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Pacemaker & Tachycardia

Proefschrift

Ter verkrijging van de graad van doctor
in de Geneeskunde
aan de Rijksuniversiteit Limburg te Maastricht,
op gezag van de Rector Magnificus Prof. Dr. H.C. Hemker,
volgens besluit van het College van Dekanen
in het openbaar te verdedigen in de aula van de universiteit
op 22 november 1984
des namiddags te vier uur.

door

Karel den Dulk geboren te Pretoria.

Heerlen 1984
Drukkerij Schrijen-Lippertz BV-Voerendaal

Promotor Prof. Dr. H.J.J. Wellens.

Referenten Dr. M.A. Allessie.
Prof. Dr. H.E. Kulbertus.
Prof. Dr. E.O. Robles de Medina.

Het verschijnen van dit proefschrift werd mogelijk gemaakt door steun van de Nederlandse Hartstichting en Rescar (stichting ter bevordering research cardiologie) Maastricht.

To my parents.

To Jennie, Kirsty and Yolanda.

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Chapter 1

Introduction

1.1 Pacemaker History (1,2)

Pacemaker history as presented here comes mostly from a book entitled "Exploring the Origins of Electrical Cardiac Stimulation" by Schechter (1) and from a chapter entitled "History of Cardiac Pacing" written by the late Dr Thalen (2).

a) Early History

As was suggested by Schechter in his extensive review on pacemaker history (1), the application of electricity in medicine was probably prompted by observing the electrical effect of certain fish. In 1615 Godinko noted that when a live torpedo ray was dropped on a pile of dead fish, the latter convulsed as if revivified. Galvani, the great Italian investigator, did many experiments with electrical stimulation on living tissue, including frogs' hearts. In 1791 he apposed a live torpedo ray to the nerves, muscles and hearts of dead frogs. Convulsive contractions occurred but disappeared after withdrawal of the fish.

Vassali and colleagues, working in Turin, Italy, received permission to experiment on the bodies of decapitated criminals immediately after execution. Using Volta's early battery, the "Voltaic pile", they stimulated the heart and produced strong contractions. These studies were described by Aldini in 1804.

The main impetus to the study of electrophysiology came from the empirical employment of electrostatic machines and Leyden jar type capacitors for therapeutic purposes. The visible reaction of muscles to electrical discharges made the use of electricity inevitable for neuromuscular diseases. Simultaneously, physiologists studied muscle function by examining the effects of electricity.

Electricity was first used successfully for human resuscitation in 1774. This was recorded in the Annals of the Royal Humane Society, London. Interest in electrical stimulation of all parts of the body increased during the mid-1800's.

Prior to the development of pacemakers, patients having a very slow heart rate due to complete atrioventricular block, had a very poor life expectancy. In patients with heart block without other serious underlying

cardiac disease, restoration of a normal heart rate by means of a pacemaker could return the patients' life expectancy to almost normal.

The feasibility of rectifying a too fast or too slow heart rate by electricity was first demonstrated clinically by Duchenne in 1869. "These were the principal morbid signs which I discerned: extremely pale face, discolored lips, slight coolness of the nose and limbs; precordial oppression with stridor; mildly panting respiration but with normal breathing rhythm, without diaphragmatic paralysis, emphysema, or rales; smallness and extreme rapidity of the pulse (136 to 140 pulsations), with such irregularity and intermittences that often six or eight successive pulsations missed; impossible to distinguish the rhythm of valvular sounds on auscultation of the heart; all I could hear were a succession of muted, unequal, disorganized small noises; no temperature rise heralding a febrile state....".

"It was after having thoroughly weighed all of those considerations that I practised cutaneous faradization, by means of the "electric hand", on the precordial region, principally over the apex of the heart, taking care to start with a very feeble dose. Then I gradually increased the intensity of the induction current, of which the intermittences were rapid, until a mild pricking was sensed by the skin. We were gratified to see our anticipations achieved; for after a few minutes and during passage of the current, the pulse diminished in frequency, became regular and formed; finally the precordial oppression and suffocation vanished. Several times during the day I had to reinstitute precordial cutaneous faradization, so as to definitely chase the diptheritic perturbation of cardiac innervation which displayed some tendency to remit."

During a remission he once used a very intense current" .... I was undertaking to reverse them, by the method of faradization which hitherto had proved successful in that patient, when the apparatus was moved and its graduator tube left completely out without my being forewarned. The resultant vivid excitation was followed by faint syncope and momentary cardiac arrest. However, cutaneous faradization of the precordial region which I promptly conduced made me master again over these nervous disorders."

Many great physicians such as Jean Charcot and Frantisek Chvostek applied current to the neck and precordium of patients to curb tachycardias associated with hyperthyroidism. Interest in the reduction of heart rate by giving extra stimuli began in 1885 with the work of Langendorff.
In 1915 De Boer described the halving of the ventricular rate by means of induction shocks. The first clinical application of paired electrical

stimulation was undertaken by Francis Dammann and his associates in 1954.

In 1850 Ludwig and Hoffa showed that strong currents to the heart caused it to assume peculiar uncoordinated movements resulting in circulatory collapse. "Herz-delirium," "fibrillar contraction," "tremulations fibrillaires," "delirium cordis," or "intervermiform movements" as ventricular fibrillation became variously known. In 1887 Mac Williams advanced the startling theory that it was fibrillation which probably precipitated the strange deaths sometimes occurring after anesthesia. The increasing incidence of fatalities from accidental electrocution provided the real incentive for thoroughly examining the matter of ventricular fibrillation.

Prevost and Batelli were the first to demonstrate beyond doubt at the end of the 19th century that fibrillatory contractions induced by electricity could often be abolished by another powerful discharge of either alternating or direct current. It is of interest that a resuscitation apparatus designed in 1788 by Charles Kite has features that would make it suitable for ventricular defibrillation. It consisted of an electrostatic generator, a Leyden jar (for storing electrical charge-a capacitor-), connecting chains, and glass-insulated electrodes. It took two centuries before an implantable defibrillator became available (3).

b) Recent History

In the early 1930's Hyman developed a machine for ambulance use (Fig 1). It was powered by a clock spring (wound by a crank every six minutes). It produced pulses instead of galvanic current as used earlier. The pacing pulses were fed into the heart through a bipolar electrode needle that could be put through the chest into the heart. This enabled Hyman to save the lives of at least two patients for 24-48 hours in 1932. He also coined the term artificial pacemaker and was the first to clearly state the principle of pacing: that a small electrical stimulus, applied to a very small area, can give rise to a wave of contraction which will then spread through the entire heart muscle, giving a relatively normal contraction. He tried to replace the spring motor that had to be rewound every six minutes by a battery-powered unit and was negotiating for a special battery with the German Siemens company. Unfortunately the last Hyman unit was destroyed in the Siemens factory during the bombing of Dresden in the Second World War.

Early clinical work in pacing was done by Zoll, in 1952. He used skin electrodes on the patients' chest, transmitting electrical stimuli of sufficient strength to cause the heart to contract.

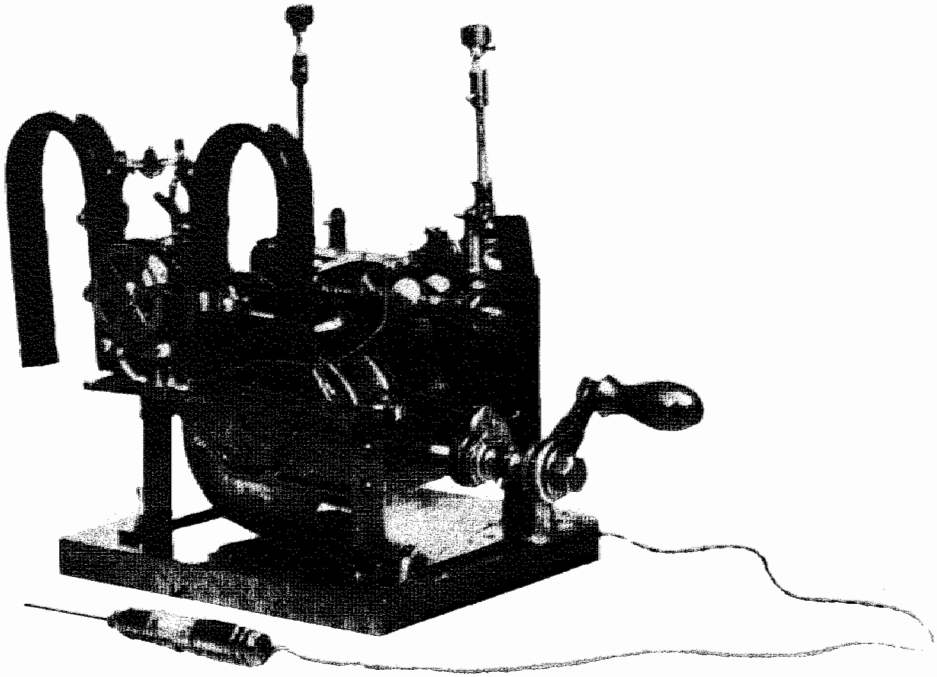


Figure 1.
Artificial pacemaker developed by Hyman in 1932. (Provided by Siemens Ltd, The Netherlands).

In 1958 Furman (4,5) developed a technique for long term stimulation of the heart by passing an endocardial catheter electrode into the right ventricle. The external pacemakers used at that time were large vacuum-tube electronic pulse generators, which were placed on a bedside table. The patient could take short walks by using a long extension cord and a rolling table. The first clinically usable portable external pacemaker was developed by Bakken in 1958, in cooperation with Weirich and Lillehei, from the University of Minnesota. By using transistors this unit was made small and independant of 110-volt power outlets.

In Stockholm, Elmqvist and Senning were the first to develop a small implantable pacemaker by using the newly-developed transistors (6-8). On 8th October 1958, they implanted their first unit in a patient, Arne H.W. Larsson, who is still alive to-day (6). Nickel-cadmium batteries were used with an energy of 60 mA that could be recharged inductively. In 1960 Chardack and Greatbatch developed a pacemaker which used mercury-zinc batteries (9). These 12-gram, 3-cm3 batteries produced about 1.3 volts. Depending upon the pacemaker type, 3-5 batteries were used to create pacemaker life times of initially 2-3 years.

Significant improvements have been made by the use of solid state lithium batteries, which were introduced in 1971 by Greatbatch (10). The application of integrated circuits is the most recent improvement and this has enabled the construction of smaller, more complicated, and more efficient pacemakers. In some cases the circuit consumes only ten percent of the energy that was necessary in the early designs. These improvements in generator design have resulted in a decrease of pacemaker volume from about 60-70 cm^3 to 30-35 cm^3 and of pacemaker weight from about 130-140 to 40-45 grams (Fig 2).

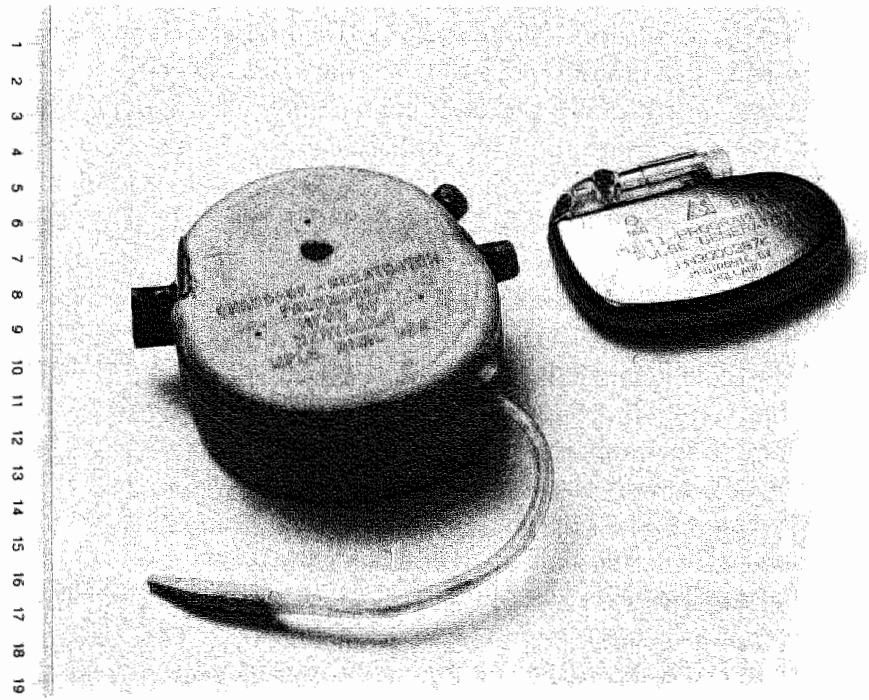


Figure 2.
Photo illustrating the reduction in pacemaker size in the past 24 years. (Provided by Medtronic BV, The Netherlands).

Pacemaker programming technology started in 1962, when Kantrowitz reported on the first programmable pacemaker implant (11).
The first unit that had the ability to have more than one parameter altered was developed by Medtronic in 1963 (12). Altering one of the two adjustable parameters, rate and output, required transcutaneous insertion of a surgical Keith needle into one of two self-sealing ports in the pacemaker enclosure. Several programming systems have been developed since. The

systems are of two basic types: those employing a pulsed magnetic field to communicate between programmer and pacemaker, and those employing a radiofrequency link. Through the use of microprocessor technology, digital codes and radiofrequency transmission, pacemaker programmers can now relay information to the implanted pacemaker and, through telemetry, also receive and process feedback from the implanted pacemaker.

The pacing modes of the pulse generators kept up with the technical improvements. The initial units could not sense the cardiac activity and operated independently thereof (asynchronous fixed rate pacemakers). These early fixed rate devices (9,13) required a minimum of components and were insensitive to interference from external electrical fields. This was followed by the development of non-competitive devices in 1965 which could detect naturally occurring cardiac activity (15). Spontaneous cardiac activity is sensed by the pacing electrode and amplified within the pacemaker. This amplified signal resets the timing circuit. Two types of response were developed: a sensed event immediately triggers an output pulse that reaches the myocardium when the heart is still unresponsive to stimuli (VVT*) and a sensed event inhibits a pacing impulse (VVI). In the presence of ventricular activity slower than the escape rate of the pacemaker, this system functions identically to a fixed rate pacemaker. Should the patients intrinsic heart rate exceed the pacemaker escape rate then no pacemaker activity will occur (VVI), or an output pulse is immediately triggered when the heart is unresponsive to stimuli (VVT).

The next development was the "physiological pacemaker", which allows automatic rate control and/or maintains ventricular filling by the atrial contraction to increase the cardiac output and thus derive an increase in functional capacity. Such designs are the VAT, VDD, AAI, DVI and the DDD pacemakers. It is interesting to note that the first application of physiological pacing was the VAT or P-wave synchronous pacemaker in 1962 (16) before the non-competitive devices. In this system spontaneous atrial activity is sensed by the atrial electrode. This signal triggers the pacemaker to produce an output pulse which is transmitted to the ventricles via the ventricular electrode after a suitable atrioventricular delay. In this way near-physiological atrioventricular activation can occur during sinus rhythm and the pacing rate can be increased according to physiological

* The 3-position pacemaker code (14) proposed by the Intersociety Commission for Heart Disease is used in this thesis to describe the mode of pacing (See table I).

Table I

Position	1	2	3
Category	Chamber(s) paced	Chamber(s) sensed	mode of response
Letters used	V-Ventricle A-Atrium	V-Ventricle A-Atrium	T-Triggered I-Inhibited
	D-Double S-Single[1]	D-Double S-Single[1] O-None	D-Double[2] O-None R-Reverse[3]

1 S in position 1 and 2 will be used by manufacturers only, and indicates that the device can be used in atrium or ventricle.

2 D in position 3 stands for atrial triggered and ventricular inhibited.

3 R in position 3 describes a system that starts pacing at high spontaneous rates.

demands (reflected in the sinus rate). The VAT pacemaker has no ventricular amplifier. Therefore, premature ventricular complexes are not sensed and the pacemaker may deliver a stimulus in the ventricle at an unfavourable moment.

To overcome this potential problem, the VDD or atrial synchronous ventricular inhibited pacemaker was developed. Currently the most advanced pacing mode is the fully automatic or DDD mode. During the abscence of atrial and ventricular activity it stimulates both chambers in sequence to preserve atrioventricular synchrony. It can track sinus rhythm like the VAT or VDD pacemaker, maintaining A-V synchrony and allowing a rate increase. It can also be inhibited by ventricular events. Moreover, in patients with contra-indications for DDD pacing, non-invasive multimode programmability is available and reprogramming to the DVI or VVI pacing mode is possible. The physiological advantage of DDD pacing is that it maintains AV synchrony and also allows for rate increase.

Accumulating evidence shows that patients derive an increase in functional capacity when pacemakers allow sinus rate variation and preservation of A-V synchrony (17-19). The best candidates for implantation of a DDD pacemaker are those, with high degree A-V block, who are physically active, not limited by other handicaps and have a normally functioning sinus node. Contra-indications for the implantation of a DDD pacemaker are the presence of chronic atrial fibrillation. Possible

contra-indications might be extremely inactive or bedridden patients, patients who have paroxysmal atrial flutter, atrial tachycardia or sick sinus syndrome.

Pacemakers can also be used for the treatment of certain types of tachycardia.

1.2 Mechanisms of Tachycardia

Animal experiments and clinical studies have resulted in the identification of three mechanisms of tachycardia which seem to be of clinical importance:

a) abnormal automaticity b) triggered activity (delayed after depolarisations) and c) re-entry.

a) In abnormal automaticity, impulse formation is based upon the spontaneous discharge of cardiac cells. Pacemaker cells are normally found in the sinus node, the atrioventricular node, and the purkinje system. Other cardiac cells may discharge spontaneously under abnormal conditions such as ischemia, sympathetic stimulation, electrolyte changes, and stretch. When rapid impulse formation is based on abnormal automaticity, the arrhythmia can at best be suppressed only temporarily by pacing.

b) Triggered activity (20) is based on the ability of the membrane action potential of certain cardiac cells to develop delayed afterpotentials able to reach threshold, thereby leading to ectopic impulse formation. Though generally the role of triggered activity in the genesis of tachycardias in the human heart seems to be small, evidence is increasing that under certain circumstances, for example during epinephrine administration or digitalis intoxication, this mechanism may be operating and be the reason for abnormal impulse formation (21). The significance of this mechanism as a cause of recurrent tachycardias in the human heart is not clear. Therefore, the role of pacing in the treatment of such arrhythmias is unknown at the present time.

c) Re-entry: Tachycardias based on a re-entry mechanism are thought to be most suited for pacing therapy. The concept of a circulating impulse was first described and studied by Mayer (22) in his experiments with jellyfish. "If we cut off the marginal sense-organs of the scyphomedusa Cassiopea, the disk becomes paralyzed and does not pulsate in sea-water. The disk will pulsate in sea-water, however, if we make either a single ring or a series of concentric broken-ring-like cuts through the muscular tissue of the sub-umbrella. Then upon momentarily stimulating the disk in any manner, it

suddenly springs into rapid, rhythmical pulsation so regular and sustained as to recall the movement of clockwork. Pulsation will not start unless the disk be momentarily stimulated, as by a mechanical or electrical shock or by a single touch with a crystal of K_2SO_4, but once started it continues indefinitely in normal sea-water without further external stimulation. The waves of pulsation all arise from the stimulated point, and the labyrinth of sub-umbrella tissue around this center must form a closed circuit."

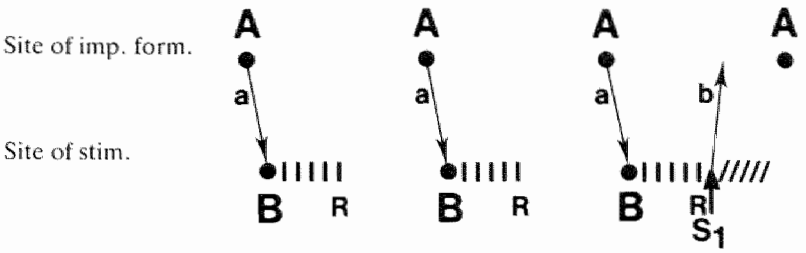
A number of other important observations were made by Mayer (22).

1) He realized that the initial circulating impulse must travel in only one direction around the closed circuit from the point of its initiation, so that block in the other direction must exist at the time of impulse initiation. Mayer cut the subumbrella tissue into a ring, stimulated the ring at one point and noted that if two waves of contraction of equal magnitude start at the same point and progress in opposite directions around the ring, only one contraction of the ring occurs. However, by applying pressure near the site of stimulation a strong contraction could be induced to progress in one direction around the ring, the area of compression preventing a strong wave from progressing in the other direction. The wave conducting in one direction returned to its point of origin and then conducted around the ring again. Once started this continues indefinitely in normal sea-water without further external stimulation.
2) He recognized the importance of the relationship between path length, conduction velocity, and the refractory period. "The wave will maintain itself indefinitely provided the circuit be long enough to permit each and every point in it to remain at rest for a certain period of time before the return of the wave through the circuit."
3) "The point which was stimulated and from which the contraction wave first arises is of no more importance in maintaining rhythmical movement than any other point in the ring". He demonstrated this by cutting away the tissue around the site at which the stimulus was applied.
4) To prove that the impulse was circulating around the ring causing it to pulsate, he showed that it stopped instantly when the circuit was interrupted.

Mines (23) made decisive contributions to the development of the concept that re-entry can cause cardiac arrhythmias. In 1913 he described a "reciprocating rhythm" which he had occasionally observed in his experiments on the auricle-ventricle preparation of the heart of the electric ray and in one experiment on the ventricle bulbus preparation from the frog. The rhythm could be initiated and terminated by appropriately timed

stimuli. "The rhythm of each chamber was directly dependant on that of the other". He postulated;" let us start with the auricular excitation. This is transmitted over the slowly conducting tissue to the ventricle. But immediately after, the excitation of the ventricle is transmitted back to the auricle. It is unlikely that the actual tissue which transmitted the impulse from auricle to ventricle could immediately transmit another impulse in the reverse direction. Therefore in all probability the portion of the auriculo-ventricular connexion responsible for the transmission from ventricle to auricle was quiescent during the previous transmission from auricle to ventricle. "..."A slight difference in the rate of recovery of two divisions of the A-V connexion might determine that an extrasystole of the ventricle, provoked by a stimulus applied to the ventricle shortly after activity of the A-V connexion, should spread up to the auricle by that part of the A-V connexion having the quicker recovery process and not by the other part. In such a case, when the auricle became excited by this impulse, the other portion of the A-V connexion would be ready to take up the transmission again back to the ventricle. Provided the transmission in each direction was slow, the chamber at either end would be ready to respond (its refractory phase being short) and thus the condition once established would tend to continue, unless upset by the interpolation of a premature systole."..."I venture to suggest that a circulating excitation of this type may be responsible for some cases of paroxysmal tachycardia as observed clinically."

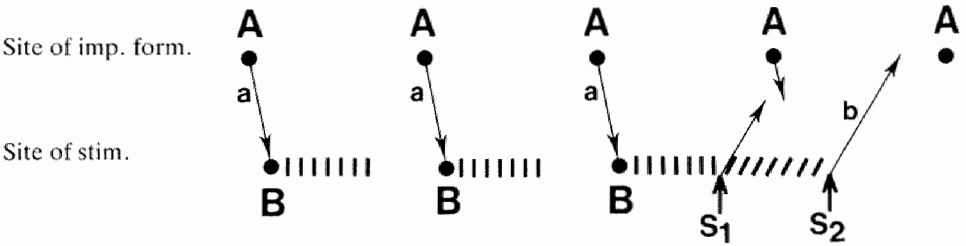
The circuit used by a circulating impulse can be anatomically (as reported by Mines) or functionally determined as described by Allessie et al (24). When the circuit is anatomically defined, as during a circus movement tachycardia in the Wolff-Parkinson-White syndrome, an excitable gap is usually present between the head and the tail of the circulating wavefront. The tissue constituting this excitable gap is not refractory and can be depolarized. The width of the excitable gap depends on the duration of the refractory period within the circuit, the length of the circuit and the conduction velocity of the circulating impulse. In the presence of an excitable gap, a correctly timed impulse from outside the circuit is theoretically able to invade and create bidirectional block in the circuit, thereby terminating the arrhythmia. Certain conditions have to be fulfilled before a single paced impulse will be able to enter a re-entrant circuit. These are illustrated in figure 3. Figure 4 shows that entrance into the circuit is facilitated by a second paced impulse, which is closely coupled to the first one.



A A = circuit time
BR = refract. period at site B
a = conduction time from A to B
b = conduction time from B to A

Arrival in circuit before next impulse (A) by one premature beat (S_1) only possible when the A-A time is longer than the sum of a + BR + b.

Figure 3.
Diagram illustrating the factors which determine entrance into the re-entry circuit by a single premature impulse from outside the re-entry circuit. (From Wellens HJJ, den Dulk K, Brugada P: Pacemaker management of cardiac arrhythmias. In: Dreifus LS, Brest AN eds: Pacemaker Therapy. Philadelphia, FA Davis Company, 1983, P 165).



Arrival in circuit before next impulse (A) of a second premature beat (S_2) only possible when the A-A time is longer than the sum of the refractory period of S_1 + b.

Figure 4.
Diagram showing how a second premature beat facilitates entrance into a re-entry circuit. (From Wellens HJJ, den Dulk K, Brugada P: Pacemaker management of cardiac arrhythmias. In : Dreifus LS, Brest AN eds: Pacemaker Therapy. Philadelphia, FA Davis Company, 1983, P 165).

Therefore, the factors which play an important role in the ability to terminate re-entrant tachycardias by pacing are; 1) tachycardia rate 2) the duration of the refractory period of the components of the re-entry circuit, and the tissue between the re-entry circuit and the pacing site, 3) the distance between the tachycardia circuit and the site of stimulation, 4) the conduction velocity in the circuit and in the tissue between the re-entry circuit and the pacing site (25).

Allessie et al (24) were able to demonstrate in their experiments on circus movement tachycardia in rabbit atrial muscle that in a small piece of atrial myocardium the induction of a properly timed premature beat could force the impulse to conduct in a circuitous route and thus initiate circus movement tachycardia. They were the first to devise a model of circus movement tachycardia that is applicable to circulating excitation in the absence of a nonexcitable central obstacle. Fig 5 shows a comparison of the anatomically (Mines) and functionally (Allessie) determined circus movement tachycardia (24).

1.3 Programmed Electrical Stimulation of the Heart

In 1963, 50 years after the experiments by Mines, Moe (26) renewed interest in the use of electrical stimulation of the heart for the study of tachycardias by describing intranodal tachycardias in dogs which could be terminated by single or double extrastimuli. The ability to record endocardial signals from strategically placed electrodes (introduced via the venous system) and the development of a safe and dependable stimulation technique led to the introduction of programmed stimulation of the heart to study arrhythmias in the intact human heart. The use of programmed electrical stimulation of the heart in the study of patients suffering from tachycardia was independently reported in 1967 by Coumel et al (27) and Durrer et al (28). This was followed by several papers on the electrical initiation and termination of different types of tachycardia (29-33).

In 1971 Wellens reviewed the use of programmed stimulation of the heart, in patients with atrial flutter, AV nodal tachycardias and circus movement tachycardia using accessory pathways (25). In 1972 he introduced the technique in patients suffering from ventricular tachycardia (32). A critical analysis of the value and limitations of programmed stimulation of the heart in determining the mechanism of tachycardia and the selection of treatment was published in 1978 (34).

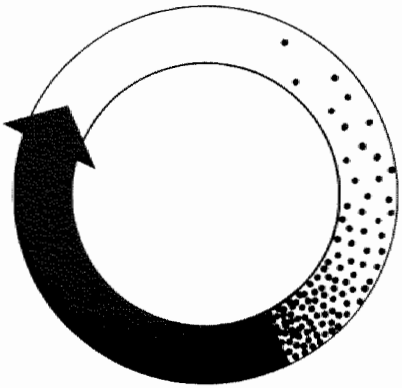
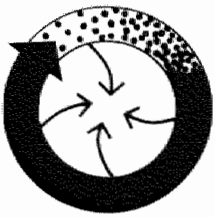
Circus movement around anatomic obstacle (Minus 1913	Circus movement without anatomic obstacle (Leading circle model)
	
1. Length of circular pathway determined by perimeter of anatomic obstacle.	1. Length of circuit determined by conduction velocity, stimulating efficacy, and refractory period.
2. Length of circular pathway fixed.	2. Length of the circuit can change with alterations in electrophysiologic properties.
3. Excitable gap between crest and tail of the impulse (white part of circuit).	3. No gap of full excitability.
4. Impulse can not short-cut the circuit.	4. Short-cut of the circuit possible.
5. Revolution time inversely related to conduction velocity.	5. Revolution time proportional to refractory period.

Figure 5.
Comparison of the two types of re-entry mechanism.
(Provided by Dr. M.A. Allessie. From Allessie MA, Bonke FIM, Schopman FJG: Circus movement in rabbit atrial muscle as a mechanism of tachycardia. III The "leading circle" concept. Circ Res, 41:9,1977).

1.4 Pacing for Termination of Tachycardia

Re-entry tachycardias can be terminated by appropriately timed stimuli, which invade the circuit and create refractoriness ahead of the circulating impulse. The importance of the site of stimulation, and the timing of the extrastimuli have been extensively demonstrated (32,34,35). Pacing as a chronic form of therapy for the interruption of re-entry tachycardias is gaining more and more interest and acceptance.

Various pacing techniques with implantable devices are currently in use or under investigation.

Underdrive Pacing: This refers to asynchronous stimulation at a rate slower than the tachycardia rate. The pacing impulses occur at various times during the tachycardia cycle, as long as the tachycardia rate is not an exact multiple of the pacing rate (36). As described by Ryan (37) in 1968, underdrive pacing can be achieved with a demand pacemaker by placing a magnet over the pacemaker and converting it to an asynchronous magnet mode. Automatic uni- and bifocal (A-V sequential with a shortened A-V interval) underdrive pacemakers are available. The effectiveness of A-V sequential dual demand automatic pacemakers has been demonstrated in selected patients (38,39). Application of unifocal underdrive pacing is limited to tachycardias with rates ‹160 bpm. Rates above 160 are rarely terminated by one premature beat unless an accessory pathway is incorporated in the circuit and the pacing catheter is placed close to the re-entry circuit (35).

Overdrive or Burst Pacing: During overdrive pacing the heart is stimulated at a rate above the tachycardia rate. Addition of a second stimulus facilitates entrance into the re-entrant circuit from a distant stimulation site by "peeling back" the refractory period at the site of stimulation and the intervening tissue (26,40). By giving more than 2 premature stimuli, the refractory period of the tissue at the stimulation site and the intervening tissue can be further shortened allowing even earlier arrival of the stimuli at the site of the re-entry circuit. This method can be used in patients with atrial flutter, A-V nodal tachycardia, circus movement tachycardia using an accessory pathway, and carefully selected cases of ventricular tachycardia. Before implanting such a device for use in the atrium, one must exclude the existence of an accessory pathway with a short anterograde refractory period.

Burst pacing is not without risk if applied to the ventricle because rate

acceleration and fibrillation can be induced in patients with ventricular tachycardias. The risk of inducing ventricular fibrillation may be reduced by late synchronization of the first beat of the burst. The pacing intervals can be determined in milliseconds or as a percentage of the tachycardia cycle length (adaptive/orthorhythmic) to compensate for a change in tachycardia rate (41).

Programmed Extrastimuli: This technique uses exact and independent timing of one or more extrastimuli to penetrate the re-entrant circuit. The coupling interval can be in milliseconds or a percentage of the tachycardia cycle length. If tachycardia has not been terminated after a first attempt, the coupling intervals or number of stimuli of subsequent treatments may be the same or may be changed. This form of pacing has been developed because number and timing of required stimuli may change with changes in posture, autonomic tone, tachycardia rate, refractory period, drug level or for other, unknown reasons.

Several pacing methods are presently available to adjust to such changes:

1) sequential incremental or decremental scanning in which the coupling interval is changed by a small value each time until the interval to be scanned has been covered or tachycardia has been terminated (42,43). Scanning can be done with the first or the last stimulus or with both if 2 stimuli are given. Currently, most commercial systems have only 2 stimuli available. Other scanning techniques have been or are currently under clinical investigation. These include the so called self-searching stimulation, centrifugal geometric scanning, concertina pacing and entrainment with premature stimulation.
2) high frequency stimulation (train) (44,45) in which a short burst of pacing is delivered with a very high rate. The train is started during the refractory period and timed to give 1 or 2 captures (directly after the refractory period) with the shortest possible coupling intervals regardless of the tachycardia rate. An inherent risk is the induction of acceleration or fibrillation;
3) automatic increase of the number of stimuli one at a time until termination of tachycardia occurs or to a predetermined number of stimuli, if termination is not achieved;
4) the adaptive mode where the coupling interval of the paced stimuli is a predetermined percentage of the tachycardia cycle length.

All these pacing techniques are available in an implantable form for long-term therapy. The three types of implantable devices are:

1) a fully automatic one (power supply and circuitry implanted) and two types

of patient-activated systems;
2) one with the power supply implanted; and
3) one with the power supplied by an external electromagnetically coupled unit. There are three types of automatic pacing systems available today. They are the dual-demand (unifocal or A-V sequential (38,39,46), the scanning (42,43), and the burst pacemaker (47,48). Of these, the scanning pacemaker does not yet have a back-up pacing mode and can give a maximum of 2 stimuli.

The less sophisticated externally activated systems have a one-way communication. The patient recognizes tachycardia and activates the system to deliver a treatment which usually consists of an asynchronous burst of high-rate pacing. The more sophisticated systems allow two-way communication and synchronization of stimulation.

The electromagnetically coupled system as described by Critelli (49,50) has the advantage that the implanted unit is small and weighs only 33 grams (power supply not implanted). However, this system is limited by being able to give only 2 stimuli and by not having a back-up pacing mode because no energy supply is implanted. The more sophisticated patient-activated systems with bidirectional communication have the general advantage of greater versatility.

1.5 Pacemaker Circus Movement Tachycardia

As has been previously mentioned, pacemakers which pace and sense in atrium and ventricle and which synchronize atrial and ventricular activity have been developed recently (DDD). These so called physiological pacemakers have however introduced a new artificial re-entry tachycardia (36,51-53).

In these patients the atrioventricular conduction system (or an accessory atrioventricular pathway) supplies the retrograde arm of the re-entry circuit by conducting the ventricular events retrogradely to the atrium. The pulse generator supplies the artificial anterograde bypass tract by synchronizing ventricular paced events to the sensed retrogradely conducted atrial events.

These artificial circus movement tachycardias (ACMTs) or pacemaker circus movement tachycardias (PCMTs) resemble re-entry tachycardias in several ways.

1) They are initiated by critically timed atrial or ventricular premature beats
2) They can be terminated by critically timed atrial or ventricular premature beats, carotid sinus massage (like in supraventricular re-entry tachycardias)

or medication.

3) Perpetuation and/or initiation of a re-entry tachycardia depends upon conduction velocity and refractoriness of the different parts of the circuit. The artificially anterogradely synchronizing bypass tract complies to these two properties as well. The conduction velocity is determined by the programmed A-V interval and the maximum atrial tracking rate programmed. The refractoriness is determined by the atrial refractory period of the device (fixed or programmed). Similar to re-entrant tachycardia, these tachycardias can also be prevented by influencing the refractory period or conduction velocity of the A-V or V-A connections. By programming the pacemaker the conduction velocity and refractory period of the anterograde artificial pathway can be changed. The retrograde arm of the re-entry circuit can be influenced by medication or vagal manoeuvres.

Ventriculoatrial conduction, which is essential for these PCMTs to occur, is not uncommon as was shown by Schuilenburg (54). The presence of ventriculoatrial conduction need not preclude the use of DDD pacing. Rather, as shown in this thesis, V-A conduction should be tested invasively before implantation by programmed stimulation and noninvasively at follow-up to ensure that PCMTs are controlled. Most PCMTs can be prevented by proper programming of the pacemaker. Occasionally the addition of medication is required.

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Chapter 2

A Versatile Pacemaker System for Termination of Tachycardias

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Externally activated pacemaker systems were implanted in 13 patients to control their drug-resistant tachycardias. Four patients had ventricular tachycardia, 2 had atrioventricular nodal reentrant tachycardia, 3 had tachycardias due to a left-sided concealed accessory pathway, and 4 had Wolff-Parkinson-White syndrome. Nine patients were paced from the right ventricle, 2 from the right atrium and 2 from the coronary sinus. The pacing system consisted of an implantable pacemaker, an external pacemaker activator and a prescription formulator. The pacemaker can signal sensing by way of radiofrequency signals to the pacemaker activator or prescription formulator. Either 1 of the latter 2 devices then determines whether the sensed rhythm fulfills the tachycardia detection criteria and, if so, controls the delivery of the selected stimulation treatment by the pacemaker. With this bidirectional radiofrequency coupling, tachycardias were noninvasively initiated by the prescription formulator after implantation and at follow-up visits to test and eventually reprogram the pacemaker activator. During a follow-up of 116 patient-months, 624 episodes of tachycardia were effectively terminated by the patients. Incidental failure to terminate occurred in 3 patients because of a defective activator, changes in the electrophysiologic substrate and inappropriate use of the device. These problems were solved by reprogramming, replacement of the activator and education of the patient. Hospital admissions for termination of tachycardia decreased from an average of 2.6 per patient-month (in the 3 months before implantation) to 0.03 per patient-month after implantation (follow-up 4 to 16 months). It is concluded that (1) this programmable externally activated pacemaker system effectively manages drug-resistant tachycardia; (2) this system has the advantage of easy testing, multiple pacing modes and ready reprogrammability; and (3) the marked reduction in hospital admissions makes the system cost-effective.

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The use of cardiac pacing as a chronic form of therapy for the interruption of paroxysmal reentrant ventricular tachycardia (VT) and supraventricular tachycardia is gaining interest and acceptance. The importance of the site of stimulation and the correct timing of pacing stimuli have been extensively demonstrated.[1,2] Pacing may be the therapy of choice in patients who do not respond to or cannot tolerate drug therapy, patients who are not suited for or refuse surgery, or for whom elective surgery is not readily available, and patients who cannot tolerate prolonged episodes of tachycardia because of the development of cardiac failure, angina pectoris or syncope. Many patients may prefer treatment with small and reliable pacemakers over chronic intake of antiarrhythmic agents which may have adverse effects and are certainly not inexpensive. Fischer et al[3] and Barold et al[4] recently reviewed the pacing techniques being used or under investigation.

This report presents our experience with a patient-activated Medtronic (Medtronic BV) interactive tachycardia pacing system to terminate VT and supraventricular tachycardias.

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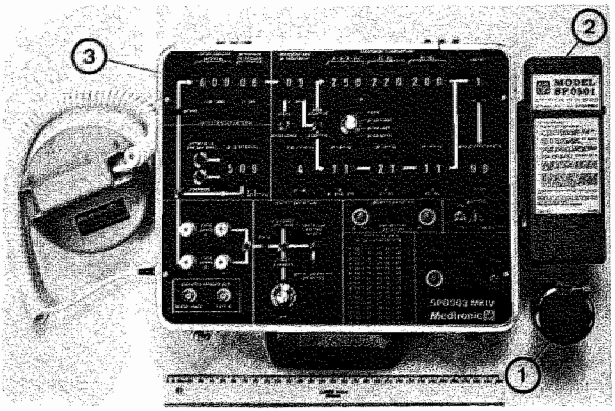

FIGURE 1. The 3 components of the Medtronic Interactive Tachy System. (1) The implantable pulse generator; (2) the hand-held pacemaker activator; and (3) the prescription formulator.

Methods

Patients: Thirteen patients (5 women, 8 men) had a Medtronic model SP0500 patient-activated pacemaker implanted for termination of their paroxysmal tachycardias. They were 18 to 61 years old (mean 46). Seven patients were from St. Annadal Hospital, Maastricht, The Netherlands, and 6 were from the Bavière Hospital, Liege, Belgium. In all patients, multiple long-term oral drug regimens failed to control their arrhythmias satisfactorily and they had been admitted many times to the hospital for conversion of arrhythmias. A complete electrophysiologic evaluation was performed in each patient to determine the mechanism of tachycardia and the most suitable stimulation site and mode to reproducibly terminate tachycardia. The pacing catheter was placed as close to the reentrant circuit as possible, using the pacing site that was most successful during electrophysiologic study. Atrial stimulation was avoided if atrial fibrillation or flutter was easily induced. Two patients were paced from the right atrium, 2 were paced from the coronary sinus and 9 from the right ventricle.

System description: The Medtronic Interactive Tachy System consists of 3 parts: (1) model SP0500 implantable pacemaker, (2) model SP0501 pacemaker activator, and (3) model SP0503 prescription formulator (Fig. 1). The pacemaker is a bipolar, atrial or ventricular inhibited device that weighs 82 g and is 56 mm in diameter. The nonprogrammable variables are: 50 pulses/min pacing rate, 5.6 V stimulus amplitude, 0.5 ms stimulus width (antibradypacing only), 375 ms refractory period and 0.75 mV sensitivity for 10 ms sine,[2] and 2.5 mV sensitivity for 40 ms sine[2] test wave form. (This sensitivity curve allows proper ventricular as well as atrial sensing.) During application of the magnet, (1) inhibited function continues, (2) the refractory period shortens to 190 ms (to allow sensing of fast rhythms), (3) a radiofrequency (RF) pulse is emitted at each sensed event, and (4) RF pulses of proper frequency content will trigger the pacemaker to deliver a stimulus, whose duration is controlled by these RF pulses. This magnet mode allows bidirectional RF communication between pacemaker and either pacemaker activator or prescription formulator. Although the implanted pacemaker is not programmable, antitachycardia pacing modes and other variables such as "tachytrigger interval," number of intervals to trigger, pulse duration, treatment repetition, delay between treatments and activator off-time can be easily modified after implantation by having the pacemaker activator reprogrammed by the manufacturer.

On recognition of tachycardia, the patient activates the pacemaker activator and places it over the pacemaker. The battery-powered activator contains a microcomputer, RF receiver and transmitter, and a magnet to initiate magnet mode. When the RF link is established, the microcomputer will determine whether the rhythm (transmitted by RF pulses) fulfills the programmed tachycardia detection criteria. If tachycardia is confirmed, the pacemaker activator delivers a preprogrammed sequence of RF pulses that are transformed into stimuli by the pacemaker. Proper sensing, stimulation and battery status are indicated by light-emitting diodes on the activator. If tachycardia is not confirmed, the activator switches off after 5 seconds. If stimulation sequences have not interrupted the tachycardia, the activator cannot be switched on again for a programmable off-time (0.5 to 34 minutes).

The prescription formulator (Fig. 1), which is an external stimulator with the same microcomputer and software as the pacemaker activator, was used after implantation to define the parameters defining tachycardia recognition, treatment and repetition. Tachycardia is recognized if the intervals between sensed depolarizations become shorter than the tachy trigger interval (selectable between 190 to 996 ms) for a programmable number of consecutive intervals (selectable between 1 to 99). On recognition of tachycardia, a treatment that may consist of 1 to 99 stimuli will be delivered (Fig. 2). The first stimulus, S_1, is synchronized to the last sensed event, R. The R-S_1 interval and S_1-S_2 interval can be individually selected, as can the remaining (S_2-S_n) intervals. These 3-interval values can be selected either in milliseconds (nonadaptive mode) or as a percentage of the last sensed tachycardia interval (adaptive mode). Interval values can be chosen between 12 and 996 ms or 3 and 97%. The last stimulus of the treatment activates a delay, selectable from 0 to 9 seconds, after which the tachycardia recognition system is again activated. If the tachycardia persists, the treatment is repeated until the maximum number of treatments (1 to 99) has been reached. The R-S_1 interval of the second treatment can be the same as before (interval "fixed") or it can be automatically shortened by 8 ms (interval "scan").

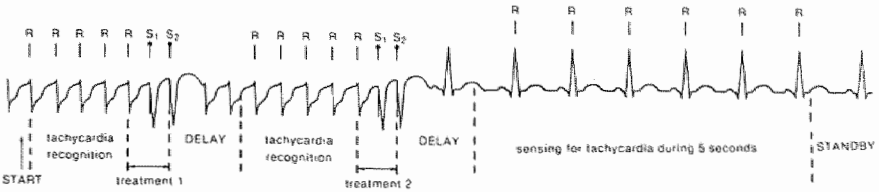

FIGURE 2. The sequence of tachycardia recognition and treatment. R = sensed QRS complex; S = stimulus.

This automatic interval scanning can also be applied to the last extrastimulus interval or to both the first and the last intervals if 2 extrastimuli are selected. This scanning continues until the maximal number of treatments or an interval shorter than 148 ms has been reached. The number of stimuli can be identical for all treatments or it can automatically increase from 1 to the selected "maximum number of pulses in treatment" (1 to 99) if the "increasing" mode was selected. Pulse width of the treatment stimuli can be chosen between 0.25 and 2.0 ms in steps of 0.25 ms. The prescription formulator can be directly connected to a temporary endocavitary lead or it can be coupled to an implanted model SP0500 pulse generator through the RF link. This requires positioning of a programmer head over the pacemaker. The last feature allows noninvasive electrophysiologic studies, induction of tachycardias and verification of safety and effectiveness of treatments after implantation. The prescription formulator also has an asynchronous stimulation capability with S-S intervals of 74 to 999 ms. Extrastimuli can be coupled to the asynchronous stimuli every N beat (N between 1 and 99). One

TABLE I Patient Data

Case	Age (yr) & Sex	Associated Cardiac Disease	Pacing Site	Antiarr. Therapy	Pacing Mode	Repro-gramming	Hospital Admissions: Before Impl. (3 mo)	Hospital Admissions: After Impl.	Follow-Up (mo)
				Ventricular tachycardia					
1	61M	PostMI	RV	+	Scan 2 VPB	TTI	4	0	9.5
2	59M	PostMI	RV	+	Scan 2 VPB	...	7	0	9.5
3	18M	Corrected tetralogy	RV	+	Fixed 3 VPB	...	9	0	4
4	35F		RV	0	Fixed 5 VPB	...	5	1	10
				AV nodal tachycardia					
5	52F	Parox. atrial fib.	CS	+	1–6 APB in adaptive mode	...	6	0	4
6	31F	...	RA	+	Scan 2 APB	...	9	1	9.5
				Concealed accessory pathway					
7	41M	...	RV	0	Scan 2 VPB	...	5	0	11
8	33M	...	RV	+	Fixed 2 VPB	Fixed 3 VPB	5	0	16
9	59M	CAD	RA	+	Scan 2 APB	Scan 3 APB	15	1	10
				Wolff-Parkinson-White syndrome					
10	48M	Amyloid DC	RV	+	Adaptive 4 VPB	...	18	0	4
11	48M	MI	CS	+	Fixed 1 APB	Fixed 2 APB	2	0	12
12	52F	...	RV	+	Adaptive 1 APB	TTI	8	1	6.5
13	60F	SSS	RV	+	Scan 3 VPB	Scan 2 VPB	7	0	10

Antiarr. = antiarrhythmic; APB = atrial premature beats; AV = atrioventricular; CAD = coronary artery disease; CS = coronary sinus; DC = dilated cardiomyopathy; fib. = fibrillation; impl. = implantation; MI = myocardial infarction; parox. = paroxysmal; RV = right ventricle; SSS = sick sinus syndrome; TTI = tachycardia trigger interval; VPB = ventricular premature beats; + = yes; 0 = no.

set of stimulation variables can be stored in the prescription formulator's memory to allow easy switching from tachycardia-initiating to tachycardia-interrupting stimulation sequences. The selected values for tachycardia detection, treatment, repetition measurements and pulse width are then programmed into the memory of the pacemaker activator microcomputer at the factory. Whenever necessary, the patient activator can be reprogrammed with a new treatment after selecting the new values with the help of the prescription formulator. Although the use of an RF link between pacemaker and activator reduces the risks of mistriggering due to interference/noise signals, the user should be aware of potentially dangerous situations such as the application of programmers other than the model SP0503 prescription formulator. Patients should be warned not to use their pacemaker activator in areas where strong sources of RF energy are present.

Procedure before discharge: In the 2 to 3 days after pacemaker implantation, tachycardias were repeatedly initiated and terminated with the prescription formulator. The maximal coupling intervals with the least number of stimuli able to terminate tachycardia reproducibly were used. The selected values for tachycardia detection and treatment, along with the selected pulse width, were then programmed into the memory of the pacemaker activator's microcomputer by the manufacturer. In the remaining days before patient discharge, tachycardias were repeatedly initiated by the prescription formulator and terminated by the pacemaker activator to test the safety and efficacy of the system and to familiarize the patient with its use.

Follow-up: During outpatient clinic visits, the number of episodes of tachycardia, the success rate of the system, complications and problems that had occurred in the interim were recorded. During each visit tachycardias were initiated several times with the prescription formulator and terminated with the pacemaker activator. If pacing treatment was inadequate, another pacing mode was tested using the prescription formulator. The pacemaker activator was then reprogrammed at the factory using the newly determined values.

Results

Table I is a summary of the data from our 13 patients who underwent pacemaker implantation.

Ventricular tachycardia: Patient 1 and 2 had VTs on the basis of an old myocardial infarction. On amiodarone, 200 mg/day, VT cycle lengths ranged from 450 to 475 ms. VTs were reproducibly terminated with 2 stimuli using the scanning mode. No acceleration of VT or degeneration into ventricular fibrillation has been seen in the respective 9.5-month follow-up periods. Patient 3 had VT that started 6 years after total correction of tetralogy of Fallot. Figure 3 illustrates termination of his VT by 3 consecutively given ventricular premature beats. It also illustrates the bradycardia support function. Patient 4 had an idiopathic VT. This patient showed different responses to identical pacing modes. Figures 4 and 5 illustrate that identical modes of pacing may lead to different modes of termination of tachycardia. Patients 3 and 4 have been followed up for 4 and 10 months, respectively, and no adverse effects have been noted.

In the 3 months before pacemaker implantation, Patients 1 to 4 were admitted many times for conversion of their VT (25 times in 12 patient-months). After pacemaker implantation, 1 patient was admitted to the hospital once over 33 patient-months with VT, because the pacemaker activator was not working. Three of the 4 patients are still on antiarrhythmic therapy. Only 1 of the 4 pacemaker activators were reprogrammed. The tachycardia trigger interval was changed after a change in medication in Patient 1.

Atrioventricular nodal reentrant tachycardia: Two female patients with atrioventricular (AV) nodal reentrant tachycardias received the model SP0500 pacemaker. In 1 patient the scanning mode with 2 stimuli from the right atrium terminated tachycardia reliably for 2 months. After this period the patient was admitted once with a sustained episode of tachycardia. After the addition of a beta-blocking agent to reduce the tachycardia rate, her tachycardias were once again promptly terminated at home by the pacemaker.

The other patient had both drug-resistant AV nodal

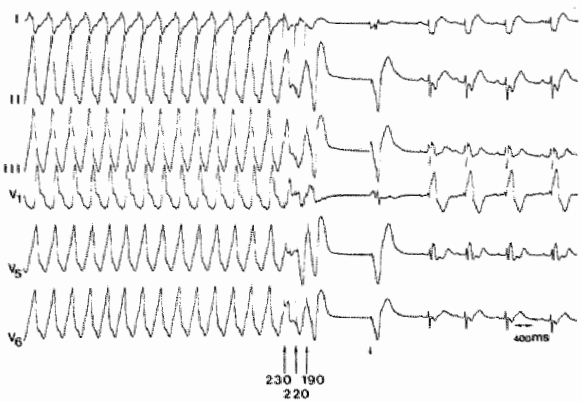

FIGURE 3. Six-channel electrocardiographic recording of termination of a ventricular tachycardia by 3 stimuli with fixed coupling intervals. After termination a bradycardia, back-up stimulus with an escape interval of 1,200 ms (50 pulses/min) can be observed.

reentrant tachycardias as well as paroxysmal atrial fibrillation. The AV nodal reentrant tachycardias could not be reliably terminated from the ventricle, but were easily terminated from the atrium. A coronary sinus position was chosen to get close to the AV nodal reentrant circuit. The drugs prescribed for this patient's atrial fibrillation lengthened the atrial refractory period, which prevented reproducible termination of tachycardia with the scanning mode. By choosing the adaptive mode (enabling adjustment to the changes in rate of tachycardia) and by giving an increasing number of stimuli, tachycardias were reproducibly terminated with the least number of stimuli (Fig. 6).

Both patients are receiving antiarrhythmic therapy. The patient activators did not have to be reprogrammed and only 1 patient has been admitted for ineffective

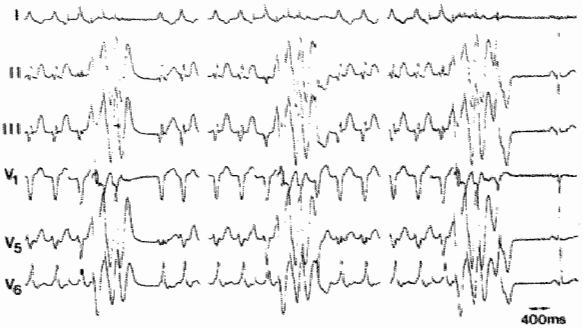

FIGURE 4. Three strips of different 6-channel electrocardiographic recordings showing the effects of a synchronized burst with identical coupling intervals in a patient with an idiopathic ventricular tachycardia. In all 3 strips the first stimulus did not capture the ventricle, while the second stimulus gave rise to a fusion complex. **Left-hand strip,** the tachycardia is reset. **Middle strip,** the ventricular premature beat after the paced QRS complexes probably reinitiates the tachycardia. **Right-hand strip,** the ventricular premature beat after the paced QRS complexes terminates the tachycardia.

FIGURE 5. Same patient as in Figure 4—3 strips of 6-channel electrocardiographic recordings illustrating 2 unsuccessful and 1 successful attempt to terminate an idiopathic ventricular tachycardia with a synchronized burst of 5 stimuli. Although the timing and number of stimuli is exactly the same, tachycardia is terminated in only 1 of 3 attempts. This observation suggests a search for a more effective mode of treatment.

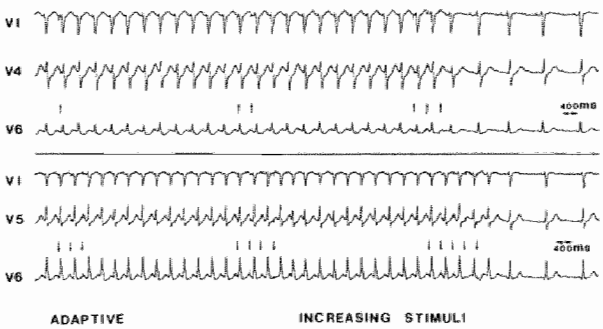

FIGURE 6. Two 3-channel electrocardiographic recordings of termination of 2 episodes of atrioventricular nodal reentrant tachycardia with different rates in the same patient. The adaptive mode with an increasing number of stimuli was used. **Top strip,** 3 stimuli from the coronary sinus with equal coupling intervals of 84% of the tachycardia cycle length were required for termination. **Bottom strip,** at a faster tachycardia rate 5 stimuli with shorter and equal coupling intervals (also 84% of the tachycardia cycle length) were needed for termination.

pacemaker termination of tachycardia in 13.5 months, compared with 15 admissions in 6 patient-months before pacemaker implantation.

Concealed accessory pathway. Three patients had left-sided concealed accessory pathways. In Patients 7 and 8, tachycardias were observed with narrow QRS complexes and with left and right bundle branch block. Because of the larger tachycardia circuit during left bundle branch block, the rate of the tachycardias was slower than that with a narrow QRS complex or right bundle branch block (Fig. 7). In Patient 7 the appearance of left bundle branch block after pacemaker implantation prevented a Medtronic Byrel dual-demand pacemaker from terminating tachycardia.[5] Not only did left bundle branch block cause changes in rate, but the tachycardia rate also fluctuated at rest and after slight exercise. The interval scanning mode with 2 stimuli resulted in reproducible termination of tachycardia. In 2 of the 3 patients, tachycardias were successfully controlled in the scanning mode using 2 stimuli; in the third patient 2 fixed stimuli were used. Patient 8 had very rapid tachycardias that were accompanied by lightheadedness and anxiety. He had 1 to 3 episodes of tachycardia per week. In the 13 months after implantation he has had 8 episodes of sustained tachycardia, during which the pacemaker activator had to be activated 2 to 4 times before successful termination. Since implantation, he has not been admitted to the hospital for termination of tachycardia. Of the patients with concealed accessory pathways, only 1 patient (patient 9) was admitted once in 37 patient-months with tachycardia, compared with 25 admissions in 9 patient-months before implantation. Patient 8 now requires 3 stimuli instead of 2 to reproducibly terminate tachycardia. Patients 8 and 9 are still receiving antiarrhythmic therapy.

Wolff-Parkinson-White syndrome: Four patients had the Wolff-Parkinson-White syndrome and in all patients the circus movement tachycardia made use of the AV node in the anterograde direction and the accessory pathway in the retrograde direction. Two patients had a short (<270 ms) and 2 a long anterograde refractory period of their accessory AV pathway. In 3 patients the accessory pathway was left-sided and in 1 it was right-sided. All 4 patients are receiving antiarrhythmic therapy. Patient 10 had amyloid congestive cardiomyopathy, frequent episodes of tachycardia (3 to 4/week) and was admitted to the hospital 18 times (often in cardiac failure) in the 3 months before implantation for termination of tachycardias. Patient 11 had 2 previous myocardial infarctions and coronary artery bypass grafting. He had infrequent episodes of tachycardia, which required hospital admission for termination. He can now terminate his tachycardia at home. When amiodarone was discontinued because of adverse effects, his tachycardia rate increased. He then required 2 stimuli instead of 1 from the coronary sinus to reproducibly terminate tachycardia. This change was made during an outpatient clinic visit. Patient 12 has been admitted once because of a sustained episode of tachycardia. She was not using her pacemaker activator correctly, and after reeducating her she could terminate her tachycardias at home. Because of slowing in the rate of tachycardia, after a change in medication, the tachycardia trigger interval had to be reprogrammed (on an outpatient basis). In Patient 13 a beta-blocking drug was given to slow down the tachycardia rate so that tachycardia could be reproducibly terminated with 2 stimuli with longer coupling intervals instead of 3 from the right ventricle. In the group of patients with Wolff-Parkinson-White syndrome there were 35 admissions in 12 patient-months before implantation and there was only 1 admission after pacemaker implantation. The latter admission was for termination of a tachycardia because the patient was not using the device properly (as previously mentioned). The total follow-up period was 32.5 patient-months.

Number of hospital admissions because of tachycardia before and after pacemaker implantation: Figure 8 indicates the number of admissions for termination of tachycardia for each patient in the 3 months before implantation. The mean number of admissions was 7.7 per patient (range 2 to 18). During a follow-up period of 4 to 16 months, only 4 patients were admitted once for termination of tachycardia. Therefore, the mean number of hospital admissions per pa-

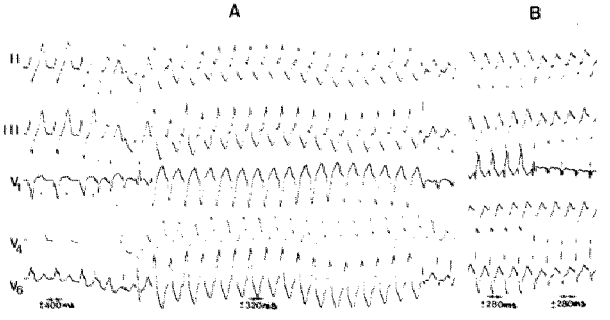

FIGURE 7. Initiation of an orthodromic circus movement tachycardia using the prescription formulator. Initiation is achieved by driving the ventricle at 120 pulses/min and delivering 2 ventricular premature beats. **Panel B** was recorded 1 minute after **panel A.** The tachycardia with left bundle branch block has a longer tachycardia cycle length than the tachycardia with right bundle branch block or without bundle branch block because the accessory pathway is left-sided.

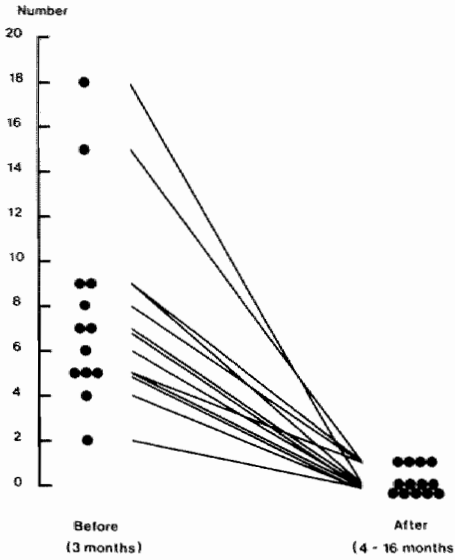

FIGURE 8. Left, number of admissions per patient for termination of tachycardia in the 3 months before pacemaker implantation. **Right-hand side** of the figure indicates that in a follow-up period of 4 to 16 months, only 4 patients had to be admitted once with tachycardia.

tient-month was reduced from 2.6 before to 0.03 after implantation. The reasons for admission were: (1) Defective pacemaker activator. (2) Increase in required number of stimuli. During the first 4 months of pacing, tachycardias were terminated with 2 stimuli. The patient was then admitted with tachycardia and, by means of the prescription formulator, was found to require a third stimulus to reproducibly terminate tachycardia. (3) Additional drug therapy. After a period of 2 to 3 months of successful termination, a beta-blocking drug was required to reproducibly terminate tachycardia without changing the pacing mode. (4) Inappropriate use of the pacing device. After reeducation, the patient had no further problems. In the first 3 instances the prescription formulator was used to noninvasively terminate the tachycardia and to determine the new most appropriate treatment.

Tachycardias at home: During a follow-up of 116 months, 627 episodes of tachycardia were reported; 612 were promptly terminated at home by the patient using the pacemaker activator. Twelve episodes needed a second, third or fourth attempt. In only 3 patients did the system fail to terminate tachycardia, for reasons already given. The fourth patient was not using the system properly.

Patient opinion: Twelve patients are very satisfied with the system. It has changed their lifestyle completely, avoiding repetitive hospital admissions. In only 1 patient has the clinical result been less than satisfactory. Initially his tachycardias were effectively terminated with 2 stimuli from the right ventricle. Thereafter, the rate of his tachycardia increased spontaneously to 260 beats/min and medication could not effectively reduce his tachycardia rate. He is now waiting for surgical sectioning of his accessory pathway.

Reprogramming of the pacemaker activator: In the follow-up of 4 to 16 months, 6 pacemaker activators were reprogrammed, 4 on an outpatient basis. In 4 patients reprogramming was required because of a change in medication. In 2 patients a third stimulus was added to reproducibly terminate tachycardia. Five of the 6 reprogrammings were done within the first 3 months after implantation. The sixth patient required a change in coupling intervals and a third stimulus 13 months after implantation. This was due to a spontaneous change in tachycardia rate.

Medication: In this selected group of patients with tachycardia, only 2 of the 13 did not require antiarrhythmic therapy after pacemaker implantation. Antiarrhythmic therapy was not always related to the tachycardia for which the system was implanted. Antiarrhythmic therapy was administered in 1 patient to prevent paroxysmal episodes of atrial fibrillation, in 1 to lengthen the anterograde refractory period of the accessory pathway, in 2 patients to reduce the rate of the tachycardia because of hemodynamic intolerance, and in 1 patient to reduce the incidence of the tachycardia.

Discussion

This RF-coupled, patient-activated system is reliable, safe and effective in terminating paroxysmal reentrant tachycardias. Its versatility is essential to allow adaptation to changing stimulation requirements, and this has avoided the need for explantation due to ineffectiveness. One to 99 stimuli are available for a treatment, with a wide range of coupling intervals. The tachycardia trigger intervals can be selected in 4-ms steps from 190 to 996 ms, compared with a limited number of preset tachycardia detection values in other devices. Other useful pacemaker activator programmable features are pulse width, delay between treatments, maximal number of treatments and the off-time. Several pacing modes are available that can be applied in different combinations. Both adaptive and fixed coupling intervals can be selected. One can choose to scan not only the first but also the last stimulus interval, or both, if 2 extrastimuli are selected.

Two modes seem to be especially effective for treating tachycardias that have a variable rate: the scanning mode and the adaptive mode using an increasing number of stimuli (Fig. 6). The adaptive mode adjusts the coupling intervals to changes in rate, and the increasing number of stimuli makes it possible to automatically use the least number of stimuli. The number of stimuli required to terminate tachycardia varies with the rate of the tachycardia (Fig. 6).

The prescription formulator is a sophisticated, portable stimulator that enables one to initiate and ter-

minate tachycardia by way of the implanted pacemaker and, thus, test and eventually tailor the treatment selected during the electrophysiologic study to different circumstances such as exercise, changes in posture, and so forth; to check the effectiveness of the selected treatment easily and noninvasively on an outpatient basis; and to ensure that the patient is using the patient activator properly.

Termination of VT by pacing has inherent risks. Therefore, implantation of these units can be done only after careful and extensive electrophysiologic evaluation not only before implantation of the unit, but also by repeated induction and termination of tachycardia during the follow-up visits. Our 4 patients with VT are still alive and unwanted rhythm disturbances were never observed.

Two types of antitachycardia pacemakers are commercially available: fully automatic systems and patient-activated systems.[3,4,6–16] Both systems have advantages, disadvantages, indications and contraindications. The patient-activated system relies on the patient as well as on the pacemaker system for recognition of tachycardia. This reduces the incidence of unnecessary treatments in the presence of sinus tachycardia, another rhythm disturbance that can be distinguished by the patient (for example, paroxysmal atrial fibrillation), and a malfunctioning system. Restrictions or disadvantages of such systems are that the pacemaker activator is breakable, can be lost or stolen, has to be within reasonable reach, and each termination is a conscious effort and the patient has to be able to tolerate the tachycardia for a longer time. In the learning phase of the application of pacing for tachycardia, we prefer the patient-activated system described because it enables us to obtain accurate information about variables such as the number of episodes of tachycardia, success of the device and frequency of reprogramming.

This system appears to have prevented many hospital admissions. Our patients have safely and quickly terminated their tachycardias at home. The system is flexible, allowing multiple pacing modes, ready reprogrammability, and easy noninvasive testing.

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Chapter 3

A Case Report Demonstrating Spontaneous Change in Tachycardia Terminating Window

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Summary

A patient who received a patient-activated pacemaker system for termination of paroxysmal, drug-resistant, ventricular tachycardia is described. During the pre-implant invasive electrophysiological (EP) study and the 3 post-implant non-invasive EP studies, tachycardias were easily and reproducibly terminated by 2 stimuli using the interval scanning mode. On the 4th day post-implant, this pacing mode no longer terminated tachycardia at rest. A new pacing mode using more stimuli was then found to terminate the tachycardia reproducibly under various physiological conditions. This case report demonstrates a spontaneous change in the tachycardia terminating window and the usefulness of versatility in anti-tachycardia pacemaker systems.

Key words: patient-activated pacing system, externally-activated pacing system, pacemaker termination of tachycardia, change in tachycardia terminating window.

Introduction

Pacing is gaining acceptance as a chronic form of therapy for re-entry tachycardias (1-6). Before implantation of an anti-tachycardia pacemaker, the tachycardia mechanism and the most appropriate site and mode of pacing to terminate tachycardia reproducibly is sought. It has previously been shown that physiological factors may change the termination window of a re-entry tachycardia (3). In this case report a successful pacing sequence became ineffective for no apparent reason. It illustrates the usefulness of a flexible pacemaker system which enables easy non-invasive electrophysiological studies under different physiological conditions and which offers several pacing modes with an adequate number of stimuli, if required.

Case report

A 50-year-old man was referred to our clinic for treatment of ventricular tachycardia. The patient developed palpitations in March 1967. The palpitations, of sudden onset and associated with anxiety, were haemodynamically well-tolerated, lasted from minutes to weeks, and had a rate of 130 to 150 beats per minute (bpm). A precipitating cause could not be identified. Numerous drugs alone or in combination had been tried unsuccessfully, including propranolol, apridin, oxprenolol, procainamide and amiodarone. Physical examination was normal. The electrocardiogram during sinus rhythm was normal. During tachycardia the QRS complexes had a width of 0.12 s left axis deviation and a right bundle branch block pattern and an R/S ratio ‹ 1 in lead V6 (Fig.1). Periods of A-V dissociation alternated with periods of 1-to-1 V-A conduction with a short V-A interval. Chest X-ray and echocardiogram were normal. Heart disease was further excluded by left and right heart catheterization with right and left ventriculography and coronary angiography. Following informed consent and without having received antiarrhythmic medication for several weeks an invasive electrophysiological study was performed according to methods described previously (7).

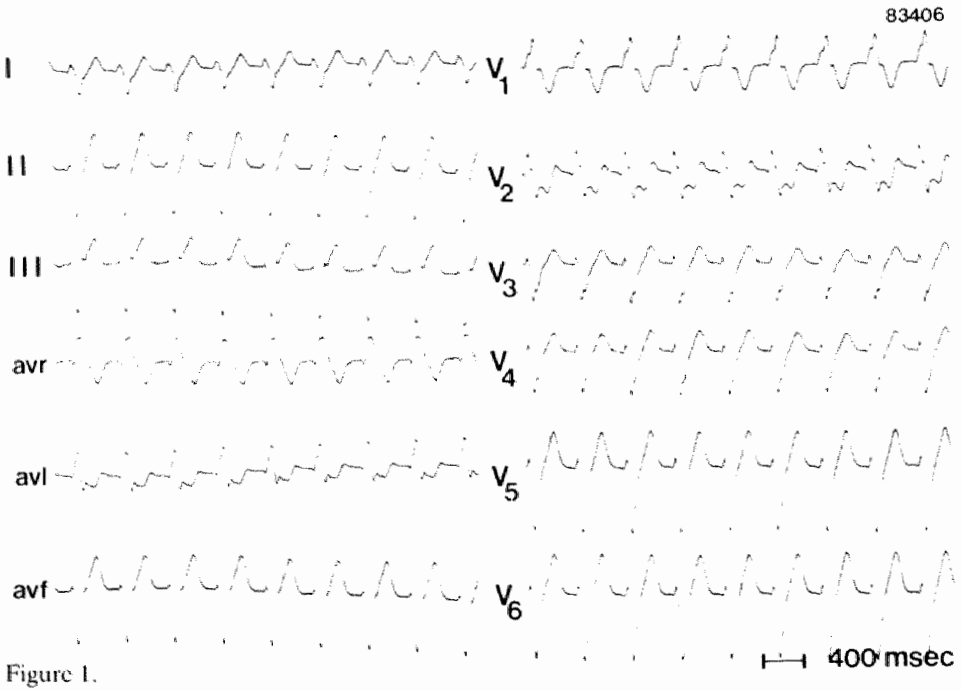

Figure 1.
Twelve-lead ECG of a ventricular tachycardia with a rate of 130 bpm. In lead III 1-to-1 V-A conduction can be seen.

Only one morphological type of monomorphic ventricular tachycardia, similar to the one clinically recorded (rate 140 bpm), could be initiated by 2 atrial premature beats during atrial pacing and also by 1 or 2 ventricular premature beats during sinus rhythm and ventricular pacing. At rest tachycardia was reproducibly terminated with 2 ventricular premature beats and overdrive ventricular pacing. During the electrophysiological study no arrhythmias other than the clinical ventricular tachycardia were noted during atrial or ventricular stimulation and following pacing to terminate tachycardia. The termination window of premature beats was wide. Because of failure of drug therapy in this patient, alternate modes of therapy were considered. The tachycardia was thought to be suitable for pacing therapy. We thought that a fully automatic pacing system was contra-indicated because of the rather slow tachycardia rate and the possible problem of differentiating between sinus tachycardia and ventricular tachycardia. Therefore, a patient-activated Medtronic model SPO500 interactive tachy pacemaker was implanted (6). A bipolar lead was placed in the right ventricular apex. By means of the prescription formulator (a sophisticated portable stimulator belonging to the interactive tachy system), tachycardias were initiated and terminated non-invasively via the radio-frequency (R-F) link with the pacemaker.No tachycardia acceleration or degeneration into ventricular fibrillation was observed during over hundred episodes of termination of tachycardia by pacing. During the first three post-operative days, 25 episodes of tachycardia were reproducibly terminated at rest by 1 or 2 stimuli using the interval scanning mode. On the 4th day, however, the same monomorphic ventricular tachycardia could not (for no apparent reason) be terminated at rest with 2 stimuli in the scanning mode. The increasing number of stimuli mode (270/250/240/240/240 ms) was subsequently chosen and it was found to terminate tachycardia reproducibly under different physiological conditions (Fig 2).
Using this modality termination is first attempted with an R-S interval of 270 ms. If this is unsuccesful, termination is automatically attempted with two premature beats with intervals of 270/250 ms. The number of stimuli are thereafter automatically increased until tachycardia has been terminated or the maximum programmed number of stimuli is reached.

In our patient a maximum of 5 stimuli were programmed. Of the 75 tachycardia terminations using this pacing mode under various physiological conditions (supine, sitting, standing and post exercise), tachycardia was sometimes terminated with 1 stimulus, but usually with 2 or 3 stimuli. A 4th stimulus was needed on 1 occassion. A 5th stimulus was

Figure 2.
ECG illustrating termination of ventricular tachycardia using the increasing number of stimuli mode. In this instance 4 stimuli were required to terminate tachycardia with coupling intervals of 270/250/240/240 ms.

never required. This pacing mode was therefore 100% effective in the 75 episodes studied. The model SPO501 pacemaker activator was subsequently programmed to this treatment and its use was explained to the patient.

In the follow-up period of 1 month the patient has been able to terminate promptly 2 tachycardias at home. It was interesting to note that when using an increasing number of stimuli with coupling intervals of 240 or 250 ms, tachycardia was terminated with an uneven number of stimuli, but reinitiated with an even number of stimuli (see fig 3).

Discussion:

During the first three post-operative days, 25 episodes of tachycardia were reproducibly terminated at rest by 1 or 2 stimuli using the interval scanning mode. The tachycardia termination window may change, due to a change in posture, autonomic tone, tachycardia rate, refractory period, drug level or for other unknown reasons. On the 4th day after implantation the previously successful pacing sequence became ineffective for no apparent reason. It became evident that frequently more than 2 stimuli were required for reproducible tachycardia termination at rest. A new pacing mode with more than 2 stimuli was then evaluated non-invasively under different physiological conditions and was effective. A versatile pacemaker system like the one described has the advantage of easy and adequate testing of the system and eliminates explantation due to ineffectiveness because of a limited number of stimuli and/or pacing modes.

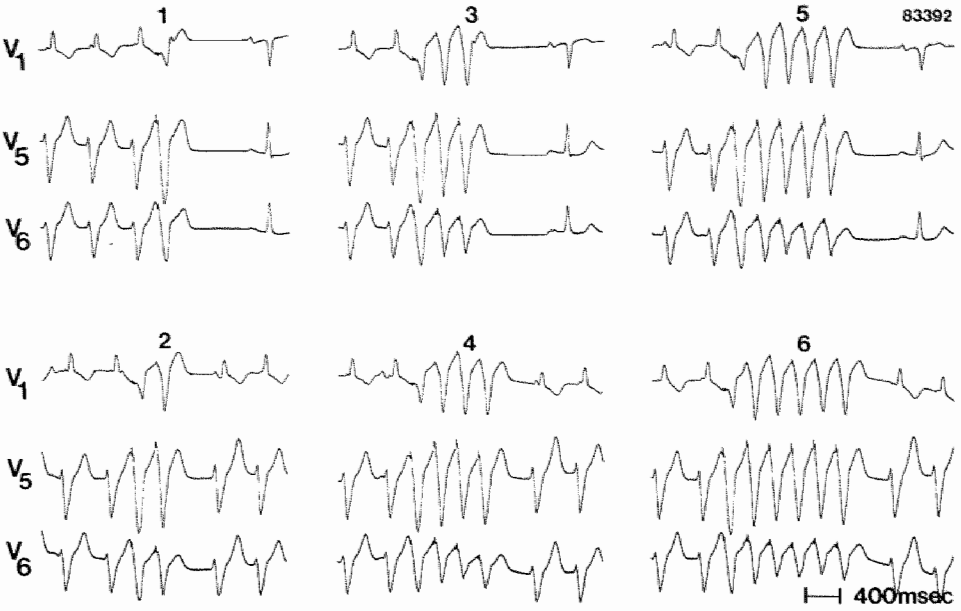

Figure 3.
Six ECG strips illustrating termination of ventricular tachycardia with an uneven number of stimuli and reinitiation with an even number. The interstimulus interval was 250 ms.

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Chapter 4

Pacemaker Related Tachycardias

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DEN DULK, K., ET AL.: Pacemaker related tachycardias. *Three cases of pacemaker interactive tachycardia are presented. The first two are (artificial) circus movement tachycardias. In the first one the retrograde arm of the tachycardia circuit was provided by the A-V node and the antegrade arm by an atrial synchronous pulse generator. In the second case, the A-V node and, coincidentally, an A-V sequential pulse generator alternately provided the antegrade arm while the retrograde arm was by way of an accessory pathway. In the third case ventricular inhibition during A-V sequential pacing gave the paced atrial events the chance to be conducted to the ventricles with a long A-V interval. This resulted in a tachycardia with a rate of 150 bpm, instead of the programmed rate of 110 bpm. (PACE, Vol. 5, July-August, 1982)*

A-V universal pacing, artificial circus movement tachycardia, pacemaker related tachycardia, dual demand pacing, retrograde conduction, electrode polarization

Pacemaker technology has made rapid progress in recent years. At the present time there are many devices available for the treatment of symptomatic bradycardias and tachycardias. The interaction, that is, the synchronization or non-synchronization between some of these sophisticated devices and the intrinsic heart rhythm, can unexpectedly give rise to new and interesting mechanisms of tachyarrhythmias. Three cases will be described that demonstrate the importance of understanding the behavior of these advanced pacing systems in order to interpret and correct the sometimes complex interaction. Carotid sinus massage has proven to be an extremely useful maneuver, both diagnostically and therapeutically.

Case No. 1

An A-V universal pulse generator (Medtronic‡ 7000) was implanted in an 81-year-old woman to prevent dizzy spells caused by high degree A-V nodal block. The pulse generator was programmed in the DDD† mode with a lower pacing rate of 60 ppm, an A-V interval of 200 ms and an upper pacing rate of 100 ppm.

On the second day after implantation, a tachycardia was noted while she was being monitored. Initiation of the tachycardia occurred after a ventricular premature beat (Fig. 1), which conducted retrogradely to the atrium. The atrial activation was sensed by the atrial amplifier, because it fell outside the atrial refractory period and it inhibited the atrial output circuit of the pulse generator. However, it did not result in a synchronized ventricular stimulus at the programmed upper rate of 100 bpm, because the A-V disable mechanism was activated. In the type of pacemaker implanted, the presence of two or more ventricular events without an interpolated atrial event disables the A-V interval timing circuit for 342 ms beginning with the second ventricular event. This feature (the so called A-V disable mechanism) was designed to prevent the device from synchronizing to a P-wave and pacing the ventricle shortly after a ventricular premature beat.

As shown in Figure 1, the next paced ventricular complex therefore occurred at the pro-

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‡Medtronic, Inc., Minneapolis, Minnesota, U.S.A.

†The three position pacemaker code proposed by the Intersociety Commission for Heart Disease is used in this article to describe the mode of pacing.

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grammed escape interval of 1000 ms. This paced ventricular event was also conducted retrogradely via the A-V node to the atrium. This time the A-V disable mechanism was not activated (because this particular ventricular complex was preceded by a sensed atrial complex) and the pulse generator produced a synchronized ventricular stimulus at an R-R interval corresponding to the programmed upper rate (100 bpm, or 600 ms.)

The second paced ventricular complex after the ventricular premature beat was also conducted retrogradely to the atrium. As can be seen in Fig. 1 the retrograde conduction time to the atrium following this ventricular complex was much longer than the ventriculo-atrial conduction interval after the spontaneous ventricular premature beat and the first paced ventricular beat, suggesting the presence of two retrograde A-V nodal pathways.

Following the second paced ventricular complex the pulse generator once again sensed the retrogradely conducted atrial complex leading to synchronization of a new pacing pulse at the ventricular level. Perpetuation of this mechanism resulted in a tachycardia. This patient, therefore, shows an artificial circus movement tachycardia. The antegrade arm is the result of pulse generator A-V synchronization and the retrograde arm is provided by ventriculo-atrial conduction over a slowly conducting A-V nodal pathway. Carotid sinus massage (Fig. 2) terminated the tachycardia by creating ventriculo-atrial Wenckebach block in the slowly conducting A-V nodal pathway.

If, as shown in Fig. 3, the atrial complex following retrograde conduction from the ventricular premature beat falls in the pacemaker's atrial refractory period, no synchronization occurs and the atrial output is not inhibited. Therefore, the pacemaker escapes at the programmed lower rate by emitting both an atrial and a

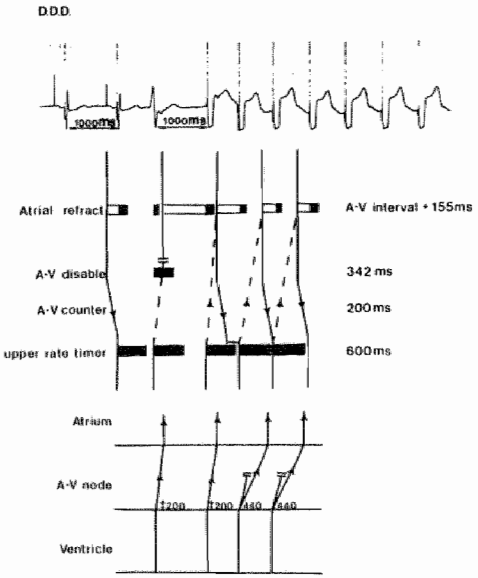

Figure 1. *Initiation of an artificial circus movement tachycardia in a patient with a Medtronic 7000 pulse generator, which was programmed to function in the A-V universal mode. See text for explanation.*

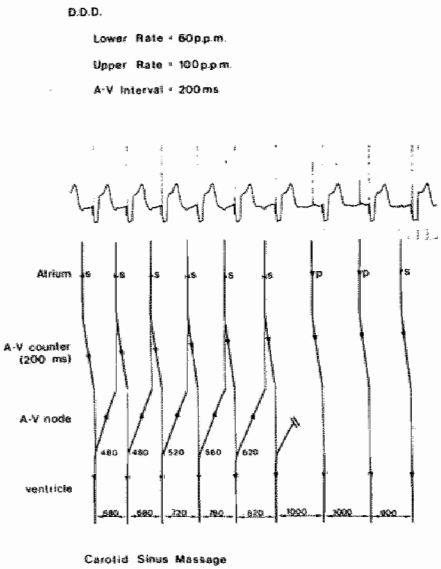

Figure 2. *Termination of the artificial circus movement tachycardia by means of carotid sinus massage by creating ventriculo-atrial Wenckebach block in the slowly conducting A-V nodal pathway.*

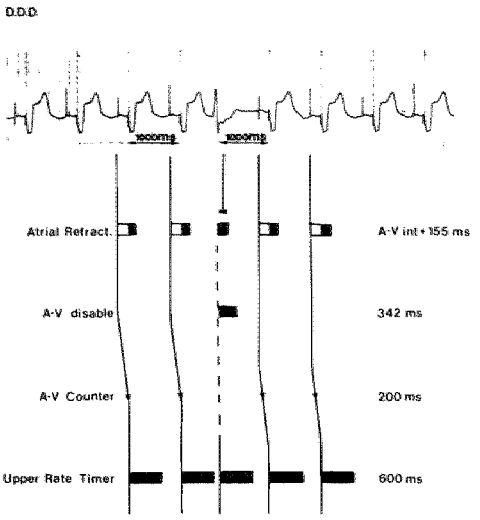

Figure 3. *Prevention of initiation of an artificial circus movement tachycardia. This occurs because the retrogradely conducted atrial event falls just inside the refractory period of the atrial amplifier and the atrial output is therefore not inhibited after the ventricular premature beat.*

ventricular stimulus. This atrial depolarization prevents the initiation of the pacemaker-induced circus movement tachycardia.

Case No. 2

A Medtronic Byrel Dual Demand (Model 5992 DD) pulse generator was implanted in a 40-year-old man, with circus movement tachycardias in the presence of a left-sided concealed accessory pathway. In this patient tachycardias could not be satisfactorily controlled by drug therapy. The pulse generator was set to function in the DVI mode with a ventriculo-atrial escape interval of 700 ms and an A-V interval setting of 150 ms.

When a tachycardia is detected this pulse generator automatically switches over to an asynchronous A-V sequential pacing mode with the same ventriculo-atrial escape interval of 700 ms, but the A-V interval is shortened to 65 ms. When the tachycardia terminates, the pacing system automatically reverts back to its original DVI mode.

Because attempts to place the atrial lead in the coronary sinus were unsuccessful, the lead was placed in the right atrial appendage. Following implantation of the atrial and ventricular leads and the pulse generator, a temporary quadripolar catheter was advanced into the right atrium to test the pulse generator.

Circus movement tachycardias with a narrow QRS complex were successfully terminated by the pulse generator (Fig. 4). The retrograde atrial activation following the paced ventricular complex was advanced to such an extent that it was blocked in the A-V node. However, following implantation of the pacing system the patient also developed circus movement tachycardias with QRS complexes showing left bundle branch block.

The tachycardia rate was slower in the presence of left bundle branch block because of the left-sided location of the accessory pathway. During left bundle branch block the pulse generator failed to terminate the tachycardia.

As shown in Fig. 5, the interstimulus interval of the pulse generator was approximately 10 ms shorter than twice the R-R interval during the circus movement tachycardia with a left bundle branch block configuration. The pulse generator marched through the tachycardia changing the form of the circus movement tachycardia. Initially, a regular tachycardia showing a left bundle branch block configuration was present; this tachycardia circuit consisted of atrium, A-V node, right bundle branch, right ventricle, left ventricle, left-sided accessory pathway, and atrium. Thereafter, following a period of fusion complexes (Fig. 5) the tachycardia was changed to a stable tachycardia with a rate equal to twice that of the pulse generator (Fig. 6).

During this tachycardia every other tachycardia complex was the result of a paced ventricular complex. The antegrade arm of the circuit was alternately provided by the A-V node and, coincidentally, by the pulse generator, while the retrograde arm was provided by the accessory pathway. Figure 7 shows how carotid

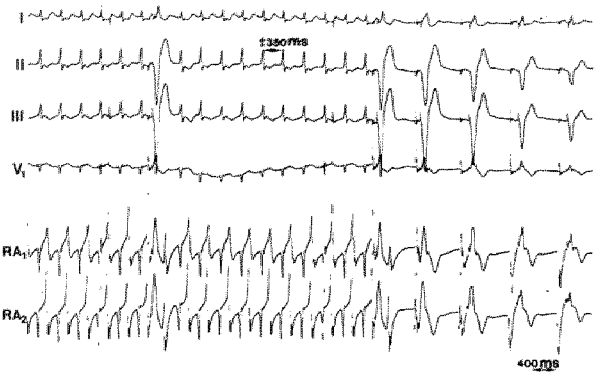

Figure 4. *Termination of a circus movement tachycardia by a Medtronic Byrel Dual Demand (Model 5992 DD) pulse generator. The atrium was retrogradely activated via a left-sided concealed accessory pathway. The pulse generator was inhibited after the first paced ventricular event, because the interval between the paced ventricular event and the next tachycardia QRS complex was longer than the tachycardia trigger interval. The pulse generator was once again activated and the second paced ventricular event advanced the atrial activation to such an extent that it blocked in the A-V node. This was followed by A-V sequential back-up pacing.*

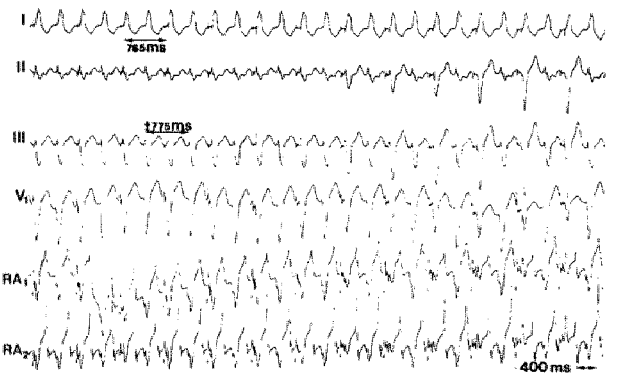

Figure 5. *Transformation of a regular orthodromic circus movement tachycardia, using a left-sided concealed accessory pathway into an irregular artificial circus movement tachycardia. Antegrade conduction is alternately provided by the A-V node and coincidentally by the pulse generator (see text). RA_1 and RA_2 are intra-atrial recordings via a quadripolar catheter.*

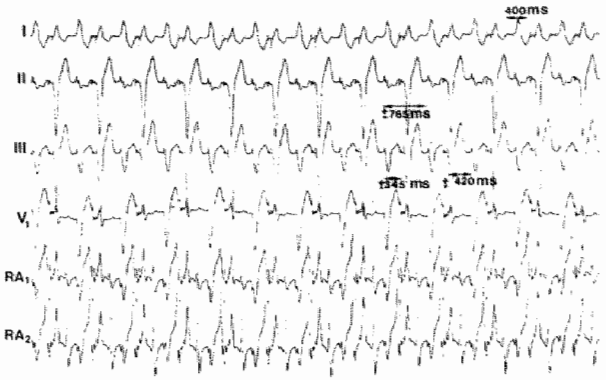

Figure 6. *Same tachycardia as in Fig. 5. The tachycardia was sustained and had to be terminated by carotid sinus massage as shown in Fig. 7.*

sinus massage facilitated termination of the tachycardia.

Case No. 3

The third tachycardia also occurred in the 81-year-old lady with the A-V universal pulse generator (Medtronic 7000) for extensive evaluation. Her pulse generator was programmed as follows: mode DVI, rate 110 bpm, A-V interval 200 ms. Although the pulse generator was programmed at a rate of 110 bpm, a tachycardia with a rate of 150 bpm was recorded during a routine check-up (Fig. 8). Figure 9 shows how this tachycardia was initiated by inappropriate

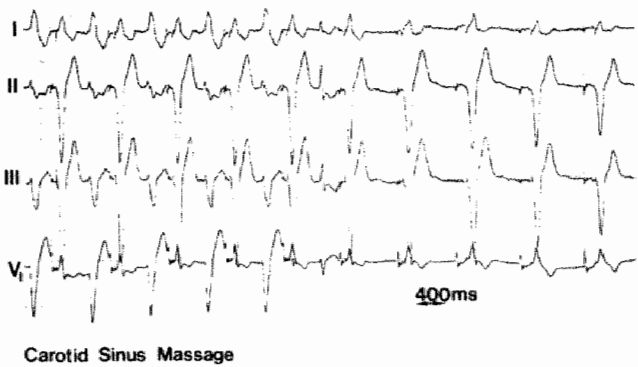

Figure 7. *Facilitation of termination of the same type of tachycardia, as shown in Fig. 6, by carotid sinus massage. During carotid sinus massage conduction resumes in the left bundle branch. This restores the ability of the pacemaker to stimulate at the appropriate interval. We cannot completely exclude the possibility that A-V block induced by carotid sinus massage terminated tachycardia. This is followed by A-V sequential back-up pacing.*

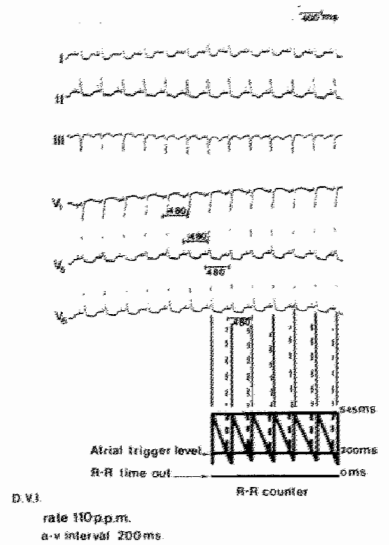

Figure 8. *Pacemaker related tachycardia. The spikes seen on the tracing are atrial stimulus artifacts. The paced atrial events are conducted to the ventricles with A-V intervals of approximately 480 ms, because ventricular outputs are inhibited by the intervening conducted ventricular complexes. As seen in the diagram the atrial and ventricular escape intervals are repeatedly reset by the intervening ventricular complexes.*

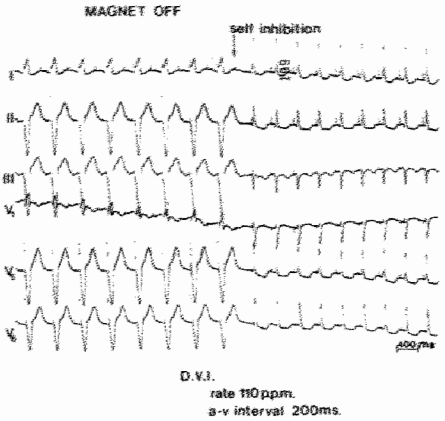

Figure 9. *Illustration of the mode of initiation of the tachycardia shown in Fig. 8. After removal of the magnet there is inhibition of the ventricular output at, or just after, the atrial stimulus artifact, as indicated. This mechanism gives the paced atrial complex the chance to be conducted with a long A-V interval by way of the normal conduction system. Just before the resulting QRS complex, a second atrial stimulus occurs, the atrial escape interval having been reset at the previous atrial stimulus artifact (by the self-inhibition).*

ventricular sensing of the atrial stimulus artifact or related polarization potentials (self-inhibition). This resulted in inhibition of the ventricular output (as shown in Fig. 9 after the removal of the magnet). The paced atrial complex was then given the chance to be conducted to the ventricles via the normal conduction system with a first degree A-V block. Just before the occurrence of the resulting QRS complex a second atrial stimulus occurred, the R-R counter having been reset at the preceding atrial stimulus. This second paced atrial event was once again conducted with a first degree A-V block. The pulse generator correctly sensed the normally conducted QRS complex and therefore inhibited its ventricular output.

This sensed ventricular event which was conducted from the previously paced atrial event, reset the R-R counter and started the timing for the next paced atrial event. The ladder diagram in Figure 8 illustrates the mechanism of this tachycardia.

Termination of such a tachycardia can easily be accomplished by placing a magnet over the pulse generator or by carotid sinus massage as shown in Fig. 10.

In Fig. 10 the tachycardia was terminated by means of an A-V nodal Wenckebach block. This was followed by pacing in a DVI mode at a rate of 110 ppm with an A-V interval of 200 ms for 6 beats. The seventh ventricular output was inhibited by polarization potentials. The atrial output which should have followed at the next reset atrial escape interval was also inhibited,

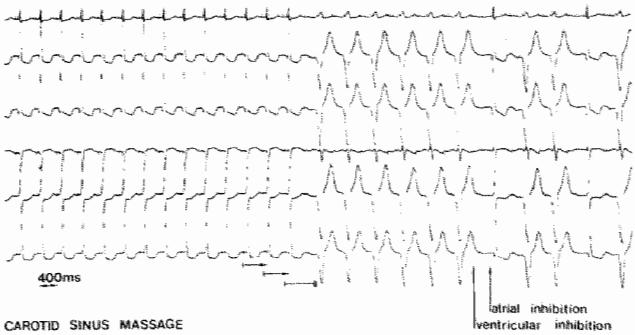

Figure 10. *Termination of the tachycardia shown in Figs. 8 and 9, by carotid sinus massage, creating A-V nodal Wenckebach block. Termination is followed by A-V sequential pacing for 6 beats. The seventh ventricular output is inhibited. The atrial output, which should have followed at the next reset atrial escape interval was also inhibited, because the analog atrial rate limit of the pulse generator was reached. This happens once again 3 beats later.*

because the shortest allowable interatrial stimulus interval was reached (analog atrial rate limit of the pulse generator).

The shortest R-R interval of such a tachycardia is a little longer than the programmed pacing rate interval minus the A-V interval, or a little longer than the atrial escape interval. The maximum possible rate of such a tachycardia can therefore be decreased by shortening the A-V interval and/or by decreasing the pacing rate. Shortening the A-V interval or decreasing the pacing rate can also be used to prevent such tachycardias. This is further illustrated in Tables I and II, which show the maximum achievable tachycardia rates with different pulse generator settings. In the first table the values are listed with an A-V interval setting of 250 ms and in the second with an A-V interval setting of 150 ms.

In the first column the pulse generator pacing rates are listed in the DVI pacing mode. In the second column the minimum P-R intervals required for such tachycardias are listed. This interval is a little longer than the maximum achievable tachycardia rate shown in column 3.

In Figure 11 there is a variation of the previously described tachycardia. This was due to deterioration of A-V conduction leading to a 3:2 Wenckebach block.

Table I.

The Maximal Possible Tachycardia Rate in a DVI Pacing Mode with the Generator Set at an A-V Interval of 250 ms

Pacing Rate (bpm) (ms)	Minimum P-R Interval (ms)	Maximal Tachy Rate (bpm)
110 (545)	295	(± 200)*
100 (600)	350	± 170
90 (666)	416	± 145
80 (750)	500	± 120
70 (857)	607	± 100
60 (1000)	750	± 80

*Atrial rate limit model 7000: 175 ppm

Table II.

The Maximal Possible Tachycardia Rate in a DVI Pacing Mode with the Pulse Generator Set at an A-V Interval of 150 ms

Pacing Rate (bpm) (ms)	Minimum P-R Interval (ms)	Maximal Tachy Rate (bpm)
110 (545)	395	± 150
100 (600)	450	± 135
90 (666)	516	± 115
80 (750)	600	± 100
70 (857)	707	± 85
60 (1000)	850	± 70

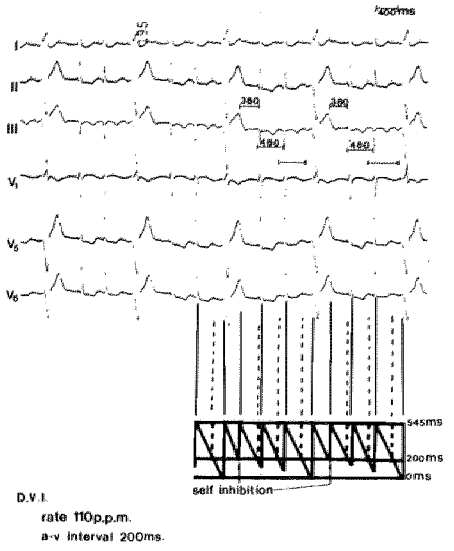

Figure 11. *This figure illustrates a variation of the previously described tachycardia. The tachycardia is repeatedly initiated by self-inhibition and terminated by a 3:2 Wenckebach block. The diagram illustrates how the pulse generator atrial and ventricular escape intervals are reset.*

As shown in Fig. 11 the sequence was repeatedly started by the self-inhibition phenomenon. This gave the first atrial paced event the chance to be conducted to the ventricles with a P-Q interval of approximately 380 ms. Just before the arrival of the conducted impulse to the ventricles there was a second atrial output. Synchronization of a ventricular output to this paced atrial complex was prevented by the conducted ventricular complex. The second atrial pacing complex was then conducted to the ventricles with a P-Q interval of approximately 480 ms. The third paced atrial complex was not conducted to the ventricles (3:2 Wenckebach block) and synchronization of a ventricular output was inhibited by the intervening conducted QRS complex resulting from the second paced atrial complex. The fourth paced atrial complex was followed by a synchronized ventricular output 200 ms later.

Discussion

Case No. 1

Ventriculo-atrial conduction is not uncommon in the human heart and presents, as shown in Fig. 1, a potential problem in using atrial synchronous pacing systems. Schuilenburg[2] showed that two-thirds of patients with normal A-V conduction had ventriculo-atrial conduction during ventricular pacing. In the presence of impaired A-V conduction the percentage of patients who had ventriculo-atrial conduction during ventricular pacing was related to the site and degree of antegrade A-V block. Roughly two-thirds of patients with uni- or bifascicular block had ventriculo-atrial conduction.[2]

In patients with second degree A-V nodal or second degree intrahissian block the incidence of ventriculo-atrial conduction diminished to 20% and 36%, respectively, while there was no ventriculo-atrial conduction in patients having complete A-V nodal or intrahissian block.[2] Fifteen percent of patients with complete subhissian block had ventriculo-atrial conduction.[2]

It is, therefore, recommended to test for the occurrence of retrograde conduction before using atrial synchronous pacing modes in pacemaker patients. If retrograde conduction is present, DDD and VDD pacing modes are to be applied with caution and special attention should be paid to the programmed upper rate. In some instances, a high upper rate may be more beneficial in preventing these artificial circus movement tachycardias or in facilitating termination by retrograde block in the A-V node.

On the other hand, a low upper rate may be indicated to limit the tachycardia rate. This has the disadvantage that the patient may not recognize that he or she has a tachycardia.

One might imagine that if the artificial circus movement tachycardia rate were 125 bpm, that by programming an upper rate of 100 bpm one would induce a pacemaker Wenckebach phenomenon. However, this is not the case, because by programming an upper rate of 100

bpm instead of 150 bpm one lengthens the interval between the retrogradely activated atrial activity and the synchronized ventricular output until the intervals between ventricular outputs have a rate of 100 bpm. The Wenckebach mechanism is dependent upon an atrial activation which falls in the refractory period of the atrial amplifier, thus not being sensed. Here the interval between the ventricular output and the succeeding atrial activation is fixed by the ventriculo-atrial conduction time.

Prolongation of the refractory period for atrial sensing after a ventricular complex will diminish the incidence of these pacemaker related reciprocating tachycardias in patients with ventriculo-atrial conduction. Such prolongation, however, will limit the response of the pacemaker to high atrial rates.

Another mode of preventing these tachycardias would be the inhibition of a single atrial synchronized ventricular output by the pulse generator on recognition of a sudden significant increase in atrial rate. This would interrupt the artificial re-entry circuit, thus ending the tachycardia. If the sudden change in rate were due to a supraventricular tachycardia a single atrial event would not be followed by a ventricular paced event. Such a sequence will have no consequences. A ventricular escape stimulus in DDD mode should always be preceded or accompanied by an atrial stimulus to reduce the likelihood of initiating an artificially induced circus movement tachycardia.

Both the prolongation of the atrial refractory period and the dual chamber escape modality in the DDD mode are presently being implemented in the circuits of the pulse generator (Medtronic 7000) described here.

Case No. 2

Recently there have been several reports on the successful use of dual demand systems. Curry et al.[3] described their experience using single chamber stimulation in patients suffering from supraventricular tachycardias; Castellanos et al.[4] reported on their success using dual chamber stimulation in patients with paroxysmal AV junctional tachycardia. Our patient shows that the occurrence of bundle branch block to the ventricle in which the accessory pathway link of the tachycardia circuit inserts may prevent interruption of the arrhythmia by a previously successful pacing system. Therefore, the effect of bundle branch block in patients having accessory pathways incorporated in their tachycardia circuit should preferably be tested before implantation of such a pacing unit.

This case illustrates some of the factors that must be considered before choosing the appropriate pacing system. In this patient the interstimulus interval of the pulse generator was coincidentally approximately equal to twice the R-R interval of the circus movement tachycardia with a left bundle branch block and the A-V node could adjust to the slight difference. This prevented the device from terminating the arrhythmia when left bundle branch block occurred during circus movement tachycardia.

Case No. 3

This tachycardia was presumed to be the consequence of self-inhibition in the early design of this universal pulse generator. The self-inhibition gave the paced atrial complex the chance to be conducted with a long A-V interval to the ventricles. The succeeding paced atrial complexes also conducted with long A-V intervals to the ventricles, because synchronization of ventricular outputs was inhibited by the intervening conducted ventricular complexes.

This situation was initiated by inhibition of a ventricular output presumably due to sensing of polarization potentials directly after the ventricular blanking period following an atrial output. This self-inhibition could only be provoked at pacing rates of at least 110 ppm. This and similar observations have initiated some design changes in this pulse generator which resulted in:

1. a reduction of polarization potentials;
2. the impossibility of generating self-inhibition in response to ventricular sensing immediately after an atrial stimulus.

Tachycardia initiation as described here requires a long atrioventricular conduction time. Also, the ventricular conducted beat must appear after the atrial output but before the ventricular output. The latter can easily be influ-

enced by changing the pulse generator rate or by shortening the A-V interval.

An atrial premature beat resulting in a conducted ventricular complex occurring after the atrial output but before the ventricular output, could also give rise to this type of tachycardia if the above mentioned requirements are fulfilled after the atrial premature beat. Similarly a ventricular extrasystole, an A-V nodal extrasystole, paroxysmal supraventricular tachycardias or sinus tachycardias could initiate such a tachycardia.

The above case cautions one to program a long A-V interval to prolong pulse generator life (by avoiding ventricular pacemaker stimuli) in patients having long atrioventricular conduction times at normal or faster atrial rates, or patients with dual antegradely conducting A-V nodal pathways. It is also important to realize that medication may influence A-V nodal conduction.

Tachyarrhythmias associated with normally functioning atrioventricular sequential demand pacemakers have been described by Santos et al.,[5] Ricci et al.,[6] and Furman et al.[7] Our observations further extend our knowledge about possible problems related to implantation of these sophisticated units. They underline the necessity of awareness by the physician that certain tachyarrhythmias can be pacemaker related. Thorough understanding of the causes of these complex arrhythmias is required to take the right programming measures to correct them.

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Chapter 5

Noninvasive Evaluation of Pacemaker Circus Movement Tachycardias

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Ventriculoatrial (VA) conduction was studied in 11 patients before DDD pacemaker implantation by incremental right ventricular pacing while recording right atrial endocavitary signals. After implantation and at 3-month outpatient visits, VA conduction and ability to initiate and sustain pacemaker circus movement tachycardia (PCMT) were systematically assessed noninvasively by testing the response of the pacemaker to asynchronous and single synchronized chest wall stimuli, muscle signals, and programmer pertubations. VA conduction was demonstrated in 3 of 11 patients before implantation as well as by noninvasive techniques after implantation with induction of PCMT. VA conduction became apparent in 2 additional patients after implantation. One of the 2 patients presented clinically with PCMT. In both patients, PCMT could be induced noninvasively. The presence or absence of VA conduction was variable, as was the rate of right ventricular pacing at which VA block occurred. In conclusion, (1) noninvasive testing techniques were effective in inducing PCMT in all patients with VA conduction and helpful in determining control of PCMTs, and (2) thorough preimplantation and repeated postimplantation assessment is needed to control and prevent PCMT.

(Am J Cardiol 1984;53:537–543)

A pacemaker circus movement tachycardia (PCMT)[2–5] is an artificial reentry tachycardia in which the retrograde limb of the circuit is provided by the atrioventricular (AV) node or accessory pathway, while the anterograde limb is by way of AV synchronization of the pacemaker. Atrial synchronous pacemakers have existed since 1962.[6] These first devices had no ventricular sensing and very long atrial refractory periods that induced 2:1 AV pacemaker block at atrial rates of 125 beats/min or greater. To sustain PCMT, all retrograde P waves must be sensed, and long atrial refractory periods make this less likely to occur. In most new DDD* systems, atrial refractory periods have been shortened to allow higher atrial tracking rates, thereby preventing the hemodynamically undesirable 2:1 AV pacemaker block[7,8] at PP intervals shorter than the atrial refractory period. Although the theoretical possibility of PCMTs was already recognized in 1973 by Castellanos and Lemberg,[9] the problem has only recently been properly realized. This study presents several techniques to evaluate noninvasively ventriculoatrial (VA) conduction in patients with a DDD pacemaker. The chronic results obtained in this way are compared with the findings at preimplant electrophysiologic evaluation.

* The 3-position pacemaker code[1] proposed by the Intersociety Commission for Heart Disease is used in this study to describe the mode of pacing.

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Methods

Patients: From December 1980 to June 1982, 11 Medtronic model 7000 pacemakers (atrial refractory period 155 ms) were implanted. All were programmed into the DDD pacing mode. Two patients were men and 9 were women, ages 36 to 84 years (mean 62). Follow-up ranged from 10 to 33 months (mean 20). The indications for implantation were symptomatic AV nodal conduction disturbances in 10 patients and symptomatic episodic sinus bradycardia with cardiac failure and first-degree AV nodal block in 1 patient (Table I). All patients except Patient 11 had normally functioning sinus nodes as judged from Holter monitor studies, preimplantation electrocardiographic monitoring and responses to exercise, carotid sinus massage and atropine.

Pacemaker description: The Medtronic Versatrax® pacemakers are unipolar, multiprogrammable pulse generators that can operate in DDD, DVI and VVI modes. Assuming that these pacing modes do not need further explanation, we will briefly describe some characteristics of these devices that are especially relevant to understanding initiation and maintenance of PCMTs. This description applies to the Versatrax models with an atrial refractory period equal to the

TABLE I Clinical Data

Case	Age (yr) & Sex	Degree AVB	Before Implant		After Implant	
			VA Cond	VAB (P/M)	VA Cond	VAB (P/M)
1	F 84	2°	+	100	+	100–130
2	M 39	3°	+	135	+	130–150
3	F 54	2°	+	90	+	100–140
4	F 64	2°	0	0	+	150–175
5	M 63	3°	0	0	+	150
6	F 36	3°	0	0	0	0
7	F 56	2°	0	0	0	0
8	F 42	3°	0	0	0	0
9	F 76	3°	0	0	0	0
10	F 84	2°	0	0	0	0
11	F 79	1°, SB	0	0	0	0

AVB = atrioventricular block; Cond = conduction; P/M = pulses per minute; SB = sinus bradycardia; VA = ventriculoatrial; VAB = ventriculoatrial block; + = present; 0 = absent.

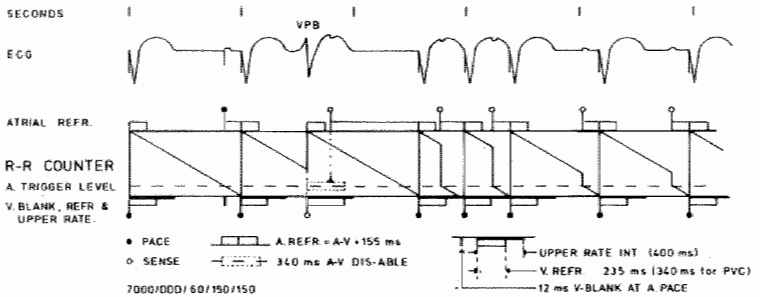

FIGURE 1. The response of the Versatrax model 7000 to a ventricular premature beat (VPB). The VPB extends the ventricular refractory period (V. REFR.) to 340 ms and starts the atrioventricular (AV) disable (DIS-ABLE) mechanism of the same duration. A retrograde P wave, occurring after the atrial refractory (A. REFR.) period but during the AV disable period, cannot start an AV interval. This retrograde P only starts the atrial refractory period, which prevents an atrial stimulus when the RR counter reaches the atrial (A.) trigger level. This causes a single ventricular stimulus at the end of the escape interval (when the R-R counter reaches its lower level). This escape, paced ventricular event conducts retrogradely to the atrium. This P wave occurs outside the atrial refractory period, is sensed, and starts an AV interval. When the AV interval is timed out, the ventricular stimulus has to wait for the end of the upper rate interval (400 ms in this example, corresponding to 150 pulses/min upper rate) to be timed out. This ventricular paced event is also conducted retrogradely to the atrium and pacemaker circus movement tachycardia is initiated. Pacemaker circus movement tachycardia terminates by means of VA block. This is followed by synchronization of ventricular paced events to sinus P waves. ECG = electrocardiogram; PVC = premature ventricular complex; V. BLANK = ventricular blanking period; VPB = ventricular premature beat.

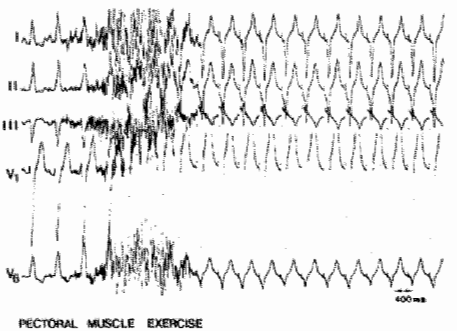

FIGURE 2. Simultaneous 5-channel electrocardiographic recording demonstrating synchronization of ventricular paced outputs to pectoral muscle artifacts with initiation of pacemaker circus movement tachycardia.

AV interval plus 155 ms (initial devices of the model 7000 series). If the pacemaker senses a ventricular premature beat (VPB) without previous atrial stimulation or sensing, the ventricular refractory period is extended from 235 to 340 ms (Fig. 1). During these 340 ms, atrial synchronization is automatically disabled. Atrial activity (sinus or retrograde) that is detected after the atrial refractory period (155 ms) but before the end of the AV disable period (340 ms) only starts an atrial refractory period. This atrial refractory period will end 155 ms after the next ventricular event. Atrial stimulation is inhibited during the total atrial refractory period. Therefore, a VPB that conducts retrogradely to the atrium with a VA interval between 155 and 340 ms will be followed by a ventricular escape stimulus. This paced ventricular event may conduct retrogradely to the atrium and initiate PCMT. The ventricular pacing rate is limited by a Wenckebach mechanism: When the AV interval times out before the programmed upper rate interval has finished, the ventricular stimulus will be suspended until completion of the upper rate interval (Fig. 1).

To avoid confusion, model 7000 pacemakers in Europe and all model 7000A pacemakers as they are now available have

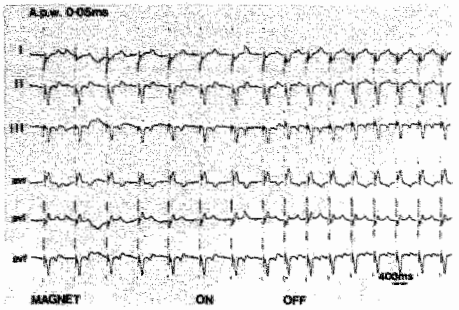

FIGURE 3. Simultaneous 6-channel electrocardiographic recording illustrating induction of a pacemaker circus movement tachycardia by programming an atrial pulse width (A.p.w.) that does not pace the atrium in the DDD pacing mode. With the magnet over the pacemaker, the ventricle is paced asynchronously at the magnet rate of 85 pulses/min. The paced ventricular events are conducted retrogradely. On removal of the magnet with the sensing systems again functional, pacemaker circus movement tachycardia is initiated.

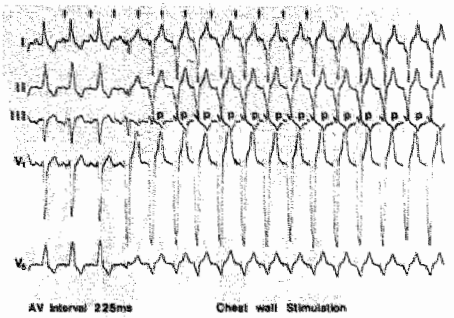

FIGURE 4. Simultaneous 5-channel electrocardiographic registration illustrating induction of pacemaker circus movement tachycardia by asynchronous chest wall stimulation due to synchronization of ventricular pacing by the pacemaker to the chest wall stimuli. **Arrows** indicate chest wall stimuli. AV = atrioventricular; p = retrograde atrial activation.

a total atrial refractory period equal to the AV interval plus 235 ms; more important, they escape in DVI instead of VVI mode after a VPB followed by VA conduction that is sensed during the AV disable period (i.e., the atrial refractory period does not inhibit the atrial stimulus). This modification was implemented to prevent the initiation of PCMT after a VPB by the paced ventricular escape beat.

Evaluation before implant: Before pacemaker implantation, VA conduction was assessed by right ventricular pacing while simultaneously recording right atrial electrograms and surface electrocardiographic leads. The right ventricle was paced incrementally. Pacing was started at a rate that captured the ventricle. This was gradually increased to a rate just above the spontaneous sinus rate. In the presence of retrograde conduction, the maximal VA conduction time and the right ventricular pacing rate at which retrograde block occurred were determined. These values, in combination with the feasibility of programming a high enough upper rate setting and an appropriate AV interval to create retrograde block at the onset of PCMT, determined whether DDD pacing was contraindicated.

Postimplantation and follow-up evaluation: All patients underwent continuous electrocardiographic monitoring for at least 24 hours after pacemaker implantation. Noninvasive assessment of VA conduction and PCMT was done before discharge and repeated at 3-month intervals. At follow-up, treadmill exercise electrocardiographic lead placement was used to provide adequate recording during the different maneuvers. Holter studies and exercise tests were not performed routinely. The following methods were used to evaluate VA conduction and PCMTs noninvasively:

Isotonic pectoral muscle exercise: Pectoral muscle artifacts were sensed as P waves. The resulting paced ventricular events could then be conducted retrogradely to the atria and PCMT could ensue (Fig. 2).

Use of the programmer: Programming a subthreshold atrial pulse width: In the DDD mode, the atrial pulse width was programmed to its minimal value (0.05 ms) to lose atrial capture. (This did not always occur.) When the magnet was then placed over the pacemaker, only the ventricles were stimulated asynchronously at the magnet rate of 85 pulses/min, AV synchrony was lost and VA conduction could occur. On magnet removal, atrial and ventricular sensing were restored and PCMT was sometimes initiated (Fig. 3).

Use of the inhibit mode: When the "inhibit" function is selected on the programmer, the programmer head will emit radiofrequency signals at about 500-ms intervals that instruct the pacemaker to inhibit pacing. When the programmer head is kept just far enough from the implanted device to prevent closure of the reed-switch, the pacemaker will not go into magnet mode and will therefore not be susceptible to programming, in this case to inhibition. However, the radiofrequency signals picked up by the lead antenna may be "sensed" by the atrial or ventricular sense amplifier. A ventricular stimulus, if picked up at the atrial amplifier only, can be synchronized to this event, which may then be conducted retrogradely to the atrium, thereby permitting induction of PCMT.

Asynchronous chest wall stimulation: Electrocardiographic electrodes were placed on the chest wall and connected to an

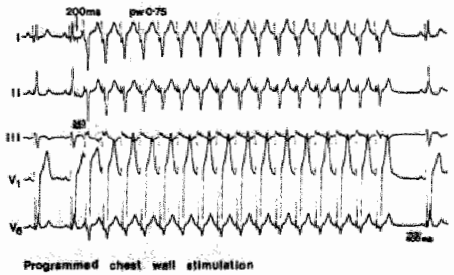

FIGURE 5. Simultaneous 5-channel electrocardiographic registration illustrating induction of pacemaker circus movement tachycardia by a timed chest wall stimulus. A timed chest wall stimulus (with a pulse width [pw] of 0.75 ms) was delivered 200 ms after a ventricular output by the pacemaker. The chest wall stimulus is sensed by the atrial amplifier and the pacemaker synchronizes a ventricular output at the pulse generator's programmed upper rate interval (343 ms). This ventricular event is conducted retrogradely to the atrium followed by pacemaker circus movement tachycardia.

external stimulator. The stimulator output was adjusted so that chest wall stimuli were sensed as P waves, but not as ventricular depolarizations. The pacemaker-synchronized ventricular outputs to these stimuli and PCMT could be initiated (Fig. 4).

Programmed chest wall stimulation: The same setup was used for programmed chest wall stimulation as was described for asynchronous chest wall stimulation. However, now a single chest wall stimulus was coupled with a programmed interval (R-S_1) to a ventricular pacemaker stimulus. This ventricular stimulus was sensed by the sensing system of the external stimulator (Medtronic model SP 503 Interactive Tachy System prescription formulator). The pacemaker AV interval was programmed to a value ensuring ventricular (fusion) pacing and the ventricular pulse width was programmed to 1.5 ms to facilitate external sensing of the pacing artifacts. The timing of the premature ventricular stimulus during sinus rhythm was determined by the selected chest wall stimulation interval (R-S_1) and the programmed AV interval. The degree of prematurity was limited by the programmed upper rate (Fig. 5). (The shortest possible premature interval was 340 ms at an upper rate setting of 175 pulses/min.)

The chest wall stimulus had to be of an amplitude that was sensed by the atrial amplifier of the pacemaker but not by the ventricular amplifier. If the chest wall stimulus was only sensed by the ventricular amplifier, all timing sequences were reset and the ventricular output was inhibited. If it was simultaneously sensed by the atrial and the ventricular amplifier, a ventricular output was triggered by the sensed atrial event. This did not occur after the programmed AV interval, but only after the upper rate interval was timed out from the chest wall stimulus, because the latter had reset the upper rate counter through the ventricular amplifier (Fig. 6). Resetting of the upper rate counter in this way may prevent one from pacing the ventricle as prematurely as one may wish to. When necessary, the ventricular sensitivity value was programmed to a less sensitive value to prevent this from happening.

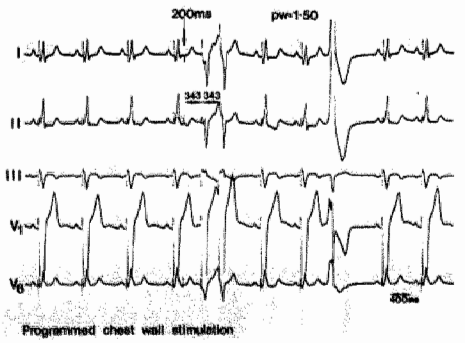

FIGURE 6. Simultaneous 5-channel electrocardiographic recording illustrating the reaction of the pulse generator to a chest wall stimulus sensed by atrial and ventricular amplifiers. The chest wall stimulus (with a pulse width [pw] of 1.5 ms) is given 200 ms after a pacemaker ventricular output, and is sensed by the atrial amplifier. This starts the pacemaker atrioventricular interval timer. The chest wall stimulus is also sensed by the ventricular amplifier. This delays the synchronization of the ventricular output after the atrioventricular interval is timed out because the upper rate interval (343 ms) is timed from the chest wall stimulus. The next ventricular event synchronizes to a sensed sinus P wave at the programmed upper rate limit.

VVI pacing: The ventricle was paced at different rates in VVI mode and VA conduction was assessed from the surface ECG (Fig. 7).

Chest wall thump: This procedure was used occasionally to induce ventricular premature beats that could initiate PCMT.

Inducement of the Wenckebach response: By increasing the sinus rate (for example, with exercise) and programming a low upper rate, the Wenckebach mode was induced when the sinus rate exceeded the programmed upper rate limit. The P waves and paced ventricular events dissociated and VA conduction and PCMT were made possible (Fig. 8).

Results

VA conduction was demonstrated in 3 of the 11 patients before pacemaker implantation (by means of incremental right ventricular pacing) as well as by noninvasive techniques after implantation (Table I). PCMT could be induced in all 3 patients. Two of the 8 patients who did not have VA conduction before pacemaker implantation had VA conduction afterwards. One (Patient 4) was admitted to the hospital with PCMT 2 weeks after implantation. The other (Patient

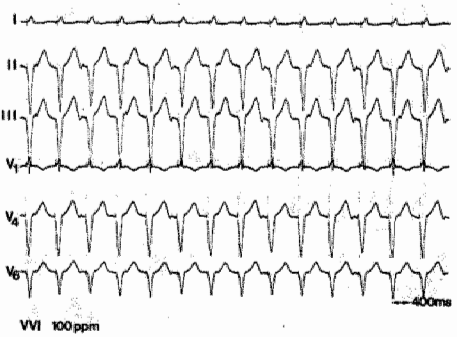

FIGURE 7. Simultaneous 6-channel electrocardiographic recording illustrating 3:1 ventriculoatrial block during VVI pacing at a rate of 100 pulses/min (ppm).

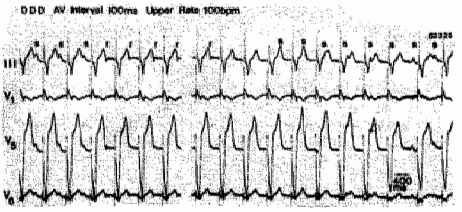

FIGURE 8. Simultaneous 4-channel electrocardiographic recording illustrating pacemaker circus movement tachycardia induction by a Wenckebach pacemaker response during exercise. Pacemaker circus movement tachycardia terminates because of ventriculoatrial block. AV = atrioventricular; bpm = beats/min; r = retrograde P waves; S = sinus P waves.

5) was found to have PCMT in the outpatient clinic by the previously described noninvasive testing methods. In both patients PCMT could then be induced noninvasively.

Patient 4, who presented with PCMT 2 weeks after implantation, had frequent VPBs originating from the right ventricular outflow tract for many years. No medication was given at discharge to prevent these VPBs because they were not troublesome and we did not have evidence of VA conduction before implantation, while monitoring after implantation, or by way of noninvasive techniques before discharge. This patient was admitted 1 week later because of paroxysmal PCMT initiated by VPBs originating from the right ventricular outflow tract. Tachycardias terminated spontaneously after a few seconds because of failure of VA conduction (Fig. 9).

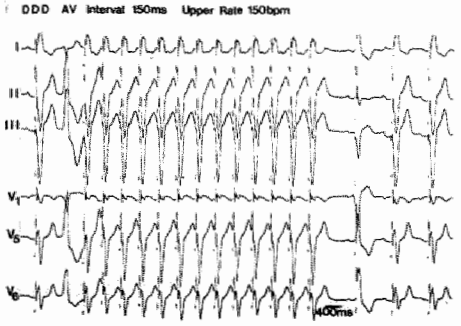

FIGURE 9. Simultaneous 6-channel electrocardiographic recording illustrating initiation of pacemaker circus movement tachycardia by a ventricular premature beat originating from the right ventricular outflow tract (see text for explanation). The tachycardia terminates spontaneously because of retrograde ventriculoatrial block. AV = atrioventricular; bpm = beats/min.

The mode of initiation of PCMT in Patient 4 was different from that which we previously described.[3] VA conduction and PCMT were assessed noninvasively 1 hour after admission. On admission, the pacemaker was functioning as follows: DDD mode, lower rate 50 pulses/min; AV interval 150 ms, upper rate 150 pulses/min. During testing the upper rate had to be lowered from 150 to 125 pulses/min to initiate and sustain PCMT for several seconds (Fig. 10). In Figure 10, an atrial output was programmed that did not pace the atrium. The ventricle was paced at the magnet rate of 85 pulses/min. Each paced ventricular event was conducted retrogradely to the atrium. When the programmer head was removed (in the panel on the right with a programmed upper rate of 125 pulses/min) and the sensing systems were functional again, a PCMT was initiated at the upper rate setting of 125 pulses/min.

An upper rate setting of 150 pulses/min was not able to maintain 1:1 VA conduction, and PCMT terminated after the first atrial synchronized ventricular paced event after removal of the magnet. An AV interval of 125 ms and an upper rate setting of 175 pulses/min was then chosen to prevent PCMTs by means of VA block of the first atrial synchronized ventricular paced event. The next morning several self-terminating PCMTs with a rate of 175 pulses/min were registered. The patient was then given disopyramide. This diminished the number of VPBs and abolished VA conduction and PCMT. Absence of VA conduction was also demonstrated during and after a treadmill exercise test.

In Patient 5, VA conduction appeared 3 months after pacemaker implantation. The patient had no complaints of palpitations at that time. When the tachycardia was initiated at the outpatient clinic, he admitted that he had felt a similar sensation on several occasions, but it never bothered him and it never lasted long. He

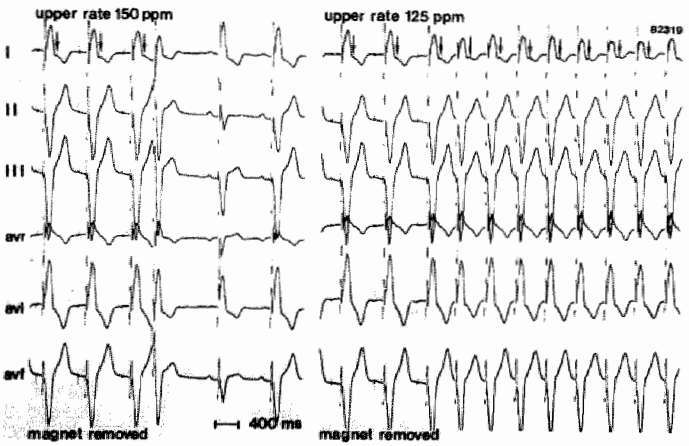

FIGURE 10. Two strips of simultaneous 6-channel electrocardiographic recordings during noninvasive induction of pacemaker circus movement tachycardia. In both cases an atrial pulse width was programmed to ensure loss of atrial capture during pacing. To the **left of each panel** the pulse generator paces in the DDD mode at the magnet rate of 85 pulses/min (ppm). Each of these ventricular pace events is conducted retrogradely to the atrium. The panel on the **left** illustrates ventriculoatrial block after the first atrial synchronized ventricular paced event after removal of the magnet when the pacemaker was programmed to an upper rate setting of 150 ppm. In the panel on the **right** pacemaker circus movement tachycardia is sustained with the pulse generator programmed to an upper rate setting of 125 ppm when the magnet was removed.

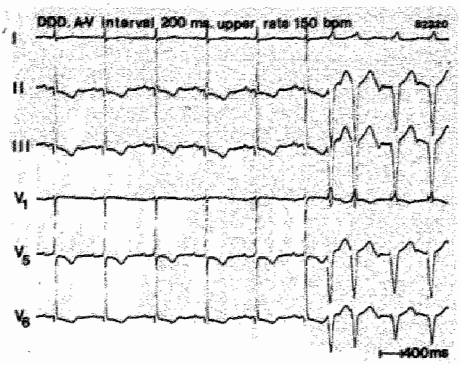

FIGURE 11. Simultaneous 6-channel electrocardiographic registration illustrating initiation of pacemaker circus movement tachycardia in the same patient as that in Figure 7 at an upper rate setting of 150 pulses/min. AV = atrioventricular; bpm = beats/min.

was treated with disopyramide, which resulted in disappearance of the sensation. VA conduction could no longer be demonstrated at repeated noninvasive assessments.

VA conduction was variable in these 5 patients, as was the right ventricular pacing rate at which VA block occurred in 4 of the 5 patients. In Patient 5 VA conduction was observed only once; after administration of disopyramide no more VA conduction was observed.

The right ventricular pacing rate at which VA block occurred ranged from 20 to 40 pulses/min in each of 4 patients. As described previously, in Patient 4, it varied from 150 to 175 pulses/min. One patient had dual retrograde AV nodal pathways illustrated by the initiation of a PCMT (Fig. 11). The first paced ventricular complex was conducted retrogradely, with a VA interval of ±200 ms, and the subsequent paced complexes were conducted retrogradely with VA intervals of 440 to 480 ms. The variability in VA conduction is demonstrated in Figure 11. An upper rate setting of 150 beats/min could not prevent PCMT with the first 2 ventricular pace events close to the programmed upper rate interval (400 ms); it had been effective at 100 beats/min (ventricular coupling interval of 600 ms).

Discussion

There are 2 possible mechanisms of initiation of the PCMT (Fig. 9). These 2 possible mechanisms are different from the one previously described[3] in which PCMT was not initiated directly by the VPB, but by the paced ventricular escape event thereafter (Fig. 1). Tachycardia starts directly after the VPB at the upper rate interval of 400 ms (Fig. 9). One possible mechanism is that the sinus P waves and the VPBs occurred and were sensed simultaneously by the respective amplifiers. The other possibility is the simultaneous sensing of VPBs by the atrial amplifier[10] as well as by the ventricular amplifier. In both cases the pacemaker then synchronized a ventricular output to the event sensed by the atrial amplifier. This ventricular stimulus did not occur after the AV interval, but after the upper rate interval that was reset by the VPB.

This is the same kind of response described in the Methods section during chest wall stimulation. We could not program an atrial sensitivity value that did not sense VPBs but that did still sense the spontaneous atrial events. Not every VPB initiated such a response. This could have been because VPBs were only intermittently sensed by the atrial amplifier or that timing of the sinus P wave in the initial portion of the VPB was not right (i.e., ± simultaneous sensing). VPBs did not conduct retrogradely (possibly due to concealed anterograde penetration[11] from the sinus P wave in the beginning of the QRS complex), but the next paced ventricular event after the VPB was conducted retrogradely and PCMT ensued.

Retrograde conduction through the AV node to the atrium is not uncommon in the human heart. Akhtar[12] recently reviewed retrograde conduction in human subjects and the functional behavior of the cardiac conduction system in retrograde direction. Schuilenburg[13] demonstrated that 68% of patients with normal AV conduction had VA conduction during ventricular pacing, whereas the remaining 32% exhibited complete retrograde AV nodal block.

In 150 patients who had AV conduction problems, the incidence of VA conduction decreased to about 44%.[13] Schuilenburg also tried to relate the presence or absence of VA conduction to the site of block in the AV conduction system. It is difficult to draw conclusions because the number of patients in the subgroups was small. For example, in 6 patients who had third-degree anterograde block in the His bundle and in 6 patients who had third-degree block in the AV node, he observed no VA conduction, whereas from a total of 13 patients with trifascicular block, 2 or 15% had VA conduction.

It is therefore recommended to test for the occurrence of retrograde conduction before using atrial synchronous pacing modes. If VA conduction is present, DDD and VDD pacing modes are to be applied with caution. The presence of VA conduction does not preclude the use of DDD or VDD pacing. Rather, the test results may be used to determine the proper pacing variables. It is relevant to determine before implantation whether the maximal VA interval falls within the atrial refractory period of the pacemaker and to determine at what ventricular pacing rates VA block will be induced. If block is created by pacing the ventricle at, for example, 90 pulses/min, then a programmed upper rate of 150 or 175 with an appropriately short AV interval will allow the creation of retrograde block within 1 or 2 beats of the PCMT and terminate it. The prematurity of the atrial synchronized ventricular paced event (that coupled to the retrogradely conducted atrial event) is also determined by the spontaneous VA interval. If this is too long, the atrial synchronized ventricular paced event may occur too late to create VA block.

Incremental right ventricular pacing before implantation did not reliably identify all patients having the ability to conduct retrogradely to the atrium (Table I).

In the setting of a diseased conduction system, a Mobitz II type VA block may exist, and one therefore cannot be sure that another pacing protocol with or without the addition of medication would have identified all patients who had retrograde conduction to the atrium.

The AV sequential pacing method may identify additional patients with the capacity to conduct retrogradely to the atrium[14] by preexciting the atrium and thereby clearing the VA pathway. However, this method does not reveal if a PCMT will be sustained or what the maximal VA interval is. The maximal VA interval will determine the ultimate rate of the PCMT, and this is the interval that must be considered if one relies on the atrial activation to occur during the atrial refractory period of the pacemaker. The VA interval at induction of a PCMT by way of an atrial premature beat (which induces the pacemaker Wenckebach behavior) is not necessarily the same as during AV sequential pacing with the induction of a VPB; the VA interval could be longer because of concealed penetration by the atrial premature beat.[11] Initiation by a VPB with this particular model pacemaker was through the ventricular escape paced beat after the VPB.

Because of its variability, VA conduction (even if initially absent) should be assessed repeatedly to be sure that PCMT has been properly controlled. Initiation of PCMT with these noninvasive techniques was done during ventricular pacing and during AV synchrony. The value of these techniques is the ability to study the condition with the pacemaker and leads in situ (after the initial invasive screening test) and the tests can be done under different physiologic conditions. It permits evaluation of whether VA conduction is paroxysmal and assessment of whether sufficient measures have been taken to prevent PCMT. This may become important after having discontinued a drug or after the addition of a drug after pacemaker implantation.

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Chapter 6

Management of Pacemaker Circus Movement Tachycardias

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DEN DULK, K., LINDEMANS, F., AND WELLENS, H.J.J.: **Management of pacemaker circus movement tachycardias.** *DDD pacemakers were implanted in 11 patients of whom 5 had the capacity to conduct retrogradely to the atrium. Methods to prevent or terminate pacemaker circus movement tachycardia (PCMT) were evaluated in these patients. V-A conduction was assessed before implantation by incremental right ventricular pacing while recording right atrial electrograms. Following implantation and at quarterly outpatient clinic visits, V-A conduction and ability to initiate and sustain PCMT were systematically assessed by non-invasive techniques. PCMT could be induced non-invasively in all 5 patients. The methods used to reduce and terminate the incidence of PCMT were: 1) decreasing the atrial sensitivity; 2) stressing the V-A conduction system by programming a high upper rate with an appropriately short A-V interval; 3) programming a low lower rate; 4) avoiding the Wenckebach response (by programming a high upper rate); 5) medication; and 6) occasionally by using a magnet. PCMT was controlled in all patients, in 2 patients by programming measures only and in 2 with the addition of medication. One patient who refused medication had to be programmed into another pacing mode. We conclude that : 1) the presence of V-A conduction is not an absolute contraindication to the use of a DDD pacing system; 2) pacing the ventricle early enough to cause V-A block was the most useful method to terminate PCMT; 3) future generation DDD pacemakers should prevent initiation of PCMTs while maintaining the possibility to synchronize to exercise-induced high atrial rates. (PACE, Vol. 7, May-June, 1984)*

pacemaker circus movement tachycardia, artificial circus movement tachycardia, ventriculo-atrial conduction, pacemaker mediated tachycardia, endless loop tachycardia

Introduction

As shown by Schuilenburg,[1] ventriculo-atrial (V-A) conduction is not rare, even in the presence of abnormal A-V conduction. He found that 34 (68%) of 50 patients with normal A-V conduction and 66 (44%) of 150 patients with abnormal A-V conduction had V-A conduction. In patients with a DDD* pacemaker the presence of V-A conduction may give rise to pacemaker circus movement tachycardias (PCMT) when the retrograde atrial activation is sensed by the atrial amplifier of the pacemaker, which in turn causes the delivery of a ventricular stimulus, which can be followed by V-A conduction, and so on. If ventricular depolarizations are preceded by atrial depolarizations, V-A conduction is not likely to occur because the atrial activity will have depolarized part of the retrograde pathway, thereby causing refractoriness. If, however, the atrial and ventricular events are sufficiently separated so that the retrograde pathway is no longer refractory, V-A conduction and PCMT may ensue. This can occur with VPBS, Wenckebach upper rate behavior during sinus tachycardia or APBS, or in the case of synchronization of a ventricular output to external artifacts sensed as P waves.

To manage PCMT in patients with a DDD pacemaker properly one must be 1) well-informed about the V-A conduction properties and times; 2) well-acquainted with the pacemaker system and pos-

*The three-position pacemaker code[2] proposed by the Intersociety Commission for Heart Disease is used in this article to describe the mode of pacing.

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sible measures to prevent PCMT; 3) able to evaluate V-A conduction and PCMT non-invasively. Our experiences in the management of PCMT are presented in this paper.

Material and Methods

Patient Population

Between December, 1980 and June, 1982 11 Medtronic* Model 7000 (Versatrax) pacemakers were implanted and programmed to the DDD pacing mode. Three of the 11 patients were male and 8 were female. Their ages ranged from 36 to 84 years (mean, 62 years).

The indications for implantation were symptomatic A-V nodal conduction disturbances in 10 patients and symptomatic episodic sinus bradycardia with cardiac failure and 1st degree A-V nodal block in 1 patient.

All patients except the last had normally functioning sinus nodes as judged from Holter monitor studies, pre-implantation ECG monitoring and responses to exercise, carotid sinus massage and atropine.

Pacemaker Description

The Medtronic Versatrax pacemaker is a multiprogrammable, double unipolar device that can operate in DDD, DVI or VVI mode. In the DDD mode the atrium is paced at the programmed lower rate, unless the spontaneous atrial rate is higher than the programmed lower rate, in which case the atrial output will be inhibited. Atrial (pace and sense) events trigger an A-V interval of programmable duration. After this interval, a ventricular stimulus is delivered unless spontaneous ventricular activity is detected earlier. In this way, ventricular stimulation is synchronized to both inherent atrial activity and atrial stimuli.

If the sensed atrial rate exceeds the programmed upper rate, the ventricular stimulation rate will not exceed the programmed upper rate. This is realized by gradual prolongation of the A-V interval which results in Wenckebach behavior. The atrial refractory period is started by all atrial and ventricular events and ends 155 ms** after the ventricular event. During the pulse generator's atrial refractory period, signals at the atrial input will be ignored and atrial pacing cannot occur during this period.†

If a ventricular event is not preceded by an atrial event, as in the case of a ventricular premature beat, the ventricular refractory period is extended from the normal 235 ms to 340 ms, during which atrial synchronization is disabled. During this A-V disabled period, a sinus or retrograde P-wave occurring after the atrial refractory period but before the end of the A-V disable period of 340 ms, starts an atrial refractory period.

This extends to 155 ms** after the next ventricular sensed or paced (lower rate) event. As stated before, atrial pacing is inhibited during the atrial refractory period.†

Therefore, a VPB followed by a P-wave with a V-A time longer than 155 ms but shorter than 340 ms results in only a ventricular stimulus[3] at the pacemaker escape (lower rate) interval.† This VVI-escape may again be followed by V-A conduction which can then start PCMT. Retrograde P-waves occurring after the A-V disable period will be sensed and PCMT may ensue.

Pre-implant Evaluation

V-A conduction was assessed before pacemaker implantation as follows: a quadripolar catheter was placed in the right atrial appendage for recording and stimulation and a bipolar catheter was placed in the right ventricular apex for stimulation. Simultaneous recordings were made of two unipolar right atrial endocavitary signals (using precordial ECG leads) and four surface ECG leads. The right ventricle was paced incrementally, starting at a rate which captured the ventricle. This was gradually increased to a rate just above the spontaneous sinus rate. In the presence of retrograde conduction, the maximum V-A conduction time and the right ventricular pacing rate at which retro-

*Medtronic, Inc., Minneapolis, Minnesota, U.S.A.

**235 ms for Versatrax with presently available modified circuitry.

†This no longer holds for the presently manufactured Versatrax pacemakers with a 235 ms atrial refractory period. They escape in DVI instead of VVI mode after a VPB which is followed by retrograde atrial activation sensed during the 340 ms ventricular refractory period.

grade block occurred were determined. These values, in combination with the feasibility of programming a sufficiently high upper rate setting and an appropriate A-V interval to create retrograde block at the onset of PCMT, determined whether DDD pacing was contraindicated.

Post-implant and Follow-up Evaluation

All patients underwent continuous ECG monitoring for at least 24 hours after pacemaker implantation. Non-invasive assessment of V-A conduction and PCMT was done before discharge and was repeated every 3 months. At follow-up, treadmill exercise ECG lead placement was used to provide adequate recording during the different maneuvers. Holter studies and exercise tests were not performed routinely. The following methods were used to evaluate V-A conduction and PCMTs non-invasively. All of these methods induce A-V dissociation.

Isotonic pectoral muscle exercise. Pectoral muscle artifacts were sometimes sensed as P-waves. The resulting paced ventricular events could then be conducted retrogradely to the atria and PCMT could enuse.

Using the programmer. 1) *By programming a subthreshold atrial pulse width.* In DDD mode, the atrial pulse width was programmed to its minimum value (0.05 ms) in order to lose atrial capture. (This did not always occur.) When the magnet was then placed over the pacemaker, only the ventricles were stimulated asynchronously at the magnet rate of 85 bpm, A-V synchrony was lost and V-A conduction could occur. Upon magnet removal, atrial and ventricular sensing were restored and PCMT was sometimes initiated, as shown in Fig. 1.

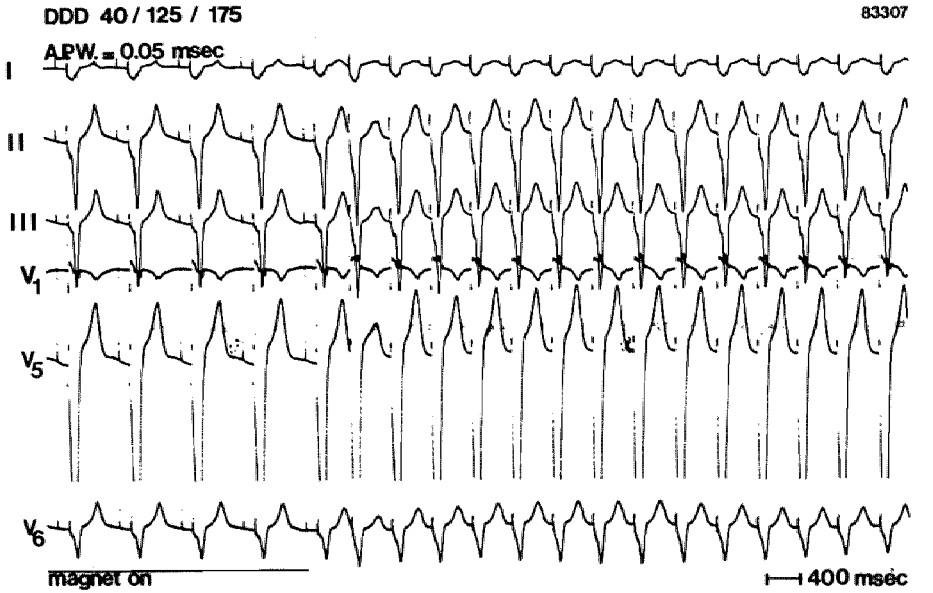

Figure 1. *Simultaneous 6-channel ECG illustrating induction of a PCMT by programming an atrial pulse width not pacing the atrium in the DDD pacing mode. With the magnet over the pacemaker the ventricle was paced asynchronously at a magnet rate of 85 bpm. The paced ventricular events were conducted retrogradely, and when the magnet was removed and the sensing systems again functional, PCMT was initiated.*

2) *By using the inhibit mode.* When the "inhibit" function is selected on the programmer, the programmer head will emit radio-frequency (RF) signals at intervals of about 500 ms, which will instruct the pacemaker to inhibit pacing. When the programmer head is kept just far enough from the implanted device to prevent closure of the reed-switch, the pacemaker will not go into the magnet mode and will therefore not be susceptible to programming—in this case, to inhibition. The R-F signals picked up by the lead antenna may be "sensed" by the atrial or ventricular sense amplifier, however. If picked up at the atrial amplifier only, a ventricular stimulus can be synchronized to this event which may then be conducted retrogradely to the atrium, thereby making induction of PCMT possible.

Asynchronous chest wall stimulation. ECG electrodes were placed on the chest wall and connected to an external stimulator. The stimulator's output was adjusted to allow chest wall stimuli to be sensed as P-waves but not as ventricular depolarizations. The pacemaker-synchronized ventricular outputs to these stimuli and PCMT could be initiated.

Programmed chest wall stimulation. For programmed chest wall stimulation, the same set-up was used as described for asynchronous chest wall stimulation. However, now a single chest wall stimulus was coupled with a programmed interval (R-S1) to a pacemaker ventricular stimulus. This ventricular stimulus was sensed by the sensing system of the external stimulator (Medtronic Model SP 503 Interactive tachy system prescription for-

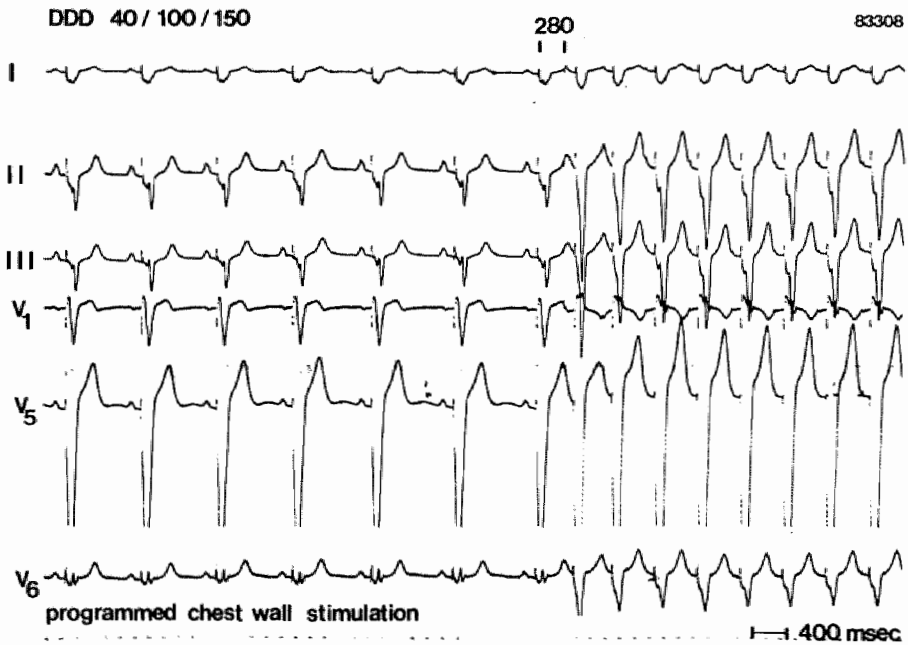

Figure 2. *Induction of PCMT by a timed chest wall stimulus delivered 280 ms after a ventricular output by the pacemaker. The chest wall stimulus is sensed by the atrial amplifier and the pacemaker synchronizes a ventricular output 120 ms after the sensed event at the pulse generator's programmed upper rate limit (150 ppm). This ventricular event is conducted retrogradely to the atrium followed by PCMT.*

mulator). The external stimulator could not sense spontaneous QRS complexes from the chest leads. Therefore, the pacemaker A-V interval was programmed to a value ensuring ventricular (fusion) pacing and the ventricular pulse width was programmed to 1.5 ms to facilitate external sensing of the pacing artifacts. The timing of the premature ventricular stimulus during sinus rhythm was determined by the chosen chest wall stimulation interval ($R\text{-}S_1$) and the programmed A-V interval. The degree of prematurity was limited by the programmed upper rate (Fig. 2), the shortest possible premature interval being 340 ms. The chest wall stimulus had to be of an amplitude which was sensed by the atrial amplifier of the pacemaker but not by the ventricular amplifier. If the chest wall stimulus was only sensed by the ventricular amplifier, all timing sequences were reset and a ventricular output was inhibited. To prevent ventricular sensing of the chest wall stimuli the ventricular sensitivity was sometimes programmed to a less sensitive value.

VVI pacing. The ventricle was paced at different rates in VVI mode and V-A conduction was assessed from the surface ECG (Fig. 3).

Chest wall thump. This was used occasionally to induce ventricular premature beats that could initiate PCMT.

By inducing the Wenckebach response. By increasing the sinus rate (e.g. exercise) and programming a low upper rate the Wenckebach mode was induced when the sinus rate exceeded the programmed upper rate limit. The P-waves and paced ventricular events dissociated and ventriculo-atrial conduction and PCMT were made possible.

When PCMT had been demonstrated by one of

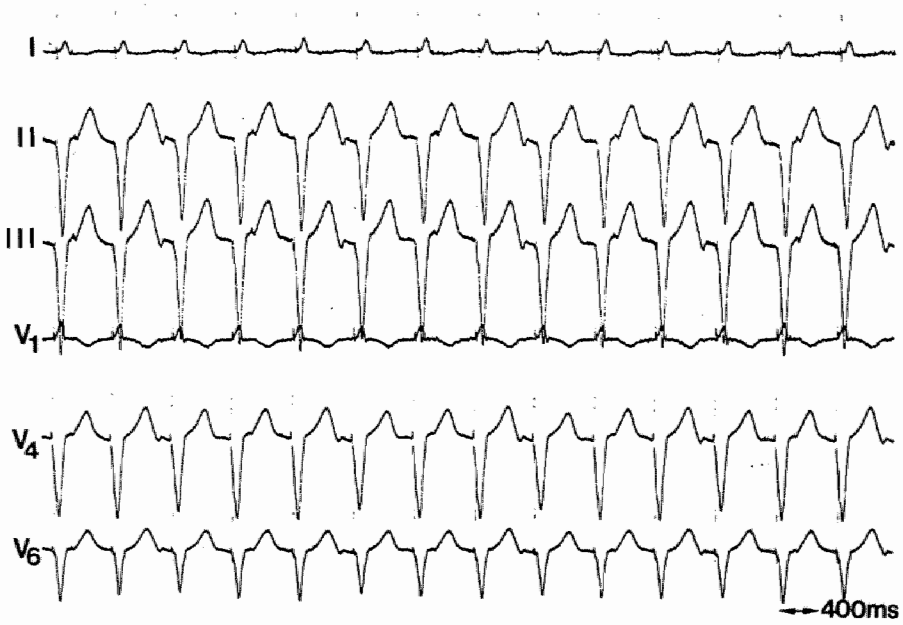

Figure 3. *Simultaneous 6-channel ECG recording showing 3:1 V-A block during VVI pacing at a rate of 100 bpm.*

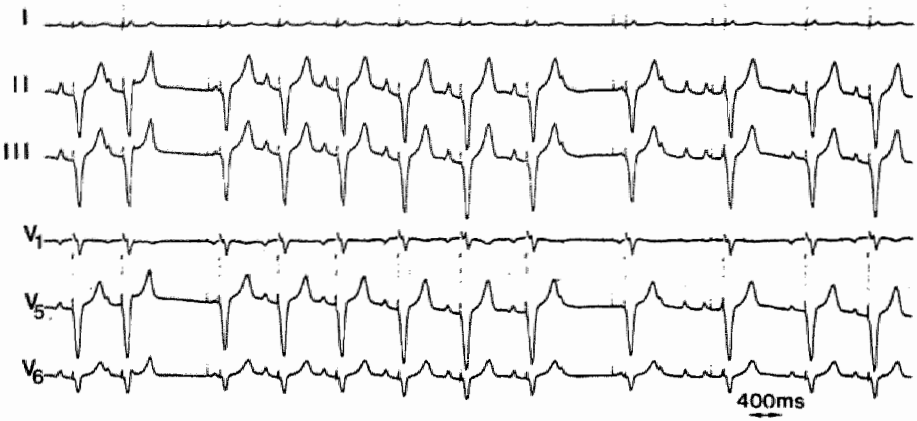

Figure 4. *Simultaneous 6-channel ECG illustrating synchronization to the sinus P-waves, but not to the atrial tachycardia P-waves, at an atrial sensitivity value of 1.5 mV. The difference in P-wave morphology can be seen in lead V_1.*

the above methods, the following measures were tested for their effectiveness in preventing or interrupting PCMT:

Methods To Prevent or Terminate PCMT

Programming a lower atrial sensitivity (higher mV value) (provided that the sinus atrial P-waves are still being sensed). This step prevents or reduces the incidence of synchronization to pectoral muscle artifacts and, hence, the incidence of PCMT.

In Figure 4 the atrial tachycardia P-waves were not sensed, while the sinus P-waves were sensed. Similarly, the retrograde P-waves may not be sensed at a different atrial sensing value while the sinus P-waves are being sensed.

Programming a high upper rate with an appropriately short A-V interval. The right ventricular pacing rate at which retrograde block occurred is determined invasively prior to implant and noninvasively after implantation by VVI pacing at different rates or by chest wall stimulation.

Figure 3 illustrates VVI pacing at 100 bpm with 3:1 V-A block. The first ventricular paced event was probably conducted over a fast retrogradely conducting pathway, the second over a slowly conducting pathway and the third blocked in both pathways. Figure 5 illustrates termination of PCMT at upper rate settings of 100 bpm and 175 bpm respectively in the same patient. At the upper rate of 175 bpm PCMT was terminated more efficiently. The coupling interval of the paced ventricular event after a sensed retrograde P-wave and the tachycardia rate of a PCMT depend upon the ventriculo-atrial conduction time, the programmed A-V interval and the programmed upper rate.

Avoiding Wenckebach behavior by programming a higher upper rate. This may not be possible in the presence of paroxysmal supraventricular tachycardias[4] (atrial tachycardia, atrial flutter and sometimes atrial fibrillation).

Programming a low lower rate. After a ventricular premature beat the pacemakers of the initial Versatrax series escape with only a ventricular output, if the retrogradely conducted P-wave occurs after the atrial refractory period but during the A-V disable period.[3]

If a low lower rate is programmed, the sinus P-wave can become manifest and retrograde conduction prevented, as seen in Fig. 6. Unfortunately, the pulse generator cannot synchronize to this P-wave because the amplifier is still refractory and spontaneous A-V conduction or a P-wave that occurs at a time just before a ventricular escape

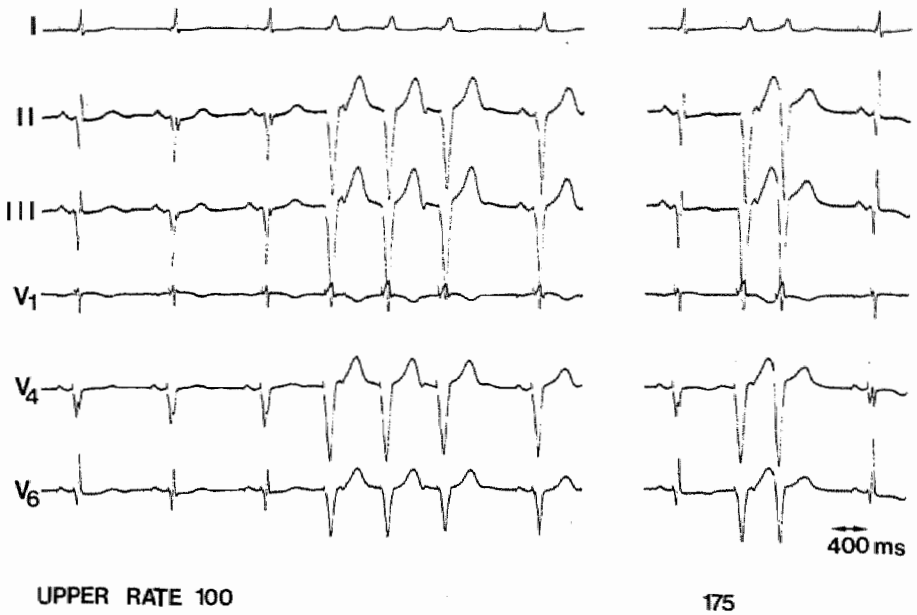

Figure 5. *Termination of PCMT by retrograde block. Left: an upper rate setting of 100 bpm; right: an upper rate of 175 bpm.*

output determines the prevention of V-A conduction.

Medication. Class I drugs like quinidine, procainamide or disopyramide can block V-A conduction and thereby prevent the occurrence of PCMT.

Magnet. If the tachycardias occur infrequently the patient can be given a magnet to terminate PCMT.

Results

If PCMT is sustained it can be terminated by interrupting either the anterograde or the retrograde arm of the artificial reentry circuit. A-V synchronization can be interrupted by placing a magnet over the pulse generator. This renders the device asynchronous. The retrograde arm of the circuit can be interrupted by medication or vagal maneuvers such as carotid sinus massage. In our experience both carotid sinus massage and placing a magnet over the pulse generator have been very effective ways of terminating PCMT.

Before implantation, ventriculo-atrial conduction was demonstrated in 3 of the 11 patients. In all 3, non-invasive induction of PCMT was possible after implantation. After implantation PCMT could be prevented in 2 of these 3 patients through stressing V-A conduction by programming an appropriately short A-V interval and a high upper rate. The third patient had very long retrograde conduction times (320-400 ms). This was one of the first implantations before we realized the significance of long V-A conduction times. His tachycardias were repeatedly and easily initiated by pectoral muscle exercise. The programming of a high atrial sensitivity value made induction of PCMT via pectoral muscle exercise more difficult and also reduced the incidence of PCMT. The latter was determined by careful history taking and repeated 24-hour Holter ECG recording. This patient was given a magnet to terminate PCMT dur-

Figure 6. *Six-channel ECG illustrating induction of PCMT by a VPB at a programmed lower rate value of 80 bpm (right panel). By programming a lower rate of 40 bpm, PCMT was prevented (left panel). The VPBs were induced by chest wall thumps. See text for explanation.*

ing the trial program settings. Because it was not possible to take sufficient precautionary programming measures to completely prevent or terminate PCMT, and because the patient did not want to take any medication, his pacemaker was programmed into another pacing mode (DVI).

After implantation, V-A conduction became apparent in an additional 2 patients with the occurrence of PCMT, which could be induced noninvasively in both. The previously discussed programming measures were not sufficient to control PCMT, but following oral administration of disopyramide, ventriculo-atrial conduction and PCMT could no longer be demonstrated in either patient at several follow-up visits.

Discussion

Our results demonstrate that retrograde conduction is not an absolute contraindication for the use of DDD-pacemakers. Even though all patients received the first-generation Versatrax pulse generator, with the very short atrial refractory period of 155 ms after the ventricular event, only one of the 5 patients showing V-A conduction had to be programmed to DVI because of PCMTs. His tachycardias were mainly induced by atrial sensing of pectoral muscle signals and were characterized by a very long V-A interval. Programming atrial sensitivity to a less sensitive setting reduced the incidence of his PCMTs but did not completely abolish them and the patient refused medication to prevent V-A conduction. The use of coated pacemakers or of an insulating pouch (boot) may be helpful in such cases, but the final solution should come from the application of bipolar systems.

VPBs are the most common cause of PCMTs[5] in patients with retrograde conduction. As explained in the pacemaker description section, the tachycardia is not directly initiated by the VPB but by

the VVI escape mode after the VPB. The obvious solution, therefore, has been to make the pacemaker escape in DVI instead of VVI mode after a VPB with sensed retrograde conduction during the A-V disable period. Although this modification has been shown to be effective in the clinical evaluation,[5] PCMT may still occur when the atrial stimulus of the DVI escape falls in the refractory period of a preceding sinus P-wave. In such a case, the interval between atrial and ventricular events may be sufficiently long to allow V-A conduction.

The V-A conduction properties will determine the ability to induce V-A block (and, hence, prevent or terminate PCMT) by shortening the ventricular stimulation interval. Three factors will determine the prematurity of the ventricular stimulation interval at the onset of PCMT or during PCMT. The spontaneous V-A interval, the programmed A-V interval and the programmed upper rate limit. When V-A block occurs, for instance, at a ventricular rate of 150 bpm (400 ms), and when the V-A time equals 325 ms, V-A block will only be induced by the pacemaker during the PCMT when the A-V interval is programmed shorter than 75 ms (and when the upper rate is programmed to 150 or 175 bpm). The longer the V-A time, the shorter an A-V interval will be required to bring the resulting ventricular stimulus close to the preceding one, which is a must in order to provoke V-A block. It should also be kept in mind that V-A conduction properties are influenced by autonomic tone and medication. In patients with dual V-A pathways, V-A block must be achieved by synchronization to the P-wave with the shorter V-A interval. Once the V-A interval has been prolonged the prematurity of the next atrial synchronized ventricular stimulus will be delayed by the prolonged V-A interval and block will become unlikely, as shown in Fig. 7.

PCMTs can also be induced by the prolongation of the A-V interval that results from the Wenckebach rate-limiting mechanism. The A-V interval, when started by an atrial premature beat or during supraventricular tachycardia, may time out before the upper rate interval has ended. The ventricular stimulus will be given at the end of the upper rate interval and the resulting prolongation of the A-V interval may set the stage for V-A conduction. These considerations indicate, that the upper rate should be programmed sufficiently high to avoid such rate-limiting behavior, especially in patients with retrograde conduction. The presence of paroxysmal supraventricular tachycardias may prevent one from programming a high upper rate.

Apart from the already described techniques to prevent and interrupt PCMTs, future DDD-pacemakers should, by design, avoid this artificial arrhythmia. As mentioned before, they should be bipolar to avoid the A-V dissociation resulting from synchronization to muscle signals. Having a programmable refractory period will prevent most PCMTs, but the ones with a long V-A interval will still be a problem. Simply prolonging the atrial refractory period in these patients defeats the intention of physiological pacing to offer rate responsiveness and A-V synchronization during high sinus rates. Pehrsson[6] and Olson[7] have demonstrated that the 2:1 blocking behavior resulting from long atrial refractory periods has negative hemodynamic consequences. A more acceptable solution can be found in extending the atrial refractory period just once following a VPB. This does not interfere with further synchronization while avoiding sensing of a retrograde P following the VPB. A situation as described previously, where an atrial pace event could occur during the refractory period of the atrium during the DVI escape mode, should be avoided.

In patients with extremely long V-A intervals, when even an automatically extended atrial refractory period or the 340-ms A-V disable in Versatrax would offer no protection, PCMT could be avoided by stimulating the atrium in synchrony with, or slightly after, the VPB. This atrial stimulus would cause concealed antegrade penetration of the V-A pathway,[8] thereby preventing V-A conduction. If the techniques mentioned here fail in some cases, the pacemaker should recognize the possibility that PCMT is present and act accordingly. (Recognition by means of continued upper rate pacing may not be helpful in patients with a long V-A interval, because their PCMT rates will be low, thus requiring the programming of a low upper rate to trigger.) In such circumstances, dropping a P-wave once or simultaneous atrial and ventricular stimulation to cause concealed antegrade conduction[8] may interrupt the tachycardia.

Such features in future DDD pacing systems will, hopefully, make programming considerations and medications to avoid V-A conduction unnecessary.

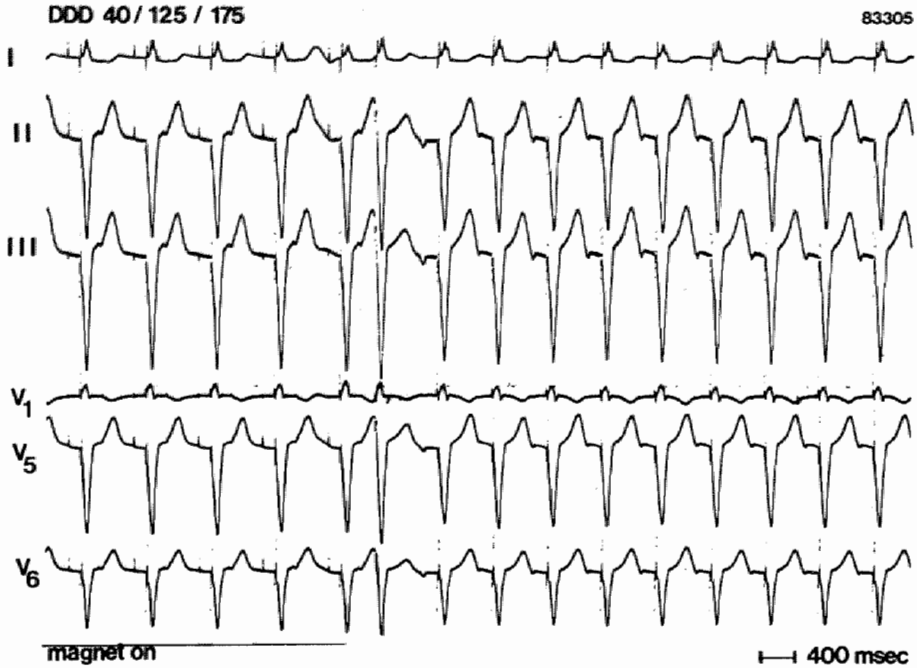

Figure 7. *Six-channel ECG showing the initiation of PCMT in a patient with dual retrograde A-V nodel pathways.*

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Chapter 7

Failure of the Post Ventricular Premature Beat DVI Mode in Preventing Pacemaker Circus Movement Tachycardia.

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Summary

To prevent initiation of pacemaker circus movement tachycardia (PCMT) by a ventricular premature beat(VPB), some DDD pacemakers have been designed to automatically change to the DVI mode (temporarily) after a VPB. In a 72-year-old man with symptomatic A-V conduction disturbances, electrophysiological investigation demonstrated intact V-A conduction with a V-A interval of 260 ms. Following implantation of a DDD pacemaker, PCMTs were sometimes initiated by the DVI mode after the VPB because the atrial stimulus of the temporary DVI mode occurred during the refractory period of the atrium following a preceeding sinus P-wave. The ventricular paced event was conducted retrogradely to the atrium and followed by PCMT. This illustrates that although the DVI mode after a VPB is frequently effective in preventing initiation of PCMT, it does not work under all circumstances. It also suggests that a DDD escape mode after a VPB with an extended atrial refractory period is a better escape mode.

Introduction

Pacemaker circus movement tachycardia (PCMT) (1-4) is common in DDD pacing. Several methods of managing PCMT have been suggested: prolonging the atrial refractory period, omitting a single atrial synchronized ventricular paced event upon recognition of PCMT, bipolar systems (to avoid initiation via pectoral muscle artefacts), and temporary DVI mode pacing after a ventricular premature beat (VPB). This last method has been implemented in several DDD pacemakers. After having sensed a VPB, the device functions in the DVI mode for one or more cycles. The rationale is

that if the VPB is conducted retrogradely to the atrium, it is not sensed. Our case report illustrates that this post-VPB DVI mode is not fool proof in preventing PCMT and that the device should function in the DDD mode rather than the DVI mode after a VPB.

Case report

A Pacesetter AFP model 283 DDD pacemaker was implanted in a 72-year-old man because of symptomatic episodes of total atrio-ventricular(A-V) block. Electrophysiologic investigation demonstrated that ventriculo-atrial (V-A) conduction was intact; the V-A interval was 260 ms,1-to-1 V-A conduction was present to a right ventricular pacing rate of 150 ppm. After implantation, while being monitored, the patient had frequent VPBs. A short atrial refractory period and the DVI response after a VPB was programmed to test the response of the device to these VPBs. PCMT was initiated by the DVI mode after the VPB (Fig 1). This happened because the atrial stimulus of the temporary DVI mode occurred during the refractory period of the atrium after a preceding sinus P-wave (which was not sensed in the DVI mode). This allowed the sinus P-wave to be sufficiently separated from the paced ventricular event to allow the paced ventricular event to be conducted retrogradely to the atrium. A similar situation is shown in figure 1B, except that the atrial paced artefact did not find the atrial tissue refractory and this paced atrial event prevented the initiation of PCMT by creating refractoriness of the V-A conduction system. PCMT was prevented in this patient by programming the atrial refractory period longer than the V-A interval.

Discussion

PCMT can be prevented by prolonging the atrial refractory period after a VPB. However, prolongation of the atrial refractory period beyond the next ventricular event (temporary DVI mode after a VPB) has the disadvantage that the PCMT can be initiated by the paced escape ventricular event. If the atrial refractory period after a VPB is prolonged to a long value and the device remains in the DDD mode, PCMT can be prevented (if the retrograde P-wave occurs during this prolonged refractory period). This prevents initiation, as illustrated in our patient, by either synchronizing to a sensed sinus P-wave or by escaping in the DVI mode. The escape atrial stimulus (after the extended atrial refractory period after the VPB) should occur at a time that would still enable the capture of the atrial stimulus if a preceding atrial event were to occur in the last part of

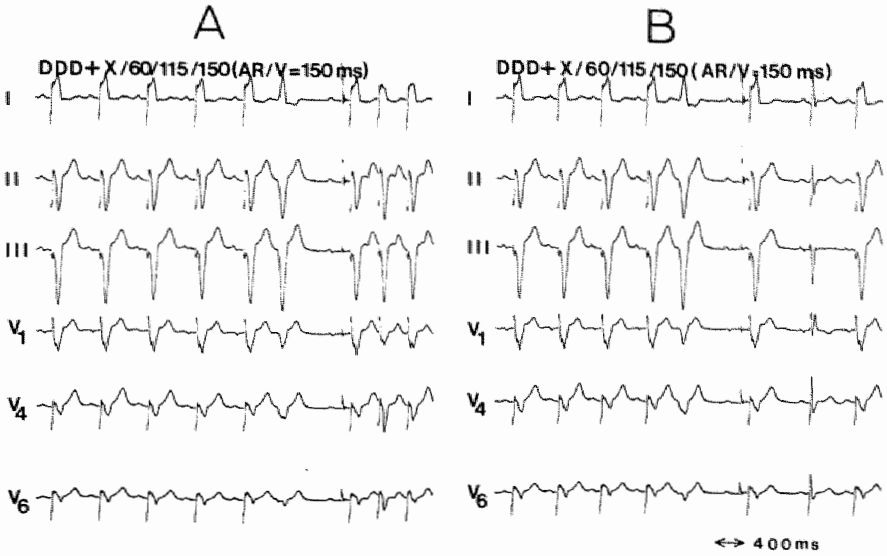

Figure 1A
Simultaneously recorded 6 channel electrocardiogram illustrating the initiation of PCMT by the ventricular escape stimulus after the VPB. The device was functioning in the DVI mode after the VPB, and therefore did not synchronize to the sinus beat after the VPB. The atrial escape stimulus occurred during the refractory period of the atrium. This allowed sufficient seperation of atrial and ventricular events to allow retrograde conduction of the paced ventricular event.

Figure 1B
PCMT is prevented by A-V conduction of the atrial stimulus after the VPB. The programmed pacing mode, lower rate, atrioventricular interval and upper rate are listed. X = temporary DVI mode after a VPB; AR/V = atrial refractory period after a sensed or paced ventricular event.

the atrial refractory period after the VPB.

The DVI mode after a VPB of the Pacesetter and the Medtronic DDD pacemakers is different. Ventricular events are recognized as VPBs by these pacing devices if two ventricular events are sensed without an intervening atrial event. In the Medtronic Versatrax series, the DVI mode is switched off after a sensed or paced ventricular event after the VPB. The Pacesetter (model 283) remains in the DVI mode until atrial pacing occurs. Thus, the P-wave after the conducted QRS-complex in figure 2 is not sensed by the pacemaker. Synchronization to P-waves is restored after the atrial escape stimulus. The device could therefore remain in the DVI mode for a long time if the escape rate or underlying heart rate were higher than the programmed atrial escape interval. For example, if the escape rate is 40 beats per min = 1500 ms, the A-V interval = 150 ms, the atrial escape

interval will be 1500-150 = 1350 ms = ± 44 beats per min. This means that if the underlying rhythm is higher than 44 beats per min the pacemaker will remain in the DVI mode after a VPB. The DVI mode after a VPB is frequently an effective means of preventing the initiation of PCMT by VPBs (5). However, the present report illustrates that it will not prevent PCMT under all circumstances, and a DDD escape mode combined with a long atrial refractory period after a VPB is a better method.

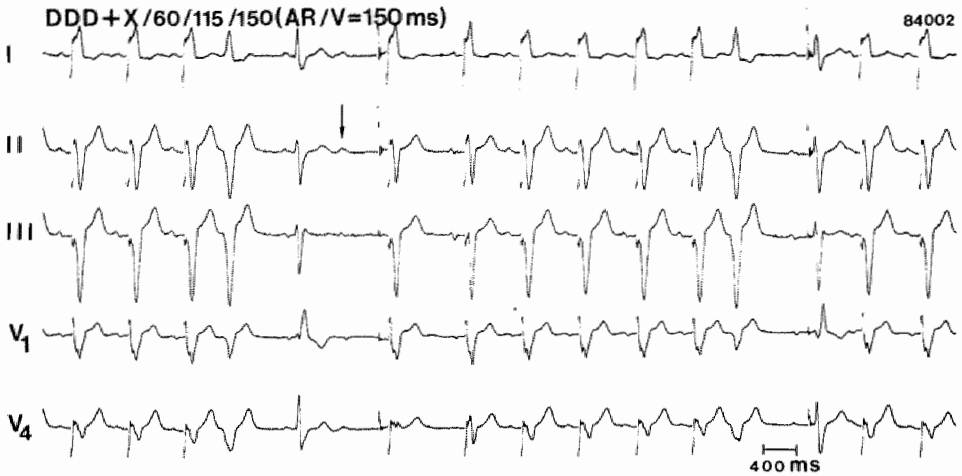

Figure 2
In this registration the sinus beat (arrow) is not sensed because the pacemaker is still in the DVI mode after the VPB. See text for explanation.
The programmed pacing mode, lower rate, atrioventricular interval and upper rate are noted in this order. X = temporary DVI mode after a VPB; AR/V = atrial refractory period after a sensed or paced ventricular event.

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Chapter 8

Do All Pacemakers Need Anti-Bradycardia as well as Anti-Tachycardia Pacing Features?

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Summary

Anti-bradycardia pacemakers which have additional anti-tachycardia pacing modes are currently being developed. The purpose of this study was to determine the need for all pacemakers to have both anti-tachycardia and anti-bradycardia pacing features. Medical and pacemaker files of 212 patients who visit our pacemaker clinic regularly, were reviewed retrospectively. One hundred and ninety three anti-bradycardia pacemakers were implanted for symptomatic bradycardia, 2 anti-bradycardia pacemakers to prevent, as well as to terminate tachycardia, 1 anti-bradycardia pacemaker to terminate tachycardia and 16 anti-tachycardia pacemakers to terminate paroxysmal episodes of drug resistant tachycardia. Four of 193 patients (2%) who received a pacemaker to prevent bradycardia, had re-entry tachycardias. Two were A-V nodal, 1 made use of an accessory pathway and 1 was an idiopathic ventricular tachycardia. The latter was not suited for pacing. In all patients tachycardia was adequately controlled by drug therapy. None of the 19 patients, who had a pacemaker implanted for symptomatic tachycardia had symptomatic episodes of bradycardia before implantation. In the group of 16 patients receiving an anti-tachycardia pacemaker, 1 patient consistently required 1 or 2 back up stimuli at 50 ppm after termination of tachycardia while 2 others used this feature occasionally.

Conclusions:

1) Supplementary anti-tachycardia pacing features would not have formed an essential part in the treatment of any of the patients who received a pacemaker for symptomatic bradycardia 2) Simple bradycardia support pacing was occasionally required after termination of tachycardia.

Introduction

The role of programmed stimulation of the heart in the study of tachycardias was first reported in 1967 (1,2). This was soon followed by the development of implantable pacing systems (3-9) for long term treatment of re-entry tachycardias by timed stimuli. Two basic types of pacemaker systems are available today; one for bradycardia support and one to interrupt paroxysmal re-entrant tachycardias. Both types of systems have undergone considerable technological refinement and sophistication in the last years. Anti-bradycardia pacemakers, which have additional anti-tachycardia pacing modes are currently being developed. Soon these additional features will be advertised as being of great benefit to the pacemaker population. The purpose of this study was to determine the need for all pacemakers to have anti-bradycardia as well as anti-tachycardia pacing capabilities.

Methods

Medical and pacemaker files of the 212 patients who visit our pacemaker clinic regularly were reviewed retrospectively. (See table I). Of these 212 patients, 16 received an anti-tachycardia pacemaker to terminate their drug resistant paroxysmal episodes of tachycardia. The remaining patients received anti-bradycardia pacemakers which did not have anti-tachycardia pacing features; the indication being, symptomatic bradycardia in 193 patients, tachycardia prevention as well as termination in 2 patients, and tachycardia termination in one patient.

Table I

Patient Population

193 ABP	Symptomatic Bradycardia
2 ABP	Symptomatic Tachycardia (Prevention and Termination)
1 ABP	Symptomatic Tachycardia (Termination)
16 ATP	Symptomatic Tachycardia (Termination)
212	

ABP = Anti-bradycardia pacemaker.
ATP = Anti-tachycardia pacemaker.

Eighty-one of the 193 patients who received an anti-bradycardia pacemaker were men and 112 were women. Their ages ranged from 24 to 97 years (mean 74). Of the 193 bradycardia support pacemakers implanted, 89 were implanted because of sinus node disease, 102 because of A-V conduction disturbances (In 3 patients the AV conduction system was interrupted to control tachycardia. In 2 patients it was performed surgically and in one by means of an electric shock delivered locally via a catheter) and 2 for the prevention of tachycardia in patients with a prolonged Q-T interval.

Two patients received a bradycardia support pacemaker to prevent and to terminate symptomatic tachycardia. One patient had a WPW syndrome, and received a dual chamber pacemaker system for prevention and automatic termination of his drug resistant nearly continuous circus movement tachycardia. The DAT and the DDD pacing modes with a very short A-V interval of 25 ms prevented and terminated circus movement tachycardia by means of artificial pre-excitation of the re-entry circuit (10). The other patient also had a WPW syndrome with very frequent episodes of tachycardia. This patient received a fixed rate ventricular pacemaker. Pacing the ventricle at 90 ppm changed the electrophysiological properties sufficiently to significantly reduce the number of episodes of tachycardia. If tachycardia did occur it was easily terminated by means of underdrive stimulation at the set rate of 90 ppm.

In one patient with a WPW syndrome and circus movement tachycardia an anti-bradycardia pacemaker was implanted and used to terminate tachycardia by means of underdrive stimulation. Underdrive pacing was achieved by placing a magnet over the implanted demand pacemaker and converting it to an asynchronous magnet mode.

Sixteen patients (8 women, 8 men) received a pacemaker to terminate their paroxysmal re-entrant tachycardias. In all patients multiple long term oral drug regimens failed to control their arrhythmias and they had been admitted frequently to hospital for conversion of arrhythmias. These patients were 19 to 67 years old (mean 43). Four patients had ventricular tachycardia, 4 had atrioventricular nodal re-entrant tachycardia and eight had tachycardias due to an accessory pathway.

Results

As can be seen in Table II, 4 of the 193 patients (2%) who had an anti-bradycardia pacemaker implanted, had re-entry tachycardias. Two of the tachycardias were A-V nodal, one made use of a concealed accessory pathway and one was an idiopathic ventricular tachycardia. The ventricular tachycardia was not suited for pacing therapy. In all 4 patients tachycardia was adequately controlled by drug therapy. Anti-tachycardia pacing was therefore an optional form of therapy in 1.5% of patients.
Of the three patients who received an anti-bradycardia pacemaker for prevention and/or termination of tachycardia, none required a pacemaker for symptomatic bradycardia.

Table II

Incidence of paroxysmal re-entry tachycardias in 193 patients who received a pacemaker for symptomatic bradycardia.

Re-entry tachycardia:		Suitable for anti-tachycardia pacing.
AV nodal tachy	2	2
Circus movement	1	1
Idiopathic VT	1	0
	–	–
	4	3
	2%	1.5%

Of the 16 patients with an anti-tachycardia pacemaker, none required a pacemaker for symptomatic bradycardia. During follow up, the bradycardia back up pacing feature of 50 ppm was consistently used in one patient for one or two beats following termination of tachycardia and occasionally in 2 other patients. One patient was bothered by the bradycardia support pacing (which he did not need) at night.

Discussion

Supplementary anti-tachycardia pacing features would not have formed an essential part in the treatment of any of the 193 patients with symptomatic bradycardia. It would have been an optional form of therapy in 1.5% of these patients.

In the relatively small group of patients with an anti-tachycardia pacemaker, only 3 patients may have benefitted from a simple bradycardia support function after tachycardia termination. However, in this group of patients a unifocal programmable form of bradycardia support was sufficient. Anti-tachycardia pacemakers should have a bradycardia back up pacing capability and the rate should preferably be programmable.

This retrospective study indicates that anti-tachycardia pacing capabilities were not essential in any of the anti-bradycardia pacemakers. Nor was physiological back up pacing required in the small, selected group of patients, in whom the primary indication for pacing was to treat symptomatic paroxysmal episodes of re-entry tachycardia which were resistant to conventional drug therapy.

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Summary

Re-entry tachycardia forms the basis of this thesis. The treatment of re-entry tachycardia by means of a pacemaker is a simple, effective alternative to drug therapy and surgery when these are ineffective, inappropriate or undesirable. The application of certain "physiological" pacing modes in anti-bradycardia pacemakers has created a completely new artificial form of re-entry tachycardia (Pacemaker circus movement tachycardia). This thesis deals with both of these aspects. The application of anti-tachycardia pacemakers for supraventricular and ventricular tachycardias is discussed with emphasis on clinical results, safety and versatility. Pacemaker circus movement tachycardias are analysed in terms of their occurrence, mechanism, evaluation and prevention.

Pacemaker history and mechanisms of tachycardia, especially re-entry tachycardia are reviewed in chapter one. Furthermore the various pacing techniques with implantable anti-tachycardia devices currently in clinical use are discussed. Finally, the mechanism of pacemaker circus movement tachycardia in DDD pacemakers is discussed. Chapter two describes the combined experience from the departments of Cardiology in Liege and Maastricht, with a versatile pacemaker system for termination of supraventricular and ventricular tachycardias. A unique case report in chapter three illustrates the need for pacemakers to be able to cope with changes in tachycardia termination window, demonstrating that flexible anti-tachycardia pacemaker systems are required. In chapter four, three patients suffering from pacemaker related tachycardias are reported. In the first case report, an artificial (pacemaker) circus movement tachycardia is analysed. The second case report illustrates the importance of various factors that have to be considered before choosing an anti-tachycardia pacing system, while the third describes a tachycardia resulting from a technical problem in one of the early DDD devices.

Our experience with ventriculo-atrial conduction and noninvasive evaluation of ventriculo-atrial conduction and initiation of pacemaker circus movement tachycardia in the first generation Medtronic Versatrax DDD pacemaker is presented in the fifth chapter. Chapter six describes how the problem of pacemaker circus movement tachycardia was managed in our patients. Possible solutions for treatment and prevention of this problem are discussed. Chapter seven illustrates that the DVI mode after a ventricular premature beat does not always prevent initiation of pacemaker circus movement tachycardia. A retrospective review of all our

pacemaker patients is given in chapter eight to determine the need for pacemakers to have anti-tachycardia features next to their ability to treat bradycardia.

Samenvatting

In dit proefschrift nemen de re-entry tachycardieën een centrale plaats in. Als medicamenteuze of chirurgische behandeling van de re-entry tachycardieën niet helpt, niet toepasbaar of ongewenst is, kan een pacemaker een simpel, efficiënt alternatief zijn. Echter het gebruik van bepaalde "fysiologische" vormen van stimulatie met behulp van anti-bradycardie pacemakers heeft een nieuwe artificiële vorm van re-entry tachycardie geïntroduceerd, de zogenaamde pacemaker cirkel tachycardieën. In dit proefschrift wordt op beide aspecten ingegaan. De wijze van toepassing van anti-tachycardie pacemakers voor supraventriculaire en ventriculaire tachycardieën wordt besproken en de klinische resultaten worden gepresenteerd. Er wordt aangetoond dat deze pacemakers veilig en veelzijdig zijn.
Daarnaast wordt de incidentie, het mechanisme, onderzoek naar en de preventie van pacemaker cirkel tachycardieën beschreven. In hoofdstuk 1 wordt een overzicht gegeven van de geschiedenis van de pacemaker. Tevens wordt op de mechanismen van tachycardieën, in het bijzonder de re-entry tachycardieën, ingegaan. Verder worden verschillende pacing technieken besproken zoals die worden gebruikt bij de huidige in de kliniek gebruikte anti-tachycardie pacemakers. Tenslotte wordt het mechanisme van pacemaker cirkel tachycardieën besproken. Hoofdstuk 2 geeft een overzicht van de gezamenlijke ervaringen van de cardiologische klinieken in Luik en Maastricht met een "veelzijdige" (door de patient zelf te activeren) pacemaker voor het beëindigen van supra-ventriculaire en ventriculaire tachycardieën. In hoofdstuk 3 wordt aan de hand van een bijzonder geval aangetoond, dat het noodzakelijk is om de wijze van het beëindigen van de tachycardie te kunnen variëren. Dit geeft aan hoe belangrijk het is dat een anti-tachycardie pacemakersysteem flexibel is.
In hoofdstuk 4 worden drie patiënten besproken met een geheel of mede door de pacemaker veroorzaakte tachycardie. De eerste patiënt heeft een artificiële (pacemaker) cirkel tachycardie. Naar aanleiding van de tweede patiënt wordt gewezen op een aantal factoren die van belang zijn bij de keuze van het anti-tachycardie pacemakersysteem. Bij de derde patiënt is de tachycardie ontstaan door een technisch probleem in de "eerste generatie" DDD pacemakers.
Onze ervaring met ventriculo-atriale geleiding wordt in hoofdstuk 5 weergegeven. Het niet-invasief evalueren van de retrograde geleiding en het initiëren van pacemaker cirkel tachycardieën ten gevolge van retrograde

geleiding wordt beschreven zoals gevonden bij de "eerste generatie" Medtronic Versatrax DDD pacemakers. Hoofdstuk 6 beschrijft onze aanpak van pacemaker cirkel tachycardieën. Methoden ter behandeling c.q. preventie van dit probleem worden besproken. Hoofdstuk 7 illustreert dat de DVI mode na een kamer extrasystole niet altijd het initiëren van pacemaker cirkel tachycardieën voorkomt. Hoofdstuk 8 geeft een retrospectief overzicht van al onze pacemakerpatiënten. Hierbij wordt ingegaan op de vraag of anti-bradycardie pacemakers ook van anti-tachycardie eigenschappen moeten worden voorzien.

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