Ultrasonic Absorption and Reflection by Lung Tissue

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§ 1. Introduction

It is well known that ionizing radiation affects biological systems in a cumulative fashion (Blum 1959). Each exposure to the radiation increases the probability that pathologic growth will become apparent in the irradiated tissue structure in some specified time interval. X-ray procedures have been long employed as diagnostic tools but it is only relatively recently that an awareness of the effects of even very small doses has become generally known. The radiation dose can be reduced in lengthy diagnostic procedures such as cardiac catheterization or examination of the GI tract, for example, by the employment of fluoroscopic gastro-intestinal image intensifiers (Mallams and Miller 1956). However, it is of considerable importance to devise procedures to accomplish the purpose of X-ray methods whenever possible, further reducing hazards of ionizing radiation. In addition, a method which reveals soft tissue structure would have considerable application in medicine. One such procedure which suggests itself is the ultrasonic method of visualization of soft tissue structures developed by Howry (1957), Kikuchi, Uchida, Tanaka and Wagai (1957), and Wild and Reid (1957). To examine this possibility for lung tissue, which is especially difficult because of the presence of undissolved gas and which represents a tissue structure that annually receives considerable diagnostic radiation attention, experiments were carried out to determine the acoustical properties of excised dog lung.

§ 2. Experimental Method and Results

The tissue was used immediately after excision and was kept continuously in normal physiological saline. For the measurements, the tissue was encapsulated in a stainless steel frame having thin polyethylene windows (0·003 in. thick), the same diameter as that of the lung sample and sufficiently large to prevent interference with the acoustic beam. Care was taken not to compress the tissue from its original thickness and it is estimated that two-thirds of the residual air remained in the lung sample. The slight surface curvature of the major faces of the sample was eliminated by the confining capsule. The capsule was filled with degassed physiological saline to minimize possible interface difficulties which would result if gas bubbles were occulded on the tissue surface. The volume of the degassed saline was a small fraction (approximately 5%) of the volume of the lung tissue in the capsule so that only a small

fraction (a maximum of 0·2%) of the gas present within the lung could be dissolved by the degassed saline. This estimate assumes that the lung contains residual air.

A thermocouple probe (Fry and Fry 1954, Dunn and Fry 1957) was used as the detector of the pulsed acoustic energy. All measurements were carried out at a temperature of $35{\cdot}0 \pm 0{\cdot}5°C$. The acoustic source was a circular X-cut quartz plate $1\frac{1}{2}$ in. in diameter and having a fundamental thickness resonant frequency of 0·98 Mc/sec. The experimental configuration was arranged so that essentially plane waves of sound were propagated over the path lengths of interest in this study, as shown by investigation of the acoustic field distribution normal to the direction of propagation. Fig. 1 schematically illustrates the experimental arrangement. Determinations of the fraction of the incident acoustic energy

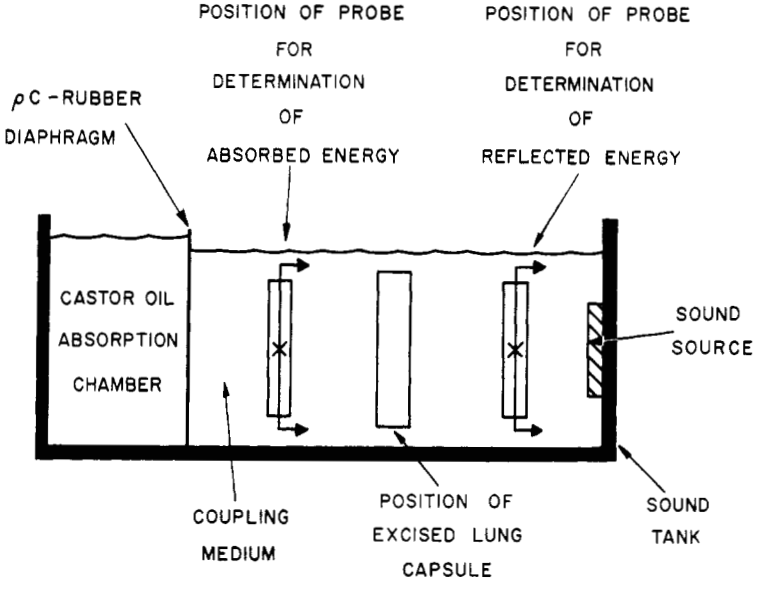

Fig. 1. Schematic illustration of the experimental arrangement.

reflected from the encapsulated lung and that propagated through the same sample were made. The coupling medium through which the ultrasound propagated from source through probe assembly and sample to the absorber was degassed physiological saline. The absorption chamber absorbed sound incident upon it to the extent that no reflected component interfered with the measurements.

For the determination of the fraction of the energy reflected at the lung-saline interface, the thermoelectric probe was placed between the encapsulated lung and the sound source. The lung sample was held in a fixed position while the probe was moved along the axis of propagation of the

acoustic field. The transient response of the probe to 0·1 sec pulses of sound was observed as a function of the probe position (0·1 mm intervals) and yields a well defined standing wave pattern. The ' standing wave ' pattern measured in this fashion can be used to deduce values of reflection coefficients by the use of an appropriate analysis.† In this way, the standing wave pattern existing in the lung–saline–probe system was determined quantitatively. Similar measurements of the standing wave pattern were made with the capsule filled with degassed saline and air to check the method. The experimentally determined reflection coefficient obtained in this fashion (with two samples of dog lung) is 0·71 implying that 50% of the incident energy is reflected at the lung-saline interface.

The density of normal lung tissue, determined from measurements of the weight and volume, is 0·40 g/cm^3. The sound propagation velocity in lung tissue can be obtained from the following relation relating the acoustic impedances (ρv), in the two media and the reflection coefficient, r (the ratio of the amplitudes of the reflected to incident acoustic pressure),

$$(\rho v)_{\text{lung}} = (\rho v)_{\text{saline}} \left(\frac{1-r}{1+r}\right),$$

where ρ is the density and v is the sound velocity. Using the values given above for the reflection coefficient and the density of lung tissue and $1{\cdot}53 \times 10^5$g/cm^2 sec for the acoustic impedance of saline, the sound propagation velocity in the lung tissue is found to be $6{\cdot}5 \times 10^4$cm/sec. The wavelength of the sound in the lung at 0·98 Mc/sec (the frequency at which the experiments were carrried out) is thus 0·66 mm.

The acoustic absorption coefficient per unit path length within lung tissue was determined by placing the encapsulated lung between the sound source and the thermoelectric probe (see fig. 1), i.e. the probe was used to investigate the intensity of the sound wave after it traversed the lung sample. The air-filled capsule was used in place of the encapsulated lung to demonstrate that within the limits of detectability (approximately one part in 10^5) no energy was received at the probe. This configuration was used to determine whether leakage of acoustic energy around the capsule would be of sufficient magnitude to interfere with the measurements. Values of the acoustic amplitude absorption coefficient per unit path length were determined from a knowledge of the energy reflected at the two lung-saline interfaces, the thickness of the sample, and the acoustic intensity detected by the probe in accordance with the relation

$$I_1 = I_0 \exp(-2\alpha l),$$

where I_0 and I_1 are, respectively, the acoustic intensitites at the lung-saline interfaces nearest to and farthest from the source, l is the thickness

† The ' standing wave ' ratio determined by the thermocouple is a function of both the acoustic pressure and particle velocity amplitudes and their spatial distributions.

of the lung sample, and α is the acoustic amplitude absorption coefficient per unit path length. The attentuation of the acoustic energy is sufficiently great such that energy reflected need not be considered. The data are tabulated in table 1. Fig. 2 shows the total absorption as a

Table 1. Tabulation of Experimental Data and Results

Sample	ρ (g/cm^3)	l (cm)	$(I_1/I_0)\times 10^3$	αl	α (cm^{-1})	$v\times 10^{-4}$ (cm/sec)
normal	0·40	0·76	0·68	3·6	4·8	6·5
normal	0·40	0·60	4·1	2·8	4·6	6·5
pneumonitis	0·76	0·99	1·08	3·4	3·5	3·4

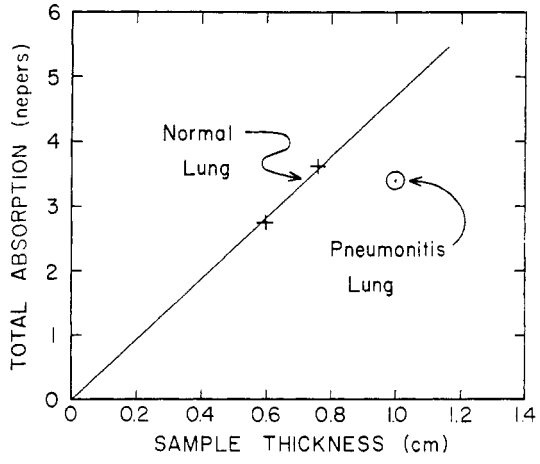

Fig. 2. Total acoustic amplitude absorption *vs* lung sample thickness.

function of sample thickness. The average value for the amplitude absorption coefficient for normal dog lung (excised) is found to be 4·7 cm^{-1}. One of the animals had a pneumonitis at the time the lung was excised. This resulted in an increased density by approximately a factor of two, a decrease in the velocity of propagation in the lung by the same factor, but a decrease in the absorption coefficient by only 25%.

§ 3. Acoustic Model of Lung Tissue

The unusually high absorption displayed by lung tissue requires comment. For example, the value obtained is more than an order of magnitude greater than that found for dry oxygen or nitrogen (Beranak 1949). In order to account for the absorption greatly in excess of that displayed by soft tissue, an acoustic model based upon the gross structure of lung tissue is postulated.

The structure of lung tissue pertinent to the present discussion can be described as follows (Best and Taylor 1945). The bronchioles branch and rebranch repeatedly as they proceed toward the periphery of the lung. The first branchings are approximately 1·5 mm in length and 0·4 mm in diameter. The terminal and respiratory bronchioles are approximately 0·5 mm in length, but the diameters are approximately the same as that of earlier branchings. That is, even though the bronchioles become shorter, virtually no decrease in diameter takes place as they pass toward the periphery. Each respiratory bronchiole gives rise to five or six alveolar ducts each of which in turn rebranch a number of times and each gives rise to approximately five alveolar sacs. The hemispherical protuberances in the walls of the sacs constitute the

Table 2. Tabulation of Quantities Appearing in Eqn. (2) and Associated Numerical Values

Quantity	Definition	Numerical Value
ω_0	$\frac{1}{R_0}\left\{\left(\frac{3\gamma P_0}{\rho}\right)\left(\frac{g}{\epsilon}\right)\right\}^{\frac{1}{2}}$, angular resonant frequency of bubble	
g	$1+\frac{2\sigma}{P_0 R_0}\left(1-\frac{1}{3h}\right)$	
ϵ	$1+\frac{3(\gamma-1)}{2\Phi R_0}\left\{1+\frac{3(\gamma-1)}{2\Phi R_0}\right\}$	
Φ	$\left(\frac{\omega_0 \rho_g C_p}{2K}\right)^{\frac{1}{2}}$	
v	acoustic velocity in liquid	$1{\cdot}5\times10^5$ cm/sec
γ	ratio of specific heats of gas	1·4
p_0	static pressure	10^6 dyne/cm^2
ρ	density of liquid	1·0 g/cm^3
ρ_g	density of gas	$1{\cdot}29\times10^{-3}$ g/cm^3
σ	surface tension	75 dyne/cm
h	γ/ϵ	$1<h<\gamma$
C_p	heat capacity at constant pressure of gas	0·24 cal/g
K	thermal conductivity coefficient of gas	$5{\cdot}6\times10^{-6}$ cal/cm sec°C
η	viscosity of liquid-like medium	$1{\cdot}5\times10^2$ poise*
N	number of bubbles per unit volume	$0{\cdot}143\ R_0^{-3}$
b	total dissipation parameter, $b_t+b_r+b_v$	
b_t	thermal dissipation parameter (does not contribute appreciably)	
b_r	radiation dissipation parameter, $\frac{\rho\omega^2}{4\pi v}$	
b_v	viscous dissipation parameter, $\frac{\eta}{\pi R_0^3}$	
R_0	radius of gas bubble	
ω	angular frequency of sound field	$6{\cdot}28\times10^6$ radians/sec

* von Gierke, Oestreicher, Franke, Parrack and von Wittern (1952)

pulmonary alveoli which are approximately 0·1 mm in diameter. The alveolar sacs have dimensions considerably greater than that of both the respiratory bronchiole and the alveolar duct.

The very large number of spheroid-like and cylindrical gaseous elements with dimensions comparable to the wavelength of sound in the lung at the frequency of the measurements (0·98 Mc/sec), suggests a model composed of a uniform distribution of spherical gas bubbles imbedded in a liquid-like medium. It is considered that sound energy excites the bubbles to pulsate and that the bubbles dissipate their energy by radiating spherical sound waves, by polytropic compressions and expansions of the enclosed gas, and by viscous dissipation attributed to viscous forces acting at the gas-liquid interface. Assume that the liquid-like material has properties similar to water (except as noted in table 2) and that the gas has properties similar to air. In order to be consistent with the experimentally determined density of lung tissue (0·40 g/cm^3), the number of bubbles per unit volume, N, is related to the mean bubble radius as

$$N = \frac{0{\cdot}6}{4/3\pi {R_0}^3} = 0{\cdot}143\ {R_0}^{-3}, \quad . \ . \ . \ . \ . \ . \ . \quad (1)$$

i.e., this requires a packing of the bubbles approximately midway between hexagonal closest packing (each sphere touched by twelve neighboring spheres) and cubic packing (each sphere touched by six neighboring spheres). The acoustic amplitude absorption coefficient per unit path length for such a system is (Devin 1959; Fry and Dunn 1961)

$$\alpha = \frac{bNv}{4}\left[\frac{\dfrac{3\gamma P_0}{{R_0}^2} + \omega^2\rho}{\left\{\dfrac{1}{4\pi R_0}\left(\omega^2\rho - \dfrac{3g\gamma P_0}{\epsilon {R_0}^2}\right)\right\}^2 + b^2\omega^2}\right] . \ . \ . \ . \ . \quad (2)$$

The quantities appearing in eqn. (2) are defined in table 2 and values for these quantities useful in making the computation are also tabulated.

Fig. 3 shows the absorption coefficient described by eqn. (2) as a function of frequency for a bubble radius of 0·3 mm, a value consistent with the dimensions of the gaseous elements of lung, as discussed above. Fig. 4 shows the absorption coefficient as a function of bubble radius at a frequency of 1 Mc/sec. In the range of values chosen for the frequency and the bubble radius in the computations, radiation of spherical sound waves contributes the greatest source of energy dissipation; dissipation attributed to viscous forces acting at the gas-liquid interface is approximately an order of magnitude less and thermal conduction is negligible. On the assumption that the bubble radius is 0·3 mm, giving $5{\cdot}3 \times 10^3$ bubbles/cm^3 from eqn. (1), fig. 3 shows that the absorption coefficient at 1 Mc/sec is 5·7 cm^{-1} as compared with the experimentally determined value of 4·7 cm^{-1}. Further, fig. 4 shows that, at 1 Mc/sec, the experimentally determined value of the absorption coefficient is obtained for a bubble radius of 0·32 mm corresponding to a bubble population of $4{\cdot}4 \times 10^3$ bubbles/cm^3.

The agreement obtained between the experimentally determined value of the absorption coefficient and that of the model is considered to be sufficiently good to lend support to the view that the mechanism of ultrasonic absorption in lung tissue at the sonic frequency of 1 Mc/sec is primarily the result of radiation of sound waves by pulsating gaseous structures.

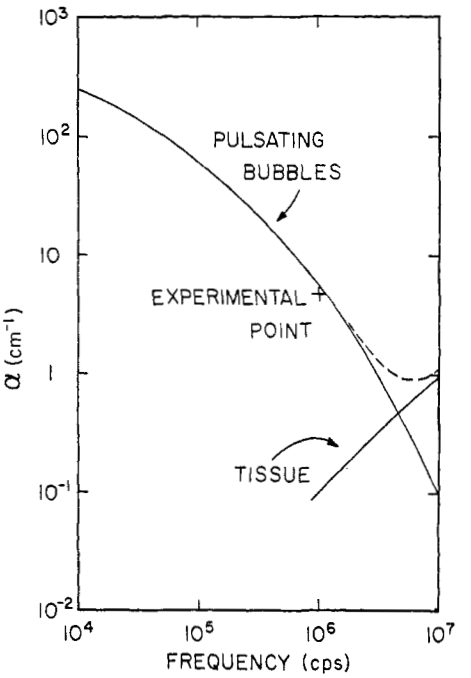

Fig. 3. Acoustic amplitude absorption coefficient per unit path length frequency for bubble radius of 0·3 mm.

§ 4. Discussion

Fig. 3 shows that as the sound frequency increases, the acoustic amplitude absorption coefficient per unit path length resulting from pulsating bubbles decreases and has the value of approximately 0·1 cm^{-1} at 10 Mc/sec (for bubbles of 0·3 mm radius). It is reasonable to assume that a contribution to the total absorption coefficient of lung also results from absorption of acoustic energy in the tissue. An examination of the literature (Goldman and Hueter 1956) indicates that with the exception of bone and lung, virtually all other mammalian tissues thus far investigated exhibit a linear dependence of the absorption coefficient per unit path length upon frequency and, at a frequency of 1 Mc/sec, the values lie between the limits of 0·025 cm^{-1} and 0·25 cm^{-1}. Let it be assumed that the total absorption coefficient, α_T, is the algebraic sum of that due to pulsating bubbles, α_b, and that due to tissue, α_t, i.e.,

$$\alpha_T = \alpha_b + \alpha_t \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad (3)$$

Let it be assumed further that $\alpha_t = 0{\cdot}1\ \mathrm{cm}^{-1}$ at 1 Mc/sec and that it increases linearly with frequency as shown in fig. 3. Then α_T, determined in accordance with eqn. (3) and illustrated in fig. 3 exhibits a minimum which (for the numerical values chosen) occurs at approximately 6 Mc/sec and has the value of $0{\cdot}9\ \mathrm{cm}^{-1}$.

The lung samples used in this study contained approximately one-third the average resting respiratory air of normal lung *in vivo*. If it is assumed that inflation of the lung to normal respiratory level has the effect of increasing the bubble radii (without altering the bubble population), then the acoustic absorption decreases, as evident from fig. 4. For such a case, the curve for pulsating bubbles would appear in fig. 3 below the one shown, and consequently would intersect the tissue absorption curve at a lower frequency yielding smaller values for the total absorption coefficient over the frequency range where the absorption due to pulsating bubbles is appreciable compared with that of tissue.

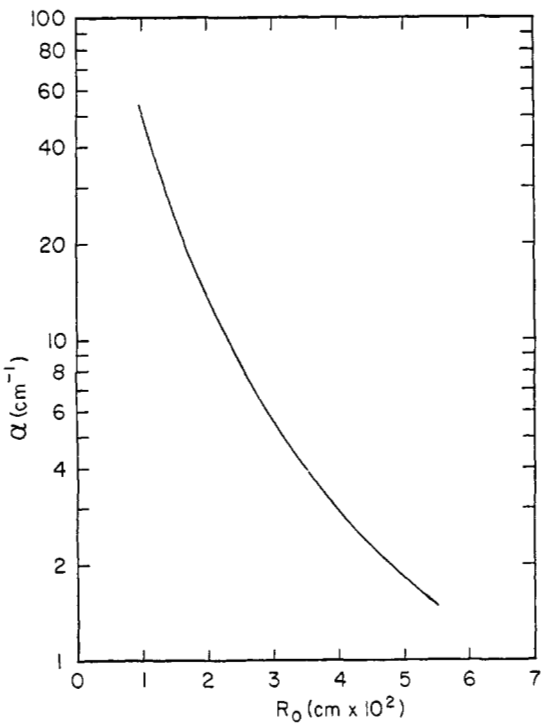

Fig. 4. Acoustic amplitude absorption coefficient per unit path length *vs* bubble radius at a frequency of 1 Mc/sec.

It thus appears that if ultrasound is to be used as a diagnostic tool to examine lung tissue *in vivo*, it is desirable to operate at frequencies near that of minimum absorption per unit path length and, in all probability,

also to increase considerably the sensitivity of the receiver for a given depth of penetration and incident sound intensity over that commonly employed for the examination of soft tissue structures of comparable thickness. Using the numerics obtained above, if internal lung structure visualization is to be accomplished comparable with the visualization of structures at the same depth in other soft tissue, it would be necessary to increase the sensitivity of the receiving system by approximately a factor of two for each centimeter depth as compared with that employed for the examination of other soft tissues. Further, there is a loss of 3 db on passing through the lung interface. With systems presently available, ultrasound could be used to examine the surface of the lung for uniformity and the immediate sub-surface tissue (the first few millimeters) for pathologic changes.

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Summary

The acoustic reflection and absorption coefficients of both normal and diseased (pneumonitis) excised lung tissue (dog) were experimentally determined at a frequency of 0·98 Mc/sec. It is found that the physiological saline-lung interface reflects 50% of the sound energy falling on it at normal incidence. The acoustic amplitude absorption coefficient per unit path length of lung tissue is 4·7 cm^{-1}. The very high absorption exhibited can be explained as caused by radiation of acoustic energy by the pulsating gaseous structures in the lung tissue. The theory indicates that the absorption coefficient of lung tissue should approach a minimum as the frequency is increased above 1 Mc/sec and should then increase at still higher frequencies. The diseased lung exhibited an acoustic absorption coefficient approximately 25% less than that of normal lung specimens.

Résumé

On a determiné expérimentalement pour la fréquence de 0·98 Mc/sec les coéfficients de réflexion et absorption acoustique pour les tissus pulmonaires normaux et morbides (pnéumonie) d'une chien. On a trouvé que la surface-frontière entre la solution physiologique et le poumon réflète 50% de l'énergie acoustique à l'incidence normale. Le coéfficient d'absorption d'amplitude acoustique par longueur unitaire de trajectoire dans le tissu pulmonaire s'élève à 4,7 cm^{-1}. La très grande absorption observée peut être expliquée par l'émission de l'énergie acoustique par les structures gaseuses vibrantes dans le tissus pulmonaires. La théroie indique que le coéfficient d'absorption du tissu pulmonaire doit s'approcher d'un minimum quand la fréquence surpasse 1 Mc/sec et qu'il doit augmenter aux fréquences plus hautes. Le poumon morbide avait un coéfficient d'absorption acoustique s'élevant à environ 75% de celui des échantillons du poumon normal.

Zusammenfassung

Die Schallreflexions- und Absorptionskoeffizienten sind bei einer Frequenz von 0,98 MHz für gesunde und kranke (Pneumonia) herausgeschnittene Hundeslungengewebe experimentell bestimmt worden. Es wurde gefunden, dass die Grenzfläche zwischen der physiologischen Salzlösung und der Lunge 50% der normal einfallenden Schallenergie reflektiert. Der Schallamplituden-Absorptionsbeiwert für Einheitsweglänge im Lungengewebe beträgt

4,7 cm^{-1}. Die äusserst hohe Absorption kann dadurch erklärt werden, dass die pulsierenden gasförmigen Gebilde im Lungengewebe Schallenergie ausstrahlen. Die Theorie deutet an, dass der Absorptionsbeiwert des Lungengewebes sich einem Minimum nähern dürfte, wenn die Frequenz über 1 MHz gesteigert wird, worauf er bei noch höheren Frequenzen ansteigt. Die kranke Lunge zeigte einen um etwa 25% kleineren Schallabsorptionsbeiwert als die normalen Lungenausschnitte.

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