FDTD analysis of human body-core temperature elevation due to RF far-field energy prescribed in ICNIRP guidelines

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Abstract

This study investigated the relationship between the specific absorption rate and temperature elevation in an anatomically-based model named NORMAN for exposure to radio-frequency far fields in the ICNIRP guidelines. The finite-difference time-domain method is used for analyzing the electromagnetic absorption and temperature elevation in NORMAN. In order to consider the variability of human thermoregulation, sets of parameters for sweating are derived and incorporated in a conventional sweating formula. First, we investigated the effect of blood temperature variation modeling on body-core temperature. Computational results show that the modeling of blood temperature variation was the dominant factor influencing the body-core temperature. This is because the temperature in the inner tissues is elevated via the circulation of blood whose temperature was elevated due to EM absorption. Even at different frequencies, the body-core temperature elevation at the identical whole-body average specific absorption rate (SAR) was almost the same, suggesting the effectiveness of whole-body average SAR as a measure in the ICNIRP guidelines. Next, we discussed the effect of sweating on the temperature elevation and thermal time constant of blood. The variability of temperature elevation caused by the sweating rate was found to be 30%. The blood temperature elevation at the basic restriction in the ICNIRP guidelines of 0.4 W/kg is 0.25 ^{o}C even for a low sweating rate. The thermal time constant of blood temperature elevation was 23 min and 52 min for a man with lower and higher sweating rate, respectively, which is longer than the average time of SAR in the ICNIRP guidelines. Thus, the whole-body average SAR required for blood temperature elevation of 1^{o}C was 4.5 W/kg in the model of human with the lower sweating coefficients for 60-min exposure. From comparison of this value with the basic restriction in the ICNIRP guidelines of 0.4 W/kg, the safety factor was 11.

1. Introduction

There has been increasing public concern about the adverse health effects of human exposure to electromagnetic (EM) waves. Elevated temperature (1-2^{o}C) resulting from the radio frequency (RF) energy absorption is known to be a dominant factor inducing adverse health effects such as heat exhaustion and heat stroke (ACGIH 1996). This fact was reflected in the ICNIRP (International Commission on Non-Ionizing Radiation Protection) guidelines (1998). In the guidelines, the whole-body average specific absorption rate (SAR) is used as a measure of human protection for RF far-field exposure. The basic restriction is defined in terms of whole-body average SAR, and its limit is 0.4 W/kg for occupational exposure. The rationale of this limit is that exposure for less than 30 min. caused a body-core temperature elevation of less than 1 ^{o}C under the conditions in which the whole-body average SAR was less than 4 W/kg (Chatterjee and Gandhi 1983, Hoque and Gandhi 1988, e.g.). Then, a safety factor of 10 has been applied to the above value to provide adequate human protection in the guidelines. However, the temperature and SAR distributions are not identical, which is attributed to heat transfer in the human body.

This problem has encouraged researchers to investigate MW-induced temperature rise in humans (Bernardi *et al* 2003, Foster and Adair 2004) and animals (Hirata *et al* 2006b, e.g.). Bernardi *et al* (2003) calculated SAR and temperature elevation in an anatomically-based human body model with the finite-difference time-domain (FDTD) method. In that study, a human standing on a perfect electrical ground was considered. The power density of EM wave was chosen to be 1 mW/cm^2 at 40 MHz and 900 MHz. The main findings of the same study were as follows: i) sweating prevented temperature from elevating, and ii) the blood temperature rise is marginal though detailed formula was not shown. The temperature elevation at the intensity prescribed in the ICNIRP guidelines (1998) was not investigated clearly. Foster and Adair (2004) used a compartmental model for the human thermoregulatory system (Douglas 1977) to calculate body temperature elevation. Then, the calculated temperature elevation was compared with the measurement for volunteers. Comparison between calculation and measurement shows fair agreement in the temperature elevation.

In the present study, we investigated the relationship between SAR and temperature elevation in an anatomically-based model named NORMAN (Dimbylow 1997) for RF far-field energy at the intensity prescribed in the ICNIRP guidelines. It should be noted that EM absorption of the human body at the ICNIRP reference level shows double-humped frequency characteristics: the first peak appears at about 65 megahertz, at which the body resonates electrically and the second peak appearing around 2 GHz is caused by the relaxation of the reference level with the increase of the frequency (Dimbylow 2002, Wang *et al* 2005, Hirata *et al* 2007). Then, we focused on the frequencies of 65 MHz and 2GHz. Two sweating models (Fiala *et al* 2001, Bernardi *et al* 2003)

are used in the present study. For the model by Fiala *et al* (2001), different sets of parameters for sweating are derived to consider the variability of body-core temperature attributed to the individual development of the sweating glands.

2. Model and Methods

2.1 Human Body Model

The whole-body average SAR and temperature elevation in the anatomically-based human model NORMAN was investigated with the FDTD method. The NORMAN developed at the Health Protection Agency, UK (former National Radiation Protection Agency) was used in the present study (Dimbylow 1997). This model was developed so that the height and weight coincide with those of the standard male and with its organs also rescaled to match the weight given in the standard man defined by ICRP (1994). The NORMAN has a height of 176 cm and a weight of 73 kg, and is comprised of 38 tissues with a resolution of 2 mm.

2. 2. SAR Calculation

The FDTD method (Taflove and Hagness 2003) is used for calculating SAR in NORMAN. A vertically-polarized plane wave is incident on these models standing in free space. The total-field / scattered-field formulation was applied for generating a plane wave. To incorporate the anatomically-based model into the FDTD method, electrical constants of tissues were required. These values were taken from measurement by Gabriel (1996). The computational region has been truncated by applying a twelve-layered, perfectly-matched layer absorbing boundary. For harmonically varying fields, the SAR is defined as

$$\mathrm{SAR} = \frac{\sigma}{2\rho}|\mathbf{E}|^2 = \frac{\sigma}{2\rho}(|\hat{E}_x|^2 + |\hat{E}_y|^2 + |\hat{E}_z|^2) \tag{1}$$

where $\hat{E}_x$, $\hat{E}_y$, and $\hat{E}_z$ are the peak values of the electric field components, σ and ρ, denoting the conductivity and mass density of the tissue, respectively.

2. 3. Temperature Calculation

For calculating the temperature elevation in NORMAN, the bioheat equation (Pennes 1948) was used. A generalized bioheat equation is given by the following equation:

$$C(\mathbf{r})\rho(\mathbf{r})\frac{\partial T(\mathbf{r},t)}{\partial t} = \nabla\cdot(K(\mathbf{r})\nabla T(\mathbf{r},t)) + \rho(\mathbf{r})SAR(\mathbf{r}) + A(\mathbf{r},t) - B(\mathbf{r},t))(T(\mathbf{r},t) - T_B(\mathbf{r},t)) \tag{2}$$

where $T(\mathbf{r},t)$ and $T_B(\mathbf{r},t)$ denote the temperatures of tissue and blood, respectively, C the specific heat of tissue, K the thermal conductivity of tissue, A the basal metabolism per unit

volume, and *B* the term associated with blood perfusion. The thermal constants of tissues were taken from our previous work (Hirata *et al* 2006a). Specific heat and thermal conductivity of tissues were obtained in terms of the equation derived by Cooper and Trezek (1971) with water content (ICRP 1975). The basal metabolism was estimated by assuming that it was proportional to the blood perfusion (Gordon *et al* 1976), just as Bernardi *et al* (2003) did. The boundary condition between air and tissue for Eq. (2) is expressed as:

$$-K(\mathbf{r})\frac{\partial T(\mathbf{r},t)}{\partial n} = H(\mathbf{r})\cdot(T_s(\mathbf{r},t)-T_e(t)) + SW\left(\mathbf{r},T_s(\mathbf{r},t)\right) \tag{3}$$

where H, T_s, and T_e denote, respectively, the heat transfer coefficient, body surface temperature, and air temperature, while SW indicates the heat loss due to sweating. T_e is chosen as 28°C at which thermal equilibrium is obtained in a naked man (Hardy and Du Bois 1938). In other words, the effect of garment insulation on the temperature elevation (Nelson *et al* 2005) is beyond the scope of the present study. The heat transfer coefficient between tissue and internal air and that between skin and external air were chosen as $50\ \mathrm{W/m^2/^oC}$ and $5.8\ \mathrm{W/m^2/^oC}$, respectively. The latter value was adjusted so that the heat radiation from the skin surface in a discretized model (NORMAN) coincides with the basal metabolism of the body: namely, the surface area of discretized models for computation is larger than that of an actual human (Samaras *et al* 2006), and thus this effect was compensated for in the present study.

For taking into account body-core temperature elevation in the bioheat equation, it is reasonable to consider the blood temperature as a variable of time $T_B(\mathbf{r},t) = T_B(t)$. Namely, the blood temperature is assumed to be constant over the whole body, since the blood circulates throughout the human body in one minute or less (Follow and Neil 1971). Thus, the following formula is not applicable to transient exposure for a brief period of time less than a few minutes. The blood temperature variation is given by the following equation (Bernardi *et al* 2003):

$$T_B(t) = T_{B0} + \int_t \frac{Q_{BTN}(t)}{C_B\rho_B V_B}\mathrm{d}t \tag{4}$$

where Q_{BTN} is the net rate of heat acquisition of blood from body tissues. C_B (=4000 J/kg·°C), ρ_B (=1050 kg/m^3), and V_B (=5000 ml) denote the specific heat, mass density, and total volume of blood, respectively (ICRP 1975). A discrete vasculature thermal model, which allows for the effect of individual vessels, was proposed by Kotte *et al* (1996), in which the spatial distribution of blood temperature can be considered quite naturally. However, a complex process is required to develop vascular distribution. Our interest is not in the spatial distribution of blood temperature itself but in the elevation of body-core temperature. This is the main reason why the spatial distribution of blood temperature is not considered here. Note that the above-defined blood temperature can be approximately considered as body-core temperature, since the blood perfusion rate in the body core is high and close to the blood temperature. In the present study,

the following two equations are used to compute the rate of heat acquisition of blood from body tissues:

$$Q_{BT}(t) = \int_V B(t)(T_B(t) - T(\mathbf{r},t))dV \quad (5)$$

$$Q_{BTN}(t) = Q_{BT}(t) - Q_{BT}(0). \quad (6)$$

It should be noted that Eq. (6) was required to compensate for unphysical time evolution of blood temperature, and enables us to evaluate the net rate of heat acquisition of blood from body tissues. This is because Eq. (5) is not zero even at time t=0, which is attributed to some computational simplification or assumption, including discretized anatomically-based model with finite resolution, uniform distribution of blood temperature over the body, and uncertainty in the basal metabolism of tissues depending on the individual and body parts. The effectiveness of this formula was demonstrated in comparison with measurement for a rabbit (Hirata *et al* 2006b).

2. 4. Thermoregulatory Response

2. 4. 1. Influence of temperature elevation on blood perfusion rate. For a temperature elevation above a certain level, the blood perfusion rate was increased in order to carry away excess heat produced. The variation of blood perfusion rate in the skin through vasodilatation is expressed in terms of the temperature elevation in the hypothalamus, $T_H - T_{H0}$, and the average temperature increase in the skin, ΔT_S:

$$B(\mathbf{r},t) = (B_0(\mathbf{r}) + F_{HB}(T_H(t) - T_{H0}) + F_{SB}\Delta T_S(t)) \cdot 2^{(T(\mathbf{r},t)-T_0(\mathbf{r}))/6} \quad (6)$$

where

$$\Delta T_S(t) = \frac{\int_S (T(\mathbf{r},t) - T_0(\mathbf{r}))dS}{S}. \quad (7)$$

T_0, F_{HB}, and F_{SB} are the steady-state temperature without heat load and the weighting coefficients of signal from the hypothalamus and skin, respectively. T_H and T_{H0} are the temperature at the hypothalamus and its initial temperature, respectively. The coefficients of F_{HB} and F_{SB} in Eq. (6) were 17,500 $\mathrm{W/m^3/^oC^2}$ and 110 $\mathrm{W/m^3/^oC^2}$ (Douglas 1977, Bernardi *et al* 2003).

In blood perfusion in all tissues except the skin, the regulation response was assumed to be governed by local tissue temperature. When that temperature remained below a certain level, blood perfusion was equal to its basal value B_0. Once the local temperature exceeded a threshold, the blood perfusion increased almost linearly with the temperature in order to carry away the heat evolved. For human legs, these responses are expressed by the following equations (Hoque and Gandhi, 1988):

$$B(\mathbf{r},t) = B_0(\mathbf{r}), \qquad T(\mathbf{r}) \le T_A \qquad (8)$$

$$B(\mathbf{r},t) = B_0(\mathbf{r})\left(1+(\gamma-1)\frac{T(\mathbf{r},t)-T_A}{T_B-T_A}\right), \qquad T_A < T(\mathbf{r}) \le T_B \qquad (9)$$

$$B(\mathbf{r},t) = \alpha B_0(\mathbf{r}), \qquad T(\mathbf{r}) > T_B \qquad (10)$$

where T_A and T_B denote the threshold temperatures at which the blood perfusion activates and saturates, respectively. The coefficient γ must be larger than 1. In the present study, this formula was applied not only to legs but also to the remaining body parts. The threshold temperatures for activating blood perfusion in the inner tissues T_A was chosen as 39 ^{o}C (Hoque and Gandhi 1988). Note that this formula does not work in the following calculations, since our attention is paid to the temperature elevation at most 1 ^{o}C or so (ICNIRP 1998), which is well below the level activating the above equations.

2. 4. 2. Influence of temperature elevation on sweating. Two sweating modelings were considered. One is the formula proposed by Spiegel (1984) and improved by Bernardi *et al* (2003), which is given by the following equation:

$$SW(\mathbf{r},t) = [SW_o + F_{HS}(T_H(t) - T_{HO}) + F_{SS}\Delta T_S] \times 2^{(T(\mathbf{r})-T_0(\mathbf{r}))/10} \qquad (11)$$

where SW_o is insensible perspiration, or the basal evaporative heat loss from the skin. The coefficients of F_{HS} and F_{SS} in Eq. (11) were 140 W/m^2/^{o}C^2 and 13 W/m^2/^{o}C^2 (Douglas 1977, Bernardi *et al* 2003).

The other formula is presented by Fiala *et al* (2001). The sweating coefficients are assumed to depend on the temperature elevation in the skin and/or hypothalamus. An appropriate choice of the coefficients could enable us to discuss the uncertainty in the temperature elevation attributed to individual differences in sweat gland development.

$$SW(\mathbf{r},t) = \{W_S(\mathbf{r},t)\Delta T_S(t) + W_H(\mathbf{r},t)(T_H(t) - T_{Ho})\} / S \times 2^{(T(\mathbf{r})-T_0(\mathbf{r}))/10} \qquad (12)$$

$$W_s(\mathbf{r},t) = \alpha_{11}\tanh(\beta_{11}T_s(\mathbf{r},t) - T_{so}(\mathbf{r})) - \beta_{10}) + \alpha_{10} \qquad (13)$$

$$W_H(\mathbf{r},t) = \alpha_{21}\tanh(\beta_{21}T_s(\mathbf{r},t) - T_{so}(\mathbf{r})) - \beta_{20}) + \alpha_{20} \qquad (14)$$

where S is the surface area of the human body, and W_S and W_H are the weighting coefficients for the sweating rate associated with the temperature elevation in the skin and hypothalamus, respectively. Fiala *et al* (2001) determined the coefficients of α and β for average sweating rate based on the basis of measurement by Stolowijk (1971). In addition to the set of coefficients in Fiala *et al* (2001), we determined the coefficients for a man with higher and lower sweating rates parametrically, as listed in table 1. Figures 1 (a) and (b) show the dependence of coefficients W_s and W_H on the temperature elevation in the skin and hypothalamus, respectively, for different sweating rates.

2.5. Exposure Scenario

The anatomically-based human model named NORMAN is located in free space. A front-incident plane wave with vertical polarization was considered as a wave source. As presented in Wang *et al* (2005), whole-body average SAR has two peaks for plane wave exposure at the ICNIRP reference level: namely, it becomes maxima at 65 MHz and 2 GHz. The first peak is caused by the whole-body resonance in the human body. The latter peak, on the other hand, is caused by the relaxation of the ICNIRP reference level with the increase in the frequency. Note that the power density at the ICNIRP reference level is 1 mW/cm^2 at 65 MHz and 5 mW/cm^2 at 2 GHz. The whole-body average SAR in NORMAN was 0.30 W/kg at 65 MHz and 0.32 W/kg at 2 GHz (Hirata *et al* 2007), which is close to the basic restriction for occupational environment prescribed in the ICNIRP guidelines. It is noteworthy that the SAR distributions at these frequencies are quite different (Hirata *et al* 2007). EM absorption occurs over the whole body at 65 MHz and around the body surface at 2 GHz. In the following discussion, we concentrated on the SAR and temperature elevation at these frequencies.

3. Computational Results and Discussion

3. 1. Analysis of Temperature Elevation with Conventional and Proposed Modelings

In the present study, we defined 'conventional' and 'proposed' modelings to discuss the effect of thermal transfer modeling on the temperature elevation. In the conventional modeling, the blood temperature variation and the thermoregulatory responses are not taken into account. In the proposed modeling, the blood temperature variation of Eq. (4) is used, and then the thermoregulatory responses were taken into account. Note that, in this subsection, Eq. (12) with a set of the parameters for the standard sweating rate was used for sweat modeling (see figure 1).

Figure 2 shows the layered-average SAR and temperature elevation in NORMAN at frequencies of 65 MHz and 2 GHz. The duration of exposure was 60 min. From the comparison of figures 2 (a) and (b), the layered-average SAR and temperature elevations resembled each other. Specifically, the layered-average SAR and temperature elevation peak at the ankle, knee, and neck at 60 MHz, while they both are rather uniform along the body height direction at 2 GHz. No clear difference is observed in the profile of temperature elevation calculated with the conventional and proposed modelings. It is worth pointing out that some differences in the temperature elevation are observed at heights of around 80, 120 and 165 cm. At these heights, the temperature elevation with the proposed modeling is higher than that with the conventional modeling. These heights correspond to the rectum, internal organs, and hypothalamus, whose blood perfusion rate is larger than that in the other organs. This implies that the difference in

temperature elevation would be caused by the blood temperature variation governed by Eq. (4).

Next, we investigated the time evolution of temperature elevation at the hypothalamus with the conventional and proposed modeling. As seen from figure 3, at both frequencies, the temperature elevations with the conventional modeling were much smaller than those with the proposed modelings. In the conventional modeling, the temperature elevation at the hypothalamus is caused by the SAR or EM absorption only. In the proposed modeling, the additional temperature elevation would be caused by the blood temperature elevation. Note that the curve for 'proposed' in figure 3 does not change obviously, even though the temperature-dependent blood perfusion rate (Eq. (6)) was neglected, implying that the blood temperature variation is the essential factor in this difference. In order to confirm this inference, we showed in the same figure the time evolution of temperature elevation of the blood. From this figure, the thermal time constants of temperature elevation at the hypothalamus were 1134 s at 65 MHz and 1114 s at 2 GHz, while those of the blood were 962 s and 880 s, respectively. This thermal constant would be somewhat larger in an actual human. This is because it takes time for blood warmed by EM absorption around the surface to reach the body core, which can not be taken into account in our formula. The difference between these time constants suggests that, first, the blood temperature elevates due to the EM absorption, inducing the temperature elevation in deep tissues. Table 2 summarizes the whole-body average SAR and temperature elevation at the hypothalamus at 65 MHz and 2 GHz. As is evident from this table, the ratio of temperature elevation at the hypothalamus to whole-body average SAR is almost the same despite the different frequency and different SAR distribution. This result suggests that the whole-body average SAR could be a decisive factor in determining the temperature elevation, implying the effectiveness of the measure used in the ICNIRP guidelines. In addition, the temperature elevation for exposure to RF fields at the ICNIRP reference level is much smaller than 1^{o}C, which is a criterion considered in the ICNIRP guidelines. This discussion will be made in detail in the next section.

3. 2. Influence of Sweating Rate on Blood Temperature Elevation

It is well known that the development of sweat glands depends on the individual. With the proposed modeling, we investigated the influence of the sweating rate on the blood temperature elevation. In this subsection, we scaled the SAR so that the whole-body average SAR coincides with 0.4 W/kg, which is the basic restriction for occupational exposure in the ICNIRP guidelines. Note that any duration is allowed if the whole-body average SAR is less than 0.4 W/kg for any 6-min average.

First, as the worst case condition, we consider an infinite duration with the whole-body average SAR close to but smaller than 0.4 W/kg. Figure 4 shows the temperature elevation with

different coefficients of sweating rate. As seen from this figure, different temperature elevation and thermal time constant were observed for different sweating rates. A lower sweating rate results in a higher temperature elevation, because the heat transfer from the skin to air is not enhanced so much even for the activation of thermoregulatory responses. For a smaller sweating rate, the blood temperature elevation reaches the steady state at 200 min., while the thermal time constant was 52 min. The thermal time constant was 22 min and 34 min. for the man with higher and standard sweating rate, respectively. The steady-state temperature elevation was 0.25^{o}C for smaller sweating rate, which is still sufficiently smaller than 1^{o}C. The blood temperature elevation is reduced to 0.18^{o}C and 0.10^{o}C, respectively, for the standard and higher sweating rates, suggesting that the uncertainty of $\pm 30\%$ in the blood temperature elevation is caused by the sweating rate.

Figure 5 shows the dependency of blood temperature elevation on the whole-body average SAR. The duration is chosen as 60 min so as to be longer than that considered in the ICNIPR guidelines (30 min.) and the thermal time constant for the man with the smaller sweating rate (52 min). In this figure, the result with Eq. (11) is also presented for comparison. As seen from this figure, the relationship between blood temperature elevation and whole-body average SAR is almost linear for the modeling with Eq. (11), while nonlinear for Eq. (12). The main reason for this difference is caused by the nonlinear dependency of sweating rate on temperature elevation for Eq. (12) (see figure 1). For the duration of 60 min., the whole-body average SAR required for a blood temperature elevation of 1^{o}C was 6.3 W/kg for the man with the standard sweating rate, 8 W/kg for the higher rate and 4.5 W/kg for the lower rate. When Eq. (5) is used, the value was 5.9 W/kg, which is comparable to that of the standard coefficients with Eq. (6).

4. Conclusion

In the present study, we investigated the relationship between SAR and temperature elevation in an anatomically-based model named NORMAN for RF far-field exposure. The motivation of the present study was that the dominant factor for RF and microwave exposures on human is the temperature elevation (1-2^{o}C), while the whole-body average SAR is used as a measure of human protection in the ICNIRP guidelines (1998). In the previous works on animal experiments, the relationship between SAR and body-core temperature elevation was not clarified. The temperature elevation due to RF exposure at the ICNIRP basic restriction was also unclear. To consider the variability of temperature elevation due to the development of sweating glands, we derived different sets of parameters for sweating applicable to conventional equations for sweat modeling.

First, we investigated the effect of blood temperature variation modeling on the temperature elevation in body-core temperature. The blood temperature modeling was found to be the

dominant factor influencing the body-core temperature. Especially, blood temperature elevation due to EM absorption induced the temperature elevation in the inner tissues via its circulation. The point to be stressed here is that the effect of frequency on the temperature elevation was marginal, even though the SAR distribution is quite different. This finding suggests the usefulness of whole-body average SAR for predicting body-core temperature elevation, and also supports the measure being used in the ICNIRP guidelines.

Next, we discussed the effect of the sweating rate on the temperature elevation and thermal time constant of blood temperature. Computational results suggested that variability of blood temperature elevation caused by different sweating rates is up to 30%. For the EM exposure of 60 min., the whole-body average SAR required for blood temperature elevation of 1^{o}C was 4.5 W/kg for a man with a low sweating rate, corresponding to the safety factor of 11 as compared with the basic restriction in the ICNIRP guidelines.

In future work, we will investigate the temperature elevation in child models exposed to far field, taking the thermo-physiology into account. In addition, we will discuss the applicability of the formula to the temperature elevation for smaller exposure duration, especially considering occupational exposure.

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FIGURE AND TABLE CAPTION

Figure1. Dependence of coefficients for sweating rate on the temperature elevation in the skin (a) and hypothalamus (b). These coefficients correspond to Eqs. (13) and (14).

Figure 2. Layer-average SAR (a) and temperature elevation (b) for exposure to plane wave with the ICNIRP reference level for occupational environment.

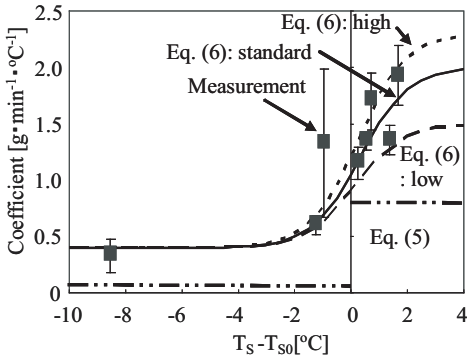
Figure 3. Temperature elevation of (a) blood and (b) hypothalamus for exposure to plane wave with the ICNIRP reference level for occupational environment. The duration of exposure was 60 min.

Figure 4. Temperature elevation of blood for different sweating rates. The whole-body average SAR was scaled to 0.4 W/kg at 65 MHz and 2 GHz.

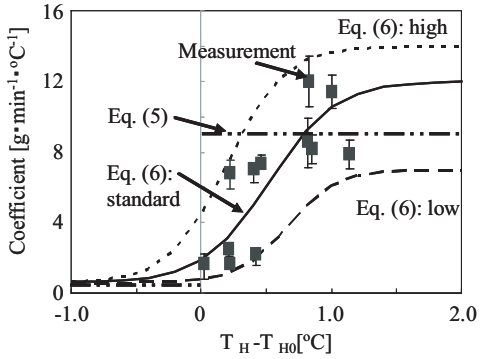
Figure 5. Temperature elevation versus whole-body average SAR. The duration of exposure was 60 min.

Table 1. Coefficients associated with sweating rates for the temperature elevation in the skin and hypothalamus.

Table 2. Temperature elevation at the hypothalamus and whole-body average SAR at 65 MHz and 2 GHz.

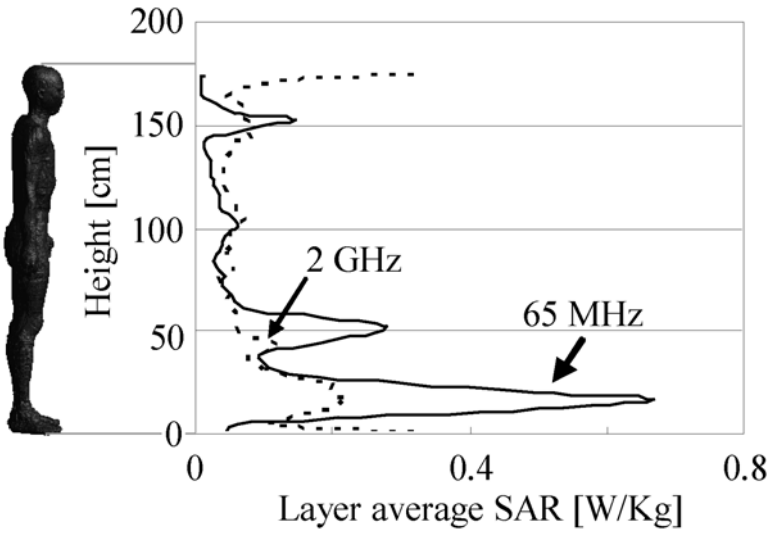

Eq. (6): high
Eq. (6): standard
Measurement
Eq. (6)
: low
Eq. (5)
Coefficient [g•min-1•°C-1]
2.5
2.0
1.5
1.0
0.5
0
-10
-8
-6
-4
-2
0
2
4
TS -TS0[°C]

(a)

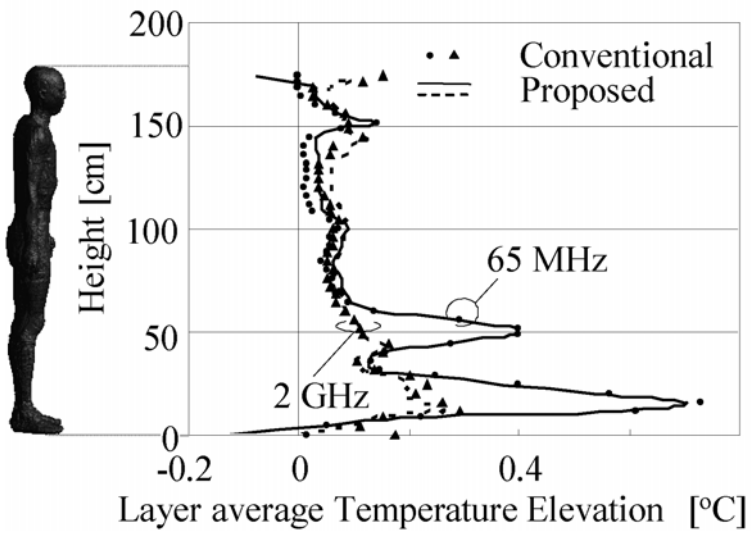

Eq. (6): high
Measurement
Eq. (5)
Eq. (6):
standard
Eq. (6): low
Coefficient [g•min-1•°C-1]
16
12
8
4
0
-1.0
0
1.0
2.0
T H -T H0[°C]

(b)

Fig. 1


200
150
100
50
0
Height [cm]
2 GHz
65 MHz
0
0.4
0.8
Layer average SAR [W/Kg]

(a)


200
150
100
50
0
Height [cm]
Conventional
Proposed
65 MHz
2 GHz
-0.2
0
0.4
Layer average Temperature Elevation [°C]

(b)

Fig. 2

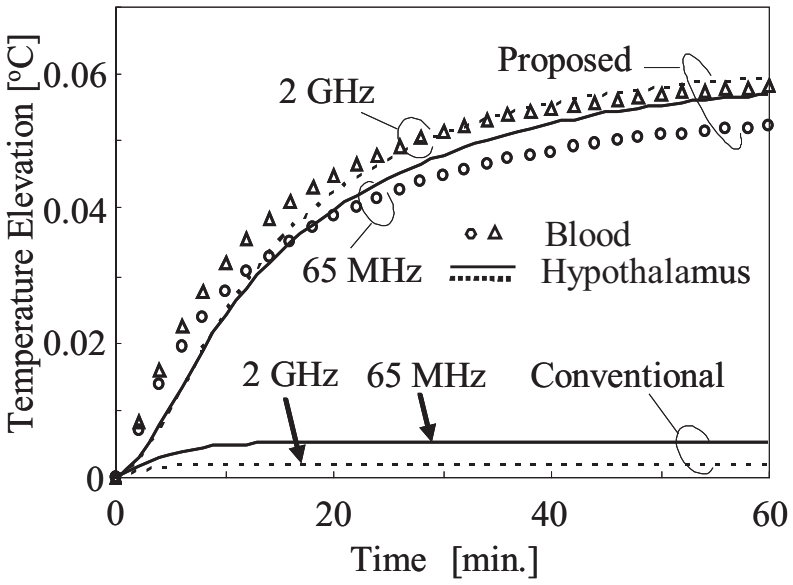

Temperature Elevation [°C]
0.06
0.04
0.02
0
Proposed
2 GHz
65 MHz
Blood
Hypothalamus
2 GHz
65 MHz
Conventional
0
20
40
60
Time [min.]

Fig. 3

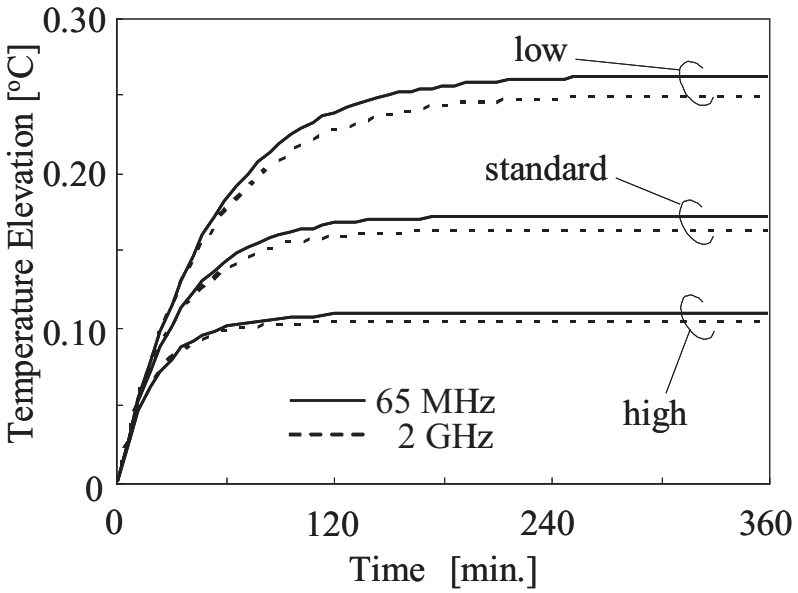

0.30
0.20
0.10
0
Temperature Elevation [°C]
low
standard
high
65 MHz
2 GHz
0
120
240
360
Time [min.]

Fig. 4

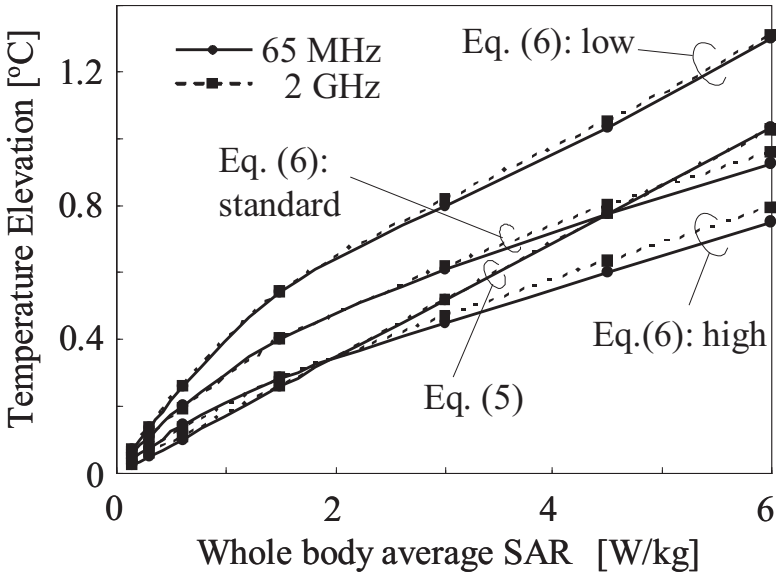

65 MHz
2 GHz
Eq. (6): low
Eq. (6):
standard
Eq.(6): high
Eq. (5)
Temperature Elevation [°C]
1.2
0.8
0.4
0
0
2
4
6
Whole body average SAR [W/kg]

Fig. 5

	Coefficients of skin signal				Coefficients of hypothalamus signal			
	α_{10}	α_{11}	β_{10}	β_{11}	α_{20}	α_{21}	β_{10}	β_{11}
high	1.35	0.95	0.15	0.59	7.30	6.70	0.47	2.30
standard	1.20	0.80	0.19	0.59	6.30	5.70	1.03	1.98
low	0.95	0.55	0.09	0.59	3.80	3.20	1.80	2.70

Table 1.

Frequency	65 MHz	2 GHz
SAR [W/Kg]	0.300	0.315
ΔT in Blood [^{o}C]	0.0570	0.0594
ΔT/SAR	0.190	0.189

Table 2