

Effect of Considering Physiological Response in Temperature Calculations for MRI of the Human Head

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Introduction

In previous simulations of temperature for MRI of the human head (1,2), no consideration of physiological response to temperature was included, as it sometimes is in other fields of study (3). Here we present a numerical comparison of the SAR and the temperature elevation in a model of a human head in a TEM coil from 64MHz to 400 MHz both with and without consideration of temperature-induced changes in rates of metabolism and perfusion. We investigate a more thorough mathematical model (including physiological response) into our MRI coil work. Results indicate that the effect of considering the physiological response is very small for an average SAR over the head near the limits currently allowable, but are more significant at higher SAR levels.

Method

The TEM coil and the investigation were formulated using a home-built implementation of the FDTD method (4). All models had a resolution of 3mm in each direction. An eight-layer Berenger's PML was implemented at the absorption boundary. A 4-Cole-Cole extrapolation technique was used to determine values for the dielectric properties of the tissues at different frequencies (4). The tissue parameters used in the computer simulation were acquired from the Brooks Air Force Laboratory database. The coil had 16 copper elements, a 30-cm inner diameter, and a 16-cm length. The diameter and length of the shield were 38 cm and 24 cm, respectively. Current sources were placed in each of four break points of each rung of the MRI coil and a 22.5-degree phase shift was set between currents in adjacent rungs. The temperature distribution was modeled by using the bio-heat equation: $\rho C_p(\partial T/\partial t) = K \nabla^2 T + \rho \text{SAR} + Q - B(T-T_b)$ with the condition $-K(\partial T/\partial n)_s = H(T_s-T_a) + \text{SWEAT} + \text{RAD}$ at the boundary between tissue and air. Here ρ is the tissue density, T is the temperature, C_p is the specific heat, K is the thermal conductivity, B is blood perfusion coefficient, T_b is the blood temperature, T_a is the ambient temperature, T_s is the temperature of skin, n is the unit vector normal to the surfaces of the skin, H is the convective heat transfer coefficient, Q is the metabolic heat production, SWEAT represents the heat losses of sweat and/or the heat flux due to evaporation, RAD is heat loss from radiation, and SAR is the RF source for heat generation in the tissue.

Local Q and B are functions of local tissue temperature. The metabolic processes are slightly accelerated when the temperature rises so that $Q = Q_0 (1.1)^{(T-T_0)}$ where Q_0 is the basal metabolism and T_0 is the initial temperature. For the internal tissues the temperature-dependence of the blood perfusion coefficient can be expressed as

$$B = \begin{cases} B_0 & T \leq 39^\circ\text{C} \\ B_0[1 + S_B(T - 39)] & 39^\circ\text{C} < T < 44^\circ\text{C} \\ B_0(1 + 5S_B) & T \geq 44^\circ\text{C} \end{cases}$$

where B_0 is the basal blood perfusion and S_B is a coefficient set to $0.8 \text{ } ^\circ\text{C}^{-1}$. The formula to calculate the blood perfusion coefficient for skin is defined as $B = [B_0 + W_1(T_H - T_{H0}) + W_2\Delta T_S] \cdot 2^{\frac{T-T_0}{6}}$ where T_H is hypothalamic temperature at time t and T_{H0} is the initial hypothalamic temperature approximated by the initial temperature of the brain center. ΔT_S is skin temperature rise. W_1 and W_2 are set to $17500 \text{ W}/(\text{m}^3 \text{ } ^\circ\text{C}^2)$ and $1100 \text{ W}/(\text{m}^3 \text{ } ^\circ\text{C}^2)$, respectively.

The mechanism of regulating sweat is described as $\text{SWEAT} = [P + W_3(T_H - T_{H0}) + W_4\Delta T_S] \cdot 2^{\frac{T-T_0}{10}}$ where P is the basal evaporation heat loss from the skin, set to $4 \text{ W}/\text{m}^2$. W_3 and W_4 are weights for hypothalamic and skin temperature rise and set to $140 \text{ W}/(\text{m}^2 \text{ } ^\circ\text{C})$ and $13 \text{ W}/(\text{m}^2 \text{ } ^\circ\text{C})$, respectively. To consider radiation the following equation is adopted: $\text{RAD} = \delta \varepsilon \{ [T + 273.15]^4 - [T_a + 273.15]^4 \}$ where δ is Stefan-Boltzman constant equal to $5.696 \times 10^{-8} \text{ W}/(\text{m}^2 \text{ } \text{K}^4)$, ε is the dimensionless emissivity of skin, approximately equal to 1. The constant 273.15 is for conversion to absolute temperature.

Result and Discussion

All field values were normalized for a head-average SAR of 3 W/kg and the time course of temperature changes were computed for 30-mins duration. The existence of thermal conduction and blood flow in the brain served to modulate the temperature in the upper part of the head. Convection, evaporation and radiation transferred the heat to the environment. With a head-average SAR level of 3 W/kg, the temperature rise distributions were not much different between the results with and without physiological response at these frequencies: because most of the tissues temperature did not exceed 39°C , the blood flow coefficient B did not change. On the other hand, the metabolism rate did increase with the temperature rise, resulting in minor temperature rises with the physiological response. Therefore, the temperature of most tissues is $0.01\text{-}0.02^\circ\text{C}$ higher than that without the thermal regulation. When the head-average SAR was increased to 6, 9, and 12W/kg, the effect of the physiological response becomes more obvious. The increasing perfusion rates of tissue decreased the temperature significantly – by more than 2.5 degrees in some locations at 12 W/kg. These also can be seen from ΔT difference distribution in Fig 1. Also, some tissues' temperature still increased a little due to the increase in metabolism rate.

References:

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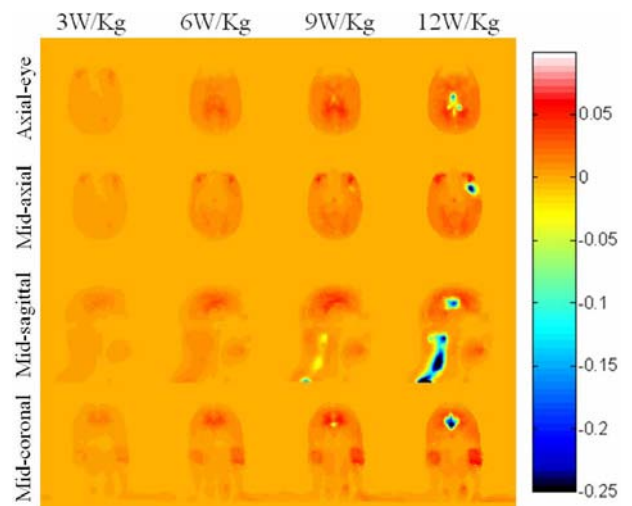


Figure 1. Difference in Temperature increase with and without consideration of physiological response (with minus without). At a head-average SAR of 3.0 W/kg, the difference is negligible.