Estimating the Thermal Dose From Backscattered RF Echoes

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Abstract. Over the years many different investigators have attempted to estimate the temperature rise resulting from ultrasound exposure in therapy applications. The developed methods typically rely on thermal expansion or temperature related sound speed variations both of which are dependent on tissue type and cannot be known *a priori* on a patient-specific basis. We have developed a method to estimate the thermal dose (temperature over time) from the backscattered RF echoes. RF echoes from within the same tissue type can be used to estimate the *in vivo* local attenuation (assumed to be the same as tissue absorption) as has been shown by other investigators. Similarly, the RF echoes can be compared to reference echoes while assuming a model for the scattering structures to estimate the total attenuation along the propagation path (i.e., the *in vivo* power spectrum). Hence, the temperature over time can be estimated from the measured in vivo power spectrum and the measured tissue absorption by solving the bioheat equation directly. The estimated thermal dose can then be used to monitor or plan ultrasound therapy on a patient-specific basis.
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INTRODUCTION

As the effectiveness of ultrasound thermal therapy continues to be demonstrated in new applications, there is a need to quantify the temperature increase resulting from the ultrasound exposure. In this work, we investigated a new method for determining the temperature increase, T', on a patient specific basis by directly solving the bioheat transfer equation given by,

$$\rho_c c_V \frac{\partial T'}{\partial t} = q_{blood} + \nabla \cdot \left(\kappa_t \nabla T' \right) + \sum_{i=0}^{\infty} \alpha_{loc} \left(\omega_i \right) I' \left(\omega_i \right) \qquad (1)$$

where t is time, ω_i is the radian frequency, $\rho_c c_V$ is the heat capacity of the tissue, κ_t is the thermal conductivity of the tissue, q_{blood} is the rate of heat removal due to blood flow, α_{loc} is the absorption of the ultrasound at the location of the heat generation, and I' is the ultrasound intensity at the location of heat generation. The heat capacity of most tissues is approximately 4 J $\cdot$ cm^{-3} $\cdot$ ^{o}C^{-1} while the thermal conductivity is approximately 0.5 W $\cdot$ m^{-1} $\cdot$ ^{o}C^{-1} [1]. Hence, the resulting temperature rise can be determined once the local absorption, blood perfusion, and in vivo ultrasound exposure intensity has been determined. Quantifying blood perfusion and local attenuation (assumed to be the same as local absorption) has been investigated in detail by other researchers [2,3]. In addition, the *in vivo* exposure intensity can be calculated from a calibrated power spectrum once the attenuation along the

propagation path has been determined on a patient specific basis. Hence, our work focused on reliably determining the attenuation along the propagation path.

ATTENUATION ESTIMATION

The attenuation along the propagation path can be measured if the impedance distribution (i.e., shape) of the structure responsible for the scattering can be estimated. One common impedance distribution hypothesized for tissue scatterers at frequencies less than ~10 MHz is the Gaussian impedance distribution. The expected backscattered voltage spectrum returned from a random distribution of Gaussian scatterers satisfying the born approximation assuming a plane wave in the focal region (i.e., weakly focused source) is given by,

$$E\left[\left|V_{refl}\right|^2\right] \propto k^4 \left|V_{plane}\left(f\right)\right|^2 e^{-0.827\left(ka_{eff}\right)^2} e^{-4\alpha f z_T} \tag{2}$$

where V_{refl} is the voltage spectrum returned by the scatterers, V_{plane} is the voltage spectrum returned from a reference plane placed at the focal plane, k is the wavenumber, f is the frequency, a_{eff} is the effective radius of the scatterer, α is the attenuation along the propagation path, and z_T is the distance from the aperture of the transducer to the focal plane. In tissue, both a_{eff} and α are unknown. Hence, both must be estimated simultaneously on a patient specific basis by minimizing

$$ASD = \underset{f}{\text{mean}}\left[\left(X\left(f, a_{eff}, \alpha\right) - \bar{X}\left(a_{eff}, \alpha\right)\right)^2\right] \tag{3}$$

where

$$\begin{aligned} X &= \ln\left(E\left[\left|V_{refl}\right|^2\right]\right) - \ln\left(k^4 \left|V_{plane}\right|^2 e^{-0.827\left(ka_{eff}\right)^2} e^{-4\alpha f z_T}\right) \\ \bar{X} &= \underset{f}{\text{mean}}\left[X\left(f, a_{eff}, \alpha\right)\right]. \end{aligned} \tag{4}$$

SIMULATION RESULTS

The total attenuation measurement was validated using computer simulations of an f/4 source with a 5 cm focal length radiating into an infinite half-space containing scatterers with Gaussian impedance distributions. The reference voltage spectrum from a plane placed at the focal plane in the simulations was given by,

$$\left|V_{plane}\left(f\right)\right| \propto \left|f\right|^2 \exp\left(-2\left(\frac{f - f_o}{\sigma_o}\right)^2\right) \tag{5}$$

where f_o was 8 MHz and σ_o was 2, 4, or 6 MHz. The attenuation of the half-space was homogenous for each case and was varied from 0 to 1 dB/cm-MHz for the different cases. The scatterers were placed at a density of 35/mm3 for all of the cases while a_{eff} was varied from 5 to 150 μm for different cases. However, for a particular half-space, all of the scatterers had the same size. The simulated RF echoes were sampled at 53

MHz and windowed with a Hamming window with a length of 3 mm. The effect of noise was also considered by adding white zero mean random noise to some of the cases so that the signal to noise ratio varied from 3 to 36 dB. (The main effect of the noise was to reduce the usable frequency range). The algorithm used all signal values greater than 6 dB above the noise floor of the power spectrum except for low noise levels where the –20 dB bandwidth of the power spectrum limited the frequency range used. For each case considered in the simulations, 1000 waveforms were generated then grouped/averaged in sets of 25 to yield 40 estimates of $E[|V_{refl}|^2]$ resulting in 40 estimates of a_{eff} and α per case. The error in the average attenuation estimate was consistently better than 0.2 dB/MHz whenever the frequency range used to obtain the estimate was larger than 4 MHz.

DISCUSSION

Now that the accuracy and precision of the attenuation estimate has been assessed for many different cases, the errors in the attenuation can be translated to errors in the resultant temperature increase estimate assuming that there are no errors in the other parameters of the Bioheat equation. In general, the temperature increase will be directly proportional to the *in vivo* intensity regardless of the beam geometry (neglecting nonlinear effects) [4]. Hence, the percent error in temperature increase due to an error in the attenuation along the propagation path is given by,

$$\%\text{Error in Temperature} = 100\left(\exp\left(-2\Delta\alpha f\right)-1\right) \tag{6}$$

where $\Delta\alpha$ is the error in the attenuation. Also, since the temperature increase at a specific location in tissue is strongly effected by other heat sources within a perfusion length, deviations in the attenuation estimate will be smoothed by the temperature calculation and errors in the temperature estimate will be correlated with error in the average attenuation value. Therefore, an error in the attenuation estimate less than 0.2 dB/MHz (i.e., accuracy for a frequency range greater than 4 MHz) assuming a 2 MHz therapy exposure translates to an error less than 9% in the temperature increase estimate.

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