Experimental Studies

Physiological Interpretation of Negative Circumferential Tension in Vascular Walls

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SUMMARY

The balance of force in a cylindrical tube (Oka-Azuma equation) indicates existence of a negative circumferential tension in the wall of blood vessels *in vivo*. However, we conventionally consider only a positive wall tension due to a positive transmural pressure in blood vessels. In this paper, the nature of such a negative circumferential tension in the vascular wall was reconsidered. The same type of negative tension or compressive force was shown to exist in any hollow and solid biological tissues placed in the atmospheric pressure. In living tissues, which have abundant water and are incompressible to external pressure, the compressive force by atmospheric pressure is simply the internal fluid pressure. The fluid pressure is a scalar and does not produce any effective net force vector. For this reason, there is no need to consider the compressive force component produced by the environmental pressure in the balance of force in living tissues.

Additional Indexing Words:

Laplace relation Circumferential tension Compression Thick-walled vessel External pressure Transmural pressure

OKA^{1),2)} and Oka and Azuma³⁾ theoretically considered the balance of force in a blood vessel and found that the circumferential tension in a thick-walled blood vessel is usually negative, i.e., compressive. Azuma and Oka⁴⁾ calculated circumferential tension and mean stress in blood vessels of various diameters and wall thicknesses and confirmed them to be usually compressive under physiological transmural pressures. However, Caro et al⁵⁾ stated that the compressive forces generated in thick-walled vessels by atmospheric pressure are not very important in practice. Other recent books on biological mechanics^{6),7)} do not touch upon such compressive forces.

This study reconsidered the nature of the state of compression in a vascular wall proposed by Oka and Azuma³⁾ and found that such compression in water-abundant and incompressible biological tissues is simply a tissue fluid pressure. Pressure is a scalar and does not produce any effective net force vector. Therefore,

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Fig. 1. Unit-length segment of thick-walled blood vessel. The cross-hatched area in Panel A is a longitudinal, radial cross-section perpendicular to the circumference. T =circumferential tension; p_e =extraluminal pressure; p_i =intraluminal pressure. Panel B shows the balance of force. r_e =external radius; r_i =internal radius; d =wall thickness.

the compression produced by the environmental pressure does not need to be considered in the balance of force in living tissues.

Theoretical background

In the following text, "force", "tension", and "stress" should be distinguished from "pressure". "Force" is the generic term of the pulling, attracting, or pushing effect between two objects. "Tension" is a force working perpendicular to a unit length of an object. "Stress" is a force working perpendicular to a unit cross-sectional area of an object. Force, tension, and stress are often used interchangeably in physiology. In contrast, "pressure" is a force working perpendicular to a unit area of an actual or hypothetical surface of an object in contact with gas or liquid. Pressure is a scalar, i.e., working equally in all directions, whereas force, tension, and stress are vectors, i.e., working in specific directions. All objects to be considered are stationary with no acceleration. Gravitational force is neglected.

Oka^{1),2)} and Oka and Azuma³⁾ considered the balance of force in a blood vessel, where *absolute* intraluminal pressure is p_i , *absolute* extraluminal pressure is p_e , and the circumferential wall tension is T , as shown in Fig. 1A. The absolute pressure stands for the pressure relative to the vacuum, not relative to the atmospheric pressure; the absolute value of the normal atmospheric pressure is 1 atm. The circumferential tension is assumed to be positive if it is tensile, and *negative if it is compressive*.

Oka^{1),2)} and Oka and Azuma³⁾ considered the resultant forces acting on the outer and inner surfaces of the cylinder, as shown in Fig. 1B. They are given by $2r_e p_e$ and $2r_i p_i$, respectively, where r_e and r_i are the outer and inner radii of the vessel. Then, they derived the following equation for the force equilibrium:

$$2r_e p_e + 2T = 2r_i p_i$$

or

$$T = p_i r_i - p_e r_e. \quad (1)$$

Eq. 1 holds quite generally; it is valid whether the wall is homogeneous or inhomogeneous, whether the wall is isotropic or anisotropic, and whether the elasticity of the wall is Hookean or nonlinear.¹⁾⁻³⁾ Eq. 1 holds whether or not the material is a living tissue.

Oka and Azuma's interpretation

Oka^{1),2)} and Oka and Azuma,³⁾ and Azuma and Oka⁴⁾ applied Eq. 1 to actual conditions.

i) When the wall is infinitely thin ($r_e=r_i=r$), Eq. 1 reduces to

$$T = (p_i - p_e)r. \quad (2)$$

Eq. 2 is simply Laplace's law for a thin-walled tube.⁸⁾

ii) When the internal pressure is equal to the external pressure ($p_i=p_e=p$), Eq. 1 reduces to

$$\begin{aligned} T &= p(r_i - r_e) \\ &= -pd \end{aligned} \quad (3)$$

where d is the wall thickness and is equal to $r_e - r_i$.

Eq. 3 indicates that T is always negative in the thick-walled cylinder placed in any positive atmospheric pressure. Oka,^{1),2)} Oka and Azuma,³⁾ and Azuma and Oka⁴⁾ interpreted the negative T as indicating that "the blood vessel is actually in a state of compression instead of tension as a whole."

iii) Based on Eq. 1, Azuma and Oka⁴⁾ calculated T and mean stress (T/d) values of different vessels from the aorta via capillaries to the vena cava under the conditions of atmospheric external pressure and physiological intravascular blood pressure. Their results indicated that T and mean stress are generally negative, i.e., compressive, except in the aorta, large arteries, and the vena cava.

General responses

After deriving the same equation as Eq. 1, Fung⁹⁾ did not discuss the possibility of compression in the vascular wall. Following the same equation as Eq. 1, Caro et al⁵⁾ wrote "if the wall of a tube is thick enough, i.e. if r_e is greater than r_i by a large enough amount, this tension can be negative, even when p_i exceeds p_e . In other words there is a compressive stress in the wall." However, they wrote "The fact that the underlying hoop stress may be compressive is not necessarily very important for the mechanical behavior of blood vessels in vivo." This is because in the circulation we normally wish to analyze small departures from some equilibrium state. . . . it is common to quote values of tension and pressure relative to a given equilibrium state, and to ignore the absolute values."⁵⁾ More recently, Fung^{6),7)} has discussed the balance of forces in a blood vessel, but neglected the compression in a thick-walled vessel placed in atmospheric pressure. Other investigators have referred to the Oka-Azuma equation,¹⁰⁾⁻¹³⁾ without considering compression of the vascular wall. However, the rationale for ignoring the compression has not been fully developed.

New physiological interpretation

None of the reports referring to Oka and Azuma's proposal³⁾ provide con-

Fig. 2. A cubic (Panel A) and an irregular shaped object (Panel B) under atmospheric pressure (p). Cross-hatched area is a hypothetical cross-section. F = total force working perpendicular to the cross-sectional area.

vincing evidence about the plausibility of negative circumferential tension or compression in vascular walls and the nature of the compression. The following interpretation can clarify the reason why we do not need to adopt Oka and Azuma's compression concept in living tissues, including blood vessels.

i) Non-specificity of compression to blood vessels

Let us first consider a solid cube as shown in Fig. 2A. In the atmosphere, all surfaces of the cube are subjected to atmospheric pressure. The pressure (p) is the only external force acting on this cube and the net force vector is zero. The total force (F) perpendicular to any hypothetical total cross-section of the cube must be compressive. The compression vanishes only when the cube is placed in the vacuum.

The same situation holds in a piece of living tissue under atmospheric pressure. However, living tissues usually contain abundant water (60–80%) and are permeable to gases. The water in living tissues not only dissolves atmospheric gases according to their partial pressures and solubilities, but also immerses various structural materials. In addition, living tissues can reasonably be assumed incompressible to environmental pressures with a Poisson's ratio of approximately 0.5.^{4),14)} Namely, living tissues are not deformed by the atmospheric pressure when all dissolved gases are equilibrated with the environmental pressures (see "Final remarks" below). Thus, the tissue structural materials are not deformed as compared to a reference state in the vacuum (see "Final remarks" below). Therefore, the compression in the living tissue is nothing but the tissue fluid pressure.

This tissue fluid pressure is equal to the atmospheric pressure (see "Final remarks" below). This situation depends on neither the shape of the living tissue nor whether the object is solid or hollow, as shown in Fig. 2B. The compressive force (F) per hypothetical total cross-section of the object is given by the tissue fluid pressure (=atmospheric pressure) times the total cross-sectional area, as shown in Figs. 2A and B.

The compression of a vascular segment placed in the atmosphere ($p_o = p_i = p$) has a compressive tension $T = -pd$ (Eq. 3). This tension is equal to the atmospheric pressure (p) times the cross-sectional area ($d \times \text{unit length}$) perpendicular to the circumference. The compression produces a tissue fluid pressure of the same magnitude as the atmospheric pressure. The same compressive tension ($-pd$) is equally obtained in an intact vascular tube or in both longitudinal pieces, as

Fig. 3. Cross-section of a vessel (Panel A) and two longitudinal strips of a vessel (Panel B). T = circumferential tension produced by the atmospheric pressure (p); d = wall thickness.

Fig. 4. Compression of an object by atmospheric pressure (Panel A) and by fingers (Panel B). Thin arrows indicate atmospheric pressure and thick arrows manual forces. The tissue structure is not deformed (dotted lines) by compression under pressure, whereas it is deformed (dotted curves) by compression with fingers. Cutting out the cross-section in the middle of the pressure-compressed object decreases the compressive force between the two halves (Panel C), whereas the same procedure on the manually compressed object keeps the same compressive force between the two halves (Panel D). In Panels C and D, the thick horizontal lines are opened cross-sections, whereas the dashed lines are intact cross-sections.

shown in Figs. 3A and B. Thus, the tubular vascular segment is not a necessary condition for the compressive force given by the Oka-Azuma equation. The compression by atmospheric pressure is not specific to a hollow tissue such as a blood vessel. It is general to any living tissue of any shape.

ii) Two types of compression

In a stationary fluid compressed by atmospheric pressure, equal pressures are working on any region of the fluid in all directions. The net force vector on this fluid is zero. Therefore, the compression in a vascular wall by the atmospheric pressure is working not only in the circumferential direction but also equally in all

other directions. This compression is substantially different in nature from the compression produced by a finite net force vector in a specific direction on the object. In tissue compressed by atmospheric pressure, the only force that can exist in the fluid is the fluid pressure because a force vector in the fluid causes flow until all fluid becomes stationary. There is no deformation of the tissue structure because of the incompressibility to pressure (Fig. 4A). However, if the same living tissue is placed in a vacuum and manually compressed to the same compressive force at the same cross-section (Fig. 4A), the force vector causes both tissue fluid compression and tissue deformation, which change the strain and stress conditions in the tissue structural materials (Fig. 4B).

The difference between these 2 types of compression can be better visualized when the objects are cut in half (Figs. 4C and D). In the pressure-compressed object (Fig. 4C), the gradually increased cut-out cross-section is pressurized by the atmosphere and the compressive force through the gradually decreased remaining cross-section decreases although compressive mean stress remains unchanged. The completely separated two halves remain compressed by the atmospheric pressure, but the two halves are no more compressed against each other. The same situation holds in the case of Fig. 3B.

In contrast, the two halves of the manually compressed object is always pressed together while it is being cut into halves as shown in Fig. 4D or even after it has been cut. This discussion holds good regardless of the material of the object as in the case of Fig. 1.

The substantial differences between these 2 types of compression provide the rationale for neglecting the compression generated by the atmospheric pressure in living tissues. It is therefore reasonable that we conventionally measure both active and passive forces of contractile and non-contractile tissues with respect to the zero force level, adjusted for an unloaded force transducer. This is standard practice for calculating myocardial forces.¹⁵⁾

iii) Biological effect of pressure

Does the fluid pressure in contractile tissues affect their contractile properties? The contractile properties of muscle tissues have been studied under a wide range of fluid pressures. The rat diaphragm muscle showed an increase in force with a 1.5 fold increase in fluid pressure from 1 atm to 137 atm.¹⁶⁾ A similar increase in force and a doubling of contraction time were observed when intact fish muscles were subjected to a fluid pressure rise to 400 atm.¹⁷⁾ By contrast, glycerinated muscles displayed an 8% decrease in force with increases in fluid pressure to 100 atm.¹⁸⁾ These data indicate that a fluid pressure change by 1 atm from the normal atmospheric pressure does not significantly affect contractile properties of living tissues, probably including blood vessels.

Final remarks

Several points must be added to the discussion in the preceding sections. First, at steady state under a given atmospheric pressure, all dissolved gases in the tissue are equilibrated with atmospheric gases. When the tissue is rapidly decompressed by a lower atmospheric pressure, dissolved gases will form bubbles within the tissue, leading to *Kaisson's disease* and ebullism. However, only the equilibrium state was considered in our discussion. Second, only the total compressive force across the entire thickness of the wall of a tube or across an entire

solid tissue has been considered. The distribution of forces, tensions and stresses depends entirely on the degree of inhomogeneity and anisotropy of the material. Although compressive force per a cross-section is finite, tensions and stresses can vary among the layers and the sites in a cross-section. Although the compressive force and mean stress are zero in the vacuum, there may be positive and negative stresses (residual stress) in different sites. A good example of this is the wide opening when a tubular segment of a blood vessel or a heart is longitudinally cut open.⁷⁾

A third point is that the fluid pressure in a tissue may not be equal at all sites because the tissue fluids are separated by cell membranes. Intracellular pressure will be higher than extracellular pressure when the myofibrils contract.¹⁹⁾ A positive transsarcolemmal pressure is generated by the increased mechanical potential energy that reflects the deformation of the cell structure during shortening.¹⁹⁾ Moreover, tissue fluid pressures may vary from site to site.²⁰⁾ These issues were not considered.

To summarize, any solid or hollow object is compressed by atmospheric pressure. However, in aqueous living tissues, pressure-induced compression exists in the form of tissue fluid pressure. Therefore, contractile forces can be measured without consideration of the pressure-induced compression.

REFERENCES

1. Oka S: Theoretical studies on hemorheology (abstract in Japanese). The 16th Rheology Symposium, Sapporo, 1967, *Zairyo* **17**: 300, 1968
2. Oka S: Cardiovascular Hemorheology, Cambridge Univ Press, Cambridge, p 114, 1981
3. Oka S, Azuma T: Physical theory of tension in thick-walled blood vessels in equilibrium. *Biorheology* **7**: 109, 1970
4. Azuma T, Oka S: Mechanical equilibrium of blood vessel walls. *Am J Physiol* **221**: 1310, 1971
5. Caro CG, Pedley TJ, Schroter RC, Seed WA (ed): The Mechanics of the Circulation, Oxford Univ Press, Oxford, p 100, 1978
6. Fung YC: Biomechanics: Mechanical Properties of Living Tissues, Springer-Verlag, New York, p 17, 1981
7. Fung YC: Biodynamics: Circulation, Springer-Verlag, New York, p 23, 1984
8. Burton AC: On the physical equilibrium for the small blood vessels. *Am J Physiol* **164**: 319, 1951
9. Fung YC: Biomechanics: Its scope, history, and some problems of continuum mechanics in physiology. *Appl Mech Revs* **21**: 1, 1968
10. Ashton H: The effect of increased tissue pressure on blood flow. *Clinic Orthopaed Rel Res* **113**: 15, 1975
11. Furness JB, Marshall JM: Correlation of the directly observed responses of mesenteric vessels of the rat to nerve stimulation and noradrenaline with the distribution of adrenergic nerves. *J Physiol* **239**: 75, 1974
12. McCaffrey J: Aspects of the microcirculation. *Aust NZ J Surg* **47**: 413, 1977
13. Misra JC, Choudhury KR: A study on the stability of blood vessels. *Rheol Acta* **21**: 340, 1982
14. Lawton RW: The thermoelastic behaviour of isolated aortic strips of the dog. *Circ Res* **2**: 344, 1954
15. Suga H, Goto Y, Nozawa T, Yasumura Y, Futaki S, Tanaka N: Force-time integral decreases with ejection despite constant oxygen consumption and pressure-volume area in dog left ventricle. *Circ Res* **60**: 797, 1987

16. Kendig JJ, Cohen EN: Neuromuscular function at hyperbaric pressures: pressure-anesthetic interactions. *Am J Physiol* **230**: 1244, 1976
17. Wardle CS, Tetteh-Lartey N, MacDonald AG, Harper AA, Pennec JP: The effect of pressure on the lateral swimming muscle of the European eel *Anguilla Anguilla* and the deep sea eel *Histobranhus Bathybius*; results of Challenger cruise 6B/85. *Comp Biochem Physiol* **88A**: 595, 1987
18. Fortune NS, Geeves MA, Ranatunga KW: Pressure sensitivity of active tension in glycerinated rabbit psoas muscle fibers: effects of ADP and phosphate. *J Mus Res Cell Motil* **10**: 113, 1989
19. Rabbany SY, Kresh JY, Noodergraaf A: Internal load of the contracting myocyte. Proceedings of the 9th International Conference and the Satellite symposium, The Cardiovascular Systems Dynamics Society, Halifax, Nova Scotia, Canada, p 77, 1988
20. Heineman FW, Grayson J: Transmural distribution of intramyocardial pressure measured by micropipette technique. *Am J Physiol* **249**: H1216, 1985