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A DISPERSIVE MODEL FOR THE PROPAGATION OF ULTRASOUND IN SOFT TISSUE

K. V. Gurumurthy[1] and R. Martin Arthur[2]

Biomedical Computer Laboratory and
Department of Electrical Engineering
Washington University
St. Louis, MO 63130

Although the dispersion of tissue is small and difficult to measure, it can be calculated from a knowledge of the tissue's attenuation. A minimum-phase function, which characterizes tissue dispersion was derived using the Hilbert transform. This function was incorporated into a tissue model which has a causal impulse response and from which accurate estimates of the slope of attenuation times path length can be extracted. Predictions of phase velocity closely match available dispersion measurements. The model suggests that phase velocity measurements must be much more accurate than attenuation measurements for a comparable description of tissue.

Key words: Hilbert transform relations; impulse response; Kramers-Kronig relations; linear- and minimum-phase systems; phase velocity; slope of attenuation; tissue characterization.

INTRODUCTION

Ultrasonic tissue characterization requires localized measurement of ultrasonic parameters which can discriminate among different tissue types and their pathological states. Measurements of both transmitted and reflected ultrasound contain information concerning the status of localized tissue, such as specific acoustic impedance, attenuation coefficient, propagation velocity, scattering parameters, density, and bulk modulus ⟨1,2⟩.

Correlation of changes in these ultrasonic properties with tissue pathology may yield indices of diagnostic significance. For example, in in vitro studies Calderon and coworkers ⟨3⟩ found that 1 cm of malignant tumor, benign tumor, and normal tissue presented 19.5, 9.0 and 2.3 dB of attenuation at 2.25 MHz, respectively. Lele and coworkers ⟨4⟩ reported that the slope of attenuation-versus-frequency curves was different for different tissues, and that the slope for necrotic tissue was markedly steeper than that of healthy tissue of the same type. O'Donnell and coworkers ⟨5⟩ observed a correlation between the change in attenuation with frequency and the collagen content of dog myocardium. Furthermore, they observed changes in the slope with time after the onset of ischemia. In in vivo studies Kuc ⟨6⟩ observed that inflamed livers exhibited lower than normal attenuation, whereas in cirrhotic livers attenuation was higher than normal.

Many problems remain to be solved, however, before noninvasive quantification of ultrasonic tissue properties can be performed routinely.

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[1] Present address: RCA New Products Laboratory, Indianapolis, IN 46201.

[2] Author to whom correspondence should be addressed.

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We believe that an appropriate tissue model is basic to an understanding of the effects of inhomogeneities and dispersion on quantitative measurements. An accurate description of a single layer of soft tissue is essential 1) to allow accurate calculation of tissue properties based on signal features, and 2) to account for the effects of intervening layers of tissue on estimates of the properties of tissue regions remote from the transducer. Precise simulations of tissue effects may provide a step in the development of the next generation of imaging systems, which may be able to determine acoustic properties of specific tissue regions, in addition to the usual mapping of tissue interfaces, during pulse-echo imaging.

If soft tissue responds linearly to ultrasound, then its behavior is fully described by either its impulse response in the time domain or its frequency response in the frequency domain. Of these descriptors only the magnitude of the frequency response of soft tissue is well known; the phase of the frequency response is not ⟨7,8⟩. Limited experimental evidence shows that tissue is dispersive, but that the dispersion is small. It is on the order of 0.7 percent per frequency decade for an attenuation of 1 dB/cm/MHz ⟨7⟩. Over the frequency range from 1-10 MHz, the phase of the frequency response is usually assumed to be linear ⟨8⟩. Unfortunately, if one makes the assumption of exponential attenuation in a dispersionless medium (constant phase velocity), the impulse response is non-causal.

Certainly one would expect tissue response, or for that matter the response of any passive, memoryless, real-world, physical system to be causal. It is not in this case because the assumption of constant phase velocity is not valid. Our objective then was to synthesize the frequency response of a single layer of tissue from a knowledge of the magnitude of its response under the causality constraint and to use the resultant model to predict tissue behavior in both the time and frequency domains. In particular we 1) predict the impulse response of soft tissue, 2) develop relations for estimating the product of the slope of attenuation and path length from the impulse response, 3) compare predicted and measured phase velocities, and 4) determine error limits which must be achieved to obtain meaningful measurements of phase velocity.

TISSUE MODELS

Tissue is almost always assumed to respond linearly to diagnostic levels of ultrasound. This assumption appears to be valid because the signal levels used in diagnostic systems are small. The intensity of diagnostic ultrasound, which may have a frequency content ranging from 1 to 20 MHz, varies from a few milliwatts/cm² for continuous-wave Doppler methods to a maximum of about 1000 watts/cm² for pulse-echo methods ⟨9⟩. Pulse-echo methods typically employ 1 μs pulses with pulse repetition frequencies up to 4000 Hz. With these parameters, diagnostic systems operate at average energy levels several orders of magnitude below those which produce structural changes ⟨9⟩. Thus, although nonlinear effects have been reported ⟨10⟩, for a wide range of clinical applications tissue responds linearly to ultrasound. In addition the nature of that response is not likely to change rapidly because during the 0.1 ms that it takes ultrasound to propagate through about 15 cm of tissue, the fastest moving tissues, the heart valves, move less than 0.1 mm ⟨11⟩. Thus neither a change in tissue property nor a movement of a different type of tissue into an ultrasonic beam during one pulse period is likely. Therefore tissue may be thought of as a linear, time-invariant system.

Any linear system is characterized by its frequency response. For an ultrasonic tissue model that response is the relation between the ultrasonic field after it has passed through a tissue specimen and the excitation into that specimen as a function of frequency. The logarithm of

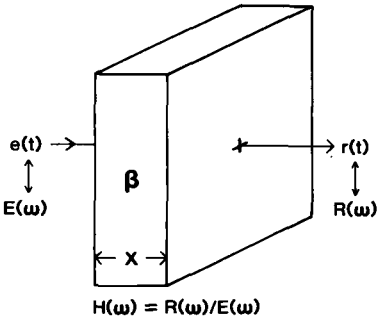

Fig. 1 Ultrasonic transmission through a uniform, homogeneous layer. E(ω) and R(ω) are the Fourier transforms of the excitation e(t) and the response r(t), respectively. For unity transmittance at each surface the magnitude of the frequency response H(ω) of the layer is a function of its thickness x and its slope of attenuation β.

the magnitude of the frequency response is usually assumed to be a linear function of frequency for a given path length. Indeed there are a number of experiments which support a power-law dependence of attenuation on frequency where the exponent of the frequency term is not significantly different from unity; the constant of proportionality is termed the slope of attenuation ⟨4,7,12-14⟩.

Given a power-law response with linear dependence of attenuation on frequency, a layer of tissue may be characterized by its slope of attenuation versus frequency β and its thickness x as shown in figure 1. If the transmittance at both surfaces of the tissue layer is unity, the magnitude of the frequency response for this configuration is usually written ⟨8,15⟩,

$$|H(\omega)| = e^{-\beta x \frac{|\omega|}{2\pi}} \quad . \tag{1}$$

This well-documented expression is the basis for existing tissue models and for the dispersive model that we propose in this paper.

LINEAR-PHASE MODEL

The phase velocity of tissue does not change much with frequency, so that to a first approximation it is a constant ⟨7,16⟩. If the phase velocity Vp is assumed to be a constant, then the phase response is a linear function of frequency,

$$\theta(\omega) = \omega \tau_b x \quad , \tag{2}$$

where τ_b is the bulk propagation delay per unit length. Combining the magnitude of the frequency response with the linear-phase term, the frequency response is

$$H(\omega) = e^{-\beta x \frac{|\omega|}{2\pi}} e^{-j\omega\tau_b x} \quad . \tag{3}$$

This expression is the response assumed by Kak and Dines ⟨8⟩. Although Eq. (3) is a good first order approximation to the transfer function of tissue, this model's impulse response is non-causal because phase velocity in not really constant. Even though tissue dispersion is small, a small change in the phase function can have a significant effect on the impulse response.

DISPERSIVE TISSUE MODEL

Because the dispersion of tissue is small, it is very difficult to measure <7>. To our knowledge, only the measurements of dispersion in hemoglobin solutions by Carstensen and Schwan <16> and in dog myocardium by Bhagat and coworkers <13> have been reported. Thus because of very limited experimental evidence, one can assume that only the magnitude of the frequency response is known. Our task then was to synthesize the frequency response of a layer of tissue from a knowledge of the magnitude of its response alone.

It is well known that for a bounded, stable, minimum-phase system, the real and imaginary parts of its transfer function are related by the Hilbert transform <17,18>. If the magnitude of the frequency response is square integrable and satisfies the Paley-Weiner condition, then a suitable imaginary term can be found, so that the system is causal. According to the Paley-Weiner condition the magnitude of the response must not go to zero faster than an exponential. To meet this condition we imposed a high-frequency limit on the magnitude of the response of tissue, i.e., on Eq. (1). (See Appendix A.)

The Hilbert transform gives the phase for a minimum-phase system, but any non-minimum phase system can be described by the product of an all-pass function and a minimum-phase sytem <18>. We assumed that the phase of the all-pass function of our tissue model had the same form as the linear-phase model. Thus as shown in Appendix A, the total phase for our tissue model consists of a linear-phase term plus the minimum-phase relation given by the Hilbert transform:

$$\theta(\omega) = \omega\tau x - \frac{\omega\beta x}{\pi^2} \ln(\omega) \tag{4}$$

$$\tau = \tau_b + \frac{\beta}{\pi^2} \tau_m \quad ,$$

where τ_b is the bulk delay and τ_m is the miminum-phase delay factor. The frequency response for the resultant causal system is

$$H(\omega) = e^{-\beta x \frac{|\omega|}{2\pi}} e^{-j\omega\tau x} e^{j\frac{\omega\beta x}{\pi^2} \ln(\omega)} \quad . \tag{5}$$

The frequency response differs from that of the linear-phase model by the presence of the final exponential term, whose argument is logarithmically dependent on frequency. The inclusion of this dispersive term makes the system causal. The effects of this term on the phase velocity are examined after we point out the marked differences between the linear-phase and Hilbert-dispersive models in the time domain.

TIME-DOMAIN RESPONSE OF SOFT TISSUE

The response of a linear system to any excitation is the convolution of that excitation and the impulse response of the system, which is dependent only on system parameters. Proper processing of appropriate features of a system's impulse response allows determination of those system parameters, which in this case are the ultrasonic properties of tissue.

IMPULSE RESPONSE

Once the frequency response of a tissue model is established, its impulse response can be found by either analytically or numerically

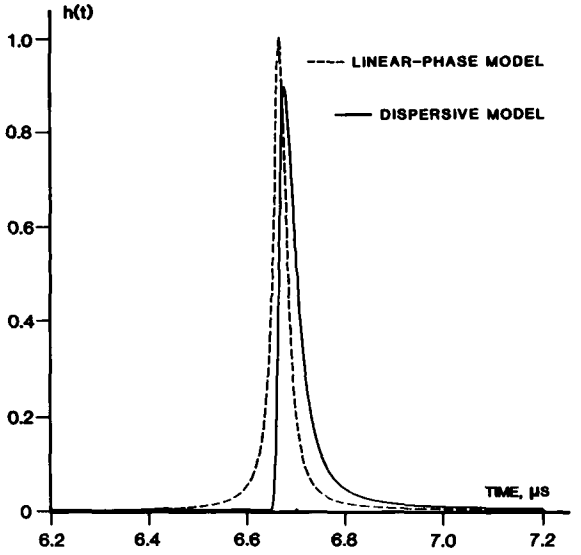

Fig. 2 Impulse response of both the linear-phase and Hilbert dispersive models. Slope of attenuation β was set at 0.1 /cm/MHz; path length was 1 cm. The bulk delay τ_b was 6.67 μs/cm; the minimum-phase delay factor was 20. Amplitudes were normalized to the peak of the linear-phase response.

calculating the inverse Fourier transform of the frequency response. In this section we present and compare the impulse responses of the linear-phase and Hilbert dispersive models.

Linear-phase Model

The impulse response of the linear-phase model (Eq. (3)) with slope of attenuation β and thickness x was derived by Kak and Dines ⟨8⟩. They showed that

$$h(t) = \frac{1}{\pi} \frac{\xi}{\xi^2 + (t - \tau_b x)^2} \tag{6}$$

$$\xi = \frac{\beta x}{2\pi} \quad .$$

This result is plotted in figure 2 for a slope of attenuation β of 0.1 /cm/MHz and a phase velocity of 1500 m/s over a path length x of 1 cm. As expected for a linear-phase system, the impulse respone is symmetric. Unfortunately, it is also non-causal. It takes 6.67 μs for sound to travel 1 cm at a velocity of 1500 m/s. Note that the peak of the impulse response occurs at this time. Thus half the energy in h(t) appears before any output should exist. This contradiction occurs because the assumption of constant phase velocity is not valid.

Hilbert Dispersive Model

The impulse response for the Hilbert dispersive model was obtained by numerically calculating the inverse Fourier transform of its frequency response (Eq. (5)). It is also shown in figure 2. The time between samples of the calculated response was 2 ns. The response is causal; the lack of symmetry in its shape implies that dispersion is associated with

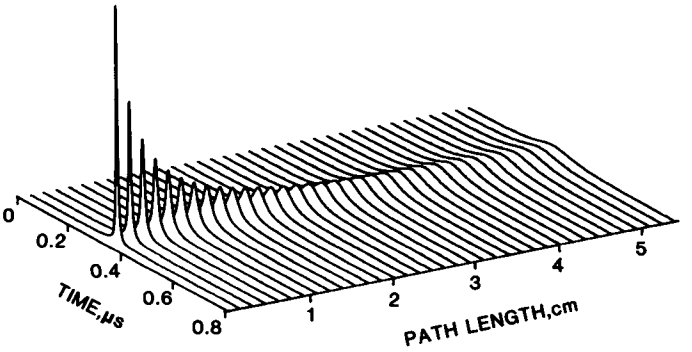

Fig. 3 Temporal and spatial characteristics of the impulse response of the Hilbert dispersive model. The slope of attenuation was set at 0.1 /cm/MHz. Peaks of the responses for tissues whose thicknesses differed by about 15 mm were aligned within a 1 μs window to form the response surface.

the system it represents. The peak is lower than that of the linear-phase model, but its duration is longer. These differences in shape occur because the frequency components of the propagating signal travel at difference velocities in the dispersive model. Higher frequencies travel slightly faster than lower ones.

The propagation delay for the dispersive model is given by Eq. (4) and contains the same bulk delay (6.67 μs/cm) as the linear-phase model plus a term dependent upon β. The value for τ_m, the minimum-phase delay factor was taken to be 20. It was found by comparing the minimum-phase term of the Hilbert model with that of a single-pole model for the same tissue ⟨19⟩. This value for τ_m made the minimum-phase terms for the two dispersive models agree for a wide range of values of the slope of attenuation. Here the effect is that the peak of the non-causal, linear-phase model response and the rising edge of the Hilbert model response approximately coincide. The peak of the response for the dispersive system is at about 6.65 μs, which corresponds to a wavefront velocity of 1504 m/s. A similar result would have been obtained if τ_m had been assigned so that the wavefront arrived at a time consistent with the propagation delay of the high frequencies in the response.

The temporal and spatial characteristics of the impulse response of the Hilbert dispersive model may be better appreciated in figure 3, where the response is shown as it propagates through increasing tissue thickness. At each thickness the rising edge changes more rapidly than the falling edge. Clearly the width of the response increases as path length increases.

ESTIMATION OF THE ATTENUATION COEFFICIENT

Kak and Dines ⟨8⟩ proposed a tissue characterization scheme based on the impulse response of soft tissue. Their tissue model was the linear-phase system whose frequency response is given by Eq. (3) and whose impulse response is given by Eq. (6). They derived estimates for the product of the slope of attenuation β and the path length x from both the root-mean-square (RMS) duration and the peak value of the impulse response. Here we show that because the assumption of constant phase velocity in the linear-phase model is inconsistent with the form of the impulse response,

estimates based on that response are in error. Those errors are quantified in this section which also gives the proper estimates of βx from both the RMS duration and the peak value of the causal impulse response of the Hilbert dispersive model.

Energy

The energy of the impulse response can be calculated using Parseval's Theorem

$$E = \frac{1}{2\pi}\int_{-\infty}^{\infty} |H(\omega)|^2 \, d\omega = \int_{-\infty}^{\infty} |h(t)|^2 \, dt \quad . \tag{7}$$

Using the magnitude of the frequency response assumed for both the linear-phase and Hilbert dispersive models (Eq. (1)), the energy is

$$E = 1/\beta x \quad . \tag{8}$$

Numerical integration of the square of the Hilbert impulse response agreed with the energy determined by the magnitude of the frequency response (Eq. (8)) within 0.4 percent. The major source of this small error was missing the peak of the response with the time quantization employed (2 ns).

Root-Mean-Square Duration

RMS duration D is a measure of the length of time that a signal persists. When applied to the impulse response, it may be thought of as an approximation to its 3 dB width. It is defined by Kak and Dines <8> as

$$D^2 = \frac{\int_{-\infty}^{\infty} (t-t_c)^2 |h(t)|^2 \, dt}{\int_{-\infty}^{\infty} |h(t)|^2 \, dt} \quad , \tag{9}$$

where the mean time t_c is

$$t_c = \frac{\int_{-\infty}^{\infty} t|h(t)|^2 \, dt}{\int_{-\infty}^{\infty} |h(t)|^2 \, dt} \quad . \tag{10}$$

For the linear-phase model Kak and Dines <8> evaluated the RMS duration and showed it to be

$$D_{LPM} = \beta x/2\pi \quad . \tag{11}$$

This linear relation in βx, the slope of attenuation times path length, is plotted in figure 4, along with the numerical evaluation of RMS duration for the Hilbert dispersive model. Although both models give straight-line relationships passing through the origin for RMS duration versus path length, the slopes differ. The duration for the Hilbert model

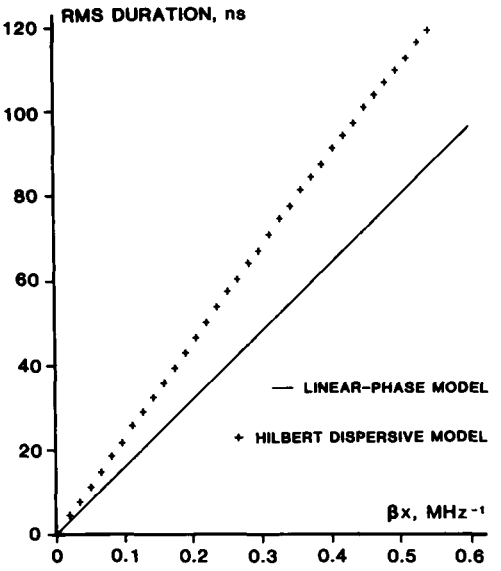

Fig. 4 RMS duration of the impulse response as a function of βx, the slope of attenuation times path length. The crosses were calculated from the data plotted in figure 3.

is always greater than that for the linear-phase model. A first order polynomial fit of the duration predicted by the Hilbert model yields

$$D_{HDM} \simeq 0.21\ \beta x \quad . \tag{12}$$

Recently Pohlig ⟨20⟩ has shown that the square of the RMS duration of a signal can be separated into the sum of two components, one the square of the duration of the zero-phase (magnitude only) signal, and the other the variance of a weighted phase derivative. Furthermore, he showed that for a given spectral magnitude, the signal with the shortest duration will have linear phase. Therefore, the observation that the RMS duration predicted by the Hilbert model is longer than that of the linear-phase model is to be expected.

More importantly, the expression for the RMS duration from the Hilbert model correctly predicts the product βx, whereas the estimate given by the linear-phase model is too high. The error in the linear-phase model estimate is found by comparing the two expressions for the RMS duration. The duration of the non-causal, linear-phase model yields an estimate of βx which is 2.3 dB too high.

Peak Value of the Impulse Response

The product of the slope of attenuation and path length βx can also be estimated from the peak of the impulse response. For the linear-phase model the peak of its non-causal impulse response as shown by Kak and Dines ⟨8⟩ is

$$h_{LPM}(t_c) \simeq 2/\beta x \quad . \tag{13}$$

The peak of the impulse response versus βx for both the linear-phase and Hilbert models is plotted in figure 5. The peaks of the causal Hilbert response are taken from the data used to plot figure 3. Both curves are

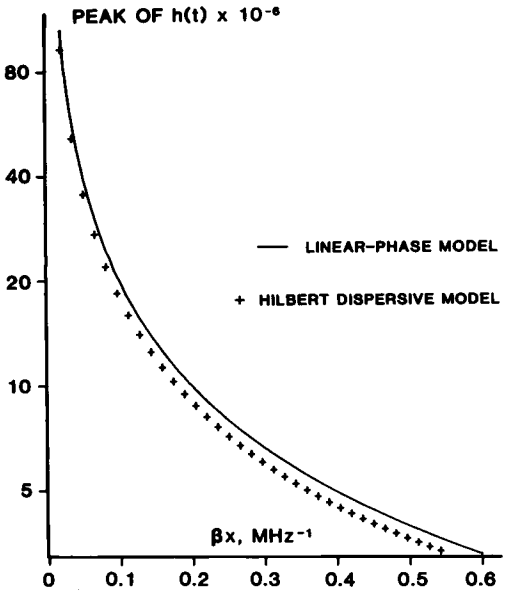

Fig. 5 Peak value of the impulse response as a function of βx, the slope of attenuation times path length. The crosses were calculated from the data plotted in figure 3.

similar in shape. If we assume an inverse relation between peak value and the product of the slope of attenuation and path length for the causal Hilbert model, then the constant of proportionality is 1.75. Thus

$$h_{HDM}(t_c) = 1.75/\beta x \quad . \tag{14}$$

If the peak value of the Hilbert response is indeed inversely proportional to βx, then the difference in the logarithm of its peak response (Eq. (13)) and that of the linear-phase model (Eq. (14)) will be constant. The difference in logarithms is

$$\ln h_{LPM}(t_c) - \ln h_{HDM}(t_c) = 0.134 \quad . \tag{15}$$

Peak values of the two models are plotted on a logarithmic scale in figure 5. The difference is almost constant. Thus the assumption of an inverse relation for both models appears to be justified.

Although the functional relation between peak value and βx appears to be the same for both models, the constant of proportionality is not the same. There is a 1.2 dB difference. The prediction by the Hilbert dispersive model is correct, so that the linear-phase model's estimate of βx based on peak value is 1.2 dB too high.

Thus for both RMS duration and peak value estimates of the slope of attenuation times path length, the linear-phase model is in error whereas the Hilbert dispersive model correctly predicts the βx product in the time domain. Table 1 summarizes the time-domain estimates given by both models along with the error in the non-causal, linear-phase model predictions.

Table 1. Determination of βx the slope of attenuation times path length from the impulse response of soft tissue.

	Linear-phase (non causal) model	Dispersive (causal) model	Difference dB
Energy	$1/\beta x$	$1/\beta x$	0
RMS duration	$\beta x/2\pi$	$\beta x/4.8$	2.3
Peak value	$2/\beta x$	$1.75/\beta x$	1.2

PHASE VELOCITY OF SOFT TISSUE

In general phase velocity $V_p(\omega)$ and the phase $\theta(\omega)$ of the frequency response are reciprocally related:

$$V_p(\omega) = \frac{\omega}{\theta(\omega)/x} \quad , \tag{16}$$

where ω is the angular frequency and x is the path length. As we showed in Eq. (4), the expression for the phase per unit length from the Hilbert dispersive model is

$$\frac{\theta(\omega)}{x} = \omega \left[\tau - \frac{\beta}{\pi^2} \ln(\omega) \right] \quad , \tag{17}$$

where the term in the square brackets is the propagation delay per unit length. The propagation delay contains a constant plus a frequency-dependent term. The amount of dispersion is controlled by the frequency-dependent term, which says that the change in either phase or phase velocity should be logarithmically dependent on frequency. Nevertheless, because the dispersion of soft tissue is small, i.e., the phase velocity is almost constant, phase should be a nearly linear function of frequency. The two components of the phase of the Hilbert dispersive model are plotted as functions of frequency in figure 6, which clearly shows that the linear-phase term dominates the dispersive term. Even the dispersive term which is dependent on the product of frequency and the logarithm of frequency is very nearly a linear function. Figure 6 emphasizes how small the dispersion in tissue is. Yet, as we have seen, this small effect is significant in determining time-domain behavior. Although not apparent here, logarithmic dependence on frequency is clearly seen in the plots of phase velocity which follow.

If we combine Eqs. (16) and (17), the phase velocity predicted by the Hilbert dispersive model can be expressed as

$$\frac{1}{V_p(\omega)} = \tau - \frac{\beta}{\pi^2} \ln(\omega) \quad . \tag{18}$$

Clearly, as ω goes to zero, so does Vp(ω) as given by Eq. (18). To complete the Hilbert dispersive model, we must specify the phase velocity at low frequency. If the medium were dispersionless, 1/Vp(ω) would be constant and equal to τ_b. If we assume in the dispersive case that Vp(ω) is fixed below some frequency and maintain consistency with the dispersionless case, then

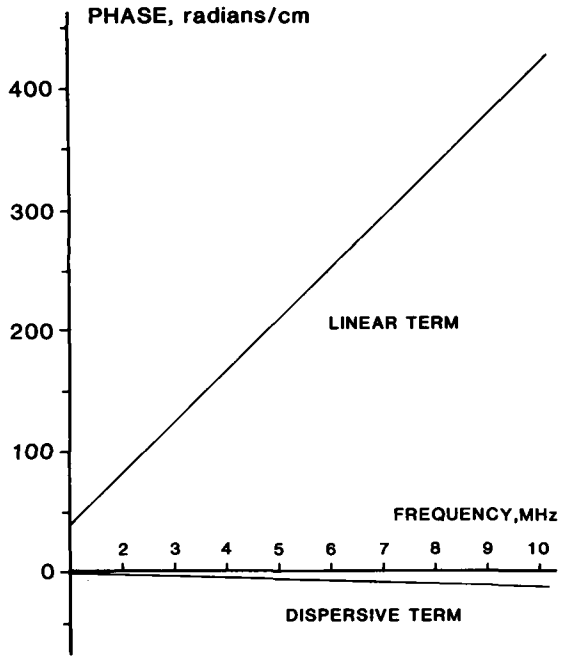

Fig. 6 Phase response of soft tissue predicted by the Hilbert dispersive model. The linear-phase term dominates the dispersive term which is dependent upon the frequency times the logarithm of frequency. Phase shown was predicted for a 1 cm layer of tissue with a slope of attenuation of 0.115 /cm/MHz (1 dB), a bulk delay of 6.67 μs/cm, and a minimum-phase delay factor of 20.

$$\frac{1}{V_p(0)} = \tau \tag{19}$$

and

$$\frac{1}{V_p(\omega)} = \frac{1}{V_p(0)} - \frac{\beta}{\pi^2} \ln(\omega) \qquad \omega \geq 1$$

$$= \frac{1}{V_p(0)} \quad . \qquad \omega < 1 \tag{20}$$

Because we are primarily interested in the 1-10 MHz range, $\omega = 1$ is essentially zero frequency. The choice of 1 rad/s at which to fix velocity is convenient because it simplifies Eq. (19) and makes the phase velocity at low frequency the same as the phase velocity of the linear-phase model. That choice, however, does not strongly influence the value of Vp(0). Vp(0) will vary by only ± 2 percent, if the frequency at which velocity is fixed varies from 1.9 μHz to 0.52 MHz for a bulk delay of 6.67 μs and a slope of attenuation of 0.1 /cm/MHz.

PHASE VELOCITY AT LOW FREQUENCY

The phase velocity at low frequency Vp(0) can be determined for the Hilbert dispersive model, if the phase velocity is known at any other single frequency ω_0. Rearranging Eq. (20) yields

Table 2. Acoustic parameters of hemoglobin solutions.

Measured (from Carstensen and Schwan, <16>)			Calculated from the Hilbert dispersive model	
Hemoglobin Concentration g/100cc	β Slope of Attenuation /cm/MHz	Phase Velocity at 2 MHz m/s	$V_p(0)$ m/s	τ_b Bulk Delay μs
8.0	0.0077	1522.9	1519.9	6.56
13.0	0.0128	1541.6	1536.6	6.48
19.2	0.0197	1562.8	1554.8	6.39
24.0	0.0257	1582.2	1571.6	6.31
30.0	0.0344	1608.3	1593.6	6.21

$$\frac{1}{V_p(0)} = \frac{1}{V_p(\omega_o)} + \frac{\beta}{\pi^2} \ln(\omega_o) \quad . \tag{21}$$

The Hilbert dispersive model is complete once the bulk delay per unit length τ_b is known. It is found from the expression for τ in Eq. (4) and from the definition of $V_p(0)$ in Eq. (19):

$$\tau_b = \frac{1}{V_p(0)} - \frac{\beta}{\pi^2} \tau_m \quad . \tag{22}$$

In order to determine whether or not Vp(0) from the Hilbert model was consistent with measurements that apply at low frequency, we analyzed the hemoglobin solutions <16> characterized in table 2 with our model. Vp(0) for these solutions increases linearly (r = 0.9991) with concentration, as shown in figure 7. The least-squares fit for $V_p(0)$ versus hemoglobin concentration is

$$V_p(0) = 1493 + 3.32 \left[Hg\right] \quad , \tag{23}$$

where $\left[Hg\right]$ is hemoglobin concentration. Vp(0) at zero hemoglobin concentration is about 1493 m/s. The experimental value for the phase velocity of distilled water at 25°C is about 1497 m/s <21>, a difference of only 0.2 percent. A straight-line fit of the phase velocity at 2 MHz versus hemoglobin concentration predicted the velocity in water to be 1491 m/s, a 0.4 percent error.

The least-squares fit of phase velocity versus hemoglobin concentration is consistent with the empirical relation between phase velocity at 1 MHz and tissue constituents reported by Goss and coworkers <22>. They showed that

$$V_p(1 \text{ MHz}) = V_o + 3.2 \left[P + 2.0\ C\right] \quad , \tag{24}$$

where P is the wet weight percentage of globular protein, C is the wet weight percentage of collagen present in the tissue, and V_o is the velocity which is observed when P and C are zero. Note the similarity in the slope for hemoglobin in Eq. (22) and that for globular protein in Eq. (23). A straight-line fit of the phase velocity of hemoglobin at 2 MHz versus concentration has a substantially different slope; it is 3.8.

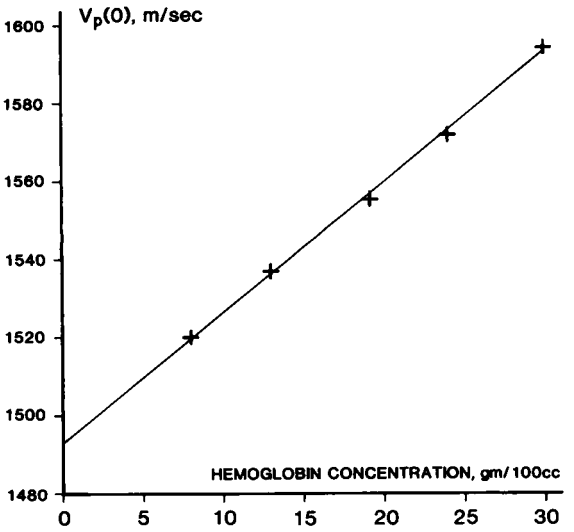

Fig. 7 Phase velocity at zero frequency versus hemoglobin concentration. The crosses were predicted by the Hilbert dispersive model; their values are given in table 2. The straight line is a least-squares fit of those points.

Thus the choice of $1/\tau$ as the low frequency value for the phase velocity, in addition to being a logical extension of the dispersionless case, is justified by its success in matching measured data. Moreover, the logarithmic factor in Eq. (21), the expression for $1/Vp(0)$, improves our model's representation of the phase velocity at low frequency over merely using values measured at 2 MHz to approximate low frequency behavior.

TISSUE DISPERSION

With the low frequency behavior of the Hilbert model determined, we are now in a position to examine phase velocity as a function of frequency. If the phase velocity $Vp(\omega)$ decreases with frequency, then the dispersion is 'normal'; if it increases with frequency the dispersion is 'anomalous' <23>. The behavior of tissue predicted by the Hilbert model may be found by differentiating the general expression for phase velocity (Eq. (16)) with respect to ω:

$$V_p'(\omega) = x \frac{\theta(\omega) - \omega\,\theta'(\omega)}{\theta^2(\omega)} \quad . \tag{25}$$

This derivative can be positive, negative, or zero:

1. $V_p'(\omega) = 0$ if the phase velocity is constant.

$$\frac{\theta(\omega)}{\omega} = \theta'(\omega) \tag{26}$$

2. $V_p'(\omega) > 0$ if the dispersion is anomalous.

$$\frac{\theta(\omega)}{\omega} > \theta'(\omega) \tag{27}$$

3. $V_p'(\omega) < 0$ if the dispersion is normal.

$$\frac{\theta(\omega)}{\omega} < \theta'(\omega) \tag{28}$$

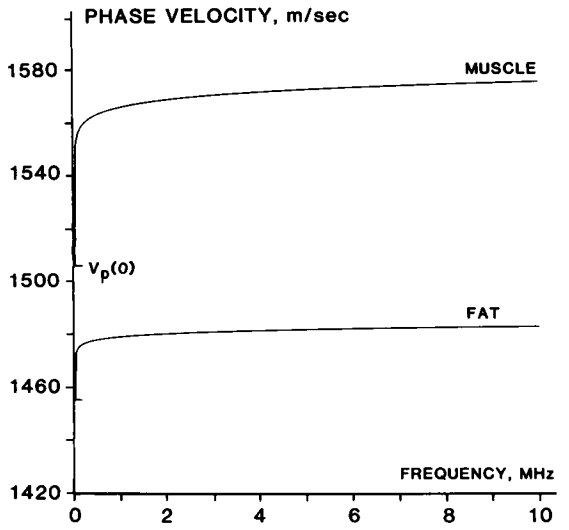

Fig. 8 Phase velocity of muscle and fat predicted by the Hilbert dispersive model. The slopes of attenuation of 0.161 /cm/MHz (1.4 dB) for muscle with insonification parallel to fibers and 0.069 /cm/MHz (0.6 dB) for fat were taken from the compilation of Goss and cowokers ⟨24⟩. The data also permitted calculation of the bulk delays, which were 6.32 μs/cm and 6.73 μs/cm for muscle and fat, respectively. The minimum-phase delay factor for both muscle and fat was 20.

For the phase given by the Hilbert dispersive model, the relationship between phase and its derivative is

$$\frac{\theta(\omega)}{\omega} = \theta'(\omega) + \frac{\beta}{\pi^2} \quad . \tag{29}$$

Because the slope of attenuation is always positive, the Hilbert model predicts that tissue dispersion will be anomalous. This prediction agrees with the limited measurements of tissue dispersion in existence ⟨13,16⟩.

PHASE VELOCITY PREDICTED BY THE HILBERT DISPERSIVE MODEL

According to the Hilbert dispersive model phase velocity can be uniquely specified at all frequencies from a knowledge of the slope of attenuation alone if the phase velocity is known at just one frequency. The phase velocity at one frequency is used to fix the phase velocity at low frequency Vp(0) and thereby the bulk delay per unit length τ_b of the Hilbert model. Phase velocity predicted by the Hilbert dispersive model for muscle and for fat is plotted in figure 8. These curves were generated with the Hilbert model from slopes of attenuation compiled by Goss and coworkers ⟨24⟩. These values are listed in table 3. The phase velocity at 1 MHz was used to calculate $V_p(0)$ and the bulk delay τ_b.

Similar calculations were carried out using measurements of hemoglobin solutions at 25°C made by Carstensen and Schwan ⟨16⟩. Table 2 shows the numerical values used in finding the phase velocity as a function of frequency for 5 hemoglobin concentrations. As shown in figure 9 phase velocities cover almost a 100 m/s range. The dispersion, however, is small, so that changes on the order of 1 m/s are difficult to see on that scale. Figure 10 shows the dispersion for a 30g/100cc concentration on an expanded scale. The predicted dispersion covers a 2.1 m/s range. Also

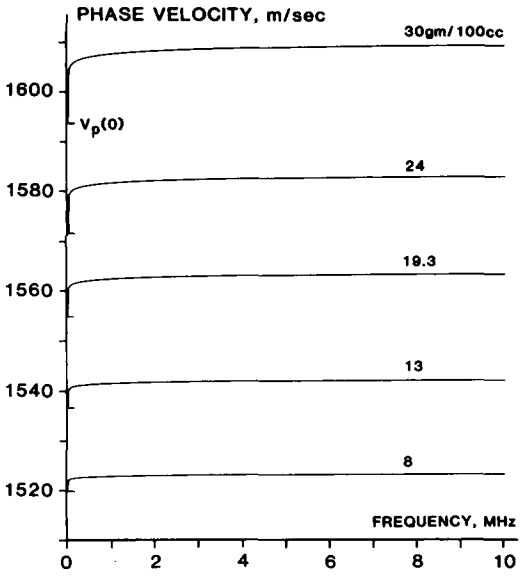

Fig. 9 Phase velocity of hemoglobin solutions predicted by the Hilbert dispersive model. Curves were calculated from measurements of attenuation reported by Carstensen and Schwan <16>. The values of the slope of attenuation β and bulk delay τ_b used for each concentration are given in table 2.

shown in figure 10 are the velocities measured by Carstensen and Schwan at 5 frequencies. The small discrepancy at high frequencies may be due to the assumption of linear variation of attenuation with frequency.

Recently, propagation velocity and attenuation measurements have been reported by Bhagat and coworkers <13> for liver and kidney tissue of Sprague-Dawley rats and for dog myocardium. Their results indicate that the propagation velocity for the above soft tissues lies in the range of 1530-1580 m/s and that the dispersion in the frequency range 1-10 MHz is about 1.5 percent for dog myocardium. They also found that attenuation was

Table 3. Change in phase velocity.

	Measured parameters			ΔV calculated from the Hilbert dispersive model		
	Slope β		V_p at 1 MHz	$\Delta V(\omega)$[1] 1-10 MHz	$\Delta V_\beta(\omega)$[2] at 10 MHz	$\frac{\Delta V_\beta(\omega)}{Vp}$
	dB	/cm/MHz	m/s	m/s	m/s	percent
Hemoglobin <16>						
8g/100cc	0.07	0.0081	1523	0.44	0.044	0.003
30g/100cc	0.3	0.035	1608	2.1	0.21	0.013
Fat <24>	0.6	0.069	1479	3.6	0.36	0.024
Muscle <24>	1.4	0.16	1566	9.2	0.92	0.059
Cartilage <24>	5.0	0.58	1665	86.	8.6	0.520

[1] Change over the 1-10 MHz range

[2] Change for a 10 percent change in β

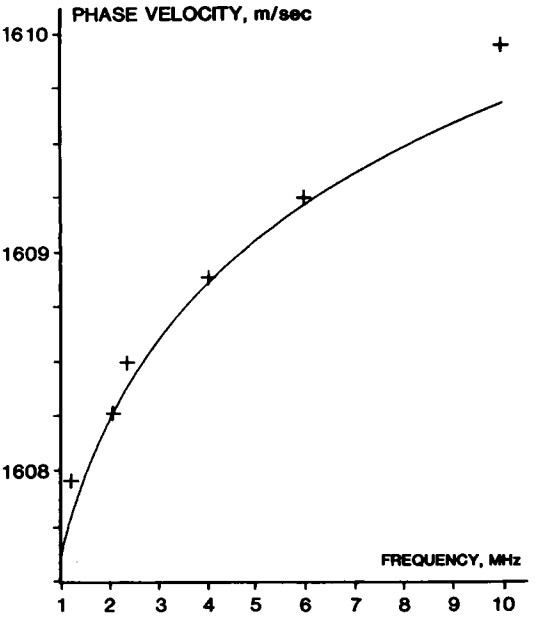

Fig. 10 Phase velocity of a 30g/100cc solution of hemoglobin predicted by the Hilbert dispersive model. This plot gives an expanded view of the top curve in figure 9, along with the phase velocities measured by Carstensen and Schwan ⟨16⟩. Measured phase velocities were not used to generate the predicted curve. The bulk delay τ_b, however, was determined from the phase velocity at 2 MHz, so that the predicted curve goes through that data point. As shown in table 2 the slope of attenuation β was 0.034 /cm/MHz; the bulk delay τ_b was 6.56 μs/cm. The minimum-phase delay factor was 20.

a nearly linear function of frequency in the 1–10 MHz range and that the slope of the least-squares fit line was differernt for different tissue types. We used the slope of the least-squares fit of their attenuation-versus-frequency curves to find the slope of attenuation. We calculated β of the left venticular muscle of dog to be 0.154 /cm/MHz. The phase velocity of 1570 m/s measured by Bhagat and coworkers at 5 MHz gave a bulk delay τ_b equal to 6.33 μs/cm. The phase velocity found from these values of β and τ_b using the Hilbert model is plotted in figure 11 along with the experimental data obtained by Bhagat and coworkers ⟨13⟩. The disperson predicted by the Hilbert model is well within the experimental error. It should be noted, however, that Bhagat and coworkers used the pulse-transit-time method to find the phase velocity, so that their measurements signals had a 20 percent bandwidth, i.e., they were not single frequencies. Thus the error in their estimates of phase velocity may be even greater than the error bars they showed.

Even with the error bars shown in figure 11, β could vary considerably and the predicted dispersion would fall within experimental limits. For example, if the 5 MHz value for phase velocity is used to fix Vp(0) and τ_b, β could be increased to 1.25 /cm/MHz before Vp(ω) exceeded the upper error limit at 10 MHz. The slope of attenuation could be increased to 1.02 /cm/MHz before Vp(ω) fell below the lower error limit at 2 MHz. Thus an error of more than 500 percent in β is consistent with the errors in phase velocity measured by Bhagat and coworkers.

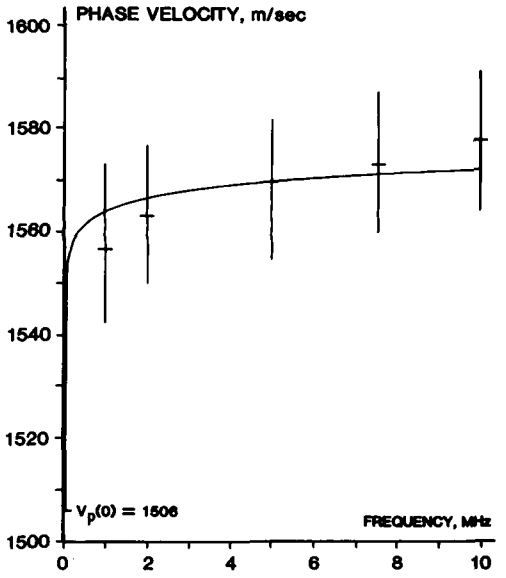

Fig. 11 Phase velocity of dog myocardium predicted by the Hilbert dispersive model. The curve was predicted from a slope of attenuation β of 0.154 /cm/MHz (1.34 dB) taken from measurements by Bhagat and coworkers ⟨13⟩. The phase velocity at 5 MHz was used to determine the bulk delay τ_b, which was 6.33 μs/cm. The minimum-phase delay factor was 20.

We can use the Hilbert dispersive model to establish allowable errors in measurements of phase velocity by calculating dispersion over the 1–10 MHz range and the change in phase velocity at a given frequency with a change in attenuation. We define the change in phase velocity over the range ω_1 to ω_2 as

$$\Delta V(\omega) = V_p(\omega_2) - V_p(\omega_1) \quad . \tag{30}$$

Applying Eq. (20) and assuming $V_p(\omega_1)V_p(\omega_2) \simeq V_p^2(\omega_1)$ because dispersion is quite small,

$$\Delta V(\omega) = \frac{\beta}{\pi^2} \ln(\omega_2/\omega_1) V_p^2(\omega_1) \quad . \tag{31}$$

Dispersion expected over the 1–10 MHz range is listed in table 3 for hemoglobin solutions and various soft tissues which cover a wide range in the slope of attenuation. As Eq. (31) states and table 3 demonstrates, the amount of dispersion is proportional to the size of the slope of attenuation.

Naturally the phase velocity predicted at a given frequency will change if the slope of attenuation changes. The sensitivity of the phase velocity to a small change in the slope of attenuation which might be caused by an error in the measurement of β suggests the accuracy needed in a phase velocity measuement to provide a representation of soft tissue equivalent to an attenuation measurement. We define the change in velocity at frequency ω for a change in the slope of attenuation from β_1 to β_2 as

$$\Delta V_\beta(\omega) = V_p(\beta_2,\omega) - V_p(\beta_1,\omega) \quad . \tag{32}$$

If the phase velocity at frequency ω_0 is used to fix the phase velocity at low frequency (Eq. (21)), then

$$\Delta V_\beta(\omega) = \frac{\beta_2-\beta_1}{\pi^2} \ln(\omega/\omega_0) V_p^2(\omega_0) \quad . \tag{33}$$

This change is also listed in table 3 for a 10 percent change in the slope of attenuation, assuming that $\omega_0 = 2\pi$ x 1 MHz and that the change $\Delta V_\beta(\omega)$ occurs at $\omega = 2\pi$ x 10 MHz. The percentage change in velocity is much much smaller than the 10 percent change in β which induced it. The results in table 3 suggest that the error in phase velocity measurements must be 2 to 3 orders of magnitude smaller than the error in the slope of attenuation, depending on the size of β, for a comparable ultrasonic characterization of soft tissue.

DISCUSSION

The Hilbert model introduced three closely-related parameters. Two appeared only in the derivation of the model. They were the high cutoff frequency ω_h and the decay factor γ which were used to modify the frequency response so that the Paley-Weiner conditon was met (see Eq. (A4)). The third, the minimum-phase delay factor τ_m, is dependent on both ω_h and γ. There are, of course, no measurements of these latter two quantities. In this paper τ_m was inferred from prior knowledge. We matched the phase of the Hilbert model to that of another causal tissue model, a single-pole model <19>. Actually τ_m can be calculated directly from Eq. (A9). The value of τ_m which we inferred from comparison to the single-pole tissue model, i.e., 20, is also obtained from Eq. (A9) if the cutoff frequency ω_h is 50 MHz and the decay factor γ is 0.9. These values are appropriate choices both for representing tissue in the 1-10 MHz frequency range and for satisfying the Paley-Weiner condition. The value of τ_m can be kept equal to 20 if both ω_h and γ are increased together. We presume that for a given value of τ_m, γ can be made as close to unity as desired if ω_h is high enough. This result says that the magnitude of the frequency response, as modified in Appendix A to satisfy the Paley-Weiner condition, can be made arbitrarily close to the expression usually assumed for the magnitude of the frequency response, namely Eq. (1), which was our starting point for the Hilbert model derivation.

Because the magnitude of the frequency response given by Eq. (1) has considerable experimental support, it was the basis for both the linear-phase and Hilbert dispersive models. Only the phase terms of the two models differ. In the derivation of our dispersive model we noted that any non-minimum phase system can be described by the product of a minimum-phase function and an all-pass function. The Hilbert transform gave the minimum-phase component of the total phase function of our model. We assumed that the phase of the all-pass function was a linear function of frequency, i.e., the same function as the phase of the linear-phase model. Such an all-pass function accounts for the delay encountered as ultrasound propagates through tissue. Because the linear-phase model is a good first order approximation to the frequency response of tissue, we do not expect the phase of an all-pass function representing tissue to differ by much from a linear term.

Although the phase functions of the linear-phase and Hilbert dispersive models differ in general, they are the same in a lossless medium, where the Hilbert model has linear phase (see Eq. (4)). Both models say that ultrasonic waveforms should be delayed, but not changed in shape as they propagate through a lossless medium ($\beta = 0$). If we consider the estimators of βx given in table 1, we find that in a lossless medium the RMS duration of the impulse response for both models becomes zero and the peak value for both becomes infinite. Thus in the lossless case these

estimators describe an impulse. If the impulse response is itself an impulse, then the waveform was indeed unchanged by propagation through lossless tissue. If the medium is not lossless, the Hilbert model says it must be dispersive and that the dispersion is anomalous. Dispersion is called anomalous if the group velocity is greater than the phase velocity. A low-loss transmission line is another example of a system in which dispersion is anomalous <26>.

According to the Hilbert model, the amount of dispersion expected for a given tissue sample can be quantified with Eq. (31) if the slope of attenuation β is known. O'Donnell and coworkers <25> quantified dispersion using the Kramers-Kronig relations. They predicted dispersion for two concentrations of hemoglobin (13 and 30g/100cc) measured by Carstensen and Schwan <16>. Our predictions for these and three other concentrations are shown in figure 9. The dispersion we measured from their curve for a concentration of 30g/100cc was 2.4 m/s. The dispersion we predicted with Eq. (31) and plotted in figure 10 is 2.1 m/s. One would expect these results to be identical because the Kramers-Kronig relations and the Hilbert transform are equivalent <19>. O'Donnell and coworkers, however, did not explicitly solve the Kramers-Kronig relation assuming a linear dependence of attenuation on frequency; they apparently integrated the attenuation as a function of frequency as measured by Carstensen and Schwan <16>.

If the assumptions of the Hilbert dispersive model are valid, then measurements of attenuation and phase velocity are not independent. We could just as easily have found, to within a constant, the attenuation of tissue from a knowledge of its dispersion <25>. Careful measurements of phase velocity or time-of-flight over a wide frequency range are needed to verify more fully the predictions of the Hilbert model. If however, attenuation and phase velocity are indeed dependent on one another, measurement of either would give an equivalent description of tissue. Attenuation would, however, be the measurement of choice because measurement error can be 2 to 3 orders of magnitude greater than that required to get an equivalent description of tissue by measuring phase velocity or time of flight.

CONCLUSIONS

The Hilbert dispersive model is a phenomenological tissue model which successfully describes the interaction of ultrasound with tissue in both the time and frequency domains. It should be valuable in simulations used to aid in the design of transducer arrays and signal processing schemes for quantitative, tissue-imaging systems.

In contrast to a linear-phase model the Hilbert model predicts a causal impulse response for tissue. Furthermore, both the RMS duration and peak value of the impulse response yield accurate estimates of the slope of attenuation times path length. Using only the slope of attenuation the Hilbert model can accurately predict tissue dispersion. The success of the model's predictions of dispersion suggests that attenuation and phase velocity measurements as functions of frequency are functionally related to within a constant. If indeed the frequency dependence of attenuation and the the frequency dependence of phase velocity can be inferred from one another, as given by the Hilbert dispersive model, then attenuation would be the measurement of choice. This choice follows because according to the model, phase velocity measurements must be much more accurate than attenuation measurements for a comparable description of tissue.

APPENDIX A

DERIVATION OF THE PHASE TERM FOR CAUSAL RESPONSE

A suitable phase function $\theta(\omega)$ can be found for a linear system from the magnitude of its frequency response $|H(\omega)|$, so that the impulse response of the system is causal. The complete frequency domain description, the frequency response, can be written

$$H(\omega) = |H(\omega)| \, e^{-j\theta(\omega)} \quad , \tag{A1}$$

where

$$|H(\omega)| = e^{-\alpha(\omega)} \quad . \tag{A2}$$

If $|H(\omega)|$ is square integrable (finite energy condition) and satisfies the Paley-Weiner conditon, then $|H(\omega)|$ is the Fourier spectrum of a causal function <17,18>. To find $\theta(\omega)$ for such a function, we note that any non-minimum-phase transfer function can be written as the product of an all-pass function times a minimum-phase function <18>. We assumed that the all-pass function in our tissue model fixed the propagation delay, i.e., that the phase of the all-pass function was a linear function of frequency. The minimum-phase component of the total phase expression is given by the Hilbert transform

$$\theta_{min}(\omega) = \frac{\omega}{\pi} \int_{-\infty}^{\infty} \frac{\alpha(s)}{s^2 - \omega^2} \, ds \quad . \tag{A3}$$

Unfortunately the magnitude of the frequency response usually assumed for tissue (see Eq. (1)) does not satisfy the Paley-Weiner condition. With a minor modification, however, it does. We must impose a high-frequency limit, beyond which the magnitude function does not go to zero faster than an exponential <27>. With this limit equal to ω_h and with γ, which must be positive and less than one, as the factor which controls the rate of decay at high frequencies, the magnitude of the response becomes

$$\begin{aligned} |H(\omega)| &= e^{-\beta x \frac{|\omega|}{2\pi}} && 0 < |\omega| < \omega_h \\ &= e^{-\beta x \frac{|\omega_h|}{2\pi}} \, e^{-\beta x \frac{(|\omega| - |\omega_h|)^{\gamma}}{2\pi}} . && |\omega| > \omega_h \end{aligned} \tag{A4}$$

This choice of magnitude function satisfies the Paley-Weiner condition because the integral expression below is bounded:

$$\int_{-\infty}^{\infty} \frac{|\ln|H(\omega)||}{1 + \omega^2} \, dx < \frac{\beta x}{2\sin((1+\gamma)\pi/2)} \quad . \tag{A5}$$

The finite energy condition is also met for this choice of $|H(\omega)|$:

$$\int_{-\infty}^{\infty} |H(\omega)|^2 \, d\omega < \frac{\Gamma(1/\gamma)}{\gamma(\beta x/2\pi)^{1/\gamma}} \quad , \tag{A6}$$

where $\Gamma(\cdot)$ is a gamma function.

For $|H(\omega)|$ as given in Eq. (4), $\theta_{min}(\omega)$ becomes

$$\theta_{min}(\omega) = \frac{\beta x}{2\pi^2}\,\omega\,\ln\left[\frac{\omega_h^2 - \omega^2}{\omega^2}\right] + \frac{\beta x}{\pi^2}\,\omega\int_{\omega_h}^{\infty}\frac{\omega_h + (s-\omega_h)^2}{s^2 - \omega^2}\,ds \quad . \tag{A7}$$

If we require that $\omega_h^2 \gg \omega^2$ and if we let $\xi = 1/s$ as suggested by Seshu and Balabanian <17> in their discussion of the contribution to the phase of frequency components in the stop band of a low-pass filter, then

$$\theta_{min}(\omega) = \omega x\,\frac{\beta}{\pi^2}\left[\,\tau_m - \ln(\omega)\right] \tag{A8}$$

where

$$\tau_m = 1 + \ln(\omega_h) + \int_0^{1/\omega_h}(\xi^{-1} - \omega_h)^{\gamma}\,d\xi \quad . \tag{A9}$$

The total phase per unit length was found by adding the bulk delay term, $\omega\tau_b x$ (Eq. (2)), of the linear-phase model to the minimum-phase term <28> and dividing by the path length x:

$$\frac{\theta(\omega)}{x} = \omega\tau_b + \omega\,\frac{\beta}{\pi^2}\left[\,\tau_m - \ln(\omega)\right] \quad . \tag{A10}$$

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