General relationships between ultrasonic attenuation and dispersion

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General relationships between the ultrasonic attenuation and dispersion are presented. The validity of these nonlocal relationships hinges only on the properties of causality and linearity, and does not depend upon details of the mechanism responsible for the attenuation and dispersion. Approximate, nearly local relationships are presented and are demonstrated to predict accurately the ultrasonic dispersion in solutions of hemoglobin from the results of attenuation measurements.

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The purposes of this letter are twofold: (1) to explore the implications of the existence of general relationships between the ultrasonic attenuation and the frequency dependence of the ultrasonic phase velocity (i.e., dispersion) for identifying specific mechanisms responsible for the propagation of ultrasound in some media, and (2) to present nearly local forms of these general relationships. We illustrate the use of these nearly local relationships for cases of ultrasonic propagation in media of biomedical interest, an area in which the mechanisms governing the propagation remain obscure.

Relationships between attenuation and dispersion, sometimes called Kramers–Kronig or generalized dispersion relationships, have proved useful in several areas of physics.[1–4] Expressed in a form appropriate to ultrasonic studies, these relationships take the form

$$K_1(\omega) - K_1(\infty) = \frac{2}{\pi} \int_0^\infty \frac{\omega' K_2(\omega')}{\omega'^2 - \omega^2} d\omega' \,, \tag{1}$$

$$K_2(\omega) = -\frac{2\omega}{\pi} \int_0^\infty \frac{K_1(\omega') - K_1(\infty)}{\omega'^2 - \omega^2} d\omega' \,, \tag{2}$$

where $K_1(\omega)$ and $K_2(\omega)$ are the real and imaginary parts, respectively, of the dynamic compressibility (inverse of the bulk modulus). If the ultrasonic wave vector is written as $k = \omega/C(\omega) + i\alpha(\omega)$, then $C(\omega)$ is the phase velocity and $\alpha(\omega)$ is the attenuation coefficient for the incident wave, as observed in transmission (i.e., direct "straight-line" propagation) measurements.

The following considerations involve only this total attenuation coefficient, and do not depend on details of the ultimate physical mechanism (i.e., absorption, scattering, or a mixture of both). That is, the effect on the incident wave of removing energy from it is the same whether that removed energy is converted immediately into heat, or whether it goes first into scattered waves which subsequently decay into heat. Indeed, there is no sharp distinction between these mechanisms; a local absorption of energy can always be thought of as the limit of a scattered wave which propagates only a short distance. In either case, the effect on the incident wave can be represented by a phenomenological compressibility $K(\omega)$ obeying Eqs. (1) and (2), which can be used to define the relationship between the attenuation and dispersion. In the limit $\alpha(\omega)C(\omega)/\omega \ll 1$ (i.e., if the real part of the wave vector is much larger than its imaginary part), the real and imaginary parts of the compressibility can be directly related to the attenuation coefficient and phase velocity such that

$$C(\omega) \simeq \frac{1}{[\rho_0 K_1(\omega)]^{1/2}} \,, \tag{3}$$

$$\alpha(\omega) \simeq \tfrac{1}{2}\rho_0 C(\omega) K_2(\omega) \,. \tag{4}$$

Using Eqs. (1) and (3) the dispersion at a specified frequency can be computed from a knowledge of the attenuation at all frequencies. Conversely, if the dispersion is known at all frequencies, the attenuation at any specified frequency can be computed from Eqs. (2) and (4).

The validity of the general form of the Kramers–Kronig relationships [Eqs. (1) and (2)] hinges only on the properties of causality (that an effect does not precede its cause) and linearity (that a response is approximately proportional to its stimulus). We make the usual physical assumption that a spatially local compressibility exists, relating pressure and density, in a re-

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gion large compared to atomic dimensions, but small compared to a wavelength.

For present purposes the significant feature of these relationships is that they do not depend upon details of the specific mechanism responsible for the attenuation and dispersion. As illustrated below, the existence of these completely general relationships renders invalid attempts to compare the attenuation and dispersion as a means of validating any specific model proposed to account for the propagation of ultrasound in some medium, as some authors appear to have attempted.[5,6]

In the form given by Eqs. (1) and (2) the Kramers–Kronig relationships are limited in usefulness because of their nonlocal character; i.e., a knowledge of either the attenuation or the dispersion for all frequencies is required. More useful, approximate attenuation–dispersion relationships in a nearly local form can be obtained from the nonlocal form using an approach analogous to that used by Bode in the study of the relationship between the gain and phase shift of an amplifier.[7] The nearly local forms of the attenuation–dispersion relationships are obtained from the exact nonlocal forms given in Eqs. (1) and (2) under the assumptions that the attenuation and dispersion are sufficiently small and do not change rapidly over the frequency range of interest. A review of the derivation of Eqs. (1) and (2) and a discussion of the range of validity of the approximate relationships presented below will be presented in a subsequent publication.[8] The nearly local relationships are

$$\alpha(\omega) \simeq (\pi\omega^2/2C_0^2)\frac{dC(\omega)}{d\omega}\ , \tag{5}$$

$$\Delta C(\omega) = C(\omega) - C_0 \simeq \frac{2C_0^2}{\pi}\int_{\omega_0}^{\omega}\frac{\alpha(\omega')}{\omega'^2}d\omega'\ , \tag{6}$$

where ω_0 is some convenient reference frequency and $C_0 \equiv C(\omega_0)$ is the phase velocity at this reference frequency.

Although strictly appropriate only under rather restrictive conditions, Eqs. (5) and (6) appear to be excellent approximations for cases encountered in the study of soft tissue. In Table I we indicate the frequency dependences observed for classical viscous losses and for the attenuation in most soft tissues. The corresponding frequency dependences for the dispersion are predicted using Eq. (6). From Table I, if the attenuation varies linearly with frequency, the dispersion should vary logarithmically with frequency, regardless of the details of the specific mechanism responsible for the linear frequency dependence of the attenuation.

To illustrate the use of the nearly local attenuation–dispersion relationships, we present in Fig. 1(a) the attenuation measured as a function of frequency by Carstensen and Schwan[5] in two solutions of hemoglobin. In Fig. 1(b) we compare the dispersion measured by Carstensen and Schwan to the dispersion predicted on the basis of the measured attenuation and Eq. (6), where C_0 was taken to be the phase velocity at 1 MHz. Agreement between the measured and predicted dispersion is excellent. Further, if the dispersion data are plotted on a logarithmic frequency scale, the data lie approximately on a straight line, in agreement with the prediction of Table I for the case of a nearly linear dependence of attenuation on frequency.

TABLE I. Observed frequency dependences of attenuation coefficient α for classical viscous losses and for the case of soft tissue, and corresponding frequency dependences of ΔC predicted from Eq. (6).

Description	Frequency dependence of attenuation $\alpha(\omega)$	Frequency dependence of $\Delta C(\omega)$
Classical Viscous	ω^2	ω^1
"Soft Tissue"	ω^1	$\ln \omega$

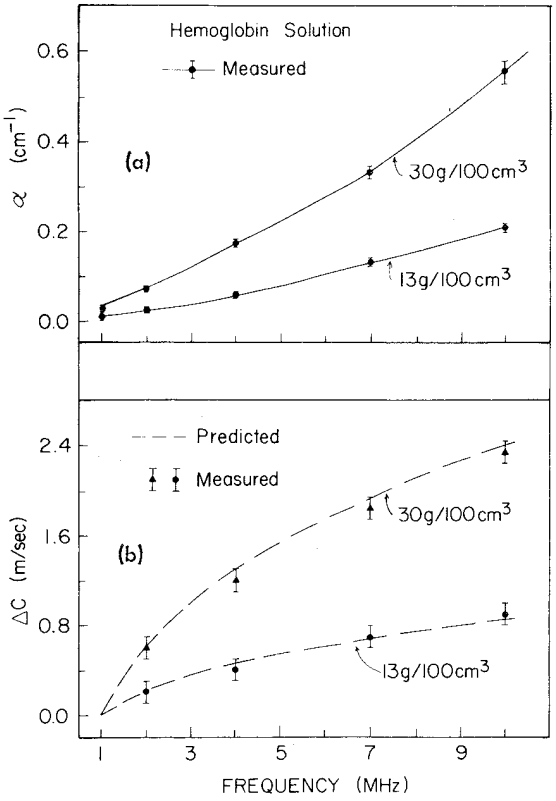

FIG. 1. (a) The attenuation coefficient measured by Carstensen and Schwan[5] is plotted as a function of frequency for hemoglobin solutions of two concentrations. (b) The change ΔC in the velocity of sound from its value C_0 at 1 MHz is displayed as a function of frequency for the same two solutions. The data points correspond to measurements made by Carstensen and Schwan, and the dashed curves were computed using Eq. (6) and the attenuation data of Fig. 1(a).

These results demonstrate the need for determining which features of ultrasonic propagation are determined by general laws of physics, as opposed to those features which are specific to the particular mechanism of propagation. An appreciation of the distinction should prove useful in establishing the mechanisms responsible for the propagation of ultrasound in biologically interesting specimens. The same lesson was learned many years ago by workers in ferromagnetism and ferroelectricity, with the emergence of a general rule that only experiments at the molecular level can distinguish reliably between different molecular models.

ACKNOWLEDGMENT

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