Temperature dependence of ultrasonic propagation speed and attenuation in excised canine liver tissue measured using transmitted and reflected pulses

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Previous reported data from our laboratory demonstrated the temperature dependence of propagation speed and attenuation of canine tissue *in vitro* at discrete temperatures ranging from 25 to 95 °C. However, concerns were raised regarding heating the same tissue specimen over the entire temperature range, a process that may introduce irreversible and, presumably, cumulative tissue degradation. In this paper propagation speed and attenuation vs temperature are measured using multiple groups of samples, each group heated to a different temperature. Sample thicknesses are measured directly using a technique that uses both transmitted and reflected ultrasound pulses. Results obtained using 3 and 5 MHz center frequencies demonstrate a propagation speed elevation of around 20 m/s in the 22–60 °C range, and a decrease of 15 m/s in the 60–90 °C range, in agreement with previous results where the same specimens were subjected to the entire temperature range. However, sound speed results reported here are slightly higher than those reported previously, probably due to more accurate measurements of sample thickness in the present experiments. Results also demonstrate that while the propagation speed varies with temperature, it is not a function of tissue coagulation. In contrast, the attenuation coefficient depends on both tissue coagulation effects and temperature elevation.
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I. INTRODUCTION

Numerous articles on ultrasonic propagation speed and attenuation in soft tissue can be obtained from the retrievable literature. Several papers, for example by Wells,[1] Goss,[2,3] and Duck[4] have summarized data on ultrasonic propagation speeds and attenuation coefficients in human and animal tissues. However, previously reported data on the temperature dependence of the propagation speed and attenuation in tissues generally are limited to maximum temperatures of about 65 °C.[5–9] The emerging use of thermal ablative therapies necessitates the acquisition of information on the variation in these parameters at a higher temperature. As tissue is heated during ablation, it expands as well as undergoes changes in the ultrasonic propagation speed and attenuation. These changes introduce time shifts in backscattered ultrasound echo signals from the heated region. The variations in these parameters during heating have been used to compute the temperature distribution in tissue during ablative procedures.[10–16]

Conventional transabdominal ultrasound imaging is presently used during rf ablation procedures for guidance, monitoring, and placement of the electrodes.[17,18] Advantages of ultrasound include widespread availability, real-time guidance for electrode placement, and accurate, convenient needle puncture guides. However a significant drawback of current ultrasonic methods is that the zone of necrosis produced during rf, microwave or high-intensity focused ultrasound (HIFU) ablation is not easily visualized by transabdominal sonography because of the low intrinsic contrast between normal and ablated liver. The poor visualization of the ablated region has prompted clinicians to seek other methods such as x-ray CT to guide the ablation procedure.[19]

Ultrasound guidance of ablation therapy could be im-

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proved if changes in echo arrival time or other temperature related parameters during heating could be used to monitor heating patterns and estimate temperature distributions. Several ultrasonic methods have been proposed to estimate temperature. These include monitoring attenuation,[16] backscattered power,[20] and propagation speed[21] changes with temperature. Thermal expansion either without[11] or with propagation speed variations[11–16] have also been monitored. Several of these techniques are based on tracking the echo-shift in the time-domain, and differentiating the time-shift estimates along the axial direction to obtain a temperature profile.[11–16] Although promising, none of the ultrasonic methods proposed above[12,13] has been applied to measure temperature changes larger than a few degrees, with Simon *et al*. estimating the largest temperature rise—from 23 to 43 °C over 208 minutes.[12] Our preliminary results both *in-vitro* and *in-vivo* suggest the possibility to provide real-time tracking of 2-D temperature changes over the large range of temperatures traditionally used in ablative therapy (40–100 °C) during the procedure.[14]

We have previously reported the temperature dependence of propagation speed and attenuation of canine tissue samples *in vitro* at discrete temperatures, ranging from 25 to 95 °C.[22] The procedure used in Techavipoo *et al.*[22] involved raising the same tissue sample to all measurement temperatures. This procedure where the same tissue sample was raised to all the temperatures in the measurement protocol, however, may introduce an irreversible and presumably cumulative degradation of the tissue structure, thereby altering the values of the propagation speed and attenuation obtained. In this paper, the measurement protocol was changed such that each tissue specimen was raised to a single elevated temperature. The results obtained in this paper demonstrate that any tissue degradation incurred by subjecting samples to the entire range of temperature elevations did not significantly affect the propagation speed or attenuation coefficient changes in tissue.

II. MATERIALS AND METHODS

The method used to measure the propagation speed and attenuation coefficient is similar to the one discussed by Techavipoo *et al.*[22] However, information obtained from reflected pulses off the sample walls is used here for an accurate estimation of tissue sample thickness in the direction of insonification.[23] In addition, the experimental protocol was modified such that sound speeds and attenuation in each tissue specimen was measured at only one temperature.

A. Apparatus

The equipment used is shown in Fig. 1. Two water baths are shown: one for performing acoustical measurements (measurement bath) and the other for heating and retaining the sample (heating bath). Ultrasound pulses were generated by a computer-controlled pulser/receiver (Panametrics model 5800, Waltham, MA) driving a single element, unfocused transducer (Panametrics V309). The acoustic signal was transmitted through a tissue sample positioned within a tissue holder and received by a second transducer (Aerotech Delta PN2794-3, Krauthkramer Inc., Lewistown, PA). In addition, ultrasound echoes from the interfaces at the front and the back of the tissue holder were detected by the Panametrics transducer, operated in a pulse-echo mode. Both transducers have a 5 MHz center frequency. The signals from the receiving transducer as well as the echo signals from the pulse-echo transducer were amplified in the pulser/receiver, and each was digitized at a 100 MHz sampling rate using a 12 bit data acquisition board (Gage Applied Sciences Inc., Lachine, QUE, Canada). Signals were stored for offline processing.

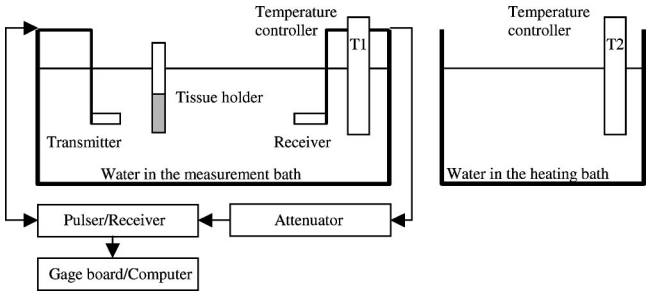

FIG. 1. The apparatus used for the experimental measurements of the propagation speed and attenuation coefficient.

B. Tissue preparation

Canine livers were obtained from 10-month old male hounds available from unrelated studies in an adjoining lab. The excised liver tissues were obtained from animals that do not undergo any procedures on the liver, and hence were considered to be normal specimens. Each individual liver was cut into samples in approximate dimension of $1.6\times5\times5$ cm. Samples did not contain large blood vessels or bile ducts. Five samples (S1, S2, S3, S4, S5) were obtained from each excised liver for the five different temperature elevations in the measurement protocol. Each of these specimens was placed into a sample holder (shown in Fig. 1) along with a degassed saline buffer. Ultrasound propagation measurement was initiated less than 5 hours after excision of the liver. The experiment was repeated using tissues from 10 different animals to obtain the mean and standard deviation of the propagation speed and attenuation over different animals.

C. Data acquisition

Propagation speeds and attenuations for all five liver samples (S1, S2, S3, S4, S5) in each group were first measured at temperatures of 22 °C and 37 °C. The temperature of 37 °C was used since this temperature is close to the animal body temperature *in-vivo*. The samples in their holders were heated by placing them into the heating bath with a preset measured water temperature for duration of 20 minutes. We assumed that the tissue temperature within the holder would reach the preset temperature of the heating bath within the 20 minute interval. The five different tissue samples (from the same excised liver) were heated to different preset temperatures, i.e., S1 to 50 °C, S2 to 60 °C, S3 to 70 °C, S4 to 80 °C, and S5 to 90 °C in our measurement protocol. The propagation speeds and attenuations of the samples were also measured again at 37 °C after the tissue

was cooled. For the measurements at 22 and 37 °C, the temperature in the measurement bath was set to the target temperature, and every sample was equilibrated to the measurement temperature. In contrast, for the measurements at the higher target temperatures, the temperature in the measurement bath was held at 37 °C while samples were heated and retained in the heating bath at the target temperature. Propagation speed and attenuation of the tissue specimen in the sample holder was then immediately measured in the measurement bath. The measurements were done in the order of lowest to highest temperatures. Because of the temperature difference between the temperatures of the tissue sample and the water in the measurement bath, care was taken to ensure that time shifts and transmitted amplitudes were recorded within 5 seconds after transferring the sample to the measurement bath. The variations in the tissue temperatures were limited to the amount of heat transferred between the tissue and the water in the measurement bath during the measurement.

D. Method

The propagation speed and attenuation coefficient were determined using the substitution technique.[22,23] For each measurement four signals were recorded, as described below, with and without the sample inserted into the ultrasound path. We record four pulses, namely the transmission pulse through water without the sample present $P_w(t)$, the transmission pulse with the sample inserted $P_s(t)$, and echoes from the proximal and distal sample windows, $P_1(t)$ and $P_2(t)$, respectively. Note that $t=0$ corresponds to the time when the pulser/receiver transmits the corresponding pulse.

Propagation speeds in tissue samples were calculated[23] using the equation

$$V_p(f)=c_w\left[1+2\times\frac{\phi_w(f)-\phi_s(f)+2\pi f(t_w-t_s)}{\phi_2(f)-\phi_1(f)+2\pi f(t_2-t_1)}\right], \quad (1)$$

where c_w is the sound speed in water, ϕ denotes the phase spectrum of a pulse that was shifted from its center to zero, and t is the corresponding time-shift. The subscripts w and s refer to the water reference and the sample, respectively. The sample thickness along the beam propagation path was estimated using

$$\hat{L}=\frac{c_w}{4\pi f}[\phi_2-\phi_1+2\phi_w-2\phi_s+2\pi f(t_2-t_1+2t_w-2t_s)]. \quad (2)$$

The frequency dependence of the phase spectra on the right side of Eq. (2) cancels out making $\hat{L}$ a constant. However, $\hat{L}$ estimated from the experimental data may vary with the frequency. The variance of $\hat{L}$ can be used to ascertain the reliability of the experiment.[23]

The measurement of propagation speed and sample thickness involves the utilization of the absolute phase spectra of the pulses. The phase information from Fourier transforms wraps around zero, making it difficult to recover the absolute phase spectra. Before implementing Eqs. (1) and (2), the center of each pulse was shifted to $t=0$; therefore the absolute phase evolves slowly with frequency and is easier to track and unwrap. However, the transducers used in the experiment have a 60% bandwidth, with phase information outside the bandwidth not suitable for tracking the phase evolution with frequency.

We defined the center of the pulse at the position of the maximum of its envelope, and we unwrapped the phase starting at 4-MHz frequency for 5 MHz center frequency pulses. Since the transducer used in the experiment was narrowband, phase unwrapping could not be initiated starting at zero frequency. The magnitude spectra of pulses at frequencies lower than 4 MHz were very small, resulting in high uncertainty in the phase spectra. Assuming that the maximum phase evolution from 0 to 4 MHz lies within $\pm 2\pi$, the possible phase after shifting the center to zero can be written as

$$\phi(f)=\phi_{\text{unwrap}}(f)\pm 2n\pi, \quad \text{for } n=0 \text{ or } 1, \quad (3)$$

where ϕ_{unwrap} is the phase after unwrapping. We obtained phase estimates by substituting the phase spectra in Eq. (2) using Eq. (3) and selecting the combination of the phases that provided the lowest thickness variation. The standard deviation of the thickness estimates for each measurement at the frequencies between 4 MHz and 6 MHz after this process is less than 0.05 mm. This combination of the phases was then substituted into Eq. (1) to obtain the propagation speed.

The attenuation coefficients in units of dB/cm were calculated from the magnitude spectra of $P_w(t)$ and $P_s(t)$ using

$$\alpha(f)=(20/\hat{L})\log_{10}[A_w(f)T_{\text{total}}(f)/A_s(f)], \quad (4)$$

where A_w and A_s are the magnitude spectra, and T_{total} is the combined amplitude transmission coefficient through the sample windows.[24] T_{total} is calculated using

$$T_{\text{total}}(f)=\frac{4Z_1Z_3}{(Z_1+Z_3)^2\cos^2(2\pi fl/c_2)+(Z_2+Z_1Z_3/Z_2)^2\sin^2(2\pi fl/c_2)}, \quad (5)$$

where the subscripts 1, 2, and 3 refer to the properties of the water in the measurement bath, the thin layer material, and the tissue sample, respectively; Z denotes the acoustic impedance, l is the thin layer thickness of the tissue holder; and c_2 is the sound speed in the thin layer. We assume that the thin layer properties are constant with temperature, $Z_2=4.25\times10^6$ kg/m^2 s, $c_2=2540$ m/s, and $l=25\ \mu$m.[24] The acoustic impedances of the tissue samples and water are cal-

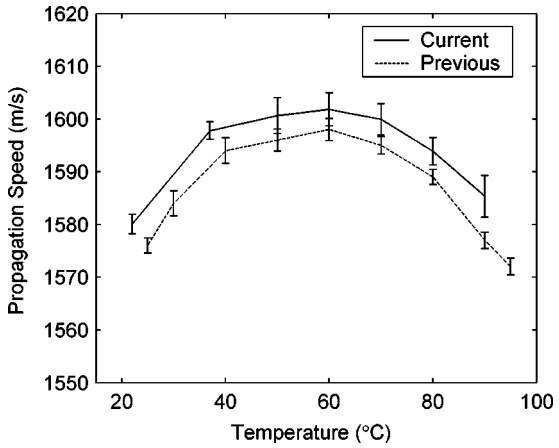

FIG. 2. Plots of mean propagation speed in canine liver tissue measured at different temperatures compared to the previous results reported in Techavipoo *et al.* (Ref. 22). Note that error bars are plus and minus one standard error across 10 different animals.

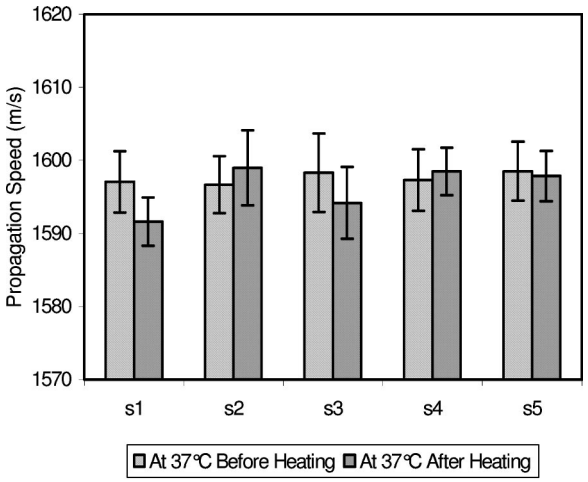

FIG. 3. Bar plots comparing the propagation speeds in canine liver tissue measured at 37 °C before and after the tissue samples were heated to the elevated target temperatures. Tissue sample S1 was raised to a single target temperature of 50 °C, S2 to 60 °C, S3 to 70 °C, S4 to 80 °C, and S5 to 90 °C, respectively.

culated from the multiplication of their densities and sound speeds. Canine liver tissue density is assumed to be equal to that of human liver, 1.050×10^3 kg/m^3,[4] and constant with temperature. The sound speeds in water and the water densities at different temperatures were adopted from Greenspan and Tschegg[25] and Gill,[26] respectively. Using these constants and the mean values over the 4 MHz to 6 MHz range for the propagation speed in samples and their thicknesses, the attenuation coefficient can be estimated using Eq. (4).

III. RESULTS

Figure 2 presents the speed of sound vs temperature for samples evaluated in this experiment. The solid line denotes the results obtained here, while the dashed line illustrates our previous results heating each sample to all measurement temperatures, as reported in Techavipoo *et al.*[22] These values are also shown in Table 1. Both plots demonstrate the same trend in the results, with the propagation speed increasing with temperature below 60 °C, reaching a maximum value at 60 °C, and then decreasing with a further temperature increase.

TABLE I. A comparison between the current and previous measurements of the propagation speeds in canine liver tissue at different temperatures.

	Current propagation speed (m/s)		Previous propagation speed (m/s)	
Temperature (°C)	Mean	Standard error	Mean	Standard error
22	1580	1.85	-	-
25	-	-	1576	1.40
30	-	-	1584	2.38
37	1598	1.66	-	-
40	-	-	1594	2.44
50	1601	3.39	1596	2.10
60	1602	3.16	1598	2.08
70	1600	2.97	1595	1.68
80	1594	2.61	1589	1.45
90	1585	3.95	1577	1.54
95	-	-	1572	1.58

All sound speeds measured in this experiment are about 5 m/s larger than our previous results. The cause of this difference likely can be explained as follows. In our previous experiment, propagation speed in tissue was calculated using $c_m = c_w/(1-c_w\,\Delta t/d)$, where c_w, Δt, and d are the sound speed in water, time-shift between the pulses with and without sample insertion, and sample thickness, respectively. The sample thickness was measured using a caliper outside the water tank, but the time-shift was measured while the sample was under water. Water pressure may have caused the sample windows to adhere more closely to the tissue sample in the measurement path than in air. Therefore, the real sample thickness for the time-shift measurement was smaller than the sample thickness measured using a caliper. As a result, the term in the parentheses in the above equation should be smaller, and c_m should be larger, assuming that the Δt is positive. In addition the thickness measurement is more accurate using the technique described in this paper since the measurement is performed at the same site where the sound speed is measured.

Comparisons between the propagation speeds in tissue measured at 37 °C before and after the samples heated to the target temperatures are shown in Fig. 3. These data evaluate whether tissue coagulation introduces irreversible changes in the propagation speed after the tissue is returned to body temperature. Observe from the figure that all the speeds of sound in all samples before and after heating lie between 1590 and 1600 m/s. No statistically significant differences are observed in the propagation speed at 37 °C measured before vs after tissue coagulation. These results demonstrate that the changes observed in the propagation speed are entirely due to the elevated temperature in tissue.

Attenuation coefficients (dB/cm) vs tissue temperature for three ultrasound frequencies are shown in Fig. 4. The attenuation coefficients vary within a 1-dB/cm range over the temperatures spanned and reach a minimum at a temperature of around 50 to 60 °C. Data for each frequency show the same trend with heating, while variations between frequen-

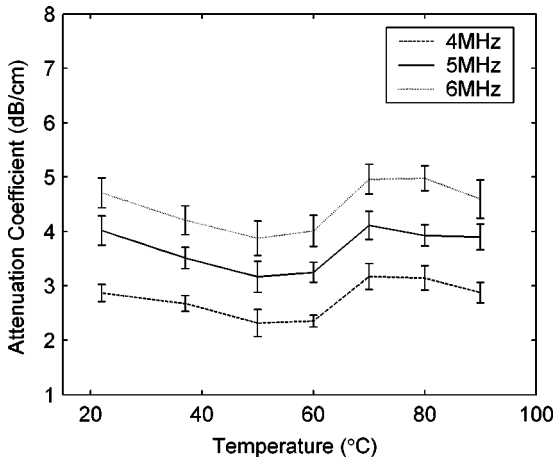

FIG. 4. Attenuation coefficients in canine liver tissue measured at different temperatures for center frequencies of 4, 5, and 6 MHz.

cies are consistent with attenuation being proportional to frequency. The attenuation coefficients obtained at 5 MHz are compared to the results obtained using the previous experimental protocol[22] in Fig. 5. These values are also provided in Table II. Note that the attenuation coefficient curves in both experiments are in the range of 3 to 4 dB/cm, and that the errorbars overlap. These results again demonstrate that attenuation coefficients derived using the two experimental protocols are nearly identical, and any differences between these and our previous results are not statistically significant.

However, the plots in Fig. 6 comparing the attenuation coefficient at 5 MHz obtained at 37 °C for the same tissue sample before and after temperature elevation indicates that there is an increase in the attenuation coefficient of the cooled tissue following coagulation. The difference between pre- and post-heating attenuation coefficients is greater for the tissues elevated to 80 °C and 90 °C (S4 and S5) than for the tissues whose target temperatures were lower (S1, S2, and S3). The third bar in each group in Fig. 6 represents the difference between the first and second measurements. The attenuation coefficient after heating increases almost linearly from 3 to 6 dB/cm when the target temperature increases

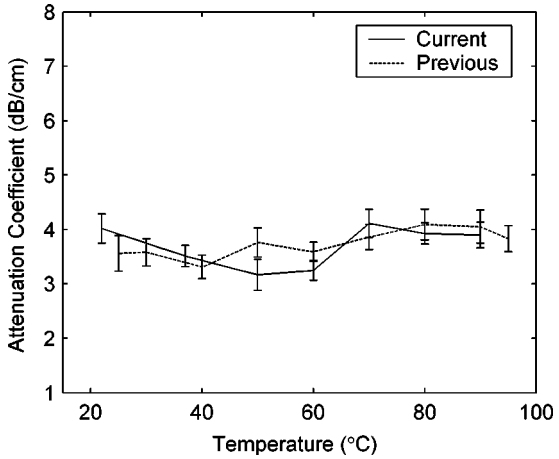

FIG. 5. Plots of the attenuation coefficients at 5-MHz frequency in canine liver tissue measured at different temperatures compared to the previous results reported in Techavipoo *et al.* (Ref. 22). Note that error bars are plus and minus one standard error across 10 different animals.

TABLE II. A comparison between the current and previous measurements of the attenuation coefficients at 5 MHz in canine liver tissue at different temperatures.

	Current attenuation (dB/cm)		Previous attenuation (dB/cm)	
Temperature (°C)	Mean	Standard error	Mean	Standard error
22	4.01	0.269	-	-
25	-	-	3.56	0.326
30	-	-	3.58	0.250
37	3.51	0.195	-	-
40	-	-	3.31	0.215
50	3.17	0.285	3.76	0.269
60	3.25	0.185	3.59	0.177
70	4.11	0.258	3.86	0.231
80	3.93	0.194	4.09	0.285
90	3.90	0.237	4.05	0.304
95	-	-	3.83	0.237

from 50 to 90 °C. These results depict that an irreversible change occurs, likely caused by tissue coagulation that occurs with increasing temperature. Another interesting facet that can be gleaned from comparing Figs. 5 and 6 is that the large increase in the attenuation coefficient is not present while the tissues are still at the elevated temperature.

Figure 7 illustrates this further, comparing the attenuation coefficient for each temperature group measured at 37 °C before heating to the attenuation at the elevated temperature. Again, the third bar in each group of three represents the difference between the first and second measurements, denoting the contributions of both the temperature elevation and tissue coagulation effects. Finally we attempt to decouple the individual contributions of tissue coagulation and temperature elevation in Fig. 8. Shown for each temperature group are differences in 37 °C attenuation before and after heating (B–A); differences between attenuation results when measured at the target temperature and when measured at 37 °C (C–A); and differences between attenuation results

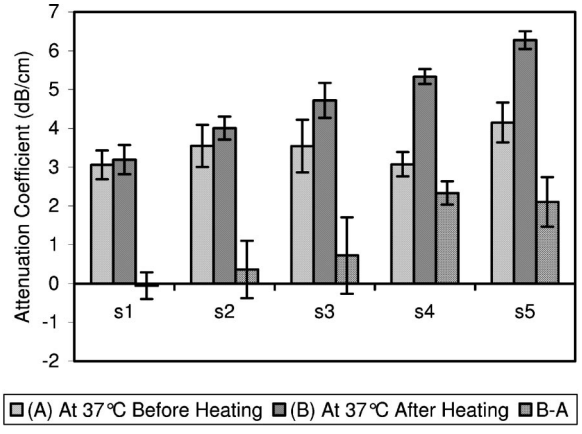

FIG. 6. Bar plots comparing the attenuation coefficients in tissue measured at 37 °C before and after the tissue samples were heated to the elevated target temperatures. The third bar-plot in each set represents the difference between the first and second measurements (B–A), demonstrating the increased attenuation due to tissue coagulation. Tissue sample S1 was raised to a single target temperature of 50 °C, S2 to 60 °C, S3 to 70 °C, S4 to 80 °C, and S5 to 90 °C, respectively.

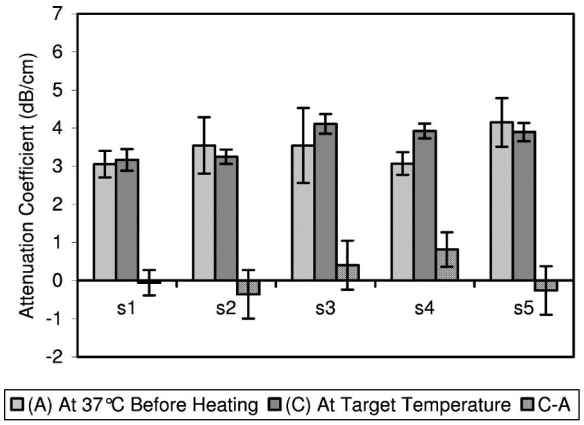

FIG. 7. Bar plots comparing the attenuation coefficients in tissue measured at 37 °C before heating and at the elevated target temperature. The third bar plot in each set represents the difference between the first and second measurements (C–A), demonstrating the increased attenuation due to both tissue coagulation and temperature elevation. Tissue sample S1 was raised to a single target temperature of 50 °C, S2 to 60 °C, S3 to 70 °C, S4 to 80 °C, and S5 to 90 °C, respectively.

when measured at the target temperature and when measured after heating but at 37 °C (C–B). Some of the larger differences in the (C–B) results in Fig. 8 is observed for the 80 °C and 90 °C temperatures (where the errorbars do not overlap), pointing out that temperature elevation itself introduces changes in the attenuation coefficient that negates the increase in the attenuation coefficient caused by tissue coagulation.

IV. DISCUSSION AND CONCLUSIONS

The propagation speed and attenuation coefficient in canine liver tissue measured in this paper agree with our previous results (Techavipoo *et al.*[22]). This implies that continuously heating and measuring properties for the same tissue samples over the entire temperature range produces similar results for both attenuation and propagation speed as the results from applying a single target temperature to each specimen. The only other difference between the measurement procedures is the more accurate measurement of specimen thickness in this paper.

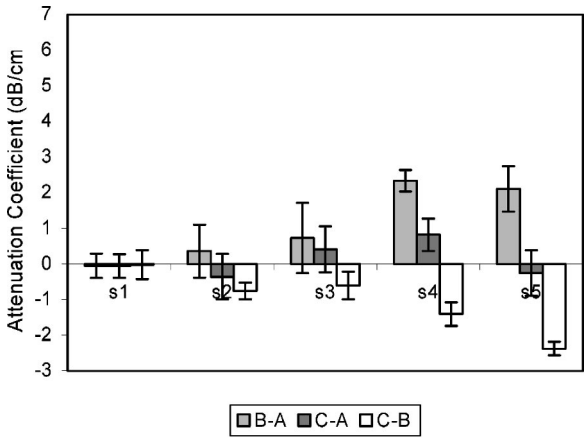

FIG. 8. Bar plots comparing the components of the attenuation coefficient due to tissue coagulation (B–A) both tissue coagulation and temperature elevation (C–A) and the component due to temperature elevation only (C–B). Note that the variation in the attenuation coefficient due to temperature elevation is opposite to that die to tissue coagulation.

In addition, we also demonstrate that no statistically significant changes in propagation speed estimates are observed before and after temperature elevation on the same tissue specimens measured at 37 °C. The higher temperatures reached almost certainly lead to tissue coagulation. The results indicate that variations in propagation speed with temperature are not a function of tissue coagulation effects that are induced with temperature elevation. Thus, it appears that tissue denaturation does not have an appreciable impact on the propagation speed. Therefore, the resultant shape of the propagation speed curve depends primarily on the temperature elevation, thereby making it an ideal parameter to measure temperature elevation in tissue.

On the other hand, the attenuation coefficient depends on both tissue coagulation and temperature elevation. By measuring the attenuations at 37 °C before and after heating, the tissue coagulation effect could be observed. The attenuation coefficient increases with the temperature that was used to heat the tissue. An increase in the temperature produces a concomitant increase in tissue coagulation and a subsequent increase in the attenuation coefficient upon cooling. In addition, temperature elevation and coagulation appear to have opposite effects on attenuation, thereby accounting for relatively unchanged values for the attenuation coefficients measured at the elevated temperatures. These results suggest that attenuation coefficients are probably not suitable for monitoring tissue temperature. However, the quantifiable and statistically significant increases in the attenuation obtained after heating (to a level that induces coagulation) followed by cooling of the tissue clearly demonstrate the potential of using the local maps of attenuation coefficients to demarcate the ablated regions. It also shows the possibility of ascertaining the degree of thermal damage by the change or the increase of the attenuation coefficient.

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