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A theoretical study of the scattering of ultrasound from blood

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A theoretical treatment of the scattering of ultrasound from blood is given, assuming that the blood behaves essentially as a continuum. The scattering then arises from fluctuations in the mass density and compressibility of the blood, which is caused by a fluctuation in the red cell concentration. An expression for the received signal in ultrasonic blood velocity measurements is given. The stochastic properties of the signal are discussed with reference to the information content about the velocity field of the blood. Since the signal is Gaussian, all available information is contained in the power spectrum, which is a blurred approximation to the velocity distribution in the region of observation.

1. Introduction

In recent years there has been considerable interest in using the Doppler shift of backscattered ultrasound from blood to study the blood velocity in arteries. Blood is a mixture of formed elements in a surrounding liquid, the plasma. The scattering arises mainly from the red cells (erythrocytes) which by far outnumber the rest of the formed elements, both in quantity and volume (Burton 1965, Shung, Siegelmann and Reid 1976).

Brody and Meindl (1974) and Newhouse, Bendick and Varner (1976) have given theoretical treatments of the received scattered signal in ultrasonic blood velocity measurements, using the assumption that the cells are independent scatterers.

In normal blood the volume concentration of cells is approximately 45%. The closest packing that may be obtained without deforming the cells is 58% (Burton 1965). This means that almost all of the cells will be in contact with one or more of the others at each instant of time. This strongly indicates that the assumption of non-interacting scatterers will not hold. This has also been confirmed by experiments of Shung *et al.* (1976).

The compressibility and mass density of the cells differ only slightly from those of the plasma. The scattering is therefore weak and can be approximated by first-order scattering in which waves which are scattered twice or more are neglected. To avoid the problem of interaction between the cells we treat the blood as an isotropic continuum. The scattering then arises from fluctuations in the compressibility, $\kappa(\boldsymbol{r}, t)$, and the mass density, $\rho(\boldsymbol{r}, t)$, of the continuum. By this the cell interaction is contained in ρ and κ which simplifies the problem.

Each red cell has a volume of approximately 90 μm (Burton 1965) while the ultrasonic wavelength at 2 MHz (an actual frequency) is 750 μm. A blood element with each side equal to $\lambda/10$ will contain more than 1000 cells in the average (Shung *et al.* 1976).

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Concerning wave-motion this element will be a point. Since the cells are strongly interacting, this fortifies the assumption of our continuum approach.

The fluctuation of κ and ρ is caused by the fluctuation in the cell concentration $n_T(r, t)$. The correlation length of n_T will be so small compared to the wavelength that it can be considered δ-correlated in space. By this the spectral properties of the scattered wave can be computed. Due to the δ-correlation of n_T, these will basically be the same as for non-interacting particles. The interaction will mainly affect the total power of the scattered wave.

2. Stochastic properties of cell concentration

Let

$$n_T(r, t) = n_0(r, t) + n(r, t) \tag{1}$$

where $n_0 = \langle n \rangle$ is the local average of n_T and n is the fluctuation of n_T around n_0. $\langle\ \rangle$ denotes ensemble average. The local average, n_0, might be a function of space due to, e.g., axial migration in the blood vessels, and also of time for time-varying velocity fields. However, the variation in n_0 will be slow compared to that of n.

In the following we shall need the correlation function of n in space and time. We will not need the details of the function over distances below $\lambda/10$. We note that the interaction between the cells is not likely to extend more than a couple of cell diameters, and the volume of $(\lambda/10)^3$ contains a large amount of cells. Therefore we can assume n to be approximately δ-correlated in space for a fixed time, i.e.

$$\langle n(r, t) n(r+\xi, t) \rangle = \langle n^2(r, t) \rangle \delta(\xi) \tag{2}$$

For the correlation function at two different times two factors are of importance:

(i) Diffusion.
(ii) The convection of the cells in the velocity field $v(r, t)$.

Diffusion in blood is a slow process and it is, therefore, evident that convection will dominate the time change of n in flowing blood. If n is strictly δ-correlated in space for a fixed time, convection will give the following correlation function.

$$\langle n(r, t) n(r+\xi, t+\tau) \rangle = \langle n^2(r, t) \rangle \delta[\xi - \zeta(r, t, \tau)] \tag{3}$$

where $r+\zeta$ is the position of the fluid element at time $t+\tau$ which at time t had the position. r. However, since δ-correlation is an approximation only, variations of the velocity field over the correlation length may cause violations of eqn. (3).

As an example, consider parabolic flow in a circular tube with radius a. Take two points on the radius, one $a/2$ and the other $a/2+\lambda/10$ from the tube axis. When the inner point has moved a distance L, the outer point has lagged $(\lambda/10) \cdot (2L/a)$ behind the inner point. We are interested in $L \lesssim a/2$ which gives a distance between the points of $\sqrt{2}\lambda/10$ which still indicates validity of eqn. (3). Thus velocity gradients will broaden the correlation peak. For flows which are not too turbulent, the δ-correlation still seems a good approximation for actual values of τ (< 100 ms). The effect of turbulence becomes more clear, perhaps, if we look at the motion of a single scatterer. If the velocity vector of a single scatterer changes drastically as it passes through the range cell, turbulence will broaden the Doppler spectrum received from that scatterer.

$\langle n^2 \rangle$ will depend on the velocity field and the *interaction* between the cells. *Turbulence* will increase $\langle n^2 \rangle$ since local accelerations in the velocity field will cause a separation between cells and plasma due to their different mass densities.

The cells will both attract and repel each other. The *attraction* has a complex nature, and reveals itself in the formation of rouleaux (Burton 1965). However, this bondage is so weak that the rouleaux break down into individual cells when exposed to the shear gradients of normal velocity fields. The repelling force is mainly mechanical in origin and occurs as a reaction when the cells get in contact and are pushed towards each other.

For non-interacting particles in a fluid at rest $\langle n^2 \rangle = n_0$ (Landau and Lifshits 1963). When the cells interact, *attraction* will increase the fluctuation while *repulsion* will decrease the fluctuation. If the blood is at rest for a sufficiently long time that rouleaux may form, we may have $\langle n^2 \rangle > n_0$. Under moderate flow velocities where the rouleaux have broken down, we have $\langle n^2 \rangle < n_0$, while turbulence will increase $\langle n^2 \rangle$ due to the separation described above.

3. Wave motion in blood

Let $p(\boldsymbol{r}, t)$ be the wave pressure at position $\boldsymbol{r}$ and time t, and $\boldsymbol{u}(\boldsymbol{r}, t)$ be the velocity of displacement of a fluid located at $\boldsymbol{r}$. According to the above discussion, we assume that adiabatic elastic compressions of the blood can be described by the law for single phase fluids

$$\frac{\partial p}{\partial t} = -\frac{1}{\kappa} \nabla \boldsymbol{u} \tag{4}$$

The momentum equation for a fluid element will be

$$\frac{\partial (\rho \boldsymbol{u})}{\partial t} = -\nabla p \tag{5}$$

where the convective part of the acceleration has been neglected since it is small. We split both ρ and κ into their averages plus a fluctuation term

$$\left.\begin{aligned} \kappa(\boldsymbol{r}, t) &= \kappa_0(\boldsymbol{r}, t) + \kappa_1(\boldsymbol{r}, t) \\ \rho(\boldsymbol{r}, t) &= \rho_0(\boldsymbol{r}, t) + \rho_1(\boldsymbol{r}, t) \end{aligned}\right\} \tag{6}$$

where $\kappa_0 = \langle \kappa \rangle$ and $\rho_0 = \langle \rho \rangle$.

We further assume that

$$\left.\begin{aligned} \kappa_1(\boldsymbol{r}, t) &= \frac{d\kappa}{dn_0} n(\boldsymbol{r}, t) \\ \rho_1(\boldsymbol{r}, t) &= \frac{d\rho}{dn_0} n(\boldsymbol{r}, t) \end{aligned}\right\} \tag{7}$$

In wave motion $\boldsymbol{u}$ will change much faster than ρ in eqn. (5) by which ρ may be taken outside the differentiation. Combining eqn. (4) and eqn. (5) we obtain

$$\nabla \left(\frac{1}{\rho} \nabla p \right) - \kappa \frac{\partial^2 p}{\partial t^2} = 0 \tag{8}$$

By substituting eqn. (6) into eqn. (8) and rearranging terms, we obtain

$$\left.\begin{aligned} \nabla^2 p - \frac{1}{c^2}\cdot\frac{\partial^2 p}{\partial t^2} &= \frac{1}{c^2}\cdot\frac{\kappa_1}{\kappa_0}\cdot\frac{\partial^2 p}{\partial t^2} + \nabla\left(\frac{\rho_1}{\rho}\nabla p\right) \\ c^2 &= \frac{1}{\rho_0\kappa_0} \end{aligned}\right\} \tag{9}$$

Possible variations in the wave velocity c due to cell migration are small and may be neglected. The coefficients κ_1/κ_0 and ρ_1/ρ are small quantities and eqn. (9) may be regarded as an inhomogeneous wave equation with a weak source-term given by the wave itself. This is an appropriate form to be treated by standard methods used in scattering theory (Morse and Ingard 1968).

Suppose there is an incident wave $p_0(\boldsymbol{r}, t)$, and write the total field as $p = p_0 + p_1 + p_2 + \ldots$ The partial term p_{i+1} is the solution of eqn. (9) when p_i is inserted on the right-hand side. The zero order term, p_0, will propagate by a homogeneous wave equation with wave velocity c, be scattered to produce p_1, which in turn is scattered to produce p_2, etc. Since the scattering is weak, the magnitude of the terms will decrease rapidly, and p_1, which is the first-order scattering term, will be a good approximation to the scattered field (Born approximation).

We shall study the scattered field for a time harmonic incident wave

$$p_0(\boldsymbol{r}, t) = \mathrm{Re}\,\{\hat{p}_0(\boldsymbol{r})\exp(i\omega_0 t)\} \tag{10}$$

Since κ_1 and ρ_1 in eqn. (9) are time dependent (blood motion), the scattered field will not contain a single frequency. We therefore assume a solution of the form

$$p(\boldsymbol{r}, t) = \mathrm{Re}\,\{\hat{p}(\boldsymbol{r}, t)\exp(i\omega_0 t)\} \tag{11}$$

Inserting this into eqn. (9) and neglecting the time derivative of $\hat{p}$ compared to that of $\exp(i\omega_0 t)$, we obtain

$$\nabla^2\hat{p} + k_0^2\,\hat{p} = -k_0^2\,\gamma_\kappa n\hat{p} + \gamma_\rho\nabla(n\nabla\hat{p}) \tag{12}$$

where $k_0 = \omega_0/c$, $\gamma_\kappa = \kappa_0^{-1}\,d\kappa/dn_0$ and $\gamma_\rho = \rho_0^{-1}\,d\rho/dn_0$. Equation (12) is studied in Morse (1968). The Born approximation to the scattered field may be written

$$\left.\begin{aligned} \hat{p}_s(\boldsymbol{r}, t) &= \int_R d^3\xi\{k_0^2\,\gamma_\kappa G(\boldsymbol{r}-\boldsymbol{\xi})\hat{p}_0(\boldsymbol{\xi}) + \gamma_\rho\nabla_\xi G(\boldsymbol{r}-\boldsymbol{\xi})\nabla_\xi\hat{p}_0(\boldsymbol{\xi})\}n(\boldsymbol{\xi}, t) \\ G(\boldsymbol{r}) &= \frac{\exp(-ik_0|\boldsymbol{r}|)}{4\pi|\boldsymbol{r}|} \end{aligned}\right\} \tag{13}$$

R is the region containing scatterers. The first term under the integral is a monopole source term which arises from the fluctuation in the compressibility, while the fluctuation in the mass density produces the second term, which is a dipole term.

4. Scattering of a plane wave

Let the incident wave be given by

$$\hat{p}_0(\boldsymbol{r}) = A\exp(-i\boldsymbol{k}_0\boldsymbol{r}) \tag{14}$$

where A is the amplitude and $\boldsymbol{k}_0$ the incident wave vector. We study the far field (i.e. $\xi \in R \Rightarrow \xi/r \ll 1$) which permits the approximation

$$\left.\begin{aligned} G(\boldsymbol{r}-\boldsymbol{\xi}) &= \frac{\exp\left[-i(k_0 r - \boldsymbol{k}_s \boldsymbol{\xi})\right]}{4\pi r} \\ \boldsymbol{k}_s &= k_0 \frac{\boldsymbol{r}}{r} = \frac{\omega_0}{c} \cdot \frac{\boldsymbol{r}}{r} \end{aligned}\right\} \tag{15}$$

Equation (13) then takes the form

$$\hat{p}_s(\boldsymbol{r}, t) = \frac{A}{4\pi} \cdot \frac{{\omega_0}^2}{c^2} \cdot \frac{\exp(-ik_0 r)}{r} \left\{\gamma_\kappa + \gamma_\rho \frac{\boldsymbol{k}_s \cdot \boldsymbol{k}_0}{{k_0}^2}\right\} \int_R d^3\xi n(\boldsymbol{\xi}, t) \exp\left[i(\boldsymbol{k}_s - \boldsymbol{k}_0)\boldsymbol{\xi}\right] \tag{16}$$

The volume integral is the space Fourier transform of n. We thus get interaction between the incident wave and the partial wave of the scattering fluctuation which satisfies the Bragg condition of interference. This illustrates a well known result in incoherent scattering theory (Morse and Ingard 1968).

The differential scattering cross-section $\sigma(\theta, \phi)$ per unit volume is defined by

$$\sigma(\theta, \phi) = \frac{\langle dP \rangle}{d\Omega} = \frac{\langle I \rangle r^2}{I_0 V}$$

where $\langle dP \rangle$ is the relative power scattered through the differential solid angle $d\Omega$ $\langle I \rangle$ is the intensity scattered in the direction (θ, ϕ) and I_0 is the total intensity incident on R. V is the volume of R and θ is the angle between $\boldsymbol{k}_0$ and $\boldsymbol{k}_s$. This gives

$$\sigma(\theta, \phi) = \frac{1}{16\pi^2} \frac{{\omega_0}^4}{c^4} \{\gamma_\kappa + \gamma_\rho \cos\theta\}^2 \langle n^2 \rangle \tag{17}$$

where

$$\langle n^2 \rangle V = \int_R d^3\xi \langle n^2(\boldsymbol{\xi}, t) \rangle$$

The scattering intensity is space dependent and proportional to the frequency in the fourth power. For a suspension of solid elements in a liquid as blood is, the total mass density will depend on the cell concentration as

$$\rho_0(n_0) = \rho_p + n_0(\rho_c - \rho_p) \tag{18}$$

where ρ_p and ρ_c are the mass densities of plasma and cells respectively. By measuring the wave velocity, the compressibility may be calculated as $\kappa_0 = c^{-2}{\rho_0}^{-1}$. This has been done for several values of n (Angelsen 1975). Using $\rho_c = 1{\cdot}1 \times 10^3$ and $\rho_p = 1{\cdot}03 \times 10^3$ kg/m^3 (Burton 1965), we obtain

$$\left.\begin{aligned} \gamma_\rho &\approx 0{\cdot}065 \\ \gamma_\kappa &\approx -0{\cdot}2 \end{aligned}\right\} \tag{19}$$

This gives an angular dependency of the scattering cross-section as shown in Fig. 1.

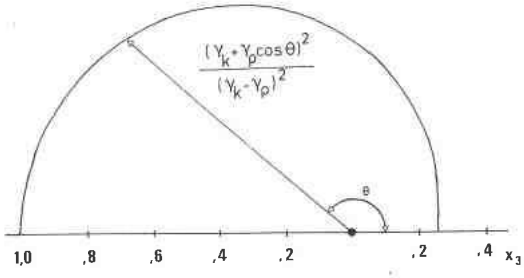

Figure 1. Polar diagram for the differential scattering cross-section of a plane wave from blood. HCT ~ 40–50%.

5. Receiver output in ultrasonic blood velocity measurements

In ultrasonic blood velocity measurements the blood vessel will normally be in the near field region of the transducers. The complex pressure amplitude of a single frequency transmitted wave may be written

$$\hat{p}_T(\omega_0, \boldsymbol{r}) = A_T(\omega_0, \boldsymbol{r}) \exp\left[-i\phi_T(\omega_0, \boldsymbol{r})\right] \tag{20}$$

where ω_0 is the angular frequency of the wave. A_T and ϕ_T are real. If the transducers and distances are such that the plane wave approximation may be performed, $\phi_T = \boldsymbol{k}_T \boldsymbol{r} + \text{const}$, $\boldsymbol{k}_T = \boldsymbol{n}_T \omega_0 / c$ where $\boldsymbol{n}_T$ is the unit normal vector to the transmitting transducer surface.

The scattered field may from eqn. (12) be represented by the following source density

$$-k_0^2 \gamma_\kappa \hat{p}_T(\boldsymbol{r}) n(\boldsymbol{r}, t) + \gamma_\rho \nabla[n(\boldsymbol{r}, t) \nabla \hat{p}_T(\boldsymbol{r})] \tag{21}$$

By reciprocity, a monopole source density with angular frequency ω_0 and magnitude $m(\boldsymbol{r}, t)$ in a region R will produce the following output of a receiving transducer

$$e(t) = -\operatorname{Re}\left\{\alpha \exp(i\omega_0 t) \int_R d^3\xi m(\xi, t) \hat{p}_R(\xi)\right\} \tag{22}$$

α is a complex constant of proportionality and $\hat{p}_R(\xi)$ is the complex amplitude of the pressure wave when the receiving transducer is excited with a voltage of angular frequency ω_0.

$$\hat{p}_R(\omega_0, \boldsymbol{r}) = A_R(\omega_0, \boldsymbol{r}) \exp\left[-i\phi_R(\omega_0, \boldsymbol{r})\right] \tag{23}$$

The time variation of m is supposed to be slow compared to the wave motion.

In continuous wave ultrasonic blood velocity measurements we then get the following expression for the signal from the receiving transducer

$$e(t) = \operatorname{Re}\left\{\alpha \exp(i\omega_0 t) \int_R d^3\xi \{k_0^2 \gamma_\kappa \hat{p}_T(\xi) \hat{p}_R(\xi) + \gamma_\rho \nabla p_T(\xi) \nabla \hat{p}_R(\xi)\} n(\xi, t)\right\} \tag{24}$$

where we have performed integration by parts on the last term. The above expression may be written in the following form

$$e(t) = \operatorname{Re}\{\exp(i\omega_0 t) a \int d^3\xi R(\xi) \exp[i\psi(\xi)] n(\xi, t)\} \tag{25}$$

where $a=|\alpha|$ and

$R=|A|$

$\psi=-\phi_T-\phi_R+\angle\alpha+\angle A$

$A=A_TA_R\{k_0{}^2\,\gamma_\kappa-\gamma_\rho\nabla\phi_T\nabla\phi_R\}+$small terms

By defining $R(\xi)\equiv 0$ for $\xi\notin R$, the integration in eqn. (23) may be extended to whole space.

When a pulsed wave Doppler meter is used, a distribution of frequencies is transmitted. As shown above the scattered intensity is proportional to the frequency in the fourth power. However, the relative width of the frequency band is generally so small (200 kHz/2 MHz) that the scattering may be considered to be frequency independent across this band. The effect of pulsing may therefore be incorporated by multiplying the transmitting transducer field pattern $\hat{p}_T$ by a window function $S(\xi)$ which moves with the transmitted pulse. Let z be the distance between the transducer and the range cell along the axis of the transducer, i.e. $z=|z|=\frac{1}{2}ct$. t is the elapsed time between the pulse transmission and reception. Then we obtain the following expression for the received signal from the kth pulse

$$\left.\begin{aligned} e_k(z)&=\mathrm{Re}\,\{\hat{x}_k(z)\exp(i2k_0z)\}\\ \hat{x}_k(z)&=a\int d^3\xi R_p(\xi,z)\exp[i\psi(\xi)]n\left\{\xi,\frac{z}{c}+kT_s\right\}\\ R_p(\xi,z)&=R(\xi)S(\xi-z)\end{aligned}\right\}\qquad(26)$$

$S(\xi)$ is the normalized envelope of the received pulse from a point scattered and T_s is the pulse repetition period. In the plane wave approximation

$$\left.\begin{aligned}\psi(\xi)&=q\xi+\text{const}\\ q&=-(k_T+k_R)=-\frac{\omega_0}{c}(n_T+n_R)\end{aligned}\right\}\qquad(27)$$

where n_T and n_R are the unit normal vectors of the transmitting and receiving transducer surfaces respectively.

6. Statistical properties of the signal

We study the properties of $\hat{x}$ given by eqn. (26) and the result may easily be applied to the continuous wave case. It is obtained as a linear operation on the stochastic process $n(\xi,t)$. It is reasonable to assume n to be Gaussian since it is the result of a large number of mainly independent events (the interaction between the cells extends for a few cell diameters only). By the linear operation $\hat{x}$ will also be Gaussian. Even if n were not Gaussian $\hat{x}$ could be approximated by a Gaussian process since it is obtained as a weighted average of an approximately δ-correlated process (Central limit theorem). $\hat{x}$ has zero mean since $\langle n\rangle=0$.

The velocity field of the blood is coded into $\hat{x}$ through the stochastic process n. All information of the velocity field available in $\hat{x}$ is therefore contained in the stochastic properties of $\hat{x}$. This is determined by the second moment of $\hat{x}$, i.e. its autocorrelation function, since $\hat{x}$ is zero mean Gaussian. For time invariant velocity fields, $\hat{x}$ will be stationary and its autocorrelation function is the inverse Fourier transform of the power spectrum of $\hat{x}$.

In the following we restrict ourselves to time invariant velocity fields and plane waves. The autocorrelation function for $\hat{x}$ is then

$$\begin{aligned} R_{\hat{x}}(k, l; z_1, z_2) &= \langle \hat{x}_k^*(z_1)\hat{x}_l(z_2)\rangle \\ &= a^2 \int d^3\xi_1 \int d^3\xi_2 R_p(\xi_1, z_1)R_p(\xi_2, z_2) \exp\left(i[\psi(\xi_2)-\psi(\xi_1)]\right) \\ &\quad \times \left\langle n\left(\xi_1, \frac{z_1}{c}+kT_s\right) n\left(\xi_2, \frac{z_2}{c}+lT_s\right)\right\rangle \end{aligned} \tag{28}$$

which by the use of eqn. (3) may be integrated to

$$\left.\begin{aligned} R_{\hat{x}}(k, l; z_1, z_2) &= a^2 \int d^3\xi R_p(\xi, z_1)R_p(\zeta, z_2) \\ &\quad \times \exp\left(iqv(\xi)\left[\!\left[(l-k)T_s-\frac{1}{c}(z_2-z_1)\right]\!\right]\right)\langle n^2(\xi)\rangle \\ \zeta &= \xi+v(\xi)\left[\!\left[(l-k)T_s-\frac{1}{c}(z_2-z_1)\right]\!\right] \end{aligned}\right\} \tag{29}$$

We may note that it will not be possible to deduce a general velocity field from $R_{\hat{x}}$. The velocity enters in $R_{\hat{x}}$ via ζ in R_p and in qv. The term $qv(l-k)T_s$ is the Doppler oscillation and will have the most significant influence on $R_{\hat{x}}$, while $R_{\hat{x}}$ is much less sensitive to its other dependencies on v.

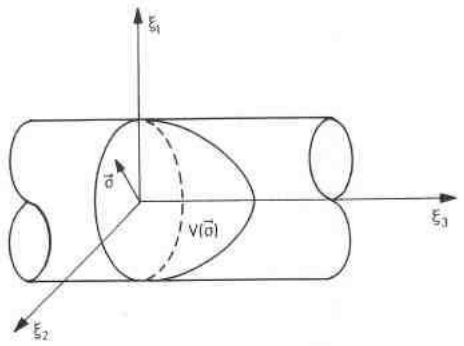

Figure 2. Coordinate system for velocity field in a tube, eqn. (30).

In the common pulsed Doppler measurements $z_1=z_2$. We orient the ξ_3-axis along v and denote the position in the ξ_1, ξ_2 plane by σ (Fig. 2). We also change $(l-k)T_s \to \tau$ to give $R_{\hat{x}}$ for the continuous Doppler signal from a certain depth.

$$\left.\begin{aligned} R_{\hat{x}}(\tau) &= a^2 \int d^2\sigma F[\sigma, v(\sigma)\tau]\langle n^2(\sigma)\rangle \exp\left[iqv(\sigma)\tau\right] \\ F[\sigma, v(\sigma)\tau] &= \int d\xi_3 R_p[\xi, z]R_p[\xi+v(\sigma)\tau, z] \end{aligned}\right\} \tag{30}$$

The fluctuation $\langle n^2\rangle$ is a function of σ only, since all points along a streamline in rectilinear flow have identical properties.

The power spectrum of $\hat{x}$ is obtained as the Fourier transform of $R_{\hat{x}}$

$$\left.\begin{aligned} G_{\hat{x}}(\omega) &= \int_{-\infty}^{\infty} d\tau R_{\hat{x}}(\tau) \exp\left(-i\omega\tau\right) = a^2 \int d^2\sigma \langle n^2(\sigma)\rangle \hat{F}\,\frac{\left[\!\left[\dfrac{\sigma,\ \omega-qv(\sigma)}{v\sigma}\right]\!\right]}{v(\sigma)} \\ \hat{F}(\sigma, \omega) &= \int_{-\infty}^{\infty} d\tau F(\sigma, \tau) \exp\left(-i\omega\tau\right) \end{aligned}\right\} \tag{31}$$

The physical interpretation of this expression is that a fluctuation travelling along a streamline through $\boldsymbol{\sigma}$ gives a burst of oscillations in the Doppler signal as it passes through the range cell. The power spectrum of the burst is a frequency band given by $\hat{F}$, centred around the Doppler frequency $\omega = \boldsymbol{q}\boldsymbol{v}(\boldsymbol{\sigma})$. The width of the band is inversely proportional to the duration of the burst in time and thus proportional to $v(\boldsymbol{\sigma})$. The average power is proportional to $\langle n^2(\boldsymbol{\sigma})\rangle$.

When the transit time is sufficiently large, we may use the following approximation (Angelsen 1975)

$$\frac{1}{v}\hat{F}\left[\boldsymbol{\sigma}, \frac{\omega - \boldsymbol{q}\boldsymbol{v}}{v}\right] \approx 2\pi A^2 L(\boldsymbol{\sigma})\delta[\omega - \boldsymbol{q}\boldsymbol{v}(\boldsymbol{\sigma})] \tag{32}$$

where

$$A^2 L(\boldsymbol{\sigma}) = \int d\xi_3 R_p{}^2 [\boldsymbol{\xi}, z]$$
$$A = R_p(z, z)$$

In this expression L is an equivalent transit length. This gives the following approximation to the spectrum

$$\tilde{G}_{\hat{x}}(\omega) = 2\pi A^2 a^2 \int d^2\,\sigma \langle n^2(\boldsymbol{\sigma})\rangle L(\boldsymbol{\sigma})\delta[\omega - \boldsymbol{q}\boldsymbol{v}(\boldsymbol{\sigma})] \tag{33}$$

The above equation may be integrated to give

$$\tilde{G}_{\hat{x}}(\omega) = 2\pi A^2\, a^2 \int_{\Gamma(\omega)} dp\, \frac{L(\boldsymbol{\sigma})\langle n^2(\boldsymbol{\sigma})\rangle}{|\nabla \boldsymbol{q}\boldsymbol{v}(\boldsymbol{\sigma})|} \tag{34}$$

$\Gamma(\omega)$ is the family of curves in the $\boldsymbol{\sigma}$-plane satisfying

$$\omega = \boldsymbol{q}\boldsymbol{v}(\boldsymbol{\sigma}) \tag{35}$$

and $\hat{p}$ is the arc length parameter along Γ. If $\hat{F}$ is the same for all $\boldsymbol{\sigma}$ satisfying eqn. (35) for fixed ω, $G_{\hat{x}}$ may be obtained from $\tilde{G}_{\hat{x}}$ by a convolution

$$G_{\hat{x}}(\omega) = \int_{-\infty}^{\infty} dw\, \frac{q_v}{w} \hat{f}\left[w, \frac{q_v(\omega - w)}{w}\right] \tilde{G}_{\hat{x}}(w) \tag{36}$$

where

$$\frac{q_v}{w}\hat{f}\left[w, \frac{q_v(\omega - w)}{w}\right] = \frac{q_v}{w} \frac{\hat{F}\left[\boldsymbol{\sigma}(w), \dfrac{q_v(\omega - w)}{w}\right]}{\displaystyle\int_{-\infty}^{\infty} d\omega \hat{F}[\boldsymbol{\sigma}(w), \omega]} \tag{37}$$

is the spectral band obtained from the scatterers with velocity $v = w/q_v$, normalized to unit power. We have defined q_v as the component of $\boldsymbol{q}$ along $\boldsymbol{v}$. A convolution formula like the above cannot be obtained in the general case.

As an example we calculate $\tilde{G}_{\hat{x}}$ for the following velocity profile in a circular tube of radius a

$$v(\sigma) = v_0\left[1 - \frac{\sigma^p}{a^p}\right] \quad 0 < \sigma < a \tag{38}$$

σ is the radial position coordinate. $p=2$ gives parabolic flow, while $p\to\infty$ gives plug flow. We assume that the region of observation is uniform with length L across the tube and that $\langle n^2\rangle=$const. Normalizing to unit power we obtain from eqn. (34), $\omega_0=qv_0$

$$\tilde{G}_{\xi}(\omega)=\begin{cases}\dfrac{4\pi}{p\omega_0\left(1-\dfrac{\omega}{\omega_0}\right)^{1-(2/p)}} & \omega\in[0,\,\omega_0] \\ 0 & \text{else}\end{cases} \tag{39}$$

The velocity profiles and the spectra are shown in Figs. 3 and 4. The singularities in $\tilde{G}_{\xi}$ will not be found in G_{ξ} due to the convolution in eqn. (36). These spectra are the same as those obtained by Roevros (1974) using a simplified analysis on independent scatterers.

7. Discussion

The received signal is the projection of the fluctuation $n(\xi, t)$ onto the kernel $R_p \exp(i\psi)$, eqn. (26). For the plane wave approximation, this extracts the Fourier component of n which satisfies the Bragg condition of reflection, § 4. We note that it is the fluctuation rather than the total concentration which produces the scattering. The reason for this is that if n_T were constant, we could always find a scatterer which

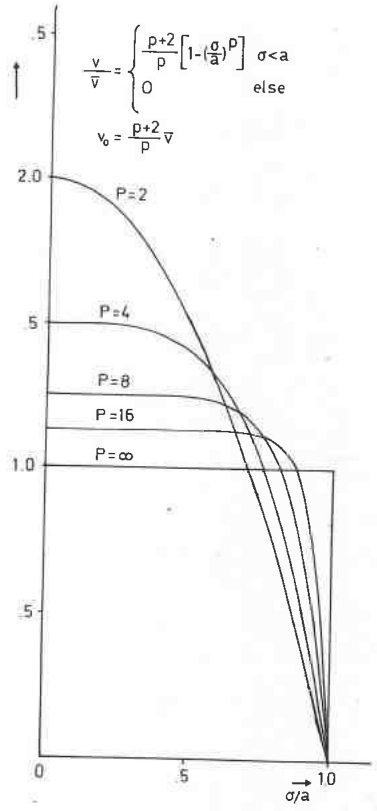

Figure 3. Blunt velocity profiles in a circular tube. $\bar{v}$ is the mean velocity across the vessel lumen.

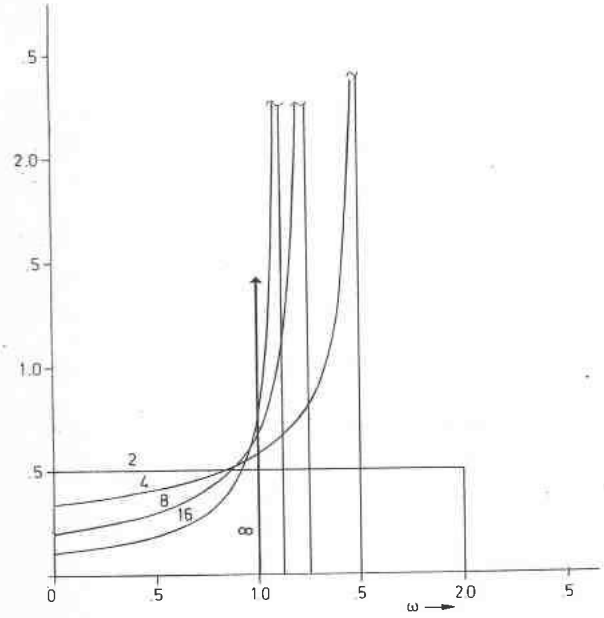

Figure 4. Approximate power spectra, $\tilde{G}_{\dot{x}}(\omega)$, for the Doppler signal from the blunt velocity profiles. $\bar{\omega}$ is the mean angular frequency of the spectrum.

cancels the signal from another scatterer. Thus it is the variation in n_T which causes scattering. This is a general situation in incoherent scattering.

The continuum approach has resolved the problem of interacting scatterers. Since the wavelength is long compared to the correlation length of n, δ-correlation of n can be assumed, and thus the effect of interaction is contained in $\langle n^2 \rangle$. In normal flowing blood, we will have $\langle n^2 \rangle = n_0$, the value for non-interacting scatterers. This has been experimentally confirmed by Shung *et al.* (1976). Turbulence will increase $\langle n^2 \rangle$ due to the difference in mass density between cells and plasma which causes separation. This will increase the scattering, which is confirmed by *in vivo* measurements. The scattered signal is generally strong from turbulent jets as in mitral and aortic stenosis. The amount the scattering is increased is left for experimental investigation.

The effect of turbulence upon scattering from a mixture of solid elements in a liquid such as blood, is different than for a pure liquid. For a pure fluid the interaction between flow and a wave arises from two effects. The one is the compression of the fluid caused by the turbulence. This modulates the mass density and compressibility of the medium. The other arises when the convective velocity becomes comparable to the sound velocity, by which the convective acceleration in eqn. (5) cannot be neglected.

The first effect is negligible in normal turbulence for practically incompressible fluids such as plasma. When particles are present in the liquid, however, we get a similar modulation of the mass density and compressibility by the flow, as discussed above.

Apart from increasing the scattering, turbulence will also affect the Doppler spectrum. This can be viewed as a violation of the δ-correlation property in eqn. (3). As discussed in § 2, a more illustrative way to describe the effect of turbulence is to follow a single scatterer. If this has a straight path through the range cell, we will

obtain a spectral band from the scatterer given by $\hat{F}$ in eqn. (31). If the velocity component along $\boldsymbol{q}$, v_q changes during the transition, additional broadening of F will occur. The amount of broadening will depend on $\langle \Delta v_q^2 \rangle / \langle v_q \rangle^2$, where Δv_q is the variation in the velocity of the scatterer.

Assume that the signal from the scatterer has the form

$$\hat{x}(t) = a(t) \exp(iw(t)t)$$

$$w(t) = \boldsymbol{q} \cdot \boldsymbol{v}(t)$$

The Fourier-transform of this signal is

$$F(\omega) = \int_{-\infty}^{\infty} dt\, a(t) \exp(iw(t)t) \exp(-i\omega t)$$

and

$$\bar{\omega} = \frac{\int_{-\infty}^{\infty} d\omega\, \omega |\hat{F}(\omega)|^2}{\int_{-\infty}^{\infty} d\omega |\hat{F}(\omega)|^2} = \frac{\int_{-\infty}^{\infty} dt\, a(t) \exp(iw(t)t) \frac{d}{dt}[a(t) \exp(iw(t)t)]}{\int_{-\infty}^{\infty} dt\, a^2(t)}$$

$$\overline{\omega^2} = \frac{\int_{-\infty}^{\infty} d\omega\, \omega^2 |\hat{F}(\omega)|^2}{\int_{-\infty}^{\infty} d\omega |\hat{F}(\omega)|^2} = \frac{\int_{-\infty}^{\infty} dt \left[\frac{d}{dt} a(t) \exp[iw(t)]\right]^2}{\int_{-\infty}^{\infty} dt\, a^2(t)}$$

If the transit time broadening of the spectrum can be neglected and $\dot{w} \ll w$, we obtain

$$\langle \bar{\omega} \rangle \approx \langle w \rangle$$

$$\langle \overline{\omega^2} \rangle \approx \langle w^2 \rangle$$

where ensemble averaging over different $w(t)$ is performed.

The scattering intensity is proportional to the frequency in the fourth power, which is also confirmed by the experiments of Shung *et al.* (1976).

The value of γ_κ given by eqn. (19) is greater than $-0{\cdot}166$ which is obtained for a single scatterer, Shung *et al.* (1977). This is reasonable since the wave in blood observes an average compressibility which is less than that for pure plasma. In their experiments Shung *et al.* have used a cell concentration of 10%. This is at the upper limit of what would give independent scatterers, and a smaller value of γ than that obtained in whole blood should be expected. (In addition the blood sample in our measurement is likely to have been different from theirs.) The scattering cross-section of blood obtained here will thus have a smaller space variation than that obtained by Shung *et al.*

The general form for the power spectrum of the received signal for rectilinear time invariant flow and plane waves is given in eqn. (31). Actually the plane wave assumption, is an approximation since the beams have finite extent, and thus are composed of a distribution of plane waves with different $\boldsymbol{q}$-vectors. In our approximation, the finite extent is taken care of by the amplitude factor R_p. This gives a finite transit time of a fluid element through the region of observation which in turn gives the line width of $\hat{F}$, eqn. (31). $\tilde{G}_{\hat{x}}$, eqn. (33), is the distribution of the velocity component along $\boldsymbol{q}$, $\omega = \boldsymbol{q}v$, in the region of observation, weighted with

$\langle n^2 \rangle L$. $\tilde{G}_{\hat{x}}$ will thus be a blurred version of this velocity distribution, the blurring caused by the finite transit time.

For small transducers and by focussing when $\psi = \boldsymbol{q}\boldsymbol{\xi} + \text{const}$ is not a good assumption, additional broadening of the single scatterer line will occur.

All available information in $\hat{x}$ of the velocity field is contained in $G_{\hat{x}}$ (§ 6). The maximum information that can be extracted from ultrasonic blood velocity measurements is this blurred velocity distribution. By knowing $G_{\hat{x}}$ as a function of z and knowing the beam pattern, the velocity field can, however, be restored in simplified situations (Jorgensen, Campan and Baker 1973).

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