

Journal of Biomechanical Engineering: Legacy Paper 2017

The *Journal of Biomechanical Engineering* has been in continuous production since 1977. To honor papers published at least 30 years ago that have had a long-lasting impact on the field, the Editors now recognize “Legacy Papers.” The journal is pleased to present the following paper as this year’s Legacy Paper:

“*On Residual Stress in Arteries*,” by C. J. Chuong and Y. C. Fung, *ASME J Biomech Eng* **108**(2): 189–192.

This work has been cited directly over 650 times (Google Scholar), but certainly has been referenced many more times via citations of Y. C. Fung’s trilogy of books on *Biomechanics* published by Springer, New York, which together have been cited more than 10,000 times.

This 1986 paper by Chuong and Fung revealed a remarkable influence of residual stress (i.e., the stress that exists in a body in the absence of external loads) on intramural stress in arteries subjected to in vivo conditions. In particular, the in-plane residual stresses—compressive in the inner portion of the wall and tensile in the outer portion—tend to homogenize the transmural distribution of circumferential and axial stresses in vivo, which has myriad biomechanical and mechanobiological implications.

Chuong and Fung reported, for example, that the predicted circumferential stress at the inner wall of the thoracic aorta is 6.5 times larger than the mean (Laplace) value when residual stress is neglected, but only 1.42 times larger when residual stress is included. That is, neglecting residual stress can result in an over 350% error in calculating maximum stress.

To appreciate better both the novelty of this work and its implications, it is useful to consider some chronological developments in modern vascular mechanics. Albeit not widely recognized, in a 1960 Ph.D. thesis at the University of London, D. H. Bergel observed: “When an artery is split longitudinally it will unroll itself to a varying degree to the form of a flat ribbon. This surely indicates some degree of stress even when there is no distending pressure.” The potential importance of this residual stress in arteries was not appreciated for the next 20+ years, however, and numerous papers reporting increasingly more sophisticated nonlinear constitutive relations and stress analyses came to the same mistaken conclusion—that intramural stresses are highly nonuniform in vivo, with a concentration of circumferential stress in the inner (intimal) layer of an artery. Though some suggested that such a stress concentration could help explain the development of atherosclerosis, an intimal disease, much remained to be learned.

In 1983, Y. C. Fung (University of California, San Diego, CA) and R. N. Vaishnav (Catholic University, Washington, DC) independently rediscovered Bergel’s 1960 observation and presented at different conferences images of longitudinally cut arterial rings “springing open” to relieve the underlying residual stress. Vaishnav and his colleague, J. Vossoughi, calculated associated values of residual strain and used a simple model based on linear

elasticity to estimate the associated residual stress. They suggested that the residual stress was approximately 14% of the mean (Laplace) value. A paper was promptly submitted (November 1983), though not published until early 1987 (*Journal of Biomechanics*, **20**, pp. 235–239). In it, they correctly concluded that residual stress “does help in reducing the severity of the nonuniformity of stress distribution.” Apparently appealing to principles of superposition common in linear elasticity, however, the reduction in stress at the inner wall was predicted to be a modest 14%.

In contrast, in his first report (published in *Biomechanics in China, Japan, and USA: Proceedings of an International Conference held in Wuhan, China, 1983*), Fung did not address the existence of residual stress quantitatively. Rather he suggested potential implications, including that “the residual stress in a tissue distributes itself in a way to assure such [optimal] performance,” and “Growth and resorption may reduce stress concentration, but in the process residual stresses are created.” In other words, Fung suggested that residual stresses develop naturally in soft tissues via growth and remodeling processes, and they serve to improve or optimize mechanical performance. Clearly, there was a pressing need for quantitative understanding.

It was on this backdrop that Chuong and Fung published, in 1986, their seminal paper on residual stress in arteries. Rather than appealing to linear elasticity and superposition, they appropriately employed an important result from nonlinear elasticity (pp. 49 in Green, A. E., and Adkins, J. E., *Large Elastic Deformations*, 2nd ed., Clarendon Press, Oxford, UK, 1970) to prescribe the motion of a material particle when mapped from an opened circular sector to a closed (residually stressed) cylinder. Similarly, though not elaborated, they used an appropriate multiplicative (not additive) decomposition of the subsequent motion from the intact, residually stressed configuration to the in vivo configuration. Despite not checking the axial equilibrium equation, their intuition was correct—the residual stress-related axial stretch was nearly unity. Their calculated values of residual stress ranged from -1.5 (inner wall) to 1.5 (outer wall) kPa, values an order of magnitude smaller than those estimated by Vaishnav and Vossoughi (-19 kPa at the inner wall). Because of the sequential, large deformations, the impact of even small residual stresses on the computed in vivo stress field was found to be dramatic, reducing the predicted inner wall stress from over 650 kPa to less than 250 kPa, a more than 2.5-fold reduction rather than the mere 14% reduction predicted previously.

Subsequent constitutive refinements by others, including the addition of basal smooth muscle tone and differential properties of the media and adventitia, suggested further reductions in the inner wall stress and the existence of nearly uniform transmural stresses under normal conditions (see Chapter 7 in Humphrey, J. D., *Cardiovascular Solid Mechanics: Cells, Tissues, and Organs*, Springer, New York, 2002), thus supporting Fung’s original

insight that residual stresses appear to optimize the mechanical function of arteries. Indeed, a uniform stress field (on average overall, but also separately in each layer) suggests the important mechanobiological hypothesis that intramural cells seek to establish and then maintain a preferred mechanical state. As foreseen by Fung, therefore, the concept of residual stress and its influence on arterial wall mechanics stimulated an important new era of research in mechanobiology and modeling mathematically the growth and remodeling of arteries as well as many other soft tissues. See, for example, the 2001 paper by Taber and Humphrey (*ASME J Biomech Eng* **123**(6), 528–535), and the 2003 paper by Rachev and Greenwald (*Journal of Biomechanics*, **36**, pp. 661–670) for developments in arterial mechanics that were inspired by the publication of Chuong and Fung’s seminal paper and appeared within ~15 yr thereafter. The subsequent ~15 yr has continued to yield increasing understanding of the fundamental processes of growth and remodeling in diverse soft tissues, which often implicitly address residual stress but increasingly focus on the “optimal performance” highlighted by Fung in 1983.

In conclusion, it is suggested here that it was because Y. C. Fung combined a broad appreciation of fundamental mathematical results in nonlinear elasticity with an appreciation of the wonders of biological processes such as growth that he and C. J. Chuong were able to understand residual stresses in arteries and anticipate their far reaching consequences. We should all seek the same. Finally, I would recommend that the interested reader consider the personal account by Fung regarding the discovery of the importance of residual stress as delivered in the 1989 ALZA Lecture at New Orleans, LA (*Annals of Biomedical Engineering*, **19**, pp. 237–249, 1991), titled “What are the residual stresses doing in our blood vessels?” The answer, as we now know, is “they are doing a lot”.

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