Single and dual chamber pacing in patients with conduction disorders

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SINGLE AND DUAL CHAMBER PACING
IN PATIENTS
WITH CONDUCTION DISORDERS

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PROEFSCHRIFT

TER VERKRIJGING VAN DE GRAAD VAN DOCTOR
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All work and no play makes Jack a dull boy.

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CHAPTER 1

INTRODUCTION

The clinical application of cardiac pacing has evolved and expanded since the introduction of endocardial stimulation in the early 1960s (1-4). At present permanent cardiac pacing is employed in patients with symptomatic conduction disorders as well as in patients with certain types of tachyarrhythmias.

In this dissertation some aspects of cardiac pacing in patients with symptomatic conduction disorders are presented. Permanent pacing in patients with supraventricular or ventricular tachycardias is beyond the scope of this work.

Patients with symptomatic conduction disorders

In this chapter the clinical manifestations of sinus node dysfunction and atrio-ventricular (AV) block are discussed. In addition, the clinical evaluation and management of patients with these conduction disorders will be presented. For the sake of clarity, the two types of conduction disorders are discussed as isolated clinical problems. In general this separation reflects reality, however in a minority of patients, sinus node dysfunction and AV block may be present simultaneously.

SINUS NODE DYSFUNCTION

1. Clinical manifestations

Sinus node dysfunction is associated with a variety of nonspecific signs and

symptoms. In 1974 Ferrer (5) presented a completely understandable picture of the complex clinical problem encountered in patients with dysfunction of the sino-atrial node. She was the first to group the following electrocardiographic observations under the title "Sick Sinus Syndrome":

- a. persistent, or unexpected sinus bradycardia (50 beats/min or less);
- b. short periods of sinus-arrest leading to profound episodes of cardiac asystole (3-10 seconds);
- c. inability of the heart to resume sinus rhythm after DC cardioversion from atrial fibrillation or atrial flutter;
- d. chronic atrial fibrillation occurring with a slow ventricular response that cannot be related to the effects of digitalis; the slow ventricular rate is due to coexisting atrio-ventricular node dysfunction, producing various degrees of atrio-ventricular block;
- e. episodes of sino-atrial exit block not related to drugs;
- f. bradycardia alternating with supra-ventricular tachycardia.

2. Clinical evaluation

History

The clinical symptoms related to sinus node dysfunction are: syncope, dizziness, lightheadedness and dyspnea. Syncope, dizziness and lightheadedness express cerebral hypoperfusion, whereas dyspnea in this setting is an expression of heart failure. Information on the duration and severity of these complaints, together with information gathered about other medical problems that may cause episodic dizziness or syncope, are the cornerstones of subsequent decision making. Table I shows a classification of the common causes of recurrent dizziness and syncope. In addition information is obtained on pharmacologic therapy, that may adversely affect sinus node function. It is established that antiarrhythmic drugs such as quinidine, flecainide, procainamide, disopyramide, cardiac glycosides, amiodarone, beta-adrenergic and calcium entry blocking agents, can severely depress sinus node function (6). In addition, lithium and anti-hypertensive agents such as clonidine, alpramethyldopa, reserpine and guanethidine may also depress sinus node function (7).

Physical Examination

Although self-evident, it is essential that a complete physical examination

TABLE 1*

**CLASSIFICATION OF CAUSES OF RECURRENT WEAKNESS, FAINTNESS
AND DISTURBANCES OF CONSCIOUSNESS**

-
1. CIRCULATORY
 - A. CARDIAC
 - BRADYCARDIA : SINUS NODE DYSFUNCTION, AV BLOCK, CAROTID SINUS HYPERSENSITIVITY
 - TACHYCARDIA : VENTRICULAR OR SUPRAVENTRICULAR TACHYCARDIAS
 - HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY
 - AORTIC STENOSIS, ATRIAL MYXOMA, ATRIAL THROMBUS
 - B. PERIPHERAL
 - POSTURAL ORTHOSTATIC HYPOTENSION
 - PSYCHOGENIC SYNCOPE
 2. OTHER CAUSES
 - A. METABOLIC AND BIOCHEMICAL
 - HYPOGLYCAEMIA
 - HYPERVENTILATION
 - INTERNAL HEMORRHAGE, HYPOVOLEMIA
 - DRUGS: CERTAIN SEDATIVES AND ANTIHYPERTENSIVE AGENTS
 - B. CEREBRAL
 - EPILEPSY
 - CEREBRAL VASCULAR DISTURBANCES
 - EMOTIONAL DISTURBANCES
-

* Modified after R.D. Adams and T.R. Harrison (69).

may offer clues to causes of recurrent dizziness and syncope (Table 1). With regard to sinus node dysfunction no specific signs exist.

Carotid sinus stimulation

Recently several reports appeared about an association between hypersensitive carotid sinus syndrome and sick sinus syndrome.

Mandel et al. (8) observed in their series of 31 patients with suspected sinus node dysfunction a hypersensitive carotid sinus response (sinus pause > 3 seconds) in 7 patients (23%). Similar findings were reported by Thormann et al. (9) and Morley et al. (10). Although carotid sinus hypersensitivity originally connoted a rather specific clinical entity, it is now viewed by these investigators as having a considerable broader meaning. Nevertheless, we would like to emphasize that the clinical relevance of this finding remains unclear, since a marked bradycardia response to carotid sinus mas-

sage may occur in patients without clinical symptoms or prior suspicion of carotid sinus hypersensitivity (11).

From a diagnostic and therapeutic standpoint differentiation between the two syndromes is important. In patients with sinus node dysfunction, pacemaker therapy is likely to alleviate symptoms. In patients with clinical evidence of carotid sinus hypersensitivity, pacemaker therapy is only effective in the subset of patients with the cardio-inhibitory type of carotid sinus hypersensitivity. In patients with the vasodepressor type or mixed type (cardio-inhibitory and vasodepressor) the value of pacemaker therapy has not been unequivocally proven (12).

24-Hour Holter recording and ECG transmission via telephone

The most definitive method to diagnose sinus node dysfunction is to document an inappropriate sinus bradycardia, sinus arrest, sinus atrial block or bradycardia alternating with tachycardia. At present, ambulatory monitoring and ECG transmission via the telephone have become the most important tools to demonstrate the presence or absence of a relationship between symptoms and ECG abnormalities.

Exercise testing

Although the purpose of exercise testing is to evaluate the ability of the sinus node to accelerate in an appropriate fashion in response to physical stress, it is still unclear if an abnormal heart rate response to exercise will facilitate the identification of patients with sinus node dysfunction (13).

Pharmacologic testing

The effects of the autonomic nervous system on sinus node function has led some investigators to recommend that heart rate response to sympathomimetic, sympatholytic, vagotonic and vagolytic drugs must be employed routinely in the clinical evaluation of patients suspected of sinus node dysfunction due to the effects of the autonomic nervous system, Jose et al. (16, 17) have described a technique to determine the intrinsic sinus rate, i.e. the rate of spontaneous sinus node depolarization independent of the effects of the autonomic nervous system. To obtain pharmacologic denervation, atropine (0.04 mg/kg) and propranolol (0.2 mg/kg) are intravenously administered, and the sinus rate representing the intrinsic heart is

measured. The intrinsic heart rate is age dependent and it can be defined by the following regression equation: $IHR = 117.2 (0.53 \times \text{age})$. A depressed intrinsic heart rate will be observed in patients with sinus node disease, whereas in patients with a normal intrinsic heart rate the documented bradycardia is attributed to a disturbance of autonomic regulation (16, 17). Since this test may accurately identify patients who have sinus node disease, it is interesting from a diagnostic standpoint. The meaning of this test in terms of management and pacemaker therapy however is unclear.

Invasive electrophysiologic evaluation

Over the past decade, a number of invasive electrophysiologic techniques have been developed to evaluate sinus node function. Two possible pathophysiologic mechanisms underly sinus node dysfunction. First, disturbances of sinus node automaticity and second, disturbances of sino-atrial conduction. Sometimes both abnormalities exist simultaneously.

The tests used to assess sinus node automaticity and sino-atrial conduction time will be discussed. In addition, the value of recording the sinus node electrogram and the assessment of sinus nodal refractoriness will be presented.

- Sinus node automaticity

Determination of the sinus node recovery time (SNRT) was originally described by Mandel et al. (18). Atrial pacing is initially performed at a rate approximately 20 beats/min faster than the spontaneous sinus rate. Pacing is continued for 30, 60, or 180 seconds (s) at which time it is abruptly terminated. SNRT is measured in milliseconds (ms) as the time elapsing from the last paced P wave to the first spontaneous beat of sinus origin (Fig. 1, A, B, C). Pacing trials are comprised of atrial pacing at progressively faster rates, often in rates with increments of 20 beats/min up to a rate of 170 beats/min.

To control for differences in spontaneous sinus rate, many authors use the corrected SNRT in assessing sinus node automaticity. The corrected SNRT is defined as the difference between the SNRT and the spontaneous sinus cycle length (19, 20). Although accurate definition of the upper limit of normal for SNRT is essential for a correct interpretation, many studies offer a wide range of normal values, i.e. 1200 ms to 1750 ms for SNRT and 375 ms to 550 ms for corrected SNRT (19, 20).

Fig. 1. Sinus Node Recovery Time.

- The atrium is paced at a rate of 80 beats/min. When atrial pacing is abruptly terminated the SNRT is measured in ms as the time elapsing from the last paced P wave to the first spontaneous atrial beat of sinus origin. A SNRT is measured of 1000 ms.
- Atrial pacing at a rate of 100 beats/min. A SNRT is measured of 1200 ms.
- Atrial pacing at a rate of 120 beats/min and a SNRT of 1360 ms.
Pacing the atrium at approximately 120 beats/min for at least 15 s results in reproducible SNRTs (18).

In patients with *prolonged recovery times*, Chadda et al. (21) have also suggested measuring SNRTs and calculating the corrected SNRTs after autonomic blockade has been achieved according to Jose's pharmacologic denervation technique (16). When SNRTs remain pathologically prolonged after pharmacologic denervation sinus node function is disturbed, whereas autonomic dysregulation of the sinus node is present when subsequently normal SNRTs are measured.

In patients with *normal recovery times*, Reiffel et al. (22) have emphasized that sinus node function is normal only when one can demonstrate the absence of atrio-sinus entrance block. Reiffel and coworkers were the first to note that maximum SNRTs in patients with sinus node dysfunction frequently occurred at rates much slower than those seen in normal subjects. To explain this finding they suggested that in patients with sinus node dysfunction, atrio-sinus entrance block also occurred at relatively slower atrial pacing rates than in normal subjects.

When atrial pacing trials are performed at different pacing rates, according to Mandel's description (18), atrio-sinus entrance block will always be recognized. On the other hand, when atrial pacing is performed at only one pacing rate, the presence of atrio-sinus entrance block might not be appreciated and the time elapsing from the last paced beat to the first spontaneous sinus beat may erroneously be interpreted as a normal SNRT.

- Sino-atrial conduction time

(1) Premature atrial stimulation

Analysis of the sinus node response to atrial premature beats was originally described by Strauss et al. (23). Fig. 2 illustrates the various types of sinus node response to premature atrial stimuli. Fig. 3 illustrates the calculation of the unidirectional sino-atrial conduction time (SACT). Reported normal values for calculated SACTs vary from 50 ms to 120 ms (24, 25).

(2) Atrial overdrive pacing

Narula et al. (26) have described an alternative method to determine SACT, utilizing atrial overdrive pacing. This method assumes that pacing the atrium at a rate slightly faster than the spontaneous sinus rate will not result in suppression of sinus node automaticity, but that the pacing beats will gradually achieve capture and reset the spontaneous sinus cycle. The atrium is paced at a rate < 10 beats/min faster than the spontaneous sinus rate for 8 consecutive beats. The interval from the last paced beat to the

Fig. 2. Sinus node response to atrial extrastimuli.

- Compensation.** An atrial extrastimulus is delivered with a coupling interval of 850 ms. This premature beat is followed by a spontaneous sinus beat after a pause of 1150 ms. Since $850 + 1150 = 2000$ ms and 2000 ms is exactly two times the basic sinus cycle length, the compensatory pause is complete.
- Resetting.** An atrial extrastimulus is delivered with a coupling interval of 650 ms. The premature beat is followed by a spontaneous sinus beat after a pause of 1150 ms. Since $650 + 1150 < 2000$ ms, the compensatory pause is incomplete. Therefore the sinus node has been depolarized by the atrial premature beat causing the next sinus depolarization to occur earlier.
- Interpolation.** An atrial extrastimulus is delivered with a coupling interval of 400 ms. The premature beat is followed by a spontaneous sinus beat after a pause of 600 ms. Since $400 + 600 = 1000$ ms and 1000 ms is exactly the basic sinus cycle length, the normal sinus discharge rate is not effected by the atrial premature beat.
- Reentry.** An atrial extrastimulus is delivered with a coupling interval of 300 ms and is followed by an early spontaneous discharge of the sinus node. The sum of the two intervals 700 ms ($300 + 400$), is less than the spontaneous sinus cycle length. Provided that the first atrial beat following the atrial premature beat is of sinus origin, this beat is called a sinus node echo beat.

(From Jordan et al., reference 20. By permission of the publisher.)

first return sinus beat is taken as the time (T) representing the sum of retrograde sino-atrial conduction time (RSACT), sinus cycle length (SCL) and anterograde sino-atrial conduction time (ASACT). Assuming that RSACT equals ASACT, unidirectional SACT equals $(T - SCL)/2$. This assumption has not been proven. Since there may be differences between anterograde and retrograde conduction velocities in the AV node, His bundle, accessory pathways and Purkinje fibers, why should the anterograde and retrograde conduction time be the same in the sino-atrial node (27)?

Fig. 3. Sino Atrial Conduction Time (SACT).

The horizontal axis represents the coupling interval of atrial extrastimuli with the preceding spontaneous sinus beats expressed as a percentage of the spontaneous sinus cycle length. The vertical axis represents the intervals between the atrial premature beat and the following spontaneous sinus beat, also expressed as a percentage of the spontaneous sinus cycle length. A, B, C and D correspond with the atrial extrastimuli delivered at resp. 850 ms, 650 ms, 400 ms and 300 ms as explained in Fig. 2. SACT is calculated as follows: The A2A3 interval during resetting of the spontaneous discharge of the sinus node is 1150 ms. The spontaneous sinus cycle length is 1000 ms (A1A1). $A2A3 - A1A1 = 2 \times \text{SACT}$. $\text{SACT} = 1150/2 - 1000/2 = 150/2 = 75$ ms, provided that the atrio-sinus conduction time equals the sino-atrial conduction time.

(From Jordan et al., reference 20. By permission of the publisher.)

SACTs calculated with this method are reproducible and are comparable to those calculated according to the method of Strauss. The advantage of this method is that complex equipment and laborious calculations required for the Strauss method became superfluous. A disadvantage of this technique as well of the Strauss method is, that SACTs are determined indirectly. Although SACT can be precisely and directly measured using the micro-electrode technique, this can only be performed in the experimental animal laboratory.

- Recording of the sinus node electrogram

At present it is unclear whether direct sinus nodal recordings can separate patients with normal from those with abnormal sinus node function (28).

- Sinus node refractoriness

Goldreyer and Damato (29) suggested that critically coupled atrial premature impulses that become interpolated are caused by sinus node refractoriness. Kerr and Strauss (30) verified this contention on isolated rabbit sinoatrial preparations with the microelectrode technique and concluded that the sudden transition between the zone of reset and the zone of interpolation was a good indication of the sinus node refractory period. They extended their observations to man and demonstrated a clear difference in sinus nodal refractoriness between normal individuals and patients with sinus node dysfunction (31). However this is the only reported study in man and additional studies of this nature are necessary to establish the role of this parameter in the diagnosis of sinus node dysfunction.

Do invasive electrophysiologic studies give additional information on patients suspected of sinus node dysfunction?

In terms of underlying pathophysiologic mechanisms the answer is yes. Electrophysiologic testing before and after autonomic blockade will aid in separating true sinus node dysfunction from sinus node dysfunction due to autonomic effects in patients with prolonged SNRTs. In contrast, the value of SNRT and CSNRT as tests to establish a diagnosis of sinus node dysfunction, is only rarely of clinical benefit. Sensitivity and specificity as statistical indexes of the efficiency of these tests have been calculated (18, 19, 23-25). Almost all patients with a pathologically prolonged SNRT or CSNRT have sick sinus syndrome. However, in the majority of patients with sick sinus syndrome no abnormal prolongation of SNRT and CSNRT have been found (18, 19, 24). Since the purpose of the test is to identify patients with sinus node dysfunction, a positive result (prolonged SNRT and CSNRT) will only be observed in a minority of cases.

The incidence of an abnormal prolongation of SACT in patients with sick sinus syndrome varied in different series from 29 to 38 percent, whereas calculated SACT of ostensibly normal subjects was prolonged in 15 to 22 percent (22-25). Therefore, SACT calculation is also of limited value to identify patients with sinus node dysfunction. Even when the results of the 2 tests are combined (SACT and SNRT), as was done in a study by Seipel et

al. (24), the diagnostic value is small.

In addition, it is not yet known if assessment of sinus node refractoriness or recording of the sinus node electrogram will offer useful clinical information.

3. Management

Pacemaker therapy for sinus node dysfunction is only indicated when a relationship between symptoms and ECG abnormalities characteristic for sinus node dysfunction can be documented, either by ECG, Holter recording, monitoring by telemetry or ECG transmission via telephone. A thorough medical and neurologic work-up is also essential. Such a work-up should include a detailed medical and pharmacologic history, routine laboratory examination, neurologic consultation and carotid sinus stimulation during continuous ECG monitoring (9-11).

Only when central nervous system symptoms or dyspnea have been shown to be associated with episodes of sinus node dysfunction and failure of subsidiary pacemakers to escape, then cardiac pacing will be successful in eliminating these symptoms (32). Regardless whether the bradycardia is due to intrinsic sinus node dysfunction or autonomic dysregulation of the sinus node function, pacemaker implantation is indicated in symptomatic patients.

There is no further need to perform exercise tests, to administer drugs to assess the influences of the autonomic nervous system on sinus nodal function or to determine SNRTs and SACTs (Fig. 4).

Fig. 4. Decision making tree in patients with sinus node dysfunction.

ECG & SND = patients with electrocardiograms characteristic for sick sinus syndrome; S+ = subset of symptomatic patients; S- = remaining patients without symptoms; PM = Pacemaker Implantation; W/S = wait & see.

With regard to pacemaker selection, it is important to assess if anterograde and retrograde AV conduction are intact (33-37). Pacemaker selection will be discussed in the last section of this chapter. Assessment of anterograde and retrograde conduction will be discussed in Chapter 2.

ATRIO-VENTRICULAR BLOCK

1. Clinical manifestation

Atrio-ventricular (AV) block is an electrocardiographic diagnosis. It is customary to distinguish 3 types of heart block:

- first degree AV block, which means prolonged but not blocked AV conduction. Propagation of the atrial impulse to the ventricle is delayed at the level of the AV node, common His bundle or both;
- second degree AV block, which means that some but not all atrial impulses originating either in the sinus node or within the atrium reach the ventricle;
- third degree AV block or complete heart block, which means a complete failure of impulse propagation from the atrium to the ventricle. This is attributed to the inability of the AV conduction system to transmit atrial impulses in anterograde direction.

AV block may be either functional and potentially reversible or may be fixed, due to intrinsic disease of the AV conducting system. Complete heart block may occur to congenital or acquired lesions.

2. Clinical evaluation

History

The clinical symptoms related to AV block vary from recurrent syncope, dizziness or dyspnea to no complaints at all (42). Unfortunately, while symptoms may be completely lacking in patients with first and second degree AV block, they may also be absent in patients with third degree AV block.

It is also important to obtain information on drug therapy, since several drugs may severely depress AV conduction (43). In general, antiarrhythmic and anti-ischemic drugs that adversely affect sinus node function, may also depress anterograde AV conduction. The drugs most likely to compromise AV conduction are: digitalis, amiodarone, beta-adrenergic and calcium

entry blocking agents. Some of the patients in whom these agents must be continued for clinical reasons, require a permanent pacemaker because of drug induced heart block.

Physical Examination

The presence of regular A and V in the jugular venous pulse, a stable loudness of the first heart sound and regular diastolic filling sounds all establish AV synchrony. Intermittent cannon waves, varying loudness of the first heart sound and intermittent diastolic filling sounds suggest AV dissociation. If this occurs in patients with a slow heart rate, AV block is likely. In patients with a fast heart rate these findings suggest ventricular tachycardia.

ECG

The ECG may indicate the site of the AV block. In patients with *first degree AV block* a narrow QRS complex (< 0.10 s) is highly suggestive for an AV nodal localization (43, 50).

Second degree AV block is subdivided into 3 types: Type I, Type II and advanced second degree AV block (2 : 1 or 3 : 1 AV conduction). Type I block is characterized by a progressive lengthening of the PR interval before conduction of a P wave to the ventricle is blocked.

Different patterns of PR intervals may be noted before AV block occurs. However, the PR interval following the non conducted P wave is always shorter than the PR interval of the preceding beat.

Type I second degree AV block is most commonly localized in the AV node (45, 47). Type II block is characterized by the absence of PR interval lengthening before AV block occurs. The site of type II block is almost entirely limited to the His bundle or bundle branches.

For many years, it was generally accepted that Type I second degree AV block would not lead to sudden asystole, as escape pacemakers within the common His bundle were usually adequate to protect the patient. In contrast, patients with second degree AV block Type II would have a worse prognosis, since they have to rely on less stable escape pacemakers localized in the distal His Purkinje system. It has generally been accepted that second degree AV block type II is a harbinger of asystole as a result of the sudden onset of third degree AV block (43, 47-49, 52).

Recently, Shaw et al. (48) presented data which challenged the correctness

of these long held concepts. These investigators showed that 3 and 5 years survival rates in unpaced patients were similarly poor regardless of the type of second degree AV block. Based on this evidence these investigators suggested that electrocardiographic features can no longer be used to separate high and low risk patients with second degree AV block. The main limitation however of this study is its design. The investigators collected ECGs that were taken in the past of patients with second degree AV block and studied the follow-up of the paced and non paced patients from data in the patient's records. Retrospective studies may be clinically important, however they are the most unproductive methods devised for studying the natural history of a disease (60).

Advanced second degree AV block (2 : 1 or 3 : 1 AV block) may be a variant of either Type I or Type II block (47, 52). Unfortunately the site of block can not be predicted by means of the ECG alone.

In *third degree AV block*, QRS complexes can either be narrow or wide. Narrow QRS complexes (0.08-0.10 s) are only shown in patients with heart block localized in the AV node or common His bundle. Wide QRS complexes (0.10-0.12 s) may be present in AV nodal as well as infranodal block. However bizarre broadened QRS complexes with a width of > 0.12 s are almost exclusively observed in patients with AV block localized below the His bundle.

Carotid sinus stimulation

Carotid sinus massage may sometimes give a clue to the site of the antero-grade AV block. When this maneuver aggravates the conduction disorder i.e. a progression of first or second degree AV block to third degree AV block, the block is most likely localized in the AV node. In some patients a decrease in block is observed. In these cases it is important to measure the spontaneous sinus rate. Slowing of the sinus rate may result in improved AV nodal as well as infranodal conduction (43), since both AV nodal and infranodal second degree AV block may be rate dependant. In this setting, carotid sinus massage does not give a clue to the site of AV block. However, when the spontaneous sinus rate does not decrease, improved AV conduction suggests an infranodal site of the AV block. Carotid sinus pressure has very little effect on AV conduction in patients with complete heart block, regardless of its site (44).

24-Hour Holter recording and ECG transmission via the telephone

Although these techniques are often employed in patients with a history of unexplained syncope, they rarely document intermittent symptomatic third degree AV block. On the other hand, it is not uncommon to observe intermittent asymptomatic first or second degree AV block due to increased vagal tone e.g. during the night in patients with apparently intact AV conduction (45, 46). If this is the only time, that second degree AV block is documented, pacemaker therapy is certainly not recommended.

Exercise testing

Exercise testing may be a helpful diagnostic tool to assess the site of the AV block. When improvement of AV conduction occurs during exercise i.e. from second degree or third degree AV block to 1 : 1 AV conduction the block is most likely localized in the AV node, since AV nodal conduction improves due to sympathetic drive.

In contrast, when exercise increases the AV block i.e. from first or second degree AV block to third degree AV block, heart block is more likely localized in the His bundle or bundle branches due to an increase of atrial impulses reaching the abnormal His Purkinje System (43).

The potential reversibility of AV block was demonstrated in young athletes (47). In these healthy individuals AV block on the rest ECG is an expression of increased vagal tone. The effects of exercise on the reversibility of AV block has not been systematically studied in elderly patients with second or third degree AV block. This is to be expected, as the majority of these patients are physically limited at the time a diagnosis of the AV block is established. As a consequence, limited data are available on the reversibility of AV block and on the physical work capacity of these patients during exercise (50, 51).

Pharmacologic testing

As opposed to sinus node dysfunction, sympathomimetic, sympatholytic, vagotonic and vagolytic drugs have not been routinely employed in the clinical evaluation of patients with first, second or third degree AV block. The administration of an anti-cholinergic drug like atropine may offer a clue to the site of block. When AV conduction temporarily improves after the administration of atropine, AV block is localized in the AV node. On

the other hand, when atropine aggravates the AV conduction disturbance, block is localized below the AV node. In rare cases, like bradycardia dependant infranodal block the opposite is true. In these patients atropine may abolish AV block despite its infranodal localization (42, 43).

Invasive electrophysiologic study

The site of AV block can be demonstrated by electrophysiologic study. In addition, invasive study allows (43, 52) further elucidation of the underlying mechanism of the conduction disorder. Fig. 5 shows the different

Fig. 5. Localization of AV block.

Diagrammatic representation of the AV conducting system shows different varieties of AV block.

Abbreviations: SAN = Sino-atrial node, AVN = AV node, BH = bundle of His, LB = left bundle branch, RB = right bundle branch, BE = His bundle electrogram, L-2 = lead II of the standard ECG, P-A = intra-atrial conduction time, A-H = AV nodal conduction time, H-V = infra-nodal conduction time. (From Narula et al. Atrioventricular block localization and classification by His bundle recordings. *Am J Med* 50; 146, 1971. By permission of the publisher.)

varieties of AV block as a result of lesions in different segments of the AV conducting system.

- *First degree AV block*

In the majority of cases (> 80 percent) AV nodal delay causes PR prolongation (> 0.21 s) (52, 53). First degree AV block may be associated with a wide or narrow QRS complex. Patients with a wide QRS complex especially those with a left bundle branch block have a very high incidence (50-90 percent) of concomitant infranodal conduction delay (HV interval > 65 ms) (52).

- *Second degree AV block*

Second degree AV block may be localized in or below the AV node. Puech et al. (53) observed, that chronic Type I second degree AV block (Mobitz Type I, Wenckebach type) was localized in 72 percent of the cases in the AV node and in 28 percent in the His bundle or bundle branches. Those cases that meet the strict criteria of Type II second degree AV block (Mobitz Type II) were almost invariably localized in the His bundle or bundle branches.

Advanced second degree AV block (high grade AV block with conduction ratios 2 : 1, 3 : 1) may be Type I or Type II variants. Narula et al. (52) observed that the site of conduction block in patients with advanced second degree AV block was localized in the AV node in 33 percent, in the His bundle in 17 percent and in the bundle branches in the remaining 50 percent.

- *Third degree AV block*

In different series, the incidence of third degree AV block ranges from 16 to 25 percent in the AV node, 14 to 20 percent in the His bundle and 56 to 68 percent in the bundle branches (52, 53).

Another aspect of electrophysiologic investigation is the evaluation of retrograde AV conduction. With regard to presently available pulse generators, evaluation of retrograde conduction is of major clinical importance (37, 54-56). The clinical evaluation of retrograde AV conduction in patients with second or third degree AV block is elaborated upon in Chapter 5.

3. Management

For the present, all authorities would agree that symptomatic patients with second or third degree AV block should be offered permanent cardiac pacing. However, no consensus has been reached on whether symptomatic patients with first degree AV block or some subgroups of patients with documented infranodal conduction delay should be paced (43, 44, 47, 49, 57-61).

Generally, the following guidelines are suggested:

Pacemaker implantation is not indicated in asymptomatic patients with first, second or third degree AV block (49). In symptomatic patients with second or third degree AV block pacemaker implantation is indicated.

- When *first degree AV block* is present on the ECG of a patient with syncope episodes, electrophysiologic study is performed to determine the level of conduction delay. In addition, programmed electrical stimulation may be performed to rule out arrhythmias that might cause syncope. In patients with a prolonged AH interval (> 160 ms) but a normal HV interval (< 65 ms) either during baseline recording or during incremental atrial pacing at rates of at least 120 beats/min, no further action is usually required. The patient is followed in the outpatient clinic and depending on further symptoms 24-hour holter recordings are repeated. When a significantly prolonged HV time (> 65 ms) is documented during baseline recording or infranodal block occurs during incremental atrial pacing at rates less than 120 beats/min, pacemaker therapy is indicated (43, 58, 62) (Fig. 6). The value of stressing the His-Purkinje system by Vaughan Williams class I antiarrhythmic drugs as suggested by Fisher (61) is still unknown;
- When *second degree AV block* is present on the ECG of patients with symptoms of syncope or dizziness, irrespective of the site of block pacemaker therapy is indicated (52, 58) (Fig. 7);
- Symptomatic patients with *third degree heart block* are also candidates for permanent pacemaker insertion irrespective of the site of lesion (Fig. 7). In the setting of complete heart block in acute myocardial infarction, temporary pacing might be indicated when deterioration of haemodynamic performance is present.
Permanent pacing is rarely indicated in patients with inferior wall myo-

Fig. 6. Decision making tree in patients with first degree AV block.

AVB I represents with first degree AV block. S+ = patients with symptoms of syncope or dizziness and first degree AV block; S- = asymptomatic patients with first degree AV block; W/S = wait & see; EPS = Electrophysiologic Study; + / - infranodal conduction delay is present or absent; PM = pacemaker implantation.

cardial infarctions, since the conduction abnormality is usually of functional origin due to increased vagal tone and hence is reversible during evolution of the infarction.

The conduction abnormality in anterior wall myocardial infarction is usually not reversible and a permanent pacemaker may be implanted. Unfortunately, pacemaker therapy rarely improves the prognosis of these patients. The major cause of death is pump failure, whether or not a pacemaker is implanted (64).

Fig. 7. Decision making tree in patients with second or third degree AV block.
(For abbreviations see Fig. 4 and 6.)

- Symptomatic patients with *bifascicular block* (LBBB, or RBBB + LAHB/LPHB) and symptomatic patients with *trifascicular block* (bifascicular block + PR prolongation > 0.20 s) are candidates for electrophysiologic study, provided that other causes for syncope have been excluded. When during baseline recording a prolonged HV interval (> 65 ms) is found or second degree infranodal block occurs during incremental atrial pacing at pacing rates less than 120 beats/min, pacemaker implantation is indicated (57, 63). In addition programmed electrical stimulation is performed to document or to rule out other causes of recurrent syncope, like ventricular tachyarrhythmias.

In patients with *unexplained syncope* and a *normal ECG* electrophysiologic evaluation is indicated, provided that other causes for dizziness or syncope have been excluded.

In these patients electrophysiologic study may reveal underlying infranodal conduction disease (65), however such a finding is rather exceptional. Patients who are found to have infranodal conduction disease are candidates for permanent pacing. In addition, programmed electrical stimulation may be performed to rule out or document arrhythmias that might cause syncope.

A discussion on the value of programmed electrical stimulation with regard to the initiation of supraventricular or ventricular tachycardias in patients without clinical documentation of the arrhythmia is beyond the scope of the clinical evaluation of AV block, although it still is an important part of the evaluation of the patient with unexplained syncope.

PACEMAKER SELECTION

In this section, the most commonly used pacemakers for patients with sinus node dysfunction and patients with AV block will be discussed. In addition the concept of rate responsive pacing is introduced.

Fig. 8. Schematic representation of the ECG during sinus rhythm and during atrial and ventricular on demand pacing in the patient with sinus node dysfunction.

- Sinus rhythm with intact AV conduction.
- Ventricular pacing in a patient without retrograde AV conduction results in AV dissociation.
- Ventricular pacing in a patient with intact retrograde AV conduction results in atrial contraction against closed mitral and tricuspid valves.
- Atrial pacing in a patient with intact AV conduction results in preservation of AV synchrony.

Ventricular demand pacing

For more than 2 decades, this was the only available permanent pacing system. Over the years several reports appeared in the literature emphasizing the disadvantages of this pacing mode, based on haemodynamic studies performed in the catheterization laboratory (33, 34). Certainly, one of the major shortcomings of ventricular pacing is that appropriate AV synchrony is lost either as a result of AV dissociation or as a consequence of retrograde atrial conduction (Fig. 8). Another limitation especially in patients with sinus node dysfunction and intact anterograde AV conduction is that ventricular depolarization and ventricular contraction during ventricular pacing is different from and haemodynamically inferior to ventricular activation during normal AV conduction (39).

On the other hand, the majority of patients with symptomatic conduction disorders in whom a ventricular unit was implanted, did remarkably well, since ventricular demand pacing proved to be successful in alleviating cerebral symptoms (32, 47). At present, ventricular demand pacemakers are still the most commonly used pacemakers in patients with symptomatic conduction disease (66).

Atrial demand pacing

Atrial pacing may be used with sinus node dysfunction and intact anterograde AV conduction. In patients with AV block, atrial pacing is contraindicated. From a haemodynamic standpoint, this pacing mode is superior to on demand ventricular pacing (35). So far, permanent atrial pacing has never gained a great popularity. This is partly due to the fact that only recently reliable atrial leads have become available for clinical application, but it also relates to the restraint of many clinicians to implant a pacing system which is inadequate if heart block would occur during follow-up.

AV universal (DDD) pacing

A DDD pacemaker operates as an atrial demand unit as long as anterograde AV conduction is intact. However when the natural AV conduction time exceeds a programmed interval (AV interval) the ventricle is paced. Therefore, during DDD pacing AV synchrony is preserved (38-40). Fig. 9 schematically illustrates the differences between ventricular and AV synchronous pacing. At present, DDD pacers are implanted, especially in pa-

Fig. 9. Schematic representation of the ECG during normal sinus rhythm, during ventricular and AV synchronous pacing.

Upper tracing: sinus rhythm with intact AV conduction.

Middle tracing: ventricular pacing by a single chamber pacemaker.

Lower tracing: AV synchronous pacing by a dual chamber pacemaker.

tients who were physically active before the onset of AV block, irrespective of their age (70, Chapter 5).

Initially, AV synchrony was thought to play a more important role in maintaining cardiac output in the failing heart than in the normal heart (66). Recently, Wirtzfeld et al. (70) demonstrated that atrial contribution to stroke volume is relatively greater in patients with pulmonary capillary wedge pressures less than 20 mm Hg than in patients with wedge pressures greater than 20 mm Hg. Based on these results these investigators suggested, that it is more likely that the opposite is true.

The benefits of DDD pacing are: AV synchrony is preserved at rest in contrast to ventricular demand pacing; during exercise not only AV synchrony is maintained, but in addition the sinus node of the patient determines the AV synchronous pacing rate. In other words as long as the sinus node normally accelerates with exercise or during conditions of increased metabolic demand, a physiologic rate response occurs (40, 41, 70). Critical to a pacing system that uses the native P wave as a trigger for ventricular pacing is the proper and consistent recognition and discrimination of the P wave

from both other intracardiac signals and extracardiac sources of interference (71).

A poor rate increase is provided by the pacemaker in patients with either sinus node dysfunction or patients treated with drugs that may suppress sinus node function.

Rate responsive pacing

One can define rate responsive pacing as pacing to optimize cardiac output to meet metabolic needs in the absence of normal atrial control (72). Control is achieved by sensing some physiologic variable that is correlated to actual physiologic need. The pacing rate is increased or decreased, dependant on the sensed parameter value. Several sensors have now been developed to adjust the atrial or ventricular pacing rate to metabolic demand (41). The ideal form of pacing is one that responds to the body's need for varying cardiac output in a way a normal sinus node would respond while maintaining AV synchrony (70). Table II lists the physiologic sensors available for rate responsive pacing, which are presently available in singel chamber pacing systems.

TABLE II*
**PHYSIOLOGIC SENSORS PROPOSED FOR RATE
RESPONSIVE PACING**

PARAMETER	YEAR**
RESPIRATION RATE	1975
BLOOD pH	1977
CENTRAL BODY TEMPERATURE	1978
QT INTERVAL	1981
O2 CONTENT OF VENOUS MIXED BLOOD	1981
ASYNCHRONOUS ATRIAL RATE	1982
MUSCLE NOISE	1983
RV STROKE VOLUME	1984
RIGHT ATRIAL PRESSURE	1984

* From A. Wirzfeld et al. (70).

** First publication in the medical literature.

- Rate responsive atrial pacing

As far as sinus nodal function is concerned, neither ventricular and atrial on demand pacing nor AV synchronous pacing correct for a blunted sinus

node response to exercise, emotion or metabolic demand. A rate responsive *atrial* pacing system would be a substitute for a failing sinus node in the subgroup of patients with sinus node dysfunction and intact AV conduction (67, 68).

- Rate responsive ventricular pacing

When patients are allowed to increase heart rate and maintain AV synchrony by use of AV synchronous pacing, a statistically significant increase in exercise capacity has been demonstrated in comparison with fixed rate ventricular pacing (50, 51).

Is this increase in exercise capacity a result of the chronotropic response of the ventricle alone or a result of the maintenance of AV synchrony during the chronotropic response of the ventricle?

Fananapazir et al. (73) studied this subject and observed that the chronotropic response of the ventricle is the primary determinant in enhancing exercise capacity. These investigators demonstrated that a similar heart rate response with or without atrial synchrony appeared to allow a similar enhancement of exercise capacity over ventricular demand pacing. Rate responsive *ventricular* pacing therefore has, similar to DDD pacing, the potential for improved exercise capability in comparison with ventricular demand pacing at a constant rate.

- Rate responsive AV synchronous pacing

As mentioned before, a limitation during AV synchronous pacing is that an inadequate rate increase will occur during exercise in patients with sinus node dysfunction or patients treated with drugs that may suppress sinus node function. Rate responsive *AV synchronous* pacing might substitute for this default with preservation of AV synchrony. With the development of appropriate sensors, rate responsive pacing may open a completely new field of investigation for the treatment of cardiac conduction disorders (67, 68, 70).

Two decades ago syncope due to complete heart block was the primary indication for permanent cardiac pacing. At present, pacemakers are implanted not only to prevent recurrent syncope but also with the objective to improve the patient's working capacity and feeling of well-being (70).

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Atrioventricular and Ventriculo-Atrial Conduction in Patients with Symptomatic Sinus Node Dysfunction

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VAN MECHELEN, R., ET AL.: Atrioventricular and ventriculo-atrial conduction in patients with symptomatic sinus node dysfunction. In 14 patients with symptomatic sinus node dysfunction—sinus bradycardia, sino-atrial exit block, or sinus arrest—electrophysiological studies were performed before implantation of a pacemaker. In 8 patients incremental high right atrial pacing showed AV-nodal Wenckebach at pacing rates equal to or above 130/min (group I); in 6 patients AV-nodal Wenckebach was reached at pacing rates less than 130/min (group II). During ventricular pacing at a rate 10-15% faster than the existing sinus rate, ventriculo-atrial (VA) conduction was present in all patients of group I, while VA conduction was present in only 2 patients of group II ($p < 0.05$). Patients with symptomatic sinus node dysfunction but with intact AV conduction frequently show VA conduction during ventricular pacing and thus are particularly at risk for developing a pacemaker syndrome when a ventricular demand (VVI) pacemaker is implanted. This complication can be avoided by atrial demand (AAI) pacing or AV sequential (DVI) pacing. When adequate experience has been gathered with AV universal (DDD) pacemakers, the indications for selection of a pacemaker in patients with symptomatic sinus node dysfunction will probably change. (PACE, Vol. 6, January-February, 1983)

pacemaker syndrome, permanent pacing, sick sinus syndrome, ventriculo-atrial conduction

Ventriculo-atrial (VA) conduction may occur under a variety of circumstances. VA conduction has occasionally been demonstrated in patients with ventricular premature beats,¹ spontaneous or catheter-induced ventricular tachycardia,²⁻⁴ complete AV block during ventricular pacing,⁵⁻⁷ normal AV conduction, and in a subset of patients with sinus node dysfunction undergoing ventricular demand pacing.⁸

The surface ECG is often helpful in identifying retrograde P waves during ventricular pacing, but definite proof of VA conduction is only possible by electrophysiological investigation. However, electrophysiological studies are not employed routinely in patients with sinus node dysfunction and, therefore, the true incidence of VA conduction during ventricular pacing in these patients has not been established.

Patients with sinus node dysfunction exhibiting VA conduction in response to ventricular pacing may be particularly sensitive to the loss of atrioventricular synchrony and may be inappropriate candidates for ventricular demand (VVI) pacing.⁹⁻¹⁴ We performed electrophysiological investigations in these patients to study VA conduction during ventricular pacing. In addition, sino-atrial, AV-nodal, and infranodal conduction were evaluated in order to select the most appropriate mode for permanent pacing.

Patients were divided into two groups, one with normal AV conduction (group I), and the other with impaired AV conduction (group II), in order to study the relationship between AV conduction and the capacity for 1:1 VA conduction.

Methods

The decision to implant a pacemaker in patients with symptomatic sinus node dysfunction was based upon the following conditions:

1. symptoms of dizziness, dyspnea, and/or syn-

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cope in relation with sinus bradycardia, sinus arrest, or sino-atrial block documented by ambulatory electrocardiography or by telemetry;

2. absence of drug therapy (digitalis and beta-blockade), that might impair sino-atrial function.

Electrophysiological studies

Patients who met these criteria underwent an electrophysiological investigation after having given informed consent, in order to study VA conduction and to evaluate sino-atrial, AV nodal, and infranodal conduction.

Three catheters were introduced by puncture of the femoral vein. One quadripolar catheter was used to stimulate the atria and record a bipolar high right atrial (HRA) electrogram; one bipolar catheter was positioned across the tricuspid valve to record the His bundle electrogram (HBE); and one bipolar catheter was used to stimulate the ventricles. If no adequate His bundle electrogram could be recorded within 10 minutes after introduction of the catheters, AV and VA conduction were studied using the atrial and ventricular catheters only. All recordings were made on a Meddars 300 catheterization laboratory computer (Honeywell*). An external pulse generator (Janssen SU2) was used for atrial and ventricular stimulation. Three ECG leads, a high right atrial and a His bundle electrogram were recorded simultaneously.

The AH interval and HV interval were measured during sinus rhythm. Ventricular pacing was then initiated at a rate 10-15% faster than the existing sinus rate. The effective ventricular refractory period (VERP) was determined with premature ventricular beats induced with regularly shortening degrees of prematurity. Criteria for the acceptance of 1:1 VA conduction during pacing were the following: [1] identical ventricular and atrial cycle length at the ventricular pacing rate; [2] constant VA conduction times at one and the same pacing rate; [3] a pause prior to the resumption of sinus rhythm

when ventricular pacing was abruptly interrupted (Figs. 1a, b).

Incremental ventricular pacing was also performed up to a pacing rate of 160/min or up to a rate at which second degree VA block occurred.

The high right atrium was paced at an initial pacing rate slightly above the patient's sinus or AV junctional rate.

The atrial pacing rate was increased in steps of 10/min until 1:1 AV conduction was lost and second degree AV block occurred. A normal response to atrial pacing at incremental rates was defined as the development of Wenckebach periods at a heart rate of 130/min or higher.¹⁰⁻¹¹ The development of Wenckebach block below a heart rate of 130/min suggested impaired AV conduction. An estimate of cardiac automaticity was obtained by pacing the atrium at approximately 120/min for a minimum of 30 seconds. Upon cessation of pacing, the interval from the last pacing impulse to the first sinus node recovery beat was measured (SNRT). The corrected sinus node recovery time (CSNRT) was calculated by subtracting the basic sinus rhythm cycle length from the sinus node recovery time. If asystole persisted for 5 seconds without escape rhythm, atrial pacing was resumed.

Automatic tone was assessed by carotid sinus massage (CSM) and the intravenous administration of atropine, starting with 0.5 mg up to a maximum dose of 2 mg. A normal response to atropine was defined as an increase of sinus rate to greater than 90/min, and an increase over the spontaneous rate of 20-50%.

These data were used to sub-divide our patient population into two categories: Group I: patients with normal AV conduction with AV nodal Wenckebach occurring at a HRA pacing rate of at least 130/min; Group II: patients with impaired AV conduction with AV nodal Wenckebach occurring at a HRA pacing rate slower than 130/min.

Policy for pacemaker implantation and follow up

In patients with normal AV conduction, an atrial demand pacemaker (AAI) was implanted. In patients with impaired AV conduction a ven-

*Honeywell, Denver, Colorado, U.S.A.

Figure 1a. Criteria for 1:1 VA conduction at ventricular pacing. The ventricles are driven at a rate of 70/min (CL, 857 ms). Atrial and ventricular cycle lengths are equal; VA conduction time is constant (160 ms). The His bundle electrogram (HBE) shows retrograde His bundle depolarizations during ventricular pacing. The VA interval is measured from the ventricular spike to the onset of atrial depolarization in the HRA lead.

tricular demand pacemaker (VVI) was implanted when VA conduction was absent during ventricular pacing at rates 60-100/min. In patients with impaired AV conduction but intact VA conduction during ventricular pacing, an AV sequential (DVI) pacemaker was implanted. Patients were discharged from the hospital 7 days after pacemaker implantation. Pacemaker follow-up included analysis of impulse duration, impulse frequency, impulse amplitude, and impulse form. Patients came to the outpatient clinic the tenth day, one month, three months, and six months after discharge from the hospital, and thereafter every six months.

Results

Patient characteristics

During a two-year period from September 1979 through September 1981, 14 patients were admitted to our department because of symptomatic sinus node dysfunction. The clinical and electrocardiographic data are listed in Table I.

Sino-atrial conduction

Table II shows the results of electrophysiological studies. The response to CSM was nor-

Figure 1b. Criteria for 1:1 VA conduction at the termination of ventricular pacing. After the second paced beat, electrical stimuli are abruptly terminated. A 1.7 s pause results before the first atrial depolarization of AV junctional origin occurs. The first atrial depolarization of sinus origin in this patient appeared after a pause of 5.5 s.

mal in all patients. The response to atropine was blunted in 10 patients (71%). Sinus node recovery time (SNRT) and corrected sinus node recovery time (CNSRT) are also listed in Table II.

AV-nodal conduction

The AH interval during sinus rhythm was normal in 7 patients and prolonged in 4 patients. The HV interval during sinus rhythm was normal in 11 patients. In 3 patients His potentials could not be recorded. Second degree AV block during HRA pacing was always proximal to the His bundle in patients in whom an adequate His bundle depolarization could be recorded. In 8 patients AV Wenckebach developed during pacing from the HRA at rates ranging from 130/min to 160/min (Group I). Six patients developed AV nodal Wenckebach during HRA pacing at rates below 130/min, ranging from 50/min to 120/min. Of these 6 patients there were 4 who had prolonged AH intervals (≥ 140 ms) during sinus

rhythm, or at atrial pacing slightly above the spontaneous rate.

Infranodal conduction

There were 3 patients with intraventricular conduction defects, 1 patient with right bundle branch block and left axis deviation, and 2 patients with left anterior hemiblock. The HV interval was normal (< 60 ms) in three patients.

VA conduction

VA conduction was present in 10 patients and absent in 4 patients (Table III). VA intervals measured from the stimulus artifact to the onset of atrial depolarization in the HRA lead ranged from 120-230 ms at ventricular pacing rates 10-15% faster than the sinus rate. VA Wenckebach was present in all 10 patients: in 7 at ventricular pacing rates equal to or greater than 100/min (ranging from 100-150/min) and in 3 patients at ventricular rates slower than 100/min (90/min).

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Table I.
Clinical and Electrocardiographic Data

Patient	Age (yrs)	Sex	Arrhythmia prior to pacing	Electrocardiogram					
				Spontaneous Sinus pause (sec)	HR (bpm)	PR (sec)	QRS (sec)	Other	Symptoms
1. J.B.	73	f	AVJ, SB	3.5	50-120	0.16	0.06	—	syncope
2. L.B.	67	m	AVJ, SB	—	30-100	0.18	0.08	—	dizziness
3. D.L.	75	f	SB, PSVT	—	35-140	0.18	0.06	—	dizziness
4. A.V.	40	f	SAB, PVST	4.2	70-140	0.16	0.06	—	dyspnea, dizziness
5. A.L.	78	m	PSVT, SB, AVJ	—	40-150	0.14	0.08	—	dizziness
6. J.C.	81	m	SB, PAF, SA	5.5	50-120	0.16	0.06	—	syncope
7. A.E.	85	f	SB, AVJ	—	37-140	0.18	0.08	—	dizziness
8. M.R.	83	f	SB, AVJ	—	40- 60	0.22	0.14	RBBB, LAHB	dizziness
9. E.S.M.	77	f	SB, AVJ	—	40-100	0.18	0.10	LAHB	dizziness
10. J.S.K.	73	f	SB, AVJ	—	28- 60	0.16	0.06	—	syncope, dizziness
11. G.D.	81	f	SB, AVBII	—	40- 80	0.20	0.06	—	dizziness
12. D.P.	83	f	SB, AVBII	—	40- 80	0.20	0.10	LAHB	dyspnea, dizziness
13. G.E.	71	f	SA, SB	4.5	40- 60	0.16	0.06	—	dizziness, syncope
14. A.J.	67	m	PAF, AVJ	—	16-100	0.18	0.08	—	dizziness

Abbreviations: AVJ = AV Junctional rhythm; SB = sinus bradycardia; PSVT = paroxysmal supraventricular tachycardia; SAB = sino-atrial block; PAF = paroxysmal atrial fibrillation; AVBII = second degree AV block; RBBB = right bundle branch block; LAHB = left anterior hemiblock.

VA conduction was present in all patients of group I, but in only 2 patients of group II ($p < 0.05$) (Table IV).

Pacemaker therapy and follow-up

All patients were discharged from the hospital within 7 days after pacemaker implantation. Eight patients had an AAI unit, 4 patients had a VVI unit, and 2 patients had a DVI pacemaker. All patients could be followed for at least 3 months. The follow-up data are listed in Table V. None of the patients with an AAI pacemaker developed impaired AV conduction. There were no pacemaker-related complications during the observed follow-up period; in particular, there were no symptoms suggestive of pacemaker syndrome. All patients are alive.

Discussion

Ventriculo-atrial conduction was studied in patients with tachyarrhythmias by Goldreyer et al.¹⁷ in 1970. These investigators could demonstrate an 89% incidence of VA conduction in patients with normal AV conduction, compared with an 8% incidence of VA conduction in patients with impaired AV conduction.

Schuilenburg¹⁸ could demonstrate a 68% incidence of VA conduction in patients with normal AV conduction and a 44% incidence of VA conduction in patients with impaired AV conduction.

In comparison with the vast literature on the subject of AV conduction accumulated in recent years, the number of studies in which antegrade and retrograde AV conduction were compared systematically is relatively small.¹⁷⁻²¹

Table II.
Results of Electrophysiological Studies

		Atrial Overdrive Response					Response to atropine		
Patient	HR and Rhythm during study (bpm)	AH (ms)	HV (ms)	AVN-W (bpm)	SNRT (ms)	CSNRT (ms)	HR (bpm)	Increase in rate (%)	
1. J.B.	SR	50	100	50	160	2000	350	70	10
2. L.B.	SR	70	110	55	140	—	—	87	15
3. D.L.	SR	50	80	50	130	2000	600	50	10
4. A.V.	SR	42	—	—	130	—	—	70	16
5. A.L.	AVJ	50	150	50	110	3000	1100	100	50
6. J.C.	SR	80	—	—	140	4000	—	130	60
7. A.E.	AVJ	45	—	—	70	4500	500	45	0
8. M.R.	SR	40	180	60	100	1100	500	70	12
9. E.S.M.	SR	60	100	50	130	1400	450	—	—
10. J.S.K.	AVJ	40	130	50	130	1800	350	40	0
11. G.D.	SR, AVB _{II}	40	140	50	60	1600	250	60	10
12. D.P.	SR, AVB _{II}	30	180	55	50	5800	350	80	12
13. G.E.	SR	60	110	45	120	6400	560	100	50
14. A.J.	AVJ	45	90	50	150	5500	250	40	0

Abbreviations: SR = sinus rhythm; AVJ = AV junctional rhythm; AVB_{II} = second degree AV nodal block; AVN-W = AV nodal Wenckebach block; SNRT = sinus node recovery time; CSNRT = corrected sinus node recovery time.

Wirtzfeld⁹ observed in 50 patients with sinus node dysfunction a 40% incidence of VA conduction during permanent ventricular pacing. However, antegrade and retrograde AV conduction were not compared systematically. Morley et al.²¹ observed an 89% incidence of VA conduction in 42 patients with sick sinus and carotid sinus syndrome and normal antegrade AV conduction.

In 14 patients with symptomatic sinus node dysfunction, we observed a 100% incidence of VA conduction in patients with intact AV conduction and a 33% incidence of VA conduction in patients with impaired AV conduction ($p < 0.05$). For patients who require permanent pacing these electrophysiological findings may have important practical implications. During ventricular pacing VA conduction will result in contraction of the atria against closed mitral and tricuspid valves. As a consequence systemic and pulmonary vein pressures will increase and arterial blood pressure will fall. The symptoms

of this so called pacemaker syndrome are, in some patients, so disabling that their ventricular demand pacemaker has to be explanted.¹²⁻¹⁴ We, therefore, believe that evaluation of retrograde conduction is mandatory, when one considers the implantation of a ventricular demand pacemaker in these patients.

In patients with normal AV conduction, but with intact VA conduction, atrial demand pacing will prevent a pacemaker syndrome. This pacing mode has been recommended^{9,13,14} and we have used atrial demand (AAI) pacemakers successfully in 8 of our patients. If, however, AV conduction deteriorates at a later stage, a new lead placement and a new pacemaker implantation might be required. AV sequential (DVI) pacing will also avoid the risk of a pacemaker syndrome and may anticipate the deterioration of AV conduction at a later stage. Unfortunately, many of the available units have an AV delay, which is too short to respect normal AV conduction, and artificial ventricular pacing occurs in

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Table III.
Results of Electrophysiological Studies

GROUP I				
patients with normal AV conduction	VERP (ms)	VAC	VAW (bpm)	VACT (ms)
1. J.B.	280	+	110	150
2. L.B.	210	+	120	200
3. D.L.	240	+	100	160
4. A.V.	280	+	150	180
6. J.C.	300	+	140	130
9. E.S.M.	310	+	90	160
10. J.S.K.	260	+	110	230
14. A.J.	220	+	90	120
GROUP II				
patients with impaired AV conduction				
5. A.L.	260	+	120	150
7. A.E.	240	—	—	—
8. M.R.	280	—	—	—
11. G.D.	240	—	—	—
12. D.P.	320	—	—	—
13. G.E.	300	+	90	150

Abbreviations: VERP = effective refractory period at ventricular pacing rates ranging from 60/min to 90/min.; VAC = VA conduction; VAW = VA Wenckebach pacing rate; VACT = VA conduction time, VA interval was measured from the stimulus artifact to the onset of AHRA.

Table IV.
VA conduction in patients with normal and impaired AV conduction

	VA conduction present	VA conduction absent
GROUP I patients with normal AV conduction	8	0
GROUP II patients with impaired AV conduction	2	4
Total	10	4
		(p < 0.05)*

*two-sided Fisher exact test.(22)

spite of normal AV conduction. Another problem with these units is that the initial slew rate of the sensed ventricular depolarization may be too slow, and the result is a fusion beat or even artificial pacing very late in a spontaneous QRS complex in spite of normal ventricular sensing. For this reason programmability of the AV delay with this form of pacing will allow greater flexibility.

In patients with impaired AV conduction, atrial demand (AAI) pacing is contraindicated. In 2 of our patients with impaired AV conduction but with intact VA conduction, retrograde conduction was precluded by pacing the atria and ventricles sequentially. In these patients an AV sequential (DVI) pacemaker was implanted.

Initially, when we started our study, the atrial synchronous ventricular inhibited (VDD) pace-

Table V.
Pacemaker therapy and follow-up

Patient	AV conduction		VA conduction		PM	F-U (months)
	N.	IMP.	P.	A.		
1. J.B.	+		+		AAI	3
2. L.B.	+		+		AAI	4
3. D.L.	+		+		AAI	8
4. A.V.	+		+		AAI	13
5. A.L.		+	+		DVI	16
6. J.C.	+		+		AAI	7
7. A.E.		+		+	VVI	3
8. M.R.		+		+	VVI	18
9. E.S.M.	+		+		AAI	14
10. J.S.K.	+		+		AAI	6
11. G.D.		+		+	VVI	7
12. D.P.		+		+	VVI	10
13. G.E.		+	+		DVI	9
14. A.J.	+		+		AAI	24

Abbreviations: N = normal; Imp. = impaired; P = present; A = absent; PM = pacemaker type; F-U = follow-up.

maker and the AV universal (DDD) pacemaker were not available. However, these units are available at present. The VDD unit is not indicated in patients with sinus node dysfunction, because normal sino-atrial function is a prerequisite for its application. The DDD unit might be used in patients with symptomatic sinus node dysfunction, but when VA conduction is intact, this unit should be programmed in a DVI mode. The reason to choose the DVI mode of pacing rather than the DDD pacing mode is that in the latter VA conduction might induce a re-entry tachycardia, in which the retrograde arm of the circuit is provided by the VA conduction pathway and the antegrade arm by the atrial synchronous ventricular pacing pathway.²¹ By programming the unit in a DVI mode,

VA conduction will not result in an atrial synchronous ventricular response and thereby not initiate a re-entry tachycardia.

In conclusion, patients with symptomatic sinus node dysfunction, in particular patients with normal AV conduction, will frequently evidence VA conduction during ventricular pacing. These patients may develop a pacemaker syndrome after implantation of a ventricular demand (VVI) pacemaker. This complication can be avoided by atrial demand (AAI) pacing or AV sequential (DVI) pacing. When adequate experience has been gained with AV universal (DDD) pacemakers, the indications for selection of a pacemaker in patients with symptomatic sinus node dysfunction will probably change.

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Serial electrophysiologic studies after single chamber atrial pacemaker implantation in patients with symptomatic sinus node dysfunction

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KEY WORDS: Anterograde AV conduction, antiarrhythmic drugs, atrial pacing, symptomatic sinus node dysfunction.

After single chamber atrial pacemaker implantation, serial electrophysiologic studies were performed noninvasively at intervals of 3 months over a total period of 3 years in 24 patients with symptomatic sinus node dysfunction. All patients underwent invasive electrophysiologic studies before pacemaker implantation and demonstrated intact anterograde AV conduction. Patients were divided into 2 groups: group 1 did not require antiarrhythmic drugs during follow-up whereas group 2 received antiarrhythmic drugs.

In group 1 (11 patients) the atrial paced heart rate producing AV Wenckebach phenomenon (AVWHR) remained stable during a mean follow-up of 22 ± 10 months; with a variability not exceeding 10 beats min^{-1} with respect to the initial AVWHR obtained during preoperative electrophysiologic study. In group 2 (13 patients) with a mean follow-up of 15 ± 8 months a mean decrease of AVWHR of 19.2 ± 17.5 beats min^{-1} was present between AVWHR before and 3 months after initiation of oral antiarrhythmic drugs ($P < 0.01$). During chronic (> 3 months) antiarrhythmic drug therapy the variability of the AVWHR never exceeded 10 beats min^{-1} with respect to the AVWHR obtained 3 months after the initiation of oral drug therapy.

Deterioration of anterograde AV conduction during long-term follow-up of patients with symptomatic sinus node dysfunction and intact anterograde AV conduction at the time of pacemaker implantation is a consequence of orally taken antiarrhythmic drugs, rather than a consequence of degeneration of the AV conducting system.

Introduction

Single-chamber atrial pacing in patients with symptomatic sinus node dysfunction and intact anterograde AV conduction at the time of pacemaker implantation is still a controversial issue. Ferrer^[1], Rosen *et al.*^[2] and Narula^[3] have suggested that sinus node dysfunction is a part of a diffuse disease of the AV conducting system. They assumed that, although anterograde AV conduction may be intact at the time of pacemaker implantation, deterioration during follow-up is likely, as a result of degeneration of the AV conducting system. However, since antiarrhythmic drugs are frequently used in these patients to treat coexisting tachyarrhythmias or other medical problems^[4], deterioration of

anterograde AV conduction may also be a result of the electrophysiologic effects of these agents on the AV conducting system.

The aim of our study, was to determine the relative importance of these two factors on deterioration of anterograde AV conduction during follow-up of patients with symptomatic sinus node dysfunction and intact anterograde AV conduction at the time of single-chamber atrial pacemaker implantation.

Methods

PATIENT SELECTION

Patients with symptomatic sinus node dysfunction and intact anterograde AV conduction were selected for this study. The diagnosis of symptomatic sinus node dysfunction was based upon symptoms of dizziness, dyspnoea or syncope with: (a) sinus bradycardia (< 50 beats min^{-1}), sinus arrest (≥ 4 s) and/or sino-atrial block (≥ 4 s), documented by Holter tracing and/or telemetry;

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Table 1 Clinical and electrocardiographic data

Patients	Age (yrs)	Sex (m/f)	CD	ECG			Other	Symptoms
				HR (bpm)	PR (s)	QRS (s)		
1	84	f	SB	40-50	0.18	0.08	-	dizziness
2	75	f	SA, SB	50-120	0.16	0.08	-	syncope
3	70	m	SB	30-100	0.18	0.08	-	syncope
4	77	f	SB	42-70	0.18	0.08	-	dizziness
5	94	f	SB	40-70	0.24	0.08	-	dizziness
6	73	f	SB	40-60	0.16	0.08	-	dizziness
7	84	f	SB, AVJR	30-50	0.20	0.08	-	dizziness
8	76	f	SA, SB	20-60	0.18	0.08	-	syncope
9	61	m	SB	35-50	0.20	0.08	-	syncope
10	82	f	SB, AVJR	40-50	0.18	0.08	-	dizziness
11	76	m	SA	10-90	0.18	0.08	-	syncope
12	75	f	SB, AVJR	28-60	0.16	0.08	LVH	syncope
13	71	m	SB	40-60	0.14	0.08	-	dizziness
14	78	f	SB, AVJR	40-70	0.18	0.08	-	dizziness
15	75	f	SB, PAF, VT	30-140	0.18	0.08	-	syncope
16	76	f	SAB, SB	45-70	0.20	0.08	-	dizziness
17	85	f	SA	30-140	0.18	0.08	-	syncope
18	65	f	SB, PAFI	40-150	0.16	0.08	-	dizziness
19	83	m	SA	30-90	0.20	0.10	AMI	dizziness
20	78	f	SA	15-75	0.16	0.08	AMI	syncope
21	85	m	SB	30-70	0.20	0.08	-	syncope
22	80	f	SB	30-65	0.18	0.08	-	dizziness
23	73	m	SB	30-90	0.20	0.12	RBBB, AMI	syncope
24	55	f	SB, PAF	40-180	0.14	0.08	-	dizziness

CD = conduction disorder; SA = sinus arrest; SB = sinus bradycardia. SAB = sino-atrial block; AVJR = AV junctional rhythm; PAF = paroxysmal atrial fibrillation; PAFI = paroxysmal atrial flutter; VT = ventricular tachycardia; LVH = left ventricular hypertrophy; AMI = anterior myocardial infarction; RBBB = right bundle branch block.

(b) the absence of oral drug therapy that might impair sino-atrial function¹⁸⁻²¹.

The clinical and electrocardiographic data are listed in Table 1. The presence of intact antero-grade AV conduction was assessed during electrophysiologic study prior to pacemaker implantation.

ELECTROPHYSIOLOGIC STUDIES

After informed consent was obtained, patients underwent an electrophysiologic study according to previously described catheter techniques^{23,8}. Cardio-active drugs were withheld for at least 48 h prior to the study.

Intact antero-grade AV conduction was defined as:

1. normal HV interval (≤ 60 ms) during baseline recording;
2. persistence of 1:1 AV conduction during

incremental high right atrial pacing at rates equal to or greater than 120 beats min^{-1} (Table 2).

In all patients with intact antero-grade AV conduction a single chamber atrial pacemaker was implanted (Medtronic 5985, 5977 or 8423). This particular pulse generator was selected because the programmability of the pacing system allowed us to perform limited electrophysiologic studies non-invasively during follow-up. With this pacemaker the atrial paced rate producing AV Wenckebach phenomenon was determined at intervals of 3 months after hospital discharge. The latter was assessed by increasing the atrial pacing rate in steps of 10 beats min^{-1} , until 1:1 AV conduction was lost and second-degree AV block occurred. Each pacing period lasted 1 min. Studies were recorded on a ECG machine with a paper speed of 50 mm s^{-1} (Fig. 1).

Table 2 Electrophysiologic studies prior to pacemaker implantation

Patients	Baseline			SNRT (ms)	AVWHR (bpm)
	Rhythm	HR (bpm)	AH (ms)	HV (ms)	
1	SR	42	—	—	130
2	SR	50	100	50	160
3	SR	70	110	50	160
4	AVJR	50	—	50	140
5	—	—	—	—	≥140
6	SR	45	80	50	150
7	SR	50	—	—	170
8	AVJR	30	—	40	120
9	SR	55	130	45	130
10	AVJR	50	—	—	150
11	SR	60	80	50	140
12	AVJR	40	—	50	130
13	SR	50	70	40	170
14	AVJR	50	—	50	190
15	SR	70	90	50	≥160
16	SR	50	100	50	120
17	SR	70	90	60	140
18	SR	70	80	40	180
19	SR	90	—	—	190
20	SR	60	130	50	130
21	SR	50	90	50	120
22	SR	70	—	—	140
23	SR	70	—	—	120
24	SR	65	60	40	180

HR=heart rate; AH=AH interval; HV=HV interval. SNRT=sinus node recovery time; AVWHR=atrial pacing rate producing AV Wenckebach phenomenon; SR=sinus rhythm; AVJR=AV junctional rhythm.

PATIENT FOLLOW-UP

After initial study and pacemaker implantation, patients were prospectively followed in the out-patient clinic at intervals of 3 months. The first patient entered the study in September 1980 and the last patient entered the study in April 1983. Every pacemaker check-up visit included the history, physical examination, 12-lead ECG, and a noninvasive determination of the atrial paced heart rate producing AV Wenckebach phenomenon (AVWHR). One 24-h Holter recording was performed routinely during the first 6 weeks after hospital discharge and thereafter when history suggested transient brady- or tachy-arrhythmias.

When follow-up was completed in September 1983, patients were divided into 2 groups; group 1 patients had not received antiarrhythmic drugs during follow-up and group 2 patients were taking antiarrhythmic drugs. Group 2 consisted of patients

who were taking antiarrhythmic drugs in hospital, and patients in whom antiarrhythmic drugs were prescribed during follow-up.

STATISTICAL ANALYSIS

Each patient served as his or her own control. Differences between values were compared by means of the two-tailed paired Student's *t*-test. All data are presented as means $\pm$ s.d.

Results

There were 11 patients (group 1) who did not require antiarrhythmic drug therapy after pacemaker implantation during a mean follow-up of 22 ± 10 months. The mean age of group 1 patients was 77 ± 9 years. Previous to pacemaker implantation the electrocardiogram showed a normal PR-interval (<0.20 s) in 8 patients (73%). In all

Figure 1. The atrial paced heart rate producing AV Wenckebach phenomenon is determined during incremental atrial pacing (AP).

patients of group 1 the QRS complex was narrow (≤ 0.10 s). The permanent atrial pacing rates ranged from 70 to 80 beats min^{-1} . The mean atrial pacing rate producing AV Wenckebach phenomenon (AVWHR) during preoperative electrophysiologic studies was 144 ± 17 beats min^{-1} . Table 3 shows the results of serial electrophysiologic testing at intervals of 3 months after hospital discharge. The variability of AVWHR was equal to or less than 10 beats min^{-1} , with respect to the initial AVWHR obtained during preoperative electrophysiologic study. One patient (no. 1) demonstrated transiently an increase of the AVWHR of more than 10 beats min^{-1} 24 months after pacemaker implantation.

Group 2 consisted of 13 patients, in whom a variety of antiarrhythmic drugs were prescribed after pacemaker implantation, to treat cardiac failure or coexisting tachyarrhythmias. Table 4 shows the specific agents that were taken orally and the date these drugs were prescribed during follow-up of each patient.

The mean follow-up of group 2 was 15 ± 8 months. The mean age was 75 ± 8 years. Prior to

pacemaker implantation the electrocardiogram showed a normal PR interval in 9 patients (69%). In 12 patients (92%) the QRS complex was narrow. In one patient the QRS duration was 0.12 sec. Permanent atrial pacing rates ranged from 70 beats min^{-1} to 80 beats min^{-1} . The mean AVWHR during preoperative electrophysiologic studies was 151 ± 29 beats min^{-1} . Table 5 shows the results of serial electrophysiologic testing during follow-up observations in the outpatient clinic. A mean decrease of the AVWHR of 19.2 ± 17.5 beats min^{-1} occurred between the AVWHR before and 3 months after initiation of oral antiarrhythmic therapy ($P < 0.01$).

In patient no. 20 no fall of the AVWHR was observed after 3 months of oral quinidine sulfate (200 mg t.i.d.). In all other patients of group 2 either receiving digoxin, beta-blocking agents, verapamil or amiodarone, a drop of the AVWHR was noted after 3 months of oral drug therapy. Patient no. 16 complained of dizziness and fatigue during the first 3 months of amiodarone therapy (600 mg daily), with a concomitant drop of the AVWHR from 120 beats min^{-1} to 80 beats min^{-1} . The ECG showed

Table 3 AVWHR of patients without antiarrhythmic drugs during follow-up

Patients	Follow-up													
	EPS	3 mths	6 mths	9 mths	12 mths	15 mths	18 mths	21 mths	24 mths	27 mths	30 mths	33 mths	36 mths	
1	130	140	130	—	130	130	130	130	150	130	130	140	130	
2	160	150	150	150	160	160	160	150	160	150	160	150		
3	160	—	140	160	160	160	160	160	150	160	150			
4	140	160	160	150	160	170	140	160	150					
5	≥140	160	160	160	160	150	160	150						
6	150	140	140	140	150	150	140							
7	170	—	—	170	—	160	180							
8	120	120	120	120	130	120	120							
9	120	110	110	110	120									
10	150	140	140	140	140									
11	140	140	150											

AVWHR = the atrial rate producing AV Wenckebach phenomenon during incremental atrial pacing; EPS = electrophysiologic study before pacemaker implantation.

Table 4 Antiarrhythmic drugs during follow-up

Patients	Initiation (mths of FU)	Indication	Drugs (mg/day ⁻¹)
12	6	PAT	digoxin 0.125 metoprolol 50
13	3	CHF	digoxin 0.125
14	1	PAT	digoxin 0.125 metoprolol 50
15	3	VT, PAF	propranolol 80 digoxin 0.625
16	1	PAFI	amiodarone 600
	3		amiodarone 200
17	1	PAT	digoxin 0.125 metoprolol 50
18	1	PAFI	digoxin 0.25 verapamil 240
19	1	CHF	digoxin 0.125
		AP	atenolol 100
20	1	PAF	quinidine 600
21	1	CHF	digoxin 0.125
22	1	CHF	digoxin 0.125
23	1	VT	amiodarone 200
			digoxin 0.0625
24	1	PAF	metoprolol 200

PAF=paroxysmal atrial fibrillation; CHF=congestive heart failure; PAFI=paroxysmal atrial flutter; PAT=paroxysmal atrial tachycardia; AP=angina pectoris; VT=ventricular tachycardia; MO/FU=months of follow-up.

second degree AV block (type 1) at the permanently programmed atrial pacing rate of 80 beats min⁻¹. The amiodarone dosage was reduced to 200 mg daily and she remained asymptomatic during further follow-up. This patient was the only one of group 2 in whom the dosage of antiarrhythmic drug therapy was changed during follow-up. No other patient of group 2 showed signs or symptoms of second or third-degree AV block. During chronic (>3 months) antiarrhythmic drug therapy the variability of the AVWHR of group 2 patients was equal to or less than 10 beats min⁻¹, with respect to the AVWHR obtained 3 months after the initiation of oral drug therapy.

One patient of group 1 was hospitalized during follow-up because of a transient ischaemic attack and 2 patients of group 2 were hospitalized because of anginal pain. None of our patients developed chronic atrial fibrillation and all patients were alive and well when follow-up evaluations were completed in September 1983.

Discussion

There has been considerable reluctance in the past to implant atrial demand pacemakers in patients with symptomatic sinus node dysfunction. Ventricular demand or dual chamber pacemakers have been used in preference because of a concern about deterioration of anterograde AV conduction during follow-up.

Several investigators^[9-11] have reported an increased risk of second- or third-degree AV block during long-term follow-up of patients with an atrial demand pacemaker. Others^[12,13] observed a very low incidence of impaired anterograde AV conduction during long-term follow-up.

Our results suggest that deterioration of anterograde AV conduction was related to the use of antiarrhythmic drugs during follow up, rather than to degeneration of the AV conducting system.

In our 11 patients, who did not require antiarrhythmic drug therapy, the development of second-

Table 5 AVWHR of patients with antiarrhythmic drugs during follow-up

Patients	Follow-up										
	EPS	3 mths	6 mths	9 mths	12 mths	15 mths	18 mths	21 mths	24 mths	27 mths	30 mths
12	130	130	130	120*	120*	120*	110*	-	120*	120*	120*
13	170	170	160*	160*	160*	160*	160*	160*	170*	160*	
14	190	170*	170*	160*	170*	160*	160*	160*			
15	≥160	190	160*	160*	170*	160*	160*				
16	120	80*	110*	110*	110*	160*					
17	140	130*	130*	120*	130*						
18	180	140*	140*	130*	140*						
19	190	140*	130*	130*	130*						
20	130	130*	130*	130*	130*						
21	120	110*	110*	120*							
22	140	130*	120*	130*							
23	120	110*	110*	110*							
24	180	150*	150*								

*The AVWHR after 3 months of oral antiarrhythmic drug therapy.

AVWHR = atrial pacing rate producing AV Wenckebach phenomenon; EPS = electrophysiologic study before pacemaker implantation.

or third-degree AV block was not observed during long-term follow-up. In one patient of our 13 patients who did require chronic antiarrhythmic during therapy symptomatic second-degree (type 1) AV block occurred, as a consequence of amiodarone therapy. In all other patients on chronic antiarrhythmic drug therapy no symptoms or signs of second- or third-degree AV block developed.

Several factors may explain the discrepancy between our results and those of other investigators:

(1) *Variability in patient population chosen for study.* Since the diagnostic criteria for sinus node dysfunction are necessarily broad [14], it is understandable that our candidate population was different from the patient population of other investigators. Rasmussen *et al.* [8] observed that more than 50% of patients with sinus node dysfunction demonstrated some kind of atrio-ventricular conduction disease. However, a prolonged sinus node recovery time was used as the sole criterion for defining an abnormal sinus node dysfunction.

(2) *Criteria for atrial pacemaker implantation.* In our series we based the decision to implant an atrial demand pacemaker on two electrophysiologic criteria, a normal HV interval during baseline recording and the persistence of 1:1 AV conduction during incremental high right atrial pacing at a rate of at least 120 beats min^{-1} . In the study of Perrins *et al.* [9], criteria to assess intact AV conduction prior to pacemaker implantation were not mentioned. In the study of Bertholet *et al.* [10] complete electrophysiologic studies were performed in only 6 out of 26 patients. It is therefore conceivable that these investigators had included in their study patients with already impaired AV nodal or infra nodal conduction.

The presence of a normal PR interval (<0.20 s) and a narrow QRS complex (<0.10) on the electrocardiogram or Holter tracing can also be used as an indication of intact AV conduction. Unfortunately, there would appear to be no definitive criterion, since normal intervals are no absolute guarantee of intact AV conduction and prolonged intervals may not always indicate impaired AV conduction when compared with invasive electrophysiologic studies [2, 11].

(3) *Antiarrhythmic drug therapy after pacemaker implantation.* Certain antiarrhythmic drugs are known to adversely affect AV nodal function in

individuals with intact anterograde AV conduction [16–22]. However, the exact influence of these drugs on anterograde AV conduction in patients with symptomatic sinus node dysfunction has not been systematically studied. Dekker *et al.* [11] followed 104 patients with symptomatic sinus node dysfunction for at least 28 months after single-chamber atrial pacemaker implantation. A progressive impairment of AV conduction was noted especially in patients with supraventricular arrhythmias after pacemaker implantation, a finding not confirmed by other investigators [10, 13]. These conflicting observations may result from differences in choice and dosage of antiarrhythmic agents that may impair anterograde AV conduction.

In conclusion, our data suggest that deterioration of anterograde AV conduction may be more related to the use of antiarrhythmic drugs, rather than to degeneration of the AV conducting system. In patients with symptomatic sinus node dysfunction but intact anterograde AV conduction at the initial electrophysiologic study single chamber atrial pacemaker proved to be safe, even when antiarrhythmic drug therapy was used. Further studies with a long period of follow-up will be necessary to confirm the safety of atrial pacing in patients with apparently intact AV conduction.

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Comment by K. Rasmussen (Tromsø, Norway)

Van Mechelen and coworkers have performed serial electrophysiological measurements of the A-V system, by a simple but adequate method, in patients with sinus node dysfunction. They have interpreted their results as indicating that, in most subjects with isolated sinus node disease, it is sufficient to perform simple atrial pacing. This message may be important since it permits us to simplify a pacing technology which has increasingly tended to overwhelm us.

These results are apparently opposed to the common concept of a generalized conduction disease which inevitably attacks various levels of the conduction system, and in which A-V conduction disturbances are always threatening patients with sinus node disease. However, this discrepancy may be only apparent. The patients were selected as having no A-V conduction disease. It is not surprising that this did not emerge in the 11 patients followed from 6 to 36 months without drugs. In the presence of a generalized conduction disease progressing in a stepwise and random manner, a longer observation period in a larger number of patients would be needed in order to assess accurately the incidence of cases needing additional ventricular pacing.

Even the practical conclusions from this paper should be drawn with some caution. It is required that the A-V conduction system has been tested and found normal. The patients may need a more frequent and accurate follow up than was otherwise necessary. Furthermore, as is evident from the report, antiarrhythmic drugs will have to be used cautiously in order not to unmask latent A-V conduction disease. If these requirements can be met, the authors are probably correct in saying that simple atrial pacing, as opposed to the more elaborate pacing systems, imposes a very small additional risk on these patients. The conclusion is in accordance with a good general rule, treat what you know about and take care of progressions when they appear.

Responses of an AV Universal (DDD) Pulse Generator (Cordis 233D) to Programmed Single Ventricular Extrastimuli

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VAN MECHELEN, R., ET AL.: Responses of an AV universal (DDD) pulse generator (Cordis 233D) to programmed single ventricular extrastimuli. To evaluate factors playing a role in initiation and perpetuation of pacemaker-mediated tachycardias (PMTs), 22 consecutive patients with symptomatic conduction disorders were studied after implantation of an AV universal (DDD) pulse generator (Cordis 233D). Patients were divided into two groups, depending upon the presence or absence of ventriculo-atrial (VA) conduction during electrophysiological study (EPS) performed before pacemaker implantation. PMTs could be initiated in six of eight patients of Group I and in none of 14 patients of Group II. Initiation and perpetuation of PMTs during DDD pacing were dependent upon the capacity of the patient to conduct ventricular premature beats (VPBs) and subsequent paced ventricular beats retrogradely to the atria, and upon three programmable parameters of the pulse generator (AV delay period, upper rate limit, tachycardia response).

Programmed single ventricular extrastimulation demonstrated that: (1) merely the presence of VA conduction during EPS, although necessary, was not sufficient to induce PMTs after DDD pacemaker implantation; (2) VPBs introduced late rather than early in the cardiac cycle initiated PMTs in a different way; (3) the initiation of PMTs could be prevented during study by adjusting the programmable parameters (AV delay period, upper rate limit, tachycardia response); (4) one of the two available tachycardia responses of the pulse generator (gradual fall-back response) was able to terminate and initiate PMTs consistently.

These observations helped in understanding the responses of the Cordis 233D pulse generator to ventricular premature beats. They indicate that additional refinement of the pulse generator is necessary to solve the problem of PMT. (PACE, Vol. 7, March-April 1984)

AV universal (DDD) pulse generator, pacemaker arrhythmias, pacemaker-mediated tachycardia, programmable pacemaker, ventriculo-atrial (VA) conduction.

Introduction

With the introduction of AV universal (DDD) pulse generators, clinicians have a dual-chamber pacemaker at their disposal that can restore AV synchrony and/or could offer an atrial rate on demand. Therefore, these units have been applied to patients with symptomatic sinus node dysfunction,

with symptomatic second or third degree AV block, and with a combination of both conditions.

Unfortunately, the DDD pacing mode in presently available AV universal (DDD) pulse generators is not recommended for patients with ventriculo-atrial (VA) conduction^{1,2} because blocked atrial premature beats, ventricular premature beats and myopotentials sensed by the atrial channel may induce tachycardias mediated by the pulse generator.¹⁻³ Also, the application of a magnet above a DDD pulse generator, or chest wall stimulation at a rate faster than the spontaneous sinus rate, may induce pacemaker-mediated tachycardias.⁴

As a consequence of this limitation, clinicians must establish the presence or absence of VA con-

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duction in patients eligible for AV universal (DDD) pulse generators before deciding to apply the DDD pacing mode permanently.

The electrocardiogram is often of little help in establishing VA conduction before pacemaker implantation; only electrophysiological investigation is a reliable indicator. For this reason, all patients in this study underwent an electrophysiological investigation before an AV universal DDD pulse generator* was implanted. After pacemaker implantation, single ventricular extrastimuli were introduced during DDD pacing in order to demonstrate the consequences of ventricular premature beats for the DDD pacing mode of this unit.

Material and Methods

Patients

The decision to implant a DDD pulse generator in patients with symptomatic conduction disorders was based upon: (1) atrial and ventricular electrophysiological function; (2) physical activity of the patient.

The unit was not used in patients with chronic atrial fibrillation, with paroxysmal supraventricular tachycardias, with known ventricular arrhythmias or in patients whose physical activity was limited even before the onset of conduction disorders.

Electrophysiological Studies

Following informed consent, electrophysiological studies were performed in patients who met the selection criteria. The patients received no medication prior to the study. Catheters were placed for both stimulation and recording of intracardiac electrograms at the right ventricular apex, tricuspid valve ring, and high lateral right atrium. Patterns of AV and VA conduction and refractory periods were determined using the extrastimulus technique during pacing from the high right atrium and right ventricular apex at several basic cycle lengths.⁵

Incremental ventricular pacing at steps of 10 bpm was initiated at a beginning rate 10-15% faster than the existing sinus rate, up to a pacing rate of 160/min, or up to a rate at which second degree VA block occurred. Each period lasted one minute. Criteria for determining the presence of 1:1 VA conduction during pacing were: (1) atrial activation sequence from low right atrium (LRA) to high right atrium (HRA); (2) identical ventricular and atrial cycle lengths at the ventricular pacing rate; (3) constant VA conduction times at one and the same pacing rate. Only data relevant to the DDD pacing mode of the AV universal pulse generator will be presented.

Pulse Generator

The microprocessor-based DDD unit consists of an atrial and a ventricular channel, with separate timing cycles, which operate in synchrony. The timing cycles of both channels are divided into two parts: an alert period and a refractory period. During the alert periods the pulse generator responds to spontaneous atrial and ventricular events; during the refractory periods of either atrial or ventricular channel or both, the pulse generator will not produce output pulses to cardiac signals. A sub-part of the refractory period is the blanking period. This blanking period (50 ms) will only be effective when an output pulse is delivered by the atrial or ventricular channel. During the blanking period atrial and ventricular sensing amplifiers are blocked to prevent cross-talk. The specifications of the pulse generator are listed in Table I.

Atrial Tachycardia Responses

To prevent atrial synchronous rates exceeding the upper rate limit of the pulse generator, two independent modes of response are available in this unit: the AV block response and the gradual fall-back response.

AV Block Response

When the pulse generator is programmed to the AV block response, the atrial refractory period equals the cycle length (ms) of the programmed maximum atrial synchronous pacing rate (Table

*Cordis 233D, Cordis Corp., Miami, Florida, U.S.A.

Table I.

Specifications of the AV Universal (DDD) Pulse Generator

<i>Programmable specifications</i>	
Minimum rate, bpm	: 50, 60, 70, 80
Maximum rate, bpm	: 100, 130, 160, 180
Atrial tachycardia response	: 2:1 AV block, gradual fall back*
AV delay period, ms	: 80, 120, 165, 250
Atrial sensitivity, mV	: 0.8, 1.5, 7.0, off.
Ventricular sensitivity, mV	: 0.8, 1.5, 2.5, off.
Atrial output, ms	: 0.5, 1.0, 1.5, off.
Ventricular output, ms	: 0.5, 1.0, 1.5, 2.0
Atrial refractory period,** ms	: 600, 460, 375, 333
<i>Non-programmable specifications</i>	
Atrial refractory period,* ms	: 305
Ventricular refractory period, ms	: 305

*When the pulse generator is programmed to gradual fall-back response the atrial refractory period is not programmable but fixed. For explanation of the gradual fall-back response see text.

**The atrial refractory period is only programmable when the 2:1 AV block response to atrial tachycardia is programmed. The atrial refractory period equals the cycle length of the programmed maximum rate.

I). At atrial rates exceeding the maximum atrial synchronous pacing rate, the pulse generator shifts abruptly from a 1:1 AV synchronous pacing mode to a 2:1 AV synchronous pacing mode.

Fall-back Response

The fall-back response is aimed at a more gradual slowing down of the ventricular rate when the maximum atrial synchronous pacing rate is exceeded. This mode is based on the computational power of the incorporated microprocessor. As the atrial rate exceeds the upper rate limit, the fall-back algorithm allows up to 4 atrial synchronous ventricular pacing pulses (limited by a 190/min pace limit). After a pause, the pulse generator automatically shifts from atrial-synchronous pacing to ventricular demand pacing at a rate slightly below the programmed maximum rate (for a period of 3.6 seconds). The ventricular rate then gradually decreases until the programmed fall-back rate is achieved. When, during the gradual fall-back response, evidence exists of an atrial rate below the programmed upper rate limit, the fall-back routine is discontinued and AV synchronous pacing is resumed.

Programmed Single Ventricular Extrastimulation

After pacemaker implantation all pulse generators were programmed in the DDD pacing mode: upper rate limit, 180/min; AV block response; minimum rate, 50/min; AV delay period, 250 ms; atrial sensitivity, 0.8 mV; ventricular sensitivity, 2.5 mV; atrial output, 1.5 ms; ventricular output, 2 ms.

The studies were performed using a temporary bipolar catheter electrode already positioned in the right ventricular apex before pacemaker implantation, a programmable stimulator (Janssen SU2) and an electrocardiograph. To avoid overcharging of the sensing amplifiers in the pulse generator and to prevent sensing of the stimulation pulse by the atrial amplifier (far-field sensing), the output voltage was set to an amplitude just above the diastolic threshold for ventricular stimulation (less than 0.75 V). Capture of the ventricular extrastimulus could be assured by adjusting pulse duration (greater than 0.5 ms).

After every 8 atrial synchronous (paced) ventricular beats, a single ventricular extrastimulus was introduced at progressive prematurity in steps of 50 ms decrements, until ventricular refractoriness was reached. When a tachycardia was initiated, a magnet was applied over the pulse generator to terminate the arrhythmia. Magnet application during pacemaker-mediated tachycardia produced asynchronous emission of both atrial and ventricular stimuli at the programmed minimum rate. The tachycardia zone was determined and the studies were repeated after reprogramming the AV delay period from 250 ms to 165 ms and 120 ms, respectively; after programming the upper rate limit of the pulse generator below the tachycardia rate; and after programming the gradual fall-back response to atrial tachycardia instead of the AV block response.

Results

Patient data are shown in Table II; electrophysiological data are listed in Table III and Table IV.

ECG Analysis

When a ventricular extrastimulus was introduced late in the cardiac cycle, the subsequent spontaneous sinus beat could elicit a ventricular

Table II.
Clinical Data

	<i>Patients</i>	22
	<i>Age Range (yrs)</i>	52-93
<i>Sex</i>	male	10
	female	12
<i>Symptoms</i>	syncope	7
	dizziness	9
	CHF, dizziness	6
<i>Conduction disorders:</i>	SND	5
	AVB II	2
	type I	1
	type II	1
	AVB III	15

SND = sinus node dysfunction; AVB II = second degree AV block; AVB III = third-degree AV block; CHF = congestive heart failure.

response from the pulse generator, but no response from the ventricular myocardium because of refractoriness (Fig. 1a). With progressively premature ventricular extrastimuli the following sensed atrial event elicited a ventricular response from the pulse generator and the ventricular myocardium. In patients without VA conduction, ventricular extrastimuli did not influence the spontaneous cycle length. However, in patients with VA conduction the ventricular response of the pulse generator to the following spontaneous sinus beat induced ventriculo-atrial (VA) conduction, thereby initiating a pacemaker-mediated tachycardia (Fig. 1b). The retrograde limb of the artificial re-entrant tachycardia was provided by the VA conduction pathway and the anterograde limb was provided by the programmed AV delay period of the pulse generator.

Still shorter coupled ventricular extrastimuli also initiated pacemaker-mediated tachycardias in patients with VA conduction. However, the sequence of initiation was different (Fig. 1c). When ventricular extrastimuli were introduced late in the car-

Table III.
Electrophysiological Data (I)

<i>Patients without VAC</i>	<i>sex</i>	<i>age (yrs)</i>	<i>CD</i>	<i>ECG</i>	<i>AVC (bpm)</i>	<i>AH (ms)</i>	<i>HV (ms)</i>
1	f	77	AVB III	SR,AVJR	—	—	40
2	f	93	AVB III	SR,VR	—	100	—
3	f	78	SND	SB,LAHB	60-120	250-340	60
			AVB I				
4	m	86	AVB III	SR,VR	—	110	—
5	m	60	AVB III	SR,VR	—	150	—
6	m	89	AVB III	SR,VR	—	120	—
			AVB I	SR,LBBB	50-80	120	100
7	m	72	AVB III	SR,VR	—	140	—
8	f	70	AVB II	SR,RBBB	70-110	90-100	50
			(type 2)				
9	f	80	AVB III	SR,VR	—	100	—
10	m	73	AVB III	SR,VR	—	110	—
11	m	74	AVB III	SR,VR	—	100	—
12	f	90	AVB III	SR,VR	—	140	—
13	m	52	AVB III	SR,AVJR	—	—	70
14	f	68	AVB III	SR,VR	—	100	—

VAC = VA conduction; CD = conduction disorder; SR = sinus rhythm; SB = sinus bradycardia; AVJR = AV junctional rhythm; VR = ventricular rhythm; AVC = range of 1:1 AV conduction during incremental atrial pacing; LAHB = left anterior hemiblock; RBBB = right bundle branch block; LBBB = left bundle branch block; AVB I, II, III = first, second and third degree AV block.

Table IV.
Electrophysiological Data (II)

Patients with VAC	sex	age (yrs)	CD	ECG findings	AVC (bpm)	AH (ms)	HV	VAC (bpm)	VACT (ms)
15	f	77	SND	SB,SAB,AVJR	50-110	110-170	45	60-80	200-210
16	f	72	SND	SB,AVJR,LAHB	50-110	90-190	45	60-120	180-230
17	f	73	AVB III	SR,AVJR	—	—	50	60-140	180-200
18	m	93	AVB III	SR,AVJR	—	—	55	80-150	200-230
19	m	70	SND	SR,RBBB	60-110	180-250	50	70-110	270-300
20	f	72	SND	SAB,SB,A Flutter	50-140	110-170	45	60	400
21	m	50	AVB III	SR,VR	—	100	—	90-130	200-230
			NAVC	SR,RBBB	60-120	100-120	50		
22	f	78	AVB II (type I)	SR,RBBB	60-80	100	50	70-130	170-180

VAC = VA conduction, range of 1:1 VA conduction during incremental ventricular pacing; AVC = range of 1:1 AV conduction during incremental atrial pacing; CD = conduction disorder; VACT = VA conduction times measured from the ventricular spike to the onset of A HRA; SB = sinus bradycardia; AVJR = AV junctional rhythm; SND = sinus node dysfunction; SAB = sino-atrial block; LAHB = left anterior hemiblock; RBBB = right bundle branch block; VR = ventricular rhythm; AVB I, II, III = first, second and third degree AV block.

diac cycle, collision of these ventricular beats occurred with the following spontaneous sinus beat; the paced ventricular beat in response to this sinus beat could (when the retrograde pathway was no longer refractory) be conducted retrogradely to the atria, and initiate a pacemaker-mediated tachycardia. When ventricular extrastimuli were introduced more prematurely, VA conduction initiated a pacemaker-mediated tachycardia.

When ventricular extrastimuli were introduced during the refractory period of the ventricular channel, refractoriness of the ventricular myocardium was reached in 8 of 14 patients without VA conduction and in 5 of 8 patients with VA conduction. In patients without VA conduction these premature beats did not interfere with the spontaneous cardiac cycle. In patients with VA conduction the response of the pulse generator was dependent upon the presence of VA conduction at these short coupling intervals. Again, if VA conduction was present, artificial re-entrant tachycardias could be initiated; if VA conduction was absent, the spontaneous cardiac cycle was not influenced by these extrastimuli.

The cycle lengths of the tachycardias could be shortened (1) as long as 1:1 VA conduction was present by reducing the programmed AV delay period (Table V and Fig. 2a, b, c); (2) as long as the

rates of the tachycardias did not exceed the upper rate limit of the pulse generator.

All tachycardias could easily be terminated by applying a magnet over the pulse generator (Fig. 3). We also tested the responses of the pulse generator to atrial rates exceeding the maximum atrial synchronous pacing rate. By programming the upper rate limit below the tachycardia rate, in combination with the AV block response, all tachycardias could be terminated (Fig. 4). It was, however, impossible to terminate these tachycardias by programming the fall-back response (Fig. 5). The reason for this was that if 1:1 VA conduction was present at a ventricular paced rate exceeding the upper rate limit of the pulse generator, 1:1 VA conduction was also present at ventricular pacing rates below that rate, unless the spontaneous atrial rate exceeded that particular ventricular pacing rate. As a consequence, atrial activity sensed by the atrial channel at a rate below the programmed upper rate limit terminated the fall-back response. AV synchronous pacing resumed and the upper rate limit was again exceeded. This, in turn, activated the fall-back tachycardia intervention program and the sequence was repeated perpetually as long as 1:1 VA conduction was present.

In 2 cases (patients 15 and 20) with symptomatic sinus node dysfunction and 1:1 AV conduction

Figure 1. (A) During spontaneous sinus rhythm (CL = 1040 ms) one ventricular extrastimulus was introduced with a coupling interval of 700 ms. The pulse generator was programmed in the DDD pacing mode; minimum rate 50/min; upper rate limit 180/min; AV delay period, 250 ms; tachycardia response, AV block. A ventricular spike is produced by the pulse generator in the T wave of the ventricular premature beat as a consequence of synchronization with a sinus beat hidden in the QRS complex of the premature ventricular beat. (B) A ventricular extrastimulus is introduced with a coupling interval of 600 ms. A pacemaker-mediated tachycardia is initiated. Note the mechanism of initiation (see text). The tachycardia cycle length is 550 ms (110/min). Sensing of the retrograde P wave occurs 300 ms (550-250) after the preceding ventricular pacer spike. (C) A ventricular extrastimulus is introduced with a coupling interval of 400 ms. A pacemaker-mediated tachycardia is initiated by VA conduction of the ventricular premature beat. Note the difference of tachycardia initiation with ventricular extrastimuli occurring very late during the cardiac cycle (see text).

during incremental atrial pacing up to a rate of 110/min and 140/min, VA conduction was only present during incremental ventricular pacing up to a rate of 60/min and 80/min (Tables IV and V). In these patients, pacemaker-mediated tachycardia could not be initiated and VA conduction was not sustained during the ventricular extra-

stimulus study, even at the largest programmed AV delay period of 250 ms.

When ventricular extrastimuli were introduced during the atrial blanking period (50 ms), a ventricular pacer spike was produced after termination of the programmed AV delay periods (Fig. 6).

Depending upon the presence or absence of VA

Table V.
Pacemaker-mediated Tachycardias in Patients with VA Conduction

Patients	HR (bpm)	TR (bpm) AVD 250 ms	TR (bpm) AVD 165 ms	TR (bpm) AVD 120 ms	VACT (ms)	VERP (ms)
15	70-75	—	—	—	—	300-350
16	80-82	130	160	—	210	250-300
17	80	130	150	170	210-230	250
18	60-65	140	170	—	180-190	250
19	50-55	110	130	—	300	300
20	80-90	—	—	—	—	300
21	70-80	120	—	—	250	300
22	70-80	120	144	160	250	250

HR = heart rate during DDD pacing; TR = tachycardia rate; AVD = programmed AV delay period; VACT = VA conduction times, calculated by subtracting the programmed AV delay period from the tachycardia cycle length (VACT = TCL - AVD). Note: all pulse generators were programmed at an upper rate limit of 180/min in combination with AV block response to atrial tachycardias exceeding the upper rate limit. This programming does not allow induction of tachycardias at rates exceeding 180/min.

conduction the duration of these studies varied from 15 minutes to 30 minutes.

Reprogramming of the Pulse Generator

In patients without VA conduction, the DDD pacing mode was applied permanently. The AV delay period was adjusted depending upon the presence of intact or impaired AV conduction, and

the minimum rate of the pulse generator was adjusted according to the presence or absence of sinus node dysfunction.

In patients with VA conduction, either the DVI mode was used permanently, or the DDD mode was applied and programmed parameters were adjusted to prevent pacemaker-mediated tachycardia (AV delay period, upper rate limit, tachycardia response).

Figure 2a. A pacemaker-mediated tachycardia is induced by one ventricular extrastimulus. The first two retrograde P waves are marked with an arrow. At a programmed AV delay period of 250 ms, the rate of the tachycardia is 128/min (CL = 470 ms, VACT = 220 ms).

Figure 2b. At a programmed AV delay of 165 ms, the rate of the tachycardia is 150/min (CL = 400 ms, VACT = 235 ms). Retrograde P waves are clearly seen on top of the T wave of the paced ventricular beats.

Discussion

The patterns of response of an AV universal (DDD) pulse generator to programmed single ventricular extrastimuli were analyzed in 22 patients with symptomatic conduction disorders.

The unit functioned well as a DDD pacer in 14 patients without VA conduction. Nevertheless, pacemaker spikes could occur during the vulner-

able period of the ventricles (Fig. 1a), and might induce ventricular tachycardia and fibrillation, particularly in patients with ischemic heart disease.⁶⁻⁸ However it should be recalled that all early pacemakers functioning in the VOO mode could cause these phenomena without any evidence of significant adverse effects.⁹

In 6 of 8 patients with VA conduction, tachycardias could be initiated and sustained by the

Figure 2c. At a programmed AV delay of 120 ms, the rate of the tachycardia is 170/min (CL = 350 ms, VACT = 230 ms). Synchronization with retrograde P waves occurs for 5 beats. The fifth synchronized ventricular beat does not conduct retrogradely to the atria. Tachycardia is terminated by block in the retrograde limb of the circuit.

Figure 3. (A) Ventricular extrastimulus and tachycardia termination (same patient as in Fig. 1). A pacemaker-mediated tachycardia is terminated by 1 ventricular extrastimulus. The coupling interval with the preceding ventricular event is 360 ms. Because VACT equals 300 ms, the extrastimulus occurred (360-300) 60 ms after sensing of the retrograde P wave (during the AV delay of the pulse generator). By shortening the AV delay to 60 ms, tachycardia is terminated by block in the retrograde limb of the circuit. (B) Magnet application and tachycardia termination. Magnet application during pacemaker-mediated tachycardia produces asynchronous emission of both atrial and ventricular extrastimuli at the programmed minimum rate.

Figure 4. AV block response to pacemaker-mediated tachycardias. The upper rate limit of the pulse generator is programmed below the tachycardia rate. In this patient (same as in Fig. 2b) a tachycardia could be induced with a rate of 150/min (CL = 400 ms) at a programmed AV delay of 165 ms. The upper rate limit was programmed at 130/min; the atrial refractory period of the pulse generator then equals the cycle length of the programmed upper rate, 460 ms. The second retrograde P wave occurs within the atrial refractory period of the pulse generator and no tachycardia is induced. AC = atrial channel of the pulse generator; VC = ventricular channel of the pulse generator; AVD = AV delay period. Shaded areas represent refractory periods of atrial and ventricular channels.

Figure 5. Gradual fall-back response to atrial tachycardias. In this patient with a pacemaker-mediated tachycardia of 110/min at a programmed AV delay of 250 ms, the upper rate limit of the pulse generator is programmed to 100/min. The tachycardia is initiated by a ventricular extrastimulus with a coupling interval of 500 ms, and subsequent retrograde conduction to the atria. The pulse generator synchronizes with four atrial events, and after a pause, starts VVI, operating at a rate slightly below the programmed upper rate limit of 90/min (CL-650 ms) for a period of 3.6 seconds. One cycle is deliberately increased by 70 ms (CL-720 ms) to detect atrial activity that might occur during the atrial blanking period. The ventricular rate then gradually decreases until the programmed fall-back rate is achieved. However, if atrial events are sensed at a rate below the programmed maximum rate, AV synchronous pacing is resumed.

It is obvious that synchronization with atrial events after termination of the gradual fall-back response results in an atrial synchronous ventricular rate exceeding the programmed upper rate limit of the pulse generator; this in turn activates the fall-back tachycardia intervention program again. The sequence is repeated as long as 1:1 VA conduction is present.

pulse generator as long as the programmed upper rate limit of the pulse generator was greater than that of the tachycardia. The cycle lengths of the tachycardias were determined by the patients' VA conduction times and by the programmed AV delay periods. The tachycardia cycle lengths varied between 110/min up to 170/min; VA conduction times ranged from 170 ms to 300 ms during tachy-

cardia. In the remaining two patients with VA conduction it was impossible to initiate pacemaker tachycardia. This was due to the fact that sustained VA conduction, a prerequisite for pacemaker-mediated tachycardia, was not present during the study.

Initially, we programmed the pacer to the DVI mode in patients with VA conduction to avoid the

Figure 6. Ventricular extrastimulus during the atrial blanking period. When a programmed, single ventricular extrastimulus is introduced during the atrial blanking period (50 ms), neither the atrial channel nor the ventricular channel is alert for incoming signals. The pulse generator delivers a ventricular pulse at the end of the programmed AV delay period.

risks of pacemaker-mediated tachycardia. However, although DVI pacing will prevent this tachycardia, dual-chamber pacing without atrial sensing might induce atrial fibrillation,¹⁰ and atrial synchrony will be limited to the programmed pacing rate. Because of these imperfections we later changed our policy and programmed to the DDD pacing mode and programmable parameters (AV delay period, upper rate limit, tachycardia response) were adjusted to prevent pacemaker-mediated tachycardia. So far, we have not observed pacemaker-mediated tachycardias in our patients during follow-up. However, an adjusted DDD pacing mode is no absolute guarantee against these tachycardias, because the parameters at implantation might become ineffective at a later stage when the functional properties of the conduction

system during retrograde conduction change. This would leave the patient without any protection against the risks emerging from these pulse generators.

The introduction of programmed, single ventricular extrastimuli during DDD pacing helped our understanding of the possible risks of a presently available DDD unit. Our observations indicate that additional refinement of the pulse generator (Cordis 233D) is necessary to eliminate the problem of pacemaker-mediated tachycardia.

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Prevalence of Retrograde Conduction in Heart Block After DDD Pacemaker Implantation

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Electrophysiologic studies were performed before DDD pacemaker implantation in 50 patients with symptomatic heart block. The patients were separated into 2 groups. Group I consisted of patients with intact retrograde conduction and group II consisted of patients with blocked retrograde conduction. After pacemaker implantation, postventricular atrial refractory periods in patients in group I were programmed at 50 to 100 ms, in excess of the retrograde conduction times measured during electrophysiologic studies. In group II patients, postventricular atrial refractory periods were routinely programmed at 300 ms. During follow-up, patients visited the outpatient clinic at 3-month intervals for noninvasive assessment of the prevalence of retrograde conduction, and to test the inducibility of pacemaker-mediated tachycardias.

The mean follow-up of group I (15 patients) was 27 ± 10 months, whereas the mean follow-up of group II (35 patients) was 19 ± 9 months. The

mean number of noninvasive tests performed during follow-up was 8 ± 3 per patient for group I and 5 ± 3 per patient for group II. In group I, retrograde conduction remained intact in 12 patients ($p < 0.01$). In 29 of 31 patients in group II, retrograde conduction remained absent ($p < 0.01$). In 4 patients in group II, chronic atrial fibrillation occurred during follow-up. Chronic atrial fibrillation did not occur in any patient in group I. During serial electrophysiologic testing, no pacemaker-mediated tachycardias could be induced in any patient in group I or II.

These results suggest that electrophysiologic studies performed before pacemaker implantation reliably predict the prevalence of retrograde conduction during follow-up of patients with symptomatic heart block and that adjusted atrial refractory periods of the pulse generator at implantation prevent the induction of pacemaker-mediated tachycardias during serial electrophysiologic testing.

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Pacemaker-mediated tachycardias in patients with DDD units can be prevented by adjusting the postventricular atrial refractory period.¹⁻⁵ However, an adjusted atrial refractory period may become ineffective in avoiding pacemaker-mediated tachycardias when the functional properties of the atrioventricular (AV) conducting system for retrograde conduction change during follow-up.

We studied retrograde conduction invasively before and noninvasively after DDD pacemaker implantation to find out if the electrophysiologic studies performed before pacemaker implantation would reliably predict the prevalence of retrograde conduction

during long-term follow-up, and if selected postventricular atrial refractory periods at implantation would prevent pacemaker-mediated tachycardias during serial electrophysiologic testing.

Methods

Patients: DDD units were implanted in 50 patients with symptomatic second- or third-degree AV block. All patients were physically active before the onset of conduction disorder.

Electrophysiologic studies: After informed consent was obtained, patients underwent an incremental ventricular pacing study to assess the prevalence and persistence of retrograde conduction. For both stimulation and recording, 3 catheters were placed at the lateral high right atrial wall, tricuspid valve ring and the right ventricular apex. Incremental pacing at steps of 10 beats/min was initiated at a beginning rate that was slightly faster than the spontaneous sinus rate, up to a pacing rate at which second-degree ventriculo-

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TABLE I Clinical and Electrophysiologic Data

	Group I RC Present	Group II RC Absent
Patients	15	35
Age (yr)		
Range	57 - 97	37 - 90
Mean	75 ± 11	74 ± 11
Male/female	1.1	1.1
Site of AV block		
AV node	6	9
Intra-Hisian	1	
Infra-Hisian	5	19
Unknown	3	7
Retrograde conduction		
Conduction times (ms)	170 - 360	
Mean	237 ± 58	
Persistence (beats/min)	100 - 200	
Mean	129 ± 35	
FU duration	27 ± 10	19 ± 9
Complications during FU		
Pocket infection	1	
Chronic atrial fibrillation		4
Atrial malsensing	1	
Deaths	2 (13%)	7 (20%)

AV block = atrioventricular block; FU = follow-up; RC = retrograde conduction.

atrial block occurred. Each pacing period lasted 1 minute. Retrograde conduction time was defined as the interval from the ventricular spike to the onset of atrial activation at the high right atrial electrogram. Measurements were made at a paper speed of 100 mm/s.

Pulse generators: In all patients, DDD units were used with programmable postventricular atrial refractory periods. In patients with intact retrograde conduction during electrophysiologic studies (group I), a post-ventricular atrial refractory period was selected 50 to 100 ms in excess of the measured retrograde conduction time at electrophysiologic studies.

In patients with retrograde block (group II), a post-ventricular atrial refractory period of 300 ms was programmed.

Patient follow-up: Patients were prospectively followed in the outpatient clinic at intervals of 3 months. Holter recordings were performed in all patients during the first 6 weeks after hospital discharge and thereafter when history suggested suggested brady- or tachy-

FIGURE 1. Atrioventricular dissociation during VVI pacing. When the pulse generator is programmed from the DDD to the VVI pacing mode at a rate slightly faster than the spontaneous sinus rate, atrioventricular dissociation is present.

TABLE II Retrograde Conduction and Antiarrhythmic Drugs During Follow-Up in Group I

Pt	FU (mo)	Drugs (mg/day)	RC Tests	RC
1	42	...	14	+
2	39*	Digoxin 0.25	13	+
3	38*	Digoxin 0.25	9	+
		Digoxin 0.25 +	3	0
		Amiodarone 400		
4	34	—	10	0
5	33	Digoxin 0.25	5	+
		Digoxin 0.25 +	5	0
		Amiodarone 200		
6	32	...	10	+
7	31	...	9	+
8	28	...	8	+
9	23	...	7	+
10	22	...	5	+
		Diltiazem 180	3	+
11	22	Digoxin 0.125	7	+
12	21	Digoxin 0.125	6	+
13	19	Digoxin 0.0625	5	+
14	14	Digoxin 0.125	4	+
15	8	...	3	+
Mean ± SD	27 ± 10		8 ± 3	

* Patient died

FU = follow-up; RC = retrograde conduction.

arrhythmias. The noninvasive assessment of retrograde conduction was divided into 2 parts:

Prevalence of retrograde conduction: By programming the pulse generator from the DDD to the VVI pacing mode at a rate 10 to 15 beats/min faster than the spontaneous sinus rate, either AV dissociation or retrograde conduction was present. When spontaneous P waves were still present on the electrocardiogram, it was concluded that AV dissociation was present (Fig. 1). However when P waves disappeared or when P waves with a different polarity were visible in every ST-segment of the ventricular paced beats, it was inferred that retrograde conduction was present (Fig. 2).

Prevention of pacemaker-mediated tachycardia: In patients with intact retrograde conduction, the pulse generator was programmed in the VDD pacing mode

FIGURE 2. Retrograde conduction during VVI pacing. When the pulse generator is programmed from the DDD to the VVI pacing mode at a rate slightly faster than the spontaneous sinus rate, P waves are no longer visible, indicating the presence of retrograde conduction.

at a minimal rate of 10 to 15 beats/min faster than the spontaneous sinus rate. When ventricular pacing at the programmed minimal rate was observed, the postventricular atrial refractory period was adequate in preventing pacemaker-mediated tachycardia (Fig. 3). When ventricular pacing occurred at a rate faster than the programmed minimal rate, pacemaker-mediated tachycardia was present and the postventricular atrial refractory period had to be prolonged to prevent sensing of retrograde P waves (Fig. 4).

Statistical analysis: Comparisons between groups were made using Fisher's exact test for unpaired data. All data are presented as mean $\pm$ standard deviation.

Results

Table I lists the clinical and electrophysiologic data of group I and II patients. Table II and III list the antiarrhythmic drugs prescribed during follow-up.

Group I: After hospital discharge, a pacemaker pocket infection in 1 patient necessitated the removal of the endocardial DDD system, and an epicardial VVI unit was replaced 6 months after the first implantation. In this patient, the prevalence of retrograde conduction could still be assessed during follow-up. However, the inducibility of pacemaker-mediated tachycardia could no longer be studied. In patients in group I, the mean number of noninvasive tests performed after hospital discharge was 8 ± 3 per patient. In 12 patients, retrograde conduction remained intact ($p < 0.01$) throughout the complete follow-up period. In 2 of 3 patients in whom retrograde block occurred, the incidence was related to the initiation of amiodarone therapy to control supraventricular arrhythmias. During serial electrophysiologic studies, pacemaker-mediated tachycardias could not be induced in any of these patients. However, in 1 patient, it was necessary to reprogram the postventricular atrial refractory period,

because telemetry recording after pacemaker implantation demonstrated pacemaker-mediated tachycardia during the night (Fig. 5 and 6).

Holter tracings did not show pacemaker-mediated tachycardia in any patient in group I. During follow-up, 2 patients (13%) died: 1 in the hospital because of a cerebrovascular accident and 1 suddenly outside the hospital.

Group II: In 1 patient, atrial malsensing occurred within 3 months after pacemaker implantation, and

FIGURE 4. VDD pacing with an insufficiently prolonged postventricular atrial refractory period (PVARP) (in the same patient as in Figure 3). The pulse generator is again programmed from the DDD to the VDD pacing mode. However, the postventricular atrial refractory period is shortened from 300 to 200 ms and the atrioventricular delay period (AVD) is prolonged to 250 ms. A pacemaker-mediated tachycardia is initiated at a rate of 135 beats/min (cycle length 0.44 second).

FIGURE 3. VDD pacing with an adequately prolonged postventricular atrial refractory period (PVARP) at a paper speed of 25 mm/s. The pulse generator is programmed from DDD to the VDD pacing mode at a minimal rate slightly faster than the spontaneous sinus rate. Spontaneous P waves disappear and VDD pacing occurs at the programmed minimal rate. AVD = atrioventricular delay period.

FIGURE 5. A, ventricular pacing during electrophysiologic study is initiated at a rate of 80 beats/min. Retrograde conduction is present with a retrograde conduction time of 200 ms. B, retrograde conduction remains sustained up to 150 beats/min and the retrograde conduction time increases to 220 ms. AEG = atrial electrogram.

TABLE III Retrograde Conduction and Antiarrhythmic Drugs During Follow-Up in Group II

Pt	FU (mo)	Drugs (mg/day)	RC Tests	RC
16	42	Digoxin 0.125	13	0
17	37	...	12	0
18	35	Digoxin 0.25	2 (AF)	0
19	13*	Digoxin 0.25	8	0
20	34	Amiodarone 200 Digoxin 0.125 Disopyramide 500	11	0
21	32	...	10	0
22	7*	...	2	0
23	26*	Digoxin 0.0625 Metoprolol 200	10	0
24	29	Digoxin 0.25	2 (AF)	0
25	22	...	9	0
26	21	Digoxin 0.25	2 (AF)	0
27	21*	...	4	0
28	26	Atenolol 100	8	0
29	24	...	8	0
30	13*	Digoxin 0.25	4	0
31	17*	Digoxin 0.125	5	0
32	23	Digoxin 0.25	7	0
33	22	...	5	0
			2	+
34	19	...	8	0
35	18	...	6	0
36	16	Digoxin 0.125	1	+
			4	0
37	16	...	5	0
38	16	...	5	0
39	14	...	4	0
40	14	Digoxin 0.25	4	0
41	13	Digoxin 0.25	4	0
42	13	Metoprolol 200	4	0
43	12	Digoxin 0.25	1 (AF)	0
44	12	...	3	0
45	11	...	3	0
46	11	Metoprolol 200	3	0
47	6*	Digoxin 0.0625 + Amiodarone 200	2	0
			0	0
48	9	...	3	0
49	9	Digoxin 0.25	3	0
50	9	Sotalol 80	3	0
Mean $\pm$ SD	19 $\pm$ 9		5 $\pm$ 3	

* Patient died.

AF = atrial fibrillation; FU = follow-up; RC = retrograde conduction.

despite repositioning of the atrial lead, malsensing remained present. In this patient, it was also impossible to study the inducibility of pacemaker-mediated tachycardia, although the prevalence of retrograde conduction could be studied by programming the VVI pacing mode. In 4 patients, chronic atrial fibrillation occurred despite the administration of antiarrhythmic drugs or elective cardioversion. These 4 patients were excluded from further study. In the remaining 31 patients, the mean number of noninvasive tests performed after hospital discharge was 5 ± 3 per patient. In 29 patients, retrograde conduction remained absent throughout the complete follow-up period ($p < 0.01$). In 2 patients, retrograde conduction was intermittently present, but pacemaker-mediated tachycardias could not be induced.

Pacemaker-mediated tachycardias were not observed during either monitoring by telemetry or holter

FIGURE 8. Telemetry recording during the night after pacemaker implantation (same patient as in Figure 5). **A**, atrial premature beats (asterisks) elicit a ventricular response of the pulse generator when they occur later than 300 ms after a sensed ventricular complex (postventricular atrial refractory period [PVARP] = 300 ms). The ventricular paced beats are retrogradely conducted to the atrium and retrograde P waves (arrows) reset the spontaneous sinus cycle. No pacemaker-mediated tachycardia is initiated because the retrograde conduction time is shorter than 300 ms. **B**, atrial premature beats appear again, and a pacemaker-mediated tachycardia is initiated because the retrograde P waves now occur outside the PVARP of 300 ms. The cycle length of the pacemaker-mediated tachycardia is 560 ms, which means that with a programmed atrioventricular delay period (AVD) of 200 ms, the retrograde conduction time equals 360 ms (560–200). **C**, tachycardia is terminated by a sudden change of the retrograde conduction time to less than 300 ms. Panels **A**, **B** and **C** are continuous tracings.

recording. Seven patients (20%) died during follow-up. In the hospital, 3 of them died from heart failure, 2 patients from carcinoma and 1 patient from renal failure. Out of the hospital, 1 patient died from unknown cause.

Discussion

Serial electrophysiologic testing in humans has been limited by the invasive character of the procedure. However, several pacemakers are currently available that enable us to perform noninvasively serial electrophysiologic studies.

In our series, the prevalence of retrograde conduction was assessed by ventricular pacing. Recently, Mahmud et al⁶ suggested that the introduction of ventricular extrastimuli, coupled to AV sequential pacing, may elicit a higher prevalence of retrograde conduction than would be observed with ventricular pacing alone. We therefore may have underestimated the prevalence of retrograde conduction. However, Mahmud et al studied 13 patients with intact AV conduction, and the site of retrograde block in all patients was at the level of the AV node. It is unknown if

extrapolation of these results to patients with heart block is warranted. Schuilenburg⁷ demonstrated in patients with AV block that retrograde infranodal block is not uncommon, which may eliminate the facilitative effect of the AV sequential method on retrograde conduction to the atrium.

Taking this limitation into account, our results suggest that retrograde conduction remains stable in most patients with heart block during long-term follow-up after DDD pacemaker implantation. Pacemaker-mediated tachycardias could not be induced in any of our patients during serial electrophysiologic testing, suggesting adequate protection against these tachycardias. Unfortunately, these observations do not absolutely guarantee protection against pacemaker-mediated tachycardia, as was shown by 1 of our patients (Fig. 5 and 6). In addition, for safety precautions, we routinely programmed a postventricular atrial refractory period of 300 ms in patients with retrograde block during preoperative studies, thereby precluding the consequences of retrograde conduction with retrograde conduction times less than 300 ms (which might have occurred intermittently during follow-up).

Our approach of lengthening postventricular atrial refractory periods proved to be a safe strategy during long-term follow-up. However, we had to accept that maximal tracking rates of the pulse generators were limited. In this regard, several suggestions were made

by Den Dulk et al⁸: Bipolar atrial sensing will prevent myopotentials from initiating a pacemaker-mediated tachycardia; reset of a prolonged postventricular atrial refractory period after a sensed atrial or ventricular premature beat will prevent sensing of retrograde P waves; and DDD escape, instead of DVI escape, after a premature beat will block subsequent retrograde conduction. In future DDD systems the problem of retrograde conduction must be solved without compromising the upper rate limit of the pulse generator.

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Pacemaker Electrocardiography of Rate Smoothing During DDD Pacing

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VAN MECHELEN, R., ET AL.: Pacemaker electrocardiography of rate smoothing during DD pacing. A rate smoothing option is available in a new bipolar AV universal (DDD) pacemaker. In three patients, two with intact retrograde conduction and one with retrograde block, rate smoothing values of 3% and 6% were programmed. Irregular pacemaker-mediated tachycardia occurred in one patient and AV synchrony was temporarily lost in the other two patients. In this report, we describe the pacemaker electrocardiography of rate smoothing during DDD pacing. (*PACE*, Vol. 8, September-October, 1985)

AV universal (DDD) pacing, pacemaker electrocardiography, rate smoothing

Introduction

The rate smoothing option in a new bipolar DDD pacemaker (CPI* 925) is intended to eliminate large cycle-to-cycle variations in rate by preventing the paced rate, either atrial or ventricular, from changing by more than a programmed percentage from one cardiac cycle to the next.¹ In this report we present three patients in whom the rate smoothing option was used.

Methods and Materials

Patients

AV universal (DDD) pacemakers were implanted in three patients with complete heart block. Patients had been physically active before the onset of conduction disorder.

Electrophysiologic Studies

Patients underwent an electrophysiologic evaluation to assess the prevalence and persistence of retrograde conduction. The study was conducted according to previously described catheter techniques.²⁻⁴

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Pulse Generator

The CPI 925 bipolar DDD pulse generator is a multiprogrammable, microprocessor-based pacemaker that can function in any standard single or dual chamber pacing mode. Rate smoothing values (RSV) may be set at 3%, 6%, 12.5%, or 25%. When the rate smoothing option is on, the pulse generator stores in its memory the most recent RR interval and does not allow the next RR interval to be less than the preceding RR interval minus the programmed percentage nor does it allow it to be greater than the preceding RR interval plus the programmed percentage.

Based on the previous RR interval and the selected smoothing value, the pulse generator sets up two windows—one for atrial events and another for ventricular events:

atrial window

$$= (\text{previous RR interval} \pm \text{RSV}) - \text{AV delay}$$

ventricular window

$$= \text{previous RR interval} \pm \text{RSV}$$

The timing for both windows is initiated at the end of a ventricular event. When an atrial event occurs before the atrial synchronization window (e.g., APB), the ventricle is paced after an interval that equals the previous RR interval - RSV. When an atrial event occurs within the atrial synchronization window (e.g., sinus beat), the programmed AV delay period is initiated and

the ventricle is paced at the end of the AV delay period. When no atrial event occurs during the time represented by (previous RR interval + RSV) - AV delay, the atrium is paced at the end of this interval and the ventricle at the end of the programmed AV delay period. When a ventricular event occurs before the ventricular window (e.g., VPB), the timing circuit is reset at an interval equal to the previous RR interval - RSV and a new cycle begins. When a ventricular event occurs within the ventricular synchronization window, ventricular output of the pulse generator is inhibited and the next RR interval will be equal to the previous RR interval $\pm$ RSV.

Case Reports

Patient No. 1

A 75-year-old man was admitted to the hospital because of Adam-Stokes attacks. The ECG on admission showed third-degree AV block with a ventricular rate of 30 beats/min, and a sinus rate of 70 beats/min. The QRS duration was 0.16 s; the morphology of the QRS complex suggested a ventricular origin of the secondary pacemaker. Monitoring by telemetry showed that heart block was intermittently present.

Electrophysiologic study performed prior to pacemaker implantation demonstrated sinus rhythm with intact AV' conduction. The AA interval was 1,050 ms, the AH interval was 100 ms, and the HV interval was 50 ms. During incremental atrial pacing, infranodal block occurred at an atrial pacing rate of 90 beats/min. During ventricular pacing retrograde conduction was blocked. The site of retrograde block was infra-His.

A CPI 925 pacemaker was implanted. At implantation, a postventricular-atrial refractory period (PVARP) was programmed to 250 ms, the AV delay period to 250 ms, and the RSV to 3%.

The fourth day after pacemaker implantation, the patient was discharged from the hospital. A routine 24-hour Holter recording was performed during the first 6 weeks after hospital discharge. An emergency call from the Holter center warned us that there was something wrong with the pacemaker. Although the patient did not have any complaints written in his diary, irregular tachycardias occurred repeatedly.

Figure 1 shows a representative tracing of the 24-hour Holter recording, during which 20 episodes of irregular tachycardia occurred. Analysis of the ECG (Fig. 1A) demonstrated that tachycardia was initiated by a sensed atrial event. Although the Holter lead does not clearly show a premature atrial beat, it can be inferred that the pulse generator must have sensed an atrial event that occurred before the atrial synchronization window because during DDD pacing a ventricular response of the pulse generator is always preceded by a sensed or paced atrial event. As a result of the programmed percentage of 3%, the ventricular pacing cycle length gradually decreased (Fig. 1B). The ventricular pacing rate therefore gradually increased. At a rate of ± 110 beats/min, (Fig. 1C) atrial pacing suddenly started at a gradually decreasing rate (Fig. 1C, D) until the spontaneous sinus rate exceeded the atrial pacing rate.

The most likely explanation of this pacemaker behavior is that although retrograde conduction was blocked during electrophysiologic study, it was no longer blocked during Holter recording and with a retrograde conduction time to the atrium that exceeded the programmed PVARP (250 ms) a pacemaker-mediated tachycardia could occur. The tachycardia was increasing in rate because of the rate smoothing option. At a rate of 110 beats/min, retrograde conduction was no longer sustained and subsequently atrial pacing occurred because no atrial event was sensed before or during the atrial synchronization window. The pacing rate then gradually decreased again due to the rate smoothing option as long as the paced atrial rate exceeded the spontaneous sinus rate. The problem was solved by programming a prolonged PVARP. The rate smoothing option was programmed off.

Patient No. 2

A 62-year-old female was admitted to the hospital because of symptomatic third-degree AV block. The ECG on admission showed complete heart block. The spontaneous sinus rate was 90 beats/min, and the ventricular rate was 40 beats/min. The QRS duration was 0.16 s; the morphology of the QRS complex suggested a ventricular origin of the secondary pacemaker. The patient did not use cardioactive medication prior to the

Figure 1. Irregular pacemaker-mediated tachycardia during ambulatory monitoring (continuous tracing). **A.** After the first two sinus beats, an APB is sensed before the atrial synchronization window. Due to the programmed RSV (3%), ventricular pacing with 1:1 VA conduction is initiated. **B.** The ventricular pacing rate gradually increases as long as 1:1 VA conduction remains present. **C.** At a ventricular cycle length of 520 ms, VA conduction is no longer sustained and because no P-wave occurs before or during the atrial synchronization window, atrial pacing is initiated at a gradually decreasing rate. **D.** When spontaneous P-waves occur during the atrial synchronization window, atrial pacing is inhibited.

onset of conduction disorder. Electrophysiologic study showed complete anterograde infranodal block. Retrograde conduction was blocked at the level of the AV node. A CPI 925 pacemaker was implanted. A PVARP was programmed at 300 ms, the AV delay period to 200 ms, and the RSV to 3%.

Monitoring by telemetry during 48 hours after implantation revealed loss of AV synchrony, which disappeared after switching the rate smoothing option off. The patient was transferred to the electrophysiology laboratory and a tem-

porary bipolar lead was inserted via the femoral vein and advanced into the right ventricular apex. Programmed single ventricular extrastimulation was performed during DDD pacing.⁴ Figure 2 shows the ECG when the rate smoothing option was programmed off. Figure 3 shows the ECG when an RSV of 3% was programmed and a premature ventricular beat was introduced during DDD pacing, leading to loss of AV synchrony as long as the spontaneous sinus rate remained unchanged. Figure 4 shows that AV synchrony was not lost when an RSV of 6% was

Figure 2. Complete compensatory pause after a sensed VPB. When the rate smoothing option is programmed off, no loss of AV synchrony is noted after a sensed VPB. The pacemaker was programmed in the DDD mode, minimum rate 50 beats/min, AV delay period of 200 ms, PVARP of 300 ms.

programmed. Figure 5 demonstrates that when an RSV of 12.5% was programmed AV synchrony was immediately restored. In this patient, an RSV of 12.5% was programmed. Holter recording performed after hospital discharge demonstrated AV synchronous pacing. So far her course (6 months after implantation) has been uneventful.

Patient No. 3

An 81-year-old female was admitted to the cardiology department because of dizziness. The ECG on admission showed sinus rhythm at a rate of 65 beats/min, 3:1 AV conduction, a QRS duration of 0.12 s, with RBBB and LAHB morphology.

Electrophysiologic study demonstrated advanced second-degree AV block. The site of anterograde block was infra-His; the AH interval was 80 ms and the HV interval of the anterogradely conducted beats (3:1) was 90 ms. During ventricular pacing, retrograde conduction was intact up to 120 beats/min. Retrograde conduction time, during sustained retrograde conduction, ranged from 280 to 300 ms. A CPI 925 pacemaker was implanted. The RSV was programmed to 6%, the AV delay period was 200 ms, and the PVARP was programmed at 350 ms. After pacemaker

implantation, the patient was monitored by telemetry and loss of AV synchrony was noted. Analysis of the ECG (Fig. 6) demonstrated loss of AV synchrony at a spontaneous sinus rate of 70 beats/min, and a paced ventricular rate of ± 60 beats/min. When the AV delay period was extended beyond the programmed maximum value, retrograde conduction occurred. The rate smoothing option was programmed off and AV synchronous pacing was restored.

Discussion

Although dual chamber pacing has corrected some of the original deficiencies of single chamber ventricular pacing, other problems have been created. In this report, some aspects of the electrocardiographic complexity of rate smoothing in patients with heart block are presented.

The problem of irregular pacemaker-mediated tachycardia, which we encountered in the first patient, was a result of the incidence of APBs, an insufficiently long PVARP, and the programmed RSV. Although this problem could easily be solved by prolongation of the PVARP, loss of AV synchrony could still occur as a consequence of the rate smoothing option. In the other two patients, we found that loss of AV synchrony

RSV 3%
PVARP 300

Figure 3. Programmed single ventricular extrastimulation during DDD pacing with an RSV of 3%. **A.** Due to the rate smoothing option, the compensatory pause after a ventricular extrastimulus is incomplete. AV sequential pacing is initiated at a gradually decreasing rate. During AV sequential pacing, the spontaneous sinus cycle is reset by the atrial paced beats. When P-waves occur during or before the atrial synchronization window, AV synchronous pacing is not restored because the RSV does not allow a reduction of the ventricular cycle length > 3%. **B.** When P-waves march into the PVARP, AV sequential pacing is resumed. As long as the spontaneous sinus rate remains unchanged AV dissociation remains present. Tracings A and B are continuous. Note that the first beat of tracing B shows poor atrial pacing.

could occur in patients with intact retrograde conduction as well as in patients with retrograde block. We observed that loss of AV synchrony in our patients was initiated by premature beats and that the endurance of AV dissociation was related to the programmed smoothing value, the programmed PVARP, and the spontaneous sinus rate.

Why does AV dissociation occur when low RSVs are programmed? Normally the interval of

the sinus beats that surround a VPB equals two times the PP interval (Fig. 2). However, when the rate smoothing option is used, the compensatory pause after a VPB is always less than complete (preceding RR interval - RSV) and AV sequential pacing or atrial pacing is initiated (Figs. 3, 4, 5). Since every following AV sequential paced cycle equals the preceding cycle plus the programmed RSV, the pacing rate gradually decreases. Initially, sinus P-waves may collide or

RSV 6%

PVARP 300

Figure 4. Programmed single ventricular extrastimulation during DDD pacing with an RSV of 6%. Before P-waves march into the PVARP, AV synchronous pacing is restored.

may be reset by the atrial paced beats during AV sequential pacing. However, because the AV sequential pacing rate decreases and the sinus rate remains unchanged, sinus P-wave will become manifest during or before the atrial synchronization window and AV sequential pacing is discontinued. Unfortunately, AV synchronous pac-

ing is not immediately restored, because the ventricular paced rate can only increase by decreasing every cycle with the programmed percentage. It is obvious that the duration of AV sequential pacing after a sensed VPB is dependent on the programmed RSV. When high RSVs (12.5%, 25%) are programmed, fewer AV sequentially

RSV 12.5%

Figure 5. Programmed single ventricular extrastimulation during DDD pacing with an RSV of 12.5%. After one cycle of AV sequential pacing, AV synchronous pacing is restored.

Figure 6. Intact retrograde conduction and rate smoothing. In this patient an RSV of 6% and a PVARP of 350 ms were programmed. Although the prolonged PVARP prevents pacemaker-mediated tachycardia, loss of AV synchrony is present. QRS complexes 1, 4 are ventricular paced beats, QRS complexes 2, 6 are anterogradely conducted beats, QRS complexes 3, 7 are ventricular paced beats with intact retrograde conduction, and QRS complexes 5, 8 show ventricular pseudofusion.

paced cycles are needed before sinus P-waves become manifest during or before the atrial synchronization window than when low RSVs (3%, 6%) are programmed. Subsequently, fewer cycles are needed to restore AV synchronous pacing when high RSVs instead of low RSVs are programmed. Figures 3, 4, and 5 illustrate that when a low RSV is programmed, restoration of AV synchronous pacing may fail because spontaneous P-waves may occur during the PVARP before AV synchronous pacing is restored. As a result, AV sequential pacing is initiated, leading to persistent AV dissociation as long as the spontaneous sinus rate remains unchanged.

We are not yet decided as to whether the rate

smoothing option is useful for clinical application. For patients with intact retrograde conduction as well as patients without retrograde conduction the risk of losing AV synchrony is present due to the rate smoothing option, especially in patients in whom low RSVs are programmed in combination with prolonged PVARPs.

In conclusion, the rate smoothing option available in a new DDD pacemaker might be of clinical importance because it eliminates large cycle-to-cycle variations in rate. However, irregular pacemaker-mediated tachycardias and loss of AV synchrony may occur. Fortunately, the pacing system enabled us to solve these problems noninvasively.

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Atrial Capture and Dual Chamber Pacing

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VAN MECHELEN, R., ET AL.: Atrial capture and dual chamber pacing. During dual chamber pacing it is sometimes impossible to assess atrial capture even on the 12-lead ECG. We developed a strategy to identify atrial capture when it is not possible to do so by ECG, and when the ECG shows no evidence of spontaneous or paced atrial activity. (PACE, Vol. 9, January-February, 1986)

atrial capture, AV sequential pacing, dual chamber pacing

Introduction

The diagnosis of successful atrial capture during dual chamber pacing may be quite difficult on the conventional ECG, especially in patients with unipolar pacemakers.^{1,2} In patients with intact AV conduction atrial capture can be demonstrated by programming a ventricular output below threshold, or by selecting an AV delay interval that exceeds the natural AV conduction time, or by programming to the atrial demand mode. However, in patients with complete heart block this approach is not appropriate.

In selected patients the bedside physical examination has been proposed as a simple and accurate means of identifying atrial capture. The presence of regular A and V waves in the jugular venous pulse and the presence of a stable intensity of the first heart sound and regular diastolic filling sounds all establish atrioventricular synchrony. In contrast, cannon waves, varying loudness of the first heart sound, and intermittent diastolic filling sounds suggest AV dissociation. Definitive proof of atrial capture can subsequently be obtained by recording the jugular venous pulse, the phonocardiogram, the echocardiogram, or the esophageal electrocardiogram.³⁻⁴ However, patient variability may be an important factor, and application of these noninvasive techniques may sometimes be very time consuming. Therefore, we questioned the suitability of this approach in the out-patient clinic when, during a pacemaker check-up visit,

the ECG does not allow assessment of adequate atrial pacing. For the out-patient setting, it would be desirable to have a simple and rapid technique to assess atrial capture.⁵ In this paper we present a strategy to demonstrate atrial pacing in patients with dual chamber pacemakers. At present this approach is routinely used in our out-patient clinic when it is unclear on the surface ECG whether or not the atrial stimulus is followed by atrial depolarization.

Methods

The flow chart to assess atrial capture is illustrated in Figure 1. When a single ECG lead does not show any evidence of spontaneous or paced atrial activity (Fig. 2), a 12-lead ECG is recorded. Special attention must be paid to leads II and VI, because these leads will most likely demonstrate atrial activation (Fig. 3). When no spontaneous or paced atrial activity is observed, the pacemaker is programmed to the VVI mode at the slowest available stimulation rate and spontaneous P waves may become visible (Fig. 4).

PP Interval Shorter than the VV Interval

When the VV interval exceeds the measured PP interval (Fig. 4) the pulse generator is programmed to the AV sequential (DVI) pacing mode⁶ at the same rate as the VVI pacing rate. When there is poor atrial pacing the spontaneous PP interval will not be affected by the atrial stimulus artifacts of the pacemaker. However, when adequate atrial pacing is present, the atrial stimuli will interfere with the spontaneous sinus cycle in the same way as atrial premature beats will interfere during sinus rhythm (Fig. 5), with resetting, interpolation, and collision of the spontaneous sinus beats.

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Figure 1. Assessment of atrial capture. AC = atrial capture; P = sinus P wave; AF = atrial fibrillation; AFL = atrial flutter; AS = atrial standstill; RC = retrograde conduction; PP = PP interval; VV = VV interval; A = atropine; VVI = VVI pacing mode; DVI = AV sequential pacing mode.

Figure 2. Unipolar AV sequential pacing (ECG lead III). The large unipolar atrial stimulus with its characteristic overshoot makes the diagnosis of successful atrial capture difficult.

Figure 3. Atrial capture during unipolar AV sequential pacing. Although lead III of the ECG does not show any evidence of successful atrial pacing, lead II of the 12-lead ECG demonstrates atrial capture.

PP Interval Longer than VV Interval

When the spontaneous sinus rate is slower than the programmed VVI pacing rate, sinus P waves will be absent during adequate AV sequential pacing as a result of override suppression of the sinus node. This means that when spontaneous P waves still are present after programming to the DVI pacing mode, the atrium is not captured by the pulse generator.

PP Interval Equals the VV Interval

When the PP interval equals the VV interval, the absence of P waves during DVI pacing at the same rate does not exclude poor atrial pacing, because dissociated P waves may be buried in the QRS complexes of the ventricular paced beats. In this setting, atropine 0.5–2.0 mg is administered intravenously to speed up the spontaneous sinus

rate, and atrial capture can be demonstrated by programming the DVI pacing mode to a rate slower than the sinus rate.

When P waves do not become visible after programming to the minimum VVI pacing rate, atrial fibrillation, atrial flutter, or, more rarely, atrial standstill may be present. However, an isoelectric baseline between the ventricular paced beats, does not, per se, mean that P waves are absent. Dissociated sinus P waves may be buried in the ventricular paced complexes or retrograde P waves may be hidden in the ST segments of the ventricular paced beats. Also, in this setting, atropine (0.5–2.0 mg) is administered to increase the underlying sinus rate in order to create a PP interval that is shorter than the ventricular paced VV interval. Subsequently, successful atrial pacing can be demonstrated by programming the AV sequential pacing mode at a slower rate than the spontaneous sinus rate.

Figure 4. VVI programming. At a rate of 70 ppm (upper tracing) it is not clear if P waves are present. At a VVI rate of 50 ppm (lower tracing) the PP interval equals 0.8 s; the ventricular paced interval is 1.2 s.

Results

During a 2-year period 87 dual chamber (DDD) pacemakers were implanted in our hospital. All patients were followed in our outpatient clinic at intervals of 3 months. In 13 patients the ECG during pacemaker follow-up did not allow assessment of adequate atrial pacing. All 13 patients had symptomatic second- or third-degree AV block. In eight patients our technique demonstrated atrial capture, which was confirmed by echocardiography. In three patients atrial fibrillation was manifest after programming the pulse generator to the minimum VVI pacing rate. In two patients inadequate atrial pacing could be demonstrated. Atrial standstill was not observed in any of our patients.

Discussion

On the conventional ECG the atrial stimulus, in particular the unipolar stimulus, may sometimes make a diagnosis of successful atrial capture

quite difficult. We developed a strategy to obtain rapid and definitive proof of appropriate atrial pacing by means of reprogramming the pulse generator and recording the ECG. Our technique is based on the electrophysiologic principles of overdrive suppression of the sinus node, and on the responses of the sinus node to premature atrial stimulation.

A limitation of our approach is that in patients with atrio-sinus entrance block no resetting of the sinus cycle will be present during adequate AV sequential pacing at a slower rate than the spontaneous sinus rate. However, the prevalence of atrio-sinus entrance block during premature atrial stimulation has been reported to be less than 7% in patients with sinus node dysfunction and may be extremely rare in patients with normal sinus node function.⁷⁻⁹

In contrast to echocardiography, venous pulse tracings, and esophageal electrocardiography, the electrocardiographic assessment of atrial capture

Figure 5. DVI pacing. Although paced P waves cannot be seen during DVI pacing (upper tracing), resetting of the sinus pacemaker demonstrates successful atrial capture. In this particular case atrial capture could clearly be proven by recording lead II of the ECG (lower tracing).

is independent of patient variability, and is available in any out-patient clinic. In addition, this approach is less time consuming than other noninvasive techniques. Therefore, we believe that this

simple technique may be applied during pacemaker check-up visits, when definitive and rapid proof of successful atrial pacing is desired.

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Failure to Sense P Waves During DDD Pacing

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VAN MECHELEN, R., ET AL.: Failure to sense P waves during DDD pacing. ECG tracings of three patients in whom AV universal (DDD) pacemakers were implanted intermittently demonstrated dropped P waves. In one patient, true atrial undersensing was present; in the others, sensing of the atrial electrode was appropriate, but sensing of sinus P waves was intermittently blocked by normal pacemaker operation. In this report we discuss the electrocardiographic diagnosis of atrial undersensing in order to avoid unnecessary reinterventions. (PACE, Vol. 9, July-August, 1986)

atrial malsensing, AV universal (DDD) pacing, atrial undersensing

Introduction

Undersensing as an expression of single chamber demand pacemaker malfunction (AAI or VVI unit) is manifest when proper pacemaker inhibition fails intermittently or continuously in association with reversion to fixed rate pacing for one or more timing cycles. True single chamber undersensing is present when a ventricular or atrial depolarization occurring during the alert part of the pacemaker escape interval is not detected by the sensing electrode, or when pacemaker malfunction is present.¹ Likewise, true undersensing of sinus P waves during DDD pacing is present when P waves are not sensed by the atrial electrode although they occur during the alert part of the atrial escape interval or when malfunction of the atrial lead, the atrial connector, or atrial sensing circuitry is present. In this report we discuss ECG tracings of three patients with DDD pacemakers in whom undersensing of sinus P waves was presumed.

Case Reports

Patient No. 1

An 87-year-old man was admitted to the CCU because of dyspnea and fainting spells. The patient

had been in good health until three days prior to admission.

Physical examination showed the patient to be a well-developed man in no obvious distress. The arterial blood pressure was 160/80 mmHg. The radial pulse was 30 beats/min. There was no neck vein distention. The lungs were clear to percussion and auscultation. The heart sounds were dull, with changing loudness of S₁. The liver was not palpable. There was no peripheral edema and the peripheral arterial pulses were normal.

The ECG showed complete heart block with a sinus rhythm of 70 beats/min, a ventricular rate of 30 beats/min, a QRS duration of 0.12 s, a RBBB morphology with left axis deviation; the QT interval was 0.48 s. Electrophysiologic study demonstrated complete infranodal block. During incremental ventricular pacing retrograde conduction was blocked below the AV node. A DDD pacemaker (Cordis* 418) was implanted and the patient was monitored by telemetry for 48 hours. The patient was discharged from the hospital the fourth day after admission.

A 24-hour Holter recording, performed three weeks after hospital discharge, intermittently demonstrated poor sensing of sinus P waves (Fig. 1). Interpretation of the ECG tracing confirmed a diagnosis of true atrial undersensing. When P waves were not sensed, they occurred during the alert part of the programmed atrial escape interval. Chest x-ray showed a displaced atrial lead on the right anterior oblique projection. The next day the

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Figure 1. Holter tracing (2-channel) of patient no 1. The pulse generator is programmed to a minimum rate of 50 beats/min, an AV interval of 200 ms, and a maximum rate of 120 beats/min. The atrial escape interval is 1000 ms (1200–200). The atrial refractory interval is 300 ms (500–200). Two sinus P waves (marked with an arrow) do not elicit a ventricular response from the pulse generator, although they occur during the alert part of the atrial escape interval.

atrial lead was repositioned, and atrial sensing was restored.

Patient No. 2

A 61-year-old woman was admitted to the CCU because she lost consciousness while she was cleaning her kitchen. She had never been hospitalized before.

On physical examination, blood pressure was 180/90 mmHg. The radial pulse was 45 beats/min. Central venous pressure was normal. The lungs were clear. The apical impulse was palpated in the 5th intercostal space in the midclavicular line. The heart sounds S1 and S2 were loud. A systolic murmur was audible at the aortic area. The murmur radiated towards the carotid arteries. The liver was not palpable. The arterial pulsations were normal and there was no peripheral edema.

The ECG showed complete heart block, a sinus rate of 70/min, a ventricular rate of 40/min,

a QRS duration of 0.08 s, and a QT interval of 0.38 s. Electrophysiologic study showed complete heart block. The anterograde block was localized in the common His bundle, whereas retrograde conduction was blocked below the level of the AV node. A DDD pacemaker (Cordis 415 A) was implanted. The patient was monitored by telemetry for 48 hours and thereafter discharged from the hospital. For the first six weeks after hospital discharge, a 24-hour Holter recording was performed routinely.

Figure 2 shows four representative ECG tracings of the Holter tape. Although atrial malsensing was presumed, interpretation of the ECG revealed adequate atrial sensing. Figure 2A shows a sinus P wave that is not followed by a ventricular stimulus. When this P wave would have been dropped because of atrial undersensing, an atrial stimulus would have been emitted after completion of the atrial escape interval. Because no atrial stimulus artifact was produced 950 ms after the preceding ventricular complex, this P wave could not be

Figure 2. Holter tracings of patient no 2. A. A sinus P wave (asterisk) does not elicit a ventricular response from the pulse generator. However, after completion of the atrial escape interval (950 ms) no atrial stimulation occurs. B. Every second P wave is blocked due to the 2:1 AV block response. C. AV synchronous pacing at a rate of 105 beats/min. D. An atrial premature beat occurs during the atrial refractory interval (arrow), and after completion of the atrial escape interval atrial stimulation follows. Minimum rate = 50 beats/min; AV interval = 250 ms; maximum rate = 110 beats/min; atrial refractory interval = 300 ms (550–250); atrial escape interval = 950 ms (1200–250).

dropped due to atrial undersensing. The most likely explanation for this pacemaker behavior is T wave sensing by the ventricular electrode, which resets the timing cycle and reinitiates the atrial refractory interval during which the sinus P wave occurred. Alternatively, electromagnetic or myographic interference may also be detected by the ventricular electrode, producing the same phenomenon. However, it is very unlikely that this interference would occur for only one timing cycle.

Figure 2B shows 2:1 AV block when the spontaneous sinus rate exceeded the programmed up-

per rate (110 beats/min). Figure 2C shows AV synchronous pacing at a sinus rate of 105 beats/min. Figure 2D shows a normal pacemaker response to a premature atrial beat which occurred during the atrial refractory interval. After completion of the atrial escape interval, an atrial stimulus artifact was produced. In this patient, the ventricular refractory interval was prolonged to prevent T wave sensing; in addition, ventricular sensitivity was programmed from 1.3 mV to 2.5 mV. The atrial refractory interval and the AV interval were shortened in order to avoid the 2:1 AV block response. A 24-hour Holter recording was

repeated after reprogramming of the pulse generator and no loss of AV synchrony was noted.

Patient No. 3

An 85-year-old woman underwent a DVI pacemaker implantation five years prior to admission because of symptomatic second degree AV block. She was admitted to the CCU because of output failure of the pacemaker.

On physical examination, her blood pressure was 130/70 mmHg. Radial pulse rate was 50 beats/min. Neck veins were not distended. The lungs were clear and heart sounds were normal. The liver was not palpable. There was pitting edema on both ankles. Arterial pulsations were normal.

The ECG on admission showed AV sequential pacing at a rate of 50 beats/min, whereas the minimum rate of the pacemaker was programmed to 70 beats/min. The ECG during pacemaker replacement showed sinus rhythm, 80 beat/min 2:1 AV block, RBBB morphology of the QRS complex, a QRS duration of 0.12 s, an intermediate axis, and a QT interval of 0.44 s.

During ventricular stimulation at a rate slightly faster than the spontaneous sinus rate, AV dissociation was present. A DDD pulse generator (Cordis 415 A) was connected with the already implanted atrial and ventricular leads. The amplitude of the atrial signal was 4 mV. After pacemaker implantation, the patient was monitored by telemetry and a monitor tracing showed atrial undersensing (Fig. 3).

A 12-lead ECG was recorded; Figure 4 shows two representative tracings of lead I. When the

spontaneous sinus rate exceeded the programmed upper rate of the pulse generator, 2:1 AV block occurred; in addition, the subsequent sinus P wave did not generate a ventricular response of the pacemaker. After a blocked P wave, due to the 2:1 AV block response, neither a sinus P wave (upper tracing) nor a ventricular premature beat (lower tracing) elicited an appropriate response of the pulse generator. The most likely explanation for this pacemaker behavior is that when a P wave occurs during the terminal portion of the atrial refractory interval, this P wave might be interpreted by the pulse generator as noise.² The response of this pulse generator to noise is that no sensing occurs either in the atrium or in the ventricle during the complete atrial escape interval. In this patient, atrial sensing was appropriate. In order to avoid the 2:1 AV block response, the atrial refractory interval and AV interval were shortened. Also the sensitivity setting of the atrial channel was changed from 0.5 mV to 2.5 mV, which lowered the risk that P waves would be recognized as noise by the pulse generator when they occurred during the terminal part of the atrial refractory interval.²⁻⁴ A 24-hour Holter recording performed after hospital discharge showed normal AV synchronous pacing.

Discussion

The variety and complexity of dual chamber pacemakers have introduced complex and intricate diagnostic problems that may be related to benign functional variations or to serious malfunction of the pacing system. In this report we

Figure 3. Telemetry tracing of patient no. 3. After exceeding the programmed upper rate of the pulse generator, 2:1 AV block occurs. The sinus P wave (asterisk) that follows a blocked P wave is not sensed. Minimum rate = 50 beats/min; AV interval = 200 ms; maximum rate = 120 beats/min; atrial escape interval = 1000 ms (1200-200); atrial refractory interval = 300 ms (500-200).

Figure 4. ECG lead I of patient no 3. (Top) After exceeding the programmed upper rate of the pulse generator 2:1 AV block occurs; again, the sinus P wave (asterisk) following a blocked sinus P wave is not sensed as was shown in Figure 3. (Bottom) A ventricular premature beat (arrow) is not sensed when it occurs during the presumed alert interval of the ventricular escape interval. Ventricular refractory interval = 300 ms.

present three patients with dropped P waves during DDD pacing. The pacemaker of the first patient showed failure to sense sinus P waves during the alert part of the atrial escape interval (Fig. 1) which is an example of atrial undersensing. The second patient showed appropriate atrial sensing, but when P waves occurred shortly after a T wave sensed by the ventricular channel, atrial channel refractoriness was reinitiated. The third patient also showed appropriate atrial sensing but dem-

onstrated the so-called noise mode response³ when the atrial rate reached the programmed upper rate of the pulse generator.

In conclusion differentiation between true and pseudo-failure to sense P waves during DDD pacing has important therapeutic consequences in terms of surgical reintervention. In the presented cases a correct diagnosis was made noninvasively. Full understanding of normal pacemaker operation was essential to interpret the ECGs.

References

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SUMMARY

The clinical work-up of sinus node dysfunction and atrio-ventricular (AV) block is described in Chapter I.

In Chapter II to VIII some electrophysiologic and electrocardiographic aspects of conduction disorders related to pacemaker therapy are discussed. The prevalence of retrograde AV conduction in patients with sinus node dysfunction was studied and the results of this study are presented in Chapter II. Especially patients with intact anterograde AV conduction demonstrated intact retrograde AV conduction. Ventricular demand pacing, in patients with symptomatic sinus node dysfunction, is therefore not the ideal pacemaker therapy. Atrial pacing is an alternative pacing mode for patients with intact anterograde AV conduction, which is haemodynamically superior to ventricular demand pacing. In Chapter III the reliability of permanent atrial pacing is presented during long term follow-up. It appeared that permanent atrial pacing was safe, even in patients who used antiarrhythmic drugs that adversely affected AV nodal function.

The introduction of dual chamber (DDD) pacemakers offered patients with symptomatic AV block an alternative for ventricular demand pacing. As was already discussed in Chapter I, DDD pacemakers have solved some of the original deficiencies of single chamber ventricular pacing, however other problems have been created. One of these problems is pacemaker mediated tachycardia, a subject which is addressed in Chapter IV.

Chapter V reports on the stability of retrograde AV conduction of patients with symptomatic heart block, after DDD pacemaker implantation. During long term follow-up, retrograde AV conduction remained stable in the majority of patients. In addition, antiarrhythmic drug therapy adversely affected anterograde as well as retrograde AV conduction. These findings were of clinical importance with regard to the prevention of pacemaker mediated tachycardia.

Chapter VI, VII and VIII focus on electrocardiographic aspects of DDD pacing. In Chapter VI, some unfamiliar ECG patterns are discussed as a result of an algorithm in a dual chamber pulse generator that prevents large fluctuations in pacing rate.

In Chapter VII an electrocardiographic technique is presented to assess atrial capture during AV sequential pacing. The value of the ECG to assess atrial malsensing during AV synchronous pacing is described in Chapter VIII.

SAMENVATTING

In Hoofdstuk I wordt de klinische evaluatie besproken van patiënten, met een gestoorde sinus knoop functie of een gestoorde atrio-ventriculaire (AV) geleiding.

In de Hoofdstukken II t/m VIII komen enkele electrofysiologische en electrocardiografische aspecten van geleidingsstoornissen in relatie tot pacemaker therapie aan de orde.

Hoofdstuk II handelt over de prevalentie van teruggeleiding naar de boezem tijdens hartkamer stimulatie bij patiënten met een gestoorde sinus knoop functie. Vooral bij patiënten met intacte AV geleiding bestaat tevens intacte teruggeleiding naar de boezem. Dit maakt permanente kamerstimulatie niet tot de ideale stimulatie vorm voor patiënten met een gestoorde sinus knoop functie. Boezemstimulatie is een alternatieve stimulatie vorm voor patiënten met een gestoorde sinus knoop functie, waarbij de AV geleiding intact is. Deze stimulatie vorm is bovendien in haemodynamisch opzicht superieur aan kamerstimulatie.

In Hoofdstuk III wordt de betrouwbaarheid van boezemstimulatie besproken. Tijdens een prospectief follow-up onderzoek bleek boezemstimulatie veilig te zijn. Symptomatisch AV blok trad bij geen der patiënten op, zelfs niet bij patiënten die behandeld werden met anti-aritmica die een remmende invloed uitoefenen op de AV knoop.

De introductie van DDD pacemakers, die boezem-kamer synchronie kunnen nabootsen, boden patiënten met een symptomatisch hartblok een alternatief voor pacemakers, die uitsluitend de hartkamer stimuleren. De voordelen van een dergelijke pacemaker kwamen reeds in Hoofdstuk I ter sprake. Hoofdstuk IV is gewijd aan een van de problemen van DDD pacemakers: artificiële pacemaker tachycardie.

Hoofdstuk V gaat over de stabiliteit van teruggeleiding naar de boezem bij patiënten met AV blok. Tijdens een vervolgstudie na DDD pacemaker

implantatie, werd op noninvasieve wijze vastgesteld, dat teruggeleiding stabiel blijft, bij de meerderheid van patiënten. Bovendien bleek, dat anti-aritmica zowel anterograde als retrograde AV geleiding kunnen blokkeren. Deze gegevens zijn van klinisch belang, met betrekking tot het voorkomen van pacemaker cirkeltachycardie.

Hoofdstuk VI, VII en VIII handelen over electrocardiografische aspecten van DDD pacemakers. In Hoofdstuk VI worden enkele merkwaardige ECG registraties besproken, die het gevolg zijn van een pacemaker algoritme, dat tot doel heeft om plotselinge veranderingen in stimulatie frequentie tegen te gaan.

Hoofdstuk VII gaat over een electrocardiografische techniek om vast te stellen of de boezem wordt gedepolariseerd tijdens AV sequentiële hartstimulatie.

In Hoofdstuk VIII wordt de betekenis van het ECG besproken, om aan te tonen of DDD pacemakers atriale signalen adequaat of inadequaet detecteren.

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