Strategy for Safe Performance of Extrathoracic Magnetic Resonance Imaging at 1.5 Tesla in the Presence of Cardiac Pacemakers in Non–Pacemaker-Dependent Patients
A Prospective Study With 115 Examinations

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Background—The purpose of the present study was to evaluate a strategy for safe performance of extrathoracic magnetic resonance imaging (MRI) in non–pacemaker-dependent patients with cardiac pacemakers.

Methods and Results—Inclusion criteria were presence of a cardiac pacemaker and urgent clinical need for an MRI examination. Pacemaker-dependent patients and those requiring examinations of the thoracic region were excluded. The study group consisted of 82 pacemaker patients who underwent a total of 115 MRI examinations at 1.5T. To minimize radiofrequency-related lead heating, the specific absorption rate was limited to 1.5 W/kg. All pacemakers were reprogrammed before MRI: If heart rate was <60 bpm, the asynchronous mode was programmed to avoid magnetic resonance (MR)–induced inhibition; if heart rate was >60 bpm, sense-only mode was used to avoid MR-induced competitive pacing and potential proarrhythmia. Patients were monitored with ECG and pulse oximetry. All pacemakers were interrogated immediately before and after the MRI examination and after 3 months, including measurement of pacing capture threshold (PCT) and serum troponin I levels. All MR examinations were completed safely. Inhibition of pacemaker output or induction of arrhythmias was not observed. PCT increased significantly from pre- to post-MRI (P=0.017). In 2 of 195 leads, an increase in PCT was only detected at follow-up. In 4 of 114 examinations, troponin increased from a normal baseline value to above normal after MRI, and in 1 case (troponin pre-MRI 0.02 ng/mL, post-MRI 0.16 ng/mL), this increase was associated with a significant increase in PCT.

Conclusions—Extrathoracic MRI of non–pacemaker-dependent patients can be performed with an acceptable risk-benefit ratio under controlled conditions and by taking both MR- and pacemaker-related precautions. (Circulation. 2006;114: 1285-1292.)

Key Words: magnetic resonance imaging □ pacemakers □ safety □ imaging

The presence of a cardiac pacemaker is currently considered an absolute contraindication to magnetic resonance imaging (MRI), and most patients with pacemakers are excluded from having MRI. In 2002, there were approximately 2.4 million patients in the United States with cardiac pacemakers, and this number is growing by 80 000 annually. A previous study has shown that MRI is indicated in 17% of all patients with pacemakers within 12 months of device placement,1 which demonstrates the need for a practical, safe approach for performing MRI on pacemaker patients.

Editorial p 1232
Clinical Perspective p 1292

The aim of the present study was to develop a strategy for safe performance of MRI at 1.5T, which included exclusion of pacemaker-dependent patients and those requiring imaging of the thorax, restriction of specific absorption rate (SAR) values to minimize the risk of lead heating, and pacemaker reprogramming to avoid interference from time-varying gradient fields. The safety of this approach was then evaluated in a large group of pacemaker patients, including assessment of
TABLE 1. Inclusion and Exclusion Criteria for Patients With Permanent Pacemakers

<table>
<thead>
<tr>
<th>Inclusion criteria</th>
<th>Exclusion criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urgent need for an MRI examination</td>
<td>Presence of a Medtronic pacemaker system manufactured between 1993 and 2004</td>
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<tr>
<td>Presence of a Medtronic pacemaker system manufactured between 1993 and 2004</td>
<td>Stable pacemaker physical parameters</td>
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<tr>
<td>Stable pacemaker physical parameters</td>
<td>Battery voltage &gt;2.7 V</td>
</tr>
<tr>
<td>Battery impedance &lt;2000 Ω</td>
<td>Battery estimated remaining lifetime &gt;6 months</td>
</tr>
<tr>
<td>Lead impedances 200 to 2000 Ω</td>
<td>Stable pacing parameters</td>
</tr>
<tr>
<td>Stable pacing parameters</td>
<td>PCT &lt;2.5 V at a pulse duration of 0.4 ms</td>
</tr>
<tr>
<td>Sensing &gt;5 mV</td>
<td>Minimum 3 months since pacemaker and lead implantation</td>
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</table>

Potential myocardial thermal injury by measuring serum troponin I and pacing capture thresholds and performance of a 3-month follow-up to evaluate long-term effects.

**Methods**

**Study Subjects**

A total of 103 consecutive patients with a pacemaker and an urgent clinical indication for MRI were prospectively enrolled. Inclusion and exclusion criteria for the present study are shown in Table 1. This study included only pacemakers from a single manufacturer (Medtronic, Minneapolis, Minn). Pacemakers eligible to be included were single, dual, and biventricular models manufactured between 1993 and 2004 (Legend II, Elite, Prodigy, Minix, Minuet, Sigma, Thera, Kappa, and InSync).

The study design is summarized in Table 2. The institutional review board of the relevant institution approved the study protocol. Signed informed consent was obtained from all subjects, who were counseled about the potential risks of MRI, including irreversible damage to pacemaker components or the system integrity, thermal injuries, and pacemaker malfunction potentially leading to death.

**MRI System and Imaging Protocol**

The maximum SAR value was limited to 1.5 W/kg. Exposure to the static magnetic field was limited to 45 minutes and total active scan time to 30 minutes. Pulse sequences that would have violated the SAR limit were not used or were modified before use. Studies of the chest, including the thoracic spine, heart, and breasts, with full exposure of the pacemaker/lead loop to the radiofrequency (RF) pulses, were not performed. All studies were performed on a 1.5T unit (Intera, Philips Medical Systems, Best, Netherlands). This system is actively shielded with a maximum gradient amplitude of 30 mT/m and a maximum slew rate of 150 T·m⁻¹·s⁻¹. The RF body coil was used as the transmission coil, and the gradient amplitude was limited to 10 mT/m.

**Pre-MRI Pacemaker Evaluation**

The same electrophysiologist interrogated all pacemakers immediately before the MRI examination. All available data on the technical and functional status of the pacemaker system (battery voltage, battery resistance, lead impedance, and pacing capture thresholds [PCTs]) were measured and recorded. PCTs were obtained at a pulse duration of 0.4 ms with pulse amplitude being the dependent variable. If the pacemaker was then reprogrammed to settings that depended on the intrinsic patient rhythm. In non–pacemaker-dependent patients with an intrinsic heart rate <60 bpm, the pacemaker was reprogrammed to asynchronous pacing (A00, V00, D00) with a pacing rate of 80 bpm, whereas in patients with a heart rate ≥60 bpm, the pacemaker was reprogrammed to a sensing (monitor-only mode) (A00, V00, D00). If a monitor-only mode was not available on the specific model, it was reprogrammed to subthreshold pacing alone. Lead polarity was reprogrammed to bipolar if possible. All additional diagnostic and therapeutic features, such as rate response, capture management, and mode switch, were turned off before MRI.

**Patient Monitoring During MRI**

Heart rate and oxygen saturation were monitored continuously with magnetic resonance (MR)–compatibly encoded ECG and pulse oximetry (Maglife C; Bruker, Wissembourg, France; Figure). Audio contact was established via an intercom system, and patients were asked to inform the investigator immediately of any torque or heating sensation, palpitations, dizziness, pain, or other unusual symptoms during imaging. An electrophysiologist and full resuscitation equipment were present during all examinations. The status of the reed switch was assessed in the subgroup of patients reprogrammed to asynchronous stimulation as follows: The reed switch was considered closed when asynchronous pacing at the magnet rate (85 bpm) was observed and open when asynchronous pacing at the programmed lower rate (80 bpm) was observed. In the subgroup of patients reprogrammed to a sensing-only mode or to subthreshold pacing, assessment of the reed switch status was not possible because there was no effective stimulation.

**Post-MRI Pacemaker Evaluation**

All data on the technical and functional status of the pacemaker system were again measured and recorded. Each lead was evaluated for changes in PCT. A clinically significant change was defined as a threshold increase of ≥1.0 V, because changes smaller than 1.0 V...
may be related to normal variations in the underlying electrophysiological conditions. A chest radiographic examination was performed in all patients with a clinically significant change in PCT to exclude lead fracture or macrodislocation. Any observed changes in programmed parameters were also recorded. Finally, the pacemaker was reprogrammed to its previous parameters with appropriate adjustments to the output and sensitivity, if needed.

Serum Troponin I
Serum troponin I, a marker of myocardial damage, was measured within 1 hour before and 12 to 24 hours after MRI to detect potential myocardial thermal injury at the lead tips.4,5

Long-Term Follow-Up
A 3-month follow-up examination was performed as before to assess possible long-term or late damage to the pacemaker system or the endocardial/myocardial tissue surrounding the lead tip.

Statistical Analysis
Statistical analysis was performed with SAS 9.1.3 service pack 3 (SAS Institute, Cary, NC). A mixed repeated-measures regression model was fit to the PCT and lead impedance data, which included covariates for cardiac chamber, timing of measurement (prescan, postscan, and follow-up), and number of scans. Correlation between measurements in the same cardiac chambers was modeled by including the subject as the random effect. Correlation between measurements in the same cardiac chamber was modeled with an autoregressive correlation structure. Battery voltage was analyzed similarly. A probability value ≤0.05 was considered to be statistically significant. For patient rates, the exact binomial 95% confidence intervals (CIs) were calculated.

Results
Twenty-one patients with Medtronic pacemakers were excluded owing to pacemaker dependence (n=9) or requested MRI of the thoracic region (n=12). The study group then consisted of 82 patients (53 males, 29 females; mean age 66.9 years, range 3.9 to 88.5 years) on whom a total of 115 MRI examinations were performed. Repeat examinations were performed on 11 patients; examinations were considered separately if they were performed on different days.

The 3-month follow-up interrogation was performed after 101 (87.8%) of 115 (95% CI 80.4% to 93.2%) examinations (mean follow-up interval 96 days, range 81 to 114 days). Follow-up was not possible after 14 examinations (12.2%, CI 6.8% to 19.6%) because 6 patients had died, and the remaining 8 refused to return for follow-up. The deaths occurred at a mean interval of 58 days (range 42 to 81 days) after MRI, and all deaths were related to the underlying disease (1 melanoma with cerebral metastases, 1 pancreatic carcinoma, and 4 brain tumors). None of the deaths was classified as pacemaker or MRI related. A pacemaker interrogation at or close to the time of death was not available in these 6 patients.

Pacemaker Reprogramming
In 47 (40.9%) of 115 examinations (95% CI 31.8% to 50.4%), patients showed an intrinsic heart rate below 60 bpm, and the pacemaker was reprogrammed to asynchronous pacing (A00, V00, D00) with a rate of 80 bpm. In 68 (59.1%) of 115 (95% CI 9.6% to 68.2%) examinations, the intrinsic heart rate was ≥60 bpm, and the pacemaker was reprogrammed to a sensing (monitor)-only mode (0A0, 0V0, 0D0). In 2 examinations with a heart rate >60 bpm, a monitor-only mode was not available for the specific pacemaker model (1 Legend II, 1 Minix); therefore, these were reprogrammed to subthreshold pacing.

Pacemaker Models
Pacemaker models implanted in the 82 patients were as follows: 19 Sigma, 22 Thera, 31 Kappa, 3 Minix, 2 InSync, 2 Prodigy, and 1 each of Legend II, Minuet, and Elite.

Pacemaker Lead Models
A total of 195 pacemaker leads were present (84 atrial and 111 ventricular). No restrictions were imposed on manufacturer, type of fixation (active versus passive fixation), polarity (unipolar versus bipolar), or age of the leads, and thus, a large variety of leads from the following manufacturers were included: Medtronic (n=103), Guidant (Indianapolis, Ind; n=18), Biotronik (Berlin, Germany; n=28), St. Jude Medical (St. Paul, Minn; n=16), Osypka (Rheinfelden-Herten, Germany; n=14), and unknown (n=16).

Anatomic Regions
Regions examined were brain (64 examinations), neck (4 examinations), lumbar spine (17 examinations), abdomen (12 examinations), pelvis (8 examinations), and lower extremities (10 examinations).

Clinical Events During the MRI Examinations
All 115 MRI examinations were completed safely. None of the patients reported any torque or heating sensations, palpitations, dizziness, pain, or other unusual symptoms. No procedures were terminated owing to clinical events or patient complaints.

Changes in Programmed Parameters
In 7 (6.1%) of 115 (95% CI 2.5% to 12.1%) examinations, the post-MR interrogation revealed an electrical pacemaker reset with subsequent alteration of the programmed pacing parameters. An electrical reset restores factory settings with predefined synchronous pacing mode (usually VVI) and parameters. The specific models that underwent an electrical reset are given in Table 3. In all cases (7/7, 100%; 95% CI 59.0% to 91.3%), patients showed an intrinsic heart rate below 60 bpm, and the pacemaker was reprogrammed to asynchronous pacing (A00, V00, D00) with a rate of 80 bpm. In 68 (59.1%) of 115 (95% CI 9.6% to 68.2%) examinations, the intrinsic heart rate was ≥60 bpm, and the pacemaker was reprogrammed to a sensing (monitor)-only mode (0A0, 0V0, 0D0). In 2 examinations with a heart rate >60 bpm, a monitor-only mode was not available for the specific pacemaker model (1 Legend II, 1 Minix); therefore, these were reprogrammed to subthreshold pacing.
to 100%), the pacemaker could be reprogrammed to the parameters present before MRI. All other pacemakers (108/115, 93.9%; 95% CI 87.9% to 97.5%) did not show any changes in the programmed pacing parameters.

Change of Heart Rate or Rhythm
No unexpected changes in heart rate or rhythm, which indicates oversensing, inhibition of pacemaker output, false triggering, episodes of pacing above the upper rate limit, or sustained atrial and ventricular arrhythmias were observed. In addition, appropriate stimulation was recorded during all examinations in which pacemakers were reprogrammed to asynchronous pacing mode (47 of 115, 40.9%; 95% CI 31.8% to 50.4%). In this subgroup, determination of the reed switch status during MRI was possible; in 21 (44.7%) of 47 (95% CI 30.2% to 59.9%), pacemaker stimulation was observed at the programmed lower rate limit, which indicates an open reed switch, whereas stimulation at the magnet mode rate was observed in 26 (55.3%) of 47 (95% CI 40.1% to 69.8%), which indicates closure of the reed switch. MRI-induced inhibition of the pacemaker output was not observed in any examination in which an electrical reset (n=7) with a resultant switch of the pacing mode to VVI occurred; in 5 of these 7 examinations, no pacemaker output was recorded because the intrinsic heart rates were above the lower rate limit (which is 65 bpm after an electrical reset). In the other 2 examinations, the reed switch was activated, with resultant asynchronous stimulation at the magnet mode rate.

Threshold Changes
Mean atrial and ventricular PCT before MRI, after MRI, and at follow-up are given in Table 4. The statistical analysis demonstrated a significant increase in PCT from before to after MRI (P=0.017). Clinically significant changes in PCT, defined as an increase in PCT ≥1.0 V, were observed in 6 (3.1%) of 195 leads (95% CI 1.1% to 6.6%). Four of the 6 increases in PCT were observed immediately after MRI, and these did not return to baseline at 3-month follow-up. No lead fracture or macrodislocation was found on chest radiography in these 6 patients. None of the leads required a change in programmed output to maintain appropriate function.

Changes in Lead Impedance
Mean atrial and ventricular lead impedance before and after MRI and at follow-up are given in Table 4. The statistical analysis demonstrated a significant decrease of lead impedance from pre- to post-MRI (P=0.025).

Changes in Battery Voltage
Mean battery voltage was 2.782±0.034 V before MRI, 2.780±0.035 V after MRI, and 2.779±0.026 V at follow-up (Table 4). The statistical analysis showed a significant decrease in battery voltage from pre- to post-MRI (P=0.0012; Table 4). This decrease in battery voltage was transient in most examinations, with full recovery at follow-up (76 [66.1%] of 115; 95% CI 56.7% to 74.7%).

Troponin I
A total of 114 blood samples were analyzed; in 1 patient, no post-MRI sample was available. The mean troponin I level was 0.02±0.03 ng/mL before MRI and 0.03±0.03 ng/mL afterward (Table 4). A 1-sided paired Student t test did not show a statistically significant increase (P=0.0693) when pre- and post-MRI levels were compared (Table 4). However, in 4 of 114 patient examinations (3.51%; 95% CI 1.0% to 8.7%), the troponin level increased from a normal baseline value to above normal afterward (threshold 0.1 ng/mL). One of these 4 patients also showed an increase in PCT (Table 5).

Discussion
Potential risks associated with MRI of patients with pacemakers are related to numerous interactions with the static magnetic field and pulsed fields of the MRI system. Translational forces and torque on pacemaker leads have been evaluated in several studies and have been shown to be small enough not to represent a safety concern at 0.5T and 1.5T for

| Table 4. Means and Standard Deviations of PCT, Lead Impedance, Battery Voltage, and Serum Troponin I Before and After MRI and at 3-Month Follow-Up |
|---------------------------------|-----------------|-----------------|-----------------|-----------------|
| PCT @ 0.4-ms pulse duration, V  | Before MRI      | After MRI       | Follow-Up       | PCT @ 0.4-ms pulse duration, V  | Before MRI      | After MRI       | Follow-Up       |
| Chamber                        | n    | Mean±SD     | n    | Mean±SD     | n    | Mean±SD     | n    | Mean±SD     |
| Atrium                         | 78   | 0.94±0.39   | 78   | 0.98±0.41   | 67   | 0.96±0.40   | 100  | 0.98±0.56   |
| Ventricle                      | 112  | 0.93±0.54   | 112  | 0.97±0.55   | 100  | 0.98±0.56   | 115  | 0.97±0.56   |
| Lead impedance, Ω              | 0.025 | 579.0±142.7 | 83   | 578.2±143.8 | 73   | 548.9±143.1 | 99   | 662.5±181.6 |
| Atrium                         | 83   | 579.0±142.7 | 83   | 578.2±143.8 | 73   | 548.9±143.1 | 99   | 662.5±181.6 |
| Ventricle                      | 111  | 667.0±181.2 | 112  | 658.0±182.2 | 99   | 662.5±181.6 | 115  | 658.0±182.2 |
| Battery voltage, V             | 0.0012 | 2.782±0.034 | 115  | 2.780±0.035 | 101  | 2.779±0.026 | 114  | 2.780±0.035 |
| Serum troponin I, ng/mL        | 0.0693 | 2.782±0.034 | 115  | 2.780±0.035 | 101  | 2.779±0.026 | 114  | 2.780±0.035 |

*P values for PCT (both chambers) comparing pre-MRI and post-MRI measurements.
†Indicates statistical significance.
Therapy DR 7960i

Therapy DR 7940

Kappa DR731

NP-53-BP

Sigma SDR

Kappa DR731

Therapy DR 7940

Pacemaker Model

Therapy DR 7940

Programmed data caused by an electrical reset.10–14

Switch, inhibition of pacemaker output, and change or loss of
dysfunction due to interaction with the MRI fields that leads
to RF-induced heating of the lead tips and pacemaker

The principal risks of MRI of pacemaker patients are related
to MRI, damage to the pacemaker or its components related to MRI.8

In vivo animal studies19 have revealed a temperature increase
in vivo9 studies at 0.5T and 1.5T revealed no irreversible

The extent of

In vivo studies simulating worst-case conditions in a
no-flow phantom have shown temperature increases (∆T) at
the lead tip of 23.5°C at 0.5 Tesla8 and up to 88.8°C at 1.5T.18

In modern pacemakers.6,7 Furthermore, extensive in vitro8 and in vivo9 studies at 0.5T and 1.5T revealed no irreversible damage to the pacemaker or its components related to MRI. The principal risks of MRI of pacemaker patients are related to RF-induced heating of the lead tips and pacemaker dysfunction due to interaction with the MRI fields that leads to asynchronous stimulation caused by closure of the reed switch, inhibition of pacemaker output, and change or loss of programmed data caused by an electrical reset.10–14

Heating and Stimulation Threshold Changes

Heating, which results from the RF radiation used in MRI, is
carried by power deposition into an ohmic resistance. Maximum
RF-induced heating occurs at the electrode-tissue boundary;
that is, the area of endocardium and myocardium close to the
tip of the electrode has a potential risk of thermal injury,15 which may result in deterioration of pacing thresholds
and/or atrial or ventricular perforation. The extent of
RF-related heating depends on the SAR16,17 of the sequence
used, the position of the pacemaker lead loop in the RF coil,
the configuration of the lead, and the specific lead model.8

In vitro studies simulating worst-case conditions in a
no-flow phantom have shown temperature increases (∆T) at
the lead tip of 23.5°C at 0.5 Tesla8 and up to 88.8°C at 1.5T.18

In vivo animal studies19 have revealed a temperature increase
at the pacemaker lead tip of up to 20.4°C at 1.5T at an SAR
value of 3.8 W/kg. These are in the range of temperatures
used for ablations (usually 50°C to 70°C) and have the
potential to cause serious harm to the patient due to loss of
pacemaker capture, as demonstrated in a recent in vivo
animal study of implantable cardioverter defibrillator leads at
1.5T.10

In the present study, we took several precautions designed
to reduce the risk of RF-induced heating. First, the SAR value
was limited to <1.5 W/kg (a value that allows almost all clinical MRI sequences to run with only minor modification).
Second, the amount of RF power transmitted to the leads of
the pacemaker system was reduced by excluding anatomic
regions with full coverage of the pacemaker lead loop, ie, the
thoracic spine, heart, and breasts. Finally, the total active scan
time (ie, the length of RF exposure) was limited to 30
minutes, because RF-induced thermal tissue damage is a
function of both temperature and exposure time.20–22

Using these precautions, we observed a total of 6 PCT
changes greater ≥1.0 V in 195 leads (3.1%), which were
considered to be MRI related, and statistical analysis revealed
a significant increase of PCT (P=0.0017) from pre- to
post-MRI. However, in none of these patients did this
increase have clinically relevant effects, ie, increase of the
pacemaker output was not necessary in any patient to ensure
stimulation with a sufficient safety margin (twice the mea-
sured PCT value).

The present study data confirm the results of Martin et al.9
who reported significant PCT changes (defined as a change
>1 voltage or pulse-width increment or decrement) in 9.4%
(10/107) of the leads from pre- to post-MRI. Both studies

<table>
<thead>
<tr>
<th>Pacemaker Model Chamber and Lead</th>
<th>PCT @ 0.4 ms, V</th>
<th>Impedance, Ω</th>
<th>Battery Voltage, V</th>
<th>Troponin I, ng/mL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thera DR 7940</td>
<td>Before MRI</td>
<td>After MRI</td>
<td>Follow-Up</td>
<td>Before MRI</td>
</tr>
<tr>
<td>A: CPI 4169</td>
<td>1.00</td>
<td>1.00</td>
<td>1.50</td>
<td>811</td>
</tr>
<tr>
<td>V: CPI 4169</td>
<td>2.00</td>
<td>2.00</td>
<td>3.00*</td>
<td>1198</td>
</tr>
<tr>
<td>Kappa DR731</td>
<td>Before MRI</td>
<td>After MRI</td>
<td>Follow-Up</td>
<td>Before MRI</td>
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<td>A: Medtronic 5076</td>
<td>0.50</td>
<td>1.50</td>
<td>1.50*</td>
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<td>V: Medtronic 5076</td>
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<td>1.50</td>
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<tr>
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<td>Follow-Up</td>
<td>Before MRI</td>
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<tr>
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<tr>
<td>V: Medtronic 5067</td>
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<td>1.50</td>
<td>1.50*</td>
<td>442</td>
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<td>After MRI</td>
<td>Follow-Up</td>
<td>Before MRI</td>
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<td>1.50</td>
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<td>657</td>
</tr>
<tr>
<td>V: Medtronic 4081</td>
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<td>0.50</td>
<td>1.50*</td>
<td>843</td>
</tr>
<tr>
<td>Thera DR 7960i</td>
<td>Before MRI</td>
<td>After MRI</td>
<td>Follow-Up</td>
<td>Before MRI</td>
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<tr>
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<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>731</td>
</tr>
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</table>

A indicates atrial lead; V, ventricular lead. Before to after MRI or before MRI to 3-month follow-up.

*Indicates increases of PCT ≥1.0 V at a pulse duration of 0.4 ms compared with baseline value (before MRI).

†Indicates an increase of serum troponin I to a value above the normal range (>0.1 ng/mL) after MRI.

TABLE 5. Examinations With Clinically Significant PCT Changes (≥1.0 V @ 0.4 ms)
Interference of MRI With Pacemaker Function

The present study confirms the findings of recent in vitro24 and in vivo studies7,8 that reed switch activation does not necessarily occur when a pacemaker is placed in an MR magnet, despite the presence of up to a 1.5T magnetic field (reed switch activation 50% to 69%8,9,24). In the present study, the reed switch remained inactivated in 44.7% of examinations. Therefore, the behavior of the reed switch is not predictable for a given patient, which has important ramifications for imaging of these patients.

The pacemaker reprogramming strategy used in the present study—intrinsic heart rate <60 bpm: asynchronous mode with 80 bpm; intrinsic heart rate ≥60 bpm: sense-only mode—minimizes both possible risks related to interference of MRI with reed switch behavior. In patients with low intrinsic heart rates, inhibition of pacemaker output by the pulsed magnetic fields becomes impossible, and in patients with high intrinsic heart rates, the risk of induction of cardiac arrhythmias related to competitive rhythms due to closure of the reed switch is minimized. However, in patients with heart rates >60 bpm, this approach still bears a small risk of competitive pacing and proarrhythmia, especially if there is an increase in the patient’s intrinsic heart rate during the MRI examination. Likewise, in patients with an initial heart rate >60 bpm with reprogramming of the pacemaker to a sense-only mode, bradycardia may occur if the heart rate unexpectedly drops during the MR examination. Furthermore, the examination strategy used in the present study does not eliminate the theoretical risk of MRI-related induction of currents in the pacemaker leads, with subsequent induction of arrhythmias.

With this approach, all examinations were completed safely, with continuous stimulation in all pacemakers reprogrammed to asynchronous mode and no stimulation in pacemakers reprogrammed to sense-only mode: Neither inhibition of pacemaker output nor generation of competitive rhythms occurred in the present study.

Change of Programmed Parameter/Electrical Resets

An electrical reset is an emergency mode that represents a safety feature to guarantee minimal pacemaker functionality in case of battery voltage dips due to electromagnetic interference or battery depletion. An electrical reset implies a change in the programmed parameters to default settings, usually an inhibited pacing mode (VVI). In the present study, electrical resets caused by exposure of the patient to the static and/or pulsed magnetic fields were observed in 7 of 115 examinations. This finding is important from a safety point of view for 2 reasons. First, in the case of an electrical reset and an open reed switch, pacemaker output may be inhibited by the time-varying gradient fields, which could potentially lead to bradycardia/asytole in patients with low intrinsic heart rates. Second, the default pacing mode and output may provide inadequate pacemaker functionality for a given patient: (1) all pacemaker-dependent patients (due to potential inhibition of pacemaker output); (2) children, who are known to have high intrinsic heart rates (the emergency VVI 65 mode after an electrical reset may not provide a sufficient cardiac output); (3) patients requiring a high pacemaker output to ensure effective stimulation (the default output parameters after an electrical reset may not provide effective stimulation); and (4) pacemaker patients who also have an ICD. In these patients, the pacemaker usually is inactivated to avoid undersensing of ventricular fibrillation as bradycardia by the pacemaker. In the case of an electrical reset with subsequent switch to VVI mode, the occurrence of ventricular fibrillation could result in pacemaker stimulation, which could lead to fatal inhibition of ICD therapy delivery. Therefore, a pacemaker interrogation should be performed immediately after MRI.

Battery Voltage

The effect of MRI on battery voltage, demonstrated in the present study by significantly (P=0.0012) decreased values immediately after MRI (Table 4), has also been shown previously at 0.5T.25 Activation of telemetry circuits and increased pacing rate due to closure of the reed switch during MRI burdens the battery, especially in pacemakers approaching elective replacement criteria.11 However, the observed decrease of battery voltage immediately after MRI was minimal (with a maximum decrease of 0.03 V); was transient in most examinations, with full recovery at follow-up (66.1%); and did not interfere with pacemaker function. These small changes are unlikely to affect the longevity of the pacemaker dramatically, because a decrease of 0.05 V is estimated to reduce pacemaker longevity by only ≈2 months.25 Therefore, the decrease in battery voltage demonstrated in the present study is of minor clinical importance and does not represent a safety risk.

Study Limitations

All devices investigated in the present study were Medtronic devices. This might limit the results and conclusions of the study as being valid only for Medtronic pacemakers. However, interactions of the static magnetic field and the reed switch, inhibition of the pacemaker output by time-varying gradient fields, and a safety mode with default pacing parameters in case of an electrical reset due to electromagnetic interference are events/phenomena that are related to underlying basic principles of pacemaker technology, and
they have also been reported for pacemakers of other manufacturers in previous studies.8–10 In addition, no restrictions on the leads implanted were imposed in the present study, which provided general and important insights into the problematic field of RF-induced lead heating.

Another limitation of this study is that patients requiring MRI examinations of the thoracic region and patients with pacemaker dependency were excluded. However, only 8.8% (12/136, 95% CI 4.6% to 14.9%) of the requested MRI examinations were thoracic MRI examinations, and only 5.1% (9/136, 95% CI 3.1% to 12.2%) were requested in pacemaker-dependent patients.

Finally, the power of the gradient system of the MRI system in the present study was limited by restricting the maximum gradient amplitude to10 mT/m. Therefore, the use of sequences that require stronger gradients may impose higher safety risks, especially the induction of ventricular arrhythmias and inhibition of pacemaker output.

Conclusions

The results of the present study demonstrate that extrathoracic MRI of non–pacemaker-dependent patients can potentially be performed safely under controlled conditions, including (1) limitation of RF exposure to minimize the risk of RF-related thermal myocardial injury, by restriction of SAR values and scan time, and exclusion of high-risk anatomic regions with full coverage of the pacemaker lead loop; (2) reprogramming of the pacemaker to a sense-only mode or to asynchronous pacing, depending on the individual’s heart rate, to avoid competitive rhythms in patients with high intrinsic heart rate, and gradient field-induced inhibition in patients with low intrinsic heart rates; (3) continuous monitoring of ECG and pulse oximetry to detect any change in heart rate or rhythm related to MRI-induced pacemaker inhibition, loss of pacemaker capture, or ventricular arrhythmias; and (4) presence of an electrophysiologist and full resuscitation facilities at the MRI site. Pacemaker interrogation immediately after the MRI is necessary to detect any change in or loss of programmed parameters and to restore the original settings. Additional testing, ie, long-term follow-up pacemaker interrogation after MRI, should be performed to assess potential late effects.

We strongly believe that reasonable safety levels can potentially be achieved under these well-controlled circumstances, but the absolute safety of MRI in pacemaker patients at present cannot be guaranteed. It is especially notable that despite the use of strict precautionary measures in the present study to minimize the risk of RF-related heating of the lead tip, subclinical myocardial injury, as indicated by increases of PCT and serum troponin I levels, could not be eliminated entirely. Therefore, each case requires a careful risk-benefit evaluation. At the present stage of development in this area, MRI of patients with pacemakers should be performed only in experienced centers, with close cooperation between electrophysiologists and radiologists.

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Disclosures

Dr Sommer is a consultant for Medtronic; as the corresponding author, he had full access to all of the data in the study and the final responsibility for the decision to submit for publication. The salary of Dr Hackenbroch as a radiology research resident was supported by Medtronic. Dr Zeijlemaker is an employee of Medtronic. The other authors report no conflicts.

References


**CLINICAL PERSPECTIVE**

Magnetic resonance imaging (MRI) has emerged as one of the most important diagnostic tools in current clinical practice, with an expected annual growth rate of >10% in the Western world. MRI is currently considered the imaging modality of choice in many fields such as imaging of the brain, spinal cord, liver, breast, and musculoskeletal system. In 2002, there were approximately 2.4 million patients in the United States with implanted cardiac pacemakers, and this number is growing by 80 000 per year. With Europe offering comparable numbers, a large group of patients is currently denied magnetic resonance examinations by most institutions. The present study, which included a 3-month follow-up to assess potential long-term damages, confirms the shift from cardiac pacemakers being an absolute contraindication to MRI to a relative contraindication. If adequate safety precautions are taken, acceptable risk-benefit ratios can be achieved for pacemaker patients with a clinical need for MRI. These precautions included a specific pacemaker reprogramming strategy, as well as some modification to the magnetic resonance scanning techniques (limitation of radiofrequency energy and active scan time). Furthermore, imaging of the thoracic region was not performed to reduce radiofrequency energy deposition in the pacemaker leads. Reasonable safety levels can potentially be achieved under these well-controlled circumstances, but at present, absolute safety still cannot be guaranteed. Therefore, each case requires a careful risk-benefit evaluation, and MRI on pacemaker patients should only be performed in experienced centers with close cooperation between radiologists and electrophysiologists.
Strategy for Safe Performance of Extrathoracic Magnetic Resonance Imaging at 1.5 Tesla in the Presence of Cardiac Pacemakers in Non-Pacemaker-Dependent Patients: A Prospective Study With 115 Examinations

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