[2] W. J. Crochetiere, L. Vodovnik, and J. B. Reswick, "Electrical stimulation of skeletal muscle—A study of muscle as an actuator," *Med. Biol. Eng.*, vol. 5, pp. 111-125, 1967.
[3] S. Rebersek and L. Vodovnik, "Proportionally controlled functional electrical stimulation of the hand," *Arch. Phys. Med. Rehab.*, vol. 45, pp. 378-382, 1972.
[4] U. Stanic and A. Trnkoczy, "Closed-loop positioning of hemiplegic patient's joint by means of functional electrical stimulation," *IEEE Trans. Biomed. Eng.*, vol. 21, pp. 365-370, May 1974.
[5] R.Merletti, R. Acimovic, S. Grobelnik, and G. Cvilak, "Electrophysiological orthosis for the upper extremity in hemiplegia: A feasibility study," *Arch. Phys. Med. Rehab.*, vol. 56, pp. 507-513, 1975.
[6] R. H. Nathan, "Closed loop control of the isometric joint moment in a multi-degree of freedom joint," in *Proc. III Med. Conf. Med. Biol. Eng.*, paper 3.3, 1983.
[7] ——, "The development of a computerized upper limb electrical stimulation system," *Orthopaed.*, vol. 7, pp. 1170-1180, 1984.
[8] Y. Handa, M. Ichie, T. Handa, R. Yagi, and N. Hoshimiya, "Control of the paralyzed hand by a computer-controlled FES system," in *Proc. 7th Ann. Conf. IEEE Eng. Med. Biol. Soc.*, Chicago, IL, 1985, pp. 322-326.
[9] P. E. Crago, P. H. Peckham, and G. B. Thrope, "Modulation of muscle force by recruitment during intramuscular stimulation," *IEEE Trans. Biomed. Eng.*, vol. BME-27, 1980.
[10] P. H. Peckham, J. T. Mortimer, and E. B. Marsolais, "Controlled prehension and release in the C-5 quadriplegic elicited by FES of the paralyzed forearm musculature," *Ann. Biomed. Eng.*, vol. 8, pp. 369-388, 1981.
[11] H. J. Chizeck, P. E. Crago, and L. S. Kofman, "Robust closed-loop control of isometric muscle force using pulsewidth modulation," *IEEE Trans. Biomed. Eng.*, vol. 35, pp. 510-517, July 1988.
[12] P. H. Peckham, "Control of contraction strength of electrically stimulated muscle by pulse width and frequency modulation," in *Proc. 29th Ann. Conf. Eng. Med. Biol.*, 1976.
[13] J. S. Petrofsky, "Control of the recruitment and firing frequencies of motor units in electrically stimulated muscles in the cat," *Med. Biol. Eng. Comput.*, vol. 16, pp. 302-308, 1978.
[14] P. E. Crago, J. T. Mortimer, and P. H. Peckham, "Closed loop control of force during electrically stimulated muscle," *IEEE Trans. Biomed. Eng.*, vol. 27, pp. 306-312, 1980.
[15] R. H. Nathan, "An FNS-based system for generating upper limb function in the C4 quadriplegic," *Med. Biol. Eng. Comp.*, vol. 27, pp. 549-556, 1989.
[16] L. Vodovnik, C. Long, and A. Lippay, "Pain response to different tetanising currents," *Arch. Phys. Med. Rehab.*, vol. 46, pp. 187-192, 1965.
[17] F. Gracanin and A. Trnkoczy, "Optimal stimulation parameters for minimum pain in chronic stimulation of innervated muscle," *Arch. Phys. Med. Rehab.*, vol. 56, pp. 243-249, 1975.
[18] P. H. Peckham, J. P. Van Der Muelen, and J. B. Reswick, "Electrical activation of skeletal muscle by sequential stimulation" in *The Nervous System and Electric Currents*, N. Wulfson and A. Sances, Eds. New York: Plenum, 1970, pp. 45-50.
[19] J. S. Petrofsky, "Sequential motor unit stimulation through peripheral motor nerves," *Med. Biol. Eng. Comp.*, vol. 17, pp. 87-93, 1979.
[20] P. H. Peckham and J. T. Mortimer, "Restoration of hand function in the quadriplegic through electrical stimulation," in *Functional Electrical Stimulation Applications in Neural Prostheses*, F. T. Hambrecht and J. B. Reswick, Eds. New York: Dekker, 1977.
[21] J. S. Petrofsky, C. A. Phillips, and H. H. Heaton, "Feedback control system for walking in man," *Comp. Biol. Med.*, vol. 14, no. 2, pp. 135-149, 1984.
[22] R. H. Nathan, "Fatigue of electrically stimulated muscles," *Artif. Organs*, vol. 8, pp. 388-389, 1984.
[23] M. Solomonow, "Restoration of movement by electrical stimulation," *Orthopaed.*, vol. 7, no. 2, pp. 245-250, 1984.
[24] M. Solomonow, E. Eldred, J. Lyman, and J. Foster, "Control of muscle contractile force through indirect high-frequency stimulation," *Amer. J. Phys. Med.*, vol. 62, no. 2, pp. 71-82, 1983.
[25] B. Zhou, R. Baratta, and M. Solomonow, "Manipulation of muscle force with various firing rate and recruitment control strategies," *IEEE Trans. Biomed. Eng.*, vol. BME-34, pp. 128-139, Feb. 1987.
[26] R. H. Nathan, "A dynamometer for biomechanical use," *J. Biomed. Eng.*, no. 2, pp. 83-88, 1979.
[27] P. H. Gorman and J. T. Mortimer, "Effect of stimulus parameters on the recruitment characteristics of direct nerve stimulation," *IEEE Trans. Biomed. Eng.*, vol. BME-30, pp. 407-414, July 1983.

Pulsed Doppler Accuracy Assessment Due to Frequency-Dependent Attenuation and Rayleigh Scattering Error Sources

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Abstract—**All engineering measurements are subject to inaccurate and imprecise estimates, including the estimate of blood flow velocity. An assessment of specific error sources can minimize such uncertainties. Frequency-dependent attenuation and Rayleigh scattering are significant error sources for pulsed Doppler ultrasound because the transmitted ultrasonic signal has a finite width spectrum. The former causes a frequency downshift and the latter a frequency upshift, both of which are independent of the actual Doppler frequency shift. This communication evaluates these error sources through computer simulation and compares the computed error to experimental data.**

INTRODUCTION

The measurement of human blood flow by ultrasound is a valuable tool for clinical diagnosis of vascular disease. Unfortunately, Doppler-based measurement techniques are plagued with practical as well as theoretical difficulties which result in inaccurate and imprecise flow measurements. A review of its accuracy and sources of error [1] concluded that Doppler methods are capable of good absolute accuracy when suitably designed equipment is used in appropriate situations, with systematic errors of 6% or less. One error source not evaluated in that review was that due to the frequency-dependent tissue attenuation of the intervening tissue. The influence of Rayleigh scattering and frequency-dependent attenuation, two spectral-broadening mechanisms, have been theoretically treated for a Gaussian spectrum [2] in which the attenuation coefficient varied linearly with frequency. In addition to these two spectral-broadening mechanisms, transducer characteristics have also been shown to influence significantly the Doppler spectrum [3], [4]. Experimental results concerning the effects of tissue attenuation on the accuracy of pulsed Doppler ultrasound were presented in [4], and compared to the model in [2]. In this paper, the

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frequency-dependent attenuation error source is evaluated with a more general tissue attenuation model through computer simulation of the spectrum generated by a simple mechanically-focused transducer and compared to previous results [4].

To assess quantitatively the effect of frequency-dependent attenuation, this simulation assumes that the flow meter measures the mean Doppler frequency determined from the ultrasonic backscattered signal. Because the pulsed Doppler spectral estimate is biased due to a finite signal to noise ratio [5], it is also assumed that no noise is present in the received signal. For simplicity, blood motion is modeled to be moving away from the transducer at a constant, uniform velocity. An approximation valid for long bursts of sine-wave excitation (greater than approximately 5 cycles) of the pressure signal spectrum from a mechanically focused transducer is given by [6]

$$T(f) = H(f)\,\mathrm{Sinc}\left(M\left(\frac{f}{f_o} - 1\right)\right) \tag{1}$$

$$H(f) = \frac{ff_o}{Q\sqrt{(f^2 - f_o^2)^2 + (ff_o/Q)^2}} \tag{2}$$

where

$T(f)$ = spectrum of transmitted pressure waveform.
$H(f)$ = transducer second order transfer function.
Sinc (f) = $\sin(\pi f)/\pi f$.
M = number of cycles of center frequency excitation.
f_o = center frequency (Hz).
Q = transducer quality factor (f_o/bandwidth).

The frequency-dependent total attenuation of tissue is modeled by [7], [8]

$$A(f) = A_o f^b \tag{3}$$

where

$A(f)$ = total attenuation as a function of frequency (dB).
A_o, b = frequency-independent tissue constants.
f = frequency in MHz.

The ultrasonic propagation distance is included in A_o. For a one-way tissue path length d, at a frequency of 1 MHz, the total attenuation A_o is equal to $a_o d$ where a_o is the attenuation coefficient at 1 MHz (unit of dB/cm-MHz for example when $b = 1$). The value of a_o at 1 MHz varies from nearly zero (<0.001 dB/cm) for water to about 1 dB/cm for some soft tissues [7]-[11]. For peripheral vascular blood flow measurements, the one way path length may be less than 1 cm resulting in an A_o value of less than 1 dB/MHz whereas for deep abdominal blood flow measurements, the value of A_o may be as great as 10 dB/MHz. The value of b varies from 1.0 to 1.4 for many soft tissues [7]-[10] and is 2 for water [11]. The frequency-dependent attenuation was assumed to vary linearly with frequency ($b = 1$) in previous analyses [2]-[4].

The magnitude of the incident pressure $|B(f)|$ at the blood vessel is dependent upon the transmitted pressure waveform spectrum and the total attenuation:

$$B(f) = |T(f)|\,10^{-[A(f)/20]}. \tag{4}$$

When ultrasound is scattered by a moving object, each of the frequency components of the scattered waveform is shifted according to the Doppler relation,

$$f_{sc} = f\left(1 - \frac{2V}{c}\right) \tag{5}$$

where

f_{sc} = frequencies backscattered by blood.
f = frequencies incident on blood.
V = magnitude of the scatterer's velocity (positive is away from the transducer).
c = speed of sound.

Also, because the moving blood is a collection of particles which are much smaller than the wavelength of the ultrasound, the backscattering coefficient varies with frequency. It is normally assumed that blood is a Rayleigh scatterer [2]-[4] implying that the scattered power is proportional to f^4. This frequency dependent backscattering shifts the mean spectral frequency to higher frequencies and counteracts the tissue attenuation effects. The backscattered pressure $S(f_{sc})$ is given by

$$S(f_{sc}) = f_{sc}^2 B(f_{sc}). \tag{6}$$

As the wave propagates back through the tissue, it is again attenuated according to (3). The transducer then filters the pressure signal incident on the transducer TP(f_{sc}). The received electrical signal $R(f_{sc})$ is

$$R(f_{sc}) = H(f_{sc})\,S(f_{sc})\,10^{-[A(f_{sc})/20]}. \tag{7}$$

The example shown in Table I illustrates the simulation method for a typical soft tissue with A_o = 1.0 dB/MHz, b = 1.4, and c = 1540 m/s [7]-[10]. The transducer is modeled by (2) with Q = 3 and f_o = 5 MHz for two values of M, 5 and 10. The magnitude of the scatterer's velocity (target speed), V for this case is 10 m/s. For the M = 5 case, the mean frequency (f_c) which is incident at the scattering target is 4.936 MHz. The tissue attenuation caused a −63.96 kHz shift from the transmitted spectrum. The Doppler shift caused by the 10 m/s scatterer motion in this case is −64.94 kHz as determined from (5). Without the Rayleigh scattering, the mean frequency of the scattered acoustic spectrum would be 4.8711 MHz (a shift of −128.90 kHz from 5.000 MHz). However, from (6) the mean frequency of $S(f_{sc})$ is 4.954 MHz (a shift of −46 kHz) which demonstrates the upward frequency shift due to the Rayleigh scattering assumption. Fig. 1 demonstrates the mean frequency shift due only to Rayleigh scattering as a function of M (f_o and M are the only variables which affect this frequency shift).

During the reverse transit back to the transducer, the tissue attenuation causes a further −61 kHz shift in the mean frequency of the ultrasonic signal. The received echo signal after transducer filtering has a mean frequency shift of −83 kHz. The transducer filtering causes the mean value of the spectrum to shift towards f_o. Thus, a Doppler shift of −64.94 kHz results in a mean frequency shift of −83 kHz in the received echo signal.

The results for M = 10 are also shown in Table I. Here a mean frequency shift of −72 kHz (less than that for M = 5) in the received echo signal compared to the Doppler shift of −64.94 kHz. In general, as M increases, the received echo signal shift approaches the Doppler shift.

Fig. 2 shows the simulated mean frequency shift relative to f_o of $R(f_{sc})$ versus target speed and Table II lists the mean frequency shift at a target speed of zero. A clear relationship between target speed and mean frequency shift is demonstrated. The slope of the mean frequency shift versus target speed is constant for a constant M and decreases as M increases (−5.08 kHz/ms^{-1} for M = 5 and −5.96 kHz/ms^{-1} for M = 10) with a limiting slope of −6.49 kHz/ms^{-1} ($-2f_o/c$) for the Doppler frequency shift. Furthermore, the mean frequency shift decreases as attenuation (either A_o or b) increases.

Fig. 3 shows the mean frequency shift of $R(f_{sc})$ relative to f_o versus the number of transmit cycles. When A_o = 0, the frequency shift is due only to Rayleigh scattering (upper most curve). The other curves show the mean frequency shift for A_o = 1.0 dB/MHz to 4 dB/MHz for b = 1.0. Because the frequency-dependent attenuation shift is directly proportional to A_o, the shift for other combinations of tissue thickness and attenuation coefficient values can be determined by interpolating the value given in Fig. 3. Received frequency shift due to frequency-dependent attenuation decreases as the number of transmitted cycles increases and the transmitted bandwidth decreases.

These simulation results show that a large error would result if the mean frequency of $R(f_{sc})$ were used to estimate the magnitude of blood flow velocity. For example, considering only the fre-

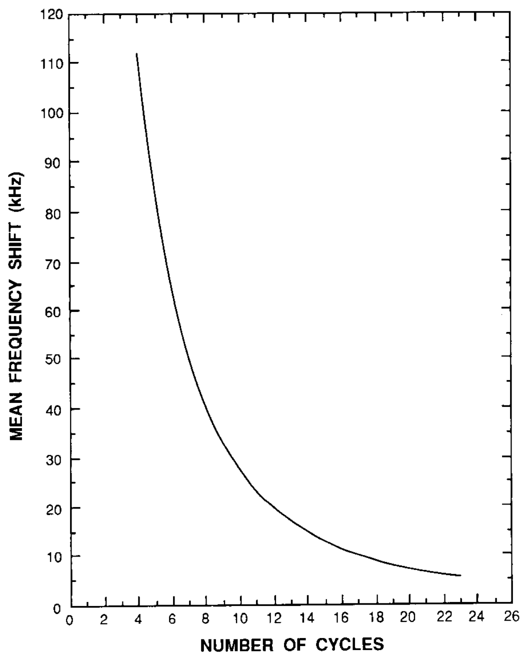

Fig. 1. Mean frequency shift due only to Rayleigh scattering versus M [see (1)].

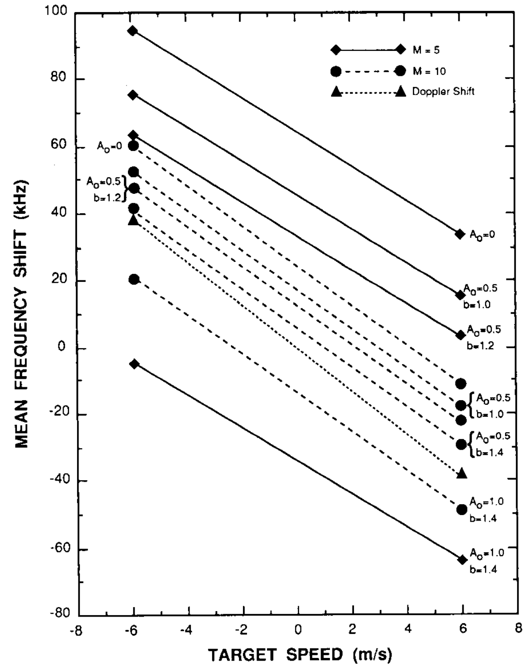

Fig. 2. Simulation results of the mean frequency shift relative to f_o of $R(f_{sc})$ versus target speed for different A_o (in dB/MHz) and b values for $M = 5$ and 10 cycles.

TABLE I

Mean Frequency (f_c) and − 3 dB Bandwidth (BW) Simulation Results of Typical Spectra at $M = 5$ and 10 for $f_o = 5$ MHz, $Q = 3$, $A_o = 1.0$ dB/MHz, $b = 1.4$, $V = +10$ m/s and $c = 1540$ m/s

Spectrum	f_c (MHz)	BW (kHz)	f_c/BW
For $M = 5$			
$H(f)$	5.000	1666.7	3.0
$T(f)$	5.000	765.6	6.5
$B(f)$	4.936	758.4	6.6
$S(f_{sc})$	4.954	746.2	6.7
$R(f_{sc})$	4.917	660.9	7.6
For $M = 10$			
$H(f)$	5.000	1666.7	3.0
$T(f)$	5.000	427.2	11.7
$B(f)$	4.979	424.8	11.8
$S(f_{sc})$	4.941	419.3	11.9
$R(f_{sc})$	4.928	400.3	12.5

quency-dependent attenuation as an error source where $f_o = 5$ MHz, $M = 5$, $A_o = 1$ dB/MHz, $b = 1.0$, and $V = -1$ m/s (a rather large speed for blood), the mean frequency shift of $R(f_{sc})$ for attenuation only is -48.94 kHz and for attenuation and flow is -42.45 kHz. The Doppler shift is 6.49 kHz. The resulting speed estimate considering only frequency-dependent attenuation is $+7.54$ m/s and considering frequency-dependent attenuation and flow is $+6.54$ m/s. If frequency shift due to Rayleigh scattering is also considered ($+83.61$ kHz), the speed estimate is -6.34 m/s. And, *via* the simulation which also includes the transducer transfer function, the resulting speed estimate is -4.94 m/s.

The above error analysis indicates why pulsed Doppler flow meters estimate the Doppler shift from the frequency of the complex demodulated signal, acquired at one range gate normally the same length of the transmit pulse) at a complex sampling rate equal to the pulse repetition frequency (PRF). In order to reduce the noise level in the range gated signal, the complex signal is usually integrated throughout the range gate and sampled with analog-to-dig-

TABLE II

Mean Frequency Shift Relative to f_o of $R(f_{sc})$ at a Target Speed of Zero for Various Values of A_o, b, and M. $f_o = 5$ MHz and $c = 1540$ m/s

		Mean Frequency Shift (kHz)	
A_o (dB/MHz)	b	$M = 5$	$M = 10$
0.0		64.57	24.97
0.25	1.0	55.32	21.06
	1.2	49.14	18.72
	1.4	39.60	15.43
	1.6	25.23	9.92
0.50	1.0	46.09	17.81
	1.2	33.66	13.08
	1.4	15.11	5.87
	1.6	−13.24	−5.38
0.75	1.0	36.79	13.93
	1.2	18.15	6.98
	1.4	−9.53	−3.67
	1.6	−51.27	−20.21
1.0	1.0	27.41	10.65
	1.2	3.19	1.18
	1.4	−33.90	−13.20
	1.6	−88.08	−34.81
1.25	1.0	18.00	6.80
	1.2	−12.30	−4.69
	1.4	−57.88	−22.70
	1.6	−125.10	−49.31
1.50	1.0	8.61	3.48
	1.2	−27.43	−10.71
	1.4	−81.47	−32.19
	1.6	−161.47	−63.73

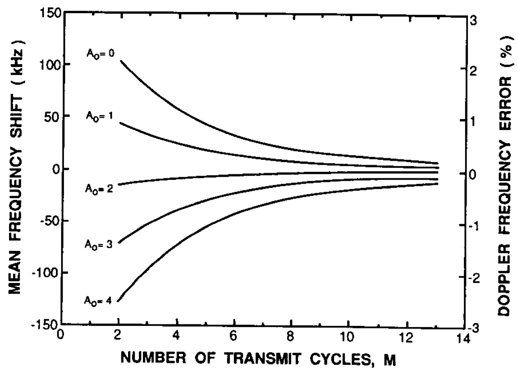

Fig. 3. Simulation results of the mean frequency bias of a received echo signal and the Doppler frequency error for $f_o = 5$ MHz versus number of cycles transmitted (M) for $b = 1.0$ and different A_o values from 0 to 4 dB/MHz. The top curve represents $A_o = 0$ dB/MHz for any b value. The Doppler frequency error comes from (10) for $f_o = 5$ MHz.

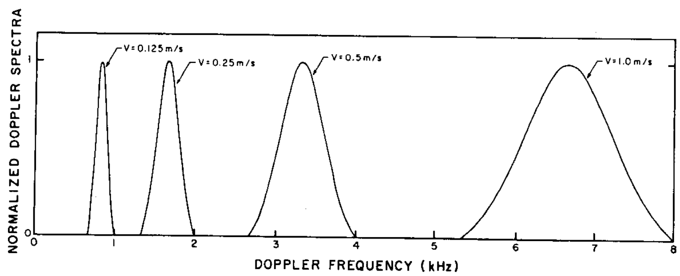

Fig. 4. Illustration of the normalized Doppler spectrum (linear magnitude) versus Doppler frequency for four different target velocities with $f_o = 5$ MHz and $M = 5$.

ital converters at the end of the range gate. This averaging of the demodulated signal results in an additional sinc frequency response centered at f_o. The spectrum of this detected range gated complex signal from a number of consecutive echoes is then determined by real time FFT spectral analysis (typically 128 complex samples are used). The mean Doppler shift can then be computed from the first moment of the output of the FFT spectral estimate. The Doppler spectrum resulting from an ideal complex envelope detector $D(f)$ is related to the received RF spectrum $R(f)$ as follows [3], [13]:

$$D(f) = R\left(\frac{f_{sc}c}{2V}\right) \text{Sinc}\left(M\left\{\frac{f_{sc}c}{2f_oV} - 1\right\}\right). \quad (8)$$

This relationship is illustrated in Fig. 4 for target speeds of 0.125, 0.25, 0.5, and 1 m/s and a center frequency of 5 MHz. Note that if the target speed were zero, as it is for stationary wall echoes, then the Doppler spectrum $D(f)$ becomes an impulse at zero frequency indicating a dc component in the complex signal. The frequency bias in the received RF echo signal $R(f)$, indicated in Fig. 3, gives rise to a change in the moment of the Doppler spectrum which is related to the mean received signal and the target speed. The error in the Doppler frequency shift ΔD can be shown to be related to the mean frequency shift due to tissue attenuation (ΔR) as follows

$$\Delta D = \frac{2V}{c}\Delta R. \quad (9)$$

Thus, the error for $V = 0$ is zero and independent of the receive signal spectrum. For nonzero target speeds, the fractional Doppler error ($\Delta D/D$) is

$$\frac{\Delta D}{D} = \frac{\Delta R}{f_o} \quad (10)$$

where D is the Doppler frequency shift without tissue attenuation and f_o is the transducer center frequency. The percentage Doppler frequency error is indicated on the right hand side of Fig. 3 for the 5 MHz center frequency.

The results presented here generally agree with the experimental findings in [4] which were obtained with a tissue phantom and a commercial Doppler system with a center frequency of 3.5 MHz. The transducer was excited with five cycles ($M = 5$). Even with the low transducer center frequency, errors of 12-15% were reported at a depth of 7 cm. A simulation using the measured parameters for the experiments in [4] ($A_o = 3$ dB, $b = 1.67$) indicated a mean receive frequency shift of −334 kHz. The resulting Doppler error for this case should be −9.6% which agrees quite well with the experimental findings. From the experimental results in [4] and the computer simulation results presented here, it is clear that quantitative use of conventional Doppler ultrasound should be restricted to peripheral vessels where tissue path lengths are less than a few centimeters.

References

[1] R. W. Gill, "Measurement of blood flow by ultrasound: Accuracy and sources of error," *Ultrasound Med. Biol.*, vol. 11, pp. 625-641, 1985.

[2] V. L. Newhouse, A. R. Ehrenwald, and G. F. Johnson, "The effect of Rayleigh scattering and frequency-dependent absortion on the output spectrum of Doppler blood flowmeters," in *Ultrasound in Medi-*

cine, Vol. 3B, D. White and R. E. Brown, Eds. New York: Plenum, 1977, pp. 1181–1191.

[3] S. K. Holland, "Estimation of blood flow parameters using pulsed Doppler ultrasound with corrections for spectral broadening," Ph.D. dissertation, Yale Univ., New Haven, CT, Dec. 1985.

[4] S. K. Holland, S. C. Orphanoudakis, and C. C. Jaffe, "Frequency-dependent attenuation effects in pulsed Doppler ultrasound: Experimental results," *IEEE Trans. Biomed. Eng.*, vol. BME-31, pp. 626–631, Sept. 1984.

[5] K. Kristoffersen, "Time-domain estimation of the center frequency and spread of Doppler spectra in diagnostic ultrasound," *IEEE Trans. Ultrason. Ferroelect. Freq. Contr.*, vol. 35, no. 6, pp. 685–700, Nov. 1988.

[6] Y. Kihuchi, Ed., *Ultrasonic Transducers.* Tokyo: Orona, Tokyo, 1969.

[7] J. D. Pohlhammer, C. A. Edwards, and W. D. O'Brien, Jr., "Phase insensitive ultrasonic attenuation coefficient determination of fresh bovine liver over an extended frequency range," *Med. Phys.*, vol. 8, no. 5, pp. 692–694, Sept./Oct. 1981.

[8] T. Lin, J. Ophir, and G. Potter, "Frequency-dependent ultrasonic differentiation of normal and diffusely diseased liver," *J. Acoust. Soc. Amer.*, vol. 82, no. 4, pp. 1131–1138, Oct. 1987.

[9] S. A. Goss, R. L. Johnston, and F. Dunn, "Comprehensive compilation of empirical ultrasonic properties of mammalian tissues," *J. Acoust. Soc. Amer.*, vol. 64, pp. 423–457, 1978.

[10] ——, "Compilation of empirical ultrasonic properties of mammalian tissues II," *J. Acoust. Soc. Amer.*, vol. 68, pp. 93–108, 1980.

[11] J. M. M. Pinkerton, "The absorption of ultrasonic waves in liquids and its relation to molecular constituents," in *Proc. Phys. Soc. London*, vol. B62, 1949, pp. 129–133.