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Numerical study of RF exposure and the resulting temperature rise in the foetus during a magnetic resonance procedure

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Abstract

Numerical simulations of specific absorption rate (SAR) and temperature changes in a 26-week pregnant woman model within typical birdcage body coils as used in 1.5 T and 3 T MRI scanners are described. Spatial distributions of SAR and the resulting spatial and temporal changes in temperature are determined using a finite difference time domain method and a finite difference bio-heat transfer solver that accounts for discrete vessels. Heat transfer from foetus to placenta via the umbilical vein and arteries as well as that across the foetal skin/amniotic fluid/uterine wall boundaries is modelled. Results suggest that for procedures compliant with IEC normal mode conditions ($\text{SAR}_{\text{MWB}} \leq 2 \text{ W kg}^{-1}$ (continuous or time-averaged over 6 min)), whole foetal SAR, local foetal SAR\textsubscript{10g} and average foetal temperature are within international safety limits. For continuous RF exposure at $\text{SAR}_{\text{MWB}} = 2 \text{ W kg}^{-1}$ over periods of 7.5 min or longer, a maximum local foetal temperature $> 38 \degree C$ may occur. However, assessment of the risk posed by such maximum temperatures predicted in a static model is difficult because of frequent foetal movement. Results also confirm that when $\text{SAR}_{\text{MWB}} = 2 \text{ W kg}^{-1}$, some local SAR\textsubscript{10g} values in the mother’s trunk and extremities exceed recommended limits.

(Some figures in this article are in colour only in the electronic version)

1. Introduction

MRI is increasingly being used for foetal imaging. Applications include the study of foetal malformations, visualization of pathologies prior to surgical intervention and morphological
Table 1. SAR and temperature limits specified in the IEC (2008) safety standard for three modes of operation of MR equipment.

<table>
<thead>
<tr>
<th>Operating mode</th>
<th>Normal</th>
<th>First-level controlled</th>
<th>Second-level controlled</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whole-body SAR (W kg⁻¹)</td>
<td>2</td>
<td>4</td>
<td>&gt;4</td>
</tr>
<tr>
<td>Partial body SAR (W kg⁻¹)</td>
<td>2–10</td>
<td>4–10</td>
<td>&gt;4 (4–10)</td>
</tr>
<tr>
<td>Head SAR (W kg⁻¹)</td>
<td>3.2</td>
<td>3.2</td>
<td>&gt;3.2</td>
</tr>
<tr>
<td>Local SAR₁₀ｇ (W kg⁻¹)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Head</td>
<td>10</td>
<td>10</td>
<td>&gt;10</td>
</tr>
<tr>
<td>Trunk</td>
<td>10</td>
<td>10</td>
<td>&gt;10</td>
</tr>
<tr>
<td>Extremities</td>
<td>20</td>
<td>20</td>
<td>&gt;20</td>
</tr>
<tr>
<td>Rise in core body temperature (°C)</td>
<td>0.5</td>
<td>1</td>
<td>&gt;1</td>
</tr>
<tr>
<td>Spatially localized temperature limits (°C)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Head</td>
<td>38</td>
<td>38</td>
<td>&gt;38</td>
</tr>
<tr>
<td>Torso</td>
<td>39</td>
<td>39</td>
<td>&gt;39</td>
</tr>
<tr>
<td>Extremities</td>
<td>40</td>
<td>40</td>
<td>&gt;40</td>
</tr>
</tbody>
</table>

changes of the placenta (Prayer et al 2006). Although there is no indication that the use of clinical MR procedures during pregnancy produces adverse effects, the positive proof of safety is difficult to achieve (IEC 2008) and there is currently uncertainty regarding the risk associated with MRI examinations of pregnant patients (ICNIRP 2004, 2009b, MHRA 2007, IEC 2008, HPA 2008, Gowland and De Wilde 2008).

The IEC (2008) safety standard limits specific absorption rate (SAR) and temperature as shown in table 1 for each of three modes of operation of MR equipment. These are (a) the normal mode which causes no physiological stress to patients, (b) the first-level controlled mode which may cause physiological stress to patients that needs to be controlled by medical supervision and (c) the second-level controlled mode in which a significant risk for patients may be produced and for which explicit ethical approval is required. ICNIRP (2004) and MHRA (2007) contain similar exposure guidelines (except that in these cases the limit for SAR averaged over the head is 3 W kg⁻¹). Specific limits on temperature and SAR for the equivalent of the second-level controlled mode are given in HPA (2008). In principle, if the recommended exposure limits in the normal and first-level controlled modes are exceeded, a MR examination can still be performed, but under the conditions of the next higher level mode. However, exposure guidelines regarding imaging of pregnant patients state:

... exposure duration should be reduced to the minimum and ... only the normal operation level is used. (ICNIRP 2004)

Pregnant women should not normally be exposed above the advised lower levels of restriction. (MHRA 2007)

Particular consideration should be made to restrict the use of controlled mode as far as possible for imaging ... pregnant women . . . . (HPA 2008)

Although none of the documents referred to in this paragraph contains a specific limit for the foetus, ICNIRP (2004) states: ‘It seems reasonable to assume that adverse developmental effects will be avoided with a margin of safety if the body temperature of pregnant women...
RF exposure and temperature rise in the foetus during MRI does not rise by more than 0.5 °C and the temperature of the foetus is less than 38 °C. This guidance refers to all modes of operation.

Several authors have reported numerical evaluations of the SAR in models of pregnant females exposed to the RF field from body birdcage coils (Hand et al 2006, Shamsi et al 2006, Stuchly et al 2006, Wu et al 2006, Saito et al 2007, Pediaditis et al 2008, Wang et al 2009). Hand et al (2006) and Pediaditis et al (2008) used the finite integration technique (FIT): the former considered a truncated model of a 28-week pregnant woman exposed to 64 and 127 MHz fields from a shielded birdcage coil whilst the latter considered a whole-body 30-week pregnant female model exposed to fields at 13, 43, 64, 85, 127 and 170 MHz. Both studies showed that local SAR_{10g} (averaged over 10 g of tissue) in the mother exceeded 10 W kg^{-1} before the maternal whole-body averaged SAR (SAR_{WMB}) reached 2 W kg^{-1}, and that axial displacement of the mother’s body by several centimetres inside the coil did not result in a shift of the location of the maximum local SAR_{10g}. The remainder of the studies referred to above used finite difference time domain (FDTD) methods for solving the electromagnetic fields. Stuchly et al (2006) considered a truncated, homogeneous (except for the foetus, foetal brain, placenta and amniotic fluid) model of a 27-week pregnant woman exposed to a 64 MHz birdcage coil and showed that SAR in the foetus was lower than that in the mother. Shamsi et al (2006) and Wu et al (2006) considered nine pregnant female models—one for each month of pregnancy—exposed to fields at 64 and 128 MHz. When normalized to the IEC (2008) whole-body SAR limits (2 or 4 W kg^{-1} for the normal and first-level controlled modes, respectively), the local SAR_{10g} within the mother exceeded the limit of 10 W kg^{-1} at both 64 and 128 MHz. The SAR_{10g} in the foetus exceeded this limit in the case of the first-level controlled mode at 64 MHz. Saito et al (2007) considered a whole-body 28-week pregnant female model exposed to 64 MHz fields and investigated the effect of changing the position of the coil with respect to the body model on foetal SAR averaged over the entire foetus (SAR_{F}), the foetal eye (SAR_{FE}) and foetal brain (SAR_{FB}). An axial shift of the coil of ±150 mm resulted in variations of SAR_{F}, SAR_{FE} and SAR_{FB} of approximately ±6%, ±5% and ±10%, respectively. Wang et al (2009) considered three pregnant woman models representing gestational ages of 3, 6 and 9 months positioned within a 24 rung 64 MHz birdcage coil and reported that the maternal maximum local SAR did not increase significantly with gestational age and that foetal local SAR was <10 W kg^{-1} (when SAR_{WMB} was 2 W kg^{-1}) for all three cases.

Relatively few of these studies modelled temperature changes within the pregnant female model resulting from the exposure to the RF field. Wu et al (2006) used a finite difference (FD) solver to Pennes’ bio-heat equation (Pennes 1948, Lagendijk 1990) with a convective boundary condition at the skin/air interface and predicted that in the case of the normal mode at both 64 and 128 MHz the maximum foetal temperature did not exceed 38 °C. Temperature increases were generally greater during the later stages of pregnancy. In the case of the first-level controlled mode at 64 MHz, the mother’s maximum temperature exceeded 39 °C at 6 months gestation and later, and temperatures in the foetus and amniotic fluid were greater than 38 °C from month 5 onwards. In general greater temperature increases were observed for exposure at 64 MHz compared to those at 128 MHz. Kikuchi et al (2008) modelled the RF exposure and resulting temperature distribution for the 26-week pregnant female model developed by Nagaoka et al (2007) within a 64 MHz birdcage coil. They predicted a maximum foetal temperature of 37.5 °C when the SAR_{WMB} was 2 W kg^{-1}. However, as noted by Gowland and De Wilde (2008), these studies appear not to have accounted for the isolation between foetal circulation and maternal circulation and the fact that under normal conditions the temperature of the foetus is approximately 0.5 °C greater than that of the mother (Laburn 1996, Schröder and Power 1997). Gowland and De Wilde (2008) also discussed likely foetal temperature changes based on heat balance arguments for normal mode and first-level controlled mode.
exposure and highlighted the general lack of information regarding the SAR averaged over the foetal volume during MR procedures.

In this work a commercial FDTD package is used to solve the time-dependent Maxwell’s equations and predict SAR distributions for the case of a pregnant female model positioned within a 16-rung shielded birdcage coil driven at either 64 MHz or 128 MHz. The resulting tissue temperatures are predicted using a commercial FD solver for the bio-heat equation that includes the ability to account for heat transfer between discrete blood vessels and the local tissue.

2. Methods

2.1. Birdcage coil model

A generic low-pass, 16-rung, shielded circular birdcage coil was modelled. The coil was 0.6 m in diameter and the end rings were 10 mm wide with an axial centre–centre spacing of 0.4 m. The rungs were 10 mm × 10 mm square section rods with their centres located on a circle of radius 0.3 m. To reduce parasitic capacitance, four capacitors (\(C_{\text{rung}}\)) were associated with each rung and were placed in 5 mm gaps between the rungs and the end rings and ±55 mm from the mid-point of each rung. Sixteen capacitors (\(C_{\text{end-ring}}\)) were also inserted in similar gaps in the end rings, midway between rungs. The coil was driven in quadrature by two voltage sources located across 5 mm gaps in the rungs at angular positions 135° and 225° relative to the vertical (y) axis. The coil was shielded by a 1.0 m long metallic cylinder of thickness 1 mm and internal radius 0.339 m. All metallic conductors were assumed to have a conductivity of \(5.997 \times 10^7\) S m\(^{-1}\). The coil was tuned to 64 MHz or 128 MHz by adjusting the values of \(C_{\text{end-ring}}\) and \(C_{\text{rung}}\). A coordinate system is defined with origin at the geometrical centre of the coil.

2.2. Whole-body pregnant female model

The pregnant female model used was the whole-body human database of the Japanese average pregnant woman jointly developed by the National Institute of Information and Communications Technology, Tokyo, and Chiba University and described by Nagaoka et al. (2007). This model of a 26-week pregnant woman was based on MR data from the abdomen of a pregnant volunteer from which a model of the foetus was constructed; this was then combined with a whole-body voxel model of a non-pregnant woman (Nagaoka et al. 2004). The model consisted of approximately \(7 \times 10^6\) cells, each 2 mm × 2 mm × 2 mm, segmented into 56 tissue types. The height and weight of the pregnant female were 1.6 m and 58.4 kg and the masses of the foetus and placenta were 826 g and 400 g, respectively. A sagittal section of the model in the plane \(x = 0.0125\) m is shown in figure 1(a), and the position of the model relative to the birdcage coil and shield, such that the foetal brain is located at the isocentre of the coil, is shown in figure 1(b). Physical properties of foetal and peri-foetal tissues used in the simulations are listed in table 2. The dielectric properties of foetal tissues were those proposed by Hand et al. (2006). The properties of other tissues were taken to be those reported by Nagaoka et al. (2004, 2007). Temperature dependences of tissue properties are not taken into account in the present study.

2.3. Numerical simulations

2.3.1. Electromagnetic. Numerical simulations of the electromagnetic problem were carried out using the FDTD solver within the SEMCAD X v14.0 (Schmid & Partner Engineering
Figure 1. (a) Sagittal section (y–z plane at x = 0.0125 m) of the pregnant female model highlighting the foetus, foetal brain, amniotic fluid, placenta and uterine wall. The foetal brain is located at the origin of coordinates, which is defined to be at the centre of the birdcage coil. (b) Location of the pregnant female model within the shielded birdcage coil. Left: top view; right: side view.

Table 2. Physical properties of foetal and peri-foetal tissues used in the models. \( \varepsilon \) is the relative permittivity, \( \sigma \) is the electrical conductivity, \( k \) is the thermal conductivity, \( c \) is the specific heat, \( M \) is the volumetric metabolic rate, \( w \) is the perfusion and \( \rho \) is the density.

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Amniotic fluid</th>
<th>Foetal brain</th>
<th>Foetus</th>
<th>Placenta</th>
<th>Uterine wall</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \varepsilon ) \n64 MHz</td>
<td>97.3</td>
<td>97.2</td>
<td>94.2</td>
<td>72.0</td>
<td>92.1</td>
</tr>
<tr>
<td>128 MHz</td>
<td>84.2</td>
<td>76.6</td>
<td>73.6</td>
<td>64.0</td>
<td>75.4</td>
</tr>
<tr>
<td>( \sigma ) \n(S m(^{-1})) \n64 MHz</td>
<td>2.07</td>
<td>0.76</td>
<td>0.92</td>
<td>0.71</td>
<td>0.91</td>
</tr>
<tr>
<td>128 MHz</td>
<td>2.14</td>
<td>0.83</td>
<td>1.00</td>
<td>0.74</td>
<td>0.96</td>
</tr>
<tr>
<td>( k ) \n(W m(^{-1}) C(^{-1}))</td>
<td>0.50</td>
<td>0.53</td>
<td>0.39</td>
<td>0.50</td>
<td>0.50</td>
</tr>
<tr>
<td>( c ) \n(J kg(^{-1}) C(^{-1}))</td>
<td>3840</td>
<td>3672</td>
<td>3195</td>
<td>3840</td>
<td>3434</td>
</tr>
<tr>
<td>( M ) \n(W m(^{-3}))</td>
<td>0</td>
<td>3399</td>
<td>3432</td>
<td>2222</td>
<td>2209</td>
</tr>
<tr>
<td>( w ) \n(kg m(^{-3}) s(^{-1}))</td>
<td>0</td>
<td>4.55</td>
<td>4.59</td>
<td>32.90</td>
<td>5.15</td>
</tr>
<tr>
<td>( \rho ) \n(kg m(^{-3}))</td>
<td>1055</td>
<td>1030</td>
<td>1040</td>
<td>1058</td>
<td>1052</td>
</tr>
</tbody>
</table>

AG, Zurich) software package to predict spatial distributions of E-fields, H-fields and SAR. Mass or volume averaged values of SAR, namely SAR\(_{10g}\), SAR\(_{M WB}\) and SAR averaged over specified tissue types, were calculated. SAR\(_{10g}\) was calculated according to IEEE (2002).

2.3.2. Thermal. Temperature changes resulting from the exposure to the electromagnetic fields were simulated using Pennes’ thermal solver within SEMCAD X. This provided a finite difference solution to the bio-heat equation:

\[
\frac{\partial T}{\partial t} = \nabla \cdot (k \nabla T) + \sigma |E|^2 + M - P_o(T - T_b),
\]

where \( c \) is the specific heat capacity (J kg\(^{-1}\) C\(^{-1}\)), \( \rho \) is the tissue density (kg m\(^{-3}\)), \( k \) is the thermal conductivity (W m\(^{-1}\) C\(^{-1}\)), \( \sigma \) is the electrical conductivity (S m\(^{-1}\)), \( E \) is the local electric field (V m\(^{-1}\)), \( M \) is the metabolic rate (W m\(^{-3}\)), \( P_o \) is the blood perfusion coefficient.
(W m\(^{-3}\) C\(^{-1}\)), \(T_b\) is the blood temperature (°C) and \(T\) is the local tissue temperature. \(P_o\) can be expressed as \(P_o = w c_b\) where \(w\) is the blood mass perfusion rate (kg m\(^{-3}\) s\(^{-1}\)) and \(c_b\) is the specific heat of blood. The thermal solver enables various thermal boundary conditions to be imposed.

Heat loss by the foetus to the mother is achieved via two major routes, namely heat transfer from foetal to maternal blood in the placenta, and that across the foetal skin/amniotic fluid and amniotic fluid/uterine wall boundaries. Studies in sheep indicate that the first of these routes accounts for approximately 80% of the foetal heat loss (Gilbert et al. 1985, Schröder et al. 1988) and limited data suggest that a similar thermal relationship exists between the human foetus and mother (Laburn 1996).

To account for heat exchange due to the umbilical vein and arteries, the one-dimensional temperature profile along a vessel was discretized and the thermal interaction between vessel and surrounding tissue and the vessel wall temperature were estimated using a method similar to that described by Kotte et al. (1996). Implementation of this in the SEMCAD thermal solver is available using the DIscrrete VAsculature (DIVA) option (Neufeld 2008). The umbilical vessels were assumed to be circular in cross section; the diameter of the vein was 6 mm and that of the arteries was 4 mm (Sutton et al. 1990). A cross section of the umbilical vessels (in the \(y-z\) plane) and their relative positions are shown in figure 2. The vein was centred on a spline that followed an arbitrary path from the umbilicus of the foetus, around the foetal limbs, to the placenta with variable \(x\) and \(y\) but constant \(z\) coordinates and of total length 30 cm. The arteries were centred on similar splines displaced 4 mm in \(-y\) and \(\pm 7\) mm in \(z\) with respect to the original spline. A perspective view of the foetus, tracks of the umbilical vessels and placenta is also shown in figure 2. This simple model ignored the coiled nature of the umbilical cord. A further approximation was that the dielectric properties of Wharton’s jelly within the umbilical cord were taken to be the same as those for amniotic fluid on account of its high water content (Scott and Wilkinson 1978) and similar conductivity to amniotic fluid (Oostendorp et al. 1989). This simplification avoided the need to include a sheath around the three umbilical cord vessels. The volumetric flow in the umbilical vein was taken to be \(2 \times 10^{-6}\) m\(^3\) s\(^{-1}\) (Boito et al. 2002) and that in each of the arteries to be \(1 \times 10^{-6}\) m\(^3\) s\(^{-1}\). \(T_b\) was 37 °C for maternal tissues and 37.5 °C for foetal tissues; the temperatures of blood entering the umbilical vein and arteries were assumed to be 37 °C and 37.5 °C, respectively. At the maternal skin/air boundary the heat transfer coefficient was assumed to be 10.5 W m\(^{-2}\) °C\(^{-1}\) and the heat flux was 20 W m\(^{-2}\). The ambient temperature was taken to be 24 °C, the maximum
RF exposure and temperature rise in the foetus during MRI

3. Results

3.1. SAR

At both 64 MHz and 128 MHz, the maximum SAR_{10g} in each of maternal blood, bone, fat, muscle and skin exceeded the appropriate local SAR_{10g} limit (10 and 20 W kg\(^{-1}\) in the trunk and extremities, respectively) when the SAR_{MWB} = 2 W kg\(^{-1}\). In both cases the maximum local SAR_{10g} occurred in the left wrist. In practice the peak local SAR in the arms and wrists is highly dependent upon position. Furthermore, SAR is defined as being pro-rata with the fraction of 6 min for which there is exposure. Figure 3 shows distributions of SAR_{10g} resulting from continuous exposure to the 64 MHz coil. Exposure to 128 MHz resulted in a qualitatively similar distribution of SAR_{10g}.

Table 3 lists SAR values in foetal and peri-foetal tissues when SAR_{MWB} = 2 W kg\(^{-1}\). Relatively high SAR occurs in the amniotic fluid and uterine wall compared with that in foetal tissues. The spatially averaged SARs over the volumes of foetal tissues and the SAR_{10g} maxima in these tissues are similar at both frequencies. The SAR data for amniotic fluid, placenta and uterine wall in general suggest higher values at 64 MHz compared to 128 MHz. In most cases the differences at the two frequencies are comparable with the changes in SAR that have been reported due to axial shifts of several centimetres in the relative positions of coil and body (Hand et al 2006, Saito et al 2007).

3.2. Thermal modelling

Temperature distributions were calculated at intervals of 300 s over a period of 3600 s. To determine baseline temperatures, the temperature solver was run in the absence of RF power. The resulting mean steady-state temperatures in the foetal tissues were \(\sim 37.5 \, ^\circ\text{C}\). The variation with time of mean and maximum temperatures in foetus (excluding brain), foetal...
Table 3. SAR in foetal and peri-foetal tissues assuming that SAR_{MWB} is 2 W kg$^{-1}$.

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Foetal brain</th>
<th>Amniotic fluid</th>
<th>Placenta</th>
<th>Uterine wall</th>
</tr>
</thead>
<tbody>
<tr>
<td>Foetus</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAR averaged over tissue mass (W kg$^{-1}$)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>64 MHz</td>
<td>1.17</td>
<td>3.36</td>
<td>0.63</td>
<td>2.55</td>
</tr>
<tr>
<td>128 MHz</td>
<td>1.05</td>
<td>2.74</td>
<td>0.40</td>
<td>2.38</td>
</tr>
<tr>
<td>Maximum SAR$_{10g}$ (W kg$^{-1}$)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>64 MHz</td>
<td>5.47</td>
<td>12.01</td>
<td>2.54</td>
<td>14.96</td>
</tr>
<tr>
<td>128 MHz</td>
<td>5.32</td>
<td>13.09</td>
<td>1.33</td>
<td>11.48</td>
</tr>
</tbody>
</table>

In the first set of simulations in which RF was present, a sequence in which SAR$_{MWB}$ was 2 W kg$^{-1}$ for 900 $\leq$ $t$ $\leq$ 2700 s and 0 W kg$^{-1}$ for $t$ $<$ 900 and $t$ $>$ 2700 s was used. In the second set of simulations, temperatures were calculated at 60 s intervals over a period of 1560 s during which SAR$_{MWB}$ was 2, 3 and 4 W kg$^{-1}$ for 360, 210 and 180 s, respectively, beginning at $t$ = 900 s and zero thereafter. These simulate simple power management strategies designed to ensure a net operation at 2 W kg$^{-1}$ averaged over 6 min. Finally, a calculation that simulated the SAR averaged over the duration of a particular clinical examination in which SAR$_{MWB}$ was 0.75 W kg$^{-1}$ for a period of 4200 s beginning at $t$ = 900 s was carried out.

Table 4 lists temperatures predicted in foetal and peri-foetal tissues following 600 and 1800 s of RF exposure, at both 64 and 128 MHz, for which SAR$_{MWB}$ = 2 W kg$^{-1}$. The mean temperature averaged over a specific tissue volume and the maximum temperature within that tissue volume are listed, as are the increases in these parameters measured with respect to their values at the moment the RF exposure begins. Mean foetal temperatures show an increase of approximately 0.2°C and remain below 38°C as recommended by ICNIRP (2004, 2009b), MHRA (2007) and HPA (2008) for both frequencies, even for a relatively long continuous exposure of 1800 s. Since the uterine wall is subject to a higher SAR, a larger increase in its mean temperature (up to $\sim$0.3°C) is predicted, whilst the highly perfused placenta undergoes a smaller temperature change. The greatest temperature change of up to $\sim$1°C is seen in the highly conducting amniotic fluid.

Figure 4 shows the time dependence of mean and maximum temperatures in foetal and peri-foetal tissues during 1800 s of continuous RF exposure at 64 MHz and 128 MHz. All mean temperatures remain below 38°C. The maximum temperature in the foetus, which occurs at the interface between the foetal head and amniotic fluid, exceeds 38°C after approximately 450 s of continuous exposure and is predicted to reach 38.9°C (for 64 MHz) and 38.7°C (for 128 MHz) after 1800 s. The maximum temperature within the uterine wall remains below 39°C although that in amniotic fluid reaches $\sim$40.1°C for 1800 s exposure at both 64 and 128 MHz.

Figure 5 shows the predicted time-dependent maximum and mean temperatures in the foetus, foetal brain and amniotic fluid for three cases in which the SAR$_{MWB}$ averaged over 6 min, as permitted by IEC (2008), is 2 W kg$^{-1}$. The shorter durations of these continuous exposures are more aligned with those used in practice. In all cases, the temperature of foetal
Table 4. Predicted temperatures in foetal and peri-foetal tissues assuming that SAR\text{whole body} = 2 W kg\(^{-1}\). Mean temperatures are averaged over the tissue type. Temperature changes \(\Delta_{\text{meanT}}\) and \(\Delta_{\text{maxT}}\) are differences in predicted mean and maximum temperatures at \(t = 1500\) or \(2700\) s relative to those at \(t = 900\) s, the time at which RF exposure begins, and correspond to RF exposures of 600 or 1800 s.

<table>
<thead>
<tr>
<th>Tissue/RF exposures</th>
<th>64 MHz</th>
<th>128 MHz</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean temp. ((^\circ)C)</td>
<td>(\Delta_{\text{meanT}}) ((^\circ)C)</td>
</tr>
<tr>
<td>Foetus (excluding brain)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>600 s</td>
<td>37.6</td>
<td>0.15</td>
</tr>
<tr>
<td>1800 s</td>
<td>37.7</td>
<td>0.24</td>
</tr>
<tr>
<td>Foetal brain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>600 s</td>
<td>37.6</td>
<td>0.16</td>
</tr>
<tr>
<td>1800 s</td>
<td>37.6</td>
<td>0.19</td>
</tr>
<tr>
<td>Placenta</td>
<td></td>
<td></td>
</tr>
<tr>
<td>600 s</td>
<td>37.0</td>
<td>0.02</td>
</tr>
<tr>
<td>1800 s</td>
<td>37.0</td>
<td>0.03</td>
</tr>
<tr>
<td>Uterine wall</td>
<td></td>
<td></td>
</tr>
<tr>
<td>600 s</td>
<td>37.3</td>
<td>0.23</td>
</tr>
<tr>
<td>1800 s</td>
<td>37.4</td>
<td>0.32</td>
</tr>
<tr>
<td>Amniotic fluid</td>
<td></td>
<td></td>
</tr>
<tr>
<td>600 s</td>
<td>37.5</td>
<td>0.47</td>
</tr>
<tr>
<td>1800 s</td>
<td>38.0</td>
<td>0.95</td>
</tr>
</tbody>
</table>
Figure 4. Variation of mean (open symbols) and maximum (solid symbols) temperatures in foetal (solid lines) and peri-foetal (dashed lines) tissues with duration of exposure to RF at 64 MHz (upper) and 128 MHz (lower) for continuous $\text{SAR}_{\text{MWB}} = 2 \text{ W kg}^{-1}$. Foetus (○, ●), foetal brain (♦, □), amniotic fluid (□, ■), uterine wall (△, ▲) and placenta (×, ⋆). The maximum temperature in amniotic fluid at 1800 s was $\sim 40.1 ^\circ \text{C}$ for both 64 and 128 MHz exposures.

Figure 6 shows an example of $\text{SAR}_{\text{MWB}}$ versus time recorded during a clinical foetal examination for which the mean SAR over a period of 4200 s was 0.75 W kg$^{-1}$. The predicted mean temperature in both foetus and foetal brain at $t = 4200$ s was 37.6 $^\circ \text{C}$, with corresponding maximum local temperatures 37.9 and 37.7 $^\circ \text{C}$, respectively.

4. Discussion

The results of the SAR simulations suggest that foetal exposure due to the RF fields from these generic birdcage coils does not differ greatly for operation at 64 and 128 MHz (coil
Figure 5. Temperature versus time relationships for foetus (○, ●), foetal brain (□, ●) and amniotic fluid (□, ■) due to short exposures at differing SAR_{MWB}. Mean temperatures (open symbols) and maximum temperatures (solid symbols) are shown. Exposures are (from top to bottom): SAR_{MWB} = 2 W kg\(^{-1}\) for 360 s, SAR_{MWB} = 3 W kg\(^{-1}\) for 240 s, SAR_{MWB} = 4 W kg\(^{-1}\) for 180 s. Data for 64 MHz and 128 MHz are shown in the left-hand, right-hand columns, respectively.

When SAR_{MWB} is 2 W kg\(^{-1}\), the SAR averaged over the entire foetus (including foetal brain) SAR\(_F\) is 1.24 and 1.14 W kg\(^{-1}\) at 64 and 128 MHz, respectively. The corresponding maximum foetal SAR\(_{10g}\) values are approximately 5.5 and 5.3 W kg\(^{-1}\). These maximum local SARs are in good agreement with the data of Shamsi \textit{et al} (2006) who predicted the maximum SAR\(_{10g}\) in a foetus at \(~\)26 weeks exposed to a 64 MHz birdcage coil to be \(~\)6 W kg\(^{-1}\) when SAR_{MWB} was 2 W kg\(^{-1}\). Wu \textit{et al} (2006), using a similar coil model driven at 128 MHz, found the corresponding maximum SAR\(_{10g}\) to be \(~\)3 W kg\(^{-1}\). Pediaditis \textit{et al} (2008) also found that foetal SARs were slightly greater at 64 MHz compared to 128 MHz when normalized to the same SAR_{MWB}. However they found that SAR_{MWB} and SAR\(_F\) were similar, and that the maximum SAR\(_{10g}\) in the foetus was approximately six times
greater than SARF. Saito et al (2007), using the same pregnant woman model as in the work described here within an 8 rung 64 MHz birdcage coil, predicted that the spatially averaged SAR in foetal brain was approximately 30–50% greater than that averaged over other foetal tissues depending upon the position of the coil relative to the mother and foetus. The present work not only confirms this finding at 64 MHz but also extends it to 128 MHz.

Some of the differences in results obtained in the studies referred to above arise from the use of differing mother/foetus models, dielectric properties (particularly for foetal tissues), coil designs and dimensions and positions of the body models relative to the coil. For example, the dependence of SAR on gestational age has been investigated by Wu et al (2006) and the use of different pregnant woman models for plane wave dosimetry has been discussed by Dimbylow et al (2009). Other studies have shown that shifts of several centimetres in the axial position of the body model relative to a birdcage coil result in only minor changes in the location of the maximum local SAR, suggesting that the intracorporal SAR distribution is primarily dependent on the dielectric properties of the tissues (Hand et al 2006), but do affect SAR values averaged over the foetal eyes, foetal brain and the foetus itself as well as those for local SAR by up to 10% (Saito et al 2007). The SAR_{WWB} is insensitive to such changes in the relative positions of coil and body.

Gabriel and Peyman (2006) discussed uncertainty in the measurement of dielectric properties. As examples, overall uncertainties in measuring the permittivity $\varepsilon$ and conductivity $\sigma$ of porcine liver and fat over the frequency range 50–300 MHz were 1.5% (for $\varepsilon$) and 2.8% (for $\sigma$) and 7.1% (for $\varepsilon$) and 10.6% (for $\sigma$), respectively. Hurt et al (2000) and Gajšek et al (2001) studied far-field exposure of whole-body models and found that the uncertainty in complex permittivity had relatively little effect on whole-body SAR values but did influence local SAR values significantly, particularly when these were low compared with the whole-body SAR value, and for muscle tissue which represented a substantial proportion of the body. The effect of the size of Yee cells (ranging from 8 to 100 cells per cm$^3$) on predicted SAR in a FDTD model of a head exposed to the near field of a 64 MHz saddle coil was studied by Collins and Smith (2003). Although the maximum SAR in a single cell increased as the cell size was reduced, the 1 cm$^3$-averaged and whole-head-averaged SARs varied only by 16% and 7%, respectively, for the cell sizes studied. Laakso (2009) studied far-field exposure of the brain and eyes and commented that 2 mm resolution might be sufficient for frequencies below 2.5–3 GHz since SAR and temperature predictions obtained at this resolution differed from

![Figure 6. Maternal whole-body SAR versus time—data from a clinical foetal examination.](image)
RF exposure and temperature rise in the foetus during MRI

those derived at higher resolution (0.5 mm) by less than 10%. Interpreting SAR data predicted using a single voxel model (or from all of the relatively few voxel models available) in terms of safety for the general population is associated with greater uncertainty. For example, Conil et al. (2008) investigated the variation of FDTD predicted SAR in six adult models (with 2 mm voxels) exposed to plane waves over the frequency range from 20 MHz to 2.4 GHz. At 100 MHz, the deviation of whole-body SARs of specific models varied from the mean value of the six models by up to +40% and −35%. The standard deviation of the SAR averaged over the torso was approximately 25% of the mean value at 100 MHz. Dimbylow et al. (2009) reported on simulations of plane wave exposure of three pregnant women models using different FDTD codes. When the same assumptions regarding dielectric properties and SAR averaging algorithms were used, differences of results were generally within ∼5%. However, the foetal SAR predicted in specific models varied by a factor of approximately 2 due to the variability in gestational age and foetal position within models.

Although previously reported MRI foetal dosimetry studies vary in detail, there are several findings that are common to most. These include the following: the maximum local SAR_{10g} in the mother’s extremities and trunk exceeds the relevant limits in exposure guidelines (ICNIRP 2004, MHRA 2007) and the IEC (2008) safety standard when SAR_{MWB} is 2 W kg\(^{-1}\) (as is the case for non-pregnant subjects), the maximum SAR_{10g} in the mother is at least several times that in foetal tissues, and for operation within the IEC (2008) safety standard normal mode (SAR_{MWB} = 2 W kg\(^{-1}\)), the foetal maximum SAR_{10g} is < 10 W kg\(^{-1}\). Thus, in terms of SAR_{10g}, the evidence suggests that foetal exposures due to MR procedures using birdcage coils operated in the normal mode are compliant with ICNIRP (2004, 2009b) and MHRA (2007) exposure guidelines, HPA (2008) recommendations and the IEC (2008) safety standard. Our results are similar to those of Pediaditis et al. (2008) who raised the issue of the appropriate limit to be applied to foetal exposure. They commented that some European countries may use ICNIRP (1998, 2009a) exposure limits recommended for health protection of the general population. However, in the case of MR-related exposure, such restriction appears to be intended to limit exposure of pregnant staff (IEC 2008). In the current work we have considered safety rather than health protection guidelines on the grounds that during deliberate MR imaging of pregnant subjects the foetus is part of the examination.

As shown in table 4, the predicted increase in the mean temperatures of the foetus and foetal brain is ∼0.15 °C and ∼0.2 °C following 600 s and 1800 s of continuous RF exposure, respectively. These values are in reasonable agreement with the predictions of Gowland and De Wilde (2008). These authors used an analytical model and suggested increases in foetal temperature of 0.2 °C and 0.3 °C following 600 s and 3600 s, respectively, of RF exposure associated with SAR_{MWB} of 2 W kg\(^{-1}\). Our predicted mean temperatures of foetal and peri-foetal tissues are below 38 °C as recommended by ICNIRP (2004). However, the maximum temperature predicted in foetal tissues exceeds 38 °C by ∼0.2 °C following 600 s exposure and by ∼0.7–0.9 °C after 1800 s. The maxima in uterine wall and placenta are compliant with the IEC (2008) limit of 39 °C in the trunk but the maximum in amniotic fluid exceeds that limit by up to ∼1 °C. Wu et al. (2006) predicted maximum temperatures of ∼37.6 °C in the foetus, ∼37.1 °C in placenta, ∼37.3 °C in the uterus and ∼37.9 °C in amniotic fluid for a 26-week foetus and normal mode operation at 64 MHz. Kikuchi et al. (2008) predicted a maximum foetal temperature of 37.5 °C in the same pregnant woman model as we have used for normal mode operation at 64 MHz. Their results also suggested that the temperature of the foetal body exceeds that of foetal brain. However, both of these studies appear not to have accounted for the fact that the foetal temperature normally exceeds that of the mother by ∼0.5 °C, and the duration of exposure is not stated.
Foetal MRI is constrained by motion artefacts arising from movement of the foetus which can be so vigorous that almost all foetal examinations use single shot methods designed to freeze foetal motion in individual slices (Levine et al 2006, Fulford and Gowland 2009). In view of such frequent and significant foetal movement over a time scale of several minutes, cautious interpretation is required of the predicted maximum foetal temperature in particular, and also of the maximum foetal SAR10g which may be time averaged over a period of 6 min, both of which are based on the assumption of a stationary foetus. For this reason average foetal temperature may provide a more realistic assessment of risk until more detailed knowledge of the time dependence of local temperature and SAR10g that accounts for realistic foetal motion becomes available.

Hand et al (1999) commented on the challenges involved to achieve accurate quantitative agreement between predicted temperatures and real ones in view of the number of parameters that influence predicted values. The assumptions inherent in Pennes’ (1948) formulation of the bio-heat transfer equation (BTE) and their consequences have been discussed at length in the thermal modelling literature (Wulff 1974, Arkin et al 1994, Van Leeuwen et al 2000). In particular, Pennes’ model assumes that all heat exchange between vessels and tissue takes place in the capillaries—in contrast to conclusions drawn from subsequent studies by Chen and Holmes (1980). Models that describe vessels discretely (Baish et al 1986, Huang et al 1996, Kotte et al 1996) offer a more accurate means of estimating heat transport within tissues. However, Collins (2009) commented that these more complex thermal models may result in a less conservative estimate of temperature increase for the purposes of ensuring safety. In the case of maternal/foetal heat transfer, the umbilical vessels present a highly significant parallel pathway for heat transfer in addition to that occurring across the foetal skin/amniotic fluid/amniotic fluid/uterine wall boundaries.

Nguyen et al (2004) compared temperatures measured in a head phantom exposed to the RF field of a 63 MHz head coil with those predicted using the bio-heat transfer equation (BTE) and found RMS differences of approximately 10–14%. Raaymakers et al (2000) compared temperature measurements made in a perfused bovine tongue with predictions made using BTE and a discrete vessel (DIVA) thermal model. For temperature increases of approximately 10 °C, the maximum difference between the DIVA predictions and measurements was ~1 °C, and DIVA predicted temperature profiles were in better agreement with measurements than BTE predictions. Hand et al (1999) studied heating of a human leg due to a 64 MHz butterfly surface coil and compared BTE predicted temperatures with those measured on the skin. With 2 W applied to the coil for periods up to ~30 min, the maximum predicted and measured increases in temperature were 1 °C and 1.2 °C, respectively, although for longer periods of heating there was a tendency for the predictions to underestimate measurements by approximately 0.2–0.3 °C. Moros et al (1993) investigated ultrasound induced heating in dog thighs and found that agreement between BTE predicted and measured local temperatures was better than ~0.7 °C (for temperature elevations of ~5 °C) away from large, thermally significant vessels.

In most reports on mother/foetus dosimetry for MRI exposures, discussion is restricted to the normal mode of operation for which SAR_{MWB} = 2 W kg⁻¹. However, Wu et al (2006) and Shamsi et al (2006) also considered the IEC (2008) first controlled mode for which SAR_{MWB} = 4 W kg⁻¹ and reported that at 64 MHz, foetal SAR_{10g} > 10 W kg⁻¹ and the maximum foetal temperature was >38 °C after the fifth month. For 128 MHz exposure, foetal SAR_{10g} and maximum foetal temperature were compliant with IEC (2008), ICNIRP (2004) and MHRA (2007) limits. Our results extrapolated to a whole-body SAR of 4 W kg⁻¹ are more restrictive in that at both 64 and 128 MHz the maximum foetal SAR_{10g} would exceed 10 W kg⁻¹ and that, for continuous exposure of >180 s the maximum foetal temperature is
predicted to exceed 38 °C. In the case of longer continuous exposures (600 s) the mean foetal temperature is predicted to be close to 38 °C. Gowland and De Wilde (2008) also refer to the IEC (2008) normal mode limit for partial body exposure which is $\text{SAR}_{\text{partialbody}} = 10 - (8 \times \text{exposed patient mass/total patient mass}) \ W \ kg^{-1}$. For example if ~25% of the maternal mass is exposed, the limit on $\text{SAR}_{\text{partialbody}} = 8 \ W \ kg^{-1}$. Extrapolation of data in table 4 suggests that at this SAR, the mean foetal temperature would increase by 0.6 °C after 600 s exposure and would exceed 38 °C. However, when $\text{SAR}_{\text{MWB}} \leq 4 \ W \ kg^{-1}$ for periods such that the time-averaged $\text{SAR}_{\text{MWB}}$ over 6 min is 2 $W \ kg^{-1}$ our results (figure 5) show that foetal temperatures remain $\leq 38 ^\circ C$.

The results of this study suggest that MRI procedures carried out under normal mode conditions in which $\text{SAR}_{\text{MWB}} \leq 2 \ W \ kg^{-1}$ are associated with foetal SAR which is compliant with limits in exposure guidelines (ICNIRP 2004, MHRA 2007, HPA 2008) and the IEC (2008) safety standard. This is in general agreement with previous similar dosimetric studies. Furthermore, the predicted temperature changes averaged over foetal tissues are also in line with ICNIRP (2004) recommendations, even when allowing ~20% uncertainty typical in predicted temperatures. The maximum foetal temperature is predicted to be close to 38 °C when $\text{SAR}_{\text{MWB}} = 2 \ W \ kg^{-1}$ for periods of ~450 s or longer. When operating at these RF levels, the time structure of clinical examinations and the total duration of foetal exposure can have significant effects in decreasing both the 6 min time averaged SAR (figure 6) and the attendant temperature changes. Finally, this study, in accord with many reports of numerical modelling of SAR due to body birdcage coils, indicates that the maximum local $\text{SAR}_{10g}$ in the mother’s extremities and trunk exceeds the relevant recommended limits when $\text{SAR}_{\text{MWB}} = 2 \ W \ kg^{-1}$. A further level of protection could be afforded to the mother by limiting exposure to a maximum $\text{SAR}_{10g}$ of 10 $W \ kg^{-1}$ in the trunk or 20 $W \ kg^{-1}$ in the extremities rather than to $\text{SAR}_{\text{MWB}} = 2 \ W \ kg^{-1}$. This would also reduce foetal SAR and consequent temperature increase. However, to be feasible in practice this would require more rapid prediction of the SAR distribution on a patient-specific basis than is currently possible.

Our results are based on simulations of a single static model with a number of inherent assumptions and uncertainties. Improvements that could be made to the model include a time-dependent $T_{\text{blood}}$ parameter in the bio-heat transfer equation to account for maternal core temperature changes, a longer, coiled, sheathed umbilical cord and temperature-dependent perfusion values. Sensitivity analyses for a range of parameter values (e.g. reduced umbilical flow due to restriction in these vessels) would also improve confidence in interpretation of results. However, accounting for foetal movement and reports of similar simulations using a variety of pregnant women models are expected to make a greater contribution to more realistic assessment of foetal exposure during MR examinations.

5. Conclusions

Numerical simulations of SAR and temperature changes in a pregnant woman model within birdcage body coils typical of those used in 1.5 T and 3 T MRI scanners have been performed. In particular, a thermal model that accounts for heat transfer from the foetus to the placenta via the umbilical vein and arteries was used. Results suggest that for MRI procedures carried out under IEC normal mode conditions such that the maternal whole-body averaged SAR is $\leq 2 \ W \ kg^{-1}$, foetal whole-body SAR is 1.24 and 1.14 $W \ kg^{-1}$ at 64 and 128 MHz, respectively. Furthermore, the average temperature of the foetus remains below 38 °C although the maximum temperature predicted in foetal tissues may exceed this value following continuous exposures of ~7.5 min or longer. However, assessment of risk posed by such maximum temperatures predicted using a static foetal model is difficult in view of frequent
foetal movement. Since it is generally recognized that a SAR_{SWM} of 2 W kg\(^{-1}\) is expected to result in SAR\(_{10g}\) values in the mother that exceed the limits recommended for the trunk and extremities, a further level of protection could be afforded limiting exposure to a maximum SAR\(_{10g}\) of 10 W kg\(^{-1}\) in the trunk or 20 W kg\(^{-1}\) in the extremities.

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