Computation of induced electric field and temperature elevation in human due to lightning current

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The present study investigated induced electric field and temperature elevation in specific tissues/organs of an anatomically based human body model for the lightning current. The threshold amplitude of the current inducing ventricular fibrillation and skin burning are estimated from computed induced electric field and temperature elevation with formulas for electrical stimulation and thermal damage. The computational results obtained herein were reasonably consistent with clinical observation. © *2010 American Institute of Physics*. [doi:10.1063/1.3427359]

Lightning fatalities and injuries in Japan have been reported to be 15 and 59, respectively, for the period from 2004 and 2008.[1] In the same period, fatalities in United States were 189.[2] Direct effect of lightning current on the human is summarized as ventricular fibrillation, respiratory arrest, and the burning around the body surface.[3,4] In addition, cataracts may develop later in some patients.[4] Postmortem examination cannot always indicate the exact mechanism of death.[3] The lightning effects have been discussed from the standpoint of different expertise; clinical aspect,[5] electrical circuit,[4,6] and animal experiment.[3,4] The induced field and temperature in specific organs/tissues could not be estimated from a physical modeling with electrical circuit theory.[4,6] Thus, the direct cause of fatalities could not be discussed from the standpoint of physics.

The following processes are considered as physical phenomena of lightning strike on humans.[4] In the first phase, the internal current may be the most causative for the development of cardiac arrest and respiratory arrest. In the second phase, as the body potential builds up in response to the current, it then produces an electric field over the surface of the body. At a certain level, current can emerge of to escape the body at various points of the body surface. In the third phase, as the current continues to increase, the surface flashover bridges the strike point and the ground.

Our attention here focuses on the physical modeling of the first phase.[6] The induced electric field/current density and resultant temperature elevation in an anatomically based human body model has been quantified for lightning current in order to obtain insight for the threshold current amplitude inducing harmful effect on humans.

For evaluating the possibility of ventricular fibrillation, electrical stimulation to tissue should be investigated. A threshold for excitation of nerve and muscle cells can be empirically derived from a strength-duration curve, as follows:[7]

$$E_T = E_0\left[1 - \exp\left(-\frac{\tau}{\tau_e}\right)\right]^{-1}, \tag{1}$$

where E_T denotes the threshold *in situ* electric field, E_0 the minimum threshold, τ_e the strength-duration time constant, and τ the pulse duration. Values of E_0 and τ_e were estimated as 0.075 V/m and 25 ms for synapse activity alteration, 6.15 V/m and 0.149 ms for 20 μm nerve (peripheral nerve) excitation, 12 V/m and 3 ms for cardiac excitation.[7] The minimum threshold for ventricular fibrillation typically exceeds that for excitation by a factor of 50 or more.[8] The threshold electric field of respiratory arrest is unsure and a passing phenomenon for diaphragm muscle, and thus not discussed here.

The temperature elevation should be investigated to discuss the skin burning due to lightning current. The duration of the lightning current is the order of several dozen microseconds or larger,[9,10] which is much smaller than the thermal time constant of biological tissue of several minutes. The temperature elevation can be approximately obtained by the following equation:

$$\Delta T \approx \frac{\sigma}{\rho C}\int_0^t |\boldsymbol{E}(t)|^2 dt, \tag{2}$$

where $\boldsymbol{E}$, σ, ρ, and C denote the *in situ* electric field, electrical conductivity, mass density, and specific heat of the tissue. The heat evolved is diffused via blood perfusion and heat conduction.[11,12] The temperature distribution becomes smooth with time, which is governed by the bioheat equation.[13] The thermal damage of skin can be evaluated with the following equation:[14]

$$\Omega = 3.1 \times 10^{98}\int_0^t \exp[-75000/(T_t + 273)]dt, \tag{3}$$

$$T_t = T_s - (T_s - 35)\left[\frac{2}{\sqrt{\pi}}\int_0^{0.15/\sqrt{t}} \exp(-y^2)dy\right], \tag{4}$$

where Ω denotes the degree of injury to be expected, T_s the surface temperature of the skin during heat exposure, and T_t the temperature of the basal epidermal layer. An $\Omega \geq 1.0$ results in a time-temperature relationship which produces complete epidermal necrosis.

An anatomically based Japanese adult male model (Ref. 15) was used in our computation. That model was developed based on the magnetic resonance images of a 22 year male volunteer having a height of 1.73 m and a weight of 65 kg.

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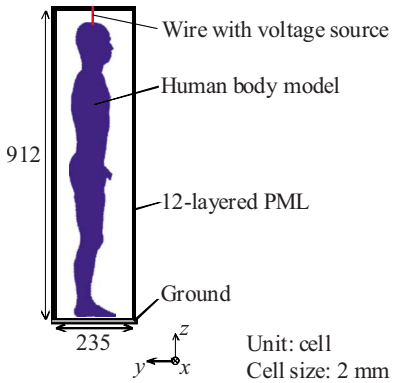

FIG. 1. (Color online) FDTD model for analyzing induced quantities caused by injected current into the head.

This model is comprised of voxels with the resolution of 2 mm and segmented to define 51 kinds of discrete organs/tissues.

In order to evaluate the *in situ* electric field and temperature elevation, the finite-difference time-domain (FDTD) method[16] was used to conduct electromagnetic and thermal dosimetry in an anatomically based human body model. The duration of lightning currents is more than dozen microseconds.[9,10] It is unpractical to conduct electromagnetic dosimetry for realistic lightning current waveforms, since the FDTD simulation with high spatial-resolution needs small time increments with the order of picoseconds. The essential factors to be evaluated were *in situ* electric field and its time integral [see Eqs. (1) and (2)]. In addition, the displacement current in the biological tissue is much smaller than the conduction current for the frequency spectrum of the lightning current. Thus, a quasistatic FDTD method was employed in order to overcome this difficulty.[17,18] The temperature elevation is computed with the bioheat equation[13] by substituting computed electromagnetic power absorbed in the model. The electrical conductivity of different tissues was taken from Ref. 19 at a frequency of 50 Hz. The thermal constants of different tissues were taken from those listed in Refs. 12 and 20.

Figure 1 illustrates an FDTD domain to analyze induced quantities caused by injected current into the head. The side length of the FDTD cell was chosen as 2 mm, which coincides with the human model resolution. The human model was placed on the bottom ground. A semi-infinite wire was stretched from the computational boundary to the top of head with a one-cell gap. For truncation of the computational region, 12-layered perfectly matched layer was used. A lightning current was simulated by an equivalent voltage source with the one-cell gap at the wire.[21] Our computational code has been validated for electromagnetic stimulation for short electromagnetic pulse[22] and thermal dosimetry.[12] For simulating a lightning strike, we considered a condition of a direct strike or side flash[3,4] to the head of a human standing on a ground with both feet.

TABLE I. Maximum and median values of *in situ* electric field strength ($|\boldsymbol{E}|$) per 1 A current in selected organs/tissues for different sites of current injection.

	$\|\boldsymbol{E}\|$ (V/m)			
	Head		Left shoulder	
Organ/tissue	Max	Median	Max	Median
Grey matter	1924	254	9.3	0.9
White matter	1910	335	5.2	1.3
Cerebellum	1150	273	10	1.6
Spinal cord	2073	234	650	30
Cerebrospinal fluid	1833	140	312	0.6
Eye	157	34	1.3	0.3
Heart	433	110	447	122

Figure 2 illustrates the *in situ* electric field strength for current injection from the top of the head for the injection of 1 A. The maximum current appeared at the top of the head or the entry site of lightning strike. Then, the current flows from the head to the feet. The *in situ* electric field takes larger values where the cross-sectional area is small, such as neck and knee. When the current was injected from the shoulder instead of the head, the induced electric field in the body except in the head and shoulder was comparable (see Table I).

Figure 3 illustrates the resultant temperature elevation distribution in the human body. The temperature elevation at the sites where the current flows in and out becomes larger.

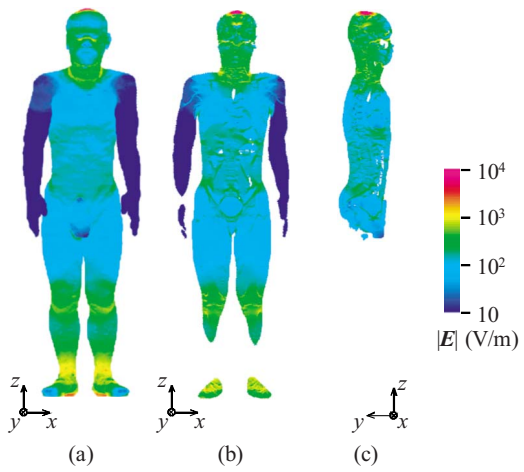

FIG. 2. (Color online) Distribution of *in situ* electric field strength ($|\boldsymbol{E}|$) per 1 A current. (a) Front view, (b) coronal, and (c) sagittal sections of the human model.

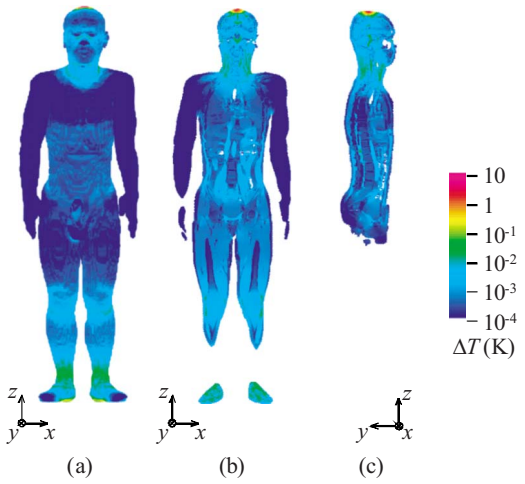

FIG. 3. (Color online) Distribution of temperature elevation (ΔT) per 1 A^2 s current. (a) Front view, (b) coronal, and (c) sagittal sections of the human model.

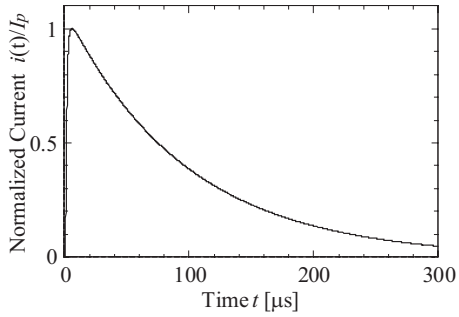

FIG. 4. Normalized current waveform given by Eq. (5) with parameters of $\eta=0.92$, $n=3.0$, $\tau_1=1.65$ μs, and $\tau_2=96.6$ μs.

In addition, notable temperature elevation is also observed around the neck and ankle where the cross-sectional area is relatively small. This distribution becomes smoother with time due to thermal diffusion.

Let us discuss our computational results for a realistic lightning current waveform. Parameters which characterize lightning current have large variability.[9,10] The waveform of the current is then simplified to be given with an empirical equation by considering the negative first stroke, as follows:[23]

$$i(t) = \frac{I_p}{\eta}\frac{k_s^n}{1+k_s^n}\exp\left(-\frac{t}{\tau_2}\right), \tag{5}$$

where I_p denotes the peak current, η the correction factor of the peak current, $k_s=t/\tau_1$, and τ_1 and τ_2 time constants determining current rise- and decay-time, respectively. A set of parameters is estimated as $I_p=30$ kA, $\eta=0.92$, $n=3.0$, $\tau_1=1.65$ μs, and $\tau_2=96.6$ μs.[18] Then, a current waveform is normalized as in Fig. 4. The waveform was truncated at 300 μs, which is three times larger than τ_2. For this waveform, the time integral of current, corresponding to electric charge, and the action integral were 9.8×10^{-5} A s and 5.4×10^{-5} A^2 s, respectively.

The strength-duration curve is considered as a constant charge for the current duration smaller than that of the time constant of the strength-duration curve.[7] The threshold amplitude inducing the ventricular fibrillation was estimated as $E_T\times\tau_e=1.8$ V s/m. For the maximum electric fields induced in the heart of 433 V/m as listed in Table I, the threshold current amplitude was 42.4 A. For the corresponding action integral of 9.7×10^{-2} A^2 s, the maximum temperature elevation in the skin averaged over 1 cm^2 around the top of the head was 40 K. Note that this averaging area corresponds to the diameter of a central core of lighting flash.[3] For the time course of temperature solved by the bioheat equation, Ω in Eq. (3) was 1.5. The temperature elevation in the remaining part of the skin and the eye were much smaller than the threshold of thermal damage. It is noteworthy that sufficient data is not available for the respiratory arrest. However, the threshold of not only respiratory nerves but also other nerve systems would be violated before the ventricular fibrillation for the lightning strike to the head. This is because the threshold for nerve stimulation in the brain is much lower than that of the heart,[8] in addition to larger induced electric field in the brain than in the heart (Table I).

In conclusion, the electrical stimulation in the brain is the dominant effect when the lightning current is injected from the top of the head. On the contrary, the ventricular fibrillation is the most possible effect for lightning strikes on the shoulder. For these two scenarios, the temperature elevation was not significant except for the entry sites of the current. These computational results are consistent with some clinical observation reported in earlier works;[3–5] cardiac arrest would be the dominant factor of lightning-induced death, and the skin burn is minor except entry and exist sites.

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