Effects of thermal properties and geometrical dimensions on skin burn injuries

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Abstract

A one-dimensional multi-layer model is presented to characterise the skin burn process resulting from the application of a high temperature heat source to a skin surface. Transient temperatures were numerically calculated using a finite difference method to solve the Pennes bioheat equation. A damage function denoting the extent of burn injury was then calculated using the Arrhenius assumptions. The model was used to predict the effects of thermal physical properties and geometrical dimensions on the transient temperature and damage function distributions. The results show that the epidermis and dermis thicknesses significantly affect the temperature and burn injury distributions, while variations of the initial temperatures and the blood perfusion have little effect.
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1. Introduction

Accurate analysis of the skin burn process facilitates the development of animal models. Burns result from a temperature elevation in tissue over a threshold value for a period of time. Both the temperature and the exposure duration greatly affect the extent of the burn. Henriques and Moritz [1] first discussed the relationship between the temperature and the time required to produce a specific degree of thermal injury. Their results showed that a burn injury of standard threshold severity can be produced by progressively decreasing temperatures as the thermal insult period is logarithmically increased.

Many investigators have discussed burn injuries for different situations. Diller and Hayes [2] presented a finite element model of burn injuries in blood-perfused skin. Torvi and Dale [3] developed a finite element model of skin for a flash fire exposure and discussed the sensitivity of burn predictions to variations in thermal physical properties. Ng and Chua [4] presented a mesh-independent model to predict skin burn injuries using the finite element method. Ng and Chua [5] compared one- and two-dimensional programmes for predicting skin burns and showed that the temperature distributions predicted by the one- and two-dimensional programmes were similar. Recently, Diller [6,7] proposed a one-dimensional model to calculate the transient temperature and injury distributions in skin using the finite difference method.

The model developed here presents the transient temperature and damage function distributions variations for the variations of the initial temperature, blood perfusions and skin layer thicknesses. The results show that the epidermis and dermis thicknesses significantly affect the temperature and burn injury distributions.

2. Mathematical model

A one-dimensional multi-layer tissue model is shown in Fig. 1. The skin is divided into three layers, the epidermis, the dermis, and the subcutaneous region. The region from the inner surface of the subcutaneous region to the core is called the inner region in this paper. The typical thermal physical properties [3] used in this paper are listed in Table 1. For blood, $\rho_b = 1060 \, \mathrm{kg \, m^{-3}}$ and $C_b = 3770 \, \mathrm{J \, kg^{-1} \, K^{-1}}$.

The analysis assumes that no interfacial resistance exists between the heating source and the skin surface. Therefore, the surface temperature remains constant during the exposure. The tissues are assumed to be homogeneous within each layer. Blood perfusion, thermal conductivity, and heat capacity are assumed to be constant in each layer despite the temperature elevation. Metabolic heat generation is assumed to be zero relative to the other heat fluxes involved.

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Nomenclature

C	specific heat ($\mathrm{J\,kg^{-1}\,K^{-1}}$)
H	distance from skin surface to body core
k	thermal conductivity ($\mathrm{W\,m^{-1}\,K^{-1}}$)
P	pre-exponential factor ($\mathrm{s^{-1}}$)
R	universal gas constant ($8.314\,\mathrm{J\,kmol^{-1}\,K^{-1}}$)
t	time (s)
T	temperature (°C or K)
x	space co-ordinate (m)
ΔE	activation energy ($\mathrm{J\,kmol^{-1}}$)

Greek letters

ρ	density ($\mathrm{kg\,m^{-3}}$)
ω_b	blood perfusion ($\mathrm{m^3\,s^{-1}\,m^{-3}}$ tissue)
Ω	damage function

Subscripts and superscripts

a	ambient
b	blood
c	core
e	east
f	final
i	initial; node number
k	time step
w	west; wall

The most common model for simulating the thermal behaviour of tissue is the Pennes' bioheat transfer equation [8]. The one-dimensional form of Pennes' bioheat equation without metabolic heat generation can be written as:

$$\rho C \frac{\partial T}{\partial t} = \frac{\partial}{\partial x}\left(k\frac{\partial T}{\partial x}\right) + \omega_\mathrm{b}\rho_\mathrm{b}C_\mathrm{b}(T_\mathrm{b} - T) \tag{1}$$

The burn process is modeled using the following initial and boundary conditions:

$$T(x, 0) = T_i(x) \quad \text{for } t = 0 \tag{2}$$

$$T(0, t) = T_\mathrm{w} \quad \text{for } x = 0 \tag{3}$$

$$T(H, t) = T_\mathrm{c} \quad \text{for } x = H \tag{4}$$

where the initial temperature distributions, $T_i(x)$, could be obtained by solving Eq. (1) with the following boundary conditions:

$$-k\frac{\partial T_i}{\partial x} = -h_\mathrm{a}(T_i - T_\mathrm{a}) \quad \text{for } x = 0 \tag{5}$$

$$T_i = T_\mathrm{c} \quad x = H \tag{6}$$

Thermal damage begins when the tissue temperature rises above 44 °C. The burn damage is predicted by calculating the damage function, Ω, at each point basing on the assumptions of Henriques and Moritz [9] as follows:

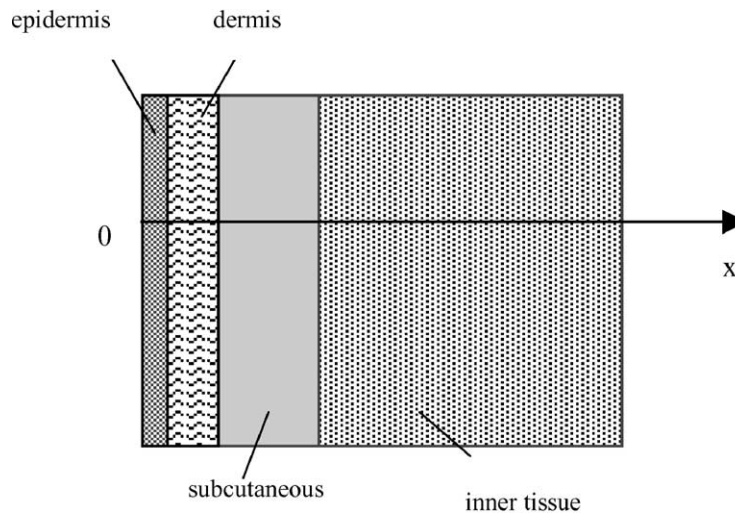$$\Omega(x) = \int_0^t P\exp\left(-\frac{\Delta E}{RT(x, t)}\right)\mathrm{d}t \tag{7}$$

Fig. 1. Schematic diagram of the multi-layer tissues.

Table 1
Thermal physical properties used in the skin

	Thickness (m)	Specific heat ($\mathrm{J\,kg^{-1}\,K^{-1}}$)	Blood perfusion rate ($\mathrm{m^3\,s^{-1}\,m^{-3}}$ tissue)	Thermal conductivity ($\mathrm{W\,m^{-1}\,K^{-1}}$)	Density ($\mathrm{kg\,m^{-3}}$)
Epidermis	80×10^{-6}	3590	0	0.24	1200
Dermis	0.002	3300	0.00125	0.45	1200
Subcutaneous	0.01	2500	0.00125	0.19	1000
Inner tissue	0.03	4000	0.00125	0.5	1000

Threshold burn injuries occur if $\Omega = 0.53$ and third-degree burn injuries occur if $\Omega = 10\,000$ [2].

3. Numerical method

A one-dimensional finite difference method was used to solve the bioheat Eq. (1). Non-uniform space meshes and uniform time steps were used in the model. The space steps and time steps are small enough to ensure that the transient temperatures were mesh-independent. An implicit time scheme was used to discretize the differential bioheat equation as follows:

$$\rho C \frac{T_i^{\,k} - T_i^{\,k-1}}{\Delta t} \Delta x = k_e \frac{T_{i+1}^{\,k} - T_i^{\,k}}{\Delta x} - k_w \frac{T_i^{\,k} - T_{i-1}^{\,k}}{\Delta x} + \omega_b \rho_b C_b (T_b - T_i^{\,k}) \Delta x \qquad (8)$$

where k was the effective thermal conductivity calculated using the harmonic mean. The Tridiagonal Matrix Algorithm [10] is used to solve the discretized equations. Then the damage function at nodes with the temperatures higher than the threshold was calculated using:

$$\Omega(i) = \sum_{k=k_i}^{k_f} P \exp\left(-\frac{\Delta E}{RT_i^{\,k}}\right) \Delta t \qquad (9)$$

where k_i indicated the time step when the node temperature was above the threshold and k_f indicated the final time step.

4. Results

The time step independent temperature distribution was chosen by calculating for $\Delta t = 0.01, 0.001$, and 0.0001 s. The results showed that $\Delta t = 0.001$ s was small enough to obtain time step independent temperature and damage function distributions. The numerical results also showed that the temperature variation decreased with depth. Therefore, the grid space was increased from the epidermis layer towards the inner tissue.

The temperature field in the tissue after 15 s of heating with a surface temperature of 90 °C was shown in Fig. 2. The temperatures of the epidermis and dermis tissue changed significantly, while the deeper tissue temperatures remained almost the same. Fig. 3 showed the damage function ($\Omega = 0.53$, $\Omega = 10\,000$) distributions with time. Most of the tissues near the surface suffered severe injury immediately after exposure, while in the deeper tissues, serious damage occurred after a relatively longer time period. The present results agreed well with those of Ng and Chua [5] for the conditions in their paper.

In another series of calculations, the initial temperature distribution was assumed to be uniform (34 °C) to evaluate the effect of the initial temperature distribution. The temperature distribution after 10 s and the damage function were
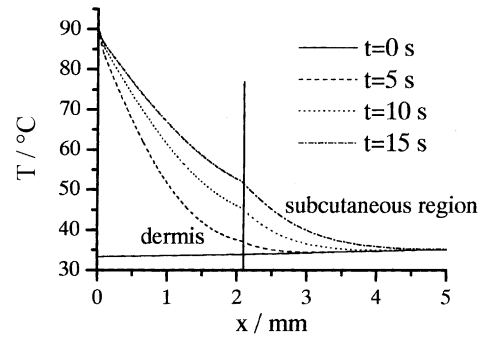

Fig. 2. Tissue temperature variations for a surface temperature of 90 °C.
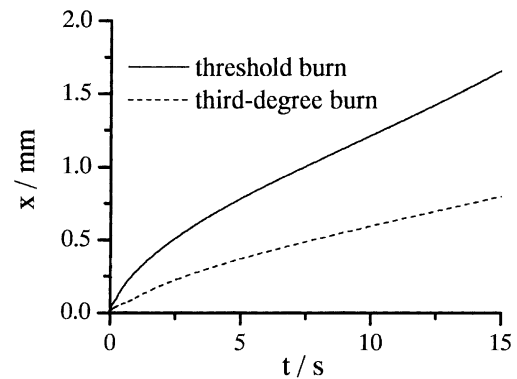

Fig. 3. Threshold ($\Omega = 0.53$) and third-degree ($\Omega = 10\,000$) burn distributions for a surface temperature of 90 °C.
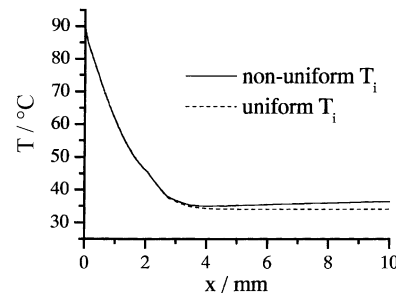

Fig. 4. Temperature distributions 10 s after exposure for uniform and non-uniform initial temperature distributions.
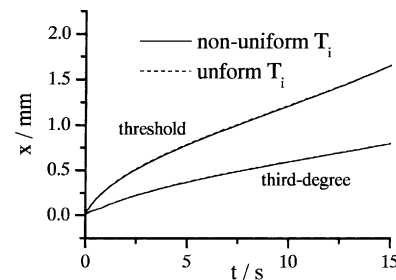

Fig. 5. Burn damage burn injuries for uniform and non-uniform initial temperature distributions.
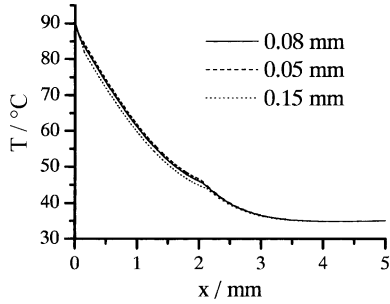
Fig. 6. Temperature distributions 10 s after exposure for different epidermis thickness and a dermis thickness of 2.0 mm.

Fig. 9. Threshold and third-degree burn injuries for different dermis thickness and an epidermis thickness of 0.08 mm.

compared with the previous results in Figs. 4 and 5. The temperature distributions within about 5 mm of the surface were almost the same with differences of several degrees deeper in the tissue where the damage was less extensive. Fig. 5 showed that the damage burn injuries for both conditions are almost the same. Therefore, the assumption of initial uniform temperature distributions in the previous analyses [2,4,5] provided reasonable estimates of the burn injuries resulting from short exposures.

The thicknesses of the epidermis and dermis differed for different peoples and different locations. The transient temperature and burn injury distributions for different epidermis thickness and dermis thickness were shown in Figs. 6–9. They showed that the epidermis and dermis thickness both significantly affected the transient temperature and burn injury distributions. Burn injuries greatly decreased with

increasing epidermis and dermis thickness. Therefore, in establishing an animal skin burn model, the animals should have similar epidermis and dermis thickness to ensure the repeatability. In addition, the heat source should be applied essentially the same location each time. An alternative would be to apply different exposure times to each animal according to their specific epidermis and dermis thickness.

The effects of variations of blood perfusion in the dermis layer and in deeper layers on the transient temperature and burn injury distributions were also calculated. The results showed that the effect of blood perfusion can be ignored in predicting the burn injuries for short duration exposures. The heat capacity of the tissues played an important role in preventing the deeper tissues from thermal damage.

5. Conclusion

A one-dimensional multi-layer finite difference model was developed in this paper to calculate transient temperature and burn injury distributions in living tissues. The model was used to analyse the effects of variations in thermal physical and geometrical properties on the transient temperature and damage function distributions. The results showed that the epidermis and the dermis thickness significantly affected the transient temperature and burn injury distributions, while the initial temperature distribution and the blood perfusion had little effect. Therefore, animal skin burn studies should consider the effects of different dermis and epidermis thicknesses, different heating locations and different exposure times. The model accuracy was restricted by the accuracy of the thermal properties of the tissues during exposure and the accuracy of the Henriques model.
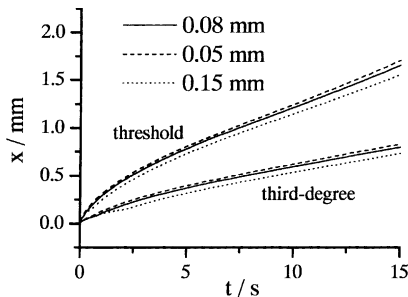

Fig. 7. Threshold and third-degree burn injuries for different epidermis thickness and a dermis thickness of 2.0 mm.
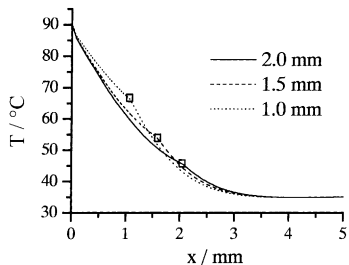

Fig. 8. Temperature distributions 10 s after exposure for different dermis thickness and an epidermis thickness of 0.08 mm. The dermis thickness is indicated by the square symbol.
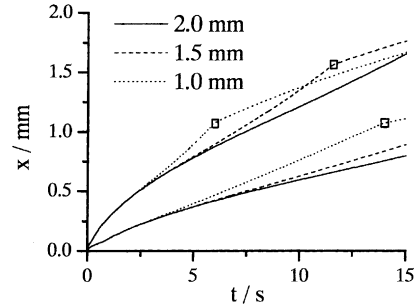
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