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In vivo heating of pacemaker leads during magnetic resonance imaging

Roger Luechinger[1], Volkert A. Zeijlemaker[2], Erik Morre Pedersen[3], Peter Mortensen[3], Erling Falk[3], Firat Duru[4], Reto Candinas[4], and Peter Boesiger[1]*

[1] *Institute of Biomedical Engineering, University and Swiss Federal Institute of Technology, Gloriastrasse 35, 8092 Zurich, Switzerland*
[2] *Medtronic, Bakken Research Center, BV., Maastricht, The Netherlands*
[3] *Skejby Sygehus, MR-Center and Department of Cardiology, Aarhus University Hospital, Denmark*
[4] *Division of Cardiology, University Hospital of Zurich, Switzerland*

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Aims Magnetic resonance imaging (MRI) is well established as an important diagnostic tool in medicine. However, the presence of a cardiac pacemaker is usually regarded as a contraindication for MRI due to safety reasons. In this study, heating effects at the myocardium–pacemaker lead tip interface have been investigated in a chronic animal model during MRI at 1.5 Tesla.
Methods and results Pacemaker leads with additional thermocouple wires as temperature sensors were implanted in nine animals. Temperature increases of up to 20°C were measured during MRI of the heart. Significant impedance and minor stimulation threshold changes could be seen. However, pathology and histology could not clearly demonstrate heat-induced damage.
Conclusions MRI may produce considerable heating at the lead tip. Changes of pacing parameters due to MRI could be seen in chronic experiments. Potential risk of tissue damage cannot be excluded even though no reproducible alterations at the histological level could be found.

Introduction

Magnetic resonance imaging (MRI) is a widely accepted tool for the diagnosis of a variety of disease states. However, the presence of an implanted cardiac pacemaker is considered to be a strict contraindication to MRI in most medical centres, precluding a substantial number of patients from the diagnostic advantages of this imaging modality.[1–6] On the other hand, a few reports of safe MRI investigation in pacemaker patients have recently become available.[7–11] Potential safety

risks of MRI on pacemaker systems are: force and torque effects on the pacemaker,[12,13] undefined reed-switch state within the strong magnetic field,[14] potential risk of heart stimulation and inappropriate pacing,[15,16] and heating effects at the lead tip.[5,17]

MRI-induced heating is a known risk for various metallic implants.[17–21] The radio frequency (RF) field used in MRI procedures may be concentrated by metallic implants producing hot spots. The RF field will induce high currents. Due to the limited conductivity of the tissue, most of the energy will be deposited as heat as a result of ohmic loss in the heart tissue around the lead tip.[22] Therefore, there is a potential risk of thermal injury, which may theoretically result in the deterioration of

* Corresponding author. Tel: +41 1 632 45 81; fax: +41 1 632 11 93.
 E-mail address: boesiger@biomed.ee.ethz.ch

stimulation thresholds or even, in extreme circumstances, in atrial or ventricular perforation. In the study by Martin *et al.*[7] significant simulation threshold changes from pre-, to post-MRI could be found in about 10% of the patients undergoing MRI, however, none of these was clinically significant. No long-term follow-up data were available for that study. Specific absorption rate (SAR) was limited to 2 W/kg and only half of the scans were performed with the thorax or neck in the isocentre of the magnet. *In vitro* studies showed high heating effects near the tip of the pacing leads.[5,17] In order to understand the effects of MRI-induced heating around pacemaker leads and pacing performance, it is important to measure heating at the lead tip together with the pacing parameters of chronically implanted pacemaker systems.

For the *in vitro* evaluation of heating effects in the MRI environment, fibreoptic sensors are the industry standard.[18,23] These sensors are not influenced by electromagnetic fields and will not influence the MRI procedure either. However, fibreoptic sensors are too fragile to be chronically implanted in animals. An alternative is the use of thermocouples that have long been used as reliable temperature sensors in catheter ablation procedures.[24] However, these sensors have to be validated for the MRI environment.

The aim of this study was to measure heating effects at the lead tip of a pacing lead in a chronic animal model during standard cardiac MRI scans at 1.5 Tesla (T). For this purpose, a pacing lead with an additional thermocouple as temperature sensor was developed and evaluated. Heat-induced tissue damage and changes of pacing performance were evaluated.

Methods

Pacemaker systems (InSync III, Medtronic Inc., Minneapolis, MN, USA) with three leads were implanted in nine pigs to evaluate heating effects induced by the RF field of an MRI scanner on the pacing parameters. With three initial experiments (Animals 1–3) an optimal set-up for investigation was defined. With this optimized set-up, the investigations were performed in the remaining six pigs (Animals 4–9).

Pacing leads and pacemakers

Both passive and active fixation leads were used for the initial evaluation. In the first animal, three custom-made passive fixation leads (CapSure SP, Medtronic, tip surface area: 5.8 mm^2) with integrated T-thermocouple sensor (Copper-Constantan) were used. Active fixation leads (similar to Medtronic Model 5072, tip surface area: 6.3 mm^2) were used in the other animals. The leads had handling properties (stiffness, torque, etc.) comparable to those of commercial leads, and the tip emitted steroids to suppress inflammation of the heart wall. The use of a stylet during implantation was possible without any limitations.

For the chronic experiments, the thermocouple wire was coiled inside the lead body. The temperature sensor was placed inside the screw (~2 mm from the top of the helix), because highest temperature increase is expected within and

on the surface of the screw. The sensor tip was electrically insulated from the tissue to prevent auxiliary currents from the temperature-measuring device to the animal. The temperature was measured by a high-precision micro-voltmeter card in a battery-operated portable computer (NI 4350, National Instruments, Austin, TX, USA). The accuracy of the temperature measurement was verified during *in vitro* MRI experiments using a fibreoptic temperature sensor (Model 790, Luxtron, Santa Clara, CA, USA). For the lead used in animal 1, the thermocouple overestimated the temperature increase by 2.8°C at a total increase of 56°C, for the lead used in the chronic experiment the thermocouple underestimated by 3.4°C at a temperature increase of 30°C (averaged over 21 s). The differences may partly be due to the mechanical restrictions in placing the fibreoptic sensor at the lead tip. Overall, the temperature measurements should agree between the two methods within a 10% margin. The comparison of two identical leads, one with and the other without a thermocouple, showed similar heating effects, thus excluding the influence of the thermocouple on heating in the set-up.

To assess heating for different pacing positions, the leads were placed in the right atrial appendage (RAA), the right ventricular apex (RVA), and at the right ventricular outflow tract (RVOT). The pacing function was turned off for all three leads and, to prevent any inappropriate stimulation, only bipolar sensing was enabled during MRI (ODO mode); using a modified programmer head (magnet removed for safety reasons), it was possible to interrogate and program the pacemaker between MRI scans without removing the animal from the MRI table (*Figure 1*). The pacemaker programmer (Medtronic 9790 C) was placed outside the RF cage at all times. Stimulation threshold measurements, pacing impedance, and sensing measurements were performed in each animal on five occasions: first after implantation, then three times at the MRI suite (before, during, and after MRI), and before the pigs were sacrificed, using the capability of the InSyncIII pacemaker. To achieve a higher sensitivity in voltage stimulation threshold evaluation, thresholds were measured at multiple pulse durations: 0.4, 0.2, and 0.06 ms. Available voltage steps were 0.5 V between 0.5 and 4 V plus two additional voltages of 5 and 7.5 V. Capturing pulse duration was determined at 2.5 V. The pulse duration could be increased from 0.03 to 0.15 ms in steps of 0.03 ms, then up to 1 ms in steps of 0.05 ms and finally in steps of 0.1 ms up to 1.5 ms.

MRI procedures

All measurements were performed on a 1.5 T Gyroscan NT (Philips Medical Systems, Best, The Netherlands). The animals were anaesthetized, artificially ventilated, and ECG and CO_2 were monitored continuously. The animals were placed in supine position with the heart at the isocentre of the MRI unit. A steady-state free precession (SSFP) sequence was used for surveys and anatomical images. To provoke heating, an untriggered turbo-spin echo sequence (field of view 400 mm, matrix 256 × 205, echo time 19 ms, repetition time 300 ms, eight slices, flip angle 90°, turbo factor 6, SAR = 3.8 W/kg, image acquisition with the built-in 56-cm long transmit body-coil) was used. The averaged whole body SAR-value of the sequence calculated by the scanner software (Release 8.1.1) was near the limits for the thorax of 4 W/kg mentioned in the IEC 60601-2-33 standard.[25] By changing the number of averages from 4 to 32 and by increasing the slice number, the scan duration could be changed from 52 s to 16 min 30 s, without changing the SAR-value.

Figure 1 The animal was placed feet first, lying supine, into the scanner. It was possible to interrogate and program the pacemaker using a modified programmer head (white arrow) in the position shown above. For stimulation threshold measurements, additional ECG cables were connected to the four legs. During MRI measurements these ECG cables and the programmer head were removed due to safety reasons. During the whole time in the MRI room, the pig was monitored using the ECG from the MRI unit.

In vivo measurements

The animal study protocol was approved by the Danish National Inspection Board for animal research. The pacemaker leads were evaluated in pigs (Danish Landrace) weighing between 60 and 65 kg (aged 130 days). Pigs are known to show the best agreement to humans in dimensions of the thorax and the heart.[26] The animals were pre-anaesthetized with midazolam and isofluran was used for maintaining anaesthesia.

In the acute experiments (Animals 1 and 2), MRI was performed directly after pacemaker implantation. In the chronic experiments (Animals 3–9), the MRI procedures were performed 4 weeks after the implantation. Animals 1–3 were sacrificed 1 day after the MRI and Animals 4–9 were sacrificed 2 weeks after MRI. In all animals, standard morphological and pathological examinations were performed. The histology preparations of the regions around the lead tip were examined. Animals 4–9 were used as a group for the statistical evaluation, since the initial three animals had a different set-up. Blood samples for troponin tests were drawn before, 8 h after, and 2 weeks after MRI. The detection of the troponin levels in the blood is a sensitive method to detect cardiac cell death in myocardial infarction.[27] Similarly, radiofrequency catheter ablation will increase the serum troponin levels as a result of heating at the myocardium–lead tip interface.[28,29]

Animals 1–2
Three passive fixation (Animal 1) and active fixation (Animal 2) leads were implanted in the RAA, RVA, and the coronary sinus (CS). In Animal 1, 13 MRI scans, including five turbo-spin echo sequences with an SAR-value 3.8 W/kg of overall 4 min 20 s were performed, and in Animal 2, 10 MRI scans including three scans with 3.8 W/kg totalling 8 min.

Animal 3
Three screw-in leads (RAA, RVA, and RVOT) with coiled thermocouple wires were implanted. Twenty-two MRI scans (among 18

turbo-spin echo sequences with an SAR-value of 3.8 W/kg and a total duration of 30 min) were performed 4 weeks later. To evaluate the influence of the position of the pig, and hence, of the pacemaker lead within the 56 cm long transmit/receive body coil of the MRI scanner, the animal was shifted in 5 cm steps over a range of 40 cm in the direction of the main magnetic field (*Figure 2*).

Animals 4–9
Three screw-in leads (RAA, RVA, and RVOT) were implanted. Four weeks after implantation, one to two survey scans and three to five scans with an SAR-value of 3.8 W/kg (mean scan time 38 min ± 5.2 min comparable to a longer clinical MRI procedure) (*Table 1*) were performed.

Statistics
Results are expressed as means ± SD. All statistical analyses were performed using commercially available statistical software (INSTAT 3.01, GraphPad Software Inc., San Diego, CA, USA). Performing comparisons among the pacing impedance measured for each animal at three time points (pre-, post-, and 2 weeks post-MRI), all possible pairwise comparisons between the three time-points were assessed simultaneously using a repeated measures ANOVA with Tukey–Kramer analysis, testing for a significant pacing impedance change induced by the MRI procedure. ANOVA with Tukey–Kramer analysis was used to adjust the significance levels for the pairwise testing. $P < 0.05$ for two-sided comparisons was considered statistically significant.

Results

Animal 1

All three temperature sensors functioned appropriately. The temperature increases were 3°C for the lead in the RAA, 18°C for the RVA lead, and 1°C for the CS lead. About 15 s after initiation of MRI scanning a regular tachycardia at 240 b.p.m. was observed (*Figure 3*). Within 5 s of scanning termination, the tachycardia converted to sinus rhythm at 85 b.p.m. This observation was reproducible in all MRI scans with high SAR-values but not in the survey scans (SAR-value < 1 W/kg).

Animal 2

All three thermocouple sensors functioned appropriately. The measured temperature increase was up to 14°C for the CS lead, 9.5°C for the RVA lead, and 1.9°C for the RAA lead.

In both acute studies, post-mortem examination revealed no macroscopic tissue damage that could be attributed to heating. In the microscopic evaluation, local tissue injury could be seen at the lead tip, but it was not possible to distinguish whether it was caused by physical damage during the implantation or thermal damage.

Animal 3

The thermocouples functioned appropriately 4 weeks after implantation. The peak heating was reached within a few seconds for all three leads (*Figure 4*). This could be seen in all animals. In Animal 3 the temperature
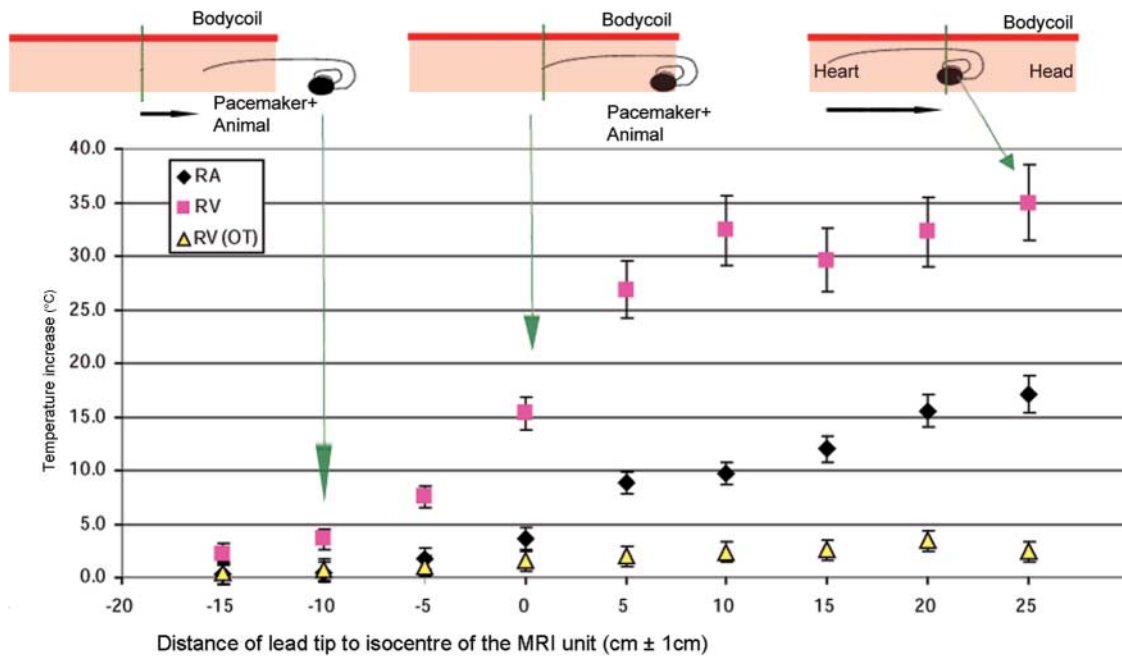
Figure 2 The heating at the lead tip depended strongly on the position of the pacing lead within the body-coil of the MRI unit. Maximal temperature increase was seen if the pacemaker and the whole lead were inside the RF-field transmitting body-coil (as shown in the schema above right). The body-coil of the 1.5 Tesla Gyroscan NT has a length of 56 cm. Because of the limited length of the respirator tubes the experiment could not be continued beyond 25 cm.

Table 1 Temperature increases and scan times

Animal	Total scan time (min)	RAA (°C)		RVA (°C)		RVOT (°C)	
		ΔT_{avg}	ΔT_{max}	ΔT_{avg}	ΔT_{max}	ΔT_{avg}	ΔT_{max}
4	30	—	—	—	—	—	—
5	41	—	—	5.7[a]	5.8[a]	5.4[a]	5.5[a]
6	41	15.7	20.4	11.1	15.6	12.2	17.2
7	37	5.7	7.0	9.3	9.7	9.7	10.6
8	40	9.1	16.6	7.5	9.7	9.0	12.1
9	42	3.7	4.2	3.9	4.7	11.4	14.7

[a]Temperatures could only be measured during part of the MRI scans.

increase at the lead tip was measured for different positions of the pig in the MR system (*Figure 2*). If the whole lead and the pacemaker were inside the body-coil, heating was highest. As soon as a part of the lead was outside the body-coil, the heating was reduced.

Animals 4–9

In Animal 4, no heating measurements were possible because all thermocouples were damaged either during the 4 weeks between implantation and MRI or during the small operation before MRI to assess the thermocouple connectors. In Animal 5, no temperature measurements were available for the RVA lead and, in addition, the heating measurements of the other two leads could only be performed for a subset of the MRI procedures. All temperature measurements and scan

durations are summarized in *Table 1*. *Table 2* shows the results of the lead impedance and stimulation threshold measurements. The averaged impedance changed from 532 ± 70 (before MRI) to 569 ± 69 (after MRI) and to 587 ± 62 Ohm (2 weeks after MRI). There was a significant increase of impedance ($F = 6.3$, df = 2, $P < 0.05$, paired for the three time-points for each pig, Tukey–Kramer ANOVA test) between pre- and post-MRI and a highly significant increase of impedance ($P < 0.001$) between pre- and 2 weeks post-MRI. Pacemaker leads which showed no capture before MRI, for example due to lead dislocation, were excluded from the statistics. All troponin tests were negative, indicating that no major heart tissue damage occurred. Pathology showed no evidence for heat-induced damage. Cell damage induced by the implantation could not be distinguished from potential cell damage from heating effects (*Figure 5*). Mild inflammation
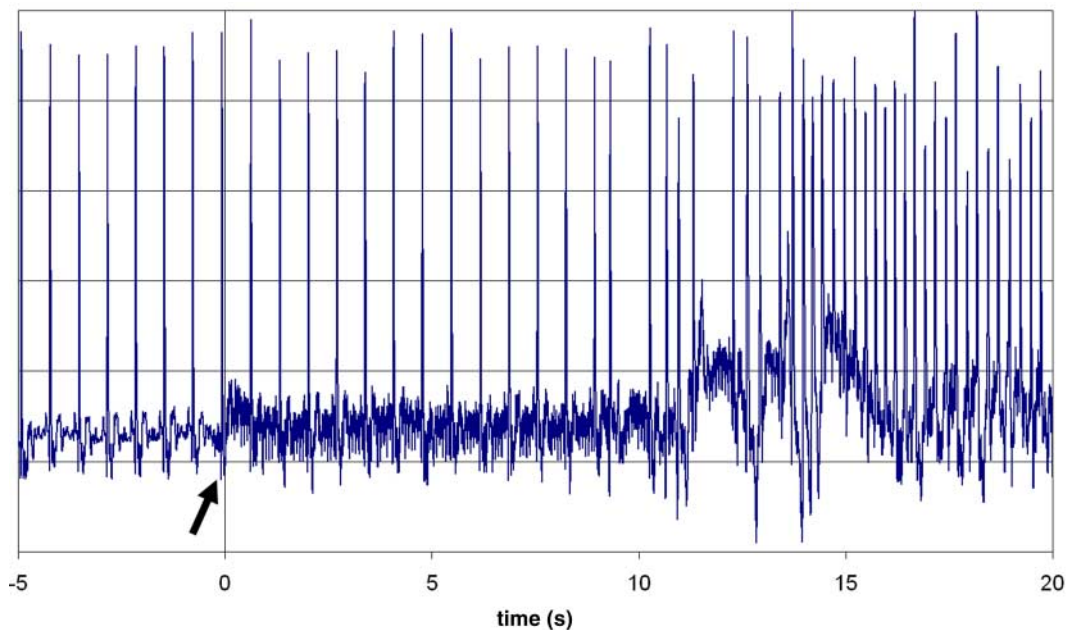
Figure 3 ECG recording of the MRI unit of Animal 1. The MRI scan starts at time-point zero (arrow), seen in the increased noise induced by the RF and gradient fields of the MRI unit. After about 10–15 s a regular tachycardia at 240 b.p.m. was observed. Within a few seconds after the scan (not shown) the heart rate was stable at 85 b.p.m.
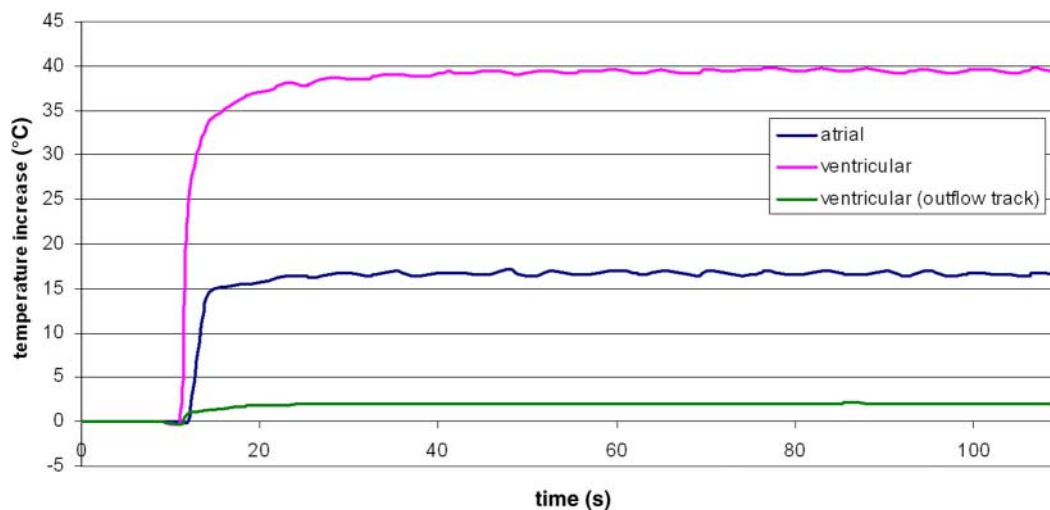

Figure 4 The graph shows the highest temperature increase in Animal 3 with chronically implanted pacemaker leads. The curve shapes are typical for the temperature increase in all animals. The small variations (<1°C) of the temperature curve of the ventricular and atrial leads were consistent with the breathing frequency (10 /min).

could be seen in Animals 3–6. The mean diameter from the scar tissue around the lead tip was 2–3 mm.

No pacemaker resets or changes in programmed parameters during any MRI scan were observed. There was no observable mechanical or electrical damage to the pacemaker components.

Discussion

Our study is the first reported investigation of heating effects of MRI at 1.5 T in pigs with chronically implanted

pacemakers. Thermocouples were chosen as temperature sensors, since they are very thin, non-magnetic, and can easily be integrated into pacing leads. Thermocouple sensor wires may, however, themselves be a potential source of heating as may any long conducting wire in an MRI device.[23,30–32] In an *in vitro* study we verified that no additional heating was produced by the thermocouple sensor integrated into the used pacing leads.

The animal experiments showed heating effects with amplitudes comparable to those measured in *in vitro* measurements.[5,33] The lack of blood flow cooling during the *in vitro* experiment seemed to be compensated by

Table 2 Impedance and stimulation threshold (ST) changes

Animal	Before MRI	Pre- to post-MRI				Pre- to 2 weeks post-MRI			
	Impedance (Ohm)	ST changes (V)			Impedance (Ohm)	ST changes (V)			Impedance (Ohm)
		0.4 ms	0.2 ms	0.06 ms		0.4 ms	0.2 ms	0.06 ms	
RAA									
4	507	0	0.5	−2.5	516	1	1	−3.5	540
5	599	LD	LD	LD	571	LD	LD	LD	599
6	566	1	1	++	557	0	0	0	700
7	446	0.5	1	++	493	0	0.5	0	557
8	472	LD	LD	LD	494	LD	LD	LD	487
9	449	0.5	0.5	0	497	1.5	1	++	503
RVA									
4	600	0.5	0	0.5	721	1	1	2.5	542
5	707	0	−0.5	−0.5	729	2.5	2.5	4	640
6	610	0.5	0.5	2.5	652	1	0.5	2.5	694
7	439	0.5	0	0	517	0.5	0.5	0.5	551
8	534	0	0	0.5	571	0	0	0	571
9	514	2	2	4.5	586	0	0	0	605
RVOT									
4	542	1.5	1	0	569	1	2.5	0	652
5	573	LD	LD	LD	564	LD	LD	LD	658
6	492	0	0	0.5	534	0	0.5	1.5	604
7	550	0	0	0	550	0	0.5	3.5	568
8	468	LD	LD	LD	539	LD	LD	LD	538
9	514	0	0.5	0.5	586	0	0.5	0.5	555

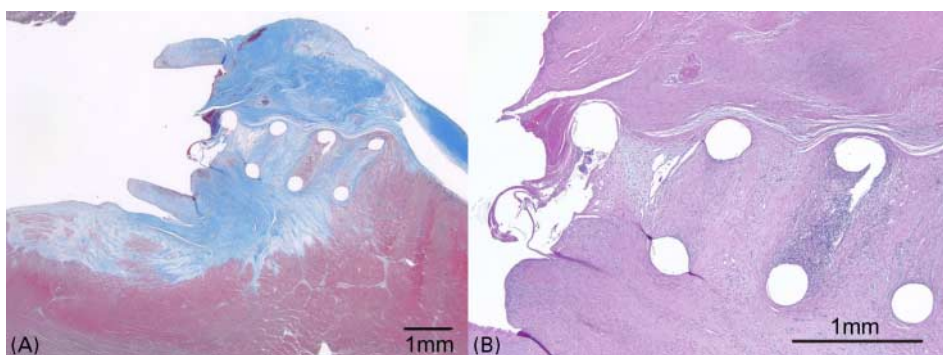++: Pacing threshold out of range (>7.5 V), LD: Lead dislocation.

Figure 5 Histology slices from the RVOT of Animal 9. (*A*) (H&E) Nuclei stain blue. Cytoplasm and other tissue constituents stain varying shades of red/pink. (*B*) (Masson's trichrome). Muscle, keratin, and cytoplasm are stained red. Collagen and mucin stain blue.

reduced heat conduction in cardiac muscle tissue compared with saline water solution. The anatomical placing of the leads near the isocentre (in left–right direction) of the MRI device may further reduce the heating in the animal model.

All three positions within the heart could show high heating effects. These findings do not exclude the possibility that cooling effects due to the blood flow are less pronounced at the RVOT than at the RAA, but other effects such as myocardial perfusion, lead configuration, or proximity of the lead to the RF coil may play a more important role.

Heat-induced tissue damage at the histological level could not be seen with certainty in any of the acute or chronic experiments. In the chronic experiments (Animals 3–9), pathology showed a 2–3 mm thick layer of scar tissue around the lead screw. Such formation of fibrosis and myocardial scar is also known to occur in humans with chronically implanted pacing leads.[34] Much higher temperatures are probably needed to induce further damage in scar tissue. *In vitro* experiments showed that the temperature drops markedly with increasing distance from the lead tip.[33] Comparable results could be found for a bare wire.[35] The expected temperatures 3 mm away from the lead tip will be 2–4 times lower, and hence, below the critical temperature for permanent cell damage. In addition a thicker scar tissue will isolate the lead tip better thermally due to

the lack of perfusion compared with a thin layer. Therefore, different scar tissue thickness may explain why no correlation between threshold changes and measured temperature increases could be seen.

Tissue damage around the pacing lead tip may increase the stimulation threshold, which has been reported in patients with implanted pacemaker systems after undergoing external defibrillation.[36] In our study significant impedance changes were found after the MRI procedure, but these changes did not cause pacemaker dysfunction. The observed changes in stimulation threshold were not likely to be clinically relevant. Due to the voltage steps of 0.5 V for the threshold measurements, smaller changes could have been missed. By more extensive threshold testing, as described by Irnich,[37] it could be possible to observe smaller changes. The threshold changes seen in this study were more pronounced than those seen in patients in the paper from Martin *et al.*[7] However, in this study we investigated worst-case situations (SAR = 3.8 W/kg, long overall scan time and pacemaker system within the body-coil). Two of the threshold changes were reversed to the original values 2 weeks after implantation. The lack of larger pacing parameter changes may be indirect evidence for a lack of extended cellular damage around the lead tip. However, threshold changes may not detect heating-induced damage, and therefore cannot be used as monitoring for long-term effects of heating. For these reasons, *in vitro* testing of lead heating effects using worst-case settings is preferable to demonstrate safety.

The dependencies of the heating effects relative to the lead position within the MRI unit in the direction of the main magnetic field have been evaluated *in vitro*.[5,33] Animal 3 showed the same reduction of heating when the pacing lead was no longer inside the body-coil. If the pacing lead was outside the body-coil, such as in most head and lower extremity scans, the heating was reduced by a factor of more than five. On the other hand, high heating has to be expected for investigations of the heart or spine. This is in good agreement with the publication from Martin *et al.*[7] since all significant stimulation changes could be found in scans either from the heart, neck, or from the abdomen, whereas no changes could be seen for the brain or lower extremity scans.

One of the reported effects of MRI on pacemakers is the potential for rapid pacing. In animal experiments Hayes *et al.*[4] showed heart rates up to the maximal tracking rates of the dual chamber devices used and speculated that this effect was due to over-sensing in the RAA. In our study, Animal 1 showed a stable tachycardia with rates of over 200 b.p.m. ~10 s after beginning the MRI scan. However, the pacemaker used was programmed in the ODO mode, which makes fast pacing induced by the pacemaker very unlikely. It is generally assumed that the frequency of induced voltages from the RF field is too high to stimulate the heart directly. However, if either the pacemaker or the myocardium–lead tip interface rectifies the induced current, then myocardial stimulation may be possible. Stimulation of the heart by the gradient fields in this case is rather unlikely, because the survey scans used comparable gradient

strength. Taking into account the 10–15 s delay between the beginning of the scan and the rapid heart rate, this tachycardia may be induced, or at least supported, by the heating around the lead tip.

Based on these findings, we recommend using continuous ECG and pulsoximetry monitoring to detect fast heart rates when MRI is an absolute necessity and the risk–benefit ratio is considered to be in favour of performing the MRI scan. Immediate discontinuation of the MRI procedure is likely to prevent ventricular fibrillation in such situations. Nevertheless, an experienced cardiologist, capable of interrogating and programming the implanted pacemaker, must be present continuously during the MRI procedure with resuscitation equipment. Imaging of the thorax or spine shows a higher risk regarding heating effects, compared with brain or lower extremity scans.

Study limitations

The use of a pig represents the best anatomical model of the human heart, but the position of the pacing leads within the thorax will be slightly different. The wider thorax of humans and the larger distance between the heart and the pacemaker pocket below the clavicular bone was partly compensated by positioning the pacemaker pocket near the neck of the pig. The position of the pig within the MRI represents the case of MRI investigation of the heart in humans. This study did not investigate the heating effects if the thorax was placed further from the isocentre of the MRI device. The method used for temperature measurements allowed a measurement at only one location. The exact temperature distribution around the lead tip could not be measured using a single sensor. No control animals were available, however, due to the use of steroid-elution lead, pacing parameters are expected to be stable 4 weeks after implantation.

Even in the non-favourable configuration of a heart scan with an SAR-value of 3.8 W/kg, no severe clinical consequences could be seen. Therefore, the absolute contraindication of pacemakers in MRI should be re-evaluated as was also concluded by Martin *et al.*[7] However, as mentioned by Gimbel *et al.*,[1] failing to identify an adverse event is not equivalent to demonstrating safety—especially when only a limited number of patients, or in our case animals, are studied. The potential risk of high heating in cardiac scans could be shown in the results and, in special situations, much higher heating has to be expected.

Conclusion

The measured temperature increase of up to 20°C showed that MRI may produce considerable heating. Changes in stimulation threshold due to MRI in chronic experiments could be shown. The pacing impedance increased significantly during MRI. The location of implanted pacing leads and the position of the animal within the MRI unit may result in marked differences in

heating. Potential risk of tissue damage cannot be totally excluded, even though no reproducible alteration at the histological level could be found. Reduction of the SAR-value by changing MRI sequence and measuring only lower extremities or the head are likely to reduce the risk of MRI-induced heating at the lead tip. Future developments in lead design and technology may reduce the problem of MRI-induced heating.

Acknowledgements

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