We recommend that His bundle electrograms be obtained for patients whose history portends of Stokes-Adams attacks and that intracardiac electrophysiological study should be seriously considered in any patient with transient postoperative CHB or electrocardiograms demonstrating bifascicular block or progressively lengthening P-R intervals. Our results and previous reports of late postoperative CHB in patients whose electrocardiograms did not show bifascicular block present some rationale for follow-up electrophysiological studies in all children following ventricular defect repair. At the present time, we cannot recommend the prophylactic use of permanent pacemakers in asymptomatic patients with evidence of trifascicular damage, but this group certainly deserves very careful follow-up and continued study.

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3. Iizkawa T, Clarke M, Trusler GA: Late complete trifascicular heart block resulting from cardiac surgery. (abstr) Circulation 44: (suppl II) II-151, 1971

Temporary Inhibition of Permanently Implanted Demand Pacemakers

Pervaize Latif, M.D., and Gordon A. Ewy, M.D.

SUMMARY Temporary inhibition of permanently implanted demand pacemakers has been previously described. Demand pacemakers may be inhibited by wave a magnet over the region of the pacemaker generator or by chest wall stimulation. The former may not inhibit most of the bipolar pacemakers, whereas the latter may be time consuming and may cause patient discomfort.

Another method is described which utilized a commercially available Cordis Omnicor Programmer, Model 166-B, to temporarily inhibit bipolar and unipolar pacemakers. By placing the programmer over the skin where the pacemaker generator is implanted and/or over the area of the subcutaneous perivenous lead and activating the programmer multiple times at a rate faster than the pacing rate, the demand pacemakers are inhibited. After testing the efficacy in vitro, the method was successfully tried on 45 patients. Fifteen of these patients had unipolar pacemakers. Pacemakers marketed by Medtronic, Cordis, Starr-Edwards, C.P.I., and Arco were tested.

Temporary inhibition of permanent demand pacemakers is desirable under various clinical situations. The method herein described has the advantages of being simple, quick, painless, and is effective for both unipolar and bipolar pacemakers.

TEMPORARY INHIBITION OF PERMANENTLY IMPLANTED DEMAND PACEMAKERS, so that one can record the patient's intrinsic nonpaced electrocardiogram, is desirable under a variety of clinical situations. Three basic methods have been described.1-4 Chest wall stimulation with an external pacemaker or waving a magnet over the pacemaker generator are the two methods most commonly used. The third utilized electromagnetic impulses generated by the Medtronic Model 5855 external rate control transmitter.5, 6 Chest wall stimulation is time consuming since various areas of the chest may have to be paced to determine the precise position for inhibition.1, 2 In addition, chest wall stimulation causes some discomfort to the patient.
Figure 1. Top — Cordis Omnicor Programmer Model 166-B. Bottom — Close-up view of control dials. The three dials are used to determine the rate, current output and pacemaker type.

When higher energy is needed to inhibit the pacemaker,1 waving a magnet over the generator does not consistently result in pacemaker inhibition.4 The Medtronic Model 5855 external rate control transmitter is no longer commercially available.

This paper describes another method for inhibiting both unipolar and bipolar pacemakers by transmitting electromagnetic impulses with a commercially available unit. This noninvasive method is simple, safe, quick, painless and effective.

Method

A Cordis Omnicor Programmer Model 166-B (henceforth called programmer) was used. This programmer (fig. 1) is designed to change the rate and/or current output of any of the four Cordis Omnicor programmable pacemakers at or after implantation by sending pulsatile magnetic waves at preselected frequency into the pacemaker generator.7,8 Three dials are available on the programmer to alter the frequency of the magnetic pulsations generated (fig. 1).

It was postulated that the impulses sent by the programmer were of sufficient strength to cause pacemaker inhibition. Accordingly, the following in vitro experiments were conducted. A Medtronic bipolar pacemaker, Model 5942 with intact pervenous lead was used as the initial test instrument. The proximal and distal electrodes were attached to the right and left arm leads of the electrocardiogram. The right and left leg leads of the electrocardiogram were attached to a normal subject (P.L.) and a lead II electrocardiogram obtained at a speed of 25 mm/sec. This produced pacing spikes 9 to 10 mm in height on the electrocardiogram. The Cordis programmer was then placed over the pacemaker generator or pervenous lead and the activating button was pushed and released several times in succession at a rate slightly faster than the pacemaker rate. This resulted in pacemaker inhibition as illustrated in figure 2. Tests of two Medtronic Model 5950 pulse generators revealed that they could be inhibited when the programmer was placed over the pervenous lead.

Once it was established that the in vitro method of pacemaker inhibition was effective and that the programmed pulsatile magnet field caused only transient inhibition of pacemaker function, it was utilized in the Pacemaker Clinic on 45 patients. The programmer was placed on the skin over the implanted pacemaker generator. In general, it was centered near the connection of the pervenous lead and the generator. Thus, the programmer was placed over the generator and/or proximal pervenous lead. The pacemaker make, model, mode of pacing, and number of pacemakers

Table 1. Types of Pacemakers Inhibited

<table>
<thead>
<tr>
<th>Make</th>
<th>Model</th>
<th>Mode</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medtronic</td>
<td>5950</td>
<td>VVI*</td>
<td>12</td>
</tr>
<tr>
<td>Medtronic</td>
<td>5944</td>
<td>VVI</td>
<td>10</td>
</tr>
<tr>
<td>Medtronic</td>
<td>5942</td>
<td>VVI</td>
<td>8</td>
</tr>
<tr>
<td>Medtronic</td>
<td>5951</td>
<td>VVI</td>
<td>2</td>
</tr>
<tr>
<td>Medtronic</td>
<td>5945</td>
<td>VVI</td>
<td>1</td>
</tr>
<tr>
<td>Cordis</td>
<td>Omni-Stanigor</td>
<td>VVI</td>
<td>6</td>
</tr>
<tr>
<td>Arco</td>
<td>Li 2D</td>
<td>VVI</td>
<td>3</td>
</tr>
<tr>
<td>Starr-Edwards</td>
<td>8114</td>
<td>VVT**</td>
<td>2</td>
</tr>
<tr>
<td>C.P.I.</td>
<td>Maxilith-301</td>
<td>VVI</td>
<td>1</td>
</tr>
</tbody>
</table>

*VVI = ventricular inhibited
**VVT = ventricular triggered

Table 2. Type of Electrodes Used in Pacemakers

<table>
<thead>
<tr>
<th>Type</th>
<th>Number</th>
<th>Endocardial</th>
<th>Epicardial</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unipolar</td>
<td>15</td>
<td>12</td>
<td>3</td>
</tr>
<tr>
<td>Bipolar</td>
<td>30</td>
<td>28</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>45</td>
<td>40</td>
<td>5</td>
</tr>
</tbody>
</table>
inhibited are summarized in Table 1. Table 2 shows the type of electrode used in these pacemakers. The duration of implantation varied from one to 169 weeks.

Figure 3 shows that each time the programmer was activated, the electrocardiogram registered an artifact which was distinctly different from the pacemaker artifact. Lead III had the minimum artifact when the pacemaker was implanted in the right upper chest. One would postulate that lead II would have the minimum artifact if the pulse generator were implanted in the left upper chest. Figures 4 and 5 illustrate how a complete 12 lead electrocardiogram can be recorded with a few paced and inhibited impulses on each lead. The Cordis Omni-Stanicor (programmable) pacemakers were inhibited by setting the dials at the appropriate model, rate and current output desired for that particular patient. All of the nonprogrammable pacemakers tested could be inhibited by setting the dials in any manner.

Discussion

Temporary inhibition of permanent pacemaker spikes is desirable under a variety of clinical situations. Examples are to document patient's own intrinsic rhythm, to document pacemaker induced ST-T wave changes, to diagnose myocardial infarction, to diagnose and study various arrhythmias, to determine the demand capability of pacemakers, and to inhibit a runaway pacemaker.

The method described herein has definite advantages over the other methods presently in use. Voukydis and associates reported that by waving a magnet over the implanted generator they could inhibit Stanicor pacemakers (Cordis unipolar ventricular inhibited type), but were unable to inhibit bipolar or unipolar pacemakers marketed by Medtronic and Starr-Edwards Laboratories. The magnet has the additional disadvantage of distorting the electrocardiogram baseline in multiple leads. Chest wall stimulation...
can inhibit both unipolar and bipolar pacemakers. However, at times, extensive chest wall exploration is required to find the proper positions to cause inhibition of the pacemaker. Annoying chest wall stimulation may occur when it is necessary to use higher energy to inhibit the pacemaker. Center and associates have described another method of inhibiting unipolar ventricular triggered (VVT) pacemaker using hypodermic needles. This method was used only with the unipolar VVT pacemakers. It is an invasive technique and has the potential hazard of damaging the lead sleeve and/or boot, or introducing infection. The final method of inhibiting pacemakers utilizes electromagnetic impulses. The Medtronic Model 5855 external rate control transmitter had been used to inhibit pacemakers, but has been obsolete since 1972 and hence is no longer available for pacemaker inhibition. Medtronic, Inc. presently has a Model 1700 pulse generator inhibitor for investigational use only. Their manual states it is for use with Medtronic Model 5950 and 5951 pulse generators only.

The exact mechanism by which the programmer inhibits demand pacemaker generators is unknown. One possibility is that the electromagnetic field temporarily closes or opens the magnetic reed switch in the pulse generator. Suppression is then explained in terms of changes in the electrochemical potential between the two electrodes. Each time the reed switch closes, this potential difference is shorted out and the pacemaker senses this voltage change. If this is the mechanism by which the pulse generator is inhibited, one could postulate that a pacemaker with malfunction of the magnetic reed switch would not be inhibited. Most difficulties in activating a normally functioning reed switch with a magnet are related to the position of the magnet or insufficient strength of the magnetic field. This occurs in obese patients and in those with pulse generators implanted below the pectoralis major. Again, one would postulate that in these clinical situations, or if the batteries of the programmer were insufficiently charged, the electromagnetic impulses of the programmer might likewise fail to inhibit a pulse generator. The second postulated mechanism for pacemaker pulse generators inhibition by the programmer is the induction of a voltage spike in the coiled wires of the pervenous leads by the electromagnetic impulse. This theory is supported by our observation that two Medtronic 5950 generators tested in vitro could not be inhibited by placing the programmer directly over the generator, but were inhibited when the programmer was placed over the pervenous lead.

The use of the programmer for inhibition is a noninvasive, simple, rapid and effective method that inhibits both unipolar and bipolar demand pacemakers.

Addendum

Since this article was accepted for publication we have encountered some Medtronic 5950 demand pacemakers that could not be inhibited with the Cordis programmer. These pacemakers do not have amplifiers before the sensing mechanism and thus may require stronger electromagnetic fields than can be generated by the Cordis programmer — especially as the batteries age. All of the Medtronic 5950 pacemakers could be inhibited with a Medtronic 1700 external pacemaker inhibitor.

Acknowledgment

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References

QUANTIFICATION OF ORTHOGONAL ECG/Kan et al.

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Quantification of the Normal Frank and McFee-Parungao Orthogonal Electrocardiogram at Ages Two to Ten Years

JEAN S. KAN, M.D., JEROME LIEBMAN, M.D., MOO HEE LEE, M.D., AND ALLAN WHITNEY, M.S.

SUMMARY A quantification of the Frank and McFee-Parungao orthogonal electrocardiogram of 175 normal children, ages 2-10 years is presented. There are significant differences in age groups 2-5 and 6-10. The QRS voltages of the younger group demonstrate 1) greater Z anterior, 2) greater initial X right, 3) greater terminal X right, 4) smaller Y inferior, and 5) the T vector is oriented more posteriorly.

There are significant differences between the two lead systems. 1) Frank spatial voltages are 70% of those of McFee. 2) Frank X left is 70% of McFee, but terminal X right is 75% and initial X right is 60% of McFee. 3) Frank Y inferior is 78% of McFee, but initial Y superior is 83% and terminal Y superior 61% of McFee. 4) Frank Z anterior is 64% and Frank Z posterior is 70% of McFee. 5) In terms of ratios the Frank is relatively less inferior, about equally posterior and more terminally right. 6) The T vector is more posterior in Frank than McFee.

STANDARD ELECTROCARDIOGRAPHY is known to be relatively inaccurate, yet it remains the system of choice for recording the summation of electrical potentials on the surface of the human. The distortion between the myocardial cell and the surface is considerable for many reasons; and this is so with any surface technique. However, there is much theoretical and practical evidence that orthogonal "dipolar" electrocardiography is less distorted and can better reflect cardiac events than does standard electrocardiography. Therefore, it is necessary to continue to collect a detailed normal data base from various age groups utilizing the best available practical lead systems. The Frank and McFee-Parungao orthogonal electrocardiographic lead systems, on both theoretical and practical bases, appear to have the best chance of becoming standard. Only limited data are available for adults and children.

We have published a very detailed quantification of both the Frank and McFee-Parungao orthogonal electrocardiographic systems from age two to 19 years. That study included only 105 children, but it provided a model for complete linear, circular and spherical quantitation and statistical analysis of electrocardiographic data. More recently published was an analysis of 166 adolescents between the ages of 11 and 19 years, which included 57 subjects from the previous report. The present study is of 175 children, ages two to ten years, including 48 of the original 105.

Material and Methods

We studied 175 normal children with an approximately equal number of blacks and whites.

The Frank and McFee vectorcardiograms were obtained using methods previously described with the subjects in the supine position. The Hart PV3 vectorcardiograph was used for the first 48 children with the vector trace interrupted every 0.002 sec. A specially constructed Electronics for Medicine (E for M) vectorcardiograph was utilized for the next 127 subjects. Simultaneous X, Y, Z scalars could be taken alone or with any two loops (horizontal, sagittal, frontal). With the E for M machine, the vector trace was interrupted both every 0.002 sec for double loops and 0.001 sec for single loops. The T wave was analyzed only for the maximal planar and spatial vectors. The P wave was not measured. The data were analyzed with the aid of an IBM
Temporary inhibition of permanently implanted demand pacemakers.
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