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The optical properties of lung as a function of respiration

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Abstract. Lung consists of alveoli enclosed by tissue and both structures contribute to volume-dependent scattering of light. It is the purpose of this paper to determine the volume-dependent optical properties of lung. *In vivo* interstitial fibre measurements of the effective attenuation coefficient μ_{eff} at 632.8 nm differed during inspiration ($\mu_{\text{eff}} = 2.5 \pm 0.5 \text{ cm}^{-1}$) from that during expiration ($\mu_{\text{eff}} = 3.2 \pm 0.6 \text{ cm}^{-1}$). *In vitro* measurements on a piglet lung insufflated with oxygen from 50 to 150 ml showed the effective attenuation coefficient at 632.8 nm decreases as a function of oxygen volume in the lung (at 50 ml $\mu_{\text{eff}} = 2.97 \pm 0.11 \text{ cm}^{-1}$, at 100 ml $\mu_{\text{eff}} = 1.50 \pm 0.07 \text{ cm}^{-1}$, and at 150 ml $\mu_{\text{eff}} = 1.36 \pm 0.15 \text{ cm}^{-1}$). This was explained by combining scattering of alveoli (Mie theory) with optical properties of collapsed lung tissue using integrating sphere measurements. Theory and measured *in vitro* values showed good agreement (deviation $\leq 15\%$). Combination of these data yields the absorption coefficient and scattering parameters of lung tissue as a function of lung volume. We conclude that the light fluence rate in lung tissue should be estimated using optical properties that include scattering by the alveoli.

1. Introduction

The light fluence rate distribution inside tissue can be estimated from the solution of the equation of radiative transfer (Chandrasekhar 1960). This requires data on the absorption coefficient, μ_a (cm^{-1}), the scattering coefficient, μ_s (cm^{-1}), and the single-particle scattering phase function $p(\theta)$ (sr^{-1}), where θ denotes the scattering angle. Measurement of the optical properties, μ_a , μ_s , and the average cosine of the scattering angle, g , is often performed with an integrating sphere setup using *in vitro* tissue samples. However, there is now increasing evidence that this method overestimates absorption coefficients (Torres *et al* 1994). Furthermore, the optical properties of healthy lung tissue measured in a double-integrating-sphere set-up are expected to differ significantly from those of an insufflated lung because the *in vitro* sample of elastic lung is collapsed during the integrating sphere measurements and the alveoli are filled with saline instead of air.

In this paper, the optical properties of piglet lung are determined as a function of lung volume. First, the effective attenuation coefficient, μ_{eff} (cm^{-1}), is determined for insufflated lung using interstitial fibre measurements. Second, the contribution of lung tissue to the scattering parameters μ_s and g of an insufflated lung is determined using double integrating spheres and an inverse adding–doubling algorithm to a sample of collapsed lung tissue. Third, the contribution of the alveoli to μ_s and g of an insufflated lung are determined using Mie theory of scattering. Finally, the absorption coefficient μ_a of insufflated lung is determined from the experimental values of μ_{eff} , and the values obtained for μ_s and g .

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2. Materials and methods

The diffusion approximation to the equation of radiative transfer is expected to predict fluence rate distributions reasonably well in regions far from sources and boundaries, provided the absorption coefficient is much smaller than twice the reduced scattering coefficient $\mu'_s = \mu_s(1 - g)$ (Star 1995). In this paper we deal with optical properties of tissue for which $\mu_a \ll 2\mu'_s$ and hence we assume the diffusion approximation to be adequate for our analysis (see section 4).

For a point source in an infinite turbid medium the energy fluence rate $\phi(r)$ (W m^2) at distance r (mm) from the point source in the diffusion approximation is given by (Chandrasekhar 1960)

$$\phi(r) = \frac{P\mu_{\text{eff}}^2}{4\pi\mu_a r} \exp(-\mu_{\text{eff}} r) \quad (1)$$

where P (W) is the output power of the point source and μ_{eff} the effective attenuation coefficient:

$$\mu_{\text{eff}} = \sqrt{3\mu_a[\mu_a + \mu_s(1 - g)]}. \quad (2)$$

From (1), μ_{eff} follows as

$$\mu_{\text{eff}} = -\frac{d}{dr} \ln[\phi(r)r]. \quad (3)$$

Hence, μ_{eff} can be determined from the negative slope of $\ln[\phi(r)r]$ versus r . In this paper, a least-squares fit was used for $3 \text{ mm} \leq r \leq 17 \text{ mm}$, to relate the measurements of $\phi(r)$ to μ_{eff} .

Figure 1. Setup for the *in vivo* and *in vitro* interstitial fibre measurements.

Figure 1 shows the setup for the *in vivo* and *in vitro* interstitial fibre measurements. An HeNe laser at 632.8 nm was used as a light source. The fluence rate within the tissue was measured by a detection fibre as a function of distance from the source fibre with a translation stage. First, the spherical diffusers were positioned adjacent to each other outside the tissue. This implies a distance of 1 mm between centres of the diffusers as the diffusing tips have a diameter of 1 mm (see following paragraph). The fibres were then introduced into the tissue through needles, the fibre ends pointing towards each other. The needles, and the fibres, were inserted in such a way that the initial distance between the diffusers was 17 mm, which was the distance between the diffusers during the first measurement. This distance was decreased by translation steps of 0.5 to 1.0 mm. We determined the actual distance r between centres of the tips by subtracting the translation in millimetres from the initial 17 mm. A measurement of the transmitted power was then made which was considered proportional to the fluence rate because we assumed the (local) light distribution to be diffuse (Arnfield *et al* 1988, 1992).

Accurate measurements of the effective attenuation coefficient of turbid media require that the applicator and detector disturb the fluence rate inside the turbid media as little as possible. We used small isotropic spherical light diffusers with a 1 mm diameter sphere to meet this requirement (Star *et al* 1987). The construction, testing of isotropicity, and validation in a liquid phantom of the spherical light diffusers, are described elsewhere (Beek 1993). Their isotropicity at 632.8 nm was better than 20%, both as detector and as source.

For the *in vivo* experiments one piglet (6 kg) was used. The piglet received Atropin 0.1 mg kg⁻¹ intraperitoneally and Fluothane 4% through a mouth cap. An endotracheal tube was inserted and the measurements were performed under total anaesthesia using a mechanical respirator (Dräger Pulmomat), a ventilatory frequency of 30 min⁻¹, a ventilatory pressure of 20–25 cm H₂O and an O₂/N₂O/Fluothane (1%) gas mixture. At 632.8 nm the fluence rate in the right lung was measured three times as a function of distance r , reintroducing source and detector for each series of measurements at a different location in the lung as explained above.

For the *in vitro* experiments the right lung of another piglet (6 kg) was used immediately after sacrifice. At 632.8 nm the fluence rate in the lung was measured three times as a function of r at three different lung volumes (50 ± 5 , 100 ± 8 , and 150 ± 10 ml). The source and detector were reintroduced for each series of measurements at different locations in the lung. The lung volume was controlled by insufflation with 100% oxygen gas, and was measured by submerging the lung in 0.9% saline before and after each series of measurements. After the experiment and after suction was applied to remove all the air out of the lung the volume of the collapsed lung was 25 ± 3 ml.

The dependence of μ_{eff} on the volume of air in the lung was analysed assuming that there are two types of scatterer in the lung: scatterers intrinsic to the tissue (such as cell organelles) and alveoli. We assume neither the number of the scatterers intrinsic to the tissue nor their scattering properties change during respiration. However, total lung volume V_l that contains these scatterers does change and therefore the scattering coefficient of the tissue component $\overline{\mu_{st}}(V_l)$ should be weighted by the lung volume:

$$\overline{\mu_{st}}(V_l) = C_{st} / V_l \quad (4)$$

where C_{st} is a constant (ml cm⁻¹). For a given lung volume V_l , we have

$$V_l = V_t + V_a \quad (5)$$

where V_t is the volume occupied by the lung tissue itself (i.e. the collapsed lung without air), which is a constant, in the piglet lung measured as 25 ml, and V_a is the alveolar volume filled with air or gas. The constant C_{st} is defined in (4) by the product of $\overline{\mu_{st}}$ and V_l at (any) V_0 :

$$C_{st} = \overline{\mu_{st}}(V_0) V_0. \quad (6)$$

Using (4) and expressing C_{st} in (6) using $V_0 = V_t$ gives

$$\overline{\mu_{st}}(V_l) = \overline{\mu_{st}}(V_t) V_t / V_l. \quad (7)$$

We assume first that the scattering coefficient of the lung tissue itself, $\overline{\mu_{st}}(V_t)$, can be determined by integrating sphere measurements on an *in vitro* sample of collapsed lung in between two glass slides with a drop of saline to fill the alveoli (and to prevent refractive index mismatches with the glass slides). These measurements, described in the previous paper (Beek *et al* 1997), yielded $\mu_s = 301$ cm⁻¹ for piglet lung at 632.8 nm. Thus, $C_{st} = 301 \times 25 = 7525$ ml cm⁻¹ (4).

We further consider that the scattering properties of alveoli change during respiration. We will describe these changes using Mie theory, assuming the alveoli are approximately

spherical (Van de Hulst 1981, Oldmixon *et al* 1985). We assume that the number of alveoli, N , remains constant (independent of whether the lung is in the inspiration or in the expiration phase). Alveolar diameter d is assumed to be equal for all alveoli, reaching a value of about $d = 150 \mu\text{m}$ when total lung volume is inspired to $V_l = 150 \text{ ml}$ (Klinge and Staub 1970, Butler *et al* 1985). The total number of alveoli, $N = 7.1 \times 10^7$, can be derived from the ratio of total alveolar volume, V_a , and the volume of one alveolus $(4/3)\pi(d/2)^3$. Here, $V_a = 125 \text{ ml}$, equal to lung volume (150 ml) minus the volume of the collapsed lung (25 ml) (5). For an alveolus of diameter d , Mie theory calculates a scattering phase function, and hence an anisotropy factor of scattering, and a scattering cross section $\sigma(d)$ (cm^2). These calculations require the refractive indices of tissue and air which are taken here as $n_t = 1.37$ and $n_a = 1$ respectively (Bolin *et al* 1989). The (weighted) contribution of scattering $\overline{\mu_{sa}}(V_l)$ due to the alveoli in the tissue of volume V_l is given by (Van de Hulst 1981)

$$\overline{\mu_{sa}}(V_l) = \frac{N\sigma(d)}{V_l}. \quad (8)$$

The volume dependent (total) scattering coefficient of lung tissue, $\mu_{sl}(V_l)$, is defined as the sum of the weighted contribution of the two different scattering components (Van de Hulst 1981, Graaff *et al* 1992):

$$\mu_{sl}(V_l) = \overline{\mu_{st}}(V_l) + \overline{\mu_{sa}}(V_l). \quad (9)$$

In a similar way, the anisotropy of scattering of lung tissue, $g_l(V_l)$, follows as (Van de Hulst 1981, Graaff *et al* 1992)

$$g_l(V_l) = \overline{g_t}(V_l) + \overline{g_a}(V_l) \quad (10)$$

where $\overline{g_t}(V_l)$ and $\overline{g_a}(V_l)$ are the weighted anisotropy of scattering intrinsic to tissue and alveoli, respectively. Here, the anisotropy of scattering has to be weighted by the concentration of the scatterers and the scattering efficiency, i.e. by the ratio of (weighted) scattering coefficient of the scatterer and total scattering (Van de Hulst 1981, Graaff *et al* 1992):

$$\overline{g_t}(V_l) = \frac{g_t(V_t)\overline{\mu_{st}}(V_l)}{\mu_{sl}(V_l)} \quad (11)$$

and

$$\overline{g_a}(V_l) = \frac{g_a(V_a)\overline{\mu_{sa}}(V_l)}{\mu_{sl}(V_l)}. \quad (12)$$

We assume anisotropy for scattering in the tissue itself, $g_t(V_t)$, can be determined by integrating sphere measurements on an *in vitro* sample of collapsed lung.

Furthermore, for any lung volume, the lung's absorption coefficient, μ_a , is caused by the lung tissue only. Assuming a constant number of absorbing centres present in tissue volume V_t (and hence the same number in any other volume V), the weighted absorption coefficient $\overline{\mu_a}(V_l)$ as a function of lung volume (V_l) is defined similarly as in (4):

$$\overline{\mu_a}(V_l) = C_a/V_l \quad (13)$$

where C_a is a constant (ml cm^{-1}) which can be related to the product of μ_a and V at (any) V_0 :

$$C_a = \overline{\mu_a}(V_0)V_0. \quad (14)$$

For one specific lung volume V_0 of 150 ml, $\overline{\mu_a}(150 \text{ ml})$ was obtained from $\mu_{\text{eff}}(V_0 = 150 \text{ ml})$ (2), and $\mu_{sl}(150 \text{ ml})$ and $g_l(150 \text{ ml})$ from (8)–(10), using $\overline{\mu_{st}}(V_t) = 301 \text{ cm}^{-1}$ at $V_t = 25 \text{ ml}$

(7), and $d = 150 \mu\text{m}$ for the Mie calculations (Klingele and Staub 1970, Van de Hulst 1981, Oldmixon *et al* 1985). Combining the experimental results ($\mu_{\text{eff}}(150 \text{ ml}) = 1.36 \text{ cm}^{-1}$) and theoretical results ($\mu_{sl}(150 \text{ ml}) = 217.3 \text{ cm}^{-1}$, and $g_l(150 \text{ ml}) = 0.85$) yields $\mu_a(150 \text{ ml}) = 0.02 \text{ cm}^{-1}$ from (2). From this value we calculate from (14) that $C_a = 0.02 \times 150 = 3 \text{ ml cm}^{-1}$. Thus, the absorption coefficient $\bar{\mu}_a(V_l)$ of any lung volume V_l can now be derived from (13). Subsequently, for lung volume V_l , we can determine $\mu_{\text{eff}}(V_l)$ from (2) using the calculated values of μ_{sl} and g_l at V_l , and the value of $\bar{\mu}_a(V_l)$ assessed from (13).

3. Results

The *in vivo* effective attenuation coefficient differed during expiration and inspiration in each series of measurements (Student's *t* test, $p < 0.001$). Figure 2 gives the results of three *in vivo* measurements at 632.8 nm. The means $\pm$ standard deviations of these three measurements are at inspiration $\mu_{\text{eff}} = 2.5 \pm 0.5 \text{ cm}^{-1}$ and at expiration $\mu_{\text{eff}} = 3.2 \pm 0.6 \text{ cm}^{-1}$.

In vitro measurement at 632.8 nm on a lung that was insufflated with oxygen gas from 50 to 150 ml confirmed that the effective attenuation coefficient depends on the volume of air in the lung (at 50 ml $\mu_{\text{eff}} = 2.97 \pm 0.11 \text{ cm}^{-1}$, at 100 ml $\mu_{\text{eff}} = 1.50 \pm 0.07 \text{ cm}^{-1}$, and at 150 ml $\mu_{\text{eff}} = 1.36 \pm 0.15 \text{ cm}^{-1}$).

Figures 3 and 4 show the scattering coefficient and the anisotropy of scattering (intrinsic to the tissue, of the alveoli and of the total lung) respectively versus the lung volume. Figure 5 shows the effective attenuation coefficient $\mu_{\text{eff}}(V_l)$ for volumes V , now calculated using values of $\mu_{sl}(V_l)$, $g_l(V_l)$ and $\bar{\mu}_a(V_l)$ according to (9), (10) and (13), and fitting the theoretical curve at the measured value of μ_{eff} at $V_0 = 150 \text{ ml}$. This theoretical curve compares favourably with the other two measured values of μ_{eff} (at $V_l = 100$ and 50 ml), showing a maximum deviation of only 15% at $V_l = 100 \text{ ml}$.

4. Discussion

In this study we used diffusion theory to represent the lung fluence rate in response to a point source. The argumentation is as follows. Lung tissue can be considered as a very large number of scattering 'cells'. Each cell consists of (i) a volume of absorbing and scattering lung tissue with refractive index 1.37, and (ii) a sphere of air (alveolus) enclosed by a membrane with refractive index 1.37. Scattering of this latter structure is according to Mie theory. Scattering, therefore, results from two scattering mechanisms, each with their own individual scattering phase function. The overall scattering phase function for a 'cell', in principle, can be measured by goniometric measurement. We combined scattering measurements of the tissue and Mie calculations of alveoli to obtain the weighted overall scattering coefficient of lung. For this random medium, consisting of absorbing and scattering 'cells', diffusion theory is valid if $\mu_a \ll \mu_s(1-g)(1+g)$ (Star 1995). Because g is close to unity this becomes $\mu_a \ll 2\mu'_s$. Our results certainly meet this requirement, because for $25 \text{ ml} \leq V_l \leq 150 \text{ ml}$: $0.12 \text{ cm}^{-1} \geq \mu_a \geq 0.02 \text{ cm}^{-1}$ and $21.1 \text{ cm}^{-1} \leq \mu'_s \leq 28.6 \text{ cm}^{-1}$. Consequently, there are no limitations to use diffusion theory in our results.

In this study we assumed alveoli to be spheres of air surrounded by tissue. We did not consider that airways have a tubular shape. Nevertheless, the distribution of alveolar septa is highly regular, and angles between adjacent septa are highly correlated (they peak near 120°) (Butler *et al* 1985, Oldmixon *et al* 1985, Suzuki *et al* 1985). Although this simplification might have influenced the results of the Mie calculations, we believe the

Figure 2. Experimental results at 632.8 nm of $\ln[\phi(r)r]$ versus distance r between detector and source during *in vivo* inspiration and expiration. Results of μ_{eff} for the three series of measurements ((a), (b) and (c)) are presented. For each series of measurements μ_{eff} during inspiration differs significantly from μ_{eff} during expiration ($p < 0.001$, Student t test).

results of optical properties of lung tissue as described in this paper are more accurate than those measured with a double-integrating-sphere set-up (Beek 1993, Pickering *et al* 1993, Torres *et al* 1994).

At 632.8 nm the integrating sphere measurements yielded $\mu_a = 2.0 \pm 0.1 \text{ cm}^{-1}$ (Beek 1993) versus (this work) $\mu_a = 0.12 \text{ cm}^{-1}$ for collapsed piglet lung, confirming

Figure 2. (Continued)

the assumption that integrating sphere measurements overestimate μ_a (Beek 1993, Torres *et al* 1994). However, compared to the expected absorption of human lung, absorption of piglet lung as measured in this work is low. If we consider human lung of 1.2 kg containing a volume of capillary blood of 150 ml, a lung capacity of 4–9 l, and blood absorption at 632.8 nm of oxyhaemoglobin and reduced haemoglobin of 4 and 22.5 cm^{-1} respectively, the absorption coefficient due to capillary blood alone in fully inflated lung is expected to be in the range of 0.12–0.06 cm^{-1} for the 100% oxygenated case, and 0.22–0.11 cm^{-1} in the 80% oxygenated case. The corresponding absorption coefficient for deflated lung is, provided that the capillary blood volume remains unchanged, 0.5 and 0.96 cm^{-1} for the 100 and 80% oxygenated case respectively. However, for our experiments we have used the lungs of newborn piglets. One striking aspect of these lungs is their whitishness. The observation that piglet lungs are much more whitish than human lungs suggests a low absorption compared to absorption in human lung. We believe this explains the factor of about five between absorption in piglet lung and human lung.

The *in vivo* effective attenuation coefficient at 632.8 nm during inspiration differed from that during expiration in each of three sets of measurements ($p < 0.001$, Student's t test). The means $\pm$ standard deviations for three sets of measurements were $\mu_{\text{eff}} = 2.5 \pm 0.5 \text{ cm}^{-1}$ and $\mu_{\text{eff}} = 3.0 \pm 0.6 \text{ cm}^{-1}$ during inspiration and expiration respectively. *In vitro* measurement at 632.8 nm on a lung that was insufflated with oxygen gas at 50 and 100 ml gave $\mu_{\text{eff}} = 2.97 \pm 0.11 \text{ cm}^{-1}$ and $\mu_{\text{eff}} = 1.50 \pm 0.07 \text{ cm}^{-1}$, respectively. It is interesting to note that the respiratory volume in the *in vivo* experiment was 30–40 ml, giving a smaller decrease in effective attenuation coefficient as a function of volume compared to the *in vitro* measurements. However, the *in vitro* measurements do not reflect contributions from perfusion, oxygenation and other tissue conditions *in vivo*. During the *in vitro* experiments we assume the number of absorbers in the tissue and the saturation of haemoglobin are constant. The number of absorbers per volume decreases during inspiration and increases during expiration, resulting in a lower absorption coefficient during inspiration than during

Figure 3. The weighted scattering coefficient intrinsic to the tissue, $\overline{\mu_{st}}$, using (7), the weighted scattering coefficient of the alveoli, $\overline{\mu_{sa}}$, using (8) and alveolar diameter $d = 150 \mu\text{m}$ at lung volume $V_0 = 150 \text{ ml}$, and the scattering coefficient of lung tissue, μ_{sl} , using (9), at 632.8 nm , as a function of the total lung volume V_l of the insufflated lung.

Figure 4. The weighted anisotropy of scattering intrinsic to the tissue, $\overline{g_t}$, using (11), the weighted anisotropy of scattering of the alveoli, $\overline{g_a}$, using (12) and $d = 150 \mu\text{m}$ at $V_0 = 150 \text{ ml}$, and the anisotropy of scattering of lung tissue, g_l , using (10), at 632.8 nm as a function of the total lung volume V_l of the insufflated lung.

Figure 5. The effective attenuation coefficient, calculated using theoretical values for μ_{sl} and g_l and a volume-dependent $\bar{\mu}_a(V)$ assuming that $\bar{\mu}_a(V)V$ is constant (13), and the measured *in vitro* effective attenuation coefficient of lung tissue at 632.8 nm, as a function of the total lung volume V_l of the insufflated lung. Theory and experiments are fitted at $V_l = 150$ ml.

expiration. *In vivo* the same phenomenon occurs. In addition, *in vivo* absorption of blood during inspiration is lower than that during expiration because blood is oxygenated during inspiration. However, the increased intrathoracic pressure during inspiration results in an increase in the venous return to the heart, resulting in an increase in blood pumped into the lungs and a decrease of blood pumped into the aorta (this phenomenon is explained by the Frank–Starling law of the heart). Therefore, the volume of blood in the lung increases during inspiration, resulting in an increase in absorption. This increase in absorption might explain the smaller difference in the effective attenuation coefficient as a function of volume during the *in vivo* experiment compared to the *in vitro* experiment.

In conclusion, at 632.8 nm, we observed under *in vivo* conditions that μ_{eff} was larger during expiration than during inspiration. The dependence of the effective attenuation coefficient on lung volume was predicted combining experimental results and theoretical calculations at a lung volume of $V_0 = 150$ ml and assuming that the number of scattering and absorbing centres in the lung remains unchanged. We conclude that the light fluence rate in lung should be estimated using an absorption coefficient that is low compared to integrating sphere data and a scattering coefficient and scattering anisotropy function that take into account scattering both in the tissue itself and by the alveoli.

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