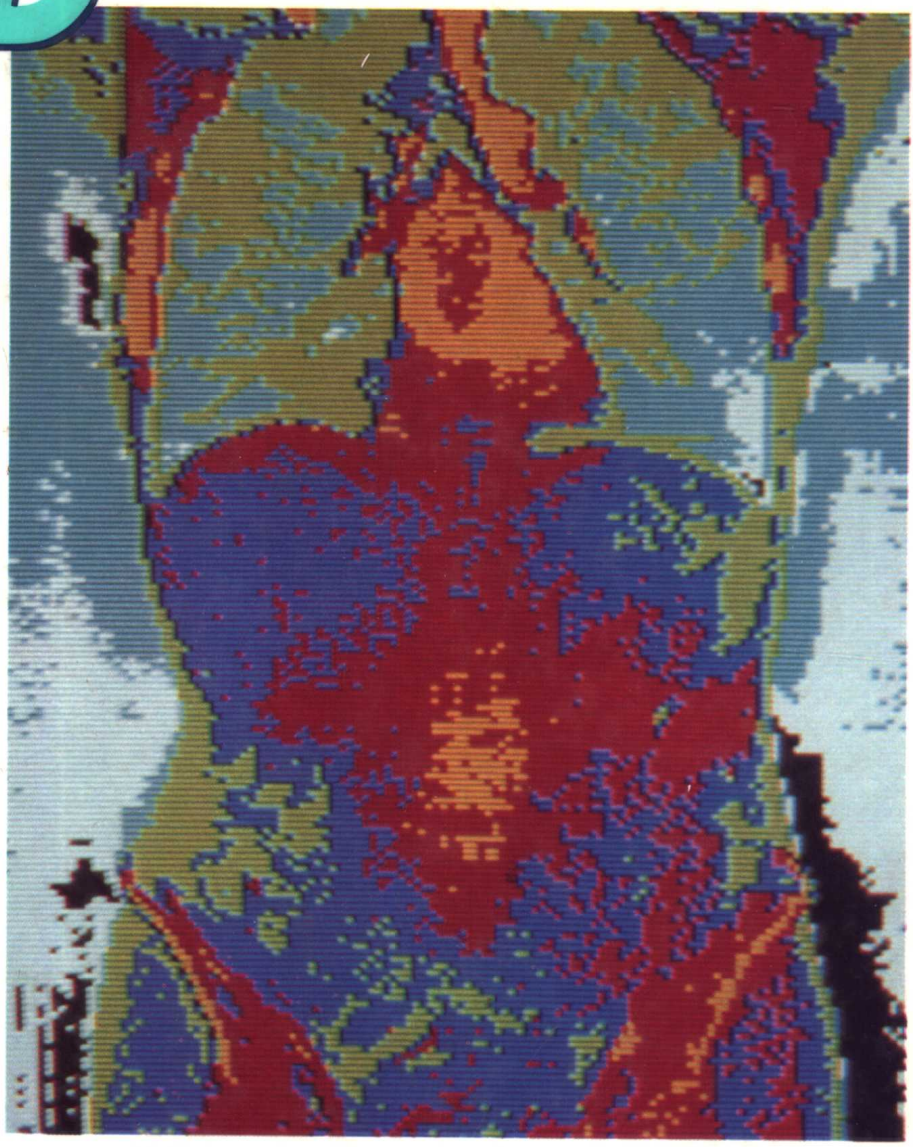
HANDBOOK OF BIOENGINEERING



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Handbook of Bioengineering

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Preface

The field of bioengineering has been developing rapidly in the last two decades. Although its precise definition is still evolving, there is a growing body of knowledge that forms the basis of this interdisciplinary subject. The purpose of this handbook is to collect, in one place, authoritative summary accounts of the various topics that comprise the field of bioengineering. The handbook is intended to serve as a reference for practicing bioengineers, medical doctors, biological scientists, and students interested in the subject. It will also serve to introduce bioengineering to other physical scientists such as physicists, mathematicians, and engineers who seek to enter the field.

The handbook is intended to be representative of the main areas of research and technology which have become known as bioengineering. The principal areas are orthopedics, cardiovascular mechanics, blood rheology, respiratory mechanics, and properties of hard and soft tissues. There are many additional topics that are not covered in this handbook, but sometimes included in the term bioengineering, such as fermentation processes, genetic engineering, and agricultural or food technology. To include all the possibilities would yield an unwieldy volume, and the present handbook is centered on aspects related to biomedical engineering.

The interdisciplinary nature and the diverse subject matter of bioengineering necessitates a collaborative effort of many individuals to present the state-of-the-art of the field. The authors of each chapter of the handbook have been chosen for their expertise in the particular fields. The technical merit of this work is entirely to the credit of the chapter authors, as the editors have served mainly in a coordinating capacity. The choice of subject content and presentation within each chapter has been the prerogative of the authors. We are most grateful and appreciative of their contributions, because they have given comprehensive accounts derived from their professional experience, condensing and presenting the best information available. Their insights define the developing field of bioengineering.

The handbook is organized to present the basic tissue properties first and proceeds to more applied subjects in the later chapters. Properties of soft

tissues, bone, tendon, ligaments, muscle, skin, and blood are covered in the early chapters. Later chapters cover models of the arterial system, the microcirculation, lung mechanics, cochlear mechanics, joint replacement, and other applications. It is the hallmark of bioengineering that quantitative, mathematical, and computational techniques such as those used in other disciplines of engineering are brought to bear on biomedical problems, both in the interpretation of biological phenomena and in the design of medical devices.

While the accounts in the handbook are as mathematical as the subject demands, sufficient descriptive background has been included so that nonbiologists can follow the medical applications and nonengineers can appreciate the physical principles involved.

It is anticipated that this handbook will be useful to students and faculty in the many departments of bioengineering that have been established at engineering and medical schools as a reference in lecture courses and as a starting point in research. It is also intended to be useful to bioengineers in industry, responsible for research and development of medical instrumentation and devices. It should also be helpful to biologists and physicians whose research or clinical practice calls for the acquisition of new instruments and in the quantitive modeling of research results. As medical practice becomes increasingly quantitative, bioengineering aspects become more important to both research and clinical applications.

Many people have contributed to the development of this handbook, particularly the many authors who have given it substance. It has been a pleasure to work with Betty Sun and Jim Halston of McGraw-Hill who have given us unfailing cooperation during the entire process of producing this volume. We also acknowledge with thanks, the very competent and thorough assistance of Louis Soslowsky in proofreading and coordinating manuscripts at Columbia University. His help has facilitated the progress of the project greatly.

For the omissions or imbalance of the material covered, the editors take responsibility and will appreciate comments from readers as to topics or types of coverage that may be desirable in future editions.

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Contents

CHAPTER 1

Mechanics of Soft Tissues

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INTRODUCTION

In this chapter the general features of the mechanics of soft tissues are presented. It will be shown that the mechanical properties of soft tissue are related to its structure. Moreover, the mechanical properties of an organ depend not only on its tissue, but also on its geometry and relationship to the neighboring organs. A typical example is the blood vessel. The capillary blood vessels of the mesentery are "rigid," whereas those in the bat's wing, for example, are "distensible." The capillaries of the lung can be both rigid and distensible. They are rigid in the plane of the capillary network, and compliant in the direction perpendicular to this plane. The stress-strain relationship of the systemic arteries is highly nonlinear, showing stiffening exponentially with increasing strains; yet that of the pulmonary arteries in the lung is linear. The systemic veins are easily collapsible; yet the pulmonary veins in the lung are not: they remain patent when the blood pressure falls below the alveolar gas pressure. These differences in mechanical properties arise because of the interaction between the blood vessels and the tissues surrounding the vessels. These differences in the mechanical properties of the blood vessels in different organs produce significant, sometimes dramatic, effects on the blood circulation of different organs.

Similarly, the mechanical properties of each tissue depend not only on its chemical composition but also on its structural or ultrastructural details. Two tissues made of the same proportions of collagen, elastin, and ground substances may behave quite differently depending on how these basic elements are put together.

More details about the properties of muscle, blood vessels, skin, uterus, lung, kidney, cochlea, spine, bone, cartilage, tendons, and ligaments are presented in separate chapters of this handbook.

1.1 PSEUDOELASTICITY

Soft tissues are pseudoelastic; that is, they are not elastic, but under periodic loading and unloading each tissue will have a steady-state stress-strain relationship which is not very sensi-

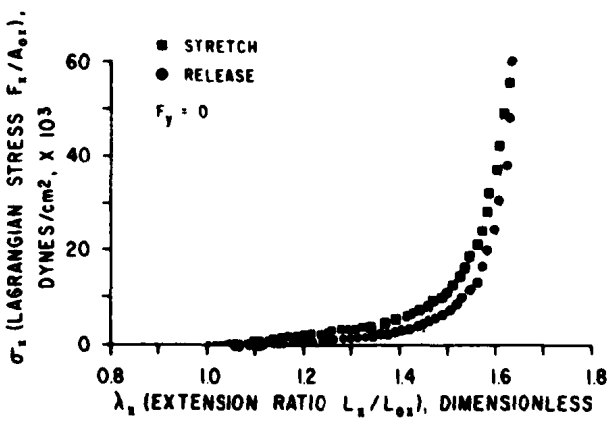

FIG. 1.1 A typical stress-strain curve for uniaxial loading. Every fourth data point is plotted. Note that the unloading curve is different from the loading curve, showing the existence of hysteresis. (*From Vawter, Fung, and West, 1978, by permission.*)

tive to strain rate. For example, Fig. 1.1 shows the stress-strain relationship of the lung tissue of the dog (with the airspace filled with saline so that the surface tension acting on the alveolar walls is very small) subjected to biaxial loading. After a number of cycles of loading and unloading, a repeatable stress-strain loop as shown in Fig. 1.1 was obtained. The existence of the loop shows that the tissue is viscoelastic and not elastic. But since the loop is repeatable, we can treat the loading and unloading curves separately and borrow the method of the theory of elasticity to describe the mechanical properties. Hence the term *pseudoelasticity*. Figure 1.2

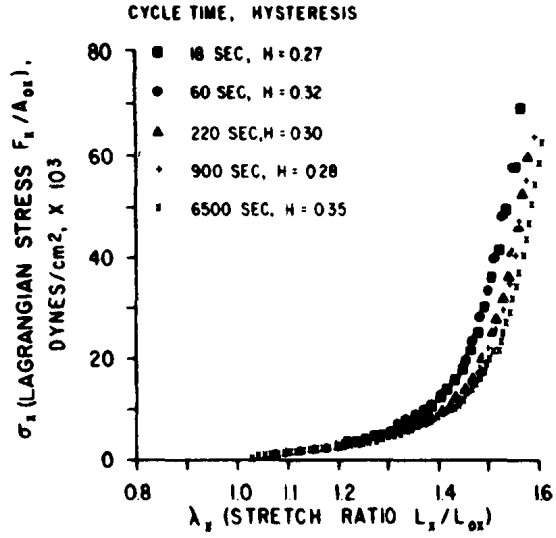

FIG. 1.2 Loading phase at different strain rates. Varying strain rate over 2.5 decades caused only small changes in response. The hysteresis H is the ratio of the area of hysteresis loop (not shown) to the area under the loading curve. The period of cycling and the values of H are given in the insert. (*From Vawter, Fung, and West, 1978, by permission.*)

shows the stress-strain relationship of the same lung tissue in *loading* at different strain rates. Each cycle was done at a constant rate. The period of each cycle is noted in the figure. It is seen that over a 360-fold change in strain rate there was only a minor change in the stress-strain relationship. The *hysteresis H*, defined as the ratio of the area of the hysteresis loop divided by the area under the loading curve, is also noted in Fig. 1.2. H is seen to be variable, but its variation with strain rate is not large. Similar experience is encountered with other tissues. Records of skeletal and cardiac muscles, ureter, teniae coli, arteries, veins, pericardium, mesentery, bile duct, skin, tendon, elastin, cartilage, and other tissues show similar characteristics. Typically, in a 1000-fold change in strain rate, the stress at a given strain in a loading (or unloading) process does not change by more than a factor of 2.

The features shown in Figs. 1.1 and 1.2 may be described by saying that living soft tissues are *nonlinearly pseudoelastic*. The stress-strain relationship is nonlinear, the viscoelasticity is pseudoelastic; hysteresis may be sizable, but it varies only mildly over a wide range of strain rates.

1.2 NONLINEAR STRESS-STRAIN RELATIONSHIP

Treating the loading and unloading curves separately, we can borrow the method of elasticity to describe the stress-strain relationship. The simplest way to represent the pseudoelasticity of a nonlinear material is to introduce a *pseudoelastic potential* (or *strain energy function*) $\rho_0 W$, which is a function of Green's strain components E_{ij}, symmetric with respect to E_{ij} and E_{ji}. The partial derivatives of $\rho_0 W$ with respect to E_{ij} gives the corresponding Kirchhoff stress components S_{ij}. W is defined for a unit mass of the tissue, and ρ_0 is the density of the tissue in the initial state; hence $\rho_0 W$ is the strain energy per unit initial volume. Thus

$$S_{ij} = \frac{\partial \rho_0 W}{\partial E_{ij}} \qquad (i, j = 1, 2, 3) \tag{1.1}$$

If the material is incompressible (volume does not change), then it can take on a *pressure* that is independent of the deformation of the body. In that case a pressure term should be added to the right-hand side of Eq. (1.1). The value of the pressure (as in water) can vary from point to point, and it can be determined only when the equations of motion, continuity, constitutivity, and boundary conditions are all satisfied.

An example of pseudoelastic potential for arteries and veins is the following (Chuong and Fung, 1983):

$$\rho_0 W = Ce^Q \tag{1.2}$$

where

$$Q = a_1 E_1^2 + a_2 E_2^2 + a_3 E_3^2 + 2a_4 E_1 E_2 + 2a_5 E_2 E_3 + 2a_6 E_3 E_1 \tag{1.3}$$

Here the vessel is assumed cylindrical and the coordinate axes x_1, x_2, x_3 are pointing in the circumferential, longitudinal, and radial directions, respectively; and $E_1 = E_{11}$, $E_2 = E_{22}$, $E_3 = E_{33}$, whereas E_{ij} $(i \neq j)$ is assumed to be zero. The constants C, $a_1, \ldots, a_6$ are the material constants that characterize the artery. For simplicity, cylindrical symmetry is assumed in this expression; otherwise one would have to generalize the quadratic form Q to include all six strain components. If the vessel wall can be considered homogeneous, then the constants C, $a_1, \ldots, a_6$ are independent of the location in the blood vessel. If, in addition, the arterial wall material is assumed to be incompressible, then the condition of incompressibility

$$(1 + 2E_1)(1 + 2E_2)(1 + 2E_3) = 1 \tag{1.4}$$

may be introduced through a lagrangian multiplier H. We define a new pseudoelastic function

$$\rho_0 W^* = Ce^Q + H[(1 + 2E_1)(1 + 2E_2)(1 + 2E_3) - 1] \tag{1.5}$$

so that

$$S_i = \frac{\partial(\rho_0 W^*)}{\partial E_i} \qquad (i = 1, 2, 3) \tag{1.6}$$

It is well known that H has the significance of a hydrostatic pressure which can assume an arbitrary value. The true value is determined by the equations of motion (or equilibrium) and boundary conditions.

For a body subjected to small changes in strains, the range of the incremental strains may be chosen to be so small that the relationship between the incremental stresses and strains is linear. This linear relationship is Hooke's law, for which the familiar material constants are the incremental Young's modulus and incremental shear modulus. For soft tissues a general feature implied by Eq. (1.2) is that the incremental moduli increase with increasing stresses.

1.3 VISCOELASTICITY

When a tissue is stretched suddenly to a new dimension which is then held constant, it will show the phenomenon of stress relaxation; i.e., the stress gradually decreases with increasing time.

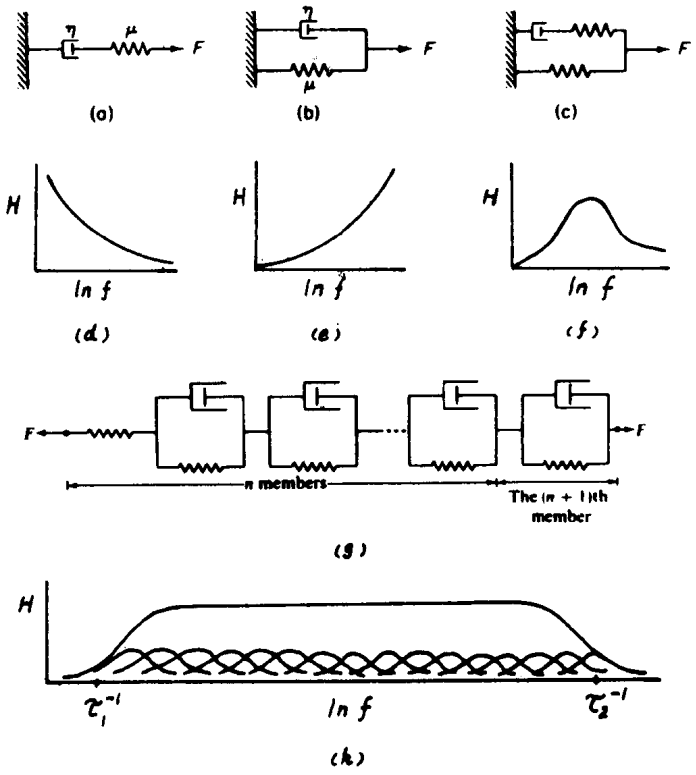

FIG. 1.3 Several models of viscoelasticity and their corresponding hysteresis-frequency relationships: (*a*) Maxwell model, (*b*) Voigt model, (*c*) Kelvin model; (*d*), (*e*), and (*f*) show hysteresis of models (*a*), (*b*), and (*c*), respectively. The hysteresis H is defined as the ratio of the area of hysteresis loop to the area under the loading curve. The abscissa is frequency in log scale. (*g*) is a generalized model for living soft tissues; (*h*) is the corresponding hysteresis versus frequency curve. Each member of the generalized model contributes a small bell-shaped curve. The sum of these small bell-shaped curves yields a curve of H which is almost constant over a wide range of frequencies. (See text for explanation.)

The degree of relaxation depends on the tissue. In time a ureter can relax off almost all the stress, whereas an artery can relax off only 20 to 30 percent, and a ligament nuchae relaxes off only a few percent. When the tissues are stressed suddenly by a step function and then the stress is kept constant, the tissue creeps; i.e., its strain increases with increasing time. The degree of creep also depends on the tissue but is usually fairly small in tendons, ligaments, skin, and arteries.

Under cyclic loading, these tissues show the phenomenon of hysteresis. As shown in Figs. 1.1 and 1.2, the hysteresis loops of soft tissues are relatively insensitive to strain rate.

These features—relaxation, creep, hysteresis, and strain rate insensitivity—can be condensed into a mathematical formulation called *quasi-linear viscoelasticity theory* (Fung, 1972). In this theory, the instantaneous stress response to strain is called the "elastic stress." The relationship between the elastic stress and strain is nonlinear. The stress at any given time is, however, related to the entire past history of the elastic stress. This history dependence is linear. The present stress is obtained by multiplying the past elastic stress increment with a relaxation function and integrating the product convolutionally from the beginning of time to the present. The relaxation function has a *continuous relaxation spectrum*, the meaning of which will be explained presently in the following paragraphs.

One of the two most popular models of viscoelasticity is the Maxwell model of a spring in series with a dashpot. Another is the Voigt model with a spring and a dashpot in parallel. A third is the Kelvin model, which is a combination of a spring in parallel with a Maxwell body. See Fig. 1.3*a* to *c*. None of these models can represent a soft tissue because, when a material represented by any one of these models is subjected to a cyclic strain, the hysteresis will not be insensitive to strain rate. As frequency increases, the dashpot in the Maxwell body will move less and less at the same load so that damping decreases with frequency. On the other hand, as frequency increases, the dashpot in the Voigt body will take up more and more of the load so that the damping increases with frequency. See Fig. 1.3*d* and *e*. For the Kelvin body there exists a characteristic frequency at which the damping is a maximum (see Fig. 1.3*f*). None of these has the feature of a nearly constant damping, independent of frequency, as soft tissues do.

A model suitable for the soft tissue is shown in Fig. 1.3*g*; it has an infinite number of springs and dashpots. In the corresponding hysteresis diagram shown in Fig. 1.3*h*, there are an infinite number of bell-shaped curves which add up to a continuous curve of nearly constant height over a very wide range of frequencies. In this situation, we say that the soft tissue has a *continuous relaxation spectrum*. The two ends of the spectrum, marked by frequencies τ_1^{-1} and τ_2^{-1} in Fig. 1.3*h*, define two characteristic times τ_1 and τ_2 which can be determined from experimental data. (See Fung, 1972, or Fung, 1981, pp. 232 ff., for mathematical details.) Tanaka and Fung (1974) have found τ_1 and τ_2 for various arteries of the dog: τ_1 lies in the range of several hundred to thousands of seconds; τ_2 lies in the range of 0.05 to 0.36 s. Chen and Fung (1973) showed that for the mesentery τ_1, τ_2 are 1.869×10^4 and 1.735×10^{-5} s, respectively. Woo et al. (1979) have found that for the cartilage $\tau_2 = 0.006$ s, $\tau_1 = 8.38$ s.

1.4 WHY DIFFERENT BLOOD VESSELS BEHAVE SO DIFFERENTLY

The aorta and thoracic arteries have nonlinear stress-strain curves, as indicated in Eq. (1.2). The pulmonary arteries and veins, in contrast, have linear pressure-diameter relationships, as shown in Fig. 1.4. The capillary blood vessels of the mesentery appear to be rigid—that is, without measurable change in diameter when blood pressure changes from 0 to 100 mmHg (Baez et al., 1960; Fung et al. 1966). However, the capillary blood vessels in the lung are very distensible. Figure 1.5 shows the variation of the thickness of the capillary sheet (the interalveolar septa) with the transmural pressure ΔP (blood pressure minus alveolar gas pressure). The pulmonary capillaries are closely knit into a sheetlike dense network which occupies about 90 percent of the total space in the interalveolar septa. Each sheet is exposed to gas on both sides. Figure 1.5 shows that the thickness of the pulmonary capillary sheets of the cat increases linearly with increasing transmural pressure at a rate of 0.22 μm/cmH_2O when ΔP is positive. But when ΔP is negative, the thickness quickly drops to zero. There is a sudden change of

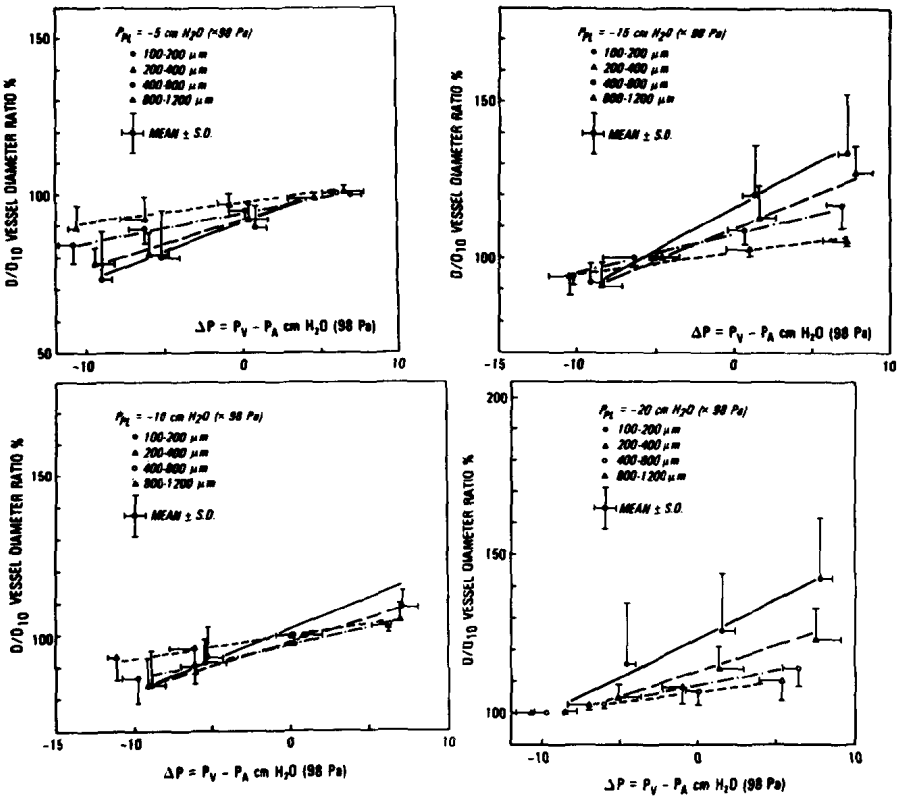

FIG. 1.4 The distensibility of pulmonary veins subjected to positive and negative $p_v - p_A$. The vessel diameter is normalized against its value when $p_v - p_{pL}$ is 10 cmH_20, at which the vessel cross section is circular. The points are classified according to their diameter at $p_v - p_{pL} = 10$. (*From Yen and Foppiano, 1981, by permission.*)

thickness at $\Delta P = 0$. When ΔP tends to 0 from the positive side, the limiting value of the sheet thickness is 4.28 μm for the cat. When $\Delta P < -1$ cmH_2O, the capillaries are all collapsed. Why do the capillaries of the lung behave so differently from those of the peripheral circulation?

There is another property of the blood vessels that has an important physiological effect: the stability of the vessel when the external pressure exceeds the internal pressure. We have seen in Fig. 1.5 that the pulmonary capillaries collapse when $\Delta P < 0$. But we know that peripheral capillaries do not collapse when blood pressure falls below tissue pressure (Fung et al., 1966). On the other hand, it is common knowledge that peripheral veins and venae cavae collapse when the blood pressure falls below the pressure in the surrounding media. But the pulmonary veins and venules do not collapse when the airway pressure exceeds the blood pressure (see Fig. 1.5 and Fung et al., 1983). Why do these vessels behave so differently when their composition and histology are very similar?

The answer is that the properties of a blood vessel depend not only on the intrinsic properties of the blood vessel wall but also on the properties of neighboring tissues. The peripheral capillaries in the mesentery are embedded in a gel. Since a gel behaves as a solid, a capillary blood vessel embedded in it behaves as a tunnel in solid earth. That is why the capillary is so rigid with respect to blood pressure and so stable with respect to compression (Fung et al., 1966). On the other hand, a pulmonary capillary is exposed to alveolar gas which provides little