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Numerical Models of Laser Fusion of Intestinal Tissues

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Abstract— **Numerical models of continuous wave Tm:YAG thermal fusion in rat intestinal tissues were compared to experiment. Optical and thermal FDM models that included tissue damage based on Arrhenius kinetics were used to predict birefringence loss in collagen as the standard of comparison. The models also predicted collagen shrinkage, jellification and water loss. The inclusion of variable optical and thermal properties is essential to achieve favorable agreement between predicted and measured damage boundaries.**

I. Introduction

Tissue fusion is dominated by irreversible thermal alteration in tissue collagen. As the temperature increases collagen fibers shrink in length and swell in caliber in an approximately iso-volumic process. The fibers, which normally assume a twisted rope-like array due to inter-fibril bond sites, unravel and lose their rope-like structure — up to approximately 65 % shrinkage. [1, 2] The native state collagen array is birefringent — i.e. able to rotate polarized light. It loses this property as it shrinks and the bonds break. [3] As further heating takes place the fibrillar molecular structure breaks down and the collagen jellifies, forming an amorphous mass. Apposed amorphous collagen masses can "agglutinate", as it were, fusing the apposed tissues.

Intestinal tissues are not collagen-rich but are potentially good candidates for laser fusion since the mucosa contains a collagenous scaffold. Additionally, rat bowel wall is similar in size and anatomical structure to the distal third of the human Fallopian tube. Studies of tissue response to laser wavelengths at 2.01 μm are of interest since minimally invasive endoscopic procedures may be facilitated by an effective laser fusion technique.

This process was studied experimentally and reported in a previous paper. [4] However, attempts at that time to effectively model the histologic and thermal field results fell woefully short of acceptable accuracy. The cause of this failure was the assumption of constant optical and thermal properties in the numerical model work. New numerical models, presented here, clearly illustrate the overwhelming importance of including variable physical properties in numerical model studies.

When numerical models are sufficiently well behaved they can be used to study transient microstructural thermodynamic events, and to compare dominant processes and effects on a fine spatial and temporal scale not possible in experiments alone. These models assume an idealized tissue (linear, homogeneous and isotropic) and an idealized geometry (smooth flat surface) in order to study the governing processes. Despite that tissue properties are not particularly well known, excellent agreement was obtained between the experimental and model results by the inclusion of variable physical properties.

The results obtained in this new study agree in both the transient surface temperature and dimensions of the histologic findings, indicating that all of the dominant physical processes are adequately represented in the model work.

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II. Numerical Model Methods

A. Laser Power Absorption

The Finite Difference Method FDM model space was 2-D axisymmetric, 101 nodes radially (r) by 51 nodes axially (z). The laser wavelength modeled was from a thulium YAG laser, TM:YAG, at λ = 2.01 μm. This wavelength is dominantly absorbed by tissue water; consequently, scattering is not an important consideration. The laser beam was considered gaussian in cross-section, confirmed by thermographic imaging of the tissue surface. This is in keeping with the separation distance between the optical fiber and the tissue surface — typically 1 cm, or about 30 fiber diameters. Consequently, the laser heat generation term is:

$$Q_{gen}(r,z) = \left[0.8\, w(r,z) + 0.2\right] \Phi(0,0)\, e^{\left[-\mu_{eff} z\right]}\, e^{\left[\frac{-r^2}{2\sigma^2}\right]} \quad (1)$$

where: Q_{gen} = the local volumetric power density (W/m^3), w = tissue water volume fraction, Φ = beam surface fluence rate coupled to the tissue (W/m^2), μ_{eff} = effective optical absorption coefficient (m^{-1}), and σ = effective beam radius (m). In the models, 80% of the tissue heating was from the tissue water and 20% from the residual tissue components (proteins), as suggested by experience in other experiments (not part of this work).

B. Thermal Model

The thermal model calculates local temperatures, T, from the volumetric heat generation using the energy equation:

$$\rho c \frac{\partial T}{\partial t} = k\nabla^2 T + Q_{gen} - Q_{fg} - \text{Surface Losses} \qquad (2)$$

where: ρ = density (kg/m^3), c = specific heat (J/kg/K), k = thermal conductivity (W/m/K), Q_{fg} = latent heat of water vaporization (W/m^3), and surface losses includes convection and radiation heat transfer and also surface evaporation at both the upper and lower surfaces. The centerline (r = 0) is adiabatic. Equilibrium boiling at 1 atmosphere is assumed for tissue water (i.e. 100 °C), which turns out to be a deviation from the measured surface temperature history, most likely due to increased subsurface pressures in the tissues. Tissue was only allowed to increase above equilibrium boiling if fully dry.

Key to the success of the numerical model work was the inclusion of variable thermal properties. Model thermal properties were estimated from correlations first presented by Cooper and Trezek in 1971, and later summarized by Diller *et a*l. [5] The correlations are:

$$\rho = \frac{1}{m_w + 0.649\, m_p + 1.227\, m_f} \qquad (3a)$$

$$k = \rho\,(6.28\, m_w + 1.17\, m_p + 2.31\, m_f) \qquad (3b)$$

$$c = 4.2\, m_w + 1.09\, m_p + 2.3\, m_f \qquad (3c)$$

where: ρ = density (g cm^{-3}), k = thermal conductivity (mW cm^{-1} K^{-1}), c = specific heat (J g^{-1} K^{-1}), and m_w, m_p and m_f are the mass fractions of water, protein and fat, respectively. Bowel was assumed to have an initial water mass fraction of m_w = 0.55 on a wet weight basis (volume fraction = 65.3 %), and zero fat (adipose) tissue.

C. Tissue Thermal Damage Model

An Arrhenius kinetic model was used to predict birefringence loss in tissue collagen and in an analogous formulation for collagen shrinkage based on [1, 2]. The Arrhenius model calculates a damage parameter, Ω, which is the natural log of the ratio of initial birefringence intensity, C(0) to the final value, C(τ):

$$\Omega(\tau) = ln\left\{\frac{C(0)}{C(\tau)}\right\} = \int_0^{\tau} A\, e^{\left[\frac{-E_a}{R\,T(t)}\right]}\, dt \qquad (4)$$

where: R = the gas constant (8.3143 J/mole/K) and T is the absolute temperature (K). The kinetic model is hyperbolically sensitive to absolute temperature in the exponent and linearly dependent on time. This is a classic thermal damage formulation modified to provide a probabilistic prediction. [5] At the conclusion of the transient thermal model the probability of resulting irreversible thermal damage is then determined from:

$$\text{Damage}(\%) = 100\left(1 - e^{-\Omega}\right) \qquad (5)$$

Birefringence loss in collagen is unique to thermal damage and is due to disruption in the regularity of the array of collagen fibers. Kinetic coefficients for this process were: E = 306 (kJ/mole) and A = 1.606 x 10^{45}, as in [6].

The collagen shrinkage model of Chen et al [1, 2] reduces their data to a single shrinkage curve characterized by an equivalent exposure time, τ_2, given by:

$$\tau_2 = e^{\left[\alpha + \beta P + M/T\right]} \qquad (6)$$

where: α = -152.35; β = 0.0109 (kPa^{-1}); P = applied stress (kPa); and M = 53,256 (K). Here the exponential term is positive because it appears in the denominator: $\alpha = -\ln\{A\}$ and M = E/R. The βP term compensates for the applied stress, and is not included in this model (P = 0). The equivalent exposure is normalized to a nondimensional time axis, $\nu = \ln\{t/\tau_2\}$, and then curve fit parameters are applied to calculate the overall shrinkage, ξ (%):

$$\xi = \left(1 - f(\nu)\right)\left[a_0 + a_1\,\nu\right] + f(\nu)\left[b_0 + b_1\,\nu\right] \qquad (7a)$$

where: a_0 = 1.80, a_1 = 0.983, b_0 = 42.4, b_1 = 3.17 (all in %),

$$f(\nu) = \frac{e^{a(\nu - \nu_m)}}{1 + e^{a(\nu - \nu_m)}} \qquad (7b)$$

and: a = 2.48, and $\nu_m = \ln\{\tau_1/\tau_2\}$ = -0.77.

III. RESULTS

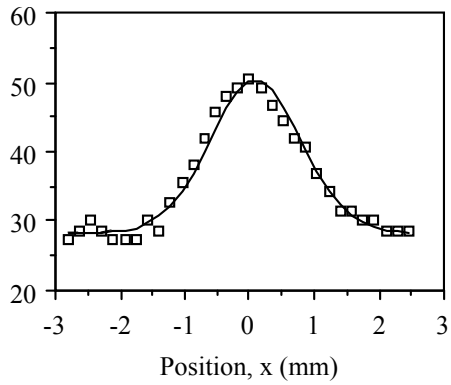

Fig. 1. Experimental exposure that resulted in a successful fusion. Squares are the thermographic data, and the line is a Gaussian fit to the experimental data at 0.2 s with total power 0.55 W and 2σ spot diameter of 2.84 mm.

The model results were compared to a successful bowel fusion experiment. [4] The laser spot had a total power of 0.55 W for 5 s, and 2σ spot diameter = 2.84 mm. The spot surface temperature was measured with an 8 to 12 μm thermal camera (Inframetrics Model 525). At 0.2 s into the 5 s exposure the spot center temperature was 52 °C (Fig. 1)

A. Comparison of Transient Temperatures

By the end of the laser pulse the center temperature had risen to approximately 105 °C. The experimental and

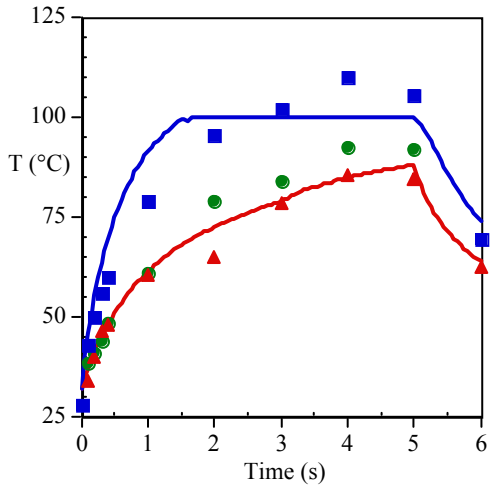

Fig. 2. Model temperature prediction at r = 0 and r = σ (lines). Squares are spot center experimental data, circles and triangles are respectively at ± σ (r = 0.71 mm).

numerical model temperatures are shown in Fig. 2. The numerical model has successfully predicted the surface temperatures at ± σ (r = 0.71 mm), and is quite close at the center (r = 0). Confirmation that the heat transfer effects are successfully modeled is given by the cooling curve, which matches the experimental result quite closely. The 1σ (r = 0.71 mm) temperature rise curve at about 3 s in Fig. 2 shows the effect of tissue water loss, corresponding to an

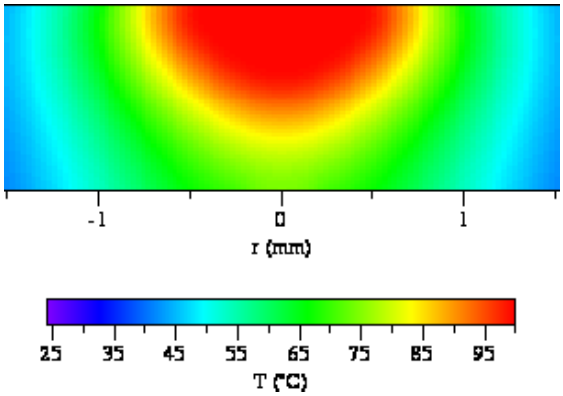

Fig. 3. Model temperature prediction at the end of heating. Width: ± 1,5 mm, thickness 1 mm (to scale).

analogous event in the experimental data.

In Fig. 3 the 2D model temperature rise plot has much deeper penetration than the previous constant property model owing to the deeper penetration of the laser flux following surface water loss, and to decreases in surface tissue thermal properties (see Table 1).

B. Comparison to Histologic Data

Histologic results for the experiment modeled are in Fig. 4. Steam vacuoles (V) are clearly seen in the light microscopic image (top), as is damage to the villae and mucosa (M). The image is at an original magnification of

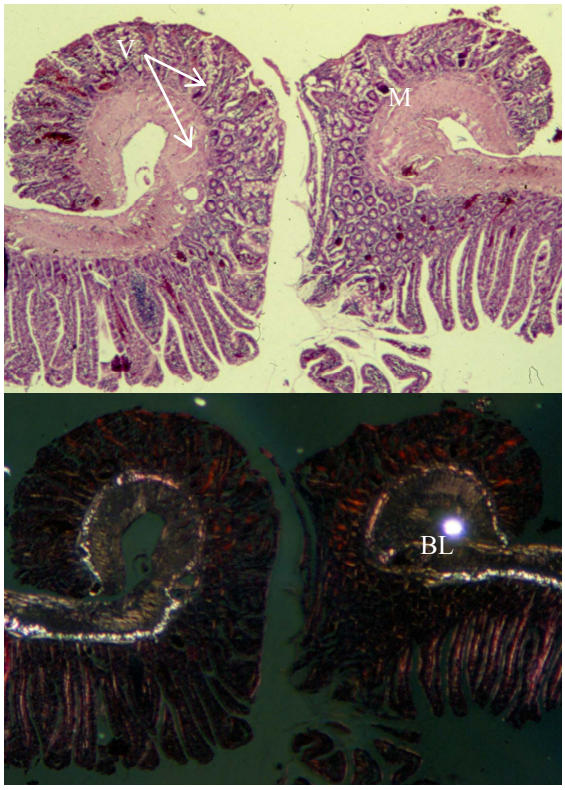

Fig. 4. Histologic result for the experiment. (Top) Light microscopic (LM) image (H&E stain) with (Bottom) corresponding transmission polarizing microscopy (TPM) birefringence image, both at original magnification of 10X. Birefringent collagen creates a bright field.

10X with hematoxylin and eosin stain. The bowel wall has a tendency to evert when resected, so the villae wind up in the illumination field of the laser. The numerical model geometry is a considerably simplified version of this since it

TABLE I
COMPARISON OF EXPERIMENT AND MODEL RESULTS

Tissue Damage Marker	Experiment	Variable Property Model	Constant Property Model
80 °C Contour			
r_{max}		0.82 mm	0.64 mm
z_{max}		0.78 mm	0.41 mm
Collagen 50% Biref. Loss			
r_{max}	0.75 mm	0.81 mm	0.67 mm
z_{max}	0.80 mm	0.84 mm	0.46 mm
Collagen Jellification			
r_{max}		0.60 mm	
z_{max}		0.50 mm	
50% Water Loss			
r_{max}		0.28 mm	
z_{max}		0.21 mm	

Collagen jellification boundary = 65% shrinkage.

is treated as a flat surface. The strain of histologic sectioning has separated the originally-successful fusion seam in these images.

The lower image in Fig. 4 is a transmission polarizing microscopy (TPM) version of the same histologic section. The mucosal collagen layer constitutes the bright field in this image. Birefringence intensity is lost in the heated collagen (BL).

In Table I, histologic section birefringence loss data for the experiment are compared to the model results (assuming a 50% birefringence loss criterion). Including temperature dependent optical and thermal properties has yielded results that compare very favorably to the measurements despite the geometric simplifications. The improvement in result accuracy over the constant property model is remarkable. Using birefringence loss as a credible calibration source suggests that the other damage process predictions, for which histologic assessment is not feasible, have substantial validity as well.

IV. Conclusion

The improved numerical model study illustrates the importance of including variable optical and thermal properties. Direct comparison to the thermographic imagery and histologic data confirm that the essential physical phenomena are now adequately represented in the model. The much-improved validity of this approach is demonstrated in the results.

Acknowledgments

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