricular paced event is not determined by the timing of the atrial sensed event and the programmed AV interval. After timing out of the AV interval ventricular pacing is postponed until the upper rate interval is elapsed. This will result in prolongation of the AV interval, the interval between atrial sensing and ventricular pacing (Fig. 6.6).
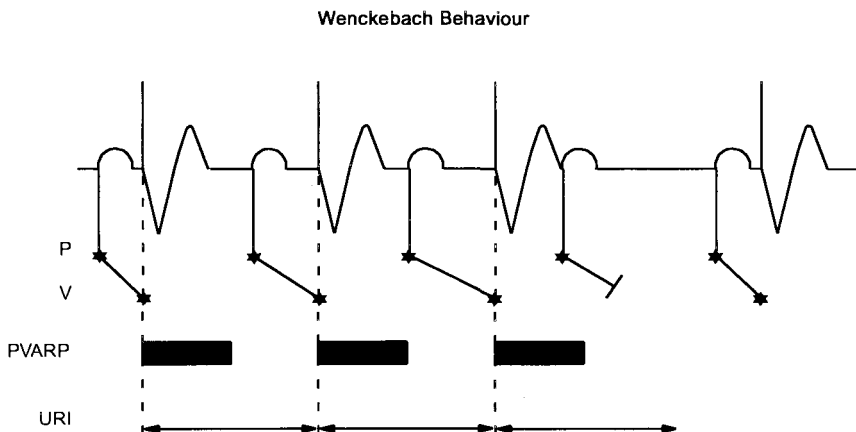
Fig. 6.8. Schematic presentation of Wenckebach type behavior in a DDD pacemaker where URI > TARP. There is gradual prolongation of the AV interval finally resulting in a P wave inside PVARP, which is not followed by ventricular stimulation.

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For the following P wave, assuming that the atrial rate is still higher than the upper rate ventricular pacing limit, the AV interval will be further prolonged. Figures 6.9 and 6.10 are a graphic illustration of the upper rate behavior for respectively URI = TARP and URI > TARP.

Ventricular Rate

PVARP 350
AVI 150
UR 120
LR 60

140
120
100
80
60

60 80 100 120 140
Atrial Rate
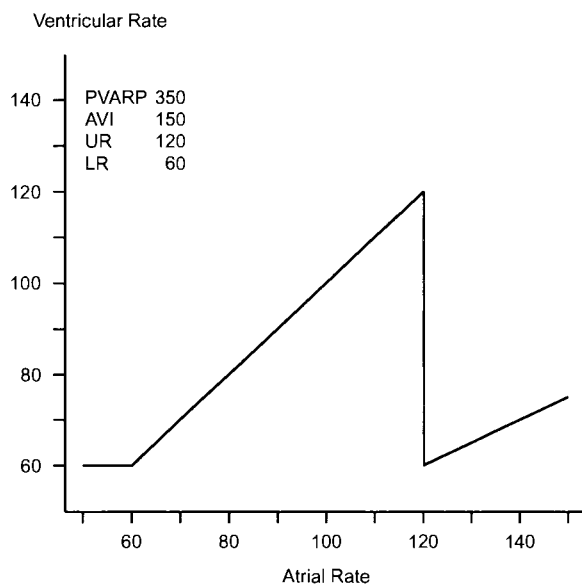
Fig. 6.9. Relationship between the atrial rate and the ventricular pacing rate in a DDD pacemaker in which URI = TARP. When the atrial rate exceeds the upper rate, there is rapid halving of the ventricular pacing rate. Values used for URI and TARP correspond with the ECG represented in figure 6.5.

Ventricular Rate

TARP 600
UR 80
LR 40

120
100
80
60
40

40 60 80 100 120
Atrial Rate
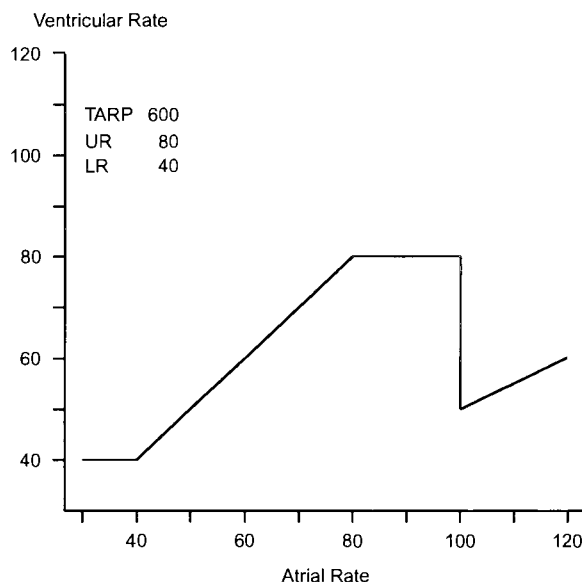
Fig. 6.10. Relationship between the atrial rate and the ventricular pacing rate in a DDD pacemaker in which URI > TARP. When the atrial rate exceeds the upper rate the ventricular pacing rate remains at the programmed upper rate and Wenckebach type behavior will be observed. As soon as the atrial rate exceeds the maximum atrial tracking rate (defined by TARP), 2:1 AV block will develop. Values used for URI and TARP correspond with the ECG represented in figure 6.7.

6.4 Factors affecting TARP

As explained in the previous paragraphs, the upper rate behavior is determined by the maximum atrial tracking rate and the upper rate ventricular pacing limit. The latter is a fixed parameter

chosen by the clinician depending upon the cardiac performance of the patient. The maximum atrial tracking rate is determined by the TARP. TARP, however, is rarely a programmable feature in dual chamber pacemakers but is indirectly programmed by the setting of the AV interval and PVARP. Because the value of the AV interval is not always fixed, as for instance in pulse generators with an adaptive AV interval, TARP and thus the maximum atrial tracking rate varies with the AV interval. In rate adaptive dual chamber pacemakers PVARP can be determined by the sensor, which shortens PVARP during exercise thus allowing higher atrial tracking rates.

6.4.1 Effect of the AV interval and PVARP

In pulse generators with an adaptive AV interval, the AV interval is a function of the preceding P-P interval. If the P-P interval increases, the AV interval becomes longer, while decreasing P-P intervals shorten the AV interval in order to imitate the physiological variation in the PQ time with varying heart rates [7,8].
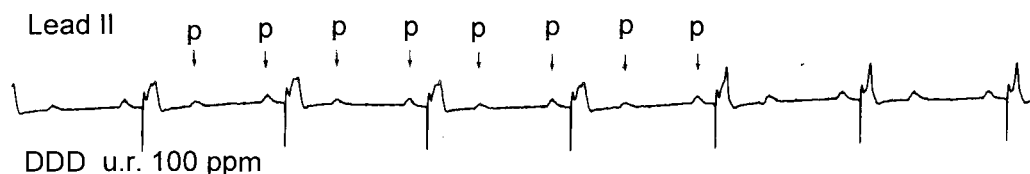
Fig. 6.11. Single channel ECG (lead II) of a patient with an implanted DDD pacemaker programmed at a lower rate of 40 ppm, PVARP 425 ms, AV interval 175 ms, upper rate 100 ppm. The TARP is 600 ms (425 + 175 = URI), thus giving a maximum atrial tracking rate of 100 bpm. Because the atrial rate is 104 bpm, 2:1 AV block is observed. See also figure 6.12.

The effect of an adaptive AV interval on the upper rate behavior of the dual chamber pacing system is best explained by an example. Assume that a pulse generator is programmed to the following setting: AV interval 150 ms, PVARP 350 ms and the upper rate interval 500 ms, which is equal to 120 ppm. Because AV interval + PVARP = 500 ms (TARP), 2:1 block will occur as soon as the atrial rate exceeds 120 bpm.
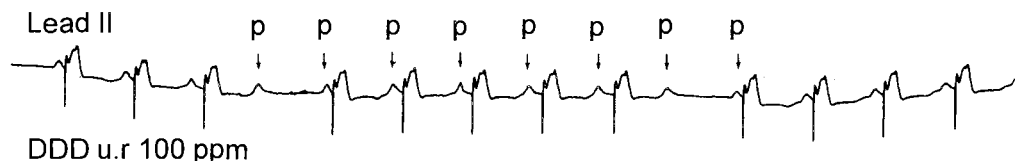
Fig. 6.12. Single channel ECG (lead II) of the same patient as figure 6.11. Programmed settings as well as the atrial rate are unchanged except for the AV interval that is programmed in the adaptive mode. Programming the AV interval in the adaptive mode results in a minimum AV interval of 75 ms, which in combination with a PVARP of 425 ms, gives a minimum TARP of 500 ms and thus a maximum atrial tracking rate of 120 bpm. By only programming an adaptive AV interval the pacing system now shows Wenckebach type behavior instead of 2:1 AV block at the same atrial rate.

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In the case of an adaptive AV interval, the AV interval shortens with decreasing P-P intervals. Assume that PVARP = 350 ms and the AV interval decreases from 150 to 75 ms when the atrial rate increases to 120 bpm. The AV interval of 75 ms together with the PVARP of 350 ms gives a TARP of 350 + 75 = 425 ms. This implies that the maximum atrial tracking rate rises from 120 bpm to 141 bpm. Therefore the upper rate behavior will change from 2:1 AV block at an atrial rate exceeding 120 bpm in case of a fixed AV interval of 150 ms and a PVARP of 350 ms, into Wenckebach type behavior for atrial rates between 120 and 141 bpm if an adaptive AV interval together with a PVARP of 350 ms was used. The change in upper rate behavior by programming an adaptive AV delay is illustrated in the figures 6.11 and 6.12.

A recent report, however, described in a specific DDD pulse generator using an adaptive AV delay an unexpected early 2:1 AV block response. The expected 2:1 block point was calculated using the shortest adaptive AV delay and the programmed PVARP. When the Wenckebach cycle was initiated, the P wave in PVARP was ignored by the pulse generator. Because of this, there was a sudden doubling of the P-P interval that was sensed by the pacemaker, resulting in prolongation of the AV interval. The prolonged AV interval together with the fixed PVARP decreased the maximum atrial tracking rate below the level, of 2:1 AV block development [9]. This phenomenon was defined as upper rate "lockout".

Similar effects can be observed in rate adaptive dual chamber pacemakers, where shortening of the PVARP as well as the AV interval can be dictated by the sensor [10].

6.4.2 Effect of PVARP extension on upper rate behavior

The effect of the PVARP extension, an option that is available in a number of dual chamber pacemakers, on upper rate behavior is explained in the following case report.

Apparent P Wave Undersensing in a DDD Pacemaker Post Exercise.

Berry M. van Gelder, Rob van Mechelen, Karel den Dulk, and Mamdouh I.H. El Gamal.

Summary
Wenckebach behavior of DDD pacemakers occurring when the P-P interval varies between the programmed upper rate interval and the total atrial refractory period is symmetrical in a sense that the pacemaker response during atrial rate acceleration is similar to the pacemaker response during atrial rate deceleration. This phenomenon can be observed in all patients with persistent AV block in whom a DDD pacemaker is implanted, during exercise testing when the spontaneous atrial rate exceeds the selected upper rate, i.e.,the programmed upper rate interval. However, this phenomenon will not be observed in all patients with intermittent intact AV conduction during excercise. In this case report we describe a patient who showed an asymmetrical reponse during a bicycle exercise test. There was 1:1 atrial sensing ventricular pacing until the atrial rate exceeded the upper rate of 140 ppm, while atrial sensing was restored during recovery when the conducted sinus rhythm had decreased to 105 beats/min.

Introduction
In a patient with complete heart block with no episodes of spontaneous AV conduction, it may be anticipated that given an upper rate interval (URI) programmed longer than the selected total

atrial refractory period (TARP), a pacemaker Wenckebach sequence is initiated when the atrial rate exceeds the programmed upper rate. With further increase of the atrial rate up to a rate that corresponds to the selected TARP, 2:1 AV block is noted. This phenomenon is symmetrical in the sense that during subsequent decrease of the spontaneous atrial rate, the 2:1 AV block response disappears and Wenckebach AV block reappears as the spontaneous atrial rate drops below the rate corresponding to the selected TARP. At atrial rates slower than the programmed upper rate, 1:1 AV conduction is resumed and the AV interval equals the programmed AV interval. In clinical practice this kind of pacemaker behavior is observed during excercise tests of patients with complete AV block in whom a DDD pacemaker is implanted. Recently we encountered a pacemaker patient who underwent a DDD pacemaker implantation because of intermittent AV block. In this patient, an exercise test was performed and a pacemaker behavior was noted that differed from what was to be expected in patients with persistent AV block. This patient showed after completion of exercise a conducted sinus rhythm with a PR interval significantly longer than the programmed AV interval, even when the rate of this sinus rhythm dropped below the programmed upper rate. Only when the atrial rate decreased to a rate significantly lower than the upper rate was 1:1 AV synchronous pacing at the programmed AV interval observed. There was apparent P wave undersensing in the recovery phase when the atrial rate dropped below the upper rate. In this article, the case study is presented and an explanation is given for this phenomenon.

Case study

A bipolar DDD pacemaker (Cosmos II[Intermedics Inc., Angleton, TX, USA]) was implanted in a 73-year-old male with right bundle branch block, left anterior hemiblock, prolonged PR interval, and symptomatic intermittent complete heart AV block.
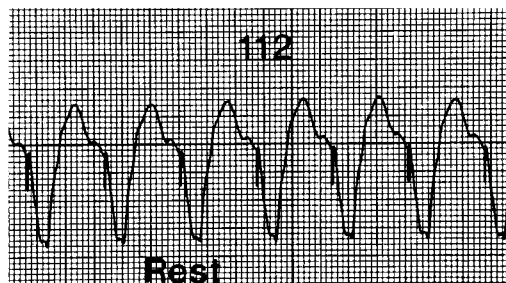
Fig. 6.13. ECG lead II at rest showing atrial synchronous ventricular paced rhythm of 112 ppm.
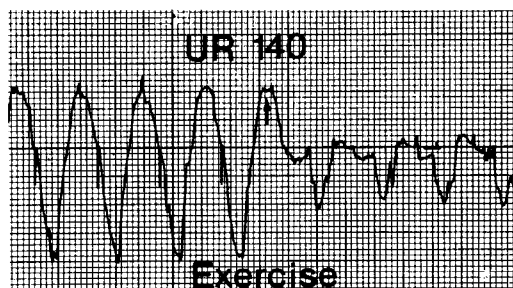
Fig. 6.14. ECG lead II showing atrial synchronous ventricular paced rhythm at the upper rate (UR). Ventricular pacing is not synchronized to a P wave (arrow), which occurs in the postventricular atrial refractory period during a pacemaker Wenckebach sequence. This allows conduction of the P wave to the ventricle with a PQ interval of approximately 260 msec and complete right bundle branch block. AV synchrony with a short AV interval is no longer maintained by the pacemaker.

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Leads were implanted in the right atrial appendage and right ventricular apex, respectively. Three weeks after implantation a bicycle exercise test was performed in our institution. The pulse generator was programmed as follows: lower rate 60 ppm, upper rate 140 ppm, and AV delay after P wave sensing 135 msec. AV delay of 75 msec. Postventricular atrial refractory period (PVARP) was 320 msec. Atrial refractory extension (ARE) was 100 msec.

ARE is activated and results in prolongation of PVARP if the pacemaker senses a ventricular event not preceeded by an atrial pace or sense event. This setting implicates that the Wenckebach upper rate reponse will occur at atrial rates between 140 and 152, and that 2:1 AV block occurs when the atrial rate exceeds 152 beats/min (PVARP + minimum AV delay = 320 + 75 = 395 msec, corresponding to a rate of 152 beats/min).
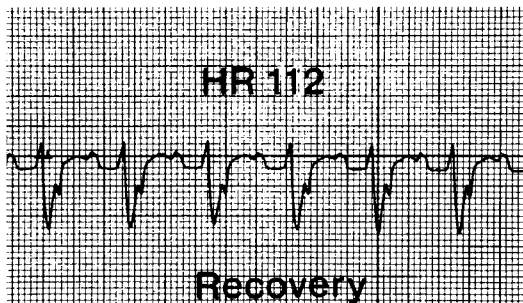
Fig. 6.15. ECG lead II showing a decrease in heart rate (HR) to 112 beats/min during the recovery phase. There is a conducted sinus rhythm with a long PQ time (200-220 msec) and right bundle branch block. An atrial synchronous ventricular paced rhythm with a short AV interval (adaptive AV interval 75-135 msec) is still absent despite a sinus rate below the upper rate limit
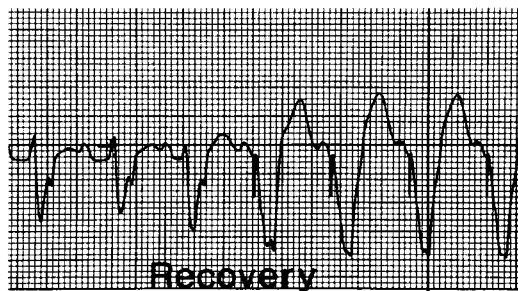
Fig. 6.16. Later on in the recovery phase, ECG lead II shows a change in rhythm from sinus with a PQ of 200 msec to atrial synchronous ventricular paced rhythm with an AV interval of 100 msec. Analysis of the PQ intervals preceding ventricular pacing shows an abrupt shortening of approximately 40 msec, without a change in P-P interval. This results in earlier detection of the conducted QRS complex and subsequently the next P wave falls outside PVARP + ARE interval of 420 msec.

At rest the electrocardiogram showed atrial sensing ventricular pacing at a rate of 112 beats/min with a short AV interval of approximately 100 msec (Fig. 6.13). After 1 minute of exercise with a load of 80 watts, atrial sensing followed by ventricular pacing suddenly changed to a conducted sinus rhythm with type I AV block and right bundle branch block (Fig. 6.14).

During the recovery period a gradual decrease in sinus rate to 112 beats/min with a PQ interval of 200 msec, atrial synchronous ventricular pacing had not returned (Fig. 6.15).

Further observation revealed a sudden change in rhythm from a conducted sinus with a PQ interval of 200 msec to an atrial synchronous ventricular paced rhythm with an AV interval of approximately 100 msec (Fig. 6.16).

During the remaining recovery time no further abnormalities occurred. After exercise test, the pacing system was checked for atrial sensing. There was proper atrial sensing at 2.0 mV

sensitivity. During the exercise test, the pacemaker was programmed to 0.8 mV sensitivity, thus excluding atrial undersensing due to marginal programming.

Discussion

Upper rate behavior in DDD pacemakers depends on the programmed URI and the TARP. The URI is defined as the shortest interval between two consecutive ventricular stimuli or from a sensed ventricular event to the succeeding ventricular stimulus. The TARP is identical to the effective atrial refractory period and corresponds to the minimum effective atrial sensing interval [1]. TARP is equal to the sum of the AV interval and PVARP. In the presence of rate adaptive AV interval, TARP shortens as the AV interval shortens. A sensed AV interval can be shorter than a paced AV interval in which case the TARP is shortened by the same amount.

Intrinsic Rhythm > Upper rate
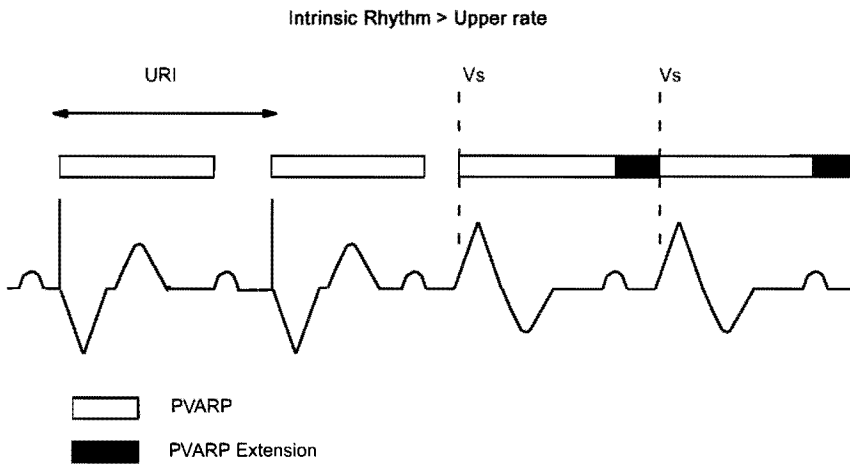
Fig. 6.17. Timing diagram of the ECG in Figure 6.14 illustrating the mechanism that prevents atrial synchronous ventricular pacing when the atrial rate exceeds the upper rate limit. The blocked P wave of the pacemaker Wenckebach cycle is conducted to the ventricle through the normal conduction system. This ventricular event is seen by the pacing system as a premature ventricular event, resulting in extension of the postventricular atrial refractory period (PVARP). This repeated for the next ventricular events. URI = upper rate interval; Vs = ventricular sensing.

In the pulse generator of our patient (Cosmos II), PVARP can be automatically extended if the generator percieves a condition that may lead to retrograde conduction. It is implemented when a premature ventricular event is sensed. Pacemaker reprogramming, magnet removal, noise sensing, and change from temporary to normal operation also activates the atrial refractory extension [2]. DDD pacemakers where URI>TARP show Wenckebach or pseudo Wenckebach behavior when the P-P interval becomes shorter than the URI. This results in prolongation of the AV interval to a maximum, the so-called Wenckebach period (W= URI-TARP). The next atrial event, following the atrial event with maximum AV interval, will be ignored by the atrial amplifier (falls in PVARP) and will not result in ventricular stimulation.

Decrease of the P-P interval to an interval shorter than TARP will result in 2:1, 3:1 AV block.

Several reports have been published on the subject of upper rate behavior, with special attention to the Wenckebach phenomenon [3-5]. In general they focus on DDD pacing for complete heart

block and are restricted to the period when the atrial rate exceeds the programmed upper rate limit, while little or no attention has been paid to the recovery phase when atrial rate drops below the upper rate. In the case of complete heart block, this phase is of no interest because atrial sensing ventricular pacing with a 1:1 reponse is restored when the P-P interval drops below the URI. There is a symmetrical ventricular response to atrial sensing during acceleration and deceleration of the atrial rate.

In patients with DDD pacemakers in whom antegrade AV conduction with a 1:1 ventricular response is still present, there is a different behavior in the exercise as compared to the recovery phase, when the atrial rate increases above and subsequently decreases below the upper rate limit. A prerequisite for this asymmetrical behavior is that the programmed AV interval after P wave sensing is shorter than the antegrade AV conduction time; this implies that at rest there is atrial sensing ventricular pacing. Initiation of exercise gives a rise in atrial rate and atrial synchronized ventricular pacing will be observed. When the P-P interval becomes shorter than the URI, the atrial sensing ventricular pacing interval prolongs and the Wenckebach cycle will be initiated. If the last P-wave of the Wenckebach cycle falls in the atrial refractory period and this P-wave is conducted antegradely to the ventricle, there is a change in the timing cycle of the pulse generator. The first ventricular conducted sinus beat (Fig. 6.17) detected by the pulse generator is regarded as a ventricular premature beat because the preceeding P wave falls in the atrial refractory period and is ignored by the system. This results in extension of PVARP and the next P wave also falls in the extended atrial refractory period. This means that the next sensed ventricular event (R wave) is again regarded as a ventricular premature beat with resulting prolongation of PVARP.

Mechanism for restoration atrial sensing
PP interval > PR + PVARP + PVARP extension
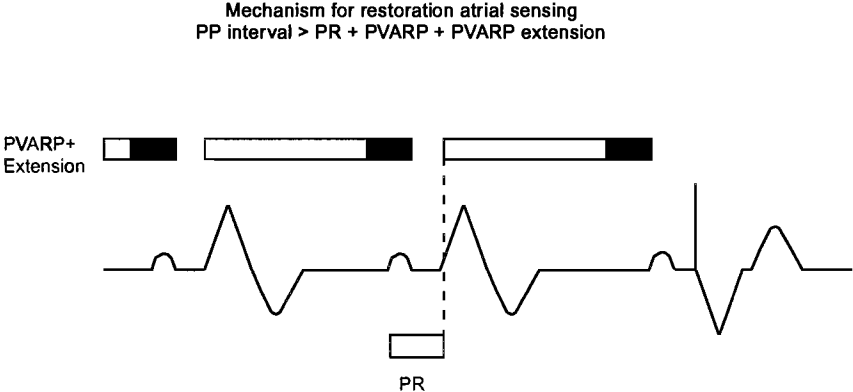
Fig. 6.18. Timing diagram of restoration of atrial sensing. P wave sensing is restored when the PP interval is equal to the sum of PR interval + PVARP + PVARP extension.

Continuation of this timing sequence implies that decrease of the atrial rate below the upper rate of the pulse generator does not restore atrial sensing immediately.The diagram in figure 6.18 shows that atrial sensing is restored when the PP interval is longer than the sum of PR interval + PVARP + ARE. In our patient, Figure 6.16 shows restoration of atrial sensing at an atrial rate of approximately 110 beats/min. The measured PQ interval of the conducted beat preceding the first ventricular paced beat is approximately 140 msec, and together with the programmed PVARP

(320 msec) and ARE (100 msec) results in an interval of 560 msec corresponding to a rate of 105 beats/min, which confirms the hypothesis of the phenomenon.

Under the conditions described in our patient, it is clear that during exercise atrial sensing is maintained until the atrial rate exceeds the programmed upper rate and the Wenckebach cycle is initiated. Restoration of atrial sensing occurs when the P-P interval is longer than the sum of PR interval + PVARP + ARE. If PR + PVARP + ARE is longer than the programmed URI, restoration of atrial sensing will occur at a lower rate than the programmed upper rate. This asymmetrical response can be defined as a hysteresis effect in atrial sensing depending upon the increase and decrease of the atrial rate with the respect to the upper rate. The phenomenon of apparent P wave undersensing by extension of PVARP has been published previously by several authors [6-9]. The phenomenon in these case was initiated by ventricular premature beats, intermittent ventricular noncapture, and artificial ventricular sensing.

The clinical importance of this phenomenon is that it can easily be misinterpreted as atrial undersensing.

References

1. Barold SS. Upper rate of DDD pacemakers. The view from the atrial side. PACE 1988;11:2149.

2. Cosmos II. Physician's Manual Models 283-03 and 284-05. Freeport, TX, Intermedics, Inc., March 1990.

3. Furman S. Dual chamber pacemakers. Upper rate behavior. PACE 1985;8:197.

4. Barold SS, Falkoff MD, Ong LS, et al. Upper rate response of DDD pacemakers. In SS Barold, J Mugica (eds.): New Perspectives in Cardiac Pacing. MT. Kisco, NY, Futura Publishing Co., Inc., 1988, p.121.

5. Furman S. Comprehension of pacemaker timing cycles. In S Furman, DL Hayes, DR Holmes (eds.): A Practice of Cardiac Pacing. Mt. Kisco, NY, Futura Publishing Co., Inc., 1989, p.135.

6. Satler LF, Rackley CE, Pearle DL, et al. Inhibition of a physiologic pacing system due to its anti-pacemaker-mediated tachycardia mode. PACE 1985; 8: 806-810.

7. Greenspon AJ, Volosin KJ. "Pseudo" loss of atrial sensing by a DDD pacemaker. PACE 1987; 10: 943-948.

8. Wilson JH, Lattner S. Undersensing of P waves in the presence of an adequate P wave due to automatic postventricular atrial refractory period extension. PACE 1989; 12:1729-1732.

9. Den Dulk K, Wellens HJJ. Failure of the post-ventricular premature beat DVI mode preventing pacemaker circus movement tachycardia. Am J Cardiol 1984; 54: 1371-1372.

6.5 Crosstalk

Crosstalk is often defined as inappropriate sensing of a pacemaker output pulse or an intrinsic event by the pacemaker sensing channel for the chamber other than the one the paced or intrinsic event occurred in [11]. Although this definition can be applied to atrial events sensed by the ventricular channel and vice versa, in practice it is only used when an atrial output pulse is sensed by the ventricular channel of the pacemaker [12]. If no measures are taken, this can result in self-inhibition of the pacing system, a potentially life-threatening situation for pacemaker dependent patients. This form of crosstalk can be encountered in DDD, DDI as well as DVI pacemakers [13].

If a ventricular event is sensed by the atrial channel, it also meets the definition of crosstalk. If this occurs, however, the ventricular event sensed by the atrial channel results in a ventricular paced event, which in turn can be sensed by the atrial channel, resulting in pacemaker tachycardia or arrhythmia [14,15]. In practice this will be defined as pacemaker mediated tachycardia or arrhythmia, instead of crosstalk. This paragraph will be limited to the effect of atrial stimulation on ventricular sensing.

6.5.1 Factors affecting crosstalk

In the definition of crosstalk, crosstalk is caused by the atrial output pulse. In practice, however, it is not the atrial output pulse itself but the afterpotential of the atrial output pulse, since all modern pulse generators initiate a ventricular blanking period during atrial stimulation, which is longer than the duration of the atrial pulse. The ventricular blanking period is a temporary disabling of the ventricular sensing amplifier following delivery of an atrial output pulse. The ventricular blanking period can vary between 12 and 60 ms, depending on the manufacturer and in some models it is automatically adjusted by the setting of atrial output and ventricular sensitivity. Because crosstalk can still be encountered in pacemakers with ventricular blanking periods, the atrial output pulse itself can be excluded as the cause of crosstalk. The following factors can affect crosstalk:

1. Atrial output (amplitude, pulse width)
2. Ventricular sensitivity
3. Duration of the ventricular blanking period
4. Electrode configuration (unipolar/bipolar)
5. Location and distance of the atrial and ventricular electrode
6. The stimulation rate
7. Pulse generator circuitry
8. Lead impedance

The first two factors, atrial output and ventricular sensitivity, are obvious. The larger the atrial output, the greater the chance is that the afterpotential of the atrial pulse will be sensed by the ventricular channel. A higher sensitivity of the ventricular channel will make sensing of the atrial pulse afterpotential more likely.

Increase atrial output   decrease
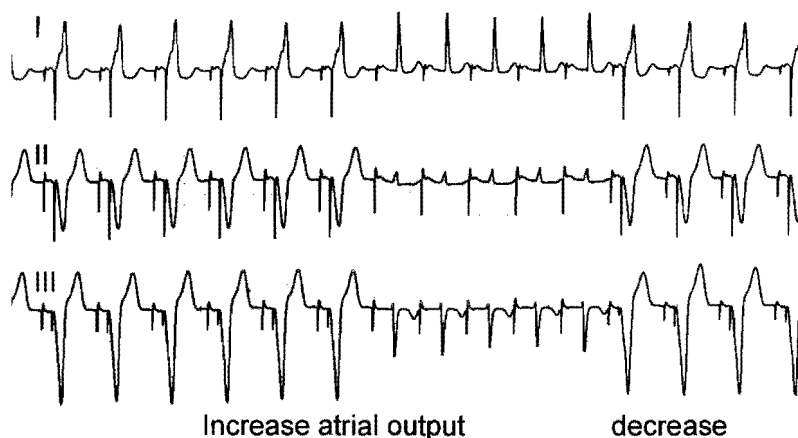
Fig. 6.19. Three channel ECG (leads I,II,III) of a patient with an external DVI pacemaker. During atrial output increase, crosstalk with inhibition of the ventricular stimulus is observed. After decreasing atrial output, normal pacemaker function is restored.

Both phenomena are illustrated in figures 6.19 and 6.20 in a patient with an external DVI pacemaker. Increasing of the atrial output as well as the ventricular sensitivity provokes

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crosstalk, in this pulse generator resulting in complete inhibition of the ventricular output. Decrease of output and sensitivity restores normal AV sequential pacing. A longer ventricular blanking period reduces the risk of crosstalk as afterpotentials are time dependent phenomena due to the fact that the afterpotentials decrease in time after emission of a stimulus.

A longer ventricular blanking period, however, also increases the period that the ventricular channel is disabled for ventricular sensing, with the potential risk of ventricular undersensing (see paragraph 6.7).
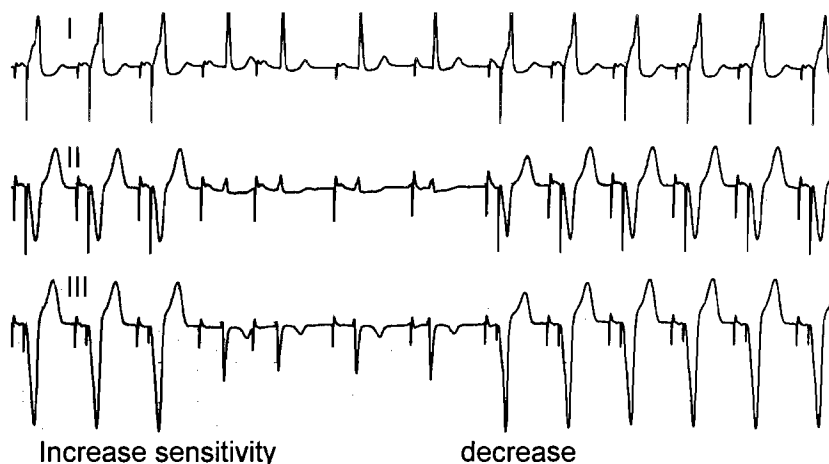
Fig. 6.20. Three channel ECG (leads I,II,III), same patient of figure 6.19 with an external DVI pacemaker. Crosstalk with inhibition of the ventricular stimulus is now observed after increasing the ventricular sensitivity. Decreasing ventricular sensitivity results in normal AV sequential pacing.

The electrode configuration plays a role in crosstalk. It is merely bipolar ventricular sensing that prevents crosstalk. The ventricular electrode dipole in the bipolar configuration is small and relatively remote from the site of atrial output pulse generation and the bipolar configuration makes it less prone to far-field phenomena. In the unipolar configuration the ventricular electrode dipole is large, and the atrial ouput pulse is generated between the electrodes of the ventricular channel, which facilitates sensing of the afterpotentials. Because crosstalk is a far-field phenomenon, the distance between the atrial and ventricular electrode plays a role in the presence or absence of crosstalk. The larger the distance between the two electrodes, the less risk of crosstalk induction.

The stimulation rate also affects crosstalk. According to Byrd et al.[16] no crosstalk was observed in unipolar DDD pacing at a lower rate of 60 ppm, but it was present in 31% of patients if pacing was performed at a lower rate of 130 ppm and in 54,5% when the pacing rate was increased to 160 ppm. The incidence of crosstalk was significantly lower but not completely eliminated in bipolar pacing systems: only 25% at a pacing rate of 160 ppm. At high stimulation rates the shortened pacing intervals decrease the time allowed for dissipation of charge generated by the atrial output pulse.

Although theorically the incidence of crosstalk is lower in bipolar systems [17], a few reports describe a higher incidence of crosstalk in the bipolar setting, is related to the pacemaker

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circuitry. Beaver et al.[18] report crosstalk in a series of bipolar pacemakers, in which the anodal electrodes of the leads were interconnected by a wire inside the pulse generator. A similar bipolar pulse generator of the same manufacturer, without the connecting wire showed no crosstalk. The direct galvanic coupling between the anodal electrodes was the cause of crosstalk and can be considered as design dependent.
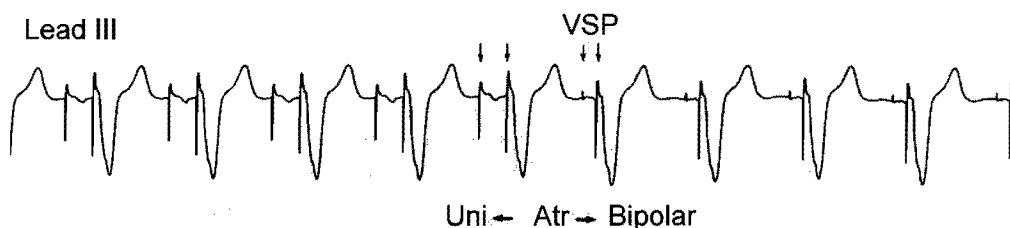
Fig. 6.21. Single channel ECG (lead III) of a patient with an implanted DDD pacemaker. The electrode configuration is programmable in this pulse generator. After programming the atrial electrode configuration from unipolar to bipolar, shortening of the AV interval indicating ventricular safety pacing (VSP) due to crosstalk is observed.

In a dual chamber pacemaker from another manufacturer crosstalk has been observed after programming the atrial electrode from the unipolar to the bipolar configuration (Fig. 6.21). During this observation the ventricular channel was programmed in the unipolar configuration. The explanation for this phenomenon is unclear, neither other authors [19,20] nor the manufacturer [21] could given the explanation, hence it also has to be considered as design dependent, since similar settings in pulse generators of other manufacturers reduce crosstalk in this configuration.
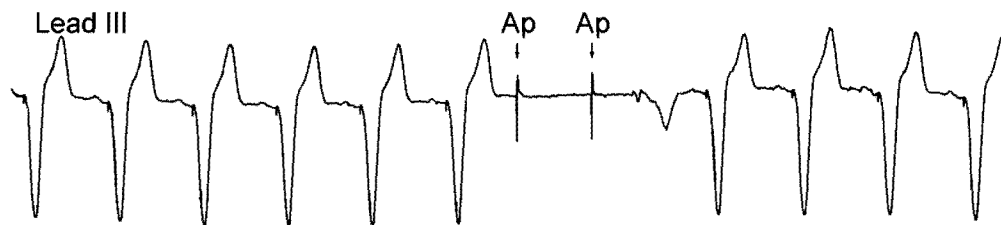
Fig. 6.22. Single channel ECG (lead III) of a patient with an implanted DDD pacemaker. Initially there is normal pacemaker function, showing atrial sensing ventricular pacing. After emission of two consecutive atrial stimuli (Ap), ventricular stimulation is inhibited due to crosstalk. After the second atrial stimulus a ventricular escape beat is recorded, after which normal function is restored. Ventricular safety pacing is not incorporated in this particular pulse generator.

An unusual type of crosstalk has been reported by Barold [19], this was not induced by atrial stimuli but during activation of atrial sense markers. Upon activation of the atrial sense markers, the pulse generator delivers a series of markers in the form of P-wave triggered atrial stimuli at 0.025 ms in duration at the programmed voltage output of the atrial channel. These markers are used to identify atrial sense events on the ECG. Under certain circumstances these stimuli may

cause crosstalk when they are sensed by the ventricular sensing amplifier. Emission of the marker pulses does not initiate a blanking period or a ventricular safety period (see next paragraph) in contrast to the conventional emission of atrial stimuli during DDD pacing.

Occasionally crosstalk can be caused by lead failure or lead displacement. One report describes self inhibition of an AV sequential demand (DVI) pulse generator due to polyurethane lead insulation disruption [22]. The insulation defect facilitated detection of the atrial stimulus by the ventricular lead. Also, lead displacement, in those cases where the distance between the atrial and ventricular electrodes becomes smaller, can induce crosstalk. E.g. displacement of the atrial lead to the lower right atrium near the tricuspid valve, or even in the right ventricular inflow or outflow tract.

6.5.2 Crosstalk protection
Previous paragraphs have elucidated the potential risk of crosstalk in patients with no underlying intrinsic rhythm. If no measures are taken it can result in complete inhibition of the ventricular channel as illustrated in figure 6.22. In order to prevent this undesired complication, two protection mechanisms are incorporated in the majority of today's pulse generators:
1. Ventricular blanking period.
2. Ventricular safety pacing.

6.5.2.1 Ventricular blanking period
The ventricular blanking period is the period after delivery of an atrial stimulus during which the ventricular channel is temporarily disabled for ventricular sensing. Because afterpotentials due to the atrial pulse are time dependent, the longer the ventricular blanking period the lower the risk of crosstalk. Timing of the ventricular blanking period can be very critical as illustrated in figure 6.23.

Fig. 6.23. Single channel ECG (lead III) of a patient with an implanted DDD pacemaker programmed at a lower rate of 90 ppm and an AV interval of 200 ms. After reducing the ventricular blanking period from 27 to 26 ms, shortening of the AV interval is recorded, indicating ventricular safety pacing induced by crosstalk.

Crosstalk is observed with a ventricular blanking period of 26 ms and disappears after programming ventricular blanking to 27 ms. However, disabling the ventricular input channel for a longer period of time induces the risk of undersensing ventricular intrinsic activity. In some pulse generators the duration of the ventricular blanking period is automatically adjusted on the basis of the currently programmed atrial output and ventricular sensitivity settings. It may also be manually programmed, in case the automatically programmed blanking interval is still insufficient to give full protection against crosstalk [23].

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6.5.2.2 Ventricular safety pacing

This feature is designed to augment the ventricular blanking function to ensure that crosstalk inhibition will not occur. If for some reason the ventricular blanking period is too short, the crosstalk sensing window, which begins at the end of the ventricular blanking period, serves as a backup. Every signal sensed in the ventricular channel during this interval is interpreted as crosstalk, and causes the pulse generator to deliver a ventricular output at the end of this interval (ventricular safety pacing).

Atrial pulse width 0.3 ms   ⌐760⌐   0.5 ms   ⌐660⌐
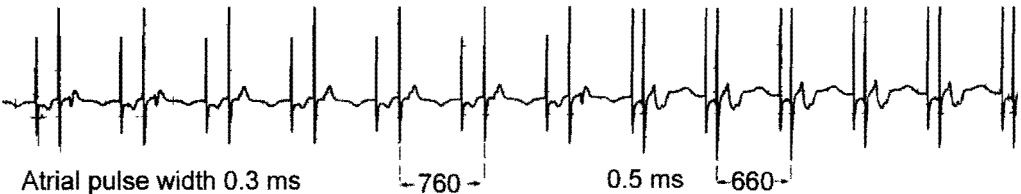
Fig. 6.24. Single channel ECG (lead I) of a patient with an implanted DDD pacemaker. After programming the atrial pulse width from 0.3 to 0.5 ms, crosstalk is signaled on the ECG in the form of recurrent "early" ventricular pacing, or AV sequential pacing with a shortened AV delay. Ventricular safety pacing. Shortening of the AV interval results in shortening of the A-A interval (760 -> 660 ms) in this atrial based timing system.

The duration of this interval is determined by the specifications of the pulse generator. In practice this interval will be between 100 - 110 ms. The reason for limiting this interval to 110 ms is

V Sensing during AV Interval.
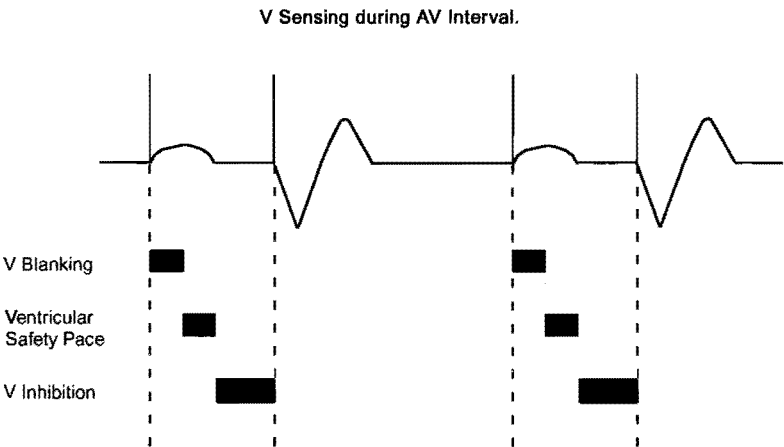
Fig. 6.25. Schematic presentation of the segments of the AV interval, the ventricular blanking period, the ventricular safety pacing period and the ventricular sensing inhibiting period (V inhibition).

explained in paragraph 6.7. Some manufacturers define ventricular safety pacing as pacing with a nonphysiological AV delay, this in contrast with the programmed AV delay, which will be

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longer in the majority of cases. If the AV interval after an atrial paced event is programmed longer than the safety pacing interval, crosstalk will be signalled on the ECG in the form of recurrent "early" ventricular pacing, or AV sequential pacing with a shortened AV interval (Fig. 6.24). This indicates that crosstalk is present and that the atrial output (amplitude, pulse width), the ventricular sensitivity or the blanking period may need adjustment [24,25].

6.6 Electrocardiographic consequences of ventricular safety pacing and ventricular blanking

In dual chamber pacemakers in which ventricular blanking and ventricular safety pacing are incorporated, the AV interval can be divided into three parts: the ventricular blanking period, the ventricular safety pacing period and the ventricular sensing inhibiting period (Fig. 6.25). Presence of crosstalk will be indicated by shorthening of the AV interval (interval between atrial pace and ventricular pace event). However, not only crosstalk, but every ventricular sense event, whatever its origin, which occurs during the safety pacing interval will shorten the AV interval. Because shortening of the AV interval or ventricular safety pacing is not only initiated by crosstalk, some authors consider the term "ventricular triggering period" to be more appropriate [26]. During the ventricular triggering period, no signal (atrial stimulus producing crosstalk, QRS complex, myopotentials, etc.) sensed by the ventricular channel inhibits the DDD pulse generator.
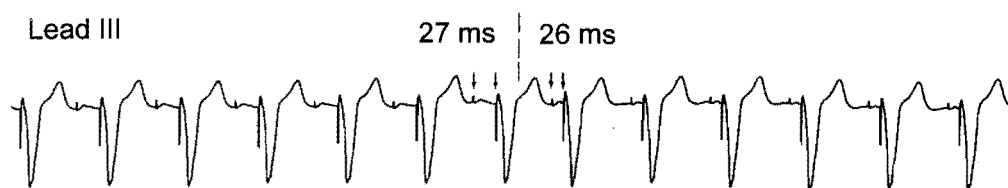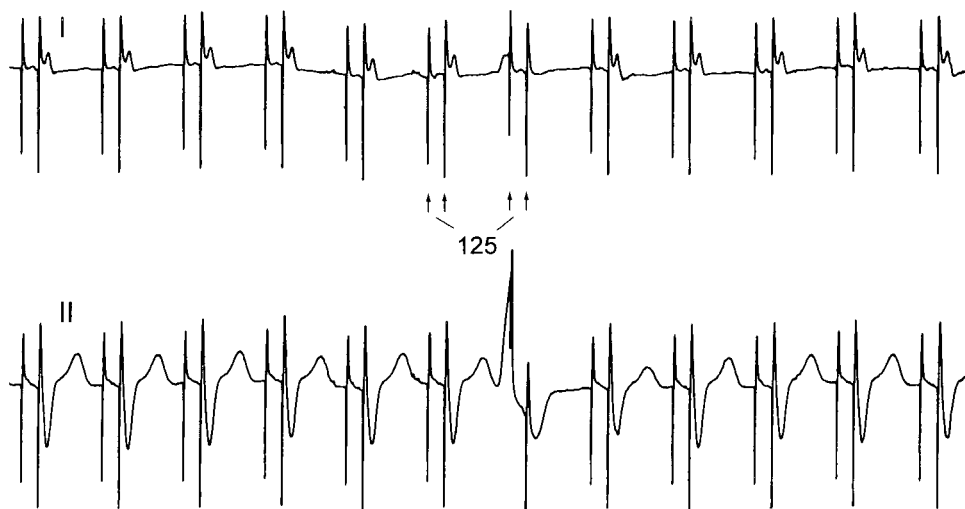

Fig. 6.26. Two channel ECG (lead I,II) of a patient with an implanted DDD pacemaker, programmed at a lower rate of 100 ppm and an AV interval of 125 ms. An atrial stimulus is emitted before the ventricular premature beat is sensed in the ventricular channel. Because the ventricular event occurs in the ventricular blanking period, the AV interval is not affected.

Rather it initiates or triggers a ventricular stimulus delivered prematurely only at the completion of the ventricular triggering period, producing a characteristic abbreviation of the paced AV interval. Although the arguments for using the term ventricular triggering period were valid, the term is not generally acceptedat the moment.

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Ventricular events occurring during the ventricular blanking period will be ignored by the ventricular channel and the ventricular stimulus will be emitted at the end of the programmed AV interval, provided no other ventricular sensed events occur in the remaining part of the AV
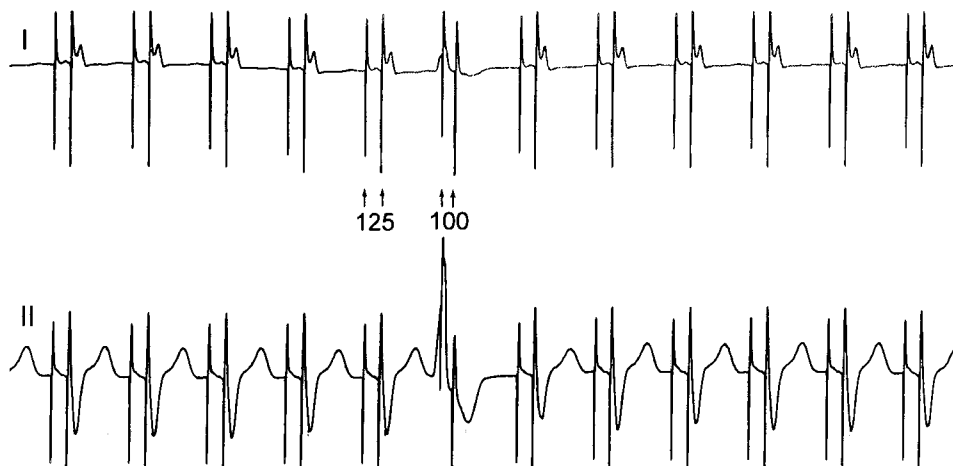
Fig. 6.27. Two channel ECG (lead I,II), same patient and same setting as figure 6.26. The ventricular premature beat has a slightly longer coupling interval with the preceding ventricular paced beat, resulting in sensing of this beat during the ventricular safety pacing period and shortening of the AV interval to 100 ms.

Fig. 6.28. Two channel ECG (lead I,II), same patient and same setting as figure 6.26 and 6.27. The coupling interval between the ventricular premature beat and the preceding ventricular paced beat is slightly longer when compared to figure 6.27. The ventricular premature beat occurs during the AV interval outside the ventricular safety pacing period resulting in inhibition of the ventricular stimulus.

interval. Figure 6.26 shows an ECG of a patient with a DDD pacemaker, stimulating at a lower rate of 100 ppm and an AV interval of 125 ms. A ventricular premature beat occurs more or less

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simultaneously with the atrial stimulus. The following AV interval is equal to the programmed interval of 125 ms, indicating that the moment of ventricular sensing occurs during the blanking period and the AV interval is not effected. However, the ECG can be easily misinterpreted as ventricular undersensing. In the same patient an identical ventricular premature beat with a longer coupling interval to the preceding ventricular paced beat illustrated proper ventricular sensing. In this recording (Fig. 6.27) the ventricular sense event occurs during the ventricular safety pacing interval, which results in shortening of the AV interval, thus confirming ventricular safety pacing. In figure 6.28, from the same patient, the same ventricular premature beat occurs with a longer coupling interval than in figure 6.27. The atrial stimulus is now just preceding the ventricular premature beat. Ventricular sensing occurs outside the ventricular safety pacing period and the ventricular output pulse is inhibited. All three ECGs illustrate the relationship between the moment of the ventricular sense event during the different parts of the AV interval and the reaction of the ventricular channel.
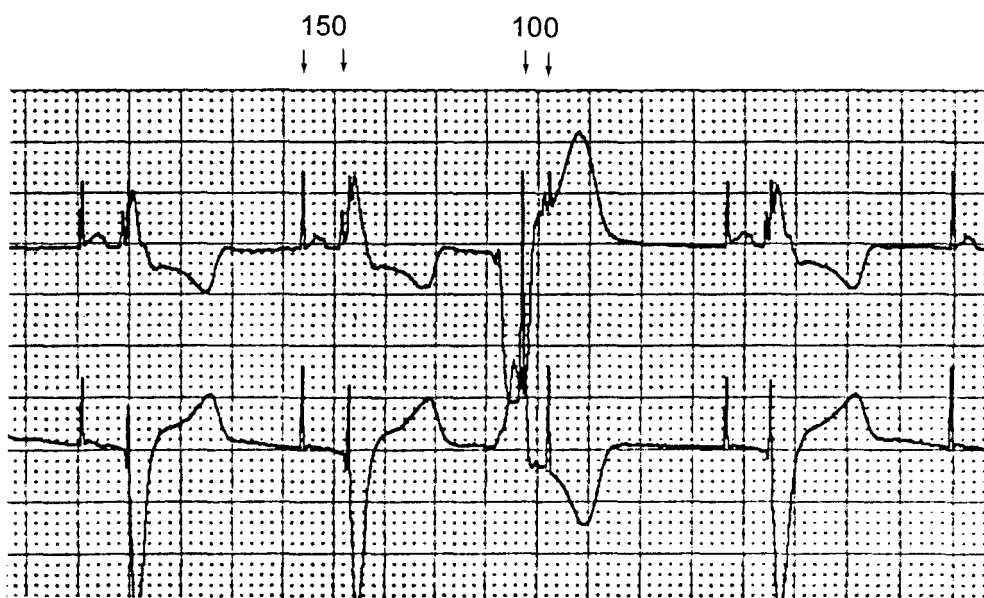
Fig. 6.29. Two channel Holter recording of a patient with an implanted DDD pacemaker; AV interval 150 ms. The atrial impulse in the ventricular premature beat is emitted before the ventricular premature beat is sensed. The ventricular premature beat is sensed during the ventricular safety pacing period resulting in a ventricular stimulus with a shortened AV interval (100 ms). The ventricular stimulus is emitted in the first portion of the intrinsic T wave.

To summarize, a ventricular sense event during the blanking period will not affect the programmed AV pace interval. A ventricular sense event during the ventricular safety pacing interval will evoke a ventricular stimulus with a shortened AV interval. A ventricular sense event during the AV interval outside the blanking and safety pacing interval will inhibit the ventricular ouput pulse.

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Ventricular safety pacing can sometimes be misleading in the ECG interpretation, especially when ventricular safety pacing is caused by ventricular premature beats originating from the left ventricle. The detection time by the right ventricular electrode for these premature beats, is relatively late. This implies that the atrial stimulus can still be emitted late in the QRS complex. Emission of the atrial impuls initiates a blanking period followed by the ventricular safety pacing period. The ventricular stimulus can be emitted in the first portion of the intrinsic T wave (Fig. 6.29).

6.7 Ventricular blanking and a long paced AV interval
The effect of the ventricular blanking period combined with a long paced AV interval in rate adaptive dual chamber pacemakers is illustrated and discussed in the following case report.

Programming a Long Paced Atrioventricular Interval May Be Risky in DDDR Pacing

Mattie G.C. Pieterse, Karel den Dulk, Berry M van Gelder,Rob van Mechelen, and Hein J.J. Wellens.

Summary
In patients with intermittent AV block and dual chamber pacemakers, a long paced AV interval of 200 msec or more can be selected to prolong pulse generator life (by avoiding the ventricular pace output) and to enable a more physiological and hemodynamically superior activation sequence. This case report describes the potential riks of programming a long paced AV interval in a patient with a DDDR pacemaker. T wave pacing, as described here, can occur if the conducted QRS complex is not sensed because it occurs during the ventricular blanking period (delivery of the atrial stimulus). This can be initiated by the mechanisms that induce apparent and actual P wave undersensing of the conducted QRS complex. In this case report apparent P wave undersensing and subsequent T wave pacing with ventricular capture (in a patient with intermittent AV block) occurred frequently during an exercise test done in the DDDR mode with a paced AV interval of 200 msec, according to the clinical evaluation protocol.

Introduction
In patients with persistent atrioventricular (AV) block, a paced AV interval of 150-200 msec is often selected when programming a DDD pacemaker. However, in patients with intermittent AV block a long paced AV interval of 200 msec or more can be selected to give natural AV conduction the chance to depolarize ventricular myocardium. As will be demonstrated in this case report, undersensing of conducted QRS complexes can occur frequently in the DDDR mode with blanking periods as short as 20 msec. This case report illustrates the potential danger of T wave pacing that can occur when programming a long paced AV interval during DDDR pacing. In addition the advantages and disadvantages of programming a long paced AV interval are also discussed.

Case Report
A bipolar dual chamber activity rate variable pacemaker (model 7086, Elite II, Medtronic Inc., Minneapolis, MN, USA) was implanted in a 35 year old male for symptomatic paroxysmal

Mobitz II AV block. To evaluate the response of the activity sensor during the clinical evaluation protocol an excercise test was performed. According to protocol the pacemaker was programmed to the DDDR mode with a lower rate of 50 ppm, and an upper tracking rate of 150 ppm, and an upper activity rate of 120 ppm. The atrial and ventricular amplitudes were 5.0 V with a pulse width of 0.48 msec. Atrial and ventricular sensitivity were 0.5 and 2.5 mV, respectively. The pace and sense configuration was bipolar, rate adaptive AV interval and safety window pacing (SWP) were programmed on. The ventricular blanking was set at 20 msec, the paced AV interval was 200 msec, and the sensed AV interval 180 msec. The postventricular atrial refractory period (PVARP) was programmed to 300 msec and the atrial blanking period was set to 125 msec.

When the rate adaptive AV interval is programmed on in the Elite II pacemaker, the sensed AV delay is shortened from the programmed value to 65 msec as soon as the P-P interval is shorter than 500 msec (120 beats/min)[1]. As can be seen in Figures 6.30 and 6.31 an atrial sensed ventricular paced rhythm with a rate adaptive AV interval of 65 msec was changed, after a ventricular premature beat (VPB), to a sinus rhythm with spontaneous conduction because the first atrial sensed ventricular paced AV interval after the VPB changed back to the programmed sensed AV interval of 180 msec (ventricular spike can be seen in the last part of the QRS complex).
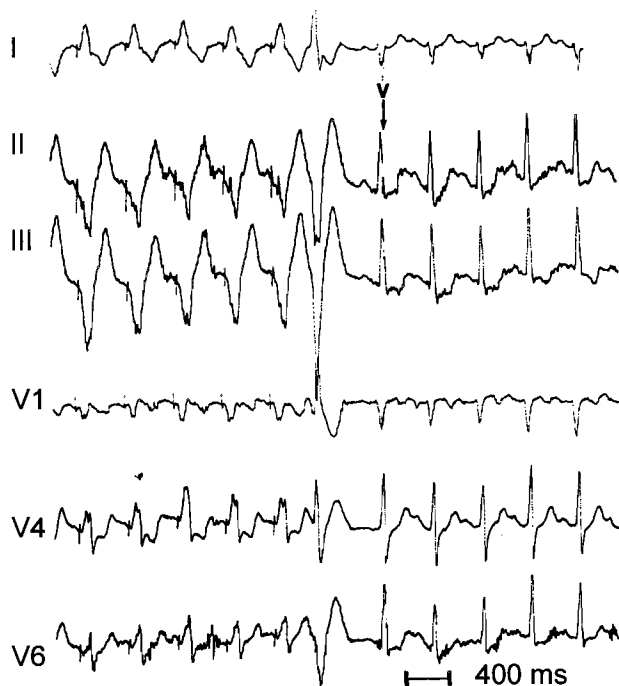
Fig.6.30. Simultaneously recorded 6-channel ECG illustrating loss of the atrial synchronized ventricular paced rhythm (with a rate adaptive atrioventricular [AV] interval of 65 msec) after the first P wave following a ventricular premature beat (VPB), because the subsequent P waves occur during the postventricular atrial refractory period (PVARP). In this device a rate adaptive AV delay is initiated when the P-P interval is shorter than 500 msec as is seen before the occurrence of the VPB. After the VPB the device times out the programmed sense AV interval of 180 msec, which explains the artifact seen in the last part of the QRS complex after the VPB (V). The next P wave occurs within 300 msec of this ventricular stimulus and, therefore, occurs within the PVARP of 300 msec and the following P wave occurs within the PVARP.

The switching from a rate adaptive AV delay of 65 msec back to the programmed sensed interval of 180 msec, occurred because one sinus P wave occurred during the atrial blanking period after

sensing of the VPB. The pacemaker sensed P-P interval surrounding the VPB was, therefore, longer than 500 msec. The PVARP of 300 msec is started late during the QRS complex (after
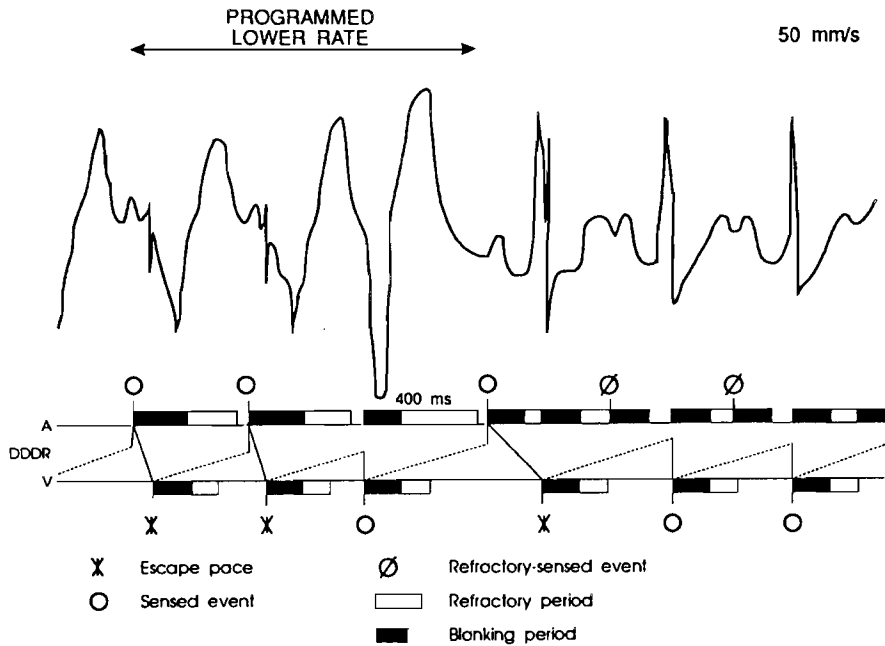
Fig. 6.31. Timing diagram explaining the pacemaker behavior in Figure 6.30.
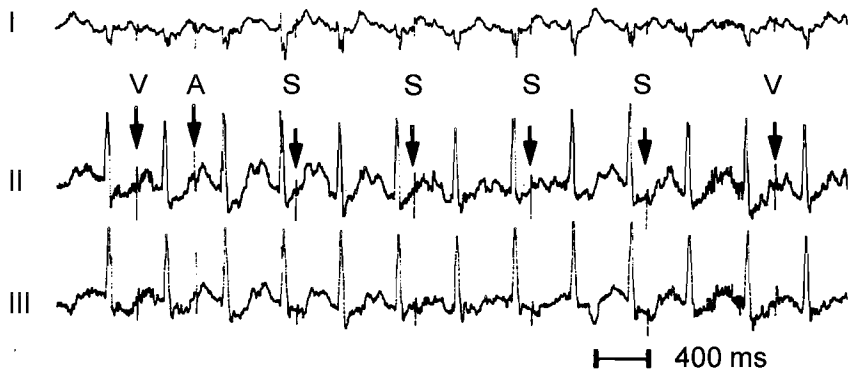
Fig. 6.32. Simultaneously recorded 3-channel electrocardiogram at the onset of the exercise test. Sinus rhythm with conducted QRS complexes occurring in and just after the ventricular blanking period after an atrial stimulus. The atrial stimulus which is often hidden in the QRS complex, is followed by a ventricular stimulus (V) at the programmed AV interval of 200 msec (arrows) or by a shortened AV interval of 110 msec due to safety window pacing (S). A = atrial stimulus not coinciding with a QRS complex.

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having timed out the above mentioned sensed AV interval of 180 msec) and the next and subsequent sinus P waves occur during the PVARP, preventing synchronization of ventricular stimuli to the P waves in the PVARP. Activity-based V-A intervals are timed out from each conducted QRS complex. Ultimately these activity-based atrial stimuli (which are timed out from the sensed conducted QRS complexes) may occur simultaneously with the intrinsic R wave, which belongs to the spontaneous P wave that was not sensed because it occurred in the PVARP.
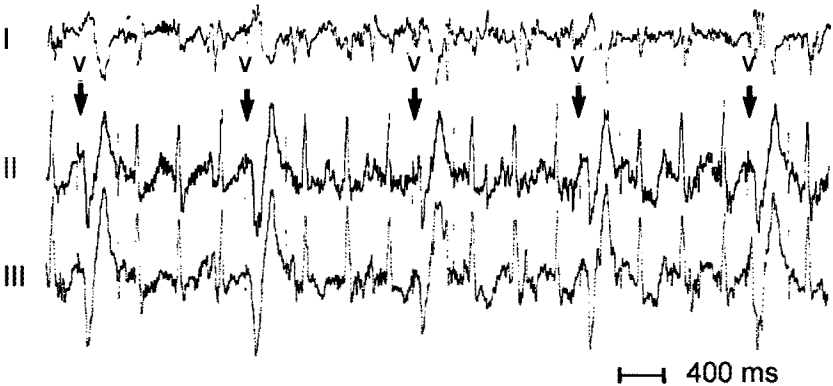
Fig. 6.33. Simultaneously recorded 3-channel electrocardiogram near the end of the exercise test demonstrating repetetive ventricular capture of the ventricular pace output (V) (due to undersensing of the spontaneously conducted QRS complex occurring in the ventricular blanking period). The situation is the same as in figure 6.32 except for the fact that the refractory period of the ventricular myocardium has shortened to such an extent that there is capture even with a paced AV interval of 200 msec.
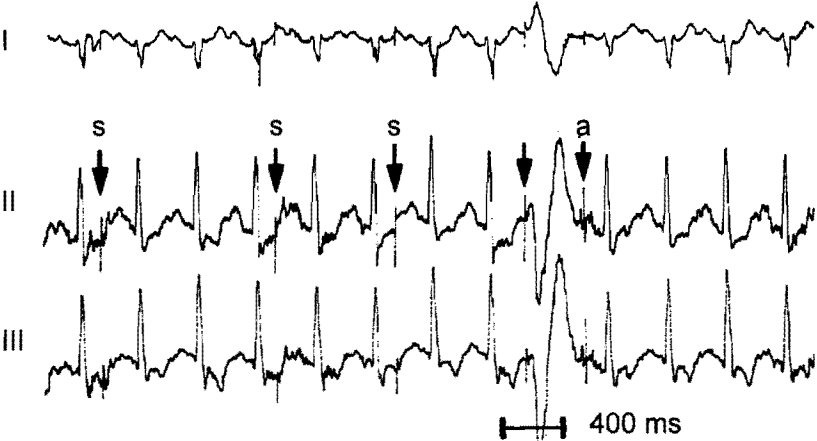
Fig. 6.34. Simultaneously recorded 3-channel ECG direct following the exercise test. The arrows labeled S indicate the occurence of safety window pacing. The isolated arrow indicates ventricular pacing with a paced atrioventricular interval of 200 msec. The arrow labeled with an A indicates the atrial stimulus artifact. The A-A interval is 550 msec.

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During atrial stimulation the ventricular channel is blanked for incoming signals and the intrinsic R wave may not be sensed. As a result a normal AV delay is timed out and a ventricular spike is produced after completion of the AV delay.

This resulted in frequent T wave pacing with paced AV intervals of 200 msec (programmed; Figs. 6.32-6.35). If the intrinsic R wave occurs within 110 msec of an atrial stimulus the paced AV interval is shortened to 110 msec due to the feature known as SWP.Figure 6.32 illustrates T wave pacing without ventricular capture at the onset of exercise after occurence of the VPB as shown in Figures 6.30 and 6.31. Near the end of the exercise test the refractory period of the ventricular myocardium had shortened sufficiently to enable capture of the ventricle with a paced AV interval of 200 msec (Figs. 6.33-6.35).

Figures 6.34 and 6.35 shows the situation immediately after exercise. Because of the rapid decrease of the activity determined rate, T wave pacing disappeared in the first 30 seconds of rest.
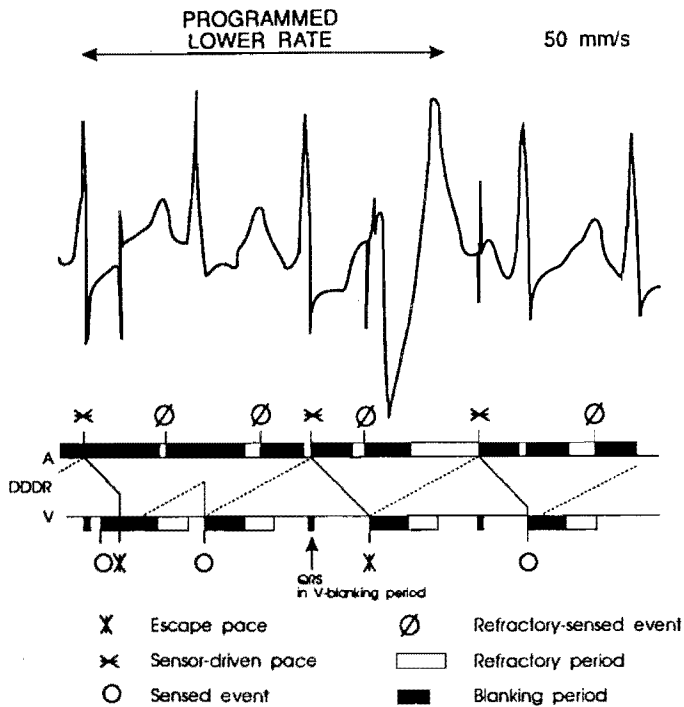
Fig. 6.35. Timing diagram explaining the pacemaker behavior in Figure 6.33.

Discussion

Long paced AV intervals are often programmed to prolong pulse generator life (by avoiding the ventricular pace output) and to enable a more physiological and hemodynamically superior activation sequence. As is demonstrated by this case report, programming a long paced AV interval ([3] 200msec) can be potentially dangerous when using a dual chamber rate responsive

mode. As shown in this case report, T wave pacing occurred frequently during the exercise test. This is especially relevant for patients with coronary artery disease, when during exercise and ischemia an inappropriate ventricular pace in the T wave could be lethal [2].

T wave pacing as seen in this case report was initiated by apparent P wave undersensing (Figs. 6.34 and 6.35). Each P wave just occurred within the 300 msec PVARP so that ventricular stimuli were not synchronized. This was induced after a VPB, as shown in figures 6.30 and 6.31, because the rate adaptive AV interval of 65 msec was extended back to the sensed AV interval of 180 msec, which permittent later initiation in the QRS complex of the next PVARP. Apparent P wave undersensing has been described previously and is initiated by extension of the PVARP by a VPB [3-5], artificial ventricular sensing,[6] QRS sensing of the conducted complex in the relative refractory period of the pacemaker due to intermittent noncapture [6], induction of pacemaker Wenckebach block [7], or by later initiation of the PVARP, as in this case.

In the Elite II pacemaker a VPB is defined as the second of two consecutive ventricular events without an intervening P wave. Such a VPB automatically starts a PVARP of 400 msec or of the programmed duration (if the value is >400 msec). The Elite II pacemaker has a programmable atrial blanking period during which the atrial sense amplifier is disabled. When sensing takes place during the remainder of the PVARP, the sensed atrial event is marked as an atrial refractory sensed event. If an atrial refractory sensed event conducts, the resultant R wave is not detected as a pacemaker defined VPB, so that the conducted QRS does not extend the PVARP to 400 msec, as was possible in previous models. This modification reduces the incidence and extent of apparent P wave undersensing. This PVARP extension is, however, still possible in several dual chamber rate responsive devices from other manufacturers.

SWP offers protection because the ventricular pace output is delivered during the absolute refractory period of the ventricular myocardium. This protection is, however, not offered if the QRS occurs during the ventricular blanking period initiated by an atrial pace and the only way to ensure safety is to program short paced AV intervals. In this patient the refractory period of the ventricular myocardium shortened to 200 msec, as demonstrated by ventricular capture at the end of the exercise test (Figs. 6.33-6.35), so that a paced AV interval of 200 msec was too long to be safe. In this patient the pacemaker settings, as well as the rate responsive mode, were programmed according to protocol to evaluate the response of the activity sensor during an exercise test. Because the patient had a normal sinus node function there was no need to program the rate reponsive mode on, and the device was programmed back into the DDD mode with a paced AV interval of 160 msec.Programming a long paced AV delay can potentially give rise to several other problems:

1. Separating atrial and ventricular events sufficiently could allow retrograde conduction of paced ventricular events, which can give rise to pacemaker circus movement tachycardia [8] if the retrograde P wave falls outside the PVARP or pacemaker syndrome if the retrograde P wave falls inside the PVARP.

2. Programming a long AV interval results in a long total atrial refractory period (AV interval+PVARP), which reduces the 2:1 AV block point and limits the atrial tracking capability of the device.

3. In the absence of spontaneous activity, the atrial sensing window starts at the end of the PVARP and ends when the V-A interval has been timed out with an atrial pace output. The atrial sensing window is enlarged by shortening the PAVRP and/or a paced AV interval. During the atrial sensing window a sensed P wave inhibits an atrial output in

the DDI(R) mode and synchronizes a ventricular pace output after the programmed AV interval in the DDD(R) mode. During excercise (in the DDDR and DDIR mode) the atrial sensing window becomes smaller until the device functions in the DVIR mode when the total atrial refractory period equals the sensor driving pacing cycle length. This can give rise to pseudo atrial malpacing as described by van Gelder et al.[9] when an atrial stimulus does not capture the atrium because the atrial tissue is still refractory from a retrogradely conducted ventricular paced\event. In this patient the PVARP+paced AV interval equals 300+200 =500 msec, which means that the device functions in the DVI mode at a sensor driven rate of 120 beats/min. In addition, a sensor determined atrial escape stimulus could induce atrial fibrillation or flutter while the device is functioning in the DVIR mode because spontaneous atrial activity will not inhibit an atrial escape stimulus. If an atrial event occurs in the last part of the PVARP this will also not inhibit a sensor determined atrial escape stimulus, so that induction of atrial fibrillation or flutter could occur at even lower sensor driven rates. In our patient, with the previously mentioned settings, this could already occur at sensor driven rates of about 85 beats/min. The risk of these problems can be reduced by enlarging the atrial sensing window by programming shorter PVARPs (if possible) and shorter paced AV intervals.

4.   In dual chamber pacemakers with ventricular-based timing, a situation can arise in which a QRS complex occurring after an atrial pace output resets the ventricular escape interval and can unexpectedly give rise to a pacemaker related tachycardia in which the atrial paced rate is higher than the programmed (or sensor driven) lower rate [10]. In the Elite II pacemaker this is no longer possible because the timing of the V-A escape interval is only started after the programmed AV interval has been timed out [1], thus preventing premature resetting of the V-A escape interval by a sensed ventricular event. (Not unique to Elite II).

Conclusion

In spite of the advantages of prolonging battery life and allowing a more physiological and hemodynamically superior natural activation sequence, the programming of a long paced AV interval must be done with caution. T wave pacing, as described here, can occur during dual chamber rate reponsive pacing in the presence of spontaneous AV conduction and can be induced by the mechanisms that induce apparent or actual P wave undersensing.

Sensor driven pacing enables frequent occurrence of T wave pacing under these circumstances.

In this case report, conducted QRS complexes occurred during the 20-msec blanking period and were, therefore, not sensed. This gave rise to T wave pacing. This behavior is particularly dangerous in the presence of ischemia and can be avoided by programming a sufficiently short paced AV interval or not programming the rate responsive mode if not needed. T wave pacing can also occur during DDD pacing [11], but it is not likely to occur as often and as repetitively as described here. In patients with sick sinus syndrome and intact AV conduction the AAI(R) mode would be better. In patients with isolated AV conduction disturbances the DDD mode is preferred.

References

1. Technical Manual for the Elite II Activity Responsive Dual Chamber Pacemaker models 7084/7085/7086. Medtronic, Inc., Minneapolis, MN, 1992.

2. Bilitch M, Cosby RS. Ventricular fibrillation and competitive pacing. N Engl J Med 1967; 11: 598-604.

3. Satler LF, Rackley CE, Pearle DL, et al. Inhibition of a physiologic pacing system due to its anti-pacemaker-mediated tachycardia mode. PACE 1985; 8: 806-810.

4. Wilson JH, Lattner S. Undersensing of P waves in the presence of an adequate P wave due to automatic postventricular atrial refractory period extension. PACE 1989; 12: 1729-1732.

5. Den Dulk K, Wellens HJJ. Failure of the postventricular premature DVI mode in preventing pacemaker circus movement tachycardia. Am J Cardiol 1984; 54: 1371-1372.

6. Greenspon AJ, Volosin KJ."Pseudo" loss of atrial sensing by a DDD pacemaker. PACE 1987; 10: 943-948.

7. Van Gelder LM, Van Mechelen R, Den Dulk K, et al. Apparent P wave undersensing in a DDD pacemaker post exercise. PACE 1992; 15: 1651-1656.

8. Den Dulk K, Lindemans FW, Wellens HJJ. Noninvasive evaluation of pacemaker circus movement tachycardias. Am J Cardiol 1984; 53: 537-543.

9. Van Gelder LM, El Gamal MIH. Ventriculoatrial conduction: A cause of atrial malpacing in AV universal pacemakers. A report of two cases. PACE 1985; 8: 140-143.

10. Den Dulk K, Lindemans FW, Bar F, et al. Pacemaker related tachycardias. PACE 1982; 5: 476-485.

11. Lamas GA, Antman EM. Pacemaker-mediated tachycardia initiated by by coincident P-wave undersensing and ventricular blanking period. PACE 1985; 8: 436-439.

6.8 Atrial depolarization not followed by ventricular stimulation (atrial undersensing)

Every atrial depolarization occurring outside the atrial refractory period should evoke a ventricular output stimulus, provided there is no ventricular sense event during the AV interval.
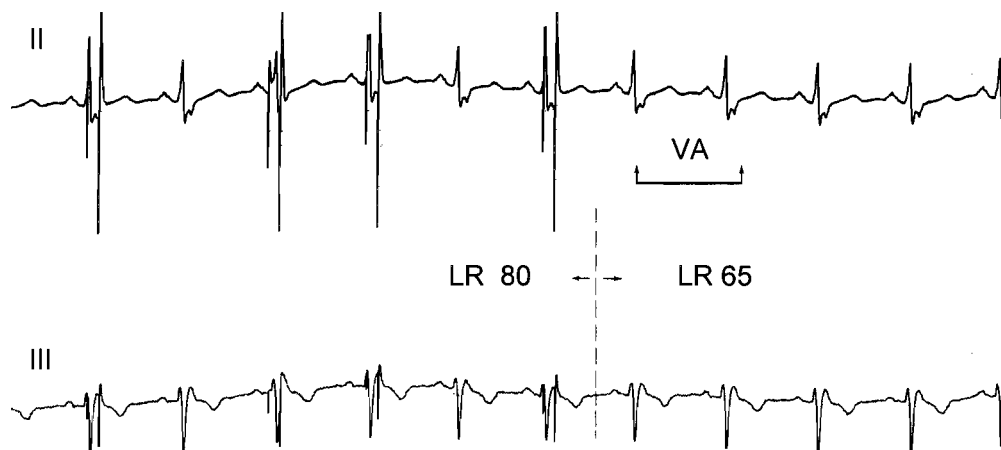
Fig. 6.36. Two channel ECG (lead II,III) of a patient with an implanted DDD pacemaker. The atrial sensitivity was programmed to 2.8 mV, inducing atrial undersensing, the lower rate to 80 ppm and the AV interval to 180 ms. In the first part of the ECG atrial undersensing is observed and the AV interval is shortened due to ventricular safety pacing. After programming the lower rate to 65 ppm electrocardiographic atrial undersensing has disappeared, because the VA interval is reset by ventricular sensing, whereas atrial undersensing is still present.

One should take into consideration the fact that the functional AV interval is only equal to the programmed AV interval if the atrial rate is lower than or equal to the ventricular upper rate limit. If the atrial rate is higher than the ventricular upper rate, but can still be sensed because P waves fall outside the atrial refractory period, the atrial sense-ventricular pace interval can be longer than the programmed AV interval (see paragraph 6.3.3).

Lead III

Atrial sense 2.8 mV ↑   1.4 mV
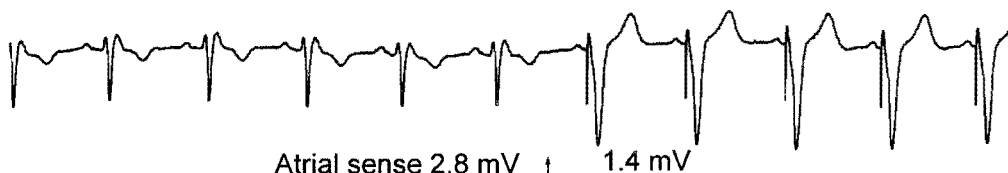
Fig. 6.37. Single channel ECG (lead III), same patient as figure 6.36. The AV interval is shortened to 80 ms, lower rate still 65 ppm. Although in the first part of the recording (atrial sensitivity 2.8 mV) atrial undersensing was present, it can easily be overlooked by the absence of atrial stimuli (explanation see figure 6.36). Restoring atrial sensing by programming the atrial sensitivity to 1.4 mV immediately reveals normal atrial sensing and demonstrates atrial undersensing in the first part of the recording.

If under the conditions mentioned above atrial depolarization does not result in a ventricular stimulus, the electrocardiographic pattern should be defined as atrial undersensing. Atrial undersensing will not under always result in an atrial stimulus following an intrinsic atrial depolarization. The latter depends on the relationship between the time of emission of the atrial pulse and, if present, the timing of the intrinsic ventricular depolarization.

Lead II

DDD AVI 150 ms   Ap   Ap
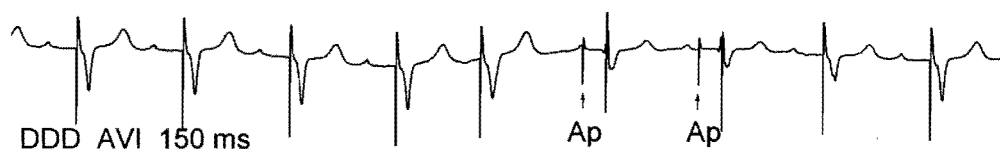
Fig. 6.38. Single channel ECG (lead II) of a patient with an implanted DDD pacemaker programmed at a lower rate of 60 ppm and an AV interval of 150 ms. The interval between the onset of the P wave and the subsequent ventricular paced beat is approximately 320 ms. In the middle of the recording two consecutive atrial (Ap) and ventricular pace pulses are observed. The second atrial stimulus suggests atrial undersensing. See also figures 6.39, 6.40.

If the intrinsic ventricular depolarization takes place before the preceding VA interval has been timed out, the lower rate timing will be reset and no atrial output pulse will be emitted, in spite of the fact that the preceding P wave is not sensed and the atrial output pulse is not inhibited by the intrinsic P wave. This implies that in the presence of sinus rhythm with a RR interval shorter than the VA interval, and a PR interval shorter than the AV interval of the pacemaker, atrial

undersensing cannot be concluded from the ECG (Fig. 6.36). Therefore in order to prove efficient atrial sensing, the AV interval should be programmed shorter than the interval between atrial sensing and ventricular sensing, which generally corresponds to the intrinsic PR interval (Fig. 6.37). However, atrial sensing can be delayed in patients where the atrial electrode is not positioned in the right atrial appendage or high right atrium.

Lead II
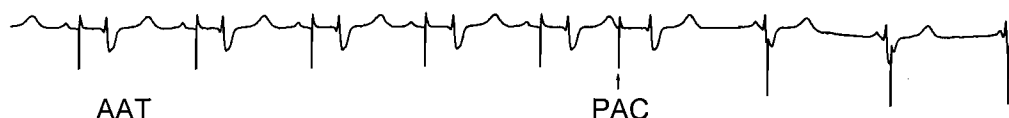
Fig. 6.39. Single channel ECG (lead II) of the same patient as in figure 6.38. The pulse generator is now programmed in the AAT mode at a rate of 50 ppm. Pacemaker stimuli indicate that atrial sensing occurs approximately 170 ms after the onset of the P wave. "Early" emission of an atrial stimulus (arrow) is caused by a premature atrial contraction (PAC). This recording illustrates that the ECG in figure 6.38 showed normal pacemaker function with late sensing of the intrinsic P wave. Late sensing is not only explained by the position of the atrial lead but by the presence of intra-atrial conduction disturbance.

Lead II
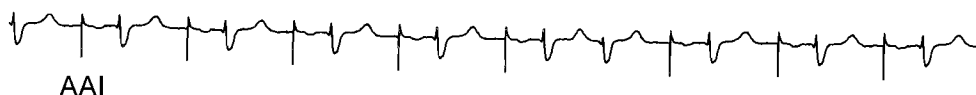
Fig. 6.40. Single channel ECG (lead II) of the same patient as the two previous ECGs. The pulse generator is now programmed in the AAI mode at a rate of 65 ppm. There is effective atrial stimulation with a delay between the atrial stimulus and the recorded atrial depolarization. The stimulated P waves have a negative deflection in lead II and III, indicating low atrial stimulation, which was confirmed by the position of the epicardial electrode.

This is illustrated in the ECG of figure 6.38. The unipolar DDD pacemaker is programmed at an AV interval of 150 ms. The interval between the onset of the P wave and the triggered ventricular pace response is approximately 320 ms. In the middle of the recording there are two atrial stimuli. The first atrial stimulus looks like atrial fusion, while the second gives an impression of atrial undersensing. The atrial stimulus is delivered approximately 140 ms after the onset of the P wave. The prolonged interval between P wave and ventricular stimulation, however, indicates that the last portion of the P wave, which has no visible deflection in the ECG, is sensed by the pacemaker. This is confirmed by programming the pulse generator to the AAT mode (Fig. 6.39). In the AAT mode the pacemaker pulse is indicating the time of atrial sensing, which is approximately 150 ms after the onset of the P wave. This also confirms that the atrial stimuli in ECG figure 6.38 are not indicating atrial undersensing, but normal pacemaker function. When the pulse generator was programmed to the AAI mode (Fig. 6.40) effective atrial stimulation was observed. The negative deflection of the paced P waves in this ECG (leads II,III)

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demonstrates that the atrium was stimulated from the low right atrium, which was explained by the position of the epicardial atrial lead. It is most likely that late sensing of the P wave is not only related to the position of the atrial electrode but also to intra atrial conduction disturbance [27,28].

6.8.1 Causes of atrial undersensing

As already explained in chapter V 5.13.1 the causes of undersensing can be categorized into three groups, when assessing sensing in the atrial channel separately:

1. The intrinsic intracardiac electrogram is of sufficient amplitude to be sensed by the pacemaker but occurs in the refractory period of the pacing system.

2. The intrinsic intracardiac electrogram is not of sufficient amplitude and/or slew rate to be sensed by the pacemaker at the programmed sensitivity level.

3. The intrinsic intracardiac electrogram is of sufficient amplitude and or slew rate to be sensed by the pacemaker but the signal is attenuated by electrical dysfunction of the lead or the pulse generator.

Atrial sense events during the relative part of PVARP will prolong PVARP similar to the mechanism described in chapter V 5.12. In the presence of continuous interference of sufficient intensity, the refractory period will continuously be reset, which ultimately results in asynchronous pacing in the interference or noise mode. Because interference can be detected in the atrial, the ventricular and in both channels, interference behavior will be explained in paragraph 6.13.

Battery depletion and extraneous interference (e.g. DC shock, electrocautery) may cause reversion of the pacing system to the VVI mode semi-permanently, thereby showing apparent atrial undersensing [29]. Cold exposure during shipment has also been reported as a cause of reversion from DDD to VVI mode [30]. These examples of pacing mode reversion, that not recover spontaneously, can be corrected by reprogramming the pulse generator.

Magnet application resulting in asynchronous pacing (DOO), thus presenting electrocardiographically undersensing in the presence of intrinsic atrial and/or ventricular activity, should not be considered as undersensing.

The explanation for the mechanism of undersensing in single chamber systems described in chapter V 5.11-5.14.3 can be applied to the atrial channel of a dual chamber system.

6.9 Mechanisms of atrial undersensing related to dual chamber systems

As explained in the previous paragraph assessment of atrial undersensing may be performed separately for the atrial channel. In dual chamber systems, however, sensing properties of the atrial channel are not only determined by the specifications of the atrial channel but can also be affected by sense events in the ventricular channel.

6.9.1 Postventricular atrial refractory period extension

In the majority of the currently available pacemakers a protection mechanism against pacemaker mediated tachycardia (see chapter VII 3.1.2) is incorporated [31,32]. These pacing systems were designed to prolong the atrial refractory period only at times when retrograde VA conduction would be likely to occur, such as following a premature ventricular contraction (PVC). Automatic extension of the atrial refractory period following a PVC protects against long retrograde VA conduction times while additionally allowing P wave synchronous pacing at high

atrial rates. The pacemaker defines a PVC as the second of two ventricular events without an intervening paced or sensed atrial event. If a pacemaker determined PVC is recorded, the postventricular atrial refractory period is extended with a programmable interval or a fixed interval in order to prevent sensing of retrograde P waves. Prolongation of PVARP, however, not only prevents sensing of retrograde P waves, but also can give rise to undersensing normal P waves [33,34]. Ignoring normal P waves under these circumstances depends on the coupling interval between P wave and the preceding PVC and the degree of PVARP extension. If the normal P wave following a PVC is ignored by extension of the PVARP and this P wave is conducted to the ventricle, the QRS complex will also be interpreted as a PVC, again resulting in extension of the PVARP. This phenomenon can lead to an asymmetrical behavior of atrial sensing during acceleration and deceleration of the atrial rate, as explained in paragraph 6.4.2 of this chapter.

6.9.2 Effect of ventricular (over)sensing on atrial sensing in dual chamber systems during the VA interval

The timing diagram in paragraph 6.1 of this chapter shows that a ventricular sense or pace event temporarily disables the atrial channel by initiation of the postventricular atrial refractory period.
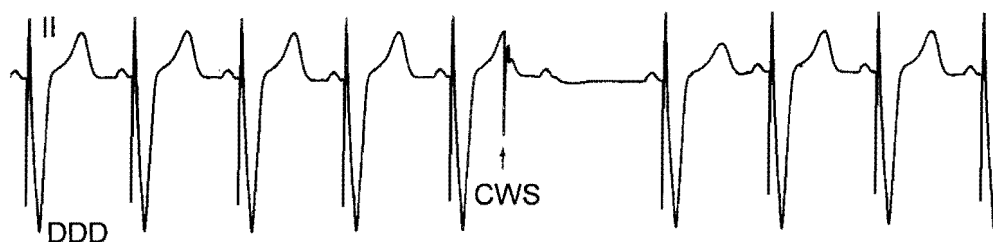
Fig. 6.41. Single channel ECG (lead II) of a patient with an implanted DDD pacemaker programmed at a lower rate of 40 ppm. The atrial channel was programmed in the bipolar configuration and a sensitivity of 3.0 mV; the ventricular channel in the unipolar configuration with a sensitivity of 1.0 mV. These settings were chosenso that a timed chest wall stimulus (CWS) could be applied just outside PVARP, which was only sensed by the ventricular channel and not by the atrial channel, thus imitating ventricular oversensing. Due to ventricular oversensing induced by the chest wall stimulus, electrocardiographic P wave undersensing is recorded. Ventricular oversensing was confirmed by the marker channel

However, it is not only the real pace and sense events that initiate PVARP but every false signal detected by the ventricular channel. This means that every possible cause of ventricular oversensing initiates a PVARP and in the majority of cases it is not only initation of PVARP, but this PVARP will also be extended, due to the fact that the event causing ventricular oversensing will be interpreted by the pacemaker as a PVC. Because an important proportion of the signals responsible for false ventricular inhibition (and thus initiation of PVARP) are invisible on the normal ECG, PVARP can be initated unknown to the interpreter. An intrinsic P wave following ventricular oversensing is often ignored by the pacemaker due to the initiation of the (extended) PVARP [35]. The patient will present with electrocardiographical atrial undersensing, while the cause of atrial undersensing is ventricular oversensing. When atrial undersensing is present in the

ECG of a patient with a dual chamber pacemaker, ventricular oversensing should be excluded as the cause, by recording of the marker channel and/or the intracardiac electrogram, if these options are available in the pacing system (see chapter I, Fig. 1.5). If none of the options are available in the pacing system and atrial undersensing is observed one can consider programming the pacing system to the VVI mode. If in the VVI mode prolongation of the escape interval is recorded, ventricular oversensing is the most likely explanation of atrial undersensing. The ECG presented in figure 6.41 illustrates ventricular oversensing by a timed chest wall stimulus. The atrial channel is programmed in the bipolar configuration at a sensitivity of 3.0 mV, while the ventricular channel was programmed in the unipolar configuration with a sensitivity of 1.0 mV. This setting was choosen in order to apply a chest wall stimulus that was only sensed by the ventricular channel. According to the marker channel an extended PVARP was initiated by the chest wall stimulus thus resulting in undersensing of the next P wave.

6.10 Premature ventricular pacing without preceding atrial depolarization (atrial oversensing)

If in a DDD pacemaker a ventricular stimulus is emitted without a preceding atrial depolarization and the interval between the preceding ventricular paced or sensed event is shorter than the lower rate interval it is electrocardiographically determined as atrial oversensing.

Fig. 6.42. Three channel ECG (lead I,II,III) of a patient with an implanted DDD pacemaker programmed at a lower rate of 60 ppm, an AV interval of 250 ms and a ventricular upper rate limit of 125 ppm. The ECG shows effective ventricular stimuli with a short coupling interval to the preceding QRS complex. Atrial oversensing? See also figure 6.43.

If the coupling interval between the ventricular stimulus and the preceding ventricular event is equal to the lower rate without an intervening paced atrial event, this should be classified as atrial no output.
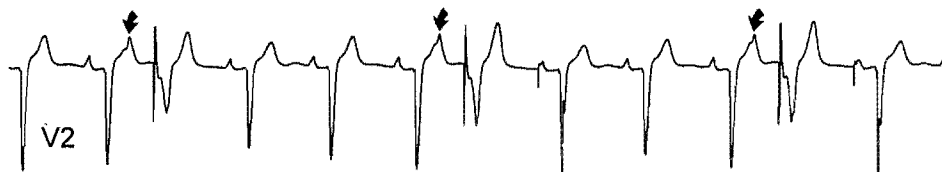
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Fig. 6.43. Single channel ECG (lead V2) of the same patient as figure 6.42. In the precordial lead premature atrial contractions (arrows) are recorded, thus confirming normal pacemaker function.

Before atrial oversensing can be concluded from the ECG, the interpreter should be sure, that no atrial depolarization is preceding the ventricular stimulus. Sometimes even a multichannel ECG can be misleading in this respect, as illustrated in figure 6.42. In this patient with an implanted unipolar DDD pacemaker the three channel ECG shows premature ventricular stimulation without a clearly visible atrial depolarization. However, recording of a V2 precordial lead (Fig. 6.43) illustrates a premature atrial contraction superimposed on the preceding T wave. So it must be concluded from the latter ECG that there is normal pacemaker function and not atrial oversensing.

Atrial oversensing resulting in ventricular stimulation is sometimes indicated in the ECG by the occurrence of retrograde P waves, because the ventricular stimulus is not preceded by atrial depolarization. The presence of retrograde P waves after ventricular stimulation is, however, not unequivocal proof of the presence of atrial oversensing. E.g. a premature atrial contraction can be sensed by the atrial channel, resulting in ventricular stimulation. If the timing of the atrial premature contraction in combination with the programmed upper rate and AV interval gives sufficient separation of the atrial depolarization and the ventricular pace event, this can also lead to retrograde conduction and pacemaker circus movement tachycardia (chapter VII 7.3).

The causes of atrial oversensing in dual chamber systems are identical to those in single chamber systems, that have been already discussed in chapter V 5.3 - 5.8.3. The only additional cause is sensing of the far-field ventricular afterpotentials or the last portion of the paced ventricular electrogram by the atrial electrode [36].

6.11 Ventricular oversensing in dual chamber pacemakers

Because the reaction on ventricular oversensing in dual chamber systems is strongly dependent on the timing of oversensing during the timing cyclus it is not possible to give a concise formulation of its electrocardiographic presentation. On examining the intervals of the timing diagram, different reactions of the pacing system to sensed signals in the ventricular channel are observed.

1. The ventricular blanking period

Because the ventricular amplifier is completely disabled during the blanking period false signals will have no effect on pacemaker function and timing.

2. The ventricular safety pace interval

A false signal sensed by the ventricular channel during the ventricular safety pace interval will evoke a ventricular stimulus at the end of this interval. So when no intrinsic ventricular depolarization is present ventricular safety pacing (shortening of the AV interval) indicates ventricular oversensing. If this is the only observation and it is not associated with other signs of

ventricular oversensing like prolongation of the VA or VV interval, crosstalk is the most likely explanation for this phenomenon (see paragraph 6.5.2.2).

3. The ventricular sensing inhibiting period of the AV interval

If ventricular oversensing is present during this period the ventricular stimulus will be inhibited. Depending upon the timing system of the pulse generator (atrial or ventricular based timing), the interval between the preceding atrial event and the following atrial paced event is equal to the lower rate interval in atrial based timing systems, and shorter than the lower rate interval in ventricular based timing systems. It is assumed that no intervening atrial and ventricular sense events are present. In the presence of intact AV conduction, where the AV conduction time is longer than the programmed AV interval, ventricular oversensing in this period can be easily overlooked. Absence of AV conduction results in atrial pacing only, which is a life threatening situation in pacemaker dependent patients, see also figure 6.22.

4. The ventricular absolute refractory period

False signals occurring in the ventricular channel during this period will have no effect on pacemaker function. Pacemaker timing will not be affected.

Lead III   CWS
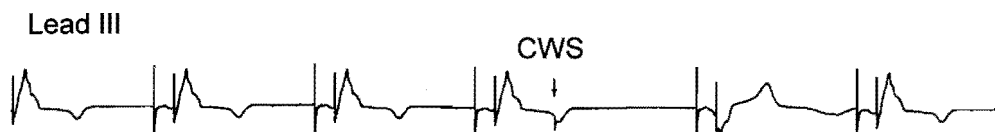
Fig. 6.44. Single channel ECG (lead III) of a patient with an implanted DDD pacemaker. One programmed chest wall stimulus (CWS), outside the ventricular refractory period, was applied to imitate ventricular oversensing. Because no intrinsic events (atrial and ventricular) followed ventricular oversensing normal AV sequential pacing is restored with a prolonged VA interval. Note that the first ventricular stimulus after the CWS gives rise to ventricular fusion.

Lead III   CWS
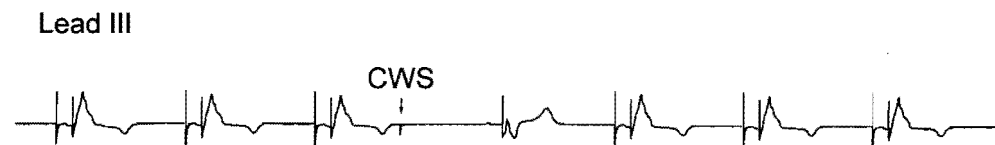
Fig. 6.45. Single channel ECG (lead III) of a patient with an implanted DDD pacemaker. One programmed chest wall stimulus (CWS), outside the ventricular refractory period, was applied to imitate ventricular oversensing. After oversensing, the following P wave triggers a ventricular stimulus, restoring normal pacemaker function with prolongation of the VV interval.

5. The ventricular relative refractory period

A false signal detected in the ventricular channel during the ventricular relative refractory period prolongs the ventricular refractory period and restarts the PVARP or even an extended PVARP. Sensing during the ventricular relative refractory period does not affect the lower rate timer but by the restart of the (extended) PVARP and prolongation of the ventricular refractory period, the ECG can represent atrial undersensing as well as ventricular undersensing as a manifestation of ventricular oversensing.

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6. The ventricular sensing inhibiting period of the VA interval

In practice this is the VA interval minus the ventricular refractory period. Ventricular oversensing during this interval initiates a ventricular refractory period and a PVARP or even an extended PVARP. If no intrinsic atrial and ventricular events occur after ventricular oversensing, atrial and ventricular pacing will follow with prolongation of the VA interval (Fig. 6.44).
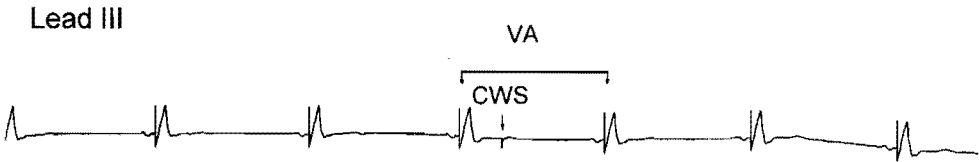
Lead III

Fig. 6.46. Single channel ECG (lead III) of a patient with an implanted DDD pacemaker. One programmed chest wall stimulus (CWS), outside the ventricular refractory period, was applied to imitate ventricular oversensing. Because the P wave, following the chest wall stimulus, occurred before the VA interval was timed out completely, pacemaker function is apparently not affected by ventricular oversensing.

Effect of Ventricular Oversensing
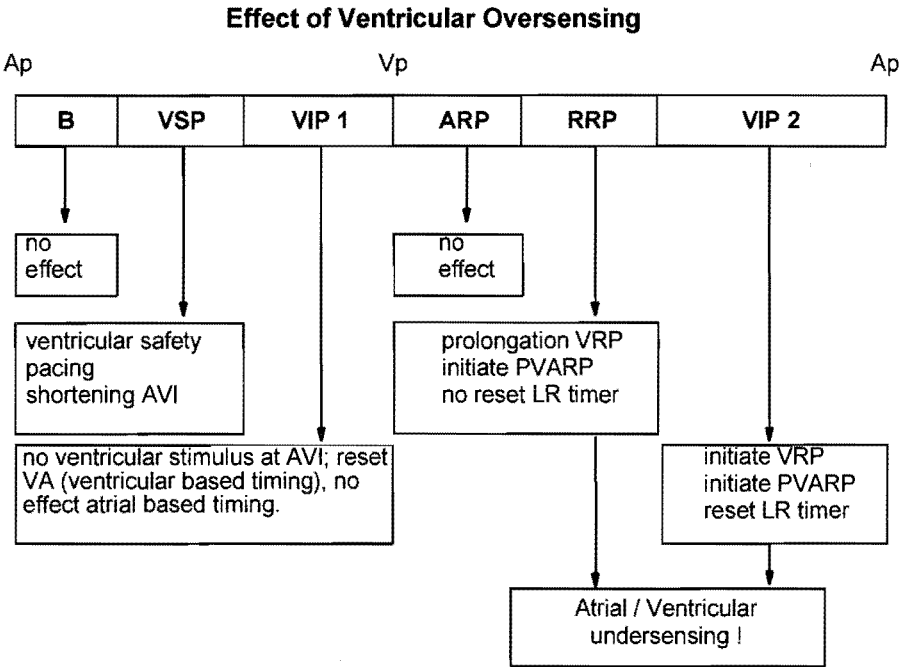
Fig. 6.47. Diagram showing the intervals of the timing cycle and the result of oversensing during these intervals. Ap= Atrial pacing, Vp= Ventricular pacing, B= blanking (ventricular), VSP= ventricular safety pacing, VIP1= Ventricular inhibiting period (during AV interval), ARP= Absolute refractory period (Ventricular), RRP= Relative refractory period (ventricular), VIP2= Ventricular inhibiting period (during VA interval), AVI= AV interval, LR timer= Lower rate timer.

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If intrinsic atrial and/or ventricular intrinsic events occur during respectively PVARP and the ventricular refractory period initiated by the false signal, atrial and/or ventricular undersensing can be observed. Atrial and/or ventricular intrinsic events occurring after the refractory periods initiated by ventricular oversensing will restore normal pacemaker function, but a VV or VA interval longer than the programmed values can be observed (Fig. 6.45). If however, an intrinsic atrial event following ventricular oversensing occurs before the VA interval, initiated by the ventricular event preceding ventricular oversensing is elapsed, no pacemaker malfunction is observed, so ventricular oversensing can be easily overlooked under these conditions (Fig. 6.46). The diagram represented in figure 6.47 illustrates the intervals of the timing cycle and the response to ventricular oversensing.

6.11.1 Causes of ventricular oversensing
The causes of ventricular oversensing in a dual chamber system are identical to those of single chamber systems mentioned in chapter V. The only exception is oversensing of the atrial stimulus, resulting in crosstalk. Crosstalk has been previously explained in paragraph 6.5 of this chapter. When oversensing is observed in a dual chamber system the most practical approach is to programme the pacing system to the VVI mode and to analyze the systems as has been described for single chamber systems showing prolongation of the escape interval.

6.12 Ventricular undersensing in dual chamber systems
In dual chamber systems the electrocardiographic presenstation of ventricular undersensing depends on the timing of the intrinsic ventricular event in the timing cycle of the pacing system. Undersensing can occur during the AV interval and during the alert section of the VA interval. The alert section of the VA interval is the remaining part of the VA interval after the elapse of the ventricular refractory period. The timing of the intrinsic ventricular activity that is ignored by the pacing system (during the AV interval or the VA interval) determines whether undersensing is characterized by a shortening of the coupling interval similar to single chamber systems.

6.12.1 Ventricular undersensing during the AV interval
In order to determine ventricular sensing during the AV interval one should differentiate between an AV interval initiated by atrial pacing and an AV interval initiated by atrial sensing.

6.12.1.1 AV interval initiated by an atrial paced event
When an AV interval is initiated by atrial pacing one has to take into account the fact that atrial stimulation is followed by the ventricular blanking period. Intrinsic ventricular events occurring during the blanking period are ignored by the pacing system as explained in paragraph 6.6 of this chapter. Under these conditions a ventricular stimulus will be emitted at the programmed AV interval. Although the electrocardiographic appearance is comparable to that of ventricular undersensing it should not be considered as such [37]. Similarly intrinsic ventricular events occurring during the ventricular safety pacing interval resulting in a ventricular stimulus just after or in the intrinsic QRS complex should not be considered as undersensing.
If undersensing of an intrinsic ventricular event occurs during the alert period of the AV interval a ventricular stimulus will be emitted during or just after the QRS complex leading to a pattern of ventricular fusion or pseudofusion. Undersensing under these circumstances is not always easy to recognize and depends on the timing of the intrinsic QRS complex and the programmed AV

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delay. If ventricular fusion or pseudofusion is present on the ECG, effective ventricular sensing should be proven by prolongation of the AV interval in order to evaluate whether the ventricular stimulus is inhibited. An even better method is to programme the device in the VVT mode at the same level of ventricular sensitivity. Ventricular sensing and the area of the QRS complex sensed is now indicated by ventricular stimuli in the QRS complex.

6.12.2.2 AV interval initiated by an atrial sensed event

If ventricular undersensing occurs during the AV interval following an intrinsic atrial depolarization, a ventricular stimulus will be emitted during or just after the QRS complex, leading to a pattern of ventricular fusion or pseudofusion (Fig. 6.48). Only when the ventricular stimulus is emitted after intrinsic ventricular depolarization is completely accomplished, can ventricular undersensing be concluded from the ECG. It is clear that in patients with absence of AV conduction ventricular sensing during the AV interval cannot be evaluated.

As stated in the previous paragraph, the best proof of adequate ventricular sensing is programming the device into the VVT mode. It not only indicates whether sensing of the device is functioning properly but it also shows which portion of the QRS complex is sensed.
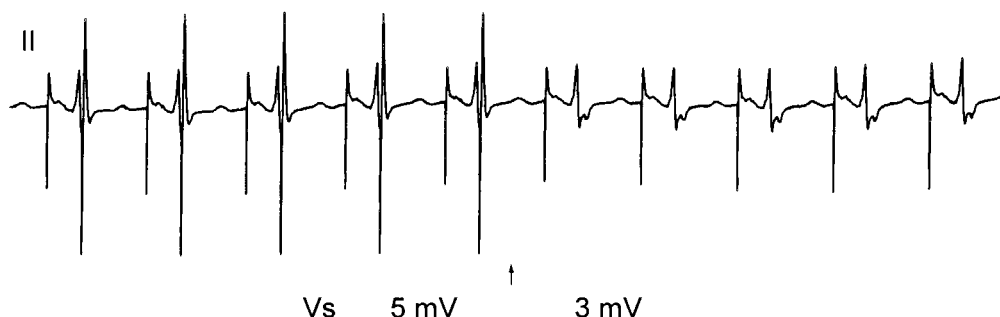
Fig. 6.48. Single channel ECG (lead II) of a patient with an implanted DDD pacemaker. In the first sectiont (ventricular sensitivity 5 mV) the ventricular stimulus is inscribed in the QRS complex showing a pseudofusion pattern. After programming ventricular sensitivity to 3 mV, there is inhibition of the ventricular stimulus, indicating ventricular undersensing in the first section of the ECG recording.

When late ventricular sensing occurs e.g. in the presence of right bundle branch block resulting in ventricular pseudofusion beats, it should not be interpreted as ventricular undersensing.

6.12.2 Ventricular undersensing during the VA interval

In order to determine ventricular sensing during the VA interval one should differentiate between a VA interval terminated by an atrial stimulus (completely elapsed VA interval) and a VA interval prematurely terminated by an intrinsic atrial depolarization (incompletely elapsed VA interval).

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6.12.2.1 Ventricular undersensing during a completely elapsed VA interval

In the case of a completely elapsed VA interval ventricular undersensing is easily recognized in the ECG. The unsensed ventricular event has not reset the VA timer and the interval between the preceding ventricular event and the following atrial paced event is equal to the VA interval of the pacing system. In the presence of this electrocardiographic pattern undersensing can be concluded, but oversensing in the ventricular channel resulting in prolongation of the ventricular refractory period or noise rate reversion should be excluded as the cause of undersensing. Recording of a marker channel, indicating ventricular sense events, will give an immediate answer to this question. If a marker channel is not available in the pacing system programming the VVT mode will give the solution. The pacing rate in VVT mode should be programmed below the intrinsic rate. If ventricular undersensing is present, no pacemaker stimuli will be inscribed in the intrinsic QRS complex and asynchronous ventricular stimulation will be observed. If oversensing is the cause of undersensing, ventricular stimuli causing depolarization will be emitted with a coupling interval shorter than the programmed escape interval.

6.12.2.2 Ventricular undersensing during an incompletely elapsed VA interval

If a VA interval is not completely elapsed because a P wave occurs before the end of the VA interval with an intervening QRS complex between this P wave and the last sensed or paced ventricular event, undersensing of the intervening QRS complex is hard to determine from the ECG. Without recording of a marker channel it can be easily overlooked and considered as normal pacemaker function, provided that the P wave triggers a ventricular output pulse. Under these circumstances the interpreter should be aware of the result of undersensing.

1. Because the intervening QRS complex is not sensed by the pacing system, no (extended) PVARP is initiated. If the coupling interval between the intervening QRS complex and the following P wave is shorter than the programmed (extended) PVARP, the following P wave should be ignored and not result in ventricular stimulation. However, if the P wave is not ignored in spite of the shorter coupling interval, ventricular undersensing is the most likely explanation.

2. If the coupling interval between the intervening QRS complex and the following ventricular stimulus, triggered by P wave sensing is shorter than the programmed upper rate interval, ventricular undersensing can be concluded.

If one or both of the above described phenomena are observed electrocardiographic ventricular undersensing is present, but oversensing still has to be excluded as the primary cause, as explained in the previous paragraph. See also chapter VIII 8.7.5.

6.13 Interference mode in dual chamber pacemakers

As explained in chapter V 5.12 all single chamber systems are equipped with a protection mechanism against continuous interference in order to prevent complete inhibition of the pulse generator. The criteria for reversion to the interference mode are dependent on the pulse generator specifications, since there is no uniformity of devices produced different manufacturers. In dual chamber pacemakers where sensing occurs in two channels, a wide variety of criteria exists for reversion to the interference mode. It is not only the criteria that differ, but also the pacing mode initiated by the interference.

Because of the non-uniform criteria as well as the pacing mode induced by the interference, it is not helpful to document all the information concerning the different pulse generators. The ECG interpreter should be informed of the system of interference mode reversion for a specific pulse

generator. Some pulse generators use only one channel for interference detection, while others use both channels. The pacing mode during interference may depend on in which channel interference is detected, and it can alter when interference is detected in one channel or in both. In the majority of pulse generators the interference mode is either VVI or DOO. Both pacing modes have in common the fact that the ECG will represent atrial undersensing, so the interpreter has to consider the possibility of interference mode reversion if atrial undersensing is present.

Although generally continuous interference of sufficient intensity is necessary to initiate the interference mode, some reports describe interference mode reversion due to cardiac signals of sufficient high rate, such as atrial or ventricular arrhythmias [38,39].

6.13.1 Influence of myopotentials on DDD pacemakers

Detection of myopotentials in unipolar dual chamber pacemakers may also induce interference mode reversion [40], but a variety of reactions on myopotentials can be observed [41]. Myopotentials detected by the pacing system can result in:

1. Total inhibition by oversensing in the ventricular channel. Because the ventricular channel is usually less sensitive than the atrial channel, oversensing might be present in the atrial channel also, but due to ventricular oversensing it does not result in ventricular stimulation.

2. Artificial atrial triggering resulting in ventricular stimulation. Myopotentials are sensed in the atrial channel and interpreted as P waves (oversensing in the atrial channel only).

3. Phenomena described under 1 and 2 can be observed alternatingly (Fig. 6.49).

4. Initiation of pacemaker mediated tachycardia due to artificial atrial triggering in the presence of retrograde conduction (see chapter VII 7.3).

5. Shortening of the AV interval as a result of ventricular safety pacing, indicating ventricular oversensing during the ventricular safety pacing interval.
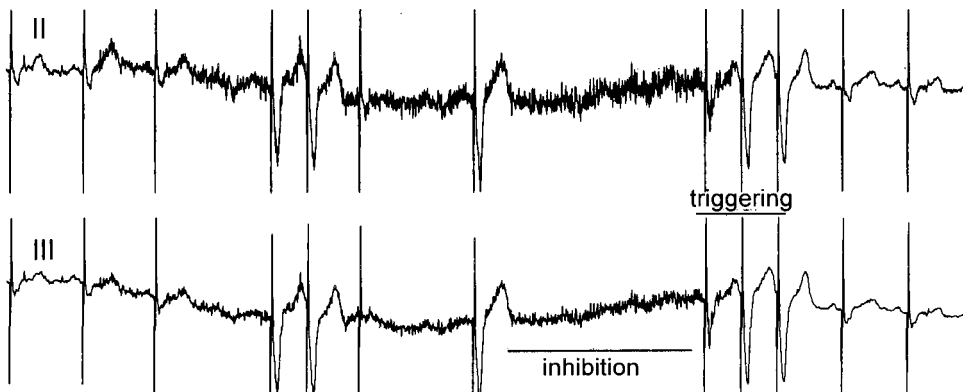
6. Interference mode reversion (DOO pacing).

Fig. 6.49. Two channel ECG (lead II,III) of a patient with an implanted unipolar DDD pacemaker. During contraction of the pectoral muscle adjacent to the pulse generator, alternating periods of artificial triggering (trig.) and total inhibition are recorded.

6.13.2 Interference mode reversion by a bipolar lead verification system

The Omega family of Intermedics pulse generators includes a function that monitors the presence and integrity of a bipolar lead in each channel [42,43]. The process of bipolar lead verification involves a very short duration (1 microsecond), small amplitude (20 mV) subthreshold pulse to the anodal ring of the channel being tested. The energy required to produce this pulse is extremely low and has virtually no effect on the longevity of the pulse generator. The sense amplifier is active immediately after the test pulse. If a bipolar lead is connected and functioning in the programmed configuration, the pulse is delivered to the body tissue where it dissipates immediately. Nothing is detected by the sense amplifier and the pulse generator continues to operate normally in the bipolar configuration. If a bipolar lead is fractured or a high lead impedance is present, the sense amplifier detects the test pulse, interprets it as interference and converts the pulse generator to the interference reversion mode using unipolar stimulation. Depending upon the channel where a high lead impedance indicating lead fracture is detected, the pacing system reverts to VVI or DOO pacing. If lead fracture is detected in the atrial channel the systems reverts to the VVI pacing mode, if lead fracture is detected in the ventricular channel or in both channels, the system reverts to the DOO pacing mode. This is illustrated in the ECGs of figures 6.50-6.51. In this patient an atrial and ventricular unipolar lead were connected to a unipolar/bipolar programmable pulse generator. By programming the atrial channel of the pulse generator to the bipolar electrode configuration the pulse generator detected a high impedance and reverted to the VVI mode (Fig. 6.50).
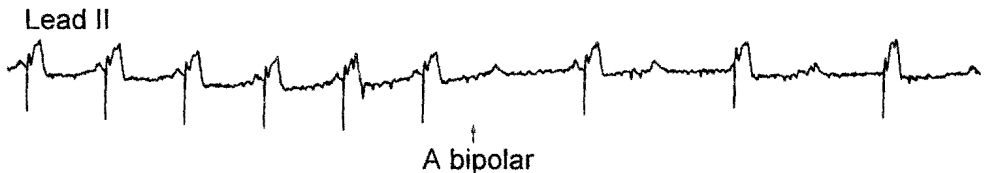
Lead II

A bipolar

Fig. 6.50. Single channel ECG (lead II) of a patient with an implanted DDD pacemaker with programmable electrode configuration. Both leads however were unipolar. The pulse generator is equipped with a bipolar lead verification system. After programming the atrial lead configuration to bipolar, the pacing system detected the absence of a bipolar lead and reverted to VVI pacing.
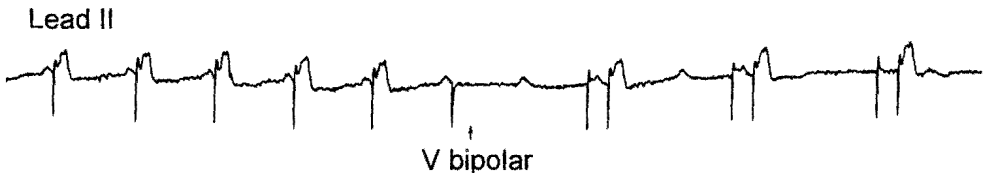
Lead II

V bipolar

Fig. 6.51. Single channel ECG (lead II), same patient as figure 6.50. After programming the ventricular channel to the bipolar configuration the pacing system detected absence of a bipolar ventricular lead and reverted to DOO pacing.

After programming the electrode configuration of the ventricular channel to bipolar DOO pacing was observed (Fig. 6.51). The same was observed after programming the electrode configuration of the atrial and ventricular channel to bipolar.

6.14 Summary and conclusions

Deviation in timing intervals in dual chamber systems can be present on the ECG even in the absence of pacemaker malfunction. In atrial based timing systems, for instance, the effective ventricular rate may be below the programmed lower rate (Fig. 6.3) without ventricular oversensing. In ventricular based systems with continuous atrial stimulation (suggesting lower rate pacing) and normal AV conduction within the programmed AV interval, the stimulation rate is higher than the programmed lower rate (Fig. 6.4).

The interval between atrial sensing and ventricular pacing is not only determined by the programmed AV interval but also by the programmed upper rate interval and PVARP. PVARP extension, initiated after a premature ventricular beat, may even lead to apparent P wave undersensing (paragraph 6.4.2). Accurate knowledge of programmed parameters and specific properties of the pulse generator are necessary to distinguish normal pacemaker function from malfunction.

Ventricular safety pacing and ventricular blanking, both protection mechanisms for crosstalk which are incorporated in the majority of dual chamber pacing systems, have to be well understood by the interpreter. Spontaneous ventricular events occurring during the ventricular blanking period or the ventricular safety period give rise to an ECG pattern of ventricular undersensing, but should be considered as normal pacemaker function.

Atrial undersensing is easily masked on the ECG if the programmed AV interval is longer than the PR interval and the lower rate VA interval is longer than the intrinsic RR interval (Fig. 6.36). Programming a shorter AV interval than the intrinsic PR interval is the best method to evaluate normal P wave sensing (Fig. 6.37). If atrial undersening is observed on the ECG, ventricular oversensing, occurring during the VA interval should also be excluded as the possible cause (Fig. 6.41). A marker channel is very valuable in this respect because it immediately elucidates the cause of undersensing. If no marker channel is available ventricular oversensing can be excluded by programming the pacing system to the VVI mode. If ventricular oversensing is present, it is demonstrated by prolongation of the escape interval. Causes of atrial undersensing are described in chapter V, and should be evaluated in a similar manner. Atrial undersensing caused by pacing mode reversion (DOO, VVI) will be indicated by pacemaker telemetry.

Atrial oversensing is demonstrated on the ECG by emission of a ventricular stimulus without preceding atrial depolarization. Recording of precordial leads or intracardiac electrograms is necessary to visualize the absence of intrinsic atrial depolarization, thus confirming atrial oversensing. Oversensing cannot be confirmed by the marker channel. Causes of atrial oversensing in dual chamber systems are indentical to those in single chamber systems as described in chapter V. The only additional cause is sensing of far-field ventricular afterpotentials or the last portion of the paced ventricular electrogram by the atrial electrode. The latter condition may lead to pacemaker tachycardia (chapter VI 7.4.2).

The effect of ventricular oversensing in dual chamber pacing systems depends on the point of time of the ventricular sense event in the timing cyclus of the pacing system. Ventricular oversensing during the ventricular safety pacing interval, which can be induced by crosstalk, gives rise to ventricular stimulation with a shortened AV interval. Oversensing during the ventricular inhibiting period of the AV interval resets the VA timer in ventricular based timing systems, the ventricular stimulus is inhibited and the Ap-Ap interval will be shortened, provided no intrinsic atrial depolarization is present. In atrial based timing systems ventricular oversensing in this period will also inhibit the ventricular stimulus but there is no effect on the Ap-Ap

interval. Ventricular oversensing during the ventricular relative refractory period has no effect on the lower rate timing. A ventricular sense event in this period initiates prolongation of the ventricular refractory period but also restarts PVARP. Prolongation of the ventricular and the atrial refractory period may give rise to a pattern of undersensing (atrial or ventricular) on the ECG. Ventricular oversensing during the inhibiting period of the VA interval resets the lower rate timer, which can result in prolongation of the V-A interval (Fig. 6.44) or the V-V interval (Fig. 6.45). Depending on the occurrence of intrinsic activity after oversensing, undersensing can be observed. If, however, ventricular oversensing is present but the intrinsic P wave following oversensing, resulting in ventricular stimulation, occurs before the VA interval is elapsed the ECG will show normal pacemaker function (Fig. 6.46).

Marker channel recording will elucidate ventricular oversensing but if this option is not available, ventricular sensing can best be evaluated by programming the device in the VVI mode.Causes of oversensing have already been discussed in chapter V.

In the case of observing ventricular undersensing, oversensing resulting in prolongation of the refractory period or interference mode reversion should be excluded. This can be done by using the marker channel or, if the marker channel is not available by programming the system to the VVT mode. The VVT mode will show effective ventricular stimulation with shortening of the escape interval if oversensing is present.

Undersensing is obvious on the ECG if the VA interval is terminated by an atrial stimulus. The coupling interval between the atrial stimulus and the preceding ventricular depolarization is shortened if undersensing is present. If the VA interval is terminated by an intrinsic atrial depolarization, recognition of ventricular undersensing may be more complicated. A coupling interval between the intrisic ventricular depolarization and the following ventricular stimulus shorter than the upper rate interval indicates ventricular undersensing. If the P wave following the intrinsic ventricular event falls in the PVARP or extended PVARP of the ventricular event, but in spite of this timing, results in ventricular stimulation it can be concluded that PVARP was not initiated by the ventricular event because of undersensing. Undersensing can be confirmed by the marker channel or if not available, programming the system to the VVT mode at a rate below the intrinsic rate. In this mode no ventricular stimulus will be emitted in the intrinsic QRS complex if undersensing is present.

Causes of ventricular undersensing are discussed in chapter V.

References

1. Barold SS. Upper rate of DDD pacemakers. The view from the atrial side. PACE 1988; 11; 2149-2159.

2. Olson WH, Fillpovich MD, Biallas RM. Cardiac output during upper rate limiting in VDD and DDD pacemakers.(abstract) PACE 1983; 6: A-81.

3. Barold SS, Falkoff MD, Ong LS , et al. Function and electrocardiography of DDD pacemakers. In S Barold (Ed): Modern Cardiac Pacing. Mount Kisco, New York, Futura Publishing Co., Inc., 1985, pp6454-675.

4. Furman S. Dual chamber pacemakers: Upper rate behavior. PACE 1985; 8: 197-214.

5. Stroobandt R, Willems R, Holvoet G, et al. Prediction of Wenckebach behavior and block response in DDD pacemakers. PACE 1986; 9: 1040-1046.

6. Higano ST, Hayes DL. Quantitative analysis of Wenckebach behavior in DDD pacemakers. PACE 1990; 13: 1456-1465.

7. Daubert C, Ritter P, Mabo P, et al. Physiological relationship between AV interval and heart rate in healthy subjects: Application to dual chamber pacing. PACE 1986; 9: 1032-1039.

8. Ritter P, Pioger G, Vai F, et al. Metabolic benefit of rate responsive atrioventricular delay (AVD) in DDD pacing.(abstract) PACE 1990; 13: 531.

9. Hessen SE, Sheppard RC, Kutalek SP. Upper rate lockout: An unexpected feature of rate adaptive atrioventricular delay. PACE 1994; 17: 103-107.

10. Meta DDDR, dual chamber rate responsive pacing system. Physian's manual, 1991.

11. Hayes DL, Maue-Dickson W, Stanton MS. Dictionary of cardiac pacing electrophysiology and arrhythmias. Miami Lakes, Florida, Peritus Corporation, 1993, p. 42.

12. Furman S, Reichter-Reiss H, Escher DJW. Atrioventricular sequential pacing and pacemakers. Chest 1973; 63: 783-789.

13. De Keyser F, Vanhaecke J, Janssens L, et al. Crosstalk with external bipolar pacing: A case report. PACE 1991; 14: 1320-1322.

14. Pimenta J, Soldá R, Pereira CB. Tachycardia mediated by an AV universal (DDD) pacemaker triggered by a ventricular depolarization. PACE 1986; 9: 105-107.

15. Johnson CD. Atrial synchronous ventricular inhibited (VDD pacemaker mediated arrhythmia due to atrial undersensing and oversensing of far-field ventricular afterpotentials or paced beats: Crosstalk. PACE 1986; 9: 710-719.

16. Byrd CL, Schwartz SJ, Gonzales M, et al. Rate responsive pacemakers and crosstalk.(abstract) PACE 1988; 11: 798.

17. Combs WJ, Reynolds DW, Sharma AD, et al. Crosstalk in bipolar pacemakers. PACE 1989; 12: 1613-1621.

18. Beaver BB, Maloney JD, Castle LW, et al. Design-dependent crosstalk in a second generation DDD pacemaker. PACE 186; 9: 65-77.

19. Barold SS, Falkoff MD, Ong LS, et al. Crosstalk due to activation of atrial sense marker function of DDD pulse generators. PACE 1987; 10: 293-301.

20. Dodinot B, Costa AB, Godenir JP, et al. Ventricular crosstalk a frequent complication of dual chamber pacing depending on polarity and electronics.(abstract) PACE 1992; 14: 738.

21. Brunner U. Personal communication. October 1992.

22. Sanford CF. Self-inhibition of an AV sequential demand (DVI) pulse generator due to polyurethane lead insulation disruption. PACE 1983; 6: 840-844.

23. Physician's manual Cosmos II. Intermedics Inc, March 1990, pp.32-33.

24. Barold SS, Ong LS, Falkoff MD, et al. Crosstalk or self-inhibition in dual chamber pacemakers. In Barold SS (Ed): Modern cardiac pacing. Mount Kisco, New York, Futura Publishing Company, 1985, p 618.

25. Levine PA, Mace RC. Assessment and management of crosstalk. In Pacing Therapy: A guide to cardiac pacing for optimum hemodynamic benefit. Mount Kisco, New York, Futura Publishing Company, 1983, pp. 239-251.

26. Barold SS, Belott PH. Behavior of the ventricular triggering period of DDD pacemakers. PACE 1987; 10: 1237-1252.

27. Josephson ME, Seides SF. Clinical Cardiac Electrophysiology. Techniques and Interpretations. Philadelphia, Lea & Febiger, 1979, pp. 81-85.

28. Josephson ME, Scharf DL, Kastor JA, et al. Atrial endocardial activation in man. Electrode catheter technique of endocardial mapping. Am J Cardiol 1977; 39: 972-978.

29. Lamas GA, Antman EM, Gold JP, et al. Pacemaker backup mode reversion and injury during cardiac surgery. Ann Thorac Surg 1986; 41: 155-157.

30. Barold SS, Falkoff MD, Ong LS, et al. Resetting of DDD pulse generators due to cold exposure. PACE 1988; 11; 736-743.

31. Physicians's manual Cosmos II. Intermedics Inc, March 1990, pp 31-32.

32. Physician's information manual, dual chamber pulse generators, volume 1. Medtronic Inc, 1992, p 21,38.

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33. Satler LF, Rackley CE, Pearle DL, et al. Inhibition of a physiologic pacing system due to its anti-pacemaker-mediated tachycardia mode. PACE 1985; 8: 806-810.

34. Wilson JH, Lattner S. Undersensing of P waves in the presence of an adequate P wave due to automatic postventricular atrial refractory period extension. PACE 1989; 12: 1729-1732.

35. Van Mechelen R, 't Hart C, de Boer H. Failure to sense P waves during DDD pacing. PACE 1986; 9: 498-502.

36. Johnson CD. Atrial synchronous ventricular inhibited (VDD) pacemaker-mediated arrhythmia due to atrial undersensing and atrial lead oversensing of far-field ventricular afterpotentials or paced beats: crosstalk. PACE 1986; 9: 710-719.

37. Ajiki K, Sagara K, Namiki T, et al. A case of pseudomalfunction of a DDD pacemaker. PACE 1991; 14: 1456-1460.

38. Sudduth BK, Morris DL, Gertz EW. Noise mode response at peak exercise in a DDD pacemaker. PACE 1985; 8: 746-752.

39. Fröhlig G, Dyckmans J, Doenecke P, et al. Noise reversion of a dual chamber pacemaker without noise. PACE 1986; 690-696.

40. Van Gelder LM, Nagelkerke D, Mulder H, et al. Unipolar versus bipolar electrode configuration in cardiac pacing. Neth J of Cardiol 1992; 5: 280-285.

41. Van Gelder LM, El Gamal MIH. Influence of myopotentials on implanted DDD-M pacemakers. In Steinbach K, Glogar D, Laszkovics A, et al (Eds): Cardiac Pacing. Darmstadt, Steinkopff Verlag, 1983, pp. 555-560.

42. Risser T, Drumm GW. A safety feature for protecting against loss of output with bipolar lead fracture.(abstract) Eur J Cardiac Pacing Electrophysiol 1992; 2: A64.

43. Mauser JF, Huang SK, Risser T, et al. A unique pulse generator safety feature for bipolar lead fracture. PACE 1993; 16: 1368-1372.

44. Intermedics Technical Memo. Bipolar lead verification in Omega family pulse generators. Intermedics Inc., August, 1993.

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Chapter VII. **Pacemaker Tachycardia**

Berry van Gelder* and Karel den Dulk**,
*   *Department of Cardiology, Catharina Hospital, Eindhoven, The Netherlands,*
**   *Department of Cardiology, Academic Hospital Maastricht, Maastricht, The Netherlands.*

Introduction

Pacemaker tachycardia can be defined as a non-physiological tachycardia in which the pacemaker plays an essential role in initiating and sustaining the tachycardia. Many types of pacemaker tachycardias have been published in the literature. The purpose of this chapter is to review the various types of pacemaker tachycardia according to their mechanism. To facilitate discussion five categories of tachycardia have been defined:

I.   Runaway pacemaker.
II.   Sensor mediated tachycardia.
III.   Pacemaker circus movement tachycardia.
IV.   Timer related tachycardia.
V.   Pacemaker synchronization to supraventricular tachycardia

7.1. Runaway pacemaker

Runaway pacemaker has been defined as a pacemaker malfunction resulting in an inappropriately fast pacing rate (100 to 2100 ppm has been reported). This problem is unpredictable, can be intermittent and usually represents a medical emergency with removal of
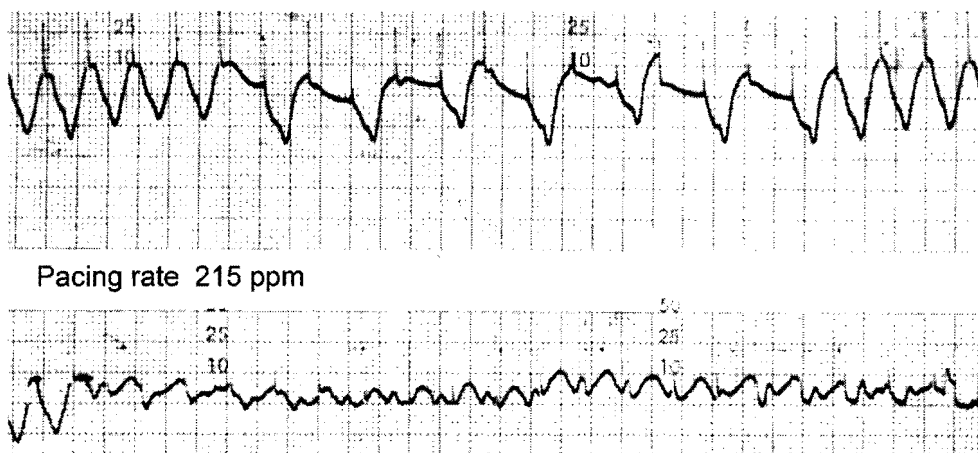

Pacing rate 215 ppm

Fig. 7.1. Implanted VVI runaway pacemaker firing at a rate of approximately 215 ppm, showing periods of complete and 2:1 capture (upper tracing) with ultimate deterioration to ventricular fibrillation (lower tracing).

the pulse generator. Most of these reports were published before 1980 [1-6]. Basically there are two categories of runaway pacemakers.

7.1.1 Spontaneous runaway pacemaker

The main reason for pacemaker runaway was battery depletion or the influx of body fluids into the electric circuitry. This body fluid affected the electrical components resulting in changes in pacemaker timing thus allowing high pacing rates (Fig. 7.1) up to 1500 ppm [7]. Hermetically sealed pacemaker housing, preventing the penetration of body fluids and electrical protection circuits, has practically eliminated this problem.

7.1.2 Externally induced runaway pacemaker.

Several externally induced runaway pacemakers have been reported, such as following the use of electrocautery [8], application of a coil of a pacemaker analyzer (Fig. 7.2) [9,10], and radiation therapy [11]. All reported cases were irreversible.

Measures have been taken to prevent pacemakers from runaway by incorporating runaway protection circuits. A runaway protection rate of 140 ppm in single chamber devices may not prevent symptoms related to the higher pacing rate, but will rarely result in hemodynamic deterioration with a fatal course. The runaway protection rate in dual chamber systems varies from 180 to 200 ppm.
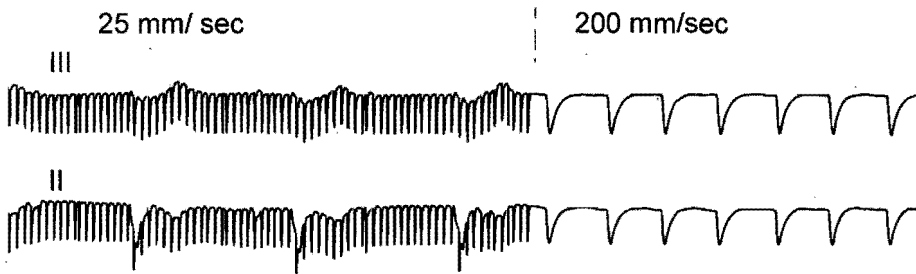
Fig. 7.2. Two channel ECG (lead I,II)recorded at 25 mm/s and 200 mm/s, showing rapid artifacts with a rate of 1200 ppm masking an underlying heart rhythm with a rate of 50 bpm. Pacemaker runaway was induced by the use of a pacemaker analyzer.

The outcome of patients with a runaway pacemaker with a high runaway rate will be determined by four main factors:
1.   The repetition rate of the pulse generator
2.   The output of the pulse generator, whether it is below or above the stimulation threshold at the time of runaway. At higher pacing rates the output capacitor cannot be recharged completely, thus reducing pacemaker output.
3.   The presence or absence of an underlying heart rhythm, when the output of the pulse generator is below the stimulation threshold.
4.   Induction of arrhythmias in which factors such as rate, ischemia, substrate and asynchronous competetive pacing also play a role.

The mortality of runaway in unprotected pacemakers was 34% in a series of 44 cases [2].

7.1.3 Management of runaway pacemaker

A characteristic of pacemaker runaway is that magnet application does not affect the pacing rate. Two reports [6,12] describe successful inhibition of the implanted device by overdrive chest wall

stimulation. However, in one case [6] this maneuver had to be terminated promptly as there was no underlying escape rhythm present. Although overdrive stimulation and decrease of pacemaker output below the stimulation threshold can be attempted, the recommended therapy is immediate replacement or disconnection of the pulse generator

.

7.2 Sensor Mediated Tachycardia (SMT)

Sensor mediated tachycardia can be defined as a tachycardia which is due to false triggering of the sensor, which subsequently gives rise to an inappropriate high pacing rate. Apart from technical failure ("sensor runaway"), SMT can be divided into two categories:
1.   SMT induced by sensor detected "interference".
2.   SMT induced by positive sensor feedback. The feedback signal is generated by the pacing system.

7.2.1 Sensor mediated tachycardia induced by interference

SMT induced by interference is caused by lack of specificity of the sensor to discriminate between interference from the appropriate sensor indicated changes. When interference stops or is eliminated, normal pacemaker function is restored. SMTs related to interference have been reported for activity, temperature and respiration sensors.

7.2.1.1 Activity sensor

The activity sensing pacemaker senses body activity through a piezoelectric pressure sensor bonded to the inside of the pulse generator's can. When the piezoelectric crystal is stressed or bent by body activity, it produces a signal that causes the pacing rate to increase. Vibrations

Programmer head on

Pacing rate 114 ppm

2 min. after removal

Pacing rate 76 ppm
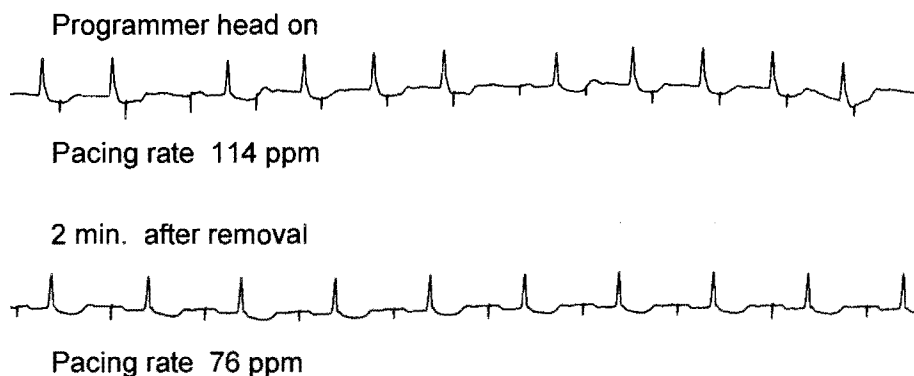
Fig. 7.3. Single lead ECG of a patient with an activity based rate adaptive AAI pacemaker implanted in the left pectoral region. After application of the programmer head an increase in stimulation rate to 114 ppm is recorded (upper panel). Two minutes after removal of the programmer head stimulation rate decreased to 76 ppm (lower panel).
Note: Increase in stimulation rate gives rise to second degree AV block.

originating from various forms of transportation [12] or direct pressure on the pulse generator give rise to inappropriately high pacing rates [13,14]. Vibrations from external sources such as automobile or bus may give rise to an increase in pacing rate. In practice, however, this seldom

leads to symptomatic rate increase unless the vibrations are extreme (e.g. tractor, helicopter).Upper rate pacing has been described in a patient after programming the pacing mode from DDD to DDDR [16]. During programming the programmer head was resting on the pacemaker pocket. Marker channel recording revealed sensor activity after each QRS complex indicating mechanical cardiac activity as a source of internal interference. The weight of the programmer head was just enough to push the pulse generator towards the heart, enabling detection of mechanical heart activity. The pulse generator was implanted in the left pectoral region. A similar observation was described in a patient with retromammary implantation of a VVIR pacemaker [17] and with a left sided pectoral implantation of an AAIR pacemaker (Fig.7.3).

A very unusual case of internal interference was described in a patient with an arterio-venous shunt for chronic intermittent hemodialysis, at the same site as the pulse generator [18]. Particularly in the supine position a faint flutter was detected by the piezo electric crystal of the sensor, resulting in an increase in pacing rate.

7.2.1.2 Respiration sensor

In the first respiratory rate adaptive pacemakers, respiration rate was detected by measuring intrathoracic impedance between the pulse generator can and an auxiliary lead that had to be implanted subcutaneously across the chest. Movement of the arm caused movement of the auxiliary lead thus increasing impedance and pacing rate [19].
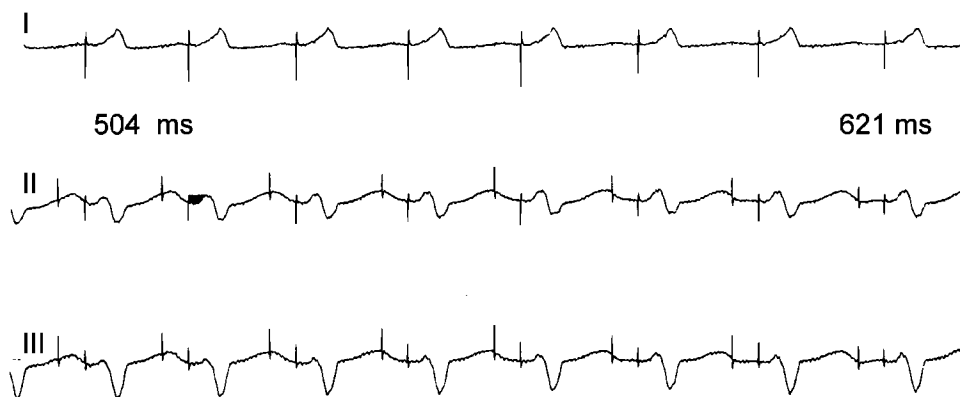
Fig. 7.4. Upper rate pacing (120 ppm) after RF ablation in a patient with a respiration sensing DDDR pacemaker. A gradual decrease in pacing rate was noticed after termination of ablation application.

In the currently available pacing systems no auxiliary electrode is needed, since impedance is measured between the tip of a conventional bipolar lead and the pulse generator can. Respiratory rate adaptive pacing systems using intrathoracic impedance as a sensor are however prone to interference from high density electrical fields like electrocautery and radio frequency ablation equipment. The electrical field generated by this equipment, is measured by the sensing circuit for intrathoracic impedance. When a high voltage is detected this is interpreted as a high impedance; upper rate pacing will result (Fig 7.4)[20,21].

Recently a similar phenomenon has been observed after connecting a patient with a minute ventilation rate adaptive pacemaker (AAIR) to monitoring equipment that also measures respiration rate. The current applied to the ECG electrodes in order to measure respiration rate was detected by the sensing circuit for intrathoracic impedance and resulted in upper rate pacing (Fig.7.5)[22].

16:54:51 02-SEP-1993   25mm/sec

16:56:57 02-SEP-1993   25mm/sec

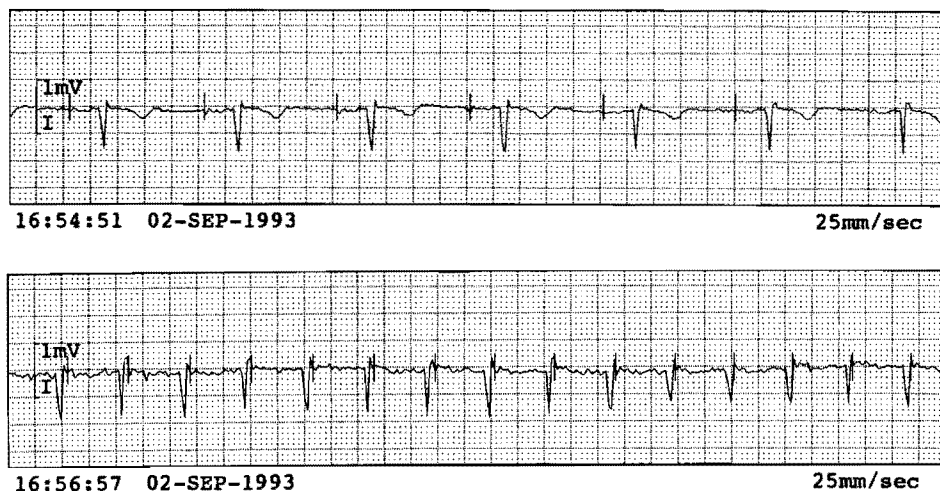*Fig. 7.5. Upper rate pacing (lower panel) in a minute ventilation rate adaptive AAIR pacemaker 2 minutes after connecting the patient to the ECG monitor. Current applied to the ECG electrodes to measure respiration rate was interpreted by the pacemaker as an increase in intrathoracic impedance, resulting in upper rate pacing.*

7.2.1.3 Temperature sensor
Rate adaptive pacemakers using central venous temperature as a sensor are affected by internal or external changes in temperature and can increase the pacing rate without any metabolically indicated need. Reported changes however are within acceptable limits and rarely lead to tachycardia [23].

7.2.2 Sensor Mediated Tachycardia induced by positive feedback
In SMT with positive feedback, a secondary reaction to the pacemaker stimulus (e.g. muscle twitching) is detected by the sensor thus increasing the pacing rate, which in turn activates the sensor followed by a further increase in pacing rate [24]. Pacing rate can rise to the programmed upper rate. Positive feedback SMT is usually sustained but is easily terminated and prevented by reprogramming the pacing system. SMTs due to a positive feedback mechanism have been described for activity, QT and temperature pacemakers.

7.2.2.1 Activity sensor
Pocket pacing in unipolar pacemakers (which can be due to high output settings, lack of an insulating cover or flip of the pacemaker [25] if one site has been isolated) can give rise to muscle twitching in the pocket, which is detected by the sensor which in turn increases the

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frequency of pacing and pocket stimulation, subsequently followed by further increase in pacing rate, and finally resulting in upper rate pacing (Fig. 7.6). The increase in pacing rate following sensor activation leads to a positive feedback increase in sensor activity.
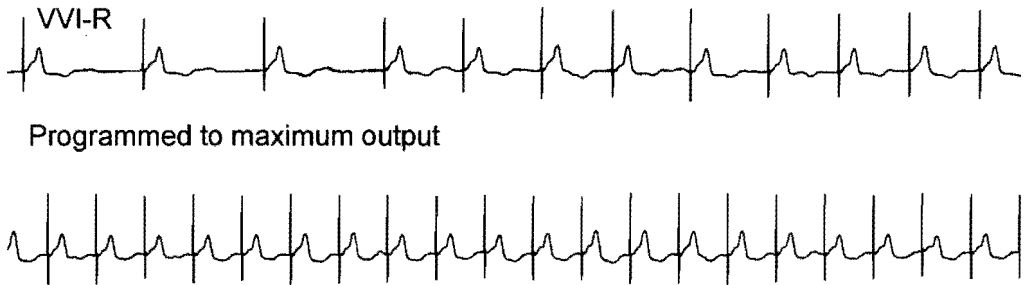

Fig. 7.6. Activity sensing pacemaker showing lower rate pacing (upper tracing) evolving to upper rate pacing (lower tracing) after programming the pacemaker to its maximal output of 8 Volt. High output setting is associated with muscle twitching which in turn is detected by the activity sensor which then increases the pacing rate.

7.2.2.2 QT sensor
Another form of sensor mediated feedback tachycardia was observed in the early versions of the QT sensing pacemaker [26]. In this device the slope determining the relationship between the shortening of the QT interval and pacing rate was linear. At low stimulation rates, the rate related shortening of QT is smaller compared to higher stimulation rates. This implies that a programmed slope appropriate for the lower rates was too sensitive when the pacing rate increased. An increase in pacing rate resulted in a rate related QT shortening interpreted by the pulse generator as an increase in metabolic need, leading to a further rate increase. This problem was only encountered in the initial versions of the device.

7.2.2.3 Temperature sensor
Sensor mediated tachycardia with sensor feedback was also reported in a single chamber temperature sensitive pacemaker [27]. The pacing system measures central venous blood temperature. Diaphragmatic stimulation from the right ventricular pacing electrode gave rise to an increase in central venous blood temperature, which in turn was sensed by the pacemaker. Pacing rate increased to the programmed upper rate.

7.3 Pacemaker Circus Movement Tachycardia (PCMT)
Antidromic PCMT
A pacemaker circus movement tachycardia (PCMT) is an artificial reentry tachycardia in which the retrograde limb of the circuit is provided by the atrioventricular (A-V) node or accessory pathway [28], while the anterograde limb is by way of synchronization of the pacemaker [29]. These PCMTs are sometimes also called artificial circus movement tachycardias (or, less specifically, pacemaker mediated or endless loop tachycardias), because the circulating event is conducted from atria to the ventricles via the pacemaker and returning to the atria via the His bundle-AV node. Occasionally it is called antidromic PCMT to distinguish it from orthodromic

PCMT, where the circulating event is conducted from atrium to ventricle via the AV conduction system and the retrograde limb of the reentry circuit is provided by the pacemaker as will be discussed later (see paragraph 7.3.2).
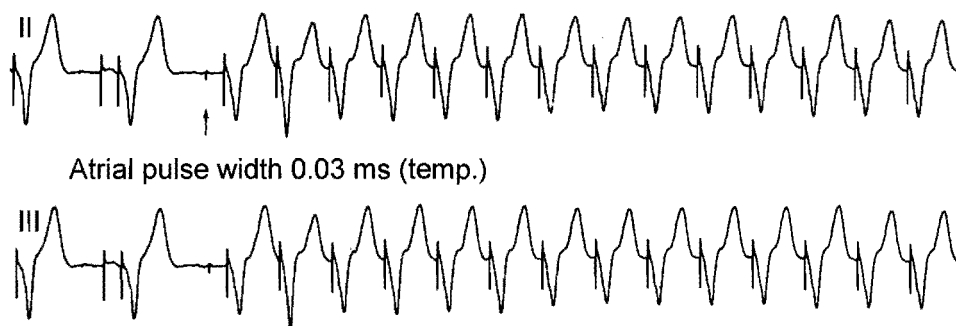

Fig. 7.7. Induction of PCMT by temporarily programming atrial pulse width below atrial stimulation threshold (arrow). Because the ventricular paced beat is not preceded by an atrial depolarization ventriculo-atrial conduction takes place and PCMT is initiated.

PCMT can occur in every pacing system with atrial synchronous ventricular stimulation possibilities (VAT, VDD, DDD), but clinical relevance became obvious with the introduction of DDD pacemakers with short non-programmable post ventricular atrial refractory periods. A prerequisite for initiation of PCMT is ventriculo-atrial (VA) conduction, which is present in 68% of patients with normal A-V conduction and even in 44% of patients with abnormal A-V conduction [30].

If stimulated ventricular depolarization is preceded by atrial depolarization, V-A conduction is not likely to occur, as the atrial activity will have depolarized part of the retrograde pathway, thereby causing refractoriness. If however the atrial events and the paced ventricular events are sufficiently separated, so that the retrograde pathway is no longer refractory, V-A conduction and PCMT may result [29,31,32,33].

This can occur under the following circumstances:

1. Ventricular premature beats
2. Atrial premature beats, which prolong AV interval significantly
3. Wenckebach upper rate behaviour
4. Artificial triggering in the atrial channel (e.g., myopotentials, chest wall stimulation, magnet application [34])
5. Loss of capture in the atrial channel (Fig 7.7)
6. Atrial undersensing [35]
7. Rate smoothing [36]
8. Fall back mode (in VVI mode)

Magnet application can initiate PCMT in dual chamber pacemakers without atrial pacing capability (VDD, VAT), because magnet application results in VOO pacing, thus enabling VA conduction and induction of PCMT after removal of the magnet (Fig. 7.8) [37]. Similarly PCMT

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can be initiated if the sinus rate decreases below the programmed lower rate giving rise to ventricular escape pacing (VDD) which could result in PCMT in the presence of VA conduction.
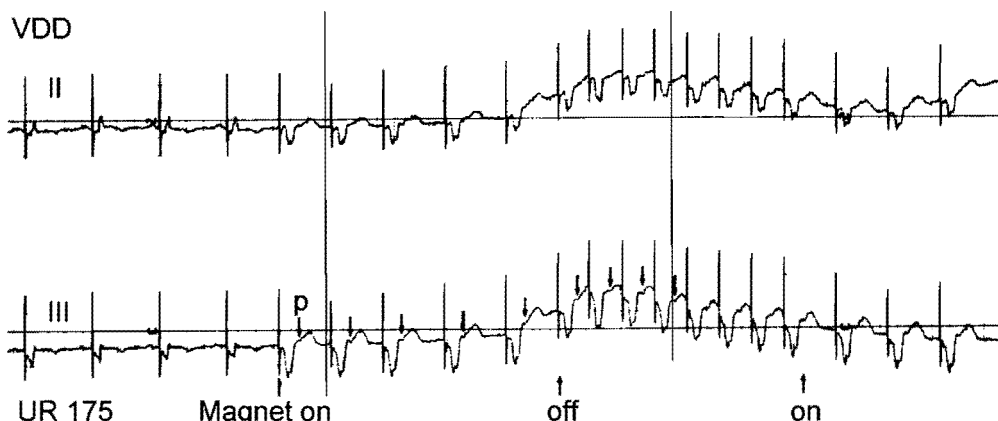

Fig. 7.8. Two channel ECG (lead II,III) of a patient with an implanted VDD pacemaker. Application of the magnet resulted in fixed rate ventricular pacing with VA conduction. Arrows indicate retrograde P waves. When the magnet was removed retrograde P waves were sensed by the atrial amplifier and PCMT was initiated. The magnet was subsequently reapplied.

7.3.1 Management of PCMT

Management of PCMT can be divided into preventing the onset of tachycardia or the termination of tachycardia. Preventing the onset of PCMT is based on ignorance of the retrogradely conducted P wave. A number of the programmable parameters can be used for prevention but they often are associated with a limitation of the upper rate behavior of the pacing system. Termination is achieved by recognition of tachycardia followed by an interruption of atrial sensing for one cycle. Several algorithms are developed for recognition of PCMT.

7.3.1.1 Post ventricular atrial refractory period (PVARP)

An effective way of preventing PCMT is by programming an appropriately long PVARP, thus ignoring the retrograde P-wave [29,38]. It will be clear that the length of PVARP, in order to give sufficient protection against PCMT, has to be longer than the VA conduction interval. PCMTs with relatively short VA intervals are easily controlled by programming an appropriately long PVARP without affecting the upper tracking rate adversely by induction of 2:1 AV block. Long PVARP required for a long VA conduction interval, will adversely affect the upper tracking rate. The upper tracking rate is determined by the total atrial refractory period (TARP), sum of PVARP and the programmed AV interval. If the P-P interval is shorter than TARP, every second P wave will not be sensed, resulting in 2:1 AV block. To illustrate the point, programming an unusual and exceptionally long PVARP of 400 ms with an AV interval of 150 ms gives a TARP of 550 ms with induction of 2:1 block at an atrial rate of 109 bpm. Programming a shorter AV interval of 100 ms in the previous example improves the situation by shortening the TARP to 500 ms (400 + 100 ms); 2:1 block now occurs at 120 bpm. Shortening of the AV interval during rate increase or having PVARP shortened during a physiological rate increase (controlled by a sensor [39] for example) could further increase the upper tracking

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capabilities. The sensor indicated rate also offers the possibility of discrimination between physiological and pathological tachycardia, thus preventing pacemaker mediated tachycardia.

7.3.1.2 Ventricular Premature Beats

Automatic extension of PVARP after a ventricular premature beat (VPB) has made a major contribution to the prevention of PCMTs intiated by VPBs. Initially some pacemakers reverted to DVI mode upon detection of a VPB. However, PCMTs can still occasionally be initiated by the DVI mode after the VPB. If the atrial stimulus of the temporary DVI mode occurs during the refractory period of the atrial tissue following a preceding sinus P wave, the ventricular paced event is now able to conduct retrogradely to the atrium. A DDD escape mode after a VPB with an extended atrial refractory period is therefore a better option.

7.3.1.3 PVC synchronous atrial stimulation

Because premature ventricular contractions (PVCs) can initiate PCMT some pulse generators (e.g., Quintech DDD 931, Vitatron Medical BV) have a programmable feature to prevent the
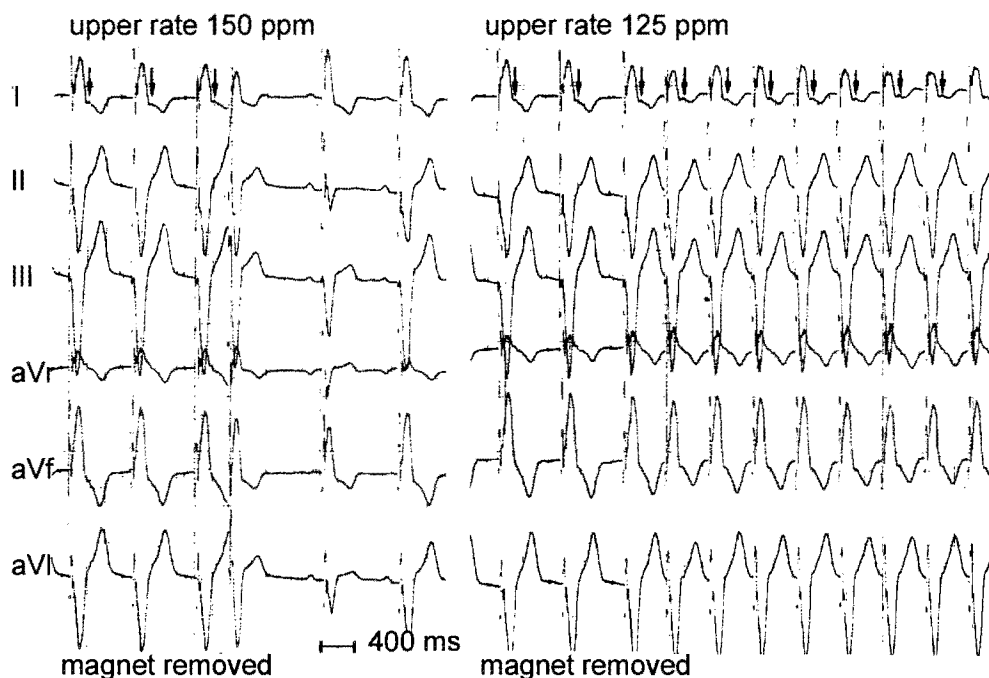
Fig. 7.9. Illustration of retrograde block by upper rate programming. In the left panel the retrograde P wave is sensed after removal of the magnet. The upper rate of 150 ppm allows a coupling interval between the retrograde P wave and the subsequent ventricular stimulus short enough to provoke VA block. In the right panel the upper rate of 125 ppm is low enough to enable 1:1 retrograde conduction.

onset of PCMT by PVCs [39]. This feature is designed to stimulate the atrium 30 ms after the detection of a PVC by the ventricular lead, thereby causing concealed antegrade penetration of

the VA pathway to prevent VA conduction. The pacemaker defines a ventricular event as a PVC, if the ventricular depolarization is not preceded by a sensed or paced atrial event. If an atrial event is a PVC-synchronous atrial stimulus, the next ventricular event is also interpreted as a PVC with resulting PVC-synchronous atrial stimulation, provided no atrial activity was sensed in between

7.3.1.4 Atrial sensitivity
An elegant way of preventing PCMT would be a device which could distinguish sinus P waves from retrograde P waves. Unfortunately this is not possible on a reliable basis yet [39,40,41,42]. Application of bipolar atrial leads has prevented initiation of PCMT by artificial triggering of ventricular stimuli to sensed myopotentials or other sources of external interference.

7.3.1.5 The role of the AV interval and upper rate
When the atrial rate exceeds the programmed upper rate (TARP < upper rate interval), there is a gradual prolongation of the AV interval, finally resulting in a Wenckebach block. With a further increase in atrial rate 2:1 AV block will occur when the P-P interval decreases below the TARP. It has been demonstrated that programming a higher upper rate prevents initiation of tachycardia [45], by preventing initiation of Wenckebach behavior during sinus tachycardia or by an atrial premature beat. The VA conduction properties will determine the ability to induce VA block by shortening the ventricular stimulation interval, thus preventing or terminating PCMT. Three factors determine the prematurity of the ventricular stimulation interval at the onset of or during PCMT. The spontaneous VA interval, the programmed AV interval and the programmed upper rate limit. The longer the VA interval, the shorter the AV interval will have to be, to result in a stimulus close enough to the previous one to provoke VA block (Fig. 7.9). However, one should take care when using this option for prevention of PCMT, because VA conduction properties are influenced by autonomic tone and 1:1 VA conduction may then be maintained.

7.3.1.6 Tachycardia termination algorithm
From the preceding paragraphs it will be clear that PCMTs with long VA intervals are the most difficult to prevent because they require long PVARPs to ignore retrograde P waves, which in turn compromises maintenance of 1:1 atrial sensing ventricular pacing. Because timing can be critical and VA conduction times can vary due to variations in autonomic tone or medication, PCMT cannot always be prevented. Different algorithms have been developed to recognize and terminate PCMT in those cases.

PVARP prolongation for 1 beat
PCMT is easily terminated by not synchronizing a ventricular paced event to a retrograde P wave by prolonging the PVARP for one beat (Fig. 7.10). Several PCMT detection algorithms have been developed:
1. In the first algorithms PCMT detection was achieved after a certain number of consecutive ventricular paced beats at the programmed upper rate limit [46,47,48]. If this was due to sinus tachycardia the device did not also synchronize every Nth beat, if a beat had already been dropped due to the Wenckebach behavior. Recognition by means of continued upper rate pacing may not be helpful in patients with a long VA interval. PCMT rate will be low, thus requiring programming a low upper rate to trigger with its disadvantages as previously discussed.

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2. Others use an algorithm that can be activated at a selectable ventricular paced rate, below the upper rate limit [49,50].

3. Varying the V-V interval. In this algorithm the stability of retrograde conduction during PCMT is used as a detection algorithm. This algorithm does not depend on the rate of PCMT wheter it is equal to or below the programmed upper rate [51].

Discrimination between PCMT and sinus or atrial tachycardia is made as follows: during tachycardia every second AV interval is shortened by a programmable short interval, resulting in a similar variation in the V-V interval [52,53,54]. If the VA conduction time as measured by the device remains stable (within a programmable range) during this variation in the V-V interval, PCMT is assumed to be present.

Muscle interference
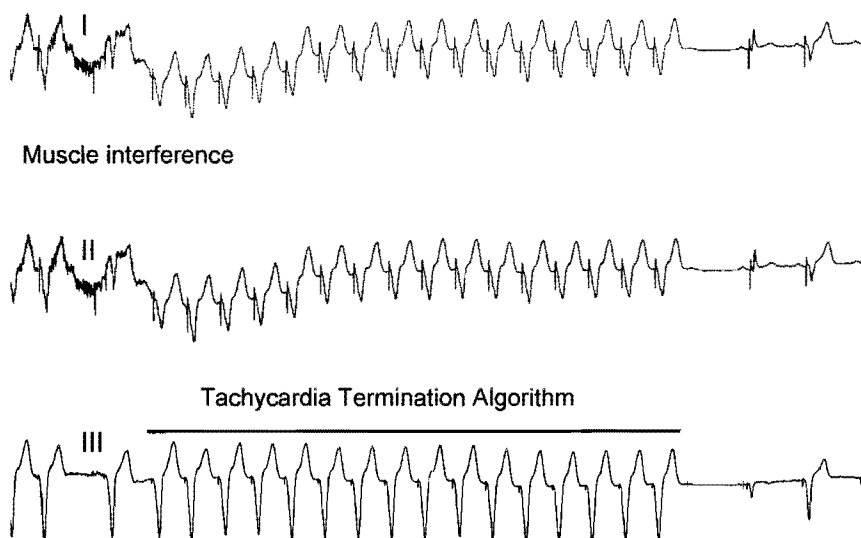
Tachycardia Termination Algorithm

Fig. 7.10. Initiation of PCMT by synchronization of ventricular paced outputs to pectoral muscle artifacts. PCMT is detected by the tachycardia termination algorithm. It is allowed to proceed for 15 consecutive ventricular pace events at the programmed upper rate and then does not synchronize a ventricular pace event, thus breaking the reentry loop.

If however the delay between the paced ventricular beat and the sensed P wave increases by the value of the shortened V-V interval in case of atrial tachycardia due to dissociation during V-V variation, PCMT is assumed to be absent. If PCMT is assumed to be present because the VA interval remains stable during V-V interval variation then PCMT is interrupted by lengthening of PVARP for one cycle.

7.3.2 Orthodromic PCMT

As previously mentioned some manufacturers have incorporated PVC-synchronous atrial stimulation as a protection mechanism for initiation of PCMT. A rare type of circus movement tachycardia has been observed in the presence of atrial undersensing and intact AV conduction. The pacemaker defines a ventricular event as a PVC, if the ventricular depolarization is not preceded by a sensed or paced atrial event [55]. After atrial undersensing the normally conducted

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QRS complex is defined by the pacemaker as a PVC. Therefore a PVC-synchronous atrial stimulus is delivered. This paced atrial complex is subsequently conducted to the ventricles with a prolonged AV interval. The resulting ventricular depolarization is once again recognized as a PVC, giving rise to PVC synchronous atrial stimulation (Fig. 7.11).

Because the antegrade limb of the tachycardia circuit is formed by the antegrade AV nodal pathway and the retrograde conduction by means of the pacemaker this tachycardia is called orthodromic PCMT. Orthodromic PCMT can be initiated by a junctional escape beat, if the pacemaker is programmed to a lower rate below the rate of the escape rhythm [56].
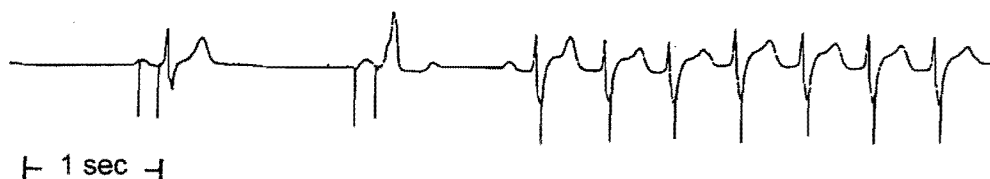

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Fig. 7.11. ECG demonstrating onset of orthodromic PCMT. After atrial undersensing, the pacemaker detects a normally conducted QRS complex. This QRS complex is interpreted as a PVC, since no previous atrial event was sensed. Therefore a PVC-synchronous atrial stimulation is delivered. This stimulated atrial complex is conducted to the ventricles with a long AV interval. The next QRS complex is again recognized as a PVC, and so on. (Courtesy Dr. P. den Heijer)

Orthodromic PCMT can occur under the following conditions:
1.   PVC-synchronous atrial stimulation mode programmed on
2.   Intermittent atrial undersensing or escape rhythm
3.   Intact AV conduction with a conduction time which is longer than the ventricular refractory period of the device

Intermittent atrial undersensing has to be considered as the mechanism for initiation of orthodromic PCMT. However, it should be realized that orthodromic PCMT can be initiated in a normally functioning device if the lower rate is programmed below the rate of the escape rhythm, provided that the escape rhythm is no sinus rhythm.

7.4 Timing related tachycardia (TRT)
There are a number of pacemaker related tachycardias that are hard to classify in one of the previously mentioned subgroups. These tachycardias have in common the fact that tachycardia is maintained by undesirable sensed events. In this respect an undesirable sensed event is not a synonym for interference, because the sensed events can be generated by or in the pacing system itself. For example atrial arrhythmias are not considered as undesirable sensed events generated by the pacing system. Because tachycardia is maintained by sensing undesirable signals thus affecting the timing cycle, we used the term timing related tachycardia (TRT).

TRT is encountered in single as well as in dual chamber pacemakers.

7.4.1 Timing related tachycardia in single chamber systems
In single chamber pacemakers TRT is only encountered in the triggered mode (VVT, AAT). In an early model of VVT pacemakers tachycardia has been observed at a rate of about 130 beats/min, corresponding to the refractory period of the pulse generator [57]. Tachycardia was

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initiated and sustained by sensing the pacemaker afterpotential at the end of the refractory period. Sensing the afterpotential evoked an effective pacemaker pulse. The afterpotential of this pulse was subsequently sensed and formed the trigger for the next pacemaker pulse resulting in tachycardia with a tachycardia interval equal to the refractory period. Because at that time no pacemakers were available with programmable features (sensitivity, output, refractory period, pacing mode) able to solve the problem, tachycardia could only be prevented by replacing the pulse generator.

The mechanism of tachycardia in AAT pacemakers is related to far-field QRS sensing, which forms the trigger for the high pacing rate [58]. In unipolar atrial pacemakers the QRS complex resulting from the conducted stimulated P-wave can be sensed by the atrial electrode. If this QRS is falling outside the atrial refractory period it evokes an atrial stimulus that is conducted to the ventricle and which once again can be sensed by the atrial electrode thus initiating tachycardia (Fig. 7.12).
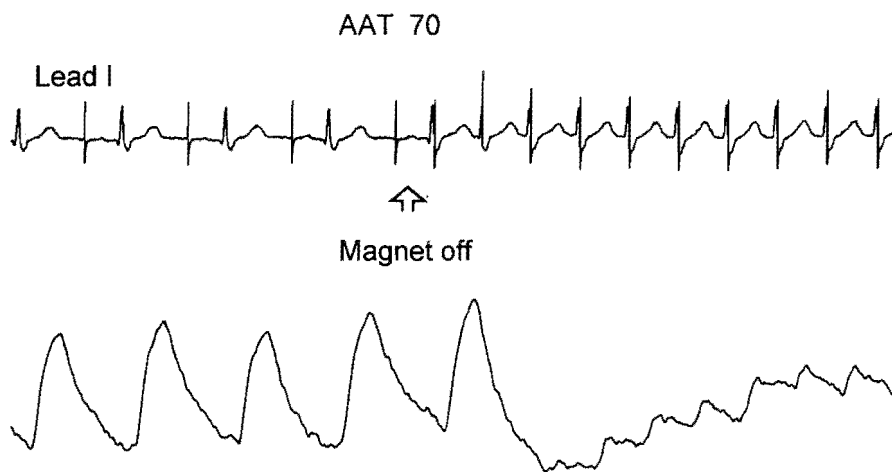
AAT 70

Fig. 7.12. ECG and atrial pulse pressure curve of a patient with an atrial pacemaker (AAT). After removal of the magnet there is far-field QRS sensing on the atrial lead, which evokes an atrial stimulus. The atrial stimulus is conducted to the ventricles and the QRS complex is again sensed by the atrial electrode, thus evoking tachycardia. The drop in arterial pulse pressure is due to the near simultaneous occurrence of atrial and ventricular events during tachycardia which affected ventricular filling adversely.

There are a number of prerequisites [58] for this tachycardia to occur:
1. Use of the atrial triggered mode (AAT)
2. Stimulus-QRS interval > atrial refractory period
3. Far-field QRS sensing
4. Tachycardia rate < maximum pacing rate

Because the AAT mode is used sometimes in patients with unipolar atrial pacemakers, in order to prevent inhibition of the pacing system by myopotentials, far-field QRS sensing should be excluded in these patients.

Initiation of tachycardia can be prevented by reducing sensitivity, provided normal atrial sensing is maintained or by extension of the atrial refractory period.

7.4.2 Timing related tachycardia in dual chamber systems

Timing related tachycardia can be encountered in dual chamber systems, either programmed in the DDD mode or the DVI mode. In the DDD mode tachycardia is initiated and maintained by inappropriate triggering of the ventricular stimulus. The erroneous signals responsible for inappropriate triggering are generated in or by the pacing system. In the DVI mode tachycardia was maintained by a specific programmed setting related to the escape interval, but initiated by pacemaker malfunction (self inhibition; myopotential interference).
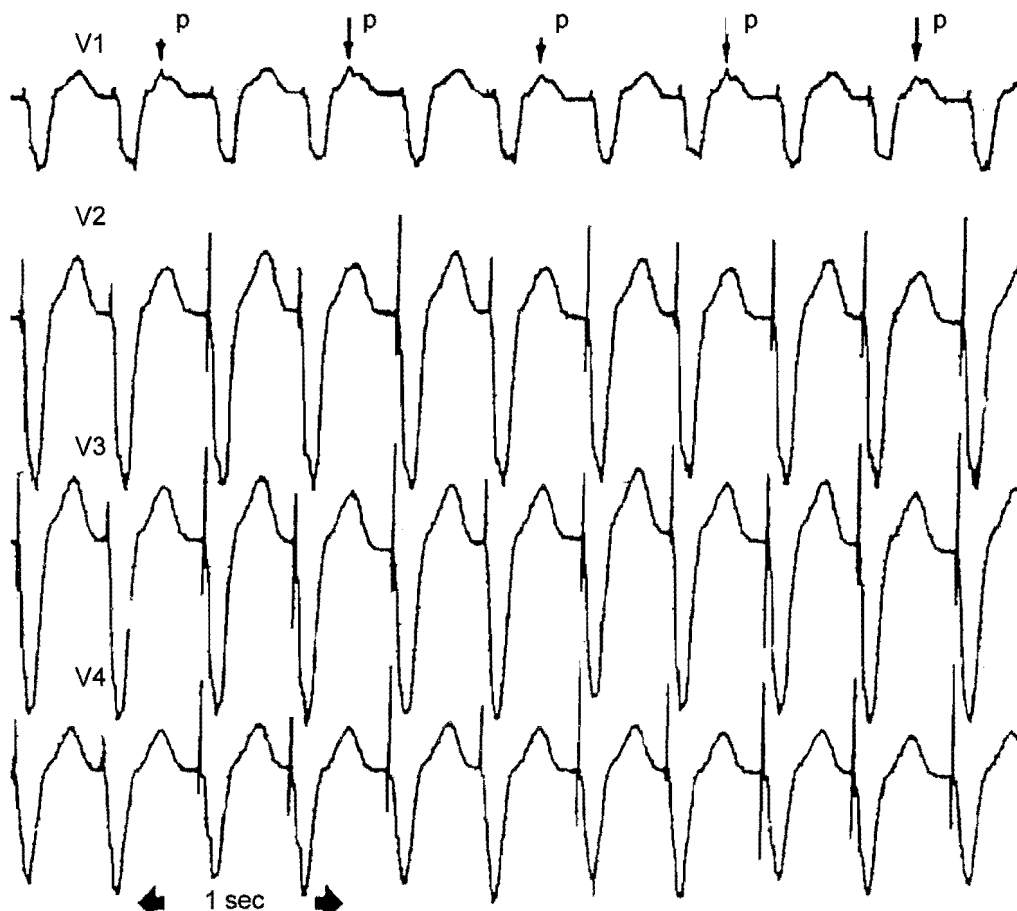
Fig. 7.13. Four channel ECG recording (precordial leads V1 to V4) showing TRT at a rate of 132 bppm. TRT is caused by sensing of a voltage dip in the atrial channel just after the atrial refractory period. (Courtesy Dr. R. Stroobandt)

7.4.2.1 TRT by inappropriate triggering of the ventricular stimulus

Recently a case of pacemaker mediated tachycardia was reported that can be considered as a TRT [60]. Tachycardia was encountered in a DDD pacemaker after exceeding the recommended replacement time. Battery impedance had increased to 11.8 kOhm. In this particular pacemaker,

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as it had passed the recommended replacement time, the state of the battery may have caused a voltage dip on the pacemaker supply line [61]. The drop of the supply voltage is caused by switching on the pacemaker microprocessor. The microprocessor is switched of during a part of the pacing cycle to save battery energy. When the microprocessor is switched on, the current drain from the battery is increased and battery voltage decreases due to the high internal resistance of the battery. Because power supply to the microprocessor is identical to the power supply to the atrial sensing amplifier, the voltage drop is detected by the atrial sensing amplifier and may erroneously be interpreted as a P wave, which in turn initiates ventricular pacing. Synchronously with ventricular pacing a PVARP is started, during which the microprocessor is switched off. At the end of the PVARP the microprocessor is turned on again, provoking a voltage dip with resulting ventricular pacing thus initiating tachycardia (Fig. 7.13).

Careful analysis of the ECG during tachycardia shows alternating variations in the ventricular paced intervals. When P waves were present between two ventricular paced beats, the pacing interval was approximately 30 ms longer. In addition to the V-V pacing interval being longer, the "spontaneous" P waves had a fixed coupling interval to the preceding ventricular stimulus, suggesting the presence of retrograde conduction. The slight prolongation in ventricular pacing interval occurring synchronously with the interpolated P waves raises the question as to whether the voltage dip was the only explanation for this tachycardia. As it happens this particular pulse generator is able to sense atrial events in the relative refractory period of PVARP [62]. Sensing during PVARP can also activate the microprocessor that was switched off. The time used to process the atrial event (approximately 30 ms) and switching off the microprocessor is added to PVARP, so that the atrial sense caused by the voltage dip at the end of PVARP is delayed by 30 ms. This explains the longer pacing interval when P waves were interpolated between two ventricular pace events.

Most of the reported dual chamber pacemakers TRTs are caused by far-field sensing of ventricular related activity by the atrial electrode [63]. This can be the QRS complex, the T wave or the afterpotential of the ventricular stimulus. Sensing of the QRS complex by the atrial electrode in dual chamber pacemakers is unusual. A QRS complex, whether spontaneous or pacemaker induced, initiates the PVARP thus eliminating sensing of the QRS complex by the atrial electrode. However, sensing the QRS complex of a paced ventricular beat by the atrial electrode has been reported [64].

It was demonstrated by intracardiac electrograms showing a prolonged ventricular spike-to-peak R wave interval. If this interval exceeds the programmed PVARP and the far-field QRS complex is of sufficient amplitude, a ventricular stimulus is synchronized to the sensed event.

Similar observations have also been described [65,66] but in these cases T-wave or far-field ventricular afterpotentials were considered to be the cause of TRT. Displacement of the atrial lead into the right ventricular outflow tract has also been responsible for the sensing of ventricular activation by the atrial electrode [67].

TRT in dual chamber pacemakers caused by sensing of ventricular related activity, including afterpotentials of the ventricular stimulus, is rarely sustained. Sometimes it is even limited to occasional triggering, leading to pacemaker mediated arrhythmias instead of tachycardia. The phenomenon can easily be prevented by prolonging the PVARP. In case of atrial lead dislodgement repositioning is the obvious solution.

7.4.2.2 Escape interval related tachycardias

Two other forms of TRT have been encountered in DDD pacemakers programmed in the DVI mode. In one patient in whom the DVI mode was programmed to a lower rate of 110 ppm and an AV interval of 200 ms (to test for self inhibition due to afterpotentials) a tachycardia of 150 ppm occurred [29].
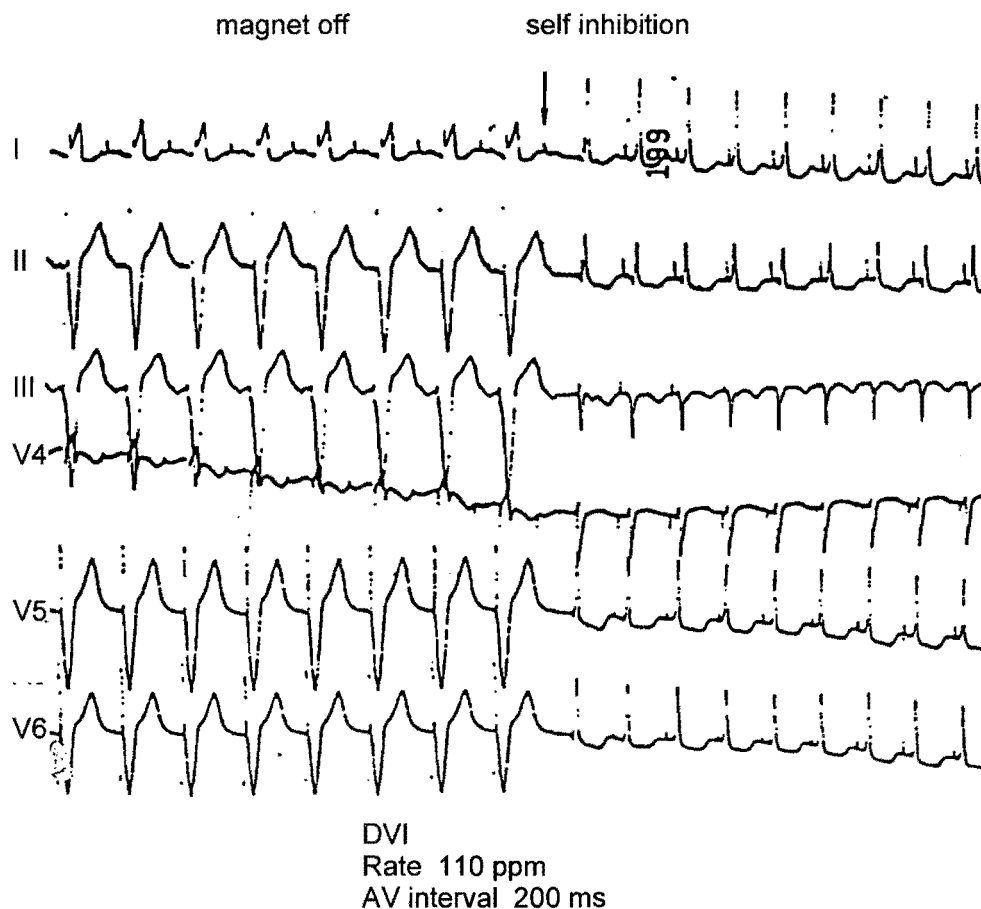

Fig. 7.14. Initiation of TRT in a DDD pacemaker programmed in the DVI mode. After removal of the magnet there is inhibition of the ventricular output at, or just after, the atrial stimulus artifact (arrow). This mechanism gives the paced atrial complex the chance to be conducted with a long AV interval. Just before the resulting QRS complex, a second atrial stimulus occurs and tachycardia is initiated. See text for further details.

Tachycardia was initiated by inhibition of the ventricular output after removal of the magnet. The paced atrial complex was conducted to the ventricles with a prolonged AV conduction time. Just before the occurrence of the conducted QRS complex a second atrial stimulus occurred. This second atrial paced event was again conducted with a prolonged AV interval thus repeating the timing cycle (Fig.7.14).

Because the pacing device had ventricular based timing, the VA interval equals the programmed lower rate minus the programmed AV interval; in this case 545-200 = 345 ms. The second atrial stimulus however was preceding the conducted QRS complex of the first stimulus. Thus, although the AV conduction time was prolonged, in pacemaker timing there was only a short AV interval (the interval between the second atrial impulse and the subsequent QRS complex generated by the first atrial stimulus). Due to this short AV interval of approximately 55 ms, the total cycle length became 55 (AV interval) plus 345 (VA interval) which equals 400 ms corresponding to the tachycardia rate of 150 ppm. The maximum rate of tachycardia depends on the programmed lower rate, the AV interval and the interval by which the AV interval is shortened by the sensed QRS complex. It will be higher at a long AV interval and a high lower rate, with resetting of the VA escape interval immediately after the atrial stimulus.

DVI 100 ppm
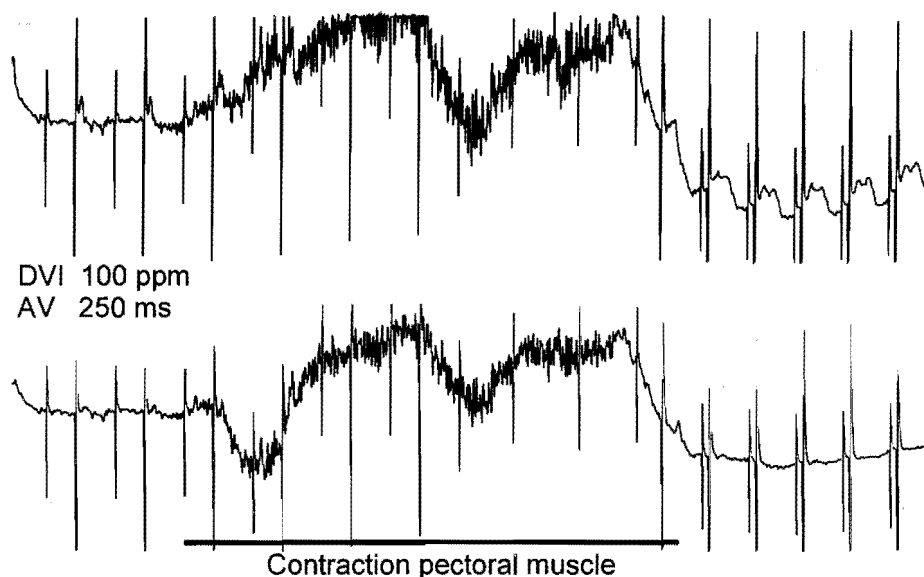AV 250 ms

Contraction pectoral muscle

Fig. 7.15. Two channel ECG (lead I,II) showing initiation of TRT (rate 150 ppm, AV interval 50 ms) after contraction of the pectoral muscle in a patient with an implanted DDD pacemaker programmed in the DVI mode. See text for further details.

Tachycardia could be terminated by application of a magnet or by carotid sinus massage. Tachycardia in this patient was initiated by crosstalk in an early model of DDD pacemaker and will rarely be encountered in currently available pacemakers because of effective crosstalk protective mechanisms which are incorporated in the new devices. The second TRT was encountered in a DDD pacemaker, programmed in the DVI mode, with a lower rate of 100 bpm and an AV interval of 250 ms [68]. In this setting pectoral muscle contraction initiated AV sequential pacing at a rate of 150 ppm and an AV interval of 50 ms (Fig.7.15).
The mechanism for initiation was explained as follows. The DVI mode in the pulse generator is realized by disabling the atrial tracking capability of the DDD mode. The upper rate mechanism is still fully functional although it is not normally "visible" in the DVI mode. The upper rate was "blindly" programmed to 150 ppm by the programmer when DVI mode was selected. Due to the

discrete processing time in the pacemaker circuitry, ventricular sensing that occurs in a 3.9 ms window directly preceding the ventricular stimulus may fail to reset the escape interval. It may lead to a ventricular stimulus at the end of the upper rate interval while timing for the atrial stimulus (the VA interval), remains unchanged. At the programmed settings the VA interval equals 600-250= 350 ms, with the upper rate programmed at 150 ppm (= 400 ms), resulting in an AV interval 50 ms. This mode of tachycardia, defined as a normal VA interval, in combination with pacing at the upper rate interval, can be sustained only if the VA interval is shorter than the upper rate interval. By "blindly" programming (which is later incorporated in the programmer) the upper rate to 175 ppm (= 343 ms) and excluding selection of combinations of lower rate and AV interval resulting in a VA interval < 343 ms, initiation of this tachycardia was further prevented.

7.5. Pacemaker synchronization to supraventricular tachycardia.

Atrial tachyarrhythmias like atrial flutter/fibrillation (AF) preclude the use of DDD pacing since the device synchronizes ventricular stimuli to the sensed atrial tachyarrhythmia.The incidence of atrial fibrillation after implantation of a DDD pacemaker varies from 5.6 [69] to 9.8% [70], even in the absence of a previous history of atrial fibrillation. Onset of atrial fibrillation after implantation of a pacemaker which synchronizes ventricular stimuli to sensed atrial events may result in an inappropriate ventricular pacing response (Figs.7.16,7.17).

Lead III Pacing rate 112 ppm
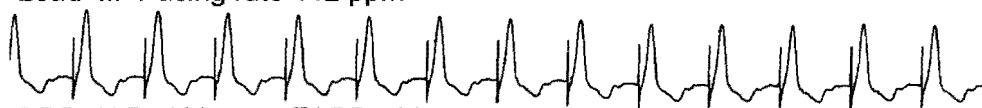
DDD U.R. 120 ppm; TARP 400 ms

Fig. 7.16. Single channel ECG (lead III) of a patient with an implanted DDD pacemaker. Ventricular pacing at a rate of 112 ppm (=535 ms) caused by sensing atrial flutter (rate 224/min=268 ms) with 2:1 block. (TARP 400 ms; URI 500 ms). See also fig. 7.17.
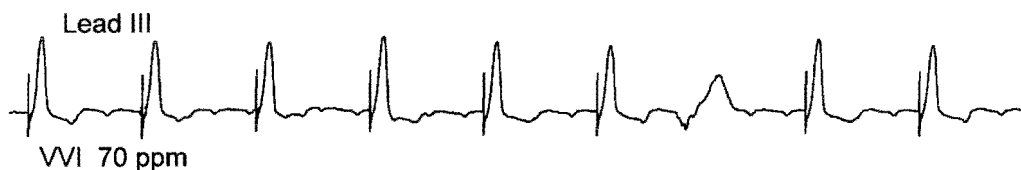
Lead III

VVI 70 ppm

Fig. 7.17. Same patient as figure 7.16 after programming the pacemaker to VVI mode 70 ppm. ECG (lead III) shows atrial flutter waves with a rate of 224/min.

During atrial fibrillation intermittent or complete undersensing of the fibrillation waves can be observed, resulting in an irregular ventricular pacing reponse or even pacing at the lower rate [71]. Ventricular pacing rate depends on the rate of the atrial arrhythmia, the programmed total atrial refractory period (TARP) and upper rate limit. If during atrial flutter or tachycardia, sensing of the atrial electrogram is consistent and the atrial arrhythmia is regular, the programming of TARP and the upper rate limit results in a predictable ventricular pacing

response. TARP determines the rate at which atrial tachycardia will be sensed. E.g. an atrial flutter at a rate of 224/min (= 280 ms interval) will result in a sensed atrial rate of 112/min if 280 < TARP < 560 ms , because every second flutter wave falls inside TARP. The ventricular pacing rate will be 112 ppm, if the upper rate limit is [3] 112 ppm (Figs. 19,20) and equal to the upper rate limit, with a pacemaker Wenckebach reponse if the upper rate limit is < 112 ppm.

7.5.1 Mode switching

Several mode switching devices are currently undergoing clinical evaluation. These devices change to a non-tracking mode upon recognition of a supraventricular tachyarrhythmia (Fig.7.18).
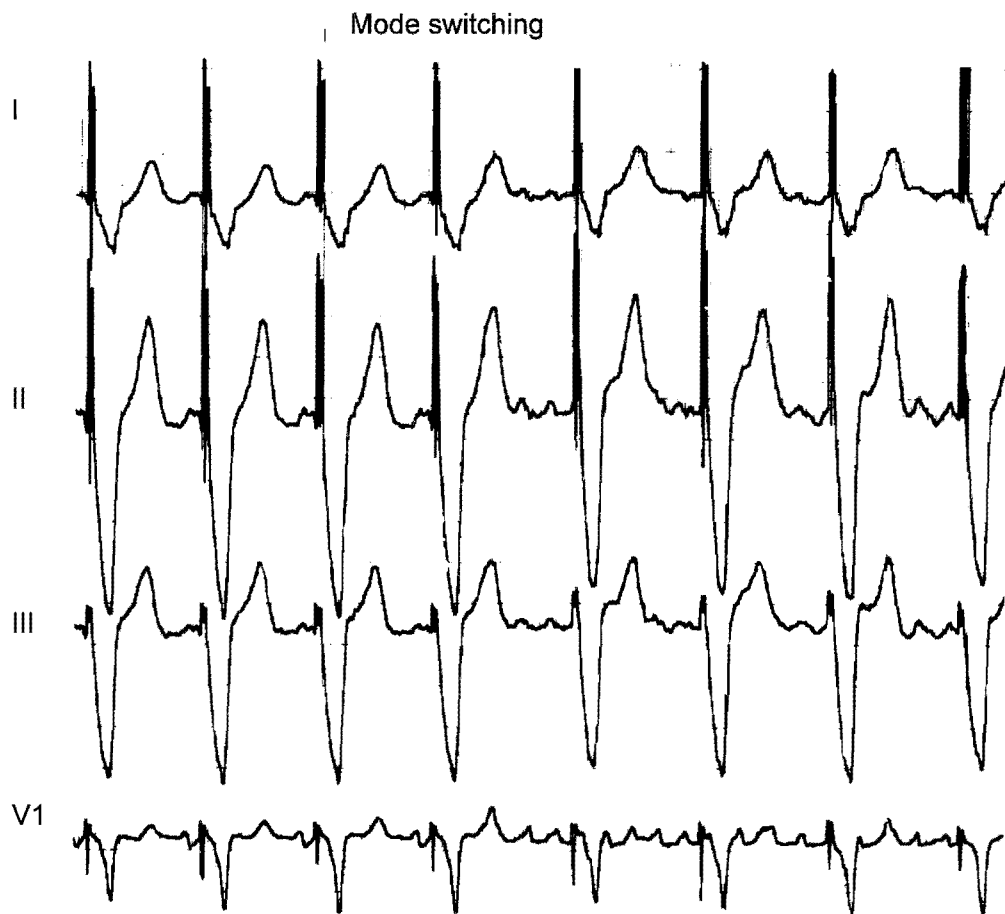
Fig. 7.18. Simultaneously recorded four channel ECG, showing tracking of sinus rhythm followed by detection of atrial flutter and reversion to VVI pacing (Mode switching).

Recognition of supraventricular tachycardia can be achieved by first detecting a certain number of beats above a "tachycardia detection rate". This has the disadvantage of producing several ventricular paced beats at the programmed upper rate. After reaching the upper rate limit and

having detected supraventricular tachycardia the device goes into the fallback mode and the ventricular paced rate is decreased gradually to the programmed lower rate or to the sensor determined rate. One of the initial devices demonstrated undesirable mode switching because the device switched to the VVIR mode for at least 7 beats, upon detection of an atrial event in PVARP [72]. Inappropriate mode switching could be initiated by atrial premature beats occurring in PVARP or ventricular premature beats with an atrial event in PVARP. Although patients did not have the increase in ventricular paced rate during supraventricular tachycardia detection, they experienced inappropriate mode switches with loss of AV synchrony for several beats, which could induce symptoms in patients in whom the atrial contribution to ventricular filling is important.

Limiting the ventricular rate increase during detection of tachycardia by algorithms such as rate smoothing has the potential disadvantage of creating AV dissociation during increase in atrial rate which is too rapid or after an atrial premature beat. This temporary loss of AV synchrony could once again induce symptoms in patients with an important atrial contribution to ventricular filling [73].

It is worth noting that atrial fibrillation or flutter can be induced by dual chamber pacemakers when there is competition between atrial activity and the atrial stimulus [37,74]. Atrial stimulation during the atrial vulnerable period can occur due to initiation of asynchronous pacing during magnet application, atrial undersensing or when the atrial stimulus is not inhibited because the P wave occurs in the PVARP. The latter situation is more likely to occur in a sensor driven DDI or DDD mode. A recently developed algorithm (atrial synchronization pulse) avoids this competitive pacing at the atrial level because atrial pacing only occurs after a "safe interval" has been timed out from the previously detected atrial event. This algorithm also enables quick resynchronization and optimizes AV synchronization [74].

Lead III

Atrial burst   10 mm/s

Fig. 7.19. ECG (10 mm/s) of a patient with a DDD pacemaker and atrial flutter (same patient as fig. 7.16). Ventricular pacing rate 112 ppm due to sensing atrial flutter waves (rate 224/min) with 2:1 block. Overdrive atrial pacing with the implanted pulse generator terminated atrial flutter and normal DDD pacing was restored. Note that ventricular pacing is interrupted during the atrial burst.

7.5.2 Atrial overpacing by the implanted pulse generator

Atrial flutter can be successfully terminated by rapid atrial pacing at a rate approximately 125% of the flutter rate [75]. Pulse generators equipped with the feature of rapid atrial pacing can be useful in the treatment of atrial flutter [76]. Currently this is only available in a temporary mode

without ventricular back-up pacing during the burst (Fig.7.19). Future devices will provide ventricular back-up pacing during burst atrial pacing for patients with total AV block.

7.6 Summary and conclusions

Pacemaker tachycardia defined as a non-physiological tachycardia in which the pacemaker plays an essential role to initiate and sustain the tachycardia are known throughout the history of cardiac pacing. The mortality caused by extremely high pacing rates (pacemaker runaway) is almost completely eliminated by the hermetical sealing of the pacemaker housing and runaway protection circuitry. Pacemaker tachycardia encountered in to day's pulse generators is rarely fatal but can result in disabling symptoms for the patient.

Pacemaker circus movement tachycardia (PCMT) and its mechanism is well known. After implantation of a DDD pacemaker it is relatively easy to determine the presence or absence of retrograde VA conduction. In the presence of retrograde conduction programming a PVARP long enough to ignore retrograde P waves is an effective measure for PCMT prevention. The only exception is the patient with a long VA conduction time, in whom the upper rate behavior is adversely affected by a long PVARP. The development of "intelligent" pacemakers allowing discrimination between normal and retrograde P waves would be the best solution for the prevention of PCMT.

Sensor mediated tachycardia are more tricky. They can be initiated completely unexpectedly, as for instance tachycardia in minute ventilation rate adaptive systems in patients who are connected to ECG monitoring equipment that also measure respiration rate (see fig. 7.5). Every sensor, based on the principle of measurement of an electrical signal, is potentially prone to sensor interference resulting in tachycardia. In this respect the hospital environment carries the highest risk for the patient with such a device, because electrical current is frequently applied to patients for diagnostic and therapeutic purposes. In the rapidly developing field of electronics and electronic communication new sources of interference may come up that will interfere with pacemaker function and initiate new sensor mediated tachycardias.

Timing related tachycardia (TRT) are rare phenomena, with a very low incidence. In some of them the underlying cause is difficult to find (Figs. 7.13, 7.15), but the other forms of TRT are in general easy to prevent by programming the appropriate refractory period.

In tachycardia in dual chamber systems due to synchronization to supraventricular tachycardia 2 mechanisms are involved. First is the development of supraventricular tachycardia and secondly the synchronization of the pacing system to this tachycardia. The solution of this problem is mainly sought by prevention of synchronization to the supraventricular tachycardia by mode switching devices, thus precluding symptoms related to rapid ventricular pacing. Although in a proper mode switching device this might be a solution in symptom preclusion, the primary cause is not treated. If in case of mode switching the patient is not aware of the supraventricular tachycardia, this arrythmia can persist for longer and treatment might be more difficult. If on the other hand in a device without mode switching, tachycardia is perceived by the patient, he will come to the clinic earlier and medical or electrical (overpacing, cardioversion) treatment can be initiated immediately, which increases the success rate, thus eliminating the primary cause of tachycardia.

In the diagnosis of pacemaker tachycardia the flow chart presented in figure 7.20 can be helpful. If a patient is presenting with pacemaker tachycardia the first diagnostic step is magnet application. If the tachycardia rate is not affected pacemaker runaway can be concluded.

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Inhibition of the implanted system can be tried by chest wall stimulation at a rate higher than the tachycardia rate, thus inhibiting the implanted system. Because chest wall stimulation is not always successful in inhibiting the implanted system in case of pacemaker runaway and the risk of inhibiting the system in a pacemaker dependent patient, emergency pulse generator replacement is indicated.

If magnet application results in magnet rate pacing, the clinician has to determine what type of pacing system is implanted. In case of a single chamber without rate adaptation, the system is in the triggered mode and the tachycardia is a timing related tachycardia. This can be prevented by programming the inhibited mode or by prolongation of the refractory period.

If the pacing system is a sensor driven system, whether single chamber or dual chamber the rate adaptive mode should be swichted off by disabling the sensor. If normal pacing is restored sensor mediated tachycardia can be concluded. The source of sensor interference, responsible for this tachycardia has te be eliminated. If tachycardia persists after disabling the sensor, the system can only be a dual chamber system.

In a dual chamber system without rate adaptation attention should be paid to the chamber(s) being paced during tachycardia. AV sequential pacing during tachycardia indicates that the tachycardia is timing related which is only encountered if the device is programmed in the DVI mode. Programming to the DDD mode is the solution for this very rare type of tachycardia. Atrial pacing during tachycardia, which can be confusing because the ECG resembles an intrinsic tachycardia with pacemaker stimuli in the QRS complex, similar to VVT pacing, orthodromic PCMT can be concluded. Because this can only occur in pulse generators with PVC synchronous atrial stimulation, disabling of this option solves the problem.

Providing ventricular pacing is observed during tachycardia the presence of supraventricular arrhythmias should be checked. If supraventricular arrhythmias are present tachycardia is due to synchronization of ventricular stimuli to this arrythmia. Disabling atrial sensing by programming the system to VVI(R) or DDI(R) mode prevents tachycardia.

If no supraventricular arrhythmias are present, VA conduction should be checked. Presence of VA conduction indicates that the tachycardia is an antidromic PCMT, which can best be prevented by programming the PVARP to an interval longer than the VA conduction time. If no VA conduction is present the tachycardia is a timing related tachycardia (TRT) caused by inappropriate triggering of the ventricular stimulus. The source for inappropriate triggering should be traced. If far-field QRS, T wave or afterpotential of the ventricular stimulus is sensed by the atrial electrode, it can be prevented by prolongation of PVARP. If inappropriate triggering is caused by the pulse generator itself, it is considered as generator malfunction and the pulse generator has to be replaced.
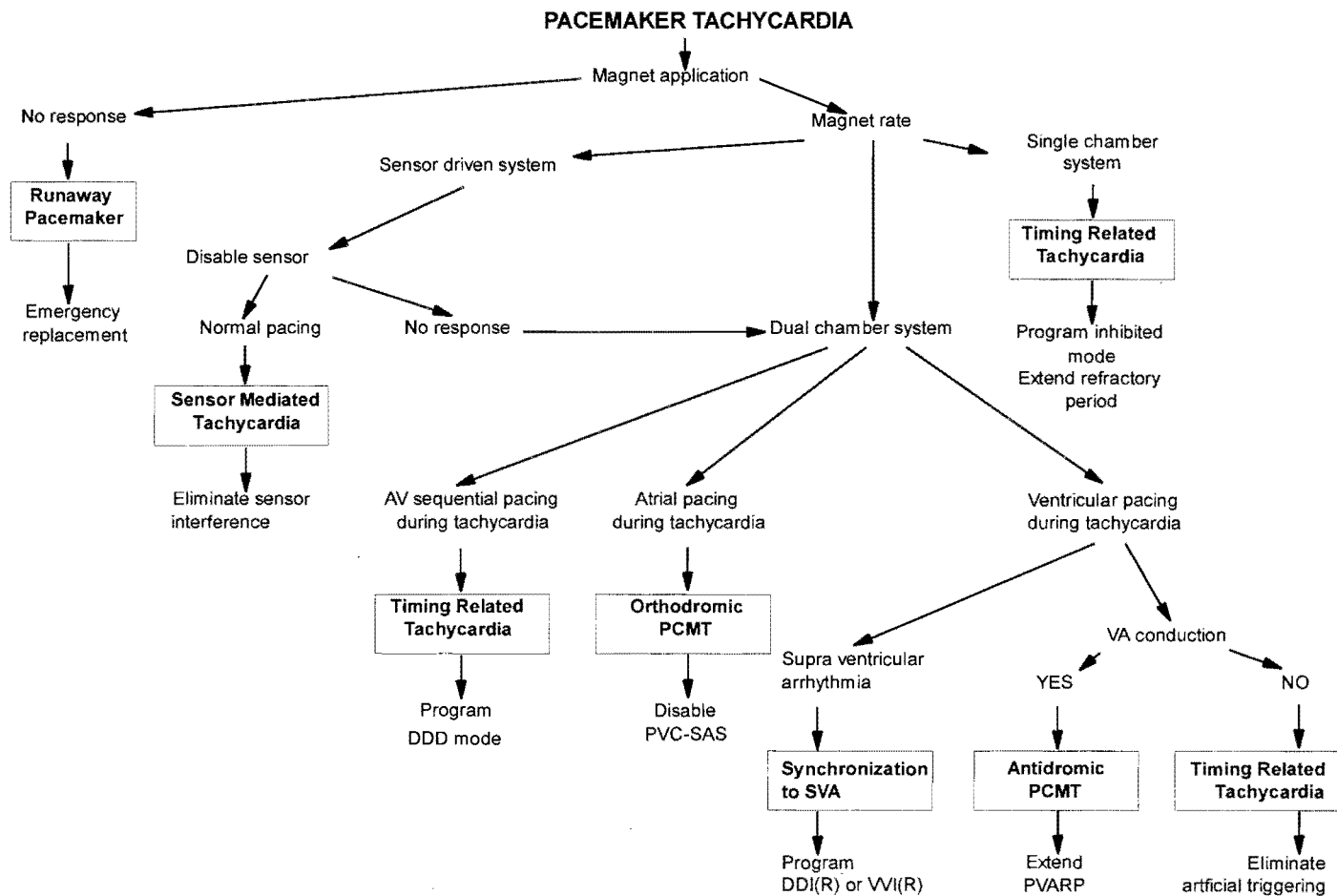
PACEMAKER TACHYCARDIA

Magnet application

- **No response**
  - **Runaway Pacemaker**
    - Emergency replacement

- **Magnet rate**
  - **Sensor driven system**
    - **Disable sensor**
      - **Normal pacing**
        - **Sensor Mediated Tachycardia**
          - Eliminate sensor interference
      - **No response** → **Dual chamber system**
  - **Dual chamber system**
    - **AV sequential pacing during tachycardia**
      - **Timing Related Tachycardia**
        - Program DDD mode
    - **Atrial pacing during tachycardia**
      - **Orthodromic PCMT**
        - Disable PVC-SAS
    - **Ventricular pacing during tachycardia**
      - **Supra ventricular arrhythmia**
        - **Synchronization to SVA**
          - Program DDI(R) or VVI(R)
      - **VA conduction**
        - **YES**
          - **Antidromic PCMT**
            - Extend PVARP
        - **NO**
          - **Timing Related Tachycardia**
            - Eliminate artficial triggering
  - **Single chamber system**
    - **Timing Related Tachycardia**
      - Program inhibited mode
      - Extend refractory period

Fig. 7.20. Flow chart used for the determination of pacemaker tachycardia.

References.

1. Comer TP, Saxena N, and Salzman SH. The case of two-timing pacemaker. PACE 1981; 4: 465-466.

2. Wallace WA, Abelmann WA, and Norman JC. Runaway demand pacemaker. Ann Thorac. Surg. 1970; 9: 209-210.

3. Shettigar UR, Kleiger RE, Krone R, et al. Runaway pacemaker with demonstration of supernormal period of excitation. Chest 1974; 65: 227-231.

4. Harper R, Peter T, Hunt D, et al. Runaway pacemaker rhythm after 36 hours of electrical inactivity. Br. Heart J. 1974; 36: 610-614.

5. Nasrallah A, Hall RJ, Garcia E, et al. Runaway pacemaker in seven patients. J. Thorac. Cardiovasc. Surg. 1975; 69: 365-370.

6. Odabashian HC and Brown DF. Runaway in a modern generation pacemaker. PACE 1979; 2: 152-156.

7. Santini M, Messina G, Masini V. Intermittent and arrhythmic "runaway" pacemaker. PACE 1980; 3: 730-732.

8. Heller LI. Surgical electrocautery and the runaway pacemaker syndrome. PACE 1990; 13: 1084-1985.

9. Inoue H, Ueda K, Ohkawa S, et al. Runaway pacemaker. A case report with a runaway rate of 2100 ppm. PACE 1979; 608-613.

10. Van Gelder LM and El Gamal MIH. Externally induced irreversible runaway pacemaker. PACE 1981; 4: 578-581.

11. Lee RW, Huang SK, Mechling E, et al. Runaway atrioventricular sequential pacemaker after radiation therapy. Am. J. Med. 1985; 5: 883-886.

12. Bluhm G, Sundstedt CD. Runaway pacemaker inhibited by external overdrive stimulation: A case report. PACE 1984; 7: 440-442.

13. Toff WD, Leeks C, Joy M, et al. The effect of aircraft vibration on the function of an activity sensing pacemaker.(abstract) Br Heart J 1987; 57: 573.

14. Wilkoff BL, Shimokochi DD, Schaal SF. Pacing rate increase due to application of steady external pressure on an activity sensing pacemaker. PACE 1987; 10: 423. (Abstract)

15. Lau CP, Tse WS, Camm AJ.. Clinical experience with Sensolog 703: a new acivity sensing rate responsive pacemaker. PACE 1988; 11: 1444-1455.

16. Ruiter JH. Clinical aspects of cardiac pacing for bradyarrhythmias. Thesis, Rotterdam 1992, p.147.

17. Ahmed R, Gibbs S, Ingram A, et al. Pacemaker mediated tachycardia with left retromammary implantation of VVIR (activity) pacemaker.(abstract) PACE 1990; 13: 1189.

18. Res J. Personal communication,1993.

19. Lau CP, Ritche D, Butrous GS, et al. Rate modulation by arm movements of the respiratory dependent rate responsive pacemaker. PACE 1988; 11: 744-752.

20. Van Hemel NM, Hamerlijnck RPHM, Pronk KJ, et al. Upper rate limit ventricular stimulation in respiratory rate responsive pacing due to electrocautery. PACE 1989; 12: 1720-1723.

21. Van Gelder BM, Bracke FALE, El Gamal MIH. Upper rate pacing after radio frequency catheter ablation in a minute ventilation rate adaptive DDD pacemaker. PACE 1994; 17: 1437-1440.

22. Van Gelder LM, Bracke FALE, El Gamal MIH. ECG monitoring and minute ventilation rate adaptive pacemakers.(abstract) Cardiologie 1994; 1: 301.

23. Alt E, Völker R, Högl B, et al. Function of the temperature controlled Nova MR pacemaker in patient's everyday life: Preliminary clinical results.(abstract) PACE 1987; 10: 1206.

24. Lau CP. Sensors and pacemaker mediated tachycardias. PACE 1991; 14: 495-498.

25. Lau CP, Tai YT, Fong PC, et al. Pacemaker mediated tachycardias in single chamber rate responsive pacing. PACE 1990; 13: 1575-1579.

26. Winter UJ, Behrenbeck DW, Hoher M, et al. Problemen bei der Slop-Einstellung un der Frequenzanpassung in frequenz variabelen Schrittmachern: Oszillationsphanomene und plotzliche Frequenzhinbruche. Herzschrittmacher 1985; 5: 50-54.

27. Volosin KJ, O'Connor WH, Fabiszewski R, et al. Pacemaker mediated tachycardia from a single chamber temperature sensitive pacemaker. PACE 1989; 12: 1596-1599.

28. Wehr M, Schmitt CG, Peter JH. Irregular tachycardia in a patient with WPW syndrome and an A-V universal pacemaker. PACE 1984; 7: 320-324.

29. Den Dulk K, Lindemans FW, Bär FW, et al. Pacemaker related tachycardias. PACE 1982; 5: 476-485.

30. Schuilenburg RM. Patterns of V-A conduction in the human heart in the presence of normal and abnormal A-V conduction. In H.J.J. Wellens, K.I. Lie, M.J. Janse (Eds): The conduction system of the heart. Leiden, Stenfert Kroese BV, 1976, p. 485.

31. Bathen J, Gundersen T, Forfang K. Tachycardias related to atrial synchronous ventricular pacing. PACE 1982; 5: 471-475.

32. Furman S, Fisher JD. Endless loop tachycardia in an AV universal (DDD) pacemaker. PACE 1982; 5: 486-489.

33. Den Dulk K, Lindemans FW, Wellens HJJ. Management of pacemaker circus movement tachycardias. PACE 1984; 7: 346-355.

34. Barold SS, Falkoff MD, Ong LS, et al. Paradoxical induction of endless loop tachycardia by magnet application over a DDD pacemaker. PACE 1986; 9: 503-510.

35. Lamas GA, Antman EM. Pacemaker mediated tachycardia initiated by coincident P wave undersensing and ventricular blanking period. PACE 1985; 8: 436-439.

36. Van Mechelen R, Ruiter J, de Boer H, et al. Pacemaker electrocardiography of rate smoothing during DDD pacing. PACE 1985; 8: 684-690.

37. Van Gelder LM, El Gamal MIH. Magnet application, a cause of persistent arrhythmias in physiological pacemakers. Report of 2 cases. PACE 1982; 5: 710-714.

38. Den Dulk K, Lindemans FW, Wellens HJJ. Merits of various antipacemaker circus movement tachycardia features. PACE 1986; 9: 1055-1062.

39. Manual META DDDR; dual chamber rate responsive pacing system, model 1250.

40. De Jongste MJL, van Binsbergen EJ, Nagelkerke D, et al. Early experience with the Quintech 931 DDD pacemaker. In Perez Gomez F (Ed): Cardiac Pacing. Madrid, Editorial Grouz, 1985; 713.

41. Mcalister HF, Klementowicz PT, Calderon EM, et al. Atrial electrogram analysis: Antegrade versus retrograde. PACE 1988; 11: 1703-1707.

42. Timmis GC, Westveer DC, Bakalyar DM, et al. Discrimination of anterograde from retrograde atrial electrograms for physiologic pacing. PACE 1988; 11: 130-140.

43. Shandling AH, Castellanet MJ, Thomas LA, et al. Variation in P-wave amplitude immediately after pacemaker implantation: possible mechanism and implications for early programming. PACE 1989; 12: 1797-1805.

44. Fröhlig G, Blank W, Schwerdt H, et al. Atrial sensing performance of AV universal pacemakers during exercise. PACE 1988; 11: 47-60.

45. Den Dulk K, Lindemans FW, Wellens HJJ. Noninvasive evaluation of pacemaker circus movement tachycardias. Am. J. Cardiol. 1983; 53: 537-543.

46. Van Gelder LM, El Gamal MIH, Baker R, et al. Tachycardia termination algorithm: A valuable feature for interruption of pacemaker mediated tachycardia. PACE 1984; 7: 283-287.

47. Fontaine JM, Maloney JD, Castle LW, et al. Noninvasive assessment of ventriculo-atrial conduction and early experience with the tachycardia termination algorithm in pacemaker mediated tachycardia. PACE 1986; 9: 212-222.

48. Calfee RV. Pacemaker mediated tachycardia: engineering solutions. PACE 1988; 11: 1917-1928.

49. Duncan JL, Clark MF. Prevention and termination of pacemaker mediated tachycardia in a new DDD pacing system (Siemens-Pacesetter Model 2010 T). PACE 1988; 11: 1679-1683.

50. Franklin JO, Blake K, Kristall R. Pacemaker mediated tachycardia slower than the maximum tracking rate: A new termination algorithm. (abstract) PACE 1989; 12: 1179.

51. Ausubel K, Gabry MD, Klementowicz PT. Pacemaker mediated endless loop tachyacrdia at rates below the upper rate limit. Am. J. Cardiol. 1988; 61: 465-467.

52. Lamaison D, Girodo S, Limousin M. A new algorithm for a high level of protection against pacemaker mediated tachycardia. PACE 1988; 11: 1715-1721.

53. Limousin M, Bonnet JL, and the investigators of the multicenter study. A new algorithm to solve endless loop tachycardia in DDD pacing: A multi-center study in 91 patients. PACE 1990; 13: 867-874.

54. Nitzsché R, Gueunoun M, Lamaison D, et al. Endless-loop tachycardias: Description and first clinical results of a fully automatic protection algorithm. PACE 1990; 13: 1711-1718.

55. Den Heijer P, Crijns HJGM, van Binsbergen EJ, et al. Orthodromic pacemaker circus movement tachycardia. PACE 1987; 10: 955-957.

56. Von Knorre GH, Ismer B, Voss W, et al. Orthodromic and antidromic pacemaker circus movement tachycardia. PACE 1991; 14: 1233-1238.

57. Taniguchi K, Takaoka T, Fujiwara H, et al. Clinical experiences and experimental studies of false sensing with demand pacemakers. In Cardiac pacing. Watanabe, Y. (Ed). Amsterdam, Excerpta Medica, 1977, pp.333-339.

58. Dodinot B, Medeiros P, Galvao S, et al. Single chamber endless loop tachycardia; A possible complication of AAT pacing.(abstract) PACE 1985; 8: A-19.

59. Van Gelder LM, El Gamal MIH, Bracke FALE, et al. Pacemaker reentry tachycardia in a single chamber pacemaker.(abstract). Neth J Cardiol 1991; 2: 67.

60. Stroobandt R, Vandenbulcke F, Falleyn H, et al. Voltage dip in pacemaker battery supply: A new cause of pacemaker mediated tachycardia. PACE 1993; 16: 806-811.

61. Dodinot B. PMT (DDD)-PM arrest (AAI)-related to "atrial hypersensitivity caused by battery depletion". Stimucoeur 1991; 19: 249.

62. Reineman H. Vitatron Medical BV. Personal communication, september 1993.

63. Schüller H, Brandt J, Fahraeus. QRS sensing via atrial lead in AAI and DDD pacing.(abstract) PACE 1985; 8: A-95.

64. Shandling AH, Castellanet MJ, Messenger JC, et al. Utility of the atrial endocardial electrogram concurrent with dual-chamber pacing in the determination of a pacemaker mediated arrhythmia. PACE 1988; 11: 1419-1425.

65. Johnson CD. Atrial synchronous ventricular inhibited (VDD) pacemaker mediated arrhythmia due to atrial undersensing and atrial lead oversensing of far-field ventricular afterpotentials or paced beats: Crosstalk. PACE 1986; 9: 710-719.

66. Levander-Lindgren M, Pehrsson SK. Occurrence and significance of arrhythmias associated with atrial triggered ventricular pacing. PACE 1984; 7: 628-639.

67. Pimenta J, Soldá R, Pereira CB. Tachycardia mediated by an AV universal (DDD) pacemaker triggered by a ventricular depolarization. PACE 1986; 9: 105-107.

68. Van Gelder LM, El Gamal MIH. Myopotential interference inducing pacemaker tachycardia in a DVI programmed pacemaker. PACE 1984; 7: 970-972.

69. Byrd CL, Schwartz SJ, Gonzales M, et al. DDD pacemakers maximize hemodynamic benefits and minimize complications for most patients. PACE 1988; 11: 1911-1916.

70. Gross J, Moser S, Benedek M, et al. Clinical predictors and natural history of atrial fibrillation in patients with DDD pacemakers. PACE 1990; 13: 1828-1831.

71. Kerr CR, Mason MA. Amplitude of atrial electrical activity during sinus rhythm and during atrial flutter-fibrillation. PACE 1985; 8: 348-355.

72. Pitney MR, May CD, Davis MJ. Undesirable mode switching with a dual chamber rate responsive pacemaker. PACE 1993; 16: 729-737.

73. Lau CP, Tai YT, Li JPS, et al. Initial clinical experience with a single pass VDDR pacing system. PACE 1992; 15: 1894-1900.

74. Furman S, Cooper JA. Atrial fibrillation during AV sequential pacing. PACE 1982; 5: 133-135.

75. Josephson ME, Seides SF. Clinical cardiac electrophysiology. Techniques and interpretations. Philadelphia, Lea & Febiger, 1979, pp.202-205.

76. Luceri RM, Castellanos A, Thurer RJ, et al. Noninvasive conversion of atrial flutter using a multiprogrammable DDD pulse generator. PACE 1986; 9: 137-140.

Chapter VIII. Troubleshooting Flow Chart

Introduction

For the analysis of pacemaker function or malfunction a systematical stepwise approach should be used. To analyze a pacemaker problem, the problem first should be identified. After identification a strategy for the problem solution should be described. For the description of the identification and the stepwise strategy a flow diagram or flow chart can be used. The flow diagram describes the steps in decision making and it reflects the rationale of the reasoning process.

In the design of a flow chart for the diagnosis of pacemaker malfunction or apparent malfunction it appears that there are a large number of possibilities that have to be confirmed or excluded before the final diagnosis can be made. If a flow chart is designed as a decision tree, it rapidly grows to a size unsuitable for book publishing and impractical for the ECG interpreter. This was one of the main reasons for incorporating the knowledge used for the analysis of the pacemaker ECG in an expert system, which is described in chapter IX.

In the draft of the flow chart we will use a schematic presentation of the pacemaker ECG illustrating malfunction, followed by the steps used for the analysis. In these steps smaller, but surveyable decision trees can be used. It needs to be easy for the interpreter to find the schematic ECG, corresponding with the actual ECG. This approach links up best with the practical situation.

For the analysis the following assumptions are made:

1)   The interpreter knows the specifications of the pulse generator and the programmed pacing mode.
2)   Lead impedance can be measured by pacemaker telemetry or pulse waveform analysis.
3)   Depolarization following stimulation can be established on the ECG.

In the flow chart the motivation for the queries will be omitted or only indicated by key words. For the sake of compactnesss, the same will be done for the answers and the final diagnosis. Explanation of the phenomenon or diagnosis will be referred to the corresponding chapter, paragraph and figure if applicable. If two or more conditions are mentioned they all have to be fulfilled, unless otherwise stated.

The flow chart is divided into the following electrocardiographic manifestations of pacemaker malfunction:

Single chamber systems
8.1.1 Continuous no output
8.1.2 Intermittent no output
8.1.3 Continuous noncapture
8.1.4 Intermittent noncapture
8.2.1 Oversensing; fixed prolongation escape interval
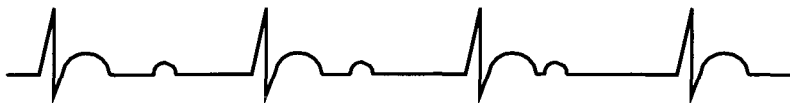8.2.2 Oversensing; varying prolongation escape interval
8.3   Undersensing

Dual chamber systems
8.4   Atrial oversensing

8.1.1 Continuous no output (single chamber system)

ECG : No pacemaker stimuli visible.

Conditions:
1) RR interval > escape or hysteresis interval
2) No pacemaker stimuli after magnet application (12 lead ECG and supplementary modified chest leads). If magnet application results in pacemaker stimuli proceed to oversensing.

Communication by telemetry:
Not possible → pulse generator failure.
Check lead impedance during replacement to exclude lead malfunction (low impedance) as the cause of generator failure (battery depletion).

Still possible → measure lead impedance.

High lead impedance (> 2000 Ohms)
- conductor break
- improper lead connection
- air entrapment, unipolar system (chapter IV, fig. 4.55).

If manipulation results in pacemaker stimuli on the ECG, the galvanic discontinuity is located between pulse generator and venous entry.
Use fluoroscopy to determine the site of discontinuity.
Program a bipolar system in the unipolar configuration, if available. If no effect, discontinuity is located in the cathodal conductor or its connection. If pacemaker stimuli are observed, discontinuity is located in the anodal conductor or its connection.
Recurrence of pacemaker stimuli after application of slight pressure on the generator indicates air entrapment. This problem is only observed in unipolar systems shortly after surgical procedures.

Low lead impedance (< 100 Ohms)
- short circuit, insulation defect
In unipolar systems located between pulse generator and cathodal conductor (chapter IV, fig. 4.56).
In bipolar systems located between anodal and cathodal conductor. If programmable in unipolar configuration, stimulation can be restored; increased stimulation threshold, increased current drain → accelerated battery depletion !

8.1.2 Intermittent no output (single chamber system)

ECG: Intermittent pacemaker stimuli.
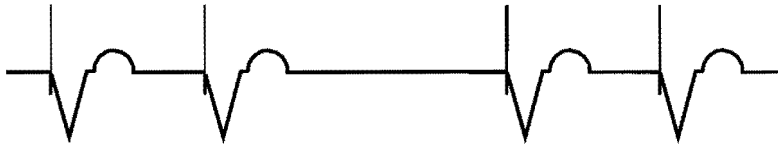
Conditions:
1) Prolonged interval is an exact multiple of the escape interval (chapter V, fig. 5.1).
2) Not affected by magnet application
If condition 1 or 2 is not fulfilled, proceed to oversensing 8.2.1, 8.2.2.

Measure lead impedance
Repetitive measurements might be necessary to determine lead impedance during no output.

High lead impedance (> 2000 Ohms)
- conductor break
- improper lead connection

If manipulation results in aggravation of the problem or restoration of continuous pacing, the galvanic discontinuity is located between pulse generator and venous entry.
Use fluoroscopy to determine the site of discontinuity.
Program a bipolar system in the unipolar configuration, if available. If no effect, discontinuity is located in the cathodal conductor or its connection. If continuous pacing is observed, discontinuity is located in the anodal conductor or its connection.
Restoration of regular stimulation after application of slight pressure on the pulse generator indicates air entrapment. This problem is only observed in unipolar systems shortly after surgical procedures.

Low lead impedance (< 100 Ohms)
- short circuit, insulation defect
In unipolar systems located between pulse generator and cathodal conductor.
In bipolar systems located between anodal and cathodal conductor. If programmable in unipolar configuration stimulation can be restored; increased stimulation threshold, increased current drain → accelerated battery depletion !

8.1.3 Continuous noncapture (single chamber system)

ECG: Pacemaker stimuli not followed by depolarization
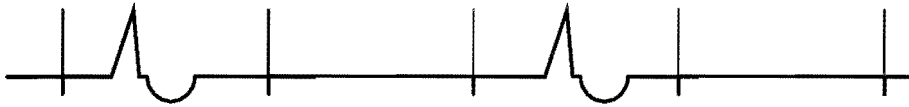
Conditions:
1) Myocardium is not refractory at the time of pulse delivery.
2) Proper output setting, not inadvertently programmed below stimulation threshold.
3) Battery depletion excluded.

Additional observations
1) Sensing from a chamber other than the original implantation site → macrodislocation.
2) Sudden onset of diaphragmatic stimulation → lead displacement or perforation.
If none of these two observations are present:

Measure lead impedance

Lead impedance within normal limits
- Administration of threshold elevating drugs ?
- Electrolyte imbalance ?
- Myocardial infarction ?
- Obsevred immediately after DC shock ? (Chapter IV, fig.4.63)
- Cardioplegia during open heart surgery ?
If none of above mentioned causes applicable :
Fluoroscopy for lead displacement or perforation. Usually shortly after implantation.
If none of above mentioned causes applicable → exit block.

High lead impedance
- conductor break (fluid bridge between both ends).
- improper lead connection.
If manipulation results in (intermittent) capture, the defect is located between pulse generator and venous entry. Use fluoroscopy to determine the site of the defect.
Program a bipolar system in the unipolar mode, if available. If no effect, the defect is located in the cathodal conductor or its connection. If capture is restored, defect is located in the anodal conductor or its connection.

Low lead impedance
- insulation defect
In unipolar systems located between pulse generator and cathodal conductor. In bipolar systems located between anodal and cathodal conductor. If programmable in unipolar configuration, capture can be restored; increased stimulation threshold, increased current drain → accelerated battery depletion !

8.1.4 Intermittent noncapture (single chamber system)

ECG : Pacemaker stimuli intermittently followed by depolarization
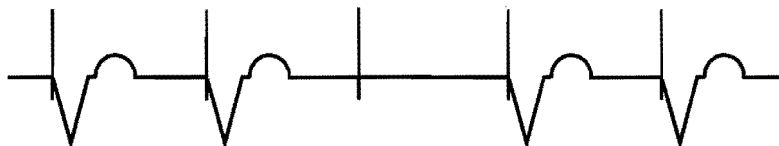
Conditions:
1) Myocardium is not refractory at the time of pulse delivery.
2) Appropriate output setting, not inadvertently at the level of the stimulation threshold.
3) Battery depletion excluded.

Additional observations
1) Changes in the morphology of the stimulated depolarization in comparison with previous recordings → lead displacement or perforation.
2) Attenuation of the pacemaker stimulus during noncapture → intermittent galvanic discontinuity (high lead impedance) or insulation defect (low lead impedance).
If none of these two observations are present :

Measure lead impedance
Repetetive measurements might be necessary to determine lead impedance during noncapture.

Lead impedance within normal limits
- Administration of threshold elevating drugs ?
- Electrolyte imbalance ?
- Myocardial infarction ?
- Observed immediately after DC shock ?
If none of above mentioned causes applicable:
Fluoroscopy for lead displacement. Usually shortly after implantation.
If none of above mentioned causes applicable → fibrous tissue formation around stimulation electrode. (Chapter IV, fig. 4.62)

High lead impedance
- conductor break with intermittent contact and fluid bridge between both ends.
- improper lead connection
If manipulation results in aggravation of the problem or restoration of continous capture, the defect is located between pulse generator and venous entry (chapter IV, fig. 4.48).
Program a bipolar system in the unipolar configuration, if available. If no effect, the defect is located in the cathodal conductor or its connection. If continuous capture is restored, defect is located in the anodal conductor or its connection.

Low lead impedance

- insulation defect

In unipolar systems located between pulse generator and cathodal conductor (chapter IV, fig. 4.56).

In bipolar system located between anodal and cathodal conductor. If programmable in unipolar configuration, stimulation can be restored; increased stimulation threshold, increased current drain → accelerated battery depletion !

Note

Intrinsic events following a noncaptured stimulus within the refractory period will show apparent undersensing.

8.2.1 Oversensing (single chamber system).

ECG: More or less fixed prolongation of the escape interval.
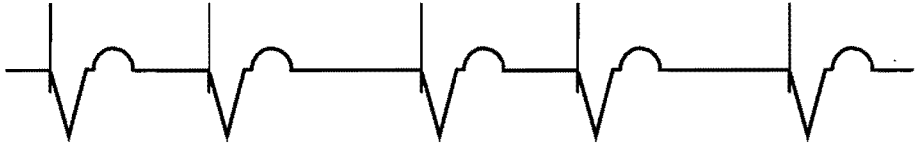
Conditions:
1) Prolonged interval is not a multiple of the escape interval
2) Prolonged interval after intrinsic depolarization > hysteresis interval
3) Magnet application results in regular asynchronous pacing at a preset interval. If there is no effect then irregular pulse formation is concluded; pulse generator failure. Unlikely in currently available pulse generators. (Chapter V, fig. 5.2)
4) EMI from the patient's environment is excluded as the cause of oversensing.

Additional observation
More or less fixed prolongation of the escape interval, but shorter than twice the escape interval → prolongation of escape interval is related to stimulation or cardiac response to stimulation (Chapter V, fig. 5.3).

Intracardiac Electrogram (IEGM)
The IEGM will elucidate the nature of signals responsible for oversensing.
- T wave sensing (VVI pacemaker) Chapter V, fig. 5.6
- R wave sensing (AAI pacemaker) Chapter V, fig. 5.7
- Afterpotential sensing
- Spurious signals, caused by abrupt changes in lead impedance, provoked by atrial or ventricular contraction; Lead malfunction. (Chapter V, fig. 5.23). A second (abandoned) lead touching the functioning lead may evoke these signals. Fluoroscopy.

No IEGM available → Measure lead impedance

Lead impedance within normal limits
- T wave sensing (VVI pacemaker)
- R wave sensing (AAI pacemaker)
- Afterpotential sensing
Afterpotential sensing can be confirmed by normalizing the escape interval after reducing pacemaker output or by shortening the prolonged escape interval by reducing the refractory period.
Oversensing can be corrected by reducing sensitivity or extending the refractory period.
High or low lead impedance
Impedance changes are caused by either a conductor defect (high lead impedance) or insulation defect (low lead impedance) but provoked by atrial or ventricular contraction (Chapter I, fig. 1.6). Differentiation between insulation and conductor defect has no practical value.

8.2.2 Oversensing (single chamber system).

ECG: Varying prolongation of the escape interval

Conditions:
1) Prolonged interval after intrinsic depolarization > hysteresis interval
2) Magnet application results in regular asynchronous pacing at a preset interval. If no effect, irregular pulse formation (pulse generator failure). Unlikely in currently available pulse generators.

Intracardiac Electrogram (IEGM)
The IEGM will elucidate the nature of signals responsible for oversensing.
- P wave sensing (VVI pacemaker), Chapter V, fig. 5.9
- Spurious signals, caused by abrupt changes in lead impedance; lead malfunction (Chapter V, fig. 5.17.) or by intermittent contact of the functioning lead with an abandoned lead. Fluoroscopy.

Marker channel
If sense markers coincide with P waves (VVI pacemaker) → P wave oversensing (Chapter V, fig. 5.11).
Sense markers randomly distributed over cardiac cycle → spurious signals, lead malfunction. Intermittent contact abandoned lead.

Triggered mode
If neither IEGM nor marker channel is available program to triggered mode. P-synchronous pacing → P wave sensing. Pacing with a varying interval → spurious signals.

Measure lead impedance

High lead impedance
- Intermittent contact in conductor
- Unstable lead connection
If manipulation results in aggravation of oversensing, the defect is located between pulse generator and venous entry. If a bipolar system is programmed in the unipolar configuration with abolition of oversensing, the defect is located in the anodal conductor.

Low lead impedance

- insulation defect

In bipolar systems located between anodal and cathodal conductor (Chapter V, fig. 5.23). Insulation defect in the outer insulation will be indicated by "unipolarization" of the pacemaker stimulus (Chapter III, fig. 3.11). In unipolar systems, chest wall stimulation can be used to confirm and to localize the insulation defect.

Note

If an event caused by oversensing preceeds an intrinsic event with a coupling interval shorter than the refractory period, the ECG may show undersensing.

8.3 Undersensing (single chamber system).

ECG: Interval between an intrinsic event and subsequent paced event is shorter than the escape interval.

Conditions:
1) The unsensed intrinsic depolarization occurs outside the refractory period initiated by a paced event.
2) Pacemaker is not in the asynchronous mode by magnet application or inadvertently programmed in the asynchronous mode.

Exclude oversensing (Chapter V, fig. 5.29.)
1) Marker channel recording
2) Triggered mode, if no marker channel available
If oversensing is present proceed to oversensing 8.2.2.

Measure lead impedance

Lead impedance within normal limits
- Pacemaker not inhibited by chest wall stimuli → pulse generator failure
- Morphology of the stimulated depolarization is changed compared with previous recordings → electrode displacement
- Morphology of the stimulated depolarization is not changed compared with previous recordings → fibrous tissue formation; increased stimulation threshold.
Exclude changes in medication, electrolyte disturbances and myocardial infarction as the cause of undersensing.

High lead impedance
- Conductor failure with intact insulation. Always associated with an increase in stimulation threshold. For unipolar systems use fluoroscopy to determine the site of the conductor failure.
Program a bipolar system in the unipolar configuration, if available. If lead impedance returns to normal the defect is located in the anodal conductor, if not it is located in the cathodal conductor.

Low lead impedance
- insulation defect
In unipolar systems, chest wall stimulation can be used to confirm and localize the insulation defect.
In bipolar systems the insulation defect is located between anodal and cathodal conductor.

8.4 Atrial oversensing (dual chamber system).

ECG: Ventricular paced event not preceded by a P wave.
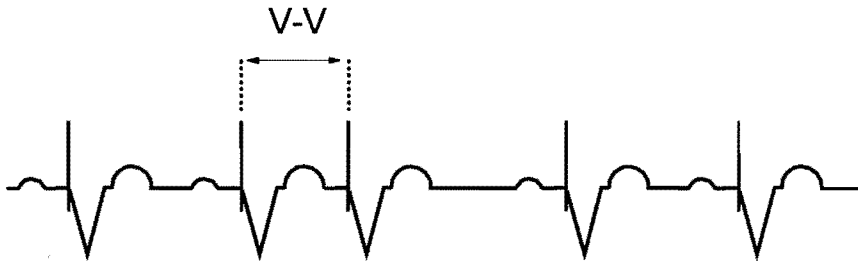
Conditions:
1) No atrial depolarization is preceding Vpace. Use precordial leads or the intracardiac electrogram, if available, to exclude atrial premature contraction.(Chapter VI, fig. 6.43)
2) The V-V interval is shorter than the lower rate interval. If the V-V interval is equal to the lower rate interval and condition 1 is fulfilled → atrial no output.
Proceed to 8.1.2 for the atrial channel.

Sensing of far-field ventricular activity ?
1) Confirm the presence of far-field ventricular activity detected at the atrial electrode by recording of the intracardiac electrogram and/or the marker channel.
2) If neither of these two options is available, program PVARP to an interval longer than the total duration of ventricular depolarization. If oversensing disappears, far-field sensing of ventricular depolarization can be concluded.

Sensing of far-field ventricular activity excluded → proceed to oversensing 8.2.2 for the atrial channel.

8.5 Atrial undersensing (dual chamber system)

ECG: P wave not followed by inhibition of the atrial stimulus and ventricular stimulation at the preset AV interval.
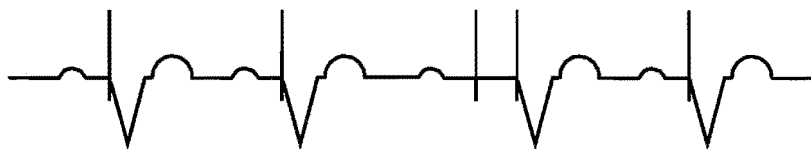
Conditions:
1) P waves occur outside PVARP or extended PVARP
2) Pacemaker is not in the asynchronous mode by magnet application, back-up mode pacing or inadvertently programmed in the asynchronous mode

Exclude ventricular oversensing
Ventricular oversensing initiates or prolongs PVARP, which may result in an ECG showing atrial undersensing. Chapter I, fig. 1.5.

Ventricular oversensing excluded by
1) Marker channel recording
2) If not available, program to the VVI mode. Normal escape interval excludes ventricular oversensing.

If ventricular oversensing is present proceed to 8.2.1 or 8.2.2 for the ventricular channel.

If ventricular oversensing is excluded proceed to 8.3 for the atrial channel.

Note
1) The ECG may show atrial noncapture due to atrial stimulation during the refractory period of the atrial myocardium.

2) Atrial undersensing may be overlooked if the RR interval is shorter than the programmed VA interval ! (Chapter VI, figs. 6.36- 6.37).

8.6.1 Ventricular oversensing; ventricular safety pacing.

ECG: Shortening of the Apace-Vpace interval.

Conditions:
1) Ventricular safety pacing present for crosstalk protection
2) Atrial stimulation not followed by intrinsic ventricular activity
3) The programmed AV interval is longer than 110 ms

Program the pacing system in the VVI mode
If the escape interval remains constant and is equal to the preset lower rate interval, oversensing is caused by atrial stimulation and crosstalk can be concluded. The ventricular safety pace mechanism is activated by crosstalk resulting in shortening of the Apace-Vpace interval. Ventricular safety pacing is characterized by an AV interval between 100 and 110 ms (Chapter VI, figs. 6.23-6.24).

If the escape interval shows prolongation or is longer than the programmed lower rate interval oversensing in the ventricular channel is concluded. Proceed to 8.2.1 or 8.2.2 for the ventricular channel.

Prevention of crosstalk
1) Reduce atrial output (pulse amplitude or pulse width).
2) Reduce ventricular sensitivity.
3) Program bipolar electrode configuration, if available.*
4) Extend the ventricular blanking period.

*Note
In some pulse generators (Intermedics, Inc.) crosstalk can be aggravated when programming the atrial lead configuration from unipolar to bipolar (Chapter VI 6.5.1).

8.6.2 Ventricular oversensing during AV interval outside ventricular safety pace interval.

ECG: Atrial stimulus not followed by a ventricular stimulus at the preset AV interval.

Conditions:
1) Ventricular safety pacing present for crosstalk protection
2) Interval between atrial stimulus and intrinsic ventricular activity (if present) is longer than the programmed AV interval

**Ventricular safety pacing absent or not activated**
Exclude crosstalk, see 8.6.1.

Proceed to oversensing 8.2.1 or 8.2.2 for the ventricular channel.

Note:
Ventricular oversensing during the AV interval initiates a ventricular refractory period. Intrinsic events occurring during this refractory period may give rise to an ECG pattern of ventricular undersensing.

8.6.3 Ventricular oversensing during VA interval.

ECG: Prolongation of the VA interval
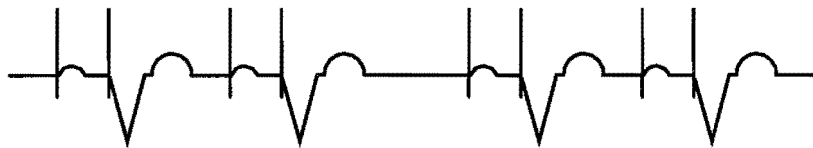
If the prolongation of the VA interval is a more or less fixed interval proceed to 8.2.1 for the ventricular channel.

If the prolongation of the VA interval is a varying interval proceed to 8.2.2 for the ventricular channel.

Note
1) Ventricular oversensing can be masked if the interval between two consecutive atrial sense-ventricular paced events is shorter than the VA interval (Chapter VI, fig.6.46). If this condition exists, record the marker channel and if not available program the system to the VVI mode to exclude oversensing.

2) Ventricular oversensing may also give rise to a pattern of atrial undersensing !
Chapter VI, fig. 6.41.

8.7.1 Ventricular undersensing during the AV interval initiated by atrial stimulation (premature beat, shortening AV interval).

ECG: Intrinsic ventricular depolarization during the AV interval followed by a ventricular stimulus with shortening of the AV interval.

Conditions:
1) Intrinsic ventricular depolarization is a premature ventricular contraction
2) Intrinsic ventricular depolarization is preceded by an atrial stimulus
3) AV interval is shortened to 100-110 ms.

Conclusion
Premature ventricular contraction occurs during the ventricular safety pacing interval resulting in shortening of the AV interval and an ECG pattern of ventricular undersensing. Normal pacemaker function (chapter VI, fig. 6.27).

8.7.2 Ventricular undersensing during the AV interval initiated by atrial stimulation (premature beat, normal AV interval).

ECG: Intrinsic ventricular depolarization during the AV interval followed by a ventricular stimulus at the preset AV interval.

Conditions:
1) Intrinsic ventricular depolarization is a ventricular premature contraction
2) Intrinsic ventricular depolarization is preceded by an atrial stimulus
3) AV interval is unchanged

Exclude real ventricular undersensing
Exclude real ventricular undersensing by programming the system to the VVI or VVT mode to verify proper sensing of ventricular premature contractions.
If real ventricular undersensing is present, proceed to 8.3 for the ventricular channel.

Conclusion
Premature ventricular contraction occurs during the ventricular blanking period resulting in an ECG pattern of ventricular undersensing (Chapter VI, fig. 6.26). Although this is normal pacemaker function, programming a long AV interval should be avoided under these conditions (Chapter VI, 6.7).

8.7.3   Ventricular undersensing during the AV interval initiated by atrial stimulation (conducted beat).

ECG: Intrinsic conducted beat preceded by atrial stimulation and followed by a ventricular stimulus with a shortened or unchanged AV interval.
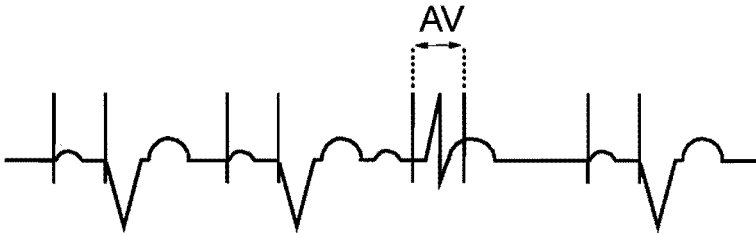
Conditions:
1) Intrinsic ventricular depolarization is a conducted beat.
2) Intrinsic ventricular depolarization is preceded by an atrial stimulus
3) AV interval is unchanged or shortened to 100-110 ms

Conclusion
Ventricular depolarization is not sensed because it occurs during the ventricular blanking period initiated by the atrial stimulus (AV interval unchanged).
Ventricular stimulus is evoked by the ventricular depolarization occurring in the ventricular safety pacing interval (AV interval shortened).
In both cases the primary cause of the ventricular stimulus following intrinsic depolarization is failure to sense the intrinsic P wave.
Proceed to undersensing 8.3 for the atrial channel.

8.7.4 Ventricular undersensing during the VA interval terminated by atrial pacing.

ECG: Interval between intrinsic ventricular event and subsequent paced atrial event shorter than the programmed VA interval.

Conditions:
1) Intrinsic ventricular depolarization occurs outside the ventricular refractory period initiated by the preceding ventricular paced event.
2) Pacemaker is not in the asynchronous mode by magnet application, back-up mode pacing or inadvertently programmed in the asynchronous mode.

Proceed to undersensing 8.3 for the ventricular channel.

8.7.5 Ventricular undersensing during the VA interval terminated by atrial sensing

ECG: Intrinsic ventricular depolarization followed by atrial sensing-ventricular pacing.
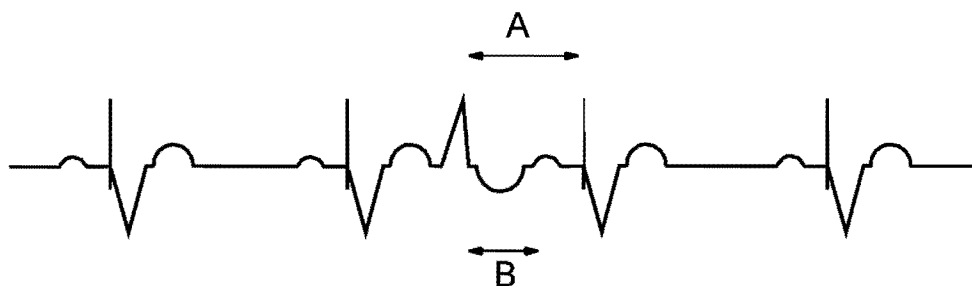
Condition:
Intrinsic ventricular activity occurs outside the ventricular refractory period initiated by the preceding ventricular paced event.

Ventricular undersensing is present, if

1) The coupling interval (A) between the intrinsic ventricular depolarization and subsequent ventricular pace event is shorther than the upper rate interval.

or

2) The coupling interval (B) between the intrinsic ventricular depolarization and the subsequent atrial depolarization is shorter than the (extended) PVARP.

If one of both conditions is fulfilled proceed to 8.3 for the ventricular channel.

If none of the two conditions is fulfilled -> normal pacemaker function.

In case of doubt, program the system to the VVT mode, which confirms presence or absence of ventricular sensing.

8.8 Summary and conclusions

A flow chart or flow diagram used for the analysis of pacemaker function and malfunction as presented in this chapter has limitations. In the first place, it gives only the main lines of identification and the strategy for problem solution. Completeness will be difficult to obtain. The rationale for the reasoning process and the explanation of the physical background for some phenomena cannot be incorporated in this flowchart. To overcome this latter problem a number of references to the text of the previous chapters are made.

Secondly the flow chart is based on the information compiled in the preceding chapters. This information is based on personal experience of the author combined with reports from the literature. This implies that the reasoning process is based on this experience and that conflicts in the reasoning process will be hard to verify from the flow chart. The application of the flow chart has not been checked by other interpreters.

To prevent the previous mentioned limitations of the flow chart and to evaluate correctness of the reasoning process, the flow chart is implemented in an expert system. After formalization of the expert knowledge and testing of the expert system the answer to these questions may be found.

Chapter IX. **A Prototype Expert System**

Introduction
Although the troubleshooting flow chart described in the previous chapter can be very helpful in the practical solution of pacemaker problems, the rationale for decision making is not explained. The knowledge used for decision making, based on empirical associative knowledge -heuristics- of the author is implemented in a prototype expert system developed in collaboration with the Eindhoven University of Technology [1]. Application of an expert system offers the possibility of justification and explanation of the solution of a problem. In case of a correct implementation it is also a guidance to the expert concerning the completeness and consistency of his/her knowledge. After clinical evaluation and validation, an expert system can be a valuable tool in the pacemaker follow-up clinic and is also useful for educational purposes.

An expert system is a computer program representing specific knowledge. This knowledge is used in a reasoning process to provide a solution or advise on a problem [2]. Expert systems are a spin-off of the research in the field of artificial intelligence.

9.1 Distinction between expert systems and conventional programs
There are a number of differences between an expert system and conventional programs:

1) The expert system simulates human reasoning in a small but wel defined domain, instead of simulating the domain itself as in a mathematical model.

2) Reasoning performed by the expert system is based on human knowledge, which makes the process symbolic. This in contrast with the usually purely numeric process in conventional systems.

3) In expert systems, in general, the "knowledge base" (the part of the system that contains a representation of the domain knowledge) is separate from the "inference engine" (the part of the system that performs the reasoning). The advantage is that both parts can be upgraded independently.

4) Problem solving in expert systems uses heuristic methods, which means the use of rules of thumb, that contain knowledge about solutions for problems in a particular domain. Heuristics are used if finding a solution is time consuming or cannot be solved in an algorithmic way. A heuristic method gives an acceptable, often practical solution, whereas an algoritmic method gives a nondisputable answer.

5) The expert system offers limited possibilities of justification and explanation of the solution and the reasoning process leading to the solution.

The more general expression "knowledge based system" is also used to describe an expert system. A knowledge based system employs heuristics and consists of a knowledge base and an inference engine. If the heuristics employed by a knowledge based system are based on the knowledge of one or more domain experts the system is called an expert system. The knowledge based system described in this chapter is therefore called an expert system.

9.2 Construction of an expert system
For the development of the expert system a heuristic approach, based on the empirical associative knowledge of a domain expert, is chosen for the following reasons:

1)   Expert systems may offer tests for logical completeness and consistency of the expert knowledge in the case of a correct implementation.
2)   Detailed expert knowledge is described in this thesis.
3)   The final version of the expert system contains the knowledge and experience of the expert. The reasoning process is based on a practical approach, which makes it easy to understand for other users and has an educational character.
4)   For a model based approach it would be difficult to make a good model description due to the numereous elements of such a system (pulse generator, lead, lead connection, electrode, electrode-myocardial interface, myocardium) and to verify or to exclude the numerous hypotheses generated by such a complex system.
5)   If a model based approach was employed the heuristics used in a previous expert system could be used to limit the number of hypotheses generated by a model based approach.

Development of an expert system is also called "knowledge engineering" and belongs to the professional area of the "knowledge engineer". Knowledge acquisition is defined as: "the transfer and transformation of potential problem-solving expertise from some knowledge source to a program" [2].
The development of an expert system consists of 5 phases:
1) Identification
In this phase the problem should be identified, which means that the problem should be properly described and the requirements for the solution should be defined.
2) Conceptualization
In this phase the essential elements and their relationships, which are used for the problem solution, are exposed.
3) Formalization
In this phase the underlying strategy used for the problem solution is explained. The problem-solving knowledge is formalized which means that it is transformed into a formal structure used for the implementation of the knowledge.
4) Implementation
In this phase the formalized problem-solving knowledge is transformed into a program, employing a programming language used in artificial intelligence or an expert system shell.
5) Testing
In this phase the expert system is tested and evaluated.

In newer expert system development theories, more attention is paid to the analysis of knowledge. This results in a knowledge model, which will be transferred into a design model in which knowledge will be implemented. Knowledge based systems Analysis and Design Support (KADS) is a system employing this methodology [3,4]. In KADS knowledge analysis is called expertise analysis. In this phase an expertise model, also called conceptual model or knowledge model is created. This knowledge model should be created in such a way that the approach to problem-solving is similar to the approach of the expert. KADS offers a frame for analysis and modelling of knowledge. During knowledge acquisition the frame will be "filled in", thus creating a knowledge model.

During the development of our expert system we used the KADS approach. The phase of conceptualization is replaced by an extensive phase of knowledge analysis. Almost simultaneously with this phase, conceptualization and analysis of the problem-solving knowledge is incorporated. During this phase the strategy, task description and inference structure are identified. This phase results in a knowledge model showing similarity to the problem-solving approach of the expert.

In contrast to the formalization phase as described by Buchanan et al.[2] no further knowledge analysis is performed in the formalization phase, but only a transformation into a formal structure.

One of the aids used in KADS is the identification of generic task models used for the problem-solving process. The task models contain a domain independent description of the knowledge necessary to generate class of similar tasks. With the task models a frame or outline for strategic, task and inference knowledge can be constructed. This approach based on a direct knowledge analysis provides a good insight into the problem-solving knowledge and the inference structure of the system. If also offers some control over the knowledge acquisition process. This set up leads to a clearly structured knowledge base.
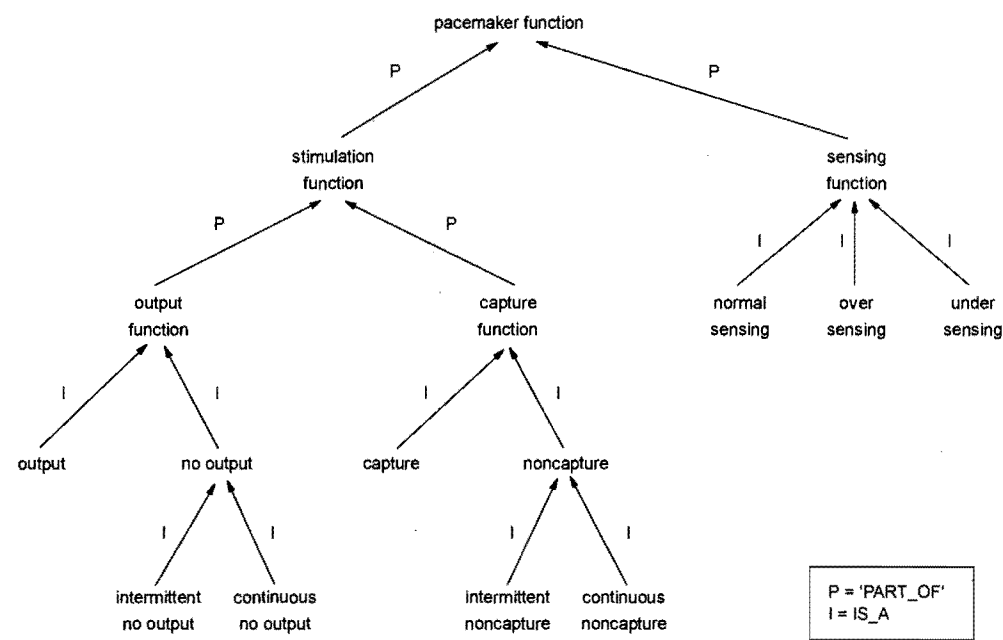
Fig. 9.1. Diagram of the elements of pacemaker function and malfunction.

9.2.1 Analysis of the expert knowledge

The purpose of the expert system is to support the ECG interpreter during the follow-up of pacemaker patients. In case of pacemaker malfunction or apparent malfunction the system supports the ECG interpreter in making the diagnosis or a number of differential diagnoses. It can also advise progression to additional investigations e.g. fluoroscopy.

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To begin with pacemaker functions and their corresponding states of normal function and malfunction have to be identified. An overview of pacemaker function and possible corresponding states per function are represented in figure 9.1.

Pacemaker functioning can be divided into:

1) Output function
2) Capture function
3) Sensing function

The system is limited to single chamber pacemakers and the approach is similar to that of the flow chart in the previous chapter. If during one of the analyses of pacemaker function malfunction is identified, the analysis process will be followed by a diagnostic process. As explained in figure 9.1, the diagnostic process refers to the following manifestations of malfunction :

no output (continuous or intermittent), noncapture (continuous or intermittent), over and undersensing.

Figure 9.2 illustrates the general problem solving strategy. In this diagram the tasks are marked within a rectangular field. The diamond shaped figures and the different descending paths are used to denote plural selections following the analysis tasks. These selections are determined by the result of the analysis. The foundation for this diagram was a thorough analysis of the steps in which the expert judges pacemaker function.

As first identification of the expert reasoning process during the analysis tasks, we used the generic task model "heuristic classification". Heuristic classification is characterized by the heuristic match between data abstractions and solution abstractions, as described by Tansley and Hayball [4].

In the analysis of output function, the first analysis is whether the pulse generator is able to emit a pacemaker stimulus e.g. is the stimulation rate higher than the intrinsic rate. If this criterium is fulfilled pacemaker stimuli should be visible on the ECG. If pacemaker stimuli are still absent the cause of malfunction should be identified.

In the analysis of capture function, effectiveness of the pacemaker stimulus is evaluated; does the pacemaker stimulus give rise to depolarization of the myocardium (capture). Essential to the evaluation of capture function is recognition of the pacemaker stimulus and the subsequent depolarization on the ECG.

In the analysis of sensing function, the timing of the pacemaker stimulus is evaluated. Essential to the evaluation of sensing function are the timing intervals between two subsequent pacemaker stimuli and the interval between an intrinsic event and the subsequent pacemaker stimulus.

During the diagnostic process as schematized in figure 9.2, the expert often uses causal tracing, a variant of the generic task model "systematic diagnosis". Sometimes the inference process of the expert has elements of the task model "systematic refinement". In causal tracing the expert follows a causal network for the possibilities of malfunction, whereas in systematic refinement an "is_a" structure is followed. However, the inference structure is similar for both task models.

In causal tracing, problem solving starts at the first level of the causal network. At this level one hypothesis is chosen, after which this hypothesis is verified or rejected by other
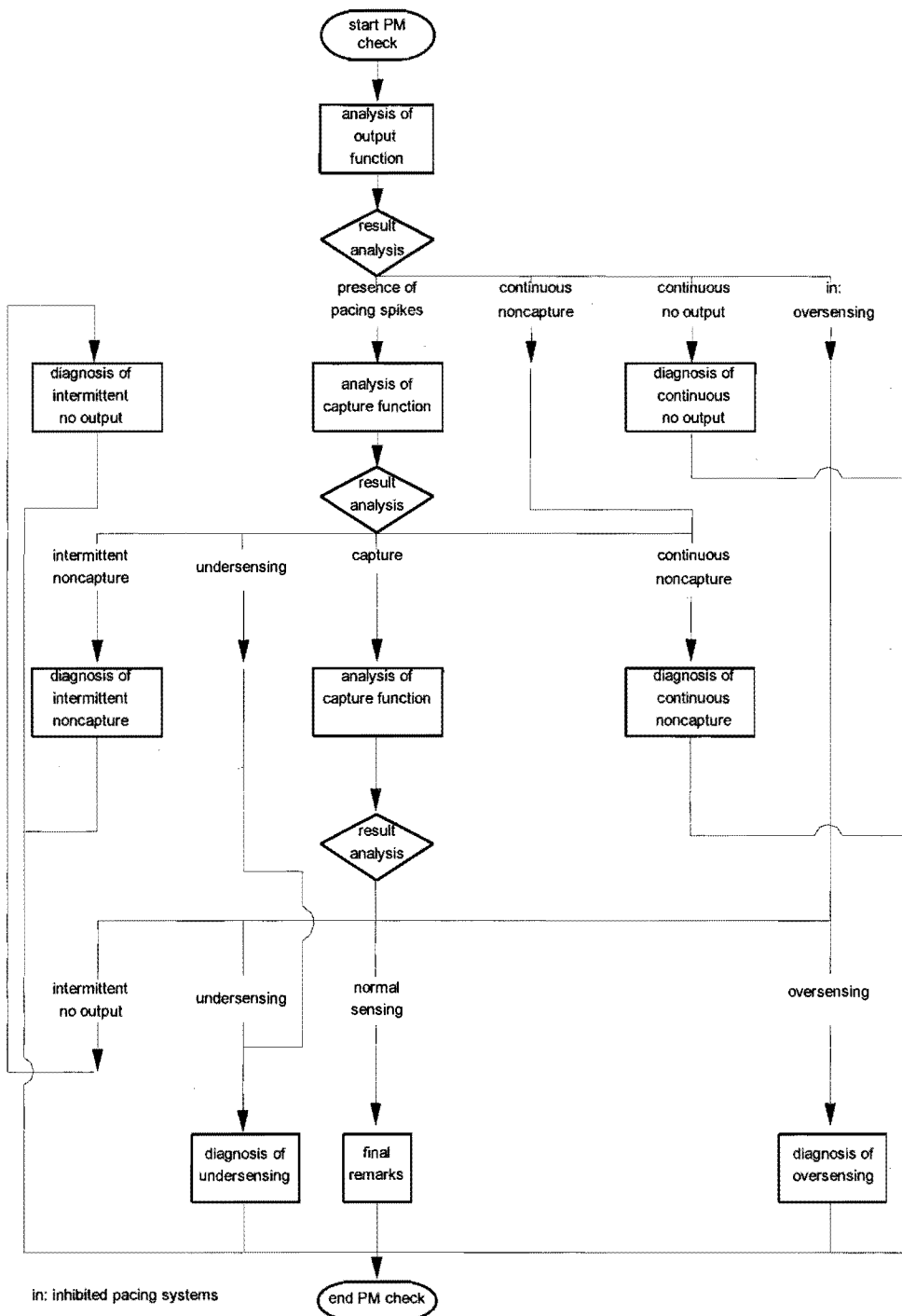
Fig. 9.2. Diagram showing the general problem solving strategy.

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observations and/or tests. If the hypothesis cannot be verified, an attempt will be made to verify a different hypothesis at the same level in the causal network. After confirmation of a hypothesis one has to proceed to a lower level in the causal network and repeat the process until the final diagnosis has been made. If no hypothesis is confirmed, the task has formally failed.

9.2.2 Formalization of the analyzed expert knowledge

During this phase in the development of an expert system the analysed knowledge from the previous phase is converted into a formal structure.

The knowledge from the previous phase is mainly human oriented, which means that it is presented in such a way that there is a good communication between the knowledge engineer and the domain expert. Knowledge is represented in normal language and in diagrams, which implies that this form may contain ambiguity.

The main purpose of formalization is to obtain an unequivocal representation of knowledge that can be used for implementation in the system. Formalization can be represented by rules in pseudocode or network structures.

The representation formalism used in the development of this expert system is the AND/OR TREE. The AND/OR TREE consists of an inverted tree structure with nodes and interconnections. The top node or root symbolizes a target or a hypothesis. This top node is interconnected with a number of nodes representing a subtarget or one or more conditions that have to be fulfilled. These subtargets or conditions can be AND or OR operated. This is illustrated by the word AND or an arc under the node, or the word OR or omission of the arc under the node. Figure 9.3 shows a formalized presentation of a part of the output function analysis.
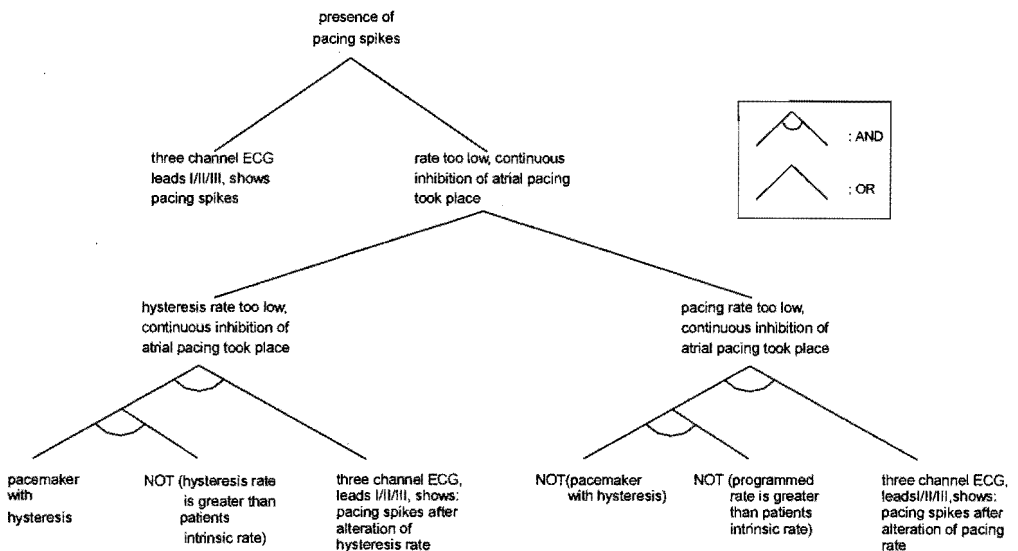
Fig. 9.3. AND/OR representation of the problem space of part of the output function analysis.

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We have chosen the AND/OR TREE formalism:
1) It offers a better surveillance than rules formalized in pseudocode;
2) It is a clear presentation of formalization for the domain expert;
3) The way of presenting shows similarity to the flow chart and appears to be a suitable tool for this application.

A disadvantage of the AND/OR TREE is that the order of evaluation is not always clear. This problem can be solved by an informal agreement about the order of evaluation, which should be implemented in a way similar to that agreement.
The order of evaluation is important because in the prototype of the expert system a dialogue between user and system is created. Therefore the order of evaluation by the expert system should be a logical one, because the user should not be confused.

9.2.3 Implementation of the formalized expert knowledge
After formalization of the analyzed knowledge it should be implemented in the system. For the implementation programming languages like LISP, PROLOG, PASCAL or a knowledge system development environment (shell) can be used.
Programming languages can be considered as general purpose tools, whereas shells are dedicated tools with a specific application and provide additional tools like debugging and testing.
In the development of our expert system we used the expert system building tool SIMPLEXYS [5].

9.2.4 The expert system building tool SIMPLEXYS
Simplexys was developed at the Eindhoven University of Technology for application in intelligent patient monitoring during anaesthesia. The knowledge necessary for incorporation in a patient monitoring system was unsuitable for an algorithmic approach. For this reason artificial intelligence techniques were used.
Limitation of many existing shells is the real-time performance necessary in patient monitoring. To overcome this limitation of real time application SIMPLEXYS was developed. SIMPLEXYS is a collection of tools for the design and development of real-time expert systems for clinical problem solving. In SIMPLEXYS goals and protocols can be implemented. The basic unit of presentation is a rule, which has the form of a definition "...is defined as...", this in contrast to production systems, which use the form "if ... then ....".
Most rule based expert systems have a rule interpreter. The rule interpreter determines how and when rules have to executed; it also functions as the inference engine of the system. In SIMPLEXYS a rule compiler is incorporated. This rule compiler translates knowledge into an internal network. Due to this internal network representation there is no need for a knowledge base search for the evaluation of a rule when the expert system is consulted.
For the evaluation of an expression, SIMPLEXYS has three "values"; "TRue", "FAlse" or "POssible". The latter means that an expression cannot be shown to be either true or false. SIMPLEXYS contains operators applicable to one but also to two expressions and the use of history operators. History operators enable the system to perform numeric instead of logical operations, e.g. how long is a condition true ?

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The inference engine can use any combination of backward chaining and forward chaining in its inferencing.

SIMPLEXYS is chosen for the following reasons:
1) The formalized expert knowledge is simple and surveyable by the application of rules that resembles nodes of an AND/OR tree
2) High level presentation of a problem by goals and protocols
3) Possibility of backward and forward chaining
4) Inference engine implemented in PASCAL, which increases efficiency as compared to LISP and PROLOG
5) The use of PASCAL enables application of a user interface and connection with other data bases
6) Immediately available, without extra costs
7) Specific knowledge about SIMPLEXYS is available and easily accessible, because it was designed at the Eindhoven University of Technology
8) Modification of source code of the inference engine and other parts of SIMPLEXYS is possible
9) In the standard program numerous tools and testing capabilities are available

SIMPLEXYS lacks the ability to reason with uncertain data or domain knowledge by means of possibilistic or probabilistic logic. This, however, is not important for the expert system because during problem solving the domain expert clearly differentiates between mutually excluding hypotheses.

In the design of the knowledge base we have chosen a clear definition of the rules. We also used the possibility of application of capitals or lower case letters in the uncompiled knowledge base. Words in capitals, with exception of the term "_atr" (=atrium), "_ven" (=ventricle), "_in" (=inhibited) and "_tr" (=triggered) symbolize hypotheses.

Words in lower case letters symbolize symptoms, test results or facts (e.g., pacemaker parameters) used to prove hypotheses.

The logic expression as formalized in figure 9.3 is implemented in SIMPLEXYS as follows:

{1}   AS_atr_in:'Presence of pacing spikes'
        aS OR RATE_LOW_atr
        THEN DO
        write_dialogue ('Pacing spikes present');
        write_dialogue ('Proceeding with capture check..');
        ENDDO
        THEN FA: DIAGNOSE_NOOUTPUT

{2}   RATE_LOW_atr:'Rate too low, continuous inhibition of atrial pacing took place'
        HYST_RATE_LOW_atr OR PACE_RATE_LOW_atr

{3}   HYST_RATE_LOW_atr:'Hysteresis rate too low, continuous inhibition of atrial
                pacing took place'
        pace_hyst AND NOT hyst_great_intrin AND aS_retryh

THEN DO
write-dialogue ('Hysteresis rate was too low. Continuous inhibition of pacing by the
intrinsic rate took place')
ENDDO

{4}   PACE_RATE-LOW_atr:'Pacing rate too low, continuous inhibition of atrial pacing
took place'
NOT pace_hyst AND NOT pace_great_intrin AND aS_retryp
THEN DO
write_dialogue ('Pacing rate was too low. Continuous inhibition of pacing by the
intrinsic rate took place');
ENDDO

{5}   aS:'Three channel ECG, leads I/II/III, shows: Pacing spikes'
ASK

{6}   pace_hyst:'Pacemaker with hysteresis'
BTEST paceinfo.hyster=true

{7}   hyst_great_intrin:'Hysteresis rate is greater than patients intrinsic rate'
ASK
ELSE DO
write_dialogue ('Please enlarge hysteresis rate so the above condition is satisfied
otherwise no valid conclusions can be drawn!');
ENDDO

{8}   aS_retryh:'Three channel ECG, leads I/II/III, shows: Pacing spikes after alteration
of hysteresis rate'
ASK

{9}   pace_great_intrin:'Programmed pacing rate is greater than patients intrinsic rate'
ASK
ELSE DO
write_dialogue ('Please enlarge programmed pacing rate so that above condition is
satisfied otherwise no valid conclusions can be drawn');
ENDDO

{10}   aS_retryp:'Three channel ECG, leads I/II/III, shows: Pacing spikes after alteration
of pacing rate'
ASK

The rules 1, 2, 3 and 4 are EVAL-RULES; the value of the rule's conclusion is determined by
the result of the evaluation of its logic expression. Rules 5, 7, 8, 9 and 10 are ASK-RULES;
the value of the rule's conclusion is determined by the answer of the user to a question. Rule 6

is a BTEST-RULE which contains a boolean PASCAL expression. This rule results in a conclusion TRue or FAlse. Rules 1,3,4,7 and 9 contain THELSES.

Some additional contructions can be explained by examples in which X and Y represent rules.

1. X: "........", THEN FA : Y

Means if X is true, then set Y to false. Evaluation of one rule (X) results in more than one conclusion (about X and Y).

2. X: "........" THEN DO Y ENDDO.

Means if X is true, then execute code Y. This allows e.g. output to the user.

3. X: "........" THEN GOAL: Y.

Means is X is true, then evaluate Y, which implements a forward chaining step.

9.2.5 The user-interface

During implementation attention should also be paid to the construction of a user-interface. The user-interface is the communication tool between expert system and user and it checks the user on incorrect supply of information to the system, as far as possible.

```
┌──────────────────── Pacemaker data ────────────────────┐
│                                                          │
│  Type   : VVI, Bipol unipol prog    prog.rate : 72  ppm (833ms) │
│                                     hyst.rate : 65  ppm (923ms) │
│  Options: real-time telemetry       refr.per  : 325 ms   │
│           marker channel telemetry  magn.rate : 80  ppm (750ms) │
│           telemetry of IEGMs                             │
│                                                          │
└──────────────────────────────────────────────────────────┘
```

```
┌──────────────────── Progress of analysis ────────────────────┐
│                                                               │
│  Output   check :  Pacing spikes present                      │
│  Capture  check :  EVALUATING : Ventricular capture           │
│  Sensing  check :                                             │
│                                                               │
│                                                               │
└───────────────────────────────────────────────────────────────┘
```

```
┌──────────────────── Dialogue ────────────────────┐
│  Starting with output check .....                 │
│                                                    │
│  Three channel ECG, leads I/II/III, shows : Pacing spikes ? y │
│  Pacing spikes present.                            │
│  Proceeding with capture check .....               │
│                                                    │
│  Three channel ECG, leads I/II/III, shows : Ventricular pacing spike │
│  continuously followed by a paced or paced fusion QRS complex ? │
│                                                    │
└────────────────────────────────────────────────────┘
```

Fig. 9.4. Screen layout during consultation of the expert system.

The screen lay-out during the use of the expert system is represented in figure 9.4. We chose a window structure for the display which offers the user a clear surveillance.

The upper window displays pacemaker specifications regarding the type of pacemaker, electrode configuration, available options and timing intervals.

The middle window is called the "progress of analysis" window. It is a type of "WHY" facility and illustrates which part of the pacemaker system is analyzed and which hypothesis is being evaluated. By representing the hypothesis under "evaluating" the user understands the background for the questions in the dialogue window.

In the dialogue window the user answers the questions raised by the expert system simply by "y", "n" or "?" (yes, no, unknown).

9.2.6 Conclusions

Task models of the KADS methodology we used during knowledge elicitation and acquisition. These task models contain a domain independent description of knowledge necessary to realize a class of corresponding tasks. Identification of these models employing problem solving in a similar way to the expert, resulted in a well-defined knowledge acquisition process. The approach gave a better insight in the expert knowledge and led to a well-defined knowledge base. For diagnostic purposes it offered reasoning capabilities at different levels of abstraction.

During the development it is important to be aware that the expert cannot be guided in his/her reasoning by the strategy of the task model if this task model is not an exact copy of his own strategy. If this occurs some heuristics, that could be valuable, may be missed. (See 9.3.6, ECG 03).

9.3 Evaluation of the prototype expert system

The prototype expert system was constructed to investigate the feasibility of such a system for the evaluation of the pacemaker ECG. If the expert knowledge is correctly implemented in the system, it also offers an adequate test of completeness and consistency of the knowledge of the domain expert. For the initial set up it was decided to limit the system to application in single chamber systems. If the results of the tests with the prototype expert system are positive, it indicates that application of an expert system in this domain is feasible. Construction of a system suitable for dual chamber pacing systems is a logical step forward into further evolution. The expert knowledge as described in this thesis was used by the knowledge engineer to construct the expert system. The following paragraphs describe the results of the analysis of 40 pacemaker ECGs by the prototype expert system. Specific features of these ECGs did not play a role in the construction of the rule base.

9.3.1 Material and Methods

Forty ECGs obtained from pacemaker patients were evaluated by the prototype expert system. Fifteen ECGs (Group A) showed normal or apparently normal pacemaker function, whereas twenty five ECGs (Group B) showed pacemaker malfunction.

The normal and apparently normal ECGs were randomly selected from ECGs obtained during pacemaker patient follow-up. These ECGs can be divided into 3 categories:

1) ECGs showing continuous stimulation without intervening intrinsic rhythm (5 ECGs)

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Table I. Diagnosis and cause of malfunction on 40 ECGs evaluated by the prototype expert system

ECG	Diagnosis	Cause of Malfunction
01	continuous noncaputure	broken conductor
02	continuous no output	air entrapment
03	continuous noncaputure	fibrous tissue formation
04	intermittent noncapture	short circuit
05	intermittent no output	short circuit
06	intermittent noncapture	incorrect lead connection
07	continuous noncapture	fractured anodal conductor
08	intermittent noncapture	fractured conductor
09	oversensing	R wave sensing (AAI)
10	continuous noncapture	DC shock
11	oversensing	T wave sensing (VVI)
12	intermittent noncapture	lead displacement
13	intermittent noncapture	insulation defect
14	continuous no output	set screw inproperly thightened
15	continuous noncaputure	set screw inproperly thightened
16	oversensing	P wave sensing (VVI)
17	intermittent noncapture	lead dsiplacement
18	intermittent noncapture	fractured conductor
19	intermittent noncapture	fibrous tissue formation
20	intermittent noncapture	Wenckebach exit block
21	continuous noncapture	DC shock
22	continuous noncaputure	inproper lead connection
23	undersensing	magnet application
24	continuous no output	battery depletion
25	continuous noncapture	fractured conductor
26	normal function	not applicable
27	normal function	not applicable
28	normal function	not applicable
29	normal function	not applicable
30	normal function	not applicable
31	normal function	not applicable
32	normal function	not applicable
33	normal function	not applicable
34	normal function	not applicable
35	normal function	not applicable
36	normal function	not applicable
37	normal function	not applicable
38	normal function	not applicable
39	normal function	not applicable
40	normal function	not applicable

2) ECGs showing exclusively intrinsic rhythm at a rate above the programmed rate of the pulse generator (5 ECGs)
3) ECGs showing alternating intrinsic rhythm and pacemaker rhythm (5 ECGs)

The ECGs showing malfunction were obtained from patients with single chamber systems (17 ECGs) and from patients with dual chamber systems (8 ECGs). All evaluated ECGs were from well documented cases, in which the cause of malfunction was previously identified. The diagnosis of malfunction was continuous noncapture in 8 ECGs, intermittent noncapture in 9 ECGs, continuous no output in 3 ECGs, intermittent no output in 1 ECG, oversensing in 3 ECGs and undersensing in 1 ECG. Tabel I summarizes the 40 ECGs according to the diagnosis and the cause of malfunction, if applicable.
Although the expert system was initially designed for single chamber systems, it can also be applied to the atrial or ventricular channel of a dual chamber system for the evaluation of effective stimulation.

9.3.2 Operation of the expert system
Before the test by the expert system can be performed the following information should be supplied by the interpreter:
1) Pacing modality
2) Electrode configuration
3) Programmed pacing rate
4) Hysteresis / hysteresis rate, if applicable
5) Refractory period
6) Magnet rate
7) Real time telemetry available ? (lead impedance, battery voltage)
8) Marker channel available ?
9) Intracardiac electrogram available ?

These pacemaker data are displayed in the upper window of the monitor screen. After supplying these data the dialogue is started. The questions of the dialogue are displayed in the lower window. The interpreter has to answer the questions by yes (y), no (n) and if the answer is unknown by (?). In the middle window the expert system indicates the progress of analysis. It shows which function is checked and which possibility is evaluated by the question in the dialogue window.
The following information should be available to the operator:
1) Lead impedance and changes in lead impedance.
2) Time interval between recording of the ECG and pacemaker implantation.
This information is normally available in the clinical setting during which a pacemaker ECG is evaluated. Lead impedance or changes in lead impedance can be obtained from pacemaker telemetry or calculated by pulse waveform analysis.
In the prototype expert system the following phenomena can be distinguished:
1) Normal pacemaker function
2) Continuous no output
3) Intermittent no output
4) Continuous noncapture

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5) Intermittent noncapture
6) Oversensing
7) Undersensing

In the currently available version of the prototype expert system (400 rules), the cause of pacemaker malfunction can only be evaluated for the malfunctions mentioned under 2,4 and 5. Because of this limitation of the expert system in the evaluation of the cause of malfunction, the majority of the selected ECGs showing malfunction were related to stimulation problems (21 ECGs) and limited to oversensing in 3 ECG and undersensing in 1 ECG.

9.3.3 Evualation of the expert system results
In the evaluation of this prototype expert system three crucial questions have to be answered:
1) Is the expert system leading to the right conclusion with respect to diagnosis and cause of malfunction ?
2) Is the reasoning process leading to the conclusion correct ?
3) If the conclusion is false or the reasoning process is not correct, is this related to incompleteness of knowledge acquisition or limitations of the implemented reasoning structure ?
In order to answer these questions the following aspects were evaluated during the test with the expert system:
1) Is the diagnosis classified correctly or incorrectly ?
2) If the diagnosis is correct, is the order of questioning logical ?
3) If the diagnosis is correct, are there unnecessary questions ?
4) Are unnecessary questions as mentioned under 3 related to incompleteness of knowledge acquisition ?
5) Are unnecessary questions as mentioned under 3 related to limitations in the implemented reasoning structure ?
6) If the diagnosis is incorrect is this related to incompleteness of knowledge acquisition ?
7) If the diagnosis is incorrect, is this related to limitations in the implemented reasoning structure ?
8) Is the cause of malfunction classified correctly or incorrectly ?
9) If the cause of malfunction is correct, is the order of questioning logical ?
10) If the cause of malfunctioning is correct, are there unnecessary questions ?
11) Are unnecessary questions as mentioned under 10 related to incompleteness in knowledge acquisition ?
12) Are unnecessary questions as mentioned under 10 related to limitations in the implemented reasoning structure ?
13) If the cause of malfunction is incorrect, is this related to incompleteness in knowledge acquisition ?
14) If the cause of malfunction is incorrect, is this related to limitations in the implemented reasoning structure ?

Table II. Results expert system test

ECG	Diagnosis	Logical order	Effective questioning	Cause of malfunction	Logical order	Effective questioning
01	Correct	Yes	Yes	Correct	Yes	Yes
02	Correct	Yes	Yes	Correct	Yes	Yes
03	Correct	Yes	Yes	Correct	No, LRS	No, LRS
04	Correct	Yes	Yes	Correct	Yes	Yes
05	Incorrect, LRS	No, LRS	No, LRS	Correct	Yes	Yes
06	Correct	Yes	Yes	Correct	Yes	Yes
07	Correct	Yes	Yes	Correct, IKA	Yes	Yes
08	Correct	Yes	Yes	Correct	Yes	Yes
09	Correct	Yes	Yes	NA	NA	NA
10	Correct	Yes	Yes	Incorrect, IKA	No, LRS	No, LRS
11	Correct	Yes	Yes	NA	NA	NA
12	Correct	Yes	Yes	Correct	No, LRS	No, IKA
13	Correct	Yes	Yes	Correct	Yes	Yes
14	Correct	Yes	Yes	Correct	Yes	Yes
15	Correct	Yes	Yes	Correct	Yes	Yes
16	Correct	Yes	Yes	NA	NA	NA
17	Correct	Yes	Yes	Correct	No, LRS	No, IKA
18	Correct	Yes	Yes	Correct	Yes	Yes
19	Correct	Yes	Yes	Correct, IKA	Yes	Yes
20	Correct	Yes	Yes	Correct	Yes	Yes
21	Correct	Yes	Yes	Incorrect, IKA	No, LRS	No, LRS
22	Correct	Yes	Yes	Correct	Yes	Yes
23	Correct	Yes	Yes	NA	NA	NA
24	Correct	Yes	Yes	Correct	Yes	Yes
25	Correct	Yes	Yes	Correct	Yes	Yes
26	Correct	Yes	Yes	NA	NA	NA
27	Correct	Yes	Yes	NA	NA	NA
28	Correct	Yes	Yes	NA	NA	NA
29	Correct	Yes	Yes	NA	NA	NA
30	Correct	Yes	Yes	NA	NA	NA
31	Correct	Yes	Yes	NA	NA	NA
32	Correct	Yes	Yes	NA	NA	NA
33	Correct	Yes	Yes	NA	NA	NA
34	Correct	Yes	Yes	NA	NA	NA
35	Correct	Yes	Yes	NA	NA	NA
36	Correct	Yes	Yes	NA	NA	NA
37	Correct	Yes	Yes	NA	NA	NA
38	Correct	Yes	Yes	NA	NA	NA
39	Correct	Yes	Yes	NA	NA	NA
40	Correct	Yes	Yes	NA	NA	NA

Abbreviations: LRS = Limitations in reasoning structure; IKA = Incomplete knowledge acquisition; NA = Not applicapble.

9.3.4 Results Group A
In this group 15 ECGs (ECGs 26-40 in table I and II) with normal or apparently normal pacemaker function were evaluated by the expert system. This group was subdivided into 3 categories.

1. ECGs showing continuous stimulation without intervening intrinsic rhythm (5 ECGs).
All these ECGs were selected because capture could be recognized from the ECG and effective stimulation was confirmed. If this is the only ECG available, sensing function cannot be evaluated, because no intrinsic activity is present on the ECG. The answers to the questions regarding normal sensing and undersensing are unknown. Only the absence of oversensing can be concluded from the ECG, if the interval between pacemaker stimuli is fixed and equal to the pacemaker escape interval. The expert system confirms ventricular (atrial) capture but indicates "no differentiation possible" for the sensing check.
If a secondary ECG, obtained during the same follow-up, would have been available, in which the stimulation rate was decreased and intrinsic rhythm became manifest, normal sensing of intrinsic depolarization could have been confirmed.
The diagnosis is correct in all of the cases, the order of questioning is logical, there are no unnecessary questions.

2. ECGs showing intrinsic rhythm at a rate above the programmed rate of the pulse generator (5 ECGs).
Because effective stimulation cannot be concluded from this recording, the expert system suggests increasing the stimulation rate in order to initiate pacing. If pacing is effective and sensing is normal, normal pacemaker function has been established. Diagnosis is correct in all cases, the order of questioning is logical, there are no unnecessary questions.

3. ECGs showing alternating intrinsic rhythm and pacemaker rhythm (5 ECGs).
From 4 out of these 5 recordings it is relatively easy to conclude normal function. Effectiveness of stimulation and normal sensing can be observed on the ECG and normal pacemaker function can be concluded. The diagnosis is correct in all cases, the order of questioning is logical, there are no unnecessary questions (Fig. 9.5).

One two-channel ECG obtained from a patient with an atrial pacemaker (AAI) was selected for evaluation because atrial depolarization following stimulation was not visible on the ECG. The expert system reached the conclusion that effective stimulation is present, based on the information that there is a 1:1 relation between atrial stimuli and the following intrinsic QRS complex (Fig. 9.6). Also in this case the diagnosis is correct, the order of questioning is logical and there are no unnecessary questions.

In all 15 ECGs showing normal or apparently normal pacemaker function the expert system comes to the right conclusion with logical and no unnecessary questions.
After the observation that normal pacemaker function has been established, the expert system suggests that the operator pay attention to the quality of the pacing system. This is done by advice to check battery voltage, stimulation threshold, lead impedance, sensing threshold and morphology changes in the stimulated depolarization. These parameters should be checked to

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```
──────────────────────── Pacemaker data ────────────────────────
Type    : VVI Unipolar                    prog. rate :   70ppm ( 857ms)
                                          rafr. per. : 320ms
Options: real-time telemetry              magn. rate :   70ppm ( 857ms)
         marker channel telemetry
         telemetry of IEGMs
```

```
──────────────────────── Progress of analysis ────────────────────────
Output  check: Pacing spikes present
Capture check: Ventricular capture
Sensing check: Ventricular normal sensing
```

```
──────────────────────── Dialogue ────────────────────────
Starting with output check .....

Three channel ECG,leads I/II/III, shows: Pacing spikes ? y
Pacing spikes present.
Proceeding with capture check .....

Three channel ECG,leads I/II/III, shows: Ventricular pacing spike
continuously followed by a paced or paced fusion QRS complex ? y
Ventricular capture has been established.
Ventricular pacing parameters should be determined:
        · Battery voltage      (for check on EOL)
        · Stimulation threshold (for check on encapsulation)
        · Lead impedance       (for check on lead integrity)

Proceeding with sensing check .....

Ventricular normal sensing ? y
Normal sensing has been established.
Sensing parameter should be determined:
        · Sensing threshold

Normal pacemaker operation has been established.

Measured pacing and sensing parameters should be compared with
the parameters gathered during former follow-ups.
This to exclude/keep track of evolving problems.
Morphology changes should also be documented, to keep track of possible
dislocation of the stimulation electrode.
```

Press any key to continue

Fig. 9.5. ECG and print-out of the expert system evaluation of an ECG showing normal pacemaker function.

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determine sufficient safety margins for pacing and sensing (stimulation threshold, sensing threshold, battery voltage) and to exclude or keep track of evolving problems (lead impedance, changes in morphology of depolarization). Pacing and sensing parameters should be compared with the parameters gathered during former follow-ups.

9.3.5 Results Group B

In this group 25 ECGs showing pacemaker malfunction were evaluated by the expert system. Malfunction was subdivided into the following 6 categories.

1) Continuous no output (3 ECGs)
The diagnosis continuous no output is correctly made by the expert system. The diagnosis is based on the absence of pacing spikes in the 12 lead ECG and nonstandard precordial leads at double standard (at higher amplification). By magnet application, not resulting in pacing spikes, no output is differentiated from oversensing. The order of questioning is logical and there are no unnecessary questions.
The causes of no output in the 3 analyzed cases were air entrapment in a unipolar system (ECG 02), inappropriate lead connection (ECG 14), and battery depletion (ECG 24).

The expert system points to the cause of no output in case of air entrapment (ECG 02). Questioning is logical and there are no unnecessary questions (Fig. 9.7).
In the evaluation of the cause of no output in ECG 14 (inappropriate lead connection), the expert systems leads to the conclusion "Galvanic discontinuity in the atrial lead system has been established", which is correct. If no X-ray or fluoroscopy is available, the expert system generates a list of possible outcomes, in which "interruption at lead connector" is mentioned.

In ECG 24, in which no output is caused by battery depletion, the expert system leads to the conclusion that no output is caused by pulse generator failure. Pulse generator failure can be differentiated between component failure and battery failure, but the expert system gives both possibilities. The questioning for cause evaluation is logical and there are no unnecessary questions.

2) Intermittent no output (1 ECG)
The diagnosis intermittent no output cannot be made by the prototype version of the expert system. This will be discussed later (see discussion). In order to perform an analysis by the expert system, only the period of no output on the ECG is taken into account and the ECG is classified as no output (ECG 05). If the section of the ECG in which no output is observed is analyzed by the expert system, the expert system leads to the right conclusion and also the cause of malfunction (short circuit in ventricular lead system) is correctly determined. The order of questioning is logical and there are no unnecessary questions.

3) Continuous noncapture (8 ECGs).
The diagnosis continuous noncapture is correctly made by the expert system. The diagnosis is based on the presence of pacing spikes not followed by atrial/ventricular depolarization. In one ECG (ECG 25), the diagnosis could only be made by using a nonstandard precordial lead at double standard, which was requested by the expert system. Only this lead showed pacing

```
─────────────────────── Pacemaker data ───────────────────────
Type    : AAI Unipolar                    prog. rate :   70ppm ( 857ms)
                                          refr. per. : 325ms
                                          magn. rate :   70ppm ( 857ms)
```

```
─────────────────────── Progress of analysis ───────────────────────
Output  check: Pacing spikes present
Capture check: Atrial capture
Sensing check:
```

```
─────────────────────── Dialogue ───────────────────────
Starting with output check .....

Three channel ECG,leads I/II/III, shows: Pacing spikes ? y
Pacing spikes present.
Proceeding with capture check .....

Three channel ECG,leads I/II/III, shows: Atrial pacing spike continuously
followed by a paced or paced fusion P wave ? ?
Three channel ECG,leads I/II/III, shows: Atrial pacing spike intermittent
followed by a paced or paced fusion P wave ? ?
Three channel ECG,leads I/II/III, shows: Atrial pacing spike is always part
of pseudofusion P wave, when not follwed by a paced or paced fusion P wave
? ?
Three channel ECG,leads I/II/III, shows: Atrial pacing spike, after
relative fixed AV interval, continuously followed by intrinsic (normal) QRS
complex ? y
Atrial capture has been established.
Atrial pacing parameters should be determined:
      · Battery voltage      (for check on EOL)
      · Stimulation threshold (for check on encapsulation)
      · Lead impedance       (for check on lead integrity)
```
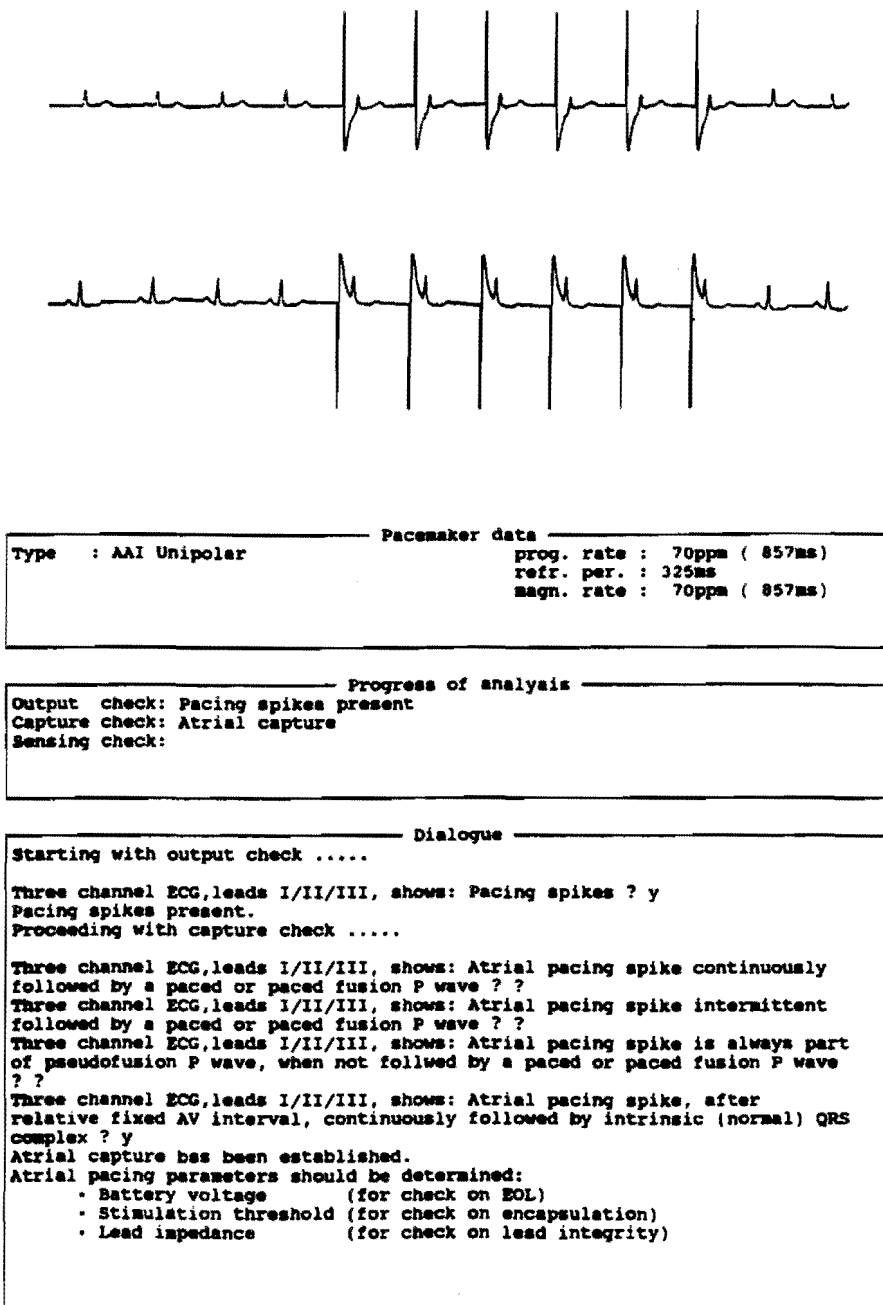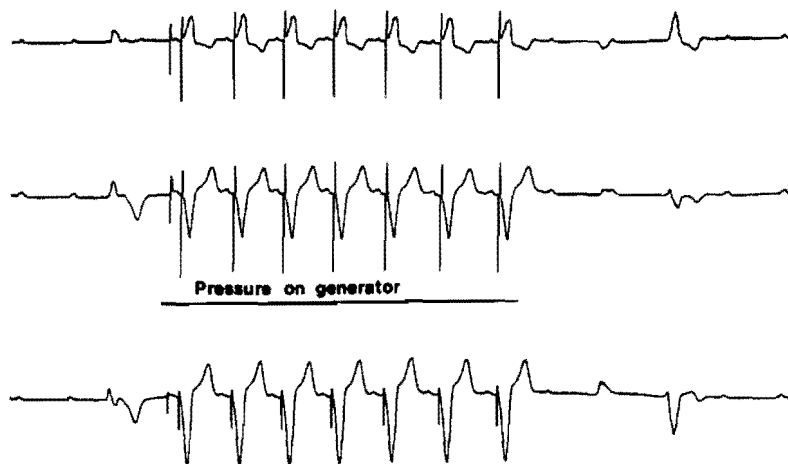
Press any key to continue

Fig. 9.6. Two-channel ECG and print-out of the expert system evaluation of an ECG (AAI pacemaker) in which atrial depolarization after stimulation is not visible on the ECG. Effective stimulation is concluded by the expert system based on the 1:1 relation between atrial stimuli and ventricular response.

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Pressure on generator

```
──────────────────── Pacemaker data ────────────
Type    : VVI Unipolar                  prog. rate :  70ppm ( 857ms)
                                        refr. per. : 325ms
Options: real-time telemetry            magn. rate :  70ppm ( 857ms)
```

```
──────────────────── Progress of analysis ──────────
Output  check: No output
Capture check:
Sensing check:

Cause: Anodal insulation through air entrapment
```

```
──────────────────────── Dialogue ──────────
Three channel ECG,leads I/II/III, shows: Pacing spikes ? n
Programmed pacing rate is greater than patients intrinsic rate ? y
Three channel ECG,leads I/II/III, shows: Pacing spikes during magnet
application ? n
12 lead ECG, or nonstandard precordial leads ECG (/DS), shows: Pacing
spikes ? n
12 lead ECG, or nonstandard precordial leads ECG (/DS), shows: Pacing
spikes during magnet application ? n
Ventricular no output has been established.
Proceeding with diagnosis for probable causes .....

ECG shows: Ventricular pacing spikes during manipulation of PM ? y
Ventricular no pulse transmission has been established.
Proceeding for a more detailed diagnosis .....

Ventricular lead impedance is very high ? y
Galvanic discontinuity in ventricular lead system has been established.
Proceeding for a more detailed diagnosis .....

The following questions should be answered during slight pressure on over
PG lying skin :
Three channel ECG,leads I/II/III, shows: Ventricular pacing spike
continuously followed by a paced or paced fusion QRS complex ? y
Anodal insulation through air entrapment has been established.
```

Press any key to continue

Fig. 9.7. ECG and print-out of the expert system evaluation of an ECG showing only pacing spikes during slight pressure on the pulse generator. The cause of malfunction was subcutaneous air entrapment, which was correctly diagnosed by the expert system.
Note: The pacing system is a dual chamber sytem but the analysis is performed for the ventricular channel.

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spikes not followed by depolarization, whereas in the standard 12 lead ECG no pacing spikes were visible. The order of questioning is logical and there are no unnecessary questions.

The causes of noncapture in the 8 analyzed cases were a fractured conductor (ECG 01,07,25), inappropriate lead connection (ECG 15,22), after DC-shock (ECG 10,21) and, fibrous tissue formation (ECG 03).

The 3 cases with a fractured conductor (ECG 01,07,25) and 2 cases with inappropriate lead connection (ECG 15,22) have in common that the stimulation current is reduced by an increase in lead impedance. The expert system points to the correct cause of noncapture by the conclusion "Discontinuity in atrial/ventricular lead system has been established" (Fig. 9.8). If no X-ray or fluoroscopy is available, the expert system generates a list of possible outcomes, in which "Incorrect ventricular connector position" and "Ventricular lead conductor fracture" are mentioned. The solution for the problem was found by a logical order of questioning without unnecessary questions.

In the 2 cases (ECG 10,21), in which continuous noncapture was observed after DC-shock, noncapture is caused by either a local reaction around the stimulation electrode due to the high intensity of the DC-shock or an elevated stimulation threshold due to reduced oxygen extraction (Chapter IV, 4.8.4.1). The expert system leads to the conclusion "Pulse generator malfunction (by DC-shock) due to component failure can be possible".

The order of questioning is not logical. After confirming the question "Lead impedance is normal ?" the expert system concludes "Problem with the ventricular myocardial interface or pulse generator has been established", which still is correct. The following question "PM check is the first after implantation ?" is answered in the negative. Under these conditions the expert first has to evaluate the causes of rise in stimulation threshold or inappropriate output programming, because lead displacement is unlikely. The expert system, however, first excludes perforation by 2 questions. After confirming the question "Increase in ventricular stimulation threshold ?", the question "PM check is the first after implantation ?" is repeated. After answering "no" to this question again, the position of the electrode is evaluated, which is not logical as previously stated. Finally the expert system raises a number of questions to differentiate between causes of elevation of the general state of excitability and inappropriate programming or changes in output setting. Confirming the question "Patient was submitted to external defibrillation ?" leads to the conclusion "Pulse generator malfunction due to component failure can be possible".

In the ECG showing continuous noncapture (ECG 03) due to fibrous tissue formation 2 test runs were performed by the expert system. In the first test run it was assumed that no X-ray or fluoroscopy information was available, whereas this information was used in the second test run. In the first test run the cause of noncapture, as analyzed by the expert system, was "Problem with ventricular electrode myocardial interface or pulse generator has been established". After confirming that lead impedance is normal and that this is the first PM check after implantation, the expert system first tries to rule out or confirm lead displacement, which is a likely explanation for noncapture under these conditions. The order of questioning is not logical because an important number of questions address findings on X-ray and fluoroscopy, whereas electrocardiographic signs of displacement should be

V1

vvl 40 ppm

V6
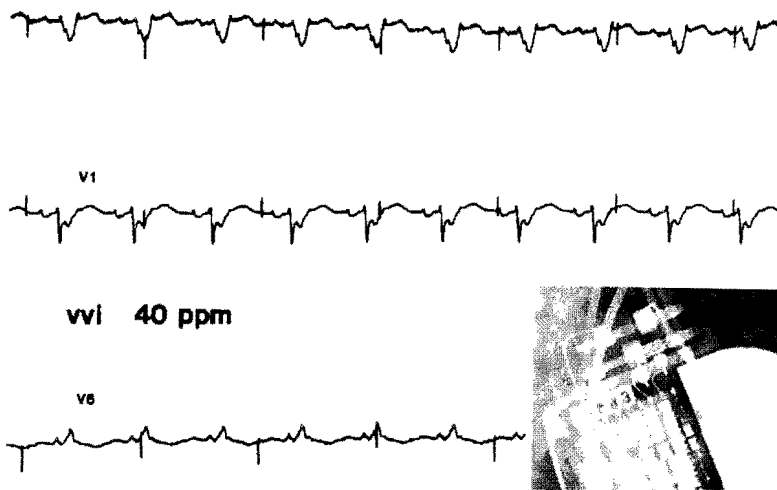
```
────────────────────── Pacemaker data ──────────────────────
Type    : VVI Bipolar                    prog. rate :   40ppm (1500ms)
                                         refr. per. : 200ms
Options: real-time telemetry             magn. rate :   40ppm (1500ms)
```

```
────────────────────── Progress of analysis ──────────────────────
Capture check: Ventricular continuous noncapture
Sensing check:

Cause: EVALUATING: Problem with ventricular electrode myocardial interface or
Cause: Discontinuity in lead system
```

```
────────────────────── Dialogue ──────────────────────

Three channel ECG,leads I/II/III, shows: Pacing spikes ? y
Pacing spikes present.
Proceeding with capture check .....

Three channel ECG,leads I/II/III, shows: Ventricular pacing spike
continuously followed by a paced or paced fusion QRS complex ? n
Three channel ECG,leads I/II/III, shows: Ventricular pacing spike
intermittent followed by a paced or paced fusion QRS complex ? n
Ventricular continuous noncapture has been established.
Proceeding with diagnosis for probable causes .....

Ventricular lead impedance is normal ? n
Problem with integrity of ventricular lead system has been established.
Proceeding for a more detailed diagnosis .....

Ventricular lead impedance is high ? y
Discontinuity in ventricular lead system has been established.
Proceeding for a more detailed diagnosis .....

Xray shows: Incorrect ventricular connector position ? ?
Xray shows: Problem with integrity of ventricular adapter ? ?
Xray shows: Ventricular lead conductor fracture ? ?
Symptoms and/or testresults do not provide for a more detailed diagnosis.
```

Press any key to continue

Fig. 9.8. ECG and print-out of the expert system evaluation of an ECG showing continuous noncapture. The cause of malfunction was an inappropriately connected ventricular lead of a dual chamber system (see X-ray), which was correctly diagnosed by the expert system.
Note: For the evaluation it was assumed that no X-ray information was available. If this information is available the question "Incorrect ventricular connector position ?" can be answered positively.

evaluated first. The question "ECG shows: Ventricular pacing spike is followed by a RBBB pattern instead of a LBBB pattern ?" is inappropriate. Because there is continuous noncapture on the ECG this question cannot be answered and should not be posed.

In the second test run information from X-ray and fluoroscopy was used to confirm a correct position of the stimulation electrode. In this test run the question "PM check is the first after implantation?" is also posed twice. The question regarding QRS morphology (RBBB or LBBB) is also raised, but cannot be answered because continuous noncapture is present. The expert system finally concludes that the cause of noncapture is "Stimulation threshold rise due to local tissue reaction around ventricular stimulation electrode has been established", which is correct.

4) Intermittent noncapture (9 ECGs).
The diagnosis intermittent noncapture is correctly made by the expert system. The diagnosis is based on the presence of pacing spikes not always followed by depolarization. To confirm intermittent noncapture the presence of pseudofusion and/or myocardial refractoriness at the time of pulse delivery are excluded by the expert system. The order of questioning is logical and there are no unnecessary questions.
The causes of intermittent noncapture were short circuit (ECG 04,13), fractured conductor (ECG 08,18), inappropriate lead connection (ECG 06), lead displacement (ECG 12,17), fibrous tissue formation (ECG 19), Wenckebach exit block between stimulation electrode and myocardium (ECG 20).

In the 2 cases with short circuit (which was an intermittent phenomenon; ECG 04,13) the stimulation current passing the myocardium was reduced because the leakage current through the short circuit exceeded the maximum output capacity of the pulse generator.
The expert system used the attenuation of the pacemaker spike on the ECG as an indicator for a variation in lead impedance and measurement of lead impedance discriminated between short circuit and galvanic discontinuity. The order of questioning is logical and there are no unnecessary questions.

The ECGs showing intermittent noncapture caused by a broken conductor (ECG 08,18) and inappropriate lead connector (ECG 06) have in common the fact that the stimulation current is reduced by an intermittent increase in lead impedance. For ECG 06 (inappropriate lead connector) the expert system concludes the correct cause of malfunction without unnecessary questions and a logical order of questioning. For the ECGs 08,18 (fractured conductor) the expert system leads to the conclusion "Intermittent discontinuity or short circuit in ventricular lead system has been established". Because lead impedance could not be determined in these pulse generators, neither the expert system nor the expert is able to find the final cause under these circumstances. The expert system advices to proceed to fluoroscopy or X-ray in order to differentiate between the two possibilities, which is a logical step that also would have been followed by the expert.

For the ECGs showing intermittent noncapture due to electrode displacement (ECG 12,17) the expert system concludes "Unstable ventricular electrode endocardial interface has been

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established". This conclusion is correct. However, after excluding attenuation of the pacemaker spike during noncapture, the following question refers to Wenckebach periodicity between electrode and myocardium. This is a very rare phenomenon and in the order of questioning it should be evaluated at the end. The morphology of the stimulated depolarization during capture is not compared with previous recordings, whereas changes in the morphology of stimulated depolarization is a definite proof of lead dislodgement. In ECG 12 it is obvious that a properly timed atrial contraction preceding the pacemaker spikes results in ventricular capture. This observation, that there is a relation between mechanical action and effective stimulation indicating an unstable electrode position, has not yet been incorporated in the knowledge base.

For the ECG showing intermittent noncapture due to fibrous tissue formation (ECG 19) the expert system concludes "Ventricular PG output close to stimulation threshold". This conclusion is correct, but the final cause for the rise in stimulation threshold is not determined. The morphology of the stimulated QRS complex is not compared with previous recordings, which would eliminate displacement as the possible cause.

For the ECG showing intermittent noncapture due to a Wenckebach exit block between electrode and myocardium (ECG 20), the expert system draws the right conclusion. There are no unnecessary questions and the order of questioning is logical.

5) Oversensing (3 ECGs)
In these ECGs (ECG 09,11,16) effective stimulation is confirmed by the expert system. Oversensing is recognized by prolongation of the escape interval. Because the reasoning process for the analysis of the the cause of oversensing is not yet implemented, no detailed analysis can be made.

6) Undersensing (1 ECG)
In this ECG (ECG 23) not every pacemaker stimulus is followed by ventricular depolarization. Although this can be interpreted as noncapture, the expert system concludes that noncapture is caused by refractoriness of the myocardium. Refractoriness of the myocardium for a pacemaker stimulus indicates that the preceding depolarization is not sensed by the pacemaker. This has to be classified as undersensing, which is the correct conclusion.

The results obtained by the expert system analysis are summarized in table II. The ECG numbers correspond with the numbers of table I. In the second column is indicated whether the diagnosis is correct or incorrect. The third column (logical order) indicates whether the order of questioning leading to the diagnosis is logical or not. Effective questioning in the fourth column indicates whether unnecessary questions are posed. The fifth column indicates whether the cause of malfunction analyzed by the expert system is correct or incorrect. Logical order (column six) and effective questioning (column seven) refer to the logical order of questioning and whether unnecessary questions are raised to conclude the cause of malfunction.

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If in the columns for diagnosis and cause of malfunction is mentioned "Incorrect", the reason for this outcome is mentioned. This outcome can be caused by limitations in the implemented reasoning structure (LRS) or incompleteness of knowledge acquisition (IKA). The same symbols are used when the order of questioning is not logical or when unnecessary questions are raised.

On some occassions (ECG 07,19) the outcome in the cause of malfunction is true, but is followed by IKA (incomplete knowledge acquisition). In these cases the answer is not false but can be more sharply defined by an increase in knowledge acquisition.

If the cause of malfunction cannot be evaluated because the evaluation of the sensing function of the pacing system is not yet implemented in the expert system, this is indicated by NA (not applicable). The same symbols are used for the ECGs 26 to 40, in which normal pacemaker function was present.

The diagnosis made by the expert system was correct in 39 out of 40 ECGs (97.5 %). The cause of malfunction was successfully determined by the expert system in 19 out of the 21 ECGs (90,5 %). In 2 of these cases (ECG 07,19) the cause of malfunction would be more accurately defined by an increase in knowledge acquisition.

In 3 cases (ECG 03,12,17) in which the outcome of the cause of malfunction was correct the implementation of the reasoning structure can be improved, thus reducing the number and the order of questions.

In two cases in which the cause of malfunction was incorrectly determined by the expert system, this was due to incomplete knowledge acquisition and limitations in the implemented reasoning structure.

9.3.6 Discussion

Normal and apparently normal pacemaker function can be determined reliably by the expert system. From the ECGs showing alternating intrinsic rhythm and pacemaker rhythm determination of the pacing and sensing function is relatively easy. Even in a case, in which depolarization caused by the pacemaker stimulus is difficult to see on the ECG (Fig. 6), the expert system leads the interpreter to the right conclusion.

In those cases, in which the ECG shows exclusively intrinsic rhythm, the expert system suggests increasing the stimulation rate in order to initiate pacing. If stimulation is effective normal pacemaker function has been established. Normal sensing can be concluded from the absence of pacemaker stimuli when the intrinsic rate is higher than the programmed rate of the pulse generator.

In the ECGs showing continuous stimulation without intervening intrinsic rhythm, normal sensing cannot be concluded. Because evaluation of the sensing function is not yet accomplished in the expert system, the rules for evaluation of normal sensing are not yet implemented. When sensing function is implemented in the system, this should also be done for evaluation of ECGs showing apparently normal stimulation.

In the ECGs showing malfunction the diagnosis is false in only one case (ECG 05). The expert system is not leading to the conclusion "intermittent no output". The explanation for this failure is relatively simple. An ECG showing intermittent no output is characterized by prolongation of the escape interval, which is also true for oversensing. To differentiate

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between oversensing and intermittent no output, the pacing sensing has to be converted to an asynchronous mode. If prolongation of the escape interval disappears after conversion to the asynchronous mode, the underlying mechanism is oversensing. If prolongation of the escape interval is maintained, intermittent no output is established. In the prototype expert system, the section which deals the evaluation of the sensing function and if necessary the following diagnosis in which reasoning with intervals is involved, is not yet implemented. However, if only the no output period present in the ECG, is evaluated by the expert system, the cause of malfunction is determined correctly.

For ECG 03, in which continuous noncapture is present due to fibrous tissue formation around the stimulation, the final conclusion is correct but the reasoning process must be improved significantly. After confirmation of the diagnosis, normal lead impedance rules out the possibility of lead integrity failure as the cause of noncapture. The remaining possibilities for noncapture are electrode displacement or reduced excitability of the myocardium by either local effects (fibrous tissue formation) or general effects. The following question "PM check is the first after implantation ?" does not differentiate between these two possibilities, but states which possibility should be evaluated first. The rationale behind this is, that electrode displacement is more likely to occur shortly after implantation, and therefore this possibility is evaluated first. Until this point the reasoning structure is logical. However, in the evaluation of lead displacement two questions address the findings during X-ray or fluoroscopy. This is not logical as this information is not available at the pacemaker check-up. It is more logical to evaluate electrocardiographic or clinical signs of lead displacement and the best suggestion is to increase pacemaker output, in order to restore capture. If capture can be restored, electrode displacement can be confirmed or ruled out by comparing the morphology of the stimulated depolarization. If the depolarization is identical to the morphology on previous recordings electrode displacement is excluded and the other possiblities should be evaluated. If capture cannot be restored, lead displacement can only be indicated by excluding the other possibilities of noncapture and in the last instance confirming by X-ray or fluoroscopic findings.

For the ECGs 12 and 17, showing intermittent noncapture due to electrode displacement, the cause of malfunction is correct. The reasoning process, however, needs some improvement. To begin with, if noncapture is not associated with attenuation of the pacemaker spike on the ECG, lead integrity is excluded as the possible cause. The opposite reasoning is true, that noncapture associated with attenuation of the pacemaker spike is caused by a defect in lead integrity, but if no attenuation is observed lead integrity should be confirmed by a normal lead impedance.
A Wenckebach exit block between electrode and myocardium as the possible cause is evaluated first. This should be done finally, because it is a very rare phenomenon.
In ECG 12 the unstable myocardial-electrode interface is indicated on the ECG by the observation that capture is present if the ventricular stimulus is preceded by a P wave with an interval corresponding to the normal PQ time. Capture is probably caused by the atrial contraction, bringing the electrode in contact with the ventricular myocardium. In ECG 17 lead displacement is indicated by a change in morphology of the stimulated QRS complex. Both observations should be added to the knowledge base.

For the ECGs 10 and 21, showing continuous noncapture after cardioversion, the cause of noncapture is incorrect and the implemented reasoning structure has limitations. After answering "no" to the question "PM check is the first after implantation ?" the expert system excludes myocardial perforation of the electrode by 2 questions. Perforation is most likely to occur in the first months after implantation, so this order of questioning is not logical. The question "PM check is the first after implantation ?" is posed twice, the second time followed by a question concerning X-ray findings. Finally the question is raised "Was the patient submitted to external defibrillation ?". After confirmation of this question the expert system comes to the conclusion "Pulse generator malfunction (by DC-shock) due to component failure can be possible". This conclusion is incorrect, because in one of the previous questions "change in pacing parameters ?" is answered in the negative. If pacing parameters are not changed component failure is not possible. If noncapture is observed after external defibrillation there are 3 possibilities:

1) There is an elevated stimulation threshold after cardioversion, which usually recovers after a short period of time.

2) The pacing system is reverted to a back up mode pacing, which may alter output parameters. This can be corrected by resetting the pacing system by the pacemaker programmer.

3) Component failure, which may result in changing output parameters or even no output. Component failure in the sensing circuit will result in sensing abnormalities.

This information should be added to the knowledge base.

In ECG 7, showing continuous noncapture, the expert system concluded that discontinuity in the lead system is the cause of noncapture. This conclusion is correct but can be more sharply definied by using the programmability of the electrode configuration (uni/bipolar). By measuring lead impedance in both configurations the discontinuity can be located in the anodal conductor if lead impedance is normal in the unipolar configuration and increased in the bipolar configuration, which was the case in this example.

In ECG 19, showing intermittent noncapture, the expert system concluded "Ventricular PG output close to stimulation threshold". This conclusion is also correct but by comparing morphology of the QRS complex of the effective stimuli to previous recordings displacement of the electrode can be excluded.

9.3.7 Conclusions

For the total results in the test with respect to the diagnosis the expert system comes to the right diagnosis in 39 out of the 40 ECGs (97,5%). In the only case that was missed, the diagnosis is not incorrect but cannot be determined because the sensing check is not yet implemented in the prototype version. The cause of malfunction is correctly determined in 19 out of the 21 ECGs (90,5%) showing malfunction, but we have to take into consideration that in the 2 cases in which the cause of malfunction was incorrect, the cause was identical.

The reasoning structure was implemented correctly, which means that there were no unnecessary questions and a logical order of questioning, in 16 out of the 21 ECGs (76,2 %) showing malfunction.

When comparing the reasoning process of the expert with the expert system, it appeared that the expert system did not make use of the search limiting heuristics of the expert in every

situation. The explanation of this phenomenon is that during elicitation and acquisition of knowledge too much attention was paid to the task model, which had a negative effect on the knowledge acquisition. More knowledge acquisition, thus using the limiting search heuristics of the expert, would have reduced the number of questions, but this had only limited effect on the final result.

More knowledge acquisition would also improve the quality of determination of the cause of malfunction. As illustrated by ECG 7 and 19, the cause of malfunction is correctly determined but can be more sharply defined by extending knowledge acquistion.

During testing the user interface gave a clear surveillance by the window structure. The presence of the "WHY" facility enlarges the insight of the user in the reasoning process of the system and adds an educative character to the system. Operation of the system is simple and hardly needs explaning to the potential user.

Although for the prototype version of the system, the initial choice was made to limit the system for application only to single chamber systems, it appeared that the system can be used without limitations for dual chamber systems with respect to the evaluation of effectiveness of stimulation. The only restriction is that the atrial and ventricular channel of the dual chamber system have to be analyzed separately as an AAI and VVI pacemaker.

It is our opinion that with this prototype expert system a promising start has been made on a system that offers a useful support for the ECG interpreter during the follow-up of pacemaker patients. After improving the order of questioning the cause of malfunction can be determined without the use of X-rays or fluoroscopy. This does not imply that X-rays or fluoroscopy are not necessary, but that these investigations can be focussed directly on the cause of malfunction.

After completion of an expert system for single chamber pacing systems, which is still a limitation in the present prototype, the system should be extended to dual chamber pacing systems. The most likely future development is coupling an expert system to pacemaker programming equipment. Coupling with the pacemaker programmer enables the expert system to collect pacemaker specifications directly. Data obtained by pacemaker telemetry such as lead impedance can be used immediately in the reasoning process, which can be shortenedby this. Simultaneous coupling of the expert system to a pacemaker patient data base would offer the possibility of comparison of previous observations and measurements with the actual information.

References

1. Bourgonje RWM. A prototype expert system for analysis and diagnosis of the functioning of permanent single chamber pacemakers. A decision support system (in Dutch). MSc. thesis, Dept. of Electr. Engng, Eindhoven University of Technology, 1994.

2. Jackson P. Introduction to expert systems. Second edition. Wokingham: Addison-Wesley, 1990.

3. Breuker J, Wielenga B. Analyse van expertise voor expertsystemen. In : Nijholt en-Steels, 1986, pp. 5.1-5.28.

4. Tansley DSW, Hayball CC. Knowledge-based system analysis and design. A KADS developer's handbook. New York: Prentice Hall, 1993.

5. Blom JA. The Simplexys experiment. Real time expert systems in patient monitoring. PhD thesis. Eindhoven University of Technology, 1990.

Conclusions and Recommendations

The purpose of this thesis was to describe a systematic approach for the analysis of the ECG representing pacemaker malfunction or apparent malfunction. Normal pacemaker function is characterized by the occurrence of correctly timed pacemaker stimuli at a preset interval followed by myocardial depolarization.

The ECG, however, reveals more information about the pacemaker system than only effective stimulation and correct sensing. Morphology of stimulated depolarization contains information about the location of the stimulation electrode, which is important in case of lead dislocation or inadvertent lead malplacement.
The presentation of the pacemaker stimulus on the ECG contains information about the location of the anodal electrode in unipolar systems. Abrupt variations in amplitude of the pacemaker stimulus on the ECG may be an indicator of lead failure or impending lead failure. An increase in amplitude in bipolar systems is an indicator for the presence of a defect in the outer insulation.

Recognition of capture or noncapture can be elucidated by secondary characteristics in the ECG or by the use of nonstandard precordial leads. In this thesis I have tried to describe as comprehensively as possible the electrocardiographic presentation of malfunction and how to analyze the possible causes, using the above mentioned elements.

After writing a thesis on this subject, it is still difficult to answer the questions "Is it complete ?" and "How systematical and consistent is the approach ?".
Therefore I am grateful to my promotor Prof. J.E.W. Beneken, who suggested during my initial proposal for this thesis, to construct an expert system based on the knowledge and rules used in the analysis of the pacemaker ECG, as described in this thesis.

A prototype expert system for analysis and diagnosis of the functioning of permanent single chamber pacemakers, constructed and described by Ir. René Bourgonje in cooperation with Dr.J.A. Blom, my co-promotor, resulted from this suggestion. The expert knowledge and the reasoning process described in this thesis formed the elements of the knowledge and rule base of the expert system.

The prototype expert system affords us the opportunity to investigate the feasibility of such a system for the analysis of the pacemaker ECG. Along with this, clinical testing has answered the previous questions about the completeness, consistency and the systematical approach described in the thesis.

From the results of the tests, it can be concluded that:
1. An expert system as described in chapter IX by Bourgonje is feasible for the evaluation of the pacemaker ECG.
2. Knowledge acquisition in the prototype expert system is at a very acceptable level. In those cases where the cause of malfunction could not be correctly determined or more sharply defined, it was not due to the fact that the knowledge was not available in the thesis but because it was not yet implemented in the system.

3. Determination of the cause of malfunction was correct in 90.5% of the investigated cases and a correctly implemented reasoning structure was present in 76,2%.

The conclusions mentioned under 2 and 3 are an indicator for the quality and the completeness, consistency and systematics of the thesis. A restricting factor is that these conclusions can only be drawn for that part of the thesis, which is implemented in the expert system.

The expert system in its current version still has limitations. Minor changes in the reasoning structure and knowledge acquisition will improve the functioning and results of the system. The system also has to be completed for the analysis of the sensing function of the single chamber pacing system.

After completion of the system for single chamber pacemakers, it should be extended to a system for dual chamber pacemakers. This step is relatively easy, because it was shown by the tests performed by the prototype expert system that analysis of dual chamber pacemakers for effectiveness of stimulation can be carried out without any problems.

During the further development of the expert system it will initially be used as a "stand alone" system until it is completed. After completion, the most likely course is integration of an expert system in pacemaker programming equipment. This integration enables the expert system to collect pacemaker specifications and telemetry data directly. These data can be used in the reasoning process, which can be shortened thereby and lead the ECG interpreter to the cause of malfunction with minimal interaction with the system.

Summary

The ECG is the most important tool to determine pacemaker function and malfunction. Effective stimulation, appropriate sensing of intrinsic atrial and ventricular depolarization and ignoring inappropriate signals are the factors determining normal pacemaker function.

Chapter I describes the additional techniques used to confirm the electrocardiographical diagnosis or to differentiate between the possible causes of malfunction. Some of these techniques are also used to evaluate the quality of the pacing system with respect to the safety margins for pacing and sensing. Determination of pacing/sensing threshold and lead impedance are important parameters for the early recognition of impending pacemaker problems.

Chapter II describes the relationship between the site of stimulation and the morphology of the resulting depolarization. This information is important in order to recognize malplacement of the stimulation electrode during implantation. A changing morphology of the stimulated depolarization during follow-up indicates a changing site of stimulation, which can be associated with electrode displacement.

Chapter III describes the presentation of the pacemaker stimulus on the ECG. The pacemaker stimulus can be distorted by some (digital) recording equipment, which implies that no conclusions can be drawn from the appearance of the pacemaker stimulus from ECGs recorded by this type equipment. In the analog recorded ECG abrupt variation in amplitude of the pacemaker stimulus may contain information in case of pacemaker malfunction, because there is a direct relationship between amplitude of the stimulus and the strength and direction of the stimulation current.

Chapter IV describes the effectiveness of stimulation in single and dual chamber pacing systems. To conclude effective stimulation the pacemaker stimulus has to be recognized to discriminate between no output and noncapture. Depolarization following stimulation has to recognized to determine capture or noncapture. Recognition of atrial capture can be problematic and sometimes requires nonstandard precordial leads at double standard amplification. ECGs showing some pitfalls in the recognition of effectual stimulation and the causes of ineffectual stimulation are described in this chapter.

Chapter V delineates the timing intervals in single chamber systems. Timing intervals play an essential role in the evaluation of pacemaker sensing. Pacemaker oversensing is characterized by prolongation of the escape interval, whereas undersensing is characterized by shortening of the escape interval following an intrinsic depolarization. The mechanisms and causes of sensing abnormalities are elucidated.

Chapter VI delineates the timing intervals in dual chamber systems, which can be more complicated than in single chamber systems. Except from the problems encountered in single chamber systems, the ECG in dual chamber systems may show apparent malfunction due to the interaction between programmable parameters. Therefore an important part of this chapter is devoted to the timing intervals in the normal functioning dual chamber pacing system. In case of

malfunction in one of the channels of a dual chamber system, this malfunction may give rise to complex ECGs, even suggestive for malfunction of the other channel.

Chapter VII summarizes the various types of pacemaker tachycardia, the underlying mechanism and the management of prevention or termination of these pacemaker tachycardia.

Chapter VIII gives a practical approach to pacemaker malfunction or apparent malfunction. In this approach schematic ECGs are used to illustrate pacemaker malfunction or apparent malfunction. In this way the electrocardiographic problem is relatively easy identified and the strategy to find the underlying cause is described.

Chapter IX describes the development and first clinical testing of a prototype expert system. In this protype expert system, developed in collaboration with the Eindhoven University of Technology, the rules for analysis of the pacemaker ECG have been implemented. The first results of clinical tests are reported.

Samenvatting

Het ECG is het belangrijkste hulpmiddel om pacemaker functie en malfunctie te kunnen vaststellen. Effectieve stimulatie, het op de juiste wijze detecteren van intrinsieke atriale en ventriculaire depolarisatie en het niet reageren op stoorsignalen zijn de factoren die de pacemaker functie bepalen.

In hoofdstuk I worden de aanvullende technieken beschreven die gebruikt worden om de electrocardiografische diagnose te bevestigen of om te differentieren naar de verschillende oorzaken van malfunctie. Een aantal van deze technieken wordt ook gebruikt om de kwaliteit van het stimulatiesysteem te evalueren met betrekking tot de veiligheidsmarges voor stimulatie en detectie van intrinsieke signalen. Bepaling van deze parameters samen met de stimulatie impedantie zijn belangrijk voor de vroegtijdige herkenning van dreigende pacemaker problemen.

Hoofdstuk II beschrijft de relatie tussen de plaats van stimulatie en de door stimulatie veroorzaakte depolarisatie. Deze relatie is belangrijk om een onjuiste positionering van de stimulatie electrode tijdens implantatie tijdig te herkennen. Een verandering in morfologie van het gestimuleerde complex tijdens follow-up van pacemaker patienten kan op dislocatie van de stimulatie electrode wijzen.

In hoofdstuk III wordt aandacht besteed aan de vorm van stimulatie impuls in het ECG. In bepaalde (digitale) registratie apparatuur wordt de stimulatie impuls vervormd of kunstmatig gegenereerd zodat geen conclusies verbonden kunnen worden aan de vorm van deze impuls. In het ECG geregistreerd door middel van analoge apparatuur kunnen conclusies verbonden worden aan de abrupte veranderingen in amplitude van de pacemaker stimulus in geval van malfunctie, omdat er een directe relatie bestaat tusen de amplitude van de stimulus en de sterkte en richting van de stimulatie stroom.

Hoofdstuk IV beschrijft de efffectiviteit van stimulatie in zowel een- als twee-kamer systemen. Om effectiviteit van stimulatie te herkennen is het nodig dat de pacemaker stimulus herkend wordt om onderscheid te kunnen maken tussen geen volgcomplex (noncapture) en geen pacemaker stimulus (no output).
Herkenning van depolarisatie is noodzakelijk om te kunnen discrimineren tussen capture en noncapture. Herkenning van effectieve atriale stimulatie is soms moeilijk en maakt het gebruikt van niet gestandaardiseerde precordiale afleidingen met en dubbele versterking van de electrocardiograaf noodzakelijk.In een aantal ECGs worden de moeilijkheden in de herkenning van effectieve stimulatie gedemonstreerd en de oorzaken van ineffectieve stimulatie beschreven.

In hoofdstuk V worden de tijdsintervallen in een-kamer systemen uiteengezet. Tijdsintervallen spelen een belangrijke rol in de evaluatie van het detectie systeem van de pacemaker. Pacemaker oversensing is gekarakteriseerd door een verlenging van het stimulatie interval, terwijl bij ondersensing het interval tussen intrinsieke depolarisatie en de daarop volgende stimulatie impuls korter is dan het stimulatie interval. De onderliggende mechanismen en oorzaken van abnormaliteiten in het detectie systeem worden beschreven.

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In hoofdstuk VI worden de tijdsintervallen van de twee-kamer systemen beschreven, welke complexer kunnen zijn dan in de een-kamer systemen. Behalve de stoornissen welke beschreven zijn in de een-kamer systemen, kan het ECG in twee-kamer systemen schijnbare malfunctie laten zien welke veroorzaakt wordt door interactie tussen de verschillende parameters. Om deze reden is in dit hoofdstuk een belangrijk deel gewijd aan de tijdsintervallen bij normaal functionerende twee-kamer systemen. In geval van malfunctie in een van de kanalen van een twee-kamer systeem, kan deze malfunctie aanleiding geven tot complexe ECGs, welke soms zelfs suggestief zijn voor malfunctie van het andere kanaal.

Hoofdstuk VII geeft een overzicht van de verschillende soorten pacemaker tachycardiën. Het onderliggend mechanisme en de handelswijze voor preventie of beeindigen van deze tachycardiën is beschreven.

Hoofdsstuk VIII geeft een praktische benadering van pacemaker problemen door middel van schematische ECGs. Op deze manier zijn electrocardiografische problemen op een envoudige wijze herkenbaar gemaakt, waarbij de strategie om tot de oorzaak van het probleem te komen is beschreven.

Hoofdstuk IX beschrijft de ontwikkeling en eerste klinische test resultaten van een prototype kennis systeem (Expert System). In dit kennis systeem, ontwikkeld in samenwerking met de Technische Universiteit van Eindhoven, zijn de regels voor analyse van het pacemaker ECG geimplementeerd. De eerste resultaten van de klinische testen worden hierin gerapporteerd.

Pacemaker Glossary

Edited by Karel den Dulk, Berry van Gelder and Rob van Mechelen,
Workshop Pacemaker & ECG.

AAI
The Intersociety Commission on Heart Disease Resources code for atrial inhibited pacing. Pacing and sensing occur in the atrium (AA); the mode of response is inhibited (I). When spontaneous atrial activity is sensed the atrial pace response is inhibited and the timer is reset to time out the programmed escape interval once more. When atrial activity is not observed an atrial pace response is produced after the programmed pacing (escape) interval is timed out. AAI pacing is also called atrial demand pacing.

AAT
The Intersociety Commission on Heart Disease Resources code for atrial triggered pacing. Pacing and sensing occurs in the atrium (AA); the mode of response is triggered (T). An atrial response is generated:
(1) when atrial activity is sensed during the atrial sensing interval. In the presence of spontaneous atrial activity this pace response does not contribute to atrial depolarization. The escape interval of the pacemaker is reset.
(2) when atrial activity is not sensed during the atrial sensing interval. An atrial pace response is generated at the end of the programmed pacing interval.This response results in atrial depolarization.

Absolute Refractory Period (ARP); Myocardium.
In physiology it represents the length of time from the onset of an action potential until repolarization is more than one-third complete. An electrical stimulus that falls within the absolute refractory period will not initiate a new action potential; the tissue is unresponsive.

Absolute Refractory Period (ARP); Pacing system.
During this part of the refractory period, which is initiated by a sensed or paced event, the pulse generator is unable to sense.

Action Potential
Rapid change in membrane potential of nerves or muscles. Each action potential begins with a sudden change from the normal negative resting potential to a positive membrane potential (depolarization) and then ends with a return to the negative resting potential (repolarization).

Adapter
Connecting element of pacemakers and leads which are otherwise incompatible; especially used to facilitate the connection of already implanted leads.

AH Interval
The AH interval is a measure of the conduction time through atrial tissue, node to the bundle of His. It is measured from a catheter placed at the tricuspid valve ring adjacent to the His bundle.

The interval is measuredfrom the onset of atrial depolarization to the onset of the His bundle deflection. Normal values range from 50 to 150 milliseconds.

Alert Period
The part of the atrial or ventricular cycle during which the pacemaker is sensitive, "alert" to incoming signals.

Algorithm
Calculation instruction, i.e. to understand how subtraction works is not difficult. However, to subtract 435 beans from 678 beans can be a time consuming job. First count 678 beans, put them in a pot and than take 435 beans out of the pot. Finally count the beans left in the pot. Applying the subtracting algorithm provides a more efficient way to get the answer.

Ampere
The basic unit of electrial current: electrical charge (coulomb) per second. One ampere (A) represents a current of one coulomb per second. Pacemaker systems require a small amount of current, expressed in milliamperes (mA) and microamperes (muA).

Amplitude
Pacemaker amplitudes are expressed in volts and represent the valvue of the electrical potential difference between the anode and cathode of the pacing system.

Analog
Opposite of digital. A continuous signal that varies in amplitude and direction and is a representation of a corresponding valvue for measured valvues of a physiocal quantity. For example, an electrocardiogram is the visual analog of cardiac electrical activity.

Anode
The positive terminal of a battery or circuit. The terminal that receives electrons. In a unipolar pacing system the housing of the pulse generator is the anodal terminal. In a bipolar system, the anode is the proximal ring that receives the electrons, whereas the distal ring or cathode is the stimulation terminal that produces electrons.

Antegrade Conduction
Normal conduction from sino-atrial node to AV node to the bundle of His and finally to the Purkinje fibers. Antegrade conduction also refers to atrio-ventricular or AV conduction.

AOO
The Intersociety Commission on Heart Disease Resources code for atrial-asynchronous pacing. Pacing occurs in the atrium (A); the mode of response is fixed or asynchronous at the programmed pacing interval. There is no sensing of spontaneous atrial activity (O).

Artficial Circus Movement Tachycardia (ACMT)
Sometimes also called Pacemaker Circus Movement Tachycardia (PCMT) or less specifically Pacemaker Mediated Tachycardia or Endless Loop Tachycardia A Pacemaker Circus Movement

Tachycardia is an artificial reentry tachycardia in which the retrograde limb of the circuit is provided by the AV node or accessory pathway, while the anterograde limb is by way of AV synchronization of the pacemaker.

AR interval
The interval from the atrial pacing stimulus to the onset of the next QRS complex, as measured on the electrocardiogram.

Artifact
Extraneous signals superimposed on the electrocardiogram. Artifacts may be caused by pacemaker pulses, electromagnetic interference, myopotentials, programming transmission and defibrillation.

Asynchronous
A mode of operation in which the pacemaker is insensitive to intrinsic cardiac signals and paces at the programmed pacing interval.

Atrial capture
The depolarization of the atria by a pacemaker stimulus. In pacing, atrial capture can be confirmed on an electrocardiogram or intracardiac electrogram.

AV interval
The interval between the initiation of a paced or sensed atrial event and the consecutive ventricular output pulse.

AV sequential Pacing
Dual-chamber pacing in which the atrium is paced before the ventricle at the programmed AV interval and where sensing occurs either in the ventricle (DVI mode) or not at all (DOO mode).

AV Synchrony
The normal physiological relationship between atrial and ventricle systole. The relationship is lost in patients with AV block. It is also lost in patients with single chamber ventricular pacing.

Backup mode
A pacing mode automatically activated when the pacemaker is subjected to defibrillation or electrocautery, or when pacemaker component malfunction occurs.

Battery
One or more power source cells that produce electrical energy. A battery consists of a negative pole, the cathode; a positive pole, the anode; and an electrolyte through which electrically charged particles are transported. The chemical interaction between the anode and cathode poles causes current flow through an external circuit. In pacing ,the anode in most pacemakers is lithium and the cathode usually is cupric sulfide (lithium-cupric sulfide) or iodine (lithium iodine).

Bipolar lead
A lead with two electrical poles that are external to pulse generator. Both pacemaker terminals, anode and cathode are located at the distal part of the lead positioned in the right atrium or right ventricle.

Blanking
A time period during which one of the pacemaker sensing amplifiers is temporarily disabled following delivery of an output pulse from the other channel. The blanking period prevents inappropriate sensing of afterpotentials from the pacemaker output pulse and, in dual chamber pacemakers, prevents sensing of pacemaker output pulses or intrinsic events in the chamber other than that in which the event occurredd. In dual chamber pacemakers, sensing of the atrial output pulse in the ventricular channel is prevented by ventricular blanking.

Capacitor
An element that can store an electrial charge.It is made of two conductors separated by an insulator. A capacitor is incorporated in the output circuit of the pacemaker to eliminate the risk of inducing fibrillation by applying direct current to the heart as a potential consequence oif component failure. The capacitor is charged by the battery and then quickly and partially discharged over the heart, thus producing the stimulus.

Capture
Depolarization in response to an electrical stimulus emitted by the pacemaker.

Cathode
The negative terminal of a circuit or a battery, i.e. the terminal that emits electrons. In a pacing system the distal tip of the lead usually serves as a cathode.

Channel
In dual-chamber pacing, channel 1 or the atrial channel controls atrial sensing and atrial pacing, channel 2 or the ventricular channel controls ventricular sensing and pacing.

Charge
The international unit for electrical charge (Q)is the coulomb. In pacing, electrical charge is expressed in microcoulombs. Since current is by definition the amount of electrical charge per second (Coulombs/s or microcoulombs/ms) is the electrical charge expressed in microcoulombs.

Charge Density
In pacing, charge density refers to the amount of charge (current x pulse duration) delivered to the myocardium at the electrode/myocardial interface per unit surface area (mm2). Charge density is thus expressed by the quotient of charge and surface area (microcoulombs/mm2).When stimuli of the same amplitude are delivered either at an electrode with a large surface area and an electrode with a small surface area. This stimulus delivered at the smallest surface area has the greatest charge density.

Chest Wall Stimulation
Delivery of stimuli by an external pacemaker to the chest wall via 2 electrodes pasted on the skin. The stimuli are sensed by the implanted pacemaker and depending on the programmed pacing mode inhibit or trigger output pulses from the implanted pacing system.

Chronaxie
The pulse duration of an electrial stimulus that has a strength twice that of the rheobase. The voltage (or current) corresponding to an infinitely large pulse width in the strength-duration curve (plateau of the curve) is called the rheobase.

Chronic pacing threshold
With a pulse generator of a variable output pulse (the pulse amplitude and/or the pulse width), one can measure the lowest pulse amplitude for a given pulse width or the lowest pulse width at a fixed amplitude at which the heart is captured. The minimum value is called the threshold, i.e. the voltage or current threshold at a certain pulse width or the pulse width threshold at a certain current or voltage. The chronic pacing threshold is the minimum output pulse required to depolarize the myocardium consistently at least 1 month after pacemaker implantation.

Chronotropic incompetence
Inability of the heart to increase its rate appropriately in response to increased metabolic demand.

Committed mode
A dual-chamber pacemaker modality in which an atrial output pulse forces (commits) the delivery of a ventricualr output pulse.

Coulomb
The unit of measurement of an electrical charge; a quantification of electrons. One coulomb is equal to the amount of electrical charge transported by a current of one Ampere for one second.

Crosstalk
The phenomenon that one channel has an undersirable influence on the other channel has been called pacemaker cross-talk. Dual-chamber pacemakers might emit a stimulus in one channel while the sensing amplifier in the other channel is still alert for incoming signals. The stimulus has an amplitude between 2.5-5 volts and the amplifier in the other compartment is tuned to detect signals of a few millivolts amplitude. Without special precautions, such a stimulus would clearly be detected by the sensing amplifier of the other channel and would cause inappropriate inhibition (self-inhibition)

Crosstalk blanking
To prevent undue interference of stimulation in one channel on sensing in the other channel, detection of stimulus artefact is prevented by ensuring that the sensing amplifier will be refractory (blanked) during stimulation.
For example, blanking of the ventricular sense amplifier during delivery of the atrial output pulse.

Current

The net transfer of electrical chage through a cross section of a conductor in a given period of time. Current is expressed in amperes or milliamperes.

Current density

In pacing current density commonly is measured as the amount of current at the electrode surface that is delivered to the excitable tissue. The current density at the electrode surface is the quotient of current and surface area (milliAmperes/ mm2).

Current drain

The average amount of current drawn from a battery by the external load of a system. In pacing, the amount of current drain depends on the circuitry; the percentage of paced events; such programmable parameters as rate, amplitude and pulse duration ; the electrode surface area and the impedance of the pacing system. Current drain occurs with each paced or sensed event; typically the amount of current drain is lower for a sensed event than for a paced event.

Cycle length

Cycle length is a measurement of distance. The distance between one event and another event on a time scale. If the cycle length is constant, the rate (beats or pulses per minute) can be calculated. For example, a heart rate of 60 beats per minute equals a cycle length of 1 second. Cycle length (ms)=60,000/heart rate.(1 minute= 60 secondes= 60,000 milliseconds)

DDD

The Intersociety Commission on Heart Disease Resources code for one of the dual-chamber pacing modes. Pacing (D) and sensing (D) occur in both the atrium and ventricle (D=dual). In the absence of spontaneous cardiac activity, atrium and ventricle are paced sequentially at the programmed pacing interval. A ventricular sense event inhibits the ventricular stimulus (and atrial stimulus is delivered) and resets the timer for the atrial and ventricular escape intervals. A sensed atrial event inhibits an atrial stimulus but also triggers a ventricular stimulus after a programmed AV delay. The latter represents an inhibited as well as triggered response to a sensed atrial event which explains the 3rd D (D=dual repose).

DDI

The Intersociety Commission on Heart Disease Resources code for dual-chamber inhibited pacing in which pacing and sensing occur in both the atrium and ventricle (DD) and the mode of response is inhibited. Sensed atrial activity inhibits the atrial output inpulse, but does not trigger a ventricular output pulse. DDI pacing is simular to DVI pacing. However, atrial sensing in DDI pacing reduces the possibility of atrial competition between a spontaneous atrial depolarization and an atrial output pulse. In the absence of spontaneous cardia activity, atrium and ventricle are paced sequentially.

Default

The automatic selection of nominal values by a pacemaker programmer at the inititiation of programming. Comparable with the automatic selection of a television station after switching on a TV set.

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Demand pacing

The output pulse is inhibited when a spontaneous depolarization is sensed. If no intrinsic activity is sensed before the end of the alert period of the pacemaker timing cycle, an output pulse is delivered. Demand pacing is often used synonymously with VVI pacing, although it refers to any pacemaker taht provides an inhibited mode of response.

DOO

The Intersociety Commission on Heart Disease Resources code for AV sequential ventricular inhibited pacing. Pacing occurs sequentially in the atrium and ventricle (D), sensing occurs only in the ventricle(V) and the mode of response is inhibited (I). In the absence of spontaneous cardiac activity, atrium and ventricle are paced sequentially at the programmed pacing interval.

Elective Replacement Time (ERT)

The time at which the usble battery capacity is approaching depletion and replacement is indicated (ERT). In some pacemakers the ERT may be determined by the placement of a magnet over the pulse generator and the subsequent evaluation of the pacing rate.

Electrode (for stimulation)

The interface with living tissue across which the stimulus is transmitted. It is usually metal for example platinum or titanium.

Electrogram

With every depolarization an electrogram can be detected by the pulse generator via the pacing lead. In waveform of the electrogram, one can distinguish a rapid biphasic voltage change called the intrinsic deflection and a slowly varying voltage. The intrinsic deflection corresponds to the depolarization wave front passing under the electrode.

Endless Loop Tachycardia (ELT)

More specifically called Pacemaker Circus Movement Tachycardia or a Artificial Circus Movement Tachycardia. It is an artificial reentry tachycardia in which the retrograde limb of the circuit is provided by the AV node or accessory pathway, while the anterograde limb is by the way of AV synchronization of the pacemaker.

End of Life (EOL)

The electronic characteristics of a pulse generator when it should be replaced.

Escape Interval

The time between a paced or sensed cardiac event and the subsequent pacing stimulus in the same chamber, provided there is no hysteresis. The escape interval is measured in milliseconds.

Event Counter

A mechanism of the circuitry that the counts output pulses or intrinsic cardiac events. The information compiled by the event counter is stored in the pulse generator and may be obtained via telemetry.

Exit block
The failure of a pacing stimulus to capture the heart. Exit block occurs when the stimulation threshold of the heart exceeds the output of the pacemaker.

Fallback
A programmable upper rate response of dual-chamber pacemakers. Fallback occurs when the intrinsic atrial rate exceeds the rate at which ventricular output pulses can occur in 1:1 synchrony with intrinsic atrial events, that is at the maximum tracking rate or upper rate limit. The ventricular paced rate decelerates to and is maintained at a programmable fallback rate that is lower than the original programmed maximum tracking rate.

Far-Field Potential
An intrinsic electrial signal visible on an intracardiac electrogram. A far-field potential may originate from pectoral, myocardial cells or other muscle cells outside the heart. When a ventricular depolarization is visible on the atrial electrogram or an atrial depolarization on the ventricular electrogram the term far-field potential is also used.

Fusion beats
In pacing, a fusion beat may occur when an intrinsic cardiac depolarization of a particular chamber merges with an output pulse of a pacemaker. Fusion beats, as seen on the ECG exhibit varying morphologies i.e. from nearly pure paced beats to nearly pure spontaneous beats.

HV interval
The period of time that it takes for a wave of depolarization to travel from the His bundle to the ventricular myocardium. In electrophysiological studies, the HV interval is measured as the interval between the onset of the His deflection to the earliest onset of the ventriculardeflection in any of the simultaneously recorded surface ECG leads.
Normal values range from 30-55ms. A prolonged HV interval, especially one greater than 100 ms is a strong predictor for the impending development of complete heart block.

Hysteresis
In cardiac pacing hysteresis is the extension of the escape interval after a sensed event. Hysterseis increases the escape interval following an intrinsic event to allow more time for intrinsisc activity.

ICHD code
Intersociety Commission on Heart Disease Resources code. A system of letters to describe pacing modes. Originally it was 3 letter code but was subsequently extended to a 5 letter code. The first letter represents the heart chamber(s) being paced; the second letter represents the heart chamber(s) being sensed by the pacemaker; the third letter represents the mode of response to the sensed events; and the fourth letter represents the programmability; and the fifth letter represents any special antitachyarrhythmia function. Usually only the first three letters of the code are used.

Impedance

The total barrier to the flow of an electrical current in a conductive system. It is a complex value that includes next to the pure electrical resistance (or Ohms resistance), the resistance of the myocardial interface and myocardial tissue and the opposition to current produced by the capacitive and inductive characteristics of the system.

Inhibited

A pacemaker reponse in which a stimulus is suppressed in response to a sensed event.

Interference rate

See noise rate

Intrinsic Deflection

The large, rapid peak to peak movement seen in atrial and ventricular electrograms. The intrinsic deflection correlates with the local conduction, that is the depolarization of cardiac cells adjacent to the electrode from which the signal is recorded.

Joule

The international unit of measurement for energy. When one coulomb of charge moves from one terminal of a 1 volt battery to the other an energy of 1 joule is dissipated. In pacing microjoules are used to quantify the amount of energy delivered by the output of the pulse generator.

Latency

The interval between the introduction of and the response to a stimulus. In pacing, latency describes the interval between the output pulse and the subsequent depolarization.

Lead Configuration

The form or shape of a pacing lead, for example an atrial J lead.

Lead Impedance

The opposition to current by the components of the pacing system, that is the lead terminal pin, the conductor(s) and the electrode(s).

Lithium Iodine

A battery chemistry in which the anode is lithium and the cathode is iodine. Lithium iodine batteries provide 2.8 volts of potential per battery cell.

Lower Rate

The preset or programmed rate at which the pacemaker will emit an output pulse in the absence of intrinsic cardiac activity.

Lower Rate Interval

The preset or programmed maximum period of time (interval) after which the pacemaker will emit an output pulse in the absence of intrinsic cardiac activity.

Magnet mode
A pacing mode actived when a magnet is placed over the pacemaker. The magnet mode usually consists of fixed-rate pacing that contiues as long as the magnet is held over the pulse generator. The rate and pulse width of the magnet mode differs from pacemaker to pacemaker and especially from manufacturer to manufacturer.

Marker Channel
Data derived from the pacemaker (telemetrically), indicating what the pacemaker is doing (pacemaker timing). The marker channel indicates when the pacemaker senses or paces, as well as when the refractory period begins and ends in one or both channels.The marker channel recording may be superimposed on the surface ECG or may be recorded as an independant tracing.
Marker channel is a registered trade mark of Medtronic Inc.

Maximum Atrial Tracking Rate
The fastest atrial rate that can be sensed by the atrial channel and can result in ventricular stimulation, provided that the latter is allowed by the programmed upper rate interval. The maximum atrial tracking rate is limited by the total atrial refractory period. For example, if the TARP = 400 ms, the maximum atrial tracking rate is limited to 150 bpm (60.000 divided by 400 ms). If the atrial rate exceeds the maximum tracking rate, a 2:1 or even higher degree of AV bloick will occur, depending upon the atrial rate.

Mode
The preset or programmed response of a pacemaker. For instance, the VVI mode is a preset response of a pacemaker to operate as a ventricular inhibited pacemaker unit.

Myopotential Inhibition
The inhibition of an output pulse (atrial or ventricular) by electriocal signals that do not originate from the atrium or ventricle but from skeletal muscle. Myopotential inhibition occurs commonly in unipolar pacing systems.

Myopotential Triggering
The triggering of a pacemaker output pulse by electrical signals that originate from skeletal muscle. For instance the atrial channel of a dual-chamber pacemaker senses a myopotential signal as intrinsic atrial activity and triggers a ventricular output pulse after completion of the AV interval.

Noncapture
No depolarization in response to an electrical stimulus emitted by the pacemaker.

No output
No current delivered to the myocardium by the pacemaker. This can be caused by failure of pulse formation or pulse transmission.

Noise
The current or voltage that can interfere with an electrical device or system. In cardiac pacing, noise refers to the extraneous spike, waveform or signal from the external environment.

Noise rate
Reversion of the pacemaker to asynchronous pacing due to sensing of extraneous electrical (noise) signals. Signals should occur at a certain rate and sensed in subsequent relative refractory periods. The number of sensed events necessary to reverse to asynchronous pacing is determined by the specifications of the pulse generator.

Noise Sampling Period
The period of time during the refractory period of the pacemaker when the sensing channel is alert for noise signals. If noise is detected, the pacemaker changes to its noise rate. Noise sampling periods may occur in the atrial as well as in the ventricular channel. This period is also called relative refractory period.

Noncommitted
A type of AV sequential pacing in which ventricular sensing occuring during the programmed AV interval will inhibit and not commit a ventricular pace output.

Oversensing
The sensing of undesired electrical signals by the pacemaker amplifier. For example, myopotentials, electromagnetic interference, T waves. Depending on the programmed pacing mode oversensing may cause inhibition or triggering of an output pulse.

Pacemaker
A pacing system consisting of a pulse generator connected to a lead with a distal tip with one or two electrodes. The system is called a pacemaker.

Pacemaker Circus Movement Tachycardia (PCMT)
Sometimes also called Artificial Circus Movement Tachycardia or less specifically Pacemaker Mediated Tachycardia or Endless Loop Tachycardia. A Pacemaker Circus Movement Tachycardia is an artificial reentry tachycardia in which the retrograde limb of the circuit is provided by the AV node or accessory pathway, while the anterograde limb is by the way of AV synchronization of the pacemaker.

Pacemaker Mediated Tachycardia (PMT)
Nonphysiological tachycardia maintained by the pacing system.

Pacemaker Syndrome
A syndrome that may be characterized by dizziness, faintness, tightness in the chest, shortness of breath, during episodes of ventricular pacing. Pacemaker syndrome is most commonly seen in patients with 1:1 VA conduction during ventricular pacing, but also in patients with AV dissociation during ventricular pacing. The loss of AV synchrony is responsable for the patient's complaints.

Physiologic Pacing
Commonly used to refer to dual-chamber pacing that mimicks, the atrio-ventricular contraction sequence of the normal heart. Current physiological pacing systems optimize the atrial contribution to cardiac output. The cardiac output may increase at rest by 10-30% over that of single chamber ventricular pacing systems.

Postventricular Atrial Refractory Period (PVARP)
The time after a ventricular paced or sensed event during which the lower rate and upper rate timer cannot be reset by any atrial sense event.

Pseudofusion beat
Emission of a pacemaker pulse simultaneous or nearly simultaneous with intrinsic cardiac activity, without producing fusion.

Pseudopseudofusion beat
Emission of an atrial stimulus simultaneous or nearly simultaneous with intrinsic ventricular activity. Has been observed in dual chamber systems programmed in the DVI mode.

Pulse Amplitude
Pulse output expressed in Volts

Pulse Generator
A pacing system consists of a pulse generator connected to one or two leads. Such a pacing system is called pacemaker. The pulse generator represents the power supply and the electronic circuitry of the pacing system.

Pulse Duration
The length of time during which an electrical stimulus is applied to the heart by the pacemaker. Pulse duration is expressed in milliseconds.

P-wave synchronous pacing
Pacing in which sensed atrial events trigger ventricular pace outputs unless the latter are inhibited. After intrinsic atrial activity is sensed a preset AV interval is initiated and at the end of the AV interval the ventricle is stimulated.

Rate Limit
Maximum rate at which permanent stimulation can be performed. Protection circuit against high rate failures (pacemaker runaway) and high rate pacing in the triggered mode (AAT, VVT). In some pulse generators the rate limit can be temporarily disabled in order to perform overdrive pacing.

Rate Responsive Pacing
Pacing in which an automatic increase or decrease in the paced ventricular rate occurs in response to changes in the intrinsic atrial rate. Rate responsive pacing is also called rate

modulated or rate adaptive pacing. Rate responsive pacing is also refers to automatic adjustments in the pacing rate in response to variables (signals) other than P waves to increase the pacing rate. Adaptive rate pacemakers utilize various types of sensors that respond to physical, chemical or electrical signals.

Rate Smoothing
A response of the pacemaker that prevents the paced rate, either atrial or ventricular, from changing by more than a programmed percentage from one cardiac cycle to the next. Rate smoothing is also used as an upper rate response in some dual-chamber pacemakers to prevent sudden changes in rate when the atrial upper rate is exceeded.

Redundancy
The deliberate duplication of essential components or functions in a system in order to decrease the probability of a system failure by providing an alternate arrangement for use upon failure of the primary arrangement.

Relative refractory period
See noise sampling period.

Resistance
Electrical. Opposition to the flow of a current through a conducting material.

Retrograde Conduction
Ventriculo-atrial conduction as opposed to anterograde or normal conduction from atrium to ventricle.

Rheobase
The voltage (or the current) corresponding to an infinitely large pulse width in the strength-duration curve (plateau of the curve) is called the rheobase.

Sensing
The ability of a pacing system to detect intrinsic cardiac activity. Detection depends upon the amplitude and frequency content of the signal.

Slew Rate
Slew rate corresponds to the maximum voltage change per unit time (rate of change of voltage dV/dt) usually contained within the intrinsic deflection.

Strength-Duration Curve
With a pulse generator of a variable output pulse (the pulse amplitude and/or the pulse width), one can measure the lowest pulse amplitude for a given pulse width or the lowest pulse width at a fixed amplitude at which the heart is captured. The minimum value is called the threshold, i.e. the voltage or current threshold at a certain pulse width or the pulse width threshold at a certain current of voltage. By measuring voltage or current thresholds for different pulse widths, or vice versa, and by plotting them on a graph with voltage or current on the vertical axis and the pulse

width along the horizontal axis, one obtains the strength-duration curve. For all amplitude and pulse width combinations above the strength duration curve, the heart is effectively stimulated, and for points below the curve, electrical stimulation is ineffective. For very large pulse widths, the amplitude necessary for effecive stimulation is low and increases with shortening of the pulse width.

Total Atrial Refractory Period (TARP)
The length of time during which the atrial sensing amplifier is unresponsive to input signals. TARP equals the sum of the AV interval and postventricular atrial refractory period (PVARP).

T wave Recycling
The resetting of a pacemaker timing cycle when a T wave is sensed by the pacemaker and is interpreted as a spontaneous ventricular depolarization.

Unipolar
In a coventional unipolar pacing system, one electrode (the cathode) is external to the pulse generator and is located at the distal tip of the pacing lead. The other electrode (anode) is the metal case of the pulse generator.

Upper Rate Interval (URI)
The shortest interval between two ventricular paced events or a sensed ventricular event followed by a paced event.

VAT
The Intersociety Commission on Heart Disease Resources code for atrial synchronized ventricular pacing. Pacing occurs in the ventricle (V), sensing in the atrium (A) and the mode response is triggered (T). A sensed atrial event triggers a ventricular output pulse.

VDD
The Intersociety Commission on Heart Disease Resources code for atrial synchronized ventricular inhibited pacing. Pacing occurs in the ventricle (V). Sensing in the atrium and ventricle (D=dual). An atrial sensed event triggers a ventricular output. A sensed ventricular event inhibits a ventricular output. The mode of response is therefore triggered as well as inhibited (D=dual). In the absence of cardiac activity the ventricle is paced at the programmed pacing interval.

Ventricular Refractory Period (VRP)
The time after a ventricular paced or sensed event during which the lower rate and upper rate timer cannot be reset by any ventricular sense event.

VOO
The Intersociety Commission on Heart Disease Resources code for asynchronous pacing in the ventricle. Also called fixed rate pacing. Pacing occurs in the ventricle (V); the mode response is fixed or asynchronous (O) at the programmed pacing interval. There is no sensing of spontaneous ventricular activity (O).

VVT

The Intersociety Commission on Heart Disease Resources code for ventricular triggered pacing. Pacing and sensing occurs in the ventricle (V); the mode of response is triggered (T). A ventricular pace response is generated;

(1) when ventricular activity is sensed during the ventricular sensing interval. In the presence of spontaneous ventricular activity this response does not contribute to the ventricular depolarization. The escape interval of the pacemaker is reset.

(2) when ventricular activity is not sensed during the ventricular sensing interval. A ventricular pace response is generated at the end of the programmed pacing interval. This response results in ventricular depolarization.

Watt

The International unit of electrical power. One watt is dissipated when one ampere of current (Coulomb/second) flows through a resistance of one Ohm. Watts may also be expressed as Joules per second.

Wenckebach reponse

A pacemaker-Wenckebach response occurs whenever the sensed atrial rate is faster than the ventricular upper tracking rate (upper rate interval). In such cases, the sensed AV interval may be prolonged until the ventricular upper tracking rate timer (upper rate interval) expires. The AV interval continues to elongate as the atrial rate rises until an atrial event falls into the postventricular atrial refractory period, thereby stopping the initiation of a pacemaker AV interval and the subsequent ventricular pacing output.

Nawoord

Het schrijven van een woord van dank aan het einde van dit proefschrift is niet eenvoudig. De kennis vergaard voor het schrijven hiervan stamt niet uit de laatste drie en een half jaar, maar strekt zich uit tot de afgelopen decennia. Velen hebben hierin een rol van betekenis gespeeld en een totaal opsomming zou mij in conflict brengen met de voorschriften in het promotieregelement, wat zegt dat dankbetuigingen slechts "in bescheiden vorm" vermeld mogen worden. Daarom wil ik iedereen bedanken die in het recente of verdere verleden het mij mogelijk gemaakt hebben dit proefschrift te voltooien.

Mijn bijzondere dank gaat echter uit naar:

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Dr.ir. Hans Blom, mijn co-promotor, voor zijn opbouwende kritiek, zijn snelle respons op de door mij geschreven hoofdstukken en zijn onontbeerlijke hulp bij de realisatie van het prototype expert system en hoofdstuk IX.

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Ir. René Bourgonje, die in staat is geweest mijn hoofdstukken over pacemaker falen om te zetten in een eerste model van een expert system, waarvan de resultaten veelbelovend zijn.

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Mamdouh verdient hierin een extra vermelding vanwege onze reeds meer dan 25 jaar durende vriendschap en zijn begeleiding toen ik probeerde mijn eerste artikelen op papier te zetten. Zijn bijdrage aan de artikelen welke de basis hebben gevormd voor dit proefschrift is van zeer grote waarde geweest.

Frank Bracke, voor zijn werk in het EFO-lab, waarin hij met veel geduld en vakmanschap mij de gevraagde plaatjes die de morphologie van het gestimuleerde complex illustreren, heeft geleverd.

Curriculum Vitae

The author of this thesis was born on May 26 1944, in Eindhoven, The Netherlands. He attended secondary school at the Sint Joris College in Eindhoven. In 1963 he started working in the former R.K. Binnenziekenhuis in Eindhoven and studied electronics at night at the R.K. Uitgebreid Technische School (1966) and the Instituut voor Hoger Beroepsonderwijs (1968), both in Eindhoven.

Since 1967 he worked in the department of Cardiology and in 1973 he became head of the Department for Cardiac Catheterization and Pacemaker Clinic. In 1980 he was elected in the board of the Dutch Working Group on Cardiac Pacing and is secretary of the Working Group since 1982. He is member of the board of the Dutch Pacemaker Registry since 1982 and consultant of the Dutch Heart Foundation since 1983. He is part-time teacher at the Hogeschool Eindhoven.

Stellingen

behorende bij het proefschrift

The ECG in the Evaluation of Pacemaker Function and Diagnosis of Malfunction

door
Berry van Gelder

1. Voor de beoordeling van pacemaker malfunctie verdienen lange, meer-kanalige ECG registraties de voorkeur boven het standaard formaat 12 afleidingen ECG (dit proefschrift).

2. Registraties van pacemaker ECG's, verkregen uit digitale apparatuur, waarbij de pacemaker impuls vaak kunstmatig gegenereerd wordt, dienen voor de beoordeling van pacemaker functie met de nodige achterdocht bekeken te worden (dit proefschrift).

3. In de huidige impulsgeneratoren is de "stoorfrequentie" vaak gelijk aan de geprogrammeerde stimulatie frequentie. Om reden van herkenbaarheid op het ECG als wel om interferentie met het spontane hartritme te voorkomen zou het aanbeveling verdienen hier een andere waarde voor te kiezen welke dan ook hoger dient te liggen dan de geprogrammeerde frequentie (dit proefschrift).

4. Digitale Röntgen technieken verdienen de voorkeur boven conventionele doorlichting of "harde" thoraxfoto's voor het opsporen van fouten in de elektrische integriteit van het pacemaker systeem (dit proefschrift).

5. Uitbreiding van pacemaker programmeringsapparatuur met een kennissysteem voor functiediagnose, zal leiden tot een snellere en verbeterde diagnostiek (dit proefschrift).

6. Het feit dat een groot aantal van de huidige impulsgeneratoren met 2 sensoren wordt uitgerust, betekent dat de ideale sensor nog niet gevonden is.

7. Belangrijke buitenlandse congressen zijn gebleken de beste volume beperkende maatregel te zijn in de gezondheidszorg.

8. Als autoverzekeringen dezelfde regels zouden hanteren als ziektekostenverzekeringen was de Nederlandse bevolking reeds lang in opstand gekomen.

9. In de interventie cardiologie worden door fabrikanten nog steeds 3 verschillende eenheden gebruikt om de dimensie van de gebruikte materialen aan te geven. Desondanks zal het de operateur niet ontgaan dat de buitendiameter nog steeds groter zal blijven dan de binnendiameter van een catheter.

10. Het toenemend aantal management opleidingen vormt een belemmering voor het werkproces, dat door middel van deze opleidingen verbeterd zou moeten worden.

11. Bij de huidige staat van onderhoud van wegen binnen de bebouwde kom lijkt de aanleg van nog meer verkeersdrempels op geld verspilling.

12. Gezien de handelswaar zou "drugstore" een toepasselijker naam zijn dan "coffee shop".

Eindhoven, 2 november 1995