

Hannah Weisbecker

Softening and Damage Behavior of Human Arteries

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Softening and Damage Behavior of Human Arteries

Experiments, Constitutive Modeling and Numerical Simulation

This work is based on the dissertation "*Softening and Damage Behavior of Human Arteries. Experiments, Constitutive Modeling and Numerical*", presented by Hannah Weisbecker at Graz University of Technology, Institute of Biomechanics in February 2014.

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HANNAH WEISBECKER



Hannah Weisbecker holds a Diploma degree from Dresden University of Technology, Germany. She has a background in Mechanical Engineering with specialization in Applied Mechanics, having completed her master degree with distinction. During her studies, she worked on a research project at the University of Concepción, Chile dealing with the numerical evaluation of the acoustic eigenvalue problem with present ambient flow velocity. Hannah's Diploma Thesis dealt with damage detection via 3D scanning laser vibrometer measurements and was realized as a joint project between the Dresden University of Technology and the University of Adelaide, Australia. During her undergraduate studies, Hannah has actively collaborated with international researchers from various fields and published two research papers in international journals.

At the Institute of Biomechanics at Graz University of Technology, Austria, Hannah completed her PhD with distinction. Under the supervision of Professor Gerhard A. Holzapfel, she worked on modeling the softening and damage of human arterial tissue. Her research aimed at better understanding the mechanical response of arterial tissue during catheter based procedures spanning from experimental studies and constitutive modeling to development and application of the finite element method for arterial tissue. It describes the behavior of the three arterial layers and analyzes the influence of the constituents on this behavior. Hannah's scientific findings have resulted in four original papers and have been presented at several international conferences.

Abstract

Cardiovascular diseases are the leading cause of death in the western world. Modern medicine is moving towards less invasive surgery such as catheter-based interventions that reduce pain and hospitalization for the patient. However, these interventions are highly demanding for the surgeon due to the lack of direct feedback from the surgical site. During these interventions high stresses and even damage may occur, e.g., due to stent expansion and balloon inflation. Numerical models, informed by experimental data on the mechanical response of human tissue at supra-physiological loads, can improve the design of stents, balloons and surgical procedures. This PhD Thesis aims to develop a computational framework for modeling catheter-based interventions with an appropriate constitutive damage model fitted to experimental data for human arterial tissues. Consequently, the work comprises three major topics, namely, experiments, mathematical modeling and computational simulation.

The experimental work involves uniaxial extension tests on human aortas in both physiological and supra-physiological load ranges. These tests are conducted on the intact wall, on the separated layers, intima, media and adventitia, and on the enzyme treated media. In the enzyme treated specimens elastin and collagen, the main load bearing components of the arterial wall, are degraded to better understand the contributions of these components on the damage and softening behavior.

For modeling the softening behavior of the tissue, an established hyperelastic constitutive model was extended with a damage function. Such a strain-energy function is capable of describing the Mullins-like behavior of arterial tissue. The experimental data, together with results from fitting the model, suggest that softening and damage are primarily associated with the collagen fibers.

The framework for numerical simulations to model damage in patient-specific geometries accounts for initial stresses, i.e. residual stresses and prestresses. Residual stresses are those stresses present in an unloaded artery to adjust transmural stresses at physiological loads that result from the different material properties of intima, media and adventitia. A method to include the residual stresses on patient-specific geometries is developed. If the geometry of an artery is derived from *in vivo* medical imaging, it is loaded by blood pressure. Hence, the finite element algorithm has to account for this load on the given geometry. This work presents an advanced method to include prestresses which reduces computational costs compared to available algorithms.

In the future, more detailed experimental data on the inter-patient and intra-patient variability in the mechanical response of human tissue and thus of material parameters will further enhance the framework for patient-specific modeling of damage in human arteries. Furthermore, multiscale models may lead to a better understanding of the damage mechanism.

Zusammenfassung

Krankheiten des Herz-Kreislauf-Systems sind die führende Todesursache in der westlichen Welt. Die moderne Medizin entwickelt katheterbasierte Eingriffe, durch die Schmerzen gelindert und Krankenhausaufenthalte verkürzt werden. Solche Eingriffe stellen hohe Ansprüche an den Chirurgen, da dieser kein direktes Feedback von dem Ort des Eingriffes erhält. Außerdem kann es z. B. beim Einsetzen eines Stents und beim Aufblasen eines Ballons zu hohen Spannungen und Schädigungen im Gewebe kommen. Mit numerischen Modellen ist eine Verbesserung der Formgebung von Stents und Ballons möglich. Eine Voraussetzung dafür sind experimentelle Daten der mechanischen Eigenschaften des menschlichen Gewebes. Diese Arbeit stellt eine numerische Simulationsmethode für katheterbasierte Operationen vor, die auf ein durch experimentelle Daten gestütztes konstitutives Modell für menschliches Arterien Gewebe zurückgreift. Sie befasst sich mit den drei Teilaspekten: Experimente, mathematische Modellierung und numerische Simulation.

Der experimentelle Teil der Arbeit untersucht das mechanische Verhalten von menschlichen Aorten bei physiologischen und supra-physiologischen Dehnungen anhand uniaxialer Zugversuche. Unterschieden wird dabei zwischen den drei Schichten, Intima, Media und Adventitia. Die mechanisch relevanten Komponenten der Arterienwand, Elastin und Kollagen, werden in der Media spezifisch von Enzymen verdaut. Auf diese Weise lässt sich der Einfluss beider Komponenten auf die Schädigung im Gewebe untersuchen.

Zur Modellierung der Entfestigung des Gewebes dient ein etabliertes hyperelastisches Modell, das durch die Erweiterung mit einer Schädigungsfunktion den „Mullinseffekt“ in arteriellem Gewebe beschreibt. Die experimentellen Daten zeigen, zusammen mit den Ergebnissen aus dem Parameterfit des Modells, dass die Entfestigung des Gewebes hauptsächlich mit den Kollagenfasern zusammenhängt.

Das numerische Schädigungsmodell berücksichtigt sowohl Residuumsspannungen als auch Vorspannungen im Gewebe. Residuumsspannungen treten in der unbelasteten Arterienwand auf, um die Spannungsgradienten über die Wandstärke auszugleichen, die durch die unterschiedlichen Materialeigenschaften von Intima, Media und Adventitia auftreten. Eine in der Arbeit entwickelte Methode bindet die Residuumsspannungen in patientenspezifische Geometrien ein. Wird die Geometrie einer Arterie aus *in vivo* Bildgebungsverfahren rekonstruiert, ist sie bereits mit Blutdruck belastet und dadurch verformt. Dies muss im finite Elemente Algorithmus berücksichtigt werden. Dieser Teil der Arbeit beschreibt eine Methode, um Vorspannungen zu berücksichtigen, welche den notwendigen Rechenaufwand gegenüber bestehenden Algorithmen verringert.

In der Zukunft können detailliertere experimentelle Daten über die Veränderlichkeit von Materialkennwerten in einem Patienten und zwischen verschiedenen Patienten numerische Modelle weiter verbessern. Außerdem ermöglicht die Weiterentwicklung von mehrskaligen Modellen ein besseres Verständnis für die Schädigungsmechanismen.

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