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Lung Tissue Rheology and 1/*f* Noise

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Abstract—The mechanical properties of lung tissue are important contributors to both the elastic and dissipative properties of the entire organ at normal breathing frequencies. A number of detailed studies have shown that the stress adaptation in the tissue of the lung following a step change in volume is very accurately described by the function t^{-k}, for some small positive constant k. We applied step increases in length to lung parenchymal strips and found the ensuing stress recovery to be extremely accurately described by t^{-k} over almost 3 decades of time, despite the quasi-static stress–length characteristics of the strips being highly nonlinear. The corresponding complex impedance of lung tissue was found to have a magnitude that varied inversely with frequency. We note that this is highly reminiscent of a phenomenon known as 1/*f* noise, which has been shown to occur ubiquitously throughout the natural world. 1/*f* noise has been postulated to be a reflection of the complexity of the system that produces it, something like a central limit theorem for dynamic systems. We have therefore developed the hypothesis that the t^{-k} nature of lung tissue stress adaptation follows from the fact that lung tissue itself is composed of innumerable components that interact in an extremely rich and varied manner. Thus, although the constant k is no doubt determined by the particular constituents of the tissue, we postulate that the actual functional form of the stress adaptation is not.

Keywords—Stress adaptation, Fractals, Viscoelasticity, Lung tissue impedance

INTRODUCTION

The mechanical properties of lung tissue are important contributors to the overall mechanical behavior of the entire organ. Indeed, the dissipative (resistive) properties of these tissues are known to be the major determinants of lung resistance in many species at frequencies within the breathing frequency range. Furthermore, lung tissue resistance decreases markedly as the frequency at which flow is oscillated at the airway opening is increased. This phenomenon is a consequence of the fact that lung tissue, like all biological soft tissues, is viscoelastic. This was first alluded to by Bayliss and Robertson (4) and Mount (23). However, the real significance of viscoelasticity in lung tissue has been fully appreciated only since the seminal work of Hildebrandt (16,17) and Bachofen (1) on isolated cat lungs. More recently, Bates *et al.* (3) used the alveolar capsule technique in dogs to determine that, under normal conditions, the apparently multi-compartment behavior of the organ is due to tissue viscoelasticity rather than regional ventilation inhomogeneity, as had been the popular conception since the influential model analysis of Otis *et al.* (26). Ludwig *et al.* (19) also used the alveolar capsule technique to demonstrate the importance of tissue resistance to the resistance of the whole lung. Now the literature contains numerous studies in humans (*e.g.*, 11,32) and various animal species (*e.g.*, 12,13,30) demonstrating the typical frequency-dependent nature of lung impedance that is the hallmark of a viscoelastic structure.

Over the normal range of breathing frequencies, the viscoelastic nature of lung tissue may be conveniently and accurately described by the linear viscoelastic solid, or Kelvin body (10) shown in Fig. 1A. The single spring spanning the structure from top to bottom gives rise to its static elastic properties. The series spring and dashpot (together known as a Maxwell element) act much like a shock absorber on an automobile and produce an exponential decay in stress (pressure) following a step change in strain (volume) as shown in Fig. 1B. The Maxwell element also enables the system to achieve a balance between conservative and dissipative behavior that varies with frequency in a manner similar to that observed in nature (Fig. 1C).

Nevertheless, nature is not quite this simple. Detailed examination by Hantos *et al.* of the frequency dependence of mechanical impedance in dog lungs (13,14) and cat lungs (15) has shown that the Kelvin body does not describe low frequency lung tissue mechanics with perfect accuracy. Rather, the real part of impedance is significantly better described as being hyperbolic with frequency, instead of sigmoidal as the Kelvin body predicts

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(Fig. 1C). Similarly, the product of the imaginary part of impedance with angular frequency, commonly called the dynamic elastance, is more closely linear with the logarithm of frequency (f) than sigmoidal with f, as predicted by the Kelvin body (Fig. 1C). Hantos *et al.* (13) showed that their results are in accord with the original observations in isolated cat lungs made by Hildebrandt (16,17), who found the time course of stress adaptation following a step change in lung volume to be almost perfectly linear with the logarithm of time (t). Peslin *et al.* (28) found the same in rat lungs. Hildebrandt (16), however, also found stress adaptation in a rubber balloon to be very well described by a function of the form t^{-k}, for some positive constant k, as is the case for various polymers (8). Recently Hantos *et al.* have shown that the frequency domain equivalent of the t^{-k} model is an even better description of lung tissue mechanics *in vivo* in dogs (14) and cats (15) than is the model predicting a linear course of stress adaptation with the logarithm of t, although the two descriptions are very similar over a wide range of t because

$$\begin{aligned} t^{-k} &= \exp[-k \ln(t)] \\ &= 1 - k \ln(t) + k^2 \ln(t)^2/2! - \ldots \\ &\quad + (-1)^n k^n \ln(t)^n/n! + \ldots \\ &\approx 1 - k \ln(t) \end{aligned} \tag{1}$$

provided $k \ll 1$ (which seems to be the case as k has been shown to have values less than 0.1 [14]).

It is intriguing that such apparently unrelated materials as lung tissue, rubber, and polymers can have stress adaptation time courses that are qualitatively so similar, all being extremely accurately described by a function on the form t^{-k}. Consequently, one is compelled to ponder why stress adaptation should always demonstrate this particular dynamic. In this paper we present further experimental evidence for the universality of t^{-k} and argue for a theory of stress adaptation based on the ensemble behavior of systems with many interacting components. We suggest that important aspects of the bulk dynamic properties of a viscoelastic material reflect the complexity of the system per se and may be quite independent of the specific properties of the material's components, something like the central limit theorem of statistics.

METHODS

We prepared and tested degassed strips of dog lung parenchyma under uniaxial strain as described by Maksym *et al.* (20). Briefly, tissue strips of approximately 3 cm unstressed length (l_0) and 0.2 cm^2 in cross section were cut from the left lobes of Krebs–Ringer-washed degassed dog lungs with the pleura removed from the strips. Each strip was mounted horizontally in a uniaxial testing apparatus and submerged in a temperature-controlled organ bath. One end of the strip was connected to a piezoresistive temperature-compensated load cell, and the other to the moving arm of a linear motor. The position of the motor arm was measured by a linear variable differential transformer (LVDT). A 486 personal computer sampled the LVDT signal and implemented a servo-control algorithm that gave the motor a flat bandwidth to 20 Hz with a 1 cm peak-to-peak displacement and a step response with no overshoot. The force measured by the load cell was sampled at 50 Hz and normalized to the cross-sectional area of the tissue strip to give an output in kilopascals.

Before making measurements, the strips were subjected to a preconditioning protocol consisting of four consecutive cycles, at constant rate of strain of 2% $l_0 \cdot sec^{-1}$, between rest length and a stress of 5 kPa. After reaching 5 kPa for the final time, the strips were returned at the same strain rate to the desired operating stress. Once the desired operating stress was reached, the tissue was held at a fixed length for 6 min, after which a sudden step stretch (rise time = 10 msec) was applied. The new length was maintained for an additional 60 sec, during which the axial stress in the tissue was measured. Preconditioning protocols were repeated prior to each measurement in order to standardize the strain history. In this way, we measured the stress relaxation in the tissue strips occurring from 0.02 to 60 sec following 10% length changes from three different initial lengths. Curves of the form $S_0 + At^{-k}$ (S_0 being the initial stress prior to the step) were fitted to the stress relaxation data using SigmaPlot v 4.0 (Jandel Scientific, Corte Madera, CA).

We also subjected the same tissue strips to sinusoidal oscillations of amplitude equal to 10% of the resting length after establishing an operating stress of 1.08 ± 0.04 kPa following preconditioning as described above. Five oscillatory cycles were performed at frequencies of 0.03, 0.1, 0.3, 1, and 3 Hz. Tissue impedance was calculated as the complex frequency domain ratio of stress to rate of change of strain from the last four cycles.

RESULTS

Figure 2 shows an example of the final preconditioning stress–stretch curve from a typical lung strip undergoing uniaxial stretching at constant velocity during both increasing length (top curve) and decreasing length (bottom curve). The curves are highly nonlinear and demonstrate a degree of hysteresis in that the increasing and decreasing curves are not identical.

Figure 3 shows log–log plots of three stress adaptation curves from a typical tissue strip obtained by subjecting the strip to 10% step changes of resting length from three different initial resting lengths. The initial stresses (S_0) prior to each step correspond to three points on the de-

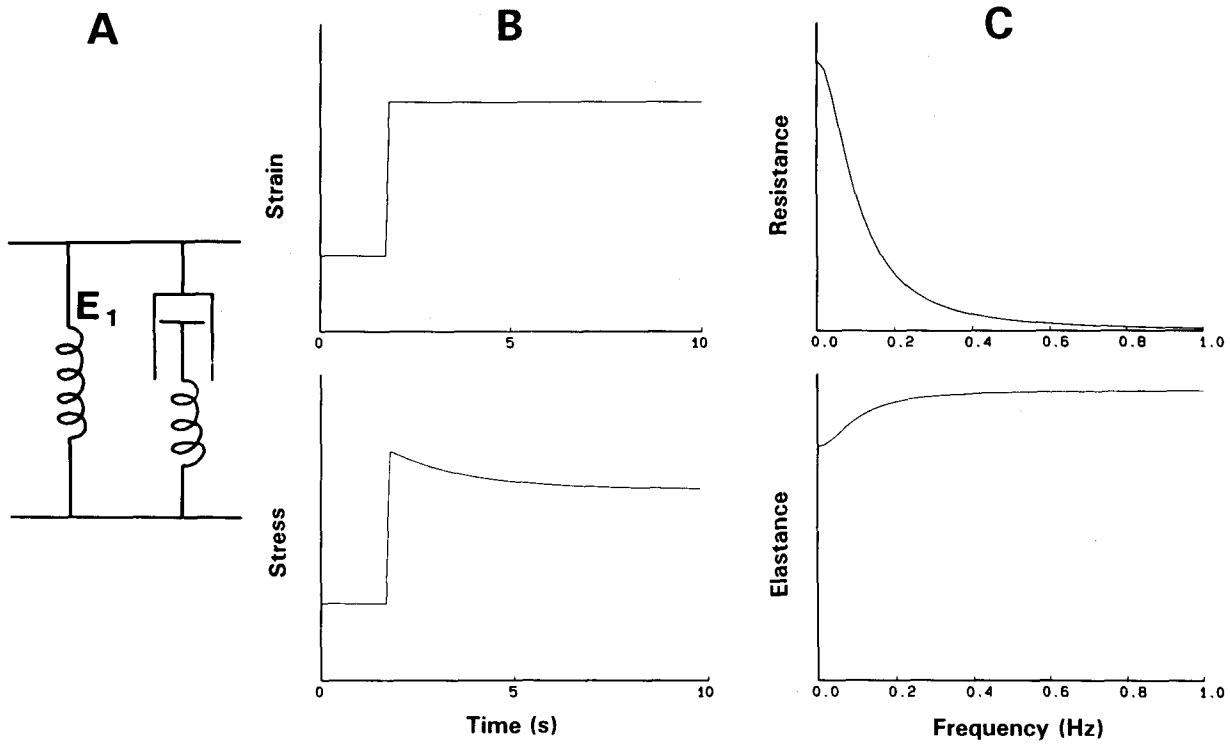

FIGURE 1. The elements of the linear viscoelastic solid, or Kelvin body. (A) The Kelvin body. (B) The time domain behavior of the Kelvin body. A step increase in strain results in a corresponding immediate increase in stress that then relaxes exponentially to a lower level. (C) The frequency domain behavior of the Kelvin body. The overall resistance of the Kelvin body decreases toward zero in a sigmoidal fashion with frequency. The initial value of resistance equals that of the dashpot in the Kelvin body. The overall elastance of the Kelvin body at zero frequency equals that of the single spring spanning the horizontal bars in the Kelvin body. Overall elastance then increases with frequency toward a value at infinity equal to the sum of the two springs in the Kelvin body.

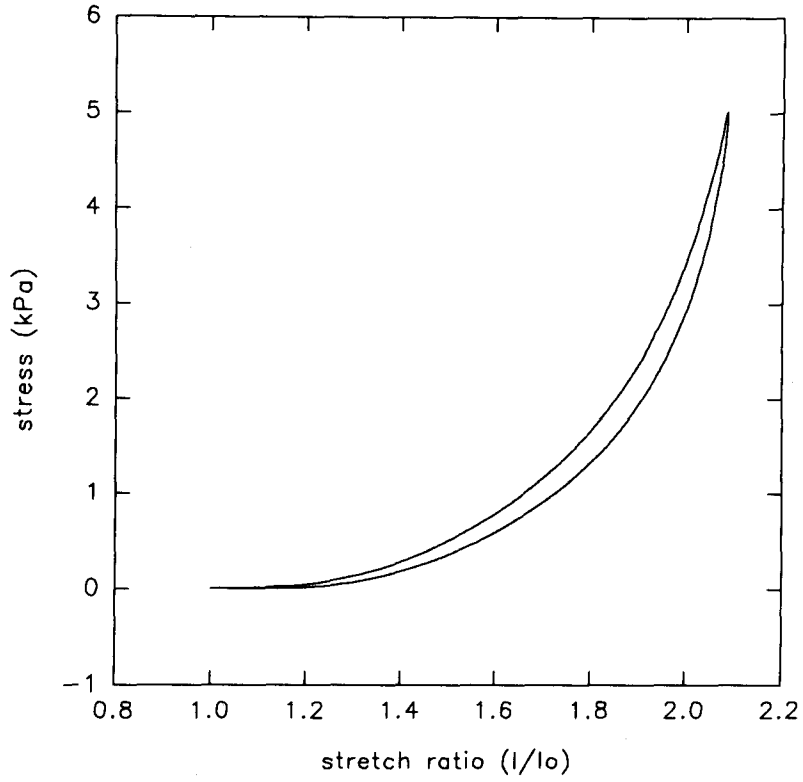

FIGURE 2. Stress *versus* stretch in a strip of lung parenchyma subjected to slow cycling at a constant strain rate.

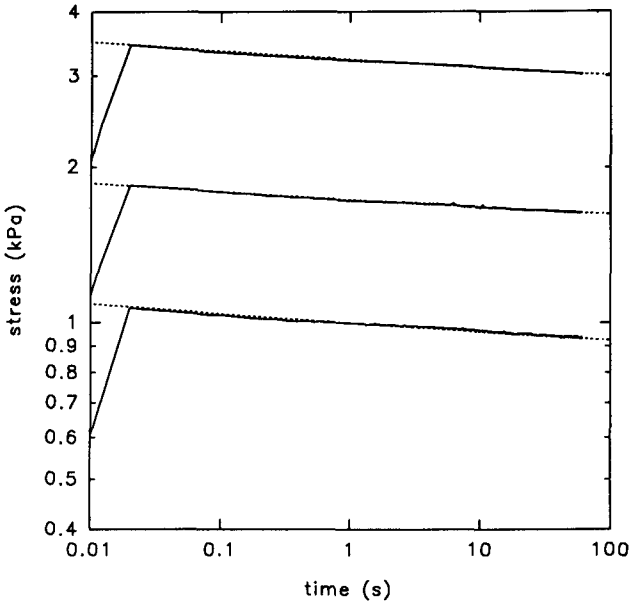

FIGURE 3. Three stress recovery curves obtained from the same lung parenchymal strip subjected to 10% stretch from three different starting lengths.

scending limb of the stress–stretch curve shown in Fig. 2. Also shown in Fig. 3 are the fitted curves of the form $S_0 + At^{-k}$, which appear as straight lines in the log–log plot. Although the values of A for the three curves varied considerably (0.389, 0.603, and 1.174 kPa, respectively, from the lowest to the highest), the values of k were remarkably similar (0.0451, 0.0424, and 0.0446, respectively). The root-mean-square residuals between the three sets of relaxation data and their fitted curves were 0.0046, 0.0028, and 0.0025 kPa, respectively, from the lowest to the highest.

The results shown in Figs. 2 and 3 are typical of those from all lung tissue strips studied. Table 1 shows the results obtained from five strips, each from a different dog, each subjected to the same procedure as that shown in Fig. 3. Again, it is clear that, despite a marked dependence of A on operating stress (S_0), k is almost perfectly independent of S_0.

Figure 4 shows a log–log plot of the magnitude of tissue strip impedance (average of five strips) *versus* frequency together with a straight line fit. From the fitted line we estimate the magnitude of tissue strip impedance to be a/f^{α}, where $a = 0.991 \pm 0.088$ kPa sec^{-1} and $\alpha = 0.959 \pm 0.022$ (mean $\pm$ standard deviation estimated from the fit).

TABLE 1. Parameters of the model $S - S_0 = At^{-k}$ fitted to stress relaxation data obtained from lung tissue strips subjected to 10% step increases in resting length from three different initial stresses (S_0).

S_0 (kPa)	A (kPa)	k	rms Residual (kPa × 10^{-3})
0.6	0.42 ± 0.05	0.044 ± 0.005	5.2 ± 3.5
1.1	0.60 ± 0.05	0.045 ± 0.004	3.8 ± 1.7
2.1	1.25 ± 0.22	0.046 ± 0.006	4.9 ± 2.0

The values of A and k are means (±standard deviation) from five strips, each taken from a different dog lung.

DISCUSSION

Viscoelasticity

Viscoelasticity is characterized by transient changes in stress following sudden changes in strain; indeed, this is virtually the definition of viscoelasticity. In general, such behavior can be modeled by a collection of Maxwell elements having appropriate values of resistance (R) and elastance (E). When the R and E values in such a model are constant, the phenomenon it represents is known as linear viscoelasticity, which alternatively may be characterized by a distribution of time constants (the ratios of the R to the E values). The stress adaptation transient exhibited by such a model following a step change in strain consists of a sum of decaying exponential functions. The time constants of the exponentials are precisely those of the Maxwell elements, and the coefficients of the exponentials are proportional to the respective E values.

It is possible, in principle, to find a distribution of time constants $B(\tau)$ that corresponds to any conceivable shape of stress adaptation transient $S(t)$ so that

$$S(t) = \int_0^{\infty} B(\tau)\, e^{-t/\tau} d\tau. \qquad (2)$$

In particular, when $B(\tau)$ is hyperbolic in t between two limiting values and zero elsewhere, then the system so defined has an impedance with a constant phase between corresponding frequency limits (10). This corresponds to an $S(t)$ that is proportional to t^{-k} between corresponding time limits, for some $k > 0$. Thus, it is possible to characterize stress adaptation in terms of linear systems theory as a continuous distribution of time constants. Indeed, it has often been viewed this way (10), perhaps for no better reason than the mathematics of linear systems are tractable and well developed. This does not mean, however, that a model of linear interconnected compartments accurately reflects the underlying physical processes giving rise to the stress adaptation. As our data show (Fig. 2), the quasi-static stress–strain behavior of lung tissue is markedly nonlinear. The same applies to the quasi-static behavior of whole lungs (27) and its dynamic behavior when oscillated about different mean strains (1,15,17), so that a nonlinear model of tissue (*e.g.*, 31,33) must be invoked to describe it over a range of strains.

A particularly significant aspect of our stress adaptation data (Fig. 3) is that, despite the highly nonlinear quasi-

static stress–length properties of the lung tissue strips, their stress recovery time courses showed almost exactly the same time dependence over nearly 3 decades of time regardless of the length from which they were stretched. That is, the stress relaxation curves were almost perfectly described by a function of the form $A(S_0)t^{-k}$, where $A(S_0)$ is a markedly nonlinear function of initial stress (or tissue length) while the time dependence is confined to the factor t^{-k}. This separation of dependencies has been described before and is the basis of Fung's quasi-linear theory of viscoelasticity (10). Navajas *et al.* (25) recently applied quasi-linear viscoelasticity to the mechanical behavior of strips of diaphragm. Despite the success of Fung's theory, however, one cannot take the independence of the value of k on S_0 as evidence of linear underlying dynamics, because the highly nonlinear forms of both the quasi-static stress–strain curve (Fig. 2) and $A(S_0)$ (Table 1) clearly show otherwise. Rather, it stands as further compelling evidence of the universality of t^{-k} as a description of soft tissue adaptation.

Another philosophical problem with the linear dynamic viewpoint is that it requires nature to have chosen the same special type of time constant distribution (*i.e.*, hyperbolic) for its various soft tissues. Why should the widely disparate constituents of materials such as lung tissue, polymers, and rubber have this common property? We feel that it is unreasonable to consider a specific type of linear model structure for soft tissues and so have looked for a more general genesis for soft tissue mechanical behavior. In the course of our search we were struck by another natural phenomenon whose ubiquity throughout the physical world gives it the same character of generality as the t^{-k} form for stress adaptation. This phenomenon is the so-called $1/f$ noise.

1/f Noise

Many dynamical systems produce apparently random behavior that has a power spectral density which varies with the inverse of frequency to some power g, where $0 < g < 2$. This has come to be known as $1/f$ noise and has been described in many apparently unrelated systems, such as electronic components (34), economic data (21), and rate of insulin uptake by diabetics (6). Indeed, even the loudness and pitch of music exhibit this type of behavior (35)! There have, of course, been attempts to understand the basis of $1/f$ noise in terms of linear systems theory (18). On the other hand, Murch and Bates (24) recently showed that a system of coupled nonlinear difference equations also produces this type of behavior. In fact, the precise origin of $1/f$ noise remains something of a mystery. Nevertheless, its widespread occurrence compels one to speculate that $1/f$ noise reflects, in the words of Keshner (18), "some profound law of nature that applies to all nonequilibrium systems." West and Schlesinger (36) also decided to "adopt the point of view that $1/f$ noise is the consequence of a system being complex, irrespective of the context."

To see how the $1/f$ noise phenomenon might bear on soft tissue mechanics, consider the function

$$P(t) = At^{-k}, \tag{3}$$

which, as we have already pointed out, seems to describe accurately stress adaptation of lung tissue following a step change in volume, where $P(t)$ is the pressure applied to inflate the lungs (referenced to the preinflation pressure) and A and k are positive constants. Considering lung tissue for the moment as a linear system, Eq. 3 can be viewed as the step response of the system when volume is the input and pressure the output. Equivalently, if flow into the lungs is taken as the input, then Eq. 3 is the impulse response of the system, which makes its Fourier transform the complex impedance, $Z(f)$, of the lung tissue. The Fourier transform of Eq. 3 can be found in standard tables to be

$$Z(f) = A(2/\pi)^{1/2}\Gamma(1 - k)\{\cos[(1 - k)\pi/2] + i\sin[(1 - k)\pi/2]\}/(2\pi f)^{1-k}, \tag{4}$$

where Γ is the Gamma function and $i = -1$. Thus if a white noise flow signal is applied to the lungs, then the resulting $P(t)$ has the character of noise with a power spectral density proportional to $1/f^{2-2k}$. For $0 < k < 1$, this falls within the definition of $1/f$ noise (18).

The above shows that a system with a transient dynamic response like that of soft tissue will shape a general (*i.e.*, white) noise source into $1/f$ noise, provided that k is somewhere in the interval 0 to 1. This condition is met by the value of k (mean value 0.045; Table 1) that we found in lung tissue strips. It is also strongly supported by our measurements of impedance as a function of frequency in the strips (Fig. 4), in which we found impedance magnitude to be almost perfectly proportional to $f^{-\alpha}$. Furthermore, the value of α we obtained from the impedance measurements (0.959) is very close to that predicted from Eq. 4 using the measured value of $k(\alpha = 1 - k = 0.955)$. Other estimates of k are also compatible with our hypothesis. For example, Hantos *et al.* (14) studied the low frequency impedance of dog lungs and found a mean value for k for normal lungs of 0.09. We also fitted Eq. 3 to the stress relaxation data of Peslin *et al.* (28) and found k to be about 0.15 (unpublished observations). These findings thus led us to wonder whether $1/f$ noise and general soft tissue stress adaptation might be reflections of the same underlying physical process. If this were true, we would be able to use the time domain expression Eq. 3 and the frequency domain $1/f$-type relation interchangeably as empirical descriptions of the same dynamic system, as depicted in Fig. 5. Of course, the question remains as to the

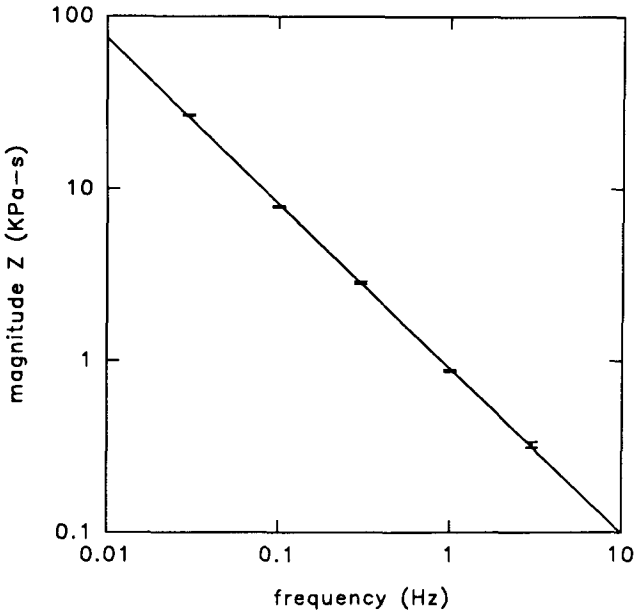

FIGURE 4. A log–log plot of tissue strip impedance magnitude *versus* oscillation frequency, together with the line of best fit. The data points shown are means (±standard deviation) from five strips, each from a different dog.

identity of the underlying phenomenon that must be common to soft tissues and all those myriad systems that produce $1/f$ noise. The only thing that we can think of which is common to all these systems is the fact that they are complex; that they are composed of innumerable, mutually interacting components or subsystems.

Actually, the link between $1/f$ noise and power law relaxation processes has already been made recently through a phenomenon called self-organized criticality (SOC) (2,7). SOC occurs in a system of interconnected components that are each able to absorb energy up to a certain point, beyond which the energy spills out into the nearest neighbors. It has been shown that such a system naturally organizes itself so that, regardless of where energy is put into the system, the spatial and temporal distributions of energy within the system assume power law forms (7). This results in the temporal dynamics of the system being characterized by $1/f$ noise.

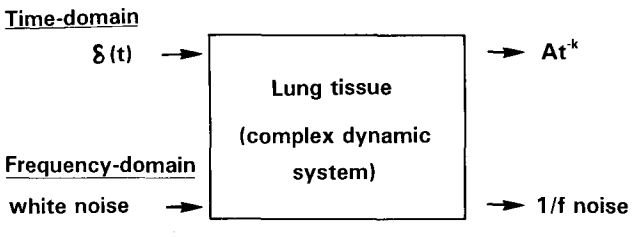

FIGURE 5. The time/frequency domain duality of lung tissue postulated as arising from its inherent dynamic complexity. In the time domain an impulse input (flow) gives rise to an output (pressure) having the form t^{-k}. In the frequency domain a white noise input produces a $1/f$ type output.

Also highly relevant to our thesis is the fact that a log normal probability distribution occurs inevitably as the ensemble behavior of many stochastic processes whose interactions are conditional upon each other's behavior. That is, the overall system behavior results from the product of many individual probabilities, rather than from their sum, as is the case for the normal distribution (36). The tail of the log normal distribution bears a very close resemblance to an inverse power function, which may explain the latter's ubiquity (36). Now consider a model of biological soft tissue in which energy is dissipated among its component fibers or molecules in a series of cascades as the yield stresses of the contact points between components are overcome in a random fashion. That is, each yield event alters the stresses in nearby contact points, thereby altering the probabilities that the nearby points themselves will yield. When a number of contact points yield in series, a cascade occurs. Furthermore, if each event contributing to a cascade is conditional upon the preceding event occurring, then the probability of the entire cascade occurring is given by the product of the probabilities of the individual events and so should follow a log normal probability distribution. Such an interdependence of events eventually leading to a cascade is precisely the type of mechanism that has been shown to produce SOC (2,7). This suggests that SOC and the log normal probability distribution may be governed by the same underlying phenomenology, namely, the multiplying of many independent probability distributions.

Our hypothesis, then, is that $1/f$ noise and the t^{-k} form of soft tissue stress adaptation are characteristics that arise from the ensemble properties of the components of complex systems. Biological soft tissue is composed of countless molecules, cells, and fibers and thus certainly qualifies as a complex system. The interactions between its components are also themselves certain to be complex, such as the fiber–gel interactions described by Burlatsky and Deutch (5). If a mechanism like SOC is ultimately responsible for power law relaxation functions in soft tissue, then strong nonlinearities at the elemental level are also necessary. Indeed, such a mechanism has recently been postulated for interacting fibers by Mijailovich *et al.* (22). Although these authors considered only the dynamics of a single pair of fibers, one can easily imagine that a network of them might exhibit the stochastic cascade behavior likely to produce SOC.

A corollary of our postulate is that the precise natures of the individual components and their interactions are not important for producing stress adaptation of the type given by Eq. 3—all that matters is that there are lots of them and that they interact in a sufficiently rich and general manner. Indeed, it has even been shown that in the much simpler system of a polymer in dilute solution, containing sufficiently long and coiled macromolecules, the form of the

macroscopic relaxation function of the solution does not depend on the specific chemical constituents of the polymer (29). This is not to say, of course, that every macroscopic tissue property should be independent of its components' properties. Presumably, for example, the actual value of k in Eq. 3 is very much influenced by the nature of the components—some components produce rapid stress adaptation while others adapt slowly. A complete understanding of tissue mechanics, however, requires that we know which aspects of a tissue's behavior follow directly from its components' individual properties and which result solely from their connectedness.

Implications for Lung Tissue Function

It is instructive to consider whether it is advantageous for lung tissue to behave like a $1/f$ system (or its t^{-k} time domain equivalent). The cardinal feature of such as system is that it has no resonant frequency or characteristic time constant and therefore has no preference for the particular frequency range or time scale over which it operates. Indeed, it is perhaps more enlightening to consider what the implications would be if lung tissue were not like this. Since lung tissue must oscillate during breathing, it would clearly be problematic if it preferred to do so at a particular frequency, because the frequency of breathing must vary over nearly two orders of magnitude, or even more, across species and under various conditions of rest and exercise (a mouse breathes at about 5 Hz whereas a resting human may breathe at about 0.1 Hz). Furthermore, lung tissue is in intimate contact with a number of other biological soft tissues (such as those of the heart, esophagus, pleural sac, and diaphragm). If each of these tissues had their own different optimal frequencies, there would clearly be problems of efficient operation of the entire organism unless the frequencies were all carefully matched. Since there are no preferred frequencies, however, the problem does not arise. Thus, from a functional point of view, time/frequency scale invariance allows the organ to adapt to a wide range of conditions. Such dynamic tolerance is similar to the geometric tolerance exhibited by a fractal airway tree to genetic or environmentally induced errors in its development during growth (37).

Another implication of Eq. 3 for lung tissue function, as pointed out by Hantos *et al.* (14), is that it automatically couples together the dissipative and conservative properties of the tissue. This fits with the well-known observation that the dissipative and conservative parts of lung tissue impedance always seem to change in concert in response to various interventions. Fredberg and Stamenovic (9) formalized this in terms of the structural damping hypothesis, in which they defined a quantity η, termed hysteresivity, as

$$\eta = R(f)/X(f), \tag{5}$$

where $R(f)$ and $X(f)$ are, respectively, the real (dissipative) and imaginary (conservative) parts of impedance. From Eq. 4 we get

$$\eta = \cos[(1 - k)\pi/2]/\sin[(1 - k)\pi/2], \tag{6}$$

which has a value of 0.14 when k is 0.09, as measured in whole lungs by Hantos *et al.* (14). This is similar to the values for η found by Fredberg and Stamenovic (9) under normal conditions. Indeed, other biological soft tissues associated with the respiratory system exhibit values of η that are similar, which points to the generality of Eq. 3.

Conclusions

In conclusion, we suggest that the qualitative nature of stress adaptation of lung tissue (indeed, biological soft tissue in general) is determined principally by the fact that it is composed of many richly interacting components, without particular regard to the nature of the individual components themselves. This is a feature that appears to be shared by many complex dynamic systems encountered in nature. We further suggest that our hypothesis has important implications for the current trend in biology of examining systems at ever increasing levels of magnification. Although cellular and molecular studies are obviously essential for a complete understanding of an entire organ, they will not lead to a complete understanding on their own. There are important aspects of an entire organ's behavior that arise independently of its components' individual properties. These aspects arise from the connectivity between the components and can be understood only by considering the organ as an entire system. In other words, an organ is more than just the sum of its parts.

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