How to design a structure able to mimic the arterial wall mechanical behavior?

M. R. JOUAN
Department of Material Engineering, Laval University, Québec, Canada; Unit of Bioengineering and Biotechnology, St François d'Assise Hospital, Québec, Canada

M. N. BUREAU, A. AJJI, M. A. HUNEAULT
Industrial Materials Institute, National Research Council, Boucherville, Canada

D. MANTOVANI
Department of Material Engineering, Laval University, Québec, Canada; Unit of Bioengineering and Biotechnology, St François d'Assise Hospital, Québec, Canada

Cardiovascular diseases, particularly arterioscleroses, count among the most frequent and fatal ones in the industrialized countries [1]. Arteriosclerosis is caused by the formation of atheromateous plate constituted by proliferation and deposits of cholesterol in lipidic scratches involving thickening of the intima, which leads to a stenosis. Clinical complications are also related to the possibility of calcification eventually leading to a rupture, cholesterol embolus and ulceration of the thrombotic plate. From a clinical point of view, it is often required to replace the diseased and structurally damaged section of the artery and implant a vascular substitute, made from either synthetic (Teflon or Dacron prosthesis) or biological (autologous graft) materials. These substitutes must conform to several requirements such as hemocompatibility, mechanical reliability and iso-compliance, i.e., graft compliance as similar as possible to that of arteries to ensure their harmonious implantation. This compliance requirement is essential to obtain similar deformation under pressure in the prosthesis and adjacent artery. Failure to obtain acceptable compliance may result in improper anastomosis and mechanical mismatch, which eventually lead to the failure of the implant [2]. Several studies have focused on the compliance of arteries to better understand their behavior [3–6].

Quite compelling insights on successful vascular grafts can also be obtained from a good understanding of the architecture of the arterial wall. In fact, the arterial wall is composed of three principal layers with very specific properties [7, 8]. The *intima*, the internal layer of the arterial wall (∼10% thickness of the wall), is composed of a unique layer of endothelial cells and serves as an internal artery liner reducing shearing of blood on the wall. It is not considered to contribute significantly to the elastic properties of the artery. The *media*, the intermediate layer of the arterial wall (∼70% thickness of the wall), is the structural component of the artery. It is composed of elastin and collagen fibers circumferentially organized that define most the elastic properties of the artery. Smooth muscles are also present in muscular arteries, but their mechanical properties can be considered as negligible. The *adventitia*, the external layer of the arterial wall (∼20% thickness of the wall), is composed of elastin and collagen fibers and contributes to the elastic properties of the artery, although to a lesser extent than the *media* does. The principal role of this layer is to allow anchoring to the surrounding tissues.

Since the *media* accounts for most of the elastic properties of the artery, it is considered to play a key role in regard to the elastic properties of the artery. The elastic moduli of the elastin and collagen fibers are reported to be approximately 3 and 1000 MPa, respectively [9]. The elastin fibers form a network of random macromolecular entanglements that constitute the matrix of the *media*. The collagen fibers form a 3D network of aligned fibers within the elastin matrix in the *media*. Due to their large differences in modulus and structure, the resulting pressure vs. deformation curve for the *media*, which constitutes the compliance curve, shows a characteristic concave shape, also called "J" shape [10], schematized in Fig. 1. As suggested in Fig. 1, this characteristic shape is caused by the sequential deformation of the elastin and collagen fibers upon loading. First, elastin fibers undergo re-orientation into the principal directions by rotation and translation due to their low modulus and random orientation. This results in the first part of the compliance curve characterized by a slope E_1 characteristic of the elastin property with a limited contribution from the collagen fibers. Upon deformation, the 3D network of collagen fibers is progressively oriented, which increases the stiffness of the material in the deformation direction and results in an increasing slope up to the E_2 value of the collagen fibers. It thus appears that, when the pressure increases in the artery, the wall is submitted to tension, which leads to artery radius increases up to the structural limit imposed by the stronger and more rigid collagen fiber network.

A striking similarity between this behavior of the arteries and the behavior of orthogonal fabrics made from rovings of structural continuous fiber reinforced polymer composites can be observed. When mechanically loaded for in-plane rotation and elongation, these fabrics initially oriented at $\pm 45^\circ$ with respect to the loading

0022–2461

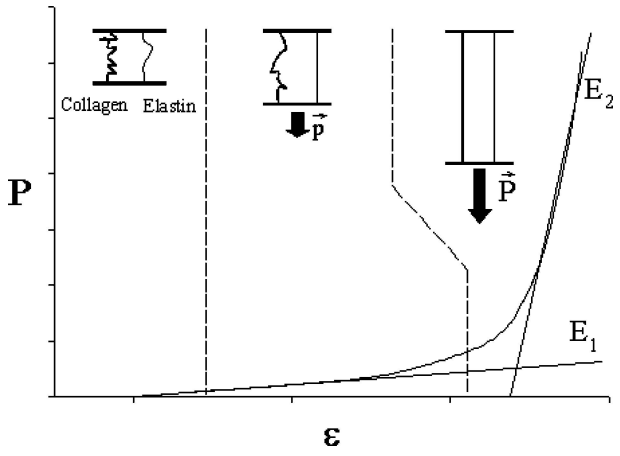

Figure 1 Schematic load (P) vs. deformation (ε) curve, or compliance curve, of the *media*. E_1 and E_2 are elastic moduli characteristic of the elastin and collagen phases, respectively. The schematics represent conceptually the deformation of both phases under pressure.

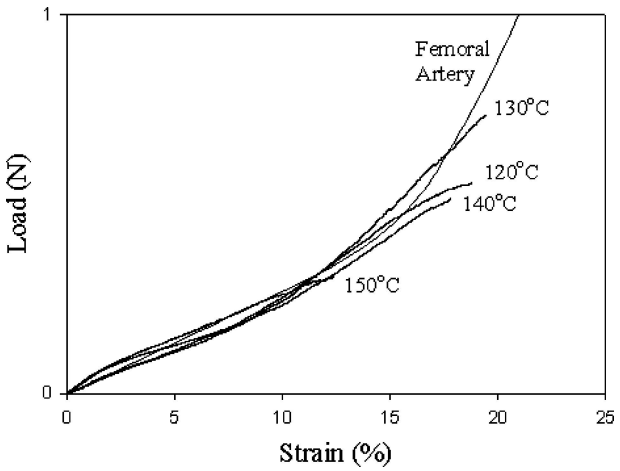

Figure 2 Stress–strain curves of fabric samples prepared (under different consolidation temperatures) and femoral artery from Ref. 13. Fabric samples with 50% of longitudinal fibers in the stretching direction are represented for two veils (120 °C), four veils (130 °C) and three veils (140 °C and 150 °C). Temperatures refer to consolidation.

direction present a concave shape, or "J" shape, load vs. extension curve [11]. This shape was attributed to a pure intraply shear deformation, or in-plane rotation, of the fabric structure under uniaxial elongation during which the angle between the two roving directions decreases gradually until a critical angle value, or locking angle, is obtained as a result of the complete rotation of the rovings. Once this locking angle is reached, the composite rovings progressively undergo extension and the slope of the load vs. extension curve increases rapidly as a result of their high modulus. This behavior has been discussed in the literature [11]. It was shown that the first part of the curve was controlled by the in-plane transverse shear of the fabrics associated to a "transverse modulus factor" and the second part of the curve was controlled by the longitudinal deformation of the fabrics associated to a "longitudinal modulus factor" [11]. From this description of the behavior of composite fabrics, it appears that iso-compliant vascular scaffolds could be obtained from continuous polymer fiber fabrics.

Preliminary results were obtained from thin non-woven mats of polypropylene (PP) fibers using the melt blowing process coupled with an attachment that controls the fiber entanglement and fiber distribution in the veil [12]. These mats were then stacked into multi-ply (20) veils and submitted to a heat treatment. Polypropylene was selected because of its hemocompatibility and its abundant use for manufacturing commercial suture wires. Polypropylene fibers with 6–14 μm in diameter were produced. The heat treatment, or consolidation step, lasted typically 15 min at a temperature ranging between 120 and 150 °C. The nominal thickness of these non-woven fabrics was 0.5 mm. Although PP fibers show an elastic modulus considerably higher than that of the arterial wall, it is possible to control and tailor the shape and moduli of the load vs. extension curve of the non-woven fabrics by controlling the inter-fiber bonding and the respective orientation of the individual veils upon stacking. Flat specimens with a nominal 40-mm width were obtained from the non-woven fabrics and tested in tensile mode at a crosshead speed of 12.5 mm/min. These tensile curves are shown in Fig. 2 at different consolidation temperatures. These curves show the characteristic J-shape aspect mentioned, with a weaker first slope (E_1) that gradually increases to a second stiffer slope (E_2) at approximately 15% in deformation. The first slope can be attributed to in-plane rotation of the non-woven fabric structure under uniaxial elongation until the PP fibers are individually stretched, which defines the second slope of the curve at high deformation. These results show that the conditions in which the nonwoven fabrics are consolidated, especially the temperature, have an important effect on their shape and first and second slope values. Also included in Fig. 2 is the curve of *in vitro* femoral arteries taken from Blondel [13]. Stress-strain curve comparison between the fabrics and femoral arteries reveal a close similarity in their behavior. They show the same characteristic J-shape aspect and the same moduli and critical deformation. From these similarities in behavior, it appears that the first part of the curve in the non-woven fabrics, during which alignment of fabric fibers occurs, corresponds to the elastin fiber re-orientation, while the second part of the curve during which PP fibers are progressively stretched corresponds to collagen fiber deformation.

It can be concluded that the behavior of an arterial wall can be mimicked using consolidated stacks of non-woven veils of polypropylene. The number of veils, their stacking angle, and their consolidation conditions can be used to tailor the mechanical properties of the material. Finally, from the rationalization of this J-shape compliance behavior of textile composites fabrics into two "modulus factors" [11], it will be possible to adjust the properties of the non-woven PP fabrics to match those of the arterial wall.

References

1. S. YUSUF, S. REDDY, S. OUNPUU and S. ANAND, *Circulation* (2001) 104.

2. C. GIANNATTASIO, F. ACHILLI, A. GRAPPIOLO, M. FAILLA, E. MELES, G. GENTILE, I. CALCHERA, A. CAPRA, J. BAGLIVO, A. VINCENZI, L. SALA and G. MANCIA, *Hypertension* **1** (2001) 38(6).
3. K. HAYASHI, K. MORI and H. MIYAZAKI, *Am. J. Physiol. Heart. Circ. Physiol.* (2003) 284(2).
4. M. E. HANSEN, E. K. YUCEL, J. MEGERMAN, G. J. L'ITALIEN, W. M. ABBOTT and A. C. WALTMAN, *Cardiovasc. Intervent. Radiol.* (1994) 17(1).
5. M. STEVANOV, J. BARUTHIO and B. ECLANCHER, *J. Appl. Physiol.* (2000) 88(4).
6. N. STERGIOPULOS, J. J. MEISTER and N. WESTERHOF, *Am. J. Physiol.* (1995) 268(4).
7. A. STEVENS and J. LOWE, in "Human Histology" (Mosby, London, UK, 1999) Vol. 2.
8. W. KAHLE, H. LEONHARDT and W. PLATZER, in "Color Atlas and Textbook of Human Anatomy" (Georg. Thieme. Pub., Stuttgart, 1978).
9. Y. C. FUNG, *Am. J. Physiol.* (1967) 213(6).
10. C. S. ROY, *J. Physiol.* (1880) 3.
11. G. LEBRUN, M. N. BUREAU and J. DENAULT, *Composite Structures* **4** (2003) 61.
12. A. AJJI, in Proceedings of the 18th annual meeting of the Polymer Processing Society. Guimarães, Portugal, June 2002, CD-ROM.
13. W. C. P. M. BLONDEL, B. LEHALLE, X. WANG and J. F. STOLTZ, *Rheol. Acta.* (2000) 39.

Received 14 July
and accepted 19 October 2004